American Journal of Industrial Medicine 9:543–550 (1986)

# Transmission of Occupational Disease to Family Contacts

**Barry Knishkowy, MD, MPH, and Edward L. Baker, MD, MPH**

As recognition of occupational illness increases, the scope of health problems related to work widens. An important area of concern is the worker's family, which has been shown to be at increased risk of disease attributable to the hazards previously thought to be relevant only to the worker. Such "para-occupational" disease occurs particularly in spouses and children through transport by the worker of hazardous materials from the worksite into the home. The most common vehicle has been contaminated work clothing brought home for cleaning. Outbreaks of severe illness caused by lead, beryllium, asbestos, and other compounds have been traced to home contamination by industrial dust. In this review, we describe reports of "para-occupational" illness that demonstrate the importance of early recognition by medical professionals of this cause of illness and of strict control of the dissemination of hazardous materials outside the workplace.

**Key words: occupational disease, lead, asbestos, polychlorinated biphenyls**

## INTRODUCTION

Illness related to occupational exposure is increasingly recognized as a major public health problem. Concern has extended to workers' families whose health may be adversely affected as a result of the workers' exposure to hazardous substances. Childhood malignancy rates have been reported to be increased in families where parents are employed in hydrocarbon-related and other industries [Fabia and Thuy, 1974; Peters et al, 1981]. Mutagenic or teratogenic mechanisms are postulated as being responsible for these and other occupation-related family health consequences, including infertility, spontaneous abortions, and low birth weight [Strobino et al, 1978; Whorton, 1983]. Breast milk has also been implicated in the transmission of occupational toxins to nursing infants, causing elevated blood levels and disease [Wolff, 1983; Bagnell and Ellenberger, 1977].

Another type of "para-occupational" disease occurs when toxic substances are carried home on contaminated work clothing and disease occurs in family members,

Occupational Health Program Harvard School of Public Health, Boston.
Current address for Dr. Barry Knishkowy is Department of Family Medicine, University Hospitals of Cleveland, Cleveland, OH 44106.
Current address for Dr. Edward L. Baker is Assistant Director, Centers for Disease Control, National Institute for Occupational Safety and Health, 1600 Clifton Road, N.E., Atlanta, GA 30333. Address reprint requests there.
Accepted for publication November 29, 1985.

particularly young children [Fischbein et al, 1980; Bellin, 1981; Chisolm, 1978]. In this paper we summarize reports of such outbreaks and suggest strategies for controlling this problem (Table I).

## DESCRIPTION OF OUTBREAKS

### Lead

Increased lead absorption has been documented in families of workers at lead mines [Martin et al, 1984], smelters [Baker et al, 1977; Rice et al, 1978; Lead poisoning—Tennessee, 1976; Winegar et al, 1977; Dolcourt et al, 1981], and storage battery factories [Dolcourt et al, 1981; Increased lead absorption in children of lead workers—Vermont, 1977; Dolcourt et al, 1978; Lead poisoning in children of battery plant employees—North Carolina, 1977; Elwood et al, 1977; Watson et al, 1978]. One of the first reports involved an outbreak of symptomatic lead poisoning among children of workers at a secondary lead smelter in 1975. Eight of ninety-one children studied required hospitalization and chelation therapy. Blood lead and erythrocyte protoporphyrin (EP) levels were significantly higher among these children than in a group of neighborhood control children. These lead and EP levels were well correlated with the lead content of household dust. Nonoccupational sources of lead in the home, such as lead-based paint or dust from local industrial sources, were shown to be unimportant contributors to this outbreak [Baker et al, 1977].

**TABLE I. Exposures and Manifestations of Para-Occupational Disease**

| Exposure | Manifestations | References |
|---|---|---|
| 1. Lead | Increased absorption | Baker et al, 1977 |
| | Hematologic toxicity | Dolcourt et al, 1981 |
| | | Elwoood et al, 1977 |
| | | Martin et al, 1984 |
| | | Rice et al, 1978 |
| | | Watson et al, 1978 |
| 2. Beryllium | Berylliosis | Chamberlin et al, 1957 |
| | | Chesner, 1950 |
| | | Eisenbud et al, 1949 |
| | | Hardy et al, 1967 |
| | | Hardy et al, 1948 |
| | | Lieben and Williams, 1969 |
| 3. Arsenic | Increased absorption | Klemmer et al, 1975 |
| | Hepatic angiosarcoma | Falk et al, 1981 |
| 4. Asbestos | Mesothelioma | Anderson et al, 1976 |
| | | Anderson et al, 1979 |
| | | Li et al, 1979 |
| | | Newhouse and Thompson, 1965 |
| | | Vianna, Polan, 1978 |
| | | Whitwell et al, 1977 |
| 5. Polycyclic Compounds (PCBs and related substances) | Increased absorption | Baker et al, 1980 |
| | Chloracne | Good and Pensky, 1943 |
| | | Jensen and Walker, 1972 |
| | | May, 1973 |
| 6. Chlordecone (Kepone) | Increased absorption | Taylor et al, 1978 |
| | Systemic toxicity | Cannon et al, 1978 |
| 7. Synthetic estrogens | Hyperestrogenism | Pacynski et al, 1971 |

