Chapter 10

Case Studies: Exposure to Lead, Pesticides in Agriculture, and Organic Solvents in the Workplace

"If we were to judge the interest excited by any medical subject by the number of writings to which it has given birth, we could not but regard the poisoning by lead as the most important to be known of all those that have been treated up to the present time. "

M.P. Orfila
A General System of Toxicology
1817

"*mere* is . . . no systematic monitoring of the health or exposure to pesticides of the more than 2 million farmworkers, applicators, harvesters, irrigators, and field hands who work around pesticides. Industrial workers who produce these pesticides receive the benefits of such monitoring. "

National Academy of Sciences
Alternative Agriculture
1989

"When I was in the Navy, I remember my commanding officer called me in and he was very upset because an air control operator had abandoned the tower, his position of duty, with seven aircraft stacked up calling for landing instructions. I was supposed to examine him. As I look back, I completely missed what was happening until years later. He was working in his off hours loading pesticides into spray planes, which caused a tremendous change in his personality and his behavior and his ability to cope. "

Gordon Baker, M.D.
Testimony before the Committee on Environment and Public Works
U.S. Senate
March 6, 1989

"..+ doctors tell me my nervous system has been heavily damaged, my brain has been damaged, and I suffer chemically induced asthma. I also have kidney, liver, and vision difficulties. I had a tumor removed from my eyes less than 1 year ago, and have been told that I have more, not to mention the chronic muscle pains throughout my body . . . . Throughout my entire 8 years at this truck manufacturing company, I was never informed of the hazards of the solvents I used, None of these products were adequately, clearly, or should I say, truthfully labeled. Yet the hazards for most of the products had been known for years by the chemical manufacturers and other people.

Frank Carsner
Testimony before the Committee on Science and Technology
U.S. House of Representatives
October 8, 1985

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INTRODUCTION

The best way of illustrating the adverse effects of toxic substances on the nervous system is by looking at substances or classes of substances that are known to be neurotoxic. These case studies discuss attempts to control human exposure to lead, pesticides, and organic solvents. They illustrate the prevalence of neurotoxic substances, the susceptibilities of certain subpopulations, special hazards in occupational settings, and how Federal agencies address these concerns.

As it exists in the earth, lead is bound in chemical compounds and presents little risk to humans. As it is mined and utilized, however, it is distributed throughout the environment, presenting a risk to the entire population, but especially children, who are most vulnerable to its effects. Research shows that children are directly exposed to multiple sources of lead, are more sensitive to exposure, and suffer worse effects than adults. A great deal of progress has been made by Federal agencies in reducing public exposure by regulating the lead contents of paint, gasoline, plumbing systems, and food containers, but lead poisoning continues to be a major national health problem.

Chemical pesticides also present a significant risk to the population as a whole, but especially to agricultural workers and others who apply them or work close to them. Several Federal agencies have regulations that are intended to protect these workers from pesticide poisoning, but critics argue that more could be done. Many States have their own regulations, some of which are more stringent than Federal regulations, especially in protecting farmworkers. This chapter reviews the different types of pesticides in use and summarizes what is known about their neurotoxic effects.

Many solvents are neurotoxic and threaten the health of the industrial workers who come in contact with them. Solvents may cause a variety of functional changes, ranging from temporary memory loss to unconsciousness, depending on the duration and extent of exposure; major structural changes in the nervous system may also result. Engineering controls to avoid contamination, isolation of workers, and issuance of protective equipment to workers are some of the preventive measures currently in use. This chapter gives examples of how various solvents have been regulated under the Occupational Health and Safety Act, including the new standards for worker protection proposed by the Occupational Health and Safety Administration in 1988. It discusses criticisms of the existing regulations and offers suggestions as to how they might be improved.

EXPOSURE TO LEAD

As discussed in previous chapters, regulation of neurotoxic substances is a two-part process, one being identification of new hazardous chemicals and prevention of human exposure to them, the other being reduction of exposure to existing toxic substances. Lead is a prominent example of a substance long known to be toxic to the human nervous system (see box 1O-A). Unlike some elements, such as sodium or zinc, lead serves no useful biological purpose; since the body can neither use nor metabolize it, lead accumulates in body tissues, especially bones and teeth. Debate continues as to what maximum level is tolerable, although the only way to prevent any toxic accumulation is to limit exposure to zero. This chapter highlights some of the difficulties of removing or preventing exposure to a neurotoxic substance that has been extensively used in industry and therefore is especially prevalent in the environment.

Efforts to reduce public exposure to lead by removing current sources and preventing new ones have been undertaken by several Federal agencies. The Environmental Protection Agency (EPA) and the Food and Drug Administration (FDA) have taken steps to reduce the amount of lead in gasoline and food, and EPA is currently considering more stringent methods for controlling exposure to lead from drinking water. Other sources of lead, however, are more difficult to control. Lead has been used consistently in industrial and commercial activities and, despite awareness of its inherent dangers, continues to be used in product manufacturing. The use of lead in manufacturing ultimately results in its distribution in the environment in the form of waste.

Box 10-A—Lead: A Historical Perspective

Lead is the oldest, most extensively studied, and probably most ubiquitous neurotoxic substance. It is mentioned in ancient Egyptian manuscripts and was used by the Egyptians as a cosmetic; both the Egyptians and the Remans used lead in cooking tools and vessels. The Remans used it as a sweetener and preservative in wines and eiders; lead acetate is often called "sugar of lead" because of its sweet taste. The Remans also used lead in building houses and transporting water. In fact, the words plumber and plumbing originate from the Latin word for lead, plumbum. Lead was mined in Great Britain as far back as the reign of Julius Caesar. Remnants of these mines contaminate local farms and gardens today.

At least some of the toxic effects of lead were known early on. The Greek thinker Dioscorides stated in the 2nd century B.C. that "Lead makes the mind give way.' Pliny the Elder cautioned that inhaling the fumes of molten lead was dangerous (although he continued to recommend that it be used in making wine). Indeed, the continued use of lead, despite recognition of its dangers, has caused many outbreaks of lead poisoning over time. Benjamin Franklin may have been the first person to recognize lead as an occupational hazard: in a letter about lead poisoning he wrote, "How long a useful truth may be known and exist, before it is generally receiv'd and practis'd on."

SOURCES: A. Fischbein, "Environmental and Occupational Lead Exposure, " *Environmental and Occupational Medicine,* W.N. Rom (cd.) (Boston, MA: Little, Brown, 1983); J.S. Lin-Fu, "Lead Poisoning and Undue Lead Exposure in Children: History and current Status,' *Low Level* Lead *Exposure: The Clinical Implications of Current* Research, H.L. Needleman (cd.) (New York, NY: Raven Press, 1980); R.H. Major, ''Some Landmarks in the History of Lead Poisoning, " *Annals of Medical History* 3:218-227, *1931;* H.L. Needleman and D. Bellinger, "The Developmental Consequences of Childhood Exposure to Lead: Recent Studies and Methodological Issues,' *Advances in Clinical Child Psychology, vol. 7,* B.B. Lahey and A.E. Kazdin (eds.) (New York, NY: Plenum Press, 1984); H. Waldron, ''Lead Poisoning in the Ancient World," *Medical History* 17:391-398, *1973.*

For example, lead is found in commodities such as solders, batteries, and paint, but it is also present in dust and soil as waste material. There is no agreement as to who bears responsibility for removing the various forms of lead from the environment. Although the Consumer Product Safety Commission has reduced the amount of lead permitted in paint to prevent future exposure, the danger of lead poisoning from leaded paint in old housing remains.

In addition to the remedial measures being taken, preventive measures must be considered for some currently minor sources that may become larger problems in the future, Incinerators, for example, may significantly increase exposure to lead in the environment as we attempt to reduce our reliance on landfills.

Sources of Exposure

Lead exists in both organic and inorganic forms. Although organic lead is more toxic than inorganic lead because it degrades quickly in the atmosphere and the body, it constitutes only a small proportion of the total lead to which the population is exposed (16). Organic lead is most commonly found as a fuel additive and can reach significant levels in heavy traffic areas and underground garages (16), but it is rapidly converted to the inorganic form. This chapter will therefore focus on inorganic lead. Significant sources of exposure to inorganic lead include water, food, soil, lead-based paint, leaded gasoline, and industrial emissions (see table 10-1).

Levels and sources of exposure vary according to surroundings. In remote areas, proximity to stationary sources of lead such as smelters maybe the main source of exposure, whereas in older cities leaded paint may be the most common source (165). Individuals living near industrial sources of lead, people who drink contaminated water, adults with occupational exposure, and children who ingest lead-contaminated paint, soil, or dust have the greatest exposure to lead (109,172).

When discussing exposure to lead, a distinction is often made between children and adults, since children both ingest and inhale more lead per unit of body weight than adults and are more vulnerable to its effects (165). Children, given their normal tendency to put things in their mouths, are likely to ingest paint, soil, or dust, all of which are potential sources of lead. Lead gives paint a sweet taste, increasing its appeal for children. Children also have a higher absorption rate of ingested lead than adults: whereas adults absorb between 5 and 15 percent of ingested lead and usually retain less than 5 percent of what is absorbed, one study found that infants on regular diets absorb an average of over 40 percent of ingested lead and retain over 30 percent

Table 10-l Significant Sources of Exposure to Lead

Leaded paint
. lead released into the air through destruction and weathering of structures painted with leaded paint
• lead ingested by children from household dust, less commonly by eating leaded paint chips

Leaded gasoline
. lead released into the air in exhaust fumes
. lead released into the air during fueling

Stationary sources
. lead released into the air by industrial activity, e.g., smelting, refining, and battery recycling
. occupational exposure of factory workers, exposure of children to lead on the clothing of parents

Dust, soil
. paint
. industrial activity
. gasoline

Water, plumbing
. **lead in water** source
. leaching from lead pipes
. leaching from lead solder
. leaching from brass or bronze

Food
. lead contained in food items from contaminated water or soil
. lead-soldered food cans
• lead deposited on crops from automobile exhaust
. lead deposited on crops from industrial activity
• lead contamination during food processing
. lead glazes in dishes and pottery

SOURCE: Office of Technology Assessment, 1990.

of that amount (57). Children also retain more of the lead they absorb than do adults, since lead in blood is stored in growing bones (165). The effects of lead on children are more severe than the effects of lead on adults: children have less bone tissue in which lead can be stored, and thus lead remains in the bloodstream, free to exert toxic effects on various organs of the body. Nutritional deficiencies, more likely to occur in the growing child, can also contribute to higher absorption levels of lead (165,169). Children's nervous systems, especially their blood-brain barriers, are not yet fully developed, and the same cellular lead exposure may produce disproportionate results in children compared to adults (84,145). Also, cognitive effects occur at lower levels in children. For similar reasons, fetuses may be even more vulnerable to lead's toxicity than children (84). There is some evidence that lead stored in women's bones from previous exposure may be mobilized during pregnancy and lactation, and thus expose the fetus and infant through the placenta and breast milk (148).

Estimates of the number of children exposed to lead, listed by source, are found in table 10-2. The

Table 10-2-Estimated Number of Children Exposed to Sources of Lead

Source	Number of children[a] (millions)
Leaded paint	12.00
Leaded gasoline	5.60
Stationary sources	0.23
Dust, soil	5.90-11.00
Water, plumbing	10.40
Food 0	1.00

[a]Numbers in the table are not additive since children are usually exposed to multiple sources of lead in the environment.

SOURCE: U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry and Center for Environmental Health and Injury Control, "Childhood Lead Poisoning—United States: Report to the Congress by the Agency for Toxic Substances and Disease Registry," *Morbidity and Mortality Weekly Report* 37:481-485, 1988.

type and availability of data for each of these sources vary considerably, therefore the estimates are not comparable and cannot be used to rank the severity of the problem by source of exposure (165).

For adults, the workplace is a major source of exposure. The National Institute for Occupational Safety and Health has listed 113 occupations that potentially increase workers' exposure to inorganic lead (74). In adults not exposed to occupational sources of lead and in children older than 6 to 8 years, food and water are most likely to be the major sources (74). For most adults, lead in the air is no longer as significant a source of exposure as lead in the diet, but as one study found, levels of lead in the blood of adults remain correlated with levels of lead in air (74), as do levels of lead in children's blood (15). Before the phase-out of lead from gasoline, however, airborne lead was the predominant source of exposure to lead for adults and children (6,173, 175).

Routes of Exposure

Lead can enter the human body through three routes: inhalation, ingestion, or absorption through the skin, although the latter is significant only for organic compounds of lead (51). Intake through inhalation depends on particle size and volubility in body fluids (51). Gastrointestinal absorption is influenced by a number of factors, primarily age and nutritional status (72). The proportion of lead absorbed through ingestion and inhalation differs by age and principal source of exposure, as discussed earlier.

Levels of Exposure

Lead is stored in the circulating blood, soft tissue, and bone. Because it has a long biological half-life and is only slowly excreted from bone, lead can accumulate in the body. Thus, the concentration of lead in the blood (the blood lead level) is not an accurate indicator of total exposure to lead, only of recent exposure. The amount of lead found in teeth and bones is a more useful indicator of cumulative exposure, but it yields no information about the time or duration of exposure, nor of current exposure. Furthermore, teeth are easily obtained only from young children, who lose their baby teeth. A technique using X-ray fluoroscope was developed in 1984 to measure lead in bone (28,77); its feasibility as a testing method is being evaluated (186).

For the most part, neurological deficits in adults have not been noted below a blood lead level of 40 micrograms of lead per deciliter of blood (ug/dl) (192), although elevations in blood pressure have been noted at 5 ug/dl (125, 126, 144). In children, however, adverse neurological effects are seen at much lower levels (33,87,165), and since 1943 the blood lead level found to be associated with neurobehavioral dysfunction has steadily decreased. Before that year, the cumulative effects of

Photo credit: U.S. Environmental Protection Agency

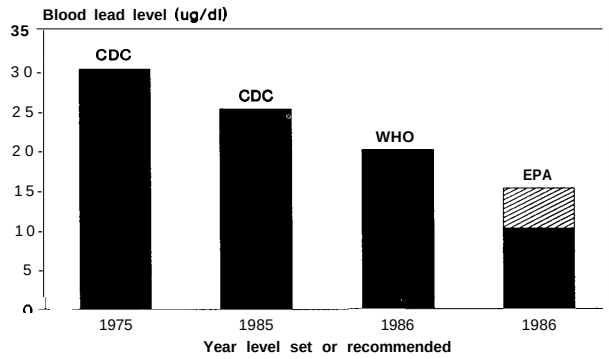
EPA has estimated that exposure to lead in drinking water is keeping more than 240,000 children from realizing their full intellectual potential.

lead poisoning went unrecognized, and physicians generally believed that if a child did not die of lead poisoning there would be no lasting effects (1 18). In 1943, however, researchers found that a group of children with mild lead poisoning in infancy did not progress satisfactorily in school, and they suggested that lead poisoning early in life might be widespread (22). Since then, the aggregate effects of lead poisoning have been recognized and its long-term effects have been studied. Researchers have correlated blood lead levels with neurobehavioral dysfunction.

Before the 1960s a blood lead level below 60 ug/dl was not considered dangerous (169). In 1975, the Centers for Disease Control (CDC) lowered the acceptable level for children to 30 ug/dl, and in 1985 it lowered the level again, to 25 ug/dl (169). The World Health Organization (WHO), in a 1986 report, stated that 20 ug/dl was the upper acceptable level (193). EPA% Clean Air Scientific Advisory Committee associated lead levels of 10 to 15 ug/dl and possibly lower with adverse effects (see figure 10-1) (172) and recommended 10 to 15 ug/dl as the maximum acceptable level. Recently, subtle deficits in neurobehavioral performance have been reported in fetuses and newborn babies exposed to low levels of lead (12,33,87,121,165).

In 1986, Congress requested that the Agency for Toxic Substances and Disease Registry (ATSDR) prepare a report on lead poisoning in children. One of the report's mandates was to estimate the total number of children exposed to potentially hazardous concentrations of lead. Approximately 2.4 million U.S. children age 6 months to 5 years living in Standard Metropolitan Statistical Areas (SMSAs) (or 17.2 percent) have blood lead levels greater than 15 ug/dl; 200,000 (1.5 percent) have blood lead levels greater than 25 ug/dl. No economic stratum of children was found to be free from the potential health risk of lead poisoning. However, since the data covered only black and white children, no reliable prevalence rates could be calculated for Hispanic children and children of all other races; further, since SMSAs include only about 80 percent of the children in the United States, the actual number of children with blood lead levels above 15 ug/dl may be higher than the ATSDR report indicates: more likely estimates are between 3 and 4 million affected children (21.4 to 28.6 percent) (165). The CDC is considering lowering its target level for medical intervention again.

Figure 10-l- Children's Blood Lead Levels Considered Acceptable by Various Agencies



= 10 to 15 ug\dl is the recommended maximum acceptable level.

SOURCE: Office of Technology Assessment, 1990.

