

WATCH/2005/3

WATCH COMMITTEE

Portland cement

Annex 1

Portland Cement Dust: Hazard Assessment (2004)

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GLOSSARY

ATS	American Thoracic Society
CI	Confidence interval
COPD	Chronic obstructive lung disease
FEF	Forced expiratory flow
FEV1	Forced expiratory volume in one second
FVC	Forced vital capacity
MRC	Medical Research Council (UK)
MVV	Maximum voluntary ventilation
OR	Odds ratio
PEFR	Peak expiratory flow rate
RR	Relative risk
SD	Standard deviation of the mean
SE	Standard error of the mean
SIR	Standardised incidence ratio
SMR	Standardised mortality ratio
TWA	Time weighted average
VC	Vital capacity
WHO	World Health Organization

1. SUMMARY

This hazard assessment updates the health effects sections of an earlier HSE review of Portland cement (HSE 1994). The latter is taken to represent an accurate assessment of the literature available up to 1991. This assessment concentrates on the evidence for non-malignant respiratory disease and carcinogenicity. The primary literature on skin sensitisation has not been reviewed because the potential for cement to cause such reactions, due to the presence of hexavalent chromium, has been established by other reviews. Toxicological endpoints such as reproductive toxicity and mutagenicity have not been considered because there are no data.

Concerning non-malignant respiratory disease, HSE (1994) noted evidence that repeated exposure to Portland cement has produced chronic bronchitis and impaired pulmonary function, but firm conclusions could not be drawn because of limitations of the available studies. The effects of long-term exposure remain poorly studied, particularly because there are no longitudinal prospective studies in workers exposed to cement. However, many of the 15 recent studies, all conducted outside of the UK, provide evidence of the presence of respiratory problems among cement factory workers, consistent with the earlier literature. Overall, the pattern of evidence indicates that cement dust has the potential to cause chronic productive cough and an obstructive impairment of pulmonary function. It is not possible to draw reliable conclusions on dose-response relationships, but the available data do raise concerns that respiratory deficits could develop with long-term average exposures to respirable cement dust above 1 but below 4 mg.m⁻³.

HSE (1994) concluded that there was no convincing evidence for an increased incidence of any site-specific cancer resulting from cement exposure, but acknowledged that the data available at that time were not consistently and reassuringly negative. Since the early 1990s, a number of relevant cancer cohort and case control studies have been published, conducted in groups exposed to cement either through employment in cement factories or in the construction industry. Together with the earlier studies, these have identified associations between occupational exposure to cement and cancers at several specific sites: the stomach, lungs, colon, pharynx and larynx. However, taking account of strength and consistency observed associations and limitations of the available studies, it is concluded that a causal association between Portland cement exposure and cancer has not been established.

2. INTRODUCTION

This hazard assessment document updates the health effects sections of an earlier HSE review of Portland cement (HSE 1994). The latter is taken to represent an accurate assessment of the literature available up to 1991. Consequently this hazard assessment concentrates mainly on literature published after this time, although certain pre-1991 studies are considered in some detail where they help with the interpretation of the recent literature. A copy of the earlier HSE (1994) review is provided as Annex 2 to the WATCH/2005/03.

There were two reasons for producing this updating document. Firstly, work-related chronic obstructive pulmonary disease (COPD) is a priority within HSE's Occupational Respiratory Disease (ORD) sub-programme. The evidence-base for this sub-programme indicates that occupational exposure to dusts in general leads to an increased risk of COPD. Cement dust is a particularly alkaline and irritant dust, and might therefore be considered to pose a greater risk of respiratory tract damage than many other poorly soluble dusts (generically often referred to as "low toxicity dusts"). The HSE (1994) review described evidence for an increased risk of chronic bronchitis and impairment of pulmonary function (consistent with COPD) in cement dust exposed workers, but there was a lack of data on dose-response relationships. Hence, one reason for undertaking this updating assessment was to see whether the scientific literature on cement dust published over the last 10 years is now better able to characterise the risks of COPD, and consequently to help determine whether or not cement dust should be regarded as a particular priority for action within HSE's ORD sub-programme.

In addition, it has been drawn to the attention of HSE that in recent years a number of case-control studies have been published suggesting that exposure to cement dust may be a cause of laryngeal and pharyngeal cancer. It was felt important to examine the evidence from these studies particularly as the earlier HSE review of Portland cement (HSE 1994) noted that the evidence for carcinogenicity with cement dust was not reassuringly negative, albeit largely based on concerns for stomach cancer. Given the widespread occupational exposure to cement dust, it was felt important that HSE develop an updated position in relation to carcinogenicity.

Annex 1 provides a detailed explanation of the composition and manufacture of Portland cement. In brief, Portland cement is produced by crushing and grinding calcareous materials (e.g. limestone, chalk) and argillaceous materials (e.g. clay, shale), as a wet slurry or in a dry state. The mixture is calcined in a rotary kiln and the resulting clinker is finely ground. The product is composed largely of calcium silicates, aluminates, and alumino-ferrites. Other constituents are then added, depending on the desired concrete properties.

Hexavalent chromium (Cr (VI)) can be present in cement. The sources of chromium are in the raw materials, refractory bricks in the kiln and chromium

steel grinders. Because of concerns about Cr (VI) sensitisation (see section 2.3), during recent years several countries have introduced measures to limit the amount of Cr (VI) in cement products. This can be achieved by the addition of ferrous sulphate to cement, which transforms Cr (VI) to form Cr (III) compounds. In 2003 the European Commission introduced new legislation to restrict the marketing and use throughout the EU of cement products containing soluble Cr (VI) at a concentration of more than 2 ppm.

This document focuses mainly on the evidence for non-malignant respiratory disease and carcinogenicity. Studies published between 1990 and 2004 were identified from a literature search using TOXLINE and PubMed, or from report reference lists. All relevant reports have been critically appraised. The primary literature on skin sensitisation was not reviewed because the hazardous properties of cement in relation to this adverse health effect had been established in the pre-1991 literature, and the primary literature has been the subject of two very recent critical reviews (summarised in section 3.3). Other toxicological endpoints such as reproductive toxicity and mutagenicity have not been considered.

Information on the adverse health effects of cement dust comes from observations of its effects in humans. As noted in the earlier HSE (1994) review, animal data are extremely limited in terms of their scope and quality and do not provide any useful information.

3. THE HEALTH EFFECTS LITERATURE

3.1 Effects of single exposure

HSE (1994) reported that there were no data on the effects of a single exposure to cement by relevant routes of exposure.

Only one recent study looking at short-term effects of cement exposure was identified. Changes in pulmonary function during a work shift were investigated in workers at three Portland cement factories in Saudi Arabia (Ali et al. 1998). From the payrolls, 150 exposed male workers were selected randomly for the study, in a manner that ensured that all production areas were represented. Their mean age (\pm SE) was 38.0 ± 0.8 years. As an unexposed control group, 355 male workers were randomly chosen from among administrative staff in the cement, ammonia, petrochemical and asbestos industries. The control group was slightly younger, with a mean age of 34.3 ± 0.5 , and their ethnicity was Arab (68%), Indian subcontinent (22%) and South East Asia (9%). Personal information was collected by questionnaire. The proportion of current smokers, ex-smokers and non-smokers was similar in the control and exposed groups. Respirable dust was measured using a gravimetric method for a total of 97 samples collected from various production areas; the report does not state the sampling time or whether these were personal or static samples. Spirometry was conducted immediately before and after the morning shift. The highest values from three acceptable expirations for FEV₁, FVC and FEF_{25-75%} were recorded and the FEV₁/FVC ratio was calculated.

The geometric mean concentration (\pm SD) of respirable dust ranged from 20 ± 1 mg.m⁻³ in the quarry to 7 ± 2 mg.m⁻³ in the kiln area. Spirometry data were reported as mean cross-shift changes. The spirometry readings did not appear to have been adjusted for height. Reductions in FEV₁, FEV₁/FVC ratio and FEF_{25-75%} occurring during the workshift were significantly greater in the exposed workers. For example, the mean (\pm SE) reduction in FEV₁ was 0.022 ± 0.008 l for the control group, compared with 0.077 ± 0.011 l for the exposed group. For FEV₁/FVC the mean reduction was $0.011 \pm 0.165\%$ and $1.316 \pm 0.319\%$ for the control and exposed group. Regression analysis demonstrated no relationship between cross-shift pulmonary changes and age or smoking status.

The results of this study suggest that bronchoconstriction had occurred during the workshift for the cement workers, who were exposed to relatively high dust concentrations. The health significance of this effect is uncertain.

3.2 Irritation

HSE (1994) concluded that cement has irritant and corrosive properties, especially when wet, towards the skin. Also, it was concluded that due to its alkalinity, moistened cement would also produce eye irritation.

As these properties of cement are well established, no further evaluation of the literature has been conducted for these adverse health effects in the present review.

Portland cement is generally considered to be a respiratory tract irritant, although there are no data on this aspect in the scientific literature.

3.3 Sensitisation

HSE (1994) stated, based on a review of the pre-1979 literature (Fleetwood and Soutar 1979), 'that there have been many cases of cement dermatitis appearing to arise as a result of skin sensitisation to hexavalent chromium (Cr(VI)) in cement'. Concerns for sensitisation relate in the main to contact with the wet cement.

Recently the National Institute of Occupational Health, Norway (NIOH, 2003) conducted a detailed critical review of the literature, to address these questions:

1. Does Cr(VI) in wet cement cause allergic dermatitis (skin sensitisation) in construction workers?
2. Is there any causal association between the reduction of Cr(VI) in cement and the occurrence of allergic dermatitis?

Twenty four papers published between 1975 and 2001 were systematically reviewed. These were selected as studies of chromate allergy in humans, especially those involving construction workers, which included the conduct of patch tests for chromate allergy. Most of the investigations were cross-sectional studies based on patients attending dermatology clinics, in the general population, or in groups of construction workers. Some studies provided limited comparative information on the prevalence of Cr(VI) sensitisation in groups of workers exposed to cement with 'high' and 'low' (less than 2 ppm) Cr(VI) content, conducted in countries where the practice of reducing the Cr(VI) content of cement by the addition of ferrous sulphate has been introduced.

All the studies reviewed were considered by NIOH to have significant limitations, notably the absence of exposure characterisation, problems with the chromate allergy diagnosis, subject selection bias and the lack of prospective follow-up. Nevertheless, the reviewers identified a consistently high prevalence of Cr(VI) allergy in studies of construction workers. Also, in general population studies, the prevalence of allergy was found to be higher in construction workers than in other occupational groups. Furthermore, analyses of patients attending dermatology clinics with a diagnosis of chromate allergy revealed a high proportion of patients with occupations involving contact with cement.

In view of the consistency and strength of the associations it was concluded that, despite their limitations, the available studies supported the hypothesis of a causal relationship between Cr(VI) in wet cement and allergic contact dermatitis in construction workers.

In answer to the second question, it was concluded that there is insufficient information to decide if there is a causal association between the reduction of Cr(VI) in cement and a reduced occurrence of allergic dermatitis. However, the information that is currently available supports such a relationship. Furthermore, it is biologically plausible that a reduction in exposure to Cr(VI) will lead to a lower risk of allergic contact dermatitis.

Recently the European Commission's Scientific Committee on Toxicology, Ecotoxicology and the Environment (SCTEE, now the Scientific Committee on Health and Environmental Risks) expressed an opinion of the risks to health from Cr(VI) in cement (European Commission, 2002). On the basis of a selective review of the published literature, SCTEE concluded that Cr(VI) compounds in cement induce sensitisation and cause serious allergic reactions in construction workers. Also they concluded that water-soluble Cr(VI) compounds penetrate the skin to a much greater extent than the less water-soluble Cr (III) compounds, and that Cr(VI) readily penetrates wet leather gloves. SCTEE also concluded that, based on studies in Denmark (Avenstorp, 1989a,b, 1991, 1992), a reduction of Cr(VI) compounds in cement to less than 2 ppm will reduce the prevalence of allergic cement eczema in workers, and will reduce the risk of becoming sensitised to chromate. They also emphasised that education and the use of personal protective measures would also contribute to a lower frequency of skin sensitisation due to Cr(VI).

Subsequently the European Commission introduced new legislation, through an amendment (2003/53/EC) to the Marketing and Use Directive (76/769/EEC), to restrict the marketing and use of cement and cement containing preparations where the level of soluble Cr(VI) is more than 2 ppm.

Overall, it can be concluded that contact with wet cement can cause sensitisation reactions to Cr(VI) in the cement and, though not proven, is likely that reducing the Cr(VI) content of cement will reduce the risks of Cr(VI) sensitisation reactions in workers who have contact with cement.

3.4 Effects of repeated exposure to cement dust

3.4.1 Recent studies of non-malignant respiratory disease in workers with long-term exposure to cement dust

HSE (1994) noted evidence that repeated exposure of humans to Portland cement has produced rhinitis, chronic productive cough (chronic bronchitis), slight abnormalities on chest radiography and impaired pulmonary function. However, it was considered that the quality of studies indicating adverse effects on pulmonary function was such that the evidence for a true effect of cement dust is inconclusive. From the limited exposure data available, and from subjective descriptions of working conditions, the respiratory effects appeared to be associated with very dusty working conditions. Although no firm conclusion could be drawn about the dose-response relationship for the respiratory effects, attention was drawn to several studies which reported no significant effects associated with airborne concentrations of cement dust of 2.9 mg.m⁻³ total dust and 0.57 mg.m⁻³ respirable dust (geometric means), up to 5 mg.m⁻³ total dust and 1.1 mg.m⁻³ respirable dust, or 2-7.8 mg.m⁻³ total dust and 1-2.2 mg.m⁻³ respirable dust.

