RISK ASSESSMENT AND COMMUNICATION AFTER CHILDREN'S EXPOSURE TO ENVIRONMENTAL TOXICANTS

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SUMMARY
EXECUTIVE SUMMARY

Environmental toxicants are a persistent and increasing cause of preventable diseases in children. Childhood exposures are also known and suspected to be associated with adult-onset diseases. With technological advancements in our society, the number and variety of chemicals to which humans are exposed have increased significantly in the last 20 years. Children are exposed to a diverse number of environmental toxicants at home, school, play, and daycare settings as well as indirectly through their parents'/guardians’ occupational and environmental exposures. There are over 80,000 chemicals in commercial use today in the United States with hundreds of new chemicals being developed and released into the environment every year. In addition, children are at risk for exposure to nearly 15,000 high-production-volume synthetic chemicals, most of which have been developed in the past 50 years. Thus, the extent of children’s exposures to potentially toxic substances will undoubtedly continue to increase. Many of these materials are contained in household products and dispersed widely in the environment. A significant proportion of chemicals have never been tested for their potential toxicity, and thus potential dangers to children are unknown. Since the toxic effects of most of these chemicals, particularly long-term effects, are largely unknown, it is difficult to assess and communicate accurate concerns to health care providers and the public.

Historical incidents demonstrating environmental poisoning of children provide the scientific foundation for further research into the relationship between environmental exposure and disease. Chemicals including PCBs (polychlorinated biphenyls), hexachlorobenzene, lead, and organic mercury have caused exaggerated toxicity in children compared to adults, illustrating the vulnerability and unique susceptibility of children. Unique exposure patterns, immaturities in physiological development, and different target organ susceptibilities as well as dependence on others to separate them from potentially harmful exposures make infants and children an especially vulnerable segment of population that requires special attention.

Children’s environmental health issues are clearly an important health priority for the new millennium. Effects of environmental toxicants on children are a growing concern. It has been estimated that over a million children less
than the age of 6 live within one mile of toxic waste sites on the National Priority List. (The National Priority List is the Environmental Protection Agency’s listings of the most seriously contaminated hazardous waste sites in the country in need of cleanup because of their threat to human health and the environment.) From a public health standpoint, what are the potential short- and longterm effects of these chemicals on developing children? Recent research has suggested that one of every 200 children in the United States now suffers from a developmental or neurological disability which was caused by an environmental toxicant. As a result of increasing awareness and publicity of environmental threats to children, pediatric health care providers (PHCPs) will need to be able to address questions and concerns from a child’s caretaker/parents as well as the local daycare and school. Parents may have specific requests for concrete information such as “What chemicals in our well water can be harmful to my children?”, “What lead level will be harmful to my child?”. Other questions may be more difficult to answer; for example, “What is the chance my child will develop cancer from the contaminants found in our garden soil?” , “Is my child’s learning disability due to the carbon monoxide leak from our faulty furnace system?” or “What long-term effects might my child encounter from exposure to low levels of radon in the basement?”. It is of utmost importance for pediatricians and other pediatric health care providers to educate themselves and their colleagues about these issues which are threatening America’s 70 million children.

General pediatric textbooks have little detailed information on environmental illness while original research and specific topics on these issues are dispersed throughout the literature and internet in sources not traditionally read or accessed by pediatric health care providers. Medical schools are estimated to provided no more than four to six hours of instruction in environmental and occupational health. Until the recent change in pediatric residency curriculum requirements and some teaching about lead poisoning, pediatric training programs were generally not teaching about children’s environmental health. The American Academy of Pediatrics recently published a Handbook of Pediatric Environmental Health which provides an excellent overview and concise summaries of environmental hazards to children, as well as practical information to be used in the office setting on diagnosing,
treated, and preventing childhood diseases associated with environmental exposures. However, once an environmental exposure has occurred or is suspected to have occurred, there is a need to provide an accurate assessment of the exposure and determine the risk of potential harmful effects for the child, if any. Furthermore, it is important to be able to communicate information to parents and the public about potentially hazardous environmental exposures in an accurate, ethical, and practical manner.

The objective of this monograph is to discuss the processes of risk assessment and risk communication as they apply to children’s exposures to potentially toxic substances in the environment. The terms “children” and “child” refer to infants, children, and adolescents except where specific differences are noted. First, the basics of taking an exposure history will be reviewed in association with brief discussions of particular environmental toxicants that the pediatric health care provider should be familiar with. Next, the traditional components of quantitative risk assessment (QRA) will be outlined, discussing in detail these analyses and their limitations. Special characteristics of the pediatric population that need to be considered in assessing exposure and toxicity will also be discussed. Unique susceptibilities of children to cancer as they relate to environmental toxic exposures will be reviewed as well as the role of biological markers in pediatric risk assessment. New approaches to risk assessment in children will be proposed. National initiatives that include the pediatric population in the risk assessment process will be addressed. Finally, selected information resources that might be helpful in understanding risk assessment and the various analyses will be reviewed.

The second section of the monograph will focus on the process and components of successful risk communication in relation to pediatric environmental health exposures. Specifically, it will discuss the multidimensional definition of risk and qualitative factors affecting people’s perception and acceptability of risk—both issues essential for communicating risk successfully. In addition, general principles and specific components about explaining risk will be reviewed. Accurate and practical communication to parents, caretakers, school personnel, and the media about hazardous exposures and potential adverse health effects is necessary in order to
protect our children, to prevent unnecessary fear and anxiety, and to further promote healthy environments for our children.

**PART I – RISK ASSESSMENT**

I. TAKING AN EXPOSURE HISTORY

It is important to be familiar with the essential components of an environmental exposure history. Identifying hazards in a child’s environment first requires that the health care provider take an adequate exposure history and be familiar with adverse health effects of many common pollutants. There are five general areas of questions that the PHCP can incorporate into health supervision and illness visits to assess for environmental causes. (Table 1) These questions have been previously developed and elicit information on exposures in the home and environment including indoor air pollution, common household products, pesticides and lawn care products, lead products and waste, recreational hazards, water supply, and soil contamination.  

**Exposures in the home, daycare, or school:**

Infants and toddlers spend most of their time indoors in their own home or daycare settings, while older children spend time at school and various outdoor and indoor activities. Important features of these various environments to consider are listed below.

*Type of dwelling:* Children living in private homes, apartments or mobile homes may be exposed to asbestos, radon, or formaldehyde. Homes, daycares or apartments may have high levels of radon in basements or lower floors where this colorless, odorless gas tends to concentrate. Radon is a natural by-product of uranium decay. It has no immediate health effects, but is associated with an increased risk of lung cancer after a long latency period. The progeny of radon attach to airborne particulates such as cigarette smoke and can be inhaled. The progeny emit high-energy alpha particles that may further injure bronchial cells, subsequently causing lung cancer. Exposed individuals who smoke further increase their risk of developing lung cancer. Approximately 5-10% of single-family homes in the
United States exceed the EPA radon recommended guidelines of 4 picocuries per liter of air. PHCPs should consult their local health department to determine whether radon is considered to be a significant risk in their area.

Asbestos was widely used from the 1950s to early 1970s in areas of the home requiring sound proofing, thermal proofing, or durability (floor and ceiling coverings, heating and water pipe insulation). When asbestos becomes frayed or friable, its fibers can be released into the air. Exposure to these fibers has been associated with lung cancer, mesothelioma, and asbestosis. Adults who smoke and are exposed to asbestos have more than 50 times the background rate of lung cancer.149

Children living in mobile homes may be exposed to formaldehyde. Formaldehyde may volatilize from particle board, insulation materials, carpet adhesives, and other materials used in mobile home construction.138 Formaldehyde exposure can cause upper respiratory and eye irritation as well as skin irritation, headache, nausea and vomiting.

Age and condition of the building – Buildings and homes constructed before 1960 are likely to still contain high amounts of lead in paint which may be peeling or chalking and thus accessible for ingestion and inhalation by children. Lead poisoning continues to be a significant health problem for children in the United States. Buildings with plumbing problems, roof leaks or history of flooding may have problems with mold growth. Mold can be a significant respiratory irritant and allergen. Some children exposed to mold have persistent upper respiratory tract symptoms such as rhinitis, sneezing, eye irritation as well as lower respiratory tract symptoms such as coughing and wheezing.46,87 Exposure to Stachybotrys and other molds has been associated with acute pulmonary hemorrhage in young infants.34,56,109,124

Newer homes may be built to conserve energy and subsequently have inadequate ventilation, resulting in increased concentrations of indoor air pollutants. These “tight” homes may be the source of numerous complaints including cough, asthma precipitation, upper respiratory irritation, eye irritation, and headache.
Ongoing or planned renovation – Improper renovations may expose children and adults to lead or other dusts, asbestos, and molds. Newly installed carpets may release irritating or toxic vapors.

Heating sources – Wood stoves and fireplaces emit noxious gases including carbon monoxide, nitrogen oxides, respirable particulates and polycyclic aromatic hydrocarbons, especially when they are not properly maintained and vented. Carbon monoxide can cause fatigue and lethargy at low concentrations and headaches, dizziness, weakness, confusion, nausea/vomiting, and death at higher concentrations. The other gases are respiratory irritants. Studies have shown that children living in homes heated with wood stoves have a significant increase in respiratory symptoms compared with children living in homes without wood stoves.\textsuperscript{79} Gas ranges may produce nitrogen oxide which is a respiratory irritant. In low-income areas, gas stoves may be used not only for cooking but as a supplemental source of heat. In addition, nitrogen dioxide exposure can result from using gas stoves for humidifying a room by keeping a pan of water steaming on the stove.

Indoor and outdoor pesticides – Pesticide exposure can occur through dermal contact, inhalation, or ingestion. Common pesticides used in the home, lawn-care products, or schools include organophosphates and carbamates, although there has been an increasing use of relatively less toxic pyrethrin products for common household bug sprays. Acute high-level exposures to organophosphates and carbamates can cause a constellation of symptoms including excessive salivation, tearing, sweating, runny nose, wheezing, nausea, vomiting, diarrhea, and muscle paralysis. Effects due to chronic low-level exposures in children are less clear. Pyrethrins, which are found in many household insecticide sprays, have low levels of acute toxicity, but may cause respiratory symptoms in selected people who are allergic to chrysanthemums. Information about chronic toxicity is lacking.

Proximity to sites of potential hazardous exposures – It is important to determine if a child’s home, daycare, or school is near a polluted lake or river, current or former industrial plant, specific commercial businesses, or dump site. Children may be exposed to lead if they live downwind from a lead smelter or battery plant. They may be exposed to various water pollutants if these drain into home wells or soil where home gardens are planted. In addition, if they
live near or on a farm, they may be exposed to pesticides either directly or indirectly. PHCPs should be aware of recent incidents of toxic emissions in their area.

**Schools** – Many environmental hazards found in school environments are similar to those found in homes. Children and adolescents may be exposed to lead and asbestos as these materials in older schools age and deteriorate. The EPA estimated in 1986 that friable asbestos may be present in as many as 35,000 schools in the United States, potentially exposing 15 million school children. The EPA requires school districts to follow certain preventive approaches to controlling asbestos in schools. Children engaging in arts and crafts projects may encounter hazards through their use of clay products and oil-based paints which are legally allowed to contain lead, cadmium, or chromium.

Problems involving potentially hazardous chemical exposure and poor indoor air quality (IAQ) are associated with increased use of manufactured construction materials, energy conservation measures that have sealed school buildings more tightly, inadequate air exchange that fails to eliminate pollutants from inside school buildings and moisture problems that cause biological growth like mold inside school buildings. The U.S. Government’s General Accounting Office (GAP) states that 46% of American schools have problems with indoor air quality, with many of the schools found in poorer school districts. This problem may play a role in the rise of asthma among children in the last 20 years. Sources of indoor air pollution in schools include: dust and dirt in heating and ventilation systems; venting of furnace gases into air ducts; dust and gases from photocopiersons, computers, and printers concentrating in poorly ventilated spaces; maintenance products—disinfectants, cleansers, pesticides, solvents, or carpet glues—release volatile organic vapors into the breathing zone; new furniture and floorings off-gas volatile components for months after installation; molds and spores thrive in water-damaged furniture and carpets. The EPA has developed an “Indoor Air Quality Tools for Schools” program which shows schools how to carry out a practical plan of action to improve indoor air problems.

Concern over children’s exposures to pesticides in the school setting has led to government action. Comprehensive nationwide information on the amount of pesticides used in this country’s public schools is not
available. Data from the American Association of Poison Control Centers shows that from 1993 through 1996, about 2,300 pesticide-related exposures involved individuals at schools. The Federal Insecticide, Fungicide, and Rodenticide Act regulates the use of pesticides in the United States, but there are no specific provisions in the law about the use of pesticides in schools. The Environmental Protection Agency and a number of states have taken initiatives and actions over the last decade to reduce the use of pesticides in schools by employing alternative pest management strategies. These alternatives are commonly referred to as integrated pest management. Further details can be found in the GAO Report on Pesticides—Use, Effects, and Alternatives to Pesticides in Schools.183

**Hobbies** – Numerous hobbies and recreational activities are associated with exposures to potentially toxic substances. (Table 2) It is important to be familiar with adverse health effects of common hobbies that the child or parent engages in to accurately assess a toxic exposure.

**Exposure to environmental tobacco smoke (ETS):**

Children exposed to secondhand ETS are at risk for significant morbidity and mortality. ETS is associated with increased frequency of respiratory infections, reactive airway disease, and recurrent otitis media in children, in addition to increasing a child’s risk for sudden infant death syndrome (SIDS) and development of lung cancer as an adult.5,89,161,178 Parents should be educated about the associations between ETS and children’s illnesses and they should be advised to quit if they currently smoke. Furthermore, visitors including babysitters and relatives should not be allowed to smoke around children. “I don’t smoke around the baby” is a common parental response but certainly not an adequate one as air exchanges and smoke on clothes spread smoke rapidly through the home. Dangers of cigarette smoking should be discussed directly with school-age children and teenagers.

**Parental and other care provider occupations and adolescent employment:**

“Take-home” exposures may result from parental/care provider occupational exposures. Workplace contaminants may be brought home on clothes, shoes, skin surfaces, and in cars of the parent or caretaker. (Table 3) Take-home exposures reported include lead poisoning in children of lead storage battery workers, elevated mercury
levels in children whose parents worked in a mercury thermometer plan, and asbestos-related diseases in families of shipyard workers.\textsuperscript{81,92,192} Workers are legally entitled to be notified of potential exposure to toxic substances under federal “right-to-know” and “hazard communication” laws. They should shower and change clothes and shoes before leaving work or immediately upon entering the house in a specially designated area. Parents may also work at home with toxic substances or participate in certain recreational activities that may expose children to toxicants. Children should not be allowed in these areas or rooms of the house.

Recent EPA investigations and reports of increased number of asbestos-related deaths in Libby, Montana, the world’s largest known deposit of natural vermiculite, demonstrate take-home contamination as a potential significant source of toxic environmental exposure for non-workers/family members.\textsuperscript{2} Workers were occupationally exposed to high concentrations of asbestos fibers released during the mining and milling of vermiculite.\textsuperscript{3,167} Household members of workers were potentially exposed to asbestos fibers adhering to hair, shoes, and work clothes brought home. Exposure and resulting health effects due to asbestos from worker take-home have been thoroughly documented in other cohorts.\textsuperscript{7}

**Diet:**

A child’s diet may place him/her at risk for exposure to environmental toxicants. Questions to parents and caregivers should include whether mothers are breastfeeding, smoke, or taking any medications. Some pesticides such as PCBs (polychlorinated biphenyls) and PBBs (polybrominated biphenyls) are lipid-soluble and concentrate in the breast milk.\textsuperscript{4}

Lead in water may contribute to low-level lead poisoning in formula-fed infants and toddlers. The water supply in the home should be tested for lead if it used for cooking and drinking purposes. Hot tap water and water from “instant hot taps” should not be used in making formula or drinking because of the possibility of lead contamination. Overboiling water may concentrate lead and should be avoided.\textsuperscript{150}
Pesticide residues in fruits and vegetables may be a harmful source of toxic chemicals to children.\textsuperscript{119} Toddlers’ diets typically contain large amounts of fresh fruits and vegetables per body weight, potentially exposing them more to chemicals with possible carcinogenic, neurotoxic, endocrinologic, and immunotoxic effects. Currently, there are still a lot of unknowns about the long-term effects of exposures to pesticides. Parents should encourage children to eat a variety of fruits and vegetables. Produce should be washed with water only to remove residues. Buying organic produce may reduce pesticide consumption if it is available and affordable.

**Exposure to lead:**
Childhood lead poisoning is a significant cause of morbidity in young children in the United States. Lead-related questions have been outlined in detail in other sources and in Table 1 based on recommendations from the Centers for Disease Control. For many reasons, lead poisoning is one of the most preventable environmental illnesses today. Identifying high-risk children through blood lead screening programs and appropriate questions has resulted in successful management, further exposure, and better primary prevention. However, primary prevention activities through anticipatory guidance, public education, identification and control of sources of lead exposure before children are born will ultimately eliminate this significant environmental toxicant.

**Questions Relating Illness to Environmental Exposure:**

Environmental causes of illness may not always be easily discernible unless an appropriate exposure history and assessment is obtained. Environmental or occupational illnesses may present with nonspecific symptoms and as common medical problems like headache, nausea, and upper respiratory symptoms. If the illness is atypical or unresponsive to supportive or specific interventions, the diagnosis may be linked to an environmental exposure. The following questions may provide information about whether the illness is related to the environment:\textsuperscript{41}

1.) Do symptoms subside or worsen in a particular location (i.e. home, child care, school)?
2.) Do symptoms subside or worsen on weekdays or weekends? Any particular time of day?
3.) Do symptoms worsen during hobby activities such as working with arts and crafts?
4.) Do children your child spends time with experience similar symptoms to your child’s symptoms?

II. RISK ASSESSMENT PROCESS

GENERAL PRINCIPLES ABOUT RISK ASSESSMENT

Heightened awareness of the possible health risks of chemicals in the environment has led to public demands for more information about the toxic effects of both new and existing chemicals. The process of health risk assessment has emerged as a systematic approach to responding to questions and concerns being raised. By definition, risk assessment is the analysis of a risk situation. (Table 4) Health risk assessment denotes a process of research and evaluation to quantify the probability of physical harm to humans attributable to a particular agent or agents. Risk assessment was originally developed for well-defined and easily analyzed mechanical problems such as building and bridge constructions. In the 1960s risk assessment first began to be used to estimate safety and risk associated with environmental exposures to carcinogens, especially in food products. The National Research Council published *Risk Assessment in the Federal Government: Managing the Process* (also known as the “Redbook”) in 1983 which established the four-step process that has become the dominant paradigm for risk assessment. Today risk assessment is the basis for most regulations and legislation concerning potentially hazardous substances.

Risk assessment integrates the disciplines of toxicology and exposure assessment in an attempt to understand and measure what types of adverse effects and harm a human might experience from exposure to a chemical or pollutant. Quantitative risk assessment (QRA) is a complex and sophisticated process that uses available scientific evidence as well as assumptions, mathematical modeling, and policy judgments in an attempt to estimate risk. Scientific techniques used to provide evidence include epidemiologic studies, laboratory studies involving whole animals or cultures of bacteria, cells or tissues, case studies of disease outbreaks, and long-term animal bioassays. The goal of QRA is to derive a quantitative or numerical value equating specific exposure conditions to the probability of the endpoint in question, with the endpoint being some form of adverse health effect such as asthma, cancer, or death. Although quantitative methods have been extensively used and are often appropriate, qualitative
approaches to risk assessment may be more appropriate and justifiable, given the inherent uncertainties in the underlying science. Ultimately, this process can then provide a tool that will aid risk managers in making decisions about potential hazards in the environment and human health.

The concept of risk assessment provides a basis for determining what we do know and what we don’t know, so that we can set bounds on the ranges of uncertainty. A methodologic approach has been proposed that can ensure consistency and complete analysis of different situations. There are four components traditionally included in the formalized risk assessment process: hazard identification and data collection, exposure assessment, toxicity assessment, and risk characterization (Figure 1). Applying these processes of risk assessment to pediatric environmental exposures is not such a straight-forward task. Most of the information presented is obtained from the EPA’s document Risk Assessment Guidance for Superfund: (RAGS) published in 1989. Incorporating all of the details and complexities of this process may not be applicable to the wide variability of scenarios that a health care provider or toxicologist may encounter in addressing a potentially toxic environmental exposure in a child. In addition, the large gaps in knowledge and data as well as tremendous amount of uncertainties involved in this process for adults becomes even further magnified when this process is applied to children. Assessing risks for children requires more information and understanding about both the unique susceptibility and exposures of children that may result in a final risk characterization that is quite different from one involving an adult. The following case scenario will help to illustrate the information obtained in a risk assessment.

**Case Scenario:** The parents of elementary school children in a small town are concerned about the presence of toxic wastes on the school playground and the pond adjacent to the school property. There was some recent information released in the media that 40 years ago the playground was a dumping site for surrounding chemical plants in the area. Many of the children have attention deficit hyperactivity disorder and asthma. In the last year, two children have been diagnosed with leukemia and one child with a brain tumor.

- What steps must be taken to assess the potential hazards on this playground, the school, and the school’s water supply?
- What limitations and assumptions must be acknowledged in performing a risk assessment which involves numerous children of various ages?
- What is the risk of adverse health effects to the children?
- Are the parents’ fears justified?
How should this information be communicated to the parents, school, and community?

Specially trained personnel in the health department and/or the EPA will carry out baseline risk assessments for circumstances similar to this scenario. Further consultation may be done with toxicologists and pediatricians to discuss specific aspects of the investigation. The goal of this section of the monograph is for pediatric health care providers to understand the general principles of QRA. Although they may not be intimately involved in the investigative processes, they should be able to explain to patients and parents the general process and general medical issues.

STEP 1 – HAZARD IDENTIFICATION / DATA COLLECTION

This process of hazard identification, data collection, and evaluation includes the identification of chemicals/substances that are health hazards based on toxicological and epidemiological evidence. This step may entail the gathering and analysis of pertinent historical data as well as site-specific information to identify potential chemicals/substances of concern. (Table 5) This process attempts to identify what substances or chemicals are present, their sources, pathways by which they contact the environment, and whether they have the ability to potentially produce adverse health effects. Specifically, site data include contaminant identities, contaminant concentrations in the key sources and media of interest (air, ground water, surface water, soil), characteristics of sources and characteristics of the environmental setting that may affect the fate, transport, and persistence of the contaminants. Detailed guidelines and specifications for sample collections and examples of modeling parameters for which information may need to be obtained are available. Based on a review of the existing data, the risk assessor should formulate a conceptual model of the site. This model should identify all potential or suspected sources of contamination, types and concentrations of contaminants detected at the site, potentially contaminated media, and potential exposure pathways, including receptors. (Table 6)
An example of this first step would be an investigation of a potentially hazardous waste site as noted in the case scenario. The pediatric health care provider or toxicologist would elicit the help of formally trained personnel from the health department or Environmental Protection Agency to complete this initial step. Investigation of the former chemical plants needs to be undertaken to identify types of chemicals expected on the playground. Then, a detailed investigation of appropriate media needs to be performed based on careful analysis of the potential routes of contaminant transport through the environment. This information is important to determine if the playground was purely a waste dumping site or whether contaminated groundwater or the flooding of a nearby potentially contaminated river may have contributed also.

Unfortunately, the basic health effects data for the majority of nonpesticidal industrial chemicals produced in the greatest quantities in the United States is not available.\textsuperscript{54} A study conducted by the Environmental Defense Fund indicated that the most basic toxicity testing results cannot be found in the public record for 71\% of the approximately 3000 high-production-volume nonpolymeric industrial chemicals in commercial use.\textsuperscript{54} Thus, there is no reliable basis to determine if these chemicals are safe or not. Forty-seven of the 100 high-production-volume chemicals sampled in this study are known to be emitted into air, land, and water in quantities of more than 10,000 pounds per year.\textsuperscript{54,116} However, toxicity data was available for less than half of these chemicals. In addition, almost 60\% of the 100 chemicals sampled which met the U.S. EPA criteria for bioaccumulation and persistence did not meet basic screening requirements for health hazard data.\textsuperscript{63} Even more disturbing is that even less toxicity information is known about the 80,000 chemicals on the U.S. EPA Inventory.

It is important for the PHCP to be familiar with some of the more common substances found on hazardous waste sites and their health effects. The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) requires the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA) to list, in order of priority, substances that are most commonly found at facilities on the National Priorities List (NPL) and which are determined to pose the most significant potential threat to human health
due to their known or suspected toxicity and potential for human exposure at these sites. Approximately 11 million people, of whom 25% to 35% are children 6 to 17 years of age, live within 1 mile of an NPL site. African Americans, Native Americans, and people of Hispanic origins comprise a greater proportion of these communities than those outside the waste site areas.

The top 20 substances and selected health effects are listed in Table 7. This list is not a compilation of “most toxic” substances, but rather a prioritization of substances based on a combination of their frequency, toxicity, and potential for human exposure at NPL sites. The objective of this priority list is to rank substances across all NPL hazardous waste sites to provide guidance in selecting which substances will be the subject of toxicological profiles prepared by the ATSDR. There are numerous information resources freely accessible to the public that provide general information on the known toxicity of numerous substances in the environment as well as results from assessments of previous hazardous sites. Some of these include the ATSDR Toxicological Profiles and Tox FAQ Sheets which can be accessed through the ATSDR HazDAT database. (See section on Information Resources for the Risk Assessment Process).

The National Priority List may not be purely applicable to the pediatric population. Special characteristics of children may place them at greater or lesser risk for toxicological effects. However, it is practical to assume that if these substances are high priority for adults, then they are more likely to be higher priority for infants and children.

**STEP 2 – EXPOSURE ASSESSMENT**

**General Information**

Exposure is defined as the contact of an organism (humans in the case of health risk assessment) with a chemical or physical agent. Exposure assessment involves the measurement or estimation of the amounts of substances to which people are exposed. It includes identification of exposed populations, analysis of contaminant releases, identification of potential exposure pathways, estimation of exposure concentration for pathways, and estimation of contaminant intakes for these pathways. The routes of exposure must be established,
whether they are oral, inhalation, dermal or any combination of these. Also, the magnitude, frequency, and duration of exposures must be determined. Exposure doses are then calculated to give estimates of chronic daily and lifetime exposures. The exposure assessment may contain three steps. (Figure 2).

The first step in evaluating exposure is to characterize the site with respect to its physical characteristics as well as those of the human population on and near the site. These site characteristics should include the climate, meteorology, geologic setting, vegetation, soil type, ground-water hydrology, and location and description of surface water. The population on or near the site should be analyzed with respect to their location relative to the site, particular activity patterns, and the presence of sensitive subgroups. For example, it is important to determine if the land under question is used for residential, recreational, school, or business/industrial purposes. Human activities and activity patterns associated with each particular land use should then be identified. The amount of time that the potentially exposed populations spend should be determined. This time may be quite variable for children. The amount of time that activities occur indoors, outdoors, or both, how activities change with the seasons, how access is restricted or limited, and any site-specific population characteristics that might influence exposure should be determined. For example, children are likely to play outdoors more in the summertime with less clothing, potentially increasing their risk for exposure. Finally, subpopulations that may be at increased risk from chemical exposures due to increased sensitivity and behavior patterns should be identified. Subpopulations which may be more sensitive include infants, children, adolescents, elderly people, pregnant and nursing women, and people with chronic illnesses. Thus, it is important to determine the locations of schools, day care centers, and residential areas with children. The characteristics that place children at increased risk for exposures will be described below.

