Predicting Children's Short-Term Exposure to Pesticides: Results of a Questionnaire Screening Approach

Ken Sexton,[1] John L. Adgate,[1] Lynn E. Eberly,[2] C. Andrew Clayton,[3] Roy W. Whitmore,[3] Edo D. Pellizzari,[3] Paul J. Lioy,[4] and James J. Quackenboss[5]

[1]Division of Environmental and Occupational Health, and [2]Division of Biostatistics, School of Public Health, University of Minnesota, Minneapolis, Minnesota, USA; [3]Research Triangle Institute, Research Triangle Park, North Carolina, USA; [4]Environmental and Occupational Health Science Institute, Robert Wood Johnson Medical School, University of Medicine and Dentistry of New Jersey, Piscataway, New Jersey, USA; [5]U.S. Environmental Protection Agency, National Exposure Research Laboratory, Las Vegas, Nevada, USA

The ability of questionnaires to predict children's exposure to pesticides was examined as part of the Minnesota Children's Pesticide Exposure Study (MNCPES). The MNCPES focused on a probability sample of 102 children between the ages of 3 and 13 years living in either urban (Minneapolis and St. Paul, MN) or nonurban (Rice and Goodhue Counties in Minnesota) households. Samples were collected in a variety of relevant media (air, food, beverages, tap water, house dust, soil, urine), and chemical analyses emphasized three organophosphate insecticides (chlorpyrifos, diazinon, malathion) and a herbicide (atrazine). Results indicate that the residential pesticide-use questions and overall screening approach used in the MNCPES were ineffective for identifying and oversampling children/households with higher levels of individual target pesticides. ***Key words:*** **atrazine, children, chlorpyrifos, diazinon, dietary ingestion, exposure, inhalation, malathion, nondietary ingestion, pesticides, questionnaires, urinary metabolites.** *Environ Health Perspect* **110:123–128 (2003). [Online 11 December 2002]**
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Children can be exposed to pesticides through a variety of pathways, including dietary and nondietary ingestion, inhalation of indoor and outdoor air, dermal contact with contaminated surfaces, and use of medications and personal care products (NRC, 1993). Because children's activities often occur in or near their residences, realistic risk assessments must necessarily involve characterization of children's exposure in residential settings (Adgate and Sexton 2001; Cohen Hubal et al. 2000; Gurunathan et al. 1998; Landrigan et al. 1999; Zartarian et al. 2000). Today, diverse efforts are under way to measure children's residential and nonresidential exposure to pesticides and a variety of other hazardous chemicals (Needham and Sexton 2000). The Minnesota Children's Pesticide Exposure Study (MNCPES) was one of the first attempts to conduct a relatively comprehensive home-based exposure assessment by measuring multipathway exposures to multiple pesticides in a cross-sectional probability sample of urban and nonurban children.

The MNCPES used a stratified random sampling strategy to select families with age-eligible children (3–13 years old) living in either the cities of Minneapolis and St. Paul (designated urban households), or Rice and Goodhue Counties (designated nonurban households), located approximately 50 miles south of the Twin Cities metropolitan area. The primary objective was to characterize children's exposure to selected pesticides through a combination of personal exposure measurements (air, duplicate diet, hand rinse) and complementary monitoring of biologic samples (pesticide metabolites in urine), environmental samples (residential indoor/outdoor air, drinking water, dust on residential surfaces, soil), and children's activity patterns. Chemical analyses focused primarily on three organophosphate insecticides (chlorpyrifos, diazinon, malathion) and a herbicide (atrazine), which were selected because of their frequent use, presence in multiple environmental media, expected population exposures, and associated toxicity.

We used data from the MNCPES to examine the issue of whether questionnaire responses about general household pesticide use can predict children's exposure to four target compounds over the short term (weeks, months). We briefly describe the study design and then examine statistical associations between questionnaire responses and measured exposure parameters. We conclude by discussing plausible reasons for our findings and commenting on implications for future studies of children's pesticide exposure.

MNCPES Design Strategy

Previous publications provide relevant details about the MNCPES, including *a*) an in-depth description of the study design and chemical analysis methods (Quackenboss et al. 2000); *b*) a summary of results from the residential pesticide-use survey (Adgate et al. 2000b); *c*) an examination of outcomes from the MNCPES design strategy (Adgate et al. (2000a); *d*) a quantitative analysis of children's activity patterns (Freeman et al. 2001); *e*) a summary of chemical analyses of house dust and urine samples (Lioy et al. 2000); and *f*) a statistical analysis of measured urine metabolite levels (Adgate et al. 2001). Below, we provide a brief synopsis of the MNCPES strategic framework and the household pesticide-screening procedure.

