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Nonagricultural and residential exposures to pesticides

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Epidemiologic studies and risk assessments conducted to assess the chronic effects of pesticides are limited by inadequate measurements of pesticide exposures, and surrogates for these data are frequently used. In this paper, pesticide use and absorbed dose previously measured in residential and occupational settings are used to evaluate the hypothesis that there is a relationship between pesticide use and exposure. For homeowner applicators of 2,4-dichlorophenoxyacetic acid (2,4-D) and chlorpyrifos, exposures were poorly correlated with the amount of herbicide used (r^2 = 0.01 to 0.40); however, exposures from a granular product were consistently less than those with liquid formulation. For professional landscape applicators, exposure over 14 days and 7 days of use was poorly correlated with the amount of 2,4-D sprayed (r^2 = 0.17 and 0.21, respectively). However, inclusion of the type of spray nozzle used and the use of gloves while spraying in the model explained increased predictability and explained 68% of the variation.

Key terms 2,4-dichlorophenoxyacetic acid; chlorpyrifos; exposure determinant; home applicator; landscape pesticide.

The importance of exposures to pesticides through nonfood routes has been emphasized in recent changes to regulations in the United States after the introduction of the Food Quality Protection Act and the need to assess and aggregate exposures to pesticides from multiple sources, including food, water, and residential uses. Much of the interest in these sources of exposures has been driven by public concerns expressed in response to pesticides in the environment (1) and studies suggesting that the pesticides may adversely affect the health of children (2–10). From the point of view of risk assessment, the extent and frequency of these exposures are important. However, they are also important for the epidemiologic assessment of possible links between pesticides and cancer. For example, a review of several studies on childhood cancer in relation to pesticides has pointed out that, for many studies, exposures are poorly characterized (11). One of the most common surrogates used to characterize pesticide exposure is the amount of pesticide used or the area of crop sprayed; however, the appropriateness of this surrogate for exposure has rarely been tested experimentally, especially for herbicides. One study has shown that pesticide use data collected shortly after application are poor predictors of exposures to 2,4-dichlorophenoxyacetic acid (2,4-D) among children of farmer applicators and may give a high false positive rate of classification (12). This issue is also addressed in another paper in this volume (13).

Several studies of exposures have been conducted among home applicators and bystanders after urban insecticide and herbicide applications on outdoor turf sites. Two of these, involving the application of 2,4-D and chlorpyrifos in landscapes, are discussed in more detail in this paper to illustrate the type of data that can be obtained and to test the hypothesis that the amount of pesticide used is proportional to the exposures or doses received.

The terms "exposure" and "dose" are often used interchangeably, generally with the understanding that important distinctions exist between the two. Exposure generally refers to the concentration of an agent at the boundary between a person and the environment, as well as the duration of contact between the two (14). Dose, or more specifically internal dose, is the amount of an agent that is absorbed, inhaled, or ingested into the body, and it is generally expressed over a given period of time.

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A large number of host factors affects the relationship between potential exposure and the resulting absorbed dose. Although all people who work or reside within a defined area may have the same opportunity for potential exposure, activity patterns and the location of work areas relative to a primary source of exposure affect a person's personal level of exposure. For agents for which inhalation is the primary route of exposure, host factors such as breathing rates and the amount of physical exertion can modify the resulting internal dose. When the primary route of exposure is dermal, the internal dose can be modified by factors such as the area and location of skin exposed, skin damage, the number of hair follicles, and other environmental considerations, such as temperature and humidity, and the presence of other compounds on the skin (15–17). The relationship between potential exposure and internal dose can also be modified by work-hygienic practices, such as the wearing of gloves or the use of respirators, and may be further modified by attitudes, avoidance behavior, and risk perception, to name a few (18).

Sources of exposure

The major sources of nonfood exposures come through the use of pesticides in the home and garden, although bystanders to agricultural pesticide applications may also receive some exposure via indirect routes (figure 1).

Strata of exposures in residential settings are home applicators, bystanders to outdoor and indoor exposures, and "re-entry" exposure through contact with treated surfaces both indoors and outdoors. The greatest potential for indoor exposures is from broadcast or fogger applications for flea control (19); however, these types of applications are infrequent (once or twice a year). Crack and crevice treatments are more frequent (every 6–8 weeks), but the applications are more directed and likely result in smaller exposures. In these situations, respiration has been identified as a major route of exposure (20), but contact with treated surfaces and household dust can be a major source of exposure, especially for children (21–23). Outdoor exposures may originate from landscape pesticide use, through homeowner applicator exposure (24), through drift or contact with treated surfaces (25), or through nearby agricultural applications (26–28).

