JEAN D. BRENDER, RN, PhD
PATRICIA A. HONCHAR, MS, PhD
CHARLES E. ALEXANDER, MD, Dr PH
RICHARD A. BEAUCHAMP, MD

Occupational pesticide poisoning: reportable disease in Texas

The incidence, recognition, and control of acute occupational pesticide poisoning are discussed. Few estimates are available to describe the scope and incidence of this problem in Texas and in the United States. Previous reports have noted that organophosphates, carbamates, and fumigants cause the majority of acute poisonings; recognition of these types of poisonings is discussed. Occupational groups that have an increased risk of pesticide poisoning include ground applicators, gardeners and nurserymen, harvesters or field-workers, warehousemen who handle and transport pesticides, formulators and manufacturers, fumigators, aerial applicators and, occasionally, police and fire fighters. Acute occupational pesticide poisoning was made a reportable disease in Texas under the Occupational Disease Reporting Law of 1985. Case reports from physicians will enable public health officials to understand the occurrence and distribution of these poisonings in Texas and to plan and implement measures to prevent further incidents.

KEY WORDS: OCCUPATIONAL DISEASES, PESTICIDE POISONING, ORGANOPHOSPHATES, CARBAMATES, INSECTICIDES.

Jean D. Brender, RN, PhD, Director, Environmental Epidemiology Program; Charles E. Alexander, MD, Dr PH, Bureau Chief, Bureau of AIDS and Sexually Transmitted Disease Control; Richard A. Beauchamp, MD, Public Health Physician, Environmental Epidemiology Program, Texas Department of Health, Austin; and Patricia A. Honchar, MS, PhD, Chief, Epidemiology and Surveillance Coordinating Activity, Office of the Director, National Institute of Occupational Safety and Health, Centers for Disease Control, Atlanta. Send reprint requests to Dr Brender, Bureau of Disease Control and Epidemiology, Texas Department of Health, 1100 West 49th St, Austin, TX 78756.

Implicit in their name, pesticides are poisonous, whether insecticides, fungicides, herbicides, rodenticides, or fumigants. Despite efforts to make these compounds specific for a particular target organism, most remain more or less toxic to humans as well. In the last 20 years, usage in the US has shifted from solid organochlorine compounds (eg, DDT, aldrin, and chlordane) to those which have less bioaccumulation in humans and animals, such as carbamates and organophosphates. However, the latter compounds are much more acutely toxic than most chlorinated hydrocarbons (1), and numerous instances of acute poisoning from them have occurred, especially in agricultural occupations. In this paper, we describe the scope, occurrence, recognition, and control of acute occupational pesticide poisoning, as well as the requirement to report diagnosed and suspected cases of such poisoning under the Texas Occupational Disease Reporting Act of 1985.

The problem

Few estimates are available of the number of pesticide poisonings occurring in the US. In California since 1973, detailed information has been available on pesticide illnesses from workers' compensation claims and through mandatory reporting (2). Since this intensive surveillance effort began, the total number of reported pesticide-related illnesses has varied from less than 1,200 to more than 2,400 cases per year in California (2–4). In 1985, there were 1,888 cases of confirmed or suspected pesticide-related poisonings reported (4). Of these, 80% (1,516) resulted from occupational exposures while the remaining 20% were nonoccupational or of unknown exposure. The incidence of pesticide-related illness or injury was estimated at 2.7 cases per 1,000 persons at risk for farm field workers and 60 cases per 1,000 for pesticide mixers, loaders, or applicators (4). Among the occupational exposures, the type of illness was reported as systemic in 43% (655) of the cases, while the remaining 57% (861) were limited to skin or eye symptoms. The type of pesticide was determined for 432 of these occupational cases, and over half were associated with exposure to organophosphates or carbamates; another 30 or more cases were associated with exposure to fumigants (aluminum phosphide, chloropicrin, and methyl bromide).

