



## Biological Monitoring Survey of Organophosphorus Pesticide Exposure among Preschool Children in the Seattle Metropolitan Area

Chensheng Lu, Dianne E. Knutson, Jennifer Fisker-Andersen, and Richard A. Fenske

Department of Environmental Health, University of Washington, Seattle, Washington, USA

In this study we assessed organophosphorus (OP) pesticide exposure among children living in two Seattle metropolitan area communities by measuring urinary metabolites, and identified possible exposure risk factors through a parental interview. We recruited children in clinic and outpatient waiting rooms. We obtained spot urine samples in the spring and fall of 1998 from 110 children ages 2–5 years, from 96 households. We analyzed urine samples for six dialkylphosphate (DAP) compounds, the common metabolites of the OP pesticides. Through parental interviews we gathered demographic and residential pesticide use data. At least one of the DAP metabolites was measured in 99% of the children, and the two predominant metabolites (DMTP and DETP) were measured in 70–75% of the children. We found no significant differences in DAP concentrations related to season, community, sex, age, family income, or housing type. Median concentrations of dimethyl and diethyl DAPs were 0.11 and 0.04  $\mu\text{mol/L}$ , respectively (all children). Concentrations were significantly higher in children whose parents reported pesticide use in the garden (0.19 vs. 0.09  $\mu\text{mol/L}$  for dimethyl metabolites,  $p = 0.05$ ; 0.04 vs. 0.03  $\mu\text{mol/L}$  for diethyl metabolites,  $p = 0.02$ ), but were not different based on reported pet treatment or indoor residential use. Nearly all children in this study had measurable levels of OP pesticide metabolites. Some of this exposure was likely due to diet. Garden pesticide use was associated with elevated metabolite levels. It is unlikely that these exposure levels would cause acute intoxication, but the long-term health effects of such exposures are unknown. We recommend that OP pesticide use be avoided in areas where children are likely to play. **Key words:** biological monitoring, children, dialkylphosphate compounds, organophosphorus pesticides, urine. *Environ Health Perspect* 108:299–303 (2001). [Online 5 March 2001]

<http://ehpnet1.niehs.nih.gov/docs/2001/109p299-303lu/abstract.html>

Concerns regarding children's exposure to pesticides have increased in recent years with the reported association between childhood cancers and residential pesticide use or parental pesticide use in the workplace (1–4). According to the National Home and Garden Pesticide Use Survey prepared by the U.S. Environmental Protection Agency (U.S. EPA) in 1990 (5), 75% of American households use insecticides. Children may be particularly susceptible to pesticide health effects because of behavioral, dietary, and physiological characteristics associated with development (6). Children's daily activities, proximity to floors, carpets, lawns, soil, and the frequency and duration of their hand-to-mouth activity may put them at higher risk for pesticide exposure than adults (7). Infants and children also differ quantitatively and qualitatively from adults in their exposure to pesticide residues in foods (8). They have greater average daily food consumption per unit of body weight than do adults and differ in the specific foods that they eat. Moreover, the typical diet of infants and young children, including a high proportion of fruits, fruit juices, milk, drinking water, and processed foods, is less diverse than that of adults. Tissues, organs, biological systems, and detoxification mechanisms of children are undergoing rapid growth and development,

predisposing them to potentially more severe consequences of toxic chemicals. Organophosphorus (OP) pesticides have become a special concern for regulatory agencies because of their widespread use, acute toxicity, and neurotoxic properties (9).

Despite the common use of pesticides in residential environments and in agriculture, few studies have measured children's exposure levels. Some have focused on acute poisoning incidents with known or probable sources (10,11); others have examined low-level, chronic pesticide exposures in agricultural communities (12–16). There are no published studies identified to date that have examined OP pesticide exposures in children residing in urban/suburban communities. The objectives of this study were to assess OP pesticide exposure among children living in two different communities in the Seattle metropolitan area using urinary dialkylphosphate (DAP) metabolite concentrations as biomarkers, and to identify possible risk factors for OP pesticide exposure of children through a parental interview.

