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# Children — Unique and Vulnerable. Environmental Risks Facing Children and Recommendations for Response

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Children may be more susceptible to exposures to environmental toxins than adults and may be more vulnerable to their effects. Because of this, the health care community and those responsible for children need to be alert to possible environmental factors in identifying and responding to the health problems of children. Their focus should be on the causes of the health problem, emphasizing environmental sources, and not on simply treating the symptoms. — *Environ Health Perspect* 103(Suppl 6):13–18 (1995)

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## Introduction

Children may be more susceptible to environmental exposures than adults and, because of their developing systems, uniquely vulnerable to their effects.

There is a growing urgency for researchers in the private and public sectors to move to fill gaps in the data and for decision makers to incorporate available information into pollution control and prevention strategies. Damage caused to children can be devastating and permanent, and the latency period for certain effects can be decades.

Because of this potential, the health care community and those responsible for children need to be alert to possible environmental factors in identifying and responding to health problems confronting children. All too often, the immediate focus is on symptoms and their treatment, rather than causes, and environmental sources of effects are the last considered, if ever.

There are particular environmental exposures, pesticides, and air pollutants for which the combination of increased susceptibility and increased opportunity for exposure combine to increase the hazards and risks for children. One of the most effective strategies immediately available is to prevent pollution and so preclude potentially toxic exposures.

## A Changing Environment

With the synthetic organic chemical revolution in the post-World War II period came an enormous influx of new chemical substances together with frequently unchecked releases of pollutants into the air, water, and land. Some of those pollutants, such as DDT, are particularly persistent and pervasive, as Rachel Carson eloquently warned in *Silent Spring*.

Past pollution practices, combined with inefficient use of fossil fuels, served to create an environment in which the air and waterways all too often became living laboratories for toxic damage. Love Canal symbolized what could go wrong when an elementary school was built directly over a hazardous waste disposal site.

Environmental legislation of the 1970s and 1980s responded to the public outcry against the pattern of environmental destruction in America, creating a network of laws and regulations to control it. Congress enacted hallmark statutes, including the Clean Air and Water Acts; Toxic Substances Control Act; Resource Conservation and Recovery Act; Comprehensive Environmental Response, Compensation, and Liability Act (Superfund law); and Safe Drinking Water Act. States adopted implementing laws and programs, in numerous cases going beyond the federal mandate.

The resulting legislative and regulatory framework delivered substantial benefits to public health and environmental protection. But what is becoming clear is that their predominant emphasis on command-and-control strategies has limits. From the point of view of protecting public health and the environment, pollution prevention is essential. Preventing pollution and consequent exposure to potential environmental risk is

all the more necessary to protect children from harm.

This is particularly important in the case of pesticides. The sheer volume of pesticide use necessitates a pollution prevention approach. In 1993, for instance, an estimated 4.23 billion pounds of pesticides were used in the United States; this total is based on the amount of active ingredients only (1). The figure includes conventional pesticides used in agriculture, wood preservatives, disinfectants, water treatment, such as swimming pool chemicals, and use in households and buildings.

## NAS Report Examines Diet, Pesticides

One of the primary routes of exposure to potential environmental risk for children is diet, underscoring the importance of scrutinizing pesticides under the two federal statutes governing their use in food—the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and the Federal Food, Drug, and Cosmetic Act.

Concern about the link between pesticide residues in food and children's health prompted Congress in 1988 to ask the National Academy of Sciences (NAS) to examine the issue. NAS established a committee to do so through its National Research Council. The committee examined scientific and policy issues confronted by government agencies, particularly the U.S. Environmental Protection Agency (U.S. EPA), in regulating pesticide residues in foods eaten by children and infants.

The resulting NAS report, *Pesticides in the Diets of Infants and Children*, issued in 1993, includes two major findings of particular importance to the protection of children from pesticide exposures (2). The

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first is that the federal government is not doing enough to protect children from exposures to pesticides. The second is that risk assessments for pesticides and toxic chemicals do not differentiate between risk to children and risk to adults, but should do so.