The second step in the exposure assessment involves identification of exposure pathways. An exposure pathway generally consists of four components: 1.) a source and mechanisms of chemical release; 2.) a retention or transport medium; 3.) a point of potential human contact with the contaminated medium (i.e. exposure point); and 4.)
an exposure route (i.e. ingestion, inhalation, dermal exposure). See Figure 3. Details of exposure pathway analysis
are described elsewhere and meant to be a qualitative evaluation of pertinent site and chemical information.176

The final step in the assessment of exposure includes the quantification of the magnitude, frequency, and
duration of exposure for the populations and exposure pathways. First, exposure concentrations are estimated using
monitoring data and/or chemical transport and environmental fate models. Direct use of monitoring data is applicable
where exposure involves direct contact with the monitored medium (i.e. a residential drinking water well or public
water supply) and provides the best estimate of current exposure concentrations. Specific details and considerations in
estimating exposure concentrations in water, soil, air, and food are found in numerous resources by the EPA.181
Modeling may be used to estimate current concentrations in media for which there are no data as well as predict
future chemical concentrations in media in cases where the time span of the monitoring data is not adequate.
Superfund Exposure Assessment Manual (SEAM) and Exposure Assessment Methods Handbook provide
descriptions of some of the models available and guidance in selecting appropriate modeling techniques.172,173

The final step in exposure assessment involves the calculation of intakes for each exposure pathway identified.
Exposure estimates are expressed in terms of the mass of substance in contact with the body per unit body weight per
unit time (i.e. mg chemical per kg body weight per day, mg/kg-day). Chemical intakes are calculated using equations
which include variables for exposure concentration, contact rate, exposure frequency, exposure duration, body weight,
and exposure averaging time. (Table 8) The values of many of these variables depend on site conditions and the
characteristics of the potentially exposed population. Intake is not equivalent to an absorbed dose. The reasonable
maximum exposure or RME should then be estimated for each exposure pathway. The RME is the highest exposure
that is reasonably expected to occur at a site and will require both quantitative information and professional judgment.
Previously, exposures were estimated for an average and an upper-bound exposure case instead of a single exposure
case as described here. The limitation of the two case approach is that the upper-bound estimate of exposure may be
well above the range of possible exposures, whereas the average estimate is lower than exposures potentially incurred
by the population. Thus, the objective of the RME is to estimate a conservative exposure case that is still within the range of possible exposures. Specific methods for calculating intakes for water, soil, and air are described elsewhere. Sources of uncertainty are also analyzed before a final summary of the estimated intakes for each pathway is developed.

**Special Considerations of Exposure Assessment in Children**

Infants and children differ from adults, both qualitatively and quantitatively, in their exposures and susceptibilities. (Table 9) Assessing exposures in infants and children can be challenging because of their numerous and varied routes of exposure, sites of exposure, unique developmental behaviors and diets. To accurately assess children’s exposures, it is first necessary to define their unique environments, link their environments to their behaviors, and then characterize the differences in specific exposures. Children’s micro- and macroenvironments are different from adults and change through their development. Children have a variety of environmental exposure routes. They are at home and go to school, day care, play and recreational activities. Older children and children of farm workers are exposed at their homes and at their parents’ worksites. All must theoretically be accounted for to determine their maximum daily exposures. In addition, children’s environments may vary demographically and across cultural groups.

Exposures that occur to a mother prior to conception may also have significant health effects on an infant. For example, a woman who was inadequately treated for lead poisoning in childhood may give birth to an infant with congenital lead poisoning. This is probably due to mobilization of lead out of bone stores during pregnancy. Exposures to the fetus are usually dependent on the exposures to the mother. Another example is infants born to mothers who conceived after eating cooking contaminated with PCBs. These infants had yusho disease. The mechanisms felt to cause this disease in infants is storage of PCBs in maternal adipose tissue during exposure which are then mobilized during pregnancy.
Children can be exposed to chemicals through multiple sources including food, water, direct inhalation and contact with agents inside and outside the home. As discussed previously, some exposures are related to “take-home” exposures from parents’ and caregivers’ occupations. They may carry home chemical residues on their clothing or may expose infants to chemicals from the work place that subsequently appear in the mother’s breast milk. In addition, a child may be exposed from living or playing near a toxic waste site, and from air and water pollution through their living sites and recreational activities. Adolescents are exposed to the same work place chemicals as their adult counterparts. Thus, accounting for all of these potential avenues of exposure can be extremely challenging and complex.

The physical location of children changes with their development. The newborn’s exposures will be similar to the mother’s exposures due to the close and intimate contact between the two in the first months of life.13 The newborn and young infant spend more time in a single environment for prolonged periods of time, i.e. a crib, rather than several different environments. As a result, cumulative exposure to a single chemical/substance may result in more toxicity, i.e. leaded dust from renovations of an adjacent room or carbon monoxide from a faulty vent in the infant’s room. Premature infants will have different exposures than full-term infants due to their sometimes prolonged stays in the intensive care unit and nurseries. Environmental exposure including light, compressed gases, intravenous solutions, various indoor air pollutants unique to hospitals and benzyl alcohol (as a diluent in medications) will occur with much higher frequency to this cohort.24 Preambulatory infants also may sustain prolonged exposures to environmental toxicants because they cannot remove themselves from the environment.

Infants and toddlers frequently crawl on various floor surfaces such as carpet and linoleum inside or on the grass, dirt, or concrete outside. Therefore, they have much more exposure to chemicals associated with these different surfaces such as formaldehyde and volatile organic solvents from synthetic carpet and glues, or pesticide residues on the indoor floor or outside grass from insecticide sprays.20,57 School-age children spend a significant period of time at school which may expose them to both similar and different chemicals than those found at home. The risk of
asbestos and mold in schools have been previously discussed. Schools are frequently built on undesirable land for economic reasons. They may be on old industrial sites or waste sites where exposure to multiple different types of toxicants may occur as the case scenario presents. Adolescents begin to determine their own environments and may misjudge or ignore the risks to themselves. In addition, they may have jobs or activities that increase their risk to certain exposures as described previously.

The breathing zones for adults and children are different. While the breathing zone is typically four to six feet above the floor for an adult, it is much closer to the floor for a child and dependent on his/her height and mobility. Chemicals heavier than air such as mercury and large respirable particulates may settle out in this lower zone and radon may accumulate. This difference in breathing zones may have been responsible for a case of mercury poisoning due to latex house paint.

The metabolic rate of children is higher than adults. Hence their oxygen consumption is greater and subsequent exposure to any air pollutant is potentially greater. For example, if radon is present at 2 pCi/l, an adult with an average oxygen consumption rate of 3.5 ml/kg/body weight/min will receive an exposure of 48 pCi/kg in 24 hours. On the other hand, a 6-month old child with an average oxygen consumption rate of 7 ml/kg/bodyweight/min will receive twice the exposure or 96 pCi/kg in 24 hours.

The quantity and quality of food consumed in an infant and child is much different than an adult. During the first 6 months of life, infants drink 7 times as much water per pound of body weight. Thus, they may have a higher level of exposure than adults to the same level of toxic contaminants in drinking water. A striking example of this exposure difference is the finding of elevated blood lead levels in infants less than 1 year of age which is presumed to be due to exposure to tap water in formula. In addition, total body water comprises a higher percentage of their body weight and they also have a higher daily rate of water replacement. All three factors contribute to the increased exposure of infants to toxic contaminants in water compared with that of adults.
Children consume greater quantities and different types of food than adults. Caloric requirements are higher for children because of their higher surface-to-volume ratios and need to grow. Thus, the amount of food they consume per kilogram body weight is higher than that of the adult. Between the ages of 1 through 5 years, they eat 3 to 4 more times per pound than the average adult. The recommended diet for the first 6 months of life is limited to formula or breast milk. Breast milk has been documented to contain many environmental toxins including lead, PCBs, and dioxins. However, the benefits of breast milk are believed to outweigh the risks associated with the contaminants.

Children’s diets contain more milk products and certain fruits and vegetables than the typical adult diet. Although they consume far fewer types of foods than adults, infants and young children may consume much more of certain foods per unit of body weight than adults do. They eat more processed foods such as fruit juices, baby food, milk, and infants’ formula. For example, the average 1-year-old drinks 21 times more apple juice and 11 times more grape juice and eats 2 to 7 times more grapes, bananas, pears, carrots, and broccoli than the average adult. The foods most commonly eaten by infants are shown in Table 10 which were identified by the National Academy of Science in their assessment of children’s diets. In the NRC’s report on pesticides in the diets of infants and children, they concluded that differences in diet and thus in dietary exposure to pesticide residues accounted for most of the difference in pesticide-related health risk and were a more important source of differences in risk than were age-related differences in toxicologic vulnerability.

Normal behavior development of children will also influence the type and extent of environmental exposures. A nonambulatory infant or child will not be able to remove himself from a toxic environment. During the toddler years, children have a normal developmental stage of intense oral exploratory behavior. These behaviors may persist in older children with developmental delay, autism, or other behavioral problems. Hand-to-mouth behavior, like sucking on thumbs and fingers, increases children’s ingestions of toxicants in dust, soil, and water. This behavior is an important etiology of lead poisoning in children who have significant concentrations of lead dust in their
environments. Furthermore, residues that persist on carpets, floors, furniture, grass, and other objects may all be sources of toxic exposure for children due to their hand-to-surface followed by hand-to-mouth activity. Children may also place their mouths on various surfaces. Wood used in some playground equipment may be treated with arsenic and creosote, thus increasing the risk of poisoning to those children who place their mouths on this equipment. Curious children may be attracted to used waste drums, mud puddles, piles of dirt, and empty lots which may contain dangerous and toxic chemicals. Older children and adolescents who are gaining more independence may place themselves in situations with greater risk. They may not consider immediate cause and effect of certain situations that place them at increased risk of toxic exposures.\textsuperscript{13}

\textit{External Exposure Monitoring in Children}

Exposure assessment can be conducted through direct and/or indirect approaches. Direct measurements of exposure may be obtained in external environmental media or through the determination of toxicants or their metabolites in a biologic medium (biologic monitoring). The latter will be described in a later section of the monograph (See Using Biologic Markers in the Pediatric Risk Assessment Process). Traditionally, exposure assessment has relied on external or ambient exposure monitoring of airborne toxicants.\textsuperscript{176,193} This process involves measuring a chemical either by area sampling with a monitor in a fixed location, or by personal monitoring in which small pumps are worn by the monitored participants. This process works well for chemicals in which reference levels are available for comparison, but has its limitations in monitoring exposure in children. This monitoring may not be representative of total exposure if wide variation in exposure occurs as described above. Furthermore, monitoring may underestimate total exposure and intake doses because airborne exposure assessment measures only one route of exposure. Ingestion, inhalation, and dermal exposure of toxicants are important routes of exposure in children for the reasons described above. Finally, external monitoring indicates only the current level of chemical present in the environment which does not always predict the internal dose of chemical. Differences in absorption at similar exposure levels may occur in children because of their increased respiratory rate, and larger surface-to-volume ratio
than adults as described later (See Special Considerations in Assessing Toxicity Due to the Biologic Environments of Children).

**STEP 3 - TOXICITY ASSESSMENT**

Toxicity assessment is the process of quantitatively evaluating the toxicity information and characterizing the relationship between the dose of the contaminant received and the incidence of adverse health effects in the exposed population. This step is also referred to as dose-response evaluation. Toxicity values such as slope factors and reference doses are derived from the quantitative dose-response relationship to be used to estimate the incidence of potential for adverse effects as a function of human exposure to the agent.\(^\text{166}\) Toxicity assessments are performed for carcinogenic and noncarcinogenic effects, each category using its own toxicity values and operational schemata.\(^\text{142}\) Information sources considered in toxicity assessment include controlled epidemiologic investigations, clinical studies, and experimental animal studies. Unfortunately there are large deficiencies in these informational sources, especially with regards to children, making this step of the risk assessment process extremely challenging.

Toxicity assessment for noncarcinogenic effects have utilized a “threshold” model approach. This model assumes that there is a threshold dose below which no toxic effects occur. The toxicity values used most often in evaluating noncarcinogenic effects are the acceptable daily intake or ADI and the reference dose or RfD.

The ADI is defined as the amount of a toxic agent in milligrams per kilogram of body weight per day which is not expected to result in an adverse effect after chronic exposure in the general population of humans, including sensitive subgroups.\(^\text{201}\) The RfD is similar in concept to the ADI but is derived using a strictly defined methodology.\(^\text{180}\) It is defined as an estimate (with uncertainty spanning an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of adverse effects during a lifetime.\(^\text{176}\) Different RfDs are defined based on the duration of exposure. A chronic RfD should generally be used to evaluate the potential noncarcinogenic effects associated with exposure
periods between 7 years and a lifetime. They are specifically developed to be protective for long-term exposure to a
compound. Subchronic RfDs (RfD<sub>s</sub>) should be used to evaluate exposure periods between two weeks and seven
years. Developmental RfDs (RfD<sub>d</sub>) are used to evaluate the potential effects on a developing organism following a
single exposure event.

A RfD can be calculated in the absence of a known value by dividing an experimentally derived safe exposure
level by an uncertainty or safety factor. (Table 11) This safe value, the No Observed Adverse Effect Level or
NOAEL, is available from experimental data on the chemical and most closely approximates a level or concentration
at or below which no toxic effects were detected. Uncertainty or safety factors are established by the type of data
available for the evaluation and generally consist of multiples of 10 (Table 12). These factors make allowances for
uncertainties or knowledge deficiencies in the available data. Thus, RfD = NOAEL / Uncertainty Factor. (Table 10)
As a result of the 1996 Food Quality Protection Act, an additional 10-fold safety factor must be added to account for
prenatal or postnatal developmental toxicity. For nonthreshold effects, the U.S. EPA adds a 100-fold uncertainty
factor where prenatal/postnatal toxicity and exposure data for children are such that the risks for children have not
been well defined and may be greater than the risks for adults.63 (See New Approaches to Risk Assessment in
Children).

When a site-specific risk assessment is done, established toxicity values may be compared with the site-
specific exposure doses (ED) that the population at risk is expected to incur where exposure doses are calculated as
chronic daily intakes (CDIs). A noncancer hazard quotient is calculated which divides the exposure dose by the
reference dose (ED/RfD). (Table 11) This hazard quotient assumes that there is a level of exposure (i.e. RfD) below
which it is unlikely for even sensitive populations to experience adverse health effects. When the RfD is less than the
ED, the hazard quotient has a value greater than 1, and the risk associated with that exposure is assumed to be
significant.174,176 As a rule, the greater the value of E/RfD above unity, the greater the level of concern. However,
these ratios should not be interpreted as statistical probabilities. Furthermore, it is important to understand that the
level of concern does not increase linearly as the RfD is approached or exceeded because different RfDs do not have equal accuracy or precision.

The ATSDR developed Minimal Risk Levels (MRLs) as an initial response to a mandate defined by the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA). This agency chose to adopt a practice similar to that of the EPA’s RfD for deriving substance-specific health guidance levels for noncarcinogenic endpoints. An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specific duration of exposure. The ATSDR uses the No-Observed-Adverse-Effect-Level / Uncertainty Factors to derive MRLs for hazardous substances. They are set below levels that, based on current information, might cause adverse health effects in the people most sensitive to such substance-induced effects. MRLs are derived for acute (1-14 days), intermediate (15-364 days), and chronic (365 days and longer) exposure durations. Currently, MRLs have been derived for the inhalation routes of exposure but not for the dermal route of exposure.

MRLs are generally based on the most sensitive substance-induced end point considered to be of relevance to humans. Exposure to a level above a MRL does not mean that adverse health effects will occur. MRLs, like RfDs, are often based on animal studies because relevant human studies are lacking. Because the ATSDR assumes that humans are more sensitive than animals to the effects of a hazardous substance and that certain persons may be particular sensitive, the resulting MRL may be as much as hundredfold below levels shown to be nontoxic in laboratory animals. These substance-specific estimates, which are intended to serve as screening levels, are used by the ATSDR health assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. MRLs are not intended to define clean-up or action levels for the ATSDR or other agencies.

Assessing toxicity for carcinogens have utilized a “nonthreshold” methodology because it is believed that there is no level of exposure to a chemical that does not pose a risk, i.e. no dose of a chemical is risk-free. Risks are
estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen (i.e. incremental or excess individual lifetime cancer risk). The guidelines presented here are consistent with EPA’s Guidelines for Carcinogen Risk Assessment. A two-part evaluation has been used to assess carcinogenic effects—a weight-of-evidence calculation and slope factor calculation.

Available data are first evaluated to determine the likelihood that the chemical is a human carcinogen. Evidence is characterized, separated for human studies and animal studies, as sufficient, limited, inadequate, no data, or evidence of no effect. Based on the extent to which the chemical has been shown to be a carcinogen in animals or humans or both, it is given a provisional weight-of-evidence classification (Table 13). The slope factor is the toxicity value used for this particular modeling which defines quantitatively the relationship between dose and response. It is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime, i.e. the probability of an individual developing cancer as a result of exposure to a particular level of a particular carcinogen. Slope factors should always be accompanied by weight-of-evidence classification to indicate the strength of evidence that the agent is a human carcinogen.

One limitation of the dose-response methodology is extrapolating toxicity assessment to lower doses. Generally, the slope factor is derived from data that contain high exposure doses. Evaluating risk at lower exposure levels is much more difficult. Numerous complex mathematical models have been developed to extrapolate effects of carcinogens seen at high doses to effects expected at low doses. Furthermore, different extrapolation methods may result in large differences in the projected risk at low doses. EPA guidelines suggest that low-dose linearity models are preferred when limited information is available. After the data are fit to an appropriate model, the upper 95th percent confidence limit of the slope of the dose-response curve is calculated which is known as the slope factor. Thus, there is only a 5% chance that the probability of a response could be greater than the estimated value on the basis of the data and model used. It is usually expressed as risk per mg/kg-day. Slope factors are then verified by
the Carcinogen Risk Assessment Verification Endeavor (CRAVE) Workgroup of the EPA and review summaries are included in the IRIS database. (See Information Resources for the Risk Assessment Process)

A linear low-dose cancer risk equation may be computed to assess risk where exposures are low—Risk = CDI x SF. (Table 14) This equation assumes that the slope factor is a constant, the dose-response relationship will be linear in the low-dose portion of the multistage model curve, and risk will be directly related to intake. A one-hit equation may be calculated where chemical intakes might be high (i.e. risk above 0.01) (Table 14). Toxic exposures that result in cancer have been analyzed by mathematical modeling to derive levels at which an increase in the incidence of cancer is at an acceptable level. This level has been established at 1 excess cancer death in 1,000,000 lifetime exposures (1 x 10^{-6}). Under site-specific circumstances, EPA guidance allows a risk range between 10^{-4} and 10^{-7}.58,165,201

There are numerous uncertainties associated with calculated toxicity values. These include: using dose-response information from effects observed at high doses to predict the adverse health effects that may occur following low-level exposures; using dose-response information from short-term exposure studies to predict the effects of long-term exposures and vice-versa; using dose-response information from animal studies to predict effects in humans; and using dose-response information from homogeneous animal populations or healthy human populations to predict the effect likely to be observed in the general population consisting of individuals with a wide range of sensitivities.37,39,72

**Special Considerations in Assessing Toxicity Due to the Biologic Environments of Children**

Quantitative differences in toxicity between children and adults are due, in part, to age-related differences in both pharmacokinetic and pharmacodynamic processes. The biologic environments of children are different from adults and vary with their developmental stages. (Table 15) The biologic environment consists of the internal physiologic interactions of the body with chemicals including absorption, distribution, metabolism, excretion, and
target organ susceptibility. Differences in size, immaturity of biochemical and physiological functions in body systems, and variation in body composition (water, fat, protein, and mineral content) all can influence the extent of toxicity.

The toxicokinetics of absorption result in increased toxicity of many substances. Absorption generally occurs by four major pathways: transplacental, percutaneous, respiratory, and gastrointestinal. Several classes of compounds readily cross the placenta, including those with low-molecular weight, those that are fat-soluble, and other specific elements. Even low levels of carbon monoxide exposure in a pregnant female may be detrimental to the fetus. Experimental models and clinical reports have demonstrated the widespread toxic effects of CO on the fetus exposed during all stages of gestation. These include teratogenicity, neurological dysfunction, decreased birth weight, and increased fetal death.\textsuperscript{60,93,112} The fetus is more susceptible to CO poisoning than its mother for two reasons.\textsuperscript{101} First, fetal COHb levels at equilibrium are 10 – 15\% higher than maternal levels and elimination time of fetal COHb is longer. Second, the fetal oxyhemoglobin dissociation curve is normally shifted to the left and the PO2 is normally low. After acute CO exposure, the curve will shift further to the left, exaggerating the effects of decreases in fetal oxygen content, resulting in increased hypoxia to the fetus. Carbon monoxide has a higher affinity for fetal hemoglobin compared to adult hemoglobin; thus, the concentration of CO is higher in the fetus compared to the mother.\textsuperscript{188}

Lipophilic compounds such as polycyclic aromatic hydrocarbons which are found in cigarette smoke, PCBs, and methyl mercury may readily cross the placenta and affect the fetus.\textsuperscript{31} Specific calcium-dependent transport mechanisms in the placenta actively transport metals such as lead into the fetal circulation. Fetal blood lead concentrations have been reported to be equivalent to maternal blood lead concentrations.\textsuperscript{68}

Percutaneous absorption of chemicals is particularly important for lipophilic compounds. Although chemicals such as nicotine and cotinine have been described in amniotic fluid, their absorption through fetal skin, which is unkeratinized, has not been studied.\textsuperscript{186} The newborn is most susceptible to absorption of various chemicals because
the skin does not start to develop keratin until 3 to 5 days after birth. Toxic epidemics involving absorption of chemicals through the skin in newborns include hypothyroidism from iodine in Betadine scrub solution, neurotoxicity from hexachlorophene, and hyperbilirubinemia from a phenolic disinfectant used to clean hospital equipment. Newborns also have a larger surface-to-body mass ratio compared with older children and adults. The newborn’s surface to mass ratio is three times larger than an adults and an older child’s surface to mass ratio is two times larger. Thus, there is the potential for a chemical to have 2-3 times more absorption in this population compared to an adult.

In the fetus, some chemicals in the amniotic fluid may come in contact with the lining of the respiratory tract. However, studies on this pathway of exposure are limited. Although the surface absorptive properties of the lung do not probably change during development, the alveoli and capillaries of the lung continue to proliferate until 5 to 8 years of age. The alveolar surface area increases from approximately 3 m² at birth to about 75 m² at adulthood. Thus, the air-tissue gas exchange area increases more than 20-fold from infancy to adulthood. In addition, there are quantitative differences in respiratory minute ventilation between children and adults as discussed previously. Thus, for inhalation exposures to equivalent environmental chemical air concentrations, both indoor and outdoor, infants and children are at least at the same or possibly greater risk for exposure.

The gastrointestinal tract undergoes numerous developmental changes, affecting the absorption of chemicals and drugs. Following birth, stomach acid secretion is low, but achieves adult levels by several months of age, which may markedly affect absorption of certain chemicals from the stomach by changing the ionization status of these chemicals. If acidity levels are too low, bacterial overgrowth in the small bowel and stomach may result in the formation of chemicals that can be absorbed. This phenomenon is well demonstrated by the acquisition of methemoglobinemia in infants whose formula was reconstituted with nitrate-containing well water. The nitrates were converted to nitrites by intestinal bacteria. Different chemicals may undergo differential transport across the
small intestine. Toddlers absorb a significantly higher percentage of ingested lead than adults—a 1 to 2-year-old child will absorb 50% of ingested lead compared to an adult who will absorb only 10%. Distribution of environmental chemicals within the body can be important in the production of toxicity in children. The tissue distribution of chemicals within the body varies with the child’s developmental stage. Lead accumulates more rapidly in children’s bones than adult’s bones. Also, animal models have shown that lead is retained to a larger degree in the infant animal brain than in the adult brain. A child’s ability to metabolize and detoxify many toxicants is different from that of an adult. Enzyme activity involved in metabolic reactions are subject to developmental changes as well as genetic polymorphisms. The cytochrome P450 enzyme system undergoes complex developmental changes. There is great interindividual variability in the amounts and types of metabolizing enzymes, some of which are under direct genetic control. Phase I (oxidation) and Phase II (conjugation) metabolism varies considerably by age and by individual chemicals and drugs.

A number of metabolic pathways responsible for bioactivation or detoxification are not present in the fetus or are less developed in the infant. For example, glucuronic acid conjugation is significantly depressed at birth, although a well-developed capability for sulfate conjugations exists. Thus, some children may be genetically more susceptible to adverse effects from certain exposures. Smokers with lung cancer have a higher incidence of lacking a specific glutathione transferase and thus have a genetic susceptibility to carcinogenesis from cigarette smoke. Children with glucose-6-phosphate dehydrogenase (G-6PD) deficiency are at risk for hemolytic anemia if exposed to certain chemicals such as naphthalene. Oxidation and detoxification metabolic processes have implications for children for risk of chemical carcinogenesis from those chemicals which undergo metabolic activation to a reactive intermediate.
Kidney function is also developmentally regulated. In the newborn, renal clearance is low for a variety of chemicals. Glomerular filtration rate is low at birth and gradually increases to adult values by approximately 1 year of age. By 16 months of age, renal function has reached adult capabilities.

As detailed below, children’s organs are undergoing growth and differentiation, both of which may be affected by exposure to environmental pollutants. Many organ systems in the fetus and the young child, particularly the central nervous system, lungs, immune system, and reproductive organs, undergo extensive growth and development throughout pregnancy and in the first months and years of extrauterine life. During this period, vital structures and differentiation of organ function develop. Furthermore, these organ systems are more susceptible to damage because they lack adequate repair mechanisms. Thus, there is high risk that the resulting dysfunction and damage in the developing infant and child will be permanent and irreversible.

Brain growth in children occurs rapidly for the first two years of life, with approximately 75% of the total number of brain cells of all types present by 2 years of age.\textsuperscript{154} Subsequent growth continues more slowly until full brain cell numbers are reached at adulthood. Additional brain growth is due to myelination of subcortical white matter, elaboration of neuronal dendrites and axons, and an increase in glial cells. As a result, the infant and child human brain represents a relatively larger portion of body mass compared to the adult human brain. The brain’s high lipid composition and relatively greater mass have implications for steady-state distribution and body storage of lipophilic environmental chemicals.

Differentiation occurs when cells take on specific tasks within the body and lose the ability to divide. It may be triggered by certain hormonal interactions. The endocrine system that regulates many functions in the body including growth, sexual maturation, and homeostasis, may be an important target for environmental toxicants. Some environmental agents may mimic or block the action of hormones and thus alter the differentiation of some tissues. Environmental contaminants have been associated with abnormal organ development and referred to as environmental endocrine disruptors (EEDs). In the early 1990s, vinclozolin, a fungicide for fruits and vegetables, was
reported to be associated with feminization of the male fetus in animal studies, acting as an antiandrogen.61,69,90,91

DDE, a metabolite of the insecticide DDT, was found to be associated with antiandrogen effects and abnormal sexual development in developing alligators.77 In addition, DDT has been associated with estrogen effects. Many endocrine disruptors like DDT/DDE bioaccumulate or concentrate in the food chain; thus, having the potential to expose a wide age range of people including the developing fetus and rapidly growing infants and children. Recent studies have demonstrated the negative effects of chlordecone, an organochlorine pesticide, on the reproductive system.184

The embryo, fetus, and young child appear to be particularly susceptible to adverse consequences following early exposure to endocrine disruptors.100 Early exposure to endocrine-disrupting chemicals may interfere with reproductive development. They may be responsible, in part, for increases in the incidence of testicular cancer, a recently reported doubling in the incidence of hypospadias in the United States, and for the increasingly early onset of puberty in young girls.50,76,127

Cell migration is necessary for certain cells to reach their destination for particular functions. Neurons in the central nervous system originate in the germinal matrix, then migrate to a predestined location in one of the many layers of the brain.13 An example of substances affecting cell migration is alcohol. Prenatal exposure to ethanol may result in the interruption of cell migration and cause lissencephaly and fetal alcohol syndrome.36,108

Synaptogenesis occurs rapidly in the first 2 years of life. Dendritic trimming is the active removal of synapses to allow more specificity of the resulting neuronal network. There is also age-related neurotransmitter development which has implications for sensitivity to those environmental chemicals that might alter neurotransmitter brain function. Data suggest that lead may interfere with all these processes.67

The brain and the lungs have prolonged periods of postnatal development, increasing the vulnerability of these organs to multiple toxic exposures. Lead affects the brain and central nervous system significantly more in the child than in the adult. This difference is the basis for the large differences of blood lead concentration as cause for concern. Several studies have demonstrated that blood lead levels as low as 10 mcg/dl may result in significant
decreases in neurobehavioral development in children.\textsuperscript{9,16,17,30} Recent data have suggested that this threshold for neurocognitive deficiencies may occur at blood lead concentrations lower than 5 mcg/dL.\textsuperscript{97} In contrast, limits of blood lead concentrations for adult occupational exposures are usually around 60 µg/dL. Thus, if cells in the developing brain are destroyed by chemicals such as lead, mercury, or solvents, there is a high risk that the resulting neurobehavioral dysfunction will be permanent and irreversible.