From recent Florida surveys of 436 citrus growers and 1,811 citrus workers, the incidence of confirmed pesticide poisonings was estimated at 3.4 cases per 1,000 field-workers at risk (5,6). Morgan et al (7) found 53 self-reported pesticide "poisoning" incidents between 1971 and 1977 in a cohort of workers from 13 states who were occupationally exposed to pesticides, most of which involved exposure to organophosphates and carbamates. Hayes and Vaughn (8) examined mortality from pesticide poisoning in the US in 1973 and 1974 as reported in death certificates: a total of 78 accidental deaths occurred, 12 of which occurred at work.

No current estimate of the incidence of pesticide poisoning is available for Texas, though several case series described this problem in the past (9,10). Hospital records were studied for 129 fully documented cases of pesticide poisoning which occurred between 1961 and 1967 in the Lower Rio Grande Valley (9). Ninety-three percent of the cases occurred during June, July, and August (which coincided with the cotton insect pest control season). Ninety-eight percent of the cases were exposed to pesticides dermally, and 95% were poisoned by ethyl or methyl parathion. In 1968, the number of acute organophosphate poisonings increased substantially over the 1961 to 1967 average; investigators attributed this increase to the more frequent use of ethyl parathion during that year (10).

Recognition of acute pesticide poisoning

Organophosphates, carbamates, and fumigants cause the majority of acute pesticide poisonings; Fig 1 lists the characteristics of acute pesticide poisoning by chemical group. In addition to acute effects, some pesticide exposures are associated with chronic health problems (eg, organophosphates and delayed neurotoxicity). These chronic effects will not be reviewed. A complete discussion of acute pesticide poisoning and the chronic health problems associ-

1. Characteristics of acute pesticide poisoning. Compiled from references 1, 11, 12, 18 and United States Navy Disease Vector Ecology and Control Center, Jacksonville, Fla, Emergency Medical Treatment for Acute Pesticide Poisoning *(chart), September, 1982.*

