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# Workers' Home Contamination

## Report of a Study Conducted Under

## The Worker's Family Protection Act

## (29 U.S.C. 671a)

**DO NOT CITE OR QUOTE**


**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES**
**PUBLIC HEALTH SERVICE**
**CENTERS FOR DISEASE CONTROL AND PREVENTION**
**NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH**

PREFACE

In November 1993, when NIOSH published a request for existing information on employee transported home contamination in the Federal Register, only some 60 references to the published literature were in hand. Following an intensive search of the literature together with a substantial response to a public request for information, over 350 citations became available for this report.

Although the Legislative directive (Public Law 102-522, Section 209, the Worker' 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 only enacted on October 26, 1992, this is not a new problem. In 1905, W. Lehmann published an article in Germany in which he described a family in which the mother and young child of a worker with chloracne also developed the disease. Lehmann wrote ". . . the worker in whose family the diseases occurred was a notoriously unclean person who had repeatedly failed to follow the instructions and whose wife was known to be sloppy. Was it not possible that the worker who thought nothing of wearing his work clothing at home carried the substances in with his clothing? And could these substances not have been ingested during meals with unclean hands on objects that had come in contact with them?" 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 in 1936 from the Pennsylvania Department of Labor and Industry. In this case a child's father was exposed to

hexachloronaphthalene and chlorodiphenyl. The man returned home from work and played with the child in his soiled clothing. The father and son slept together, the father in his underwear. A younger sister was also developing chloracne.

An additional ten pre-1960 articles have been uncovered that identified beryllium, diethylstilbestrol, mercury vapor, toxaphene, and lead as hazards to the families of workers. These reports also call attention to the special hazards that exist where the home and workplace are closely associated such as farms and "cottage" industries.

Beginning with reports in 1965 by Kiviluoto and by Newhouse and Thompson of asbestosis and mesothelioma among family members of asbestos workers, a substantial number of such cases have been, and continue to be identified worldwide. A particular hazard to wives of asbestos workers was laundering the contaminated clothing.

Lead poisoning in a family engaged in recovery of gold from jewelry in a "cottage" type operation was reported from India in 1971 that demonstrated the hazards to children playing in the work area. There followed during the 1970s an additional 15 citations to contamination of the homes of lead workers, and such reports continue to appear in the current literature. During the past two years 15 new reports of contamination of the homes of lead workers have become available; nine of these were submitted to NIOSH in response to its request for current information and would not otherwise be known.

This report to Congress and to the Workers' Family Protection Task force summarizes the incidents of employee transported home contamination that this study has discovered including the health consequences, the sources, and the level of contamination. The report contains information on the effectiveness of preventative measures and of cleaning and decontamination procedures that have been used or studied. Finally the report summarizes the laws and regulations that are relevant and responses of federal and state governmental agencies to incidents of employee transported 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 needless illnesses.

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# INTRODUCTION

Because of repeated reports of contamination of workers' homes in their states, identical bills, HR845 and S353, were introduced in 1991 by Mr. Ballenger (North Carolina) and Mr. Jeffries (Vermont) in the U.S. House of Representatives and the U.S. Senate, respectively. The Senate Subcommittee on Labor of the Committee on Labor and Human Resources held a hearing on S353 on July 26, 1991. Following the hearing, the Committee on Labor and Human Resources revised S353 and issued a report on November 27, recommending the revised bill to the Senate. 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. 671a.

The Workers' Family Protection Act requires the Director of the National Institute for Occupational Safety and Health (NIOSH) to conduct a study not later than 18 months after the date of its enactment. The study is to be conducted 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 Federal Government agencies the Director determines to be appropriate. The purpose of the study is 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 the workers. 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.

NIOSH is to prepare a report consisting of a summary of 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.
- The effectiveness of workplace housekeeping practices and personal protective equipment in preventing such incidents.
- The effectiveness of normal house cleaning and laundry procedures for removing hazardous materials and agents from workers' homes and personal clothing.
- Indoor air quality, as the research concerning such pertains to the fate of chemicals transported from a workplace into the home environment.
- Methods for differentiating exposure health effects and relative risks associated with specific agents from other sources of exposure inside and outside the home.

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. In general, studies that involved

reproductive effects related to the exposure of workers were excluded. Although these could in a broad sense be related to protection of the family, they are usually not related to contamination of the home by the worker. Some of the 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.

The report is arranged to address the issues identified in the Act. First, the studies relating to health effects are reviewed by substances. Details of the studies are presented in Tables and overviews of the findings for each contaminant are presented in the text. The second section is a review of the industrial hygiene aspects. The industrial hygiene data from the various studies was extracted into a table and the findings are summarized in the text for: (a) sources of contamination; (b) concentrations found in homes, automobiles and clothing; (c) the measures taken at the workplace that impact on or prevent contamination of the home and their effectiveness; and (d) procedures used and the effectiveness of home decontamination and (e) laundering.

In the final sections federal and state laws that are operative and the responses of federal and state agencies to incidents of home contamination are reviewed.

## METHODOLOGY

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 Federal Register Notice entitled "National Institute for Occupational Safety and Health: Request for Existing Information Relevant to Implementing the Workers' Family Protection Act," was published [Appendix 1]. 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 Mortality Weekly Report (MMWR) on December 10, 1994 [Appendix 2], by E-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.

Over 50 written and several telephone responses were received. (The mailing lists used and copies of written responses are available from the NIOSH Docket Office.) 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.

Key-word literature searches were conducted in various databases. 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.

By the end of February 1994, most of the responses to the various requests for information had been received and most identified published literature obtained. This information was distributed to the working group members for their review and compilation into the sections of this report.

## FINDINGS

### (1) Incidences of Take-Home Contamination - Health Effects

#### (a) Beryllium

Occupational lung disease from beryllium exposure has been recognized for over 50 years [Chamberlin et al. 1957; Hardy 1965; Hardy 1948; Hardy et al. 1967]. Chronic beryllium disease, an occupational granulomatous lung disorder, is still identified and it afflicts the small percentage of beryllium-exposed workers who develop beryllium-specific, cell-mediated immunity [Kreiss 1989; Newman 1989; Kreibel 1988].

Historically, non-occupational chronic beryllium disease has been described among residents living in the community surrounding a beryllium production plant [Eisenbud et al. 1949; Sterner and Eisenbud 1951; Chesner 1950] and among family members of beryllium workers who were presumably exposed to beryllium-contaminated clothing [Eisenbud et al. 1949; Sterner and Eisenbud 1951; Eisenbud and Lisson 1983].

Although chronic beryllium disease is generally described among workers in whom a history of past beryllium exposure is identified, 65 non-occupational cases were reported to the U.S. Beryllium Case Registry during the 1940s and 1950s [Eisenbud and Lisson 1983], these were recognized during a time when the beryllium industry did less to control environmental exposures. Twenty-three of these cases were attributed to household exposure to dust brought home on work clothes and 42 to ambient air pollution [Eisenbud and Lisson 1983]. It is noteworthy, that even with improvements in control of industrial exposure, occupationally related

beryllium disease continues to occur in an unchanged small percentage of exposed workers [Cullen 1986; Rossman 1988] and the actual number of current and former beryllium-exposed workers in the United States remains uncertain.

Until a 1992 report [Newman and Kreiss 1992], no household or air pollution cases had been reported in more than 30 years [Lieben 1959; Sussman 1959]. The relative disappearance of such cases has been attributed to improved control of air emissions and improved work practices such as mandatory work clothes exchange [Eisenbud and Lisson 1983]. Although it is also possible that household and community cases of beryllium disease may still be occurring but are unrecognized or misdiagnosed. Because chronic beryllium disease is readily confused with sarcoidosis [Sprince 1976], persons in the community with this disorder may be misclassified as having granulomatous lung disease of unknown etiology.

The correct diagnosis of chronic beryllium disease has implications both for individuals and for public health prevention activities [Cullen 1987; Coates 1983; Hasan and Homayoun 1974]. Individuals with granulomatous lung diseases should have a careful occupational and environmental history, and if a history of direct or indirect contact with beryllium is identified, additional testing should be considered [Kreiss 1989; Newman 1989]. Reports of non-occupational cases suggest that until more is known, even persons with seemingly minor, incidental beryllium exposure should be considered to be at risk [Bohne and Cohen 1985] and efforts to reduce exposures should continue.

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(b) Asbestos

Household contacts of asbestos-exposed workers are at increased risk of mesothelioma, lung cancer, and non-malignant parenchymal and pleural abnormalities. Three cohort studies (Table 1), six case-control studies (Table 2), and numerous case reports provide evidence of adverse effects in family members of asbestos workers.

Mesothelioma among household contacts of asbestos-exposed workers is a sentinel event for exposure from home contamination by asbestos [Gardner and Saracci 1989]. 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 by workers' inadvertent contamination of the home.

Most cases of mesothelioma among workers' family members occur in households where asbestos-contaminated work clothing were laundered at home. It is of interest to note that male children of asbestos workers appear to be at increased risk when compared with female children [Anderson 1979b; Kilburn et al. 1985, 1986].

Investigators from Mount Sinai School of Medicine, The City University of New York [Anderson 1983; Anderson et al. 1976, 1979a, 1979b; Joubert et al. 1991; Nicholson 1983; Nicholson et al. 1980] studied household

contacts of 1664 amosite asbestos workers who manufactured thermal insulation (Table 1). The prevalence of parenchymal and pleural abnormality twenty or more years after first household exposure was 48% among wives, 21% among daughters, 42% among sons, and 37% among siblings [Anderson 1979b].

Quantitative risk assessment for para-occupational exposure is difficult because fiber exposure measurements are not available. Nicholson [1983] has measured fiber levels in homes of asbestos miners and millers. 38.5% of 13 chrysotile samples taken between 1973-1976 from homes of asbestos workers exceeded 200 ng/$m^3$ (range 100-5000 ng/$m^3$).

Risk estimation is also difficult because most studies of para-occupational exposure are embedded within larger studies. Cases are individually matched to controls, but results from matched analysis are rarely reported for para-occupational exposure, making risk estimation problematic [Gardner and Saracci 1989].

The Mount Sinai [Anderson 1983; Anderson et al. 1976, 1979a, 1979b; Joubert et al. 1991; Nicholson 1983; Nicholson et al. 1980] investigators studied morbidity and mortality among a cohort of household contacts of amosite workers employed in a New Jersey asbestos insulation materials factory between 1941 and 1945.

Occupational, residential, smoking, and medical history 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 were at significantly increased risk of pleural abnormality (12% observed vs. 2% expected; $p<0.02$). Nicholson has documented increased asbestos fibers in dust from homes of former asbestos workers as long as 30 years after cessation of employment [Anderson et al. 1979].

Selikoff and colleagues 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 factory employees, Nicholson [1983] estimates that the expected number of mesothelioma deaths is 0.04, assuming an ambient air concentration of 200 ng/m$^3$.

The 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 1.7 (8 observed vs. 4.7 expected). Among males with 20+ years latency, there were 12 lung cancer deaths observed vs. 6.1 expected (SMR=1.97).

Magnani et al. [1993] conducted a retrospective cohort mortality study of 1964 wives of asbestos cement workers with no history of occupational exposure. Cancer of the pleura was significantly elevated, with an SMR of 792.3 (95% CI 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.

There was one cohort study by Navratil et al. [1972] of Czechoslovakia. There were 4 groups:

1. occupational
2. environmental
3. domestic (family members) >20 years old
4. general adult population in general area >40 years old

The first 3 groups all had hyaline pleural plaques much higher than expected compared to Group 4.

Several case-control studies (Table 2) have documented increased risk of respiratory disease among household contacts of asbestos workers [Newhouse and Thompson 1965; Whitwell et al. 1977]. Vianna and Polan [1978] conducted a matched case-control study of histologically

confirmed mesothelioma among New York State women. They report a relative risk of 10 (95% CI=1.4-37.4) for domestic exposure including hand-laundering work clothes. Results remain significant after elimination of occupationally exposed women from the analysis (p=0.02).

McDonald et al. [1970, 1973, 1980] have conducted several analysis on a population based series of North American autopsies. 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 para-occupational exposure histories were obtained from relatives. Women with mesothelioma were significantly more likely to have been exposed to asbestos dust on work clothes of household contacts (p=0.08).

There was one case control study by Ashcroft [1970] from Britain. Twenty-three patients with mesothelioma were matched on sex, age with 46 hospital controls, free of malignant disease. This study was primarily designed to show asbestos (primarily occupational) exposure was linked with mesothelioma. Pertinent to take home exposure, however, was that one patient with mesothelioma was a widow of an asbestos worker who was exposed for 3 years to the asbestos dust brought home on her husband's hair and shoes.

Kilburn et al. [1985, 1986] studied shipyard workers, most of whom had bystander (secondary) exposure to asbestos on the job. The prevalence

of radiographic evidence of asbestosis was 11% among wives, 8% among sons, and 2% among daughters of these shipyard workers [Table 3].

For the purpose of clarity and ease of presentation, the group of 41 papers on case histories has been divided into four subgroups: case reports (17 papers) (Table 4), case series (16 papers) (Table 5), health hazard evaluations (four papers) (Table 6) and miscellaneous reports (four papers) (Table 7). In addition, within each subgroup, the studies are listed in chronological order by year of publication. The oldest study is the study by Newhouse and Thompson of mesothelioma in London which was published in 1965, and is considered a classic paper in the field of take-home asbestos exposure.

In general, the problem of indirect exposure to industrial toxicants appears to be an international problem. Of the 33 papers that are epidemiology studies, 11 are from the United States, eight from Great Britain, five from Scandinavia, four from Germany, two from Canada, two from Italy, and one from Australia.

Areas of asbestos mining and manufacturing where the original exposures occurred include prospecting, mining, textiles, tile making, insulation workers, boiler makers, bakery oven building, shipbuilding and associated trades, certain railroad shop trades, welding rods, and asbestos products such as cord, seals, plates and other products. Secondary or indirect exposures included near-by workers not engaged directly with asbestos work, laundering of dusty work clothes by family

members, exposure to asbestos air pollution in vicinity of factory, living with workers in the industry, asbestos dust in home workshops, and other shops such as a pub located in a residence. There is also some evidence, which will be discussed later, of a genetic predisposition in some families, for mesotheliomas to develop.

The health effects of this indirect exposure seem universally to result in pleural or peritoneal mesotheliomas, pleural plaques, and/or asbestosis. Furthermore, these diseases seem to be specific to crocidolite asbestos exposure. Amphiboles, as a type of asbestos, are more important than chrysotile [Gibbs et al. 1989].

The first group of 17 papers (Table 4) are made up of case reports. These are generally detailed descriptions of one or more cases that have been, in this instance, subjected to take-home asbestos and have developed a specific health problem. These descriptions are limited to a maximum of six patients (in this group of 17 reports) and may be family members of the exposed or totally unrelated patients that a physician has seen. Thirteen of the seventeen reports reported mesothelioma as the end health result of this exposure. Age at death ranged from 31 in a non-occupationally exposed son [Champion 1971] to 74 in a mother who was married to a worker in a lumber and shingle mill [Krousel et al. 1986].

Looking at some specific and unusual indirect exposures, Epler et al. [1980] reported on two brothers who developed pleural changes in young

adulthood. It seems that in childhood that they had played in a room that was being used as an automobile muffler repair shop. Magee et al. [1986] reported on an individual that had developed a mesothelioma. He apparently had been exposed as a child in Corsica to tremolite asbestos in a room in his home that was being used as a local bar. The patrons of this bar were miners at the Carni asbestos mine and came into the bar in their dusty work clothes. Li et al. [1989] looked at 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 end result was the deaths of the mother, one sister and a young uncle that lived there of mesotheliomas. The father died of asbestosis.

Three case reports by Risberg et al. [1980], Martensson et al. [1984b], and Krousel et al. [1986] report that heredity may be important in cases where several family members develop mesotheliomas. There may possibly be a predisposing genetic factor that is involved. In a family made up of a sister, a brother, and two identical twin brothers, three are dead of mesotheliomas and the sister survives. The twins were directly exposed to asbestos for only a period of eight years [Martensson et al. 1984b].

Knappmann [1972] reported that in four cases of 251 cases of mesothelioma that he looked at in detail all tumors had metastasized, and that 70 percent had distant metastases. These cases all had long

latency periods.

The second group of 27 papers (Table 5) contain the case series studies. These are generally groups of unrelated (non-family) people who were seen and/or autopsied at large hospitals or clinics in large urban areas. Cases may include both direct and indirect exposures. The size of study ranges from 10 cases of indirect exposure [Gibbs et al. 1990] to a study of 133 cases of pleural plaques and 145 cases of asbestosis in Hamburg by Dalquen et al. 1970. The major diseases are again asbestosis, mesothelioma (pleural and peritoneal) and pleural plaques.

Three of the case series studies by Newhouse and Thompson 1965; Lieben and Pistawka 1967; and Dalquen et al. 1970 report that neighborhood exposure (defined as living within a half mile of an asbestos factory) may be an important source of indirect exposure to asbestos. Five studies report that laundering of dusty work clothes by wives or children of asbestos workers is an important source of indirect exposure [Ashcroft and Heppleston 1970; Dalquen et al. 1970; Edge and Choudhury 1978; Lander and Viskum 1985; and Konetzke et al. 1990]. Another possible source of indirect exposure is being a member of a family where one or more family members is an asbestos worker [Lieben and Pistawka 1967; Dalquen et al. 1970; Rubino et al. 1972; and Bianchi et al. 1982]. Two reports [Lieben et al. 1967, and Milne 1976] found mesothelioma patients with no known asbestos exposure.

Bittersohhl and Ose [1971] from Germany report on workers in two

chemical combines and a metal foundry that developed mesothelioma. These patients included workers that were not directly working with asbestos, but whose work stations were nearby to where asbestos work was being done in the factories.

The presence of asbestos bodies in indirectly exposed workers is considered important in these studies. The following case series studies report evidence and/or counts of asbestos bodies: [Newhouse et al. 1965; Ashcroft et al. 1970; Rubino et al. 1972; Milne 1976; Edge et al. 1978; Bianchi et al. 1982; and Gibbs et al. 1989.]

In a letter to the editor of THE LANCET, Browne [1991] pointed out that the end user of asbestos materials may be more severely compromised than the primary manufacturer. Handling the finished asbestos product may be more hazardous than manufacturing it. He reports that in a study of histologically confirmed cases of mesothelioma in south-east England that only 5.5% were among employees engaged in primary asbestos manufacture of asbestos products. All the rest of the cases were attributable to user industries.

Three papers are primarily concerned with methodology. Heller et al. [1970] did a radiological review of x-rays of 10 people diagnosed with malignant pleural mesothelioma. The purpose was to provide a technical discussion of evaluating x-rays for malignant pleural mesothelioma. Gibbs et al. [1989] did a study to correlate lung mineral count with the Zielhuis groupings, to determine whether any mesotheliomas were

unrelated to asbestos exposure, and to determine which form of asbestos fiber was most important as far as mesotheliomas were concerned. He determined that the Zielhuis method was too complex, that mesotheliomas develop in the absence of asbestos exposure, and that amphiboles are more important than chrysotile in respect to mesothelioma occurrence. Gibbs et al. [1990] also did a comparison study of types of lung fibers and size distribution in a series of indirect exposure cases of mesothelioma with a series of known occupational exposure in female gas mask makers. The gas mask workers showed consistently high crocidolite concentrations, while the indirect exposure cases showed mixed concentrations of various forms of asbestos fiber.

The third group of papers is composed of four health hazard evaluations (Table 6). These are basically small industrial hygiene studies of a particular building, manufacturing facility, mine, mill, quarry or other work place done by NIOSH. These surveys can be requested by a company, a union, or individual workers as a result of a perceived hazard. Results must be reported back within a short time span.

These four studies were done in a floor tile factory [Belanger et al. 1979], an automobile and truck brake shoe factory [Seixas and Ordin 1986], a U.S. Postal Service vehicle maintenance facility specializing in servicing and replacing brake shoes [Godby et al. 1987], and a friction products and adhesive manufacturing facility [Driscoll and Elliott 1990]. All facilities, with the exception of the postal facility which had brake drum service controls in place, had significant

environmental asbestos contamination. The postal facility was within NIOSH acceptable limits. The adhesive factory also had exposures to lead and solvents.

Recommendations to all four facilities included the use of company work clothes that would be provided, laundered, and kept at the company. Adequate decontamination and personal hygiene would be encouraged in order to prevent possible contaminants from being taken home on workers clothes or in their automobiles in the form of dust. Other recommendations to the three facilities that had hazard problems included issuing and enforcing the use of proper respirators, and the use of engineering controls where practical.

The final group of papers (Table 7) is called miscellaneous because of the diverse nature of the reports. This group is made up of a technical assistance report, a congressional asbestos oversight report, a report on the problems associated with asbestos removal, and an engineering report that gives in detail the proper sampling technique and proper housecleaning technique for five houses that suffered asbestos contamination from a toxic site.

Lemen [1972] observed the spraying of fireproof material on portions of a new research center being built in Cincinnati, Ohio. The hazard being investigated was asbestos exposure during spraying operations. The mixer-pump operator was found to have the highest exposure of the four men involved in the operation. This man was supplied with a respirator

which he did not use. Lemen's recommendations included enforced respirator use for all men, work supplied uniforms which are laundered by the company and not taken home, and to substitute some other fire retardant than asbestos in the fireproof mix.

The second paper in Table 7 is the minutes of an asbestos oversight hearing held by the Subcommittee on Elementary, Secondary, and Vocational Education of the Committee on Education and Labor, House of Representatives, of the Ninety-Sixth Congress, on H.R. 1435 and H.R. 1524, the purpose of these bills was "To establish a program for the inspection of schools for the presence of asbestos materials, to provide funds for the testing and evaluation of potential hazards, to create a loan program to assist in the containment or removal of imminent hazards to health and safety, and for other hazards." These minutes, among other things, contain a discussion of a published report by William J. Nicholson and others for NIEHS to describe the situation in New Jersey primary and secondary schools concerning asbestos in schools and suggestions on how to prevent further contamination.

Hinze and Hinze [1986] reported on the problems of asbestos removal. Asbestos has been known for over 2000 years, and its' use is widespread and worldwide. It has been considered a useful construction material. However, it is now considered a health hazard and as such consideration has been given to the removal of asbestos from buildings. Some of the problems include high cost of removal, changing and nonspecific regulations, various agencies claiming jurisdiction over the substance,

lack of information about asbestos-related diseases, and insurance-liability considerations.

The final paper in Table 7 concerns asbestos related housecleaning activities in five houses at a former lead-acid battery manufacturing site [Beegle and Forslund 1990]. Specific instructions are given for sampling of asbestos dust. Then detailed instructions are given for the decontamination of the houses. This paper concerns environmental cleanup of contaminated sites.

There were 3 review articles, covering the international literature. Rom et al. [1982] and Berry [1986] reviewed the association between asbestos exposure mesothelioma, primarily the occupational exposures. On domestic exposure, they reviewed many of the articles already discussed. Grandjean and Bach [1986] from Denmark did a literature review of effects of bystander exposure in general. They indicated that the effects of domestic exposure to asbestos stemmed from contaminated clothing worn or brought home.

**(c) Lead**

Over 60 reported incidents of take-home lead exposure were identified. Of these, there are 22 published retrospective cohort studies (Table 8), 14 published community screening studies (Table 9), and 28 case series or case reports, of which 10 are unpublished reports or letters (Table 10).

The cohort studies (Table 8) were designed such that households were selected on the basis of exposure (e.g., a lead worker lived in the household) and blood lead levels in the exposed group were compared to those of a comparison group of households where no one worked with lead. The community screening studies (Table 9) were designed to screen for elevated blood lead levels among residents near a lead industry, usually a lead smelter. Community studies were included in this review if investigators also compared blood lead levels in families of workers to those of other community members. The group of case reports and case series (Table 10) are reports of take-home lead incidents or assembled series of exposed family members without a comparison group.

Of the cohort and community screening 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 10) came from the U.S. and many of these were reported by state and federal agencies as a result of a 1993 Federal Register notice soliciting information on incidents of take-home lead exposure for this review.

The industries in which take-home lead exposure has been reported include: lead smelting, battery manufacturing/recycling, radiator repair, electrical components manufacturing, pottery/ceramics, and stained glass making. Take-home lead exposure 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 OSHA Lead Standard and was therefore relatively unregulated and understudied. A NIOSH study of take-home lead exposure among families of construction workers in New Jersey is currently underway.

The cohort studies (Table 8) 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 blood lead levels (BLLs) in excess 30 micrograms per deciliter ($\mu$g/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 six 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.

Morton et al. [1982], in a study of lead storage battery workers, showed 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 75% of workers' children had BLLs of 10 $\mu$g/dL or higher compared with 40% of control children.

The cohort studies also point to the issue of home-operated shops and cottage industries where work is conducted near or adjacent to the home, such as "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., employees in smaller industries, such as radiator shops are potentially at higher risk since OSHA regulations give inspection priority to businesses with ten or more employees.

In general, cohort studies have found that workers' children have significantly higher BLLs than control children. The BLLs for exposed children across all studies ranged from 10.2 to 81 $\mu$g/dL, while the BLLs for control children ranged from 6.2 to 27 $\mu$g/dL. Comparison groups were chosen from neighborhoods, community residents, birth registries, local schools and hospitals/clinics.

The community studies (Table 9) were included in the review if they addressed worker take-home exposure as part of the community investigation. The majority of the studies were conducted in smelter communities Of the 14 studies reviewed, all but three were conducted outside the U.S. Four studies were conducted in Germany. All the studies but one reported an association between child exposure to lead (as measured in blood, hair, or teeth) and parental occupation in a lead industry. A small study conducted by Landrigan and Baker [1981], that

included only 3 households with lead-exposed workers, reported no exposed children with BLLs over 40 μg/dL. However, It is difficult to conclude that this study is negative since today's guidelines indicate that the health effects of lead exposure may occur at levels as low as 10μg/dL.

The community study that reported the highest BLLs was conducted in a large smelter community in Brazil [Carvalho et al. 1984]. Children (age 1-9) of lead workers had a significantly higher mean BLL (67.5 μg/dL) than similarly aged children of non-lead workers (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). Parental occupation as a miner was an independent predictor of child's BLL.

The case report and case series collection of studies (Table 10), 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 own home [Dolcourt et al. 1981]. The highest BLLs among 22 family members were observed in a 3-year-old male and female (256 and 220 μg/dL, respectively using capillary sampling).

This series of reports undoubtedly represents 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 exposure. Ten of the 28 case reports (36%) are unpublished.

(d) Pesticides

There are nineteen reports in the scientific literature that describe situations in which pesticides from the workplace have the potential to cause adverse health effects in the home. Details of these reports are described in Table 11. Although the health effects are often presented in detail in the reports, information on how to differentiate between the health effects and relative risks from pesticides brought in from the workplace and pesticides used in the home is not always provided. Workplace procedures that contribute to the transmission of the chemical from the workplace to the home and housekeeping practices that could reduce home exposure are occasionally discussed. Information on pesticide contamination in the home is rarely available. Furthermore, several of the articles discuss the potential of transmission from the workplace to the home environment, without providing case histories.

Many of the reports in the literature describe incidents in which children were poisoned after exposure to residual pesticides left in discarded drums or other containers. In addition, children were often poisoned after they played with items that were contaminated with pesticides, or ingested pesticide from containers used to store or mix

pesticides. The majority of these cases involved children living on farms where pesticides were used. 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]. McGee et al. 1952, reported several unrelated poisonings by toxaphene, including one in which a 2 year old boy died after playing in a yard where strips of metal from flattened storage drums had been removed from a processing plant to cover the walls of a garden shed. 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.

The necessity of prudent handling of pesticides during transportation has been emphasized in several of the reports describing cases of pesticide poisoning in the home. For example, Anderson et al. [1965] describe a near-fatal incident of parathion poisoning in two friends, 5 and 12 years old boys, who became ill after sleeping on flannelette sheets that had been brought home by the father of one of the boys. The father was a salvage dealer who, in the line of business, bought the sheets as damaged goods from an insurance adjustor. The sheets were later discovered to have been contaminated with parathion during shipment in the hold of a ship that also contained drums of parathion. In a similar case, six boys became poisoned by wearing blue jeans that became contaminated with Phosdrin during transportation on a truck that also carried the insecticide [Warren et al. 1963]. Occupation of the

families was not discussed.

Three reports in the literature describe how poor hygiene practices in a chemical plant that produced the pesticide kepone lead to contamination of the homes of workers. Ninety-four percent of the family members that were examined had detectable levels of kepone in their blood, compared to nineteen percent 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.

Poisoning by exposure to residual parathion in clothing was described by Clifford and Nies 1989. Three workers in a plant that manufactured parathion were admitted to the hospital with symptoms of organophosphate poisoning. Although strict safety measures in the plant were enforced, a uniform contaminated with parathion that was designated to be burned was accidentally washed. It was later discovered that the workers who became ill had worn coveralls that had been washed along with this uniform. After it was determined that 5 additional sets of uniforms washed with the contaminated uniform contained from 135 to 1500 ppm parathion, all the uniforms in the plant were destroyed and replaced. Because of the practice of laundering the uniforms at the plant, no pesticide was transferred to clothes in the home laundry.

**(e) Mercury**

As summarized briefly in Table 12, there are nine reports on home

contamination by mercury. These cases occurred in a variety of settings, from mining operations to manufacturing plants. Home contamination has occurred when exposed workers carried mercury home in their automobiles and on clothes (including shoes). Automobiles and laundering facilities have been found to have the highest levels of mercury. In spite of these environmental exposures, blood levels of mercury in exposed families were not always elevated from control levels.

