Report to Congress
on
Workers’ Home Contamination Study
Conducted Under
The Workers’ Family Protection Act
(29 U.S.C. 671a)

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
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FOREWORD

In 1992, the U.S. Congress passed the Workers’ Family Protection Act (Public Law 102-522, 29 U.S.C. 671), which requested that the CDC’s National Institute for Occupational Safety and Health (NIOSH) conduct a study to “evaluate the potential for, prevalence of, and issues related to the contamination of workers’ homes with hazardous chemicals and substances...transported from the workplaces of such workers.” With this request, Congress identified a compelling public health issue, bridging health concerns in the workplace and the home. NIOSH found that contamination of workers’ homes is a worldwide problem, with incidents reported from 28 countries and from 36 States in the United States. Such incidents have resulted in a wide range of diseases and, in some cases, death among workers’ families.

This report represents an important step in addressing the concerns outlined in the Act. It puts us on the road to preventing the exposure of families to potentially harmful substances unknowingly brought home from the job. It also serves as a reminder of the importance of occupational safety and health research to CDC’s overall mission of promoting health and quality of life by preventing and controlling disease, injury, and disability.

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PREFACE

The legislative directive (Public Law 102-522, Section 209, the Workers' Family Protection Act, [29 U.S.C. § 671a]) to conduct this study of contamination of workers' homes by substances carried home on workers' clothing or bodies was enacted on October 26, 1992. However, this is not a new problem. Holt [1923] cited two early studies of lead-workers' families that were published in 1860 and 1896. Oliver [1914] reported on lead poisoning in wives of house painters who washed their husbands' overalls, observations that resulted in a series of laws in Great Britain to protect the workers' families from lead poisoning. Lead poisoning continues to be a problem; this report cites about 65 incidents of lead poisoning among workers' families. Of these, 35 are from the United States, 24 of which were reported in the last 10 years.

Lehmann [1905] reported that the mother and child of a worker exposed to chlorinated hydrocarbons developed chloracne (a condition similar to acne caused by certain chlorinated chemicals) ascribed to the worker's contaminated clothing. Lehmann also wrote of a laundress who developed chloracne as a result of washing the contaminated clothing of workers. Thirty years after Lehmann's report was published in Germany, a similar case was reported by Fulton and Matthews [1936] from the Pennsylvania Department of Labor and Industry. In this case a child's father who was exposed to hexachloronaphthalene and chlorodiphenyl wore his soiled clothing home from work. Additional cases of workers' homes being contaminated with chlorinated hydrocarbons have been reported in the last 10 years.

Prior to 1960, beryllium, toxaphene, mercury vapors, and diethylstilbestrol were also identified as hazards to the families of workers. In the last 10 years, 10 additional chemical substances have been identified in incidents of workers' home contamination, as well as allergens, radioactive materials, and infectious agents.

This report to Congress and the Workers' Family Protection Task Force summarizes the incidents of home contamination this study has discovered, including the health consequences, the sources, and the levels of contamination. The report contains information on the effectiveness of preventive measures and of decontamination procedures that have been used or studied. The report summarizes the relevant laws and regulations and responses of Federal and State agencies and industry to incidents of workers' home contamination.

The report should be useful not only to Congress and the Workers' Family Protection Task Force in deciding future actions, but also to all who have responsibilities and concern for protecting workers and their families from preventable illnesses.

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EXECUTIVE SUMMARY

The Workers’ Family Protection Act of 1992 (Public Law 102-522, 29 U.S.C. § 671a) directed the National Institute for Occupational Safety and Health (NIOSH) to conduct a study of contamination of workers’ homes with hazardous chemicals and substances (including infectious agents) transported from the workplace. NIOSH found that contamination of workers’ homes is a worldwide problem; incidents have been reported from 28 countries and from 36 States in the United States. Such incidents have resulted in a wide range of health effects and death among workers’ families exposed to toxic substances and infectious agents. About half of the reports of health effects have appeared in the last 10 years, revealing new sources of contamination.

In completing the study, NIOSH solicited information from Federal and State health, labor, and environmental agencies, groups with special circumstances such as firefighters, and the public. NIOSH then reviewed and compiled the information received along with information in published reports on contamination of workers’ homes by substances brought home from the workplace. The report includes a survey of reported health effects, information on sources and levels of contamination, preventive measures, decontamination procedures, a review of Federal and State laws, and responses of agencies and industry to incidents involving contamination of workers’ homes. This report is being considered by the Workers’ Family Protection Task Force, which is charged under the Workers’ Family Protection Act with evaluating the need for additional research.

Health Effects of Workers’ Home Contamination

Workers can inadvertently carry hazardous materials home from work on their clothes, skin, hair, tools, and in their vehicles. As a result, families of these workers have been exposed to hazardous substances and have developed various health effects. Health effects have also occurred when the home and the workplace are not distinct—such as on farms or in homes that involve cottage industries. For some contaminants, there are other potential sources of home contamination such as air and water pollution and deteriorating lead paint in the home. Only a few of the studies found in the literature used epidemiologic methods to estimate the relative risks of health effects from the contaminant transported home by the worker independent of health risks due to other sources of the contaminant in the home.

Little is known of the full range of health effects or the extent to which they occur as a result of workers’ home contamination. There are no information systems to enable tracking of illnesses and health conditions resulting from these circumstances. Many of the health effects among workers’ family members described below were recognized because of their uniqueness their clear relationship to workplace contaminants, or their serious nature.
• **Chronic beryllium disease**
  This potentially fatal lung disease has occurred in families of workers exposed to beryllium in the nuclear and aviation industries and workplaces involved in the production of beryllium and fluorescent lights and gyroscopes.

• **Asbestosis and mesothelioma**
  Fatal lung diseases have occurred among family members of workers engaged in the manufacture of many products containing asbestos, including thermal insulation materials, asbestos cement, automobile mufflers, shingles, textiles, gas masks, floor tiles, boilers, ovens, and brakeshoses and other friction products for automobiles. Families have also been exposed to asbestos when workers were engaged in mining, shipbuilding, insulating (e.g., pipe laggers and railway workers), maintenance and repair of boilers and vehicles, and asbestos removal operations.

• **Lead poisoning, neurological effects, and mental retardation**
  These health effects have occurred in children of workers engaged in mining, smelting, construction, manufacturing (pottery, ceramics, stained glass, ceramic tiles, electrical components, bullets, and lead batteries), repair and reclamation of lead batteries, repair of radiators, recovery of gold and silver, work on firing ranges, and welding, painting, and splicing of cables.

• **Deaths and neurological effects from pesticides**
  Farm families and families of other workers exposed to pesticides have suffered these serious effects.

• **Chemical burns from caustic substances**
  Chemical burns of the mouth and esophagus and fatalities from ingesting caustic substances have occurred in farm families when hazardous substances were improperly used and stored on farms.

• **Chloracne and other effects from chlorinated hydrocarbons**
  Family members have been exposed when these substances were transported home on clothing of workers manufacturing or using these compounds in the production of insulated wire, plastic products, ion exchange resins, and textiles. Family members have been similarly exposed when workers’ clothes became contaminated during marine electrical work, transformer maintenance, municipal sewage treatment, rail transportation, wood treatment, and application of herbicides.

• **Neurological effects from mercury**
  Family members have developed various neurological effects as a result of being exposed to mercury carried home on clothing of workers engaged in mining, thermometer manufacture, and cottage-industry gold extraction.
• Abnormal development from estrogenic substances
  Enlarged breasts have occurred in boys and girls and premature menstruation has
  occurred in girls from estrogenic substances brought home on contaminated clothing
  of pharmaceutical and farm workers.

• Asthmatic and allergic reactions from dusts
  Farm families and others have suffered asthmatic and other allergic effects from
  animal allergens, mushrooms, grain dust, and platinum salts.

• Liver angiosarcoma from arsenic
  Families of workers engaged in mining, smelting, and wood treatment have been
  exposed to arsenic from contaminated skin and clothing; one child developed liver
  angiosarcoma.

• Dermatitis from fibrous glass
  Family members have developed dermatitis when their clothing was contaminated
  with fibrous glass during laundering of insulation workers' clothing.

• Status epilepticus from chemical exposure
  A child experienced epileptic seizures following ingestion of an explosive compound
  brought home on the clothing of a worker engaged in the manufacture of explosives.

• Diseases from infectious agents
  Family members have contracted infectious diseases such as scabies and Q fever from
  agents brought home on contaminated clothing and skin of workers engaged in
  agriculture, hospital, and laboratory work. As intended by Congress, infectious agents
  are included as hazardous substances to the extent that pathogens can be transported
  on a worker's person or clothing.

Measures for Preventing Home Contamination

Preventive measures that were found to be effective when used in the workplace include:

• Reducing exposures in the workplace;
• Changing clothes before going home and leaving the soiled clothing at work to be
  laundered by the employer;
• Storing street clothes in separate areas of the workplace to prevent their
  contamination;
• Showering before leaving work; and
• Prohibiting removal of toxic substances or contaminated items from the workplace.

Preventive measures that have been used successfully at home include:

• Separating work areas of cottage industries from living areas;
• Properly storing and disposing of toxic substances on farms and in cottage industries;
• Preventing family members from visiting the workplace;
• Laundering contaminated clothing separately from family laundry when it is necessary to launder contaminated clothing at home; and
• Informing workers of the risk to family members and of preventive measures.

Other preventive measures that need to be used include:

• Educating physicians and other health professionals to inquire about potential work-related causes of disease;
• Developing surveillance programs to track health effects that could be related to home contamination; and
• Educating children, parents, and teachers about the effects of toxic substances.

Procedures for Decontaminating Homes and Clothing

Decontamination procedures include air showers, laundering, airing, vacuuming and other methods of surface cleaning, and destruction and disposal of contaminated items. These procedures appear to have widely varying effectiveness, depending on the specific methods employed, the contaminants, and the surfaces. In general, hard surfaces can be far more easily decontaminated than clothes, carpets, and soft furniture. In most cases effective decontamination requires relatively intensive methods. Normal house cleaning and laundry practices appear to be inadequate for decontaminating workers’ clothes and homes. Lead, asbestos, pesticides, and beryllium contamination can be especially persistent. In some instances even intensive decontamination procedures may be ineffective.

Another serious concern is that decontamination methods can increase the hazard to the person performing the operation and to others in the household. Home laundering of contaminated clothing exposes the lauderer. Vacuuming of floors contaminated with mercury can substantially increase air concentrations, and vacuuming of carpets contaminated with lead can increase lead concentrations on the carpet surface.

The difficulty of decontaminating work clothing, the prominence of clothing as a source of home contamination, and the potential exposure of the lauderer are problems that can be avoided through the use of disposable work clothing. The use, availability, and cost of this alternative need to be assessed.

Federal and State Laws

Seven statutes provide Federal agencies with some mechanisms for responding to or preventing workers’ home contamination. Twenty rules or standards in the Code of Federal Regulations (CFR) address workers’ home contamination or have elements that serve to protect workers’ families.
Under the Occupational Safety and Health Act of 1970 (Public Law 91-596), NIOSH research assessing the health of workers has also addressed the exposure of their families to workplace contaminants, resulting in recommendations to prevent home contamination. The Occupational Safety and Health Administration (OSHA) regulations and actions intended to protect workers also help assure that families are protected. In addition, OSHA can promulgate standards to protect workers' family members when workers are required to live in housing provided by the employer as a condition of employment. Under the Federal Mine Safety and Health Act of 1977 (Public Law 95-164), the Mine Safety and Health Administration (MSHA) has limited regulatory authority to address issues of workers' home contamination.

The U.S. Environmental Protection Agency (EPA) has broad authority under the Toxic Substances Control Act (Public Law 94-469) to regulate chemicals and to obtain information about the adverse effects of chemicals. In addition, EPA has specific authority and responsibility regarding the use of asbestos and lead. Under the Federal Insecticide, Fungicide, and Rodenticide Act (Public Law 92-516), EPA also regulates the use and disposal of pesticides (which also helps to protect workers' families). EPA and the Agency for Toxic Substances and Disease Registry (ATSDR) are authorized under the Superfund Amendments and Reauthorization Act of 1986 (Public Law 99-499) to address hazardous waste and releases of hazardous substances that may relate to identifying contamination of workers' homes and assuring decontamination.

Thirty States and Puerto Rico responded to the requests from NIOSH for information about State laws. Most indicated that there were no laws specific to workers' home contamination or protection of workers' family members. Some States identified laws requiring the reporting of cases of elevated blood lead levels and pesticide poisonings to a State agency; other States identified laws related to work at hazardous waste sites and emergency responses to releases of hazardous substances. An examination of occupational safety and health regulations of States with OSHA-approved occupational safety and health programs revealed none more stringent than Federal OSHA regulations - with respect to the protection of workers' families. However, extension of occupational safety and health regulations to State and local government employees in these States also helps protect the families of public employees in these States.

Responses to Incidents of Workers' Home Contamination

Several Federal agencies have responded to incidents of workers' home contamination, often working together with State or local government agencies. These responses have resulted in identification of workers' home contamination, decontamination of workers' homes, and recommendations for instituting workplace changes that would prevent further contamination. NIOSH has conducted approximately 40 health hazard evaluations that address potential home contamination. In several cases, Federal agencies have referred incidents to State or local health departments for follow-up actions.
State agencies have investigated incidents of workers' home contamination, made referrals to Federal agencies for follow-up actions, and recommended workplace improvements to prevent further contamination of workers' homes.

Responses to incidents of workers' home contamination include educational materials such as those of the Lead Industries Association, Inc. on preventing workers' home contamination as well as responses of various employers to specific incidents of home contamination.

Limitations of the Report

The health information available for the report, which includes incidents of illness and home contamination obtained from public agencies and published literature, does not provide a basis for estimating the prevalence of this public health problem.

The Workers' Family Protection Act requires NIOSH to evaluate relevant information about indoor air quality as it relates to workers' home contamination and to study the special circumstances of firefighters as they relate to contamination of their homes.

- The only report found on indoor air quality applicable to workers' family protection involved tetrachloroethylene exposures in living quarters located in the same building as dry-cleaning establishments. Indoor air quality studies would be useful to protect family members in cottage industries.

- Incidents of contamination of firefighters' homes were not identified. However, NIOSH has conducted several studies of contamination and decontamination of protective clothing used by firefighters. These studies are reviewed in this report and NIOSH will continue to pursue the issues related to potential contamination of firefighters' homes.

Other limitations of the report include:

- Little research has documented the frequency and distribution of health effects among the families of workers in various industries and occupations. NIOSH is undertaking one study addressing lead exposure among families of bridge repair workers.

- Lead and pesticides are the only contaminants for which monitoring or reporting programs help to identify and prevent cases of poisoning from workers' home contamination.

- Despite various case reports, the prevalence of health effects from workers' home contamination is not known because there are no surveillance systems in place for tracking or monitoring such health conditions.

- Many diseases have long latency periods between exposure and manifestation of the disease, making identification and intervention difficult.
• The workplace origin of many common diseases that occur in workers’ families (such as asthma, dermatitis, and infectious diseases) is probably unrecognized because physicians and other health professionals fail to inquire about the occupation of family members and to consider whether these diseases are work-related.

• The literature reviewed in this report contained only nominal information about contamination levels in workers’ homes. Most measurements were of surface dust, for which there are no guidelines for acceptable levels of contamination.

Recommendations for Research and Education

• The prevalence of health effects of contaminants transported from the workplace should be determined. One possible approach would be to conduct surveys among occupational and environmental medicine health care providers and clinics.

• The employment practices and controls that work best in preventing the transport of contaminants from the workplace to the home should be identified.

• Educational programs to prevent home contamination should be developed for employers, workers, children, teachers, and parents, physicians, and other health professionals.

• The special needs and problems of individuals who work in home or cottage industries need to be identified.

Conclusions

• Workers’ home contamination may pose a serious public health problem. Health effects and deaths from contaminants brought home from the workplace have been reported in 28 countries and 36 States.

• The extent to which these health effects occur is not known because there are no information systems to track them, and physicians do not always recognize the occupational contribution to various common diseases.

• About half of the reports of health effects from home contamination are less than 10 years old. The literature on the health effects involved approximately 30 different substances or agents. The potential exists for many of the thousands of other chemicals used in commerce to be transported to workers’ homes or to be used in home-centered businesses.

• Health effects and deaths from contaminants brought home from the workplace are preventable using known effective measures. Educational programs are needed to promote their use.
• Normal house cleaning and laundry practices are often inadequate for decontaminating workers' homes and clothing and can increase the hazard to the person performing the tasks and others in the household.

• Only two Federal laws have elements that directly address workers' home contamination. However, other laws provide agencies with certain mechanisms for responding to, or preventing workers' home contamination. Operating under existing laws OSHA, MSHA, DOE, ATSDR, EPA, and CDC, including NIOSH and the National Center for Environmental Health have responded to incidents of workers' home contamination, made recommendations to prevent such incidents, and conducted relevant research.
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INTRODUCTION

Because of repeated reports of contamination of workers' homes in their States, identical bills [WFPA 1991a], S. 353 [WFPA 1991b] and H.R. 845 [WFPA 1991c], were introduced in 1991 by Mr. Jeffords (Vermont) and Mr. Ballenger (North Carolina) in the U.S. Senate and the U.S. House of Representatives, respectively. The Senate Subcommittee on Labor of the Committee on Labor and Human Resources held a hearing on S. 353 on July 26, 1991 [U.S. Senate 1991a]. Following the hearing, the Committee on Labor and Human Resources revised S. 353 and issued a report on November 27, recommending the revised bill to the Senate [U.S. Senate 1991b]. The revised bill was incorporated into the Fire Administration Authorization Act of 1992 (Public Law 102-522) as Section 209 of that law, which was enacted on October 26, 1992. Section 209, the Workers' Family Protection Act, appears in the United States Code at 29 U.S.C.1 § 671a (Appendix 1).

The Workers' Family Protection Act requires the National Institute for Occupational Safety and Health (NIOSH) to conduct a study on workers' home contamination in cooperation with the Secretary of Labor, the Administrator of the Environmental Protection Agency, the Administrator of the Agency for Toxic Substances and Disease Registry and other appropriate Federal Government agencies. The purpose of the study is to evaluate contamination of workers' homes with hazardous chemicals and substances, including infectious agents, transported from the workplaces. The study is to consist of: (1) a review of past incidents of home contamination reported in the literature and in the records of NIOSH, the Occupational Safety and Health Administration (OSHA), the States, and other governmental agencies, including the Department of Energy (DOE) and the Environmental Protection Agency (EPA); and (2) an evaluation of current statutory, regulatory, and voluntary industrial hygiene or other measures used by small, medium, and large employers to prevent or remediate home contamination.

The Act directs NIOSH to report existing research and case histories conducted on incidents of employee transported contaminant releases, including:

- The health effects, if any, of the resulting exposure on workers and their families;
- Methods for differentiating exposure health effects and relative risks associated with specific agents from other sources of exposure inside and outside the home;
- The effectiveness of workplace housekeeping practices and personal protective equipment in preventing home contamination;
- The effectiveness of normal house cleaning and laundry procedures for decontaminating workers' homes and personal clothing; and
- Indoor air quality, as the research concerning such pertains to the fate of chemicals transported from a workplace into the home environment.

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1United States Code.
In conducting the study and preparing the report, NIOSH has taken a broad approach to the problem of workers’ home contamination in order to ensure that relevant information is included. Some reports that may relate to hobbies were included because the distinction between hobby and "cottage industry" is not always clear and the situations may be similar. Reports where family members were exposed by visiting the workplace were included, as were reports where living quarters adjacent to workplaces were contaminated. Studies of contamination of homes from other sources were included if they provided relevant information about levels of contamination, methods of measurement, or decontamination. As intended by Congress, infectious agents are included as hazardous substances to the extent that pathogens can be transported on a worker’s person or clothing. Congress did not intend for the Workers’ Family Protection Act to apply to the spread of infectious diseases by other means.

In July 1993, a working group was formed with representatives from each NIOSH Division to plan and implement a strategy to conduct this study. Specific task areas were assigned to members of this working group. Several Federal agencies including the Agency for Toxic Substances and Disease Registry (ATSDR), the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), the Department of Energy (DOE), the Mine Safety and Health Administration (MSHA), and the Centers for Disease Control and Prevention (CDC) provided assistance in conducting the study.

The NIOSH working group obtained information for this report through a variety of routes. On November 15, 1993, a notice entitled "National Institute for Occupational Safety and Health: Request for Existing Information Relevant to Implementing the Workers' Family Protection Act" was published in the Federal Register (Appendix 2). The notice requested information on several topics including measurements of home contamination, reports on government actions occurring as a result of home contamination incidents, preventive measures used by employers, and effectiveness of industrial hygiene practices. This notice was announced in CDC’s Morbidity and Mortality Weekly Report (MMWR) on December 10, 1993 (Appendix 2), by electronic mail to State agencies involved in NIOSH occupational health programs and cooperative agreements and to county agriculture extension agents. A request for information was also distributed to Poison Control Centers.

In January 1994, NIOSH sent over 1,100 letters to associations and State and Federal agencies and programs requesting information relevant to this study. The Federal Register Notice was enclosed with these letters. The mailing lists used and copies of written responses are available from the NIOSH Docket Office.

Over 50 written and several telephone responses were received. Working group members followed up on several Federal, State, and local agency responses. All State-plan occupational safety and health offices were contacted by telephone to obtain a copy of relevant State laws on occupational safety and health.
Several previous review articles provided an entry to the world literature [Bellin 1981; Chisolm 1978; Lehmann 1977; McDiarmid and Weaver 1993].

Key-word literature searches were conducted in various databases, including TOXLINE and NIOSHTIC. Articles and reports identified in these searches were obtained and reviewed for relevance. In most cases, cited references from these reports and articles were retrieved and reviewed as well.

The report is arranged to address the issues identified in the Act. In Chapter 1, the studies relating to health effects are reviewed. Details of the studies for each contaminant are presented in Tables 1-14 and overviews of the findings for each contaminant are presented in the text. In Chapter 2, the sources of contamination are discussed by contaminant, where information was available. Chapters 3, 4, and 5 present discussions of the studies for each contaminant on: levels of contamination; preventive measures; and procedures for decontamination, respectively. Table 15 presents the details on industrial hygiene studies cited in Chapters 2-5 by contaminant, incorporating the process, the industrial hygiene methodology, observations, and comments or recommendations. Studies on laundry procedures for pesticides which are discussed in Chapter 5 are summarized in Table 16.

In Chapter 6, Federal and State laws that are operative are discussed. The Federal statutes are summarized in Table 17 and rules of various Federal agencies found in the Code of Federal Regulations are tabulated and explained in Table 18. In Chapter 7, the responses of Federal and State agencies and industry to incidents of home contamination are reviewed; these are summarized in Tables 19-23.
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, asthmagons/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/m³. 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.

9
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; Teysier 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.


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 [Dolcort 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 μg/m³ (time-weighted averages). Mercury levels in the urine of the workers ranged from 1 to 345 μg/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 μg/m³ (range 0.02-10 μg/m³) and that the levels of mercury in the urine of the children averaged 25 μg/L, some five times higher than that reported in controls. Mercury in the urine of one child was in excess of 50 μg/L and for another child it was in excess of 100 μg/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 children's asthma improved when the fathers ceased working at the incinerator.

Occupational asthmato gens 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 (As₂O₃) 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 stabilizers, 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 cyclotrimethylenetriminitramine (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 then 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 coccidioidomycosis, 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.
CHAPTER 2. SOURCES OF WORKERS' HOME CONTAMINATION

CHAPTER SUMMARY
Sources of contamination of workers homes and poisonings of workers' family members reviewed in this chapter include: work clothing; the worker's body; tools and equipment; taking items home from work (such as scrap material); cottage industries (where work is done in or adjacent to the home); farms; and visiting a family member's workplace.

Clothing contamination was documented in 18 reports: 1 on beryllium; 7 on lead; 7 on pesticides; and 1 each on PCB's, an estrogenic substance (zeranol), and 3,4-benzo(a)pyrene. For lead, measurements of both clothing and home contamination were included in some studies. However, these were inadequate for establishing a quantitative relationship between the two. Other evidence of clothing as a source of home contamination includes: high levels of contamination in areas of homes where soiled clothing is stored and laundered (lead, mercury); contamination of washing machines (mercury) or dryers (3,3'-dichlorbenzidine, MOCA); and poisoning of launderers (beryllium, asbestos, lead, kepone).

The workers' body has been considered as a source of home contamination, and showering before leaving work has often been recommended as a preventive measure. However, reports have only documented contamination of workers' hands.

Hand tools and other equipment have been found to contribute to home and vehicle contamination by mercury and pesticides. The potential for contamination of homes by tools was also demonstrated for PCB's and radioactive substances.

Items taken home from work (beryllium-ore bags, cotton shipping-bags for asbestos, cloths from discarded filters, metal drums, contaminated milk, and radioactive scrap lumber) have resulted in serious, and sometimes fatal, poisonings of workers' family members.

Cottage industries, where work is undertaken in the same building or on the property where the family resides have been recognized as a hazard to family members since at least 1914. Cottage industries are the subject of 22 reports of home contamination or family poisonings from asbestos, lead, parathion, and mercury which are reviewed in this chapter. The levels of contamination were often extremely high and the poisonings were severe.

Farms are similar to cottage industries in that families live on the property where work is performed. Three types of products used on farms: pesticides; caustic substances; and estrogenic substances have resulted in several cases of poisoning of family members.
Visiting the workplace of a family member has been shown to be a hazard for families of dry cleaners and veterinarians.

**CONTAMINATED CLOTHING**

**Overview**
This section reviews reports that provide evidence for clothing worn, or otherwise taken home from work, as a source of home contamination. The reports are summarized in Table 15. In the health effects studies reviewed in Chapter 1, home contamination and family exposures were often attributed to contaminated clothing brought home from the workplace. This attribution was based on: information elicited by questioning household members; descriptions of workplaces and work practices; and the practice of wearing and laundering work clothes at home. Clothing contamination was documented in 18 reports reviewed below: 1 on beryllium; 7 on lead; 7 on pesticides; 1 on chlorinated hydrocarbons (PCBs); 1 on an estrogenic substance (zeranol); and 1 on 3,4-benzo(a)pyrene. Only for lead were measurements of both clothing contamination and home contamination included in the same studies; these few studies are inadequate for establishing any quantitative relationship between clothing contamination and home contamination.

Other evidence of clothing as a source of home contamination includes: the findings discussed below of high levels of contamination in laundry areas of workers' homes and in areas where contaminated clothing is stored (lead, mercury); contamination of washing machines (mercury) or dryers (3, 3'-dichlorobenzidine, MOCA); and poisoning of home launderers (beryllium, asbestos, lead, kepone). Estimates of exposure levels that could have occurred during home laundering of beryllium and asbestos suggest that such exposures could have exceeded OSHA occupational exposure limits for these substances.

**Beryllium**
There were no reports of measurements of home contamination by beryllium, although the case histories and epidemiology studies generally assumed that cases of berylliosis in workers' family members were due to laundering contaminated clothing. In support of this assumption, the following studies on clothing contamination indicate that substantial amounts of beryllium dust could have been brought into the workers' homes by contaminated clothing.

Fabrics experimentally exposed at a beryllium production worksite contained beryllium up to 2.8 mg/m² [Bohne and Cohen 1985]. In a subsequent study Cohen and Positano [1986], found that work shirts contained from 12 to 37 mg/m² of beryllium. It is likely that inhalation exposures of workers' family members occurred during laundering of the contaminated clothing, since resuspended beryllium dust concentrations in air from unwashed shirts at up to 0.64 µg/m³ were found. In an earlier laboratory study, Eisenbud et al. [1949] found beryllium concentrations in air at 125-1,200 µg/m³ when soiled clothes
were shaken and estimated an inhalation dose of 17 μg during a single home laundry. The OSHA permissible exposure limit (PEL) for beryllium is 2 μg/m³ as an 8-hr. time-weighted average (TWA) with permissible excursions up to 25 μg/m³ for up to 30 minutes (29 CFR² 1910.1000).

Asbestos
Several studies of asbestos workers' families inferred that asbestos-related diseases were due to home contamination emanating from clothes contaminated at work, especially due to laundering the clothes [Anderson et al. 1979a,b; Bianchi et al. 1987; Giarelli et al. 1992; Gibbs et al. 1990; Huncharek et al. 1989]. However, no studies evaluated the relationships between home contamination by asbestos, contamination of clothing brought home from work, and exposures during home laundering. The few studies reported and reviewed in this section indicate that clothing probably was a source of home contamination by asbestos and support the hypothesis that home laundering of asbestos contaminated clothing could be especially hazardous.

One study reported measurements of asbestos contamination in workers' homes; however no measurements of clothing as a source of the contamination were made [Nicholson et al. 1980].

Two studies of workplace clothing contamination by asbestos have been reported [Seixas and Ordin 1986; Driscoll and Elliott 1990]. Chrysotile asbestos was found in all clothing vacuumed as employees left work at a brake shoe manufacturing facility, but neither report provided quantitative data on asbestos recovered from the workers' clothing.

No studies of exposure during home laundering were found. However, a study on laundering clothing contaminated by an asbestos removal operation produced an average of 0.4 fibers/cm³ while picking up clothing and loading the washer. A maximum of 1.2 fibers/cm³ was found during the total laundry operation [Sawyer 1977]. Although the study was not conducted in a home laundry and measurements of the level of clothing contamination that generated these concentrations were not made, the study is consistent with the hypothesis that home laundering of asbestos-contaminated clothing is hazardous. Another important aspect of laundering asbestos contaminated clothing is that the fibers can transfer to uncontaminated clothing washed with the contaminated clothing, as was found by NIOSH [1971] in a study of dry cleaning a coat made with 8% asbestos fiber.

Lead

Overview. The 18 studies reviewed in this section provide both direct and indirect evidence that lead-contaminated clothing is a source of home contamination.

Lead contamination for both clothing and homes was included in three reports reviewed in this section: one on ore smelter workers; one on a lead products trucker; and one on a radiator repairman. In addition there are two reports on electric cable splicers, one of which reported on clothing contamination and the other of which reported on home contamination. The studies on smelter workers and electric cable splicers, which are the only ones that had comparable data, indicate that contamination of both the smelter workers' homes and clothing was much greater than that of the cable splicers. However the studies are inadequate for establishing a quantitative relationship between levels of contamination in clothes and in homes.

Other evidence presented in this section that clothing is a source of lead contamination in homes includes: (1) additional measurements of clothing contamination; (2) the findings of the highest lead loadings in areas of homes where contaminated clothing was stored and laundered; (3) elevated BLLs in children of parents who wore their contaminated clothing home; and (4) elevated BLLs in home launderers.

Reports of clothing and home contamination. Measurements of lead contamination include: concentrations in collected dust, expressed as weight of lead/weight of total dust (e.g., μg/g or ppm); or lead loading which is the weight of lead within a square area of surface (e.g., μg/cm²).

Homes of secondary lead smelter workers were found by Winegar et al. [1977] to contain lead in house dust at 120-26,000 ppm. In this study, lead concentration in dust of pants cuffs worn under coveralls of two workers were 60,000 and 600,000 ppm and the lead loading of the trouser bottoms of six workers was 280-7,600 μg/cm². There was no correlation between measurements of lead in house dust and the lead loading of trouser bottoms of the six workers. Only 1 of 33 workers showered at work, 8 took work clothes home for cleaning and 21 took home street clothes that were worn under coveralls. There was also no correlation between house dust levels and the type of clothing brought home.

Pollock [1994] found lead at 240 μg/ft² (2.2 μg/cm²) on the shoes of a worker who was engaged in trucking lead and lead products; lead up to 0.2 μg/cm² was found on surfaces of the worker's home.

Lead carried home from a radiator repair shop was reported by Pitts [1986] and Garrettson [1988]. Wipe samples were collected at various locations in the
home and automobile and from the workers’ shoes; data were reported in 
μg/filter but the areas sampled were not reported. Lead in house dust was 183-
284 μg/filter in the bathroom closet where dirty linen was kept and 284 
μg/filter in the kitchen near the washing machine. Other areas of the home 
were less contaminated. Higher levels were found in the worker’s car (1,295 
μg/filter on the driver’s seat and 7,580 μg/filter on the floor of the car). The 
highest level was found on the worker’s shoes (11,030 μg/filter). Lead in house 
paint and in soil outside the house were eliminated as sources of home 
contamination.

In a study of electric cable splicers, lead concentrations in house dust in the 
homes were measured by Rinehart and Yanagiswa [1993]. As with the study of 
battery factory workers’ homes [CDC 1977b], the highest concentrations of lead 
in dust were found in the laundry areas (621-1,606 ppm), but measurements of 
lead in clothing were not reported. However, in an earlier study of these cable 
splicers [Venable et al. 1993], their clothing was found to contain lead at 600-
4,800 μg/ft² (5.7-45 μg/cm²), and their cars contained up to 12,400 μg/ft² (17 
μg/cm²). Most workers took their soiled clothing home and many of them 
washed their work clothes with other laundry. Taken together, these two 
reports, [Venable et al. 1993; Rinehart and Yanagiswa 1993] provide evidence 
that the electric cable splicers’ clothes were contaminated, resulting in 
subsequent contamination of their homes.

Supporting studies. Other studies that provide information on either lead 
contamination of homes or clothing support the inference of contaminated 
clothing as a source of lead in homes.

Lead concentrations in dust from seven battery factory workers’ homes were 
studied and found to be highest (average 31,840 μg/g, maximum 84,000 μg/g) 
in closets where the work clothes were stored [CDC 1977b; Dolcourt et al. 
1978]. The average concentration of lead in dust of cars that were driven to 
work was 2,770 μg/g. Measurements of the lead-contaminated clothing were 
not made, but paint, water supply and air were ruled out as sources of lead.

Lead at 1,700 ppm was found in the dust of the home of a worker engaged in 
cutting down old cables [Osorio 1994]. He wore his work clothing home and 
laundered it with the family laundry. In a report on workers who soldered or 
welded with lead, wipe samples were taken from two workers’ shoes and the 
floor under the gas pedal of a car. Shoes had lead at 4-20 μg/cm² and the floor 
had 4 μg/cm² [CDC 1992a].

Kaye et al. [1987] measured lead up to 3,400 ppm in vacuum cleaner dust of 
homes of workers making ceramic-coated capacitors and resistors. 
Measurements of clothing contamination were not made; however, lead 
concentrations in the workplace were 50-1,700 μg/m² (OSHA PEL 50 μg/m²)
the workers wore no protective clothing and there were no shower facilities at work, suggesting that contaminated clothing could have been a source of home contamination.

Lead loadings were determined at various sites in the homes of workers exposed to lead at a bridge site in Ohio [Piacitelli and Whelan 1995]. Measurements of clothing contamination were not reported, except for the highest lead loading of clothing (2,278 µg/m²), and of the sofa or chair (639 µg/m²) used by the worker. Paint in the home was excluded as a potential source of the lead contamination.

Cook et al. [1993] found lead in floor dust up to 11,000 ppm, and in window sill dust up to 28,000 ppm in a study of 105 homes in Leadville, Colorado. An unspecified number of workers were engaged in lead mining and smelting; children of those who wore their work clothing home had elevated BLL’s. Czachur et al. [1995] found that elevated BLLs of children of workers in a variety of industries were related to the practice of washing dirty work clothing at home. Similarly, Morton et al. [1982] found a significant correlation between the practice of battery workers bringing dirty clothing home and BLLs of their children. Piclette et al. [1989] found that BLLs were elevated in family members who laundered the clothing of battery recycling workers.

Contamination of clothing by lead was documented without any information on home contamination for: (1) workers engaged in abrasive blasting of lead-based paint on a bridge by Ewers et al. [1994a, 1995] who found lead levels as high as 300 µg/cm² on work shirts; (2) secondary lead smelter workers by Grandjean and Bach [1986] who found lead up to 2 g/pair of socks; and (3) workers engaged in lining tanks with lead plates (up to 20 µg/cm² on shoes and up to 2 µg/cm² on shirt collars) [McCammon et al. 1991].

Baker et al. [1977] assumed that contaminated clothing was the source of lead contamination (up to 89,000 ppm) in the homes of the secondary lead smelter workers they studied. This assumption is supported by clothing contamination and house dust studies of smelter workers by Winegar et al. [1977], Grandjean and Bach [1986], and Cook et al. [1993].

Pesticides
A single report of measurements of contamination of workers' homes by pesticides was found [Osorio 1994]. Although this study demonstrated much higher levels of diazinon, chlorpyrifos and propoxur in house dust from floors of farm workers' homes than from floors of non-farm workers' homes, no information on clothing as a source of the contamination was included in the report.
Several reports on contamination of clothing were found; however no associated measurements of home contamination were found. Finley and Rogilico [1969] found up to 12 ppm methyl parathion and up to 136 ppm of dichlorodiphenyl-trichloroethane (DDT) in cloth worn by workers for 8 hrs. in a cotton field the day after spraying. Clothing worn for 30 minutes while working in a freshly sprayed cotton field was analyzed for methyl and ethyl parathion by Ware et al. [1973]. Blue jeans contained 6-16 mg of methyl parathion and about 8 mg of ethyl parathion, whereas T-shirts contained less than 1 mg of each of these contaminants. Finley et al [1977] found methyl parathion at up to 32 ppm in samples of cloth worn during 6 hours of work in a freshly sprayed cotton field, and Graves et al. [1980] found permethrin at 25.8 ppm in a similar study of cotton field workers.

A study of corn-field sprayers’ clothing contamination by a water dispersible granule of the herbicide atrazine after 4 hours of work in the field was reported by Oakland et al. [1992]. Atrazine at up to 7 µg/cm² of fabric was found.

A pair of coveralls that had been worn during pesticide applications for 4 seasons and washed after each use were analyzed for residual pesticides by Stone and Stahr [1989]. Trelfan® was found in samples of the coveralls at up to 43 ng/cm², Lorsban® at up to 92 ng/cm², and Counter at up to 15 ng/cm², demonstrating the persistence of these substances in contaminated clothing.

Clothing contaminated by pesticides can contaminate laundry equipment [Laughlin et al. 1985; Laughlin et al. 1981; Laughlin and Gold 1988, 1989b] and clothing washed with [Clifford and Nies 1989; Easely et al. 1983; Finley et al. 1974; Kim and Wang 1992; Kim et al. 1993; Oakland et al 1989; Braun et al. 1989], or subsequent to [Laughlin et al. 1985; Laughlin and Gold 1989b; Laughlin et al. 1981], the contaminated clothing. Clifford and Nies [1989] found that a uniform on which ethyl parathion was spilled contained 7 g of ethyl parathion/100g of clothing (70,000 ppm) after two washings. Clothes that had been laundered with the originally contaminated uniform contained ethyl parathion at 135-1,500 ppm. Other pesticides that have been shown to transfer to clean clothing when washed with contaminated clothing include: 2,4-dichlorophenoxyacetic acid (2,4-D)ester and amine [Easely et al. 1983]; methyl parathion, toxaphene and DDT [Finley et al. 1974]; atrazine [Kim and Wang 1992; Oakland et al. 1989]; diazinon [Oakland et al. 1989]; and pyrazophos [Braun et al. 1989].

Two reports of family poisonings associated with pesticide-contaminated clothing also provide information on clothing as a source of home contamination. West [1959] found that shoes worn home by a crop sprayer were sufficiently contaminated with demeton to poison the worker’s child who contacted either the shoes or the paper towels that were used to clean them.
Two wives who washed the clothes of workers engaged in the manufacture of kepone developed signs of kepone poisoning [Cannon et al. 1978].

Chlorinated Hydrocarbons
Contamination of homes by 3,3'-dichlorobenzidene and 4,4'-methylene-bis(2-chloroaniline) (MOCA) with some evidence of contaminated clothing as the source have been reported. 3,3'-Dichlorobenzidene in vacuum cleaner dust of homes of workers engaged in its production was found at concentrations up to 10.5 ppm and in dryer lint up to 0.74 ppm [ATSDR 1991b]. MOCA was found in vacuum cleaner dust of homes of workers engaged in plastics manufacture at concentrations up to 2.6 ppm and in dryer lint up to 0.65 ppm [ATSDR 1989b].

Contamination of firefighters protective clothing was documented by Kominski [1987a] who found polychlorobiphenyls (PCBs) up to 1,060 µg/g of clothing following a transformer fire. This study adds credibility to the assumption of clothing contamination by PCBs in the several cases where workers' family members developed chloracne.

Mercury
Evidence for clothing as a source of workers' home contamination with mercury derives from observations of clothing contamination, and the finding of elevated mercury concentrations in areas of homes where soiled clothing was stored and laundered. There are no quantitative data on levels of mercury contamination of clothing.

In a study where mercury was used to calibrate scientific glassware, Danzinger and Possick [1973] reported that mercury particles became embedded in the workers' clothing, especially in knitted fabrics. No measurements of mercury contamination in the homes were made, but the author stated that some female workers would shake mercury particles out of their clothing at home.

Workers milling cinnabar ore wore their mercury-contaminated clothing home and contaminated their cars and their homes [Zalesac 1994]. Mercury contamination of workers' clothes contained in plastic bags was confirmed by sampling the air in the bags; mercury was found in workers' cars at 30-60 µg/m³, and in workers' homes near washers and dryers at 5-50 µg/m³. The occupational exposure limit for inorganic mercury in mining is 50 µg/m³ as an 8-hr. time-weighted average (30 CFR 57.5001).

Additional support for contaminated clothing as a source of home contamination by mercury is provided by Hudson et al. [1985, 1987] and ATSDR [1990a].

Workers exposed to mercury in a thermometer manufacturing plant also brought work clothes and shoes home and contaminated their homes [Hudson
Mercury concentrations in the air of living areas in the workers' homes were 0.02-10 µg/m³ compared to 0.01-1 µg/m³ in control homes. While measurements of clothing contamination were not made, the authors noted that elevated mercury concentrations were found in places where work clothes and shoes were located and in some washing machines.

Workers exposed to high concentrations of mercury during a maintenance operation in a chlor-alkali plant did not shower at the end of the day, and took their work clothing and tools, which were visibly contaminated with mercury, home in their private cars [ATSDR 1990a]. The clothing was washed at home, and the highest concentrations of mercury in the homes were found in the air over washing machines (54 µg/m³) and sinks (7 µg/m³). The mean concentration of mercury in living room air of the 45 contaminated homes was 0.92 µg/m³ (range 0.1-5.0 µg/m³).

While these studies [Zalesak 1994; Hudson et al. 1985, 1987; ATSDR 1990(a)] did not quantitatively measure mercury contamination of clothing, the findings of high levels of contamination in areas where work clothes were located and in washing machines provide evidence that clothing was a source of the home contamination.

**Estrogens**

Measurements of home contamination by estrogenic substances were not found. However, the reports reviewed in this section on documentation of clothing contamination and the effectiveness of measures to prevent home contamination by soiled clothing, support the assumption of clothing as a source of the contamination.

While investigating a pharmaceutical manufacturing plant where children had developed gynecomastia, Aw et al. [1985] found that clothing worn by one of the mothers contained 32 mg of zeranol, a compound with estrogenic properties. The mother washed her clothing at home. Other workers from the plant whose children were similarly effected had also washed their work clothes at home. Specificity of the toxic response together with the documentation of clothing contamination provides evidence of clothing as a source of the children's toxic responses.

Workers engaged in manufacturing diethylstilbestrol wore their soiled clothes home and their children developed signs of estrogen poisoning [Katzenellenbogan 1956]. When controls (special shoes and clothing, showers and laundry) were instituted to prevent home contamination, the health effects were alleviated, providing a basis for the assumption that taking contaminated clothing home was the source of the problem.
Other Substances
Masek et al. [1972] found 3,4-benzo(a)pyrene at up to 14,000 µg/g of clothes in the clothing of workers in a pitch coking plant. No measurements of home contamination were made in this study.

Fibrous glass from contaminated work clothes can be transferred to non-contaminated clothing washed with the contaminated clothing [Peachey 1967; Abel 1966; Madoff 1962].

In the case of silica, one small study indicated that laundering contaminated clothing could be done without contamination of the home area [Versen and Bunn 1989].

Infectious Agents
There is one example where an infectious agent was isolated from clothing contaminated at work [Marmon and Stoker 1956]. In this case, C. burnetii was isolated from a shepherd's clothing following an outbreak of Q fever among the shepherd's contacts. In another case, Q fever occurred in family members of workers at a sheep research station. The family members had no contact with infected animals and their infection may be explained by fomite spread [Rauch et al. 1987], perhaps from contaminated clothing.

Radioactive Substances
Documented cases of home contamination by thorium, americium, and an unidentified radioactive substance exist in the Occurrence Reporting and Processing System (ORPS) database of the Department of Energy (DOE) [Boyle 1994]. In the case of thorium, contaminated clothing was taken home and a pillow case became contaminated, the americium was detected on a worker's hat and his child's diaper, and in the other case, the radioactive material was found in the home on personal clothing worn home the previous day.

Files of the U.S. Nuclear Regulatory Commission (NRC) were found to contain three reports of nuclear power workers' clothing being contaminated with potential for, but unconfirmed, contamination of the workers' homes [Brockman 1993]. These files also contain two reports of laboratory workers shoes being contaminated by radioactive phosphorus (P-32) with subsequent contamination of their cars; however, no mention of home contamination was made in the two cases.

THE WORKER'S BODY
Although it is considered good industrial hygiene practice for many work situations to shower before leaving work, and this practice is often recommended to prevent home contamination, there is no quantitative information about contamination of workers' bodies, except for the hands.
Hands of bridge workers engaged in abrasive blasting of lead-based paint were found by Ewers et al. [1994a, 1995] to be contaminated with lead up to 5 mg/pair. Hands of radiator repair shop workers were found by Piacitelli and Rice [1993] to be contaminated with lead at up to 78 mg/m² (since the combined surface area of both hands is about 1,000 cm², this equates to about 8 mg of lead on two hands). Hands of utility workers engaged in cable splicing were contaminated with lead at up to 5 mg/ft² [Venable et al. 1993] (this equates to about 5 mg of lead on two hands). In an experimental study of fiber contamination on fingers, Schneider et al. [1986] found up to 82 fibers/cm² on fingers after contact with dusty surfaces.

TOOLS AND EQUIPMENT
Workers may take their tools and equipment home with them [Hartle et al. 1987] or transport them in company or private vehicles, as in the case of the workers at the chlor-alkali plant in Tennessee who transported mercury contaminated tools in their private cars [ATSDR 1990a]. Although mercury on tools was not measured, the tools were described as visibly contaminated and air in workers' cars contained mercury at 8-10 µg/m³. Barnett [1994] described an incident where a pesticide applicator took the company truck home, ready for the next day's work. During the night, chloropicrin leaked from its container and contaminated the worker's driveway and the neighbor's premises.

The NRC files contain a record of finding a radioactive hand tool in a nuclear power reactor contractor's home; in this case, the radioactivity was confined to the tool [Brockman 1993]. The only quantitative measurements of tool contamination found were of PCB contamination of tools used in an aluminum extrusion process [Hartle et al. 1987]. A hand wrench had PCB contamination at 308 µg/m² and the external surface of a lunch pail had 14 µg/m²; the authors cited contamination limits of 50-250 µg/m² for low contact surfaces.

TAKING ITEMS HOME FROM WORK
There are 10 reports that document home contamination by workers taking home contaminated items from work for their own use. This practice resulted in serious poisonings or exposures of family members from beryllium [Chesner 1950], asbestos [Li et al. 1989], lead [Carvalho et al. 1984; Dolcourt et al. 1981; Osorio 1994], pesticides [McGee et al. 1952], hormones [Pacynski et al. 1971], and radioactive lumber [Brockman 1993].

Beryllium
Beryllium-ore bags were taken home by a worker at a beryllium plant and given to a neighbor woman who used them for dish cloths and who later died from chronic bilateral granulomatous pneumonitis; beryllium at 0.07 µg/100g of lung tissue was found [Chesner 1950].
Asbestos
Asbestos-contaminated cotton cloth bags that had been used to transport molded asbestos insulation were taken home by a worker and used as diapers [Li et al. 1989]; three family members died of mesothelioma at an early age. It should be noted that dirty clothes were also brought home.

Lead
Lead-contaminated cloths from discarded pollution control filters at a lead smelter were taken home by workers for use at home [Carvalho et al. 1984]. The children of these lead workers had a mean BLL of 67.5 μg/dL. In another case, discarded lead battery casings were taken home for fuel by a worker engaged in recovering lead from used batteries [Dolcourt et al. 1981]. The battery casings were burned in the family’s wood-burning stove. House dust contained up to 43,281 ppm of lead; one child had a BLL of 220 μg/100 mL and developed encephalopathy with seizures. Osorio [1994] reported that when lead contaminated telephone poles were taken home for firewood by a worker, the soil in the yard of the worker’s home where the poles were located contained lead at 1,500-1,600 μg/dL.

Pesticides
Toxaphene-contaminated metal brought home from a processing plant resulted in the death of 2-year-old boy [McGee et al. 1952]. The metal, which consisted of flattened strips made from drums that had contained toxaphene, was used to cover the walls of a tool shed on the day the child, who played in the area, was poisoned. In another case, a loaded company truck was parked in an employees driveway overnight [Barnett 1994]. Part of the load was chloropicrin which leaked from the vehicle, poisoning the next-door neighbors.

Estrogens
Diethylstilbestrol poisoning of family members was considered by Pacynski et al. [1971] to be due in part to women bringing home contaminated factory-supplied milk which was consumed by the children.

Radioactive Substances
Radioactive waste lumber was used to construct a garage at home by a worker engaged in the manufacture of catalysts containing depleted uranium. About 20 years later the garage was found to be contaminated in excess of NRC release criteria [Brockman 1993].

COTTAGE INDUSTRIES
Cottage industries, those where work is undertaken in the same building or on the property where the family resides have been recognized as a hazard since at least 1914 [Oliver 1914]. Poisonings by asbestos, lead, parathion, and mercury have occurred in cottage industries.
Asbestos
Asbestos sheets brought home from work were used in a cottage industry to repair burned out mufflers [Epler et al. 1980]. The asbestos sheets were stored in the basement where the children played and were also used to construct a tree house in which the children played. Both children developed asbestos related lung disease at about age 30. Asbestos cement was produced in the basement of another home for about 20 years [Otte et al. 1990]. The mother, father and one son died of mesothelioma some 40 years after the beginning of the exposures to asbestos.

Lead
In addition to the early report by Oliver [1914] of lead poisoning in family members of home pottery manufacture, 14 recent reports on cottage-industry home contamination and poisoning of family members by lead were found, 6 of them involved pottery.

In the report by Oliver [1914], lead at up to 10,000 ppm was found in dust of potters’ homes where the pottery was dipped in lead glaze in the same room in which the family lived and slept; lead was also found in the clothes of a young boy and a baby. Koplan et al. [1977] reported on six home potters and their families in Barbados and found BLLs up to 71 μg/mL, and average concentrations of lead in dust for the six households of 2,333-88,159 ppm with a maximum value of 325,892 ppm. The State of Alabama [1992] reported finding lead at up to 177,000 μg/ft² in a home pottery workshop where children with elevated BLLs spent some time; elevated lead levels were also found on the kitchen floor of the family’s dwelling.

Other studies of home pottery manufacture did not report levels of contamination, but did report elevated BLLs. BLLs up to 74 μg/100 mL were found for children of workers engaged in ceramics (plates, cups, vases, etc.) production at home in Italy [Abbotti et al. 1979]. Molina-Ballesteros et al. [1983] found BLLs up to 98 μg/dL in children of potters working in their homes in Mexico; and in Japan, Katagiri et al. [1983] reported lead in urine of children of home pottery workers up to 79.3 μg/L compared to 59.9 μg/L in control children; 11.2% of children of home potters had lead in urine greater than 30 μg/L vs. 2.7% of control children. More recently in the United States, Fischbein et al. [1992] reported finding a BLL of 48 μg/dL in a child of a home potter in New York.

Manufacture, repair and recycling of lead batteries by cottage industries have also resulted in contamination of living areas and exposure of family members. Lead loadings up to 53,140 μg/m² were found in households of cottage industry battery repair shops in Jamaica [Matte and Burr 1989; Matte et al 1989]. Matte and Burr [1989] also found that playing in the area of the battery repair shops was an independent predictor of elevated BLLs in children. Other reports did not measure home contamination but reported lead poisoning or elevated BLLs.
Three cases of severe lead poisoning in children whose fathers manufactured lead storage batteries in their homes were reported from the Philippines in 1952 [Anonymous 1952]. An Alaskan battery manufacturing shop investigated by Apol and Single [1980] was located in the building where the owner and his family lived; three of the owner's children who also worked in the shop had elevated BLLs. In a home where battery recycling took place, two children had BLLs of about 65 µg/100 mL [Dolcourt et al. 1981].

Other cottage industries where family exposures to lead have occurred include: (1) backyard smelters in Jamaica (lead on floors of children's' area at up to 109,000 µg/m²) [Matte et al. 1991]; (2) recovery of gold from scrap jewelry in India [Joshua et al. 1971]; (3) quench hardening of cutlery in Japan; and (4) type printing in Japan [Kawai et al. 1983].

Pesticides
Parathion contaminated sheets that had been purchased by a salvage dealer operating out of his home were used by the family; one child who slept in the sheets was severely poisoned on two occasions [Anderson et al. 1965].

Mercury
Mercury poisoning resulting from its use in homes for extracting gold from sand has been reported on four occasions [Haddad and Stenberg 1963; Halle 1969; Hatch 1990; King 1954]. In all of these reports, the person doing the extracting was hospitalized; in two cases family members were also hospitalized [Haddad and Stenberg 1963; Halle 1969]. Mercury excreted in urine over 24 hours by 2 of the extractors were 557 µg and 2,100 µg; for family members 22-176 µg. One of the extractors had a blood mercury level of 193 mg/dL.

FARMS
Farms and ranches are similar to cottage industries in that families live on the property where work is performed. Three types of products used on farms — pesticides, caustics, and estrogenic substances have resulted in several incidents of poisoning of family members.

Children playing with discarded pesticide containers [Wolfe et al. 1961; Johnston 1953] and pesticide contaminated items [Johnston 1953; Eitzman and Wolfson 1967] on farms have resulted in poisonings by toxaphene and parathion. Farm children also have been poisoned by: drinking from containers, such as cups and soft drink bottles containing pesticides [McGee et al. 1952; Eitzman and Wolfson 1967]; and by playing with or eating pesticides that have been improperly stored [Johnston 1953; Simon 1963; MacMillan 1964].

Caustic products used on farms including dehorning products, disinfectants, drain cleaners and pipe line cleaners containing sodium and potassium hydroxide have been responsible for poisoning of over 40 children on United States farms in recent
years [Neidich 1993; Edmonson 1987; Young 1994; Pelegrin 1995; Geisinger Medical Center 1991; Leach and Leach 1992]. Similar poisonings have been reported from Norway [Christensen 1994]. Often the poisonings result from children drinking the caustic solutions from other than the original containers, e.g., soda bottles. These poisonings have caused second degree burns to the esophagus, esophageal perforation, and in one case death.

Estrogenic substances used in animal feed resulted in poisonings of farm children [Bierbaum 1993]. Farm homes have been shown to be contaminated with fungal spores from barns [Pasen et al. 1989]. Livestock or wild animals may serve as reservoirs for infectious agents.

FAMILY VISITS TO WORKPLACE
Visiting the workplace is a source of poisoning of family members that has been identified and is relevant to the concept of workers' family protection. Although it deviates somewhat from the concept of the worker contaminating the home by bringing contaminated items home from work, it is similar to cottage industry and farm situations where family members enter the work area.

Lundquist [1980] discussed the hazards of lead contaminated grounds outside the plant where a parent works. Not only can the workers' shoes become contaminated while walking to the car, but also while waiting to pick up a working parent, children may play on the contaminated grounds.

