Community Exposures to Airborne Agricultural Pesticides in California: Ranking of Inhalation Risks

Sharon Lee, Robert McLaughlin, Martha Harnly, Robert Gunier, and Richard Kreutzer

California Department of Health Services, Environmental Health Investigations Branch, Oakland, California, USA

We assessed inhalation risks to California communities from airborne agricultural pesticides by probability distribution analysis using ambient air data provided by the California Air Resources Board and the California Department of Pesticide Regulation. The pesticides evaluated include chloropicrin, chlorothalonil, chlorpyrifos, *S,S,S*-tributyl phosphorotrithioate, diazinon, 1,3-dichloropropene, dichlorvos (naled breakdown product), endosulfan, eptam, methidathion, methyl bromide, methyl isothiocyanate (MITC; metam sodium breakdown product), molinate, propargite, and simazine. Risks were estimated for the median and 75th and 95th percentiles of probability (50, 25, and 5% of the exposed populations). Exposure estimates greater than or equal to noncancer reference values occurred for 50% of the exposed populations (adults and children) for MITC subchronic and chronic exposures, methyl bromide subchronic exposures (year 2000 monitoring), and 1,3-dichloropropene subchronic exposures (1990 monitoring). Short-term chlorpyrifos exposure estimates exceeded the acute reference value for 50% of children (not adults) in the exposed population. Noncancer risks were uniformly higher for children due to a proportionately greater inhalation rate-to-body weight ratio compared to adults and other factors. Target health effects of potential concern for these exposures include neurologic effects (methyl bromide and chlorpyrifos) and respiratory effects (1,3-dichloropropene and MITC). The lowest noncancer risks occurred for simazine and chlorothalonil. Lifetime cancer risks of one-in-a-million or greater were estimated for 50% of the exposed population for 1,3-dichloropropene (1990 monitoring) and 25% of the exposed populations for methidathion and molinate. Pesticide vapor pressure was found to be a better predictor of inhalation risk compared to other methods of ranking pesticides as potential toxic air contaminants. *Key words:* agriculture, air monitoring, fumigants, inhalation exposures, pesticides, risk assessment. ***Environ Health Perspect* 110:1175–1184 (2002). [Online 30 September 2002]**

http://ehpnet1.niehs.nih.gov/docs/2002/110p1175-1184lee/abstract.html

Agricultural pesticides have historically been used in proximity to rural communities in California. Use near populated areas is increasing nationwide, as population growth expands into formerly rural farmland. Pesticides applied in agriculture can travel in the air through processes such as spray drift and post-application volatilization, sometimes for substantial distances (*1–3*). A wide range of agricultural pesticides has been found in ambient air (*1,4,5*). Agricultural pesticides have also been measured in indoor air, sometimes at increased concentrations (*6–8*). There is increasing public health concern regarding potential residential exposures to these agricultural pesticides and limited understanding about the potential for such exposures. Acute health effects, such as eye, respiratory, and gastrointestinal irritation, fatigue, and headaches, have been associated with some instances of agricultural pesticide drift into California communities (*9–11*). However, there is a risk of other, nonacute health effects from airborne agricultural pesticides, many of which are less readily apparent than irritant effects.

Some methods for ranking agricultural pesticides by their potential hazard as air contaminants have been proposed based on use, volatility, toxicity, and so on (*12,13*). Ultimately, the rankings are used to determine exposure reduction or public health priorities. One of the initial uses of the ranking developed by the California Department of Pesticide Regulation (CDPR), called the (pesticide) toxic air contaminant (TAC) ranking, is to direct air monitoring for agricultural pesticides in California (*13*). The California Air Resources Board (CARB) conducts this ambient air monitoring in agricultural communities, which are selected on the basis of area use of the monitored pesticide, and in regional urban centers (*4*). For the monitored pesticides, an opportunity exists to calculate inhalation risk.

In this report we present a screening risk assessment (for both cancer and noncancer effects) of inhalation exposures to agricultural pesticides measured in California community ambient air in high-use agricultural areas between 1986 and 2000. Pesticides included in the assessment are among the top 20 pesticides ranked as potential toxic air contaminants (TACs) by the CDPR or as hazardous air pollutants (HAPs) by the U.S. Environmental Protection Agency (EPA), which have CARB air monitoring data (*13,14*). The pesticide monitoring data include fumigants: chloropicrin (*15*), 1,3-dichloropropene (*16–19*), methyl bromide (*18–20*), and methyl isothiocyanate [MITC (*21*)]; fungicides: captan (*22*) and chlorothalonil (*23*); herbicides: eptam [EPTC (*24*)], linuron (*25*), molinate (*26*), simazine (*27*), and *S,S,S*-tributyl phosphotrithioate [DEF (*28*)]; and insecticides: aldicarb (*29*), chlorpyrifos (*30*), diazinon (*31*), dichlorvos (*32*), endosulfan (*33*), fenamiphos (*34*), methidathion (*35*), phorate (*36*), and propargite (*37*). The air monitoring data for MITC and dichlorvos are based on agricultural use of their parent compounds, metam sodium and naled, respectively. Some of these air monitoring data have been previously reported (*4*). Chronic and short-term inhalation exposures are assessed for adults and for children (*38*).

The conventional approach to risk assessment typically uses single health-conservative exposure values, such as inhalation rate (*39,40*). The resulting risk estimate, while health conservative, gives little information about the likelihood of risk in an exposed population. In contrast, probability analysis, presented here, uses distributions for exposure variables to estimate a range (likelihood) of risks. These risks expressly apply to the populations in the vicinity of the air monitoring stations. The monitored pesticides can be ranked by inhalation risk in the exposed communities using these risk estimates. Estimates of the total California population with a similar exposure potential can also be made by determining the agricultural pesticide use density near the air monitoring locations and then enumerating the California population living in areas with similar or higher use densities.

Methods

Pesticide monitoring. Air monitoring methods have been discussed in detail by Baker et al. (*4*). Briefly, pesticides under evaluation by the CDPR as possible TACs are sampled by CARB in the California county and in month(s) with the reported highest use of

Address correspondence to S. Lee, California Department of Health Services, Environmental Health Investigations Branch, 1515 Clay Street, Suite 1700, Oakland, CA 94612 USA. Telephone: (510) 622-4478. Fax: (510) 622-4505. E-mail: sseidel@dhs.ca.gov

We thank staff of the California Air Resources Board and Department of Pesticide Regulation for discussion of the air monitoring reports, C. Wilder for preparation of the manuscript, and A. Bradman for reviewing the initial manuscript.

The opinions expressed are the views of the authors. They do not necessarily reflect the policies of the California Department of Health Services.

Received 9 November 2001; accepted 26 April 2002.

each pesticide in recent years. In California, all agricultural applications of pesticides are reported, including geographical location and date of application [Pesticide Use Report (PUR) data] (*41*). Complete agricultural pesticide use reporting has been a California requirement since 1990, with restricted pesticide usage reported pre-1990. These PUR data are error checked and maintained by the CDPR. On average, three to four rural agricultural communities and a regional urban comparison site were selected for monitoring of each pesticide. One to two air monitors were placed on the roofs of community buildings such as schools. Air samples were typically collected by pump-and-adsorbent-cartridge capture methods, using low- and medium-volume flow rates. Monitoring for methyl bromide and 1,3-dichloropropene in 2000 also used evacuated Silcosteel canisters (*18,19*). Generally, 24-hr samples were collected each week for several weeks. The community air data are descriptive of average pesticide air concentrations in high-use agricultural regions, since community monitors were not positioned near known field applications.

Pesticides among the top 20 potential California TACs and U.S. EPA HAPs with monitoring data are listed in Table 1. Monitoring data were not available for potential TAC pesticides ranked 6th (*p*-dichlorobenzene), 7th (cyanazine), 12th (alachlor), and 13th (dimethoate). All pesticides have at least one month of community air-monitoring data, except molinate (1.5 weeks), 1,3-dichloropropene in 1990 (2 weeks), and MITC (2 weeks). The statistical analysis of air data in this report differs from that of Baker et al. (*4*), who did not include nondetectable analytic results in the statistical estimate of the mean. We entered nondetectable compounds in estimates of the mean as zero values for pesticides detected in < 10% of air samples, and at one-half the minimum quantitation limit for all other pesticides. 1,3-Dichloropropene monitoring includes community air data collected before its use was suspended in California in 1990 and community data collected following reintroduction in 1996 (*16–19*).

PUR data were obtained electronically from the CDPR for 1986–1999, the most recent year available at the time of this report (*41*). Agricultural pesticide use was evaluated within 1.5–3 miles of each monitoring station, for the year of monitoring. PUR data are available by township, range, and section, a section being approximately 1 mi^2. The adjacent years 1999 and 1989/1991 were used, respectively, for PUR analysis for 2000 monitoring and for 1990, which was a transition year to complete use reporting. We calculated annual pesticide use density (pounds/square mile) within 1.5–3 miles of each air monitoring station for selected pesticides, based on PUR data for the year of monitoring (1999 proxy year for 2000 monitoring). Population estimates were derived for all 1990 California census block groups with annual average pesticide use (pounds/square mile) greater than or equal to that in the vicinity of the air monitoring sites, using methods previously described (*12*).

Risk assessment. The risk assessment evaluates inhalation exposures to adults and children ≤ 12 years of age. Noncancer risks are assessed for chronic (> 1 year), subchronic (≥ 15 days), and acute exposures (typically 1–24 hr) (*40*). Cancer risks assume a lifetime exposure. The following equations were used to estimate inhalation risk:

$$\text{Average Daily Intake (mg/kg/day)} = C_{\text{air}} \times IR \times CF \times EF \times ED, \quad [1]$$

where C_{air} = concentration of pesticide in community air (mg/m^3); IR = inhalation rate (liters/kilogram body weight-day); CF =

Table 1. Community air concentrations (µg/m^3) in California.

