

# Evaluation of Take-Home Organophosphorus Pesticide Exposure among Agricultural Workers and Their Children

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We analyzed organophosphorus pesticide exposure in 218 farm worker households in agricultural communities in Washington State to investigate the take-home pathway of pesticide exposure and to establish baseline exposure levels for a community intervention project. House dust samples ( $n = 156$ ) were collected from within the homes, and vehicle dust samples ( $n = 190$ ) were collected from the vehicles used by the farm workers to commute to and from work. Urine samples were obtained from a farm worker ( $n = 213$ ) and a young child ( $n = 211$ ) in each household. Dust samples were analyzed for six pesticides, and urine samples were analyzed for five dialkylphosphate (DAP) metabolites. Azinphosmethyl was detected in higher concentrations ( $p < 0.0001$ ) than the other pesticides: geometric mean concentrations of azinphosmethyl were  $0.53 \mu\text{g/g}$  in house dust and  $0.75 \mu\text{g/g}$  in vehicle dust. Dimethyl DAP metabolite concentrations were higher than diethyl DAP metabolite concentrations in both child and adult urine ( $p < 0.0001$ ). Geometric mean dimethyl DAP concentrations were  $0.13 \mu\text{mol/L}$  in adult urine and  $0.09 \mu\text{mol/L}$  in child urine. Creatinine-adjusted geometric mean dimethyl DAP concentrations were  $0.09 \mu\text{mol/g}$  in adult urine and  $0.14 \mu\text{mol/g}$  in child urine. Azinphosmethyl concentrations in house dust and vehicle dust from the same household were significantly associated ( $r^2 = 0.41$ ,  $p < 0.0001$ ). Dimethyl DAP levels in child and adult urine from the same household were also significantly associated ( $r^2 = 0.18$ ,  $p < 0.0001$ ), and this association remained when the values were creatinine adjusted. The results of this work support the hypothesis that the take-home exposure pathway contributes to residential pesticide contamination in agricultural homes where young children are present. **Key words:** biologic monitoring, children, dialkylphosphate metabolites, dust, exposure, organophosphorus pesticides, take-home. *Environ Health Perspect* 110:A787–A792 (2002). [Online 12 November 2002] <http://ehpnet1.niehs.nih.gov/docs/2002/110pA787-A792curl/abstract.html>

Children represent a sensitive subpopulation in terms of exposures to pesticides because they have higher rates of metabolism, less-mature immune systems, unique diets, and distinct patterns of activity and behavior when compared with adults (NRC 1993). Of particular concern are organophosphorus (OP) pesticides, because of their acute toxicity and widespread use both residentially and agriculturally (WHO 1986). Children in families of farmers and farm workers may receive higher pesticide exposures than other children (Loewenherz et al. 1997; Lu et al. 2000; Simcox et al. 1995). Families of farm workers have increased risks of neuroblastoma, nervous system tumors, Hodgkin disease, bone and brain cancer, and childhood leukemia (Buckley et al. 1989; Bunin et al. 1990; Daniels et al. 1997; Kristensen et al. 1996; Lowengart et al. 1987; Wilkins and Koutras 1988), which may stem from increased exposures to agricultural chemicals.

One pathway by which children may be exposed to higher levels of agricultural chemicals is take-home exposure. This pathway involves the transport of contaminants from the workplace to the residence on a worker's clothing or person. Compounds that are likely to cling to and are difficult to remove from clothing, shoes, skin, or hair (e.g., beryllium,

asbestos, lead, pesticides) are potential take-home contaminants, and the take-home pathway has been well documented for several of these compounds (Knishkowsky and Baker 1986; McDiarmid and Weaver 1993; NIOSH 1995). A recent study by Lu et al. (2000) indicated that the take-home pathway is a significant contributor to residential contamination in the homes of agricultural workers. The National Institute for Occupational Safety and Health recommends educating workers and their families about the risks of take-home exposure and about ways that they can minimize their risks (NIOSH 1995).

The research presented here is part of an intervention project designed to reduce take-home pesticide exposure through behavioral change at the community level (Thompson et al., in press). The goals of the current work were to investigate the take-home pathway of pesticide exposure among agricultural families and to establish a baseline of exposure in communities participating in the larger intervention project.

## Materials and Methods

**Study design.** Twenty-four agricultural communities in the Yakima Valley of Washington State were selected to take part

in the intervention project. Thompson et al. (in press) describe the design of the intervention project in detail. Urine and dust samples were collected at the outset of the project (1999) to establish baseline exposure measurements. After sample collection, the communities were randomized into either intervention or control status. In 2002, a similar set of measurements was collected in these communities and will be compared with the 1999 measurements to evaluate the effectiveness of the intervention program.