Measurement of exposures

Methods and techniques for measuring worker and residential exposures have been reviewed recently (29, 30). Inhalation exposure can be estimated with the use of air monitoring of work areas or personal monitoring of breathing zones. Dermal exposures can be evaluated with the use of patches, whole body dosimetry, or fluorescent tracers. Depending on the physical characteristics of the agent of interest and the primary routes of exposure, a combination of these methods can be used to evaluate exposure. When these exposure estimates are converted to internal dose estimates, constant breathing rates, body weights, dermal absorption rates, and a direct linear relationship between exposure and dose are generally assumed. This assumption does not allow for any intraindividual variation in the relationship between exposure and dose (18).

Matrix samplers or dosimeters are usually absorptive substrates that are used to measure potential dermal exposures. These samplers range from gloves to entire suits that can be extracted with solvents after exposure and analyzed for pesticide residues. A very traditional technique for estimating exposure is the use of small patches of absorptive material attached to clothing. The method was pioneered by Durham & Wolfe (31) and has been widely used since that time (29, 32, 33). In practice, several of these pads are placed on key locations on the body and after the exposure activity, a section of the pad is cut out and analyzed for pesticides. Total body exposure is determined by extrapolation to the areas of the particular body parts and summing the totals. A novel variation of the patch technique is the use of sticky backed sanitary pads, which attach easily to clothing and have little background contamination (34). Such techniques assume that deposition is uniform, which it is not.

Special garments or devices can be used to dislodge pesticides from treated surfaces such as outdoor or indoor surfaces. Whole-body dosimeter clothing was first reported to be used by Chester & Ward (35) for assessing agricultural exposures, but it has also been used for indoor exposures when coupled with choreographed exercises (Jazzercises) to reduce the variability between participants and to standardize contact times

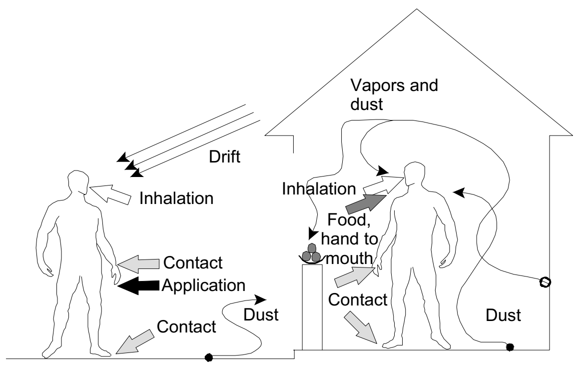

Figure 1. Potential sources of exposure in the home and landscape.

and movements (32, 33). These dosimeters can also be worn under clothing to assess the effectiveness of protective garments. While giving better estimates of total exposures than patches, the cotton or absorptive fabrics are a poor model of human skin and are thus more suitable for characterizing dermal deposition (spray droplets) than dislodgeable residues. A novel use of dosimeters is the recent use of a "child-like mannequin" for assessing the potential for spray-drift pesticide exposure among children (36).

An extension of the dosimeter approach has been the use of fluorescent tracers added to the spray formulation to assess dermal exposure. First used to monitor spray deposition by Staniland (37), this technique was adapted to illustrate nonuniformity by Chester & Ward (35) and further developed and used by Fenske and his co-workers (38 and earlier papers) and was recently reviewed (39). Archibald and his co-workers (40–42) used the technique for assessing the exposure of greenhouse workers to pesticides, as did others in The Netherlands [43; personal communication: Brouwer DH. Exposure of greenhouse workers to pesticides. Presented at "Workshop on Video Imaging Techniques for Assessing Exposure", June 20–21, 1996. Iowa City (IA): 1996.]. The technique was further developed by Roff and his colleagues (44–46) and used to assess exposures of homeowners to wood preservatives applied in the domestic situation (45). Once the compatibility and calibration of the ratios of pesticide and tracer have been completed, the technique is fast and noninvasive, and it gives immediate visual results that are useful in training applicators. It can be used to assess the effectiveness of protective clothing but, because of differences in adsorption to fabric between the tracer and the pesticide, cannot be used to measure penetration through protective clothing (47). Tracers can also be used to assess the dislodgeability of pesticides from treated surfaces; however, the differences in the dissipation of the residues of the pesticide and the tracer over time require extensive calibration for consistent results (48, 49).

The utility of various techniques for measuring the dislodgeability of pesticides from turf has been studied, and the polyurethane foam (PUF) roller was found to be the least suitable. The weighted cloth-covered (DOW) sled (50) was more suitable, but a modification of the California roller (33) was found to be the best (51). In practice, a weighted roller of polyvinyl chloride (PVC) covered with an absorptive fabric is moved across the surface of the turf. The fabric is then removed for analysis.

The extraction of absorptive surfaces, such as toys, has been used to estimate exposures through the oral route when these are mouthed by young children (52). However, the applicability of solvent-extracted residues to those that could be removed with saliva was not assessed, and there was great variation in the study due to low replication. The relevance of this source of exposure is not clear.

