OCCUPATIONAL SAFETY AND HEALTH HAZARDS IN AGRICULTURE: A REVIEW OF THE LITERATURE

FINAL REPORT

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CHAPTER I. INTRODUCTION

Since passage of the North American Free Trade Agreement (NAFTA) in 1993, the United States, Canada, and Mexico have been working together to improve the health and safety of workers on both sides of the border. To that end, representatives from the three countries have met to develop and refine an Action Plan for coordinating tripartite activities in the area of workplace safety and health. One of the activities being conducted by OSHA is this review of the literature on safety and health hazards in agriculture. Meridian Research, Inc. assisted in this effort by providing OSHA with technical support under Task Order No. 10, Base Year, DOL Contract No. J-9-F-3-0043. The body of this report is organized into sections, based on the type of hazard or agent causing the adverse health effects. For example, the literature describing the health effects associated with exposure to **herbicides** is discussed separately from the literature on nematocide-related health effects. Similarly, infections are described under various headings, depending on whether they are caused by a **bacterium**, a **virus**, a **protozoan**, etc. In addition, for each topic, the adverse health effects in humans are described, followed by a discussion of the most relevant literature.

CHAPTER II. THE WORKPLACE AND WORKFORCE

Farming in the United States is performed on 2.1 million farms, defined by the United States Department of Agriculture (USDA) as independent areas of land that could (but do not necessarily) sell $1,000 or more worth of agricultural products per year (USDA 1990). The average farm size is 455 acres; more than 1.7 million U.S. farms have fewer than 500 acres. In 1987, nearly 1.5 million farms produced poultry and livestock, and 1.6 million farms produced crops (USDA 1990). Hay, corn, wheat, soybean, sorghum, and cotton make up the largest crops, and cultivation and harvesting of these crops is highly mechanized. The cultivation and harvesting of fruit and vegetable crops is more labor intensive, however.

Farm labor is provided by 2.1 million farm owners, their families, and hired hands. In 1987, an estimated 600,000 unpaid family members worked on farms, in addition to almost 1 million paid employees. The Occupational Safety and Health Administration (OSHA) oversees job-related safety and health practices on farms with 11 or more employees; however, fewer than 5 percent of farms are large enough to employ 11 or more employees (Purschwitz and Field 1990). Cordes and Rea (1988) report that 25 percent of all farm workers are younger than 16 years, and that most of these workers are the children of farmers. According to Engberg (1993), women comprise 20 percent of the farming workforce.

A significant proportion of the hired farm labor workforce migrates from one farming region to another during the course of the growing season. Estimates of the number of migrant farm workers in the U.S. are imprecise because of the nomadic nature of the group, the variation in crop harvesting patterns, and the reluctance of some migrant farm workers to be counted, and estimates of the size of this workforce therefore vary greatly. The Office of Migrant Health estimates that migrant and seasonal farmers (and their families) number approximately 3 million, while others estimate the number of migrant farmers to be 5 million or more (Mobed, Gold, and Schenker 1992). Women and children comprise a significant proportion of the migrant farmer workforce (Mobed, Gold and Schenker 1992).

The agricultural workplace has many potential health and safety hazards, including those posed by machinery, animals, agricultural chemicals, infectious agents, ergonomic

factors, and the elements. The sheer number of farm animals and farm machines on U.S. farms casts light on the potential magnitude of the hazards posed by animals and machines. In 1987, approximately 4.6 million tractors were in operation on U.S. farms, as were 823,000 hay balers, 667,000 combines, and 43,000 corn pickers. According to U.S. Department of Commerce data for 1991, approximately 360 million chickens, 100 million cattle, and 57 million hogs reside on U.S. farms (Statistical Abstract 1993).

CHAPTER III. STUDIES OF ACCIDENT-RELATED MORTALITY AND MORBIDITY AMONG FARMERS

Farmers are at increased risk of death or disability due to work-related illness and injury. Risk estimates provided by the National Safety Council (NSC), Bureau of Labor Statistics[1] (BLS), National Institute for Occupational Safety and Health (NIOSH), and other sources vary somewhat, but all place farming among the most hazardous of the occupations. A number of epidemiological studies of the mortality experience of farmers in several states have been published (Une, Schuman, Caldwell, and Whitlock 1987; Delzell and Grufferman 1985; Stark, Chang, Fitzgerald et al. 1987; Stubbs, Harris, and Spear 1984; Burmeister and Morgan 1982). These studies have consistently shown increased risk of death due to external (accidental) causes among farmers.

Fatal Accidents

Merchant (1991) analyzed estimates of traumatic death rates among farmers and found them to be 2.5 to 5 times greater than the corresponding death rates for workers in all other industries. In 1992, the NSC reported 47.2 accidental deaths per 100,000 farm residents (including non-work-related deaths) (NSC 1993). For the same year, estimates show the rate of fatal injuries (37 deaths per 100,000 workers) to be higher among agricultural workers than the rate in the mining or construction industry (29/100,000 and 22/100,000, respectively) (NSC 1993). According to NIOSH estimates, the average annual injury-related death rate among farmers between 1980 and 1985 was 21.7 per 100,000, higher than for all other sectors (Baker, O'Neill, Ginsburg, and Li 1992). Between 14 percent and 24 percent of fatal farm accidents involve children, depending on the state of residence (Purschwitz and Field 1990).

According to data from the National Traumatic Occupational Fatality surveillance project, death rates attributable to farm machinery dropped by 20 percent between 1980 and 1986, although agricultural machinery continues to account for 51 percent of all deaths nationally due to workplace machinery (based on 1988 data) (Baker, O'Neil, Ginsburg, and

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[1] Based upon those working on farms with 11 or more workers.

Li 1992). According to BLS data, 28 percent of deaths occurring in 1980 among agricultural workers at work were caused by over-the-road motor vehicle accidents, and 20 percent were caused by accidents involving farm equipment or vehicles. Heart attacks, being struck by objects, fires and explosions, and airplane crashes accounted for 15 percent, 8 percent, 7 percent, and 6 percent of these deaths, respectively. Electrocutions, falls, machinery operations, and gunshots each accounted for less than 5 percent of agricultural worker deaths (Kraus 1985).

Farmers 60 years old or older are at greatest risk of death due to injury on the farm. The average annual injury-related death rate among older farmers (65 years or more) is very high, averaging 55.7 per 100,000 between 1980 and 1985 (Myers 1990). The annual death rate from accidents involving farm machinery roughly quadruples between 40 and 70 years of age (Baker, O'Neill, Ginsburg, and Li 1992). Estimates of the proportion of farm-related fatalities occurring to older workers range from 20 percent to 40 percent (Baker, O'Neill, Ginsburg, and Li 1992).

A study of farm fatalities in Indiana between 1968 and 1985 reported 250 fatal injuries to older farm workers. Fifty-three percent of the fatalities were due to farm tractors or to equipment associated directly with tractors, such as power take-off devices. Of the tractor-related fatalities, 39 percent were due to roll-over accidents, 18 percent to run-over accidents, and 6 percent each to falls from tractors and power take-off entanglements. Other farm machinery (augers, combines, corn-pickers, etc.), falls, tree cutting, and livestock-related accidents accounted for most of the remaining accidental deaths (Purschwitz and Field 1991).

Stallones (1989) reported an annual injury-related mortality rate of 52.2 per 100,000 male farm workers in Kentucky between 1979 and 1985. This rate increased from 29.1/100,000 among those younger than 25 years to 110.7/100,000 among those 65 years and older. Consistent with other studies, this study reports that nearly 50 percent of injury-related farm deaths are due to agricultural equipment, including tractors. Smoke inhalation, drowning, firearms, falls, and animal-related injuries accounted for most of the remainder of these deaths (Stallones 1989).

Non-Fatal Accidents

In 1992, an estimated 140,000 disabling injuries occurred among U.S. farm workers (4.3 disabling injuries per 1,000 farm workers), and the occupational injury/illness rate among farm workers involved in crop production was 13.2 injury/illnesses per 100 workers (NSC 1993). More than half of these injuries and illnesses resulted in lost work days (average 39 days away from work) (NSC 1993). In comparison, the manufacturing sector had an injury/illness incidence rate of 10.8 per 100 workers and the construction sector had a rater of 5.4 per 100 workers in 1992 (NSC 1993).

Purschwitz and Field (1990) reviewed farm injury data from 35 states and determined that 65 percent of all farm injuries were severe enough to require medical attention. Nearly half of the accidents resulting in permanent disability were caused by agricultural machinery (other than tractors). In a large case series of farm trauma patients, farm animals, farm machinery (including augers and hay balers), tractors, falls, power take-offs, and corn pickers accounted for 30 percent, 23 percent, 16 percent, 10 percent, 7 percent, and 6 percent of all non-fatal injuries, respectively (Cogbill, Steenlage, Landercasper, and Strutt 1991).

McDermott and Lee (1990) found lower accident rates among the migrant farm population: 1.7 per 100 persons per year due to moving vehicles and 2.7 per 100 persons per year for other work accidents. One survey of migrant farmers randomly chosen in work camps showed that 8.4 percent had had an occupational injury during the previous 3 years; of these injuries, 20 percent were vehicle-related, while 80 percent involved fractures, lacerations, and sprains. Selection effects (i.e., selecting out of the workforce those most susceptible to injury) may cause an underestimation of injury risk among migrant farm workers (Ciesielski, Hall, and Sweeney 1991).

CHAPTER IV. STUDIES OF ADVERSE HEALTH EFFECTS AMONG FARMERS

MORTALITY STUDIES OF NEOPLASTIC DISEASE AND GENERAL STUDIES OF REPRODUCTIVE EFFECTS

Neoplastic Disease

Farmers are generally less at risk of dying of a chronic illness than other groups in the population. However, their risk of dying from specific neoplastic diseases may be increased compared with that in other groups. The association between the occupation of farming and certain kinds of cancer is strengthened by the fact that plausible etiologic candidates have been suggested for those cancers that are most consistently associated with farming. The following paragraphs briefly describe these analyses.

Davis, Blair, and Hoel (1992) reviewed 20 mortality studies of farmers in the United States and found the risk of several neoplasms to be increased in this group. Cancer of the lip, stomach, prostate, and brain, and Hodgkin's disease, non-Hodgkin's lymphoma, leukemia, multiple myeloma, and melanoma were significantly more common among the farmers studied.

A meta-analysis of several mortality studies of farmers found that the risk of death from leukemia, prostatic cancer, stomach cancer, multiple myeloma, Hodgkin's disease, melanoma, and cancer of the lip were all increased (weighted relative risks from 1.07 to 2.08). Although most of the studies analyzed reported an increased risk of mortality due to non-Hodgkin's lymphoma, the weighted relative risk (across all studies reviewed) was not significantly different from 1.0 for this cancer (Blair, Zahm, Pearce et al. 1992).

After a review of studies of the neoplastic risk associated with farming, Moses (1989b) noted that the risk of malignant lymphoma was elevated among farmers in California, Iowa, Minnesota, Utah, Wisconsin, and Kansas, and leukemia risk was increased among farmers in Iowa, Minnesota, Nebraska, North Carolina, Oregon, Washington, and Wisconsin. In many of these states, multiple myeloma risk among farmers was also elevated.

To further define the association between non-Hodgkin's lymphoma and farming, Zahm and Blair (1992) reviewed 21 cohort studies or surveys addressing this topic. Among

these studies, 11 demonstrated positive associations between farming and the development of non-Hodgkin's lymphoma (but only three of these studies had statistically significant results). One study even reported a statistically significant protective effect associated with farming. The relative risk estimates shown by these studies ranged from 0.6 to 2.6. In a group of 19 case-control and cross-sectional studies reviewed by the same authors, the estimated relative risks for non-Hodgkin's lymphoma among farmers ranged from 0.6 to 1.6. Eight of these studies demonstrated statistically significant positive associations for this disease.

Alavanja, Blair, and Masters (1990) described an apparent dose-response relationship that may further implicate agricultural exposures in the development of non-Hodgkin's lymphoma. These authors reported a pronounced increase in the estimated relative risk for non-Hodgkin's lymphoma among maintenance and elevator workers in flour mills (odds ratios 8.1 and 2.8, respectively). Among workers in the flour mills, the relative risk of developing non-Hodgkin's lymphoma increased as duration of employment increased (odds ratio 9.1 for workers employed for 25 years or more). These risks appeared to be related to exposures within the flour-processing plants. An increased relative risk for pancreatic cancer was also observed in this cohort.

The risk of leukemia may also be increased among farmers. Brown and colleagues (1990) observed a small (odds ratio 1.2) but statistically significant increased risk for some forms of leukemia among those who lived or worked on a farm as adults. The odds ratio for developing chronic lymphocytic leukemia among farmers was 1.4. In a European epidemiological investigation of occupational risk factors for acute leukemia, farmers were shown to be 4.6 to 5.6 times more likely than their age, gender, and residence-matched controls to develop acute leukemia (Richardson, Zittoun, Bastuji-Gorin et al. 1992).

The risk of multiple myeloma may also be increased among farm workers. Eriksson and Karlsson (1992) performed a matched-pair, case-control study and found farmers to be the only occupational group with significantly increased risk (odds ratio 1.7) for developing multiple myeloma. Brown, Burmeister, Everett, and Blair (1993) also identified a non-significant increase in the risk of multiple myeloma (odds ratio 1.2) among farmers in a different case-control study of multiple myeloma.

Pearce and Reif (1990) review the epidemiologic evidence for the associations between human malignancy and chemical exposures in the agricultural workplace. Although the available evidence is sparse, these authors suggest that infection with farm animal viruses (bovine leukemia virus, the herpes virus causing Marek's disease in poultry, bovine papilloma virus, and avian leukosis virus) may predispose farmers to the development of malignancies. They further argue that chronic antigenic stimulation in the farm environment may be related to the apparent increased risk of multiple myeloma (Pearce and Reif 1990).

In addition to these specific types of cancer, other studies have shown that farmers are at increased risk of death due to other cancers, respiratory disease, tuberculosis, chronic nephritis, central nervous system disease, rheumatic fever, parasitic disease, and infections (Saftlas, Blair, Cantor et al. 1987; Stubbs, Harris, and Spear 1984; Delzell and Grufferman 1985; Une, Schuman, Caldwell, and Whitlock 1987).