The literature search conducted in 2004 identified a number of recently published cross sectional studies, together with one retrospective cohort study, of non-malignant respiratory disease conducted in workers at cement production plants. In 10 studies, pulmonary function testing was conducted and information on the presence of self-reported respiratory symptoms was usually collected. Pulmonary function testing was by spirometry, except for one study that used a peak flow meter. One cross sectional study investigated workers using chest radiography. A further 4 studies in cement production workers looked only at self reported respiratory symptoms. All these 15 studies are appraised below, and summarised in Table 1.

Two general health surveys of populations of construction workers are available (Petersen and Zelling 1998 and Arndt et al. 1996) but these studies do not provide any information on health that can be related to cement dust exposure so these studies were not included in this review.

3.4.1.1 Retrospective cohort study, which included pulmonary function testing

Fell et al. (2003) conducted a retrospective cohort study in 119 Norwegian cement factory workers. A respiratory questionnaire was administered and spirometry was conducted. Current dust level measurements were available and a semi-quantitative cumulative exposure matrix was developed. The study showed no differences in the respiratory health of the cement workers exposed to average respirable dust levels of at least 1 mg.m⁻³ when compared with matched blue-collar controls

In a recent retrospective cohort study, pulmonary function and the presence of respiratory symptoms were investigated in a group of Norwegian cement factory workers (Fell et al. 2003). The investigation was conducted in 1998 and 1999. The study population comprised men born between 1918 and 1938 who had worked at a particular cement-producing factory for at least one year; presumably most of the study population were retired. A total of 226 workers fulfilling these criteria were identified, of which 148 were still alive. No information was available on the cause of death of those who had died. After exclusions due to white collar work, workplace exposures to paint or welding fume, medical conditions unrelated to work that would affect ability to undergo spirometry testing, refusal to participate or not being contactable, 119 workers were available for the study. Only 4 workers refused to participate. They had a mean age of 69 years, and a mean of 21.8 years exposure to cement dust. As controls, a group of 94 men born between 1918 and 1938 and who had worked at an ammonia plant for at least one year were identified; 63 were still alive at the time of the study. The ammonia plant was located about 10 km from the cement plant. It used closed processes, so the workers were not exposed to ammonia. None of the control workers had ever experienced exposure to ammonia at levels sufficient to cause symptoms of sensory irritation. After exclusions due to refusal to participate or not being contactable, 50 controls, of mean age 67 years were available for investigation. The percentage of deceased workers in the cement workers and controls was similar, at about 35%. The proportion of current smokers was slightly higher among the cement workers (35%) than the controls (26%).

Previous dust exposures for particular work processes were estimated from documented information on changes in work practice and from detailed interviews with a subgroup of workers. Four levels of historical exposure were established, and assigned semiquantitative exposure numbers. Cumulative exposure indices were calculated for each worker for an exposure-response analysis. Contemporary dust exposure was measured by personal sampling, over 8 hour periods on three days of work; 20 person-related measurements were conducted. Silica levels were measured in the three samples showing the highest amounts of dust. Information on respiratory symptoms was gathered, using a Norwegian modification of the MRC questionnaire, answered at home. Also, information on job history, cement dust exposure level estimates, asbestos exposure and smoking habits were gathered. Spirometry was conducted in accordance with ATS guidelines by an investigator who was only 'partially' blinded to exposure status. A diagnosis of COPD was made in subjects with a history of chronic cough, phlegm when coughing, breathlessness and/or wheezing and also with FEV₁/FVC ratio of less than 70%.

In the contemporary workplace air samples the mean concentration for total dust was 7.4 mg.m^{-3} (range $0.4 - 53.7 \text{ mg.m}^{-3}$) and for respirable dust 0.91 mg.m^{-3} (all respirable dust measurements were below the Norwegian exposure limit of 5 mg.m^{-3}). Silica was not detected in two samples and was 0.06 mg.m^{-3} in the third sample, below the local limit. The prevalence of respiratory symptoms was slightly higher in the cement workers, and positively associated with cumulative exposure index for three of the symptoms; however, there were no statistically significant differences between the control and exposed workers. There were no significant differences for the spirometry parameters, after adjustment for age, height, tobacco use and asbestos exposure. For the controls and exposed workers, FVC was 86 and 89% of predicted (according to age, height and gender), FEV₁ was 85 and 90% of predicted and FEV₁/FVC ratio was 73 and 74%, respectively. Among the cement workers the spirometry results were similar for the workers within the highest and lowest cumulative exposure quartiles. The prevalence of COPD was 14% for controls and 14.3% for exposed workers.

Overall, this study showed no differences in the respiratory health of surviving workers exposed to cement plant dust and a comparable group of non-exposed workers. This study included an assessment of workers who had left the cement plant, so the potential for selection bias due to the exclusion of individuals who may have left the cement plant because of work related ill-health was reduced. However, no information on the causes of death among the 35% of workers who were deceased was available so the possibility of selection bias resulting from the exclusion of individuals who died of respiratory disease cannot be excluded. Nevertheless, the percentage of deaths in the two groups was similar suggesting that if selection bias was present it was minimal. A weakness of the semi-quantitative dose-response assessment is that this relied on estimates of cumulative exposure in the absence of historical dust measurement. Inaccuracies in the exposure estimation would result in a tendency for the masking any exposure-response relationship, if present.

One limitation of this study is that it is not possible to draw firm conclusions about the maximum airborne concentration of cement plant dust that would not be associated with adverse respiratory health because of the absence historical quantitative exposure measurements. Nevertheless, if it is assumed that historical exposures were at least as high as contemporary exposures, the study provides reassurance that the control of cement dust levels to around 1 mg.m^{-3} (respirable dust) will provide protection against adverse effects on respiratory health.

3.4.1.2 Cross sectional studies which included pulmonary testing

Mwaiselage et al. (2004) studied 115 Tanzanian cement factory workers. Pulmonary function was investigated by spirometry and peak flow measurement. Current dust exposure measurements were available, from which cumulative exposures were estimated. FVC, FEV₁, FVC/FEV₁ and PEF were reduced for the exposed workers in comparison with controls. The risk of developing a notable obstructive impairment,

represented by a FEV₁/FVC ratio <0.7, was significantly increased for cumulative dust exposures in excess of 300 mg.m⁻³year.

In a recent study, pulmonary function was investigated in male workers at Tanzanian Portland cement factory (Mwaiselage et al. 2004). The exposed study population was all 126 production workers at the plant. A control group was made up of all 88 maintenance workers and 32 randomly selected administrative workers. After exclusions because of refusal to participate (about 4%), unsatisfactory spirometry performance, physician diagnosed asthma or the presence of other diseases that could affect pulmonary function, 115 exposed subjects and 102 controls were available for the final analysis. The mean duration of employment for the exposed group was 12 years and for the controls 15 years. Information on personal characteristics, smoking, use of personal protective equipment and occupational history were obtained by standardised questionnaire. Spirometry was conducted during the morning, and to ATS guidelines. PEFr was measured by Vitalograph peak flow meter. FEV₁, FVC and PEFr were expressed as absolute values and as % of predicted with reference to age and height adjusted values for the male Tanzanian population. A contemporary dust exposure assessment was based on 120 full-shift personal samples of total dust, collected from 52 exposed and 28 control subjects. Dust exposures were expressed for each occupational group as a cumulative mean, calculated as the product of the geometric mean concentration and the number of years worked in the particular group.

Smoking was more prevalent in the exposed group, with 32% being ever-smokers, compared with 25% among controls. Mean cumulative dust exposure was 11 mg.m⁻³year (range 0.3-38) for the controls and 69 mg.m⁻³ year (range 3-926) for the exposed group. Approximately 30% of production and maintenance works reported wearing disposable protective face masks regularly, although the quality and effectiveness of these masks could not be determined. The report stated that there was no correlation between face mask use and lung function, but no data were presented. Among the exposed workers, mean cumulative exposure was highest for crane operators (378 mg.m⁻³ year, 13 workers), packers (230 mg.m⁻³year, 30 workers) and crushers (158 mg.m⁻³ year, 18 workers) and lowest for raw mill operators (21 mg.m⁻³ year, 17 workers).

FEV₁ for the exposed group (mean ± SD, 82% ± 15) was lower than for controls (99% ± 12). Similarly, FEV₁/FVC for the exposed group (0.77 ± 0.1) was lower than for controls (0.87 ± 0.1), as was PEFr (80% ± 5 vs. 88% ± 7). These differences were statistically significant. Similar differences were observed for comparisons between ever smokers of the exposed and control groups (after adjusting for number of packs smoked) and never smokers of the exposed and control groups. Linear regression analysis, taking account of height, age and smoking, showed a negative correlation between cumulative dust exposure and the following lung function parameters: FEV₁, FVC, FEV₁/FVC ratio and PEF.

An analysis was conducted of persons with FEV₁/FVC ratio <0.7, considered by the authors to have 'airflow limitation'. It was found that the % of workers

with FEV₁/FVC ratio <0.7 was greatest in the occupational groups with the highest cumulative exposure, which were crane operatives (23%), packers (47%) and crushers (28%), as compared with raw mill operators (7%) and controls (6%). OR relating to FEV₁/FVC ratio <0.7 for occupational groups in the cumulative exposure band 100-300 mg.m⁻³ year was 1.6 (95% CI 0.5-5.1) and for >300 mg.m⁻³ year was 9.9 (95% CI 3.5-27.8), after adjustment for age and smoking.

To conclude, this study demonstrated reduced lung function in cement production workers in comparison with a control group with relatively low dust exposure. A strength of this study is that a meaningful analysis for dose-response relationships was possible because subgroups with very contrasting dust exposure levels were identified. This analysis showed a negative correlation between measurements of lung function and total dust exposure levels, and that the risk of developing a notable obstructive impairment (i.e. represented by a FEV₁/FVC ratio <0.7) was significantly increased for cumulative total dust exposures in excess of 300 mg.m⁻³ year. A weakness of this study is that only contemporary exposure data were available and so the reliability of the cumulative exposure estimates is uncertain. The authors stated that conditions at the factory and production capacity had changed little since opening in 1965, and they considered it was reasonable to assume that dust exposure levels had remained constant, but questions still remain. The report did not mention whether workers changed jobs within the factory; if this did occur, then cumulative exposure group misclassification is possible, which would have the effect of masking a dose response relationship. Overall, this study provided evidence of an obstructive impairment associated with cumulative cement exposures likely to be in excess 300 mg.m⁻³ year.

Laraqui Hossini et al. (2002) investigated the respiratory health of 280 Moroccan cement factory workers by respiratory questionnaire and spirometry. No dust exposure measurements were taken, but levels were described as being relatively high. There was a higher prevalence of respiratory symptoms and minor spirometry deficits among the cement workers, compared with controls.

Pulmonary function and the presence of respiratory symptoms were investigated in workers at a Moroccan cement factory (Laraqui Hossini et al. 2002). The study population was 280 workers exposed to cement dust, of mean (\pm SD) age 41 \pm 6 years and duration of employment 14 \pm 4 years. As a control group, 73 non-exposed administrative workers from the same factory were recruited, of mean age and duration of employment of 37 \pm 7 and 13 \pm 4 years, respectively. Recruitment methods were not described. All exposed and control subjects had been employed at the factory for at least 2 years, and none were found with respiratory or cardiac symptoms at a pre-employment medical examination. The proportion of current, ex- and non-smokers in the two groups was similar. Demographic information and medical histories were obtained using a standardised questionnaire based on those of the MRC and WHO. Chronic bronchitis was defined as productive cough for at least 3 months of the year, for at least 2 years. Respiratory function was assessed by spirometry, using standard methods. The highest values from three expirations for FVC and FEV₁ were recorded and FEV₁/FVC ratio was calculated. Workers with deficits were graded as having an 'alveolar', slight

(FEV₁ 71-80% of predicted), moderate (FEV₁ 61-70% of predicted), serious (FEV₁ 41-60% of predicted) or severe (FEV₁ below 40% of predicted) deficit according to an official French government grading system. Cement plant dust exposure for the exposed group was graded in a semi-quantitative manner as low, moderate and high, depending on the job within the plant.

Dust concentration in the workplace was not measured, but in the crushing shop, production plant and packing areas the concentration was such that vision was impeded and dust was deposited on hair and clothing. The prevalence of respiratory symptoms was higher among the exposed workers, with 65% reporting symptoms compared with 35% of controls. Most common symptoms were coughing, phlegm, dyspnoea and rhinitis. Approximately 30% of exposed workers were considered to have chronic bronchitis compared to 10% of controls, with the prevalence being greatest for workers with the highest dust exposure, those employed for more than 10 years, and among current and ex-smokers. Abnormal spirometry results were found in 22% of exposed workers compared with 14% of controls, although most (78%) abnormalities were graded as an 'alveolar' or slight deficit. There was no clear relationship between the level of exposure and spirometry results.