Chemical basis (examples of compounds)*	Pharmacologic action or site of toxicity	Routes of absorption	Major acute signs and symptoms	Laboratory tests
Chlorinated hydrocarbons [chlorobenzilate, Kelthane, Thiodan, methoxychlor, lindane, heptachlor, toxaphene, chlordane]	Neurotoxin; CNS, kidney, liver	Ingestion, dermal, inhalation	Apprehension, excitability, dizziness, headache, disorientation, weakness, paresthesia, convulsions	Pesticide and/or metabolites measured in blood; concentration more important than mere presence
Organophosphates [diazinon, malathion, methyl parathion, parathion, Guthion, chlorpyrifos (Dursban), Di-Syston, dichlorvos, S-Seven]	Irreversible inhibition of acetyl-cholinesterase enzyme	Ingestion, dermal, inhalation	*Mild:* fatigue, headache, blurred vision, dizziness, numbness of extremities, nausea, vomiting, excessive sweating and salivation, tightness in chest *Moderate:* weakness, difficulty talking, muscular fasciculations, miosis *Severe:* unconsciousness, flaccid paralysis, moist rales, respiratory difficulty, and cyanosis *Other:* cardiac arrhythmias	Red blood cell cholinesterase, plasma cholinesterase
Carbamates [aldicarb (Temik), methomyl, oxamyl, carbaryl (Sevin), carbofuran, Baygon]	Reversible inhibition of acetyl-cholinesterase enzyme	Ingestion, dermal, inhalation	Diarrhea, nausea, vomiting, abdominal pain, excessive sweating and salivation, blurred vision, difficulty breathing, headache, muscular fasciculations	Red blood cell and plasma cholinesterase may be normal and thus not reliable detectors of poisoning; carbamate metabolites in urine
Halocarbon and sulfuryl fumigants [methyl bromide, carbon disulfide, chloropicrin, ethylene dibromide, dibromochloropropane]	CNS, enzyme systems, liver, kidney, lungs	Ingestion, dermal, inhalation	Dizziness, headache, nausea, vomiting, abdominal pain, mental confusion, tremor, convulsions, pulmonary edema	Methyl bromide—blood bromide concentrations; carbon disulfides in urine
Phosphine fumigants [aluminum phosphide (Phostoxin)]	Lungs, CNS, liver, kidney	Inhalation, dermal, ingestion	Dizziness, headache, nausea, vomiting, dyspnea, pulmonary edema	None known; victim's breath may smell like garlic or acetylene
Cyanide fumigants [Cyclon]	Inactivates the cytochrome oxidase of cells in critical tissues, primarily the heart and brain	Ingestion, inhalation, dermal (rare)	*Large dose:* collapse and cessation of respiration *Smaller dose:* headache, weakness, confusion, nausea, vomiting, dizziness, hyperpnea, apprehension, convulsions *Other:* breath may smell like bitter almonds	Cyanide in blood and tissues; thiocyanate metabolite in urine and saliva
Nitrophenolic and nitrocresolic herbicides [dinitrocresol, dinoseb (Dinitro-3), dinitrophenol]	Liver, kidney, and nervous system; stimulation of oxidative metabolism in cell mitochondria	Ingestion, inhalation, dermal	Yellow staining of skin and hair; profuse sweating, headache, thirst, malaise, warm flushed skin, tachycardia, fever	Nitrophenols and nitrocresols in urine and serum
Chlorophenoxy compounds [2,4-D, Silvex, 2,4,5-T, Dicamba]	Skin, eyes, respiratory and gastrointestinal linings	Ingestion, dermal, inhalation	*Inhalation:* burning sensations in the nasopharynx and chest, dizziness *Ingestion:* vomiting, esophagitis, abdominal pain, diarrhea, fibrillary muscle twitching, stiffness of muscles of extremities, metabolic acidosis in large doses	Chlorophenoxy compounds in blood and urine
Dipyridyls [diquat (Aquacide), paraquat (Dextrone X)]	Injury of epithelial tissue: skin, nails, cornea, liver, kidney, and linings of gastrointestinal and respiratory tracts	Ingestion, dermal, inhalation	*Ingestion early:* nausea, vomiting, diarrhea, melena, pain (oral, substernal, abdominal) *48–72 hours after exposure:* oliguria, jaundice, cough, dyspnea, tachypnea, pulmonary edema, convulsions, coma	Paraquat and diquat in blood and urine

* Use of trade names is for identification only and does not imply endorsement by the Texas Department of Health or the National Institute for Occupational Safety and Health, Centers for Disease Control.

ated with these compounds can be found in Hayes, *Pesticides Studied in Man* (11), and the Environmental Protection Agency (EPA) publication by Morgan, *Recognition and Management of Pesticide Poisonings* (12). The EPA booklet is an excellent reference for the medical practitioner who treats pesticide poisoning on an emergency basis. The Texas State Poison Center (telephone number: 1–800–392–8548), the North Central Texas Poison Center (1–800–441–0040) and local and regional poison control centers are also valuable resources for clinicians in treating pesticide poisoning.

There are three principal routes of exposure to pesticides for workers: the respiratory, oral, and dermal routes (13). Most persons are aware of the potential for inhalation and ingestion, but many do not appreciate the potential danger of skin absorption. Occupational exposures to organophosphate and carbamate pesticides frequently occur by the dermal route (14). Pesticide residues can accumulate on skin, clothes, and hair during direct handling or application of the pesticide, or from plants and soils after application.

In a study of dermal penetration of insecticides in mice, the carbamates (carbofuran, methomyl, carbaryl) penetrated the most rapidly, and malathion, an organophosphate, penetrated the least rapidly (15). Other organophosphates (ie, parathion and chlorpyrifos) had intermediate penetration rates.

