WORKING FOR A HEALTHY FUTURE

HISTORICAL RESEARCH REPORT

Research Report TM/79/20
1979

Medical aspects of the manufacture and use of Portland cement. A review of the literature

Fleetwood L, Soutar CA

HISTORICAL RESEARCH REPORT
Research Report TM/79/20
1979

Medical aspects of the manufacture and use of Portland cement. A review of the literature

Fleetwood L, Soutar CA

The scanning of this and the other historical reports in the Research Reports series was funded by a grant from the Wellcome Trust. The IOM's research reports are freely available for download as PDF files from our web site: http://www.iom-world.org/research/libraryentry.php

INSTITUTE OF OCCUPATIONAL MEDICINE
Research Avenue North, Riccarton, Edinburgh, EH14 4AP
Tel: +44 (0)870 850 5131 Fax: +44 (0)870 850 5132
e-mail publications@iomhq.org.uk

Report No. TM/79/20
UDC 613.62: 666.94

MEDICAL ASPECTS OF THE MANUFACTURE AND USE OF PORTLAND CEMENT

A Review of the Literature

L. Fleetwood
C.A. Soutar

December 1979

Report No. TM/79/20
UDC 613.62: 666.94

INSTITUTE OF OCCUPATIONAL MEDICINE

MEDICAL ASPECTS OF THE MANUFACTURE AND USE OF PORTLAND CEMENT

A Review of the Literature

by

L. Fleetwood
C.A. Soutar

Medical Branch,
Institute of Occupational Medicine,
Roxburgh Place,
Edinburgh EH8 9SU.

(Tel. 031-667-5131)

December 1979

CONTENTS

Report No. TM/79/20
UDC 613.62: 666.94

INSTITUTE OF OCCUPATIONAL MEDICINE

MEDICAL ASPECTS OF THE MANUFACTURE AND USE OF PORTLAND CEMENT

A Review of the Literature

by

L. Fleetwood and C.A. Soutar

SUMMARY

The published literature on the medical hazards of cement manufacture and use (not including the effects of asbestos cement) is principally concerned with effects on the respiratory system. A much smaller part is concerned with effects on the skin, and there are references to effects on hearing and the gastro-intestinal tract.

Critical appraisal of the literature on medical effects on the respiratory system reveals important defects in the methodology of most published work, principally a lack of adequate control groups and failure to allow for the effects of age and smoking habits. National differences of medical terminology, regional differences in composition of cement and probable differences in dust concentrations between plants compound the difficulties of interpretation of results.

Claimed effects of cement dust on the respiratory tract include rhinitis, abnormalities of the chest radiograph, abnormalities of lung function tests, symptoms such as chronic productive cough (chronic bronchitis), and frequent chest infections. Tentative conclusions which may be drawn from the literature are that exposure to cement dust is probably related to both rhinitis and an increase in chronic productive cough (though the effects of smoking habits have not been allowed for in most studies), but that cement is unlikely to cause a severe fibrotic pneumoconiosis (like silicosis). Reports of slight abnormalities of the chest radiograph in cement workers are sufficiently common to justify concern that exposure to cement dust may cause tissue changes in the lungs. It is not known whether these abnormalities are associated with abnormal lung function. Studies of lung function have generally been inadequately designed, and no definite effect of cement on lung function has been demonstrated.

Cement dermatitis (eczema) is well recognised, but its prevalence in cement workers has not been studied. It is usually associated with sensitivity to hexavalent chromium salts, which probably play an important part in causation. Nevertheless cement dermatitis may occur without chromate sensitivity, and the abrasive, alkaline nature of cement is probably also an important factor. Cement ulcers are an occasional complication of prolonged contact with wet cement.

No evidence was found for an increased risk of cancer as a result of exposure to cement dust, nor of diseases of the gastro-intestinal tract.

Doubt remains whether cement dust causes chronic productive

cough, abnormalities of the chest radiograph, or abnormalities of lung function tests, but these questions could be answered by a properly designed epidemiological study of current cement workers and workers who have left the industry, and comparison of symptoms, chest radiographic appearances and lung function measurements, with estimates of dust exposure, taking account of age and smoking habits.

CEMENT MANUFACTURE

Cement manufacture involves the quarrying and transporting of limestone, clay and other materials. These are crushed and calcined at about 1400°C and then allowed to cool. The clinker is ground, sieved and packed. Mixed with water and other substances, cement is used as a bonding and sealing agent. There are two main kinds of cement, natural and artificial. Artificial cements are chiefly of two kinds, Portland and aluminous. Special purpose cements are produced by the addition of substances which give resistance to heat or acid, or other desirable properties.

In modern cement plants, raw materials which are quarried from sources close to the plant are stored until used. These raw materials are blended in the correct proportions for the type of end product desired, and then crushed in roller mills. After crushing they are calcined and it is during this process that the cement is formed. The clinker is cooled. Grinding occurs in roller mills and the ground cement is then conducted to hoppers from which it may be packed in bags or loaded into tankers. In modern plants the handling of materials may be automated, and the plant managed as a closed circuit to prevent the escape of raw materials or finished product.

Components of raw materials and the cement dust are set out in Table 1.

TABLE 1

RAW MATERIALS:	Silicious and argillaceous composites.	
	Limestone.	
PROCESSES:	Raw materials:	quarrying - may involve blasting.
	" " :	transport.
	Crushing.	
	Clinkering.	
	Clinker grinding.	
	Cooling.	
	Packing.	
INTERMEDIATES:	None of significance.	CO_2, H_2O and small amounts of SO_3 evolve.
FUELS:	Gas, oil and coal : singly, or in combination.	

COMPOSITION OF CEMENT:

Portland		Aluminous	
Ca O	60 - 70%	$Al_2 O_3$	50%
$Si O_2$	19 - 24%	Ca O	40%
(free $Si O_2$	5%)	$Fe_2 O_3$	6%
$Al_3 O_3$	4 - 7%	$Si O_2$	4%
$Fe_2 O_3$	2 - 6%		
Mg O < 5%			

OTHER MATERIALS - may be included in cement because:

(1) they are locally available, or

(2) they confer special properties.

Quartz	up to 94%
Tuff (volcanic ash)	up to 35%
Soluble silicic acid	up to 20%
Diatomaceous earth	
Hexafluorosilicates	up to 4%
Blast furnace slag	
p - toluenesulphonyl chloride	10%
Barium Sulphate	70%

From: Occupational Health and Safety, International Labour Organisation (1974), pp 277-279.

LITERATURE ON MEDICAL ASPECTS OF EXPOSURE TO CEMENT DUST

INTRODUCTION

There is a large body of literature on medical aspects of cement manufacture and use. Writers in the USA, the USSR, Yugoslavia, Argentina, Egypt, India, Germany, and elsewhere have studied cement plants in their own countries over a period of time from 1887 to 1978. The earliest reference to cement workers we have found was in the analysed records of the Leipzig Local Sick Fund covering the years from 1887 to 1905.

This medical literature deals mainly with affections of the respiratory tract such as rhinitis, pneumoconiosis, bronchitis and emphysema. A smaller proportion is concerned with skin conditions and the remainder deals with a variety of other conditions. It was therefore thought appropriate to consider the literature under these main headings:

1. Respiratory tract - upper
2. Respiratory tract - lower
3. Gastro-intestinal tract
4. Skin
5. Hearing loss
6. Other systems
7. Morbidity and mortality data.

Effects of asbestos cement are not included in this review.

Dust inhaled into the lungs may be deposited on the walls of the airways, where by non-specific irritation or by chemical reaction they may cause cough, sputum production and occasionally narrowing of the airways leading to wheeze and breathlessness. A large number of dusts met with in industry may have these effects, which are sometimes described by the medical term 'chronic bronchitis'. Study of those symptoms is confused by their known associations with tobacco smoking, ageing and general urban atmospheric pollution. This review of the literature points out where proper account has been taken of those factors other than cement dust that are known to be associated with the development of such symptoms.