		Urban community		All data—rural communities				15-Day max-high community[a]	
Pesticide[b]	TAC rank	*n* > MQL/total[c]	Mean ± SD[d]	*n* > MQL/total[c]	Mean ± SD[d]	GM	Range[e]	Mean ± SD[d]	GM
Propargite	1	3/22	0.014 ± 0.0043	67/152	0.046 ± 0.12	0.024	< 0.023–1.3	0.32 ± 0.39	0.21
Chlorothalonil	2	0/15	< 0.0039[f]	3/45	0.00029 ± 0.0011	0.000053	< 0.0039–0.0046[f]	0.0011 ± 0.0021	0.00013
MITC	3	8/8	2.1 ± 2.4[f]	20/24	4.9 ± 5.6	0.88	< 0.01–18[f]	8.4 ± 5.6	6.4
DEF	4	6/36	0.0013 ± 0.0022	121/125	0.064 ± 0.073	0.028	< 0.0011–0.34[f]	0.19 ± 0.083	0.17
Endosulfan	5	0/19	< 0.0038	66/75	0.018 ± 0.025	0.0011	< 0.0038–0.17	0.047 ± 0.061	0.024
Fenamiphos	8	0/24	< 0.0093	0/92	< 0.0093	< 0.0093	< 0.0093	< 0.0093	< 0.0093
Phorate	9	0/24	< 0.0093	0/96	< 0.0093	< 0.0093	< 0.0093	< 0.0093	< 0.0093
Chlorpyrifos[g]	10	8/21	0.015 ± 0.022	75/82	0.10 ± 0.15	0.058	< 0.0094–0.91	0.23 ± 0.18	0.2
Chloropicrin[h]	11	0/21	< 0.085[f]	20/71	0.21 ± 0.59	0.077	< 0.085–4.6[f]	0.48 ± 1.1	0.15
Molinate	14	NC	NC	10/10	0.54 ± 0.3	0.47	0.16–1.2[f]	0.72 ± 0.31	0.67
Aldicarb	15	0/23	< 0.03	0/92	< 0.03	< 0.03	< 0.03	< 0.03	< 0.03
Linuron	16	0/23	< 0.015	0/90	< 0.015	< 0.015	< 0.015	< 0.015	< 0.015
Methidathion[g]	17	1/17	0.0068 ± 0.028	12/65	0.041 ± 0.092	0.021	< 0.03–0.67	0.13 ± 0.22	0.042
Diazinon	18	3/12	0.011 ± 0.012	30/48	0.025 ± 0.030	0.015	< 0.01–0.16	0.063 ± 0.051	0.047
EPTC	19	0/24	< 0.072	21/96	0.057 ± 0.047	0.047	< 0.072–0.24	0.1 ± 0.078[i]	0.078
Simazine	20	0/24	< 0.0042	21/96	0.0029 ± 0.002	0.0026	< 0.0042–0.018	0.0054 ± 0.0051	0.0041
Captan	HAP	0/14	< 0.013[f]	0/42	< 0.013	< 0.013	< 0.013[f]	< 0.013	< 0.013
1,3-Dichloropropene[j]	HAP								
1990		8/8	0.9 ± 0.98[f]	32/32	24 ± 39	8.9	0.3–160[f]	42 ± 54	22
1996		16/21	0.57 ± 0.78	64/84	1.4 ± 2.3	0.43	< 0.1–13	3.1 ± 4.3	1.5
2000a[k]		9/23	0.76 ± 1.5	41/118	2.7 ± 13	0.1	< 0.05–135	22 ± 45	0.99
2000b[k]		5/30	0.048 ± 0.072	36/149	0.2 ± 0.59	0.046	< 0.05–4.3	1.1 ± 1.5	0.24
Dichlorvos[l]	HAP	3/16	0.013 ± 0.0065	11/64	0.014 ± 0.0094	0.012	< 0.02–0.059[f]	0.023 ± 0.018	0.018
Methyl bromide	HAP								
1986[h]		0/21	< 4.2[f]	2/71	0.12 ± 0.69	0.048	< 4.2–4.4[f]	0.34 ± 1.2[m]	0.062
2000a[k]		23/23	0.69 ± 1.0	117/118	2.5 ± 6.7	0.58	< 0.036–55	9 ± 13[m]	2.9
2000b[k]		30/30	5.2 ± 6.0	149/149	12 ± 21	3.9	0.23–119	33 ± 34[m]	19

Abbreviations: GM, geometric mean; MQL, minimum quantitation limit; *n* > MQL/total, number of samples > MQL, over the total number of samples; NC, not conducted.
[a]Community with the highest ambient air concentrations over a 15-day consecutive period, unless noted otherwise. [b]No air monitoring was conducted for potential TAC pesticides ranked: 6th, *p*-dichlorobenzene; 7th, cyanazine; 12th, alachlor; 13th, dimethoate. [c]Number of samples excludes blanks, spikes, and co-located samples. [d]Nondetects are included as one-half the MQL for pesticides detected in ≥ 10% of samples, and as zero values (arithmetic means) or MQL/100 (geometric means) for those detected in < 10% of samples. [e]MQL (or minimum if all samples > MQL) to maximum sample concentration. [f]Previously reported (*4*). [g]Chlorpyrifos and methidathion oxon data summed with parent data using the conversion (molecular weight parent/molecular weight oxon) × oxon concentration = parent equivalent. [h]Two consecutive 4-hr samples per 24 hr for chloropicrin and methyl bromide (1986 only); all others 24-hr samples. [i]22-Day mean. [j]1990 monitoring for 1,3-dichloropropene before suspension in California. (*16*); 1996, 2000 monitoring following reinstatement in California (*17–19*). [k]The 2000a monitoring location had high use of 1,3-dichloropropene and secondary use of methyl bromide, whereas 2000b had high use of methyl bromide and secondary use of 1,3-dichloropropene. [l]Dichlorvos, breakdown product of naled. [m]6-Week mean in 2000; length of monitoring (3.5 weeks) in 1986.

conversion factor (0.001 m^3/L air); *EF* = exposure frequency (all chronic; months/12 months); *ED* = exposure duration (cancer risk; years/70 years). For risk,

$$\text{Noncancer Risk (hazard quotient)} = \text{Intake (mg/kg-day)/RfD (mg/kg-day)} \quad [2]$$

$$\text{Cancer Risk} = \text{Intake (mg/kg-day)} \times \text{PF (mg/kg-day)}^{-1}. \quad [3]$$

Noncancer risk is defined as the ratio (hazard quotient; HQ) of the estimated intake to the reference dose (RfD). The RfD is the dose at or below which adverse noncancer health effects are not estimated to occur. Noncancer risks from exposures to pesticides with nonsystemic (portal-of-entry) effects (e.g., respiratory irritation) were assessed by eliminating IR and CF from Equation 1; in other words, the effect is dependent on air concentration. The resulting exposure estimate in milligrams per cubic meter is divided by the reference value in milligrams per cubic meter. Cancer risk estimates (Equation 3) use the potency factor (PF), a numerical estimate of the potency of the carcinogen. The PF multiplied by the estimated intake yields an estimate of the cancer risk over a lifetime from that exposure alone.

For probability distributions of risk, we conducted Latin hypercube analysis using commercial software, Crystal Ball v. 2000 (*42*). We used 10,000 equation solutions, or trials, to define the range of risk for each exposure scenario. The sample size for Latin hypercube sampling was 5,000. Contribution to variance was used for sensitivity analysis. Probability distributions were defined for pesticide air concentrations, inhalation rates, and exposure frequencies. Variables are listed in Tables 2 and 3.

Air concentrations. Log-normal distributions were identified for the pesticide air monitoring data based on preliminary histogram analysis (not shown). Pesticide concentrations listed in Table 1 in micrograms per cubic meter were converted to milligrams per cubic meter, natural log transformed, and means and standard deviations calculated on the transformed data set for the log-normal distribution (not shown). Air data from all rural communities combined (Table 1) were used to estimate chronic exposures. We used the highest 15-consecutive-day air concentration to estimate subchronic exposures (Table 1), except for EPTC and methyl bromide. These used 22-day and 6-week intervals, respectively, corresponding to the intervals used to establish their subchronic RfDs (Table 4). As with the air data used for chronic exposure estimates, means and standard deviations were calculated on natural log-transformed data for subchronic exposures (not shown). Acute exposure estimates used the sample maximum concentration measured in community ambient air (Table 1). Because of this, the air concentration in the equation for all acute exposures is a single value rather than a distribution.

Inhalation rate. Inhalation rate distributions are based on analysis of ventilation rate data for a population cross-section (*43*). Inhalation rates follow a gamma distribution. They are defined for a child (≤ 12 years) and an adult (> 12 years) for noncancer risk, and a lifetime (0–70 years) for cancer risk.

Exposure frequency. Exposure frequency refers to the fraction of a year over which an exposure occurs (e.g., 3 months/12 months = 0.25). It applies only to chronic exposures, which are, by definition, a year or more (*40*). EF estimates were based on analysis of pesticide use report (PUR) data for the agricultural sections immediately surrounding each air monitoring site, typically within a 1.5-mile radius (*41*). For methyl bromide, MITC, and 1,3-dichloropropene, the monitoring data showed elevated air concentrations in background urban sites, and/or there was no reported use in a 1.5-mile radius at the time when air monitoring showed the presence of the pesticide. For these pesticides, we expanded the radius for PUR analysis to 3 miles. "Triangular distributions," used when estimates of the minimum, most likely mean, and maximum points in the distribution are available, were used to describe exposure frequency. In the triangular distribution, the minimum chronic exposure was assumed to be 1 day/year for all pesticides. The most likely exposure period included months with agricultural pesticide use at least 50% of that in air monitoring month(s), in the radius around each monitoring site. In the case of DEF, the most likely exposure period equaled the monitoring period, approximately 10 weeks of the year (*28*). The maximum exposure period included all months where use of each pesticide was ≥ 10 pounds in the defined radius. [In the absence of indoor air monitoring data, daily exposures assume a 24-hr exposure at the measured ambient air concentrations. Supporting this are other study findings of comparable or higher concentrations of a range of agricultural pesticides, including MITC, indoors compared to outdoors (*6–8*)].

