Pesticide-Related Health Problems and Farmworkers

by Marion Moses, MD

Farmworkers labor under some of the worst conditions of any group of workers in the United States. Farm work not done by farm owners and their families is largely performed by ethnic minorities, primarily Hispanics of Mexican origin, who may comprise 80% to 90% of the work force (Martin, 1985). American blacks comprise the next largest group, with a smaller number of Haitians, Filipinos, Vietnamese, Laotians, Koreans, Jamaicans, and others. The U.S. Department of Agriculture (USDA) estimates an annual agricultural work force of approximately two million hired workers and three million farm owners and their families (unpaid). California, Texas, and Florida are the leading states in number of hired farmworkers (USDA, 1986).

THE WORKERS

Farmworkers are excluded, completely or partially, from federal laws that protect other workers including: the National Labor Relations Act (which guarantees the right to join a union and bargain collectively); the Fair Labor Standards Act (which governs minimum wage and child labor); and the Occupational Safety and Health Act (which governs standards of health and safety in the workplace). Furthermore, most migrant farmworkers are excluded from state laws such as workers' compensation and unemployment insurance.

THE WORKPLACE

The USDA defines a farm as a place that sells or could sell $1000 of agricultural products during the year. There are 2,214,429 farms in the U.S., and one billion acres of cropland (not including pasture and rangeland). The average farm size is 455 acres; however, 14% of the farms with the highest income control 50% of the land (USDA, 1986).

Farmworkers labor under some of the worst conditions of any group of workers in the United States.

The largest amount of acreage in the U.S. is in crops such as corn, wheat, soybeans, and cotton, in which cultivating and harvesting is almost completely mechanized. Labor-intensive crops, primarily fruits and vegetables still require large numbers of workers for hand-cultivating and harvesting. It is the picking of these crops, which Edward R. Murrow called "The Harvest of Shame," that is the major work of the migrant and seasonal farmworker.

The agricultural workplace poses many hazards related to the use of tractors, harvesters, ladders, irrigation and other equipment and machinery. Other hazards include heat stress, bee stings, snake bites, dusts, and airborne allergens. This review will focus on an additional and more insidious hazard faced by agricultural workers—pesticide exposure.

PESTICIDE USE IN AGRICULTURE

Almost all commercial crops in the U.S. are heavily and repeatedly sprayed with chemical pesticides, the majority of which are toxic materials that pose both acute and chronic health problems to exposed workers.

The largest single user of pesticides in the U.S. is agriculture, which, in 1985, accounted for 77% of nationwide usage of 1.08 billion pounds (does not include wood preservatives); and 78% of expenditures of $4.6 billion (U.S. Environmental Protection Agency, 1986). In California, where approximately 250 million pounds of pesticides are used annually, 92% is used in agriculture.

Pesticides must be registered with the Environmental Protection Agency (EPA) before they can be legally sold or used in the U.S. Of the 1,200 pesticide active ingredients currently registered, about half are used in agriculture. These pesticide active ingredients are combined with other so-called "inert" ingredients into approximately 35,000 different commercial products or formulations.

Inert ingredients, which may be as toxic or even more toxic than the pesticide itself, are neither required to be tested for acute and chronic health effects nor listed by name on the pesticide label. Inert ingredients may comprise 90% or more of a registered pesticide product, but due to "trade secret" provisions of the Federal Insecticide Fungicide Rodenticide Act (FIFRA), the identity of these ingredients cannot be released to the public (even in cases of serious poisoning) by state or federal regulatory agencies.

The pattern of pesticide use in

agriculture has changed greatly, with a 175% increase in herbicide use from 25 years ago; herbicides now represent two thirds of all usage. Widely used herbicides include: alachlor (Lasso), Atrazine, 2,4-D, Paraquat (Gramoxone), Simazine (Princep), and trifluralin (Treflan).

The use of the less persistent but more acutely toxic organophosphate and N-methyl carbamate insecticides has increased with the banning or restriction of the environmentally persistent chlorinated hydrocarbons (DDT, Aldrin, Dieldrin, Lindane, Chlordane, Heptachlor, and Toxaphene). Widely used organophosphates include chlorpyrifos (Dursban, Lorsban), diazinon (Spectracide), malathion, parathion, Phosdrin, methamidophos (Monitor), and DDVP (Vapona). Widely used N-methyl carbamates include aldicarb (Temik), carbaryl (Sevin), carbofuran (Furadan), and methomyl (Lannate, Nudrin).

Highly toxic fumigants such as methyl bromide and D-D (Telone or 1,2-dichloropropane/1,3-dichloropropene) are being used in increasingly larger amounts as replacements for DBCP, banned in 1979, and ethylene dibromide, banned in 1984.

While fungicides represent a small percentage of total volume of pesticide use, they are important because many are carcinogens and/or teratogens. They are used extensively on fruits and vegetables and may persist as residues in the marketed fresh or processed product (National Academy of Science, 1987). Widely used fungicides include Benomyl, Captan, chlorothalonil (Bravo, Daconil), Maneb, and Mancozeb (Dithane).

ENVIRONMENTAL PESTICIDE EXPOSURE

Pesticides are among the few toxic materials deliberately added to the environment. Their use in agriculture is a major source of involuntary exposure of the general public to carcinogens due to contamination of fresh and processed food by persistent pesticide residues, most of which cannot be washed off or degraded by cooking.

The dominance of chemical pest control in agriculture in the U.S. began in the middle and late 1940s. By the middle 1950s, evidence of widespread contamination of fish, birds, and wildlife by pesticides was mounting. EPA and other monitoring agencies throughout the world have shown that pesticide contamination is global, including snow caps of the highest mountains and core samples from the arctic ice pack. A recent California study shows that pesticides concentrate in fog (Glotfelty, 1987).

The primary route of worker exposure to the majority of pesticides is the skin, and not, as commonly believed, the respiratory system.

Humans are contaminated with pesticides, especially fatty tissue. Breast milk is contaminated with a variety of pesticides (Rogan, 1980; Savage, 1981), with high levels in the U.S. found in women in the rural south (Barnett, 1979). The highest have been reported in farmworker women in cotton growing areas in Central America (deCampos, 1979). Many pesticides cross the placenta, and newborn infants are already contaminated at birth.

A problem inherent in current pesticide application technology is drift, or dispersal of the pesticide away from the site of application. Only 10% to 15% of applied pesticides actually reach the target pest, with the remaining 85% to 90% dispersed off-target to air, soil, and water through drift, runoff, volatilization, off-gassing, etc. (Matthews, 1982).

Pesticides can drift as far as 50 miles from the site of application, depending on particle size and wind conditions. Pesticide residues can be persistent soil contaminants and a continuing source of contamination from runoff as well as dust. A dust storm was found to have carried pesticides from Texas to Ohio (Akesson, undated). Significant concentrations of almost all pesticides applied aerially or by ground rig sprayers can drift up to a mile or more from the site of application, even under the best wind conditions (Akesson, 1964; Matthews, 1982).

Communities that abut agricultural land are at risk from pesticide drift, and while some episodes of illness have been reported (Goldman, 1987; Ratner, 1986), the extent of the problem is essentially unknown and undocumented. In 1987 in California, pesticide drift caused three major evacuations of community residents—two from Guthion being used in peach orchards and one from methyl bromide off-gassing from a gladiola field.

Agricultural pesticide use is the major cause of non-point source contamination of groundwater (a non-point source means there is not a single identifiable place such as a toxic dump, factory, sewer line, etc. causing the contamination, see Table 1). Nationwide, 50% of the drinking water supply is from groundwater and in rural areas, 90% or more.

OCCUPATIONAL PESTICIDE EXPOSURE IN AGRICULTURE

The primary route of worker exposure to the majority of pesticides is the skin, and not, as commonly believed, the respiratory system. Fumigants, which are in the form of gases, are a notable exception. This accounts, in part, for their greater toxicity. However, the skin is a source of absorption for them as well (van den Oever, 1982, 1984). Persistence of pesticides on the skin for many months after the last known exposure has been shown (Kazen, 1974).

Workers who mix, load, and apply pesticides are exposed to the concen-

trated form of the pesticide. A large number of workers are exposed from the use of ground rig spray equipment. A common method uses tractor-mounted or drawn tanks with attached booms and nozzles that vary in number and size. Most are open-cab with minimal protection for the applicator. Air blast sprayers, used in nut and fruit groves and orchards, are among the most highly polluting equipment.

Nationwide, the percentage of pesticides applied by fixed-wing aircraft or helicopters (called crop dusters) is unknown. In California, it is estimated that more than half are applied by such methods. The pilots are less at risk of poisoning than the ground crews who mix and load the pesticide, and especially the flaggers who direct the crop duster from the ground.

Chemigation, or putting the pesticide in the irrigation water, is increasing. Pesticides are also incorporated into soil (usually granular formulations), or as in the case of fumigants, injected into the soil and then tarped with plastic sheeting to minimize loss from off-gassing. Animals are also treated with pesticides, externally through the use of "dips," or in their feed. Residues of pesticides from such use can then contaminate the meat, milk, eggs, gelatin, and other animal products.

Fieldworkers who harvest and cultivate crops are exposed to residues of pesticides, primarily on leafy surfaces (dislodgeable residues), but also on the crop itself or in the soil or duff (decaying plant and organic material that collects under vines, trees, etc.). Farmworkers are also exposed to pesticides by crop dusting aircraft or ground rig sprayers applying pesticides to adjacent fields, often the very field in which they are working.

ACUTE HEALTH EFFECTS OF PESTICIDES

Acute health effects of pesticide exposure range from eye and upper respiratory tract irritation and contact dermatitis to systemic poisoning, which can lead to death.

