

**REVIEW OF THE EVIDENCE FOR CHRONIC RESPIRATORY
ILL HEALTH IN CONSTRUCTION WORKERS**

CONTENTS

EXECUTIVE SUMMARY	4
INTRODUCTION.....	7
BACKGROUND	7
OCCUPATIONS AND EXPOSURES IN THE CONSTRUCTION INDUSTRY.	8
HEALTH EFFECTS	10
COPD	10
Emphysema	11
Chronic bronchitis	11
Pneumoconiosis	12
Silicosis	12
EVIDENCE BASE.....	13
Large-scale general population studies.....	13
General population studies with lung function tests	14
Studies reporting on respiratory symptoms.....	15
Studies reporting on physician-diagnosed COPD	18
Information from published statistics in the UK	19
Summary of the general population studies.....	20
Mortality studies	21
Mortality studies in construction workers.....	21
Mortality studies in specified construction trades.....	24
Heavy equipment operators	24
Asphalt workers	25
Roofers and waterproofers.....	27
Road construction workers	27
Labourers.....	27
Plasterers	28
Construction carpenters (joiners)	28
Ironworkers	29
Electricians	29
Bricklayers.....	29
Construction painters	30
Summary of mortality studies	30

Case reports	31
Cross-sectional studies	32
Studies in various groups of construction workers	32
Studies informing on silicosis/mixed-dust pneumoconiosis	36
Summary of studies informing on silicosis/mixed-dust pneumoconiosis ..	41
Studies in insulation workers	42
Painters	44
Asphalt workers	46
Electricians	47
Carpenters	48
Tunnel workers	48
Summary of studies in tunnel workers	55
Summary of the cross-sectional studies	56
Longitudinal studies	58
Case-control studies	58
DISCUSSION	59
REFERENCES	62

EXECUTIVE SUMMARY

The aim of this review was to evaluate the evidence for an association between respiratory ill health and construction work, focussing on the conditions of chronic obstructive pulmonary disease (COPD), and silicosis/mixed-dust pneumoconiosis. A further aim was to identify causal substances, and particular tasks and construction trades at most risk.

There are numerous studies that provide information on respiratory ill health in construction workers, including population-based prevalence surveys, mortality studies and cross-sectional workplace studies. However, few studies provided information on exposure conditions or potential causative agents.

The findings from 10 general population surveys were broadly consistent in showing that construction workers tend to have increased prevalences (averaging around 2 to 3-fold higher) of COPD and of symptoms of chronic bronchitis compared to age-and smoking-matched controls from other occupational groups, although the magnitude of the odds ratios varied from study to study. In the one study in which it was investigated, the severity of COPD appeared worse in retired construction workers compared to retired workers from other “blue-collar” occupations. The general population surveys do not provide any information on particular construction trades at most risk. Most studies did not explore causative agents except at a very general level, and noted that dusts, including wood dust and silica, were associated with COPD and respiratory symptoms.

There are numerous studies that inform on mortality in construction workers. Overall, the findings reveal a moderate excess mortality from COPD in construction workers, with statistically significant PMRs/SMRs ranging between 109-207. However, as smoking status was not taken into account in any of these studies, it is uncertain to what extent occupational exposures may have contributed to these findings. In a study of over 300,000 Swedish construction workers with over 30 years of follow up, the proportion of COPD mortality attributable to dust/irritant exposure was estimated at 10.7% in the overall cohort and 52.6% in the never-smokers. These estimates suggest that smoking is the predominant cause of COPD mortality in construction workers but that occupational exposures are also involved. In terms of specific trades at most risk, it is difficult to draw any firm conclusions from the available mortality studies, mainly because generally only one or two isolated studies exist for any individual trade.

The findings from workplace cross-sectional studies generally indicate prevalences of COPD/chronic respiratory symptoms in construction workers of about 12-22%. Although external control groups were often not included for comparison, given that construction workers are often engaged in physically demanding work and should therefore be in relatively good health, these prevalences are suggestive of an effect of occupation. Although there are studies that have investigated individual construction trades such as painters, asphalt workers, electricians and carpenters, they are of limited usefulness in terms of identifying trades that may be at particular risk, mainly because generally only one or two isolated studies exist for any individual trade.

Tunnel workers are the one construction trade that has been investigated in most detail particularly with respect to exposure conditions, although only a few studies are available. Evidence from Norway and Germany showed that tunnel workers were at increased risk of an accelerated decline in lung function of about double the normal

age-related decline. In the Norwegian tunnel workers, this decline was found to be particularly associated with exposures to respirable dust, silica and to nitrogen dioxide, but because of the mixed exposure conditions other substances may also have contributed to the decline in lung function. A study in Norwegian underground tunnelers found a prevalence of COPD of 14% as compared with a prevalence of 8% in outdoor construction workers. Overall, with the possible exception of tunnel workers, it is difficult to single out individual construction trades as being at particularly increased risk of respiratory ill health, although it should be noted that in general, in all of the trades studied, there was generally a higher prevalence of COPD and/or chronic respiratory symptoms than in external reference groups.

Overall, the findings from numerous studies of different designs and in different countries are reasonably consistent in indicating that construction workers show moderate increases (2 to 3-fold) in the COPD morbidity and mortality compared to age- and smoking-matched reference groups. Although a number of studies consistently point to a role of dusts and irritants in general, given the diversity of sectors, trades, activities and workplaces investigated, the available data do not allow one to specify with sufficient reliability a particular causative agent or agents. Information on specific trades is limited, but tunnel workers clearly stand out as being at higher risk compared to outdoor construction workers.

There is very little data relating specifically to UK construction workers. However, it is noted that many of the studies are relatively recent and all are based on industrialised countries such that it is reasonable to infer that the findings would be broadly relevant to current UK construction workers.

There is a less substantive body of evidence concerning silicosis/mixed-dust pneumoconiosis than for COPD. There are reports of silicosis deaths in construction workers, including plasterers, cement masons and road construction and maintenance workers. However, often these reports refer to only a very small number of deaths drawn from very large worker populations, suggesting that these may be relatively isolated cases. There are no reports of silicosis deaths in UK construction workers.

In the available cross-sectional studies, the prevalence of silicosis/mixed-dust pneumoconiosis in construction workers has not been systematically or widely investigated. Evidence from a single study each in Dutch and Swedish construction workers suggests that there is likely to be a low prevalence (around 1%) of silicosis in general construction workers, but of a low radiological category (ILO 1/0 and ILO 1/1). A higher prevalence of silicosis (16%) was found in a study in Hong Kong underground caisson workers. In a study of over 2,000 mixed construction workers employed at US nuclear plants, there was a 2.2% prevalence of pneumoconiosis (ILO category $\geq 1/0$), and in another study of US construction painters the prevalence was 3.7%, and 3.3% in the reference group of sheet metal workers. In a Swedish study of nearly 6,000 construction workers, the prevalence of pneumoconiosis (ILO category $\geq 1/1$) was 4.4%. In a study of about 1,300 Dutch construction workers from building sites there was a prevalence of pneumoconiosis (ILO categories 1/0 and $\geq 1/1$) of approximately 10% and 3% respectively.

Overall, there would appear to be a rather low prevalence (around 1%) of silicosis of low radiological severity in general construction workers, but particular trades such as caisson workers may be at particular risk. A higher prevalence (2-10%) of mixed-dust pneumoconiosis, also of low radiological grade, has been found. Unfortunately, no

reliable exposure information is available to characterise dose-response relationships. These radiological changes are likely to be of little or no health significance, although it should be noted that these studies were cross-sectional in design and so lack information on the progression of pneumoconiosis over time.

There are no documented cases of silicosis in UK construction workers although 27 German construction workers received compensation for silicosis in 1998, suggesting that there may be some undetected cases in the UK. Overall, it is difficult to draw any firm conclusions concerning pneumoconiosis risks in UK construction workers except perhaps that there is likely to be a relatively widespread prevalence (up to 10%) of pneumoconiotic changes of low radiological severity, such that the health impact is likely to be little or none.

INTRODUCTION

The aim of this review is to characterise the evidence on the extent of respiratory ill health in construction workers, and to identify where possible the causes (substances/processes) and specific occupational groups at most risk.

The review should therefore form a significant contribution to the evidence base on occupational lung disease for the Disease Reduction Programme (DRP), and should help facilitate decisions regarding whether or not respiratory disease in construction workers deserves further attention.

The ill health conditions covered in this review are chronic obstructive pulmonary disease (COPD), chronic bronchitis and emphysema, and silicosis/mixed-dust pneumoconiosis. Asthma, respiratory tract cancers and diseases related to asbestos are not included in the scope of this review given that such conditions are dealt with elsewhere within the DRP.

The information presented in this review derives from a range of epidemiological studies including mortality studies, cross-sectional (prevalence) surveys, a limited number of prospective studies, case-control studies, as well as a number of population-based prevalence studies.

The studies evaluated in this review were located by a Medline search of the scientific literature reaching back from the present day to the mid-1980s. Search terms such as “COPD”, “chronic bronchitis”, “emphysema”, “respiratory symptoms”, “silicosis”, “pneumoconiosis”, “respiratory ill health” and “construction” were used. Additional studies cited in the identified papers but not located by Medline were also evaluated.

BACKGROUND

The reason for undertaking this review is that in the early stages of the HSE DRP, when the evidence-base for occupational lung diseases was first being compiled, it was observed that a number of population surveys pointed to a possible association between construction work and an increased risk of COPD. Subsequently, HSE decided that work-related COPD should be a priority within the respiratory disease element of the DRP. This decision was based on evidence indicating that occupational exposures to dusts and irritants is responsible for about 15% of all cases of COPD in society (ATS, 3003). Given the serious nature of this condition, and the significant morbidity and mortality associated with COPD, it is important to identify the industries/occupations at most risk. In terms of ranking or prioritising such industries/occupations, the size of the industry and the number of workers affected needs to be taken into account. The DTI website (www.dti.gov.uk/construction/stats/050304/pressrel050304.htm) estimates that a total of 1,600,000 persons were employed in construction in October 2003.

Other industries/occupations suggested by the results of population surveys to have increased prevalences of COPD include agriculture, rubber, plastics, leather and synthetics manufacture, textile mill products manufacture, food products manufacture, bakers and cleaners. HSE has already commissioned work to further characterise the extent of occupational ill health in the agricultural sector, but there is nothing of specific relevance to COPD for construction work. Overall, given the size of the construction sector in the UK, and the absence of documented evidence concerning

respiratory health in UK construction workers, it was deemed worthwhile to explore the published literature to try to characterise this issue further.

Respirable crystalline silica (RCS) is another priority within the respiratory disease element of the DRP. HSE is currently carrying out a survey to obtain measured exposure data for RCS in four industry sectors where there is judged to be the most concern for exposure but for which there is little or no current measurement data available: these sectors are construction, quarrying, brick manufacture and stonemasonry. Although it is known that certain tasks within the construction industry have the potential for high exposures to dusts containing RCS, there is no data on the possible extent of silicosis/mixed-dust pneumoconiosis in UK construction workers. It therefore seemed appropriate that this review should cover not only COPD but also silicosis and mixed-dust pneumoconiosis.

Although there is no direct evidence from published literature or from UK sources such as the Industrial Injuries Scheme on work-related COPD or pneumoconiosis in construction workers, based on the information presented in this review, WATCH members are asked if in their opinion, any inferences on the risk of these diseases may be drawn based on the evidence from other countries.

OCCUPATIONS AND EXPOSURES IN THE CONSTRUCTION INDUSTRY

The construction sector includes a large set of diverse trades: labourers, carpenters, plasterers, painters, brick layers, stone masons, craft workers, electricians, plumbers, concrete and terrazzo finishers and glaziers, sandblasters, mechanics and repairers, drywall installers, roofers, asphalt workers, insulators, ironworkers, engineers, tunnel workers, welders and cutters. Since welding involves specific exposures to welding fumes, data on welders will not be dealt with by this review.

The construction industry is vast and encompasses a very large range of activities and workplaces. The construction environment is dynamic, with frequent adoption of new processes and materials. Construction is not only defined as new construction, but also includes a range of activities such as plant refits, specialised and ongoing maintenance, renovations and repairs.

Exposures to a number of pollutants may be intermittent, with short-term excessive peak exposures coupled with intervals of low exposure. Because each construction worker moves about a site, the worker's position in relation to exposure sources may change regularly. At some moments, a worker may be using and be directly exposed to a hazardous substance, but at other times the worker may be exposed to other substances as a bystander downwind (para-occupational exposure). It is therefore difficult to anticipate all the substances and degrees of exposure that someone will encounter on a given day. Construction work sites are usually complex environments where exposures to mixtures of agents under a variety of outdoor, indoor and confined space settings are common. Diverse materials or contaminants may be encountered during demolition or renovation at construction sites, producing unpredictable and variable mixes of workplace exposures. In addition to the transient and serial nature of exposures at construction sites, the diversity of construction-related sectors (commercial, residential, industrial, etc.), trades and activities that may coexist at a particular work site also contributes to the complexity of the exposure. Because of

this, most occupational health studies of construction workers tend to focus on occupation and tenure rather than attempting quantitative exposure assessment. In addition, attributing a health response to any one agent is often not feasible, particularly with health effects such as COPD, which has multiple potential causes.

The mobility of the construction workforce poses special problems for epidemiological research, which ideally requires a stable workforce amenable to follow-up investigation. Trade union membership provides a convenient way to identify and access potential subjects, but only 20% of known construction workers are unionised, and other workers may work off-the-record for independent construction contractors (Ringen et al., 1995).

Construction workers are potentially exposed to a variety of hazardous agents including cement dust, wood dust from sawing, dust from the ground, dust from using pneumatic tools on concrete or other stone work, fumes from welding, roofing or paving, and diesel exhaust from machines. Substances that may pose threats to the respiratory system include asbestos, silica and other mineral dusts, synthetic vitreous fibres (including for example, rock wool), PAHs, metal fumes and dust, formaldehyde, resin adhesives, creosote, gasoline, oils, diesel fumes, vapours and dusts from paints, pitch, sealers, and solvents.

Airborne dust is probably the most prevalent inhalation hazard in construction. Exposure is experienced not only by the worker directly involved, but also by other workers within the work vicinity. For example electricians and glaziers are significantly exposed to particulates generated by other trades. The most significant sources of dust in construction are activities involving the use of power tools, and more specifically hand-held tools, grinding and dry cutting activities, sweeping, fire-proof spraying and heavy machinery (Chisholm, 1999). The highest dust levels are observed during cleanup, demolition, compressed air cleaning, mixing and spraying cement insulation, abrasive blasting, masonry, road milling, concrete grinding, cutting, chipping, and concrete mixing activities. Median 8h-TWA respirable dust concentrations of 13.5 and 11.9 mg.m⁻³ have been reported for compressed air cleaning and concrete cutting (Verma et al., 2003). On a trade basis, labourers, painters and bricklayers have the highest cumulative exposures to respirable dust (Verma et al., 2003). On a task basis, the highest total dust levels have been measured for abrasive blasting and fireproof mixing and the highest respirable dust levels have been measured during dry concrete grinding (Verma et al., 2003). Welding tasks, and in particular gouging tasks, generate high dust levels. Abrasive blasting is considered to be a very dusty task. Alkaline dust tends to be generated in association with concrete activities and in fireproof mixing. Wood dust exposure is significant in residential or commercial construction.

Other important contaminants in construction are gypsum, silicates, diesel exhaust, welding fumes, metals and man-made mineral fibres (MMMF). MMMF (mineral wool, rock wool, slag wool, fibreglass) are found in common construction material such as drywall, roofing material and thermal insulation (Verma et al., 2004). On some occasions, particulates may contain other hazardous materials such as lead or coal tar pitch. Organic solvents and lead tend to be present in industrial painting operations.

Exposure to silica occurs during processing of materials containing sand and stone (e.g. brick, concrete, granite, mortar, tile) with equipment that generates respirable particles, such as grinders, electrical saws, (jack)hammers and drills. Workers significantly exposed to silica are those employed in concrete removal and demolition work, bridge and road construction, tunnel construction, and concrete or granite cutting, chipping, drilling, sanding and grinding. Sandblasters, rock drillers, demolition workers and recess millers have the potential for particularly high silica exposures (Lumens and Spee, 2001). In construction work, materials containing silica do not have a homogeneous composition and a variety of tools generate dust with different characteristics (Nij et al., 2004). The silica content of building materials may vary from negligible in gypsum to 40% in sand lime. Exposure levels to respirable silica have been reported to be as high as 10.0 mg.m⁻³ (8h-TWA) for concrete sawing and abrasive blasting (Linch, 2002) and 26.2 mg.m⁻³ (8h-TWA) for painters (Rappaport et al., 2003). In general, respirable silica levels for certain tasks in the construction industry can be high and have been reported to exceed national occupational exposure limits (Nij et al., 2003a; HSE, 1998).

HEALTH EFFECTS

There are a number of respiratory ill health conditions that may be associated with work in the construction industry as a result of long-term repeated exposures to dusts and irritants. This review concentrates on the evidence relating to chronic obstructive pulmonary disease (COPD), a condition that encompasses chronic bronchitis and emphysema, and silicosis and mixed dust pneumoconiosis. Other respiratory conditions such as asthma, lung cancer and asbestosis, are not included in the scope of this review because they are covered elsewhere in HSE's Disease Reduction Programme. This section is provided to help in the interpretation of the health effects information covered in this review.

COPD

COPD is characterised by airflow obstruction that is not fully reversible. The condition is usually progressive and associated with abnormal inflammatory responses of the lungs to noxious particles or gases. The signs and symptoms of COPD include chronic cough and sputum production, and shortness of breath. The diagnosis is confirmed by spirometry. The criteria for diagnosis are the presence of FEV₁¹ <80% of the predicted value in combination with an FEV₁/FVC² ratio <70%. Chronic cough and sputum production may precede the development of airflow obstruction by many years; although not all individuals with chronic cough and sputum production go on to develop COPD (GOLD 2003). Conversely, not all individuals with airflow obstruction suffer from cough and sputum production.

COPD is a slowly developing condition that is not usually manifest until midlife, and it would be very unusual for COPD to be diagnosed in someone <40 years old. Thus, it is important to look at the age distribution of subjects in the studies in construction workers to ensure that the relevant age group is covered.

¹ FEV₁ is the Forced Expiratory Volume in one second

² FVC is the Forced Vital Capacity

In normal adults, FEV₁ declines by 20- 30 ml per year. An accelerated annual decline in FEV₁ is an indication of COPD development. In subjects who develop COPD, FEV₁ declines by more than 40 ml per year (MacNee and Zielinski, 2000), indicating an annual loss of 10 ml over and above the normal age-related decline. Reductions in FEV₁ have been shown to be predictive of premature mortality (Peto et al., 1983).

The main cause of COPD in the general population is cigarette smoking. Individuals with COPD caused by occupational exposures cannot be distinguished clinically from those in whom the disease is caused by cigarette smoking.

Emphysema

COPD is an umbrella term that mainly covers chronic bronchitis and emphysema. Often these two conditions co-exist, particularly as they advance in severity. Many of the studies evaluated in this review have investigated the prevalence of chronic bronchitis in construction workers, but the only data on emphysema in construction workers comes from mortality studies where emphysema has been given as a cause of death on death certificates.

Emphysema is a condition in which there are enlarged alveolar airspaces with destruction of alveolar walls. The condition leads to a loss of elasticity in the lung tissue and a loss of structural support for the small airways in the lungs. This causes premature closure of the airways on exhalation, causing airways obstruction (airflow limitation). Emphysema is diagnosed by pathological examination of lung tissue, but may also be indicated using certain radiological techniques. Traditional chest X-rays would not usually be sufficient to provide a reliable diagnosis of emphysema.

Chronic bronchitis

Chronic bronchitis is a condition in which there is excess mucus production in the bronchial airways. This is associated with an increase in the volume of submucosal glands and the numbers of goblet cells in the bronchial airways. The composition of the mucus in chronic bronchitis may be altered (increased viscosity and thickening) and this may lead to an increased risk of respiratory tract infection. The mucus leads to irritation of the airways and hence coughing.

The diagnosis of chronic bronchitis is based on the reporting of respiratory symptoms and these are typically investigated in epidemiological studies using a questionnaire. There are a number of validated questionnaires available; chiefly there is a questionnaire originally produced by the British Medical Research Council (BMRC) but there are other questionnaires available. The questionnaires typically investigate smoking history, and ask questions on a range of respiratory symptoms, such as difficulty in breathing when walking on the level with someone of one's own age. However, the epidemiological definition of chronic bronchitis is the presence of chronic cough with sputum production, daily for at least 3 months of the year, for at least the preceding two years. Some questionnaires only ask about current symptoms and so are not able to reliably identify chronic bronchitis.

Symptoms data can be subject to seasonal variability and so the design of the study including the use of an appropriate control group, needs to be carefully considered in the interpretation of results. It should be noted that there is often a poor correlation

between the presence of symptoms and reductions in FEV₁. This may be because the development of excess mucus production in the bronchial airways and the changes in the smaller airways and lung parenchyma leading to airflow limitation, are separate consequences of exposure to dusts and irritants.

In some epidemiological studies, clinical examinations are conducted in addition to administration of a symptoms questionnaire. These examinations may involve auscultation (listening to breath sounds with a stethoscope). This may identify a range of abnormal sounds including those referred to as “crackles or rales” usually in the base of the lungs, and “rhonchi”, emanating from the larger airways. Such sounds usually indicate the presence of fluid secretions, such as pulmonary oedema or mucus, and may relate to the presence of COPD or chronic bronchitis; however they may be due to other conditions including infections. Hence the results from auscultation are probably of limited usefulness for the purposes of this epidemiological review, but are included when available in order to provide a comprehensive account of the evidence.

Pneumoconiosis

This is a condition in which there is dust accumulation in the lungs, which may or may not be accompanied by fibrosis (scarring) of the lungs, leading to changes that are visible on chest X-ray. There are various types of pneumoconiosis depending on the type of dust exposure, the principal types being silicosis, asbestosis and coalminers’ pneumoconiosis (or black lung). The pattern of fibrosis and consequent chest X-ray changes may allow discrimination between these conditions. There is also mixed-dust pneumoconiosis (or mixed-dust fibrosis), caused by exposure to mineral dusts containing a small amount of crystalline silica. Cases of mixed dust pneumoconiosis and also cases of silicosis have been reported in construction workers. Silicosis is characterised on chest X-ray by the presence of small rounded nodules predominantly in the upper lobes of the lung, whereas mixed-dust pneumoconiosis is identified as the presence of opacities that are irregular in shape and not restricted to the upper lobes. All of the pneumoconioses are purely occupational in causation i.e they are industrial diseases that do not occur in the general population unexposed to workplace dusts.