This study shows an increased prevalence of respiratory symptoms and deficits in spirometry in cement exposed workers in comparison with the control group. A key weakness of this study is the lack of quantitative exposure data. Also, the cement dust exposed group were compared to administrative workers, who may have a different respiratory health status irrespective of the influence of cement exposure.

Meo et al. (2002) conducted spirometry in 50 Pakistani cement mill workers. No information on dust exposure levels was available. FVC and FEV₁ were reduced and FEV₁/FVC ratio was increased for the cement workers, in comparison with matched controls.

Pulmonary function was investigated in a group of 50 male workers from a Karachi cement mill (Meo et al. 2002). The study subjects were volunteers, selected at random. They were described as apparently healthy, aged 20 - 60 years (mean 36.9), and had been exposed to cement dust for a mean of 13 years. The control group comprised 50 healthy males, selected from the local Karachi population. They were matched with the exposed group for age, height, weight and socio-economic status. Subjects with a range of medical conditions or who smoked or abused drugs were not included in the study. Spirometry was conducted in accordance with ATS guidelines. The highest values from three expirations of the following parameters were recorded: FVC, FEV₁, FEV₁/FCV ratio, PEF and FEF_{25-75%} and MVV.

Mean FVC and FEV₁ were significantly reduced in the group of cement workers. For the controls and cement workers FVC (mean ± SE) was 4.03 ± 0.09 and 3.18 ± 0.11 litres and FEV₁ was 3.04 ± 0.08 and 2.54 ± 0.10 litres, respectively. FEV₁/FCV ratio was significantly increased for the cement workers; the ratios were 75.8 ± 1.4 and 81.5 ± 2.3% in controls and cement workers, respectively. PEF and MVV were significantly lower in the cement workers, but FEF_{25-75%} was similar to the controls.

The reductions in FVC and FEV₁ accompanied by an increase in FEV₁/FVC ratio seen in the cement workers are consistent with the presence of a restrictive impairment of lung function. Overall, this study suggests that long-term exposure to cement dust can cause an impaired respiratory function, consistent with the previous study. However, a limitation of this study is that the exposed and control subject selection procedures were not adequately described, so it is not possible to judge if selection bias was introduced. Another limitation of the study is that there is no information on dust exposure, or a description of the working conditions at the cement mill so it is not possible to relate the lung function differences to dust exposure levels in a quantitative manner.

Al-Naomi et al. (2001) investigated the respiratory health of 75 cement factory workers in the United Arab Emirates by respiratory questionnaire and spirometry. No information on cement dust exposure levels was available. Among the cement workers there was a higher prevalence of respiratory symptoms and a reduction in spirometry measurements, in comparison with matched controls.

Pulmonary function and the presence of respiratory symptoms were investigated in a group of workers of Pakistani or Bangladeshi origin at a United Arab Emirates cement-producing factory (Al-Naomi et al. 2001). All 75 workers at the factory were invited to participate. Eight workers did not participate, either because they were on leave at the time of the study, or were senior management personnel. None reported a history of allergic or chronic respiratory disease prior to joining the cement industry. A control group was selected as a group of similar age, nationality and socio-economic status, and with no history of chronic respiratory disease or history of occupational exposure to dust chemicals fumes or gases. They were selected from those seeking a certificate of good health from a medical clinic so that they could renew their residency permit; the report described them as 'retail salesmen'. Of the 136 unexposed workers invited to participate, 134 accepted. Information on general and respiratory health and lifestyle was collected using an interviewer-administered modification of the MRC standardised questionnaire on respiratory symptoms. It was not stated if the interviewer was blinded to exposure status. Also, information on the use of personal protective equipment was collected. Body mass index (BMI) was calculated from height and weight measurements, and blood pressure was measured. Smoking was analysed as 'pack-years' (smoking 20 cigarettes a day for one year was defined as a pack-year). Spirometry was conducted according to ATS recommendations.

The control and exposed groups were similar with respect to age (mean \pm SD 44.4 \pm 7.5 and 44.2 \pm 9.9 years, respectively, although elsewhere in the report the mean age of the control group is given as 32.1 years), BMI, blood pressure and education. However, there was a large and statistically significant difference in cigarette consumption; pack-years were 0.5 \pm 0.05 for controls and 4.6 \pm 1.1 for the exposed group. The percentage of workers self-reporting the following chronic respiratory symptoms were higher in the exposed group: cough (10% in control vs. 30% in exposed group), phlegm (5% vs. 25%), wheeze (3% vs. 8%), dyspnoea (5 vs. 21%), sinusitis (11% vs.

27%). Asthma was higher in the exposed group (3% vs. 6%). Bronchitis, defined by the presence of phlegm/cough for at least 3 months of the year, was reported in 4% of controls compared with 13% of exposed workers. All spirometry parameters were significantly lower in the exposed group. In the control and exposed groups, respectively, FVC was (mean \pm SE) 3.42 ± 0.05 and 3.17 ± 0.08 l, FEV₁ was 2.99 ± 0.04 and 2.59 ± 0.07 l/s, and FEV₁/FVC ratio was 87.64 ± 0.58 and $81.78 \pm 1.11\%$. Within the group of exposed workers, spirometry values were lower among supervisors (13 workers), attendants (5) and machine operators (23) who were said to work on the factory floor longer than maintenance workers. The percentages of exposed workers with a mild impairment of FEV₁ (defined as below the lower 95% CI for the predicted value but >60% of predicted) were greater than the controls (43% in controls vs. 67%). Only one subject in the study, who was in the exposed group, had a more severe impairment. A comparison of the spirometry results for exposed workers who were smokers and non-smokers showed few differences, suggesting the smoking did not have an effect on the spirometry results (although the report text described 'significantly lower values' for smokers which were not apparent in the results table). Overall, this study showed a higher prevalence of chronic respiratory symptoms and reduced pulmonary function in cement workers, compared with a group of workers who were not exposed to dusts and chemicals. However, there are inconsistencies in the reporting of the study, and uncertainties regarding the role of smoking in the differences observed between the cement workers and controls. Consequently the reliance that can be accorded to this study is limited.

Noor et al. (2000) investigated 62 Malaysian cement factory workers. A respiratory questionnaire was administered and spirometry was conducted. Current dust measurements were available. There was a higher prevalence of respiratory symptoms and a reduction of FVC, FEV₁ and FEV₁/FVC ratio among the cement workers compared with controls that were university staff or students.

In a briefly reported study, pulmonary function and respiratory symptoms were investigated in workers at a Malaysian Portland cement-producing factory (Noor et al. 2000). Subject selection procedures were not described. Sixty-two cement workers were selected for study; 32 were non-smokers with a mean age of 35 years and 30 were smokers with mean age of 37. They had worked at the factory for between 0.5 and 38 years. Seventy university students and staff, all not current smokers with mean age of 37, served as controls. Airborne dust levels were determined from 2 hour samples taken at six static sampling points in the cement factory and one at the university. Dust was fractionated into 'fine' (diameter <10 μ m) and 'course' diameter <10 μ m particles. Spirometry was conducted according to standard methods. Lifestyle and medical information were collected using an ATS questionnaire.

Dust measurements in the cement factory were highest in a packing area, with total dust concentrations of 10.2 mg.m^{-3} of which 8.0 mg.m^{-3} were fine particles. In other areas the total dust concentrations ranged from 3.4 to 0.2 mg.m^{-3} . FEV₁ and FEV₁/FVC ratio, unadjusted for height and age, were reduced in both non-smoking and smoking cement workers, in comparison with the control group. These differences were more marked when the

analysis was limited to subjects aged 30 – 40. For these subgroups, for controls, non-smoking and smoking cement workers, respectively, mean (\pm SE) FEV₁ was 3.0 \pm 0.1, 2.7 \pm 0.1 and 2.7 \pm 0.2 l and FEV₁/FVC ratio was 95.5 \pm 1.1, 85.4 \pm 1.6 and 81.1 \pm 3.7%. The differences between the controls and both groups of cement workers were statistically significant. The height distribution within each of these subgroups was similar. Mean FEV₁ when expressed as a percentage of predicted (using a Malaysian reference population) was reduced in both non-smoking and smoking cement workers compared with controls; values for the control and two respective control groups were 102 \pm 2, 92 \pm 4 and 96 \pm 4%. The cement workers were stratified according to whether they worked in areas with high (mean of 8.5 mg.m⁻³), medium (2.8 mg.m⁻³) or low (0.58 mg.m⁻³) dust concentrations and it was found the spirometry parameters generally had the lowest mean values in the high exposure group. The prevalence of three respiratory symptoms was significantly higher among cement workers. These were morning cough (in 6% of controls vs. 25% of cement workers), morning phlegm (11% vs. 24%) and chest tightness (6% vs. 19%).

Overall, this study showed a higher prevalence of chronic respiratory symptoms and reduced pulmonary function in cement workers, compared with university students/staff. One weakness of the design of this study is that the exposed and control groups were unlikely to be equivalent in terms of socio-economic status, a possible confounding factor. Nevertheless, among the cement workers the effects on pulmonary function were most marked for those with the highest dust exposures, suggesting that cement exposure can affect respiratory health.

Mengesha et al. (1998) administered a respiratory questionnaire and conducted spirometry and peak flow measurements in 53 Ethiopian cement factory workers. Current dust measurements were available. There was a higher prevalence of respiratory symptoms among cement workers, compared with controls. FVC, FEV₁ and FEV₁/FVC ratio were lower for the group with the highest dust exposures.

Pulmonary function and the presence of respiratory symptoms were investigated in persons working in dusty environments in Ethiopia (Mengesha et al. 1998). Among those investigated were 53 cement factory workers, all non-smokers, chosen at random. No information on their age, gender or duration of employment was reported. The control group was 111 males and 100 females, all non-smokers who worked in areas free of dusts. They were said to live in the same area and to be of the same socio-economic status as the exposed workers. The method of recruiting the controls was not stated. Information on respiratory symptoms was gathered using a modification of the MRC and ATS questionnaires and used to identify respiratory conditions referred to as chronic cough, chronic bronchitis and bronchial asthma. Spirometry was conducted using standard methods for 71 controls and 28 cement workers prior to commencing a work shift. PEF_R was determined using a Wright peak flow meter. Current respirable airborne dust concentrations were determined from personal air samples. A total of 11 samples, taken over 6 - 8 hour periods, were analysed.

The mean dust level (\pm SD, presumably a 4 - 6 h TWA) for rotary kiln workers was $3.2 \pm 3.5 \text{ mg.m}^{-3}$ and for packing workers $43.1 \pm 35.3 \text{ mg.m}^{-3}$. The prevalence of respiratory conditions was significantly higher among the cement workers; the percentage of workers in the control and exposed groups, respectively, was 9% and 30% with chronic cough, 9 and 26% with chronic bronchitis and 8% and 32% with bronchial asthma. The spirometry results for the controls and cement dust workers were not presented in a way that allowed a comparison. However, it was possible to compare the results for 12 operators of the rotary kiln with those for 16 cement packers. Mean FVC was similar, FEV₁ was slightly lower for the packers (91% vs. 98% of predicted) and FEV₁/FVC was significantly lower for the packers (90% vs. 98%). PEF_R was lower for the packers, but the difference was not statistically significant (92% vs. 98% of predicted). Study shows increased prevalence of respiratory symptoms in cement workers, compared with the unexposed controls, and poorer lung function test results in the cement workers with the highest current dust exposure.

Abuhaise et al. (1997) investigated the respiratory health of 348 Jordanian cement factory workers, who were divided into three groups exposure groups, based on current dust exposure measurements. A respiratory questionnaire was administered and spirometry was conducted. There was a higher prevalence of asthma in the group with the highest dust exposure, but the spirometry results were similar for all three groups.

Pulmonary function and respiratory symptoms were investigated in workers at a Jordanian Portland cement-producing factory (Abuhaise et al. 1997). All workers employed at the factory for more than 3 years were invited to participate; 442 (73%) agreed. After exclusions due to family or pre-employment history of respiratory disease, being an ex-smoker (only smokers and non-smokers were included), participation in other activities involving dust exposure, unacceptable spirometry performance, 348 workers were available for study. They were predominantly middle-aged men, with a mean age of 41 years and most had worked at the plant for between 3 and 14 years. There was no unexposed control group. Demographic information and occupational and medical histories were collected using a modification of the MRC and ATS questionnaire on respiratory symptoms. Spirometry was conducted once during a workshift using procedures that were in accordance with ATS recommendations. Observed readings were reported as percentage of predicted. Current respirable dust exposure levels were measured by personal sampling. Between 3 and 5 workers in each of 4 plant areas were sampled. Three exposure groups were identified; group 1, the lowest exposure group, included administrative and transport workers; group 2 included maintenance staff, clinker workers and foremen; group 3 included packers and cement mill workers.

