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RISKS TO HEALTH

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THE INTERACTION OF SMOKING AND WORKPLACE HAZARDS

RISKS TO HEALTH

by

K. Rothwell

Consultant, WHO Tobacco or Health Programme, Geneva
and WHO Office for Occupational Health, Geneva

THE RISK TO HEALTH FROM SMOKING AT WORK

Introduction

There are no longer doubts that tobacco use, particularly cigarette smoking, is the principal cause of several debilitating and often terminal diseases. It has also long been recognized that the workplace is a source of hazards that can cause disease and early death in many occupations. During the past two decades it has become clear that when workers smoke at their place of work, they are not merely exposed to two mutually isolated types of hazard: one attributable to the occupation and the other to tobacco smoke, but are subject to detrimental health effects arising either from a combination or from an interaction of the two.

Tobacco is used throughout the world; in countries with low income economies and in the most affluent industrialized nations. It is used by men and women, by children and adults, and millions of others are involuntarily subjected to environmental tobacco smoke. There are numerous explanations for the tobacco habit but the main reason for its ubiquity is the addictive drug nicotine present in all forms of tobacco leaf and delivered in varying amounts to the user by all the methods of usage that people worldwide have, over the ages, devised.

In many lesser developed countries, and particularly in the rural areas of these countries, traditional methods of tobacco use are widely practised but are being supplanted by cigarette smoking; a habit which by and large has replaced all other forms of use in the industrialized countries. Traditional ways of using tobacco have been legion but are all based on either smoking tobacco in some form of pipe or hand rolled tube of tobacco, or on chewing a mixture of tobacco with a variety of flavouring materials. The cigarette appeared and began to replace such methods of use in Europe, possibly as an imitation of papyrosi, after the Crimean War, and records dating back as far as the 1880's detail cigarette consumption in France and the United Kingdom from then to the present day. The reasons for the popularity of cigarette smoking emerging in Europe and the USA were probably twofold; on the one hand availability following the invention of a machine for large scale cigarette manufacture, and on the other, the greater ease and convenience that cigarettes brought to tobacco use. By the end of the second world war, cigarette smoking had become completely established as a socially acceptable habit for both men and women in the developed countries where, for a period during the 1950's, there may have been more smokers than non smokers. Today, in most developed countries, cigarettes account for at least 80% of the overall tobacco consumption and in most developing countries cigarette smoking is increasing rapidly and, in all but the poorest and most remote areas, is replacing other forms of tobacco use.

During the past 40 years, smoking has been recognized as being a serious health hazard and the main cause of death from many common diseases in most developed countries. In these countries, where the effects of smoking on health are generally appreciated and the economic costs and losses realized, legislation is being enacted, control is being taken through taxation, and action is being taken to educate the public not only on the dangers of smoking but also on the benefits to be gained from stopping. In only a small number of developing countries have the hazards and economic disadvantages that are associated with widespread tobacco use by the population been fully accepted even at government level, and there are not many of these countries that have as yet taken any decisive action to curtail the behaviour.

All working situations involve an element of danger: frequently it is associated with accidents, but harmful health effects often arise from the work itself, or in the environment of the working activities. Thus there are airborne mineral dusts in mining and biological dusts in farming and industries using biologically produced raw materials; fume is produced during welding; smokes, mists, vapours and gases present hazards in many industrial situations; excessive heat, ultra violet light and high levels of noise are frequently detrimental to the well being of workers; ionizing radiations in mining and modern technology are now recognized as workplace hazards and in many occupations workers are subjected to harmful mechanical vibration. These occupational conditions all take their toll on health and well-being but it is now also realized that their effects are far greater when they are combined with the added effects from tobacco smoking.

Tobacco growing involves the use of chemical agents which can harm the health of workers; harvesting can cause sickness due to skin absorption of nicotine; and processing exposes workers to health hazards from airborne dust and fungal spores. However, the greatest impact of tobacco on the health of the workforce as a whole is in the effects of smoking on other diseases related to the workplace. Smoking, particularly cigarette smoking, is detrimental to health in a wide variety of occupations not only because of the diseases it causes <u>per se</u> but because it adversely affects disease conditions in which other agent(s) may be aetiologically implicated. It has become clear that work hazards and the hazards associated with smoking at work cannot be separated, nor considered in isolation in the workplace. Apart from the now well known danger to non smokers of the smoking by coworkers being allowed to contaminate the shared atmosphere, there are other situations where smoking increases the severity of a disease to a level far in excess of what could be expected from smoking alone, from the occupational hazard alone, or even from the two effects added together.

In many industrialized countries, the hazards of the workplace have been recognized, regulations have been formulated and legislation has been enacted to protect workers and

provide for their education on the dangers arising from the nature of the occupations in which they are engaged; although in some countries, implementation of the rules and law is not always strictly imposed and workers sometimes neglect the training they have been given. In many newly industrialized countries where development is progressing swiftly, the health problems associated with the work have not yet been addressed and many workers are completely ignorant of the dangers to their health by which they are surrounded.

In most countries and in many working situations, the combined effects on health of smoking and occupational hazards have as yet been seldom fully recognized, or accepted by governments, employers, unions, or workers and the dangers of smoking at work have not been given serious attention except where there might be risks of large scale accidents from fire or explosion.

It is hoped that the following brief survey of the interaction of smoking with the hazards of the workplace will serve to illuminate some aspects of the situation and stimulate further research into problems which can affect not only the health of individuals but also the economic success of many industries and ultimately national economies.

1. The nature of cigarette smoke

Cigarette smoke is an aerosol consisting of a particulate phase composed of liquid droplets dispersed in a gas/vapour phase. When a cigarette is smoked, a large number of compounds are formed by pyrolysis at, and/or distilled from, the burning end. These either pass down the cigarette as mainstream smoke, some being condensed a short distance behind the burning cone, or are emitted into the air from the burning end as sidestream smoke. With each subsequent puff, the procedure is repeated but the smoke becomes progressively stronger because not only is the previously condensed material distilled again and added to the smoke, but the length of cigarette available for further condensation is constantly decreasing.

The nature of any tobacco smoke depends on a whole range of variables concerned with the type of tobacco and its growing and processing and many factors associated with its burning. In the case of a cigarette (or a bidi) there are many further variables that define the smoke chemistry such as dimensions, wrapper porosity and smoking parameters, i.e. the puff volume, frequency, and duration. However, in spite of the many variable factors that affect the nature of cigarette and tobacco smoke, the variation in smoke chemistry is mainly confined to changes in the concentration balance of smoke constituents rather than to the presence or absence of particular compounds.

Some 4000 compounds present in tobacco smoke have been identified and many have been quantified. Many remain that have not yet been identified. Numerous lists of smoke constituents and their concentrations in the smoke from different types of cigarette have been published: a WHO compilation(173) gives an extensive list of compounds and a large number of references. In the same way that the compounds can be classified according to their chemical structure, they can also be listed according to their biological activity in groups such as: chemical asphyxiants, irritants, carcinogens, enzyme inhibitors, neurotoxins, and pharmacologically active compounds.

Although the main point of entry into the body by cigarette smoke constituents is the lungs, many constituents, particularly in smoke from pipes and cigars, dissolve in saliva and are absorbed through the tissues of the buccal cavity or swallowed. Alcoholic beverages also provide an effective smoke solvent by which materials reach the oesophagus and stomach. Mainstream cigarette smoke particles vary in size from 0.15μ to 1.3μ, with a mean size around 0.4μ. In sidestream smoke the particles are somewhat smaller, varying from 0.01μ to 0.1μ(174). Thus, from size considerations, the aerosol particulate matter along with the vapour phase constituents and the permanent gases, is capable of reaching the alveoli when smoke is inhaled. Whilst the deposition of components of all the phases of smoke in the tracheobronchial tree is complicated by the behaviour of hydrophilic constituents in the high humidity conditions and by the fact that some electrostatic precipitation occurs, it has nevertheless been demonstrated that smoke reaches every part of the trachea, bronchi and alveoli.

Some methods of cigarette smoking

Information on the physical and chemical nature of cigarette smoke is obtained from smoke produced under standard conditions, e.g. a smoking pattern consisting of 35mm^3 puffs of two second duration taken at one minute intervals down to a specified butt length. It can be shown that the constitution of the smoke is sensitive to changes in these parameters which, in only a general way reflect the way in which a smoker produces smoke. Furthermore, the way in which each individual uses the smoke produced is far from standard. One smoker might light a cigarette and then leave it to burn away, whilst another will feverishly puff to the last millimeter of available tobacco. Some draw the smoke into the mouth and then blow it out again whereas others inhale each puff into the deepest recesses of the lungs. Some smokers retain a lit cigarette between the lips, infrequently taking a definite puff but inhaling a continuous stream of both mainstream and sidestream smoke: a habit frequently seen among workers who need to have both hands free to perform their task. It is clear that methods of smoking must have an impact on the fact that some smokers more quickly fall victim to smoking related disease than do others.

The most unfortunate smoker is the involuntary, or obligatory smoker, condemned to breathing the smoke laden atmosphere produced by other smokers in the vicinity.

2. Diseases caused by workplace hazards and/or smoking

Chronic Obstructive Lung Disease (COLD)

Poor social conditions, inadequate nutrituion, overcrowding and excessive atmospheric pollution were the social and environmental factors considered to be responsible for the widespread bronchitis in nineteenth century England. Bronchitis was, however, ill defined and occurred in other countries but was defined there otherwise. With the amelioration of working and living conditions, the amount of bronchitis should have decreased, but the cigarette boom of the twentieth century has ensured high prevalence of the disease throughout the world.

Bronchitis has remained ill defined until recent years. It is now considered to be one of several diseases classified under the collective heading of Chronic Obstructive Lung Diseases (COLD). In addition to chronic bronchitis, the other members of the group are: small airways disease, toxic bronchiolitis obliterans (other forms of bronchiolitis obliterans occur but are of less importance in the context of occupation related disease), emphysema and fibrosis.

Bronchitis. "Industrial bronchitis", is defined(1) "as a condition characterized by cough and sputum for at least three months of the year, which may or may not be accompanied by airways obstruction, and which is a consequence of prolonged inhalation of dust or irritant gases at the workplace". In smokers lungs the effect of dust is overwhelmed by the irritant constituents of tobacco smoke which not only cause hypersecretion of mucus and alter it's physical properties and chemical structure, but also impair the ciliary clearance mechanism.

Mineral dusts, particularly in all forms of mining, cause bronchitis, as do many biological dusts, irritant vapours and gases and both inorganic and organic chemical dusts and sprays.

Small Airways Disease (SAD) is a widespread narrowing of membranous bronchioli; it is inflammatory in origin and is often associated with excess mucus and an accumulation of macrophages in the respiratory bronchioli. SAD can be associated with environmental and industrial pollutants, but it arises mainly as an injury caused by smoking. When dust is deposited in membranous bronchioli, it can induce fibrosis.

Toxic bronchiolitis obliterans, an extreme form of SAD is caused by the inhalation of toxic gases and fumes, such as phosgene, oxides of nitrogen, sulphur dioxide, and smoke from fires, and it leads to widespread bronchiolar obliteration.

Emphysema is defined as an enlargement of the air spaces distal to the terminal non respiratory bronchioli, accompanied by destructive changes in the alveolar walls. It tends to be prevalent in older age groups and follows small airways disease. Macrophages which have engulfed foreign particles, including smoke particulate matter in the lungs of smokers, and which have been found accumulated in the bronchioli(2) and in the lung parenchyma(3) have been implicated in the pathogenesis of emphysema. There are different forms of emphysema recognized which vary with the nature of the insult to the tissues. For purposes of post mortem examination of slices of lung, emphysema has been defined(4) as the presence of air spaces of 1 mm or more in size.

Pulmonary fibrosis, the abnormal formation of fibrous tissue, or scar tissue, is the response of bronchiolar tissue to deposition therein of inhaled particles. Different types of particles, for example: coal dust, silica or asbestos, cause different types of reaction by the lung tissue. The various types of radiological changes seen in early stages of these fibroses are associated with relatively minor pulmonary impairments, but continuous exposure leads to a greater degree of fibrosis and to progressive massive fibrosis in some subjects. On histological, animal experimental and roentgenographic evidence, Weiss(5) concluded that cigarette smoking results in diffuse fibrosis.

It could be argued that the COLD diseases, although perhaps not the most dramatic in the public's eye, are probably among the most important of diseases caused by smoking and/or occupational and environmental pollution, not only from the health aspect but also because of their negative economic impact on a community. In a study of 215 229 adults in a region of Czechoslovakia(6), smoking was the most important risk factor. Risks for male non smokers and light smokers under 30 years of age were 1.18% and 2.28% respectively; for men aged 50 years, smoking more than 20 cigarettes a day, the risk was 20.36% compared with 3.31% for non smokers of the same age.

Cancer

There are many forms of cancer that have been associated with particles, vapours, fumes and gases present in the working environment. Lung cancer is probably the commonest and has been associated with many industries using asbestos; with working in metal molding, or in coking plants; with motor vehicle drivers; with several types of mining because of the accumulation of α-emitting radioisotopes; and with industrial contact with such materials as arsenic, chromates, chloromethyl ethers, mustard gas and polynuclear hydrocarbons. Mesothelioma has been associated with exposure to asbestos;

nasal and sinus cancer with nickel refining and among woodworkers; leukaemia with ionizing radiations and with occupations using benzene; bladder cancer has been found in workers involved in the manufacture and use of dyes and with rubber industry workers; liver cancer has been linked with the use of vinyl chloride; and an association of scrotal cancer with coal tar is suggested from the fact that a decrease in the disease coincided with the closure of a tar distillery. Excesses of cancers of the large intestine, bladder and stomach have been found in machinists and related occupations; of lymphoma and cancers of the bladder and brain in electricians; and cancers of the buccal cavity, pharynx, larynx and brain in telegraph, telephone and power linesmen.

There have been many examples of an association between occupational hazards and lung cancer and yet, "the vast majority of the evidence available at the present time(1) suggests that (only) around 4 to 5 per cent of all lung cancer is related to occupational exposure". Thus, occupation is of limited importance in the problem of lung cancer as a whole. Smoking accounts for 90%. It is likely that the occupational contribution to some other forms of cancer is also limited.