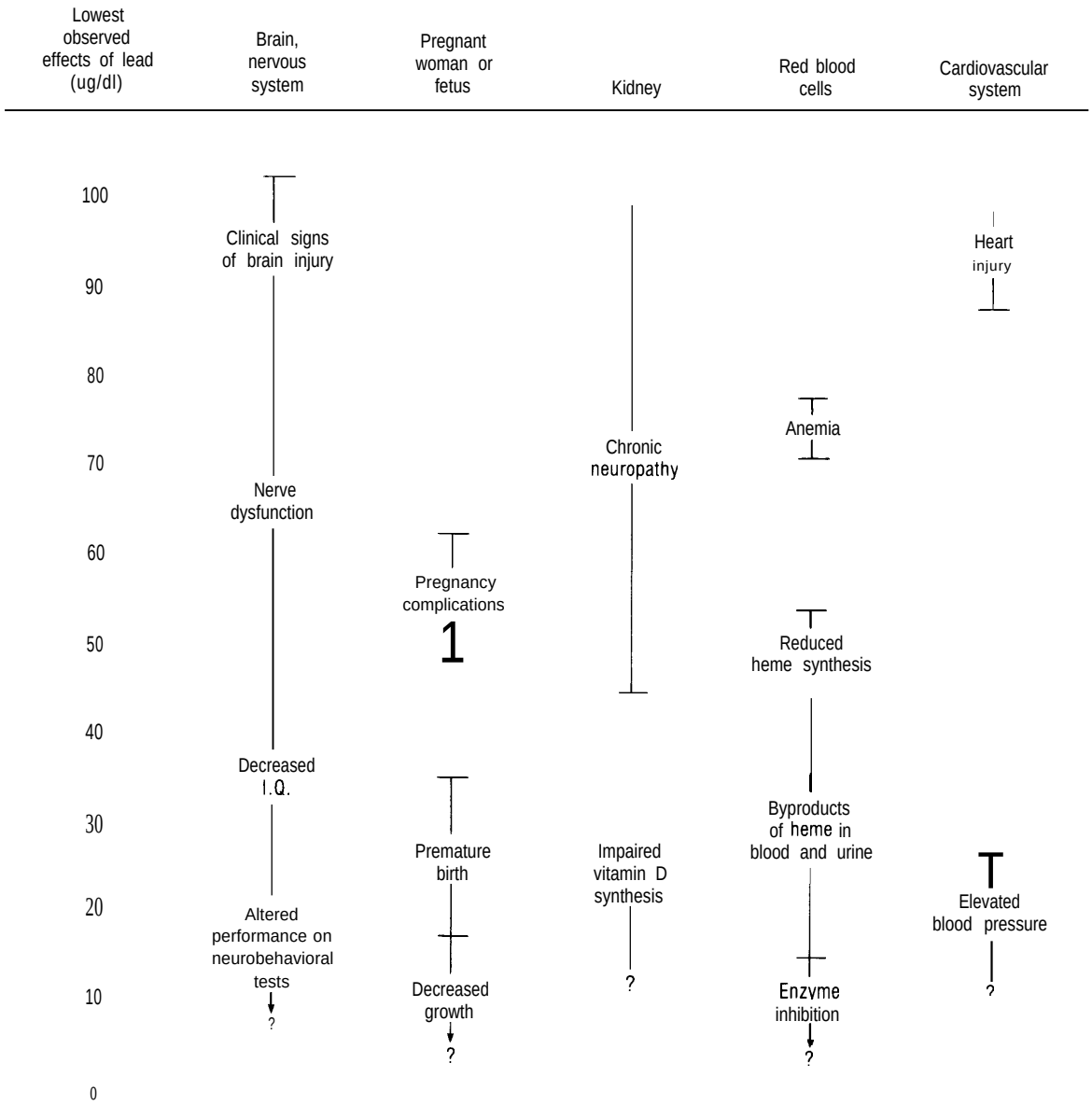
It is significant that some of the studies on children have not detected a threshold for adverse effects of lead (87,117,123), indicating that as tests for various impairments become more sensitive, the level at which adverse effects are observed may decrease further. Accurate, current information as to the lowest blood lead levels associated with neurotoxic effects is crucial for policymaking, since the regulations that set safety levels at 25 ug/dl do not adequately protect the many children whose blood lead levels fall below that; these children may be endangered at levels of 10 **to 15** ug/dl, or possibly lower.

Effects of Lead on the Human Body

Lead causes numerous adverse health effects. A summary of some observable effects and the blood lead levels with which they have been correlated is given in figure 10-2. In children, brain damage resulting from exposure to lead can range in severity from inhibited muscular coordination to stupor, coma, and convulsions at high levels (72). Acute brain damage is rare in adults; when it appears it is usually a result of high exposures to lead and is often accompanied by other factors, such as alcoholism. High exposures to lead can also damage the peripheral nervous system.

Since the discovery of chelation treatment, which removes lead from the blood, mortality from acute lead poisoning has declined. Yet as our ability to

Figure 10-2-Adverse Health Effects of Lead



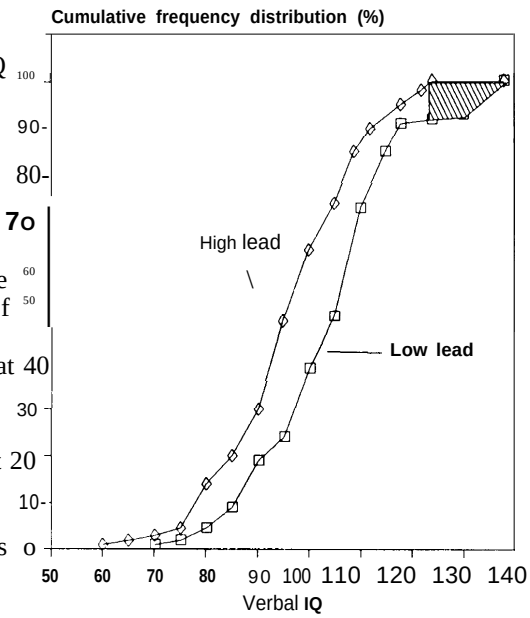
SOURCE: U.S. Environmental Protection Agency, "Drinking Water Regulations; Maximum Contaminant Level Goals and National Primary Drinking Water Regulations for Lead and Copper; Proposed Rules" (53 FR 31565), 1966.

detect subtle neurological deficits has improved, estimates of morbidity have increased. Effects of permanent damage to the central nervous system—for example, mental retardation, hyperactivity, seizures, optic atrophy, sensory-motor deficits, and behavioral dysfunctions-have been observed (see box 10-B). There is also some recent evidence that lead may cause minor hearing impairments (146). Chronic low-level exposure may ultimately be more damaging than acute exposure that is treated immediately (21).

Factors such as genetic variation in susceptibility, nutritional status, behavior, and age may alter an individual's vulnerability to lead poisoning (1 18). Most of these factors affect toxicity by altering the absorption of lead.

Box IO-B—Lead Poisoning and IQ

A study in 1979 found that children exposed to lead had intellectual, attentional, and behavioral deficits, It also found a difference of about 5 points in the mean IQ (intelligence quotient) of children with elevated lead levels and those with low lead levels. While this number is statistically significant, some question was raised as to whether it was biologically significant.



As the figure shows, the significance of this difference in **IQs** shows up most clearly at the ends of the IQ spectrum. Children with elevated lead levels were three times more likely to have a verbal IQ below 80; furthermore, none of them had superior IQ scores (greater than 125), while 5 percent of the children with low lead levels had scores in that range.

A follow-up study published in January of 1990 concluded that the effects of lead exposure upon cognitive development in early years persist into early adulthood. In this study, children who were originally examined in the first grade were reexamined as high school students. The subjects underwent extensive neurobehavioral analysis using a variety of tests for hand-eye coordination, grammatical reasoning, and reaction times. Deficits in central nervous system functioning resulted in poorer classroom performance, reduced vocabulary and reasoning scores, and higher absentee rates in school.

SOURCES: H.L. Needleman, C. Gunnoe, A. Leviton, et al., "Deficits in Psychologic and Classroom Performance of Children With Elevated Dentine Lead Levels," *New England Journal of Medicine* 300:689-695, *1979;* H.L. Needleman, A. Leviton, and D. Bellinger, "Lead-Associated Intellectual Deficit," New *EnglandJournal of Medicine* 306:367, *1982;* B. Weiss and T.W. ClarkSon, "Toxic Chemical Disasters and the Implications of Bhopal for Technology Transfer," *The Milbank Quarterly* 64:216, *1986.* H.L. Needleman, A. Schell, D. Bellinger, et al., "The Long Term Effects of Exposure to Low Doses of Lead in Childhood,' New *England Journal of Medicine* 322(2):83-88, *1990.*

Regulatory Activity Regarding Exposures to Lead

Action by Congress and various executive agencies has led to a reduction in exposure to lead in the United States. Their response marks the first time that specific neurobehavioral effects of a toxic substance were considered in determining regulatory policy. Although progress has been made, there is evidence that lead poisoning in the United States still occurs in epidemic proportions.

Lead in the Air

Removing lead from the air is the responsibility of EPA, whose statutory authority comes from the Clean Air Act, passed in 1970 and amended in 1977. The two major sources of lead in the air are leaded gasoline and stationary sources, such as lead smelt-*ers.*

In 1978, EPA promulgated regulations stating that the level of lead in the air must not exceed 1.5 micrograms per cubic meter (ug/m^3). Under the Clean Air Act, the States had to take steps to meet that standard by 1982. The standard includes contributions from both automobiles and industrial sources and was designed to prevent children from being exposed to concentrations of lead in the air that could lead to blood lead levels of more than 30 ug/dl (96).[1]

In 1973, EPA promulgated regulations requiring that major gasoline dealers sell at least one grade of "unleaded" gasoline (defined as containing no more than 0.05 gram of lead per gallon of gasoline).

[1]The recommended maximum for children's blood lead levels has been repeatedly revised: EPA Science Advisory Board established 10 to 15 ug/dl and possibly lower as the blood lead level of concern in 1986 (173).

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Lead in Food

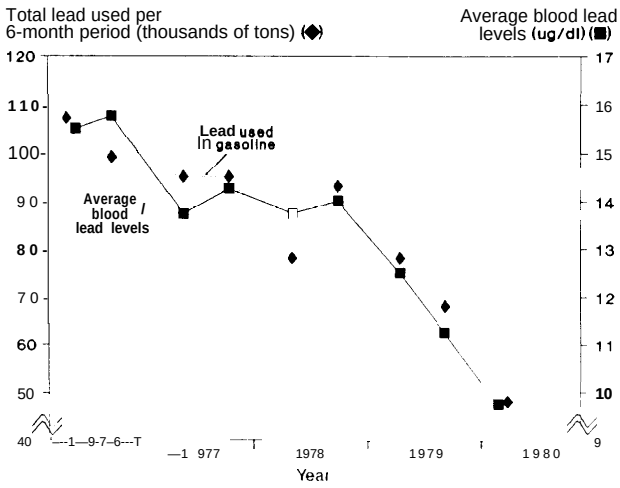
Regulation of lead in food is the responsibility of FDA. Although the agency has set acceptable levels of lead for pesticides and food utensils in domestically produced food, much of its activity has focused on eliciting voluntary cooperation from domestic food manufacturers and processors (165). The success of this effort is illustrated in figure 10-4.

Regulation of lead by FDA began in the 1930s, when the agency established guidelines for limits on the use of lead in pesticides.[2] The next item of concern was lead in canned evaporated milk. In 1974, the agency proposed a tolerance level of 0.30 part of lead per million parts of milk (ppm) (39 FR

Photo credit: National Archives

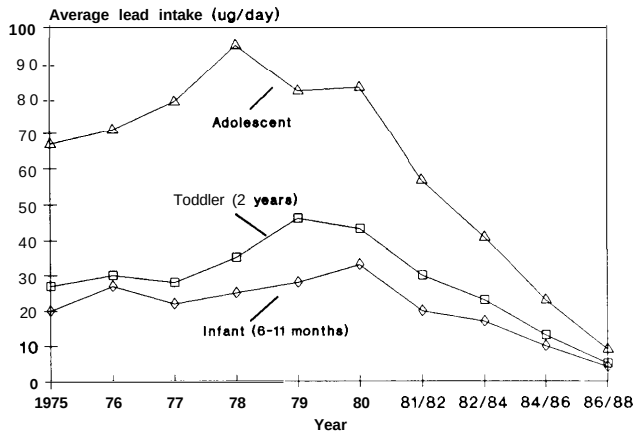
[2]Current permissible levels of lead in pesticides are 1 microgram per gram (ug/g) on citrus fruits and 7 ug/g on other fruits and vegetables.

Figure 10-3-Lead Used in Gasoline Production and Average Blood Lead Levels



SOURCE: J. Schwartz, H. Pitcher, R. Levin, et al., Costs *and Benefits of Reducing Lead in Gasoline: Final Regulatory Impact Analysis, EPA-230-05-85-O06* (Washington, DC: U.S. Government Printing Office, 1985).

Figure 10-4-Dietary Lead Intake



SOURCE: U.S. Department of Health and Human Services, Public Health Service, Food and Drug Administration, Center for Food Safety and Applied Nutrition, *FDA Total Diet Study* (Washington, DC: 1989).

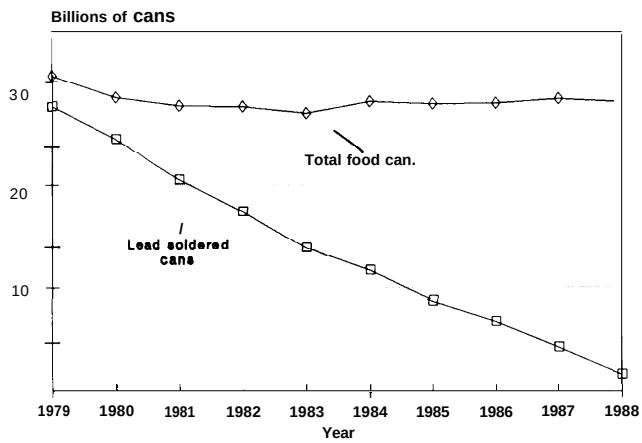
42745). As a result of its own studies and FDA's recommendations, the milk industry reduced the levels of lead in evaporated milk from 0.52 ppm in 1972 to 0.08 ppm in 1982 (30,109). Manufacturers of infant juices also took steps to lower lead levels in their products, eventually switching voluntarily from tin cans to glass jars (109), as did manufacturers of canned infant formula, who switched from lead-soldered cans to other types of cans (96).

There has been a significant decrease in the use of lead solder for food cans manufactured in the United States. In 1979, more than 90 percent of such cans contained lead solder; by 1989, less than 4 percent did. Figure 10-5 demonstrates the trend in reducing lead solder in cans and reflects the can manufacturing industry's plans to eliminate lead solder in all domestically produced food cans in the next 2 to 3 years (24). The number of imported cans containing lead solder is not known but maybe large (165).

Materials used for packing food have also been a source of concern. These materials are considered indirect food additives, because contaminants may migrate from packaging materials into the food. As of 1980, three indirect food additives were subject to limitations on the amount of lead they can contain (109).

Regulations concerning lead used in food utensils, specifically ceramic and hollowware products, have been promulgated by FDA (54 FR 23485). Large containers (in which food is likely to be stored) and cups used by children have lower limits on permissible lead content than do small utensils (100,109). FDA is currently considering lowering the acceptable limit for large containers. Although these limits apply to both imported and domestic utensils, few imported utensils are tested for lead content. In response to public concern, some retailers are testing imported dishes on their own (100, 182).

Figure 10-5-Food Can Shipments



SOURCE: Can Manufacturers Institute, Washington, DC, personal communication, 1989.

Occupational Exposure to Lead

In contrast to the reduction of lead in food, where strict regulations have not had to be imposed by government, the reduction of occupational exposure to lead has required more intervention. In response to the Occupational Safety and Health Act of 1970, the Occupational Safety and Health Administration (OSHA) promulgated regulations in 1978 (29 CFR 1910.1025) that set a maximum permissible level for lead in the air inhaled by workers.[3]

The lead industries immediately sued OSHA, challenging the validity of the standard. In 1980, the U.S. Court of Appeals for the District of Columbia Circuit upheld the limit and most other provisions of the regulation but ordered that the feasibility of engineering controls be reconsidered for many affected industries (180). OSHA states explicitly that industries must use engineering controls to reduce the overall level of lead in the air at the workplace, as opposed to simply giving workers respirators to remove lead from the air they inhale. The court instructed OSHA to reassess the feasibility of such engineering and work controls for approximately 40 industries. Only one of these studies has not been completed; however, because the courts will reexamine all the studies at once, these 40 industries are currently exempt from the requirement to achieve 50 ug/cm^3 through engineering and work practice controls.

The regulatory framework for ensuring minimal occupational exposure to lead is in place. Occupational exposure has been reduced considerably in most large industries, as indicated by decreases in cases of high-dose lead poisoning, mean blood lead levels in workers, and mean air lead levels in most workplaces (75). It remains a problem in small shops, however, which are covered by OSHA regulations but may not be routinely inspected. Some critics assert that enforcement of OSHA regulations is inadequate. Others state that, as revealed by several State screening programs, many employers are unaware of their responsibilities, and others ignore them. Many employees are not aware of their rights or are reluctant to report employers for fear of losing their jobs.

Lead in Paint

Although lead-based paint is now only rarely used, the paint that remains on the walls of older housing is the most significant source of lead poisoning today. Many children are exposed to lead-based paint, and efforts to remove paint from the walls as a preventive measure vary greatly from State to State. The U.S. Department of Health and Human Services reported in 1988 that 52 percent of all residential buildings have paint **containing lead in concentrations greater than or equal to that considered dangerous by the CDC (165, 169).**

In 1971, Congress attempted to address the issue of lead poisoning from lead-based paint. The Lead-Based Paint Poisoning Prevention Act and its 1973 and 1976 amendments directed the Consumer Product Safety Commission (CPSC) to establish a level of safety for lead in paint.[4] Most paints are regulated under this standard, but lead is still used in some paints (most often as a weather-resistant coating for metals) (51), and the yellow paint used for lining highways and roads contains lead as well (42). The CPSC has no control over lead-based paints already in houses and other dwellings or lead-based paint manufactured before 1977, when the regulation went into effect (165).