### Method

**Study design.** This cross-sectional study included repeated spot urine sample collection and is part of a larger study that aims to assess children's exposure to pesticides,

identify risk factors, and develop strategies for pesticide exposure reduction. Two communities located in the Seattle metropolitan area were selected for subject recruitment. Community 1 is south of the city of Seattle in King County. This area is urban and densely populated. The residents in this area are predominantly lower to middle income and many reside in multifamily dwellings. Community 2 is a suburb north of Seattle in south Snohomish and north King counties. The area is predominantly inhabited by middle- to upper middle-income families residing in single-family dwellings.

**Subject recruitment.** Families were recruited in the lobbies of a public-funded Women, Infants, and Children (WIC) clinic in community 1 and in a private pediatric clinic in community 2. The WIC clinic provides nutritional counseling and midwifery services to families meeting certain income criteria. The pediatric clinic is a group practice providing outpatient care. To be eligible for our study, the child had to be toilet trained and between 2 and 5 years old. The procedures used in the study were reviewed and approved by the University of Washington Human Subjects Review Committee; written consent was obtained from each parent, and oral assent was obtained from each child.

**Sample collection.** Participants included 58 children from 50 families recruited from community 1, and 52 children from 46 families recruited from community 2. Upon recruitment, parents were provided with polypropylene specimen cups for collecting a

Address correspondence to C. Lu, Box 357234, University of Washington, Seattle, WA 98195-7234 USA. Telephone: (206) 685-9299. Fax: (206) 616-2687. E-mail: calu@u.washington.edu

We thank S. Kong for her assistance in subject recruitment and sample collection, T. Moate for the analytical work, and the families who participated in this study.

This work was supported by the U.S. Environmental Protection Agency (U.S. EPA) through Science to Achieve Results (STAR) program (grant R819186-01) and by Cooperative Agreement U07/CCU012926-04 (Pacific Northwest Agricultural Safety and Health Center) from the NIOSH/CDC. Its contents are solely the responsibility of the authors and do not necessarily represent the official view of the U.S. EPA or the National Institute for Occupational Health/Centers for Disease Control and Prevention.

Received 11 September 2000; accepted 23 October 2000.

urine sample at home. Commode inserts were also provided for children who were unable to urinate directly into the specimen cup (usually females). If the insert was to be used for sample collection, the parent was asked to transfer the urine from the insert to the provided specimen cup. Appointments were made to pick up the children's urine samples at their residences. In some cases, urine samples were obtained at the clinic at the time of recruitment.

We collected two spot urine samples from each child. The first (spring) sample was collected from 7 May to 6 June 1998 and the second (fall) from 29 September to 18 November 1998. Spring through fall was determined to be a period of high residential pesticide use in the Seattle area, based on information gathered from local pest control and lawn care services and veterinarians. We selected these sampling periods to increase the chances of obtaining urine samples with detectable OP metabolites. For the second (fall) sampling period, specimen cups and cover letters containing abbreviated pesticide use surveys were mailed to all families who participated in the first sampling. Participants included 49 and 51 children who provided samples from community 1 and 2, respectively (10 children were lost to follow-up).

**Sample handling.** Parents were given instructions on assisting their child to collect the specimen. When samples were collected at the time of recruitment, parents gave the sample to the investigator for transport to the University of Washington laboratory. When samples were collected in their residences, the parent was asked to place the sample in the refrigerator until it was picked up by our staff. All samples were picked up within 48 hr of the void, most in less than 24 hr. All urine samples were transported on ice. Urine samples were processed immediately after arrival in the laboratory. Total sample volume was recorded and the urine was aliquoted into three centrifuge tubes with volumes of 5, 10, and 15 mL. Samples were stored at  $-20^{\circ}\text{C}$  until analysis.

**Parental interview.** An interview was administered at the time of sample pickup. We collected general information regarding the child's age and weight, parental occupation, and income level of the family. Questions regarding residential environment included home ownership status, length of time at current residence, and housekeeping practices (presence of a floor mat, frequency of vacuuming). We gathered residential pesticide use information by establishing whether the household had any pets, a lawn, or a vegetable or flower garden. We asked families if a family member or a professional had used pesticides on pets, lawn, garden, or inside their home within the previous 6 months.

We also asked which specific pesticide products were used and asked to see them if available. When possible, we recorded the product name, EPA registration number, date of application, and location where the pesticide was applied. Finally, we asked questions about the child's activities and behaviors, such as the child's frequency of hand washing, placement of hands in the mouth, and thumb sucking. A brief follow-up questionnaire was administered with the fall sample collection, which focused on insecticide use since the previous sample collection.

