Pesticide Exposure and Children

Part 1: Why Focus on Kids?

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Dr. Richard Fenske, Professor of Environmental Health, UW

As the year 2000 begins, the link between pesticides and children has become a high profile issue for consumer groups, the chemical manufacturing industry, and government agencies. Even Congress is getting into the act. "Pesticide risk unknown at schools" reads the headline of an Associated Press article published in the January 5 *Seattle Times*. Senator Lieberman from Connecticut, Senator Torricelli from New Jersey, and our own Senator Murray are proposing new legislation that would require schools to notify parents before pesticides are used. Their concerns have been spurred by a recent General Accounting Office report which concluded that little is known about pesticide use in schools and the potential exposure of children.

Once again we find ourselves confronted with controversy and uncertainty about the health risks of pesticides. We have stepped into what has recently been called the "risk information vacuum" by two Canadian academics, Douglas Powell and William Leiss, in their book*, Mad Cows and Mother's Milk: The Perils of Poor Risk Communication* (McGill-Queen's University Press, 1997). It is always refreshing to view U.S. risk controversies through the eyes of our northern neighbors. Powell and Leiss sit outside the fray, and are able to take a more sanguine look at the heated health risk debates that seem to thrive in this country.

If we imagine the risk information vacuum from a chemist's perspective, we might see three sealed glass vessels in a line connected by stopcocks. The central vessel is a vacuum; one of its neighboring vessels contains various types of scientific knowledge; its other neighbor contains a mix of anecdotal information, speculation, anxiety, and even dread—let's call it "caution." If the two stopcocks are opened simultaneously, some combination of knowledge and caution will fill the vacuum. With pesticides and children we have a low concentration of scientific knowledge, but plenty of caution, so the vacuum quickly fills with a lopsided mixture. Until more scientific knowledge can be developed to supplant caution, the controversy continues.

Problems with Fleas

My own concern about children and pesticides was sparked by a series of informal side meetings held at national conferences of the American Chemical Society, starting about 1986. At the time I was at the Agricultural Experiment Station at Rutgers University in New Jersey, and my work dealt with fluorescent tracer evaluation of exposures during pesticide applications. In these meetings scientists from government, industry, and academia got together to discuss what we came to call the "indoor occupant exposure" issue. Our attention focused quickly on the use of indoor broadcast spraying and "bombs" (total release aerosol canisters) to control fleas. Several organophosphates and carbamates with moderate acute toxicity were registered for this use. Scientists at North Carolina State University (Wright, Leidy, and others) had done some controlled spraying in dormitories, and measured residues of such compounds as chlorpyrifos and diazinon. Scientists at Dow had also conducted a study of broadcast spraying of Dursban™. But none of these studies had systematically estimated risks to children. What kinds of risks did these treatments pose, we wondered? Scientists from one major chemical manufacturer had done some controlled spraying with their product, and concluded that the possible risk for a crawling infant in a home soon after broadcast treatment exceeded their comfort zone. The company voluntarily withdrew its product registration for broadcast application around 1987.

I had done several research projects with scientists at Health Canada in Ottawa, and we soon found a common interest in this "new" issue of children's residential pesticide exposure. I was asked to develop exposure assessment guidelines for indoor environments, and in 1988 we tested the guidelines in a study with Dursban, following label instructions for broadcast treatment, and using some middle-of-the-road assumptions regarding skin contact and absorption. We published our findings in 1990 in the *American Journal of Public Health* (vol. 80, pp. 689-693), concluding that exposure levels within the first twenty-four to forty-eight hours "could result in doses at or

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above the threshold of toxicological response." Our findings were quite similar to those of the aforementioned industry scientists who had withdrawn their product for broadcast use. Seven years later Dow and United States Environmental Protection Agency (USEPA) agreed to remove broadcast and total release aerosol applications from the Dursban product label on the basis of potential risks to children.

