

Persons using assistive technology might not be able to fully access information in this file. For assistance, please send e-mail to: mmwrq@cdc.gov. Type 508 Accommodation and the title of the report in the subject line of e-mail.

Leading Work-Related Diseases and Injuries -- United States

The National Institute for Occupational Safety and Health (NIOSH) has developed a suggested list of 10 leading work-related diseases and injuries. Summaries of the first five disease categories have appeared previously (1-5); a discussion of the sixth category, disorders of reproduction, appears below. **DISORDERS OF REPRODUCTION**

Since antiquity, certain chemical and physical agents have been recognized as having detrimental effects on human reproduction. For example, the effect of industrial lead poisoning in inducing abortions was noted by the Romans and again in the first decade of this century (6). Evidence from more recent laboratory studies and clinical investigations indicates that a wide range of microbiologic, physical, and chemical agents, such as Brucella, rubella, ionizing and nonionizing radiation, heat and vibration, tobacco, alcohol, and certain drugs, can adversely affect reproductive outcomes. At least 50 chemicals--including heavy metals, such as lead and cadmium, glycol ethers, organohalide pesticides, organic solvents, and chemical intermediates, such as styrene and vinyl chloride--in widespread use in industry have been shown to produce impairment of reproductive functions in animals (7).

Until recently, the potential hazards to human reproduction posed by occupational exposures received little attention. However, adverse effects after thalidomide exposure in the 1960s and the occurrence in 1970 of methylmercury poisoning among residents of Minamata, Japan, dramatically demonstrated the teratogenic potential of chemical exposures. Those events and the increasing entry of women into the workforce focused greater attention on the potential hazards to female reproductive function of occupational exposures. In the late 1970s, the demonstration of sterility among male workers exposed to dibromochloropropane was described; this drew attention to the concomitant potential for hazards to male reproductive function (8).

Occupational exposures can produce a wide range of adverse effects on reproduction. The effects of parental exposure before conception to agents toxic to reproductive functions may be evident as reduced fertility, unsuccessful fertilization or implantation, or an abnormal fetus. Maternal exposure after conception may result in death of the fetus or structural and functional abnormalities in the newborn. Other possible adverse outcomes include spontaneous abortions

(both early and late), major and minor birth defects, perinatal death, low birth weight, altered sex ratio, developmental or behavioral disabilities, and transplacental exposure to carcinogen (9-11).

Estimates of the prevalence of adverse reproductive outcomes indicate that these events occur with considerable frequency in the U.S. population. For example, an estimated 560,000 infant deaths, spontaneous abortions, and stillbirths occur each year. The March of Dimes estimates that 200,000 live infants with some type of birth defect--benign or disabling--are born in the United States each year (9).

The causes of most of these adverse outcomes are unknown. For example, 6%-30% of the infertile couples have no recognized anatomic or physiologic abnormalities to account for the infertility (10); neither the etiology of sperm abnormalities nor the cause of sister-chromatid exchange in spontaneous abortions has been established (11,12). The causes for as many as 65%-70% of the birth defects are not known (13).

Maternal Exposures. Studies of occupational reproductive hazards to date have consisted mainly of epidemiologic surveys of pregnancy outcomes following maternal exposures. Such studies have shown increased rates of spontaneous abortions among laboratory and chemical workers (14,15) and among workers exposed to lead (16), ethylene oxide (17), and anesthetic gases (18,19). Studies of adverse outcomes of pregnancy, however, are subject to several methodologic limitations. For example, the detection of rare outcomes, such as birth defects, requires the study of several thousand pregnancies, and retrospective studies are subject to problems of recall and misclassification, both of reproductive events and of exposures (20,21). The timing, duration, and frequency of exposure before and during pregnancy may critically affect reproductive outcomes (22). For example, exposure to ionizing radiation during the first trimester may result in microcephaly and mental retardation, and exposure during the third trimester may produce low birth weight and neonatal death (11). Other studies have been limited by the selection of inadequate comparison groups or the failure to examine the influence of other factors, such as alcohol and tobacco consumption or maternal age, that affect reproductive outcomes.