Another source of mercury contamination of homes involves home gold mining operations. Several reports suggest that families can be exposed during home use of mercury to extract gold from soil. In some cases, severe poisoning occurred. These cases are included because of their similarities to situations that may occur in cottage industries, and also because some of these may be subject to regulations of the Mine Safety and Health Administration.

West and Lim [1968] described exposures of mine workers to mercury during milling of cinnabar. The urine levels reached as high as 8640 μg/L and some workers had severe neurological symptoms of mercury exposure. The authors reported that some of the workers had unknowingly contaminated their living quarters with mercury from their boots and other work clothes. Data were not provided. Two additional reports provided information on home contamination from mining.

Zalesak [1994] reported mercury contamination of cars and homes of

workers employed by a California gold mine. In homes, the washers and dryers were the most heavily contaminated, with mercury levels ranging from 0.05 to 0.005 $mg/m^3$. Zalesak also reported that a technician from a second work facility wore contaminated clothing home. Traces of mercury were found in her car for several months after the exposures, but none was detected in the home.

Occupational exposure to mercury, followed by home contamination, was described by Ehrenberg et al. [1986; 1991] and Trost 1985]. Company records at a thermometer-manufacturing reported mercury vapor levels from 24-308 $\mu g/m^3$ (time-weighted averages). Vapor levels ranged from 26 to 271 $\mu g/m^3$, with urine levels from 1-345 $\mu g/g$ creatinine. Subsequent to the study by Ehrenberg et al. [1986], Hudson et al. [1987] investigated the exposure to mercury in children of the workers. The investigators reported that urine mercury levels in the children averaged 25 $\mu g/L$, some five times higher than that reported in controls. Furthermore, they observed a significant correlation between the urine mercury levels of the workers' children and the urine mercury levels of the parents. Zirschky and Witherell [1987] described the clean-up program implemented to decontaminate the homes of these children in order to reduce the likelihood of their additional exposure to mercury.

A recent report [ATSDR 1990a] summarizes the exposures of workers in another industry. Chlor-alkali chemical workers were exposed to high levels of mercury during a scheduled maintenance operation. Workers used oxyacetylene torches to remove old pipes and fittings, some of

which contained mercury. The heated mercury volatilized, and then condensed on the ceiling, walls, and floors, as well as on the clothing of the workers. Although protective clothing was used, work gloves, clothes, and boots were soaked with mercury. The workers returned home to their families in the evening, transporting mercury from the workplace into their homes. Thus families were also exposed to mercury. As a result of notification of EPA by a worker concerned about his family, EPA and ATSDR investigators evaluated the extent of exposure to mercury. High levels of mercury in various areas of the workers' homes were found but family urine mercury levels were not higher than those of control subjects.

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

King [1954] described the exposure of an amateur prospector to mercury when the prospector heated gold sand with mercury in a clay dish over the kitchen stove. The prospector developed severe coughing, vomiting, and became cyanotic. The author stated "The warning implied by this case as to the improper handling of mercury seems clear."

Another case of family mercury poisoning 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 miner's pan. The father's urine levels ranged from 200 to 560 μG/24 hr over four days following exposure. The mother who was in an adjacent room was also symptomatic, but urine levels apparently were not checked. The symptoms of the children (who were asleep in another room) included frequent coughing, fever, and nausea; their urine levels ranged from 33 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.

Hatch [1990] reported another case in which a woman was poisoned when she used mercury to extract gold ore in a cast-iron ladle over her kitchen stove. 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 three weeks of chelation therapy, her blood level was 193 mg/dl of mercury, suggestive of continued exposure. Concentrations of mercury in her home dissipated over time.

**(f) Chlorinated Hydrocarbons**

Twenty-eight reports in the literature present examples where chlorinated hydrocarbons in the workplace have the potential to be carried by the workers into their residences. Details of these reports are given in Table 13. These reports can be classified into four exposure categories. A few reports describe cases in which family

members exhibited toxicities resulting from exposure to chemicals brought home from the workplace. Other reports describe instances in which exposure to family members is documented by an increase in chlorinated hydrocarbon content in blood or urine. Other reports indicate that there is potential for exposure to workers' families due to the presence of chlorinated hydrocarbons found to be present in the home. Many reports suggest that there is a potential for exposure to occupants of the residences because of contamination of personal possessions, such as work clothing or tools, which are brought home by the worker.

Only four reports document instances in which health effects are seen in family members which are attributable to exposure to chlorinated hydrocarbons from the workplace. The first report, the earliest report found on home contamination by any substance, was by Lehman [1905] in Germany on family members who developed chloracne ascribed to the father wearing work-soiled clothing home. The earliest report from the U.S. of exposure to a chlorinated hydrocarbon in the home was in 1936, when the wife, daughter, and son of a worker who was exposed to hexachloro-naphthalene and chloro-diphenyl in an electrical insulation plant developed the same type of dermatitis seen in seventy-eight percent of the workers in the plant. The worker wore dirty work clothes home, and played with his children before changing into clean clothes. In this early report, it was recommended that the workers should be provided with protective clothing, lockers, and other sanitary facilities [Fulton and Matthews 1936]. In another instance, the son of a pipefitter

developed chloracne similar to that of this father, after his father wore his dirty work clothes at home after working around equipment contaminated with dioxin [Jensen et al. 1972a]. In addition, a six-week old daughter developed jaundice and hepatomegaly due to exposure to tetrachloroethylene in breast milk. It was discovered that the mother frequently visited the father during lunch at the dry-cleaning establishment where he worked [Bagnell and Ellenberger 1977].

Five reports describe cases in which exposure in the home was documented by measuring chlorinated hydrocarbons in the urine or blood of family members. No adverse health effects were described in any of these cases. Polychlorinated biphenyl was detected in the blood of wives of railway maintenance workers who repaired transformers [Fischbein and Wolff 1987] and in family members of municipal sewage treatment workers [Baker et al. 1980]. Methylenebis-2-chloroaniline, a suspected bladder carcinogen, was found in urine of family members of workers from two different specialty plastics manufacturing plants [ATSDR 1989a,b; ATSDR 1989a,b; ATSDR 1990b]. In addition, a potential human carcinogen, 3,3'-dichlorobenzidine, was present in urine of family members and employees of a chemical production facility [ATSDR 1991b].

Six reports document contamination of homes with chlorinated hydrocarbons from the workplace, but do not present data on content in biological samples from the residents of the homes. Dioxin was found to be present in homes in four areas, near roads where waste oil containing dioxin had been sprayed to retard dust [Doherty 1984; Ramsey 1987; Hess

1988; MacDonald 1988]. Tetrachloroethylene was found to be present in residences located in buildings with dry cleaning establishments [Stasiuk 1993]. In addition, polychlorinated biphenyls were present in house dust in vacuum sweepings of houses of occupationally exposed workers [Price and Welch 1972].

The remainder of the reports describe conditions in the workplace where workers would be exposed to chlorinated hydrocarbons where the workers could carry the contamination home. For example, chlorinated hydrocarbons used in the workplace were found to be present in lunch and locker rooms. One of the reports documented contamination of personal tools used by the workers that may be taken home. Data were not provided on work clothes contamination. However, in many cases, the workers did not wear company laundered work clothes. These reports are described in detail in Table 13.

**(g) Estrogens**

There are 7 references in Table 14 discussing take-home exposure to hormones specifically estrogens. Two references from Israel (1950s) and three from Poland (1960s), all case report series, deal with hyperestrogenic syndromes among children of pharmaceutical workers exposed to estrogens. The children experienced improvement with reduction of exposure or change of jobs in the parent. One reference [Aw et al. 1985] is a NIOSH health hazard evaluation in a pharmaceutical manufacturer where children of workers exposed to zeranol developed hyperestrogenic syndromes. The Bierbaum reference [Bierbaum 1993]

for pesticides or wood preservation, compared to homes where the residents did other work not involving arsenic. This lends credence to the argument that the arsenic was brought to the home by the worker on clothing and inanimate objects. The second reference [Falk et al. 1981] is a case report of hepatic angiosarcoma in a child associated with arsenic contamination of parental clothing, the water supply, and the environment.

(j) Cadmium

Four studies reported home contamination with cadmium which originated from parental occupation in a lead smelter (Table 14). In three of these reports a significant association between parental employment in the smelter and the concentration of cadmium in the blood (CdB) or in hair (CdH) of children was found. 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.

(k) 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 of a current potential case where an insulation worker's clothes were washed with the family

laundry. The wife and child suffered dermatitis in this case. Surface contaminations of fibrous glass can be detected using sticky sampling media and polarized microscopy [Schneider 1986; Schneider et al. 1989].

(l) Other substances

Tin is the only other chemical substance found to have been studied as a take-home contaminant [Rinehart and Yanagisawa 1993; Wainwright et al. (in press)]. Health effects of tin were not detected in theses studies.

(m) Infectious agents

Infectious agents (i.e., organisms that cause communicable diseases) are similar to toxic chemical such as lead and asbestos, in that they may be brought home on the clothes or bodies of workers. The intent of the Act is to investigate the extent to which workers' families might be exposed to life threatening pathogens because a working family member is routinely exposed to such agents in the workplace. It was not the intent of the Act that non-deadly infectious agents like the common cold be investigated [U.S. Senate 1991b].

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 direct contact, indirect contact, and airborne contact [adapted from Benenson 1985]:

In direct contact the immediate transfer of infectious materials may occur 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).

Through indirect contact the 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, vector-borne 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.

Airborne disease results from aerosols containing infectious agents being 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 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 in **aerosols** contained in dust brought to the home from the workplace (e.g., Q fever, anthrax and possibly fungal diseases). Infectious diseases that are spread by other means were not intended to be covered by the legislation. For example, tuberculosis spread by an infectious emergency service worker to family members via aerosols is not included nor is HIV infection that may be transmitted to a spouse during intercourse. 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 potential transmission of a bloodborne pathogen on soiled linen is considered to be negligible [CDC 1987].

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. However, since this legislation is intended to include agents that may be brought home on the worker's clothes or person, diseases that appear likely to be transmitted to the home on the worker include parasitic, vector-borne and air-borne diseases [adapted from Benenson]:

**Parasitic diseases** such as (1) scabies, 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. (2) Roundworm infection (*Ascaris lumbricoides*) from contaminated soil may be brought into houses and automobiles on the shoes of workers. The infection may than be transmitted to members of the household in dusts or via ingestion. Infection is usually highest in children aged 3-8 years. (3) 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.

Vector-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.

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 and murine typhus fever. In addition, approximately 90 arthropod-borne viral diseases have been identified. These include Colorado tick fever and encephalitis that are tick-borne. The mosquito is the vector for many arboviruses that infect humans; however, mosquitos are less likely than ticks 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.

Air-borne 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.

There are several fungal diseases that could potentially be transmitted via the clothes of workers including Aspergillosis (caused primarily by *Aspergillus fumigatus*, *A. niger*, and *A. flavus)*. 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].

The following are a number of examples from the literature that are indicative of the circumstances where infectious agents have been transmitted to the homes of workers:

(1) 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].

(2) In 1991, an immunocompromised patient (non-HIV related) was admitted to a hospital in Kansas. Forty-nine hospital staff members were subsequently 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 were also infected. Fourteen family members of the staff were infected with scabies from the

index patient [Clark et al. 1992].

(3) In 1984 an outbreak of Q fever in Idaho was associated with a sheep research station. Two of the 18 cases of Q fever 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 fomites 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].

(4) 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. One of the members of the play was a shepherd who came to rehearsals in his work clothes. *C. burnetti* was isolated from the dust collected from the shepherd's clothes [Marmon and Stoker 1956].

(5) 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. Nineteen of the 60 employees of the school system met the case definition for giardiasis. Three members of the employees' households also had persistent diarrhea consistent with giardiasis infection [Osterholm et al. 1981].

(2) Incidences of Take-Home Contamination - Industrial Hygiene Aspects

(a) Sources of Take-Home Contamination

In reviewing the numerous studies listed in Table 15, one finds that sources of chemicals exposure for family members include the classic mode of taking chemicals home on work clothes and several instances of atypical sources. Chemicals reported in the table include lead, mercury, pesticides, cadmium, asbestos, etc. Lead is the most frequent agent identified due perhaps to its widespread use and routine screening. Industries associated with lead being taken home include lead battery production, manufacture of pewter objects, pottery manufacturing, lead mines and lead smelters.

Several case studies include atypical situations that do not involve strictly carrying toxin chemicals home on the clothes. In a study reported by Bagnell and Eklenberger [1977], a baby was exposed to tetrachloroethylene (TCE) via the mother's breast milk. The mother did not work with TCE, but was exposed via daily visits to her husbands place of work (dry cleaning establishment) to have lunch (0.5 to 1 hour). A second atypical case, reported by Benning [1958], involved emotional upset at home due to employees being exposed to high levels of

mercury. A tamping powder used in the production of electric motors was found to contain 22-24% mercury. Fifty-two of ninety employees (all females) had symptoms of mercury poisoning. Symptoms included antisocial behavior, sore mouths, shaking and in some cases inability to care for their families. After the company replaced the tamping powder with a powder not containing mercury and installed local exhaust ventilation, the employees symptoms began to disappear.

Some of the case studies include contaminated cloth goods taken home by the worker. A study by Anderson et. al. [1965], describes a case from Canada in which two boys who played together were admitted to a hospital at separate times. Both boys nearly died from anti-cholinesterase poisoning. The exposure resulted from the boys sleeping on sheets that were contaminated with parathion during transport in the hold of a ship. In another report, Eitzman and Wolfson [1967] describe 30 deaths of children due to parathion poisoning that occurred in the state of Florida between 1959 and 1964. In one episode, three siblings were poisoned after making a swing from a burlap sack that was heavily contaminated with parathion. The children made the swing, played with it that day and the next day - when they became ill.

Other examples which could be very important involve cottage industries. In a case from India reported by Joshua et al. [1971] three of nine children died from lead poisoning. In this study the authors note that the parents refused to recognize the hazard and did not take any corrective action. In a second case study reported by Hung [1980],

battery processing in a Taiwan home resulted in blood lead levels of 100, and 124 ug/dL in three children. Two of the children were diagnosed with lead encephalopathy. Cottage industries have been associated with lead (e.g., ceramics, lead batteries, pottery, battery processing, quench hardening & type printing), asbestos (e.g., asbestos cement production), and mercury (e.g., home gold ore processing).

(b) Concentrations of Contaminants

Take home contamination exposures have been documented for several chemicals, including arsenic [Klemmer et al. 1975]; asbestos [Driscoll and Elliott 1990], [Gibbs et al. 1989], [Huncharek et al. 1989], [Nicholson 1980], [Nicholson 1983], [Seixas 1986]; beryllium [Chamberlin 1957], [Cohen and Positano 1986], [Eisenbud et al. 1949]; dioxin [Doherty 1984]; fungi [Pasanen 1989]; lead [Abbritti 1979], [Abbritti, 1987], [Anonymous 1992], [Baker et al. 1977], [Barrett 1944], [Brockhaus et al. 1988], [Carvalho et al. 1984], [Chernard et al. 1987], [Cook et al. 1993], [Dolcourt et al. 1978], [Dolcourt 1981], [Elwood et al. 1977], [Ewers et al. 1982], [Ewers et al. 1994], [Fischbein et al. 1991], [Gittleman et al. 1991], [Hung 1980], [Katagiri et al. 1983], [Kawai 1983], [Kaye et al. 1987], [Kelly 1994], [Koplan et al. 1977], [Landrigan et al. 1980], [Lyngbye et al. 1991], [Marvelius et al. 1989], [Martin 1974], [Martin et al. 1974], [Matte and Burr 1989], [Matte et al. 1991], [McCammon et al. 1991], [Menrath et al. 1993], [Milar and Mushak 1982], [Millar 1978], [MMWR 1992b], [MMWR 1989a], [MMWR 1985], [MMWR 1977b], [Molina-Ballesteros 1983], [Morton 1902], [New Jersey Dept. of Health], [Piacitelli 1994], [Piccinini 1986], [Pitts 1986],

[Pollock 1994], [Quinn 1985], [Remakrishna 1982], [Rice 1978], [Ritcher 1985], [Richter 1980], [Rinehart 1993], [Schumacher 1991], [State of Alabama 1992], [Watson 1978], [Winegar 1977]; mercury [Ehrenberg et al. 1986], [NIOSH 1973], [Trost 1985]; methyl parathion [Finley et al. 1977]; PCB [Fischbein, 1986]; pesticides [Osario 1994]; tetrachloroethylene [Bagwell and Ellenberger 1977]; and Zeranol [Aw and Stephenson 1985]. The majority of these measurements involved biological monitoring (such as blood lead determination) to document exposure of family members to take home contamination. However, air, vacuum, dust, surface wipe and several other types of samples were also used in some cases. Levels of contaminants found to document the presence of take home contamination extracted from Table 15 are summarized below.

| Contaminant | Type of Sample | Range |
|---|---|---|
| Arsenic | Home vacuum samples | 1.1-1,080 ug As/g dust |
| Asbestos | Clothing Vacuum Samples | Asbestos present |
| | Car Seat Vacuum Samples | Asbestos present |
| | Lung Tissue Fiber Counts | 2,000-221,000 f/g lung tissue |
| | Air samples - homes | 50-5,000 $ng/m^3$ |
| Beryllium | Lung Tissue Analysis | Beryllium present |
| | Clothing samples | 12-37 $mg/m^2$ shirt fabric |
| | Air Samples - simulated cleaning of work clothes | Dose - 17 ug Be |
| Dioxin | Soil samples | 3-48 ppb |
| | Carpet samples | 2.6 ppb |
| | Home vacuum samples - post cleanup | < LOD |
| Fungi | Air samples - homes | $10^3$ - $10^4$ $CFU/m^3$ |
| Lead | Blood lead | 220ug/dl |
| | Home vacuum samples | 13 $mg/m^2$<br>ND-3,400 ppm |
| | Car wipe samples | 1.7-3 mg<br>814-3,910 ppm |
| | Hair lead | 12.69 ppm |
| | Soil samples | 51-520,000 ppm |
| | Carpet samples | 11.8-282.8 $mg/ft^2$<br>1,700-84,000 ppm |
| | Air samples - homes | 2-50 $ug/m^3$ |
| | Surface dust samples - homes | 260-326,000 ppm<br>20-295,000$ug/m^2$ |
| Mercury | Air samples - examination trailer | 21.5-23.4 $ug/m^3$ |
| PCB | Serum PCB | 69-101 ng/mL |
| | Adipose PCB | 20.7 ng/mL |

| | | |
|---|---|---|
| Tetrachloroethylene | Breast milk tetrachloroethylene | Tetrachloroethylene present |
| | Serum tetrachloroethylene | Tetrachloroethylene present |
| Zeranol | clothing | Zeranol present |

Standards with which to compare these levels are lacking in most cases. Blood lead levels are one exception with a reccommendation from CDC of 10 ug/dl for children. Milar and Mushak propose that levels of contamination exceeding 1,000 ppm of lead in housedust or 50 ug lead/$m^2$ surface may cause a definite health hazard to children. In cases where air levels are measured, they are most often compared to recommended standards for worker populations which may not be protective for children and for the prolonged exposure periods that may occur at home. For many of the measures, the only comparison is to levels found in control groups or background levels. This means that in these cases, even though it is known that a take-home contaminant is present, the significance of this presence in the causation of health effects may be unclear.

**(c) Effectiveness of Preventative Measures**

One cross-sectional study conducted in Brazil [Silvany-Neto et al. 1989] of blood lead levels in children living near a lead smelter, indicated that controls implemented between 1980 and 1985 by the company to reduce emissions did not successfully prevent children from being overexposed to lead. A comparison of the results from a follow-up survey in 1985 with the results from an initial survey in 1980 demonstrated a reduction in the proportion of children who had moderately elevated zinc

protoporphyrin levels ($2.11 \leq ZPP \leq 4.79$ umol/l) and children who had extremely elevated levels ($\geq 4.80$), but the proportion of children with above normal levels as a whole was similar to that found in the 1980 survey. The sources of exposure present in 1985 were believed to be those generated before the implementation of controls. This study reminds us that preexisting sources of exposure, as well as the currently-generating sources of exposure, must be addressed in order to prevent overexposures.

A publication of two case studies of workers' spouses who had allergic reactions to antigens brought home by their spouse [Venables and Newman-Taylor 1989], indicates that the practices of showering and changing clothes before leaving work were effective at preventing allergic reactions. In the first case, the antigen was rodent urine which the wife brought home from the research laboratory she worked at. 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 from his work at a precious metal 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.

The report of an investigation of a stained glass window-making studio [Donovan 1994(a) and (b)], which was conducted by NIOSH to evaluate workers' exposures to lead, indicated that the use of traditional

industrial hygiene controls were affective at preventing contamination of the worker' home that was adjacent to the studio. Industrial hygiene practices used at the studio included local exhaust ventilation for use 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, and prohibiting work shoes from leaving the studio. Based on the results of surface-wipe sampling, which demonstrated elevated levels in the studio (1.2 to 1600 $mg/m^3$) but not in the house (non-detected or trace), the author concluded that the industrial hygiene practices prevented the contamination of the house. The author suggests that these industrial practices could benefit others in the trade. These findings are particularly important since many of the examples of gross home contamination found in the literature are associated with home businesses or "cottage industries."

There were three publications [Baker et al. 1977; Barnett 1994; and MMWR 1977b], which did not demonstrate the effectiveness of controls, but instead, demonstrated that investigations can be effective at getting controls implemented. This is a reminder that evidence of a problem, or at least awareness of a potential problem, may be needed in some situations before controls are implemented.

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 from the investigation.

Barnett [1994] reported on a pesticide spill (chloropicrin) that occurred at an employee's house 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 Hazmat 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 appropriate storage and means to secure containers while transporting chloropicrin must be used.

One article discusses the effectiveness of washing work clothes, worn by workers at a pitch coking plant, at removing 3,4 Benzo(a)pyrene [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-benzopyrene from the fabric . . . ." It is assumed that the "present procedure" was normal detergent and rinse.

Excessive lead exposure was identified for workers at a battery factory and some of the workers' children [MMWR 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.

(d) Effectiveness of Home Decontamination Procedures

There are few reports in the literature on the effectiveness of methods for decontamination of household carpets and floors.

Repeated steam cleaning (Calgon* - detergent) reduced lead (Pb) dust in carpets by 61% compared to a 12% reduction when cleaning used detergent only. [Milar and Mushak 1982]

A study of contaminated homes near a lead smelter found that combining HEPA filtered dry vacuuming and shampooing of carpets removed less than 8% of Pb. [CH2M Hill 1991]

Repeated HEPA dry vacuuming of carpets was ineffective in reducing Pb contamination at the surface, and in some conditions actually increased; dry HEPA vacuuming removed 95% of total dust from bare wood floors; dry HEPA vacuuming removed 75% of total dust from linoleum floors, with an additional 20% in a final wet washing. [Ewers et al. 1994]]

Use of HEPA filtered dry vacuuming was also ineffective in removing asbestos fibers from carpets; HEPA filtered hot-water extraction reduced

asbestos concentration by 70%. [Kominsky et al. 1990]

**(e) Effectiveness of Laundry Procedures**

This section will review articles dealing primarily with the ability of laundering procedures to adequately clean workplace clothing. Properly cleaned protective clothing should prevent contamination of other clothing and the skin, thus reducing the chance of bringing workplace toxins into the home. It must be emphasized that the wearing or taking home of workplace (protective) clothing, often for the purpose of laundering, is the major mechanism of exposure of homes and family members to workplace toxins. As described elsewhere, this mechanism of exposure has been documented for asbestos, silica, lead, fiberglass, arsenic, beryllium, mercury, asthmatogens, pharmaceuticals, many pesticides, chlorinated hydrocarbons, explosives, and other toxins. As many articles note, it follows that great care must be utilized in laundering contaminated clothing, and that one must consider appropriate risk evaluation, work practices, engineering and other controls, and protective clothing when involved in this process.

An obvious solution (besides ending or reducing the hazardous exposure itself) to the problem of home contamination by soiled work clothes is to leave all such clothing at work, as required for several workplace hazards in federal regulations. Even this may not be totally effective unless scrupulous separation of home and work clothes and other measures are followed [Richter et al. 1985; Lundquist 1980]. Because of the often incomplete cleaning, some articles have recommended the

replacement of work clothes on a periodic basis [Masek et al. 1972]. The use (and subsequent proper disposal) of disposable protective clothing is another means of avoiding home contamination.

A related issue is the potential contamination of waste water effluents during laundering. This is under federal control for commercial laundering operations [Weller 1994]. Other potential hazards of laundering include local exposures to perchloroethylene from commercial dry cleaning facilities [Stasiuk 1993].

Through the years, most sources have recommended daily laundering of work clothes for virtually all exposures [Katzenellenbogen 1956; Marceleno et al. 1974; Benning 1958; NIOSH 1973; Seixas and Ordin 1986; Venable et al. 1993]. As noted below, the effectiveness of this practice is often inadequately studied, and is frequently incomplete.

(1) **Fibers**

Studies are limited regarding the effectiveness of cleaning of materials for fibers. Fiberglass from contaminated work clothes or fiberglass textiles can contaminate other clothing in the wash [Peachey 1967; Abel 1966; Madoff 1962]. In addition, the Peachey paper found continued dermatitis after wearing clothes undergoing two washes of fiberglass-contaminated clothing, indicating incomplete fiber removal during the laundering. A NIOSH study of dry cleaning a coat made with 8% asbestos fiber also showed that fibers were picked up by other clothes in the same wash [NIOSH

1971]. Unpublished data show that in one commercial laundering setting, clothing contaminated with ceramic fibers (50 to 500 fibers per square millimeter) were, after washing, reduced to not detectable to 7 fibers per square millimeter [Textile Rental Services Association of America 1994].

(2) Non-Fibrous Dusts

The general practice of laundering assumes that appropriate laundering should remove most non-fibrous dusts from clothing. However, Cohen and Positano [1986] in a small study showed considerable retention of beryllium in old shirts versus new ones, even after laundering. The ability to resuspend the dust in the laundered clothing was quite reduced, however. In the case of silica, one small study found that laundering could be done without contamination of the home area. [Versen and Bunn 1989]. However, in other studies, as noted above, the process of home laundering was a major cause of home contamination.

(3) Firefighter's Protective Clothing and Chemical Protective Clothing

Several NIOSH investigations in this area have been done [Kominsky 1984a,b,c; Kominisky 1987; Kominisky and Singal 1987; Orris and Kominisky 1984]. Most were related to contaminations with polychlorobiphenyls (PCBs) during electrical fires. These efforts indicated that, in one setting, a standard detergent wash followed by a water wash did not effectively remove the PCBs from the (Nomex) protective clothing, and may in fact have increased the

contamination. The recommended wash was an alkaline (trisodium phosphate base) or nonionic (octylphenoxypolyethoxyethanol base) synthetic detergent. It was noted that the lack of visible contamination should not be used to guide cleaning needs in this setting; testing for residua is needed. The need to collect, test, and properly dispose of the wash water was also noted.

One research effort indicated that a trichlorotrifluoroethane [Freon 113] based dry cleaning machine with a revolving chamber system reduced site-contaminated garment PCB levels by an average of 88%, and of laboratory-contaminated garments by 99% [Kominsky 1987]. Since safe levels of surface contamination are not known, it could not be stated if this would prevent worker or home contamination. Perkins et al [1987] found that Freon decontamination reduced toluene to 0.8 percent 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 degrees Centigrade for 24 hours reduced the level to 0.25%. This research also showed that the 50 degree temperature air drying reduced seven other solvent contaminants to the limit of detection levels. Finding evidence of no 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 toxin.

(4) Laundering for Pesticide Removal from Work Clothing

Numerous studies have examined the effect of laundering for pesticide (or herbicide) removal from work clothing [Anonymous 1994; Chiao-Cheng et al. 1988; Clifford and Nies 1989; Easley et al. 1981a, 1981b, 1982a, 1982b, 1983; Easter 1983; Finley and Rogillio 1969; Finley et al. 1974, 1979, undated; Goodman et al. 1988; Graves et al. 1980; Hild et al. 1989; Keaschall et al. 1986; Kim et al. 1982, 1993; Kim and Wang 1982; Laughlin and Gold 1988, 1989a, 1989b; Laughlin et al. 1981; Lillie et al. 1981, 1982; Mullen and Lehrburger 1991; Nelson et al. 1992; Oakland et al. 1989; Obendorf and Solbrig 1986; Rigakis et al. 1987; Satoh 1979; Stone and Wintersteen 1988]. Other articles have addressed related issues, such as the effectiveness of various types of clothing, typical use patterns, etc. [Cloud et al. 1983; Stone et al. 1986; Orlando et al. 1981; Oakland et al. 1992; Kim and Kim 1988; Rucker et al. 1986]. Most of these articles deal with pesticide exposure. In general, herbicides are less toxic than insecticides, although the former have a wide range of toxicity and must be handled carefully [Lavy 1988].

Several review articles and recommendations are of particular use to those interested in the hazard of bringing toxins into workers' homes Easley Laughlin, and Gold [1981]; Laughlin and Gold [1988]; Nelson et al. [1992]; Stone and Wintersteen [1988]. Nelson et al.

[1992] found a range of pesticide post-wash residue of 0 to 41% of the pre-wash level for six classes of pesticides, with an average of 13%. Keaschall [1984] found laundering to reduce contamination of several pesticide classes approximately 90% to 95%. However, Easley et al. [1982] and Laughlin et al. [1985] found only 67% of concentrated (54%) methyl parathion was removed from clothing after ten washes. Because of the many unknowns, some authors recommend yearly replacement of coveralls in pesticide application work [Stone and Stahr 1989].