The chest X-ray changes associated with pneumoconiosis may be categorised using a scoring system devised by the International Labour Organisation (ILO). This is a 12-point scoring system that is based on scoring the size, shape and profusion of opacities seen on chest X-ray by trained medical readers. A score of 1/0 represents only a mild change that probably has no health significance in itself, although there is a concern it is an early indicator of a condition that may progress in severity. A score of 1/1 is a more pronounced change but is also not likely to be associated with functional impairment. ILO scores of 2/1 or greater are more likely to be associated with some impairment in lung function and respiratory symptoms; however it has been widely observed that there is a lack of correlation between chest X-ray score, respiratory symptoms and lung function.

Silicosis

Silicosis is a slowly developing irreversible lung disease caused by the inhalation of dusts containing respirable crystalline silica (usually in the form of quartz). Quartz is

present in many materials used in the construction industry: sand, concrete, grout/mortar, gravel, bricks, tiles, and stone. Cases of silicosis are diagnosed on the basis of chest X-ray changes in combination with a relevant history of occupational exposure.

Particles of respirable crystalline silica (RCS) cause damage and inflammation in the lungs. Over time, this damage can lead to the development of scar tissue (fibrosis), which shows up characteristically as small rounded opacities on chest X-ray. In severe cases, silicosis leads to premature death. In cases of exceptionally heavy exposure over just a few months or years, the condition of “acute silicosis” can develop which can be rapidly fatal. Cases of acute silicosis have been noted in sandblasters. Silicosis increases susceptibility to infection, and historically was strongly associated with pulmonary tuberculosis.

EVIDENCE BASE

There are numerous epidemiological studies that inform on the nature and extent of respiratory disease in construction workers. These studies range from large-scale surveys in the general population, to specific workplace studies. This review begins with an evaluation of the general population studies, then mortality studies in construction workers, followed by cross-sectional workplace studies. The reason for this ordering is that in the early stages of HSE’s Disease Reduction Programme it was the general population studies that first drew attention to a possible link between work in the construction industry and COPD.

Large-scale general population studies

General population studies gather information from very large numbers of people and thus have the potential to be statistically powerful. However, such studies rely on self-reporting of occupation and/or occupational exposures and usually lack specific information on the features of exposure, although in some studies duration of exposure/employment is assessed. In the investigations included in this section, health information was obtained by questionnaire or telephone surveys, although in some studies the subjects were brought into centres for lung function testing and clinical assessment, giving more reliability to the results.

In general, subjects with asthmatic symptoms were either excluded from the analyses or were analysed separately. Most of the studies in this section are cross-sectional prevalence studies, although there are a few longitudinal studies that have followed a cohort over time. The potential weaknesses of cross-sectional studies, such as failure to take account of workers who have left employment because of work-related ill health, would, if anything, tend to obscure the ability to detect associations between employment and ill health. The general population studies are summarised in tabular form in Table 1 in the Appendix to this document.

General population studies with lung function tests

Data from a survey conducted from 1988 to 1994 in the United States were used to estimate odds ratios (ORs) for the association between COPD and employment (Hnizdo et al., 2002).

The original survey, the Third National Health and Nutrition Examination Survey (NHANES III), was designed to select a representative sample of the US adult population aged 17-90 years (n=20,500). However, only the data for adults aged 30-75 years (n=11,447) were used for this particular study on COPD. After exclusions for reasons such as missing occupational codes or lung function tests, or diagnosis of asthma, there were 9,823 subjects for the final analyses. The authors of the study considered that the large sample size allowed for an unbiased estimation of the effect of confounding factors, and provided varied industrial and job exposures. However, they noted that the original NHANES III survey was not designed to be representative of US employment patterns and may have omitted some high-risk occupations.

Two separate analyses were conducted; one for the longest held job in relation to industry sector, and the other in relation to occupation. For each analysis, office workers comprised the comparison group for calculation of odds ratios (ORs). In this study, COPD was defined as $FEV_1/FVC < 70\%$ and $FEV_1 < 80\%$ predicted.

In the analysis by industry sector, the OR for COPD in construction workers (n=493), adjusted for age, smoking status, pack-years of smoking, body mass index, education and socioeconomic status was increased at 1.3 (95% CI 0.8-2.3) compared to office workers (n=2653). However, when the data for never-smokers were analysed separately, the OR for COPD was 3.5 (95% CI 0.9-14) in construction workers, based on comparison with never-smoking office workers (n=1335).

In the analysis by occupation, for construction trades and labourers (n=456), the OR for COPD in all subjects was 1.2 (95% CI 0.6-2.5) compared to office workers (n=2277). In never-smokers, the OR for construction trades and labourers (n=119) was 3.4 (95% CI 1.1-10.5) based on comparison with never-smoking office workers (n=1159).

The results for both analyses are very similar, and reveal a more prominent relationship between employment as a construction worker and COPD in never-smokers than in smokers.

To help set the results for construction into context; the industry with the highest OR for COPD (all subjects) was rubber, plastics, leather manufacturing (OR = 2.5; 95% CI 1.4-1.4), and the industry with the highest OR (never-smokers only) was utilities (OR 27.7; 95% CI 3.6-214). For occupations, there was not such a great spread in OR values; the occupation with the highest OR for COPD (all subjects) was freight stock, material handlers (OR = 2.2; 95% CI 1.3-3.7) and the occupation with the lowest OR >1 was waitresses (OR 1.1; 95% CI 0.5-2.5). Fourteen occupations with ORs <1 were grouped together as "other occupations" and were not specified further. Many of the raised ORs for particular industry sectors and occupations were not statistically significant. However, the fraction of COPD in the US population attributable to work was estimated at 19.2% overall, and 31.1% among never smokers.

In summary, this was a well-reported large-scale study in which cases of COPD were identified by spirometry according to internationally agreed diagnostic criteria. Two separate analyses, one by industry and one by occupation, indicated higher prevalences of COPD in construction workers compared to office workers of the same

age and other demographic characteristics. However, the increased prevalence of COPD was only statistically significant in the never-smoking construction workers, among whom there was a more than 3-fold increased prevalence of COPD compared to non-smoking office workers.

Fishwick et al (1997) studied the effect of occupation on COPD in New Zealand. The study population (1,609 subjects aged 20-44) was drawn from a random sample of the New Zealand population (only non-asthmatics selected for this study). Subjects completed a respiratory symptoms questionnaire, and pulmonary function testing was conducted; 1132 (70%) of subjects completed satisfactory baseline lung function assessment. The data were used to assess the effect of occupation (current job) on the presence of symptoms suggestive of COPD (cough, phlegm and shortness of breath) either alone or in combination with evidence of mild airway obstruction ($FEV_1/FVC < 75\%$). An odds ratio adjusted for age, sex and smoking status of 2.7 (95% CI 0.7-10.2) for symptoms suggestive of COPD (cough and phlegm) was obtained for construction workers ($n = 18$) compared to a reference group of professional, administrative and clerical workers ($n = 1,022$). None of the construction workers with cough and phlegm were classed as having mild airway obstruction. However, only 14 construction workers completed lung function testing so the authors felt that little emphasis could be placed on this finding. Overall, this is a relatively small-scale study, which is limited by the fact that the age group of the study subjects (20-44) is too young for investigation of COPD, and the group size of construction workers was too small ($n=18$) to provide meaningful results. The results weakly suggest an increase in the reporting of respiratory symptoms in construction workers compared to office workers.

A population survey was conducted in 1971-72 in Tucson, Arizona on 1,132 male residents who completed a questionnaire on respiratory symptoms and occupation and underwent lung function tests (Lebowitz, 1977). Selected subjects must have been employed full time for 6 months or more. Subjects were classified as having abnormal lung function on the basis of an FEV_1/FVC ratio less than 80% of the predicted value and having an FEV_1 less than 75% of predicted. It was found that the prevalence of abnormal lung function results in construction workers was 11.9% ($n=140$) compared to 6.5% in a non-dust exposed reference group ($n=518$) after adjusting for age and smoking. The prevalence of abnormal lung function results in construction workers was said to be above the upper confidence limit for the non-exposed group, but otherwise there was no clear reporting of the statistical significance of this difference. The distribution of respiratory symptoms showed no clear differences between the construction workers and the non-exposed group. Occupational exposures to sawdust and to silica were associated with higher prevalences of abnormal lung function. The criteria for abnormal lung function used in this study do not quite match the currently agreed international criteria for COPD (FEV_1/FVC ratio less than 70% of predicted in combination with an FEV_1 less than 80% of its predicted value) but are reasonably close. These results suggest that there was a nearly a 2-fold higher prevalence of COPD in construction workers compared to a non-dust exposed group during the 1970s in this study population.

Studies reporting on respiratory symptoms

Vermeulen et al (2002) conducted a nested case-control study on a random sample of the population in the town of Doetinchem, in the Netherlands in 1993. A respiratory

questionnaire was completed by 1,104 subjects (representing an 80% response rate) aged 20-59. The results were used to identify occupations (analysed as the longest held jobs) at increased risk of chronic bronchitis or asthma. Cases of chronic bronchitis were identified by self-reported episodes of cough and phlegm daily for more than three months a year or by self-reported attacks of shortness of breath while walking on flat terrain at normal speed. A total of 274 cases of chronic bronchitis or asthma were selected and compared with 274 age-matched controls randomly sampled from those respondents reporting no history of respiratory symptoms. An odds ratio adjusted for age, sex, smoking and socio-economic status of 3.38 (95% CI 1.02-11.27) for chronic bronchitis was obtained in construction workers compared to a reference group of workers employed in “occupations with few chemical exposures” (e.g. business, law, communications, sales, etc.). Duration of employment in the construction industry was also positively associated with chronic bronchitis. For comparison, the results of this study showed that the industry with the highest OR for symptoms of chronic bronchitis was rubber, plastics and synthetics (OR = 6.52; 95% CI 1.26 – 53.8).

Data obtained by postal questionnaire from a community-based study conducted in Scotland in 1994 on 16,900 subjects were used to identify occupations at increased risk of chronic bronchitis or asthma by applying a case-control design (IOM, 1997). Cases of chronic bronchitis were identified by self-reported episodes of cough and phlegm on most days for at least three months each year. A total of 408 cases of chronic bronchitis were selected and compared with 424 controls randomly sampled from those respondents reporting no history of respiratory symptoms. In construction workers who had ever been employed in this occupation, the odds ratio for chronic bronchitis, after adjustment for smoking status and age, was 1.96 (95% CI 1.07-3.61).

Data obtained from a population survey conducted in 14 industrialised countries on 13,253 subjects (aged 20 to 44 years) were used to identify occupations at increased risk of chronic bronchitis (Zock et al., 2001). Subjects completed a detailed respiratory questionnaire and underwent lung function testing. The countries in this survey were included in the European Community Respiratory Health Survey (ECRHS). Chronic bronchitis was identified by self-reported regular expectoration of phlegm on most days for at least three months each year. A second definition was a regular cough with phlegm production for at least 3 months of the year. Subjects were classed according to current occupation, or for subjects reporting a change of job because of respiratory symptoms, by occupation at that time. To calculate prevalence ratios, office workers were used as the comparison group. In never-smoking construction workers (group size not stated) the prevalence ratio for phlegm, adjusted for gender, age and country was increased at 2.2 (95% CI 0.8-61) compared to never-smoking office workers; the corresponding prevalence ratio for cough with phlegm was 1.8 (95% CI 0.2 – 14). Lung function testing did not show any statistically significant reductions in FEV1 in any occupational group, although FEV1 values tended to be lower in most job categories compared to office workers. This study also found that self-reported occupational exposure to vapours, gas, dust or fumes significantly increased the risk of chronic bronchitis in never-smoking workers compared to never-smoking subjects non-exposed subjects (age-gender-country-adjusted OR of 1.4; 95% CI 1.0-2.0). The results of this study do not provide evidence for an increased prevalence of COPD in construction workers but the age group

studied (20-44) is probably too young for a worthwhile investigation of chronic bronchitis and COPD.

Heederick et al (1989) conducted a population survey to assess the effect of occupational exposure on the occurrence of chronic non-specific lung disease (CNSLD) in 1985 on 828 men (aged 65-84) from the industrial town of Zutphen in the Netherlands. Subjects were administered a respiratory questionnaire and were examined by a trained physician. This population of 828 men included the 555 subjects still alive of a cohort of 804 men selected in 1960 for a longitudinal study (see below Heederick et al., 1990) and 273 newly recruited residents. Cases of CNSLD were identified by either reports of episodes of cough and phlegm for longer than three months a year or episodes of wheezing and shortness of breath or by diagnosis of chronic bronchitis or emphysema by a clinician. Subjects ever treated for asthma were excluded from the analysis. By using a job exposure matrix, specific exposures for a selected occupation in a particular sector of industry were generated. This was only done for the longest held occupation. Comparable exposures were then grouped into categories. These included exposure to fumes, organic dust, mineral dust, heavy metals, organic solvents, adhesives and paint. Each of these exposures was also graded as low or high.

Working in the construction industry was significantly correlated, after adjustment for age, smoking and socio-economic status, to the occurrence of symptoms of cough and sputum compared to non-industrial work (OR=2.6; $p<0.05$). Although not statistically significant, elevated ORs were also obtained for symptoms of shortness of breath (2.20), CNSLD diagnosis (2.26) and 'ever treated for emphysema or chronic bronchitis' (1.99). Within the construction industry there was also a significant relationship between symptoms of cough and sputum and the numbers of years in this sector of industry. In general, an increased risk of symptoms of cough and sputum, CNSLD diagnosis or being treated for emphysema or chronic bronchitis was observed, after adjustment for age and smoking habits, for exposure to organic dust (presumably wood dust), mineral dust, organic solvents, adhesives or paint compared to no exposure. For example, statistically significant ORs, adjusted for age and smoking habits, of 2.36, 2.46 and 2.66 were obtained for symptoms of cough and sputum, CNSLD diagnosis or being treated for emphysema or chronic bronchitis, respectively in workers exposed to organic dust compared to non-exposed workers. A significant relationship was also seen, after adjustment for age, smoking and socio-economic status, between exposure to dust, fumes and/or gases and symptoms of cough and sputum (OR=3.13; $p<0.05$), CNSLD diagnosis (OR=2.41; $p<0.05$) or being treated for emphysema or chronic bronchitis (OR=1.75; $p<0.05$).

Overall, these results suggest that construction workers had nearly three times the probability of having symptoms of CNSLD compared to workers from non-industrial occupations. This study also shows that occupational exposure to organic dust (most likely to mean wood dust in the construction industry) in particular, and more generally to dust, fumes and/or gases, is an important factor in the development of CNSLD.

The same population was also studied in a longitudinal investigation aimed at obtaining more stable and reliable estimates of the occurrence of CNSLD in relation to occupation (Heederick et al., 1990). A cohort of 804 men (aged 40-59), which largely overlapped with that of the cross-sectional analysis described above, was

selected in 1960 and followed for 25 years until 1985. Information about occupation was obtained by questionnaire and the occurrence of CNSLD was monitored through regular medical examinations. By dividing the number of people with CNSLD in a specific occupational group by the total number of person-years in that group, the incidence density (ID) for CNSLD for each occupational group was calculated. A person contributed person-years of observations until CNSLD was diagnosed or until death occurred. Incidence density ratios (IDRs) for each occupation were then calculated by comparison with the ID for CNSLD of a reference group of white-collar workers. Subjects ever treated for asthma were excluded from the analysis only towards the end of the follow-up, which implies that the risk of CNSLD might have been slightly overestimated by including some asthmatics. Occupation was analysed as the job held in 1960. Of the cohort members who were still alive in 1985 less than 17% had ever worked in another type of industry. In construction workers (n = 60) an increased risk of CNSLD, adjusted for current age, calendar period and smoking status, (IDR=2.3; 95% CI 1.44-3.67) was found in comparison with white-collar workers (n = 369). This study benefits from the longitudinal design and the fact that it focused on a more relevant age group (40-59) for investigation of chronic respiratory symptoms compared to some of the cross-sectional surveys reported above.

A 13-year follow-up study of occupational exposure and incidence of chronic respiratory symptoms was carried out among 2730 residents of Cracow, Poland (Krzyzanowski and Jedrychowski, 1990). Subjects were interviewed three times, in 1968 (start of the study), 1973 and 1981 (end of follow-up). The results showed that prolonged exposure to dusts was associated with a doubling of the risks of developing chronic phlegm and attacks of breathlessness (odds ratios adjusted for age, smoking and socio-economic status of 2.1 [95% CI 1.5 –3.0] and 2.4 [95% CI 1.5 – 3.7] respectively). In males exposed to dusts, the observed incidence of attacks of breathlessness and chronic cough was greater than expected (p=0.05) among construction workers. No additional information specifically related to construction workers was available in this study report.

Studies reporting on physician-diagnosed COPD

Data obtained by interviews in 1992 from the Health and Retirement survey, a nationally representative cohort of older people in the United States, were used to examine health outcomes of construction workers (age 51-61 years) compared to workers in the same age range from other blue-collar occupations (Petersen and Zwerling, 1998). A group of 312 male construction workers was identified in this survey. Two comparison reference groups were selected: a group of 1,716 male blue-collar workers (service employees, farming, fishing or forestry, mechanics or repair, precision production, operators and armed forces) and a group of 2,064 male white-collar workers (managerial, professional specialty, technical, sales and administrative support). Cases of COPD were identified among those respondents who reported a physician's diagnosis of chronic bronchitis or emphysema.

The three groups were similar with respect to age and race; however, the proportion of fully retired construction workers was almost double (15.5%) that of the other two groups (7.2% and 8.2% in the blue-collar and white-collar workers respectively). Construction workers exhibited a higher prevalence of COPD (9.6%) compared to the other blue-collar workers (7.3%) and to the white-collar workers (5.1%). Although smoking was not taken into account in this comparison, it is reasonable to assume that the other blue-

collars and the construction workers would have had similar smoking habits and thus to infer that the excess prevalence of COPD observed in the construction workers was likely to be due to occupational factors. Furthermore, a separate analysis for never-smokers showed that the prevalence of COPD among never-smoking construction workers was a lot higher (10.4%) than in never-smoking blue-collars (3.7%) with an age- and working status-adjusted OR of 3.2 (95% CI 1.04-10.05). Surprisingly, the group of construction workers who had the highest prevalence of COPD were those who never smoked (10.4%), followed by those who formerly smoked (9.9%) and current smokers (8.6%). This seems to suggest that relatively old construction workers who never smoked were more likely to have COPD than their counterparts who currently or formerly smoked. The most likely explanation of this paradox is that construction workers that smoke self-select out of the trade. Construction workers who smoke may begin to encounter symptoms of COPD at an earlier stage of their work life than non-smokers, becoming unable to keep up with the physical demand of the job and hence, selecting themselves out of the trade in another occupation. Workplace dust exposures may have contributed to the onset of the symptoms in such smoking workers. This suggests that the results of this cross-sectional analysis may be an underestimation of the true risk of developing work-related COPD in those who smoke. It is also noted that about 60% of the construction workers said the COPD symptoms limited their normal activities compared to only 19% of the other blue-collars, implying that not only was there a higher prevalence of COPD in the construction workers, but that the condition was of a greater severity.

Overall, this study shows that in a survey conducted in the United States in 1992, never-smoking construction workers were over three times more likely to have a physician's diagnosis of COPD than other never-smoking blue-collar employees, and that the COPD was frequently of higher severity.

Information from published statistics in the UK

There are various sources of information on the incidence of occupational respiratory disease in the UK and these are published on HSE's website. One source is the Department of Work Pensions (DWP) Industrial Injuries Scheme (IIS). This records the numbers of workers receiving compensation for prescribed industrial diseases such as pneumoconiosis. However there are no compensated cases of pneumoconiosis recorded for construction workers under this scheme. The only occupational group for whom compensation is available for COPD is underground coalminers; hence this scheme is unable to provide statistics on the incidence of work-related COPD in UK construction workers.

Another potentially relevant source of information is The Health and Occupational Reporting network (THOR). This network of reporting schemes is run by Manchester University and includes SWORD (Surveillance of Work-related and Occupational Respiratory Disease) and OPRA (Occupational Physicians Reporting Activity). The statistics from this scheme are likely to grossly underestimate of the true incidence of work-related COPD. There are a number of reasons for this, but the main reason is that most cases of COPD occur in smokers, and the primary care doctor may not suspect an occupational causation or contribution. Hence very few cases will be referred to the specialist physicians participating in THOR. The annual incidence of work-related COPD reported under THOR between 2000-2004 ranged between 30-144. The most important occupations contributing to these cases were coalmining,

dockworking, welding, and petroleum working. There is no specific information relating to the incidence of work-related COPD in construction workers.

In relation to pneumoconiosis (including asbestosis), SWORD data reveals that, with the exception of miners and quarrymen, the annual incidence per 100,000 employed people during the years 1996-1999 was higher (8.1) in construction workers than in other occupational groups (e.g. 0.7 in transport and communication workers; 1.3 in engineers) (Meyer et al., 2001). This provides only limited information because the pneumoconiosis category includes asbestosis. Hence, it is uncertain whether or not any of the cases involved would have been silicosis or mixed dust pneumoconiosis. Hence, for the purposes of this review, no useful conclusions can be drawn from the SWORD data.

At the time of writing this document, the most recently published HSE Occupational Health Decennial Supplement was that for 1995. This includes a section covering the results from the Labour Force Survey (LFS) for 1990. This was a survey on a representative sample of 40,000 households in England and Wales. A questionnaire was used to identify self-reported illnesses that respondents believed to have been caused or made worse by work. The results allowed occupational groups with relative risks >2 for the main disease categories to be identified. This showed that male construction workers were in the top five occupational groups for increased reporting of work-related lower respiratory disease, with a rate of 105 cases per 10,000 and a relative risk of 3.34 (statistically significant at a 1% level). Lower respiratory disease referred to “lung and breathing problems”. This disease category did not include asthma, pneumoconiosis or upper respiratory disease, which were each listed separately. Only coal mining and “other” (mainly labourers) showed higher rates for lower respiratory disease than construction workers. It is difficult to interpret these data from the LFS because they are based on self-reporting and perceptions of the effect of working conditions. It was reported that 85% of respondents had consulted a doctor about their illness, but on the other hand, nearly half the cases took no sickness absence as a result of their illness, implying that the cases were not severe. The interpretation of these results is further limited by the lack of detail on the specific nature of the respiratory ill health conditions. Overall this is rather weak evidence, but it does fit with the findings from other studies in this section pointing to a higher prevalence of chronic bronchitis and COPD in construction workers than in many other occupational groups.

There is no other relevant information available from UK sources of statistics on the prevalence of non-malignant respiratory ill health conditions in construction workers.

