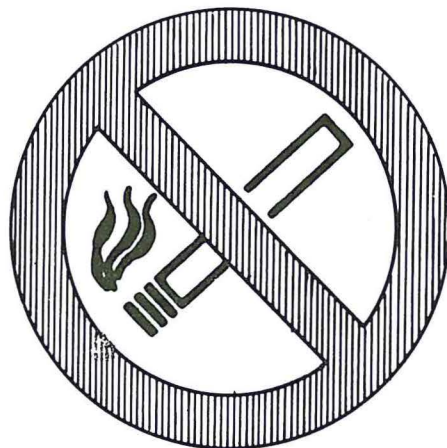


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ADVERSE HEALTH EFFECTS of SMOKING and the OCCUPATIONAL ENVIRONMENT



U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health

The NIOSH Current Intelligence Bulletin is the primary product of the Current Intelligence System. The purpose of the Current Intelligence System is to promptly review, evaluate, and supplement new information received by NIOSH on occupational hazards that are either unrecognized or are greater than generally known. The staff of the NIOSH Technical Evaluation and Review Branch, Office of Extramural Coordination and Special Projects was responsible for the preparation of this Bulletin.

As warranted by this evaluation, the information is capsulized and disseminated to NIOSH staff, other government agencies, and the occupational health community, including labor, industry, academia, and public interest groups. With respect to currently known hazard information this system also serves to advise appropriate members of the above groups of recently acquired specific knowledge which may have an impact on their programs or perception of the hazard. Above all, the Current Intelligence System is designed to protect the health of American workers and to allow them to work in the safest possible environment.

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CURRENT INTELLIGENCE BULLETIN

ADVERSE HEALTH EFFECTS OF SMOKING AND THE OCCUPATIONAL ENVIRONMENT

February 5, 1979

There is increasing evidence of adverse health effects due to the combined actions of tobacco use and exposure to chemical and physical agents in the workplace. The National Institute for Occupational Safety and Health (NIOSH) recommends that the use of and/or carrying of tobacco products into the workplace be curtailed in situations where employees may be exposed to physical or chemical substances which can interact with tobacco products. Additionally, curtailment of the use of tobacco products in the workplace should be accompanied by simultaneous control of worker exposure to physical and chemical agents. These recommendations are based on evidence which indicates that smoking can act in combination with hazardous agents to produce or increase the severity of a wide range of adverse health effects. Six ways have been identified by which smoking can interact with workplace exposures, and this Bulletin has been prepared to advise you of the hazards involved. NIOSH requests that chemical producers and distributors transmit the information in this Bulletin to their customers and employees, and that professional associations and unions inform their members.

In this Bulletin, smoking and/or tobacco products are defined as cigarettes, cigars, pipe tobacco, chewing tobacco, and any by-products resulting from their burning and/or use. The discussions and illustrations used in this Bulletin may relate to any one or more of these products. It is important to note that many of the adverse effects can still occur if an exposed tobacco product (contaminated) is subsequently smoked away from the workplace.

BACKGROUND

The National Institute for Occupational Safety and Health recently prepared a chapter entitled "Interaction Between Smoking and Occupational Exposures," for SMOKING AND HEALTH - A Report of the Surgeon General - January 11, 1979 (1). This Bulletin contains a summary of the information in the above chapter of the Surgeon General's Report.

The smoking habits among workers in various occupations provide an opportunity for interaction to occur between smoking and workplace exposure to physical and chemical agents. More blue-collar workers smoke [51%] than white-collar workers

[37%]. Also, the blue-collar workers have the highest risk for workplace exposure to hazardous physical and chemical agents. The use of tobacco products and workplace exposure to industrial agents increased steadily from 1920 to 1960. Since 1966 the percentage of blue-collar workers who smoke has decreased while the number of workplace exposures continues to increase. Studies have shown that more non-whites[22.6%] work in jobs associated with an increased risk of lung cancer than do whites[13.5%](2-5).

Despite increasing recognition that both smoking and workplace exposures contribute to the development of certain disease states, few investigators have addressed the ways in which these two factors can interact to produce or enhance disease in workers. Some of the effects historically attributed to smoking may actually reflect interactions between smoking and workplace exposure to physical and chemical agents. These cannot always be quantified, and it should be noted that the six different mechanisms by which smoking may adversely act with physical and chemical agents found in the workplace are not mutually exclusive and several may prevail for any given agent. The six modes of interaction follow.

ILLUSTRATIONS OF MODES OF ACTION

1. Certain toxic agents in tobacco products and/or smoke may also occur in the workplace, thus increasing exposure to the agent.