The number of workers in the United States affected by pesticides is unknown, although estimated at 300,000 a year (Wasserstrom, 1985). A Nebraska study of emergency room visits and hospitalizations for pesticide-related illness, conducted during the 1984-85 crop season, found an annual incidence of 1.35 cases per 10,000 population.

The great majority of workers do not know the names of pesticides to which they are exposed or the acute and chronic risks to their health.

Organophosphates were responsible for 25% of the incidents and anhydrous ammonia for 33% (Rettig, 1987).

Only the state of California requires mandatory reporting of pesticide-related illness, with 1,211 cases in 1986 (California Department of Food and Agriculture, 1987). However, the California system is based on doctor reporting through the workers' compensation system. Many affected workers never see a doctor, are not properly diagnosed or are unaware of their rights under the law (Kahn, 1976; Wasserstrom, 1985). The most frequently mistaken diagnoses in workers with pesticide poisoning are flu and gastroenteritis.

The organophosphate pesticides, similar to nerve gas in their effects on the nervous system (inhibition of the enzyme cholinesterase), are the most toxic, and have been responsible for the great majority of systemic poisonings and deaths in agricultural workers. Other highly toxic pesticides that have caused serious illness and death include the dinitrophenol class of chemicals and the fumigants. The large body of literature on the diagnosis and management of acute pesticide poisoning will not be discussed in detail in this review (Hayes, 1982; Namba, 1971; Morgan, 1982; Tafuri, 1987).

Many poisonings of entire crews of farmworkers from skin absorption of pesticide residues on crops they were picking have been documented (Midtling, 1984; Peoples, 1978; Saunders, 1987). Most are from organophosphate pesticides, especially Parathion.

To prevent these poisonings, California set quarantine times in 1972, or "reentry intervals" for some pesticides—making it illegal for workers to enter a field for a specified amount of time after it had been sprayed. California is the only state to have its own reentry intervals, which are much longer and cover more crops and pesticides than the weaker EPA standards promulgated later (see Table 2). If no specific reentry intervals have been set, the fields may be reentered legally (but not necessarily safely) "when dusts have settled and sprays have dried."

These intervals do not always protect workers from acute illness, and there have been poisonings even when the legal reentry time was observed (Saunders, 1987). The author investigated the following situation, which illustrates the failure to protect workers: A 32-year-old worker who was sent into a tomato field that had been sprayed one hour before with the organophosphate, Monitor (methamidophos), died six hours later. Reentry intervals do not address the problem of worker exposures to residues of carcinogenic pesticides or pesticides with other potential chronic effects.

EFFECTS OF PESTICIDES ON THE SKIN

More than 40% of all reported occupational diseases in the U.S. are disorders of the skin. The actual incidence is estimated to be ten to 50 times higher than the reported incidence of 1.5 cases/1,000.

TABLE 1

Oncogenic and Suspect Pesticides with Classification Code* (as of April 1988) **Based on EPA Data**

Pesticide	Classification*	Pesticide	Classification*
Acephate (Orthene)	C	CPA	NA
Acetochlor	B2	Cypermethrin	C
Aciflurofen (Tackle/Blazer)	B2	2,4-D	C
Alachlor	B2	Dalapon	NA
Aliette	C	Daminozide (Alar)	B2
Amdro	B2	DBCP	B2
Amitraz	C	DDT, DDE, DDD	B2
Amitrole	B2	DDVP	B2
Apollo (Clofentezine)	C	Diallate	NA
Assert	D	Diclofop-methyl (Hoelon)	NA
Assure	C	Dicofol	C/B2
Asulam	C	p-Dichlorobenzene	C/B2
Atrazine	C	Dieldrin	B2
Barban (Carbyne)	NA	Dimethoate	NA
Baygon (Propoxur)	B2	Dinoseb	C
Baytan (Triadimenol)	C	2,4-DP Acid	NA
Benomyl	C	Ethalfluralin	NA
Biphenox (Mowdown)	NA	Ethylene dibromide	B2
Biphenthrin	C	Ethylene thiourea (ETU)	B2
Bromacil	NA	Fenarimol	D/E
Bromoxynil	C	Fluridone	E
Cadmium	B1	Folpet	B2
Captafol	B2	Fomesafen	C
Captan	B2	Formaldehyde vapor	NA
CDEC	NA	Furmecyclox	B2
Chloramben	NA	Galben	NA
Chlordane	B2	Gardona	C
Chlordimeform	B2	Glyphosate (Roundup)	C
Chlorobenzilate	NA	Guthion	D
Chlorothalonil	B2	Haloxyfop-methyl (Verdict)	C
4-chloro-o-toluidine	NA	Harvade	C

* *Classification Code*
A—Human Carcinogen.
B—Probable Human Carcinogen.
B1—Sufficient evidence of carcinogenicity from animal studies with limited evidence from epidemiologic studies.
B2—Sufficient evidence of carcinogenicity from animal studies, with inadequate or no epidemiologic data.

Workers in agriculture are at a four times greater risk of skin disease than workers in other industries. Most pesticide-related skin problems are primary irritant or contact dermatitis. However, pesticides can also cause allergic contact dermatitis, which can become a chronic problem—in some cases the workers can be permanently disabled since they cannot tolerate even minute exposures to the pesticide. Often the condition is aggravated by sunlight, adding to the disability.

Pesticides shown to be sensitizers, ie, cause allergic contact dermatitis include: alachlor (Lasso), Benomyl (Benlate), Botran, Captan, Captafol (Difolatan), Dazomet, Dyrene (Anilazine), malathion, Maneb, Mancozeb, Naled (Dibrom), PCNB (Pentachloronitrobenzene), Propachlor, Pyrethrum, Rotenone, Thiram, Zineb, and Ziram, among others (Adams, 1983).

A major difficulty in making the diagnosis of allergic contact dermatitis from pesticide exposure in agricultural workers is determining if the dermatitis is from the pesticide or the crop itself. Crops that can cause allergic dermatitis include: artichokes, asparagus, brussel sprouts, cabbage, chicory, chives, citrus, garlic, leeks, onions, and pineapple, among others. Patch testing is neces-

Pesticide	Classification*	Pesticide	Classification*
HCB (cont. with PCNB)	B2	Permethrin	NA
Heptachlor	B2	PHMB	NA
Hoelon	NA	Picloram	NA
Imidan (Phosmet)	C	Primicarb	NA
Isoxaben	C	Prochloraz	C
Kerb (Pronamide)	C	Profluralin	NA
Lactofen	B2	Propazine	C
Larvadex (Cyromazine)	NA	Propioconazol (Tilt)	C
Lindane	B/C	Pydrin	NA
Linuron	C	Rabon	NA
Maleic hydrozide	NA	Resmethrin	NA
Mancozeb	NA	Ronilan	E
Maneb	NA	Rotenone	pending
MBC	NA	Savey	C/B2
Metalaxyl	E	Sutan	NA
Methane arsenic acid	NA	Telone II	B2
Methidathion	C	Terbuthylazine	NA
Metolachlor	pending (C)	Terbutryn	C
Methoxychlor	NA	Tetrachloroethylene	A
Metronidazole	NA	Toxaphene	NA
Mirex	NA	1,1,2-Trichloroethane	NA
Monuron	NA	Trichlorfon	NA
Nemacur	NA	Tridiphane	NA
Nitrofen (TOK)	NA	Trifluralin	C
Norflurazon	NA	UDMH (Daminozide metab.)	B2
OPP	NA	Vel	NA
Oryzalin (Surflan)	C	Vinylidene chloride	NA
Oxadiazon (Ronstar)	B2	Zineb	NA
Paraquat	C		
Parathion	C		
PCNB	D		

C—Possible Human Carcinogen. Limited evidence of carcinogenicity in the absence of human data.
D—Not classifiable as to human carcinogenicity. Inadequate or no human and animal data for carcinogenicity.
E—Evidence of noncarcinogenicity for humans. No evidence of carcinogenicity in at least two animal species in adequate studies, based on available evidence and does not mean is not a carcinogen under any circumstances.
NA—Not available or not provided.

sary to identify the allergen and confirm the diagnosis.

CHRONIC EFFECTS OF PESTICIDES

Little is known about the extent or magnitude of chronic health problems related to occupational exposure to pesticides because appropriate studies have not been done. Nor is the maintenance of records of pesticide usage that would document exposures required by current law. Most workers are exposed to many different pesticides (and "inert" ingredients) over a working lifetime and have many different employers, often for only short periods of time. The great majority of workers do not know the names of the pesticides to which they are exposed or the acute and chronic risks to their health.

Lack of record-keeping and the failure to document exposures and illness are reasons for little worker population-based scientific data on the extent of chronic health problems in farmworkers from pesticide exposures. In this regard, the almost universal response of the agricultural industry to concerns of farmworker health effects from chronic, low-level exposure to pesticides is: if there were a problem we would know about it.

Another problem in documenting chronic and delayed health effects from farmworkers' exposure to pesticides is the long period of clinical latency for some of the effects, especially cancer. The time from last exposure to development of disease may range from 15 to 30 years.

A large percentage of the pesticides widely used in agriculture are animal carcinogens and/or teratogens (see Tables 1 and 3). Enormous deficiencies in the toxicology submitted to EPA by the agrichemical companies on the acute and chronic toxicity of their products have been reported (Begley, Scandal in the testing lab. *Newsweek*, May 30:83, 1983). These data gaps and their implications for the safety of workers, consumers and the environment have been the subject of many reports (National Academy of Science, 1987; Mott, 1984; U.S. General Accounting Office, 1980, 1986a, 1986b, 1986c).

