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THE NEED FOR FURTHER RESEARCH
ON CHILDHOOD PESTICIDE EXPOSURE

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by

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The Need for Further Research on Childhood Pesticide Exposure

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ABSTRACT. Because of their activity patterns and physiological characteristics, children are particularly vulnerable to health risks resulting from pesticide exposure. Although existing studies suggest statistically significant associations between chemical exposure and health effects, the true magnitude of the risks is unknown, and there is a paucity of pesticide-related research and legislation involving this sensitive population. This paper discusses reasons for the vulnerability of children to pesticide exposure; describes health effects resulting from such exposure; summarizes several relevant existing studies; and outlines the need for further exposure assessment, health research and policy involving pesticides and children.

Introduction

The five components which comprise the complete human health risk model are pollutant source identification, pollutant fate and transport characterization, human exposure analysis, dosage estimation, and health effect calculation. Because each component is sequentially dependent on the preceding one, obtaining accurate estimates of each is crucial for determining the extent to which identified pollutant sources should be controlled in order to reduce associated health risks.¹ Although environmental regulatory programs have recognized the need to protect public health from the adverse effects of pesticides, current legislation focuses on adults rather than children.

Working with experts on Mexican-American issues, talking with farm laborers, and reviewing the literature summarized in this paper has stimulated Stanford University Civil Engineering Department's on-going research project to accurately quantify pesticide exposure and intake dose incurred by children (in particular, 2-5 year old Latino children of farm laborers in the Salinas Valley of California). The goals of the project include expanding

the database on pesticide health effects on children; developing techniques to quantify exposure-related human activity patterns; and coding a statistical model that will use collected pesticide concentration data and videotaped activity pattern data to quantify exposure and dose for a given population. After the model is validated with data collected in a field study being conducted by the Stanford Civil Engineering Department, it may be used by public policy decision makers as a tool for evaluating the effectiveness of exposure reduction scenarios for a given population. These outputs may be used as inputs to pharmacokinetic models, which determine human health risks caused by pollutants. This paper reviews the literature examining the association between pesticide exposure and child health risks, in an effort to inspire further research in this area.

Vulnerability of Children to Pesticides

Children are more physiologically susceptible to neurotoxins in pesticides than adults are. Because the cells of children divide rapidly, chemicals become quickly incorporated into the developing organs, which are vulnerable to toxic substances. Pesticides can more readily harm developing biochemical and physiological systems because developing organs cannot detoxify foreign substances as easily as developed organs can.²

Introducing pesticides into the body can disrupt the delicate balance of chemicals required for proper functioning of the brain, spinal cord, and nerves. With their vast surface area, nerve cells provide great opportunity for attack by toxic compounds. Unlike many other cells of the human body, nerve cells do not normally regenerate. Thus, if a pesticide interacts with a nerve cell, an irreversible alteration is likely to occur. Without the blood-brain barrier that acts as a filter in the fully developed system, the brain and spinal cord of children are not well protected from toxic substances.²

Children exposed to pesticides may be at a greater risk than adults who are exposed later in life, because the time for cancer to manifest itself is greater. It is estimated that 55% of the lifetime cancer risk from exposure to carcinogenic pesticides in foods is incurred by age 6.³ Mechanisms of carcinogens in pesticides, unlike neurotoxins, are not well understood and require further study.

Exposure Opportunities

In addition to being more physiologically susceptible to health risks from pesticides, children also have greater exposure opportunities than adults because of their varied activities, playing habits, and hygiene practices. The three exposure pathways are inhalation, dermal contact, and ingestión.

Inhalation Exposure

Because pesticides are often applied as aerosols, and sprayed in the vicinity of children, inhalation exposure poses a common health threat to children. This is especially true in the agricultural setting, in which the dwellings of farm laborers are often adjacent to crops that are sprayed. In this setting, children who work or play in fields are sometimes sprayed accidentally or are sprayed by pesticide drift from neighboring fields.⁴ The results of a New York State survey illustrate an extreme case of agricultural pesticide exposure: of 50 migrant farmworker children, 36% had been sprayed in the fields, and 34% replied that the farm dwelling had been sprayed.⁵ Studies have shown that infants and small children who crawl on contaminated carpets breathe dust contaminated with high concentrations of pesticides.⁶ Cases of pesticide poisonings have also been reported for children who mowed lawns sprayed with organophosphates.^{5,7}