Exposures through the respiratory route have been commonly assessed through the use of filters and adsorption devices attached to portable pumps to measure concentrations of particulate and volatile pesticides in the breathing zone of workers or in outdoor and indoor environments. Suffice it to say that these samplers have been widely used in occupational (53) and nonoccupational settings (20). This technique has recently been used to assess exposures to metals from dust generated during the manufacture of wooden items from treated wood (54).

Adult exposure through the oral route from surfaces such as food and utensils contaminated during domestic pesticide applications has been shown to be a relatively minor source (55), but the evaluation of this route of exposure among children is more challenging and is the object of recent and ongoing research (28, 56–62). It has been estimated that, because of smaller body mass and greater consumption, children have an approximately 12-fold greater potential exposure to pesticides in dusts than do adults (63). Regional and national differences in the use of pesticides indoors, differences in housekeeping, and child-rearing practices further complicate the assessment of exposure through this route and extrapolation from one region to another.

Biomonitoring studies, in which pesticides or their metabolites are measured in body fluids, such as saliva, blood, and urine, have been used to characterize exposures and absorbed dose and have been reviewed (64). These studies have used both continuous and spot samples to characterize total exposures over time, as well as during shorter periods. To determine the total absorbed dose of a chemical agent following a single occupational or environmental exposure or multiple exposures, the toxicokinetics and toxicodynamics of the compound must be known. This information is generally obtained through the use of in vitro and in vivo animal studies and, in some cases, may be further supported by human volunteer studies. Concentrations of the agent of interest in biological samples such as urine, feces, blood, or serum can be used to estimate the total absorbed dose, and the accuracy of this dose estimation is largely based on the availability of relevant human studies and the timing and completeness of the sample collection (65). Biomonitoring is held up as the "gold-standard" for exposure measurement as it integrates all sources of exposures and accounts for differences in penetration between persons and chemicals and thus usually gives lower (but more realistic) measures of exposure or dose (64). The technique is best applied to compounds with well-known pharmacokinetics and metabolism in humans but suffers from a lack of sensitivity at low concentrations for fluids, such as urine, that are relatively difficult to analyze. The issue here is that measurement below the level of detection (LOD) or a nondetected level (ND) does not mean zero exposure. While this may be of little significance for highly exposed persons, it can become relevant when aggregate exposures to many pesticides are assessed for bystanders, when additive action of individual chemicals, all below the LOD, may represent a significant risk. The issue of aggregate exposures has been discussed elsewhere (66).

Exposure analyses for turf uses of pesticides

Exposures to liquid and granular 2,4-D, a commonly used turf herbicide, have been measured for homeowner applicators, professional applicators, and bystanders to both types of application (18, 24, 65, 67, 68). In addition, exposure to liquid and granular chlorpyrifos has also been measured among homeowner applicators and bystanders (69).

The homeowner applicator studies on 2,4-D were conducted using biomonitoring based on a collection and analysis of all urine produced during the 96-hour postapplication period after a single application (24). Homeowner applicators were divided into four strata, using a granular (weed and feed 2,4-D) formulation with and without supplied protective clothing (polyester-cotton overalls, rubber boots, and rubber gloves) or a liquid 2,4-D formulation applied with a hose-end sprayer with and without supplied protective equipment. Total 24-hour urine samples were collected from professional landscape applicators over a 14-day period early in the spray season. The application of 2,4-D started on the same day as the study and ceased 11 days into the study (at the start of a long weekend). The collection and analysis of urine continued over the long weekend to allow a more-complete excretion and collection of any absorbed 2,4-D (67). In both the homeowner and professional landscape applicator studies, 96-hour urine samples were collected from bystanders, not involved in the application but living in the same house. The total amount of 2,4-D measured in the 96-hour urine samples collected from the homeowners and bystanders was used as an estimate of the total absorbed dose resulting from exposure on the day of application.

The chlorpyrifos studies were conducted in a manner similar to those described for 2,4-D, except that an exposure study of professional applicators was not conducted. Urine samples collected from homeowner applicators and bystanders were hydrolyzed to convert all chlorpyrifos to trichloropyridinol (TCPY) which was then analyzed using gas chromatography with mass spectroscopic confirmation (69). The total amount of TCPY excreted over the 96-hour period was used to calculate the total absorbed dose of chlorpyrifos for each volunteer.