A second aspect of the lead-based paint legislation involves removing lead paint from housing under Federal jurisdiction, an activity that falls to the Department of Housing and Urban Development (HUD). HUD can only regulate paint in public housing or federally assisted dwellings (165). The Department's regulations currently ensure notification of residents in and purchasers of HUD-associated housing constructed before 1950 of the hazards of lead poisoning from lead-based paint. The regulations also prohibit the use of lead-based paint in HUD housing and federally owned and assisted construction or rehabilitation of residential structures, and ensure removal of lead-based paint in HUD-associated housing and federally owned prop-

[3]Before 1978, the permissible exposure limit was 200 ug/m^3 (over an average time period of 8 hours). The regulations lowered the limit to 50 ug/m^3 (43 FR 52952 and 43 FR 54354) and set an action level of 30 ug/m^3 (an action level is based on the same criteria as a tolerance). At this action level, the industry must initiate environmental monitoring, recordkeeping, education, training, and medical surveillance. Medical removal protection (removing the employee to an area with exposure below the action level) is directed by the medical surveillance findings (109).

[4]CPSC's authority in this area comes from the Consumer Product Safety Act, which gives the Commission the power to ban as hazardous any consumer product that presents an unreasonable risk of injury (15 U.S.C. 2057). The current regulations state that paint may contain no more than 0.06 percent lead.

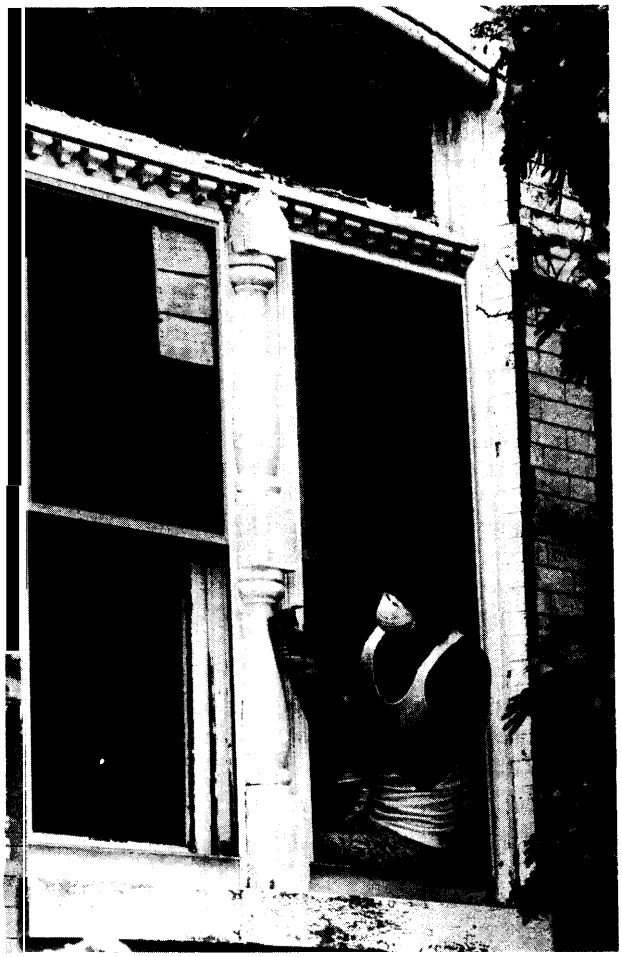


Photo credit: U.S. Environmental Protection Agency

The paint that remains on the walls of older housing is a significant source of lead poisoning.

erties (109). Removal of lead-based paint from walls is dangerous in itself. Workers can be exposed to lead dust if not adequately protected, and dust and paint chips can be released into the nearby environment if not properly disposed of, resulting in markedly increased exposure of inhabitants. HUD is currently conducting a study to determine the extent of the lead-based paint problem in public housing and to study the efficacy of alternative abatement procedures.

The Lead-Based Paint Poisoning Prevention Act also created a Federal program to fund lead poisoning prevention programs for children. Initially funded through the Bureau of Community Environmental Management, the program was transferred to the CDC in 1973, and until 1981 the CDC administered grants to the States for prevention programs. In 1981, the Omnibus Budget Reconciliation Act rolled a number of categorical health programs, including the lead poisoning prevention program, into the Maternal and Child Health Services Block Grant. Thus, the allocation of money among the various health programs, previously dictated by the Federal Government, became the decision of each individual State. Accordingly, States now choose how much money, if any, to apply to lead poisoning prevention programs (see box 10-C). Because many of these programs have been reorganized at the State level and because reporting of lead poisoning prevention expenditures is now voluntary, it is difficult to determine how expenditures on lead poisoning prevention programs have changed. According to a 1984 General Accounting Office study on the Maternal and Child Health Block Grant, lead screening projects have received "the greatest reduction in emphasis' (179), and a 1987 survey by the National Center for Education in Maternal and Child Health indicated 10 States have no lead poisoning prevention activities at all (1 11). In 1988, the Lead Poisoning Prevention Act authorized $66 million for community screening between 1989 and 1991 in order to compensate for deficits in lead poisoning prevention programs at the State level. Lead-based paint remains a significant source of lead poisoning, despite the laws and regulations that specifically address this problem.

Lead in Drinking Water

Both EPA and Congress are currently addressing the problem of lead in drinking water. In 1986, EPA estimated that 42 million Americans drank tap water containing more than 20 parts of lead per billion parts of water, the proposed drinking water standard (which has since been lowered). The Agency further estimated that exposure to lead in drinking water is keeping more than 240,000 children from realizing their full intellectual potential (171).

Lead rarely originates from source water but leaches out of plumbing containing lead pipes and fixtures or lead solder. EPA estimates that there are approximately 4.4 million lead service lines in use in the United States and that approximately 25 percent of water suppliers have some lead service lines within their distribution system (53 FR 31521). Since more acidic water leaches more lead out of plumbing systems, lead in drinking water may be regulated by controlling its pH (a measure of

Box 10-C-State Lead Poisoning Prevention Programs

Some States do nothing about lead poisoning, largely because it is not considered a significant problem. Others identify children with high blood lead levels through mandatory reports from laboratories that conduct blood tests, then follow up by treating the children and removing the environmental source of lead, if possible. Other States have an outreach program, whereby children in high-risk areas are screened and appropriate follow-up action is taken. Some communities have lead poisoning prevention programs.

A number of States have either passed legislation or are considering legislation addressing the issue of childhood lead poisoning. Massachusetts, for example, has extensive legislation that requires statewide screening of children under age 6, reporting of cases of childhood lead poisoning by physicians, and art education campaign about the dangers and sources of lead poisoning. The law also outlines lead-based paint abatement standards and a program for removing or covering lead in soil, among other provisions. Generally, areas of the country with industrial pollution, older housing, and large cities appear to have the most active lead poisoning prevention efforts.

Given the variability of these prevention efforts, it is difficult to characterize the extent of screening at the State and local levels. However, a survey conducted by the Public Health Foundation in 1983 yields some relevant data. Of the 48 State and territorial health agencies surveyed, 33 operated lead poisoning prevention services. Thirty of these programs reported screening 676,600 children ages 1 to 5. Of the children screened, 9,317, or 1.6 percent, had confined lead toxicity (defined as blood lead levels greater than 30 ug/dl and erythrocyte protein levels greater than 50 ug/dl, the CDC standard at that time). Of these children, 92 percent received medical care,[1] and environmental investigations were conducted for 96 percent. The source of lead was determined in 80 percent of the cases of confirmed lead toxicity, and 98 percent of those sources were lead-based paint. Of the children with identified hazards, the hazards were abated for 91 percent.

In one sense, these data are encouraging, since the majority of children with elevated blood lead levels evidently obtained medical treatment and hazard abatement. On the other hand, the number of children identified and treated is only a small percentage of the 200,000 children estimated to have elevated blood lead levels. Thus, a large number of children with potentially dangerous exposure to lead are not being helped.

[1]From this point on, percentages are based on data reported by those State and territorial health agencies that could provide both the numerator and the denominator for their percentages. As not all agencies reported all the relevant data, not all are represented in these numbers.

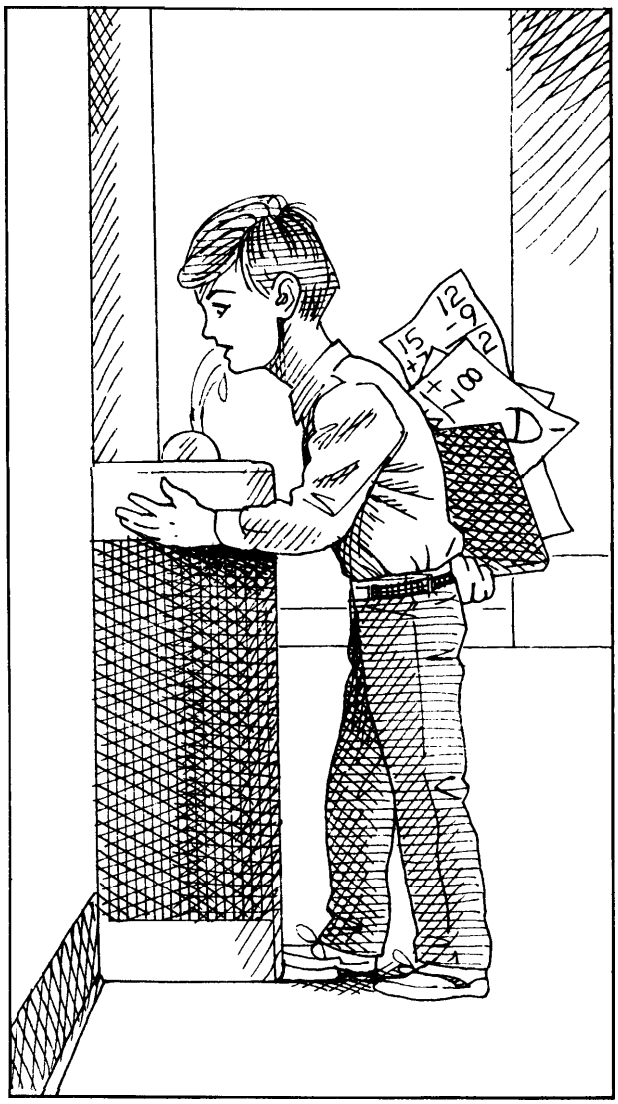
SOURCES: Public Health Foundation, ***Special Report: State Health Agency Lead Poisoning Prevention Activities, 1983*** (Washington, DC: 1986); U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, *The Nature and Extent of Lead Poisoning in **Children** in the United States: A Report to Congress* (Washington, DC: 1988).

acidity), and EPA argues that the least expensive method for reducing lead in drinking water is central corrosion control treatment (84,171).

Under the Safe Drinking Water Act (1974), EPA must establish maximum contaminant level goals (MCLGs) and national primary drinking water regulations (NPDWRs) for contaminants that may have an adverse effect on the health of the population drinking the water. While MCLGs are nonenforceable health goals, NPDWRs are enforceable standards. NPDWRs include maximum contaminant levels (MCLs) or treatment technique requirements, or both. In 1986, amendments to the Safe Drinking Water Act listed 83 contaminants, including lead, for which EPA had to develop MCLGs and NPDWRs.[5]

In 1988, EPA proposed an MCLG of zero for lead. The proposed NPDWRs establish an MCL of 0.005 milligram of lead per liter of water (mg/l) for water entering the distribution system (to replace the current MCL of 0.05 mg/l); require corrosion control treatment techniques if specified levels of lead, copper, and water acidity are not met (the Agency issued regulations for copper and lead simultaneously); and require public education if other meas-

[5]The 1986 amendments to the Safe Drinking Water Act also banned the use of lead solder or flux and lead-bearing pipes and fittings. This ban was effective in 1986, and States were required to implement and enforce it as of June 1988. EPA is currently developing a program to withhold Federal grants for programs to improve the quality of drinking water from States that fail to enforce the ban (53 FR 31516).



Illustrated by: Ray Driver

ures fail.[6] Some argue that the proposed regulations are not strict enough and claim that EPA has both the authority and the responsibility to set MCLs at the tap.[7] Water suppliers, on the other hand, find the corrosion control program to be unwarranted and expensive.

The debate over regulation of lead in drinking water focuses on whether the public water supplier or the consumer is ultimately responsible for preventing high levels. Public water systems control the quality of the water they distribute, including the parameters that determine how much lead will leach from plumbing into the water. On the other hand, the water passes through a distribution system that is owned partially by the water supplier and partially by the consumer. If the regulation is enforced at the tap, the water supplier must assume responsibility for some lead contributions from the consumer's plumbing. If lead levels are enforced at the beginning of the distribution system, the consumer must assume responsibility for some of the water supplier's plumbing or the corrosivity of the water supplied by the water system, or both. Undercurrent EPA regulations, the supplier is responsible both for lead levels in the water in the distribution system and for the water quality at the tap.

Lead in drinking water remains a serious problem in some water supplies, especially in schools. The efficacy of the regulations promulgated by EPA will be crucial in determining how serious a problem it remains. (Another widely discussed issue concerns lead in water coolers-see box 10-D.)

Lead in Incinerator Ash

The United States produces approximately 160 million tons of solid waste every year. Currently, approximately 83 percent of this waste is put in landfills, 11 percent is recycled, and 6 percent is incinerated (86). As landfills are rapidly being filled, there is much discussion concerning other methods of disposing of this waste. EPA estimates there will be a sixfold increase in the capacity for waste incineration in the United States over the next 15 years (76).

Incineration has both advantages and disadvantages. Its major advantage is that it reduces the volume of waste by 75 to 80 percent. Furthermore, it can be used to generate electricity and can be linked with recycling methods to remove such solids as iron, steel, glass, and paper from the waste stream

[6]Under these proposed relations, all water leaving the treatment plant would have to meet the 0.005 mg/l standard. To regulate how much lead the water can pick up as it travels from the distribution point to the consumer, EPA proposed that targeted samples be taken from consumers' taps. If the average lead level is less than or equal to 0.01 mg/l, the average copper level less than or equal to 1.3 mg/l, and the pH greater than or equal to 8.0 in at least 95 percent of the samples, then the supplier is not required to take any further action. If any of these three standards is not met, the water supplier would be required to implement or improve its corrosion control. If the lead levels are above 0.02 mg/l, the supplier would have to launch a public education program to encourage consumers to reduce their exposures to lead in drinking water (53 FR 31516).

[7]There is some concern that EPA's measures do not adequately treat the problem, since: 1) the NPDWR of 0.005 mg/l does not reflect the MCLG of O mg/l; 2) the tap standard is not enforceable; and 3) limited sampling at the tap will necessarily overlook some households with high lead levels (1 16).

Box 10-D-Lead in Water Coolers

Another source of lead in drinking water, water coolers, has received considerable attention in the press and is the subject of legislation passed in the 100th" Congress. Some water coolers may contain lead-lined tanks or lead solder that comes into contact with the water. Data solicited by Congress from manufacturers reveal that close to 1 million water coolers currently in use contain lead (U.S. Congress, Committee on Energy and Commerce, 1988). These water coolers are of special concern because they are frequently used in schools.

The Lead Poisoning Prevention Act of 1988 addresses this situation through the following provisions: 1) recalling all water coolers with lead-lined tanks; 2) banning the manufacture or sale of water coolers that contain lead; 3) setting up a Federal program to assist schools in evaluating and responding to lead contamination problems; and 4) making funds available for the initiation and expansion of lead poisoning prevention programs (for all sources of lead poisoning). This last provision is designed to expand on Federal funds for lead screening from the Maternal and Child Health Services Block Grants. The legislation also requires that the Environmental Protection Agency publish a list of water coolers that are not lead-free within 100 days of enactment (U.S. Congress, Committee on Energy and Commerce, 1988). The Agency released a proposed list in April 1989. The original draft of the legislation contained a section that set a Safe Drinking Water Act maximum contaminant level at the tap, but this section was eventually deleted because of political pressure (''House Staffers, ' 1988).

SOURCES: "House Staffers Scrap Lead Standard to Speed Drinking Water Bill's Passage," *inside EPA 9:6, 1988;* U.S. Congress, Committee on Energy and Commerce, Subcommittee on Health and the Environment, *Lead Contamination* (Washington, DC: U.S. Government Printing Office, 1988).

(76). However, byproducts of incineration may have adverse effects on the environment and on human health. Residue remaining from incineration (bottom ash), particles removed from the air after combustion (fly ash), and airborne emissions (stack emissions and fugitive emissions) may contain high concentrations of toxic substances, including lead and other toxic heavy metals (76). Compared to landfills, stack and fugitive emissions may greatly increase exposure. On the other hand, when ash is placed in landfills, the lead may leach out of the ash into the groundwater, eventually ending up in lakes, ponds, and rivers that may be used for recreation or drinking water.

EPA has the authority to regulate incinerator ash under the Resource Conservation and Recovery Act, but there is some debate as to whether incinerator ash should be considered a hazardous substance because the municipal solid waste which is burned to create it is not designated hazardous waste. Some environmentalists call for testing all incinerator ash and treating it as hazardous waste if the tests indicate it has hazardous properties.