Paternal Exposures. Since azoospermia (absence of living spermatozoa in the semen) and oligospermia (subnormal concentration of spermatozoa) were reported in 1977 among workers exposed to dibromochloropropane (8), at least 14 studies have examined the quality of semen in workers exposed to lead, carbon disulfide, anesthetic gases, ionizing radiation, toluenediamine, dinitrotoluene, carbaryl, and several other pesticides (10). Adverse effects on the quality of semen were reported in workers exposed to lead or ionizing radiation. In other studies (e.g., of exposures to ethylene dibromide) results were inconclusive because of problems in design of the study or inadequate numbers of participants (10). CDC recently used data collected by the Metropolitan Atlanta Congenital Defects Program to examine the risk of serious structural birth defects among the children of male Vietnam veterans; no statistically excessive risks were noted (23). In general, relatively few studies have been conducted of reproductive outcomes associated with paternal exposures (9).

Extent of potential exposures. Estimates have been made of the number of workers potentially exposed to selected agents known or suspected to be toxic to reproductive function (Table 1). NIOSH estimates that approximately 200,000 workers are potentially exposed to various glycol ethers (24), several of which exhibit marked testicular toxicity in animals (25). An estimated 9 million workers are exposed to radiofrequency/microwave radiation (26), which has been shown to cause embryonic death and impaired fertility in animals but which has yet to be studied

adequately in humans. NIOSH has estimated that approximately 50,000 personnel in hospital operating rooms are potentially exposed to waste anesthetic gases, and 139,000 hospital and other industrial workers may be exposed to ethylene oxide (24); both agents have been linked to an increased risk of spontaneous abortions in humans. Reported by Industrywide Studies Br, Surveillance Br, Div of Surveillance, Hazard Evaluations, and Field Studies, National Institute for Occupational Safety and Health, CDC.

Editorial Note

Editorial Note: The extent to which occupational exposures in American workers produce adverse reproductive outcomes is largely unknown. However, the information presented here suggests that the problem is both widespread and serious. Epidemiologic and toxicologic research into the reproductive effects of occupational exposures is in its infancy. There is a continuing effort to elucidate the etiology of adverse reproductive outcomes, such as fetal chromosomal abnormalities or abnormal spermatogenesis and to develop improved animal models for screening agents for possible mutagenic and toxic effects related to human reproduction. Registries for the surveillance of outcomes of reproduction, such as CDC's Birth Defects Monitoring Program (9), and improved methodologies developed to evaluate such parameters as quality of semen (12) and outcomes of pregnancy (20), will permit further identification of specific occupational hazards to reproduction. When such hazards are identified and controlled in the workplace, the prevention of reproductive disorders in the population as a whole will be substantially improved.

References

1. CDC. Leading work-related diseases and injuries--United States. *MMWR* 1983;32:24-6, 32.
2. CDC. Leading work-related diseases and injuries--United States. *MMWR* 1983;32:189-91.
3. CDC. Leading work-related diseases and injuries--United States. *MMWR* 1984;33:125-28.
4. CDC. Leading work-related diseases and injuries--United States. *MMWR* 1984;33:213-5.
5. CDC. Leading work-related diseases and injuries--United States. *MMWR* 1985;34:219-22, 227.
6. Hamilton A. Women in the lead industries. Bureau of Labor Statistics Bulletin No. 253. Washington, D.C.: U.S. Department of Labor, 1919. In: Hunt V. Work and the health of women. Boca Raton, Florida: CRC Press, Inc. 1979.
7. Barlow SM, Sullivan FM. Reproductive hazards of industrial chemicals: an evaluation of animal and human data. London: Academic Press, 1982.
8. Whorton D, Krauss RM, Marshall S, Milby TH. Infertility in male pesticide workers. *Lancet* 1977;ii:1259-61.
9. Bloom AD, Paul NW, eds. Guidelines for studies of human populations exposed to mutagenic and reproductive hazards: proceedings of conference held January 26-27, 1981, in Washington, D.C. sponsored by Centers for Disease Control. White Plains, New York: March of Dimes Birth Defects Foundation, 1981.
10. Ratcliffe JM. Altered fertility. In: Review of occupational hazards to reproductive health. Geneva, Switzerland: World Health Organization, (in press).
11. Strobino mBR, Kline J, Stein Z. Chemical and physical exposures of parents: effects on human reproduction and offspring. *Early Hum Dev* 1978;1:371-99.