Ingestión Exposure

In 1989, the Natural Resources Defense Council (NRDC) published a report charging that children are exposed to "intolerable" cancer risks from pesticide residues in fruits and vegetables. Estimating that at least 17 percent of preschoolers, or 3 million children, are exposed to neurotoxic organophosphorous pesticides in fruits and vegetables above Federal safety standards, the NRDC criticized the U.S. Environmental Protection Agency for disregarding children when setting food residue standards. Because children eat more food relative to their body weights than adults do, and because children consume, on average, more contaminated fruits, they face higher risks than adults from pesticide residues.³

Ingestión exposure may also occur via contaminated beverages, drinking water, breast milk, hand-to-mouth contact, and pica behavior (soil ingestión). A study in Dallas revealed that, of 37 cases of pesticide poisonings, almost all occurred in the home, and ingestión of liquid was the most common exposure route (76%). These oral poisonings occurred when the child drank improperly stored liquid agents, contacted a contaminated container or object, or ate insecticide granules.⁷ Other studies have identified pesticides (including DDE and PCBs) in human breast milk,⁸ suggesting the possibility of ingestión exposures to children who breastfeed.

Dermal Exposure

Because children's playing habits involve contact with many surfaces, dermal exposure is thought to incur the greatest potential exposure of the three pathways. Playing with surfaces or dirt contaminated with pesticides, and contacting occupationally exposed parents provide opportunities for dermal exposure via clothing and skin. After being transferred from a surface to the skin, the pesticides may cause dermatitis before they are absorbed into the system. Skin of infants is more permeable than adult skin, so the rate of dermal absorption is higher.⁶ Also, skin conditions of infants, such as dryness and rashes, reduce the protective properties of the skin, so chemicals may be absorbed more readily into the body.⁹ A study of doses in infants, following broadcast application of chlorpyrifos in a residence infested with fleas, revealed that total absorbed doses were 1.2-5.2 times the human No Observable Effect Level, and that dermal absorption represented 68% of the total dose.⁶

Dermal exposure is influenced by factors such as the child's clothing, the hygiene practices of the child and the parents, and the cleanliness of the child's home. The amount of dermal exposure depends on how much of the child's skin contacts contaminated surfaces. Thus, the more clothing a child wears, the less dermal exposure he or she incurs. However, if the laundry facilities or practices are inadequate for removing pesticides, washing clothes may distribute the pesticides on clothes surfaces which contact the skin. Because the dose of pesticide that enters the child's body depends on how long the pesticides remain on the skin, cleanliness is another important factor in dermal exposure.

A child that bathes or washes his or her hands frequently will be less likely to accumulate high doses of pesticides than a child who cleans himself or herself less frequently. Because parents carry, hug, and hold hands of their children, the parents' hygiene is also important. Furthermore, since contaminated objects contribute to dermal exposure, a house that is cleaned (i.e., vacuumed, dusted, swept) often is likely to contribute less to dermal exposure than a house with contaminated surfaces.

Special Risks to Children of Farm Laborers

The above discussion of opportunities for pesticide exposure suggests that pesticides pose a significant risk to children in the agricultural setting. Because farmworkers are a segment of society that have low earnings, job security, education, political power, and health status, their children must often help in the fields to help the family survive economically;¹⁰ it is estimated that 25% of farm labor is performed by children in the United States.⁴ Even if farm labor children are too young to pick crops, many are brought into the fields because their parents cannot afford day care.⁴ In the fields, they inhale sprayed pesticides, eat food from contaminated crops, and play in contaminated areas. In the homes, pesticide spray drift enters through open windows, and pesticides tracked in on shoes from the fields contaminate floors on which children crawl. Because this sector lives in poverty, farm laborers often lack sanitary facilities and laundry facilities, which also increases their likelihood to incur pesticide exposure.⁵

HEALTH EFFECTS

Neurotoxic Effects

Organophosphates and carbamates are the most commonly used pesticides in agriculture and households, and their toxicology has been widely studied.¹¹ 1989 statistics from the American Association of Poison Control Centers show that among 5,947 insecticide exposures to children under age six, 5,025 were organophosphate exposures.¹⁰ By inhibiting acetylcholinesterase, the enzyme that breaks down the neurotransmitter acetylcholine, these chemicals result in nervous system dysfunctions. In adults and animals, acute symptoms of these pesticides include hyperactivity, neuromuscular paralysis, visual problems, breathing difficulty, abdominal pain, vomiting, diarrhea, restlessness, weakness, dizziness, and in severe instances, convulsions, coma, or death.¹¹ Chronic

organophosphate pesticide exposure have been associated with frontal lobe impairment, as indicated by lower scores on neuropsychologic tests and EEGs.¹³