Strategic framework. The strategic framework for the MNCPES comprised three overlapping phases: identification of eligible households, initial screening of eligible households to preferentially select those with high pesticide use, and intensive monitoring of households and children. These phases were implemented on a rolling basis over a 5-month period (May–September 1997). The goal was to enroll at least 100 families/children for the intensive-monitoring portion of the MNCPES.

Identification phase. A total of 2,303 telephone numbers were selected from a commercially available list of residences predicted to have age-eligible children based on birth records and other publicly available data. Because of concerns that the list might underrepresent families from lower socioeconomic (SES) strata, telephone numbers from lower SES census tracts were sampled proportional to their rate of occurrence in the study area. Of the initial 2,303 telephone numbers, 2,057 were determined to be residential.

Screening phase. Telephone screening was completed for 1,388 of these households. A combination of selection criteria (residence located in target areas, age-eligible

Address correspondence to K. Sexton, Environmental and Occupational Health, Room 1260, MMC 807, 420 Delaware Street SE, Minneapolis, MN 55455 USA. Telephone: (612) 626-4244. Fax: (612) 626-0650. E-mail: ksexton@umn.edu

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child present, reported use of pesticides, and use of a well as a water source in nonurban households) and probability sampling was used to identify 477 families eligible to participate in the screening-phase survey of in-home pesticide storage and use. This residential pesticide survey was completed within the scheduled period for 294 families, and 181 families were selected and available to complete the baseline questionnaire. A subset of 173 families completed the baseline questionnaire.

Monitoring phase. A total of 109 families/children were selected to begin the intensive-monitoring phase, of which 102 families/children were subsequently enrolled and completed the final phase of the study. The age distribution of the 102 children in the intensive-monitoring phase of the MCPES was as follows: 3 years, 10 children; 4 years, 10 children; 5 years, 10 children; 6 years, 15 children; 7 years, 10 children; 8 years, 6 children; 9 years, 10 children; 10 years, 8 children; 11 years, 12 children; 12 years, 9 children; and 13 years, 2 children.

Screening procedure to select higher pesticide exposure households. In the MNCPES survey design, larger proportions of households deemed to have more frequent pesticide use (based on a telephone interview during the identification phase) and with more than one age-eligible child were selected for the household-screening phase, and families with private wells in nonurban areas were preferentially selected. During the subsequent screening phase, interviewers administered the household pesticide screening questionnaire to an adult residing in each of 294 households with age-eligible children. The questionnaire consisted of 46 questions related to *a*) consent and eligibility (1 question), *b*) occupant characteristics (12 questions), *c*) household characteristics (4 questions), *d*) household pesticide use (23 questions), and *e*) occupant activities (6 questions). At the same time, study personnel also obtained informed consent and conducted the pesticide inventory. Based on the integrated results, each household was assigned (subjectively) a numeric score, computed as the sum of the factors listed below.

A score of 12 points indicated that at least one primary pesticide was found either inside or out, and there was reported use in the past year. A score of 6 points indicated that pesticides were reportedly used to control pests inside the home in the past 6 months. A score of 5 points indicated that pesticides were reportedly used to control pests outside the home in the past 6 months. A score of 4 points indicated that a household member reported regular occupational exposure to pesticides. A score of 3 points indicated that at least one primary pesticide was found inside or out, but it was not reported used in the past year. A score of 2 points indicated that only nonprimary pesticides were found in the inventory, but there was use reported in the past year. A score of 1 point indicated that only nonprimary pesticides were found in the inventory and there was no reported use in the

Table 1. Summary statistics for target pesticides measured in selected media.

Sampling medium and measurement parameters	Atrazine	Diazinon	Malathion	Chlorpyrifos
Personal air				
No. of valid analyses[a]	42	48	61	60
Percent detectable[b]	16.7	64.1	54.1	95.0
Geometric mean (ng/m^3)[c]	0.1	0.3	0.3	1.9
Geometric SD[c]	12.7	5.1	5.6	4.4
Average detection limit[d]	0.09	0.1	0.1	0.09
Indoor air				
No. of valid analyses	60	75	88	82
Percent detectable	21.6	68.0	67.0	91.5
Geometric mean (ng/m^3)	0.1	0.3	0.5	1.6
Geometric SD	8.8	5.2	6.2	5.6
Average detection limit	0.08	0.1	0.1	0.1
Outdoor air				
No. of valid analyses	46	52	51	52
Percent detectable	15.2	13.5	11.8	9.6
Geometric mean (ng/m^3)	0.06	0.06	0.06	0.6
Geometric SD	2.7	2.4	2.6	2.2
Average detection limit	0.08	0.1	0.09	0.1
Daily intake of solid foods[e]				
No. of valid samples	100	101	96	96
Percent detectable	NA[f]	NA	NA	NA
Geometric mean (µg/day)	0.06	0.05	2.0	0.3
Geometric SD	2.9	2.8	2.7	2.6
Average detection limit	NA	NA	NA	NA
Daily intake of beverages[g]				
No. of valid analyses	101	101	101	101
Percent detectable	NA	NA	NA	NA
Geometric mean (ng/day)	NE[h]	NE	NE	NE
Geometric SD	NE	NE	NE	NE
Average detection limit	NA	NA	NA	NA
Surface dust loading				
No. of valid analyses	99	99	99	99
Percent detectable	3.0	7.1	0.0	61.6
Geometric mean (ng/cm^2)	2.0	1.8	NE	0.7
Geometric SD	1.3	1.3	NE	1.6
Average detection limit	4.1	3.5	3.0	1.2
Soil concentration				
No. of valid analyses	102	102	102	102
Percent detectable	0.0	3.9	0.0	2.9
Geometric mean (µg/kg)	NE	5.2	NE	5.0
Geometric SD	NE	1.3	NE	1.2
Average detection limit	81.6	10.1	10.2	10.2
Urine concentration (metabolite)[i]				
No. of valid analyses	90	—[j]	90	90
Percent detectable	4.4		46.6	96.6
Geometric mean (µg/L)	0.4		0.7	6.3
Geometric SD	1.5		1.9	2.0
Average detection limit	0.7		0.8	1.4

