CHAPTER 1. HEALTH EFFECTS OF WORKERS' HOME CONTAMINATION

CHAPTER SUMMARY

Reports of health effects among workers' family members from beryllium, asbestos, lead, caustic farm products, pesticides, chlorinated hydrocarbons, mercury, estrogenic substances, asthmatogens/allergens, arsenic, cadmium, fibrous glass, cyclotrimethylenetriamine, and infectious agents are reviewed in this chapter.

Beryllium was responsible for approximately 40 cases of chronic beryllium disease among workers' family members; the most recent case was reported in 1992 suggesting that cases may still be occurring.

Asbestos contamination of workers' homes has been a world-wide problem resulting in all forms of asbestos disease among workers' family members, including over 100 identified deaths from mesothelioma in the United States. Although many uses of asbestos have been abandoned and occupational exposures are regulated, potential exposures of workers' family members may still exist in the United States, especially in the construction industry.

Lead contamination of workers' homes resulting in elevated blood lead levels (BLLs) of workers' children and other household members is currently a substantial problem in the United States. Elevated BLLs have been correlated with hematologic abnormalities and abnormalities of neurologic and neurobehavioral testing, especially in children. Nearly 80 reported incidents of workers' family exposure to lead were identified in 22 published retrospective cohort studies, 14 published community studies, and 30 case series or case reports, of which 10 are unpublished reports or letters. Of the 34 reports on BLLs of workers' children, 19 have appeared since 1990. These included five reports that identified children with BLLs in excess of 40 μg/dL. The 19 reports suggest that workers' home contamination by lead is a current health problem in the United States.

Of the 80 reported incidents, about 8% (5 reports/6 cases) involved elevated BLLs in adult family members. In all six cases, BLLs exceeded 10 μg/dL. In two of these, BLLs exceeded 40 μg/dL, one of which reported a BLL greater that 50 μg/dL. These indicate a concern for the health of workers' adult family members, and for prenatal exposure.

Accidental ingestion and skin contact with caustic farm products have been responsible for over 40 cases of poisonings of farm children. Effects of ingestion of caustic farm products include chemical burns of the mouth and esophagus which can be fatal. Accidental body contact can cause chemical burns of the skin and eyes. Since most of these 40 cases have been reported in the last 5 years, this is a current problem.

Pesticide poisoning resulted in fatal cases and serious non-fatal cases in workers' children and adult contacts. Although most of the reports are dated 1980 or before, the three reports since 1990 indicate that pesticides continue to be of concern for families of applicators and farmworkers.

Chlorinated hydrocarbons resulted in five reports of health effects in family members of exposed workers. There were other cases in which chlorinated hydrocarbons in the urine or blood of family members were measured, but no adverse health effects were reported.

Mercury was responsible for six incidents of workers' homes being contaminated. The most severe cases of family poisonings occurred in cottage-industry type gold extraction operations, but family members of chlor-alkali plant workers and workers engaged in thermometer manufacturing were also exposed to mercury in recent years.

Exposure to estrogenic substances resulted in children of pharmaceutical and agricultural workers developing hyperestrogenic syndromes consisting of menstrual irregularities in women, breast development in men and boys, and premature onset of breast development and menstruation in girls.

Asthmatic and allergic reactions of family members were associated with animal allergens, platinum salts, mushrooms, grain dust, and Otto fuel.

Arsenic in mine and smelter dust brought home on a worker's clothing was considered to be one source of poisoning of his child which resulted in a liver angiosarcoma.

Cadmium contamination of lead-smelter workers' homes resulted in elevated concentrations of cadmium in the blood and hair of the workers' children.

Fibrous glass contaminated clothing has been shown to contaminate other clothing during laundry operations and to result in dermatitis of workers' family members.

Cyclotrimethylenetrinitramine (RDX) resulted in an episode of status epilepticus in a child due to ingesting RDX. The child's mother worked in an explosives manufacturing plant and transported clumps of RDX home on clothing and shoes.

Infectious agents as workers' home contaminants was verified by five reports of household members being infected with contagious diseases brought home on the worker. In these 5 reports, 35 household members were reported to have been infected with scabies, Q fever, mites, or giardiasis. It is believed that many additional cases exist that were either not reported in the literature or were reported in such a manner as to make them difficult to locate.

BERYLLIUM

Overview

The reports discussed in this section and summarized in Table 1 document approximately 40 cases (sometimes called household or contact cases) of chronic beryllium disease which occurred among family members of beryllium workers prior to 1967. The report of another case of chronic beryllium disease in 1992 [Newman and Kreiss 1992] indicates that cases may still be occurring among workers' family members but are not being diagnosed accurately.

Background

Beryllium, the second lightest metal, was discovered in 1798. Currently it has many uses. It is added to copper, glass, plastics, and ceramics to be used in connectors in electronic equipment, semiconductor packages, satellites, rockets, springs, gyroscopes, aircraft brakes and engines, submarine cable housings, dental prostheses, nuclear reactors, missile guidance systems, and military vehicle armor [Lang 1994]. It has also been used in rocket fuels, fluorescent lamps and neon signs, radio tubes, incandescent lamps and fluorescent powders, and cathode ray tubes.

Chronic beryllium disease is a potentially fatal granulomatous lung disorder characterized by a beryllium-specific cell-mediated immunity [Kreiss et al. 1989; Newman et al. 1989; Kreibel et al. 1988].

Only one-third of those dying from chronic beryllium disease were found to have mention of berylliosis on the death certificate [Lieben and Williams 1969]. Therefore complete case ascertainment for beryllium disease cannot be assured. Chest X-rays do not make the distinction between sarcoidosis and beryllium disease. This is of special concern for non-occupational cases, where a history of beryllium exposure may be difficult to obtain. In addition, chronic beryllium disease can be confused with sarcoidosis [Sprince et al. 1976]; therefore a misdiagnosis of sarcoidosis in a person with chronic beryllium disease is possible, as happened at first with the patient reported on by Newman and Kreiss [1992].

Several diagnostic testing methods are available. Lung biopsy specimens can be tested for beryllium. Bronchoalveolar lavage fluid can be tested for specific lymphoblastic response to beryllium salts. Kreiss et al. [1989] reported the use of a peripheral blood beryllium-reactive lymphocyte transformation test along with confirmation methods (more sensitive than those used in the past) such as bronchoalveolar lavage and transbronchial biopsy. Blood tests for beryllium sensitization may be positive when chest radiographs and pulmonary function tests are normal, thus presenting the possibility of an early clinical or subclinical case. The authors cautioned that issues relating to the use of the peripheral blood beryllium-reactive lymphocyte transformation test as a screening test still need to be worked out.

Review of Studies

Prior to World War II, there was little use of beryllium and few workers were exposed. During World War II, production of beryllium compounds from the ore increased and took place in two plants in Ohio and one in Pennsylvania. In this same period, beryllium was used in the production of fluorescent lights in a number of manufacturing plants in New England. In connection with these activities during the 1940's, disease from beryllium exposure first appeared in the United States [Van Ordstrand et al. 1943; Kress and Crispell 1944].

Subsequent to the report by Hardy [1948] of chronic beryllium disease in residents living near facilities manufacturing fluorescent lights, a number of reports identified neighborhood cases among residents living near the beryllium production facilities in Ohio [Eisenbud et al. 1949; DeNardi et al. 1949; Chesner 1950; Sterner and Eisenbud 1951] and Pennsylvania [Chamberlin et al. 1957]. Some of these reports [Hardy 1948; Eisenbud 1949; Chesner 1950; Chamberlin 1957] also identified beryllium workers' family members with beryllium disease ascribed to exposure to beryllium-contaminated clothing or other contaminated material. Additional reports continued to identify cases of beryllium disease in family members of workers which were ascribed to exposure to beryllium-contaminated clothing [Lieben and Metzner 1959; Tepper et al. 1961; Lieben and Williams 1969].

A registry of beryllium disease cases was initiated in 1951 [Hardy et al. 1967]. Additional reports on this registry have been published [Hardy 1965; Hasan and Kazemi 1974; Sprince and Kazemi 1980; Eisenbud and Lisson 1983]. In a review by Tepper et al. [1961], 32 cases of beryllium disease in beryllium workers' family members are cited. In 24 cases, the patients lived with workers who brought home beryllium-contaminated clothing and in 8 cases the patients had been exposed to beryllium plant discharges as well as contaminated clothing. Hardy et al. [1967] identified a total of 40 cases of chronic beryllium disease from the Beryllium Case Registry where the chief exposure was believed to be contaminated clothing.

The first case of beryllium disease in workers' family members was reported by Hardy [1948]. A woman developed and died of chronic beryllium disease after caring for her daughter, who also died of the disease. The daughter, who worked in a fluorescent lamp plant, would come home with beryllium powder on her clothes and shoes. Eisenbud et al. [1949] reported on a case of a worker's wife who developed beryllium disease after she routinely washed his beryllium-contaminated clothing.

Chesner [1950] discussed a 26-year-old woman whose neighbor brought sacks from the beryllium plant to her home. She used the sack material for dishcloths. She died after two years of progressive cough and weight loss. Chamberlin et al.

[1957] reported on five persons with beryllium disease who had exposure to clothing of beryllium extraction workers.

Other authors [DeNardi et al. 1949; Sussman et al. 1959; Eisenbud and Lisson 1983] discuss varying numbers of contact (household) cases. The cases discussed by these authors are likely included in the Beryllium Case Registry, but it is difficult to be certain of this because detailed descriptions are not always given.