Exposure duration. An ED equal to 1 (a lifetime) is assumed for the cancer risk assessment. It was chosen by default because factors

Table 2. Distribution parameters for inhalations rates and air concentrations.

Variable (reference)	Distribution	Parameters: Location	Scale	Shape
Inhalation rate (L/kg-day)	Gamma			
Child ≤ 12 years (*43*)		301.67	29.59	5.06
Adult > 12 years (*43*)		163.95	45.39	1.51
Lifetime (*43*)		193.99	31.27	2.46
Air concentration (mg/m^3)	Log normal		μ, σ of ln-transformed data	

Table 3. Distribution parameters for exposure frequencies (months/12 months).

Pesticide	Parameters: Minimum[a]	Likeliest[b]	Maximum[c]
Chloropicrin	0.003	0.25	0.33
Chlorothalonil	0.003	0.67	1.0
Chlorpyrifos	0.003	0.25	0.75
DEF	0.003	0.19	0.21
Diazinon	0.003	0.25	0.67
1,3-Dichloropropene			
1990	0.003	0.25	0.33
1996	0.003	0.17	0.17
2000a	0.003	0.42	0.5
2000b	0.003	0.67	0.83
Dichlorvos	0.003	0.17	0.17
Endosulfan	0.003	0.17	0.17
EPTC	0.003	0.25	0.25
Methidathion	0.003	0.25	0.58
Methyl bromide			
1986	0.003	0.42	0.75
2000a	0.003	0.42	0.42
2000b	0.003	0.17	0.5
MITC	0.003	0.5	0.58
Molinate	0.003	0.08	0.25
Propargite	0.003	0.17	0.25
Simazine	0.003	0.17	0.5

Data from the CDPR (*41*). Distribution was triangular.
[a]A minimum exposure of 1 day/365 days was assumed for all pesticides. [b]Number of months per 12 months with reported pesticide use ≥ 50% of the use during air sampling month(s) within a 1.5-mile radius (or 3 mile radius for 1,3-dichloropropene, methyl bromide, and MITC) of the sampling site. [c]Number of months per 12 months with reported pesticide use ≥ 10 pounds within a 1.5-mile radius (or 3 mile radius for 1,3-dichloropropene, methyl bromide, and MITC) of the sampling site.

such as mobility of the population are not well defined. In the risk assessment, we averaged air concentrations over all rural communities monitored for a pesticide as a proxy for chronic "regional" exposures. These regions include distances within which an average resident might reasonably be expected to move, using national data on number of miles moved by home buyers (*44*).

Reference doses and potency factors. Table 4 lists noncancer RfDs and cancer PFs. These are from the U.S. EPA (*45–54*), the CDPR (*55–64*), the California Office of Environmental Health Hazard Assessment (OEHHA) (*65,66*), and the Agency for Toxic Substances and Disease Registry (ATSDR) (*67,68*). RfDs are listed in Table 4 by exposure duration (acute, subchronic, or chronic) and corresponding target organ/toxicity. Note that the term "RfD" is used in Table 4 headings to indicate all noncancer reference values cited from various sources, avoiding the use of multiple terms developed by various agencies, such as reference concentration (*45*), minimum risk level (*67*), or reference exposure level (*65*). Reference values shown in air concentration units of milligrams per cubic meter, rather than milligrams per kilogram body weight, are based on portal-of-entry effects. PFs are listed, with the cancer classification of the U.S. EPA Office of Pesticide Programs (*50*). When available, values based on inhalation studies were always chosen over oral values. Values based on oral studies are indicated in Table 4 by (o), or (o→i), indicating oral to inhalation extrapolation by the listing agency. Where agencies/programs listed values that differed from one another by > 2-fold, the high and low values are both listed in Table 4. This occurred for the following: chlorpyrifos, 1,3-dichloropropene, dichlorvos, EPTC, and molinate with chronic RfDs; diazinon, EPTC, and molinate with subchronic RfDs; 1,3-dichloropropene, methyl bromide, and MITC with acute RfDs; and 1,3-dichloropropene and dichlorvos with cancer PFs. In these cases, risks were estimated separately using each of the two values.

With two exceptions, all of the RfDs and PFs listed in Table 4 are based on administered doses, with no adjustment for absorption by the listing agency. The exceptions, for DEF and dichlorvos, are footnoted in Table 4, and the absorption factor is included in Equation 1 of their exposure assessment. Acute RfDs have not been published for endosulfan, propargite, and simazine. In these cases, we identified the no-observed-adverse-effect level (NOAEL) from the most sensitive teratology study on file with the CDPR (*58*). The teratology studies were chosen because they are well-reviewed, short-term studies, albeit oral, on a potentially sensitive subpopulation of pregnant animals. The NOAEL was divided by the standard default uncertainty factor of 100 (10 for extrapolation from an animal to human population and 10 for potentially sensitive human subpopulations) to estimate an acute RfD for these three pesticides.

The federal Food Quality Protection Act (FQPA) of 1996 directs the U.S. EPA to use an additional safety factor of up to 10-fold, if necessary, to account for data uncertainties when evaluating pesticide risks to infants and children (*38*). The U.S. EPA is in the process of assigning these FQPA safety factors to pesticides that may pose additional risks to children. Available FQPA factors are listed in Table 4. In this risk assessment, the "adult" RfDs shown in Table 4 were divided by the available FQPA factor when assessing all risks to children.

Results

Noncancer and cancer risks are estimated for the 50th, 75th, and 95th percentiles of likelihood of risk (probability estimates, Tables 5–7) in the monitored communities. Two risk entries per pesticide exposure scenario indicate use of two different RfDs or PFs (Table 4). These pairs denote a range of RfD or PF values used by different agencies or programs, and, consequently, a greater range in the risk estimates for these pesticide exposure scenarios. The range was most notable for MITC acute noncancer risks.

Table 4. RfDs and PFs of pesticides found in air.[a]

Pesticide	FQPA factor[b]	Acute RfD (mg/kg/24 hr)[c]	Target toxicity	Subchronic RfD (mg/kg/day)[c]	Target toxicity	Chronic RfD (mg/kg/day)[c]	Target toxicity	Cancer classification[d]	PF (mg/kg/day)$^{-1}$[d]
Chloropicrin	NE	0.029 mg/m^3 (1hr) (*65*)	Ie,Ir	0.001 mg/m^3 (*64*)	R	0.001 mg/m^3 (*64*)	R	NE	
Chlorothalonil	1×	0.02 (o→i) (*46*)	K	0.02 (o→i) (*46*)	K	0.02 (o→i) (*46*)	K	Likely	7.66×10^{-3} (o) (*46*)
Chlorpyrifos	10×	0.001 (*47*)	Nch	0.001 (*47*)	Nch	0.0003 (o→i) (*47*) 0.003 (o) (*45*)	Nch Nch	Not likely	
DEF	10×	0.006 (*55*)	Nch	0.006 (*55*)	Nch	0.009 (*48*)	Nch	Likely (↑ dose) Not likely (↓ dose)	8.4×10^{-2} (o→i) (*55*)
Diazinon	1×	0.00009 (*49*)	Nch	0.00009 (*49*) 0.0026[e]	Nch Nch	0.00009 (*49*)	Nch	Not likely	
Dichloropropene	1×	0.55 (*56*) 0.1[h]	W W	0.014 mg/m^3[f]	R	0.02 mg/m^3 (*45*) 0.009 mg/m^3[i]	R R	Likely	1.4×10^{-2}[g] 5.5×10^{-2} (*56*)
Dichlorvos	3×	0.0033 (*57*)	Nch	0.0008[j]	Nch	0.00014[k] 0.0005 (*51*)	Nch Nch	Possible/ likely	7.68×10^{-2} (o) (*50*) 3.5×10^{-1} (o→i) (*57*)
Endosulfan	NE	0.007 (o) (*58*)	D	0.006 (o) (*52*)	K, W	0.006 (o) (*45*)	K, W	Not likely	
EPTC	10×	0.15[l]	C	0.007 (*59*) 0.022 (> 21 d)[m]	H, R H, Nch, W	0.005 (o) (*59*) 0.025 (o) (*53*)	N C, Rep	Not likely	
Methidathion	1×	0.002 (o→i) (*54*)	Nch	0.002 (o→i) (*54*)	Nch	0.002 (o→i) (*54*)	Nch	Possible	5.3×10^{-1} (o) (*60*)
Methyl bromide	NE	0.21[n] 0.056[p]	D N	0.002 (6 wk)[o]	N	0.005 mg/m^3 (*45*)	R	Inadequate evidence	
MITC	NE	0.066 mg/m^3 (1–8 hr) (*62*) 0.001 mg/m^3 (4 hr) (*66*)	Ie Ie	0.003 mg/m^3 (*62*)	R	0.0003 mg/m^3 (*62*)	R	NE	
Molinate	NE	0.12 (o→i) (*63*)	Rep	0.0048 (o→i) (*63*) 0.002 (o) (*52*)	Rep Rep	0.002 (o) (*45*) 0.01 (o→i) (*63*)	Rep N	Possible	4.92×10^{-2} (o) (*50*)
Propargite	NE	0.02 (o) (*58*)	D	Adopted chronic		0.02 (o) (*45*)	D	Likely	2.01×10^{-1} (o) (*50*)
Simazine	NE	0.05 (o) (*58*)	D	0.005 (o) (*52*)	W, H	0.005 (o) (*45*)	W, H	Possible	1.2×10^{-1} (o) (*50*)

Abbreviations: C, cardiovascular; D, developmental; H, hematologic; Ie, eye irritation; Ir, respiratory irritation; K, renal; N, neurologic; Nch, cholinesterase inhibition; NE, not established; R, respiratory tract; Rep, reproductive; W, whole body. Values are based on inhalation studies, unless noted, as oral (o) or oral-to-inhalation (o→i) route extrapolation by listing agency.
[a]Where values differed by > 2-fold between agencies/programs, the high and low values are both listed; values are based on administered doses except the DEF PF and dichlorvos acute RfD (70% and 50% assumed absorption, respectively). [b]Food Quality Protection Act (FQPA) safety factor (*38*); adult RfDs are divided by the FQPA factor when assessing risks to infants/children. [c]RfDs are in units of milligrams per kilogram per day except those based on nonsystemic (portal-of-entry) effects (mg/m^3). Tabled RfDs have not been divided by FQPA factors; acute RfDs are 24 hr unless noted otherwise. [d]Human cancer classification (*50*); PF, Q_1^*. Original citation units, if different from above, and unit conversion references: [e]0.009 mg/m^3 (*52,68*); [f]0.003 ppm (*52,68*); [g]4×10^{-6} (µg/m^3)$^{-1}$ (*45,52*); [h]0.024 ppm (*61,64*); [i]0.002 ppm (*52,68*); [j]0.0003 ppm (*52,68*); [k]0.0005 mg/m^3 (*45,52*); [l]0.58 µg/L (*53,101*); [m]0.083 µg/L (*53,101*); [n]0.21 ppm (*61*); [o]0.002 ppm (*61*); [p]0.05 ppm (*52,68*).