Cardiovascular Disease

In countries where cigarette smoking has been a long established custom it is responsible for around 25% of deaths from cardiovascular disease. Since, in many of these countries, up to 45% of all deaths are caused by cardiovascular disease, compared with up to 8% of all deaths being due to lung cancer, it is clear that, of the two, most smoking related deaths are attributable to CVD.

Two components of tobacco smoke, nicotine and carbon monoxide, are the principal materials responsible for the harmful cardiovascular effects. Nicotine from cigarette smoke is rapidly and completely absorbed in the lungs; from pipe, cigar smoke, or chewing tobacco, it is slowly absorbed from the buccal mucosa. Nicotine causes increases in heart rate and blood pressure; it stimulates structures, such as the central nervous system, that are activated by acetylcholine release; it causes an increased mobilization of free fatty acids in the serum; and enhances platelet adhesiveness. These effects are responsible for more cardiac work (which for individuals with some forms of heart disease will not be met by increased coronary heart flow), with an interference with metabolic exchange across capillary walls, with ischemic episodes and an initiation of thrombosis. Carbon monoxide from cigarette smoke increases the carboxyhaemoglobin concentrations in the blood and lowers its oxygen carrying capacity: increased oxygen debt after exercise and impairment of endurance performance are evident in smokers, compared to non smokers. Carbon monoxide also has a great affinity for myoglobin and may interfere with oxygen uptake by the myocardium. Cigarette smoking has been generally considered to be the

primary cause of thromboangiitis obliterans since the disease was first described in 1908, and its effects are alleviated by smoking cessation. Its effect on the peripheral microvascular system is evident from the reduction in temperature caused by cigarette smoking, that can be demonstrated by hand thermography, and by measurable reductions in skin temperature that occur. There is a large excess of deaths, frequently sudden deaths, from cardiovascular disease in smokers, which is related to the number of cigarettes smoked per day, and reduced CVD mortality is associated with smoking cessation. Cigarette smoking is considered to be the most important cause of chronic bronchitis and could also be the primary cause of emphysema. Both conditions aggravate heart disease and may cause chronic cor pulmonale(7). It is likely that the hydrogen cyanide, the oxides of nitrogen and some of the several inorganic elements such as arsenic, cadmium and lead, present in tobacco smoke will also contribute to cardiovascular disease.

Cardiovascular disease is also associated with various forms of occupational exposure, which can be classified as physical, chemical and biological, and with occupational lifestyle. Physical exposures include extremes of barometric pressure, vibration, noise, and extremes of temperature and humidity. Chemical agents, which may be absorbed by inhalation, skin absorption, or ingestion, can produce cardiovascular toxicity by directly affecting the myocardium or by impairing the oxygen carrying capacity of the blood and causing anoxic damage to the vascular system, or by enzyme inhibition. Biological agents may be encountered in laboratories, hospitals or endemic areas where a worker becomes exposed. Occupational lifestyles may be sedentary or involve great physical activity and within the spectrum of these the "personality type" can also influence the incidence of cardiovascular disease.

The combination of smoking and an occupational exposure will, in every case, predispose to an increased detrimental cardiovascular effect. The anoxia concomitant with working at high altitude along with reduced oxygen carrying capacity of the blood due to carboxyhaemoglobin from cigarette smoke will act in concert to cause anoxic damage and oxygen debt. The normal bodily reactions to heat and humidity or to cold and submersion will be changed by the effects of nicotine on vasodilation and vasoconstriction. Chemical agents which may occur in the working environment and are known to be present in cigarette smoke, and which are known to have cardiovascular effects, will have an increased effect when accumulated from the two sources. Interaction effects can be expected when the cardiovascular system, itself affected by hazardous materials from one source, is stressed to overcome toxicity or impaired physiological activity arising elsewhere in the body because of agents from another source.

The many interactions giving rise to cardiovascular disease, whether occupational, dietary, hereditary, or due to lifestyle and physical activity, create difficulties for research into understanding the nature of the effects which result in the high levels of morbidity and mortality attributable to cigarette smoking and the combined effects of smoking and occupational hazards on CVD have not been as extensively studied as have the interactions between smoking and COPD or cancer.

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3. The combined effect of smoking and workplace hazards on health

There are four principal ways in which smoking can interact with workplace hazards to impair the health of the worker who smokes. They are not mutually exclusive and in fact there are many situations in which the first two occur together in many workplace situations.

3.1. Tobacco smoke can modify the detrimental health effects associated with materials encountered in the workplace, in some cases causing a highly elevated disease risk, for example, the effects of cigarette smoke on diseases related to asbestos, coal dust, α-radiation.

3.2. Chemical compounds associated with injury to health are often found in both tobacco smoke and the working environment and each source can augment the dose obtained from the other, e.g. carbon monoxide, acrolein, benzene, heavy metal elements.

3.3. Materials used in the workplace, which produce harmful chemical agents when they are burnt or vaporized, can be transferred from the workplace onto smoking materials and cause the smoke to be far more injurious when the tobacco is smoked, e.g. polytetrafluoroethylene, methylparathion.

3.4. Tobacco smoke can affect a physiological process and increase the impairment of physical or physiological functions initiated by certain occupational conditions, e.g. the effect of smoking on the peripheral vascular system can enhance the effects of vibration and noise.

Although there are now many well documented examples of all these types of interaction between smoking and various occupational hazards, it would be useful if further studies were carried out in industries where they may hitherto have been neglected, perhaps because sufficiently large sample sizes of

workers/smokers/non-smokers/controls were lacking, or because information on occupational and smoking histories had not always been obtained from employees.

In devising questionnaires and interpreting results, it is necessary to consider job migration and distinguish between ex-employees who resigned for age or economic benefits and those who may have left because of the onset of symptoms of ill health; and in choosing controls within a community consideration must be given to the possibility that some may have avoided an occupation because of fear of, or predisposition to, a disease condition.

3.1. The interaction of tobacco smoke and exposure to workplace hazards

Tobacco smoke can modify the detrimental effects associated with materials encountered in the workplace, in some cases causing a highly elevated risk.

Airborne Mineral Dust

- Mining, exposure to coal dust
- Industries using asbestos and asbestos products
- Operations using non-asbestos fibrous minerals.
- Occupations involving exposure to silicaceous materials.
- Occupational exposure to α-Radiation
- Other non radioactive, non fibrous mineral dusts

Airborne Biological Dusts

- Textiles
- Grain and other vegetable dusts
- Fungal spores and microorganisms
- Woodworking

Waterborne hazards

Airborne inorganic elements and compounds

- Elements and compounds in fume, smoke, mists
- Welding
- Inorganic gases

Organic compounds encountered in various industries, such as:

- chemicals
- petroleum
- dyestuffs and dying
- coking
- rubber
- aluminium
- agriculture

Airborne mineral dust

Coal Dust

Chronic bronchitis of coal miners who are smokers is probably a combination of mucus hypersecretion due to dust; masked by mucus hypersecretion and impaired lung clearance caused by smoke; along with small airways disease caused by smoke; and the effect of dust on small airways inflamed by smoke. It is possible that emphysema proceeds from small airways disease, rather than being primarily caused by dust, and thus there are more cases of emphysema in coal miners who are smokers than in non smokers. The primary cause of coal workers pneumoconiosis is coal dust. There is no association between coal dust and lung cancer; only between smoking and cancer.

It was realized in the 16th century that coal mining carried a risk of respiratory disease and, in the 19th century, black masses in miners lungs were described. It is now known that coal miners can suffer from Chronic Bronchitis, Simple Coal Workers Pneumoconiosis, Progressive Massive Fibrosis, and Emphysema. The respiratory impairment appears as radiologically observed and functional changes in the lungs, but whilst some of these changes are associated solely with coal dust, some are also closely associated with smoking. In the past it has been difficult to apportion attributable risk to the two causes because coalface workers, the group subjected to the highest dust exposure, have a different smoking pattern and perhaps a different daily consumption of smoking materials than non coalface workers because smoking in the mine is forbidden. With the limitation of dust in modern mining, the effect of the two factors: coal dust and smoking, is now becoming clearer. An added difficulty in interpreting data comes from the fact that non smokers may accumulate greater dust exposure because they have less work absenteeism and live longer.

In most of the countries surveyed, the prevalence of smoking by miners tends to have been somewhat higher than in either the male population as a whole, or among most other occupational groups: although a distinction is seldom drawn between miners and coal miners except in studies centred on coalmining populations. Nothing is known of the situation in countries where women are employed in mining.

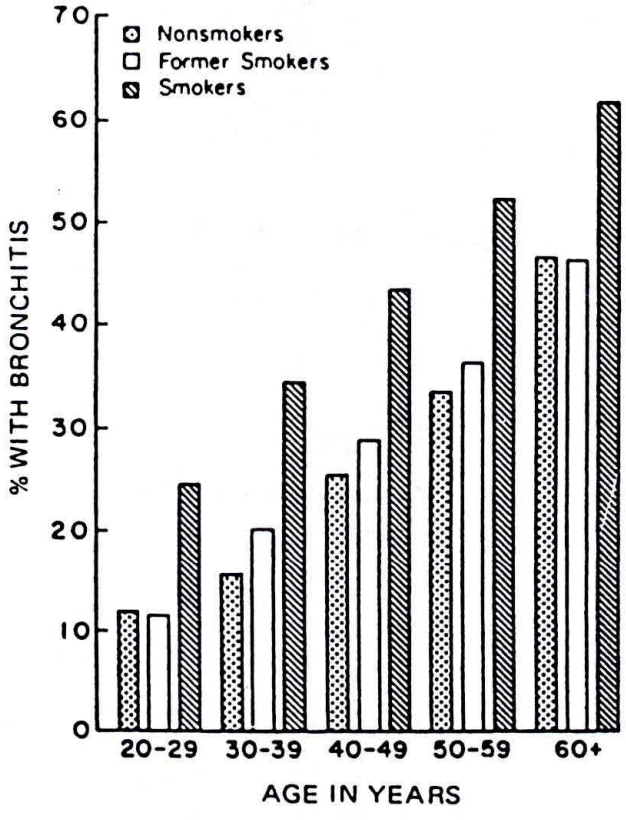
In the UK, from 1963 to 1975, smoking prevalence in the male population(8) (and among miners) fell from 54% (miners 77%) to 47% (miners 49%).

The New Zealand population census 1976 gave the smoking prevalence for the male population as being 39% and for miners, 57%; similar to transport workers and labourers. In a report on the 1981 census(9) the smoking prevalence quoted for men was 35% and for the group comprising production workers, transport operatives and labourers, (the group which included miners in the earlier census) the figure given was 43%.

In a study of 8555 miners from 29 bituminous coal mines(10), over 50% were smokers and 25% were ex-smokers. In another study(11), only 13% of 1677 coal miners from 5 British collieries, who were examined in a lung function study, were non smokers; 66% were regular smokers and the remainder were intermittent or ex-smokers. In a 20 year follow-up study of a population of coal miners and others(12), 69% of the coal miners were smokers. These examples typify the smoking prevalence of coal miners prior to the early 1980's. A 1982 survey of the smoking habits of 800,000 American men and women in relation to their occupations(13) found that among miners (type unspecified) 29.4% had never smoked regularly, 31.5% were current smokers and 39.1% were former cigarette smokers. This may reflect a general trend in the countries with higher income economies where smoking has been tending to decreased throughout many sections of the population over the past 10 years.

Bronchitis. Kilbestis et al.(10) found that the prevalence of bronchitis in coal miners who smoke was higher than in their non smoking coworkers (figure 1). Coalface workers had more bronchitis and more airway obstruction than surface workers and the difference between smokers working at the coalface and non smoking surface workers showed that the effect of smoking was five times greater than that of coal dust. The following table shows the results of an epidemiological evaluation of chronic obstructive bronchitis in 5605 miners (M), 1276 ex-miners (EM) and 3898 individuals who had never worked in a mine (NM)(14).

Fig. 1. Prevalence of bronchitis by age and smoking status in 8492 bituminous miners.



	NM%		M%		EM%	
	<29 yr	>50 yr	<29 yr	>50 yr	<29 yr	>50 yr
Chronic bronchitis						
Non smokers	1.1	20.5	1.6	9.6	0	15.0
Smokers	2.4	28.2	4.8	34-6	3.3	31.5
Obstructive symptom						
Non smokers	4-6	10.3	0	10.8	0	12.5
Smokers	6.1	14.2	10.3	16.4	6.6	16.0

Smoking is seen from these figures to have a more serious effect on miners than upon other groups. It is not unlikely that within a mining community, the other two groups may have a predisposition to the disease and have achieved their status by job selection or job escape: from a study of new entrants into coal mining(15) it was found that those who drop out of mining tend to be less physically fit and more prone to chest problems than those who remain.

Morgan(1) discussed analyses of the effects of cigarette smoking, dust exposure and environmental factors on respiratory disease, from which it appeared that bronchitis and airways obstruction are two separate responses to cigarette smoking. Bronchitis leads to hypersecretion of mucus and affects the large airways. The airflow obstruction found in smokers is due to small airways disease and involvement of respiratory bronchioles leading to the development of emphysema. In coal miners, prevalence of bronchitis in non smokers lungs is related to the degree of dust exposure. This is most evident in non smokers because, in smokers, the effects of tobacco smoke overwhelm and obscure the effect of dust. Airways obstruction is caused more by cigarette smoke than by dust. From a study(16) of the relationship between chronic bronchitis, smoking and dust (unspecified source), heavy smoking was equated with 20 years of exposure to dust.

It is reasonable to conclude that the chronic bronchitis of miners is probably a combination of:

- mucus hypersecretion caused by dust;
- mucus hypersecretion, mucus modification and clearance impairment caused by tobacco smoke;
- small airways disease caused by tobacco smoke;
- the effect of dust on small airways tissue already inflamed by smoking.