Congress has been interested in this issue as well. Legislation has been introduced in the 10lst Congress to amend the Clean Air Act, directing EPA to promulgate regulations that would control emissions of specified air pollutants, including lead, from municipal waste incineration sites and ensure safe management of municipal incinerator ash.

Although the amount of human exposure to lead from municipal waste incinerators is not large now, the projected increase in the number of such incinerators indicates that it could become a problem in the future.

Lead in Soil

EPA is conducting a project under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA, or Superfund) to determine whether abatement of soil lead (by removal or some form of isolation) will reduce

Photo credit: National Archives

Lead may be released into the air through the weathering of structures painted with lead-based paint.

childhood exposure to lead (as determined by the amount of lead measured on the hands and in the blood of children). Studies are being conducted in Boston, Cincinnati, and Baltimore. The studies will not focus on high-lead areas, such as areas near lead smelters, or on children who need clinical attention. Instead, the focus is on intermediate lead levels, which are more typical urban exposures (42).

Summary and Conclusions

Public health measures have achieved a substantial decrease in human exposure to lead in recent years; lead poisoning, however, remains a significant problem, especially in children. As tests become more sensitive, studies indicate that neurobehavioral dysfunction is associated with lower blood lead levels than previously believed. The precise level of exposure which causes impairment is controversial: there may be no threshold level for adverse effects, in which case the more sophisticated our ability to detect impairments from lead poisoning becomes, the lower the levels at which impairments may be found. Since 10 to 15 ug/dl is the limit most recently proposed as a maximum blood lead level and the medical treatment techniques now available are not able to reduce blood lead levels below approximately 20 ug/dl, prevention is crucial.

Since lead poisoning was clearly identified as a public health problem, it has received a great deal of attention from Congress and a number of Federal agencies. EPA's reduction of lead in gasoline has greatly reduced the amount of lead in the air; FDA and the food industry have together reduced the amount of lead in food; and EPA has recently implemented a regulatory program to control the amount of lead in water. OSHA regulations have reduced lead exposure in most large lead-using industries. Federal and State programs have begun to remove lead paint from older housing. The regulatory framework that now exists, if properly enforced, could continue to reduce many sources of exposure to lead.

Despite these areas of success, progress remains to be made. Not everyone is satisfied with the steps that have been taken. Some argue that the existing regulations fail to treat the problem of lead in drinking water adequately. Some feel the OSHA regulations for lead exposure in the workplace are not properly enforced and have too many exceptions. Also, there are no Federal programs to remove lead-based paint in old houses or to establish mandatory, centralized reporting of lead poisoning.

Many argue for stronger measures to prevent lead toxicity. Prevention might be improved by a general screening program for all children and by adopting alternatives to incineration of waste, thus avoiding increased exposure to lead in the air. Federal programs to improve conditions in the workplace and remove lead-based paint from all houses could be implemented. Lead content in water could be monitored strictly, and if need be, regulations could be revised. Public education programs could be introduced in high-risk areas near industrial or waste-disposal sites. Federal money could be designated for specific lead poisoning prevention programs rather than including lead poisoning programs under the block grant umbrella.

Designing programs to remove lead from the environment is most problematic when responsibility for removing contamination is not clear. A baby poisoned by lead from canned milk is clearly the food industry's responsibility, therefore that industry was prompt and thorough in its response to the lead poisoning problem. In many cases, however, such as controlling lead in drinking water, responsibility for lead poisoning cannot be so clearly ascertained: some public water suppliers question whether they or consumers are responsible for plumbing with lead pipes or lead solder. The Nation must address difficult questions such as this if continued progress is to be made in reducing public exposure to lead.

EXPOSURE TO NEUROTOXIC PESTICIDES IN AGRICULTURE

The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) of 1947 defines a pesticide as:

> . . . any substance or mixture of substances intended for preventing, destroying, repelling or mitigating any insects, rodents, nematodes, fungi, or weeds or any other form of life declared to be pests. . . and any substance or mixture of substances intended for use as a plant regulator,[8] defoliant or dessicant.

[8] Alar (daminozide), for example, is called a pesticide for regulatory purposes, even though it does not kill pests. It is used as a growth regulator to promote a uniform red color in apples and to prolong shelf-life.

Human exposure to pesticides can occur in a number of ways—through contaminated drinking water, through eating foods containing pesticide residues (see box 10-E), through pesticides used in the yard, home, and office, and through exposure in various occupational and agricultural settings. Besides field workers and pesticide applicators, those at risk in agricultural settings include nursery, greenhouse, forestry, and lawn care workers. Although pesticides are a major health concern in the home and for exterminators, highway workers, grain elevator operators, and pesticide manufacturing and formulating employees, this section focuses on pesticide exposure in the agricultural setting.

Approximately 1 billion pounds of pesticides are used annually in agriculture in the United States, and approximately 4 billion pounds are used annually worldwide (102,174). Approximately $7 billion is spent annually on pesticides in the United States. Agriculture accounts for more than two-thirds of the expenditures and approximately three-fourths of the quantity used (174).

Agricultural workers who may be exposed to pesticides include pesticide handlers (handling is defined as mixing, loading, applying, flagging,[9] and equipment cleaning, repairing, and disposal), who work with concentrated forms of pesticides; workers performing hand labor in fields treated with pesti-

Box 10-E—Pesticides in Food

A report released in February 1989 by the Natural Resources Defense Council (NRDC), *Intolerable Risk: Pesticides in Our Children's Food,* has spurred considerable debate about the risks to humans, particularly children, of pesticide residues in food. The report analyzed the extent of children's exposure and attempted to determine the potential hazards, focusing on increased risk of cancer and neurobehavioral damage. Data analyzed in the study were obtained from the Environmental Protection Agency (EPA), the Food and Drug Administration, and the Department of Agriculture.

After examining 23 pesticides known to have adverse health effects, the report concluded that preschoolers are being exposed to hazardous levels of pesticides in fruits and vegetables. Twenty of these pesticides were found to be neurotoxic. NRDC estimated that, from raw fruits and vegetables alone, at least 17 percent of the preschool population, or 3 million children, are exposed to neurotoxic organophosphorous pesticides above levels the Federal Government has described as safe.

NRDC criticized EPA for setting legal limits for pesticides in foods based on data collected 20 years ago and on adult consumption of fruit and vegetables (children generally eat more produce than adults). A few of NRDC's primary recommendations follow:

Congress must clarify EPA's authority to change tolerance levels quickly.
. EPA must consider risks from ''inert' ingredients when regulating pesticides.
. Neurotoxicity testing should be required for all pesticides used on food.
. Congress should establish national definitions of "integrated pest management" and ''organic" farming technologies and develop a national certification process for goods grown using these technologies.

The report also includes recommendations to the public to reduce their exposure to pesticides.

EPA believes that the NRDC study overstates the risks from pesticides. The Agency stated that the benefits of pesticide use outweigh the minimal risks and that EPA routinely takes into account the potentially higher exposure of children. However, EPA officials do concede that the report raises valid questions. In a news release distributed the same day as the NRDC study, the National Food Processors Association (NFPA) stated that pesticides are virtually nonexistent in packaged foods and that, when detected, they are far below allowable levels. The NFPA attributes this absence of residues mainly to the NFPA Pesticide Protective Screen Program, which spells out proper pesticide control and monitoring practices for growers producing crops for the food industry.

SOURCES: Office of Technology Assessment, 1990; D. Duston, ''Hotline Helps Growers Find Alternatives to Pesticides,' Associated Press, Mar. 22, 1989; D. Duston, "Eight in 10 Americans Prefer Chemical Free Food; Half Would Pay More," Associated Press, Mar. 19, 1989; A.K. Naj, "Panel Assails Pesticide Study, Calls Food Safe," *Wall Street Journal,* Apr. 6, 1989, sec. B, p. 3; Natural Resources Defense Council, intolerable *Risk: Pesticides in Our Children's Food* (Washington, DC: 1989).

[9]Flaggers are workers who direct crop dusters as they spray pesticides on fields.

cides (called farmworkers in this report); and workers in forests, nurseries, and greenhouses where pesticides are used.

Extent of Exposure of Agricultural Workers

Agriculture is the primary source of income for an estimated 4 to 5 million Americans, a significant proportion of whom are children under the age of 16 (102). Many of these persons are exposed to higher levels of pesticides than the general public. Approximately 2.7 million agricultural workers in the United States are migrant and seasonal farmworkers (164), and most seasonal work involves contact with pesticide residues on crops such as cotton, vegetables, fruits, and nuts. Another group with significant exposure to pesticides is pesticide handlers: EPA estimates there are approximately 1.3 million certified pesticide applicators in the United States (176). The number of agricultural workers performing other pesticide-handling jobs is unknown.

The seventy of illnesses caused by pesticides depends mostly on the dose absorbed and the inherent toxicity of the product. Farmworkers are exposed to pesticides primarily through residues on foliage and crop surfaces, during aerial and hand spraying, picking, packing, and sorting, but also during hoeing and other field work. Forest, greenhouse, and nursery workers are exposed by similar means. Mixers, loaders, and applicators may be exposed to concentrated doses of pesticides in the course of their daily work. Exposure usually occurs by absorption through the skin, except in the case of fumigants, which are inhaled. The amount of pesticide absorbed depends on the nature of the work being performed, the clothing the worker is wearing, the part of the body exposed, and the condition of the worker's skin (absorption increases with dermatitis, cuts, and abrasions). Another relevant factor in exposure is the rate at which pesticides degrade, which varies with conditions such as heat and moisture.

Estimates of the incidence of pesticide-related health problems among workers vary. The annual worldwide incidence of pesticide poisonings is estimated to be between 500,000 (192) and 2.9 million (69), with a fatality rate of approximately 1 percent (102). In the United States, the prevalence of pesticide-related illness among farmworkers may be as high as 300,000 cases,[10] only 1 to 2 percent of which are thought to be reported (31). The majority of reported cases of pesticide-related illness involve exposure to neurotoxic pesticides (102,185), but the lack of reporting of most cases complicates the assessment of any persisting neurological and psychiatric problems. Some observers have estimated that in developed countries 4 to 9 percent of acutely poisoned individuals suffer long-term neurological and psychiatric effects (46).

Special Risks to Children

Pesticides are thought to pose a considerably higher risk to children than to adults (106,114). Children can be exposed in a number of ways: through prenatal maternal exposure, from being in the fields where their parents work, contact with pesticide residues on parents' clothing, living in migrant camps next to fields being treated, and working in the fields themselves. Since they absorb more pesticide per pound of body weight, children may receive substantially higher doses of pesticides than adults, and their immature development may make them more susceptible to neurotoxic effects. EPA and OSHA standards for worker safety are based on adult exposure only.[11] Many organ systems, including the nervous and reproductive systems, are still developing in infants and young children. The effects of pesticides on these developing systems are largely unknown. There are important lessons to be drawn from the case of lead, which has severe effects on the developing nervous system and other organs of children.

Documented Adverse Effects on the Nervous System

Although many pesticide-induced illnesses among agricultural workers are thought to be severe and acute, some evidence suggests that they are in fact moderate and chronic (31). The full effects on learning and perception and the emotional changes associated with pesticide exposure are not known because of the difficulty of testing these functions and establishing a normal range (5). Failure to report illness and the lack of comprehensive studies of the agricultural worker population may result in under-

[10]This figure is based on extrapolation of data collected in California. Tracking the prevalence of farmworkers' pesticide-related illnesses is difficult because of the lack of reporting requirements in most States and the limitations of those that do exist. These limitations are discussed later in this chapter,

[11]The National Academy of Sciences recently initiated a 2-year study to assess the risk of exposure of children to pesticides.

Photo ***credit: U.S. Environmental Protection Agency***

Current EPA regulations establish basic protective clothing requirements for agricultural workers who enter treated fields. However, recent studies document significant pesticide exposures despite the use of typical protective clothing.

estimation of the true extent of both short- and long-term neurological effects. Organophosphorous insecticides, which make up approximately 40 percent of all pesticides used in the United States, are currently the most commonly reported source of worker illness. The more persistent organochlorine pesticides, used extensively in the 1940s through 1970s, are now either banned or restricted in the United States and thus do not contribute as much to worker illness. What is known about the effects on worker health of a few commonly used classes of pesticides is examined later in this chapter.

Short-Term Effects on the Central Nervous System

Some cases of worker illness are mild and persist for a few hours. In more severe cases, symptoms may not peak until 4 to 8 hours after onset and may persist from 1 to 6 days. Some recovery periods are longer (90):

- In a moderately severe poisoning of 24 field workers, including children, exposed to residues of two pesticides, mevinphos (Phosdrin) and phosphamidon (Dimecron), in California, anxiety and other symptoms were reported after 70 days (98,184). In this case, farmworkers were working in cauliflower fields prior to the legal reentry interval.
- There have been several documented poisonings of entire crews who entered fields after the permissible reentry interval. In 1987,78 farmworkers in three different crews developed moderate to severe pesticide poisoning from contact with phosalone (Zolone), used in California vineyards, long after it was thought safe to reenter. Because of its persistence and risk to farmworkers, phosalone is no longer used on grapes in California (23).
- In 1988, two crews were poisoned by a highly toxic insecticide, methomyl, in California. In the first case, 34 orange harvesters went into a methomyl-treated orchard 1 day after application, and 17 developed symptoms of pesticide poisoning that required hospital treatment. In the second case, grape workers were hospitalized after exposure to methomyl. As a result of these poisonings, the reentry interval for methomyl in California was increased from 2 to 14 days (23).

Long-Term Effects on the Central Nervous System

The nature of long-term neurobehavioral effects of exposure to organophosphorous insecticides is unresolved and deserves further investigation. The evidence supporting the existence of delayed, persistent, or latent effects in humans includes case reports, epidemiological studies of agricultural workers with and without histories of acute poisoning, and deaths resulting from neurobehavioral disease among agricultural workers.

Case Studies—The pesticides parathion, mevinphos (Phosdrin), and malathion are frequently reported as causing health problems. Case reports and studies of acute poisonings of agricultural and other workers indicate that 4 to 9 percent of the acutely poisoned individuals experienced delayed or persistent neurological and psychiatric effects (46). These effects include agitation, insomnia, weakness, nervousness, irritability, forgetfulness and confusion, and depression (56,64,65,1 55); persistent mental disturbances—reported as delirium, combativeness, hallucinations, or psychoses—are noted in some cases of pesticide poisonings (62). Occupations most frequently mentioned in case reports include mixers, loaders, applicators, pilots, flaggers, nursery and greenhouse workers, pesticide manufacturing workers, agricultural and pest control operators, and inspectors. Farmworkers tend not to appear in the reports, for reasons that are discussed later in this chapter.

Epidemiological Studies-Although few epidemiological studies of agricultural workers have been done, approximately 500 subjects from various cohorts have been subjected to standardized neurobehavioral assessments examining memory, reaction time, behavior, visual ability, and mood. Subjects tend to be young, mostly male, and employed in agricultural occupations for unspecified periods. In field studies, quantitative data on exposure are lacking.

In general, this research demonstrates that pesticide poisoning can lead to poor performance on tests involving intellectual functioning, academic skills, abstraction, flexibility of thought, and motor skills; memory disturbances and inability to focus attention; deficits in intelligence, reaction time, and manual dexterity; and reduced perceptual speed. Increased anxiety and emotional problems have also been reported. Exposed groups included farmers without symptoms (73), industrial workers with accidental exposures (97), pest control workers (90), and a wide variety of agricultural workers tested an average of 9 years after an acute poisoning was diagnosed by a physician (140).

Neurobehavioral Disorders, Mortality, and Accidents—Analysis of occupation and causes of death reported on death certificates suggests that agricultural workers are at risk of dying from neurobehavioral disorders and accidents. Approximately twice the expected mortality from behavioral disorders (i.e., those resulting from altered perception or judgment) has been reported among white male farmworkers and orchard laborers from Washington (99) and among California farmworkers (154).

Both of these studies and one of British Columbia farmworkers (55) found disproportionate mortality due to external causes, particularly motor vehicle accidents. The precise role of pesticides, if any, in the mortality patterns is unknown. Based on worker reports of feeling "fuzzy" at the end of the work day, researchers have speculated that farmworker exposure to pesticides impairs judgment and coordination and may contribute to motor vehicle accidents (155). There are numerous case reports of near misses and fatal workplace accidents involving farm machinery and crop-dusting aircraft in which behavioral effects of pesticides are implicated (38,62, 135,136,149,191).

Suspected Adverse Effects and Limitations of Existing Data

The occurrence of neurobehavioral disorders after chronic low-level exposure in the absence of acute poisoning has not been adequately studied. Neuropsychological assessments of occupational groups have yielded inconsistent results, perhaps reflecting differences among pesticides and differences in the type and scope of tests used. Subtle neurobehavioral effects have been observed most consistently in young, asymptomatic male workers who have been employed for a long time (19,194), who have been previously diagnosed as having acute pesticide poisoning, or who are recovering from an acute exposure (38,73,140). Few studies have assessed the duration of impairment. Field studies have not provided sufficient data on exposure levels or duration to understand dose-response relationships, nor have most studies controlled for age, education, or other potential confounding factors. Few studies have examined exposed workers prospectively, subgroups of women or aging workers, interactions between pesticides, or interactions between pesticides and pharmacological agents (including ethanol or common medications).