Summary of the general population studies

This review has identified 10 general population studies that have investigated the occupational distribution of COPD and chronic bronchitis (see Table 1). Only one of these studies was conducted in the UK (Scotland). The findings from these studies are broadly consistent in showing that construction workers tend to have an increased prevalence of COPD and of symptoms of chronic bronchitis compared to most of the other occupational groups. In general, the prevalences of COPD and symptoms of chronic bronchitis ranged between 2- to 3-fold higher in construction workers compared to age and smoking matched controls from other occupations, although this varies from study to study. In the majority of studies the reference group was composed of office workers or other administrative staff. However, in one study the

reference group comprised other “blue-collar” workers, but still there was a 3-fold increase in the prevalence of COPD in the construction workers; also, in this study the severity of COPD appeared to be worse in construction workers than in the other blue-collar workers.

In some studies, it was observed that higher odds ratios were obtained for the association between construction work and COPD/chronic bronchitis when analyses were conducted in never smokers, compared to analyses on all subjects combined or for smokers/ex-smokers. This is probably because smoking can obscure the ability to detect an occupational contribution to COPD development, and the impact of occupational dust exposures is more apparent when comparisons are made on never-smokers.

These general population studies do not provide any information on particular trades most at risk of COPD within the construction industry. Most studies did not explore potential substances/processes associated with increased risks of COPD. The few studies that did investigate causative agents did so only at a very general level, and found that, dusts, including wood dust and silica, were noted as being associated with COPD and the symptoms of chronic bronchitis.

It is noted that there is only one large-scale population study on the work-relatedness of COPD conducted in Great Britain. However, among the remaining studies, all in industrialised countries, most were conducted in the 1990s, and hence are relatively recent. On balance, given that the type of construction materials and processes undertaken in construction work are likely to be broadly similar across different industrialised countries, it seems reasonable to conclude that the evidence for an association between COPD and symptoms of chronic bronchitis in construction workers is likely to be relevant to current working conditions in Great Britain.

Mortality studies

COPD is an umbrella term that can cover a number of disease states including chronic bronchitis, emphysema, asthma, bronchiectasis, extrinsic allergic alveolitis and chronic airways obstruction not otherwise classified. This means that mortality data on COPD can refer to a number of different codes in the International Classification of Diseases (ICD) system. The European Lung White Book notes that the term COPD can be used in a variety of ways when it is judged to be a contributing factor but not the main cause of death, leading to misclassification and omissions from medical records. For example, COPD predisposes to lung infections such as pneumonia, which would be given as the cause of death. Hence, death certificate information may lead to underestimates of the true extent of mortality from COPD.

For ease of digesting the information in the following summaries of mortality studies, the various ICD codes referring to deaths from COPD have been omitted. All of the mortality studies considered in this review are also summarised in tabular form in Table 2 in the Appendix to this document.

Mortality studies in construction workers

A prospective cohort study of 317,629 Swedish male construction workers followed from 1971 to 1999 investigated whether occupational exposure to dust, fumes or

gases increased the risk for death from COPD (Bergdahl et al., 2004). In 1968, the Swedish Foundation for Occupational Safety and Health was established to coordinate activities concerning occupational health in Swedish construction workers. Construction workers were invited to health examinations at intervals of 2-5 years. Although the programme was voluntary, at least 80% of the eligible workers participated at least once. Data from the health examinations included occupational titles and smoking habits. Those examined before age 15 or after age 67 were excluded from analysis. The loss of subjects to follow-up was low (0.15%) and such persons were also excluded from the analysis. The occupational title at the time of the first health examination was used in the analysis; however, most workers (up to approx 74%) remained in the same occupation during the follow-up period.

The exposure assessment was based on information obtained on each job from a previous survey conducted in the mid-1970s by occupational hygienists and physicians visiting five different sites in different geographical regions of Sweden. Based on this information, a job-exposure matrix was developed. This allowed estimation of each individual's exposure. Exposures to asbestos, asphalt fumes, cement dust, concrete dust, diesel exhaust, epoxy resins, isocyanates, man-made mineral fibres, metal fumes, organic solvents, quartz dust and wood dust were assessed. However, in the final analysis, categories were combined, resulting in four major exposure groups: inorganic dust (asbestos, man-made mineral fibres, cement, concrete and quartz), gases and irritants (epoxy resins, isocyanates and organic solvents), fumes (diesel exhaust, metal fume and asphalt) and wood dust. Of the 317,629 workers included in the cohort, 200,735 reported exposure to any airborne exposure (with 154,324 to inorganic dust, 52,434 to gases and irritants, 69,657 to fumes and 21,479 to wood dust) and 116,894 constituted the non-exposed reference group. Through a linkage to the National Cause Death Register, subjects who died (underlying cause) from COPD were identified. Altogether 523 deaths due to COPD occurred among workers exposed to any airborne exposure and there were 200 deaths among the non-exposed workers.

After adjusting for smoking and age, there was a slightly increased RR of death due to COPD (RR 1.12 – 95% CI 1.03-1.22) among workers exposed to any airborne exposure as compared to the non-exposed workers. The RR of death from COPD was also increased among those exposed to inorganic dust (RR 1.16 – 95% CI 1.05-1.28), those exposed to gases and irritants (RR 1.18 – 95% CI 0.98-1.41) and those exposed to fumes (RR 1.22 – 95% CI 1.04-1.42) as compared to the non-exposed workers. When restricting the analysis to never-smokers, mortality from COPD further increased among workers with any airborne exposure (RR 2.11- 95% CI 1.43-3.00), those exposed to inorganic dust (RR 2.38 – 95% CI 1.59-3.44), those exposed to gases and irritants (RR 2.92 – 95% CI 1.46-5.22) and those exposed to fumes (RR 2.72 – 95% CI 1.31-3.68) as compared to the non-exposed never-smoking workers. In a different type of analysis, using a Poisson regression model including smoking, age and the four major exposure categories, only exposure to inorganic dust was associated with an increased risk of mortality from COPD (hazard ratio HR 1.10 – 95% CI 1.06-1.14), especially among never-smokers (HR 2.30 – 95% CI 1.07-4.96).

The fraction of COPD among the exposed attributable to any airborne exposure was estimated as 10.7% overall and 52.6% among never-smokers. This implies that 56 of the 523 deaths from COPD would not have occurred but for workplace exposures to dust, gases and fumes in this cohort of construction workers.

Overall, this is a detailed study of a very large Swedish cohort with nearly 30 years of follow-up. The results indicate that mortality from COPD among construction workers is linked to exposure to dusts and irritants. Although the choice of an internal reference group acted as a control for differences in socio-economic status, an important risk factor for COPD, it is likely that construction workers in the “unexposed” group would also have had some exposure to dust, fumes or gases, (albeit at lower levels than in the exposed group), leading to a possible underestimate of the effects of dusts/irritants exposure. Also, the study did not analyse risk in relation to cumulative exposure, which may have refined the evidence on the effects of exposure. It should be pointed out that this study did not compare mortality from COPD in construction workers with that from the general population.

Robinson et al (1995) conducted a study of mortality in the construction industry in the United States (US) between 1984 and 1986 based on an analysis of 876,731 death certificates. Proportionate mortality ratios (PMRs) for specific causes of death were calculated using the US age-, gender- and race-specific proportional mortality rates for the years of the study. Statistically significantly increased PMRs were obtained for COPD of 122 (95% CI 113-131, based on 733 deaths), and for silicosis PMR=327 (95% CI 149-620, based on 9 deaths) among white male construction workers who died under the age of 65 (n = 5,357). Analysis among various trades showed an elevated PMR for silicosis of 449 in painters (based on 4 deaths). It is noteworthy that these deaths were in men under the age of 65. Smoking status was not taken into account in this study and this makes it difficult to interpret the increased PMR for COPD. However, smoking is not a cause of silicosis, and this can be more reliably attributed to occupation. There was a concern raised by the authors about silicosis and the use of silica for abrasive blasting in construction work.

PMRs for specific causes of death were evaluated among 29,554 male construction workers who resided and died in North Carolina, US during the period 1988-1994 using the North Carolina age-, gender- and race-specific proportional mortality rates for the years of the study (Wang et al., 1999). A statistically significant excess of death was observed for pneumoconiosis and other non-malignant respiratory diseases (PMR=111, $p < 0.05$). Analysis among various trades showed statistically significant elevated PMRs ($p < 0.05$) for non-malignant respiratory diseases including COPD specifically in carpenters (PMR=114), labourers (PMR=118), painters and plasterers (PMR=152) and insulators (PMR=218). In addition, there was a statistically significant ($p < 0.05$) excess mortality from emphysema in operating engineers and heavy equipment operators (PMR=191). The high PMR in insulators probably reflects an excess of asbestosis.

In a registry based study, statistics obtained from the silicosis mortality surveillance scheme in the US between 1968 and 1990 (total number of deaths with silicosis 13,744) show that the construction industry accounted for the highest proportion (over 10%) of silicosis deaths (Bang et al., 1995).

Only one published study of mortality in UK construction workers was identified (Dong et al 1995). This study was based on analysis of 15007 death certificates of members of the Building and Civil Engineering Holiday and Benefit Scheme who had died between 1975-1987 aged 20-64 years. The key findings of the study and focus for analysis were deaths from cancer and accidental deaths. However, there were 422

observed deaths from non-malignant diseases of the respiratory system versus 937 expected, based on male deaths occurring in England and Wales over the same period over the same age range, leading to a PMR of only 45. This might appear to suggest a substantial deficit in deaths from non-malignant respiratory diseases in UK construction workers. However, it should be noted that the mortality data derived from those who died in service or shortly afterwards; the mean age at death was 53 years and 92% of subjects were still in employment at the time of death. Given the nature of COPD, it is most unlikely that those with severe COPD would still be in employment at or close to the time of death, and hence this study is unable to contribute meaningful evidence on the relationship between construction work and COPD. Although there were 41 deaths from mesothelioma in this study, there was no mention of deaths from asbestosis or other pneumoconioses such as silicosis, which presumably would have been rolled in with the general category of deaths from respiratory diseases. In view of the way the data were obtained in this study, this study will not be included in Table 2 or in the final summary analysis of the mortality studies.

Mortality studies in specified construction trades

Heavy equipment operators

The only studies in heavy equipment operators were conducted in the US. In the US, operating engineers operate and maintain heavy earthmoving equipment such as cranes, bulldozers, graders and backhoes. This equipment is used in four main activities; building roads, bridges, tunnels and dams; construction of buildings and power plants; removal of earth materials and grading earth surfaces, and in the replacement of concrete and other paving materials; construction of drainage systems, pipelines and other related activities such as blasting.

A cohort mortality study was conducted in 3,243 construction heavy equipment male operators who had been members of the US International Union of Operating Engineers during 1964 and 1978 and had died as of 31 December 1978 (Wong et al., 1985). SMRs for specific causes of death were estimated using the US age- and gender-specific mortality rates for the years of the study. Overall mortality for the entire cohort and several subgroups was significantly lower than expected. However, a statistically significant increase in mortality was observed for emphysema (SMR=165, 95% CI 136-198, based on 116 deaths). When mortality was analysed in relation to duration of union membership, for durations of <5, 5-9, 10-14, 15-19 and ≥ 20 years, the corresponding SMRs for emphysema were 99, 107, 158, 194, and 175 (the latter two SMRs being statistically significant). Duration of union membership was taken as a surrogate for exposure to diesel engine exhaust emissions. The study did not take smoking status into account, but it seems unlikely that the observed mortality from emphysema could be entirely explained by cigarette smoking. There was also an analysis in this study of mortality in retirees (n=6678, of which there were 1345 deaths). Among the deaths, there was a statistically significant excess of non-malignant respiratory disease; the excess came primarily from emphysema (75 deaths versus 27 expected, SMR = 277.3 p <0.01). A further analysis of retirees was undertaken, to exclude those who had retired early (before age 65) due to ill health. This comprised a group of 4075 subjects among which there were 796 deaths. In this group, there were 59 deaths from emphysema versus 21 expected, leading to an SMR

of 274.8 (95% CI 209-354.6). Among the cancer deaths in this cohort, only liver cancer was found in excess, with lung cancer mortality almost identical with expected rates. This suggests that cigarette smoking is unlikely to entirely account for the excess mortality from emphysema observed in this cohort. This was not a very large-scale study but the results are reasonably convincing for an excess mortality from emphysema in construction heavy equipment operators. The relevance of this evidence to current employment conditions in the UK construction industry is uncertain.

In another mortality study of 15,843 construction heavy equipment operators who had been members of the US International Union of Operating Engineers and had died between 1988 and 1993, PMRs for specific causes of death were calculated using the US age-, gender- and race-specific proportional mortality rates for the years of the study (Stern et al., 1997a). Deaths from emphysema (PMR=137, 95% CI 120-155) and from pneumoconioses including silicosis and other respiratory diseases including COPD (PMR=111 95% CI 104-119) were found in excess. There were 43 deaths from bronchitis (PMR = 128; 95% CI 93-173). There were 3 deaths from silicosis (PMR =149; 95% CI 0.3-4.34), although a PMR is probably meaningless for such a low number of deaths. The study did not take smoking status into account making it difficult to judge the likely role of occupation in contributing to the observed excess mortality from emphysema. Lung cancer mortality was raised with a PMR of 114 (95% CI 109-119); this does not particularly help to clarify the likely role of smoking in relation to the excess mortality from emphysema.

Asphalt workers

Asphalt consists of bitumen mixed with sand and/or gravel (Burstyn et al 2003). Although bitumen can contain coal tar, the use of coal tar in the asphalt industry has been largely discontinued in Western Europe. Fumes are emitted when hot asphalt is applied to surfaces during paving. The fumes consist of particles containing adsorbed polycyclic aromatic hydrocarbons (PAHs).

Burstyn et al., (2003) reported a cohort mortality study of asphalt workers drawn from studies in Denmark, Finland, France, Germany, Israel, the Netherlands and Norway. The overall cohort study was coordinated by the International Agency for Research on Cancer (IARC) to investigate cancer mortality, although data on deaths from non-malignant causes were also collated. The study by Burstyn et al., was to specifically examine the relationships between mortality from obstructive lung diseases and occupational exposures among asphalt workers. The reason for the study was that the authors were aware of various studies pointing to an association between respiratory disease and asphalt work, although the authors felt that these studies were not definitive.

For example, note was made of a cohort study of 679 Danish male mastic asphalt workers followed from 1959 to 1986 (Hansen, 1991). Deaths from bronchitis, emphysema and asthma grouped were found to occur in excess (SMR=207; 95% CI 95-393), based on 9 deaths. However, this excess was not statistically significant and this was a relatively small-scale study. Attention was also drawn to a study in highway maintenance workers in California (Maizlish et al 1988). There were 1570 deaths in workers employed between 1970 and 1983; among these deaths there were 8

deaths from emphysema versus 3.2 expected, leading to a PMR of 250. However, this excess could not be related to any specific exposure, whether to asphalt, herbicides, diesel engine exhaust emissions etc. A number of cross-sectional studies were identified that linked exposure to asphalt fumes to symptoms of chronic bronchitis but these studies had not controlled for smoking and tended to have low power. Given this body of uncertain evidence relating asphalt work to obstructive lung disease, Burstyn et al analysed the evidence relating to mortality from obstructive lung disease drawn from the large European cohort assembled by IARC.

The study included 58,862 men first employed between 1913 and 1999 in 217 companies applying and mixing asphalt in seven countries. Oil refineries where bitumen is distilled from crude oil were excluded from the study. Within the study population, there were 12,367 persons who were employed only in asphalt paving and for whom quantitative exposure estimates were available for PAHs and bitumen fume. There were 36,831 persons never exposed to coal tar. A study specific exposure matrix (Road Construction Worker's Exposure Matrix, ROCEM) was produced taking into account personnel records and job classes for the subjects in the study.

Within this study loss to follow-up was 0.9%. Follow-up began in 1953 and ceased in 2000. The average duration of follow-up was 17 years. Because of the poor ability of death certification in Europe to discriminate between deaths from asthma (ICD-9 code 493) and COPD (chronic bronchitis and/or emphysema, ICD-9 codes 490-492) no distinction was made between deaths from asthma and COPD.

Exposure-response analyses of mortality from chronic bronchitis, emphysema and asthma were conducted in relation to three estimates of exposure to PAHs; duration of exposure, cumulative exposure and average exposure, with a further separate analysis for the pavers only, using Poisson regression. For all workers, positive associations were observed in relation to estimates of cumulative and average exposure, but not with duration of exposure. The associations were stronger for the pavers. To illustrate this, quantitative estimates of cumulative exposure were expressed as benzo[α]pyrene ($\text{ng}\cdot\text{m}^{-3}$) multiplied by years; for cumulative exposures of >0 - <233 , 233 - <624 , 624 - <1414 , >1414 , the corresponding RRs and 95% CIs were 1.00, 1.55 (0.68-3.54), 2.97 (1.25,7.07), and 4.06 (1.35,12.19). These values showed a statistically significant trend of increased risk with increased exposure ($p<0.001$).

A separate analysis for coal tar was said to have yielded similar results. A further separate analysis for bitumen fume revealed positive but not statistically significant associations with mortality from obstructive lung diseases. However, it was felt that this association could have been confounded by simultaneous exposure to coal tar. The authors noted that there were not enough deaths in the various exposure categories in the subcohort lacking exposure to coal tar to allow any meaningful analysis of bitumen fume exposure unconfounded by coal tar. Cumulative and average exposures to silica, diesel exhaust and asbestos were not positively associated with mortality from obstructive lung disease after adjustment for country, age, calendar period, and duration of employment. The authors considered that smoking was the principal uncontrolled confounding factor in the study.

Overall, the authors concluded that exposure to PAHs in asphalt workers was associated with an increased mortality from obstructive lung disease; that the main source of the PAH exposure in these workers was from coal tar, although some exposure via bitumen fume also occurs. The authors felt that they could not rule out a role for bitumen fume exposure in contributing to the mortality from obstructive lung

disease. Overall, the findings suggest that contemporary asphalt work, which should not involve the use of coal tar, may be of only limited concern for an increased risk of mortality from obstructive lung disease.

The analysis presented above by Burstyn et al., was aimed at characterising the causative agents associated with mortality from obstructive lung disease in asphalt workers from seven countries. However, the publication does not give an indication of the extent of mortality from obstructive lung disease in comparison with the relevant general populations. In the time available for completing this review it has not been possible to obtain and assess the individual cohort studies involved. However, for the study in Norway (obtained as an abstract only) reported by Randem et al (2003), it is noted that the SMR for mortality from non-malignant respiratory disease was 125 (95% CI 0.97-158) and was associated with time since first employment in the asphalt industry. Mortality from respiratory diseases was highest among those first employed in the 1960s. Among the different job types, mortality from respiratory diseases was highest among the pavers and mastic asphalt workers. This is in line with the findings noted above by Hansen (1991) for Danish mastic asphalt workers.

Roofers and waterproofers

In a mortality study of 11,144 members of the US Union of roofers, waterproofers and allied workers who died between 1950 and 1996, PMRs for specific causes of death were estimated using the US age-, gender- and race-specific proportional mortality rates for the years of the study (Stern et al., 2000). Roofers may be exposed to bitumens (asphalt and/or coal tar pitch), as well as asbestos and fibreglass. A statistically significant elevated risk of mortality was observed for pneumoconioses, including silicosis and asbestosis, and other non-malignant respiratory diseases including COPD (n=326; PMR=115; 95% CI=103-128). There were also non-statistically significant increases in mortality from bronchitis (PMR = 123; 95% CI 85-172), and emphysema (PMR=119; 95% CI 99-139).

Road construction workers

In a study of 863 Italian male road construction and maintenance workers receiving work disability compensations who died between 1980 and 1993, PMRs for specific causes of death were estimated using the Italian age- and male-specific proportional mortality rates for the years of the study (D'Errico et al., 2002). A statistically significantly increased PMR of 307 (95% CI=166-567; n=10) was obtained for silicosis.

Labourers

In a mortality study of 11,685 construction labourers, members of the Labourers' International Union of North America who died between 1985 and 1988, PMRs for specific causes of death were estimated using the US age- and gender-specific proportional mortality rates for the years of the study (Stern et al., 1995). A PMR of 112 (95% CI=92-136) was obtained for emphysema. As smoking status was not taken into account in this analysis, and given the magnitude of the PMR, these results do not provide evidence for an occupational link to the excess of emphysema deaths.

Burkhart et al., (1993) undertook a literature review to characterise the exposures and health risks of construction labourers. Their paper cites data from the NIOSH Occupation Coded Mortality Surveillance Data 1984-1986. This source presented a PMR for COPD of 133 ($p \leq 0.05$) in white men, and of 122 ($p < 0.05$) in black men. The only other relevant health risk information presented derived from the California Occupational Mortality Study (COMS) 1979-1981. This provided SMRs for specified causes of death in construction labourers, adjusted for smoking and alcohol consumption. However, the authors pointed out that the data from COMS only covered deaths up to age 64, and concern was expressed about the accuracy of classification as construction labourers, which they felt may have included other workers such as tile setters, carpet and dry wall installers etc. However, bearing these limitations in mind, the smoking and alcohol-adjusted SMR for COPD in white male construction labourers was 120 (95% CI 89-158), and in black male construction labourers was 148 (95% CI 74-265).

Overall, the limited information available does not provide evidence for an association between work as a construction labourer and mortality from COPD.

Plasterers

PMRs for specific causes of death were calculated among 12,873 members of the US Operative Plasterers' and Cement Masons' International Association who died between 1972 and 1996 using the US age-, race- and calendar-specific proportional mortality rates (Stern et al., 2001). There were 159 deaths from emphysema versus 148 expected, with a non-statistically significant PMR of 107. A statistically significant excess mortality from non-malignant respiratory diseases including COPD was observed (PMR=109; $p < 0.05$). There were 4 deaths from silicosis versus 2 expected, leading to a PMR of 179 (not statistically significant). This study does not provide evidence for a link between occupation as a plasterer or cement mason with mortality from COPD. However, the four silicosis deaths in plasterers and cement masons indicate potential for exposure to respirable crystalline silica in these construction trades, but they were only a very small fraction of the total mortality (>12000 deaths).

Construction carpenters (joiners)

In a mortality study of 27,362 members of the US Carpenters' Union who died between 1987 and 1990, PMRs for specific causes of death were estimated using the US age-, gender- and race-specific proportional mortality rates for the years of the study (Robinson et al., 1996). A statistically significantly increased PMR of 115 (95% CI=102-130) was obtained for emphysema among white male carpenters who were last employed while in the construction industry. As smoking status was not taken into account in this analysis, and given the magnitude of the PMR, these results do not provide clear evidence of an occupational link to an increased risk of emphysema deaths. There were 4 deaths from silicosis with a PMR of 112 (95% CI 31-288). It is interesting to note that there were 121 deaths from mesothelioma in this study population, and 17 deaths from asbestosis, indicating exposure to asbestos-containing dust.