**Sample analysis.** We analyzed urine samples for six common dialkylphosphate (DAP) metabolites: dimethylphosphate (DMP), dimethylthiophosphate (DMTP), dimethyldithiophosphate (DMDTP), diethylphosphate (DEP), diethylthiophosphate (DETP), and diethyldithiophosphate (DEDTP). Urine samples collected in the spring sampling period were not analyzed for DEDTP because no analytical standard was available at that time.

Analysis was performed using a gas chromatograph equipped with a flame photometric detector and a splitless injector (Hewlett Packard, Palo Alto, CA) and a Supelco SPB-20 column (J&W Scientific, Folsom, CA). Sample preparation procedures included solid phase extraction, azeotropic distillation, and pentafluoro(methyl)benzylbromide (PFBBR) derivitization.

We determined the limit of quantitation (LOQ) for each DAP compound on the basis of the mean recovery of the lowest fortification level minus 3 standard deviations (SD). Metabolite residues that were less than the LOQ were designated as  $< \text{LOQ}$  and were assigned values of  $1/2 \text{ LOQ}$  for statistical analysis. The LOQs were 7.4 ng/mL for DMP, 6.6 ng/mL for DMTP, 1.1 ng/mL for DMDTP, 1.2 ng/mL for DEP, 0.7 ng/mL for DETP, and 1.1 ng/mL for DEDTP. DMTP and DETP were the most frequently detected DAP compounds in urine samples collected during the spring (70% and 71%) and fall (74% and 71%). These results were consistent with our previous study in which urine samples were collected from children living in an agricultural community (13).

**Data analysis.** The dimethyl (DMP, DMTP, and DMDTP) and diethyl (DEP and DETP) metabolite concentrations were converted to their molar concentrations ( $\mu\text{mol/L}$ ) and summed to produce a single methyl or ethyl dialkylphosphate concentration for each sample (16). Because only one urine sample contained a detectable DEDTP concentration, DEDTP was excluded from the data analysis.

The distributions for the dimethyl and diethyl molar concentrations were skewed

and were not effectively normalized using either a  $\log_{10}$  or a square-root transformation. Therefore, we performed statistical analyses with nonparametric tests using SPSS 8.0 (SPSS Inc., Chicago, IL). A focus child was selected for families with more than one child enrolled in the study, to remove within-household dependence. The primary criteria for focus child selection were contribution of two spot urine samples and acceptable creatinine measurements. A 95% confidence interval of creatinine measurement was constructed based on the urine samples collected from this study and from a previous study of 109 children ages 2–5 years old (15). Creatinine values falling within this confidence interval range were considered acceptable. If two children from the same family met these criteria, selection was random.

## Results

Participating families included in the analysis consisted of 50 families from community 1 and 46 families from community 2. The mean ages of the participating children were 3.9 years and 4.0 years for communities 1 and 2, respectively. There were 29 male (58%) and 21 female (42%) children from community 1, and 26 males (57%) and 20 females (43%) from community 2. The study population was predominantly Caucasian, and the ethnicity of the two communities was similar. The socioeconomic status of the study communities, however, differed distinctly. Community 2 participants were predominantly upper-middle income: 96% of these families (44 families) reported annual incomes above \$35,000 and resided in single-family homes. Conversely, families recruited from community 1 were primarily low to middle income: 88% of these families (44 families) reported annual incomes below \$35,000, and 74% (37 families) resided in multiunit buildings.

Eighty-six percent of the study children (83 children) had at least one measurable DAP metabolite in the spring sampling, and 92% (88 children) had at least one measurable DAP metabolite in the fall sampling. Only 1 of the 96 children had no measurable metabolites in either sample. DAP concentrations were compared across seasons (spring and fall) for each community. We found no significant differences for either dimethyl or diethyl concentrations (Wilcoxon matched-pairs signed-ranks test,  $p > 0.05$ ). We then averaged the two samples from each child to represent the DAP concentrations during the study period (May–November 1998). Table 1 provides descriptive statistics of DAP concentrations in urine collected from the 96 focus children. We found no significant differences or the median concentrations of either dimethyl or diethyl DAP

concentrations across communities (Mann-Whitney *U*-test,  $p > 0.05$ ). However, dimethyl DAP concentrations were higher than diethyl DAP concentrations in both communities. Pooling data from the two communities, the median concentrations of dimethyl and diethyl DAPs were 0.11 and 0.04  $\mu\text{mol/L}$ , respectively. Neither median dimethyl nor diethyl DAP concentrations were significantly different between male and female children (Table 1; Mann-Whitney *U*-test,  $p > 0.05$ ). The boxplot in Figure 1 indicates that there was no trend for age of the child and DAP concentration.

