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More Effective Prevention in Occupational Health Practice?*

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Summary

Official figures for occupational disease in the United Kingdom underestimate the true incidence of work-related disease. Successful prevention depends first on identifying risks, either by preliminary testing before anyone is exposed or by observing adverse effects on work people. Methods of identification need to be systematic and more sensitive to detect long term effects and their causes. They can be improved by studying geographical patterns of disease and classifying work by exposures rather than job titles. Incidence and severity of disease are the product of interacting causes, environmental exposures, host factors and individual behaviour—all of which need to be borne in mind if prevention is to be more effective. Occupational health has special opportunities to undertake a wider range of preventive activities which include modification of personal habits. Smoking, diet, physical exercise and control of infection by immunization and screening for non-occupational disease come into this category.

Introduction

It is a special honour for me to give the first Jameson–Parkinson Memorial Lecture, endowed by the late Dr Margaret Dobbie-Bateman. These are names that will mean little to many of you, but they are three people to whom this Society owes much for its foundation and early development.

Nearly 50 years ago a small group of industrial medical officers sought the advice of Professor Wilson Jameson, Dean of the London School of Hygiene and Tropical Medicine, about founding an association of industrial medical officers. He encouraged the idea and offered to provide an address and a meeting place at one of the main academic centres of preventive medicine in Europe. This gave the new Association of Industrial Medical Officers† an air of respectability at a time when industrial medicine was a relatively new discipline and was not regarded highly by the medical profession. When Professor Jameson became Chief Medical Officer at the Ministry of Health he was succeeded as Dean by Brigadier Parkinson, who continued to give the fullest support to our Society. Parkinson and Jameson, at that time, were household names as the authors of the *Synopsis of Hygiene*, an authoritative reference book on Public Health and the 'bible' of every student of the subject. Dr Margaret Dobbie-Bateman, who was Medical Officer to Harrods, was the first honorary treasurer of the Association. She will be remembered for her kindness to the fledglings in industrial medicine and for the support she gave to this Society which will be continued through her generous bequest.

*The first Jameson–Parkinson Memorial Lecture delivered at the Annual Provincial Meeting of the Society of Occupational Medicine in July 1983.

†In 1965, with its wider scope, it became the Society of Occupational Medicine.

At the Society's twenty-first Anniversary Meeting, 27 years ago, I was privileged to be President and to be invested by Sir Wilson Jameson with the new Presidential Jewel, bearing the portrait of Charles Turner Thackrah, the first British physician to advocate a new discipline of occupational health, based on prevention of disease and conservation of health (Meiklejohn, 1957). It was an inspiring ceremony for me as director of a newly established Occupational Health Unit at the London School of Hygiene and Tropical Medicine.

Today's lecture seems an appropriate occasion to attempt to answer the question—can occupational health achieve more effectively the objectives of Thackrah (1832), 'prevention, rather than the relief of evils'?

Incidence of Occupational Injury and Disease

Official Figures

The official figures for fatal and non-fatal industrial injury and disease represent a small fraction of the total mortality and morbidity in the British working population. Out of some 100 000 deaths per year of men and women aged between 15 and 64, about 2 per cent (1300 injuries and 1000 prescribed industrial diseases) are directly due to work. For all non-fatal injuries, the proportion among men due to work is 45 per cent and for women 23 per cent (*Table I*).

Table I. Annual non-fatal injuries* in working population (Great Britain) thousands

	Males	*Females*
Total	1390	390
Work	630	90
Motor vehicle	150	50
Other	610	250

* Estimates in round numbers based on data for 1973–75 for injuries leading to 4 or more days off work or an equivalent degree of severity for people not at work. *Source*: Royal Commission on Civil Liability (1978).

The working population in Great Britain makes more than 9 million claims a year for new spells of sickness, of which about 9500 are due to prescribed industrial disease (*Table II*).

Do these figures give a true picture of the incidence of work-related injury and disease? A work injury is easy to identify because it can be related to a particular event. The figures may even exaggerate incidence. The larger benefits received for work injuries and the prospect of a common law claim against the employer may understandably encourage an injured person to claim industrial injury benefit for a non-work injury.

Table II. Annual new spells of sickness in Working Population (Great Britain, 1979–80) thousands

	Males	*Females*
All causes	6200	3000
Respiratory	1800	1000
Musculoskeletal	790	230
Mental	240	190
Circulatory	220	40
Prescribed	9·5	

Source: Department of Health and Social Security (1982) from data based on sickness and invalidity benefits paid to insured population of about 26 million.

Occupational disease is more difficult to identify because cause and effect are not closely related in time. All the evidence indicates that the official figures underestimate incidence. They are based on prescribed occupational diseases and omit many disorders not meeting the rigorous conditions required for prescription.

Other Data

A random sample of households in Great Britain was surveyed in 1973, at the request of the Royal Commission of Civil Liability. The survey identified 413 household members who had contracted illnesses between 1970 and 1973, which they believed arose from their work. On further enquiry, 290 considered the work link was probable but only 45 of them had a prescribed occupational disease (*Table III*). Results such as these, which are based on the subject's opinion, have to be interpreted with caution. Nevertheless, they indicate a widespread belief that many illnesses which are not prescribed can be attributed to working conditions and may be of the order of five times as numerous as those which are prescribed.