The developing lung may also be more susceptible to various components of indoor and outdoor pollution, thus placing children at higher risk than adults to the adverse effects of air pollution.\textsuperscript{189} Numerous studies have documented the toxic effects of air pollutants on otherwise healthy children.\textsuperscript{47,147,202} Several studies report that FEV\textsubscript{1} and lung growth rate in children ages 6 to 10 years is reduced after exposure to cigarette smoke.\textsuperscript{18,160,191} Neither the nervous system nor the lungs are well able to repair any structural damage that is caused by environmental toxicants. Recent data show that 17 out of every 100 nonsmoking adults who develop lung cancer had significant exposure to indoor cigarette smoke as a child.\textsuperscript{89}

The immune system can have long-lasting alterations after perinatal exposure to environmental toxicants. Besides changing the response to infection, such alterations can also affect the regulation of the development of other systems such as the nervous and reproductive systems.\textsuperscript{21}

Because most children have more future years of life than adults, they have more time to develop chronic diseases triggered by early environmental exposures. Many diseases caused by environmental toxicants develop decades after the exposure. Examples of this latency effect include lung cancer and malignant mesothelioma caused by exposure to asbestos; leukemia caused by benzene; breast cancer that may be associated with DDT exposure; and Parkinson’s disease that might be caused by exposures to environmental neurotoxicants.\textsuperscript{52,118} Many of these diseases are probably the result of multistage processes within the body’s cells that require many years to progress from earliest initiation to actual manifestation of illness. Consequently, certain toxic exposures encountered early in life may be more likely to lead to disease than the same exposures encountered later in life.
Children’s Special Susceptibilities to Cancer in Risk Assessment

It has been estimated that about 80 to 90% of all cancers are attributable to environmental factors interacting with both genetic and acquired susceptibilities.\textsuperscript{130} For infants and children, the proportion of cancers attributable to environmental factors is probably lower than in adults, although this is not well known. During the last two decades, there has been relatively little change in the incidence of children diagnosed with all forms of cancer. However, childhood leukemias appeared to increase in incidence in the early 1980s. Also the overall incidence of childhood brain tumors rose from 1973 through 1995.\textsuperscript{113} Children may be at an increased risk for developing cancer for numerous reasons. These include increased absorption and retention of environmental agents, reduced detoxification and repair systems during the early stages of development, a higher rate of cell proliferation, and the fact that cancers initiated in the prenatal period and early years of infancy have the opportunity to develop over many decades.\textsuperscript{130}

Children’s susceptibility to certain carcinogens may be influenced by the presence and activity of specific metabolizing enzymes. The absence of or lower activity of enzymes that are required to metabolize carcinogens to their active form can reduce a child’s risk of cancer following exposure.\textsuperscript{32} On the other hand, decreased amounts or activity of enzymes that detoxify carcinogens can increase cancer risk. As discussed previously, there is interindividual variability and genetic susceptibility in the amounts and types of metabolizing enzymes.\textsuperscript{43,59,105} The CYP1A1 enzyme (one of the P450 enzyme subtypes), whose product metabolizes polycyclic aromatic hydrocarbons (PAH) such as benzo[a]pyrene, is a polymorphic enzyme.\textsuperscript{32} Approximately 10% of the Caucasian population has a highly inducible form of the enzyme which has been associated with increased lung cancer in smokers.\textsuperscript{130} PAH-DNA adducts are also elevated in the umbilical cord blood and placenta of newborns with the CYP1A1 MDP1 polymorphisms, suggesting that the genetic polymorphism may increase the risk from transplacental PAH exposure.\textsuperscript{32}

The rapid cell proliferation seen in growing children may contribute to an increased risk of cancer in this population. Polycyclic aromatic hydrocarbons and aflatoxin B\textsubscript{1} produce liver tumors when administered to newborn rodents but not when administered to older animals.\textsuperscript{32,114} It is presumed that the liver is proliferating much more
rapidly during the newborn period, thereby increasing its susceptibility. Another example is the increased risk of radiation-induced breast cancer in women who were in their teens at the time of the atomic bombings.\textsuperscript{164}

DNA repair mechanisms also play an important role in cancer protection.\textsuperscript{32} An example is the association of mutations of the p53 genes and tobacco smoking. The p53 gene encodes a protein that modulates DNA repair and cell division.\textsuperscript{84,145,159} Mutations of the p53 genes are involved in at least 50\% of all cancers.\textsuperscript{12} Thus, it is plausible that p53 mutations may occur in the offspring of smoking mothers, making these children more susceptible to certain types of cancer.

The proliferation and terminal differentiation of different tissues make them particularly susceptible to carcinogenesis. This increased susceptibility is due to the shortened time period for DNA repair and the multiple changes that are occurring within the DNA during cell growth.\textsuperscript{13} A clinical example of this process is the epidemic of scrotal cancer among the pubertal chimney sweeps of Victorian England.\textsuperscript{35} These chimney sweeps were usually adolescents with developing secondary sexual characteristics. It is hypothesized that the scrotum, while undergoing terminal differentiation, had increased susceptibility to the carcinogens in the soot, as this was an uncommon site for tumors except in this particular population.

**RISK CHARACTERIZATION**

Risk characterization involves the synthesis of information from the previous three steps of QRA and characterizes the potential for adverse health effects to occur. It provides a summary of the toxicity and exposure assessment and may provide a quantitative and qualitative expression of risk. (Figure 5) Major assumptions, scientific judgments, and estimates of the uncertainties involved in the assessment process are also presented. Risk characterization serves as the bridge between risk assessment and risk management. It provides important information for the risk manager to consider along with other economic, technical feasibility, and regulatory factors which are important for the decision-making process.
The target value of the risk characterization step is determined by the goal of the risk assessment. This risk may be presented in several different ways depending on the particular assessment: maximum lifetime cancer risk, population cancer risks, distribution of individual risks, and noncancer risks based on a health reference dose (RfD). For example, if the chemical being analyzed is a carcinogen, then the target is a cancer risk value. If the chemical causes noncarcinogenic effects, the target value may be a determination as to whether an exposure will result in a significant likelihood of an adverse toxic health effect which may be expressed as a hazard quotient. Both carcinogenic and noncarcinogenic effects can occur with the same substance (i.e. benzene or arsenic); thus, these effects must be separately calculated and presented.

The first step is to review and compare the results of the exposure assessment (intakes for all exposure pathways and land-uses and for all relevant substances) and toxicity assessment (toxicity values for all exposure routes and relevant substances). The consistency and validity of three assumptions, in particular, common to the exposure outputs and toxicity outputs for each contaminant and exposure pathway of concern must be checked. First, the averaging period for exposure must be consistent for each substance. For example, if the toxicity value is based on average lifetime exposure (e.g., slope factors), then the exposure duration must also be expressed in those terms. For estimating cancer risks, average lifetime exposures should always be used. If noncarcinogenic effects of less-than-lifetime exposures are being evaluated, chronic RfDs cannot be compared to short-term exposure estimates nor can short-term exposures be converted to equivalent lifetime values for comparison to chronic RfDs. Second, all toxicity values used for each exposure pathway being evaluated should be consistent with the route of exposure (e.g., oral toxicity values compared to oral exposure pathway). Extrapolating between exposure routes for some substances that only produce effects dependent upon the specific route of exposure is not appropriate. For example, a toxicity value based on a localized lung tumor that results only from inhalation exposure would not be appropriate for estimating risks associated with dermal exposure. Currently, the EPA considers it appropriate only to extrapolate dermal toxicity values from values derived for oral exposure. Finally, the exposure estimates and toxicity values
should be expressed both as absorbed doses or both expressed as intakes. Adjustments made for dermal exposures, absorbed-dose toxicity values, and medium of exposure are detailed elsewhere.  

The second step in risk characterization is quantifying risk or hazard indices for both carcinogenic and noncarcinogenic effects of each substance and each exposure pathway being analyzed. Specific details regarding risk quantification for individual substances are discussed in the section on toxicity assessment. Probabilistic analyses are performed for estimating the risk of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen (i.e. the incremental or excess individual lifetime cancer risk). The linear low-dose cancer risk equation and the one-hit equation are used depending on whether the risk level is high or low (Table 14). For noncarcinogenic effects, a probabilistic approach is not used for estimating risk. Instead, an exposure level over a specified time period is compared with a reference dose derived for a similar exposure period—a hazard quotient. Separate consideration is needed for the three exposure duration periods—chronic, subchronic, and shorter-term exposure—as defined previously.

Guidelines for assessing potential health effects of multiple chemicals have been outlined. A simple additive equation for cancer risk is appropriate for most risk assessments where the total cancer risk is the sum of the separate risk estimates for each substance. Resulting cancer risk estimates should be expressed using one significant figure only (i.e. it should not exceed 1). Similarly, to assess the total potential for noncarcinogenic effects of more than one chemical, a hazard index (HI) approach has been developed. (Table 11) The hazard index is equal to the sum of the hazard quotients. When the HI exceeds unity, potential health effects may occur. A hazard index should be calculated separately for chronic, subchronic and shorter-term exposure periods.

There are several limitations to these rather simple additive approaches in assessing overall risk. For carcinogenic effects, substances with different weights of evidence for human carcinogenicity are included. The cancer risk equations sum all carcinogens equally, although their weight of evidence is not necessarily equal. The total cancer risk estimate might become artificially more conservative because the probability distributions for each
substance are not strictly additive. Slope factors from animal data are given the same weight as slope factors derived from human data. Finally, the action of each substance being analyzed may not be exclusively independent. Possible interactions between different carcinogens will affect the validity and reliability of the quantitative risk assessment.

There are many limitations to the additive summary for noncarcinogenetic analysis. The level of concern for potential harmful effects associated with noncarcinogenic substances does not increase linearly as the reference dose is approached or exceeded. This is because the RfDs do not have equal accuracy or precision and are not based on the same severity of effect. In addition, hazard quotients are combined for substances with different RfDs which are based on critical effects of varying toxicological significance. Also, multiple RfDs will include different uncertainty adjustments and modifying factors, making the combinations of these varying levels of confidence not totally exact. Applying the hazard index equation to various compounds that do not induce the same type of effects or act by the same mechanism could overestimate the potential for effects. It may be necessary to segregate hazard indices by these two parameters.

Finally, the risk characterization process should consider comparison of the current risk assessment with other health assessment and site-specific human studies that might be available. The baseline risk assessment should be compared to the ATSDR health assessments if available. The baseline risk assessment should be compared to the ATSDR health assessments if available. (See section on Information Resources for the Risk Assessment Process) If there are differences in the qualitative conclusions of these health assessments and the quantitative conclusions of the baseline risk assessment, these differences and their implications should be discussed. It is also important to include any epidemiological or health studies for populations exposed from the site if they are available. Specific areas of agreement and disagreement between these health studies and the risk assessment should be described as well as factors that could account for the discrepancies.

Risk assessment information should be summarized and the discussion should include the key elements described above as well as a level of confidence in the toxicity information and exposure estimates and the major factors reducing the certainty in the results and the significance of these uncertainties. It is the responsibility of the
risk assessment team to develop conclusions about the magnitude and kinds of risk at the site and the major uncertainties affecting the risk estimates. In addition, a summary of the cancer risks and noncancer hazard indices should be presented and accompanied by explanatory test. An example of a risk characterization is shown in Table 16. The discussion should summarize both the qualitative and the quantitative findings of cancer risks and noncancer hazards. These summaries should also be properly qualified by discussion of major assumptions and uncertainties in the assessment process.

Uncertainties and Weaknesses of the Risk Assessment Process

Uncertainties associated with site risk assessments need to be explained in the risk characterization analysis. The risk measures used in site risk assessment usually are not full probabilistic estimates of risk, but conditional estimates given a considerable number of assumptions about exposure and toxicity. Thus, it is important to fully delineate the assumptions and uncertainties inherent in the risk assessment to put the risk estimates into proper perspective. Highly quantitative statistical uncertainty analysis is usually not practical or necessary for site risk assessments. Uncertainty about the numerical results in all environmental risk assessment is large, generally on the range of at least an order of magnitude or greater. Sometimes a qualitative approach is the most practical approach to describing uncertainty given the use of the information.

Several categories of uncertainties are associated with risk assessments. These include: 1.) definition of the physical setting; 2.) dose-response model applicability and assumptions; 3.) transport, fate, and exposure parameter values; and 4.) tracking uncertainty-how uncertainties are magnified through the various steps of the assessment process. In addition, the initial selection of substances used to characterize exposures and risk on the basis of the sampling data, the toxicity values for each substance, and the exposure assessment for individual substances and exposures contribute to the uncertainties. The basis for these uncertainties is usually the chemical monitoring data and the models used to estimate exposure concentrations in the absence of monitoring. Additional uncertainties
must be incorporated into the risk assessment when exposure to several substances across multiple exposure pathways is summed as discussed above.

The weaknesses associated with the risk assessment process stem from these sources of uncertainties and will be further discussed. (Table 17) Extrapolation of data may occur in several manners and use many assumptions that introduce uncertainties into the final risk value. These include extrapolation of data from animals to humans, from a high-dose region on a dose-response curve to a low-dose region, from one route of exposure in test animals to another route of exposure in humans, or from continuous, static exposure conditions in the laboratory to uneven, discontinuous human exposures.39,72

Various studies and techniques may weaken the risk assessment process and their limitations should be acknowledged. Studies may be used inappropriately to provide data that is not directly transferrable to risk assessment processes.29 These toxicity studies may not have been designed to provide risk assessment data. Epidemiologic studies may have limited reliability because of their retroactive nature, inability to detect small differences in effects, and limitations in recovering adequate exposure data and disease incidence.129,141,145

Risk assessment techniques may not account for the different mechanistic classifications of carcinogens. DNA-reactive carcinogens that react with genetic material are genotoxic and those that act without evidence of a direct interaction with genetic material are epigenetic. This distinction may have substantial significance in risk assessment.157 Genotoxic carcinogens may cause tumors at a very low level without a threshold value while epigenetic carcinogens must reach some threshold levels before tumorigeneis, thus having some safe low level of exposure. As a result, human risk may not be the same for different types of carcinogens.

The risk assessment process makes conclusions about risks from processes and modeling techniques based on populations.8 Averages and standardized values applicable only to general populations are examples of assumptions that are used to characterize exposures to populations. These assumptions are sources of uncertainty that arise in exposure assessments. Data from laboratory studies done in animals that lack genetic heterogeneity may not be
applicable to a genetically heterogeneous human population. Using this data to estimate and characterize risk will not accurately reflect risk imposed on susceptible subgroups of the population like children.

There are weaknesses associated with assessing toxicity values. The uncertainty factor used to determine an RfD may give a false impression that risk disappears as the RfD drops below some value. Risk may vary significantly for different individuals under the same conditions as well as with the inherent properties of the toxicant. Certainly, in assessing risk in children, due to the large gaps in knowledge, multiple uncertainty factors are used to provide a conservative estimate of risk. When calculating risks for carcinogenic substances, the slope factor gives a 95% upper confidence limit as the upper bound for the cancer risk value. Thus, there is a 5% chance that the true risk is actually greater than the calculated risk, giving a small degree of unreliability. Some authors have suggested that it may be more appropriate to report the best estimate as well as the upper and lower confidence limits in order to enhance the evaluation and interpretation of the risk value. In addition, high to low dose extrapolation models may give significantly different risk estimates in the low dose region as previously discussed. Finally, calculated risks due to multiple exposures may not be reliable due to the possibility of additive, synergistic, or antagonistic effects as discussed previously.

The exposure assessment is purported to be the weakest aspect of regulatory risk assessment and prone to widely variable estimates. These weaknesses are further accentuated when assessing exposure in children as discussed previously. Exposure values may be widely divergent depending on the sampling techniques, the inability to quantitate or characterize the exposure sources, and the differences between experimental conditions under which the data are collected and the actual exposure situation. Furthermore, exposure doses are calculated using standard intakes for an average 70 kilogram human which obviously inadequately characterizes the pediatric population. Finally, the level, frequency, duration, and routes of exposure differences are sources of uncertainty that limit the applicability of laboratory data to actual human exposure.
Many assumptions must be made in the risk assessment process to fill in the numerous areas of deficient knowledge regarding specific chemicals. A traditionally accepted procedure is to apply the assumption that appears least likely to lead to an underestimate of the actual human health risks. This assumption then leads to a very conservative estimate of risk and consequently a very imprecise risk value, perhaps overestimating the potential risk of a substance too much. On the other hand, a conservative risk estimate may provide a high degree of assurance that the actual risk will not exceed the estimate of risk and subsequently may ensure more protection of public health.

**Reconceiving Risk Characterization**

It has been suggested that risk characterization may fail if it is conceived only as a summary or translation of the results of technical analysis for the use of a decision maker. It may frame the scientific and technical information in such a manner that leads to an ill-judged decision or it may provide this information in such a way that is really not useful for the risk managers. Furthermore, a danger to risk managers is a misconception about how risk characterization should relate to the overall process of understanding and managing risk. The Committee of Risk Characterization (a group of experts convened by the National Research Council in 1994) proposed that it was necessary to reconceive risk characterization in order to increase the likelihood of achieving sound and acceptable decisions. They offered seven principles for implementing the process, which were published in 1996, *Understanding Risk: Informing Decisions in a Democratic Society*. These principles are conceptualized from a process of both analysis and deliberation and outlined in Table 18.

Analysis and deliberation will ideally rectify each other. Analysis enhances deliberation by informing discussion with facts, predictions, and basic understanding of risk-generating processes. Deliberation can help clarify areas of consensus and dispute, define needs for analysis and improved understanding, and bring new information and new perspectives to analysis. This expanded concept of risk characterization which uses a broad analytic-deliberative process appears to offer benefits for both the risk management and ultimately the risk communication processes by better risk characterizations and better risk decisions.
III. NEW APPROACHES TO RISK ASSESSMENT IN CHILDREN

MODIFYING THE RISK ASSESSMENT PARADIGM IN CHILDREN

Because of the difficulties encountered in risk assessment methodology, applying these methods to infants and children further magnifies the uncertainties and weaknesses. The current paradigm of risk assessment places the toxicant or hazardous substance at the center of the discussion and subsequently analyzes data on effects, exposures, and toxicity. Children’s unique susceptibilities and distinct exposure patterns that have been previously discussed must be considered in the risk assessment process. These considerations necessitate that children, not toxicants, are placed at the center of the risk assessment paradigm.\textsuperscript{96} (Figure 6) This new approach to risk assessment would then focus on a new set of questions that take into account children’s special vulnerabilities.\textsuperscript{1}

- What is the child exposed to?
- How is the child exposed and at what stage of development?
- Are these exposures of particular concern for children on the basis of available data?
- What are the effects of acute exposures or long-term, low-level exposure?
- What are the delayed effects?
- What are the effects of multiple and cumulative exposures?
- What are the transgenerational effects?
- Are children more or less sensitive than adults?
- What additional information/research is needed to adequately characterize health concerns?

Using this model, data would need to be collected and analyzed on the basis of children’s exposures and not extrapolated from adult data.

The ATSDR has addressed the inclusion of child-oriented health risk assessment in their Toxicological Profiles.\textsuperscript{6} These documents are current reviews of the potential hazards to humans of the 275 chemicals on the
National Priorities List. (See Information Resources for the Risk Assessment Process). Several pediatric sections are currently being added to the Toxicological Profiles. These include:

- How can the substance affect children? (This general question will be addressed in the Public Health section)
- How can families reduce the risk of exposure to a substance?
- Children’s Susceptibility. (See Table 19 for specific questions)
- Identification of Data Needs: Children’s Susceptibility
- Exposures to Children. (See Table 19 for questions)
- Identification of Data Needs: Exposures to Children

All Toxicological Profiles developed after 1996 will have these new child health sections.

As discussed previously, several studies have demonstrated that there is very little toxicity data available for the majority of chemicals produced, giving us no reliable information about their safety. A high priority for improving the hazard identification process is to target toxicity screening for those chemicals to which children are most exposed, that is, chemicals in their home, schools, and play areas. It is necessary to develop a strategy to assess chemicals and make toxicity data available to focus the risk assessment process even further for infants and children.

Defining and characterizing exposure patterns in children is probably the most important factor in developing new approaches to risk assessment in children. The National Research Council concluded in their 1993 report that differences in exposure between adults and children were more likely to account for differences in risk than were age-related differences in toxicity.\(^{119}\) As the new paradigm for child health risk assessment demonstrates, it is imperative that new research focus on answering those crucial questions related to types and durations of exposures.

Exposure research in children needs to improve. Studies of children’s activity patterns are an essential component of estimating their exposures. In addition, a better understanding of how to quantify exposures from children’s activities is needed. For example, how often does a child bring a toy to his/her mouth that has touched a contaminated surface? What is the distribution of ingestion rates of soil and dust among children in various age
ranges? Very little to no data is available on the exposures derived from dermal contact, ingestion and inhalation of chemicals on floors and household dust, and in the unique indoor breathing zone of a child. Research initiatives are underway to look at the dietary intake of children more precisely so that exposure and risk to chemicals in food can be better understood.

Because children consume much more of certain foods on a body-weight basis than adults, it is important to have accurate data on food and water consumption patterns for infants and children. This model must also allow for changes in exposure and susceptibility with age. Thus, it is necessary to delineate with more precision what children eat, drink, and inhale during the first few years of life by addressing each age group separately—infancy, toddler, school-age, and adolescence-- because of the wide variability during these time periods. Exposure estimates should be developed differently depending on whether acute or chronic effects of a chemical are of concern. Individual daily ingestion/inhalation/absorption is more appropriate for assessing acute toxicity of exposures while averages of exposure parameters are an appropriate measure of exposure for assessing the risk of chronic toxicity. However, the wide variability of children’s daily exposures makes this assessment of chronic toxicity extremely challenging.

Because children may have multiple chemical exposures in different environments which may be difficult to evaluate, it may be easier to construct a model for assessing toxicity which categorizes toxicants by their mechanism of toxicity. As discussed previously in the section on exposure assessment, it is unclear how to combine the effects from multiple exposures and estimate a cumulative potential risk. Some chemicals may be simply additive while others may inhibit each other or even be synergistic and magnify risk. Thus, if one is able to determine that multiple chemicals have the same mechanism of action, it may be more accurate to evaluate multiple exposures. For example, many pesticides are organophosphates and thus inhibit acetylcholinesterase. It may then be rational to develop a risk assessment procedure which accounts for multiple exposures of this class of chemicals and may provide a more accurate estimate of risk of harmful effects.
It is also imperative that highly exposed populations of children be identified to obtain more toxicity information. Research must target highly exposed children, i.e. children of migrant farm workers, or children of lead battery plant workers, which may help to identify further information from particular chemicals. In addition, information from unique cancer clusters (i.e. DES-exposed children) may help to elucidate some additional exposure issues in children. There also may be subsets of children who are highly susceptible on the basis of genetic variability.

Determining whether current techniques used to collect environmental samples and measure contaminant concentrations are representative of children’s exposures is also very important. Environmental sampling must take into account the special characteristics of children’s environments as previously discussed. For example, air-monitoring measurements may need to be obtained closer to the ground where children breathe. Adaptation of exposure assessment techniques for children are necessary to provide a more accurate evaluation of this step in the risk assessment process. Several strategies have been suggested to study the differences in exposure between children and adults: recruitment of pediatric clinics and child-specific facilities such as schools or day care for monitoring; and parent-child study pairs for meaningful comparisons between genetically similar people and similar exposures on the same day. Smaller air monitoring pumps to be placed in backpacks or fanny packs are available that are more comfortable and practical for children. Passive badge monitoring with relatively tamper-resistant instruments is also available. Use of activity pattern diaries is another modality that may help to more accurately assess exposure patterns in children.

Finally, it is imperative to characterize exposures that occur during critical periods of development in which susceptibility to toxicants may be significantly increased. As discussed previously, exposures at periods of development when tissues or organ systems are rapidly developing, may greatly increase the child’s risk of adverse health effects, especially cancer. Developmental toxicants are important to recognize as they may result in spontaneous abortions, malformations, early postnatal mortality, reduced birth weight, and/or neurological abnormalities. Scientists generally agree that approximately 3% of all developmental defects are attributable to
exposure to toxic chemicals and physical agents, including environmental factors. Also, it is generally agreed that 25% of all developmental defects may be due to a combination of genetic and environmental factors which include chemicals. Several environmental agents are known developmental toxicants for humans. These include lead, PCBs, methyl mercury, ionizing radiation, glycol ethers, and ethanol. Other agents are suspected human developmental toxicants based on animal studies and include pesticides such as DDT and vinclozolin. Developmental and reproductive toxicants added to the U.S. Toxic Release Inventory in 1994 are shown in Table 20. The EPA’s Guidelines for Developmental Toxicity Risk Assessment provides a complete risk assessment guideline for developmental toxicity.\textsuperscript{177}

Assessing toxicity in children requires that multiple uncertainty factors be used until better data is obtained so that risk is not underestimated. As a result of the 1996 Food Quality Protection Act, an additional 10-fold uncertainty/safety factor must be added to account for prenatal or postnatal developmental toxicity. (The implementation of this legislation is still in progress.) In addition, the U.S. EPA must consider all available data on in utero effects. For nonthreshold effects like carcinogens, the U.S. EPA adds a 100-fold uncertainty factor where prenatal/postnatal toxicity and exposure data for children are such that the risks for children have not been well defined and may be greater than the risks for adults.\textsuperscript{63,177} In the future, smaller uncertainty factors may be able to be used if the hazards for children are well understood and characterized.

The use of biologically based dose response (BBDR) modeling may have a significant impact on quantitative risk assessment in infants and children.\textsuperscript{62} These models incorporate underlying biologic processes, pharmacokinetics, and dose-reponse relationships into a comprehensive model that more accurately predicts risks from animal data to humans and from high doses to low doses.\textsuperscript{120} Knowing the inherent limitations of the current risk assessment paradigm for children, this modeling technique may provide much more accurate information for developmental toxicity risk assessment. Ongoing research is continuing to look at multiple toxic endpoints taking into account age-specific changes in organ size and growth rates.
It is imperative that scientists and regulators focus their efforts on understanding the developmental toxicity of environmental agents in order to protect developing embryos, fetuses, and children against any potential hazards. Extraordinary advances in genomics and developmental biology in the last five years provide an enormous opportunity for scientists to improve substantially the detection of developmental toxicants and determine their mechanisms of toxicity. As a result, the opportunities to improve the risk assessment process for developmental toxicants greatly increases. The National Research Council undertook an extensive project in the 1990s to evaluate and clarify the role of environmental agents in human developmental defects. They published their findings and recommendations in *Scientific Frontiers in Developmental Toxicology and Risk Assessment*.122 This report provides a comprehensive evaluation and analysis of the current understanding of the mechanisms of action of chemicals that result in developmental defects and makes recommendations for the improvement of developmental toxicity risk assessment. Specifically, the Committee on Developmental Toxicology concludes that:

“…. the major recent advances in developmental biology and genomics can be used to improve qualitative and quantitative risk assessments by integrating toxicological and mechanistic data on a variety of model test animals with data on human variability in genes encoding components of developmental processes, genes encoding enzymes involved in the metabolism of chemicals, and genes encoding receptors and transporter proteins that move these chemicals and their metabolites in and out of the cell.” 122

The report outlines a multidisciplinary, multilevel, interactive approach to improve risk assessment for human developmental defects. This novel approach to risk assessment includes a wide variety of information sources on risk assessment—assessment of toxicity and mechanism of action of chemicals in a variety of model systems (in vitro assays, nonmammalian models, and mammalian models) as well as assessment of toxicity, susceptibility and exposure in human populations. It should provide a guide for obtaining the type of data which is needed for a comprehensive cross-species model of exposure and development. This multidisciplinary, multilevel approach is needed to “bridge the gap” between the emerging scientific information and the assessment of human risk.122 Finally, the report discusses three recommendations of the committee:
1.) To improve the understanding of the mechanisms of action of toxicants, critical molecular targets of toxicants must be identified among the components of developmental processes.