Ironworkers

In a mortality study of 13,301 members of the North America International Union of construction ironworkers who died between 1984 and 1991, PMRs for specific causes of death were estimated using the US age-, gender- and race-specific proportional mortality rates for the years of the study (Stern et al., 1997b). The Ironworkers Union members were said to be mainly bridge, structural, ornamental and reinforced concrete iron workers, welders, rodmen, machinery movers, stone derrick-men, shop men and navy yard riggers, among other skilled members. A statistically significant elevated risk of mortality was observed for pneumoconioses, including silicosis and asbestosis, and other non-malignant respiratory diseases including COPD (n=690; PMR=111; 95% CI=103-120) and for emphysema (PMR=122; 95% CI=104-143). As smoking status was not taken into account in this analysis it is difficult to interpret the raised PMR for emphysema, but overall, these findings do not provide clear evidence of a link between work as an iron-worker in the construction industry and COPD.

Electricians

The mortality of 31,068 US members of the International Brotherhood of Electrical Workers, who primarily worked in the construction industry and died between 1982 and 1987, was evaluated for a possible association between employment and death from specific causes (Robinson et al., 1999). PMRs were calculated using US age-, gender-, race- and calendar-specific proportional mortality rates. A statistically significant excess mortality from emphysema was observed (n=161; PMR=130; 95% CI 110-151) among those electrical workers classified as outdoor workers (n = 12,071). Smoking status was not taken into account in this analysis and it is uncertain whether or not smoking could have entirely accounted for the magnitude of the PMR (130) for emphysema in this study population.

Bricklayers

PMRs for specific causes of death were evaluated among 10,921 members of the US International Union of bricklayers and allied craftworkers who died during the period 1986-1991 using the US age-, gender- and race-specific proportional mortality rates for the years of the study (Salg and Alterman, 2005). An increase in mortality was observed for emphysema (PMR=133; 95% CI 111-157) and for other non-malignant respiratory diseases including COPD (PMR=119; 95% CI 110-129). Smoking was not taken into account in this study making it difficult to assess the possible role of occupational dust exposure in contributing to the excess mortality from emphysema. There were four deaths from silicosis, with a non-statistically significant PMR of 322. Within this paper, there was reference to unpublished data from NIOSH based on death certificates coded for occupation and industry for 28 US States in the National Occupational Mortality Surveillance system for the period 1984-1998. An elevated PMR for COPD in brickmasons and stonemasons of 120 (95% CI 114-126) was found. Again, without any adjustment for smoking habits, it is difficult to interpret this finding.

Finkelstein and Verma (2005) reported a cohort mortality study in 11,000 Ontario members of the International Union of Bricklayers and Allied Craftworkers. Subjects entered follow-up at the date of joining the union, or 1950, whichever was later. Follow-up was terminated at age 85; the paper did not state the year in which follow-up was terminated, and only brief information was provided concerning the design of the study. The reference population for calculating SMRs was the male population of Ontario. There were 836 deaths observed in the cohort. Among the deaths, mortality from respiratory disease was not elevated, with SMRs and 95% CIs for obstructive lung disease of 91 (37-190), 120 (62-210) and 86 (46-150) for the periods 10-19, 20-29 and ≥ 30 years since start of union membership.

Overall, the mortality data for bricklayers do not provide clear evidence for an increased mortality from COPD.

Construction painters

In a cohort mortality study of 42,170 male painters, members of the US International Brotherhood of Painters and Allied Trades, followed for 15 years from 1979 to 1994, SMRs for specific causes of death were estimated using the US age-, gender- and calendar-specific mortality rates (Steenland and Palu, 1999). During this period 18,259 deaths occurred. An elevated risk of mortality was observed for emphysema (n=232; SMR=113; 95% CI 99-128) and for other non-malignant respiratory diseases including COPD (SMR=107; 95% CI 99-114). These SMRs did not quite reach statistical significance, and as smoking status was not taken into account, it can be concluded that these results do not provide evidence for an increased mortality from COPD in painters from the construction trade.

Summary of mortality studies

There are numerous studies that inform on mortality from respiratory disease in construction workers (see Table 2). Three studies cover construction workers in general, and the remaining studies focus on particular trades within the construction industry such as heavy equipment operators or asphalt work.

There are 11 studies that reported PMRs or SMRs for deaths due to COPD or emphysema (these do not include the studies that grouped together the deaths from non-malignant respiratory diseases including pneumoconioses and COPD). Most of these studies found statistically significantly raised PMRs/SMRs with magnitudes ranging between 109-207. One study in US heavy equipment operators found that the SMR for emphysema was 275 in 'normal' retired workers (those who had not retired prematurely due to ill health). Overall, the findings suggest that in general, there is a moderate excess mortality from COPD in construction workers compared to the general population. However, this is difficult to interpret given that smoking was not taken into account in any of these studies. In this regard, it may be relevant to note that a study in over 300,000 Swedish construction workers with 30 years of follow-up estimated that the proportion of COPD mortality attributable to dust/irritant exposure was 10.7% in the overall cohort, and 52.6% in the never-smokers.

It is uncertain if the estimated attributable fractions from the Swedish study are generalisable to mortality findings from other countries. However, they provide some

indication of the likely role of exposures to dusts and irritants in contributing to mortality from COPD in construction workers.

In terms of specific trades within the construction industry that may be at most risk of mortality from COPD, it is difficult to draw any firm conclusions from the available data, mainly because generally only one study exists for any individual trade. Two separate US studies in heavy equipment operators both found an excess mortality from COPD with statistically significant SMR and PMR values of 194 and 134 respectively. In one of these studies there was a suggestion that exposure to diesel engine exhaust emissions may have been a relevant factor. Overall however, no firm conclusions can be drawn concerning particular construction trades that may be most at risk from an increased mortality from respiratory disease.

There is little information concerning the precise exposures that may be associated with mortality from COPD in construction workers. Dusts and irritants have been shown to be associated with mortality from COPD, not just in construction workers, but also for employment in general. More specifically, a large-scale European study that included a detailed job exposure matrix revealed a positive association between PAH exposure and mortality from obstructive lung disease in asphalt workers. However, the main source of PAH exposure was judged to be coal tar, the use of which is now largely discontinued in asphalt work in Europe. Although there is still some exposure to PAHs from the bitumen used in asphalt work, it is uncertain whether this would be sufficient to warrant concern for COPD development.

The mortality studies also provide evidence to show that among construction workers, deaths can occur from silicosis but this has not been widely investigated. A US surveillance scheme showed that of 13,744 deaths from silicosis between 1968-1990, 10% were in construction workers. Other studies revealed raised PMRs for silicosis in general construction workers, and in plasterers, cement masons and in road construction and maintenance workers. Although the PMR values were relatively high, they typically represent only small numbers of deaths.

In conclusion, mortality statistics are likely to suffer from a number of weaknesses particularly in relation to construction workers. This is because as they grow older, many workers are likely to leave the construction industry for less physically demanding work. Hence, the occupation stated on the death certificate may not reflect the main occupation held in life. From the studies available, there is a reasonably consistent body of evidence pointing to a low to moderate excess of mortality from COPD in construction workers compared to the general population. Cigarette smoking and socioeconomic factors are likely to account for much of this excess, but occupational exposures to dusts and irritants play a role. A single study in a very large cohort of Swedish workers, suggests that occupational exposures to dusts or irritants may be responsible for almost 11% of the mortality from COPD in construction workers, rising to about 50% in never-smokers.

Case reports

In an Alert by NIOSH (2005), six case reports of construction workers who have died or are suffering from silicosis are described. These are included here only for completeness.

A 39-year-old man was diagnosed with silicosis (progressive massive fibrosis) and tuberculosis in April 1993 after working 22 years as a sandblaster.

A white male non-smoker was diagnosed with advanced silicosis, emphysema, and asthma at age 49 after working 23 years as a tile installer. His work included polishing and drilling tile, and he was exposed to grout dust and sandblasting.

A white male non-smoker was diagnosed with silicosis, emphysema, and lung cancer at age 70 after working 41 years as a mason laying brick.

A 47-year-old man was diagnosed with severe silicosis after working 22 years as a rock driller. He was diagnosed in 1992. In the spring of 1994, while he was on a ventilator, he died from respiratory failure. His autopsy confirmed advanced silicosis.

A white male worker died of silicosis at age 69 after working 2 years as a tunnel construction worker.

A 55-year-old man was diagnosed in 1994 with simple silicosis after working 30 years as a building renovation mason. Although a lung biopsy revealed silicotic nodules, he was still working as of 1995. This mason used sand (or occasionally coal slag) as the usual abrasive. Periodically, the mason used a handheld masonry saw with no water on the blade.

Cross-sectional studies

Cross-sectional studies (prevalence surveys) are limited in their ability to identify cause-effect relationships. In general, they cannot take adequate account of workers who have left employment because of poor health, and so may under-estimate the effects of occupational exposure. There may also be considerable day-to-day variability in symptoms and inter-individual variability in lung function, which contribute to produce background “noise” in a study. Such factors can mask the ability to detect an effect of occupational exposure. Cross-sectional studies often lack any historical exposure data and information on when ill-health conditions began to develop, and therefore are limited in their ability to characterise dose-response relationships and demonstrate causality. Bearing these limitations in mind, cross-sectional studies can be influential in drawing attention to populations at risk and potential risk factors. All of the cross-sectional studies considered in this review are also summarised in a tabular form in Table 3 in the appendix to this document.

Studies in various groups of construction workers

In a cross-sectional study conducted to determine the prevalence of chronic respiratory disease in construction workers, 4,958 male employees of the German construction industry, aged 40-64 years, were examined by the Occupational Health Service of the Workmen’s Compensation Board for Construction Workers between August 1986 and December 1988 (Arndt et al., 1996 and Rothenbacher et al., 1997). About 78% of the employees invited (n=6356) agreed to participate in the medical examinations, which consisted of an assessment of the occupational and medical history, a physical examination and lung function tests. The following professions were identified: plumbers, carpenters, painters, plasterers, bricklayers, unskilled workers, office employees, engineers and architects. The latter three professions were combined in a common reference group of 207 white-collar employees. Three

different measures were used to characterise the respiratory disease morbidity: 1) the presence of clinical findings on lung auscultation (rales, rhonchi or crackling, or prolonged duration of expiration); 2) $FEV_1 < 70\%$ predicted in the publication by Rothenbacher et al. (1997) and $FEV_1 < 80\%$ predicted in the publication by Arndt et al. (1996); and 3) medical diagnosis of chronic respiratory disease (ICD-9: 490-496, COPD). An active follow-up of all employees was conducted between October 1992 and July 1994 to ascertain whether early retirement due to permanent disability or death had occurred after the initial examination. Life status and employment status at follow-up could be ascertained for 96% and 92% of the participants, respectively. In total, during the period of follow-up (4-5 years), 340 employees were granted a disability pension for health-related reasons, and 141 employees died.

The mean age of the employees varied between 47.9 years (plumbers) and 51.0 years (carpenters). Overall, the employees had worked for about 30 years on average. Smoking habits varied considerably among the various professions with only 41% of the white-collar employees being current smokers compared to 63% of the unskilled workers, 56% of the plasterers and painters, 54% of the bricklayers, 51% of the plumbers and 48% of the carpenters.

A tendency towards a higher prevalence of chronic respiratory disease was seen in most occupations of the construction industry, when compared with white-collar employees. The prevalence of clinical findings on lung auscultation ranged from 2.9% in the white-collar employees to 12.6% in the unskilled workers. The prevalence of reduced FEV_1 ($< 70\%$ of predicted) ranged from 4.5% in the white-collar employees to 12.9% in the unskilled workers, and the prevalence of chronic respiratory disease diagnosis ranged from 3% in the white-collar employees to 9.1% in the unskilled workers.

Although not statistically significant, elevated age-adjusted prevalence rate ratios (PRRs) for the three morbidity measures were calculated for all occupations when compared with the white-collar employees. For example, the prevalence rate ratio for clinical findings on lung auscultation was about 2 (95% CI 0.8-4.8) in carpenters and unskilled workers compared to the white-collar employees.

The only statistically significant finding was an increased prevalence of reduced FEV_1 ($< 80\%$ of predicted) in the unskilled workers compared to the white-collar employees (age-adjusted PRR of 1.5, 95% CI 1.01-2.22). However, it is noted that, since the differences in smoking habits between the exposed workers (up to 63%) and the white-collar employees (41%) were not taken into account, the higher prevalence of chronic respiratory disease seen in most occupations in the construction industry when compared with white-collar employees cannot exclusively be attributed to occupational exposure/factors. As expected, there was a strong association of all three morbidity measures with smoking status (PRRs of 3.8, 2.1 and 4.5 for clinical findings on lung auscultation, $FEV_1 < 70\%$ of predicted, and diagnosis of chronic respiratory disease respectively in smokers compared to non-smokers).

The presence of chronic respiratory disease was associated with early retirement due to permanent disability and death during the follow-up period. For example, relative risks (RR) adjusted for age, occupation, nationality, company size and health centre of 3.2 (95% CI 1.8-5.6) and 2.9 (95% CI 2.0-4.2) for permanent disability and mortality respectively were obtained in employees with a $FEV_1 < 70\%$ of predicted compared to employees with a $FEV_1 \geq 100\%$ of predicted.

Overall, although these results suggest that there was a tendency towards a higher prevalence of chronic respiratory disease in these construction blue-collar workers from Germany when compared with white-collar employees, by failing to take account of the smoking status and of potential socio-economic differences, the evidence is rather weak. However, consistent with other evidence available in the literature (Peto et al., 1983), this study clearly showed that subjects with chronic respiratory disease were at higher risk of disability and death.

In a cross-sectional study conducted to investigate the prevalence of pneumoconiosis and pulmonary function defects in the construction industry, 2,602 construction workers (mean age 58 years \pm 12.7 years) employed at US nuclear weapons sites for facility construction, maintenance and renovation underwent between 1996 and 2001 a respiratory examination (Dement et al., 2003). This included a respiratory history and symptom questionnaire (adapted from the American Thoracic Society questionnaire), a posterior-anterior (P-A) chest radiograph and spirometry testing. Workers also provided a detailed work, exposure, smoking and medical history. Information on exposure histories was obtained through detailed interviews and by incorporating maps, photos and other site-specific materials intended to assist with recall. Workers with prior lung disease diagnoses were excluded from this survey. However, participants were required to have had significant exposures to silica, asbestos or welding fumes during their work in order to be included in the medical screening programme. Chest radiographs were classified according to the ILO (1980) Classification of Radiographs of Pneumoconioses. A parenchymal abnormality consistent with pneumoconiosis was defined as a profusion score of 1/0 or greater for any shape or size of small opacity. Workers with normal chest X-rays were used as the internal referent group. A minimum of 3 acceptable spirometric tracings with reproducibility of FEV₁ and FVC within 5% was obtained. Abnormal spirometry results were identified and classified according to an obstructive pattern (FEV₁/FVC < 95% CI of the predicted value and FVC \geq 95% CI of the predicted value), a restrictive pattern (FVC < 95% CI of the predicted value and FEV₁/FVC \geq 95% CI of the predicted value) and a mixed obstructive/restrictive pattern (FEV₁/FVC < 95% CI of the predicted value and FVC < 95% CI of the predicted value). Workers with FVC \geq 95% CI of the predicted value and FEV₁/FVC \geq 95% CI of the predicted value were considered as having normal lung function.

On average, these workers had been employed at these sites for 14.5 (\pm 10) years and up to 28.6 (\pm 12.5) years in the construction trades. The study population included a wide variety of trades with the highest numbers being pipefitters (19.3%), electricians (18.2%), labourers (15.6%) and carpenters (8.5%). A high proportion of these workers had ever smoked (47.6% and 19.1% of former and current smokers respectively) with only 33.4% of never smokers. Approximately half of the workers reported regular exposure to silica, asbestos or welding fumes during their work history, and more than 80% of the workers reported exposures to these materials on a weekly basis or more often. The prevalence of pneumoconiosis (profusion score \geq 1/0), obstructive lung disease, restrictive lung disease and mixed obstructive/restrictive disease was 2.2% (n=57), 12.2% (n=317), 20.5% (n=533) and 10.0% (n=261) respectively. Symptoms of cough (on most days for at least 3 consecutive months of the year), phlegm (on most days for at least 3 consecutive months of the year) and grade 2 or higher dyspnoea (walking slower than people of the same age because of breathlessness)

were reported by 21.1% (n=706), 28.7% (n=746) and 35.0% (n=911) of the workers, respectively.

Among workers with a normal chest X-ray, 39% (n=758) were found to have an abnormal pulmonary function (11.5% obstructive, 19.2% restrictive and 8.3% mixed). However, only 1.8% (n=27) of those with normal lung function were found to have pneumoconiosis. Among workers with pneumoconiosis, 47.4% (n=27), 21.1% (n=12), 21.1% (n=12) and 10.5% (n=6) were found to have normal lung function, obstructive lung disease, restrictive lung disease and mixed obstructive/restrictive disease, respectively.

Pneumoconiosis was associated with length of employment: an OR adjusted for age, sex, race and smoking habits of 3.6 (95% CI = 1.1-11.6) was obtained by an unconditional logistic regression model for workers employed for 35 years or more compared to those employed for less than 5 years. Workers who reported regular exposure to silica were found to be at higher risk of pneumoconiosis (age-, sex-, race- and smoking-adjusted OR of 1.6) although this did not achieve statistical significance (95% CI 0.9-2.8). Millwrights, sheetmetal workers and ironworkers were found to have a higher prevalence of pneumoconiosis compared to other trades.

The prevalence of obstructive and mixed lung defects increased by smoking category whereas the prevalence of restrictive defects demonstrated no trend. No consistent trend was observed for any of the pulmonary defects with increasing duration of employment after adjustment for age and smoking. A higher prevalence of restriction and a lower prevalence of obstruction were observed among sheetmetal workers.

The results of this study indicate that 12% of this large sample of relatively old US construction workers employed in the trade for a mean of 28 years showed obstructive lung disease (COPD). Unfortunately, there was no comparison to the prevalence of COPD in the general population, and no analysis in terms of age-distribution to put this value into context. Also, there was no information on the severity of the COPD experienced by these workers. These results also showed that the prevalences of restrictive and mixed restrictive/obstructive defects were relatively high (20.5% and 10.0% respectively) with an overall prevalence for lung function abnormalities of 42.7%. This study also showed that pneumoconiosis (profusion score $\geq 1/0$) was present in 2.2% of these workers and was associated with duration of employment and with the reporting of regular past exposure to silica. The possibility of self-selection of diseased workers into the screening programmes cannot be completely excluded; however, 582 workers participated in the work and exposure history component of the survey but chose not to undergo the medical examination. Exposures to asbestos, silica and welding fumes were reviewed for these workers and compared to those for the 2,602 workers included in these analyses. The overall exposure patterns were found to be very similar indicating that it is unlikely that the disease prevalence estimates obtained in this study were biased by a pattern of self-selection.

In a study conducted in France, Pham et al (1972) investigated chronic bronchitis and lung function in a cross-sectional study of 200 male construction workers aged 40-60 years. Chronic bronchitis was identified through a questionnaire administered by a trained physician as the reporting of chronic productive cough for at least 3 months of the year for at least the past 2 years. Workers were placed into subgroups according to their perceived levels of exposure to inhaled contaminants in the workplace. There was no mention of any particular types of construction work. Across the 200 subjects,

21% fulfilled the criteria for chronic bronchitis, a prevalence that was believed by the authors to be higher than one would expect in a non-dust exposed population. However, no clear relationship of chronic bronchitis with perceived workplace exposures was seen. The lack of such a relationship may be due to small group sizes and inaccuracies in exposure categorisation. Lung function data showed that “airways obstruction”, defined as FEV₁/VC ratio <65%, was found in 22% of the study population, with the highest frequency (34%) in those with symptoms of chronic bronchitis.

Overall, the results of this study are suggestive of an excess of chronic bronchitis (21%) and obstructive airways disease (22%) among a group of French construction workers surveyed in the early-1970s; however the precise role of workplace dust and/or irritant exposures could not be determined.

Studies informing on silicosis/mixed-dust pneumoconiosis

Chest X-ray findings were investigated in a cross-sectional study of 1339 Dutch construction workers with expected high exposure to respirable dust containing quartz (Nij et al., 2003b). Originally, 4173 construction workers were selected from a nationwide database based on the description of the current job title. These included tuck pointers, demolition workers, concrete workers (involved with drilling, repairing or blasting concrete and cutting, grinding and sawing grooves in walls), natural stone workers (involved with sawing, engraving and polishing of natural stone), terrazzo workers and pile-top crushers (involved with drilling to break up tops of concrete piles). Of these 4173 subjects, 1339 (32%) participated in the survey, which took place between January and March 1998. All participants were asked to complete a questionnaire with items on occupational history and smoking habits, and a validated questionnaire on respiratory symptoms, and underwent a posterior-anterior chest radiograph. Chronic bronchitis was identified as the reporting of chronic productive cough for at least 3 months of the year for at least the past 2 years. The chest radiographs were read by 3 independent certified “B” readers according to the ILO (1980) Classification of Radiographs of Pneumoconioses. If at least one reader recorded rounded opacities as the predominant type of opacities, the subject was classified as having rounded opacities. All other small opacities classified as category 1/0 and greater were classified as irregularly shaped small opacities.

For the non-response analysis, a randomly selected group of 344 non-participants were contacted by telephone and asked to address questions on the reason for not participating, age, smoking habits, repeated dust exposure at work, respiratory disease and respiratory symptoms. A limited number of personal respirable dust measurements (67 over 34 subjects) were performed among construction workers whose jobs mainly involved concrete drilling, tuck point grinding, cleaning of construction sites and demolition and clearing of rubble. Sampling was conducted during full workdays (6-8 hours). After gravimetric determination of dust on the filters, the quartz content was determined by infrared spectroscopy. By using these measurements, three industrial hygienists, with experience in exposure assessment among construction workers, classified a total of 36 different jobs for quartz exposure on a 10-point arbitrary scale and calculated a proxy for the cumulative quartz exposure (quartz exposure index x duration of exposure).

The mean age of the participants was 42 (± 7.8) years and the average duration of work in the construction industry was 19 (± 9.5) years. Fifty percent of the workers

were current smokers and 30% were ex-smokers. Most of the workers (95%) reported current exposure to mineral dust. The non-response survey did not reveal systematic differences between the participants and the non-participants with regard to age and smoking habits. However, less non-responders than responders reported being exposed to dust (78% vs 95%) and the prevalence of chronic cough was higher in the non-participants (19%) compared to the participants (13%). The prevalence of profusion category $\geq 1/0$ and of profusion category $\geq 1/1$, irrespective of the shape of the opacities, was 10.2% and 2.9% respectively. Most of the predominant small opacities were classified as irregularly shaped and only a minority as rounded (prevalence of 0.77% for rounded opacities of profusion category $\geq 1/0$). In agreement with other experts in this field, the study authors considered the rounded opacities as early signs of nodular silicosis, whereas the irregular opacities as indicative of mixed-dust pneumoconiosis. The highest mean 8 hr TWA respirable dust levels and respirable quartz levels were found for tuck pointers (3.5 and 0.56 $\text{mg}\cdot\text{m}^{-3}$ respectively) and concrete drillers and grinders (2.8 and 0.84 $\text{mg}\cdot\text{m}^{-3}$ respectively) with the lowest values (0.99 and 0.032 $\text{mg}\cdot\text{m}^{-3}$ respectively) in construction site cleaners. A large task-related variability in exposure was observed and the average quartz content of the dust was 12% (range 0.4-40%).