Noncancer risks are presented in Table 5 for children ≤ 12 years of age and in Table 6 for adults. Risks are ranked in approximately ascending order. Results are presented as HQs, that is, intake divided by the reference dose in milligrams per kilogram-day, or exposure divided by the reference value in milligrams per cubic meter (Equation 2). [MITC and chloropicrin acute risk estimates are presented as point estimates. For these, the only exposure distribution (inhalation rate) is eliminated from the equation because MITC and chloropicrin acute target health effects are nonsystemic.] The risks to children are consistently greater than for adults because children have a greater inhalation-to-body weight ratio and, in some cases, because the FQPA factor for children lowered the reference dose (Table 4). Four pesticides have HQs > 1 for an estimated 25–50% of the exposed populations of children (75th and 50th percentiles of risk). These include MITC for subchronic and chronic exposures; chlorpyrifos for acute and subchronic exposures; 1,3-dichloropropene for subchronic exposures in 1990; and methyl bromide for subchronic exposures in 2000a and 2000b (Table 5). (The uncertainty between the two acute HQs for MITC, 18 versus 0.3, limits the acute MITC risk interpretation.)

In 2000, joint air monitoring was conducted for methyl bromide and 1,3-dichloropropene in two regions in California, identified in Tables 1 and 3 and Tables 5–8 as 2000a and 2000b. The 2000a monitoring was in the county with high use of 1,3-dichloropropene (and secondary use of methyl bromide), while 2000b was in counties with high use of methyl bromide (and secondary use of 1,3-dichloropropene). While the available methyl bromide air monitoring data do not reflect the history of regulatory actions, this may be due to sampling limitations in the 1986 air monitoring, which used a method with a much higher minimum quantitation limit (4.2 μg/m^3) compared to later sampling (Table 1) (*4,18,19*).

Reference doses are based on studies identifying the most sensitive target organ(s) and critical health effect(s) for a length of exposure. For MITC, the critical effect for subchronic and chronic exposures is respiratory: nasal epithelial atrophy in animal studies (*62*). The critical subchronic effect for methyl bromide exposure is neurologic: decreased responsiveness in animal studies (*61*). Chlorpyrifos acute and subchronic critical effects are also neurologic: enzyme cholinesterase inhibition in animals (*47*). The 1,3-dichloropropene critical subchronic effect is respiratory: nasal epithelial changes, also in animal studies (*56*). An HQ > 1 is generally a trigger for regulatory scrutiny. However, because uncertainty (safety) factors, typically 100-fold, are incorporated into the reference values, this does not necessarily mean that an individual will become ill from such an exposure.

In some cases, particularly chloropicrin and MITC, the sampling intervals were greater than the RfD interval. The chloropicrin samples were taken over 4 hr, while the acute RfD is for a 1-hr exposure. MITC samples were over 24 hr, while the acute MITC RfDs are for 1- to 8-hr exposures (Tables 1 and 4). These acute RfDs were used here without modification, as it was beyond the scope of our analysis to rescale them. Risks for these acute exposures consequently may be underestimated.

Lifetime cancer risks for pesticides with cancer potential and available cancer potency factors are presented in Table 7. Increased regulatory scrutiny of cancer risk often occurs when the estimated risk reaches 1×10^{-6} to 1×10^{-5}, 1/1,000,000 to 1/100,000, excess lifetime cancer risk. Lifetime cancer risks that reach or exceed 1×10^{-6} for an estimated 25–50% of the exposed populations include 1,3-dichloropropene for 1990, methidathion, and molinate (Table 7). The uncertainties are relatively greater for methidathion and molinate estimates compared to 1,3-dichloropropene. Methidathion and molinate are listed by the U.S. EPA as possible human carcinogens (limited evidence), while 1,3-dichloropropene is a probable human carcinogen and, unlike methidathion and molinate, has cancer PFs specific to the inhalation route (Table 4). As with noncancer risks, the true cancer risks to an individual are likely to be lower, due to upper bound estimates established for potency factors and other health-conservative assumptions.

1,3-Dichloropropene use permits were suspended in California in 1990 after high concentrations were found in community air (*16*). The reinstatement of 1,3-dichloropropene permits in 1996 included a number of California-specific regulatory controls. Cancer risks for 1,3-dichloropropene are reduced for the subsequent monitoring years in 1996 and 2000 (Table 7).

In this risk assessment, the variability in ambient air concentrations contributed the largest part of the variance in probability distributions for chronic and subchronic exposures (acute exposure estimates used the maximum air concentration). Several pesticides have air concentrations that span two to

Table 5. Child noncancer HQs (two HQs are calculated where two reference values are available).

	50th, 75th, 95th percentile probability estimates (≤ 12 years old)		
Pesticide	Acute HQ	Subchronic HQ	Chronic HQ
MITC	18.0[a,b]	2.1, 3.8, 8.5[a]	1.0, 6.8, 118[a]
	0.3[a,b]	NA	NA
Methyl bromide			
2000b	0.9, 1.0, 1.2	4.3, 9.1, 27.0	0.2, 0.4, 2.0[a]
	0.3, 0.3, 0.3	NA	NA
2000a	0.4, 0.5, 0.6	0.6, 2.4, 15.4	0.03, 0.09, 0.4[a]
	0.1, 0.1, 0.2	NA	NA
1986	0.03, 0.04, 0.05	0.01, 0.03, 0.1	0.003, 0.006, 0.01[a]
	0.009, 0.01, 0.01	NA	NA
Chlorpyrifos	4.0, 4.5, 5.2	0.9, 1.3, 2.2	0.3, 0.6, 1.7
	NA	NA	0.03, 0.06, 0.2
1,3-Dichloropropene			
1990	0.7, 0.8, 0.9	1.6, 3.5, 11.5[a]	0.2, 0.5, 2.0[a]
	0.1, 0.1, 0.2	NA	0.08, 0.2, 0.9[a]
2000a	0.6, 0.6, 0.7	0.07, 0.6, 15.5[a]	0.003, 0.01, 0.1[a]
	0.1, 0.1, 0.1	NA	0.001, 0.006, 0.05[a]
1996	0.06, 0.06, 0.07	0.1, 0.3, 0.9[a]	0.005, 0.02, 0.08[a]
	0.01, 0.01, 0.01	NA	0.002, 0.007, 0.03[a]
2000b	0.02, 0.02, 0.02	0.02, 0.08, 0.7[a]	0.002, 0.006, 0.02[a]
	0.003, 0.004, 0.004	NA	0.001, 0.003, 0.01[a]
Diazinon	0.8, 0.9, 1.0	0.2, 0.4, 0.9	0.02, 0.05, 0.1
	NA	0.008, 0.01, 0.03	NA
Chloropicrin	0.2[a,b]	0.2, 0.4, 1.4[a]	0.01, 0.03, 0.09[a]
DEF	0.3, 0.3, 0.3	0.1, 0.2, 0.3	0.002, 0.005, 0.02
Methidathion	0.1, 0.2, 0.2	0.009, 0.02, 0.09	0.001, 0.002, 0.006
Molinate	0.004, 0.005, 0.006	0.2, 0.2, 0.3	0.01, 0.02, 0.04
	NA	0.06, 0.09, 0.1	0.002, 0.004, 0.007
EPTC	0.007, 0.008, 0.009	0.05, 0.09, 0.2	0.007, 0.01, 0.02
	NA	0.02, 0.03, 0.06	0.001, 0.002, 0.004
Dichlorvos	0.01, 0.01, 0.02	0.03, 0.05, 0.1	0.01, 0.02, 0.03
	NA	NA	0.004, 0.005, 0.009
Propargite	0.03, 0.03, 0.04	0.005, 0.009, 0.02	0.00007, 0.0001, 0.0004
Endosulfan	0.009, 0.01, 0.01	0.002, 0.004, 0.01	0.00008, 0.0002, 0.0005
Simazine	0.0002, 0.0002, 0.0002	0.0004, 0.0006, 0.001	0.00005, 0.00007, 0.0001
Chlorothalonil	0.0001, 0.0001, 0.0001	0.000003, 0.00001, 0.0001	0.0000006, 0.000001, 0.000005

NA, not applicable. HQ = intake (mg/kg/day)/reference value (mg/kg/day), unless otherwise indicated.
[a]Exposure (mg/m^3)/reference value (mg/m^3) (see Table 4). [b]Point estimate (no probability distributions in equation).

three orders of magnitude (Table 1). For chronic exposures, the order of percent contribution to variance (mean ± SD) was air concentration (75 ± 16), exposure frequency (22 ± 14), and inhalation rate (3 ± 3). The percent contribution to variance for subchronic exposures was air concentration (96 ± 4) and inhalation rate (4 ± 4; mean ± SD). (Exposure frequency applies only for chronic exposures.)