All the studies measured the amount of pesticide used by the applicator. The data thus allow a test of the null hypothesis that the common surrogate for exposure, amount of pesticide used, is not related to exposure, or more specifically, the total absorbed dose. For this purpose, a simple linear regression of the amount used versus the total exposure was used. Since no bystanders to the 2,4-D applications had any measurable exposures, no analyses of these data were conducted. Likewise, as only one of the bystanders to the chlorpyrifos applications had measurable exposure, these data also were not analyzed. Regressions of the amount of 2,4-D applied and the total absorbed dose of homeowner applicators showed poor correlations (figure 2); this finding suggested that the amount of chemical used, or a co-variate, such as area treated, is a poor surrogate for exposure. However, it is clear that exposures from the liquid formulation were much greater than those from the granular formulation, despite the greater amounts of the latter that were applied. Similar relationships were observed for chlorpyrifos (figure 3), the granular formulation again resulting in smaller exposures.

Although these data do not support the use of the amount applied as a surrogate for exposure, the data can be used to estimate the likelihood of exposure through the use of a distributional analysis. Probabilistic approaches have been used for assessing environmental risks from pesticides (70). For this purpose, exposure data were ranked from the lowest to the highest, and plotting positions were calculated from the equation $j = i/n+1 \times 100$, where j is the plotting position, i is the rank, and n is the total number of data points in the data set. Data from sets with two or more nonzero values were then plotted as cumulative frequency distributions on

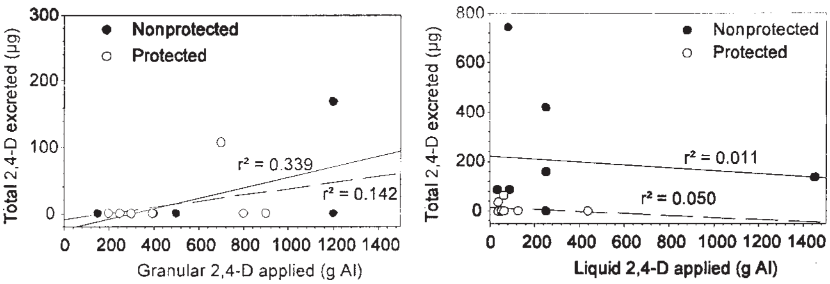

Figure 2. Illustration of the poor correlation between the amount of 2,4-dichlorophenoxyacetic acid (2,4-D) applied and the exposure of homeowner applicators.

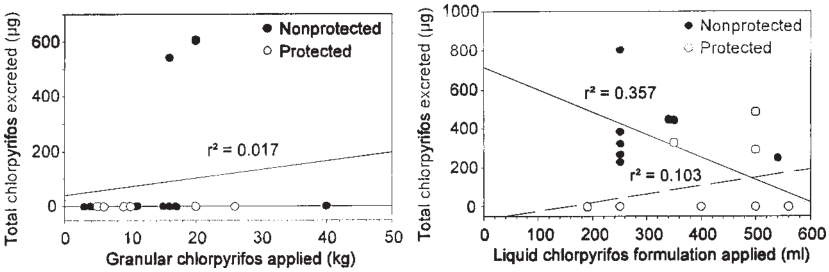

Figure 3. Illustration of the poor correlation between the amount of chlorpyrifos applied and the exposure of homeowner applicators.

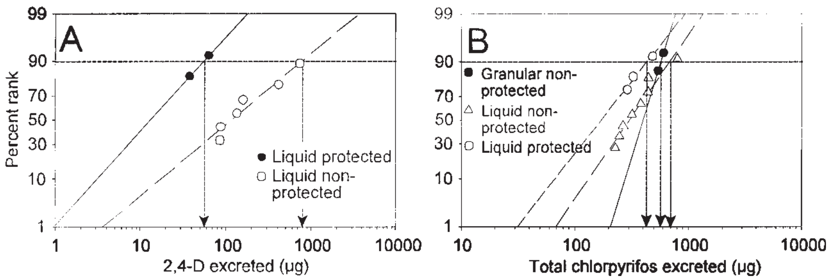

Figure 4. Cumulative frequency distributions of exposure to chlorpyrifos (A) and 2,4-dichlorophenoxyacetic acid (2,4-D) (B) for homeowner applicators.

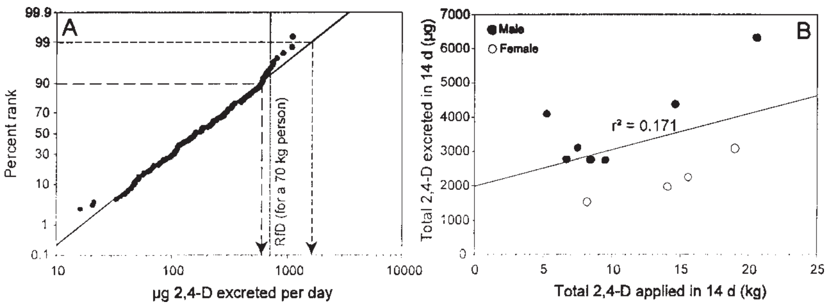

Figure 5. Cumulative distribution of the daily amounts of 2,4-dichlorophenoxyacetic acid (2,4-D) excreted by landscape applicators (A) and the relationship between the total amount of 2,4-D applied during a 14-day period and the total amount of 2,4-D excreted over the same time (B).