While the cited articles discuss laundering considerations, several also note the importance of primary prevention [Ware et al. 1973]. For example, it has been shown that clothing contamination with methyl parathion is reduced by 90% if one waits two days before entering the treated field instead of just one day [Finley et al. 1977; Finley et al. 1979]. Because of the incomplete removal of clothing contaminants, heat, ultraviolet light, microwave [Kim 1989], and antitoxic chemicals have been suggested as soil degraders. However, these have not been extensively studies, and are not commonly used.

The following summary of findings and practical recommendations is based primarily on research of pesticide exposures cited above, which generally involved home washing equipment. It is important to note, therefore, that different or additional recommendations, as described in other parts of this section, may apply to the

decontamination of substances other than pesticides.

Summary of Laundry Findings:

1. Although many generalizations can be made, the effectiveness of laundering depends on the specific toxic chemical exposure, and its formulation.

2. Pesticides can leach from contaminated clothing during laundering to contaminate other parts of the same garment, to other clothing in the same wash, and to a lessor extent to the washing equipment, thus potentially contaminating later washes. The solubility of the pesticide is a major factor in this process.

3. The pesticides that leach from contaminated clothing to other clothing or surfaces have been found to be biologically active, and thus capable of causing disease.

4. Although laundering can effectively reduce the level of contamination of clothing, it almost never completely removes the toxic residues.

5. Most experts note that, although further research is needed, it has not been established that the amounts of residue left after a careful laundering process constitute a health hazard for humans. This statement assumes complete washing after every use, using a prewash and the optimal wash conditions, and "normally" soiled

clothing. It should be emphasized that clear examples of human toxicity have occurred in laundered work clothes [Clifford and Nies 1989].

6. The removal of pesticides from clothing is increased by using hot water (e.g., 60° C.); a prewash or pre-rinse stage; multiple washings; a heavy duty detergent, especially for those chemicals that are oil-based; full volumes of water and a full wash time; and a prolonged drying time.

7. Heavily contaminated clothing (such as from a spill of full strength solutions) has been shown to be toxic to humans even after multiple washings.

8. The repeated use of clothing in exposure settings without laundering after each use leads to an accumulation of residues.

9. Laundry additives such as bleach or ammonia have not been found to contribute to the removal of pesticide residues.

**Summary of Laundering Recommendations from Cited Literature:**

In most work settings, there is no quantitative assessment of the toxin contamination of the clothing. The recommendations thus rely on qualitative assessments of exposure, and are meant to represent practical yet effective approaches to the problem, taking various factors into account.

1. One should always read the chemical toxin label carefully for information regarding handling and recommended laundering procedures.

2. Heavily contaminated clothing should be discarded and/or burned. Because of the many unknowns, some authors recommend yearly replacement of coveralls in pesticide application work, even when heavy contamination is not known to have occurred [Stone and Stahr 1989].

3. Pesticide-contaminated clothing should be washed separately from other clothing.

4. Laundering methods should include the use of a prewash/pre-rinse step, hot water (e.g., 60° C.), and full cycle water volumes and time (12-14 minutes, and a double rinse if possible). It is best to wash only a few garments in any one wash.

5. Two washes are recommended routinely by some; and where significant contamination is expected by others.

6. Contaminated clothing should be laundered after each use.

7. Line air drying is recommended, both to avoid contamination of an automatic dryer which is difficult to clean, and to allow sunlight and time to further reduce the toxic residues.

8. After laundering contaminated clothing, the washer should have an "empty load" wash with detergent and full volume and time settings to reduce the chance of contamination of later wash cycles.

9. One should wear rubber gloves to handle pesticide-soiled clothing, and dispose of these when deterioration is noted, and at the end of the pesticide season. A separate garbage-bag lined cardboard box should be used as a hamper, and discarded at the end of the application season. All appropriate safety and health measures should be taken when handling the clothing.

**Airshowers and Shoecleaners**

Simonson and Mecham [1983] showed that airshowers removed from 5 to 72% of lead dust from clothing in workplace studies, and 23% to 69% in laboratory studies. Some small amount of breakthrough the clothing (posing a possible skin exposure) was noted. Shoe cleaners were observed (non-quantitatively) to be effective, although potential problems with adequate maintenance were noted.

**Other Agents**

Little is reported 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].

**(3) Review of Federal and State Laws**

manufacturers, processors and distributors to maintain records of significant adverse reactions to health, and §2607(e) requiring manufacturers, processors and distributors to report immediately information that a substance or mixture presents unreasonable risk of injury to health or the environment.

**Asbestos Hazard Emergency Response Act of 1986**

This act specifically requires that state plans for accrediting asbestos removal contractors contain procedures to prevent asbestos exposure to an employee's family.

**Residential Lead - Based Paint Hazard Reduction Act of 1992**

This Act has several requirements that could be relevant to protection of workers' families from lead-contaminated dust in their homes. These include definition of lead-contaminated dust, development of a comprehensive lead-exposure abatement program, studies of sources of lead exposure in children, including occupational contributions.

**Federal Insecticide Fungicide, Rodenticide Act**

Requirements in this Act on storage and disposal of pesticide containers and the subsequent regulation (40 CFR 165) are particularly relevant to protection of workers' families, especially in agriculture where cases of childhood poisoning from improper disposal has been a problem.

**(4) Responses of Federal Agencies to Incidences of Home Contamination**

The purpose of this section is to review documented investigations and

enforcement of regulations by state and federal agencies relative to home contamination incidents. The documentation for this section is compiled in Table 17.

## (a) Centers for Disease Control and Prevention (CDC)

Many of the centers making up the Centers for Disease Control and Prevention, including the Institute, have been involved in investigations of potential take-home exposures.

### (1) National Center for Environmental Health (NCEH)

The mission of NCEH is to prevent and control disease injury and disability related to the interactions between people and their environment outside of the workplace. The center's applied research has evaluated potential take-home exposures of toxic substances, including lead, pesticides, arsenic, tin, and PCBs.

#### Lead

Three studies [Baker et al. 1977, Landrigan and Baker 1981, Matte et al. 1991] were conducted involving smelters. One [Baker et al. 1977], of children and spouses of lead smelter workers determined that the children had higher blood-lead levels and that their homes had higher concentrations of lead than did controls. Families of battery plant workers were found to have elevated blood lead levels in three studies [Dolcourt et al. 1978; Watson et al. 1978; Dolcourt et al. 1981]. Novotny et al. [1987] looked at the blood lead levels of firing range workers and their spouses finding elevated levels in the workers but not

in the spouses. An ongoing study of workers exposed to the Firing Range at the FBI academy suggests that there was potential for take-home exposure of lead among these workers (based on contamination of workers' vehicles with lead) although lead dust levels in the workers' homes and blood lead levels among the workers' children were low (suggesting that the children were not being exposed to significant amounts of lead at the time of the study).

In March, 1993 NCEH and NIOSH collaborated on an Exposure assessment for heavy metals associated with a smelter in Oruro, Bolvia. The investigators evaluated biological and environmental samples for lead, arsenic, antimony, and tin. Both biological and environmental testing suggested take-home of tin, although the biological results were not elevated to levels documented to cause adverse health effects.

(2) **National Institute for Occupational Safety and Health**

Health Hazard Evaluations (HHEs) are conducted by investigators from NIOSH. Employers or employees (or their representatives) can request a Health Hazard Evaluation which studies potentially hazardous exposures to material or conditions in the workplace. A review of reports from these HHEs and other NIOSH studies conducted under its general research authority indicates about 30 looked at potential or actual instances of toxic materials travelling home with the worker.

**Asbestos**

Construction [Lemen 1972], flooring material manufacturing [Belanger et

al. 1979], friction product manufacturing [Seixas and Ordin 1986], and a chemical plant [Driscoll and Elliot 1990] were among sites investigated. 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 hygiene practices. In two cases [Seixas and Ordin 1986; Driscol and Elliot 1990] asbestos was detected on the workers' clothes as they left the worksite. In a NIOSH evaluation of a brake service facility [Godby et al. 1987] it was noted that most workers did not change out of work clothes at work and took their work clothes home to laundry.

**Lead**

Stained glass [Landrigan et al. 1980], battery manufacturing and recycling facilities [Apol and Singal 1980; Matte and Burr 1989; Gittleman et al. 1991], tank lining companies [McCammon et al. 1991; MMWR 1992a], assay labs [Gunter et al. 1987], and building renovation contractors [Kiefer 1994] were among situations investigated for worker exposure to lead and the possibility of lead brought home from the workplace by workers contaminating workers' homes. In one case [Matte and Burr 1989], a back-yard battery repair shop, contamination of the home and elevated blood-lead levels of family members were found. Another case showed that the elevated blood lead levels of stained glass workers' families were related to workplace exposure [Landrigan et al. 1980]. In another case, lead was detected in workers' cars, indicating a potential for transfer to the home [McCammon et al. 1991]. In most cases, improved hygiene practices were recommended to prevent

contamination of homes.

PCBs

Investigations of two of these cases, one a manufacturing plant and one a rail yard contaminated with PCBs, resulted in hygiene recommendations [Hartle et al. 1987; Hartle 1987]. Several HHEs dealt with firefighters PCB exposure [Kominsky 1984, 1984b, 1984c, 1987, 1987a; Seligman 1984]. Generally, the firefighters' clothing was potentially contaminated. Recommendations for laundering clothing and using protective gear were made.

**Other**

Children of chemical plant workers had enlarged breasts probably due to Zeranol (estrogenic animal growth promoter) exposure at home. Zeranol was thought to be brought home on the worker's clothing. NIOSH made recommendations to prevent home contamination [Aw et al. 1985].

During the Hazard Evaluation of a thermometer plant, the NIOSH trailer where tests were given became contaminated with mercury, suggesting take-home contamination with mercury was possible [Ehrenberg 1986].

**(3) Other CDC Centers**

In a joint study by CDC and EPA [Canon et al. 1978], kepone poisoning of wives of kepone workers was investigated showing a connection with the affected wives washing their husbands' clothes. Wolfe et al. [1961] made recommendations for pesticide applicators that include proper

decontamination of empty drums and applicators' clothing.

(b) Agency for Toxic Substances and Disease Registry (ATSDR)

The Agency for Toxic Substances and Disease registry has conducted several relevant studies of workers homes in connection with contaminated site studies. Investigation of a chemical manufacturer in Adrian Michigan showed detectable levels of MBOCA in the urine of workers families and tests showed the homes were contaminated. The material may have been tracked out on the employees' clothing and shoes. Homes were decontaminated. [ATSDR 1989a, 1990b, 1993b]

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

ATSDR reports describe other investigations into MBOCA, lead [ATSDR 1991a, Alabama Department of Public Health 1991] and mercury [ATSDR 1990a] home exposures.

(c) Occupational Safety and Health Administration (OSHA)

Although in general OSHA's jurisdiction is limited to the workplace itself, several reports on take home contamination exist.

One report [Natarajan 1994] describes an investigation into high blood-lead levels in a child. The child's father (who also had an elevated blood-lead level) worked as a radiator repairman. In Kankakee IL

[Wiehrdt 1994], the local health department referred a case to OSHA of gross lead contamination of a home. Two children were hospitalized and chelated. The father, an employee of a battery plant, was bringing lead home on his clothing. OSHA conducted a comprehensive inspection of the plant. In Indianapolis IN, [Wiehrdt 1994] OSHA suspected a potential home contamination problem while inspecting a local plant in 1984. OSHA referred the potential problem to the local county health department. Subsequent investigation by this department determined that at least one of the children had a blood lead level of 50 $\mu$g/dL. In 1990, the Cleveland Area OSHA office [Wiehrdt 1994] investigated a company where it was determined that employees were being exposed to lead. Hearing that three employees had children with elevated blood lead levels, OSHA conducted sampling in the employees' homes. Later, the case was reported to the state's Department of Health and the Cleveland Lead Hazard Abatement Center.

**(d) Environmental Protection Agency (EPA)**

Reports from the Environmental Protection Agency indicate various investigation and remediation efforts for Dioxin contamination of homes from contaminated fill and roadway material. [Ramsey 1987; MacDonald 1988; ERM-Southeast Inc. 1989; CH2M Hill 1991; Doherty 1984; Hess 1988; Beegle and Forshund 1990.] Although these reports do not describe take home contamination, i.e., contamination brought home by workers from their workplace, they do outline various protocols for cleaning contaminated homes.

(e) Department of Energy (DOE)

Beginning in October 1990, the Department of Energy (DOE), under DOE order 5000.3A, requires 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 data base 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.

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.

The ORPS Program Manager provided 16 reports related to contamination of workers' homes with hazardous substances transported from the workplace. 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:

1. 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."

2. 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."

3. Initially-undetected damage to an americium source resulted in contamination of a worker's hat, which was found on routine survey several days after the event. 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, which concluded that most likely explanation 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 the offending lab. The team reviewing the incident recommended more careful handling of and administrative controls for americium sources.

**(f) Nuclear Regulatory Commission (NRC)**

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 should 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 (ref: personal communication, Ken Brockman, NRC).

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.

1. 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.

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

3. 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.

4. Low-level contamination, initially undetected at a portal monitor, was discovered on clothing brought home by a power reactor worker.

5. 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.

6. 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.

7. 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:

1. 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.

2. 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 de-contaminated. There were no reports of adverse health effects.

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

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

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

6. Contamination found at a residence recently vacated by owner of a licensed laboratory. No additional information available.

(g) 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 driers were the most heavily contaminated part of the homes. The company cleaned the homes.

(5) Responses of State Agencies to Incidences of Home Contamination

Information on state agency investigations into incidents of home contamination was obtained in two ways. The first was via 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 18 and 19. The second way documentation of state investigations was obtained was through literature searches in the open literature. Studies by state agencies identified in this way are compiled in Table 20.

In some instances where states reported they had no information on home contamination investigations or incidents, such reports were found in the literature. Likewise, states which did not respond to inquiries also had

reports in the literature of investigations that took place in the state. Commonly, in these instances, these investigations were conducted by local health departments collaborating with federal agencies and reported in journal articles or CDC's MMWR.

The reports of these state agency responses to, or investigations of, home contamination incidents are divided into three groups: Lead; Pesticides; 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 poisonings through local health departments and physician reporting and as a result can identify incidents for investigation.

In 1975, 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 blood lead levels greater than or equal to 30μg/dL. [MMWR 1976]. Again, the source for lead appeared to be the parents' contaminated clothing. This level differed significantly from similar local populations of children. Oklahoma investigators found similar results in an investigation of blood lead levels in children of battery manufacturing workers [Morton et al. 1982]. In North Carolina, 72% of the children of battery plant workers in an investigation had blood lead levels of 30μg/dL or above [MMWR 1977b]. An investigation by the Alabama Department of Health in 1991 of a battery recycling operation revealed elevated blood lead levels in most of the workers. When the local County Health Department measured the blood lead levels of the children of these workers, mean blood lead levels were 22.4μg/dL [MMWR 1992b]. These investigations resulted variously in chelation therapy for victims, recommendations for improved hygiene practices, improved engineering controls, OSHA fines, and, in one case, a court order to remove all workers from the workplace.

Elevated blood lead levels in children have also been traced to radiator repair workers. Investigators from the New York City Department of Health [Nunez et al., 1993] found that three of seven radiator repair workers' children tested had blood lead levels at or above 10 μg/dL. The Minnesota Department of Health [Lussenhop et al., 1989] investigated blood lead levels in radiator repair workers and their children.

Other cases investigated by states where elevated blood lead levels in

children were attributed to the parents' occupation include a Mississippi Department of Health investigation into lead contamination of a lead transporter's home [Pollock 1994] a capacitor and resistor plant in Colorado investigated by the local Health Department and OSHA [MMWR 1985], a belt buckle, plaques, and awards manufacturer investigated by a Colorado local Health Department [MMWR 1989], a soil nutrient manufacturer investigated by the Iowa Bureau of Labor [Hooper 1991], and an un-identified company in Indiana reported through the state's Department of Labor [Molovich].

In 1992, a pilot study of take-home lead was conducted by the New Jersey Department of Health [Czachur et al. in press] 98 persons with elevated blood lead levels (≥ 40μg/dL) were identified 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 level testing for their children. Blood lead levels were obtained on 28 children from the families of 15 of these workers. Nine (32%) of these children had blood lead levels now considered to be a potential risk for adverse health effects (≥ 10μg/dL). Interestingly, of six children whose parents did not bring their clothes home to be laundered, none had blood lead levels at 10μg/dL or above while the parents of all nine children at 10μg/dL or above did bring their clothes home to be washed.

**Pesticides**

Surprisingly, no states responded with nor was any information found about pesticide home contamination. As a consequence, there are no reports

listed in the tables. However, awareness of the potential for such cases was demonstrated by the existence of programs and printed materials to educate about the potential for such home contamination and ways to prevent it. Agricultural extension services in Arkansas [Lavy 1988; Huitink 1994], Florida [Anonymous 1994], Iowa [Stone et al. 1986; Stone and Wintersteen 1988], Louisiana [Finley et al. no date], Michigan [Branson and Henry 1982], and Nebraska [Easley et al. 1981] have developed training courses for pesticide safety, brochures describing safe application practices, brochures on proper laundering techniques for clothes worn during application, or brochures on proper disposal technique of pesticides containers.

**Other**

A 1936 study by the Pennsylvania Department of Labor and Industry of the effects of Hexachloro-Naphthalene and Chloro-Diphenyl exposure on wire insulation workers attributed dermatitis in a young child to exposure from his father [Fulton and Matthew 1936].

In 1948 [Hardy 1948], a paper published on beryllium exposure and disease by a physician with the Massachusetts Department of Labor mentioned the case of the mother of a worker who developed beryllium related disease. The suspected cause was beryllium exposure from her daughter. In the 1960's, contact cases of beryllosis 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]. Interestingly, although the investigators 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 family member exposure.

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Table 1. Studies of Take-Home Asbestos Exposure (Cohort Studies)

| Author (year) | Location Industry/ Population at Risk | Study Design | RESULTS | Comments |
|---|---|---|---|---|
| Anderson [1983] Anderson et al. [1976, 1979a,b] | Patterson, New Jersey<br><br>Amosite workers employed 1941-1945 in thermal insulation materials factory | Cohort.<br><br>Morbidity and mortality among 2218 household contacts of amosite workers identified. 679 of 1545 alive 1980 were examined. Occupational, residential, smoking, medical history questionnaire administered to the exposed cohort. Vital status follow-up is through 1980. | 3/663 observed deaths were due to mesothelioma.<br><br>Lung cancer SMR=152 (25 obs./16.4 exp.); SMR=185 after 20 years latency. 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). The SMR=1.7. Among males with 20+ years latency, there were 12 lung cancer deaths observed vs. 6.1 expected (SMR=1.97). | 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. |
| | | 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 from chest radiograph 1975-1976. | 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 ($p<0.001$). Prevalence of abnormalities increased with duration since first exposure; 40% prevalence among those with longest latency ($p<0.01$). 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.<br><br>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. | Dust from work clothes, shoes, hair assumed causal; no changing facilities at factory. |
| Magnani et al. [1993] | Italy<br><br>Asbestos cement workers | Retrospective cohort mortality study of 1964 wives of asbestos cement workers; cohort had no history of occupational exposure. Husbands employed 1950-1985; deaths occurred 1965-1988. | Between 1965-1988, there were 4 pleural tumors (1 mesothelioma) obs. vs. 0.5 exp.; 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). | 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. |

Table 1. (Continued) Studies of Take-Home Asbestos Exposure (Cohort Studies)

| Author (year) | Location<br>Industry/<br>Population at Risk | Study Design | Results | Comments |
|---|---|---|---|---|
| Joubert et al.<br>[1991] | New Jersey<br><br>Amosite asbestos workers | Cohort study.<br>Followed household contacts of amosite asbestos workers employed at a single facility 1941-1954. Of 4044 household contacts, 878 were examined 1973-1976. | Vital status follow-up through January 1990 indicates that 28% died of lung cancer, 23% died of digestive cancer, and 9% died of mesothelioma. The authors state that cancer deaths were 2 times expected based on national estimates. | Some figures reported in the paper seem contradictory.<br><br>This appears to be additional follow up of Selikoff study. |
| Navratil et al.<br>[1972] | Czechoslovakia<br><br>Chrysotile asbestos product processing | cohort<br>Group1<br>800 workmen employed for more than 10 years at Factory A.<br><br>Group2<br>155 persons living in the neighborhood of the factory.<br><br>Group3<br>114 persons who were blood relations of factory employees, older than 20 years.<br><br>Group4<br>8133 persons over the age of 40 who lived in the same district as the factory but not in the same neighborhood as the factory. | Each group was evaluated by xray for the prevalence of pleural calcifications, with or without other signs of asbestosis.<br><br>Group1<br>42 persons (5.3%)<br><br>Group2<br>9 persons (5.8%)<br><br>Group3<br>4 persons (3.5%)<br><br>Group4<br>36 persons (0.44%)<br><br>Expected cases (based on the general population) was:<br>Group1<br>2.75 (not42)<br><br>Group2<br>0.53 (not9)<br><br>Group3<br>0.39 (not4) | In group 2, 5 were also blood relatives but were counted in group2 rather than group3.<br><br>Blood relations were assumed to have increased exposure due to contact with workers wearing contaminated work clothes. In group4, 8 of the cases were later discovered to have a history of occupational asbestos exposure. Subtracting those cases changed the prevalence in group4 to 28 cases (0.34%). |

Table 2. Studies of Take-Home Asbestos Exposure (Case-Control Studies)

| Author (year) | Location | Industry/ Population at Risk | Study Design | Results | Comments |
|---|---|---|---|---|---|
| Newhouse and Thompson [1965] | London, England | Hospital-based | 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. | 9/76 cases (7 female; 2 male) reported domestic exposure compared with 1/76 controls from the inpatient series. | Most female exposures from laundering work clothes; 2 males exposed in childhood to family members who worked in asbestos factory; latency ranged from 16-55 years. |
| Vianna and Polan [1978] | New York State | Population-based | 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. | Relative Risk from matched pairs analysis for domestic exposure (included 2 with occupational exposure) reported as 10 (95% CI=1.4-37.4).<br><br>Analysis on subset of 46 non-occupationally exposed cases: 8/46 reported domestic exposure vs. 1/46 controls (p=0.02). | All 10 domestic cases exposed during hand-laundering of work clothes. |

Table 2. (Continued) Studies of Take-Home Asbestos Exposure (Case-Control Studies)

| Author (year) | Location | Industry/ Population at Risk | Study Design | Results | Comments |
|---|---|---|---|---|---|
| McDonald and McDonald [1980] | Canada and USA | Population-based autopsy series [Canada 1960-1972: USA 1972] | 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 para-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. | 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). | Among males with no occupational exposure, 5/156 cases vs. 2/156 controls reported exposure from hobbies or home projects. 5/8 para-occupational cases were exposed in childhood. 3 cases and one control were exposed to clothing of a chrysotile production worker; 5 cases and 1 control were exposed to contaminated clothing of insulation of factory workers. |
| Whitwell et al. [1977] | England | Hospital-based | 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. | 1 mesothelioma reported associated with para-occupational exposure. Although not explicitly stated in published report, apparently no cases associated with para-occupational exposure were observed in either control series. | Father worked in gas mask production and brought work home. |

Table 2. (Continued) Studies of Take-Home Asbestos Exposure (Case-Control Studies)

| Author (year) | Location | Industry/ Population at Risk | Study Design | Results | Comments |
|---|---|---|---|---|---|
| McEwen et al. [1971] | Scotland | Population-based | 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. | No statistically significant differences between cases and controls with respect to para-occupational exposure. | The number of para-occupational cases was not reported in the publication. |
| Ashcroft [1970] | Britain | Shipbuilding (asbestos) | Case-control. 23 cases of mesothelioma (20 pleura, 3 peritoneum) 19 males, 4 females. 46 hospital controls matched on sex and age, free from malignant disease controls. | 91% of the cases had a history of exposure to asbestos.<br><br>41% of the controls had a history of exposure to asbestos. 19 were exposed at work (described as trivial exposure for 4). | One patient was a widow of an asbestos worker. She was exposed for 3 years to the asbestos dust brought home on her husband's hair and shoes. |

Table 3. Studies of Take-Home Asbestos Exposure (Community Studies)

| Author (year) | Location/ Industry/ Population at Risk | Study Design | Results | Comments |
|---|---|---|---|---|
| Kilburn et al. [1985, 1986] | Los Angeles County<br><br>Shipyard workers | 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. | 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. | Possible selection bias resulting from volunteer study participants. No difference in prevalence observed by smoking status. Most shipyard workers had indirect (bystander) exposure.<br>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 familiar asbestosis occurred in families of insulators.) |

Table 4. Studies of Take-Home Asbestos Exposure (Case Reports)

| AUTHOR(S) (YEAR) COUNTRY | INDUSTRY | HEALTH EFFECT | #/RELATIONSHIP | AGE(S) AT DEATH | COMMENTS/ISSUES |
|---|---|---|---|---|---|
| Rusby [1968] England | Asbestos Related | Pleural changes Meso-thelioma | 5 unrelated cases - 4 males & 1 female | 50,54, 55,59,67 | Cases include both direct and indirect exposures. Asbestos bodies present in some cases. |
| Teyssier and Lesobre [1968] | | Asbestosis/asbestos | Case report of asbestosis in a man exposed as a teen to his father's work clothes worn home from an asbestos plant. | | Paper in French. This man was a railway worker. |
| Champion [1971] Canada | Asbestos | Meso-thelioma | 2 unrelated males | 31 and 32 | Father of case 1 (pipe laggar) had asbestosis. Son had never had occupational exposure. Case 2 lived in Asbestos, Quebec and worked as an asbestos prospector for 10 years. |
| Knappmann [1972] West Germany | Asbestos | Meso-thelioma | 4 case reports out of 251 cases diagnosed in pathology departments of the Hamburg central hospitals. | Age at death is as in bronchogenic cancer. | None of tumors remained restricted to site of origin. The proportion with distant metastases was surprisingly high at 70 percent. Four typical case reports are quoted with long latency periods. |
| Lillington et al. [1974] USA | Asbestos | Familial Meso-thelioma | 1 wife | 52 | Domestic case resulting from residence in the same house as an asbestos worker. |
| Li et al. [1978] USA | Asbestos | Familial Meso-thelioma | 1 father, 1 mother, and 3 daughters | 71,51,34,30, and 23 | Father was shipyard worker who had asbestosis and died of lung cancer. His wife and eldest daughter died of meso-thelioma. Dusty work clothes were implicated. |
| Epler et al. [1980] USA | Asbestos | Pleural changes Meso-thelioma | 2 wives of asbestos workers 2 brothers | 60,56, 33 and 27 | Wives were involved with cleaning husbands' clothes resulting in mesothelioma in one and pleural changes in the other. Brothers played as children in room used as muffler shop. Both developed pleural changes in young adulthood. |
| Risberg et al. [1980] USA | Asbestos (building industry) | Familial Meso-thelioma | Father, 2 brothers and 1 sister | 61,71, 60, and 52 | Heredity may be important predisposing factor in this case. All cases smoked. Father died of peritoneal mesothelioma. The two sons died of tubulo-papillary mesothelioma. Sister was similar to eldest son. |

Table 4. (Continued) Studies of Take-Home Asbestos Exposure (Case Reports)

| | | | | | |
|---|---|---|---|---|---|
| Joergensen [1981] Denmark | Asbestos (insulation work) | Pleural plaques | 3 wives with indirect exposure | 71,54 and 58. | These three cases had no obvious symptoms. Exposure was limited to laundering of clothes, dusty shoes, etc. Two of these women smoked. One case had a father and five brothers in insulation business. |
| Martensson et al. [1984b] Sweden | Asbestos (foundry) | Familial Meso-thelioma | Sister and brother and identical twin brothers | 52,58, 44 and 43. | There may be a genetic factor involved. Tenure was only 8 years in the case of the twins. Three of the four are dead, and the sister survived. The twins had occupational exposure, and the others did not. |
| Krousel et al. (1986) USA | Asbestos | Familial pleural meso-thelioma | Three first-degree relatives, mother, son and daughter. | 74, 40, and 35. | Again there is a question of a predisposing genetic factor. Son was only one with possible direct exposure. Other exposure was from father who worked at lumber and shingle company. Both daughter and son died young. |
| Magee et al. [1986] Canada | Tremolite asbestos | Meso-thelioma | Single case | 41 | Indirect exposure to asbestos from Carni Mine in Corsica. Exposed as child in Pub in house. Analysis of lung mineral content showed elevated levels of tremolite and actinolite asbestos. Issue of fiber size and mesothelial carcinogenesis discussed. |
| Huncharek et al. [1989] USA | Asbestos | Pleural meso-thelioma | Single case (female) | 67 | Indirect exposure by exposure to husband, a shipyard mechanic, who dismantled boilers and other related machinery for 34 years. She laundered his clothes. |
| Li et al. [1989] USA | Asbestos | Familial Meso-thelioma | Family cluster of four. Father worked in asbestos plant. | 32, 53, 49, 43 | Cotton cloth sacks in which molded asbestos insulation had been transported had been used to make diapers for children. Results were the deaths of the mother, one sister, and a young uncle who lived there. In addition, the father died of asbestosis. |
| Otte et al. [1990] Denmark | Asbestos cement | Familial Meso-thelioma | Family cluster of six (three deceased). | 74, 79, 45. | Family produced asbestos cement in their home. Used dry hand mixing procedure. Mother, Father and one son died of meso-thelioma. Two sons and daughter survived. All decedents smoked. |
| Oern et al. [1991] Norway | Asbestos (various) | Familial asbestos related disease | Family cluster of four. Both direct and indirect exposure | 84,79 68, 76. | Family made up of two brothers, a sister and her husband. All males were asbestosis insulators and two were smokers. Oldest brother (alive) has asbestosis. Second brother and sister died from mesothelioma. Brother-in-law died from cancer bronchiale. |