A nursing mother regularly spent her lunch hour with her husband in his dry cleaning establishment [Bagnell and Ellenberger 1977]. Her infant developed obstructive jaundice and her breast milk contained tetrachloroethylene at 1.0 mg/dL. In another study, Agazzotti et al. [1994] collected alveolar air samples from family members of dry cleaners, and from family members in control homes. Perchloroethylene (tetrachloroethylene) concentrations in alveolar air of family members who visited the workplace were nearly as high at 4.1 mg/m³ as they were in the dry cleaners (6.56 mg/m³). For family members who did not visit the workplace the alveolar air concentration averaged 0.27 mg/m³ compared to 0.008 mg/m³ for controls.

Wilken-Jensen [1983] reported that children of a veterinarian suffered from asthma every time they went to work with their father.
CHAPTER 3. LEVELS OF CONTAMINATION IN HOMES AND CARS

CHAPTER SUMMARY
Measurements of contamination in workers' homes and cars were reported for asbestos, lead, pesticides, mercury, a few chlorinated hydrocarbons, arsenic, and fungi (Table 15). However, for the other substances reviewed as contaminants of workers' homes, data on levels of contamination have not been reported; this is true for beryllium, estrogenic substances, asthmatoxins, cadmium, fibrous glass, and radioactive substances.

For asbestos, there are no studies of contaminated surfaces, but in one study of the air of workers' homes asbestos concentrations up to one-half of the current 8-hr. time-weighted average OSHA exposure limit for workers were found.

There are many studies of workers' home contamination by lead that document the substantial contamination that has occurred. Lead contamination of surfaces is measured either as concentration of lead in dust, expressed as ppm or as the amount of lead covering an area of surface, expressed as weight of lead per unit of area, and referred to as lead loading.

- When the concentration of lead in household dust was measured, average concentrations in workers' homes ranged from 1,600 ppm to 5,000 ppm with maximum values up to 84,000 ppm. In control homes, concentrations were usually less than 1,000 ppm.

- When lead contamination was measured as weight/unit area, workers' homes had lead loadings that were greater than 2,500 \( \mu g/m^2 \), ranging up to 109,000 \( \mu g/m^2 \). Control houses had lead loadings that were less than 1,000 \( \mu g/m^2 \).

- Lead loadings in workers' cars ranged from 1,000 to 300,000 \( \mu g/m^2 \). Control cars had lead loadings that were less than 1,000 \( \mu g/m^2 \).

While measurements of lead in control homes provide some basis for evaluating contamination of workers' homes, guidelines for critical levels of contamination are needed. A value of 500 ppm for the concentrations of lead in dust was used in one study as a threshold for cleaning homes. For lead loading after lead-based paint removal, 2,152 \( \mu g/m^2 \) has been used for floors as a practical, not health-based level. A level of 1,500 \( \mu g/m^2 \) has been stated as a level of concern for children's health.

In three studies of workers' homes contaminated with mercury, concentrations of mercury in air ranging from 0.02 \( \mu g/m^3 \) to 50 \( \mu g/m^3 \) were found. In one study of control homes, concentrations in air ranged from 0.01-1 \( \mu g/m^3 \). Mercury concentrations in contaminated automobiles were 8-60 \( \mu g/m^3 \). The MSHA
permissible occupational exposure limit for inorganic mercury vapor is 50 μg/m³ as an 8-hr. time-weighted average (30 CFR 57.5001).

The few reported measurements of workers' home contamination by pesticides, chlorinated hydrocarbons, arsenic, and fungi also demonstrated high levels of contamination.

**ASBESTOS**

Only one report on measurements of asbestos contamination in workers' homes was found. Nicholson et al. [1980] reported that chrysotile asbestos in 13 air samples from homes of miners and millers in California and Newfoundland ranged from less than 50 to somewhere in the range of 2,000 ng/m³ to 5,000 ng/m³ (1,000 ng/m³ equates to about 0.01 fiber/cm³ [Cossette 1984]). The OSHA maximum permissible concentrations for workplace exposures are 0.1 fiber/cm³ as an 8-hr. average and 1.0 fiber/cm³ as a 30-minute average (29 CFR 1910.1001; 1915.1001; 1926.1101).

**LEAD**

Most of the measurements of lead contamination in workers' homes and cars are of lead concentration in dust expressed as ppm (or the equivalent μg/g) or of lead loading on surfaces expressed as μg/m² or μg/ft² (1 μg/ft² = 10.76 μg/m²). Similar units are used for expressing measurements of contamination of carpets, furniture, and cars.

**Concentrations of Lead in Dust.** Concentrations of lead in house dust of control homes were reported in several studies. Baker et al. [1977] found lead at an average of 404 ppm in control homes for a study of smelter workers in Tennessee, and Rice et al. [1978] found 1,240 ppm in control homes of secondary smelter workers. In control homes for a study of ceramic workers in Colorado, Kaye et al. [1987] found lead concentrations from non-detectable levels up to 320 ppm. For a study of electric cable splicers, Rinehart and Yanagisawa [1993] found 121-879 ppm, in control homes. Watson et al. [1978] found lead at an average of 718 ppm in hosedust of control homes used for a study of battery manufacturing workers in Vermont. As a guideline for cleaning lead contaminated homes in Idaho, an action level of 500 ppm was used [CH₂M Hill 1991].

By contrast to these control measurements, Baker et al. [1977] found an average concentration of lead in house dust of smelter workers of 2,687 ppm, Rice et al. [1978] found 3,310 ppm in homes of secondary lead smelter workers, Kaye et al. [1987] found lead up to 3,400 ppm in homes of the ceramics workers, Rinehart and Yanagisawa [1993] found lead up to 1,600 ppm in homes of electric cable splicers, and Watson et al. [1978] found an average of 2,239 ppm in homes of battery manufacturing workers.

High concentrations of lead in house dust were also found in other studies of smelter workers, cable workers, and battery manufacturing workers. Smelter workers'
homes in Minnesota were found by Winegar et al. [1977] to be contaminated to about the same extent (median lead concentration 2,400 ppm; range 120-26,000 ppm) as the homes of smelter workers reported by Baker et al. [1977]. Homes of workers cutting down lead cable contained lead in dust at 1,700 ppm [Osorio 1994]. Homes of battery factory workers in North Carolina had lead concentrations in house dust ranging from 1,695 ppm to 84,074 ppm [Dolcourt et al. 1978; CDC 1977b].

Lead in house dust of cottage pottery industries in Barbados contained lead at an average concentration of 5,000 ppm [Koplan et al. 1977]. Homes of workers manufacturing pewter products in Ohio contained lead in window sill dust at 1,700 to 25,000 ppm [Kelly 1994].

Kawai et al. [1983] found 100-5,000 ppm of lead in dust from carpets of cottage-industry homes where work with lead took place. Carpets in homes of workers engaged in manufacture of pewter products contained 675-7,200 ppm of lead in dust [Kelly 1994]. Dust from carpets in homes of foundry workers contained lead at 105-1,535 ppm [Nelson and Clift 1992]. Furniture in a home where lead-battery casings were burned as fuel had lead in dust at 13,283 ppm [Dolcourt 1981].

Concentrations of lead in dust of workers’ cars have also been reported. Cars of miners had lead in dust at 3,900 ppm compared to control cars at 917 ppm [Menrath et al. 1993]. Dust in cars of workers manufacturing pewter products contained lead at 700 ppm [Kelly 1994].

**Lead Loading.** For lead loading, that is the total amount of lead per unit surface area, the U.S. Department of Housing and Urban Development (HUD) recommends as feasible levels for samples collected by wipe methods after lead-paint abatement: for hard floors, 200 µg/ft² (2,152 µg/m²); and for window sills 500 µg/ft² (5,380 µg/m²) [Jacobs 1994]. Only three studies of lead loadings in control homes were found. Abbritti et al. [1989] reported an average of 800 µg/m² in homes used for controls in a study of ceramics workers in Italy. Menrath et al. [1993] reported 602 µg/m² in control homes for a study of lead miners in the United States, and Matte et al. [1991] reported 690 µg/m² in control homes for a study of lead smelter workers’ homes in Jamaica.

Lead loadings on floors in homes of backyard lead smelter workers in Jamaica were found by Matte et al. [1991] up to 109,000 µg/m² (geometric mean 2,790 µg/m²). About half the homes had peeling paint with 1%-6% lead. In cottage industry battery repair shops in Jamaica, Matte and Burr [1989] found 190-53,140 µg/m² in wipe samples from the floors of the workers’ homes. Paint samples contained less than 1% lead. Homes of ceramics workers in Italy had lead loadings of 2,700-4,700 µg/m² [Abbritti et al. 1989], and in a cottage pottery industry in Alabama, lead loadings of 172 µg/m² on a bedroom carpet to 4,196 µg/m² on the kitchen floor were found [State of Alabama 1992].

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Lead loading of carpets and furniture was reported by CH₂M Hill [1991]. In this study, lead loadings in carpets and furniture of homes in a lead smelter area were 138,000-2,054,000 µg/m² and 613,000-11,118,000 µg/m², respectively.

Lead loadings in automobiles of workers engaged in: removing lead-based paint from a bridge; radiator repair; electric cable splicing; lead welding and soldering; and battery recycling have been reported. Floors of the bridge workers’ cars had lead loadings ranging from 340µg/m² to 2,000 µg/m² (mean 630 µg/m²); other surfaces had lead loadings less than 500 µg/m² [Ewers et al. 1995; Piacitelli et al. (in press)]. Lead loadings in cars of radiator repair workers were up to 96,000 µg/m² [Piacitelli and Rice 1993]. Cars of electric cable splicers had lead up to 133,000 µg/m² [Venable et al. 1993]. The floor of a car of a worker who soldered and welded with lead had lead at 40,000 µg/m² [CDC 1992a]. Cars of workers engaged in battery recycling had lead loadings of 190,000 µg/m² on the floor, 300,000 µg/m² on the drivers’ seat, and 170,000 µg/m² on the dashboard [Gittleman et al. 1991, 1994].

MERCURY
Measurements of mercury contamination in workers’ homes and cars were reported by ATSDR [1990a], Hudson et al. [1985, 1987], and Zalesak [1994]. Following a single day of exposure to mercury in a maintenance operation, workers contaminated their homes and cars from clothing and tools worn and used at work [ATSDR 1990a]. Mercury concentrations in 25 workers’ homes ranged between 1 µg/m³ and 5 µg/m³, and in cars between 8 µg/m³ and 10 µg/m³. When workers in a thermometer plant contaminated their homes, Hudson et al. [1985, 1987] reported mercury concentrations in air of living areas at 0.02-10 µg/m³ (median 0.24 µg/m³) compared to control homes which had mercury at levels of 0.01-1 µg/m³ (median 0.05 µg/m³). Zalesak [1994] reported that contaminated homes of gold mine workers had concentrations of mercury near washers and dryers at 5-50 µg/m³ and their cars had mercury concentrations at 30-60 µg/m³. The MSHA permissible occupational exposure limit for inorganic mercury vapor [30 CFR 57.5001] is 50 µg/m³ as an 8-hr. time-weighted average.

CHLORINATED HYDROCARBONS
Concentrations of 3,3'-dichlorobenzidine (DCB) in vacuum cleaner dust from homes of workers engaged in its manufacture were at 10.5 ppm [ATSDR 1991b]. House dust in homes of workers exposed to PCBs contained PCBs at concentrations up to 180 ppm [Price and Welch 1972]. No guidelines exist for judging the significance of these contaminant concentrations. No other measurements of home contamination levels for this class of compounds were found.

PESTICIDES
Concentrations of diazinon, chlorpyrifos, and propoxur were found at much higher concentrations in floor dust collected in farmworkers’ homes than in non-farmworkers’ homes [Osorio 1994]. Diazinon was found at averages of 56 µg/m² and 39 ppm in four of five (not detected in the fifth home) farmworkers’ homes.
compared to 0.29 μg/m² and 0.19 ppm in the one non-farmworkers’ homes where it was found. Chlorpyrifos was found in three of the five farmworkers’ homes at averages 6.9 μg/m² and 11.1 ppm compared to 1.1 μg/m² and 0.71 ppm in the one non-farmworker’s home in which it was found. Propoxur was found in one farmworker’s home at 16.9 μg/m² and 0.52 ppm compared to 0.15 μg/m² and 0.10 ppm in the one non-farmworker’s home in which it was found. Twelve other pesticides were found at low levels in some homes of both farmworkers and non-farmworkers.

Since pesticides have many applications and some, such as DDT, are very stable compounds, it is important to determine sources of pesticides when evaluating workers’ home contamination. Lewis et al. [1994] found a total of 23 pesticides in 9 homes in North Carolina. From 8 to 18 different pesticides were found in individual homes. No guidelines for levels of concern for contamination of workers’ homes by pesticides were found.

ARSENIC
Dust in homes of workers exposed to arsenic in Hawaii contained arsenic at 5.2 to 1,080 ppm, compared to 1.1-31 ppm in dust of control homes [Klemmer et al. 1975]. Guidelines by which to judge the significance of these contamination levels were not found.

INFECTIONIOUS AGENTS
Concentrations of fungal spores in farm homes in Finland were 10⁴-10⁷/m³, which was 10-1,000 times the concentrations found in urban homes [Pasanen et al. 1989]. Reports of contamination levels for other infectious agents in workers’ homes were not found.
CHAPTER 4. PREVENTIVE MEASURES

CHAPTER SUMMARY
Several measures that have been taken to prevent contamination of workers' homes and to protect workers' families are identified in the reports reviewed in this Chapter. The measures include:

- reducing exposures in the workplace;
- changing clothes before going home and leaving the soiled clothing at work to be laundered by the employer;
- storing street clothes in separate areas of the workplace to prevent their contamination;
- showering before leaving work;
- prohibiting taking toxic substances or contaminated items home;
- separating work areas from living areas of cottage industries;
- storing and disposing of toxic substances on farms and in cottage industries properly;
- preventing family members from visiting the workplace;
- laundering separately from family laundry when it is necessary to launder contaminated clothing at home; and
- informing workers of the risk to family members from home contamination and ways to prevent it.

The few studies evaluating these measures indicate that they can be effective in reducing or eliminating home contamination. There have also been instances in which home contamination has occurred when one or more of these measures has been omitted.

BERYLLIUM
Following reports of occupational and non-occupational (community and workers' families) cases of berylliosis, the beryllium industry instituted a number of preventive measures, including: engineering controls to reduce air-borne exposures of workers'; community air pollution controls; and measures to prevent exposure of family members to contaminated clothing [Eisenbud et al. 1949; Metzner and Lieben 1961]. In one plant, a double locker system was installed in 1955 which prevented removal of work clothes, underwear, socks and shoes from the facility [Lieben and Metzner
1959]. Until Newman and Kreiss [1992] reported on a case, there were no new cases of berylliosis in beryllium workers' families reported for more than 30 years. This recent case report demonstrates the dangers of any relaxation of preventive measures as the uses of beryllium, the number of workplaces where it exists, and the number of workers exposed expand.

**ASBESTOS**

Although poisoning of asbestos workers' families has been known since the report by Newhouse and Thompson [1965], and has been repeatedly associated with laundering contaminated clothing, no information exists on effectiveness of preventive measures. Belanger et al. [1979] recognized the hazard in evaluation of a factory where asbestos was used in the manufacture of floor coverings. They specifically recommended that work clothes not be taken home because this could expose others at home.

Seixas and Ordin [1986] and Driscoll and Elliott [1990] investigated plants manufacturing brake linings and made recommendations for providing protective clothing, keeping street clothes separate from work clothes, company laundering and showering before leaving work. The OSHA asbestos standards [29 CFR 1910.1001, 29 CFR 1915.1001, and 29 CFR 1926.1101] require these actions when employee exposures exceed 0.1 fiber/cm³ averaged over 8 hrs. or 1.0 fiber/cm³ averaged over 30 minutes. In the absence of information on clothing and personal contamination levels when workers are exposed to asbestos at concentrations below these limits, the adequacy of the OSHA standards for protecting workers' families cannot be judged.

**LEAD**

The report of an investigation of a stained glass window-making studio [Donovan 1994a,b], documented that the use of controls by the studio effectively prevented lead contamination of the worker's home that was adjacent to the studio. Preventive measures used at the studio included local exhaust ventilation during soldering, general dilution ventilation equipped with an electrostatic filter, adhesive mats at doorways to decrease the migration of lead dust on shoes, a laundry room located between the studio and the house that was also used as a changing room, designated work clothing that was only worn in the studio, washing work clothes separately from other clothes, prohibiting work shoes from leaving the studio, and prohibiting the child from entering the studio. Based on the results of surface-wipe sampling, which demonstrated elevated lead levels in the studio (1.2 mg/m³ to 1,600 mg/m³) but not in the home (non-detected or trace), the author concluded that the measures used prevented contamination of the home. The Lead Industries Association, Inc. has produced a video tape entitled "Controlling Lead Exposure for Stained Glass Professionals and Hobbyists" [LIA 1994a].

In another cottage industry, a home-pottery operation, the concerned potter and her family were asked to discontinue being exposed in the facility because of their elevated BLLs [Fischbein et al. 1992]. Two years later, the BLLs were normal, indicating that corrective measures, though not described, were effective. The Lead
Industries Association, Inc. has produced a video tape entitled "Control of Lead Exposure in the Ceramics Industry" [LIA 1993b].

Piacitelli et al. [in press] studied contamination in cars of workers engaged in removing lead-based paint from a bridge. They found that lead contamination was lower in cars of abrasive blasters (379 μg/m³) than in those of other workers (1,100 μg/m³). Abrasive blasters had the highest air-borne exposures at the worksite, but regularly changed clothes and showered before entering their vehicles whereas the other workers (industrial hygiene/safety and security personnel) did not. This study provides evidence of the effectiveness of the preventive measures, but also indicates that the measures should be extended to the lesser exposed workers. The OSHA lead standard for construction workers (29 CFR 1926.62) does not require showering and changing clothes unless exposures exceed 50 μg/m³ as an 8-hr. time-weighted average.

Rinehart and Yanagiswa [1993] found that even though electric cable splicers shower and change clothes at work, they contaminate their homes by taking their contaminated clothing home to wash. Since these workers' exposures are less than the OSHA standard of 50 μg/m³ (29 CFR 1910.1025), employers are not required to launder the employees' clothes.

Excessive lead exposure was identified for workers at a battery factory and for some of the workers' children [CDC 1977b]. The factory initiated a program designed to reduce worker and family exposures. Plant processes, including exhaust ventilation systems, were improved and coveralls and improved shower facilities were provided. Under the direction of the local health department, the homes of the affected children were thoroughly cleaned.

Morton et al. [1982], in a study of BLLs in children of workers engaged in battery manufacture, found that only changing clothes at work did not reduce the risk of elevated BLLs in the workers' children. They recommended showering before leaving work in addition to changing clothes. Similar findings were reported for backyard battery repair shops [Matte and Burr 1989; Matte et al. 1989] where changing from work clothes before entering the home did not result in lower concentrations of lead in house dust.

An article specifically directed at protecting lead battery workers' families was published in Battery News in 1980 [Lundquist, 1980]. This article informs the readers that levels of contamination that may be considered innocuous in the workplace may be of concern in the home where children are exposed and daily exposures are for 24 hours. The article also informs the readers of several sources of home and automobile contamination. In addition to the workers body and clothing, the reader is advised of the hazard of children visiting the workplace and taking home contaminated items such as scrap or surplus material.
Baker et al. [1977] reported on an investigation of a secondary lead smelter. The authors state that "Since this investigation, remedial action has taken place at the smelter and in the workers' homes to reduce lead exposure: work clothes are no longer worn home, workers shower before leaving work, plant processes have been redesigned to reduce lead exposure, and homes have been thoroughly cleaned." Although it is not clearly stated, the article implies that the remedial action was taken in response to the results of the investigation. The effectiveness of preventive measures was not determined.

The Lead Industry Association [LIA 1989; 1991; 1993a,b; 1994a,b] has produced brochures, flyers, and videotapes that provide information for a wider audience that is relevant to preventing workers' home contamination.

CAUSTIC FARM PRODUCTS
After incidents in which children were poisoned by caustic farm products, farm journals published preventive measures [Morris and Morris 1992, 1993; Devries and Devries 1993; Jorgenson 1990]. Morris and Morris [1992, 1993] designed a storage box with a child-proof lid and this design was published in Hoard's Dairyman in 1992 and in the Farm Journal in 1993. Another design for storing hazardous chemicals in dairy barns was also published in Hoard's Dairyman in 1993 [Devries and Devries 1993]. Several precautions that farmers need to take with caustic dairy cleaners were enumerated by Jorgenson [1990]. These include:

- rinsing the measuring containers immediately;
- mounting the original container and attachments out of children's reach;
- leaving labels on containers;
- never storing chemicals in food containers, soda bottles, or cups;
- storing chemicals in a locked storage area out of a child's reach; and
- properly disposing of empty containers. "Don't leave them—even rinsed ones—around for children to find."

PESTICIDES
Barnett [1994] reported on a pesticide spill (chloropicrin) that occurred at the house of an employee who was preparing a work vehicle for a 12-day trip to treat utility poles. Next door residents, two adults and three children, became ill and the local fire department was called to clean up the spill. As a result of this incident, the employer instituted workplace changes which included a policy that company vehicles were not to be taken home, and that appropriate storage and means to secure containers while transporting chloropicrin be used.
Finley et al. [1977] demonstrated that delaying entering a field for 4 days after spraying with methyl parathion reduced clothing contamination by 99% from that on the first day after spraying. Thus delayed entry would substantially reduce exposure of launderers and potential for contamination of laundry equipment.

A number of publications have addressed hazards to workers and to workers' family members and advised on preventive measures [Wyant-McNutt 1983; Lavy 1988; Branson and Henry 1982; Rigakis et al. 1987; Easley et al. 1981a; Laughlin and Gold 1989c; Stone and Wintersteen 1987; Anonymous 1994; Finley et al. (no date)]. Several of these publications are pamphlets produced and distributed by Agricultural Experiment Stations and Extension Services to advise workers and their families on proper procedures for handling and laundering the pesticide contaminated clothing, based on research reviewed in Chapter 5 and summarized in Table 16. Briefly the recommendations in these publications and pamphlets for handling and laundering clothing contaminated with pesticides include:

- Discard or burn heavily contaminated clothing (e.g., after a spill);
- Store soiled clothing separately from other clothing;
- Use rubber gloves when handling soiled clothing;
- Launder soiled clothing separately from other clothing;
- Launder contaminated clothing after each use, and on the day of use, if possible;
- Laundry methods should include a pre-wash treatment, heavy duty detergent, hot water (e.g., 60°C), a complete wash cycle, full water volume, a wash time of 12-14 minutes, and a double rinse;
- Clean the washing machine after laundering contaminated clothing by running the machine through a complete cycle with a full volume of water and detergent; and
- Line air dry the clothes to avoid contamination of an automatic dryer and to allow sunlight and time to further reduce the toxic residues.

Four surveys on how workers and workers' family members handle pesticides or contaminated clothing have been reported. The first survey, conducted in 1982, was of licensed professional agricultural workers in Louisiana [Cloud et al. 1983]. More than half of the respondents were unaware of the existence of disposable protective garments, and the common clothing worn was short sleeved shirts and denim or khaki pants. Home laundering was the rule, the clothes were usually stored in clothes hampers and laundered within two days of use. About 30% of the respondents laundered the contaminated clothing with other family clothing, and none reported using any pretreatments.
Grain growers in Alberta, Canada who did their own pesticide application were asked to respond to a laundry practices questionnaire in 1984 [Rigakis et al. 1987]. The persons who did the laundry (97% were wives) were the responders. In 34% of the families, other family members assisted with the laundry. Pesticide contaminated clothing was stored apart from other clothing prior to laundering by 62% of the responders, 59% washed contaminated clothing separately from other clothing, and 60% used pre-wash treatment of the clothing. However, only 18% of the responders reported washing the contaminated clothing on the day it was used, only 25% used water heated to the usually recommended temperature, 73% used less detergent than recommended on the container, 76% used a clothes dryer, and only 6% used rubber gloves for handling contaminated clothes. Based on these results, information on handling and laundering contaminated clothing was developed and distributed to farmers.

In 1983, a pamphlet entitled "What to Do when Clothes Are Soiled With Pesticides" was published by the Iowa Cooperative Extension Service [Stone and Wintersteen 1988]. A survey of laundering practices among farm families was conducted in 1984 [Stone et al. 1986]. The recommendations made in 1983 appear to have influenced laundry practices in Iowa. The findings of this survey of 368 registered pesticide applicators indicated much better laundry practices in Iowa, than were found by Cloud et al. [1983] in Louisiana. Nearly all (98%) of the applicators' clothing was washed at home and most families (greater than or equal to 90%) stored and washed the soiled clothing separately from other clothing. Full water levels were used by three-fourths of the launderers and about half used hot water. However, 68% of the launderers did not clean the washing machine after washing the contaminated clothing and 73% did not destroy clothing on which concentrated liquid pesticides had been spilled. The recommendations made in 1983 appear to have influenced laundry practices in Iowa.

Pesticide applicators (23) and farmers (15) in California responded to a questionnaire that solicited information on their attitudes about some factors relevant to family protection [Rucker et al. 1986]. The applicators all considered that it wasn't safe for children to be around when they were working with pesticides; the growers were less certain on this item. When asked where they stored their contaminated clothing, most of them responded that they never stored their clothes with the family laundry or in closets with other clothes. Also, most of them reported that the contaminated clothing was always washed in separate loads from other clothing; however, most of them did not pre-rinse the clothing before washing and most of them used a single wash.

HORMONES
Effectiveness of controls in the manufacture of diethylstilbestrol in eliminating the hyperestrogenic signs in the children of workers who manufacture the compound was documented by Katzenellenbogen [1956] and Pacynski et al. [1971]. The preventive
measures included use of gloves, special shoes and clothing, and laundering of work clothes by the company.

ASTHMATOGENS/ALLERGENS
Two case studies of workers' spouses who had allergic reactions to antigens brought home by their spouse indicate that the practices of showering and changing clothes before leaving work were effective at preventing the allergic reactions. In the first case, the antigen was of animal origin which the wife brought home on her clothing and body from the research laboratory where she worked. The symptoms of the worker's husband resolved after the worker began wearing different clothes at work and at home, and showering and washing her hair before leaving work. In the second case, the antigen was platinum salts which the husband brought home on his clothing and body from his work at a precious metals refining company. The symptoms of the worker's wife resolved after her husband's company instituted a policy that employees should shower and change clothes before leaving work.
CHAPTER 5. DECONTAMINATION PROCEDURES

CHAPTER SUMMARY
In this Chapter studies relevant to decontamination of clothing, homes and equipment are reviewed. Decontamination procedures include: air showers; laundering; dry cleaning; dispersal into the air; vacuuming; shampooing; washing; and chemical or physical destruction of contaminated items. The effectiveness of decontamination procedures depends upon the physical and chemical characteristics of the contaminant, the level of contamination, and the physical characteristics of the contaminated material or item.

Clothing and carpets are perhaps the most difficult items to decontaminate. Most studies on decontamination of clothing have been on laundry procedures for pesticides, although some clothing decontamination information exists for: fibrous materials, including asbestos; beryllium; PCBs; lead; 3,4-benzo(a)pyrene; and infectious agents.

Reports on decontamination of homes that were contaminated by workers’ take-home activities are limited to lead, mercury, and 4,4’-methylene-bis-(2-chloroaniline) (MOCA). Information on decontamination of buildings contaminated by dioxin and polychlorinated biphenyl (PCB) from sources other than workers’ take-home activities provides additional information on the effectiveness of decontamination procedures. For asbestos, only laboratory studies are available.

Decontamination is the last resort in protecting workers’ family members, a step that must be taken when preventive measures have not been used or were inadequate. The decontamination process can be hazardous to persons involved in the process. Hazardous concentrations of contaminants can be generated when handling contaminated clothing, vacuuming and mopping floors. Contaminants can be transferred to other clothing during laundry and dry cleaning. Laundry and dry cleaning equipment can become contaminated with subsequent contamination of other clothing.

Normal house cleaning and laundry practices are usually not effective. To achieve acceptable levels of decontamination, special procedures are required. These procedures include specially designed vacuum cleaners, special cleaning compounds, and use of appropriate laundry procedures, including decontamination of laundry equipment after each use. Destroying highly contaminated items such as carpets, furniture, and clothing may be the most effective and practical decontamination procedure.

DECONTAMINATION GUIDELINES
While the studies cited in this section provide information on decontamination procedures and effectiveness for several contaminants, guidelines on levels of contamination that are acceptable were found only for lead, mercury, and PCBs.
For lead loading, the Department of Housing and Urban Development (HUD) has
guidelines for floors of 200 μg/ft² (2,152 μg/m²) and for window sills of 500 μg/ft²
(5,380 μg/m²) [Jacobs 1994]. The HUD guidelines are not based on health
considerations, they are based on levels that can be practically achieved following
lead-paint abatement. These values were used by the State of Alabama [1992] and
Pollock [1994]. Matte and Burr [1989] cited 1,500 μg/m² as a level of concern for
children’s health. CH₂M Hill [1991] used a concentration of lead in dust of 500 ppm
as an action level for cleaning residences.

Mercury concentrations in air of workers’ homes of 0.5 μg/m³ and 1.0 μg/m³ were
used as decontamination goals in the reports by ATSDR [1990a] and Zirschky and
Witherell [1987] respectively. In a recent report on decontamination of homes in
Florida, 0.3 μg/m³ was the level at which families were allowed to return to their
homes following decontamination [CDC 1995].

For PCBs, EPA guidelines for indoor solid surfaces and high contact outdoor solid
surfaces state that post clean-up levels should not exceed 10 μg/100 cm² (40 CFR
761.125). Based on PCB levels found in non-manufacturing buildings, 0.11 μg/100
cm² was used as a guideline for decontaminating a school building [Orris and
Kominsky 1984]. Other guidelines cited by Hartle et al. [1987] were 0.5 μg/100 cm²
for office buildings; for an aircraft plant, 2.50 μg/100 cm² was cited for low contact
surfaces, and 1 μg/100 cm² for high contact surfaces.

REVIEW OF DECONTAMINATION PROCEDURES
BERYLLIUM
Shirts worn for one day in a beryllium plant were studied by Cohen and Positano
[1986]. Three shirts were classified as “nearly new” and three were classified as “old.”
One “nearly new” and one “old” shirt was laundered at the workplace. Beryllium was
present at 22 mg/m³ and 30 mg/m³ in the “old” washed shirt. Beryllium was present
at 12 mg/m³ and 20 mg/m³ in the “nearly new” unwashed shirts and at 0.2 mg/m³ in
the “nearly new” washed shirt. Although this was a pilot study, it is the only study
found that provides information on laundering clothing contaminated with beryllium
or similar particulate material. The study indicates that the beryllium was laundered
from the “nearly new” shirts, but that beryllium had accumulated and was well
entrenched in the “old” shirts. Substantial levels of beryllium dust in air were
generated during laundry procedures. The concentrations were up to 1.2 mg/m³
[Eisenbud et al. 1949] compared to the occupational exposure limit of 2 μg/m³ as an
8-hr. time-weighted average [29 CFR 1910.1000].

ASBESTOS
There were no studies on the effectiveness of any methods for removal of asbestos
from clothing contaminated in the workplace. One study conducted on dry cleaning
a coat which contained 8% asbestos in its fabric, indicated that some of the loose
fibers were removed [NIOSH 1971]. Concentrations in the air of asbestos fibers
longer than 5 μ that were generated by wearing the coat before cleaning were around 2/cc whereas after cleaning the concentrations were about 0.5/cc. Since the fibers were part of the fabric, the study may underestimate the ability of laundry procedures to remove asbestos from contaminated clothing. Asbestos fibers were transferred to sport coats dry cleaned with the coat containing asbestos.

In a laboratory study, asbestos-contaminated carpets were cleaned for about 65 minutes by either dry vacuuming or hot water extraction, using vacuum cleaners equipped with high efficiency particulate air (HEPA) filters [Kominsky et al. 1990]. The carpets were artificially contaminated with \(9.3 \times 10^4\) and \(9.3 \times 10^6\) asbestos structures per meter squared (s/m²), based on levels found in carpets from an asbestos-containing building. Dry vacuuming removed little or no asbestos from the carpets whereas hot water extraction removed about 70%. An important aspect of this study was the effect of the cleaning procedures on airborne asbestos concentrations. During carpet cleaning, by either method and at either level of carpet contamination, average asbestos concentrations in room air of 0.15-0.25 s/cm³ were generated. The OSHA permissible exposure limit for asbestos is 0.1 fiber/cm³ as an 8-hr. time-weighted average [29 CFR 1910.1001; 29 CFR 1915.1001; 29 CFR 1926.1101].

Resuspension of asbestos fibers was observed with a fiber aerosol monitor during a daily cleaning period of a classroom by Litzistorf et al. [1985]. Resuspension of dust by cleaning activities is an important consideration not only for the decontamination process, but also for persons living in the home and performing routine cleaning operations.

LEAD
No studies on laundering of clothes contaminated with lead were found. However, Simonson and Meecham [1983] showed that a workplace airshower removed from 5% to 72% of lead oxide dust from clothing samples contaminated with about 1 mg/cm², and from 23% to 69% from samples contaminated with about 0.6 - 2.5 mg/cm² in laboratory studies. A small amount of lead was blown through the clothing to the underclothing and body of the workers (up to 1% of the dust loading).

Ewers et al. [1994b] studied the effectiveness of dry vacuuming for removal of lead from carpets taken from homes of children who had high BLLs. These carpets were highly contaminated with surface lead loadings of 114,000 μg/m² to 5,650,000 μg/m². The carpets were vacuumed with commercially available vacuum cleaners intended for industrial use. The vacuum cleaners were equipped with HEPA filters and fitted with a commercial beater bar nozzle. The carpets were vacuumed 10 times for 1 min/m² each time. Surface loadings and amount of lead removed were measured after each vacuuming. After some of the earlier vacuumings, lead loading on the surface increased by up to four times, but by the tenth vacuuming the surface lead loading was reduced to 6%-61% (average 20%) of the initial loading. The
investigators concluded that it may be more practical to replace than clean contaminated carpets.

In a study of contaminated homes near a lead smelter, the carpets were first vacuumed with a high efficiency particulate vacuum cleaner equipped with a beater bar attachment, then shampooed three times with an industrial grade shampooer [CH_2M Hill 1991]. The carpets in this study had lead loadings of 130,000 μg/m² to 2,500,000 μg/m². The cleaning procedures reduced the loadings by only 0.9%-13.5%. The authors estimated that 74 separate shampooings would be needed to remove all lead from the carpets.

Vacuuming of lead-containing carpets with a power carpet beater followed by steam cleaning with a commercial carpet cleaner containing a water-detergent mixture was also found by Milar and Mushak [1982] to have little effect on the level of lead contamination. Two steam cleanings 24 hours apart using detergent in the vacuum cleaner reduced the concentration of lead in dust by 12% and the lead loading by 38%. When a Calgon® (sodium hexametaphosphate) solution (1 lb/5 gal water) was used for the initial steam cleaning followed a day later by steam cleaning with detergent, lead concentration in the carpet dust was reduced by 61% and the lead loading by up to 91%. The authors suggested that Calgon® coats the particulate surface with phosphate or polyphosphate groups, reducing electrostatic interaction with carpet fiber and allowing easier removal by detergent. When this method was applied to decontaminating a home where a worker took home battery casings to use for fuel in the family stove, a surface lead loading of 4,125 μg/m² was reduced to 1,961 μg/m² [Dolcourt et al. 1981].

Decontamination of other surfaces is generally more effective than cleaning carpets. Farfel and Chisolm [1990] reported that the ability to reduce lead dust levels on household surfaces after lead-paint abatement activities depended on their condition. Smooth floor surfaces such as vinyl tile and linoleum tended to have lower dust levels than wooden floors which tended to be pitted, splintered and worn.

HEPA vacuuming of bare wooden floors for 1 min/m², removed from 14% to 62% of the total lead removable (95% of that present) by vacuuming for 5 min/m² followed by washing with tap water [Ewers et al. 1994b]; the condition of the floors was not described. For linoleum floors, most of the lead dust that could be removed by vacuuming for 5 min/m² (75% of total dust present) was removed in the first two minutes. With linoleum floors, about 20% of lead dust was removed by the post-vacuuming washing, whereas less than 5% was removed by the post-vacuuming washing of bare wooden floors.

PESTICIDES
Twenty-eight studies on decontaminating clothing contaminated with pesticides are summarized in Table 16. Many of these studies were included in a review article by Laughlin and Gold [1988]. The studies involve 33 pesticides with a wide range of
chemical and physical properties including: 14 acetylcholinesterase inhibiting insecticides (9 organophosphates and 5 carbamates); 5 organochlorine insecticides; 4 pyrethroid insecticides; 9 herbicides; and 1 fungicide. The studies were conducted with various formulations including liquids, emulsifiable concentrates, encapsulated, wettable powders, and water dispersible granules. Various fabrics were contaminated, usually by laboratory procedures, to determine the effectiveness of different laundry procedures. Fabrics of different weights and weaves (e.g., twill denim, poplin) were chosen to represent clothing worn by exposed workers; they included 100% cotton, various cotton polyester blends, and fabrics treated with soil repellents. Most of the studies were conducted using an accelerated laundering apparatus (Atlas Launder-Ometer), usually using 150 mL water. Laundry variables studied included water temperature, detergent, and pre-wash treatment.

The various laundry procedures applied to pesticides removed from about 20% to over 99% of the contaminant from the cloth, depending on pesticide characteristics, clothing characteristics, and laundry variables.

Pesticide characteristics that make cleaning difficult include: (1) formulation as an emulsifiable concentrate [Easley et al. 1981b; Kim et al. 1993; Laughlin and Gold [1989a,b; Nelson et al. 1992; Laughlin et al. 1985; Easter 1983]; (2) high concentration of pesticide [Easley et al. 1982a; Laughlin and Gold 1989a; Laughlin et al. 1985]; and (3) low solubility in water (e.g., 1 mg/L) [Easley et al. 1983]. Repeated contamination without laundering after each use also makes it more difficult to remove pesticides from clothing [Goodman et al. 1988].

Clothing characteristics that may influence the effectiveness of laundering include: weight of the fabric, chemical composition of the fiber (e.g., cotton, wool, nylon, polyester), and functional finishes (durable press, soil-release, and soil repellent). Heavier fabrics, such as denim pants, may be more difficult to clean than lighter ones, such as shirts [Kim et al. 1982].

Most studies on the effect of chemical composition of the fibers have been with various blends of cotton and polyester, ranging from 100% cotton to 65%/35% polyester-cotton. The results of these studies are variable, and most of them have found no effect with methyl parathion [Easley et al. 1981b; Easley et al. 1982b; Finley et al. 1974; Goodman et al. 1988; Laughlin et al. 1985; Laughlin and Gold 1989a]. Finley and Rogillo [1969] studied fabrics worn in cotton fields the day after spraying with a mixture of DDT and methyl parathion. They found that after-washing residues of DDT were greater for 100% cotton and 65%/35% cotton-polyester fabrics than for 50%/50% and 35%/65% cotton-polyester fabrics. The same was found for methyl parathion but to a lesser extent. By contrast, Nelson et al. [1992] found greater after-washing residues on 50%/50% cotton-polyester fabrics than on 100% cotton fabrics when organophosphates (methyl parathion, fonofos, and terbufos) were the contaminants. In the same study, Nelson et al. [1992] found residues greater on the 100% cotton fabric when carbaryl and atrazine were the
contaminants. Lillie et al. [1981] found no differences in after-washing residues between 100% cotton and 100% polyester fabrics when diazinon, propoxur, carbaryl and prometon were the contaminants.

Functional finishes generally make laundering less effective [Laughlin and Gold 1988]. Keaschall et al. [1986] found that fluorocarbon finishes reduced absorption of pesticides, but did not facilitate removal by laundering. Laughlin and Gold [1989a,b] found that while contamination of 100% cotton and 50%/50% cotton-polyester fabrics was decreased by finishing the fabrics with a fluoroaliphatic soil repellent, residual methyl parathion after washing was greater in the finished fabrics. Similar results were reported by Hild et al. [1989] for 50%/50% cotton-polyester fabric.

Laundry variables that have been studied include: water temperature, detergents, pre-wash treatment, water level, drying method (air/machine), repeated washing, and other laundry additives (bleach, ammonia).

Hot water (60°C), in general, removes more pesticides than warm (49°C) or cold (30°C) water [Lillie et al. 1981; Kim et al. 1982; Kim et al. 1986; Lillie et al. 1982; Kim and Wang 1992; Easter 1983; Easter and DeJange 1985; Laughlin et al. 1985]. With 2,4-D ester which is classified as insoluble, Easter et al. [1983] found 26% removal from contaminated fabrics washed at 30°C and 45% removal when washed at 60°C. With the 2,4-D amine (solubility 4,400-18,000 mg/L at 30°C), more than 99% was removed when washed either at 30°C or 60°C. Similarly, Chiao-Cheng et al. [1988] found that more than 99% of carbofuran (solubility 700 mg/L) and methomyl (solubility 60,000 mg/L) were removed by washing at either 49°C or 60°C.

Detergents that have been studied include heavy-duty liquid detergents without phosphates, detergents with phosphates, and those with carbonates. Easley et al. [1982b] and Laughlin et al. [1985] found that a heavy duty liquid detergent resulted in lower residual methyl parathion in fabrics after washing than when commercially available detergents containing phosphates or carbonates were used. A heavy-duty liquid detergent without phosphate was also found to be superior to a 12% phosphate detergent for laundering clothes contaminated with 2,4-D ester. By contrast, Kim et al. [1986] found that detergents containing phosphate were superior to a heavy-duty liquid detergent for removing alachlor from contaminated fabrics. Hild et al. [1989] found that a heavy-duty nonionic liquid detergent and an anionic phosphate detergent were equally effective in removing parathion from contaminated fabrics.

Pre-wash treatments have generally been found to contribute substantially to removal of pesticides from fabrics. Nelson et al. [1992] found that a commercial pre-wash product lowered the amount of contaminants remaining in fabrics after washing for 11 pesticides, and Keaschall et al. [1986] found that a pre-wash spray and a degreaser were both beneficial for removing another group of 11 pesticides from contaminated fabrics. Rigakis et al. [1987] confirmed the effectiveness of a pre-wash treatment to enhance removal of three of the pesticides studied by Nelson et al. [1992]. Kim et al.
[1986] found perchloroethylene to be a more effective pre-wash treatment than ethyl alcohol for removing alachlor from fabrics.

Water level was shown by Hild et al. [1989] to result in lower levels of methyl parathion in laundered fabrics.

Kim et al. [1986] found that machine drying of fabrics that had been contaminated with alachlor resulted in lower residual contaminant than air drying. On the other hand, Kim and Wang [1992] found no difference in residual atrazine between machine and air drying.

Repeated washing has been studied with parathion, 2,4-D ester, pyrazophos, triallate, trifluralin, and deltamethrin. Satoh [1979] found that a single washing removed 75%-95% of parathion from clothing contaminated from one day of work in a cotton field; the second washing removed a smaller percentage of the remaining contaminant. It was noted that the more contaminated the clothing the harder it was to clean.

The effect of concentration of methyl parathion on laundry effectiveness was reported by Easley et al. [1982a] and Laughlin et al. [1985]. When an emulsifiable concentrate was applied to fabrics at a concentration of 1.25%, 18% of that applied remained after the first wash, 4% after the second wash, and 0.37% after the tenth wash. When the emulsifiable concentrate was applied at a concentration of 54%, 84% of that applied remained after the first wash, 65% after the second wash, and 33% remained after the tenth wash.

Pyrazophos was applied to fabrics to simulate contamination in greenhouses by spraying or spilling [Braun et al. 1989]. When applied by spraying, the first wash removed 78% of the contaminant and the second wash removed an additional 14%. When applied by spilling, the first wash removed 92% of the contaminant but the second wash removed only an additional 2%.

Easley et al. [1983] found that a single washing of fabrics contaminated with 2,4-D ester removed about 30% of that applied and that two washings removed about 41%.

Fifty-two percent of triallate was removed by the first washing and an additional 30% by the second washing in a study by Rigakis et al. [1987]. In this same report, the first washing removed 77% of trifluralin and 84% deltamethrin; the second washings removed an additional 14% of trifluralin, and 15% of deltamethrin.

Other laundry additives such as bleach and ammonia have been found to have little effect on the effectiveness of laundering fabrics contaminated with methyl parathion [Easley et al. 1981b; Laughlin et al. 1985], or with chlorpyrifos, diazinon, and chlordane [Lillie et al. 1982].
Other clothing decontamination methods that have been studied include storage with and without air flow, and chemical decomposition by heat. Laughlin and Gold [1989a] found that residues of methyl parathion remaining in fabrics after a single wash decreased when stored in moving air up to six months. Alachlor was found to degrade rapidly in contaminated fabrics heated in a convexion oven at 200°C and after 30 minutes when heated at 150°C; at 150°C for 60 minutes, the residue was 0.005% of the contamination level [Kim 1989]. Microwaves of 2,450 MHZ at 50W, 250W, and 500W for up to 200 seconds were not very effective.

CHLORINATED HYDROCARBONS
Kominsky [1984b, 1987a] reported on decontamination of clothing contaminated by PCBs during a fire at an electric transformer oil reclamation facility. In one setting, the Nomex® protective clothing was dipped into a tub containing detergent followed by a water rinse in a second tub. The procedure reduced the PCB surface contamination (from 15.8 µg/cm² to 0.35 - 7.2 µg/cm²), but may have increased the concentration of PCBs in the fabric (0.76 - 601 µg/gm fabric before washing; 14-1050 µg/gm fabric after washing).

Subsequently, Kominsky [1987a] conducted a dry-cleaning experiment on Nomex® clothing that was contaminated during the same fire. The clothing contained PCBs at 5.3-480 µg/g fabric. Laboratory-contaminated Nomex® clothing containing PCBs at 10,000-1,000,000 µg/g fabric were also included in the study. A trichlorotrifluoroethane (Freon® 113)-based dry cleaning machine with a revolving chamber system was used. This process reduced site-contaminated garment PCB levels by an average of 88%, and of laboratory-contaminated garments by 99%. Since safe levels of surface contamination are not known, it could not be stated if this would protect workers or prevent home contamination.

Homes were contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (dioxin) when contaminated waste oil was used for dust suppression in two neighborhoods [Hess 1988; Doherty 1984]. Hess [1988] reported on decontamination of eight residences and three commercial buildings in one of the areas. After vacuuming with a high efficiency electric vacuum equipped with a HEPA filter, every surface was wiped with a damp cloth and detergent wash. The procedure resulted in non-detectable levels of dioxin in the eight residences and two commercial buildings, and reduced the level in the most contaminated building from 36.6 ppb to 13 ppb. In the other area, Doherty [1984] removed the contaminated carpet, then thoroughly vacuumed and washed the residence with a detergent solution. A post-clean-up vacuum dust sample was negative for dioxin.

Homes contaminated with MOCA, including homes of exposed workers, were cleaned by a commercial carpet cleaner [Hesse 1991]. In the more contaminated homes, the amount of MOCA removed by vacuuming carpets before commercial cleaning was 30-300 times that collected after commercial cleaning. However, in a laboratory study, a single commercial cleaning technique removed only 31% of the
MOCA contained in a carpet from a worker's home. In this study, it was found that dry vacuuming with a good household style vacuum was equal to or better than the wet commercial method for removing MOCA from carpet.

When a transformer in a school building malfunctioned, dielectric fluid was vented into the transformer vault [Orris and Kominsky 1984]. Contamination by PCBs, trichlorobenzene, and tetrachlorobenzene occurred to varying degrees throughout the building. All vertical and horizontal surfaces in the building were washed with liquid alkaline synthetic detergent formulated for penetration and removal of PCBs. The cleaning procedure reduced PCB surface concentration in the most contaminated area by 98%, from 2,620 µg/100 cm² to 46 µg/100 cm². The cleaning procedure was also effective in reducing concentrations of PCBs, trichlorobenzene, and tetrachlorobenzene in air by 90-98%.

MERCURY
No information was found on decontamination of clothing contaminated with mercury; however contamination of washing machines after laundering contaminated clothing has been found [ATSDR 1990a; Hudson et al. 1985, 1988; Zalesak 1994].

Workers' homes contaminated with mercury have been successfully decontaminated [Zirschky and Witherell 1987; ATSDR 1990a; CDC 1995]. However, unless special techniques are used, mercury vapor can be generated in the cleanup process [Votaw and Zey 1991; Zey 1988] and vacuum cleaners can become contaminated [Tubbs and Galson 1989; Zey 1984; Zey 1988]. An employee's exposure to mercury while vacuuming in a dental office was at 69 µg/m³ compared to 8.5 µg/m³ when not vacuuming [Votaw and Zey 1991; Zey 1988]. Workers' family members who vacuumed and mopped floors in workers' contaminated homes were found to be at increased risk for elevated levels of mercury in urine [ATSDR 1990a].

Methodology used to successfully decontaminate workers' homes includes vacuum cleaners specially designed for mercury and use of mercury suppressants for cleaning surfaces [Zirsky and Witherell 1987; ERM Southeast, Inc. 1989; ATSDR 1990a, CDC 1995].

Vacuum cleaners for mercury are equipped with a suction hose, vacuum pump, inline mercury trap, charcoal filters to remove mercury vapor and HEPA filters to remove mercury droplets [Reisdorf and D’Orlando 1984]. Mercury suppressants chemically combine with mercury, enhancing the ability to remove it from contaminated surfaces [Murphy 1978]. Using these procedures, 25 workers' homes in Tennessee were decontaminated to mercury concentrations that were less than 0.5 µg/m³ from decontamination levels that were up to 5.0 µg/m³ [ATSDR 1990a]. The homes of thermometer plant workers were decontaminated to levels below 1 µg/m³ [Zirschky and Witherell 1987]; the contaminant levels were not well described, but one report indicated the levels may have been as high as "4 times the levels allowed at work" [Trost 1985]. In a recent report of homes contaminated by children playing with
abandoned mercury, the homes were decontaminated by a combination of techniques including destruction of contaminated items, vacuuming, and ventilation [CDC 1995].

OTHER SUBSTANCES
Commercial laundering of clothing contaminated in the workplace with ceramic fibers at 50 to 500 fibers/mm² reduced the contamination by 86%-100%, resulting in levels that ranged up to 7 fibers/mm² [Weller 1994].

One article discusses the effectiveness of removing 3,4 Benzo(a)pyrene by washing work clothes worn by workers at a pitch coking plant [Masek et al. 1972]. The author states that "the present procedure of washing working underwear and clothes by no means ensures an efficient removal of the carcinogenic 3,4-benzo(a)pyrene from the fabric . . . ." It is assumed that the "present procedure" was normal detergent and rinse.

Perkins et al. [1987] found that Freon® decontamination reduced toluene to 0.8% of the original exposure on a butyl rubber test material. A soap and water decontamination reduced the level to 1.1 percent. However, air drying the test material at 50°C for 24 hours reduced the level to 0.25%. This research also showed that air drying at 50°C reduced seven other solvent contaminants to limits of detection. Finding no evidence of damage to the material with this process, the authors recommended it as the preferred means of decontaminating chemical protective clothing against solvents. They noted that with small amounts of contamination from solvents with "substantially different solubility properties from the protective clothing," air drying at room temperature for 24 hours should be adequate to remove the solvent.

INFECTIOUS AGENTS
Several articles, and general recommendations [Joint Committee on Health Care Laundry Guidelines 1983] exist regarding laundering to remove biologic agents, such as anthrax, which can be transmitted to laundry personnel via work clothes [Hardy 1965], or fungal spores which can be brought into farmers' homes on work clothes [Pasanen et al. 1989].

In the medical facility setting, laundering is universally recommended, and is believed to be effective in killing or markedly reducing biological contamination of clothing and linens [Garner and Favero 1987]. Although a major emphasis of laundering in this setting is to prevent contagion spread in the medical facility, effective laundering and other decontamination practices also help to protect employees from bringing infectious diseases into their homes. A number of mechanisms are probably active in this process, including dilution and inactivation or the microbicidal properties of heat, detergents, pH changes, chlorine, and drying. Studies of bacterial survival after various types of hospital laundering have shown marked reduction of viable bacteria [Walter and Schillinger 1975], [Christian et al. 1983], [Blaser et al. 1984]. Careful procedures and appropriate equipment are needed to ensure that the laundry staff
themselves are not contaminated with the hazardous biological materials [Garner and Favero 1987; McKay-Ferguson and Mortimer 1977].
CHAPTER 6. REVIEW OF EXISTING FEDERAL AND STATES LAWS

CHAPTER SUMMARY

FEDERAL LAWS

Statutes and rules are reviewed that provide Federal agencies with relatively limited authority to prevent or remediate workers' home contamination. Table 17 displays, in summary form, the text of those statutes most relevant to this topic, while Table 18 provides a brief explanation of the relevant regulations. Examples of workers' home contamination in which Federal agencies exercised their statutory and regulatory authority are presented in the following chapter, as well as Table 19. Seven statutes were identified in the United States Code (U.S.C.), and 20 regulations were found in the Code of Federal Regulations (CFR), that addressed workers' home contamination. Below are summaries of Federal and State laws that are relevant to the issue of workers' home contamination.

Under the Occupational Safety and Health Act of 1970 (OSH Act), the Occupational Safety and Health Administration (OSHA) has limited authority to develop and promulgate standards for protecting workers' families directly; under this authority, OSHA can protect workers' families from workplace contaminants if workers are required to reside in employer-provided housing as a condition of employment. OSHA has extensive authority, however, to require that workers not carry home workplace contaminants on their clothing, in their automobiles, or by other means (i.e., by promulgating standards that require workers to remove contaminants from their skin and clothing prior to leaving the workplace). OSHA consultations conducted pursuant to 29 U.S.C. 670 also may promote, indirectly, prevention of take-home contamination.

The OSH Act does not provide specific authority to the National Institute for Occupational Safety and Health (NIOSH) to conduct studies on family protection from workers' home contamination. To the same extent that OSHA regulations and actions intended to protect workers also help assure that families are protected, however, NIOSH research assessing work hazards enables the agency to identify the potential for home contamination and make preventive recommendations. While NIOSH has no specific legal authority to evaluate conditions in workers' homes, the agency can conduct such studies with the cooperation of workers and their families.

The Federal Mine Safety and Health Act of 1977 (Mine Act) provides the Mine Safety and Health Administration (MSHA) with authority comparable to OSHA's. The Mine Act also authorizes MSHA to regulate home contamination if the mine is solely owned and operated by the miner.
The Environmental Protection Agency (EPA) has general authority under the Toxic Substances Control Act to regulate chemicals and to obtain information on the adverse effects of chemicals, thereby permitting EPA, at least indirectly, to prevent workers' home contamination. EPA has specific authority to prevent workers' home contamination under the Asbestos Hazard Emergency Response Act of 1986 and the Residential Lead-Based Paint Hazard Reduction Act of 1992.

Under the Federal Insecticide, Fungicide, and Rodenticide Act, EPA has broad authority to regulate the application and disposal of pesticides; EPA has used this authority, at least to a limited extent, to promulgate standards that prevent workers and farm owners from contaminating their homes with pesticides.

The Comprehensive Environmental Responses, Compensation, and Liability Act, and the Superfund Amendments and Reauthorization Act, authorize EPA and the Agency for Toxic Substances and Disease Registry to regard workers' contaminated homes as hazardous-waste-release sites, thereby allowing these agencies to take those measures necessary to decontaminate workers' homes and to control the sources of home contamination.

STATE LAWS

Thirty States, and Puerto Rico, responded to requests from NIOSH for information regarding State laws on this topic. These respondents indicated they had no laws currently in force relating directly to the protection of workers' families. Some of these States identified laws requiring that cases of elevated blood lead and pesticide poisoning be reported to a State agency, as well as laws addressing work practices at hazardous-waste sites and during emergency responses to the release of hazardous substances. Examination of occupational safety and health laws of States with OSHA-approved occupational safety and health programs did not find any laws that were more stringent than the commensurate Federal laws. Extension of occupational safety and health laws to State and local government employees in these States, however, provides added protection to their family members that is not available in States without OSHA-approved programs.