Table III. Four hundred and thirteen subject's opinion on work as a cause of illness and the number with prescribed disease

Probable link 290	Doubtful link 123
Prescribed disease 45	3

Source: Royal Commission on Civil Liability (1978).

In Finland the Institute of Occupational Health in Helsinki, which is the envy of many countries because of the quality of its research and teaching, has an Occupational Disease Register. This Register obtains its data from three sources: 1, accident reports and physicians' diagnoses obtained by insurance companies; 2, occupational diseases diagnosed at the Institute; and 3, occupational disease reports by provincial medical officers. The Register includes categories of disease such as salmonella infection, brucellosis, viral hepatitis and vibration syndrome, which are considered to be of occupational origin. None of these is a prescribed occupational disease in this country. Taking account of their much smaller working population of 2·36 million, Finland records about three times more occupational disease than Britain (*Table IV*).

The figures in *Table IV* are a relatively small fraction of the total burden of disease in both countries, but there should be scope for more effective prevention as they are caused by known environmental agents and other factors which can be eliminated or controlled.

Table IV. Incidence of occupational disease in Great Britain and Finland

Occupational disease	*Finland (1979)*	*Great Britain (1979–80)†*
Infections	80	27
Poisoning and other diseases	141	95
Respiratory system	377	935
Musculoskeletal connective tissue	1049	3672
Skin	1183	5660
Hearing loss	1474	5900
Vibration syndrome	78	—*
Total	4382	16 289

* Not prescribed
† 12 months period
Sources: Institute of Occupational Health (1980) and DHSS (1982).

Stages in Prevention

Preventive action can be taken at three stages—*primary* through identifying causes of illness and injury and eliminating or controlling them; *secondary* by detecting early manifestations of disease through health surveillance; and *tertiary* through minimizing disability and restoring function through rehabilitation.

Both secondary and tertiary prevention are important functions of any health care system and, in particular, occupational health, with the opportunities it offers for screening workers exposed to health risks, for assessing capacity for work and for rehabilitating the injured in their own work place. I have no doubt that secondary and tertiary prevention could be made more effective, but the real need in terms of reducing the burden of injury and illness and suffering is to have a more effective primary prevention programme. In this lecture I shall focus on primary prevention.

There are two ways of achieving more effective primary prevention—first by better identification and control of all types of work-related disease and injury, and secondly by helping work people to reduce the risk of chronic disease by modifying their life styles with respect to smoking, diet and exercise. The first is not controversial, but the second may be regarded as outside the scope of occupational health services and would be unacceptable to many managements and representatives of the work force.

Sir Richard Doll (1983), in his Harveian Oration, gave modification of personal habits, particularly smoking, as offering the greatest opportunities for prevention, apart from socioeconomic improvements for the poorer members of the community. I do not think any health service can afford to ignore this opportunity.

Work-related Disease and Injury

The incidence and severity of a disease in a working population is the product of many interacting causes that may be grouped as host factors inherent in the worker, the external environment and individual behaviour (*Fig.* 1). A single cause may be necessary to produce an occupational disease like lead poisoning, but in itself may not be sufficient. Other factors like host susceptibility, attitudes to safe methods of working and personal hygiene will determine which individuals are

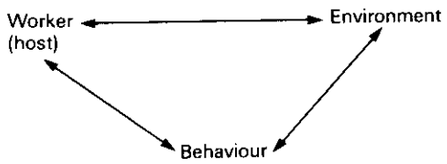

Fig. 1. Multiple causes of disease (from Morris (1975)).

affected and how severely. This concept of multiple causality is even more relevant to the control of other types of work-related disease and in the prevention of injury from work accidents. Work-related diseases can be grouped into three categories (*Table V*), in which work is the necessary cause (I), a contributory cause (II) and an aggravating factor (III).

Table V. Categories of work related disease

	Example
I Work—the necessary cause	Lead poisoning
II Work—a contributory causal factor, not a necessary one	Coronary heart disease
III Work—provokes a latent disorder or aggravates established disease	Peptic ulcer Eczema

Although occupational diseases, i.e. those in category I, can usually be readily identified, a necessary cause may be obscured. Epidemiological evidence indicates cotton dust as the necessary cause of the prevalent specific cotton workers' respiratory disease (World Health Organization, 1983). For many years the wet climate and polluted atmosphere of Lancashire cotton towns were regarded as the major causes of chronic bronchitis in cotton workers. More recently smoking has been incriminated as the major cause. Long term low level exposures to toxic chemicals may be both contributory and necessary aetiological factors in chronic disease. It may also be difficult to distinguish between diseases which are caused by and those which are merely aggravated by work. Such a distinction may be relevant in the award of compensation, but once the adverse work affect has been identified, it has little relevance to prevention or to patient care.

Identification of Work Factors Causing Disease and Injury

Clues which point to a health hazard are the first stage in identifying a risk of work-related disease or injury.

There are two distinct methods of identification: 1, to predict a risk in a new chemical and to prevent its use before anyone is exposed. The same principle applies to machines—the hay baler reported to have killed six British farmers and seriously injured others should have been identified as potentially dangerous in the design stage and modified before being put on the market. 2, to detect risks by observing work people exposed to adverse environmental agents.

Predicting Risks before Use

Risks may be predicted by toxicity testing, which is now required for new substances used in quantities of 1 tonne or more per annum in member states of the European Communities (Statutory Instruments 1496, 1982).