Based on what is known from laboratory animal studies and from the few human studies, chronic effects of concern in pesticide-exposed populations include cancer, birth defects, neurobehavioral deficits, neuropsychological changes, and reproduction and fertility problems. Such chronic effects may occur with no indication of any acute health effects, even with long-term exposure over a working lifetime. Chronic effects can occur though exposure to the pesticide ceased many years before.

PESTICIDES AND CANCER IN HUMANS

Several of the early studies of cancer in humans exposed to pesticides were done at pesticide manufacturing plants and reported no evidence of increased risk of cancer. Such early reports were used to conclude that pesticides could be used safely and posed no carcinogenic risk to occupationally exposed workers, or to the general population exposed to much lower levels.

Drawing such conclusions from these studies is problematic. The number of deaths was very small, and a low incidence cancer (such as lymphoma, liver, brain) may not have been found because the sample was too small or the clinical latency period was too short. Since cancer is a disease of long latency, too few years might have passed for the effect to be demonstrated. Both of these factors could bias the studies toward finding no significant effects (Shindell, 1986). In addition, many of these studies had serious design flaws, and some were based on poorly documented company records.

Little is known about the extent or magnitude of chronic health problems related to occupational exposure to pesticides because appropriate studies have not been done.

Several occupational groups whose pesticide exposure may put them at increased risk of cancer have been studied. Among them are farm owners and managers, farmworkers, pesticide sprayers, structural pest control operators (exterminators), pesticide manufacturing workers, and grain mill workers.

Malignant Lymphoma

Increased risk for malignant lymphoma has been found in farmers in California (Stubbs, 1984), Iowa (Burmeister, 1981, 1983), Minnesota (Cantor, 1985), Utah (Schumacher, 1985), Wisconsin (Saftlas, 1987), and New Zealand (Pearce, 1985); and in grain mill workers (Alavanja, 1987b).

A case-control study in Kansas found increased risk of non-Hodgkin's lymphoma in farmers who used herbicides more than 20 days a year; even higher risk was found for farmers who frequently mixed or applied herbicides themselves. The excess mortality was associated with exposure to the herbicide 2,4-D (Hoar, 1986).

In Iowa and Minnesota farmers, excess mortality for non-Hodgkin's lymphoma was associated with exposure to methyl bromide, pentachlorophenol, insecticides, and herbicides (Everett, 1985). Increased risk for small cell lymphocytic lymphoma was associated with exposure to nicotine, lindane, glyphosphate (Roundup), Atrazine, and Cyanazine, and, for all types of non-Hodgkin's lymphoma, with exposure to DDT, chloramben, and carbofuran (Cantor, 1985).

One case study reports two California firemen who had cleaned up a tank truck spill of the fumigant 1,3-dichloropropene and died six years later of malignant lymphoma (Markovitz, 1984).

Leukemia

Increased risk for leukemia has been found in farmers in Iowa (Burmeister, 1981, 1982), Minnesota (Blair, 1985a), Nebraska (Blair, 1979, 1985b), North Carolina (Delzell, 1985), Oregon and Washington (Milham, 1971), Wisconsin (Saftlas, 1987), and British Columbia (Gallagher, 1984).

In Iowa and Minnesota farmers, excess risk of leukemia was associated with exposure to Ethoprop, nicotine, Methoxychlor, and DDT (Blair, 1985a), and with exposure to insecticides and herbicides (Everett, 1985). In Iowa, the risk was higher for farmers who lived in the counties using the most herbicides (Burmeister, 1982), and in Nebraska for farmers from high insecticide use counties (Blair, 1979).

Multiple Myeloma

Increased risk for multiple myeloma has been found in farmers in Iowa (Burmeister, 1981, 1983), Washington (Milham, 1971), Wisconsin (Cantor, 1984; Saftlas, 1987), Australia (Nandakumar, 1986), and Sweden (Wiklund, 1986a); in Finnish herbicide sprayers (Riihimaki, 1982), and in orchard farmers in New Zealand (Pearce, 1985). In Wisconsin

TABLE 2

Reentry Intervals (REI)* in Days (1988 California Department of Food and Agriculture and the Environmental Protection Agency for Selected Pesticides)

Pesticide	CALIFORNIA						EPA
	Apples	Citrus	Corn	Grapes	Peaches	Other	All Crops
All Category I†	1	1	1	1	1	1	—
Aldicarb (Temik)	1	1	1	1	1	1	1
Anilazine (Dyrene)	2	2	2	2	2	2	1
Carbofuran	—	—	14	—	—	—	2
Chlorpyrifos	—	2	—	—	—	—	4
Diazinon	—	5	—	5	5	—	—
Dimethoate	—	2	—	2	—	—	4
Dioxathion	—	30	—	30	30	—	1
Disulfoton	2	2	2	2	2	2	1
Endosulfan	2	2	2	2	2	2	2
Ethion	2	30	2	14	14	2	1
Fenamiphos (Nemacur)	1	1	1	1	1	1	2
Fensulfothion	1	1	1	1	1	1	1
Guthion	14	30	—	21	14	14	1
Malathion	—	1	—	1	1	—	—
Methidathion	2	30,40	2	2	2	2	1
Methomyl (Lannate)	2	2	2	2	2	—	2
Methyl bromide	—	—	—	—	—	—	2
Methyl parathion	14	14	14	14	21	14	1
Mevinphos (Phosdrin)	2	4	2	4	4	2	—
Monitor	2	2	2	2	2	2	1
Monocroptophos	2	2	2	2	2	2	1
Naled (Dibrom)	—	1	—	1	1	—	1
Parathion-ethyl	14	30,45,60,90	14	21	21	14	2
Phorate (Thimet)	2	2	7	2	2	2	1
Phosalone (Zolone)	—	7	—	—	7	1	1
Phosmet (Imidan)	—	—	—	5	5	—	1
Propargite (Omite)	—	14	—	14	—	—	7
Propargite (Omite CR)	—	42	—	—	—	—	—

* *A reentry level (REI) is a quarantine period after a pesticide has been sprayed before workers are permitted to enter the field. When a longer interval is not on the label, workers can enter "when sprays have dried and dusts have settled."*

† *The most acutely toxic, with oral LD50 less than 50 mg/kg, dermal LD50 less than 200 mg/kg or inhalation LD50 less than 2000 mg/l/h. In California (but not EPA) all Toxicity Category I pesticides have a one day REI.*

farmers, excess risk for multiple myeloma was found in those born after 1905 and living in high insecticide use counties (Cantor, 1984).

A case-control study using Surveillance Epidemiology and End Results (SEER) data in selected counties in Washington State and Utah, and in metropolitan Atlanta and Detroit, found the highest risk for multiple myeloma in subjects who reported past exposure to pesticides (Morris, 1986).

Testicular Cancer

Increased risk of testicular cancer has been found in farmers and farm managers in England and Wales (McDowall, 1984) and in agricultural workers in Texas (Mills, 1984).

Statistically nonsignificant increased risk of testicular cancer has been found in pest control operators in Sweden, where mortality ratios were shown to increase from 1961-66 to 1967-73 to 1974-79 (Wiklund, 1986b). Statistically nonsignificant increased risk was found in farmers in Wisconsin (Saftlas, 1987), and in pesticide manufacturing workers in Michigan and Arkansas where the only common exposure the workers had was

to methyl bromide (Wong, 1984).

Two cases of testicular cancer in 30-year-olds, diagnosed within a year of each other, were reported in Illinois. Both had had occupational exposure to pesticides at the same canning plant (Prabhakar, 1978).

Cancer of the Gastrointestinal Tract

Increased risk for liver cancer has been found in agricultural workers in New Jersey (Stemhagen, 1983) and in Swedish grain millers (Alavanja, 1987b). A trend for increasing risk of liver cancer from 1961 to 1979 was found in Swedish farmers (Wilkund, 1986a). Statistically nonsignificant elevated risk has been found in pesticide manufacturing workers (Wong, 1984); and for liver/gallbladder cancer in California farmworkers, both white (includes Hispanics) and nonwhite, and in farm owners, both white (includes Hispanics) and nonwhite (Stubbs, 1984).

A five-center collaborative study found statistically nonsignificant elevated risk of liver cancer for pesticide exposure, employment in agriculture, employment in livestock agriculture, and for occupation as farmer or farmworker. No consistent trend between years of farming and risk was demonstrated (Austin, 1988).

Increased risk of stomach cancer has been found in farmers in Iowa (Burmeister, 1981), British Columbia (Gallagher, 1984a), Wisconsin (Saftlas, 1987), and Sweden (Wiklund, 1986a); and in farmworkers in California (Stubbs, 1984). Statistically nonsignificant elevated ratios were found in British Columbia farmworkers (Gallagher, 1984b); and in pesticide manufacturing workers (Mabuchi, 1980).

Excess mortality from pancreatic cancer has been found in grain mill workers (Alavanja, 1987a); and in farmers in British Columbia (Gallagher, 1984b), Iowa (Burmeister, 1981), and Wisconsin (Saftlas, 1987). Statistically nonsignificant elevated ratios were found in East German pesticide sprayers (Barthel, 1981) and British Columbia farmworkers (Gallagher, 1984a, b).

Lung Cancer

Excess mortality from lung cancer has been found in Florida pest control operators (Blair, 1983); in Maryland pesticide manufacturing workers (Mabuchi, 1980); and in East German pesticide sprayers (Barthel, 1981). Statistically nonsignificant increased ratios were found in U.S. pest control operators (Wang, 1979); in California farmers (Stubbs, 1984); in pesticides sprayers in England and Wales (Coggon, 1986); and in Finnish pesticide sprayers (Riihimaki, 1982).