a log-percent probability scale and a regression conducted on the transformed data (figure 4), on the assumption of a log-normal model. For the purposes of ranking, nondetected levels were assigned a dummy value of zero, but were not plotted. The values for the nondetected levels were assumed to have the same distribution as the observed values. These cumulative distributions can be used to estimate exposures that may occur under similar conditions of use or to assess risks from the measured values. The 90th centile of the exposure values is shown on the graphs to illustrate this use; however, those based on few data points obviously have greater uncertainty.

The value for protective clothing in reducing exposure was the most obvious in the case of 2,4-D (figure 4B).

The exposures of the landscape applicators were analyzed in a similar manner (figure 5). Although the amounts of 2,4-D excreted by a landscape applicator in a day are dependent on exposures occurring over several days, the daily amounts of 2,4-D excreted were treated as independent data for the purposes of a distributional analysis. This procedure resulted in a relatively large data set (figure 5A), from which the centiles of exposure could be estimated. The median exposure (50th centile) and 90th centile were smaller than the reference dose for a 70-kg person (figure 5A). The 99th centile from the fitted distribution exceeded the reference dose and was also greater than the greatest concentration measured.

An analysis of the amount of 2,4-D applied over the 14-day period and the total excreted by the individual applicators showed a poor correlation (figure 5B); however, the four least-exposed persons (in relation to the amount of 2,4-D used) were all females. Again, the amount of pesticide applied was a poor surrogate for exposure, and individual differences between applicators appeared to modify the relationship between the amount of pesticide applied and the total absorbed dose.

Similarly, the results of a study designed specifically to address the issue of the relationship between the amount of pesticide sprayed and the total absorbed dose among professional landscape applicators showed a poor relationship between the amount of 2,4-D applied and the total body dose (over a 1-week period), as estimated from the urinary excretion of 2,4-D (65, 68). In this case, the use and excretion of 2,4-D was monitored for 94 landscape applicators. A questionnaire was used to collect information on spraying practices, hygiene practices, the perception of risks, smoking, and self-reported exposures. A simple one-variable model (pesticide use) of ln-ln-transformed data, gave a model r^2 of

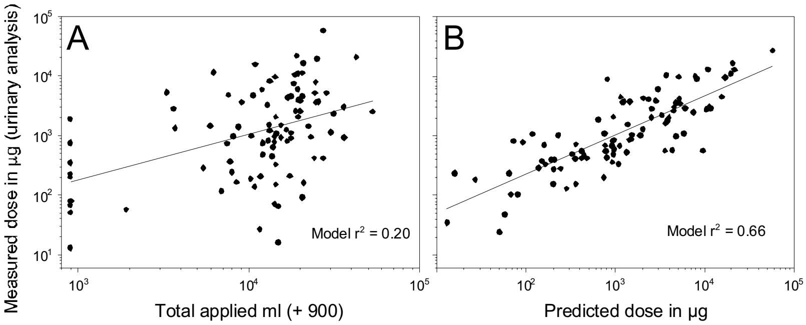

Figure 6. Relationship between the amount of herbicide sprayed and body dose estimated from urinary output for two consecutive 24-hour periods for 94 professional landscape applicators (A) and the relationship between the measured and modeled dose of 2,4-dichlorophenozyacetic acid (2,4-D) for the same data set (B). [Redrawn from Harris et al (69)].

0.20. When a predictive model was developed that incorporated variables to indicate the type of nozzle used, the wearing of gloves, job satisfaction, and smoking in addition to the amount sprayed, the model r^2 was 0.66 (figure 6). The authors concluded that the volume of pesticide used was not strongly related to body dose, as measured by biological monitoring, and that semi-quantitative classifications of job titles and tasks performed were inadequate proxies of exposure, as were self-reported exposures. They also pointed out that current epidemiologic studies lack sufficient power when quantity of pesticide applied is used is a surrogate for exposure.

Concluding remarks

The use of individual pesticide application records as proxy or surrogate measures for pesticide dose has been shown to be clearly inadequate in both occupational and residential settings. If future epidemiologic studies are to be conducted with sufficient power to detect subtle health risks in these settings, the estimation of dose must be improved, and relevant proxies must be established.

