

# Pesticides and Inner-City Children: Exposures, Risks, and Prevention

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Six million children live in poverty in America's inner cities. These children are at high risk of exposure to pesticides that are used extensively in urban schools, homes, and day-care centers for control of roaches, rats, and other vermin. The organophosphate insecticide chlorpyrifos and certain pyrethroids are the registered pesticides most heavily applied in cities. Illegal street pesticides are also in use, including *tres pasitos* (a carbamate), *tiza china*, and methyl parathion. In New York State in 1997, the heaviest use of pesticides in all counties statewide was in the urban boroughs of Manhattan and Brooklyn. Children are highly vulnerable to pesticides. Because of their play close to the ground, their hand-to-mouth behavior, and their unique dietary patterns, children absorb more pesticides from their environment than adults. The long persistence of semivolatile pesticides such as chlorpyrifos on rugs, furniture, stuffed toys, and other absorbent surfaces within closed apartments further enhances urban children's exposures. Compounding these risks of heavy exposures are children's decreased ability to detoxify and excrete pesticides and the rapid growth, development, and differentiation of their vital organ systems. These developmental immaturities create early windows of great vulnerability. Recent experimental data suggest, for example, that chlorpyrifos may be a developmental neurotoxicant and that exposure *in utero* may cause biochemical and functional aberrations in fetal neurons as well as deficits in the number of neurons. Certain pyrethroids exert hormonal activity that may alter early neurologic and reproductive development. Assays currently used for assessment of the toxicity of pesticides are insensitive and cannot accurately predict effects to children exposed *in utero* or in early postnatal life. Protection of American children, and particularly of inner-city children, against the developmental hazards of pesticides requires a comprehensive strategy that monitors patterns of pesticide use on a continuing basis, assesses children's actual exposures to pesticides, uses state-of-the-art developmental toxicity testing, and establishes societal targets for reduction of pesticide use. **Key words:** children's environmental health, chlorpyrifos, environmental justice, neurodevelopmental impairment, organophosphates, pesticides, pyrethroids. — *Environ Health Perspect* 107(suppl 3):431–437 (1999).

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The hazards of pesticides to children's health have been the subject of intense concern in the United States since publication in 1993 of the report "Pesticides in the Diets of Infants and Children" by a committee of the National Academy of Sciences (NAS) (1). More than one billion pounds of pesticides are purchased each year in the United States (2), and the NAS report called attention to the disproportionately heavy exposures of children to these pesticides as well as to their unique biologic vulnerabilities. The report expressed concern that previous approaches to assessment of the risks of pesticides and to their regulation in the United States had not adequately considered the susceptibilities of infants and children. The NAS report noted that early exposures to pesticides can cause disease in childhood as well as in later life, including cancer, reproductive anomalies, disorders of the immune system, and neurologic and behavioral dysfunction.

The risks of pesticide exposure appear to be particularly great for the six million American children who live in poverty in inner cities (3). These children live in crowded, substandard housing, and they are at high risk of exposure to the chemical pesticides—legal as well as illegal—that are heavily applied in the urban environment (2,4). New information from New York State indicates that the greatest use of pesticides in all counties statewide is in the densely populated, highly urbanized boroughs of Manhattan and Brooklyn (5). Detailed information is scant, however, on urban children's actual levels of exposure to pesticides and on the adverse health effects that may result from these exposures.

This report has three goals: *a*) To review data on children's exposures to pesticides with emphasis on exposures in the inner-city and their relation to issues of environmental justice. *b*) To review data on the vulnerability of children to pesticides, with

particular reference to the developmental toxicity of chlorpyrifos and of certain pyrethroids; and *c*) To consider the current state of neurodevelopmental toxicity testing for pesticides and to review the adequacy and sensitivity of current test procedures.

## Pesticides

### Types of Pesticides

Synthetic pesticides are a diverse group of chemical compounds (Table 1) and include insecticides, fungicides, herbicides, and rodenticides. Most are derived from petroleum (6). In homes and apartments, pesticides control termites, roaches, and rodents. In gardens and lawns, as well as along highways and under power line right-of-ways, chemical herbicides control the growth of unwanted plants. By controlling agricultural pests, pesticides have contributed to dramatic increases in crop yields and in the quantity and variety of the diet. By controlling insect vectors, they have helped to limit the spread of disease. Thus pesticides have helped indirectly to enhance children's health (1).