Although extensive studies have been conducted to assess the neurotoxicologic effects of these pesticides on animals and adults, however, few have examined the toxic effects on infants and children.⁷ Since the nervous systems of children are particularly vulnerable to pesticides, it seems logical that the effects of pesticide poisoning in children are at least as detrimental as in adults.¹³ A case-control study of poisoned and unexposed children by Colorado State University showed that exposed children scored worse in tests of intellectual functioning, academic skills, abstraction, and simple motor skills.⁴

The Southwestern Medical School in Dallas conducted a retrospective study of 37 children diagnosed and hospitalized with organophosphate or carbamate toxicity at the Children's Medical Center, Dallas between June, 1975 and November, 1986. Almost all of the reported poisonings occurred in the home; 15% of the children developed symptoms within 36 hours after the residence was sprayed or fogged by an unlicensed exterminator; and poisonings occurred with almost equal frequency throughout the year. Twenty of 24 patients whose serum was tested showed increased cholinesterase activity⁷

Childhood Cancer

Although it is more difficult to quantify carcinogenic effects of pesticides than neurotoxic effects, various epidemiologic studies suggest significant links between pesticide exposure and childhood cancer cases. Before properly evaluating findings of these studies, it is necessary to consider their potential sources of bias. The following section discuss factors that may have contributed to bias in the studies of interest.

Potential Sources of Bias in Epidemiologic Studies

There are four major potential sources of bias in epidemiologic studies: sample size; selection of subjects; misclassification of exposure; and confounding factors. Because few studies pertaining to childhood malignancies

contain a large sample size, a large effect may not be statistically significant because the sample size is too small. Thus, findings from larger studies should be given more credence than findings from smaller studies.¹⁴

The method of subject selection, particularly in case-control studies, may introduce bias because the control group often consists of siblings and friends of case parents, rather than the ideal random sample of the population that gave rise to the cases.¹⁴ Non-response in telephone interviews, or inability to trace controls selected from birth certificates or medical records, will also introduce bias.¹⁵

Methods used to obtain exposure information may contribute greatly to recall and interviewer bias. These methods include: inferring exposure from clinical records; inferring exposure by interviewing the subject's parent(s); and inferring parental occupational exposure histories from interviews (especially surrogate interviews with a spouse).¹⁴ In a pilot study conducted by Stanford University in September, 1993, in which the caretaker parents of four 2-5 year old Latino children were asked to recall the details of their child's activities on the previous day, it was found that the parent's recall was inaccurate compared to data collected from videotapes. The conclusion was that using recall information may greatly distort exposure calculations, which are based on the duration of time a person spends in a location or contacting a contaminated object.¹⁶

Confounding factors, extraneous factors that are associated with cancer and the exposure status (e.g., parental occupation), may bias the outcome of an analysis unless they are accounted for. When interpreting findings from such studies, greater consideration should be placed on those which attempt to minimize potential biases.¹⁴

These sources of bias should be considered when interpreting the studies summarized in the following sections.

Colorectal Carcinoma

Colorectal cancer is a rare form of cancer in children and adolescents. Based on an extensive literature review, Pratt et al. (1977)¹⁷ found only 300 reported cases of patients under age 20 with carcinoma of the colon. The mean age at diagnosis for this type of cancer is 69 years.¹⁷ However, between September, 1974 and November,

1976, 13 adolescent patients were diagnosed with colorectal cancer at the St. Jude Children's Research Hospital in Memphis, Tennessee. Ten of these patients came from the rural areas of the Mississippi Delta, areas of high pesticide use. Nine of these subjects had been exposed to farm sprays or agricultural chemicals, and three of the nine had sprayed herbicides and insecticides. In Mississippi, the reporting of five cases between October, 1974 and August, 1975 was significantly different from the one expected case.^{17,18}

Caldwell et al. (1981)¹⁸ collected blood specimens of the 10 children for pesticide residue analysis, and also interviewed the patients and their families about personal, family, medical, work, and residential histories; dietary habits; water supplies; and exposure to pesticides and herbicides. Although they did not find an association between serum pesticide levels in the blood samples of the cases, and the controls (children without malignancies visiting the Mississippi Rheumatic Fever Clinic), they suggested that serum pesticide levels may not accurately reflect pesticide levels of colon contents; that the levels may be deceptive because specimen collection may have been far removed from actual exposure; and that exposure histories may have been imprecisely documented.