Abbreviations: NA, not applicable; NE, not estimated.
[a]Number of samples sent to the laboratory that complies with calibration and quality control standards applicable to each analyte and sampling medium. [b]Percentage of valid samples for which measured pesticide concentration was above the analytic detection limit reported by the laboratory. [c]The geometric mean and SD were calculated using the measured value of the sample if it was above the reported analytic detection limit. For samples that were below the analytic detection limit, a value of one-half the detection limit was used. If there were duplicate samples and both were above the analytic detection limit, they were averaged to obtain a single value. If one duplicate sample was above the analytic detection limit and the other below, then only the detectable value was used. [d]The average detection limit is the average analytic detection limit for multiple batches of samples processed at different times by the laboratory. Units are the same as the geometric mean. [e]Daily intake of solid food has no analytic detection limit because it is the product of pesticide concentration in food and the mass of food collected per day. Percentage of measured pesticide concentrations in food samples above the analytic detection limit was 8% for atrazine, 3% for diazinon, 46% for malathion, and 57% for chlorpyrifos. [f]Not applicable because intake is calculated by multiplying the pesticide concentration by mass collected in the duplicate diet. [g]Daily intake of beverages has no analytic detection limit because it is the product of pesticide concentration in beverages and the volume of beverages consumed per day. All measured pesticide concentrations in beverage samples were below the analytic detection limit. [h]NE, not feasible to estimate because all samples were below the analytic detection limit. [i]Most children provided three urine samples (88 provided three samples, two provided two samples and one child provided a single sample) over a 1-week period. Measured urine metabolites from all samples for each child were averaged for each analyte to calculate a geometric mean and SD. One-half the analytic detection limit was used for those samples below the analytic detection limit. Concentrations were not adjusted with creatinine. [j]Chemical analysis not performed for this pesticide and medium.

past year. Maximum points possible = 27 (because some factors are mutually exclusive).

Those households deemed by the investigators to have greater potential for exposure to target pesticides were selected at a higher rate for the intensive-monitoring phase of the MNCPES. This selection was based on a subjective (numeric) scoring process that integrated information from subject-specific data (household/occupant information obtained from both the identification and screening phases) and household-specific data (presence of products with one or more of the target pesticides according to results of the in-home pesticide inventory).

For approximately 8 weeks, each of two MNCPES field teams was assigned specific target cohorts (subset of the 294 households) on which to focus their recruiting efforts for a particular week. On average, each team successfully recruited about 10 families/children per week, of which 173 eventually completed the intensive-monitoring baseline questionnaire. Within each weekly target cohort, five households with the highest pesticide screening scores (based on the scoring procedure described above) were selected with certainty, and the other five were selected at random. Of the 102 households that ultimately participated in the intensive-monitoring phase of the MNCPES, 54 (53%) were designated subjectively as having higher pesticide screening scores.

It is worth noting that the screening scores were initially incorrect because some products were classified incorrectly as containing diazinon. This error was corrected later and scores were recalculated, which resulted in changed scores for about 30 households. All statistical analyses were run on both sets of scores and produced similar results. The following discussion presents data for the corrected scores only.

Statistical methods. Statistical tests were carried out on log-transformed data to stabilize variances of measurement error, and pesticide concentrations below the analytic detection limit were imputed as half the detection limit reported by the relevant laboratory (Adgate et al. 2001). Pesticide concentrations in many of the sampling media were still highly skewed even after log transformation due to large numbers of nondetects. Nonparametric Wilcoxon tests were done to explore differences between higher pesticide use and other homes. Statistical analyses were carried out in two ways to determine whether the household pesticide-screening score (described above) predicted measured pesticide values (Table 1). First, logistic regression was used to test whether higher scores predicted presence of a detectable amount of one of the target pesticides (categoric variable, yes/no); second, ordinary least-squares regression was used to test whether higher scores predicted higher measured log concentration of the target pesticides (continuous variable).