2.) To study how the new information about development and developmental toxicity can address the uncertainties in quantitative and qualitative risk assessment. Specific areas which need to be addressed include intraspecies differences in sensitivity to toxicants, cross-species extrapolation, extrapolation of high-dose exposure of small populations of test animals to low-dose, long-term exposure of a large human populations, and expanded test information for numerous chemicals and mixtures of chemicals.

3.) To improve the interdisciplinary advances in developmental toxicology, databases of developmental toxicology, developmental biology, and genomics must be better linked on the Internet, and that multidisciplinary outreach programs be established for the effective exchange of information and techniques related to the analysis of developmental defects and to the assessment of toxicity for risk assessment.

This report will serve as an outstanding guide for investigators and the other numerous people involved in assessing risk in children in the 21st century.

**USING BIOLOGIC MARKERS IN THE PEDIATRIC RISK ASSESSMENT PROCESS**

Using biologic markers is another new approach to further strengthen the risk assessment process in children. Although the increased susceptibility of infants and children to environmental toxicants has been demonstrated in multiple studies, the type and extent of pediatric illness secondary to environmental exposure has not been well characterized. Few data are available on the magnitude of children’s exposure to most environmental toxicants. There are many reasons for this lack of information. Documentation of exposure is difficult in the fetal and pediatric population. Modeling of exposure is difficult because there are few studies documenting where children spend their time. The individual dose to a child is difficult to document even in situations with known exposures. In addition, the long latency of many environmentally induced diseases makes their etiology difficult to determine, making
retrospective studies difficult to conduct. A child may be exposed to more than one environmental toxicant as well as other agents, which may confound the association of one toxicant to a particular illness. Extrapolation of animal models to human children is difficult. Many of the critical stages of development are not well characterized in animals. Thus, it is important to have a tool that can overcome many of these deficiencies. One of the primary purposes of biomarkers in environmental health research is to identify exposed persons, so that risk can be predicted and disease prevented.

Biologic markers, or biomarkers, are cellular, biochemical or molecular alterations that are measurable in biologic media such as human tissues cells or fluids. Biologic markers have been viewed as forming a continuum of events from external exposure to an environmental agent to the development of disease. Three general categories of biomarkers have been defined: biomarkers of exposure to chemical or physical agents, biomarkers of effects of those exposures, and biomarkers of susceptibility. (Figure 7)

A biomarker of exposure is an exogenous chemical or its metabolite(s) or the product of an interaction between a xenobiotic agent and some target molecule or cell that is measured in a compartment within an organism. Biomarkers of exposure can be divided into internal dosimeters or markers of biologically effective dose. An internal dose biomarker measures the amount of a toxicant or its metabolite present in cells, tissues, or body fluids. Markers of internal dose indicate that an exogenous agent has entered the body of an exposed individual. An internal dose marker may measure the agent itself unchanged (e.g. blood lead) or a metabolically altered form of the agent. Most biomarkers of exposure are measured in blood, urine, or exhaled alveolar air.

An example of a successful pediatric internal dosimeter is blood lead. This biomarker has had a significant impact on the prevention and treatment of lead exposure in children. Other examples include cord blood measurements of polychlorinated biphenyls and cotinine (a metabolite of nicotine) which document fetal exposure to these chemicals. Measurement of cotinine (a metabolite of nicotine) in serum, urine, and saliva is a biomarker for children’s exposure to environmental tobacco smoke. Urinary nitrophenol concentration has been used as a
marker for methyl parathion exposure and urinary trichloroacetic acid has been used as a marker for trichloroethylene exposure, a common pollutant at Superfund sites.\textsuperscript{55}

The measurement of internal dose biomarkers assesses the amount of a chemical that is ultimately absorbed into the body after the influence of exposure factors such as behavior, contact rates, protective measures, and differences in respiratory rate.\textsuperscript{193} Heavy metals such as mercury and arsenic can be directly measured in urine samples. Benzene is a human leukemogen and found in tobacco smoke and gasoline. Urine \textit{trans,trans}-muconic acid (MA), a metabolite of benzene, has been used as a measurement of environmental exposure to benzene in children.\textsuperscript{193}

Other examples of biomarkers of exposure are listed in Table 21. Urinary 1-hydroxypyrene has been utilized as an exposure biomarker for polycyclic aromatic hydrocarbons.\textsuperscript{27,28,158} One study estimates that 65\% of an infant’s total exposure to benzo[a]pyrene comes from house dust.\textsuperscript{139} Because of the variability in children’s nondietary ingestions of dust and difficulty in quantifying hand-to-mouth activity, a PAH biomarker such as urinary 1-hydroxypyrene may be particularly useful in assessing the house dust exposure. Another example is the use of breath sampling as a method for evaluating environmental exposures to volatile organic compounds (VOCs).\textsuperscript{51,190} This methodology is especially well suited for children because of its noninvasiveness and ease with which samples can be collected.

Unfortunately, few chemicals have well-developed and well-validated assays for quantification. Individual metabolite biomarkers may be nonspecific because of formation from more than one parent compound like MA. Finally, internal dosimeters do not provide information about the interactions of the compound with critical cellular targets—or the biologic effective dose. For example, a child 2 years of age and a newborn may have the same blood lead concentration, but the interaction of the lead with critical sites in the central nervous system may be greater for the newborn because of a underdeveloped blood-brain barrier.\textsuperscript{15}

Biologically effective dose measurement assesses damage at the target organ affected. It is the amount of toxicant that has interacted with a critical molecular site where the biologic effect is initiated or with an established surrogate.\textsuperscript{15} Because marker categories represent a continuum, determining a clear distinction between markers of
exposure and markers of effect is difficult. Carboxyhemoglobin is an example of this type of biomarker for carbon monoxide poisoning. Frequently surrogate biomarkers are used because of lack of knowledge of the actual target molecule and/or inaccessibility of the target tissue such as the brain. Measurements of red blood cell and plasma cholinesterase in children exposed to organophosphate or carbamate pesticides are an example of biomarkers that determine biologically effective doses by effects on surrogate tissues.

Examples of biologically effective dose markers include DNA and protein adducts that are electrophilic molecules covalently bonded with DNA or protein (e.g. albumin or hemoglobin). DNA-adduct formation is believed to be a step in the carcinogenic process. The measurement of carcinogen-DNA adducts in white blood cells is a surrogate for the measurement of the specific DNA-carcinogen interaction initiating the cancer in the target tissue. DNA-adduct formation is believed to be a step in the carcinogenic process. The measurement of carcinogen-DNA adducts in white blood cells is a surrogate for the measurement of the specific DNA-carcinogen interaction initiating the cancer in the target tissue. It is more mechanistically relevant to disease than internal dose, since it takes into account differences in metabolism of the chemical as well as the extent of repair of DNA adducts. Numerous studies have shown PAH-DNA adducts in people associated with specific occupational exposures. Carcinogen-protein adducts in blood have also been used as surrogates for the biologically effective dose in the target organ. Recent studies have demonstrated that children of smoking mothers had significantly higher levels of PAH-albumin adducts than children of nonsmoking mothers. Another example is urinary aflatoxin-DNA adduct measurements which have demonstrated that aflatoxin-exposed individuals who are also infected with the hepatitis B virus are at high risk for hepatic cancer. The aflatoxin-albumin adduct has been measured in fetal cord blood at lower levels than maternal blood. This measurement suggests that the fetal liver is able to metabolize aflatoxin, resulting in a carcinogenic exposure to its cells.

A biomarker of effect is a measurable alteration of an endogenous component within an organism, that, depending on magnitude, can be recognized as a potential or established health impairment or disease. A variety of biomarkers fall into this category including gene mutations, alterations in oncogenes and tumor suppressor genes, sister chromatid exchanges, and chromosomal aberrations. One example is the alteration in pulmonary function
tests in children after exposure to environmental tobacco smoke. Biomarkers of effect are not chemical- or agentspecific and can be affected by other environmental exposures or by lifestyle. Numerous other indoor air pollutants may cause alteration of pulmonary function in children. Individuals exposed to high concentrations of ethylene oxide have higher levels of sister chromatid exchanges compared with less exposed individuals. Another example is lead which alters neurotransmission and synaptogenesis in the central nervous system.

A biomarker of susceptibility is an indicator of an inherent or acquired property of an organism to increase the internal dose of a xenobiotic or to alter the response to the challenge of exposure to a specific xenobiotic substance. Biomarkers of susceptibility indicate individual factors, namely variations in genetic structure, that can affect response to environmental agents. Genetic variabilities may make an individual more susceptible to health effects from environmental exposures as discussed previously. Examples include individual variability in Phase I and Phase II metabolic enzyme activities. CYP1A1, a P450 enzyme, is highly responsive to PAH exposures. Its inducibility has been associated with higher risks of lung cancer in smokers. However, exposure can also decrease susceptibility to the effects of later exposure. An example would be an exposure to a toxicant that induces the enzyme responsible for its excretion. A subsequent dose would be metabolized more readily and hence cause less harm. A biologic marker of susceptibility may also be a marker of resistance when the internal dose or the health effect is less than the general population in children with this marker. An example of increased susceptibility would be the acquisition of methemoglobinemia due to nitrate exposure in the face of glucose-6-phosphate dehydrogenase deficiency.

Biomarkers can be utilized in all steps of the risk assessment process. Sensitive biomarkers can be used to assess early indicators of harm in human populations with suspected environmental exposure. In the health hazard identification step, biomarkers can be substituted for end points located at the exposure-disease continuum such as a specific disease or tumor formation. Biomarkers can also provide information on the metabolic and physiologic differences between animals and humans, potentially eliminating the need for uncertainty factors. In the toxicity
assessment step, they may also be used for low-dose extrapolation. It may be possible to characterize dose-response relationships at response rates orders of magnitude lower and over a much greater dose range than is currently possible by using sensitive exposure and effect biomarkers that show strong correlation with tumor development. Finally, incorporating exposure and effect biomarkers in the characterization of interindividual variability will allow protection of vulnerable individuals, while avoiding the need to use uncertainty factors to account for this variability.49

Biomarkers could enhance and even simplify the exposure assessment process in children. Assessing exposure in infants and children is complex because exposures occur through multiple routes and pathways in children and there is a high ratio of sampling burden to subject ability (cognitive and physical limitations).193 Biomarkers may provide a much more straightforward assessment of exposure. Used in conjunction with more traditional methods of exposure assessment such as environmental monitoring and questionnaires, biomarkers will hopefully improve the accuracy of exposure measures in the following ways: they may help to reduce the misclassification of exposure measures, identify the most relevant exposure dose with respect to health outcome, integrate exposures over time and from various routes of entry into the body, identify specific exposures within a mixture of compounds, associate time of exposure with dose, and help to validate more traditional measures of exposure. As a result, the more accurately one can measure exposure, the more sensitive epidemiologic studies will be in detecting exposure/disease associations, especially weak associations.

Although a few epidemiologic studies have been conducted on children living near toxic dump sites, application of biomarkers to detect environmental toxic exposures in the clinical setting has been primarily used for lead poisoning and passive exposure to ETS. Certainly, the use of biomarkers in a clinical setting could facilitate the diagnosis of conditions in children that occur as a result of exposure to specific toxic substances. It should help to confirm that a child has been exposed to a particular toxicant and may provide a tool to monitor either the effects of
the toxicant or the effects of specific therapy. Incorporation of all these types of biomarkers into the risk assessment process can significantly enhance the documentation of exposure and toxicity.

Ideally, biologic markers for assessing risk in children should be inexpensive to collect and analyze, require a small amount of a practical biologic specimen, and be obtainable with low-to-noninvasive sampling techniques, making collection as painless as possible. In addition, the biomarker should be specific to a particular exposure, persist for a reasonably long time in the body, and integrate exposures over time and from various routes of entry into the body. Finally, to use biomarkers confidently in a child’s risk assessment, they need to have a high probability of being detected in the exposed population; i.e., have an assay that is sensitive, specific, reliable, and validated in human field studies. These criteria provide a great challenge for future researchers studying these markers in the dynamic pediatric population.

IV. NATIONAL INITIATIVES TO IMPROVE THE RISK ASSESSMENT PROCESS IN CHILDREN

In the last 10 years, there has been significant progress in the acknowledgement of the need for and development of national initiatives that address issues related to children’s environmental health. For decades, the federal government has registered, regulated and sometimes restricted potential environmental pollutants, from industrial chemicals to lawn fertilizers. Yet, until very recently, the potential adverse effects of these substances on the health of children had rarely been addressed.

Concern about the potential vulnerability of infants and children to dietary pesticides led the U.S. Congress in 1988 to request that the National Academy of Sciences/National Research Council examine this issue. In 1993 they published a landmark study on environmental risks to children entitled Pesticides in the Diets of Infants and Children. This report highlighted the unique vulnerability of children to environmental toxicants through their analysis of information on pesticides in the diets of infants and children. In addition, it emphasized the relative paucity of data relating environmental exposures and children’s health. This report also made several
recommendations that set the stage for future initiatives. Specifically, they recommended that the EPA modify its decision-making process for setting tolerances (the legal limit of pesticide residue allowed in or on a raw agricultural product and processed foods) to account for human health considerations rather than agricultural practices. Second, the committee felt it was essential to develop toxicity testing procedures for pesticide residues that specifically evaluate the vulnerability of infants and children.

In 1995 the EPA established an agency-wide policy that requires inclusion of infant and child health risk evaluation in all agency standard setting. This policy requires the development of a separate assessment of risk to infants and children. In 1996 the Environmental Protection Agency issued a national assessment of children’s environmental health issues and established a comprehensive National Agenda to Protect Children’s Health from Environmental Threats, mandating that the U.S. EPA major existing health standards be reviewed and new standards be set that ensure the protection of children.179 Also in 1996 the ATSDR launched their Child Health Initiative whose major objective was to emphasize children’s health in all of its agencies’ programs.6 As part of this project, they have trained officials to incorporate child health issues into public health assessments at Superfund hazardous waste sites. In addition, they are reformatting their Toxicological Profiles to expand coverage of the specialized toxicology of children including a section on children’s susceptibility and special exposures.

The Food Quality and Protection Act of 1996 pays special attention to the health considerations of children and other vulnerable populations in defining food standards. Special provisions focus on the development and implementation of survey procedures to ensure that adequate data on food consumption patterns of infants and children are collected. In addition, it states that the EPA must determine that a given tolerance level is not harmful to infants and children in establishing, modifying, leaving in effect, or revoking a tolerance or exemption for a pesticide chemical residue. This act is the first piece of environmental legislation since the legislation on lead poisoning in the 1980s that addresses children’s health and provides a framework on how to do it.
Finally, in 1997 an Executive Order was signed by President Clinton to reduce environmental health and safety risks to children. This order, “Protection of Children from Environmental Health Risks and Safety Risks”, requires federal agencies to assign high priority to evaluating and managing special environmental risks that may disproportionately affect children, ensure that regulatory standards account for these special risks, and coordinate research priorities on children’s health issues.

The field of pediatric environmental health has also emerged in the last 5 years as a response to the need for adequate knowledge and expertise for the diagnosis, treatment, and prevention of illness due to perinatal and pediatric exposures to environmental hazards. Pediatric Environmental Health Specialty Units (PEHSU) have been established in the country to provide much of this expertise. This integration of clinical toxicology, pediatrics, and environmental/occupational medicine will undoubtedly further advance the information and research gaps by helping to implement many of the recommendations made by the aforementioned national initiatives.
V. INFORMATION RESOURCES FOR THE RISK ASSESSMENT PROCESS AND PEDIATRIC ENVIRONMENTAL HEALTH

Numerous resources are available for the pediatric health care provider to gather detailed information on toxic chemicals and substances in addition to some web sites specifically dealing with pediatric environmental health issues. The following is a selected list of resources that may be helpful in gathering information on children’s environmental health in general and for the different steps of the risk assessment process. Most the information may be downloaded directly from the internet or ordered via information given on the website.

ATSDR (Agency for Toxic Substances and Disease Registry) Office of Children’s Health


The web site provides information and comprehensive resources on pediatric environmental issues including pediatric toxicology references.

ATSDR Public Health Assessments

http://www.atsdr.cdc.gov/HAC/PHA

ATSDR is required by law to conduct a public health assessment at each of the sites on the EPA National Priorities List. The aim of these evaluations is to determine if people are being exposed to hazardous substances and, if so, whether that exposure is harmful and should be stopped or reduced. Sections of the reports include review of exposure data, determination of health effects, conclusions about the level of health threat posed by a site, and recommendations to stop or reduce exposure in a public health action plan. The reports focus on public health, or the health impact on the community as a whole, rather than on individual risks.
ATSDR Toxicological Profiles

http://www.atsdr.cdc.gov/toxpro2.html

The ATSDR has developed 261 profiles for hazardous substances found at National Priorities List (NPL) sites. These profiles contain general toxicity information and levels of exposure associated with lethality, cancer, genotoxicity, neurotoxicity, developmental and reproductive toxicity, immunotoxicity, and systemic toxicity. Health effects in humans and animals are discussed by exposure route and duration.

The Children’s Environmental Health Network

http://www.cehn.org/

The Children’s Environmental Health Network is a national multi-disciplinary project whose mission is to promote a healthy environment and protect the fetus and the child from environmental health hazards. The web site provides information on the Network, children’s environmental health issues, and links to sources of information and resources in the field.

Exposures Factors Handbook Volumes I, II, III.

http://www.epa.gov/ncea/pdfs/efh/front.pdf

The Exposure Factors Handbook provides a summary of the available statistical data on various factors used in assessing human exposure. The handbook was developed for risk assessors who need to obtain data on standard factors to calculate human exposure to toxic chemicals. These factors include drinking water consumption, soil ingestion, inhalation rates, dermal factors, consumption of fruits and vegetables, fish, meats, dairy products, homegrown foods, breast milk intake, human activity factors, consumer product use, and residential characteristics. Recommended values are for the general population and also for various segments of the population who may have characteristics different from the general population.
**HazDat**


HazDat is the ATSDR’s Hazardous Substance Release and Health Effects Database. It is the scientific and administrative database developed to provide access to information on the release of hazardous substances from Superfund sites or from emergency events and on the effects of hazardous substances on the health of human populations. Information included in HazDat are site characteristics, activities, and events, contaminants found, contaminant media and maximum concentration levels, impact on population, community health concerns, ATSDR public health threat categorization, ATSDR recommendations, environmental fate of hazardous substances, exposure routes, and physical hazards at the site/event. HazDat also contains substance-specific information such as health effects by route and duration of exposure, metabolites, interactions of substances, susceptible populations, and biomarkers of exposure and effects.

**Health Effects Assessment Summary Tables (HEAST).**

This resource includes toxicity information and values for chemicals for which Health Effects Assessments (HEAS), Health and Environmental Effects Documents (HEEDS), Health and Environmental Effects Profiles (HEEPs), Health Assessment Documents (HADs), or Ambient Air Quality Criteria Documents (AAQCDs) have been prepared. HEAST summarizes interim RfDs and slope factors in addition to other toxicity information as well as directs readers to current sources of supporting toxicity information. This resource may be helpful when verified information for a chemical is not in IRIS. HEAST can be obtained by request from the Superfund Docket (202-382-3046) or through EPA Center for Environmental Research Information (CERI) in Cincinnati, OH (513-569-7562).
Healthy Children—Toxic Environments: Acting on the Unique Vulnerability of Children Who Dwell Near Hazardous Waste Sites


This report by the Child Health Workgroup of the ATSDR recommends actions and major directions to be undertaken for children living near hazardous waste sites. It discusses the unique susceptibility of children, the imperative of programs for children’s environmental health, the impact of ATSDR’s programs on children, and the child health workgroup recommendations.

IRIS- Integrated Risk Information System

http://www.epa.gov/iris/

The Integrated Risk Information System (IRIS) is an electronic database, prepared and maintained by the U.S. EPA, containing information on human health effects that may result from exposure to various environmental chemicals. It was initially developed for EPA staff in response to a growing demand for consistent information on chemical substances for use in risk assessments, decision-making and regulatory activities. The chemical files contain descriptive and quantitative information on: 1.) oral reference doses and inhalation reference concentrations for chronic noncarcinogenic health effects (RfDs and RfCs, respectively); and 2.) hazard identification, oral slope factors, and oral and inhalation unit risks for carcinogenic effects. IRIS contains only those RfDs and slope factors that have been verified by the RfD and CRAVE (Carcinogen Risk Assessment Verification Endeavor) workgroups and considered to be the preferred source of toxicity information. For technical questions about the scientific information content in IRIS, call the U.S. EPA Risk Information Hotline at 1-513-569-7159.

National Pesticide Information Center (NPIC)

http://www.ace.orst.edu/info/nptn/index/html 1-800-858-7378

The NPIC takes calls from both the public and health professionals and answers both general and technical questions on pesticides, provides emergency help and referrals, and offers advice on pesticide poisoning prevention. The NPIC
web site allows users to download a brochure from the network (that pediatricians could display in a waiting room),
as well as annual reports and various fact sheets about different pesticides. It also offers technical and general
resources, including human and animal poison control centers, regulatory issues and tips on pest control. The NPIC is
a cooperative effort between the EPA and Oregon State University.

**Toxic Release Inventory (TRI)**

http://www.epa.gov/tri/tridata/tri00/index.htm

The Toxic Release Inventory contains information about more than 650 toxic chemicals that are being used,
manufactured, treated, transported, or released into the environment. Manufacturers of these chemicals are required
to report the locations and quantities of chemicals stored on-site to state and local governments. These reports are
submitted to the EPA which then compiles this data in an on-line, publicly accessible national computerized database.
Information and chemical reports which tabulate air emissions, surface water discharges, releases to land,
underground injections, and transfers to off-site locations are included.

**MSDS Online**

www.msdsonline.com

The website provides free material safety data sheets which contain immediate information on industrial products.
MSDS are required by OSHA to contain certain information including the identify of the product and health hazards
of the chemicals.
VI. REFERENCES


100. Longnecker MP, Rogan WJ, Lucier G. The human health effects of DDT (dichlorodiphenyltrichloroethane) and PCBs (polychlorinated biphenyls) and an overview of organochlorines in public health. Ann Rev Publi Health 1997;18:211-244.


103. Luyens JN. The legacy of well-water methemoglobinemia. JAMA 1987;257:2793-2795.


197. Whyatt RM, Perera FP. Application of biologic markers to studies of environmental risks in children and the developing fetus. Environ Health Perspect 1995;103 (Suppl 6);105-110.


Table 1. Taking a Pediatric Exposure History - Selected to Questions to Ask

<table>
<thead>
<tr>
<th>Exposure Issue</th>
<th>Questions to Ask</th>
</tr>
</thead>
</table>
| Child’s environment including home, school, daycare | • Do you live in a house, apartment, or mobile home?  
• On what level of the dwelling is the child’s room located?  
• What is the age and condition of your home?  
• How is your home heated? Do you have a fireplace or wood stove?  
• Do you use pesticides inside or outside your home?  
• What hobbies do your child and other family members have?  
• Is your home, the child’s daycare, or school near a polluted body of water, industrial plant, commercial business, or dump site? |
| Adolescent work history; family members’ jobs | • What is your occupation?  
• What is your spouse’s occupation?  
• Do other members of the family have jobs? What are they?  
• For adolescents: Do you work? What kind of job do you have and what hours do you work? |
| Exposure to environmental tobacco smoke | • Do you smoke tobacco products?  
• If so, 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? |
| Child’s Diet | • For breastfeeding mothers:  
  Do you take any drugs or medications?  
  Do you smoke?  
• 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 products do you buy? Organic? Local, in-season? |
| Risk Factors for Lead Poisoning | • Does the child live in or visit a house built before 1960 that has peeling or chipping paint?  
• Does the child live in or regularly visit a house built before 1960 that recently was renovated or where renovation is ongoing or planned?  
• Is there a brother, sister, housemate, or playmate being followed or treated for lead poisoning (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? |

Table 2. Potential Toxic Exposures Related to Hobbies

<table>
<thead>
<tr>
<th>Activity</th>
<th>Potential Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Painting</td>
<td>• Toxic pigments, i.e., arsenic, cadmium, chromium, lead, mercury</td>
</tr>
<tr>
<td></td>
<td>• Acrylic emulsions</td>
</tr>
<tr>
<td></td>
<td>• Solvents</td>
</tr>
<tr>
<td>Ceramics</td>
<td>• Raw materials</td>
</tr>
<tr>
<td></td>
<td>• Firing</td>
</tr>
<tr>
<td></td>
<td>• Gas-fired kilns</td>
</tr>
<tr>
<td></td>
<td>• Colors and glazes containing barium carbonate; lead, chromium, uranium, cadmium</td>
</tr>
<tr>
<td></td>
<td>• Fumes of fluoride, chlorine, sulfur dioxide</td>
</tr>
<tr>
<td></td>
<td>• Carbon monoxide</td>
</tr>
<tr>
<td>Sculpture and Casting</td>
<td>• Silica (silicon dioxide)</td>
</tr>
<tr>
<td></td>
<td>• Metal fumes, sand(silica) from molding, binders of phenol formaldehyde or urea formaldehyde</td>
</tr>
<tr>
<td>Welding</td>
<td>• Metal fume, ultraviolet light exposure, welding fumes, carbon dioxide, carbon monoxide, nitrogen dioxide, ozone or phosgene</td>
</tr>
<tr>
<td>Woodworking and Refinishing</td>
<td>• Wood dust</td>
</tr>
<tr>
<td></td>
<td>• Solvents (methylene chloride)</td>
</tr>
<tr>
<td>Photography</td>
<td>• Developer</td>
</tr>
<tr>
<td></td>
<td>• Stop bath</td>
</tr>
<tr>
<td></td>
<td>• Stop hardener</td>
</tr>
<tr>
<td></td>
<td>• Fixer</td>
</tr>
<tr>
<td></td>
<td>• Hardeners and stabilizers</td>
</tr>
<tr>
<td></td>
<td>• Hydroquinone, phenylenediamine, alkalis</td>
</tr>
<tr>
<td></td>
<td>• Acetic acid</td>
</tr>
<tr>
<td></td>
<td>• Potassium chrome alum (chromium)</td>
</tr>
<tr>
<td></td>
<td>• Sodium thiosulfite, acetic acid</td>
</tr>
<tr>
<td></td>
<td>• Formaldehyde</td>
</tr>
</tbody>
</table>