There is a wide variety of tests including long term animal tests and short term *in vitro* tests for mutagenicity and cell damage. They are, however, beset with difficulties (Royal Society, 1983); first because of their doubtful relevance for man, secondly because of their cost. A spectrum of tests, including acute toxicity and chronic studies, costs about £500 000. Thirdly, there is a world-wide scarcity of skilled manpower and facilities for carrying out the animal and other laboratory tests currently demanded by authorities. A Royal Society Group (1983) suggests 'limited testing' before authorizing the temporary use of a chemical for which a good case has been made. If the chemical structure of a compound is similar to a known carcinogen or the results of short term tests give rise to grounds for concern, more extensive testing would be necessary. If, on the other hand, screening tests were satisfactory, it might be reasonable to give temporary approval to use for, say, 5 years. During the trial period there would be appropriate surveillance to identify levels of exposure and any adverse health effects. A similar procedure could be adopted for potentially dangerous machines.

Except for chemicals which are clearly toxic or machines which are recognized as potentially dangerous in the design stage, identification of health hazards ultimately depends on observations on man.

Finding Clues by Observing Work People

There are several ways of finding clues concerning work-related disease.

Clinical Observation

In 1775 Percivall Pott, a surgeon whose name is associated with a fracture and spinal tuberculosis, described scrotal cancer in chimney sweeps. One hundred and fifty years later Dr Wilson, a House Surgeon at the Manchester Royal Infirmary, reported the occurrence of this disease in cotton mule spinners (Southam and Wilson, 1922).

Clinical observations by Amor (1960) gave the first clues to a risk of nasal cancer in nickel refiners and by Esme Hadfield of a similar risk in woodworkers (Macbeth, 1965).

Observations by Managers and Workers

A works manager in an arsenical sheep dip factory thought an unduly high proportion of his workers were dying of cancer. An epidemiological study by Bradford Hill and Faning (1948) confirmed his suspicions and led to improved control measures. A farm worker was among the first to suspect that mouldy hay caused Farmer's Lung.

Such observations are likely to be made only for rare types of disease or conditions with characteristic symptoms. But, they are still an important source of identification.

Routinely Collected Data

There is a wide variety of mortality and morbidity data routinely collected by government departments, corporate bodies, hospitals and work places. These can give help in identifying health risks.

In the nineteenth century and early years of the twentieth century, national mortality data in England and Wales, published every 10 years by the Registrar General, revealed gross excesses of deaths in hazardous occupations such as coal and tin mining and preparing cotton for spinning. These and other dangerous occupations have since been made safer. Such data have become less useful because they fail to detect the more obscure occupational hazards which still have fatal consequences, for example exposure to carcinogens, causing common types of malignant disease, such as lung and bladder cancers.

Other mortality data collected by local Registrars of Death, hospitals and companies are seldom analysed routinely to generate hypotheses, but rather to test hypotheses. Examples are the death registers in a country town, used to detect a suspected cancer risk in a sheep dip factory (Bradford Hill and Faning, 1948); the analysis of hospital records to examine the relationship between asbestos exposure and mesothelial tumours (Newhouse and Thompson, 1965); and a study of company pension records to identify a mortality excess from cerebrovascular disease in lead workers (Dingwall-Fordyce and Lane, 1963).

Routinely collected *morbidity* data at national level give a broad picture of the burden of sickness absence and invalidity among the gainfully employed, but they seldom bring to the fore new health problems. Sickness absences, recorded by occupational health services, are more useful. They are helpful in the management of individual health problems and can be used epidemiologically to identify adverse working conditions.

National figures for work injuries, based on compulsory reporting of certain types of accident to the Health and Safety Executive, may highlight dangerous work practices such as a dramatic increase in injuries from overturning farm tractors (Ministry of Agriculture, Fisheries and Food, 1971).

Occupational diseases, recorded in *Special Registers*, are fairly crude epidemiological tools. They are incomplete because physicians neglect their responsibility to report or they may fail to connect diseases with work conditions. However, a register, for example of beryllium or asbestos related disease, may indicate the occurrence of occupational disease in jobs not previously recognized as being hazardous.

It is necessary for occupational health services to keep records of medical examinations and treatments for dealing with the problems of individual workers. They also have an epidemiological value in identifying health hazards of all kinds, ranging from the apparently trivial, such as arc eye or irritation of the respiratory tract, to more serious injuries and diseases.

It is an important function of an occupational health service to identify health risks, but more than half the working population in Britain do not have such services (Employment Medical Advisory Service, 1978). Moreover, most occupational physicians are not trained to undertake this kind of preventive medicine.

Better Identification of Risks

The identification of undisclosed hazards tends to be unsystematic and to depend on methods that are not sensitive enough to detect less obvious risks, particularly those in which ill effects become apparent after many years.

There are two recent developments in clue finding which should lead to better identification of work-related disease.

Geographical Patterns of Disease

Studies of variations in mortality for large areas such as regions or counties are likely to obscure clues to causes of disease occurring in small areas where particular types of industry are located. Professor Donald Acheson and his colleagues from the Medical Research Council's Environmental Epidemiology Unit have investigated geographical variations in mortality among the 1366 local authority areas of England and Wales, covering the 11 years between 1968 and 1978 (Gardner et al., 1982a, b).