Further statistical analyses were carried out on adult responses to six pesticide-use questions (Table 2) that were asked as part of the household screening questionnaire. We examined whether the six pesticide-use questions, individually and in combination, predicted either presence of a detectable amount of one of the target pesticides (categoric variable, yes/no) or the measured log concentration of one of the target pesticides (continuous variable). Significance for the categoric response was assessed using an approximate chi-square test. Because of small cell counts, associations between detectable amount (yes/no above analytic limit of detection) and each of the six pesticide-use survey questions were tested again using Fisher's exact test (Agresti 1990). Regressions were also run with all six survey questions considered at once to determine how combinations of questions were associated with log concentration. Model reduction was carried out via backward stepwise regression (Neter et al. 1990).

All analyses were done using SAS, Version 8 (SAS Institute, Inc., Cary, NC), and reported *p*-values were not adjusted for multiple comparisons. Analyses were not weighted according to the study sampling weights so that we could assess, within our preferentially selected sample, whether the pesticide-use scoring procedure or the questionnaire effectively predicted which households/children had higher pesticide concentrations.

Table 2. Summary of responses to selected pesticide-use questions plausibly related to children's exposure.

Question from the MNCPES pesticide-use questionnaire	No. responding yes	No. responding no
17. Is this property used as a farm? (*n* = 102)	11 (10.8%)	91 (89.2%)
18. In the past 6 months, were any chemicals for the control of fleas, roaches, ants, or other insects used inside this residence? (*n* = 102)	65 (63.7%)	37 (36.3%)
If yes to question 18, then an interviewer asked questions 19 and 20		
19. What room(s) in your home were treated? (*n* = 65)		
a. Living room	16	49
b. Family room	12	53
c. Dining room	12	53
d. Kitchen	55	10
e. Bathroom(s)	18	47
f. Bedroom(s)	16	49
g. Basement	20	45
h. Other rooms	11	54
20. Which areas within the rooms were treated? (*n* = 65)		
a. Floors	41	24
b. Baseboards	24	41
c. Lower half of walls	5	60
d. Upper half of walls	4	61
e. Ceilings	6	59
f. Cupboards with dishes	6	59
g. Cupboards with food	9	56
h. Cabinets for storage	12	53
i. Closets	8	57
j. Windowsills	15	50
k. Other	28	37
26. In the past 6 months, were any chemicals for the control of fleas, roaches, ants, or other insects used on the exterior or foundation of this residence? (*n* = 102)[a]	27 (26.5%)	74 (2.6%)
32. In the past 6 months, have there been any regular treatments by anyone on the lawn or yard outside of this residence? (*n* = 102)	41 (40.2%)	61 (59.8%)
38. Do you have a flower, vegetable, or fruit garden here or elsewhere to which you apply chemicals for the control of weeds or insects? (*n* = 102)[a]	28 (27.5%)	73 (71.6%)
39. Do you have pets such as dogs, cats, gerbils, hamsters, rabbits, guinea pigs, birds, or horses? (*n* = 102)	73 (71.6%)	29 (28.4%)
If Yes to question 39, then an interviewer asked Question 40:		
40. Are any chemicals or collars used on any of these pets to control fleas or ticks? (*n* = 73)	20	53

[a]One person responded "I don't know."

Results

Measured pesticide concentrations (atrazine, chlorpyrifos, diazinon, malathion) in eight types of sampling media from the 102 households/children enrolled in the intensive-monitoring phase of MNCPES are summarized in Table 1. A detailed description of sample collection techniques and chemical analysis methods has been published previously (Quackenboss et al. 2000), as has a summary of samples collected (both targeted and valid) and associated numbers of valid analytic results (Adgate et al. 2000a). Personal (child's breathing zone), indoor (inside the residence), and outdoor (outside the residence) air concentrations are based on 144-hr integrated samples (approximate). All were collected using the same type of sorbent cartridge and

battery-operated constant-flow pump. Samples of solid food and beverages (not including tap water) consumed at home during the 4-day sampling period were collected using a duplicate diet approach. Daily intake was calculated using food mass of the duplicate diet samples. Surface dust was collected inside the residence from an accessible area in the child's play area and in another room where he or she routinely spent time (Lioy et al. 2000). A soil sample was collected from exposed soil in the child's primary outside activity areas. Most children (88/102) provided three first morning void urine samples on days 3, 5, and 7 of the sampling week. (Concentrations were averaged to obtain a single value.)