According to the literature review, there were no more contact (household) cases added to the registry after Hardy et al. [1967] until the article by Newman and Kreiss [1992] who reported on a 56-year-old woman with chronic beryllium disease who had first been diagnosed with sarcoidosis. When it was determined that her husband was a beryllium worker, she was evaluated for beryllium disease. The clinical picture was compatible with beryllium disease and her blood test showed beryllium sensitization.

Because of the long period of time between the prior contact cases and this case, a review of her exposures to beryllium is useful. She was a non-smoker who had always lived in Ohio. She was self-employed and had sold cosmetics, done babysitting, brought up her children, and from 1973 until the time of the article, had done stockroom work for a retailer. Her husband had worked from 1959 to the current time at a beryllium production plant, with daily exposure to beryllium. When working directly with beryllium, he always changed clothing after work, showered before leaving for home, and did not bring his work clothing home. The family had always lived at least 28 miles from his work. She sought medical attention for this illness in November 1988. Her exposures consisted of the following:

- She took a tour of the plant in the 1960's;
- She took another tour in the 1970's at a time when it was not operating;
- During some months in 1976, her husband was an advisor to a new ceramics plant, where he did not do hands-on work and wore street clothes, which his wife cleaned on several occasions. Thus although beryllium was used at the plant, clothes worn to work were not left at work;
- A hydrogen furnace containing beryllium oxide exploded in her husband's face in February 1979. He was sent to the emergency room in his contaminated work clothes. When he was discharged from the emergency room, she was given the contaminated clothes which she put in a plastic bag at home before returning them to the plant guardhouse. Over the next several months, she scrubbed her husband's face several times daily with a motorized rotating brush to remove embedded metallic debris; and
- The husband injured his ankle while at work in September 1987. When she picked him up at the hospital, he was still wearing work clothes. He rode home in her car and she placed the dusty clothes in a plastic bag.

This case illustrates the need for vigilant application of industrial hygiene controls for beryllium even when exposures do not seem high or consistent. Moreover, it is possible that household and community cases of beryllium disease may still be occurring but are unrecognized or misdiagnosed as in this case.

ASBESTOS

Overview

Based on the studies reviewed in this section, families of asbestos-exposed workers have been at increased risk of pleural, pericardial, or peritoneal mesothelioma, lung cancer, cancer of the gastrointestinal tract, and non-malignant pleural and parenchymal abnormalities as well as asbestosis. Four cohort studies (Table 2), one community study (Table 3), seven case-control studies (Table 4), numerous case reports (Table 5) and case series (Table 6) provide evidence of these adverse effects in family members of asbestos workers.

The occupations associated with asbestos-related disease in family members are those where workers were exposed to asbestos dust during: construction and renovation; prospecting and mining; manufacturing textiles, tiles, boilers, and ovens; shipbuilding and associated trades; certain railroad shop trades; welding; insulation; use and manufacture of asbestos products such as cords, seals, and plates; and renovation and demolition projects within the construction industry.

Although many past uses of asbestos have been abandoned, and asbestos uses and occupational exposures are now subjected to regulation, potential exposures of family members in the United States may still exist, especially in the construction industry [Sullivan et al. 1995].

Background

Asbestos is a generic term for a number of silicate minerals with a fibrous crystalline structure. The asbestiform varieties of silicate minerals can be found in both the amphibole and serpentine mineral groups, in veins or small veinlets within rock containing or composed of the common (non-asbestiform) variety of the same mineral. The major asbestiform varieties of minerals used commercially are chrysotile, tremolite-actinolite asbestos, cummingtonite-grunerite asbestos, anthophyllite asbestos, and crocidolite. Asbestos is marketed by its mineral name (e.g., anthophyllite asbestos), its variety name (e.g., chrysotile, crocidolite), or its trade name (e.g., Amosite).

Mesothelioma is a tumor arising from the pleural, pericardial, and peritoneal membranes. When it occurs in asbestos workers' household contacts, it is a sentinel event for exposure to asbestos from home contamination [Gardner and Saracci 1989]. Lung cancer is a malignant tumor of the lung. Cancer of the gastrointestinal tract is a malignant tumor of any part of the gastrointestinal tract including the mouth, pharynx, esophagus, stomach, small intestine, pancreas, colon, rectum, and anus. Asbestosis is a fibrotic disease of the lungs caused by

asbestos fibers which results in reduced lung volumes and difficulty in breathing. Pleural and hyaline plaques are localized thickenings which may be evident on radiographs 20 or more years after exposure. Pleural and hyaline plaques generally occur without symptoms but do provide a clinical marker of asbestos exposure.

Mesothelioma has occurred following short term asbestos exposures of only a few weeks, and can result from very low levels of exposure. There may be a latency period of 40 years or longer between exposure and clinical disease. Symptoms include chest pain, shortness of breath, and weight loss. Analysis of tissue obtained by biopsy (or at autopsy) is required for a definitive diagnosis [Dement et al. 1986]. Treatment is ineffective, with rapid disease progression and death [Lilis 1986].

Lung cancer may be associated with a range of symptoms including cough, shortness of breath, bloody sputum, and weight loss. Definitive diagnosis is made by tissue biopsy. Metastasis is common, and may present as bone pain or fracture, seizure, or various other syndromes. Progression of lung cancer is generally rapid, and treatments (including surgery, chemotherapy, and radiation) are unlikely to result in long term survival [Hodous and Melius 1986]. Although increased risk of lung cancer among household contacts of asbestos workers has been observed, the high prevalence of cigarette smoking among lung cancer cases frequently makes it difficult to detect cases which may be caused by exposure to asbestos resulting from workers' inadvertent contamination of the home.

Review of Studies

Information on exposure of family members has been elicited by questioning patients or relatives about the practice of bringing work clothes home and laundering the asbestos contaminated clothing at home. Other identified sources of exposure of workers' family members to asbestos include taking contaminate items home from work and using asbestos in cottage industries [Magee et al. 1986; Bittersohl and Ose 1971]. Additional evidence that exposures occurred in the homes of asbestos workers is the finding of asbestos in lungs of asbestos workers' family members who had no known exposures, other than contact with an exposed worker [Whitwell et al. 1977; Ashcroft and Heppleston 1970; Huncharek et al. 1989; Gibbs et al. 1989, 1990; Giarelli et al. 1992].

Most cases of asbestos disease among workers' family members occurred in households where information indicated that asbestos-contaminated work clothing was brought into the home and women were exposed during home laundering of the contaminated work clothing [Ashcroft and Heppleston 1970; Dalquen et al. 1970; Edge and Choudhury 1978; Lander and Viskum 1985; Konetzke et al. 1990]. Children were exposed by playing in areas where asbestos-contaminated shoes and work clothes were located, or where products containing asbestos were used or stored. It is of interest to note that male children of asbestos workers appear to

be at increased risk when compared with female children [Anderson et al. 1979b; Kilburn et al. 1985, 1986; Grundy and Miller 1972].

Three review articles discuss the adverse effects in family members of asbestos workers and the bases for inferring that these adverse health effects result from transporting contaminated clothing and other articles into the home. Grandjean and Bach [1986] reviewed the literature on effects of asbestos exposure on workplace bystanders and family members and Rom and Lockey [1982] and Berry [1986] reviewed the association between asbestos exposure and mesothelioma.

Based on the health effects studies reviewed in this section, contamination of workers' homes by asbestos dust appears to be an international problem. Of the 50 reports summarized in Tables 2-6, 16 are from the United States, 10 from Great Britain, 9 from Italy, 7 from Scandinavia, 3 from Germany, 2 from Canada, and 1 each from Australia, France, and Czechoslovakia.

Cohort Studies. Investigators from Mount Sinai School of Medicine [Anderson 1983; Anderson et al. 1976, 1979a, 1979b; Joubert et al. 1991; Nicholson 1983; Nicholson et al. 1980] studied household contacts of 1,664 amosite asbestos workers who manufactured thermal insulation (Table 2). The prevalence of parenchymal and pleural abnormality 20 or more years after first household exposure was 48% among wives, 21% among daughters, 42% among sons, and 37% among siblings [Anderson 1979b].

The Mount Sinai investigators [Anderson 1983; Anderson et al. 1976, 1979a, 1979b; Joubert et al. 1991; Nicholson 1983; Nicholson et al. 1980] studied morbidity and mortality among a cohort of household contacts of amosite asbestos workers employed in a New Jersey asbestos insulation materials factory between 1941 and 1945. Occupational, residential, smoking, and medical histories were obtained from the exposed cohort. Radiographs were taken 20 or more years after first exposure. Results for radiographic analysis were compared with a control group of similar age and gender from the same urban community. A statistically significant increased frequency of asbestos-associated radiographic abnormalities was observed among household contacts of asbestos workers. The prevalence of radiographic abnormality associated with secondary exposure was 35% vs. 5% expected, based on the comparison population ($p<0.001$). The prevalence of abnormalities increased with duration since first exposure ($p<0.01$). Those with 10 or more years of household exposure had a prevalence of abnormal radiographs of 53%. Household contacts of former asbestos workers who entered the home only after cessation of employment also were at significantly increased risk of pleural abnormality (12% observed vs. 2% expected; $p<0.02$) [Anderson et al. 1979a].