In the 1970s, several case reports suggested a possible association between pesticide exposure and cancer in children.

Brain Cancer

Excess mortality from primary brain cancer was found in California farmworkers (Stubbs, 1984) and farmers in Italy (Musicco, 1982). Statistically nonsignificant elevated ratios were found in Florida pest control operators (Blair, 1983); in herbicide sprayers in England and Wales (Coggon, 1986); in pesticide manufacturing workers (Wong, 1984); and in farmers in Iowa (Burmeister, 1981), North Carolina (Delzell, 1985), and Wisconsin (Saftlas, 1987).

CANCER IN CHILDREN AND PESTICIDE EXPOSURE

In the 1970s, several case reports suggested a possible association between pesticide exposure and cancer in children. In Ohio, five children were diagnosed with neuroblastoma at the same hospital in the same year and all of the mothers of the children had prenatal exposure to chlordane (Infante, 1978).

Nine cases of colorectal cancer, a rarity in children, were diagnosed at the same hospital within a two-year period; eight of the children were from rural areas in Mississippi, Arkansas, or Tennessee and had had insecticide exposure (Pratt, 1977). This same investigator reported a case of colon cancer in a child who had lived in Times Beach, Missouri, where he had potential exposure to dioxin (Pratt, 1986).

A study in Baltimore, Maryland, found children with primary brain cancer were more likely to have had pesticide exposure in the home than children without cancer. Household insecticides were reported to be associated with leukemia and aplastic anemia in children (Reeves, 1982). A study of children in Los Angeles with acute lymphocytic leukemia found increased risk for children whose parents used pesticides in the home, with the risk being even higher for lawn or garden use (Lowengart, 1987).

A cluster of childhood cancer has been reported from agricultural communities in the San Joaquin Valley of California. In McFarland, over a period of three years, eight children were diagnosed with cancer when two cases would have been expected. The types of cancer were two cases of Wilms' tumor and one case each of non-Hodgkin's lymphoma, neuroblastoma, astrocytoma, osteogenic sarcoma, fibrosarcoma and rhabdomyosarcoma (California Department of Health Services, 1988; Kern County Health Department, 1986). Since this report, two more cases of cancer have occurred in children in McFarland; a 14-year-old boy died of hepatoblastoma in November 1987 and a case of primary brain tumor has been diagnosed recently.

No direct evidence implicates pesticides in these cancers but Kern county, where McFarland is located, is the second leading pesticide use county in California, annually using approximately 11% to 12% of all restricted-use pesticides in the state (California Department of Food and

TABLE 3
Teratogenic Pesticides* (Based on EPA Data as of June, 1988)

Pesticide	Use	Pesticide	Use
Acrolein (Aqualin)	H	Fenarimol (Rubigam)	F
Altosid (Methoprene)	IGR	Fenoxaprop ethyl	H
Amiben (Chloramben)	H	Fluazifop-butyl (Fusilade)	H
Avermectin	I	Folpet	F
Bacquacil		Hexachlorobenzene	F
Baycor (Bitertanol)	F	Imidan (Phosmet)	I
Bayleton (Triadimefon)	F	Kinoprene	IGR
Benazolin-ethyl	H	Larvadex (Cyromazine)	I
Benomyl	F	Mancozeb	F
Bentazon (Basagran)	H	Methyl parathion	I
Bladex (Cyanazine)	H	Mirex	I
Bromoxynil	H	Nemacur (Fenamiphos)	N
Cacodylic acid	H	Nitrofen (TOK)	H
Captafol	F	Omite (Propargite)	A
Captan	F	OPP	D,F
Carbaryl (Sevin)	I	OPP-sodium salt	D,F
Chlordimeform	I,A	Paclobutrazol	PGR
Chlorpropham	H,PGR	PCNB	F
Copper sulfate	F	Picloram	H
Cycloheximide (Acti-dione)	F	Potassium maleic hydrazide	PGR
2,4-D acid	H	Sodium arsenate	I
Dichlobenil	H	Sodium arsenite	F,H,I
Dichlorophene	F,B	Sodium omadine	
DMF		2,4,5-T	H
2,4-DP Acid (Dichlorprop)	H	Terrazole	F, NI
Dinocap (Karathane)	F,A	Tributyltin oxide	F
Dinoseb	H	Trichlorfon	I
Diquat	H	Trifluralin	H
Endosulfan	I	Triphenyltin fluoride	F
Endothall	H	Triphenyltin acetate	F,H,M
Ethion	I	Triphenyltin hydroxide	F
2-Ethyl 1,3-hexanediol		Vinyzene	
Ethylene dichloride	Fum	Warfarin	R

**A—acaracide*
B—bactericide
D—disinfectant
F—fungicide
Fum—fumigant
H—herbicide
I—insecticide
IGR—insect growth regulator
M—molluscicide
N—nematicide
NI—nitrification inhibitor
PGR—plant growth regulator
R—rodenticide
Rep—repellant
S—solvent
WP—wood preservative

Agriculture, 1986). Restricted-use pesticides are those that require a permit for use and must be applied by, or under the supervision of a state-certified applicator. In California only, amount used must be reported. An estimated one third of the pesticides used in agriculture in Kern County are proven or suspected animal carcinogens.

REPRODUCTIVE OUTCOME AND PESTICIDE EXPOSURE

Many pesticides have been shown to be teratogenic, embryotoxic or fetotoxic, causing the equivalent of spontaneous abortion, death of the fetus in utero, or a broad range of developmental, behavioral or growth problems. Other effects on the reproductive system of laboratory animals include infertility, sterility and chromosomal abnormalities (Schardein, 1985b).

DBCP and Sterility in Humans

In 1977, several men working in

the pesticide formulation division of a California chemical company noticed they had not recently fathered children. All of the men had exposure to the agricultural fumigant 1,2-dibromo-3-chloropropane (DBCP). Five of the first men tested were shown to be either azoospermic, with total absence of sperm, or oligospermic, with very low sperm counts of less than 20 million.

Further study of male employees at the plant found that of those men exposed to DBCP, 13% were sterile or azoospermic (compared to 2.9% of workers with no exposure), 16.8% had very low sperm counts or were oligospermic (compared to none of the nonexposed), and 15.8% had lowered sperm counts (compared to 5.7% of the nonexposed workers). Two of the sterile workers had not had any exposure to DBCP in nine and 13 years respectively, and both had fathered children prior to their exposure to the pesticide. None of the sterile men showed any improvement one year later (Whorton, 1979, 1980). A follow-up five to eight years after all exposure to DBCP ceased found that for some of the workers the damage to their testes was permanent (Eaton, 1986).

When the sterility in the workers in California was discovered, studies of other workers exposed to DBCP and a similar pesticide, ethylene dibromide (widely used as a grain fumigant), were done. Sperm counts of agricultural workers in six different southern states with exposure to DBCP showed low sperm counts (Sandifer, 1979).

Decreased fertility was found in workers at a plant in Arkansas that manufactured ethylene dibromide (Wong, 1979). Sterility was found in men working at a DBCP plant in Israel (Potashnik, 1978).

DBCP was used by injecting it into the soil. It is now the most widespread contaminant of groundwater in the state of California and of drinking water in many agricultural valley towns, where thousands of wells have been capped due to the contamination (California Legislature, 1985; Russell, 1987). Both DBCP and ethylene dibromide are potent animal carcinogens.

Birth Defects

Birth defects are the leading cause of infant mortality and a major cause of infant morbidity in the U.S. They are relatively rare, occurring in 3% to 7% of all births. The cause is known for only a small percentage of birth defects (eg, alcohol, certain drugs, certain viruses, ionizing radiation, chromosomal defects). For 65% of birth defects, the cause is unknown (Janerich, 1983).

First trimester pesticide exposures have been associated with birth defects and occasional case reports and legal cases.

In one of the few studies of farmworkers, all births in a California County Hospital were investigated to determine any difference in the prevalence of birth defects in infants born to agricultural workers and nonagricultural workers. An increased prevalence of limb-reduction defects was found in infants of agricultural workers (Schwartz, 1986). A larger follow-up study showed an association between residence in agricultural counties and limb-reduction defects (Schwartz, 1988).

In occasional case reports and legal cases first trimester occupational pesticide exposures have been associated with birth defects. In a recent limb-reduction defect case settled out of court, the farmworker mother's exposure was to the fungicides Captan and Benomyl sprayed in the vineyard where she was picking grapes. In another recent case, a woman was exposed while picking cauliflower that had been sprayed with Metasystox-R, a known animal teratogen. She delivered a chromosomally normal child with multiple severe defects that only survived a short time (Midtling, in press).

Studies in Arkansas (Nelson, 1979), New Zealand (Hanify, 1981), and Hungary (Thomas, 1980) have been done to determine if parental environmental or occupational exposures to the phenoxy herbicides, mainly 2,4,5-T and 2,4-D, were associated with birth defects. None have found any significant associations between these herbicides and major structural defects.

Vietnam veterans have also raised concerns about their exposure to the combination of the phenoxy herbicides 2,4-D and 2,4,5-T known as "Agent Orange." Two well-conducted case-control studies done by the Centers for Disease Control in Atlanta and by the Australian Government found no relationship between service in Vietnam and fathering a child with a birth defect (Australian Government, 1983; Erickson, 1984).

The State of California has recently set up a birth defects monitoring program for 60% of all births in the state, which includes major agricultural areas. A California law (SB950, 1984) requiring pesticide product registrants to supply adequate chronic toxicity testing for their products or lose their California registration is called "The Birth Defects Prevention Act."