References

1. Colborn T, Dumanoski D, Myers JP. Our stolen future. New York (NY): Dutton; 1996.
2. Bell EM, Hertz-Picciotto I, Beaumont JJ. A case-control study of pesticides and fetal death due to congenital anomalies. Epidemiology 2001;12:148–56.
3. Bell EM, Hertz-Picciotto I, Beaumont JJ. Case-cohort analysis of agricultural pesticide applications near maternal residence and selected causes of fetal death. Am J Epidemiol 2001;154:702–10.
4. Bosma H, van Boxtel MP, Ponds RW, Houx PJ, Jolles J. Pesticide exposure and risk of mild cognitive dysfunction. Lancet 2000;356:912–3.
5. Daniels JL, Olshan AF, Teschke K, Hertz-Picciotto I, Savitz DA, Blatt J, et al. Residential pesticide exposure and neuroblastoma. Epidemiology 2001;12:20–7.
6. Daniels JL, Olshan AF, Savitz DA. Pesticides and childhood cancers. Environ Health Perspect 1997;105:1068–77.
7. Ernst P. Pesticide exposure and asthma. Am J Respir Crit Care Med 2002;165:563–4.
8. Meinert R, Schuz J, Kaletsch U, Kaatsch P, Michaelis J. Leukemia and non-Hodgkin's lymphoma in childhood and exposure to pesticides: results of a register-based case-control study in Germany. Am J Epidemiol 2000;151:639–46 and 647–50.
9. Nurminen T. Maternal pesticide exposure and pregnancy outcome. J Occup Environ Med 1995;37:935–40.
10. Leiss JK, Savitz DA. Home pesticide use and childhood cancer: a case-control study. Am J Public Health 1995;85:249–52.
11. Hoar Zahm S, Ward MH. Pesticides and childhood cancer. Environ Health Perspect 1998;106 S3:893–908.
12. Arbuckle TE, Cole DC, Ritter L, Ripley BD. Children's exposure to pesticides: comparison of exposure estimates derived from biomonitoring and questionnaire data. Epidemiology 2004;15:187–94.
13. Arbuckle TE, Cole DC, Ritter L, Ripley BD. Biomonitoring of herbicides in Ontario farm applicators. Scand J Work Environ Health 2005;31 suppl 1:90–7.
14. Hatch M, Thomas D. Measurement issues in environmental epidemiology. Environ Health Perspect 1993;101 suppl 4:49–57.
15. Maibach H, Feldman R, Milby T, Serat W. Regional variation in percutaneous penetration in man. Arch Environ Health 1971;23:208–11.
16. Moody R, Wester R, Melendres J, Maibach H. Dermal absorption of the phenoxy herbicide 2,4-D dimethylamine in humans: effect of DEET and anatomic site. J Toxicol Environ Health 1992;36:241–50.
17. Harris SA, Solomon KR. Percutaneous penetration of 2,4-dichlorophenoxyacetic acid and 2,4-D dimethylamine salt in human volunteers. J Toxicol Environ Health 1992;36:233–40.
18. Harris SA. The development and validation of a pesticide dose prediction model [dissertation]. Toronto (Ontario): University of Toronto; 1999.
19. Edminston S. Human Illness/injuries reported by physicians in California involving indoor exposure to pesticides containing chlorpyrifos, DDVP, and/or propoxure (1983–1986). Sacramento (CA): Worker Health and Safety Branch, California Department of Food and Agriculture; 1990. Report no HS 1431.
20. Whitmore RW, Immerman FW, Camman DE, Bond AE, Lewiss RG, Schaum JL. Non-occupational exposures to pesticides for residents of two US cities. Arch Environ Contam Toxicol 1994;26:47–59.
21. Fenske RA, Black KG, Elkner KP, Lee CL, Methner MM, Soto R. Potential exposure and health risks of infants follow-