Federal Regulation

Most workers in the United States are protected by the Occupational Safety and Health Act, which affords them certain rights, including permissible exposure limits, personal protective equipment and clothing, access to medical and exposure records, training about the risks of exposure, and protection against employer retaliation. Pesticide handlers and workers in forests, nurseries, and greenhouses are covered under these regulations. OSHA requires that field workers be provided with toilets, drinking water, and water for hand washing; however, handling of pesticides is covered under FIFRA, which is administered by EPA. Since 1983, manufacturing workers have had the right to information on the hazards of the chemicals with which they work under OSHA's Hazard Communication Right-to-Know Standard. Since 1988, other industrial workers have also had this right.

FIFRA was enacted in 1947 to protect farmers from ineffective and dangerous pesticides by requiring that a pesticide be registered before it is marketed. The legislation was amended extensively in 1972 (Public Law 92-516), with new provisions

allowing for direct controls over the use of pesticides, classification of selected pesticides into a restricted category, registration of manufacturing plants, a national monitoring program for pesticide residues, the inclusion of environmental effects in the cost-benefit analysis of the pesticide regulation process, and the required reregistration of older pesticides to ensure that they meet new data requirements (2).

Since FIFRA was amended in 1972, controversies about its implementation and its ability to protect farmers and farmworkers have received repeated congressional attention. In 1988, after considerable political debate, a compromise bill (dubbed "FIFRA lite" because of its restricted scope) was passed by Congress and signed by the President.

Photo credit: Douglas Watts/Christophar Brady

The new law requires EPA to review within the next decade the 600 active pesticide ingredients and to charge manufacturers for some of EPA's costs (under previous law, the government was responsible for virtually all of the cost). The bill also partially repeals the indemnification provision that required the government to pay manufacturers or users of pesticides for existing stock whose registration was canceled by the Agency. This provision was a major obstacle to EPA's cancellation or suspension of some of the most toxic pesticides.[12] Many issues, however, were lost in the final bill, including farmworker protection standards and specific requirements for EPA review and testing of pesticides. Two efforts to strengthen Federal authority were defeated: 1) synchronization of data requirements, which would have prevented States from requiring additional data before registering pesticides, and 2) preemption of States from setting more stringent tolerances for pesticide residues in food.

EPA promulgated regulations under FIFRA in 1974. Of particular interest here are those regulations dealing with the occupational safety and health of agricultural workers (40 CFR 170 and 156). The 1974 regulations apply only to workers performing hand labor in fields during or after pesticide application. Their main provisions are a prohibition against spraying workers; specific reentry intervals (i.e., the time that must elapse between application of a pesticide and the return of workers to the treated area) for 12 pesticides and a general reentry interval for all other agricultural pesticides; a requirement that protective clothing be worn by any worker who has to reenter a treated area before the reentry interval has expired; and a requirement for "appropriate and timely" warnings to workers when they are expected to work in fields that have been or will be treated with pesticides.

FIFRA has been criticized as inadequate to protect workers and the public from pesticides known to cause or suspected of causing serious chronic effects, including cancer, reproductive problems, and neurological damage (178). EPA has set reentry intervals for only 68 of more than 400 active ingredients currently used to manufacture thousands of agricultural pesticide products.

In addition, FIFRA requires a balancing of risks and benefits to determine whether a hazardous pesticide should be canceled or suspended. This provision can delay or prevent EPA from regulating pesticides that are potentially neurotoxic, depending on whether the perceived benefits of its use outweigh the perceived risks. Risk-benefit analysis, however, rarely includes the costs of ill health to those exposed, including lost work time, hospital care, and other medical care.

In 1983, EPA reviewed the regulations under FIFRA and determined that they were inadequate to protect workers occupationally exposed to pesti-

[12]Chlordane was originally proposed for cancellation in 1974 because of its adverse health effects. There is some feeling that the considerable cost to EPA of indemnifying chlordane's manufacturers and users may have influenced its decision not to cancel the registration. While agricultural uses of chlordane were canceled in 1984, it was still used widely to kill termites until 1988. On Feb. 3, 1989, the U.S. Court of Appeals overturned an earlier decision and permitted the sale of existing chlordane stocks (112).

cides. The Agency proposed new regulations in 1988. These regulations would cover workers in forests, nurseries, and greenhouses; pesticide handlers; and workers performing hand labor in treated fields. Some of the key items of the proposed regulations follow:

- General pesticide safety information must be placed in a prominent location at each farm, forest, nursery, and greenhouse during the growing season. Workers who do not speak English must be given a written warning in their own language to obtain a translation of this information. Training must be provided for all persons who handle agricultural pesticides and for all persons who enter treated areas before the reentry interval has expired. Any person who handles a pesticide must be provided, on request, all information from the labeling of that pesticide.
- All workers must be clearly and adequately notified about pesticide application and relevant reentry intervals. The methods of notification will vary according to the site, but will include a requirement that warning signs be posted outside pesticide-treated areas with a reentry interval of more than 48 hours.
- All pesticide handlers and early reentry workers must wear minimum personal protective equipment, as specified by pesticide labels. Determination of the appropriate equipment must take into account the toxicity of the pesticide, the handling technique, and the route and type of exposure.
- The minimum reentry interval will be "until sprays have dried, dusts have settled, or vapors have dispersed. " Reentry intervals will be set at 48 hours for organophosphorous and n-methyl carbamate insecticides in toxicity category I (most acutely toxic) and 24 hours for the same pesticides in toxicity category II and all other pesticides in toxicity category I.
- Workers must be provided with water, soap, and disposable towels after exposure to pesticides or pesticide residues. Information about and transportation to nearby medical facilities must be provided to workers in emergency cases of pesticide poisoning or injury.
- Commercial handlers who are exposed to toxicity category I or II organophosphorous insecticides for 3 consecutive days or any 6 days in a 21-day period must be monitored for cholinesterase inhibition (177).

The proposed regulations have been criticized by farmworkers' and farmers' groups, growers, and pesticide users and producers. Critics argue that the standards fail to address many needs, including those for mandatory education of all farmworkers concerning the neurotoxic and other health effects of pesticides and safety training in the use of pesticides; telling workers what pesticides they have been exposed to; more protective reentry intervals; and consideration of the additive and synergistic effects of exposure to multiple pesticides. Critics also argue that the proposed standard could increase farmworkers' risks by permitting early reentry into treated fields as long as workers are given protective equipment.

Pesticide regulation and policy have historically been made at the Federal level, yet the Office of Pesticide Programs has consistently had one of the smallest budgets of any EPA program. Resources for the review of toxicological data, monitoring programs, and worker protection standards have been limited. EPA currently provides no funds to State agencies to conduct worker and public health evaluations. Indeed, EPA officials have stated that farmworker protection standards are not part of current State enforcement grants under FIFRA (105).

Areas of Particular Concern

Pesticide Registration-An important obstacle to protecting farmworkers from neurotoxic pesticides is the major gaps in data in many pesticide registration files. In 1984, the National Academy of Sciences found that 67 percent of pesticides studied had undergone no neurotoxicity testing at all, and all of the neurotoxicity tests performed were judged inadequate (108). The 1988 FIFRA amendments gave EPA 9 years to complete its pesticide registration review, but the battery of tests currently required by EPA for pesticide registration is geared toward detecting only the most obvious neurotoxic effects. Only one type of test specifically intended to detect nervous system impairments is currently included in EPA's pesticide assessment guidelines, although new test guidelines are being devised (see ch. 5). EPA was petitioned by a group of consumer advocates and professional organizations to develop more extensive neurotoxicity test guidelines (26).

Another gap in protection is the lack of data on effects of exposure to the so-called inert ingredients in pesticides. These ingredients are used as carriers of the active ingredient and do not appear on pesticide labels because of their trade secret status.[13] They are inactive only to the extent that they are thought to have no effect on the targeted pest. Hence, they may be defined as inert yet be toxic to humans. Of the 1,200 substances designated as inert, EPA concludes that 55 are "toxicologically significant," with another 65 structurally related to substances known to be toxic. As of 1987, EPA did not know the toxicity of some 800 inert ingredients contained in pesticide products and regarded some 200 as generally safe (2); the Agency has since incorporated inert ingredients into its ongoing review of the toxicity of pesticide ingredients.

FIFRA permits States to register for 5 to 8 years pesticides needed to fill "special local needs" and "crisis" situations. This may, under certain conditions, provide a substantial loophole in farmworker protection, because it allows States to register pesticides that have not met Federal testing requirements. There has been considerable criticism of this practice.

Public attention was drawn to the issue of the quality of data submitted for the registration of new products by the discovery that one of the major laboratories providing data to EPA had falsified findings (31,143). In 1984, EPA's internal review process for evaluation of toxicological data was criticized because of cases in which EPA reviewers had incorporated information provided by manufacturers, apparently without any independent analysis. In 1989, the Senate Environment and Public Works committee initiated an oversight review of EPA's registration standards when it was learned that seven of the eight members of EPA's Science Advisory Panel had apparently served as consultants to the chemical industry (93,163). Thus, although EPA is working to fill the data gaps in pesticide registration, there remain questions about the impartiality of the Agency's regulation process.

Reentry 1ntervals-Unlike industrial workers, farmworkers are not protected by specific maximum levels of exposure to chemicals. Rather, they are protected by reentry intervals, which restrict entry to a field after pesticide application (40 CFR 1988 ed. 170). When they were first instituted in 1974, specific reentry intervals were set only for the 12 chemicals with the highest observed toxicity; access to all other active ingredients was restricted only "until sprays have dried or dusts have settled." Currently, specific reentry intervals have been set for 68 active ingredients for which animal studies demonstrated need. These 68 active ingredients are used in about 90 percent (by volume) of pesticides used in agriculture.

EPA claims that these reentry intervals protect workers from the most toxic active ingredients used in pesticides, but many observers are concerned that the existing regulations do not adequately protect farmworkers from neurotoxic pesticides. Farmworker protection advocates argue that the blanket reentry interval which covers other pesticides improves farmworker safety somewhat, but more adjustments need to be made for specific chemicals. There have been episodes of worker poisoning and even fatalities, particularly involving parathion, due to inadequate reentry intervals (102,151). Toxic residues can persist on foliage for weeks after application and are known to persist longer in dry climates (102). In California, most farmworker poisonings from neurotoxic pesticides have occurred because of inadequate reentry intervals (185). Several States have gone beyond EPA's standard and imposed longer reentry intervals based on local conditions. California, for example, has set many longer reentry intervals based on local conditions. Texas has set a minimum 24-hour reentry for all labor-intensive activities and has set longer reentry intervals for a number of pesticides. New Jersey and North Carolina require a 24-hour reentry interval for all toxicity category I pesticides. Other States, too, are revising their standards for reentry intervals.

The 1988 FIFRA amendments address some of the shortcomings of piecemeal regulation. EPA is currently drawing up proposals for stricter regulations, including longer reentry intervals for more chemicals.

Protective Clothing-current EPA regulations establish a basic protective clothing requirement for workers who must enter treated fields before the reentry interval has elapsed. Proposed EPA regula-

[13]Pesticides are not generally applied in a pure form. The pesticide (also known as the active ingredient) is usually diluted by a solvent or an inactive solid (known as the inert ingredient).

tions would specify particular items to be worn, depending on the task being performed, the circumstances of potential exposure, and the toxicity class of the pesticide. However, some persons argue that protective clothing and equipment are not adequate to protect workers from harmful exposures. All too frequently, employers do not provide protective clothing and equipment or employees do not wear them because of the excessive heat or their constraints on movement. Furthermore, recent studies document the significant exposures workers may receive even while using an approved respirator or wearing typical protective clothing (48).

Lack of Pesticide Illness Reporting—Because there are inadequate reporting mechanisms for acute pesticide poisoning episodes and none for adverse chronic effects among farmworkers in the United States, the true rate of pesticide-related illness among farmworkers may be underestimated. Even if there were more centralized reporting, physicians often have little training in occupational medicine and thus may not recognize instances of pesticide poisoning, and patients rarely have access to information about the pesticides to which they are exposed. The lack of occupational histories and accurate exposure data make proper diagnosis and treatment difficult, if not impossible (103). Furthermore, many ill workers never actually see a doctor.

Farmworkers, especially migratory farmworkers, whose immigration status and language barriers make them especially vulnerable, are often not represented by unions that influence standards of health and safety in the workplace. On a State level, most migrant farmworkers are excluded from workers' compensation and unemployment insurance (103). These exclusions from governmental protections prevent accurate estimates of pesticide illness, lost work time, and medical costs. Persons who advocate greater protection for farmworkers argue that reporting requirements for national pesticide illness and pesticide use would enable regulators to target pesticides for regulatory action and better assess their effects on health (134).

Monitoring Methods and Needs

There is no regular or required biological monitoring of agricultural workers exposed to pesticides in the United States, except for periodic cholinesterase tests for a small group of certified applicators exposed to organophosphorous and carbamate insecticides on a regular basis in California. Proposed EPA regulations would require monitoring of commercial pesticide handlers under certain circumstances. One direct means of assessing workers' exposure to chemicals is by measuring the parent substance or its metabolizes in the blood or urine; however, this methodology is available for only a limited number of pesticides (101). A promising new field cholinesterase test has been developed and used in Central America to identify workers suffering adverse effects (88); such a test might improve worker awareness and enhance preventive medical care (157) if workers can be induced to participate.

Monitoring programs are most effective when they are based on an understanding of the nature of farmworker exposure and the patterns of pesticide use. More extensive monitoring would allow better assessment of the extent of neurobehavioral problems caused by pesticide exposure among farmworkers; but conducting assessments of non-English-speaking, migratory populations may be difficult, there may not be qualified medical personnel and adequate equipment in rural areas, and the availability of monitoring devices may be a disincentive for employers to prevent exposures in the first place.

State Regulation

Under current law, States may set more stringent requirements for pesticide use than those provided in Federal statutes. Several States, notably Texas, California, and Washington, have initiated their own worker and public programs to fill the gaps in Federal regulations. Other States, for example, Iowa, Minnesota New Jersey, New York, North Carolina, and Wisconsin, have also taken steps to address critical needs at the State level. Nine States have laws requiring reporting systems for pesticide illness or pesticide use, although most of them are unenforced; 16 other States have limited forms of data collection; and 16 States have mandatory worker compensation programs for agricultural workers (53 FR 25973).

California has an extensive and well-funded pesticide registration and worker safety program that exceeds EPA standards in addressing local conditions and patterns of pesticide use. As mentioned earlier, California and Washington require reporting of pesticide illness. California enacted the Birth Defects Prevention Act of 1984 to require adequate data on the 200 most widely used pesticides

suspected to be hazardous to humans. This law prohibits the conditional registration of any new pesticide without complete and valid data on health effects. It also requires cancellation of any pesticide containing an active ingredient that causes significant adverse effects on health.

Texas has adopted several farmworker protection measures, including a 24-hour minimum reentry interval for all pesticides used on labor-intensive crops and certain prior notification and posting provisions for workers and other persons adjacent to treated fields. The most far-reaching development is Texas' Agricultural Hazard Communication (right-to-know) Law, the first such law in the Nation. It requires agricultural employers to provide their workers with information about the health risks of pesticides and ways to minimize these risks. Employers are required to maintain a list of all pesticides used and to make it accessible to workers, their physicians, and other designated representatives. Farmworker training (in a form and language understood by workers) is also guaranteed by this law, through crop sheets and other written and audiovisual materials (185,187).

New Approaches to Pest Control

The simplest way to protect farmworkers is to reduce the overall use of pesticides, particularly the most toxic ones. Movements to build sustainable agricultural systems based on limited use of pesticides and fertilizers and on integrated pest management (IPM) systems have been initiated in several States (see box 10-F). IPM relies on the coordination of a number of control tactics. It attempts to minimize the use of pesticides by making maximum use of biological controls (e.g., natural predators and parasites, disease-causing microorganisms, pheromones, and pest-resistant plants) and cultural controls (e.g., crop rotation and removal of crop residues that shelter pests after harvest). Chemical controls are used prudently, in conjunction with these other methods (176). IPM practices can potentially reduce pesticide use by as much as 50 percent (161).

Research on IPM techniques is slowly spreading to the more labor-intensive crops, but limited Federal funding has delayed implementation of this promising technology (187). The U.S. Department of Agriculture is researching and developing sustainable agriculture strategies which include IPM [14 U.S.C. 1463(C)]. In 1988, an estimated 8 percent of crop land (27 million acres) was enrolled in some 30 State IPM programs (104).

Examples of Neurotoxic Pesticides

The following discussion introduces several of the most common classes of pesticides known to have neurotoxic effects.

Cholinesterase-Inhibiting Insecticides

Organophosphorous and carbamate insecticides, the cholinesterase-inhibiting pesticides, represent a large and important class of neurotoxic substances (see table 10-3). Because of their widespread use and high toxicity at acute exposures, they are the most common cause of agricultural poisonings. Both affect target insects and humans by inhibiting acetylcholinesterase, an enzyme that breaks down the neurotransmitter acetylcholine. Inhibiting this enzyme creates a build-up of the transmitter, which causes nervous system dysfunction.