**Sample population.** Subjects were recruited initially from within the population of a concurrent study conducted by the Fred Hutchinson Cancer Research Center (FHCRC) in the Yakima Valley. Thompson et al. (in press) provide a complete and detailed discussion of the sample population recruitment. Eligible households included an adult farm worker actively involved in field-work or pesticide application (warehouse workers were excluded) and a child between the ages of 2 and 6 years. Adult respondents signed informed consent to participate. Study protocol and data collection procedures were reviewed and approved by the Human Subjects Review Board at the University of Washington (UW) and the Institutional Review Board at the FHCRC.

**Questionnaire.** A questionnaire consisting of 73 items relating to agricultural job tasks, protective practices, and sociodemographic characteristics was administered to all adult participants (Thompson et al., in press). Information from two questions relevant to pesticide exposure pathways has been incorporated into this analysis. First, farm workers were asked to estimate the distance between

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their homes and the nearest field or orchard to which pesticides or farm chemicals were applied. Response categories consisted of < 1 block, 1–2 blocks, 2–4 blocks, 4–8 blocks, 8 blocks–1 mile, > 1 mile, don't know, and refused. Second, farm workers were asked if they had worked on each of the following crops in the past three months: apples, hops, pears, peaches, cherries, and grapes. Response categories for each crop consisted of yes, no, don't know, and refused. Workers were also asked to specify any other crops on which they had worked.

**Dust sampling and analysis.** Dust samples were collected from July through October 1999 with a Nilfisk vacuum cleaner unit (GS-80; Nilfisk of America, Malvern, PA) by trained field staff from the Yakima Valley. House dust was collected in each household in the area where the parent or adult participant said the child played most frequently. Sampling was avoided within 3 feet of the home entryway. The area vacuumed depended on the floor surface type, and a square half-meter by half-meter template was used as a guide. In general, four template areas were vacuumed if the floor was plush carpet (e.g., thick or shag carpet), six if it was thin/flat carpet (e.g., thin carpets, area rugs),

and eight if it was hard/smooth floor (e.g., linoleum, wood).

Vehicle dust collection was added to the study protocol based on the recommendation of the project's community advisory board. A vehicle dust sample was collected from each of the households in which the adult farm worker regularly used a vehicle to get to and from work. Both the front and back footwells were vacuumed, except in the case of trucks without rear footwells, and mats were not removed before vacuuming. All dust samples were stored at  $-10^{\circ}\text{C}$  in the field laboratory until shipped on ice to the UW laboratory, where they were again stored at  $-10^{\circ}\text{C}$  until analysis.

Dust was analyzed for six OP pesticide residues—four dimethyl pesticides (azinphosmethyl, malathion, methyl parathion, and phosmet) and two diethyl pesticides (chlorpyrifos and diazinon)—following the extraction and gas chromatographic procedures described by Moate et al. (2002). These six pesticides represent the major organophosphates applied in the lower Yakima Valley. Samples were transferred from the vacuum cleaner bags to 150- $\mu\text{m}$  metal sieves (VWR, West Chester, PA) and were sieved for 10 min in a sieve shaker (Model RX-24; WS

Tyler Inc, Mentor, OH). At least 0.7 g fine (< 150  $\mu\text{m}$ ) dust was necessary for analysis.

**Urine sampling and analysis.** Urine was collected from one adult farm worker and one child 2–6 years of age in each household concurrent with dust sampling. Child samples were collected using commode specimen collection pans (Sage Products, Inc., Crystal Lake, IL) and were subsequently transferred into 100-mL polypropylene containers with screw-cap lids. Samples from adults and some older children were collected directly into the polypropylene containers. A complete urine sample consisted of a composite of either two or three independent voids, each separated by a minimum of 3 days, and all collected within a 2-week period. Each void was collected by field staff on the day that it was provided and stored in the field lab at  $-10^{\circ}\text{C}$  until all voids were available. Composites consisted of equal volumes of the independent voids. Ideally, each contributed 15 mL; however, if one void was less than 15 mL, that volume was matched by the other void(s). All urine samples were stored at  $-10^{\circ}\text{C}$  in the field lab until shipped on ice to the UW laboratory, where they were stored at  $-10^{\circ}\text{C}$  until analysis.

Urine was analyzed for five of the six dialkylphosphate (DAP) compounds that are produced by metabolism of most OP pesticides, following the extraction and gas chromatographic procedures described by Moate et al. (1999). The five DAPs included dimethylphosphate (DMP), dimethylthiophosphate (DMTP), dimethyldithiophosphate (DMDTP), diethylphosphate (DEP), and diethylthiophosphate (DETTP). Diethyldithiophosphate (DEDTP) was not analyzed because none of the pesticides targeted in this study metabolize into DETTP. Creatinine concentrations were ascertained using a colorimetric procedure based on the Jaffé reaction (Creatinine Procedure No. 555; Sigma Diagnostics, Dorset, UK).