The measured geometric mean (\pm SE) respirable dust concentrations (presumably 8 h TWAs) were $0.5 (\pm 2.1)$, $1.6 (\pm 2.6)$ and $3.9 (\pm 4.0) \text{ mg.m}^{-3}$ in exposure groups 1, 2 and 3, respectively. Overall, 58% of workers reported no respiratory symptoms during the last 3 years. The most frequent symptoms were cough reported by 19% and dyspnoea by 18%. About 16% were considered to have asthma and about 15% were considered to have chronic

bronchitis (defined as phlegm/cough for at least 3 months each year for at least 2 years). The prevalence of asthma was higher in group 3 (27%), but for other respiratory symptoms and conditions there were no meaningful differences between the groups. Age or duration of employment had no influence on questionnaire responses. The prevalence of respiratory symptoms was higher among smokers. Mean FEV₁, FVC, FEF_{25-75%} and FEV₁/FVC ratio were similar for all three exposure groups. Overall, this study did not provide any convincing evidence that the exposure conditions at this factory were causing respiratory problems in the workforce. Because there were considerable overlaps in the dust exposure concentration ranges for the three study groups the power of the study to detect a dose-response relationship would be low. The participation rate of 73% raises the possibility that the study population was not representative of the workforce.

Chanrawanshi and Pati (1996): this study does not contribute any useful information on the effects of cement dust.

PEFR was measured in Indian cement-producing factory workers, with the primary aim of assessing the effects of shift work on pulmonary function (Chanrawanshi and Pati 1996). The results showed poorer lung function in shift workers, but no exposure data was provided and no conclusions regarding cement can be drawn.

Yang et al. (1996) administered a respiratory questionnaire and conducted spirometry on 412 Taiwanese cement factory workers. They were exposed to mean respirable dust levels of about 4 mg.m⁻³. The prevalence of respiratory symptoms was higher and FVC and FEV₁ were reduced, in comparison with controls.

In Taiwan, pulmonary function and respiratory symptoms were investigated in workers at four cement factories (Yang et al. 1996). The study considered all male workers who had been employed for more than 5 years at one of these factories. After a small number of exclusions because of refusal to participate or unacceptable spirometry performance, 412 production workers were investigated. Their mean age was 44 years and their mean duration of employment was 17 years. A group of 179 administrative workers at these plants served as controls. Their ages, duration of employment and prevalence of smokers was similar to the exposed group. Demographic information and employment histories were obtained by interview. Information on respiratory symptoms was obtained using a modification of the ATS questionnaire. Chronic bronchitis was defined as cough/phlegm on most days for at least 3 months of the year. Spirometry was conducted according to ATS recommendations. It was not stated if the interviewer and spirometry technicians were blinded to exposure status. Current respirable dust exposure levels were measured from personal air samples, for persons working in all the principal job categories. A total of 198 samples, taken over 4 - 8 hour periods, were analysed.

Geometric mean (\pm SD) respirable dust concentration for control subjects was 0.41 ± 0.98 mg.m⁻³ and for the exposed group, 3.58 ± 4.89 (range 1.28-8.12) mg.m⁻³. The prevalence of cough (19 vs. 12%) and phlegm (18 vs. 13%) was significantly higher in the exposed group. However, the prevalence of

wheezing, dyspnoea and chronic bronchitis was similar in the two groups. After adjustment for smoking, age and height, FVC (3.80 l for controls vs. 3.54 l for exposed), FEV₁ (2.98 vs. 2.73 l) and FEV₁/FVC (78.42 vs. 77.12%) were significantly reduced in the exposed group, in comparison with the controls. There were similar reductions in FEF₅₀, and FEF₇₅. The study showed an increase prevalence of respiratory symptoms and reduced pulmonary function in workers exposed to cement dust. A potential weakness of this study is that there may have been socio-economic differences between the cement exposed workers and administrative controls, which could have contributed to the observed differences between the groups.

3.4.1.3 Study using chest radiography

Abrons et al. (1998) conducted chest radiography in 2640 US cement factory workers. Information on current dust exposure levels was available. A slight increase in the prevalence of minor pulmonary radiographic abnormalities, which seems to be confined to current smokers, was observed for the cement workers in comparison with blue collar controls. Very few of the cement workers had radiographic findings that were indicative of significant pulmonary disease.

In a cross sectional study, chest radiography was conducted in 2640 randomly selected workers from 16 US Portland cement producing factories (Abrons et al. 1997). This is a report of an additional investigation conducted on the same subjects as a study reported in 1988, which showed no convincing evidence of a relationship between cement dust exposure and the presence of respiratory symptoms or and changes in pulmonary function (Abrons et al 1988, cited in HSE 1994). These factories, were a random sample of the 153 US cement factories. All workers were invited to participate, and 87% agreed. The mean age of the exposed group was 41 years, and 95% were male. The control group comprised of 1458 blue-collar workers drawn from the food, textile and electrical equipment manufacturing industries, judged on the basis of workplace inspections by a hygienist not to have been exposed to known respiratory hazards. Only 55% of the unexposed workers invited to participate agreed. There were age and gender differences in comparison to the exposed group; for controls, the mean age was 34 years, and 50% were male. Standard postero-anterior chest radiographs were assessed according to the 1971 ILO-U/C system, by three independent, qualified, readers. Demographic information and medical and employment histories (including information on asbestos exposure) were obtained by an interviewer-administered questionnaire. Current dust exposure levels were determined by personal full-shift sampling.

The overall geometric mean levels of respirable (211 samples) and total dust (1011 samples) were 0.57 mg.m⁻³ (range 0.01-46.22) and 2.90 mg.m⁻³ (range 0.01-78.61) (presumably an 8 h TWA), respectively. Crystalline silica was detected in about 15% of personal respirable dust samples, and levels in about 10% of these exceeded a limit of 0.05 mg.m⁻³. Additionally, no asbestos fibres were found in any area air samples taken, or in bulk material samples. Radiography revealed low prevalence of abnormalities for both exposed and control workers. Rates for both rounded and irregular small opacities (category 1 profusion or greater) were about 1% among cement workers

compared with about 0.1% for the control group. Three cement workers had rounded small opacities of category 2; all other cases of opacity were category 1. The prevalence of pleural abnormalities was about 1.6% in exposed workers and 0.2% in controls. These differences were statistically significant, and remained so after adjustment for age and smoking. The prevalence of abnormalities for the subset with limited previous exposure to other agents was similar to that of the whole group of cement workers. A comparison of the findings for this subset with controls by smoking status indicated that the significant differences were confined to current smokers. An analysis that took account of duration of employment in current and previous current job and cement exposure levels based on current measurements indicated a positive relationship between exposure and with pleural abnormalities, but not with opacities.

This study indicates an association between employment in the cement industry and a slight increase in the prevalence of minor pulmonary radiographic abnormalities, which seems to be confined to current smokers. It is noted that very few of the cement workers had radiographic findings that were indicative of significant pulmonary disease. Because current exposure levels covered such a wide range, and historical exposure data were not available, no information on dose-response relationships can be gained from this study.

Laraqui et al. (2001) found a higher prevalence of respiratory symptoms together with spirometry deficits among Moroccan concrete manufacturing workers, compared with controls.

Laraqui et al. (2001) investigated the prevalence of respiratory problems in workers at two ready-made concrete production plants in Morocco. This study is considered briefly, as the workers would have been exposed to gravel and sand (and hence silica) as well as to cement dust. The study population was 120 concrete workers, who were compared with 120 civil servants. No quantitative airborne dusts measurements were available. A higher prevalence of respiratory symptoms, rhinitis, conjunctivitis and dermatitis was reported among the concrete workers. Also, the proportion of workers with spirometry deficits, as defined by an official French government grading system, was higher among the concrete workers.

3.4.1.4 Studies investigating symptoms only

Alvear-Galindo et al (1999) administered a respiratory questionnaire to 425 Mexican cement factory workers. A semi-quantitative dust exposure assessment was conducted. The prevalence of respiratory symptoms was highest among those with the highest exposures.

The association between levels of exposure to cement dust and respiratory symptoms was studied in workers at a Mexican Portland cement-producing factory (Alvear-Galindo et al. 1999). The study population was 425 workers at a factory, representing 95% of the workforce. Their mean age was 39 years. All had worked at the factory for their entire working life. Six semi-quantitative levels of dust exposure were established from interviews with a small group of

experienced workers, taking account of time spent at various work positions and the visibility in those areas. Each level was given a score of 1 (the lowest) to 6. A list of 85 work positions were drawn up and exposure scores were assigned to each position. Information on the work history was obtained by questionnaire, and a cumulative exposure score was calculated for each worker. It was stated that exposure to other respiratory tract irritants, including tobacco smoke, was minimal. Information on respiratory symptoms was gathered using the ATS questionnaire. Also, information on respiratory illness history was available in personnel records. An occupational health physician considered the information from these two sources to make an assessment of the presence of respiratory disease. Some workers were said to have received compensation for occupational diseases.

Most workers had been exposed to levels of 4, 5 or 6 for most of their working life. Very few workers under the age of 25 reported respiratory symptoms. However, 8% of workers aged 25-44 years and 16% aged 45 or more were diagnosed with bronchitis; 72% and 83%, respectively, had dyspnoea and 24% and 32% had wheezing. A diagnosis of 'occupational respiratory disease' (the diagnostic criteria were not described) was made for 32% and 52% of workers from the two respective age groups. The prevalence of respiratory problems was greatest among those with the highest cumulative exposure scores, after adjusting for age.

This study reported a high prevalence of respiratory problems at the cement factory, which was positively associated with the estimated cumulative dust exposure. A limitation to the value of this study is that quantitative exposure information was not available.

Abou-Taleb et al. (1993) administered a respiratory questionnaire to 304 cement factory workers in the United Arab Emirates. Chronic bronchitis was present in 12% of workers, but there was no control group for comparison.

The respiratory health of a sample of United Arab Emirates cement workers was assessed (Abou-Taleb et al. 1995). From four cement production factories, 304 workers were selected at random. These represented about 50% of the workers from each factory. Their mean age was 38 years, and 88% were of Indian nationality. Demographic and lifestyle information and medical histories were obtained by interviewer-administered questionnaire. Respiratory health problems most frequently reported were chronic cough in 19% of workers and chronic bronchitis (diagnostic criteria were not stated) in 12%. Other health problems reported in a minority of workers included sneezing with runny or stuffy nose, burning eyes, dermatitis, headache and fatigue. It is not possible to draw firm conclusions from this study as it is difficult to assess the contribution of cement dust to the reported health problems in the absence of either an unexposed group or groups with contrasting dust exposure levels for comparisons.

Yang et al. (1993) administered a respiratory questionnaire to 661 Taiwanese cement factory workers. Current dust measurements were available. The prevalence of respiratory symptoms was similar in low, medium and high dust exposure groups.

The respiratory health of cement workers at the four plants in Taiwan was investigated (Yang et al. 1993). It is possible that some of the data reported in this publication was also used in the Yang et al. (1996) report, summarised above. This earlier report provides information on respiratory symptoms and spirometry for 661 cement factory workers. The workers were classified into three groups according to respirable dust exposure concentration. Group 1 were 146 administrative workers, with mean dust exposures of 0.22 (range 0.03-1.4) mg.m⁻³. Group 2 were 140 maintenance workers, machine operators and drivers, with mean dust exposure of 0.55 (0.13-1.94) mg.m⁻³. Group 3 were 375 workers in jobs with the highest exposures, for example packing or preparing raw materials; mean dust concentration was 1.24 (0.38-8.12) mg.m⁻³. The mean age and duration of employment of the three groups were similar, although the percentage of smokers was higher in groups 2 and 3. Respirable dust exposure levels were determined by personal breathing zone sampling over 4 to 8 hour periods. Between 48 and 78 samples were taken for each exposure group.

The only indication of a difference in respiratory health between the groups was the prevalence of cough was higher for group 3 (17% compared with 11% for groups 1 and 2); this difference was statistically significant after adjusting for smoking. The prevalence of other symptoms and spirometry results were similar for all groups. Overall, this study provides no convincing evidence of differences in respiratory health between the three exposure groups.

3.4.1.5 Summary of information on the effects of long-term exposure on non-malignant respiratory disease

HSE (1994) noted evidence that repeated exposure to Portland cement has produced chronic bronchitis and impaired pulmonary function, but firm conclusions could not be drawn because of limitations of the available studies. The effects of long-term exposure remain poorly studied, particularly because there are no longitudinal prospective studies. Of the 15 recent studies available, most were cross sectional in design, conducted in Portland cement production plants outside of Europe. The exception was a retrospective cohort study of a relatively small number of Norwegian cement plant workers (Fell et al. 2003). This study provided evidence that long-term exposure to an average of respirable dust concentration of about 1 mg.m⁻³ is not associated with deficits in lung function or with an excess of COPD. HSE (1994) highlighted several earlier studies that produced no convincing evidence an association between respiratory impairment and long-term exposure to cement dust at mean airborne concentrations (respirable dust) ranging from 0.6-2.2 mg.m⁻³. The consistency of these studies provides evidence that long-term exposure to cement plant dust at mean concentrations of about 1-2 mg.m⁻³ will not affect respiratory health.