Standardized mortality ratios (SMRs) were calculated for rare types of disease like pleural mesothelioma, bladder cancer and nasal cancer. They were based on the populations of the 1366 areas at the time of the 1971 census. Areas with SMRs in the top tenth of the distribution and raised above the average 100 at the 1 per cent level of statistical significance have been plotted. The method picks out known hazards. The high risk of mesothelioma among women is seen in *Fig.* 2 in some relatively small areas where asbestos was used in the

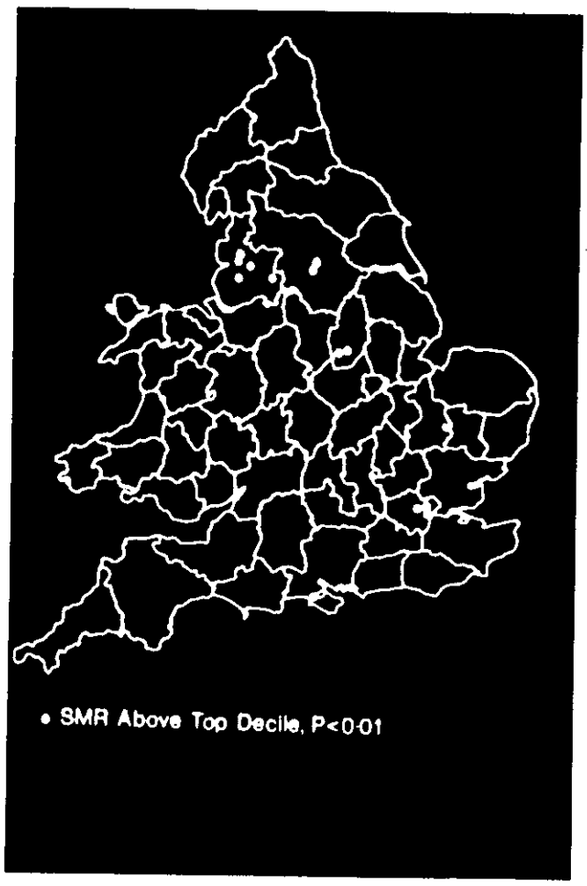

Fig. 2. Local Authority areas with raised mortality from mesothelioma of the pleura in women (1968–78). Gas mask manufacturing—Leyland, Carlton, Preston, Blackburn and Nottingham. Textile manufacturing and insulation materials—Spenborough, Leeds, Rochdale, Barking, Newham and Bexley (from Gardner et al. (1982a)).

manufacture of textiles and of gas masks (Gardner et al., 1982a).

The areas with a high mortality from bladder cancer in men (*Fig.* 3) are located mainly in parts of England where the dye stuffs and rubber industries are concentrated but there are apparent excesses in several places such as Weymouth and Melcombe Regis and the London Borough of Southwark, which cannot be explained on the basis of known industrial carcinogens. This has led to further investigations (Gardner et al., 1982b).

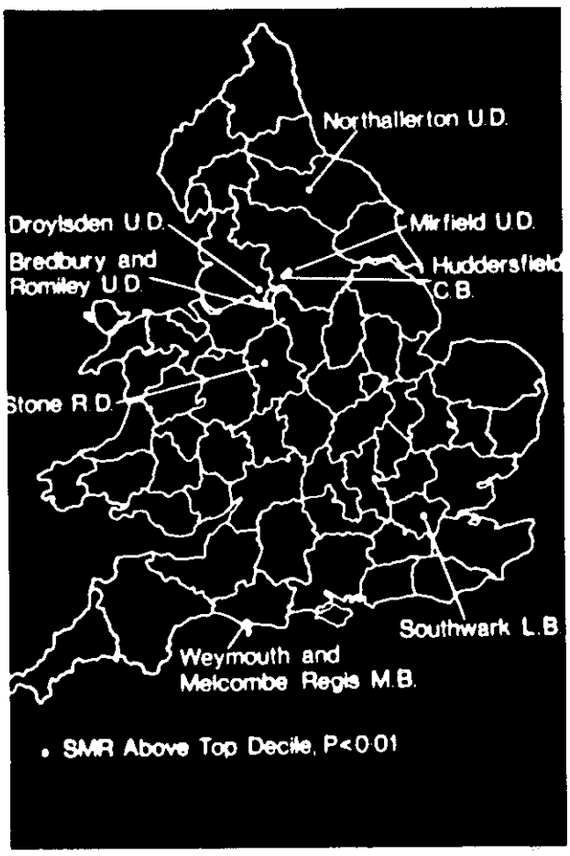

Fig. 3. Local Authority areas with raised mortality from bladder cancer in men (1968–78) located mainly in northern areas with dye stuffs and rubber industries. Three areas in south have no recognized industrial risk (from Gardner et al. (1982b)).

Thus, systematic study of mortality in much smaller areas than have been studied in the past may give clues to possible causative factors of disease in the work environment.

It is now possible to produce computer drawn maps of England and Wales, depicting the incidence of 37 sites of cancer in local authority areas (Gardner et al., 1983). The National Institute for Occupational Safety and Health (NIOSH) is also using computer drawn maps for the 3073 United States counties to generate hypotheses on occupational hazards by relating mortality data to types of work in the same small areas (Frazier et al., 1983).