General Studies of Reproductive Effects in the Farming Population

Although specific agricultural chemicals known to have adverse reproductive effects in humans (such as 1,2-dibromo-3-chloropropane (DBCP)) have been removed from use, recent studies suggest that reproductive toxins may remain in the agricultural workplace. Strohmer, Boldizsar, Plockinger, and colleagues (1993) compared differences in the distributions of male-partner occupation between 103 couples seeking fertility assistance due to male sterility and 103 couples seeking fertility assistance due to female sterility. The estimated relative risk of male infertility given male-partner agricultural work was 12.2 (highly significant). Farming women also have been found to be at increased risk for stillbirth and spontaneous abortion, and an increase in limb-reduction birth defects has been found among the offspring of farmers in California (Moses 1989b).

STUDIES OF PESTICIDE-RELATED EFFECTS AMONG FARMERS

General

Pesticides include herbicides, insecticides, fungicides, rodenticides, slimicides, bactericides, acaricides, molluscicides, and nematicides (Moses 1989b). Two-thirds of all pesti-

cides used (by weight) are herbicides. Alachlor, atrazine, 2,4-D, paraquat, simazine, and trifluralin were among the most commonly used herbicides (Moses 1989b).

Organophosphate and carbamate insecticides are most commonly used now that the use of chlorinated hydrocarbon insecticides (DDT, aldrin, dieldrin, chlordane, heptachlor, lindane, and toxaphene) has been restricted. Commonly used organophosphate insecticides include: chlorpyrifos, diazinon, malathion, parathion, phosdrin, methamidophos, and DDVP. The most often used N-methyl carbamate insecticides include: aldicarb, carbaryl, carbofuran, and methomyl. Since DBCP and ethylene dibromide have been banned, other fumigants, including 1,2-dichloropropane/1,3-dichloropropene (DD) and methyl bromide, are being used more frequently. Commonly used fungicides include benomyl, captan, chlorothalonil, maneb, and mancozeb (Moses 1989b).

Occupational exposures to pesticides occur most commonly in the form of dermal contact. The persistence of pesticides on the skin for months following exposure has been described (Moses 1989b). However, there are many opportunities for agricultural workers to be exposed to pesticides. For example, most pesticides require mixing and loading into equipment before they can be applied to seed, soil, crops, or harvest. Workers involved in pesticide application are at risk for dermal and respiratory exposure, whether applying the substance with ground rig spray equipment, air blast sprayers, crop dusters, or as animal dips. Once applied, pesticide exposure can occur from off-gassing, drift (dispersal from the point of application by wind), or contact with residues (on plants, animals, skin, clothing, and farm equipment) (Moses 1989b).

Personal control measures commonly used to minimize worker exposure to pesticides include the use of protective clothing and gloves, respirators, eye protection, and other hygiene measures. Environmental controls are more difficult to institute, but establishment of reentry intervals following pesticide application, i.e., establishing periods of time during which workers are not allowed into the fields after spraying, is the most frequently used control.

STUDIES OF ACUTE PESTICIDE-RELATED HEALTH EFFECTS

The inhalation or ingestion of pesticides is associated with serious acute systemic toxicity. These acute health effects can be classified according to their systemic or topical effects. Topical effects include injuries to the skin and eyes, such as contact dermatitis, chemical burns, conjunctivitis, iritis, and corneal ulceration. Systemic effects include intoxication and respiratory distress syndromes (Davies and Enos 1980).

The incidence of toxic exposure among those working in the agricultural sector is difficult to assess. Many farm chemicals are used sporadically or seasonally, and minor exposure to toxins may produce vague or non-specific symptoms. For these reasons, many researchers studying the epidemiology of toxic exposures among farmers have generated conservative estimates of the incidence of toxic exposures by including as cases only those coming to medical attention for signs and symptoms related to the exposure (for example, by visiting the emergency room) (Rettig, Klein, and Sniezek 1987).

The Nebraska Department of Health studied emergency room and hospital admission and discharge diagnoses to assess the incidence of toxic exposures to agricultural chemicals in a 21-county section of Nebraska. Over a 2-year period, an annual incidence of 13.5 acute exposures per 100,000 population was observed. The proportion of the population in these counties working in the agricultural industry was not reported; however, one-third of these exposures were to anhydrous ammonia (a fertilizer); 2,4-D and parathion were the next most common exposures. The remaining exposures were to a variety of pesticides and herbicides. The route of exposure was evenly divided between inhalation and dermal exposure. Spray drift, equipment failure, and spills resulted in 47 percent, 23 percent, and 21 percent of exposures, respectively (Rettig, Klein, and Sniezek 1987).

STUDIES OF CHRONIC PESTICIDE-RELATED HEALTH EFFECTS

The long-term health effects associated with exposure to pesticides include chronic or recurrent injuries to the skin and eyes, pulmonary fibrosis, neoplastic transformation in various tissues, neurological dysfunction and behavioral disturbance, infertility, liver dysfunction, and kidney dysfunction (Davies and Enos 1980; Lings 1982). A collection of abstracts on the human carcinogenicity of pesticides has been published (Moses 1989a).

The estimated annual incidence of pesticide-related skin disease among all California agriculture workers between 1978 and 1983 was 2.4/10,000. The rate was considerably greater among horticulture, crop, and poultry farmers. Skin diseases represent approximately 70 percent of occupational diseases in the agricultural industry in California. Among these, irritant contact dermatitis is most common (usually resulting from contact with inorganic compounds such as copper sulfate), and allergic contact dermatitis is also observed (usually resulting from contact with carbamate insecticides) (Abrams, Hogan, and Maibach 1991).

STUDIES OF HERBICIDE/DEFOLIANT-RELATED HEALTH EFFECTS AMONG FARMERS

General

The phenoxy herbicides currently in use include 2,4-D and 2-methyl-4-chlorophenoxyacetic acid. 2,4,5-T is a phenoxy herbicide that has been banned in the United States since 1978 (Zahm and Blair 1992). Several case-control studies have implicated 2,4,5-T as a carcinogen; exposure has been linked both to soft-tissue sarcomas and lymphomas (Bond and Rossbacher 1993). Atrazine is a triazine herbicide and is the most commonly used herbicide in the United States (Zahm and Blair 1992). In experimental models, atrazine has been shown to have carcinogenic potential. Atrazine alters steroid hormone metabolism by inhibiting several enzymes that are involved in steroid production and degradation (Zahm, Weisenburger, Cantor et al. 1993).

Phenoxyacetic acid herbicides and chlorophenols may be contaminated with known carcinogens, such as dioxins and nitrosodimethylamine, although significant contamination is less likely now than in the past. However, the presence of contaminants complicates the interpretation of the epidemiologic evidence on the carcinogenicity of phenoxyacetic acid herbicides and chlorophenols (Kelly and Guidotti 1989/1990).

Studies of Acute Effects Related to Herbicides

The acute toxicity of most herbicides is low. The signs of symptoms of acute herbicide-related toxicity in humans include headache, dizziness, nausea, vomiting, hyperthermia, sweating, diffuse erythema, hypotension, and injury to the liver, kidneys, and heart.

Paraquat has been described as the most toxic of the herbicides, causing oxidant injury to epithelial membranes and other tissues with which it comes into contact. Initially, non-specific oxidant damage occurs to the mucous membranes, liver, kidneys, and heart. Initially, paraquat causes a severe acute alveolitis, which leads to pulmonary fibrosis and hyaline membrane formation. Death from asphyxia may result (Moses 1989b). The absolute lethal dose of paraquat is 6 grams; no survivors have been reported at greater doses. Many, but not all, persons exposed to less than 1 gram have survived. Delay in treatment worsens the prognosis at a given exposure dose (Bainova 1983). Many deaths have been attributed to acute paraquat toxicity, not uncommonly from intentional ingestion.

In one case series of 15 agricultural workers with dermal exposure to paraquat involving less than 10 percent of the body surface, 13 suffered burns (first to third degree), vesicles, contact dermatitis, and conjunctivitis. Onset of skin manifestations was delayed by 1 to 3 days in all workers. None suffered systemic toxicity, although paraquat was measured in the blood or urine of five of these workers (Hoffer and Taitelman 1989).

Studies of Chronic Effects Related to Herbicides

The signs and symptoms of chronic herbicide-related toxicity in humans include depression, ataxia, hypotonia, weight loss, abdominal pain, vomiting, diarrhea, thyroid and pituitary dysfunction, kidney and liver damage, and dermatitis (Bainova 1983). The carcinogenic and teratogenic potential of several herbicides has also been studied.

Blair and Zahm (1990) reviewed many of the epidemiological studies linking herbicides and cancer and focused their discussion on soft-tissue sarcoma and lymphoma. Based on the reviewed studies, relative risk estimates for the development of soft-tissue sarcomas following exposure to phenoxyacetic acid herbicides ranged from 0.7 to 6.8. Eight of 11 soft-tissue sarcoma studies reviewed had relative risk estimates that exceeded one. Relative risk estimates for non-Hodgkin's lymphoma ranged from 0.4 to 4.8. Again, eight of the 11 studies reviewed for this endpoint showed relative risk estimates greater than one (Blair and Zahm 1990). All five of the studies reviewed for multiple myeloma reported an excess risk associated with phenoxyacetic acid exposure (relative risk (RR) range 1.3 to 5.0). Testicular and ovarian cancer risk estimates were also elevated in the three reviewed studies evaluating

these outcomes (RR 2.2 to 4.6, and 4.4, respectively). Leukemia also was more common among the exposed groups in all studies reviewed (RR 1.1 to 2.2) (Blair and Zahm 1990). Other reviewers of the published literature have come to similar conclusions, i.e., that phenoxy herbicide exposure is fairly consistently associated with non-Hodgkin's lymphoma and may also be associated with soft-tissue sarcoma, leukemia, and cancer of the colon, lung, nose, prostate, and ovary (Morrison, Wilkins, Semenciw et al. 1992).

Zahm and Blair (1992) later reported on the results of several case-control studies in farmers that evaluated the association between non-Hodgkin's lymphoma and previous exposure to phenoxy herbicides, predominantly 2,4-D. The relative risk estimates were 1.5 to 6.0. Two of the studies demonstrated statistically significant trends of increasing risk with increasing exposure (either number of days exposed per year, amount of time between exposure and skin decontamination, or use of protective clothing) (Zahm, Weisenburger, Cantor et al. 1990; Hoar, Blair, Holmes et al. 1986). Farm workers who mixed or applied herbicides frequently had an 8-fold increase in risk for non-Hodgkin's lymphoma (Hoar, Blair, Holmes et al. 1986). These results are supported by the consistency of the findings among studies of workers from other industrial groups who were exposed to these chemicals. In addition, a few studies demonstrate an association between exposure to triazine herbicides and non-Hodgkin's lymphoma. Atrazine use was significantly associated with non-Hodgkin's lymphoma (odds ratio 1.4), but this association was minimized after controlling for 2,4-D and organophosphate use (Zahm, Weisenburger, Cantor et al. 1993).

Richardson and colleagues (1992) conducted a matched-pair case-control study of acute leukemia and found a 6-fold increased risk associated with medium to high weed killer (not further specified) exposures for more than 10 years. The relative risk was much greater for acute lymphoblastic leukemia than for acute myelogenous leukemia.

Kelly and Guidotti (1989/1990) reviewed the available experimental and epidemiological evidence for the potential carcinogenicity of the phenoxyacetic acid herbicides and chlorophenols, and concluded that 2,4-D exposure may be weakly related to the subsequent development of non-Hodgkin's lymphoma. Considerably less evidence was found to support an association between exposure to these chemicals and the development of soft-tissue sarcomas, however (Kelly and Guidotti 1989/1990).

However, all of the evidence does not support an association between herbicide exposure and cancer. Bond and Rossbacher (1993) reviewed a subset of animal, ecological, case-control, and historical cohort studies of the associations between cancer and three chlorophenoxy herbicides: 4-chloro-2-methyl phenoxyacetic acid (MCPA); 2-(4-chloro-2 methyl-phenoxy) propionic acid (MCPP); and 2-(2,4-dichlorophenoxy) propionic acid (2,4-DP). Animal studies have shown liver and kidney effects after exposure to these herbicides but no increase in the risk of neoplasms. These authors further reported that, based on the studies they reviewed, MCPA, MCPP, and 2,4-DP do not appear to cause cancer in humans (Bond and Rossbacher 1993). Brown, Burmeister, Everett, and Blair (1993) conducted a case-control study of multiple myeloma, and found no increased risk among those exposed to phenoxy herbicides (individually or as a chemical class).

In a review of occupational cohort studies evaluating the association between phenoxy herbicide and chlorophenol use and neoplastic disease, Johnson (1990) reported insufficient follow-up periods in all studies for soft-tissue sarcoma risk to be adequately evaluated. Only four of twelve studies had sufficient follow-up to evaluate malignant lymphoma risk, and none of these four studies showed a statistically significantly elevated risk (although relative risk estimates were 2.0 or greater for two of the studies) (Johnson et al. 1990).

Skin manifestations following dermal exposure to herbicides are relatively common. Several herbicides have been associated with contact dermatitis: phenylmercury nitrate, dazomet, glyphosphate, atrazine, dichlobenil, propazine, simazine, trichlorobenzyl, 2,4-D, and choline chloride. Allergic contact dermatitis following exposure to metam-sodium, amitrole, chloridazon, phenmedipham, alachlor, barban, and dazomet have been reported. Inorganic arsenical insecticides and herbicides, and the herbicide paraquat, have been implicated as skin carcinogens (Abrams, Hogan, and Maibach 1991).

Dermal paraquat exposure has been associated with contact dermatitis, keratoses, Bowen's disease, and squamous cell carcinoma of the skin. Fingernail abnormalities including discoloration, onycholysis, deformity, and loss of nails also have been described following nail exposure to paraquat (Abrams, Hogan, and Maibach 1991).

Moses (1989b) reports that data available to EPA as of 1988 supported the teratogenicity of the following herbicides: acrolein; amiben; benazolyn-ethyl; bentazon; bladex;

bromoxynil; cacodylic acid; chlorpropham; 2,4-D acid; dichlobenyl; 2,4-DP acid; dinoseb; diquat; endothall; fenoxaprop-ethyl; fluazifop-butyl; nitrofen; picloram; 2,4,5-T; trifluralin; and triphenyltin acetate.