Children's Environmental Health

If we have eliminated some of the high exposure scenarios for children and pesticides, why do concerns continue? Are children really at risk in schools? Do crack-and-crevice or lawn applications pose a hazard? To answer these questions we need to step away from the issue of pesticide safety and look more broadly at concerns about children's health. In the early 1990s a national network of public health professionals formed to focus on environmental hazards and children. The primary concern of this group was that children were being overlooked in research and health risk assessments. One result of their efforts was a 1996 Executive Order directing all federal agencies to develop an explicit strategy for including children's health in their evaluations. Now, researchers who apply for funding from the National Institutes of Health need to explain why they are **not** including children in their projects. The spotlight has clearly been shifted to reach children. Environmental health research now includes such questions as: what do children eat and how does it differ from adult diets? Where do children spend their time and how do they interact with their environment? How does hand-to-mouth activity in infants and toddlers affect exposure to environmental contaminants? Results from this research will add new knowledge to our understanding of pesticide health risks and reduce the uncertainty that currently fills the risk information vacuum. The National Institute for Environmental Health Sciences and the USEPA recently partnered in funding eight new "pediatric environmental health" research centers, one of which is here in the University of Washington's Department of Environmental Health. These new centers are part of the national effort to understand health risks in children.

TABLE 1
CDC Action Levels for Blood Lead in Children

Years	Blood Lead Level (µg/dL)
1960-1970	60
1970-1985	30
1985-1991	25
1991-	10

Children and Susceptibility

The final element of concern related to children's health is children's susceptibility to certain environmental hazards. The discovery over the past two decades of the health effects of lead on children has been instructive. Since 1960 our estimate of an acceptable lead exposure level for children has decreased steadily, dropping from 60 to 10 micrograms per deciliter of blood, according to the Centers for Disease Control and Prevention (Table 1). Some scientists believe that effects can occur from exposures below 10 µg/dL; work is underway to test this hypothesis.

The lesson to be learned from lead exposure is that children may have very different susceptibilities than adults, particularly in the very early years of life. It is well known, for instance, that infants have very low levels of the enzyme methemoglobin reductase, making them particularly susceptible to anemia, or "blue baby syndrome." (See related article in *AENews* Issue 150, Oct. 1998.) Also, the enzyme that breaks down the pesticide parathion and its oxon derivative is not fully expressed until about two years, so until that time young children are probably at elevated risk from exposure. These examples point to a need for a better understanding of developmental factors in young children. Public health is about the prevention of disease, and it is only with a solid scientific base that we can develop policies that are protective, fair, and cost-effective. Part 2 will review our recent work on pesticide exposure in children in Wenatchee.

Dr. Richard Fenske is Professor of Environmental Health at the University of Washington's School of Public Health and Community Medicine, and Director of the Pacific Northwest Agricultural Safety and Health Center (PNASH). He also serves on EPA's Science Review Board, a congressionally mandated advisory board for pesticide science policy. He can be reached at ***rfenske@u.washington.edu*** *or (206) 616-1958.*

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Pesticide Exposure and Children

Part 2: Children in Agricultural Communities

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New concerns about pesticide health risks and children in the late 1980s were the foundation for the 1996 Food Quality Protection Act. Those concerns also spawned new efforts among public health scientists. We saw the need for a better understanding of exposure if we were to produce more accurate estimates of risk. Equally important, we needed to identify special populations at high risk.

Risk is often defined as the probability of harm. Groups at increased risk are normally those who either have high exposures or enhanced susceptibility to a particular disease agent. In the case of pesticides, for example, mixers, loaders, and applicators are considered "high risk" because of the relatively high exposure that can result from direct contact with commercial products and spray. Children are considered "high risk" because of possible increased susceptibility and the ongoing development of their organ systems.

So what about children of pesticide handlers and others who work with agricultural chemicals? Aren't their risks potentially high both from the point of view of exposure and of susceptibility? Our studies here at the University of Washington School of Public Health and Community Medicine for the past eight years have tried to answer these questions. We decided that children in farming communities should be defined as a special population for research, and that we needed to find out if their exposures and risks were different from those of other children. Furthermore, we knew that children in farming communities were probably exposed to more than one pesticide, and that pesticides that work by a common mechanism of action may produce an additive or cumulative risk. In the end we decided to focus our efforts on younger children (1-6 years old), and we examined their exposure to the organophosphorus (OP) insecticides. Nearly all OP pesticides have a similar mode of action: they inhibit the nervous system enzyme acetylcholinesterase.