**Quality control and quality assurance.** Urine surrogate solutions (5 mL) consisted of double-deionized water and the 10 most significant components of urine by weight. Dust surrogates (1 g) were subsamples of house dust or vehicle dust previously determined to be free of OP pesticides. Fortified and blank samples were taken into the field intermittently with actual samples. Transport into the field neither contaminated blank samples nor degraded fortified samples.

**Data management.** Urinary metabolite and dust residue data sets included samples containing concentrations less than the limit of quantitation (LOQ), which yielded analyte peaks with signal-to-noise ratios either greater than 3:1 (termed "< LOQ") or less than 3:1 [termed nondetectable (ND)]. Percentile-percentile plots demonstrated that the concentrations of metabolites and pesticides above the

**Table 1.** Percentage of house dust and vehicle dust samples containing six target OP pesticides at levels above the LOQ, at levels < LOQ, and at ND levels.

	Azinphosmethyl	Malathion	m-Parathion	Phosmet	Chlorpyrifos	Diazinon
House dust ( $n = 156$ )						
Above the LOQ (%) <sup>a</sup>	133 (85)	24 (15)	20 (13)	22 (14)	41 (26)	6 (3.8)
< LOQ (%) <sup>b</sup>	6 (3.8)	26 (17)	26 (17)	74 (47)	54 (35)	22 (14)
ND (%)	17 (11)	106 (68)	110 (71)	60 (38)	61 (39)	128 (82)
Vehicle dust ( $n = 190$ )						
Above the LOQ (%)	165 (87)	30 (16)	23 (12)	41 (22)	34 (18)	4 (2.1)
< LOQ (%) <sup>b</sup>	14 (7.4)	71 (37)	42 (22)	81 (43)	17 (9.0)	12 (6.3)
ND (%)	11 (5.8)	89 (47)	125 (66)	68 (36)	139 (73)	174 (92)

ND, nondetectable.

<sup>a</sup>Percentage of total samples. <sup>b</sup>< LOQ; LOQ ( $\mu\text{g/g}$ ) are as follows: house dust: azinphosmethyl = 0.09, malathion = 0.16, m-parathion = 0.12, phosmet = 0.13, chlorpyrifos = 0.15, diazinon = 0.17; vehicle dust: azinphosmethyl = 0.11, malathion = 0.08, m-parathion = 0.12, phosmet = 0.09, chlorpyrifos = 0.11, diazinon = 0.11.

**Table 2.** GM, GSD, maximum values, and selected percentiles of six OP pesticide residue levels<sup>a</sup> in house dust and vehicle dust ( $\mu\text{g/g}$ ).

	GM	GSD	Percentiles				Maximum
			25th	50th	75th	95th	
House dust ( $n = 156$ )							
Azinphosmethyl	0.53	4.3	0.22	0.53	1.42	5.31	14.9
Malathion	0.05	3.0	0.02	0.04	0.14	0.29	1.03
m-Parathion	0.03	4.8	0.01	0.04	0.10	0.28	1.71
Phosmet	0.02	11	0.00	0.02	0.09	1.30	16.9
Chlorpyrifos	0.05	4.6	0.02	0.05	0.14	0.68	2.56
Diazinon	0.01	5.1	0.00	0.01	0.03	0.12	0.77
Vehicle dust ( $n = 190$ )							
Azinphosmethyl	0.75	5.3	0.21	0.85	2.81	9.56	38.3
Malathion	0.02	5.3	0.01	0.02	0.05	0.31	2.12
m-Parathion	0.01	8.1	0.00	0.01	0.03	0.38	3.13
Phosmet	0.01	20	0.00	0.02	0.07	1.19	34.9
Chlorpyrifos	0.03	4.8	0.01	0.03	0.09	0.42	1.97
Diazinon	0.00	7.6	0.00	0.00	0.01	0.05	0.55

GM, geometric mean.

<sup>a</sup>Includes generated values for NDs and < LOQ s.

LOQ were normally distributed following a log transformation, a trend reported previously for biologic and environmental samples from residential locations (Gordon et al. 1999; U.S. EPA 1998). We therefore elected to treat the censored values as log-normally distributed. This approach has been used in previous work with left-censored data (Lyles et al. 2001; Lynn 2001). Quartile-quartile plots were created, and random values ranging from 0 to the LOQ and falling along the log-normal distribution of the points above the LOQ were assigned to the ND and < LOQ samples. Geometric means and SDs were calculated for the overall data sets, including the assigned values, and new log-normal distributions with the same characteristics and size were generated for each compound, as described by Cohen (1991). The NDs and < LOQs in the original data set were replaced with the data from the generated distribution, with NDs considered smaller than < LOQs, and these values were used in subsequent analyses. All concentrations above the LOQ were retained from the original data set. No adult urine samples contained concentrations of DEP above the LOQ, and therefore the distributional characteristics of DEP in child urine were used.