BACKGROUND

The first laws addressing workers' home contamination were enacted in England between 1903 and 1911 [Oliver 1914]. The purpose of these laws was to prevent workers from exposing their families to lead dust that was deposited on the workers' clothing during the work process. Enactment of these laws occurred after Oliver observed lead poisoning among workers' wives, and attributed this poisoning to the wives being exposed to lead while washing their husbands' work clothes. The following industries or work activities were subject to these laws: manual file cutting; manufacturing batteries, paints and colors, decorative pottery, and lead-containing compounds; heading lead-dyed yarn; and smelting lead-containing materials. The laws required employers to provide their
workers with clean work clothes (that employees were mandated to wear), rooms for donning and removing work clothes, and facilities for storing work clothes; employers also had to communicate and enforce prohibitions against removing work clothes from the workplace.

In the United States, a variety of statutes and rules currently provide some protection against workers’ home contamination. This review found only two statutes that specifically addressed protection of workers’ families. These two statutes are the:


Details of these statutes are discussed below under the appropriate statute headings.

The remaining federal statutes that permit agencies to promulgate requirements addressing prevention of workers’ home contamination, as well as remedial actions to be taken should such contamination occur, include the:


The sections of these statutes related to workers’ home contamination are presented in Table 17; this table also lists citations to the case law associated with these sections. The following discussion provides a summary of the manner in which each statute has been used to promulgate rules that address workers’ home contamination.

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3Note that this case law will not be discussed extensively in this review.
REVIEW OF RELEVANT FEDERAL STATUTES AND RULES

Occupational Safety and Health Act of 1970 (OSH Act)
The purpose of the OSH Act is to protect workers while they are at their place of employment. Workers' home contamination has been addressed only in a limited fashion by the two principle agencies established under the OSH Act, the Occupational Safety and Health Administration (OSHA, responsible for the promulgation and enforcement of occupational safety and health standards) and the National Institute for Occupational Safety and Health (NIOSH, responsible for research needed to identify and prevent occupational safety and health problems).4

OSHA. In general, OSHA appears to have limited authority under the OSH Act to develop and promulgate standards for protecting workers' families directly; under this authority, OSHA can protect workers' families from workplace contaminants if workers are required to reside in employer-provided housing as a condition of employment. This determination was made by a federal appellate court in Frank Diehl Farms v. Secretary of Labor. In this case, the employer (i.e., Frank Diehl Farms) provided employees, who were seasonal workers, with temporary housing while they were harvesting vegetables; employee use of this housing was voluntary. The housing was readily available, and little or no rent was paid by the employees for the housing; nevertheless, some employees chose to stay elsewhere. On inspecting this housing, OSHA cited the employer under the standard that regulated temporary labor camps; this standard is codified at 29 CFR 1910.142. This enforcement action was based on an OSHA instruction interpreting the standard to apply to any housing provided by employers to employees, whether or not employee use of the housing was voluntary. The federal appellate court that reviewed this case, however, interpreted OSHA's authority differently. Finding that the term "workplace" is commonly and ordinarily defined as "the place where one must be in order to do his job," the court held that OSHA could enforce this standard "only if company policy or practical necessity force workers to live in employer provided housing . . . ." This decision, therefore, implies that OSHA could cite an employer for contamination of workers' homes only if these workers were forced to reside in the housing as a condition of employment or because no reasonable alternative housing was available.5

4Section 20(a)(6) of the OSH Act authorizes NIOSH to conduct health hazard evaluations. This section requires that HHEs be performed after NIOSH receives "a written request by any employer or authorized representative of employees...." Requests submitted by individual employees, members of their families, or other parties do not satisfy this requirement.

5While injury or death to employees resulting from workplace hazards usually are addressed under state or federal workers' compensation systems, family members who suffer health effects associated with workers' home contamination may, under some circumstances, bring tort actions against culpable employers through the appropriate state or federal courts. Even children who have been injured in utero as a result of their parents' exposure to workplace hazards have brought negligence claims against the responsible employers (see, for example, Widera v. Ettco Wire & Cable Corp. [1994] and Agnew-Watson v. County of Alameda [1994]; the outcome of these cases, however, has been mixed, and appears to depend in large part of the
Despite the enforcement limitations implied by the decision in Diehl, OSHA has been successful in promulgating several standards that serve, indirectly, to protect workers' families from take-home contamination. These rules include the substance-specific standards for asbestos, lead, arsenic, and cadmium. While these standards contain provisions that reduce workers' home contamination, the specific purpose of these provisions is to prevent excessive worker exposure to these contaminants (i.e., employers need not comply with these provisions unless workplace contamination exceeds permissible exposure limits). These standards require, in part, that employers clean or replace contaminated work clothes periodically, train workers to handle and store contaminated work clothes properly, and provide shower and washing facilities for employee use after each work shift. (See Table 18 for a detailed listing of these requirements. Note, however, that these decontamination requirements do not apply to the hundreds of hazardous substances regulated by OSHA under 29 CFR 1910.1000.)

Several of these standards recognize the importance of preventing workers' home contamination. For example, a statement in the preamble to the cadmium standard [57 Federal Register 42349 (1992)] notes that "wearing contaminated street clothing outside the worksite would lengthen the duration of the employee's exposure and could cause cadmium to accumulate in employees' cars and homes, exposing other individuals to the hazard." Later, at 57 Federal Register 42350, the preamble mentions that, to prevent this contamination, the cadmium standard requires employees "to change out of work clothes, which are then segregated from their street clothes, to shower before leaving the plant, and to leave work clothing at the workplace, [which] significantly reduces the movement of cadmium from the workplace." The purpose of this provision is to limit additional worker exposure to cadmium and to "provide added protection to employees and their families."

Statements in the preamble to OSHA's asbestos standards [59 Federal Register 49964 and 41012 (1994)] recognize the hazard of asbestos to workers' family members, noting that "studies have documented that in the past workers have brought asbestos contaminated clothing home with them and thereby caused exposure and asbestos-related disease among family members." OSHA found that this situation warranted "special consideration"; therefore, the asbestos standards for construction and shipyards [29 CFR 1926.1101 and 29 CFR 1915.1001, respectively] require that employers control the release of asbestos particles from contaminated work clothing using several different procedures depending on the type (or class) of work being performed by the employees.

OSHA's lead standards [29 CFR 1910.1025, 29 CFR 1915.1025, and 29 CFR 1926.62] have similar hygiene requirements. Appendix B of these standards, entitled "Employee Standard Summary," advises that "contaminated work clothing or equipment must be

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willingness of a court to recognize a child's claims as independent from any cause of action that could be asserted by a parent.
removed in change rooms and not worn home or you will extend your exposure and expose your family since lead from your clothing can accumulate in your house, car, etc."

The OSHA standards addressing hazardous-waste operations and emergency response [29 CFR 1910.120, and 29 CFR 1926.65] have requirements for wearing protective clothing, decontaminating employees, and decontaminating or disposing of contaminated clothing and equipment before leaving the worksite. These standards, however, contain no specific advisory information regarding contamination of workers’ automobiles and homes.

OSHA’s hazard-communication standards [29 CFR 1910.1200, 29 CFR 1915.1200, and 29 CFR 1926.59] require that employees be advised about hazards with which they work. While not required by OSHA under these standards, information on workers’ home contamination could be included in Material Safety Data Sheets that accompany hazardous chemicals, and could be incorporated as well into employee training.

NIOSH. NIOSH’s research authority to study occupational safety and health hazards [29 U.S.C. 669] enables the agency to identify instances for which the potential for home contamination exists, and to make preventive recommendations regarding this problem. NIOSH also is authorized to recommend safety and health standards to OSHA [29 U.S.C. 671(c)]. Operating under the same limitations as OSHA, the basis of NIOSH-recommended standards must relate directly to the protection of workers, but the recommendations may be equally effective in protecting the workers’ families. Under this authority, the agency compiled information regarding workers’ home contamination in NIOSH criteria documents for asbestos [NIOSH 1972a, 1977], beryllium [NIOSH 1972b], and mercury [NIOSH 1973]). In a recommended standard on the manufacture and formulation of pesticides, NIOSH recommended that work clothing not be worn or taken home to be laundered, and that the clothing be laundered by the employer [NIOSH 1978].

Federal Mine Safety and Health Act of 1977 (Mine Act)

The Mine Act at 30 U.S.C. 802 and 803 provides indirect authority for preventing workers’ home contamination. These sections require that an employer who also is an employee in his/her workplace (i.e., an owner-miner of a mining operation) must comply with applicable safety and health rules developed under the statute by the Mine Safety and Health Administration (MSHA). If a mine, for example, is located on the owner’s home/family property, and the owner is the only miner involved in extracting minerals from the mine, the owner must provide himself/herself with the safety and health measures prescribed by the appropriate MSHA rules. Under these circumstances, compliance with the MSHA rules will reduce exposure of the owner-miner’s family to mineral dusts and other mining contaminants.

Having more general implications for the protection of miners’ families, § 811 of the Mine Act grants MSHA authority to establish rules to protect miners from exposure to
toxic substances, and specifically grants MSHA the authority to establish rules addressing suitable protective equipment. As with OSHA standards, these rules can ultimately protect both miners and their families.

Additional protection against home contamination by miners is provided implicitly in § 877 of the Mine Act. This section authorizes MSHA to require that employers make sanitary and bathing facilities available at the worksite for use by miners in removing mining-related contaminants; also, these facilities must be adequate for miners to change and store their work clothes between work shifts. These requirements have been incorporated into regulations for coal mines [30 CFR 71.400-404 and 30 CFR 75.1712].

NIOSH has authority under § 951 of the Mine Act to conduct research on the health effects of exposure to mining operations, and to make preventive recommendations.

**Toxic Substances Control Act (TSCA)**

The Environmental Protection Agency (EPA) has extensive authority under TSCA to regulate chemical hazards. Under 15 U.S.C. 2604 and 2605, EPA can regulate the manufacturing, processing, use, distribution in commerce, and disposal of new and existing chemicals, respectively. While another section, 15 U.S.C. 2682, does not refer explicitly to the hazard of take-home lead to workers' families, paragraph (a)(1) of this section directs EPA to "promulgate final regulations governing lead-based paint activities to ensure that individuals engaged in such activities are properly trained, that training programs are accredited, and that contractors engaged in such activities are certified." This provision states further that "[s]uch regulations shall contain standards for performing lead-based paint activities, taking into account reliability, effectiveness, and safety." A later provision of this section requires a "Study of Certification," and states that "[t]he Administrator [of EPA] shall conduct a study of the extent to which persons engaged in various types of renovation and remodeling activities in target housing...are exposed to lead and create a lead-based-paint hazard on a regular or occasional basis." A subsequent paragraph of this section requires that regulations promulgated under paragraph (a)(1) of this section be amended as appropriate using the results of such a study. Should such a study find hazards to the health of workers' families resulting from the workers' lead-based paint activities, EPA is obligated to establish training and certification requirements to reduce or eliminate the risk of injury to these families.

The following provisions of the TSCA also are useful in reducing the risk of workers' home contamination: 15 U.S.C. 2604 (requiring an evaluation by EPA of the health and environmental effects of new chemicals, and of significant new uses of existing chemicals, prior to the manufacture, or new use, of these chemicals); 15 U.S.C. 2605(a) (mandating that chemical manufacturers and processors provide notice of unreasonable risk of injury resulting from their chemicals); 15 U.S.C. 2607(c) (providing that chemical manufacturers, processors, and distributors maintain records of significant, adverse health effects resulting from chemicals for which they are responsible); and 15 U.S.C. 2607(e) (imposing on chemical manufacturers, processors, and distributors a duty to report
immediately information that a substance or mixture for which they are responsible presents substantial risk of injury to health).

Under 15 U.S.C. 2605(a) (i.e., the provision of the TSCA granting EPA authority to regulate hazardous-chemical substances and mixtures), EPA promulgated a standard [40 CFR 763.121] that prescribes full-scale decontamination procedures following asbestos-abatement actions performed by state and local government workers; these asbestos-decontamination procedures are somewhat abbreviated for workers involved in small-scale, short-duration asbestos-abatement actions. Among the asbestos-decontamination procedures specified under this standard is a requirement that workers wear protective clothing, and that this protective clothing be handled appropriately to avoid release of asbestos fibers; this standard, therefore, indirectly prevents asbestos contamination of workers’ homes.

Asbestos Hazard Emergency Response Act of 1986

This act contains a provision [15 U.S.C. 2646(b)(1)(B)(xi)] that addresses, explicitly, workers’ home contamination. This provision requires implementation of "[h]ousekeeping and personal hygiene practices, including the necessity of showers, and procedures to prevent asbestos exposure to the employee’s family." Additionally, this act specifically requires that state plans for accrediting asbestos-removal contractors contain procedures to prevent asbestos contamination, including contamination of an employee’s family. This requirement was implemented by EPA under Appendix C to 40 CFR part 763, subpart E.

Residential Lead-Based Paint Hazard Reduction Act of 1992

This Act has several provisions that indirectly protect workers’ families from lead-contaminated dust in their homes. These provisions include the development of a health-based standard for lead-contaminated household dust, development of a comprehensive, lead-exposure abatement program, and studies of the sources of lead exposure among children, including the occupational contribution to this exposure. In addition to the sections of this act presented in Table 17, the following sections may be considered relevant to preventing workers’ home contamination: 15 U.S.C. 2682(a); 15 U.S.C. 2682(c)(2); and 15 U.S.C. 2682(c)(3).

Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)

Under 7 U.S.C. 136(w), EPA has broad regulatory authority to establish standards that prevent, indirectly, contamination of workers’ homes. This section authorizes the promulgation of standards "with respect to the package, container, or wrapping in which a pesticide or device is enclosed for use or consumption, in order to protect children and adults from serious injury or illness resulting from accidental ingestion or contact with pesticides or devices regulated by this subchapter, as well as to accomplish the other purposes of this subchapter." Using this authority, EPA has promulgated a number of
rules that protect workers' families. These standards include: Child Resistant Packaging [40 CFR 157]; Certification of Pesticide Applications [40 CFR 171]; Labeling Requirements for Pesticides and Devices [40 CFR 156]; and the Worker Protection Standard [40 CFR 170].

The provisions of the Worker Protection Standard contain requirements that, at least indirectly, prevent workers' home contamination. The principal purpose of this standard is to protect workers from exposure to pesticides that are used during normal pesticide operations by the agricultural, nursery, greenhouse, and forestry sectors; with regard to the agricultural sector, this standard also requires prevention of accidental exposure of workers and other persons to pesticides. The phrase "other persons" would include family members (of both workers and the owners of agricultural establishments) who may be in the vicinity of pesticide operations. Another provision of this standard [40 CFR 170.112] requires owners of agricultural establishments to prevent workers from entering pesticide-treated areas until the pesticides have dissipated from these areas. This provision also requires that protective clothing be: worn by workers while applying pesticides; cleaned daily after use according to clothing manufacturers' instructions and instructions provided on pesticide-product labels; cleaned separately from other clothing; and stored, after cleaning, away from contaminated areas and separately from other clothing. In addition, those who launder protective clothing must be informed of the: pesticide-contamination problem; harmful effects of pesticide contamination; correct methods of handling and cleaning protective clothing; and procedures to use in protecting themselves from contamination. While the requirements of this provision could, indirectly, do much to prevent workers' home contamination, prevention is incomplete because the owners of agricultural establishments are not required to provide this protection for themselves or members of their immediate families; the standard does, however, encourage them to do so.

The Certification of Pesticide Applications standard [40 CFR 171] requires certification of pesticide applicators, including farm owners and farm workers, who apply restricted-use pesticides; these applicators must be certified for each restricted-use pesticide they apply. To be certified, these applicators must be able to read and understand the pesticide-product label, and have practical knowledge of the correct use, storage, handling, and disposal of pesticides and pesticide containers. Full compliance with these certification requirements would do much to prevent workers' home contamination.

The provisions of 40 CFR part 165, authorized under FIFRA at 7 U.S.C. 136(q), address specifically the storage and disposal of pesticide containers. If these provisions were properly implemented, the incidence of child poisoning resulting from improper disposal

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The Labeling Requirements for Pesticides and Devices standard requires that labels on pesticide containers provide information regarding worker protection. These labels, however, are not required to prescribe the decontamination procedures to be used on protective clothing. Shirts, short pants, shoes, and other items of ordinary work clothing are not considered protective clothing and, therefore, are not subject to these requirements.
of pesticide containers, a serious problem in the agriculture sector, would be substantially reduced.

Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) and Superfund Amendments and Reauthorization Act (SARA)

CERCLA was enacted in 1980 in response to concerns by Congress regarding hazardous-waste disposal problems [Ways and Means Committee 1980]. These concerns involved sites that contained large quantities of hazardous wastes, unsafe hazardous-waste disposal practices, and the substantial dangers to health and the environment resulting from improper hazardous-waste disposal. A primary purpose of SARA, the 1986 Superfund Amendments, was to decontaminate abandoned hazardous-waste sites and leaking, underground-storage tanks that present the most serious public health and/or environmental hazards [Energy and Commerce Committee 1986].

The primary purpose of CERCLA is to address major hazardous-waste issues and to protect the public from emergency releases of hazardous substances; however, as discussed by Zirschky et al. [1987] and Zirschky [1990], several sections of CERCLA and SARA provide, at least indirectly, protections against workers’ home contamination (Table 17). These protections are inferred from the: broad definitions of "facility," "hazardous substance," "release," and "pollutant or contaminant"; and the authority assigned to EPA and to the Agency for Toxic Substances and Disease Registry (ATSDR) to designate hazardous substances, respond to releases of hazardous substances, and perform health assessments near hazardous-waste sites.

Under the CERCLA provisions, two occupational groups have been identified that are at enhanced risk of workers’ home contamination. The first occupational group consists of workers who remediate (i.e., "clean up") hazardous-waste sites; this workforce is both large and highly mobile. Remediation work involves exposure of workers’ protective clothing to chemical contaminants, and these contaminants can be carried to workers’ homes unless adequate workplace safeguards are in place. The second occupational group is composed of chemical-emergency responders. ATSDR data indicate that chemical emergencies are frequent, and often result from industrial-plant mishaps, transportation accidents, and improper disposal of chemicals. Emergency responders can transport chemical contaminants into the home unless clothing changes and other safeguards are effected.

The worker protection standards [29 CFR 1910.120 and 40 CFR 311] required under § 126 of SARA have provisions for changing and decontaminating work clothing, providing change rooms, and showering before leaving a hazardous-waste site. Adherence to these requirements should prevent home contamination by hazardous-waste workers.

Constraints incorporated into CERCLA reduce the likelihood that incidents of workers’ home contamination will be addressed extensively by either EPA or ATSDR. These constraints include: in responding to hazard-waste releases, the highest priorities must be
assigned to releases that present the largest public health threat [42 U.S.C. 9604]; and specification of strict criteria for determining these priorities [42 U.S.C. 9605]. Criteria to be considered in determining these priorities are the: population at risk; harmful effects of toxic substances located at hazardous-waste sites; likelihood that these substances have contaminated, or will contaminate, drinking water; and the potential for direct human contact with these substances.

Under these provisions, the following rules have been promulgated: 40 CFR 300, which resulted in the National Priorities List consisting of high-priority, hazardous-waste sites; and 40 CFR 302, which specifies hazardous substances and the reportable-release quantities of these substances (i.e., quantities of hazardous substances that, if released, may be harmful to public health and/or the environment and must, therefore, be reported to the National Response Center). While these constraints may limit EPA and ATSDR in prevention and control of workers’ home contamination, recent case law indicates that State and Federal courts may provide an alternate vehicle for addressing this issue under CERCLA and SARA.\(^7\)

Although decontamination of workers’ homes is not addressed directly under any of the above-mentioned rules, some hazardous-waste sites (Alaska Battery Enterprises, Anderson Development Company, and Borfos Nobel, Inc.) designated on the National Priorities List have been identified as sources of workers’ home contamination. As EPA and ATSDR conduct studies at other sites on the National Priorities List, additional cases of workers’ home contamination may be found. Many of the chemicals noted in previous chapters as being involved in workers’ home contamination are on the priority list of hazardous substances [40 CFR 302], and these chemicals typically have low reportable-release quantities (i.e., 1-10 pounds).

REVIEW OF STATE LAWS

NIOSH requested information (Appendices 2 and 3) on State and local laws that were relevant to preventing workers’ home contamination. The responses from State agencies to these requests are summarized in Tables 20 and 21. Agencies from 30 States and Puerto Rico responded to this request; these responses also are summarized in Tables 20 and 21. Eleven States and Puerto Rico replied that no statutes or rules related to workers’ home contamination currently are in force. Arizona, California, and Idaho reported that laws existed for reporting elevated blood-lead levels and/or pesticide poisonings. Michigan, Maine, and Pennsylvania reported on laws, similar to CERCLA, that regulated hazardous-waste sites and the emergency response to hazardous-chemical releases. Oregon wrote that, while no laws or regulations dealing directly with workers’

\(^7\)In Vermont v. Staco, Inc. [1988], the court awarded the plaintiffs (the state of Vermont and the village of Poultney) nearly $74,000 in damages for decontamination costs incurred by the plaintiffs in responding to a release of mercury from the defendants’ thermometer-manufacturing facility into Poultney’s sewer system. In finding the defendants liable, the court stated that “the defendants released mercury to the environment through the movement of workers to and from the...facility in [the village of] Poultney.”
home contamination had been enacted, many occupational safety and health regulations had been promulgated that help, indirectly, to prevent this problem.

About half of the respondents have occupational safety and health programs approved by OSHA. To be approved, a State’s occupational safety and health laws have to be at least as protective of workers as the Federal laws. To determine whether any of these laws had requirements that are more stringent than the commensurate Federal OSHA regulations, the occupational safety and health laws of States with OSHA-approved programs were obtained and evaluated. Most of the State laws were identical to the Federal OSHA regulations with regard to workers’ family protection; unlike the Federal OSHA regulations, however, the State laws apply to State and local government employees. Thus, in States that have OSHA-approved programs, the families of government employees have benefitted indirectly from provisions of the arsenic, asbestos, cadmium, and lead standards requiring that workers shower and change clothes before leaving the workplace; in contrast, the families of State and local government employees in the remaining States may not have benefitted from these requirements because compliance by these State and local governments is voluntary.
CHAPTER 7. RESPONSES TO INCIDENTS OF HOME CONTAMINATION

CHAPTER SUMMARY
In this Chapter, responses of Federal and State agencies and industry to incidents of workers' home contamination are reviewed. NIOSH found that several Federal agencies have responded to incidents of workers' home contamination, often working together or working with State or local governmental agencies. These responses by Federal agencies have resulted in identification of workers' home contamination that otherwise would have not have been known, decontamination of workers' homes and recommendations for instituting workplace changes that would prevent further contamination. In several cases, Federal agencies have referred incidents to State or local health departments for follow-up actions.

A number of State agencies have also investigated incidents of workers' home contamination, made referrals to Federal agencies for follow-up actions and made recommendations for workplace improvements to prevent further contamination of workers' homes.

In some instances where States reported they had no information on home contamination investigations or incidents, such reports were found in the literature. Likewise, for some States that did not respond to inquiries, reports were found in the literature of investigations that took place in the State. Often, in these instances, the investigations were conducted by local health departments collaborating with Federal agencies and reported in journal articles or CDC's Morbidity and Mortality Weekly Report (MMWR).

Only a few responses of industry to incidents of workers' home contamination were found. However, these reports indicate how industry can contribute to prevention by informing workers of hazards, as well as by taking specific actions to correct situations where workers' families are at risk or by use of preventive measures.

RESPONSES OF FEDERAL AGENCIES
In this section, responses of Federal agencies to incidents of workers' home contamination are reviewed. The information for this section, compiled in Table 19, was derived from published reports and responses to requests for information by NIOSH.

OVERVIEW
Agencies that have responded to incidents of workers' home contamination include: (1) the Centers for Disease Control and Prevention (especially the National Center for Environmental Health and the National Institute for Occupational Safety and Health); (2) the Occupational Safety and Health Administration; (3) the Mine Safety and Health Administration; (4) the Department of Energy; (5) the Environmental Protection Agency; (6) and the Agency for Toxic Substances and Disease Registry. In many cases, Federal, State, and local agencies collaborated on the investigations. In all
of the incidents in which OSHA was involved, State or local health departments were also involved by either notifying OSHA of probable home-contamination cases or by being informed by OSHA of such cases. Several of the investigations conducted by CDC and ATSDR have also been in collaboration with agencies of State or local governments. These Federal agency investigations and responses to incidents of workers’ home contamination have resulted in:

- Recommendations for instituting changes in industrial hygiene practices to prevent further home contamination.
- Decontamination of workers’ homes.
- Identification of workers’ family members who have been exposed to or poisoned by toxic substances introduced into the home from the workplace.

Centers for Disease Control and Prevention (CDC)
The Centers for Disease Control and Prevention has conducted about 20 studies on incidents of workers’ home contamination; about half of these have been conducted by NIOSH, and the rest by other Centers, especially the National Center for Environmental Health. NIOSH has further identified potential for workers’ home contamination in a number of its evaluations of individual workplaces and made recommendations for improved industrial hygiene measures for its prevention. CDC has also presented information to Congress on issues of home contamination and published reviews to assist professionals that may be confronted by cases of workers’ home contamination and resulting family poisonings.

National Center for Environmental Health (NCEH)
The mission of NCEH is to prevent and control disease and disability related to the interactions between people and their environment outside of the workplace. The Center’s applied research has evaluated incidents of workers’ home contamination by lead, pesticides, arsenic, tin, and PCBs.

Lead. Three studies [Baker et al. 1977; Landrigan and Baker 1981; Matte et al. 1991] were conducted by NCEH on families of lead smelters workers. These studies found that homes of exposed workers had higher concentrations of lead than controls and that family members BLLs were elevated. Following the study by Baker et al. [1977] the homes were cleaned and workers showered and changed clothes before going home.

Families living in contaminated homes of battery plant workers were found to have elevated BLLs in four studies [Watson et al. 1978; Dolcourt et al. 1978; Dolcourt et al. 1981; Matte et al. 1989]. Following the studies by Dolcourt et al. [1984], home decontamination was undertaken.
Kaye et al. [1987] found elevated BLLs among children of workers exposed to lead in a plant manufacturing electrical components and made recommendations for taking preventive measures in the workplace.

Novotny et al. [1987] studied the BLLs of firing range workers and their spouses, finding elevated levels in the workers but not in the spouses.

In an ongoing study of workers exposed on the firing range of the FBI Academy, the workers' vehicles have been found to be contaminated with lead; however, lead dust levels in the workers' homes and BLLs among the workers' children were low, suggesting that the children were not being exposed to significant amounts of lead [Briss 1994].

In March, 1993 NCEH and NIOSH collaborated on an exposure assessment for heavy metals associated with a smelter in Oruro, Bolivia. The investigators evaluated biological and environmental samples for lead, arsenic, antimony, and tin. Both environmental testing and biological monitoring suggested that workers' homes were contaminated by tin. However, the biological results were not elevated to levels documented to cause adverse health effects [Briss 1994].

Other. In a study of a community that had used sludge contaminated with PCBs, Baker et al. [1980] found higher levels of PCBs in the blood of family members of sewage treatment workers than in other members of the community.

Falk et al. [1981] reported on a case of angiosarcoma in a young girl whose father worked with arsenic and wore contaminated clothing home.

Wolfe et al. [1961] studied cases of pesticide poisoning in children and made recommendations for pesticide applicators that included decontamination of empty drums and clothing.

A joint study by CDC and EPA [Canon et al. 1978], found that wives of workers' exposed to kepone had signs of kepone poisoning. These women had washed their husbands' clothes.

National Institute for Occupational Safety and Health (NIOSH) NIOSH conducts evaluations of health hazards in the workplace. About 40 NIOSH studies have addressed potential or actual incidents of workers' home contamination.

Asbestos. Five investigations of potential workers' home contamination by asbestos were reported by NIOSH. These investigations were made at: a construction site [Lemen 1972]; a plant manufacturing flooring material [Belanger et al. 1979]; a plant manufacturing friction products [Seixas and Ordin 1986]; a chemical plant [Driscoll and Elliott 1990] and a brake-service facility [Godby et al.
In each case it was determined that the potential existed for workers to bring asbestos home on their clothing as a result of inadequate or inconsistently applied industrial hygiene practices. In two cases [Seixas and Ordin 1986; Driscoll and Elliott 1990], asbestos was detected on the workers' clothes as they left the worksite. In an evaluation of a construction site [Lemen 1972] and a brake-service facility [Godby et al. 1987], NIOSH found that most workers did not change clothes before leaving work and that their work clothes were laundered at home. In all of these studies, recommendations were made that would prevent workers' home contaminations such as reducing exposures at work and leaving contaminated clothing at work.

**Lead.** Five investigations of exposure of workers to lead and potential home contamination were reported by NIOSH: stained glass manufacturing [Landrigan et al. 1980]; battery manufacturing and recycling facilities [Apol and Singal 1980; Matte and Burr 1989; Gittleman et al. 1991]; tank lining [McCammon et al. 1991; CDC 1992a]; gold assaying [Gunter et al. 1987]; and building renovation [Kiefer 1994]. In a study by Matte and Burr [1989] of back-yard battery repair shops, contamination of the homes and elevated BLLs of family members were found. Elevated BLLs of stained-glass workers' families were related to occupational exposures of the worker [Landrigan et al. 1980]. Lead was detected in workers' cars by McCammon et al. [1991], indicating a potential for transfer to the home. In these reports, improved hygiene practices were recommended to prevent contamination of homes.

**Pesticides.** Kominsky [1984c] studied the contamination of firefighters protective clothing by malathion and diazinon following a fire and made recommendations for laundering the contaminated clothing.

**Chlorinated Hydrocarbons.** Investigations of two cases involving PCBs, one a manufacturing plant and one a railroad yard, resulted in industrial hygiene recommendations to prevent transportation of the contaminant from the workplace [Hartle et al. 1987; Hartle 1987]. Several studies addressed instances in which firefighters' clothing was contaminated with PCBs [Kominsky 1984a, 1984b, 1987a, 1987b; Kominsky and Singal 1987; Orris and Kominsky 1984; Seligman 1984]. The reports provided recommendations for laundering clothing and using protective clothing.

**Mercury.** During the health hazard evaluation of a thermometer plant in Vermont, the NIOSH trailer where workers received medical tests became contaminated with mercury, suggesting that contamination of the workers' homes with mercury was possible [Ehrenberg 1986]. Since the plant was closed soon after the study, industrial hygiene recommendations were not included in the report.

**Estrogenic Substances.** Children of chemical-plant workers who contaminated their homes had enlarged breasts due to zeranol, an estrogenic animal growth
promoter. Zeranol was brought home on contaminated work clothing. NIOSH made recommendations to prevent home contamination [Aw et al. 1985]. NIOSH also participated in a study on poisoning of farm children by diethylstilbestrol [Bierbaum 1993].

Agency for Toxic Substances and Disease Registry (ATSDR)
The Agency for Toxic Substances and Disease registry has conducted several studies of hazardous waste sites that are relevant to workers' home contamination. An investigation of a chemical manufacturer in Adrian, Michigan showed detectable levels of MOCA in the urine of workers' families. The workers' homes were contaminated [ATSDR 1989a, 1990b, 1993b] by MOCA that may have been tracked out of the workplace on the employees' clothing and shoes. The homes were decontaminated.

An investigation at the Bofors-Nobel, Inc. Company in Michigan found 3,3'-dichlorobenzidene contamination in workers' homes and 3,3'-dichlorobenzidene in the urine of some workers and family members [ATSDR 1991b].

Other ATSDR reports describe investigations of contamination by MOCA [ATSDR 1989b], lead [ATSDR 1991a], and mercury [ATSDR 1990a]. In other cases, attempts by ATSDR to evaluate workers' home contamination failed because of inadequate participation by workers' families [ATSDR 1993a, Alabama Department of Public Health 1991].

Occupational Safety and Health Administration (OSHA)
Although in general OSHA's jurisdiction is limited to the workplace, through interactions with State and local health departments some reports on workers' home contamination have resulted from its investigations. In addition, a recent review article [McDermid and Weaver 1993] addresses issues of poor industrial hygiene, cottage industries, and physician awareness as they relate to workers' home contamination.

[Natarajan 1994] described an OSHA investigation of a workplace in Texas after learning of high BLLs in a child. The child's father (who also had an elevated blood lead level) worked as a radiator repairman. Recommendations were made to prevent further contamination.

The local health department in Kane County, Illinois referred a case to OSHA of gross lead contamination of a home [Wiehrdt 1994]. Two children were hospitalized and chelated for lead poisoning. OSHA conducted a comprehensive inspection of the battery plant where the father worked and found that he was bringing lead home on his clothing.

OSHA suspected a potential home contamination problem while inspecting a local plant in 1984 in Indianapolis, Indiana and referred the potential problem to the
county health department [Wiehrdt 1994]. Subsequent investigation by the county health department determined that at least one of the children had a BLL of 50 μg/dL.

In 1990, the Cleveland area OSHA office investigated a company where it was determined that employees were being exposed to lead [Wiehrdt 1994]. Learning of three employees whose children had elevated BLLs, OSHA conducted sampling in the employees’ homes. Later, the case was reported to the Ohio Department of Health and the Cleveland Lead Hazard Abatement Center.

Mine Safety and Health Administration (MSHA)
MSHA has investigated two instances where workers inadvertently brought mercury into their homes and cars [Zalesek 1994]. In one case, the workers’ washers and dryers were the most heavily contaminated part of the homes. The company cleaned the homes.

Environmental Protection Agency (EPA)
The Environmental Protection Agency investigated, and decontaminated homes of workers contaminated by mercury in Tennessee [ERM-Southeast, Inc. 1989]. In another study supported by EPA, PCB contamination of workers’ homes was documented [Price and Welch 1972]. EPA has also conducted various investigations and remedial activities on homes contaminated by: dioxin [Ramsey 1987; MacDonald 1988; Doherty 1984; Hess 1988]; lead [Beegle and Forslund 1990; CHM Hill 1991]; and asbestos [Beegle and Forslund 1990]. Although these latter reports are not about homes contaminated by workers’ activities, they do provide protocols and information relevant to cleaning contaminated homes of workers.

Department of Energy (DOE)
Beginning in October 1990, the Department of Energy (DOE), under DOE order 5000.3A, required the reporting of any event which could "affect the health and safety of the public, seriously impact the intended purpose of DOE facilities, have a noticeable adverse effect on the environment, or endanger the health and safety of workers." In February 1992, this order was superseded by DOE order 5000.3B, with some modifications in reporting criteria. The requirements cover "events" related to radioactive as well as other hazardous materials and replaced a previous "unusual occurrence reporting system" instituted in 1984. The central DOE operational database containing all post-1989 occurrence reports is called ORPS (Occurrence Reporting and Processing System) and is maintained by the DOE Office of Nuclear Safety. There is no central repository of pre-1989 records or reports [Boyle 1994].

Both chemical and radiologic contamination incidents are covered by the DOE reporting policy. The database is not classified; if any of the reports involve classified information, a computer entry notes that there is a classified report, with the detailed description maintained in a classified hard copy file. The reported
incidents are summarized weekly in a publication prepared by the Nuclear Safety Office of DOE.

There are approximately 19,000 reports from 1990-present in the database. Since off-site contamination of a home is not uniquely coded; the use of word searches with ORPS can lead to under-counting of relevant cases of potential take-home contamination. In addition, there may be reports of workers' home contamination in the pre-1990 files which were not searched.

The ORPS Program Manager provided 16 reports related to contamination of workers' homes with hazardous substances transported from the workplace [Boyle 1994]. These reports primarily describe breaks in procedure or poor work practices with potential rather than actual take-home contamination, or with take-home activity that did not result in contamination of the workers' homes or family members. The three incidents involving possible contamination of workers' homes or family members include:

- Workers contaminated with thorium and protactinium while changing valves on cylinders, apparently ignored positive readings on contamination monitors, resulting in contamination of one employee's pillow case and shirt and another employee's shoe. The incident led to major revisions in the facility's monitoring program and contamination control procedures. Based on survey information and monitoring data, which indicated no internal contamination of the workers and "minute" external (skin) contamination, the incident was anticipated to have "negligible effect on the health of the workers or the public."

- An employee was found to have contaminated hands when monitored upon entering the facility; the employee had not gone through the monitoring process when exiting from work the previous night. Survey of the employee's home found that two items of personal clothing worn the previous day were contaminated. Levels of contamination were "extremely low" and there was felt to be no exposure to the employee's family. The employee and his clothes were decontaminated and the employee was terminated for "willful and flagrant disregard of health and safety procedures."

- Initially-undetected damage to an americium source resulted in contamination of a worker's hat, which was found on a routine survey several days after the event. A follow-up investigation identified americium on the diaper of a worker's infant child. A panel of independent experts from the national radiation dosimetry community, the radiological medicine community, and a local pediatrician guided the follow-up evaluation. The panel concluded, the most likely explanation was that this was a false positive because of poor laboratory performance. The poor laboratory performance was well-documented by the evaluators and no subsequent samples were sent to this laboratory. The team reviewing the incident
recommended more careful handling of and administrative controls for americium sources.

**Nuclear Regulatory Commission (NRC)**

NRC did not report cases of Agency responses to incidents of workers’ home contamination. However, it has established reporting requirements that identified cases of potential home contamination and responses of employers involved. The U.S. Nuclear Regulatory Commission has regulatory jurisdiction over byproduct material of reactors plus "special nuclear material" used as reactor fuels or bomb material. Users of radioactive materials falling under NRC jurisdiction include commercial nuclear power plants, university and hospital laboratories using radioactive materials, and industrial users of radiation sources. Twenty-nine States have agreements with NRC delegating to the States regulation of nuclear materials within their borders; NRC directly administers regulatory activities in the remaining 21 States.

Reporting regulations contained in 10 CFR 20 are intended to cover all significant incidents of off-site contamination, including contamination by radioactive material accidentally or intentionally brought home by workers.

NRC maintains two databases potentially containing reports of off-site contamination of workers' homes: the database of events called in to the NRC Operations Center (dealing mostly with reactor-related events) and the Non-Reactors Event Reporting (NRER) database, which is a compilation of significant Non-Reactor licensee reports that were originally sent to the NRC regions [Brockman 1993].

At the request of NIOSH, NRC personnel searched these two databases to identify events involving radioactive contamination brought home from the workplace. A search of NRC Operations Center data from 1985 to mid-September 1993 identified 34 incidents of off-site contamination; in seven of these, the brief reports directly address the possibility for take-home radioactive material.

- Contaminated hand tools were found in the home of a nuclear power reactor contractor’s home. One tool was radioactive, but no personnel or items in the contractor’s home were contaminated.

- A deliberate ingestion of uranium acetate was associated with contamination of the ingester's home.

- Four nuclear power plant contract workers contaminated their socks and shoes, which went initially undetected by monitor with potential contamination of a home and a hotel.

- Low-level contamination, initially undetected at a portal monitor, was discovered on clothing brought home by a power reactor worker.
• A worker in a fuel cycle facility was burned by a radioactive acid solution. Although no contamination was discovered during the worker's self-frisk before he was transported to the hospital, external contamination was subsequently detected on the acid burn areas.

• Low-level contamination was found on the accelerator pedal of a worker's vehicle during employee screening after detection of P-32 contamination in a laboratory.

• Four contract workers set off portal monitors when reporting to work at a nuclear power reactor for the first time.

No additional information is available for any of these reports.

A search of 1985-92 NRER data identified 80 contamination events resulting in off-site contamination. The reports generally lack detail, but those which raise the possibility of take-home contamination include:

• In the 1970s, in accordance with his employer's policy at the time, a worker used waste lumber from his workplace to construct a garage in his home. The employer manufactured catalysts containing depleted uranium. In 1991, following newspaper articles concerning radioactive contamination at the site, the employee contacted the State department of health. Surveys of the garage revealed contamination in excess of the NRC release criteria. The licensee replaced the garage. There were no reports of adverse health effects.

• Phosphorus P-32 was spilled in a university laboratory over a weekend; the spill was discovered when contamination was found on an individual's shoes. Contamination was found in the laboratory, in the building outside the laboratory, in at least one automobile, and on the shoes of about 40 individuals. Contaminated areas were isolated and cleaned up and all contaminated items were impounded and decontaminated. There were no reports of adverse health effects.

• A contamination event at a hospital resulted in contamination of a pharmacy truck driver, his truck, and a transport box. No additional information available.

• Radioactive sand from a Federal facility was disposed of in a septic tank on a farm. No additional information available.

• "Small areas of contamination" were found in a worker's residence. No additional information available.

• Contamination found at a residence recently vacated by the owner of a licensed laboratory. No additional information available.
RESPONSES OF STATE AGENCIES
Information on State agency investigations into incidents of home contamination was obtained in two ways. The first was by direct solicitation of various State agencies, including State agriculture and State and local health departments, State departments of labor, and State environmental departments. Responses received from these State agencies are compiled in Tables 20 and 21. The second way documentation of State investigations was obtained through literature searches in the open literature. Studies by State agencies identified in this way are compiled in Table 22.

The reports of the State agency responses to, or investigations of, home contamination incidents are discussed by groups: Lead; Pesticides; and Other.

Lead
Because of its wide use in a number of common industries, and particularly because of its serious neurological impact on children, the most commonly cited incidents of home contamination from the States involve lead. Many States maintain active surveillance of lead poisoning through local health departments and physician reporting and as a result can identify incidents for investigation.

In California, laboratories that analyze blood for lead content are required to report BLLs of 25 µg/dL or above to the California Department of Health Services and to the local county health department [Osorio 1994]. Programs in the California Department of Health Services, the Occupational Lead Poisoning Prevention Program and the Childhood Lead Poisoning Prevention Branch, coordinate investigations of elevated BLLs with local health departments. Take-home exposure cases are typically identified during the investigation of workers with high BLLs or follow-up of childhood cases where lead exposure is identified in the job of a parent or other household member. In terms of prevention activities, the Occupational Lead Poisoning Prevention Program includes information on take-home lead exposure in their outreach and educational efforts. Data from the Childhood Lead Poisoning Prevention Branch show that of the 1,232 cases of elevated BLLs in children under age 16 with follow-up information, 106 had a potential exposure in a worker's contaminated home. Of those with information reported about the lead workers in the household, 33% changed clothes before leaving work, 13% took showers before going home, and only 18% ever had a blood lead test for work.

The California Department of Health Services provided five case studies of take-home lead exposure in response to the NIOSH Federal Register request for information [Osorio 1994]. Industries involved in these cases include: lead recycler/bullet manufacturing; radiator repair; and cable cutting operations.

In the cable cutting operations case, the county health department conducted a follow up investigation of a 3-year-old child with a BLL of 28 µg/dL whose uncle, a lead cable cutter, for 4-5 years lived with the family. The father was also employed in this trade for 3 months prior to the investigation. Their employer did not provide
hand washing or showering facilities at the worksite. Work clothing was laundered at home, and a lead concentration of 1,700 ppm was found in a composite sample of dust in the home. In addition, the home's back yard was contaminated with lead from lead contaminated telephone poles which were brought from the worksite and stored in the back yard for firewood. The State industrial hygienist recommended and the company complied with the following preventive measures:

- test all workers blood lead levels;
- provide a medical monitoring program;
- provide a testing facility;
- provide protective clothing;
- provide worker training;
- provide an air monitoring program; and
- implement safe clean-up methods.

The Minnesota Department of Health investigated the potential for elevated blood lead levels among household contacts of employees of a lead smelter [Winegar et al. 1977]. Data gathered as a result of this study showed high levels of lead in the workers' clothing and hair and elevated blood lead levels in some children of the workers. This smelter reclaimed lead from old batteries. Home contamination incidents from similar battery manufacturing, recycling, or reclamation operations were investigated by health departments in several States:

- In a Tennessee case investigated by the State's Department of Public Health and the local county health department, 49% of the battery reclamation workers' children (50 of 102) had elevated BLLs equal to or greater than 30 μg/dL [CDC 1976]. The source for lead appeared to be the parents' contaminated clothing.

- Oklahoma investigators found similar results in an investigation of BLLs in children of battery manufacturing workers [Morton et al. 1982].

- In North Carolina, 72% of the children of battery plant workers had BLLs of 30 μg/dL or above [CDC 1977b].

- An investigation by the Alabama Department of Health in 1991 of a battery recycling operation revealed elevated BLLs in most of the workers. When the
local county health department measured the BLLs of the children of these workers, mean BLLs were 22.4 µg/dL [CDC 1992b].

These investigations resulted in chelation therapy for some victims, recommendations for improved hygiene practices, improved engineering controls, and, in one case, a court order to remove all workers from the workplace.

Reports of elevated BLLs in children of radiator repair workers include:

- The New York City Department of Health found that three of seven radiator repair workers' children had BLLs at or above 10 µg/dL; recommendations for industrial hygiene consultation were made and on-site educational programs were undertaken [Nunez et al. 1993];

- The Minnesota Department of Health investigated BLLs in radiator repair workers and their children and identified the need for worker education, safer work practices, better shop design, and better ventilation in the industry [Lussenhop et al. 1989];

Other cases investigated by States where elevated BLLs in children were attributed to the parents' occupation include:

- A Mississippi Department of Health investigation into lead contamination of a workers' home and made recommendations for changing shoes and clothes before entering the home, washing clothing separately, and cleaning the home [Pollock 1994];

- A capacitor and resistor plant in Colorado was investigated by a local health department and OSHA, finding workers and children with elevated BLLs resulted in enforcement of the OSHA lead standard to reduce exposures of the workers and their families [CDC 1985];

- A local health department in Colorado investigated a belt buckle, plaques, and awards manufacturer and assisted the company in reducing exposure of workers and their families to lead [CDC 1989b];

- The Iowa Bureau of Labor investigated a soil nutrient manufacturer whose raw material contained lead [Hooper 1991]. The employees were not required to shower or change clothes before leaving work. One workers' child was "tested for lead poisoning, the probable cause was washing work clothes with the child's clothes. Actions were taken to remedy the problem; and"

- A welder in Indiana whose family car was contaminated with lead and whose child had seizures was reported by the State's Department of Labor [Molovich
1991]. Action taken to prevent further contamination of the home included washing the worker’s car once per week and the company laundered his socks.

The New Jersey Department of Health [Stanbury 1994; Czachur et al. 1995] conducted a pilot study of home contamination by lead in 1992. Elevated BLLs (> 40 μg/dL) were identified in 98 persons through the State’s occupational lead registry and 45 were contacted, interviewed about their occupations and age of their homes, and offered free blood lead testing for their children. BLLs were obtained on 28 children from the families of 15 of these workers. Nine (32%) of these children had BLLs considered to be a potential risk for adverse health effects (> 10 μg/dL). The parents brought their work clothes home for laundering. Of six children whose parents did not bring their clothes home to be laundered, none had BLLs at 10 μg/dL or above.

Pesticides
Agricultural extension services in Arkansas [Lavy 1988; Huitink 1994], Florida [Anonymous 1994], Iowa [Stone and Wintersteen 1988], Louisiana [Finley et al. no date], Michigan [Branson and Henry 1982], and Nebraska [Easley et al. 1981a; Laughlin and Gold 1989c] have developed informational materials for farmers that advise on preventive measures. These advisories include:

- training courses for pesticide safety;
- brochures describing safe application practices;
- brochures on proper laundering techniques for clothes worn during application; and
- brochures on proper disposal of pesticides containers.

Other
A 1936 study by the Pennsylvania Department of Labor and Industry on the effects of hexachloronaphthalene and chlorodiphenyl (PCB) exposure of wire insulation workers attributed dermatitis in a young child to exposure from his father bringing home contaminated work clothes [Fulton and Matthew 1936].

Hardy [1948] (a physician with the Massachusetts Department of Labor), in a published paper on beryllium exposure and disease, cited a case of beryllium disease in the mother of a beryllium worker. The suspected source of beryllium was exposure to her daughter’s contaminated clothes. In the 1960’s, cases of beryllosis among workers’ family members were investigated by the Pennsylvania Department of Health [Lieben and Williams 1969].
A study published in 1978 by researchers with the New York Department of Health showed a correlation between mesothelioma in women and asbestos related employment of husbands and fathers [Vianna and Polan 1978].

Investigators from the Vermont State Department of Health studied home contamination and health effects in children of thermometer plant workers exposed to mercury [Hudson et al. 1985].

In California, the State Department of Public Health investigated mercury exposure and poisoning of cinnabar miners and mill workers [West and Lim 1968] and developed a document on how to prevent mercury poisoning [Anonymous 1968]. Although the investigators [West and Lim 1968] noted that home contamination with mercury brought home on workers' boots and clothes could increase workers' exposure to mercury, no mention was made of potential exposure of family members.

**RESPONSES OF INDUSTRY**

NIOSH has attained little information on how industry has responded to incidents of workers' home contamination. This section describes information from NIOSH studies (primarily those that were requested by employers) and from other reports, articles, and submissions by industry in response to the Federal Register Announcement requesting information (Appendix 2). They are tabulated in Table 23.

In a study requested by the owner of a stained glass studio attached to her home, NIOSH investigators determined that the exemplary industrial hygiene and housekeeping practices in use were adequate, since no lead was detected in the home [Donovan 1994a,b]. The owner requested the study because of concerns about exposures both in the home and the workplace.

In the case of a mercury thermometer plant where children of the workers were found to have been exposed to mercury, the plant voluntarily closed [Hudson et al. 1987]. Part of the plant re-opened when appropriate controls had been implemented. Another example of industry response occurred when an employee of a wood treating company brought home and spilled chloropicrin from a company truck, which made neighbors ill, and the company instituted a policy forbidding bringing company vehicles home [Barnett 1994].

Examples of educational material produced by industry (or their associations or trade magazines) involve lead. The Lead Industries Association, Inc. has produced brochures, flyers, and videotapes that warn of the dangers of lead exposure and provide advice on how to reduce these exposures and prevent workers' home contamination [LIA 1989; 1991; 1993a,b; 1994a,b]. Several of the videotapes are specific to particular hobbies or industries that involve the use of lead containing material. *Battery Man*, a trade magazine, published an article on protecting the families of workers involved in battery production [Lundquist 1980].
CHAPTER 8 - RECOMMENDATIONS AND CONCLUSIONS

Based on the information compiled and reviewed in this report a number of recommendations for research and education needed to prevent workers’ home contamination are identified in this Chapter. Also, a number of conclusions can be drawn from the report, as discussed below.

Recommendations for Research and Education
The prevalence of health effects of contaminants transported from the workplace need to be determined. One possible approach would be to conduct surveys among occupational and environmental medicine health care providers and clinics. Employment practices and controls that work best in preventing the transport of contaminants from the workplace to the home should be identified, and the special needs and problems of individuals who work in home or cottage industries need to be identified.

Educational programs to prevent home contamination should be developed for employers, workers, children, teachers, and parents, physicians, and other health professionals.

Conclusions
Workers’ home contamination may pose a serious public health problem. Health effects and deaths from contaminants brought home from the workplace have been reported in 28 countries and 36 States. The extent to which these health effects occur is not known because there are no information systems to track them, and physicians do not always recognize the occupational contribution to various common diseases. About half of the reports of health effects from home contamination are less than 10 years old. The literature on the health effects involved approximately 30 different substances or agents. The potential exists for many of the thousands of other chemicals used in commerce to be transported to workers’ homes or to be used in home-centered businesses.

Health effects and deaths from contaminants brought home from the workplace are preventable using known effective measures, however educational programs are needed to promote their use. Preventive measures are necessary because normal house cleaning and laundry practices are often inadequate for decontaminating workers’ homes and clothing and can increase the hazard to the person performing the tasks and others in the household.