As shown in Table 1, more than 90 valid samples (88% of 102) were obtained for individual pesticides in five of eight sampling media (daily intake of solid foods and beverages, surface dust loading, soil concentration, and urine metabolite concentration). Because of problems related to sample collection and chemical analysis (Adgate et al. 2000a), the number of valid air samples (personal, indoor, outdoor) ranged from a low of 42 (41% of 102) for atrazine in personal air to a high of 88 (86% of 102) for malathion in indoor air. For most media (except personal and indoor air), the percentage of valid analyses with detectable pesticide levels was 15% or less. (The only exceptions were chlorpyrifos in surface dust and both chlorpyrifos and malathion in urine.) It was not feasible to determine the percent detectable for daily intake of food and beverages, because they are calculated as the products of concentration measurements and mass collected in the duplicate diet.

Fifty-four of 102 households enrolled in the intensive-monitoring phase were designated higher pesticide usage based on the screening phase pesticide-use scoring procedure described above. These 54 households had a mean screening score of 18.2 (median 18, SD 4.7), compared with a mean of 6.2 (median 7, SD 4.0) for the other 48 lower pesticide usage households. Statistical analysis (nonparametric Wilcoxon two-sample test) revealed a significant difference ($p < 0.0001$) between the two sets of calculated scores (an expected result based on the study design). The nonparametric Wilcoxon two-sample test was also used to test for differences in measured log pesticide concentrations (for applicable sampling media in Table 1) between the 54 higher pesticide usage and 48 lower pesticide usage households. The only statistically significant difference was for a metabolite of malathion in the child's urine [$p = 0.04$; Wilcoxon test statistic = 2.48; 95% confidence interval (CI), 1.06–5.8].

Logistic regression and ordinary least-squares regression were used to test whether higher assigned household pesticide-screening scores (regardless of whether the household had been designated as higher or lower pesticide usage) were associated with higher measured pesticide concentrations for the sampling media shown in Table 1. When the dependent variable was defined as the presence of a detectable amount of pesticide in a particular sampling medium (categoric variable, yes/no), the only statistically significant finding was for atrazine levels in personal air [$p = 0.028$; odds ratio (OR) = 1.22; 95% CI, 1.02–1.45]. Defining the log pesticide concentration (continuous variable) as the dependent variable produced only two statistically significant results: atrazine in personal air ($p = 0.020$) and malathion (metabolite) in urine ($p = 0.033$). However, in both cases the parameter estimate was in the direction of higher pesticide-screening scores associated with lower pesticide concentrations (probably due to chance). Similar results for logistic regression and ordinary least-squares regression were obtained when we examined the relationships between pesticide-screening scores and the total concentration of all four target compounds combined (i.e., no meaningful statistical associations).

The evidence indicates that the use of subjective pesticide-screening scores to preferentially select higher pesticide use households failed to identify households with higher concentrations (in seven different environmental media) as well as children with higher short-term pesticide exposures (urine metabolite concentrations). However, the screening system was based on a subjective assignment of points to household attributes plausibly related to pesticide exposure. It is possible that individual pesticide-use questions or combinations of questions may provide better predictive capability. To investigate this possibility, we selected the key pesticide-use questions likely to be most directly related to measured pesticide concentrations from the pesticide-screening questionnaire. The six key pesticide-use questions (along with three subquestions) are listed in Table 2 (the question number is from the screening questionnaire). Table 2 also provides data on the number of respondents answering yes or no to each question.

Logistic regression and ordinary least-squares regression were used to test whether responses to any of these individual questions predicted either presence of a detectable amount or measured log concentration of one of the target pesticides in any of the eight sampling media listed in Table 1. No statistically significant results were observed for any of these tests. For example, selected logistic regression results are presented in Table 3 for malathion and chlorpyrifos. (Their metabolites were found most frequently in the children's urine.) No outcomes were significant at the $p = 0.05$ level when we tested whether the answer to Question 18 on the household pesticide-screening questionnaire (was there indoor pesticide application within the past 6 months—yes or no?) predicts detectable pesticide levels (yes/no). Similar results were obtained for the other five questions and when Fisher's exact test was used instead of logistic regression.

To test whether a combination of questions might be more predictive of log concentrations in various media, regressions were run with all six pesticide-use questions in the model. Backward stepwise regression was used to select the most parsimonious models, and the six that explained the highest proportion of variance (r^2) are listed in Table 4. The best predictive model produced an r^2 of 12% [a combination of Questions 17 (property a farm) and 26 (exterior use of pesticides in past 6 months)] to predict chlorpyrifos in indoor air; and a combination of questions 18 (inside use of pesticides in past 6 months), 26 (exterior use of pesticides in past 6 months), 38 (apply chemicals to garden), and 39 (have pets) to predict a metabolite of malathion in urine. Again, however, the parameter estimates in most cases were in the direction of higher potential for exposure associated with

Table 3. Logistic regression results for pesticide concentration (detectable/nondetectable) on reported indoor pesticide application in the past 6 months (yes/no).