But pesticides also have harmful effects (Table 1). Pesticides cause injury to human health as well as to the environment. The range of these adverse health effects includes acute and persistent injury to the

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**Table 1.** Examples of pesticides found in the U.S. inner-city environment.

	Examples	Toxic effects
Chemical group		
Organochlorines (halogenated hydrocarbons)	DDT, lindane, dieldrin, chlordane	Carcinogenic, hormonal agonists, neurotoxic
Organophosphates	Parathion, chlorpyrifos	Neurotoxic, dermatotoxic
Carbamates	Malathion, aldicarb	Neurotoxic, dermatotoxic
Pyrethroids	Cyfluthrin, permethrin, fenvalerate	Possibly immunotoxic and neurotoxic, hormonal agonists, antagonist
Herbicides		
Dipyridyl	Paraquat, diquat	Pulmonary fibrosis
Other	Atrazine, alachlor	Carcinogenic

nervous system, lung damage, injury to the reproductive organs, dysfunction of the immune and endocrine systems, birth defects, and cancer (1).

Approximately 600 pesticide active ingredients—insecticides, herbicides, rodenticide and fungicides—are currently registered with the U.S. Environmental Protection Agency (U.S. EPA). These compounds are mixed with one another and blended with inert ingredients to produce more than 20,000 commercial pesticide products. EPA estimates that each year domestic users in the United States spend \$8.5 billion for 1.1 billion pounds of pesticide active ingredients (3).

### Historical Patterns of Pesticide Use

The era of chemical pesticides began in the 19th century when sulfur compounds were developed as fungicides. In the late 19th century, arsenical compounds were introduced to control insects that attack fruit and vegetable crops; for example, lead arsenate was used widely on apples and grapes. These substances were acutely toxic (3).

In the 1940s the chlorinated hydrocarbon pesticides, most notably DDT (dichlorodiphenyltrichloroethane), were introduced. DDT and similar chemicals were used extensively in agriculture and in the control of malaria and other insect-borne diseases. Because they had little or no immediate toxicity, they were widely hailed and initially were believed to be safe (7,8).

In 1962, with publication of Rachel Carson's *Silent Spring*, the potential of the chlorinated hydrocarbon pesticides for long-term endocrine and reproductive toxicity and for accumulation in the food chain became widely known (9). Carson's work showed, for example, that DDT caused reproductive failure in eagles and ospreys, species that had accumulated large doses of DDT because of their position high in the food chain. In 1972 DDT was banned in the United States by the newly

created the U.S. EPA. Manufacture and use have continued, however, in many nations, especially in less industrially developed countries (10).

### Current Patterns of Pesticide Use

The principal classes of insecticides in use in the United States and in most industrialized countries today are the organophosphates, carbamates, and pyrethroids (8). Unlike the chlorinated hydrocarbons, these compounds are short-lived in the environment and do not bioaccumulate. However, the organophosphates and carbamates are toxic to the nervous system and cause many cases of acute poisoning in the United States each year (11). Certain of the pyrethroids are also suspected to be reproductive toxins and endocrine disruptors (12,13).

Insecticide use has declined in recent years. In part, this reduction reflects introduction of newer, more potent insecticides that are effective in smaller amounts, and in part, it reflects the adoption of pesticide use reduction strategies such as integrated pest management (IPM) (14) and sustainable agriculture. IPM programs emphasize the use of nonchemical means of pest control to replace and complement chemical pesticide use. In homes, schools, and hospitals, IPM programs incorporate the cleanup of food residues, the sealing of foundation cracks, and good maintenance. IPM programs have been successfully introduced into urban areas and have been effective in reducing pesticide applications and levels of vermin in public housing (15).

### Children's Exposures to Pesticides

Children are at risk of exposure to multiple pesticides via many routes (1). Children may be exposed to pesticides in their homes, schools, and day-care centers, as well as in parks and gardens. Approximately 90% of American households use pesticides

(4). Homeowners accounted for the purchase of an estimated 74 million pounds of the pesticides used in the United States in 1995, representing a nearly \$2 billion industry (16). Diet, including drinking water, is a second important source of children's pesticide exposure (1).