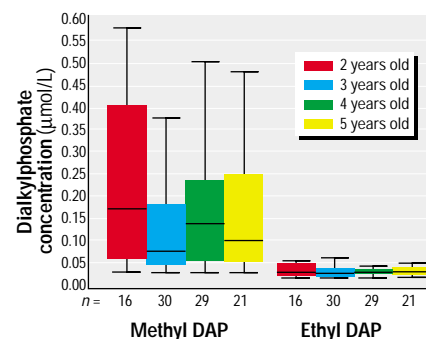
The reported residential pesticide use and the corresponding median DAP concentrations in children are listed in Table 2. Forty-nine families (most in community 2) reported having a garden, and 27 of them had applied pesticides in the garden during the previous 6 months. Only one family reported use of pesticides in the week preceding sample collection. Children living in a household with a garden had significantly higher diethyl DAP concentrations than those without a garden (Mann-Whitney *U*-test,  $p = 0.04$ ). Children had significantly higher DAP concentrations (both dimethyl and diethyl) when living in households where garden pesticide use was reported (Mann-Whitney *U*-test,  $p = 0.05$  and  $p = 0.02$  for dimethyl and diethyl DAP, respectively). We found significantly higher dimethyl DAP concentrations in children who had pets in

the household, but found no association for either dimethyl or diethyl DAP concentrations and the use of pesticides on family pets. Twenty-three families reported having their homes treated for fleas, cockroaches, or other insects, and 45 families reported using pesticides on their lawns, but children's DAP concentrations were not significantly different from those whose reported no pesticide use. Figures 2 and 3 show the boxplots of dimethyl and diethyl DAP concentrations in children's urine, grouped by different residential use of pesticides. Analysis of data gathered through parental interviews regarding child behavior and family hygienic practices did not reveal any significant associations with DAP concentrations.

### Discussion

This biological monitoring survey documents exposures to OP pesticides among children living in urban/suburban communities. The use of urinary metabolites as biomarkers provides an estimate of exposure by all routes (dermal, respiratory, and oral) and assesses actual rather than potential absorption. Common urinary metabolites that are identified after exposure to OP pesticides are the DAP metabolites that are formed when OP pesticides undergo cleavage of the leaving group with substitution for a hydrogen atom. Therefore, it is not possible to attribute exposure to specific OP pesticides when using DAP metabolites without

detailed knowledge of sources and exposure pathways. Although a few specific urinary metabolites exist (e.g., 3,5,6-trichloro-2-pyridinol for chlorpyrifos; nitrophenol for parathion), they are not yet identified for most OP pesticides. At least 39 OP pesticides are used in the United States, nearly all of which produce DAP metabolites. Thus, the DAP metabolite method provides an integrated exposure estimate for the OP pesticides. For the findings reported here, it is likely that children's exposure to OP pesticides was the result of direct exposure not only to agricultural OP pesticides in food



**Figure 1.** Dialkylphosphate (both dimethyl and diethyl) concentrations in children living in the greater Seattle area grouped by age. Concentration trend with age showed a nonsignificant difference (Kruskal-Wallis one-way ANOVA,  $p = 0.36$  and  $p = 0.64$  for methyl and ethyl DAP, respectively). Boxplot: the horizontal lines in each plot represent 10th, 25th, 50th, 75th, and 90th percentiles, bottom to top.

**Table 1.** Dialkylphosphate concentrations ( $\mu\text{mol/L}$ )<sup>a</sup> in urine samples collected from children living in two communities in the Seattle metropolitan area.