Total molar quantities ( $\mu\text{mol/L}$ ) were calculated by combining individual metabolites according to their chemical structures as follows:

$$[\text{DAP}_{\text{dimethyl}}] = [\text{DMP}]/125 + [\text{DMTP}]/141 + [\text{DMDTP}]/157 \quad [1]$$

$$[\text{DAP}_{\text{diethyl}}] = [\text{DEP}]/153 + [\text{DETP}]/169, \quad [2]$$

where the metabolite concentrations are in units of micrograms per liter and the molecular weights by which they are divided are in units of grams per mole. Results were also adjusted by urinary creatinine concentration by dividing the individual metabolite concentrations (micrograms per liter) by urinary creatinine concentration (milligrams per deciliter) to yield creatinine-adjusted metabolite concentrations (micrograms metabolite per grams creatinine). Adjusted metabolite concentrations (micrograms per gram) were then summed to their molar equivalents (micromoles metabolite per gram creatinine) as described by Equations 1 and 2. All analyses were conducted using both the adjusted and nonadjusted values.

The analytic method for determining DEP concentration consistently overestimated by 37%, and therefore DEP values were adjusted for recovery. All other metabolites had mean recovery efficiencies within one SD of the mean. Dust concentrations were not corrected for recovery because variations were inconsistent. The data were log-transformed,

and linear regression and analysis of variance (ANOVA) calculations were performed in STATA (STATA 6, College Station, TX).

## Results

**Study participants.** A total of 218 households were enrolled in this study. Ninety-seven percent of the participants were Hispanic and 3% were Caucasian. Seven children and five adults who were enrolled in the study declined to provide urine samples at the time of collection. Therefore, urine samples were provided by 211 children and 213 adults. House dust samples were collected in 210 homes, but 54 of these samples did not contain sufficient mass for analysis. Commuter vehicles were available and were sampled at 205 households, but 15 of these samples did not contain sufficient mass for analysis. Thus, pesticide residue analysis was conducted for 156 house dust samples and 190 vehicle dust samples.

**Environmental and biologic samples.** Azinphosmethyl was the most commonly detected pesticide in both house dust and vehicle dust (Table 1): 85% of house dust samples and 87% of vehicle dust samples contained concentrations of azinphosmethyl above the limit of quantitation. The geometric mean concentration of azinphosmethyl was 0.53  $\mu\text{g/g}$  [geometric standard deviation (GSD) =

4.3] in house dust and 0.75  $\mu\text{g/g}$  (GSD = 5.3) in vehicle dust. Residue concentrations for all six target pesticides in house dust and vehicle dust are presented in Table 2. Concentrations of azinphosmethyl were more than an order of magnitude higher than concentrations of any of the other pesticides in both house dust and vehicle dust ( $p < 0.0001$ ).

DMTP was the most commonly detected metabolite (Table 3): 88% of child urine samples and 92% of adult samples contained concentrations of DMTP above the limit of quantitation. Table 4 presents the urinary DAP results, both as urine concentrations and creatinine-adjusted concentrations. The geometric mean concentration of dimethyl DAP metabolites was 0.09  $\mu\text{mol/L}$  (GSD = 2.9) in child urine and 0.13  $\mu\text{mol/L}$  (GSD = 6.9) in adult urine. When the levels were adjusted for creatinine concentration, the geometric mean concentration of dimethyl DAP metabolites was 0.14  $\mu\text{mol/g}$  (GSD = 3.2) in child urine and 0.09  $\mu\text{mol/g}$  (GSD = 7.2) in adult urine. Dimethyl DAP levels were higher than diethyl DAP levels in both child and adult urine ( $p < 0.0001$ ), when the data both were and were not adjusted for urinary creatinine concentration. Adult urine samples contained significantly higher concentrations of dimethyl DAP metabolites than did child samples when the results were

**Table 3.** Percentage of child and adult urine samples containing five DAP metabolites at levels greater than the LOQ, at levels < LOQ, and at ND levels.

	DMP	DMTP	DMDTP	DEP	DETP
Child urine ( $n = 211$ )					
Above the LOQ (%) <sup>a</sup>	41 (19)	186 (88)	95 (45)	2 (0.9)	78 (37)
< LOQ (%) <sup>b</sup>	16 (7.6)	10 (4.7)	5 (2.4)	82 (39)	57 (27)
ND (%)	154 (73)	15 (7.1)	111 (53)	127 (60)	76 (36)
Adult urine ( $n = 213$ )					
Above the LOQ (%)	43 (20)	197 (92)	114 (54)	0 (0.0)	103 (48)
< LOQ (%) <sup>b</sup>	17 (8.0)	2 (0.9)	3 (1.4)	69 (32)	58 (27)
ND (%)	153 (72)	14 (6.6)	96 (45)	144 (68)	52 (24)

ND, nondetectable.