Only two Federal laws have elements that directly address workers’ home contamination. However, other laws provide agencies with certain mechanisms for responding to, or preventing workers’ home contamination. Under existing laws, OSHA, MSHA, DOE, ATSDR, EPA, and CDC, including NIOSH and the National Center for Environmental Health have responded to incidents of workers’ home contamination, made preventive recommendations, and conducted relevant research.
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<tr>
<th>Author (year)</th>
<th>Location</th>
<th>Industry</th>
<th>Study Design</th>
<th>Results</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Hardy [1948]</td>
<td>Massachusetts</td>
<td>Fluorescent bulb manufacture</td>
<td>Case report</td>
<td>Woman with chronic beryllium disease from household contact.</td>
<td>Exposed for 2 years caring for a fatally ill daughter in home (daughter was a beryllium worker who died of beryllium disease).</td>
</tr>
<tr>
<td>Eisenbud et al. [1949]</td>
<td>Ohio</td>
<td>Beryllium plant</td>
<td>Case report</td>
<td>Woman with chronic beryllium disease from household contact.</td>
<td>Woman laundered husband's work clothes for 3 months. Estimated that a single laundering resulted in inhalation of 17 µg of beryllium.</td>
</tr>
<tr>
<td>Chesner [1950]</td>
<td>Ohio</td>
<td>Beryllium plant</td>
<td>Case reports</td>
<td>Chronic beryllium disease in: 26-year-old woman whose neighbor worked at the plant. 8-year-old girl.</td>
<td>Woman used bags in which beryllium had been shipped as dish clothes; 10 year latency; infant child may also be a case.</td>
</tr>
<tr>
<td>DeNardi et al. [1949]</td>
<td>Ohio</td>
<td>Beryllium plant</td>
<td>Case report</td>
<td>26-year-old woman.</td>
<td>Girl's father worked at plant for 1 year before her birth; her uncle had chronic pneumonitis and lived in home for a few months the year before the girl became ill. He had worked at the plant for a few weeks about 7 years earlier. Husband worked in beryllium plant for 8 weeks; she denied cleaning his clothes.</td>
</tr>
<tr>
<td>Newman and Kreiss [1992]</td>
<td>Ohio</td>
<td>Beryllium plant</td>
<td>Case report</td>
<td>56-year-old woman whose husband worked in plant for 26 years before initiation of symptoms of chronic beryllium disease.</td>
<td>Woman had only incidental exposure; plant required change of clothes and shower before going home.</td>
</tr>
<tr>
<td>Chamberlin et al. [1957]</td>
<td>Pennsylvania</td>
<td>Beryllium plant</td>
<td>Case series</td>
<td>5 women ages 24-56, with chronic pulmonary fibrosis all deceased.</td>
<td>Exposed to contaminated work clothes. Beryllium in lungs 0.02-0.20 µg/100g of dried lung.</td>
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<tr>
<td>Lieben and Metzner [1959]</td>
<td>Pennsylvania</td>
<td>Beryllium plant</td>
<td>Case series</td>
<td>19 cases of beryllium disease ages 10-60 yrs, 9 deceased, 21 neighborhood cases, exposures ages 10-80 yrs.</td>
<td>All had contact with contaminated clothing for 2 months to 13 years; beryllium was found in the lungs.</td>
</tr>
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<td>Metzner and Lieben [1961]</td>
<td>Pennsylvania</td>
<td>Beryllium plant</td>
<td>Case series</td>
<td>Added 3 contact cases and 5 neighborhood cases to those reported by Lieben and Metzner [1959].</td>
<td>Two of the cases were a brother and sister who were often present when the mother washed their father's clothes.</td>
</tr>
<tr>
<td>Dattoli et al. [1964]</td>
<td>Pennsylvania</td>
<td>Beryllium plant</td>
<td>Case series</td>
<td>Added 1 contact case and 3 neighborhood cases to those reported by Metzner and Lieben [1961].</td>
<td>Beryllium found in lungs of contact case who handled contaminated clothes for 5 years and also had neighborhood exposure.</td>
</tr>
<tr>
<td>Lieben and Williams [1969]</td>
<td>Pennsylvania</td>
<td>Beryllium plant</td>
<td>Case series</td>
<td>Added 3 cases of beryllium disease to those reported by Dattoli et al. [1964]. Total of 26 cases ages 17-59 among household contacts of workers; 22 were female, 18 deceased.</td>
<td>Exposed to clothing or home environment of beryllium workers before 1959.</td>
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<td>Author (year)</td>
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<td>Hardy [1965] Hardy et al. [1967]</td>
<td>U.S.A.</td>
<td>Various</td>
<td>Beryllium Case Registry (1952-1966)</td>
<td>40 cases from household contact.</td>
<td>All exposed to contaminated work clothes; 13 also exposed by air pollution.</td>
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<td>Author (year)</td>
<td>Location Industry/ Population at Risk</td>
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<td>Anderson [1983]</td>
<td>Patterson, New Jersey</td>
<td>Cohort study. Morbidity and mortality among 2,218 household contacts of amosite workers identified. 679 of 1,545 alive through 1980 were examined. Occupational, residential, smoking, medical history questionnaire administered to the exposed cohort. Vital status follow-up is through 1980. Radiographs were taken 20+ years after first exposure. For radiographic analysis, a frequency matched (age, gender) control group was assembled of 326 unexposed people from the same urban New Jersey community who presented for chest radiograph 1975-1976.</td>
<td>3/663 observed deaths were due to mesothelioma. Lung cancer overall SMR=152 (25 observed/16.4 expected) after 20 years latency SMR=185. Among females, there were 8 respiratory cancers observed vs. 6.4 expected. Excess risk was confined to those with 20+ years latency (8 observed vs. 4.7 expected, SMR=170). Among males with 20+ years latency, there were 12 lung cancer deaths observed vs. 6.1 expected (SMR=197). Increased frequency of asbestos-associated radiographic abnormalities among household contacts. Prevalence of radiographic abnormality associated with secondary exposure was 35% vs. 5% expected based on the comparison population (p&lt;0.001). Prevalence of abnormalities increased with duration since first exposure; 40% prevalence among those with longest latency (p&lt;0.01). Those with 10+ years of household exposure had a prevalence of abnormal radiographs of 53%. For 1971 ILO classification 1/1 and greater, a prevalence of 10.3 observed vs. 0.6% in controls. Prevalence of parenchymal or pleural abnormality 20+ years after first household exposure (1979b): 48% among wives, 21% among daughters, 42% among sons, and 37% among siblings.</td>
<td>Mesothelioma deaths occurred 20+ years after childhood domestic exposure (2 female; 1 male). There were 2 additional mesotheliomas among children of workers that were excluded from analysis. Dust from work clothes, shoes, hair assumed causal. No changing facilities at factory.</td>
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<tr>
<td>Anderson et al. [1976, 1979a, 1979b]</td>
<td>Amsite workers employed 1941-1945 in thermal insulation materials factory</td>
<td>Retrospective cohort mortality study of 1,964 wives of asbestos cement workers; cohort had no history of occupational exposure. Husbands employed 1950-1985; deaths occurred 1965-1988.</td>
<td>Between 1965-1988, there were 4 pleural tumors (1 mesothelioma) observed vs. 0.5 expected; 6 lung cancer vs. 4.0 expected. Expected based on local rates. Among women with domestic exposure, cancer of the pleura was significantly elevated SMR = 792.3 (95% CI 215.9 - 2,028.8).</td>
<td>This plant had no laundering facilities, and work clothes were laundered at home. All 6 cases reported more than 10 years of exposure. There were 2 additional mesotheliomas observed after 1988.</td>
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<td>Joubert et al. [1991]</td>
<td>New Jersey Amosite asbestos workers</td>
<td>Cohort study. Followed household contacts of amosite asbestos workers employed at a single facility 1941-1954. Of 4,044 household contacts, 878 were examined 1973-1976.</td>
<td>Vital status follow-up through January 1990 indicates that 28% died of lung cancer, 23% died from cancer of the gastrointestinal tract, and 9% died of mesothelioma. The authors state that cancer deaths were 2 times expected based on national estimates.</td>
<td>Some figures reported in the paper seem contradictory. This appears to be additional follow-up of the Anderson et al. studies.</td>
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<tr>
<td>Navrátil and Trippa [1972]</td>
<td>Czechoslovakia Chrysotile asbestos product processing</td>
<td>Cohort study. Prevalence of pleural calcification in three asbestos exposed groups compared with prevalence in non-exposed residents of the same area (Group #4).</td>
<td>Each group was evaluated by X-ray for the prevalence of pleural calcification, with or without other signs of asbestosis. Observed statistically significant (p&lt;0.01) increased risk of calcification among each asbestos exposed group compared with Group #4.</td>
<td>In group #2, 5 were also relatives but were counted in group #2 rather than group #3. Blood relations were assumed to have increased exposure due to contact with workers wearing contaminated work clothes.</td>
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<p>| Group #1 | 800 workmen employed for more than 10 years at a factory. | Group #1 | 42/800 (5.3%) observed vs. 2.75 expected |
| Group #2 | 155 persons living in the neighborhood of the factory. | Group #2 | 9/155 (5.8%) observed vs. 0.53 expected |
| Group #3 | 114 persons older than 20 years who were relatives of factory employees. | Group #3 | 4/114 (3.5%) observed vs. 0.39 expected |
| Group #4 | 8,127 persons over the age of 40 who lived in the same district as the factory but not in the same neighborhood as the factory. | Group #4 | 25/8127 (0.34%) |</p>
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<tr>
<td>Kilburn et al. [1985, 1986]</td>
<td>Los Angeles County Shipyard workers</td>
<td>Community-based cohort. Prevalence of radiographic evidence of asbestos among shipyard workers and their household contacts with at least 20 years latency ( n = 1017 ) was compared with that of 2 previously studied comparison groups (Long Beach census tract and Michigan adults). Medical and occupational history obtained by examination and interview.</td>
<td>Prevalence among household contacts without occupational exposure was reported. Among 274 wives of shipyard workers, 11.3% had radiographic evidence of asbestosis (profusion 1/0 or greater), compared with prevalence of 0.6% in the California and 0.0% in the Michigan comparison groups. Prevalence increased with time since first exposure; the prevalence rate among those with longest latency was 32%. Among 140 female children, the prevalence rate was 2.1%; a prevalence of 7.6% was observed among 79 sons of shipyard workers.</td>
<td>Possible selection bias resulting from volunteer study participants. No difference in prevalence observed by smoking status. Most shipyard workers had indirect (bystander) exposure. Families of insulators appear to be at increased risk of asbestosis compared with other shipyard workers. (1% of shipyard workers were insulators; about 25% of asbestosis in workers' families occurred in families of insulators.)</td>
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<td>Newhouse and Thompson [1965]</td>
<td>London, England</td>
<td>Hospital-based</td>
<td>Matched case-control. Cases (n=83) were autopsy series who died of mesothelioma (pleural and peritoneal) 1917-1964; matched on gender and birth date (+/- 5 years) to in-patient controls from same hospital who were hospitalized 1964. Two other comparison groups were used to verify results, but not reported in paper: 1. matched on gender, birth date, and date of admission, and 2. 17 patients from same hospital pathology series who were misdiagnosed as mesothelioma.</td>
<td>9/76 cases (7 female; 2 male) reported domestic exposure compared with 1/76 controls from the inpatient series.</td>
</tr>
<tr>
<td>Vianna and Polan [1978]</td>
<td>New York State</td>
<td>Population-based</td>
<td>Matched case-control. Cases were 52 (30 pleural/20 peritoneal) histologically confirmed female mesothelioma (pleural and peritoneal) deaths (1967-1977); one-to-one matching on gender, race, county of residence, marital status, age and year at death; controls died from causes other than cancer; occupational history by questionnaire, medical and industrial records.</td>
<td>Relative Risk from matched pairs analysis for domestic exposure (included 2 with occupational exposure) reported as 10 (95% CI = 1.4-37.4). Analysis on subset of 46 non-occupationally exposed cases: 8/46 reported domestic exposure vs. 1/46 controls (p=0.02).</td>
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<td>McDonald and McDonald [1980]</td>
<td>Canada and USA</td>
<td>Population-based autopsy series (Canada 1960-1972: USA 1972)</td>
<td>Matched case-control. 557 pleural and peritoneal mesotheliomas with autopsy; matched on hospital, gender, age, and year of death to controls with pulmonary metastases from non-pulmonary primary who were autopsied; occupational, residential, smoking, and non-occupational exposure histories from interview (blind) with relatives for 490 matched pairs. Occupational exposures coded blind and cumulated to 10 years before death of case.</td>
<td>8/557 cases vs. 2/557 controls reported domestic exposure to asbestos dust on work clothes of household contact (p=0.08 for matched pairs analysis).</td>
</tr>
<tr>
<td>Whitwell et al. [1977]</td>
<td>England</td>
<td>Hospital-based</td>
<td>Case-control. Asbestos fiber content in lungs of 100 consecutive pleural mesothelioma autopsies compared with 100 lung cancer cases and 100 lungs of people who died from causes other than industrial lung disease or lung cancer for whom occupational histories were available. Occupational and residential history obtained from patients or relatives.</td>
<td>1 mesothelioma reported in asbestos worker's family. Although not explicitly stated in published report, apparently no cases were observed in either control series in asbestos workers' families.</td>
</tr>
<tr>
<td>Author (year)</td>
<td>Location</td>
<td>Industry/Population at Risk</td>
<td>Study Design</td>
<td>Results</td>
</tr>
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<tr>
<td>McEwen et al. [1971]</td>
<td>Scotland</td>
<td>Population-based</td>
<td>Matched case-control. 83 mesothelioma cases who died 1950-1967 from all pathology departments in Scotland. Two control groups were matched on age and gender to the nearest chronologic pathology report from the same hospital: (1) coronary artery disease deaths, and (2) lung and gastric carcinoma cases were matched to pleural and peritoneal mesothelioma cases, respectively.</td>
<td>Only a few cases and controls had shared households with asbestos workers. No statistically significant differences between these two groups.</td>
</tr>
<tr>
<td>Rubino et al. [1972]</td>
<td>Italy (Piedmont)</td>
<td>Various industries;</td>
<td>Case-control study of 50 confirmed cases of pleural mesothelioma admitted to 2 Turin clinical settings 1960-1970. Controls were 50 patients from the same institution matched on gender and age.</td>
<td>12% of mesothelioma reported in the case series were in workers' family members; 3 cases (2 men and 1 woman) had lived with persons employed in the asbestos industry, compared with none in the control group.</td>
</tr>
<tr>
<td>Ashcroft and Heppleston [1970]</td>
<td>Britain</td>
<td>Shipbuilding</td>
<td>Case-control study of 23 cases of mesothelioma (20 pleura, 3 peritoneum; 19 males, 4 females) that came to autopsy compared with 46 hospital controls matched on sex and age, free from malignant disease.</td>
<td>91% of the cases had a history of exposure to asbestos vs. 41% of 46 matched controls (p&lt;0.001); 1 of the cases resulted from domestic exposure.</td>
</tr>
<tr>
<td>AUTHOR(S) (YEAR) COUNTRY</td>
<td>INDUSTRY</td>
<td>HEALTH Effect</td>
<td># CASES/RELATIONSHIP</td>
<td>AGE(S) AT DEATH OR DIAGNOSIS</td>
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<tr>
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</tr>
<tr>
<td>Rusty [1968] England</td>
<td>Asbestos</td>
<td>Mesothelioma</td>
<td>1 Female</td>
<td>71</td>
</tr>
<tr>
<td>Teysaier and Lesobre [1968]</td>
<td>Asbestos</td>
<td>Asbestosis Pleural plaques</td>
<td>Case report of asbestosis in a man exposed as a teen to his father's work clothes worn home from an asbestos plant.</td>
<td></td>
</tr>
<tr>
<td>Champion [1971] Canada</td>
<td>Asbestos</td>
<td>Mesothelioma</td>
<td>1 Male</td>
<td>31</td>
</tr>
<tr>
<td>Knappmann [1972] West Germany</td>
<td>Asbestos</td>
<td>Mesothelioma</td>
<td>Man lived for several years with sister who was an asbestos worker who came home with dusty clothes and hair.</td>
<td>66</td>
</tr>
<tr>
<td>Lillington et al. [1974] USA</td>
<td>Asbestos</td>
<td>Familial mesothelioma</td>
<td>1 Wife</td>
<td>52</td>
</tr>
<tr>
<td>Li et al. [1978] USA</td>
<td>Asbestos</td>
<td>Familial mesothelioma</td>
<td>1 Mother, and 1 daughter</td>
<td>51,34</td>
</tr>
<tr>
<td>Epler et al. [1980] USA</td>
<td>Asbestos</td>
<td>Pleural changes Mesothelioma</td>
<td>2 Wives of asbestos workers 2 Brothers</td>
<td>60,56, 33 and 27</td>
</tr>
<tr>
<td>Rieberg et al. [1980] USA</td>
<td>Construction</td>
<td>Familial pleural and peritoneal mesothelioma</td>
<td>Father, 2 brothers and 1 sister</td>
<td>61,71, 60, and 52</td>
</tr>
<tr>
<td>Jorgensen [1981] Denmark</td>
<td>Asbestos (insulation work)</td>
<td>Pleural plaques</td>
<td>3 Wives of insulation workers</td>
<td>71,54 and 58.</td>
</tr>
<tr>
<td>AUTHOR(S) (YEAR) COUNTRY</td>
<td>INDUSTRY</td>
<td>HEALTH EFFECT</td>
<td># CASES/RELATIONSHIP</td>
<td>AGE(S) AT DEATH OR DIAGNOSIS</td>
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<tr>
<td>Martensson et al. [1984b] Sweden</td>
<td>Asbestos (foundry)</td>
<td>Familial pleural mesothelioma</td>
<td>Sister and brother</td>
<td>52-58</td>
</tr>
<tr>
<td>Krousel et al. (1986) USA</td>
<td>Asbestos</td>
<td>Familial pleural mesothelioma</td>
<td>Mother, son and daughter of a lumber and shingle company worker.</td>
<td>74, 40, and 35.</td>
</tr>
<tr>
<td>Magee et al. [1986] Italy</td>
<td>Chrysotile ore contaminated with tremolite and actinolite.</td>
<td>Mesothelioma</td>
<td>Single male case.</td>
<td>41</td>
</tr>
<tr>
<td>Huncharek et al. [1989] USA</td>
<td>Asbestos</td>
<td>Pleural mesothelioma</td>
<td>Single case (female)</td>
<td>76</td>
</tr>
<tr>
<td>Li et al. [1989] USA</td>
<td>Asbestos</td>
<td>Familial mesothelioma</td>
<td>2 Female cases. Father worked in asbestos plant.</td>
<td>32, 49</td>
</tr>
<tr>
<td>Otte et al. [1990] Denmark</td>
<td>Amosite asbestos cement</td>
<td>Familial mesothelioma</td>
<td>Family cluster of 3 deceased.</td>
<td>74, 79, 45.</td>
</tr>
<tr>
<td>AUTHOR(S) (YEAR) COUNTRY</td>
<td>INDUSTRY</td>
<td>HEALTH EFFECT</td>
<td># CASES/RELATIONSHIP</td>
<td>AGE(S) AT DEATH OR DIAGNOSIS</td>
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<tr>
<td>Oem et al. [1991] Norway</td>
<td>Asbestos (various)</td>
<td>Pleural mesothelioma</td>
<td>1 Woman</td>
<td>79</td>
</tr>
<tr>
<td>Anonymous [1993b] England</td>
<td>Asbestos</td>
<td>Pleural plaques</td>
<td>3 Daughters</td>
<td>63, 62, 60</td>
</tr>
<tr>
<td>AUTHOR(S)</td>
<td>INDUSTRY</td>
<td>HEALTH EFFECT</td>
<td>#/RELATIONSHIP</td>
<td>AGE AT DEATH EXPOSURE</td>
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<tr>
<td>Lieben and Platawka [1967] USA</td>
<td>Asbestos (insulation, shipbuilding)</td>
<td>Mesothelioma: 1 pleural and 2 peritoneal.</td>
<td>3-year-old girl whose father worked with asbestos insulation. 40-year-old woman whose father and brother were asbestos insulation workers. 67-year-old woman whose two sons were shipyard asbestos insulation workers.</td>
<td>3, 40, 67. No numerical exposure data given.</td>
</tr>
<tr>
<td>Dalquen et al. [1970] Germany</td>
<td>Asbestos</td>
<td>Pleural plaques</td>
<td>22 cases among domestic contacts</td>
<td></td>
</tr>
<tr>
<td>Heller et al. [1970] USA</td>
<td>Asbestos</td>
<td>Pleural mesothelioma</td>
<td>1 woman. Washed pipefitter husband's asbestos-contaminated work clothes.</td>
<td></td>
</tr>
<tr>
<td>Bittersohl and Ose [1971] Germany</td>
<td>Asbestos (insulation and products such as cords, seals, plates, etc.)</td>
<td>Pleural mesothelioma</td>
<td>1 woman whose husband was exposed to asbestos insulation at a chemical plant. She laundered his work clothes.</td>
<td>Exposure data given in terms of East German standard.</td>
</tr>
<tr>
<td>AUTHOR(S)</td>
<td>YEAR</td>
<td>COUNTRY</td>
<td>INDUSTRY</td>
<td>HEALTH EFFECT</td>
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</tr>
<tr>
<td>Vianna et al. [1981]</td>
<td></td>
<td>USA</td>
<td>Six New York counties</td>
<td>Mesothelioma</td>
</tr>
<tr>
<td>Greenberg and Davies [1974]</td>
<td></td>
<td>England/Wales</td>
<td>Asbestos</td>
<td>Mesothelioma</td>
</tr>
<tr>
<td>Milne [1976]</td>
<td></td>
<td>Australia</td>
<td>Asbestos (asbestos/cement)</td>
<td>Mesothelioma</td>
</tr>
<tr>
<td>Edge and Choudhury [1978]</td>
<td></td>
<td>England</td>
<td>Asbestos (shipyard workers)</td>
<td>Pleural mesothelioma</td>
</tr>
</tbody>
</table>
### Table 6. (Continued) Health Effects of Take-Home Asbestos Exposure (Case Series)

<table>
<thead>
<tr>
<th>AUTHOR(S)</th>
<th>YEAR</th>
<th>COUNTRY</th>
<th>INDUSTRY</th>
<th>HEALTH EFFECT</th>
<th>#/RELATIONSHIP</th>
<th>AGE AT DEATH EXPOSURE</th>
<th>COMMENTS/ISSUES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bianchi et al.</td>
<td>1982</td>
<td>Italy</td>
<td>Asbestos (shipyard work)</td>
<td>Pleural mesothelioma</td>
<td>1 woman whose husband was a shipyard worker.</td>
<td></td>
<td>Of 70 cases (64 men; 6 women) seen at Institute of Pathological Anatomy of Trieste 1967-1980 1 was due to probable domestic exposure. Necropsy findings available in 63 cases. Remaining 7 cases were diagnosed at thoracotomy. Of the cases, 43 employed in shipyards, most prior to 1940. Intervals between first exposure and death ranged from 28 to 61 years. Asbestos bodies found in 48 of 61 cases.</td>
</tr>
<tr>
<td>Bianchi et al.</td>
<td>1987</td>
<td>Italy</td>
<td>Asbestos (shipyard, sodium carbonate factory)</td>
<td>Hysaline pleural plaques</td>
<td>59 cases were attributed to domestic exposure (laundring asbestos-contaminated work clothes of family members). 9 cases with occupational and domestic exposure.</td>
<td>Not stated</td>
<td>74 women with hysaline pleural plaques found at necropsy. 2 cases with occupational exposure. Sufficient exposure data could not be obtained on 4 cases. Pleural malignant mesotheliomas was noted in 2 cases with a history of household exposure.</td>
</tr>
<tr>
<td>Bianchi et al.</td>
<td>1990</td>
<td>Italy</td>
<td>Shipyard, sodium carbonate factory, textile, artisans, domestic maids</td>
<td>Hysaline pleural plaques</td>
<td>Pleural plaques at necropsy were found in 55% of 121 women with history of domestic exposure.</td>
<td></td>
<td>1,620 necropsies (1,040 men, 580 women) were performed from Oct 1979 to Dec 1987 in Monfalcone, Italy. 121 women with history of domestic exposure were compared to 37 women with no history of domestic exposure. Pleural plaques were significantly more prevalent in those women with domestic exposure (p&lt;0.001). The prevalence of hysaline plaques was higher in every occupational category for women with domestic exposure than for women without domestic exposure.</td>
</tr>
<tr>
<td>Bianchi et al.</td>
<td>1991</td>
<td>Italy</td>
<td>Shipbuilding, sodium carbonate factory</td>
<td>Asbestos bodies Hysaline pleural plaques</td>
<td>1,765 necropsies (1,127 men; 638 women). In women, cleaning of work clothes polluted with asbestos was the main source of exposure. Domestic exposure resulted in pleural plaques in about half the necropsies on female patients.</td>
<td></td>
<td>Prevalence of pleural plaques and asbestos bodies varied by occupation in men; the highest prevalence was in those who had worked in the sodium carbonate factory. Only 21 of the 638 necropsies on females had a history of occupational exposure.</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>AUTHOR(S) YEAR COUNTRY</th>
<th>INDUSTRY</th>
<th>HEALTH EFFECT</th>
<th>#/RELATIONSHIP</th>
<th>AGE AT DEATH EXPOSURE</th>
<th>COMMENTS/ISSUES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bianchi et al. [1993] Italy</td>
<td>Asbestos (shipyard, sodium carbonate factor)</td>
<td>Malignant mesothelioma</td>
<td>6 women had history of domestic exposure (laundring asbestos-contaminated clothing of family members).</td>
<td></td>
<td>92 malignant mesotheliomas were diagnosed between Oct. 1979 and April 1992 at Monfalcone Hospital. 6.5% of these were associated with domestic exposure. 75 cases had occupational history of exposure to asbestos. One case had a history of probable environmental exposure.</td>
</tr>
<tr>
<td>Lander and Viskum [1985] Denmark</td>
<td>Asbestos work</td>
<td>Pleural plaques, pleural calcification, pulmonary fibrosis, asbestosis</td>
<td>Of 63 women (spouses of workers exposed to asbestos) with indirect (non-occupational) exposure to asbestos, 9 (17%) had radiological changes characteristic of exposure to asbestos. Exposure consisted of laundering asbestos-contaminated work clothes.</td>
<td>Adult women</td>
<td>The researchers attempted to enroll 125 spouses of asbestos-exposed workers. 90 participated in the study. 20 were excluded due to lack of exposure (X-rays were normal). 5 were excluded due to occupational exposure (one had pleural plaques). 2 were excluded for other pulmonary diseases.</td>
</tr>
<tr>
<td>Gibbs et al. [1989] Wales</td>
<td>Asbestos</td>
<td>Pleural mesothelioma</td>
<td>1 male and 12 females with non-occupational exposure (Zielhuis group II, e.g., the wives of asbestos workers) included in the study.</td>
<td>47-72</td>
<td>84 cases diagnosed 1979-1986 chosen because the history of asbestos exposure was absent, indirect, or ill-defined. 3 purposes of study were: 1. correlate lung mineral count with Zielhuis (1977) occupational exposure groupings; 2. determine whether any mesotheliomas were unrelated to asbestos exposure; and 3. compare the role of amphiboles and chrysotile in causation. Conclude that 1. Zielhuis method too complex; 2. mesotheliomas may develop in absence of asbestos exposure; and 3. amphiboles are more important than chrysotile. Electron microscope mineral fiber analysis data provided by exposure group.</td>
</tr>
<tr>
<td>AUTHOR(S) YEAR COUNTRY</td>
<td>INDUSTRY</td>
<td>HEALTH EFFECT</td>
<td>#/RELATIONSHIP</td>
<td>AGE AT DEATH EXPOSURE</td>
<td>COMMENTS/ ISSUES</td>
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</tr>
<tr>
<td>Gibbs et al. [1990] Wales</td>
<td>Asbestos (shipyard work, legging, building, ordinance)</td>
<td>Malignant pleural mesothelioma</td>
<td>10 cases in family members, 9 of whom were spouses of asbestos workers and 1 was daughter of a man who died of asbestosis.</td>
<td>Age range: 47 to 72.</td>
<td>This was a comparison of types of lung fibers and size distribution in a series of non-occupational cases of mesothelioma with a series of known occupational exposure in female gas mask workers. The non-occupational group fiber exposure was variable; 6 showed high crocidolite; 7 showed high amosite; 1 high chrysotile; and 2 showed normal for all fiber (several showed more than 1 high fiber group).</td>
</tr>
<tr>
<td>Konetzke et al. [1990] Germany</td>
<td>Asbestos</td>
<td>Asbestosis Mesotheiloma</td>
<td>48 non-occupational registry cases of mesothelioma and 19 cases of asbestosis (11 male; 56 female). 10 of these lived near an asbestos plant.</td>
<td></td>
<td>Confirmed report that even in the non-occupational area, asbestos represents a non-negligible risk for diseases of the lung. Non-occupational risk factors identified in this study included: laundering (46%); use of asbestos containing materials in the house (21%); and leisure activities (15%).</td>
</tr>
<tr>
<td>Kivliuoto [1965] Finland</td>
<td>Asbestos</td>
<td>Pleural plaques, Pleural adhesions, Pulmonary fibrosis, Mesotheiloma.</td>
<td>4 cases of asbestoses in 4 sisters whose father had been occupationally exposed to mixed dusts.</td>
<td></td>
<td>The father 50 years earlier had been occupationally exposed to mixed dusts and presumably brought them home on his clothes.</td>
</tr>
<tr>
<td>Martensson et al. [1984a]</td>
<td>Asbestos work</td>
<td>Mesotheiloma</td>
<td>Woman who had been exposed to asbestos during childhood via her father's work clothes.</td>
<td></td>
<td>Analysis of 32 cases of malignant mesothelioma. All but one case was occupational.</td>
</tr>
<tr>
<td>Sider et al. [1987] U.S.A.</td>
<td>Insulation work</td>
<td>Radiographic pleural changes (plaques, calcification, thickening)</td>
<td>18 (19.4%) wives of asbestos-exposed insulation workers screened had radiographic abnormalities.</td>
<td></td>
<td>117 wives of asbestos-exposed insulation workers were screened with X-rays and pulmonary function tests. None of the 24 women under age 40 had any X-ray abnormalities. Exposure for all of them was less than 8 years. These 24 were excluded. 18 (19.4%) of the remaining 93 had radiographic abnormalities.</td>
</tr>
<tr>
<td>AUTHOR(S) YEAR COUNTRY</td>
<td>INDUSTRY</td>
<td>HEALTH EFFECT</td>
<td>#/RELATIONSHIP</td>
<td>AGE AT DEATH EXPOSURE</td>
<td>COMMENTS/ ISSUES</td>
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<tr>
<td>Giarelli et al. [1992] Italy</td>
<td>Asbestos (shipyard)</td>
<td>Mesothelioma</td>
<td>5 women with a history of domestic exposure to asbestos (laundring the asbestos-contaminated clothing of family members).</td>
<td>Age at death of all cases 33 to 92 years (median 70 years).</td>
<td>170 pleural mesotheliomas examined at necropsy between 1968-1987 (Trieste University). Occupational histories consistent with asbestos exposure in 150 cases. 5 had no asbestos exposure history but lung sections showed asbestos bodies. 5 women had a history of domestic exposure to asbestos (laundring the asbestos-contaminated clothing of family members).</td>
</tr>
</tbody>
</table>
Table 7. Health Effects of Take-Home Lead Exposure (Cohort Studies)