STUDIES OF INSECTICIDE/FUMIGANT-RELATED HEALTH EFFECTS AMONG FARMERS

General

Insecticides include organophosphates, carbamates, organochlorines, and other compounds. Organophosphate insecticides may be aliphatic or aromatic, and within each group there are halogenated and non-halogenated members. Dichlorvos and trichlorfon are halogenated aliphatic organophosphates; chlorophyrifor, coumaphos, crufomate, ronnel, and tetrachlorvinphos are halogenated aromatic organophosphates; and azinphos-methyl, crotoxyphos, dioxathion, famphur, fensulfothion, methyl parathion, parathion, and phosmet are non-halogenated aromatic organophosphates. Organophosphates are degraded in the environment in a relatively short period of time following application (Brown, Blair, Gibson et al. 1990).

Carbamates include aldicarb, carbaryl, propoxur, carbofuran, methomyl, and bendiocarb. The organochlorine insecticides include DDT, aldrin, endrin, dieldrin, chlordane, heptachlor, and toxaphene. As a group, organochlorine insecticides have an extremely long half-life in the environment, and tend to accumulate in the fat tissues of exposed animals (and humans) (Shaver and Tong 1991).

Fumigants are pesticides applied as a gas, vapor, or smoke to prevent spoilage of agricultural products contained in an enclosure. Fumigants are also used to decontaminate the soil. As a group, the fumigants tend to be very toxic and to have little specificity for particular organisms (that is, they may be used as insecticides, fungicides, ascaricides, and rodenticides). Carbon tetrachloride/carbon disulfide mixtures (80/20), ethylene dibromide, phosphine, malathion/methoxychlor mixtures, chloroform, methyl bromide, chloropicrin, ethylene dichloride, dichloropropene/dichloropropane mixtures, dibromochloropropane, ethylene oxide, propylene oxide, propanyl, formaldehyde, hydrogen cyanide, and acrylonitrile are among the compounds classified as fumigants. Many of these fumigants are restricted or banned due to their acute or chronic toxicities (Shaver and Tong 1991).

Organophosphate and carbamate insecticides are neurotoxins, potent inhibitors of carboxylic esterases (a class of enzymes that includes acetylcholinesterase and pseudocholinesterase). Organophosphates are irreversible inhibitors, and carbamates inhibit acetylcholinesterase competitively. Acetylcholinesterase is the enzyme responsible for the degradation and subsequent deactivation of acetylcholine at the neural synapse. In the presence of organophosphates, the active site of acetylcholinesterase is bound and unavailable for acetylcholine degradation. The concentration of acetylcholine in the synapse rises and the efferent nerve fiber is initially stimulated and subsequently inactivated. Cholinergic nerves are found in the central nervous system, somatic nerves, ganglia of the sympathetic nervous system, parasympathetic nervous system, and some sympathetic nerve endings (including those to the sweat glands) (Namba, Nolte, Jackrel, and Grob 1971). Organochlorine insecticides cause central nervous system excitation that is most pronounced in the sensory and motor neurons (Klaassen et al. 1986).

Toxic exposures to organophosphates, carbamates, organochlorine compounds, and the fumigants most frequently occur by absorption through the skin and respiratory tract, although absorption from the gastrointestinal tract and conjunctiva may occur. Many organophosphates are converted in the liver to even more potent cholinesterase inhibitors (i.e., parathion to paraoxon and malathion to malaoxon) (Namba, Nolte, Jackrel, and Grob 1971).

Studies of Acute Effects Related to Insecticides/Fumigants

The signs and symptoms of acute organophosphate or carbamate insecticide toxicity in humans involve multiple organ systems. The airways and breathing may be adversely affected: central respiratory depression, bronchoconstriction, increased airway secretions, pulmonary edema, dyspnea, cough, wheezing, and cyanosis may all be observed within the first 12 hours following exposure. Adverse effects on the central and peripheral nervous systems may be manifested as: personality or mood changes, auditory or visual hallucinations, headache, tremor, slurred speech, decreased level of consciousness, seizures, generalized weakness or flaccid paralysis, fasciculations, decreased or absent deep tendon or cranial nerve reflexes, miosis, blurred vision, increased salivation, increased lacrimation, increased sweating, and urinary incontinence. The cardiovascular system is also adversely affected, as

manifested by bradycardia or tachycardia, first-degree heart block, widened pulse pressure, hypertension or hypotension, and decreased cardiac output. Nausea, vomiting, diarrhea, fecal incontinence, tenesmus, and abdominal pain are commonly observed in even mild organophosphate or carbamate poisoning. In the blood, decreased cholinesterase activity in serum and erythrocytes is found, and leukocytosis is common (Namba, Nolte, Jackrel, and Brob 1971; Medved and Kagen 1983; Shaver and Tong 1991; López-Carillo and López-Cervantes 1993). The acute effects of moderate organophosphate toxicity, especially visual disturbance, headache, and weakness, have been reported to persist up to several months after exposure to mevinphos and phosphamidon (Whorton and Obrinsky 1983). Atropine and pralidoxime (PAM) may be used to treat the adverse effects of organophosphate and carbamate toxicity.

Acute exposure to organochlorine insecticides is associated with a delay in the onset of symptoms of from 30 minutes to 12 hours. Symptoms include nausea, vomiting, diarrhea, abdominal pain, headache, dizziness, ataxia, paresthesia, bulbar tremors followed by tremors of the extremities, hyperthermia, convulsions, apnea, bleeding diathesis, and death. Laboratory evidence of liver, kidney, and bone marrow damage evolves after significant organochlorine exposure (Kaloyanova-Simeonova 1983; Klaassen et al. 1986).

Fumigants most commonly cause injury to the respiratory tract. Some are severe respiratory irritants (including sulfur dioxide, chloropicrin, acrolein, and formaldehyde) and cause cough, laryngospasm, and bronchospasm. Others are not very irritating and may cause serious airway injury without irritant symptoms (these include methyl bromide, phosphine, and ethylene oxide). Most fumigants cause respiratory injury with subsequent pulmonary edema, central nervous system depression, liver damage, and kidney damage following significant acute exposure. Skin exposure may result in irritation or, in some cases, desquamation. Carcinogenic and teratogenic potential has been demonstrated for dibromochloropropane, ethylene dibromide, 1,2-dichloropropane, and some other fumigants (Shaver and Tong 1991). Methyl bromide is a fumigant used to sterilize soil. More acute occupational deaths have been reported for methyl bromide than for any other pesticide (Moses 1989b).

Studies of Chronic Effects Related to Insecticides/Fumigants

Neurological symptoms and skin manifestations have been observed among farm workers exposed to insecticides and fumigants. As with herbicides, several epidemiological studies suggest that insecticides and fumigants are associated with neoplastic disease among these workers. Leukemia and multiple myeloma have shown the most consistent associations. Teratogenicity and other adverse reproductive effects have also been associated with exposure to some insecticides and fumigants.

Neoplastic Effects. Richardson, Zittoun, Bastuji-Garin, and colleagues (1992) conducted a matched-pair case-control study of acute leukemia and found a 4-fold, statistically significant risk elevation associated with medium to high (not further specified) insecticide exposure for more than 10 years. The relative risk was much greater for acute lymphoblastic leukemia than for acute myelogenous leukemia. Brown, Blair, Gibson and colleagues (1990) found in a case-control study that exposure to halogenated aliphatic, halogenated aromatic, and non-halogenated aromatic organophosphate insecticides significantly increased the risk of developing chronic lymphocytic leukemia (the odds ratios were 2.9, 2.7, and 2.5, respectively). The risk of leukemia (all subtypes) was extremely elevated among those who had handled, mixed, or applied crotoxyphos (odds ratio 11.1). Statistically significant excesses in risk were also observed for dichlorodiphenyltrichloroethane (DDT) (OR 1.3), dichlorvos (OR 2.0), famphur (OR 2.2), methoxychlor (OR 2.2), nicotine (OR 1.6), and pyrethrins (OR 3.7) (Brown, Blair, Gibson et al. 1990).

Other hematologic and soft tissue sarcomas may occur at increased rates among those exposed to insecticides. Eriksson and Karlsson (1992) conducted a matched-pair, population-based, case-control study of multiple myeloma in Sweden and found that exposure to DDT was associated with a 1.8-fold increase in risk. In a case-control study of non-Hodgkin's lymphoma among women, mixing or handling organophosphate insecticides was associated with an increased risk (OR 4.5, statistically significant). An increased risk was also suggested by results seen in women who applied insecticides to farm animals (Zahm, Weisenburger, Saal et al. 1993). A case-control study of soft-tissue sarcoma showed increased risk of myomatous and fibrous sarcomas among those who mixed and applied insecticides to ani-

mals, and failure to use personal protective measures was associated with even greater risk (Zahm, Blair, Holmes et al. 1988).

Reproductive Effects. Moses (1989b) reports that data available to EPA as of 1988 supported the teratogenicity of several insecticides, including avermectin, carbaryl, chlordimeform, DBCP, dinocap, endosulfan, ethion, ethylene dibromide, imidan, larvadex, methyl parathion, mirex, omite, sodium arsenate, sodium arsenite, and trichlorfon. Ethylene dibromide and DBCP may cause sterility in exposed workers. Both are animal carcinogens, as is arsenic (Moses 1989b; Pearce and Reif 1990). A recent cohort study of pregnancy outcome showed no increased risk of spontaneous abortion, preterm delivery, low birth weight, or toxemia among Hispanic women exposed to organophosphate insecticides (Willis, de Peyster, Molgaard et al. 1993).

Chronic Toxicity. Signs and symptoms of chronic organophosphate toxicity include headache, weakness, memory impairment, disturbed sleep and orientation, anorexia, hallucinations, anxiety, neuritis, paresis, and paralysis (Medved and Kagen 1983). Some organophosphate insecticides have been shown to cause a neuropathy several weeks following exposure. Pathologically, the larger and longer neurons of both the central and peripheral nervous systems are affected, and demyelination occurs. Weakness progressing to paralysis and death has been described. Organophosphates implicated as likely etiologic agents of this delayed neuropathy include EPN, trichlorfon, DDVP, DEF, isofenphos, and chlorpyrifos (Moses 1989b). Signs and symptoms of chronic toxicity from prolonged exposure to organochlorine insecticides include headache, dizziness, paresthesia, sensory and vestibular disturbance, vascular tone lability, abdominal pain, convulsions, tachycardia, increased cardiac stroke volume, chest pain, liver and kidney function abnormalities, pancytopenia from marrow suppression, and hemolytic anemia (Kaloyanova-Simeonova 1983).

Dermal Effects. Skin manifestations of insecticide exposure are relatively common. Contact dermatitis has been observed following dermal exposure to rotenone, inorganic arsenic compounds, ethylene oxide, DD (a mixture of 1,3-dichloropropene, 1,2-dichloropropane, epichlorohydrin, and related compounds), lindane, malathion, dichlorvos, thiometon, promecarb, carbaryl, and propargite. Allergic contact dermatitis has been reported following contact with pyrethrum (especially pyrethrosin), inorganic arsenic compounds, ethylene

oxide, metam-sodium, parathion, methyl parathion, malathion, and naled. Dermal contact with pyrethroid insecticides has also been associated with paresthesias. Methyl bromide causes chemical burns when it comes in contact with the skin. Acrylonitrile exposure has been associated with the development of toxic epidermal necrolysis (and is reportedly no longer used in industry). Nail dystrophy has been associated with exposure to 4,6-dinitro-*o*-cresol, and severe phototoxic reactions have been reported following exposure to phenothiazine (Abrams, Hogan, and Maibach 1991).

STUDIES OF FUNGICIDE-RELATED EFFECTS AMONG FARMERS

General

Fungicides are pesticides that kill members of the *Phycomycetes*, *Ascomycetes*, *Basidiomycetes*, and *Imperfecti* groups of organisms. Many compounds may be used in agriculture as fungicides, including: sulfur, copper, mercury, tin, and zinc compounds, organophosphorus compounds, dithiocarbamates, carbamates, halogenated hydrocarbons, aromatic nitro compounds, quinones, anilines, phthalimides, pyrimidines, pyridines, thiodaoaxoles, triazines, isoxazolones, imidazoles, oils, and other compounds (Kaloyanova-Simeonova 1983).

Studies of Acute Fungicide-Related Health Effects

The acute toxicity of selected fungicides illustrates the many mechanisms of action by which some compounds with fungicidal properties act. Hexachlorobenzene, a chlorinated hydrocarbon, is stored in fatty tissues and is highly toxic to the liver. It interferes with porphyrin metabolism, and causes a porphyria similar to that seen in porphyria cutanea tarda. Hirsutism and arthritis have been descried among workers exposed to this fungicide. 2-(1-Methyl-2-propyl)-4,6-dinitrophenyl isopropyl-carbonate, an aromatic nitro compound, inhibits cellular respiration and results in depletion of adenosine triphosphate in the cytoplasm. It causes severe liver and convoluted renal tubular injury. Manifestations of toxicity include hyperthermia, methemoglobinemia, hemolysis, and central nervous system disturbance. Acute poisoning with dinitrocapryl crotonate, another aromatic nitro compound, causes disturbed thermal homeostasis, clonic activity, and respiratory difficulties. 2,4-Dinitrophenyl-

thiocyanate is another aromatic nitro compound that causes leukocytosis and anemia. *cis*-N-(trichloromethyl)thio-4-cyclo-hexene-1,2-dicarboximide (captan) is a phthalimide fungicide that interferes with many cellular metabolic processes, including cellular respiration, nucleic acid and protein synthesis, and glycolysis. In addition, it reacts with thiol moieties, thus interfering with the catalytic function of many enzyme systems. Central nervous system symptoms and irritation of the skin, conjunctivitis, and mucous membranes have been described among the acute effects of exposure. Tetrahydro-3,5-dimethyl-2H-thiadiazine-2-thione (dazomet), a thiodiazole fungicide, may cause tachycardia, salivation, anxiety, tachypnea, cramps, and ataxia following acute exposure. The quinones may cause methemoglobinemia and hemolysis following acute exposure. 2,3,5,6-Tetrachloro-1,4-benzoquinone also causes central nervous system depression. 6-Methyl-1,3-dithiolo(4,5b)quinoxalino-2-one (chinomethionate) is a heterocyclic fungicide that has high cumulative toxicity. Its acute toxicity is manifested by central nervous system effects, gastrointestinal disturbances, and decreased phagocytic activity (Kaloyanova-Simeonova 1983; Shaver and Tong 1991).