Children are not simply little adults, the NAS report emphasized. Children are different from adults in terms of sensitivity because they are growing and their internal organs are developing and maturing. Children are also different in terms of exposures because they have distinctly different behavioral and eating patterns.

Doing a better job of assessing risks for children requires more information about both susceptibility and exposures. Too many critical gaps in existing data persist. Although developing the needed information is a complex matter, scientists in government, academe, and elsewhere have succeeded in filling some of the gaps; and research currently underway needs continued support. At the same time, however, incorporating existing information into the assessment of children's risks must become a priority.

This article will examine how children may be more vulnerable to potential toxic effects of chemicals because of the developmental nature of their systems, behavior patterns, and environmental conditions. It will also draw on a series of examples to illustrate risk issues involving children. These include sensitivity, exposure patterns, multiple sources of exposure to the same chemicals, and multiple exposures to chemicals that can act in the same way and can affect the same child.

### Sensitivity and Pesticides

The first case illustrates the importance of considering sensitivity in determining environmental risks to children. Sensitivity, in an environmental context, is the capacity to be harmed. It varies among different populations, ethnic groups, and genetic backgrounds, as well as by age and childhood experience and development. Age-related differences have a significant effect on metabolism (or how humans handle toxic substances), physiology (or how the body works), developmental stages, behavior, and diet.

In 1981, vinclozolin was registered for use as a fungicide on fruits and vegetables, having satisfied registration criteria under FIFRA. Federal regulations allow registration only if there are "no unreasonable adverse health effects" when compared with

benefits gained. If that test is met, the risks involved are not considered unreasonable.

However, in 1988, the manufacturer of vinclozolin had important new findings from hormonal studies of rats and reported them to the U.S. EPA, as mandated by law when new significant findings of an adverse effect from a pesticide are found. The company determined that *in utero*, or during fetal development, vinclozolin was associated with feminization of the male fetus. During *in utero* development, male fetuses were developing feminine sexual characteristics.

The Health Effects Research Laboratory in the U.S. EPA's Office of Research and Development, which had been working on hormonal effects of pesticides, took a closer look at vinclozolin and determined that effects were found at doses six times lower than those reported when the pesticide was originally registered. Feminization of male fetuses, sterility later in life in the male animals, and other developmental variations were all confirmed. The mechanism of action is vinclozolin, which acts as an antiandrogen, blocking androgen effects (3,4).

As a result, no new uses have been allowed for vinclozolin, and as vinclozolin goes through the reregistration process under the 1988 FIFRA amendments, all uses currently allowed will be reassessed.

The potential implications of the effects of vinclozolin for children are inconclusive, but the data underscore the need for a cautionary regulatory approach and continued vigilance in regulating pesticide residues in food. Growing children are sensitive to imbalances in hormone levels, and the question of potential adverse health effects from exposure to pesticide residues in food needs to be the subject of continued research.

### Sensitivity and Wildlife

Evidence of abnormal sexual development possibly due to environmental contaminants also comes from case studies involving wildlife, raising the issue of potential implications for childbearing and children's postnatal development. Much more research needs to be done in this area.

In the highly evolved chain of ecologic connections, air, water, land, vegetation, and animals are linked in a complex web of interactions. For example, pollutants move among air, water, and land and are taken up by plants, which in turn are consumed by animals and humans. What goes into the air from near and far becomes deposited in rivers and lakes, for example, contaminating fish and fowl to varying degrees.

In recent years, scientists have begun observing marked effects on the reproductive systems of wildlife in areas that have been subjected to significant environmental contamination, provoking questions about potential implications for human beings' ability to have healthy children. Many of the instances have involved a group of widely used chemicals called organochlorines.

Perhaps the most noted case involves alligators living in Lake Apopka in central Florida (5). A number of endocrine-related effects were observed, including low hatching rates, males with abnormal reproductive tracts, and females with ovaries bearing abnormal eggs. These effects were inferred to be due to a large quantity of DDE, a potent metabolite of the insecticide DDT, which had been spilled there. DDE's effects apparently led to an imbalance between androgens and estrogens in the developing alligators, which in turn caused abnormal sexual development.