Conventional point estimates of risk are typically more conservative than 50th percentile estimates of risk presented here. This is due to the use of conservative exposure assumptions (e.g., arithmetic mean air concentrations, upper bound inhalation rate estimates). In comparison to stochastic risk estimates, use of conventional assumptions generally resulted in point estimates of risk at or above the 75th percentile (not shown).

In this risk assessment, community exposures and risks were characterized for the populations within a few miles of the air monitoring stations. We also estimated the total California population living in census block groups with a similar or greater pesticide use density, compared to the monitored communities (Table 8). Annual pesticide use density (pounds per square mile) was calculated in the vicinity of the air monitoring sites for chlorpyrifos, metam sodium, methyl bromide in 2000 monitoring (2000b), and 1,3-dichloropropene in 2000 monitoring (2000a). Methyl bromide had the largest estimate of the total exposed California population, 208,757, followed by metam sodium (MITC), 185,441, and 1,3-dichloropropene, 43,246. Chlorpyrifos had the lowest estimate of the total exposed California population, 2,523.

Table 9 shows pesticide use in the county of air monitoring. Use in the year of monitoring is compared to average use during 1991–1999, for both total use and use per square mile of agricultural land in the county (*69*). For many pesticides, use in the year of monitoring is generally representative of average use over the past several years. For naled (dichlorvos parent), a year-by-year analysis shows an apparent steady decline in use (not shown). The greatest increase in county use occurs for metam sodium (the parent of MITC). A yearly analysis shows an increase of 3- to 5-fold every year from 1995 onward, compared to 1993 (not shown).

Average annual MITC use, within 3 miles of the community air monitoring locations, has increased more than 2-fold since the 1993 monitoring (not shown).

We evaluated several predictors of the chronic inhalation risks estimated in this report, using Spearman rank correlation coefficients. The California ranking for potential pesticide toxic air contaminants (Table 1) was not significantly correlated with the child chronic risk ranking ($r = 0.22$, $p = 0.43$). For example, propargite and chlorothalonil were first and second in the pesticide toxic air contaminant ranking, but these pesticides were found to have among the lowest inhalation risks. The chronic reference dose ranking (Table 4) was significantly correlated with the child risk ranking ($r = 0.63$, $p = 0.01$). The pesticide vapor pressure ranking (*70*) was the best predictor of the child chronic risk ranking ($r = 0.70$, $p = 0.003$). Similarly, vapor pressure ($r = 0.60$, $p = 0.12$) was a better predictor of lifetime cancer risk ranking (Table 7) than the cancer potency factor ($r = -0.07$, $p = 0.87$). Vapor pressure has been highly correlated with downwind pesticide concentrations in previous studies (*71*). Among the 15 pesticides in this study with detectable air concentrations, vapor pressure was also highly correlated with the geometric mean air concentrations in rural communities ($r = 0.77$, $p < 0.001$).

Discussion

Of the pesticides ranked in this screening risk assessment, the agricultural fumigants present the highest noncancer and cancer inhalation risks. These include MITC, methyl bromide, and 1,3-dichloropropene. MITC and 1,3-dichloropropene are two of the fumigants being proposed as replacements for methyl

Table 6. Adult noncancer HQs (two HQs are calculated where two reference values are available).

Pesticide	50th, 75th, 95th percentile probability estimates		
	Acute HQ	Subchronic HQ	Chronic HQ
MITC	18.0[a,b]	2.1, 3.8, 8.5[a]	1.0, 6.8, 118[a]
	0.3[a,b]	NA	NA
Methyl bromide			
2000b	0.5, 0.5, 0.7	2.2, 4.7, 13.9	0.2, 0.4, 2.0[a]
	0.1, 0.1, 0.2	NA	NA
2000a	0.2, 0.3, 0.3	0.3, 1.2, 7.9	0.03, 0.09, 0.4[a]
	0.06, 0.07, 0.09	NA	NA
1986	0.02, 0.02, 0.03	0.007, 0.02, 0.06	0.003, 0.006, 0.01[a]
	0.005, 0.005, 0.007	NA	NA
1,3-Dichloropropene			
1990	0.3, 0.4, 0.5	1.6, 3.5, 11.5[a]	0.2, 0.5, 2.0[a]
	0.06, 0.08, 0.1	NA	0.08, 0.2, 0.9[a]
2000a	0.3, 0.3, 0.4	0.07, 0.6, 15.5[a]	0.003, 0.01, 0.1[a]
	0.05, 0.06, 0.08	NA	0.001, 0.006, 0.05[a]
1996	0.03, 0.03, 0.04	0.1, 0.3, 0.9[a]	0.005, 0.02, 0.08[a]
	0.005, 0.006, 0.008	NA	0.002, 0.007, 0.03[a]
2000b	0.009, 0.01, 0.01	0.02, 0.08, 0.7[a]	0.002, 0.006, 0.02[a]
	0.002, 0.002, 0.003	NA	0.001, 0.003, 0.01[a]
Chloropicrin	0.2[a,b]	0.2, 0.4, 1.4[a]	0.01, 0.03, 0.09[a]
Diazinon	0.4, 0.5, 0.6	0.1, 0.2, 0.5	0.01, 0.02, 0.07
	NA	0.004, 0.007, 0.02	NA
Chlorpyrifos	0.2, 0.2, 0.3	0.04, 0.07, 0.1	0.02, 0.04, 0.1
	NA	NA	0.002, 0.004, 0.01
Methidathion	0.07, 0.09, 0.1	0.005, 0.01, 0.05	0.0006, 0.001, 0.003
Molinate	0.002, 0.003, 0.003	0.08, 0.1, 0.2	0.005, 0.009, 0.02
	NA	0.03, 0.04, 0.07	0.001, 0.002, 0.004
DEF	0.01, 0.01, 0.02	0.006, 0.009, 0.02	0.00009, 0.0003, 0.001
Propargite	0.01, 0.02, 0.02	0.002, 0.004, 0.01	0.00004, 0.00007, 0.0002
Dichlorvos	0.002, 0.002, 0.003	0.005, 0.009, 0.02	0.002, 0.003, 0.006
	NA	NA	0.0006, 0.0009, 0.002
Endosulfan	0.004, 0.005, 0.007	0.0009, 0.002, 0.007	0.00004, 0.00009, 0.0002
EPTC	0.0003, 0.0004, 0.0005	0.003, 0.004, 0.01	0.0003, 0.0005, 0.001
	NA	0.0008, 0.001, 0.003	0.00007, 0.0001, 0.0002
Simazine	0.00008, 0.00009, 0.0001	0.0002, 0.0003, 0.0007	0.00002, 0.00004, 0.00007
Chlorothalonil	0.00005, 0.00006, 0.00008	0.000001, 0.000006, 0.00005	0.0000003, 0.0000007, 0.000003

NA, not applicable. HQ = intake (mg/kg/day)/reference value (mg/kg/day), unless otherwise indicated.
[a]Exposure (mg/m^3)/reference value (mg/m^3) (see Table 4). [b]Point estimate (no probability distributions in equation).

Table 7. Lifetime cancer risks.

Pesticide	Percentile probability estimates		
	50th	75th	95th
1,3-Dichloropropene[a]			
1990	2×10^{-5}	6×10^{-5}	3×10^{-4}
	6×10^{-6}	2×10^{-5}	7×10^{-5}
2000a	4×10^{-7}	2×10^{-6}	2×10^{-5}
	1×10^{-7}	5×10^{-7}	4×10^{-6}
1996	7×10^{-7}	2×10^{-6}	1×10^{-5}
	2×10^{-7}	5×10^{-7}	3×10^{-6}
2000b	3×10^{-7}	8×10^{-7}	3×10^{-6}
	8×10^{-8}	2×10^{-7}	8×10^{-7}
Methidathion	7×10^{-7}	1×10^{-6}	4×10^{-6}
Molinate	6×10^{-7}	1×10^{-6}	2×10^{-6}
Propargite	2×10^{-7}	3×10^{-7}	9×10^{-7}
DEF	6×10^{-8}	2×10^{-7}	7×10^{-7}
Dichlorvos	1×10^{-7}	2×10^{-7}	3×10^{-7}
	3×10^{-8}	4×10^{-8}	7×10^{-8}
Simazine	2×10^{-8}	3×10^{-8}	5×10^{-8}
Chlorothalonil	6×10^{-11}	1×10^{-10}	5×10^{-10}

Risk = intake(mg/kg/day) × potency factor $(mg/kg/day)^{-1}$. Risk interpretation examples: 1×10^{-6} = 1/1,000,000 lifetime excess cancer risk; 2×10^{-4} = 2/10,000 lifetime excess cancer risk.
[a]Two estimates are calculated because two potency factors were available (see Table 4).

bromide. The actual health impacts of exposure to these pesticides may be less than estimated here, due to the use of health-conservative factors common in risk assessment (e.g., uncertainty factors of 10–100 or more incorporated into reference doses and upper-bound estimates of cancer potency factors). This is also a screening risk assessment with a number of uncertainties, including the existence of differing reference values and potency factors which can influence the risk estimates (e.g., the acute hazard quotients for MITC). These differences, however, do not alter the overall risk ranking and conclusions of the report. Our risk estimates suggest caution in the expanded use of these fumigants.

Atmospheric dispersion modeling has been used across census tracts in the contiguous United States to rank risks for a broad range of federal HAPs (*72*). Our report is distinct in using actual community air monitoring data to rank risks from inhalation exposures to agricultural pesticides. Our risk assessment focuses on California communities in high-use pesticide areas, at or above the 90th percentile of density of use for most pesticides (*12*). Hence, the risk assessment characterizes the most exposed populations of California. It is worth noting that the pesticide air concentrations in this risk assessment are ambient community air measurements, not measurements near field applications. Near-field air concentrations are typically much higher than ambient community air data (*4*). In the absence of effective regulatory controls, proximity to field applications could contribute significantly to a higher short-term exposure burden to nearby residents.