Subsequent studies have produced similar findings. A report of 27 children of employees at a lead storage battery plant revealed significantly higher blood lead levels and concentrations of lead in household dust than in a group of neighborhood control children [Increased lead absorption in children of lead workers — Vermont, 1977]. Another study found that 40 of 58 children whose mothers worked in a battery factory had lead levels greater than or equal to 30 $\mu$g/dl of blood, and that six required chelation therapy at least once [Dolcourt et al, 1978]. Young children, ages one through six, had the highest blood lead levels and the greatest prevalence of lead poisoning. Ingestion of particulate lead in house dust through hand-to-mouth activities normally seen in preschool children was felt to be the primary mechanism of exposure.

These studies attributed the increased lead absorption in family members to lead brought home on workers' clothing. One of the other reports of transmission to family members involved workers in two secondary lead smelters who generally showered and changed their clothing at the end of a work shift and did not bring the contaminated clothing home. The authors suggested that lead sources, such as cars parked at the plant, dust on objects passed through a contaminated parking lot, and dusty street clothing that had been stored in dirty lockers, may have been responsible for the exposure at home [Rice et al, 1978].

### Beryllium

Numerous studies have shown that chronic berylliosis may develop in household contacts of beryllium workers [Lieben and Williams, 1969; Chamberlin et al, 1957; Chesner, 1950; Hardy, 1948; Hardy et al, 1967; Eisenbud et al, 1949]. Many of the individuals in these studies not only lived with beryllium industry employees, but also lived near the plant. An attempt was therefore made to identify the principal source of exposure. In one study, 27 of 60 neighborhood cases were exposed to beryllium only through contaminated clothing [Hardy et al, 1967].

To quantify home exposure resulting from the laundering of work clothing, Eisenbud and colleagues [1949] determined that shaking contaminated clothing produced an average beryllium content in air of 500 $\mu g/m^3$. (The present federal standard for beryllium permits a ceiling concentration at the workplace of 5 $\mu g/m^3$.) The development of strict occupational hygiene measures in 1949 has largely reduced home contamination and the incidence of beryllium-related diseases among both workers and their home contacts has fallen markedly since that time [Hardy et al, 1967; Hasan and Kazemi, 1974].

### Arsenic

In one report, agricultural use of toxic substances led to contamination of the home environment. The arsenic content of house dust among Hawaiian families that used arsenical herbicides was found to be higher than the level of contamination in the homes of families that did not. Additionally, several extremely high values (greater than 100 $\mu$g arsenic/gm dust) were recorded in both groups. In each instance the home owner was found to be employed either by a pest control firm or by a firm specializing in the treatment of construction lumber with wood preservative chemicals. The authors concluded that excess arsenic in these cases may have been carried home on work clothing [Klemmer et al, 1975].

Arsenic from the workplace may have contributed to the development of cancer in a child. A report in the literature describes four cases of hepatic angiosarcoma, a

rare tumor which is especially uncommon in children. One of the four was a girl who lived within one mile of the copper smelter and mine where her father worked in Ajo, Arizona. Exposed to arsenic in her water supply, in the local soil, and on her father's clothing, this girl ingested large amounts of the substance as a result of her habit of eating dirt both from the yard and from her father's shoes. The contaminated clothing may well have played a role in the development of her illness [Falk et al, 1981].

## Asbestos

Asbestos from the workplace has been implicated in numerous cases of mesothelioma in family members [Newhouse and Thompson, 1965; Anderson et al, 1976; Vianna and Polan, 1978; Li et al, 1978; Whitwell et al, 1977; Anderson et al, 1979]. One of the first studies of this association, published in 1965, reported that 9 of 83 patients at the London Hospital with a diagnosis of mesothelioma had lived with asbestos workers. Typically, the patient was a woman who washed her husband's contaminated clothing. In one case, the husband was described as coming home "white with asbestos every evening for three or four years." [Newhouse and Thompson, 1965].