In assessing the risks to health of children's exposures to pesticides, it is necessary to bear in mind that the effects of children's multiple exposures may be additive (1).

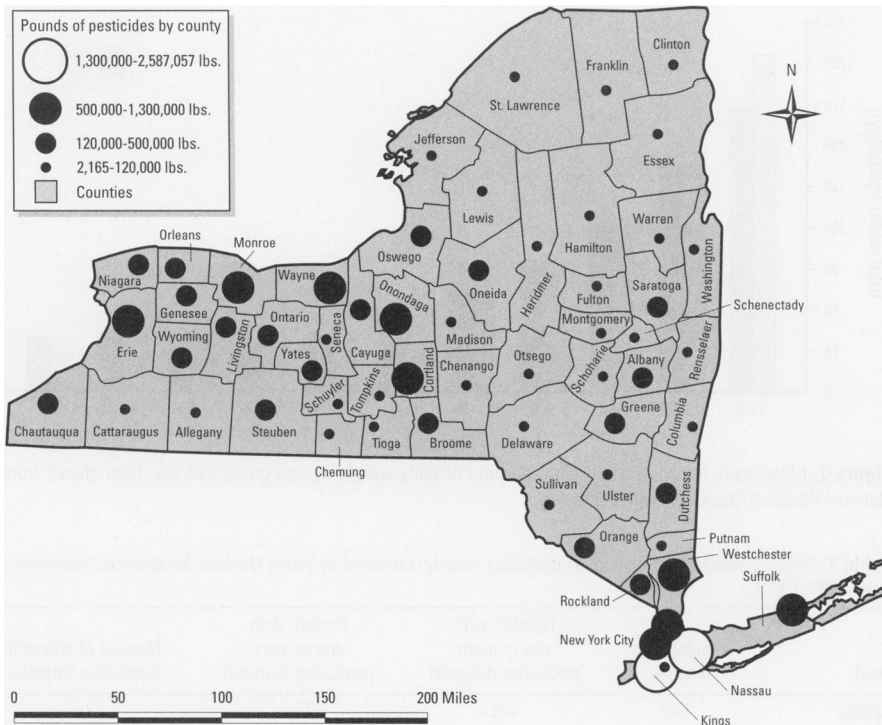
### Children's Exposures to Pesticides in the Urban Environment

The urban environment requires continuous control of insects and rodents. Moreover, urban pest control must occur in apartments and in other confined spaces where great potential exists for intimate exposure of children (15).

New statewide data for New York State, collected under legislative authority by the New York State Department of Environmental Conservation and compiled by the New York Public Interest Research Group and Environmental Advocates, show that very heavy use of pesticides occurs in the inner-city environment (5). This survey, based on reports filed by commercial pesticide applicators, found that the heaviest application of legally registered pesticides in all of New York State, including the agricultural upstate region, occurred in the two highly urbanized boroughs of Manhattan and Brooklyn (Figure 1).

Chlorpyrifos was the pesticide most heavily applied throughout New York State in 1997, including New York City (5). The number of gallons of chlorpyrifos applied in Manhattan exceeded the total number of gallons of all pesticides applied in any other single county. In the urban setting, chlorpyrifos is applied around baseboards and injected into cracks and crevices to control termites and cockroaches. It may be broadcast in rooms to control fleas. Occasionally, it is applied with a fogger. The National Nonoccupational Pesticide Exposure Study found that chlorpyrifos is one of the pesticides most widely detected in American homes. In Jacksonville, Florida, chlorpyrifos residues were found in air samples in 83–97% of homes, and in Massachusetts, chlorpyrifos residues were found in 30–40% of homes (17).

Specific data on residential pesticide use in East Harlem, New York City, are available for the 16,000 housing units in the 15 housing projects in East Harlem that are owned and operated by the New York City



**Figure 1.** Total pounds of pesticide products applied by commercial applicators and sold to farmers in 1997 in New York State, by county. Pesticide data are reported by DEC on a county wide basis, but actual pesticide use may not be uniform throughout each county. Map is updated from Thier et al. (5) by the New York Public Interest Research Group and Environmental Advocates. Map prepared by NYPIRG's Community Mapping Assistance Project (CMAP) using ArcView 3.1 provided by the Conservation Technology Support Program.