ing indoor residential pesticide applications. Am J Public Health 1990;80:689–93.
22. Lewis RG, Fortmann RC, Camann DE. Evaluation of methods for monitoring the potential exposure of small children to pesticides in the residential environment. Arch Environ Contam Toxicol 1994;26:37–46.
23. Lewis RG, Fortune CR, Willis RD, Camann DE, Antley JT. Distribution of pesticides and polycyclic aromatic hydrocarbons in house dust as a function of particle size. Environ Health Perspect 1999;107:721–6.
24. Harris SA, Solomon KR. Human exposure to 2,4-D following controlled activities on recently sprayed turf. J Environ Sci Health B 1992;27:9–22.
25. Harris SA, Solomon KR, Stephenson GR. Exposure of homeowners and bystanders to 2,4-dichlorophenoxyacetic acid (2,4-D). J Environ Sci Health B 1992;27:23–38.
26. Ames RG, Howd RA, Doherty L. Community exposure to a paraquat drift. Arch Environ Health 1993;48:47–52.
27. Gladen BC, Sandler DP, Zahm SH, Kamel F, Rowland AS, Alavanja MC. Exposure opportunities of families of farmer pesticide applicators. Am J Ind Med 1998;34:581–7.
28. 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 2000;84:290–302.
29. Chester G. Worker exposure: methods and techniques. In: Krieger RI, editor. Handbook of pesticide toxicology. 2nd edition. New York (NY): Academic Press; 2001. p 425–34.
30. Driver JH, Ross HH, Pandian MD, Evabs JB, Whitmyre GK. Residential exposure assessment: an overview. In: Krieger RI, editor. Handbook of pesticide toxicology. 2nd ed. New York (NY): Academic Press; 2001. p 435–42.
31. Durham WF, Wolfe HR. Measurement of the exposure of workers to pesticides. Bull World Health Organ 1962;26:71–91 and 279–81.
32. Ross J, Thongsinthusak T, Fong HR, Margetich S, Krieger R. Measuring potential dermal transfer of surface pesticide residue generated from indoor fogger use: an interim report I. Chemosphere 1990;20:349–60.
33. Ross J, Fong HR, Thongsinthusak T, Margetich S, Krieger R. Measuring potential dermal transfer of surface pesticide residue generated from indoor fogger use: an interim report II. Chemosphere 1991;22:975–84.
34. Machado Neto JG, Matuo T, Matuo YK. Dermal exposure of pesticide applicators in staked tomato (Lycopersicon esculentum Mill) crops: efficiency of a safety measure in the application equipment. Bull Environ Contam Toxicol 1992;48:529–34.
35. Chester G, Ward M. An accurate method for measuring dermal exposure to pesticides. Hum Toxicol 1983;2:555–6.
36. Williams RL, Bernard CE, Krieger RI. Child-like mannequin for observation and measurement of spray drift particle deposition for pesticide exposure assessment. Bull Environ Contam Toxicol 2001;67:179–86.
37. Staniland LN. Fluorescent tracer techniques for the study of spray and dust deposits. J Agric Eng Res 1958;3:110–25.
38. Fenske RA, Birnbaum SG. Second generation video imaging technique for assessing dermal exposure (VITAE System). Am Ind Hyg Assoc J 1997;58:636–45.
39. Houghton D, Archibald BA, Solomon KR. Review of video imaging techniques for estimating dermal exposure to pesticides. In: Ecobichon DJ, editor. Occupational Hazards of pesticide exposure: sampling, monitoring, measuring. Philadelphia (PA): Taylor and Francis; 1998. p 135–86.
40. Archibald BA, Solomon KR, Stephenson GR. Fluorescent tracer and pesticide penetration through selected protective clothing. Bull Environ Contam Toxicol 1994;53:479–85.
41. Archibald BA, Solomon KR, Stephenson GR. Estimating pirimicarb exposure to greenhouse workers using video imaging. Arch Environ Contam Toxicol 1994;27:126–9.
42. Archibald BA, Solomon KR, Stephenson GR. Estimation of pesticide exposure to greenhouse applicators using video imaging and other assessment techniques. Am Ind J Hyg 1995;56:226–35.
43. Bierman EPB, Brouwer DH, van Hemmen JJ. Imprementation, application and evaluation of the fluorescent tracer technique to assess dermal exposure. Den Haag: TNO Nutrition and Food Research Institute; 1995. Report no V95–629, project 255301.
44. Roff MW. Accuracy and reproducibility of calibrations on the skin using the fives fluorescence monitor. Ann Occup Hyg 1997;41:313–24.
45. Roff MW. Dermal exposure of amateur or non-occupational users to wood-preservative fluids applied by brushing outdoors. Ann Occup Hyg 1997;41:297–311.
46. Roff MW. A novel lighting system for the measurement of dermal exposure using a fluorescent dye and an image processor. Ann Occup Hyg 1994;38:903–19.
47. Bestari KT, Robinson IB, Solomon KR, Steele TS, Day KE, Sibley PK. Distribution and composition of polycyclic aromatic hydrocarbons within experimental microcosms treated with creosote-impregnated Douglas fir pilings. Environ Toxicol Chem 1998;17:2369–77.
48. Houghton D. Development and validation of fluorescent tracer method to estimate dermal exposure to pesticides used indoors [dissertation]. Guelph (ON): University of Guelph; 1997.
49. Roff M, Wheeler J, Baldwin P. Comparison of fluorescence and rinsing methods for assessing dermal exposure. Appl Occup Environ Med 2001;16:319–22.
50. Vaccaro JR, Nolan RJ, Hugo JM. Evaluation of dislodgeable residue and adsorbed dose of chlorphyrifos to crawling infants following indoor broadcast applications of a chlorpyrifos-based emulsifiable concentrate: confidential report. Midland (MI): The Dow Chemical Company; 1992.
51. Fortune CR. Evaluation of methods for collecting dislodgeable pesticide residues from turf. Research Triangle Park (NC): United States Environmental Protection Agency (US EPA), National Exposure Research Laboratory; 1997. Report no EPA/600/R-97/119.
52. Gurunathan S, Robson M, Freeman N, Buckley B, Roy A, Meyer R, et al. Accumulation of chlorpyrifos on residential surfaces and toys accessible to children. Environ Health Perspect 1998;106:9–16.
53. Nigg HN. Occupational monitoring. In: Ecobichon DJ, editor. Occupational hazards of pesticide exposure: sampling, monitoring, measuring. Philadelphia (PA): Taylor and Francis; 1998. p 107–34.
54. Decker P, Cohen B, Butala JH, Gordon T. Exposure to wood dust and heavy metals in workers using CCA pressure-treated wood. J Am Ind Hyg Assoc 2002;63:166–71.
55. Leary JS, Keane WT, Fontenot C, Feichtmeir EF, Schultz D, Koos BA, et al. Safety evaluation in the home of polyvinyl chloride resin strip containing dichlorvos (DDVP). Arch Environ Health 1974;29:308–14.
56. Adgate JL, Clayton CA, Quackenboss JJ, Thomas KW, Whitmore RW, Pellizzari ED, et al. Measurement of multi-pollutant and multi-pathway exposures in a probability-based sam-