In 1976, 33 case reports of mesothelioma in family contacts of asbestos workers had been reported. An additional four cases were reported in an article that summarized all family cases to that date. The authors of this paper also carried out a study of 326 people who had lived in the homes of asbestos workers during their employment, and who themselves had had no other exposure to asbestos. One hundred and fourteen, or 35% of those studied, were found to have chest x-ray abnormalities, including pleural thickening, pleural calcifications and irregular opacities [Anderson et al, 1976].

In another report, 52 women with mesothelioma were found to have a significantly greater number of fathers or husbands who had worked in asbestos-related industries than did a group of controls [Vianna and Polan, 1978]. Three cases of asbestos-related disease have been reported in a single family: Mesotheliomas developed in both the wife and the daughter of an asbestos worker who had died with severe asbestosis and lung cancer [Li et al, 1978].

An attempt has been made to quantify the actual exposure to asbestos fibers in the homes of workers. Chrysotile asbestos concentrations in air in the homes of 13 asbestos mine and mill employees were found to range from 50 to over 2000 $\mu g/m^3$. (The current federal standard allows a ceiling concentration at the workplace of ten fibers longer than 5 $\mu m/cc$, or approximately 300,000 $\mu g/m^3$.) This contrasted with samples from three neighboring homes of nonminers, in which the airborne concentrations ranged from 32–65 $\mu g/m^3$ [Nicholson et al, 1980].

## Polycyclic Compounds

One of the first reports of occupation-related disease in family members was published in 1943 and involved Halowax, a mixture of pentachloronaphthalene, hexachloronaphthalene, and chlorinated biphenyl, which was used in insulating wire for electric cables. A total of 52 cable workers using Halowax developed acneiform lesions (chloracne), referred to in this outbreak as "Halowax Acne" or "Cable Rash." Workers' wives also developed an eruption resembling that of their husbands, most probably as a result of contact with contaminated work clothing [Good and Pensky, 1943].

Another occurrence of chloracne in family members involved the chemical tetrachlorodibenzodioxin (dioxin) at a German plant in the 1950s. Dioxin, a minor contaminant in the manufacture of trichlorophenol, becomes an important byproduct of the reaction above 200°C. Increased production of dioxin due to chronic overheating led to the development of chloracne in all the plant's workers, as well as in many of their wives, children, and domestic animals [May, 1973].

A third outbreak of chloracne occurred in 1968 when an explosion in a 2,4,5-trichlorophenol production facility resulted in skin lesions in 70 workers. Three additional cases involving two temporary workers and a son of one of these men occurred in 1971. The father's work clothing was apparently the source of the son's exposure [Jensen and Walker, 1972].

Polychlorinated biphenyls (PCBs) are compounds that have been used as insulating fluids and dielectrics. Elevated serum levels of PCBs have been found in family members of workers in an electrical capacitor manufacturing plant [Baker et al, 1980].

### Chlordecone (Kepone)

Chlordecone is a chlorinated hydrocarbon insecticide that was produced between the mid-1950s and the mid-1970s. A study carried out in 1975 showed that 76 (57%) of 133 people who had worked at a plant that manufactured Kepone had developed a new clinical illness characterized by nervousness, tremor, weight loss, opsoclonus, pleuritic and joint pain, and oligospermia. A study of community residents and of family members revealed that many had detectable Kepone concentrations in their blood. However, clinical evidence of Kepone poisoning (objective tremor) was found in only two cases: Both were workers' wives who had washed their husbands' work clothing [Cannon et al, 1978]. In another study, the percentage of workers' family members with detectable chlordecone in their blood (94%) was greater than that for workers in contiguous businesses and industries (72%) or for residents throughout the locality (19%) [Taylor et al, 1978].

### Synthetic Estrogens

One of the few available studies of occupational exposure to synthetic estrogens describes hyperestrogenism resulting from exposure to DES and other estrogens at a pharmaceutical plant. Seven of the seventeen affected individuals were children whose parents worked at the plant [Pacynski et al, 1971]. Chemicals brought home from the workplace may have been responsible for the effects seen in the children.

## PREVENTION

The most direct means of preventing these exposures is by ensuring that contaminated clothing remains in the workplace. The Occupational Safety and Health Administration (OSHA) has promulgated regulations covering lead, arsenic, and asbestos exposure requiring, among other things, that employers provide work clothing, changing facilities, and showers for employees. Unfortunately, other hazardous substances are not covered by similar regulations and the standards that exist are not universally enforced.