Housing Authority (NYCHA). These data were obtained by our group through annual surveys of pest control operators (PCOs) employed by the Housing Authority. PCOs routinely apply pesticides to all apartments on a monthly basis. Chlorpyrifos and cyfluthrin, a pyrethroid, are the main insecticides used by PCOs, along with lesser amounts of bendiocarb (Ficam). Chlorpyrifos has been one of the two insecticides most heavily used by NYCHA in each of the past 10 years. In 1997, between 15 and 25 gal of commercial chlorpyrifos concentrates (Dursban 4E or Dursban LO) were applied in each housing project in East Harlem. Anecdotal reports suggest that domestic pesticide use has increased in recent years in the wake of reports linking cockroach droppings to childhood asthma (18).

Organochlorine (OC) pesticides (e.g., chlordane, DDT, dieldrin, and lindane) have also been found widely in residential air and indoor surfaces in homes in cities in the United States. Many of these OCs have been banned for decades, and hence they are found more often in older homes. Typical residential concentrations of OC and other

pesticides in air range from 1–400 ng/m<sup>3</sup> leading to average exposures among children as high as 4 ng/day mm (Table 2). Chlordane has been used in 24 million U.S. homes, usually as a termiticide, and it has been detected in the home environment as long as 35 years after use (19). Children of color residing in old, poorly maintained housing are especially likely to be exposed to persistent pesticides (20).

Illegal pesticides are of great concern in the urban environment—the so-called street pesticides. For example, a very highly concentrated and illegal preparation of the carbamate insecticide Aldicarb is available in inner-city communities under the name Tres Pasitos (“three little steps,” the distance a rodent is said to be able to walk after ingesting this agent). A recent case report described a girl 2 years of age in New York City who had an acute cholinergic episode after eating Tres Pasitos (21). Another roach killer bought on street corners in East Harlem is called Tiza China (Chinese chalk); it is reported to contain boric acid. Finally, a recent episode involved methyl parathion. Eleven hundred homes in Chicago, Cleveland, and on the

**Table 2.** Estimated daily exposures to selected pesticides in two U.S. cities.

	Air (µg/day)	Diet (µg/day)
Chlordane	4,000	70
DDT	90	1,900
Permethrin	20	170
Chlorpyrifos	3,800	140

Data from Whitmore et al. (17).

Gulf Coast were illegally sprayed in 1996 with this highly toxic, restricted-use pesticide. Such incidents point to the potential for domestic misuse of pesticides in inner-city communities, with resulting extreme effects on the health of children (22).

### Residential Concentrations of Pesticides in the Inner City

Pesticides are used in 90% of homes and apartments in the United States (16).

Long persistence of pesticides in the urban indoor environment, especially semi-volatile pesticides such as chlorpyrifos, is a recently recognized phenomenon (4). Plush toys, carpets, and soft furniture are especially likely to absorb chlorpyrifos, to retain it for long periods of time, and then to release vapors to the air. In two apartments studied in a recent investigation, chlorpyrifos remained on soft, absorbent surfaces for up to 2 weeks after an experimental application. Areas affected were not only those directly sprayed; fallout was widespread (4). In such circumstances, children may receive exposures to chlorpyrifos up to 20 times the U.S. EPA reference dose (23). Other organophosphates and carbamates are often found in domestic settings: propoxur, diazinon, dichlorvos, malathion, bendiocarb, and pyrethrins (17,24,25).

Levels of pesticides in blood and breast milk of urban residents are higher in recently sprayed homes or in homes where extensive applications have been made (18,26). Children's exposures to these residual deposits are higher than those of adults, because of their normal hand-to-mouth behavior and because they crawl and sit on floors and carpets where surface residues are high (1). The skin of neonates and young children is more permeable than adult skin to lipophilic agents (27,28). Surface contamination by OCs, particularly heptachlor, can lead to dermal uptake that easily exceeds the acceptable daily intake for adults (0.5 µg/kg/day) and therefore is likely to be even greater for children. For home-use pesticides, inhalation exposures overshadow those from the diet and are higher in summer than in winter (17).