Some dust inhaled may reach the furthest parts of the lungs where exchange of gases takes place, the respiratory bronchioles and alveoli. The amount of dust that reaches this part of the lungs is determined by the particle size distribution of the airborne dust cloud. In general, only dust particles between about 0.5 and 7 μm in diameter reach the lung periphery. Dust cannot be removed as rapidly from here as from the airways and is dealt with by engulfment by phagocytic cells. This mechanism is slow and relatively inefficient at clearing large amounts of dust. Moreover it is dependent on the dust not being toxic to the phagocytic cell. In the case of asbestos, silica and coal, dusts may damage the cells and initiate a reaction that causes pulmonary fibrosis or scarring. This is called pneumoconiosis. Occasionally this dust retention in the lungs occurs without the development of fibrosis, although the radiographic appearances may be identical, and the pneumoconiosis is regarded as benign. Differentiation of benign from fibrotic pneumoconiosis is made by consideration of the progression of the condition, from the association of abnormalities of the measured function of the lung and by studying the pathological changes found in the lungs post-mortem. Any studies of pneumoconiosis during life should include a control population of unexposed individuals, as there is much variability in interpretation of radiographs and the reader of the radiograph is likely to be highly influenced by what he knows of the subjects' occupations. This literature review considers where studies have taken such precautions in studying pneumoconiosis or other diseases in cement workers and what pathological evidence there is for the existence of such conditions.

The literature studied covers a considerable period of time and its geographical origins are world-wide. Use and definitions of such terms as rhinitis, chronic bronchitis, emphysema, asthma, pneumoconiosis, and silicosis are unlikely to be identical in medical literature of such scattered origins, and in the papers themselves are often undefined. For instance, a diagnosis of 'chronic bronchitis' has been based variously on a history of productive cough, breathlessness, abnormality of lung function tests, or abnormality of the chest radiograph. In this review, therefore, emphasis is placed on the observations (if given) on which the authors based their diagnostic conclusions.

1. Cement and the upper respiratory tract

The upper respiratory tract consists of those portions of the respiratory tract lying between the external nares (the nostrils) and the tracheal bifurcation. It includes the nostrils, the accessory nasal sinuses, the nasopharynx, and the larynx. Rhinitis, rhinolithiasis, influenza, and a diminished sense of smell are among problems mentioned in the literature in connection with cement dust, and the papers dealing with these have been critically assessed.

VACCAREZZA (1950) studied 3,083 workers in Argentinian cement plants. He found that 6.8% had an encrustation of cement dust within the nasal passages, a condition he called rhinolithiasis, which was thought to be a form of rhinitis characteristic of cement workers. This was an uncontrolled study.

GIULIANI and BELLI (1955) undertook the clinical examination of 180 Italian cement workers and found that 72.3% had some disorder of the nasal mucosa, including acute and chronic hypertrophic or atrophic rhinitis. They did not state their diagnostic criteria, nor did they examine any controls.

THOMPSON *et al*. (1928) reported their findings in 274 American cement workers studied over a period of three years. By comparing sickness absence records of these workers with those of workers at a nearby rubber factory they found that the incidence of disease of the pharynx and tonsils was 3.2 times as high among cement workers. They also had 1.9 times as much disease of the nasal fossae, and 3.2 times as much influenzal illness as workers in the rubber factory. The cement workers worked a seven-day week, rubber workers a five-day week, and such differences must cast doubt on the validity of results based on sickness absence records.

GALLETTI *et al*. (1972a) studied 30 Sicilian cement plant operatives and found that eight of these (26.7%) had a 'diminished' sense of smell when their response to odorific substances was measured. They took no account of age, smoking habit, or medical history, did not examine the olfactory organs, and studied no controls.

1.1 Conclusion

The lack of properly controlled studies leaves the relationship between cement dust and rhinitis unclarified, although the high prevalence of disease in some studies suggests that cement dust probably contributes to rhinitis.

2. Cement and the lower respiratory tract

2.1 Pneumoconiosis - abnormalities of chest radiographs

PRODAN (1971) stated that 'Vaccarezza (1950) reported the first cases of severe pneumoconiosis among cement workers'. This statement has been referred to by individuals concerned about the health of cement workers; however, examination of the source reveals that the statement is incorrect. VACCAREZZA (1950) reported on 2,557 cement workers who were examined radiologically without discovering any cases of 'pulmonary fibrosis of silicotic type'. Signs of active or inactive tuberculosis were discovered in 210 (8.2%). Increased bronchovascular markings were found in the chest radiographs of 57 men (2.2%), but the significance of this finding is not clear, for no controls were studied.

THOMPSON et al. (1928) in their three-year study, X-rayed 53 cement workers (but no controls). In those early days, it may have been impractical to X-ray the whole factory population. Of 37 men who had been in the industry for three years or more, 15 showed evidence of pneumoconiosis as defined by Thompson's own criteria (Table 2). However, the authors stated that cement pneumoconiosis was of a benign type because it produced no symptoms and did not predispose to other disease.

GARDNER et al. (1939) studied 2,278 workers in cement manufacturing plants in the USA, and found no cases of advanced changes of the chest radiograph except in workers who had been exposed to silica dust in previous employments, or who were thought to have non-occupational medical conditions. Slight diffuse abnormalities of the chest radiograph were found in 17% of the workforce, but the authors could not relate prevalence to the duration of employment at the plant, and concluded that these appearances were the result of ageing. This study may be criticised in that workers who had left the factory were not examined, and it is possible that those workers staying at work in the plant for long periods were healthier than those who left, many of whom may have left for health reasons. The method of recording abnormalities of the chest radiograph, while conventional in 1939,

TABLE 2

CRITERIA FOR RADIOLOGIC DIAGNOSIS OF SILICOSIS

(THOMPSON *et al.* 1928)

1. Bilateral increased hilar shadows.
2. These shadows are generally reticular, least profuse in the periphery and the apices.
3. Diaphragmatic adhesions and densities in the lower lung fields, especially on the right.
4. No calcifications (except with tuberculosis).
5. No cavitation.
6. Shadows predominantly right middle and lower zones.
7. Tendency to beading along linear markings at junction of trunk shadows.
8. Fuzzy diaphragm.
9. Chest contour and heart shadow usually normal.

would not now be regarded as sufficiently precise for epidemiological studies.

SANDER (1958) reported the results of a re-examination of 195 workers who had been examined in 1934 and 1937 and since then had remained engaged in cement manufacture until 1957. Before discussing his findings he defined 'benign pneumoconiosis' as dust retention in the lungs without fibrosis, bronchial irritation, allergy, impairment of lung function or increased susceptibility to infection. 'Fibrotic pneumoconiosis' he defined as 'a fibrous tissue reaction to retained dust, of which silicosis and asbestosis are the leading examples'. He classified the chest X-rays of 120 men (61.5%) as 'within normal limits'; 68 men (35%) had 'moderate linear exaggeration'; six men (3%) had 'marked linear exaggeration with some ill-defined micronodulation', and two men had 'definite micronodulation'. Although he concluded that these changes were those of benign pneumoconiosis and not of silicosis, it is difficult to be certain that age and smoking habits had not contributed to the radiological appearances. No controls were studied, and lung function tests were not performed.

DOIG (1955) reported eight cases of disabling pneumoconiosis in limestone workers, diagnosed by chest radiographic appearances. However, these workers had also been employed in occupations in which exposure to quartz dust occurred, and it is likely that the changes were due to silicosis and not related to the limestone.

TARNOPOLJSKAYA and OSETINSKIJ (1957) stated that as a result of their long experience at the Ukraine Central Institute for Industrial Hygiene and Occupational Diseases, they were able to conclude that a localised and non-progressive form of pneumoconiosis occurs in a proportion of cement workers after long exposure to cement dust. They gave no epidemiological evidence in support of this statement.

HUBLET (1968) studied 478 cement workers. The X-rays of 253 (52.9%) of these showed nodular opacities of type p, m, or n. These were all workers who had been exposed for 25 years or more. There were no cases of progressive massive fibrosis (a severe form of pneumoconiosis) or of other lung disease. No controls were studied.