Table 4. (Continued) Studies of Take-Home Asbestos Exposure (Case Reports)

| | | | | | |
|---|---|---|---|---|---|
| Sword [1993] England | Asbestos | Pleural plaques | Three daughters | 63,62<br>60 | Daughters of pipe laggar. Exposure was laundering of work clothes. Two younger daughters were asymptomatic but all three had varying degrees of pleural plaques. Father died of peritoneal mesothelioma. |
| Stapleton [1993] USA | Asbestos | Asbestos disease? | Wife | Unknown | Note in Bits & Pieces about indirect exposure and suggestion that one contact the Asbestos Trust Fund if similarly exposed. |

Table 5. Studies of Take-Home Asbestos Exposure (Case Series)

| AUTHOR(S) YEAR COUNTRY | INDUSTRY | HEALTH EFFECT | #/RELATIONSHIP | AGE@DEATH EXPOSURE | COMMENTS/ ISSUES |
|---|---|---|---|---|---|
| Newhouse et al. [1965] England | Asbestos (Factory work, domestic, laggers and insulators, living in neighborhood of factory) | Mesothelioma,27 peritoneal & 56 pleural. | Eighty-three cases of mesothelioma confirmed by necropsy or biopsy. There were 41 males and 42 females. | Youngest dead at 33. Seven deaths occurred at 65 or older. | This is a complex study covering both direct and indirect exposure. There is evidence that neighborhood exposure may be important (living within a half mile of asbestos factory). Contains table of evidence of asbestos bodies. |
| Lieben et al. [1967] USA | Asbestos (textiles, insulation, tile, boiler maker, bakery oven builder, etc.) | Mesothelioma, 34 pleural and 8 peritoneal. | 42 cases of mesotheliomas reported from 152 hospitals over a five-year period. | Various ages. No numerical exposure data given. | Ten patients worked in asbestos plants, eight lived or worked close to an asbestos plant, three patients were family members of asbestos workers. In ten patients a history of assumed exposure to asbestos was obtained after close questioning. In 11 patients no history of asbestos exposure was obtained. |
| Ashcroft et al. [1970] England | Asbestos (Shipbuilding) | Mesothelioma of pleura or peritoneum. | 23 cases which came to necropsy. | Age range: 43 to 76. All but one case direct exposure to asbestos. | Case control study (2 controls per case). Has tables of fiber and asbestos body counts. Association between mesothelioma, asbestos bodies in the lungs and occupational exposure to asbestos is confirmed in a British shipbuilding area. One patient was exposed to dusty work clothes at home. The incidence of mesothelioma was 91% in the case population vs. 41% in the controls. |
| Dalquen [1970] Germany | Asbestos | Pleural plaques, Asbestosis | 133 cases of pleural plaques and 145 cases of asbestosis from the Hamburg area. | ? | Occupational and endemic exposure to asbestos seem to be the main cause of pleural plaques. Of 92 cases with dust anamnesis, 34 were occupationally exposed, 22 domestically, 21 by urban dwelling and 10 by multiple cause. The latency period for plaques is 40.2 years. |
| Heller et al. [1970] USA | Asbestos | Mesothelioma | Radiological review of x-rays of ten people diagnosed with malignant pleural mesothelioma. | Age range; 34-74. Eight male and two female. Six exposed to asbestos. | This is a technical discussion of evaluating x-rays for malignant pleural mesothelioma. It is basically intended as training for the radiologist. |

Table 5. (Continued) Studies of Take-Home Asbestos Exposure (Case Series)

| | | | | | |
|---|---|---|---|---|---|
| Bittersohl et al. [1971] Germany | Asbestos (insulating and products such as cords, seals, plates, etc.) | Mesothelioma, pleural | 26 cases of pleural mesotheliomas from chemical plants of the district of Merseburg, East Germany. | Age range: 34-65+. Exposure data given in terms of East German standard. | This group is made up of 22 patients from the Leuna Chemical combine, 2 patients from the Beuna Chemical Combine, and one patient from a metal foundry. All had been exposed to dust levels exceeding their standard.. This group includes workers not directly working with asbestos, but working near-by. Forty-six percent of the cases occurred after retirement. |
| Vianna et al. [1971] | Six New York counties | Mesothelioma | Of 31 cases, 17 were occupationally exposed; 7 reported paraoccupational exposure; 7 had no known exposure to asbestos. 7/14 cases with no occupational exposure history reported domestic exposure. | | Population-based incidence study (case series). Descriptive survey of 31 (22 male; 9 female) histologically confirmed mesothelioma diagnosed 1973-1978; occupational histories from cases or relative. No control group. |
| Grundy et al. [1972] USA | N/A | Childhood Mesothelioma | 13 cases of childhood mesothelioma in US children. | Age range: 4 through 17. | Death certificate search. Asbestos may not be a factor in childhood mesothelioma. Latency is short in children compared to adults. Need more environmental data. |
| Rubino et al. [1972] Italy (Piedmont) | Various industries | Pleural Meso-thelioma | 54 cases of pleural mesothelioma admitted either to the Chest Surgery Center or Department of Medicine at U. of Turin. | Age range: 33 to 75. | Occupational exposure was unequivocally demonstrated in five men. Three other patients (two men and one women) had lived with persons employed in the asbestos industry. Asbestos bodies were found in only one individual. Piedmont produces only chrysotile. Most research implicates crocidolite as causing pleural mesothelioma. |
| Greenberg and Davies [1974] | England/Wales | Mesothelioma/ Asbestos | | Reported 2 female paraoccupational mesotheliomas associated with household exposure of 2 and 3 years duration. One husband worked in asbestos factory, and one was exposed to brother's work clothes. | 1967-1968 mesothelioma case series (n=413: 365 pleural; 48 peritoneal) from population-based registry. Asbestos exposure history obtained by interview from cases, relatives, employers and workmates. For 246 of the 413 cases, the diagnosis was histologically confirmed. |

Table 5. (Continued) Studies of Take-Home Asbestos Exposure (Case Series)

| | | | | | |
|---|---|---|---|---|---|
| Milne [1976] Australia | Asbestos (textiles, welding rods, lagging, boiler repair, asbestos/cement, etc.) | Mesothelioma, 29 pleural and 3 peritoneal | Retrospective survey of 32 cases of mesothelioma in Victoria, Australia. | Age range: Not given. Exposures: Six months to 30 years. | Confirms relationship between occupational cancer and development of mesothelioma (elapsed time about 20 years). Occupational history is equally effective as an asbestos body count to indicate past exposure. In 16% of cases, there was no evidence of exposure to asbestos. Two cases of peritoneal cancer were in siblings without asbestos exposure. |
| Edge et al. [1978] England | Asbestos (shipyard workers) | Malignant pleural meso-thelioma | 47 men and 3 women. All men plus one woman occupationally exposed. One woman exposed at home, the other had no known exposure. | Age at diagnosis ranged between 32 and 74. | Fifty cases of pleural mesothelioma histologically proved and accepted by Pneumoconiosis Panel. One woman married to shipyard plumber who may have brought dust home on clothes. Asbestos content of last 20 cases was measured. Eighteen cases substantially exceeded that of the general population. Metastases, frequent at necropsy, occurred in 25 of 47 cases. |
| Bianchi et al. [1982] Trieste, Italy | Asbestos (various shipyard trades) | Pleural Mesothelioma | 64 men and 6 women. Forty-three patients employed in shipyards, most prior to 1940. | Age at death: 40 to 83. | Seventy cases seen at Institute of Pathological Anatomy of Trieste. Necropsy findings available in 63 cases. Remaining seven cases were diagnosed at thoracotomy. Intervals between first exposure and death ranged from 28 to 61 years. One case was due to probable domestic exposure. Asbestos bodies found in 48 of 61 cases. |
| Bianchi et al. [1987] Italy | Asbestos (shipyard, sodium carbonate factory) | Hyaline Pleura Plaques | 74 women with hyaline pleural plaques found at necropsy. | Not stated | 59 cases were attributed to domestic exposure (laundering asbestos-contaminated work clothes of family members)<br>2 cases with occupational exposure<br>9 cases with occupational and domestic exposure.<br>Sufficient exposure data could not be obtained on 4 cases.<br>Pleural malignant mesothelioma was noted in 2 cases with a history of household exposure. |
| Bianchi et al. [1990] Italy | Shipyard (asbestos) | Hyaline Pleural Plaques | 1620 necropsies (1040 men, 580 women) were performed from Oct 1979 to Dec. 1987 in Monfalcone, Italy.<br><br>121 women with history of domestic exposure were compared to 57 women with no history of domestic exposure. | | The prevalence of hyaline plaques was higher in every occupational category for women with domestic exposure than for women without domestic exposure. |

Table 5. (Continued) Studies of Take-Home Asbestos Exposure (Case Series)

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|---|---|---|---|---|---|
| Bianchi et al. [1991] Italy | Shipbuilding, sodium carbonate factory | Asbestos bodies hyaline pleural plaques | 1765 necropsies<br><br>1127 men<br>638 women | | 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.<br><br>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.Only 21 of the 638 necropsies on females had a history of occupational exposure. |
| Bianchi et al. [1993] Italy | Asbestos (shipyard, seamen, insulation workers) | Mesothelioma | 92 malignant mesotheliomas were diagnosed between Oct. 1979 and April 1992 at Monfalcone Hospital. | Age at diagnosis 42 to 89 years (median 68 years). | 75 cases had occupational history of exposure to asbestos. 5 women had history of domestic exposure (laundering asbestos-contaminated clothing of family members). One case had a history of probable environmental exposure. |
| Lander et al. [1988] Denmark | Asbestos work | Pleural plaques, pleural calcifications, pulmonary fibrosis, asbestosis | 63 women (spouses of workers exposed to asbestos) with indirect (nonoccupational) exposure to asbestos. Of the 63 women, 9 (17%) had radiological changes characteristic of exposure to asbestos. | Adult women | It was attempted to enroll 125 spouses. 90 participated in the study. 20 were excluded due to lack of exposure (Xrays were normal). 5 were excluded due to occupational exposure ( one had pleural plaques). 2 were excluded for other pulmonary diseases.<br>63 were left in the study, exposure consisting of laundering asbestos-contaminated work clothes. |
| Lander et al. [1985] Denmark | Asbestos (unknown) | Benign asbestotic pulmonary changes. | 63 women whose husbands had been exposed to asbestos dust. | Not given | Out of a group of 63 women who husbands had been occupationally exposed to asbestos dust, 17% were found to have radiologically benign asbestotic pulmonary changes. This exposure was caused by cleaning work clothes which had been contaminated with asbestos dust. |
| Gibbs et al. [1989] Wales | Asbestos | Pleural meso-thelioma | 84 cases chosen because the history of asbestos exposure was absent, indirect, or ill-defined. | Used Zielhuis age grouping. | Three purposes of study were: 1. correlate lung mineral count with Zielhuis groupings; 2. determine whether any mesotheliomas were unrelated to asbestos exposure; and 3. compare the role of amphiboles and chrysotile in causation. Results indicate: 1. Zielhius method too complex; 2. mesotheliomas may develop in absence of asbestos exposure; and 3. amphiboles are more important than chrysotile. |

Table 5. (Continued) Studies of Take-Home Asbestos Exposure (Case Series)

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|---|---|---|---|---|---|
| Gibbs et al. [1990] Wales | Asbestos | Malignant mesothelioma | 10 paraoccupational cases, nine of whom were spouses of asbestos workers. one was daughter of gas-mask factory worker. | Age range: 47 to 72. | This was a comparison of types of lung fibers and size distribution in a series of paraoccupational cases of mesothelioma with a series of known occupational exposure in female gas mask workers. Gas mask workers showed consistently high crocidolite concentrations. The paraoccupational group was variable; six showed high crocidolite; seven showed high amosite; and two showed normal for all fiber (several showed more than one high fiber group). |
| Konetzke et al. [1990] Germany | Asbestos | Pleural plaques Mesothelioma | 48 cases of meso-thelioma and 19 cases of pleural plaque. | Not given in summary | Confirmed reports that even in the non-occupational area, asbestos represents a non-negligible risk for diseases of the lung. Some figures from this study; laundering (46%); use of asbestos containing materials in the house (20.9%); and in connection with leisure activities (14.9%). |
| Browne [1991] England | User asbestos industry | Environmental | Letter to editor of THE LANCET. | N/A | Letter pointing out that the end user of asbestos materials may be more severely compromised than the primary manufacturer (5.5% of cases were in primary sector). Also points out the problem with para-occupational exposure. |
| Kiviluoto [1965] Finland | Asbestos | Pleural plaques, pleural adhesions, pulmonary fibrosis, mesothelioma. | 4 cases of asbestoses in 4 sisters whose father had been occupationally exposed to mixed dusts. | Childhood | The father 50 years earlier had been occupationally exposed to mixed dusts and presumably bought it home on his clothes. |
| Martensson et al. [1984a] | Asbestos work | Mesothelioma | Analysis of 32 cases of malignant mesothelioma. All but one case was occupational. | Childhood | One case of a woman who had been exposed to asbestos during childhood via her father's work clothes. |
| Sider et al. [1987] U.S.A. | Insulation work | Radiographic pleural changes (plaques, calcification, thickening) | 117 wives of asbestos-exposed insulation workers were screened with xrays and PFT's. | Of the 93 women, the year of initial exposure for those with abnormal photographs was 1952. The year of initial exposure for those with normal xrays was 1958. | None of the 24 women under age 40 had any xray abnormalities . Exposure for all of them was less than 8 years. These 24 were excluded.<br><br>18 (19.4%) of the remaining 93 had radiographic abnormalities.Only the year of initial exposure was significant. |

Table 5. (Continued) Studies of Take-Home Asbestos Exposure (Case Series)

| | | | | | |
|---|---|---|---|---|---|
| Giarelli et al. [1992] Italy | Asbestos (shipyard, navy, merchant marine, docks) | Mesothelioma | 170 pleural mesotheliomas examined at necropsy between 1968-1987 (Trieste University). | Age at death of all cases 33 to 92 years (median 70 years). | 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 (laundering the asbestos-contaminated clothing of family members. |

Table 6. Studies of Take-Home Asbestos Exposure (Health Hazard Evaluations)

| HETA NUMBER AUTHOR | COMPANY AND LOCATION | HAZARD | RESULTS AND RECOMMENDATIONS (ASBESTOS RELATED) |
|---|---|---|---|
| 78-73-612<br>Belanger et al.<br>[1979] | Kentile Floors, Inc.<br>Chicago, IL<br><br>Floor tile<br><br>50 "affected workers" | Exposure to asbestos, PNAs, vinyl chloride monomer and other organic and inorganic substances. | Asbestos was the only contaminant found to exceed a NIOSH recommended criteria. Eight of thirty asbestos samples exceeded the criteria of 10 fibers/cc of air. No cases of asbestosis were found in present or past employees of this plant. Only one out of three cases of cancer could be regarded as being of industrial origin and it could not be definitely traced to exposure at this plant.<br><br>All work clothes of men potentially exposed to asbestos should be collected each day and placed in plastic bags for proper cleaning. Under no circumstances should they be taken home. |
| 84-262-1734<br>Seixas and Ordin<br>[1986] | Friction Division Products<br>Trenton, NJ<br><br>Auto and truck brake shoes<br><br>120 production workers | Exposure to asbestos and the potential for home contamination. | Results of air sampling and x-ray review indicate a significant risk of asbestos disease in the plant and a potential risk to families of employees. Seven of twenty workers with >10 years at this plant had x-ray findings consistent with asbestos exposure. Two had asbestosis, two had pleural thickening, and one had a pleural plaque. Two had lung cancer.<br><br>Personal air sampling results indicated asbestos TWA exposures in the range of 0.06 to 1.56 f/cc. The OSHA exposure limit is 0.2 f/cc and the NIOSH limit is 0.1 f/cc. This indicates significant asbestos contamination in the plant. In addition, samples taken from work clothes as workers went home indicated the presence of asbestos.<br><br>A whole series of recommendations were made from discouraging smoking to providing clean uniforms, and enforcing respirator use. |
| ECTB 152-20b<br>Godby et al.<br>[1987] | United States Postal Service<br>Vehicle Maintenance Facility<br>Nashville, TN<br><br>Garage facility for fleet of 575 mail service vehicles serviced by 9 mechanics, 1 lead mechanic, 4 garagemen and 2 supervisors. | Exposure to asbestos when servicing and replacing brake shoes. | This paper is an evaluation of brake drum service controls within a major US Postal System vehicle service center. Exposure data suggest that the present technique was substantially effective in controlling asbestos dust during brake servicing. Suggestions are made about personal hygiene and not taking dirty work clothes home. |
| 87-126-2019<br>Driscoll and Elliott [1990] | Chrysler Chemical Division<br>Trenton, MI<br><br>Friction products and adhesives for auto manufacture.<br><br>138 hourly workers | Exposures to asbestos, lead and solvents. | NIOSH investigators concluded that a health hazard did exist at this plant based on observation of existing conditions and results of environmental sampling.<br><br>Recommendations made to improve conditions at this plant included the use of supplied-air respirators, use of company supplied work clothes, establishment of a decontamination area, improved industrial hygiene monitoring, and the use of engineering controls. |

Table 7. Studies of Take-Home Asbestos Exposure (Miscellaneous Reports)

| AUTHOR (YEAR) COUNTRY | INDUSTRY | HEALTH EFFECT | #/RELATIONSHIP | EXPOSURE | COMMENTS/ISSUES |
|---|---|---|---|---|---|
| Lemen [1972] USA | Asbestos Fireproof insulation | Asbestos exposure while spraying. | 4 men, a mixer-pump operator, two sprayers, and a hose handler. | All samples were below emergency standard of 5.0 fibers/ cc> 5u. | Mixer-pump operator had highest exposure to spray of 3.00 fibers/cc> 5u. He was supplied with a respirator which he did not use. Recommendations included respirators for each man, company provides clothing and laundrying which is not worn home, that other men in the area be protected, and that a asbestos free material be substituted in the fireproofing. |
| House of Repre-entatives [1979] USA | Asbestos Oversight Hearings | Hazards to school children. | School children in New Jersey, Kentucky and other places. | Asbestos coated walls and ceilings which had begun to flake. | These are the minutes of a hearing in the US House of Representatives concerning exposure of schoolchildren to asbestos fibers. The purpose was to establish a program for inspection of schools for the presence of asbestos materials, to provide funds for the testing and evaluation of potential hazards, to create a loan program to assist in the containment or removal of imminent hazards to health and safety, and for other purposes. |
| Rom et al. [1982] (review of literature from various countries) | Asbestos work | Mesothelioma | N/A | Occupational, environmental, and household contact discussed. | This article is a review of mesothelioma. In discussing household contact with asbestos and subsequent mesothelioma, it discusses many of the articles reviewed for the Worker Family Protection Act. |
| Berry [1986] (review of data from various countries) | Asbestos work | Mesothelioma | Not indicated | Household contact | It is mentioned that household contact with exposed workers can result in a mesothelioma. Articles reviewed elsewhere are cited (Newhouse et al. 1965, Anderson et al. 1976. The article is a review of asbestos effects in general. |
| Grandjean et al. [1986] Denmark | Asbestos work, other exposures | Hyaline plaques, asbestosis, mesothelioma | Cites numerous cases of these health effects in household contacts of asbestos workers. | Household contact with asbestos workers. | This article is a review of bystander exposures in general. Eight of the articles discussed deal with bystander exposure to asbestos. The asbestos exposure was felt to stem from exposure to contaminated clothing worn or brought home. |
| Hinze and Hinze [1986] USA | Asbestos | Safe removal of asbestos from buildings. | N/A | N/A | This paper outlines some of the problems concerned with asbestos removal and suggests some recommendations to ameliorate the situation. |
| Beegle and Forslund [1990] USA | Asbestos (former battery plant) | Asbestos related housekeeping activities. | Mulea home plus four other homes. | Houses built on former site of battery plant have asbestos contamination in basement | This is an engineering report giving detailed instructions for sampling asbestos contamination in the homes, and comprehensive cleaning instructions in order to decontaminate the five houses. |

Table 8. Studies of Take-Home Lead Exposure (Cohort Studies)

| Author (year) | Location | Industry | Study Design | Results | Comments |
|---|---|---|---|---|---|
| Baker et al. [1977] | Tennessee, USA | Lead smelting | Cohort<br>20 exposed Children<br>17 neighborhood controls | Mean BLL significantly higher in exposed children; higher dust lead levels in exposed houses (2,687 vs. 404 ppm); children's BLLs correlated with dust levels. | Matched on neighborhood and measured lead content in paint by x-ray fluorescence. Earlier report in MMWR 1976 25:85. |
| Elwood et al. [1977] | England | Battery plant | Cohort<br>192 exposed children<br>273 children from birth registry | Workers' children had significantly higher BLLs than registry children (mean 33 vs. 27 $\mu$g/dL). | Used capillary sampling; 3-year-olds had highest BLLs. |
| Koplan et al. [1977] | Barbados | Pottery | Cohort<br>12 potters<br>19 family members<br>24 controls | Mean BLL of potters' family members (35 $\mu$g/dL) was significantly higher than that of controls (17-19 $\mu$g/dL). | Homes were adjacent to potteries |
| Watson et al. [1978] | Vermont, USA | Battery plant | Cohort<br>27 exposed children<br>32 neighborhood controls | 56% of exposed vs. 12.5% of controls had BLL $\geq$ 30 $\mu$g/dL; higher mean dust lead levels in exposed houses (2,239 ppm) vs. controls (718 ppm). | Used capillary sampling. Earlier report in MMWR 1977; 26:61. |
| Millar [1978] | England | Lead smelting/refining | Cohort<br>71 children of workers<br>191 community children (living near plant) | Difference in BLLs between workers' children (21.1 $\mu$g/dL) and community children (18.2 $\mu$g/dL) was statistically significant for children age $\leq$ 10. | |
| Rice et al. [1978] | USA (city unspecified) | Secondary lead smelter | Cohort<br>33 exposed homes<br>19 neighborhood homes | 7 exposed children had ZPPs over 50 $\mu$g/dL compared to 1 control child. Significantly higher lead levels were found in wipe and dust samples from exposed homes compared to control homes. | No BLLs were measured. |
| Abbritti et al. [1979] | Italy | Ceramics | Cohort<br>40 children of ceramic workers<br>47 children of ceramic workers who work at home<br>89 unexposed children in community | Mean BLLs for the three groups: 25.1, 27.5, 23.0 (p-value for difference between the latter two < 0.001). | Many of the pottery factories were home-operated. |
| Landrigan et al. [1980] | Georgia, USA | Stained glass | Cohort<br>12 workers<br>5 hobbyists<br>4 workers' family members | Mean BLLs were 20.7 $\mu$g/dL for workers, 11.6 $\mu$g/dL for hobbyists and 11.3 $\mu$g/dL for family members of workers. BLL was associated with duration of work and percentage of work involving lead. | |

Table 8. Continued Studies of Take-Home Lead Exposure (Cohort Studies)

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|---|---|---|---|---|---|
| Molina-Ballesteros et al. [1980] | Mexico | Pottery | Cohort<br>198 workers and their families<br>187 controls and their families | Children aged < 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. | |
| Morton et al. [1982] | Oklahoma, USA | Battery factory | Cohort<br>34 exposed children (age < 7)<br>34 age-matched neighborhood control children | Significantly different BLLs were found between groups p<0.001). BLL > 30 μg/dL found in 53% of exposed children versus 0% in controls. Statistically significant differences found in children's BLL between good and poor worker hygiene practices. | Used capillary sampling. |
| Milar and Mushak [1982] | North Carolina, USA | Battery factory | Cohort<br>17 exposed children (age < 5)<br>30 control children (age < 5) | Average BLL of exposed children was 44 μg/dL and of control children, 18 μg/dL. | |
| Ramakrishna et al. [1982] | Sri Lanka | Gold and silver recovery | Cohort<br>33 members of exposed families<br>21 neighborhood controls | 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 2 years (42 and 56 μg/dL). | The youngest child tested was 9 years old. |
| Molina-Ballesteros et al. [1983] | Mexico | Pottery manufacturing | Cohort<br>153 children (age 5-15) from pottery-making families<br>80 control children from local schools | 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. | |
| Katagiri et al. [1983] | Japan | Pottery manufacturing | Cohort<br>89 3-yr olds from homes where pottery made<br>70 3-yr olds from homes where parent works in pottery factory<br>947 3-yr olds from homes where no one works in pottery<br>768 3-yr old controls | 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). | Urine samples are questionable in evaluating lead exposure. |
| Richter et al. [1985] | Israel | Battery factory | Cohort<br>18 exposed children<br>729 control children | Among exposed children > 10 years old, ZPP > 40 μg/dL was 4.1 times higher and among exposed children < 10, ZPP > 40 μg/dL was 2.9 times higher than controls. | No BLLs were measured. Elevated ZPP can also be influenced by iron deficiency. |

Table 8. Continued Studies of Take-Home Lead Exposure (Cohort Studies)

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|---|---|---|---|---|---|
| Piccinini et al. [1986] | Italy | Ceramic tile | Cohort<br>22 children of tile workers exposed to lead<br>27 children of tile workers not exposed to lead<br>24 control children | Children in group 1 had a mean BLL of 13.5 $\mu$g/dL compared with group 2 mean of 112.2 and group 3 mean of 10.7. Hair lead levels for the 3 groups were 17.0, 9.8 and 7.8 respectively. | Used capillary sampling. No sex differences found. |
| Kaye et al. [1987] | Colorado, USA | Electrical components plant | Cohort<br>89 exposed family members<br>62 clinic controls | Exposed family members had significantly higher mean BLL (10.2 $\mu$g/dL) compared to unexposed (6.2 $\mu$g/dL). | Also reported in MMWR 1985; 34:384. |
| Abbritti et al. [1988] | Italy | Ceramic pottery factories | Cohort<br>136 exposed children<br>199 community children | Exposed children had higher mean BLL (10.7 $\mu$g/dL) compared to community children (9 $\mu$g/dL) ($p<0.05$). | Many of the pottery factories were home-operated. No differences found by age or sex of child. |
| Wang et al. [1989] | Taiwan | Multiple | Cohort<br>105 newborns of lead workers<br>102 nonexposed newborns | Mean cord BLL of exposed newborns was significantly higher (8.8 $\mu$g/dL) than mean cord BLL of unexposed newborns (6.9 $\mu$g/dL). | Paternal contribution to cord BLL appears to be through either working at home (n=12 fathers) or bringing lead dust home and exposing mother. |
| Matte et al. [1989] | Jamaica | Battery repair | Cohort<br>24 exposed households (112 workers and family members)<br>18 neighborhood control households (74 family members) | Geometric mean BLLs were significantly higher among exposed households compared to controls. 43% of exposed children aged < 12 years had BLL greater than 70 $\mu$g/dL. | These were "backyard" battery repair shops. Also reported in NIOSH HETA 87-371-1989. |
| MMWR [1989a] | Jamaica | Battery repair | Cohort<br>17 exposed households<br>18 neighborhood controls | All exposed children aged 0-5 had BLLs $\geq$ 25 $\mu$g/dL. | |
| Gittleman et al. [1994] | Alabama, USA | Battery reclamation | Cohort<br>16 children of 11 workers<br>7 neighborhood control families<br>(5 children age 6-17, 11 adults) | Exposed children had higher mean BLL (22.4 $\mu$g/dL) compared to controls (9.8 $\mu$g/dL). 75% of workers' children had BLLs $\geq$ 10 $\mu$g/dL compared with 40% of control children. | Also reported in NIOSH HETA 91-213-2123 and MMWR 1992; 41:301. |

TABLE 9. Studies of Take-Home Lead Exposure (Community Studies)

| Author (year) | Location | Industry | Study Design | Results | Comments |
|---|---|---|---|---|---|
| Martin et al. [1974] | England | Lead factory | Community screening<br>39 children < age 5 living within 400 m of factory<br>80 children living 400-500 m from factory<br>252 children at local schools | Of 4 children < age 5 with highest BLLs, 3 with levels of 75, 74, and 65 $\mu$g/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). | |
| Landrigan and Baker [1981] | Texas, USA | Ore smelting | Community survey<br>3 households in survey include smelter workers | No children in worker households had BLL $\geq$40 $\mu$g/dL. | |
| Ewers et al. [1982] | Germany | Lead smelting | Community survey<br>302 exposed children<br>86 children in control area | Children of lead workers had higher BLLs than other children (geometric mean = 19.7 vs. 14.2 $\mu$g/dL; $p < .05$). Higher tooth lead levels were associated with father's occupational exposure to lead. | Capillary sampling; blood samples were collected from only a sample of children (n=83). |
| Carvalho et al. [1984] | Brazil | Lead smelting | Community survey<br>104 children (age 1-9) of lead workers<br>357 children (age 1-9) of non-lead workers | Exposed children had a significantly higher mean BLL (67.5 $\mu$g/dL) than unexposed children (56.6 $\mu$g/dL). | Results originally reported in an unpublished thesis by Carvalho (1982). |
| Chenard et al. [1987] | Canada | Copper smelting | Community survey<br>128 children<br>Group 1 (35) exposed through residence and father's work<br>Group 2 (63) exposed through residence only<br>Group 3 (30) exposed through father's work only<br>189 control children from nearby community | All exposed children had significantly higher BLLs than control children. BLL ratios of exposed groups 1, 2, and 3 to nonexposed were 1.83, 1.79, and 1.23 respectively. | Additional sources of lead exposure such as hobbies and home assessment not measured. |
| Brockhaus et al. [1988] | Germany | Lead smelter | Community survey<br>9 children of lead workers (age 4-5)<br>195 control children (age 4-5) | Children of lead workers had significantly higher mean BLL (18.4 $\mu$g/dL) than controls (10.4 $\mu$g/dL). | |