### Children's Dietary Exposures to Pesticides

A major finding of the NAS report "Pesticides in the Diets of Infants and Children" is that children have proportionately greater dietary exposures to pesticides than adults (1). Pound for pound of body weight, children drink more water and eat more food than adults. Thus they are more heavily exposed than adults to pesticides and other toxins that are present in food and water. For example, children in the first 6 months of life consume 7 times as much water per pound as does the average American adult (29) (Figure 2), and thus they are more heavily exposed than adults to pesticides in drinking water. Two behavioral characteristics of infants and children further magnify their exposures: their normal hand-to-mouth activity, and their play close to the ground (30).

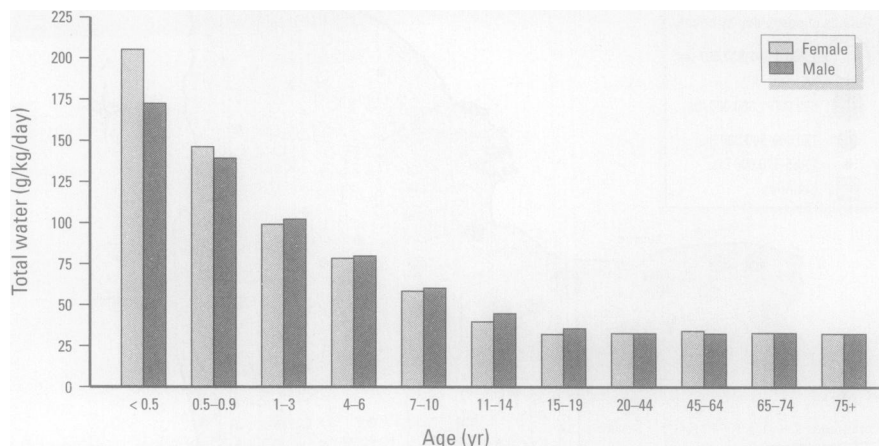
Surveys undertaken by the U.S. EPA and compiled by the Environmental Working Group of foods commonly consumed by children have shown that a high proportion contain pesticide residues (Table 3). Moreover, these studies indicate that these foods often contain residues of multiple pesticides (31). A recent report from Consumers Union has confirmed and extended these findings (32).

Pesticides have also been found in some of the baby foods most commonly sold in the United States (33). The pesticides detected in baby food included eight compounds that are toxic to the nervous system, five that affect the endocrine system, and eight that are potential carcinogens.

### The Unique Vulnerability of Infants and Children to Pesticides

In addition to being proportionately more heavily exposed to pesticides than adults, infants and children are biologically more vulnerable to them. The NAS report, "Pesticides in the Diets of Infants and Children" (1), identified three biological bases for that vulnerability.

First, children's metabolic pathways, especially in the first months after birth, are immature compared to those of adults. In some instances, children are actually better able than adults to cope with environmental toxicants. They are unable, for example, to metabolize toxicants to their active form (34). More commonly, however, fetuses, infants, and children are less able to detoxify chemicals such as organophosphate pesticides and thus are more vulnerable to them (30,35,36).



**Figure 2.** Mean daily intake of total water per unit of body weight by age group and sex. Reproduced from National Research Council (1) with permission.

**Table 3.** Pesticide residues on fruits and vegetables heavily consumed by young children. Supermarket warehouse data, 1990–1992.

Food	Number of samples	Number with one or more pesticides detected	Percent with one or more pesticides detected	Number of different pesticides detected
Apples	542	425	78	25
Bananas	368	134	36	9
Broccoli	63	16	25	9
Cantaloupes	225	78	35	19
Carrots	252	125	50	12
Cauliflower	65	26	40	13
Celery	114	85	75	13
Cherries	90	72	80	13
Grapes	313	192	61	22
Green beans	249	95	38	20
Leaf lettuce	201	136	68	22
Oranges	237	190	80	25
Peas	191	87	46	19
Peaches	246	194	79	20
Pears	328	240	73	11
Potatoes	258	120	47	17
Spinach	163	88	54	19
Strawberries	168	138	82	17
Tomatoes	395	203	51	22
Total	4,468	2,644	59	81

Data from Wiles and Campbell (31).