Physicians and other health practitioners have an important role to play in detecting and preventing environmental disease. The occupational and environmental

history, increasingly recognized as a valuable clinical tool, should be used in identifying family contact disease. Three general questions should be included in any routine history [Goldman and Peters, .1981]: (1) What are your current and past longest-held jobs? (social history); (2) Do you have, or have you ever had exposure to chemicals, dust, fumes, loud noise, or radiation? (review of systems); and (3) Is there any relationship between the presenting illness and any job or exposure, the time of work or of home activities, or any other contributing factors? (chief complaint).

Consultation regarding the significance of specific workplace exposures can be obtained from specialists in occupational and environmental medicine and/or federal or state agencies. Through appropriate intervention the primary care provider can educate the patient and the employer regarding the hazards of family contact disease and ultimately lead to a reduction in this eminently preventable condition.

## SUMMARY

Health may be affected by toxic substances in the home, in the community, and in the workplace. Industrial substances transported home by the worker are increasingly being implicated as agents causing illness in family contacts. Lead, beryllium, and asbestos are substances for which this process has been demonstrated convincingly. Other toxins, including arsenic, synthetic estrogens, and various chlorinated hydrocarbons, have also been implicated. Work clothing is the most important vehicle by which occupational substances are brought home, but other personal objects may provide sources of home contamination.

The magnitude of the problem may be reduced by workplace regulation and by increased health provider awareness. Current OSHA standards indirectly protect family members from exposure to asbestos, lead, and arsenic, while recommendations to leave contaminated clothing at work are nonexistent for numerous other toxic substances.

Control of family contact disease depends significantly upon public awareness and involvement. The responsibility falls in large part upon the health care practitioner, whose environmental and occupational history-taking for all members of the family will facilitate protection of the health of workers and their household members.

## REFERENCES

Anderson HA, Lilis R, Daum SM, Fischbein AS, Selikoff IJ (1976): Household-contact asbestos neoplastic risk. Ann NY Acad Sci 271:311–323.

Anderson HA, Lilis R, Daum SM, Selikoff IJ (1979): Asbestosis among household contacts of asbestos factory workers. Ann NY Acad Sci 330:387–399.

Bagnell PC, Ellenberger HA (1977): Obstructive jaundice due to a chlorinated hydrocarbon in breast milk. Can Med Assoc J 117:1047–1048.

Baker EL Jr, Folland DS, Taylor TA, Frank M, Peterson W, Lovejoy G, Cox D, Houseworth J, Landrigan PJ (1977): Lead poisoning in children of lead workers. N Engl J Med 296:260–261.

Baker EL Jr, Landrigan PJ, Glueck CJ, Zack MM, Liddle JA, Burse VW, Houseworth WJ, Needham LL (1980): Metabolic consequences of exposure to polychlorinated biphenyls (PCBs) in sewage sludge. Am J Epidemiol 112:553–563.

Bellin JS (1981): Don't take your 'work' home with you. Occup Health Saf June:39–42.

Cannon SB, Veazey JM Jr, Jackson RS, Burse VW, Hayes C, Straub WE, Landrigan PJ, Liddle JA (1978): Epidemic kepone poisoning in chemical workers. Am J Epidemiol 107:529–537.

Chamberlin GW, Jennings WP, Lieben J (1957): Chronic pulmonary disease associated with beryllium dust. Pa Med J April:497–503.