ple of children: practical strategies for effective field studies. J Exp Anal Environ Epidemiol 2000;10:650–61.
57. Fenske RA, Lu C, Simcox NJ, Loewenherz C, Touchstone J, Moate TF, et al. Strategies for assessing children's organophosphorus pesticide exposures in agricultural communities. J Exp Anal Environ Epidemiol 2000;10:662–71.
58. Hubal EA, Sheldon LS, Zufall MJ, Burke JM, Thomas KW. The challenge of assessing children's residential exposure to pesticides. J Exp Anal Environ Epidemiol 2000;10:638–49.
59. Lu C, Knutson DE, Fisker-Andersen J, Fenske RA. Biological monitoring survey of organophosphorus pesticide exposure among pre-school children in the Seattle metropolitan area. Environ Health Perspect 2001;109:299–303.
60. Zartarian VG, Ozkaynak H, Burke JM, Zufall MJ, Rigas ML, Furtaw EJ Jr. A modeling framework for estimating children's residential exposure and dose to chlorpyrifos via dermal residue contact and nondietary ingestion. Environ Health Perspect 2000;108:505–14.
61. Tulve NS, Suggs JC, McCurdy T, Cohen Hubal EA, Moya J. Frequency of mouthing behavior in young children. J Exp Anal Environ Epidemiol 2002;12:259–64.
62. Quackenboss JJ, Pellizzari ED, Shubat P, Whitmore RW, Adgate JL, Thomas KW, et al. Design strategy for assessing multi-pathway exposure for children: the Minnesota Children's Pesticide Exposure Study (MNCPES). J Exp Anal Environ Epidemiol 2000;10:145–58.
63. Hawley JK. Assessment of health risks from contaminated soil. Risk Anal 1985;5:289–302.
64. Krieger RI. Biomonitoring human pesticide exposures. In: Ecobichon DJ, editor. Occupational hazards of pesticide exposure: sampling, monitoring, measuring. Philadelphia (PA): Taylor and Francis; 1998. p 187–208.
65. Harris SA, Corey PN, Sass-Kortsak AM, Purdham JT. The development of a new method to estimate total daily dose of pesticides in professional turf applicators following multiple and varied exposures in occupational settings. Int Arch Occup Environ Health 2001;74:345–58.
66. Dong MH, Ross JH. Coping with aggregate pesticide exposure assessment: an integrated approach. In: Krieger RI, editor. Handbook of pesticide toxicology. 2nd ed. New York (NY): Academic Press; 2001. p 479–92.
67. Solomon KR, Harris SA, Stephenson GR. Applicator and bystander exposure to home garden and landscape pesticides. In: Racke KD, Leslie AR, editors. Pesticides in urban environments: fate and significance. Washington (DC): American Chemical Society; 1993. p 262–74.
68. Harris SA, Sass-Kortsak AM, Corey PN, Purdham J. Development of models to predict dose of pesticides in professional turf applicators. J Exp Anal Environ Epidemiol 2002;12:130–44.
69. Stephenson GR, Bowhey CS, Lang SR, Solomon KR. Homeowner-applicator and bystander exposure to chlorpyrifos applied to turfgrass. Int Turf Soc Res J 2001;9:45–9.
70. Solomon KR, Takacs P. Probabilistic risk assessment using species sensitivity distributions. In: Posthuma L, Traas T, Suter GW, editors. Species sensitivity distributions in risk assessment. Boca Raton (FL): CRC Press; 2002. p 285–313.