The Mt. Sinai investigators also examined mesothelioma and lung cancer mortality for vital status follow-up through 1980. There were 3 mesothelioma

deaths among 663 observed deaths for this cohort. In evaluating the significance of the mesothelioma mortality observed among these household contacts of amosite asbestos factory employees, Nicholson [1983] estimated the expected number of mesothelioma deaths to be 0.04, assuming an ambient air concentration of 200 ng/m^3. The standardized mortality ratio (SMR) for lung cancer was 152 (25 observed vs. 16.4 expected); after 20 years latency, an SMR of 185 was observed [Anderson 1983]. Among females, those with 20 or more years latency had an SMR of 170 (8 observed vs. 4.7 expected). Among males with 20+ years latency, there were 12 lung cancer deaths observed vs. 6.1 expected (SMR = 197).

A retrospective cohort mortality study of 1,964 wives of asbestos cement workers in Italy was conducted by Magnani et al. [1993]. The wives had no history of occupational exposure. Cancer of the pleura was significantly elevated, with an SMR of 792.3 with a 95% confidence interval of 215.9-2,028.8. The women who died from respiratory disease had washed their husband's work clothes in the home for more than 10 years.

The prevalence of hyaline pleural calcification in the general population in one area of Czechoslovakia was compared with three groups exposed to asbestos by Navratil and Trippe [1972]. All three exposed groups had a statistically significant increased risk of pleural plaques ($p < 0.01$) compared with the general population group. Pleural calcification was found in 0.34% (28/8,127) of the general adult population who lived in the same district as the factory and who were more than 40 years old, compared with 5.3% (42/800) among 800 asbestos-exposed workers, 5.8% (9/155) among people living in the neighborhood of the asbestos factory, and 3.5% (4/114) for relatives of asbestos workers, who were more than 20 years old.

Community-Based Cohort Studies. Shipyard workers, most of whom had bystander (secondary) exposure to asbestos on the job, and their families were studied by Kilburn et al. [1985, 1986]. The prevalence of radiographic evidence of asbestosis was 11% among their wives, 8% among their sons, and 2% among their daughters (Table 3).

Case-Control Studies. Six of seven case-control studies (Table 4) documented cases of mesothelioma among household contacts of asbestos workers. Newhouse and Thompson [1965] found 9 (7 female; 2 male) family-member cases among 76 mesothelioma cases versus only 1 control who was an asbestos worker's family member. Most of the women with mesothelioma had laundered their husband's work clothes. Whitwell et al. [1977] found a case of mesothelioma in a man whose father brought home gas mask canisters for packing with asbestos; there were no cases of domestic exposure to asbestos in the 100 case controls.

A matched case-control study of histologically confirmed mesothelioma among New York State women was reported by Vianna and Polan [1978]. They reported a relative risk of 10 (95% CI=1.4-37.4) for domestic exposures, including hand-laundering of work clothes. Results remained significant after elimination of occupationally exposed women from the analysis ($p=0.02$).

Several analyses on a population-based series of North American autopsies were conducted by McDonald and co-workers [McDonald et al. 1970; McDonald and McDonald 1973, 1980]. They studied 557 pleural and peritoneal mesothelioma cases from the U.S. and Canada matched on hospital, gender, age, and year of death to controls with pulmonary metastases from non-pulmonary primary cancers. Occupational, residential, smoking, and non-occupational exposure histories were obtained from relatives. Women with mesothelioma were more likely to have laundered work clothes of household contacts ($p=0.08$).

Rubino et al. [1972] reported on 102 cases of mesothelioma that occurred from 1960 to 1970 in two clinical settings in Turin, Italy. Of these, the diagnosis was confirmed in 54 cases, and an occupational history was obtained for 50 of these. Fifty matched controls from the same institution were selected, matched on age and gender. Of mesotheliomas reported in this case series, 12% resulted from home contamination, including one man whose wife worked in the asbestos industry, and a woman whose brother worked in an asbestos cement factory.

A case-control study of British shipbuilders was reported by Ashcroft and Heppleston [1970]. Patients with mesothelioma (23) were matched on gender and age with 46 hospital controls who were free of malignant disease. Of the cases, 91% had a history of asbestos exposure, compared with 41% of the controls ($p<0.001$). This study was designed to demonstrate a link between asbestos exposure (primarily occupational) and mesothelioma. Pertinent to workers' home contamination, one patient with mesothelioma was the widow of an asbestos worker. She was exposed for 3 years to asbestos dust brought home on her husband's hair and shoes.

One case-control study reported no significant differences in mesothelioma cases and controls with respect to non-occupational exposure [McEwen et al. 1971].

Case Reports. In Table 5, 16 case reports describing 1 or more cases of an asbestos-related health effect in family members of asbestos workers are summarized [Rusby 1968; Teyssier and Lesobre 1968; Champion 1971; Lillington et al. 1974; Li et al. 1978; Epler et al. 1980; Risberg et al. 1980; Jorgensen 1981; Martensson et al. 1984b; Krousel et al. 1986; Magee et al. 1986; Huncharek et al. 1989; Li et al. 1989; Otte et al. 1990; Oern et al. 1991; Anonymous 1993b]. Of these reports, 13 are of mesothelioma, 2 of pleural plaques, and 1 of asbestosis. Nine of the studies report on effects in multiple family members.

These case reports provide information on exposure scenarios not usually elicited in other types of studies. Epler et al. [1980] reported on two brothers who developed pleural changes as young adults. As children, they played in a room that was used as an automobile muffler repair shop. Magee et al. [1986] reported on an a case of mesothelioma in a 41-year-old male with no occupational exposure. He was exposed as a child in Corsica to tremolite asbestos in a room in his home that was used as a local bar. The patrons of this bar were miners at the Canari asbestos mine and came into the bar in their dusty work clothes. As a child, this man had used asbestos ore from the mine to filter wine. Li et al. [1989] reported on a family of four in which the father worked in an asbestos products plant. The father brought home cotton cloth sacks in which molded asbestos insulation had been transported. The mother cut the sacks into diapers for her children. The mother and one daughter died of mesothelioma. The father died of asbestosis. A young uncle who lived in the home and worked briefly in an asbestos-exposed job also died of mesothelioma. Otte et al. [1990] studied a family who produced asbestos cement in their homes. The mother, father and one son died of mesothelioma.

Case-Series. Table 6 summarizes 22 papers describing studies of case series [Lieben and Pistawka 1967; Dalquen et al. 1970; Heller et al. 1970; Bittersohl and Ose 1971; Vianna et al. 1981; Grundy and Miller 1972; Greenberg and Davies 1974; Milne 1976; Edge and Choudhury 1978; Bianchi et al. 1982; Bianchi et al. 1987; Bianchi et al. 1990; Bianchi et al. 1991; Bianchi et al. 1993; Lander and Viskum 1985; Gibbs et al. 1989; Gibbs et al. 1990; Konetzke et al. 1990; Kiviluoto 1965; Martensson et al. 1984a; Sider et al. 1987; Giarelli et al. 1992].

These reports are on series of patients seen and/or autopsied at large hospitals or clinics in urban areas. The reports may include cases with occupational and community exposures in addition to cases due to workers' home contamination. Sixteen of the reports were of studies on mesothelioma and six were reports on asbestosis and pleural plaques.

Fiber type and size distribution in a series of mesothelioma cases occurring among family members of asbestos-exposed workers were reported by Gibbs et al. [1990]. These cases had various forms of asbestos fibers in their lungs reflecting the types of asbestos to which the working family member was exposed.

LEAD

Overview

Based on the reports reviewed in this section, children and adult family members of lead workers have been at risk of developing lead poisoning from contamination of their homes with lead. These reports also raise concern about the effect of prenatal exposure as a result of an adult family member being exposed to lead.

Nearly 80 reported incidents of workers' family exposure to lead were identified in 22 published retrospective cohort studies (Table 7), 14 published community studies (Table 8), and 30 case series or case reports, of which 10 are unpublished reports or letters (Table 9). Of the 34 reports of workers' children's blood lead levels (BLLs), 19, including 5 which identified children with BLLs in excess of 40 μg/dL, were reported since 1990, indicating that workers' home contamination by lead continues to be a current health problem in the United States.

In five reports (six cases) BLLs in adult family members of U.S. lead workers were reported. In all six cases, BLLs exceeded 10 μg/dL. In two of these, BLLs exceeded 40 μg/dL, one of which reported a BLL greater than 50 μg/dL. Thus, indicating a need for concern for the health of workers' adult family members and concern for prenatal exposure.

This series of reports may represent only a small portion of the documented cases of take-home lead exposure. Many of the case reports were solicited from State health departments as part of the NIOSH effort to summarize existing accounts of such exposures. Of the 28 case reports, 10 (36%) are unpublished.

Background

Lead is a bluish gray metal that has been used since ancient times because of its useful properties, such as low melting point, pliability, and resistance to corrosion. The ancient Romans and Greeks first discovered the toxic effects of lead. Lead is ubiquitous in U.S. urban environments due to the widespread use of lead compounds in industry, gasoline, and paints during the past century. Exposure to lead occurs via inhalation of dust and fume, and ingestion through contact with lead-contaminated hands, food, water, cigarettes, and clothing.

Lead poisoning of workers' family members has been known since Oliver [1914] reported cases in Great Britain of "double wrist drop" (peripheral neuropathy) in women who laundered the clothes of their husbands who were painters. Oliver also reported severe cases of lead poisoning (infant mortality, paralysis, blindness, and severe mental retardation) among children of home pottery makers in Hungary.