Spontaneous Abortion

It is possible that more birth defects are not associated with pesticide exposures because of the toxicity to the embryo and fetus. The fetus may die very early in the pregnancy and be spontaneously aborted (Schardein, 1985a). Spontaneous abortion, unfortunately, is an even more difficult area of reproductive epidemiology to study than birth defects. A study of maternal occupation and fetal death found farmworker women to be at increased risk for spon-

taneous abortion (Vaughan, 1984). A study in India found increased spontaneous abortion, stillbirth, and sterility in grape garden workers (Rita, 1987).

CHRONIC EFFECTS FROM ORGANOPHOSPHATE PESTICIDES

Pesticide-Induced Delayed Neuropathy

While the acute toxic effects of organophosphate exposure are well characterized and documented (Namba, 1971; Morgan, 1982; Hayes, 1982), little is known about neuropathological and neurobehavioral delayed or chronic effects in humans.

Certain organophosphates can cause a pesticide-induced delayed neurotoxicity (PIDN). Long and large diameter fibers in the spinal cord and peripheral nervous system are damaged. Demyelination results in muscle weakness that may progress to paralysis. The feet and legs are usually more severely affected than the hands and arms. Onset is usually two to four weeks after the acute exposure (Cherniak, 1986; Johnson, 1980; Soliman, 1982; Wilson, 1985). Currently registered organophosphate pesticides found to cause PIDN in hens (the experimental animal used for testing) include EPN, trichlorfon (Dipterex), DDVP (Vapona), DEF, and isofenphos (Oftanol).

PIDN is thought to be the cause of severe neurological disease in 12 workers at a pesticide plant in Texas that manufactured leptophos (Phosvel) for export from 1971 to 1976. Four employees were diagnosed with multiple sclerosis, two with psychiatric disorders, and three with encephalitis, encephalomyelitis, and postinfectious encephalomyelitis. The National Institute for Occupational Safety and Health (NIOSH) investigated the plant in 1976 when the company applied to the EPA to market the product in the U.S. for use on cotton. NIOSH determined that the workers' neurological disease was work-related (Xintaras, 1978). Leptophos had caused paralysis and death in thousands of water buffalo in Egypt, where it was being used on cotton.

Two case reports in the literature of suicidal ingestions of organophosphates indicate that humans may be more susceptible to PIDN than the hen. Chlorpyrifos (Lorsban, Dursban) may be implicated in PIDN in a 20-year-old man in Italy (Lotti, 1986). Since chlorpyrifos is negative in the hen assay, is coming into increasingly wider use, and persists in body tissues longer than other organophosphates, it should be reevaluated in this regard.

Amendments to federal and state pesticide laws and regulations that would protect farmworkers and better their working conditions are resisted in the agricultural and agrichemical industries.

Neurobehavioral Effects

Several early case reports document that organophosphate pesticides can cause profound mental and psychological changes in human beings. In one of the earliest studies, mental patients were used as research subjects; administration of the pesticide resulted in an apparently permanent exacerbation of acute psychosis in one previously stable patient (Rowntree, 1950). This was before the days of human rights research committees.

Several other cases reports of mental illness or severe psychological disturbances in pesticide applicators have been reported. Behavioral changes such as anxiety, difficulties in concentration, memory deficits, and other more subtle effects have also been widely reported (Bowers, 1964; Brown, 1971; Dille, 1964; Duffy, 1979; Levin, 1976; Rodnitzky, 1975). Neurobehavioral effects of pesticides were discussed in a recent review of delayed effects of pesticides (Sharp, 1986).

Few follow-up studies have been done in people poisoned with pesticides to determine if any long-term or delayed effects were present. One such study examined 117 of 235 individuals three years after they had been reported by California physicians as occupationally poisoned by organophosphate pesticides, mainly parathion and Phosdrin (Tabershaw, 1966). Thirty-three still had complaints three years later, in ten of whom the central nervous system was affected—mainly visual disturbances. No major psychiatric or neurological sequelae were found.

A recent study investigated neuropsychological status of 100 people who had been poisoned by organophosphate pesticides (mainly parathion) an average of nine years prior. Their scores on a battery of four tests were compared to the same number of nonpoisoned controls. The poisoned subjects had significant differences in measures of memory, abstraction and mood; twice as many had scores consistent with cerebral damage or dysfunction (24% compared to 12% of controls). MMPI (Minnesota Multiphasic Personality Inventory) scores showed greater distress and complaints of disability in the poisoned subjects (Savage, 1988).

CHRONIC EFFECTS OF N-METHYL CARBAMATE PESTICIDES

The N-methyl carbamates such as aldicarb (Temik), carbaryl (Sevin), methomyl (Lannate, Nudrin), and carbofuran (Furadan), are similar to the organophosphates in their acute toxicity, and like them are inhibitors of the enzyme cholinesterase. Except for an aldicarb-related tractor accident death of a young farmworker (Lee, 1984), no occupational deaths have been reported from these compounds in the U.S.

Several episodes of consumer poi-

soning from food contaminated with aldicarb have occurred, however. Two episodes of poisoning from aldicarb residues in hydroponic cucumbers have been reported (Goes, 1980); and in July, 1985, over 1,000 consumers became ill from eating aldicarb contaminated watermelon (Centers for Disease Control, 1986). The pesticide intermediate, methyl isocyanate (MIC), which caused the deaths and illnesses in Bhopal, India, is used in the manufacture of N-methyl carbamates.

The N-methyl carbamates have not been thought to pose chronic problems. However, a study from Wisconsin found adverse effects on the immune system in women who drank water contaminated with low levels of aldicarb (Fiore, 1986). Animal experimental data supports the findings in humans in that immune system effects were seen at very low levels (Olson, 1987). Aldicarb is a low-level groundwater contaminant in Wisconsin, California, and other states (Cohen, 1984; Zaki, 1982).

EFFECTS OF FUMIGANTS

Fumigants are the most highly toxic of all pesticide chemicals used in agriculture. They are highly biologically reactive compounds. Many are alkylating agents, mutagens, carcinogens, neurotoxins, and hepatotoxins.

These chemicals are gases, penetrating readily into the lungs where they enter the blood and are rapidly distributed throughout the body. Full respiratory protection is mandatory when working with these pesticides and death can occur rapidly if the fumes are inhaled, even for a short period of time. However, skin absorption can be a significant worker hazard as well (van den Oever, 1982, 1984; Zwaveling, 1987).

Fumigants, as a class, have caused severe human illness and death (Behrens, 1986; Hine, 1969; Letz, 1984; Radimer, 1974). Methyl bromide, a widely used soil sterilant and post-harvest fumigant in agriculture, as well as a structural fumigant, has been responsible for more occupational deaths in California than any other pesticide (Hine, 1969). Toxic psychosis and irreversible neurological and neurobehavioral sequelae after recovery from acute poisoning, as well as more subtle effects from chronic exposures, have been reported (Anger, 1986; Greenberg, 1971; Hine, 1969; Prockop, 1986).

The highly toxic fumigants DBCP and ethylene dibromide, chemically

Workers in agriculture are at a four times greater risk of skin disease than workers in other industries.

similar to methyl bromide and D-D (Telone) which has replaced them, were discussed in relation to sterility in male workers.

EFFECTS OF FUNGICIDES

Fungicides are not acutely toxic compounds, with acute median lethal dose (LD 50) being 5,000 mg/kg or more for the most widely used ones. However, they pose the greatest risk of cancer, and several widely used fungicides are teratogens as well. The case of limb-reduction defect in a child whose mother was exposed to Captan and Benomyl was previously discussed. A recent report made some risk estimates for cancer in consumers who eat contaminated food (National Academy of Science, 1987); however, no studies on exposed workers have been done.

Several of the fungicides cause allergic contact dermatitis as well as primary contact dermatitis, with Captafol (Difolatan), Maneb, Mancozeb, and Benomyl being responsible for many of the reported cases (Adams, 1983).

EFFECTS OF HERBICIDES

Herbicides are the most frequently applied agricultural pesticide product. Although most herbicides are not acutely toxic, chronic toxicity and environmental contamination and drift are serious concerns.

Paraquat (Gramoxone) is one of the most widely used herbicides in the world. It is also the most toxic, and has been responsible for thousands of deaths. While many of the fatalities are due to suicidal ingestions (especially in Japan where it is estimated that over 1,300 suicides a year involve Paraquat), a large number have occurred from accidentally drinking the herbicide from unlabeled beverage containers or food jars, especially among children. Paraquat poisoning has no antidote or effective treatment if a sufficient amount of the chemical has been absorbed.

Acute poisoning with Paraquat can cause severe injury to the eyes, skin, nose and throat, as well as damage to the liver, kidneys, and myocardium. These effects, though severe, are reversible, with death being from asphyxiation due to pulmonary fibrosis. It was initially thought that death from Paraquat could not occur from dermal absorption. However deaths have been reported in farmers spraying dilute mixtures whose only exposure was through the skin (Fitzgerald, 1978) and from use as a scabicide, or to kill lice (Crome, 1986). Paraquat is an epithelial toxin and a powerful irritant that can cause epistaxis and severe dystrophy or complete loss of the fingernails (Howard, 1979).

The mechanism of toxicity of Paraquat is believed to be reaction with molecular oxygen in the lung to form superoxide ion. How this causes the acute alveolitis that leads to progressive pulmonary fibrosis and deaths is not known.

MPP is the metabolite of a street drug MPTP (1-methy-4-phenyl-1,2,3,6-tetrahydropyridine), which has been shown to cause Parkinson's

disease in drug abusers. MPP is similar in structure to Paraquat, which raises the issue of the herbicide as a possible etiologic agent or risk factor for the disease. Cases of Parkinson's disease in pesticide applicators (Bocchetta, 1986; Sanchez-Ramos, 1987), as well as higher prevalence in agricultural areas of Quebec where there is pesticide use, have been reported (Barbeau, 1987).