Emphysema and pneumoconiosis. The issue of emphysema in coalworkers appears to have been controversial. From a study(17) of coalworkers and non coalworkers it was concluded, after taking any effect of smoking into account, that there was a seven times excess of

emphysema in coalworkers. Results of post mortem examinations of 866 Australian miners(18) showed a positive correlation between dust exposure and emphysema and pneumoconiosis for which the severity was highest in non smokers. However, smoking and non smoking coal-face workers were not compared. Chronic bronchitis was positively correlated with smoking. From a post mortem comparison of lungs from 450 coal miners(4) it was found that emphysema occurred more frequently in those that smoked (72%) than in ex-smokers (65%) or in non smokers (42%) and the relative frequency increased with age at death. The study considered the possibility that coal dust may cause emphysema which inhibits clearance and in turn promotes fibrosis, or alternatively that fibrosis caused by dust increases the chance of emphysema. A study of South Wales coal miners(19) militated against dust induced emphysema. It has been suggested that differences in emphysema between coalworkers and non coalworkers can be accounted for by current smokers. Morgan(1) concluded a brief review by considering that the evidence militates against obstructive emphysema occurring more commonly in coal miners than in the general population or that more dust inhalation leads to a greater likelihood of emphysema developing. There have been several reviews of small airways disease (SAD) which suggest that emphysema proceeds from smoking induced SAD. Cosio, et al.(20) considered their observations to support the hypothesis that SAD is causally related to centrilobular, but not necessarily to panlobular emphysema. Dust in coal mining appears to be the primary cause of coal workers pneumoconiosis.

Cancer. Perhaps due to failure to control confounding factors, there has been a lack of consistency among reports on the relationship between coal mining and the incidence of lung cancer in miners. A direct evaluation of the relationship between lung cancer mortality and coal mine dust exposure, controlling for smoking status(21), found no evidence of a link between coal mine dust exposure and lung cancer risk, nor of an interaction effect, although the expected lung cancer risk in cigarette smokers was observed. A hypothesis(22) that increased pulmonary retention of carcinogenic particles as a result of impaired ventilatory function would predispose to lung cancer and that normal ventilatory function, permitting efficient lung clearance and subsequent swallowing of carcinogens, would lead to stomach cancer among coal miners has been tested(23). The results contradicted the hypothesis, suggesting that obstruction may set a condition for stomach cancer and normal lung clearance may set a condition for lung cancer and that the inhaled carcinogenic agent is different for each type of cancer. For miners with airways obstruction or long term smoking, coal dust poses a stomach cancer risk and for miners with normal ventilatory function, current cigarette smoking poses a disproportionately high lung cancer risk. From a study of dust exposure, pneumoconiosis and mortality of coal miners(24) it was found that lung cancer mortality among miners who smoked was 5.5 times higher than in never smokers but that the effect was entirely due to smoking.

Radon and radon daughter contamination of the dust in coal mines would be expected to be as prevalent as in all other mines and thus the apparent very low lung cancer risk in coal mining might be unexpected. However, because of the explosion risk in coal mines, their ventilation is usually efficient and a build-up of radioactivity is probably far less likely than in other types of mine.

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Occupational exposure to asbestos

> Exposure to asbestos can cause lung cancer in both smokers and non smokers but the risk of lung cancer from a combination of both asbestos and smoke is far higher than the sum of the two separate risk factors and the great preponderance of lung cancers occurs in asbestos workers who are smokers. Death rates from chronic lung disease in smoking asbestos workers are higher than those for either non smoking asbestos workers or smoking non asbestos workers and the effects of asbestosis and smoking are probably additive. Asbestos causes mesothelioma, but this is not affected by smoking.

Asbestos is a generic name for a group of fibrous silicates that have found widespread use over the past 100 years for their heat, fire, acid and to some extent alkali resistant properties. Asbestos types are classified according to their physical characteristics as serpentine or amphibole. Chrysotile is in the first group and consists of long pliable white fibres. Amosite and crocidolite are two varieties in the second group, members of which tend to have straight needle-like fibres. The long fibre asbestos varieties can be spun into yarn which can be woven into fabric; shorter fibre varieties are incorporated into cement, board and tiles. Asbestos products have been used for the fabric of buildings, for electrical and thermal insulation, for fire and safety equipment and for the brake linings of cars. It has found use in shipbuilding and the glass industry, in domestic electrical and heating equipment and in theatres and cinemas for fire curtains. Just as workers in many manufacturing industries may be exposed to various forms of asbestos, many other workers are exposed in maintenance work and in demolition and recycling operations: sometimes more intimately than the workers using new asbestos products in manufacturing. Workers in mining and separation of the mineral from rock also have high exposure.

Airborne asbestos was recognized as a cause of pulmonary fibrosis in 1898 and with its increasing use came an increasing realization of the health hazards inherent in it's use. Case reports were published in 1924 and 1927(25,26) and in the ensuing years the range of asbestos related diseases has been extended to include not only asbestosis

(parenchymal and pleural fibrosis) but also lung cancer(27), pleural mesothelioma(28) and cancer of other sites(29), and there have been many studies on the combined effects of smoking and asbestos exposure. The several forms of asbestos differ not only in their colour and fibre length but also in their relative carcinogenic potential, although there is still some debate on this. However, the amphibole varieties appear to carry the highest carcinogenic risk. Eronite, a fibrous zeolite, and tremolite are used for building materials in some parts of the world and there is a high prevalence of mesothelioma in these regions. Different degrees of synergism that have been observed between smoking and asbestos exposure are probably partly linked to the different carcinogenic potential of the various types of asbestos. However, there is no doubt that the cancer risks for any smoker who may encounter asbestos are extremely high.

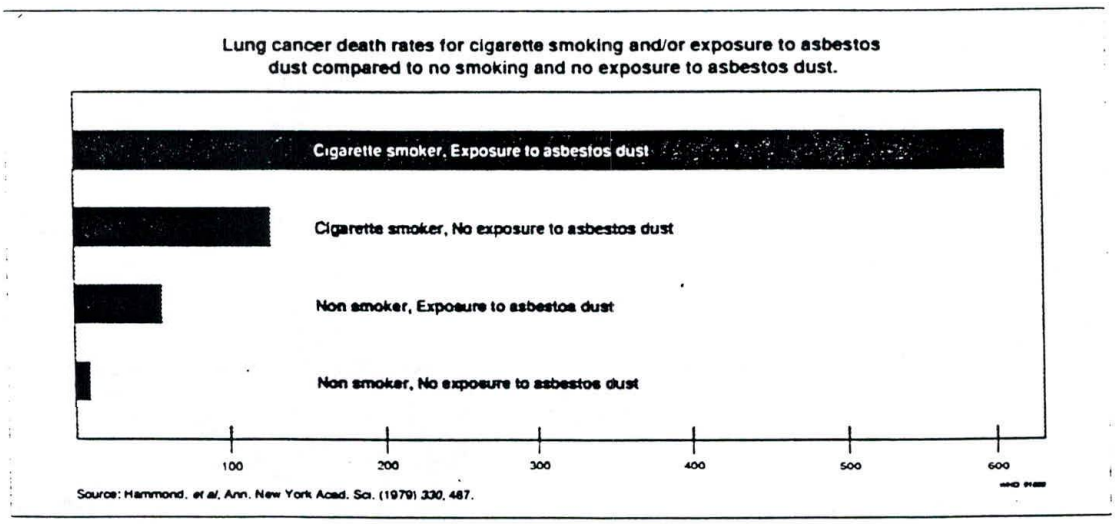
Because there are so many very different occupations in which asbestos exposure might occur, along with a range of possible levels of exposure and a variety of types of asbestos in use, it is difficult to generalize on the smoking habits of all asbestos exposed workers. Furthermore, in many studies, the number of smokers within groups of workers with asbestos related disease have been recorded rather than the smoking habits of all the exposed population. From the information available, it appears that the smoking habits of asbestos exposed workers have probably reflected those of blue collar workers in general. The following are a few examples of smoking prevalence in different groups of asbestos workers:

Asbestos textiles workers	Ref(30)	75% smokers
Asbestos miners	Ref(31)	46% cigarette smokers 36% ex cigarette smokers 5.5% pipe/cigar smokers
Electrochemical plant (two areas)	Ref(32)	84% to 87% were smokers or ex-smokers
Population in Telemark, Norway	Ref(33)	Asbestos exposed: 44.6% smokers 36.0% ex smokers No asbestos exposure: 40.9% smokers 28.6% ex smokers
Survey/800,000 American men & women in 1982	Ref(13)	Asbestos exposed: 33.6% smokers 47.3% ex smokers.

Lung cancer and asbestos. In 1968(34) cigarette smoking was shown to be an additional hazard among asbestos workers. This was a small study and no lung cancer deaths were found among non smoking asbestos workers. A similar observation was also recorded in another small scale study(35). Since then, many reports on the interaction of cigarette

smoking and asbestos exposure have been published. It has become clear that, in combination, the two hazards are associated with very high lung cancer rates (figure 2) and attempts have been made to understand the nature of the effect. Either cigarette smoke and asbestos could act independently and their combined effect would be the sum of their individual effects, or there could be synergism between the two and the ultimate effect would be a product of the two risk factors. Many studies have been carried out(36 to 41) and have been discussed by Steenland,K. & Thun,M.(42). In some studies, the effects of smoke and asbestos appear to be additive and in others either multiplicative or somewhere between the two. Several reasons for lack of consistency can be invoked: sample size, type of asbestos, time scale, age and intensity of exposure to asbestos and smoking history. Both smoking prevalence and cigarette consumption are difficult to obtain with accuracy, particularly from sources other than the individual concerned, furthermore, over the 25 to 30 year period that the individuals who were the subjects of surveys had worked with asbestos, and been smokers, there was a considerable change in the tar yields of cigarettes. Between 1933 and the late 1940's, the yield from an average cigarette could have varied from 33 to 49 mg tar and up to 3 mg nicotine(43), whereas in the 1960's and 1970's the average yield fell to around 16 mg tar and 1.5 mg nicotine. However, whatever may be the smoking/asbestos interaction influencing the incidence of lung cancer, the fact remains that there is an extraordinary increase in the risk for the asbestos exposed worker who smokes (figure 2).

Figure 2



Mesothelioma

There is no evidence that smoking affects the other types of neoplasm that have been associated with asbestos. Whilst up to 90% of mesothelioma cases have been attributed to asbestos, the incidence is not affected by smoking(41,44,45).

Asbestosis. The normal biological response to tissue injury is fibrosis and the lungs react to asbestos in this way. The type of roentgenographic opacities formed are different from those attributable to silica or coal dust and hence the term asbestosis is used. The formation of fibrotic tissue might be expected as the natural reaction of lung tissue to the many harmful constituents of tobacco smoke, however, chronic lung injury caused by smoking alone does not appear to lead to extensive fibrosis. In a review(5) of histological evidence, evidence from animal experiments and roentgenographic evidence, it was concluded that cigarette smoking can result in diffuse fibrosis similar to that caused by asbestos and that the changes showed a dose response to the duration and degree of smoking: although the two agents cause apparently similar effects, it was concluded that an assessment of the contribution of each during combined exposure required further study. The question of whether cigarette smoke causes changes in the lung consistent with fibrosis remains controversial. Nevertheless, it has been shown that death rates from chronic lung disease are increased by both smoking and asbestos exposure and death rates of asbestos workers who were also smokers is greater than the sum of the effect of the two individual exposures.

It has been evident that a dose of asbestos capable of an excess risk of lung cancer would also produce asbestosis, but no connection between the two was implied. A recent prospective mortality study(46) has shown that asbestosis is a precursor of asbestos related lung cancer. In this study, all cases of lung cancer developing from asbestosis were smokers and thus it was not possible to make any assessment of the interaction of hazards.

A summary of the situation on smoking and asbestos is contained in the following quotation(45): "1) the relative risk of contracting lung cancer from asbestos is approximately the same for smokers and non smokers; 2) the great preponderance of lung cancers, in absolute terms, occurs in asbestos workers who are smokers; 3) asbestos exposure and smoking act synergistically in causing lung cancer, producing an effect that is greater than the sum of their individual contributions; 4) smoking appears to have the more powerful effect on the incidence of lung cancer; and 5) the risk of death in asbestos exposed persons who stop smoking is decreased considerably". Although it is unlikely that smoking initiates the fibrotic process that leads to asbestosis, it may affect it in terms both of the extent to which clinical asbestosis becomes manifest and of its rate of progression.

Occupational exposure to non-asbestos fibrous minerals

Fibrosis (and mesothelioma) have been attributed to certain non-asbestos fibrous minerals. The added effect of smoking has not been assessed. Bronchitis and related symptoms are the effects found in smokers working with other fibrous silicates, such as wollastonite, used as substitutes for asbestos. There is insufficient evidence from which to draw conclusions on the health effects of fibreglass.

From a study in the Anatolian Region of Turkey(47) it was concluded that fibrous zeolite minerals in the soil and building materials used in the area were the cause of pleural and parenchymal lung fibrosis among the inhabitants. Pleural mesotheliomas have also been described(48) in communities exposed to zeolite minerals. Several other non-asbestos fibrous mineral have also been associated with pulmonary fibrosis.

Wollastonite is a fibrous monocalcium silicate which has been used as a substitute for asbestos as a filler and flux in ceramics, in grinding wheels, refractory products, building blocks and acoustic tiles. Exposed workers were surveyed in 1976 and 1982(49). A significant effect was observed for chronic cough, phlegm and bronchitis due to smoking. Reduced pulmonary function was attributed to wollastonite. However, the study population was small and interaction between smoking and dust exposure was not considered.

Although it has been suggested(50) that fibreglass may be carcinogenic, because it can be in a form with physical properties similar to those of asbestos, the matter remains controversial and there is insufficient evidence from which to draw definite conclusions concerning either exposure to fibreglass alone or the added effect of smoking. The effect of other vitreous fibres such as glass wool remain debatable.

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Occupational exposure to silica

Silica dust appears to be the sole cause of silicosis. A complication of silicosis is pulmonary insufficiency and this will be enhanced by smoking. Silica dust would be expected to cause bronchitis, but in smokers, smoking induced bronchitis masks any other.

In addition to coal-workers pneumoconiosis, silicosis is the other major occupational, non-asbestos, mineral dust generated fibrosis. Within different industrial communities it has been realized for as long as their occupations have been in existence that there are certain diseases peculiar to their work; known colloquially by terms such as "potters' rot", "miners' phthisis" and "grinders' asthma". Silica related pulmonary diseases are not only a hazard of most mining operations but they can affect workers in a wide variety of occupations where silica dust is generated. Bricklayers; cement makers; pottery, porcelain and ceramic workers; anyone involved in rock drilling or in chipping, grinding or polishing stone, or in sandblasting; in using grinding stones to smooth or polish other materials such as precious stones, or metals, or optical glass; or in the manufacture of polishing materials such as sandpaper, metal polishes and toothpaste, represent a small number of those exposed to silica. The number of industries in which silica dust can occur is very large and the extent of silica exposure of workers in different occupations very variable: the percentage of silica in the respirable dust can vary and silica being an active adsorbent, it can become contaminated with other materials in the workplace atmosphere and be altered in it's potential for toxicity. Furthermore, it has been suggested that freshly fractured silica dust may exhibit a different surface reactivity and cytotoxicity from that of aged silica(51).