Lead deposited in the respiratory and digestive systems is released to the blood, which distributes the lead throughout the body. More than 90% of total body lead content is accumulated in the bones, where it is stored for decades. Lead from bones may be released into the body long after the initial external exposure. There are several biological indices of exposure to lead. Measurement of protoporphyrin (free or zinc protoporphyrin [ZPP]) concentration in red blood cells (erythrocytes) can be a good indicator of inhibition of heme synthesis by lead; however there are other causes (i.e., anemia) of elevated protoporphyrin levels. Lead concentrations in urine, skeletal bones, teeth and hair can be used as biological indicators of exposure to lead. Recent advances in the measurement of

bone lead levels will eventually provide a more accurate method for determining cumulative lead exposure and the total body burden of lead. However, the best available method for evaluation of the current biological exposure to lead is measurement of the BLL.

The toxic responses to lead at various BLLs differ somewhat for children and adults [ATSDR 1988; EPA 1986]. In children, BLLs greater than 80 μg/dL may result in coma, convulsions, profound irreversible mental retardation, seizures, and death. At BLLs greater than 40 μg/dL, the effects include reduced hemoglobin synthesis and peripheral nerve dysfunction. At BLLs greater than 25 μg/dL, lower IQS and slower reaction times occur; and BLLs as low as 10 μg/dL may result in deficits in neurobehavioral development and enzyme inhibition. Evidence also indicates that children exposed *in utero* are at increased risk for adverse neurobehavioral and growth effects if their mothers' BLL is as low as 8 μg/dL and the umbilical cord BLL at birth is greater than about 6 μg/dL. The Centers for Disease Control and Prevention (CDC) defines pediatric lead poisoning as a BLL of 10 μg/dL or higher [CDC 1991].

In adults, BLLs at or above 100-120 μg/dL may result in encephalopathic signs and symptoms and chronic nephropathy. At BLLs greater than 80 μg/dL, frank anemia may occur. At BLLs greater than 40 μg/dL, adults may experience interference with hemoglobin metabolism, peripheral nerve dysfunction, and male and female reproductive system effects, and at BLLs as low as 10 μg/dL, enzyme inhibition and elevated blood pressure may develop.

Review of Studies of Blood Lead Levels

Exposure of lead workers' families was identified in nearly thirty different industries/occupations. The industries in which exposure of family members has been reported most often include: lead smelting, battery manufacturing/recycling, radiator repair, electrical components manufacturing, pottery/ceramics, and stained glass making. Family members exposure to lead has rarely been reported in construction (two case reports, one published). In part, this may be due to the fact that prior to June of 1993, the construction industry was exempt from the Occupational Safety and Health Administration (OSHA) lead standard and was therefore relatively unregulated and understudied. A NIOSH study of lead exposure among families of construction workers in New Jersey is currently underway; preliminary findings suggest that children of construction workers are at increased risk of BLLs greater than 10 μg/dL [Whelan and Piacitelli 1995].

The cohort studies (Table 7) selected households on the basis of exposure (e.g., a lead worker lived in the household) and BLLs in the exposed group were compared to households where no one worked with lead. The community studies (Table 8) were designed to screen for elevated BLLs among residents near a lead industry, usually a lead smelter. Community studies were included in this review if investigators also compared BLLs in families of workers to those of other

community members. The group of case reports and case series (Table 9) are reports of take-home lead incidents or assembled series of exposed family members without a comparison group.

Of the cohort (Table 7) and community studies (Table 8), 11 were conducted in the United States, and 25 were conducted in other parts of the world including England, Italy, Mexico, Greece, and the Caribbean. All but four of the case series/case reports (Table 9) came from the U.S. and many of these were reported by State and Federal agencies as a result of the 1993 Federal Register notice soliciting information on incidents of take-home lead exposure for this review.

Cohort Studies. The cohort studies (Table 7) date back to the late 1970's, beginning with the widely-cited report by Baker et al. [1977] published in the *New England Journal of Medicine*. Over 40% of smelter workers' children had BLLs in excess 30 μg/dL. The work clothing was implicated as the vehicle of contamination. This was one of the first studies to note the differences in exposure by age; highest BLLs were found in children less than 6 years of age. The investigators used a comparison group matched on neighborhood and measured lead content in household paint. Both are ways to account for background sources of lead exposure in the child's environment.

In a study of lead storage battery workers, Morton et al. [1982] found statistically significant differences in BLLs between children of workers with good hygiene practices (e.g., showering and changing clothes before leaving work) and those with poor hygiene practices.

The most recent cohort investigation of secondary lead exposure was conducted as part of a NIOSH health hazard evaluation of a battery reclamation site in Alabama. The small take-home component of the investigation found that 12 of 16 (75%) workers' children had BLLs of 10 μg/dL or higher compared with 2 of 5 (40%) control children.

The cohort studies also raise the issue of home-operated shops and cottage industries where work is conducted in or adjacent to the home. These include "back-yard" radiator shops in Jamaica [Matte and Burr 1989], home-operated pottery factories in Barbados [Koplan et al. 1977], and ceramic tile shops in Italy [Abbritti et al. 1979; Abbritti et al. 1988]. Exposures in these settings pose a special problem since employees and families are often unaware of the hazards of working with lead. In the U.S. smaller businesses such as radiator shops are not likely to have the services of an industrial hygienist and therefore may be unaware of measures that could be used to prevent take-home contamination (e.g., protective clothing, showers) [Pedersen and Sieber 1988].

In general, cohort studies have found that lead workers' children have significantly higher BLLs than control children after controlling for relevant non-occupational factors, such as neighborhoods and income. The BLLs for exposed children across all studies ranged from 10.2 to 81 μg/dL, while the BLLs for control children ranged from 6.2 to 27 μg/dL.

Community Studies. The community studies (Table 8) addressed worker take-home exposure as part of community investigations. The majority of the studies were conducted in smelter communities. Of the 14 studies reviewed, all but 3 were conducted outside the U.S. Four studies were conducted in Germany. All but one of the studies reported an association between children's exposure to lead (as measured in blood, hair, or teeth) and parental occupation in a lead industry.

The community study that reported the highest BLLs was conducted in a large smelter community in Brazil [Carvalho et al. 1984]. Children (ages 1-9) of lead workers had a significantly higher mean BLL (67.5 μg/dL) than similarly aged children of non-lead workers from the same community (56.6 μg/dL). The most recent study in the U.S. was reported by Cook et al. [1993] who found that the mean BLL in children in a smelting/mining community was 10.1 μg/dL (range 0.5-30.1 μg/dL). In this study, children whose parents were miners had higher BLLs than children whose parents were not occupationally exposed to lead.

Case Reports and Case Series. The case report and case series collection of studies (Table 9), dating back to the first report in 1952, illustrates the breadth of industries in which take-home lead exposure has been documented. More unusual industries include a polyvinyl chloride (PVC) factory, cutlery tempering, plaque production, propane tank manufacturing, cable cutting and salvage, and trucking. The most striking case report was from North Carolina where battery factory workers were taking home discarded battery casings and burning them as fuel in their home [Dolcourt et al. 1981]. The highest BLLs among 22 family members were observed in a 3-year-old male and 3-year-old female (256 and 220 μg/dL).

Review of Studies of Other Health Effects

All 34 U.S. reports (Tables 7-9) with quantitative blood lead information reported children with BLLs at or over the CDC intervention level of 10 μg/dL. In 24 of the 34 reports, children with BLLs greater than 25 μg/dL were identified, including 15 reports of children with BLLs at or over 40 μg/dL, and 3 reports of children with BLLs greater than 80 μg/dL. Specific health effects or lead poisoning severe enough to require chelation therapy were found in 12 of the 34 reports. In two reports, health effects were not found. The remaining 20 reports did not address health effects.

Reports of cases requiring chelation therapy, and cases of encephalopathy and elevated erythrocyte protoporphyrin (EP) were associated with BLLs greater than 50 μg/dL. Health effects reported at BLLs greater than 30 μg/dL included elevated erythrocyte protoporphyrin, metaphyseal lead lines in the long bones, and one case of possible learning and behavioral problems. The two studies that found no effects on measures of hemoglobin synthesis had BLLs in the range 8-44 μg/dL in one case and an average of 10.2 μg/dL in the other case. In all of the 20 studies in which health effects were not addressed, BLLs exceeded 10 μg/dL; in 10 of them BLLs were greater than 25 μg/dL and in 4 of these the BLLs exceeded 40 μg/dL.

CAUSTIC FARM PRODUCTS

Overview

Over 40 cases of poisonings by caustic farm products are documented by the reports reviewed in this section and summarized in Table 10. Effects of caustic farm products include chemical burns of the mouth and esophagus, eyes, and skin which may be fatal. Most of these cases have been reported in the last 5 years.

Background

Caustic products used on farms include dehorning products, drain cleaners, disinfectants, and dairy pipe line cleaners. The caustic substances include sodium and potassium hydroxide and sulfuric and phosphoric acid.

Review of Studies

There are eight reports on ingestions of caustic alkali (sodium hydroxide) and acid products (sulfuric and phosphoric) on farms. Almost all of the cases involved children, although there was at least one case where an adult ingested liquid dairy pipe line cleaner when it was in a container other than the original (a glass tumbler) in his barn [Edmonson 1987]. Christesen [1994] compared aspects and prevention of caustic alkali ingestion by children in Denmark. He noted the incidence of children being poisoned by milk pail cleaners, while originally high, decreased over the period of his study, due to an educational campaign implemented by the popular press during the same time period.