Organophosphate and carbamate (cholinesterase-inhibiting group) pesticides poison insects and mammals by binding with and inhibiting the action of acetylcholinesterase (16). Organophosphorus compounds react with acetylcholinesterase by phosphorylation with serine at the enzyme's active center which produces a relatively stable (and inactive) enzyme. If both of the methyl or ethyl alkyl groups are still present on the phosphate, significant enzyme reactivation (through dephosphorylation) is possible (16,17). However, if the phosphorylated enzyme is allowed to "age" (through loss of one of its alkyl groups), it becomes far more refractory to regeneration (17). Carbamate pesticides inactivate acetylcholinesterase through carbamylation which also involves the serine group at the enzyme's active center (18). This process is relatively reversible, and if carbamate is removed from the system (by dilution, dialysis, or metabolism and excretion), the enzyme will rapidly regain its ability to break down acetylcholine (16,18). The net effect of these reactions is an accumulation of acetylcholine at nerve synapses and motor endplates (19).

Signs and symptoms of organophosphate and severe carbamate poisoning can be classified into muscarinic (parasympathetic), nicotinic (primarily motor), and central nervous system manifestations according to site of action (20). Muscarinic signs and symptoms may include excess salivation, excess bronchial secretion, sweating, miosis and blurred vision, abdominal cramps, diarrhea, nausea, vomiting, and bradycardia (21). Nicotinic manifestations in striated muscle include muscular cramps, fasciculations, and weakness (20). In severe intoxication, nicotinic actions at the autonomic ganglia may sometimes mask muscarinic effects; these patients may have tachycardia, pallor, and an elevated blood pressure (18,20). Central nervous system effects include headache, drowsiness, dizziness, and, in severe cases, unconsciousness or coma. With organophosphate poisoning, the interval between exposure and clinical manifestations may be as short as five minutes after massive ingestion or up to 12 to 24 hours with lesser exposures (20). If death occurs, the usual cause is respiratory failure.

As in any suspected poisoning case, a thorough history of recent activities prior to the onset of symptoms is invaluable in the diagnosis of acute pesticide poisoning. Depending on the type of pesticide, route of exposure, estimated severity of poisoning, and observed signs and symptoms, some or all of the following measures may be necessary in treating organophosphate or carbamate poisoning. The patient's airway should be cleared of any excess secretions and oxygen should be administered. In severe poisonings, intubation and mechanically assisted ventilation may be necessary. The antidotes for organophosphate poisoning are atropine sulfate and pralidoxime (severe poisoning), while atropine alone is an adequate antidote for carbamate poisoning (12). Because "aged" phosphorylated acetylcholinesterase becomes resistant to reactivation, it is important to administer pralidoxime early after organophosphate poisoning to maximize its effectiveness as a cholinesterase reactivator (17,18). Pralidoxime is contraindicated for use in carbamate poisoning because of the pharmacological properties of this drug and the rapid reversal of the carbamate inhibited enzyme (11,12,17,18). If pesticide exposure has occurred through dermal contact, the patient should be decontaminated by washing the skin and shampooing the hair with an alkaline soap to prevent any further absorption (18). If exposure was through ingestion, the stomach and intestine should be cleared of unabsorbed pesticide by appropriate procedures such as the administration of syrup of ipecac (or, if the patient is unconscious, gastric intubation, suction, and lavage) followed by activated charcoal and sodium sulfate (12,18). Detailed descriptions of treatment regimens can be found in several medical references (11,12,17,18).

The monitoring of erythrocyte and plasma cholinesterase levels in organophosphate poisoning is important in determining the presence and degree of poisoning and assessing response to therapy. Several methods are available for measuring erythro-

cyte and plasma cholinesterase. Coye, Lowe, and Maddy provide a comprehensive review of the measurement and interpretation of cholinesterase activity in workers exposed to pesticides (22).