MALJTSEVA and TATANOV (1974) examined 1,247 workers radiologically and 721 clinically. Mild (category 1) pneumoconiosis was found in the chest radiographs of 0.62%. This arose after not less than eight years' exposure - typically after 20 years. No controls were studied.

JENNY et al. (1960) studied 488 workers in three Swiss cement factories. The X-rays of 97 workers taken nine months before the study started were examined. No cases of silicosis were found among them, and the chest radiographic appearances of the other 391 men were not reported.

POPOVIĆ (1964) studied 304 Serbian workers at two cement factories. Of these, 35.3% had asymptomatic pneumoconiosis (the radiological criteria used were not specified). None had silicosis - presumably by silicosis he meant pneumoconiosis with respiratory symptoms. His terms are not defined and no controls were examined.

KALAČIĆ (1973a) studied 847 cement workers from Split and found that 0.5% had 'X-ray signs of suspected fibrosis of the lungs'. No clarification of this statement was offered. None of 460 controls (shipyard workers from Split) had these appearances.

SCOTT (1973) in a review article states unequivocally that 'there are no health problems involved in the manufacture of cement'. He based this sweeping statement on the work of GARDNER et al. (1939) (see above); of DERVILLÉE and CARRERE (1935) who found that none of 550 men engaged in making or using cement showed any clinical respiratory changes on prolonged exposure to dust; and of PARMEGGIANI (1951) who said that no cases of radiological pneumoconiosis occurred in 533 workers making cement, but that silicosis was seen among the quarriers of the raw materials. Diagnostic criteria for these observations are not given.

SCANSETTI et al. (1975) compared cement workers with asbestos cement workers and asbestos workers. Presence of small opacities (ILO category 1/0 or greater), were commonly seen in asbestos and asbestos cement workers, but were also present in a substantial proportion of the cement workers (rounded opacities 18%, irregular opacities 33% of cases). Control workers not exposed to dust were not studied.

MAESTRELLI et al. (1979) reported an increased prevalence of small opacities in the chest radiographs of workers exposed to cement dust for long periods, compared to less exposed workers in the same factory. The ages and smoking habits of the groups were comparable, though these factors were not analysed in detail.

The report of H.M. Chief Inspector of Mines and Quarries (Department of Energy, 1975) stated that there were 539 new cases of pneumoconiosis in that year. The Digest of Pneumoconiosis Statistics (Health and Safety Executive, 1977) stated that there were 630 new cases in mining and quarrying, 183 in other notifiable industries and one in building. From these statistics it is clear that cement dust has not been recognised as a pneumoconiosis hazard.

2.2 Pneumoconiosis - pathology

CORTEZ PIMENTEL and PEIXOTO MENEZES (1978) demonstrated pulmonary (and hepatic) granulomata and fibrous scarring in one subject who had worked in a cement plant for 28 years. These granulomata were examined by X-ray diffraction, and patterns characteristic of Portland cement were detected within the granulomata. The significance of this report is uncertain; histological association does not necessarily imply causation, and no other report of pulmonary granulomata in response to cement dust has been published.

DOIG (1955) reported the lung pathology of a limestone worker who developed pneumoconiosis; the lung showed silicotic nodules, but this was more probably related to the nine years the man spent in coalmining than the nineteen years as a limestone miner.

2.3 Symptoms of respiratory disease

(a) Chronic cough and sputum and acute respiratory disease

THOMPSON *et al*. (1928) studied the sickness absence records of men at cement plants in the USA. Comparing these with the records of workers in nearby rubber factories they found that the frequency of all respiratory diseases was approximately twice as high among the cement workers. They used the terms 'influenza, grippe, bronchitis, pneumonia, and tuberculosis', but without specifying the diagnostic criteria for these diseases or the means by which their presence was detected. Nor were these diagnoses all made by the same individual. The cement plant workers worked a 7-day week, those in the rubber plant working a 5-day week. No account was taken of age or social class, although men in roughly similar age groups were compared.

VACCAREZZA (1950) stated that in the 3,083 Argentinian cement workers he studied, the incidence of bronchitis did not seem to be related to cement dust exposure, but varied with the climatic conditions of the different regions of the country in which the plants were situated. He did not state his diagnostic criteria for bronchitis, nor did he study any controls, measure dust levels, or give details of climatic conditions.

DURDA et al. (1976) studied 2,027 men in a cement factory where average total dust levels were 10 mg/m^3, the range extending up to 115 mg/m^3. Using the Medical Research Council criteria for chronic bronchitis (History of chronic cough and sputum, MRC 1965), they found 7.1% of non-smokers in dusty jobs for two years or more had chronic bronchitis, as against 1.9% of non-smokers in clerical jobs or in dusty jobs for less than two years. The first group had an average age of 39, the second 33, and while age and social class difference may have contributed to this difference in respiratory symptoms, an effect of cement dust seems likely.

GIULIANI and BELLI (1955) examined the sputum of 180 Southern Italian cement workers microscopically and found mucopurulent or frankly purulent sputum in 76.7% of cases. On clinical examination 83.3% were found to suffer from rhinitis, bronchitis, or emphysema. Diagnostic criteria were not stated, nor were controls studied. The diagnosis of emphysema was made radiologically, while rhinitis and bronchitis were diagnosed clinically. It is unhelpful to group these latter diseases together. No correlations between disease and smoking or age were attempted. No controls were studied.

JENNY et al. (1960) examined 488 cement workers in three Swiss factories. In reply to a questionnaire 202 (41%) said they had cough, 128 (26%) had cough and sputum. There was a relationship with smoking, 35% of those who smoked more than 20/day having cough and sputum, while 12% of non-smokers had these symptoms. Clinical bronchitis (moist and dry rhonchi audible through the stethoscope with cough and sputum) was found in 10 (2%) and clinical emphysema (undefined) in 6 (1.2%). They concluded that age for age the prevalence of bronchitis was not materially different among smokers from that quoted in the literature but that among non-smokers it was considerably higher. They themselves did not study any controls.

VYSKOČIL (1962) reported on a group of 104 cement workers seen in 1949 and followed up until 1964. In 1962 he reported that at the 1949 examination, 75 had a prolonged history of cough and on examination had signs of bronchitis (râles). This combination of symptoms and signs he called chronic bronchiolar catarrh. He was able to show a rise in the prevalence of chronic bronchitis which he diagnosed by a combination of clinical, radiological, and lung function criteria - in 1954 and again in 1959. No controls were studied, although figures taken from various authors who studied groups in different occupations were quoted. Dust counts were stated to range from 10 to 250 million particles per cubic metre. It was stated that 79% of smokers developed chronic bronchitis, versus 47% of non-smokers. No correlation with age was attempted. The numbers examined in 1954 and 1959 varied, nor were the same people examined on each occasion.

The same author (VYSKOČIL, 1968) reported findings in this group in 1964. At that stage 13 of the original group had died, five from pulmonary causes. Another 31 were not traced. The group of 60 who were examined in 1964 included 38 (63%) with chronic cough and sputum. The bronchitics were on average 4-6 years older and had worked an average of six years longer than the non-bronchitics. The cement workers were compared with four other groups consisting of one referred to as a 'control' group, and groups of street cleaners, miners with pneumoconiosis and miners without pneumoconiosis. The basis of selection of the 'control' group was not specified nor were the groups controlled for smoking habit. Details of their clinical assessment were not given. Among these groups, 'chronic bronchitis and emphysema' was less prevalent than among the cement workers.

VYSKOČIL's methods differed over the course of time. His diagnostic criteria altered between 1949 and 1964. The group studied was not followed up in its entirety, and no explanation was offered for the fact that more men were seen at subsequent than at earlier examinations. The percentage of the target group finally seen was small (58%) and no controls were studied over the same period. The prevalence of chronic bronchitis reported is high, but the methods of the study prevent satisfactory interpretation of the results.

POPOVIĆ (1964) examined 304 workers in two cement works in Serbia. Total dust concentrations in the air varied between 2,850 and 14,000 particles/cm³. On the basis of a detailed history he found dyspnoea (breathlessness) in 43.1%, cough in 26.8%, expectoration in 22% and 15.5% had chest pain. There were no controls and smoking habit was not taken into account.