Finding the Children

A major challenge for population-based exposure assessment studies is defining the study population. Sometimes this is done geographically or on the basis of existing databases such as census data. Ideally, a probabilistic sample can be drawn from a well-defined population so that results can be generalized to the larger population.

Defining "agricultural communities," however, turned out to be complicated. Such communities are widely dispersed and do not always conform to census or political boundaries.

Once the community is defined, traditional methods of access to families may not be feasible. Among agricultural workers, multiple families may live in residences designed for a single family, and telephone-based sampling methods may miss a significant fraction of the population. In our state's agricultural regions the primary language of many workers is Spanish, so bilingual capabilities are essential.

The area selected for our studies centered around Wenatchee, Washington. The region consists of an urban zone along the Columbia River, with orchards extending into the surrounding mountain canyons as well as upriver, and newer residential development interspersed with farmland. This entire region was considered the "agricultural community" for our studies. Orchard management in the area includes periodic application of several OP pesticides, including azinphos-methyl, chlorpyrifos, diazinon, phosmet, and malathion.

In our recent studies we attempted probability-based sampling using census tract data, but this approach required a randomized door-to-door contact, as much of the population did not have telephone service. We also found that families were wary of strangers approaching their doors, and were often unreceptive to our request for participation. This method was ultimately abandoned as impractical. Study participants were recruited through community organizations, including social service agencies, clinics, and producer-operated cooperatives. This approach allowed us to quickly identify families with young children.

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Our studies in 1992 and 1995 divided households into two groups based on proximity to farmland and parental occupation. "Agricultural" families were defined as households that included at least one adult working in farming. Adult workers were further classified as pesticide applicators and farm workers in the 1995 study. None of the pesticide applicators in these studies conducted this activity full-time. A smaller "reference" family population was also recruited. These families had no household members working in farming, and lived more than one-quarter of a mile (about 400 meters) from farmland. Children up to six years of age were recruited from these families. Often more than one child per family would participate in the study.

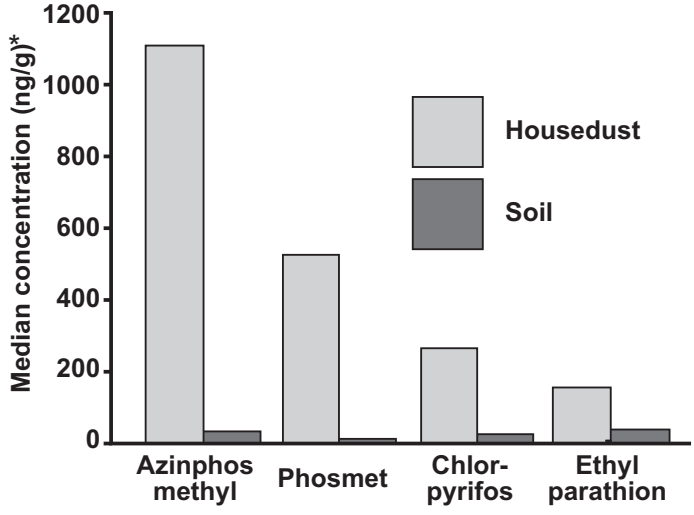
Assessing Exposures

When we began this work in 1991 there were no laboratories prepared to conduct multiple OP residue analysis in media other than food. Even acquiring appropriate standards was problematic. Our lab had to develop new analytical methods to meet our needs for environmental measurements. Our 1992 and 1995 studies focused on four OP pesticides used in Washington state orchards: azinphos-methyl, phosmet, chlorpyrifos, and ethyl parathion. We included soil and housedust sampling.

Thirty OP pesticides were registered for use in Washington State in 1998. Studies expanded to include diazinon, dichlorvos, malathion, methyl parathion, methidathion, mevinphos, ethoprop, phorate, dimethoate, and terbufos; sample media were expanded to include twenty-four-hour indoor air, indoor and outdoor surface wipes, and drinking water. Duplicate one-day diet samples were analyzed by Dr. Carol Weisskopf at the Food and Environmental Quality Laboratory at Washington State University. Nonetheless, more than half of the OP pesticides registered in Washington State still fell outside these analytical capabilities.