Since the number and range of occupations in which silica exposure is possible are large, it is not possible to generalize on the smoking habits of exposed workers. Almost all, however, will be blue collar workers and thus the smoking prevalence is likely to be higher than that of the general population, as the following examples show. Among workers in a Swedish iron ore mine in 1984 who had worked underground during all their working life (average 31 years), 37% were never smokers(52) and in a slightly older group who had spent most of their working lives underground (average 34.5 years) but now worked on the surface, only 23% were never smokers. Ex smokers in the two groups were 29% and 39% respectively. Only 25% of surface workers were smokers; 51% were ex-smokers. In a survey of slate workers in North Wales in 1981, 57% were found to be cigarette smokers and only 14.7% were never smokers(53). High smoking prevalence was found among Australian gold miners(54): in 1985 56.2% were current smokers, 18.5% ex-smokers and 25.3% non smokers. In the survey of the smoking habits of 800,000 American workers in 1982, only 19% of workers exposed to coal or stone dust had never smoked regularly(13).

There have been many studies of the mortality of silica exposed workers and mortality rates have been consistently higher than for non-exposed workers, however, in the majority of the studies, the effects of smoking were not taken into account. Occupational silica exposure can be associated with chronic silicosis, a condition which develops after 20 to 40 years of exposure, and acute silicosis which has a more rapid onset after exposure. There are also other types of silicosis which relate to the nature

of the dust. Chronic bronchitis and airways obstruction have also been associated with silicaceous dust exposure. The chief complications of silicosis are pulmonary tuberculosis, respiratory insufficiency and non specific pulmonary infection: even today, pulmonary tuberculosis is still the most frequent complication(55).

The most widely held hypothesis for the pathogenesis of chronic silicosis is that silica particles are phagocytosed by the alveolar macrophages to which they have a marked selective toxicity. Autolysis of the macrophages initiates a series of biological reactions leading to the formation of collagenous fibres. It has been suggested that acute silicosis arises from the inhalation of more highly reactive silica particles(51). From the work to date it would appear that silica is the sole aetiological agent for all forms of silicosis, although in a study of granite workers exposed to granite and quartz dust(56), 233/784 showed opacities that were considered to be due to both dust and smoking.

Bronchitis is usually a consequence of prolonged inhalation of dust and might be expected to result from silica exposure, but in a survey in the Transvaal(57) a higher prevalence of chronic bronchitis was observed in smoking gold miners than in non-smoking coworkers and there was no difference between non-smoking miners and non-smoking controls. In the case of coal miners (vide supra) the effect of dust on bronchitis was completely overwhelmed by the effect of smoking. In a study of iron ore miners in Sweden(52) a strong relationship was found between chronic bronchitis and smoking but there was no relationship between chronic bronchitis and working underground. The two risk factors, silica dust and smoking, appear to be additive but the smoking effect is far greater than that of silica dust.

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Occupational exposure to ionizing radiations

The ionizing radiations from radon and its radioactive decay products are a primary cause of lung cancer in exposed workers in many mining operations. Exposed workers who are also smokers have the usual risk attributable to smoking added to the tumour risk due to α-radiation, along with an accelerating effect due to tumour promoters occurring in cigarette smoke. Thus the chance of lung cancer is greater and the latent period shorter and so the cancers appear sooner in smokers than in non smokers.

Radon, a gas, is produced by radioactive decay of the radium present in all rocks and soils. It constantly emanates from the earth and, although present in the atmosphere in extremely small amounts because of dilution, it can collect in ill ventilated spaces and is found in caves and many underground mines in significant concentrations. Radon, not in itself the main hazard, has a half life of almost 4 days and undergoes radioactive decay to form other radioactive products, known as radon daughters, that are of greater concern to health. The radon daughters are radioactive, ionized metal atoms which become attached to respirable dust particles and because they have a short half life (collectively about 30 minutes), radioactive decay is proceeding while the particles are resident in the lungs before they can be removed by normal lung clearance. Thus radiation, 95% attributable to α-radiation, is delivered to the tissues and is the cause of excess lung cancer among miners.

Observations in several mining communities, for example in the USA, Czechoslovakia, Canada, France, among uranium miners; workers in a niobium mine in Norway; iron ore miners in Sweden; tin miners in China and the UK and fluorspar miners in Newfoundland; have been reviewed by Archer(58). A significant dose related increase in lung cancer risk with exposure to radon and radon daughter elements has been evident and in miners who are cigarette smokers there is an interaction between the radiation exposure and the smoke exposure. Several facts emerge: the induction-latent period for lung cancer is longer when the exposure to radioactivity starts at a younger age; it is shortened by high exposure rates and by cigarette smoking; and lung cancers develop at lower levels of exposure to radioactivity in miners who smoke than in those who are non smokers. Lower levels of lung cancer among coal miners than in other underground workers is probably because coal mines are well ventilated to reduce fire and explosion risk and as a result there is no build up of radioactivity. In mines where silicosis was a problem, e.g. iron ore mines, ventilation to remove the silica also reduced the radioactivity, however, in some Swedish mines, because of freezing when outside air was used for ventilation, silica was reduced by circulating the air through old underground mine workings with the result that, in the 1920's, success in reducing the potential for silicosis was marred by an increase in lung cancer because radioactive materials were not filtered out, but this only became evident many years later.

The nature of the interaction between α-radiation and cigarette smoke is not clear. From one study(59) smoking and radiation were considered to have an additive effect and in another(60) the effect was considered to be multiplicative. From a long term study on Swedish iron ore miners(61) it was concluded that tobacco smoke acts as a tumour promoter, an effect that has been demonstrated in almost all animal studies. The histological types of lung cancer among miners have been different as the time elapsing between follow-up studies has changed. It has also been shown(58) that the age range of

the population under observation can influence the conclusion, thus only for the group aged 35-65 years will the smoking-radon relationship appear to be multiplicative.

The conclusion reached by the US Surgeon General(62) was that the smoke-radon interaction consists of two parts: an additive effect of the contribution of the two agents on the number of tumours produced and an accelerating effect due to tumour promoters in cigarette smoke. Thus for a miner who smokes, not only is the chance of lung cancer greater but the latent period is shorter and therefore the cancer appears sooner in smokers.

The possibility that α-radiation from radon daughters in the home or in other working situations where there are enclosed spaces with poor ventilation, or where tight energy conservation measures have been adopted, may present a highly elevated hazard to smokers is a matter for further investigation and since the level of risk will depend upon the mechanism of the interaction, further study of this would be useful.

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Exposure to other non-radioactive mineral dusts

Talc. Examination of talc samples has shown that true talc is only one of many mineral components of the powder sold as commercial talc. Asbestos-like minerals such as tremolite and anthophyllite may constitute major fractions(63), In a study of workers exposed to this type of talc, the only significant difference compared with non talc workers was in the number and severity of cases of dyspnea in the talc workers and smoking was considered to be an aggravating factor(63). The fibrous minerals in the talc were apparently less fibrogenic than chrysotile or amosite asbestos.

In a study of the effects of silica and talc on lung cancer among pottery workers: men exposed to high levels of silica dust with no talc exposure had a non significant mortality ratio of 1.37, however, those exposed to non-fibrous talc as well as to high levels of silica had a 2.5 times lung cancer risk which rose to 3.64 among those exposed for 15 or more years(64). The effect of smoking was not considered. As with other workers in dusty occupations it is likely that smoking prevalence was high.

The talc used in the rubber industry has been implicated in lung cancer: this has been referred to in the section on exposure to hazards in the rubber industry, page 40.

Alumina. A cross-sectional study of 788 employees of an aluminium production company examined the relationship of radiographic abnormalities to smoking and dust exposure during the mining and refining of bauxite to alumina(65). Chest radiographs showed a moderate trend of increasing prevalence of small opacities with increasing duration and high cumulative exposures in non smokers. Cigarette smokers had a significantly higher prevalence of opacities and their trends with duration were accentuated in comparison with non smokers. The stronger effects were attributed to the joint effects of duration of smoking and occupational exposure.

Dusts from many industrial operations, such as from metal grinding, borax, potash mining, working with corundum and other mineral dust exposures, have not been studied extensively, nor involving sufficient numbers of workers for the effects of smoking to be assessed. Silicates such as Kaolin, mica, vermiculite have been implicated in pneumoconiosis but smoking interactions have not been reported.

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Airborne biological dusts

Smokers show an increase in both the prevalence and severity of byssinosis and cigarette smoke increases the detrimental effect of cotton dust on ventilatory capacity. Cotton dust and cigarette smoke both cause bronchitis. Cigarette smoke and not cotton dust is the cause of emphysema. Smokers are more susceptible to occupational asthma from biological dusts than are non smokers.

Occupational exposure to biological dusts

Many dusts and aerosols of biological origin which can affect the health of exposed workers arise in agricultural operations and in industries such as textiles. The worldwide population that may be exposed is thus large and these exposures are of particular importance in developing countries where most workers, including whole families from young children to the elderly, are engaged in agricultural activities and small scale manufacture using vegetable products. Agricultural workers may be exposed to vegetable dusts, airborne fungal spores and microorganisms, animal danders and feathers, and herbicide and pesticide residues. Processing of agricultural products such as cotton, flax, hemp, grain, tobacco, paprika and tea and the milling of certain varieties of wood are occupations where vegetable dust exposures have been associated with detrimental health effects.

In addition to irritation and bronchitis that can be associated with almost all types of dust, there are four further types of effect of the inhalation of biological dusts. They are byssinosis, and responses which may be allergic, immunological or non-specific, and all are affected in different measure by smoking.

Byssinosis and other effects of textiles

The respiratory disease peculiar to textile workers, and recorded in several European countries in the 18th and 19th centuries, was given the name byssinosis. Many studies of the disease were carried out particularly during the present century in the Lancashire cotton mills of England. Byssinosis is a disease with characteristic symptoms of chest tightness and shortness of breath on returning to work after a period of absence and there is the possibility of permanent respiratory disability developing. It is more prevalent in the early stages of cotton processing, in the dusty fibre preparation jobs such as carding, than in weaving. Since the 1950's it has been established that the disease occurs in many other cotton processing countries such as Brazil, China, Egypt, India, Israel, the Netherlands, Sweden and Uganda. Byssinosis, or byssinosis-like symptoms, and bronchitis have also been found among flax, hemp, jute and sisal workers.

As with many industries that are widespread throughout the world, the smoking behaviour of the workers can only be considered in a general way: they usually conform to the norm for blue collar workers of a region. In a study of textile workers in South Carolina in 1973, the smoking prevalence was almost the same for workers as for controls(66). In another study of cotton workers in 1973(70), the percentage of male current smokers was 62.5% and of ex-smokers 16.4%; among females the figures were 33.9% and 6.1% respectively. Among flax scutchers in Normandy in 1986/7, 56% were smokers, and 18% were ex-smokers; compared with 45% and 15% respectively for the controls(67). In 31 Lancashire textile factories (1988) 47.5% of the 4656 workers interviewed were smokers(68). There were 5.9% of the 800,000 American workers surveyed(69) for smoking habits in 1982 who were exposed to textile fibres or dust: 28.5% of these were regular cigarette smokers and 44.9% were former cigarette smokers; compared with 23.5% and 43.5% respectively among the other occupations not exposed to textiles.

Increases in both byssinosis and bronchitis have been attributed to cotton dust exposure and to smoking in the cotton industry(70,71). From an industrial study of the biological effects of cotton dust and cigarette smoke(72) it was concluded that smokers show an increase in both the prevalence and severity of cotton dust induced byssinosis and that cigarette smoke also increases the detrimental effect of cotton dust on ventilatory capacity. It was suggested that the impairment of lung clearance mechanisms by cigarette smoke could be responsible for the deleterious effect of cotton dust and

also that smoking might lower the threshold of susceptibility to the effects of cotton dust inhalation. Additivity and equal importance of the effects of smoke and cotton dust have been suggested(66) but since they affect different lung function parameters it would seem that the two factors affect two different sites. The fact that workers who stopped smoking, whilst remaining in the same job, lost their byssinosis symptoms was significant. A recent survey, published 1988(68), of workers in 31 textile factories in Lancashire in the UK, where the conditions of work have been considerably improved, showed that byssinotic symptoms were related, in decreasing order, to years in the industry, degree of dust exposure, quality of cotton in use, ethnic origin and smoking habits. Symptoms of chronic bronchitis were found to be significantly related primarily to smoking habit and then to factors connected with the occupation. In a study of hemp workers(73), decline in ventilatory function was more pronounced in smokers. It was suggested(67) that a surprisingly low prevalence of byssinotic symptoms in 12 flax scutching mills in Normandy, may have been due to either self selection of the workforce, or an absence of the causative agent in the dust; persistent cough and phlegm production were, however, associated with tobacco use.

Estimates of the incidence and severity of textile related pulmonary disease vary widely. From a study of emphysema and other chronic lung disease in textile workers(74) it was concluded that there was no positive evidence to support a conclusion that textile workers, even after many years of exposure, have more emphysema or other chronic lung disease than the non-textile population and that the increase in emphysema between the years 1962-1969 and the period 1970-1980 is probably explained by an increase in smoking over the years prior to the study and corresponds to an increase in lung cancer during the period. It was suggested(75), however, that conclusions on the effect of byssinosis on mortality of textile workers from pulmonary disease should await a more comprehensive study of the subject, nevertheless, the importance of smoking as a primary causative factor had also been mentioned in another independent study(76). A similar conclusion was arrived at from ventilatory function tests over a three year period on 153 women (103 smokers, 50 non-smokers) with grades 2 and 3 byssinosis(77).

Cancer deaths in general and lung cancer in particular were, if anything, lower in workers exposed to cotton dust than in others(78).