KALAČIĆ (1973a) asked 847 cement workers and 460 controls matched for age in Yugoslavia to respond to the MRC questionnaire on smoking and chest disease. He found that major symptoms - cough, expectoration, exertional dyspnoea, wheezing - occurred significantly more frequently in cement workers than in controls. However, cement workers within each group studied tended to smoke more than controls. While smoking effects could explain part of the observed difference in the prevalence of symptoms between the two groups, they appear to be insufficient to account for all of it.

KARAJOVIĆ et al. (1959) examined 456 workers in Yugoslavia clinically and found 57 cases of 'chronic bronchitis' and 21 cases of 'chronic bronchitis with emphysema' - 17% in all. The authors did not state their diagnostic criteria or methods, nor were controls studied. Age and smoking habit were not reported.

MALJTSEVA and TATANOV (1974) reported an incidence of 'chronic bronchitis' of 9.5% in the group of cement workers studied. Their criteria were not stated, age and smoking habit were not taken into account, and no controls were studied.

KALAČIĆ (1973b) studied the yearly incidence of disability retirement of workers with chronic bronchitis, asthma or emphysema in the shipyard and in the cement plant at Split from 1959-1968. On average, 0.31% of shipyard workers but 1.35% of cement plant workers fell into those categories each year, so that the frequency of early retirement because of respiratory diseases was more than four times as high among cement workers as among shipyard workers.

In a recent study of cement workers in Italy, MAESTRELLI et al. (1979) reported a raised prevalence (35%) of chronic productive cough among workers continuously exposed to cement dust, when compared to workers in non-dusty occupations in the same factory (6%). Though dust exposures and smoking habits were not analysed in detail, these differences appeared, on the data given, not to be caused by differences in smoking habit or age.

(b) Asthma

The diagnosis of asthma is made on symptoms of episodic breathlessness, supported by lung function tests showing variable airflow obstruction.

THOMPSON *et al.* (1928) reported two cases of asthma among 570 cement workers examined. This is much lower than the expected prevalence of about 16-20 men.

TORSUEV *et al.* (1971) reported that 12% of workers exposed to dry cement for 10 years or more developed 'asthma or asthmoid bronchitis'. These terms were not defined. Of 40 subjects with this disease, 17 showed a 'bronchospastic reaction' to the inhalation of 1 g of cement dust. Such a response would be typical of the effects of inhalation of any dust in an asthmatic individual. Age and smoking habit were not considered and no controls were studied.

KARAJOVIĆ *et al.* (1959) found six cases of 'bronchial asthma' and seven of 'asthmoid bronchitis' (the terms were not defined) in 456 cement workers. The prevalence of asthma in this group was about the same as that in the general population. There were no controls.

JENNY *et al.* (1960) found that 3% of 488 workers had 'asthmoid bronchitis' - i.e. chronic productive cough, with increased FEV after the administration of a bronchodilator - and 0.6% had 'bronchial asthma', a history of asthmatic attacks.

TOURAINE *et al.* (1957) and BONHOMME (1975) reported single cases of asthma in cement workers. Neither had had any history of asthma before starting work in a cement plant. Both were sensitive to bichromate patch tests. However, Bonhomme's patient was also sensitive to microbial and fungal antigens, feathers and (unspecified) dusts, suggesting that this individual was atopic and therefore liable to develop asthma irrespective of occupational exposure to noxious dusts.

Thus asthma has been reported to occur in cement workers, but has not been demonstrated to be a consequence of exposure to cement dust; nor has its prevalence in cement workers been shown to be higher than in the general population.

2.4 Lung function studies

The function of the lungs can be assessed objectively by fairly simple, reproducible and non-invasive techniques. Measurement of the forced vital capacity (FVC: the size of a full breath, expired as rapidly as possible) and the expiratory volume in one second (FEV_1: the amount of the FVC expired during the first second of forced expiration) give an indication of the volume of the lung and, with other measures of expiratory flow rate (e.g. MMEF, MEF 50 or MEF 75) give an indication of bronchial disease such as may occur in chronic bronchitis or asthma.

VYSKOČIL (1962) reported on the lung function of cement workers in a 10-year follow-up study. One hundred and four men were seen originally, 53 persons after five years and 28 after ten years. Spirometry was performed to determine the maximal breathing capacity and the FEV_1. Although he stated that the group studied had significantly reduced lung function as compared with the normal population, he studied no controls and did not allow for the effects of smoking and previous occupation. No mention is made of those not followed up.

JENNY et al. (1960) studied 488 cement workers' vital capacity and maximal expiratory flow rate (MEFR). There was no relationship between duration of exposure to dust and reduction in vital capacity or MEFR. Smoking habit and age were not considered and there were no controls.

KALAČIĆ (1973a) reported that FEV_1/FVC ratio (reduction of which indicates bronchial disease) was significantly reduced in 847 cement workers compared with 460 controls. However, he did not standardize for age in comparisons between the two sets of men. Also, there were significantly more non-smokers (29.8%) in the control group than in the cement workers (23.3%). The proportion of light smokers was also significantly higher in the control group. However, he did find a significant difference in FEV_1/FVC ratio between non-smoking cement workers and non-smoking controls. Data on lung function of 47 cement factory workers and 20 controls is omitted. All these factors make his conclusions difficult to evaluate.

However, in a study of part of the same group in 1974, Kalačić studied 39 non-smoking cement workers and 23 controls. There were no significant differences with respect to age, height, FVC, FEV_1, FEV_1/FVC ratio, MEFR 200-1200, and MMEF between the two groups.

However, the difference between the MEF 50-75 (MEFR 200-1200, MMEF and MEF 50-75 are further measurements derived from the maximal expiratory flow/volume or volume/time traces) was significant at the 5% level. He concluded that this suggests either a reduction of elastic recoil or an increase in airways resistance, or both, in the lungs of the cement workers. He pointed out that coal dust and sulphur dioxide from the furnaces may play a role. Respirable dust particle counts ranged from 511/cc to 106,600/cc. The Yugoslav maximal permitted dust level is 450/cc.

ŠARIĆ and HOLETIĆ (1975) followed up some of the workers from KALAČIĆ's (1973a or b) study. They studied 37 cement workers, eight retired cement workers and 20 shipyard workers, all with the symptoms of chronic bronchitis. They found a fall over two years of controlled ventilatory indices in all three groups. However, FEV_1/FVC ratio was significantly reduced only in the cement workers; and when smoking habit was considered separately, cement worker smokers had significantly reduced FEV_1 and FVC values. This was not the case with the smoking controls. The numbers studied were small, the basis of the selection was not specified and there was no rigorous matching by age and smoking habit of cement workers and controls. The authors' conclusion, that exposure to cement dust may contribute to the development of airflow obstruction, is open to doubt.

ŠARIĆ et al. (1976) studied samples from the same population yet again. This time 160 active cement workers and 80 controls, selected on the basis of having or not having symptoms of chronic bronchitis (chronic productive cough) were investigated. In both groups there were significant falls in spirometric indices with time. These falls were greater in cement workers and controls than for a standard population and the losses in the cement workers were not significantly greater than in the controls.

RASMUSSEN et al. (1977) studied 301 cement factory workers, 449 blue collar workers, 218 white collar workers and 102 men who had been predominantly engaged in farming, forestry, or fishing. Each category was further subdivided by smoking habit. Subjects were weighed and measured, then interviewed about smoking habit, respiratory symptoms and recent upper respiratory tract disease. Spirometry was performed. Full occupational and residential histories were taken.

No significant differences in lung function between cement factory workers and other blue collar workers with comparable smoking habits could be demonstrated by use of the MMEF, FVC or FEV_1/FVC ratio when standardized for age, height, and weight. This was a thorough study, but did not take into account estimated or measured individual exposure to dust.

2.5 Other respiratory conditions

No published reports have linked any pulmonary cancers with cement dust, and there is no evidence that tuberculosis or other infections are more common in cement workers.