Biological monitoring for multiple OP compounds is also challenging. Of the thirty pesticides used in Washington, for example, only five have compound-specific urinary metabolites. The lack of specific metabolites for OP pesticides led us to measure urinary dialkylphosphates — the common metabolites of the OPs. Six metabolic products are normally measured by gas chromatography following derivatization: dimethyl phosphate (DMP), dimethylthio phosphate (DMTP), dimethyldithio phosphate (DMDTP), diethyl phosphate (DEP), diethylthio phosphate (DETP), and diethyldithio phosphate (DEDTP). It is important to realize, though, that even this more generic assay does not necessarily capture all OP compounds. Eight of the thirty OP pesticides used in Washington are not measured with this technique.

FIGURE 1



Comparison of median OP pesticide concentrations in housedust and outdoor soil. Samples collected from agricultural families living in an agricultural community in central Washington State. (Simcox et al., 1995)

***ng/g = nanograms (of pesticide) per gram (of housedust or soil)**

Pesticide Levels in Homes

The 1992 studies included soil and housedust sampling of forty-eight agricultural families and eleven reference families. Figure 1 provides median values for four OP pesticides in

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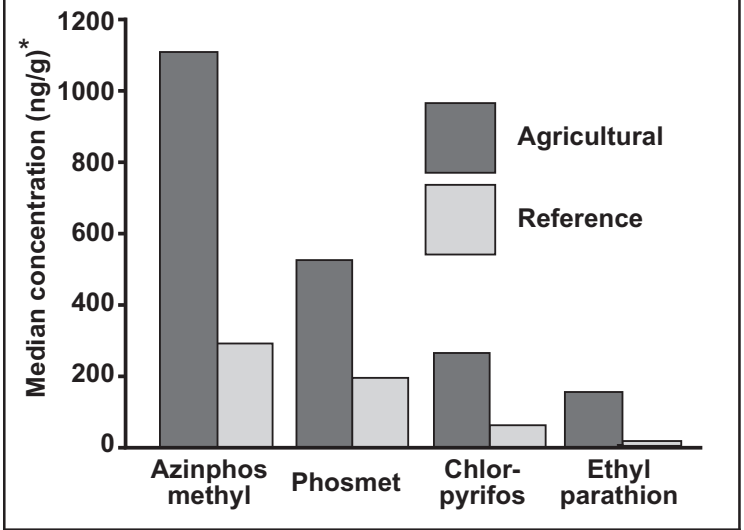
housedust and soil. These data indicated that housedust concentrations were substantially higher than soil concentrations for all compounds, and that the highest housedust concentrations were for azinphos-methyl and phosmet, both dimethyl compounds. These findings, coupled with knowledge that these children spent much of their time indoors, led to the conclusion that housedust concentration was the most useful indicator of exposure potential for this population. Figure 2 compares the OP pesticide housedust concentrations for agricultural and reference families, demonstrating that children in agricultural households had higher exposure potential than did children in reference families for all four OP compounds measured.

Our 1995 studies included housedust sampling in seventy-six homes and collection of urine samples from 109 children. An initial report of this study compared DMTP urinary concentrations of forty-eight applicator children and eleven reference children.

FIGURE 2



Comparison of median OP pesticide concentrations in housedust between agricultural and reference families in an agricultural community in central Washington State. (Simcox et al., 1995)

***ng/g = nanograms (of pesticide) per gram (of housedust)**

The patterns for metabolite concentrations were similar to those for housedust concentrations: about a four- to five-fold difference between the groups.

Our studies in 1998 included biweekly urine samples from about fifty Wenatchee children for one year, samples from 100 children in two Seattle metropolitan area communities, and a pilot multi-pathway exposure analysis in thirteen homes. We are hoping to publish results for these studies sometime this year.

What Are the Risks?