<sup>a</sup>Percentage of total samples. <sup>b</sup>< LOQ; LOQ ( $\mu\text{g/L}$ ) are as follows: DMP = 7.4, DMTP = 1.1, DMDTP = 0.6, DEP = 2.9, DETP = 1.3.

**Table 4.** GM, GSD, maximum values, and selected percentiles of dimethyl and diethyl DAP metabolite levels<sup>a</sup> in child and adult urine samples.

	GM	GSD	Percentiles				Maximum
			25th	50th	75th	95th	
Concentration ( $\mu\text{mol/L}$ )							
Child urine ( $n = 211$ )							
Dimethyl <sup>b</sup>	0.09	2.9	0.04	0.08	0.18	0.52	15.4
Diethyl <sup>c</sup>	0.06	1.5	0.04	0.06	0.08	0.11	0.23
Adult urine ( $n = 213$ )							
Dimethyl	0.13	6.9	0.03	0.09	0.44	3.02	98.0
Diethyl	0.06	1.5	0.05	0.06	0.08	0.12	0.30
Creatinine-adjusted ( $\mu\text{mol/g}$ )							
Child urine ( $n = 211$ )							
Dimethyl	0.14	3.2	0.06	0.14	0.31	1.02	8.42
Diethyl	0.09	2.0	0.06	0.09	0.14	0.29	0.72
Adult urine ( $n = 213$ )							
Dimethyl	0.09	7.2	0.02	0.06	0.25	2.12	75.8
Diethyl	0.04	2.0	0.02	0.03	0.06	0.12	1.03

<sup>a</sup>Includes generated values for NDs and < LOQs. <sup>b</sup>Dimethyl metabolites are the molar sums of DMP, DMTP, and DMDTP.

<sup>c</sup>Diethyl metabolites are the molar sums of DEP and DETP.



not creatinine adjusted ( $p = 0.01$ ). However, when the values were adjusted by urinary creatinine concentration, child urine samples contained significantly higher concentrations of dimethyl DAP metabolites than did adult samples ( $p = 0.001$ ).

**Take-home exposure pathway.** Linear regression analysis indicated a significant association between azinphosmethyl concentrations in vehicle and house dust ( $p < 0.0001$ ,  $r^2 = 0.41$ ), as shown in Figure 1. Dimethyl DAP levels in the urine of children and adults living in the same household were also significantly associated ( $p < 0.0001$ ,  $r^2 = 0.18$ ), as shown in Figure 2. When the metabolite concentrations were creatinine adjusted, the significant associations between child and adult levels remained ( $p < 0.0001$ ,  $r^2 = 0.15$ ).

Questionnaire responses ( $n = 216$ ) regarding household distance to nearest treated field were evaluated to determine whether proximity was associated with exposure, using an ANOVA procedure. First, household proximity to treated fields was defined by the six questionnaire response categories:  $< 1$  block, 1–2 blocks, 2–4 blocks, 4–8 blocks, 8 blocks–1 mile, and  $> 1$  mile. Neither azinphosmethyl concentration in house dust nor child dimethyl DAP level was significantly associated with household proximity to treated fields (house dust:  $p = 0.58$ ; nonadjusted child urine:  $p = 0.34$ ; creatinine-adjusted child urine:  $p = 0.30$ ). Previous studies have used categories of  $\leq 200$  ft (60 m) and  $> 200$  ft (60 m) to investigate the relationship between proximity to treated fields and azinphosmethyl concentrations in house dust and dimethyl DAP concentrations in urine (Loewenherz et al. 1997; Lu et al. 2000). To approximate this analysis, proximity to treated fields was categorized as  $< 1$  block ( $n = 79$ ) and  $> 1$  block ( $n = 137$ ). Again, concentrations of azinphosmethyl in house dust and of dimethyl DAPs in child urine did not differ by distance category (house dust:  $p = 0.58$ ; nonadjusted child urine:  $p = 0.30$ ; creatinine-adjusted child urine:  $p = 0.40$ ).

Linear regression analyses were conducted to evaluate the relationship between pesticide residue levels in household dust and the measured biologic levels in children. Concentrations of azinphosmethyl in household dust were found to be significantly associated with dimethyl DAP concentrations in child urine (nonadjusted:  $r^2 = 0.14$ ,  $p < 0.0001$ ; creatinine adjusted:  $r^2 = 0.15$ ,  $p < 0.0001$ ).