The crude prevalence of small irregular opacities (either category $\geq 1/0$ or $\geq 1/1$) and of small rounded opacities (category $\geq 1/0$) increased with the cumulative quartz exposure index. For example, the prevalence of irregular opacities of category $\geq 1/1$ rose from 0.8% for a cumulative quartz exposure index of less than 4 to 5.3% for a cumulative quartz exposure index of ≥ 15 . In a multiple regression analysis to adjust for potential confounding factors such as age and smoking (smoking does not cause pneumoconiosis, but there are suggestions that shadows on the radiographs can be misinterpreted as pneumoconiosis in heavy smokers, especially in the presence of emphysema), there was still an association between the prevalence of small opacities (irregular or rounded) of category $\geq 1/0$ and the cumulative quartz exposure index, although this was not statistically significant (Prevalence Ratio [PR] of 1.6; 95% CI 0.91-2.81 for a cumulative quartz exposure index of ≥ 15 compared to a cumulative quartz exposure index of 0). When the prevalence of small opacities (irregular or rounded) of category $\geq 1/1$ only was considered, the association with the cumulative quartz exposure index was stronger and statistically significant (PR of 4.69; 95% CI 1.30-16.9 for a cumulative quartz exposure index of ≥ 15 compared to a cumulative quartz exposure index of 0).

Overall, this study showed an increased risk (prevalence of 10.2%) of early signs of mixed-dust pneumoconiosis (profusion score $\geq 1/0$) among a relatively large group of Dutch construction workers involved in grinding, (jack)-hammering, drilling, cutting, sawing and polishing. More pronounced radiographic changes (profusion score $\geq 1/1$) were found in 2.9% of the workers. This study also showed that the increased risk of mixed-dust pneumoconiosis observed was associated with a proxy for cumulative exposure to quartz. Mean exposure levels for the whole studied group were 2.4 $\text{mg}\cdot\text{m}^{-3}$ (8hr-TWA) for respirable dust, 0.3 $\text{mg}\cdot\text{m}^{-3}$ (8hr-TWA) for respirable quartz and 5.7 $\text{mg}\cdot\text{m}^{-3}\cdot\text{years}$ over a period of 19 years for cumulative quartz. These results, however, should be interpreted with caution. First of all, it should be noted that, due to the low (32%) participation rate, the true prevalence of mixed-dust pneumoconiosis in these workers could have been lower or higher. Secondly, the reliability of the exposure

levels and related dose-response relationships is poor because of the limited nature of the exposure assessment and the lack of information on past exposure.

Further work by the same authors on the same study population investigated whether long-term exposure to quartz-containing dust in construction workers, which was found positively associated with mixed-dust pneumoconiosis in the original study described above, can result in lung function impairment (Nij et al., 2003c). In addition to completing a questionnaire with items on occupational history and smoking habits, and a validated questionnaire on respiratory symptoms, and to undergoing a posterior-anterior chest radiograph, all participants (1339) were also administered spirometric lung function tests. Abnormal lung function values were defined as values below the group lower 95% CI limits, corrected for age and height. Lung function data obtained from this group of construction workers were compared with values from a random sample of the Dutch population including working men not owning a business, between 30 and 60 years of age (mean age 43.7), without higher education. This resulted in a reference population of 1350 individuals. In relation to the radiographic readings, profusion category $\geq 1/1$ was considered as an indication of early signs of mixed-dust pneumoconiosis.

As already described in the original study, the prevalence of pneumoconiosis profusion category $\geq 1/1$ was 2.9%. The crude prevalences of chronic (in the past 2 years, for longer than 3 months) cough, chronic cough with sputum, shortness of breath during normal activity and frequent wheezing were 13%, 10%, 9% and 10% respectively. However, no associations between these symptoms and the cumulative quartz exposure index or duration of exposure were observed, after correction for pack-years smoked. Construction workers had a significantly lower average lung function compared to the reference group, after correction for smoking. The mean FEV₁, FVC and PEF (Peak Expiratory Flow) corrected for age and height were respectively 120 ml, 130 ml and 225 ml lower in the construction workers. When construction workers with mixed-dust pneumoconiosis (profusion category $\geq 1/1$) were excluded, the difference in lung function between the two groups was about 10% lower, but still highly significant. Although the cumulative quartz exposure index was not associated with reductions in the lung function values, duration of exposure was positively, but not statistically significantly associated with both FEV₁ ($p=0.1$) and FVC ($p=0.2$).

There was a clear association between radiographic and spirometric abnormalities corrected for age and height. Reduced FEV₁ and FVC values (corrected for age and height) were more prevalent among those with radiographic evidence of mixed-dust pneumoconiosis (profusion category $\geq 1/1$) compared to those without after adjustment for smoking (RR of 3.36; 95% CI 1.44-7.80 for reduced FEV₁ and RR of 2.77; 95% CI 1.00-7.69 for reduced FVC). Pneumoconiosis (profusion category $\geq 1/1$) was statistically significantly associated after adjustment for smoking with a mean reduction in FEV₁ (corrected for age and height) of 267 ml ($p<0.05$) and a mean reduction in FVC (corrected for age and height) of 181 ml ($p=0.09$).

Overall, this study showed that lung function was on average somewhat lower (with mean age- and height-corrected FEV₁ and FVC values 120 and 130 ml lower respectively) among a relatively large group of Dutch construction workers when compared with a comparable reference group; however, no clear dose response with cumulative exposure to quartz was observed. A more significant reduction in lung

function was found among those workers with mixed-dust pneumoconiosis (profusion category $\geq 1/1$).

Ng et al (1987) investigated the prevalence of silicosis in a cross-sectional study of 118 caisson (cylindrical foundation which after excavation is concreted in place) construction workers from Hong Kong. Of the "available" 238 site workers (it is unclear how these subjects were selected) interviewed in relation to their occupational and medical histories, 118 (58%) accepted to undergo chest radiography. The chest radiographs were then categorised according to the ILO (1980) Classification of Radiographs of Pneumoconioses. Data on respirable dust exposure levels and on silica content at these building sites were obtained by conducting an air monitoring programme of 18 caisson construction sites involving the collection of personal (34) and static (53) samples.

Of the 118 subjects (mean age 40 years; range 20 to 62 years) enrolled in the study, 74 were underground caisson workers and 44 surface workers. The number of years spent in caisson underground work averaged 7.5 years (range 1 to 19 years). No cases of silicosis were identified among surface workers, but 12 cases of silicosis (16%) were found among underground workers. All of these cases occurred in those with 10 to 20 years of work experience. One case was of grade 1, 8 cases of grade 2 and 3 cases of grade 3. Respirable silica levels (8hr-TWA) measured at these sites exceeded 0.1 mg.m^{-3} in 65% of the samples, and a median concentration as high as 6.1 mg.m^{-3} (8h-TWA) was found for dry drilling inside the caisson. Overall, these findings showed that these underground caisson construction workers from Hong Kong were at high risk of developing silicosis (prevalence of 16%) from high exposure to respirable crystalline silica.

In a retrospective cross-sectional study of 1056 workers attending the pneumoconiosis clinic in Hong Kong between 1995 and 1999 for compensation assessment, the relationship between confirmed cases of silicosis and the type of occupation, respiratory symptoms and spirometric data was investigated (Law et al., 2001). Of the 1056 workers enrolled in the study, 648 (mean age 57.4 years) were confirmed as cases of silicosis. These had an occupational history (average duration of dust exposure of 24.2 years) involving significant exposure to silica-containing dust and round and/or irregular opacities in the lungs with profusion greater than 1/0 according to the ILO (1980) Classification of Radiographs of Pneumoconioses. The majority (518, 80%) of these confirmed cases of silicosis worked in the construction industry and a high proportion of these were caisson workers or stone splitters. Approximately 69% of the confirmed cases had grade 1 profusion whereas only 2% had grade 3 profusion; 65% and 32% had round and irregular opacities, respectively, and 24.8% showed progressive massive fibrosis (PMF). Approximately 41% had chest X-ray abnormalities consistent with pulmonary tuberculosis and 8% had eggshell calcifications characteristic of silicosis. More than 94% of the workers who sought pneumoconiosis compensation assessment complained of respiratory symptoms such as shortness of breath, chronic productive cough, chest pain and wheezing, and a high proportion (37.4%) suffered from pulmonary tuberculosis. The spirometric measurements of the confirmed cases of silicosis were within the normal ranges, with a mean FVC 97.9% of predicted and a mean FEV_1/FVC 69.5% of predicted. However, significantly poorer FVC and FEV_1 were found among those with PMF. The lack of significant lung function impairment among the cases of simple silicosis

is not surprising if we consider that these workers were self-selected to be able to perform heavy manual tasks, and hence, their lung function parameters should have been on average better than those of the general population.

Overall, the results of this study showed that silica dust exposure in the construction industry was the major cause of silicosis in Hong Kong, accounting for 80% of all the confirmed cases of silicosis (648) attending the pneumoconiosis clinic between 1995 and 1999. A high proportion of these cases were caisson workers or stone splitters. However, the exact nature and composition of the silica dust and the dust exposure levels could not be determined by this study.

In a study detailed above under the heading “Studies in various groups of construction workers” (Dement et al., 2003), the prevalence of pneumoconiosis (X-ray opacities of profusion $\geq 1/0$) among 2,602 US construction workers was 2.2%. Pneumoconiosis was associated with length of employment: an OR adjusted for age, sex, race and smoking habits of 3.6 (95% CI = 1.1-11.6) was obtained for workers employed for 35 years or more compared to those employed for less than 5 years. Workers who reported regular exposure to silica were found to be at higher risk of pneumoconiosis (age-, sex-, race- and smoking-adjusted OR of 1.6) although this did not achieve statistical significance (95% CI 0.9-2.8). Millwrights, sheetmetal workers and ironworkers were found to have a higher prevalence of pneumoconiosis compared to other trades. Overall, this study showed that these construction workers were at risk of developing pneumoconiosis from exposure to silica and that this risk increased with duration of employment.

Five hundred and seventy-six confirmed cases of silicosis reported to NIOSH between 1993 and 1997 were reviewed to evaluate the risk of silicosis among workers in the fast-growing highway repair industry in the US (Valiante et al., 2004). Work history indicated that 8% of these cases resulted from work in the construction industry and that 27% of these were involved in ‘heavy’ construction such as road and highway construction/repair. Road construction and repair workers are potentially exposed to airborne silica dust from activities that create airborne dust, such as sawing, breaking and grinding concrete and other materials that contain silica. Therefore, data on silica exposure levels in this industry were obtained by conducting in 1999 a monitoring programme of 9 highway repair sites involving the collection of 52 personal samples for 7 different typical tasks (operating a jackhammer, sawing concrete, milling concrete, cleaning up concrete, drilling dowels, milling asphalt and cleaning up asphalt). For all of these tasks, except milling and cleaning up asphalt, the mean 8h-TWA levels of respirable silica were above 0.05 mg.m^{-3} and a mean concentration as high as 1.070 mg.m^{-3} (8h-TWA) was found for milling concrete.

Overall, these findings indicate that construction workers constituted 8% of all the cases of silicosis reported to NIOSH in the US between 1993 and 1997. About one quarter of these were workers engaged in road construction and repair. Exposure data were limited but suggest that exposures to respirable crystalline silica can be substantial in this industry sector.

Among 5,898 Swedish construction workers the prevalence of pneumoconiosis category 1/1 and greater was 4.4% (Albin et al., 1992). However, this study was not primarily designed to assess the relationship between dust exposure and health effects,

and exposure and population characteristics were not documented in the paper. Hence, no other relevant information was provided in the study.

Only four (1.5%) cases of silicosis (3 of grade 1/0 and 1 of grade 1/1) were found in a retrospective cross-sectional study of 271 Swedish construction workers involved in concrete work for more than 20 years (Tornling et al., 1992).

The prevalence of 'pneumoconiosis' (ILO category $\geq 1/0$) was 3.7% among 118 construction painters and 3.3% among 314 construction sheet metal workers from Boston, US (Schwartz and Baker, 1988; for more details of the study see section on painters).

From information cited by Lumens and Spee (2001) it appears that in Germany, 27 new compensations due to silicosis have been assigned in 1998 to construction workers.

Summary of studies informing on silicosis/mixed-dust pneumoconiosis

There is only limited information available on the prevalence of silicosis/mixed-dust pneumoconiosis in construction workers, with only two studies providing quantitative exposure data on levels of respirable dust and respirable silica and attempting to analyse dose-response relationships. There are no studies conducted in the UK.

In a large sample of Dutch construction workers involved in activities (grinding, (jack)-hammering, drilling, cutting, sawing and polishing) producing relatively high exposure levels to silica, 10% of the workers showed early signs of mixed-dust pneumoconiosis on chest X-ray (ILO category $\geq 1/0$), and 2.9% had more pronounced changes (ILO $\geq 1/1$). Less than 1% of the workers showed rounded opacities indicative of silicosis. In Swedish construction workers, the prevalence of silicosis (ILO category $\geq 1/0$) was reported to be 1.5% in one study, and the prevalence of 'pneumoconiosis' (ILO category $\geq 1/1$) was 4.4% among 5,898 subjects in another study. Among 2,602 US construction workers the prevalence of 'pneumoconiosis' (ILO category $\geq 1/0$) was 2.2%. It has also been reported that 8% of all cases of silicosis reported to NIOSH in the US between 1993 and 1997 were in construction workers, with approximately one quarter of these being workers engaged in road construction and repair. Among 118 construction painters and 314 construction sheet metal workers from Boston, US, the prevalence of 'pneumoconiosis' (ILO category $\geq 1/0$) was 3.7% and 3.3%, respectively. In Hong Kong, up to 80% of 648 confirmed cases of silicosis were in construction workers, and a high proportion of these were caisson workers and stone splitters. A study in these caisson workers from Hong Kong revealed very high exposures to respirable silica in underground work with a prevalence for silicosis of 16%.

Dose-response relationships were identified with duration of employment in one study and with a proxy of cumulative exposure to silica in another study. Unfortunately, in the latter study, the limited nature of the exposure assessment and the lack of information on past exposure put into question the reliability of the observed dose-response.

Information about different groups/trades is very limited, and, although there is a wide range of trades with significant exposure to silica, the only group clearly singled out

as being at most risk of developing silicosis was the underground caisson workers from Hong Kong.

Overall, these studies show that construction workers from European countries, the US and Hong Kong were at risk of developing mixed-dust pneumoconiosis and silicosis of low radiological grade from cumulative exposure to silica containing dust. Unfortunately, no reliable exposure information is available to characterise dose-response relationships.

Studies in insulation workers

Lung function was investigated in a cross-sectional study of 340 Danish male construction insulation workers and in a reference group of 166 bus drivers (Clausen et al., 1993). The insulation workers were members of the Copenhagen Union of Insulation Workers, and of those invited, 74% agreed to take part in the study. All the participants were sent a questionnaire about their medical and occupational history and underwent spirometric tests. Six years before the present study, 114 of the insulation workers and 59 of the bus drivers had undergone a similar investigation. For these subjects, a longitudinal analysis of the annual decline in lung function was also performed.

Age (ranging from 22 to 64 years with a mean of 42 years in both the insulation workers and bus drivers), height (mean of 177 cm in both the insulation workers and bus drivers) and smoking habits (20% and 16% of never smokers in the insulation workers and bus drivers respectively) were similar in the two groups. In the cross-sectional analysis, the insulation workers had statistically significantly lower FEV₁ values than the bus drivers regardless of smoking. For example, in non-smokers, the mean FEV₁ values for insulation workers and bus drivers were 2.80 and 3.71 l, respectively. FVC was roughly the same in the two groups. In the longitudinal analysis, the average annual decline in FEV₁ was statistically significantly greater in the insulation workers compared to that in the bus drivers regardless of smoking. For example, in non-smokers, the average annual decline in FEV₁ for insulation workers and bus drivers was 154 and 56 ml/year, respectively. It is noted that this decline appears excessively high compared to that reported by other studies in construction workers and therefore it should be treated with caution. Self-assessed former exposure to asbestos among insulation workers was not associated with lung function. It has therefore been speculated by the authors that the reduction in lung function observed in this study might have been due to exposure to modern insulation materials such as rock and glass wool.

Overall, these findings show that this group of Danish insulation workers were at increased risk of developing obstructive lung disease (decline in FEV₁) compared to bus drivers possibly related to exposure to rock and glass wool. It is noted that, although no standardisation of the FEV₁ and FVC values for age and height was performed, given that the two groups had similar mean age and height and that their average FVC values were the same, these results point to a real difference between these two groups. It is also noted that, although the annual decline in FEV₁ observed in these insulation workers was excessively high compared to that reported by other studies in construction workers, there was a clear difference between insulation workers and bus drivers.

Albin et al (1998) investigated whether exposure to insulation wool adversely affected lung function and respiratory symptoms in a cross-sectional study of 45,716 male Swedish construction workers. The study population was chosen to minimise the risk that it would comprise subjects with substantial exposure to asbestos. Asbestos use in the Swedish construction industry was markedly reduced in 1975. Accordingly, only subjects born in 1955 and after were included (median age at first examination 22 years). Data on spirometry, respiratory symptoms and self reported exposure were obtained through nationwide health check ups conducted between 1981 and 1993 by the construction industry's organisation for working environment, safety and health. For those workers (20,086) for which data on spirometry and self reported exposure were available for more than one time point, a longitudinal analysis (mean follow up of 4 years) of the annual decline in lung function and of its relationship with exposure to insulation wool was also performed. Exposure to insulation wool was assessed by combining a job exposure matrix, self reported exposure information and monitoring data on airborne fibre and dust concentrations (mean 8-hr TWA concentrations of respirable fibres and total dust of 0.2-0.9 f/ml and 2-11 mg.m⁻³ respectively) collected at different construction sites during 1978-1990 by personal sampling.

In the cross-sectional analysis, no association between cumulative exposure to insulation wool and reduction in lung function was found after adjustment for smoking and other occupational exposures (asbestos, silica and isocyanates). Adjusted ORs for low (<85% of predicted) VC and FEV₁ did not increase with increasing exposure to insulation wool. However, a strong association was found between exposure to insulation wool and persistent cough during the past 12 months (adjusted OR of 2.59, 95% CI 2.19-3.06 for high exposure to insulation wool compared to no exposure) and between persistent cough and reduced lung function. In the longitudinal analysis, no significant association between exposure to insulation wool and the annual decline in VC and FEV₁ was found after adjustment for smoking and other occupational exposures (asbestos, silica and isocyanates). Overall, the results of this study show that exposure to insulation wool in the Swedish construction industry had no effect on VC or FEV₁ but was associated with an increased prevalence of persistent cough. However, it should be noted that the necessity to study fairly young subjects in order to avoid effects of asbestos exposure also limited the ability to detect a chronic effect of exposure on lung function.

The possible association between chronic bronchitis and exposure to man-made mineral fibres (MMMMF) was investigated in a cross-sectional study of 135,000 male Swedish construction workers examined between 1971 and 1974 (Engholm and von Schmalensee, 1982). Chronic bronchitis was identified as the reporting of chronic productive cough almost every day for at least 3 months every year. Workers were placed into three categories according to their reported duration of exposure to MMMF (no exposure, <3 years and ≥3 years). In non-smokers (40,340), the age-standardised rate ratio of chronic bronchitis in workers exposed to MMMF for at least 3 years compared to non-exposed workers was 2.68. However, in non-smokers as in other smoking categories, a strong association between exposure to MMMF and exposure to asbestos was also found. Overall, although these findings are suggestive of an association between chronic bronchitis and exposure to MMMF among construction workers, it is unclear whether these results reflect a true effect of exposure to MMMF or an effect of exposure to asbestos. It is also unclear whether or not other exposures encountered in the construction industry may have contributed to the development of chronic bronchitis.

A high prevalence (35%) of airflow obstruction (FEV_1 and $FEV_1/FVC\%$ below the lower 95% confidence limits of the predicted values) was observed in a cross-sectional study of 88 construction insulators from British Columbia, Canada with high exposure to asbestos (Kennedy et al., 1991). However, given that this is likely to be associated with exposure to asbestos, no further details of this study are provided here.

Painters

White and Baker (1988) investigated respiratory symptoms and lung function in a cross-sectional study of 225 male construction painters (mean age 41 years), members of two local union affiliates of the US International Brotherhood of Painters and Allied Trades (IBPAT). Of a total of 663 identified members, 37.1% participated in the health screening that took place in 1984. Although both participants and non-participants had similar mean ages and years since joining the union, there is no information on the extent of the exposure to paints and other chemicals and on the prevalence of respiratory problems among the workers that did not take part in the study. It is therefore unclear whether selection bias was introduced in the analysis. At the time of the health screening, all participants were asked to complete a questionnaire covering their medical and occupational history. Questions concerning respiratory symptoms and smoking history were based on the American Thoracic Society, Epidemiology Standardisation Project questionnaire. Additional questions covered acute irritation to eyes, throat and nose. As a measure of a possible link with work, the participants were also asked whether the symptoms got better, worse or stayed the same during weekends and holidays.

Chronic bronchitis was defined as the reporting of cough with phlegm on most days for three months during the year for at least two years. At least three acceptable spirometric tracings for each participant were obtained, and FEV_1 , FVC and FEF_{25-75} measured. The observed values for FEV_1 , FVC, FEF_{25-75} and FEV_1/FVC were considered to be abnormal if, when expressed as percentages of predicted values, they were less than the lower 95% percentile values published by Knudson et al (1983) for a non-smoking, white population. For example, for men aged 40 and over, the FEV_1 was considered abnormal if it was less than the cut-off of 77.2% of the predicted value. If none of these measures of pulmonary function were below the cut-off values, the painter was classified as having no evidence of pulmonary disease. If only the percentage predicted for FEF_{25-75} was below the cut-off value, the painter was classified as having possible small airways disease. If the percentage predicted for FEV_1 was below the cut-off value or the FEV_1/FVC ratio was less than 70%, the painter was classified as having possible obstructive pulmonary disease. If only the percentage predicted for FVC was below the cut-off value, the painter was classified as having possible restrictive pulmonary disease. If the percentages predicted for both FEV_1 and FVC were below the cut-off values, the painter was classified as having possible mixed obstructive/restrictive pulmonary disease.