	Boys <sup>b</sup>		Girls <sup>b</sup>		Community 1		Community 2		All children	
	Methyl <sup>c</sup>	Ethyl <sup>d</sup>	Methyl	Ethyl	Methyl	Ethyl	Methyl	Ethyl	Methyl	Ethyl
Median	0.10	0.04	0.11	0.04	0.10	0.03	0.11	0.04	0.11*	0.04*
Mean	0.19	0.05	0.18	0.04	0.17	0.04	0.20	0.05	0.19	0.05
CV (%)	100	125	89	100	94	75	100	100	95	80
<i>n</i>	49	49	47	47	50	50	46	46	96	96
Min–Max	0.04–0.93	0.03–0.31	0.04–0.72	0.03–0.24	0.04–0.59	0.03–0.20	0.04–0.93	0.03–0.31	0.04–0.93	0.03–0.31
10th Percentile	0.04	0.03	0.05	0.03	0.04	0.03	0.05	0.03	0.04	0.03
25th Percentile	0.06	0.03	0.06	0.03	0.05	0.03	0.07	0.03	0.06	0.03
75th Percentile	0.28	0.05	0.24	0.05	0.25	0.04	0.25	0.05	0.25	0.05
90th Percentile	0.47	0.09	0.45	0.06	0.45	0.06	0.48	0.10	0.45	0.07

Abbreviations: CV, coefficient of variation; Max, maximum; Min, minimum.

<sup>a</sup>Concentrations were the average of spring and fall data. <sup>b</sup>Includes both communities 1 and 2. <sup>c</sup>Methyl is sum of DMP, DMTP, and DMDTP concentrations. <sup>d</sup>Ethyl is sum of DEP and DETP concentrations. \* $p < 0.001$  (Wilcoxon matched-pairs signed-ranks test).

**Table 2.** Residential use of pesticides and the corresponding median dialkylphosphate concentrations ( $\mu\text{mol/L}$ ) in children living in the Seattle metropolitan area.<sup>a</sup>

Question	Dimethyl DAP concentration ( $\mu\text{mol/L}$ )			Diethyl DAP concentration ( $\mu\text{mol/L}$ )		
	Positive response ( <i>n</i> ) <sup>b</sup>	Negative response ( <i>n</i> ) <sup>b</sup>	<i>p</i> -Value <sup>c</sup>	Positive response	Negative response	<i>p</i> -Value <sup>c</sup>
Do you have a flower/vegetable garden?	0.14 (49)	0.08 (46)	0.11	0.04	0.03	0.04
Do you apply any pesticides in your garden?	0.19 (27)	0.09 (22)	0.05	0.04	0.03	0.02
Do you apply any pesticides in your lawn?	0.14 (45)	0.09 (48)	0.13	0.04	0.04	0.68
Does this household have any cats or dogs?	0.16 (40)	0.09 (56)	0.04	0.04	0.04	0.40
Are any of the following used on your cats and/or dogs? (flea powder, flea collar, or shampoo) <sup>d</sup>	0.15 (18)	0.18 (18)	0.80	0.04	0.03	0.14
Since January 1998, has this home been treated for flies, fleas, cockroaches, or other insects (this includes products like Raid, fly strips, etc)?	0.11 (23)	0.11 (73)	0.35	0.03	0.04	0.27

<sup>a</sup>Concentrations were the average of spring and fall data. Seattle metropolitan area comprises communities 1 and 2. <sup>b</sup>Number of families who responded. <sup>c</sup>Mann-Whitney *U*-Wilcoxon rank-sum *W* test. <sup>d</sup>Four families who owned a dog or cat did not answer this question.

but also to other OP pesticides that are commonly used in residential environments.

Urinary metabolite measurements of environmental contaminants in adults are routinely corrected for differences in urine flow rate by using creatinine measurements or specific gravity. Normalization using creatinine is based on the assumptions that creatinine concentration is inversely proportional to urine flow and that creatinine excretion is independent of urine flow. However, it is known that both exogenous (diet and exercise) and endogenous (age, sex, muscle mass) factors can affect creatinine elimination (18). The increase in creatinine excretion from infancy to adulthood correlates with the growth of muscle mass and may further complicate the use of creatinine measurements. Because children are a much less homogeneous population than adults, the appropriateness of normalizing urinary metabolite values using children's creatinine measurements is unknown at this point. Therefore, in this study, we used creatinine measurements to determine the quality of urine samples, rather than to adjust data.