Classification of Work by Exposures

The classic epidemiological approach to studying the relationship between work and disease is to examine the frequency of disease in particular occupations. This serves its purpose where the occupation has a dominant exposure and the risk is high. It is insensitive when the risk is not severe and the job title includes workers who are not exposed or are lightly exposed. A job title may also conceal multiple exposures.

Various research centres in Europe and North America have developed job-exposure matrices (JEMs). JEMs assemble lists of substances to which workers are exposed in specified occupations and conversely lists of occupations in which exposure to a particular agent (e.g. benzene) is a common factor (Medical Research Council, 1983). The relative risks for bladder cancer associated with three job titles were studied in patients in Massachussetts. Subsequently a matrix of job titles associated with aromatic amines was compiled and the data reanalysed, using exposure rather than job title as the basis for classification (Hoar et al., 1983). A much higher relative risk was found for persons exposed to aromatic amines than to any of the job categories (*Table VI*). The analysis by exposure had the effect of excluding subjects in the job categories who had not been exposed to aromatic amines. Three potential uses of JEMs are: 1, the identification of previously undisclosed hazards; 2, spotting jobs in which workers are at risk to known hazards; and 3, identifying groups with multiple exposures and measuring their effects. JEMs may be further refined by estimates of degrees of exposure into light, moderate and heavy. For the future, with improved techniques, environmental exposures can be quantified and could thus enable JEMs to determine dose–response relationships and more reliable control limits.

Table VI. Bladder cancer in Massachusetts

Occupational category	*Relative risk*	*95% confidence limits*
Dyestuffs	2·2	0·7– 7·6
Rubber etc.	1·6	1·0– 2·4
Leather etc.	2·0	1·4– 2·9
Exposure category	8·6	6·2–12·0

Source: Medical Research Council (1983)

Control of Work-related Disease

Clue finding is the first step in the control of work-related disease and is probably the weakest link in the chain. The steps which follow include formulation of a hypothesis on causes and its testing, determining the extent and severity of risk and establishing control limits.

Category I Work-related Disease

This can be relatively easily controlled by a competent occupational health service when attention is given to host factors and individual behaviour as well as environmental control (see *Fig.* 1).

Regulations were passed in 1925 to control a rising incidence of lead poisoning in the manufacture of electric accumulators. They had some effect, except in a large company where the yearly incidence of poisoning was still rising. In 1927 the company appointed its own occupational physician, Dr Ronald Lane. In the factories under his supervision lead poisoning was almost eliminated. They produced 90 per cent of lead batteries

made in the United Kingdom. They were responsible for only 6 per cent of all cases of lead poisoning which occurred between 1930 and 1961 in workers making lead batteries. This success was achieved by environmental control, medical surveillance and education of management and work force (Lane, 1949). Medical policy was to exclude those with hypertension, nephritis, peptic ulcer or epilepsy and slovenly workers of low intelligence. Anyone showing signs of abnormally high lead absorption was temporarily removed from exposure. Lane (1949) stressed the importance of education. The worker must understand fully the danger of his work. This needs patience on the part of the doctor.

Category II Work-related Disease

This is where work is a contributory, but not a necessary cause, and both identification and control are more difficult.

A study of death rates among British viscose rayon workers exposed to carbon disulphide (CS_2) was undertaken after Vigliani had described cerebrovascular damage among Italian viscose rayon workers. There was no mortality excess from cerebrovascular disease, but, unexpectedly, a significant twofold excess of deaths from coronary heart disease (Tiller et al., 1968). This was strong suggestive evidence of an occupational risk and generated the hypothesis, which needed testing, that CS_2 exposure promotes coronary heart disease (CHD). A prospective follow-up study of exposed and non-exposed groups was undertaken by Hernberg and his colleagues in Finland. A large group of viscose rayon workers aged 25–64 years, with a minimum of 5 years exposure to CS_2, were matched individually with an equal number of subjects (the reference workers) from a nearby paper mill. Good comparability with respect to aetiological factors in CHD was achieved (*Table VII*). The results summarized in *Table VIII* revealed a significant excess of CHD deaths in the exposed group in the first 5 years (Hernberg, 1983). During this period the incidence of non-fatal infarctions and angina were also significantly increased. By the seventh and eighth years of the follow-up most workers with early manifestations of CHD (angina and ECG changes) were moved to jobs with no CS_2 exposure. In addition environmental concentrations of CS_2 had been substantially reduced to levels below 10 p.p.m. The excess of deaths declined and in the last 3 years of the follow-up, CHD mortality in the viscose rayon workers was less than that of the paper mill workers. This study shows how an occupational risk can be eliminated by a combination of *health surveillance* and *environmental control*, which are, in themselves, the basic elements of good occupational health practice.

Table VII. Comparability of CS_2 exposed and reference workers

Age Birth district Type of work Social class	Same
Cigarette consumption Physical activity Diet Body weight	No major differences between groups

Table VIII. Coronary heart disease deaths among 343 CS_2 exposed rayon workers and 343 referents

Group	*Before 1960*	*1967–72*	*1972–77**	*1977–80*
Exposed	—	14	15	5
Non exposed	—	3	8	8
Rate ratio	—	4·7	1·9	0·7
CS_2 exposures (p.p.m.)	40	10–30	<10	<10

* Removal from exposure of men with early CHD.
Source: Hernberg (1983).