Of the remaining studies, all had significant design limitations. Notably, due to the cross sectional design the adverse effects of cement may be underestimated because this type of study cannot take account of workers that may have left the industry due to work-related health problems. Another key weakness of these studies is that a number lacked quantitative exposure

data and none included historical exposure data. Nevertheless, a many of these studies provide a consistent picture of the presence of respiratory symptoms, reports of bronchitis, and lung function deficits in workers exposed to cement plant dust. Typical symptoms associated with cement plant work were cough, phlegm and dyspnoea. Deficits of lung function were usually seen as a reduction in FEV₁, FVC and FEV₁/FVC ratio, suggesting an obstructive impairment. In studies that provided some information on dust exposure conditions, the respiratory effects were generally associated with dust exposures likely to be approaching, or in excess of, the UK occupational for Portland cement of 4 mg.m⁻³ respirable dust and 10 mg.m⁻³ total dust (8h TWAs) (also the limits for dusts generally). In particular, a study in Taiwan cement factories found that at current average respirable dust levels of just under 4 mg.m⁻³ there were deficits in FEV₁ and FVC in the exposed workers and a study in Tanzanian cement factory suggested that 30 years exposure to average total inhalable dust levels of 10 mg.m⁻³ would lead to deficits in FEV₁ and FVC. Overall, the pattern of evidence indicates that cement dust has the potential to cause chronic productive cough and an obstructive impairment of pulmonary function, and hence qualitatively, this strengthens the picture presented in the earlier HSE (1994) review. However, there are no prospective cohort studies available that would allow reliable conclusions on dose-response relationships to be drawn, but the available data do raise concerns that respiratory deficits could develop with long-term average exposures to respirable cement dust above 1 but below 4 mg.m⁻³.

It is noted that cement production plant workers can be exposed to the raw materials of cement production, which include limestone, chalk, clay and shale, to clinker, as well as to Portland cement dust. The exact nature of the dust exposures experienced by cement plant workers has not been characterised in any of the studies. Consequently, one cannot exclude the possibility that the effects on respiratory health may have been due, at least in part, to the raw materials and intermediates of cement production.

3.4.2 Mortality and cancer studies involving workers with long-term exposure to cement dust

HSE (1994) concluded that mortality studies conducted on cement workers provided no evidence of increased incidence of death from non-malignant respiratory disease. With regard to cancer, three cohort mortality studies conducted in cement manufacturing workers found elevated SMRs for stomach cancer. The elevation was statistically significant in one study conducted in the UK (SMR 1.75, based on 22 observed deaths,). In other two studies the SMRs were not statistically significant (SMR 1.19 based on 24 deaths and SMR 1.35 based on 27 deaths). Several other studies found associations between exposure to cement and various site-specific cancers but these were isolated reports and no firm conclusions could be drawn. Overall, HSE (1994) came to the view that there was no convincing evidence for an increased incidence of any site-specific cancer resulting from exposure to cement dust, but it was also acknowledged that the results emerging from the studies available up to 1991 were not consistently and reassuringly negative.

Since 1991, a number of mortality and cancer cohort studies conducted in cement production workers of cement masons have been conducted. Also, a series of case control studies conducted in Germany have drawn attention to occupational contact with cement as a possible risk factor for cancers of the oral cavity, pharynx and larynx. The present review also briefly considers earlier case control studies that have investigated risk factors for laryngeal cancers, and other recent cancer related studies that include subjects that may have been exposed to cement.

3.4.2.1 Mortality and cancer cohort studies in cement production workers or masons

Recent cohort studies conducted in cement production workers in three northern European locations, in Sweden, Denmark and Lithuania, are available. Additionally, a cancer cohort study has been conducted in Icelandic cement masons. These studies are appraised below, and summarised in Table 2.

Jakobsson et al. (1993) investigated mortality and cancer morbidity in about 2400 Swedish cement factory workers. Overall mortality and cause-specific non-malignant mortality for the cement workers were similar to the national rates. However, the analysis of individual tumour sites revealed an increased risk of tumours of the right side of the colon (SIR 2.7, 95% CI 1.4 - 4.8, 12 cases). The risk was increased among those with more than 25 years cement plant work.

In a retrospective cohort study mortality and cancer morbidity experiences of a group of about 2400 workers at two Swedish cement production factories were investigated (Jakobsson et al. 1993). This study was conducted as a follow up to the Jakobsson et al. (1990) case control study summarised by HSE (1994), which reported an increased risk of colorectal cancer associated with one of the cement factories (factory A). The cohort consisted of all men employed at the factories at the start of January 1952 or since then, and with

a total employment of at least one year. Mortality and cancer morbidity information to the end of 1986 was obtained from death certificates and a national tumour registry and compared with the age-adjusted rates for the general population of Sweden (mortality) or the regions in which the plants were located (morbidity). About 90% of the cancers diagnoses were confirmed by histology or cytology. The analysis focused on cancers occurring 15 years or more after commencement of exposure.

Very limited exposure data were available. The report states that new cement ovens and automatic process controls were introduced at both plants during the 1960s and ventilation was improved during the 1970s suggesting that dust exposure during the latter years of the study period were considerable lower than earlier. At factory A, total dust measurements made from the mid-1970s onwards were generally lower than 10 mg.m^{-3} (presumably as a TWA). Dust surveys conducted in factory B during the early 1970s showed that total dust airborne concentrations were generally less than 20 mg.m^{-3} and in later years less than 10 mg.m^{-3} . Concentrations greater than 100 mg.m^{-3} were found for some cleaning and maintenance tasks, although it was suggested that respiratory protection might have been used during these operations. Factory B measurements were based on personal sampling and expressed as a TWA, although the sampling time was not stated. The quartz content of the dust was said to be generally less than 5%, and no cases of silicosis have been reported at these plants.

Overall mortality (495 observed deaths vs. 527 expected) and cause-specific non-malignant mortality for the cement workers were similar to the national rates. Also, overall tumour morbidity (162 observed cases vs. 160 expected) did not differ from the regional rates. However, the analysis of individual tumour sites revealed a significant increased risk of tumours of the right side of the colon (SIR 2.7, 95% CI 1.4 - 4.83, 12 cases). The risk was increased among those with more than 25 years cement plant work (SIR 4.3, 95% CI 1.7 - 8.9, 7 cases). However, the risk of right-sided colon cancer was not significantly increased at factory A, which was associated with an excess of colorectal cancer morbidity in the Jakobsson et al report of 1990. All colon cancer cases had been confirmed by histology or cytology. There was no excess of tumours at other sites. The authors discussed the possibility of bias due to the use of an inappropriate reference population. Farmers are known to have very low rates of colon cancer and a relatively high proportion of farmers in the localities of the cement plants may have resulted in a bias of the magnitude of the calculated SIR in the direction of an overestimate.

As an extension to their 1993 study, Jakobsson et al. (1994) reanalysed mortality and cancer morbidity for the 1520 Swedish cement factory workers. Comparisons with other groups of blue-collar workers and with the general population were conducted. An increased risk of cancer of the right side of the colon (SIR 2.6, 95% CI 1.4 - 4.6) for the cement workers was confirmed.

In a later report, the risk of right-sided colon cancer at the two Swedish cement factories was compared with that for asbestos cement workers, other industrial workers without significant exposure to mineral dust or fibres and fishermen in Sweden (Jakobsson et al. 1994). All members of the

occupational groups had a minimum employment of one year, and only cancer morbidity occurring more than 15 years after the commencement of employment was considered. Cancer mortality and morbidity for the period 1958 to about 1987 (slightly later for fishermen) was ascertained and SMRs and SIRs were calculated with reference to the general male population of Sweden. The cohort of cement workers consisted of 1526 blue-collar workers with a median birth year of 1927. The asbestos cement cohort was 981 blue-collar workers from a building materials factory, with median birth year 1913. The 'other' industrial workers cohort consisted of 3965 blue-collar workers from a variety of industries where some chemical exposures were likely. The cohort of fishermen was 8092 in number, with a median birth year of 1924.

Overall cancer mortality for the cement workers was similar to the general population (SMR 1.02, 95% CI 0.92-1.25), and to 'other' industrial workers. The cancer mortality for the asbestos workers was slightly higher, and for the fishermen slightly lower. Morbidity from right-sided colon cancer was increased among cement workers (SIR 2.61, 95% CI 1.35-4.57), but risk of left-sided colon cancer was not increased. The findings for asbestos cement workers were similar. Morbidity for colon cancers among the 'other' industrial workers and fishermen was similar to the general population. This study showed an increased risk of right-sided colon cancer among workers with long-term exposure to cement dust compared to other blue-collar workers, which constitute an appropriate reference population, and to the general population.

Vesbo et al. (1991) investigated cancer morbidity in 546 Danish cement factory workers. No evidence of an increased risk of cancer was reported, when compared with a control group drawn from the local population.

The relationship between employment at a Danish Portland cement factory and cancer morbidity was investigated in a cohort study (Vesbo et al. 1991). The cohort of cement workers comprised of all men aged 46-69 living in the city of Aalborg who had worked at the factory for at least one year prior to 1974; there were 546 men meeting these criteria. The control group was all men of the same age group living in this city who had not worked at the factory for a year or more and who were born on the 1st or 15th of each month; they numbered 856. Information on employment and lifestyle factors was gathered by questionnaire. From factory personnel records, the cement workers were classified as having either 1-20 years or ≥ 20 years employment. Information on actual occupation within the factory was not available so it was not possible to categorise individuals into more precisely defined exposure groups. The control subjects were categorised into three occupational groups, blue-collar workers, white-collar workers and 'others'. Information on cancer cases for the period 1974 to 1985 was obtained from the Danish Cancer Registry. The numbers of observed cases of cancer for each study group were compared to the expected number based on cancer incidence rates for the relevant age groups for the total male population of Denmark.

Airborne cement dust measurements were not made prior to 1974. Personal sampling during 1974 showed that 9% of respirable dust measurements were

greater than 5 mg.m^{-3} . No increased risk of all types of cancer was seen among cement workers compared with either the general male population or with each of the control groups. However, the risk of respiratory cancer was higher among cement workers when compared to the general male population. The authors provided a more detailed comparison of the risk of respiratory cancer for cement workers and white-collar controls, after excluding all men with asbestos exposure due to employment at a nearby asbestos-cement plant (approximately 5% of cement workers) and controlling for age and smoking. The RRs for respiratory cancer were 0.5 (95% CI 0.1-1.5) for men with 1-20 years cement factory employment and 1.0 (95% CI 0.4-2.6) for men with ≥ 20 years employment. The authors pointed out a drawback with respect to comparisons with the blue-collar control group, since members of this group may have been exposed to asbestos at a large local shipyard. Overall, this study provides no convincing evidence of an increased risk of cancer morbidity associated with cement dust exposure at the cement factory.

Smailyte et al. (2004) investigated mortality and cancer morbidity in 2500 Lithuanian cement factory workers. There was an increased incidence of lung cancer among cement workers (SIR 1.5, 95% CI 1.1-2.1) compared to the general population, but smoking was not taken account of in the analysis. A dose-related trend towards an increased incidence of stomach cancer was observed, although the number of cases was small.

In a cohort study, mortality and cancer incidence among workers at a Lithuanian Portland cement producing plant was investigated (Smailyte et al 2004). The study population comprised all workers employed by the cement producing departments for at least 12 months between 1956 and 2000 and who were available for the follow-up period of 1978 to 2000. The study population numbered 2498, which was about 94% of all those who had worked at the plant from 1978 to 2000. This cohort represented 43490 person years of exposure. Approximately 70% of the study population were men. Subjects were identified from factory records, which provided information on age, employment start and finish dates, and factory departments in which each subject worked. Information on vital status, cause of death and cancer cases were obtained from Government population registers, death certificates and the Lithuanian Cancer Registry. SIRs and SMRs were calculated with reference to the gender and age-adjusted rates for the general Lithuanian population.

Some dust exposure information was available from 1975, expressed as annual mean total dust concentrations. These were calculated from static sampling measurements (sampling time was not stated) conducted four times a year in various departments. Annual mean dust concentrations were highest in the packing (ranging from 19.1 to 15.0 mg.m^{-3}) and calcining (19.2 to 8.2 mg.m^{-3}) departments.

The overall mortality among male cement workers was not increased (376 observed deaths, SMR 1.0, 95% CI 0.9-1.1), but deaths from malignant neoplasms were increased (102 observed, SMR 1.3, 95% CI 1.0-1.5), mainly due to lung cancer mortality (SMR 1.4, 95% CI 1.0-1.9). There was no excess mortality for non-malignant respiratory disease. Among females, overall

mortality and mortality for specific causes was similar to that of the general Lithuanian population. The cancer incidence data for male cement workers revealed a slight excess for all malignancies (SIR 1.2, 95% CI 1.0-1.4). The only site showing a significant excess was the lung (SIR 1.5, 95% CI 1.1-2.1). A total of 389 cement workers had been also been involved with the production of asbestos cement at another plant in the same area. When these were excluded from the analysis the incidence of lung cancer remained elevated (SIR 1.4, 95% CI 1.0-2.0). There was a slight trend towards an increased incidence of lung cancer with increasing time since first employment and duration of employment. The authors presented cancer SIRs for categories of cumulative dust exposure, but it was not clear from the report how this index of exposure was calculated. There was no obvious relationship between lung cancer and cumulative exposure, although the SIRs were significantly increased for the two highest dust exposure categories designated '55-130 mg.m⁻³ year' (SIR 2.0, 95% CI 1.2-3.4) and '>130 mg.m⁻³ year' (SIR 1.8, 95% CI 1.1-3.0). The authors of the study drew attention to an increase in the incidence of cancers of the urinary bladder, but the increase was not statistically significant (8 cases, SIR 1.8, 95% CI 0.9-3.5) and there was no convincing relationship with duration of employment of cumulative dust exposure. There was a trend towards an increased incidence of stomach cancers with cumulative dust exposure; for a '55-130 mg.m⁻³ year' exposure group the SIR was 1.1 (4 cases, 95% CI 0.4-2.8) and for a '>130 mg.m⁻³ year' exposure group the SIR was 1.5 (6 cases, 95% CI 0.6-3.0).