Chesner C (1950): Chronic pulmonary granulomatosis in residents of a community near a beryllium plant: Three autopsied cases. Ann Intern Med 32:1028–1048.
Chisolm JJ Jr (1978): Fouling one's own nest. Pediatrics 62:614–617.
Dolcourt JL, Hamrick HJ, O'Tuama LA, Wooten J, Baker EL Jr (1978): Increased lead burden in children of battery workers: Asymptomatic exposure resulting from contaminated work clothing. Pediatrics 62:563–566.
Dolcourt JL, Finch C, Coleman GD, Klimas AJ, Milar CR (1981): Hazard of lead exposure in the home from recycled automobile storage batteries. Pediatrics 68:225–229.
Eisenbud M, Wanta RC, Dustan C, Steadman LT, Harris WB, Wolf BS (1949): Nonoccupational berylliosis. J Indust Hyg Toxicol 31:282–294.
Elwood WJ, Clayton BE, Cox RA, Delves HT, Ring E, Malcolm D, Ratcliffe JM, Taylor JF (1977): Lead in human blood and in the environment near a battery factory. Br J Prev Soc Med 31:154–163.
Fabia J, Thuy DM (1974): Occupation of father at time of birth of children dying of malignant diseases. Br J Prev Soc Med 28:98–100.
Falk H, Herbert JT, Edmonds L, Heath CW Jr, Thomas LB, Popper H (1981): Review of four cases of childhood hepatic angiosarcoma—elevated environmental arsenic exposure in one case. Cancer 47:382–391.
Fischbein A, Cohn J, Ackerman G (1980): Asbestos, lead and the family: Household risks. J Fam Pract 10:989–992.
Goldman RH, Peters JM (1981): The occupational and environmental health history. JAMA 246:2831–2836.
Good CK, Pensky N (1943): Halowax acne. Arch Dermatol 48:251–257.
Hardy HL (1948): Delayed chemical pneumonitis in workers exposed to beryllium compounds. Am Rev Tuber 57:547–556.
Hardy HL, Rabe EW, Lorch S (1967): United States beryllium case registry (1952–1966). J Occup Med 9:271–276.
Hasan FM, Kazemi H (1974): Chronic beryllium disease: A continuing epidemiologic hazard. Chest 65:289–293.
Increased lead absorption in children of lead workers—Vermont (1977). Morbid Mortal Weekly Rep 26:61–62.
Jensen NE, Walker AE (1972): Chloracne: Three cases. Proc R Soc Med 65:687–688.
Klemmer HW, Leitis E, Pfenninger K (1975): Arsenic content of house dusts in Hawaii. Bull Environ Contam Toxicol 14:449–452.
Lead poisoning—Tennessee (1976). Morbid Mortal Weekly Rep 25:85.
Lead poisoning in children of battery plant employees—North Carolina (1977). Morbid Mortal Weekly Rep 26:321.
Li FP, Lokich J, Lapey J, Neptune WB, Wilkins EW Jr (1978): Familial mesothelioma after intense asbestos exposure at home. JAMA 240:467.
Lieben J, Williams RR (1969): Respiratory disease associated with beryllium refining and alloy fabrication. J Occup Med 11:480–485.
Martin AE, Fairweather FA, Buxton RSJ, Roots LM (1984): Recent epidemiological studies of environmental lead of industrial origin. Proceedings of International Symposium on Environment and Health, C.E.C. June, pp 1–6.
May G (1973): Chloracne from the accidental production of tetrachlorodibenzodioxin. Br J Ind Med 30:276–283.
Newhouse ML, Thompson H (1965): Mesothelioma of pleura and peritoneum following exposure to asbestos in the London area. Br J Ind Med 22:261–269.
Nicholson WJ, Rohl AN, Weisman I, Selikoff IJ (1980): Environmental asbestos concentrations in the United States. IARC Sci Publ 2:823–827.
Pacynski A, Budzynska A, Przylecki S, Rosacynski J (1971): Hiperestrogenizm v pracownikow zakladow farmaceutyczaych i ich dziecijako choroba zawodowa. Endrokrynol Pol (Warsaw) 22:149–154.
Peters JM, Preston-Martin S, Yu MC (1981): Brain tumors in children and occupational exposure of parents. Science 213:235–237.
Rice C, Fischbein A, Lilis R, Sarkozi L, Kon S, Selikoff IJ (1978): Lead contamination in the homes of employees of secondary lead smelters. Environ Res 15:375–380.
Strobino BR, Kline J, Stein Z (1978): Chemical and physical exposures of parents: effects on human reproduction and offspring. Early Hum Dev 1/4:371–399.

Taylor JR, Selhorst JB, Houff SA, Martinez AJ (1978): Chlordecone intoxication in man. Neurology 28:626–630.

Vianna NJ, Polan AK (1978): Non-occupational exposure to asbestos and malignant mesothelioma in females. Lancet 1:1061–1063.

Watson WN, Witherell LE, Giguere GC (1978): Increased lead absorption in children of workers in a lead storage battery plant. J Occup Med 20:759–761.

Whitwell F, Scott J, Grimshaw M (1977): Relationship between occupations and asbestos-fibre content of the lungs in patients with pleural mesothelioma, lung cancer and other diseases. Thorax 32:377–386.

Whorton MD (1983): Adverse reproductive outcomes. The occupational health issue of the 1980s. Am J Public Health 73:15–16.

Winegar DA, Levy BS, Andress JS Jr, Landrigan PJ, Scurton WH, Krause MJ (1977): Chronic occupational exposure to lead: An evaluation of the health of smelter workers. J Occup Med 19:603–606.

Wolff MS (1983): Occupationally derived chemicals in breast milk. Am J Ind Med 4:259–281.