Second, infants and children are growing and developing, and their delicate developmental processes are easily disrupted. Their immune system is immature. Many organ systems in infants and children undergo extensive growth and development throughout the prenatal period and the first months and years of extrauterine life. Thus, if cells in an infant's brain are destroyed by pesticides, if reproductive development is diverted by endocrine disrupters, or if development of the immune system is altered, the resulting dysfunction can be permanent and irreversible. Some of the biologic mechanisms responsible for these developmental vulnerabilities are discussed in

detail in an accompanying report by Eskenazi et al. (37).

Third, because children have more future years of life than most adults, they have more time in which to develop chronic disease that may be initiated by early exposures. Exposures sustained early in life, including prenatal exposures, appear more likely to lead to disease than similar exposures encountered later. Also, deficits sustained early may persist lifelong (1). There is evidence, for example, that pre- and postnatal exposures to pesticides increase risk of childhood cancer (38), and concern has arisen that early exposure to neurotoxic pesticides may increase risk in later life of chronic neurologic diseases

such as dementia, Parkinson's disease, and amyotrophic lateral sclerosis (39,40).

Recent findings on the developmental toxicity of two pesticides commonly used in the inner-city environment, chlorpyrifos and certain pyrethroids, exemplify the special susceptibilities of infants and children. These data underscore the potential risks to children's health and development of exposures to these agents in early life.

### Developmental Toxicity of Chlorpyrifos

Recent experimental data indicate that chlorpyrifos may exhibit developmental toxicity to the fetal nervous system at relatively low doses (41-44). A study by Whitney et al. (41) found that administration of chlorpyrifos to neonatal rats at 1 day of age (approximately equivalent to human fetal exposure at 7 months of gestation) produced significant inhibition of DNA and protein synthesis throughout the brain. The authors interpreted these results as indicating "that low doses of chlorpyrifos target the developing brain during the critical period in which cell division is occurring, effects which may produce eventual cellular, synaptic and behavioral aberrations after repeated or prolonged subtoxic exposures." Similarly, another recent study found that repeated exposure of pregnant rats to low doses of chlorpyrifos resulted in long-term neurochemical and behavioral deficits in the offspring (44).

The mechanism of chlorpyrifos-induced neurotoxicity was studied by Song et al. (42), who found that chlorpyrifos evoked deficits in multiple components of the adenylyl cyclase cascade in brain cells, a system that mediates cholinergic as well as adrenergic signals. Furthermore, Campbell et al. reported that even at relatively low doses of exposure insufficient to compromise survival or growth, chlorpyrifos was found to "produce cellular deficits in the developing brain that could contribute to behavioral abnormalities" (43). Slotkin (45) reports that chlorpyrifos inhibits DNA synthesis *in vitro* in cultures of fetal rat neurons; additionally, cell replication is inhibited, cell acquisition is arrested, and neurotoxic apoptosis is accelerated.

### Endocrine Toxicity of Certain Pyrethroids

Pyrethroids are widely used as replacements for chlorpyrifos and other organophosphate and carbamate pesticides, especially in New York City. They have also been used

to control body lice and scabies as a replacement for more toxic agents such as lindane. Their acute and chronic toxicity is generally low, but there are reports of neurologic and respiratory reactivity to certain pyrethroids (46,47). Recently, our center has reported hormonal activity for certain pyrethroids (48,49), suggesting that they should be investigated further in terms of their capacity to affect neurologic and reproductive development.

### Assessing the Toxicity of Pesticides to Children's Health and Development

Protection of children's health against the hazards posed by pesticides requires that all pesticides be thoroughly assessed for their potential to cause toxicity. Toxicity testing provides the scientific basis for risk assessment and for pesticide regulation. Pesticide standards, termed tolerances, are only as protective of health as the testing upon which they are based (1).

To assess the health hazards of pesticides, the U.S. EPA [under the Food Quality Protection Act (FQPA) of 1996, the current federal legislation governing pesticide use in the United States] requires pesticide manufacturers to conduct a series of tests on laboratory animals and in tissues *in vitro*. Health risks to humans are then inferred from these experimental results.