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Exposure to dust in woodworking

> Woodworkers, particularly in the furniture industry have an elevated risk of cancer of the nasal cavity and sinuses; smokers are also at high risk.

The risk of developing cancer of the nasal cavity among workers manufacturing wooden furniture has been shown to be up to 1000 times that for the general population(79). The effect is worst in the more dusty areas(80). It is also associated more with certain hardwoods and the finishing of fine furniture than with all types of wood and may thus be allied to both the chemical and physical nature of the dust. In one study where the nasal cancer incidence was 100 times greater than for the general population, it did not appear to be affected by smoking habits. A similar conclusion was reached in a study in an area of Italy with a large number of cases of nasal cancer in woodworkers and leather workers(81). An association with smoking has, however, been established in more recent studies and current and past smoking habits have been shown to be a risk factor for developing squamous cell cancer of the sinus in men(82). A case control study of 121 male woodworkers seen for cancer of the nasal cavity or paranasal sinus in British Columbia in Canada between 1939 and 1977(83) showed increased relative risks associated with occupations involving exposure to wood (2.5) and with smoking (4.9) and, in a study in North Carolina and Virginia in the US, between 1970 and 1980(84), a major finding was the elevated risk of nasal cavity and sinus cancer among cigarette smokers. The interaction of wood dust and smoking needs further study, adenocarcinomata appear to be the type for which wood dust is mainly responsible whilst smoking is associated with squamous cell cancer.

The added effect of varnishes and solvents in the furniture industry is also an area for further study. Toluene diisocyanate, a component of polyurethane varnishes has been implicated in occupational asthma and more non smokers seem to have been affected by late responses but the interaction effects were not clear.

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Allergic response to vegetable dusts

This type of response can occur in the upper airways when it is manifest as Hay Fever, possibly in response to certain types of pollen, or it may be in the bronchi, as

asthma, or it may appear in both. Some of the dusts that cause allergic airways responses (occupational asthma) are grain dusts from various cereals and their products, wood dusts particularly from red cedar and iroko, teas and tobacco. Among asthmatics, environmental cigarette smoke makes the effect of the asthma worse(85) and smoking effects appear to be additive to that of asthma(86). Grain dust exposure and smoking have been found to cause increases in the prevalence of respiratory symptoms and reductions in pulmonary function of grain elevator workers(87). The effect of smoking appeared to be slightly more pronounced. The combined effect of grain dust and smoking on lung function appeared to be additive except in the least exposed workers (5 years or less) where a synergistic effect was observed in tests on peripheral airways dysfunction.

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Other diseases caused by biological agents

Allergic alveolitis

> Allergic alveolitis appears to be less prevalent in smokers than in non smokers but it is likely that this is due to a reduction in alveolar macrophage activity caused by smoke rather than to a beneficial effect.
> Schistosomiasis induced bladder cancer, a widespread occupational disease among agricultural workers in many developing countries, appears to be enhanced by smoking.

Although many biological dusts are known to have detrimental effects on workers, there have been few studies on the added effect of smoking. Allergic alveolitis: there are several forms of this disease, of which farmer's lung, bagasse pneumonitis, and bird fancier's lung are examples, caused by fungal spores in mouldy hay or mouldy sugar cane or an agent in bird feathers respectively, and it is immunologically mediated(88). It seems to be more prevalent in non smokers than in smokers, possibly because smoking may depress alveolar macrophage activity. In contrast, smokers are more likely to show higher specific antibody production and correspondingly be more susceptible to occupational asthma(88). As well as among grain, tea, coffee and rice handlers, symptoms typical of occupational asthma occur in enzyme detergent workers, amongst whom it has been found that twice as many smokers as non smokers exhibit asthmatic symptoms(88).

less intense. In the latest study(97) of a cohort of 3916 Swedish copper smelter workers, the interaction between arsenic and smoking for the risk of developing lung cancer was intermediate between additive and multiplicative and appeared to be less pronounced among heavy smokers.

From studies of the effect of arsenic trioxide in smelter workers, of poisoning by arsine, and of arsenic contamination of drinking water and beer (reviewed 92), there appears to be a causal relationship between CVD and arsenic compounds and a dose response has been demonstrated for arsenic and cardiovascular mortality. The known effects of smoking on CVD could be expected to contribute additively to any effects from workplace exposure to arsenic compounds, the possibility of a multiplicative effect has not been examined.

Cadmium occurs in zinc and lead-zinc ores and is a hazard of the smelting of these and several other metals, as well as of the production of cadmium itself and its compounds. It is encountered in electroplating, alkaline batteries, pigment manufacture and use, and is a hazard of welding. The principle route of entry to the body is through the lungs but ingestion is also possible. The tobacco plant absorbs unusually large amounts of cadmium from the soil and during burning a high proportion of this is transferred to the mainstream smoke. Various organs can be affected by long term exposure to cadmium but the most noticeable effect is impairment of the renal function giving rise to proteinuria and general ill health. Cadmium has also been associated with various types of lung disturbance (emphysema, obstructive pulmonary disease and diffuse fibrosis) and with lung cancer. Effects of cadmium on CVD and hypertension have been suggested but any causal relationship has been questioned in a review of epidemiological studies(92). The effects of smoking on any cadmium related diseases is not known but blood and urine cadmium levels have been shown to be higher in smokers than in non smokers and the levels were found to be considerably elevated in smokers working in an alkaline battery factory(98) and in smelter workers who were also smokers(99,100). Although blood or urine levels of cadmium are not good measures of the body burden of cadmium, it would nevertheless seem that the risk of developing cadmium related disease must be higher in cadmium exposed smokers than in their non smoking coworkers.

Chromium and its compounds are encountered in the metalurgical, chemical, elecroplating and leather tanning industries and workers producing ferrochrome alloys or welding stainless steel, involved in the production or use of chromium salts and pigments, or working in the leather industry are liable to suffer occupational exposure.

The principle route of entry to the body is through the lungs. The deposition of chromates on mucous membranes may cause ulceration which, in the nasal septum, can lead to perforation; an asthmatic reaction has been reported in workers exposed to chromic acid; and chromium and some of its salts, particularly hexavalent chromium salts, have been associated with lung cancer both in experimental animals and in epidemiological studies. After an evaluation of all the available data(101) it was considered that "there is sufficient evidence in humans for the carcinogenicity of hexavalent chromium compounds as encountered in chromate production, chromate pigment production and chromium plating industries". There have not been any systematic studies of the combined effects of smoking and hexavalent chromium inhalation but several small scale observations have been made(102-105) which suggest that workers with chromium compounds who are also smokers may be at greater risk than non-smokers in the industry.

Cobalt. There have been tentative suggestions that cobalt exposure and cardiomyopathies may have a causal link and if this were the case, smoking could be expected to contribute additively. However, further studies of the relationship between cobalt exposure and CVD are needed, particularly in view of the fact that this metal and its compound are in widespread use.

Lead. Levels of lead in blood vary from one area to another, between urban and rural populations and between men and women. Higher blood lead and erythrocyte protoporphyrin levels have been demonstrated in heavy smokers exposed to lead (106,107): these could have been partly due to contaminated cigarettes acting as vectors. Other studies have shown little difference between smokers and non smokers in blood lead levels. Epidemiological studies of the effect of lead on CVD have been reviewed(92) and there may be a causal relationship. The effect of smoking on any lead related CVD would represent an added burden.

Manganese is encountered by miners, workers in the ferromanganese and iron and steel industries, and by those involved in the production of dry cell batteries and in the manufacture and use of welding rods. The principal route of entry to the body is through the lungs but because most of the compounds are insoluble, only the smallest particles, capable of reaching the alveoli and being phagocytosed, are absorbed. Long term exposure to manganese causes damage to the central nervous system and impaired mental capacity, as well as lung damage resulting in an increased incidence of pneumonia and a higher rate of acute and chronic bronchitis. Data from a study of workers employed in the production of manganese alloys suggested that manganese may contribute to the development of chronic lung disease. Individuals with a history of smoking appeared to be more affected than non smokers and the relationship between the the degree of smoking and the prevalence of respiratory tract symptoms in the manganese exposed workers suggested that smoking may act synergistically with manganese(108). In an epidemiological study of workers

producing manganese salts and oxide(109), additive effects for smoking and manganese exposure were found. There has been an indication of increased spontaneous abortions and stillbirths among the wives of manganese workers: since abortions and stillbirths are also increased in women who smoke, it is reasonable to assume that the wives of manganese workers who smoke are at added risk.

The interaction of cigarette smoke and manganese compounds should be a subject for further study, particularly in view of the fact that organic manganese compound may become the cause of significant atmospheric contamination in the future as they find more widespread use as petroleum additives.

Nickel. The excess risk of cancer among workers in nickel refineries has been well documented(101). It is also known that nickel is taken up by tobacco plants and up to 20% of this is transferred to the mainstream smoke, probably in the form of nickel carbonyl. A study in the early 1980's in Norway demonstrated(110,111) that the numbers of nasal and lung cancers in nickel refinery workers were far higher than the numbers expected and when smoking habits were taken into account it was found that there was a response which was closer to being additive than multiplicative(111). The main areas of industry in which the nickel hazard occurs are in the roasting of the ore, smelting and electrolysis. Histological examination of nasal biopsy specimens, for early signs of carcinogenesis, from 59 retired nickel workers, 21 of whom were smokers and snuff dippers, showed a higher score for smokers than for non smokers and 4 of the subjects with nasal carcinoma were smokers(112).

Metal welding

Welding is fundamentally the joining of materials by fusion. From time immemorial, metals have been welded by hammering hot pieces together. During the past 100 years, several methods of welding have been developed which basically join together pieces of metal by local melting. This may be achieved by a hot flame (oxy-acetylene) or by an electric arc struck between the surfaces to be joined and another piece of metal which forms the electrode and which melts into the joint. Materials may be included to act as a flux and the molten area may be bathed in an inert gas to reduce oxidation at the joint. Oxides of many metals, including those considered in this review, along with other inorganic compounds, such as the oxides of nitrogen, carbon monoxide, ozone, carbonyl chloride, and organic compounds such as formaldehyde, acrolein, benzene, are encountered in the "fume" that arises from the different types of welding. Flashing of ultra violet light; dust, containing chromium, nickel and silica, from sandblasting and grinding; and asbestos are also hazards of welding. Metal fume fever is a common illness of welders and may give rise to siderosis. Silicosis and asbestosis have been found in

welders and chromium sensitivity and nasal septum erosion have been reported in stainless steel welders. Among welders and shipyard workers (many of whom are welders), the percentage of never smokers in a survey of 800 000 American men and women in relation to their occupation were 18.8% and 16.5% respectively(13), putting welders among the groups with the heaviest smoking prevalence.

A study of lung cancer deaths among 3247 shipyard workers(113) showed an excess of the disease of 32% relative to the general population and, when only the years at risk were considered, the excess risk was 74%. Most of the welders were also smokers and smoking was a controlled variable in this study, but from the excess lung cancer observed the subject is clearly one for further study. An increased incidence of lung and bladder cancer among welders in a Norwegian shipyard(114) was found but smoking was mentioned only as a confounder. From a case control study(115) of lung cancer in welders it was concluded that an excess of lung cancer was contributed to by a higher frequency of smoking and probably exposure to asbestos in the shipyards. In another study(116), it was concluded that arc welding fume and gases were not related to bronchopulmonary disease: in older welders smoking and age were the significant factors, however, this study was carried out in a well ventilated engineering workshop and not in the closely confined spaces often encountered by welders in shipbuilding and other heavy engineering work. From an assessment of the relationship between welding fume and respiratory symptoms(117), it was concluded that all welders show increased prevalence of respiratory symptoms relative to controls but chronic bronchitis was found to be more prevalent in smokers (37.9%) than in non smoking welders (12.5%). The post shift concentration of chromium in the urine of stainless steel welders has been found to be increased(118) and the increase was slightly greater in the case of smokers.

Traumatic vascular disease has been reported in welders; arising from repeated prolonged occupational hand trauma. The effect of smoking on peripheral vascular disease is likely to be a contributary factor to this condition and management of the disease includes abstinence from tobacco.

It is reasonable to expect an interaction between cigarette smoke and some or all of the many components of welding fume and and the several disease conditions with which they have been associated.

Sulphur dioxide is a pungent and suffocating, water soluble gas, well known as an upper respiratory irritant and a causal agent of bronchiolitis(119). It is a by-product of coal and oil fired burners and a workplace pollutant in many industries, such as textile dyeing and finishing, smelting of copper, lead and zinc; wood-pulping; and the paper industry. It occurs in cigarette smoke, but only as one of a very large number of

respiratory irritants. In a Swiss study(120), bronchitis was found to be more frequent in sulphur dioxide polluted air, but smoking was shown to be a dominant factor. In a study of the effect of sulphur dioxide in and around a pulp factory in Sweden(121) the effects of exposure to the gas and to cigarette smoking were found to be synergistic.

Compounds such as carbon monoxide, hydrogen cyanide, some of the oxides of nitrogen, and ammonia occur in both the workplace and tobacco smoke. See section 3.2, page 47.

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Occupational exposure to organic chemical compounds

In a large number of very diverse industries, many organic compounds are encountered which have properties covering a wide spectrum of both molecular structure and biological activity. Some may affect lung clearance, or the cardiovascular system, or tissues in the lungs, liver, kidneys, blood, bone, the central nervous system, or processes such as reproduction and cell division, and many may be tumour initiating or promoting. Cigarette smoke also contains a vast array of organic compounds and many of these can likewise affect these several biological tissues and processes. Exposures in industry and exposure due to smoking differ in that in the majority of situations met with in industry, the body usually encounters only one or perhaps a limited group of these compounds whereas cigarette smoke delivers the full chemical storehouse in each of a series of short sharp insults. However, in spite of the large number of biologically active organic compounds encountered in many industries and the prevalence of smoking among workers, it is surprising how little work has been published on the effects on health of the interaction of industrial organic pollutants and tobacco smoke. Nevertheless, the effects of a few specific organic compounds, or of some that are encountered in specific industries, have been studied and, from the known effects of smoking, it is possible to see some of the likely effects of adding together the effects of the two hazards and to realize the potential for interaction.