Most workers reported working on more than one crop during the 3 months before their interview. Workers most frequently reported working with apples (72%), pears (60%), and cherries (37%).

**Community status.** Two methods were employed to determine whether the intervention and control communities were

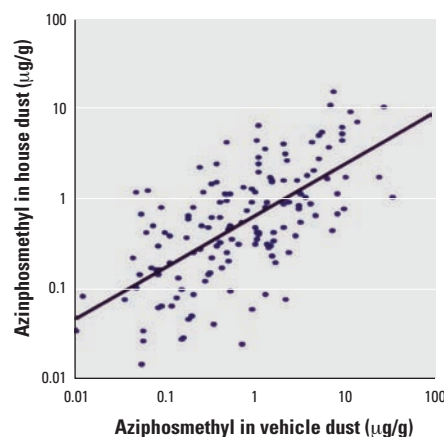
significantly different in terms of OP pesticide exposure or contamination. Five families were excluded from this analysis because the communities in which they resided were not randomized into control or intervention status. First, an ANOVA by randomized group was conducted in which the unit of analysis was the family ( $n = 213$ ). Table 5 presents the geometric mean DAP metabolite and OP residue concentrations by community status. No significant differences were found for urinary DAP levels. Table 5 presents the nonadjusted results; however, the results were unchanged when urinary metabolite levels were adjusted for creatinine concentration. Because six pesticides were targeted in this analysis, the Bonferroni adjustment for multiple comparisons was used, and the  $p$ -value necessary to indicate significance was determined to be 0.008. No significant differences were found for any of the pesticide concentrations in house dust or vehicle dust by community status. A second analysis employed an ANOVA using community as the unit of analysis ( $n = 24$ ), and the geometric means and SEs of the individual samples within the communities were compared. Again, no significant differences were found in exposure levels by community status (data not shown).

## Discussion

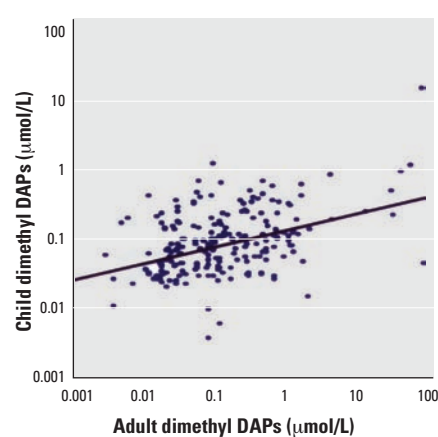
**Take-home exposure.** The results of this study are consistent with the theory of a para-occupational or take-home exposure pathway; i.e., agricultural chemicals move from the workplace to residential environments through the activities of farm workers. Workers in this study were most commonly employed on apple, pear, and cherry crops. More pounds of azinphosmethyl are applied annually on these crops in Washington State than any of the other target pesticides (USDA 2000).

Azinphosmethyl is a Toxicity I insecticide registered exclusively for agricultural use, and according to the U.S. Department of Agriculture, an estimated 360,000 lbs were applied to apple, pear, and cherry crops in Washington State in 1999 (USDA 2000). Annual use of chlorpyrifos approached this amount (300,000 lbs), but unlike azinphosmethyl, chlorpyrifos is generally sprayed before worker contact with treated fields. Therefore, the relatively high concentrations of azinphosmethyl found in house dust and vehicle dust samples in this study correspond well with pesticide use and worker activity patterns in Washington State in 1999.

Concentrations of azinphosmethyl in house dust and vehicle dust from the same household were strongly associated. This suggests that the vehicle used for travel to and from work is a vector of chemical transmission, and that the residues found in the vehicle are markers of contamination on worker clothing or skin. Although it is possible that the observed association was due to a common source other than the workplace, there is little evidence to support such an argument. Analysis of azinphosmethyl levels in house dust and residential proximity to farmland did not reveal a significant pattern, so pesticide drift from agricultural spraying seems an unlikely explanation for the association. It is also possible that workers may have brought agricultural chemicals such as azinphosmethyl home for residential use, and that both home and vehicle were thereby contaminated. This scenario is plausible for a few individuals, but is unlikely to be widespread among workers in this region. A previous study in an adjacent agricultural region found that only 6% of pesticide applicators reported using azinphosmethyl at their residences (Loewenherz et al. 1997). This practice by a small fraction of the



**Figure 1.** Plot of azinphosmethyl concentrations ( $n = 145$ ) in vehicle dust ( $\mu\text{g/g}$ ) versus azinphosmethyl concentrations in house dust ( $\mu\text{g/g}$ ) by household ( $r^2 = 0.41$ ;  $p < 0.0001$ ). This plot includes samples with residue levels below the LOQ.