In their 1993 report, the NAS Committee on Pesticides in the Diets of Infants and Children was critical of the test protocols for pesticides then mandated by the U.S. EPA (1). The committee was concerned that those protocols did not accurately or sensitively assess the neurodevelopmental and other developmental toxicities of pesticides. The Committee recommended more thorough developmental toxicity testing of all pesticides. Accordingly, the FQPA requires that the special vulnerabilities of infants and children be considered in setting all pesticide standards. Moreover, when data on the developmental toxicity of a pesticide are not available, the U.S. EPA is required as a default provision under FQPA to incorporate an extra 10-fold safety factor into the relevant standard for the protection of children's health.

The U.S. EPA Office of Prevention, Pesticides and Toxic Substances has issued guidelines for a set of tests to be performed on animals and cells to estimate the possible toxic effects that pesticides might have in humans—"The Health Effects Test Guidelines." This listing includes 59 different tests that had previously appeared

under various government documents and now have been combined into a single list. Generally, the U.S. EPA requires manufacturers of food-use pesticides to perform the following initial set of basic screening tests on all pesticides (50):

- Acute toxicity set: oral, dermal, inhalation, eye and skin irritation, dermal sensitization
- Subchronic (90 days) feeding studies in rat, dog
- Mutagenicity battery
- Structural chromosomal aberrations
- Metabolism study in rats
- Two-year chronic/carcinogenicity study in rats
- Developmental toxicity study in rabbits and rats
- Reproductive, 2-generation study in rats
- Developmental neurotoxicity study (as needed)
- Acute delayed neurotoxicity—hen (required for organophosphates)

### Limitations of the U.S. EPA Screening Tests for Pesticides

A recent critical review (16) has concluded that these new U.S. EPA guidelines still fail to adequately or sensitively detect the effects of pesticides on fetal and early childhood development.

A very serious limitation in the current array of screening tests required by the U.S. EPA is that they do not routinely require full-scale neurodevelopmental toxicity testing, nor do they routinely require functional assessment of effects on the nervous system, even in the case of neurotoxic pesticides (16). Moreover, it is not clear when and which tests of neurotoxicity are mandated for particular classes of pesticides. This wide flexibility in the implementation of developmental neurotoxicity testing allows for the perpetuation of data gaps and thus for continued use of potentially dangerous pesticides.

A second problem is that most tests required for assessment of toxicity of food-use pesticides are performed only in adult animals (16). Previous studies have shown, however, that toxic effects on the developing brain cannot be predicted by testing pesticides on adult animals. In some instances the window of vulnerability to specific neurodevelopmental effects can be quite narrow (51,52). Similarly, the time at which effects are assessed after exposure can be quite crucial. These points are illustrated by the data presented above on the developmental toxicity of chlorpyrifos and pyrethroids (41-49).

A third limitation on the U.S. EPA current test protocols is that they do not follow animals exposed in infancy over the duration of a lifetime. Thus they cannot address the hypothesis that some cases of degenerative diseases that typically develop in later life, such as Alzheimer or Parkinson disease, may result from the accumulation of toxic damage to the brain that begins in infancy and extends over a lifetime (38,39). The biologic basis for this hypothesis is the concept that the death of brain cells may not affect neurologic function or behavior during childhood when there still exists considerable cellular reserve, but that as the child ages and becomes an adult, the continued dying-off of brain cells may unmask damage that was inflicted by toxins during childhood. Extending the observation period of experimental animals through sexual maturity and into late adulthood would enhance the possibility of observing such changes. Full examination of these issues may require use of nonhuman primate test systems.

The U.S. EPA fails to routinely require that food-use pesticides be tested for toxicity to the immune system.

Neither existing tests nor tolerances based upon their results fully reflect pesticides' potential to disrupt the endocrine system.

The U.S. EPA requires no testing, nor do specific test guidelines exist, to assess the possibly interactive effects of multiple pesticides. Yet children are exposed to pesticide mixtures on a daily basis.

The U.S. EPA performance to date under FQPA has been disappointing. For example, of the first 100 pesticide tolerances reviewed under the act U.S. EPA required the imposition of an additional safety factor in the case of only 12 despite wide gaps in neurodevelopmental and other toxicity testing data (53).