In considering organic chemical compounds, there are some that occur in industry that have not been detected in cigarette smoke and the effect of smoking is to change the risk of occupational exposure (section 3.1). Many of them occur both in tobacco smoke and the workplace and the effect of smoking is one of augmentation of dose (section 3.2), although modification of effect can also occur in this situation as well. Some are added to smoke when workplace materials that have contaminated smoking materials are burnt or volatilized (section 3.3). Some organic compounds may fall into all three categories.

Exposure to dyestuffs

Bladder cancer has been associated with smoking, and with exposure to certain arylamines which occur in dyestuffs, traces of which could contaminate dyes and other materials used in occupations employing other organic compounds. Many similar compounds are also found in tobacco smoke. There is an increased risk of bladder cancer for workers who are also smokers in these industries.

There is a well established relationship between bladder cancer and exposure to certain aromatic amines encountered in the dyestuffs industry, e.g. benzidine and β-naphthylamine. In addition, although there have been a small number of conflicting reports, most of the currently available evidence clearly shows a relationship between bladder cancer and smoking. Some recent studies have considered the interaction of the two hazards. From an analysis of 991 cases(122), a significant risk of bladder cancer was associated with cigarette smoking and a dose response relationship, based on years of employment, was found for dye manufacturing workers. The two risks were considered to be additive. Overall there was a significant risk of bladder cancer associated with cigarette smoking, a risk ratio of 1.8 for males, and there were significant overall risks associated with occupations such as those of process workers in the dye manufacturing industry who had a risk of 2.9 for males. When smokers were contrasted with non smokers, among dye manufacturing process workers who were also smokers the risk was 4.6, while for non smokers the risk was 1.9. In a later report(123), part of the same case control study, it was concluded that arylamines in the dyestuffs industry pose the major threat of bladder cancer: cigarette smoking remained another risk factor. However, from another survey in an area of Spain where 44% of the adult population work in dyeing and printing textile fabrics(124), it was clear that there was an increased risk of bladder cancer for habitual smokers. RR for smokers 2.3, for occupational exposure 5.5 and for smoking and occupational exposure 11.7. Because of the large number of amines in cigarette smoke, an additive effect for dyes and tobacco smoke could be expected, however, the observed effect in this case was multiplicative.

In a more recent study among men in Spain(125), an increased risk of bladder cancer was found for textile workers (OR 1.97), mechanics and maintenance workers (OR 1.86), and workers in the printing industry (OR 2.06). The highest risk was among those first employed in the textile industry before the age of 25 and prior to 1960. Among mechanics the highest risk was for those who started after the age of 25 and later than 1960. The OR for smokers who had also been employed in one of the high risk occupations was 7.82 which is compatible with a multiplicative affect of joint exposure to tobacco and occupational hazards. Additivity of risks was found for the interaction between tobacco smoke and several occupations associated with bladder cancer in a study in Italy(133),

however the occupations were not specified. The bladder cancer risk associated with smoking black tobacco was also considered.

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Exposure hazards in the rubber industry

There are many airborne hazards in the rubber industry. They occur as dusts, fumes, aerosols, vapours and gases, and a high risk of pulmonary disability has been reported, particularly from emphysema. Excess mortality for cancers of various sites has been reported. There is no information on smoking prevalence in the industry, nor of the interaction of smoking with the industrial hazards. Some interactions will be the same as those in other industries, e.g. asbestos, dust, solvent vapour, fumes and the effect of particulates on tissues already inflamed by small airways disease caused by smoking will be the same.

Some of the principal airborne hazards in this industry are fumes, talc, carbon black, chemical additives and organic solvents but the components of the hazard differ between the several types of occupation within the industry. A high risk of pulmonary disability has been reported(126) and there was an elevated risk for workers who were smokers and who were employed in areas where exposures to respirable particulates and/or to solvents could be demonstrated. The data obtained suggested an interactive effect between smoking and occupational exposure for workers involved in mixing, (exposed to talc, accelerators, cementing materials and other particulates); in extrusion, (exposed to solvent sprays and mold release agents); and in curing, (exposed to solvents and rubber reaction products). A problem in surveying the rubber industry arises because of job migration by some workers within the industry, thus another high risk group of workers who were also smokers was involved in finishing and inspection (exposed to solvents and rubber dust) but they tend to be older employees who have had experience in other sections before moving to this particular job. Throughout the industry, emphysema was the principal pulmonary condition requiring premature termination of employment due to disability in rubber workers(126). The conclusion of this study was that there was a need not only to identify the specific hazardous agents but also to initiate an energetic and committed programme aimed at eliminating smoking in this occupational group.

Excess mortality from cancers of various sites, the site usually being associated with specific areas of work and types of exposure within the industry, has also been reported(127-131). Lung cancer has been associated with curing and inner tube

manufacture(128-130,132) but the agents responsible are not known. Curing involves the use of solvent based sprays and, during the curing process, gases and vapours, undefined in composition, are produced. The use of talc, and hence a possible exposure to asbestos type fibrous minerals, has been associated with pulmonary disease(63) and with a significant 2.5 times excess risk of lung cancer(64) in industries not associated with rubber manufacture, as well as in the rubber industry(132) where the relative risk of lung cancer for talc exposed workers was 3.2 for men and 4.4, for women. In the sections where the very high lung cancer was found, however, the levels of smoking were also very high and the possibility of an effect due to interaction of the two exposures could not be assessed.

Gastrointestinal cancer, bladder cancer and leukaemia have been associated with different jobs within the industry(127) and the possibility of causal relationships arising from exposure to carbon black, plasticizers, antioxidants, arylamino compounds and the use of benzene as a solvent have been suggested. The role of smoking, whether causative, or additive, or interactive was, however, not considered.

There is no information on the prevalence of smoking among workers in the rubber manufacturing industry: it is to be assumed that their smoking characteristics are similar to those found for other blue collar workers.

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Exposure to pesticides

The use of pesticides occupies millions of people throughout the world. Arsenical pesticides are probably carcinogenic and their effect could be synergised by smoking, as has been found for arsenic in manufacturing industry. Many organic pesticides have harmful effects on physiological processes which are also affected by smoking; thus an elevated combined effect could be expected. Chromosomal aberrations caused by many pesticides are greater in smokers than in non smokers.

Fungicides, insecticides, herbicides and all other agents that reduce the detrimental effects of life-forms that may harm agricultural production were historically derived from inorganic elements such as copper, lead, arsenic, sulphur and compounds such as lime and chlorates. During the past 30 years, however, many of these materials have been replaced by complex organic compounds and the number and variety of these, with

their multitude of trade variants, is enormous. The number of workers exposed during manufacture is relatively small but the worldwide population at risk as users is very large; in agriculture, viticulture, fruit production, cotton farming and even in growing tobacco. In spite of the size of the industry involved in the production of pesticides and the enormous amount in use throughout the world, as well as the very large population which could be at risk from any untoward effects, little work has been published on the effects on health arising from the interaction of pesticide exposure and smoking.

"Vineyard sprayers lung" has been described as an occupational disease and a study was made of the cytologic changes of the respiratory tract in vineyard workers spraying copper sulphate based Bordeaux Mixture(134). The macrophage cytoplasm of control subjects contained no copper whereas copper was detected in 64% of the vineyard sprayers: a fact which in itself shows the extent to which fungicidal sprays can be inhaled. Abnormal cytological changes were found in smokers in both the sprayers and controls and atypical squamous metaplasia were observed in 29% of smokers who were vineyard workers. Studies of the effect of arsenical pesticides have shown mixed results which is unexpected in view of the effects that have been shown to be attributable to industrial exposure to arsenic and the interaction of this with smoking.

Clearly the complex organic pesticides in use today in the form of dusts and dispersible powders, and as emulsifiable concentrates and solutions for spraying, are likely to be inhaled during use. In the case of dusts and powders there will be diseases attributable to the carrier dusts per se independent of the pesticides they are carrying; primarily bronchitis and SAD, and in smokers there will be the usual overriding effect of smoke induced bronchitis along with the effects of dust on smoke-inflamed small airways. The effects of many pesticides are associated with enzyme inhibition and effects on the respiratory-, central nervous- and cardiovascular- systems, and carcinogenic properties have been ascribed to some, but the extent of any effects in exposed workers who are also smokers has not been studied extensively and little is known of any interactive effect of smoking. There have been many studies carried out to investigate the mutagenic potential of various well known pesticides and in some the effect of smoking has been considered and shown to increase chromosomal aberrations. In a recent study, a 2- to 3-fold increase in chromosomal aberrations was noted in smokers exposed to pesticides when compared to non smokers exposed to pesticides working in the same fields and a high prevalence of aberrations has been recorded in another group of smoking workers who used pesticides in cotton fields(135). Other similar studies were listed(135).

The following are some typical types of compounds in use as pesticides:

- Several organophosphorus compounds, such as malathion, methyl parathion and parathion, are used as insecticides, as are lindane and carbaryl.

- Sulphur containing organic compounds such as chloroallyl thiocarbamates and dithiocarbamates, are herbicides, as are chloromethylphenoxyacetic acid (MCPA) and trichlorophenoxyacetic acid (245T)
- Sulphur containing and phenolic compounds have found use as fungicides
- Naphthylurea is a rodenticide
- Maleic hydrazide is a growth retardant which has been widely use by tobacco producers in sucker control.

Smoking materials could act as vectors for all these compounds (see Section 3.3).

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Exposure to chloromethyl ethers

When inhaled, chloromethyl methyl ether and bis(chloromethyl) ether are lung carcinogens. Their effect is modified by smoking, the total effect being less than additive.

Chloromethyl methyl ether (CMME) and its contaminant bis(chloromethyl) ether (BCME) are carcinogenic when inhaled, BCME far more so than CMME, and a groups of chemical workers who had been exposed to these compounds were studied from 1963 to 1972(136-139). The result show a modification of the effect in that there was a higher rate ratio for non smokers than for smokers and the effect for smokers was less than additive. The study was however, carried out on a very small number of subjects: only 11 lung cancer deaths occurred in total. In reviewing these findings(42) several explanations were quoted, such as protection by a greater mucus barrier in smokers, or less access to tissues due to constriction of small airways in smokers. However, if this protection is available for the chloromethyl ethers, it should also be available as protection against other carcinogens such as asbestos, but it appears not to be.

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Exposures in the petroleum industry

> Smoking prevalence among petroleum workers has been found to be similar to that in the general public. An association between renal cell cancer and the industry, with an interaction between long term occupation and smoking has been found. Bladder cancer has also been associated with the industry and with cigarette smoking.

Worldwide, some 500 000 people are employed in petroleum refining and may be exposed to a large number of substances that occur in crude oil, or in production processes, or as intermediates, catalysts and additives, or in the final products. Many of the compounds found in this industry also occur in cigarette smoke but there are also many more that are peculiar to the industry. Because of fire risk, there are sections of the industry where smoking will not be permissible during working hours but in a study of 10 923 male and 624 female employees of the Australian petroleum industry between 1981 and 1984 it was found that this group did not differ substantially from the general population in its smoking habits(141). There have been suggestions of an increased risk of renal cancer. From a study of 92 men with histologically confirmed renal cell carcinoma(140) it was concluded that there is an interaction between long term gasoline exposure and heavy smoking. This form of cancer among petrochemical workers has been found in other studies but without any relationship to smoking being considered. An association between bladder cancer and oil refinery workers in Argentina was found, as was an elevated risk of lung cancer in smokers but the numbers were too few for an evaluation of the interaction(142). However, since the two risk factors have been demonstrated separately, it is likely that together they will produce an increased risk.

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Exposure to polycyclic aromatic hydrocarbons

> Exposure to tar volatiles, in which the main carcinogens are PArH, causes bladder cancer and cancer of the lung and a significantly elevated alveolar macrophage count. In all cases, a synergistic interaction with cigarette smoking was found which appeared to be multiplicative.

Tobacco smoke has been shown to contain many polycyclic aromatic hydrocarbons(PArH), among them several that have known carcinogenicity. There are also several industries in

which these compounds occur and represent a prime hazard as lung and bladder carcinogens. Clearly a smoker in any of these industries will obtain a dose of PArH both from tobacco smoke and from the industrial source. Since, in many industries, interaction of smoking with occupational exposure is a possibility, the subject will be considered here rather than in section 3.2. PArH in the workplace are, furthermore, often accompanied by other hazardous materials, particularly irritants.

PArHs occur in coal gas manufacture, coking oven fumes, aluminium refining, in the use of tar and asphalt, in oil refining and the exhaust from internal combustion engines and are usually present along with irritant fumes or aerosols. There is a lack of smoking data for workers in these industries, but it can be assumed that the smoking prevalence is at least as high as the average for blue collar workers. It is known that 69% of workers at one Norwegian smelter smoked when the expected prevalence of smoking was 52%(146) and, although not specific to these industries, the percentage of smokers and ex smokers among workers exposed to chemicals and coal tar pitch in a survey of 800,000 American men and women in 1982 in relation to their occupation(13) was 49.9% against 46.1% for the average worker.

It was concluded from a Canadian study that the incidence of bladder cancer is unusually high in aluminium smelter workers, particularly among those employed in Soderberg potrooms(144). The metal is extracted by an electrolytic process, involving the use of a mixture of tar and pitch which creates air pollution by tar volatiles including PArH which, measured as benzo(a)pyrene, can reach a concentration of 800 $\mu g/m^3$/8hours(143). The PArH were considered to be the factor causing bladder cancer. There was a synergistic effect when cigarette smoking and benzo(a)pyrene exposure were combined(144), but whether the interaction was additive or multiplicative could not be assessed on the numbers studied. In a more extensive study in which the former data were augmented(145), the tar volatiles were confirmed as the cause of bladder cancer and the results suggested that exposure to tar volatiles and cigarette smoke combined multiplicatively. Workers in coke oven plants have a higher incidence of lung cancer than the general population and a measurable amount of PArH in urine, which is higher for smokers(147). Professional drivers are exposed to PArH through the exhaust of petrol and particularly diesel engines and an excess of lung cancer has been found in this occupational group, with a suggestion of a synergistic effect between smoking and occupational exposure(148).

The function of alveolar macrophages is to remove inhaled foreign material from the alveoli and respiratory bronchioles and their numbers increase when the lungs are exposed to particles and gases. Furthermore, it has been demonstrated that the macrophage count after lung lavage was higher from persons exposed to cigarette smoke than from non

exposed persons. It has been shown that the alveolar macrophage count for workers in the potrooms of an aluminium reduction plant is elevated and that for workers who were also smokers the count was higher again. From the results it was concluded that cigarette smoke and workplace pollution act synergistically in increasing the number of alveolar macrophages(149).