Dinoseb, another highly toxic herbicide from the dinitrophenol family, was suspended for most uses by the EPA in October, 1986. In 1983, a young Texas farmworker, spraying Dinoseb and wearing a leaking backpack sprayer, died of acute poisoning from the pesticide three days after he began spraying.

Several widely used herbicides in the U.S. are oncogens and groundwater contaminants, such as alachlor (Lasso), 2,4-D, glyphosate (Roundup), and Simazine (Princep).

CONCLUSION

The realities of agricultural practice, the lack of legal protections, and severe weaknesses in the existing laws, combined with the toxic pesticides that are ubiquitous in the farmworkers' environment, make agricultural work especially hazardous. Farmworkers are exposed to toxic pesticides from many sources—the crops they cultivate and harvest, the soil in which the crops are grown, drift in the air and water from pesticides applied to adjacent fields or to the very field in which they are working.

Farmworkers live in homes surrounded by fields that are heavily and repeatedly sprayed. Pesticides are likely to be in the irrigation water, which many farmworkers must use for bathing and drinking due to the substandard living quarters provided by some employers. Pesticides may contaminate the groundwater from which they get their drinking water. Farmworkers are more likely to consume produce very soon after harvesting and thus may get more pesticide residues in their food than the general public.

Toxic occupational exposures start at a very young age, since agriculture is the only industry in which children comprise a significant part of the work force. Infants and very young children are often taken to the fields with their parents.

Amendments to federal and state pesticide laws and regulations that would protect farmworkers and improve their working conditions are resisted in the agricultural and agrichemical industries. In this regard, it is of interest that the first ban on DDT in the U.S. was not by the EPA in 1972, but in a 1967 United Farm Workers' union contract with a California grape grower.

The public health task is clear. Not only must more resources and priority be given to biological monitoring and epidemiological studies of farmworkers, but also support given to the efforts of farmworkers and their unions to make their workplaces safe for themselves and their children.

REFERENCES

Adams, R.M. Occupational skin disease. New York: Grune and Stratton, 1983.

Akesson, N.B., Yates, W.E. Problems relating to application of agricultural chemicals and resulting drift residues. *Annual Review of Entomology* 1964; 9:285-318.

Akesson, N.B., Wilce, S.E., Yates, W.E. Pesticide chemicals as environmental contaminants. (Undated document)

Alavanja, M.C.R., Malker, H., Hayes, R.B. Occupational cancer risk associated with the storage and bulk handling of agricultural foodstuff. *Journal of Toxicology and Environmental Health* 1987a; 22(3):247-254.

Alavanja, M.C.R., Rush, G.A., Stewart, P., et al. Proportionate mortality study of workers in the grain industry. *Journal of the National Cancer Institute* 1987b; 78(2):247-252.

Anger, W.K., Moody, L., Burg, J., et al. Neurobehavioral evaluation of soil and structural fumigators using methyl bromide and sulfuryl fluoride. *Neurotoxicology* 1986; 7:137-156.

Austin, H., Delzell, E., Gufferman, S., et al. Case-control study of hepato-cellular carcinoma, occupation and chemical exposures. *Journal of Occupational Medicine* 1988; 29:665-669.

Australian Government: Australian Veterans Health Studies. Case-control study of congenital anomalies and Vietnam service (birth defects study). Australian Government Publishing Service, Canberra, Australia, 1983.

Barbeau, A., Roy, M., Bernier, G., et al. Ecogenetics of Parkinson's Disease: Prevalence and environmental aspects in rural areas. *Canadian Journal of Neurological Sciences* 1987; 1436-1441.

Barnet, R.W., D'Ercole, J.D., Cain, J.D., et al. Organochlorine pesticide residues in human milk samples from women living in Northwest and Northeast Mississippi, 1973. *Pest Monit J* 1979; 13:47-51.

Barthel, E. Increased risk of lung cancer in pesticide-exposed male agricultural workers. *Journal of Toxicology and Environmental Health* 1981; 8:1027-1040.

Behrens, R.H., Dukes, D.C.D. Fatal methyl bromide poisoning. *British Journal of Industrial Medicine* 1986; 43:561-562.

Blair, A., Everett, G., Cantor, K., et al. Leukemia and farm practices (abstract). *American Journal of Epidemiology* 1985a; 122(3):535.

Blair, A., Grauman, D.J., Lubin, J.H. Lung cancer and other causes of death among licensed pesticide applicators. *Journal of the National Cancer Institute* 1983; 71(1):31-37.

Blair, A., Thomas, T.L. Leukemia among Nebraska farmers: A death certificate study. *American Journal of Epidemiology* 1979; 110(3):264-273.

Blair, A., White, D.W. Leukemia cell types and agricultural practices in Nebraska. *Journal of Occupational Medicine* 1985b; 40(4):211-214.

Bocchetta, A., Corsini, G.U. Parkinson's Disease and pesticides (letter). *Lancet* 1986; 11(2):1163.

Bowers, M.B., Goodman, E., Sim, V.M. Some behavioral changes in man following anticholinesterase administration. *Journal of Nervous and Mental Disease* 1964; 138:383-389.

Brown, H.W. Electroencephalographic changes and disturbance of brain function following human organophosphate exposure. *Northwestern Medicine* 1971; 70:845-846.

Burmeister, L.F. Cancer mortality in Iowa farmers, 1971-78. *Journal of the National Cancer Institute* 1981; 66(3):461-464.

Burmeister, L.F., Van Lier, S.F., Isacson, P. Leukemia and farm practices in Iowa. *American Journal of Epidemiology* 1982; 115(5):720-728.

Burmeister, L.F., Everett, G.D., Van Lier, S.F., et al. Selected cancer mortality and farm practices in Iowa. *American Journal of Epidemiology* 1983; 118(1):72-77.

California Department of Health Services. Epidemiologic study of adverse health effects in children: in McFarland, California, Phase II Report. Epidemiological Studies and Surveillance Section, Berkeley, California, January 19, 1988.

California Legislature Assembly Office of Research. The leaching fields: A nonpoint threat to groundwater. Joint Publications Office, Sacramento, California, March, 1985.

California Department of Food and Agriculture. Pesticide use report: Annual 1985. Sacramento, California: CDFA, 1986.

California Department of Food and Agriculture. Summary of illnesses and injuries reported in California by physicians as potentially related to pesticides, 1986. Sac-

ramento, California: CDFA, Worker Health and Safety Branch, April 1, 1987.

Cantor, K., Everett, G., Blair, A., et al. Farming and non-Hodgkin's lymphoma (abstract). *American Journal of Epidemiology* 1985; 122(3):535.

Cantor, K.P., Blair, A. Farming and mortality from multiple myeloma: A case-control study with the use of death certificates. *Journal of the National Cancer Institute* 1984; 72(2):251-255.

Cherniak, M.G. Organophosphorus esters and polyneuropathy. *Annals of Internal Medicine* 1986; 104:264-266.

Coggon, D., Pannett, B., Winter, P.D., et al. Mortality of workers exposed to 2 methyl-4-chlorophenoxyacetic acid. *Scandinavian Journal of Work, Environment and Health* 1986; 12:448-454.

Cohen, S.Z., Creeger, S.M., Carsel, R.F., et al. Potential pesticide contamination of groundwater from agricultural uses. In Krieger, R.F. and Seiber, J.N.: Treatment and Disposal of Pesticide Wastes. ACS Symposium Series #259, American Chemical Society, Washington, D.C., 1984.

Centers for Disease Control. Aldicarb food poisoning from contaminated melons—California. *Morbidity and Mortality Weekly Report* 1986; 35(16):254-258.

Crome, P. Paraquat poisoning 1986. *Lancet* 1986; 1:333-334.

deCampos, M., Olszyna-Marzys, A.E. Contamination of human milk with chlorinated pesticides in Guatemala and El Salvador. *Archives of Environmental Contamination and Toxicology* 1979; 8:43-58.

Delzell, W., Grufferman, S. Mortality among white and nonwhite farmers in North Carolina, 1976-1978. *American Journal of Epidemiology* 1985; 121(3):391-402.

Dille, J.R., Smith, P.W. Central nervous system effects of chronic exposure to organophosphate insecticides. *Aerospace Medicine* 1964; 35:475-478.

Duffy, F.H., Burchfiel, J.L., Bartels, P.H., et al. Long-term effects of an organophosphate upon the human electroencephalogram. *Toxicology and Applied Pharmacology* 1979; 47:161-176.

Eaton, M., Schenker, M., Whorton, D., et al. Seven-year follow-up of workers exposed to 1,2-dibromo-3-chloropropane. *Journal of Occupational Medicine* 1986; 28:1145-1150.

Erickson, J.D., Mulinare, J., McClain, P.W., et al. Vietnam veterans' risks for fathering babies with birth defects (Summary Report). *Journal of the American Medical Association* 1984; 252:903-912.

Everett, G., Blair, A., Cantor, K., et al. Environmental chemical exposures as risk factors for leukemia and non-Hodgkin's lymphoma (abstract). *American Journal of Epidemiology* 1985; 122(3):535-536.

Fiore, M.C., Anderson, H.A., Hong, R., et al. Chronic exposure to aldicarb-contaminated groundwater and human immune function. *Environmental Research* 1986; 41:633-645.

Fitzgerald, G.R., Barniville, G., Black, J., et al. Paraquat poisoning in agricultural workers. *Journal of the Irish Medical Association* 1978; 71:336-342.