	Malathion			Chlorpyrifos		
Medium	*p*-Value	OR[a]	95% CI[b]	*p*-Value	OR[a]	95% CI[b]
Personal air	0.073	0.377	0.13–1.09	NE[c]	NE	NE
Indoor air	0.369	0.641	0.24–1.69	0.296	0.310	0.04–2.75
Outdoor air	0.373	2.76	0.30–25.7	0.715	0.700	0.11–4.66
Daily intake of solid foods	0.060	0.442	0.19–1.04	0.380	1.46	0.63–3.37
Daily intake of beverages	NE	NE	NE	NE	NE	NE
Surface dust loading	NE	NE	NE	0.436	0.71	0.30–1.67
Soil concentration	NE	NE	NE	NE	NE	NE
Metabolite in urine	0.174	0.550	0.23–1.30	NE	NE	NE

[a]Odds ratio for measured pesticide level above analytic detection limit in households reporting indoor pesticide applications in the past six months compared with measured pesticide level above analytic limit in households reporting no indoor pesticide applications in the past six months (Question #18 on the Household Pesticide-Screening Questionnaire) [b]95% CI for the OR. [c]NE, not feasible to estimate because all samples were below the analytic detection limit or because of small cell counts (e.g., only 3 nondetects for chlorpyrifos in personal air).

lower pesticide concentrations (probably due to chance). Thus, single questions and combinations of questions failed to predict higher individual pesticide concentrations in any of the eight sampling media studied. They also failed to predict the total concentration of the four target compounds combined.

Discussion

Adequate protection of children's environmental health has been the stated objective of numerous policy initiatives and research programs over the past 10 years (Carlson 1998; Clinton 1997; Galson et al. 1998; Landrigan et al. 2000; Needham and Sexton 2000; NRC 1993; Selevan et al. 2000). Although much of the concern has focused on possible adverse health effects from both dietary and nondietary exposure to organophosphate pesticides, relatively little is actually known about when, where, why, how, and for whom elevated exposures are likely to occur. Consequently, there is an acute need for better monitoring data on which to base realistic assessments of both exposure and health risks.

The MNCPES obtained baseline, population-based measurements of important exposure-related variables to allow for more realistic assessments of children's multipathway pesticide exposures. The objective of the pesticide-use screening procedure was to enrich the stratified random sample by oversampling households where children were deemed more likely to come into contact with the target pesticides. Our goal in the intensive-monitoring phase was to enroll more children living in households where measured pesticide concentrations were expected to be above the analytic limit of detection for applicable laboratory methods (i.e., to enrich the sample).

This enrichment process is potentially important for a baseline study like the MNCPES, because a probability sample is needed to estimate the distribution of pesticide exposures for a defined population of children (in this case, children living in specified census tracts in Minneapolis–St. Paul and Rice and Goodhue Counties). To describe the characteristics of the exposure distribution, measures of central tendency (e.g., mean, median, mode) and variability (e.g., SD) are often used. Several points on the distribution (i.e., point estimates) are typically of special interest for risk assessment purposes: *a*) values near the middle of the distribution; *b*) values at or above the 90th percentile, which the U.S. Environmental Protection Agency (U.S. EPA) defines as the high end of the distribution; and *c*) values at or near the extreme upper end that give an indication of concentrations experienced by the most exposed individuals in the population (Sexton et al. 1995). If too many reported pesticide concentrations are below the analytic limits of detection, especially when the sample is not large (e.g., 102 children in the intensive-monitoring phase), accurate estimation of important distribution descriptors becomes problematic. Because unequal weighting adversely affects the precision of the estimated mean for the overall population, one must consider the trade off of attempting (and perhaps not succeeding) to oversample the high end of the distribution versus getting a more precise estimate of the mean but having more nondetects.

Results indicate that the subjective pesticide-use screening procedure used in MNCPES was ineffective as a method for identifying and oversampling households/children with detectable levels of atrazine, diazinon, malathion, and chlorpyrifos. Statistical analyses reveal no meaningful differences between nominally higher pesticide use households (n = 54) and other households (n = 48) for measured pesticide concentrations in eight sampling media. Similar results were obtained for responses to six pesticide-use questions, both individually and in combination, which also failed to differentiate households/children with higher measured pesticide concentrations. In some cases with large numbers of nondetects, significant results in the opposite direction from that expected were found.

There are several plausible reasons why neither the subjective pesticide-use scoring procedure nor the pesticide-use survey questions predicted measured concentrations. First, both were based on questions about use of any pesticides for insect or weed control, whereas measurements focused on just four target compounds. In fact, results of the earlier pesticide survey (Adgate et al. 2000b) revealed that products containing nontarget insecticides were more likely to be found in MNCPES households and more likely to be used during the past year than the target insecticides. The pesticide-use questions were necessarily general because few, if any, participants were likely to know (or remember) whether they used a product with a specific active ingredient. Nevertheless, this incongruity between generalized questions and specific pesticide measurements undoubtedly contributed to the observed lack of predictive capability.