Both DDT/DDE and vinclozolin are endocrine disrupters, substances that mimic or block the action of natural hormones. DDT is an estrogen, and vinclozolin and DDE are antiandrogens. Estrogens are the group of human hormones responsible for many of the more feminine parts of sexual development throughout life, and androgens, mainly testosterone, are responsible for development of many male sexual characteristics. Both males and females naturally have levels of estrogens and androgens; it is the proper balance between the two that results in appropriate sexual development.

Endocrine disrupters such as DDT/DDE may be persistent in the environment. In theory, possible side effects of environmental estrogens and antiandrogens include abnormal pregnancy and sexual development; potential cancer risks, i.e., breast and prostate; and other diseases such as endometriosis. Many endocrine disrupters like DDT/DDE bioaccumulate or concentrate in the food chain. In humans, effects on lactation have been demonstrated. Rogan et al. (6) studied breast milk DDT levels for approximately 800 women in North Carolina. The intent was to look for health effects in children exposed to DDT in breast feeding. Researchers found no evidence of increased illnesses among the children. However, they discovered that women with the highest levels of DDT in their milk breast fed less than 40% as long as women with the lowest levels of DDT. Impaired lactation would have profound effects in circumstances where breast milk is the only safe

alternative for feeding infants (for example, when there is no safe drinking water for mixing formula).

There have also been concerns about the potential for effects on sexual development and cancer risks that might be hormonally related, such as breast cancer in women and prostate and testicular cancer in men (7).

The U.S. EPA has developed testing protocols for evaluating hormonal effects of pesticides. The revised protocols call for using extended dosing periods, testing for developmental milestones in the animals, and looking for developmental end points after birth or postnatally. The U.S. EPA's Scientific Advisory Panel, comprised of outside experts, endorsed the changes, clearing the way for them to be added to the agency's testing requirements. The new protocols will add a further layer of protection in discerning potential environmental risks to human health, but troubling questions about effects on reproductive systems remain.

### Dietary Exposure to Toxicants

On the issue of exposures, children's diets differ significantly from those of adults, the NAS report confirmed (2). They eat more fruit in proportion to their body size; they also have less varied diets. As every parent knows, as children go through the first few years of life, they develop preferences for certain foods and often only will eat those particular foods for months at a time.

But NAS found that knowledge about what children eat is much more limited than it should be. Not only are existing data for children inadequate, current information on the U.S. diet is based on surveys conducted in the late 1970s. Dietary habits have changed substantially since then. Much higher consumption of fresh fruits and vegetables is a key example.

In looking at the diets of infants and children, NAS was critical of the current system for evaluating dietary intake for children because it groups all children between 1 and 6 years of age. Data are available for children up to 1 year of age. However, the differences that exist between the diet of a 1-year-old child and that of a 5-year-old child cannot be taken into account. In assessing exposure and potential risk, NAS recommended more precision about what children eat during the first few years of life by addressing each year of life between 1 and 6 separately.

Work is underway with the U.S. Department of Agriculture (USDA) to accomplish that objective. For example,

USDA is planning to revise the national food consumption survey, called the Continuing Survey of Food Intake, to characterize more accurately consumption patterns for foods children eat most frequently.

In addition to using the USDA data, the U.S. EPA is planning to use data collected in the National Health and Nutrition Examination Survey to get a better idea of the food children eat. This survey also gathers information about young children, including those from various income and ethnic groups.

The foods children most commonly eat are identified in the NAS report (Table 1). In addition to foods that might be expected, such as milk and apples, there are some interesting and unexpected items, like coconut oil, which is in a number of processed foods, including sweet cereals that children often love.

### Exposure and Bananas

A specific example of the need to protect children from dietary exposure to risk involves the use of the pesticide aldicarb on bananas, one of the foods that many children prefer. A toddler can easily eat an entire banana. Some can eat several in a sitting, and children typically eat more of them than adults per pound of body weight.

Aldicarb is an insecticide that has been used for a number of years on fruits, nuts, potatoes, and various other vegetables. It is a systemic pesticide, which means it is taken up by the roots of the plant and ends up in the plant itself, and so cannot be removed by simply washing or peeling fruits and vegetables.