Work-related Disease from Multiple Exposures

Multiple exposures to potentially harmful agents are common and widespread. They occur in manufacturing and service industries, in mines, laboratories and agriculture.

Combined effects, in theory, may be independent, antagonistic or synergistic, but in practice little is known about the health effects of multiple exposures in the work place (World Health Organization, 1981) or indeed outside it.

Synergistic effects which may be additive or multiplicative, are the most important from the health point of view, particularly where they are multiplicative as in cigarette smoking and exposure to asbestos (Saracci, 1981). However, it is generally assumed that when there are exposures to two or more hazardous substances acting upon the same organ system, their combined effects are additive and the control limit (threshold limit value) is exceeded if the sum of the following fractions exceeds unity:

$$\text{i.e. } \frac{C_1}{T_1} + \frac{C_2}{T_2} + \quad \ldots \frac{C_n}{T_n} > 1.$$

where C_1, C_2 and C_n are measured concentrations and T_1, T_2 and T_n are the respective control limits.

A group of 102 car painters in garages in Finland was exposed to a mixture of organic solvents (*Table IX*) in concentrations which were well below the control limit. Compared with a matched reference group of loco engineers, they had significantly more acute symptoms of irritation and prenarcosis and long term effects such as fatigue and loss of memory (Husman, 1980). They also had greater reduction in nerve conduction velocities (Seppalainen et al., 1978). The TLVs may have been too high, but the more likely explanation is that there was a potentiating effect of these solvents. There is evidence of similar effects in agricultural workers from combinations

Table IX. Organic solvent exposures of 102 car painters

	$\frac{C}{T}$*
Toluene	0·15
Xylene	0·06
Butyl aectate	0·05
White spirit	0·03
Other solvents	0·04
Total additive exposure	0·33

* C is the concentration of each solvent, T is the corresponding TLV.
Source: Husman (1980).

of pesticides (Kaloyanova-Simeonova, 1977) and in firemen from the products of pyrolysis (Axford et al., 1976).

Under laboratory conditions, responses to separate and combined exposures can be measured in animals and, sometimes, in human volunteers and the type of effect can be identified. But such experiments have a limited value. In the work place the opportunity to measure responses to separate and combined exposures seldom occurs naturally. It requires a carefully planned investigation, which depends on the full cooperation of management and workers. It can only be contemplated where there is clinical or laboratory evidence of a potentiating effect. The combined effect of a work exposure and personal habit, for example asbestos and smoking, can usually be investigated without much difficulty, as seen, because cohorts of workers with combined and separate exposures occur naturally.

New Health Problems

More effective prevention of work-related disease depends on identification of risks and then on eliminating or controlling causes which may be a combination of environmental, host and behavioural factors (see *Fig.* 1).

This broad approach to prevention can help to identify and control ill effects from exposures to physical, chemical and biological agents, but looking ahead our work force, with its electronic technology and abundant leisure, will face new health problems, which are likely to be mental, psychosocial and stress-induced. Occupational health has 'to enlarge its interests and adapt its skills' to deal with them (Morris, 1982).

Prevention of Disease which is not Directly Work-related

There are many opportunities for Occupational Health Services to undertake a wide range of preventive activities which are not directly concerned with work-related disease and injury. They include modification of personal habits, control of infection by immunization and screening for non-occupational disease (*Table X*). The reasons for doing this kind of preventive work are: first, it is not done routinely by the National Health Service (NHS) and may be done more effectively by occupational health services (OHS) which have access to the younger age groups, many of whom have no contact with the NHS; secondly, frequency of contact by OHS staff makes possible a developing programme of prevention, continuous follow-up and more effective intervention; and thirdly, the firm may derive economic benefits by reducing unnecessary sickness and disability.

Some of the most important opportunities for prevention are the elimination of the smoking habit, the modifying of the twentieth century diet and increasing the level of physical activity.

Table X. Opportunities for prevention of disease not directly work-related

Modification of personal habits
Smoking
Alcohol
Addictive drugs
Diet
Physical exercise
Control of infection (immunization)
Influenza
Rubella*
Screening for non-occupational disease
Women–breast and pelvic disease
Senior executives—life style etc.

*An occupational risk in female school teachers, hospital staff and nursery attendants of child-bearing age.

Smoking

The avoidance of smoking alone would probably reduce the mortality from all cancers by about a third; it would almost eliminate chronic obstructive lung disease and the complications of peripheral vascular disease and would probably reduce the mortality from myocardial infarction by about a quarter (Doll, 1983).

There is obvious scope for smoking cessation programmes at work. The TUC at its 1981 Annual Congress resolved 'to educate and inform its members about the hazards of smoking and to advise unions to negotiate "no smoking" areas in work places and time off to attend smoking withdrawal courses'. The Employment Medical Advisory Service (EMAS 18/82) supports action at the work place to encourage people not to smoke.

Some managements are lukewarm or even hostile to smoking cessation programmes. A recent survey was made of a random sample of 100 of the top 1000 companies according to *The Times* Listing (Harris and Seymour, 1983). Most (67 per cent) of the companies were manufacturing industries and by chance no tobacco company was included in the sample. The vast majority (94 per cent) of the companies had no formal smoking policy other than for safety reasons; 45 per cent could not see any advantages in adopting a policy against smoking in the work place; 86 per cent thought there would be disadvantages in that workers would not stand for it; it would be unpopular and infringe on the individual's freedom of choice. Fifty-six per cent of the companies employed full-time occupational health staff. It seems that they had little positive impact on their firm's attitude to smoking cessation programmes.