Table 9. Continued Studies of Take-Home Lead Exposure (Community Studies)

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| Silvany-Neto et al. [1989] | Brazil | Lead smelting | Community surveys<br>1980 survey<br>131 children of lead workers<br>457 community children<br><br>1985 survey<br>108 children of lead workers<br>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). | |
| Maravelias et al. [1989] | Greece | Lead smelting | Community survey<br>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. | |
| Hoffstetter et al. [1990] | Germany | Lead and other metal smelting | Community screening<br>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) |
| ATSDR [1991]<br>Philadelphia | Pennsylvania, USA | Lead plant | Community survey<br>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 (27.7%); numbers very small; no results statistically significant. |
| Miesen [1991] | Germany | Metallurgical plant | Community screening<br>491 exposed (19 children < age 6) | Of schoolchildren living with lead-exposed family members, 16.7% had BLLs over 25 μg/dL. | |
| Lyngbye et al. [1991] | Denmark | Multiple lead industries | Community survey<br>101 first grade children with high dentine lead concentrations (above 18.7 μg/g).<br>99 control children with low dentine lead (below 5 μg/g). | A positive association (4-fold relative risk) was found between dentine lead and parental employment as a shipyard worker, welder, auto mechanic or car painter. | Only half of eligible children contributed a tooth for analysis. |
| Schuhmacher et al. [1991] | Spain | Multiple lead industries | Community survey<br>478 exposed children | 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. | No data on blood lead levels. |

Table 9. Continued Studies of Take-Home Lead Exposure (Community Studies)

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|---|---|---|---|---|---|
| Cook et al. [1993] | Colorado, USA | Smelting and mining | Community screening<br>150 children < age 6 | Mean BLL 10.1 μg/dL (range 0.5-30.1 μg/dL).<br>Parental occupation as a miner was an independent predictor of BLL. | |

TABLE 10. Studies of Take-Home Lead Exposure (Case Report/Case Series)

| Author (year) | Location | Industry | Study Design | Results | Comments |
|---|---|---|---|---|---|
| Anonymous [1952] | Philippines | Storage battery factories | Case reports<br>Case 1: 1 lead-exposed worker, 1 child age 3<br><br>Case 2: 1 lead-exposed worker, 1 child age 2.5<br><br>Case 3: 1 lead-exposed worker, 1 child age 2 | Case 1: Symptomatic child was misdiagnosed as a case of poliomyelitis and later died.<br><br>Case 2: Symptomatic child died after 3 days of treatment<br><br>Case 3: Symptomatic child was chelated and recovered. | Workers made storage batteries in or near where the families lived. No BLLs reported. |
| Joshua et al. [1971] | India | Gold and silver recovery | Case report<br>1 family (9 adults, 9 children); 3 generations | BLL levels ranged from 52 to 72 $\mu$g/dL in children and 37 to 61 $\mu$g/dL in adults. | House and work areas were adjacent. |
| Winegar et al. [1977] | Minnesota, USA | Lead smelting | Case series<br>38 workers (87 family members) | Median BLL of workers was 72.5 $\mu$g/dL (range 21-112 $\mu$g/dL) and median BLL of family members was 17 $\mu$g/dL (range 8-44 $\mu$g/dL). Five children under age 10 had BLLs 30 $\mu$g/dL. | |
| Dolcourt et al. [1978] | North Carolina, USA | Battery factory | Case series<br>58 children of unknown number of workers | 40 (69%) of children had BLLs 30 $\mu$g/dL. Levels highest in children age < 3 and statistically significant decline with age. | Used capillary sampling. Also reported in MMWR 1977;26:321. |
| Richter et al. [1980] | Jerusalem | Polyvinyl chloride (PVC) factory | Case series<br>13 workers (6 spouses, 12 children) | Workers' mean BLL was 27.6 $\mu$g/dL. Mean BLL was elevated among children (12.3 $\mu$g/dL) but not among spouses (8 $\mu$g/dL). Mean BLL in 4 children whose fathers showered and changed before leaving work was significantly lower (10.3 $\mu$g/dL) than mean BLL in children whose fathers did not (14.7 $\mu$g/dL). | |

Table 10. Continued Studies of Take-Home Lead Exposure (Case Report/Case Series)

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|---|---|---|---|---|---|
| Dolcourt et al. (1981) | North Carolina, USA | Battery factory | Case report<br>(Family 1)<br>1 worker<br>22 exposed family members<br><br>(Family 2)<br>1 worker<br>6 exposed family members | 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).<br><br>Two children age 7 and 16 months had BLLs of 64 and 63 μg/dL, respectively. | Discarded battery casings were burned as fuel in home. Used capillary sampling.<br><br>Worker was operating illicit battery recycling in home. Used capillary sampling |
| Kawai et al. (1983) | Japan | Cutlery tempering and type printing (at home) | Case series<br>62 family members from 15 exposed households | Children < 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). | |
| Pitts (1986) | Virginia, USA | Radiator repair | Case report<br>1 worker<br>4 children | Radiator worker with BLL of 78μg/dL had children under age 7 with BLLs of 79, 64, 48, and 27 μg/dL. Lead dust found in worker's van, and in house where dirty clothes stored. | |
| Novotny et al. (1987) | Colorado, USA | Firing range | Case series<br>4 workers<br>3 spouses | BLLs levels in workers ranged from 41 to 77 μg/dL. Spouse BLLs ranged from 6 to 11 μg/dL. | |
| Garrettson (1988) | Virginia, USA | Radiator repair | Case report<br>2 workers<br>3 exposed children | Two of the three children had elevated BLL (48 and 79 μg/dL). High levels of lead dust found on father's shoes and in van. | |
| MMWR (1989b) | Colorado, USA | Plaque production | Case report<br>1 worker<br>4 exposed family members | Three children's BLLs ranged from 13 to 37 μg/dL. | |

Table 10. Continued Studies of Take-Home Lead Exposure (Case Report/Case Series)

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|---|---|---|---|---|---|
| Pichette et al. [1989] | Texas, USA | Battery manufacturing and recycling | Case series<br>71 lead-exposed workers<br>101 children (50% under age 6) | 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). | |
| Lussenhop et al. [1989] | Minnesota, USA | Radiator repair | Case series<br>12 workers<br>16 children < age 6 | All but one child had BLLs below 15 μg/dL. Mean BLL was 9.3 μg/dL. | |
| | | | | | |
| Molovich [1991] | Indiana, USA | Welding | Case report<br>1 worker<br>1 child age 4 | Child was reported to have consecutive BLLs of 6.2 and 9.7 μg/dL and was symptomatic. | Family car was contaminated with lead. |
| Fischbein et al. [1992] | USA | Pottery | Case report<br>1 worker<br>2 exposed family members | Worker and her daughter had BLLs of 48 and 54 μg/100ml, respectively. Spouse's BLL was 20 μg/100ml. | |
| MMWR [1992] | Utah, USA | Construction | Case report<br>2 workers (number of family members not reported) | One family BLLs all < 4 μg/dL. Second family had a 7 mo old with BLL of 17 μg/dL. Home inspection revealed no other sources of lead exposure. | |
| State of Alabama [1992] | Alabama, USA | Pottery manufacturing | Case report<br>2 workers (parents)<br>2 children | Children (age 2 and 14 mos) had elevated BLLs (no other data reported) | Pottery shop adjacent to home. |
| Anonymous [1992] | Virginia, USA | Not available | Case report<br>1 child of 2 workers | A one-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. | |
| Nunez et al. [1993] | New York, USA | Radiator repair | Case series<br>7 children of workers | 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. | Over 50% of radiator shops declined to participate. |

Table 10. Continued Studies of Take-Home Lead Exposure (Case Report/Case Series)

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|---|---|---|---|---|---|
| de Silva [1993] | Maryland, USA | Construction | Case report<br>Adult blood lead registry<br>2 children | 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. | |
| Amato [1994] | Virginia, USA | Radiator repair<br><br>Propane tank manufacturing<br><br>Battery manufacturing | Case reports<br>1 worker<br>2 children<br><br>1 worker<br>1 family member<br><br>(number of workers not given)<br>2 children | Children had elevated BLLS (no other data reported)<br><br>Family member had elevated BLL (no other data reported)<br><br>Children had "mildly elevated" BLLs (no other data reported) | |
| Barnett [1994] | Oregon, USA | Bronze foundry | Case report<br>2 children of exposed workers | Two children under 2 years of age had BLLs of 14 and 23 μg/dL. | |
| Czachur et al. [1994] | New Jersey, USA | Construction; Battery manufacturing; General manufacturing | Case series<br>15 workers<br>28 children | 8 children (29%) had BLLs 10-19 μg/dL; highest BLL was 26 μg/dL. | Study was a follow-back of workers with BLLS over 25 μg/dL from adult blood lead registry; 46% response rate. |
| Jung [1994] | Connecticut USA | Painting | Case report<br>1 worker<br>2 children | Children's BLLs were 16 and 19 μg/dL. Worker's BLL was 29.9 μg/dL. | |
| Natarajan [1994] | USA | Radiator repair | Case report<br>1 worker<br>1 child | Child was found to have a BLL of 24 μg/dL. Father had BLLs of 52 and 64 μg/dL. | Worker changed clothes before going home but did not shower. |

Table 10. Continued Studies of Take-Home Lead Exposure (Case Report/Case Series)

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|---|---|---|---|---|---|
| Osorio [1994] | California, USA | Lead recycling/ bullet manufacturing<br><br>Radiator repair<br><br>Cable cutting<br><br>Cable salvage<br><br>Battery repair | Case reports<br>1 worker (2 children)<br><br>1 worker (2 children)<br><br>2 workers (3 children)<br><br>1 worker (1 child)<br><br>1 worker (1 child) | Two-year old with BLL of 44 μg/dL and one-year with BLL of 36 μg/dL.<br><br>Children age 4 and 1.5 had BLLs in 20's.<br><br>BLLs of children age 3, 5, and 9 mos were 28, 27, and 21 μg/dL respectively.<br><br>Child (age 10 mos) had a BLL of 26 μg/dL.<br><br>Child (age 6) had BLL of 36 μg/dL. Father had BLL of 121 μg/dL. | Environmental sources ruled out.<br><br>Home was constructed post-1978; no lead paint identified. |
| Pollock [1994] | Mississippi, USA | Trucking | Case report<br>1 worker<br>2 children | Children (age 1 and 3) had BLLS of 24 and 28 μg/dL, respectively. | No environmental sources of lead identified. |
| Wiehrdt [1994] | Illinois, USA<br><br>Indiana, USA<br><br>Ohio, USA | Battery plant<br><br>Not given<br><br>Metals | Case report<br>2 children of 1 worker<br><br>Case report<br>At least 1 child of 5 related workers<br><br>Case report<br>Unknown number of children | "Gross contamination" of home; two children hospitalized/chelated.<br><br>At least 1 child had a BLL of 50 μg/dL.<br><br>Children of 3 workers had elevated BLLs (levels not provided) | |

**Table 11. Studies of Take-Home Pesticide Exposures**

| AUTHOR<br>YEAR<br>LOCATION | INDUSTRY | HEALTH EFFECT | #/RELATIONSHIP | COMMENTS AND ISSUES |
|---|---|---|---|---|
| Wolfe et al.<br>[1961] | Agriculture | Potential for pesticide poisoning | Workers' families | Discarded pesticide containers pose a hazard to children and other family members. Glass containers, paper bags, and metal drums were shown to contain residual pesticide in amounts that could easily cause the death of children or adults if the contaminated containers were improperly handled. Adequate rinsing and immediate destruction by crushing and burying was recognized as essential to avoid accidental exposures. |
| Wyant-McNutt<br>[1983] | Farming | Potential for pesticide poisoning | Worker's children | Importance of keeping farmers' children away from pesticides was emphasized. Pesticides should be stored in their original containers in a locked area. |
| Lewis et al.<br>[1994]<br>Raleigh-Durham-Chapel Hill area, North Carolina | Pesticides in the home environment | Potential for pesticide poisoning | Children | Some pesticides can persist in the home for months or years after application or track-in. Monitoring methods were applied to nine homes to assess potential exposures of children aged 6 months to 5 years. Pesticide residues were measured in indoor air, carpet dust, outdoor soil, and on the children's hands. The number of pesticides found at each home ranged from 8 to 18 of the 30 that were monitored, and the greatest number of pesticides and highest concentrations were found in carpet dust. The pesticide residues found on the children's hands correlated well with the amount of pesticides found in the carpet dust. Therefore, dislodgeable pesticide residues in carpets or on uncovered floors may present an important exposure route for children under the age of five through dermal contact or oral ingestion. |
| McGee et al.<br>[1952]<br>US | Chemical Processing Plant | Toxaphene poisoning (convulsions or death) | 1 son | Two 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. |
| | Farming | | 1 son | 17-month old boy died after drinking from tin cup containing toxaphene while his father was mixing a spray for tobacco. |
| | | | 1 son | 2-year old boy recovered from convulsions after drinking toxaphene while his mother was working in a cotton field. |
| | | | 7 family members | Seven family members were poisoned by eating collard green picked from a patch treated with toxaphene by a farmhand, who misused the insecticide which was available to him. All recovered. |

**Table 11. Continued Studies of Take-Home Pesticides Exposures**

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|---|---|---|---|---|
| Johnston [1953] Washington | Farming | Cholinesterase inhibition (nausea to death) | | Due to gross carelessness on the part of the parents, several children were poisoned in unrelated accidents in an agricultural area following exposure to the organophosphate insecticide parathion. A cholinesterase level measured below 0.5 units in the red blood cells or plasma of the victims was highly suggestive of organic phosphate poisoning. |
| | | | 1 daughter | Nine month old daughter died after playing with a can containing parathion which her father had discarded in the yard. |
| | | | 1 son, 1 daughter | 2 1/2 year old boy and his 5 year old sister played with sacks containing powdered parathion. Boy became ill, was hospitalized, and recovered. Girl had no symptoms. |
| | | | 2 sons | 23 month old and 3 year old brothers played with a can of parathion that they found in their basement. Only the younger boy had a cholinesterase test indicating serious poisoning. The younger boy was hospitalized and recovered. The older boy showed no symptoms. |
| Simon [1963] Washington | Farming | Cholinesterase inhibition (Coma and convulsions). Recovered. | 1 son | 4-year old boy was poisoned after playing with a bag of parathion in the barn on his family's farm. |
| MacMillan [1964] Canada | Farming | Cholinesterase inhibition (Respiratory distress, semi-coma). Recovered | 1 boy | Two 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. |

Table 11. Continued Studies of Take-Home Pesticides Exposures

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| Eitzman and Wolfson [1967] Florida | Farming, home use of parathion | Cholinesterase inhibition: Death | | 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. Prohibiting the sale of parathion except to agriculturists who know the dangers of parathion was recommended. |
| | | | 6 children | Six children ages 1-3 years ingested parathion stored in improper containers, such as soft drink bottles. |
| | | | 6 children | Six children aged 9 months - 10 years ate parathion that they found on the floor or window sill where it had been placed to kill roaches. |
| | | | 3 siblings | Three siblings ate parathion given to them by a sibling who pretended it was a "medicine". |
| | | | 7 children | Seven children aged 1-9 years inhaled or had skin contact with parathion powder. Three of these children were siblings who died after playing on a swing that they made from a burlap sack heavily contaminated with parathion. |
| | | | 4 children | Four children, aged 16-17 years, died after suicide or work exposures with parathion. |
| | | | 4 children | 4 children, aged 1-4 years, died after parathion exposure suggested by the presence of paranitrophenol in the urine. The place and type of contact was uncertain. |
| Mayer and Schlackman [1975] Pennsylvania | Home use of pesticides | Cholinesterase inhibition (respiratory difficulty). All recovered. | 2 siblings, mother | Mother attempted to treat head lice on 2 1/2 and 4 year old children with a shampoo that she made using parathion. Mother and children were hospitalized and recovered. |
| | | | 1 child | 4 1/2 year old child, who had a history of pica, was hospitalized after visiting a relatives home that had been treated as part of a municipal rat and roach control project. Child was discovered to have decreased cholinesterase levels, and positive urine specimen for methyl carbamate, an ingredient of Baygon. |
| Davies and Enos [1980] | Farming | Pesticide poisoning. Symptoms not specified. | 1 boy | Three year old boy was hospitalized after ingesting Dursban (chlorpyrifos), shown by the excretion of alkyl phosphate and phenolic metabolites. Agricultural worker's wife and children may be heavily exposed from exposure in the field and from materials brought back to the home. |
| Griffin and O'Malley [1992] California | Farming | Cholinesterase inhibition (lethargia, respiratory distress). Recovered. | 1 daughter | Three 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. |

Table 11. Continued Studies of Take-Home Pesticides Exposures

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| Barnett [1994] Oregon | Wood treatment | Eye irritation, nausea, vomiting, coughing | Neighbors (2 adults, 3 children) | Employee of wood treating company brought home his company vehicle which contained 6 containers of chloropicrin. The containers fell and split open, spilling one gallon on the driveway. The pesticide was carried by the breeze to the next door residence, affecting the neighbors. As a result of this accident, the company instituted a policy not to bring company vehicles home, and to utilize secure containers while transporting chloropicrin. |
| Anderson et al. [1965] California | Salvage | Cholinesterase inhibition: mild symptoms (nausea, vomiting) to respiratory distress, coma. All recovered | 2 sons, 1 neighbor child | Father, a salvage dealer, brought home flannelette sheets which he had purchased as damaged material from an insurance adjustor. The sheets were later found to have become contaminated with parathion in the hold of a ship during shipment. The children were exposed to parathion when they slept on the sheets. |
| Warren et al. [1963] California | Transportation | Cholinesterase inhibition (nausea to coma). All recovered. | 6 boys | Children became ill after wearing new unwashed jeans. All the children had depressed plasma cholinesterase levels. The clothing became contaminated with the organophosphate Phosdrin while in shipment 8 months earlier. Children wearing jeans that were washed before wearing did not become ill. |
| Clifford and Nies [1989] Colorado | Pesticide Manufacture | Cholinesterase inhibition (Nausea and weakness) . All recovered. | 3 employees | Workers became ill after wearing uniforms that were contaminated with parathion when they were washed at the plant with a uniform heavily contaminated with parathion. Potential poisoning of family members was avoided by the plant safety practice of cleaning uniforms at the plant. |
| Ganelin et al. [1964] Arizona | Crop Dusting | Cholinesterase inhibition (Nausea, vomiting, blurring of vision). All recovered. | 3 workers | Two workers hospitalized after washing airplanes that had previously been used for parathion application. Another worker had to be hospitalized after dismantling the hopper of his airplane, which had not been used for applying parathion for the previous two weeks. Employees whose duties involve maintaining or cleaning equipment used with insecticides must be instructed to follow safety procedures identical to those for handling the insecticides themselves. |
| Cannon et al. [1978]<br><br>Taylor et al. [1978]<br><br>Kelly [1977]<br><br>Hopewell, Virginia | Chemical Manufacture | Kepone poisoning (subjective nervousness, objective tremor) | Wives of 2 workers had objective tremor<br><br>94 % of family members had elevated kepone levels in blood. | 57 percent of 133 past and current workers in a kepone manufacturing plant in Virginia were found to have had, or to have, symptoms of kepone poisoning (subjective nervousness or objective tremor). Affected workers had blood levels of kepone ranging from 0.009-11.8 ppm. 19 percent of the 214 community residents had levels ranging from 0.005-0.0325 ppm. 72 percent of workers in contiguous businesses had kepone levels of 0..003-0.031 ppm. 94 percent of family members had detectable levels of kepone in their blood, with levels ranging from 0.003-0.39. Wives of two workers had demonstrable tremor. Both gave a history of having washed their husband's work clothing. |

Table 12. Studies of Take-Homer Mercury Exposure

| Source | Who affected | Effects | Urine levels | Remediation | Comments | Reference |
|---|---|---|---|---|---|---|
| Poultney, VT (thermometer plant) | Workers | Neurological symptoms | 1-345 μg/g creatinine | (Closed Plant) | | Ehrenberg et al. [1986, 1991] |
| Poultney, VT (thermometer plant) | Workers' children | elevated blood levels; no clinical effects | 25μg/L vs. 5μg/L | Decontaminated homes | | Hudson et al. [1987] |
| Chemical workers | Workers and families | None reported | Not elevated | Cleanup contaminated homes | | ATSDR [1990a] |
| Mine workers & their homes | Mainly workers | Neurological symptoms | ≤8640 μG/L | | <800 μG/L mild; 800-1900 moderate; >1900 severe | West & Lim [1968] |
| Gold miners | Workers and families | None reported | Not elevated | | 0.05-0.005 mg/m³ near washer & dryer | Zalesak [1994] |
| California?; gold extraction | Husband, wife | acute bronchitis; fever, chills, nausea | 355 μG/L, 80 μG/L | BAL | | Haddad & Stenberg [1953] |
| Washington? gold extraction | Husband, wife, three children | acute interstitial pneumonia, nausea, hypoxemia, headache | "5 X normal" (33-560 μG/24 hr) | BAL, oxygen | | Hallee [1969] |
| gold extraction | Husband | severe coughing, vomiting, cyanosis | | chelation, oxygen | | King [1954] |
| gold extraction | Woman | gastrointestinal disturbances | > 3 weeks chelation = 193 μG/dL | chelation | | Hatch [1990] |

Table 13. Studies of Take-Home Chlorinated Hydrocarbons Exposures

| AUTHOR<br>YEAR<br>LOCATION | INDUSTRY | HEALTH EFFECT | #/RELATIONSHIP | COMMENTS AND ISSUES |
|---|---|---|---|---|
| Fulton and Matthews [1936]<br>Pennsylvania | Manufacture of insulated wire and electrical cable | Acne-like dermatitis (Chloracne) | Wife<br>1 Daughter<br>1 Son | 78% of 101 workers exposed to hexachloro-naphthalene and chloro-diphenyl used in coating wire and electrical cable showed symptoms of dermatitis. The wife of one of the workers with chloracne also had similar dermatitis, as did their 11 month old daughter and 2 1/2 year old son. 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]<br>New York | Marine electrical work | Acneform dermatitis, lassitude, occasional impotence, weight loss, taste disturbances | Unspecified | 52 electricians working with electrical cable in a shipbuilding organization developed Halowax acne or "cable rash". Halowax was a mixture of chlorinated hydrocarbons incorporated into electrical cables for insulation. After this outbreak of dermatitis, preventative measures were initiated which stressed the importance of cleanliness, frequent showering and changing clothes. Work uniforms were provided. |
| Jensen et al. [1972a]<br><br>Jensen et al. [1972b]<br><br>May [1973]<br><br>Derbyshire, Britain | 2,4,5-trichlorophenol manufacture | Chloracne | 1 son<br>1 wife | 79 maintenance workers at a factory producing 2,4,5-trichlorophenol developed chloracne several months after an explosion at the plant. Analysis of residues at the plant site after the explosion showed the presence of 2,3,6,7-tetrachlorinated dibenzodioxin, which had formed due to the high temperatures of the explosion. Two pipefitters refitting a large tank at the plant that had previously been steam cleaned developed severe chloracne. The son of one of the pipefitters who played with his father while he was wearing his dirty work clothes also developed chloracne. In addition, the wife of the second pipefitter also 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. |
| Fischbein and Wolff [1987]<br>New York | Transformer maintenance | Elevated serum or adipose polychlorinated biphenyl (PCB) levels | 2 Wives | Two railway maintenance workers who repaired transformers and handled dielectric fluid containing PCB developed chloracne, and were shown to have higher serum PCB levels (77 ng/ml, 101 ng/ml) than the general population (7 ng/ml) and to have a PCB pattern resembling Aroclor 1254, indicating occupational exposure. (90% of non-industrially exposed individuals have the Aroclor 1260 pattern.) Wives of the workers did not have significantly elevated levels of PCB in serum and adipose tissue over that seen in the general population, but the PCB pattern resembled Aroclor 1254 seen in their husbands serum. 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. |

Table 13. Continued Studies of Take-Home Chlorinated Hydrocarbons Exposures

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| Baker et al. [1980]<br><br>MMWR [1978]<br><br>Bloomington, Indiana | Municipal Sewage Treatment | Elevated serum polychlorinated biphenyl (PCB) levels | 19 family members | After polychlorinated biphenyl was found to have been released into the municipal sewage treatment plant by an electrical manufacturing firm that used PCB as a dielectric in the production of capacitors, PCB levels in the serum of workers, their family members, community residents and people who applied sludge from the plant on their yards was determined. The mean PCB value in 89 people who had applied sludge to their yards was 17.4 ppb. Mean serum levels in 18 sewage treatment workers was 75.1 ppb. The mean value in 19 family members of the workers was 33.6 ppb, elevated over that seen in 22 community residents (24.4 ppb) without unusual exposure to PCB. 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. |
| Bagnell and Ellenberger [1977]<br>Halifax, Canada | Dry-Cleaning | Obstructive jaundice and hepatomegaly | Daughter | Six week old breast-fed daughter developed jaundice and hepatomegaly and was admitted to the hospital. When it was discovered that the mother regularly visited the father during lunch at the dry-cleaning establishment where he worked, the mother's blood was examined for tetrachloroethylene (TCE) and found to contain 0.3 mg/dL. TCE, reported to cause toxic hepatitis in adults following lengthy industrial exposure, was present in the breast milk at 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. |
| ATSDR [1989b]<br><br>Hesse [1991]<br><br>Kalamazoo, Michigan | Specialty plastics manufacture | Not addressed | Potential exposure of an unspecified number of family members to 4,4-methylenebis-2-chloroaniline (MOCA) | A study was conducted in 1980 to determine the presence of MOCA, a suspected bladder carcinogen, resulting from workplace trackout in an unspecified number of homes of employees of the Roto-Finish Company. Vacuum cleaner dust and dryer lint contained a maximum level of 2.6 and 0.65 ppm MOCA, respectively. Urine sampling detected a maximum concentration of 12.1 ppb in a family member and 746 ppb in an employee. Follow-up public health actions or studies are under consideration to determine the potential for exposure to MOCA via contaminated groundwater, surface water, sediments and soils from the now abandoned Roto-Finish site. |
| ATSDR [1989a, 1990b]<br><br>Hesse [1991]<br><br>Adrian, Michigan | Chemical manufacture | Exposure to the potential human carcinogen 4,4'-methylene bis(2-chloroaniline) (MOCA) | Unspecified number of spouses, 6 children | The suspected human carcinogen, 4,4-methylene bis(2-chloroaniline) (MOCA) was produced by the Anderson Development Company (ADC) between 1968 and 1979. From May, 1979 to June, 1980, the Michigan Department of Public Health (MDPH) conducted urine analyses on workers with high potential for exposure to MOCA and on the workers' spouses and their children. MOCA was found in the workers' urine in concentrations up to 58,000 ppb, in spouses' urine, and ~~in 6 of 12 children~~ in children tested (15 ppb). Urine specimens from occupants of 50 randomly selected households within a 5 block radius of the plant did not show detectable levels of MOCA (detection limit 5 ppb), except for one preschool child (6 ppb). It was suggested that MOCA may have been "tracked" out of the manufacturing plant on the shoes and clothes of the employees and deposited in their residences. The MDPH determined that MOCA could be transferred from employees clothes to other clothing during regular household laundering. |

Table 13. Continued Studies of Take-Home Chlorinated Hydrocarbons Exposures

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| ATSDR [1991b] Muskegon County, Michigan | Chemical manufacture (pesticides, herbicides, 3,3'-dichlorobenzidine benzidine | Exposure to the potential human carcinogen 3,3'-dichlorobenzidine | Unspecified number of family members of workers | Chemical production of a variety of chemicals, including pesticides, herbicides, 3,3'-dichlorobenzidine (DCB), and benzidine, began at a 120 acre site, Bofors-Nobel, Inc., located near Muskegon, Michigan in 1960 and continued under various owners. In 1980-1981, the Michigan Department of Public Health conducted a study to examine track-out of DCB in homes of an unspecified number of the employees and to determine DCB content in soil near the site. Samples collected from vacuum cleaner bags from homes of some of the employees had up to 10.5 ppm DCB. Dryer lint contained up to 0.74 ppm. A soil survey conducted in 1984 found from 0.07 to 3.01 ppm in soil up to a half mile of the site. From 0.006-0.281 ppm DCB was found in the urine of employees and family members. No data were provided on urine content in other members of the community. It was concluded that a hazard may exist for workers involved in remediation work on the site and their families if adequate safety measures are not taken. |
| Donaldson and Johnson [1972] Redwood City, California | Manufacture of ion exchange resins | Potential for exposure to the carcinogen bis(chloromethyl) ether | Potential exposure of an unspecified number of family members to bis(chloromethyl)ether | Family members of workers that used bis(chloromethyl)ether in the manufacture of ion exchange resins at the Diamond Shamrock Chemical Company between 1945-1972 may have been exposed to this potential lung carcinogen via contaminated work clothes brought home by the workers. Although the company provided laundering, the employees contributed to the cost of work clothes, and may have taken them home. No quantitative measurements were made to determine work clothes contamination or to determine the extent to which clothes were taken home. |
| Townsend et al. [1982] Midland, Michigan | Chlorophenol production | Survey for adverse pregnancy outcomes (stillbirths, spontaneous abortions, congenital malformations) | 370 wives | 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. |
| Smith et al. [1982] New Zealand | Herbicide applicators | Surveyed for miscarriages, congenital defects: no increase found | 989 applicators and wives | Applicators who sprayed 2,4,5-T products, reported to contain the contaminant 2,3,7,8-TCDD (dioxin), 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. |