Neidich [1993], noted 14 case reports over a 5-year period for 2 pediatric inpatient facilities in Sioux Falls, South Dakota. Seven children ingested solid caustics (three were calf dehorning products, two were used as disinfectants and cleaners, and two were being used as drain cleaners). All seven of the liquid caustics were being used as dairy pipe line cleaners. Six of the seven children ingested the product from containers other than the original. The non-original containers were smaller than the original containers and included empty cans and soft drink bottles. The caustic was transferred several times a day to these smaller and more convenient containers, from which the children ingested the product. By comparison, all seven solid caustic ingestions occurred directly from the original containers. All 14 cases were found to have mouth burns at initial

examination; 3 of the solid ingestions and 5 of the liquid caustic ingestions resulted in second degree esophageal injury.

A farm-injury study by the Marshfield Medical Research Center in Wisconsin identified nine cases of children being admitted/treated by one hospital for injuries associated with caustic farm products during February 1990 through October 1992. Four cases were from ingestion of caustic dairy pipe line cleaner by children, four were eye injuries from caustic cleaners, and one was a skin burn from liquid dairy pipe line cleaner [Young 1994].

Edmonson [1987] provided reports on 10 cases which occurred over a period of 10 years presenting to 4 Wisconsin hospitals. All 10 cases for farm children involved liquid dairy pipe line cleaner (sodium hydroxide or potassium hydroxide). The 10 cases were equally distributed among sexes (5/5), ages 1-3 years. The 10 cases occurred in the milkhouse or barn and parents were present in 6 of 9 cases. In 8 of the 10 cases the type of container from which the liquid caustic was ingested was a beverage container (soda bottles and glasses). Of the 10 cases, 2 resulted in esophageal stricture and perforation, some of the most serious complications of caustic ingestions.

Within a 6-month period (December 1990-May 1991) one hospital in rural mid-state Pennsylvania reported four incidents of dairy pipe line cleaner poisonings [Geisinger Medical Center 1991]. One incident resulted in the death of a 17-month-old child and esophageal stricture and perforation in a 2½-year-old child.

Four cases of caustic dairy pipe line cleaner ingestion by children were identified from the Milwaukee Childrens Hospital (Wisconsin) Trauma Registry from March 1993 until January 1995 [Pelegrin 1995].

Finally, agricultural trade magazines periodically report incidents which have involved dairy pipe line cleaner poisonings [Leach and Leach 1992; Jorgenson 1990].

PESTICIDES

Overview

Reports of several fatal cases of pesticide poisoning as well as several serious non-fatal cases in workers' children and a few cases of poisonings in adult contacts are reviewed in this section and summarized in Table 11.

Five reports of pesticide poisoning by workers' family members relate to contamination introduced into their houses from the workplace. Other reports are of incidents of poisoning resulting from farm children playing with improperly stored or discarded containers or equipment. Two other reports are of incidents resulting from workers transporting items from the workplace to the residential area. Although most of the reports are dated 1980 or before, the three reports

since 1990 indicate that pesticide exposure may continue to be a risk for families of applicators and farmworkers.

Background

Pesticides are substances intended for preventing, destroying, repelling or mitigating an insect, rodent, nematode, fungus, weed, or other life or for use as plant regulators, defoliants or desiccants [McConnell 1994]. About 1,200 pesticides are registered for use in the United States. Pesticides can be classified in various ways. When classified by use, the categories are:

- insecticides;
- herbicides;
- fungicides;
- fumigants and nematocides; and
- rodenticides.

Insecticides include: organophosphates and carbamates, both of which inhibit the enzyme acetylcholinesterase resulting in neurotoxicity; the naturally occurring pyrethrum which may cause allergic reactions; the synthetic pyrethroids which may cause digestive system symptoms, neurotoxicity, and death; and organochlorine compounds which can cause neurotoxicity, liver damage, and cancer.

Other pesticides include: herbicides, most of which have low acute toxicity but some of which are animal carcinogens; fungicides, which encompass a wide variety of chemical classes, generally have low acute toxicity but have a wide spectrum of effects such as enzyme inhibition and carcinogenicity; fumigants and nematocides, a chemically diverse group of substances with high vapor pressure, can effect the respiratory system and also act as systemic poisons; and rodenticides which destroy the blood-clotting mechanism.

Most of the reports reviewed in this section involve the acetylcholinesterase inhibiting insecticides, but reports on organochlorine insecticides and a fumigant are also included.

Review of Studies

A 1½-year-old girl was poisoned by demeton when her father, a crop sprayer came home with contaminated shoes [West 1959]. He cleaned the shoes with paper towels, placed the towels in a wastebasket and left the shoes in the bathroom. The child contacted either the towels or the shoes and became unconscious. After treatment for organophosphate poisoning she recovered.

Three reports in the literature describe how poor hygiene practices in a chemical plant that manufactured the pesticide kepone led to contamination of the homes of workers [Cannon et al. 1978; Taylor et al. 1978; Kelly 1977]. Of the family members that were examined, 94% had detectable levels of kepone in their blood,

compared to 19% of community residents. In addition, two wives of workers had signs of kepone poisoning, displaying the same type of tremors seen in many of the workers. Both wives reported that they washed their husbands' work clothing.

In 1992, the California Department of Health Services conducted a pilot study of pesticide contamination of farmworkers' and non-farmworkers' homes located within one-quarter of a mile of agricultural fields [Osorio 1994]. In total, 12 different pesticides were detected in house dust samples. Pesticides were detected in all five of the farm workers' homes and in three of six non-farmworkers' homes. Levels of diazinon, chlorpyrifos, and propoxur were higher in farmworkers' homes than in non-farmworkers' homes. Wipe samples were taken from the hands of 1 child aged 1-5 years in each of the 11 households. Measurable levels of diazinon and chlorpyrifos were found only on hands of farmworkers' children; three children had diazinon (52-220 ng/wipe) and two of these had chlorpyrifos (20-100 ng/wipe). Based on dust levels and hand contamination, children residing in the homes with the highest diazinon levels were at substantially increased risk for acetylcholinesterase inhibition.

Reports in the literature describe incidents in which children were poisoned from: residual pesticides left in discarded containers [Johnston 1953; MacMillan 1964; Eitzman and Wolfson 1967]; from improper storage [Johnston 1953; Simon 1963], and from pesticides held in improper containers such as soda bottles and tin cups [Eitzman and Wolfson 1967; McGee et al. 1952; Fowler 1994a]. The children were often poisoned after they played with items that were contaminated with pesticides, or ingested pesticides from containers used to store or mix pesticides. For example, the 4-year-old son of a farmer was admitted to the hospital in a moribund condition after his mother discovered that he had played with a bag of parathion insecticide stored in the barn [Simon 1963]. In another case, a brother and sister died after playing in a swing that they made from a burlap sack heavily contaminated with parathion [Eitzman and Wolfson 1967]. Other similar poisonings are described in Table 11.

Another source of poisonings of workers' families has been items taken home from work. McGee et al. [1952] reported several unrelated poisonings by toxaphene, including one in which strips of metal from flattened storage drums were used to cover the walls of a garden shed. The drums were contaminated with toxaphene and a 2-year-old boy died after playing nearby. Anderson et al. [1965] described a near-fatal incident of parathion poisoning in two boys 5 and 12 years old, who became ill after sleeping on flannelette sheets that had been brought home by the father of one of the boys. The father operated a salvage dealer business from his home. He bought damaged sheets from an insurance adjustor which were contaminated with parathion.

The Health Division of the Oregon Department of Human Resources submitted a case report of illness caused by chloropicrin [Barnett 1994]. When an employee

brought home a loaded company truck containing chloropicrin, about one gallon was spilled on his driveway, causing eye irritation, nausea, vomiting, and coughing among two adults and three children living next door.

CHLORINATED HYDROCARBONS

Overview

Seven reports reviewed in this section and summarized in Table 12 document five instances in which health effects occurred in family members of workers who were exposed to chlorinated hydrocarbons. Other reports describe cases in which chlorinated hydrocarbons were found in the urine or blood of family members, but no adverse health effects were reported.

Background

Among the chemicals discussed in this section are a number of substances that cause chloracne, including: polychlorinated biphenyls (PCBs); chlorinated naphthalenes; chlorinated tars including hexachlorobenzene; and the 2,3,7,8- and 2,3,6,7-tetrachlorodibenzodioxins (TCDD). Other chemicals discussed in this section are the potential carcinogens 4,4'-methylene-bis(2-chloroaniline) (MOCA), 3,3'-dichlorobenzidine, 2,4,5-trichlorophenol, and tetrachloroethylene. In addition to chloracne, the first group of compounds may cause numerous other health effects, including cancer. Because of their toxicity and environmental persistence the uses of all these compounds except tetrachloroethylene and 2,4,5-trichlorophenol have been greatly curtailed or banned. However, workers may still be exposed to PCBs remaining in electrical transformers and at hazardous waste sites, to the dioxins which may be contaminants in certain pesticides, and to MOCA in production of certain plastics.

Review of Studies

The earliest report found on home contamination by any substance, was by Lehmann [1905] in Germany on family members who developed chloracne when the father wore his work clothing at home. The clothing was contaminated with chlorinated tars which included hexachlorobenzene and pentachlorobenzoic acid.

The earliest report from the U.S. of exposure of family members to a chlorinated hydrocarbon in a worker's home was in 1936 [Fulton and Matthews 1936]. The wife, 11-month-old daughter, and 2½-year-old son of a worker in Pennsylvania who was exposed to hexachloronaphthalene and chlorodiphenyl (also known as polychlorinated biphenyl [PCB]) in an electrical insulation plant developed the same type of acne-like dermatitis (chloracne) seen in workers in the plant. The worker wore dirty work clothes home and played with his children before changing into clean clothes.