2.6 Conclusion

Much of the published work on the effects of cement dust on the lungs of workers in the industry suffers from grave methodological defects, principally lack of adequate control groups, and difficulties in allowing for the effects of age and smoking. It is rare for adequate exposure histories or details of the types of cement to be recorded. Nevertheless most radiographic studies, though uncontrolled, comment on the presence of small opacities in a proportion of subjects. While there is no evidence that advanced pulmonary fibrosis occurs, this suggests that a type of pneumoconiosis may occur in certain circumstances. This is supported in one instance only by pathological data, which suggests that the histological basis for this radiological change may be a pulmonary granulomatous reaction. Further studies are undoubtedly required to determine the relative effects of exposure, age and other disease in causing these shadows and to ascertain whether they are associated with impaired lung function.

It seems likely that exposure to cement dust has been related to the development of cough and sputum (chronic bronchitis), more frequent spells of sickness absence and premature retirement through ill health. Differences in smoking habits, and working shifts may contribute to these differences but are unlikely to be the whole explanation.

Most studies of lung function of cement workers have found abnormalities suggesting bronchial disease, but adequate control groups have been lacking. One adequate recent study (RASMUSSEN et al., 1977) in which controls were studied and smoking habit and age were taken into account (though individual dust exposures were not estimated) did not find any lung functional abnormality attributable to cement dust. It may be concluded that cement dust has not been proved to cause abnormalities of lung function tests. Again, any effect on the airways is likely to be related to the quantity of dust inhaled and this important information is missing from most publications. For this reason it is not possible to conclude from any one study that the results apply to the industry across the board, whatever the dust conditions. Studies of dust-disease interrelationships have not been reported.

3. Cement and the gastro-intestinal tract

Diseases of the gastro-intestinal tract occur frequently in the general population and have long been known to be associated with some foods, alcohol, smoking, and stress. No conclusions about gastro-intestinal disease in cement workers can be drawn unless a control population comparable in all respects is studied at the same time.

3.1 Animal work

KOLEV and SHUMKOV (1975) administered cement to mice by intra-tracheal and intraperitoneal instillation. This produced lesions of the peritoneum including ulcers, and papillomata in the bronchi, followed by peribronchial fibrosis.

EINBRODT and HENTSCHEL (1966) reported that cement dust given into the peritoneum of the rat has a sclerotic effect when its free silica content approaches 5%.

3.2 Gastroduodenal ulceration

JULLIEN *et al*. (1949) recorded their clinical impressions that there was an increased prevalence of gastroduodenal ulceration in building workers, but gave no evidence to support this impression. They reported that 4.8% (13 of 269) of workers in a cement factory in Nice had presented with symptoms subsequently confirmed as being from peptic ulcer, but did not say over what period. They compared this with the prevalence in a quite unsuitable control group, a hospital population in Paris. Some completely inadequate animal studies are described.

THOMPSON *et al*. (1928) showed that the frequency of digestive diseases as assessed from sickness absence records among men in a cement plant was two and a half times as high as that among rubber workers. Cement workers worked a seven-day week, rubber workers five days, and these different working conditions may have influenced sickness rates.

A review article by PRODAN (1971) is often quoted. While discussing the evidence for a raised incidence of digestive disorders, he refers only to the work of JULLIEN et al. (1949) mentioned above.

3.3 Other organs of the gastro-intestinal tract

ARABSKA et al. (1976) studied the mouths of 228 cement workers and 86 controls. They found that 84% of the first and 76% of the second group had poor oral hygiene. Of the cement workers, 98.3% had parodontopathies, vs 87.2% of the controls. Considering the abrasive nature of cement coupled with the oral hygiene of the group, their findings were unsurprising though their further significance is uncertain.

CORTEZ PIMENTEL and PEIXOTO MENEZES (1978) found hepatic granulomata at necropsy in the previously mentioned case. They were able to demonstrate the presence of Portland cement particles within these granulomata by X-ray diffraction.

GALLETTI et al. (1972a) examined 30 cement workers in Sicily and found 21 with 'hepatic dysfunction', seven with gastroduodenitis, one with gastritis, two with gastric ulcers. They did not define their terms, indicate how they diagnosed these disorders or study any controls.

4. Cement and the skin

4.1 Irritant properties of cement

Cement contains large quantities of calcium oxide and small amounts of K_2O, Na_2O and MgO. These alkali oxides when dissolved in water give wet cement an alkalinity of about pH12.5 (HANNUKSELA et al., 1976). This alkalinity is sufficient:

(a) to dissolve keratin, the protein of epidermal cells and other proteins;

(b) to saponify tissue fats (BIRMINGHAM, 1977).

These actions together may cause direct tissue destructions of the skin. In addition, the soaps formed will remove sebum, thus further diminishing the skin's natural defences.

Small crystalline and irregular amorphous bodies occur in cement. These can abrade the skin, enhancing the destructive effects of the alkali.

In addition, cement is hygroscopic and can cause cracking of the skin as it withdraws water from it. Once the skin has been thus attacked, it falls easy prey to fungal and bacterial infection.

These physical and chemical effects can lead to skin irritation ranging from erythema to deep ulceration.

4.2 Cement burns

In 1700 RAMAZZINI observed that bricklayers suffered from ulceration of the skin as a result of exposure to lime. In this century, MEHERIN and SCHOMAKER (1939) reported 60 cases of cement burns and ulcers seen over 18 months, and studied the progress of the burns histologically. They found that the average duration of disability was ten days. Ulcers tended to occur on the dorsum of the foot, the malleoli, popliteal fossae, wrists, hands and fingers, and at the site of friction of boots and gloves. The authors recommended frequent changes of clean, dry boots and gloves, and showers for men coming off a shift. MORRIS (1960) reported two cases of skin ulceration due to wet cement. One of these took three weeks to heal. No details of the length of exposure were given.

Superficial to deep ulceration occurring after prolonged intimate contact with wet cement has been reported by VICKERS and EDWARDS (1975); HANNUKSELA et al. (1976) and BANDMANN and AGATHOS (1977). Healing took from 2-15 weeks. The mechanism was thought to be a direct toxic and irritant effect of the cement. Five patients were tested for sensitivity to chrome: none was sensitive.

TORSUEV et al. (1971) reported on the frequent occurrence of dermatitis and cement burns in vertical drifters whose job was the lining of shafts in coal mines.

4.3 Cement dermatitis

The occurrence of dermatitis has long been recognised in cement workers, and was a common occurrence in men building the London Underground Railway system (O'DONOVAN, 1925). It is thought to be clinically distinguishable from dermatitis of other varieties (WHITE, 1934). Characteristically it involves the backs of the hands and feet, and webs of the fingers. The palm is usually spared, and the nails become thickened and dystrophic. Furunculosis and fissuring occur.

A strikingly high prevalence of allergy to dichromate salts (hexavalent salts), demonstrated by patch tests, in workers with cement dermatitis, was originally observed by JAEGER and PELLONI (1950), and this finding has largely been confirmed by many studies, for example BURROWS and CALNAN (1965), JOVOVIĆ et al. (1971) and MEZZADRA et al. (1974). Patch testing with trichromate salts, as opposed to dichromate (hexavalent) salts, gives largely negative results (SOYINKA, 1977; JOVOVIĆ et al., 1971). Testing with extracts of whole cement has given variable results (PERONE et al., 1974; TORSUEV et al., 1971; KARAJOVIĆ et al., 1959), and is said to be non-reproducible (CALNAN, 1960).

The proportions of patients with cement dermatitis who have been reported to have positive patch tests to dichromate salts range between 60% and 94%. All authors agree that it is a high percentage, and that cases without demonstrable allergy occur. The prevalence of positive tests in cement workers without dermatitis or with other clinical forms of dermatitis is of the order of 5 to 12%.

Positive patch tests to cobalt occur with lower frequency in workers with cement dermatitis (FREGERT and GRUVBERGER, 1978).

Cement contains small quantities of hexavalent chromium salts which may be extracted with solutions of appropriate salts (CALNAN, 1960; POSSICK, 1970; FREGERT and GRUVBERGER, 1972; PERONE et al., 1974). There appears to be more hexavalent chromium in the finished cement than in the raw materials from which it is made, and the hexavalent chromium is probably derived from conversion of trivalent chromium in the raw materials, and from the chromium steel alloy of which the steel balls in the crushing mills are made (CALNAN, 1960; FREGERT and GRUVBERGER, 1972).