Table 13. Continued Studies of Take-Home Chlorinated Hydrocarbons Exposures

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| Doherty [1984] Jefferson County, Missouri | Chemical waste disposal | Exposure to dioxin | Occupants of 1 residence | Dioxin at concentrations up to 48 ppb was found in the driveway at one residence and along a length of private road (Lacy Manor Drive/Sandcut Road). In addition, carpet fibers from the residence showed dioxin contamination. Contamination occurred when a previous owner of the residence, who was a driver for the Russell Bliss Oil Service Company, sprayed the road with waste oil to control dust in the early to mid 1970s with a material contaminated with dioxin. Contamination was suspected when an occupant reported sickness in horses on the site. It was determined that the family living in the residence was at risk of developing adverse health effects from dioxin exposure, since they were exposed to levels of dioxin in excess of 1 ppb. The residents were relocated while the residence was cleaned and the road capped with asphalt. |
| Ramsey [1987] Jefferson County, Missouri | Organic chemical production | Not addressed | Potential dioxin exposure to occupants of several homes | Several residences in Jefferson, Missouri were contaminated with dioxin when contaminated fill was used in drainage ditches, gardens, and footings. The fill came from a nearby horse arena where several horses died after the arena had been sprayed with dioxin-contaminated oil to suppress dust. Concentrations of dioxin as high as 660 ppm were found in the ditches. Wipe samples inside one of the homes showed 3.6 ppb dioxin, a level greater than the 1 ppb action limit set by the Center for Disease Control. |
| Hess [1988] Ballwin, Missouri | Chemical waste disposal | Exposure to dioxin | Occupants of 24 residences and one business | The Castlewood residential community in St. Louis County, Missouri, became contaminated with dioxin after roads and parking lots were sprayed with waste oil to suppress dust by the Russell Bliss Waste Oil Company in the 1970s. This company was a subcontractor hired for the removal and disposal of chemical wastes from the Northeastern Pharmaceutical Company that manufactured 2,4,5-trichlorophenol in a process that produced dioxin as a waste product. Contamination in a tavern on one of the parking lots was 32.6 ppb. Soil samples in the area had levels up to 558 ppb. A total of 24 residences and one business had to be cleaned. |
| MacDonald [1988] Quail Run, Gray Summit, Missouri | Chemical waste disposal | Exposure to dioxin | Occupants of 28 residences | Quail Run Mobile Manor, a mobile home park located near Gray Summit, Missouri, was contaminated with dioxin when a gravel road was sprayed with dioxin-contaminated waste oil to suppress dust in the early 1970s. Up to 2200 ug/kg dioxin was found in the soil in the mobile home park. 28 homes were contaminated with dioxin. |

Table 13. Continued Studies of Take-Home Chlorinated Hydrocarbons Exposures

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| Stasiuk [1993] New York | Dry cleaning establishments | Potential exposure to perchloroethylene | Unspecified number of residents and workers in locations near dry cleaning establishments. | Indoor air exposure to perchloroethylene in residences and businesses near dry cleaning establishments was assessed in New York State, where 18 percent of the dry cleaners are in buildings with residences, and half of all dry cleaners are in buildings containing other businesses. Indoor air concentrations ranged from 15 to 197,000 ug/$m^3$ air in apartments in buildings where dry cleaners used transfer machines. Businesses near dry cleaners were found to have levels as high as 50,400 ug/$m^3$. It was estimated that 170,000 people would be exposed to perchloroethylene in New York State. |
| Schreiber [1993] | Dry-Cleaning | Potential for adverse health effects | Breast fed children | Using physiologically based pharmacokinetic modeling, authors predicted breastmilk PCE concentrations in women occupationally exposed to tetrachloroethylene (perchloroethylene, PCE) to range from 857-8440 ug/L and in women exposed to PCE in residences near dry cleaners from 16-3000 ug/L, compared to predicted concentration of 1.5 ug/L in typical residential exposures. Most of the modeled exposures to infants via breastmilk exceeded the oral reference doses calculated by the USEPA. The hepatotoxicity observed by Bagnell and Ellenberger, 1977, occurred at a doses about twice that predicted by for highest maternal exposures evaluated. Monitoring studies to measure actual concentrations of PCE in breastmilk of exposed mothers are necessary. |
| Price and Welch [1972] Michigan | Unspecified occupation | Potential for elevated levels of polychlorinated biphenyl (PCB) in serum and adipose tissue | Potential family exposure | Polychlorinated biphenyls were present in house dust in vacuum sweepings of houses of occupationally exposed workers in Michigan. Samples contained up to 180 ppm PCBs, mainly Aroclor 1254. PCB levels in adipose or serum of the family members was not presented. |
| Clapp et al. [1985] Moscow, Pennsylvania | Urethane casting | Not addressed | Potential exposure of an unspecified number of family members to 4,4-methylenebis-2-chloroaniline (MOCA) | Twenty-four hour urine samples were collected from six production workers, one maintenance worker, and one manager in the Steinmetz and Sons Machinery Co, where MOCA was being used in producing molded urethane products. All samples had detectable concentrations of MOCA. MOCA was not detected in any air samples, and in only two of eighteen surface wipes in the plant. It was recommended that the company issue clothing which is laundered daily and not worn home and that the employees shower before leaving at the end of a work shift. |
| Marceleno et al. [1974] Lancaster, South Carolina | Textiles manufacture | Potential exposure to the carcinogen bis-chloromethyl ether | Unspecified number of family members | The National Institute of Occupational Safety and Health conducted a survey at the Grace Bleacher, Spring Mills Inc., Lancaster, South Carolina, to determine if workers were exposed to the carcinogen bis-chloromethyl ether (BCME) formed during the use of formaldehyde resins and chloride-containing catalysts. Personal and area air samples taken at the textile factory failed to detect BCME. However, since the potential for the formation of BCME existed during the processes used at the plant, it was suggested that workers shower and change from their work clothing before departing from home in order to avoid taking BCME home to their families. |

Table 13. Continued Studies of Take-Home Chlorinated Hydrocarbons Exposures

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| Hartle [1987]<br>Paoli, Pennsylvania | Rail transportation | Potential for cancer, liver injury | Exposure potential to families of 105 workers | NIOSH conducted a study in 1986 to evaluate occupational exposure to polychlorinated biphenyl (PCB) among workers in a rail yard operated by the Southeastern Pennsylvania Transit Authority (SEPTA). PCB contamination, assumed to be the result of the use of PCB-containing oils in transformers on commuter rail cars, had been known since 1978. Sampling from the floors of the facility, including the repair and maintenance areas, lunch room, and locker rooms, showed high levels of contamination (32,000 and 60,000 ug PCB/$m^2$ in lunch and locker rooms). High contact items such as handles of SEPTA owned tools and crane controls sampled for surface contamination had from 21 to 9,800 ug PCB/$m^2$. Workers were required to provide their own tools, but NIOSH was not permitted to sample personal items. An advisory panel including EPA and NIOSH members, convened in 1985, recommended that high contact items not exceed PCB surface concentrations greater than 50 ug/$m^2$. It was recommended that disposable shoe covers should be used and discarded when traveling from the parking lot to the locker rooms, that separate work and street shoes be used, and that tools used on site remain on site in order to prevent off site contamination of workers' homes. |
| Todd and Timbie [1979]<br>Florence, South Carolina | Wood Preservative Treatment Plant | Potential for exposure to pentachlorophenol | Potential for exposure to family members | An industrial hygiene survey was conducted at a wood treatment plant where pentachlorophenol (PCP) and creosote were used. Monitoring showed PCP concentrations of 0.01 mg/$m^3$ in the air near the equipment, and 0.11 mg/$m^3$ on top of the treated wood. Almost half of the 141 employees wore work uniforms while the remainder brought work clothing from home. No measurements were made for PCP content on clothing, or in worker's homes. |
| Hartle et al. [1987]<br>Lafayette, Indiana | Aluminum production | Potential for cancer, liver injury | Potential exposure to families of workers | A health hazard evaluation conducted by NIOSH at plant that produced extruded aluminum found polychlorinated biphenyl (PCB) contamination in the extrusion area of the plant (39,000 and 58,000 ug/$m^3$) and in the lunch/locker rooms (636 and 2632 ug/$m^3$). The guideline for PCB surface contamination is 50-100 ug/$m^3$. The plant had previously used hydraulic fluid containing PCB. It was recommended that access to the extrusion area be limited to essential personnel, and that employees be provided with company laundered coveralls and a "clean side/dirty side" locker room for use when arriving and leaving the worksite. |

Table 14. Studies of Take-Home Exposures - Other Substances

| AUTHOR (YEAR) LOCATION | CONTAMINANT | INDUSTRY | STUDY DESIGN | RESULTS | COMMENTS |
|---|---|---|---|---|---|
| Katzenellenbogen [1956] Israel | Estrogens | Pharmaceutical manufacturing | Case reports | Five children of pharmaceutical workers developed hyperestrogenic syndromes. Some of these children were the ones also discussed in the above mentioned 1956 article. | The product was stilbestrol. |
| Klorfin and Bartine [1956] Israel | Estrogens | Poultry farm workers | Case report | Three children of workers who handled estrogens at work developed hyperestrogenic syndromes. The parents who were the workers also had hyperestrogenic syndromes. | Two children improved after the parent changed employment.<br><br>Another child was lost to followup. |
| Budzynska et al. [1967] Pacynski et al. [1968] Poland | Estrogens | Pharmaceutical manufacturing | Case report | Six children of employees who worked with estrogens developed hyperestrogenic syndromes. | Exposure through in utero exposure vs. take home exposure was discussed. However, on stepchild did not live with the worker until the child was four years old ruling out in utero exposure in that case. |
| Pacynski et al. [1971] Poland | Estrogens | Pharmaceutical manufacturing | Case report | Six children of employees who worked with estrogens developed hyperestrogenic syndromes. | Same children as in prior two articles. However, in this article it stated that after the workplace was investigated and recommendations were carried out, the hyperestrogenic syndromes disappeared in all the children and diminished in the workers. This result would argue against the in??????????? |
| Aw et al. [1985] Indiana | Estrogens | Pharmaceutical manufacturing | Cross-sectional study of employees working with hormones | Three male children of current workers and two children of former workers determined to have breast enlargement. | In the children of former workers, the breast enlargement diminished after the parent left employment at this workplace. |
| Bierbaum [1993] Kansas | Estrogens | Feedlot repair | Case reports | 1976 NIOSH memos dealing with 4 children (ages 3 and 6) with gynecomastia and pubic hair. | The two fathers of these 4 children repaired feedlot bins containing feed supplemented with DES. |

Table 14. Continued Studies of Take-Home Exposures - Other Substances

| AUTHOR (YEAR) LOCATION | CONTAMINANT | INDUSTRY | STUDY DESIGN | RESULTS | COMMENTS |
|---|---|---|---|---|---|
| Wilken-Jensen [1983] Denmark | Animal allergen | Veterinary medicine | Case report | | |
| | Grain dust | Miller | Case report | Son developed asthma if the father did not change clothes when he came home from the mill. | |
| Venables and Newman-Taylor [1989] United Kingdom | Animal allergen | Laboratory animal work | Case report | Husband developed asthma due to animal allergens brought home on the wife's person. | Initial sensitization probably due to pet rat but specific asthma symptoms occurred specifically after contact with the wife, an animal handler. |
| | Platinum | Precious metal refining | | Wife developed recurrence of asthma after change in husband's job. | Symptoms occurred when husband returned from work. No symptoms on weekends. Positive skin prick tests to platinum salts used in his job. |
| Klemmer et al [1975] Hawaii | Arsenic | Wood treatment: pesticide use | Survey of arsenic in house dust | | Higher values were found in the homes of employees of pest control firms or firms dealing with wood preservation with chemicals. |
| Falk et al. [1981] United States | Arsenic | Copper smelter | Case report | Hepatic angiosarcoma | Exposure from fathers clothing, outside environment and water supply. |

Table 14. Continued Studies of Take-Home Exposures - Other Substances

| AUTHOR (YEAR) LOCATION | CONTAMINANT | INDUSTRY | STUDY DESIGN | RESULTS | COMMENTS |
|---|---|---|---|---|---|
| Carvalho et al. [1986] Santo Amaro City, northeast Brazil | Cadmium | Lead smelter | Cohort of 396 children age 1-9 year living less than 900m from primary lead smelter. | Geometric mean and standard deviation of CdB were 0.087 and 2.5um/l resp., range 0.004-0.511<br>380 children (96%) had CdB > than 0.0089 um/l<br>The relationship between parental employment in the smelter and children CdB levels were 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 the shorter distance from home to smelter. | |
| Brockhous et al. [1988] Germany, Stolberg | Cadmium | Lead and zinc smelters | Cohort of 9 children from lead worker families and 195 children from other families, aged 4-17 years. | Children from lead worker families (n = 5) had significantly higher GM CdU of 0.34, GSD 2.6 than children from other families (n=97) whose GM CU was 0.13. GSD 2.2, $p < 0.01$.<br>CdB among children from lead worker families were higher than in children from other families but not statistically significant. | Cd in hair (CdH) was measured in children.<br>Medium CdH for girls was 4.7ppm SD 2.2, and for boys 3.4, SD 2.2.The "light" children had significantly lower CdH than the medium ($p<0.05$) and the dark $p<0.005$) children. CdB was lower in "dark" children than in "light"<br>The mean CdH was significantly ($P<0.0001$) higher for children whose fathers worked in the lead smelter compared with children whose fathers had other jobs.<br>CdH increased proportionally with increased Cd in the soil. |
| Carvalho et al. [1989] Santo Amaro, Brazil | Cadmium | Lead smelter | Cohort of 263 children 1-9 year old, living less than 900m from lead smelter, were divided into racial groups; light, medium and dark. | | |

Table 14. Continued Studies of Take-Home Exposures - Other Substances

| AUTHOR (YEAR) LOCATION | CONTAMINANT | INDUSTRY | STUDY DESIGN | RESULTS | COMMENTS |
|---|---|---|---|---|---|
| Maravelias et al. [1989] Attika, 60 km southeast of Athens, Greece | Cadmium | Lead smelter | Cohort of 514 children age 5-12 from 4 schools located within various distances from the smelter: a.500m b.900m c.1500m d.1500m | Average CdB level among these children was 0.36ug/l, range 0.1-3.1ug/dl CdB were higher in children from school closest to the smelter (500m) as compare to the other schools but no relationship was found between parental employment in the smelter and CdB. | On average boys had significantly higher CdB than girls, CdB increases with age ($r = 0.1917$, $p<0.001$). |
| Hofstetter et al. [1990] Stolberg, West Germany | Cadmium | Lead smelter | Cohort 229 children ages 6-7 years | Mean concentration of CdB 0.14 ug/dl, range <0.1 -0.5 ug/dl. Significantly highest CdB were observed among children from two school ($p< 0.01$ and $p< 0.001$) located in the vicinity of the smelter as compared to school located in the farthest distance from the smelter. CdB among children whose fathers were employed at the smelter were insignificantly higher( higher CdB than those of fathers who had other jobs. | |
| Madoff [1962] Unknown | Fibrous glass | Home | Case report | Deep excoriations of the skin were found on a 5-year old boy's trunk and shoulders. The boy's two younger sisters also developed the same symptom later. | The cause was traced to clothes washed in a contaminated washing machine where several pairs of fibrous glass curtains had been washed. |
| Abel [1966] New York | Fibrous glass | Home | Case report | A mother and her 3 children developed severe pruritis. | The mother had washed a fiberglass curtain with the family laundry in the washing machine. |
| Peachey [1967] Unknown | Fibrous glass | Home | Case reports | A woman and her six children, and another family of three, developed pruritus. The man of the former family did not have the symptom. | Both cases were traced to mixing glass-fibre curtains with the family clothes in the same washing machines. The clothes of the man who was not affected were handwashed separately. |

Table 14. Continued Studies of Take-Home Exposures - Other Substances

| AUTHOR (YEAR) LOCATION | CONTAMINANT | INDUSTRY | STUDY DESIGN | RESULTS | COMMENTS |
|---|---|---|---|---|---|
| U.S. Senate [1991] North Carolina | Otto fuel | Hazardous waste incineration | Case report | Two children developed severe asthma after exposure to hazardous waste (primarily though to be Otto Fuel) on parental clothing. | The children's illnesses improved after the fathers stopped working at the incinerator. |