Adapted from Goldman RH, Peter JM. The occupational and environmental health history. JAMA 1981;246:2831-2836. Reproduced with permission
<table>
<thead>
<tr>
<th>Occupation</th>
<th>Toxic Exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carpenters and Loggers</td>
<td>Plicatic acid; Exotic woods; Chlorine; Hydrogen sulfide; Methylmercaptan; Pentachlorophenol; Copper; Nickel</td>
</tr>
<tr>
<td>Concrete workers and masons</td>
<td>Chromium; cement; cobalt; calcium hydroxide; silica; asbestos; lime</td>
</tr>
<tr>
<td>Dry Cleaners</td>
<td>Perchloroethylene; carbon tetrachloride; trichloroethylene; stoddard solvent; naphtha; trichloroethane</td>
</tr>
<tr>
<td>Exterminators</td>
<td>Organophosphates; pyrethrin; pyrethroids; arsenic</td>
</tr>
<tr>
<td>Farmers/farm personnel</td>
<td>Ammonia; oxides of nitrogen; methane; carbon dioxide/ nonionizing radiation/ mycotoxins; pesticides</td>
</tr>
<tr>
<td>Floor and carpet layers</td>
<td>Methylmethacrylate; glues; adhesives; acrylates; epoxies; polyesters; ethyl acrylates</td>
</tr>
<tr>
<td>Jewelers</td>
<td>Cyanide; mercury; chromium-6</td>
</tr>
<tr>
<td>Mechanics</td>
<td>Gasoline; degreaser; Stoddard solvent; benzene; methylethylketone (MEK)</td>
</tr>
<tr>
<td>Morticians</td>
<td>Formaldehyde; glutaraldehyde</td>
</tr>
<tr>
<td>Office personnel</td>
<td>Volatile organic chemicals (VOCs); environmental tobacco smoke; mold; formaldehyde</td>
</tr>
<tr>
<td>Painters and furniture refinishers</td>
<td>Hydrofluoric acid; dyes; solvents; lacquers; methylene chloride diisocyanates; benzo[a]pyrene; mercury</td>
</tr>
<tr>
<td>Plumbers</td>
<td>Lead; asbestos; solder flux; zinc; tetrahydrofuran; cyclohexanone; fluoroarbons</td>
</tr>
<tr>
<td>Printers</td>
<td>Toluene; azoridine; carbon black; naphtha n-hexane; isopropyl alcohol</td>
</tr>
<tr>
<td>Roofers</td>
<td>Coal tar; benzo[a]pyrene; polycyclic aromatic hydrocarbons; fiberglass</td>
</tr>
<tr>
<td>Shoemakers</td>
<td>2,5-hexandione; benzene oxide; tri-orthocresylphosphate (TOCP); methylethylketone (MEK); toluene solvents; vinyl chloride; leather dust</td>
</tr>
<tr>
<td><strong>Table 4. Key Definitions</strong></td>
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<td>----------------------------</td>
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<tr>
<td><strong>Hazard:</strong> an act or phenomenon posing potential harm to some person(s) or thing(s); the magnitude of the hazard is the amount of harm that might result</td>
<td></td>
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<tr>
<td><strong>Risk:</strong> the chance of injury, damage, or loss; adds to the hazard the probability that the potential harm or consequence will develop as well as qualitative or outrage factors that contribute to people’s perception (i.e. voluntariness, control, fairness, familiarity, etc.)</td>
<td></td>
</tr>
<tr>
<td><strong>Risk Assessment:</strong> characterization of the potential adverse effects of exposures to hazards; quantitative risk assessment includes four components—hazard identification, exposure assessment, toxicity assessment, and risk characterization.</td>
<td></td>
</tr>
<tr>
<td><strong>Risk Communication:</strong> an interactive process of exchange of information and opinion among individuals, groups, and institutions; it may involve single or multiple messages about the nature of risk and other messages that express concerns, opinions, or reactions to risk messages</td>
<td></td>
</tr>
<tr>
<td><strong>Risk Management:</strong> the process of taking results from the risk assessment and arriving at a decision of action or inaction; strategies or options can be broadly classified as regulatory, nonregulatory, economic, advisory, or technological</td>
<td></td>
</tr>
<tr>
<td><strong>Risk Message:</strong> a written, verbal, or visual statement containing information about risk; may or may not include advice about risk reduction behavior</td>
<td></td>
</tr>
</tbody>
</table>
Table 5. Hazard Identification

- Gather and analyze relevant site data to include:
  - Contaminant identified
  - Contaminant concentrations in the sources and media of interest (air, soil, water)
  - Characteristics of sources / release potential
  - Characteristics of the environmental setting that may affect the fate, transport, and persistence of the contaminants

- Identify potential chemicals of concern
Table 6. Elements of a Conceptual Evaluation Model


<table>
<thead>
<tr>
<th>VARIABLES</th>
<th>HYPOTHESES TO BE TESTED</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOURCES</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Contaminants</td>
</tr>
<tr>
<td></td>
<td>• Concentrations</td>
</tr>
<tr>
<td></td>
<td>• Time</td>
</tr>
<tr>
<td></td>
<td>• Locations</td>
</tr>
<tr>
<td></td>
<td>• Source exists</td>
</tr>
<tr>
<td></td>
<td>• Source can be contained</td>
</tr>
<tr>
<td></td>
<td>• Source can be removed and disposed</td>
</tr>
<tr>
<td></td>
<td>• Source can be treated</td>
</tr>
<tr>
<td>PATHWAYS</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Media</td>
</tr>
<tr>
<td></td>
<td>• Rates of migration</td>
</tr>
<tr>
<td></td>
<td>• Time</td>
</tr>
<tr>
<td></td>
<td>• Loss and gain functions</td>
</tr>
<tr>
<td></td>
<td>• Pathway exists</td>
</tr>
<tr>
<td></td>
<td>• Pathway can be interrupted</td>
</tr>
<tr>
<td></td>
<td>• Pathway can be eliminated</td>
</tr>
<tr>
<td>RECEPTORS</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Types</td>
</tr>
<tr>
<td></td>
<td>• Sensitivities</td>
</tr>
<tr>
<td></td>
<td>• Time</td>
</tr>
<tr>
<td></td>
<td>• Concentrations</td>
</tr>
<tr>
<td></td>
<td>• Numbers</td>
</tr>
<tr>
<td></td>
<td>• Receptor is not impacted by migration of contaminants</td>
</tr>
<tr>
<td></td>
<td>• Receptor can be relocated</td>
</tr>
<tr>
<td></td>
<td>• Institutional controls can be applied</td>
</tr>
<tr>
<td></td>
<td>• Receptor can be protected</td>
</tr>
</tbody>
</table>
## Table 7. Top 20 Hazardous Substances on the National Priority List and Selected Health Effects

<table>
<thead>
<tr>
<th>SUBSTANCES</th>
<th>HEALTH EFFECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Arsenic</strong></td>
<td>GI: Vomiting, diarrhea</td>
</tr>
<tr>
<td></td>
<td>Neuro: Sensorimotor neuropathy</td>
</tr>
<tr>
<td></td>
<td>Skin: Dermatitis; hyperkeratoses; skin cancer</td>
</tr>
<tr>
<td></td>
<td>CV: Cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>Pulmonary: Pulmonary edema, cancer</td>
</tr>
<tr>
<td></td>
<td>Hematologic: Anemia</td>
</tr>
<tr>
<td><strong>2. Lead</strong></td>
<td>GI: Abdominal pain, constipation</td>
</tr>
<tr>
<td></td>
<td>Heme: Anemia</td>
</tr>
<tr>
<td></td>
<td>Neurological: Peripheral neuropathy; behavioral changes; encephalopathy; neurocognitive abnormalities</td>
</tr>
<tr>
<td></td>
<td>Renal: Nephropathy; hypertension</td>
</tr>
<tr>
<td><strong>3. Mercury</strong></td>
<td><strong>Elemental</strong></td>
</tr>
<tr>
<td></td>
<td>Pulmonary: Acute pneumonitis</td>
</tr>
<tr>
<td></td>
<td>Oral: Gingivitis, stomatitis</td>
</tr>
<tr>
<td></td>
<td>CNS: Neuropsychiatric changes (erythema); tremor</td>
</tr>
<tr>
<td></td>
<td><strong>Inorganic</strong></td>
</tr>
<tr>
<td></td>
<td>Pulmonary: Acute pneumonitis</td>
</tr>
<tr>
<td></td>
<td>GI: Esrroive esphagitis, gastritis; gastroenteritis</td>
</tr>
<tr>
<td></td>
<td>Renal: Proteinuria; acute tubular necrosis</td>
</tr>
<tr>
<td></td>
<td>Acrodyia</td>
</tr>
<tr>
<td></td>
<td>CNS: Behavioral and psychomotor impairments</td>
</tr>
<tr>
<td></td>
<td><strong>Organic</strong></td>
</tr>
<tr>
<td></td>
<td>Skin: Dermatitis</td>
</tr>
<tr>
<td></td>
<td>CNS: Sensorimotor changes, visual field constriction, tremor; cerebral palsy; mental retardation; neurodevelopmental delay; seizures</td>
</tr>
<tr>
<td><strong>4. Vinyl Chloride</strong></td>
<td>GI: Hepatic angiosarcoma</td>
</tr>
<tr>
<td></td>
<td>Skeletal: Acro-osteolysis</td>
</tr>
<tr>
<td></td>
<td>Extremity: Raynaud-type phenomena</td>
</tr>
<tr>
<td><strong>5. Polychlorinated Biphenyls (PCBs)</strong></td>
<td>Skin: Chloracne</td>
</tr>
<tr>
<td></td>
<td>GI: Increase in liver enzymes</td>
</tr>
<tr>
<td></td>
<td>Neuro: Neurocognitive delays in children</td>
</tr>
<tr>
<td><strong>6. Benzene</strong></td>
<td>Heme: Leukemia, aplastic anemia</td>
</tr>
<tr>
<td></td>
<td>CNS: CNS depression, ataxia, confusion</td>
</tr>
<tr>
<td></td>
<td>Skin: Dermatitis</td>
</tr>
<tr>
<td><strong>7. Cadmium</strong></td>
<td>Pulmonary: Pulmonary edema; emphysema</td>
</tr>
<tr>
<td></td>
<td>GI: Gastroenteritis</td>
</tr>
<tr>
<td></td>
<td>Renal: Proteinuria; hypertension</td>
</tr>
<tr>
<td></td>
<td>Skeletal: Osteomalacia; fractures</td>
</tr>
<tr>
<td></td>
<td>CNS: Anosmia</td>
</tr>
<tr>
<td><strong>8. Benzo[a]pyrene (a PAH)</strong></td>
<td>Lung and Skin Cancer</td>
</tr>
<tr>
<td><strong>9. Polycyclic aromatic hydrocarbons (PAHs)</strong></td>
<td>Cancer: skin, lung, bladder, GI</td>
</tr>
<tr>
<td><strong>10. Benzo[b]fluoranthene</strong></td>
<td>Skin Cancer</td>
</tr>
<tr>
<td><strong>11. Chloroform</strong></td>
<td>CNS: Dizziness, headaches</td>
</tr>
<tr>
<td></td>
<td>Skin: sores</td>
</tr>
<tr>
<td></td>
<td>Cancer</td>
</tr>
<tr>
<td><strong>13. Aroclor 1254</strong></td>
<td>See PCBs</td>
</tr>
<tr>
<td></td>
<td>Chemical</td>
</tr>
<tr>
<td>---</td>
<td>------------</td>
</tr>
<tr>
<td>14.</td>
<td>Aroclor 1260</td>
</tr>
</tbody>
</table>
| 15. | Trichloroethylene | CNS: Dizziness, headache, facial numbness, ataxia, apnea, coma; hearing loss and speech delays in children  
|   |            | Skin: Flushing and rashes                                   |
|   |            | Cardiac: Impaired heart function                             |
| 16. | Dibenzo[a,h]anthracene | See PAHs                                                     |
| 17. | Dieldrin   | CNS: Headache, dizziness, irritability, seizures, coma, muscle twitching |
| 18. | Chromium 6+ | GI: Hemorrhage, vomiting  
|   |            | Skin: Dermatitis                                             |
|   |            | Nose: Nasal mucosa ulceration; nasal septum perforation      |
|   |            | Kidney: Renal tubular necrosis                               |
|   |            | Pulmonary: Lung cancer                                       |
| 19. | Chlordane  | CNS: Confusion, weakness, seizures                           |
|   |            | GI: Vomiting, diarrhea, ? hepatotoxicity                     |
| 20. | Hexachlorobutadiene | No studies looking at effects in humans                      |
### Table 8. Equation for Calculating Chemical Intakes

\[
\text{Intake} = C \times \frac{CR \times EFD}{BW} \times \frac{1}{AT}
\]

Where:

- **I** = Intake; the amount of chemical at the exchange boundary (mg/kg body weight-day)

Chemical-related variable

- **C** = chemical concentration; the average concentration contracted over the exposure period (mg/liter water)

Variables that describe the exposed population

- **CR** = contact rate; the amount of contaminated medium contacted per unit time or event (liters/day)

- **EFD** = exposure frequency and duration; describes how long and how often exposure occurs. Often calculated using two terms EF and ED
  
  - **EF** = Exposure frequency (days/year)
  
  - **ED** = exposure duration (years)

Assessment-determined variable

- **BW** = body weight; the average body weight (kg) over the exposure period

- **AT** = averaging time; period over which exposure is averaged (days)
Table 9. Differences in Exposures between Children and Adults

| • Exposures prior to conception affecting the fetus |
| • Multiple routes of exposure  
  Food, water, direct inhalation, contact  
  “Take-home” exposures from parents’/caregivers’ occupations |
| • Developmental changes in physical location of children  
  Hospital exposure in infancy - Neonatal intensive care unit  
  Preambulatory infants – more time in a single environment  
  Crawling infants – exposure to various floor and ground surfaces  
  School-age children – exposures at school  
  Adolescents – exposures at jobs and extracurricular activities |
| • Breathing Zones closer to the floor |
| • Higher Metabolic Rates  
  Greater oxygen consumption |
| • Higher Consumption of Food and Water per body weight |
| • Consumption of Different Types of Food  
  Infants limited to formula or breast milk  
  Consumption of more processed foods |
| • Behavior Developmental Patterns Influencing Exposures  
  Nonambulatory children cannot remove themselves from toxic environment  
  Toddlers with oral exploratory behaviors (hand-to-mouth)  
  Older children and adolescents don’t consider immediate cause and effect of potentially dangerous situations |
<table>
<thead>
<tr>
<th>Table 10. Foods Most Commonly Eaten By Infants</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Milk, nonfat solids</td>
</tr>
<tr>
<td>• Milk sugar (lactose)</td>
</tr>
<tr>
<td>• Apple juice</td>
</tr>
<tr>
<td>• Apples, fresh</td>
</tr>
<tr>
<td>• Bananas, fresh</td>
</tr>
<tr>
<td>• Rice, milled</td>
</tr>
<tr>
<td>• Orange juice</td>
</tr>
<tr>
<td>• Pears, fresh</td>
</tr>
<tr>
<td>• Milk, fat, solids</td>
</tr>
<tr>
<td>• Peaches, fresh</td>
</tr>
<tr>
<td>• Carrots</td>
</tr>
<tr>
<td>• Beef, lean</td>
</tr>
<tr>
<td>• Peas, succulent, garden</td>
</tr>
<tr>
<td>• Beans, succulent, garden</td>
</tr>
<tr>
<td>• Oats</td>
</tr>
<tr>
<td>• Soybean oil</td>
</tr>
<tr>
<td>• Coconut oil</td>
</tr>
<tr>
<td>• Wheat flour</td>
</tr>
</tbody>
</table>

Table 11. Calculation of Toxicity Values for Noncancerous Effects

1. \( \text{RfD} = \frac{\text{NOAEL}}{\text{UF}} \)
   Where:
   RfD = Reference Dose (when used without other modifiers, RfD generally refers to chronic reference dose)
   NOAEL = No-Observed-Adverse-Effect-Level
   UF = Uncertainty Factor (See Table 12)

2. \( \text{Noncancer Hazard Quotient} = \frac{\text{ED}}{\text{RfD}} \)
   Where:
   ED = Exposure dose (or chronic daily intakes CDIs)
   RfD = Reference dose
   ED and RfD are expressed in the same units and represent the same exposure period (i.e. chronic, subchronic, or shorter-term)

3. \( \text{Noncancer Hazard Index} = \frac{E_1}{\text{RfD}_1} + \frac{E_2}{\text{RfD}_2} + \ldots + \frac{E_i}{\text{RfD}_i} \)
   Where:
   \( E_i \) = exposure level (or intake) for the \( i^{th} \) toxicant;
   \( \text{RfD}_i \) = reference dose for the \( i^{th} \) toxicant; and
   E and RfD are expressed in the same units and represent the same exposure period (i.e. chronic, subchronic, or shorter-term)
Table 12. Uncertainty Factors/Safety Factors Used to Establish Reference Doses or Acceptable Exposures of Chemicals

<table>
<thead>
<tr>
<th>Uncertainty Factor</th>
<th>Criteria for Application</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Used to account for variation in the general population and intended to protect sensitive subpopulations like children</td>
</tr>
<tr>
<td>10</td>
<td>Used when extrapolating data from animals to humans to account for the interspecies variability</td>
</tr>
<tr>
<td>10</td>
<td>Used when a NOAEL derived from a subchronic instead of a chronic study is used as the basis for a chronic RfD</td>
</tr>
<tr>
<td>10</td>
<td>Used when a LOAEL is used instead of a NOAEL to account for the uncertainty associated with extrapolating from LOAELs to NOAELs</td>
</tr>
<tr>
<td>0 to 10 MF or modifying factor</td>
<td>Used to reflect a qualitative professional assessment of additional uncertainties in the study and database for the chemical not explicitly addressed by the other uncertainty factors. The default value for the MF is 1.</td>
</tr>
</tbody>
</table>

LOAEL – Lowest-Observed-Adverse-Effect Level
NOAEL – No-Observed-Adverse-Effect Level

**Table 13. EPA Weight-of-Evidence Classification System for Carcinogenicity**

<table>
<thead>
<tr>
<th>Group</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Human carcinogen</td>
</tr>
<tr>
<td>B1 or B2</td>
<td>Probably human carcinogen</td>
</tr>
<tr>
<td></td>
<td>B1 indicates that limited human data are available.</td>
</tr>
<tr>
<td></td>
<td>B2 indicates sufficient evidence in animals and inadequate or no evidence in humans</td>
</tr>
<tr>
<td>C</td>
<td>Possible human carcinogen</td>
</tr>
<tr>
<td>D</td>
<td>Not classifiable as to human carcinogenicity</td>
</tr>
<tr>
<td>E</td>
<td>Evidence for noncarcinogenicity for humans</td>
</tr>
</tbody>
</table>
Table 14. Calculation of Toxicity Values for Carcinogenic Effects

<table>
<thead>
<tr>
<th>Linear Low-Dose Cancer Risk Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk = CDI x SF</td>
</tr>
<tr>
<td>Where:</td>
</tr>
<tr>
<td>Risk = a unitless probability (e.g. 2 x 10^{-5}) of an individual developing cancer</td>
</tr>
<tr>
<td>CDI = Chronic daily intake averaged over 70 years (mg/kg-day); and</td>
</tr>
<tr>
<td>SF = slope factor, expressed in (mg/kg-day)^{-1}</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>One-Hit Equation for High Carcinogenic Risk Levels</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk = 1 – exp (-CDI x SF)</td>
</tr>
<tr>
<td>Where:</td>
</tr>
<tr>
<td>Risk = a unitless probability (e.g. 2 x 10^{-5}) of an individual developing cancer</td>
</tr>
<tr>
<td>Exp = the exponential;</td>
</tr>
<tr>
<td>CDI= Chronic daily intake averaged over 70 years (mg/kg-day)</td>
</tr>
<tr>
<td>SF = slope factor, expressed in (mg/kg-day)^{-1}</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 15. Differences in Biologic Environments Between Children and Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ABSORPTION:</strong></td>
</tr>
<tr>
<td>• Transplacental absorption of toxic chemicals to the fetus</td>
</tr>
<tr>
<td>• Greater percutaneous absorption of chemicals in infants</td>
</tr>
<tr>
<td>Larger surface-to-body mass ratio in infants</td>
</tr>
<tr>
<td>Less keratinized skin</td>
</tr>
<tr>
<td>• Increased absorption of certain chemicals</td>
</tr>
<tr>
<td>Low acidity levels in the GI tract</td>
</tr>
<tr>
<td>Differential absorption of chemicals in the GI tract</td>
</tr>
<tr>
<td><strong>DISTRIBUTION:</strong></td>
</tr>
<tr>
<td>• Differential tissue distribution of chemicals in the body</td>
</tr>
<tr>
<td><strong>METABOLISM:</strong></td>
</tr>
<tr>
<td>• Differential ability to activate or detoxify substances</td>
</tr>
<tr>
<td>Immature metabolic enzyme systems</td>
</tr>
<tr>
<td>Genetic control of enzyme systems</td>
</tr>
<tr>
<td><strong>EXCRETION:</strong></td>
</tr>
<tr>
<td>• Low renal clearance in infancy</td>
</tr>
<tr>
<td><strong>TARGET ORGAN SUSCEPTIBILITY:</strong></td>
</tr>
<tr>
<td>• Growth and differentiation of organs</td>
</tr>
<tr>
<td>Increased susceptibility to environmental toxicants during cell proliferation</td>
</tr>
<tr>
<td>High lipid content of some organs</td>
</tr>
<tr>
<td>Hormonal control of organ differentiation</td>
</tr>
<tr>
<td>Prolonged periods of organ development increasing vulnerability</td>
</tr>
<tr>
<td><strong>MORE FUTURE YEARS OF LIFE:</strong></td>
</tr>
<tr>
<td>• Latency effect for development of diseases</td>
</tr>
</tbody>
</table>
Table 16. Example of Risk Characterization for Radon.

### Radon Risk Evaluation Chart if You Smoke

<table>
<thead>
<tr>
<th>Radon Level</th>
<th>If 1,000 People Who Smoked Were Exposed to This Level Over a Lifetime...</th>
<th>The risk of cancer from radon exposure compares to...</th>
<th>What To Do: STOP SMOKING and...</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 pCi/L</td>
<td>About 135 people could get lung cancer</td>
<td>&gt; 100 times the risk of drowning</td>
<td>Fix your home</td>
</tr>
<tr>
<td>10 pCi/L</td>
<td>About 71 people could get lung cancer</td>
<td>&gt; 100 times the risk of dying in a home fire</td>
<td>Fix your home</td>
</tr>
<tr>
<td>8 pCi/L</td>
<td>About 57 people could get lung cancer</td>
<td></td>
<td>Fix your home</td>
</tr>
<tr>
<td>4 pCi/L</td>
<td>About 29 people could get lung cancer</td>
<td>&gt; 100 times the risk of dying in an airplane crash</td>
<td>Fix your home</td>
</tr>
<tr>
<td>2 pCi/L</td>
<td>About 15 people could get lung cancer</td>
<td>&gt; 2 times the risk of dying in a car crash</td>
<td>Consider fixing between 2 and 4 pCi/L</td>
</tr>
<tr>
<td>1.3 pCi/L</td>
<td>About 9 people could get lung cancer</td>
<td>(Average indoor radon level)</td>
<td>(Reducing radon levels below 2 pCi/L is difficult)</td>
</tr>
<tr>
<td>0.4 pCi/L</td>
<td>About 3 people could get lung cancer</td>
<td>(Average outdoor radon level)</td>
<td></td>
</tr>
</tbody>
</table>

*pCi/L: picocuries per liter. If you are a former smoker, your risk might be lower.

### Radon Risk Evaluation Chart if You Have Never Smoked

<table>
<thead>
<tr>
<th>Radon Level</th>
<th>If 1,000 People Who Never Smoked Were Exposed to This Level Over a Lifetime...</th>
<th>The risk of cancer from radon exposure compares to...</th>
<th>What To Do:</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 pCi/L</td>
<td>About 8 people could get lung cancer</td>
<td>&gt; The risk of being killed in a violent crime</td>
<td>Fix your home</td>
</tr>
<tr>
<td>10 pCi/L</td>
<td>About 4 people could get lung cancer</td>
<td></td>
<td>Fix your home</td>
</tr>
<tr>
<td>8 pCi/L</td>
<td>About 3 people could get lung cancer</td>
<td>&gt; 10 times the risk of dying in an airplane crash</td>
<td>Fix your home</td>
</tr>
<tr>
<td>4 pCi/L</td>
<td>About 2 people could get lung cancer</td>
<td>&gt; The risk of drowning</td>
<td>Fix your home</td>
</tr>
<tr>
<td>2 pCi/L</td>
<td>About 1 person could get lung cancer</td>
<td>The risk of dying in a home fire</td>
<td>Consider fixing between 2 and 4 pCi/L</td>
</tr>
<tr>
<td>1.3 pCi/L</td>
<td>Less than 1 person could get lung cancer</td>
<td>(Average indoor radon level)</td>
<td>(Reducing radon levels below 2 pCi/L is difficult)</td>
</tr>
<tr>
<td>0.4 pCi/L</td>
<td>Less than 1 person could get lung cancer</td>
<td>(Average outdoor radon level)</td>
<td></td>
</tr>
</tbody>
</table>

*pCi/L: picocuries per liter. If you are a former smoker, your risk might be higher.

<table>
<thead>
<tr>
<th>Table 17. Weaknesses Associated with the Risk Assessment Process</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>• EXTRAPOLATION OF DATA</strong></td>
</tr>
<tr>
<td>Interspecies extrapolation (animal studies to human)</td>
</tr>
<tr>
<td>High dose region on a dose-response curve to a low dose region</td>
</tr>
<tr>
<td>Exposure routes in test animals to human exposure data</td>
</tr>
<tr>
<td>Continuous static exposure in experimental environment to uneven, discontinuous human exposures</td>
</tr>
<tr>
<td><strong>• LIMITATIONS OF INVESTIGATIONAL STUDIES</strong></td>
</tr>
<tr>
<td>Inappropriate studies for toxicity data</td>
</tr>
<tr>
<td>Limitations of epidemiologic studies</td>
</tr>
<tr>
<td><strong>• VARIATIONS IN EXPOSURE ASSESSMENT</strong></td>
</tr>
<tr>
<td>Incomplete sampling techniques</td>
</tr>
<tr>
<td>Use of standard intakes for average adult</td>
</tr>
<tr>
<td>Differences in experimental and actual exposure conditions</td>
</tr>
<tr>
<td>Level, frequency, duration, and route of exposure</td>
</tr>
<tr>
<td>Inability to quantitate source of exposure due to contamination</td>
</tr>
<tr>
<td><strong>• LIMITATIONS OF CALCULATED TOXICITY VALUES</strong></td>
</tr>
<tr>
<td>Use of uncertainty factors</td>
</tr>
<tr>
<td>Use of upper confidence interval</td>
</tr>
<tr>
<td>Selection of extrapolation models for dose-response curves</td>
</tr>
<tr>
<td>Risks due to multiple exposures</td>
</tr>
<tr>
<td><strong>• DIFFERING MECHANISMS OF ACTION FOR CARCINOGENESIS</strong></td>
</tr>
<tr>
<td>Different risk for genotoxic versus epigenetic carcinogens</td>
</tr>
<tr>
<td><strong>• VARIABILITIES IN POPULATIONS</strong></td>
</tr>
<tr>
<td><strong>• USE OF APPROPRIATE ASSUMPTIONS</strong></td>
</tr>
<tr>
<td>Conservative estimate of risk may result in very imprecise risk value</td>
</tr>
<tr>
<td>Overestimation of risk</td>
</tr>
</tbody>
</table>
Table 18. Principles for Risk Characterization

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Risk characterization should be a <em>decision-driven</em> activity, directed toward informing choices and solving problems.</td>
</tr>
<tr>
<td>2.</td>
<td>Coping with a risk situation requires a broad understanding of the relevant losses, harms, or consequences to the interested and affected parties.</td>
</tr>
<tr>
<td>3.</td>
<td>Risk characterization is the outcome of an <em>analytic-deliberative process</em>. Its success depends critically on systematic analysis that is appropriate to the problem, responds to the needs of the interested and affected parties, and treats uncertainties of importance to the decision problem in a comprehensible way.</td>
</tr>
<tr>
<td>4.</td>
<td>Those responsible for a risk characterization should begin by developing a provisional <em>diagnosis of the decision situation</em> so that they can better match the analytic-deliberative process leading to the characterization to the needs of the decision, particularly in terms of level and intensity of effort and representation of parties.</td>
</tr>
<tr>
<td>5.</td>
<td>The analytic-deliberative process leading to a risk characterization should include early and explicit attention to <em>problem formulation</em>; representation of the spectrum of interested and affected parties at this early stage is imperative.</td>
</tr>
<tr>
<td>6.</td>
<td>The analytic-deliberative process should be mutual and recursive. Analysis and deliberation are complementary and must be integrated throughout the process leading to risk characterization: deliberation frames analysis, analysis informs deliberation, and the process benefits from feedback between the two.</td>
</tr>
<tr>
<td>7.</td>
<td>Each organization responsible for making risk decisions should work to build organizational capability to conform to the principles of sound risk characterization. At a minimum, it should pay attention to organizational changes and staff training efforts that might be required, to ways of improving practice by learning from experience, and to both costs and benefits in terms of the organization’s mission and budget.</td>
</tr>
</tbody>
</table>

Table 19. Specific Questions for Pediatric Toxicologic Profiles–Children’s Susceptibility

- What health effects are observed in children, in immature animals, and in adults who were exposed as children?
- Is any developmental process altered by the toxicant?
- Are pharmacokinetics different in children? Are there appropriate physiologically based pharmacokinetic models?
- Is metabolism different in children? Is there placental metabolism?
- Are pharmacodynamics different in children?
- Are biomarkers of exposure or effect validated in children or in adults who were exposed as children? Is so, are these biomarkers unique to children?
- Are there interactions with other chemicals in children? If so, are these interactions unique to children?
- Does the susceptibility of children differ from that of adults?
- Are there pediatric-specific methods for reducing peak absorption following exposure, reducing body burden, or interfering with the mechanism of action for toxic effects? Are any methods used in adults contraindicated in children? Have any methods been validated in children?
- How can parental exposure affect children?
- Is there transfer into breast milk?
- Is there storage in maternal tissues? Is there mobilization during pregnancy or lactation?
- Do the chemical or its metabolites cross the placenta, preferentially accumulate on the fetal side of the placenta, or indirectly affect the fetus?
- Are children exposed? Are there measurements of exposure or body burden?
- Are there unique exposure pathways for children?
- Are children different in their weight-adjusted intake of the toxicant?
- Could adolescents be exposed occupationally?
- Are the parents’ work clothes likely to be a source of exposure to children?
- Is the exhaled breath of occupationally exposed parents likely to be a source of exposures?
- Are there any childhood-specific means to decrease exposure?