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Location</th>
<th>Industry</th>
<th>Study Design</th>
<th>Results</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baker et al. [1977] CDC [1976]</td>
<td>Tennessee, USA</td>
<td>Lead smelting</td>
<td>Cohort 20 Exposed children 17 Neighborhood controls</td>
<td>Mean BLL significantly higher in exposed children; some of whom had BLL &gt; 80 µg/dL. Higher dust lead levels in exposed houses (2.687 vs. 404 ppm); children's BLLs correlated with dust levels.</td>
<td>Matched on neighborhood and measured lead content in paint by X-ray fluorescence. 7 children had erythrocyte protoporphyrin &gt; 190 µg/100 mL which necessitated immediate medical attention.</td>
</tr>
<tr>
<td>Elwood et al. [1977]</td>
<td>England</td>
<td>Battery plant</td>
<td>Cohort 192 Exposed children 273 Children from birth registry</td>
<td>Workers' children had significantly higher BLLs than registry children (mean 33 vs. 27 µg/dL).</td>
<td>Used capillary sampling; 3-year-olds had highest BLLs. No health effects reported.</td>
</tr>
<tr>
<td>Kaplan et al. [1977]</td>
<td>Barbados</td>
<td>Pottery</td>
<td>Cohort 12 Potters 19 Family members 24 Controls</td>
<td>Mean BLL of potters' family members (35 µg/dL) was significantly higher than that of controls (17-19 µg/dL).</td>
<td>Homes were adjacent to potteries. An adult female member of potters' family had decreased deep tendon reflexes in ankles bilaterally; BLL of 54 and erythrocyte protoporphyrin of 209 µg/100 mL.</td>
</tr>
<tr>
<td>Watson et al. [1978] CDC [1977a]</td>
<td>Vermont, USA</td>
<td>Battery plant</td>
<td>Cohort 27 Exposed children 32 Neighborhood controls</td>
<td>56% of exposed vs. 12.5% of controls had BLL ≥ 30 µg/dL; higher mean dust lead levels in exposed houses (2,289 ppm) vs. controls (718 ppm).</td>
<td>Used capillary sampling. Significantly elevated erythrocyte protoporphyrin.</td>
</tr>
<tr>
<td>Millar [1978]</td>
<td>England</td>
<td>Lead smelting/refining</td>
<td>Cohort 71 Children of workers 191 Community children (living near plant)</td>
<td>Difference in BLLs between workers' children (21.1 µg/dL) and community children (18.2 µg/dL) was statistically significant for children age ≤ 10.</td>
<td>No deviation from normal health were found by careful study.</td>
</tr>
<tr>
<td>Rice et al. [1978]</td>
<td>USA (city unspecified)</td>
<td>Secondary lead smelter</td>
<td>Cohort 33 Exposed homes 19 Neighborhood homes</td>
<td>7 exposed children had zinc protoporphyrins over 50 µg/dL compared to 1 control child. Significantly higher lead levels were found in wipe and dust samples from exposed homes compared to control homes.</td>
<td>No BLLs were measured. Other health effects not reported.</td>
</tr>
<tr>
<td>Abbratti et al. [1979]</td>
<td>Italy</td>
<td>Ceramics</td>
<td>Cohort 40 Children of ceramic workers 47 Children of ceramic workers who work at home 89 Unexposed children in community</td>
<td>Mean BLLs for the three groups: 25.1, 27.3, 23.0 (p-value for difference between the latter two &lt; 0.001).</td>
<td>Many of the pottery factories were home-operated. No significant difference in hematocrit.</td>
</tr>
<tr>
<td>Author (year)</td>
<td>Location</td>
<td>Industry</td>
<td>Study Design</td>
<td>Results</td>
<td>Comments</td>
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</tr>
<tr>
<td>Landrigan et al. [1980]</td>
<td>Georgia, USA</td>
<td>Stained glass</td>
<td>Cohort 12 Workers 5 Hobbyists 4 Workers’ family members</td>
<td>Mean BLLs were 20.7 μg/dL for workers, 11.6 μg/dL for hobbyists and 11.3 μg/dL for family members of workers. BLL was associated with duration of work and percentage of work involving lead.</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td>Molina-Ballesteros et al. [1980]</td>
<td>Mexico</td>
<td>Pottery</td>
<td>Cohort 198 Workers and their families 187 Controls and their families</td>
<td>Children aged &lt; 9 years of exposed workers had a mean BLL of 81 μg/100g compared to control children of same age who had a mean BLL of 19.5 μg/100g.</td>
<td>No association between clinical symptomology and lead poisoning could be established because of socioeconomic conditions.</td>
</tr>
<tr>
<td>Morton et al. [1982]</td>
<td>Oklahoma, USA</td>
<td>Battery factory</td>
<td>Cohort 34 Exposed children (age &lt; 7) 34 Age-matched neighborhood control children</td>
<td>Significantly different BLLs were found between groups p&lt;0.001, BLL &gt; 30 μg/dL (maximum 72 μg/dL) in 53% of exposed children versus 0% in controls. Statistically significant differences found in children's BLL between good and poor worker hygiene practices.</td>
<td>Used capillary sampling. No health effects reported.</td>
</tr>
<tr>
<td>Milar and Mushak [1982]</td>
<td>North Carolina, USA</td>
<td>Battery factory</td>
<td>Cohort 17 Exposed children (age &lt; 5) 30 Control children (age &lt; 5)</td>
<td>Average BLL of exposed children was 44 μg/dL and of control children, 18 μg/dL.</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td>Ramakrishna et al. [1982]</td>
<td>Sri Lanka</td>
<td>Gold and silver recovery</td>
<td>Cohort 33 Members of exposed families 21 Neighborhood controls</td>
<td>Mean BLL in exposed families was 33 μg/dL versus 12 μg/dL in control families. Very high BLLs were found in two children aged 12 years (42 and 56 μg/dL).</td>
<td>The youngest child tested was 9 years old. No health effects reported.</td>
</tr>
<tr>
<td>Molina-Ballesteros et al. [1983]</td>
<td>Mexico</td>
<td>Pottery manufacturing</td>
<td>Cohort 153 Children (age 5-15) from pottery-making families 80 Control children from local schools</td>
<td>Exposed children had significantly higher mean BLL (39.5 μg/dL) than controls (24.8 μg/dL). Over 40% of exposed children had BLLs over 40 μg/dL compared to none of control children.</td>
<td>No cases of acute lead poisoning were found. Hemoglobin and hematocrits were within the lower limits of normal.</td>
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<td>Author (year)</td>
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<tr>
<td>Katagiri et al. [1983]</td>
<td>Japan</td>
<td>Pottery manufacturing</td>
<td>Cohort 89 3-yr olds from homes where pottery was made 70 3-yr olds from homes where parent works in pottery factory 947 3-yr olds from homes where no one works in pottery 768 3-yr old controls</td>
<td>Children in groups 1 and 2 had significantly higher urinary lead levels (15.8 and 13.6 μg/dL) compared to control children (10.6 μg/dL) and compared to their mothers (10.8 μg/dL).</td>
<td>Urine samples are questionable in evaluating lead exposure. δ-aminolevulinic acid in urine was not different between groups. Coproporphyrin in urine was slightly elevated in home pottery children.</td>
</tr>
<tr>
<td>Richter et al. [1985]</td>
<td>Israel</td>
<td>Battery factory</td>
<td>Cohort 18 Exposed children 729 Control children</td>
<td>Among exposed children &gt; 10 years old, zinc protoporphyrin &gt; 40 μg/dL was 4.1 times higher and among exposed children &lt; 10, zinc protoporphyrin &gt; 40 μg/dL was 2.9 times higher than controls.</td>
<td>No BLLs were measured. Elevated zinc protoporphyrin can also be influenced by iron deficiency. Other health effects not reported.</td>
</tr>
<tr>
<td>Piccinini et al. [1986]</td>
<td>Italy</td>
<td>Ceramic tile</td>
<td>Cohort 22 Children of tile workers exposed to lead 27 Children of tile workers not exposed to lead 24 Control children</td>
<td>Children in group 1 had a mean BLL of 13.5 μg/dL compared with group 2 mean of 12.2 and group 3 mean of 10.7. Hair lead levels for the 3 groups were 17.0, 9.8 and 7.8 respectively.</td>
<td>Used capillary sampling. No sex differences found. No health effects reported.</td>
</tr>
<tr>
<td>Kaye et al. [1987] CDC [1985]</td>
<td>Colorado, USA</td>
<td>Electrical components plant</td>
<td>Cohort 89 Exposed family members 62 Clinic controls</td>
<td>Exposed family members had significantly higher mean BLL (10.2 μg/dL) compared to unexposed (6.2 μg/dL).</td>
<td>No significant differences between groups in hemoglobin levels.</td>
</tr>
<tr>
<td>Abbritti et al. [1988]</td>
<td>Italy</td>
<td>Ceramic pottery factories</td>
<td>Cohort 136 Exposed children 199 Community children</td>
<td>Exposed children had higher mean BLL (10.7 μg/dL) compared to community children (9 μg/dL) (p&lt;0.05).</td>
<td>Many of the pottery factories were home-operated. No differences found by age or sex of child. No health effects reported.</td>
</tr>
<tr>
<td>Wang et al. [1989]</td>
<td>Taiwan</td>
<td>Multiple</td>
<td>Cohort 105 Newborns of lead workers 102 Non-exposed newborns</td>
<td>Mean cord BLL of exposed newborns was significantly higher (8.8 μg/dL) than mean cord BLL of unexposed newborns (6.9 μg/dL). Birth weights and gestational age not effected.</td>
<td>Paternal contribution to cord BLL appears to be through either working at home (n=12 fathers) or bringing lead dust home and exposing mother.</td>
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<td>Matte et al. [1989]</td>
<td>Jamaica</td>
<td>Battery repair</td>
<td>Cohort</td>
<td>Geometric mean BLLs were significantly higher among exposed households compared to controls. 43% of exposed children aged &lt; 12 years had BLL greater than 70 μg/dL.</td>
<td>These were &quot;backyard&quot; battery repair shops. No health effects reported.</td>
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<td>Matte and Burr [1989]</td>
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<td>CDC [1989a]</td>
<td>Jamaica</td>
<td>Battery repair</td>
<td>Cohort</td>
<td>All exposed children aged 0-5 had BLLs ≥ 25 μg/dL.</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td>Gittleman et al. [1994]</td>
<td>Alabama, USA</td>
<td>Battery reclamation</td>
<td>Cohort</td>
<td>Exposed children had higher mean BLLs (mean 22.4 μg/dL, max 42 μg/dL) compared to controls (9.8 μg/dL). 75% of workers' children had BLLs ≥ 10 μg/dL compared with 40% of control children. Adult family members of workers BLLs (mean 8.9 μg/dL, max 21 μg/dL).</td>
<td>No health effects reported.</td>
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<tr>
<td>Gittleman et al. [1991]</td>
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<td>CDC [1992b]</td>
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<td>Martin et al.</td>
<td>England</td>
<td>Lead factory</td>
<td>Community screening</td>
<td>Of 4 children &lt; age 5 with highest BLLs, 3 with levels of 75, 74, and 65 μg/mL were living close to factory and 2 of these had fathers working at factory. Five of 10 surveys in vicinity of other lead works found elevated BLLs in families of workers (no other data available).</td>
<td>No clinical symptoms of lead poisoning. Not clear whether these four were examined.</td>
</tr>
<tr>
<td>Landrigan and Baker</td>
<td>Texas, USA</td>
<td>Ore smelting</td>
<td>Community survey, 3 Households in survey include smelter workers</td>
<td>No children in worker households had BLL ≥40 μg/dL.</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td>Ewers et al.</td>
<td>Germany</td>
<td>Lead smelting</td>
<td>Community survey, 302 Exposed children, 86 Children in control area</td>
<td>Children of lead workers had higher BLLs than other children (geometric mean = 19.7 vs. 14.2 μg/dL; p &lt; .05). Higher tooth lead levels were associated with father's occupational exposure to lead.</td>
<td>Capillary sampling; blood samples were collected from only a sample of children (n=83). No health effects reported.</td>
</tr>
<tr>
<td>Carvalho et al.</td>
<td>Brazil</td>
<td>Lead smelting</td>
<td>Community survey, 104 Children (age 1-9) of lead workers, 357 Children (age 1-9) of non-lead workers</td>
<td>Exposed children had a significantly higher mean BLL (67.3 μg/dL) than unexposed children (56.6 μg/dL). Variation in hemoglobin levels was not associated with BLLs.</td>
<td>Results originally reported in an unpublished thesis by Carvalho (1982).</td>
</tr>
<tr>
<td>Chenard et al.</td>
<td>Canada</td>
<td>Copper smelting</td>
<td>Community survey, 128 Children, Group 1 (35) exposed through residence and father's work, Group 2 (63) exposed through residence only, Group 3 (30) exposed through father's work only, 189 control children from nearby community</td>
<td>All exposed children had significantly higher BLLs than control children. BLL ratios of exposed groups 1, 2, and 3 to non-exposed were 1.83, 1.79, and 1.23 respectively. All BLLs in workers children &lt; 30 μg/dL.</td>
<td>Additional sources of lead exposure such as hobbies and home assessment not measured. Free erythrocyte protoporphyrin is not significantly different between exposed and control.</td>
</tr>
<tr>
<td>Brockhausen et al.</td>
<td>Germany</td>
<td>Lead smelter</td>
<td>Community survey, 9 Children of lead workers (age 4-5), 195 Control children (age 4-5)</td>
<td>Children of lead workers had significantly higher mean BLL (18.4 μg/dL) than controls (10.4 μg/dL).</td>
<td>No health effects reported.</td>
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</table>
| Silvany-Neto et al. [1989] | Brazil | Lead smelting | Community surveys  
1980 Survey  
131 Children of lead workers  
457 Community children  
1985 Survey  
108 Children of lead workers  
142 Community children | Children of lead workers had a significantly higher mean ZPP level than controls both in the 1980 survey (35.4 vs. 24.9 μg/dL) and in the 1985 survey (26.3 vs. 22.8 μg/dL). | Other health effects in workers children not presented. |
| Maravelias et al. [1989] | Greece | Lead smelting | Community survey  
514 Children living in smelting town | The mean BLL for the children of unskilled workers (many of whom worked at the smelter) was 23.3 μg/dL. This was significantly higher than the mean BLL of children of other workers. | No health effects reported. |
| Hoffstetter et al. [1990] | Germany | Lead and other metal smelting | Community screening  
229 Children ages 6-7 | Mean BLL 6.3 μg/dL (range 2.6-15.5 μg/dL). Factors significantly associated with higher BLL were: living in urban area, second-hand smoke, living in a family of foreigners or with a lead worker. | BLLs significantly lower in 1989 than in previous test years (back to 1974). No health effects reported. |
| ATSDR [1991a] | Pennsylvania, USA | Lead plant | Community survey  
736 Study participants | Children (age 0-5) whose parents had a job with "definite" lead exposure had a mean BLL of 12.7 μg/dL compared with children whose parents were unexposed (9.0 μg/dL). | Poor response rate (27%); numbers very small; no results statistically significant. Erythrocyte protoporphyrin levels for workers' family not reported separately. |
| Miesen [1991] | Germany | Metallurgical plant | Community screening  
491 Exposed (19 children < age 6) | Of schoolchildren living with lead-exposed family members, 16.7% had BLLs over 25 μg/dL. | No effects on red blood cells, hemoglobin, hematocrit and porphyrin. |
| Lyngbye et al. [1991] | Denmark | Multiple lead industries | Community survey  
101 First grade children with high dentine lead concentrations (above 18.7 μg/g).  
99 Control children with low dental lead (below 5 μg/g). | A positive association (4-fold relative risk) was found between dental lead and parental employment as a shipyard worker, welder, auto mechanic or car painter. | Only half of eligible children contributed a tooth for analysis. No health effects reported. |
<table>
<thead>
<tr>
<th>Author (year)</th>
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<tbody>
<tr>
<td>Schuhmacher et al. [1991]</td>
<td>Spain</td>
<td>Multiple lead industries</td>
<td>Community survey 476 Exposed children</td>
<td>Mean hair lead for children whose fathers worked in lead-related occupations was 12.7 μg/g compared to 8.4 μg/g among children of workers not in lead occupations.</td>
<td>No data on blood lead levels. No health effects reported.</td>
</tr>
<tr>
<td>Cook et al. [1993]</td>
<td>Colorado, USA</td>
<td>Smelting and mining</td>
<td>Community screening 150 Children &lt; age 6</td>
<td>Mean BLL 10.1 μg/dL (range 0.3-30.1 μg/dL). Parental occupation as a miner was an independent predictor of BLL.</td>
<td>No health effects reported.</td>
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<td>Anonymous [1952]</td>
<td>Philippines</td>
<td>Storage battery factories</td>
<td>Case reports</td>
<td>Case 1: Symptomatic child was misdiagnosed as a case of poliomyelitis and later died.</td>
<td>Workers made storage batteries in or near where the families lived. No BLLs reported.</td>
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<td>Case 2: 1 Lead-exposed worker, 1 child age 2.5</td>
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<td>Case 3: 1 Lead-exposed worker, 1 child age 2</td>
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<tr>
<td>Joshua et al. [1971]</td>
<td>India</td>
<td>Gold and silver recovery</td>
<td>Case report</td>
<td>BLL levels ranged from 52 to 72 μg/dL in children and 37 to 61 μg/dL in adults.</td>
<td>House and work areas were adjacent. Severe lead poisoning including convulsions and death.</td>
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<td>1 Family (9 adults, 9 children); 3 generations</td>
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<tr>
<td>Winegar et al. [1977]</td>
<td>Minnesota,</td>
<td>Lead smelting</td>
<td>Case series</td>
<td>Median BLL of workers was 72.5 μg/dL (range 21-112 μg/dL) and median BLL of family members was 17 μg/dL (range 8-44 μg/dL). 5 children under age 10 had BLLs ≥ 30 μg/dL. Free erythrocyte protoporphyrin of family members ranged from 10-94 μg/100 mL.</td>
<td>Few family members had symptoms of lead poisonings. Headache and fatigue 20% each. 4 family members with the highest BLLs were asymptomatic.</td>
</tr>
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<td></td>
<td>USA</td>
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<td>38 Workers (87 family members)</td>
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<tr>
<td>Dolcourt et al. [1978] CDC [1977b]</td>
<td>North Carolina, USA</td>
<td>Battery factory</td>
<td>Case series</td>
<td>40 (69%) of children had BLLs ≥ 30 μg/dL. Leveis highest in children age 3 and statistically significant decline with age. Maximum 90 μg/dL.</td>
<td>Used capillary sampling. All children were asymptomatic and all had normal findings on physical and neurological exams. No anemia. 6 children showed metaphyseal lead lines. 6 children with BLL 44-90 required chelation on at least one occasion.</td>
</tr>
<tr>
<td>Huong [1980]</td>
<td>Taiwan</td>
<td>Battery processing, stabilizer manufacturing</td>
<td>Case series</td>
<td>4 children had BLLs of at least 80 μg/dL (2 had lead encephalopathy, 2 had severe abdominal symptoms); 1 child had a BLL of 50-79 μg/dL (no symptoms). 2 adults had BLLs of at least 80 μg/dL (both were symptomatic), 4 had BLLs of 50-79 μg/dL (1 symptomatic), and 2 had BLLs of 30-49 μg/dL.</td>
<td>Male heads of household worked inside the residence; living areas in both homes were contaminated with lead dust.</td>
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<td>Richter et al. [1980]</td>
<td>Israel</td>
<td>Polyvinyl chloride (PVC)</td>
<td>Case series</td>
<td>Workers' mean BLL was 27.6 μg/dL. Mean BLL was elevated among children (12.3 μg/dL) but not among spouses (8 μg/dL). Mean BLL in 4 children whose fathers showered and changed before leaving work was significantly lower at 10.3 μg/dL (9.2-12.0 μg/dL) than in children whose fathers did not (14.7 range 10.0-20.0 μg/dL). Child with BLL of 20 μg/dL had hemoglobin of 11.2 g/dL. One wife had BLL of 6 μg/dL, free erythrocyte protoporphyrin in red blood cells of 124 μg/dL and hemoglobin 9.4 g/dL.</td>
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<tr>
<td>Dolcourt et al. [1981]</td>
<td>North Carolina, USA</td>
<td>Battery factory</td>
<td>Case report (Family 1)</td>
<td>All children had BLLs over 30 μg/dL. The highest observed levels were in a 3-year-old male (256 μg/dL) and a 3-year-old female (220 μg/dL). Erythrocyte protoporphyrin of 400 hemoglobin 6 and 9.9. Erythrocyte protoporphyrin was &gt; 100 in 10 children. 20-year-old women had BLL of 52 μg/dL.</td>
<td>Discarded battery casings were burned as fuel in home. Used capillary sampling. Basophilic stripping of red blood cells in 7 family members 15 months-9 years old. Metaphyseal encephalopathy and lead lines in 15-month-old. Erythrocyte protoporphyrin 313 and 404, hemoglobin 9.6 and 10.4. Worker was operating illicit battery recycling in home. Used capillary sampling. 16-month-old had hematocrit of 38.</td>
</tr>
<tr>
<td>Kawai et al. [1983]</td>
<td>Japan</td>
<td>Cutlery tempering and type</td>
<td>Case series</td>
<td>Children &lt; age 12 had higher mean BLL levels than family members not doing lead work (21.8 vs. 13.7 μg/dL for cutlery-tempering and 27.6 vs. 11.7 μg/dL for type-printing households).</td>
<td>2 children had excessive 8-amino-levulinic acid in urine; 2 children had excessive coproporphyrin in urine.</td>
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<td>Pitts [1986]</td>
<td>Virginia, USA</td>
<td>Radiator repair</td>
<td>Case report</td>
<td>Radiator worker with BLL of 78 µg/dL had children under age 7 with BLLs of 79, 48, and 27 µg/dL. Lead dust found in worker's van, and in house where dirty clothes stored. Wife had BLL of 12 µg/dL.</td>
<td>2-year-old child with BLL of 79 and erythrocyte protoporphyrin of 100 was asymptomatic but underwent chelation therapy.</td>
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<td>Garrettson [1988]</td>
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<td>Novotny et al. [1987]</td>
<td>Colorado, USA</td>
<td>Firing range</td>
<td>Case series</td>
<td>BLLs levels in workers ranged from 41 to 77 µg/dL. Spouse BLLs ranged from 6 to 11 µg/dL.</td>
<td>No health effects reported in wives.</td>
</tr>
<tr>
<td>CDC [1989b]</td>
<td>Colorado, USA</td>
<td>Plaque production</td>
<td>Case report</td>
<td>Children's BLLs ranged from 13 to 37 µg/dL; wife's BLL 15 µg/dL; Children's free erythrocyte protoporphyrin 92-196 µg/dL.</td>
<td>4-year-old daughter's X-ray showed dense metaphyseal density in long bones.</td>
</tr>
<tr>
<td>Pichette et al. [1989]</td>
<td>Texas, USA</td>
<td>Battery manufacturing and recycling</td>
<td>Case series</td>
<td>12% of children had BLLs of 25-49 µg/dL. Mean BLLs for children by age were 19 µg/dL for 0-3; 13 µg/dL for 4-6; and 10 µg/dL for children age 7 and over. Children of battery recycling workers had significantly higher BLLs than children of other battery workers (p = .001). Mean BLL level of spouses who laundered workers' clothes was 13 µg/dL compared to 8.4 µg/dL when clothes were laundered by the company.</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td>Lussenhop et al. [1989]</td>
<td>Minnesota, USA</td>
<td>Radiator repair</td>
<td>Case series</td>
<td>All but 1 child had BLLs below 15 µg/dL. Mean BLL was 9.3 µg/dL.</td>
<td>No health effects in children reported.</td>
</tr>
<tr>
<td>Molovich [1991]</td>
<td>Indiana, USA</td>
<td>Welding</td>
<td>Case report</td>
<td>Child was reported to have consecutive lead levels of 0.3 and 0.47 (units and media not reported) and was asymptomatic.</td>
<td>Family car was contaminated with lead. Child had seizures; neurological measurements normal.</td>
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<td>Flachbein et al. [1991, 1992]</td>
<td>USA</td>
<td>Pottery</td>
<td>Case report 1 Worker 2 Exposed family members</td>
<td>Worker and her daughter had BLLs of 48 and 54 µg/100ml, respectively. Spouse's BLL was 20 µg/100ml.</td>
<td>Physical exams, complete blood counts and routine biochemistry screens were within normal limits. Erythrocyte protoporphyrin levels in artist 225 µg/100 mL of red blood cells.</td>
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<tr>
<td>CDC [1992a]</td>
<td>Utah, USA</td>
<td>Construction</td>
<td>Case report 2 Workers (number of family members not reported)</td>
<td>BLLs in 1 family all &lt; 4 µg/dL. Other family had a 7 mo old with BLL of 17 µg/dL. Home inspection revealed no other sources of lead exposure.</td>
<td>Daughter 162 µg/100 mL of red blood cells. 8-aminolevulinic acid in urine abnormal in all family members. No health effects reported.</td>
</tr>
<tr>
<td>State of Alabama [1992]</td>
<td>Alabama, USA</td>
<td>Pottery manufacturing</td>
<td>Case report 2 Workers (parents) 2 Children</td>
<td>Children (age 2 and 14 mos) had elevated BLLs (no other data reported)</td>
<td>Pottery shop adjacent to home. No health effects reported.</td>
</tr>
<tr>
<td>Anonymous [1992]</td>
<td>Virginia, USA</td>
<td>Not available</td>
<td>Case report 1 Child of 2 workers</td>
<td>A 1-year-old child had a BLL of 56 µg/dL. Mother (BLL=67 µg/dL) and father (BLL=21 µg/dL) both worked in a lead industry.</td>
<td>Child received erythrocyte protoporphyrin 73 µg/dL chelation therapy. No other health effects reported.</td>
</tr>
<tr>
<td>Nunez et al. [1993]</td>
<td>New York, USA</td>
<td>Radiator repair</td>
<td>Case series 7 Children of workers</td>
<td>Mean blood lead level 10 µg/dL (range 4-21 µg/dL); 3 children had levels ≥ 10 µg/dL. 79% of workers reported usually changing their clothes and shoes before leaving work.</td>
<td>Over 50% of radiator shops declined to participate. No health effects reported.</td>
</tr>
<tr>
<td>de Silva [1993]</td>
<td>Maryland, USA</td>
<td>Construction</td>
<td>Case report Adult blood lead registry 2 Children</td>
<td>Construction worker with elevated BLL (86 µg/dL) had a child with BLL of 26 µg/dL. Second report was of a construction worker with BLL of 35 µg/dL who had a child with BLL of 17 µg/dL.</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td>Author (year)</td>
<td>Location</td>
<td>Industry</td>
<td>Study Design</td>
<td>Results</td>
<td>Comments</td>
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<tr>
<td>Amato [1994]</td>
<td>Virginia, USA</td>
<td>Radiator repair</td>
<td>Case reports</td>
<td>Children had elevated BLLs (no other data reported)</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Propane tank manufacturing</td>
<td>1 Worker</td>
<td>Family member had elevated BLL (no other data reported)</td>
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<td></td>
<td></td>
<td>Battery manufacturing</td>
<td>1 Worker, 1 Family member</td>
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<td></td>
<td>Number of workers not given</td>
<td>Children had &quot;mildly elevated&quot; BLLs (no other data reported)</td>
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<td></td>
<td></td>
<td></td>
<td>2 Children</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Barnett [1994]</td>
<td>Oregon, USA</td>
<td>Bronze foundry</td>
<td>Case report</td>
<td>2 children under 2 years of age had BLLs of 14 and 23 µg/dL.</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2 Children of exposed workers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Czachur et al. [1995]</td>
<td>New Jersey, USA</td>
<td>Construction; Battery manufacturing; General manufacturing</td>
<td>Case series</td>
<td>8 children (29%) had BLLs 10-19 µg/dL; highest BLL was 26 µg/dL.</td>
<td>Study was a follow-back of workers with BLLs over 25 µg/dL from adult blood lead registry; 46% response rate. No health effects reported.</td>
</tr>
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<td></td>
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<td></td>
<td>15 Workers, 28 Children</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jung [1994]</td>
<td>Connecticut USA</td>
<td>Painting</td>
<td>Case report</td>
<td>Children's BLLs were 16 and 19 µg/dL. Worker's BLL was 29.9 µg/dL.</td>
<td>No health effects reported.</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td>1 Worker</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>2 Children</td>
<td></td>
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</tr>
<tr>
<td>Natarsjan [1994]</td>
<td>USA</td>
<td>Radiator repair</td>
<td>Case report</td>
<td>Child was found to have a BLL of 24 µg/dL. Father had BLLs of 52 and 64 µg/dL.</td>
<td>Worker changed clothes before going home but did not shower. No health effects reported.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1 Worker</td>
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<table>
<thead>
<tr>
<th>Author (year)</th>
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<th>Study Design</th>
<th>Results</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osorio [1994]</td>
<td>California, USA</td>
<td>Lead recycling/bullet manufacturing</td>
<td>Case reports 1 Worker (2 children) 1-year with BLL of 36 μg/dL.</td>
<td>2-year-old with BLL of 44 μg/dL and 1-year with BLL of 36 μg/dL.</td>
<td>Environmental sources ruled out.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Radiator repair</td>
<td>1 Worker (2 children)</td>
<td>Children age 4 and 1.5 had BLLs in 20's.</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Cable cutting</td>
<td>2 Workers (3 children)</td>
<td>BLLs of children ages 3 and 5 years, and 9 mos were 28, 27, and 21 μg/dL respectively.</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>Cable salvage</td>
<td>1 Worker (1 child)</td>
<td>Child (age 10 mos) had a BLL of 26 μg/dL.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Battery repair</td>
<td>1 Worker (1 child)</td>
<td>Child (age 6) had BLL of 36 μg/dL. Father had BLL of 121 μg/dL.</td>
<td></td>
</tr>
<tr>
<td>Pollock [1994]</td>
<td>Mississippi, USA</td>
<td>Trucking</td>
<td>Case report 1 Worker 2 Children</td>
<td>Children (age 1 and 3) had BLLs of 24 and 28 μg/dL, respectively.</td>
<td>No environmental sources of lead identified. No health effects reported.</td>
</tr>
<tr>
<td>Wiehrdt [1994]</td>
<td>Illinois, USA</td>
<td>Battery plant</td>
<td>Case report 2 Children of 1 worker</td>
<td>&quot;Gross contamination&quot; of home.</td>
<td>2 children were hospitalized and underwent chelation therapy. 1 child underwent chelation therapy.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Not given</td>
<td>Case report At least 1 child of 5 related workers</td>
<td>At least 1 child had a BLL of 50 μg/dL.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Indiana, USA</td>
<td>Metals</td>
<td>Case report Unknown number of children</td>
<td>Children of 3 workers had elevated BLLs (levels not provided)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ohio, USA</td>
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<tr>
<td>O'Tuama et al. [1979]</td>
<td>North Carolina, USA</td>
<td>Burning lead battery terminals</td>
<td>Case report 1 Worker 3 Children Children of coworkers</td>
<td>20-month-old son had high blood lead. 2 siblings plus children of 5 coworkers showed evidence of increased lead absorption.</td>
<td>No health effects reported.</td>
</tr>
<tr>
<td>AUTHOR (YEAR) LOCATION</td>
<td>CONTAMINANT</td>
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<tr>
<td>Christensen [1994]</td>
<td>Caustic Products</td>
<td>Agriculture</td>
<td></td>
<td>Cases were reported of milk pail cleaner poisonings.</td>
<td>Reports were of hospitalizations. Decrease attributed to media campaign generating awareness.</td>
</tr>
<tr>
<td>Denmark</td>
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<tr>
<td>Neidich [1993]</td>
<td>Caustic Farm Products</td>
<td>Agriculture</td>
<td></td>
<td>14 children identified as receiving emergency medical care.</td>
<td>8/14 cases incurred second degree esophageal burns. 6/7 CIP product ingestions occurred from other than the original containers.</td>
</tr>
<tr>
<td>South Dakota</td>
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<tr>
<td>Young [1994] Wisconsin</td>
<td>Caustic CIP products</td>
<td>Agriculture</td>
<td></td>
<td>9 cases of caustic exposure requiring treatment among farm children were identified in Wisconsin for alkali injuries during February 1990 through October 1992.</td>
<td>4 cases were liquid CIP ingestions (all admitted to hospital), 4 cases were eye injuries by caustic cleaning agents, and 1 was a skin burn from liquid CIP.</td>
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<tr>
<td>Edmonson [1987]</td>
<td>Caustic Alkali Ingestions (Clean-in-Place or CIP products)</td>
<td>Agriculture</td>
<td></td>
<td>10 children in Wisconsin were identified in which CIP poisoning occurred over a period of 10 years and presented to 4 WI hospitals.</td>
<td>All 10 cases for farm children involved liquid dairy pipe line cleaner (sodium hydroxide or potassium hydroxide).</td>
</tr>
<tr>
<td>Wisconsin</td>
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<tr>
<td>Pelegrin [1995]</td>
<td>Caustic Alkali Ingestions</td>
<td>Agriculture</td>
<td></td>
<td>4 children were admitted to Wisconsin hospital from March 1993 to Jan 1995.</td>
<td>All 4 suffered esophageal burns (2 severe) and 1 incurred gastric burns, too.</td>
</tr>
<tr>
<td>Wisconsin</td>
<td></td>
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<tr>
<td>Geisinger Medical Center [1991]</td>
<td>Caustic Alkali Ingestions (Clean-in-Place or CIP products)</td>
<td>Agriculture</td>
<td></td>
<td>4 children were identified that had been admitted to a mid-state hospital in PA. during a year period in 1990-91.</td>
<td>1 fatality, a 17-month-old boy, was attributed to CIP products and a 2½-year-old sustained esophageal stricture and perforation.</td>
</tr>
<tr>
<td>Pennsylvania</td>
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</tr>
<tr>
<td>Leach and Leach [1992]</td>
<td>CIP products and pipe line cleaner</td>
<td>Agriculture</td>
<td></td>
<td>19-month-old boy swallowed about 1 teaspoon of heavy duty CIP and pipe line cleaner.</td>
<td>He suffered esophageal burns resulting in scar tissue build-up in the esophagus.</td>
</tr>
<tr>
<td>Maryland</td>
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<tr>
<td>Wisconsin</td>
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</table>
| McGee et al. [1952] USA | Chemical Processing Plant  
Toxaphene Farming | Toxaphene poisoning (convulsions or death) | 1 Son          | 2-year-old son died after playing in yard where storage barn had been built from strips of metal taken from drums that had contained toxaphene. The metal was taken home from a processing plant. 17-month-old son died after drinking from tin cup containing toxaphene while his father was mixing a spray for tobacco. 2-year-old son recovered from convulsions after drinking toxaphene while his mother was working in a cotton field. |
Parathion | Acetylcholinesterase inhibition (nausea to death) | 1 Daughter, 1 Son, 1 daughter | 9-month-old daughter died after playing with a can containing parathion which her father had discarded in the yard. 2½-year-old son and his 5-year-old sister played with sacks containing powdered parathion. Boy became ill, was hospitalized, and recovered. Girl had no symptoms. 23-month-old son and his 3-year-old brothers played with a can of parathion that they found in their basement. Only the younger boy had an acetylcholinesterase test indicating serious poisoning. The younger boy was hospitalized and recovered. The older boy showed no symptoms. |
Parathion | Acetylcholinesterase inhibition (Coma and convulsions; Recovered) | 1 Son          | 4-year-old son was poisoned after playing with a bag of parathion in the barn on his family's farm. |
| MacMillian [1964] Canada | Farming  
Parathion | Acetylcholinesterase inhibition (Respiratory distress, semi-coma; Recovered) | 1 Child        | 2-year-old boy was poisoned after he smeared the remaining contents of "an empty jar" of parathion that he found in the barn over his face and lips. |
| Osorio [1994] California | Farming  
Diazinon  
Chlorpyrifos  
Propoxur | Risk of acetylcholinesterase inhibition by diazinon | 3 Children < 5 years of age | Diazinon, chlorpyrifos, and propoxur were found at elevated levels in homes of farmworkers. Of the farm workers' children, 3 had diazinon at 52-220 ng and 2 of these had chlorpyrifos at 20-100 ng on their hands. No pesticides were found on hands of children of non-farm workers. |
| West [1959] | Crop spraying by airplane. Demeton | Acetylcholinesterase inhibition | 1½-year-old daughter | Father came home after spraying a crop and cleaned his boots with paper towels. He threw the towels in the waste basket and placed his boots in the bathroom. His daughter either contacted the boots or the paper towels. |
| Bittman and Wolfson [1967] Florida | Farming  
Parathion | Acetylcholinesterase inhibition; Death | 5 Children, 6 Children, 7 Children | Deaths of 30 children between 1959 and 1964 were reported due to parathion exposure mainly because of adult misuse, or improper storage or disposal practices included in the report were: Children ages 1-5 years ingested parathion stored in improper containers, such as soft drink bottles. Children aged 1-9 years inhaled or had skin contact with parathion powder, 3 of these children were siblings who died after playing on a swing that they made from a burlap sack heavily contaminated with parathion. |
<table>
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<tr>
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<tbody>
<tr>
<td>Davies and Enos [1980]</td>
<td>Farming Chlorpyrifos</td>
<td>Pesticide poisoning. Symptoms not specified.</td>
<td>1 Child</td>
<td>3-year-old boy ingested chlorpyrifos, shown by the excretion of alky phosphate and phenolic metabolites. The authors note that agricultural workers' wives and children may be heavily exposed. These exposures occur in the field and also from materials brought back to the home.</td>
</tr>
<tr>
<td>Griffin and O'Malley [1992] California</td>
<td>Farming Aldicarb</td>
<td>Acetylcholinesterase inhibition (lethargia, respiratory distress). Recovered.</td>
<td>1 Daughter</td>
<td>3-year-old girl was hospitalized with symptoms of poisoning typical of those resulting from exposure to a carbamate anticholinesterase insecticide. The girl, who recovered, lived in a mobile home on a dairy farm where her father worked. It was determined that a tractor parked near the house contained a box of Aldicarb, and the soil 15 feet from the house showed 1.84 percent Aldicarb.</td>
</tr>
<tr>
<td>Barnett [1994] Oregon</td>
<td>Wood treatment Chloropicrin</td>
<td>Eye irritation, nausea, vomiting, coughing</td>
<td>Neighbors (2 adults, 3 children)</td>
<td>Employee of wood treating company brought home his company vehicle which contained 6 containers of chloropicrin. The containers fell and split open, spilling 1 gallon on the driveway and it affected the neighbors.</td>
</tr>
<tr>
<td>Anderson et al. [1965] California</td>
<td>Salvage Cottage industry Parathion</td>
<td>Acetylcholinesterase inhibition: mild symptoms (nausea, vomiting) to respiratory distress, coma. All recovered.</td>
<td>2 Sons, 1 neighbor child</td>
<td>Father operated a salvage business at home. Flannelette sheets purchased from an insurance adjustor were contaminated with parathion. The children were exposed to parathion when they slept on the sheets which were used in the home.</td>
</tr>
<tr>
<td>Cannon et al. [1978] Taylor et al. [1978] Kelly [1977] Hopewell, Virginia</td>
<td>Chemical Manufacture Kepone</td>
<td>Kepone poisoning (subjective nervousness, objective tremor)</td>
<td>Wives of 2 workers had objective tremor</td>
<td>19% of the 214 community residents had detectable levels of kepone in their blood (0.005-0.0325 ppm). 94% of family members had detectable levels of kepone in their blood (ranging from 0.003-0.39 ppm). Wives of 2 workers had demonstrable tremor. Both gave a history of having washed their husband's work clothing.</td>
</tr>
<tr>
<td>AUTHOR</td>
<td>YEAR</td>
<td>LOCATION</td>
<td>INDUSTRY</td>
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</table>
| Fulton and Matthews | 1936 | Pennsylvania | Manufacture of insulated wire and electrical cable | Acne-like dermatitis (Chloracne) | Wife  
1 Daughter  
1 Son | Workers exposed to hexachloronaphthalene and chlorodiphenyl used in coating wire and electrical cable developed chloracne. The wife, 11-month-old daughter and 21-year-old son of one of these workers also developed chloracne. The father wore dirty work clothes home and played with his son without changing into clean clothes. It was recommended that adequate protective clothing, lockers, and other sanitary facilities should be provided to the workers. |
| Good and Pensky | 1943 | New York | Marine electrical work | Acnesform dermatitis, lassitude, occasional impotence, weight loss, taste disturbances | Wives | 52 electricians exposed to Haloxaz in shipbuilding developed chloracne, as did a few of their wives. After this outbreak, preventive measures were initiated which stressed the importance of cleanliness, frequent showering and changing clothes. Work uniforms were provided. |
| Jensen et al. | 1972 | Derbyshire, Britain | 2,4,5-trichlorophenol manufacture | Chloracne | 1 Son  
1 Wife | Workers at a factory producing 2,4,5-trichlorophenol developed chloracne following an explosion. Contaminants at the plant after the explosion included 2,3,6,7-tetrachlorinated dibenzodioxin. Later 2 pipefitters working on a tank that had been steam cleaned developed chloracne. The son of 1 of these who played with his father while he was wearing his dirty work clothes, and the wife of the other developed chloracne. As a result of this outbreak, the plant initiated a program for laundering work clothes and encouraged the workers to shower regularly, wear clean undergarments, and to change into clean clothing before leaving work. |
<p>| Fischbein and Wolff | 1987 | New York | Transformer maintenance | Elevated serum or adipose polychlorinated biphenyl (PCB) levels | 2 Wives | 2 railway maintenance workers who repaired transformers containing PCBs developed chloracne. Their serum PCB levels (77 ng/mL, 101 ng/mL) had a PCB pattern resembling Aroclor 1254. Wives of the workers did not have significantly elevated levels of PCBs but the PCB pattern also resembled Aroclor 1254. Both wives reported laundering their husbands work clothes. Prudent industrial hygiene measures were recommended to prevent the transmission of chemical from the workplace to the home. |
| Baker et al. | 1980 | Bloomington, Indiana | Municipal Sewage Treatment | Elevated serum polychlorinated biphenyl (PCB) levels | 19 Family members | After PCB was released into the municipal sewage treatment plant by an electrical manufacturing firm, PCB levels in the serum of workers, their family members, community residents and people who applied sludge from the plant on their yards were determined. The mean PCB values were: 17.4 ppb in 89 people who had applied sludge to their yards; 75.1 ppb in 18 sewage treatment workers; 33.6 ppb in 19 family members of the workers; 24.4 ppb in 22 community residents without unusual exposure. No chloracne or systemic poisoning was reported. It was suggested that family members may have contacted PCBs on the shoes, clothing, skin or hair of the workers. |
| ATSDR | 1989 | Kalamazoo, Michigan | Specialty plastics manufacture | Not addressed | Potential exposure of an unspecified number of family members to 4,4'-methylene-bis(2-chloroaniline) (MOCA). | A study was conducted in 1989 to determine the presence of MOCA in homes of employees of the Roto-Flash Company. Vacuum cleaner dust and dryer lint contained a maximum level of 2.6 and 0.65 ppm MOCA, respectively. Maximum MOCA concentrations in urine of 12.1 ppb in a family member and 746 ppb in an employee were found. |</p>
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<tbody>
<tr>
<td>ATSDR [1989a, 1990b] Hesse [1991] Adrian, Michigan</td>
<td>Chemical manufacture</td>
<td>Exposure to 4,4'-methylene bis(2-chloroaniline) (MOCA)</td>
<td>Spouses and children</td>
<td>MOCA was produced by the Anderson Development Company. The Michigan Department of Public Health conducted urine analyses on the workers' spouses and their children. MOCA was found at concentrations up to 15 ppb.</td>
</tr>
<tr>
<td>ATSDR [1991b] Muskegon County, Michigan</td>
<td>Chemical manufacture (pesticides, herbicides, 3,3'-dichlorobenzidine (DCB))</td>
<td>Exposure to 3,3'-dichlorobenzidine</td>
<td>Unspecified number of family members of workers</td>
<td>Chemical production, including 3,3'-dichlorobenzidine (DCB), began at a site near Muskegon, Michigan in 1960. In 1980-1981, the Michigan Department of Public Health conducted a study of DCB in homes of employees. Samples collected from vacuum cleaner bags from homes of some of the employees had up to 10.5 ppm DCB, and dryer lint contained up to 0.74 ppm. From 0.006 to 0.281 ppm DCB was found in the urine of employees and family members.</td>
</tr>
<tr>
<td>Townsend et al. [1982] Midland, Michigan</td>
<td>Chlorophenol production</td>
<td>Survey for adverse pregnancy outcomes (stillbirths, spontaneous abortions, congenital malformations)</td>
<td>370 Wives</td>
<td>Pregnancy outcome in 370 wives of workers potentially exposed to dioxin formed as a byproduct in the production of 2,4,5-trichlorophenol were compared to outcomes in wives of workers with no exposure to dioxin. Results indicated that there was no statistically significant association between potential for exposure to dioxin and pregnancy outcome. Exposure potential was categorized on the basis of job classification and wipe tests of plant surfaces.</td>
</tr>
<tr>
<td>Smith et al. [1982] New Zealand</td>
<td>Herbicide applicators</td>
<td>Surveyed for miscarriages, congenital defects</td>
<td>989 Applicators and wives</td>
<td>Applicators who sprayed 2,4,5-trichlorophenol products, reported to contain the contaminant, an animal teratogen, were surveyed to determine pregnancy outcomes. The wives of New Zealand sprayers reported helping their husbands spray and handle the herbicide. No detectable reproductive effects were reported.</td>
</tr>
<tr>
<td>Bagnell and Ellenberger [1977] Halifax, Canada</td>
<td>Dry-Cleaning</td>
<td>Obstructive jaundice and hepatomegaly</td>
<td>Daughter</td>
<td>The breast-fed daughter of a woman who regularly visited the father during lunch at a dry-cleaning establishment developed jaundice. The mother's blood contained 0.3 mg/dL tetrachloroethylene (TCE) and the breast milk, 1.0 mg/dL. No TCE was present in the blood of the infant when examined 1-week after breast feeding was stopped. Liver function returned to normal.</td>
</tr>
<tr>
<td>Author (year)</td>
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<td>Study Design</td>
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<tr>
<td>Ehrenberg et al. [1986, 1991] Hudson et al. [1985, 1987]</td>
<td>Vermont</td>
<td>Thermometer plant</td>
<td>60 Workers' children 32 Control children</td>
<td>Workers' children had urine mercury levels of 25 μg/L vs 5 μg/L controls. Childrens' urine levels correlated with worker parents' levels. No clinical effects were found in the children.</td>
</tr>
<tr>
<td>ATSDR [1990a]</td>
<td>Tennessee</td>
<td>Chemical workers</td>
<td>115 Members of exposed workers' families.</td>
<td>Urine mercury levels of family members in normal range, mean 5.1 ± 4.4 ng/mL.</td>
</tr>
<tr>
<td>Haddad and Stenberg [1963]</td>
<td>California</td>
<td>Gold extraction in the home</td>
<td>Case report</td>
<td>Husband and wife had acute bronchitis, fever, chills and nausea. Urine mercury levels, 540 μg/L in husband, 80 μg/L in wife.</td>
</tr>
<tr>
<td>King [1954]</td>
<td>Arizona</td>
<td>Gold extraction in the home</td>
<td>Case report</td>
<td>Husband had severe coughing, vomiting and cyanosis; wife did not report symptoms.</td>
</tr>
<tr>
<td>Hatch [1990]</td>
<td>Arizona</td>
<td>Gold extraction in the home</td>
<td>Case report</td>
<td>Woman had gastrointestinal disturbances. Blood mercury level after 3 weeks of chelation was 193 ng/dL.</td>
</tr>
<tr>
<td>AUTHOR (YEAR) LOCATION</td>
<td>CONTAMINANT</td>
<td>INDUSTRY</td>
<td>STUDY DESIGN</td>
<td>RESULTS</td>
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<td>--------------------------------------------------------------------------</td>
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<tr>
<td>Katzenellenbogen [1956]</td>
<td>Diethylstilbestrol</td>
<td>Pharmaceutical manufacturing</td>
<td>Case reports</td>
<td>5 children of pharmaceutical workers developed hyperestrogenic syndromes.</td>
</tr>
<tr>
<td>Kloorin and Bantine [1956]</td>
<td>Diethylstilbestrol</td>
<td>Pharmaceutical manufacturing</td>
<td>Case report</td>
<td>6 children of employees who worked with estrogens developed hyperestrogenic syndromes.</td>
</tr>
<tr>
<td>Pacynski and Robaczynski [1968] Poland</td>
<td>Diethylstilbestrol</td>
<td>Pharmaceutical manufacturing</td>
<td>Case report</td>
<td>6 children of employees who worked with estrogens developed hyperestrogenic syndromes.</td>
</tr>
<tr>
<td>Pacynski et al. [1971] Poland</td>
<td>Diethylstilbestrol</td>
<td>Pharmaceutical manufacturing</td>
<td>Case report</td>
<td>6 children of employees who worked with estrogens developed hyperestrogenic syndromes.</td>
</tr>
<tr>
<td>Aw et al. [1985] Indiana</td>
<td>Zeranol</td>
<td>Pharmaceutical manufacturing</td>
<td>Cross-sectional study of employees working with hormones</td>
<td>3 male children of current workers and two children of former workers determined to have breast enlargement.</td>
</tr>
<tr>
<td>Bierbaum [1993] Kansas</td>
<td>Diethylstilbestrol</td>
<td>Feedlot repair</td>
<td>Case reports</td>
<td>1976 NIOSH memos dealing with 4 children (ages 3 and 6) with gynecomastia and pubic hair.</td>
</tr>
<tr>
<td>Author (Year) Location</td>
<td>Contaminant</td>
<td>Industry</td>
<td>Study Design</td>
<td>Results</td>
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<tr>
<td>Wilken-Jensen [1983] Denmark</td>
<td>Animal allergen</td>
<td>Veterinary medicine</td>
<td>Case report</td>
<td>Children suffered from asthma every time they went to work with their father or every time he came home from work.</td>
</tr>
<tr>
<td></td>
<td>Grain dust</td>
<td>Miller</td>
<td>Case report</td>
<td>Son developed asthma if the father did not change clothes when he came home from the mill.</td>
</tr>
<tr>
<td></td>
<td>Mushroom mycelium</td>
<td>Mushroom farming</td>
<td>Case report</td>
<td>The son developed allergic symptoms when mushroom mycelium was exchanged.</td>
</tr>
<tr>
<td>Venables and Newman-Taylor [1989] United Kingdom</td>
<td>Animal allergen</td>
<td>Laboratory animal work</td>
<td>Case report</td>
<td>Husband developed asthma due to animal allergens brought home on the wife's person.</td>
</tr>
<tr>
<td></td>
<td>Platinum</td>
<td>Precious metal refining</td>
<td>Case report</td>
<td>Wife developed recurrence of asthma after change in husband's job.</td>
</tr>
<tr>
<td>U.S. Senate [1991a] North Carolina</td>
<td>Otto fuel</td>
<td>Hazardous waste incineration</td>
<td>Case report</td>
<td>2 children developed severe asthma after exposure to hazardous waste (primarily thought to be Otto fuel) on parental clothing.</td>
</tr>
<tr>
<td>Klemmer et al. [1975] Hawaii</td>
<td>Arsenic</td>
<td>Wood treatment: pesticide use</td>
<td>Survey of arsenic in house dust</td>
<td></td>
</tr>
<tr>
<td>Falk et al. [1981] United States</td>
<td>Arsenic</td>
<td>Copper smelter</td>
<td>Case report</td>
<td>Hepatic angiosarcoma</td>
</tr>
<tr>
<td>AUTHOR (YEAR) LOCATION</td>
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<tr>
<td>Carvalho et al. [1986] Brazil</td>
<td>Cadmium</td>
<td>Lead smelter</td>
<td>Cohort of 396 children ages 1-9 years living less than 900 m from primary lead smelter.</td>
<td>Geometric mean and standard deviation of CdB were 0.087 and 2.5 μg/L resp., range 0.004-0.511. 380 children (96%) had CdB &gt; than 0.0089 μg/L. The relationship between parental employment in the smelter and children’s CdB levels was not significant, but the CdB level was significantly (0.0001) higher among children living in households in which “smelter dross” (an industrial residue obtained from lead ore containing variable content of Cd and used for paving) was present, than in children in whose households smelter dross was not found. Higher CdB was significantly (0.00001) associated with shorter distance from home to smelter.</td>
</tr>
<tr>
<td>Brockhous et al. [1988] Stolberg</td>
<td>Cadmium</td>
<td>Lead and zinc smelters</td>
<td>Cohort of 9 children from families of lead workers and 193 children from other families, ages 4-17 years.</td>
<td>Children from families of lead workers (n = 5) had significantly higher GM CdU of 0.34 (GSD 2.6) than children from other families whose GM CU was 0.13 (GSD 2.2), p &lt; 0.01. CdB among children from families of lead worker were higher than in children from other families but the difference was not statistically significant.</td>
</tr>
<tr>
<td>Carvalho et al. [1989] Brazil</td>
<td>Cadmium</td>
<td>Lead smelter</td>
<td>Cohort of 263 children 1-9 years old, living less than 900 m from lead smelter. Measured cadmium in hair.</td>
<td>The mean cadmium in hair was significantly (P &lt; 0.0001) higher at 5.0 ppm for children whose fathers worked in the lead smelter than for children whose fathers had other jobs (3.7 ppm).</td>
</tr>
<tr>
<td>Author</td>
<td>Year</td>
<td>Location</td>
<td>Contaminant</td>
<td>Industry</td>
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<tr>
<td>Maravelias et al.</td>
<td>1989</td>
<td>Greece</td>
<td>Cadmium</td>
<td>Lead smelter</td>
</tr>
<tr>
<td>Hofstetter et al.</td>
<td>1990</td>
<td>Germany</td>
<td>Cadmium</td>
<td>Lead smelter</td>
</tr>
<tr>
<td>Madoff</td>
<td>1962</td>
<td>Unknown</td>
<td>Fibrous glass</td>
<td>Home</td>
</tr>
<tr>
<td>Abel</td>
<td>1966</td>
<td>New York</td>
<td>Fibrous glass</td>
<td>Home</td>
</tr>
<tr>
<td>Peachev</td>
<td>1967</td>
<td>Unknown</td>
<td>Fibrous glass</td>
<td>Home</td>
</tr>
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<tr>
<td>Woody et al. [1986]</td>
<td>RDX (cyclotrimethylenetri nitramine)</td>
<td>Explosives manufacture</td>
<td>Case report</td>
<td>Child developed episodes of status epilepticus.</td>
</tr>
<tr>
<td>Author [year] Location</td>
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<td>Industrial Hygiene Methodology</td>
<td>Industrial Hygiene Observation</td>
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<tr>
<td>Bohme and Cohen [1985] Ohio</td>
<td>Beryllium</td>
<td>Beryllium production</td>
<td>Fabrics exposed at worksite.</td>
<td>Electrostatic charges increased fabric contamination of cotton, but not Nomex fabric loading up to 2,800 µg/m².</td>
</tr>
<tr>
<td>Chamberlin et al. [1957] Pennsylvania</td>
<td>Beryllium</td>
<td>Beryllium plant</td>
<td></td>
<td>5 patients were family members of beryllium workers and cleaned their dusty clothes.</td>
</tr>
<tr>
<td>Chesner [1950] Ohio</td>
<td>Beryllium</td>
<td>Beryllium plant</td>
<td>Case histories of chronic pulmonary granulomatosis</td>
<td>Woman used empty beryllium ore bags for dish clothes. She got the bags from a neighbor who worked at the plant.</td>
</tr>
<tr>
<td>Cohen and Pietz [1986] Ohio</td>
<td>Beryllium</td>
<td>Beryllium refinery</td>
<td>New and used shirts worn at work were analyzed for beryllium in the fabric. Fabrics were agitated in a glove box to measure re-suspended beryllium.</td>
<td>Beryllium concentrations ranged from 12 to 37 mg/m³ in unwashed shirt fabric. Air concentration of beryllium measured in refinery was only a fraction of the PEL of 0.002 mg/m³. The old shirts also showed significantly higher concentrations of beryllium and resuspended significantly higher quantities of beryllium to the air than newer shirts.</td>
</tr>
<tr>
<td>Eisenbud et al. [1949] Ohio</td>
<td>Beryllium</td>
<td>Beryllium production</td>
<td>Study of dust generated by laundry procedures using 100 uniforms worn for one day by plant employees.</td>
<td>Air concentrations of beryllium: shaking soiled clothes 0.1-1.2 mg/m³; scrubbing 3.7 µg/m³; shaking and folding washed clothes 4.6 µg/m³. Estimated inhalation dose during single home-cleaning of work clothes was 17 µg of beryllium.</td>
</tr>
<tr>
<td>Anderson et al. [1979b] New Jersey</td>
<td>Asbestos</td>
<td>Asbestos product manufacturing</td>
<td>Evaluate health status of 679 household contacts of asbestos workers. Interviews, X-rays, physical exams.</td>
<td>Family contacts had no other known asbestos exposure. 35% of household contacts had radiographic abnormalities vs. 5% of controls.</td>
</tr>
<tr>
<td>Author [year] Location</td>
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<tr>
<td>Belanger et al. [1979] Illinois</td>
<td>Asbestos</td>
<td>Vinyl asbestos &amp; asphalt asbestos floor covering manufacturing</td>
<td>Personal &amp; area sampling</td>
<td>Separate lockers for clean and dirty clothes. Coveralls provided. Showers provided.</td>
</tr>
<tr>
<td>Bianchi et al. [1987] Italy</td>
<td>Asbestos</td>
<td>Asbestos related industries</td>
<td>Interviews to determine occupational histories and practices of washing clothes at home.</td>
<td>For women, cleaning work clothing was the main source of exposure. Many women were exposed in this way for more than 20 years.</td>
</tr>
<tr>
<td>Driscoll and Elliott [1990] Michigan</td>
<td>Asbestos</td>
<td>Manufacturing of asbestos brake linings; production of adhesives, sealers and paints.</td>
<td>Vacuumed samples of work clothes and car seat via phase contrast microscopy (PCM) and transmission electron microscopy (TEM).</td>
<td>Vacuum Samples: all clothing (n=7) was contaminated with asbestos, 4 of 6 samples from workers car seats were contaminated with asbestos.</td>
</tr>
<tr>
<td>Claretli et al. [1992] Italy</td>
<td>Asbestos</td>
<td>A variety of work performed by shipyard workers, dock workers and sailors</td>
<td>Review of 170 clinical cases in which necropsies were performed.</td>
<td>5 cases had domestic exposures to asbestos in cleaning the work clothes of their husbands who were employed in shipyards.</td>
</tr>
<tr>
<td>Gibbs et al. [1990] United Kingdom</td>
<td>Asbestos</td>
<td>Shipyard, lagging building and ordinance work</td>
<td>Study of 10 non-occupational cases of mesothelioma</td>
<td>9 cases were due to exposures to asbestos from washing husbands' work clothes.</td>
</tr>
<tr>
<td>Huncharek et al. [1989] USA</td>
<td>Asbestos</td>
<td>Exposure of spouse to contaminated clothing for 34 years.</td>
<td>Post mortem fiber counts from removed lung</td>
<td>Fibers/g of wet lung tissue: Chrysotile 1.72 x 10^4 Amosite/crocidolite 59 x 10^3 Tremolite/actinolite/anthophyllite 221 x 10^3</td>
</tr>
<tr>
<td>Author [year] Location</td>
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<tr>
<td>Kominsky et al. [1990] Ohio</td>
<td>Asbestos</td>
<td>Wet &amp; dry vacuuming to remove asbestos fibers from contaminated carpets and determination of airborne levels during vacuuming.</td>
<td>Transmission electron microscopy was used to analyze air samples collected during vacuuming. An EPA method was used for analysis of carpet samples. Carpets contaminated at 1 million and 1 billion asbestos structures/ft². Decontamination by HEPA filtered dry vacuum and hot-water extraction cleaners.</td>
<td>Wet vacuuming reduced carpet contamination by 70%. There was no significant evidence of change following dry vacuuming.</td>
</tr>
<tr>
<td>Nicholson et al. [1980] California &amp; Newfoundland</td>
<td>Asbestos</td>
<td>Workers' home contamination from mining and milling asbestos.</td>
<td>Air samples were collected in homes of asbestos workers.</td>
<td>13 Chrysotile asbestos samples ranged from &gt; 50 to &gt; 2,000 &lt; 5,000 ng/m³.</td>
</tr>
<tr>
<td>NIOSH [1971] Ohio</td>
<td>Asbestos</td>
<td>Laundering-transfer of fibers to other clothes and cleaning solution.</td>
<td>An asbestos-containing coat (8% asbestos) was dry cleaned with non-asbestos containing clothes.</td>
<td>Demonstrated substantial transfer of fibers to other clothes and to the dry cleaning fluid.</td>
</tr>
<tr>
<td>Sawyer [1977] Connecticut</td>
<td>Asbestos</td>
<td>Laundry</td>
<td>Air samples collected on membranes filter analyzed by phase contrast microscopy. Samples collected in building laundry, not homes.</td>
<td>Asbestos fibers/cm³: Picking up clothing 0.4 Loading washer 0.4 Loading dryer 0.0 Personal 0.4 (up to 1.2)</td>
</tr>
<tr>
<td>Selkas and Ordin [1986] New Jersey</td>
<td>Asbestos</td>
<td>Brake shoe manufacturing</td>
<td>Work clothing was vacuumed as employees left work and dust was analyzed using polarized light microscopy and X-ray diffraction.</td>
<td>Vacuum samples from all work clothes contained chrysotile asbestos fibers. Quantitative data not presented.</td>
</tr>
<tr>
<td>Abbritti et al. [1989] Italy</td>
<td>Lead</td>
<td>Ceramic factories &amp; workshops at home</td>
<td>Sampling of house dust. Methodology not reported.</td>
<td>Mean lead concentrations in house dust were 2.7 and 4.7 mg/m² where workers were exposed in factories and where workshops were adjacent to houses, respectively (vs. 0.8 mg/m² in controls).</td>
</tr>
</tbody>
</table>
Table 15. (Continued) Workers’ Home Contamination-Industrial Hygiene Aspects