Polychlorinated biphenyls with a pattern resembling Aroclor 1254 (a mixture of PCBs containing 54% chlorine) were found in the blood of two railway maintenance workers who repaired transformers (77 and 101 ng/mL) [Fischbein

and Wolff 1987]. The PCB levels for the wives who laundered their husbands clothes were not elevated but their PCB pattern resembled the Aroclor 1254 pattern of their husbands, suggesting that the PCBs found in the women's blood were derived from contact with their husbands.

After PCBs were released into the municipal sewage treatment plant by an electrical manufacturing firm in Bloomington, Indiana, PCBs were found in the blood serum of sewage treatment workers (75.1 ppb), their family members (33.6 ppb), community residents (24.4 ppb) and people who applied sludge from the plant on their yards (17.4 ppb) [Baker et al. 1980]. Thus, the workers' family members had higher concentrations of PCBs in their blood serum than the other non-occupational groups.

Good and Pensky [1943] reported that 52 electricians exposed to Halowax in shipbuilding developed chloracne as did some of their wives. Halowax was a mixture of chlorinated naphthalenes and other chlorinated hydrocarbons. Other details about the wives were not reported.

After an explosion at a factory producing 2,4,5-trichlorophenol in Derbyshire, Britain, two pipefitters developed chloracne [Jensen et al. 1972a,b; May 1973]. The son of one of the pipefitters developed chloracne similar to that of his father. His father wore his dirty work clothes at home after working around equipment contaminated with dioxin which had formed at the time of the explosion. The wife of the other pipefitter also developed chloracne.

In Midland, Michigan, Townsend et al. [1982] and in New Zealand, Smith et al. [1982] measured the reproductive effects in wives of workers exposed to 2,4,5-trichlorophenol and 2,3,7,8-TCDD (dioxin) respectively. In both cases, no adverse reproductive effects were found.

A suspected bladder carcinogen, 4,4'-methylene-bis(2-chloroaniline) (MOCA), was found in excess of 12.0 ppb in urine of family members of workers from two different specialty plastics manufacturing plants [ATSDR 1989a,b; ATSDR 1990b; Hesse 1991]. Another potential human carcinogen, 3,3'-dichlorobenzidine was present at 0.006-0.281 ppm in urine of family members and employees of another chemical production facility in Michigan [ATSDR 1991b].

A 6-week old girl developed jaundice and hepatomegaly due to exposure to tetrachloroethylene (TCE) in breast milk [Bagnell and Ellenberger 1977]. TCE in the mother's blood was 0.3 mg/dL and in the breast milk it was 1.0 mg/dL. The mother frequently visited the father during lunch at the dry-cleaning establishment where he worked. One week after breast feeding was stopped, no TCE was present in the blood of the infant and liver function returned to normal.

MERCURY

Overview

As summarized in Table 13, there are six reports of workers' homes being contaminated by mercury. The most severe cases of family poisonings, requiring hospitalization, occurred in cottage industry-type gold extraction operations, but family members of chlor-alkali plant workers and workers engaged in thermometer manufacturing were also exposed to mercury.

Background

Acute exposure to mercury may produce gastrointestinal disturbances, pharyngitis, dysphagia, and shock. Chronic exposure results in central and peripheral nervous system and renal effects.

Mercury is the only metal that is liquid throughout usual temperature ranges. Mercury, found in all classes of rocks, can be recovered from ores by heating. Major uses of mercury are in chlor-alkali plants and in manufacture of electrical apparatus.

Mercury exposures occur among dentists, gold extractors, jewelers, laboratory workers, miners, and thermometer makers. Low levels of elemental mercury are difficult to measure in humans and the environment. This difficulty should be kept in mind when considering several of the reports reviewed in this section which found that levels of mercury in either air or urine of exposed family members were not elevated above background (control) levels.

Review of Studies

Occupational exposure to mercury in a thermometer-manufacturing plant, followed by home contamination, was described by Ehrenberg et al. [1986, 1991], Trost [1985], and Hudson et al. [1985, 1987]. Company records showed mercury vapor levels from 24-308 $\mu g/m^3$ (time-weighted averages). Mercury levels in the urine of the workers ranged from 1 to 345 $\mu g/g$ creatinine [Ehrenberg et al. 1986]. Hudson et al. [1985, 1987] investigated the exposure to mercury in children of the workers. These investigators reported that the median mercury concentrations in the homes was 0.25 $\mu g/m^3$ (range 0.02-10 $\mu g/m^3$) and that the levels of mercury in the urine of the children averaged 25 $\mu g/L$, some five times higher than that reported in controls. Mercury in the urine of one child was in excess of 50 $\mu g/L$ and for another child it was in excess of 100 $\mu g/L$. There was a significant correlation between the mercury levels in the urine of the workers' children and the mercury levels in the urine of the parents. Neurological studies of 23 workers' children compared to 32 control children found no significant effects of the exposures.

A recent report [ATSDR 1990a] summarizes the exposures of workers in a chlor-alkali chemical plant. The workers were exposed to high levels of mercury during a scheduled maintenance operation which involved removing old pipes and

fittings, some of which contained mercury, using oxyacetylene torches. The heated mercury volatilized and condensed on the ceiling, walls, and floor, as well as on the clothing of the workers. Although protective clothing was used, work gloves, clothes, and boots which were soaked with mercury were taken home, exposing family members. As a result of notification of EPA by a worker concerned about his family, EPA and ATSDR evaluated the extent of exposure to mercury. They found high levels of mercury in various areas of the workers' homes but they did not find elevated urine mercury levels among the family members.

Cases of mine workers' homes being contaminated have been reported [West and Lim 1968; Zalesak 1994]. However the reports do not address the impact of this contamination on the health of the family members.

Severe cases of acute mercury poisoning of family members exposed during home use of mercury to extract gold from soil have been reported. These cases are included because of their similarities to situations that may occur in cottage industries.

- A husband and wife were exposed to mercury when the husband attempted to extract gold from sand samples in the home [Haddad and Stenberg 1963]. The husband's symptoms included fever, chills, nausea, and bronchitis; his urine contained mercury at 540 μg/L. The wife, who was in an adjacent room during the extraction process, had mercury in urine at 80 μg/L.

- An amateur prospector was exposed to mercury when he heated gold sand with mercury in a clay dish over the kitchen stove [King 1954]. The prospector developed severe coughing, vomiting, and became cyanotic.

- A case of family mercury poisoning that occurred when the father attempted to extract gold from sand was reported by Hallee [1969]. Approximately 30 mL of mercury accidentally spilled into a red-hot pan on the stove. The father's urinary excretion of mercury ranged from 200 μg/24 hr. to 560 μg/24 hr. over four days following exposure. The symptoms of the children (who were asleep in another room) included frequent coughing, fever, and nausea; their urine excretion of mercury ranged from 33 μg/24 hr. to 94 μg/24 hr. on the day following exposure. The mother who was in an adjacent room was also symptomatic, but urine levels apparently were not checked.

- A woman was poisoned when she used mercury to extract gold ore in a cast-iron ladle over her kitchen stove [Hatch 1990]. The woman indicated that she had been told to perform the operation outside, but thought she would be safe having a window open and a house fan over the kitchen stove. After 3 weeks of chelation therapy, her blood mercury level was 193

mg/dL, suggestive of continued exposure. Concentrations of mercury in her home dissipated over time.

ESTROGENIC SUBSTANCES

Overview

Children of pharmaceutical and agricultural workers exposed to substances with estrogenic activity have developed hyperestrogenic syndromes as documented by studies reviewed in this section and summarized in Table 14.

Background

Estrogenic substances include: the steroid female sex hormones estradiol, estrone, and estriol; the synthetic estrogen diethylstilbestrol; and some naturally occurring compounds such as coumestrol and genestin found in certain plants such as clover, soybeans, and tulips; and zearalenone, a mycotoxin produced by numerous species of Fusarium; and zeranol, a hydrated form of zearalenone [NTP 1982].

Occupational exposure to estrogenic substances primarily occurs in the pharmaceutical industry, although, as discussed below, there are also exposures to agricultural workers. Embalmers may also be exposed [Finkelstein et al. 1988; Bhat et al. 1990]. If the exposures are not sufficiently controlled, workers and family members may develop hyperestrogenic syndromes.

Hyperestrogenic syndromes consist of menstrual irregularities in women, breast development in adult men and boys, and early onset of breast development and menstruation in young girls. There is also concern that exposure to estrogenic materials may be associated with breast cancer.

Review of Studies

There are seven references in Table 14 summarizing exposures of workers' family members to estrogenic substances. Two references [Katzenellenbogan 1950; Klorfin and Bartine 1956] describe an incident where five children of workers engaged in the manufacture of diethylstilbestrol in Israel developed hyperestrogenic syndromes. Three references [Budzynska and Robaczynski 1968; Pacynski et al. 1967, 1971] describe a similar incident in Poland of hyperestrogenic syndromes occurring among children of pharmaceutical workers exposed to diethylstilbestrol. In both incidents, the chemical was brought home on contaminated clothing. In Poland milk that had been contaminated at work was also taken home for consumption. The children in Poland improved with reduction of parental exposure or job change.

Aw et al. [1985] conducted a health hazard evaluation of a pharmaceutical manufacturer in Indiana where five children of workers exposed to zeranol developed enlarged breasts. Industrial hygiene recommendations were made to prevent further contamination of the workers' homes.