## Conclusion

The six million children who live in poverty in inner cities in the United States are a group within the American population that is at disproportionately high risk of exposure to numerous environmental toxins. These children's heavy exposures to lead, air pollution, and hazardous waste sites, as well as their disproportionately high prevalence of lead poisoning and incidence of asthma (18), have all been noted previously (54). Now to the list of these children's excessive exposures must be added pesticides.

Heavy applications of pesticides have been required in inner-city neighborhoods

because of the age and poor maintenance of the urban housing stock. The resulting heavy exposure of inner-city children to pesticides is therefore a direct consequence of poverty, overcrowding, and poor housing and must therefore be viewed as yet another manifestation of the environmental injustice that these children suffer (55).

Developmental toxicity is the major threat posed by the exposure of fetuses, infants, and children in the inner city to heavy levels of pesticides (1). The concordance of young children's disproportionately heavy exposure to pesticides, coupled with their developmental vulnerabilities, places them at seriously increased risk for neurologic, endocrine, and other developmental disabilities. The emerging data cited in this report on the previously unrecognized developmental hazards of chlorpyrifos (41-45) and pyrethroids (46-49), two of the pesticides most heavily used in inner-city environments, underscore this concern.

The prevention of neurodevelopmental toxicity caused by pesticides among inner-city children will require a coordinated program of research, public health surveillance, and prevention. The Center for Children's Environmental Health and Disease Prevention Research at the Mount Sinai School of Medicine, supported by the National Institute of Environmental Health Sciences and the U.S. EPA, is dedicated to the mission of reducing the exposure of inner-city children to neurotoxins, particularly to pesticides. The center plans to undertake a coordinated interdisciplinary program. Basic research within the center will explore the effects of pesticides upon gonadotropin-releasing hormone neurons in the developing brain. Other basic research will explore differences in children's susceptibility to pesticides that result from genetically programmed polymorphisms in the enzymes that regulate pesticide metabolism. A prospective epidemiologic study of infants born at the Mount Sinai Hospital will assess the effect of prenatal exposures to pesticides, polychlorinated biphenyls, and lead on children's development. Mother-infant pairs will be enrolled in the study during pregnancy. Maternal pesticide levels will be assessed through measurement of urinary pesticide metabolites during pregnancy, and the developmental trajectories of the babies will be assessed prospectively. The center embodies a community-based intervention project that will evaluate objectively the effectiveness of introducing IPM programs to East Harlem.

Nationally, the protection of inner-city children against the hazards of pesticides will require a comprehensive strategy that embodies the following four elements:

- Monitoring of pesticide use in inner-city communities on a continuing basis. The new pesticide-use registry in New York State (5) is an excellent model for such surveillance, and similar registries need to be established across the United States.
- Assessment of children's actual exposures to pesticides through a combination of environmental assessment and biologic monitoring.
- Utilization of state-of-the-art developmental toxicity testing, including full-scale functional neurodevelopmental and endocrine toxicity testing.
- Establishment of societal targets for pesticide use reduction through introduction of IPM and other sustainable programs.

To effectively protect inner-city children against pesticides, it is essential that our society adopt new approaches that complement and build on risk assessment, especially programs for reduction of pesticide use. Risk assessment has for the past 20 years been the predominant paradigm used by regulatory agencies to control exposure to pesticides. Although it has had its successes, it is an inherently slow process that typically proceeds by considering only one chemical at a time. Moreover, a perennial problem is that pesticides under assessment remain on the market until assessment is completed. As demonstrated by the recent data from New York State (5), overly heavy reliance on risk assessment to the virtual exclusion of other approaches to pollution prevention has not been successful in limiting the pesticide exposures of urban children.

A complementary paradigm for controlling exposure to pesticides that must be used in concert with risk assessment is pollution prevention. Under this approach, specific numerical targets are set for reducing pesticide use over a span of years. The principal approach to reduction of pesticide use in the inner-city environment is IPM. A study undertaken in public housing in inner-city Chicago has demonstrated that IPM can potentially reduce the use of pesticides in the urban environment by as much as 50% without reduction in effectiveness of control of vermin (15). Strategies for the reduction of pesticide use have also proved successful and cost effective in hospitals, schools, and day-care centers.

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