Some cholinesterase-inhibiting pesticides cause hyperactivity, neuromuscular paralysis, visual problems, breathing difficulty, abdominal pain, vomiting, diarrhea, restlessness, weakness, dizziness, and possibly convulsions, coma or death (see table 10-4) (102,141,195). The extensive literature on neurobehavioral toxicology in laboratory animals exposed to pesticides has been reviewed by others (14,29,34,41,71,189). The onset and duration of symptoms in acute poisoning of workers depends on the inherent toxicity of the insecticide, the dose, the route of exposure, and preexisting health conditions. Deaths have occurred in the past when workers were not treated properly for their exposure. The inhibition of acetylcholinesterase by both organophosphorous insecticides and n-methyl carbamates is reversible; however, inhibition caused by n-methyl carbamates is generally considered more readily and rapidly reversible than that caused by organophosphorous insecticides. For several of the organophosphorous insecticides, inhibition of acetylcholinesterase is so slowly reversible that an accumulation of the effect can occur. Once exposure ceases, however, full recovery usually results (102,106).

Some researchers have found delayed effects after an episode of acute organophosphorous insecticide poisoning: these include irritability, depression, mood swings, anxiety, fatigue, lethargy, difficulty concentrating, and short-term memory loss. These symptoms may persist for weeks and months after

Box 10-F-Organic Farming and Alternatives to Chemical Pesticides

In response to growing consumer demand, the cost of chemical fertilizers and pesticides, and evidence of risk to human health and the environment more farmers are turning to organic production. There is no single definition of organic farming, but it generally requires some degree of abstinence from use of chemical fertilizers and pesticides. In Texas, a farm is only certifiably organic if no pesticides have been used for 3 years and no chemical fertilizers have been used for 2 years, but standards may vary from State to State. Where there is no State regulation of organic farming, responsibility for setting standards usually falls to trade organizations, and there is frequent controversy over how strictly to limit pesticide use.

Organic farming is gaining the attention of consumers, growers, and legislators. A California trade organization reported that sales of organic produce in the United States doubled between 1983 and 1988, to $1 billion. A 1989 Harris poll reported that 84,2 percent of Americans would buy organic food if it were available, with 49 percent of those willing to pay more for it (organic produce currently costs between 5 and 15 percent more than crops on which pesticides are used). This public concern is reflected by distributors such as Sunkist Growers, Inc., and Dole Foods Co., who are beginning to grow organic produce, and by supermarkets, which are beginning to issue written policies requiring chemical-free produce from suppliers. State legislatures have been slower to address the pesticide problem. To date, only a small percentage of States has any regulations for organic farming, and only a few of these have certification programs.

Historically, organic farming has been more expensive because it is more labor-intensive, it is done on smaller farms, and it results in smaller yields. The resulting products, however, tend to have a higher profit margin than the more abundant crops grown on large farms where pesticides are used, As biological alternatives to pesticides are researched and developed, costs of alternative farming might be reduced further.

Despite the promises organic farming offers for human health and the environment there is awareness of its drawbacks even within the organic farming community. Complete rejection of chemical pesticides may reduce crop yields. Even some environmentally aware and health-conscious farmers agree that chemical pesticides are occasionally required.

Insufficient regulation of organic foods and farming methods is another drawback to organic farming. Apart from the lack of precise definition of what organic farming is, public safety maybe threatened by lack of enforced regulation of so-called organic produce, as well as a lack of testing at the supplier level to confirm that foods are free of toxic substances. According to the Consumers Union, most grocery stores rely on their suppliers' word that produce is pesticide-free, yet when that organization tested apples bought in stores which claimed not to sell apples treated with Alar, 55 percent of the apples contained it.

Rather than attempting to end all use of chemicals in agriculture, a solution may be found in integrated pest management or other alternative agriculture systems, which use chemicals discriminately, if at all, in conjunction with biological controls designed to fit local conditions. A National Research Council report released in September 1989 concludes that Federal farm subsidy programs encourage the use of chemical pesticides when nonchemical alternatives may be as or more effective. The report recommends that at least $40 million be allocated annually for research on alternative farming.

SOURCES: "Apple Grower Says Chemicals Sometimes Needed When All Else Fails," Associated Press, Apr. 3, 1989; D. Duston, "Eight in 10 Americans Prefer Chemical Free Food; Half Would Pay More," Associated Press, Mar. 19, 1989; "Entomologists Defend Chemicals as Necessary in Food Production," Associated Press, Mar. 25, 1989; "Farmers Hope to Replace Chemicals with Biological Fertilizers,' Associated Press, Apr. 3, 1989; P. Fikac, "Consumers Union: Ask Grocers About Reduce," Associated Press, Mar. 30, 1989; National Academy of Sciences, National Research Council, *Alternative Agriculture* (Washington, DC: National Academy Press, 1989); S.L. Nazario, "Big Firms Get High on Organic Farming: Pesticide Scare Reinforces Shift in Techniques,' *Wall Street Journal,* Mar. 21, 1989; ''Pesticide Scares Fuel Already Growing Organic Food Popularity," Associated Press, Apr. 10, 1989.

the initial exposure (17,82,83,137,147). Whether there are significant chronic effects of exposure to low-level organophosphorous and n-methyl carbamate insecticides (and, indeed, of exposure to pesticides in general) is a matter currently under debate. A number of researchers have observed persistent alteration of brain function (36,56,59,97, 141), while others have noted no long-term effects (10,13,29,153,155).

Some organophosphorous insecticides can produce delayed and persistent neuropathy by damaging certain neurons in the spinal cord and peripheral

Table 10-3-Organophosphorous and Carbamate Insecticides

Highly toxic[a]	Moderately toxic[a]
Organophosphorous insecticides	
tetraethyl pyrophosphate (TEPP)	bromophos-ethyl (Nexagan)
dimefox (Hanane, Pestox XIV),	leptophos (Phosvel)
phorate (Thimet, Rampart, AASTAR)	dichlorvos (DDVP, Vapona)
disulfoton[b] (Disyston)	ethoprop (Mocap)
fensulfothion (Dasanit)	demeton-S-methyl[b] (Duratox, Metasystox (i))
demeton[b] (Systox)	triazophos (Hostathion)
terbufos (Counter, Contraven)	oxydemeton-methyl[b] (Metasystox-R)
mevinphos (Phosdrin, Duraphos)	quinalphos (Bayrusil)
ethyl parathion (E605, Parathion, Thiophos)	ethion (Ethanox)
azinphos-methyl (Guthion, Gusathion)	chlorpyrifos (Dursban, Lorsban, Brodan)
fosthietan (Nem-A-Tak)	edifenphos
chlormephos (Dotan)	oxydeprofos[b] (Metasystox-S)
sulfotep (Thiotepp, Bladafum, Dithione)	sulprofos (Bolstar, Helothion)
carbophenothion (Trithion)	isoxathion (E-48, Karphos)
chlorthiophos (Celathion)	propetamphos (Safrotin)
fonofos (Dyfonate, N-2790)	phosalone (Zolone)
prothoate[b] (Fac)	thiometon (Ekatin)
fenamiphos (Nemacur)	heptenophos (Hostaquick)
phosfolan[b] (Cyolane, Cylan)	crotoxyphos (Ciodrin, Cypona)
methyl parathion (E 601, Penncap-M)	phosmet (Imidan, Prolate)
schradan (OMPA)	trichlorfon (Dylox, Dipterex, Proxol, Neguvon)
mephosfolan[b] (Cytrolane)	cythioate (Proban, Cyflee)
chlorfenvinphos (Apachlor, Birlane)	phencapton (G 28029)
coumaphos (Co-Ral, Asuntol)	pirimiphos-ethyl (Primicid)
phosphamidon (Dimecron)	DEF (De-Green, E-Z-Off D)
methamidophos (Monitor)	methyl trithion
dicrotophos (Bidrin)	dimethoate (Cygon, DeFend)
monocrotophos (Azodrin)	fenthion (mercaptophos, Entex, Baytex, Tiguvon)
methidathion (Supracide, Ultracide)	dichlofenthion (VC-13 Nemacide)
EPN	bensulide (Betasan, Prefar)
isofenphos (Amaze, Oftanol)	EPBP (S-Seven)
endothion	diazinon (Spectracide)
bomyl (Swat)	profenofos (Curacron)
famphur (Famfos, Bo-Ana, Bash)	formothion (Anthio)
fenophosphon (trichloronate, Agritox)	pyrazophos (Afugan, Curamil)

nervous system. The resulting muscle weakness may progress to paralysis. Onset is usually 2 to 4 weeks after the acute exposure (27,70,150). The initial symptoms of peripheral neuropathy are usually cramps in the calves and numbness and tingling in the feet. Increased weakness and flaccidity of the legs follows, accompanied by varying amounts of sensory disturbance. The arms may also be affected (106). There is no specific treatment, and the rate and extent of recovery vary considerably.

Organochlorine Insecticides

The organochlorine insecticides are chlorinated hydrocarbon compounds that act as central nervous system stimulants (see table 10-5). Organochlorines accumulate in both the environment and the body. In general, they are considered less acutely toxic than organophosphorous and n-methyl carbamate insecticides, but they have a greater potential for chronic toxicity. The prototype organochlorine, DDT, was discovered in 1939 and was used extensively in agriculture and against mosquitoes and other insects that transmit human disease before it was banned from most uses in the United States in 1972.

From 1940 through the 1970s, a number of other organochlorine compounds, such as aldrin, dieldrin, toxaphene, mirex, endrin, lindane, heptachlor, and chlordane, were widely used as insecticides. Following recognition of their accumulation in the environment and in human and animal tissues, and observation of some adverse effects on wildlife, most have been banned or severely restricted in use. For example, chlordane, introduced in 1947 and since then one of the most widely used of this family, was originally targeted by EPA for restricted use in 1974. It was banned for most uses except termite control in 1978 (102). A decade later, EPA banned almost all uses of chlordane.

The organochlorines are easily absorbed by inhalation or ingestion and may also be absorbed through the skin. They are generally distributed to fatty

Table 10-3-Organophosphorous and Carbamate Insecticides-Continued

Highly toxic[a]	Moderately toxic[a]
dialifor (Torak)	naled (Dibrom)
cyanofenphos (Surecide)	phenthoate (dimephenthoate, Phenthoate)
dioxathion (Delnav)	IBP (Kitazin)
mipafox (Isopestox, Pestox XV)	cyanophos (Cyanox)
	crufomate (Ruelene)
	fenitrothion (Accothion, Agrothion, Sumithion)
	pyridaphenthion (Ofunack)
	acephate (Orthene)
	malathion (Cythion)
	ronnel (fenchlorphos, Korlan)
	etrimfos (Ekamet)
	phoxim (Baythion)
	merphos (Fo!ex, Easy off-D)
	pirimiphos-methyl (Actellic)
	iodofenphos (Nuvanol-N)
	chlorphoxim (Baythion-C)
	propyl thiopyrophosphate (Aspen)
	bromophos(Nexion)
	tetrachlorvinphos (Gardona, Appex, Stirofos)
	temephos (Abate, Abathion)
Carbamate Insecticide	
aldicarb[b] (Temik)	dioxacarb (Elocron, Famid)
oxamyl (Vydate L, DPX 1410)	promecarb (Carbamult)
methiocarb (Mesurol, Draza)	bufencarb (metalkamate, Bux)
carbofuran (Furadan, Curaterr, Crisfuran)	propoxur (aprocarb, Baygon)
isolan (Primin)	trimethacarb (Landrin, Broot)
methomyl (Lannate, Nudrin, Lanox)	pirimicarb (Pirimor, Abel, Aficida, Aphox, Fernos, Rapid)
formetanate (Carzol)	dimetan (Dimethan)
aminocarb (Matacil)	carbaryl (Sevin, Dicarbam)
cloethocarb (Lance)	isoprocarb (Etrofolan, Ml PC)
bendiocarb (Ficam, Dycarb, Multamat, Niomil, Tattoo, Turcam)	

[a]Compounds are listed in order of descending toxicity. "Highly toxic" organophosphates have listed oral LD_{50} (median lethal dose) values (rat) less than 50 mg/kg; "moderately toxic" agents have LD_{50} values in excess of 50 mg/kg.
[b]These insecticides are systemic; they are taken up by the plant and translocated into foliage and sometimes into the fruit.

SOURCE: D.P. Morgan, *Recognition and Management of Pesticide Poisoning,* EPA pub. No. 540/9-S8-001 (Washington, DC: U.S. Government Printing Office, 19S9).

tissue, the liver, and the nervous system. Most are metabolized by the liver and excreted in urine. For some pesticides, accumulation in fat tissue occurs during chronic exposure, so elimination is slow. DDT, for example, is metabolized and excreted slowly and can still be found in the fat of most people exposed to it years after its use was terminated (62).

Acute intoxication from organochlorines can produce nervous system excitability, apprehension, dizziness, headache, disorientation, confusion, loss of balance, weakness, muscle twitching, tremors, convulsions, and coma. Uncontrolled seizures, respiratory problems, or both, may lead to brain or other organ damage. Children may be particularly sensitive to brain and nerve damage from organochlorine pesticides and may suffer from long-term behavioral and learning disabilities as a result of exposure (41).

One of the most serious cases of severe poisoning occurred in manufacturing workers handling chlordecone, commonly known as Kepone (see ch. 2). These workers suffered tremors, disturbances in vision, and difficulty in walking (156). As a result, this pesticide's registration was canceled by EPA in 1977 (42 FR 18855).

Fumigants

Fumigants-used to kill insects, insect eggs, and microorganisms-are the most acutely toxic pesticides used in agriculture. Because they are gases, fumigants are usually taken directly into the lungs, where they readily enter the blood and are distributed throughout the body. Although inhalation is the most serious source of exposure and can lead rapidly to death, absorption of fumigants through the skin can also be a significant hazard (103).

Table 10-4-Neurotoxic Effects of Acute Exposure to High Levels of Organophosphorous or Carbamate Insecticides

Function of nervous system when stimulated by acetylcholine	Effect of excessive stimulation of the nervous system
Activate salivary, sweat, and tear glands	Increased salivation, sweating, watering of eyes
Constrict bronchi	Tightness in chest, coughing and wheezing, difficulty breathing
Contract pupil of eye	Pinpoint pupils, blurring of vision
Control heart function	Abnormal heart beat, change in blood pressure
Increase spasms in digestive tract	Stomach cramps, nausea, vomiting, diarrhea
Increase spasms in urinary tract	Urinary frequency and incontinence
Activate skeletal muscles	Twitching, restlessness, tremulousness, impaired coordination, generalized muscle weakness, paralysis, and death or brain injury caused by asphyxiation after muscle paralysis
Alter brain function	Headache, giddiness, anxiety, emotional instability, lethargy, confusion; eventually severe central nervous system depression and coma

SOURCE: B.B. **Young, Neurotoxicity of Pesticides,"** *Journal of Pesticide* ***Reform 6:8-11,*** **1986.**

Table 10-5-Organochlorine Insecticides

Insecticide
endrin (Hexadrin)
aldrin (Aldrite, Drinox)
endosulfan (Thiodan)
dieldrin (Dieldrite)
toxaphene (Toxakil, Strobane-T)
lindane (gamma BHC or HCH, Isotox)
hexachlorocydohexane (BHC)
DDT (chlorophenothane)
heptachlor (Heptagran)
chlordecone (Kepone)
terpene polychlorinates (Strobane)
chlordane (Chlordan)
dicofol (Kelthane)
mirex (Dechlorane)
methoxychlor (Marlate)
dienochlor (Pentac)
TDE (DDD, Rhothane)
ethylan (Perthane)

SOURCE: D.P. Morgan, Recognition *and Management of Pesticide Poisoning,* EPA pub. No. 540/9-88-001 (Washington, DC: U.S. Government Printing Office, 1989).

Fumigants have caused severe illness and death in human beings (11,63,81,132). Poisoning initially causes headache, nausea, vomiting, and dizziness, followed by drowsiness, fatigue, slurred speech, loss of balance, and disorientation. In severe poisonings, seizures, loss of consciousness, respiratory depression, and death may occur. Tremors and generalized seizures may also occur, particularly from methyl bromide poisoning.

Methyl bromide, one of the most widely used pesticides in the United States, is a colorless gas at room temperature. It has a faint, somewhat agreeable odor, making it difficult to detect, even at toxic levels (127). This pesticide has caused death and severe neurotoxic effects in fumigators, applicators, and structural pest control workers. Acute exposure to methyl bromide can result in visual and speech disturbances, delirium, and convulsions. Both acute and chronic poisoning from methyl bromide may be followed by prolonged, and in some cases permanent, brain damage marked by personality changes and perception problems. Chronic exposure can result in progressive peripheral neuropathy, with loss of motor control, numbness, and weakness (4,63).

Chlorophenoxy Herbicides

Chlorophenoxy herbicides include 2,4-dichlorophenoxyacetic acid(2,4-D),2,4~-trichlomphenoxyacetic acid (2,4,5-T), 2-methyl-4 -chlorophenoxyacetic acid (MCPA), and 2,4,5 -trichlorophenoxypropionic acid (Silvex). These herbicides were among the most widely used until EPA suspended many of their uses because of potential adverse effects on human health (43 FR 17116). The chlorophenoxy herbicides continue to be used widely in forestry and weed control in agricultural and urban settings. Farmworkers can be exposed to these pesticides during mixing and loading or by drift from nearby applications. Although these compounds are readily metabolized and excreted and are of relatively low toxicity to mammals, they are often contaminated with dioxins, which may be toxic themselves (102). There are more than 75 different dioxin isomers, but TCDD, a contaminant of 2,4,5-T, is believed to be the most toxic (68).