Pesticide exposures and risks are characterized for the communities around the air monitoring locations. However, the potential for exposures in other residential areas clearly exists. For example, census data indicate that > 185,000 people live in areas in California with a density of use of metam sodium (MITC) greater than the community air monitoring locations. More than 208,000 people live in areas where the density of use of methyl bromide exceeds use around recent air monitoring locations. These data suggest a potential for exposures and risks, similar to those calculated in this risk assessment, for hundreds of thousands of people in California.

Children's exposures require particular attention (*38*). Risks to children are uniformly higher than those of adults due to a greater inhalation rate-to-body weight ratio and other factors. Our report specifically assesses risks to children from a rarely evaluated exposure—inhalation of agricultural pesticides. This pathway is important because an increasing number of children live along the nation's agricultural–urban edge. For example, in California we estimated that > 53,000 children lived in census block groups where methyl bromide use density exceeded the use density near recent community air monitoring locations.

There are also other states that use these pesticides nearly as extensively as California. For example, 1997 crop use shows the following rank by pounds of active ingredient: metam sodium (MITC parent), 1 = California (13.7 million), 2 = Michigan (2.4 million), 3 = Florida (2.3 million); methyl bromide, 1 = California (14.5 million), 2 = Florida (11.3 million), 3 = Georgia (1.4 million pounds); 1,3-dichloropropene, 1 = North Carolina (10.8 million), 2 = Oregon (5.8 million), 3 = Washington (3.6 million), 7 = California (1.5 million); chlorpyrifos, 1 = California (2.4 million), 2 = Iowa (1.2 million), 3 = Illinois (1 million) (*73*). Community air measurements in California may be relevant with respect to other agricultural regions with similar crops and pesticide applications. However, California has the most restrictive pesticide permit conditions of any state, aimed largely

Table 8. Total California population in areas with pesticide use density greater than air monitoring areas.[a]

Pesticide	Pounds/mile^2 in monitoring area[b]	Child population (< 15 years old)	Total population
Methyl bromide	5,893	53,731	208,757
Metam sodium (MITC)	1,296	48,410	185,441
1,3-Dichloropropene	1,306	12,819	43,246
Chlorpyrifos	800	764	2,523

[a]Population estimates for California block groups using 1990 census data (*12*). [b]Pesticide use density based on PUR data for radii around air-monitoring sites: 3 mile radius (methyl bromide, MITC, and 1,3-dichloropropene); 1.5-mile radius (chlorpyrifos). Methyl bromide air monitoring location, "2000b"; 1,3-dichloropropene monitoring location, "2000a" (see Table 1). PUR data are from the year of air monitoring (MITC, chlorpyrifos) or 1999 proxy year (methyl bromide, 1,3-dichloropropene).

Table 9. Pesticide use in year and county of air monitoring, and 1991–1999 averages.

	Year of monitoring			1991–1999 avg use	
Pesticide, county	Year	Total pounds	Pounds/mile^2 Ag land[a]	Total pounds	Pounds/mile^2 Ag land[a]
Chloropicrin					
Monterey-SCz-SB[b]	1986	738,790	1,043	1,306,775	1,845
Chlorothalonil					
Ventura	1991[c]	45,134	198	72,216	317
Chlorpyrifos					
Tulare	1996	385,776	274	348,181	248
DEF					
Fresno	1987	371,725	158	346,623	147
Diazinon					
Fresno	1998	117,799	50	158,025	67
1,3-Dichloropropene					
Kern	1996	602,527	325	666,890[e]	360[e]
Kern	1999[d]	664,042	358	666,890[e]	360[e]
Merced	1989[f]	1,927,471	1,932	204,577[e]	205[e]
Monterey-SCz-SB[b]	1999[d]	570,996	806	408,511[e]	577[e]
EPTC					
Imperial	1996	152,960	165	145,894	157
Endosulfan					
Fresno	1996	75,400	32	94,314	40
Metam sodium (MITC)					
Kern	1993	1,028,869	555	2,800,896	1,511
Methidathion					
Tulare	1991	75,075	53	80,419	57
Methyl bromide					
Kern	1999[d]	788,293	425	1,564,439	844
Monterey-SCz-SB[b]	1986	1,308,103	1,846	2,955,187	4,171
Monterey-SCz-SB[b]	1999[d]	2,971,270	4,194	2,955,187	4,171
Molinate					
Colusa	1992	321,555	575	276,063	493
Naled (Dichlorvos)					
Tulare	1991	31,316	22	25,216	18
Propargite					
Fresno-Kings-Tulare	1999	626,606	130	784,388	163
Simazine					
Fresno	1998	182,634	78	147,568	63

Abbreviations: avg, average; Ag, agricultural.
[a]Source for agricultural (Ag) land (*69*). [b]SCz-SB Santa Cruz, San Benito counties (San Benito adjacent to air monitoring sites). [c]1991 proxy year for 1990 air monitoring year; pesticide use report data not validated for 1990. [d]1999 proxy year for 2000 air monitoring year; 2000 pesticide use report data not available at time of report. [e]Use averaged over 1996–1999 for 1,3-dichloropropene; use largely suspended in California 1990–1995. [f]1989 proxy year for 1990 air monitoring year; pesticide use report data not validated for 1990.

at reducing airborne emissions, particularly for fumigants (*74–77*). This may result in lower exposures and risks under California use conditions.

Metam sodium use has increased markedly nationwide, an estimated 7- to 12-fold since the late 1980s (*78*). Methyl bromide use is likely to decrease nationwide as other fumigants, including 1,3-dichloropropene and metam sodium, are adopted as substitutes in the mandated reduction under the Clean Air Act (*79*). Chlorpyrifos was the most widely used U.S. household pesticide before the 2000 decision by the U.S. EPA to eliminate most residential, school, and park uses (and cancel agricultural use on tomatoes and greatly reduce use on apples and grapes) to reduce exposures to children (*80*). Other agricultural uses of chlorpyrifos are less impacted by regulatory changes (*80*). The California air monitoring in 1996 for chlorpyrifos was in communities in citrus-growing areas. The chlorpyrifos concentrations detected in the California monitoring are several-fold higher than those found in urban areas (Table 1) (*81*), making it less likely that these concentrations were a result of residential use.

Notable uncertainties in this risk assessment occur in hazard identification, dose–response assessment, and exposure assessment. Stochastic analysis was used to characterize exposure variability. Among the distributions used, annual exposure frequency was the least characterized, relying on pesticide use data to establish a triangular distribution. Ambient air data were used in the risk assessment in the absence of indoor air data. Both higher and lower indoor air concentrations relative to ambient air have been historically reported for agricultural-use pesticides (*6–8*).

The risk assessment only considered inhalation exposures. Ingestion and dermal pathways are also likely exposure routes (*82–84*). Young children in particular, with a higher ingestion rate of fresh fruits and vegetables and higher contact rates with soil and housedust through hand-to-mouth activities, are at risk for cumulative pesticide exposures by such routes (*82*). There is also a large subpopulation at potentially higher risk: farmworker/farm children. An estimated 20% of 5 million U.S. farmworkers live or work in California (*85*). Increased exposures of children of farmworkers and farmers have been repeatedly documented, through occupational take-home exposures and other routes (*86–89*).

With respect to existing toxicologic data, there are important gaps in health reference levels specific to the inhalation route. There are also a number of pesticides for which no FQPA safety factor for children has been established. Several pesticides have noncancer or cancer health reference levels that vary between agencies and programs, resulting in a wider range in risk estimates. Uncertainties in hazard identification are also present. Emerging concerns, such as endocrine disruption and neurologic development, may not have been fully evaluated in toxicity testing (*90,91*). There is also a lack of toxicity data on exposures to multiple pesticides. Combined exposures to pesticides have been shown to cause effects not observed individually (*92–94*) and may potentiate toxicity in some pesticide combinations, for example, cholinesterase inhibitors (*95–97*).

Several pesticides found in the air of communities (Table 1) are organophosphate (OP) cholinesterase inhibitors, including chlorpyrifos, DEF, diazinon, dichlorvos, EPTC, and methidathion. Children may be exposed to multiple OPs, all sharing a common toxicity, through multiple routes. Exposure studies have shown OP pesticide accumulation on children's toys as a result of prolonged vaporization from other deposits (*98*), indoor transport from outdoor applications of OPs, with redistribution into indoor air and surfaces (*99*) and increased OP metabolites in children living near agricultural applications (*86,87*). Exposure to organophosphate pesticides may potentially impact neurodevelopment, growth, and respiratory health in children (*100*).

Pesticides with the highest risks in this risk assessment, MITC, methyl bromide, and 1,3-dichloropropene, impact some of these same target organs. Methyl bromide is a developmental, neurologic, and respiratory toxin. MITC and 1,3-dichloropropene are also respiratory toxins. The potential for exposure to more than one of these pesticides clearly exists. Methyl bromide and 1,3-dichloropropene were detected together in dual air monitoring. Several of the California communities selected for air monitoring for a pesticide were reselected in later studies because they were in the highest-use area for another pesticide. Toxicity, epidemiology, and exposure studies addressing likely combinations of these pesticides are needed.

Risk ranking effectively identifies the pesticides most in need of further scrutiny from inhalation exposure to agricultural pesticides. Vapor pressure is a significant predictor of this ranking of inhalation risks. Candidate pesticide air contaminants may be most readily identified using a ranking system that places greater weight on vapor pressure.