Studies of Chronic Fungicide-Related Health Effects

Many commonly used fungicides, including baycor, bayleton, benomyl, captafol, captan, copper sulfate, cycloheximide, dichlorophene, fenarimol, folpet, hexachlorobenzene, mancozeb, OPP, PCNB, terrazole, tributyltin oxide, triphenyltin fluoride, and triphenyltin hydroxide, have demonstrated carcinogenic, chromosomal, teratogenic, and other reproductive effects in animals or humans (Moses 1989b; Kaloyanova-Simeonova 1983).

A four-fold excess risk of non-Hodgkin's lymphoma was described in a nested case-control study of grain millers exposed to fungicides and fumigant pesticides. A dose-response relationship was identified, such that workers exposed for 25 years or more were at nine times the risk for developing non-Hodgkin's lymphoma (Zahm and Blair 1992).

The effects of chronic exposure to many fungicides include liver and kidney damage. Hexachlorobenzene, dinobuton, dinocap, nirit, captan, dazomet, chloronil, and chinomethionate exposure may cause hepatic injury and degenerative changes. Kidney damage has also been described following chronic exposure to dinobuton, dinocap, nirit, captan, dazomet, chloronil, and chinomethionate (Kaloyanova-Simeonova 1983).

Abrams, Hogan, and Maibach (1991) noted that sulphur, triphenyltin hydroxide, tributyltin oxide, copper sulfate, hexachlorobenzene, thiophanatemethyl, captafol, folpet, pentachloronitrobenzene, thiram, chlorothalonil, and dithianone have been associated with contact dermatitis. Allergic contact dermatitis has also been seen with exposure to metam-sodium, benomyl, thiophanatemethyl, captafol, captan, ditalmifos (a European fungicide), dinitritochlorobenzene, plondrel, maneb, zineb, mancozeb, chlorothalonil, and dinobuton. Fatalities attributed to toxic epidermal necrolysis have been associated with chlorothalonil exposure (Abrams, Hogan, and Maibach 1991).

STUDIES OF RODENTICIDE-RELATED EFFECTS AMONG FARMERS

Rodenticides are categorized as acute poisons (those requiring only one dose) or chronic poisons (those requiring multiple exposures). The acute poisons are highly toxic (LD_{50}s less than 100 mg/kg), are non-specific for rodents, and generally lack antidotes. Zinc phosphide, norbromide, fluoracetamide, strychnine, sodium fluoroacetate, and alpha-chloralose are acute poisons used in the control of rodents. The exposure mechanism usually involves contact between the poison and the fur, with subsequent ingestion when the animal cleans the fur. These compounds have the disadvantage that signs of toxicity may become manifest before a lethal dose has been delivered, and may interfere with the ingestion of a lethal dose (Shenker 1983). In humans, zinc phosphide exposure may cause severe esophageal burns, nausea, abdominal pain, chest tightness, agitation, chills, fever, and shock. Cardiovascular collapse has been reported in adult humans exposed to as little as 7 drops of zinc phosphide. Norbromide causes hypothermia and hypotension in humans (Sittig 1991). Strychnine causes seizures, tonus, and respiratory embarrassment. Fluoroacetamide and sodium fluoroacetate cause cessation of aerobic cellular respiration by blocking the metabolism of citrate, and exposure results in apprehension, nausea, vomiting, seizures, ventricular fibrillation, and death (Shaver and Tong 1991).

The chronic rodent poisons, including the anticoagulants and calciferol, can be targeted more specifically to rodents but require multiple doses to be lethal. Effective antidotes for the chronic rodent poisons exist. These poisons are usually mixed into a bait, which is ingested. The animals usually remain asymptomatic until a lethal dose has been ingested. In

humans, chronic toxicity of anticoagulants is manifested as bleeding diathesis, and vitamin K is an effective antidote (Shenker 1983). According to Abrams, Hogan, and Maibach (1991), warfarin and naphthylthiourea have been associated with contact dermatitis. Also, warfarin is teratogenic in experimental animals and humans, causing fetal loss and a characteristic embryopathy (Moses 1989b).

STUDIES OF NEMATOCIDE-RELATED EFFECTS AMONG FARMERS

1,2-Dibromo-3-chloropropane (DBCP) was introduced as a nematocide in 1955 and was later found to be carcinogenic in experimental animals. Subsequent studies have shown DBCP to be mutagenic in both procaryotic and eucaryotic strains of organisms. In mammalian systems, DBCP induces heritable mutations as well as autosomal dominant, lethal mutations in the genome. Sperm from men exposed to DBCP have a higher incidence of non-dysjunction of the Y-chromosome (Teramoto and Shirasu 1989).

In exposed male workers, DBCP causes infertility (azospermia and oligospermia). Reduced reproductive function in exposed workers appears to be permanent and is believed to result from irreversible damage to the germinal epithelium. An increased incidence in congenital malformations among the offspring of workers exposed to DBCP has not been described, but a greater proportion of pregnancies conceived by partners of exposed male workers end in spontaneous abortion (Teramoto and Shirasu 1989). The nematicide nemacur has been shown to have teratogenic activity in animals (Moses 1989b).

STUDIES OF FERTILIZER-RELATED EFFECTS AMONG FARMERS

General

Fertilizers may be classified as natural and artificial on the basis of their origin. Natural fertilizers derive from human or animal excrement, either processed or not. The greatest risks to farm workers arise from exposure to off-gases and infectious agents. Each of these risks will be discussed in separate sections (confinements and manure pits, and zoonoses, respectively). Artificial fertilizers include nitrogen, phosphorus, potassium, and trace element compounds. Among these, the nitrogenous fertilizer anhydrous ammonia is most commonly used (Ersov 1983).

Anhydrous ammonia provides a rich source of bioavailable nitrogen. Under normal atmospheric conditions, anhydrous ammonia is a colorless gas with a strong odor of ammonia. For agricultural applications, it is stored and handled in pressurized containers as a liquid. Anhydrous ammonia vaporizes at -33C, is extremely caustic, and is very soluble in water (Dalton and Bricker 1978).

Most exposures to anhydrous ammonia occur during transfer between storage tanks and application equipment (Dalton and Bricker 1978). In one series, broken couplings and hoses accounted for 50 percent of exposures (Millea, Kucan, and Smoot 1989).

Studies of Acute Fertilizer-Related Health Effects

Anhydrous ammonia is a common cause of severe chemical burns among agricultural workers. In one survey of emergency room admissions for agricultural chemical exposure in Nebraska, anhydrous ammonia exposure accounted for one-third of all admissions (Rettig, Klein, and Sniezek 1987). In a series of 12 cases of anhydrous ammonia exposure, eight occurred in farm workers. Surface burns covering 3 to 22 percent of the body were noted. Two workers suffered significant respiratory tract burns (Millea, Kucan, and Smoot 1989). In another series, one of six workers acutely exposed to anhydrous ammonia died of pulmonary complications (Dalton and Bricker 1978).

When anhydrous ammonia comes into contact with the skin or mucous membranes, it rapidly vaporizes and causes severe freezing, extensive caustic burns, and liquefaction necrosis of the tissues involved. Near-instant thrombosis of affected vessels may result in vascular compromise of distal regions (Millea, Kucan, and Smoot 1989). Vapors rapidly dissolve in airway and conjunctival secretions, causing severe caustic burns to the oropharynx, nasopharynx, airways, lung parenchyma, and eyes.

At 53 parts per million, the odor of ammonia can be detected. At concentrations of approximately 400 ppm, irritation of the eyes, nose, and throat are reported. Concentrations of 700 ppm produce immediate eye injury. Laryngospasm and coughing occur immediately at concentrations of 1700 ppm, and glottic edema may ensue within a few hours of exposure to these levels. Exposures to 2500 to 4500 ppm are lethal for durations of 30 minutes or more. Exposure to 5000 ppm is rapidly fatal (Dalton and Bricker 1978; Millea, Kucan, and

Smoot 1989). The most severe pulmonary injuries occur on exposure to concentrations less than 2500 ppm, since more prolonged exposure can be tolerated at this level (Shaver and Tong 1991).

Immediately on exposure to damaging concentrations of anhydrous ammonia, severe pain of the eyes, throat, mouth, and exposed skin has been reported. Airway edema progresses rapidly, and aphonia and stridor are common. Cyanosis and asphyxia from airway obstruction and pulmonary edema have also been recorded. Burns of the skin (usually of the face and exposed skin), eyes, and mucous membranes are commonly observed. Liquefaction necrosis of surface and deeper tissues is also observed (Shaver and Tong 1991).

Resuscitative measures should include thorough washing of the affected areas with copious amounts of water. Exposed individuals usually require airway maintenance by endotracheal intubation or tracheostomy. Pulmonary parenchymal damage frequently necessitates positive pressure ventilation to increase mean airway pressure and the oxygen diffusion gradient (required because pulmonary edema and adult respiratory distress syndrome often follow acute overexposures). Eye and skin burns are treated with local care, according to the extent of the burn (Dalton and Bricker 1978).

Studies of Chronic Fertilizer-Related Health Effects

Bronchiolitis obliterans and chronic cystic bronchiectasis have been described following anhydrous ammonia inhalation injury (Shaver and Tong 1991). Glaucoma, corneal opacity, cataract, irregular astigmatism, iritis, fusion of the cornea, iris, and lens, and atrophy of the globe occur during recovery from eye injury caused by such exposure (Helmers, Top, and Knapp 1971). Chronic exposure to low-level nitrogenous fertilizers has been associated with death due to methemoglobinemia (Shaver and Tong 1991).

STUDIES OF AFLATOXIN-RELATED EFFECTS

General

Aflatoxins are a class of mycotoxins related structurally to coumadin that are produced by *Aspergillus flavus* and *Aspergillus parasiticus*. Aflatoxins may be present in peanuts, corn, cottonseed, wheat, millet, sorghum, barley, peas, sesame, soybeans, cowpeas,

Brazil nuts, pistachio nuts, almonds, beans, cassava, and sweet potatoes. Drought stresses, high temperatures, insect infestation, and high plant density each favor the growth of *Aspergillus* and the production of aflatoxin in the field. In addition, damage to crops, rain during harvesting, and elevated storage temperatures promote aflatoxin production during storage and processing (Coulombe 1991).

Aflatoxins are ubiquitous contaminants in the crops listed above. Two aflatoxins, AFB_1 and AFM_1, have been studied extensively. AFB_1 is the most potent aflatoxin, both in terms of acute effects and carcinogenicity. AFM_1 is commonly found in dairy products and moldy grains, and was the first recognized metabolite of AFB_1. AFM_1 can be found in the urine of animals and humans fed aflatoxin-contaminated substances. The Food and Drug Administration has informally set the action level for total aflatoxin at 20 parts per billion (ppb) for contaminated commodities, and at 0.5 ppb for AFM_1 in dairy products (Wood 1991).

AFB_1 is metabolized in the liver (in humans and many other species) by the cytochrome P450 system, and in the peripheral tissues by prostaglandin H synthetase. One metabolite is the 2,3-epoxide of AFB_1, which is a powerful alkylating agent. This epoxide-AFB_1 molecule results in strong cross-linking of cytoplasmic DNA, RNA, and protein, resulting in DNA-adducts, decreased transcription, and decreased translation. Glutathione conjugation with AFB_1-2,3-epoxide appears to render it inactive as an alkylating agent and protects against its carcinogenic activity. Several aflatoxins result from AFB_1 metabolism, but none has been shown to be as toxic as AFB_1. Aflatoxins are excreted in the stool and urine of animals fed contaminated grains (Coulombe 1991).

In vitro systems have shown both AFB_1 and AFM_1 to be mutagenic, and hepatocarcinogenicity had been demonstrated in trout, chicks, ducks, turkeys, mice, rats, hamsters, and monkeys ($AFB_1 >> AFM_1$ in both cases). Although less common, neoplasms of other organs have been found to result from aflatoxin exposure. Additionally, aflatoxicosis following acute aflatoxin poisoning has been described, initially in turkeys. Aflatoxin was found to be the cause of turkey X-disease, which was manifested by acute, hemorrhagic liver necrosis, fatty infiltration of the liver, hyperplasia of the bile ducts, anorexia, lethargy, and death.

Hemorrhagic necrosis of the kidneys, spleen, pancreas, heart, and gastrointestinal tract have also been observed (Coulombe 1991).

Human exposure to aflatoxins occurs most frequently by ingestion of contaminated foods, such as corn, peanut butter, and dairy products. Farmers are also at risk of inhalation exposure to aflatoxin-contaminated dusts (Coulombe 1991). Respirable dusts from corn contaminated with aflatoxin have much higher aflatoxin concentrations that those found in contaminated consumables, with the highest concentrations found in the smallest particles. Particles with less than 7 μm diameters had aflatoxin concentrations as high as 1.8 parts per million (Coulombe 1991). Experimental studies have demonstrated conversion of AFB_1 to carcinogenic metabolites in cell cultures of non-ciliated mammalian tracheal epithelial cells, and 50 percent of rats exposed to six separate 0.3-mg doses of AFB_1 by instillation into the airway over 3 weeks developed tracheal carcinomas (Coulombe 1991).

Animal waste provides another potential for farmer exposure to aflatoxins, since AFB_1, AFM_1, and other native aflatoxins and their toxic metabolites are excreted in stool and urine. In addition, concentrations of aflatoxin in excess of the FDA action level have been found in the breast milk of mothers exposed to high dietary concentrations of aflatoxin (Wood 1991). In a case-control study of primary hepatic carcinoma, milk-maids were found to have an increased risk of developing this disease (odds ratio 3.5), probably as a result of exposure to aflatoxins (Kauppinen, Riala, Seitsamo, and Hernberg 1992).

Studies of Aflatoxin-Related Acute Effects

Reports of human aflatoxicosis are rare in the United States. Reports from western India, Uganda, Taiwan, Czechoslovakia, and Senegal have described over 100 deaths caused by hepatic failure and massive gastrointestinal hemorrhage among people ingesting aflatoxin-contaminated grains. Among children in northern Thailand, aflatoxin poisoning is believed to be the cause of a syndrome consisting of encephalopathy and fatty degeneration of the viscera, manifested by rapid progression from vomiting to convulsions, coma, and (in 80 percent of cases) death. The liver, kidneys, and heart demonstrate fatty degeneration in this syndrome (Coulombe 1991).