Table 20. Developmental and Reproductive Toxicants added to the U.S. Toxic Release Inventory (TRI) in 1994. – Selected Examples

<table>
<thead>
<tr>
<th>Chemical Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>2,4-DP</td>
</tr>
<tr>
<td>Abamectin</td>
</tr>
<tr>
<td>Amitraz</td>
</tr>
<tr>
<td>Bifenthrin</td>
</tr>
<tr>
<td>Bromoxynil</td>
</tr>
<tr>
<td>Chlorsulfuron</td>
</tr>
<tr>
<td>Cyanazine</td>
</tr>
<tr>
<td>Diazinon</td>
</tr>
<tr>
<td>Diclofop methyl</td>
</tr>
<tr>
<td>Dimethoate</td>
</tr>
<tr>
<td>Disodium cyanodithiomidocarbamate</td>
</tr>
<tr>
<td>Diuron</td>
</tr>
<tr>
<td>Fluorouracil</td>
</tr>
<tr>
<td>Hydramethylnon</td>
</tr>
<tr>
<td>Linuron</td>
</tr>
<tr>
<td>Lithium carbonate</td>
</tr>
<tr>
<td>Nicotine and salts</td>
</tr>
<tr>
<td>Oxydiazon</td>
</tr>
<tr>
<td>Pentobarbital sodium</td>
</tr>
<tr>
<td>Phenytoin</td>
</tr>
<tr>
<td>Potassium dimethyldithiocarbamate</td>
</tr>
<tr>
<td>Propachlor</td>
</tr>
<tr>
<td>Sodium nitrate</td>
</tr>
<tr>
<td>Sulprofos</td>
</tr>
<tr>
<td>Terbacil</td>
</tr>
<tr>
<td>Tributylin methacrylate</td>
</tr>
<tr>
<td>Vinclozolin</td>
</tr>
</tbody>
</table>

Adapted from Goldman LR. New approaches for assessing the etiology and risks of developmental abnormalities from chemical exposure. Reproductive Toxicology 1997;11:443-451. Reproduced with permission from Elsevier Science
<table>
<thead>
<tr>
<th>SUBSTANCE</th>
<th>SPECIMEN</th>
<th>SUBSTANCE MONITORED</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arsenic</td>
<td>Urine</td>
<td>Arsenic</td>
</tr>
<tr>
<td>Benzene</td>
<td>Urine</td>
<td>Phenol</td>
</tr>
<tr>
<td></td>
<td>Exhaled Air</td>
<td>Benzene</td>
</tr>
<tr>
<td>Cadmium</td>
<td>Urine</td>
<td>β-2 microglobulin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Metallothionein</td>
</tr>
<tr>
<td>Dieldrin</td>
<td>Blood</td>
<td>Dieldrin</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>Urine</td>
<td>Formic acid</td>
</tr>
<tr>
<td>Lead</td>
<td>Blood</td>
<td>Lead</td>
</tr>
<tr>
<td>Mercury</td>
<td>Urine</td>
<td>Mercury</td>
</tr>
<tr>
<td>Methylene Chloride</td>
<td>Exhaled Air</td>
<td>Carbon monoxide</td>
</tr>
<tr>
<td></td>
<td>Blood</td>
<td>Carboxyhemoglobin</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Organophosphates</td>
<td>Blood</td>
<td>RBC Cholinesterase</td>
</tr>
<tr>
<td></td>
<td>Urine</td>
<td>Plasma cholinesterase</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Alkyl-phosphate metabolites of particular substance</td>
</tr>
<tr>
<td>PCB</td>
<td>Blood</td>
<td>PCB</td>
</tr>
<tr>
<td>Tobacco Smoke</td>
<td>Blood</td>
<td>Cotinine</td>
</tr>
<tr>
<td></td>
<td>Urine</td>
<td></td>
</tr>
<tr>
<td>Toluene</td>
<td>Urine</td>
<td>Hippuric Acid</td>
</tr>
<tr>
<td>Trichloroethylene</td>
<td>Urine</td>
<td>Trichloroacetic Acid</td>
</tr>
</tbody>
</table>
Figure 1. Risk Assessment Process

HAZARD IDENTIFICATION
What health problems are caused by the chemical?

EXPOSURE ASSESSMENT
- What people are exposed to the chemical?
- How much of the chemical are people exposed to?

TOXICITY ASSESSMENT
- What are the health problems of different exposures?

RISK CHARACTERIZATION
- What is the risk of health problems in the exposed population?
Figure 2. Exposure Assessment Process
Figure 3. Exposure Pathways
Figure 4. Steps in Toxicity Assessment

<table>
<thead>
<tr>
<th>Step 1</th>
<th>Gather qualitative and quantitative toxicity information for substances being evaluated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 2</td>
<td>Identify exposure periods for which toxicity values are necessary</td>
</tr>
<tr>
<td>Step 3</td>
<td>Determine toxicity values for noncarcinogenic effects</td>
</tr>
<tr>
<td>Step 4</td>
<td>Determine toxicity values for carcinogenic effects</td>
</tr>
<tr>
<td>Step 5</td>
<td>Summarize Toxicity Information</td>
</tr>
</tbody>
</table>

Figure 5. Steps in Risk Characterization

**Step 1: Organize Outputs of Exposure and Toxicity Assessments**
- Exposure Duration
- Absorption Adjustments
- Consistency Check

**Step 2: Quantify Pathway Risks for Each Substance**
Estimate:
- Cancer Risk
- Noncancer Hazard Quotient
For Each Pathway, Calculate:
- Total Cancer Risk
- Noncancer Hazard Index

**Step 3: Combine Risks Across Pathways that affect the same individual(s) over the same time periods**
- Sum Cancer Risks
- Sum Hazard Indices

**Step 4: Assess and Present Uncertainty**
- Site-Specific Factors
- Toxicity Assessment Factors

**Step 5: Consider Site-Specific Health of Exposure Studies**
- Compare Adequate Studies with Results of Risk Assessment

**Step 6: Summarize Results of the Baseline Risk Assessment**

Figure 6. Child-Centered Risk Assessment Paradigm

- What is the child exposed to?
- How is the child exposed and at what stage of development?
- Are these exposures of particular concern for children on the basis of available data?
- Are children more or less sensitive than adults?
- What are the transgenerational effects?
- What are the effects of acute exposures or long-term, low-level exposure?
- What are the effects of multiple and cumulative exposures?
- What are the delayed effects?
- What additional information/research is needed to adequately characterize health concerns?
Figure 7. Classes of Biologic Markers.

Indicates progressing, if it occurs, to the next class of marker. Indicates that individual susceptibility influences the rates of progress, as do other variables. Biologic markers represent a continuum of changes and the classification of change may not always be distinct. From National Research Council; Biologic Markers in Productive Toxicology. Washington, DC: National Academy Press, 1989. Reproduced with permission.
PART II RISK COMMUNICATION

Case Scenario: The parents of elementary school children in a small town are concerned about the presence of toxic wastes on the school playground and the pond adjacent to the school. There was some recent information released in the media that 40 years ago the playground was a former dumping site for surrounding chemical plants in the area. Many of the children have attention deficit hyperactivity disorder, learning disorders, and asthma. In the last year, two children have been diagnosed with leukemia and one child with a brain tumor.

The hazard assessment reveal PCBs and arsenic in the soil, chlorinated hydrocarbons, PCBs, and benzene in the pond.

• How should this information be communicated to the parents, school, and community?
• Is there any risk of disease to these children after their exposure on the playground, near the pond, and in the school?
• What is risk?
• Did the soil and water contaminants cause the children’s leukemia and brain tumor?
• Are the parents’ fears justified?
• What are general principles for communicating risk?
• What is the best manner for the pediatric health care provider to participate in explaining the findings to the parents of the school children?

I. INTRODUCTION

Reviewing the case scenario, once an assessment of potential hazards has been performed on the playground, it is important for the resulting information to be conveyed to parents, school officials, and the community in an accurate, ethical, and understandable format so that unnecessary fear, anxiety, and mistrust are not created. However, the process of communicating information on environmental hazards must entail more than a one-way communication from “expert” to “lay person” of sophisticated statistics and probabilities of disease and death. It is essential for the public to become involved in the communication process-- to come to a mutual understanding of the nature and extent of the potential health risks-- and to empower them to make rational decisions about the circumstances for themselves, their children, and their community. The comprehensive process of purposeful exchange of information about health or environmental risks between interested parties is known as risk communication. Interested parties may include government agencies, corporations and industry groups, the media, scientists, professional organizations, public interest groups, and individual citizens.11
Because they are usually viewed by parents as credible and trustworthy, pediatric health care providers are in a strategic and important position as communicators for information on various environmental issues. However, they need to be skilled in assessing and communicating information about environmental health risks to avoid creating unnecessary stress and fear for their patients, parents, and themselves. Furthermore, they need to be knowledgeable about available resources that parents/patients may obtain reliable information when they don’t know about specific issues or details. In the case scenario, a wide spectrum of questions will evolve concerning general information, specific chemicals, diseases, and future of the community: How safe is the playground? What is the risk of long-term effects of the pollutants in the soil and water? Are the PCBs found in the water and soil responsible for my child’s learning problems in school? Is my child’s leukemia related to the arsenic in the playground soil? Should we move our children to a different school because of the results of the assessment? Should our family relocate to a different city? These types of questions are very difficult to answer due to the lack of specific exposure and toxicity data in children as discussed in the previous section. It is difficult to project the future and make sound recommendations when there is so much uncertainty. In fact, the PHCP probably will not be able to answer every question. However, understanding basic principles about the process of risk communication can help the PHCP, patient, and/or parent select appropriate ways of understanding and controlling some of the health risks associated with the environmental exposure.

II. BACKGROUND AND DEFINITION OF RISK COMMUNICATION

Risk communication has received a great deal of attention in the last 10 years because it has become clear that without good communication, the processes of risk assessment and risk management are somewhat futile and ineffectual. It is a complex process which has begun to be understood even better in the last decade because of the increasing social science literature that has addressed some of its fundamental components. However, communicating knowledge about
avoiding hazardous situations probably has existed since the origins of structured risk analysis in
the Babylonian era over 5000 years ago and paralleled the risks that individuals have faced
throughout human history. In the mid- and late 1980’s, numerous conferences, articles, and
books emerged that dealt with the subject of risk communication.8,10,12,16,34,37,39,50 The literature
on risk communication encompasses such diverse fields as cognitive psychology, social
psychology, consumer behavior, marketing, advertising, economics, mass communications,
linguistics, anthropology, decision science, sociology, political science, health education,
behavioral medicine, public health, environmental health, law, and philosophy. The inclusion of
these numerous and diverse fields reflects the broad scope of the discipline of risk
communication.

In 1983, the National Research Council completed a study of managing risk which
focused on improving risk assessment and risk decisions within the government—Risk
Assessment in the Federal Government: Managing the Process.30 Risk management is the term
used to describe the process of taking risk assessment results and arriving at a decision of action
or inaction. (Table 4) Risk management strategies or options can be broadly classified as
regulatory, nonregulatory, economic, advisory, or technological. Risk assessment and risk
management are two distinct, though, closely interlinked processes. (Figure 8) However, it
became evident that these two processes could not be successful without risk communication.
Growing concern that risk communication was becoming a major problem led to the chartering
of a National Research Council committee to examine possibilities for improving social and
personal choices on technological issues by improving risk communication. The NRC
developed the Committee on Risk Perception and Communication which included members with
extensive experiences analyzing, managing, and communicating about diverse risks, including
those from radiation, chemicals, drugs, disease, and consumer products. These members had
experience in diverse settings including industry, the federal government, the mass media, and
environmental groups. The committee exhibited diverse disciplinary backgrounds including physical and social sciences, law, journalism, public health, and communications research in an effort by the NRC to achieve a balance of perspectives on all these dimensions. Their report *Improving Risk Communication*, published in 1989, provides a comprehensive discussion on the process of risk communication, the content of risk messages, and ways to improve risk communication in the service of public understanding and better-informed individual and social choice.31

This explosion of literature reflects increased attention not only by the federal government but by other organizations as well, to the need of informing the general public about the nature of the health, safety, and environmental risks associated with personal and societal choices.31 The concern with informing the public has several motivating sources including: 1.) a requirement for or desire by government to inform; 2.) a desire by government or industry officials to overcome opposition to decisions; 3.) a desire to share power between government and public groups and 4.) a desire to develop effective alternatives to direct regulatory control.31 Risk communication can take place in a variety of forms, ranging from warning labels on consumer products to interactions among government officials, industry representatives, the media, and the public.

At the federal level, the Environmental Protection Agency (EPA) has been the strongest marketer of the concept of risk communication. The EPA has elevated the concept of risk communication to a strategic level of importance in both its regulatory activities and its research agenda.34 Industries that are regulated by the EPA also see risk communication as a key policy and management issue. In the public health arena, risk communication is focused on educating the public about health risks. Health officials focus on communicating risk of health problems, such as AIDS and teenage pregnancy, to target populations. Risk communication is an important component of medical practice, including the doctor-patient relationship. Because of the
increase of chronic diseases both in children and adults, as well as the uncertainty associated with the risk factors and course of these diseases, risk communication in medicine has attained a more central and visible role.\textsuperscript{33}

What is the definition of risk communication? The term will have different meanings to different users. To many technical experts and scientists, it means the development and delivery of a one-way message that describes or characterizes hazards, risks, or risk-reducing actions that is addressed to “nonexperts” or the general public. This definition equates risk communication with the delivery of certain kinds of messages and its success in terms of merely “getting the message across.” Unfortunately, it also portrays the public as a passive, uninformed, and uninvolved receiver of information. Risk communication should be thought of as a type of “democratic dialogue.” Communication is an essential component of democratic societal decisions.\textsuperscript{31} Individuals, groups, and institutions should be allowed to express their concerns and viewpoints and listen to what other participants have to say. In turn, elected officials and public officials act in the interest of the public, sometimes adding their own opinions. Furthermore, it is important to emphasize that technological choices—i.e. whether a community should build a nuclear power plant for their electricity—are value-laden. Nonexperts’ interests, values, and concerns should be acknowledged just as the scientific knowledge about new advances in technology.

Thus, the definition of risk communication should be reflective of a more comprehensive process. The National Research Council’s report defines risk communication as:

“an interactive process of exchange of information and opinion among individuals, groups, and institutions. It involves multiple messages about the nature of risk and other messages, not strictly about risk, that express concerns, opinions, or reactions to risk messages or to legal and institutional arrangements for risk management...Risk communication is successful only to the extent that it raises the level of understanding of relevant issues or actions and satisfies those involved that they are adequately informed within the limits of available knowledge.” \textsuperscript{31}
Risk communication must be an integral component of risk management. (Figure 8) Although successful risk communication may not guarantee that risk management decisions will always maximize general welfare or provide no risk of harm to anyone, it should ensure that decision makers will understand what is known about the implications of the available options for the welfare of people. Successful risk communication does not necessarily lead to better decisions because risk communication is only part of the risk management process.

The goal of this section of the monograph is to provide a framework for understanding many general principles about risk communication; namely, the definition of risk, understanding risk perception and risk acceptability, and the multiple components of the risk communication process including how to explain risk. The scope of this subject is vast; thus, the purpose of this monograph is to highlight important and general concepts and processes. The goal is *not* to provide pediatric health care providers with a “how to” manual for risk communication. Principles that form the basis of successful risk communication, including the definition and perception of risk and the processes of communicating different messages, are based on scientific evidence and the collected experiences of individuals and organizations that have engaged in this process. It is then the hope that the PCHP will develop their own guidelines to fit their own unique positions. There are many excellent general and comprehensive references on risk communication which are highlighted in the last section *General Information Resources About Risk Communication.*

**III. WHAT IS RISK?**

In order to fully appreciate the processes of risk assessment and risk communication, it is important to understand the meaning of risk. Actually, the definition of risk is confusing and somewhat controversial. Furthermore, the choice of a particular definition can affect the outcome of various risk management decisions including allocation of resources for safety measures, distribution of political power in society, and anxiety and fear among parents for their
family’s well-being. The concept of risk is subtle and complex and no one definition of risk is suitable for all problems.\textsuperscript{20}

The Webster’s New World Dictionary defines risk as “the chance of injury, damage, or loss”.\textsuperscript{32} Many people use the word \textit{risk} inconsistently—sometimes using it to mean a hazardous activity (i.e. Where does this risk rank in the list?), sometimes to mean probability (i.e. What is the risk of death from exposure to this chemical?), sometimes to mean consequence (What is the risk of drinking the playground water?), and sometimes to mean the danger or threat associated with a hazardous activity or technology. (How great is the risk to the fetus if the mother drinks alcohol during pregnancy?)\textsuperscript{41} The risk assessment process attempts to quantitate risk by analyzing various hazards and determining the probabilities and consequences of adverse events. Developing quantitative knowledge is very important when assessing the risk of infants and children to environmental hazards. How much pesticides are children exposed to in their diets? What is the chance that children will develop asthma from various outdoor and indoor air pollutants? What is the risk of congenital abnormalities of infants born to mothers living near the playground? Unfortunately, communicating the answers to these questions is sometimes impossible because of the limitations of the risk assessment process described in the previous section.

The first important conceptual distinction to understand is that between hazard and risk. Hazard is an act or phenomenon posing potential harm to some person(s) or thing(s); the magnitude of the hazard is the amount of harm that might result, including the seriousness and the number of people exposed. Risk adds to the hazard and its magnitude the probability that the potential harm or undesirable consequence will be realized.\textsuperscript{31} One technical definition of risk is that it is the product of a measure of the size of the hazard and its probability of occurrence:

\text{Risk} = \text{Size of Hazard} \times \text{Probability of Occurrence.}
characterized by event probabilities and consequences are inadequate because they do not encompass subjective assessment techniques or intuitive risk assessments.\textsuperscript{42}

The subjectivity of risk is illustrated through the beliefs that there are many qualitative aspects of risk. The social science literature has contributed to our understanding of the multidimensionality of risk, especially risk perception. Subjectivity permeates risk assessments because judgments are used at many stages of the process including which endpoints or consequences to include in the analysis.\textsuperscript{42} The different ways that fatality risks can be measured as an endpoint to an analysis can make a particular technology or environmental problem look either more or less risky, and thus affect how the hazard is perceived and evaluated.\textsuperscript{8} (Table 22). Depending on the circumstances, these different expressions may strike a person as being more or less appropriate, more or less frightening, and more or less credible. Each way of summarizing death embodies its own set of values.\textsuperscript{8} For example, “reduction in life expectancy” treats deaths of young people more importantly than deaths of older people, who have less life expectancy to lose. Merely counting fatalities treats deaths of the old and young as equivalent. Using this measure also treats deaths from people who choose to engage in an activity by choice as equivalent to those who are exposed to an environmental hazard involuntarily. This comparison is not accurate or logical. To select one modality as an endpoint for assessing risk requires a value judgment concerning which deaths one considers most undesirable. This concept can be applied to other decisions regarding risk assessment in children that may be not as easily quantifiable; for example, is it more important to assess the long-term effects of PCBs on learning problems in young children by individual schools or look at the number of children with learning problems exposed to PCBs as a proportion of the total number of children in the country (a much smaller and different number)?

Problems in communicating about risks stem partly in the marked differences that exist between the two languages used to describe risk: the scientific and statistical language of experts
and the intuitively-grounded language of the public.35 (Table 23). Scientists, in general, define risks in the language and procedures of science itself—the nature of the harm that may occur, the probability that it will occur, and the number of people who may be affected.23 Most people seem less aware of the quantitative or probabilistic nature of a risk and more concerned with broader, qualitative attributes. A growing body of literature in the last 15 years on risk perception helps illustrate the values and other qualitative aspects involved in the evaluation of different qualities of hazards.

The differences between the two languages may constitute barriers to dialogue and understanding amongst groups of people. However, both languages for describing risk are necessary for effective risk communication. Successful risk communication attempts to break down these barriers and facilitate productive exchanges between these two languages.

**RISK ACCEPTABILITY AND RISK PERCEPTION**

Understanding the distinction between risk and risk acceptability is critical to successful risk communication. Even though the level of risk is related to risk acceptability, it is not a perfect correlation.8,9 There are two factors that affect the way people assess risk and evaluate acceptability which modify this correlation. First, the level of risk is only one among several variables that determines acceptability. Other factors that should be important in assessing risk are fairness, benefits, alternatives, control, and voluntariness as described in more detail below. Second, deciding what level of risk ought to be acceptable is not a technical question but a value question. People vary in their assessment of risk acceptability, weighing the various factors according to their own values, sense of risk, and stake in the outcome.9 Debates about risk may really be debates about accountability and control. Whose values and opinions will ultimately determine the outcome?

Research has demonstrated that the members of the public include qualitative and subjective attributes in their perception of risk. This is in contrast to the perception of risk held
by the scientific community and experts which tend to view risk as the probability of harm or expected mortality as characterized in risk assessments, an objective and rational view of the real risk. In the 1980s, several groups developed models that incorporated the value systems of individuals, peer groups and societies into risk communication theory. There appears to be general agreement that risks are viewed by individuals, peer groups and societies according to their perceived threat to familiar social relationship and practices, and not simply by numbers and statistics alone. The psychometric paradigm describes risk from a psychological perspective, emphasizing the various characteristics or dimensions which may be important in influencing risk perceptions. These theories hold that risk itself is deterministic in generating perceptions. In contrast, the cultural theory of risk holds that the characteristics of the perceiver, rather than the risk itself, are important to an understanding of risk perception. Finally, the social amplification of risk theory suggests a way to integrate the psychological and cultural aspects into a comprehensive framework accounting for the social, cultural, and individual characteristics that tend to magnify one risk over another.

Slovic described two dimensions that typically characterize people’s risk perceptions: 

*dread risk* and *unknown risk*. *Dread risk* is the extent of perceived catastrophic potential, threat to life, and lack of control (radiation leak into surrounding community) while *unknown risk* is the extent to which the risk associated with the hazard seems unobservable, unknown, new, and delayed in its harmful effects (long-term risk of low-level lead exposure during pregnancy on child’s neurological development). Peter Sandman developed an alternative risk equation—RISK = HAZARD + OUTRAGE—that takes into account qualitative aspects of risk that the traditional scientific perception of risk does not—RISK = HAZARD. These “outrage factors” are aspects of risk that scientists and technical experts tend to ignore or fail to acknowledge when relaying information and essential components of risk. Several factors that modulate risk perception are outlined below and shown in Table 24.
1. **Unfamiliar risks are less acceptable than familiar risks.** The most underestimated risks are those which people have encountered for long periods without experiencing the undesired event such as household accidents. Thus, the perceived riskiness of a hazardous waste facility may partly be a reflection of its unfamiliarity. Furthermore, emphasizing its similarity to industrial facilities that people are more familiar with can diminish the fear. In addition, detailed information about various aspects of the waste facility should reduce the fear level.

2. **Involuntary risks are less acceptable than voluntary risks.** Many studies show the acceptance of voluntary risks at one thousand times the level for involuntary risks.46 Helping to acknowledge the public’s involvement and power over various decisions regarding environmental hazards will lessen the fear and decrease the perception of increased risk. For example, actively involving the parents, school officials, and community in decisions regarding the playground clean-up should help allay fear and anxiety and decrease their perception of increased risks.

3. **Risks controlled by others are less acceptable than risks under one’s own control.** People tend to feel more comfortable with environmental risks they can do something about themselves, rather than having to rely on the government or other agencies to protect them. People need to know that they have some control over decisions in the entire risk management process.

4. **Undetectable risks are less acceptable than detectable risks.** Risks with effects that may take years to show up are more likely to be feared. A major component of the fear of carcinogenicity is its undetectability during its latency period when there are no manifestations of the hazard. This factor is an important quality when conveying long-term risks of medical problems and cancer to parents of young children. It is impossible to assure the proper clean-up of hazardous wastes and minimize pesticide exposure to limit the detectable risks of acute toxicity.
5. **Risks perceived as unfair are less acceptable than risks perceived as fair.** A coerced risk will always seem unfair. A community may have increased risk perception because their area was chosen by government officials as a hazardous waste facility, feeling they are getting stuck with the risk and getting little benefit. Part of the risk communication process, therefore, is to try to achieve equity by negotiating appropriate benefits to compensate a community. This factor explains partly why communities that depend on a particular industry for jobs sometimes see pollution from that industry as less risky.

6. **Risks that do not permit individual protective action are less acceptable than risks that do.** People prefer to know that there are things they can do as individuals to reduce the risk even of a low-probability risky event, even though it may neither be practical nor cost-effective.

7. **Dramatic and memorable risks are less acceptable than uninteresting and forgettable ones.** This principle is known as the “availability heuristic”: people judge an event as more likely or frequent if it is easy to imagine or recall.\(^{43}\) The historic legacy of environmental catastrophes such as Bhopal, India and Chernobyl has increased the public’s perception of chemical and radiation risks even greater. Furthermore, risks that receive extensive media attention are likely to be overestimated, while those that the media fails to popularize are not perceived as risky.

8. **Uncertain risks are less acceptable than certain risks.** This principle also has important implications for communicating environmental risks about children where there are so many uncertainties in the current knowledge. Basing decisions about you and your child’s or patient’s health on uncertain information arouses fear and anxiety. A parent’s question “Is it safe?” may not be able to be answered in a forthright manner. Acknowledging that the current information supports a possibility of risks, but its extent is uncertain, is the most appropriate approach.
9. **Artificial risks seem less acceptable than natural risks.** A natural risk caused by God is more acceptable than one caused by people. There appears to be less anger and fear about naturally occurring radon in homes than the high radon levels caused by uranium mills or other industrial sources.

10. **Risks that are not well understood by science are less acceptable than risks that are well understood by science.** Risks that scientists can explain to communities seem more acceptable than those about which scientists have to acknowledge a great deal of uncertainty.

11. **Risks to children are less acceptable than risks to adults.** People are more concerned about risks perceived to affect children in a disproportionate fashion.

   Other qualitative factors that may increase public concern and thus viewed as “more risky” include catastrophic potential, identifiable “victims”, lack of trust in responsible institutions, immoral activities, risk to future generations, unclear benefits, and irreversible effects. The greater the number and seriousness of these factors, the greater the likelihood of public concern about the risk, regardless of the scientific data.