Table 15. Industrial Hygiene Review of Take-Home Exposure-Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Abbritti et al. [1979] Italy | Lead | Ceramic Factories (pb) & workshops at home (pb). | 1. Looked at no lead exposure vs. lead exposure from ceramic factories and home workshops. 2. 372 children from 3 communities Blood pb | Children whose parents worked with pb had higher PbB | 1. Silent epidemic 2. Saturnism 3. Study from Italy |
| Abritti et al. [1987] Italy | lead | See above | Blood Pb | PbB up to 97 ug/l in children whose parents work with lead | 1. Abstract only 2. Followup to above study |
| Abritti et al. [1988] Italy | lead | Road Traffic & Ceramic Industry | 534 children Blood Pb via AAS | Mean PbB = 8.3 to 9.7 ug/dl 3 children had PbB of 29.5, 31.0 and 33.9 ug/dl boys had slightly higher PbB in all groups | 2 of 3 communities that were used in previous study, plus 1 new community |
| Anderson et al. [1965] Canada | Parathion | Sheets contaminated in hold of ship during transport | Sheets & other materials "stripped" in glass jars with benzene. Strips analyzed via GC-EC detector | 2 boys (friends who played together) admitted to hospital at separate times. Both nearly died. 1st boy readmitted later. Brother of 1st boy also got sick - not hospitalized | 1. Flannelette sheets contaminated with parathion resulted in anti-cholinesterase poisoning 2. Authors call for national & international regs covering Mfg, transport & handling of pesticides. 3. Canadian study. |
| Anderson et al. [1976] New Jersey | Asbestos | Amosite asbestos products mfg | 1. Evaluate health status of 679 house hold contacts of asbestos workers. 2. Interviews, X-rays, physical exams. | 1. Family contacts had no other known asbestos exposure. 2. 35% of house hold contacts had radiographic abnormalities vs 5% of controls. | 1. A total of 1664 workers (1941-1954). 2. Plant did not have change rooms or change of clothing available. 3. New Jersey study |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Anderson et al. [1979] | Asbestos | Amosite asbestos products mfg | See Above | See above | 1. This is continuation of study reported 3 years earlier. |
| Anderson et al. [1979] | Asbestos | Amosite Asbestos Products Mfg. | See Above | 1. Overall 225 (35.9%) o 626 house hold contacts had chest X-ray abnormalities vs 15 (4.6%) of 326 controls. | 1. Update of study. |
| Anderson [1983] | Asbestos | Amosite Asbestos Products Mfg. | See above | Same statistics given as in above reports | 1. In: Proceedings of World Symposium on Asbestos<br>2. Author makes point that latency could be much longer in this study due to age of some children when they entered cohort. |
| Anonymous [1952] Phillippines | Lead | Storage battery production | NA | 1. 3 children were treated at clinic or hospitals, 2 of 3 died.<br>2. Fathers made storage batteries in shops at home. | 1. One page report in Journal from 1952.<br>2. Copy is of poor quality.<br>3. Study in Philippines. |
| Anonymous [1992] Virginia | Lead | Valve thread area | Clothing & Surface Wipe Samples | 1. Paint used in as sealant was 80% lead.<br>2. 13 month old had 73 ug/dl blood EP.<br>3. Mother, father and brother had high PbB as well | 1. 7 workers & families were tested (some refused).<br>2. Paint had recently been introduced.<br>3. Most homes were free of Pb. One had lead paint. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Apol and Singal [1980] Alaska | Lead | Lead acid storage battery mfg. | Air sampling in plant - NIOSH P&CAM 341. Medical Interviews, Blood tests - coulter counter, AAS | PBZ samples (5) ranged from 111 to 1053 ug/$m^3$. | 1. Process generates lead dust & fume, no LEV except at lead melting pots.<br>2. Owner & family lived above plant.<br>3. Recommendations:<br>a. Install LEV<br>b. Improve resp prg<br>c. install change rm<br>d. Improve housekeeping<br>e. Follow OSHA Pb Stand for housekeeping, resp prg, etc.<br>f. Close up common hallway (plant/home) clean home, monitor family for pb exposure. |
| ATSDR [1991b] Michigan | 3,3'-dichlorobenzidine (DCB) + other haz chems | Hazardous Waste Site | 1. Previous Studies are discussed.<br>2. Tests included soil, water, air, medical exams, etc. | 1. Report is a hazard assessment of a Hazardous Waste Site<br>2. History of waste water leaks at pesticide mfg plant, one involved 2,000,000 gallons. | 1. 1981 "trackout" study found DCB in homes of workers. DCB found in vacuum cleaner bags & dryer lint. DCB found in urine of some workers & family members. |
| Aw and Stephenson [1985] Indiana | Zeranol (an animal growth hormone) the main ingredient in Ralgro | Pharmaceutical formulator, ingredients are purchased externally | 1. Air, surface wipes, dermal patches, clothing pieces analysis of above via HPLC. 2. Questionnaire (ask about children's health), blood samples, physical exam | 1. Some children had breast enlargement, 1 had external medical exam - found increased bone growth.<br>2. Employee's work clothing contaminated with zeranol, employee's skin also contaminated. | 1. No study of workers homes.<br>2. 2 male children were seen by NIOSH physician.<br>3. Workers often wore work clothes home.<br>4. Recs made to reduce the potential for zeranol being "taken home".<br>5. Indiana study |
| Bagwell and Ellenberger [1977] | Tetrachloro-ethylene (perchloroethylene) | Suede & leather dry cleaning | GC headspace procedure of breast milk & venous blood samples | 6 week old female child had obstructive jaundice | 1. Father worked in dry cleaning plant<br>2. Mother ate lunch with husband (30-60 min).<br>3. Evidence suggests baby exposed via breast milk. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Baker et al. [1977] | Lead | Secondary Lead Smelter | 1. Questionnaire & blood samples of wives & children of lead workers.<br>2. Sampled paint from homes for Pb content and collected wipe samples. Paint analyzed via X-ray fluorescence, wipe samples via anodic stripping voltametry. | 1. 38 (41.8%) of children had PbB of 30 or more ug/100 ml, 8 children were hospitalized & received chelation therapy.<br>2. Children's blood pb levels closely matched their father's level. | 1. Lead based paint was excluded as an important source of lead exposure.<br>2. As a result of study workers homes were cleaned. Workers started showering and changing before going home. |
| Barrett [1994] Oregon | Chloro-picrin | Wood Treating Company | NA | 1. Containers of pesticide dropped on employee's driveway. Wind carried vapors next door.<br>2. Neighbors (2 adults & 3 children) got sick. | 1. Case reported in Letter to Diane Manning (Per NIOSH request for info.)<br>2. Wood treating employee's child stayed in house - did not get sick.<br>3. As result of accident, employer changed SOPs (employees no longer take pesticides home. |
| Barrett [1944] Oregon | Lead | Bronze Foundry | NA | 2 children of foundry workers had high blood leads (14 & 23 ug/dl). | Or-OSHA documented (?) that employees were taking lead dust home on clothes. |
| Barrett [1994] Oregon | Lead | Tile Mfg | NA | 1. Study is ongoing.<br>2. One worker had blood lead of 73 ug/dl. | Investigators think at least one child (of a worker) has high blood lead. |
| Beegle and Forslund [1990] | Asbestos & Lead | Homes with asbestos containing materials and lead contamination from reclamation of used batteries. | NA | NA | (Report is in a letter to USEPA R-3 and somewhat confusing.) Report is plan to clean ACM and lead dust from residences. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Belager et al. [1979] Illinois | Asbestos | Vinyl asbestos & asphalt asbestos floor covering mfg. | Personal & area sampling in tile mfg plant. | Results were listed for in plant investigation. | 1. Rec made to change clothes & leave all work clothes at work.<br>2. Previously, showers and change of work clothes were optional.<br>3. Illinois report. |
| Benning [1958] Ohio | Mercury | Carbon brush mfg (brushes used in electric motors) | GE Mercury Vapor detector used in plant & Greenburg/Smith impinger per ACGIH analytical method | 1. Air levels in plant for total hg - 0.2 to 0.75 $mg/m^3$. Hg vapor is air - 0.18 to 0.38 $mg/m^3$. (Report also lists summary air levels of hg up to 7.5 $mg/m^3$ ?)<br>2. 36 of 90 women interviewed sore mouths, 22 had shakes.<br>3. Health effects (antisocial) had negative impact on family life.<br>4. 52 of 90 had symptoms of hg poisoning.<br>5. Tamping cmpd found to contain 22 to 24% hg. | 1. Plant had poor housekeeping, no ventilation, no showers facilities or change of work clothing policy.<br>2. Except for plant management all workers were women.<br>3. Interviews and letter from employees indicate classic exposure to hg at high levels.<br>4. As a result of study management installed local exhaust ventilation and switched to non-mercury tamping cmpd.<br>5. Ohio report. |
| Bianchi et al. [1991] Italy | Asbestos | Asbestos related industries | Study of 1765 consecutive necropsies at Monfalcone hospital | For women cleaning asbestos contaminated work clothes, work clothing was main source of exposure. | See below. |
| Bianchi et al. [1993] Italy | Asbestos | Asbestos related industries | Necropsy based study to evaluate asbestos exposure in general population of Monfalcone, Italy. | 1. 92 cases of malignant mesotheliomas diagnosed.<br>2. Cases included 84 men and 8 women.<br>3. Cases with domestic exposure had cleaned work clothes of family members employed at shipyard or sodium carbonate factory. | 1. Follow-up to previous report.<br>2. Authors suggest due to high asbestos exposure into 1970s, expect future waves of mesotheliomas. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Bohne and Cohen [1985] | Beryllium | Beryllium production | One aspect of investigation looked at re-suspension of dust from work clothing. Analysis via ICP-AES. | Personal monitors mounted on fabric had increased concentration of dust compared to "breathing zone samples". Used sampling frames (?) for all samples. | 1. Authors do not comment on significance of results for transport of chemicals to family members. 2. Research supported in part via NIOSH grant. |
| Branson and Henry [1982] | Pesticides | Pesticide application | NA | NA | This is an extension bulletin (1-page) alerting pesticide users of potential hazard for workers and families. |
| Brockhaus et al. [1988] | Lead & Chromium | ? | Biological samples of blood, urine & shed deciduous teeth. | 1. Children from lead worker families had significantly higher blood lead & chromium levels. 2. Blood lead in rural children = 5.5 to 7.0 ug/dl; children from industrial areas had 0.5 to 1.0 ug/dl more blood lead. | Children from lead worker families tend to have higher blood lead values. |
| Cannell et al. [1987a] | NA | NA | NA | NA | Report describes device to measure directly the spatial distribution & total mass of fluorescent tracer material with intent to evaluate home contamination. |
| Cannell et al. [1987b] | Particle activity (radioactivity) distribution in homes. | NA | NA | NA | Follow-up to previous report. More detailed description provided. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Carvalho et al. [1984] | Cadmium | Lead smelter | Prevalence study of anemia among children (1-9 years) living near lead smelter. 2. Blood lead samples analyzed via AAS with heated graphite furnace atomizer. | 1. 1 year olds had prevalence rate of 48.5% compared to 18.6% for all children tested. 2. Children of lead workers had higher blood lead values (3.26 u-mol/l)than other children (2.73 u-mol/l). | 1. River heavily polluted by lead & cadmium. 2. This is part of major study health effects for community around smelter. 3. lead workers took used "filters" home from the plant, for some purpose. |
| Carvalho et al. [1986] | Cadmium | Lead smelter | (see above) | Boys & girls had equal cadmium blood levels. 2. 96% of children tested had cadmium blood levels above "normal" value (1 ug/l). | (same study as above) |
| Carvalho et al. 1989] | Cadmium | Lead smelter | 1. Determine concentration of Cd in hair of children living within 900 meters of lead smelter. 2. Hair samples analyzed via energy transfer atomic absorption. 3. Soil samples analyzed via flame AAS. 4. Blood samples analyzed via flameless AAS. | Geometric mean for cadmium in hair was 4.12 ppm. Girls had higher mean values (4.7 vs 3.4 ppm) than boys. | 1. Primary lead smelter polluted Subae' river basin with at least 400 tons of cadmium between 1960-1980. 2. Smelter doss used to pave streets, private yards, etc. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Chamberlin et al. [1957] | Beryllium | Beryllium plant | clinical assessment of 20 patients whose chest roentgenogram & clinical findings indicate a form of pulmonary disease similar to that seen in beryllium workers. | 1. 5 of 20 patients were family members of beryllium workers.<br>2. 19 of 20 patients had beryllium in their lungs. | Some patients had no known exposure to beryllium. |
| Chenard et al. [1987] | Lead | Primary lead smelter | 1. Blood lead, hair lead, and FEP were determined for 132 children (2-12 years)living near smelter.<br>2. PbB via flameless AAS with a graphite furnace. | Children exposed thru fathers work had ratio of 1.23 (exposed vs non-exposed); children exposed thru residence had 1.79 ratio & children with both sources of exposure had a ratio of 1.83. | 1. Children exposed thru father's occupation and thru residence had higher blood lead values.<br>2. Authors note benefit of post shift hygiene facilities - not used at smelter. |
| Cohen and Positano [1986] | Beryllium (Be) | Beryllium processing plant | 1. New and used shirts analyzed to find the concentration of Be in the fabric.<br>2. Also fabric was agitated in a glove box to see how much Be would be re-suspended. | 1. Old shirts re-suspended significantly higher quantities of Be to the air than newer shirts.<br>2. The old shirts also showed significantly higher concentrations of Be before agitation.<br>3. Be concentrations ranged from 12 to 37 $mg/m^2$ of the shirt fabric **(Leroy is this last part correct?)** | This is a followup to study done by Bohne & Cohen (1985).<br>2. Air concentration of Be measured in refinery was only a fraction of the (TLV) 0.002 $mg/m^3$. However, there was a high concentration of Be on the shirts (noteworthy). |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Clapp et al. [1985] | 4,4'-methylenebis(2-chloroaniline) also called MBOCA | Urethane casting | 1. Air samples<br>2. Surface wipes.<br>3. Hand contact monitors.<br>4. Urine samples. | 1. Air samples = < 2.9 ug/$m^3$ (all samples).<br>2. Surface wipes = <5.3 ug/wipe (most surfaces).<br>3. Hand monitors = <5.3/hand monitor (all samples).<br>4. Two surface wipes had >5.3 ug/wipe. Both were collected on MBOCA pots(?) where it is expected to be found.<br>5. All urine samples for 6 workers contained detectable concentrations of MBOCA but were less than 100 ug/l as enforced by OSHA. | 1. No take-home toxin measurements were made.<br>2. Study investigators recommended:<br>a. Clothing not be taken home.<br>b. Shower before leaving work. |
| Cook et al. [1993] | Lead | Mining community | 1. Venous blood—samples from kids 6-71 months of age (n=150).<br>2. Floor dust: main entrance, bedroom, other rooms<br>3. Window sill dust.<br>4. Soil samples.<br>Pb in paint: exterior, interior by x-ray fluor.,<br>5. Tap water. | 1. Bl-V (m) = 10.1 +or- 5.58 ug/dl, GM = 8.7 +or- 1.79.<br>2. Floor & soil samples range = 1000 to 2453 ppm (mean value of all samples).<br>3. Pb in paint = 14% (exterior), 9.2% (interior).<br>4. Tap water <5 ug/l (n = 104) one sample = 13 ug/l. | 1. High Pb in soil throughout the community.<br>2. + assoc between Pb-B and floor dust (p=0.189)<br>3. + assoc between Pb-B and window sill (p=0.372).<br>4. No assoc between Pb-L & X-raysflor of paint in or on homes.<br>5. + Assoc between parent being a miner & wearing work clothes home and Pb-B of kids (p= 0.019). |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Chesner [1950] | Beryllium | Beryllium plant | Autopsies: 3 cases. | Two of 3 cases had indirect contact with plant. | 1. One case used beryllium ore bags she got from the a neighbor who worked at the plant.<br>2. Second case (8 year old female) had father who worked at plant<br>3. Authors note in addendum that 2 other autopsied cases occurred - one a 26 year old female whose husband worked at the plant. |
| CH2M Hill [1991] | Lead, arsenic, cadmium, mercury, zinc, copper, antimony | Lead Smelter | Vacuum & shampoo rugs etc. to evaluate lead levels. | 1. Lead loading in carpets ranged from 11.8 to 282.8 mg/ft$^2$.<br>2. Bulk of lead contamination was in carpet rather than in pad or on floor below carpet.<br>3. Vacuuming & shampooing did not remove much lead from carpet. Worked OK for throw rugs. | 1. This is a report of planned house dust remediation project of Bunker Hill site. Discusses results of pilot test to clean carpets, upholstered chairs, etc.<br>2. Lead was used as indicator of all 8 metals. Initial analysis indicated all 8 metals followed general trend for decontamination.<br>3. Previous RADER report indicated largest lead source for kids was residence. |
| Davies and Enos [1980] | Pesticides | Agriculture | 1. Urinary excretion of 6-beta-hydroxycortisol in migrant children. | 1. Significantly high levels of 6-beta-hydroxycortisol were found.<br>2. High serum DDE were found. | 1. This may be a child working in the fields problem - rather than a take home toxin problem.<br>2. This data is reported in a different reference, but references do not accompany this paper. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Danziger [1973] | Mercury (hg) | Scientific glassware mfg, calibration of glassware. | 1. Hg in air, Hg vapor detector.<br>2. 75 workers<br>3. Urine spot samples (not 24 hr).<br>4. Engineering controls were evaluated. | 1. One subject had objective tremor.<br>2. Hg in urine range = 0.000 to 2.220 mg/l.<br>3. Hg in air range = 0.00 to 0.30 $mg/m^3$. | 1. Recommended that workers not wear knitted clothing because hg can e trapped i the interstices of the fabric. (No mention of THT in this paper, but knitted clothing could be a route for take home. No mention of how much Hg could get trapped in the fabric). |
| Donovan [1994] | Lead (Pb) | Stain glass studio. | 1. Air sample @ work.<br>2. Air sample @ home.<br>3. Wipe @ home.<br>4. Vacuum carpet @ home.<br>5. Soil samples around home and studio.<br>6. Wipe @ studio. | 1. 0.0 to 2 $ug/^3$ in air @ work.<br>2. Home air, home wipes and carpet vacuum samples all were <LOD (LOD not given).<br>3. Soil samples: studio = 58-62 ug/g, home = 53-73 ug/g.<br>4. Studio wipe samples = 1200 to 1,600,000 $ug/m^2$.<br>5. Pb-B = 1.8-2.1 ug/dl.<br>6. ZPP = 31-39 ug/dl. | 1. Abstract only<br>2. The simple, low-cost ventilation and hygiene techniques used at the studio could benefit others in the trade. |
| Donovan [1994] | Lead (Pb) | Stain glass studio. | 1. Air samples in studio, home & outside.<br>2. Wipe samples in studio & home.<br>3. Soil samples studio & home.<br>4. Blood leads & Zinc Portoprophin. | 1. Air samples PBZ (studio) = o.1 to 80 $ug/m^3$.<br>2. Air samples area (studio) = <0.1 $ug/m^3$, home and outside = < 0.1 $ug/m^3$.<br>3. Wipe samples (studio) 1.2 to 1600 $mg/m^2$, (home) = <LOD.<br>4. Soil sample (studio) = 58-62 ug/g, (home) = 53-73 ug/g.<br>4. Blood samples (Pb-B) = 34 ug/dd & (ZPZ) = 31 ug/dd. | 1. See abstract by same author, same year.<br>2. Studio adjoined their home.<br>3. Engineering controls & hygiene practices account for low Pb-B and low Pb in the home. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Dolcourt [1981] | Lead (Pb) | Battery recycling | 1. Pb-B<br>2. Vacuum sample via flame AA.<br>3. Soil samples. | 1. 3 year old had 220 ug/dl.<br>2. for one family of seven (6 months - 48 years) range was 29 to 72 ug/dl.<br>3. Vacuum sample = 13 $mg/m^3$.<br>4. Soil sample results = 12% in yard to 50% in waste pile. | 1. Battery castings were burned as fuel in the one' family wood burning stove.<br>2. Illicit battery recycling out of the home kitchen. |
| Doherty [1984] | Dioxin 2,3,7,8-TCDD | CERCLA Immediate Removal Action | 1. Soil samples.<br>2. Carpet samples inside house.<br>3. Post clean up vacuuming samples. | 1. Soil - 3.0 to 48 ppb along road and drive way.<br>2. Carpet - 2.6 ppb.<br>3. <LOD for dioxin. | 1. Home owner sprayed oil contaminated with dioxin on his gravel drive & road to control dust.<br>2. Personal exposures were not measured. |
| Dolcourt et al. [1978] | Lead (Pb) | Battery factory workers family. | 1. Pb-B by AAS.<br>2. Carpet vacuum samples via AAS.<br>3. $H^2O$ via AAS (no data given).<br>4. Paint on house by X-ray Fluor. (no data given). | 1. (Pb-B) range 22 to 90 ug/dl with significant decline with increase age of children (n=58), 40 had >30 ug/dl and 10 had >40 ug/dl.<br>2. Carpets contained 1700 to 84,050 ppm of Pb. | 1. Clothing closets demonstrated particularly large degrees of contamination..<br>2. Lead contaminated work clothes was the probable source of elevated Pb-B in children of battery workers. |
| Driscoll and Elliott [1990] | Lead<br>Asbestos<br>Solvents | 1. Adhesives<br>2. Sealers<br>3. Paints production.<br>4. Asbestos brake linings mfg. | 1. Bulk dust - TEM for asbestos (plant).<br>2. PBZ air samples - PCM for asbestos.<br>3. Vacuumed samples: a) work clothes, b) car seats, via PCM & TEM.<br>4. Medical monitoring: a) chest X-ray, b) PFT, c) quest., d) Pb-B, e) ZPP. | 1. Bulk = 1 to 50% asbestos.<br>2. PBZ (n=53) 0.02 to 101.11 **(Leroy is this # correct?)** fibers.<br>3. Vacuum Samples: all clothing (n=13) was contaminated with asbestos, 11 of 13 samples from car seats (workers cars) were contaminated.<br>4. Medical results were extensive in text, n=28, 1 Pb-B was >40 ug/dl. | 1. It is postulated that much of the family members risk is a result of workers bringing contaminated clothing home for washing.<br>2. No data from homes given. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Edge and Choudhery [1978] | Asbestos | 1. Ship building.<br>2. Ship retrofitting.<br>3. Asbestos removal. | Methods are clinical. | 1. Fifty cases of malignant pleural mesothelioma from 1966-1976 in Barrow-in-furness (?). | 1. One women with mesothelioma was the wife of a shipyard plumber, who may have brought home asbestos dust on his clothes.<br>2. No other take-home related data. |
| Ehrenberg et al. [1986] | Mercury (Hg) | Thermometer and glass production for scientific use. | Flame absorption spectroscopy | 1. PBZ (n=40) 25.6 to 270.6 ug/$m^3$.<br>2. Urine Hg (n=79) 1.3 to 344 ug/g creatinine.<br>3. In NIOSH trailer (n=2) Hg in air samples = 23.4 to 21.5 ug/$m^3$. | 1. Take home not addressed in this paper but the measurements obtained in the NIOSH trailer suggest that some contamination of the trailer during the study. This also supports the possibility of offsite Hg contamination via workers inadvertently carrying Hg home on their clothes, shoes or hair, skin etc.<br>2. Issue of employees taking Hg home was addressed in a separate state health department study. |
| Eisenbud ~~et al.~~ and Lisson [1983] | Beryllium | Beryllium Production<br><br>Fluorescent lamp production | Epi study of neighborhoods near plants producing Beryllium-containing materials.<br>X-rays used for diagnosis.<br><br>Ambient air monitoring using Whatman #41 filter paper at a flow rate of 100 fpm and analyzed spectrographically | 1. Eleven persons showing non-occupational beryllium poisoning (berylliosis) were reported within vicinity of production plant. Ten were within 3/4 mile. One was two miles away from plant but was spouse to plant employee and believed to be exposed through take-home dusts.<br><br>2. Simulated home-cleaning of work clothes resulted in inhalation up to 17 ug of Be. | 1. Nonoccupational Berylliosis<br><br>2. Epi study plus ambient air monitoring lead to recommendations of ambient air average daily conc. limit of 0.01 ug/m**3. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Elwood et al. [1977] | lead | Pb smeltering in battery plant | Blood Pb via AAS<br><br>Also sampled air, dust, soil, vegetation, and tap water | Mean PbB = 6.0 ug/dl higher in children of plant workers than in otherwise comparable children<br><br>Capillary blood samples in wives of plant workers were 1.7 ug/100 ml higher than otherwise comparable wives but venous blood samples showed no significant difference.<br><br>Pb in dust, soil, and vegetation decreased in conc. w/ distance from factory.<br>3 children had PbB of 29.5, 31.0 and 33.9 ug/dl<br>boys had slightly higher PbB in all groups | The presence of a lead-worker in the household appeared to outweigh all other domestic factors such as lead pipes, cars, age of house, etc... |
| ERM Southeast Inc. [1989] | Mercury | Hg decon. of residential structures | Jerome 511 merc. vapor analyzer & dosimeter<br><br>Jerome 431 instantaneous Hg vapor monitor | Dosimeter value of 50 ug/m**3 used as clean/non-clean threshold | Article details procedure for decontamination of employee homes which were contaminated with mercury. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Ewers et al. [1982] | lead | lead smelter | Tooth lead levels from 302 German children in smelter area.<br><br>Blood leads | 1. Children of Pb workers had higher average tooth Pb and BLLs than children of non-Pb workers.<br><br>2. Tooth lead levels increased with increasing residence duration within the smelter community and with the degree of local environmental lead pollution. | 1. Correlation coef. of tooth vs. blood lead levels was 0.47.<br><br>2. Intra-indiv. variability of tooth Pb levels was low ($r=0.86$)<br><br>3. Tooth Pb levels were similar between brothers and sisters ($r=0.75$). |
| Ewers et al. [1994] | lead | Residential decontamination of carpets and floors | Repeated HEPA vacuuming to "clean" contaminated carpets. Collected dust was analyzed for Pb contamination.<br><br>Wooden floors were dry HEPA vac'd.<br><br>Linoleum floors required dry HEPA Vac followed by wet washing | Pb conc. remained about the same from initial to final carpet cleaning.<br><br>Over 95% of total dust was removed from bare wooden floors after dry HEPA vacuuming only.<br><br>Dry HEPA vacuuming removed over 75% of total dust from linoleum floors. Wet washing removed an additional 20%. | 1. Pb loading on carpet surface often increased after vacuuming because lead was brought up to the carpet's surface.<br><br>2. Tests concluded that it may be more practical to replace rather than clean contaminated carpets. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Ewers et al. [1994a] | Lead | abrasive blasting of LBP covered bridge | 1. 2 side-by-side (GA) sampling pumps at 3 locations w/in containment area.<br><br>2. BZ samples<br><br>3. Skin sampled w/moistened towelette<br><br>4. Gauze patches attached to worker for clothing simulation. | 1. Mean GA lead conc. was 1100 ug/m**3 TWA.<br><br>2. Blaster's BZ = 2000 ug/m**3<br><br>3. 82-5100 ug Pb removed from workers' hands.<br><br>4. 54 ug up to 34,000 ug total Pb contamination on gauze patches. | 1. Abstract of AIHCE presentation.<br><br>2. High variability of side-by-side GA sample results questions accuracy of traditional air sampling techniques in these environments. |
| Finley et al. [1977] | Pesticide (methyl parathion) | Cotton field work in treated fields | Cotton & cotton/polyester blends were worn by workers into a cotton field treated with methyl parathion. Samples were collected on 1st, 2nd, & 4th days after spraying. | 1. Day 1: all-cotton fabric residue concentrations were only 68% of the cotton-lend residues.<br><br>2. Day 2: Same comparison was 62%.<br><br>3. Same comparison was 49%. | 1. Report specifically concerns the absorption of residues by all-cotton vs. cotton blend garments.<br><br>2. Report also concluded that undergarments received only 55% of the outer garment residue levels. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Finley et al. [no date] | methyl parathion | Cotton field work in treated fields | Not specified | 1. "Most" pesticide residue was removed from garment after a single washing.<br><br>2. Two washings removed even more residue. | 1. Contains rehash of previous article.<br><br>2. Provides instructions for laundering to clean garments of pesticide residue. |
| Fischbein [1987] | PCB | Transformer Maint. | Serum PCB's & Adipose PCB | 1. Serum: 69 -101 ng/mL vs gen. population levels of 7 ng/mL.<br><br>2. Adipose: mean 20.7 ng/mL vs. gen. population levels of 1 ng/mL. | 1. Samples and adipose samples from 2 workers' wives also indicated possible take-home contamination of PCBs. |
| Fischbein et al. [1991] | Lead | Pottery work | Blood Pb via AAS<br><br>EP Levels<br><br>ALA-D activity | 1. BLLs:<br>worker - 48 ug/dl<br>daughter - 54 ug/dl<br>spouse - normal<br><br>2. EPs:<br>worker - elevated<br>daughter - elevated<br>spouse - normal<br><br>3. ALA-D activity was abnormal in all three. | 1. Art studio was operated out of family home.<br><br>2. Daughter (5 yr old) spent significant time in studio with artist mother. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Fish et al. [1964] | particulate | experimental re-dispersion | light-scattering particle size analyzer | Dependent upon room activity | 1. Taken from proceedings from a tech. symposium.<br><br>2. Experimental tests were non-contaminant specific. |
| Ganelin et al. [1964] | Parathion | Pesticide application | Epi case studies | Non-fatal poisonings of pesticide application workers exposed to equipment previously used for organophosphorus insecticide application. | Conclusion: All equipment which has been used for organophosphorus insecticides must be considered contaminated and dangerous until a thorough decontamination is performed. |
| Garrettson, LK [1984] | Environmental toxicity | varied | Epi case histories | varied | 1. Due to incomplete organ development in children, there is potential for effects on maturation that are not seen in adults.<br><br>2. Children between 9 mo. and 3 yrs. old account for more than 50% of poison center calls.<br><br>3. Article discusses home poisons as well as take-home toxins. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Giarelli et al. [1992] | asbestos | "naval" work | 1. Epi case studies | 1. 170 cases of asbestos-related fatalities were examined. | 1. 150/170 cases indicated work histories with occupational exposure to asbestos.<br><br>2. median interval between initial exposure was 48 years. Range (14-71).<br><br>3. malignant pleural mesotheliomas (fatal) |
| Gibbs et al. [1990] | asbestos (chrysotile, crocidolite, amosite) | Military Gas Mask Production<br><br>paraoccupational exposure | Epi study of 10 paraoccupational cases | Fatal malignant mesothelioma | 1. malignant mesothelioma<br><br>2. 9 of 10 paraoccupational cases were considered to have developed their diseases due to exposures to asbestos on husbands clothes. |
| Gibbs et al. [1989] | asbestos | nonoccupational or unknown exposures | mineral content of 84 fatal cases was estimated through electron microscopy and energy dispersive X-ray analysis. | Lung burdens for chrysotile and amphiboles were estimated. The amounts of chrysotile appeared similar for both mesotheliomas and control populations. | 1. non-occupational malignant mesotheliomas.<br><br>2. Study supports findings that amphiboles are more important than chrysotile in the causation of malignant mesothelioma. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Gittleman et al. [1991] | Lead | Battery Recycling | 1. BLLs<br><br>2. Aerosols (RAM)<br><br>3. Wipe samples in employee autos. (Whatman smear tabs) | 1. 93% of tested employees had BLLs above 40 ug/dl (range 52-86)<br><br>2. RAM: 40-400 ug/m**3<br><br>3.<br>- 3mg Pb on drivers seat<br>- 1.9 mg on driver's floor area<br>- 1.7 mg on dashboard | NIOSH HHE. |
| Goldman and Peters [1981] | non-specific | nonspecific | 1. Survey patient<br><br>2. ID potential sources of exposure.<br><br>3. ID types and use of toxic substances used.<br><br>4. Follow-up & resolution. | non-specific | Article provides sequence of steps to facilitate physician recognition of occupationally related diseases. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Grandjean and Bach [1986] | UV Radiation<br><br>Asbestos<br><br>nonspecific | welding<br><br>ship building<br><br>domestic tasks | Symptom Survey<br><br>Epi studies<br><br>Epi studies | "welder's eye" often occurs among bystanders not req'd to wear eye protection<br><br>asbestosis<br><br>elevated BLLs & asbestosis | 1. Article emphasizes importance of considering indirect or bystander exposures as a source of occupational exposures.<br><br>2. Details cases of asbestosis among ship builders not id'd as asbestos workers.<br><br>3. Discusses spouse exposure to take-home contaminants during laundry tasks. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Gunter et al. [1987] | lead | Fire assay procedures | 1. BLLs<br><br>2. FEPs<br><br>3. physicals & questionnaires<br><br>4. air sampling for Pb dust & fume | 1. Of 10 workers sampled, all were within OSHA reg. limits of 50 ug/dl however 3 were over the 40 ug/dl back-to-work level.<br>2. 3 FEP conc. exceeded "normal" levels of 50 ug/dl.<br><br>3. Physicals and questionnaires revealed no positive physical exam findings of excess lead exposures.<br><br>4. Air sampling:<br>GA - 0.1 to 0.6 mg/m**3<br>BZ - 9/14 BZ samples exceeded eval. criteria (range - 0.01 - 0.49 mg/m**3) | NIOSH HHE (HETA 86-438-1795) |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Hardy [1965] | Beryllium | fluorescent lampworker | Epi studies<br><br>Chest X-rays | Recorded 210 fluorescent lampworkers w/beryllium disease (129 living, 81 dead). | 1. Contains substantial text praising the works of Dr Alice Hamilton and providing historical perspective on the development of American Occupational Health.<br><br>2. Criticizes US PHS and US medical industry's slow recognition of Be health effects given earlier reports of Beryllium-related worker illnesses in Europe. |
| Hartle [1987] | PCB | Rail Yard Workers | GA air samples using 150 mg dual-section florisil tubes at 0.6-1.0 L/M<br><br>Wipe samples w/hexane moistened gauze pads | 1. Air Samples:<br>- 27 below LOQ<br>- 18 above LOQ w/mean = 0.5 ug/m**3.<br><br>2. Wipe samples from bottom of maint. pit averaged 90,000 ug/m**2 of PCB. | 1. NIOSH HHE<br><br>2. Evaluation site did not allow HHE investigators to perform personal interviews, BZ sampling, or wipe sampling of personal clothes.<br><br>3. All air results were below NIOSH REL of 1.0 ug/m**3. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Hartle et al. [1987] | PCB | Alum. extrusion using PCB contaminated hydraulic fluids | Environ. air samples (BZ & GA)<br><br>Wipe samples<br><br>Serum PCBs | BZ results up to 2 ug/m**3<br><br>wipe sample results up to 900 ug/m**2<br><br>Serum PCBs were not elevated | 1. NIOSH BZ REL is 1 ug/m**3<br><br>2. NIOSH wipe sample proposed limit of 50-100 ug/m**2.<br><br>3. NIOSH HHE |
| Hasan and Hamayoun [1974] | Beryllium | aviation industry<br><br>nuclear industry<br><br>gyroscope production | X-ray<br><br>biopsy<br><br>autopsy<br><br>PFT | Physical and chemical analyses were positive for Be disease. | 1. Article produces 5 criteria for the diagnosis of Beryllium disease.<br><br>2. Reports case histories of 76 cases entered into the Beryllium case registry since 1966. |
| Hatch [1990] | mercury | gold ore extraction | Jerome Hg vapor analyzer | Only 3/26 samples in the home were significant and these 3 were taken at source locations for Hg storage. (ie... storage bag, mixing pot, and dispensing ladle) | 1. Hobby-related exposures<br><br>2. Patient twice diagnosed for Hg poisoning. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Hofstetter et al. [1990] | lead<br><br>cadmium | lead smelter | BLLs<br><br>Blood Cad. Levels | BLLs ranged 2.6 - 15.5 ug/dl (mean = 6.3 ug/dl)<br><br>Cd Blood Levels ranged <0.1 - 0.5 ug/l (mean = 0.14 ug/l) | 1. 229 children in study<br><br>2. Environmental pollution |
| Hess [1988] | Dioxin | Dust-suppression w/contaminated waste oil | TCDD-soil<br><br>TCDD-Hi-vol air filters | soil: 36 ppb l<br><br>air samples: BDL | Very large environmental restoration project began in 1983 not yet completed as of article's writing. |
| Huncharek et al. [1989] | asbestos | Domestic exposure to spouse | Physical exam<br><br>biopsy<br><br>post mortem fiber counts from removed lung | 1. malignant mesothelioma w/extension to mediastinum, pericardium, and left chest<br><br>2. <u>f/g of wet lung tissue</u><br>- chrysotile 1.72 x 10**3<br>- amocite/crocidolite 59 x 10**3<br>- tremolite/actinolite/anthophyllite 221 x 10**3 | 1. Husband was a shipyard machinist.<br><br>2. Spouse exposed through contaminated take-home laundry. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Hung [1980] | Lead | Battery processing in home | physical exam<br><br>BLLs | lead encephalopathy in 2 children<br><br>16 mo. old child-BLL 100 ug/dl<br><br>2.5 yr old - BLL 86 ug/dl<br><br>3yr 8mo - BLL 124 ug/dl | Follows case history of Pb poisoning of 2 families in Taiwan. |

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| Elwood et al. [1977] | lead | Pb smeltering in battery plant | Blood Pb via AAS<br><br>Also sampled air, dust, soil, vegetation, and tap water | Mean PbB = 6.0 ug/dl higher in children of plant workers than in otherwise comparable children<br><br>Capillary blood samples in wives of plant workers were 1.7 ug/100 ml higher than otherwise comparable wives but venous blood samples showed no significant difference.<br><br>Pb in dust, soil, and vegetation decreased in conc. w/ distance from factory.<br>3 children had PbB of 29.5, 31.0 and 33.9 ug/dl<br>boys had slightly higher PbB in all groups | The presence of a lead-worker in the household appeared to outweigh all other domestic factors such as lead pipes, cars, age of house, etc... |
| ERM Southeast Inc. [1989] | Mercury | Hg decon. of residential structures | Jerome 511 merc. vapor analyzer & dosimeter<br><br>Jerome 431 instantaneous Hg vapor monitor | Dosimeter value of 50 ug/m**3 used as clean/non-clean threshold | Article details procedure for decontamination of employee homes which were contaminated with mercury. |

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| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Jensen et al. [1972] | Tetrachlorobenzo-dioxin | trichlorophenol plant | | Two employees, one employee's son & other employee's wife developed chloracne. | Contamination of equipment resulting from explosion years earlier. The probable source of son's exposure was noted to be embracing his father while he still had his work clothes on. |
| Joshua et al. [1971] | Lead | Cottage industry: extracting gold & silver from jewelers waste. | Medical monitoring | All members of a family living in India, 9 adults & 9 children, were affected. Three of the children died. | This article contains a lot of clinical information. The author notes that the family refused to recognize the hazard and did not take any corrective action. |
| Jung [1984] | Lead | self employed painter | Medical monitoring | Painters level was 29.9 ug/dl, as reported to the Ct State Health Dept. | The painter was hospitalized the previous summer as a psychiatric admission. At the time, a heavy metal workup was not performed. |
| Katagiri et al. [1983] | Lead | Pottery mfg in Japan in factories and homes. | Medial monitoring Three groups of children compared: 1) home pottery workers, 2) factory pottery workers & 3) nonpottery. | Children of family pottery workers had the highest Pb-U levels. Children of factory pottery workers had higher exposure than non-pottery worker children. | The results seen for children was not present in the parents. Therefore parents Pb-U levels should not be considered representative of child's potential exposure. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Katzenellenbogen [1956] | Stilbestrol | Production of stilbestrol in pharmaceutical factory. | | 1. Six children (4 boys) with signs & symptoms resulting from exposure.<br>2. One or both of parents worked with stilbestrol.<br>3. Health effects reduced via decrease in dust level and PPE use. | Also investigated 4 other factories where 11 other affected workers were identified.<br><br>Recs: 1) PPE including gloves & resps. 2) Ventilation, 3) changing work clothes & showering facilities, 4) Designated work clothes, & 5) medical surveillance of workers. |
| Kawai M [1983] | Lead | Cottage industries in Japan: quench-hardening & type-printing. | Air & surface sampling of dust. Pb identified via AAS. | Air: 2 to 50 ug/$m^3$<br>Surface: 260 to 20,386 ppm of Pb in total dust. | Evidence provided that exposure risk is higher for children because of habits. Rec - medical monitoring of children of households conducting lead-work. |
| Kaye et al. [1987] | Lead | Electronics components plant making ceramic-coated capacitors & resistors. | Company data = (air) 61 to 1700 ug/$m^3$. Samples of dust from vacuums of workers & controls were collected. | Lead was detected in 10/11 samples form workers, ranging from ND to 3400 ppm and 2/9 for controls, ranging from ND to 320 ppm. | Concentrations from workers homes was significantly higher than controls. |