Employees exposed in the workplace to toxic chemicals can receive additional exposures from the presence of those toxic chemicals in tobacco products. For example, cigarette smoking causes increased exposure to carbon monoxide (CO). A CO concentration of 4% (40,000 ppm) in cigarette smoke can lead to a lung CO concentration of 0.04 to 0.05% (400 to 500 ppm), which can produce CO blood concentrations, as measured by the carboxyhemoglobin (COHb) level, of 3 to 10% (6-8).

Workers are frequently exposed to carbon monoxide as part of their job and workers who smoke in those situations have increased exposure to CO. For example, in a study of COHb levels in British steelworkers, the average end-of-shift COHb concentration found in non-smoking blast furnace workers was 4.9% compared to 1.5% in non-smoking unexposed controls. For heavy cigarette smokers, the average COHb levels were 7.4% for exposed blast furnace workers and 4.0% for unexposed controls (9). The COHb levels of blast furnace workers who smoked were in a critical range. Studies have shown that levels of COHb in excess of 5% can cause cardiovascular changes which are dangerous for persons with coronary heart disease (10-11). Also, since a significant number of workers have coronary heart disease and many smoke, additional occupational exposure to carbon monoxide may increase cardiovascular morbidity and mortality.

Other chemicals found in tobacco which workers might be exposed to at their jobs, include: acetone, acrolein, aldehydes (e.g. formaldehyde), arsenic, cadmium, hydrogen cyanide, hydrogen sulfide, ketones, lead, methyl nitrite, nicotine, nitrogen dioxide, phenol, and polycyclic aromatic compounds (12).

2. Workplace chemicals may be transformed into more harmful agents by smoking.

The heat generated by burning tobacco can transform workplace chemicals into more harmful substances. Investigations of outbreaks of polymer fume fever (PFF) provide a clear illustration of this effect.

Polymer fume fever is a disease caused by inhalation of degradation product fumes from heated Teflon[®] (polytetrafluoroethylene). The particular chemical agents responsible for PFF have not been identified; however, temperatures in excess of 315[°]C (600[°]F) have been sufficient to cause their production. It is important to note that the temperature of burning tobacco in a cigarette is approximately 875[°]C (1600[°]F) (13,14). This disease is characterized by effects such as chest discomfort, fever, increased number of white blood cells, headache, chills, muscular aches and weakness (15). Because these symptoms are similar to those of other diseases, such as influenza, polymer fume fever may go undiagnosed. It has been suggested that repeated attacks of polymer fume fever may lead to permanent lung damage (16).

One report describes aviation employees whose work involved contact with door seals that had been sprayed with an unspecified fluorocarbon polymer. In one case, a worker smoking during a break realized by the taste of his cigarette that it had become contaminated. Although the worker extinguished the cigarette, he experienced shivering and chills lasting approximately six hours, beginning one-half hour after smoking (17).

Another illustrative report describes outbreaks of polymer fume fever among smokers whose hands were contaminated with polytetrafluoroethylene used as a mold release agent. There was no recurrence of symptoms among these workers after smoking at the plant was prohibited (18).

Other examples of workplace chemicals which can possibly be transformed into more toxic substances by smoking after tobacco is contaminated include a number of chlorinated hydrocarbons that have the potential for conversion to phosgene, a highly toxic chemical.

3. Tobacco products may serve as vectors by becoming contaminated with toxic agents found in the workplace, thus facilitating entry of the agent into the body by inhalation, ingestion, and/or skin absorption.

Tobacco products can become contaminated by chemicals used in the workplace thus increasing the amount of toxic chemicals entering the workers' bodies.

The effects of smoking cigarettes contaminated in the workplace with known amounts of tetrafluoroethylene polymer have been studied with the assistance of human volunteers. Nine out of ten subjects were reported to exhibit typical

polymer fume fever symptoms after each had smoked just one cigarette contaminated with 0.40 mg tetrafluoroethylene polymer.

Some other toxic chemicals found in the workplace, identified in NIOSH criteria documents as potential contaminants of tobacco products include boron trifluoride (20), carbaryl (21), dinitro-ortho-creosol (22), inorganic fluorides (23), formaldehyde (24), lead (25,26), inorganic mercury (27), methyl parathion (28), and organotin (29).

4. Smoking may contribute to an effect comparable to that which can result from exposure to toxic agents found in the workplace, thus causing an additive biological effect.

Smoking can add to the damaging biological effects which result from exposure to toxic chemicals found in the workplace. For example, combined worker exposure to chlorine and cigarette smoke can cause a more damaging biological effect than exposure to chlorine alone. In a plant producing chlorine by electrolysis of brine, 55 of 139 workers required oxygen therapy at least once during their employment after accidental exposure one or more times to high concentrations of chlorine. The maximal mid-expiratory flow (MMF) values of these workers with accidental chlorine exposures were compared with those of non-exposed smokers and non-smokers. A reduction in normal lung function is indicated by low MMF values, while a normal lung function is reflected by higher MMF values. MMF values decreased when chlorine and smoking were considered as additive toxic agents. Average MMF values in liters per second [L/sec] decreased in the following sequence: unexposed non-smokers [4.36], unexposed smokers [4.13], exposed non-smokers [4.10], and exposed smokers [3.57] (30). Other agents which can act additively with tobacco smoke include cotton dust (31), coal dust (32,33), and beta radiation (34).