Although there is no statistical evidence for the association, both studies suggest that pesticide exposure did cause this regional increase in this rare disease among adolescents. They base this belief on the unusually high number of regional cases, on the childrens' medical histories (that do not suggest high susceptibility to colon cancer), and on the patients' histories of pesticide exposure. However, to find a statistically significant association between pesticides and colorectal cancer in children, they recommend further surveillance for additional cases.^{17,18}

Leukemia

The Children's Cancer Study Group (CCSG) conducted a case-control study to investigate an association between parents' occupational exposures and acute nonlymphoblastic leukemia (ANLL). Cases obtained from registration files of the CCSG consisted of all patients under age 18 diagnosed with ANLL between 1980 and 1984. Of the 262 eligible cases, 204 mothers consented to interviews. Controls were obtained through random digit dialing, and matched by date of birth and race.¹⁹

Both parents were interviewed over the telephone about demographic characteristics, occupation, parents' exposure histories, clinical factors, and household exposures. Household exposure results of the study are summarized in Table 1.

Table 1. Household Pesticide Exposure Results for ANLL Study¹⁹

Exposure	Frequency	Case	Control	Odds Ratio	95% Confidence Interval	P-trend
Pre-Natal (Mother's Exposure)	None	134	148	1.0		
	<1/wk	50	40	1.4	0.8-2.2	
	1-2/wk	12	15	0.9	0.4-2.1	
	Most Days	8	3	3.5	0.9-13.8	0.05
Child	None	128	148	1.0		
	<1/wk	46	33	1.8	1.0-3.0	
	1-2/wk	13	9	2.0	0.8-5.0	
	Most Days	8	3	3.5	0.9-13.8	0.04

The analysis of pesticide exposures inferred from job title revealed that pesticide exposure of the father was significant (Odds Ratio=2.3, p=0.05). Mother's occupational exposure to pesticides was also a significant risk factor (Odds Ratio=inf., p-trend=0.008). Age stratification analyses revealed that for children under 6 years of age with prolonged exposure to either parent who had been exposed, the odds ratio was 11.4 with p-trend=0.003.¹⁹

This study strongly implicates a link between parental pesticide exposures and ANLL. Relevant confounding factors, including associations between occupational exposures, and between household exposures to pesticides, petroleum products, and marijuana, were considered in the analyses by conducting stepwise logistic regression analysis.¹⁴ Because the study excluded families that were not English speaking or did not have a telephone, exposure to migrant farm laborer children were underrepresented.¹⁹

A case-control study to investigate the causes of acute lymphocytic leukemia (ALL) selected 159 cases from the Los Angeles County Cancer Surveillance Program. Controls were selected from friends of the case, where possible, and otherwise from random-digit dialing, and were matched by age, sex, race, and Hispanic origin for whites. After obtaining interviews from both parents, 123 case-control pairs were analyzed. The findings revealed an

association between childhood acute leukemia and intrauterine pesticide exposure (household: Odds Ratio=3.8, $p=0.004$; garden: Odds Ratio=6.5, $p=0.007$)²⁰. Although the study found intercorrelation among different occupational exposures, it examined the independent contributions of these factors in a multivariate analysis.¹⁴

Brain Tumors

An exploratory case control study in Baltimore, Maryland found that more children with brain tumors and children with other cancers diagnosed between 1965-1975 had been exposed to insecticides than had the control children without cancer. From 15 hospitals, 73 matched pairs of patients with brain tumors and normal controls, and 78 matched pairs of children with brain tumors and cancer controls were studied.²¹

Normal controls were selected from birth certificates at the Maryland State Health Department and matched by sex, birthdate, and race. Cancer controls were matched to brain tumor cases by sex, date and age at diagnosis, and race. The cancer controls were used to determine factors associated with both brain tumors and other cancers, and to determine if selective recall bias of parents contributed to bias in the study. Interviews of all parents were questioned about characteristics of the child, maternal characteristics and exposures, and family characteristics and exposures.²¹

With regard to insecticide exposure, the odds ratio was 2.3 ($p=0.10$) for children with brain tumors versus normal controls. There was no significant difference between children with brain tumors and children with other cancers with regard to insecticide exposure. Looking only at children living on a farm which used agricultural pesticides. Gold et al. (1979) also found an association between brain tumor risk in children under age 20 and normal controls (Odds Ratio=4.0, $p=0.04$).²¹