Gallagher, R.P., Threlfall, W.J., Spinelli, J.J., et al. Occupational mortality patterns among British Columbia Farm Workers. *Journal of Occupational Medicine* 1984a; 26(12):906-908.

Gallagher, R.P., Threlfall, W.J., Jeffries, E., et al. Cancer and aplastic anemia in British Columbia farmers. *Journal of the National Cancer Institute* 1984b; 72(6):1311-1315.

Glotfelty, D.E., Seiber, J.N., Liljedahl, L.A. Pesticides in fog. *Nature* 1987; 325: 602-605.

Goes, E.A., Savage, E.P., Gibbons, G., et al. Suspected foodborne carbamate pesticide intoxications associated with ingestion of hydroponic cucumbers. *American Journal of Epidemiology* 1980; 111:254-260.

Gold, E., Gordis, L., Tonascia, J., et al. Risk factors for brain tumors in children. *American Journal of Epidemiology* 1979; 109(3):309-319.

Goldman, L.R., Mengle, D., Epstein, D.M. Acute symptoms in persons residing near a field treated with the soil fumigants methyl bromide and chloropicrin. *Western Journal of Medicine* 1987; 147:95-98.

Greenberg, J.O. The neurological effects of methyl bromide poisoning. *Industrial Medicine* 1971; 40:27-29.

Hanify, J.A., Metcalf, P., Nobbs, C.L., et al. Aerial spraying of 2,4,5-T and human birth malformations: An epidemiological investigation. *Science* 1981; 212:349-351.

Hayes, W.J. Jr. Pesticides Studied in Man. Baltimore: Williams and Wilkins, 1982.

Hine, C.H. Methyl bromide poisoning. *Journal of Occupational Medicine* 1969; 11:1-10.

Hoar, S.K., Blair, A., Holmes, F.F., et al. Agricultural herbicide use and risk of lymphoma and soft-tissue sarcoma. *Journal of the American Medical Association* 1986; 256(9):1141-1147.

Howard, J.K. A clinical survey of paraquat formulation workers. *British Journal of Industrial Medicine* 1979; 36:220-223.

Infante, P.F., Epstein, S.S., Newton, W.A. Jr. Blood dyscrasias and childhood tumors and exposure to chlordane and heptachlor. *Scandinavian Journal of Work, Environment and Health* 1978; 4:137-150.

Janerich, D.T., Polednak, A.P. Epidemiology of birth defects. *Epidemiology Reviews* 1983; 5:16-37.

Johnson, M.K. The mechanism of delayed neuropathy caused by some organophosphorus esters: Using the understanding to improve safety. *Journal of Environmental Science and Health* 1980; B15(6):823-841.

Kahn, E. Pesticide-related illness in California farmworkers. *Journal of Occupational Medicine* 1976; 18:693-696.

Kazen, C., Bloomer, A., Welch, R., et al. Persistence of pesticides on the hands of some occupationally exposed people. *Archives of Environmental Health* 1974; 29:315-318.

Kern County Health Department. Epidemiologic Study of Cancer in Children in McFarland, California, 1985-1986: Phase I. Statistical Considerations, Current Environment. Kern County Health Department, Bakersfield, California, November, 1986.

Lee, M.H., Randsell, J.F. A farmworker death due to pesticide toxicity: A case report. *Journal of Toxicology and Environmental Health* 1984; 14:239-246.

Letz, G.A., Pond, S.M., Osterloh, J.D., et al. Two fatalities after acute occupational exposure to ethylene dibromide. *Journal of the American Medical Association* 1984; 252:2428-2431.

Levin, H.S., Rodnitzky, R.L. Behavioral effects of organophosphate pesticides in man. *Clinical Toxicology* 1976; 9:391-405.

Lotti, M., Moretto, A., Zoppellari, R., et al. Inhibition of lymphocytic neuropathy target esterase predicts the development of organophosphate-induced delayed polyneuropathy. *Archives of Toxicology* 1986; 59:176-179.

Lowengart, R.A., Peters, J.M., Cicioni, C., et al. Childhood leukemia and parents' occupational and home exposures. *Journal of the National Cancer Institute* 1987; 79(1):39-46.

Mabuchi, K., Lilienfeld, A.M., Snell, L.M. Cancer and occupational exposure to arsenic: A study of pesticide workers. *Preventive Medicine* 1980; 9:51-77.

Markovitz, A., Crosby, W.H. A soil fumigant, 1,3-dichloropropene, as possible cause of hematologic malignancies. *Archives of Internal Medicine* 1984; 144:1409-1411.

Martin, P., Mines, R., Diaz, A. A profile of California farmworkers. *California Agriculture* 1985; 6:16-18.

Matthews, G.A. Pesticide Application Methods. New York: Longman, Inc., 1982.

McDowall, M., Balarajan, R. Testicular cancer and employment in agriculture. *Lancet* 1984; 1:510-511.

Midtling, J.E., Barnett, P.G., Coye, M.J., et al. Clinical management of field worker organophosphate poisoning: Case report. *Western Journal of Medicine* 1984; 143: 168-172.

Milham, S. Jr. Leukemia and multiple myeloma in farmers. *American Journal of Epidemiology* 1971; 91(1):307-310.

Mills, P.K., Newell, G.R., Johnson, D.E. Testicular cancer associated with employment in agriculture and oil and natural gas extraction. *Lancet* 1984; 1:207-209.

Morgan, D.P. Recognition and management of pesticide poisoning, 3rd Edition. Washington, D.C.: U.S. Environmental Protection Agency, 1982.

Morris, P.D., Koepsell, T.D., Daling, J.R., et al. Toxic substance exposure and multiple myeloma: A case-control study. *Journal of the National Cancer Institute* 1986; 76(6):987-994.

Mott, Lawrie. Pesticides in food: What the public needs to know. San Francisco: National Resources Defense Council, Inc., 1984.

Musicco, M., Filippini, G., Bordo, B.M., et al. Gliomas and occupational exposure to carcinogens: Case-control study. *American Journal of Epidemiology* 1982; 116(5): 782-790.

Namba, T., Nolte, C.T., Jackrel, J., et al. Poisoning due to organophosphate insecticides: Acute and chronic manifestations. *American Journal of Medicine* 1971; 50: 475-492.

Nandakumar, A., Armstrong, B.K., deKlerk, N.H. Multiple myeloma in western Australia: A case-control study in relation to occupation, father's occupation, socioeconomic status, and country of birth. *International Journal of Cancer* 1986; 37:223-226.

National Academy of Science. Regulating pesticides in food: The Delaney paradox. Committee on Scientific and Regulatory Issues Underlying Pesticide Use Patterns and Agricultural Innovation. Board on Agriculture, National Research Council. Washington, D.C.: National Academy Press, 1987.

Nelson, C.J., Holson, J.F., Green, H.G. Retrospective study of the relationship between agricultural use of 2,5,5-T and cleft palate occurrence in Arkansas. *Teratology* 1979; 19:377-384.

Olson, L.J., Erickson, B.J., Hinsdill, R.D., et al. Aldicarb immunomodulation in mice: An inverse dose-response to parts per billion levels in drinking water. *Archives of Environmental Contamination and Toxicology* 1987; 16:433-439.

Pearce, N.E., Smith, A.H., Fisher, D.O. Malignant lymphoma and multiple myeloma linked with agricultural occupations in a New Zealand cancer registry-based study. *American Journal of Epidemiology* 1985; 121(2):225-237.

Pearce, N.E., Smith, A.H., Howard, J.K., et al. Non-Hodgkin's lymphoma and exposure to phenoxyherbicides, chlorophenols, fencing work, and meat works employment: A case-control study. *British Journal of Industrial Medicine* 1986; 43:75-83.

Peoples, S.A., Maddy, K.T. Organophosphate pesticide poisoning. *Western Journal of Medicine* 1978; 129:273-277.

Potashnik, G., Ben-Aderet, N., Israeli, R., et al. Suppressive effect of 1,2-dibromo-3-chloropropane on human spermatogenesis. *Fertility and Sterility* 1978; 30:444-447.

Prabhakar, J.M. Possible relationship of insecticide exposure to embryonal cell carcinoma (letter). *Journal of the American Medical Association* 1978; 240:288.

Pratt, C.B., George, S.L., O'Connor, D., et al. Adolescent colorectal cancer and dioxin exposure (letter). *Lancet* 1986; 2:803.

Pratt, C.B., Rivera, G., Shanks, E., et al. Colorectal carcinoma in adolescents, implications regarding etiology. *Cancer* 1977; 40:2464-2472.

Prockop, L.D., Smith, A.O. Seizures and action myoclonus after occupational exposure to methyl bromide. *Journal of the Florida Medical Association* 1986; 73: 690-691.

Radimer, G.F., Davis, J.H., Ackerman, A.B. Fumigant-induced toxic epidermal necrolysis. *Archives of Dermatology* 1974; 110:103-104.

Ratner, D., Eshel, E. Aerial pesticide spraying: An environmental hazard (letter). *Journal of the American Medical Association* 1986; 256:2516-2517.

Reeves, J.D. Household insecticide-associated blood dyscrasias in children (letter). *American Journal of Pediatric Hematology/Oncology* 1982; 4:438-439.

Rettig, B.A., Klein, D.K., Sniezek, J.E. The incidence of hospitalizations and emergency room visits resulting from exposure to chemicals used in agriculture. *Nebraska Medical Journal* 1987; 7:215-219.

Riihimaki, V., Asp, S., Hemberg, S. Mortality of 2,4-dichlorophenoxy acetic acid and 2,4,5-trichlorophenoxyacetic acid herbicide applicators in Finland. First report of an ongoing prospective study. *Scandinavian Journal of Work, Environment and Health* 1982; 8:37-42.