Translating the environmental and biological measurements we have collected into a meaningful statement about health risk has not been a simple task. First, we felt that the parents of the children who participated in our studies deserved clear and understandable feedback about the study results. The letters we sent to parents included specific results for their children, but also tried to answer the question, "Should I be concerned about these levels from a health standpoint?" We told parents that the levels we measured did not pose a serious or immediate hazard to their children, and that exposures were best described as "low level." We became convinced after comparing our study results with available scientific information that these children were not at risk for an acute health effect, such as substantial decrease in their nervous system enzymes. Yet when it comes to more subtle health effects, we don't have a good answer. The jury is still out. A number of studies are exploring the effects of low-level OP pesticide exposure on neurological development in very young animals. New findings will be reported periodically in the scientific literature, and will perhaps even reach the newspapers. But it will be many years before the question of long-term effects will be answered with any reasonable degree of scientific certainty. In the meantime, what do we do?

Public health emphasizes prevention as the most effective means of reducing risk. We have encouraged parents who wish to reduce their

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children's exposure to adopt some commonsense procedures: always follow pesticide label instructions, keep pesticides in a safe place in the home, remove shoes and clothing that may have pesticide residues before entering the home, and keep kids away from pesticide-treated areas, both indoors and out. We have also joined with scientists at the Fred Hutchinson Cancer Research Center to develop a study in the lower Yakima Valley to see if a community-based education program can reduce pesticide exposure in children of agricultural workers.

The debate about pesticide health risks is likely to be a long and contentious one. The scientific uncertainty that has created the current risk information vacuum means that caution will be an important principle in regulation. In the meantime, good public health practice and common sense suggest we try to reduce our children's exposures wherever possible.

Dr. Richard Fenske is Professor of Environmental Health at the University of Washington's School of Public Health and Community Medicine, and Director of the Pacific Northwest Agricultural Safety and Health Center (PNASH). He serves on EPA's Science Review Board, a congressionally mandated advisory board for pesticide science policy. He can be reached at ***rfenske@u.washington.edu*** *or (206) 616-1958.*

REFERENCE AND ADDITIONAL RESOURCES

Eskenazi B., Bradman A., Castorina R. Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environmental Health Perspectives Supplements* 1999: Vol. 107, pp. 409-419.

Loewenherz C., Fenske R.A., Simcox N.J., Bellamy G., Kalman D.A. Biological monitoring of organophosphorus pesticide exposure among children of agricultural workers in central Washington State. *Environmental Health Perspectives* 1997, Vol. 105, pp. 1344-1353.

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COOPERATIVE EXTENSION
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A monthly report on pesticides and related environmental issues

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Pesticide Exposure and Children

Part 3: Estimating Doses for Children

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In the February and March issues of *Agrichemical and Environmental News* (*AENews* Nos. 166 and 167), I shared some background on the concerns surrounding children's exposure to pesticides and I outlined University of Washington (UW) studies on children in the Wenatchee area. In the last week of April, a new analysis of our Wenatchee studies was published in *Environmental Health Perspectives*, a scientific journal sponsored by one of the National Institutes of Health (see *Editor's Note,* p. 3). Once published, a paper like this can become news, and this one did. The information released by the University of Washington Office of Health Sciences and Medical Affairs was headlined: "UW Study Finds Many Farm Children Are Exposed to Pesticides." This was translated in the *Seattle Times* on April 25, 2000, as "Kids' Pesticide Levels Unsafe."

Why did our findings draw media attention? Did our paper really demonstrate that children are exposed to pesticides at "unsafe" levels?

In our report, we tried to answer the question that parents and others ask when they learn about our studies of children and pesticides: "What are the risks? Are the levels safe?" These are not easy questions to answer.

Methodology in Brief

Our study evaluated the exposures of 109 children living in Chelan and Douglas counties. Most (91) had parents working in agriculture. The others (18) did not have any household members involved in agriculture, and lived at least one-quarter mile from treated farmland. The metabolites we measured in the children's urine are common to several organophosphorus (OP) pesticides, including azinphos-methyl and phosmet. Our approach was to convert the OP pesticide metabolites found in the urine of children to estimates of the total amount of pesticides the children probably absorbed on the day we sampled. These dose estimates were then compared to guidelines developed by the Environmental Protection Agency and the World Health Organization (Table 1). Our analysis assumed that the metabolites were the result of exposure to either azinphos-methyl or phosmet, the two chemicals found