<table>
<thead>
<tr>
<th>Author [year] Location</th>
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<th>Industrial Hygiene Observation</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Apoll and Singal [1980] Alaska</td>
<td>Lead</td>
<td>Lead acid storage battery manufacturing</td>
<td>Air sampling in plant</td>
<td>Personal breathing zone samples (5) ranged from 111 to 1,053 μg/m³.</td>
<td>Owner &amp; family lived above plant. Recommended: Installing change room, closing up hallway between plant and home, clean home, and monitor family for lead exposure.</td>
</tr>
<tr>
<td>Baker et al. [1977] Tennessee</td>
<td>Lead</td>
<td>Secondary lead smelter</td>
<td>Sampled homes for lead content in house dust. Analyzed wipe samples by anodic stripping voltametry.</td>
<td>Dust in workers' homes contained lead at 2,687 ppm (&lt;1,000-80,000) vs. 404 ppm for controls.</td>
<td>As a result of study, workers homes were cleaned. Workers started showering and changing clothes before going home.</td>
</tr>
<tr>
<td>Barnett [1994] Oregon</td>
<td>Lead</td>
<td>Tile manufacturing</td>
<td>Not applicable</td>
<td>1 worker had a blood lead level of 73 μg/dL.</td>
<td>Investigators think at least one child (of a worker) has high blood lead.</td>
</tr>
<tr>
<td>Barnett [1994] Oregon</td>
<td>Lead</td>
<td>Bronze foundry</td>
<td>Not applicable.</td>
<td>2 children of foundry workers had high BBLs (14 &amp; 23 μg/dL).</td>
<td>Oregon OSHA documented that employees were taking lead dust home on clothes.</td>
</tr>
<tr>
<td>Carvalho et al. [1984] Brazil</td>
<td>Lead</td>
<td>Lead smelter</td>
<td>Blood lead samples analyzed via atomic absorption spectrophotometry with heated graphite furnace atomizer.</td>
<td>Children of lead workers had higher blood lead values (67.5 μg/dL) than other children (36.6 μg/dL). Lead workers took used &quot;filters&quot; home from the plant, for re-use in their home.</td>
<td></td>
</tr>
<tr>
<td>CH2M Hill [1991] Idaho</td>
<td>Lead</td>
<td>Lead smelter</td>
<td>Vacuuming and shampooing; analyses for lead and other metals.</td>
<td>Lead loading in carpets ranged from 12 to 283 mg/ft² and in furniture from 37 to 1,100 mg/ft², only 14-30% of lead was removed from carpets; 5-40% from furniture.</td>
<td>500 μg/g (lead/total dust) action level for cleanup advisory.</td>
</tr>
<tr>
<td>Cook et al. [1993] Colorado (Leadville)</td>
<td>Lead</td>
<td>Lead mining and smelting</td>
<td>Work practices questionnaire. Samples of floor dust, window sill dust, paint and tap water were analyzed for lead.</td>
<td>Lead in window sill dust 30-27,900 ppm, floor dust 8-11,000 ppm.</td>
<td>Positive association between miner wearing work clothes home and BLLs in children.</td>
</tr>
<tr>
<td>Czachur et al. [1995] New Jersey</td>
<td>Lead</td>
<td>Various lead industries</td>
<td>Telephone interviewers asked about showering at work, washing clothes at home, etc.</td>
<td>The data indicate that elevated blood lead levels in children are associated with parents washing dirty work clothing at home.</td>
<td>Data collected after June 1992.</td>
</tr>
<tr>
<td>Author [year] Location</td>
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<tr>
<td>Dolcourt et al. [1978]</td>
<td>Lead</td>
<td>Battery factory</td>
<td>Carpet vacuum samples</td>
<td>Average concentrations of lead in house dust were 1,700 to 47,534 ppm in 6 homes. Clothes closets demonstrated particularly large degrees of contamination, up to 84,000 ppm. Cars contained lead in dust at an average of 2,770 ppm.</td>
<td>Contaminated work clothing was the probable home lead contamination source; paint, water supply and air were ruled out as sources of lead.</td>
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<tr>
<td>See also CDC [1977b]</td>
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<td>North Carolina</td>
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<tr>
<td>Dolcourt et al. [1981]</td>
<td>Lead</td>
<td>Battery recycling. Battery castings were burned as fuel in one family's wood burning stove.</td>
<td>Dust samples collected by vacuum. Decontamination with Calgon®.</td>
<td>Dust from kitchen floor contained lead at 41,283 ppm (13.5 mg/m²); from bedroom floor 6,800 ppm (3.3 mg/m²); from living room floor 5,862 ppm (5.9 mg/m²); from sofa 13,283 ppm. About 5.5% was removed by a single decontamination procedure.</td>
<td>Battery recycling out of the home kitchen.</td>
</tr>
<tr>
<td>North Carolina</td>
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<tr>
<td>Donovan [1994a, 1994b]</td>
<td>Lead</td>
<td>Stained glass studio.</td>
<td>Air samples at work and home. Wipe samples at home and studio. Vacuum carpet at home.</td>
<td>Home-air, home-wipe and carpet-vacuum samples all were below the detection limit. Studio wipe samples = 1.2 to 1,600 mg/m². Breathing-zone air samples in studio = 0.1 to 80 μg/m³. General-areas air samples in studio ≤ 2 μg/m³; home and outside &lt; 0.1 μg/m³.</td>
<td>The studio adjoins the home. Engineering controls &amp; hygiene practices account for low blood lead and low lead in the home. The simple, low-cost control techniques used at the studio could benefit others in the trade. Dog and child not permitted in studio.</td>
</tr>
<tr>
<td>Ohio</td>
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<tr>
<td>Everts et al. [1994a, 1995]</td>
<td>Lead</td>
<td>Abrasive blasting of lead-based-paint from bridge</td>
<td>Wipe samples for lead taken from hands and faces of workers and smooth surfaces of personal vehicles, vacuum samples of car floor carpets and seats, and gauze patches were attached to workers clothing.</td>
<td>Lead on; unwashed faces 4-1,800 μg/wipe; washed 4-1,600 μg/wipe; unwashed hands 4-5,600 μg/wipe; washed hands 1-920 μg/wipe; clothing 1-7,700 μg/gauze pad (&lt;1-1,200 μg/cm²); cars up to 2,000 μg/cm² on floor, 1,100 μg/cm² on driver's arm rest.</td>
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<tr>
<td>See also Fiaschetti et al. [in press]</td>
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<tr>
<td>Connecticut</td>
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<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Ewers et al. [1994b] Ohio</td>
<td>Lead</td>
<td>Decontamination of carpets and floors</td>
<td>Carpets-repeated HEPA vacuuming. Wooden floors-dry HEPA vacuuming. Linoleum floors-dry HEPA vacuuming followed by wet washing. Collected dust was analyzed for lead contamination.</td>
<td>Lead concentration in dust remained about the same from first to tenth carpet cleaning. Over 95% of total dust was removed from bare wooden floors after dry HEPA vacuuming only. Dry HEPA vacuuming removed over 75% of total dust from linoleum floors. Wet washing removed an additional 20%.</td>
<td>Lead loading on carpet surface may increase if vacuuming is not done for sufficient time. It may be more practical to replace rather than clean contaminated carpets.</td>
</tr>
<tr>
<td>Fischbein et al. [1992] USA</td>
<td>Lead</td>
<td>Home pottery work</td>
<td>Blood lead</td>
<td>BLL: worker 48 μg/dL, daughter 54 μg/dL, spouse normal. Art studio was separated from the family home by a curtained-off studio. Daughter (5-years-old) spent significant time in studio with artist mother.</td>
<td></td>
</tr>
<tr>
<td>Gittleman et al. [1991, 1994] Alabama</td>
<td>Lead (Pb)</td>
<td>Battery recycling</td>
<td>Wipe samples in employee autos. Work practices observations.</td>
<td>Workers did not consistently shower and change clothes before leaving work. 3 mg Pb/100 cm² on drivers seat 1.9 mg Pb/100 cm² on driver’s floor area. 1.7 mg Pb/100 cm² on dashboard.</td>
<td></td>
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<tr>
<td>Grandjean and Bach [1986] Denmark</td>
<td>Lead</td>
<td>Secondary lead smelter</td>
<td>Airborne lead in socks</td>
<td>When air lead &lt; 0.1 mg/m³, socks contained 0.13-1.62 g lead/pair. When air lead &gt; 0.1 mg/m³, socks contained 0.06-2.19 g lead/pair. Indicates that all clothes worn at work should not be taken home.</td>
<td></td>
</tr>
<tr>
<td>Gunter et al. [1987] Colorado, Nevada</td>
<td>Lead</td>
<td>Fire assay procedures</td>
<td>Air sampling for lead dust &amp; fume.</td>
<td>General room air contained lead up to 600 μg/m³. The report recommends that the workers should shower and change clothes before leaving the workplace to prevent exposure of family members to lead-contaminated work clothes.</td>
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<tr>
<td>Kawai et al. [1983] Japan</td>
<td>Lead</td>
<td>Cottage industries: quench-hardening &amp; type-printing.</td>
<td>Air and surface sampling of dust in 2 homes where family members had elevated BLL.</td>
<td>Air in workshop: 2 to 50 µg/m³ Surface: 200 to 20,386 ppm of lead in total dust. Workshop surfaces: Floor 3,000 to 20,000 ppm Desk 4,500 ppm Window frame 1,400 ppm. Living area surfaces: Carpets 1,000 to 5,000 ppm Window frame 260 ppm Television 380 ppm Dining room floor 678 ppm.</td>
<td></td>
</tr>
<tr>
<td>Kaye et al. [1987] Colorado</td>
<td>Lead</td>
<td>Electronics components plant making ceramic-coated capacitors &amp; resistors.</td>
<td>Workplace area samples contained lead at 60-1,700 µg/m³. Dust samples were taken from vacuum cleaners of both worker and non-worker homes, and were analyzed for lead content.</td>
<td>Lead in 11 dust samples from workers' homes, ranged up to 3,400 ppm, and in 9 samples from non-worker homes it ranged from up to 320 ppm.</td>
<td>No protective clothing or worksite showers were used to prevent dust from being taken home from the plant.</td>
</tr>
<tr>
<td>Koplan et al. [1977] Barbados</td>
<td>Lead</td>
<td>Pottery cottage industry leadgazing</td>
<td>22 wipe samples of dust in work and living areas of 6 dwellings.</td>
<td>20 samples had lead in dust &gt; 1,000 µg/g. 15 samples from living areas averaged 5,000 µg/g. Lead in dust ranged from 393 µg/g from a floor in the dining room in one home to 325,892 µg/g from a table surface where glazing was done.</td>
<td></td>
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<tr>
<td>Landrigan et al. [1980] Georgia</td>
<td>Lead</td>
<td>Stained glass window production in commercial studios</td>
<td>Lead levels in bulk-dust samples in 1 home and 1 studio.</td>
<td>Mean lead concentration in studio (3 samples) was 10,696 ppm. Mean concentration in home (2 samples) = 355 ppm.</td>
<td></td>
</tr>
<tr>
<td>Lundquist [1980] USA</td>
<td>Lead</td>
<td>Manufacturing of lead batteries.</td>
<td>Informing workers of hazards of home contamination and preventive measures.</td>
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<tr>
<td>Martin [1974], Martin et al. [1974] United Kingdom</td>
<td>Lead</td>
<td>Smelters, lead and other mines.</td>
<td>Lead in clothing and automobiles.</td>
<td>Lead was found in undenoting, socks, boots and cars of workers, data were not reported.</td>
<td>Factory managers were asked to assure that workers used the cleaning facilities available so that they did not take lead dust home on their bodies, clothing, and footwear.</td>
</tr>
<tr>
<td>Matte and Burr [1989]; Matte et al. [1989] Jamaica</td>
<td>Lead</td>
<td>Cottage industry: &quot;backyard battery repair shops&quot;</td>
<td>Air, dust, and soil</td>
<td>Lead in air, geometric mean = 21 μg/m³ in workshops. Lead in household dust, range = 190 to 53,140 μg/m². Lead in soil, 51 to 400,000 ppm.</td>
<td>Changing work clothes was not associated with lower house dust lead levels. None of the facilities surveyed had adequate shower and changing facilities. Threshold for soil lead = 500 ppm; for house dust 1,500 μg/m². Playing in shop area was associated with higher BLLs in children.</td>
</tr>
<tr>
<td>Matte et al. [1991] Jamaica</td>
<td>Lead</td>
<td>Conventional &amp; cottage lead smelters</td>
<td>House dust and soil samples from cottage smelters. Dust samples collected from floor of room in which children spent most of their time.</td>
<td>Lead in house dust, 100 to 109,000 μg/m²; in soil, 9 to 320,000 ppm.</td>
<td></td>
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<tr>
<td>McCammon et al. [1991] Utah</td>
<td>Lead</td>
<td>Lining of 2 large tanks with lead sheets.</td>
<td>Wipe samples for surface lead including employees' street shoes and in employees' cars.</td>
<td>Lead on boots and shoes 1-20 μg/cm²; on shirt collars 0.3-2 μg/cm²; on floor of cars 0.3-4 μg/cm².</td>
<td>The authors state: &quot;The opportunity for lead exposure was likely increased by the lack of shower facilities &amp; the practice of wearing work clothes at home.&quot; Recommendation: install clean &amp; dirty change rooms, and provide laundry facilities at work.</td>
</tr>
<tr>
<td>Menrath et al. [1993] USA</td>
<td>Lead</td>
<td>Mining</td>
<td>Dust samples collected from the cars &amp; homes of both miners &amp; non-miners in the same community.</td>
<td>Mean lead concentrations in dust = 3,909 ppm in miners cars vs. 917 ppm in non-miners cars. Lead loadings on car floors = 3,539 μg/ft² for miners vs. 565 μg/ft² for non-miners. Lead loadings in homes were similar for both groups, approx. 56 μg/ft². Concentration of lead in house dust was higher for miners.</td>
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<tr>
<td>Milar and Mushak [1982] North Carolina</td>
<td>Lead</td>
<td>Home contamination of carpets containing 1,152-11,148 ppm of lead</td>
<td>Calgon solution in Steamex carpet cleaner followed by detergent cleaner 24 hours later.</td>
<td>Detergent cleaning alone had little effect on lead expressed either as ppm in dust or lead in μg/m². Calgon treatment followed by detergent reduced contamination by 80% on the average.</td>
<td>Details of decontamination procedure are given. Effectiveness demonstrated by concomitant reduction in child's blood lead.</td>
</tr>
<tr>
<td>CDC [1977b] See also Dolcourt et al. [1978] North Carolina</td>
<td>Lead</td>
<td>Battery factory</td>
<td>Lead in air, dust, paint &amp; drinking water from 7 workers' homes and soil samples from cars that were driven to work.</td>
<td>No lead contamination in paint or water and no airborne lead exposures from factory emissions or busy roadways. Lead in house dust 1,693 to 84,074 μg/g. Highest levels were from closet where work clothes were stored (mean 31,840 μg/g). Mean lead level in dust from cars was 2,770 μg/g.</td>
<td>The employer made changes to reduce worker &amp; family exposures, including exhaust ventilation, providing coveralls &amp; improved shower facilities. Contaminated homes were thoroughly cleaned.</td>
</tr>
<tr>
<td>CDC [1992a] Utah</td>
<td>Lead</td>
<td>Welding and soldering with lead</td>
<td>Wipe samples.</td>
<td>Shoes had lead at 4-20 μg/cm². Floor of car had lead at 4 μg/cm².</td>
<td></td>
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<tr>
<td>Morton et al. [1982] Oklahoma</td>
<td>Lead</td>
<td>Battery making</td>
<td>Questionnaires designed to ascertain potential lead exposure in homes.</td>
<td>Blood lead levels in children related to workers' potential for exposure to lead and to work practices.</td>
<td>The authors recommend controls: showering, shampooing, changing clothes and shoes before leaving work. They also noted that only changing clothes did not reduce the risk.</td>
</tr>
<tr>
<td>Nelson and Clift [1992] Oklahoma</td>
<td>Lead</td>
<td>Foundry</td>
<td>Samples were collected from homes of foundry &amp; non-foundry workers in a rural town. Samples collected included dust from carpeting, dash of work vehicle, and clothing. Samples were analyzed by atomic absorption with a graphite furnace.</td>
<td>Lead was detected in all homes with levels in carpets ranging from 105 ppm to 1,535 ppm.</td>
<td></td>
</tr>
<tr>
<td>Osorio [1994] California</td>
<td>Lead</td>
<td>Cutting down lead-sheathed cable</td>
<td>Lead in worker's home and yard.</td>
<td>Lead in backyard soil at 1,500 μg/dL; lead in indoor housedust at 1,700 ppm.</td>
<td>Worker wore dirty clothing home and laundered it with family laundry. Also took lead-contaminated used telephone poles home for firewood.</td>
</tr>
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<tr>
<td>Piacitelli and Whelan [1995] Ohio</td>
<td>Lead</td>
<td>Bridge repair</td>
<td>Wipe and vacuum samples for dust in automobiles and homes of workers. Samples of loose paint and water supply analyzed for lead.</td>
<td>Lead loading (µg/m³): Main entry floor, 2,031 Change area floor, 2,276 Washing machine lid, 801 Sofa/chair, 639 Driver's seat, 5,647 Floor of car, 10,653 Steering wheel, 790 Worker’s hands, 3,131 Family member’s hands, 140</td>
<td>Data from homes with lead in paint &gt; 0.5% excluded.</td>
</tr>
<tr>
<td>Piacitelli and Rice [1993] Ohio</td>
<td>Lead</td>
<td>Radiator repair shops</td>
<td>Surface wipe sampling of workers’ cars, hands and foreheads. Evaluation of hand washing.</td>
<td>Lead in cars up to 96,000 µg/m², on foreheads of workers, 6,000 µg/m²; on unwashed hands, 78,050 µg/m²; average on unwashed hands, 678 µg/m²; after washing hands, 593 µg/m².</td>
<td></td>
</tr>
<tr>
<td>Piacitelli et al. [in press] See also Ewers et al. [1994a, 1995] Connecticut</td>
<td>Lead</td>
<td>Bridge work: lead-based paint abatement</td>
<td>Wipe and vacuum samples were collected in 27 automobiles of abrasive blasters and other lead-exposed workers.</td>
<td>Lead contamination was found in all automobiles. The mean lead loading was lower in abrasive blasters’ cars than in other workers’ cars: 370 µg/m² vs. 2,000 µg/m². Lead loadings on floors and seats of cars were 340-2,000 µg/m² and on other surfaces &lt; 500-1,900 µg/m².</td>
<td>Abrasive blasters regularly changed out of work clothing and showered before entering their cars, whereas the other workers did not. Half the workers regularly had child passengers.</td>
</tr>
<tr>
<td>Pichette et al. [1989] Texas</td>
<td>Lead</td>
<td>Battery manufacture Batteries manufacture/Battery recycling Battery recycling</td>
<td>BLL of spouse and other adult household members by laundry practice.</td>
<td>When laundered by company BLLs were 8.4 µg/100 mL; spouse, 13.0 µg/dL; other household member, 15.6 µg/dL.</td>
<td></td>
</tr>
<tr>
<td>Pitts [1986] Garrettson [1988] Virginia</td>
<td>Lead</td>
<td>Radiator repair</td>
<td>Wipe samples of lead in dust at home and in car; area sampled not reported.</td>
<td>Lead (µg/filter) in: Bathroom closet, 183 Kitchen floor, 284 Floor board of car, 7,580 Driver’s seat, 1,295 Workers Shoes, 11,030</td>
<td>Living room paint had lead at 0.03%; yard soil had lead at 39 ppm. The father did not change clothes or shower at his work site. The highest lead level in the house was near the washing machine. Children frequently played in the car.</td>
</tr>
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<tr>
<td>Pollock [1994] Mississippi</td>
<td>Lead</td>
<td>Trucking lead and lead products</td>
<td>Lead in dust at home.</td>
<td>Lead found on surfaces in home up to 20 μg/t² on worker's shoes at 240 μg/ft².</td>
<td>200 μg/ft² is limit for dust on floors. Change clothes &amp; shoes before entering home, wash work clothes separately, clean or wash floors, carpets, furniture and toys.</td>
</tr>
<tr>
<td>Que Hye et al. [1985] Ohio</td>
<td>Lead</td>
<td>Not applicable</td>
<td>Method development for sampling &amp; analysis of house dust and hand contamination.</td>
<td>Recovery of lead dusts from surfaces by single vacuuming was 74%, by five vacuumings, 100%. Recovery of lead from hands by single wipe was 52%, by five wipes, 100%.</td>
<td></td>
</tr>
<tr>
<td>Rice et al. [1978] USA</td>
<td>Lead</td>
<td>Secondary lead smelters</td>
<td>Wipe samples of dust collected in workers' homes and control homes.</td>
<td>Lead content of wipe cloths in workers' homes, 79-112 μg; control homes, 10-29 μg. Lead concentration in settled dust, 3.31 μg/m² in workers' homes; 1.24 in control homes.</td>
<td>More stringent work practices &amp; personal hygiene are required.</td>
</tr>
<tr>
<td>Rinehart and Yangiwa [1993] See also Venable et al. [1993] Massachusetts</td>
<td>Lead, tin</td>
<td>Electric cable splicing Workers' homes contamination measurements compared to control homes</td>
<td>House dust samples collected with a hand-held vacuum, analyzed by X-ray fluorescence.</td>
<td>Laundry areas of workers' homes, 621-1,606 ppm of lead in dust; control homes, 9-1,212 ppm. Other areas of workers' homes, 227-909 ppm of lead in dust; control homes, 121-879 ppm. Tin in workers' homes: laundry areas, 73-242 ppm; other areas, 45-115 ppm. Tin in control homes: laundry areas up to 73 ppm; other areas not detected.</td>
<td>Since workers' exposures to lead were below the OSHA PEL, workers washed their soiled clothes at home.</td>
</tr>
<tr>
<td>Sherlock et al. [1983] United Kingdom</td>
<td>Lead</td>
<td>Lead levels in Caucasian and Asian children</td>
<td>BLLs and hand washing practices.</td>
<td>Children who washed hands before eating had lower BLLs levels than children who did not.</td>
<td>Children should be encouraged to wash hands before eating.</td>
</tr>
<tr>
<td>Simonson and Mechem [1983]</td>
<td>Lead</td>
<td>Smelter</td>
<td>Evaluation of airshowers and automatic shoe cleaners for lead removal from clothing.</td>
<td>Air shower removed 5 to 72% of lead from clothing. No quantitative data reported for shoe cleaners.</td>
<td>Some penetration of lead to underwear.</td>
</tr>
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<tr>
<td>State of Alabama [1992] Alabama</td>
<td>Lead</td>
<td>Pottery making, cottage industry</td>
<td>Lead wipe samples in home and workshop.</td>
<td>Wipe samples in mobile home = 16 to 390 μg/ft² in workshops 440-177,000 μg/ft².</td>
<td>Author notes HUD interim guidelines for clearance criteria for specific indoor surfaces: floor = 200 μg/ft²; window sills = 500 μg/ft²; window wells = 800 μg/ft². Grandmother's living quarters should be moved from the ceramic/pottery shop. Make shop off limits to family's children. Shower and change into clean clothes before entering house.</td>
</tr>
<tr>
<td>Venable et al. [1993] Massachusetts</td>
<td>Lead</td>
<td>Cable splicing in a utility company</td>
<td>Air sampling. Wipe samples from surfaces, employee clothing, boots, and hands before and after washing.</td>
<td>TWA exposures to lead at 0.22 to 17 μg/m³ below OSHA PEL (50 μg/m³). Lead loadings: Service vehicles 48-12,400 μg/ft²; clothes 600-4,800 μg/ft²; hands before washing 1,800-4,900 μg/ft²; after 250-680 μg/ft².</td>
<td>Although most employees showered and changed clothes at the end of the day 91% took work clothes home and 22% washed work clothes and other laundry together.</td>
</tr>
<tr>
<td>Watson et al. [1978] Vermont</td>
<td>Lead</td>
<td>Battery manufacturing</td>
<td>Lead samples in homes; lead in house dust, drinking water and paint. Dust samples analyzed via an anodic stripping voltameter, lead paint samples were analyzed by an X-ray source detector, and water samples were analyzed via atomic absorption spectrophotometry.</td>
<td>Households of workers had higher concentrations of lead in dust (2,239 ppm) than controls (718 ppm).</td>
<td>Authors note that even with a plant program of showering and changing clothes, apparently enough lead reaches the workers homes (on hair, skin, clothing) to result in higher lead levels. Recommendation that plant provide laundered uniforms at work.</td>
</tr>
<tr>
<td>Wiehrdt [1994] Indiana, Illinois, Ohio</td>
<td>Lead</td>
<td>Not identified</td>
<td>OSHA learned that 6 children of workers had elevated lead levels, and conducted wipe samples in children's homes.</td>
<td>Lead in wipe samples ranged up to 240 μg/wipe.</td>
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<tr>
<td>Winegar et al. [1977]</td>
<td>Lead</td>
<td>Secondary lead smelter</td>
<td>Lead in household dust and clothing.</td>
<td>Lead in household dust from 25 households ranging from 120 to 26,000 ppm (2,400 ppm median). Lead in dust and cloth from cuffs of pants worn under coveralls was 60,000-600,000 ppm, and 700-19,000 µg/2.5 cm² respectively.</td>
<td>One of 33 workers showered at work, 8 took work clothes home for cleaning, 21 took home clothes worn under coveralls.</td>
</tr>
<tr>
<td>Devries and Devries [1993] Ontario, Canada</td>
<td>Caustic farm products</td>
<td>Farming</td>
<td>Developed a cover for drums of acid, soap, and chlorine cleaner used on farm.</td>
<td>Preventive measures include guidelines for safe handling.</td>
<td>Published design in &quot;Hoard's Dairyman&quot; for wide distribution to dairy farmers.</td>
</tr>
<tr>
<td>Anderson et al. [1965] Canada</td>
<td>Parathion</td>
<td>Salvage Operation Sheets contaminated in hold of ship during transport</td>
<td>Strips of material analyzed for parathion.</td>
<td>Flannelette sheets contaminated with parathion.</td>
<td>Owner operated salvage business from home and used sheets purchased from insurance company for home use.</td>
</tr>
<tr>
<td>Barnett [1994] Oregon</td>
<td>Chloropirin</td>
<td>Wood treating</td>
<td>Not applicable.</td>
<td>Containers of pesticide dropped on employee's driveway. Wind carried vapors next door.</td>
<td>As result of accident, employer no longer allowed employees to take company cars home.</td>
</tr>
<tr>
<td>Branson and Henry [1982] Michigan</td>
<td>Pesticides</td>
<td>Pesticide application</td>
<td>This is an extension bulletin alerting pesticide users of potential hazard and precautions for workers and families.</td>
<td></td>
<td>Recommends cleaning clothes after every use, keeping contaminated clothing separate from other clothing, and using an extra rinse.</td>
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<tr>
<td>Cannon et al. [1978] Virginia</td>
<td>Kepone</td>
<td>Manufacture</td>
<td>Kepone in whole blood. Interviews.</td>
<td>Kepone in blood of family members 0.003-0.39 ppm; other residents 0.005-0.033 ppm.</td>
<td>2 women who washed husbands clothes developed symptoms of kepone poisoning.</td>
</tr>
<tr>
<td>Clifford and Nies [1989] Colorado</td>
<td>Ethyl parathion</td>
<td>Manufacture</td>
<td>Laundry</td>
<td>After spill, uniform was washed twice and still contained parathion at 70,000 ppm; clothes washed with it contained 135-150 ppm.</td>
<td>Potential poisoning of family members was avoided because company routinely laundered work clothes.</td>
</tr>
<tr>
<td>Finley and Rogillio [1969] Louisiana</td>
<td>1,2-bis-(p-Chlorophenyl)-2,2,2-trichloroethane (DDT) and methyl parathion</td>
<td>Cotton field work in treated fields</td>
<td>Up to 12 ppm of methyl parathion and 136 ppm of DDT were found in the exposed fabrics.</td>
<td>5 shirting-type fabrics were worn in the field for 8 hours.</td>
<td>Recommended delaying reentry to day 4 and wearing 2 layers of clothing.</td>
</tr>
<tr>
<td>Finley et al. [1979] Louisiana See Satoh [1979]</td>
<td>Pesticide (methyl parathion)</td>
<td>Cotton field work in treated fields</td>
<td>All-cotton fabric residue concentrations (21 ppm on day 1) were 49-68% of the cotton-blend residues (35 ppm on day 1). Clothing contamination level on day 4 was 1% of that on day 1. Undergarments attained 55% of the outer garment residue levels.</td>
<td>Cotton &amp; cotton/polyester blends were worn by workers into a cotton field treated with methyl parathion. Samples were collected on 1st, 2nd, and 4th days after spraying.</td>
<td>Recommended delaying reentry to day 4 and wearing 2 layers of clothing.</td>
</tr>
<tr>
<td>Ganellin et al. [1964] Arizona</td>
<td>Parathion</td>
<td>Pesticide application</td>
<td>Case studies</td>
<td>Non-fatal poisonings of pesticide application workers exposed to equipment previously used for organophosphorus insecticide application.</td>
<td>All equipment which has been used for application or handling of toxic insecticides must be considered contaminated and dangerous until a thorough decontamination is performed. Such equipment should be kept in areas forbidden to those who are uninforme of hazards.</td>
</tr>
<tr>
<td>Graves et al. [1980] Louisiana</td>
<td>Permethron</td>
<td>Farming</td>
<td>Fabrics worn for 6 hours in cotton field.</td>
<td>Fabrics contained permethron at about 25 ppm.</td>
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<td>Lavy [1988] Arkansas</td>
<td>Herbicides</td>
<td></td>
<td>Informing applicators of hazards and protective measures.</td>
<td>A total of 23 pesticides were found. From 8 to 18 pesticides were found in each home.</td>
<td>Included in this advisory are recommendations for preventing contamination of the home.</td>
</tr>
<tr>
<td>Lewis et al. [1994] North Carolina</td>
<td>Pesticides</td>
<td>Home Contamination</td>
<td>Sampling for 30 pesticides in carpets in 9 residences.</td>
<td>Atrazine at up to 7 μg/cm².</td>
<td>Demonstrates need to consider many sources when evaluating workers' homes.</td>
</tr>
<tr>
<td>Oakland et al. [1992] North Carolina</td>
<td>Atrazine</td>
<td>Crop-spraying</td>
<td>Atrazine contamination of spray's clothing after 4 hours of work.</td>
<td>Atrazine at up to 7 μg/cm².</td>
<td></td>
</tr>
<tr>
<td>Osorio [1994] California</td>
<td>Pesticides</td>
<td>Farming</td>
<td>Sampled for pesticides in 5 farm workers' homes and 6 non-farmworkers' homes.</td>
<td>In all, 15 pesticides were found in the study. Diazinon, chlordimefens and propoxur were found at much higher concentrations in farm workers' homes.</td>
<td>Sources of pesticides (air, clothing, shoes) was not determined.</td>
</tr>
<tr>
<td>Stone and Stahr [1989] Iowa</td>
<td>Pesticides</td>
<td>Farming</td>
<td>Analysis of applicator's coveralls for pesticides.</td>
<td>Measurable levels of 5 pesticides found even though the coveralls had been washed after each use.</td>
<td>Demonstrates the difficulty of removing pesticides from fabrics by laundering.</td>
</tr>
<tr>
<td>Ware et al. [1973] Arizona</td>
<td>Ethyl and methyl parathion</td>
<td>Farming</td>
<td>Measured contamination of clothing from working in treated cotton fields for 30 minutes.</td>
<td>Blue jeans contained methyl parathion at 6-16 mg/pair and ethyl parathion at about 8 mg/pair. T-shirts contained &lt; 1 mg.</td>
<td></td>
</tr>
<tr>
<td>Wolfe et al. [1981] Washington</td>
<td>Parathion</td>
<td>Agriculture</td>
<td>Analysis of parathion in empty metal drums and effect of rinsing.</td>
<td>After first rinse water contained parathion from 3 to 33 g/gal; after 4 rinses, &lt; 0.15 g/gal.</td>
<td></td>
</tr>
<tr>
<td>Fischbein [1987] USA</td>
<td>Polychlorinated biphenyl (PCB)</td>
<td>Transformer maintenance Home laundry</td>
<td>Serum PCBs in 2 workers and their wives.</td>
<td>One worker's serum PCB level, 69-101 ng/mL; wife's 11-15 ng/mL. Other worker's serum PCB level, 77 ng/mL; wife's 6 ng/mL. In both cases PCB pattern was same as husbands.</td>
<td>The author recommended &quot;appropriate industrial hygiene measures&quot; to prevent home contamination from occupational sources.</td>
</tr>
<tr>
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<tr>
<td>Hartle et al. [1987] Indiana</td>
<td>Polychlorinated biphenyl (PCB)</td>
<td>Aluminum extrusion using PCB contaminated hydraulic fluids</td>
<td>Wipe samples</td>
<td>Wipe sample results up to 900 μg PCB/m². Hand wrench 308 μg PCB/m². Lunch pail 14 μg PCB/m². Gloves 36-160 ppm PCB.</td>
<td>NIOSH proposed a wipe-sample limit 50-250 μg/m² for low contact surfaces. Change room &amp; company laundered coveralls were recommended.</td>
</tr>
<tr>
<td>Hartle [1987] Pennsylvania</td>
<td>Polychlorinated biphenyl (PCB)</td>
<td>Maintenance workers working in PCB-contaminated building in a rail yard</td>
<td>Wipe samples with hexane moistened gauze pads.</td>
<td>Wipe samples from bottom of maintenance pit averaged 90,000 μg PCB/m². Other wipe samples indicated PCB contamination on floors of lunch room, locker rooms, supply room, and foreman's office.</td>
<td>Wipe sampling of clothes and worker-owned tools were not allowed. The workers might have also contaminated their own homes from clothes, shoes and personal tools taken home with them.</td>
</tr>
<tr>
<td>Kominsky [1987a] Florida</td>
<td>Polychlorinated biphenyl (PCB)</td>
<td>Firefighting at a transformer oil reclamation facility. Decontamination of protective clothing.</td>
<td>PCBs extracted from pre-wash samples using toluene. Samples of incident-contaminated clothing &amp; &quot;spiked&quot; clothing. Detergent and water wash. RADKLEEN® clothing decontamination system with Freon®.</td>
<td>After soap and water wash PCB's in clothing 15-1,060 μg/g. After RADKLEEN® treatment PCB's 1.8-25 μg/g in incident-contaminated clothing. RADKLEEN® efficiency was 66 to 99% for incident-contaminated and &gt; 90% for &quot;Spiked&quot; samples.</td>
<td>Safe level of PCB's in clothing not known.</td>
</tr>
<tr>
<td>Orris and Kominsky [1984] Minnesota</td>
<td>Polychlorinated biphenyl (PCB)</td>
<td>Transformer fire at a school</td>
<td>Wipe samples at a school. Decontamination of surfaces by washing with liquid alkaline synthetic detergent.</td>
<td>Initial surface contamination &lt; 0.05 to 20 μg/100cm². Some surfaces required 2 washes.</td>
<td>PCBs were not detected after cleaning.</td>
</tr>
<tr>
<td>Price and Welch [1972] Michigan</td>
<td>Polychlorinated biphenyl (PCB)</td>
<td>General population</td>
<td>Human tissue analyses House-dust samples from homes of workers occupationally exposed to PCBs.</td>
<td>PCBs were found in human tissues during autopsies. Several house-dust samples contained up to 180 ppm PCBs.</td>
<td></td>
</tr>
<tr>
<td>Doherty [1984] Missouri</td>
<td>Dioxin (2,3,7,8- tetrachlorodibenzodioxin)</td>
<td>Community contamination</td>
<td>Soil samples Carpet samples inside house Post clean-up vacuum samples Replaced carpet</td>
<td>Dioxin in soil 48 ppb in front of house; 2.6 ppb in carpet, post clean-up. Dioxin in vacuum dust less than lab detection limit.</td>
<td></td>
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<tr>
<td>Hess [1988] Missouri</td>
<td>Dioxin 2,3,7,8-tetrachlorodibenzodioxin</td>
<td>Decontamination of structures contaminated with dioxin from waste oil used in dust suppression.</td>
<td>Residence and one commercial building were cleaned with HEPA equipped high-efficiency vacuum and every inside surface wiped with a damp cloth and detergent wash.</td>
<td>Residues were satisfactorily cleaned (&lt;4.0 pg/cm²) commercial building contamination reduced from 36.6 ppb to 13 ppb.</td>
<td>Additional decontamination of commercial building included removal of attic insulation and floor replacement.</td>
</tr>
<tr>
<td>Jensen et al. [1972b] United Kingdom</td>
<td>2,3,6,7-tetrachlorodibenzodioxin</td>
<td>Trichlorophenol plant refitting a previously cleaned tank</td>
<td>Two employees, one employee's son &amp; other employee's wife developed chloracne.</td>
<td></td>
<td>The equipment had been contaminated 3 years earlier, and repeatedly steam cleaned. Employees must have contacted a pocket of residual material.</td>
</tr>
<tr>
<td>ATSDR [1991b] Michigan</td>
<td>3,3'-Dichlorobenzidine (DCB)</td>
<td>DCB production</td>
<td>Sampled workers' home for DCB in vacuum cleaner bags and dryer lint.</td>
<td>DCB up to 10.5 ppm was found in vacuum cleaner bags &amp; up to 0.74 ppm in dryer lint. DCB was found in urine of some workers &amp; family members.</td>
<td></td>
</tr>
<tr>
<td>ATSDR [1989b] Michigan</td>
<td>4,4'-methylene-bis(2-chloroaniline) (MOCA)</td>
<td>Plastics manufacture</td>
<td>Vacuum cleaner dust and clothes dryer lint in workers' homes analyzed for MOCA.</td>
<td>MOCA up to 2.6 ppm in vacuum dust and 0.65 ppm in dryer lint.</td>
<td>MOCA in urine of family members up to 12.1 ppb.</td>
</tr>
<tr>
<td>Bagnell and Ellenberger [1977] North Carolina</td>
<td>Tetrachloroethylene (perchloroethylene)</td>
<td>Suede and leather dry cleaning</td>
<td>Gas chromatography headspace procedure of breast milk &amp; venous blood samples.</td>
<td>Mother often ate lunch with husband (30-60 min) in dry cleaning plant.</td>
<td>Evidence suggests that the baby was exposed via breast milk.</td>
</tr>
<tr>
<td>Clapp et al. [1985] Pennsylvania</td>
<td>4,4'-methylene-bis(2-chloroaniline) (MOCA)</td>
<td>Urethane casting</td>
<td>Air samples, Surface wipes, Hand contact monitors, Urine samples</td>
<td>Except for 2 surface wipes which had MOCA &gt; 5.3 µg/wipe, measurements were below detection level. However, urine samples for 6 workers ranged from 2 to 36 µg MOCA/L.</td>
<td>Company should issue and launder clothing daily, including underwear, require showering before going home. Shoes and disposable shoe covers should be provided.</td>
</tr>
<tr>
<td>Schreiber et al. [1993] New York</td>
<td>Tetrachloroethylene</td>
<td>Dry cleaning</td>
<td>Air sampling in apartments above dry cleaning establishments.</td>
<td>Tetrachloroethylene at 100-440 µg/m³ in apartments above dry-to-dry units, 1,350-17,000 µg/m³ above transfer units.</td>
<td>Although workers' families were not identified as living in the apartments, such a &quot;cottage industry&quot; situation could exist.</td>
</tr>
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<tr>
<td>Stasiuk [1993] (see also Schneider et al. [1999], New York)</td>
<td>Perchloroethylene (PCE)</td>
<td>Dry cleaning</td>
<td>Survey of dry-cleaning establishments</td>
<td>18% (471) of dry cleaners in New York State are in residential buildings. PCE concentrations ranged from 15 to 197,000 µg/m³ in apartments above dry cleaners using transfer machines, 160-55,000 µg/m³ above vented dry-to-dry machines, and 6-1,910 µg/m³ above non-vented dry-to-dry machines.</td>
<td></td>
</tr>
<tr>
<td>ATSDR [1990a] See also ERM Southeast Inc. [1989], Tennessee</td>
<td>Mercury</td>
<td>Chlor-alkali Chemical plant Workers' homes contaminated</td>
<td>Decontamination method not described</td>
<td>Initial mercury concentration in 6 homes was 0.92(&lt;2.0-5.0) µg/m³; 1.0-5.0 µg/m³ in living quarters of 45 homes. 54 µg/m³ over washing machines, 7 µg/m³ over sinks, and 8-10 µg/m³ in workers' cars. After decontamination concentrations in homes were 0 to 0.5 µg/m³.</td>
<td>Vacuuming and floor washing were risk factors for increased mercury absorption by workers' wives.</td>
</tr>
<tr>
<td>Benning [1958], Ohio</td>
<td>Mercury</td>
<td>Carbon-brush manufacturing</td>
<td>Plant had poor housekeeping, no ventilation, no shower facilities or change of work clothing policy so that &quot;workers could not avoid taking a certain amount of contaminant home.&quot;</td>
<td></td>
<td>Provide cotton smocks, clean at start of work shift, to be left at plant each day and laundered.</td>
</tr>
<tr>
<td>Danziger and Possick [1973], New Jersey</td>
<td>Mercury</td>
<td>Scientific glassware manufacturing; calibration of glassware.</td>
<td>Observation</td>
<td>Mercury particles became embedded in workers' clothing, especially in knitted fabric. Some female workers would shake mercury out of their clothes when they arrived home.</td>
<td>The authors recommend that workers not wear knitted clothing and that they wear impervious aprons.</td>
</tr>
<tr>
<td>Ehrenberg et al. [1986], Vermont</td>
<td>Mercury</td>
<td>Thermometer and glass production for scientific use.</td>
<td>Flame absorption spectroscopy</td>
<td>The NIOSH trailer which workers entered for medical exams became contaminated; air samples (N=2) contained mercury at 23.4 and 21.5 µg/m³.</td>
<td>The measurements obtained in the NIOSH trailer suggest the possibility of offsite mercury contamination via workers inadvertently carrying mercury home on their clothes, shoes, hair, or skin.</td>
</tr>
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<tr>
<td>ERM Southeast Inc. [1989] See also ATSDR [1990a] Tennessee</td>
<td>Mercury</td>
<td>Protocol for mercury decontamination of workers' contaminated homes.</td>
<td>Jerome 511 mercury vapor analyzer &amp; dosemeter. Jerome 431 instantaneous mercury vapor monitor. Use of Nilfisk&lt;sup&gt;®&lt;/sup&gt; mercury vacuum system and mercury binding solution.</td>
<td></td>
<td>0.5 μg/m&lt;sup&gt;3&lt;/sup&gt; were used as clean/non-clean threshold.</td>
</tr>
<tr>
<td>Hudson et al. [1985, 1987] Vermont</td>
<td>Mercury</td>
<td>Thermometer manufacture</td>
<td>Air sampling in workers' homes.</td>
<td>Mercury-in-air of living area: Workers homes 0.02-10 μg/m&lt;sup&gt;3&lt;/sup&gt; (median 0.24 μg/m&lt;sup&gt;3&lt;/sup&gt;); Control homes 0.01-1 μg/m&lt;sup&gt;3&lt;/sup&gt; (median 0.05 μg/m&lt;sup&gt;3&lt;/sup&gt;).</td>
<td>All workers brought work clothes and shoes home. Some elevations of mercury in places where work clothes and shoes were found and in some washing machines.</td>
</tr>
<tr>
<td>Trost [1985] Vermont</td>
<td>Mercury</td>
<td>Thermometer plant</td>
<td></td>
<td>State inspectors found mercury contamination in over half of 50 workers' homes. One home had over 4 times the levels allowed at work.</td>
<td>New York times story of mercury transport via workers' clothing/shoes to homes.</td>
</tr>
<tr>
<td>West and Lim [1968] California</td>
<td>Mercury</td>
<td>Milling cinnabar ore</td>
<td></td>
<td>Authors stated that miners had contaminated their homes from their boots and work clothes.</td>
<td></td>
</tr>
<tr>
<td>Zalesak [1994] California</td>
<td>Mercury</td>
<td>Gold mining</td>
<td>Air sampling in workers' homes, cars, and inside plastic bags containing work clothing.</td>
<td>Mercury at 0.005-0.5 mg/m&lt;sup&gt;3&lt;/sup&gt; near washer and dryer, 0.03-0.05 mg/m&lt;sup&gt;3&lt;/sup&gt; in cars, documented contamination of work clothing.</td>
<td>Employees wearing work clothing home contaminated their cars and homes.</td>
</tr>
<tr>
<td>Aw et al. [1985] Indiana</td>
<td>Zeranol (an animal growth hormone)</td>
<td>Pharmaceutical formulator</td>
<td></td>
<td>Employee's work clothing contaminated with 32 mg of zeranol, employee's skin also contaminated. Workers often wore work clothes and shoes home, and laundered clothes at home.</td>
<td>Recommended showering and changing clothes before leaving work.</td>
</tr>
<tr>
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<tr>
<td>Katzenellenbogen [1956] Israel</td>
<td>Diethylstilbestrol Signs of estrogen poisoning in workers and families</td>
<td>Production of diethylstilbestrol in pharmaceutical factory. Wearing dirty clothes home</td>
<td>Established control procedures including sufficient ventilation, gloves, respirators, special shoes and clothing, showers, and laundry procedures.</td>
<td>Health effects reduced by control procedures.</td>
<td>Hyperestrogenism in workers and children disappeared after installation of controls.</td>
</tr>
<tr>
<td>Pasynski et al. [1971] Poland</td>
<td>Diethylstilbestrol</td>
<td>Manufacturing</td>
<td>Observation of work practices including taking potentially contaminated food home. Institution of improved industrial hygiene.</td>
<td>In both cases, one spouse was allergic to the allergen brought home on the clothing of the other spouse.</td>
<td>Symptoms resolved after workers changed clothes and showered before leaving work.</td>
</tr>
<tr>
<td>Venables and Newman-Taylor [1989] United Kingdom</td>
<td>Rat allergen Platinum salts</td>
<td>Animal handling in laboratory Use of platinum salts in industrial laboratory</td>
<td>Concentration of arsenic in household dust from vacuums after filtering through a 0.246 mm size sieve. Analyzed via spectrophotometry.</td>
<td>Arsenic content (μg/g dust) in untreated homes without exposed worker 1.1 to 31.0; with exposed worker 5.2 to 1,080. Arsenic content of treated homes without exposed worker 3.0 to 6.4; with exposed worker 8 to 380.</td>
<td>Arsenic may have been brought home on clothing of some exposed workers.</td>
</tr>
<tr>
<td>Klemmer et al. [1975] Hawaii</td>
<td>Arsenic</td>
<td>Homes treated with pesticides against termite infestations, and homes with pretreated lumber, homes with workers exposed to arsenic.</td>
<td>Surface and finger sampling for fibers. A method was developed to measure settled fibers from hard surfaces.</td>
<td>Fiber counts on fingers before exposure to dusty surfaces were 0-1 f/cm², after exposure, 1.5-82 f/cm².</td>
<td></td>
</tr>
<tr>
<td>Schneider [1986] Schneider et al. [1989]</td>
<td>Man made mineral fibers (M MMMF) and non-MMMP</td>
<td>Surveys in office buildings and schools</td>
<td>Study of human activities affecting airborne fiber concentrations in non-occupational environments in a classroom.</td>
<td>Demonstrated a large increase in fiber concentration in air during vacuuming.</td>
<td></td>
</tr>
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<tr>
<td>Mastek et al. [1972] Czechoslovakia</td>
<td>3,4-Benz(o)pyrene</td>
<td>Pitch coking plant.</td>
<td>3,4-Benz(o)pyrene in workers clothing.</td>
<td>3,4-Benz(o)pyrene in underwear (pants) 31-930 µg/g; (shirts) 410-1,100; in work clothes (pants) 7,760-35,000 µg/g; in shirts 1,400-14,000 µg/g. In washed clothes 12-594 µg/g.</td>
<td>Author states that current washing method does not ensure effective removal of contaminant. Residual contaminant after washing increases with time of use, 41 µg/g at 2 wks, 315 µg/g at 12 months.</td>
</tr>
<tr>
<td>Versen and Bunn [1969] USA</td>
<td>Diatomaceous earth</td>
<td>Mining, processing, packaging diatomaceous earth. Laundering of work clothing in employees homes.</td>
<td>Air sampling in laundry area of home and outside home. Analysis via X-ray diffraction.</td>
<td>Total dust 0.02-0.06 mg/m³ inside home, 0.02-0.08 mg/m³ outside home. Silica was detected but amount was below quantitation level.</td>
<td></td>
</tr>
<tr>
<td>Weeks et al. [1976]</td>
<td>Aromatic amines</td>
<td>Not applicable</td>
<td>Method development—chemical spot tests (visualization &amp; UV detection).</td>
<td>The spot tests can detect low levels of aromatic amines on surfaces: on paint 5-150 ng/cm²; on metal 15 ng/cm²; smooth concrete 500-5,000 ng/cm²; rough concrete 200-5,000 ng/cm².</td>
<td></td>
</tr>
<tr>
<td>Woody et al. [1986] Arkansas</td>
<td>Cyclotrimethylene trinitramine (RDX)</td>
<td>Explosives manufacture</td>
<td>Method development—chemical spot tests (visualization &amp; UV detection).</td>
<td>Mother reported seeing child chewing on clumps of the plasticized RDX carried home on work boots and clothing worn home.</td>
<td></td>
</tr>
<tr>
<td>Pasanen et al. [1989] Finland</td>
<td>Fungi</td>
<td>Farming</td>
<td>Air sampling with impactor; cultivation; SEM.</td>
<td>In farm homes: 10³ to 10⁶ colony forming units/m³; total spores at 10⁴ to 10⁵ spores/m³ were 10-1,000 times levels in urban homes. Certain fungal genera were measured in cow barns and farmers homes but not in control homes.</td>
<td>Study suggests that fungal spores were carried on clothes from cow barns to farmers' homes.</td>
</tr>
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<td>Garrettson [1984]</td>
<td>Environmental toxicity</td>
<td>Pesticides, lead and other toxins</td>
<td>Literature review</td>
<td></td>
<td>Workers need to be informed about toxicity of compounds and the potential dangers of taking them home in one way or another. Lead regulations intended to protect workers' families need to be enforced.</td>
</tr>
<tr>
<td>Cannell et al. [1987a, 1987b]</td>
<td>Measurement of home contamination</td>
<td>Research: Shoe types, floor types, and traffic</td>
<td>Tracers: Polydisperse stilbene for carpets; monodisperse silica particles for tile.</td>
<td>Ground contact with shoes is dominant factor in transport capacity; carpet removes soil from shoes more rapidly than tile; at most 50% of contaminant in carpet can be removed by intense vacuuming.</td>
<td></td>
</tr>
<tr>
<td>Beegle and Forlund [1990]</td>
<td>Asbestos and lead</td>
<td>Not applicable</td>
<td>Protocols for cleaning homes contaminated with asbestos and lead are provided.</td>
<td>Not applicable.</td>
<td></td>
</tr>
<tr>
<td>Goldman and Peters [1981]</td>
<td>Occupational and environmental health history.</td>
<td>Occupational and environmental health history.</td>
<td>Recommendations that clinical physicians include occupation of family members to identify potential sources of exposure, including chemicals brought into the home from contaminated work clothes.</td>
<td>Article provides sequence of steps to facilitate physician recognition of occupationally and environmentally related diseases.</td>
<td></td>
</tr>
<tr>
<td>Fish et al. [1967]</td>
<td>Particulate</td>
<td>Experimental re-dispersion of settled particulates.</td>
<td>Light-scattering particle size analyzer.</td>
<td>Re-dispersion of settled particulates was dependent upon room activity. With light air movement, transfer from floor to clothing was 22%/hr.</td>
<td>Demonstrated potential for body and clothing contamination from resuspension of settled dust on workroom surfaces.</td>
</tr>
</tbody>
</table>

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<thead>
<tr>
<th>Reference</th>
<th>Pesticide (water solubility)</th>
<th>Formulation</th>
<th>Fabrics</th>
<th>Laundry or Decontamination Variables</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Braun et al. [1989]</td>
<td>Pyrazophos (4 mg/L at 20°C) Spray on three layers of cloth; spill on three layers of cloth</td>
<td>Water solution (450 mg/L)</td>
<td>Cotton polyester coverall fabric, contaminated and uncontaminated clothes washed together</td>
<td>Automatic washing machine, heavy-duty detergent, single wash, 2 rinses; double wash; pre-soak plus double wash.</td>
<td>Single wash removed 78% of pyrazophos from spray-on and 83% from spill-on; double wash removed 92% and 94%, respectively. Pre-soak did not improve on double wash. Transfer fabrics had 68% of the residual contamination of the contaminated fabrics. Second and third layers had traces of pyrazophos when sprayed on and slightly more when spilled on.</td>
</tr>
<tr>
<td>Chiao-Cheng et al. [1988]</td>
<td>Carbofuran (700 mg/L) methomyl (60,000 mg/L)</td>
<td>Water suspension Water soluble liquid</td>
<td>100% cotton and 100% polyester</td>
<td>60°C and 49°C washes. Anionic and non-ionic washes.</td>
<td>More than 99% of the pesticides were removed from both fabrics by all laundering methods</td>
</tr>
<tr>
<td>Esley et al. [1981b]</td>
<td>Methyl parathion (50-60 mg/L at 20°C) encapsulated wettable powder</td>
<td>Emulsifiable concentrate</td>
<td>Denim, 100% cotton and 50/50 cotton-polyester</td>
<td>Pre-rinse, detergent, ammonia and chlorine bleach.</td>
<td>Fabric was not a factor in removal. For encapsulated and wettable powder forms, 93-99% of methyl parathion was removed; for emulsifiable concentrate, 80-88% was removed by the different laundry procedures. Pre-rinse was the most effective variable; ammonia the least effective.</td>
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<tr>
<td>Esley et al. [1982a]</td>
<td>Methyl parathion</td>
<td>Emulsifiable concentrate solutions 0.25-54%</td>
<td>Denim, 100% cotton twill</td>
<td>Heavy duty liquid detergent, 1 wash cycle, 2 rinse cycles. Repeated up to 10 times.</td>
<td>More than 95% methyl parathion was removed when applied as 0.25 and 0.50% solutions. Only 19.5% was removed by first wash when applied as a 54% solution, and only 67% had been removed after 10 washes.</td>
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<td>Reference</td>
<td>Pesticide (water solubility)</td>
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<td>Basley et al. [1982b]</td>
<td>Methyl parathion</td>
<td>Emulsifiable concentrate 1.25% solution</td>
<td>100% Cotton and 50/50 cotton-polyester</td>
<td>30°C, 49°C, and 60°C washes; detergents: 8.7% phosphate; carbonate; non-phosphate heavy duty liquid; 12% phosphate.</td>
<td>50% removal at 30°C; 70% or greater removal at 49°C and 60°C. Heavy duty liquid detergent slightly better than others at 49°C and 60°C.</td>
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<td>Basley et al. [1983]</td>
<td>2,4-dichlorophenoxyacetic acid (2,4-D) ester and 2,4-D amine (ester, insoluble) (amine 4-15 g/L at 30°C)</td>
<td>Denim, 80/20 cotton-polyester contaminated; 50/50 cotton-polyester transfer fabric</td>
<td>Denim, 80/20 cotton-polyester contaminated; 50/50 cotton-polyester transfer fabric</td>
<td>60°C wash/49°C rinse; 30°C wash/30°C rinse; pre-rinse/no pre-rinse; heavy duty liquid non-phosphate detergent; 12% phosphate detergent; ammonia/no ammonia; repeated washing.</td>
<td>2,4-D ester, 26-45% removed, 1-2% transferred; pre-rinse had no effect; ammonia had no effect; 30°C wash, 26% removed; 60°C wash 49% removed; non-phosphate detergent removed 31%; one wash removed about 30%; two washes removed about 41%; 2,4-D amine, more than 99% removed by all treatments; transfer 0.02-0.26%.</td>
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<td>Basler [1983] Basler and DeJonge [1985]</td>
<td>Captain (3 mg/L at 77°C) Guthion® (29 mg/L at 25°C)</td>
<td>Aqueous suspension Emulsifiable concentrate</td>
<td>Denim 100% Cotton twill Nylon Microporous film fabric, 3 layers</td>
<td>38°C, 49°C, 60°C washes, 2 rinses. Heavy duty liquid detergent with both anionic and non-ionic surfactants.</td>
<td>Captain-cotton, 75%, 98%, and 99% removal at 38°C, 49°C, and 60°C; nylon, 98% removal at 38°C, greater than 99% removal at 49°C and 60°C. Guthion-cotton, 94%, 96%, and 98% removal at 38°C, 49°C, and 60°C; nylon 86%, 91%, and 92% removal at 38°C, 49°C, and 60°C.</td>
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<tr>
<td>Finley and Rogilio [1969]</td>
<td>DDT (insoluble) methyl parathion</td>
<td>Fabrics worn in cotton fields for 8 hours on the day after spraying</td>
<td>100/0, 35/65, 50/50, 65/35 cotton-polyester type fabric with durable press finish or soil release or soil release finish (65/35 only)</td>
<td>Laundered in automatic washer and electric dryer.</td>
<td>DDT averaged 81.8 ppm in clothing before laundering, 24.9 ppm after; 100/0 and 65/35 cotton-polyester collected and retained more DDT than other fabrics. Methyl parathion averaged 7.7 ppm in clothing before laundering and 0.2 ppm after laundering. 100/0 and 65/35 cotton-polyester collected more parathion than other fabrics and also retained more, but not as much as with DDT.</td>
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| Finley et al. [1974] | Methyl parathion  
Toxaphene-methyl parathion-DDT  
(toxaphene 3 mg/L)                         | Emulsifiable concentrate           | 100% Cotton. 50/50 Cotton-polyester       | 60°C, 2 rinses, standard detergent, small (19.85 L) electric portable washer. Contaminated and clean fabrics washed 1, 2, or 3 times. | Toxaphene and DDT were difficult to remove from fabric after first wash. Transfer of all three pesticides when applied as a mixture was much greater for the 50/50 cotton-polyester than for 100% cotton. When methyl parathion was applied by itself, there was no difference in transfer by fabric type. All *Drosophila* confined on washed fabrics that had been contaminated with the mixture died. |
| Goodman et al. [1988] | Methyl parathion  
5 days contamination with and without daily laundering                               | Emulsifiable concentrate 1.25%    | 100% Cotton poplin. 50/50 cotton-polyester poplin finished and unfinished with a fluoro-carbon renewable soil-repellent finish | 49°C water, 2 rinses, pre-wash, non-ionic heavy-duty liquid detergent, agitation by 25 stainless steel balls. | Residue after washing daily was less for soil-repellent fabrics. Residue increased daily when washing was not done daily. Both contamination levels and residuals were less with soil-repellent finish when laundering was not done daily. Methyl parathion concentrations in water were much less with daily laundering. |
| Graves et al. [1980] | Permethrin  
(0.2 mg/L at 20°C)                                                                     | Fabrics worn in cotton fields on days 1, 2, or 4 after spraying | Heavy all cotton fabric, 2 layers         | Single laundering in hot water, heavy-duty detergent, dry bleach, perborate additive.                  | Contamination: Outer layer day 1, 25.8 ppm; day 2, 14.4 ppm; day 4, 3.8 ppm; under layer day 1, 19.9 ppm; day 2, 14.4 ppm; day 4, 1.2 ppm.  
After laundering: Outer layer day 1, 15.3 ppm; day 2, 9.2 ppm; day 4, 1.6 ppm; under layer day 1, 9.4 ppm; day 2, 5.8 ppm; day 4, 0.6 ppm. |
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<td>Hild et al. [1989]</td>
<td>Methyl parathion</td>
<td>Emulsifiable concentrate</td>
<td>50/50 Cotton-polyester with and without soil repellent</td>
<td>40°C wash, non-ionic heavy-duty liquid detergent; phosphate-based powdered anionic detergent: Prewash product, air dried, 50, 100, 150, and 225 mL water, 0, 10, 25, 50 steel balls.</td>
<td>No effect of detergent type. Increasing detergent decreased residue, increasing water level decreased residue, agitation (steel balls) had no effect. Residues always greater with soil repellent. All treatments removed more than 98% of contaminant.</td>
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<td>Keeschall et al. [1986]</td>
<td>Organophosphates: chlopyrifos (2 mg/L), dichlorvos (5 g/L), diazinon (40 mg/L), dimethoate (25 g/L), malathion (200 g/L), methyl parathion</td>
<td>All emulsifiable concentrates except carbofuran which was a flowable formulation</td>
<td>50/50 Cotton-polyester, unfinished and fluorocarbon finishes consumer applied and commercially applied</td>
<td>49°C wash, 2 rinses, detergent, pre-wash spray or degreaser.</td>
<td>Dichlorvos, dimethoate, malathion, carbofuran and propoxur were effectively removed from all fabrics by washing; chlopyrifos, aldrin, and lindane were least effectively removed. Fluorocarbon finishes reduced absorption of pesticides by the fabrics, but did not facilitate removal by laundering. The laundry additives significantly aided residue removal.</td>
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<tr>
<td>Kim and Wang [1992]</td>
<td>Atrazine 30 mg/L at 20°C</td>
<td>Water-dispersible granules, unground and ground (dust) 1 gram sewed into pockets</td>
<td>Heavy weight cotton twill</td>
<td>60°C and 49°C, heavy duty liquid detergent with anionic and non-ionic surfactants, 10 and 30 steel balls (agitation), machine and air drying, accelerated laundering apparatus, 300 mL water, clean clothes laundered with contaminated ones.</td>
<td>Over 99% of the initial contamination was removed by the washing and drying process. The amounts in the transfer cloths were 0.05-0.25% of the initial contamination. The 60°C water removed more atrazine and resulted in lower transfer to clean cloths. Drying methods had no effect.</td>
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<tr>
<td>Kim et al. [1993]</td>
<td>Atrazine (30 mg/L) Metolachlor (530 mg/L)</td>
<td>Water dispersible granules Wettable powder Flowable liquid Emulsifiable concentrate 1 gram sewed into sockets</td>
<td>100% Cotton heavy weight twill</td>
<td>60°C wash, heavy duty liquid detergent, 2 rinses, machine dried. More than 99% of all atrazine formulations were removed, but only 90% of metolachlor. Emulsifier appeared to inhibit removal of the more soluble metolachlor. Atrazine in transfer cloths was 13-50% of residues in contaminated cloths, but only 0.2% for metolachlor.</td>
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<tr>
<td>Kim et al. [1982]</td>
<td>Fonofos (13 mg/L at 20°C) Alachlor (242 mg/L at 25°C)</td>
<td>Emulsifiable concentrate</td>
<td>100% Cotton 6 oz. shirt wt. 14 oz. pants wt. denim</td>
<td>40°C, 49°C, 60°C, detergent/no detergent, immediate wash/24-hr wash, 2 cold rinses, air dried, 150 mL water, 30 steel balls. Remaining residues after laundry (ratio of residue peak to standard peak) 42-81% for fonofos, 2-47% for alachlor. Heavier fabrics contained more residues, hot water removed more of both substances from heavier fabrics, detergents resulted in lower residues, as did immediate washing.</td>
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<tr>
<td>Kim et al [1986]</td>
<td>Alachlor</td>
<td>Emulsifiable concentrate</td>
<td>100% Cotton and 65/35 polyester-cotton broadcloth. 100% and 65/35 polyester-cotton twill.</td>
<td>49°C, 60°C, 150 mL water, standard detergent with 12.4% phosphate, commercial detergent with 6.1% phosphate, heavy duty detergent with no phosphate, three pre-treatments: denatured ethyl alcohol (7.5 mL/150 mL); perchloro-ethylene (7.5 mL/150 mL); distilled water 150 mL each for 2 minutes at 49°C; 9-minute wash, 2 rinses at 49°C. Air and machine dry.</td>
<td>Hot water removed more alachlor than warm water. Dryer drying reduced residues compared to air. Light fabrics had much lower residues than heavy fabrics; 65/35 polyester-cotton twill had more residue than 100% cotton twill. Perchloroethylene removed significantly more alachlor than alcohol or water. Phosphate detergents decreased residues in broadcloths.</td>
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<td>Kim [1989]</td>
<td>Alachlor</td>
<td>Emulsifiable concentrate</td>
<td>100% Cotton twill</td>
<td>Convection oven 60°C, 100°C, 150°C, 200°C, 15, 30, 60 min drying time. Microwave 2450 MHz, 50, 250, 500 1st, 50, 100, 150, 200 seconds.</td>
<td>Alachlor degraded rapidly at 200°C in the convection oven and after 30 minutes at 150°C. At 60 minutes and 150°C, the residue was 0.004% of the contamination level. The microwave was not very effective.</td>
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<tr>
<td>Laughlin and Gold [1989a]</td>
<td>Methyl parathion</td>
<td>Emulsifiable concentrate 1.25% and 54%</td>
<td>100% Cotton, 50/50 cotton-polyester. Unfinished or with fluorosilphatic soil repellent.</td>
<td>60°C, 2 rinses at 49°C, non-ionic liquid detergent, 12 minute wash, storage at 0°C, 20°C, and 20°C with airflow, and 20°C with relative humidity at 65. 54% formulation stored only at 20°C with airflow.</td>
<td>Initial contamination with 1.25% formulation was 50 μg/cm² for unfinished fabric and 22 μg/cm² for finished. The finished material had greater residues (3.63 μg/cm²) than unfinished (0.35-1.33 μg/cm²). Residues decreased with storage time in moving air at 20°C.</td>
</tr>
<tr>
<td>Laughlin and Gold [1989b]</td>
<td>Methyl parathion</td>
<td>Emulsifiable concentrate Wettable powder encapsulated</td>
<td>50/50 cotton polyester Unfinished durable press Fluorocarbon soil repellent</td>
<td>69°C wash or 49°C wash, 49°C rinse, heavy duty liquid non-ionic detergent or standard detergent, accelerated laundering apparatus. Also 1 m² fabric laundered in automatic home washing machines at 49°C.</td>
<td>Laundering significantly reduced contamination of all fabrics, but the fluorocarbon soil repellent fabric was more resistant to cleaning. Residues were less with the encapsulated formulation. Study demonstrated spread of contaminant from point of contact during laundering as well as contamination of laundry equipment.</td>
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<tr>
<td>Laughlin et al. [1985]</td>
<td>Methyl parathion</td>
<td>Emulsifiable concentrate 0.25-54% Wettable powder</td>
<td>100% cotton and 50/50 cotton-polyester denim fabrics. All cotton batiste was used for transfer studies.</td>
<td>60°C wash, 49°C pre-rinse/no pre-rinse, phosphate detergent, two rinses at 49°C, ammonia/no ammonia, bleach/no bleach. Clean fabric laundered immediately after contaminated fabric. Four detergents each at 30°C, 49°C, and 60°C. Multiple launderings (up to 10).</td>
<td>80-99% of parathion removed, no differences between fabrics. Emulsifiable concentrate, 80-89% removal, other forms more than 90%. Pre-rinse resulted in greater removal. Laundry equipment was sufficiently contaminated that the transfer fabric (laundered immediately after contaminated fabric) contained 0.0001-0.0001% of the original contamination. The four detergents tested gave similar results. Hot water (49°C and 60°C) was better than cold. With 1.25% methyl parathion, 18% remained after first wash and 4% after second wash and 0.37% after the tenth wash. With 54% methyl parathion, 84% remained after the first wash and 33% remained after the tenth wash. Ammonia and bleach had no effect.</td>
</tr>
<tr>
<td>Lillie et al. [1982]</td>
<td>Diazinon Chlorpyrifos</td>
<td>Emulsifiable concentrate</td>
<td>100% Cotton</td>
<td>One-speed washing machine, 30°C, 40°C, 60°C, detergent, detergent plus bleach, plain water, 2 rinses, 30 minute gas dryer.</td>
<td>78-96% of diazinon removed; 52-77% of chlorpyrifos removed; 30-84% of chlor dane removed. Removal increased with hotter water and detergent in all cases.</td>
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</table>
Table 16. (Continued) Studies on Laundering Pesticides from Contaminated Clothing

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<tr>
<th>Reference</th>
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<tr>
<td>Lillie et al [1981]</td>
<td>Diazinon (40 mg/L)</td>
<td>Emulsifiable concentrate</td>
<td>100% Cotton</td>
<td>One-speed washing machine, 68 agitations/min. 43°C wash cycle, non-phosphorus detergent, 14 minute wash, 2 rinses, gas dryer. Effect of 30°C, 43°C, and 60°C water temperatures on removal from 100% cotton fabric.</td>
<td>Absorption by cotton was greater than for polyester with chlordane, diazinon, and prometon. Penetration was greater for the polyester. The particles of the wettable powder of carbaryl were probably large enough to prevent penetration. 90-99% of all pesticides were removed by washing. Hotter water removed more pesticide except for chlordane where there was no effect of water temperature.</td>
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<td>Chlordane (1 mg/L)</td>
<td>Emulsifiable concentrate</td>
<td>100% Polyester</td>
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<td>Carbaryl (120 mg/L)</td>
<td>Wettable powder</td>
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<td>Prometon (750 mg/L)</td>
<td>Emulsifiable concentrate</td>
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<td>Bromacil (800 mg/L)</td>
<td>Oil formulation</td>
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<td>Propoxur (2 g/L)</td>
<td>Emulsifiable concentrate</td>
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<td>Malathion (200 mg/L)</td>
<td>Oil formulation</td>
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<td>Nelson et al. [1992]</td>
<td>Carbamates: carbaryl (insecticide) (120 mg/L at 30°C) triallate (herbicide) (4 mg/L at 20°C) Organophosphates: methyl parathion (insecticide) (50 mg/L at 20°C) fonofos (insecticide) (13 mg/L at 20°C) terbufos (insecticide) (10-15 mg/L at 25°C) Pyrethroids: deltamethrin (insecticide) (0.002 mg/L at 20°C) cyfluthrin (insecticide) (2 mg/L at 20°C) cypermethrin (insecticide) (0.01-0.02 mg/L at 20°C) Alachlor (herbicide) (242 mg/L at 25°C) Atrazine (herbicide) (30 mg/L at 20°C) Trifluralin (herbicide)</td>
<td>Wettlable powder and flowable liquid Emulsifiable concentrate Liquid Emulsifiable concentrate Liquid Emulsifiable concentrate Wettlable powder and emulsifiable concentrate Wettlable powder and emulsifiable concentrate Emulsifiable concentrate Wettlable powder and flowable liquid Emulsifiable</td>
<td>100% Cotton and 50/50 cotton-polyester twill.</td>
<td>49°C, 2 rinses, pre-wash/no pre-wash, heavy duty liquid detergent, air drying</td>
<td>Prewash product lowered residue for all pesticides. The residue remaining ranged from zero for carbaryl wettlable powder to 48% for deltamethrin emulsifiable concentrate without prewash. Residues were greater on the cotton-polyester for organophosphates and lower for atrazine.</td>
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Table 16. (Continued) Studies on Laundering Pesticides from Contaminated Clothing

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<tbody>
<tr>
<td>Oakland et al.</td>
<td>Atrazine (30 mg/L) Diazinon (40 mg/L) Metolachlor (330 mg/L)</td>
<td>Not stated</td>
<td>100% Cotton knit 65/35 Cotton-polyester</td>
<td>27°C and 60°C wash, detergent (not described), contaminated and non-contaminated fabrics washed together.</td>
<td>The only atrazine cross-contamination was from denim to denim in cold water. Diazinon cross-contamination was greater than metolachlor except transfer to denim. Cross-contamination was significantly greater in hot water for diazinon and metolachlor. Atrazine did not cross-contaminate in hot water. Both denim and knit fabrics accepted cross-contamination.</td>
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<td>[1989]</td>
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<td>chambray, 100% Cotton denim, 65/35</td>
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<td>polyester-cotton with fluorocarbon finish</td>
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<tr>
<td>Obendorf and Solbrig [1984]</td>
<td>Malathion Methyl parathion (tagged with osmium tetroxide)</td>
<td>Not stated</td>
<td>50/50 Cotton-polyester with and without</td>
<td>200 mL water (temperature not reported, 10 steel balls (agitation), anionic surfactant with carbonate and zeolite builders, 2 rinses, air dried. X-ray analysis for fabric distribution of pesticides.</td>
<td>Washing removed 30-40% of both pesticides, but did not reduce the amounts in the lumen of the cotton fibers.</td>
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<td>durable-press finish</td>
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<td>Rigakis et al.</td>
<td>Trifluralin (1 mg/L) Triallate (4 mg/L) Deltamethrin (0.002 mg/L)</td>
<td>Emulsifiable</td>
<td>100% Cotton fabric</td>
<td>50°C wash, 2 rinses at 40°C, pre-wash/ no pre-wash each with one or two washes, air dried.</td>
<td>Trifluralin removal was 77% after one wash and 91% after two washes with pre-wash. Triallate removal was 52% after one wash and 82% after two washes with pre-wash. Deltamethrin removal was 84% after one wash and 99% after two washes with pre-wash.</td>
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<td>[1987]</td>
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<td>concentrate</td>
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<td>Satoh [1979]</td>
<td>Note this article and the next article in the journal had the authors and titles switched. See Finley et al. [1979] for correct title and author.</td>
<td>Contamination by wearing clothes in cotton field for one day</td>
<td>50/50 Cotton-polyester twill pants and shirts</td>
<td>140°F wash, rinse, heavy duty carbonate laundry detergent, machine dry at 190°F for 30 minutes. Repeat procedure.</td>
<td>Contamination levels ranged from 0.07 to 28.97 ppm. One washing reduced residues by 75-95%. Washing seemed to be less effective as contamination level increased. The second wash removed a smaller percentage of the remaining residue.</td>
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<td>Popular Name</td>
<td>Public Law</td>
<td>U.S. Code</td>
<td>Sections Relevant to Workers' Family Protection</td>
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The Congress declares it to be its purpose and policy . . . . to assure so far as possible every working man and women in the Nation safe and healthful working conditions . . . .  

Note 9. This chapter was created for the sole purpose of protecting health and safety of workers and improving physical working conditions on employment premises C.A. 5, 1979F. 2d622.  

Note 16. This chapter covers only housing that is a condition of employment and does not apply to housing which is work related but which is not conditions of employment C.A. 11, 1983, 696F. 2d1325, rehearing denied 704F. 2d1253.  

§ 654 Duties of Employers and Employees  
Each employer shall furnish to each of his employees employment and a place of employment which are free from recognized hazards that are causing or are likely to cause death or serious physical harm to his employees.  

Note 84. This chapter does not create duties between employers and invitees, only between employers and their employees. C.A. Tex. 1981, 653F. 2d915, rehearing denied 661F. 2d931.  

Note 86. Secretary should be able to extend coverage of this chapter to certain employer-provided means of transportation and certain employer-provided housing even though such extension exceeds plain language of this chapter. C.A. 11, 1983, 696F. 2d1325, rehearing denied 704F. 2d1253.  

§ 669 Research and Related Activities  
§ 669(a)(4) The Secretary of Health and Human Services shall also conduct special research, experiments, an demonstrations relating to occupational safety and health as are necessary to explore new problems, including those created by new technology in occupational safety and health, which may require ameliorative action beyond that which is otherwise provided for in the operating provisions of this chapter.  

§ 669(a)(6) The Secretary of Health and Human Services . . . . shall determine following a written request by any employer or authorized representative of employees, specifying with reasonable particularity the grounds on which the request is made, whether any substance found in the place of employment has potentially toxic effects in such concentrations as used or found . . . .  

§ 669(e) The functions of the Secretary of Health and Human Services under this chapter shall, to the extent feasible, be delegated to the Director of the National Institute for Occupational Safety and Health established by Section 671 of this title.  