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| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Kelly [1994] | Lead | Mfg of pewter products | Dust samples from carpets, window sills, automobile, soil & exterior surface dust. | Up to 25,000 ppm. | Low Pb concentrations outside of home & high levels inside, led the author to conclude that source of exposure was the workplace. |
| Kiefer [1994] | Lead & solvents | Renovation of an antique residence. | Bulk paint analysis for Pb, surface wipes for Pb dust, PBZ air sampling for Pb. | Paint contained 0.19% to 0.28% Pb. Surface levels ranged from 47 to 158 ug/ft$^2$. PBZ levels were at or below the LOD. | This was a renovation situation where exposures will depend on whether or not occupants are in the residence during renovation, and the effectiveness of cleaning procedures used following renovation. |
| Kilburn et al. [1985] | Asbestos | Shipyard workers | Chest X-rays | | |
| Kilburn et al. [1986] | Asbestos | Shipyard workers | Chest X-rays | | |
| Klemmer et al. [1975] | Arsenic | Pesticide application or pretreatment of lumber with preservative chemicals. | Concentration of As in household dust from vacuums after filtering through a 0.246 mm size sieve. Analyzed via spectrophotometry. | 1.1 to 1,080 ug As/g of sieved dust. Study compared homes which had treated wood or pesticide application to homes that had neither (self-reported). | Questions were asked regarding occupational use of pesticides. Extremely high cases, > 100 ugAs/g dust, were associated with occupational exposure. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Klorfin and Bartine [1956] | Stilbestrol | Pharmaceutical factory producing diethylstilbestrol. | Medical tests | Six or 7 cases of stilbestrol intoxication identified, 2 of which were workers' children. | 1 8-yr old child's mother (also a case) worked in factory. Second child was 10-yr old who's father was a pharmacist in a factory. |
| Kominsky [1987] | PCBs | Structural firefighting that occurred at a transformer oil reclamation facility. Tested the effectiveness of a commercial dry cleaning system to decontaminate turn-out gear. | PCBs extracted from pre-wash samples using toluene. Samples of clothing from incident-contaminated clothing & "spiked" clothing. Analysis via GC with ECD. | Cleaning efficiency was 66 to 99% for incident-contaminated and >90% for "Spiked" samples. | The commercial system used, RADKLEEN$^{tm}$, used 1,1,2-trichloro-1,2,2-trifluoroethane (Freon 113). |
| Kominsky et al. [1990] | Asbestos | Testing the effectiveness of wet & dry vacuuming to remove asbestos fibers from contaminated carpets & also determine airborne levels during vacuuming. | TEM was used to analyze air samples collected during vacuuming. An EPA method (reference #1) was used for analysis of carpet samples. | Wet vacuuming was ~70% effective. There was no significant evidence of change following dry vacuuming. | |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Koplan et al. [1977] | Lead | Cottage industry of pottersin Barbados, West Indies. | Pb concentrations in dust samples which were collected using gauze pads. Pb release from pottery samples analyzed via AAS. | Pb in dust ranged from 393 ug/g from a floor in the DR in one home, to 325,892 ug/g from a table surface where glazing was done. Release levels ranged from 7 to 3,125 ug/ml from the pottery samples. | |
| Lander F Viskum B [1985] | ? | ? | ? | Article in foreign language (not translated). | |
| Landrigan et al. [1980] | Lead | Stained glass window production, in commercial studios (not in home). | Pb-B levels for workers & family members. Pb levels in bulk-dust samples in 1 home & 1 studio. | Mean Pb conc in studio (3 samples) was 10,696 ppm. Mean conc in home (2 samples) = 355 ppm. | |
| Lavy [1988] | Herbicides | | | | This is an informative chapter from a book directed at consumers or workers who apply pesticides. Included in this are recs. for preventing contamination of the home. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Lehmann [1977] | Asbestos, vinyl chloride, arsenic | 1. Asbestos plant<br>2. Vinyl chloride prod<br>3. Copper mines. | 1. X-rays.<br>2. Epi<br>3. Epi | One asbestos case is a daughter who took lunch to her father each day. | Authors note that mesotheliomas have occurred among laundry workers handling asbestos contaminated clothing. They stress the need for controlling dust at its source. |
| Lehmann [1905] | Chlorine-tar derivatives (?) | Electrolytic decomposition of saturated salt solution for lime chloride prod in German plant. | | | |
| Lewis et al. [1994] | Pesticides | | | | Not take-home related. Exposure sources were to commercial home treatments or household use. |
| Lindbohm et al. [1991] | Lead | No specific process | Pb-B levels | | Exposures were quantified using historical Pb-B levels in workers, & self reporting of workplace and occupation by workers & their wives. |
| Litzistorf et al. [1985] | Fibers | | | | Not related to take home problem. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Lundquist [1980] | Lead | | | | Article explains the issues of take-home lead in plain English. |
| Lyngbye et al. [1991] | Lead | Danish auto mechanics & shipyard workers | Pb in the teeth of children | | Increased concentration seen in children of 3 groups, 1) lower social class, 2) fathers were auto mechanics or shipyard workers 3) And those who lived in areas with heavy traffic. |
| Lyngbye et al. [1991] | Lead | Danish auto workers & shipyard workers | Pb in the teeth of children | | Increased concentrations seen in children whose fathers were auto repair, auto painting, or shipyard workers; and who lived in areas with heavy traffic. Pica & maternal smoking during pregnancy were also associated with higher concentrations. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Magee et al. [1986] | Asbestos | Mining? | Asbestos fibers in tissue sample | A case report of a 41 yr old man diagnosed with mesothelioma. | Self reporting indicated exposure (asb) occurred from birth to age 11, when he lived in Corsica near the Canari Mine. His parents were not miners, but his father operated a pub that mine workers visited. He & his family, took ore from the workers, trucks - to filter wine. |
| Maravelius et al. [1989] | Lead & Cadmium | Mining & smelting in Greece | Blood Pb & Blood Cd levels | Children of unskilled workers had sig. higher Pb-B than other children. | Parents of children with high PB-B worked in lead-zinc mining & smelting complex. |
| Martin [1974] | Lead | Lead works in United Kingdom | Blood Pb | 3 children with highest Pb-B (75, 74, & 65 ug/dl. Fathers of 2 worked at lead facility. | Wide spread contamination of air & soil near lead works. |
| Martin et al. [1974] | Lead | Various sites in United Kingdom e.g., smelters, lead & other mines. | Blood Pb | Levels highest for family members living close to Pb industries. | Levels also high for family members of lead workers even if home not close to industry. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Masek et al. [1972] | 3,4 benzo(a) pyrene | Pitch coking plant in Czechoslavakia | 3,4 benzo(a) pyrene in workers clothing | Up to 11 ppm in underwear & up to 350 ppm in trousers & shirts. Post washing analysis of clothing shows that 3,4 benzo(a)pyrene residue stays in clothes. | Author states that washing method does not ensure effective removal of contaminant. Method describes as "usual method". Clothes washed on site - not taken home. Thus not a THT hazard, but could be if clothes had been washed at home. Author states that contaminated clothes are a source of dermal exposure to workers. |
| Matte and Burr [1989] | Lead | Cottage industry in Jamaica: "backyard battery repair shop". | Air, dust, soil & blood Pb samples. | Air: GM = 21 ug/$m^3$, Dust: range = 190 to 53,140 ug/$m^3$. Soil: 51 to 400,000 ppm. | Authors state that "residential exposure occurs mainly by contamination of soil & house dust". |
| Matte et al. [1991] | Lead | Same study | Same study | Same study | Peer reviewed journal article of same study. Changing work clothes was not associated with lower house dust Pb levels. Possible due to adequate shower and changing facilities were not provided. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Matte et al. [1991] | Lead | Conventional & cottage lead smelters in Jamaica. | Dust, soil, peeling home paint, & blood Pb samples. Two residential areas close to smelter sampled: one with cottage smelters & one without. | Dust: 20 to 294,680 ug/$m^2$. Soil: <5 to 520,000 ppm. Paint: up to 6% Pb.<br><br>Soil & dust samples were significantly higher in the area with cottage smelters. The measured Pb levels were higher than those predicted based on data from developed countries. | Authors suggest that this higher levels might be due to presence of cottage smelters & the location of the 2 residential areas relative to the prevailing wind direction. Higher levels than models predicted (explanations include difference in time spent outdoors, hygiene & nutrition) suggest that environmental criteria in developed countries may not be protective in developing countries. |
| May [1973] | Tetrachlorodi benzo dioxin | Production of 2,4,5-trichlorophenol (2,4,5-TCP) in plant in Great Britain? | Clinical & laboratory medical tests. | Chloracne resulted from exposure to tetrachlorodibenzodioxin (formed during exothermic reaction at plant). | No cases of disease among relatives. |
| McCammon et al. | Lead | Lining of 2 large tanks with lead sheets. | Wipe samples for surface Pb inside the workplace, including change room, employee street shoes & in employees' cars. | <1 to 60 ug Pb/$cm^2$. Results indicate that Pb was taken home & thus, a potential exposure to family members. | Per authors "The opportunity for Pb exposure was likely increased by the lack of shower facilities & the practice of wearing work clothes home". Recs: 1) Install clean & dirty change rooms &<br>2) Laundry facilities at work. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Menrath et al. [1993]) | Lead | Mining | Dust samples collected from the cars & homes of both miners & non-miners in the same community. | Mean Pb in miners cars = 3909 ppm, vs 917 ppm in non-miners cars. Lead loadings were similar for two groups, ~56ug/ft$^2$. Concentration of Pb in house dust was higher for miners. | Information from abstract in presented at the American Industrial Hygiene Conference & Exhibit (1993). Thus, few details provided of methods, results, etc. Authors state, "data suggests that take worker carry-home is not a major area of concern at the present time, but is an area where some vigilance is warranted". |
| Miesen [1991] | Lead | Lead & silver plants. | Blood lead | | Living with an occupationally exposed worker was recognized as a risk factor. Also recognized was drinking water in homes with lead pipes. |
| Milar and Mushak [1982] | Lead | Not identified | Lead conc in dust inside of homes of an occupationally exposed resident & in homes of non-exposed residents. Also Pb-B. | Mean dust conc for homes of occupationally exposed resident = 3000 ppm, vs mean for non-exposed resident's homes = 250 ppm. | Blood lead levels were higher for children in homes with an occupationally exposed resident. The author suggests that dust levels of > 1000 ppm represents a health hazard for children. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Millar [1978] | lead | Lead works in England | Blood leads in children. | Mean Pb-B was significantly higher for children of Pb workers than for children of non-Pb workers. | |
| MMWR [1992b] | Lead | Automobile- & industrial-battery reclamation. | Blood lead levels of workers' children & controls. | The Pb-B levels were significantly higher in children of lead workers than in other children. | |
| MMWR [1989a] | Lead | | | | This MMWR article covers the same study as Matte & Burr (1989). |
| MMWR [1985] | Lead | Mfg capacitors & resistors. | | | This MMWR article covers the same study as Kaye et. al. (1987). |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| MMWR [1977b] | Lead | Battery factory | Blood leads: Pb conc in air, dust, paint & drinking water from 7 homes of children with the highest Pb-B levels. Also soil samples from cars that were driven to work. | No Pb contamination in paint or water and no airborne lead exposures from factory emissions or busy roadways. Dust conc from 1,695 to 84, 074 ugPb/g. Highest level were from closet where work clothes were stored. Mean Pb level in soil from cars was 2,770 ugPb/g. | The employer made changes to reduce worker & family exposures, including exhaust ventilation, providing coveralls & improved shower facilities. This study was the third report to CDC, over 2 years, of lead absorption in children of lead-exposed workers. In all 3 cases, lead dust carried home on contaminated work clothes was the apparent source of exposure. Significant lead absorption has been associated with young children exposed to lead in dust at levels of > 1000 ugPb/g. |
| Molina-Ballesteros [1979] | Lead | Pottery making | Blood-Pb | Positive Pb-B in all ages - male & female | Community population studies |
| Molina-Ballesteros et al. [1983] | Lead | Pottery making | Bld-pb | Elevated Bld-pb | Exposed children in home work shops vs control-children in homes w/o pottery making. Recommend controls: (1) separate glazing/baking from family access; (2) Eliminate letharge from process |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Morton et al. [1902] | Lead | Battery Making | Bld-pb | Elevated Bld-pb in "exposed" vs "controls" | Exposed children (fathers in battery mfg) vs control-children (fathers in non-lead occupations)<br><br>Recommend Controls: Good personal hygiene - showering, shampooing, changing clothes and shoes before leaving work was effective for lead contaminant. |
| Natarajan [1994] | Lead | Radiator repair (RR) | Blood Pb<br>Air Pb<br>Wipe Pb | Elevated blood & air Pbs; positive surface wipes | 3 RR workers & areas showed contamination - no evaluation at workers homes conducted |
| Nelson and Clift [1992] | Lead | Foundry | EPA 3050 | Pb detected in all homes | Homes sampled (clothing, dust, soil, water) of foundry & non-foundry workers. Lead smelter emissions likely source for home Pb contamination. |
| New Jersey Dept Health [no date] | Lead | Various Pb industries | Blood Pb | Parent 29-62 ug/dl<br>Children 0-26 ug/dl | Small pilot study.<br>No exposure data homes obtained. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Newhouse and Thompson [1965] | Asbestos | - | | | Hospital patients w/o occupational exposure, but likely domestic exposure to asbestos, diagnosed mesotheliomas. Exposure likely from washing ACC, living near asbestos factory. |
| Nicholson et al. [1980] | Asbestos | Environmental Contamination | Air Sampling | 43 of 89 Chrysotile asbestos samples > 50 ug/$m^3$ | Samples collected in public schools, outdoor air, construction sites, workers homes. |
| Nicholson [1983] | Asbestos | - | - | Asbestos ubiquitous in ambient air - data for U.S. Schools - 10 to 2000 ng/$m^3$; homes of workers - 50 to 5000 ng/$m^3$. | Cancer risk of non-occupational population in U.S. A review. |
| NIOSH [1973] | Mercury | Various | | | Homes contaminated from workers clothes, boots. Recommendations: shower, clothes change, work practices - food and tobacco exclusions in the work place. |
| Oern [1991] | asbestos | Asbestos insulator | | Female exposure from laundering husbands clothes soiled by asbestos. | Familial asbestos disease - mesothelioma. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Orris and Kominsky [1984] | PCBs | Transformer failure | Air & wipe samples at a school | School vault: PCB in air 50-90 ug/$m^3$, on surface 100-5000 ug per 100 $cm^2$. | Transformer fire at a school - fire fighters exposed. No assessment of worker home contamination. School PCB levels measured. |
| Osorio [1994] | Lead Pesticides | Various | Method developed to measure 12 pesticides in house hold dust. | Lead - 5 case studies showed contaminated work clothing taken home contributed to Blood Pb elevations.<br><br>(Example Organophosphate in farm worker homes.) | California DH reports of THT studies including lead and pesticides. |
| Otte et al. [1990] | Asbestos | Cement Mfg | | No exposure data | Family mesotheliomas from home production of asbestos cement. |
| Pacynski et al. [1971] | Stilbestrol | Manufacturing | | No exposure data | Hyperestrogenism is workers and children. Stilbestrol exposure from workers clothing taken home and through pregnancy. |
| Pacynski and Robacznski [1968] | Stilbestrol | Manufacturing | | No exposure data | Hyperestrogenism in female workers during stilbestrol mfg. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Pasanen et al. [1989] | Fungi | Farming | Impactor sampling - cultivation; SEM | $10^3$ to $10^4$ CFU/$m^3$ | Airborne fungal contamination carried from cow barns to farmers homes. |
| Piacitelli [1993] | Lead | Radiator Repair | NIOSH 7105, 0700 - air & wipe samples | Air - 15.2 ug/$m^3$ Wipe - up to 500,000 ug/$m^2$. | Lead contamination documented in radiator repair shops; no home contamination assessment. |
| Piacitelli et al. [1994] | Lead | Bridge work. | NIOSH 7105 wipe & vacuum samples. | Auto surfaces (floor, seats, arm rests) up to 2485 ug/$m^3$ & 814 ppm lead. | Lead contamination documented in automobiles of bridge workers. Poor hygiene - contaminated clothing. |
| Piccinini et al. [1986] | Lead | Tile workers | Blood-Pbs | | Lead workers' children had higher B-Pb than control children. No home contamination assessment. |
| Pitts [1986] | Lead | Radiator repair | Blood-Pbs, lead in dust at home. | 183-11,030 ug lead detected in home. | Child lead poisoning. Lead contamination documented at home. |
| Pollock [1994] | Lead | Trucking lead/products | Blood-Pbs, lead in dust at home | 20-240 ug/$ft^2$ | Child lead poisoning, lead contamination documented at home. |
| Price and Welch [1972] | PCB | General population | GC TLC | | PCB analysis in human tissue. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Que Hee et al. [1985] | Lead | | Method development for lead sampling of house dust | | No home contamination assessment. |
| Quinn [1985] | Lead | | | Blood lead of lead workers' children in UK generally higher than controls. | No home contamination assessment. |
| Ramakrishna et al. [1982] | Lead | Gold-silver jewelry waste recovery | | Blood lead of family members elevated - cottage industry in Srilanka | No home contamination assessment. |
| Rice [1978] | Lead | Lead smelter | Wipe/dust samples | Towels - 79 ug, settled dust - 3.3 ug/$m^3$. | 1. Smelter workers' home contamination documented vs controls.<br>2. More stringent work practices & personal hygiene are required. |
| Richter et al. [1985] | Lead | Battery mfg | Blood-lead, ZPPs | Wives/children of battery plant (HAIFA-**Jerry what is this?**) workers had elevated lead levels vs controls. | No home contamination assessment |
| Richter et al. [1980] | Lead | Israel Population | Blood lead, ambient lead | PVC workers children had elevated Pb-B; levels higher in worker group not changing work clothes. | No home contamination assessment. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Rinehart and Yanagiswa [1993] | lead, tin | Cable splicers | House dust samples - X-ray fluorescence | Laundry area - 1021 ppm lead; other areas - 585 lead. | Home contamination documented elevated lead and tin in cable splicers vs controls. |
| Schneider et al. [1989] | Non-specific dust. | | Surface dust method development via light scattering | | |
| Schneider [1986] | Man made mineral fibers, other fibers | Method development for settled fibers | | | Fiber measurement in schools. |
| Schreiber [1993] | Tetrachloroethlene (PCE) | Dry cleaning | | Pharmacokinetics modeling | Estimates of infant exposure to PCE in breast milk. |
| Schuhmacher et al. [1991] | Lead | | Hair lead | Exposed - 12.69 vs controls - 8.43 ug/g | Lead workers' children (Spain) hair lead higher than in controls. |
| Seixas and Ordin [1986] | Asbestos | Brake shoe mfg. | Chest X-rays; air & clothing samples for asbestos. | Clothing positive for asbestos. | Potential for home contamination shown by asbestos on workers clothing. |
| Sherlock et al. [1985] | Lead | UK population - children | Blood lead | No difference among 3 groups, but children who washed hands before eating had lower Pb-B. | Blood lead level in children of different ethnic origin measured. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Silvany-Neto [1989] | Lead | Smelter | Blood lead | Blood leads slightly reduced after improvements, but highly contaminated soil a long-lasting risk factor. | Blood lead in children living around smelter assessed, before and after changes to reduce pollution from smelter. Children of lead workers had highest Pb-Bs. |
| Simonson and Mecham [1983] | Lead | Smelter | | Air shower removed 5 to 72% of lead from clothing. | Evaluation of controls for lead removal: air showers & automatic shoe cleaners. |
| Stasiuk [1993] | PCE | Dry cleaning | | PCE concentrations ranged from 15 to 197,000 $ug/m^3$ (most over 100 $ug/m^3$. | PCE exposures in residences located in building with dry cleaners. |
| State of Alabama [1992] | Lead | Residence | Wipe samples | Wipe samples in mobile home = 16 to 390 $ug/ft^2$. | Elevated Pb-Bs in children living in mobile home adjacent to auto repair & pottery shop. |
| Stewart [1967] | Particulate | | | | Study of parameters affecting re-suspension of particulates from surfaces. |
| Trost [1985] | Mercury | Thermometer plant. | | | Elevated mercury in children of thermometer plant workers. Mercury transport via worker clothing/shoes to homes. |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Venables and Newman-Taylor [1989] | 1) Rat 2. Platinum salts (?) | | | | Asthma related to spousal occupation. Relived after pet removal & change of work, shower & change of clothes. |
| Venable et al. [1993] | Lead | Cable Splicing | Air, wipe, Blood lead, ZPP | Electrical transmission workers evaluated for occupational Pb exposure | No home contamination assessment |
| Versen and Baun [1989] | Crystalline silica | Laundering of clothes | Air sample - NIOSH 0500 & 0600 | $SiO_2$ - ND-LOD | $SiO_2$ inside vs outside home showed no difference in concentration. |
| Wang et al. [1989] | Lead | Taipai population | | Parental Pb-B, cord Pb-P associated with lead soldering, washing clothes at home. | |
| Watson et al. [1978] | Lead | Battery mfg | | Households of workers had higher concentrations of lead in dust (2239 ppm) vs controls (718 ppm). | Battery plant workers' children had higher Pb-B & EPP vs controls. |
| Weeks et al. [1976] | OSHA carcinogens | | Development of chemical spot tests (visualization & uv detection) | Low levels (ng) on surfaces (metal, paint, concrete). | |

Table 15. Continued Industrial Hygiene Review of Take-Home Exposure - Industrial Hygiene

| Author (year) Location | Contaminant | Process | Sampling Method | Results/ Concentrations | Comments |
|---|---|---|---|---|---|
| Whitwell et al. [1977] | Asbestos | | | Hospital patients with mesothelioma related to industrial asbestos exposure; lung fiber counts were not higher in patients who had lived near asbestos pollution sources. | |
| Wiehrdt [1994] | Lead | | | | Reference to home contamination cases (3) known to OSHA. |
| Winegar et al. [1977] | Lead | Smelter | Blood Pb, ore, dust | Household dust (pb) - 120 to 126,000 ppm (2,400 ppm median. | Smelter workers and household contacts evaluation for Pb poisoning, and contamination of household. |
| Woody et al. [1986] | RDX | Explosive plant | | | Apparent ingestion of RDX from boots/clothing worn home, resulted in child neurotoxic effects. |

Notes: * Results of other tests (hematocrit, total erythrocytal porphyrins, aminolevulinic acid, etc.) reported in interim paper.

Abbreviations & acronyms: PB-B = Blood Lead, Pb-U = Lead in Urine, Pb-H = Lead in hair, ECD = Electron Capture Detector, GC = Gas Chromatography, FID = Flame Ionization Detector

**Table 16. Federal Laws Relevant to Take-Home Exposure**

| Popular Name | Public Law | U.S. Code | Sections Relevant to Workers' Family Protection |
|---|---|---|---|
| Occupational Safety and Health Act of 1970 | Public Law 91-596 | 29 U.S.C. 651 & Seq. | §651 Congressional Statement of Findings and Declaration of Purpose and Policy<br>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 . . . .<br><br>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.<br><br>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.<br><br>§654 Duties of Employers and Employees<br>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.<br><br>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.<br><br>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.<br><br>§669 Research and Related Activities<br>§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.<br><br>§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 . . . .<br><br>§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.<br><br>§671 The Institute is authorized to - (1) develop and establish recommended occupational safety and health standards. |

**Table 16. Continued Federal Laws Relevant to Take-Home Exposure**

| Popular Name | Public Law | U.S. Code | Sections Relevant to Workers' Family Protection |
|---|---|---|---|
| Federal Mine Safety and Health Act of 1977 | Public Law 91-173<br>Public Law 95-164 | 30 U.S.C. 801 & Seq. | §802 Definitions<br>(g) "Miner" means any individual working in a coal or other mine;<br>(h)(1) "coal or other mine" means (A) an area of land from which minerals are extracted in nonliquid 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 nonliquid 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;<br><br>Note 3. Coal or other mine<br>Definition of "coal mine" under subsection (h) of this section includes a commercial purpose requirement. C.A. 3, 1984, 748F. 2d176.<br><br>Note 5. Miner<br>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.<br><br>§803 Mines subject to coverage<br>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.<br><br>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.<br><br>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.<br><br>Provisions of this chapter are applicable even though owner operators work the mine. D.C. PA. 1980, 491F, Supp. 1123.<br><br>Owner-operated mine is not outside provisions of this chapter. D.C. PA. 1980, 487F, Supp. 1376.<br><br>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. PA. 1978, 465F, Supp. 838. |

**Table 16. Continued Federal Laws Relevant to Take-Home Exposure**

| Popular Name | Public Law | U.S. Code | Sections Relevant to Workers' Family Protection |
|---|---|---|---|
| | | | §813 Inspections, investigations, and recordkeeping<br><br>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.<br><br>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 implicably consented to warrantless inspections of his quarry by representatives of the Secretary of Labor pursuant to this chapter. C.A. Cal. 1980, 628F, 2d1255.<br><br>§877(l) 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.<br><br>§951 Studies and research<br>(a) [The Secretary of Health and Human Services shall conduct studies]<br>(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[.]<br>(12) for such other purposes as . . . . deem[ed] necessary to carry out the purposes of this chapter.<br>(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 . . . .<br><br>(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. |

**Table 16. Continued Federal Laws Relevant to Take-Home Exposure**

| Popular Name | Public Law | U.S. Code | Sections Relevant to Workers' Family Protection |
|---|---|---|---|
| Toxic Substances Control Act | Public Law 94-~~469~~ 469 | 15 U.S.C. 2601 & Seq. | §2602 Definitions<br>(2)(A) Except as provided in subparagraph (B), the term "chemical substance" means any organic or inorganic substance of a particular molecular identity, including —<br>(i) any combination of such substances occurring in whole or in part as a result of a chemical reaction or occurring in nature and<br>(ii) any element or uncombined radical.<br>(B) Such term does not include—<br>(i) any mixture,<br>(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,<br>(iii) tobacco or any tobacco product,<br>(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),<br>(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<br>(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.<br><br>(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.<br><br>(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.<br><br>(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.<br><br>(10) The term "process" means the preparation of a chemical substance or mixture, after its manufacture, for distribution in commerce-<br>(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<br>(B) as part of an article containing the chemical substance or mixture.<br><br>(11) The term "processor" means any person who processes a chemical substance or mixture. |

**Table 16. Continued Federal Laws Relevant to Take-Home Exposure**

| Popular Name | Public Law | U.S. Code | Sections Relevant to Workers' Family Protection |
|---|---|---|---|
| | | | § 2605. Regulation of hazardous chemical substances and mixtures<br><br>(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:<br><br>(6)(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.<br><br>(7) A requirement directing manufacturers or processors of such substance or mixture<br>(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,<br>(B) to give public notice of such risk of injury, and<br>(C) to replace or repurchase such substance or mixture as elected by the person to which the requirement is directed.<br><br>§2607 Reporting and Retention of Information<br>(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.<br><br>(e) 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. |

**Table 16. Continued Federal Laws Relevant to Take-Home Exposure**

| Popular Name | Public Law | U.S. Code | Sections Relevant to Workers' Family Protection |
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| Asbestos Hazard Emergency Response Act of 1986 | Public Law 99-519 | 15 U.S.C. §2642 & Seq. | §2646 Contractor and laboratory accreditation<br><br>(b) Accreditation by State<br>(1) Model plan<br>(B) Plan requirements<br>(xi) Housekeeping and personal hygiene practices, including the necessity of showers, and procedures to prevent asbestos exposure to an employee's family. |
| Residential Lead-Based Paint Hazard Reduction Act of 1992 | PL 102-550<br>Title X | 15 U.S.C. §2681 & Seq. | §2681 Definitions<br>For the purposes of this subchapter:<br>(1) Abatement<br>The term "abatement" 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-<br>(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<br>(B) all preparation, cleanup, disposal, and postabatement clearance testing activities associated with such measures.<br><br>(11) Lead-contaminated dust<br>The term "lead-contaminated dust" 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.<br><br>(12) Lead-contaminated soil<br>The term "lead-contaminated soil" 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.<br><br>§2683 Identification of dangerous levels of lead<br>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 [2 U.S.C.A. § 4851 et seq.], lead-based paint hazards, lead-contaminated dust, and lead-contaminated soil. |

**Table 16. Continued Federal Laws Relevant to Take-Home Exposure**

| Popular Name | Public Law | U.S. Code | Sections Relevant to Workers' Family Protection |
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| | | | §2685<br>(a) Program to promote lead exposure abatement<br>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.<br><br>(c) Exposure studies<br>(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.<br><br>(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:<br>(A) Drinking water<br>(B) Food<br>(C) Lead-based paint and dust from lead-based paint<br>(D) Exterior sources such as ambient air and lead in soil<br>(E) Occupational exposures, and other exposures that the Secretary determines to be appropriate. |
| Federal Insecticide Fungicide, Rodenticide Act | Public Law 92-516 | 7 U.S.C. 136 | §136q. Storage, disposal, transportation, and recall<br><br>(a) Storage, disposal, and transportation<br>(1) Data requirements and registration of pesticides<br>The Administrator may require under section 136(a) or 136(d) of this title that-<br>(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;<br>(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 rinsate containing the pesticide, or any other material used to contain or collect excess or spilled quantities of the pesticide; and<br><br>(e) Container design<br>(1) Procedures<br>(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. |

**Table 16. Continued Federal Laws Relevant to Take-Home Exposure**

| Popular Name | Public Law | U.S. Code | Sections Relevant to Workers' Family Protection |
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| | | | (B) The regulations shall ensure, to the fullest extent practicable, that the containers-<br>(i) accommodate procedures used for the removal of pesticides from the containers and the rinsing of the containers;<br><br>(ii) facilitate the safe use of the containers, including elimination of splash and leakage of pesticides from the containers;<br>(iii) facilitate the safe disposal of the containers; and<br>(iv) facilitate the safe refill and reuse of the containers. |
| | | | (2) Compliance<br><br>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.<br><br>(f) Pesticide residue removal<br>(1) Procedures<br>(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.<br><br>(B) The regulations may-<br>(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;<br>(ii) specify procedures that can be implemented promptly and easily in various circumstances and conditions;<br>(iii) provide for reuse, whenever practicable, or disposal of rinse water and residue; and<br>(iv) be coordinated with requirements for the rinsing of containers imposed under the Solid Waste Disposal Act (42 U.S.C. 6901 et seq.).<br>(C) The Administrator may, at the discretion of the Administrator, exempt products intended solely for household use from the requirements of this subsection.<br><br>(2) Compliance<br>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. |