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TABLE 1

Children's OP pesticide doses relative to the U.S. Environmental Protection Agency chronic dietary reference doses (RfDs), and World Health Organization acceptable daily intakes (ADIs) for azinphos-methyl and phosmet[1]

Regulatory Reference Value	Agricultural Children	Reference Children
	% of spray season dose estimates exceeding reference value[2]	
EPA Chronic Reference Dose (RfD)		
Azinphos-methyl (1.5 µg/kg/d)	56	44
Phosmet (11 µg/kg/d)	8.9	0
WHO Acceptable Daily Intake (ADI)		
Azinphos-methyl (5 µg/kg/d)	19	22
Phosmet (20 µg/kg/d)	3.3	0

[1]Includes all children in the study; assumes doses are attributable entirely to either azinphos-methyl or phosmet.

[2]Based on 91 estimates for agricultural children and 18 estimates for reference children.

not to generalize to other times of the year, or other regions. Nevertheless, it seems reasonable to assume that these children were exposed at these levels across the 40 to 50 days of the spraying season.

The Public Health Message

What do these numbers and comparisons really mean for children's health? The major public health message is that these findings are cause for concern, but not for alarm. We can say with some certainty that these exposures fall short of causing acute health effects, since the WHO and EPA guidelines incorporate large uncertainty factors. But it is also clear that the exposures for many of these children fall into that zone of uncertainty.

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in nearly all of the housedust samples we collected from the children's homes. The results are presented in Table 2, and a graph of dose distribution is presented as Figure 1.

Findings in Brief

We found that for children whose parents worked in agriculture as either orchard applicators or field-workers, more than half of the doses estimated for the spray season exceeded the U.S. Environmental Protection Agency's chronic dietary reference dose (RfD) and about one fifth exceeded the World Health Organization's acceptable daily intake (ADI) values for azinphos-methyl. For children whose parents did not work in agriculture the values were 44% and 22%, respectively. When we considered that the metabolites were due to phosmet exposure, we found that less than 10% of the children exceeded the EPA and WHO reference values. None of the dose estimates exceeded what is called the "no effect" level determined in animal studies. We also noted that the study took place during a period of active spraying, and we cautioned readers

TABLE 2

Spray season dose estimates[1] adjusted by daily creatinine output. Children were aged 0-6 years. Doses were based on two dialkylphosphate metabolites (DMTP and DMDTP) common to the dimethyl organophosphorus pesticides.

	Dose (µg/kg/day)			
	Applicator children (n=49)	**Farmworker children (n=13)**	**Agricultural children[2] (n=62)**	**Reference children (n=14)**
Median	2.8 [3,4]	1.2 [3]	2.0 [5]	0.3 [4,5]
25th percentile	0.8	0.6	0.7	0.1
75th percentile	4.4	4.1	4.3	3.2
Mean	3.8	2.4	3.5	2
Std. Dev.	4.6	2.5	4.2	3.1
Range	0 – 19.5	0 – 7.5	0 – 19.5	0 – 10.3

[1]Spray season dose estimates were based on the average of two samples per child. Only one child was used from each family in this analysis. All samples were collected during the May-July spraying season. In cases with missing samples, a single sample was used to estimate average dose.

[2]Agricultural children are a combination of applicator and farmworker children.

[3]Applicator and farmworker children dose estimates were not statistically different (Mann-Whitney U Test).

[4]Applicator and reference children dose estimates were statistically different (p=0.05, Mann-Whitney U Test).

[5]Agricultural and reference children dose estimates were marginally different (p=0.06).

Pesticides and Children, cont.