Studies of Aflatoxin-Related Chronic Effects

Ecological evidence from India, Southeast Asia, and sub-Saharan Africa supports an association between aflatoxin consumption in humans and primary hepatocellular carcinoma. In Mozambique, average aflatoxin consumption was greatest among the countries studied (222 ng/kg/day), as was the annual incidence of primary hepatocellular carcinoma (35 cases per 100,000 persons at risk). Based on these data, the relationship between aflatoxin exposure and risk of primary hepatocellular carcinoma appears linear.

One potential confounder of the observed ecological association that has yet to be adequately explored is hepatitis B virus infection, a known risk factor for primary hepatocellular carcinoma with high prevalence rates in countries with high aflatoxin consumption per capita. Groopman, Hall, Whittle, and colleagues (1992) showed that hepatitis infection per se does not influence aflatoxin metabolism. Qian, Ross, Yu, and colleagues (1994) recently demonstrated a strong interaction between aflatoxin exposure and hepatitis B infection, such that the odds ratio for those with urinary aflatoxin metabolites and hepatitis B was 59.4 (lower 95 percent confidence limit 16).

Other epidemiological studies of the association between aflatoxin consumption or respiratory exposure and primary hepatocellular carcinoma have been difficult to conduct because primary hepatocellular carcinoma is rare and exposure histories are difficult to obtain or infer. In one cohort study of peanut processors exposed to aflatoxins (0.04 to 2.5 μg AFB_1/week), a statistically significant increase in the risk of death from all cancer and respiratory tract cancers was found (Coulombe 1991). Recently, biomarkers of aflatoxin exposure have been defined, including AFB_1 adducts of serum lysine and albumin, as well as urinary metabolites of AFB_1 (Coulombe 1991). A recent risk assessment modelling primary hepatic carcinoma risk as a function of aflatoxin exposure estimated a lifetime excess risk of 1 per 100,000 for each 253 ng/day increase in exposure (Hoseyni 1992).

STUDIES OF ADVERSE EFFECTS RELATED TO SWINE AND CATTLE CONFINEMENTS/MANURE PITS

Exposure of agricultural workers to toxic gases generated during the decomposition of swine and cattle waste may occur within animal confinements or manure pits that are used to hold the waste prior to disposal or use as fertilizer. Typically, excreta pass through slotted

floors and into a waste storage tank or pit where anaerobic degradation (fermentation) ensues, yielding an atmosphere within the confinement pit that contains alcohols, acids, amines, carbonyls, sulfides, nitrogen heterocycles, esters, mercaptans, disulfides, and the "fixed gases" (carbon monoxide, carbon dioxide, methane, ammonia, hydrogen sulfide) (H_2S), as well as particulate matter that may include animal proteins, plant and fungal proteins, bacteria, viruses, and fungal spores. Relative oxygen depletion occurs near the surface of the manure. The atmosphere created may be extremely hazardous. Proper ventilation, knowledge of the effects of disturbing the liquid manure, and use of personal protective gear (clothing, eyewear, gloves, respirator) are essential to working safely in these settings (Donham, Rubino, Thedell, and Kammermeyer 1977; Donham 1983).

Respiratory irritation, organic dust toxic syndromes, hypersensitivity syndromes, infection, acute intoxication, and asphyxia may occur in workers exposed to the atmosphere of animal confinements and manure pits. Of these, acute intoxication and asphyxia will be addressed in this section (see the sections on Hypersensitivity pneumonitis, Organic dust toxic syndrome, and Zoonoses for discussions of these other topics).

Acutely hazardous conditions within the confinement develop within hours when ventilation systems are not in operation or when the manure is agitated. The manure pit itself is always hazardous and entry should be undertaken only with a self-contained breathing apparatus. The epidemiology of agricultural worker injury associated with animal confinements and manure pit exposure is not well defined, but a disproportionate number of deaths associated with these exposures occur between April and September (CDC 1993).

In a stagnant manure pit, the layer of gas above the scum layer is oxygen depleted and rich in CO_2 and H_2S, both of which are heavier than air. Several fatalities have been reported among workers overcome by H_2S upon entering stagnant manure pits (CDC 1981). H_2S blocks cytochrome oxidase activity and causes cessation of aerobic cellular respiration (Shaver and Tong 1991). Methane asphyxia also has been described among farm workers entering stagnant manure pits (CDC 1989). Respiratory protection was not used by those workers fatally overcome by these toxic gases.

Manure pits are routinely agitated to suspend the solids before the manure is pumped out. Agitation results in the rapid release of CO_2, H_2S, and ammonia, and the concentration

of these gases may exceed safe limits even when ventilation systems are operational. Farm worker and livestock deaths within confinements have been described following agitation of manure pits (CDC 1978). Not uncommonly, several farm workers will succumb while attempting to rescue their co-workers from the pits. Between 1982 and 1992, NIOSH investigated 104 fatalities occurring in 68 incidents involving animal confinements; 35 percent of these fatalities occurred during rescue attempts (CDC 1993).

STUDIES OF ADVERSE EFFECTS RELATED TO SILO FILLER'S DISEASE

General

Farmers raising livestock frequently feed them silage, and these workers are at greatest risk for silo gas exposure. Silage, a fermented form of farm-animal feed, is formed when bacteria partially digest corn, oats, and/or alfalfa. The hypoxic and acidic silage resists spoilage due to molds, and is more nutritious than the forage used for substrate. Silage is formed within a closed fermentation environment, the silo. Silos can be classified by the location of the silage unloading area. In both top and bottom unloading silos, forage is usually blown into the silo and leveled. Bottom-unloading silos are closed once filled, and silo gases rapidly accumulate within them. Top-unloading silos (the most common form) are not sealed, but silo gases, which are heavier than air, form a concentrated layer above the silage. The concentration of silo gases is much greater in bottom-unloading silos, and after they have been sealed, entry is only survivable with a self-contained breathing apparatus. Top-unloading silos have doors for entry that are arranged vertically and are accessible by ladder. Farmers can assess the level of silage by percussion of the doors, and when above the level of silage, may open the door and walk onto the top of the silage. Silo gases may leak and accumulate in low locations in or around the silo as well (Douglas, Hepper, and Colby 1989).

The atmosphere within the silo is rapidly depleted of oxygen, and concentrations of carbon dioxide and oxides of nitrogen increase. Nitrogen dioxide (NO_2) and dinitrogen tetroxide (N_2O_4) begin to accumulate immediately. Levels of NO_2 are commonly found to be in the range of 200 to 2,000 parts per million (ppm). The production of nitrogen oxides subsides after a few days, but silo gases remain in a concentrated layer above the silage for

up to many weeks (depending on how tightly the silo is sealed) (Douglas, Hepper, and Colby 1989). Natural ventilation and the use of forage blowers before entry into silos usually provides a safe atmosphere for farmers (Groves and Ellwood 1989).

Nitrogen dioxide has a characteristic odor similar to that of bleach. It may be colorless or slightly yellow, and it may stain silage or other things that are exposed to high concentrations. When ambient humidity is low, or when an individual is exposed to low concentrations of NO_2 for more than a few minutes, the ability to smell NO_2 is blunted. The presence of dead insects, birds, or small mammals should be recognized as a warning that NO_2 concentrations are hazardous (Douglas, Hepper, and Colby 1989).

Acute Effects of Silo-Filler's Disease

In experimental models of NO_2 toxicity, damage to lung parenchyma, pulmonary microvasculature injury, decreased pulmonary and systemic vascular tone, diminished oxygen-carrying capacity of blood, and disturbed acid-base status have been described. Any of these effects may cause death or permanent injury. The pulmonary effects include bronchospasm, protein damage due to free-radicals, impaired surfactant function, capillary leakage and pulmonary edema, epithelial desquamation, and ciliary destruction. Oxygen diffusion into the circulation can be greatly decreased, resulting in hypoxemia. Nitrite ions cause vascular smooth muscle relaxation, resulting in increased pulmonary blood flow (worsening pulmonary edema) and decreased systemic blood pressures with resultant hypoperfusion and tissue hypoxia (shock). Iron in hemoglobin is reduced, with resultant methemoglobinemia and reduced ability of the hemoglobin molecule to carry oxygen to the tissues. Combined respiratory and metabolic acidosis ensues due to hypoventilation, lactic acidosis, and accumulation of nitric and nitrous acids (Douglas, Hepper, and Colby 1989).

Exposure to NO_2 in concentrations of 200 ppm or greater almost instantly renders the victim unconscious. Silo gas mixtures with high concentrations of NO_2 tend to be extremely oxygen depleted (1 percent at the silage surface) and have high levels of carbon dioxide (19 percent at the surface). The combination of these factors accounts for the rapidly fatal effects of silo gases that have been described (Groves and Ellwood 1989). Lesser exposure concentrations of NO_2 (50 to 100 ppm) may cause only mild irritation of the eyes and throat

at the time of exposure, but may later cause significant pulmonary injury to the airways and alveoli (Douglas, Hepper, and Colby 1989).

In a study of New York farmers, an estimated annual incidence of five silo gas poisonings per 100,000 farm workers having contact with silos was observed. Twenty percent of these poisonings were lethal (Zwemer, Pratt, and May 1992).

In another case series of 17 farmers exposed to silo gases, all silos were reportedly top-unloaded. In each case, exposure occurred when the farmer entered or climbed the silo within a few days or on the day of filling. Exposure concentrations were not described in this series. The symptoms described during these exposures were (in order of decreasing frequency) cough, light-headedness, dyspnea, tightness of the chest, choking, loss of consciousness, audible wheezing, chest pain, weakness, and irritation of the eyes and throat. Late effects included dyspnea (uniformly), cough, chest pain, tachypnea, chest tightness, headache, fever, insomnia, audible wheeze, chills, light-headedness, and myalgias (Douglas, Hepper, and Colby 1989).

Chronic Effects of Silo-Filler's Disease

The late effects of exposure to silo gases include susceptibility to respiratory infection, reactive airways, exertional dyspnea, and bronchiolitis obliterans (Douglas, Hepper, and Colby 1989).

STUDIES OF EFFECTS RELATED TO ORGANIC-DUST TOXIC SYNDROME

Organic-dust toxic syndrome has been described among agricultural workers involved in grain, poultry, pig, and dairy production, activities that generate large amounts of organic dust (Rylander, Essle, and Donham 1990; Donham, Leistikow, Merchant, and Leonard 1990). For example, swine confinements have high concentrations of airborne particulates, and these contaminants are 20 to 30 percent protein in composition (Donham, Rubino, Thedell, and Kammermeyer 1977). Following exposure to large amounts of respirable organic dusts, workers may develop a self-limited, inflammatory respiratory illness called organic dust toxic syndrome (Seigel, Olenchock, Sorenson et al. 1991). These symptoms have onset hours after exposure to moldy hay, silage, wood chips, grain, or pigs. Extreme

contamination of these organic media with bacteria, fungi, and endotoxin has been described (Dutkiewicz, Olenchock, Sorenson et al. 1989). The symptoms are typically seen among all workers who are exposed, and are relatively short-lived. Endotoxins, mycotoxins, proteinases, and endogenous histamine appear to be responsible for the symptom constellation (Seigel, Olenchock, Sorenson et al. 1991; do Pico 1992).

Annual period prevalence estimates for these symptoms range from 6 to 15 percent among farmers in general to 16 to 32 percent among grain workers. In some studies, the lifetime prevalence of characteristic symptoms was estimated at approximately 44 percent (do Pico 1992). Among swine confinement workers in the United States, Canada, and Sweden, the prevalence of symptoms consistent with organic-dust toxic syndrome is 10 to 30 percent (Donham 1990).

Following acute, massive exposure to organic dusts, fever, malaise, myalgia, headache, dry cough, chest tightness, wheezing, mild dyspnea, nasal and throat irritation, and other flu-like symptoms have been described. Typically, no radiographic abnormalities are noted, and pulmonary function studies remain approximately normal. Bronchoalveolar lavage and lung biopsy reveal polymorphonuclear leukocytic alveolitis and bronchitis with no granulomata. Lavage fluids have been productive of large numbers of spores, and *Aspergillus*, *Penicillium*, and *Actinomycetes* species have been isolated (do Pico 1992). Organic-dust toxic syndrome is not associated with known long-term sequelae (Zejda 1993; do Pico 1992).

STUDIES OF EFFECTS RELATED TO FARMER'S LUNG (HYPERSENSITIVITY PNEUMONITIS)

According to Zejda, McDuffie, and Dosman (1993), hypersensitivity pneumonitis resulting from IgG and IgA mediated immunologic responses to proteins of *Faeni rectivirulga* (formerly *Mycropolyspora faeni*), *Thermoactinomyces vulgaris*, other fungi, and animals is not clinically confirmed. Precipitin serum antibody to antigen present in the work environment is classically isolated (in 90 to 100 percent of cases during acute exacerbation) (Pepys 1983). Moldy hay and silage are frequently implicated as the relevant exposure (do Pico 1992), but malt, sugar cane, and mushroom workers, as well as bird breeders, have experienced the disease (Schenker, Ferguson, and Gamsky 1991). Survey data suggest that 0.1 to

15 percent of farmers may suffer from hypersensitivity pneumonitis (Zejda 1993). Others estimate the prevalence of hypersensitivity pneumonitis to be 4.2/1,000 farmers at risk (Marx, Guernsey, Emanuel et al. 1990). The disease occurs most frequently during the winter months in areas with generous rainfall (Pepys 1983).

Acute signs and symptoms include fever, malaise, rigors, myalgia, weight loss, dyspnea, hemoptysis (rarely), and basal rales that occur 5-6 hours after exposure. Diffuse, nodular, and patchy infiltrates may be seen on chest X-ray. Pulmonary function tests are abnormal, with decreases in tidal volume, total lung capacity, lung diffusion capacity, and arterial oxygenation. Bronchoalveolar lavage demonstrates lymphocytosis in the airways, and lung biopsy characteristically demonstrates a mononuclear alveolitis with granuloma formation (do Pico 1992).

Farmer's lung is a recurrent respiratory illness that may occur with even minor exposures to the inciting agents. Farmer's lung is manifested as non-specific respiratory symptoms initially but later may progress to pulmonary fibrosis and chronic respiratory insufficiency (Zejda 1993). Recurrent episodes are not required for progression to pulmonary fibrosis to occur (Pepys 1983).

Use of respiratory protection and avoidance of offending antigens are reasonable control measures. Severely affected individuals may be unable to work in the offending environment.