The authors discussed a major limitation of this study, which is that no data on tobacco consumption was available. It was stated that about half of the male population of Lithuania are smokers, and it is likely that more blue-collar workers smoke than white-collar workers. Therefore it is possible that the results of this study may have been subjected to a confounding influence of smoking. If the analysis had taken account of smoking, the statistically significant differences observed for the SIRs for lung cancers in this study may have been eliminated. For this reason it is not possible to draw conclusions about the role of cement dust in reported increase in the risk of cancers. It is noted that due to the crude nature of exposure assessment there is a high potential for exposure group misclassification, which could result in the masking of any dose response relationships.

Rafnsson et al. (1997) investigated cancer in 1172 Icelandic cement masons. The study revealed an increased risk of lung cancer among the masons in comparison with the general population of Iceland (SIR 1.7, 95% CI 1.1-2.5). The SIR was increased when the analysis allowed for a latency period of 30 years from the start of work as a mason.

Rafnsson et al. (1997) conducted a retrospective cohort cancer study in all 1172 Icelandic licensed masons who were born after 1880 and alive in 1955. The cancer experiences from 1955 to 1993, or until death if before 1993, were ascertained from the national Cancer Registry. It was possible to obtain information on the status of all study subjects. Most of the cancer cases had been verified by histological diagnosis. Masons alive at the time of the study were questioned on their smoking habits and the number of years worked as a mason. The main task of the masons was to finish and smooth the surfaces of concrete structures using wet concrete, applied by trowel or by spraying.

No quantitative exposure data were available, although it was stated that when spaying inside a thick mist of an aerosol of concrete was produced that made it difficult to see across the room. Dermal contact with the wet concrete was described as 'intensive' on hands and face. The concrete contained cement, sand, water and other compounds. A survey showed that about 88% of responding masons had worked as finishers for at least 10 years.

The study showed an increased risk of lung cancer in comparison with the general population of Iceland (SIR 1.69, 95% CI 1.09-2.49). The SIR was increased when the analysis allowed for a latency period of 30 years since completing vocational training (SIR 1.77, 95% CI 1.01-2.88). The percentage of never-smokers among the masons was estimated to be lower than in the general population, but this could not account for the increased risk. For all other tumour sites there were no increased risks for the cement masons. The masons would have been exposed to silica, but there were no identified cases of silicosis. Strengths of the study are that the design provided protection against subject selection bias, cancer ascertainment was reliable and complete, and the follow-up period was long. However, weaknesses of the study are that exposure information was not available and an analysis of cancers in relation to duration of employment was not conducted.

3.4.2.2 German case control studies for cancers of the oral cavity, pharynx and larynx

Maier et al. conducted a series of population-based case control studies in cancer patients from the Gliessen and Heidelberg University Ear Nose and Throat (ENT) clinics investigating occupational and other risk factors for cancers of these sites. These studies are appraised below, and summarised in Table 3.

Maier et al. (1991) investigated the occupational risk factors for cancers of the oral cavity, pharynx and larynx in 200 cases and 800 controls. The study reported an elevated risk of cancer associated with occupational exposure to cement. The estimated RR was 2.4 (95% CI not stated) after adjustment for smoking and alcohol consumption.

In the first study the occupational exposure histories of cases of squamous cell carcinomas of the oral cavity, oropharynx, hypopharynx and larynx were investigated (Maier et al. 1991). The investigations were conducted at the Gliessen University ENT Clinic and the Heidelberg University ENT clinic. The same patients were investigated in an earlier study, in which non-occupational risk factors for cancers at these sites were analysed (Maier et al 1990). The first 100 patients with the above diagnosis attending each clinic for consultation or treatment from September to December 1987 (Gliessen) or February 1988 to April 1988 (Heidelberg) were selected for each study. Patients who were first diagnosed more than three years prior to the study period were excluded. The Gliessen control group consisted of 400 male patients attending either the ENT (200 patients) or Medical (200 patients) Clinics of Gliessen University who had no known cancer. Four controls were assigned to each case, two from each of the ENT and Medical Clinics, who were matched for age (± 2 years) and size of their home. The selection or number of Heidelberg controls was not described in detail, although the design of this part of the study was said to be similar to that of the Gliessen study. Approximately 30% of Gliessen case and control patients and 20% of Heidelberg case and control patients who were invited to participate in the study declined.

Occupational exposure information for each of the Gliessen patients was obtained by one interviewer using a computerised questionnaire that focussed mainly on exposures to harmful substances at work. Patients were asked simply whether or not they had been exposed to asbestos, welding gases, organic compounds, heavy metals, coal products, wood dust, cement, herbicides and ionising radiation. No information on duration or frequency of exposure was collected. Details of the method of collecting exposure information for the Heidelberg patients was not described in detail, except it was mentioned that information of duration of exposure was collected and the analysis included only substances to which the patient had been in contact with for at least once a week for more than 10 years. It was not stated if the interviewer was blinded to case/control status, or if the patients were aware of the purpose of the investigation. The analysis of the results of the Heidelberg study included an adjustment for the influence of alcohol consumption and smoking. RRs for occupational risk factors were estimated from odds ratios.

The majority of cases were within the age range 49 to 66. The distribution of tumour sites was similar for both study centres; approximately 45% were carcinomas of the larynx, 23% of the oral cavity, 24% of the oro-pharynx and 8% of the hypo-pharynx.

The exposure information for the Gliessen and Heidelberg study centres was analysed separately. For the Gliessen study, the percentage of cases with cement exposure was significantly higher than controls (14.5% of controls vs. 25% of cases). The percentage of cases with asbestos exposure was also significantly higher than controls (22% vs. 40%). There were no differences between the cases and controls for the other exposures. For the Heidelberg study, the proportion of cases with occupational exposure to cement (estimated RR 4.4, 95% CI 2.4-8.4), organic compounds, wood dust or coal products were significantly greater than controls. After adjustment for the effects of alcohol and tobacco only the estimated risks for cement remained statistically significant (RR 2.4, 95% CI not stated). The RRs for cement increased with duration of exposure; compared with that for less than 5 years exposure, the estimated RR was almost 3 times higher for 5-20 years exposure, 5.5 times higher for 20-40 years, and 6.3 times higher for more than 40 years. However, the confidence intervals of the estimated RRs for these subgroups were not reported, neither were the number of cases within each exposure band, so it is not possible to assess the robustness of this trend.

A limitation of the Gliessen study is that the exposure assessment identified anyone who had come into contact with a particular substance at any time during their work; the authors consider this part to be a pilot exercise for the Heidelberg study. The Heidelberg study provides evidence of an association between cement exposure and cancer of the upper respiratory/digestive tract. As with many case control studies, the exposure assessment relied on the recall of the subjects, which may be subject to bias because the cases may have a greater motivation to recall chemical exposures than the control patients. Such a bias would result in an overestimation of risks associated with cement exposure. Similarly there may have been interviewer bias in that the interviewer may have pressed harder to gain information on occupation exposure for the cases. However, there is no logical reason for the cases to be more strongly motivated to recall cement exposure in preference to exposures to other substances, unless they were aware of a hypothesis that cement exposure was associated with cancers of the upper respiratory/digestive tract.

Maier et al. (1992) investigated the occupational risk factors for cancers of the larynx in 164 cases and 656 controls. The study reported a slightly elevated risk of cancer associated with occupational exposure to cement. The estimated RR was 1.2 (95% CI 0.9-1.4) after adjustment for smoking and alcohol consumption.

Maier et al. (1992) investigated the occupational exposure risk factors associated with squamous cell carcinoma of the larynx. The cases, numbering 164, were all male patients attending the Heidelberg University ENT clinic with this diagnosis during the periods June 1988 to October 1988 and November

1988 to April 1989, excluding those first diagnosed more than 3 years prior to the study periods. The controls were 656 male patients without a cancer diagnosis, half of whom attended the Heidelberg University ENT and half from the Medical Out-Patients Department of the University Clinic. Four controls were assigned to each case, two from each of the ENT and Medical Clinics, who were matched for age (± 2 years) and size of their home. No information on the numbers of available cases and controls refusing to participate was given. Occupational and personal information was collected by interview using a computerised questionnaire. The subjects were said to have been questioned on extent and duration of exposure to potentially harmful materials, but no detailed information on the questions, or interview conditions were given. RRs for occupational risk factors were estimated from odds ratios.

Regarding the types of laryngeal cancers among the cases, 72 were glottal cancers, 73 were supraglottal, and 19 could not be classified. Several indicators of the level of education showed that average level of education was noticeably higher for controls; for example 90% of cases had no school-leaving certificate, compared with 74% of controls. After adjusting for alcohol and tobacco consumption, a significantly elevated estimated RR was found only for cases reporting occupational exposure to coal/coal tar products (estimated RR 2.67, 95% CI 1.3-5.5). The risk associated with cement exposure showed a marginally statistically significant elevation (estimated RR 1.4, 95% CI 1.1-1.6) but the risk was not statistically significant after adjustment for alcohol and tobacco consumption (estimated RR 1.18, 95% CI 0.9-1.4).

This study points to the possibility of an association between exposure to cement and cancer of the larynx. As for the Maier et al 1991 study, the exposure assessment relied on the recall of the patients, which may have been subject to bias.

An analysis of data from what is presumed to be the same group of cases and controls investigated in the above study was included in a later publication (Maier and Tisch 1997), but this report did not provide any additional information on the risks of laryngeal cancer associated with cement exposure.

Maier et al. (1994) investigated the occupational risk factors for cancers of the pharynx in 105 cases and 656 controls. An elevated risk of cancer associated with occupational exposure to cement was reported. The estimated RR was 2.2 (95% CI 0.9-5.2) after adjustment for smoking and alcohol consumption.

In another case control study, the occupational exposure risk factors for pharyngeal cancer were investigated (Maier et al. 1994). The cases were 105 male patients with histologically confirmed cancer of the pharynx who consecutively attended the University of Heidelberg Department of Otorhinolaryngology/Head and Neck Surgery during the period February 1991 and mid-September 1991. The controls were 656 male patients without a cancer diagnosis, half of whom attended the Heidelberg University ENT and half from the Neurological Clinics. Four controls were assigned to each case, two from each of the ENT and Neurological Clinics, who were matched for age (± 5 years) and size and location of their home. Personal and occupational

information was obtained by interview, all of which were conducted by the same interviewer. In comparison with the earlier Maier studies, more detailed employment information, including the time spent working on particular activities, was gathered. Each subject was asked if they had been exposed, and for how long, to a number of specific materials/activities, including asbestos, welding fume, particular metals, cement, coal tar products, types of wood dust, herbicides, biocides, lubricants and general dusts. RRs for occupational risk factors were estimated from odds ratios.

The percentage of cases that were construction workers was 27%, compared to 7% of controls (estimated RR 4.9, 95% CI 1.3-5.9). Alcohol and tobacco consumption was much higher among cases that were construction workers than controls who were construction workers. There was a similar difference in alcohol and tobacco consumption among cases and controls from other occupations. After adjustment for alcohol and tobacco consumption, the RR estimate for construction workers for the risk of pharyngeal cancer was 2.8 (95% CI 1.1-5.9). When the analysis focused on long-term (more than 10 years) occupational contact with cement the estimated RR was elevated, at 2.2 (95% CI 0.9-5.2) after adjustment for alcohol and tobacco. The 29 cement-exposed cases had a mean duration of contact with this material for 25 (SD 9.3) years. Thus, this study demonstrates an increased risk of pharyngeal cancer among construction workers with long-term contact with cement, but when tobacco and alcohol consumption are taken into account this increased risk was not statistically significant. Overall, this study points to the possibility of an association between exposure to cement and pharyngeal cancer. As for the previous Maier studies, the exposure assessment relied on the recall of the patients, which may have been subject to bias.

Maier et al. (1999) presented an overview of the results of their cancer case control study series (Maier et al. 1991, 1992 and 1994). There was no additional information on cancer risks for cement-exposed workers in this report.

Maier et al. (2002) investigated the occupational risk factors for cancers of the oral cavity, pharynx and larynx in 206 cases and 110 controls, matched for smoking and alcohol consumption. A substantially increased risk of cancer associated with occupational exposure to cement was reported. The OR was 12.6 (95% CI 3.9-43.0) after adjustment for smoking and alcohol consumption.