In another survey, most companies were reluctant to offer a cessation service for similar reasons and because they lacked designated staff, i.e. an occupational health service to set up a programme (Moreton and East, 1982).

Diet

The beneficial effects of modifications in the twentieth century diet on the incidence of hypertension, heart and cerebrovascular disease, diabetes and diseases of the large bowel are now generally recognized. They are not so widely accepted as the benefits of stopping smoking because the scientific evidence is less certain. A recent report for the National Advisory Committee on Nutrition Education by a group of experts under the chairmanship of Professor James (1983) recommends that as a nation we consume 10 per cent less fat, sugar, salt and alcohol, and 25 per cent more fibre in cereals, fruit and vegetables over the 1980s, with bigger changes in the long term, including a halving of sugar intake.

Exercise

Since the classic observation by Jerry Morris that bus conductors have less coronary heart disease than drivers (Morris et al., 1953), evidence has accumulated that a sedentary life increases the risk of myocardial infarction. It is now accepted that exercise is an antidote to CHD and possibly hypertension, as well as making people feel better and assisting in weight reduction.

Educational programmes encouraging people to stop smoking, to modify their diet and take more exercise are now being introduced at the work place by Local NHS Health Education Services with the support of occupational physicians and nurses.

A Heart Disease Prevention Project

An international trial was planned in four European countries to find out if risk factors in coronary heart disease can be reduced and to measure the effects of any reductions on the incidence of CHD (WHO European Collaborative Group, 1983). As part of this project, a randomized controlled trial was undertaken in 24 work places in the United Kingdom in collaboration with occupational physicians (Rose et al., 1980, 1983). Similar trials were made in Belgium, Italy and Poland. The population studied in the four countries comprised nearly 50 000 men aged 40–59.

In each country the work places were paired for size, region and nature of work. Within the pairs they were randomized to either:

an 'intervention' factory, where participants were screened and given advice on smoking, diet and exercise and treated for any hypertension detected; or

a 'control' factory in which there was screening of a 10 per cent sample of its members, but no intervention.

In the intervention factories the following treatment or advice was given: cholesterol-lowering diet (all subjects); cessation of cigarette smoking (smokers); daily physical exercise (sedentary workers); weight reduction (men 15 per cent or more overweight) and hypotensive drug therapy (men with raised systolic blood pressure above 160 mmHg). There was a general campaign to modify risk factors by posters, letters and group discussions. Individual advice was also given, particularly to high risk groups.

Table XI. Percentage change in overall risk factors and incidence of fatal and non-fatal CHD

	UK	*Belgium*	*Italy*	*Poland*
Overall risk factors (MLF)*	−3·9	−15·8	−28·2	−4·2
Change in CHD incidence	+5	−24	−14	−20

* Multiple logistic function which summarizes the overall impact of intervention on risk factor levels (WHO European Collaborative Group, 1980).

Taking account of changes in cholesterol levels, cigarette smoking, weight and systolic blood pressure, the overall reduction in risk factors in the 5–6 years of the trial was small in the United Kingdom compared with other countries (*Table XI*). Belgium and Italy achieved the best results, with commensurate changes in the incidence of CHD. Poland did no better than the UK for risk factor change in the whole study population, but did exceptionally well in the high risk group. There was a correlation between success in modifying people's life style and the amount of time given to health education. For example, in Italy the net fall in overall risk factors was 28 per cent among 3131 men, using six full time staff. In the UK the net fall was 4 per cent among 9734 men with an equivalent of two full time staff.

The results suggest that intervention can reduce the incidence of CHD and the size of the reduction is consistent with the degree of risk factor change and the effort put into making such changes.

Conclusion

Environmental control on its own will seldom be wholly successful, even in preventing work-related disease and injury. It has little to offer in preventing the much larger burden of non-occupational disease carried by the working community. While the work site is the obvious place for controlling work-related disease and injury, for which managements have a legal and moral responsibility, it is also an appropriate setting for a much broader approach to preventing unnecessary disease and disability. Successful prevention, like good treatment, needs resources—resources to encourage people through personal contacts and educational programmes to take a more positive approach to health and safety and to modify personal life styles. It depends on the support and cooperation of management and unions, backed up by competent occupational health services.

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REFERENCES

Amor A. J. (1960) One generation. *Transactions of the Association of Industrial Medical Officers* **10**, 74.

Axford A. T., McKerrow C. B., Parry Jones A. and Le Quesne P. M. (1976) Accidental exposure to isocyanate fumes in a group of firemen. *British Journal of Industrial Medicine* **33**, 65.

Department of Health and Social Security (1982) *On The State Of The Public Health for the Year 1981.* London HMSO.

Dingwall-Fordyce I. and Lane R. E. (1963) A follow up study of dead workers. *British Journal of Industrial Medicine* **20**, 313.

Doll R. (1983) Prospects for prevention. *British Medical Journal* **286**, 445.

Employment Medical Advisory Service (1978). *Occupational Health Services: The Way Ahead* London, HMSO.

Frazier T. M., Lalich N. R. and Pederson D. H. (1983) Use of computer generated maps in occupational hazard and mortality surveillance. *Scandinavian Journal of Work and Environmental Health* **9**, 148.