SKIN SENSITIZERS (OTHER THAN PESTICIDES)

Many chemicals used in agriculture can sensitize the skin, cause or exacerbate derma tologic pathology, or promote systemic allergic responses. These have been reviewed by Abrams, Hogan, and Maibach (1991). Deet (insect repellent) may cause irritant dermatitis, and has been associated with exacerbations of acne vulgaris and seborrhea. Contact dermatitis has been described following exposure to cow saliva, hypochlorite, phenolic compounds, ammonium compounds, cow hair, cow amniotic fluid, animal feeds, iodine and related compounds, furazolidone, hydroquinone, and halquinol. Severe eczema has been reported following dermal exposure to quinoxaline (a feed additive used outside the United States). Allergic contact dermatitis (urticarial reactions) have been reported following exposure to

horse saliva, ethoxyquin, olaquindox, nitrofurazone, piperazine, phenothiazine, levamisole, nitrofurazone, tylosin, penethamate, and chromate. Photosensitivity following quindoxin, olaquindox, and chlorpromazine contact has been reported (Abrams, Hogan, and Maibach 1991).

ALLERGENS AND RESPIRATORY IRRITANTS

Respiratory irritant exposures are common among farmers. These irritants include inorganic dusts and gases, pollens and grains, bacteria and fungi, animal dander and other proteins, workplace chemicals (solvents, pesticides, fertilizers), animal waste decomposition products, storage mites, and household irritants. Allergic rhinitis, conjunctivitis, and respiratory symptoms are common among farmers (Schenker, Ferguson, and Gamsky 1991). Marx, Twiggs, Ault, and colleagues (1993) showed that Wisconsin dairy farmers were more likely than controls to have immunologic sensitivities to house dust and storage mites, grain smuts, *Cladosporium*, *Aspergillus*, and cattle.

Exposure to bulk soybeans has been strongly implicated as a cause of epidemic asthma during loading and unloading operations (Anto, Sunyer, Rodriguez-Roisin et al. 1989). Senthilsevan, McDuffie, and Dusman (1992) found self-reported carbamate insecticide use and asthmatic symptoms to be correlated in a cross-sectional study of male Canadian farmers. Decrements in pulmonary function and increased respiratory symptoms were associated with a history of carbamate use.

Chronic bronchitis (cough and copious phlegm production) has been reported in 5.6 percent to 21 percent of farmers surveyed, placing non-smoking farmers at three times the risk of non-smokers in other industries for having these symptoms. Among grain workers, a chronic bronchitis prevalence of 49 percent has been reported. Asthma, or frequent wheezing and shortness of breath, is also three times more prevalent among farmers and animal confinement workers than among controls (Zejda, McDuffie, and Dosman 1993).

Donham (1990) reviewed several studies that addressed respiratory symptoms among swine confinement workers. Chronic cough and copious phlegm production were reported in 12 to 55 percent of the subjects surveyed. These findings support the position that the complex mixture of gases and dusts found in swine confinements predisposes workers to

developing symptoms of occupational asthma and chronic bronchitis (Donham, Rubino, Thedell, and Ramnaermeyer 1977; Donham 1990).

STUDIES OF INFECTIONS IN FARMERS

Zoonoses

Over 150 diseases in humans have been described as zoonoses, and among these, 40 or more represent significant risks to agricultural workers in the United States. According to Cordes and Rea (1988), **tularemia** and **Rocky Mountain spotted fever** are the most common zoonotic infections in the United States. Incidence data describing occupational risk for these is not available, but some estimate thousands of farmers to be affected annually by zoonotic infections (Engberg 1993; Kligman, Peate, and Cordes 1991). Control measures for most zoonotic infections include use of personal protective gear (gloves, boots, aprons, eye protection, respirators), immunizations (of herds, domestic animals, and farm workers), ventilation, isolation or destruction of infected animals, use of insecticides and insect repellents, proper storage and processing of foods, and proper disposal of carcasses, waste products, and products of animal delivery (Kligman, Peate, and Cordis 1991). Personal hygiene measures (hand washing, changing to fresh clothes after contamination, potable water, restroom facilities) are always important for infection control.

Fungal Infections

Ringworm (*tinea corporis* and *tinea capitis*) is a common fungal infection that may be transferred from farm animals to humans. The most frequently involved organisms include *Trichophyton mentagrophytes*, *Trichophyton verrucosum*, *Trichophyton equinum*, and *Microsporum canis*. Direct contact with infected animals is not required for infection, because the hyphae remain viable on cool, dry fomites for periods as long as several years. Infection control measures rely on gloves and protective clothing and the cleaning of hands, other body parts, and equipment that comes into contact with the animals (Abrams, Hogan, and Maibach 1991).

Candidiasis (caused by *Candida* species), an infection of the skin causing itching, redness, and maceration, has been described among poultry workers. Skin trauma and wet

working environments predispose to this infection. Protective clothing, gloves, and keeping dry serve as control measures (Engberg 1993).

Coccidiomycosis (caused by *Coccidioides immitis*) may occur among those working in the soil of arid regions, which is where spores are found. Coccidiomycosis is endemic in the southwestern United States, especially Arizona and southern California (Cordes and Rea 1988). Infection causes pulmonary infection or meningitis. Dust control measures help protect against coccidiomycosis (Engberg 1993).

Histoplasmosis (caused by *Histoplasma capsulatum*) is caused by infection with spores present in soil or dust that is contaminated with the droppings of fowl. It is endemic in the Ohio and Mississippi river valleys. Farmers and poultry workers are at increased risk. Systemic and primary pulmonary infections have been described. Dust control, disinfection, and sanitation measures reduce the risk of infection (Engberg 1993).

Viral Infections

Viral encephalitis (caused by 16 groups of viruses) is the most common, serious arthropod-borne disease in the United States. In 1989, 981 cases were reported. The most common viral encephalitides are St. Louis encephalitis, Eastern Equine encephalitis, and Western Equine encephalitis. Most Western Equine encephalitis occurs in males in agricultural regions. The viral encephalitides are more common during the summer months. They are transmitted to humans by mosquitoes and ticks that have associated with infected woodland mammals and birds (Kligman, Peate, and Cordis 1991). Signs and symptoms include fever, headache, lethargy, and altered mental status. Coma and death have been reported. Preventive measures include regional insect control, use of insect repellant, and protective clothing. If continued or extensive exposure is anticipated, inactivated vaccines for Eastern and Western Equine encephalitis are available from the United States Army (Kligman, Peate, and Cordes 1991).

Pox viruses infecting cows (**pseudocowpox**) and sheep (**contagious ecthyma**) may infect humans via skin contact and cause milker's nodule and orf, respectively. The viruses are hardy, and may remain viable on fomites for many years. Infection produces a self-limited skin manifestation: hard, wart-like nodules on the hands and forearm. Infection con-

trol measures include use of gloves and protective clothing, washing after contact with animals, and vaccination of herds (Kligman, Peate, and Cordis 1991; Abrams, Hogan, and Maibach 1991).

Rabies is caused by a species of lyssavirus, and is a rare cause of disease in humans in the United States in recent decades. However, rabies infection is endemic in the small mammals of many regions of the United States, with 4,000-7,000 infected animals identified each year (Kligman, Peate, and Codis 1991). Farmers at risk for infection are those working with horses or cattle, working near the Mexican border, and those in frequent contact with wild animals. The symptoms of rabies follow infection by periods ranging from a few weeks to several years. These have insidious onset, and may include vague psychiatric complaints, fever, nuchal rigidity, and paresthesias at the original bite site. The pathognomonic signs associated with rabies include hydrophobia (secondary to uncoordinated swallow with aspiration of liquids) and aerophobia (secondary to facial muscle spasms elicited by wind) (Plotkin and Clark 1992). These are followed by excess salivation, progressive encephalitis, ascending paralysis with hyper-reactivity to stimuli, delirium, convulsions, respiratory failure, and death (Kligman, Peate, and Cordis 1991). Infection control involves thorough cleaning of wounds, avoidance of the salivary secretions of wild animals, and immunization of domestic animals, and use of human cell diploid vaccine and rabies immune globulin for high risk contacts.

Bacterial Infections

The agricultural environment can be associated with a number of disease-causing bacteria. *Francisella tularensis* (the cause of tularemia), *Bacillus anthracis* (anthrax), *Brucella* species (brucellosis), *Leptospira interrogans* (leptospirosis), *Yersinia pestis* (plague), *Clostridium tetani* (tetanus), *Salmonella dublin* (salmonella dermatitis), *Erysipelothrix rhusiopathiae* (erysipeloid), and *Staphylococcus* species (folliculitis/cellulitis) may be transmitted from wild or domestic animals to humans, causing the diseases described below (Kligman, Peate, and Cordis 1991; Engberg 1993; Abrams, Hogan, and Maibach 1991).

Tularemia is usually transmitted to humans from cottontail rabbits, although other animals and arthropods may serve as vectors. It is highly infectious. In 1989, 152 cases

were reported in the United States. Hunters, farmers, sheep shearers, and trappers are at greatest risk for tularemia. In ulceroglandular tularemia, erythematous macules appear at the site of inoculation and evolve into pruritic papules. On the fourth day after infection, an ulcer forms at the site of inoculation, accompanied by high fever, suppurative adenitis, headache, myalgia, and hepatosplenomegaly. The oculoglandular form is associated with severe conjunctivitis and facial and cervical adenopathy. The pulmonary syndrome includes cough, pleuritic chest pain, hemoptysis, tracheitis, bronchitis, pneumonia, pulmonary edema, pleural effusion, and hilar adenopathy. Exudative pharyngitis with membrane may be observed, along with symptoms of gastroenteritis (Kligman, Peate, and Cordis 1991). Infection control includes use of protective clothing, eye protection, and gloves, avoidance and isolation of infected animals, insect control, and vaccination (Kligman, Peate, and Cordis 1991; Abrams, Hogan, and Maibach 1991).

Brucellosis can be contracted following handling, contact with secretions or excretions, or consumption of unpasteurized milks or cheeses from infected animals. Placentas and abortuses from infected animals are especially infective (Kligman, Peate, and Cordis 1991). *B. suis* results from contact with infected pigs, *B. abortus* from cattle, *B. melitensis* from sheep and goats, and *B. canis* from dogs. Livestock producers and meat packers are at greatest risk. In 1989, 95 cases of brucellosis were reported in the United States (Kligman, Peate, and Cordis 1991). *B. melitensis* causes the most severe disease in humans. Symptoms include spiking and unpredictable ("undulant") fevers, chills, myalgias, weakness, night sweats, headache, anorexia, abdominal pain, diarrhea followed by constipation, and irritability (Kligman, Peate, and Cordis 1991). Skin signs of brucellosis may include a maculopapular eruption, petechiae, and a chronic ulcer at the site of inoculation, and urticarial or vesiculopapular eruption. Chronic brucellosis may include endocarditis, osteomyelitis, spondylitis, orchitis, cholecystitis, and meningoencephalitis (Kligman, Peate, and Cordis 1991). Infection control involves protective clothing, eye protection, and gloves, good personal hygiene following animal contact, pasteurization of dairy products, immunization of animals with brucella vaccine, and isolation of infected animals (Abrams, Hogan and Maibach 1991; Engberg 1993).

During the period 1980-1989, only five cases of **anthrax** were reported in the United States. Cutaneous anthrax is most commonly contracted following contact with contaminated goat hair, wool, and hides that have been imported from regions in which anthrax is endemic. Infection may follow a scrape or puncture wound, and is manifested initially as a small papule that enlarges to a purple mass with a hemorrhagic vesicle in the center. Central necrosis ensues with eschar formation, and satellite lesions may develop. Dissemination is associated with regional lymphadenopathy (Kligman, Peate, and Cordis 1991). Pulmonary anthrax from inhalation of spores presents as non-specific cough, which evolves to hemoptysis, pulmonary edema, dyspnea, hypoxia, chest pain, necrotizing and hemorrhagic mediastinal lymphadenitis, bacteremia, and sometimes death. Systemic infections are frequently associated with spread to the meninges and hypotension (Kligman, Peate, and Cordis 1991). Infection control includes use of protective clothing, gloves and respirators, fumigation of imported wools and furs, good hygiene following animal contact, and immunization of animals with anthrax vaccine (Abrams, Hogan, and Maibach 1991; Engberg 1993).

Leptospirosis results from contact with the excrement or tissues of infected farm or wild animals, or water contaminated by these sources. Ninety-three cases were reported in 1989. Trappers, farmers in dairy, rice, or sugar cane production, meat inspectors, slaughterhouse workers, and veterinarians are at greatest risk for leptospirosis. Symptoms of leptospirosis include chills, fevers, headache, myalgia, nausea, vomiting, conjunctival suffusion, bradycardia, and rash. More severe infections may be associated with hepatitis (jaundice), hemoglobinuria, nephritis, and meningitis (Kligman, Peate, and Cordis 1991). The mortality rate from leptospirosis is 3 percent to 6 percent. Personal hygiene, use of protective clothing, eye protection, and gloves, animal vaccinations, isolation of infected animals, and rodent control are the best available control measures (Engberg 1993).

Plague is rare among farmers, but can be contracted in the western United States from fleas on infected rodents, coyotes, and rabbits. Hunters and trappers, as well as dock workers, are at greatest risk. Cutaneous, pulmonary, and systemic infections occur. Signs and symptoms include fever, chills, adenitis, bubo formation, pneumonia, and septic shock. Avoidance of infected rodents and the use of protective clothing serve as the best control

measures, and a vaccine is available for workers at high risk (Kligman, Peate, and Cordis 1991; Engberg 1993).

Erysipeloid is a skin infection that workers engaged in fish, poultry and meat-handling operations may contract. The affected skin is warm, raised, pruritic, and erythematous, and can be expected to resolve without intervention in 2-3 weeks. Personal hygiene measures and glove wearing when in contact with animal products serve as the best control measure (Kligman, Peate, and Cordis 1991; Engberg 1993).

Effects Caused by Protozoa and Helminths

Infestation by *Giardia lamblia*, *Toxoplasma gondii*, *Echinococcus* species, *Ancylostoma duodenale*, *Ascaris* species, and *Necator americanus* result from the ingestion of cysts or invasion through the skin by larvae present in the feces of infected farm animals, domestic animals, and humans. Infestation usually involves the gut, but lung, liver, brain, heart, spleen, skeletal muscle, and eye involvement have been described from some of these agents. Personal hygiene, sanitary disposal of human and animal excreta, available potable water, and use of shoes and gloves will reduce the risk of infection (Kligman, Peate, and Cordis 1991; Engberg 1993).