Bierbaum [1993] reported on the occurrence of hyperestrogenic syndromes in children of workers who repaired feedlots in Kansas. The diethylstilbestrol was added to feed used in the feedlots, a practice that was abandoned during the course of the investigation.

ASTHMATOGENS/ALLERGENS

There are three references dealing with six incidents of asthma (Table 14). They are case reports involving various occupations. Wilken-Jensen [1983] discussed two cases of asthma in children in Denmark. The children developed asthmatic symptoms whenever the fathers (one a veterinarian, the other a miller) returned from work. In another case from Wilkens-Jensen, a boy developed fever, dyspnea (difficulty breathing), and general malaise regularly when exposure to mushroom mycelium occurred on the farm where he lived. These symptoms required multiple admissions to a local hospital.

Venables and Newman-Taylor [1989] discussed two cases of spouses (in the United Kingdom) who exhibited symptoms of asthma whenever the other spouse returned from work. In one case, the wife was a laboratory animal handler and the husband developed asthma. The husband's symptoms were most severe when he had contact with his wife on her return from work. These symptoms resolved after his wife started wearing different clothes at home than at work and showering and washing her hair before leaving work. In the other case, the husband worked with precious metals (platinum salts) and the wife developed asthma. The wife's symptoms resolved after the husband's company started a policy that employees should shower and change clothes before leaving the workplace. In both cases, symptoms improved when better work practices were instituted.

The third reference is from the hearings on the Workers' Family Protection Act [U.S. Senate 1991a]. It consists of a physician's testimony on cases of asthma among the children of workers at a hazardous waste incinerator in North Carolina. The childrens' asthma improved when the fathers ceased working at the incinerator.

Occupational asthmatogens generally are characterized by one of the following groups:

- airborne organic dusts with high molecular weights, generally plant or animal proteins;
- low molecular weight reactive chemicals (e.g., diisocyanates, platinum salts);
- pharmacologic bronchostrictors; or
- non-sensitizing respiratory tract irritants.

Symptoms may include wheezing, chest tightness, cough and shortness of breath, or recurrent episodic attacks of cough, sputum production, and rhinitis.

Exposures to asthmatogens occur in a number of industries including but not limited to, agriculture; manufacture of wood products, food, and chemicals; automobile body shops; and laboratories where animals are kept.

ARSENIC

Two references are included in Table 14. Falk et al. [1981] reported a case of hepatic angiosarcoma in a child associated with arsenic contamination of parental clothing, the water supply, and the environment. The father worked in a copper mine and smelter area where his clothing became contaminated with dust containing arsenic. His daughter, who exhibited a striking degree of pica (craving for unnatural food such as dirt), ate dirt from the yard and licked dirt off of her father's shoes. Klemmer et al. [1975] studied arsenic levels in homes in Hawaii and found higher values in homes of employees of firms using arsenic for pesticides or wood preservation, compared to homes where the residents did other work not involving arsenic. This lends credence to the observation by Falk et al. [1981] that arsenic was brought to the home by the worker on clothing and inanimate objects.

Arsenic (As), discovered in 1250 A.D., is a semimetallic solid which rapidly oxidizes to arsenuous oxide (As_2O_3) with the odor of garlic. It is used in bronzing, pyrotechnics, for hardening and improving the sphericity of shot, and as a doping agent in solid-state devices such as transistors. Gallium arsenide is used as a laser material to convert electricity directly into coherent light. Arsenic compounds have also been used as pesticides. Arsenic exposure has been found near copper, lead, and zinc smelters [Falk 1981].

CADMIUM

Four studies reported home contamination with cadmium which originated from parental occupation in a lead smelter (Table 14). In three of these reports, investigators found a significant association between parental employment in the smelter and the concentration of cadmium in the blood (CdB) or in hair (CdH) of children. In one report no significant relationship was found between parental occupation in the smelter and CdB in children, but a significant relationship was reported between presence of smelter dross in the household and elevated CdB concentration in children. Although, they did not identify women who had lived in homes of workers exposed to cadmium, Lauwerys et al. [1980] found that elderly women who lived in a cadmium polluted area in Belgium had a higher cadmium body burden and a higher prevalence of signs of renal dysfunction than women from a control area.

Cadmium induces cancers in laboratory animals and is associated with lung and prostate cancer in humans. Other chronic effects of cadmium include renal

disease, impaired lung function, and interference with calcium metabolism. Cadmium has a very long half-life in the body (up to 30 years in muscle); consequently, toxic levels may eventually be attained from very low levels of exposures.

Cadmium (Cd) was discovered in 1817 as an impurity in zinc carbonate. Almost all cadmium is obtained as a by-product in the treatment of zinc, copper, and lead ores. It is a soft, bluish-white metal, similar in many respects to zinc. Cadmium is used extensively in electroplating, many types of solder, for Ni-Cd batteries, in plastics stablizers, and in paint pigments. Cadmium compounds are used in black and white television phosphors and in blue and green phosphors for color television tubes. The sulfide is used as a yellow pigment.

FIBROUS GLASS

Three case reports included in Table 14 [Abel 1966; Madoff 1962; Peachey 1967] describe a dermatitis caused by wearing clothes contaminated with fibrous glass. In these cases, the sources of the contamination were family or laundromat washing machines where fibrous glass curtains had been washed. NIOSH has been made aware (personal communication) of a current potential case in which the wife and child suffered dermatitis as a result of washing an insulation worker's clothes with the family laundry.

Fibrous glass is a synthetic vitreous fiber manufactured by the blowing, spinning, or drawing of molten materials comprising silica and selected inorganic oxides. It has many uses including household and aircraft insulation, filter media, production of certain types of face masks, and the manufacture of fiberglass boats.

OTHER SUBSTANCES

Another chemical substance found to have been studied as a take-home contaminant is tin [Rinehart and Yanagisawa 1993; Briss 1994]. Health effects of tin were not detected in these studies.

Woody et al. [1986] reported a case of a child who developed an episode of status epilepticus from eating cyclotrimethylenetrinitramine (RDX). The child's mother worked in an explosives manufacturing plant and transported RDX home as clumps on clothing and shoes.

INFECTIOUS AGENTS

Overview

Reviewed in this section are infectious agents that could be transmitted from the workplace to the home on the body or clothing of the worker. Infectious agents that normally do not cause life threatening disease in healthy individuals, such as the common cold, are not considered to be under the perview of the Worker's Family Protection Act. Infectious diseases "within" a worker that are not physically on the body or the clothes are not considered in this review. For

example, tuberculosis spread by an infectious emergency service worker to family members via aerosols would not be included nor is HIV infection that may be transmitted to a spouse during intercourse. However, it should be noted that any infectious disease contracted by a worker at the workplace will be brought home and can potentially infect members of his or her household.

Based on these criteria, diseases that appear to most likely be transmitted from the worksite to the home "on" workers or their clothes include parasitic (mites and lice), vector-borne (lyme disease), and air-borne diseases (Q fever) that may be transmitted via fomites (e.g., dust).

Five reports of household members being infected with contagious disease brought home on the worker were identified. In these 5 reports, 35 "household" members were reported to have been infected with scabies, Q fever, mites, or giardiasis. It is believed that many additional cases exist that were either not reported in the literature or were reported in a manner that made them difficult to identify in the literature.

Background

Microorganisms are ubiquitous in nature. In humans, they are found naturally in many locations of the body including the skin, hair, and even internally in several locations such as the GI tract. These normal microbial flora help protect the host from pathogens and do not constitute any problems for healthy individuals. In fact, only a few of the bacteria, viruses, fungi, mycoplasmas, chlamydiae, rickettsiae, or protozoa found in nature are capable of causing disease in humans. For those organisms that are effectively able to invade and cause disease, there are several ways that they may be transported from infected workers to other members of the household. These include [Benenson 1985]:

- The transmission of infectious materials may occur by direct contact between individuals through a receptive portal of entry by touching (e.g., scabies), biting (e.g., hepatitis B virus [HBV]), kissing (e.g., Epstein-Barr virus), or sexual intercourse (e.g., human immunodeficiency virus [HIV]). When individuals sneeze, cough, sing, or even talk they exhale a cloud of tiny droplets of saliva. Direct projection of this droplet spray (usually in close proximity to the source - 1 meter or less) onto the conjunctiva or mucous membranes of another individual can transfer disease (e.g., common cold). Some diseases can also be transmitted transplacentally from mother to child (e.g., rubella, HIV).

- Indirect transmission of infectious agents may occur by contact with intermediates such as contaminated inanimate materials (e.g., toys, clothing, eating utensils, bedding) as well as contaminated food, water, milk or biological products such as blood, tissues, or organs. Also, zoonotic diseases may be transmitted by contact with animals that serve as reservoirs for infectious agents such as rabies. In addition, arthropod vectors such as ticks may transfer

rickettsiae (e.g., Rocky Mountain spotted fever), bacteria (e.g., Lyme disease) or viruses (e.g., encephalitis) through bites.