A similar exploratory case-control study of brain tumors in children under age 20 was conducted in Ontario between 1977 and 1983. Seventy-four cases out of 123 identified from two hospitals in Toronto were willing to participate. The 138 controls that participated were selected as follows: a random sample of 'potential fathers', i.e. males who were between ages 15 and 50 and residents in the study area during the year in which the matched case

was born, was drawn from Ontario population lists; then, from their children, two randomly selected controls were matched to each case by age and sex. Interviews administered to the natural mother included questions regarding life-time exposures of the child (e.g., water supply, X-rays, birth characteristics, and parental exposures).²²

Conditional logistic regression analysis revealed that brain tumors were not significantly associated with exposures to pesticides. The inconsistency between this study and the Baltimore study suggest the need for future studies with larger sample sizes to determine whether pesticides actually contribute to childhood brain tumors.²²

Although most of the above studies suggest associations between pesticide exposure and childhood cancer, other studies have been more controversial. One of the reasons that these studies undergo intensive scrutiny is that pesticides themselves are a controversial issue; the livelihoods of chemical manufacturers and agricultural workers depend on these chemicals. Unless studies are extensive and rigorous, they are subject to dismissal by critics. Several of the more publicly known pesticide controversies are described below.

Controversial Studies

McFarland "Cancer Cluster"

A well-known cluster of childhood cancers (leukemia, Wilm's tumor, astrocytoma, nonHodgkin's lymphoma, osteogenic sarcoma, fibrosarcoma, rhabdomyosarcoma) in agricultural McFarland, California between 1975 and 1985 suggests a link with the use of agricultural chemicals. Ten cases of cancer in children under age 15 were observed, but only three were expected. Between 1982 and 1985, one case was expected, but eight were observed. Furthermore, an excess of fetal and infant deaths occurred between 1981 and 1983.²³

Because epidemiologic studies could not find an environmental cause for the cancers, and because the observed cancers were biologically dissimilar, critics claim that there is no association. Because of the difficulties in conducting retrospective epidemiologic studies, however, a link that did exist may not be found. This example suggests the importance of collecting extensive data and conducting epidemiologic studies as close to diagnosis of the cancers as possible.

Alar

Another well-known controversy was in 1989 over the pesticide Alar. This pesticide, which was widely used on apples and found in apple juice and apple sauce, was associated with a preschooler cancer risk of 1 in 4200, 240 times the 1 in 1,000,000 lifetime risk that the EPA strives for.³ UDMH, a degradation product of Alar, was linked to hemangiosarcomas and lung tumors in 1967, 1977, and 1989. In 1985, a scientific advisory panel to the U.S. Department of Agriculture stated that existing studies did not provide enough information to conduct quantitative risk assessment for cancer from Alar. After further studies were conducted, EPA reduced allowable tolerances for Alar residues on crops. It was not until 1989, after the Natural Resources Defense Council conducted new studies that linked UDMH with the same kinds of tumors found in the early 1970s, that EPA canceled Alar's registration.⁸ Perhaps if more studies on UDMH had been conducted in the 1970s, the EPA would have banned Alar a decade earlier. This example also illustrates the importance of conducting extensive research in the area of pesticides and cancer.

SUMMARY OF RESEARCH AND POLICY NEEDS

The fact that the above studies used different sample sizes, questionnaires, and methods for selecting controls makes it difficult to determine which findings are the most reliable. Since there are biases and conflicting results, it is necessary to improve study methodologies, to expand the databases, and to conduct further studies. If more reliable research is conducted, policymakers might be more inclined to fill policy needs for protecting the health of children.

Research Needs

Further research appears necessary in the following areas: improving methods for exposure assessment, development and use of biomarkers of exposure, acute poisoning follow-up studies, and evaluation of toxic synergisms from exposures to pesticide mixtures;⁸ more evaluation of toxicity and human health effects of older pesticides on organ function, perinatal toxicity, immunotoxicity, and chronic effects such as cancer and neurotoxicity;⁸ development of better databases and methods for quantitative risk assessments; studies of

mechanisms of actions of pesticides to improve the use of existing chemicals and developing safer ones;⁸ studies of long-term pesticide exposure effects on reproduction;²⁴ quantitative measurements of pesticide penetration through dermal surfaces;²⁵ and determination of appropriate no observed effect levels (NOELs).²⁵

Policy Needs

Although children are especially vulnerable to the health risks of pesticides, they have been overlooked by the United States and other governments during establishment of pesticide concentration standards and laws regarding worker safety. Research findings regarding pesticide effects on childrens' health should be used to strengthen existing laws and create new ones in order to protect this population. The research conducted to date suggests policy reform in four main areas: policies protecting children in the agricultural setting; greater focus on policies and research related to neurotoxic effects; stricter policies and standards regarding residential pesticides and their applications; and environmental standards based on children rather than adults.