§ 671 The Institute is authorized to - (1) develop and establish recommended occupational safety and health standards.
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(g) "Miner" means any individual working in a coal or other mine;  
(b)(1) "coal or other mine" means (A) an area of land from which minerals are extracted in non-liquid form or, if in liquid form, are extracted with workers underground, (B) private ways and roads appurtenant to such area, and (C) lands, excavations, underground passageways, shafts, slopes, tunnels and workings, structures, facilities, equipment, machines, tools, or other property including impoundments, retention dams, and tailings ponds, on the surface or underground, used in, or to be used in, or resulting from, the work of extracting such minerals from their natural deposits in non-liquid form, or if in liquid form, with workers underground, or used in, or to be used in, the milling of such minerals, or the work of preparing coal or other minerals, and includes custom coal preparation facilities. In making a determination of what constitutes mineral milling for purposes of this chapter, the Secretary shall give due consideration to the convenience of administration resulting from the delegation to one Assistant Secretary of all authority with respect to the health and safety of miners employed at one physical establishment.  
Note 3. Coal or other mine  
Definition of "coal mine" under subsection (h) of this section includes a commercial purpose requirement. C.A. 3, 1984, 748F. 2d176.  
Note 5. Miner  
Owner operators who work the mines are "miners" within this chapter and fall within the category of persons whose safety Congress desired to protect. D.C. PA. 1980, 491F Supp. 1123. This chapter's broad definition of "miner" as any individual working in a coal mine rebuts any inference that a miner cannot also be an owner or operator. D.C. PA. 1978, 465F, Supp. 838.  
§ 803 Mines subject to coverage  
Each coal or other mine, the products of which enters commerce, or the operations or products of which affect commerce, and each operator of such mine and every miner in such mine shall be subject to provisions of this chapter.  
Note 5. One man, owner operated coal mine the products of which were sold totally intra state, was not subject to requirements of this chapter. D.C. PA. 1973, 373F, Supp. 797.  
Note 7. This chapter applied to small coal mine even though only miners working therein were four brothers who owned and operated the mine. C.A. PA. 1979, 604F, 2d231.  
Provisions of this chapter are applicable even though owner operators work the mine. D.C. PA. 1980, 491F, Supp. 1123.  
This chapter covers mines that are totally owned and operated by the same persons, that is, those mines where the only persons working therein are the owners themselves. D.C. |
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<td>Public Law 95-164</td>
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<td>Note 14. Refusal of owner-operators to permit an authorized representative of the Secretary of Labor to enter upon and to conduct an inspection of their mine constituted a continuing threat to the health and safety of miners and interfered with, hindered and delayed the Secretary and his authorized representatives in carrying out the provisions of this chapter. D.C. PA 1980, 491F, Supp. 1123.</td>
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<td>Note 15. Where operator of small, family-owned rock quarry and his wife excavated rock and marketed their product without the assistance of any employees and in view of fact that the excavation of decorative rock was not subject to the type of license and reporting requirements which place some business proprietors on notice of extensive federal oversight, circumstances did not permit conclusion that the operator of the quarry implicitly consented to warrantless inspections of his quarry by representatives of the Secretary of Labor pursuant to this chapter. C.A. Cal. 1980, 628P, 2d1255.</td>
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<td>§ 877(f) The Secretary may require any operator to provide adequate facilities for the miners to change from the clothes worn underground, to provide for the storing of such clothes from shift to shift, and to provide sanitary and bathing facilities.</td>
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<td>§ 951. Studies and research</td>
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<td>(a) [The Secretary of Health and Human Services shall conduct studies]</td>
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<td>(11) to determine upon written request by any operator or authorized representative of miners, specifying with reasonable particularity the grounds upon which such request is made, whether any substance normally found in a coal or other mine . . . . has potentially hazardous effects, and shall submit such determinations to both the operators and the miners as soon as possible;</td>
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<td>(12) for such other purposes as . . . . deem[ed] necessary to carry out the purposes of this chapter.</td>
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<td>(b) Activities under this section in the field of coal or other mine health shall be carried out by the Secretary of Health and Human Services through the National Institute of Occupational Safety and Health . . . .</td>
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<td>(d) The Secretary of Health and Human Services shall also conduct studies and research into matters involving the protection of life and the prevention of diseases in connection with persons, who although not miners, work with, or around the products of coal or other mines in areas outside of such mines and under conditions which may adversely affect the health and well-being of such persons.</td>
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(2)(A) Except as provided in subparagraph (B), the term "chemical substance" means any organic or inorganic substance of a particular molecular identity, including—  
(i) any combination of such substances occurring in whole or in part as a result of a chemical reaction or occurring in nature and  
(ii) any element or uncombined radical.  
(B) Such term does not include—  
(i) any mixture,  
(ii) any pesticide (as defined in the Federal Insecticide, Fungicide and Rodenticide Act) when manufactured, processed, or distributed in commerce for use as a pesticide,  
(iii) tobacco or any tobacco product,  
(iv) any source material, special nuclear material, or byproduct material (as such terms are defined in the Atomic Energy Act of 1954 and regulations issued under such Act),  
(v) any article the sale of which is subject to the tax imposed by section 4181 of Title 26 (determined without regard to any exemptions from such tax provided by section 4182 or 4221 or any other provision of Title 26), and  
(vi) any food, food additive, drug, cosmetic, or device (as such terms are defined in section 321 of Title 21) when manufactured, processed or distributed in commerce for use as a food, food additive, drug, cosmetic, or device.  
(5) The term "environment" includes water, air, and land and the interrelationship which exists among and between water, air, and land and all living things.  
(7) The term "manufacture" means to import into the customs territory of the United States (as defined in general headnote 2 of the Tariff Schedules of the United States), produce, or manufacture.  
(8) The term "mixture" means any combination of two or more chemical substances if the combination does not occur in nature and is not, in whole or in part, the result of a chemical reaction; except that such term does include any combination which occurs, in whole or in part, as a result of a chemical reaction if none of the chemical substances comprising the combination is a new chemical substance and if the combination could have been manufactured for commercial purposes without a chemical reaction at the time the chemical substances comprising the combination were combined.  
(10) The term "process" means the preparation of a chemical substance or mixture, after its manufacture, for distribution in commerce—  
(A) in the same form or physical state as, or in a different form or physical state from, that in which it was received by the person so preparing such substance or mixture, or  
(B) as part of an article containing the chemical substance or mixture.  
(11) The term "processor" means any person who processes a chemical substance or mixture. |
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(a) Scope of regulation.—If the Administrator finds that there is a reasonable basis to conclude that the manufacture, processing, distribution in commerce, use, or disposal of a chemical substance or mixture, or that any combination of such activities, presents or will present an unreasonable risk of injury to health or the environment, the Administrator shall by rule apply one or more of the following requirements to such substance or mixture to the extent necessary to protect adequately against such risk using the least burdensome requirements:

(5)(A) A requirement prohibiting or otherwise regulating any manner or method of disposal of such substance or mixture, or of any article containing such substance or mixture, by its manufacturer or processor or by any other person who uses, or disposes of, it for commercial purposes.

(7) A requirement directing manufacturers or processors of such substance or mixture
(A) to give notice of such unreasonable risk of injury to distributors in commerce of such substance or mixture and, to the extent reasonably ascertainable, to other persons in possession of such substance or mixture or exposed to such substance or mixture,
(B) to give public notice of such risk of injury, and
(C) to replace or repurchase such substance or mixture as elected by the person to which the requirement is directed.

§ 2607 Reporting and Retention of Information
(c) Records—Any person who manufactures, processes, or distributes in commerce any chemical substance or mixture shall maintain records of significant adverse reactions to health or the environment, as determined by the Administrator by rule, alleged to have been caused by the substance or mixture. Records of such adverse reactions to the health of employees shall be retained for a period of 30 years from the date such reactions were first reported to or known by the person maintaining such records. Any other record of such adverse reactions shall be retained for a period of five years from the date the information contained in the record was first reported to or known by the person maintaining the record. Records required to be maintained under this subsection shall include records of consumer allegations of personal injury or harm to health, reports of occupational disease or injury, and reports or complaints of injury to the environment submitted to the manufacturer, processor, or distributor in commerce from any source. Upon request of any duly designated representative of the Administrator each person who is required to maintain records under this subsection shall permit the inspection of such records and shall submit copies of such records.

d) Notice to Administrator of substantial risks—Any person who manufactures, processes, or distributes in commerce a chemical substance or mixture and who obtains information which reasonably supports the conclusion that such substance or mixture presents a substantial risk of injury to health or the environment shall immediately inform the Administrator of such information unless such person has actual knowledge that the Administrator has been adequately informed of such information.
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<td>(b) Accreditation by State</td>
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<td>(1) Model plan</td>
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<td>(B) Plan requirements</td>
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<td>(xi) Housekeeping and personal hygiene practices, including the necessity of showers, and procedures to prevent asbestos exposure to an employee's family.</td>
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<td>For the purposes of this subchapter:</td>
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<td>(1) Abatement</td>
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<td>The term &quot;abatement&quot; means any set of measures designed to permanently eliminate lead-based paint hazards in accordance with standards established by the Administrator under this subchapter. Such term includes:</td>
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<td>(A) the removal of lead-based paint and lead-contaminated dust, the permanent containment or encapsulation of lead-based paint, the replacement of lead-painted surfaces or fixtures, and the removal or covering of lead-contaminated soil; and</td>
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<td>(B) all preparation, cleanup, disposal, and postabatement clearance testing activities associated with such measures.</td>
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<td>(11) Lead-contaminated dust</td>
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<td>The term &quot;lead-contaminated dust&quot; means surface dust in residential dwellings that contains an area or mass concentration of lead in excess of levels determined by the Administrator under this subchapter to pose a threat of adverse health effects in pregnant women or young children.</td>
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<td>(12) Lead-contaminated soil</td>
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<td>The term &quot;lead-contaminated soil&quot; means bare soil on residential real property that contains lead at or in excess of the levels determined to be hazardous to human health by the Administrator under this subchapter.</td>
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<td>§ 2683 Identification of dangerous levels of lead</td>
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<td>Within 18 months after October 28, 1992, the Administrator shall promulgate regulations which shall identify, for purposes of this subchapter, and the Residential Lead-Based Paint Hazard Reduction Act of 1992 [42 U.S.C.A. § 4851 et seq.], lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil.</td>
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(a) Program to promote lead exposure abatement  
The Administrator, in cooperation with other appropriate Federal departments and agencies, shall conduct a comprehensive program to promote safe, effective, and affordable monitoring, detection, and abatement of lead-based paint and other lead exposure hazards.  
(c) Exposure studies  
(1) The Secretary of Health and Human Services (hereafter in this subsection referred to as the "Secretary"), acting through the Director of the Centers for Disease Control (CDC), and the Director of the National Institute of Environmental Health Sciences, shall jointly conduct a study of the sources of lead exposure in children who have elevated blood lead levels (or other indicators of elevated lead body burden), as defined by the Director of the Centers for Disease Control.  
(3) The studies described in paragraphs (1) and (2) shall, as appropriate, examine the relative contributions to elevated lead body burden from each of the following:  
(A) Drinking water  
(B) Food  
(C) Lead-based paint and dust from lead-based paint  
(D) Exterior sources such as ambient air and lead in soil  
(E) Occupational exposures, and other exposures that the Secretary determines to be appropriate. |
(a) Storage, disposal, and transportation  
(1) Data requirements and registration of pesticides  
The Administrator may require under section 136(e) or 136(d) of this title that-  
(A) the registrant or applicant for registration of a pesticide submit or cite data or information regarding methods for the safe storage and disposal of excess quantities of the pesticide to support the registration or continued registration of a pesticide;  
(B) the labeling of a pesticide contain requirements and procedures for the transportation, storage, and disposal of the pesticide, any container of the pesticide, any rinseate containing the pesticide, or any other material used to contain or collect excess or spilled quantities of the pesticide; and  
(e) Container design  
(1) Procedures  
(A) Not later than 3 years after the effective date of this subsection, the Administrator shall, in consultation with the heads of other interested Federal agencies, promulgate regulations for the design of pesticide containers that will promote the safe storage and disposal of pesticides. |
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<td>Federal Insecticide Fungicide, Rodenticide Act</td>
<td>Public Law 92-316</td>
<td>[7 U.S.C. § 136]</td>
<td>(B) The regulations shall ensure, to the fullest extent practicable, that the containers-&lt;br&gt; (i) accommodate procedures used for the removal of pesticides from the containers and the rinsing of the containers;&lt;br&gt; (ii) facilitate the sale use of the containers, including elimination of splash and leakage of pesticides from the containers;&lt;br&gt; (iii) facilitate the safe disposal of the containers; and&lt;br&gt; (iv) facilitate the safe refill and reuse of the containers.&lt;br&gt; (2) Compliance&lt;br&gt; The Administrator shall require compliance with the regulations referred to in paragraph (1) not later than 5 years after the effective date of this subsection.&lt;br&gt; (f) Pesticide residue removal&lt;br&gt; (1) Procedures&lt;br&gt; (A) Not later than 3 years after the effective date of this subsection, the Administrator shall, in consultation with the heads of other interested Federal agencies, promulgate regulations prescribing procedures and standards for the removal of pesticides from containers prior to disposal.&lt;br&gt; (B) The regulations may-&lt;br&gt; (i) specify, for each major type of pesticide container, procedures and standards providing for, at a minimum, triple rinsing or the equivalent degree of pesticide removal;&lt;br&gt; (ii) specify procedures that can be implemented promptly and easily in various circumstances and conditions;&lt;br&gt; (iii) provide for reuse, whenever practicable, or disposal of rinse water and residue; and&lt;br&gt; (iv) be coordinated with requirements for the rinsing of containers imposed under the Solid Waste Disposal Act (42 U.S.C. § 6901 et seq.).&lt;br&gt; (C) The Administrator may, at the discretion of the Administrator, exempt products intended solely for household use from the requirements of this subsection.&lt;br&gt; (2) Compliance&lt;br&gt; Effective beginning 5 years after the effective date of this subsection, a State may not exercise primary enforcement responsibility under section 136w-1 of this title or certify an applicator under section 136i of this title, unless the Administrator determines that the State is carrying out an adequate program to ensure compliance with this subsection.</td>
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Nothing in the subsection shall affect the authorities or requirements concerning pesticide containers under the Solid Waste Disposal Act (42 U.S.C. § 6901).  

(g) Pesticide container study  
(1) Study  
(A) The Administrator shall conduct a study of options to encourage or require-  
(i) the return, refill, and reuse of pesticide containers;  
(ii) the development and use of pesticide formulations that facilitate the removal of pesticide residues from containers; and  
(iii) the use of bulk storage facilities to reduce the number of pesticide containers requiring disposal.  
(B) In conducting the study, the Administrator shall-  
(i) consult with the heads of other interested Federal agencies, State agencies, industry groups, and environmental organizations; and  
(ii) assess the feasibility, costs, and environmental benefits of encouraging or requiring various measures of actions. |
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(B) "environment" means (A) the navigable waters, the waters of the contiguous zone, and the ocean waters of which the natural resources are under the exclusive management authority of the United States under the Magnuson Fishery Conservation and Management Act [16 U.S.C.A. § 1801 et seq.], and (B) any other surface water, ground water, drinking water supply, land surface or subsurface strata, or ambient air within the United States or under the jurisdiction of the United States;  
(9) "facility" means (A) any building, structure, installation, equipment, pipe or pipe line (including any pipe into a sewer or publicly owned treatment works), well, pit, pond, lagoon, impoundment, ditch, landfill, storage container, motor vehicle, rolling stock, or aircraft, or (B) any site or area where a hazardous substance has been deposited, stored, disposed of, or placed, or otherwise come to be located; but does not include any consumer product in consumer use or any vessel;  
(14) "hazardous substance" means (A) any substance designated pursuant to section 1321(b)(2)(A) of Title 33, (B) any element, compound, mixture, solution, or substance designated pursuant to section 9602 of this title, (C) any hazardous waste having the characteristics identified under or listed pursuant to section 3001 of the Solid Waste Disposal Act [42 U.S.C.A. § 6921] (but not including any waste the regulation of which under the Solid Waste Disposal Act [42 U.S.C.A. § 6901 et seq.] has been suspended by Act of Congress), (D) any toxic pollutant listed under section 1317(a) of Title 33, (E) any hazardous air pollutant listed under section 112 of the Clean Air Act [42 U.S.C.A. § 7412], and (F) any imminently hazardous chemical substance or mixture with respect to which the Administrator has taken action pursuant to section 2606 of Title 15. The term does not include petroleum, including crude oil or any fraction thereof which is not otherwise specifically listed or designated as a hazardous substance under subparagraphs (A) through (F) of this paragraph, and the term does not include natural gas, natural gas liquids, liquefied natural gas, or synthetic gas usable for fuel (or mixtures of natural gas and such synthetic gas).  
(18) "onshore facility" means any facility (including, but not limited to, motor vehicles and rolling stock) of any kind located in, on, or under, any land or nonnavigable waters within the United States;  
(22) "release" means any spilling, leaking, pumping, pouring, emitting, emptying, discharging, injecting, escaping, leaching, dumping, or disposing into the environment, but excludes (A) any release which results in exposure to persons solely within a workplace, with respect to a claim which such persons may assert against the employer of such persons, (B) emissions from the engine exhaust of a motor vehicle, rolling stock, aircraft, vessel, or pipe line pumping station engine, (C) release of source, byproduct, or special nuclear material from a nuclear incident, as those terms are defined in the Atomic Energy Act of 1954 [42 U.S.C.A. § 2011 et seq.], if such release is subject to requirements with respect to financial protection established by the Nuclear Regulatory Commission under section 170 of such Act [42 U.S.C.A. § 2210] or, for the purposes of section 9604 of this title of any other response action, any release of source byproduct, or special nuclear |
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<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
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<td>(23) &quot;remove&quot; or &quot;removal&quot; means the cleanup or removal of released hazardous substances from the environment, such actions as may be necessary taken in the event of the threat of release of hazardous substances into the environment, such actions as may be necessary to monitor, assess, and evaluate the release or threat of release of hazardous substances, the disposal of removed material, or the taking of such other actions as may be necessary to prevent, minimize, or mitigate damage to the public health or welfare or to the environment, which may otherwise result from a release or threat of release. The term includes, in addition, without being limited to, security fencing or other measures to limit access, provision of alternative water supplies, temporary evacuation and housing of threatened individuals not otherwise provided for, action taken under section 9604(b) of this title, and any emergency assistance which may be provided under the Disaster Relief Act of 1974 [42 U.S.C.A. § 5121 et seq.]; So in original. Probably should be &quot;necessarily&quot;.</td>
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<td>(25) &quot;respond&quot; or &quot;response&quot; means remove, removal, remedy, and remedial action;</td>
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<td>(33) The term &quot;pollutant or contaminant&quot; shall include, but not be limited to, any element, substance, compound, or mixture, including disease-causing agents, which after release into the environment and upon exposure, ingestion, inhalation, or assimilation into any organism, either directly from the environment or indirectly by ingestion through food chains, will or may reasonably be anticipated to cause death, disease, behavioral abnormalities, cancer, genetic mutation, physiological malfunctions (including malfunctions in reproduction) or physical deformations, in such organisms or their offspring; except that the term &quot;pollutant or contaminant&quot; shall not include petroleum, including crude oil or any fraction thereof which is not otherwise specifically listed or designated as a hazardous substance under subparagraphs (A) through (F) of paragraph (14) and shall not include natural gas, liquefied natural gas, or synthetic gas of pipe line quality (or mixtures of natural gas and such synthetic gas).</td>
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<td>Note 3. Hazardous substances  To be considered hazardous substance under Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), substance need only be defined as hazardous under Solid Waste Disposal Act, Clean Air Act, Federal Water Pollution Control Act or Toxic Substances Control act or be so designated by Environmental Protection Agency pursuant to its authority under CERCLA. State of N.J., Dept. of Environmental Protection and Energy v. Gloucester Environmental Management Services, Inc., D.N.J. 1993, 821 F.Supp 999.</td>
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<td>Hazardous substances under CERCLA include all substances so designated under other statutes pursuant to the grant of authority contained therein, in addition to substances so designated by the Environmental Protection Agency (EPA) pursuant to the grant of authority in CERCLA. U.S. v. Alcan Aluminum Corp., N.D.N.Y.1991, 755 F.Supp. 531,</td>
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<td>Note 3c. Concentration, quantity, volume Waste material that is not specifically listed as hazardous substance in Environmental Protection Agency's (EPA's) designation of CERCLA hazardous substances is hazardous if it contains hazardous substances, regardless of volume or concentration. State of Ariz. v. Motorola, Inc., D.Ariz.1991, 774 F.Supp. 566.</td>
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<td>§ 9602. Designation of additional hazardous substances and establishment of reportable released quantities; regulations (a) The Administrator shall promulgate and revise as may be appropriate, regulations designating as hazardous substances, in addition to those referred to in section 9601(14) of this title, such elements, compounds, mixtures, solutions, and substances which, when released into the environment may present substantial danger to the public health or welfare or the environment, and shall promulgate regulations establishing that quantity of any hazardous substance the release of which shall be reported pursuant to section 9603 of this title. The Administrator may determine that one single quantity shall be the reportable quantity for any hazardous substance, regardless of the medium into which the hazardous substance is released. For all hazardous substances for which proposed regulations establishing reportable quantities were published in the Federal Register under this subsection on or before March 1, 1986, the Administrator shall promulgate under the subsection final regulations establishing reportable quantities not later than December 31, 1986. For all hazardous substances for which proposed regulations establishing reportable quantities were not published in the Federal Register under this subsection on or before March 1, 1986, the Administrator shall publish under this subsection proposed regulations establishing reportable quantities not later than December 31, 1986, and promulgate final regulations under this subsection establishing reportable quantities not later than April 30, 1988.</td>
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<td>§ 9604. Response authorities (a) Removal and other remedial action by President; applicability of national contingency plan; response by potentially responsible parties; public health threats; limitations on response: exception (1) Whenever (A) any hazardous substance is released or there is a substantial threat of such a release into the environment, or (B) there is a release or substantial threat of release into the environment of any pollutant or contaminant which may present an imminent and substantial danger to the public health or welfare, the President is authorized to act, consistent with the national contingency plan, to remove or arrange for the removal of, and provide for remedial action relating to such hazardous substance, pollutant, or contaminant at any time (including its removal from any contaminated natural resource), or take any other response measure consistent with the national contingency plan which the President deems necessary to protect the public health or welfare or the environment. When the President determines that such action will be done</td>
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<td>Popular Name</td>
<td>Public Law</td>
<td>U.S. Code</td>
<td>Sections Relevant to Workers' Family Protection</td>
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<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
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<td>(b) Investigations, monitoring, etc., by President.</td>
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<td>(1) Information; studies and investigations.</td>
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<td>Whenever the President is authorized to act pursuant to subsection (a) of this section or</td>
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<td>whenever the President has reason to believe that a release has occurred or is about to</td>
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<td>occur, or that illness, disease, or complaints thereof may be attributable to exposure to a</td>
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<td>hazardous substance, pollutant, or contaminant and that a release may have occurred or</td>
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<td>be occurring, he may undertake such investigations, monitoring, surveys, testing, and other</td>
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<td>information gathering as he may deem necessary or appropriate to identify the existence</td>
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<td>and extent of the release or threat thereof, the source and nature of the hazardous</td>
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<td>substances, pollutants or contaminants involved, and the extent of danger to the public</td>
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<td>health or welfare or to the environment. In addition, the President may undertake such</td>
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<td>planning, legal, fiscal, economic, engineering, architectural, and other studies or</td>
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<td>investigations as he may deem necessary or appropriate to plan and direct response</td>
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<td>actions, to recover the costs thereof, and to enforce the provisions of this chapter.</td>
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<td>(e) Information gathering and access; action authorized, access to information, entry,</td>
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<td>inspection and samples; authority and samples, compliance orders; issuance and</td>
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<td>compliance, other authority, confidentiality of information; basis for withholding</td>
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<td>(1) Action authorized.</td>
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<td>Any officer, employee, or representative of the President, duly designated by the</td>
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<td>President, is authorized to take action under paragraph (2), (3), or (4) (or any</td>
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<td>combination thereof) at a vessel, facility, establishment, place, property, or location or, in</td>
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<td>the case of paragraph (3) or (4), at any vessel, facility, establishment, place, property, or</td>
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<td>location which is adjacent to the vessel, facility, establishment, place, property, or location</td>
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<td>referred to in such paragraph (3) or (4). Any duly designated officer, employee, or</td>
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<td>representative of a State or political subdivision under a contract or cooperative</td>
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<td>agreement under subsection (d)(1)(i) of this section is also authorized to take such action.</td>
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<td>The authority of paragraphs (3) and (4) may be exercised only if there is a reasonable</td>
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<td>basis to believe there may be a release or threat of release of a hazardous substance or</td>
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<td>pollutant or contaminant. The authority of this subsection may be exercised only for the</td>
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<td>purposes of determining the need for response, or choosing or taking any response action</td>
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<td>under this subchapter, or otherwise enforcing the provisions of this subchapter.</td>
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<td>(2) Access to information.</td>
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<td>Any officer, employee, or representative described in paragraph (1) may require any</td>
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<td>person who has or may have information relevant to any of the following to furnish, upon</td>
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<td>reasonable notice, information or documents relating to such matter:</td>
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<td>(A) The identification, nature, and quantity of materials which have been or are</td>
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<td>generated, treated, stored, or disposed of at a vessel or facility or transported to a vessel</td>
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<td>or facility.</td>
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<td>(B) The nature or extent of a release or threatened release of a hazardous substance or</td>
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<td>pollutant or contaminant at or from a vessel or facility.</td>
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<td>(C) Information relating to the ability of a person to pay for or to perform a cleanup.</td>
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<td>In addition, upon reasonable notice, such person either (i) shall grant any such officer,</td>
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<td>employee, or representative access at all reasonable times to any vessel, facility,</td>
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<td>establishment, place, property, or location to inspect and copy all documents or records</td>
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<td>Popular Name</td>
<td>Public Law</td>
<td>U.S. Code</td>
<td>Sections Relevant to Workers' Family Protection</td>
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<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
<td></td>
<td>(B) Any vessel, facility, establishment, or other place or property from which or to which a hazardous substance or pollutant or contaminant has been or may have been released. (C) Any vessel, facility, establishment, or other place or property where such release is or may be threatened. (D) Any vessel, facility, establishment, or other place or property where entry is needed to determine the need for response or the appropriate response or to effectuate a response action under this subchapter.</td>
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(4) Inspection and samples

(A) Authority

Any officer, employee or representative described in paragraph (1) is authorized to inspect and obtain samples from any vessel, facility, establishment, or other place or property referred to in paragraph (3) or from any location of any suspected hazardous substance or pollutant or contaminant. Any such officer, employee, or representative is authorized to inspect and obtain samples of any containers or labeling for suspected hazardous substances or pollutants or contaminants. Each such inspection shall be completed with reasonable promptness.

(B) Samples

If the officer, employee, or representative obtains any samples, before leaving the premises he shall give to the owner, operator, tenant, or other person in charge of the place from which the samples were obtained a receipt describing the sample obtained and, if requested, a portion of each such sample. A copy of the results of any analysis made of such samples shall be furnished promptly to the owner, operator, tenant, or other person in charge, if such person can be located.

(i) Agency for Toxic Substances and Disease Registry; establishment, functions, etc.

(1) There is hereby established within the Public Health Service an agency, to be known as the Agency for Toxic Substances and Disease Registry, which shall report directly to the Surgeon General of the United States. The Administrator of said Agency shall, with the cooperation of the Administrator of the Environmental Protection Agency, the Commissioner of the Food and Drug Administration, the Directors of the National Institute of Medicine, National Institute of Environmental Health Sciences, National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention, the Administrator of the Occupational Safety and Health Administration, the Administrator of the Social Security Administration, the Secretary of Transportation, and appropriate State and local health officials, effectuate and implement the health related authorities of this chapter. In addition, said Administrator shall:

(A) in cooperation with the States, establish and maintain a national registry of serious diseases and illnesses and a national registry of persons exposed to toxic substances;

(B) establish and maintain inventory of literature, research, and studies on the health effects of toxic substances;
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<th>Popular Name</th>
<th>Public Law</th>
<th>U.S. Code</th>
<th>Sections Relevant to Workers' Family Protection</th>
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<tr>
<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
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<td>(C) in cooperation with the States, and other agencies of the Federal Government, establish and maintain a complete listing of areas closed to the public or otherwise restricted in use because of toxic substances contamination;</td>
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<td>(D) in cases of public health emergencies caused or believed to be caused by exposure to toxic substances, provide medical care and testing to exposed individuals, including but not limited to tissue sampling, chromosomal testing where appropriate, epidemiological studies, or any other assistance appropriate under the circumstances; and</td>
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<td>(E) either independently or as part of other health status survey, conduct periodic survey and screening programs to determine relationships between exposure to toxic substances and illness. In cases of public health emergencies, exposed persons shall be eligible for admission to hospitals and other facilities and services operated or provided by the Public Health Service.</td>
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<td>(2)(A) Within 6 months after October 17, 1986, the Administrator of the Agency for Toxic Substances and Disease Registry (ATSDR) and the Administrator of the Environmental Protection Agency (EPA) shall prepare a list, in order of priority, of at least 100 hazardous substances which are most commonly found at facilities on the National Priorities List and which, in their sole discretion, they determine are posing the most significant potential threat to human health due to their known or suspected toxicity to humans and the potential for human exposure to such substances at facilities on the National Priorities List or at facilities to which a response to a release or a threatened release under this section is under consideration.</td>
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<td>(B) Within 24 months after October 17, 1986, the Administrator of ATSDR and the Administrator of EPA shall revise the list prepared under subparagraph (A). Such revision shall include, in order of priority, the addition of 100 or more such hazardous substances. In each of the 3 consecutive 12-months periods that follow, the Administrator of ATSDR and the Administrator of EPA shall revise, in the same manner as provided in the 2 preceding sentences, such list to include not fewer than 25 additional hazardous substances per revision. The Administrator of ATSDR and the Administrator of EPA shall not less often than once every year thereafter revise such list to include additional hazardous substances in accordance with the criteria in subparagraph (A).</td>
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<td>(6)(A) The Administrator of ATSDR shall perform a health assessment for each facility on the National Priorities List established under section 9605 of this title. Such health assessment shall be completed not later than December 10, 1988, for each facility proposed for inclusion on such list prior to the date of the enactment of the Superfund Amendments and Reauthorization Act of 1986 or not later than one year after the date of proposal for inclusion on such list for each facility proposed for inclusion on such list after October 17, 1986.</td>
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Table 17. (Continued) Federal Laws Relevant to Workers' Home Contamination

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<th>Popular Name</th>
<th>Public Law</th>
<th>U.S. Code</th>
<th>Sections Relevant to Workers' Family Protection</th>
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<tr>
<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
<td>(B) The Administrator of ATSDR may perform health assessments for releases or facilities where individual persons or licensed physicians provide information that individuals have been exposed to a hazardous substance, for which the probable source of such exposure is a release. In addition to other methods (formal or informal) of providing such information, such individual persons or licensed physicians may submit a petition to the Administrator of ATSDR providing such information and requesting a health assessment. If such a petition is submitted and the Administrator of ATSDR does not initiate a health assessment, the Administrator of ATSDR shall provide a written explanation of why a health assessment is not appropriate.</td>
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<td>(C) In determining the priority in which to conduct health assessments under this subsection, the Administrator of ATSDR, in consultation with the Administrator of EPA, shall give priority to those facilities at which there is documented evidence of the release of hazardous substances, at which the potential risk to human health appears highest, and for which in the judgment of the Administrator of ATSDR existing health assessment data are inadequate to assess the potential risk to human health as provided in subparagraph (F). In determining the priorities for conducting health assessments under this subsection, the Administrator of ATSDR shall consider the National Priorities List Schedules and the needs of the Environmental Protection Agency and other Federal agencies pursuant to schedules for remedial investigation and feasibility studies.</td>
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<td>(D) Where a health assessment is done at a site on the National Priorities List, the Administrator of ATSDR shall complete such assessment promptly and, to the maximum extent practicable, before the completion of the remedial investigation and feasibility study at the facility concerned.</td>
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<td>(E) Any State or political subdivision carrying out a health assessment for a facility shall report the results of the assessment to the Administrator of ATSDR and the Administrator of EPA and shall include recommendations with respect to further activities which need to be carried out under this section. The Administrator of ATSDR shall state such recommendation in any report on the results of any assessment carried out directly by the Administrator of ATSDR for such facility and shall issue periodic reports which include the results of all the assessments carried out under this subsection.</td>
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<td>(F) For the purposes of this subsection and section 9611(c)(4) of this title the term &quot;health assessments&quot; shall include preliminary assessments of the potential risk to human health posed by individual sites and facilities, based on such factors as the nature and extent of contamination, the existence of potential pathways of human exposure (including ground or surface water contamination, air emissions, and food chain contamination), the size and potential susceptibility of the community within the likely pathways of exposure, the comparison of expected human exposure levels to the short-term and long-term health effects associated with identified hazardous substances and any available recommended exposure or tolerance limits for such hazardous substances, and the comparison of existing morbidity and mortality data on diseases that may be associated with the observed levels of exposure. The Administrator of ATSDR shall use appropriate data, risk assessments, risk evaluations and studies available from the Administrator of EPA.</td>
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<th>Popular Name</th>
<th>Public Law</th>
<th>U.S. Code</th>
<th>Sections Relevant to Workers' Family Protection</th>
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<tr>
<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
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<td>(G) The purpose of health assessments under this subsection shall be to assist in determining whether actions under paragraph (11) of this subsection should be taken to reduce human exposure to hazardous substances from a facility and whether additional information on human exposure and associated health risks is needed and should be acquired by conducting epidemiological studies under paragraph (7), establishing a registry under paragraph (8), establishing a health surveillance program under paragraph (9), or through other means. In using the results of health assessments for determining additional actions to be taken under this section, the Administrator of ATSDR may consider additional information on the risks to the potentially affected population from all sources of such hazardous substances including known point or nonpoint sources other than those from the facility in question. (11) At the completion of each health assessment, the Administrator of ATSDR shall provide the Administrator of EPA and each affected State with the results of such assessment, together with any recommendations for further actions under this subsection or otherwise under this chapter. In addition, if the health assessment indicates that the release or threatened release concerned may pose a serious threat to human health or the environment, the Administrator of ATSDR shall so notify the Administrator of EPA who shall promptly evaluate such release or threatened release in accordance with the hazard ranking system referred to in section 905(a)(8)(A) of this title to determine whether the site shall be placed on the National Priorities List or, if the site is already on the list, the Administrator of ATSDR may recommend to the Administrator of EPA that the site be accorded a higher priority. (7)(A) Whenever in the judgment of the Administrator of ATSDR it is appropriate on the basis of the results of a health assessment, the Administrator of ATSDR shall conduct a pilot study of health effects for selected groups of exposed individuals in order to determine the desirability of conducting full scale epidemiological or other health studies of the entire exposed population. (B) Whenever in the judgment of the Administrator of ATSDR it is appropriate on the basis of the results of such pilot study or other study or health assessment, the Administrator of ATSDR shall conduct such full scale epidemiological or other health studies as may be necessary to determine the health effects on the population exposed to hazardous substances from a release or threatened release. If a significant excess of disease in a population is identified, the letter of transmittal of such study shall include an assessment of other risk factors, other than a release, that may, in the judgment of the peer review group, be associated with such disease, if such risk factors were not taken into account in the design or conduct of the study. (8) In any case in which the results of a health assessment indicate a potential significant risk to human health, the Administrator of ATSDR shall consider whether the establishment of a registry of exposed persons would contribute to accomplishing the purposes of this subsection, taking into account circumstances bearing on the usefulness of such a registry, including the seriousness of unique character of identified diseases or the likelihood of population migration from the affected area.</td>
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<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
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<td>(9) Where the Administrator of ATSDR has determined that there is a significant increased risk of adverse health effects in humans from exposure to hazardous substances based on the results of a health assessment conducted under paragraph (6), an epidemiologic study conducted under paragraph (7), or an exposure registry that has been established under paragraph (8), and the Administrator of ATSDR has determined that such exposure is the result of a release from a facility, the Administrator of ATSDR shall initiate a health surveillance program for such population. This program shall include but not be limited to: (A) periodic medical testing where appropriate of population subgroups to screen for diseases for which the population or subgroup is at significant increased risk; and (B) a mechanism to refer for treatment those individuals within such population who are screened positive for such diseases.</td>
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<td>(11) If a health assessment or other study carried out under this subsection contains a finding that the exposure concerned presents a significant risk to human health, the President shall take such steps as may be necessary to reduce such exposure and eliminate or substantially mitigate the significant risk to human health. Such steps may include the use of any authority under this chapter, including, but not limited to: (A) provision of alternative water supplies, and (B) permanent or temporary relocation of individuals. In any case in which information is insufficient, in the judgment of the Administrator of ATSDR or the President to determine a significant human exposure level with respect to a hazardous substance, the President may take such steps as may be necessary to reduce the exposure of any person to such hazardous substance to such level as the President deems necessary to protect human health.</td>
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<td>(12) In any case which is the subject of a petition, a health assessment or study, or a research program under this subsection, nothing in this subsection shall be construed to delay or otherwise affect or impair the authority of the President, the Administrator of ATSDR, or the Administrator of EPA to exercise any authority vested in the President, the Administrator of ATSDR or the Administrator of EPA under any other provision of law (including, but not limited to, the imminent hazard authority of section 7003 of the Solid Waste Disposal Act [42 U.S.C.A. § 6973]) or the response and abatement authorities of this chapter.</td>
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<td>(18) If the Administrator of ATSDR determines that it is appropriate for purposes of this section to treat a pollutant or contaminant as a hazardous substance, such pollutant shall be treated as a hazardous substance for such purpose.</td>
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<td>§ 9605 National contingency plan; preparation, contents, etc. (a) Revision and republication Within one hundred and eighty days after December 11, 1980, the President shall, after notice and opportunity for public comments, revise and republish the national contingency plan for the removal of oil and hazardous Title 33, to reflect and effectuate the responsibilities and powers created by this chapter, in addition to those matters specified in section 1321(c)(2) of Title 33. Such revision shall include a section of the plan to be</td>
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<td>Sections Relevant to Workers' Family Protection</td>
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<tr>
<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
<td>(1) methods of discovering and investigating facilities at which hazardous substances have been disposed of or otherwise come to be located;</td>
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<td>(2) methods of evaluating, including analyses of relative cost, and remedying any releases or threats of releases from facilities which pose substantial danger to the public health or the environment;</td>
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<td>(3) methods and criteria for determining the appropriate extent of removal, remedy, and other measures authorized by this chapter;</td>
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<td>(4) appropriate roles and responsibilities for the Federal, State, and local governments and for interstate and nongovernmental entities in effectuating the plan;</td>
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<td>(5) provision for identification, procurement, maintenance, and storage of response equipment and supplies;</td>
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<td>(6) a method for and assignment of responsibility for reporting the existence of such facilities which may be located on federally owned or controlled properties and any releases of hazardous substances from such facilities;</td>
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<td>(7) means of assuring that remedial action measures are cost-effective over the period of potential exposure to the hazardous substances or contaminated materials;</td>
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<td>(8)(A) criteria for determining priorities among releases or threatened releases throughout the United States for the purpose of taking remedial action and, to the extent practicable taking into account the potential urgency of such action, for the purpose of taking removal action. Criteria and priorities under this paragraph shall be based upon relative risk or danger to public health or welfare or the environment, in the judgment of the President, taking into account to the extent possible the population at risk, the hazard potential of the hazardous substances at such facilities, the potential for contamination of drinking water supplies, the potential for direct human contact, the potential for destruction of sensitive ecosystems, the damage to natural resources which may affect the human food chain and which is associated with any release or threatened release, the contamination or potential contamination of the air which is associated with the release or threatened release. State preparedness to assume State costs and responsibilities, and other appropriate factors;</td>
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|              |            | (B) based upon the criteria set forth in subparagraph (A) of this paragraph, the President shall list as part of the plan national priorities among the known releases or threatened releases throughout the United States and shall review the list no less often than annually. Within one year after December 11, 1980, and annually thereafter, each State shall establish and submit for consideration by the President priorities for remedial action among known releases and potential releases in that State based upon the criteria set forth in subparagraph (A) of this paragraph. In assembling or revising the national list, the President shall consider any priorities established by the States. To the extent practicable, the highest priority facilities shall be designated individually and shall be referred to as the "top priority among known response targets"; and, to the extent
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<tr>
<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
<td>priority facility only once. Other priority facilities or incidents may be listed singly or grouped for response priority purposes;</td>
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(9) specified roles for private organizations and entities in preparation for response and in responding to releases of hazardous substances, including identification of appropriate qualifications and capacity therefor and including consideration of minority firms in accordance with subsection (f) of this section.

The plan shall specify procedures, techniques, materials, equipment, and methods to be employed in identifying, removing, orremedying releases of hazardous substances comparable to those required under section 1321(c)(2) (F) and (G) and (j)(1) of Title 33. Following publication of the revised national contingency plan, the response to and actions to minimize damage from hazardous substances releases shall, to the greatest extent possible, be in accordance with the provisions of the plan. The President may, from time to time, revise and republish the national contingency plan.

(b) Revision of plan

Not later than 18 months after October 17, 1986, the President shall revise the National Contingency Plan to reflect the requirements of such amendments. The portion of such Plan known as "the National Hazardous Substance Response Plan" shall be revised to provide procedures and standards for remedial actions undertaken pursuant to this chapter which are consistent with amendments made by the Superfund Amendments and Reauthorization Act of 1986 relating to the selection of remedial action.

(c) Hazard ranking system

(1) Revision

Not later than 18 months after October 17, 1986, and after publication of notice and opportunity for submission of comments in accordance with section 553 of Title 5, the President shall by rule promulgate amendments to the hazard ranking system in effect on September 1, 1984. Such amendments shall assure, to the maximum extent feasible, that the hazard ranking system accurately assesses the relative degree of risk to human health and the environment posed by sites and facilities subject to review. The President shall establish an effective date for the amended hazard ranking system which is not later than 24 months after October 17, 1986. Such amended hazard ranking system shall be applied to any site or facility to be newly listed on the National Priorities List after the effective date established by the President. Until such effective date of the regulations, the hazard ranking system in effect on September 1, 1984, shall continue in full force and effect.

(d) Petition for assessment of release

Any person who is, or may be, affected by a release or threatened release of a hazardous substance or pollutant or contaminant, may petition the President to conduct a preliminary assessment of the hazards to public health and the environment which are associated with such release or threatened release. If the President has not previously conducted a preliminary assessment of such release, the President shall, within 12 months after the receipt of any such petition, complete such assessment or provide an explanation of why the assessment is not appropriate. If the preliminary assessment indicates that the release or threatened release concerned may pose a threat to human health or the
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<tr>
<td>CERCLA &amp; SARA</td>
<td>[42 U.S.C. § 9601 et seq.]</td>
<td>priority of such release or threatened release.</td>
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<td>§ 9611 Use of Fund</td>
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<td>(3) Subject to such amounts as are provided in appropriate Acts, the costs of a program to identify, investigate, and take enforcement and abatement action against releases of hazardous substances.</td>
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<td>(4) Any costs incurred in accordance with subsection (m) of this section (relating to ATSDR) and section 9604(l) of this title including the costs of epidemiologic and laboratory studies, health assessments, preparation of toxicologic profiles, development and maintenance of a registry of persons exposed to hazardous substances to allow long-term health effect studies, and diagnostic services not otherwise available to determine whether persons in populations exposed to hazardous substances in connection with a release or a suspected release are suffering from long-latency diseases.</td>
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<td>(6) Subject to such amounts as are provided in appropriation Acts, the costs of a program to protect the health and safety of employees involved in response to hazardous substance releases. Such program shall be developed jointly by the Environmental Protection Agency, the Occupational Safety and Health Administration, and the National Institute for Occupational Safety and Health and shall include, but not be limited to, measures for identifying and assessing hazards to which persons engaged in removal, remedy, or other response to hazardous substances may be exposed, methods to protect workers from such hazards, and necessary regulatory and enforcement measures to assure adequate protection of such employees.</td>
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<td><strong>Inorganic arsenic</strong>&lt;br&gt;[29 CFR 1910.1000] (general industry)&lt;br&gt;[29 CFR 1915.1018] (shipyards)&lt;br&gt;[29 CFR 1926.1118] (construction)</td>
<td>OSHA</td>
<td>The PEL is 10 μg/m³ as an 8-hr. average. Where employees work in areas where exposure concentrations exceed 10 μg/m³ or when the possibility of skin or eye irritation from inorganic arsenic exists, the employer must provide clean protective work clothing weekly (daily if exposure levels exceed 100 μg/m³). Protective clothing must be removed in change rooms and placed in a closed container prior to removal for cleaning, laundering or disposal. The container must be labeled and the launderer informed of the hazards. When exposures exceed 10 μg/m³, the employer must provide showers and separate storage facilities for street and work clothes.</td>
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<td><strong>Asbestos</strong>&lt;br&gt;[29 CFR 1910.1001] (general industry)</td>
<td>OSHA</td>
<td>PEL is 0.1 fiber/cc as an 8-hr. average. Excursion Limit (EL) is 1 fiber/cc as a 30-min. average. Where employees are exposed above these limits, or where the possibility of eye irritation exists, the employer must provide, and ensure: that the employee wears appropriate protective work clothing; that contaminated clothing is removed only in change rooms; that no employee takes contaminated work clothing out of the change room (except those authorized to do so for purposes of laundry, maintenance and disposal); that containers for contaminated clothing are labeled; that laundering is done in a way that will minimize release of fibers to the air; that the launderer be informed of the hazards; and that contaminated clothing be transported in sealed containers. The employer must provide facilities to assure that street clothes do not become contaminated if the employees' exposures exceed the PEL, that such employees shower at the end of the work shift and that they do not leave the workplace with any clothing or equipment worn during the work shift. In addition to the requirements for general industry, clothes of workers who work in certain regulated areas where the decontamination area and the shower cannot be located next to the regulated area, must be vacuumed with a HEPA vacuum cleaner before proceeding to the shower, or the employee must remove contaminated clothing in the equipment room and don clean work suits before proceeding to the shower. For other regulated areas, work clothing must be vacuumed before it is removed, but showering is not required.</td>
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<td><strong>Cadmium</strong>&lt;br&gt;[29 CFR 1910.1027] (general industry)&lt;br&gt;[29 CFR 1915.1027] (shipyards)&lt;br&gt;[29 CFR 1926.1127] (construction)</td>
<td>OSHA</td>
<td>The PEL is 5 μg/m³ as an 8-hr. average. If an employee is exposed above the PEL or where skin or eye irritation is associated with cadmium at any level, the employer must provide clothing and equipment that prevents contamination of the employee and the employee's garments. Contaminated clothing must be removed at the end of the work shift in change rooms which have separate storage facilities for street clothes and work clothes. The facilities must be designed to prevent contamination of the street clothes. The employer must assure that employees exposed above the PEL shower during the end of the work shift. The employer must assure that no employee takes contaminated protective clothing from the workplace, except when authorized to do so for laundry, cleaning, maintenance or disposal at an appropriate location or facility. Contaminated clothing must be stored in a closed container, and labeled. Launderers and cleaners must be informed of hazards.</td>
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<td><strong>Hazard Communication</strong>&lt;br&gt;[29 CFR 1910.1200] (general industry)&lt;br&gt;[29 CFR 1915.1200] (shipyards)&lt;br&gt;[29 CFR 1926.59] (construction)</td>
<td>OSHA</td>
<td>These standards have elements that could be used for preventing workers' home contamination. These elements include: the written hazard communication which employers must prepare; the requirements that all containers of hazardous chemicals be labeled; the requirement for preparation of material safety data sheets containing information on applicable precautions for safe handling and use, including appropriate hygiene practices, work practices, or personal protective equipment; the requirement for employee information and training.</td>
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<td><strong>Hazardous Waste Operations and Emergency Response</strong>&lt;br&gt;[29 CFR 1910.120] (general industry)&lt;br&gt;[29 CFR 1926.65] (construction)</td>
<td>OSHA</td>
<td>These standards require a written safety and health program for employees involved in hazardous waste operations. Among the requirements are: use of appropriate protective equipment for each hazardous waste site; appropriate decontamination of all employees and contaminated clothing and equipment before leaving the area; location of the decontamination procedures to minimize cross-contamination; removal of protective clothing or equipment from the site only by authorized employees; advising laundries and cleaning establishments of the hazards of contaminated clothing; and provision of showers and change rooms outside of the contaminated area, when the need is indicated.</td>
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<td>Lead</td>
<td>OSHA</td>
<td>The PEL is 50 µg/m³ as an 8-hr. average. Where employees are exposed above 50 µg/m³ the employer must: provide clean protective clothing at least weekly (daily if exposures exceed 200 µg/m³); provide clean change rooms equipped with separate storage facilities for work and street clothes which prevent cross-contamination; provide showers and ensure that employees shower at the end of the work shift; and ensure that employees required to shower do not leave the workplace wearing any clothing or equipment worn during the work shift. Appendix B to these standards advises the worker of the benefits imparted to the family by these requirements.</td>
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<td>[29 CFR 1926.62] (construction)</td>
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<td>Mandatory Health Standards - Surface Coal Mines and Surface Work Areas of Underground Coal Mines</td>
<td>MSHA</td>
<td>Requires each operator of a surface coal mine to provide bathing facilities and clothing change rooms in a convenient location with individual storage container or lockers for miners’ clothing during and between shifts.</td>
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<td>[30 CFR 71.400-71.404]</td>
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<tr>
<td>Mandatory Safety Standards - Underground Coal Mines</td>
<td>MSHA</td>
<td>Requires each operator of an underground coal mine to provide bathing facilities (showers with both hot and cold water) and change rooms with individual storage containers or lockers and sufficient room to permit the use of the facilities by all miners changing clothes prior to and after each shift.</td>
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<td>[30 CFR 75.1712]</td>
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<td>Labeling Requirements for Pesticides and Devices</td>
<td>EPA</td>
<td>Among the several requirements for labels are: a child hazard warning &quot;keep out of reach of children&quot; must appear on every pesticide product label, with few exceptions; clearly stated directions for use, including worker protection statements required by the worker protection standard; specific directions for storage and disposal of the pesticide and its container. Worker protection statements required on pesticide labels for products to be used in the production of agricultural plants on any agricultural establishment include statements on: restricted entry; worker notification; and personal protective equipment.</td>
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<td>[40 CFR 156.10] [40 CFR 156.20]</td>
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<td>Packaging Requirements for Pesticides and Devices</td>
<td>EPA</td>
<td>This rule requires child-resistant packaging for pesticide products and devices, however a product restricted to use by or under the supervision of a certified applicator is exempt from this requirement unless EPA determines that the product poses a risk of serious accidental injury or illness which child-resistant packaging would reduce. Certain products packaged in large-sized containers are also exempt unless EPA determines that it is to be sold to homeowners or other members of the general public.</td>
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<td>[40 CFR 157]</td>
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<td>Regulations for the Acceptance of Certain Pesticides and Recommended Procedures for the Disposal and Storage of Pesticides and Pesticide Containers</td>
<td>EPA</td>
<td>Procedures for storage and disposal of pesticides and pesticide containers are recommended, but are mandatory only for EPA in carrying out its disposal and storage operations. Recommendations for disposal of small quantities include rinsing empty containers three times, adding the rinse liquids to spray mixtures in the field, burning, where permissible, in open fields, or buried singly in open fields by the user.</td>
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<td>[40 CFR 165]</td>
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<td>Worker Protection Standard [40 CFR 170]</td>
<td>EPA</td>
<td>This standard generally applies to the use of pesticides on agricultural establishments for the production of plants for commercial purposes. The owners of agricultural establishments need not assure that the protective measures are provided to themselves and members of their immediate family while they are performing tasks related to production of agricultural plants on their own agricultural establishment, although they are encouraged to do so. The standard delineates restrictions on entering areas treated with pesticides; defines protective clothing, and its use and decontamination; storage requirements for clean protective clothing; a requirement that contaminated clothing be stored and washed separately from other clothing or laundry; a requirement that persons cleaning or laundering protective clothing be informed of the contamination and correct ways to handle and clean it; a requirement that all clean personal protective equipment be stored separately from personal clothing and apart from contaminated areas. The agribusiness employer must not allow or direct any worker to wear home or take home personal protective equipment contaminated with pesticides.</td>
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<td>Certification of Pesticide Applicators [40 CFR 171]</td>
<td>EPA</td>
<td>This standard generally requires that applicators be competent in the use and handling of pesticides they use, that they can read and understand the label and instructions for use, storage, and disposal of the pesticides and containers.</td>
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<tr>
<td>National Oil and Hazardous Substances Pollution Contingency Plan [40 CFR 300]</td>
<td>EPA</td>
<td>The purpose of the plan is to provide organizational structure and procedures for preparing for and responding to discharges of oil and releases of hazardous substances, and contaminants. The plan defines size classes of releases, includes the National Priorities List of hazardous waste sites, provides for worker protection in responding to releases and working at hazardous waste sites, and community right-to-know provisions.</td>
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<td>Worker Protection [40 CFR 311]</td>
<td>EPA</td>
<td>Applies 29 CFR 1910.120 to State and local government employees engaged in hazardous waste operations in States that do not have an OSHA approved State occupational safety and health plan.</td>
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<td>Asbestos [40 CFR 763]</td>
<td>EPA</td>
<td>Extends worker protection of the OSHA asbestos standard for construction to employees of local education agencies who perform operations, maintenance repair activities involving Asbestos-Containing Materials (ACM). Extends construction standard to employees of State and local governments not covered by OSHA, an approved OSHA State plan, or State asbestos regulation which EPA determines is comparable to or more protective than this standard (40 CFR 763). Appendix C to Subpart E (Asbestos-Containing Materials in Schools) entitled 'Asbestos Model Accreditation Plan for States' requires asbestos workers training to include information on &quot;potential exposures, such as family exposures.&quot;</td>
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<td>Occupational Safety and Health Administration</td>
<td>The OSHA office in Austin, Texas investigated a referral from the Texas State Health Department concerning a child with a high BLL. The father's BLL was also high and his workplace had high lead levels. OSHA did not inspect the home but made recommendations to prevent further contamination. The State has a hazard training standard where employers must inform employees of hazardous substances. See Natarajan [1994] in Table 9 for details.</td>
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<td>(OSHA) Austin, Texas</td>
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<td>[Nicholas 1994; Natarajan 1994]</td>
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<td>OSHA</td>
<td>The OSHA office of Wichita, Kansas had no data on home contamination and stated that its jurisdiction is the workplace only. However, based on feedback from chemical workers, home contamination was a common occurrence. Where applicable, the OSHA standards requiring showers, protective clothing, and employee training are used to prevent home contamination.</td>
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<td>Wichita, Kansas</td>
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<td>[Goldberg 1994]</td>
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<td>OSHA</td>
<td>The author pointed out that there are no provisions to ensure that contamination is removed from a worker's clothing if an over exposure cannot be proven.</td>
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<td>Hawaii</td>
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<td>[Goo 1994]</td>
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<td>OSHA</td>
<td>The health department in Kankakee, Illinois referred a case of home contamination to OSHA. Resulting in an OSHA inspection of the father's workplace. In another case the Indiana OSHA referred a case of home contamination to a Marion County health agency which then found an elevated BLL in a child. Yet in another case the Cleveland area OSHA office discovered during a workplace inspection some of the employees' children had elevated BLLs. OSHA made measurements of home contamination and referred the case to the Ohio Department of Health. See Wiehrdt [1994] in Tables 9 and 15.</td>
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<td>Chicago, Illinois (regional office)</td>
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<td>[Wiehrdt 1994]</td>
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<td>Mine Safety and Health Administration (MSHA)</td>
<td>MSHA submitted two reports of workers' home contamination by mercury.</td>
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<td>Zalesak [1994]</td>
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<td>Department of the Interior</td>
<td>Reported that home contamination has not been a problem at the National Fisheries Contamination Research Center. Personal protective equipment is used and Center has hygiene plan.</td>
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<td>[Heine 1994]</td>
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<td>Department of Energy</td>
<td>Sixteen reports of cases including 3 incidents of home contamination were extracted from DOE's Occurrence Reporting and Processing System (ORPS) reporting system for review by NIOSH. The Department takes follow-up action such as major revisions in facility decontamination procedures.</td>
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<td>[Boyle 1994]</td>
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<td>Nuclear Regulatory Commission</td>
<td>NRC has two databases, one for reactor related events and the other for non-reactor related events. A NRC search of these two databases found 34 incidents of off-site contamination in the reactor related file of which several had potential for worker home contamination. In the non-reactor events database they found 80 off-site contamination events of which 6 had potential for worker home contamination.</td>
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<td>[Brockman 1993]</td>
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<td>Agency for Toxic Substances and Disease Registry (ATSDR) [Alabama Department of Public Health 1991]</td>
<td>The Alabama Department of Health conducted an exposure study of children living near a lead reclamation factory in Alabama. Although the investigators recognized the potential for workers' home contamination, households of employees of the factory did not participate. Possible reasons for non-participation are discussed in the report.</td>
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<td>ATSDR [1989b]</td>
<td>A study in Michigan to determine the extent of trackout from the workplace was reported of the homes of workers exposed to MOCA while manufacturing plastics. See ATSDR [1989b] in Tables 12 and 15.</td>
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<td>ATSDR [1993a]</td>
<td>In this study of a North Carolina hazardous waste incinerator, EPA conducted site evaluations, NIOSH conducted neurotoxicity studies on workers, and ATSDR studied the health of area residents. ATSDR was to do study of workers' families and homes, but was unable to get sufficient volunteers.</td>
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<td>ATSDR [1991a]</td>
<td>In this Philadelphia neighborhood lead study, exposure of children was evaluated; children of lead workers were included. See ATSDR [1991a] in Table 8.</td>
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<td>ATSDR [1989a; 1990b; 1993b]</td>
<td>Anderson Development Company, Adrian, Michigan manufactured MOCA. Detectable levels of MOCA were found in urine of workers' families. Professional cleaning of homes was reported (carpets, baseboards, hard covered floors) after lab tests (dryer lint, furnace filters, vacuum bags, urine of family members) showed home contamination. Currently conducting a study for bladder cancer of workers and workers' families. See also ATSDR [1989a, 1990b] in Table 12.</td>
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<td>ATSDR [1991b]</td>
<td>At the Bofors - Nobel, Inc. chemical manufacturing site in Michigan. 3,3'-Dichlorobenzidine was found in homes of some workers (vacuum cleaner bags) and in the urine of some workers and family members. See ATSDR [1991b] in Tables 12 and 15.</td>
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<tr>
<td>Environmental Protection Agency (EPA)</td>
<td>This report contains a protocol developed by an EPA contractor for monitoring and cleaning homes contaminated with mercury.</td>
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<td>ERM-Southeast, Inc. [1989]</td>
<td>This report contains a protocol for cleaning homes contaminated with lead and asbestos.</td>
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<td>Beegle and Forslund [1990]</td>
<td>In this text of EPA answers to follow-up questions from the Senate hearing on Lead in the Environment, EPA's recognition of home contamination with lead from the workplace as a serious problem is stated. EPA is working cooperatively with OSHA and Labor groups to develop a research agenda for this issue. Although EPA's role is to identify research to minimize home exposure, EPA does not generally respond to specific home contamination incidents.</td>
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<td>Fisher [1991]</td>
<td>In this report of a Superfund site in Missouri, complete removal of dioxin contaminated soil and materials around homes and clean-up of home interiors is described.</td>
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<td>Ramsey [1987]</td>
<td>In this report of a Missouri Superfund site, removal and replacement of dioxin contaminated roads and decontamination of houses and businesses is described. See Hess [1988] in Table 15.</td>
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<td>Hess [1988]</td>
<td>In this report of a Missouri Superfund site, cleanup of mobile home park, including decontamination of mobile homes, contaminated with dioxin is described.</td>
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<td>MacDonald [1988]</td>
<td>In this report on EPA supported testing of human adipose tissue samples for PCBs, the authors also reported on PCBs in the house dust of workers homes. See Table 15.</td>
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<td>Price and Welch [1972]</td>
<td>In this report of a Missouri Superfund site, removal and replacement of a dioxin-contaminated roadway and decontamination of houses is described. See Table 15.</td>
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<td>Centers for Disease Control and Prevention (CDC)</td>
<td>A series of tests were conducted on a woman’s coat containing asbestos (8%) to determine magnitude of exposure associated with wearing, brushing, or cleaning the coat. Found transfer to companion clothing during laundry.</td>
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<td>NIOSH [1971]</td>
<td>Survey of Diamond Shamrock Company in Redwood City, California, to determine exposure of workers to bis (chlorodimethyl) ether. The employees paid half the cost of the work clothes and although the company paid for laundering, they could take their work clothes home.</td>
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<td>Donaldson and Johnson [1972]</td>
<td>Proctor and Gamble in Blue Ash, Ohio. This was a study of asbestos exposure in workers involved in spraying fireproof insulation at new building site. Workers were covered with insulation material, did not have respirators, and took clothing home at end of day. Recommended supply of respirators and clothing and laundry by contractor.</td>
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<td>Lemen [1972]</td>
<td>Grace Bleachery, Lancaster, North Carolina (textile facility). This was a study to determine the presence and extent of exposure to bis-chloromethyl ether. Workers did not shower or change work clothes before leaving for home and were seen eating in production areas at work.</td>
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<tr>
<td>Marcileno et al. [1974]</td>
<td>Testimony of John F. Finkles to Congress. RE: exposure to kepone at Life Science Products plant in Virginia. Found that Kepone contaminated work clothes were often worn home.</td>
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<tr>
<td>Finkles [1976]</td>
<td>Joseph K. Wagoner’s testimony to the subcommittee of House committee on Interstate and Foreign Commerce pointed out that wives, children, and relatives of asbestos workers have died from disease related to asbestos exposure from worker’s clothing brought home.</td>
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<tr>
<td>Beiermann [1993]</td>
<td>Survey of wood preservative treatment facility (Creosote) at Koppers Company, Inc., Florence, South Carolina. Some employees had work uniforms, but others brought work clothing from home. Most employees changed clothes before leaving work, but not all employees showered, although shower facilities were provided.</td>
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<tr>
<td>Todd and Timbie [1979]</td>
<td>Kentile Floors, Inc. in Chicago, Illinois. Investigation of worker exposure to asbestos and other chemicals. Manufacturer of vinyl floors and asbestos floor covering. Workers required to wear coveralls, respirators, and safety shoes, and can shower, but not required to do so.</td>
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</tr>
<tr>
<td>Belanger et al. [1979]</td>
<td>Published paper of stained glass workers - hobbyist, professionals and families, BLLs were related to lead in workplaces and workers’ homes. Recommended that contaminated work clothes not be worn home and be laundered separately.</td>
<td></td>
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<tr>
<td>Landigan et al. [1980]</td>
<td>Alaskan Battery Enterprises, Fairbanks, Alaska. Evaluated lead exposure among workers manufacturing lead-acid storage batteries. Looked at worker BLLs. Owner and family lived above the plant and 4 employees were children. Home entered through hallway that opened into plant. Recommended shower and change room, good housekeeping and redesign of entry to house.</td>
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<thead>
<tr>
<th>FEDERAL AGENCY</th>
<th>RESPONSE</th>
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<tbody>
<tr>
<td>Bridbord [1980]</td>
<td>Book Chapter - Lead exposure. The author points out that lead adversely affects sperm, and the fetus; that lead can be brought home as dust on shoes, clothing, and body; and that elevated blood levels have been found in children of workers exposed to lead (e.g. battery manufacturing).</td>
</tr>
<tr>
<td>Clapp et al. [1985]</td>
<td>Steinsmeier &amp; Sons, Moscow, Pennsylvania. Workers in this factory were exposed to MOCA. It was recommended that workers get clothing from company that is laundered daily and not worn home, use shoe covers, and shower before leaving work.</td>
</tr>
<tr>
<td>Aw et al. [1985]</td>
<td>Manufacturing Chemists, Inc., Indiana. An animal growth promoter (estrogenic) was found in worker's clothing which were laundered at home. Extensive recommendations were made to prevent home contamination.</td>
</tr>
<tr>
<td>Seixas and Ordin [1986]</td>
<td>Friction Division Products, Trenton, New Jersey. In this plant in which auto and truck break shoes were manufactured, samples from workers' clothing as they left the plant showed asbestos, raising potential for home contamination.</td>
</tr>
<tr>
<td>Eisenbud and Liason [1983]</td>
<td>Update of Beryllium cases - Up to 1983 no new cases of berylliosis had been reported since 1950.</td>
</tr>
<tr>
<td>CDC [1992a]</td>
<td>CDC's MMWR report on lead exposures among lead burners. Wipe samples were taken from changing room, toe of workboot, and floor under auto gas pedal. BLLs of family members of 2 workers were measured. The company implemented additional hygiene practices.</td>
</tr>
<tr>
<td>Knishkowy and Baker [1986]</td>
<td>Journal article. Contamination routes between work and home, types of illnesses that resulted, and preventive measures are discussed.</td>
</tr>
<tr>
<td>Hartle et al. [1987]</td>
<td>Aluminum Company of America, Lafayette, Indiana. Investigation of exposure to PCBs. It was recommended that exposed personnel be provided with protective equipment, including company-laundered coveralls, change rooms, and showers.</td>
</tr>
<tr>
<td>Ehrenberg et al. [1986]</td>
<td>Staco, Inc., Poulney, Vermont. Thermometer manufacturing plant. NIOSH trailer where tests were given became contaminated with mercury, suggesting possible home contamination.</td>
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<tr>
<td>FEDERAL AGENCY</td>
<td>RESPONSE</td>
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<tr>
<td>Hartle [1987]</td>
<td>This was a joint request to NIOSH from EPA and ATSDR for technical assistance in connection with a PCB contaminated Amtrak railyard. NIOSH recommendations to prevent contamination of workers' cars and homes included shoe covers, personal protective equipment, and leaving tools onsite.</td>
</tr>
<tr>
<td>Driscoll and Elliott [1990]</td>
<td>Chrysler Chemical Division, Trenton, Maine. Exposure to asbestos, solvents and lead. Found asbestos contamination of workers' clothing, autos, and on workers leaving for home. Recommended change facilities, showers, and company provision of work clothing.</td>
</tr>
<tr>
<td>Matte and Burr [1989]</td>
<td>Jamaican Ministry of Health, Kingston, Jamaica. Backyard battery repair shop - measured lead in houses and blood levels of family members. Found significant contamination and recommended that there should be separate entrances for shops and houses, that dust in workplaces be controlled, that work clothing, laundry facilities, and showers be provided.</td>
</tr>
<tr>
<td>Gittleman et al. [1991]</td>
<td>G.T. Jones Tire and Battery Distributing, Inc., Birmingham, Alabama. Lead exposure from battery recycling. Took samples from workers' autos. Found lead in autos, which was not consistent with reports that most of workforce showered before leaving and maintained good hand-washing hygiene. Observed that only 20% of workers showered before leaving work and 1/3 did not change work clothes at end of day. Recommended good personal hygiene (showers, clothing changes, hand-washing).</td>
</tr>
<tr>
<td>Venable et al. [1993]</td>
<td>Boston Edison Company, Boston, Massachusetts. Work in underground utility vaults. Wipe samples for lead from work surfaces, service vehicles, employee clothing, and hands. Recommended hand-washing procedures, showering, and changing into non-contaminated clothing at the end of day.</td>
</tr>
<tr>
<td>McCammon et al. [1991]</td>
<td>New England Lead Burning Company, Salt Lake City, Utah. Wipe samples showed contaminated clothing, shoes (which wore home) and lead in workers' cars. Opportunity for lead exposure probably increased by lack of showering and practice of wearing work clothes home. Made recommendation to prevent home contamination.</td>
</tr>
<tr>
<td>Donovan [1994a,b]</td>
<td>Kessler Studios, Loveland, Ohio. Home-based stained glass window studio. Did not appear that lead was migrating into house - results indicated that ventilation and hygiene practices employed by the artists minimize their exposure and the contamination of their house with lead.</td>
</tr>
<tr>
<td>Kominsky and Singal [1987]</td>
<td>Firefighters in Groveport, Ohio. PCB contamination. Recommended disposable protective clothing to ultimately reduce problems of decontaminating equipment.</td>
</tr>
<tr>
<td>Kominsky [1984c]</td>
<td>Fabric samples from protective clothing of 3 firefighters were analyzed for malathion and diazinon. Laundry decontamination procedures were recommended.</td>
</tr>
<tr>
<td>Kominsky [1984b]</td>
<td>PCB contaminated firefighters clothes - Decontamination procedures for clothing were insufficient.</td>
</tr>
<tr>
<td>Kominsky [1984a]</td>
<td>Measured PCB concentrations in firefighters' clothing worn at a fire. Clothing was replaced.</td>
</tr>
<tr>
<td>Orris and Kominsky [1984]</td>
<td>Maplewood, Minnesota. PCBs in high school. Made recommendation about laundering clothing to remove PCBs.</td>
</tr>
<tr>
<td>Kominsky [1987a]</td>
<td>Jacksonville, Florida. Firefighter suits contaminated with PCBs from fire at transformer oil reclamation facility. Recommendations made for decontaminating garments, based on study of two different laundry methods.</td>
</tr>
<tr>
<td>FEDERAL AGENCY</td>
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<tr>
<td>Other CDC Centers</td>
<td>A joint study by CDC, the Tennessee Department of Public Health, and the Memphis-Shelby County Health Department of a lead smelter in Memphis, Tennessee found workers' houses had significantly higher concentrations of lead dust than controls. Children's BLLs were significantly higher than control children's and correlated with the concentrations of lead in dust.</td>
</tr>
<tr>
<td>Baker et al. [1977]</td>
<td>PCBs in sewage sludge. Serum PCB levels were higher in worker's families than in other community residents.</td>
</tr>
<tr>
<td>Cannon et al. [1978]</td>
<td>Joint study by CDC and EPA, Virginia State Department of Health and NIOSH of Kepone poisoning in wives of kepone workers. It is stated that the kepone episode has stimulated the development of an active, OSHA-approved occupational safety and health plan and has stimulated the passage through the State Legislature of the Virginia Toxic Substances Information Act.</td>
</tr>
<tr>
<td>Dolcourt et al. [1981]</td>
<td>Joint study by CDC, North Carolina Department of Human Resources, and Cabarrus County Health Department of auto battery recycling. Two families in cottage industries had high BLLs.</td>
</tr>
<tr>
<td>Dolcourt et al. [1978]</td>
<td>Joint study by CDC and the Wake County Health Department of lead poisoning in children of battery workers. Dust samples were collected in homes and BLLs of children were determined. Carpeting, clothes, and closets showed especially large amounts of contamination. Took measurements to reduce workers' exposure and home contamination and to decontaminate homes. Molar and Mushak [1982] studied decontamination of these homes of in a study supported by NIEHS Grants.</td>
</tr>
<tr>
<td>Falk et al. [1981]</td>
<td>Girl with angiosarcoma was exposed to arsenic in soil, water, and dust on father's work clothes and boots. NCI and NIEHS were also involved in this study.</td>
</tr>
<tr>
<td>Kaye et al. [1987]</td>
<td>Study of family members of workers exposed to lead. Children and family members had elevated BLLs compared to a non-exposed group. Lead levels from dust samples were significantly higher in workers' homes.</td>
</tr>
<tr>
<td>Landrigan [1976]</td>
<td>In this review of lead exposure in children, workers' home contamination is cited as a source of exposure.</td>
</tr>
<tr>
<td>Landrigan and Baker [1981]</td>
<td>In a study of children exposed to heavy metals from smelters, a relation between house dust levels and BLLs of children was found in El Paso, Texas.</td>
</tr>
<tr>
<td>Matte et al. [1991]</td>
<td>Several Centers and NIOSH studied household dust and soil around houses and BLL contamination in households near both conventional and cottage lead smelters. Significant increase in BLLs in children and residents near cottage smelters were found.</td>
</tr>
<tr>
<td>Matte et al. [1989]</td>
<td>Study of battery repair shops in Jamaica. Also reported in [CDC 1989a].</td>
</tr>
<tr>
<td>Novotny et al. [1987]</td>
<td>Joint study by CDC and the Colorado Department of Health of employees of a firing range and their wives.</td>
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<tr>
<td>Watson et al. [1978]</td>
<td>This joint study by CDC, EPA, and the Vermont State Health Department found elevated BLLs of children of workers manufacturing lead storage batteries. Even though workers showered and changed clothes at work, clothes were washed at home.</td>
</tr>
<tr>
<td>Wolfe et al. [1961]</td>
<td>Recommended that if discarded pesticide drums can't be destroyed, they should be rinsed with water at least twice, so as to be less likely to have toxic residues that can affect children. Also made recommendations for decontamination of pesticide applicators' clothing.</td>
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<td>FEDERAL AGENCY</td>
<td>RESPONSIBILITY</td>
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<tr>
<td>U.S. Air Force</td>
<td>Non-occupational Beryllium poisoning. Measured concentrations of beryllium generated during laundry, folding of clothes, etc.</td>
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<tr>
<td>U.S. Atomic Energy Commission</td>
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<tr>
<td>Eisenbud et al. [1949]</td>
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<tr>
<td>U.S. Atomic Energy Commission</td>
<td>Epidemiology of Beryllium intoxication. The literature on concentrations of beryllium related to development of diseases was reviewed, and related to &quot;hygiene&quot; measures. Exposure levels for workers, and for air in vicinity of beryllium plant and recommended laundering of workers' clothing to prevent contamination of workers' homes.</td>
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<td>Sterner &amp; Eisenbud [1951]</td>
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<td>STATE</td>
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<td>Alaska</td>
<td>Department of Environmental Conservation</td>
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<td>Arizona</td>
<td>Department of Health</td>
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<td>California</td>
<td>Department of Health Services</td>
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<td>Environmental Protection Agency</td>
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<td>Connecticut</td>
<td>Department of Public Health and Addiction Services</td>
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<td>Indiana</td>
<td>Department of Health</td>
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<td>Department of Labor</td>
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<td>Iowa</td>
<td>Department of Employment Services</td>
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<td>Kentucky</td>
<td>Department of Health Services</td>
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<td>Labor Cabinet</td>
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<td>Maryland</td>
<td>Department of the Environment</td>
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<td>Michigan</td>
<td>Department of Natural Resources</td>
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<td>New York</td>
<td>Department of Environmental Conservation</td>
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<td>Oregon</td>
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<td>Puerto Rico</td>
<td>Department of Labor and Human Resources</td>
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<td>Utah</td>
<td>Department of Health</td>
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<td>Virginia</td>
<td>Department of Labor and Industry</td>
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<td>Department of Health</td>
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<td>Wyoming</td>
<td>Department of Health</td>
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Table 21. How State Agencies Respond to Incidents of Workers' Home Contamination – States that Do Not Have Their Own OSHA Program