Aerosols containing infectious agents may be generated when an individual coughs, sneezes, sings, or talks. Also, aerosols may be generated by other methods in normal work situations such as those found in slaughterhouses, rendering plants, or autopsy rooms as well as during accidents in microbiology laboratories. Droplet nuclei are aerosols that contain infectious particles that are made by the evaporation of fluid from the droplets formed during the production of aerosols. Unlike droplet spray that may remain airborne only for a few feet that are associated with direct transmission of disease, droplet nuclei may remain suspended in the air for long periods of time and are associated with respiratory diseases (e.g., tuberculosis, influenza, mumps). Some infectious diseases that are normally spread via aerosols may also be spread via fomites (e.g., in dust from contaminated clothing or bedding, combs, floors, soil, etc.) such as the microorganisms that cause Q fever, coccidioidomycosis, and anthrax. Droplet nuclei and dust particles in the 1-5 micrometer size range may remain suspended in the air for long periods and, unlike larger particles, may easily be drawn into and retained in the alveoli of the lungs bypassing many of the defense mechanisms of the respiratory system.

Infectious diseases that most likely meet the criteria of being transported to workers' homes "on workers," their clothing, or other materials brought from the workplace include those (1) that are spread through direct skin-to-skin contact or direct contact with contaminated clothing such as **parasites** (e.g., mites or lice), (2) via **arthropod vectors** such as ticks (e.g., Lyme disease) or (3) those that may be transmitted on dust particles that are inhaled (e.g., Q fever, anthrax and possibly fungal diseases). The possibility appears to exist for bloodborne diseases such as HIV or HBV to be transported home on a worker's clothing soiled with body fluids from an infected person. However, the transmission of a bloodborne pathogen on soiled linen is considered to be negligible [CDC 1987].

Infectious diseases that are spread by routes other than "on workers" were not intended to be covered by the legislation. Diseases such as tuberculosis, which is spread by breathing air contaminated with bacteria from infected individuals, is not included nor are HIV infections that may be transmitted to spouses during intercourse. However, it should be noted that virtually any infectious disease contracted by a worker at the workplace will be "brought home" and can potentially infect members of his or her household. For example, at a recent scientific symposium, a case of a correction officer with occupationally acquired multidrug resistant tuberculosis (MDR-TB) was discussed. At the time, the correction officer was sharing a hospital room with his 2-year-old son who had acquired the disease from his father [Boyles and Boggan 1994a].

Diseases that appear likely to be transmitted to the home "on the worker" include

[adapted from Benenson 1985]:

1. Parasitic Diseases
   a. Scabies is a parasitic disease of the skin caused by a mite (*Sarcoptes scabiei*) that causes severe itching and is highly contagious. It is normally spread via skin-to-skin contact but in some cases may be spread through contact with mite-infected undergarments or bedclothes.

   b. Roundworm infection (*Ascaris lumbricoides*) from contaminated soil may be brought into houses and automobiles on the shoes of workers. The infection may than be transmitted to members of the household in dusts or via ingestion. Infection is usually highest in children aged 3-8 years.

   c. Pinworm disease (*Enterobius vermicularis*) is an intestinal infection that is usually spread through direct contact from anus to mouth of infective eggs but may be spread via clothing or bedding.

2. Vector-borne Diseases
   a. Arthropod-borne diseases that occur in the United States include lyme disease, caused by a spirochete *Borrelia burgorferi*. Lyme disease was first recognized as a clinical disease in 1977 when a group of children in Lyme, Connecticut was infected. It is considered to be the most common vector-borne disease in the United States and is characterized by distinctive skin lesions, polyarthritis, and neurological and cardiac involvement.

   b. Additional vector-borne diseases that may be brought home by workers in the United States include rickettsial diseases where ticks are also the vector such as Rocky Mountain spotted fever. In addition, approximately 90 arthropod-borne viral diseases have been identified. These include Colorado tick fever and encephalitis viruses that are tick-borne. The mosquito is the vector for many arboviruses that infect humans; however, mosquitos are unlikely to be brought home on workers. Plague (*Yersinia pestis*) is a disease of domestic and wild rodents transmitted to humans by flea bite. Tularemia (*Francisella tularensis*) may be spread via ticks but also may be transmitted via inhalation of contaminated dust particles.

3. Air-borne Diseases
   a. Respiratory diseases that may be spread via the air should be considered when infectious diseases that may be taken home are considered. For example, rickettsiae are small (300-600nm) obligatory parasitic bacteria that are often transmitted to man through the bite of arthropod vectors such as ticks. However, the rickettsia that causes Q fever (*Coxiella burnetti*) is found in animals as well as ticks and may be transmitted to humans by inhalation of infected dust, indirectly via the drinking of infected milk, or by direct contact with animals, particularly cattle, sheep, and goats. It is an acute febrile

disease with pneumonitis occurring in many cases. The organisms are highly infectious and are often spread in dusts associated with parturition. Person-to-person transmission is uncommon, although the disease may be contracted by direct contact with the laundry of exposed workers.

b. There are several fungi that can be transmitted via the clothes of workers that are capable of causing disease. These include coccidiomycosis, a systemic fungal disease which begins as a respiratory infection that may become disseminated and cause death. The infectious agent (*Coccidioides immitis*) is common in the arid and semiarid areas of the United States and is commonly transmitted on dust particles when the dry soil is disturbed but may also be transmitted on sheep wool. Also, Aspergillosis (caused primarily by *Aspergillus fumigatus, A. niger,* and *A. flavus)* is a fungal disease that may be transmitted on workers. Several clinical conditions can be produced by these fungi including the formation of masses of hyphae within ectatic bronchi and pneumonic and disseminated infection. The organisms are often found in compost piles undergoing decay and fermentation, hay that has been stored damp, in decaying vegetation, and in cereal grains. Although not an infectious disease, *Aspergillus* species as well as many other fungi may cause allergic reactions such as asthma in sensitive individuals.

The occupation or job elements of workers should also be considered when "take-home infectious diseases" are considered. For example, in occupations such as farming the worksite and home are often located virtually together and infectious agents that are at the worksite may easily be transported directly or indirectly (e.g., via vectors) into the home and infect household members.

Based on the potential proximity to large reservoirs (e.g., grain storage, compost piles) of fungus on farms, there is perhaps a greater potential for fungal exposures in farm households. A study in Finland of airborne fungal spore concentrations in farm houses during the winter months indicated that some fungal genera not normally found in the urban environment (e.g., *Alternaria, Botrytis*) were found in the farmhouses as well as the cow barns.[Pasanen et al. 1989]. The results of the study indicated that airborne fungal spores may be carried from the cow barn into the farmers' homes.

Other diseases that may be directly associated with specific occupations include animal diseases such as brucellosis and anthrax. Brucellosis is primarily an occupational disease of farm workers, slaughterhouse workers, veterinarians, and meat plant workers who are exposed to infected animals or tissues. Approximately 150-250 cases per year are reported in the United States [Benenson 1985]. Transmission is primarily by direct contact with infected animals (e.g., cattle and swine) but the bacteria can survive in dust and airborne transmission is possible [Anonymous 1978]. Anthrax (*Bacillus anthracis*) is an acute bacterial disease that usually initially affects the skin but may occasionally

involve the mediastinum or intestinal tract. It rarely occurs in developed nations. It is primarily a disease of workers who process hides and veterinarians who come in contact with infected animals. It may remain viable as a spore in soil associated with infected animals for years. Inhalation anthrax may result if the spores are inhaled while intestinal anthrax may arise if the spores are ingested [Benenson 1985].

Review of Studies

In this section, a number of examples from the literature that are indicative of the circumstances where infectious agents have been transmitted to the homes of workers are discussed.

- An HIV infected 28-year-old male with a disseminated *Mycobacterium avium* infection was admitted to an Italian hospital in 1991. He was also diagnosed as being infected with the mite *Sarcoptes scabiei*. The hospital staff were aware of this infection and used protective clothing, gloves, and booties. However, within one month, 29 staff members were infected with the mite. Six relatives of the staff were infected at home [Scalzini et al. 1992].

- In 1991, an immunocompromised patient (non-HIV related) was admitted to a hospital in Kansas. He was later found to be infected with scabies. Subsequently, 49 hospital staff members were infected with scabies including: those with frequent direct care responsibilities such as nurses and respiratory therapists; ancillary staff including those from social services and housekeeping; and 14 family members of the staff. [Clark et al. 1992]

- In 1984 an outbreak of Q fever in Idaho was associated with a sheep research station. Of the 18 cases of Q fever 2 were family members of workers employed at the station. One was a 14-month-old child while the second was the wife of a worker. It is assumed that these family members were infected with Q fever rickettsiae contained on dust brought home on the clothes of the workers. It is also worth noting that a farmer who had no direct contact with the research station also contracted Q fever. It is thought that he was infected from a Q fever infected guard dog he had received from the research station [Rauch et al. 1987].

- A case was reported in England where 10 people became ill with Q fever who were performers in an Easter play at their village church. The source of the infection was a shepherd who came to rehearsals in his work clothes. *C. burnetti* was subsequently isolated from dust collected from shepherds' clothing, demonstrating contaminated clothing to be a potential source for exposure of family members on farms [Marmon and Stoker 1956].

- Giardiasis is a protozoan (*Giardia lamblia*) infection that primarily attacks the small intestine and is associated with symptoms that include diarrhea, cramps,

and bloating. It is most often contracted from fecally contaminated water or food but may be transmitted person-to-person. In 1979, the Minnesota Department of Health conducted an evaluation of an outbreak of giardiasis at a rural public school system. Of the 60 employees of the school system, 19 met the case definition for giardiasis. Three members of the employees' households also had persistent diarrhea consistent with giardiasis infection [Osterholm et al. 1981].

RADIOACTIVE SUBSTANCES

Several incidents of home contamination are discussed in Chapter 7 "Responses to Incidents of Workers' Home Contamination." However, since no adverse health effects of these incidents were reported they are not discussed in this Chapter.