5. Smoking may act synergistically with toxic agents found in the workplace to cause a much more profound effect than that anticipated simply from the separate influences of the occupational exposure and smoking.

Smoking can interact with worker exposure to toxic materials found in the workplace resulting in more severe health damage than that anticipated from adding the separate influences of the occupational exposure and smoking. Asbestos provides one of the most dramatic examples of severe health damage resulting from interaction between the smoking of tobacco products and workplace exposures. In a prospective study of 370 asbestos insulation workers, 24 of 283 cigarette smokers died of bronchogenic carcinoma during the four year period of the study, while not one of the 87 non-cigarette smokers died of this cancer (35). This study suggested that asbestos workers who smoke have eight times the risk of lung cancer as compared to all other smokers and 92 times the risk of non-smokers not exposed to asbestos. This same group of insulation workers was restudied five years later, at which time 41 of the 283 smokers had died of bronchogenic cancer. Only 1 of the 87 non-cigarette smokers, a cigar smoker, died of lung cancer (36).

Other chemicals and physical agents which appear to act synergistically with tobacco smoke include radon daughters (37), gold mine exposures (38), and exposures in the rubber industry (39).

6. Smoking may contribute to accidents in the workplace

Studies have shown that smoking contributes to accidents in the workplace. In a nine-month study of job accidents, the total accident rate was more than twice as high among smokers as among non-smokers (40). It has been suggested that injuries attributable to smoking were caused by loss of attention, preoccupation of the hand for smoking, irritation of the eyes, and cough (41). Smoking can also contribute to fire and explosions in occupational settings where flammable and explosive chemical agents are used; however, in many of these areas smoking is prohibited.

Some other situations where interaction between smoking and workplace exposure have been hypothesized include:

- A. Cadmium - Several studies suggests that exposed smokers had poorer lung function and a higher incidence of urinary abnormalities than did exposed non-smokers (42,43).
- B. Chloromethyl Ether - Chronic cough and expectoration showed a dose response relationship with chemical exposure and smoking. For each smoking category, chronic cough was more common for exposed than for unexposed men (44).
- C. beta-Naphthylamine and other Aromatic Amines - There are reports of associations between cigarette smoking and bladder cancer (45,46). Since aromatic amines, which are known bladder carcinogens, are also found in cigarette smoke (12), a smoker who works with this group of gases receives exposure to bladder carcinogens from two sources. The interaction between smoking and exposure to aromatic amines should be further assessed.

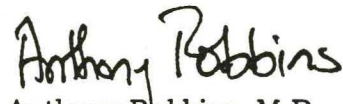
Research Considerations

- 1. Studies on the adverse health effects from smoking should take occupational exposures into consideration and vice versa. Whenever possible, studies should include data on exposed and unexposed smokers and non-smokers.
- 2. The increasing rates of lung cancer in non-white males compared to white males should be investigated further with respect to occupational exposures and smoking habits.
- 3. The change in smoking habits of blue collar workers over the last decade provides an opportunity to more critically assess the contribution of smoking vs. occupational exposure to hazardous agents to certain disease states. Cohorts should be identified and followed prospectively for this purpose.

4. Workplace physical and chemical agents which interact with the smoking of tobacco to produce adverse health effects should be identified.
5. Further investigation into the mechanisms of synergism between smoking and occupational exposures is needed.
6. The impact of the combination of smoking and workplace exposures upon reproductive functions needs further study.
7. The impact of smoking on workplace accidents merits further study.
8. The lack of information on the effect of side stream smoke in the development of occupational disease in non-smoking workers merits attention.
9. The effects of cessation of smoking upon lung cancer risk among those occupationally exposed to toxic workplace agents requires investigation.

RECOMMENDATIONS

The National Institute for Occupational Safety and Health (NIOSH) recommends that the use of and/or carrying of tobacco products into the workplace be curtailed in situations where employees may be exposed to physical or chemical substances which may interact with tobacco products. Additionally, curtailment of the use of tobacco products in the workplace should be accompanied by simultaneous control of worker exposure to physical and chemical agents. These recommendations are based on evidence which indicates that smoking can act in combination with hazardous agents to produce or increase the severity of a wide range of adverse health effects.