The prevalence of transient symptoms of irritation, which improved at weekends, increased with increasing number of weeks worked with solvents in the past years. Of the painters with reproducible values for both FEV_1 and FVC ($n=185$), 1.1% had possible small airways disease, 1.6% had possible restrictive disease, 4.9% had possible mixed disease and 16.8% had possible obstructive disease. Longer

employment as a painter was associated with increased prevalence of chronic obstructive disease. Multiple linear regression analysis with smoking (pack-years) and duration of employment as explanatory variables showed a significant association ($p=0.005$ for the t test of the regression coefficient) between years worked as a painter and a mean decrement in FEV_1 (observed $FEV_1 -$ expected FEV_1) of about 11ml in excess of the age-related loss for each year worked. Separate analyses run for painters who had never smoked and for painters who were either current or former smokers showed mean decrements in FEV_1 in excess of the age-related loss for each year worked of about 4 and 13 ml in non-smoking and smoking painters, respectively, which indicates an interactive effect of smoking and duration of employment as a painter on lung function decline.

The prevalence of chronic bronchitis was 24.9% (overall) and the prevalence of shortness of breath was 17.9% (overall). There was little correlation between lung function findings and symptoms, but this is consistent with the medical literature on this subject. The prevalence of chronic bronchitis was significantly associated (chi-squared =11.22, $p=0.0008$) with increased use of spray application methods. For example, the prevalence of chronic bronchitis was 39% in painters who had spent $\frac{3}{4}$ of their time painting using spray application methods compared to 0% in those who had never used these methods.

Overall, the results of this study showed an excess of COPD, chronic bronchitis and other respiratory symptoms such as shortness of breath and acute irritation in a group of unionised construction painters from the US. Almost 17% of the study population met the criteria for diagnosis of COPD. Although there was no comparison of this prevalence to an external control group, separate analyses in non-smokers confirmed the occupational contribution to this disease and analyses in relation to duration of employment indicated a dose-response relationship. This suggests that this group of construction painters were at increased risk of developing COPD and that this risk increased with duration of employment. After adjustment for smoking, a mean decrement in FEV_1 of about 11ml in excess of the age-related loss was estimated for each year worked as a painter. There appeared to be little correlation between impairment in lung function and symptoms, but this is a widely recognised phenomenon and may be due to the fact that the symptoms of bronchitis are largely due to inflammatory changes in the larger bronchial airways whereas the reductions in FEV_1 are more likely to be linked to changes in the smaller airways (Fletcher et al., 1976; Bauer et al., 2001). Almost 25% of the study population reported symptoms of chronic bronchitis. Again, although there was no comparison of this prevalence to an external control group, analyses in relation to the time spent using spray application methods indicated a dose-response relationship. About 1.6% of the study population showed restrictive changes.

The respiratory status of 118 construction painters, members of the Boston district council of the IBPAT was studied and compared with that of a group of construction workers not exposed to paints (314 sheet metal workers) (Schwartz and Baker, 1988). Of a total of 409 eligible union members (active and retired members who were covered under the Union Health and Welfare Plan), only 29% participated in the medical evaluation that took place in 1984. Assessment of selection bias was accomplished by mail questionnaire to non-participants and showed reasonable comparability between participants and non-participants. For comparison purposes, data from a published study of 314 construction sheet metal workers tested in 1981

were reanalysed (Baker et al., 1985). As members of the construction trades, both painters and sheet metal workers had similar demographic characteristics. All participants completed an occupational history questionnaire and the American Thoracic Society (ATS) questionnaire on respiratory symptoms. For each participant five spirometric tracings were obtained and the ATS guidelines were used to determine acceptability. Percent-predicted values for FEV₁ and the FEV₁/FVC ratio were calculated. Chest radiographs were also performed and interpreted according to the ILO (1971) Classification of Radiographs of Pneumoconioses. A parenchymal abnormality consistent with pneumoconiosis was defined as a profusion score of 1/0 or greater for any shape or size of small opacity.

Painters and sheet metal workers were found to represent comparable working populations with similar levels of union participation and similar age (mean ages of 42 and 46 years for painters and sheet metal workers respectively). However, there were more non-smokers among the painters (25.4%) compared to the sheet metal workers (17.5%). Also, work-related exposures for painters included spray paint, silica and asbestos, while sheet metal workers were most often exposed to asbestos, sheet metal dust and welding fumes. The prevalence of pneumoconiosis was similar in both working groups (3.7% in painters vs 3.3% in sheet metal workers). When compared with sheet metal workers, painters reported significant excess symptoms of cough (34.2% vs 23.7%), wheezing (34.2% vs 9.0%) and dyspnoea (40.4% vs 7.4%) even though there were fewer smokers in this group.

Compared to non-smoking sheet metal workers that had been working at least 15 years in the trade, non-smoking painters that had been working for the same length of time had significant decrements in percent predicted FEV₁ (91.1% vs 104.6%) and FEV₁/FVC (75.6% vs 80.0%). There was no significant difference in these lung function parameters between non-smoking painters and non-smoking sheet metal workers that had been working less than 15 years in the trade. However, significantly lower percent-predicted FEV₁ (87.4% vs 94.9%) and FEV₁/FVC (74.1% vs 77.2%) were found in current smoking painters that had been working less than 15 years in the trade compared to current smoking sheet metal workers that had been working for the same length of time. Restricting the analysis to painters, and controlling for smoking, a trend between years of exposure as a construction painter and airflow obstruction was observed. Using a FEV₁/FVC ratio less than 70% as an indication of airflow obstruction and comparing each category to the non-smokers with less than 15 years in the trade, both smokers with less than 15 years in the trade and non-smokers with more than 15 years in the trade had similar increased, but not statistically significant, relative risks (RR=5.0; 95% CI 0.6-39.8 and RR=4.4; 95% CI 0.6-33.7), while smokers with more than 15 years in the trade had a higher risk of airflow obstruction (RR=8.3; 95% CI 1.1-60.9).

Overall, the results of this study show that this relatively small group of unionised construction painters from Boston, US, compared to other construction workers not exposed to paints (sheet metal workers) had a higher prevalence of respiratory symptoms. This study also shows that lung function was poorer in those painters with at least 15 years in the trade.

Asphalt workers

The occurrence of respiratory symptoms, COPD and airflow limitation was investigated in a cross-sectional study (conducted in 1999) of 64 male asphalt workers

and in a reference group of 195 male outdoor construction workers (Randem et al., 2004). The asphalt workers were all the employees of one company located close to Oslo, Norway, and the outdoor construction workers belonged to the same company. Spirometric tests and a questionnaire on respiratory symptoms and smoking habits were administered. Respiratory symptoms and lung function were adjusted for age and smoking. Cases of COPD were defined by a FEV₁/FVC ratio < 0.7 combined with a history of chronic productive cough, breathlessness and/or wheezing. The prevalence of current smoking was similar in the two groups (53% in both). The asphalt workers were on average younger (36 vs 40 years) and had worked fewer years (11 vs 16 years) in the present job than the reference workers. Symptoms of chest tightness (age- and smoking-adjusted OR=2.8, 95% CI 1.3-5.9), shortness of breath on exertion (age- and smoking-adjusted OR=4.1, 95% CI 1.3-13.0), chest wheezing (age- and smoking-adjusted OR=2.6, 95% CI 1.4-4.9) and COPD (age- and smoking-adjusted OR=2.8, 95% CI 1.2-6.5) were all significantly more prevalent among the asphalt workers compared to the outdoor construction workers. FEV₁/FVC was significantly decreased in the asphalt workers compared to the outdoor construction workers (0.78 vs 0.80, p=0.01). Overall, this study shows that this relatively small group of asphalt workers from Norway had an increased risk of respiratory symptoms, lung function decline and COPD compared to other construction workers (a very comparable reference group with respect to education, socioeconomic status and selection for employment).

Electricians

Respiratory symptoms, lung function and radiographic changes among 100 actively employed electricians in Edmonton, Canada, with 20 or more years of union membership (International Brotherhood of Electrical Workers, Local 424) were compared with those of 100 telephone workers from Edmonton Telephones who had 20 or more years of union membership (Hessel et al., 1998). It was believed that the workers with longer experience would be more likely to exhibit adverse health effects if the adverse health effects were associated with employment. The workers at Edmonton Telephones were selected for comparison because, although they spend some of their time on construction sites, they are usually not involved when conditions are very dusty. Of the invited subjects, all males less than 65 years of age selected at random from the union rosters, 80.2% electricians and 72.7% telephone workers respectively participated in the survey, which took place in 1995. It is stated that some of the non-respondents were working outside the Edmonton area during the time of the study. All participants completed an occupational history questionnaire and the American Thoracic Society (ATS) questionnaire on respiratory symptoms. For each participant three spirometric tracings were obtained and the ATS guidelines were used to determine acceptability. Percent-predicted values for FEV₁, FVC, FEV₁/FVC and FEF₂₅₋₇₅ were calculated. Chest radiographs were also taken and interpreted by two experienced chest physicians according to the ILO (1980) Classification of Radiographs of Pneumoconioses.

There was a significant difference in the age distribution of these two groups with the telephone workers having more subjects (27% vs 12%) in the youngest age group (38-45 years). Smoking habits did not differ significantly for the two groups although there were more non-smokers among the telephone workers (34% vs 26%). There were no differences between the two groups for past history of respiratory conditions such as bronchitis, pneumonia or asthma. Compared with telephone workers,

electricians had more chronic phlegm (age-and smoking-adjusted OR=2.74, 95% CI 1.13-6.60) and shortness of breath (age-and smoking-adjusted OR=2.26, 95% CI 1.10-4.67). However, there were no differences in lung function. The prevalence of radiographic changes (pleural abnormalities or parenchymal small opacities) in both groups was low; however, the electricians had more changes (any abnormality) than the telephone workers (9.6% vs 2.1% with an OR=5.03, 95% CI 1.06-23.93). Chronic phlegm and chest tightness were significantly associated with at least 5 years exposure to fumes in industrial construction (age-and smoking-adjusted ORs of 1.65, 95% CI 1.10-2.47 and 1.62, 95% CI 1.14-2.32 respectively).

Carpenters

Respiratory diseases, as defined by ICD-9 diagnoses on medical insurance claims (either private health insurance claims or workers' compensation claims), were studied for a cohort of 10,938 active union carpenters from the US between 1989 and 1992 (Lipscomb and Dement, 1998). Among the 931 cases of lung disease identified, 90 (9.7%), 51 (5.5%) and 22 (2.4%) were cases of COPD (ICD-9: 496), chronic bronchitis (ICD-9: 491) and emphysema (ICD-9: 492) respectively. The majority of the lung disease claims related to bronchitis (ICD-9: 490; 54.9%) and asthma (ICD-9: 493; 22.9%). These findings suggest a high rate (nearly 10%) of respiratory disease claims among these carpenters, with about 18% of these claims related to COPD/chronic bronchitis/emphysema. However, it is noted that no conclusions on the risk of COPD/chronic bronchitis/emphysema in carpenters can be drawn from this study as it is unclear whether or not these claims were work-related and also because there is no information on smoking.

Tunnel workers

In tunnel construction work, high concentrations of dust are produced mainly because activities are undertaken in enclosed or partially enclosed spaces. Dust is generated from rock drilling, rock bolting, grinding, scaling and transport operations. Other important dust-generating activities are blasting rock and spraying wet concrete on tunnel walls for strength and finishing work. Important determinants of exposure are job group, job site, certain tasks (e.g. drilling and scaling), the presence of a cab and breakthrough of the tunnel. Other important tunnelling pollutants are those generated by diesel powered machinery such as nitrogen dioxide, oil mist and diesel exhaust emissions.

Ulvestad et al (2001a) investigated whether exposure to gases and dust in tunnel construction is associated with signs of airway inflammation. A study population of 29 Norwegian non-smoking male tunnel concrete workers (mean age 44 years) and a reference group of 26 non-smoking male outdoor concrete workers (mean age 39 years) were examined between September and November 1998 by acoustic rhinometry (nasal swelling and obstruction) nasal and exhaled nitric oxide (markers of airway diseases), spirometry and a questionnaire on respiratory symptoms. The tunnel workers were subjects who had been performing concrete work for a period of 1 year after the excavation of the tunnel site, but otherwise had no previous tunnel work experience. The reference subjects were recruited from the outdoor construction sites in the vicinity of the tunnel site. None of the subjects reported asthma, which was one of the criteria of exclusion from the study. All participants had to be free from

respiratory infections for the 3 weeks prior to testing. Personal exposures to dust and gases were measured at 15 construction sites on a random sample of workers from the two groups for at least 2 days. Exposures to a number of airborne substances were measured, including total and respirable dust, quartz and nitrogen dioxide.

The tunnel workers had significantly higher exposures to total dust (geometric mean 8h-TWA of 5.4 vs. 1.0 mg.m⁻³), respirable dust (1.61 vs. 0.21 mg.m⁻³), quartz (0.087 vs. 0.003 mg.m⁻³) and nitrogen dioxide (0.9 ppm vs. n.d.) than the outdoor workers. The occurrence of respiratory symptoms such as congested nose (76% vs. 42%), sore throat (65% vs. 23%), productive cough (38% vs. 0%) and chest tightness with wheeze (38% vs. 0%) was higher in the tunnel workers than in the reference subjects. Exhaled nitric oxide (a marker of airway inflammation) was higher in the tunnel workers than in the outdoor workers (8.4 vs. 5.6 ppb) whereas spirometric values were comparable. The tunnel workers also showed nasal mucosa swelling compared to the outdoor workers.

Overall, this study showed that in these workers from Norway, those who had worked in underground construction for 1 year had a significantly increased occurrence of early signs of inflammation of the upper and lower airways (respiratory symptoms, nasal mucosa swelling and increased exhaled nitric oxide levels), compared to subjects who had performed the same job tasks outdoors. No significant differences in spirometric values were found.

The prevalence of respiratory symptoms and airflow limitation and their relationship to years of employment were investigated in a cross-sectional study of 212 Norwegian male tunnel construction workers and in a reference group of 205 other male construction workers (Ulvestad et al., 2000). Of the 417 workers invited, which constituted the total number of subjects employed at 15 different work sites, 100% participated in the study. The tunnel workers included tunnel face workers, shotcreters and concrete workers while the reference group comprised carpenters and iron fixers. The subjects answered a questionnaire on respiratory symptoms and smoking habits and were administered spirometry tests and chest radiographs. COPD was diagnosed in those with a history of productive cough, breathlessness and/or wheezing, and a FEV₁/FVC ratio < 0.7. Personal exposures to dust and gases were measured at 15 construction sites on a random sample of 193 workers from the two groups for at least 2 days. Exposures to a number of airborne substances were measured, including total and respirable dust, quartz, nitrogen dioxide and oil mist.

The tunnel workers and the reference subjects were comparable with respect to age (mean age of 41 and 40 years respectively) and smoking habits (54% and 52% of current smokers respectively). The tunnel workers had a higher geometric mean 8h-TWA exposure to total dust (3.6 vs. 1.05 mg.m⁻³) and respirable dust (1.2 vs. 0.21 mg.m⁻³) than the reference subjects. They were also exposed to significantly higher levels of quartz (geometric mean 8h-TWA of 0.034 vs. 0.003 mg.m⁻³), oil mist (geometric mean 8h-TWA of 0.5 vs. 0.12 mg.m⁻³) and nitrogen dioxide (geometric mean 8h-TWA of 0.5 ppm vs. n.d.). The tunnel workers reported a significantly higher prevalence of respiratory symptoms (cough, shortness of breath on exercise, chest tightness and wheezing) than the reference subjects, with age- and smoking-adjusted ORs ranging from 1.94 to 3.47. The prevalence of COPD was significantly higher in the tunnel workers (14%) compared to that in the reference group (8%), and this was stated to be higher than that found in the general population (5.4%; no further details provided of the age-distribution of this prevalence). FVC (% of predicted) and

FEV₁ (% of predicted) in the tunnel workers decreased significantly with years of employment compared with the other construction workers. For example, in the tunnel workers, FEV₁ (% of predicted) was 90.3% in those with >20 years employed compared to 101.6% in those with <10 years employed while in the reference group it was 99.8% in those with >20 years employed compared to 100.3% in those with <10 years employed. A linear multiple regression model with age and smoking as explanatory variables, predicted that FEV₁ would additionally decrease by 17 ml for each year of exposure to tunnel work compared to 0.5 ml for each year of exposure in the other construction workers. Hence, compared to other construction workers, among these tunnel workers there was a clear accelerated decline in lung function, which was related to years of exposure. None of the tunnel workers had radiological signs of silicosis, which is consistent with the reportedly low levels of exposure to quartz.

Overall, these findings show that these tunnel workers from Norway were at increased risk for accelerated decline in FEV₁, respiratory symptoms and COPD compared to other construction workers (a very comparable group with respect to socio-economic status). This study also shows that the observed decline in lung function was associated with duration of employment.

Although the following study is of a longitudinal design, it is included here with the cross-sectional studies to keep all the studies about tunnel construction workers grouped together. In subsequent work, a subgroup of the cohort investigated cross-sectionally by Ulvestad et al (2000) was followed up for 8 years to obtain more reliable estimates of the effect of cumulative exposure to dust on lung function and respiratory symptoms (Ulvestad et al., 2001b). A group of 96 tunnel workers (79 drillers and 17 shotcreters) and two reference groups of 178 outdoor construction workers (shutters and iron fixers) and 71 white-collar employees (engineers and foremen) were examined in 1991 and re-examined in 1999. From the original cohort of 417 subjects recruited in 1991, 345 (83%) participated in the follow-up study in 1999. Subjects (n=20) aged ≥55 years in 1991 and subjects (n=15) diagnosed with asthma or COPD in 1991 were excluded. Lost to follow-up were 37 subjects: two had died in work accidents, two had left work due to disability (non-respiratory causes), five had changed job and 28 declined to participate. Since these 28 subjects were in 1991 comparable (in terms of mean age, duration of employment, smoking habits and FEV₁ values) to the other subjects, it is unlikely that the results of this follow-up study could have been affected by selection bias. The subjects answered a questionnaire on respiratory symptoms and smoking habits and were administered spirometry tests and chest radiographs. COPD was diagnosed in those with a history of productive cough, breathlessness and/or wheezing, and a FEV₁/FVC ratio < 0.7. Between 1996 and 1999 personal exposures to dust and gases were measured at 16 construction sites on a random sample of workers from each job group (for a total of 157 workers) for at least 2 days. Sampling times were limited to 5-8 hours but results were considered representative for the 10-hour shift periods. Exposures to a number of airborne substances were measured, including total and respirable dust, quartz, nitrogen dioxide and oil mist. Cumulative exposures to each agent were calculated for each worker by multiplying the time spent in each job group by the mean exposure for that job group.

There were no significant differences between the tunnel workers and the two reference populations for age (mean age of 39-41 years in 1991), height (179-180 cm)

and duration of employment (16-19 years in 1991). As expected, compared with the white-collar employees, the proportions of current smokers were higher in the tunnel workers (52% vs. 31%) and the outdoor workers (52% vs. 31%). At the start of the study there were no significant differences in lung function data across the different groups with mean values for FVC and FEV₁ above 100% predicted. Over the 8-year follow up period the outdoor workers and white-collar employees showed similar lung function declines; the two groups were therefore merged into one reference group. Compared with the reference group, and adjusting for age and smoking (pack-years) the drillers and the shotcreters had a significantly larger decrease in FEV₁ (0.43 l and 0.54 l respectively vs. 0.26 l).

The decrease in FEV₁ was associated with cumulative exposure to respirable dust and quartz. A linear multiple regression model with age, smoking and cumulative exposure to respirable dust and quartz as explanatory variables ($R^2_{\text{adjusted}} = 0.15$, which indicates that 85% of the FEV₁ decline is explained by the model), predicted that in a worker 40 years of age, the annual decline in FEV₁ in a non-exposed non-smoker would have been 25 ml (which corresponds to the physiological, age-related decline), in a non-exposed smoker 35 ml (which corresponds to an excess yearly decrease of 10 ml due to smoking), and in a non-smoking tunnel worker 50-63 ml (which corresponds to an excess yearly decline of 25-38 ml due to tunnel work). Hence, among these non-smoking tunnel workers there was a clear accelerated decline in lung function at least double the normal age-related decline. The results of the regression model also showed that in these workers a cumulative exposure to respirable dust of 1 mg.y.m⁻³ would be associated with an excess annual decline in FEV₁ of 10.6 ml (SE = 2.8 ml), and a cumulative exposure to quartz of 1 mg.y.m⁻³ would be associated with an excess annual decline in FEV₁ of 271 ml (SE = 121 ml). Mean 8-hr TWA concentrations of respirable dust ranged from 1.2 mg.m⁻³ for the drillers up to 3.6 mg.m⁻³ for the shotcreters and the correspondent mean cumulative exposures to respirable dust ranged from 9.6 to 25.4 mg.y.m⁻³. For quartz, the mean 8-hr TWA concentrations ranged from 0.019 mg.m⁻³ for the shotcreters up to 0.044 mg.m⁻³ for the drillers and the correspondent mean cumulative exposures ranged from 0.13 to 0.35 mg.y.m⁻³.

As correlations between cumulative exposure to respirable dust and cumulative exposure to total dust ($r_{\text{Spearman}} = 0.97$), nitrogen dioxide ($r_{\text{Spearman}} = 0.82$) and oil mist ($r_{\text{Spearman}} = 0.77$) were high as it was the correlation between cumulative exposure to quartz and cumulative exposure to nitrogen dioxide ($r_{\text{Spearman}} = 0.95$), it was difficult to distinguish between the various tunnelling pollutants in relation to their detrimental effects on the airways. The authors chose to select respirable dust and quartz for their final regression model as both have been well documented to cause chronic airflow limitation. However, it cannot be ruled out that short-term, peak exposures to nitrogen dioxide (from blasting operations and diesel exhaust) and oil mist (from diesel exhaust) might have contributed to the accelerated lung function decline found in these tunnel workers. It should be also noted that cumulative exposure to nitrogen dioxide in particular was a significant variable when tested alone, but not when combined with cumulative exposure to respirable dust or quartz.

Compared with the reference group, and after adjustment for age and smoking, the odds ratios for the occurrence of new respiratory symptoms (cough, shortness of breath on exercise, dyspnoea and wheezing) during the 8-year follow up were statistically significantly increased (ORs ranging from 3.9 to 20.2) in the tunnel workers. The incidence of these symptoms was associated with cumulative exposure

to respirable dust (ORs ranging from 2.7 to 9.1 for 10 mg.y.m⁻³ exposure), but not with cumulative exposure to quartz.

None of the tunnel workers had radiographic signs of pneumoconiosis and there were no cases with new radiographic findings during the follow-up period.