Data obtained from the parental interview and follow-up questionnaire helped identify factors that may influence a child's pesticide levels. In general, the survey achieved its purpose of obtaining information relevant to the scope of this study. Parents were able to answer most of the questions with certainty, but some households provided much more detailed information in their responses than did others. When parents were asked about residential pesticide use, they were not normally able to provide information on the type of pesticide used and the frequency of use. There may have been some recall bias in the reported frequency of use of pesticides. Unless the application had occurred recently, the parent

had trouble remembering when and where a pesticide had been used. We asked parents about home pesticide use within the previous 6 months. In many cases, the parent did not know the name of the product used. Often the parent being interviewed was not the parent who had applied the pesticides. If the product was still on hand, we asked to see the product and then recorded important information about the product, such as the active ingredients and the EPA registration number. In some of the few cases where lawn services were used, we were able to obtain information on products applied from the service.

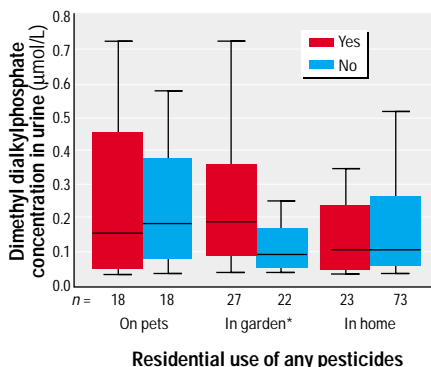
The results from this study indicate that nearly all children sampled in the Seattle metropolitan area had measurable DAP metabolites in their urine and that 70–75% had one of the two major metabolites (DMTP or DETP). The frequency of detection of DAP metabolites in this study was greater than in our previous study, in which a less sensitive analytical method was used for DAP analysis (13). However, this result was within the range found by Hill and colleagues (19), in which specific urinary metabolites for two OP pesticides, chlorpyrifos and parathion, were measured in 82% and 41% of samples, respectively, collected from 1,000 adults living in the United States.

The most striking finding from our study was the association between reported residential pesticide use and elevated DAP metabolite concentrations in children. Children whose families reported pesticide use in their gardens had significantly higher diethyl DAP concentrations than those who had gardens but did not use any pesticides. The association was also significant but weaker for dimethyl DAP compounds. According to the administered survey and our observations, 10 of 27 families who

reported using pesticides in their gardens used either chlorpyrifos or diazinon, both diethyl OP pesticides. We found this association of increased DAP levels with OP pesticide use in the garden despite the fact that the families may not have applied pesticides for months. Pesticide residues residing in outdoor soil can be tracked easily into the indoor environment and settle into the carpet along with other house dust, where it may degrade more slowly (20,21). Ingestion of soil or house dust containing pesticide residues may contribute to the exposure of young children because children spend more time on the floor than adults and may engage in hand-to-mouth and object-to-mouth behaviors.

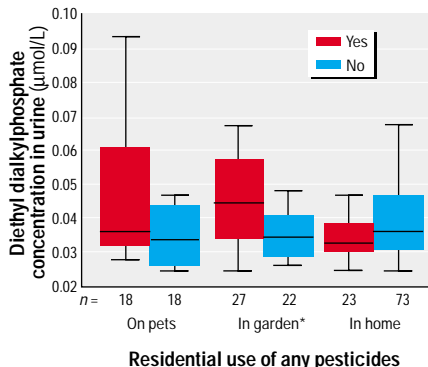
Socioeconomic indicators, such as annual household income and housing type, were not useful predictors of children's exposure to pesticides in this population. One child's parents in community 2 reported buying exclusively organic produce and did not use any pesticides at home. This child was the only subject whose urine samples showed no measurable concentrations of any of the DAP metabolites in the spring and fall samples. In this study, we found no statistically significant differences in DAP levels during the spring and fall. Many families who reported the use of pesticides during the spring sampling period continued to use pesticides through the fall sampling period, mostly in the garden. Depending on the climate in different regions in the United States, pesticide use patterns in the residential environment may vary. According to the 1997 Washington State Department of Health annual pesticide incident report, 48% of reported health complaints were associated with nonagricultural pesticide use and most incidents occurred during the spring and summer months (22). A similar seasonal trend was suggested by a study conducted to measure nonoccupational exposures to pesticides for residents of Jacksonville, Florida, and Springfield, Massachusetts (23).