In the most recent Maier study, occupational and nutritional risk factors for cancers of the oral cavity, pharynx and larynx were investigated in cases matched to controls with similar alcohol and tobacco consumption (Maier et al. 2002). The cases were 209 male German patients with histologically confirmed squamous cell carcinoma at these sites attending the Heidelberg University ENT clinic. The control subjects were 110 male German patients attending the ENT clinic without a cancer diagnosis and matched to the cases for age, alcohol consumption and tobacco consumption. Recruitment took place over a 5-year period, between 1990 and 1995. No further details of the case and control subject selection procedures were described, so it is not possible to assess if these procedures introduced bias. The report stated that access to non-cancer patients with high alcohol and tobacco consumption

was restricted and a willingness of these patients to cooperate was low. It was not stated whether or not any of the cases or controls were included in the studies reported earlier. Occupation and medical histories, and information on alcohol/tobacco consumption, nutrition and socio-economic factors were gathered by interview with the aid of a standardised questionnaire. Specific questions were asked about exposures to asbestos, organic solvents, heavy metals, coal products, wood dust, cement, herbicides and ionising radiation. In the analysis only those occupational substances to which a subject was in contact with at least once a week for more than 10 years was taken into account. It was not stated if the interviewer was aware of the subject case/control status.

The age distribution of cases (56 ± 9 years) and controls (58 ± 10) was similar. The level of educational attainment of the controls was higher, for example 10% of the cases and 29% of the controls attended further education institutes (such as trade school, technical college or university). The prevalence and type of non-cancer diseases present among the cases and controls were similar. Mean alcohol consumption for the cases (86 ± 64 g/d) and controls (97 ± 60 g/d) was similar, as was tobacco consumption (49 ± 30 pack years for cases, 56 ± 37 pack years for controls). The percentage of cases with an occupation in the construction or demolition industries was 21%, noticeably higher than the 7% of controls. When contact with individual occupational materials were considered, regular contact with two materials showed significantly elevated odds after adjustment for alcohol and tobacco consumption: asbestos (OR 8.7, 95%CI 2.0-37.0) and cement dust (OR 12.6, 95% CI 3.9-43.0).

The high OR for cement dust exposure indicated a strong association with cancers of the head and neck. A potential weakness of this study is that the cases and controls did not appear to be comparable in terms of education, suggesting there were socio-economic differences between the two groups. This potential confounding factor was not taken account of in the analysis. Also, there may have been bias in the selection of controls resulting from a lack of willingness of potential controls to participate. The extent and direction of this bias cannot be assessed from the information presented in the report. However, the magnitude of the OR is such that confounding and bias cannot be the only explanation for its elevation.

Dietz et al. (2004) investigated the occupational risk factors for cancers of the larynx in 257 cases and 769 population controls. An elevated risk of cancer associated with occupational exposure to cement was reported. The OR was 1.18 (95% CI 0.77-1.18) assessed by substance questionnaire or 2.04 (95% CI 1.16-3.56) assessed by job-specific questionnaire, after adjustment for smoking, alcohol consumption and socio-economic status.

In a very recent case control study (known as the 'Rhein-Neckar-Larynx Study') the occupational risk factors associated with squamous cell carcinoma of the larynx were investigated (Dietz et al. 2004). Eligible cases were all patients attending the Otolaryngology, Head and Neck Surgery departments of the Heidelberg, Mannheim, Ludwigshafen, Darmstadt and Heilbronn hospitals with a new, histologically confirmed, diagnosis of cancer of the

larynx between May 1998 and December 2000. All laryngeal cancer patients from the study region are treated at these hospitals. Of the eligible cases, 257 (96%) agreed to participate. Three control subjects, matched for age, gender and residential location, were assigned to each case. Controls meeting the matching criteria were chosen at random from population registries. Of the 1233 control subjects selected, 769 (62%) agreed to participate. The age range of participants was 40-75 years.

Information on occupational exposures and other risk factors was obtained by face-to-face interview, using standardised questionnaire. Five interviewers, with special questionnaire training, were used. All cases, and 554 of the controls were interviewed at one of the participating hospitals. An occupational history of jobs held for more than 6 months was obtained, and subjects were asked if they had been exposed to a checklist of named substances that the authors considered to be either known or suspected respiratory carcinogens. Detailed information on exposures was obtained by supplementary job-specific questions, with was used to assign semi-quantitative exposure scores. Detailed information on smoking and alcohol consumption was also gathered. As a validation exercise, a special cement questionnaire was administered about 2-4 years after the original interview to subjects thought to have experienced cement exposure. Information on the techniques used, the types of cement used, the methods of processing cement products on-site, and for exposures to asbestos and asbestos cement was requested. Because of death, loss of contact or refusal, only 26% of eligible cases and 75% of eligible controls participated in the validation exercise.

Smoking was found to be a very significant risk factor for laryngeal cancer. The OR for heavy smokers was 32.5 (95% CI 15.1-71.0), after adjustment for alcohol consumption. High alcohol consumption was also a risk factor, shown by an OR 2.4 (95% CI 1.5-9.7) for those consuming more than 75g alcohol per day, after adjustment for tobacco consumption. The occupation risk factor analysis focussed on cement exposure. One analysis was based on the carcinogenic substance questionnaire responses; 23.3% of cases claimed cement exposure compared to 14.4% of controls (OR 1.18, 95% CI 0.77-1.18, after adjustment for alcohol, tobacco and socio-economic status). A second analysis considered the responses to job-specific questions and found fewer cases and controls judged to have experienced cement exposure; 14.8% of cases had been exposed, compared to 5.1% of controls (OR 2.04, 95% CI 1.16-3.56, after adjustment). However, there were no indications of a dose-response when the ORs were calculated for low (less than 3000 cement working hours) and high (more than 3000 hours) exposure groups; the OR (after adjustment) for the low exposure group was 2.22 (95% CI 1.02-4.48) and for the high exposure group 1.87 (95% CI 0.88-4.01).

In the validation exercise, cement exposure of the 15 participating cases was confirmed, but cement exposure was confirmed for only 69/80 (86.3%) of controls, suggesting that cement exposure for the controls may have been overestimated. This analysis also showed that more cases had also been exposed to slaked lime (which was sometimes used in place of Portland

cement in the 1950s and early 1960s), but the numbers of subjects was too few to draw any conclusions.

Overall, the results of this study provide evidence of an association between exposure to cement and laryngeal cancer. However, this association appears weak as a dose response could not be detected and ORs, at the most, were only marginally statistically significant. As for the Maier studies, the exposure assessment relied the recall of the subjects, which may have been subject to bias in favour of recall of cement exposure by the cases.

3.4.2.3 Earlier case control studies investigating risk factors for laryngeal cancers

Earlier studies of risk factors for laryngeal cancers, identified from a PubMed search of literature published from 1980, have been briefly considered to determine whether there is corroborative evidence for the association between cement exposure and cancers at this site observed in the German studies. Six such studies were found, which are summarised below.

In a Danish study, 326 cases of newly diagnosed laryngeal cancer were investigated (Olsen and Sabroe, 1984). For each case four control subjects, matched for age and gender were identified from local person-registration lists. Information on occupational history, occupational exposures, alcohol and tobacco consumption was obtained by postal questionnaire. All RRs, estimated from odds ratios, were adjusted for age, alcohol and tobacco consumption. The RR for laryngeal cancer in unskilled/semiskilled workers was significantly elevated (RR 1.8, 95% CI 1.3-2.6). The RRs for skilled and salaried workers or professionals were not elevated. Of the occupational materials, the RR was elevated only for 'any occupational dust' (RR 1.6' 95% CI 1.2-2.0). The RR for exposure to cement was not elevated, at 0.9 (95% CI 0.6-1.3, 42 cases, 148 controls). Among 7 occupations listed with significantly elevated RRs was employment in concrete, cement and asbestos cement manufacture (RR 17.3). This RR related to just 6 cases and 2 controls. As this grouping included asbestos cement workers, and involved a small number of subjects, no conclusions about cement dust can be drawn from this observation.

Cauvin et al. (1990) studied all 2471 cases of squamous cell cancer of the oropharynx, hypopharynx, glottis, supraglottis, epilarynx and oral cavity identified at a Paris hospital between 1975 and 1984. The controls were 147 males, who were all patients attending the hospital during the study period who were healthy or with a cancer at another site. Information on occupational history, occupational exposures and alcohol and tobacco consumption was obtained by interview. The OR for the exposure category of 'cement, stone' was significantly elevated for cancers at only one of nine sites analysed, the supraglossis. After adjustment for alcohol and tobacco consumption the OR was 2.4 (95% CI 1-5.8). However, this could be a chance finding as elevated risk was limited to one tumour site. The exposure group included exposure to stone, so the role of cement dust exposure is even more uncertain.

The possible occupational risk factors for laryngeal cancer were investigated on the Texas Gulf Coast (Brown et al. 1982). The cancer cases were obtained from hospital records and tumour registries at 56 (out of possible 67) hospitals in the study area. All diagnoses of laryngeal cancer for white males aged 30 to 79 living in the study area during the period mid-1975 to mid 1980, totalling 303 cases, were identified. After exclusions, a group of 183 men (136 living and 47 dead) were studied. Controls were a sample of males, matched for ethnicity, age, and vital status, identified from various local records. Information on demographics, occupational history, tobacco and alcohol consumption and diet was collected by interview of participants or close relatives. Employment in the construction industry was one of the occupations with a significantly increased RR, after adjustment for tobacco and alcohol consumption (RR 1.70, 95% CI 1.06-2.72). However, this group would have been exposed to a wide range of substances so it not possible to relate this finding to cement dust exposure.

Flanders and Rothman (1982) studied 90 male cases of laryngeal cancer identified from a larger cancer study. The control group was 933 males identified with cancers of the haematopoietic system, prostate and rectum. Information on employment, tobacco consumption and demographics were obtained by interview. The alcohol and tobacco adjusted RR for employment in the construction industry was elevated at 1.5 (90% CI of 0.7-3.2). Among specific occupations, the RR for construction labourers was 1.3 (90% CI 0.4-4.6). Again, this study provided no information specifically on cement dust as a risk factor.

Flanders et al. (1984) studied 42 patients with newly diagnosed and histologically confirmed squamous laryngeal cancer attending a US group of hospitals between 1974 and 1979. Eighty-five control subjects, matched for age, gender, residence area, tobacco and alcohol, were selected from non-cancer hospital patients. Workers who may have been exposed to cement were grouped into a very heterogenous group of 'labourers or maintenance personnel', which showed a significantly elevated estimated RR (4.6, 90% CI 1.6-13.6). RRs for farmers and textile processors were also elevated. This small study provided no information on the potential for cement dust to cause cancer.

Zagranski et al (1986) considered male patients attending two US hospitals with histologically confirmed laryngeal cancer first diagnosed between 1975 and 1980 and who were alive at the time of the study (1980-1981). Controls were selected from male general surgical patients at the hospitals, matched for year of admission, decade of birth, county of residence, ethnicity and smoking status. After refusals and exclusions 92 cases and were 181 controls were studied. After adjustment for tobacco and alcohol consumption, the OR for working in the construction industry was less than 1. By occupation, the OR for ever working as a mason was elevated, at 2.2 (95% CI 0.9-5.8). Masons will have been exposed to dusts other than to just cement and a specific role for cement in causing laryngeal cancer cannot be identified from this study.

Rothman et al. (1980) reviewed the epidemiology literature for laryngeal cancer. The pre-1980 literature did not identify cement dust exposure as a risk factor, but it must be stated that occupational risk factors had not been adequately studied at this time. The review identified strong associations between both smoking and high alcohol consumption and laryngeal cancer.

Overall, the earlier studies of risk factors for laryngeal cancer provide limited support for the existence of an association between working in the construction industry and laryngeal cancer, but provide no useful information about the risks associated with exposure to cement dust *per se*.

3.4.2.4 Other recent mortality and cancer-related studies involving subjects with exposure to cement dust.

These studies have only limited relevance to the assessment of the hazardous properties of Portland cement, and are only briefly considered.

Potential occupational risk factors for multiple myeloma were investigated in Swedish construction workers (Lee et al. 2003). Incident cases of multiple myeloma for the period 1971-1999 among 365,000 members (about 80% of eligible workers) of a Swedish construction industry health care scheme were identified from the Swedish Cancer Registry. Occupational exposures were assessed in a semiquantitative manner on the basis of industry job codes. A total of 446 primary multiple myeloma cases were identified. The RR for cement exposure 1.0 (95% CI 0.76-1.28, 49 cases) after adjustment for age and body mass index, showing that cement was not a risk factor for multiple myeloma.

Szadkowska (2001) conducted a nested case control study in pulp and paper workers. Three cases of lung cancer among persons with previous exposure to 'cement, brick, sand, grindstone dust' were identified, compared with none with such exposure among controls. No conclusions about the effects of cement dust can be drawn from this study.

A retrospective cohort mortality was conducted in members of a US plasterers and cement masons trade union (Stern et al. 2001). All members dying during the periods 1972-1980 and 1984-1996 were included. There were 7811 cement masons in the cohort, whose exposures were said to include cement dust, silica, asphalt and various solvents. Compared to the general population, cement masons had significantly higher mortality for cancer of the stomach (proportionate cancer mortality ratio 133, $p < 0.01$) and for benign neoplasms (proportionate mortality ratio 132, $p < 0.01$). Because of the wide range of occupational exposure experienced by the cement masons no conclusions about the effects of cement dust can be drawn.