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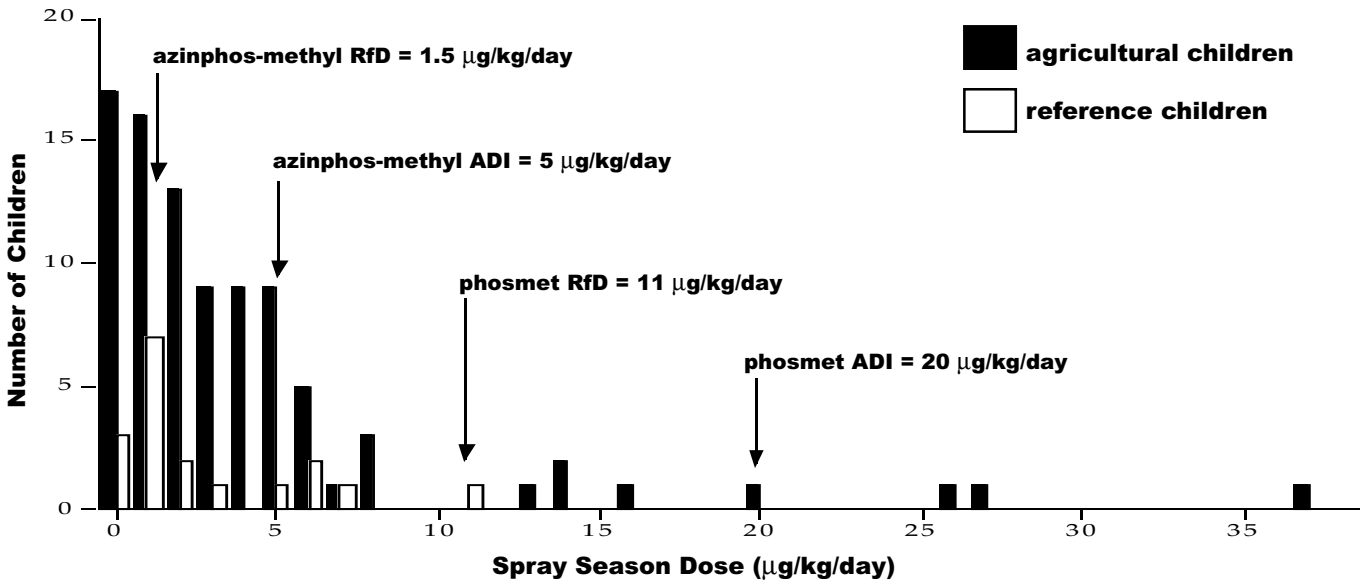
Some will argue that the current guidelines are too stringent, but others argue that they are not protective enough, particularly for children. Current regulatory methods are based on measurements of residues in food, water, and the environment, from which models are developed to estimate dose. Often these models include very conservative or protective assumptions, which can lead to high estimates and the appearance of risk that may or may not be present. Biological monitoring data are not normally used in the regulatory process, as they are very cumbersome to obtain and complex to coordinate. Yet it seems clear that they can provide a more accurate estimate of the dose that a child receives. The primary scientific message is that biological measurements, such as pesticide metabolites in urine, can give us reasonably good estimates of dose and risk. As we monitor more children we will be able to see patterns that can aid in developing commonsense and cost-effective methods to reduce exposures.

Dr. Richard Fenske is Professor of Environmental Health at the University of Washington's School of Public Health and Community Medicine, and Director of the Pacific Northwest Agricultural Safety and Health Center (PNASH). He also serves on EPA's Science Review Board, a congressionally mandated advisory board for pesticide science policy. He can be reached at ***rfenske@u.washington.edu*** *or (206) 616-1958.*

EDITOR'S NOTE: The University of Washington paper, "Biologically Based Pesticide Dose Estimates for Children in an Agricultural Community," appears in the June 2000 issue of *Environmental Health Perspectives*. General information on this publication, and abstracts of some articles, are available on the Internet at **http://ehpnet1.niehs.nih.gov/docs/**. Actual articles are available on-line by subscription only. The June article that precipitated the media attention was available electronically to subscribers the last week in April. For a printed copy of the article, you may contact Dr. Fenske at the telephone number or e-mail address above.

FIGURE 1



Distribution of OP pesticide dose estimates for children in an agricultural community, derived from urinary metabolite measurements and adjusted for creatinine concentration. Spray season dose estimates for 109 children (91 agricultural, 18 reference). The doses are expressed as micrograms of pesticide per kilogram of body weight. Arrows indicate guidelines that have been established by EPA and WHO for azinphos-methyl and for phosmet. Azinphos-methyl is a more toxic OP pesticide than phosmet, so its EPA reference dose (RfD) and WHO acceptable daily intake (ADI) levels are lower.