It is probable that chromate sensitivity is an important mechanism in the causation of cement dermatitis, though the absence of chromate sensitivity in a small proportion of cases indicates that other factors are of importance. In particular the abrasiveness and alkalinity of cement are likely to be important in causation and maintenance of the condition.

Exact figures for the prevalence of cement dermatitis are not available, but it appears to be a considerable problem. RUSSELL (1933) found that 7% of the sickness absences in a cement works were attributed to skin diseases. In Great Britain nearly 200,000 days of work are lost through 'eczema and dermatitis' in the building trade (BURROWS and CALNAN, 1965.

The prognosis of cement dermatitis appears to be poor. The condition often persists even after a change of occupation (BURROWS and CALNAN, 1965). Prevention is best achieved by meticulous protection, and careful cleaning of skin and clothing. The use of barrier creams which reduces hexavalent to trivalent chromium has been found useful (BURROWS and CALNAN, 1965), and the addition of iron sulphate to cement, to reduce the available chromate, has been recommended (FREGERT et al., 1979).

5. Hearing loss

The human ear is able to detect sounds of frequencies between 20 Hz and 20 kHz in early adulthood. Hearing acuity gradually diminishes so that a greater intensity of sound is required to produce a response at any given frequency, and the response at some frequencies may disappear altogether (HENDERSON et al., 1977). The hearing loss associated with ageing is called presbycusia.

Speech perception is not linearly related to auditory acuity and persons with a large degree of hearing loss are able to understand speech because of the high redundancy of speech signals. Hearing loss must therefore be measured audiometrically.

Various authors state that the noise levels in a cement plant, particularly in the vicinity of the rolling mills, are very high, perhaps up to 115 dB (A). The recommended maximum sustained level is 90 dB (A). However, only one study of hearing loss in a cement plant, that of GALLETTI et al. (1972b) was discovered in a search of the literature.

6. Other conditions

Conjunctivitis and blepharitis have been mentioned in the literature as occurring in cement factory workers, although no studies have been done. FRANKE and HARMS (1972) reported one case of a worker who observed a clinkering oven without protective goggles and sustained a cataract as a result.

7. Mortality and morbidity studies

Long-term medical hazards to workers in any industry may be assessed by comparing the mortality and morbidity of those workers with others who match them in age, sex, social status, habits and genetics but differ from them in occupation. Statistics of morbidity and mortality of workers in various industries have been compiled from time to time, those regarding miners being particularly good.

However, statistics on workers in the cement industry are not kept separately in this nor, as far as we are able to discover, in any other country.

CONCLUSIONS

A review of the international literature on the medical effects of cement dust reveals grave defects in the scientific methodology of much published work, principally the selection of unsuitable or inadequate control groups and failure to allow for the effects of age, smoking and general atmospheric pollution. The difficulties of drawing conclusions from these papers are compounded by international differences in medical terminology, or regional differences in the composition of cement, and likely differences in factory conditions, between old and new factory plant. Medical studies of cement workers in the United Kingdom are lacking.

The literature concentrates on the possible effects of cement dust on the respiratory tract, and to a lesser extent on the skin. In spite of the largely unsatisfactory nature of this literature, some tentative conclusions may be drawn. It is unlikely that cement dust causes a severe form of pneumoconiosis such as the extensive fibrosis (scarring) of silicosis, for in none of the large epidemiological surveys have such abnormalities been found. However, minor changes in the chest radiograph have been reported in many surveys, and although age and smoking habits have usually not been taken properly into account, these abnormalities have been reported so commonly that it is likely that cement dust does cause minor changes in the appearances of the chest radiograph. The clinical significance of these changes, and any associated lung function changes are not known.

Rhinitis has been reported so commonly among cement workers that it is very likely to be a hazard of exposure to the dust. Increased prevalence of chronic cough and sputum (chronic bronchitis) has also been reported frequently in cement workers, and although the effects of age, smoking and local general atmospheric pollution have often not been adequately taken into account, nevertheless the frequency with which an excess of chronic bronchitis has been reported in cement workers suggests that it is indeed a hazard of the inhalation of cement dust.

Cement dust has not been proved to cause abnormalities of lung function tests, nor has the converse been proven. Many poorly controlled studies have claimed lung function abnormalities in cement workers. One well-controlled study in which cement workers were not shown to have worse lung function than other outdoor workers failed to take account of individual dust exposures, so that dust/disease relationship could not be explored except very crudely.

The international medical literature, though open to many criticisms, suggests that lung disease may be a consequence of the inhalation of cement dust, and the question of whether cement workers in the United Kingdom suffer from increased illness as a result of their work can only be answered by studies in this country, taking account of local materials and factory conditions. There is a need for well planned medical epidemiological studies of men exposed to cement dust, with adequate control groups and allowance made for the effects of age and smoking. Measurements of dust levels and recording of detailed occupational histories would permit comparisons between medical indices and estimates of exposure to cement dust to distinguish the effects of cement dust from other effects not related to occupation.

Wet cement may certainly cause damage and ulceration of the skin after prolonged contact, probably a direct effect of its alkalinity and abrasiveness. Cement dermatitis is well recognised clinically, but information on its prevalence and the disability it may cause in cement workers is lacking.

Cement has not been shown to cause malignant disease or peptic ulceration.

ACKNOWLEDGMENTS

This work was carried out with the aid of financial support from the Cement Makers' Federation.

REFERENCES

ARABSKA, B., OSTROWSKA, H. and ZABINSKA, O. (1976). Parodontopathies in cement workers. Czasopismo Stomatologiczne, 29 (1), 73-76.

BANDMANN, VON H.-J. and AGATHOS, M. (1977). Toxic ulcerative contact dermatitis from precast concrete. Berufsdermatosen, 25 (3), 108-117.

BIRMINGHAM, D.J. (1977). Dermatoses. Occupational Diseases, 79-83. U.S. Department of Health, Education and Welfare, June 1977.

BONHOMME, A. (1975). Cement Asthma. Ouest Médical, 28 (7), 399-402.

BURROWS, D. and CALNAN, C.D. (1965). Cement Dermatitis. Transaction of St. John's Hosp. Derm. Soc., London, 51, 27-39.

CALNAN, C.D. (1960). Cement Dermatitis. Journal of Occupational Medicine, 2, 15-22.

CORTEZ PIMENTEL, J. and PEIXOTO MENEZES, A. (1978). Pulmonary and hepatic granulomatous disorders due to the inhalation of cement and mica dusts. Thorax, 33, 219-227.

DEPARTMENT OF ENERGY (1975). Report of H.M. Chief Inspector of Mines and Quarries for 1974. H.M.S.O., London.

DERVILLÉE, P. and CARRERE, P. (1935). Results of an investigation into occupational diseases of cement workers in the Department of Gironde. La Medecine du Travail, 7 (6), 244-247.

DOIG, A.T. (1955). Disabling pneumoconiosis from limestone dust. British Journal of Industrial Medicine, 12, 206-216.

DURDA, M., ZIEMECKA, E., SZAFRANSKI, W. (1976). Air pollution in a cement mill. Bulletin of the International Union against Tuberculosis, 51, 631-634.

EINBRODT, H.J. and HENTSCHEL, D. (1966). Experiments on animals with dust from the working environment of a blast furnace cement works. Internationalis Archiar für Gewerbepathologie in Gewerbehygiene, 22 (4), 354-366.

FRANKE, VON W. and HARMS, H.-H. (1972). Cataract from heat radiation in the cement industry. Arbeitsmedizin Sozialmedizin Arbeitshygiene, 11, 331.

FREGERT, S. and GRUVBERGER, B. (1972). Chemical properties of cement. Berufsdermatosen, 20, 238-248.

FREGERT, S. and GRUVBERGER, B. (1978). Solubility of cobalt in cement. Contact Dermatitis, 4, 14-18.