   Extensive literature supports the notion that affect—the specific qualities of “goodness” or “badness” experienced as a feeling state—plays an important role in guiding human judgments and decisions of risk and benefit. Further understanding of this “affect heuristic” is essential for successful risk communication. In 1978 Fischhoff demonstrated that perception of risk and society’s response to risk was associated to the degree to which a hazard evoked feelings of dread in people. For example, activities associated with cancer are seen as riskier and more in need of regulation and change than activities associated with less dreaded forms of illness, injury, and death. These observations have been reproduced in numerous other studies.

   Judgments of risk and benefit are negatively correlated. Thus, for many hazards, the greater the perceived benefit, the lower the perceived risk and vice versa. Smoking, alcoholic beverages, and food additives, for example, tend to be perceived by scientific experts as very
high in risk and relatively low in benefit, whereas vaccines and antibiotics tend to be seen as high in benefit and relatively low in risk.\textsuperscript{15} A later study found that the inverse relationship between perceived risk and benefit of an activity (e.g. using pesticides) was linked to the strength of positive or negative affect associated with that activity.\textsuperscript{1} This result implies that people base their judgments of an activity or technology not only on what they think about it but also on what they feel about it. For example, if parents like the idea of removal of the contaminated playground soil, they are moved toward judging the risks of this activity as low and the benefits as high; if they dislike it, they will judge the risk as high and the benefits as low.

Providing information to people about risk also changes the perception of benefit and risk. For example, information stating that risk was low for some technology should lead to more positive overall affect which would, in turn, increase the perceived benefit. Finucane and others provide evidence to support this hypothesis.\textsuperscript{15} In their experiment, when information about nuclear power was provided that changed either the perceived risk or the perceived benefit, an affectively congruent but inverse effect was observed on the non-manipulated attribute as depicted in Figure 9. For example, providing information to people about the benefits provided by nuclear power as a source of energy decreased people’s perception of the risks of that technology. Other studies have found that this inverse relationship between affect and perception of risk is strengthened under time-pressure conditions; thus, limiting the use of analytic thought and enhancing the reliance on affect.\textsuperscript{15} Another study of toxicologists’ reactions toward various chemicals (benzene, passive cigarette smoke, dioxin in food, aspirin) on an affect scale (bad to good) also illustrated this inverse relationship even when they were asked to judge the degree of risk associated with a very small exposure to the chemical.\textsuperscript{45} One might expect that these scientists would judge the risk of very small exposures of all of these chemicals as uniformly low with little or no correlation to their ratings of affect. However, most of the
toxicologists exhibited a high correspondence between their judgments of affect and risk in judging chemical risks.

The fact that hazards may differ dramatically in their qualitative aspects helps to explain why certain technologies or environmental activities, such as nuclear power and landfills, evoke much more serious public opposition than other activities such as riding in an automobile, which causes significantly more fatalities. The risk perception research demonstrates that the equating of risks with different attributes is value laden, but also that the values adopted by this practice differ from those held by most people. The research also suggests that technical experts may not know the proper weighting of hazards to equate one with another without careful thought and analysis. Disregarding the values of people and reducing different kinds of hazard to a common quantifiable number (numbers of fatalities per year) promotes misunderstanding and conflict when communicating to the public and may engender mistrust of the expertise.

There is also concern that the practice of more sophisticated quantitative risk assessment is breeding fear and increasing the public’s perception of risk.\textsuperscript{36} Certainly, the practice of QRA has increased during the past 20 years as government and industry officials have tried to develop more effective ways to meet the public’s demands for a safer and healthier environment. However, as efforts have been expended to make our lives safer and healthier, some people have become more, rather than less, concerned about risk.\textsuperscript{40} National surveys in the United States, Canada, and France demonstrated that about 70\% of the public believe that “If a person is exposed to a chemical that can cause cancer, then that person will probably get cancer some day”.\textsuperscript{27,28} More than 50\% agreed that “there is no safe level of exposure to a cancer-causing chemical.” The public’s concern that any exposure to a carcinogen is likely to cause cancer is associated with their desire to avoid these chemicals and reduce the risks of exposure to them at any cost.\textsuperscript{36}
As discussed in the previous section, there are two models for quantifying risk—the threshold and nonthreshold models. In the threshold model, the No Observed Adverse Effect Level (NOAEL) is the threshold below which adverse effects cannot be observed. On the other hand, the nonthreshold models imply that there is a linear dose-response relationship and no exposure is without risk. It has been suggested that the use of a nonthreshold model to express risk in probabilistic terms leads to higher perceived risk than does the threshold model. Investigators have demonstrated that both college students and toxicologists judged risk to be greater if it was expressed in probabilistic terms. For example, based on animal studies, the probability that one will develop cancer from exposure to Chemical Y is about 1 chance in 100,000 as opposed to nonprobabilistic terms, where Chemical X has been observed to cause cancer in animals, but only at doses more than 100,000 times greater than what you will drink. A majority of respondents in both groups judged the risk expressed as about 1 chance in 100,000 to be greater.36

IV. THE PROCESS OF RISK COMMUNICATION

The process of risk communication must include understanding people’s perceptions of risk and their emotions and concerns; dealing with concerns for which some organization or agency has control and identifying how others will be managed; advising people of environmental and public health risk assessments in language that they understand; informing the public about current and proposed actions; and providing opportunities for public involvement in risk management decisions.2 With a general understanding of these issues, the pediatric health care provider can then apply some general principles about the process of risk communication in a much broader context. Risk communication must be a two-way process to not only discuss the technical information associated with the concerning hazards, but also to understand how to respond to the public’s fear and outrage and conduct the process sincerely with people’s interests
in mind. Thus, the process includes general principles about communication as well as specific issues when explaining risk.

**GENERAL PRINCIPLES ABOUT RISK COMMUNICATION**

The purpose of risk communication, therefore, is to inform and advise the public on health risk assessment information, and to then involve them in the risk management. As a result of this process, the public can better decide on actions they must take to protect their interests, and agencies can make effective risk management decisions. In most of these situations, the PHCP is neither the public nor the agency, but should be a neutral party. (It is important to understand also that the concept of “the public” as a target for risk communication is not a single entity; instead there are many “publics”, each with its own interests, needs, concerns, priorities, and preferences.)

Risk communication tasks can be organized into four general types according to the primary objective or the intended effect of the communication: 1.) information and education; 2.) behavior change and protective action; 3.) disaster warnings and emergency information; and 4.) joint problem solving and conflict resolution. These four types of tasks overlap substantially in real life situations but conceptually are different. All are important, to some degree, in the various situations where risk communication is needed.

**Basic Communication Strategies**

To start the process or risk communication, it is important to formulate a general approach using basic communication planning strategies as outlined below:

1. **Identify the intended audience.** As the communicator, it is important to identify who the audience will be—a patient, a parent, a group of community citizens, or public officials? Furthermore, one must identify the needs of the audience which may be different for different people. Do they have informational needs, emotional needs, personal interests? What are the audience’s concerns?
Examples include: How will it affect me? Can I trust the presenter? Is it fair? Does the presenter make sincere attempts to understand my concerns and needs? Specific knowledge of the intended audience allows risk messages to be customized and allows for appropriate and perhaps different channels of communication to be chosen (single presenter in a room, radio show announcements, etc.)

2. **Set realistic goals.** The communicator must set realistic and achievable goals for his/her communication messages. Examples include: To reassure the parents of school children that the playground soil is safe; to inform public officials that concentrations of inorganic arsenic in the river by the school are higher than expected; to persuade parents that they do not need to relocate their families due to findings of chemicals in the playground soil. All of these goals have different audiences or different purposes that will impact on specific risk messages should be constructed.

3. **Explore alternative ways to achieve the goal.** If the presenter’s goal is to persuade parents that there is no need to relocate from their current houses, small group meetings with the parents might be a better way to initiate the communication to hear and discuss their concerns, needs, and the uncertainties. Then, one could do a more formal presentation with specific technical information or distribute a brief written summary of the desired hazard information. Small group meetings with two-way discussions may be more effective in reassuring various sectors of the public than a more formal presentation to a large audience. These discussions can decrease the public’s uncertainty about the communicator’s interest and commitment to their needs, help establish rapport, and help to facilitate the exchange of information. On the other hand, if the goal is to inform
public officials about specific hazards, an alternative is to prepare a more technical written summary about the hazard in question using the framework of the risk characterization process.

4. **Formulate communication objectives and main points.** Determine beforehand the key messages that are important to achieving the communication goal. For example, if the communicator’s goal is to reassure a parent about the safety of the drinking water on the school playground because he/she believes it is safe based on review of sampling results and risk assessment, a primary objective or conclusion to state upfront is “The water is safe to drink.” Another key point is to tell the parent how you came to this conclusion by presenting supporting facts.

**Risk Communication Problems**

Understanding some of the major problems with risk communication will allow the PHCP to understand better the global process of risk communication. Risk communication problems arise from four sources: message problems, source problems, channel problems, and receiver problems. These problems are outlined in Table 26. Message problems arise from limitations of scientific risk assessments and the difficulty in communicating highly technical information to the public. Source problems include the limitations of risk communicators and risk assessment experts. These include lack of trust and credibility; limited understanding of the interest, concerns, fears, and values of individual citizens; disagreements among the experts; and limited resources and expertise for addressing risk problems. Channel problems include limitations in the modalities by which scientific information about environmental risks is transmitted. This problem can be a tremendous source of anxiety and fear for the public if information is disclosed prematurely without appropriate explanation or if distortions or biases inflate the risk. Finally, receiver problems entail characteristics of the intended recipients of the communication. These “risk receivers” may have inaccurate perceptions of the levels of risk,
lack of interest, high demands for scientific certainty, and/or difficulties in understanding results of the quantitative risk assessment. Most of these problematic issues will be discussed in more detail in the ensuing sections.

**Cardinal Rules of Risk Communication**

There are no easy rules or prescriptions for effective risk communication. The EPA published their Seven Cardinal Rules of Risk Communication in 1988 which have served as a general framework for understanding major principles of risk communication. These have been modified for PHCPs in their role as risk communicators to parents and caregivers. (Table 27)

**Rule 1: Accept and involve the parent/public as a legitimate partner.**

It is important to involve the parent, community and all parties that have an interest or stake in the particular issue. They should help in identifying and solving the problems. Decisions about risk should be based on factors other than the magnitude of the risk. An important goal of risk communication should be to produce an informed public that is involved, interested, reasonable, thoughtful, solution-oriented, and collaborative.

**Rule 2: Plan carefully, have a clear message, and evaluate your efforts.**

The risk communication process should begin with the basic communication principles discussed in the prior section. Different goals, audiences and and media require different risk communication strategies. Information about risks should be evaluated, paying particular attention to its strengths, weaknesses, uncertainties, and assumptions used in the assessment process. Risk messages and communication should be practiced and tested prior to the actual communication with the intended audience. Finally, efforts should undergo evaluation and discussion so that people can learn from mistakes.

**Rule 3: Listen to the parent’s / public’s specific concerns.**

Communication is a two-way process. If the presenter does not listen to a person or a group of people, he cannot expect them to listen to him. Also, it is important to not make assumptions
about what people know, think, or want done. People are often more concerned about trust, credibility, competence, control, fairness, and compassion than specific mortality statistics or quantitative risk assessments.

**Rule 4: Be honest, frank, and open.**

In communicating risk information, trust and credibility are considered vitally important assets. State your credentials, but do not ask or expect to be trusted. If you are uncertain or do not know an answer, state that you do not know. Disclose risk information as soon as possible and do not minimize or exaggerate the level of risk. Lean toward sharing more information, not less, or people may think you are hiding something. Trust and credibility are difficult to obtain and once lost, almost impossible to regain completely. (See Trust and Credibility Section).

**Rule 5: Coordinate and collaborate with other credible sources.**

It is important to coordinate with other organizations or groups and issue communications jointly with other credible sources. Conflicts or public disagreements with other credible sources make your risk communication more difficult.

**Rule 6: Meet the needs of the media and/or provide the parent access to appropriate information.**

The media are a prime transmitter of risk information and play a critical role in the process. The media are frequently more interested in politics than in risk, simplicity than in complexity, and in danger than in safety. Learning to provide risk information tailored to the needs of each type of media is an important process. Also, accessibility to reporters and establishing relationships of trust with specific channels of the media will make specific media channels important in the risk communication process. Learning how to interact with the media and use the media as effective communication modalities are important skills to obtain, depending on your role. This knowledge is beyond the scope of this monograph. It is important for the PHCP to be familiar with general informational resources about risk assessment, risk communication, and various
chemicals and their toxicities. Much of this information is readily accessible on the internet. (See Information Resources about the Risk Assessment Process)

**Rule 7:** Speak clearly and with compassion or refer the parent to someone who can.

Technical language and jargon can be barriers to successful communication with the public. It is important to use simple, non-technical language, use concrete images that communicate on a personal level; use examples and anecdotes that make technical risk data more meaningful; use meaningful risk comparisons to help put risks in perspective and acknowledge and respond to emotions that people express. These specific aspects of explaining risk will be discussed in more detail in the next section.

**EXPLAINING RISK**

An obvious and important component of risk communication is the actual explanation of risk or the risk message. However, as Peter Sandman states in his 1986 paper on Explaining Environmental Risk: “The task of risk communication, then, isn’t just conveying information, though that alone is a challenge; it is to alert people when they ought to be alerted and reassure them when they ought to be reassured… but the ultimate goal of risk communication should be rational alertness, not passive trust.” The actual explanation of risk must take into account content of the message as well as the basic principles of communication as described above—the target audience, goal of the message, and key points. Specific aspects of the message content such as the magnitude of the risk, technical information, and the uncertainties of the information, must be communicated in an effective, understandable and ethical manner so as not to create unfounded fear, anxiety, and confusion.

**General Principles**

As previously discussed, there are numerous variables that must be taken into account when communicating risk including the definition of risk, the perception of risk, the targeted audience and their needs and concerns, the goal of the communication, and the importance of
public involvement in risk management. General guiding principles about the actual explanation of risk are outlined below and listed in Table 28. These guidelines are adapted from the New Jersey Department of Environmental Protection’s manual on risk communication, *Improving Dialogue with Communities: A Risk Communication Manual for Government.* These principles reiterate many of the important factors that must be included in any risk communication effort.

- **Consider the qualitative factors associated with people’s perception of risk.**
  
  Different people see risk differently. One must understand what is making people fearful, anxious, or angry about a potentially risk situation before they can effectively explain the risk.

- **Explore what information people want and in what form.**
  
  Before presenting risk information, determine what individual and/or community concerns are present so that information can be appropriately relayed in the communication process. This information may be quite different than the actual technical information gleaned from formal quantitative risk assessments.

- **Anticipate and respond to people’s concerns about their personal risk.**
  
  Although many regulatory agencies focus on risks to populations, people are most concerned about their own risk and that of their families. Personal concerns such as “can my family drink the water?” or “Is it safe for my children to play on the school playground?” must be addressed through information relayed by PHCP. Pediatric health care providers will find themselves in this situation much more often due to the nature of the physician-patient relationship.

- **When explaining quantitative risk assessment, adequate background information about the process must be given.**
  
  The risk assessment process should be explained before relaying actual numbers derived from this process. This explanation should include clear and simple diagrams and other visual aids that illustrate how assessments are done, what are exposure routes and generally how numbers
are generated. It may be useful to express risks in several different ways since no single presentation of risk is entirely objective. Information needs to be put into some type of perspective. Most researchers suggest that the risk communicator should avoid dichotomizing risk, i.e. seeing risks as either safe or dangerous. Instead, one suggestion is to categorize risk as low, medium, or high.

- **Understand important guidelines when presenting technical information to the public.**

Presenting technical scientific data may be a major source of anxiety for the presenter as well as a major potential source of anxiety and confusion for the recipients. Many guidelines about presenting technical information are similar to general guidelines about explaining risk and will be discussed in detail in a later section.

- **Use caution when comparing environmental risks to other risks.**

Risk comparisons can be helpful to gain a better understanding, but must be presented with caution. There are several different types of risk comparison, of which some are more appropriate than others. More details about comparing risk are discussed in a later section (see Guidelines for Appropriate Use of Risk Comparisons).

- **Acknowledge the uncertainties associated with the risk assessment process.**

As discussed previously, there are many uncertainties associated with the risk assessment process, especially in determining long-term outcomes in children with potential environmental exposures for which we currently do not have any data. It is important to convey the sometimes inevitable uncertainty of science and to be specific about which information is uncertain. People’s demand for absolute certainty might stem from their frustrations and concerns which are not being addressed as well as their lack of involvement in the process, rather than their true desire for absolute scientific information. More details about explaining uncertainty are discussed in a later section.
• *Individuals and communities must determine what is acceptable to them, not the agency or primary risk communicator.*

People’s understanding of risk should not be confused with their acceptance of it. People may be able to understand the risk, but may not want to live with it. The term “acceptable risk” should be used cautiously as it is a relative term that will have different meanings to different people. People should be given more control over the risks presented to them, so that they might feel more comfortable with them. There are other aspects of decision-making besides risk and these must be acknowledged and heard. Dialogue between people facing the same risk should be encouraged so that people may be able to better accept some risk.

**Risk Messages – Informing and Influencing**

A risk message is a verbal, written, or visual presentation containing information about risk which may or may not include advice about risk reduction behavior. Risk messages can take a variety of forms—face-to-face interaction, advertisements, presentations to groups, direct mailing, press conferences, television or radio interviews, and newspaper/magazine articles. The purpose of risk messages is to inform and/or influence, both of which may occur in a single risk message or multiple messages. Risk communication may involve a single risk message with one designated purpose or multiple risk messages from many sources, making the process more complex. As stated previously, risk communication may occur in different settings; although these settings can generally be categorized into the public and personal arenas. Risk messages may differ in their purposes of informing and influencing depending on which arena and the goal of the message.

In a setting of public debate and regulatory actions, democratic risk communication includes a wide range of message, sources, and audiences. Interested groups and citizens pose questions and issues about environmental toxins for the experts who subsequently must respond with information. Regulatory decisions in the United States must generally be based on the best
available scientific knowledge. Consequently, risk communication in this regulatory context must entail messages describing, interpreting, and summarizing scientific knowledge about risks. However, the process may also include expressions of opinion, concern, and frustration by any participant in the process. Many messages are guided by political interests and thus may present a biased assessment of the available knowledge. Public policy about tobacco smoking is an example of this context. On the other hand, risk messages may be addressed to individuals rather than a group of people in public debate. Both recipients of risk messages (the individual and the regulatory agency) have the authority to make a decision; however, the individual may not need as much information or as much detailed information. The risks associated with tobacco smoking and consumption of alcoholic beverages during pregnancy illustrate the types of communication that must inform or influence individuals to make a personal choice. Warning labels on cigarette packages or wine bottles may be sufficient risk messages for personal action to reduce risks as opposed to a report of formal risk analysis. Risk communication in this setting is considered to be successful only if it adequately informs the individual for making a choice among alternatives.³¹

A primary goal of risk messages is to inform the recipients of the relevant information and knowledge about a particular issue. Ideally, the result is that the person or group of people informed gain a complete understanding of the issue; however, full understanding does not usually occur for most important choices about risk. Thus, a reasonable and practical goal for information messages is for the recipient to gain an understanding that is adequate to make appropriate choices given his or her values.³¹ Furthermore, adequate understanding may not require knowing every detail about an issue, but rather, knowing enough to make choices in one’s own best interests.

Numerous techniques are available for constructing risk messages that can be used to influence the targeted audience as well as to inform them. These techniques include highlighting
facts, “framing” information, persuasive use of facts, appeal to authority, and appeals to emotion.\textsuperscript{31} Unfortunately, sometimes using both techniques of informing and influencing can confuse the audience as to the intent of the message and/or the communicator.

Risk messages may not include all the known scientific details and still be read and understood by most nonexperts. Thus, people who design messages may delete some information and highlight other information. For example, a message may be developed that only presents knowledge about deaths due to an environmental toxin rather than the various types of cancer which have been associated with its exposure. Similarly, presenting the effects of an environmental chemical on the whole population which may be quite small, without relaying the increased incidence of effects on children. As discussed previously, making information more “available” (the availability heuristic) or highlighting information affects the understanding and perception of risk, influencing people’s beliefs about what aspects are important.\textsuperscript{21,25,47}

Information can be presented different ways resulting in different perceptions of risk. “Framing” information may involve the choice between alternative ways of presenting the same numerical information. Stating that the risk of developing cancer is less than 1\% is less attractive than stating there is over a 99\% chance of not developing cancer. Similarly, presenting outcomes with phrases such as “lives saved” as opposed to “lives lost” or “children who develop learning problems” may “frame” the magnitude of the risk.\textsuperscript{19,48} Another important example of framing is the use of risk comparisons which is discussed in more detail in a later section.

Selective presentation of information, creation of specific arguments, and careful placement of various arguments within a message for maximum effect can augment the persuasive effect of messages.\textsuperscript{4,14,29,31} Nonexperts may appeal to various authorities and experts to learn who has taken what position on a difficult issue or choice. Designers of risk messages may present unbalanced authoritarian views; i.e., present quotations from some experts in
support of an issue but omit quotations from some experts who disagree with the issue. Appeals to emotions can be effective influence techniques in selected situations. Risk messages may be designed to generate fear, guilt, anxiety, parental concerns, or other emotions so that people will take action. A commercial relaying information that mercury poisoning causes severe neurological problems in children conveys the same information whether or not there is an accompanying film of a neurologically devastated child. However, the message using the film may have a different effect by appealing to people’s emotions.

**Communicating Technical Information**

General guidelines for communicating technical issues are similar to general principles about explaining risk. Risk-related numbers and statistics from the quantitative risk assessment need to be communicated in a manner which is understandable to the targeted audience. These include level of risk, concentrations (i.e. parts per million), probabilities (i.e. the likelihood of the event), and quantities (i.e. how many tons of water contaminated with chemical X). Several references outline in detail specific guidelines for explaining technical information about risk.\(^9,24\) Covello and colleagues outline 10 steps on how best to provide and explain risk-related numbers and statistics.

1. Do not just present the risk-related numbers and statistics as you find them. Numbers and statistics should be selected wisely.
2. Pick a risk number for which the data are good.
3. Use whole numbers and simple fractions whenever possible, such as 7 parts per billion instead of 0.007 parts per million.
4. Pick a number that will be easy for the audience to understand, avoiding unfamiliar units or overly complex concepts. It is helpful to explain the number in words. For example, a risk of 0.047 is understandable only to a select group of
people; however, most people can understand that roughly 5 people in a group of 100 people would be affected.

5. Use graphs, charts, and other visual aids.

6. Choose a number considered fair and relevant by other knowledgeable people. “Fair” means that the number does not give a misleading impression of the seriousness of the risk while “relevant” means that the number refers to the issue at hand.

7. Pick a number that the audience will also consider fair and relevant. Find out which health or environmental effects the people care about. Explaining miscarriage rates to a group of parents concerned about cancer risks to their children is not productive.

8. Strive for comprehensibility and clarity without oversimplifying. Most people are capable of understanding quite complex quantitative information if sufficiently motivated.

9. Pay close attention to the numbers that others are using. People will become easily confused if different risk communicators use different measures of the same risk.

10. Consider offering several different estimates of the same risk. For example, the best estimate of risk of cancer from water pollution is $a$. Our worst-case estimate is $b$. The highest estimate we have found from the X organization is $c$.

It may also be beneficial for the risk communicator to “personalize” the risk-related numbers and statistics to overcome some of the perceived emotionality of the risk—what might happen to me or my child if we drink the water? Several approaches have been suggested.8

- Using examples and anecdotes—hypothetical if necessary, real if possible—to make the risk data come alive.
• Talking about oneself; for example, about risks that are personally unacceptable, about feelings toward the audience at that moment.

• Using concrete images to give substance to abstract risk data.

• Avoiding distant, abstract, and unfeeling language about death and illness.

• Listening to people express their concerns.

The ability of the public to assimilate technical information should not be underestimated. Know who your audience will be and design your presentation based on what they want to know and what you want them to know. As stated previously, it is important to anticipate and respond to people’s concerns about their own personal risk rather than just giving the facts. Take care to give adequate background when explaining risk numbers. Use language that is consistent with the expertise of the audience and make it clear, concrete, and as down-to-earth as possible, avoiding jargon and acronyms. Selectively provide the technical information that helps respond to people’s concerns, but is neither too complex, too patronizing, or too much volume. On the other hand, don’t oversimplify the information and only present data that support one view. Use simple graphics to illustrate the major points to be communicated. Collaborate with other credible experts when communicating technical information.

Unfortunately, explaining complex information is a very difficult task. Further research is needed on how to explain environmental health risks. However, regardless of our ability to explain risk, people’s perception of the risk will be influenced by many more factors other than scientific data. Risk communication will fail and further problems will arise if these other factors (i.e. people’s needs, concerns, perception of risk, etc.) are not addressed.

**Explaining Uncertainty**

There are several types of uncertainty that must be addressed in the risk communication process including 1.) the uncertainty of the science in general; b.) the inexactness of the risk
assessment process; c.) the incompleteness of the information that has been gathered; and 4.) differences of opinion as to the implications of the information and the optimum risk management option. When addressing uncertainty, several guidelines have been proposed.

1.) Acknowledge the uncertainty. It is a much better strategy to admit your uncertainty than claiming to know more than you actually do. Uncertainties in the risk assessment process (previously outlined in the first section) must be acknowledged up front.

2.) Consider involving the public in resolving the uncertainty. It may be easier for people to accept uncertainty if they can play a role in its resolution. Involving the public in resolving uncertainty may also be perceived as fairer and may lead to better solutions.

3.) Give people as much individual control as possible over an uncertain situation.

4.) Stress the caution built into standard-setting and the risk assessment process. It must be explained that agencies build standards with wide margins of safety into risk assessments to account for the uncertainty and to protect the public.

5.) If people are demanding absolute certainty, pay attention to values and other concerns, not just the science. As previously discussed, the demand for absolute certainty may stem from people’s frustrations and anger about lack of involvement or control and not necessarily the science. It is important to listen to the concerns behind the demand.

When presenting the uncertainties in a risk assessment, there is a high risk that the presentation may be perceived negatively and interpreted with vastly different conclusions. The risk of being misunderstood or misperceived can be reduced if the needs and concerns of the specific audience is considered. For example, a thorough scientific presentation of uncertainties of the risk of neurological complications from chemicals found in the playground soil may be received by a group of other risk assessors and regulatory personnel with a clear understanding, concluding that the results can be used with confidence. On the other hand, the same
presentation given to parents of school children may be received quite adversely. Suspicious and angry parents might conclude that the risk must be high because the presenter focused heavily on the uncertainties of the assessments and not their concerns or interests. (“The presenter must not be competent because he is not sure about anything and he is only concerned about his own issues.”)

Degree of certainty can be represented along a scale or continuum, from “total confidence” in the data to “guesswork.” Most data generated by risk assessments are somewhere in the middle of the scale. Nonetheless, such data are better for making decisions and taking actions than guesswork. Thus, the information is helpful but not certain. When the data are near the “guesswork” end of the scale, this knowledge must be stated. It is important to state as much is known and then explain what will be done to get better risk data and what will be done in the interim to reduce the risk or to protect people against it. In communicating risk about infants’ and children’s exposures to potentially toxic environmental chemicals, risk estimates are very uncertain because the data are lacking and the measurements are difficult. In these situations, it is important to explain the risk assessment process so that the public can understand why the risk estimate is uncertain and that the estimates presented are based on the best available scientific data.

It is important to address the specific needs and concerns of the decision maker (risk assessor, citizen, or other official) in situations where a discussion of uncertainty is needed to help make a decision. Some examples of the needs and concerns of the risk assessor and the public in the form of questions are listed in Table 29. These questions can also be viewed as “uncertainty criteria”, where the more certain that these criteria are met, the more the situation will be considered, safe, acceptable, or good.
**Guidelines for Appropriate Use of Risk Comparisons**

Risk comparisons are a powerful tool in risk communication. Risk comparisons may help to put risks into perspective and thus are an important part of improving risk communication. Comparing one risk which may not be well understood to another risk that is more easily understood or the public is more familiar with may be a useful way to convey information about the former risk. However, it is often difficult to find risks that are similar on enough attributes to validate the comparisons. Furthermore, risk comparisons can be used to influence or even mislead the public, because a risk comparison may imply that if a person is willing to take the larger of two risks he or she should accept the smaller risk as well. Thus, risk comparisons must be used cautiously and in the appropriate circumstances.