Anthony Robbins, M.D.
Director

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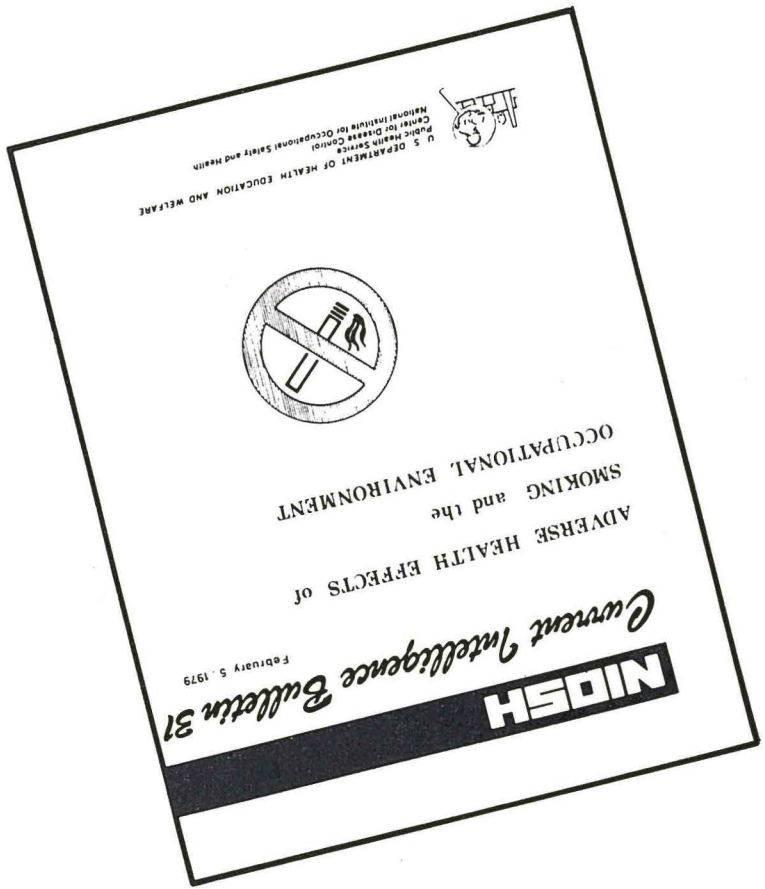
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- * 1. Chloroprene - January 20, 1975
- * 2. Trichloroethylene (TCE) - June 6, 1975
- * 3. Ethylene Dibromide (EDB) - July 7, 1975
- * 4. Chrome Pigment - June 24, 1975
- October 7, 1975
- October 8, 1976
- * 5. Asbestos - Asbestos Exposure During Servicing of Motor Vehicle Brake and Clutch Assemblies - August 8, 1975
- * 6. Hexamethylphosphoric Triamide (HMPA) - October 24, 1975
- * 7. Polychlorinated Biphenyls (PCBs) - November 3, 1975
- August 20, 1976
- 8. 4,4'-Diaminodiphenylmethane (DDM) - January 30, 1976
- * 9. Chloroform - March 15, 1976
- 10. Radon Daughters - May 11, 1976
- 11. Dimethylcarbamoyl Chloride (DMCC) Revised - July 7, 1976
- 12. Diethylcarbamoyl Chloride (DECC) - July 7, 1976
- 13. Explosive Azide Hazard - August 16, 1976
- 14. Inorganic Arsenic - Respiratory Protection - September 27, 1976
- * 15. Nitrosamines in Cutting Fluids - October 6, 1976
- * 16. Metabolic Precursors of a Known Human Carcinogen, Beta-Naphthylamine - December 17, 1976
- * 17. 2-Nitropropane - April 25, 1977
- * 18. Acrylonitrile - July 1, 1977
- * 19. 2,4-Diaminoanisole in Hair and Fur Dyes - January 13, 1978
- * 20. Tetrachloroethylene (Perchloroethylene) - January 20, 1978
- 21. Trimellitic Anhydride (TMA) - February 3, 1978
- * 22. Ethylene Thiourea (ETU) - April 11, 1978
- * 23. Ethylene Dibromide and Disulfiram Toxic Interaction - April 11, 1978
- * 24. Direct Black 38, Direct Blue 6, and Direct Brown 95 Benzidine Derived Dyes - April 17, 1978
- * 25. Ethylene Dichloride (1,2-Dichloroethane) - April 19, 1978
- 26. NIAX[®]Catalyst ESN - May 22, 1978
- * 27. Chloroethanes - Review of Toxicity - August 21, 1978
- * 28. Vinyl Halides - Carcinogenicity - September 21, 1978
- 29. Glycidyl Ethers - October 12, 1978
- * 30. Epichlorohydrin - October 12, 1978
- * 31. Adverse Health Effects of Smoking and Occupational Environment - February 5, 1979

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