<table>
<thead>
<tr>
<th>STATE</th>
<th>AGENCY</th>
<th>INFORMATION PROVIDED TO NIOSH</th>
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<tbody>
<tr>
<td>Alabama</td>
<td>Department of Health</td>
<td>The Department of Health submitted two case histories where site visits found increased BLLs, a final report to ATSDR on lead exposure of children, and a report on tire and battery plants conducted by the regional OSHA office [Williamson 1994; State of Alabama 1992; Mangum 1994; Alabama Department of Health 1991].</td>
</tr>
<tr>
<td>Arkansas</td>
<td>University of Arkansas College of Agriculture and Home Economics and Cooperative extension Service</td>
<td>The College reported that it has no reports of take-home toxins, and that the States worker protection standard applies to agriculture. The Department submitted reports to NIOSH on educational literature with segments addressing home contamination from workers [Huitink 1994; Lavy 1994].</td>
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<tr>
<td>Delaware</td>
<td>Department of Natural Resources and Environmental Control</td>
<td>The Department reported that it had no information on any take-home cases [Mohrman 1994].</td>
</tr>
<tr>
<td>Florida</td>
<td>Department of Labor and Employment Security</td>
<td>The Department of Labor reported that it has no mechanism in place to monitor worker transportation of chemicals, etc. [Koehler 1994]. The Department of Agriculture has published a brochure in English and Spanish on washing clothing contaminated by pesticides which was provided to NIOSH [Anonymous 1994].</td>
</tr>
<tr>
<td>Idaho</td>
<td>Department of Health</td>
<td>The Department of Health reported that it has no jurisdiction over private sector or other agencies. It does have a health and safety program for its own employees with specific procedures involving contamination and written procedures to comply with OSHA for lab safety and hazardous waste. It has practices and procedures in place to make sure decontamination occurs at the workplace. It has an elevated blood lead registry and conducts follow-up studies on children that are reported. Employers of adults reported to the registry are identified to see if there is an occupationally related cause of exposure [Schultz 1994; Stokes 1994].</td>
</tr>
<tr>
<td>Louisiana</td>
<td></td>
<td>No responses to requests for information were received; however, the Agriculture Experiment Station provided reports on pesticide workers' clothing contamination and laundering when contacted by telephone [Finley et al. 1977; no date].</td>
</tr>
<tr>
<td>Maine</td>
<td>Department of Environmental Protection</td>
<td>The Department of Environment reported that it has an emergency response group that responds to oil and hazardous chemical incidents. The group follows decontamination procedures and has decontamination areas for workers. All the workers are in a health monitoring program [Marriott 1994].</td>
</tr>
<tr>
<td>Mississippi</td>
<td>State Department of Health</td>
<td>The Department reported on an investigation of two cases of lead poisoning [Pollock 1994].</td>
</tr>
<tr>
<td>Montana</td>
<td>Department of Health and Environmental Sciences</td>
<td>The Department reported that it has no system of tracking home-contamination cases, any complaints of hazardous sites are referred to county health officials [Cleverly 1994].</td>
</tr>
<tr>
<td>Nebraska</td>
<td>Department of Labor</td>
<td>The Department of Labor reported that it has no statutes or regulations about home contamination. It uses applicable OSHA standards [Calcaterra 1994]. The Agriculture Experiment has been active in studying laundering of pesticide workers' clothing and informing farm families of appropriate laundering procedures [Laughlin and Gold 1988, 1989].</td>
</tr>
<tr>
<td>New Jersey</td>
<td>Department of Labor</td>
<td>Federal Occupational Safety and Health Standards are applied to public sector employees [Katz 1994]</td>
</tr>
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<td></td>
<td>Department of Health</td>
<td>The Department of Health submitted a report on a pilot project on exposure of children to take-home lead [Stanbury 1994; Czachur et al. 1995].</td>
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<tr>
<td>STATE</td>
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<tr>
<td>Oklahoma</td>
<td>Department of Health and Department of Environmental Quality</td>
<td>These Departments reported that they have no data or regulations on home contamination [Coleman 1994].</td>
</tr>
<tr>
<td>Pennsylvania</td>
<td>Department of Labor and Industry</td>
<td>The Department of Labor reported that it collects information on hazardous chemicals in the workplace from employers. It provided NIOSH with copies of the State's: Employer/Worker Community Right to Know Act; List of chemicals subject to reporting (EPA); Manual for employer compliance with Hazardous Materials Act [Tinney 1994].</td>
</tr>
<tr>
<td>South Dakota</td>
<td>Department of Health</td>
<td>The Department of Health reported that it has been conducting residential indoor air quality studies since 1990, and has initiated a simple data collection system for surveillance of hazards in the home; however, it has no reports of take-home cases [Forsch 1994].</td>
</tr>
<tr>
<td>West Virginia</td>
<td>Department of Health and Human Resources</td>
<td>The Department of Health reported that it has no reports or data on workers' home contamination and submitted the State's Asbestos Licensure Law [Wallace 1994; Pinnell 1994].</td>
</tr>
<tr>
<td>Wisconsin</td>
<td>Department of Natural Resources (DNR)</td>
<td>The Department of Natural Resources reported that it has no evidence of wastewater staff taking home contaminants. There is a possibility of home contamination by forest firefighters, since the firefighters wash their clothing at home. DNR does not systematically collect information on home contamination; it focuses on environmental protection, rather than public or environmental health [Kavanaugh 1994].</td>
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<td>STATE AGENCY</td>
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<tr>
<td>Alabama Department of Public Health</td>
<td>CDC [1992b]</td>
<td>1991 - Battery reclamation workers had elevated BLLs and BLLs of some of the workers' children were high. Workers had inadequate hygiene practices; wore work clothes home, didn't shower at work. Closed plant.</td>
</tr>
<tr>
<td>California</td>
<td>West and Lim [1968]</td>
<td>1968 - Mercury workers contaminated their homes with mercury from boots and work clothing.</td>
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<td></td>
<td>West [1959]</td>
<td>A child whose father was a crop sprayer was poisoned when his father wore contaminated shoes at home.</td>
</tr>
<tr>
<td>Colorado Department of Health</td>
<td>Cook et al. [1993]</td>
<td>1993 - Assessed BLLs of children living in mining and smelting communities. Found that the sources of exposure to lead were lead brought home on clothes and contaminated soil in yards.</td>
</tr>
<tr>
<td></td>
<td>CDC [1985; Kaye et al 1987]</td>
<td>Electrical component manufacturer. High BLLs in workers who wore work clothes home and exposed children, significantly higher BLLs in workers' children.</td>
</tr>
<tr>
<td></td>
<td>CDC [1989b]</td>
<td>Elevated BLLs in workers who manufactured lead belt buckles. Wives and children's BLLs were elevated. Noted the importance of getting occupational histories from patients admitted for treatment of lead poisoning.</td>
</tr>
<tr>
<td>Minnesota Department of Health</td>
<td>Winegar et al. [1977]</td>
<td>1977 - Lead smelter workers and families - BLLs and house dust concentrations of lead were measured. BLLs up to 44 mg were found. Also found elevated house dust lead levels in workers' homes and lead on workers and their clothing.</td>
</tr>
<tr>
<td></td>
<td>Lussenhop et al. [1989]</td>
<td>1989 - Some radiator repair workers had elevated BLLs. Screened 16 children - BLLs were normal.</td>
</tr>
<tr>
<td>New Jersey Department of Health</td>
<td>Czauch [1995]</td>
<td>1994 - Conducted a pilot study on children of workers with elevated BLLs and found lead contaminated clothing to be a source of elevated BLLs in the children.</td>
</tr>
<tr>
<td>New York Department of Health</td>
<td>Nuñez et al. [1993]</td>
<td>1993 - Radiator repair - study of workers' children and workers (67% of auto radiator repair workers in 89% of shops in city). None of children's BLLs were in excess of current guidelines.</td>
</tr>
<tr>
<td>North Carolina Wake County Department of Health</td>
<td>CDC [1977b]</td>
<td>1977 - Take-home lead from battery plant employees, 72% of children of plant employees had increased BLLs. High concentrations of lead were found in cars and in closets where shoes and work clothes were stored.</td>
</tr>
<tr>
<td>Oklahoma Department of Health</td>
<td>Morton et al. [1982]</td>
<td>1982 - Studies of children of workers in lead related industries - assessed exposure and measured BLLs and personal hygiene practices of workers. Only good personal hygiene before leaving work was effective for lead containment (showering, washing hair, changing clothes).</td>
</tr>
<tr>
<td>Pennsylvania Department of Health</td>
<td>Lieben and Williams [1969]</td>
<td>1969 - Respiratory diseases associated with beryllium refining in alloy fabrication - 95 workers and &quot;contact&quot; cases, some of whom were relatives of workers.</td>
</tr>
<tr>
<td>STATE AGENCY</td>
<td>REFERENCE</td>
<td>FINDINGS</td>
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<tr>
<td>Pennsylvania Department of Labor and Industry</td>
<td>Fulton and Matthews [1936]</td>
<td>1936 - Report on effects of exposure to naphthalene and chlorodiphenyl (dermatological and systemic effects) - case of dermatitis in child whose father had worked at plant and wore soiled clothes home. The father played with child without changing clothes. The wife also had dermatitis, as did an 11-month-old infant.</td>
</tr>
<tr>
<td>Tennessee Department of Public Health</td>
<td>CDC [1976]</td>
<td>1976 - Children of workers in secondary lead smelters. Children of workers had elevated BLLs, supposedly because of parents' contaminated clothing.</td>
</tr>
<tr>
<td>Vermont State Department of Health</td>
<td>Zirschy and Witherall [1987]</td>
<td>1987 - Study of mercury from clinical thermometer plant being carried home on workers' clothing. Found increased air mercury levels in some of workers' homes and increased urine mercury levels in some children's urine. Plant was closed and company was required to clean up workers' homes. Subsequently hired consultant, who developed protocol describing all cleaning procedures, disposition of waste, personnel to be used and procedures to determine the success of the decontamination. Paper describes contents of this protocol.</td>
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<tr>
<td></td>
<td>Watson et al. [1978]</td>
<td>Children of workers exposed to lead at a battery plant had high BLLs and their homes had elevated lead levels in house dust.</td>
</tr>
<tr>
<td>Virginia Commonwealth University</td>
<td>Garretson [1988]</td>
<td>1987 - This is a report of a study of a radiator mechanics' child with lead poisoning.</td>
</tr>
<tr>
<td>Source</td>
<td>Industry</td>
<td>Problem</td>
</tr>
<tr>
<td>-------------------------------------</td>
<td>-----------------------------------</td>
<td>-------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Barnett [1994]</td>
<td>Bronze foundry</td>
<td>Lead determined to be brought into workers homes on their clothing. Two children identified with elevated BLLs.</td>
</tr>
<tr>
<td>Gunter et al. [1987]</td>
<td>Assay laboratory</td>
<td>Workers were exposed to high lead levels and some had elevated BLLs.</td>
</tr>
<tr>
<td>Donovan [1994a,b; 1994b]</td>
<td>Stained glass</td>
<td>Work involves use of lead, residence attached to workplace.</td>
</tr>
<tr>
<td>Versen &amp; Bunn [1989]</td>
<td>Mining, processing, and packaging of diatomaceous earth</td>
<td>Possible exposure to silica when laundering workers' clothes.</td>
</tr>
<tr>
<td>Hudson et al. [1985, 1987]</td>
<td>Thermometer manufacture</td>
<td>Workers and children of workers had high levels of mercury in their blood</td>
</tr>
<tr>
<td>de Silva [1994]</td>
<td>Sandblasting</td>
<td>In a routine annual check-up, a pediatrician found an elevated BLL in a 2-year-old. The pediatrician determined that the child's father was working in a lead related occupation. The father's BLL was 35 μg/dL.</td>
</tr>
<tr>
<td>Lundquist [1980]</td>
<td>Battery manufacturing</td>
<td>Take-home lead contamination</td>
</tr>
<tr>
<td>Lead Industries Association [1989, 1991, 1993a,b; 1994a,b]</td>
<td>Multiple industries and hobbies, users of lead</td>
<td>Potential take-home contamination</td>
</tr>
</tbody>
</table>
Appendix I.
The Workers' Family Protection Act
29 U.S.C. 671a
Section 209
of the
Public Law 102-522
PUBLIC LAW 102-522 [H.R. 2042]: October 26, 1992

FIRE ADMINISTRATION AUTHORIZATION
ACT OF 1992

For Legislative History of Act, see p. 2910.

An Act to authorize appropriations for activities under the Federal Fire Prevention and Control Act of 1974, and for other purposes.

Be it enacted by the Senate and House of Representatives of the United States of America in Congress assembled,

SECTION 1. SHORT TITLE.

This Act may be cited as the "Fire Administration Authorization Act of 1992".

TITLE I—UNITED STATES FIRE ADMINISTRATION

SEC. 101. AUTHORIZATION OF APPROPRIATIONS.

Section 17(g)(1) of the Federal Fire Prevention and Control Act of 1974 (15 U.S.C. 2218(g)(1)) is amended—

(1) by striking "and" at the end of subparagraph (B);
(2) by striking the period at the end of subparagraph (C) and inserting in lieu thereof a semicolon; and
(3) by adding at the end the following new subparagraphs:
"(D) $25,550,000 for the fiscal year ending September 30, 1992;
"(E) $26,521,000 for the fiscal year ending September 30, 1993; and
"(F) $27,529,000 for the fiscal year ending September 30, 1994."

SEC. 102. PRIORITY ACTIVITIES OF THE UNITED STATES FIRE ADMINISTRATION.

(a) PRIORITY ACTIVITIES.—In expending funds appropriated pursuant to the amendments made by section 101 of this Act, the United States Fire Administration shall give priority to—

(1) reducing the incidence of residential fires, especially in residences of the very old, the very young, or the disabled in urban and rural areas, through the development and dissemination of public education and awareness programs, through arson research and technical assistance programs, and through research and development on new technologies;
(2) working with State Fire Marshals and other State level fire safety offices to identify fire problems that are national in scope;
(3) disseminating information about the activities and programs of the United States Fire Administration to State and local fire services;
(4) enhancing the residential sprinkler programs, including research, demonstration activities, and technical assistance to the public and private sectors; and
(5) enhancing research into sprinkler programs in areas or structures with limited or no domestic water supply;

106 STAT. 3410
SEC. 207. AUDITS, REPORT REQUIREMENTS, AND PETITION OF ATTORNEY GENERAL FOR EQUITABLE RELIEF.

(a) AUDITS.—For purposes of the Act entitled "An Act to provide for audit of accounts of private corporations established under Federal law", approved August 30, 1964 (36 U.S.C. 1101 et seq.), the Foundation shall be treated as a private corporation established under Federal law.

(b) REPORT.—The Foundation shall, within 4 months after the end of each fiscal year, prepare and submit to the appropriate committees of the Congress a report of the Foundation's proceedings and activities during such year, including a full and complete statement of its receipts, expenditures, and investments.

(c) RELIEF FOR CERTAIN FOUNDATION ACTS OR FAILURES TO ACT.—If the Foundation—
   (1) engages in, or threatens to engage in, any act, practice, or policy that is inconsistent with the purposes set forth in section 202(b); or
   (2) refuses, fails, or neglects to discharge its obligations under this title, or threatens to do so,
the Attorney General may petition in the United States District Court for the District of Columbia for such equitable relief as may be necessary or appropriate.

SEC. 208. IMMUNITY OF THE UNITED STATES.

The United States shall not be liable for any debts, defaults, acts, or omissions of the Foundation nor shall the full faith and credit of the United States extend to any obligation of the Foundation.

SEC. 209. WORKERS' FAMILY PROTECTION.

(a) SHORT TITLE.—This section may be cited as the "Workers' Family Protection Act".

(b) FINDINGS AND PURPOSES.—
   (1) FINDINGS.—Congress finds that—
      (A) hazardous chemicals and substances that can threaten the health and safety of workers are being transported out of industries on workers' clothing and persons;
      (B) these chemicals and substances have the potential to pose an additional threat to the health and welfare of workers and their families;
      (C) additional information is needed concerning issues related to employee transported contaminant releases; and
      (D) additional regulations may be needed to prevent future releases of this type.
   (2) PURPOSE.—It is the purpose of this section to—
      (A) increase understanding and awareness concerning the extent and possible health impacts of the problems and incidents described in paragraph (1);
      (B) prevent or mitigate future incidents of home contamination that could adversely affect the health and safety of workers and their families;
      (C) clarify regulatory authority for preventing and responding to such incidents; and
      (D) assist workers in redressing and responding to such incidents when they occur.
   (c) EVALUATION OF EMPLOYEE TRANSPORTED CONTAMINANT RELEASES.—

106 STAT. 3420
(I) Study.—

(A) In General.—Not later than 18 months after the date of enactment of this Act, the Director of the National Institute for Occupational Safety and Health (hereafter in this section referred to as the "Director"), in cooperation with the Secretary of Labor, the Administrator of the Environmental Protection Agency, the Administrator of the Agency for Toxic Substances and Disease Registry, and the heads of other Federal Government agencies as determined to be appropriate by the Director, shall conduct a study to evaluate the potential for, the prevalence of, and the issues related to the contamination of workers' homes with hazardous chemicals and substances, including infectious agents, transported from the workplaces of such workers.

(B) Matters to Be Evaluated.—In conducting the study and evaluation under subparagraph (A), the Director shall—

(i) conduct a review of past incidents of home contamination through the utilization of literature and of records concerning past investigations and enforcement actions undertaken by—

(I) the National Institute for Occupational Safety and Health;

(II) the Secretary of Labor to enforce the Occupational Safety and Health Act of 1970 (29 U.S.C. 651 et seq.);

(III) States to enforce occupational safety and health standards in accordance with section 18 of such Act (29 U.S.C. 667); and

(IV) other government agencies (including the Department of Energy and the Environmental Protection Agency), as the Director may determine to be appropriate;

(ii) evaluate current statutory, regulatory, and voluntary industrial hygiene or other measures used by small, medium and large employers to prevent or remediate home contamination;

(iii) compile a summary of the existing research and case histories conducted on incidents of employee transported contaminant releases, including—

(I) the effectiveness of workplace housekeeping practices and personal protective equipment in preventing such incidents;

(II) the health effects, if any, of the resulting exposure on workers and their families;

(III) the effectiveness of normal house cleaning and laundry procedures for removing hazardous materials and agents from workers' homes and personal clothing;

(IV) indoor air quality, as the research concerning such pertains to the fate of chemicals transported from a workplace into the home environment; and

(V) methods for differentiating exposure health effects and relative risks associated with specific

106 Stat. 3421
agents from other sources of exposure inside and outside the home;
(iv) identify the role of Federal and State agencies in responding to incidents of home contamination;
(v) prepare and submit to the Task Force established under paragraph (2) and to the appropriate committees of Congress, a report concerning the results of the matters studied or evaluated under clauses (i) through (iv); and
(vi) study home contamination incidents and issues and worker and family protection policies and practices related to the special circumstances of firefighters and prepare and submit to the appropriate committees of Congress a report concerning the findings with respect to such study.

Establishment.

(2) DEVELOPMENT OF INVESTIGATIVE STRATEGY.—
(A) TASK FORCE.—Not later than 12 months after the date of enactment of this Act, the Director shall establish a working group, to be known as the "Workers' Family Protection Task Force". The Task Force shall—
(i) be composed of not more than 15 individuals to be appointed by the Director from among individuals who are representative of workers, industry, scientists, industrial hygienists, the National Research Council, and government agencies, except that not more than one such individual shall be from each appropriate government agency and the number of individuals appointed to represent industry and workers shall be equal in number;
(ii) review the report submitted under paragraph (1)(B)(v);
(iii) determine, with respect to such report, the additional data needs, if any, and the need for additional evaluation of the scientific issues related to and the feasibility of developing such additional data; and
(iv) if additional data are determined by the Task Force to be needed, develop a recommended investigative strategy for use in obtaining such information.
(B) INVESTIGATIVE STRATEGY.—
(i) CONTENT.—The investigative strategy developed under subparagraph (A)(iv) shall identify data gaps that can and cannot be filled, assumptions and uncertainties associated with various components of such strategy, a timetable for the implementation of such strategy, and methodologies used to gather any required data.
(ii) PEER REVIEW.—The Director shall publish the proposed investigative strategy under subparagraph (A)(iv) for public comment and utilize other methods, including technical conferences or seminars, for the purpose of obtaining comments concerning the proposed strategy.
(iii) FINAL STRATEGY.—After the peer review and public comment is conducted under clause (ii), the Director, in consultation with the heads of other government agencies, shall propose a final strategy for investigating issues related to home contamination.
that shall be implemented by the National Institute
for Occupational Safety and Health and other Federal
agencies for the period of time necessary to enable
such agencies to obtain the information identified
under subparagraph (A)(ii).

(C) CONSTRUCTION.—Nothing in this section shall be
construed as precluding any government agency from
investigating issues related to home contamination using
existing procedures until such time as a final strategy
is developed or from taking actions in addition to those
proposed in the strategy after its completion.

(3) IMPLEMENTATION OF INVESTIGATIVE STRATEGY.—Upon
completion of the investigative strategy under subparagraph
(B)(iii), each Federal agency or department shall fulfill the
role assigned to it by the strategy.

(d) REGULATIONS.—

(1) IN GENERAL.—Not later than 4 years after the date
of enactment of this Act, and periodically thereafter, the Sec-
retary of Labor, based on the information developed under
subsection (c) and on other information available to the Sec-
retary, shall—

(A) determine if additional education about, emphasis
on, or enforcement of existing regulations or standards
is needed and will be sufficient, or if additional regulations
or standards are needed with regard to employee trans-
ported releases of hazardous materials; and

(B) prepare and submit to the appropriate committees
of Congress a report concerning the result of such
determination.

(2) ADDITIONAL REGULATIONS OR STANDARDS.—If the Sec-
retary of Labor determines that additional regulations or stand-
ards are needed under paragraph (1), the Secretary shall
promulgate, pursuant to the Secretary's authority under the
Occupational Safety and Health Act of 1970 (29 U.S.C. 651
et seq.), such regulations or standards as determined to be
appropriate not later than 3 years after such determination.
Appendix 2.
Request of Existing Information Relevant
to Implementing the Workers’ Family Protection Act

FR58:60202 - 60204

and

Notice to Readers, MMWR 42(48):943
GENERAL ACCOUNTING OFFICE

Federal Accounting Standards Advisory Board; Meeting Cancellation

AGENCY: General Accounting Office.
ACTION: Cancellation of November meeting.

SUMMARY: Pursuant to section 10(a)(2) of the Federal Advisory Committee Act (Pub. L. 92-463), as amended, notice is hereby given of cancellation of the November 18 meeting of the Federal Accounting Standards Advisory Board. Notice of the meeting was previously published in the Federal Register of November 8. It is currently anticipated that the December meeting will be held as scheduled. Due notice of it will be published at a later date in the Federal Register.

FOR FURTHER INFORMATION CONTACT: Ronald S. Young, Staff Director, 750 First Street NE., room 1001, Washington, DC 20002, or call (202) 512-7354.

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Centers for Disease Control and Prevention

Scientific Review of Draft Current Intelligence Bulletin on the Carcinogenic Potential of Occupational Exposure to Asphalt Products; Meeting

The National Institute for Occupational Safety and Health (NIOSH) of the Centers for Disease Control and Prevention (CDC) announces the following meeting.


Time and Dates: 9 a.m.—5:30 p.m., December 1, 1993; 8 a.m.—12 noon, December 2, 1993.

Place: Robert A. Taft Laboratories, Auditorium, NIOSH, CDC, 4676 Columbia Parkway, Cincinnati, Ohio 45226.

Purpose: The purpose of the meeting is to review the draft of the Current Intelligence Bulletin, "Carcinogenic Potential of Occupational Exposure to Asphalt Products," with a panel of invited participants selected by NIOSH for their expertise and background in this area. The scientific review will provide NIOSH with individual input and opinion from experts outside the Institute to prior the finalizing the Current Intelligence Bulletin for publication and transmittal to the Department of Labor. The review will focus on the health affects related to occupational exposures to asphalt products and on data from carcinogenicity studies in animals. Viewpoints and suggestions from industry, labor, academia, other government agencies, and the public are invited.

Contact persons for additional information: General Information may be obtained from Pam Graydon, NIOSH, CDC, 4676 Columbia Parkway, Mailstop C-30, Cincinnati, Ohio 45226, telephone 513/533-8312. Technical information may be obtained from Crystal Ellison NIOSH, CDC 4676 Columbia Parkway, Mailstop C-31, Cincinnati, Ohio 45226, telephone 513/533-8331.

Dated: November 8, 1993.
Elvis Hilyer, Associate Director for Policy Coordination, Centers for Disease Control and Prevention (CDC).

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institute for Occupational Safety and Health; Request for Existing Information Relevant to Implementing the Workers’ Family Protection Act

AGENCY: National Institute for Occupational Safety and Health (NIOSH), Centers for Disease Control and Prevention (CDC), Public Health Service (PHS), Department of Health and Human Services (DHHS).

ACTION: Notice of request for existing information.

SUMMARY: NIOSH is requesting existing information on the contamination of workers’ homes by hazardous chemicals and substances transported from the workplace on equipment, clothing, or the worker’s person. This information would include existing reports of incidents resulting in familial poisonings or illnesses, methods of preventing and remediating such incidents, relevant statutes and regulations to prevent such incidents, and past investigations, enforcement actions, and the role of governmental agencies in preventing and responding to such incidents.

DATES: Information in response to this notice should be submitted by February 14, 1994.

ADDRESSES: Please submit two copies of any information to Diane Manning, Docket Office Manager, Division of Standards Development and Technology Transfer, NIOSH, 4676 Columbia Parkway, C-34, Cincinnati, Ohio 45226.

FOR FURTHER INFORMATION CONTACT: Dr. Steven Galson, Division of Standards Development and Technology Transfer, NIOSH, 4676 Columbia Parkway, C-14, Cincinnati, Ohio 45226. Telephone 513/533-8302.

SUPPLEMENTARY INFORMATION: The Workers’ Family Protection Act (29 U.S.C. 671a), hereafter referred to as “the Act,” was enacted on October 26, 1992, as section 209 of Public Law 102-522, the “Fire Administration Authorization Act of 1992.” The purpose of the Act is to protect the health of workers and their families from hazardous chemicals and substances, including infectious agents, transported from the workplace to the...
home on equipment, clothing, or the worker’s person. The specific objectives of the Act are the following:

1. To increase understanding and awareness of the extent and impact on health of hazardous chemicals and substances transported from the workplace to the home;

2. To prevent or mitigate future incidents of home contamination that could adversely affect the health and safety of workers and their families;

3. To clarify regulatory authority for preventing and responding to incidents of home contamination;

4. To assist workers in redressing and responding to incidents.

Under the Act, NIOSH is mandated to conduct a study to evaluate the problem of contamination of workers’ homes by hazardous chemicals and substances transported from the workplace. The study is to include review of past incidents of home contamination, and consultation by governmental agencies in response to such incidents, the roles and practices of governmental agencies and NIOSH study, and an analysis of relevant statutes, regulations, and voluntary measures. In addition to requesting existing information on these matters, NIOSH is also requesting existing information on incidents of home contamination and family illness or poisoning in situations where the workplace and home located together, such as farms and certain small businesses. NIOSH is requesting both published reports, including studies, case histories, voluntary guidance, standards, and regulations, and unpublished reports including accounts from physicians, poison control centers, industry management, labor unions, and other parties. Existing information is specifically requested on the following:

1. Measurements of home contamination or incidents of familial poisoning or illness due to contamination of the home by hazardous chemicals or substances transported to the home from the workplace on the equipment (including vehicles), clothing, or a worker’s person.

2. Any measurements of home contamination or incidents of familial poisoning or illness resulting from hazardous chemicals or substances due to proximity of the workplace and to the home, such as farms or other businesses with attached living quarters.

3. Reports of Federal, state or local government actions to either enforce statutes or regulations or provide assistance in incidents of familial poisoning or illness due to hazardous chemicals or substances transported from the workplace, or due to the proximate nature of the home and workplace.

4. Measures used by employers to prevent or remedy home contamination, including statutory, regulatory, or voluntary industrial hygiene measures. (Please specify the approximate number of workers employed by the business establishment.)

5. Effectiveness of industrial hygiene practices in the workplace, such as housekeeping practices and the use of personal protective equipment, in preventing home contamination.

6. Effective... of normal house cleaning and laundry procedures in removing hazardous materials and agents from workers’ homes, personal clothing and equipment (including vehicles).

7. Information on indoor air quality research that pertains to the fate of chemicals transported from a workplace into the home environment.

8. Information on any of the above items that pertains specifically to firefighters.

NIOSH presently has copies of the references listed at the end of this request for existing information.

Information received in response to this notice (except that designated trade secret and protected under Section 15 of the Occupational Safety and Health Act of 1970 [29 U.S.C. 664], or that exempt from disclosure under the Freedom of Information Act (5 U.S.C. 552) or the Privacy Act (5 U.S.C. 552a)) will be available for public examination and copying at the above address.

Dated: November 5, 1993.

Diane D. Porter,
Acting Director, National Institute of Occupational Safety and Health, Centers for Disease Control and Prevention (CDC).

References


Flood-Related Mortality — Continued

or swiftly moving flood waters. In this report, 75% (27.36) of the drownings that occurred during the summer and fall floods in Missouri were motor-vehicle–related.

The findings in this report underscore the importance of two strategies for preventing flood-related injuries and death. First, information about flood and post-flood hazards must be disseminated rapidly and widely to groups at increased risk for injury. For example, motorists should be warned not to drive through areas inundated by flash floods, not to enter swiftly moving water, and that only 2 feet of water can carry away most automobiles (7). In addition, recreational activities, such as wading or bicycling, in flooded areas should be discouraged. Second, hydrologic studies and hazard analyses should address potentially flood-prone tributaries. The hazard potential of such areas during flash floods should be identified, and appropriate warning signs should be posted. MDH is continuing surveillance of flood-related mortality to monitor circumstances of death.

References

Notice to Readers

Workers' Family Protection Act

On November 15, 1993, CDC's National Institute for Occupational Safety and Health (NIOSH) published in the Federal Register a request for existing information relevant to implementing the Workers' Family Protection Act1. NIOSH is requesting information on incidents of family poisonings or home contaminations by substances inadvertently carried home by workers on their clothing, equipment, or person and on regulations and methods for dealing with such incidents. Copies of the Federal Register announcement are available from the Docket Office Manager, Division of Standards Development and Technology Transfer (DSDTT), NIOSH; telephone (513) 533-8304. Additional information is available from the Deputy Director, DSDTT, NIOSH; telephone (513) 533-8302.

*58 FR 60202-60204.
Appendix 3.
Letters Requesting Information
January 10, 1994

Dear Sir/Madam:

The National Institute for Occupational Safety and Health (NIOSH) has recently published a Federal Register (FR) Notice (Enclosure 1) that requests information relevant to implementation of the Workers' Family Protection Act (29 USC 671a). The Act charges NIOSH with conducting a study of hazardous chemicals and substances, including infectious agents, that are carried home by the worker. The FR Notice describes the NIOSH responsibilities under the Act, including the development of a report that will describe the extent of the home contamination problem, the efficacy of actions taken to prevent home contamination, and the role of government agencies in responding to incidences of home contamination.

A previous FR Notice (Enclosure 2) was published on September 22, 1993, by NIOSH that solicits nominations for membership on the Workers' Family Protection Task Force. This task force, which is mandated by the Act, will review the report prepared by NIOSH and determine if additional research data are needed to fully address the problem of home contamination. If additional data are needed, the task force will develop a strategy to obtain this information.

To help prepare this report, please respond to the appropriate questions in Enclosure 1 and provide to NIOSH information (including laws or regulations), reports, or data on the contamination of workers' homes that are relevant to your agency or office. Please send these comments by February 14, 1994 to Ms. Diane Manning (NIOSH Mail Stop C-15). If you have any questions on this request, please call Mr. John Whalen at 513/533-8306.

Sincerely yours,

[Signature]

Richard W. Niemeier, Ph.D.
Director
Division of Standards Development and Technology Transfer

Enclosures
January 10, 1994

Dear Sir/Madam:

The National Institute for Occupational Safety and Health (NIOSH) has recently published a Federal Register (FR) Notice (see enclosed) requesting information relevant to implementation of the Workers' Family Protection Act (29 USC 671a).

The Act charges NIOSH with conducting a study of hazardous chemicals and substances, including infectious agents, that are carried home by the worker. The FR Notice describes the NIOSH responsibilities under the Act, including the development of a report that will describe the extent of the home contamination problem, the efficacy of actions taken to prevent home contamination, and the role of government agencies in responding to incidences of home contamination.

A task force mandated by the Act will review the report prepared by NIOSH and determine if additional research data are needed to fully address the problem of home contamination. If additional data are needed, the task force will develop a strategy to obtain this information.

Since your organization may have access to information on take-home toxins, we would greatly appreciate your assistance in obtaining information for the NIOSH report. The types of information needed are described in the enclosure and include clinical and legal case reports, laws or regulations, and any other relevant data. We would also appreciate your sharing this request with other interested agencies and groups. Information should be sent by February 14, 1994 to Ms. Diane Manning (NIOSH Mail Stop C-34). If you have any questions, please call Mr. John Whalen, at 513/533-8306.

Sincerely yours,

Richard W. Niemeier, Ph.D.
Director
Division of Standards Development
and Technology Transfer
Dear Sir/Madam:

The National Institute for Occupational Safety and Health (NIOSH) has recently published a Federal Register (FR) Notice (see enclosed) requesting information relevant to implementation of the Workers' Family Protection Act (29 USC 671a).

The Act charges NIOSH with conducting a study of hazardous chemicals and substances, including infectious agents, that are carried home by the worker. The FR Notice describes the NIOSH responsibilities under the Act, including the development of a report that will describe the extent of the home contamination problem, the efficacy of actions taken to prevent home contamination, and the role of government agencies in responding to incidences of home contamination.

Since your organization may have access to information on take-home toxins, we would greatly appreciate your assistance in obtaining information for the NIOSH report. The types of information needed are described in the enclosure. I am requesting your assistance especially in regard to relevant state or local laws, any actions that may have been taken, or could be taken by your Agency in cases of home contamination, and methods and procedures used for decontamination and prevention of future incidents. Information should be sent by February 14, 1994 to Ms. Diane Manning (NIOSH Mail Stop C-34). If you have any questions, please call Mr. John Whalen, at 513/533-8306.

Sincerely yours,

Richard W. Niemeier, Ph.D.
Director
Division of Standards Development and Technology Transfer
Appendix 4.
Acknowledgements
ACKNOWLEDGEMENTS

The following individuals from the Centers for Disease Control and Prevention (CDC) participated in the study of home contamination and the preparation of this report.

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Appendix 5.
Glossary
GLOSSARY

acetylcholinesterase - an enzyme that catalyses the hydrolysis of acetylcholine to choline and acetic acid.

angiosarcoma - a malignant tumor that originates from blood vessel elements in muscle and other soft tissue.

allergen - a substance, often a protein, that stimulates cellular responses in the body resulting in allergic symptoms.

allergy - hypersensitivity of the body cells to a specific substance (allergen) that results in various types of allergic reaction.

anthrax - a disease from infection with the bacteria Bacillus anthracis.

arthropod vectors - arthropods (e.g., ticks, fleas, and mosquitoes) may transmit diseases to humans. For example, Lyme disease may be transmitted through a bite from a tick and malaria is transmitted to humans from mosquito bites.

asthma - a disease marked by shortness of breath and trouble breathing due to reversible constriction of the bronchial tubes of the lung. It is often related to allergic conditions.

asthmagen - a substance which triggers asthma attacks.

asbestosis - a fibrotic disease of the lungs caused by asbestos fibers, which results in reduced lung volumes and difficulty in breathing.

berylliosis - a granulomatous disease of the lung related to inhalation of beryllium.

bronchoalveolar lavage - the washing out of the lung by multiple injections and removals of fluid.

blood lead level - the concentration of lead in the blood determined by laboratory methods.

chlamydia - small organisms that, like viruses, grow within host cells. However, their structure is similar to bacteria. Chlamydia cause a number of human diseases including trachoma (a scarring eye disease) and psittacosis (a pneumonia transmitted from birds to humans).

chloracne - an eruption of the skin resembling acne and resulting from exposure to chlorine or its compounds.

conjunctiva - the mucous membrane covering the anterior (front) surface of the eyeball and lining the lids.

coccidiomycosis - a systemic mycotic disease caused by the fungus Coccidioides immitis.
GLOSSARY

cyanotic - relating to the dark bluish or purplish coloration of the skin and mucous membranes due to deficient oxygenation of the blood in the lungs or to an abnormally great reduction of the blood in its passage through the capillaries.

droplet nuclei - droplets that contain infectious particles and are made by the evaporation of fluid from the droplets formed during the production of aerosols. Droplet nuclei may remain suspended in the air for long periods of time and are associated with respiratory diseases.

dysphagia - difficulty swallowing.

encephalitis - inflammation of the brain.

encephalopathic - relating to any disease of the brain.

esophageal perforation - a hole in the portion of the digestive tract between the throat and the stomach.

febrile disease - a disease with a fever component.

fibrosis - formation of fibrous tissue, as a reactive or reparative process in the body.

fomites - inanimate objects that may be contaminated with infectious organisms and serve in their transmission.

fungi - molds and yeasts - they have characteristics of both plants and animals and may cause diseases (i.e., mycoses, mycotoxicoses, and allergies).

gynecomastia - excessive development of the breast in a male.

giardiasis - an intestinal infection with the protozoan Giardia lamblia.

hepatomegaly - enlargement of the liver.

imbecile - mentally deficient.

lymphoblastic - relating to a young, immature cell that is destined to mature into a lymphocyte white blood cell.

mental retardation - an intellectual deficit that causes incompetence in the performance of social roles.

metastasis - in cancer, the appearance of neoplasms (tumors) in parts of the body remote from the seat of the primary tumor.

mesothelioma - a tumor derived from the cells of the pleural or peritoneal membranes.

moribund - dying, at the point of death.
mucous membranes - membranes that line the passages and cavities of the body which communicate directly or indirectly with the exterior.

mycoplasma - small organisms that grow within host cells. They are similar to bacteria but their cell walls are less complex. Mycoplasma are often associated with a relatively mild type of pneumonia.

necropsy - autopsy; an examination of the organs of a dead body for the purpose of determining the cause of death or of studying the pathologic changes present.

nephropathy - any disease of the kidney.

paralysis - loss of power of voluntary movement in a muscle through injury or disease of its nerve supply.

parasites - an organism that lives on a different organism without contributing anything to the survival of the host. Human parasites may be classified as protozoa, helminths (worms), mites, and lice.

parenchymal - relating to the specific tissue of a gland or organ, contained in and supported by the connective tissue framework, or stroma.

parturition - the act of giving birth.

pathogen - any virus, microorganism, or other substance causing a disease.

pericardial - surrounding the heart; relating to the membrane surrounding the heart (pericardium).

peritoneal - the membrane lining the abdominal cavity and covering most of the organs contained in the abdominal cavity.

pharyngitis - inflammation of the throat (i.e. the area of the gastrointestinal track between the mouth and the esophagus).

pleura - the membrane surrounding the lungs and lining the walls of the thoracic (chest) cavity.

pleural plaques - a patch or small differentiated area on the pleura.

protozoa - normally found as a microscopic single celled organism. Also, grows in colonies and exhibits some features of lower level animals. Malaria and amebiasis are examples of parasitic diseases in humans caused by protozoa.

Q fever - a febrile disease due to infection with the rickettsiae Coxiella burnetii.

radiographic - pertaining to X-ray imaging.

rhinitis - inflammation of the nasal mucous membrane.
rickettsiae - small microorganisms that, like viruses, grow within host cells and may produce disease. Unlike viruses, these organisms, have a more complicated structure, multiply like bacteria, and are susceptible to antibiotics. Rickettsial diseases are frequently transmitted by bites from ticks or lice.

sarcoidosis - a multiple organ system disease of unknown cause which can involve the lymph nodes, lungs, skin, and eyes.

status epilepticus - a condition in which one major attack of epilepsy (i.e. seizures) succeeds another with little or no intermission.

transbronchial - across a bronchus (the breathing tubes of the lung).

viruses - the smallest organisms that produce disease. They grow within host cells and take over the metabolic functions of the host organism to reproduce themselves.

wrist drop (double wrist drop) - paralysis of the extensors of the wrist and fingers from a lesion of the nerve supply. Double wrist drop would mean both wrists are affected.

zoonotic diseases - a disease in man acquired from animals.