Overall, the results of this 8-year follow-up study show that these tunnel construction workers from Norway were at increased risk of lung function decline and respiratory symptoms. This study also shows that in these tunnel workers cumulative exposures to respirable dust and quartz were the main contributors of the observed airflow limitation.

Again, the following study is of a longitudinal design, but it is included here with the cross-sectional studies to keep all the studies about tunnel construction workers grouped together. A subsequent study by the same researchers involving a 6-year period of follow-up (Bakke et al., 2004) investigated the relation between lung function and cumulative exposure to dust and gases in a group of Norwegian tunnel construction workers that overlapped (191 subjects were the same) with that studied by Ulvestad et al (2001b). The study population was male, and worked as drill and blast workers (n=115), tunnel concrete workers (n=69), shotcreting operators (n=22), and tunnel boring machine workers (n=3). Outdoor concrete workers (n=220), foremen (n=67) and engineers (n=155) served as a low-exposed reference population. Thus the total study population comprised 651 workers followed up by spirometric measurements during 1989-2002 for an average of 6 years. These 651 subjects were employed in one of Norway's major construction companies. All the employees of this company participated in the health check ups, but only those (651) who had more than one lung function measurement were included in the study.

Personal exposures to dust and gases were measured at 16 construction sites on a random sample of workers from each job group (for a total of 157 workers) for at least 2 days. Sampling times were limited to 5-8 hours but results were considered representative for the 10-hour shift periods. Exposures to a number of airborne substances were measured, including total and respirable dust, quartz, nitrogen dioxide, oil mist, oil vapour, formaldehyde and carbon monoxide. Cumulative exposures to each agent were calculated for each worker based on multiplying the time spent in each job group by the mean exposure for that job group. There was no information on the use of respirators but the authors observed that respirators were generally not worn except by shotcreting and tunnel boring workers, who occasionally wore dust masks.

There were no significant differences between the tunnel workers and the reference populations for age (mean age of 39-41 years). However, as expected, smoking was more common among the tunnel workers and the outdoor concrete workers compared to the engineers (53% vs 37%). Among the non-smoking reference group, the annual decline in FEV₁ over the 6-year period was 21 ml. This is consistent with a normal age-related decline. The annual *excess* decline in FEV₁ in non-smoking tunnel workers was 26 ml for drill and blast workers, 31 ml in concrete workers and 21 ml in shotcreters. Hence, among these non-smoking tunnel workers there was a clear accelerated decline in lung function at least double the normal age-related decline. There were no observed excess declines in FEV₁ in the tunnel borers. This same pattern of results was observed for the tunnel workers who smoked, except that such workers suffered a slightly greater loss in lung function compared to the non-smokers.

The engineers in the reference group were a more highly educated group than the exposed population and this may have introduced a bias in the results. However, when the engineers were excluded from analysis there was no difference in the results.

Multiple linear regression analyses in relation to each exposure variable adjusted for age and smoking showed that all airborne contaminants (total dust, respirable dust, quartz, VOC, oil mist, oil vapour, formaldehyde, nitrogen dioxide and carbon monoxide) were statistically significantly associated with the annual declines in FEV₁. However, cumulative exposures to nitrogen dioxide (from blasting operations and diesel powered machines) showed the strongest association, and inclusion of other exposure variables did not improve the model significantly. Furthermore, several of these exposure variables (i.e. oil mist, oil vapour, VOC and formaldehyde) were present at such a low level that they could not be considered a likely cause of the observed lung function decline. A model with age, smoking and cumulative exposure to nitrogen dioxide as explanatory variables ($R^2_{\text{adjusted}} = 0.09$, which indicates that 91% of the FEV₁ decline is explained by the model), predicted that in these workers a cumulative exposure to nitrogen dioxide of 1 ppm.y would be associated with an excess annual decline in FEV₁ of 39 ml (SE = 9.0 ml), which is equivalent to an excess annual decline in FEV₁ of 8.2 ml for a mean 8h-TWA exposure to nitrogen dioxide of 0.21 ppm during a year. Unfortunately, short-term, peak exposures were not considered. Thus, it is uncertain to what extent the strength of this association and the magnitude of the observed FEV₁ decline would have been affected by the short-term peak exposures rather than by the mean 8h-TWA exposure values. In a previous study by the same workers, peak exposures to nitrogen dioxide >10 ppm were observed in drill and blast workers.

Based on the results of this study, the authors concluded that cumulative exposure to nitrogen dioxide was a major cause of the lung function decline observed in these tunnel construction workers.

The paper noted that part of the study population overlapped with that investigated in a previous study by the same authors (Ulvestad et al., 2001b; see above). This previous work had showed that these tunnel workers from Norway were at increased risk of lung function decline and that cumulative exposures to respirable dust and quartz appeared to be the main contributors of the observed airflow limitation. However, in this earlier study, because of the high correlation between nitrogen dioxide and respirable dust, it had not been possible to discriminate between the effects of dust and nitrogen dioxide.

Overall, the results from both studies indicate that these tunnel construction workers from Norway were at increased risk of lung function decline (at least double the age-related decrease). A number of potential causes were involved. Earlier work by these authors suggested that respirable dust and quartz were the principal contributors but the later work indicated a more prominent effect of nitrogen dioxide.

Respiratory symptoms and lung function were investigated in a cross-sectional study of 389 highway and tunnel construction workers (mean age 41.4 years) involved in a large project in the northeastern US between 1992 and 1999 (Oliver et al., 2001). Of these 389 workers, 231 (59.4%) were tunnel workers and 158 (40.6%) operating engineers (99 journeymen and 59 apprentices). The operating engineers were workers who operated heavy equipment such as backhoes, bulldozers, loaders, excavators and cranes. Subjects were recruited through their unions. Age and length of time in the

union for the study participants compared to all union members were very similar. Principal work activities included cut and cover tunnel construction using slurry walls and cast-in-place reinforced and steel composite concrete, excavation and tunnelling under an existing railroad station, and rebuilding existing off-ramp and street decking and new surface roadway exit ramps. All participants were asked to complete a questionnaire covering their medical and occupational history and a questionnaire concerning respiratory symptoms and smoking history, and underwent spirometry tests. Chronic bronchitis was identified as the reporting of chronic productive cough for at least 3 months of the year for at least the past 2 years. An obstructive lung defect was defined by an $FEV_1 < 80\%$ of predicted and an $FEV_1/FVC < 0.7$, and a restrictive lung defect by an $FVC < 80\%$ of predicted and an $FEV_1/FVC \geq 0.7$. Trade and time in the union (average of 11.3 years) were used as surrogates of exposure.

Age varied across the different trades, with the operating engineer journeymen being the oldest on average (46.7 years) and the operating engineer apprentices being the youngest (30.6 years). Participants were predominantly male (91.5%). The highest proportion of current smokers was among the tunnel workers (40.4%) and the lowest among the operating engineer journeymen (17.2%). After adjustment for smoking, restrictive changes were more common among the operating engineers than among the tunnel workers (3.8% vs 1.7%). Obstruction was found in 4.3% of the tunnel workers and in 3.8% of the operating engineers and within the operating engineers it was more common among the journeymen than among the apprentices (6.1% vs 0%). The prevalence of chronic bronchitis was 6.5% and 1.9% respectively in the tunnel workers and operating engineers. Although a non-exposed referent group was not included, the authors considered that the prevalence of chronic bronchitis in the tunnel workers was slightly higher than would be expected in the general population. The prevalence of chronic bronchitis was significantly increased in the tunnel workers compared to the operating engineers (age- and smoking-adjusted $OR=7.50$, 95% CI 1.49-37.37). There was a significant decrease in the prevalence of chronic bronchitis with increasing time in the union for both trades combined, which is consistent with the possibility that symptomatic workers tend to leave the trade.

Overall, the results of this study show that the prevalence of airflow obstruction was similar in tunnel workers (4.3%) and operating engineers (3.8%) involved in a large project in the north-eastern US. Unfortunately, in the absence of a non-exposed referent group, these prevalences cannot be put into context. However, in terms of symptoms, the findings showed that the tunnel workers reported a markedly higher prevalence of chronic bronchitis symptoms compared to the operating engineers.

Post-shift spirometric changes in lung function were investigated in a study of 30 German tunnel workers (mean age 32) working with shotcrete under compressed air (on average about 1 bar excess pressure) (Kessel et al., 1989). No information is provided in the paper on how these subjects were selected. All workers wore for the duration of a shift (8 hours) personal samplers calibrated in compressed air to assess total inhalable dust exposure. Spirometric tests were conducted before and after an 8-hr shift. In addition, potential long-term effects were studied by conducting a longitudinal investigation, in which the initial lung function data (it is unclear whether the pre-shift or the post-shift values were used) were compared with those obtained after employment for about two years.

The total inhalable dust concentrations ranged from 3.2 to 62.1 $mg.m^{-3}$ (8h-TWA) depending on the particular task. The highest 8h-TWA concentration (with a mean of

26 mg.m⁻³) was found among those who guided the shotcrete nozzle, followed by the dumper drivers with 10 mg.m⁻³ and the concrete mixer operators with 7 mg.m⁻³. A full shift exposure to shotcrete under compressed air resulted in a statistically significant increase (by 10%) of the airway resistance (measured by the area under the flow-volume curve) and in a statistically significant decrease of the FVC (by 3%) and FEV₁ (by 4%). In addition, the PEF (maximum expiratory flow) and the MEF₇₅ (maximum expiratory flow when 75% of the FVC remains to be exhaled) decreased by 6% and 8% respectively, and the total inhalable dust concentrations during a working shift correlated with the lung function data. Employment for about two years did not lead to a substantial difference in FVC and FEV₁ even though MEF₂₅ and MEF₅₀ were significantly decreased (by 22% and 18% respectively).

Overall, these findings show that these tunnel workers from Germany had an increased post-shift airway resistance and signs of longer-term airflow obstruction. Although the increased post-shift airway resistance only reflects an acute response to heavy dust exposure, the observed 2-year changes in MEF₂₅ and MEF₅₀ raise concern for the development of chronic respiratory disease in these workers.

Short-term (over a work period of 11 days) lung function changes (a 3% decrease in FVC, a 7% decrease in FEV₁ and a 8% decrease in FEF₂₅₋₇₅) were observed among 24 Norwegian tunnel workers exposed to high peak exposures of nitrogen dioxide (up to 20 ppm) from blasting fumes (Bakke et al., 2001). The lung function values returned to the baseline levels after an exposure-free period of 10 days. Since these short-term changes reflect an acute response to high peak exposures and it is unclear whether or not they may play a role in the development of chronic respiratory disease, no further details of this study are provided here.

Summary of studies in tunnel workers

Most of the research on tunnel workers has been conducted on groups from Norway, although there are also a US- and German-based studies available.

The Norwegian data (3 studies but the same study population) show that these tunnel workers compared to other construction workers were at increased risk of developing chronic respiratory symptoms, accelerated decline in FEV₁ and COPD.

For COPD, a prevalence of 14% was found in one study, and this compared to 8% in outdoor construction workers. In relation to lung function decline, the evidence showed that these tunnel workers from Norway had a clear accelerated annual decline in FEV₁ at least double the normal age-related decline. Dose-response relationships of this decline with years of employment and with cumulative exposure to respirable dust, quartz and nitrogen dioxide (from blasting operations and diesel exhaust) were also reported. One study found that for each year of exposure to tunnel work there was an excess decrease in FEV₁ of 17 ml. An 8-year follow-up study showed that cumulative exposures to respirable dust and quartz were the main contributors to the observed airflow limitation, whilst later work by the same authors indicated a more prominent effect of nitrogen dioxide. Therefore, although there have been attempts to single out the specific agents responsible for this decline, because of the mixed exposures involved, these have not been reliably characterised.

Further support for the increased risk of lung function decline observed in the Norwegian studies is provided by the German study. This study reported early signs

of longer-term airflow obstruction (slight changes in MEF₂₅ and MEF₅₀ after employment for about 2 years) among 30 tunnel workers.

No association between tunnel work and airflow obstruction was found in a US study, although the tunnel workers showed a 7.5 fold higher prevalence of chronic bronchitis compared to a reference group of operating engineers.

In conclusion, evidence from Norway and Germany shows that tunnel construction workers were at increased risk of developing airflow obstruction. Exposures to respirable dust, quartz and nitrogen dioxide were identified as being associated with reductions in FEV₁; however, because of the mixed exposures involved, these associations were not reliably characterised.

Summary of the cross-sectional studies

A large number of cross-sectional studies are available (see table 3). Of these, the majority have investigated respiratory symptoms and lung function defects either in unspecified groups of construction workers or in specific trades, with only few informing on silicosis/mixed-dust pneumoconiosis. Despite the size of the database, there is relatively little quantitative information on exposure conditions; furthermore, when this is available, its representativeness is uncertain.

Three studies, one in Germany, one in the US and one in France, have reported on the prevalence of COPD and respiratory symptoms among relatively large samples of construction workers. In the German study, 12.6% and 12.9% of the workers had chronic respiratory symptoms and COPD (FEV₁<70% of predicted) respectively. Consistent with these findings, the prevalence of COPD was 12% in the US study, while in the French study, the prevalence of chronic bronchitis and airways obstruction (FEV₁/VC<65%) was even higher (21% and 22% respectively). Although these studies failed to take smoking into account or include a comparison group, the magnitude of the prevalences, particularly in an occupational group engaged in manual work that would be expected to have better lung function than average, provides evidence of an excess of chronic respiratory symptoms and chronic airflow obstruction among construction workers. This is further supported by the findings of a study conducted in a relatively large group of Dutch construction workers, which showed a significant decrease in lung function in comparison to other blue-collar workers.

Information about the following trades is also available: insulation workers, painters, asphalt workers, electricians, carpenters and tunnel workers.

For insulation workers, four relatively old studies, one from Denmark, two from Sweden and one from Canada, were located. Of these studies, two, because of confounding by exposure to asbestos, were considered to be not within the scope of this review. Of the other two, only one study identified an increased risk of decline in lung function that was not confirmed by the other study.

For construction painters, only two studies from the late 80s in the US were identified. These studies showed that two relatively small groups of unionised painters had a higher prevalence of symptoms of chronic bronchitis (about 25%) and of other respiratory symptoms. One study also showed that the prevalence of chronic bronchitis was associated with increased use of spray application methods. These painters were also found to have poorer lung function, with one study suggesting that

they were at increased risk of developing COPD (prevalence of 17%) and that this risk increased with duration of employment. After adjustment for smoking, a mean decrement in FEV₁ of about 11 ml in excess of the age-related loss was estimated for each year worked as a painter.

For asphalt workers, electricians and carpenters, one study each was located. The study in carpenters was of poor design, hence no reliable conclusions could be drawn. The study in electricians identified a statistically significantly (approximately 2-fold) higher prevalence of chronic respiratory symptoms than in telephone workers, but there were no differences in lung function between these two groups. The study in asphalt workers showed consistently higher prevalences of at least 2-fold compared to outdoor construction workers, of chronic respiratory symptoms, lung function decline and COPD.

In relation to tunnel workers, evidence from Norway and Germany showed that these workers were at increased risk of developing lung function decline (at least double the normal age-related decrease) and that this risk increased with duration of employment. Exposures to respirable dust, quartz and nitrogen dioxide were identified as being associated with reductions in FEV₁ but because of the mixed exposure situation, other substances were likely to have contributed to the decline in lung function.

In conclusion, the information on specific trades is limited, and although the evidence seems to point towards an excess of respiratory symptoms and airflow obstruction in most trades investigated, it is difficult to identify specific trades at particular risk, with the possible exception of tunnel workers.

Overall, therefore, the available cross-sectional studies suggest that there is an excess of COPD/chronic bronchitis among construction workers with prevalences ranging from 12% up to 22%. Although the majority of the studies lacked data on exposure and were unable to reliably identify specific substances and processes of concern, several showed dose-response relationships with duration of employment. Information on specific trades was limited, but tunnel workers appear to stand out as being at particular risk, with exposures to respirable dust, quartz and nitrogen dioxide appearing to play the most important role.

The prevalence of silicosis/mixed-dust pneumoconiosis in construction workers has not been systematically or widely investigated in the available cross-sectional studies. In a study of over 2,000 mixed construction workers employed at US nuclear plants, there was a 2.2% prevalence of pneumoconiosis (ILO category $\geq 1/0$), and in another study of US construction painters the prevalence was 3.7%, and 3.3% in the reference group of sheet metal workers. In a Swedish study of nearly 6,000 construction workers, the prevalence of pneumoconiosis (ILO category $\geq 1/1$) was 4.4%. In a study of about 1,300 Dutch construction workers from building sites there was a prevalence of pneumoconiosis (ILO categories 1/0 and $\geq 1/1$) of approximately 10% and 3% respectively; chest X-rays clearly consistent with silicosis were found in only 0.77% of these workers. A higher prevalence of silicosis (16%) was found in a study in Hong Kong underground caisson workers.

Overall, there would appear to be a rather low prevalence (around 1%) of silicosis of low radiological severity in general construction workers, but particular trades such as caisson workers may be at particular risk. A higher prevalence (2-10%) of mixed-dust pneumoconiosis, also of low radiological grade, has been found. Unfortunately, no

reliable exposure information is available to characterise dose-response relationships. These radiological changes are likely to be of little or no health significance, although it should be noted that these studies were cross-sectional in design and so lack information on the progression of pneumoconiosis over time.

Longitudinal studies

In a follow-up study of the investigation conducted by Pham et al (1972), 200 male French construction workers aged 40-60 years, analysed in 1971 with respect to symptoms of chronic bronchitis and spirometry indices of obstructive airways disease, were re-examined 3½ years later (Pham et al., 1977). Chronic bronchitis was identified through a questionnaire administered by a trained physician as the reporting of chronic productive cough for at least 3 months of the year for at least the past 2 years. Thirty-two subjects (16%) were lost at follow-up as they could not be located. There was no significant difference in smoking and working habits between those lost at follow-up and the entire group. However, in general, the subjects lost at follow-up were the more severely affected in the first study, both with respect to symptoms and lung function.

Although 25% of the initially asymptomatic subjects reported to have developed symptoms of productive cough, 22% of the initially bronchitic subjects claimed improvement of their symptoms. It has been postulated by the authors that the improved symptomatology noted in 22% of the initially bronchitic subjects may be partly explained by the typical seasonal variation in respiratory symptoms (it was winter at the time of the initial survey and spring at the follow-up investigation). However, a statistically significant decline (no further details provided) in spirometric values (FEV₁ and FEV₁/VC) was observed in all age groups.

Overall, the results of this follow-up study show a decline in lung function of 137 male construction workers after 3½ years. However, it is unclear from the data provided whether this decline is higher than that expected by advancing age, and hence, no conclusions can be drawn.

Two follow-up studies in tunnel construction workers have been described together with the cross-sectional studies investigating tunnel workers.

Case-control studies

In a case-control study conducted to investigate the risk of airway obstruction and respiratory symptoms in occupations involving exposure to dust or irritants, 131 cases of COPD and selected 298 controls without respiratory disease were analysed in relation to their occupations and past exposure (Mastrangelo et al., 2003). The cases of COPD were defined by a clinical history of ≥2 years shortness of breath and/or winter phlegm and an FEV₁<80% of the predicted value, only minimally reversible with bronchodilators. The controls were subjects with other occupational diseases than respiratory disease (e.g. dermatitis, musculo-skeletal disorders, etc.). Both the cases and the referents were patients referred during 1990-1997 for COPD or another occupational disease to the Institute of Occupational Medicine in Padua, Italy either by the Italian National Institute for Workers' Compensation, whenever the diagnosis

was controversial, or by family doctors for therapy. The likely past exposure of each subject was characterised according to occupational category, years spent in a given occupation, date of hire and on the basis of a job-exposure matrix. By using the job-exposure matrix, for each occupational category the likely exposure to biological dust, mineral dust and gas/vapour/fume was classified as none/low or high. Office workers (12 cases and 60 controls) were identified as non-exposed; they were therefore used as the non-exposed group for comparison with the risk of COPD in other occupations.

In construction workers (21 cases and 45 controls) and in painters (16 cases and 21 controls) the odds ratios for COPD, adjusted for age, smoking habits and calendar year at hire were significantly (although with very large CIs) increased at 3.13 (95% CI 1.04-9.45) and 4.69 (95% CI 1.34-16.4), respectively, compared to office workers. In addition, in painters, the age-smoking-adjusted OR for COPD was significantly increased at 1.06 (95% CI 1.01-1.10) for each extra year of work compared to workers with no exposure in this trade (office workers), while in construction workers any such increase was of borderline significance (OR=1.03; 95% CI 0.99-1.07). This indicates that there is a detectable exposure-effect relationship between the risk of COPD and the numbers of years worked in the construction industry and in the painting trade. This study also found that in general the risk of COPD was associated with exposure to organic dust (age-smoking-adjusted OR of 8.86; 95% CI 2.29-34.3 for workers exposed to organic dust vs office workers), gas/vapour/fumes (age-smoking-adjusted OR of 5.83; 95% CI 1.82-18.6) and mineral dust (age-smoking-adjusted OR of 3.80; 95% CI 1.21-12.0).

Overall, these results show that these Italian construction workers and painters compared to office workers were at increased risk of COPD and that this risk increased with duration of employment. The study also claims that exposure to organic dust, gas/vapour/fumes and mineral dust was associated with the risk of COPD; however, because of the limited nature of the exposure assessment, the reliability of this finding is questionable.

DISCUSSION

There is a substantial and broadly consistent body of evidence showing that there are clear associations between the risk of COPD/emphysema/chronic bronchitis and work in the construction industry with risk estimates being more than double those in age- and smoking-matched reference populations.

Several different types of epidemiological study designs have been used in the literature reviewed, including population-based prevalence surveys, mortality studies, cross-sectional studies, a limited number of longitudinal studies and case-control studies. All have their limitations; population-based studies are based on self-reporting of occupation and/or occupational exposures and lack specific information on the nature and extent of exposure; mortality studies based on SMRs and PMRs tend to lack quantitative information on exposure and provide only crude estimates of the excess of a disease in an occupational group compared to the general population; furthermore, if PMRs are used, an excess in one disease might just be because there is a deficit in another; cross-sectional studies only provide information at one point in time making inference of causal relationships between exposure and disease outcome difficult to assess; follow-up studies may suffer from loss to follow-up and changes in

occupational and other circumstances; case-control studies are by nature retrospective and thus may suffer from recall bias.

Methods of diagnosing COPD and defining cases varied between studies. Many used self-reporting of respiratory symptoms, although often the questionnaire developed by the MRC or the ATS for this purpose was used. Others used a report of physician diagnosis of COPD, with or without confirmation by pulmonary function testing. In some cases, the focus of attention was airflow obstruction or lung function decline rather than COPD diagnosis.