12. Wyrobek AJ. Methods for evaluating the effects of environmental chemicals on human sperm production. *Environ Health Perspect* 1983; 48:53-9.
13. Wilson JG. *Environment and birth defects*. New York: Academic Press, 1973.
14. Stranberg M, Sandback K, Axelson O, Sundell L. Spontaneous abortions among women in hospital laboratory. {Letter} *Lancet* 1978;i:384-5.
15. Hemminki K, Franssilla E, Vainio H. Spontaneous abortions among female chemical workers in Finland. *Int Arch Occup Environ Health* 1980;45:123-6.
16. Nordstrom S, Beckman L, Nordenson I. Occupational and environmental risks in and around a smelter in northern Sweden. V. Spontaneous abortion among female employees and decreased birth weight in their offspring. *Hereditas* 1979;90:291-6.
17. Hemminki K, Mutanen P, Saloniemi I, Niemi ML, Vainio H. Spontaneous abortions in hospital staff engaged in sterilising instruments with chemical agents. *Br Med J* 1982;285:1461-3.
18. Cohen EN, Gift HC, Brown BW, et al. Occupational disease in dentistry and chronic exposure to trace anesthetic gases. *J Am Dent Assoc* 1980; 101:21-31.
19. Cohen EN, Brown BW, Bruce DL, et al. Occupational disease among operating room personnel: a national study. *Anesthesiology* 1974;41: 321-40.
20. Selevan SG. Design considerations in pregnancy outcome studies of occupational populations. *Scand J Work Environ Health* 1981;7:76-82.
21. Buffler PA. Some problems involved in recognizing teratogens used in industry. *Contributions to epidemiology and biostatistics* 1979;1: 118-37.
22. Gordon JE. Assessment of occupational and environmental exposures. In: Bracken, MB, ed. *Perinatal epidemiology*. New York:Oxford University Press (in press).
23. Erikson JD, Mulinare J, McClain PW, et al. Vietnam veterans' risks for fathering babies with birth defects. *JAMA* 1984;252:903-12.
24. National Institute for Occupational Safety and Health. National Occupational Hazard Survey. Projected estimates of potentially exposed workers. Cincinnati, Ohio: National Institute for Occupational Safety and Health, December 1977 (DHEW{NIOSH} publication no. 78-114).
25. National Institute for Occupational Safety and Health. Glycol ethers. *Current Intelligence Bulletin* No. 39. Cincinnati, Ohio: National Institute for Occupational Safety and Health, 1983 (DHHS {NIOSH} publication no. 83-112).
26. Centaur Associates, Inc. final report; study of radiofrequency and microwave radiation (phase 1). Prepared for the Occupational Safety and Health Administration;1982.

Disclaimer All MMWR HTML documents published before January 1993 are electronic conversions from ASCII text into HTML. This conversion may have resulted in character translation or format errors in the HTML version. Users should not rely on this HTML document, but are referred to the original *MMWR* paper copy for the official text, figures, and tables. An original paper copy of this issue can be obtained from the Superintendent of Documents, U.S. Government Printing Office (GPO), Washington, DC 20402-9371; telephone: (202) 512-1800. Contact GPO for current prices.