Aldicarb is a carbamate pesticide. It acts by inhibiting acetylcholinesterase, an enzyme necessary for the proper transmission of nerve impulses, and can be very toxic to humans, causing a number of effects, including diarrhea, vomiting, and changes in the function of the central nervous system.

The manufacturer of aldicarb notified the U.S. EPA in 1991 of some unexpected

aldicarb residues on bananas. Generally, the residues were below the legal limit or tolerance. "Tolerances," the NAS report noted, "constitute the single, most important mechanism by which the U.S. EPA limits levels of pesticide residues in foods. A tolerance is defined as the legal limit of a pesticide residue allowed in or on a raw agricultural commodity and, in appropriate cases, on processed foods. A tolerance must be established for any pesticide used on any food crop" (2). Tolerances are set on the basis of composite samples. Under this approach, bunches of bananas were blended and then analyzed.

The level detected using this sampling method was found to be below the legal limit. However, when bananas were analyzed one at a time, some of these bananas were found to have levels of aldicarb that were up to 10 times greater than the legal limit. When the legal limit was originally established, it was considered safe. This conclusion was based, in part, on the assumption that any exposure to aldicarb would be spread over a day. More recently, it has become apparent that a whole day's exposure could occur in a single serving. With chemicals like aldicarb, which can produce acute effects, the original legal limits may no longer be considered safe for certain age groups, such as young children.

The U.S. Food and Drug Administration checked aldicarb levels in bananas used for baby food. Those levels were very low, probably because the baby foods are made by blending large numbers of bananas. The problem was high levels of aldicarb in individual bananas that, at random, some children could end up eating. Some of these bananas were not only well above the legal limit but had levels potentially high enough to make a child acutely ill.

The U.S. EPA's dietary risk assessment found that, for the hottest bananas, the allowable daily limit of aldicarb would be exceeded by an adult eating more than one-eighth of a banana and by a child eating more than one bite of a banana. But even for bananas at the legal limit, just one-third of a banana would be an excess for a toddler and one-seventh of a banana would be above the allowable daily intake for an infant.

In 1991, the U.S. EPA and the manufacturer reached an agreement to stop the sale of aldicarb for use on bananas. The registration for bananas has since been canceled and the tolerance revoked. The company also has voluntarily withdrawn its use

**Table 1.** Eighteen foods most commonly eaten by infants.

Milk, nonfat solids	Milk sugar (lactose)
Apple juice	Bananas, fresh
Apples, fresh	Rice, milled
Orange juice	Peas, succulent, garden
Pears, fresh	Beans, succulent, garden
Milk, fat, solids	Oats
Peaches, fresh	Soybean oil
Carrots	Coconut oil
Beef, lean	Wheat flour

on white potatoes for the time being because of reasons similar to those that pertain to bananas. Sampling single potatoes revealed a few with residues at or above levels of concern. The company also agreed to reduce the amount of aldicarb used on citrus fruits. The pesticide is currently undergoing special review for groundwater concerns. The situation involving aldicarb residues in bananas is a good example of the need to monitor children's exposures to pesticides in food and to respond accordingly.

A cautionary approach holds true for food imported from abroad. There has been concern for some time about potential risk from pesticides banned or not registered for use in the United States but sometimes detected in imported food. The U.S. Food and Drug Administration (FDA), which oversees the safety of imported food, generally has found a 2- to 3-fold higher violation rate for imported than for domestic foods, usually due to residues of pesticides without tolerances rather than for pesticide residues above prescribed tolerance levels (8).

The food supply in the United States is considered the safest in the world, and adults as well as children should eat a diet high in fruits and vegetables. But efforts to ensure its safety with still higher certainty are key, particularly in view of children's patterns of food consumption and their vulnerability to toxicity.

### Variety of Exposure Routes

There are multiple sources and avenues of exposure to pesticides and other toxic substances for children (9). Food and water are obvious sources. There can be direct inhalation and contact with agents inside and outside the home. Some exposures are occupationally related, like parents carrying home chemical residues on their clothing or the transfer to breast milk of chemicals contacted at work. Still other exposures can come from discharges to the air and water, certain waste sites, and, on occasion, industrial accidents.