Rita, P., Reddy, P.P., Reddy, S.V. Monitoring of workers occupationally exposed to pesticides in grape gardens of Andhra Pradesh. *Environmental Research* 1987; 44:1-5.

Rodnitzky, R.L., Levin, H.S., Mick, D.L. Occupational exposure to organophosphate pesticides, a neurobehavioral study. *Archives of Environmental Health* 1975; 30:98-103.

Rogan, W. Pollutants in breast milk. *New England Journal of Medicine* 1980; 302:1450-1453.

Rowntree, D.W., Nevin, S., Wilson, A. The effects of diisopropylfluorophosphonate in schizophrenia and manic depressive psychosis. *Journal of Neurology, Neurosurgery and Psychiatry* 1950; 13:47-62.

Russell, H.H., Jackson, R.J., Spath, D.P., et al. Chemical contamination of California drinking water. *Western Journal of Medicine* 1987; 147:615-622.

Saftlas, A.F., Blair, A., Cantor, K.P., et al. Cancer and other causes of death among Wisconsin farmers. *American Journal of Industrial Medicine* 1987; 11:119-129.

Sanchez-Ramos, J.R., Hefti, F., Weiner, W.J. Paraquat and Parkinson's Disease (letter). *Neurology* 1987; 4(37):723.

Sandifer, S.H., Wilkins, R.T., Loadholt, C.B., et al. Spermatogenesis in agricultural workers exposed to dibromochloropropane (DBCP). *Bulletin of Environmental Contamination and Toxicology* 1979; 23:703-710.

Saunders, D., Ames, R.G., Knoak, J.B., et al: Outbreak of OMite-CR induced dermatitis among orange pickers in Tulare County, California. *Journal of Occupational Medicine* 1987; 29:409-413.

Savage, E.P., Keefe, T.J., Tessari, J.D., et al. National study of chlorinated hydrocarbon insecticide residues in human milk. *American Journal of Epidemiology* 1981; 113:413.

Savage, E.P., Keefe, T.J., Mounce, L.M., et al. Chronic neurological sequelae of acute organophosphate poisoning. *Archives of Environmental Health* 1988; 43:38-45.

Schardein, J.L. Chemically Induced Birth Defects. New York: Marcel Deckker, 1985a.

Schardein, J.L., Schwetz, B.A., Kenel, M.F. Species sensitivities and prediction of teratogenic potential. *Environmental Health Perspectives* 1985b; 61:55-67.

Schumacher, M.C. Farming occupations and mortality from non-Hodgkin's lymphoma in Utah, a case-control study. *Journal of Occupational Medicine* 1985; 27(8):580-584.

Schwartz, D.A., LoGerfo, J.P. Congenital limb reductions in the agricultural setting. *American Journal of Public Health* 1988; 78(6):654-657.

Schwartz, D.A., Newsum, L.A., Heifitz, R.M. Parental occupation and birth outcome in an agricultural community. *Scandinavian Journal of Work, Environment and Health* 1986; 12:51-54.

Sharp, D.S., Eskenazi, B., Harrison, R., et al. Delayed health hazards of pesticide exposure. *Annul Review of Public Health* 1986; 7:441-471.

Shindell, S., Ulrich, S. Mortality of workers employed in the manufacture of Chlordane: An update. *Journal of Occupational Medicine* 1986; 28(7):4987-5011.

Soliman, S.A., Farmer, J., Curley, A. Is delayed neurotoxicity a property of all organophosphorus compounds? A study with a model compound, Parathion. *Toxicology* 1982; 23:267-279.

Stemhagen, A., Slade, J., Altman, R., et al. Occupational risk factors and liver cancer. *American Journal of Epidemiology* 1983; 117(4):443-454.

Stubbs, H.A., Harris, J., Spear, R.C. A proportionate mortality analysis of California agricultural workers, 1978-1979. *American Journal of Industrial Medicine* 1984; 6:305-320.

Tabershaw, I.R., Cooper, W.C. Sequelae of acute organophosphate poisoning. *Journal of Occupational Medicine* 1966; 8:5-20.

Tafuri, J., Roberts, J. Organophosphate Poisoning, Collective Review. *Annals of Emergency Medicine* 1987; 16:193/93-102/202.

Thomas, H.F. 2,4,5-T use and congenital malformation rates in Hungary (letter). *Lancet* 1980; 2:214-215.

U.S. Department of Agriculture. Agricultural Statistics, 1986. Washington, D.C.: Government Printing Office, 1986.

U.S. Environmental Protection Agency. Pesticide industry sales and usage, 1985 market estimates. Economic Analysis Branch, Benefits and Use Division, Office of Pesticide Programs. Washington, D.C.: EPA, September, 1986.

U.S. General Accounting Office. EPA is slow to carry out its responsibility to control harmful chemicals. GAO Report CED-81-1. Washington, D.C., October 28, 1980.

U.S. General Accounting Office. Pesticides: EPA's formidable task to assess and regulate their risks. GAO Report RCED-86-125. Washington, D.C., April, 1986a.

U.S. General Accounting Office. Pesticides: Better sampling and enforcement needed on imported food. GAO Report RCED-86-219. Washington, D.C., September, 1986b.

U.S. General Accounting Office. Pesticides: Need to enhance FDA's ability to protect the public from illegal residues. GAO

Report RCED-87-7, Washington, D.C., October, 1986c.
van den Oever, R., Roosels, D., Lahaye, D. Actual hazard of methyl bromide fumigation in soil disinfection. *British Journal of Industrial Medicine* 1982; 39:140-144.
van den Oever, R., Roosels, D., Lahaye, D. Actual hazard of methyl bromide fumigation in soil disinfection (letter of reply to critique of above article). *British Journal of Industrial Medicine* 1984; 41:282-283.
Vaughan, T.L., Daling, J.R., Starzyk, P.M. Fetal death and maternal occupation: An analysis of birth records in the state of Washington. *Journal of Occupational Medicine* 1984; 26:676-678.
Wang, H.H., MacMahon, B. Mortality of pesticide applicators. *Journal of Occupational Medicine* 1979; 21(11):741-744.
Wasserstrom, R.F., Wiles, R. Field duty: U.S. farmworkers and pesticide safety. Washington, D.C.: World Resources Institute, July, 1985.
Whorton, D., Milby, T.H., Krauss, R.M., et al. Testicular function in DBCP exposed pesticide workers. *Journal of Occupational Medicine* 1979; 21:161-166.
Whorton, D., Milby, T.H. Recovery of testicular function among DBCP workers. *Journal of Occupational Medicine* 1980; 22:177-179.
Wiklund, K. Trends in cancer risks among Swedish agricultural workers. *Journal of the National Cancer Institute* 1986a; 77(3): 657-664.
Wiklund, K. Testicular cancer among agricultural workers and licensed pesticide applicators in Sweden. *Scandinavian Journal of Work, Environment and Health* 1986b; 12:630-631.
Wilson, B.W., Hooper, M., Chow, E., et al. Assessment of farmworker risk from organophosphate-induced delayed neuropathy. In Honeycutt, R.C. et al. (Eds): Dermal exposure related to pesticide use. ACS Symposium Series No. 273, American Chemical Society, 1985.
Wong, O., Brocker, W., Davis, H.V., et al. Mortality of workers potentially exposed to organic and inorganic brominated chemicals, DBCP, TRIS, PBB and DDT. *British Journal of Industrial Medicine* 1984; 41:15-25.
Wong, O., Utidjian, H.M.D., Karten, V.S. Retrospective evaluation of reproductive performance of workers exposed to ethylene dibromide. *Journal of Occupational Medicine* 1979; 21:98-102.
Xintaras, C., Burg, J.R., Tanaka, S., et al. Occupational exposure to Leptophos and other chemicals. NIOSH Publication No. 78-136. Washington, D.C.: Government Printing Office, 1978.
Zaki, M.H., Moran, D., Harris, D. Pesticides in groundwater: The aldicarb story in Suffolk County, NY. *American Journal of Public Health* 1982; 72:1391-1395.
Zwaveling, J.H., deKort, W.L.A., Meulenbelt, J., et al. Exposure of the skin to methyl bromide: A study of six cases occupationally exposed to high concentrations during fumigation. *Human Toxicology* 1987; 6:491-495.

Minority Workers
IN SUMMARY

Pesticide-Related Health Problems and Farmworkers. Moses, M. *AAOHN Journal* 1989; 37(3):115-130.

1. Migrant and seasonal farmworkers are primarily ethnic minorities who are excluded from federal laws that protect other workers. Farmworkers live and work under substandard conditions that place them at increased risk of pesticide-related illness.

2. Agriculture uses 80% of all pesticides in the U.S. Handlers who mix, load and apply pesticides as well as workers cultivating and harvesting crops sprayed with them are at risk of acute poisoning or even death from their exposures. Drift and run-off of agricultural pesticides pollute the air, soil and water, creating additional hazards to workers' families, community residents, and the environment.

3. Chronic effects, including cancer in adults and children, adverse reproductive outcomes, delayed neuropathy and neurobehavioral effects, are also associated with occupational and environmental exposure to pesticides.

ABOUT THE AUTHOR: Dr. Moses is a physician specializing in Environmental and Occupational Medicine. She is an assistant clinical professor at the University of California, San Francisco, and a member of the National Advisory Committee of the Environmental Protection Agency's Pesticide Farm Safety Center.

Adapted from materials prepared for the Hispanic Health Status Symposium (November 6, 1987), The University of Texas Health Science Center, San Antonio, Texas.