The level of erythrocyte cholinesterase is a better indicator of acetylcholinesterase activity at nerve synapses than that of plasma cholinesterase (23). According to Namba (20), clinical manifestations usually occur only if more than 50% of serum cholinesterase is inhibited, and severity of illness is proportional to the degree of inhibition of serum cholinesterase activity. However, Midtling and colleagues (24) followed patients with moderately severe symptoms whose erythrocyte cholinesterase was only 30% inhibited. In their case series of patients with organophosphate poisoning, the rate of decline in cholinesterase levels appeared more important than the total decline. Plasma cholinesterase usually returns to normal within a few weeks; but it may take one to three months for the erythrocyte cholinesterase levels to plateau and to return to normal (12,24). Therefore, serial determinations of erythrocyte and plasma cholinesterase levels may be more useful than single determinations. Minimum normal values of cholinesterase vary by test method and must be specified by the laboratory doing the test.

Fumigants are another group of pesticides that occasionally cause severe poisonings in workers. Poisoning is usually through inhalation, but the liquid and solid forms can be ingested or absorbed through the skin (18). These compounds can cause serious injury to the lungs, liver, heart, kidneys, and central and peripheral nervous systems (25,26). Hine provides a thorough review of ten cases of methyl bromide poisoning (25); all of which were exposed through inhalation in the food processing industry (nuts, fruits, and grains). Symptoms included malaise, headache, dyspnea, visual disturbances, weakness, nausea, and vomiting. Several patients developed acute chemical pulmonary edema. Similar symptoms were found in two children and 29 crew members who were exposed to toxic phosphine fumes from grain in storage aboard a grain freighter (26). Laboratory confirmation of fumigant intoxication depends on the compound. For many of these substances, no definitive laboratory tests are available. Blood bromide levels are useful to detect methyl bromide poisoning if the patient is not taking inorganic bromide medications. Cyanide can be measured in blood and tissues (or its metabolite thiocyanate in urine) (12).

Occupations at risk

Since the person with pesticide poisoning may present with nonspecific signs and symptoms, eliciting information about occupation and work activities may help the physician determine whether acute pesticide poisoning has occurred. Specific occupational groups have an increased risk of exposure to pesticides. These include ground applicators, gardeners and nurserymen, harvesters or field workers, warehousemen who handle and transport pesticides, formulators and manufacturers, fumigators, aerial applicators, and, occasionally, police and fire fighters, who may encounter pesticide spills as part of their work (2). In California during 1978, ground applicators had the highest number of reported occupationally related pesticide exposure illnesses and aerial applicators had the lowest number among these occupational groups (2). During 1985, however, more cases of systemic illnesses associated with pesticide exposure in California were reported in workers exposed to pesticide spray drift than in workers directly handling pesticides (4). In the 1968 survey in the Rio Grande Valley, over one third (44/118) of the pesticide poisonings occurred in persons who loaded spray planes (10).

The sites of dermal exposure may vary by occupation. In one study (27), plant workers mixing and bagging 4% and 5% carbaryl dust had the highest potential exposure on their hands, forearms, and the front body areas. In field workers applying 0.045% to 0.06% carbaryl spray, the highest exposures were on the shoulders and back of the neck.

Occasionally, an occupational death due to trauma is secondary to acute pesticide poisoning. Lee and Randsell (28) report such an incident in which a 20-year-old man died after collapsing in a field and being run over by farm machinery operated by another worker. He had been assigned to load granulated aldicarb, a highly toxic carbamate, into a hopper and apparently developed acute aldicarb poisoning through dermal exposure. Morgan et al (7) noted that death by accidental trauma was increased among a cohort of workers occupationally exposed to pesticides. Although it could not be determined whether pesticide poisoning was causally related to the fatal injuries, it is very plausible that pesticide exposure could predispose to injury by causing incoordination and altered sensorium.