Krstev et al. (1998) conducted a death certificate based case control study of prostate cancer in 24 US States. The occupations of 60900 men with prostate cancer as a cause of death were identified and compared with age matched controls that had died of causes other than cancer. 'Supervisor of brick

maisons' was one of over 70 occupations with a significantly increased odds ratio for prostate cancer. The extent of cement exposure experienced by this occupational group was not reported.

Cancer in Illinois construction workers was investigated in a series of case control studies using subjects from the state Cancer Registry (Keller and Howe 1993). The risk of lung cancer was greater for construction workers than the general population of Illinois (OR 2.11, (95% CI 1.02-1.26), but this was thought to be due to higher tobacco consumption among the construction workers. Cement exposure was not identified as a risk factor for cancer.

In a briefly reported study, the frequencies of sister chromatid exchanges (SCEs) in the lymphocytes of workers from an Indian Portland cement factory were measured (Fatima et al 1995). The exposed subjects were 59 non-smokers, age range 24-54 years, who had been exposed to cement for 1-17 years. The mean number of SCEs in the exposed workers was 8.98 per cell, compared with 3.5 among the controls. Within the exposed group the results were analysed according to whether the subjects had been exposed for 1-5, 6-11, or 12-17 years. The frequency of SCEs appeared to be positively related to exposure duration, but this could have been due to age differences between the exposure groups, which were not taken account of in the analysis. This is a briefly reported investigation, with results that are without corroboration from other studies. Furthermore, the health significance of such an increased frequency of SCEs is not known. Therefore, no conclusions about the hazardous properties of cement dust can be drawn from this study.

3.4.2.5 Summary of mortality and cancer studies involving workers with long-term exposure to cement dust

HSE (1994) came to a position that there was no convincing evidence for an increased incidence of any site-specific cancer resulting from cement exposure, but acknowledged that the data available at that time were not consistently and reassuringly negative. Since the early 1990s, a number of relevant cancer cohort and case control studies have been published, conducted in groups exposed to cement either through employment in cement manufacturing or in the construction industry. These studies have identified associations between cement exposure and cancers at several specific sites: the stomach, lungs, colon, and head/neck. The strength of evidence supporting a causal association of each of these cancers with Portland cement exposure is discussed with reference to strength of association, consistency, the presence of a dose-response relationship and biological plausibility. Evidence from experimental studies in animal or in vitro models, which can contribute to arguments for or against causality, are not available.

Elevated SMRs for stomach cancer were seen in three out the four cohort studies conducted in cement manufacturing workers covered by HSE (1994). One cohort study in cement manufacturing workers included in this review, conducted in Lithuania, identified a dose-related trend towards an increased risk of stomach cancer, although the numbers of cases involved was very small. In contrast, cohort studies conducted in two Swedish Portland cement

plants and a Danish cement plant did not show increased risks for stomach cancer. There is a degree of consistency, since an association has been seen in four out of the six available cohort studies. The strength of association is moderate, with SMRs ranging from 1.19 to 1.77. In two studies there was evidence for a dose-response relationship, but the evidence was far from convincing because of the small number of cancer cases in each exposure subgroup. Overall, the case for causality is stronger than for the cancers at other sites, but the recent data does not change the picture of uncertainty presented in the early 1990s.

An association between cement exposure and lung cancer was seen in a cohort study in Lithuanian cement manufacturing workers but this could have been due to smoking. A study in Icelandic cement masons showed an increased risk of lung cancer in comparison with the general population, reporting an SIR of 1.77 (95% CI 1.01-2.88) when allowing for a latency period of at least 30 years. Overall, evidence for causality is very weak because consistency across studies in different populations is lacking. Furthermore, the strength of the observed association was moderate, and dose-response information is not available.

A comprehensive cohort investigation into cancer at two Swedish cement manufacturing plants showed an increased risk of right-sided colon cancer among workers with long-term cement exposure. However, the right side of the colon, or colon generally, was not identified as a cancer site associated with cement exposure in the other cohort studies. Again, evidence for causality must be considered to be weak because an association has not been observed in studies in different populations. However, the strength of association was greater than found for colon and lung cancers (SIR 4.3, 95% CI 1.7 - 8.9 for workers with more than 25 years exposure) and there was some evidence suggesting an increased risk with increasing duration of exposure.

Concerning cancers of the head and neck, a series of recent population based case control studies conducted in Germany demonstrated an association between contact with cement through employment in construction and cancers at these sites, especially cancers of the pharynx and larynx. Earlier case control studies, conducted elsewhere, investigating the risk factors for laryngeal cancers provided limited evidence of an association between employment in the construction industry and an increased risk of laryngeal cancer, but these studies provided no useful information on the risks associated with exposure to cement *per se*. No information on the association between these types of cancer and cement can be derived from the cohort studies because head and neck cancers are rare and the cohort studies lacked the power to investigate such cancers. The strength of association between occupational contact with cement and cancer of the head and neck was generally moderate, with ORs or estimated RRs ranging from about 1.2 to 2.4, although for one study the association was very strong as shown by an OR of 12.6. The association showed consistency within the series of studies conducted in southern Germany but only limited corroborative support is available in terms of consistency from studies of laryngeal cancer in other

populations. A dose-response relationship has not been demonstrated. Furthermore, the exposure assessments in these studies did not determine the quantitative nature of the cement exposure, or take account of other exposures that could be experienced by persons who have occupational contact with cement, so there is no clear picture of what the subjects were exposed to. Overall, evidence for a causal association between cement exposure and head and neck cancer is weak.

Biological plausibility is considered as for all cancers together. As a highly alkaline substance cement can cause irritation at sites of contact, such as the mouth, throat, lungs and, for ingested material, the gastrointestinal tract. Persistent chronic irritation will cause repeated cycles of cell death, cell proliferation and other inflammatory responses. It is recognised that this process can be a step on the pathway to cancer. Thus, although there are no specific data for cement in support of this possible mode of action, it is biologically plausible that cement dust could have the potential to cause cancers at sites of contact.

To conclude, a causal association between Portland cement exposure and cancer has not been established. Nevertheless, the findings of the recent studies reviewed in this document add to the concerns for cancer raised by the earlier data reviewed by HSE (1994).

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Table 1 Summary of recent studies of non-malignant respiratory disease in workers with long-term exposure to cement dust

Reference	Country	Study subjects	Main investigations	Main findings in cement workers
Fell et al. 2003	Norway	119 cement factory workers, mainly retired. 50 blue collar controls, matched for age	Respiratory questionnaire, spirometry. Current dust measurements.	No differences in respiratory health between cement workers and controls
Mwaiselage et al. 2004	Tanzania	115 cement factory workers 102 controls, with maintenance or office jobs at factory	Spirometry, peak flow. Current dust measurements.	Reduction in FVC, FEV ₁ , FVC/FEV ₁ , PEFR, negative correlation with cumulative exposure estimates
Laraqui Hossini et al. 2002	Morocco	280 cement factory workers. 73 controls with office jobs.	Respiratory questionnaire, spirometry. No exposure data, but dust levels high.	Higher prevalence of respiratory symptoms and minor spirometry deficits
Meo et al. 2002	Pakistan	50 cement factory workers. 50 matched population controls.	Spirometry. No exposure information	Reduction in FVC and FEV ₁ . Increase FEV ₁ /FVC ratio.
Al-Neaimi et al. 2001	UAE	75 cement factory workers. 134 matched controls	Respiratory questionnaire, spirometry. No exposure information.	Higher prevalence of respiratory symptoms. Reduction in FVC, FEV ₁ and FEV ₁ /FVC ratio
Noor et al. 2000	Malaysia	62 cement factory workers 70 controls from university.	Respiratory questionnaire, spirometry. Current dust measurements.	Higher prevalence of respiratory symptoms. Reduction in FVC, FEV ₁ and FEV ₁ /FVC ratio.
Mengesha et al. 1998	Ethiopia	53 cement factory workers. 211 controls, non-dusty occupations	Respiratory questionnaire, spirometry, peak flow. Current dust measurements.	Higher prevalence of respiratory symptoms. FEV ₁ and FEV ₁ /FVC ratio lower in the group with highest cement exposure.
Abuhaise et al. 1997	Jordan	348 cement factory workers, divided into 3 exposure groups	Respiratory questionnaire, spirometry Current dust measurements.	Higher prevalence of asthma in those with highest exposure. Spirometry similar for all 3 groups.
Chanrawanshi & Pati 1996	India	365 cement factory workers. 45 controls	Peak flow. No exposure information	PEFR lower among cement workers who worked rotating shifts
Yang et al. 1996	Taiwan	412 cement factory workers. 147 controls with office jobs	Respiratory questionnaire, spirometry Current dust measurements	Higher prevalence of respiratory symptoms. Reduction in FVC, FEV ₁ and FEV ₁ /FVC ratio
Abrons et al. 1998	USA	2640 cement factory workers. 1458 blue collar controls	Radiography. Current dust measurements available	Increase prevalence of radiography-detected minor abnormalities of lungs, confined to mainly to smokers
Alvear-Galindo et al. 1999	Mexico	425 cement factory workers	Respiratory questionnaire. Semi-quant exposure assessment	Prevalence of respiratory problems highest among those with highest exposure scores
Abou-Taleb et al. 1995	UAE	304 cements factory workers	Respiratory questionnaire	Chronic cough in 19%, chronic bronchitis in 12% of workers, but no control group for comparison
Yang et al. 1993	Taiwan	661 cement factory workers, including 146 office workers	Respiratory questionnaire. Current dust measurements	No differences between high medium and low exposure groups
Laraqui et al 2001	Morocco	120 concrete factory workers. 120 controls, civil servants	Respiratory questionnaire	Higher prevalence of respiratory symptoms among concrete workers

Fell et al. (2003) is a retrospective cohort study, the remainder are cross-sectional studies

Table 2: Summary of mortality and cancer morbidity studies in cement manufacturing workers or masons

Reference	Country	Study subjects	Main investigations	Main findings
Jakobsson et al. 1993	Sweden	2400 cement factory workers, at two plants	Mortality and cancer morbidity, compared to general population	Overall mortality and cause-specific non-malignant mortality for the cement workers was not affected. Risk of tumours of right side of colon increased (SIR 2.7, 95% CI 1.4 - 4.8).
Jakobsson et al. 1994	Sweden	1526 cement factory workers 3965 blue collar workers 8092 fishermen	Mortality and cancer morbidity, compared to general population	Risk of tumours of right side of colon increased for cement workers (SIR 2.6, 95% CI 1.4-4.6). Cancer risks for other groups similar to that of the general population
Vesbo et al. 1991	Denmark	546 cement factory workers 856 unexposed controls	Cancer morbidity	No evidence of an increased risk of cancer for cement workers
Smailyte et al. 2004	Lithuania	2498 cement factory workers	Mortality and cancer morbidity, compared to general population	Increased incidence of lung cancer among cement workers (SIR 1.5, 95% CI 1.1-2.1), but smoking was not taken account of in the analysis. Dose-related trend towards an increased incidence of stomach cancer.
Rafnsson et al. 1997	Iceland	1172 masons, using wet concrete to finish buildings	Cancer morbidity, compared to general population	Increased incidence of lung cancer among masons (SIR 1.7, 95% CI 1.1-2.5)

Table 3 Summary of German case control studies for cancers of the oral cavity, pharynx and larynx

Reference	Location	Study population	Main findings
Maier et al. 1991	Heidelberg, Giessen	200 male cases of cancer of the oral cavity, pharynx and larynx 800 male hospital patient controls, matched for age and size of home	Elevated risk of cancer associated with occupational exposure to cement. Estimated RR 2.4 (95% CI not stated) after adjustment for smoking and alcohol consumption
Maier et al. 1992	Heidelberg	164 male cases of cancer of the larynx 656 male hospital patient controls, matched for age and size of home	Slightly elevated risk of cancer associated with occupational exposure to cement. Estimated RR 1.18 (95% CI 0.9-1.4) after adjustment for smoking and alcohol consumption
Maier et al. 1994	Heidelberg	105 male cases of cancer of the pharynx 656 male hospital patient controls, matched for age and size/location of home	Elevated risk of cancer associated with occupational exposure to cement. Estimated RR 2.2 (95% CI 0.9-5.2) after adjustment for smoking and alcohol consumption
Maier et al. 2002	Heidelberg	209 male cases of cancer of the oral cavity, pharynx and larynx 110 male hospital patient controls, matched for age, smoking and alcohol consumption	Substantially elevated risk of cancer associated with occupational exposure to cement. The OR was 12.6 (95% CI 3.9-43.0).
Dietz et al. 2004	Rhien-Neckar region	257 cases of laryngeal cancer 769 controls from the general population, matched for age, gender and location of home	Elevated risk of cancer associated with occupational exposure to cement. The OR was 1.2 (95% CI 0.8-1.2) assessed by substance questionnaire or 2.0 (95% CI 1.2-3.6) assessed by job-specific questionnaire, after adjustment for smoking, alcohol consumption and socio-economic status.