The task of trying to account for all exposures is complex and difficult. Pesticides, for example, can be ingested during food consumption, inhaled when present in the air, and absorbed through skin contact (Table 2). They are commonly found in food and drinking water; in the air; on lawns and gardens; in households; and, for adults, in the workplace.

Infants, for example, individually can face a higher level of exposure than adults

**Table 2.** Multiple sources and routes of exposure to pesticides for children.

	Ingestion	Inhalation	Dermal
Food	×		
Water	×	×	×
Air	×		
Lawn/garden	×		×
Household	×	×	×
Occupational	×	×	×

to the same level of toxic contaminants in drinking water (12). This would include pesticides. Although infants typically weigh only one-tenth as much as adults, they drink about one-third as much water each day. In addition, water constitutes a higher percentage of their body weight. They also have a higher daily rate of water replacement. These factors combine to increase the exposure of infants to toxic contaminants in water, compared with that of adults, underscoring the need for preventive action to protect them.

In communities with contaminated air, improving overall air quality is vitally important for disease prevention. In terms of protecting children's health, specifically, pediatric asthma is a major concern. Poor air quality conditions exacerbate asthma for children and possibly lead to an increased incidence of attacks, a number of studies have shown (12,13).

But indoor air environments cannot be ignored. There are a number of important sources of pollutants in indoor environments, including tobacco smoke, stove and fireplace fumes, household cleaners, paints and glues, and synthetic fabrics, as well as pesticides.

A U.S. EPA study completed in 1990, titled *The Non-Occupational Pesticide Exposure Study*, found that 85% of the total daily exposure to airborne pesticides comes from breathing air inside the home (11). Because of this finding, the U.S. EPA developed a new residential exposure research strategy. Developing additional information about how these exposures occur will be increasingly necessary.

Some of the ways in which children can be exposed involve hand-to-mouth behavior, like sucking on thumbs and fingers. Other behaviors include object-to-mouth, elbow-to-lawn, hand-to-surface, and elbow-to-floor. There are many permutations of these. As a result, residues that persist on such things as carpets, floors, furniture, grass, soil, and playground equipment may be sources of exposure for children.

The U.S. EPA programs that evaluate the risks of toxic substances need to pay more attention to the question of whether and how products in homes and the workplace lead to indoor air pollution problems. They also need to take a more preventive strategy. This means preventing chemicals in these products from being present in indoor environments in the first place and so precluding exposure to children and others.

### Health Effects and Lead

The medical community played an important role in uncovering the link between children's exposure to lead and the effects on their health. In decades past, paint containing lead was widely used in the interior of American homes. As homes began to deteriorate and suffered from the lack of upkeep, children frequently ingested the paint chips; this was particularly true in lower economic areas. Children experienced various symptoms, ranging from constipation and retardation to encephalopathy, with coma, convulsions, and even death. Still the link with lead was unclear.

In the 1980s, however, studies tracking children from birth resulted in credence being given to the idea that exposure to lead caused behavioral disorders and a lowering of intelligence. Further, the effects were detected at much lower levels than expected and lasted longer than expected; these exposures were at levels once thought to be safe.

Fortunately, lead levels are coming down, due to government action to reduce lead exposure not only from house paint, but also from gasoline, drinking water, and household products. There is still much to be done, however, particularly to protect children living in lower income areas.

### Role of Clinicians

Identifying children's exposures to the multitude of potential hazards is difficult. Following are cases that illustrate not only the problems involved but also the crucial role clinicians can play in helping to identify environmental sources of toxicity and responding to them.

The first case involves an 8-month-old infant girl who was diagnosed with chronic diazinon poisoning (14). A routine physical examination at 12 weeks of age found excessive muscle tone in her legs. A month later, when symptoms did not improve, a specialist examined the infant and found increased tone in both her arms and her legs. The specialist suspected a mild case of cerebral palsy and began treatment and therapy.