The potential for occupational pesticide exposure and poisoning in Texas is substantial. The US Bureau of the Census estimated in 1982 that Texas had 27 pesticide-formulating establishments with a total of 2,600 employees, 1,600 of whom were production workers (29). An estimated 1,200 of these production workers were employed in the Houston-Galveston area. The number of Texas manufacturing plants producing the active ingredients of pesticides is unknown. Manufacturing/formulation establishments account for minor potential worker exposure to pesticides, when compared to the agricultural industry. From 1982 estimates, insecticides were applied to 6.5 million acres, and herbicides were ap-

plied to 10.4 million acres of land in Texas (30). In terms of land area receiving herbicide or insecticide applications, pesticide use is relatively heavy in the Panhandle and Lower Rio Grande Valley compared to other regions in Texas. It is safe to assume that agricultural workers at risk of exposure may be found in all farming areas. No accurate estimate of farm workers potentially exposed to pesticides is, however, available.

Control

Measures to control acute occupational pesticide poisoning in Texas include the EPA's registration of agricultural chemicals and use restrictions (31), Texas Department of Agriculture's (TDA) recently adopted rules governing agricultural pesticide use (32), criteria set by the Occupational Safety and Health Administration (OSHA) (33) and recommendations from the National Institute for Occupational Safety and Health (NIOSH) for pesticide manufacturing and formulating establishments (14), and the Texas Department of Health's recent requirement for the reporting of cases of occupational diseases including acute occupational pesticide poisoning (34).

The EPA is responsible for the registration and regulation of pesticide use in the US (31). Enforcement of these regulations and investigation of exposure incidents have been delegated to state departments of agriculture. In 1984, the TDA adopted rules "to establish pesticide application standards designed to prevent unreasonable risk to human health and protect workers and others during the production of agricultural field crops" (32). These rules include standards for worker re-entry into fields. Re-entry intervals are established for pesticides when used on crops which require workers to do labor-intensive activities (eg, planting, thinning, detasseling, sucker removal, pruning, and harvesting). Re-entry interval refers to restricting field access for a period of time after pesticide application (35). Fig 2 shows the re-entry intervals adopted by the TDA. Rules also cover the posting of fields and the necessary procedures, if for some reason workers need to enter the field before the re-entry interval expires. Farm operators are required to know or have access to the trade and chemical name of any pesticide being used and must make this information available to workers, to persons alleging pesticide exposure, and to attendant medical personnel upon request.

For the workplace environment of pesticide manufacturing and formulating establishments, OSHA has established permissible exposure limits (PEL) for many pesticides (33). In its criteria document on manufacture and formulation of pesticides, NIOSH has emphasized work practices, engineering controls, and medical surveillance programs to protect workers from the adverse effects of pesticide exposure. Recommendations for medical surveillance include an initial medical examination, preexposure and periodic erythrocyte cholinesterase levels for workers exposed to cholinesterase-inhibitors, and periodic medical examinations (at least annually) (14).

Reporting acute occupational pesticide poisoning

Acute occupational pesticide poisoning is reportable in accordance with Texas Board of Health rules issued under the 1985 Texas Occupational Disease Reporting Law (34). Physicians and directors of laboratories with case reports must provide the name, address, age, sex, and race of the patient and the diagnosis and date of diagnosis to the director of their local or regional health department, who will then transmit this information to the Texas Department of Health, Epidemiology Division, in Austin. Reports may also be made directly to the Texas Department of Health, Epidemiology Division, by telephoning (toll-free) 1–800–252–8239. The Occupational Disease Reporting Act provides for the confidential handling of all case reports by local and state health departments, and information about individual cases will be held as confidential medical records.