Comparison of a risk with other risks does not establish the acceptability of the risk in question. If the chance of morbidity or mortality from a previously unknown risk is about the same as that from a known risk, this does not necessarily imply that the two risks are equally acceptable. The logic of using comparisons to determine acceptable risks is as follows: since one accepts the risk of driving an automobile which is > 200 annual fatalities per million persons, then one ought to accept the risk of exposure to chemical X which is projected at 10 annual fatalities per million. Per social science experts, this logic is erroneous. People should not ignore the potential fire hazards of a gas range just because the risk of fatalities due to fire is much smaller than the risk of driving an automobile. The particular level of risk is only one factor among many that determine its acceptability.

Generally, comparing risks along a single dimension is not helpful when the risks are widely perceived as qualitatively different. As previously discussed in the section on risk perception, different risks have different qualitative characteristics. These characteristics—voluntariness, fairness, benefits, alternatives and control-- can affect the way comparisons are
viewed. Hazards with very similar quantitative risks may result in different responses if their qualitative characteristics are different. Thus, avoid risk comparisons that ignore these factors or do not share similar qualitative characteristics.

A major pitfall of comparing risks is selecting risks for comparison that minimize or trivialize the risk in question. The use of risk ladders, which present a range of probabilities from lower to higher risk for a single class of risks, may give this appearance when the risk in question is much lower than other risks and when there are few risks presented with comparable levels. This technique of comparing risks may help people to better understand the magnitude of risks, although there may be some weaknesses with this approach as illustrated in Figure 10.

Risk-related numbers and statistics can be presented as part of a comparison. “Concentration” comparisons (i.e. the risk is equal to a drop of benzene in a 10 million gallon lake) are commonly used, but seldom helpful. Most concentration comparisons appear to trivialize the problem and prejudge its acceptability. Therefore, concentration comparisons—usually designed to convince the audience to stop worrying—are likely to fail and should be used sparingly. “Quantity” comparisons may be more useful. It may be helpful to translate tons of contaminated soil into number of swimming pools or football stadiums because the audience is able to visualize the amount to some extent.

Risk comparisons should focus the comparison on classes of substances, products, processes or activities that are similar or related in their characteristics, such as activities that serve the same function and whose benefits tend to be similar. Some kinds of risk comparisons are more likely than others to be perceived as an effort at minimizing or trivializing risk or as an effort to preempt judgments about the acceptability of the risk. A categorization and ranking system for risk comparisons has been reported and is outlined below with examples. The highest-ranking comparisons (first-rank risk comparisons) are those that put the least strain on the trust between groups of people, and appear to be the most relevant, appropriate and helpful.
The lowest-ranking comparisons are those that have no obvious relevance, appropriateness, or helpfulness and appear to be manipulative and/or misleading. A general rule is to select from the highest-ranking comparison whenever possible and to use a low-ranking risk comparison cautiously.

**First-Rank Risk Comparisons (most acceptable)**

1.) *Comparison of the same risk at two different times.*

Example: The risk of asthma in the school is 20% less than it was before we removed the mold from the airducts.

2.) *Comparison of the risk with a standard.*

Example: Exposure of school children to asbestos is well below the level that the Occupational Safety and Health Administrations considers safe.

3.) *Comparisons with different estimates of the same risk.*

Example: The best estimate of the risk of asthma from the assessment of the school’s playground is “X”; however the EPA has calculated a worst-case risk estimate of “Y” based on assessments of this and other playgrounds.

**Second-Rank Risk Comparisons (less desirable)**

1.) *Comparisons of the risk of doing something versus not doing it.*

Example: If the newest and most advanced water filtration equipment is installed, the risk of exposure will be \( x \), whereas if this equipment is not installed, the risk will be \( y \).

2.) *Comparison of alternative solutions to the same problem.*

Example: The associated risk with incinerating waste is \( x \), and the risk associated with using a landfill is \( y \).

3.) *Comparisons with the same risk as that experienced in other places.*
Example: The most serious soil contamination problems have been demonstrated in city $x$; the soil contamination problem in this community is only 1/10 as serious as city $x$.

**Third-Rank Risk Comparisons (even less desirable)**

1.) *Comparisons of average risk with peak risk at a particular time or location.*

Example: The risk associated with airborne dust from the playground is $x$ on an average day which is one-thousandth as great as the risk one year ago during the wind storm.

2.) *Comparisons of the risk from one course of an adverse effect with the risk from all sources of the same effect.*

The risk of cancer from exposure to soil contaminant $x$ is five-hundredths of 1 percent of the total cancer risk in the community.

**Fourth-Rank Risk-Comparisons (marginally acceptable)**

1.) *Comparisons of risk with cost, or of one cost-risk ratio with another cost-risk ratio.*

Example: Reducing the risk posed by soil contaminant $x$ by half would cost $y$ dollars.

2.) *Comparison of a risk with benefit.*

Example: The chemical product whose waste by-product is soil contaminant $x$ is used by industry as a pesticide and thus helps produce viable food supply to the community.

Risk-benefit comparisons are more acceptable when the benefits are directed to the same people. However, it may still be perceived as bribery or blackmail (i.e. the community will receive $x$ amount of money for new schools from the industry or the community will lose $x$ number of jobs if the plant is shut down). Benefits should be explained separately from its risks so that people can make their own risk-benefit comparisons.

3.) *Comparison of occupational risks with environmental risks.*

Example: The community is exposed to far less of the soil contaminant $x$ than the plant workers were exposed to who had no adverse health effects.
4.) *Comparison with other risks from the same source.*

Example: Exposure to soil contaminant \( x \) from the chemical plant \( a \) is no more serious a problem than exposure to soil contaminant \( y \) from plant \( b \) (which the community has accepted for over 50 years).

5.) *Comparison with other specific causes of the same disease, illness, or injury.*

Example: Soil contaminant \( x \) produces far less cancer than does exposure to natural background levels of the same chemical.

**Fifth-Rank Risk Comparisons (use with extreme caution)**

*Comparison of unrelated risks. (smoking, driving a car, getting struck by lightning)*

Example: The risk of cancer from exposure to soil contaminant \( x \) is less than getting struck by lightning. Comparing two totally different risks with different outcomes is both unacceptable and meaningless. To say that a child’s risk of developing leukemia from benzene exposure is less than the risk of getting struck by lightening is flawed and may promote anger among parents.

Risk comparisons should be targeted to the specific audience. They should be constructed carefully to address important and relevant health and environmental consequences. Their usefulness should then be tested and evaluated. Risk comparisons may be very helpful and serve an important role in selected situations.

**TRUST AND CREDIBILITY**

No review of risk communication would be complete without a discussion on the importance of trust and credibility in this process. Establishing trust and credibility is essential for risk communication to be successful, more so than pages of data analysis and probabilistic summaries of risk. If the audience trusts the communicator, they are much more likely to
respond to presentations of risk with less anger, fear, and anxiety. The only way to achieve credibility is to “be credible”. A critical element of credibility for a particular source is the degree to which the recipients of the risk message believe that source to be justified in the position reflected in the message.31

Guidelines for building trust and credibility are outlined in Table 30. Being consistently honest, fair, competent, and caring will earn trust. However, acting trustworthy will not necessarily guarantee that people will trust you. Failing to earn credibility will engender anger, opposition, and resentment. An important key to building trust is involving the public (the patient, the parent, or the group) in the decision-making process. It is important for the risk communicator to be forthcoming with information and listen to what people are telling him/her from the outset. Trust should not be confused with agreement—people can trust each other and still disagree on issues. Although, trust may not be earned by every citizen involved in a situation, it is important to always practice the basic principles. Furthermore, most risk communication disasters have originated from a major breakdown in trust between the public and government/regulatory agencies. Because this is such an important component of risk communication, these concepts are illustrated again in Table 31, but in a reverse manner, i.e. ten ways to lose trust and credibility.

SUCCESSFUL RISK COMMUNICATION

The risk communication process can be judged successful if it adequately informs the affected or interested individuals or groups within the limits of the available knowledge. Successful risk communication may not always lead to the optimal or best decisions because it is only one part of risk management. Also, it may not result in consensus about controversial issues or in uniform personal behavior.31 Because different people have different values and interests, a better understanding will not necessarily lead them all in the same decision-making direction. Disseminating accurate information about risk does not necessarily define the process
as successful, unless the recipients achieve an adequate understanding. Finally, risk messages describing expert knowledge to nonexperts are necessary for risk communication; however, they are not sufficient by themselves for the process to be successful. Nonexperts such as patients, parents, and school personnel play an integral role in the process by contributing to an interactive dialogue that includes the expression of their concerns, interests, and values. Risk communication should produce an informed public that is involved, interested, reasonable, thoughtful, solution oriented, and cooperative. Successful risk communication will help to engender not only public trust, but also trust among individuals.
V. INFORMATION RESOURCES FOR RISK COMMUNICATION

Publications:


Internet Sites:

Decision Research:

http://www.decisionresearch.org/

Decision Research is dedicated to helping individuals, industry, government, and society understand and cope with complex and risky decisions of modern life. Dr. Paul Slovic, a founder of Decision Research, is one of the world’s leading analysts of risk, risk perception, and risk management. The web site provides an extensive publication record of the researchers at Decision Research for the last 10 years. A limited number of reprints are also available.

Risk Communication Bibliography:

http://excellent.com.utk.edu/~mmmiller/bib.html

This web site, authored by Dr. Steve Depoe, provides an extensive bibliography of articles on risk communication since 1995.
A Primer on Health Risk Communication Principles and Practices:

http://www.atsdr.cdc.gov/HEC/primer.html

This primer, published by the Agency for Toxic Substances and Disease Registry, provides a comprehensive, yet concise, overview on the principles and practices of health risk communication with practical examples and scenarios.

The Peter Sandman Risk Communication Web Site:

http://www.psandman.com/

Dr. Sandman, an expert in risk communication, is the creator of the risk formula: Risk = Hazard + Outrage. This web site contains an extensive bibliography of publications and training videos on risk communication. Many of the publications can be directly accessed on the web.
VI. REFERENCES


Table 22. Alternative Ways of Expressing Mortality Statistics

<table>
<thead>
<tr>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Deaths per million people in the population.</td>
</tr>
<tr>
<td>• Deaths per million people within $x$ miles of the facility.</td>
</tr>
<tr>
<td>• Deaths per unit of concentration (LD-50 or any toxicity measure).</td>
</tr>
<tr>
<td>• Deaths per facility.</td>
</tr>
<tr>
<td>• Deaths per ton of the airborne toxic substance released.</td>
</tr>
<tr>
<td>• Deaths per ton of the airborne toxic substance absorbed by people.</td>
</tr>
<tr>
<td>• Deaths per ton of chemical produced.</td>
</tr>
<tr>
<td>• Deaths per million dollars of product produced.</td>
</tr>
</tbody>
</table>

Table 23. Characteristics of the “Expert” and “Public” Languages of Risk Communication.

<table>
<thead>
<tr>
<th>“Expert” Assessment of Risk</th>
<th>“Public” Assessment of Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scientific</td>
<td>Intuitive</td>
</tr>
<tr>
<td>Probabilistic</td>
<td>Yes / No</td>
</tr>
<tr>
<td>Acceptable Risk</td>
<td>Safety</td>
</tr>
<tr>
<td>Changing Knowledge</td>
<td>Is it or isn’t it?</td>
</tr>
<tr>
<td>Comparative Risk</td>
<td>Discrete Events</td>
</tr>
<tr>
<td>Population Averages</td>
<td>Personal consequences</td>
</tr>
<tr>
<td>A Death is a death.</td>
<td>It matters how we die.</td>
</tr>
</tbody>
</table>

Powell D. Setting the stage: understanding communication issues with foodborne pathogens. [http://www.plant.uoguelph.ca/safefood/risk-anal/oca-talk/oca-talk.htm](http://www.plant.uoguelph.ca/safefood/risk-anal/oca-talk/oca-talk.htm)
Table 24. Qualitative Factors Affecting Risk Perception (“Outrage Factors”)

<table>
<thead>
<tr>
<th>Factor</th>
<th>Conditions Associated with Increased Public Concern/“More Risky”</th>
<th>Conditions Associated with Decreased Public Concern/“Less Risky”</th>
</tr>
</thead>
<tbody>
<tr>
<td>Voluntariness of exposure</td>
<td>Involuntary</td>
<td>Voluntary</td>
</tr>
<tr>
<td>Controllability (personal)</td>
<td>Uncontrollable</td>
<td>Controllable by Individuals</td>
</tr>
<tr>
<td>Familiarity</td>
<td>Unfamiliar / exotic</td>
<td>Familiar</td>
</tr>
<tr>
<td>Uncertainty</td>
<td>Risks scientifically unknown or uncertain</td>
<td>Risks known to science</td>
</tr>
<tr>
<td>Catastrophic Potential</td>
<td>Fatalities and injuries grouped in time and space</td>
<td>Fatalities and injuries scattered and random</td>
</tr>
<tr>
<td>Detection of effects</td>
<td>Undetectable</td>
<td>Detectable</td>
</tr>
<tr>
<td>Effects manifestation</td>
<td>Delayed effects</td>
<td>Immediate effects</td>
</tr>
<tr>
<td>Effects on children</td>
<td>Children specifically at risk</td>
<td>Children not specifically at risk</td>
</tr>
<tr>
<td>Effects on future generations</td>
<td>Risk to future generations</td>
<td>No risk to future generations</td>
</tr>
<tr>
<td>Victim identity</td>
<td>Identifiable victims</td>
<td>Statistical victims</td>
</tr>
<tr>
<td>Comprehension</td>
<td>Not well understood by science</td>
<td>Well understood by science</td>
</tr>
<tr>
<td>Memorability</td>
<td>Dramatic, memorable</td>
<td>Uninteresting, forgettable</td>
</tr>
<tr>
<td>Fairness</td>
<td>Unfair</td>
<td>Fair</td>
</tr>
<tr>
<td>Dread</td>
<td>Effects dreaded</td>
<td>Effects not dreaded</td>
</tr>
<tr>
<td>Benefits</td>
<td>Unclear benefits</td>
<td>Clear Benefits</td>
</tr>
<tr>
<td>Morality</td>
<td>Morally relevant (immoral)</td>
<td>Morally irrelevant (moral)</td>
</tr>
<tr>
<td>Reversibility</td>
<td>Irreversible effects</td>
<td>Reversible effects</td>
</tr>
<tr>
<td>Trust in Institutions</td>
<td>Lack of trust</td>
<td>Trust</td>
</tr>
<tr>
<td>Media attention</td>
<td>Much media attention</td>
<td>Little media attention</td>
</tr>
<tr>
<td>Origin</td>
<td>Caused by human action or failures</td>
<td>Caused by acts of nature or God</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Types of Risk Communication Objectives</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Information and Education</strong></td>
</tr>
<tr>
<td>• Informing and educating people about risks and risk assessment in general.</td>
</tr>
<tr>
<td>Example: Informing parents about the risks of pollutants in the water and soil.</td>
</tr>
<tr>
<td><strong>Behavior Change and Protective Action</strong></td>
</tr>
<tr>
<td>• Encouraging personal risk-reduction behavior.</td>
</tr>
<tr>
<td>Example: Advertisements encouraging mothers not to consume alcohol while pregnant</td>
</tr>
<tr>
<td><strong>Disaster Warning and Emergency Information</strong></td>
</tr>
<tr>
<td>• Providing direction and behavioral guidance in disasters and emergencies.</td>
</tr>
<tr>
<td>Example: Sirens indicating the accidental release of toxic gas from a chemical plant</td>
</tr>
<tr>
<td><strong>Joint Problem Solving and Conflict Resolution</strong></td>
</tr>
<tr>
<td>• Involving the public in risk management decision-making and in resolving health, safety, and environmental controversies.</td>
</tr>
<tr>
<td>Example: Holding public meetings on the chemical substances found in the school’s playground and water</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 26. Risk Communication Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Message Problems:</strong></td>
</tr>
<tr>
<td>• Deficiencies in scientific understanding, data, models, and methods resulting in large uncertainties in risk estimates</td>
</tr>
<tr>
<td>• Highly technical analyses that are often unintelligible to lay persons</td>
</tr>
<tr>
<td><strong>Source Problems:</strong></td>
</tr>
<tr>
<td>• Lack of trust and credibility</td>
</tr>
<tr>
<td>• Disagreements among scientific experts</td>
</tr>
<tr>
<td>• Limited authority and resources for addressing risk problems</td>
</tr>
<tr>
<td>• Lack of data addressing the specific fears and concerns of individuals and communities</td>
</tr>
<tr>
<td>• Failure to disclose limitations of risk assessments and resulting uncertainties</td>
</tr>
<tr>
<td>• Limited understanding of the interests, concerns, fears, values, priorities, and preferences of individual citizens and public groups</td>
</tr>
<tr>
<td>• Use of bureaucratic, legalistic, and technical language</td>
</tr>
<tr>
<td><strong>Channel Problems:</strong></td>
</tr>
<tr>
<td>• Selective and biased media reporting that emphasizes drama, wrongdoing, conflicts</td>
</tr>
<tr>
<td>• Premature disclosures of scientific information</td>
</tr>
<tr>
<td>• Oversimplifications, distortions, and inaccuracies in interpreting technical risk information</td>
</tr>
<tr>
<td><strong>Receiver Problems:</strong></td>
</tr>
<tr>
<td>• Inaccurate perceptions of levels or risk</td>
</tr>
<tr>
<td>• Lack of interest in risk problems and technical complexities</td>
</tr>
<tr>
<td>• Overconfidence in one’s ability to avoid harm</td>
</tr>
<tr>
<td>• Strong belief and opinions that are resistant to change</td>
</tr>
<tr>
<td>• Exaggerated expectations about the effectiveness of regulatory actions</td>
</tr>
<tr>
<td>• Desire and demands for scientific certainty</td>
</tr>
<tr>
<td>• Reluctance to make trade-offs between different types of risk or between risks, costs, and benefits</td>
</tr>
<tr>
<td>• Difficulties in understanding probabilistic information related to unfamiliar technologies</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Rule</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Accept and involve the parent/public as a legitimate partner in identifying and solving the problem.</td>
</tr>
<tr>
<td>2</td>
<td>Plan carefully, have a clear message, and evaluate your efforts.</td>
</tr>
<tr>
<td>3</td>
<td>Listen to the parent’s/public’s specific concerns.</td>
</tr>
<tr>
<td>4</td>
<td>Be honest, frank, and open.</td>
</tr>
<tr>
<td>5</td>
<td>Coordinate and collaborate with other credible sources.</td>
</tr>
<tr>
<td>6</td>
<td>Meet the needs of the media and/or provide the parent access to the information.</td>
</tr>
<tr>
<td>7</td>
<td>Speak clearly and with compassion or refer the parent to someone who can.</td>
</tr>
</tbody>
</table>

Table 28. General Guidelines About Explaining Risks

- Consider the qualitative factors associated with people’s perception of risk.
- Explore what information people want and in what form.
- Anticipate and respond to people’s concerns about their personal risk.
- When explaining quantitative risk assessment, adequate background information about the process must be given.
- Understand important guidelines when presenting technical information to the public.
- Use caution when comparing environmental risks to other risks.
- Acknowledge the uncertainties associated with the risk assessment process.
- Individuals and communities must determine what is acceptable to them, not the agency or primary risk communicator.

Table 29. **Needs and Concerns of the Risk Assessor and the Public in the Form of Questions** ("Uncertainty Criteria" where the more certain that these criteria are met, the more it will be considered safe, acceptable, or good.)

<table>
<thead>
<tr>
<th>Risk Assessor</th>
<th>Public</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Meets Government standards?</td>
<td>• Is it safe for me (adult)?</td>
</tr>
<tr>
<td>• Population Risk &lt; 1 cancer case</td>
<td>• Is it safe for my child?</td>
</tr>
<tr>
<td>• Is it within $10^{-4}$ and $10^{-6}$ risk?</td>
<td>• Does the government understand my concerns?</td>
</tr>
<tr>
<td>• Is it detectable?</td>
<td>• Will I be kept informed?</td>
</tr>
<tr>
<td>• Is it above background levels?</td>
<td>• Will I have access to the process?</td>
</tr>
<tr>
<td>• Is it an enforceable solution?</td>
<td>• Is it fair?</td>
</tr>
<tr>
<td>• Is it feasible to clean up?</td>
<td>• Will the value of my home be maintained?</td>
</tr>
<tr>
<td>• Are sensitive populations protected?</td>
<td>• Will my quality of life remain the same?</td>
</tr>
<tr>
<td>• Will I meet my deadline?</td>
<td>• Are there favorable news reports?</td>
</tr>
<tr>
<td>• Are the actions consistent with the type of chemical or carcinogen?</td>
<td>• Are others saying good things about it?</td>
</tr>
<tr>
<td>• Are the assumptions in the risk assessment health protective?</td>
<td>• Does the government have a high degree of confidence in their actions?</td>
</tr>
<tr>
<td>• Are the uncertainties in the risk assessment health protective?</td>
<td>• Is the government honest?</td>
</tr>
<tr>
<td></td>
<td>• Will the government really listen?</td>
</tr>
<tr>
<td></td>
<td>• Will the government be considerate of my time?</td>
</tr>
<tr>
<td></td>
<td>• Is the government taking me seriously?</td>
</tr>
<tr>
<td></td>
<td>• Will the air smell the same?</td>
</tr>
<tr>
<td></td>
<td>• Will it look OK?</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 30. Guidelines for Earning Trust and Credibility</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Be aware of the factors which inspire trust.</td>
</tr>
<tr>
<td>• Pay attention to process.</td>
</tr>
<tr>
<td>• Explain agency procedures.</td>
</tr>
<tr>
<td>• Be forthcoming with information and involve the public from the outset.</td>
</tr>
<tr>
<td>• Focus on building trust as well as generating good scientific data.</td>
</tr>
<tr>
<td>• Follow up.</td>
</tr>
<tr>
<td>• Make only promises you are sure you can keep.</td>
</tr>
<tr>
<td>• Provide information that meets people’s needs.</td>
</tr>
<tr>
<td>• Get the facts straight.</td>
</tr>
<tr>
<td>• Try to coordinate with other agencies.</td>
</tr>
<tr>
<td>• Don’t give mixed messages.</td>
</tr>
<tr>
<td>• Listen to what various groups are telling you.</td>
</tr>
<tr>
<td>• Enlist the help of organizations that have credibility with communities.</td>
</tr>
<tr>
<td>• Avoid “closed” meetings.</td>
</tr>
<tr>
<td>• If dealing with a situation, in which trust is low, consider taking several steps to acknowledge the lack of trust, steps to prevent the trust-eroding actions from happening again, responding on a personal level, and efforts to recoup trust.</td>
</tr>
</tbody>
</table>

### Table 31. Ten Ways to Lose Trust and Credibility

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Don’t involve people in decisions that directly affect their lives.</td>
</tr>
<tr>
<td>2.</td>
<td>Hold onto information until people are screaming for it.</td>
</tr>
<tr>
<td>3.</td>
<td>Ignore peoples’ feelings.</td>
</tr>
<tr>
<td>4.</td>
<td>Don’t follow-up.</td>
</tr>
<tr>
<td>5.</td>
<td>If you make a mistake, deny it.</td>
</tr>
<tr>
<td>6.</td>
<td>If you don’t know the answers, fake it.</td>
</tr>
<tr>
<td>7.</td>
<td>Don’t speak plain English.</td>
</tr>
<tr>
<td>8.</td>
<td>Present yourself like a bureaucrat.</td>
</tr>
<tr>
<td>9.</td>
<td>Delay talking to other agencies involved.</td>
</tr>
<tr>
<td>10.</td>
<td>If one of your scientists has trouble relating to people, hates to do it, and has begged not to, send him or her out anyway.</td>
</tr>
</tbody>
</table>

Figure 8. Integration of Risk Assessment, Risk Management, and Risk Communication

RISK ASSESSMENT
- Hazard ID
- Exposure Assessment
- Toxicity Assessment

RISK MANAGEMENT
- Regulatory
- Economic
- Advisory
- Technological

RISK COMMUNICATION
- Identify the intended audience.
- Set realistic goals.
- Explore alternative ways to achieve the goal.
- Formulate communication objectives.
Figure 9. Model Demonstrating Effect of Information and Affect on Perception of Risk.
Model showing how information about benefit (A) or information about risk (B) could increase the global affective evaluation of nuclear power and lead to inferences about risk and benefit that are affectively congruent with the information input. Similarly, information could decrease the global affective evaluation of nuclear power as in C and D, resulting in inferences that are opposite to those in A and B. From Finucane ML et al: The Affect Heuristic in Judgments of Risks and Benefits; J Behav Dec Making 2000; 13:1-17; John Wiley & Sons Limited. Reproduced with permission.
Figure 10a. Risk Ladder

This model demonstrates poor risk comparisons. Principle weaknesses of this model are:
1.) Estimated levels of risk are presented on a log scale which some people will fail to realize; thus, equal distances between points on the scale do not represent equal differences in magnitude; 2.) All risks are presented as point estimates; 3.) Periods of exposure are not delineated; 4.) Risks with very different qualitative attributes are included (i.e. drinking a diet soda and fighting a fire). From Covello VT, Sandman PM, Slovic P. Risk communication, risk statistics, and risk comparisons: A manual for plant managers. Washington, DC: Chemical Manufacturers Association. 1988. Reprinted with permission from the American Chemistry Council.
This model demonstrates better risk comparisons. Principal weaknesses include:
1.) Exposure factors are not defined; 2.) It is not known whether most people accurately perceived the factors of smoking, asbestos, and X-rays; 3.) The original published scale uses colors which may be misleading; and 4.) The factors of smoking, asbestos, and X-rays have different qualitative attributes.

SUMMARY

Children’s environmental health issues must be an important health priority for the new millennium. Children’s increasing exposures to a wide array of environmental toxicants in the last 10 years poses new challenges for research, education, regulation, and policy regarding numerous environmental issues. Many chronic diseases in children thought to be caused by toxic environmental exposures such as asthma, cancer, and neurodevelopmental delay have been termed by some “the new pediatric morbidity.” Undoubtedly, with the increasing production of synthetic chemicals, the extent of children’s exposures to potentially toxic substances will increase in the ensuing decade.

Extensive literature has been published in the last twenty years on risk assessment and risk communication. However, only until recently, have these processes been examined in the context of the infant, child, and adolescent. Because of increasing publicity and awareness of environmental threats to children, pediatric health care providers need to understand the fundamental principles of these processes so that they are better able to answer patient and parent questions and/or refer them to the appropriate resources. This monograph discusses risk assessment and risk communication as they apply to children’s exposures to potentially toxic substances in the environment.

The traditional components of quantitative risk assessment must take into account children’s unique susceptibilities to environmental toxicants. The risk assessment paradigm should focus on additional aspects of the exposure such as the effects of long-term, low-level exposure, the effects of multiple and cumulative exposures, transgenerational effects, and delayed effects. Defining and characterizing
exposure patterns in children is one of the most important factors in developing new approaches to risk assessment in children. Furthermore, strategies for improving toxicity data on those chemicals to which children are most exposed must be developed as well as strategies for targeting research at highly-exposed children and/or children at the highest risk (i.e. during critical periods of development). Further development of specific biologic markers will strengthen the risk assessment process in children.

Successful risk communication is an integral and necessary component of the risk assessment and risk management processes. Pediatric health care providers are in a strategic position as communicators for information on various environmental issues. Accurate and practical communication to parents, caretakers, school personnel, and the media about hazardous exposures and potential adverse health effects is necessary to protect our children, to prevent unnecessary fear and anxiety, and to further promote healthy environments for our children. Understanding the multidimensionality of risk as well as the qualitative factors affecting people’s perception and acceptability of risk are essential for communicating risk successfully. General principles about risk communication are presented in this monograph to help the pediatric health care providers inform others within the limits of their knowledge and/or to provide a foundation of how to understand other risk communicators.