Effects Caused by Chlamydia and Rickettsiae

Chlamydia psittaci, the cause of ornithosis, may cause pulmonary and systemic infections among poultry workers and others who handle birds. Infection results from contact with the fluids or excreta of infected birds. Hygiene and protective clothing, gloves, eyewear, and respirators, as well as isolation and treatment of infected birds, reduce the risk of infections for agricultural workers.

Rocky Mountain spotted fever is a vasculitis caused by *Rickettsia rickettsii*, which is transmitted most commonly by ticks. Farmers, trappers, and foresters are at highest risk. Half of the reported cases in the United States occur in the Southeastern states. Almost all cases occur between April and September. A total of 592 cases were reported in the United States in 1987. A purpuric, vasculitic rash is seen, along with high fever, myalgia, weakness, abdominal pain, non-productive cough, thrombocytopenia, anemia, elevated serum

transaminases, and hyponatremia. Avoidance of ticks and the use of insect repellents and protective clothing reduce the risk of infection (Kligman, Peate, and Cordis 1991; Engberg 1993).

Q-fever can be contracted through respiration of contaminated aerosolized particles, direct skin contact, arthropod transmission, or ingestion. Agricultural workers most at risk include slaughterhouse, dairy, or livestock workers, veterinarians, and hide handlers. Placentas and excreta of infected animals are very infectious, and *Coxiella burnetti* can live for long periods in water or milk. Two to four weeks following infection, acute-onset fever, myalgias, headache, weakness, dry cough, chest pain, and rales develop. Granulomatous hepatitis with jaundice may subsequently evolve. Chronic forms of Q-fever have been described, most commonly as endocarditis (Kligman, Peate, and Cordis 1991). Infection control measures include use of protective clothing, glasses, and gloves. Local control of insect vectors can reduce the spread of epidemic Q-fever, and exposed workers may receive a killed vaccine (Kligman, Peate, and Cordis 1991).

Injury-Related Infections

Injuries resulting from farm machinery accidents typically result in deep, irregular wounds with devitalized tissue and gross contamination with plant matter, soil, and animal excreta. Bacterial flora that are associated with these media may infect farm-related wounds. Infections with enteric organisms (gram positive and negative aerobic and anaerobic bacteria), soil-related organisms, and fungi have been described. These infections can complicate recovery from wounds, and may cause more limitation of function than the original injury. Systemic illness, such as sepsis, secondary spread of infections, and tetanus may also occur (Gordon, Indeck, Bross et al. 1988; Agger, Cogbill, Busch et al. 1986; Brennan, Rhodes, and Peterson 1990).

Tetanus most frequently results from wound contamination with soil, and must be considered a potential pathogen in all agricultural wounds. Sixty-three cases were reported in 1989 in the United States. Infection is followed by the anaerobic growth of *Clostridium tetani*, with production of exotoxin that can enter nerves directly or remain systemic. Three to 21 days following infection, signs and symptoms become manifest: muscle spasms, ab-

dominal rigidity, trismus, risus sardonicus, opisthotonos, spastic skeletal muscle contraction, tonic-clonic convulsions, respiratory embarrassment, and death (in 50 to 90 percent of cases). Preventive measures include meticulous wound cleaning and debridement, healing by secondary intention, tetanus immunization, and tetanus immune globulin (Kligman, Peate, and Cordis 1991).

CHAPTER V. INJURIES RELATED TO FARM ANIMALS AND EQUIPMENT

ACCIDENTS RELATED TO POWER TAKE-OFF DEVICES

The National Traumatic Occupational Fatality surveillance survey data demonstrated that, between 1980 and 1988, an average of 16 workers aged 16 or older were killed annually in the United States as a result of power take-off (PTO) entanglement. Between 1982 and 1986, 148 emergency room visits were attributed to non-fatal injury secondary to PTO entanglement (CDC 1992). Entanglement in PTO devices is more common between May and September. Risk factors for PTO entanglement include absent or incomplete shielding, loose clothing, windy conditions, elevated driveline, and longer hair (CDC 1992).

Power take-off devices, also known as secondary drivelines, transmit energy (torque) generated by a farm tractor to other machines in the farmyard, such as augers, hay balers, and hay bale throwers. The PTO functions like a driveshaft, turning at speeds up to 1,000 revolutions per minute. Many PTO devices still in use are either not shielded or are inadequately shielded, leaving farm workers and others in the vicinity exposed to a rapidly spinning shaft unless the PTO has been disengaged. The spinning PTO may be low to the ground (as when in use to drive an auger), or at chest or head-level (when in use to drive a hay baler or hay bale thrower) (Heeg, ten Duis, and Klasen 1986).

Several types of injuries resulting from contact with PTOs have been described. Clothes and hair entangle easily in PTO devices. "Scalping," or the avulsion of the hair and skin of the scalp, neck, and face, have been described following entanglement of hair (CDC 1992). Avulsion or degloving of soft tissue of the limbs and genitals frequently results when clothing becomes caught in the PTO. Traumatic amputations of the limbs and multiple fractures of the limbs and vertebrae have been described as well. Finally, multiple blunt traumatic injuries to the head, thorax, abdomen, and extremities have been described when people whose clothes are entangled are thrown into nearby, immovable structures or machinery (Heeg, tenDuis, and Klasen 1986). In one series from the Mayo Clinic (McElfresh and Bryan 1973), 117 non-fatal PTO-related injuries occurring over the preceding 15 years were described. Forty-six percent of victims had fractures (one-third of

which were open fractures), 33 percent had extensive soft-tissue injury, and 11 percent had traumatic amputations (McElfresh and Bryan 1973).

ACCIDENTS RELATED TO AUGERS

Auger injuries have been implicated in up to 50 percent of all machinery-related deaths in North America, and in 50 percent of traumatic amputations in children in Winnipeg, Canada (Letts and Gammon 1978). In one series, the peak incidence of auger injuries to children associated with grain operations occurred in the Fall (Letts and Gammon 1978).

The auger is a corkscrew device (Archimedes screw) that resides within an encasement for its entire length (with the exception of the two ends). When an auger is turning, it will efficiently move grain, corn, meat, water, or other agricultural products from the intake area (usually on ground level) to the outflow area (usually the top of a silo or into a combine). The intake area is open to varying extent (depending on use and type of safety shield, as well as the product being moved). The intake area may lie within or beneath a hopper, or it may be buried within a pile of grain. Augers may operate at up to 600 revolutions per minute, and they are difficult to jam or stall. Injury occurs when an extremity, hair, or clothing becomes caught in the screw mechanism and the worker is pulled into the encasement (Letts and Gammon 1978).

Death due to hypovolemia (secondary to exsanguination), sepsis, and asphyxia have been attributed to auger accidents (Grogono 1973). Limb amputations are more commonly proximal, above the knee or elbow, making prosthetic fitting difficult (Letts and Gammon 1978). The limbs of children tend to be amputated at more proximal levels because they feed more easily into the encasement (Grogono 1973). Open and closed fractures, dislocations, lacerations, avulsion, and degloving injuries (in which the soft tissue is stripped from the bone) have all been described as auger injuries. Auger wounds are extremely prone to infection for two reasons: the wounds are extremely contaminated, and frequently several hours pass between the time of injury and debridement. Debridement of auger wounds is difficult because the material in the auger becomes deeply embedded within the soft tissues. Infection of auger wounds is so prevalent that primary closure (i.e. suturing the wound

closed) is usually contraindicated. Clostridial infections, and subsequent gas-gangrene, have also been described (Letts and Gammon 1978).

ACCIDENTS RELATED TO CORN-PICKERS

Corn-picker injuries are most common in October and November, and most frequently occur between the hours of 11:00 and 13:00 or between 15:00 and 17:00 (Campbell, Bryan, Cooney, and Ilstrup 1979; Proust 1993). Nearly 90 percent of farmers suffering a corn-picker injury are left with permanent disability of the affected hand (Campbell, Bryan, Cooney, and Ilstrup 1979).

The corn-picker machine consists of several pairs of side-by-side rollers rotating in opposite directions. These rollers "snap" the ear from the stalk and deposit them onto a gathering chain or into an auger that transports the ears to the combine for removal of the husks. Stalks can jam the snapping rollers, and must be removed before operations can proceed. If the corn-picker is not turned off before the jammed stalk is removed, it instantly may pull the stalk (or tool) along with the farmer's hand into the snapping rollers when the jam is relieved (Gorsche and Wood 1988). Hardin and Robinson (1950) were among the first to report the characteristic hand injuries associated with entrapment in a corn-picker.

The snapping rollers of the corn-picker characteristically cause severe hand injuries, usually of the dominant hand, involving friction burns, crushing of both soft tissue and bone, avulsion or degloving of soft tissue, and amputation of the fingers and distal hand (Melvin 1972). In one series, primary amputation of some part of a digit occurred in 36 percent of cases, and 60 percent suffered at least one fracture (Campbell, Bryan, Cooney, and Ilstrup 1979). The wounds are contaminated with debris from the corn stalk, dirt, and grease, and the farmer is frequently trapped by the corn-picker for several hours before the hand can be freed (Proust 1993). In one series, the average time that the hand was stuck in the corn-picker was 27 minutes (Campbell, Bryan, Cooney, and Ilstrup 1979). In another series, 10 percent of farmers had to self-amputate the entrapped limb to gain freedom (Melvin 1972).

Corn-picker injuries have been classified on the basis of severity. Type I injuries are those in which all fingers and the thumb are amputated or devascularized. In type II injuries,

the thumb is spared but all remaining fingers are lost. In type III injuries, the thumb and at least one other finger remain vital. Corn-pickers rarely cause amputations at a higher level than the mid-hand in adults because the metacarpal bones jam the snapping rollers. Farmers are at risk of suffering a corn-picker injury to the other hand when trying to free the hand which is jammed within the snapping rollers, and double amputations have been described (Gorsche and Wood 1988). Dislocations of the elbow and/or shoulder on the affected side are common, as are tendon and ligament injuries (Proust 1993).

Approximately 50 percent of corn-picker injuries in one series became infected to such an extent that further operative procedures or delayed hospital discharge was necessitated. Most of these infections become clinically apparent within the first five days following injury. *Klebsiella, Enterococcus, Pseudomonas, E. coli*, and *Staphylococcus* are most commonly cultured from corn-picker wounds (Melvin 1972). In another series, 24 percent of corn-picker injuries had documented infections with *Enterobacter* species, *Xanthomonas maltophila*, and *Serratia* species (Cogbill, Steenlage, Landercasper, and Strutt 1991). Tetanus has also been reported following corn-picker injury (Melvin 1972).

Corn-picker injuries are treated by provision of anesthesia, thorough debridement, and delayed closure. Reconstructive efforts to provide an optimally useful appendage may involve construction of an ulnar stump for apposition of the thumb if it remains, or toe-to-hand transplantation (Gorsche and Wood 1988; Proust 1993).

ACCIDENTS RELATED TO HAY BALERS

Modern roll hay balers rely on bale-forming belts and rollers to form and tie bales of hay weighing up to one ton. The rollers may become jammed, and when relieving the obstruction, the hand of the operator may become entrapped. Friction burns, crush injuries, neurovascular disruption, avulsion, or degloving of soft tissue may ensue. Traumatic amputation and open, comminuted fractures occur frequently (most commonly of the humerus). In one series, all cases had effective devitalization of the involved extremity, requiring amputation (Gainor 1983). In another series, 50 percent required forequarter amputation and nearly all required extensive skin grafting (McKinnon, Robinson, and Masters 1967). The

secondary drivelines (power take-off) of hay balers have been associated with scalp avulsion injuries among women farmers whose hair became entrapped (CDC 1992).

Within the last decade, a combination of injuries consisting of a compression fracture of the twelfth thoracic vertebra and a sternal fracture has been seen in farmers hit by a falling hay bale while seated behind the steering wheel of a hay bale loader. The bale causes extreme flexion of the torso and crushes the twelfth vertebra. Additionally, the bale drives the anterior chest into the steering wheel, resulting in a sternal fracture and, often, a flail chest. This combination of fractures has been named the hay baler's fracture (Mayba 1984).

ACCIDENTS RELATED TO TRACTORS

According to NSC data, approximately one-quarter of all fatal accidents occurring on farms involve tractor roll-overs. Falls from tractors and run over injuries account for a significant proportion of tractor-related fatalities as well (Stoskopf and Venn 1985). Purschwitz and Field (1990) estimates that 27 percent to 53 percent of fatal farm accidents involve tractors. NSC data (1993) suggest a decrease in the rate of tractor-related fatalities over the last 20 years. According to the Centers for Disease Control, approximately 132 deaths due to tractor roll-over occur annually in the United States (CDC 1993). In 1992, 6.8 deaths per 100,000 tractors were reported. Among these deaths, 53 percent were due to tractor roll-overs, and 25 percent resulted from run-over accidents (NSC 1993). These estimates have remained stable since the early 1980s (Etherton, Myers, Jensen et al. 1991).

According to the Centers for Disease Control, 202 deaths associated with farm tractors occurred in Georgia between 1971 and 1981, accounting for a crude annual death rate of 23.6 tractor-associated accidental deaths per 100,000 male farm residents. Ninety-eight percent of the victims were males, 82 percent were white, and 15 percent were black. Seventy-two percent of deaths occurred to people who were older than 40 years. Seasonal peaks in accidental deaths were noted in March and April, and in July and August. Most fatal accidents occurred between 4:00 and 5:00 p.m. Approximately 90 percent of the tractor fatalities occurred on farms. Roll-overs accounted for 76 percent of these fatalities, and 14 percent of the victims were run over. The most common injury accounting for death was crush-

ing of the chest (83 percent), followed by extravasation, strangulation/asphyxia, and drowning (CDC 1983; Goodman, Smith, Sikes et al. 1985).

Roll-over protective structures (ROPS) provide tractor drivers with a crush-proof cab for protection in the event of a roll-over accident. According to a large survey by NIOSH, more than half of all tractors in use in the United States lack ROPS, and 54 percent of all tractor-use hours were performed with tractors lacking ROPS (CDC 1993). ROPS are very effective in preventing death during a tractor roll-over, especially when safety harnesses are used to keep the worker within the protected zone. In one series, 40 percent of workers involved in roll-over accidents with no ROPS died, compared with 2 percent of those in roll-over accidents in which ROPS were in place (CDC 1993).