In most of the workplace conditions in which PArH contaminate the atmosphere, the gases, fumes and aerosols also contain other materials which act as irritants, and may play a role in the aetiology of chronic obstructive lung disease. The situation in aluminium smelting has been reviewed(146) and the role of smoking was considered to be important and an aspect requiring specific attention in future research. It was also considered important to consider smoking in epidemiological surveys after a study had been made of lung cancer mortality rates and smoking patterns in workers in the motor vehicle industry in which proportionate mortality rates were considerably reduced when smoking rates were taken into account.

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Exposure to carbon disulphide

Carbon disulphide is a volatile liquid which in technical grades has an unpleasant smell due to contaminants. It occurs in the petroleum and coking industries and some sections of the chemical industry but occupational exposure is mainly confined to the viscose rayon industry where it occurs along with hydrogen sulphide. Long term exposure to carbon disulphide, at concentrations as low as 10 to 20 mg/cubic metre, can affect the nervous system leading to behavioural changes and peripheral nerve dysfunction. An excess of coronary heart disease is also a well known effect. Cigarette smoke can contain from 18 to 42 μg per cigarette in mainstream smoke. Clearly, a smoker exposed to workplace carbon disulphide is subjected to doses from both sources and hence at increased risk of disease.

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3.2. Chemical compounds present in both tobacco smoke and the workplace

Some chemical compounds often found in the work environment and associated with health risks also occur in cigarette smoke.

Formaldehyde	gas	Hide preservation, textile printing foundries, hospitals & laboratories embalming.	Severe irritation allergy, cancer.
Aliphatic aldehydes & ketones	vapours liquids	antioxidants, explosives, mirror silvering, adhesives, celluloid, lacquer & varnishes, paint spraying	eye, skin, upper respiratory irritants
Acrolein	vapour	Acrylates, plastics, rubber, fire fighting, refrigeration, textile resins, foundries, welding.	intense irritant to eyes, mucous membranes.
Aromatic hydrocarbons	vapours aerosol	Aluminium smelting, petroleum,coking gas & tar industries, exhaust gases	lung and bladder cancers.
Aromatic amines	aerosol	Dying and dyestuffs	Bladder cancer.
Benzene	vapour	Furniture finishing, petrochemicals, rubber industry, adhesives, solvents	Irritation, blood changes, anaemia leukaemia, mutagenicity.
Hydrogen cyanide	gas	Blast furnaces, coke ovens, gas industry, electroplating, silver extraction.	irritant, asphyxiation, enzyme deactivation.
Ammonia	gas	Chemical industry, dying, electro-plating, fertilizer manufacture, paper & paper pulp industries, leather tanning, water treatment.	irritant,nausea, bronchitis, pulmonary oedema
Nitrogen oxides	gases	Fertilizer manufacture, welding, chemical industries, metal cleaning. necrosis,oedema, bronchiolitis.	irritation, bron -chial corrosion
Volatile compounds of As, Cd, Cr, Mn, Ni.	fume & aerosol	Smelting, electroplating, welding battery manufacture, metal recovery pesticides	respiratory irritants, dyspnea emphysema, cancer fibrosis, allergy, CVD, neurotoxicity

These compounds represent only a very small number of the harmful constituents of tobacco smoke

Another important constituent is carbon monoxide, a well known asphyxiant gas which reduces the oxygen carrying capacity of the blood by combining with haemoglobin. The mainstream smoke from a cigarette can contain up to 23mg of carbon monoxide. In some occupations, workers are subjected to significant concentrations of carbon monoxide and the smoker in these situations, therefore, receives a considerably elevated dose of the gas and is thus at a greater risk of suffering from the associated partial chemical asphyxiation, and from cardiovascular and neurotoxic effects. Typical occupations in this category are policemen on points duty and garage workers, as well as firefighters, and workers in gas and coke production and in foundries.

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3.3. Smoking materials acting as vectors

> Materials used in the workplace, which produce harmful chemical agents when they are burnt or vaporized, can be transferred from the workplace onto cigarettes or other smoking materials and cause the smoke to be far more injurious when the tobacco is smoked.

Polytetrafluoroethylene (Teflon) is used in coatings for cooking ware, for making chemical vessels, gaskets and bearings and in sprays as a mold release agent. This polymer, and polyvinyl fluoride, are inert materials but their thermal decomposition products can be very biologically reactive. Cigarettes are easily contaminated in the workplace and when smoked, the polymer burns giving fumes which cause polymer-fume fever: severe gripping chest pain causing difficulty breathing, trembling and shaking, elevated temperature and severe diaphoresis. The symptoms pass after a day or two, but recur on again smoking a contaminated cigarette. Before the cause was recognized a case was recorded of a person, who used the polymer in a mold release spray, having some 40 attacks(150). Another case was a person who referred to the disease as "mold machine pneumonia"(150) and other cases have been reported more recently(151). In the early cases there appeared to be no long term effect, but in the first case mentioned above, after some 18 months of wellbeing after the 40 attacks, the subject began to complain of shortness of breath. Post mortem examination of the lungs, after death from an unrelated condition, revealed interstitial pulmonary fibrosis. It was suggested that polymer-fume fever should be regarded as a potentially serious disorder(151).

Fluorocarbons frequently occur as volatile vapours in aerosols, and refrigerants, but some occur as liquid solvents and in plastics and when burnt produce hydrofluoric acid which is an intense irritant.

Methylparathion one of a group of organophosphorus compounds used as pesticides which are rapidly absorbed into the body, particularly through inhalation, and which cause a wide range of unpleasant and debilitating symptoms at low concentrations and severe illness at higher concentrations.

Dinitro-o-cresol is another compound used in agriculture as a herbicide and pesticide, as well as in the dyestuffs industry. It can enter the body by inhalation and causes several conditions from intoxication, excessive sweating, thirst, fatigue, and fever which may lead to rapid deterioration and death.

Mercury. Inorganic mercury occurs in many industries, as elemental mercury in scientific and electrical instruments, as amalgams with many other metals, in paints and pigments and in the chemical industry, as well as in mining and extraction of the metal. Organic mercury compounds are used as disinfectants, fungicides, herbicides, bactericides and antiseptics. Contamination of smoking materials can lead to the inhalation of mercury which leads to bronchitis, bronchiolitis and pneumonitis. A wide range of conditions can arise from long term exposure at low levels.

Cadmium has already been referred to earlier, p. 34. It can be a hazard due to its presence in the workplace; it is present in tobacco and is transferred to mainstream smoke and it could enter the body as a result of smoking materials acting as vectors.

Chlorinated hydrocarbons abound throughout many industries where they are used for cleaning, degreasing, destaining, in leather finishing, as solvents, in paints and resins, in printing, tar and wax making and wool scouring, to name but a few. The majority of these when burnt form carbonyl chloride, also known as phosgene, which causes upper respiratory tract irritation and in larger doses dyspnea and respiratory failure. Smoking materials could act as vectors.

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3.4. Exposure to Vibration and Noise

The vibration of many hand-held tools causes Raynaud's phenomenon (vibration induced white finger syndrome). The effect is far worse in smokers than in non smokers. Noise-induced hearing loss is also more pronounced in smokers than in non smokers.

Smoking has been causally related to cardiovascular, cerebrovascular and peripheral vascular diseases; it is, therefore, not unexpected that related occupational disabilities associated with these diseases are affected by smoking.

Raynaud's phenomenon (also called vibration white finger syndrome) was first associated with the use of vibrating tools in Italian miners in 1911 and the association has since been reported for a wide range of hand-held vibrating tools such as impact hammers, chipping hammers, grinders, riveters and the chain saws used by woodworkers. An American survey estimated that 1.2 million workers were exposed to hand-arm vibration. Damage to digital arteries and narrowing of the lumen has been associated with vibration syndrome(153) and because nicotine act as a vasoconstrictor it has been suggested(154) that limiting smoking could aid blood flow to the extremeties and thus reduce the condition. In a survey of forestry workers in Quebec in 1977-1978(155), among 1540 woodcutters, a prevalence of Raynaud's phenomenon was found in 30.5% of chainsaw users and there was a strong association between this and cigarette smoking, the relative risks were 3.50 for non smokers and 6.55 for smokers: corresponding to an additive effect for the two risk factors. From another study of the effect of tobacco use on a cohort of men with the disease, in which the extent of tobacco use was confirmed by blood cotinine measurements(156), it was shown that tobacco aggravates the symptoms of the disease, and that patients with advanced symptoms were found to use tobacco more frequently and to have higher blood cotinine levels than patients with less advanced disease.

Noise is also caused by vibration, usually at a slightly shorter wave length. In a study of aviators carried out at the US Naval Aerospace Medical Research Laboratory in 1963(157), two hearing level groups were identified, one with normal and the other with impaired hearing. The impaired hearing group had smoked more cigarettes for a longer period of time than had those in the normal group. The relationship between cigarette smoking and hearing loss was studied among 2348 noise-exposed workers at an aerospace company and it was found that smoking was clearly a risk factor in noise-induced hearing loss: odds ratio = 1.27 for ever smokers and odds ratio = 1.39 for present smokers, compared with non smokers(158). Vascular insufficiency of the cochlear organ has been cited as the predominant cause of progressive hearing loss that occurs with age and it was suggested that smoking reduces the blood supply by 1) vasospasm induced by nicotine,

2) atherosclerotic narrowing of vessels, and 3) thrombotic occlusions(159). In a study of 1000 candidates at a Veterans Hospital(159) it was found that whilst age and sex were the most important variables, at all measured frequencies the percentage of loss was greater for smokers, the differences being greater at higher frequencies.

Opinions differ on the influence of occupation and hand injury on Dupuytrens contracture(175) but, although not specifically caused by occupational activities, Dupuytren's disease has been statistically linked to cigarette smoking which may be involved in the pathogenesis of the disease by producing microvascular occlusion and subsequent fibrosis and contracture. Cigarette smoking is one of the most significant factors in the development of peripheral vasculopathy(176).

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4. Smoke as an Atmospheric Pollutant

The most unfortunate smoker is the one who participates in passive smoking - involuntary smoking - compulsory smoking. Whatever name is chosen, it describes the inhalation of tobacco smoke from an atmosphere polluted by smokers and frequently neither an alternative nor an escape is possible. In many developed countries, environmental tobacco smoke at work is probably a more frequent atmospheric pollutant than all the other workplace hazards put together. Furthermore, any worker who may be exposed to an industrial hazard has a still greater health risk if the environment is also polluted by tobacco smoke because of an interaction that often occurs between the two risk factors. In most developed countries today, 60% to 70% of the population of working age are non smokers and yet in offices, workrooms, storerooms, restaurants, taverns, transport vehicles, - wheresoever they work, these non smoking workers have to breathe air contaminated by tobacco smoke.

Environmental pollution by tobacco smoke comes in some measure from the smoke exhaled by smokers but the main source is sidestream smoke (SS) which contaminates the air with the same toxic compounds as occur in mainstream smoke (MS).

SS particulate matter is composed of smaller particles than that of MS and consequentially is potentially more dangerous because small particles are capable of travelling deeper into the airways before deposition than are large particles. Furthermore, because of the different flow rates of air passing over the burning cone of a cigarette and the different burning temperatures of the cone during the formation of

mainstream and sidestream smoke, SS formation involves less thermal decomposition and more compounds are distilled. Thus the concentrations of most components of both the vapour phase and the particulate phase are higher in SS than in MS. The following table lists a small number of the toxic and carcinogenic compounds that occur in tobacco smoke and shows the ratio of their concentrations in MS and SS. Sidestream smoke is also more alkaline than MS and therefore more nicotine is present in the form of the free base in environmental tobacco smoke.

Toxic and carcinogenic agents in undiluted cigarette SS (177, 178)

Compound	Type of toxicity	Amount in SS (per cigarette)	Ratio of SS/MS
Vapor phase:			
Carbon monoxide	T	26.8-61 mg	2.5-14.9
Carbonyl sulfide	T	2-3 mg	0.03-0.13
Benzene	C	400-500 µg	8-10
Formaldehyde	C	1,500 µg	50
3-Vinylpyridine	SC	300-450 µg	24-34
Hydrogen cyanide	T	14-110 µg	0.06-0.4
Hydrazine	C	90 ng	3
Nitrogen oxides (NO_x)	T	500-2,000 µg	3.7-12.8
N-nitrosodimethylamine	C	200-1,040 ng	20-130
N-nitrosopyrrolidine	C	30-390 ng	6-120
Particulate phase:			
Tar	C	14-30 mg	1.1-15.7
Nicotine	T	2.1-46 mg	1.3-21
Phenol	TP	70-250 µg	1.3-3.0
Catechol	CoC	58-290 µg	0.67-12.8
o-Toluidine	C	3 µg	18.7
2-Naphthylamine	C	70 ng	39
4-Aminobiphenyl	C	140 ng	31
Benz(a)anthracene	C	40-200 ng	2-4
Benzo(a)pyrene	C	40-70 ng	2.5-20
Quinoline	C	15-20 µg	8-11
N'-nitrosonornicotine	C	0.15-1.7 µg	0.5-5.0
NNK	C	0.2-1.4 µg	1.0-22
N-nitrosodiethanolamine	C	43 ng	1.2
Cadmium	C	0.72 µg	7.2
Nickel	C	0.2-2.5 µg	13-30
Polonium-210	C	0.5-1.6 pCi	1.06-3.7

Abbreviations: C, carcinogenic; CoC, cocarcinogenic; MS, mainstream smoke; SC, suspected carcinogen; SS, sidestream smoke; T, toxic; TP, tumor promoter; NNK, 4-(methyl-nitrosamino)-(3-pyridyl)-1-butanone.

The effects of Involuntary Smoking

In most developed countries, the prevalence of smoking among adults has fallen from the very high values of the 1950's to between 30% and 40%. Thus some 65% of adults in these countries do not smoke and, although the ratio of smokers to non smokers varies with age, sex, educational attainment, socio-economic group, and in different occupations, the situation in general terms is that 65% of adults have chosen to refrain from willingly inhaling tobacco smoke, and yet many of them are frequently in situations where they are obliged do so unwillingly. Even among smokers, there are many who dislike inhaling environmental tobacco smoke. In many developing countries, smoking prevalence among men is very high and among women very low and at least half of the adult population - mainly women - are likely to have to smoke involuntarily.