Estimation of exposure in many of the studies reviewed was often based on a simple job title, occupation or industry. Only few studies had quantitative exposure measurements, and even when these were available, the limited nature of the exposure assessment and the lack of information on past exposure question their reliability and representativeness. The potential for exposure misclassification was therefore high in the majority of the studies. Some studies focussed on current employment, others on the longest held job, with only few collecting information on lifetime exposures.

Cumulative exposure was the exposure metric most often used when quantitative exposure estimation was carried out. This metric however does not distinguish between low levels of exposure experienced over a long period of time and shorter but much higher (peak) exposures. Although the majority of the available studies failed to address this element in the exposure assessment methodology, two studies in tunnel workers specifically highlighted the importance of considering peak, intermittent exposures to irritant gases such as nitrogen dioxide. Some studies have used length of employment as a surrogate for cumulative exposure, but again this does not take account of high intermittent exposures.

Smoking has been identified as the most important risk factor in the development of COPD and this was clearly shown in many of the studies reviewed. The evaluation of smoking varied between studies with only few collecting data on pack-years. Other important confounders that were sometimes adjusted for included age and socioeconomic status.

In spite of all of these shortcomings, the evidence shows that construction workers are at increased risk of developing COPD/emphysema/chronic bronchitis compared to the general population and also when compared with a number of other occupational groups.

The population-based studies are broadly consistent in showing that construction workers tend to have an increased prevalence of COPD/chronic bronchitis compared to most of the other occupational groups. In general, the prevalences of these ill health conditions were about 2 to 3-fold higher in construction workers compared to age- and smoking-matched controls from other occupations. Most studies did not explore potential substances/processes associated with increased risks of COPD/chronic bronchitis. The few studies that did investigate causative agents did so only at a very general level, and found that, dusts, including wood dust and silica, were noted as being associated with COPD and the symptoms of chronic bronchitis. Specific trades within the construction industry were not investigated by these studies.

The mortality data provide a reasonably consistent body of evidence pointing to a low to moderate excess mortality from COPD in construction workers compared to the general population, with statistically significantly raised PMRs/SMRs ranging from 109 to 207. Cigarette smoking and socioeconomic factors are likely to account for

some of this excess, but occupational exposure plays also a role. A single study in a very large cohort of Swedish workers indicated that occupational exposures to dusts or irritants may be responsible for almost 11% of the mortality from COPD in construction workers, rising to about 50% in never-smokers. There is little information concerning the precise exposures that may be associated with mortality from COPD in construction workers. Dusts, including mineral dust, fumes and gases have been shown to be associated with mortality from COPD in construction workers in one study. Associations have also been found with PAHs specifically in asphalt workers in another study. In terms of specific trades within the construction industry that may be at most risk of mortality from COPD, although clearly raised PMRs/SMRs for COPD and/or emphysema were found in heavy equipment operators, bricklayers, painters and plasterers and electricians, it is difficult to draw any firm conclusions from the available data, mainly because only one study exists for any individual trade.

The available cross-sectional studies suggest that there is an excess of COPD/chronic bronchitis among construction workers with prevalences ranging from 12% up to 22%. Although the majority of the studies lacked data on exposure and were unable to identify specific substances and processes at risk, several showed associations with duration of employment. One study found that for each year of exposure to tunnel work there was an excess decrease in FEV₁ of 17 ml. Information on specific trades was limited, but tunnel workers clearly stood out as being at higher risk compared to outdoor construction workers.

The limited number of longitudinal studies available was conducted in tunnel workers. These workers were shown to be at increased risk of developing airflow limitation with a clear accelerated decline in lung function at least double the normal age-related decline. Exposure to respirable dust, quartz and irritant gases such as nitrogen dioxide were shown to be associated with reductions in FEV₁; however, because of the mixed exposures involved, these associations were not reliably characterised.

Overall, therefore, the findings from numerous studies of different designs and in different countries are reasonably consistent in indicating that construction workers show moderate increases in COPD morbidity and mortality compared to age- and smoking-matched reference groups. Although a number of studies consistently point to a role of dusts and irritants in general, given the diversity of sectors, trades, activities and workplaces investigated, the available data do not allow one to specify with sufficient reliability a particular causative agent or agents. Information on specific trades is limited, but tunnel workers clearly stand out as being at higher risk compared to outdoor construction workers.

It is noted that, with the exception of one population-based study and one mortality study, there are no other studies conducted in the UK. However, it is also noted that many of the studies were relatively recent and all were based on industrialised countries. Thus, given that the type of construction materials and processes undertaken in construction work are likely to be broadly similar across different industrialised countries, it seems reasonable to conclude that the evidence for an association between COPD/chronic bronchitis/emphysema and construction work is likely to be relevant to current working conditions in UK construction workers.

In relation to silicosis/mixed-dust pneumoconiosis, the mortality studies provide evidence to show that among construction workers, deaths can occur from silicosis, but this has not been widely investigated. A US surveillance scheme showed that of 13,744 deaths from silicosis between 1968-1990, approximately 10% were in construction workers. Other studies revealed raised PMRs for silicosis in general construction workers, and in plasterers, cement masons, painters and in road construction and maintenance workers. However, often these reports refer to only a very small number of deaths drawn from very large worker populations, suggesting that these may be relatively isolated cases.

In the available cross-sectional studies, the prevalence of silicosis/mixed-dust pneumoconiosis in construction workers has not been systematically or widely investigated. In a study of over 2,000 mixed construction workers employed at US nuclear plants, there was a 2.2% prevalence of pneumoconiosis (ILO category $\geq 1/0$), and in another study of US construction painters the prevalence was 3.7%, and 3.3% in the reference group of sheet metal workers. In a Swedish study of nearly 6,000 construction workers, the prevalence of pneumoconiosis (ILO category $\geq 1/1$) was 4.4%. In a study of about 1,300 Dutch construction workers from building sites there was a prevalence of pneumoconiosis (ILO categories 1/0 and $\geq 1/1$) of approximately 10% and 3% respectively; chest X-rays clearly consistent with silicosis were found in only 0.77% of these workers. A higher prevalence of silicosis (16%) was found in a study in Hong Kong underground caisson workers.

Overall, there would appear to be a rather low prevalence (around 1%) of silicosis of low radiological severity in general construction workers, but particular trades such as caisson workers may be at particular risk. A higher prevalence (2-10%) of mixed-dust pneumoconiosis, also of low radiological grade, has been found. Unfortunately, no reliable exposure information is available to characterise dose-response relationships. These radiological changes are likely to be of little or no health significance, although it should be noted that these studies were cross-sectional in design and so lack information on the progression of pneumoconiosis over time.

There are no documented cases of silicosis in UK construction workers although 27 German construction workers received compensation for silicosis in 1998, suggesting that there may be some undetected cases in the UK. Overall, it is difficult to draw any firm conclusions concerning pneumoconiosis risks in UK construction workers except perhaps that there is likely to be a relatively widespread prevalence (up to 10%) of pneumoconiotic changes of low radiological severity, such that the health impact is likely to be little or none.

REFERENCES

Albin M, Engholm G, Frostrom K, Kheddache S, Larsson S and Swantesson L (1992) Chest x ray films from construction workers: International Labour Office (ILO 1980) classification compared with routine readings. *British Journal of Industrial Medicine*; **49**: 862-868.

Albin M, Engholm G, Hallin N and Hagmar L (1998) Impact of exposure to insulation wool on lung function and cough in Swedish construction workers. *Occup Environ Med*; **55**: 661-667.

Arndt V, Rothenbacher D, Brenner H, Fraisse E, Zschenderlein B, Daniel U, Schuberth S and Fliedner TM (1996) Older workers in the construction industry: results of a routine health examination and a five year follow up. *Occup Environ Med*; **53**: 686-691.

Baker EL, Dagg T and Greene RE (1985) Respiratory illness in the construction trades: I. The significance of asbestos-associated pleural disease among sheet metal workers. *J Occup Med*; **27**: 483-489.

Bakke B, Ulvestad B, Stewart P, Lund MB and Eduard W (2001) Effects of blasting fumes on exposure and short-term lung function changes in tunnel construction workers. *Scand J Work Environ Health*; **27(4)**: 250-257.

Bakke B, Stewart P and Eduard W (2002) Determinants of dust exposure in tunnel construction work. *Appl Occup Environ Hyg*; **17(11)**: 783-796.

Bakke B, Ulvestad B, Stewart P and Eduard W (2004) Cumulative exposure to dust and gases as determinants of lung function decline in tunnel construction workers. *Occup Environ Med*; **61**: 262-269.

Bang KM, Althouse RB, Kim JH, Game SR and Castellan RM (1995) Silicosis mortality surveillance in the United States, 1968-1990. *Appl Occup Environ Hyg*; **10(12)**: 1070-1074.

Bauer TT, Schultze-Werninghaus G, Kollmeier J, Weber A, Eibel R, Lemke B and Schmidt E-W (2001) Functional variables associated with the clinical grade of dyspnoea in coal miners with pneumoconiosis and mild bronchial obstruction. *Occup Environ Med*; **58**: 794-799.

Bergdahl IA, Toren K, Eriksson K, Hedlund U, Nilsson T, Flodin R and Jarvholm B (2004) Increased mortality in COPD among construction workers exposed to inorganic dust. *Eur Respir J*; **23**: 402-406.

Burstyn I, Boffetta P, Heederik D et al., (2003) Mortality from obstructive lung diseases and exposure to polycyclic aromatic hydrocarbons among asphalt workers. *Am J Epidemiol*; **158** (6): 468-478.

Chisholm J (1999) Respirable dust and respirable silica concentrations from construction activities. *Indoor Built Environ*; **8**: 94-106.

Clausen J, Netterstrom B and Wolff C (1993) Lung function in insulation workers. *Br J Ind Med*; **50**: 252-256.

Dement JM, Welch L, Bingham E, Cameron B, Rice C, Quinn P and Ringen K (2003) Surveillance of respiratory diseases among construction and trade workers at Department of Energy nuclear sites. *American Journal of Industrial Medicine*; **43**:559-573.

D'Errico A, Mamo C, Tomaino A, Dalmasso M, Demaria M and Costa G (2002) Mortality of a cohort of road construction and maintenance workers with work disability compensation. *Med Lav*; **93**: 519-526.

Dong W, Sullivan K and Fletcher T (1995). Mortality of construction workers in the UK. *International Journal of Epidemiology*; **124** (4): 750-757.

Engholm G and von Schmalensee G (1982) Bronchitis and exposure to man-made mineral fibres in non-smoking construction workers. *Eur J Respir Dis*; **63**: 73-78.

Finkelstein M and Verma DK (2005) Mortality among Ontario members of the International Union of Bricklayers and Allied Craftworkers. *Am J Ind Med*; **47**: 4-9.

Fishwick, D, Bradshaw LM, D'Souza W, Town I, Armstrong R, Pearce N and Crane J (1997) Chronic bronchitis, shortness of breath and airway obstruction by occupation in New Zealand. *Am J Respir Crit Care Med*; **156**: 1440-1446.

Fletcher C, Peto R, Tinker C and Speizer FE (1976) The natural history of chronic airflow obstruction. *Br Med J*; **1**: 1645-1648.

GOLD. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease. Executive Summary. Updated 2003. National Institutes of Health. National Heart, Lung and Blood Institute.

Hansen ES (1991) Mortality of mastic asphalt workers. *Scand J Work Environ Health*; **17**: 20-24.

Heederick D, Pouwels H, Kromhout H and Kromhout D (1989) Chronic non-specific lung disease and occupational exposures estimated by means of a job exposure matrix: the Zutphen study. *Internat J Epidemiol*; **18**(2): 382-389.

Heederick D, Kromhout H, Burema J, Biersteker K and Kromhout D (1990) Occupational exposure and 25-year incidence rate on non-specific lung disease: the Zutphen study. *Internat J Epidemiol*; **19**(4): 945-952.

Hessel PA, Melenska LS, Michaelchuk D, Herbert FA and Cowie RL (1998) Lung health among electricians in Edmonton, Alberta, Canada. *J Occup Environ Med*; **40**(11): 1007-1012.

Hnizdo E, Sullivan PA, Bang KM and Wagner G (2002) Association between chronic obstructive disease and employment by industry and occupation in the US population: a study of data from the Third National Health and Nutrition Examination Survey. *Am J Epidemiol*; **156**: 738-746.

Institute of Occupational Medicine (IOM) A community based case-control study of asthma and chronic bronchitis in relation to occupation. Buchanan D, Donnan PT, Cowie HA, Miller BG and Soutar CA (1997). IOM Report TM/97/05.

HSE (1998) Respirable crystalline silica – Exposure assessment document EH74/2. HSE Books.

HSE Occupational Health Decennial Supplement (1995) Edited by Francis Drever. Office of Population Censuses and Surveys. London, HMSO.

Kennedy SM, Vedal S, Muller N, Kassam A and Chan-Yeung M (1991) Lung function and chest radiograph abnormalities among construction insulators. *American Journal of Industrial Medicine*; **20**:673-684.

Kessel R, Redl M, Mauermayer R and Praml GJ (1989) Changes in lung function after working with the shotcrete lining method under compressed air conditions. *Bri J Ind Med*; **46**:128-132.

Knudson RJ, Lebowitz MD, Holberg CJ and Burrows B (1983) Changes in the normal maximal expiratory flow-volume curve with growth and aging. *Am Rev Respir Dis*; **127**: 725-734.

Krzyzanowski M and Jedrychowski W (1990) Occupational exposure and incidence of chronic respiratory symptoms among residents of Cracow followed for 13 years. *Arch Occup Environ Health*; **62**: 311-317.

Lau YWS, Leung MCM, Leung CC, Yu TS and Tam CM (2001) Characteristics of workers attending the pneumoconiosis clinic for silicosis assessment in Hong Kong: retrospective study. *Hong Kong Med J*; **7**: 343-349.

Lebowitz MD (1977) Occupational exposures in relation to symptomatology and lung function in a community population. *Environ Res*; **14**: 59-67.

Linch KD (2002) Respirable concrete dust-silicosis hazard in the construction industry. *Appl Occup Environ Hyg*; **17**: 209-221.

Lipscomb HJ and Dement JM (1998) Respiratory diseases among union carpenters: cohort and case-control analyses. *Am J Ind Med*; **33**: 131-150.

Lumens ME and Spee T (2001) Determinants of exposure to respirable quartz dust in the construction industry. *Ann Occup Hyg*; **45(7)**: 858-895.

MacNee W and Zielinski J (2000) - Chapter 2 from Pulmonary Rehabilitation, Volume 5 of the European Respiratory Monograph 13. Edited by Donner CF and Decramer M.

Mastrangelo G, Tartari M, Fedeli U, Fadda E and Saia B (2003) Ascertaining the risk of chronic obstructive pulmonary disease in relation to occupation using a case-control design. *Occupational Medicine*; **53**: 165-172.

Meyer JD, Holt DL, Chen Y, Cherry NM and McDonald JC (2001) SWORD'99: surveillance of work-related and occupational respiratory disease in the UK. *Occup Med*; **51(3)**: 204-208.

Ng TP, Yeung KH and O'Kelly FJ (1987) Silica hazard of caisson construction in Hong Kong. *J Soc Occup Med*; **37**: 62-65.

Nij ET, Hilhorst S, Spee T, Spierings J, Steffens F, Lumens M and Heederick D (2003a) Dust control measures in the construction industry. *Ann Occup Hyg*; **3**: 211-218.

Nij ET, Burdorf A, Parker J, Attfield M, van Duivenbooden C and Heederick D (2003b) Radiographic abnormalities among construction workers exposed to quartz containing dust. *Occup Environ Med*; **60**:410-417.

Nij ET, de Meer G, Smit J and Heederick D (2003c) Lung function decrease in relation to pneumoconiosis and exposure to quartz-containing dust in construction workers. *Am J Ind Med*; **43**:574-583.

Nij ET, Hohl D, Borm P, Burstyn I, Spierings J, Steffens F, Lumens M, Spee T and Heederick D (2004) Variability in quartz exposure in the construction industry: implications for assessing exposure-response relations. *J Occup Environ Hyg*; **1**: 191-198.

NIOSH (2005) Preventing silicosis and deaths in construction workers. Warning! Exposure to respirable crystalline silica dust during construction activities can cause serious or fatal respiratory disease.

Oliver LC, Miracle-McMahill H, Littman AB, Oakes JM and Gaita RR (2001) Respiratory symptoms and lung function in workers in heavy and highway construction: a cross-sectional study. *Am J Ind Med*; **40**: 73-86.

Petersen JS and Zwerling C (1998) Comparison of health outcomes among older construction and blue-collar employees in the United States. *Am J Ind Med*; **34**: 280-287.

Peto R, Speizer FE, Cochrane AL, Moore F, Fletcher CM, Tinker CM, Higgins ITT, Gray RG, Richards SM, Gilliland J and Norman-Smith B (1983) The relevance in adults of air-flow obstruction, but not of mucus hypersecretion, to mortality from chronic lung disease. *Am Rev Respir Dis*; **128**: 491-500.

Pham QT, Gimenez M, Myre M, Chaspoul H and Martin J (1972) An epidemiological study of bronchitis in building workers. *Bull Physio-path Resp*; **8**: 769-795.

Pham QT, Benis AM, Mur JM, Sadoul P and Haluszka J (1997) Follow-up study of construction workers with obstructive lung disease. *Scand J Resp Dis*; **58**: 215-226.

Randem BG, Langard S, Kongerud J et al (2003) Mortality from non-malignant respiratory diseases among male Norwegian asphalt workers. *Am J Ind Med*; **43**(1): 96-103.

Randem BG, Ulvestad B, Burstyn I and Kongerud J (2004) Respiratory symptoms and airflow limitation in asphalt workers. *Occup Environ Med*; **61**(4): 367-369.

Rappaport SM, Goldberg M, Susi P, Herrick RF (2003) Excessive exposure to silica in the US construction industry. *Ann Occup Hyg*; **47**(2): 111-122.

Ringen K, Seegal J and Englund A (1995) Safety and health in the construction industry. *Annu Rev Public Health*; **16**: 165-188.

Robinson C, Stern F, Halperin W, Venable H, Petersen M, Frazier T, Burnett C, Lalich N, Salg J, Sestito J and Fingerhut M (1995) Assessment of mortality in the construction industry in the United States, 1984-1986. *American Journal of Industrial Medicine*; **28**:49-70.

Robinson CF, Petersen M, Sieber WK, Palu S and Halperin WE (1996) Mortality of Carpenters' Union members employed in the US construction or wood products industries, 1987-1990. *American Journal of Industrial Medicine*; **30**:674-694.

Robinson CF, Petersen M and Palu S (1999) Mortality patterns among electrical workers employed in the US construction industry, 1982-1987. *American Journal of Industrial Medicine*; **36**:630-637.

Rothembacher D, Arndt V, Fraisse E, Daniel U, Flidner TM and Brenner H (1997) Chronic respiratory disease morbidity in construction workers: patterns and prognostic significance for permanent disability and overall mortality. *Eur Respir J*; **10**:1093-1099.

Salg J and Alterman T (2005) A proportionate mortality study of bricklayers and allied craftworkers. *American Journal of Industrial Medicine*; **47**:10-19.

Schwartz DA and Baker EL (1988) Respiratory illness in the construction industry – airflow obstruction among painters. *Chest*; **92**(1): 134-137.

Steenland K and Palu S (1999) Cohort mortality study of 57 000 painters and other union members: a 15 year update. *Occup Environ Med*; **56**:315-321.

Stern F, Schulte P, Sweeney MH, Fingerhut M, Vossen P, Burkhardt G and Kornak MF (1995) Proportionate mortality among construction laborers. *American Journal of Industrial Medicine*; **27**: 485-509.

Stern F and Sweeney MH (1997a) Proportionate mortality among unionized construction operating engineers. *American Journal of Industrial Medicine*; **32**:51-65.

Stern FB, Sweeney MH and Ward E (1997b) Proportionate mortality among unionized construction ironworkers. *American Journal of Industrial Medicine*; **31**:176-187.

Stern FB, Ruder AM and Chen G (2000) Proportionate mortality among unionized roofers and waterproofers. *American Journal of Industrial Medicine*; **37**:478-492.

Stern F, Lehman E and Ruder A (2001) Mortality among unionized construction plasterers and cement masons. *American Journal of Industrial Medicine*; **39**:373-388.

Sullivan PA, Bang KM, Hearl FJ et al (1995) Respiratory diseases risks in the construction industry. *Occupational Medicine: State of the Art Reviews*; **10**: 313-334.

Tornling G, Tollqvist J, Askergrén A, Hallin N and Hogstedt C (1992) Does long-term concrete work cause silicosis? *Scand J Work Environ Health*; **18**: 97-100.

Ulvestad B, Bakke B, Melbostad E, Fuglerud P, Kongerud J and Lund MB (2000) Increased risk of obstructive pulmonary disease in tunnel workers. *Thorax*; **55**: 277-282.

Ulvestad B, Lund MB, Bakke B, Djupesland PG, Kongerud J and Boe J (2001a) Gas and dust exposure in underground construction is associated with signs of airway inflammation. *Eur Respir J*; **17**: 416-421.

Ulvestad B, Bakke B, Eduard W, Kongerud J and Lund MB (2001b) Cumulative exposure to dust causes accelerated decline in lung function in tunnel workers. *Occup Environ Med*; **58**: 663-669.

Valiante DJ, Schill DP, Rosenman KD and Socie E (2004) Highway repair: a new silicosis threat. *American Journal of Public Health*; **94**(5): 876-880.

Verma DK, Kurtz LA, Sahai D, and Finkelstein MM (2003) Current chemical exposures among Ontario construction workers. *Appl Occup Environ Hyg*; **18**: 1031-1047.

Verma DK, Sahai D, Kurtz LA and Finkelstein MM (2004) Current man-made mineral fibers (MMMF) exposures among Ontario construction workers. *J Appl Occup Environ Hyg*; **1**: 306-318.

Vermeulen R, Heederik D, Kromhout H and Smit HA (2002) Respiratory symptoms and occupation: a cross-sectional study of the general population. *Environ Health*; **1**: 5.

Wang E, Dement JM and Lipscomb H (1999) Mortality among North Carolina construction workers, 1988-1994. *Applied Occupational and Environmental Hygiene*; **14**:45-48.

White MC and Baker EL (1988) Measurements of respiratory illness among construction painters. *British Journal of Industrial Medicine*; **45**: 523-531.

Wong O, Morgan RW, Kheifets L, Larson SR and Whorton MD (1985) Mortality among members of a heavy construction equipment operators union with potential exposure to diesel exhaust emissions. *Br J Ind Med*; **42**: 435-448.

Zock JP, Sunyer J, Kogevinas M, Kromhout H, Burney P, Anro JM and the ECRHS Study Group (2001) Occupation, chronic bronchitis, and lung function in young adults. *Am J Respir Crit Care Med*; **163**: 1572-1577.