Moreover, it is probable that residential pesticide use in and around these homes was episodic, occurring only infrequently and intermittently, if at all. The scoring system and the questions focused on pesticide use in the preceding 6–12 months, implicitly assuming that either previous applications would subsequently result in higher concentrations or pesticides would be consistently applied more frequently in certain households, thereby giving rise to higher concentrations. The evidence suggests that under the conditions of the study neither assumption was correct.

The screening scores and pesticide-use questions were an attempt to obtain information on sources and potential exposure-related behaviors over the past several months, whereas measured pesticide concentrations provided only a short-term exposure snapshot covering a few days. Thus it is possible, though in our opinion not likely, that longer-term measurements (e.g., months) might have been better correlated with either the scoring system or the pesticide-use questions. It is interesting to note that pesticide

Table 4. Prediction of log pesticide concentration using household screening questionnaire responses.

Pesticide and medium[a]	r^2 for the model[b]	Question(s) in the model[c]	β[b]	*t*-Statistic	*p*-Value
Chlorpyrifos					
Personal air	0.07	Q18	–0.82	–2.08	0.04
Indoor air	0.12	Q17	–1.41	–2.56	0.01
Daily intake of solid foods	0.04	Q26	–0.80	–1.93	0.06
Surface dust loading	0.08	Q26	–0.66	–1.89	0.06
		Q26	–0.028	–2.87	0.01
Malathion					
Metabolite in urine	0.12	Q18	–0.22	–1.67	0.10
		Q26	–0.33	–2.14	0.03
		Q38	0.31	2.10	0.04
		Q39	–0.26	–1.78	0.08
Chlorpyrifos					
Metabolite in urine	0.08	Q26	–0.29	–1.73	0.09
		Q32	–0.26	–1.69	0.09

[a]The specific pesticide and the medium or media in which concentrations were measured. [b]Regression coefficients and r^2 values are from backward stepwise linear regression; questions that are significant predictors at level 0.10 were retained in the model. Log concentrations were computed from one-half the detection limit for those falling below the detection limit. Only those media with approximately normally distributed log concentrations were considered. [c]The question or questions included in the best fitting model: Q17, Is this property used as a farm? Q18, In the past six months were any chemicals for the control of fleas, roaches, ants, or other insects used inside this house/apartment? Q26, In the past six months were any chemicals for the control of fleas, roaches, ants, or other insects used on the exterior of this house/apartment? Q32, In the past six months have there been any regular treatments by anyone on the lawn or yard outside of this house/apartment? Q38, Do you have a flower, vegetable, or fruit garden to which you apply chemicals? Q39, Do you have pets such as dogs, cats, gerbils, hamsters, rabbits, guinea pigs, birds, or horses?

concentrations were not predicted accurately even in sampling media that might have been expected to retain residue from past pesticide applications (house dust).

It is also possible, though again doubtful from our perspective, that better predictive capability would have been achieved had the questions focused in more detail on the specifics of residential pesticide application and the child's behaviors during and directly following these applications. The problem is that at some point, the ability of parents/guardians to recall accurately the particulars of routine household activities or the activities of their children seriously limits the viability of this approach.

The picture is further complicated because the data used to calculate screening scores, including responses to pesticide-use questions, were obtained several weeks before sample collection. Although we acquired time–activity data during the sampling week, there is always the possibility that participants altered their normal behavior before and during the monitoring period as a consequence of being enrolled in the study.

Conclusions

There is an ongoing need to obtain baseline data on children's exposure to organophosphate pesticides and many other hazardous environmental chemicals. Better estimates of the distribution of exposures, including measures of central tendency and variability, as well as point estimates in the upper tail of the distribution, are necessary for more realistic assessment of children's actual exposure and related health risks. This means that well-designed probability studies will have to be undertaken to obtain the requisite exposure information. Because a high percentage of households/families/children tend to have relatively low exposures (often below analytic limits of detection), there is an ongoing need to find cost-effective ways to screen and preferentially select those households/families/children that experience higher exposures. Nevertheless, results from the MNCPES show that predicting children's short-term exposure to the four target pesticides is not straightforward. Both a relatively complex pesticide-use scoring procedure and responses to pesticide-use questions failed to predict higher concentrations of target pesticides collected over several days in multiple sampling media. These results agree with previous findings that screening scores and questionnaires are not predictive of individual pesticides (Whitmore et al. 1994). Further research is needed to identify practical and easy-to-apply screening approaches that successfully predict long- and short-term pesticide exposures for children.