In some industrial situations the problem has been alleviated indirectly because ventilation systems are obligatory for the removal of other pollutants. However, in many workshops, stores, and offices, ill conceived architectural designs based primarily on energy conservation rather than employee welfare have resulted in work situations where air changes are not at a high enough rate (for fear of heat loss and hence expense) or in which air is merely recirculated after filtration (which fails to remove many smoke contaminants). Some workplaces are not ventilated at all.

Taverns and bars are examples of occupational situations with outstanding tobacco smoke pollution. The clientele chose the environment for short periods of diversion, but bartenders are subjected to the atmosphere throughout their working day and in a Canadian survey (160) bartenders were found to be the occupational group at highest risk from lung cancer - even higher than the several building trades often associated with high risk.

Many of the effects of involuntary smoking are difficult to quantify and hence difficult to demonstrate, for example, annoyance, discomfort and pain cannot be directly measured. Annoyance by tobacco smoke is suffered by many people and its effects at work are a decrease in efficiency and productivity. Dry throat, discomfort in the nasal cavity and sinuses and headache are frequent complaints which are difficult to quantify but which can affect productivity. Eye irritation can often be seen and some people are allergic to tobacco smoke, suffering breathing distress in a smoke contaminated atmosphere.

Allergies, headaches, sore eyes, annoyance, are all reversible and since they are transient they seem to be considered to be a reasonable price that the many should be willing to pay for the satisfaction of the few compulsive smokers. The question therefore that arises is: at what level does the price become too high? Is it perhaps when the harm is measurable?

There is a measurable elevation of carbon monoxide in non smokers as a result of passive smoking(161) and in closed spaces the carbon monoxide concentration increases linearly with the rate of cigarette burning.

There is a significant and measurable concentration of cotinine in both blood and urine of non smokers exposed to passive smoking and it has been shown(162) that the half life of this nicotine metabolite is longer in non smokers than in smokers. It has been suggested that the longer residence of nicotine alkaloids and metabolites in non smokers could increase the probability of the endogenous formation of carcinogenic N-nitrosamines.

Involuntary inhalation of tobacco smoke causes a measurable increase in heart rate, probably due to the nicotine and carbon monoxide.

The traditional spirometry measurements of lung function, FVC and FEV1, often give normal values in the presence of small airways disease, but reduced mid expiratory flow rate (FEF 25-75) and end expiratory flow rate (FEF 75-85) have been shown to be commensurate with small airways disease. From measurements of these it has been shown that non smokers score well above all other groups tested. Passive smokers and non inhaling smokers have an equal measure of small airways dysfunction(163). Further increases in exposure to cigarette smoke cause a progression from small airways involvement to extensive bronchiolar and alveolar disease(164). It has been shown(165) that children living in households with parents who are smokers score a reduced FEF 25-75 and the score decreases further with increasing exposure to smoke, i.e. when more than one member of the household is a smoker.

Numerous studies on a possible relationship between involuntary smoking and lung cancer incidence, usually among the wives of smokers, have been carried out with conflicting results. However, by using large sample numbers, carefully verifying smoking status, and obtaining histological verification of cancers as being primary lung cancer(166) it was concluded that there is an elevated risk of lung cancer ranging from 13% to 31% in women who are exposed to the smoke of others. From an analysis of the results of 13 studies, the conclusion reached(167) was that there was a highly significant 35% increase in lung cancer risk from involuntary smoking.

Thus, environmental tobacco smoke can cause lung cancer, pulmonary dysfunction, irritation to the eyes, nose, throat and tracheobronchial system, increased blood carbon monoxide concentration; be responsible for the presence in the body of potentially harmful nicotine metabolites, cause allergic reaction and engender annoyance.

Quality standards for air in the working environment are maintained in many countries and "Limit Values" state the concentrations permissible for hazardous materials in the workplace. However, there is no limit for the concentration of tobacco smoke in the air. No reference has been made to tobacco smoke particulate matter, yet there is a limit for coal tar pitch volatiles which are no more rich in carcinogens than is tobacco smoke. The particulate, vapour and gas phases of tobacco smoke contain compounds which are irritant, carcinogenic, asphyxiant, or allergenic and many of them, as individual compounds have been ascribed limit values, yet when they occur in tobacco smoke, no value is assigned.

The absence of a limit for the permissible concentration of tobacco smoke in the workplace gives a shield behind which industrial welfare officers can abstain from making recommendations, employers can vacillate and refrain from action, and smokers can continue to promote ill health in their coworkers.

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5. Tobacco Chewing

Tobacco chewing is a widespread practice in the USA and South east Asia, with some use in Europe and other Asian countries. The habit, once common worldwide, lost popularity during the nineteenth century in favour of smoking and during the twentieth century cigarette smoking became the main form of tobacco use in almost all developed countries although there has been a resurgence of the habit in recent years.

Little consideration seems to have been given to the interaction of tobacco chewing with other hazards but there is no reason to doubt that conditions linked to peripheral vascular disease will be exacerbated by the nicotine absorbed during chewing. Thus similar effects on hearing acuity and Reynaud's syndrome and Dupuytren's disease may be expected from chewing as from smoking. Although the interactions occurring between smoke and industrial hazards that affect lung function and cause respiratory disease are unlikely as a result of chewing tobacco in the presence of other hazards, the fact that chewing tobacco is almost as rich a storehouse of harmful chemical compounds as is tobacco smoke (e.g. nitrosamines) suggests that systemic effects can be expected, such as synergistic effects on bladder cancer.

Chewing has remained a habit among certain working groups in many countries, particularly when the nature of the working environment is such that smoking must be

forbidden for safety reasons, as in coal mining and many sections of the chemical industry. It also remains common in groups whose occupation requires the use of both hands, such that neither remains available to help with smoking.

In the USA, chewing tobacco is preferred by many workers in heavy industry, such as steel and coal, and in such as the petroleum industry where the inflammability of the environment precludes smoking. Although tobacco chewing is uncommon in the UK, it is found in coal miners, particularly among those working underground. In Sweden, around 17% of the population are users of moist snuff.

Research into the added effects of the use of oral tobacco on the effects of environmental and workplace hazards is needed.

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6. Some economic effects of smoking in the workplace

> Smokers take more sick-leave than non smokers take. Work-time loss, decreased efficiency and increased cleaning costs are attributable to smoking at work. Employers have higher employee health insurance costs because of actuarial estimates of excess health care costs attributable to smoking. Accident insurance is higher because smokers have twice the number of accidents suffered by non smokers. Around 10% of all industrial fires result from smoking. The foregoing sections have dealt with the increase in occupational diseases that are suffered by smokers. These can be translated into increased costs and losses.

The effects at the personal and family level of incapacity due to ill health, or of death, need little elaboration. At the financial level there is hardship, problems arise at the social level and there is psychological stress which can itself lead to further physical illness(168).

Coronary heart disease and stroke caused 38% of all deaths in England in 1989 with an estimated loss of 42.7 million working days. Of these, 25% can be attributed to smoking. The second most common cause of mortality was cancer which accounted for 25% of all deaths and it is likely that one third of these deaths were smoking related.

The economic costs and losses at the industrial level are significant, and arise from several sources.

1. Absenteeism. From a review of several studies(169) the summarized conclusions were that smokers have 33% to 45% more absenteeism than non smokers. Studies of work site health-promotion programmes also found reduced absenteeism rates after a year or two of smoking cessation. Data from a U.S. Health Interview Survey in 1970 showed that the number of work days lost per year for both sexes/all ages above 17 years were 6.3, 5.2 and 4.4 for present-, former- and never-smokers respectively(170). Data from a later U.S. Health Interview Survey have shown higher work absenteeism among smokers than non smokers: 5.7 days per year for a man smoking 25 cigarettes a day, compared with 4.3 days for a non smoker. In a survey of smoking and excess sick leave in a Department of Health in the U.S. It was found that in a 20 month period during 1983-1984, 97 smokers took an average of 5.3 hours per month sick leave, whereas 309 non smokers took 4.3 hours. The excess leave by smokers was regardless of sex, marital status or age(171). In a study among healthy young men of the incidence of influenza, during an outbreak of the disease, it was found that 68.5% of the smokers had influenza compared with 47.2% of non smokers, furthermore, the influenza was more severe in the smokers: 50.6% of the smokers lost work days compared with 30.1% of the non smokers(172). Clearly the greater work absenteeism of smokers must have a significant cost element which may vary between industries. Furthermore, there will be added costs for hiring temporary replacements or redistributing work among other workers.

2. Lost productivity. Smoking takes time, adds to the costs of cleaning, the carbon monoxide decreases worker efficiency in several activities and the irritant effects of smoke can further decrease attentiveness. Lost productivity can also arise from the hostility of non smokers to conditions in which smoke pollution is permitted. A method of minimal costing has been suggested based on an assumed loss of one minute per working hour.

3. Insurance costs. The levels at which employers contribute to staff insurance is very variable but excess health insurance costs have been calculated by all insurance companies, based on the percentage of certain diseases which are caused by smoking, and excess insurance costs are borne by all employers because of the percentage of the workforce who are smokers. This topic has been reviewed by Kristein(169) for the situation in the U.S. Accident compensation insurance is also higher because of smoking: smokers have twice the number of accidents that occur among non smokers.

4. Fire and accident losses. The fire losses attributable to smoking have been estimated at 7.3% to 11% of all fire losses and half of the total value of fire losses is accounted for by industrial fires, the other half is due to residential fires (including hotels and motels)(169).

5. Occupational diseases. The foregoing section of this document have dealt with the interactive effect of smoking and various occupational hazards. In many industries, the further reduction of the levels of occupational hazards would be a far more expensive operation than controlling cigarette smoking and the latter course of action would serve the dual purpose of increasing workers health and reducing employers financial liabilities.

6. Passive smoking has been shown to be a cause of higher rates of respiratory disease than are encountered in those not exposed to tobacco smoke. Environmental tobacco smoke is thus an added cost centre in industry.

Only the economic costs at the industrial level have been considered above. The costs and losses at the national level must take into account all aspects of health and social welfare benefits to both the sick and their dependents as well as the overall losses in national production arising from the effects of ill health and disability upon industry. This is, however, a subject beyond the scope of the present monograph, which will be considered elsewhere.

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7. Summary and conclusions

Many harmful effects of smoking are now well documented and have been overwhelmingly substantiated. There have been a considerable number of investigations of the interaction of smoking with and the health hazards found in various occupations and this short survey has reviewed some of the risks that have been associated with smoking in the workplace. Several subjects have not been dealt with in any detail, such as: allergic reactions and sensitization; certain cardiovascular diseases; effects involving cell division; the effects of smoking on concentration, performance and efficiency, on sight, on the autonomic nervous system, on systems of detoxification and on reproduction. Although there is information concerning occupational hazards in these areas, and the effect of smoking is known in some, further research on the interaction of the hazards is needed.

There is now considerable information on the ways in which smoking and many industrial hazards interact to cause increases in severity or more rapid onset of

diseases, and yet an impression gained from all the studies is that the harm occasioned by smoking outweighs that for all other agents, and in some cases it is difficult to avoid the conclusion that if damage to tissues from smoking had not occurred, the effects of workplace hazards would have been far less severe.

It is known that:

- Involuntary smokers, teenage smokers, and children in households where there are smokers, suffer from pulmonary dysfunction which can be equated with small airways disease.

- Smoking has a detrimental effect on mucus secretion, mucus quality and lung clearance.

- Smoking surveys in many countries have shown a high prevalence of addicted smokers among teenagers, and most habitual smokers start the habit as teenagers.

The inevitable conclusion from these facts is that an enormous number of young people suffer from small airways inflammation and impairment of their lung clearance mechanism solely from tobacco smoke and this occurs before they ever encounter any occupational hazards. Questions arise, therefore, on the role of smoking related disabilities as precursors of many other diseases, particularly the occupational diseases. It seems reasonable to suppose that any hazardous material encountering tissue that is already suffering some degree of inflammation due to smoking will have a more profound effect than if it were to impinge upon normal healthy tissue. Furthermore, if harmful inhaled materials cannot be efficiently cleared, because smoking has damaged the clearance mechanism, within a short time of being deposited, their longer term residence in the respiratory system will occasion greater harm than if they had been rapidly removed.

In all countries where surveys have been carried out, the highest prevalence of smoking is among the so called "blue collar workers" and from many of the studies cited in this document, it is seen that there is invariably a very high prevalence of smoking among workers who are involved in jobs where dusts are generated. Thus, most workers are susceptible to the effects of dust when they first join the work force and reinforce their susceptibility throughout their working lives.

The inevitable conclusions from this review are:

1. Tobacco smoke can modify the risk associated with many hazardous materials encountered in the workplace and in some cases the interaction causes an extraordinary increase in the severity of the disease, often advancing its onset and accelerating its rate of development.

2. Chemical substances associated with health risks are often present in both cigarette smoke and in the working environment and thus each source can add to the burden imposed by the other to increase the severity of a disease.

3. Harmful materials in the workplace can contaminate smoking materials and be transferred from there to the user, in some cases causing severely debilitating and possibly life threatening diseases.

4. Innocuous materials in the workplace can be transformed by the smoking process into extremely harmful compounds.

5. The effects of smoking on the vascular, particularly the peripheral vascular, system can considerably enhance the diseases which affect the extremities and impair the use of the hands and also affect hearing acuity.

6. Passive smoking is invariably a workplace hazard whether the smoke is the only atmospheric contaminant or occurs in combination with other workplace pollutants.

7. All diseases suffered by a work force, whether caused by smoking or caused by industrial hazards, or enhanced by interaction of the two, result in absenteeism, often lead to early retirement due to disability, reduce productivity and increase employers costs. Thus the worker is put under financial stress and the employer, suffering work disruption and the loss of skilled labour, encounters economic loss. Ultimately the economic losses are reflected in national economies which are at further disadvantage because of the economic costs associated with these diseases that arise from increased medical service costs and increases in the costs of social benefits.

8. A complete ban on smoking in all places of work would be advantageous to all concerned.

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