Gardner M. J., Acheson E. D. and Winter P. D. (1982a) Mortality from mesothelioma of the pleura during 1968 in England and Wales. *British Journal of Cancer* **46**, 81.

Gardner M. J., Winter P. D. and Acheson E. D. (1982b). Variations in cancer mortality among Local Authority areas in England and Wales: Relations with environmental factors and search for causes. *British Medical Journal* **284**, 784.

Gardner M. J., Winter P. D., Taylor C. and Acheson E. D. (1983) *An Atlas of Mortality in England and Wales (1968–78.) Vol I. Cancer.* Chichester, John Wiley & Co.

Harris J. and Seymour L. (1983) No smoking still not a sign of the times at work. *Occupational Health* **35**, 308.

Health & Safety Executive (1983) *Manufacturing and Service Industries, Health and Safety, 1981.* London, HMSO.

Hernberg Sven (1983) Use of epidemiology in occupational health. *Manual of Epidemiology.* Copenhagen, WHO (in the press).

Hoar S. K., Morrison A. S., Cole P. and Silverman D. T. (1983) An occupational and exposure linkage system for the study of occupational carcinogens. Medical Research Council.

Husman K. (1980) Symptoms of car painters with long term exposure to a mixture of organic solvents. *Scandinavian Journal of Work and Environmental Health* **6**, 19.

Institute of Occupational Health (1980) *Occupational Diseases in Finland in 1978–1979.* Helsinki, Reviews 5.

James W. P. T. (1983) *Proposals for National Guidelines for Health Education in Britain NACNE.* London, Health Education Council.

Kaloyanova-Simeonova F. (1977) *Pesticides: Toxic Action and Prevention.* Sofia, Bulgarian Academy of Science.

Lane R. E. (1949) The care of the lead worker. *British Journal of Industrial Medicine* **6**, 125.

Macbeth R. G. (1965) Malignant disease of the paranasal sinuses. *Journal of Laryngology and Otology* **79**, 592.

Medical Research Council (1983) *Job Exposure Matrices. Conference Report, Southampton, 1982.* Southampton General Hospital.

Meiklejohn A. (1957) *The Life Work and Times of Charles Turner Thackrah, Surgeon and Apothecary of Leeds (1795–1833).* Edinburgh, Livingstone.

Ministry of Agriculture of Fisheries and Food (1971) *Annual Report on Safety Health Welfare and Wages in Agriculture for 1970.* London, HMSO.

Moreton W. J. and East R. (1982) *Smoking cessation programmes in the work place.* London, Health Education Council.

Morris J. N., Heady J. A., Raffle P. A. B., Roberts C. G. and Parks J. W. (1953) Coronary heart disease and physical activity of work. *Lancet* **2**, 1053.

Morris J. N. (1975) *Uses of Epidemiology*, 3rd Ed. Edinburgh, Churchill Livingstone.

Morris J. N. (1982) Epidemiology and Prevention. Opening address, 9th Scientific Meeting, International Epidemiology Association, Edinburgh, 1981. *Millbank Memorial Fund Quarterly/Health & Society* **60**, 1.

Newhouse M. L. and Thomson H. (1965) Mesothelioma of the pleura and peritoneum following exposure to asbestos in the London area. *British Journal of Industrial Medicine* **22**, 261.

Rose G., Heller R. F., Tunstall P. H. and Christie D. G. S. (1980) Heart disease prevention project: a randomized control trial in industry. *British Medical Journal* **1**, 747.

Rose G., Tunstall P. H. and Heller R. F. (1983) UK heart disease prevention project: incidence and mortality results, *Lancet* **1**, 1062.

Royal Commission on Civil Liability (1978) *Statistics and Costings, Vol. 2, Cmnd. 7054.* London, HMSO.

The Royal Society (1983) Risk Assessment, a Study Group Report. London Royal Society.

Saracci R. (1981) Personal—environmental interactions in occupational epidemiology. In: McDonald J. C. (ed): *Recent Advances in Occupational Health* Edinburgh, Churchill Livingstone.

Seppalainen A. M., Husman K. and Martenson C. (1978) Neurophysiological effects of long term exposure to a mixture of organic solvents. *Scandinavian Journal of Work and Environmental Health* **4**, 304.

Statutory Instruments 1496 (1982) *Health & Safety: The Notification of New Substances Regulations, No. 1496.* London, HMSO.

Thackrah C. T. (1832) *The Effects of Arts, Trades and Professions and of Civil States and Habits of Living of Health and Longevity*, 2nd edition. London, Longmans, reprinted in Meiklejohn (1957).

Tiller J. R., Schilling R. S. F. and Morris J. N. (1968) Occupational toxic factor in mortality from coronary heart disease. *British Medical Journal* **4**, 407.

WHO European Collaborative Group (1980) Multifactorial trial in the prevention of coronary heart disease. 1. Recruitment and initial findings. *European Heart Journal* **1**, 73.

WHO European Collaborative Group (1983) 3. Incidence and mortality results, *European Heart Journal* **4**, 141.

WHO (1981) *Health effects from combined exposures.* Tech. Rep. Series, WHO, Geneva.

WHO (1983) *Recommended health based occupational exposure limits for selected vegetable dusts.* Tech. Rep. Series, no. 684, WHO, Geneva.

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