References and Notes

1. USGS. Pesticides in the Atmosphere - Distribution, Trends, and Governing Factors. Open-File Report 94-506. Sacramento, CA:U.S. Geological Survey, 1995.
2. Tiefenbacher JP. Mapping the pesticide driftscape: Theoretical patterns of the drift hazard. Geograph Environ Model 2(1):83–102 (1998).
3. Glotfelty DE, Seiber JN, Liljedahl LA. Pesticides in fog. Nature 325:602–605 (1987).
4. Baker LW, Fitzell DL, Seiber JN, Parker TR, Shibamoto T, Poore MW, Longley KE, Tomlin RP, Propper R, Duncan DW. Ambient air concentrations of pesticides in California. Environ Sci Technol 30(4):1365–1368 (1996).
5. Majewski MS, Foreman WT, Goolsby DA, Nakagaki N. Airborne pesticide residues along the Mississippi River. Environ Sci Technol 32:3689–3698 (1998).
6. CDPR. Seiber JN, Woodrow JE, Krieger RI, Dinoff T. Determination of ambient MITC residues in indoor and outdoor air in townships near fields treated with metam-sodium. DPN 50150-151, RN 170403. Sacramento, CA:California Department of Pesticide Regulation, 1999.
7. Camann DE, Geno PW, Harding HJ, Giardino NJ, Bond AE, Lewis RG, Akland GG. A pilot study of pesticides in indoor air in relation to agricultural applications. In: Indoor Air '93: Proceedings of 6th International Conference on Indoor Air Quality and Climate, Helsinki, Finland, 4–8 July 1993, Vol 2 (Seppanen O, ed). Helsinki:Finnish Society of Indoor Air Quality and Climate, 1993;207–212.
8. Teschke K, Chow Y, Bartlett K, Ross A, van Netten C. Spatial and temporal distribution of airborne *Bacillus thuringiensis* var. *kurstaki* during an aerial spray program for gypsy moth eradication. Environ Health Perspect 109:47–54 (2001).
9. Ames RG, Stratton JW. Acute health effects from community exposure to *N*-propyl mercaptan from an ethoprop (Mocap)-treated potato field in Siskiyou County, California. Arch Environ Health 46(4):213–217 (1991).
10. Goldman LR, Mengle D, Epstein DM, Fredson D, Kelly K, Jackson RJ. Acute symptoms in persons residing near a field treated with the soil fumigants methyl bromide and chloropicrin. West J Med 147:95–98 (1987).
11. Scarborough ME, Ames RG, Lipsett MJ, Jackson RJ. Acute health effects of community exposure to cotton defoliants. Arch Environ Health 44(6):355–360 (1989).
12. Gunier RB, Harnly ME, Reynolds P, Hertz A, Von Behren J. Agricultural pesticide use in California: pesticide prioritization, use densities, and population distribution for a childhood cancer study. Environ Health Perspect 109:1071–1078 (2001).
13. California Department of Pesticide Regulation. Pesticides for Evaluation as Candidate Toxic Air Contaminants. EH 96-01. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/eh96-01.pdf [cited 14 August 2001].
14. U.S. EPA. Section 112 Hazardous Air Pollutants. Available: http://www.epa.gov/ttn/atw/188polls.html [cited 14 August 2001].
15. CARB. ARB Monitoring of Chloropicrin. Sacramento, CA:California Air Resources Board, 1987.
16. CARB. ARB 1990 Monitoring of Telone. Sacramento, CA:California Air Resources Board, 1991.
17. CARB. Report for 1996 Ambient Monitoring of Telone in Kern County. Report No. C96-045. Sacramento, CA:California Air Resources Board, 1997.
18. CARB. Ambient Air Monitoring for Methyl Bromide and 1,3-Dichloropropene in Monterey and Santa Cruz Counties – Fall 2000. Sacramento, CA: California Air Resources Board, 2001. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/tacpdfs/mebr2000.pdf [cited 20 August 2002].
19. CARB. Ambient Air Monitoring for Methyl Bromide and 1,3-Dichloropropene in Kern County – Summer 2000. Sacramento, CA: California Air Resources Board, 2000. Available: http://www.cdpr.ca.govdocs/empm/pubs/tac/tacpdfs/mthdic13.pdf [cited 20 August 2002].
20. CARB. ARB Monitoring of Methyl Bromide, 1986. Sacramento, CA: California Air Resources Board, 1987. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/tacpdfs/mebr-86.pdf [cited 20 August 2002].
21. CARB. Ambient Air Monitoring for MITC in Kern County during Summer 1993. Sacramento, CA: California Air Resources Board, 1994. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/tacpdfs/mitckern.pdf [cited 20 August 2002].
22. CARB. Ambient Air Monitoring for Captan in Kern County during Spring, 1993. Report No. C89-041. Sacramento, CA:California Air Resources Board, 1994.
23. CARB. ARB Monitoring of Chlorothalonil. Sacramento, CA:California Air Resources Board, 1991.
24. CARB. Report for the Air Monitoring of EPTC in Merced County (Application) and in Imperial County (Ambient). Sacramento, CA: California Air Resources Board, 1998. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/eptc.htm [cited 20 August 2002].
25. CARB. Report for the Application and Ambient Air Monitoring of Linuron in Kern County. Sacramento,