Follow-up investigations are conducted by the TDH epidemiology staff to verify the diagnosis, determine the source of the causative agent, obtain an

*2. Re-entry intervals for pesticides used on crops requiring workers to perform labor-intensive activities in Texas.**

Re-entry level	Pesticide
24 hours	Any pesticide with registered agricultural uses when used on crops requiring workers to perform labor-intensive activities, unless the pesticide has been granted an exemption. (As of April 4, 1986, six formulations of Dipel are exempt).
48 hours	azinphos-methyl (Guthion) carbophenothion demeton dicrotophos disulfoton endosulfan endrin ethion methidathion methyl parathion mevinphos monocrotophos oxydemeton-methyl phorate phosphamidon
7 days	ethyl parathion

* Texas Department of Agriculture. Reentry Intervals [Texas Register, Jan 1, 1985 (10 Tex Reg 39-401).] Notice of Exemption from Interim 24-hour Reentry Interval established for Agricultural Pesticides [Texas Register, Apr 4, 1986 (11 Tex Reg 1164).]

occupational and employment history, and seek related but unreported cases. As necessary, worksite investigations will be undertaken to determine whether an ongoing hazard is present and to make appropriate recommendations to eliminate the hazard. The purpose of this reporting system is to determine the epidemiology of acute occupational pesticide poisoning in Texas, and through identification of hazardous workplaces, pesticides, and practices, enable prevention of future cases.

Conclusion

Little is known about the incidence of acute occupational pesticide poisoning in Texas. Much of our understanding about this occupational problem has evolved from reports received through the reporting system in California. Yet, differences in climate, soil, topography, crops grown, and pests influence the use, location, quantity, nature, availability, toxicity, and persistence of pesticide residues (36). Understanding and control of pesticide-related health problems in Texas require active surveillance. Implementation of the new Occupational Disease Reporting Law will help us understand the frequency and distribution of cases and enable prevention of further incidents. The participation of Texas physicians by recognizing and reporting cases of acute occupational pesticide poisoning to the TDH is essential to this task.