Neither age nor sex was associated with children's exposure to pesticides in this study. In our previous study (13), a marginally significant trend of increasing DMTP concentration was observed with decreasing age, suggesting that activities associated with a child's age are an important variable for exposure. The reasons for these conflicting results could stem from differences in the communities where these children resided. In our previous study (13), children were recruited from an agricultural community where agricultural pesticide was used in close proximity to their homes and residential contamination was fairly common, as evidenced by OP pesticide levels measured in house dust. If younger children spent more time on



**Figure 2.** Residential use of pesticides and the distribution of dimethyl dialkylphosphate concentrations ( $\mu\text{mol/L}$ ) in children living in the Seattle metropolitan area.

\*Significantly higher dimethyl DAP concentrations were found in children whose parents reported use of pesticides in their gardens, Mann-Whitney  $U$ -Wilcoxon rank-sum  $W$  test,  $p = 0.05$ .



**Figure 3.** Residential use of pesticides and the distribution of diethyl dialkylphosphate concentrations ( $\mu\text{mol/L}$ ) in children living in the Seattle metropolitan area.

\*Significantly higher diethyl DAP concentrations were found in children whose parents reported use of pesticides in their gardens, Mann-Whitney  $U$ -Wilcoxon rank-sum  $W$  test,  $p = 0.02$ .

contaminated surfaces in these homes than older children, then the observed difference may have been real. A comparison between 2- to 5-year-old children and older children might help reveal such age differences.

Meinert and colleagues (4) reported an association between residential insecticide uses and childhood lymphoma (odds ratio = 2.6), and the frequency of parental use of household insecticides was a significant factor for this diagnosis ( $p = 0.02$ ). However, the authors acknowledged that the lack of insecticide exposure assessment was a major limitation of the study. A recent study in rural El Salvador (14) evaluated OP pesticide exposure in children 8–17 years old, but yielded only qualitative data. The study found a significant association between adult family member and child OP pesticide metabolite concentrations, but other statistical analyses were confounded by the pooling of adult and child data. Guillette and colleagues (24) found a difference in physiological and neurological deficits in two groups of Yaqui Indian preschool children, presumably due to pesticide exposure. The study was ecological in design, and no measurements were taken of pesticides or any other toxicants that might have affected the relative performance of the two groups. Therefore, the attribution of the observed effects to pesticide exposure remains speculative. Carefully conducted epidemiologic studies that incorporate biomonitoring are needed to ascertain the health risks of pesticide exposure levels children.

The attribution of DAP metabolite measurements to specific pesticides is difficult without detailed knowledge of exposure pathways (15), and such an analysis is beyond the scope of this paper. Symptoms related to OP pesticide exposure in this study were not specifically examined, but none were reported by parents or children, and it is unlikely that the exposures observed in this population would have caused acute intoxications. There is a lack of scientific knowledge regarding the long-term health effects of low-level exposure to OP pesticides in children. This study supports a public health recommendation that, where possible, OP pesticide use should be avoided in areas where children are likely to play. If a residential pesticide application is necessary, it is important to follow the label instructions. Special caution should be taken to avoid contamination of surfaces that are likely to be contacted by children and other occupants.