FREGERT, S., GRUVBERGER, B. and SANDAHL, E. (1979). Reduction of chromate in cement by iron sulphate. Contact Dermatitis, 5, 39-42.

GALLETTI, C., TANZARIELLO, R. and TANZARIELLO, A. (1972a). Occupational olfactory disorders in cement workers. La Clinica O.R.L., 2, 117-125.

GALLETTI, C., TANZARIELLO, R. and TANZARIELLO, A. (1972b). Occupational deafness in cement workers. La Clinica O.R.L., 2, 139-155.

GARDNER, L.U., DURKAN, T.M., BRUMFIEL, D.M. and SAMPSON, H.L. (1939). Survey in seventeen cement plants of atmospheric dusts and their effects upon the lungs of two thousand two hundred employees. Journal of Industrial Hygiene and Toxicology, 21 (7), 279-318.

GIULIANI, V. and BELLI, R. (1955). On the occupational risks of cement workers. La Medicina del Lavoro, 46 (12), 715-724.

HANNUKSELA, M., SUHONEN, R. and KARVONEN, J. (1976). Caustic ulcers caused by cement. British Journal of Dermatology, 95, 547-549.

HEALTH & SAFETY EXECUTIVE (1977). Digest of Pneumoconiosis Statistics 1975. H.M.S.O., London.

HENDERSON, T.L., DUNN, D.E., NOZZA, R.J. and WASSERMAN, D. (1977). Oscillatory vibrations. In: Key, M.M. et al. (Editors): Occupational diseases. A guide to their recognition. DHEW Publication No. 77-181. U.S. Department of Health, Education and Welfare. pp 508-520.

HUBLET, P. (1968). The relative risk of pneumoconiosis in cement manufacture. Archives Belges de Médecine Sociale Hygiène, Médecine du Travail et Médecine Legale, 26, 417-430.

JAEGER, H. and PELLONI, E. (1950). Tests épicutanés aux bichromates positifs dans l'eczéma au ciment. Dermatologica, 100: 207-216.

JENNY, VON M., BATTIG, K., HORISBERGER, B., HAVAS, L. and GRANDJEAN, E. (1960). Occupational medical investigation in cement factories. Schweizerische Medizinische Wochenschrift, 90, 705-709.

JOVOVIĆ, D., CVIJETIĆ-SMODLAKA, O. and ANDREJEVIĆ, L. (1971). The role of cobalt in cement eczema. Medicinski Glasnik, 10, 349-352.

JULLIEN, G., LEANDRI, M. and BOUVEYRON, J. (1949). A new occupational disease of cement: gastroduodenal ulcers. Archives des Malades Professionnelles du Médecine du Travail et de Sécurite Sociale, 10, 612-619.

KALAČIĆ, I. (1973a). Chronic nonspecific lung disease in cement workers. Archives of Environmental Health, 26, 78-83.

KALAČIĆ, I. (1973b). Ventilatory lung function in cement workers. Archives of Environmental Health, 26, 84-85.

KALAČIĆ, I. (1974). Early detection of expiratory airflow obstruction in cement workers. Archives of Environmental Health, 29, 147-149.

KARAJOVIĆ, D., DANILOVIĆ, V., VERBIĆ, N., DORDEVIĆ, V., POPOVIĆ, D., MILOSAVLJEVIĆ, Z., DORDEVIĆ, S., SLAVKOVIĆ, V., SAVIĆ, D. and MALESEVIĆ, L. (1959). Investigation into allergy in cement workers. Acta Medica Iugoslavia, 7, 339-345.

KOLEV, K. and SHUMKOV, G. (1975). Biological effects of cement dust on intraperitoneal and intratracheal tests. Probl. Khig., 1, 111-118.

MAESTRELLI, P., SIMONATO, L., BARTOLUCCI, G.B., GEMIGNANI, C., MAFFESSANTI, M.M. (1979). Pneumoconiosis and chronic bronchitis in a group of cement workers. La Medicina del Lavoro, 70 (3), 195-202.

MALJTSEVA, L.M. and TATANOV, Y.A. (1974). Occupational respiratory disease in workers at a modern cement plant. Gigiena Truda I Professional nye Zabolevaniya, 3, 14-17.

MEHERIN, J.M., SCHOMAKER, T.P. (1939). The cement burn: its etiology, pathology, and treatment. Journal of the American Medical Association, 112, 1322-1326.

MEZZADRA, G., RANDAZZO, S.D. and TERMINELLO, V. (1974). Studies on cutaneous sensitivity to cement. Italian General Review of Dermatology, 11 (2), 103-109.

MORRIS, G.E. (1960). The primary irritant nature of cement. First published report of cement ulcers. Archives of Environmental Health, 1, 301.

O'DONOVAN, W.J. (1925). Lime dermatitis. Lancet, 1, 599-602.

PARMEGGIANI, L. (1951). Remarks on pneumoconiosis statistics in the cement industry. Rossegna di Medicina Industriale, 20, 400-409.

PERONE, V.B., MOFFITT, Jr. A.E., POSSICK, P.A., KEY, M.M., DANZINGER, S.J. and GELLIN, G.A. (1974). The chromium, cobalt, and nickel contents of American cement and their relationship to cement dermatitis. American Industrial Hygiene Association Journal, 301-306.

POPOVIĆ, D. (1964). The incidence of pneumoconiosis among workers in the cement industry. Arhiv za Higijenu Rada, 15, 353-376.

POSSICK, P.A. (1970). Cement dermatitis. Cutis, 5, 167-170.

PRODAN, L. (1971). Cement. In: Encyclopaedia of Occupational Health and Safety, Vol. 1, 277-279, I.L.O., Geneva.

RAMAZZINI, B. (1713). De Morbis Artificum Diatriba, Geneva. Translated by W.C. Wright, 1940, University of Chicago Press, Chicago.

RASMUSSEN, F.V., BORCHSENIUS, L., HOLSTEIN, B. and SOLVSTEEN, P. (1977). Lung function and long-term exposure to cement dust. Scandinavian Journal of Respiratory Diseases, 58, 252-264.

RUSSELL, A.E. (1933). The Effect of Cement Dust upon Workers. American Journal of Medical Science, 185 (3), 330-338.

SANDER, O.A. (1958). Roentgen Resurvey of cement workers. AMA Archives of Industrial Health, 17, 96-103.

ŠARIĆ, M. and HOLETIĆ, A. (1975). A two year follow-up study of forced expiratory volumes in a group of cement workers with chronic bronchitis. Arhiv za Higijenu Rada, 27, 119-122.

ŠARIĆ, M., KALAČIĆ, I. and HOLETIĆ, A. (1976). Follow-up of ventilatory lung function in a group of cement workers. British Journal of Industrial Medicine, 33, 18-24.

SCANSETTI, G., COSCIA, G.C., PISANI, W. and RUBINO, G.F. (1975). Cement, asbestos, and cement-asbestos pneumoconioses. Archives of Environmental Health, 30, 272-275.

SCOTT, G.A. (1973). Observations on the origin and effects of the industrial dusts of East Antrim. Ulster Medical Journal, 42 (2), 153-167.

SOYINKA, F. (1977). Epidemiology of occupational dermatosis of builders in Nigeria. Berufsdermatosen, 25, 195-202.

TARNOPOLJSKAYA and OSETINSKIJ (1957). The effects of cement dust on the respiratory organs. Soviet Medicine, 21, 90-94.

THOMPSON, L.R., BRUNDAGE, D.K., RUSSELL, A.E. and BLOOMFIELD, J.J. (1928). The health of workers in dusty trades - I. health of workers in a Portland cement plant. U.S. Public Health Service, Washington, D.C.

TORSUEV, N.A., LYUBOMUDROV, V.E., GURKOVSKAYA, S.M., GUBERGRITS, E.A. and MAZURIN, A.P. (1971). Occupational dermatoses in drifters of vertical shafts of coal mines. Vestnik Dermatologii i Venerologii, 45 (12), 58-60.

TOURAINE, R.G., SOUBRIER, R. and TOLOG, F. (1957). Concomitant asthma and eczema caused by cement. Archives des Malades Professionelles, 18, 282-284.