**Figure 2.** Plot of urinary dimethyl DAP concentrations ( $n = 206$ ) in adult urine ( $\mu\text{mol/L}$ ) versus child urine ( $\mu\text{mol/L}$ ) by household ( $r^2 = 0.18$ ;  $p < 0.0001$ ). Creatinine-adjusted results are similar ( $r^2 = 0.15$ ;  $p < 0.0001$ ). This plot includes samples with metabolite levels below the LOQ.

current study population would not be sufficient to explain the observed association.

The lack of association between proximity and exposure is not consistent with previous work in Washington State (Loewenherz et al. 1997; Lu et al. 2000; Simcox et al. 1995). There are at least two possible explanations for this inconsistency. First, this study occurred in the Yakima Valley region of Washington State, which differs geographically from the Wenatchee area, where previous work took place. Wenatchee is located in a region marked by canyons, which may be more conducive to wind patterns responsible for spray drift. Alternatively, the current study was not focused on the spray drift pathway and simply may not have been effective in examining the association between proximity and exposure. The interview question regarding proximity to treated fields may have been insufficient because the type of pesticide applied on nearby fields was not specified, and distances were self-reported in terms of blocks. Therefore, though there is no evidence that spray drift confounded the reported associations, it is not possible to rule out this pathway as a potential source of exposure for this population.

The association found here between dimethyl DAP levels in child and adult urine

**Table 5.** GM concentrations of urinary DAP metabolites ( $\mu\text{mol/L}$ ) and OP pesticides ( $\mu\text{g/g}$ ) by control or intervention status.<sup>a</sup> No significant differences between control or intervention households were found.

	Intervention	Control	p-Value <sup>b</sup>
Child urine			
No.	101	105	
Dimethyl <sup>c</sup>	0.08	0.10	0.13
Diethyl <sup>d</sup>	0.06	0.06	0.12
Adult urine			
No.	102	106	
Dimethyl	0.16	0.11	0.15
Diethyl	0.06	0.06	0.78
House dust			
No.	74	78	
Azinphosmethyl	0.55	0.51	0.77
Malathion	0.05	0.06	0.16
m-Parathion	0.02	0.04	0.02 <sup>e</sup>
Phosmet	0.02	0.02	0.09
Chlorpyrifos	0.07	0.05	0.09
Diazinon	0.01	0.01	0.88
Vehicle dust			
No.	92	93	
Azinphosmethyl	0.70	0.78	0.68
Malathion	0.02	0.01	0.41
m-Parathion	0.01	0.01	0.84
Phosmet	0.01	0.01	0.45
Chlorpyrifos	0.03	0.03	0.90
Diazinon	0.001	0.003	0.07

<sup>a</sup>Five households were not included in this analysis because they were located in communities not randomized into control or intervention status. <sup>b</sup>p-Values calculated using ANOVA techniques to determine significant differences between groups. <sup>c</sup>Dimethyl metabolites represent the molar sum of DMP, DMTP, and DMDTP. <sup>d</sup>Diethyl metabolites represent the molar sum of DEP and DETP. <sup>e</sup>Not significant when corrected for multiple comparisons.

can also be viewed as supportive of a take-home exposure pathway, but this evidence is less persuasive. The dimethyl DAP metabolites may have been the result of exposure to a variety of OP pesticides and not only those used exclusively in agriculture. In addition, this association could be due to coexposures from another source such as diet. Without a reference or control population for comparison, it is difficult to draw a firm conclusion from these data. In sum, the existence of a take-home pathway in this population is best supported by the association of azinphosmethyl residue levels in vehicle dust and house dust.

These results concur with previous work in Washington State, which found that concentrations of azinphosmethyl and phosmet in the house dust of agricultural workers were elevated over concentrations of these pesticides in the house dust of nonagricultural workers, regardless of residential proximity to farmland (Lu et al. 2000). The study by Lu et al. also reported that residues of agricultural pesticides were detected on the work boots, steering wheels, and children's hands of many of the agricultural families, but not of the reference families.

**Exposure measurements.** This study provides baseline exposure measurements for the agricultural communities participating in the larger intervention project. Azinphosmethyl residues were detected in 85% of the house dust samples, and thus azinphosmethyl concentration in dust will likely serve as the most reliable indicator of take-home exposure. The high prevalence of azinphosmethyl in homes is consistent with findings from previous exposure studies in nearby agricultural regions of Washington State. A 1992 study of OP pesticide concentrations in the house dust of agricultural and reference families found detectable levels of azinphosmethyl in the house dust of every family sampled (Simcox et al. 1995). A study in the same region in 1995 reported similar results (Lu et al. 2000).