REFERENCES

1. Moses M: Pesticides, in Rom WN (ed): Environmental and Occupational Medicine. Boston, Little Brown and Company, 1983, pp 547–571.
2. Kilgore WW, Akesson NB: Minimizing occupational exposure to pesticides: populations at exposure risk. Residue Rev 75:21–31, 1980.
3. Jackson RJ: Pesticides as a public health concern in California. West J Med 139(3):363–364, 1983.
4. California Department of Food and Agriculture. Summary of Report from Physicians of Illnesses that were Possibly Related to Pesticide Exposure During the Period January 1- December 31, 1985 in California. Unpublished report. Sacramento, Calif, Feb 18, 1986.
5. Griffith J, Duncan RC: Grower reported pesticide poisonings among Florida citrus fieldworkers. J Environ Sci Health B20(1):61–72, 1985.
6. Griffith J, Duncan RC, Konefal J: Pesticide poisonings reported by Florida citrus fieldworkers. J Environ Sci Health B20(6):701–727, 1985.
7. Morgan DP, Lin LI, Saikaly HH: Morbidity and mortality in workers occupationally exposed to pesticides. Arch Environ Contam Toxicol 9(3):349–382, 1980.
8. Hayes WJ Jr, Vaughn WK: Mortality from pesticides in the United States in 1973 and 1974. Toxicol Appl Pharmacol 42(2):235–252, 1977.
9. Reich GA, Gallaher GL, Wiseman JS: Characteristics of pesticide poisoning in South Texas. Tex Med 64(9): 56–58, 1968.
10. Hatcher RL, Wiseman JS: Pesticide poisoning. Epidemiology of pesticide poisoning in the Lower Rio Grande Valley in 1968. Tex Med 65(8):40–43, 1969.
11. Hayes WJ Jr: Pesticides Studied in Man. Baltimore, Williams and Wilkins, 1982.
12. Morgan DP: Recognition and Management of Pesticide Poisonings, 3rd ed. Washington, DC, US Government Printing Office, 1982.
13. Gunther FA, Iwata Y, Carman GE, et al: The citrus problem: research on its causes and effects, and approaches to its minimization (Review article). Residue Rev 67:1–32, 1977.
14. National Institute for Occupational Safety and Health. Occupational Exposure During the Manufacturer and Formulation of Pesticides: Criteria for a Recommended Standard. DHEW Pub no (NIOSH) 78–174. Washington, DC, US Government Printing Office, 1978.
15. Shah PV, Monroe RJ, Guthrie FE: Comparative rates of dermal penetration of insecticides in mice. Toxicol Appl Pharmacol 59(3):414–423, 1981.
16. Aldridge WN: The nature of the reaction of organophosphorus compounds and carbamates with esterases. Bull WHO 44(1):25–30, 1971.
17. Taylor P: Anticholinesterase agents, in Gilman AG, Goodman LS, Gilman A (eds): Pharmacological Basis of Therapeutics, 6th ed. New York, MacMillan Publishing Co, Inc, 1980, pp 100–119.
18. Murphy SD: Pesticides, in Casarett LS, Doull J (eds): The Basic Science of Poisons. New York, MacMillan Publishing Company, 1980, pp 357–408.
19. Namba T: Cholinesterase inhibition by organophosphorus compounds and its clinical effects (Review article). Bull WHO 44(1):289–307, 1971.
20. Namba T, Nolte CT, Jackrel J, et al: Poisoning due to organophosphate insecticides. Acute and chronic manifestations. Am J Med 50(4):475–492, 1971.
21. Kipling RM, Cruickshank AN: Organophosphate insecticide poisoning. Anaesthesia 40(3):281–284, 1985.
22. Coye MJ, Lowe JA, Maddy KT: Biological monitoring of agricultural workers exposed to pesticides. I. Cholinesterase activity determinations. J Occup Med 28(8): 619–627, 1986.
23. Vandekar M: Minimizing occupational exposure to pesticides: cholinesterase determination and organophosphorus poisoning. Residue Rev 75:67–80, 1980.
24. Midtling JE, Barnett PG, Coye MJ, et al: Clinical management of fieldworker organophosphate poisoning. West J Med 142(4):514–518, 1985.
25. Hine CH: Methyl bromide poisoning. A review of ten cases. J Occup Med 11(1):1–10, 1969.
26. Wilson R, Lovejoy FH, Jaeger RJ, et al: Acute phosphine poisoning aboard a grain freighter. Epidemiologic, clinical and pathological findings. JAMA 244(2):148–150, 1980.
27. Comer SW, Staiff DC, Armstrong JF, et al: Exposure of workers to carbaryl. Bull Environ Contam Toxicol 13(4):385–391, 1975.
28. Lee MH, Ransdell JF: A farmworker death due to pesticide toxicity: a case report. J Toxicol Environ Health 14(2–3):239–246, 1984.
29. US Department of Commerce. 1982 Census of Manufacturers, vol 1. Geographic Area Series-Texas. Washington, DC, US Government Printing Office, May, 1985.
30. US Department of Commerce. 1982 Census of Agriculture, vol 1. Geographic Area Series-Texas. Washington, DC, US Government Printing Office, Sept, 1984.

31. Coye MJ: The health effects of agricultural production. I. The health of agricultural workers. J Public Health Policy 6(3):349–370, 1985.
32. Texas Administrative Code, title 4, chapter 7. Pesticides (4 TAC 7.25–7.31).
33. National Institute for Occupational Safety and Health, United States. Occupational Safety and Health Administration. Stricoff RS, Partridge LJ, Mackison FW (eds): Occupational Health Guidelines for Chemical Hazards. DHHS (NIOSH) Publication no 81–123, Jan 1981, Washington, DC, US Government Printing Office.
34. Texas Administrative Code, title 25, chapter 99. Occupational Diseases Reporting (*25 TAC 99.1).
35. Popendorf WJ, Leffingwell JT: Regulating OP pesticide residues for farmworker protection (Review article). Residue Rev 82:125–201, 1982.
36. Paynter OF: Worker reentry safety. III. Viewpoint and program of the Environmental Protection Agency. Residue Rev 62:13–19, 1976.

For additional resources on this topic, see the MORE ON THE SUBJECTS department in this issue.