VACCAREZZA, R.A. (1950). Hygiene and Health in the Portland cement industry. Investigation in the Argentine Factories, Buenos Aires: Guillermo Kraft Ltda.

VICKERS, H.R. and EDWARDS, D.H. (1975). Cement Burns. Contact Dermatitis, 2, 73-78.

VYSKOČIL, J. (1962). The problem of chronic bronchitis in Czech. cement workers. Review of Czechoslovak Medicine, 8 (1), 38-52.

VYSKOČIL, J. (1968). Long-term follow-up of chronic bronchitis in hydraulic cement workers. Vnitrni lékařstvi, 14 (4), 341-348.

WHITE, R.P. (1934). The Dermatoses. H.K. Lewis, London.

GLOSSARY

Airflow obstruction:	Increased airways resistance, i.e. difficulty in moving air forcibly in or out of the lungs - measured by lung function tests.
Airway:	Conducting tubes in the lung (bronchi, bronchioles), leading to the air spaces (alveoli) where gas exchange takes place.
Airways resistance:	Resistance to airflow in the airways, usually caused by narrowing.
Allergy:	Altered biological reactivity to a substance as a result of exposure to it.
Alveoli:	Small (1 mm diameter) air spaces, of which the lung is largely composed, in which gas exchange between air and blood takes place.
Antigen:	Protein or other large molecule which can stimulate allergy.
Apices:	Tops of the lungs.
Asthma:	Intermittent wheeze and breathlessness caused by variable narrowing of the airways: is sometimes a response to an inhaled substance.
Atrophic:	Thin.
Blepharitis:	Inflammation of the eyelids.
Bronchodilator:	Drug which relaxes the muscle of the bronchi, used in the treatment of asthma. A substantial response to a bronchodilator implies the presence of asthma.
Broncho-vascular markings:	The lines and spots on the chest radiograph caused by the normal anatomy of the lung: 'increased broncho-vascular markings' implies abnormality, e.g. pneumoconiosis. (Includes 'linear exaggeration'.)
Calcification:	Dense radiographic shadows caused by deposition of calcium in tissues: occurs in such diseases as tuberculosis and asbestos-induced lung disease.
Cataract:	Opacity of the lens of the eye, obscuring vision.

Chest radiograph:	Chest X-ray.
Chronic bronchitis:	Productive cough for three months in a year for three consecutive years or more: this usage of the term does not necessarily include breathlessness or other disability.
Conjunctivitis:	Inflammation of the surface of the eye.
Control group:	Group of individuals to act as standard of comparison: ideally should be identical in all respects to the study group except for the variables under investigation.
Dermatitis:	Inflammation of the skin, manifested by redness, roughening, cracking and weeping: same as eczema.
Dorsum:	Upper surface (of the foot).
Dyspnoea:	Breathlessness.
Dystrophic:	Growing badly: (of nails), thickened and irregular.
Eczema:	Inflammation of the skin, manifested by redness, roughening, cracking and weeping: same as dermatitis.
Elastic recoil:	Elasticity of the lungs which helps in expiration.
Emphysema:	Dilation and destruction of the air spaces in the lung, causing breathlessness. May be suspected on the results of chest radiograph or breathing tests, confirmed pathologically. This term is extensively misused in medical and lay literature.
Epidemiology: (epidemiological)	Study of large number of individuals ('population').
Fibrosis: (fibrotic)	Process of healing by scarring. In the lung this may occur in response to an inhaled dust (amongst other causes), and leads in some degree to reduced lung function and breathlessness.

Forced expiratory volume in one second (FEV_1):	The amount of the forced vital capacity expired during the first second of forced expiration.
Forced vital capacity (FVC):	The size of a full breath, expired as rapidly as possible.
Gastritis:	Inflammation of the stomach.
Granulomata:	A small area of inflammation of specific appearance visible microscopically.
Hepatic:	Of the liver.
Hilar shadows:	The shadows in the chest radiograph caused by the great vessels and bronchi in the roots of the lungs.
Hypertrophic:	Thickened and abundant.
Incidence: (of disease)	Number of individuals developing a disease over a period of time (attack rate).
Intraperitoneal:	Into the sac containing the gut.
Lung function tests:	Breathing tests.
Malleoli:	Ankle bones.
Micronodulation:	Small spots (in the chest radiograph).
Morbidity:	Illness or disability rate.
Mortality:	Death rate.
MRC:	Medical Research Council.
Mucosa:	Lining of mouth, nose, air passages, stomach, etc.
Mucropurulent:	Partly infected (refers to mucus or sputum).
Nasal fossae:	Sinuses.
Nodular opacities:	Small spots (in the chest radiograph).
Non-specific:	In this context means non-allergic, that is, the biological response is as strong on first exposure as on subsequent exposures (e.g. response to high or low pH, or abrasive material).
Olfactory:	Of the sense of smell (nose and sinuses).
Papillomata:	Benign tumours.
Parodontopathy:	Disease around the teeth (sockets).

Patch testing:	Substance is applied to skin under a patch.
Peribronchial fibrosis:	Scarring round the air passages.
Phagocytes: (phagocytic)	Cells which engulf and attempt to digest foreign particles.
Pneumoconiosis:	Disease of the lungs caused by inhalation of inorganic dust, manifested by abnormality of the chest radiograph: underlying pathology ranges from aggregations of dust in the lungs without significant tissue changes or abnormal function, to severe scarring reaction, with loss of function and breathlessness.
Popliteal fossa:	Back of the knee.
Presbycusia:	Loss of hearing as a result of ageing.
Prevalence: (of disease)	Number of individuals suffering from a disease at any one time.
Progressive massive fibrosis:	Areas of very severe scarring (fibrosis) in the lung in response to inhaled dust.
Purulent:	Infected (refers to mucus or sputum).
Radiologically:	By X-ray.
Râles:	Crackling sounds heard through the stethoscope.
Respirable dust:	Dust of a size which can be inhaled into the alveoli of the lungs (up to 7 μ diameter).
Reticular:	Of a lace-like pattern.
Rhinitis:	Inflammation of the nose manifested by symptoms of blockage, watering or sneezing, or redness or swelling of the lining of the nose.
Rhinolithiasis:	Stones in the nose.
Rhonchi:	Wheezes heard through the stethoscope.
Sclerotic:	Causing fibrosis.
Sebum:	The natural oil of the skin.
Sensitivity:	Allergy: i.e. altered reactivity as a result of exposure.
Silicosis:	The pneumoconiosis caused by silica (quartz): this type involves severe damage to the lung.
Silicotic nodules:	Areas of scarring and fibrosis seen in the lungs in response to inhaled silica (quartz).
Sinuses:	Accessory air spaces communicating with the nose.

Spirometry:	Measurement of FVC, FEV_1, and other measurements derived from the forced expiration of a maximum breath (e.g. MEFR, FEV_1/FVC ratio, MEFR 200-1200, MMEF, MEF 50-75).
Tracheal bifurcation:	The point where the main air passage from the larynx (trachea) divides into two tubes, each passing to one lung.
Ulcer:	Punched-out hole in surface.
Ventilatory: (ventilation)	Of spirometry.
Wheeze:	Musical noises from the lungs heard at the mouth or through the stethoscope.

HEAD OFFICE:

Research Avenue North,
Riccarton,
Edinburgh, EH14 4AP,
United Kingdom
Telephone: +44 (0)870 850 5131
Facsimile: +44 (0)870 850 5132

Tapton Park Innovation Centre,
Brimington Road, Tapton,
Chesterfield, Derbyshire, S41 0TZ,
United Kingdom
Telephone: +44 (0)1246 557866
Facsimile: +44 (0)1246 551212

Research House Business Centre,
Fraser Road,
Perivale, Middlesex, UB6 7AQ,
United Kingdom
Telephone: +44 (0)208 537 3491/2
Facsimile: +44 (0)208 537 3493

Brookside Business Park,
Cold Meece,
Stone, Staffs, ST15 0RZ,
United Kingdom
Telephone: +44 (0)1785 764810
Facsimile: +44 (0)1785 764811

Email: iom@iom-world.org