Although farm children stand at a higher risk to health effects from pesticides, this minority group has substandard access to medical care. A study of 262 migrant workers in Wisconsin revealed that fewer than half of the migrant children under age 16 had an annual physical checkup. Only 55% of the preschoolers had been immunized for DPT, Polio, Measles, Rubella, Smallpox, Mumps, and TB.¹⁰ Despite federal child labor laws, many migrant farmworker children work in pesticide-laden fields, where they apply pesticides themselves, play in the fields, or spend their time in sprayed homes or contacting contaminated objects in the agricultural setting.

Stricter enforcement of existing state and federal laws, such as child labor laws and re-entry intervals in the field is needed. In addition to giving more resources and priority to biological monitoring and epidemiologic studies of farmworkers and their children, the government should support farmworkers and their unions to make the fields and farm dwellings safer for children.²⁶ Efforts should be made to provide adequate child care facilities for children of farmworker and other rural parents, so as to eliminate exposure opportunities in the agricultural setting.⁴

The Office of Technology Assessment has recommended the following mandates by Congress related to neurotoxic substances: more neurotoxicity testing under the Toxic Substances Control Act and the Federal Insecticide, Fungicide, and Rodenticide Act; greater focus of regulatory concerns on neurotoxicity; enhanced research programs in federal agencies; provision of more funds for research; and improved public education (e.g., improved labeling of consumer products; distribution of information about exposure) concerning risks of toxic substances to the public.²

After concluding that current federal regulatory requirements for indoor pesticide applications are inadequate based on high infant doses of chlorpyrifos, Fenske et al., 1990⁶, recommended several strategies for minimizing exposures to infants. These include the following: establishment of notification requirements to inform residents of potential health risks; recommendations for unconditional thorough ventilation of the house prior to re-entry; well-defined re-entry periods beyond the current insufficient 1-2 hours, with special warnings that infants and small children (and pets) should not contact treated surfaces; development of application of surveillance programs and more efficient training and licensing procedures for applicators; and increased testing, modification or withdrawn registrations of household pesticides.⁶

The NRDC recommends that the EPA reassess its procedures in risk assessment techniques based on risks to children. As of 1989, the EPA was using the USDA's 1977 food consumption data rather than the 1985 NRDC survey data in setting standards for pesticide residues. Between 1977 and 1985, preschoolers' fruit consumption increased by 30%.³ Although the EPA is moving in the direction urged by the NRDC, re-registration of the 300 pesticides on the market will not be completed for at least five years.³

CONCLUSIONS

Many of the above recommendations for future research and policy changes have assumed that pesticides are used, and that we as a society must protect our children from them by altering human behavior (e.g., not reentering homes or fields for a specified interval; supervising application of indoor pesticides). Another approach to the problem is to seek alternatives to chemical pesticides, such as organic farming, or implementation of integrated

pest management techniques which minimize use of chemicals. Until such alternatives to chemical pesticides become more accepted and widely used, however, it appears necessary to protect the health of children from the adverse effects of pollutants of earlier days that persist in the environment and of pollutants that continue to be applied at alarming rates.

Although numerous studies demonstrate a statistically significant association between pesticides and health risks to children, there is a need to strengthen the argument by filling gaps in the complete human health risk model, which was discussed in the introduction of this paper. Policymakers may be more inclined to promulgate laws aimed at protecting children from the effects of pesticides if data relating a pesticide source, its movement through the environment, its specific contact with a target population, and its penetration into human body tissues and fluids can be synthesized. Thus, in addition to an epidemiological approach in which the health effects are related to common factors of the target population (risk to source), exposure assessment studies which quantify health risks from the other direction (source to risk) would enhance the evidence that children must be protected from pesticides. The exposure assessment approach also allows scientists and policymakers to identify behaviors that can be modified and sources that can be mitigated for specific populations.

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