Most current knowledge of the effects of TCDD on humans comes from overexposures of workers manufacturing 2,4,5-T or the compound from which it is derived (61,66,68). Acute exposure to high doses has led to peripheral neuropathy, sometimes accompanied by difficulty in walking and coordinating the legs. Irritability, insomnia and hypersomnia, lethargy, impotence, and psychiatric disturbances have also been reported in cases of acute exposure (102). Peripheral neuropathy resulting from dermal

absorption and death resulting from ingestion have been reported for 2,4-D (102).

The most notorious chlorophenoxy herbicide is the defoliant Agent Orange. Agent Orange consists of a 1:1 mixture of 2,4,5-T and 2,4-D and was widely used in Vietnam from 1962 to 1970. A number of adverse effects on Vietnamese and on American soldiers in Vietnam have been alleged. A recent report indicates that the probability of exposure of U.S. veterans was small (162), and whether Agent Orange was the cause of the alleged health effects is still unresolved (102).

Pyrethroids

Pyrethroids, a group of insecticides, are highly toxic to insects but less toxic to mammals, which metabolize and excrete them quickly. Pyrethroids act by altering the flow of sodium ions through the nerve cell membrane, resulting in repeated firing of the nerve cell (106).

Because pyrethroids appear to be less acutely toxic than other insecticide groups, their use is likely to increase. In response to the observation of axonal swelling in rats subsequent to pyrethroid ingestion, EPA requires a special new pathological evaluation as part of the 90-day rodent feeding study from all companies attempting to register a pyrethroid (37).

Summary and Conclusions

Approximately 1 billion pounds of pesticides products, made up of 600 active pesticide ingredients, are used annually in agriculture in the United States (102). Many of these active pesticide ingredients have never been tested for potential neurotoxic or neurobehavioral effects, damage to the reproductive system, or other effects on human health. Historically, few pesticides have been banned or restricted by EPA.

Although everyone is exposed to low levels of pesticides in food and water, an estimated 2.7 million migrant and seasonal farmworkers face greater risk because they are regularly exposed to higher levels of pesticides and because existing protections do not always cover them adequately. Pesticide applicators, loaders, and mixers, as well as nursery, greenhouse, forestry, and lawn care workers, may be exposed to particularly high levels of pesticides as well. Children, who constitute a significant proportion of the agricultural work force, are especially vulnerable because their nervous systems are not fully developed. The majority of pesticides used are organophosphorous and n-methyl carbamate insecticides, both of which are neurotoxic. They can produce acute effects (ranging from moderate symptoms to death) and perhaps chronic effects as well, although the data are inconclusive. Some organophosphorous insecticides can also cause delayed damage to the peripheral nervous system.

It is not possible to estimate accurately the extent of illness among farmworkers because there is no national pesticide illness reporting system or worker monitoring program. Extrapolations by others from available data suggest a prevalence of more than 300,000 pesticide-related illnesses among farmworkers, although only a small percentage of these cases are reported (31). The total number of worker deaths and the extent of chronic health problems caused by exposure to pesticides are also unknown.

Limiting the use of neurotoxic pesticides would be a straightforward way to control exposure. Integrated pest management systems offer alternative approaches to pest control and minimize the use of pesticides.

More research is needed to understand the neurotoxic effects of new and existing chemicals and to protect agricultural workers from them. EPA's pesticide registration review should require information on pesticide neurotoxicity based on the most current knowledge of dose-response relationships, mechanisms of action, and structure-activity relationships. Premarket testing could include effects on learning, memory, conditioned behavior, and emotional disorders, rather than being limited to motor function. More information is needed on the long-term effects of pesticides on the fetus, on children, and on the aged.

The need for epidemiological studies of the effects of pesticides on agricultural workers is critical. Reporting and monitoring procedures could be established and enforced to provide more accurate information on the prevalence and incidence of pesticide illness; furthermore, to facilitate reporting, both physicians and workers could be better educated in the signs and symptoms of pesticide illness.

In the near term, several actions could be taken to provide greater protection to agricultural workers. Establishing more specific reentry intervals, which take into account the chemical and neurotoxic properties of certain chemicals, would be a positive

step forward. EPA might also adjust its risk-benefit assessment criteria for pesticide registration to include the costs of pesticide poisoning of workers. Workers could be regularly monitored for exposure to pesticides, provided with appropriate protective clothing, trained in safe application for specific circumstances, educated about the health effects of exposure, and better informed about the chemicals they use under right-to-know laws. Mandatory recordkeeping on pesticide application could be included in the latter to ensure that workers can obtain information about previous exposures. EPA has proposed decontamination facilities and emergency provisions for all workers, but more could be done to prevent pesticide poisoning. Since the chronic effects of pesticide poisoning remain unknown, efforts may be best directed toward prevention.

EXPOSURE TO ORGANIC SOLVENTS IN THE WORKPLACE

According to the National Institute for Occupational Safety and Health (NIOSH), approximately 9.8 million workers are exposed to solvents every day through inhalation **or skin contact (166).** Acute exposure to organic solvents can affect an individual's manual dexterity, speed of response, coordination, and balance; it can also produce feelings of inebriation. Chronic exposure to some organic solvents can result in fatigue, irritability, loss of memory, sustained changes in personality or mood, and decreased learning and concentration abilities; in some cases, structural changes in the nervous system are apparent.

Organic solvents are a group of simple organic liquids that are volatile; that is, in the presence of air they change from liquids to gases and therefore are easily inhaled. Figure 10-6 illustrates the general classes of organic solvents. Solvents usually serve one of two general functions. They may be used in separation processes to selectively dissolve one material from a mixture, or they may act as a processing aid, facilitating fabrication of a material (usually a polymer) by reducing its viscosity (188). They are components of a variety of products, including paints, paint removers and varnishes; adhesives, glues, coatings; decreasing and cleaning agents; dyes and print ink; floor and shoe creams, polishes, and waxes; agricultural products; pharmaceuticals; and fuels. In 1984, approximately 49 **million tons of industrial organic solvents were produced in the United States (167).**

There are many occupations in which workers are exposed to solvents. For example, painters may come in contact with methyl alcohol, acetone, methylene chloride, toluene, and complex mixtures of petroleum products. Depending on the exposure levels in air, house painters may experience a variety of adverse effects, including fatigue, impaired memory, difficulty in breathing, slurred speech, nausea, dizziness, difficulty in concentrating, and dermatitis. Some researchers believe that painters may develop a "psycho organic syndrome" from exposure to chronic low levels of solvents (49,58). The syndrome is characterized by fatigue, difficulty concentrating, learning, and remembering, and personality changes (32).

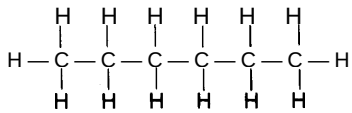
In order to protect workers, NIOSH recommends that employers educate them about the materials to which they are exposed, the potential health risks involved, and work practices that will minimize exposure to these substances (166). NIOSH also recommends that employers assess the conditions under which workers may be exposed to solvents, develop monitoring programs to evaluate the extent of exposure, establish medical surveillance for any adverse health effects resulting from exposure, and routinely examine the effectiveness of the control methods being employed in order to reduce exposures to the permissible exposure limits (PELs) mandated by OSHA. There are three basic methods for minimizing worker exposure to organic solvents: using effective engineering controls, isolating workers from the source of exposure, and using personal protective equipment (8).

Organic solvents are of particular concern because most are toxic in different ways and to varying degrees and many are also flammable. The increase in the number of available organic solvents and the development of new processes utilizing them present major occupational health challenges (8,166).

Some organic solvents are also subject to abuse by inhalation. The extent of this abuse is much greater than is generally recognized. The National Institute on Drug Abuse reports that the lifetime incidence of solvent abuse among seniors in high school (thus excluding dropouts) is exceeded only by alcohol, tobacco, marijuana, and stimulants (113). The abuse of solvents by Hispanic and Native Americans is widespread in some regions, exceeded only by

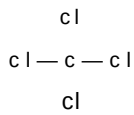
Figure 10-6-Classes of Organic Solvents

Allphatic hydrocarbons
(Acyclic)
Straight or branched chains of carbon and hydrocarbons



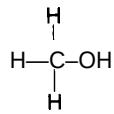
n- Hexane

Halogenated hydrocarbons
A halogen atom has replaced one or more hydrogen atoms on the hydrocarbon



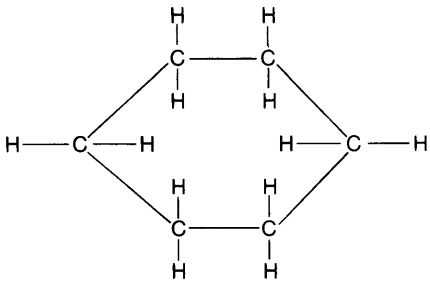
Carbon tetrachloride

Alcohols
Contain a single OH group



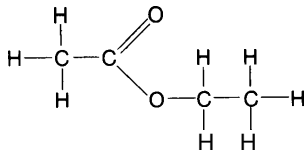
Methyl alcohol

Cyclic hydrocarbons
(cycloparaffins, naphthenes)
Ring structure saturated and unsaturated with hydrogen



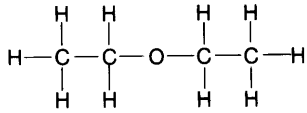
Cyclohexane

Esters
Formed by interaction of an organic acid with an alcohol



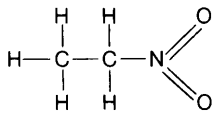
Ethyl acetate

Ethers
Contain the C–O–C linkage



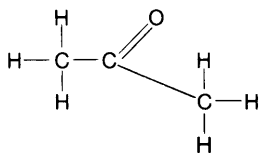
Ethyl ether

Nitrohydrocarbons
Contains an NO, group



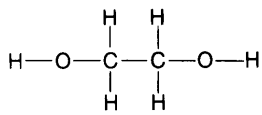
Ethyl nitrate

Ketones
Contain the double bonded carbonyl group, C = O, with 2 hydrocarbon groups on the carbon



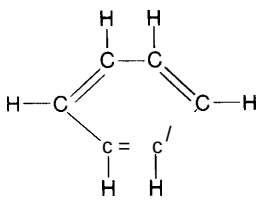
Acetone

Glycols
Contain double OH groups



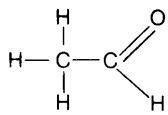
Ethylene glycol

Aromatic hydrocarbons
Contain a 6-carbon ring structure with one hydrogen per carbon bound by energy from several resonant forms



Benzene

Aldehydes
Contain the double bonded carbonyl group, C = O, with only one hydrocarbon group on the carbon



Acetaldehyde

SOURCE: Alliance of American Insurers, ***Handbook of* Organic *Industrial Solvents,* 6th** edition, 1987, pp. 1-20.

alcohol abuse (l). Such exposures greatly exceed those encountered in the workplace and can be associated with severe and irreversible toxicities.

Uptake, Distribution, and Elimination of Solvents

Solvents may enter the body by inhalation, dermal contact, or ingestion. The hazards associated with dermal exposure and ingestion can be severe; in fact, numerous fatalities have resulted from exposure to methanol by these routes. However, because of the volatility of these chemicals, a major route of exposure is inhalation. Exposure to the skin is another important route. For example, immersion of hands in methylene chloride causes neurological damage (159), and carbon disulfide produces shaking of the hands and loss of feeling (89).

The amount of the solvent entering the body depends on such factors as route of exposure, the concentration of the solvent in the air, the volubility of the solvent in blood, and the amount of physical work being performed at the time of exposure. A sedentary worker on a factory floor will absorb less solvent than a worker engaged in a vigorous physical task because the latter will be inhaling more rapidly and deeply (thereby moving more solvent to the site of uptake in the lungs) and more blood will be traveling through the lungs (carrying the solvent throughout the body).

Some solvents tend to be distributed unequally among the organs of the body. This is both because the volubility of a particular solvent varies with different tissues and because the blood supply to tissues varies greatly. Thus, an organ like the brain, with its high fat content and very rich blood supply, achieves high levels of solvents quickly. Given a constant concentration of solvent in the air, the amount of solvent present in body tissues eventually reaches a plateau in each tissue, but the time required to achieve that plateau varies among tissues and among individuals.

At the same time that the body is absorbing solvents, it is working to eliminate them. If exposure ceases or is reduced, the solvent begins to be exhaled, or "blown off. " Enzymes may change the structure of the solvent, making it more water soluble and enabling the kidneys to eliminate it. The metabolism of solvents can be a two-edged sword, however, since the metabolize may be more toxic than the parent solvent. Mixtures of solvents or

Photo credit: United Automobile, Aerospace, and Agricultural Improvmnt Winkers of America-UAW Public Relations Department

Respirators may be useful in minimizing exposure to solvent vapors when engineering or work practice controls are inadequate.

industrial grade solvents may be more toxic than pure solvents, either because of toxic contaminants or because of chemical interactions.

Neurological and Behavioral Effects

All solvents are soluble in fat and will at some level of exposure produce effects on the central nervous system (35). For a wide variety of drugs and chemicals, the more soluble the chemical is in brain membranes, the more potent it is and the longer it acts.

Interest in the effects of solvents on the central nervous system dates back to the early search for anesthetics, when many agents were examined. Short-term exposures at low toxicity may produce mucous membrane irritation, tearing, nasal irrita-

tion, headache, and nausea (35). With repeated inhalation of high levels of solvents, a state of severe narcosis may be produced; at lower levels, the effects resemble those of alcohol. There may be initial euphoria, loquaciousness, and excitement, followed by confusion, dizziness, headache, motor incoordination, ataxia, unconsciousness, and death. These so-called nonspecific narcotic effects of solvents are the major reason they are regulated in the workplace; they can impair work performance and the ability to avoid hazards (35).

Toxicity studies and health problems in the workplace have revealed other effects that are specific to individual solvents or classes of solvents. For example, neuropathies may result from chronic exposure to hexane, methyl-n-butyl ketone, and related solvents. This disorder (sometimes referred to as hexacarbon neuropathy) is characterized by numbness in the hands and feet and may progress to muscle weakness and lack of coordination (152). Some solvents produce seizures and convulsions on acute exposure, for example, such alkylcycloparaffins as methylcyclopentane and methylcyclohexane (79, 80,129). Indeed, epileptic seizures in the workplace may be mistakenly attributed to an undiagnosed neurological defect of the worker rather than to a chemical exposure.

Adverse effects on the inner ear may also be caused by exposure to solvents. For example, exposure to high levels of alkylbenzenes such as toluene and xylene can damage the inner ear, leading to high-frequency hearing loss (128,130,133). Dizziness and vertigo have been reported following acute exposure to a variety of solvents. Exposure may also adversely affect various visual functions and the sense of smell (43,94,95).

Some solvents may cause emotional disorders. Carbon disulfide can produce a raging mania and has been associated with increased risk of suicide (92). In 1902, Thomas Oliver described his visits to India-rubber factories in London and Manchester, noting "the extremely violent maniacal condition into which some of the workers, both female and male, are known to have been thrown. Some of them have become the victims of acute insanity, and in their frenzy have precipitated themselves from the top rooms of the factory to the ground" (122).

Other disorders associated with exposure to solvents include sleep disturbances, nightmares, and insomnia (18,190). Trichloroethylene or its contaminants may damage facial nerves and produce facial numbness (20). Severe brain injuries (chronic encephalopathies) have been documented following prolonged exposures to high levels of solvents, such as during deliberate self-administration of solvents. This has produced concern about the likelihood of such effects occurring in the workplace. Prolonged exposure to styrene may produce impairments in perceptual speed and accuracy, memory, and cognitive performance (60).

The Solvent Syndrome: A Current Controversy

There is considerable evidence that toxic encephalopathy may be caused by high-level, prolonged, and repeated exposure to some organic solvents (158). Encephalopathy consists of a wasting of brain matter, which leads to expansion of the fluid-filled cavities in the brain. The syndrome is associated with motor disorders and impaired mental function. Several Scandinavian countries have identified a new disease entity, a toxic encephalopathy following chronic solvent exposure, and compensate workers who develop it at the workplace (52). However, the studies used to document the syndrome's existence are the subject of controversy (45,58). A multinational study of workers exposed to solvents is being funded by a consortium of industrial groups (158). In studies of this type, many variables may obscure the detection of an effect or erroneously suggest its existence. These include age, concurrent exposure to other chemicals, excessive alcohol intake, drug abuse, and socioeconomic status. In fact, a recent reanalysis of test data failed to confirm an earlier report of a ''chronic painters' syndrome" with dementia (54). Many studies suffer from not having extensive documentation of workplace exposure levels. It was having such information on exposure that enabled investigators to do landmark studies of carbon disulfide neurotoxicity. These studies revealed differences in suicide rates among workers in a rayon factory as a function of work assignment and associated carbon disulfide exposure within the plant (92).

Although painters are exposed for long periods of time to solvents, their exposure is moderate in comparison to that of solvent abusers, who routinely expose themselves to very high concentrations. The injuries to the nervous system suffered by solvent abusers are unequivocal and severe (53,78,1 38,142). A scientific conference recommended directions that human and animal research should take (9). The

lack of an animal model inhibits the normal regulatory process of hazard identification, risk assessment, and risk management. Just as prudent regulatory actions are undertaken to minimize the risk of cancer in humans when tumors are observed in laboratory animals, a nervous system injury or behavioral disorder identified in laboratory animals could be the basis for regulation to reduce the likelihood of injury to the human nervous system. To date, little effort has been devoted to developing an animal model of the solvent syndrome.