Concentrations of azinphosmethyl in household dust were predictive of dimethyl DAP concentrations in child urine, both with and without adjustment for urinary creatinine. However, the  $r^2$  values of 0.14 and 0.15 (for nonadjusted and creatinine-adjusted urinary DAP levels, respectively) suggest that azinphosmethyl concentration in household dust did not explain more than 15% of the variability in the children's dimethyl DAP metabolite levels. Therefore, though household dust appears to be a good indicator of children's azinphosmethyl exposure, this measurement alone is likely not sufficient to fully characterize that exposure.

This study used a Nilfisk GS-80 vacuum cleaner for dust sampling instead of the HVS3 vacuum (Cascade Stamp Sampling Systems, Bend, OR), which has been used in

previous work (Lu et al. 2000; Simcox et al. 1995). Because over 400 dust samples were collected, the portability and maneuverability of the Nilfisk unit made it a more convenient choice. A study by the Agency for Toxic Substances and Disease Registry compared geometric mean lead concentrations collected by the two vacuum cleaners and found that the vacuums differed significantly in collection efficiency in living rooms and bedrooms, though not in entryways (Sterling et al. 1999). The HVS3 had consistently higher collection efficiency than the Nilfisk, and therefore the results of the current study may underpredict dust residue levels compared with studies using the HVS3.

Dimethyl DAP metabolite levels were much higher than diethyl DAP metabolite levels in both child and adult urine, which concurs with findings in previous studies (Aprea et al. 2000; Koch et al. 2002; Lu et al. 2001). However, the median DMTP level in the urine of the children in this study (5.8  $\mu\text{g/L}$ ) was lower than that of children of applicators (21  $\mu\text{g/L}$ ), as reported in a 1995 study occurring in a similar geographic region (Loewenherz et al. 1997). This difference may be due to a combination of factors. First, pesticide exposure levels in agricultural communities have a strong seasonal association, concurrent with agricultural spray periods (Koch et al. 2002). Most azinphosmethyl applications occur between May and July. Sampling in the 1995 study occurred in June, whereas most of the sampling in the current study occurred between July and October. Thus, this study probably missed some of the highest exposures. Second, Lu et al. (2000) report that dimethyl OP metabolite concentrations tend to be higher in the urine of children of pesticide applicators than children of fieldworkers. Unlike the participants in the 1995 study, many of the participants in the current study were not pesticide applicators.

**Study limitations.** This study had four notable limitations. First, urine was collected as several spot samples instead of 24-hr total voids. This necessitated the assumption of steady-state conditions for urinary output and may have introduced random variability into the metabolite measurements. Further, these spot samples were pooled to yield a single sample representing the exposure of each study participant. Ideally, spot samples would have been analyzed individually to prevent the dilution of samples containing high contaminant concentrations and to allow investigation of intraindividual variability. The decision to pool the spot samples was made out of necessity and was based on limited analytic time and resources.

The second limitation of this study was noted previously. Because sampling occurred between July and October, peak exposures occurring during the active spray period were

probably not captured. Third, for many of the compounds investigated, high proportions of the samples contained residues at levels below the limit of quantitation. Our treatment of censored data aimed to produce more realistic variation in these concentrations, but the true values of these samples are still unknown. However, we also analyzed this data set with values below the LOQ set to zero, and again with these values set to one-half the LOQ, and found all statistical results to be unchanged. Significant associations between paired data also remained when the censored data were excluded, demonstrating that the reported associations were not dependent on the generated values. Nonetheless, improved analytic methods would provide more information about the exposure patterns of pesticides commonly present at levels below the LOQ.

Finally, this research is limited by ambiguity regarding the appropriateness of creatinine adjustment for children's urinary exposure measurements (Boeinger et al. 1993). Factors known to affect urinary creatinine levels include weight, age, muscularity, and diet, and differences in these factors are expected to produce differences in creatinine concentrations between children and adults (Wilder 2001). The differential influence of creatinine adjustment on child and adult exposure measurements is illustrated by the comparison of dimethyl DAP metabolite levels within these two populations. With no adjustment, adult dimethyl DAP levels are significantly greater than corresponding child levels. However, when the data were creatinine adjusted, this finding was reversed, demonstrating that the adjustment elevated exposure estimates for children relative to adults. Without definitive evidence regarding the validity of creatinine adjustment in exposure estimates for children, analyses where adjusted and nonadjusted results do not concur cannot be fully interpreted.

## Conclusion

This work provides OP pesticide exposure measurements for a large, chiefly Hispanic population of agricultural workers and their

families. Patterns of OP pesticide exposure in this population were supportive of the hypothesis that the take-home exposure pathway contributes significantly to residential pesticide contamination in farm worker homes that include young children. The study also provides baseline exposure measurements for a community intervention project in the Yakima Valley, and has demonstrated that intervention and control communities within the study had similar exposure levels at the outset of the intervention activities.

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