CA:California Air Resources Board, 1999. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/linuron.htm [cited 14 August 2001].
26. CARB. Molinate Ambient Air Monitoring in Colusa County, May 1992. Sacramento, CA: California Air Resources Board, 1993. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/molinate.htm [cited 14 August 2001].
27. CARB. Report for the Application (Tulare County) and Ambient (Fresno County) Air Monitoring of Simazine. Sacramento, CA: California Air Resources Board, 1999. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/simazine.htm [cited 14 August 2001].
28. CARB. ARB Monitoring of DEF. Sacramento, CA:California Air Resources Board, 1988.
29. CARB. Report for the Application and Ambient Air Monitoring of Aldicarb. Sacramento, CA:California Air Resources Board, 1998. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/aldicarb.htm [cited 14 August 2001].
30. CARB. Report for the Application and Ambient Air Monitoring of Chlorpyrifos (and the Oxon Analogue) in Tulare County during Spring/Summer, 1996. Sacramento, CA:California Air Resources Board, 1998. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/chlrpfs.htm [cited 14 August 2001].
31. CARB. Report for the Application (Kings County) and Ambient (Fresno County) Air Monitoring of Diazinon during Winter, 1998. Sacramento, CA:California Air Resources Board, 1998. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/diazinon.htm [cited 14 August 2001].
32. CARB. Airborne Concentrations of Naled and Dichlorvos in Central Tulare County from Sampling Conducted in May and June 1991. Sacramento, CA:California Air Resources Board, 1993.
33. CARB. Report for the Air Monitoring of Endosulfan in Fresno County (Ambient) and in San Joaquin County (Application). Sacramento, CA:California Air Resources Board, 1998. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/endoslfn.htm [cited 14 August 2001].
34. CARB. Report for the Application and Ambient Air Monitoring of Fenamiphos in Fresno County. Sacramento, CA: California Air Resources Board, 1998. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/fenamphs.htm [cited 14 August 2001].
35. CARB. Airborne Concentrations of Methidathion and Methidaoxon in Central Tulare County from Sampling Conducted in June and July 1991. Sacramento, CA:California Air Resources Board, 1993.
36. CARB. Report for the Application (Del Norte County) and Ambient (Fresno County) Air Monitoring of Phorate. Sacramento, CA:California Air Resources Board, 1998. Available: http://www.cdpr.ca.gov/docs/empm/pubs/tac/phorate.htm [cited 14 August 2001].
37. CARB. Report for the 1999 Application and Ambient Air Monitoring for Propargite and Bifenthrin in Fresno and Kings Counties. Sacramento, CA:California Air Resources Board, 2001.
38. U.S. EPA. Food Quality Protection Act (FQPA) of 1996. Available: http://www.epa.gov/oppfead1/fqpa/ [cited 14 August 2001].
39. U.S. EPA. Risk Assessment Guidance for Superfund Human Health Evaluation Manual. EPA/540/1-89/002. Washington, DC:U.S. Environmental Protection Agency, 1989.
40. ATSDR. Public Health Assessment Guidance Manual. Atlanta, GA:Agency for Toxic Substances and Disease Registry, 1992.
41. CDPR. Pesticide Use Report Data 1986-1999. Sacramento, CA:California Department of Pesticide Regulation, 2000.
42. DI. Crystal Ball, 2000. Denver, CO:Decisioneering Inc., 2000.
43. Office of Environmental Health Hazard Assessment. Air Toxics Hot Spots Program Risk Assessment Guidelines. Part IV. Exposure Assessment and Stochastic Analysis Technical Support Document. Available: http://www.oehha.ca.gov/air/hot_spots/finalStoc.html#download [cited 20 August 2002].
44. U.S. EPA. Exposure Factors Handbook, Vol. III, Activity Factors. EPA/600/P-95/002Fc. Washington, DC: U.S. Environmental Protection Agency, 1997.
45. U.S. EPA. Integrated Risk Information System. Available: http://www.epa.gov/iriswebp/iris/index.html [cited 30 August 2001].
46. U.S. EPA. Clorothalonil Reregistration Eligibility Decision (RED). Available: http://www.epa.gov/pesticides/reregistration/status.htm [cited 30 August 2001].
47. U.S. EPA. Clorpyrifos Interim Reregistration Eligibility Decision (IRED). Available: http://www.epa.gov/pesticides/reregistration/status.htm [cited 30 August 2001].
48. U.S. EPA. Tribufos Preliminary Risk Assessment. Available: http://www.epa.gov/oppsrrd1/op/tribufos.htm [cited 30 August 2001].
49. U.S. EPA. Diazinon Revised Risk Assessment. Available: http://www.epa.gov/oppsrrd1/op/diazinon.htm [cited 30 August 2001].
50. U.S. EPA. Office of Pesticide Programs List of Chemicals Evaluated for Carcinogenic Potential. Washington, DC: U.S. Environmental Protection Agency, 2000.
51. U.S. EPA. Dichlorvos Preliminary Risk Assessment. Available: http://www.epa.gov/oppsrrd1/op/ddvp.htm [cited 30 August 2001].
52. U.S. EPA. Health Effects Assessment Summary Tables FY 1997 Update. EPA-540-R-97-036. Washington, DC: U.S. Environmental Protection Agency, 1997.
53. U.S. EPA. EPTC Reregistration Eligibility Decision (RED). Available: http://www.epa.gov/pesticides/reregistration/status.htm [cited 30 August 2001].
54. U.S. EPA. Methidathion Revised Health Risk Assessment. Available: http://www.epa.gov/oppsrrd1/op/methidathion.htm [cited 30 August 2001].
55. California Department of Pesticide Regulation. Evaluation of S,S,S-Tributyl phosphorotrithioate (DEF) as a Toxic Air Contaminant. Part C: Health Assessment. Available: http://www.cdpr.ca.gov/docs/empm/pubs/def/defaircn.htm [cited 30 August, 2001].
56. CDPR. Risk Assessment of 1,3-Dichloropropene. Sacramento, CA:California Department of Pesticide Regulation, 1997.
57. CDPR. Dichlorvos (DDVP), Second Addendum to Risk Characterization Document. Sacramento, CA:California Department of Pesticide Regulation, 1998.
58. California Department of Pesticide Regulation. Toxicology Data Review Summaries. Available: http://www.cdpr.ca.gov/docs/toxsums/toxsumlist.htm [cited 30 August 2001].
59. CDPR. EPTC (S-Ethyl-dipropylthiocarbamate) Risk Characterization Document. Sacramento, CA:California Department of Pesticide Regulation, 1995.
60. CDPR. Methidathion (Supracide) Dietary and Drinking Water Risk Assessment. Sacramento, CA:California Department of Pesticide Regulation, 2000.
61. California Department of Pesticide Regulation. Methyl Bromide Risk Characterization Document for Inhalation Exposures, Draft, 1999; NAS Review of Methyl Bromide Risk Characterization Document. Available: http://www.cdpr.ca.gov/docs/dprdocs/methbrom/mb_main.htm [cited 30 August 2001].
62. California Department of Pesticide Regulation. Evaluation of Methyl Isothiocyanate (MITC) as a Toxic Air Contaminant, 2002. Available: http://www.cdpr.ca.gov/docsempm/pubs/tac/mitc.htm [cited 4 April 2002].
63. CDPR. Molinate Risk Characterization Document. Sacramento, CA:California Department of Pesticide Regulation, 1996.
64. California Department of Pesticide Regulation. Lompoc Pesticide Air Fumigant Sampling and Analysis Plan, Appendix B, Fumigant Screening Levels. Available: http://www.cdpr.ca.gov/docs/dprdocs/lompoc/apends99.htm [cited 30 August 2001].
65. Office of Environmental Health Hazard Assessment. Acute RELs. Available: http://www.oehha.org/air/acute_rels/index.html [cited 30 August 2001].
66. Alexeeff GV, Shusterman DJ, Howd RA, Jackson RJ. Dose-response assessment of airborne methyl isothiocyanate (MITC) following a metam sodium spill. Risk Anal 14(2):191–198 (1994).
67. Agency for Toxic Substances and Disease Registry. Minimal Risk Levels (MRLs) for Hazardous Substances. Available: http://www.atsdr.cdc.gov/mrls.html [cited 30 August 2001].
68. Agency for Toxic Substances and Disease Registry. Toxicological Profiles. Available: http://www.atsdr.cdc.gov/toxpro2.html [cited 30 August 2001].
69. USGS. Geographical Analysis Program (GAP) Analysis of Mainland California: An Interactive Atlas of Terrestrial Biodiversity and Land Management (CD-ROM). Washington, DC:U.S. Geological Survey, Biological Resources Division, 1999.
70. U.S. Department of Agriculture. Agricultural Research Service (ARS) Pesticide Properties Database. Available: http://wizard.arsusda.gov/acsl/ppdb.html [cited 30 August 2001].
71. Woodrow JE, Seiber JN, Baker LW. Correlation techniques for estimating pesticide volatilization flux and downwind concentrations. Environ Sci Technol 31(2):523–529 (1997).
72. Woodruff TJ, Axelrad DA, Caldwell J, Morello-Frosch R, Rosenbaum A. Public health implications of 1990 air toxics concentrations across the United States. Environ Health Perspect 106:245–251 (1998).
73. National Center for Food and Agricultural Policy. National Pesticide Use Database. Available: http://www.ncfap.org/ncfap/index.html [cited 30 August 2001].
74. California Department of Pesticide Regulation. Review of Restrictions on the Use of Methyl Bromide. Available: http://www.cdpr.ca.gov/docs/dprdocs/methbrom/mb_main.htm [cited 24 August 2000].
75. California Department of Pesticide Regulation. Suggested Permit Conditions for Using 1,3-Dichloropropene Pesticides (Fumigant). Available: http://www.cdpr.ca.gov/docs/enfcmpli/penfltrs/penf2001/2001040.pdf [cited 20 August 2002].
76. California Department of Pesticide Regulation. Permit Conditions for Applications of Metam-Sodium and Potassium N-Methyldithiocarbamate (Metam-Potassium) Products. Available: http://www.cdpr.ca.gov/docs/enfcmpli/penfltrs/penf2000/2000044.pdf [cited 20 August 2002].
77. MST. Metam Sodium Taskforce Publications. Guidelines for All Application Methods for Metam-Sodium in California. Available: http://www.metampsc.com/tib.pdf [cited 20 August 2002].
78. U.S. EPA. Pesticides Industry Sales and Usage, 1996 and 1997 Market Estimates. Available: http://www.epa.gov/oppbead1/pestsales/ [cited 23 August 2001].
79. U.S. EPA. Methyl Bromide Phase Out Web Site. Available: http://www.epa.gov/ozone/mbr/ [cited 24 August 2001].
80. U.S. EPA. Documents for Chlorpyrifos. Available: http://www.epa.gov/pesticides/op/chlorpyrifos.htm [cited 26 February 2002].
81. Whitmore RW, Immerman FW, Camann DE, Bond AE, Lewis RG, Schaum JL. Non-occupational exposures to pesticides for residents of two U.S. cities. Arch Environ Contam Toxicol 26:47–59 (1994).
82. National Resource Council. Pesticides in the Diets of Infants and Children. Washington, DC:National Academy Press, 1993.
83. Bradman MA, Harnly ME, Draper W, Seidel S, Teran S, Wakeham D, Neutra R. Pesticide exposures to children from California's Central Valley: results of a pilot study. J Expo Anal Environ Epidemiol 7(2):217–234 (1997).
84. Fenske RA, Black KG, Elkner KP, Lee CL, Methner MM, Soto R. Potential exposure and health risks of infants following indoor residential pesticide applications. Am J Public Health 80(6):689–693 (1990).
85. Mobed K, Gold EB, Schenker MB. Occupational health problems among migrant and seasonal farm workers. West J Med 157(3):367–373 (1992).
86. Loewenherz C, Fenske RA, Simcox NJ, Bellamy G, Kalman D. Biological monitoring of organophosphorus pesticide exposure among children of agricultural workers in central Washington State. Environ Health Perspect 105:1344–1353 (1997).
87. 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 84:290–302 (2000).
88. McCauley LA, Lasarev MR, Higgins G, Rothlein J, Muniz J, Ebbert C, Phillips J. Work characteristics and pesticide exposure among migrant agricultural families: a community-based research approach. Environ Health Perspect 109:533–538 (2001).
89. Simcox NJ, Fenske RA, Wolz SA, Lee I, Kalman DA. Pesticides in household dust and soil: exposure pathways for children of agricultural families. Environ Health Perspect 103:1126–1134 (1995).
90. Cooper RL, Goldman JM, Stoker TE. Neuroendocrine and reproductive effects of contemporary-use pesticides. Toxicol Ind Health 15:26–36 (1999).
91. U.S. EPA. Special Report on Environmental Endocrine Disruption: An Effects Assessment and Analysis. EPA/630/R-96/012 Washington, DC:U.S. Environmental Protection Agency, 1997.
92. Porter WP, Jaeger JW, Carlson IH. Endocrine, immune, and behavioral effects of aldicarb (carbamate), atrazine (triazine) and nitrate (fertilizer) mixtures at groundwater concentrations. Toxicol Ind Health 15:133–150 (1999).
93. Heindel JJ, Chapin RE, Gulati DK, George JD, Price CJ,

Marr MC, Myers CB, Barnes LH, Fail PA, Grizzle TB, et al. Assessment of the reproductive and developmental toxicity of pesticide/fertilizer mixtures based on confirmed pesticide contamination in California and Iowa groundwater. Fundam Appl Toxicol 22:605–621 (1994).

94. Boyd CA, Weiler MH, Porter WP. Behavioral and neurochemical changes associated with chronic exposure to low-level concentration of pesticide mixtures. J Toxicol Environ Health 30:209–221 (1990).
95. Hodgson E. Induction and inhibition of pesticide-metabolizing enzymes: roles in synergism of pesticides and pesticide action. Toxicol Ind Health 15:6–11 (1999).
96. Abiola FA, Sere A, Sawadogo JG, Diatta F, Ly M. Cholinesterase depression among Senegalese crop protection workers exposed to organophosphorous pesticides. Bull Environ Contam Toxicol 41(4):483–488 (1988).
97. Frawley JP, Fuyat HN, Hagan EC, Blake JR, Fitzhugh OG. Marked potentiation in mammalian toxicity from simultaneous administration of two anticholinesterase compounds. J Pharmacol Exp Therap 121:96–106 (1957).
98. Gurunathan S, Robson M, Freeman N, Buckley B, Roy A, Meyer R, Bukowski J, Lioy PJ. Accumulation of chlorpyrifos on residential surfaces and toys accessible to children. Environ Health Perspect 106:9–16 (1998).
99. Lewis RG, Fortune CR, Blanchard FT, Camann DE. Movement and deposition of two organophosphorus pesticides within a residence after interior and exterior applications. J Air Waste Manage Assoc 51:339–351 (2001).
100. Eskanazi B, Bradman A, Castorina R. Exposures of children to organophosphate pesticides and their potential adverse health effects. Environ Health Perspect 107(suppl 3):409–419 (1999).
101. U.S. EPA. Revised Occupational and Residential Exposure Assessment and Recommendations for the Reregistration Eligibility Decision Document for EPTC. D258688. Washington, DC:U.S. Environmental Protection Agency, 1999.