#### REFERENCES AND NOTES

- Daniels JL, Olshan AF, Savitz DA. Pesticides and childhood cancers. *Environ Health Perspect* 105:1068–1077 (1997).
- Pogoda JM, Preston-Martin S. Household pesticides and risk of pediatric brain tumors. *Environ Health Perspect* 105:1214–1220 (1997).
- Zahn SH, Ward MH. Pesticides and childhood cancer. *Environ Health Perspect* 106(suppl 3):893–908 (1997).
- Meinert R, Schuz J, Kaletsch U, Kaatsch P, Michaelis J. Leukemia and non-Hodgkin's lymphoma in childhood and the exposure to pesticides: results of a register-base case-control study in Germany. *Am J Epidemiol* 151:639–646 (2000).
- Grossman J. What's hiding under the sink: dangers of household pesticides. *Environ Health Perspect* 103:550–554 (1995).
- Faustman EM, Silbernagel SM, Fenske RA, Burbacher TM, Ponce RA. Mechanisms underlying children's susceptibility to environmental toxicants. *Environ Health Perspect* 108(suppl 1):13–21 (2000).
- U.S. EPA. Children's Vulnerability to Toxic Substances in the Environment. EPA600/F/98/013. Washington DC:U.S. Environmental Protection Agency, 1998.
- National Research Council. Pesticides in the Diets of Infants and Children. Washington, DC:National Academy Press, 1993.
- Eskenazi B, Bradman A, Castorina R. Exposures of children to organophosphate pesticides and their potential adverse health effects. *Environ Health Perspect* 107(suppl 3):409–419 (1999).
- Wagner SL, Orwick DL. Chronic organophosphate exposure associated with transient hypertonia in an infant. *Pediatrics* 94:94–97 (1994).
- Ritcher ED, Kowalski M, Leventhal A, Grauer F, Marsouk J, Brenner S, Shkolnik I, Lerman S, Zahavi H, Bashari A, et al. Illness and excretion of organophosphate metabolites four months after household pest extermination. *Arch Environ Health* 47:135–138 (1992).
- Shealy DB, Barr JR, Ashley DL, Patterson DG Jr, Camann DE, Bond AE. Correlation of environmental carbaryl measurements with serum and urinary 1-naphthol measurements in a farmer applicator and his family. *Environ Health Perspect* 105:510–513 (1997).
- Loewenherz C, Fenske RA, Simcox NJ, Bellamy G, Kalman D. Biological monitoring of organophosphorus pesticide exposure among children of agricultural workers. *Environ Health Perspect* 105:1344–1353 (1997).
- Azaroff LS. Biomarkers of exposure to organophosphorus insecticides among farmers' families in rural El Salvador: factors associated with exposure. *Environ Res* 80:138–147 (1999).
- Fenske RA, Kissel JC, Lu C, Kalman DA, Simcox NJ, Allen EH, Keifer MC. Biologically based pesticide dose estimates for children in an agricultural community. *Environ Health Perspect* 108:515–520 (2000).
- Lu C, Fenske RA, Simcox NJ, Kalman D. Pesticide exposure of children in an agricultural community: evidence of household proximity to farmland and take home exposure pathways. *Environ Res* (in press).
- Moate T, Lu C, Fenske RA, Hahne R, Kalman DA. Improved cleanup and determination of dialkyl phosphates in the urine of children exposed to organophosphorus insecticides. *J Anal Toxicol* 23:230–236 (1999).
- Boeniger MF, Lowery LK, Rosenberg J. Interpretation of urine results used to assess chemical exposure with emphasis on creatinine adjustments: a review. *Am Ind Hyg Assoc J* 54:615–627 (1993).
- Hill RH, Head SL Jr, Baker S. Pesticide residues in urine of adults living in the United States: reference range concentrations. *Environ Res* 71:99–108 (1995).
- Simcox NJ, Fenske RA, Wolz SA, Lee I-C, Kalman DA. Pesticides in housedust and soil: exposure pathways for children of agricultural families. *Environ Health Perspect* 103:1126–1134 (1995).
- Chuang JC, Callahan PJ, Katona V, Gordon S. Development and Evaluation of Monitoring Methods for Polycyclic Aromatic Hydrocarbons in House Dust and Track-In Soil. EPA Publication 600/R94/189. Columbus, OH:Battelle, 1993.
- PIRT. Pesticide Incident Reporting and Tracking Panel, 1997 Annual Report. Olympia, WA:Washington State Department of Health, 1998.
- Whitemore RW, Immerman FW, Camann DE, Bond AE, Lewis RG, Schaum JL. Non-occupational exposures to pesticides for residents of two cities. *Arch Environ Contam Toxicol* 26:47–59 (1994).
- Guillette EA, Meza MM, Aquilar MG, Soto AD, Garcia IE. An anthropological approach to the evaluation of preschool children exposed to pesticides in Mexico. *Environ Health Perspect* 106:347–353 (1998).

When it comes to the environment ...



## YOU CAN'T BELIEVE EVERYTHING YOU READ

So you'd better know who you can trust. *Environmental Health Perspectives* draws on more than 25 years of scientific publishing, backed by the authority of the National Institutes of Health—National Institute of Environmental Health Sciences, to provide you with the most current and credible information available to help you make decisions about your health and the environment.

Each month we delve into the issues you care about such as:

- water, air, and soil pollution
- urban sprawl
- population and world health
- toxic pesticides
- environmental products and services



Call 1-800-315-3010 today  
to subscribe and visit us online at

<http://ehp.niehs.nih.gov/>