Health Protection

There are several methods for controlling worker exposure to organic solvents, including worker isolation, use of engineering controls, and personal protective equipment. Proper maintenance procedures and education programs are important ingredients of protection programs. OSHA regulations require that workers be informed about the hazards associated with the chemicals present in the workplace (29 CFR 1987 ed. 1910.1200). NIOSH recommends that employers establish a medical surveillance program to evaluate both the acute and chronic effects of exposure to organic solvents and that workers undergo periodic medical examinations (166). Both physicians and workers should be given information regarding the adverse effects of exposure to organic solvents and an estimate of the worker's potential for exposure to the solvents. This information should include the results of workplace sampling and a description of protective devices that the worker may be required to use (166).

Contaminant Controls, Worker Isolation, and Personal Protective Equipment

The primary means of preventing contamination is by applying appropriate engineering controls. These may be necessary to eliminate the potential for exposure and to prevent fires and explosions. Achieving an adequate reduction of exposure to a solvent depends on the construction and maintenance of the engineering control applied to the system, the exposed liquid surface, and the temperature and vapor pressure of the solvent. Closed system operations are the most effective method of minimizing worker exposure. Closed system equipment can be used for manufacturing, storing, and processing organic solvents. As an alternative, workers can be isolated from the process by being enclosed in a control booth.

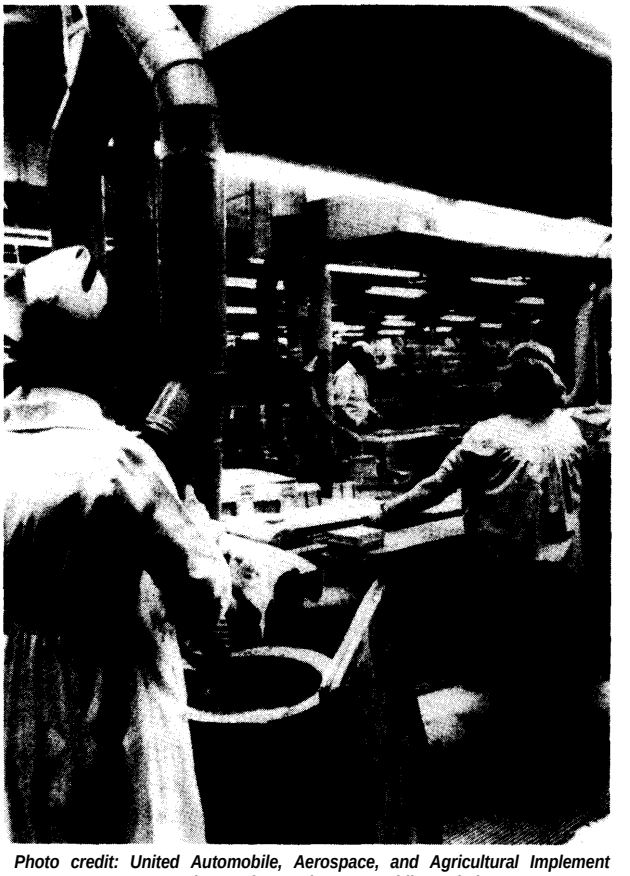


Photo credit: United Automobile, Aerospace, and Agricultural Implement Workers of America-UAWPublic Relations Department

Millions of workers come into contact with toxic substances every day through inhalation or skin contact, Many of the substances are known to be or are potentially neurotoxic.

When a closed system cannot be implemented, exhaust fans can be used to direct vapors away from workers and to prevent the contaminated air from recirculating in the workplace (166). In addition, personal protective equipment may be necessary (see box 10-G).

Respirators may be needed to minimize exposure when engineering or work practice controls are inadequate for this purpose. Respirator may be required for protection in certain situations such as implementation of engineering controls, some short-duration maintenance procedures, and emergencies. The use of respiratory protection requires that the plant or company institute a respiratory protection program (166). Direct contact of organic solvents with the skin can be prevented by wearing solvent-resistant gloves, aprons, boots, or entire work suits. Depending on the workplace and on the hazardous

Box 10-G-Engineering Controls v. Personal Protective Devices

The scientific and technical community has generally preferred engineering controls, that is, changes in the design of the physical environment and equipment used in the workplace, to personal protective devices, such as respirators, because:

- workers are erratic in their use of personal protective devices;
- such devices are cumbersome and, in the case of respirators, even the most conscientious worker may have difficulty ensuring an effective seal between face and mask;
- personal protective devices themselves require maintenance, such as periodic replacement of air filters; and
- it can be difficult to know when a personal protective device has failed.

The Occupational Safety and Health Act (OSH Act) was designed to decrease worker exposure to toxic substances and to provide information to employees about remaining occupational health risks. Maintaining low levels of toxic substances in the workplace through engineered controls has historically been given priority over the use of personal protective devices, except in those cases where it is not feasible to use engineering controls to reach the OSH Act exposure limit. Engineering controls are generally more expensive than personal protective equipment, and small plants and businesses often cannot afford to make expensive changes.

The mandate of the Act, however, has been to maintain a safe workplace, regardless of the size of the business. If it is not feasible to institute engineering controls or to engineer down to what the Act determines to be a safe level of exposure, then personal protective equipment is an acceptable choice. Recent changes in the existing rule on methods of compliance (54 FR 23991) allow respiratory protection to be used in lieu of administrative or engineering controls under the following circumstances **(54 FR 23991):**

1. during the time necessary to install or implement feasible engineering controls;
2. where feasible engineering controls result in only a negligible reduction in exposures;
3. during emergencies, recovery operations, unscheduled repairs, shutdowns, and field situations where there are no utilities for implementing engineering controls;
4. operations requiring added protection where there is a failure of normal controls; and
5. entries into unknown atmospheres (e.g., entering vessels, tanks, or other confined spaces for cleaning).

In addition to regulatory requirements, there are important ethical arguments about engineering controls versus personal protective devices. What are the important values at stake? The health, well-being, and autonomy of the worker are obviously important. (Autonomy refers to behaviors that reflect the capacity of competent adult individuals to formulate life plans and make decisions freely, without coercive influences.)

The Act is designed to ensure a safe and healthful workplace by setting exposure levels and establishing standards on behalf of the worker. One can argue that the option of using personal protective devices gives the worker a choice in determining the extent of exposure to hazardous substances. Yet it is difficult to imagine why a worker would prefer to use a cumbersome device like a respirator rather than have the workplace and equipment engineered to be safer. In situations in which it is not feasible to engineer safe levels of exposure, the use of personal protective devices may be the only option for working safely. On occasion, employees may decide to work in an area that requires the use of personal protective equipment in order to gain a particular type of work experience or to make more money. From an ethical standpoint however, circumstances in which there exists some coercive element are objectionable.

On balance, the interests of the worker seem to be best served by the use of engineering controls that lower levels of exposure to toxic substances for most workers most of the time. Some employers, however, can and do continue to argue that the greatest good for the greatest number requires at least some reliance on the use of personal protective devices.

SOURCE: Office of Technology Assessment, 1990.

properties of the substance, face shields and safety goggles may be required.

OSHA Regulations

The principal reason for the enactment of the Occupational Safety and Health Act of 1970 was to protect workers from occupational safety and health hazards. To accomplish this goal, OSHA sets minimum standards for working conditions. Hazards not mentioned in the standards are covered by the "general duty clause," which requires each employer to maintain a workplace ''free from recognized hazards. " All work environments must meet the regulations and standards set by the law (29 CFR 1987 ed. 1900-1910). OSHA has the authority to conduct inspections, determine compliance with the standards, and initiate enforcement actions against employers who are not in compliance.

If an inspector documents a violation, it is reported to the OSHA area director, who then informs the employer of the citation or proposed penalty. If the employer disagrees with the action, he or she may contest it by informing the Department of Labor within 15 working days of the citation. When notification is received that an action is being contested, the Occupational Safety and Health Review Commission is notified, and this review commission assigns the hearing to an administrative law judge. Following the hearing, the judge may issue an order to affirm, modify, or vacate the citation or proposed penalty. The order is final after 30 days unless the commission reviews the decision. If the employer decides not to contest the citation, he or she must correct the situation that is in violation of the standards. If the employer cannot do so within the proposed abatement period, an extension maybe requested. The law provides for fines of up to $1,000 for each violation and up to $10,000 if the violation is willful or repeated (29 CFR 1987 ed. 1903).

The PEL Controversy

OSHA recently published a revised standard that increased the protection of workers by implementing new or revised PELs for 428 toxic substances, including a number of organic solvents (53 FR 20960-20991). The final standard was published in January 1989. According to the Department of Labor, the new limits will reduce considerably the risk of illness, including cancer, by using the force of law to ensure that workers are not exposed at levels above the new PELs. The final rule was effective in March 1989, and the start-up date for compliance with any combination of controls (e.g., personal protective equipment) was September 1989, whereas compliance with engineering controls is delayed until December 31, 1992, or in some cases a year later.

The PELs are listed in the so-called Z tables in the OSHA regulations (29 CFR 1910.1000). The recent changes include revising the PELs, adding short-term exposure limits (STELs) to complement the 8-hour time-weighted average (TWA) limits, and where necessary designating skin or ceiling limits for the substances (54 FR 2332-2403). According to the Department of Labor:

> OSHA has reviewed health, risk and feasibility evidence for all 428 substances for which changes to the PEL were considered. In each instance where a revised or new PEL is adopted, OSHA has determined that the new limits substantially reduce a significant risk of material impairment of health or functional capacity among American workers, and that the new limits are technologically and economically feasible. This determination has been based on further review of the material discussed in the Proposal, public comments and a detailed review of the entire record for this rulemaking (54 FR 2334).

The new rule established lower exposure limits for approximately 212 substances already regulated by OSHA. PELs would be established for the first time for another 164 substances. A large number of these are established to prevent adverse effects on the nervous system. According to the Department of Labor:

> . . . Benefits will accrue to approximately 4.5 million workers who are currently exposed in excess of the PEL and are expected to include over 55,000 occupational illness cases, including almost 24,000 lost workdays annually. If not prevented, these illnesses would eventually result in approximately 700 fatalities per year. . . . The annual cost is approximately $150 per worker protected, and is never more than a fraction of 1 percent of sales and less than 2 percent of profits (usually substantially less) except for a very few segments . . . (54 FR 2335).

The approach used to develop the regulations of the new PELs has been controversial (54 FR 3272-2377). In evaluating the PELs, OSHA used the threshold limit values (TLVs) published by the American Conference of Governmental Industrial Hygienists (ACGIH) published in 1988 and the

recommended exposure limits (RELs) developed by NIOSH as its starting point. The agency compared the PELs to the Z tables and to the TLVs. If the two differed, the PEL was evaluated for revision. The agency first determined if the TLVs and RELs were similar. If they were, or if there was no REL, then OSHA studied the TLVs. If the TLV and REL differed significantly, OSHA examined the scientific basis of each recommendation and determined which was more appropriate. According to OSHA:

> In its review, OSHA determined frost whether the studies and analysis were valid and of reasonable scientific quality. Second, it determined, based on the studies, if the published documentation of the REL or TLV would meet OSHA's legal requirements for setting a PEL. Thus, OSHA reviewed the studies to see if there was substantial evidence of significant risk at the existing PEL or, if there was no PEL, at exposures which might exist in the workplace in the absence of any limit. Third, OSHA reviewed the studies to determine if the new PEL would lead to substantial reduction in significant risk. If this was so, and if the new PEL was feasible, OSHA proposed the new PEL (54 FR 2372).

The TLVs, RELs, and old and new PELs of some selected solvents are listed in table 10-6.

Table 10-6-Representative Exposure Limits to Solvents

	Measure (ppm)			
Solvent	REL[a]	TLV[b]	Old PEL[c]	New PEL
Toluene	100	100	200	100
Xylene	100	100	100	100
Cresol	2.3	5d	5d	4
Acetone	250	750	1,000	750
Styrene	50	50	100	50
Tetrachloroethylene	—	50	100	25
Methyl chloroform	200	350	350	350
Allyl chloride	1	1[d]	1	1
Furfuryl alcohol	50	10	50	10
Ethylene dichloride	1	10	50	1
Benzene	0.1	10	10	10
Carbon disulfide	1	10	20	4
Trichloroethylene	25	50	100	50
Chloroform	—	10	50	2

[a]RELs (recommended exposure limits) are set by the National Institute for Occupational Safety and Health.
[b]TLVs (threshold limit values) are set by the American Conference of Governmental Industrial Hygienists.
[c]PELS (permissible exposure limits) are set by the Occupational Safety and Health Administration.
[d]The ACGIH designation "skin"(s) refers to the potential contribution Of exposure by the cutaneous route, including mucous membranes and eyes.

SOURCES: 54 FR 2332-2983; 29 CFR 1987 ad. 1910.1000; R.B. Dick, "Short Duration Exposures to Organic Solvents: The Relationship Between Neurobehavioral Test Results and Other Indicators," *Neurotoxicology and Teratology* 10:39-50, *1988;* U.S. Department of Health and Human Services, National Institute for Occupational Safety and Health, "Organic Solvent Neurotoxicity," *Current Intelligence Bulletin* 48:1-39, 1987.

The final standard has been controversial because it represents a substantially different approach to OSHA rule-making. Until this action, OSHA addressed toxic substances individually, a process that produced standards for only 24 substances in 17 years. In this single rule-making, however, OSHA established new exposure limits for 376 toxic substances by adopting the TLVs published by ACGIH. Industry and several unions expressed concern that OSHA was delegating its regulatory authority to a nongovernment organization and that in some cases TLVs are not based on recent studies (25,139). The extent of corporate influence on TLVs has also been the subject of debate (25). Some unions contended that ACGIH is dominated by industry and that OSHA's action subverts the activities of NIOSH (139).

NIOSH offers RELs for chemicals following careful review of available data and bases its recommendations solely on the chemical's effects on health. However, OSHA, by law, cannot enforce a standard with a recommended exposure limit (REL) that is not technologically or economically feasible. These constraints often prevent OSHA from lowering a limit on the basis of health considerations alone, that is, on NIOSH's recommendation.

Public comments submitted to OSHA on the PEL proposal were in broad agreement that the PELs needed updating; however, many thought the project was being undertaken hastily and that the public interest would not be well served by such a major procedural change. Some commentors recommended that periodic updates be conducted on a more frequent and less harried basis.

By using the ACGIH list of TLVs as the basis for its selection, OSHA was able to save a great deal of the time it would have taken to address these chemicals through the usual regulatory procedures. OSHA is constrained to conduct a number of analyses by statute or executive order, including extensive economic analyses. By its own admission, OSHA states that it follows more extensive and elaborate administrative procedures than other health regulatory agencies:

> ... Clearly an improved approach to regulation is needed to solve this problem in a reasonable time period. OSHA's traditional approach, which has

> permitted on the average less than two major health regulations per year, is not adequate to address the backlog of at least 400 chemicals generally recognized as needing new or lower exposure limits. OSHA has reviewed the law, Congressional intent, its history, and the recommendations of experts . . . [and has] concluded that this approach has a greater health benefit and will prevent more deaths and various deleterious health effects, than could be achieved by allocating the same resources to comprehensive rulemaking for a small group of substances. . . (54 FR 2370).

The advisability of using the recommended exposure standards of a private organization instead of NIOSH is likely to be a subject of continuing controversy in the occupational health arena.

Summary and Conclusions

Organic solvents and mixtures of solvents with or without other toxic substances are widely used in the workplace. It is estimated that 9.8 million workers come into contact with solvents every day through inhalation or skin contact. Some solvents may profoundly affect the nervous system. Acute exposure to solvents can affect an individual's manual dexterity, response speed, coordination, or balance. Chronic exposure may lead to reduced function of the peripheral nerves and such adverse neurobehavioral effects as fatigue, irritability, loss of memory, sustained changes in personality or mood, and decreased learning and concentration abilities.

In order to protect workers, OSHA requires that employers inform and educate workers about the potential health risks of the materials to which they are exposed and adopt work practices that minimize exposure to hazardous substances. NIOSH recommends that employers assess the conditions under which workers may be exposed to solvents, develop monitoring programs to evaluate the extent of exposure, establish medical surveillance for any adverse health effects, and routinely examine the effectiveness of the control methods.

OSHA recently updated the permissible exposure limits for 428 substances, many of them solvents. The new ruling established lower PELs for 212 substances already regulated by the agency. PELs were also established for the first time for another 168 substances, while existing limits for 25 substances were reaffirmed. This marks the first time in 17 years that a new set of exposure standards has been established. The mechanism by which the new PELs were set, however, is the subject of controversy.

For many companies, meeting the new standards may require stricter engineering controls or more frequent use of respirators and other personal protective devices, or both. OSHA requires companies to educate workers about the hazards of the substances to which they are exposed, to institute control methods to prevent exposure, and to formulate plans or procedures to maintain compliance with the new rulings.

There is insufficient information available to regulatory agencies to distinguish dangerous solvents from ones that are not dangerous. Creative approaches are needed to protect workers while avoiding unnecessary and overly burdensome regulations. To fill this need, research programs in academia, industry, and government will have to be expanded significantly. If NIOSH is to play an important future role in the development and analysis of information on safe exposure levels for solvents, then additional resources will be required and the Institute will have to make a commitment to focus more attention on the neurological and behavioral effects of solvents. Improvement in the development of toxicity standards will require a substantially closer working relationship between OSHA and NIOSH.

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