Fortunately, the clinician discovered several months later that the home had been sprayed with diazinon prior to the first examination. He recommended testing and diazinon residues were found in the home. The child also had an elevated urinary alkyl phosphate level, comparable to the levels found in farm workers who work with the pesticide. Alkyl phosphate is a metabolite of diazinon. Happily, 6 weeks after leaving the home environment, the child's signs resolved.

The unusual features in this case are that the clinician took the time to take the history, which revealed this exposure and understood what was needed to do the laboratory work, to identify diazinon in the home, and to find metabolites in the child's urine. Under other circumstances, this child might have gone on to have chronic neurologic damage from the exposure and no one would have known why.

The second example concerns chronic mercury toxicity in a child, as reported in *Morbidity and Mortality Weekly Reports* in 1991 (15,16). The case involves a 4-year-old child from Michigan with sweating, itching, headaches, difficulty walking, gingivitis, hypertension, and red discoloration of the palms and the soles of the feet. The physician involved suspected mercury poisoning. Fortunately, this doctor remembered the days when mercury compounds were used for teething powders. He also knew the significance of a particular array of symptoms that are characteristic of acrodynia, which is pathognomonic for, that is, always linked with mercury exposure.

What the physician learned from the patient's history was that much of the inside of the home had recently been painted with latex paint, and the family had closed the windows and used air conditioning. Since that was the only change in the environment, he investigated further and found not only elevated urinary mercury levels in the child's urine but also mercury vapors in the house. He learned

that mercury had been used as a fungicide in the paint.

Since then, the mercury compound involved has been banned for use in house paints, but this case raises the question of whether there have been a number of instances of exposure for children in the past that went unrecognized.

### Multiple Exposures

Children may have multiple chemical exposures, which are difficult to identify and evaluate. Suppose, for example, that a child's home is treated with one pesticide. Others are used to treat the child's school for pests. Still other pesticides are in the food the child eats. All may have the same mechanism of action.

Several classes of pesticides contain specific chemicals that are likely to act by the same method of action. Examples include the organophosphates and carbamates, both of which inhibit acetylcholinesterase.

It is not known how to combine the effects from these exposures and so estimate potential risk. Not known, for example, is whether these exposures are simply additive, if these pesticides sometimes inhibit each other, or if they sometimes are additive or synergistic, multiplying each other's potential effects on children.

The NAS recommended research to evaluate the issue of multiple exposures to pesticides that act by the same mechanism. Such information could lead logically to developing procedures to take multiple exposures into account in the regulatory process. Work on this issue needs to be accelerated.

There are still many unknowns about the effects of pesticides on people and on infants and children in particular. Filling the information gaps on effects and exposures, primarily nondietary exposures, is essential, but achieving that goal will take time, focused effort, and unwavering support for research dedicated to this end.

Clinicians can play an important role in accomplishing this goal through special awareness of the potential effects of pesticide poisoning. Although environmental toxicity typically is not the first item on a doctor's mind in making a diagnosis, increased alertness to environmental toxicity can be a direct route to identifying causes of disease. Parents can contribute as well by identifying possible environmental links and advising the physician involved in treating the child's health problem about them.

One of the major strategies immediately available for protecting children from exposures to environmental risk is pollution prevention. It is time for parents and schools to take a careful look at how pesticides and other toxic chemicals are used around children. Pesticide users must learn how to use the safest possible methods of pest control to prevent exposure to children. First and foremost, pesticides should not be used on a "preventive" basis but rather to treat specific pest problems. When pest control is needed, it is important to use Integrated Pest Management (IPM) techniques to avoid the use of pesticides. Around the home and around schools, this means keeping pests out in the first instance and denying them access to food and shelter. If a pesticide is needed, use the safest product available and follow label instructions carefully. Be sure that any pest control contractors are licensed. And stay out of the house or school during treatment, ventilating it well before reoccupying it. Likewise, it is important to reduce the risks of agricultural pesticides. The U.S. EPA is working with farmers and the USDA to reduce unnecessary use, encourage IPM practices, and help the transition to safer alternatives. We are working to strengthen regulation of pesticides to protect children. What these changes will mean is fewer pesticide residues in children's environments, in drinking water, and on food.

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