References

Adgate JL, Barr DB, Clayton CA, Eberly LE, Freeman NCG, Lioy PJ, et al. 2001. Measurement of children's exposure to pesticides: analysis of urinary metabolite levels in a probability-based sample. Environ Health Perspect 109:583–590.

Adgate JL, Clayton CA, Quackenboss JJ, Thomas KW, Whitmore RW, Pellizzari ED, et al. 2000a. Measurement of multi-pollutant and multi-pathway exposures in a probability-based sample of children: practical strategies for effective field studies. J Expo Anal Environ Epidemiol 10(6):650–661.

Adgate JL, Kukowski A, Stroebel C, Shubat PJ, Morrell S, Quackenboss JJ, et al. 2000b. Pesticide storage and use patterns in Minnesota households with children. J Expo Anal Environ Epidemiol 10:159–167.

Adgate JL, Sexton K. 2001. Children's exposure to pesticides in residential settings. In: Handbook of Pesticide Toxicology (Krieger R, ed). 2nd ed. San Diego, CA:Academic Press, 887–904.

Agresti A. 1990. Categorical Data Analysis. New York: John Wiley & Sons, Inc.

Carlson J, ed. 1998. Children's Environmental Health: Research, Practice, Prevention, and Policy. Environ Health Perspect 106(suppl 3):785–862.

Clinton WJ. 1997. Children's Health. Executive Order 13045: Protection of Children From Environmental Health Risks and Safety Risks. Fed Reg 62:19885.

Cohen Hubal EA, Sheldon LS, Burke JM, McCurdy TR, Berry MR, Rigas ML, et al. 2000. Children's exposure assessment: a review of factors influencing children's exposure, and the data available to characterize and assess that exposure. Environ Health Perspect 108:475–486.

Freeman NCG, Jimenez M, Reed KJ, Gurunathan S, Edwards RD, Roy A, et al. 2001. Quantitative analysis of children's microactivity patterns: The Minnesota Children's Pesticide Exposure Study. J Expo Anal Environ Epidemiol 11(6):501–509.

Galson SK, Carroquino MJ, Landrigan PJ, eds. 1998. Preventable Causes of Cancer in Children. Environ Health Perspect 106(suppl 3):865–925.

Gurunathan S, Robson M, Freeman NCG, Buckley B, Roy A, Meyer R, et al. 1998. Accumulation of chlorpyrifos on residential surfaces and toys accessible to children. Environ Health Perspect 106:9–16.

Landrigan PJ, Claudio L, Marowitz SB, Berkowitz GS, Brenner BL, Romero H, et al. 1999. Pesticides and inner-city children: exposures, risks, and prevention. Environ Health Perspect 107(suppl 3):431–437.

Landrigan PJ, Weiss B, Goldman LR, Carpenter DO, Suk WA, eds. 2000. The Developing Brain and Environment. Environ Health Perspect 108(suppl 3):373–448.

Lioy PJ, Edwards RD, Freeman NCG, Gurunathan S, Pellizzari ED, Adgate JL, et al. 2000. House dust levels of selected insecticides and a herbicide measured by the EL and LWW samplers and comparisons to hand rinses and urine metabolites. J Expo Anal Environ Epidemiol 19(4):327–340.

National Research Council. 1993. Pesticides in the Diets of Infants and Children. Washington, DC:National Academy Press.

Needham LL, Sexton K, eds. 2000. Assessment of Children's Environmental Exposure. J Expo Anal Environ Epidemiol 10(6):611–815.

Neter J, Kutner MH, Nachtsheim CJ, Wasserman W. 1990. Applied Linear Statistical Models, Fourth Edition. Chicago, IL: Richard D. Irwin, Inc.

Quackenboss JJ, Pellizzari ED, Shubat P, Whitmore RW, Adgate JL, Thomas KW, et al. 2000. Design strategy for assessing multi-pathway exposure for children: the Minnesota Children's Pesticide Exposure Study (MNCPES). J Expo Anal Environ Epidemiol 10:145–158.

Selevan SG, Kimmel CA, Mendola P, eds. 2000. Identifying Critical Windows of Exposure for Children's Health. Environ Health Perspect 108(suppl 3):451–597.

Sexton K, Callahan MA, Bryan EF, Saint CG, Wood WP. 1995. Informed decisions about protecting and promoting public health: rationale for a National Human Exposure Assessment Survey. J Expo Anal Environ Epidemiol 5(3):233–256.

Whitmore RW, Immerman FW, Camann DE, Bond AE, Lewis RG, Schaum JL. 1994. Non-occupational exposures to pesticides for residents of two U.S. cities. Arch Environ Contam Toxicol 26:47–59.

Zartarian VG, Ozkaynak H, Burke JM, Zufall MJ, Rigas ML, Furtaw EJ. 2000. A modeling framework for estimating children's residential exposure and dose to chlorpyrifos via dermal residue contact and nondietary ingestion. Environ Health Perspect 108:505–514.