ACCIDENTS CAUSED BY FARM ANIMALS

General

Most animal-associated pathology results from trauma or infection. However, in their population-based, matched-pair, case-control study of multiple myeloma in Sweden, Eriksson and Karlsson (1992) found an association with exposure to domestic animals (especially horses, goats, and cattle).

Bovine and Equine Trauma

The incidence of trauma due to farm animals is not known. In a survey of farm injuries, 18 percent were classified as animal-related. Of these, the legs, back, hands, and arms were the sites of injury for 18.5 percent, 10.6 percent, 10.1 percent, and 9.7 percent of injuries, respectively. Thirty-five percent of these injuries occurred on the first day the victim worked with the animal. Sixty-seven percent of the accidents occurred when the animal was standing, walking, or running. Seventeen percent occurred while milking an animal. Cattle and horses accounted for 57 percent and 27 percent of injuries, respectively (Hoskin and Miller 1979).

In one case series of 134 patients admitted to the hospital for bovine or equine trauma, one-third of the injuries resulted from falls off horses, 21 percent from kicks by cows, 19 percent from bovine assaults, 13 percent from equine assaults, and eight percent

from kicks by horses. The remaining injuries (6 percent) resulted from accidents involving animal-drawn vehicles. Among the victims of these injuries, 40 percent were farmers, 30 percent were children of farmers, 13 percent were wives of farmers, and 2 percent were veterinarians (Busch, Cogbill, Landercasper, and Landercasper 1986). Cow kicks were most frequently associated with trauma to the leg or face of the victim. Most falls from horses resulted in head or arm injuries. Thoracic, abdominal, and multi-system trauma was most common among those assaulted by cows and horses (Busch, Cogbill, Landercasper, and Landercasper 1986).

Among veterinarians, 63 percent of whom reported working with large animals, most (64 percent) had sustained a serious injury within the last year while working with animals. Of these injuries, the hands, arm, and head were involved 53 percent, 28 percent, and 21 percent of the time, respectively. The mechanisms of injury most commonly included kicks (36 percent), bites (34 percent), and crush injuries (12 percent). Reports of injuries sustained by animals pushing, head butting, goring, running over, and falling on the veterinarian were also described, and these mechanisms accounted for 15 percent of the reported injuries (Landerscasper, Cogbill, Strutt, and Landerscasper 1988).

Prevention measures include effective animal restraint, avoidance of enclosed spaces containing animals, moving slowly, avoidance of threatening movements around young animals, and care when milking animals.

Wild Mammals

Wild mammals encountered by farmers may act as reservoirs for organisms that have the potential to cause serious infections. These infections will be discussed in the section on zoonoses. Other injuries, such as bites and mauling by wild animals, are very rare.

Venomous Insects and Reptiles

Farm workers may come into contact with snakes and insects that envenomate to incapacitate or digest the animals that threaten or sustain them. The venomous insects include *Hymenoptera* species (wasps, hornets, fire ants), *Latrodectus* and *Loxosceles* species

(black widow and brown recluse spiders), *Centruroides* and *Hadrorus* species (scorpions), and *Scolopendra heros* (giant desert centipede).

Local effects of envenomation by these insects include: pain, erythema, edema and localized areas of warmth. Extensive tissue destruction at the site of envenomation is characteristic of bites by the brown recluse spider. Systemic effects include neurotoxic effects, hemolysis, hypertension, respiratory compromise, and shock (either neurogenic or anaphylactic) (Hassen 1991).

The venomous reptiles that present a health risk to farm workers include members of the *Crotalus*, *Sisturus*, and *Agkistrodon* species of pit vipers (rattlesnakes, copperheads, cottonmouth moccasins), *Micrurus* and *Micruroides* species (coral snakes), and *Heloderma suspectum* (gila monster). Local effects include bite marks, retained teeth, pain, swelling, erythema, ecchymosis, bullae, paresthesias, lymphangitis, atrophy, and contracture.

Systemic effects vary by reptile group. Pit viper venoms may cause central nervous system depression, nausea, vomiting, coagulopathy, rhabdomyolysis, renal impairment, shock, and acidosis. Coral snake venom causes lethargy, weakness, fasciculations, bulbar palsy convulsions, respiratory failure, and shock. Gila monster venom may cause hypotension, weakness, diaphoresis, and shock (Hassen 1991).

ACCIDENTS CAUSED BY ELECTRICITY

According to the Centers for Disease Control (1983), farmers are at significantly increased risk for death by electrocution. The proportionate mortality ratio (PMR) for death due to electrocution among male farmers in Washington state between 1950 and 1979 was 223 (42 deaths observed, 18 expected) (Helgerson and Milham 1985). Of the 42 electrocution deaths of farmers, 23 (53 percent) were "irrigation-pipe associated," resulting from contact between an irrigation pipe held by a farmer or farm worker and a power line. Between 1970 and 1979, 79 percent of electrocution deaths of farmers resulted from contact with power lines mediated by an irrigation pipe (Helgerson and Milham 1985). Most of these deaths occurred between April and September, and workers younger than 30 years old were at greatest risk (CDC 1983).

CHAPTER VI. STUDIES OF ENVIRONMENTAL RISK FACTORS

HEAT/FLUID DEPRIVATION CONDITIONS

Farmers are at risk of suffering from "exposure" syndromes because they work outdoors for prolonged periods. In warmer climates or seasons, illnesses caused by excess heat are often seen among farmers. According to Brown (1991), 15 California farmers died of heat stroke between 1955 and 1963, and sporadic cases have been reported since. The strenuous nature of many farm activities results in increased metabolism and endogenous heat production. This state of increased metabolism, coupled with high environmental temperatures, radiant energy from direct sunlight, high humidity, and limited access to water (and electrolytes) may overwhelm the homeostatic mechanisms needed to maintain a euthermic body core.

Heat cramps, syncope, exhaustion, and stroke may result. **Heat cramps** in the skeletal and abdominal muscles are caused by hyponatremia, when adequate free water is taken to replenish losses but the sodium content is inadequate to make up for salt losses. In heat syncope, relative hypovolemia ensues from a combination of decreased fluid intake, increased insensible fluid loss, and peripheral vasodilation (to facilitate dissipation of heat). **Heat exhaustion** frequently also consists of cramps and syncope; however, somnolence, nausea, vomiting, anorexia, tachycardia, and impaired judgment are also observed. In water-depletion heat exhaustion, hypernatremia, decreased sweating, and intense thirst are seen. Core temperature is elevated (37.5° to 38.5°C). In salt-depletion heat exhaustion, hyponatremia with persistent sweating is observed, and core temperature may be normal or low. Again, mental status changes are observed. **Heat stroke** is a medical emergency. It reflects profound inadequacy or loss of control over homeostatic mechanisms. Major central nervous system dysfunction (convulsions, coma, delirium), lack of sweating, and significant elevation in core temperature (41°C) are signs of heat stroke. Among farmers, exertional heat stroke results from failure to adequately dissipate heat. Hypotension, acute renal and hepatic failure, coagulopathy, and rhabdomyolysis may be observed (Brown 1991).

Heat stress is difficult to quantify since it is a function of environmental conditions (temperature, humidity, wind, direct sunlight), the metabolic heat generated by the work

activity, and the condition of the farmer (fat content, hydration, and electrolyte status, acclimatization, clothing, other medical conditions or medications). One measure of occupational heat stress, the heat Bulb Globe Temperature, has been modified by NIOSH to yield recommended exposure and alert limits, which provide maximal exposure durations at given metabolic and environmental heats (Brown 1991).

SUN-RELATED CONDITIONS

The risk of developing non-melanotic skin cancers (basal cell and squamous cell carcinomas) increases as exposure to ultraviolet radiation increases. Therefore, outdoor workers, including farmers, are at increased risk for the development of these cancers (Abrams, Hogan, and Maibach 1991). Blair and colleagues (1991) reviewed nine studies that addressed the risk of cancer of the lip, 12 that dealt with melanoma risk, and eight that covered non-melanotic skin cancer risks among farmers. Of these studies, nine, eight, and seven showed excess risks, respectively. These findings support the hypothesis that increased ultraviolet exposure among farmers increases their risk of solar-related cancers (Blair et al. 1991). In a mortality study of Wisconsin farmers, proportionate mortality due to cancer of the eye was elevated (PMR 3.75). Sunlight was postulated to be a likely etiologic agent (Saftlas, Blair, Cantor et al. 1987).

COLD INJURIES

In general, farmers are at increased risk for cold-induced injures, but this is especially true for older, malnourished, smoking, fatigued, or alcohol-using workers. Cold injuries may be local, affecting a digit, appendage, or extremity, or they may be systemic. Incidence data on cold-induced injury among farmers are not available. **Frost nip** is not uncommon, and involves reversible ice crystal formation in the skin surface, with no skin loss during resolution. **Chilblains**, usually involving the hands or feet, result from recurrent exposure to cold, wet conditions. Pale skin with paresthesias or anesthesia is seen initially, but vascular injury becomes apparent 2 to 3 days after re-warming, and is manifested by hyperemia, ecchymosis, hemorrhage, edema, cellulitis, thrombophlebitis, and gangrene. In **frostbite**, the subcutaneous tissue and muscle are frozen; this condition is manifested as edema,

erythema, paresthesia, anesthesia, and waxy, hard skin. **Systemic hypothermia** (core temperature $<35°C$) presents clinically as depressed mental status, slurred speech, weakness, lethargy, combativeness, irritability, and cool skin and face (Brown 1991). Progressive hypothermia may result in cardiovascular collapse and apnea.

CHAPTER VII. EFFECTS RELATED TO PHYSICAL HAZARDS

NOISE-INDUCED HEARING LOSS

Farmers are exposed to excessively loud sounds when working with farm machinery or within animal enclosures. Noise-induced hearing loss typically results in damage to the hair cells of the cochlea, which sense 4000 Hz frequencies, and as the damage progresses, audition of both higher and lower frequencies becomes impaired. OSHA currently permits continuous noise exposure to 90 decibels over an 8-hour workshift. Tractors frequently expose farm workers to sound that is louder than 85 decibels (Crutchfield and Sparks 1991). Swine confinements have noise levels greater than 100 decibels during feeds, and horseshoe fitting is associated with 98 to 120 decibel exposures (Donham 1983; Kristensen and Gimsing 1988).

Epidemiologic investigations have shown a greater prevalence of hearing loss in the range of frequencies expected from noise-induced damage among farmers than in non-farming controls (Plakke and Dare 1992). Among farmers, 16.8 percent had hearing impairment sufficient to compromise speech communication (Crutchfield and Sparks 1991). Among New York dairy farmers, 37 percent had abnormal pure tone average thresholds (500 Hz, 1000 Hz, 2000 Hz, 3000 Hz), and 65 percent had abnormal perception of high frequencies. The left ear was significantly more likely to be affected than the right. A significant dose-response relationship was observed, with risk of hearing impairment increasing with years of work in farming (May, Marvel, Regan et al. 1990). Among central Wisconsin teenagers, the prevalence of early noise-induced hearing loss was greater among those working on farms (54 to 57 percent) than among those not working on farms (24 to 33 percent). The odds ratio for hearing loss, given farm work, was 2.25 to 2.46, after adjustment for other factors known to increase the risk of hearing loss. The incidence of farm noise-induced hearing loss nationally is not known, but based on the high prevalences reported, this appears to represent a major public health problem. Use of hearing protection is infrequently reported in the available surveys, but such use would reduce the risk of noise-induced hearing loss among those working on farms (Broste, Hansen, Strand, and Strueland 1989).

VIBRATION-RELATED CONDITIONS

Significant whole-body vibration occurs during the operation of agricultural vehicles. In a cross-sectional study of subjective back pain complaints, Boshuizen, Bongers, and Hulshof (1990) reported the prevalence of low back pain to be 10 percent greater among workers with a history of driving tractors. Frequent or long-lasting back pain risk was much greater among workers with a history of whole-body vibration (odds ratios greater than 4.0). Furthermore, duration of vibration dose (years driving a tractor) was positively correlated with prevalence of back pain. The odds of reported prolapsed disk was 6.8 times greater among workers exposed to whole-body vibration than among workers without such an exposure (Boshuizen, Bongers, and Hulshof 1990).

Although tractors and many hand-held agricultural tools produce significant vibrations, vibration injury among agricultural workers has not been studied extensively and the incidence in this group is unknown. Whole body vibration is transmitted through the feet in standing workers or through the buttocks of seated workers. Farm workers have exposure to whole-body vibration through the buttocks when seated on tractors. Estimates of weighted root mean square (RMS) acceleration (a measure of vibration) show farm tractor operators to be exposed to 0.4 to 1.25 m^2. Studies of whole-body vibration among agricultural workers suggest a higher incidence in this group of spinal pathology consistent with cumulative trauma or vibration injury, including degenerative changes, low back pain, and herniation of intervertebral disks. Peripheral nerve, peripheral vascular, gastrointestinal, and vestibular system injuries are more prevalent among tractor operators. Studies of workers in other occupational groups exposed to similar whole-body vibration have also shown increased risk for degenerative spine changes and intervertebral disk herniation (Crutchfield and Sparks 1991).

Farmers are also exposed to local vibration of the upper extremities when using hand-held agricultural tools. The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended threshold limit values for upper extremity exposure to vibration. Vibration injuries of the upper extremity include osteoarthritis of the wrist and elbow, bone cyst, peripheral demyelination, and arterial wall hypertrophy. Vibration-induced white finger is a vaso-occlusive response to vibration that may result in permanent disability. Forestry

workers with frequent exposure to chain saws are at great risk for vibration-induced white finger. This disorder has not been described among farmers to date (Crutchfield and Sparks 1991).

CHAPTER VIII. CONCLUSION

The agricultural workplace and worker are very productive, and productivity has improved dramatically in the last few decades, largely as a result of technological advancements that have facilitated crop planting, growth, harvesting, and production, and diminished crop loss due to infestation and spoilage. However, many of the technological advancements that account for the improved productivity of American agriculture present the farm worker with significant risks. The safety and health status of farm workers, as described in this paper, clearly reflects the burden of these risks. Farmers are more likely to suffer from accidental death, dismemberment, burns, poisoning, certain cancers, hearing loss, infection, back injury, and heat stress than workers engaged in other occupations. However, the use of available preventive technologies, and the development of others, holds great promise for markedly improving the safety and health of American farm workers.

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