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IN-DEPTH REVIEW

Occupational chronic obstructive pulmonary disease: a standard of care

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Background	Consistent evidence from population studies report that 10–15% of the total burden of chronic obstructive pulmonary disease (COPD) is associated with workplace exposures. This proportion of COPD could be eliminated if harmful workplace exposures were controlled adequately.
Aims	To produce a standard of care for clinicians, occupational health professionals, employers and employees on the identification and management of occupational COPD.
Methods	A systematic literature review was used to identify published data on the prevention, identification and management of occupational COPD. Scottish Intercollegiate Guidance Network grading and the Royal College of General Practitioner three star grading system were used to grade the evidence.
Results	There are a number of specific workplace exposures that are established causes of COPD. Taking an occupational history in patients or workers with possible or established COPD will identify these. Reduction in exposure to vapours, gases, dusts and fumes at work is likely to be the most effective method for reducing occupational COPD. Identification of workers with rapidly declining lung function, irrespective of their specific exposure, is important. Individuals can be identified at work by accurate annual measures of lung function.
Conclusions	Early identification of cases with COPD is important so that causality can be considered and action taken to reduce causative exposures thereby preventing further harm to the individual and other workers who may be similarly exposed. This can be achieved using a combination of a respiratory questionnaire, accurate lung function measurements and control of exposures in the workplace.
Key words	COPD; FEV_1 decline; lung function; occupation; workplace.

Introduction

Chronic obstructive pulmonary disease (COPD) is characterized by progressive airflow obstruction that is only partly reversible, inflammation in the airways, and systemic effects or co-morbidities [1]. Its primary cause is cigarette smoking, accounting for 80% of the disease burden. COPD is the fourth leading cause of death worldwide [2]. The prevalence of COPD is difficult to determine because the condition does not usually manifest until midlife, may be confused with other conditions and normally requires lung function assessment to confirm a diagnosis. In England and Wales, it is estimated that there are currently 900 000 diagnosed cases, and an estimated 2 million people are thought to have the disease but remain undiagnosed [3].

Other environmental risk factors for COPD have been identified. The evidence supporting these risks varies. For example, the roles of coal, cadmium, silica and biomass in the causation of COPD are relatively well established, and the role of more generic exposures to potentially harmful inhaled exposures in the workplace, subsequently referred to as vapours, gases, dusts and fumes (VGDF), are supported by evidence from a number of studies [4–11]. The

cause of COPD in an individual case is likely to be multifactorial, with multiple personal risk factors and exposures influencing its development and progression.

For the purposes of this study, occupational COPD is defined as COPD where there has been a material contribution made to its development, or severity, by inhaled workplace agent(s). When considering the influence COPD has on the fitness and ability to work, all COPD is considered, and not just that component thought to be caused or made worse by work.

National legislation to prevent exposures that cause COPD varies geographically. In Great Britain, for example, employers and their workers have responsibilities under the Control of Substances Hazardous to Health (COSHH) Regulations 2002 to prevent potentially harmful exposures to agents in the workplace [12]. However, there is little published evidence to assist employers and workers when considering either how best to reduce the risk of COPD related to work, or how best to identify and retain those with COPD in the work environment.

Various documents have highlighted these issues. For example, the recent Clinical Strategy for COPD and Asthma in England and Wales, produced by the Department of Health, emphasized the importance of workplace exposures when considering COPD [13]. This was from the perspective of not only reducing exposures to potentially harmful VGDF but also modifying other risk factors at work, including for example, smoking cessation programmes, and how best to retain those with COPD in the workplace.

Along with examining the evidence base surrounding occupational COPD, this review intends to provide evidence-based guidance for the management of workers with early or established COPD. Although there is now a wealth of literature about COPD and occupation, there remains a lack of practical guidance for health care workers in this area. The intent of this study is therefore to be practically focused and to assist all health care professionals, and others, to understand what the evidence supports in terms of practice and where evidence is still lacking.

Methods

The evidence reported here is taken from the output of a systematic literature search and subsequent rating of evidence quality, concerning the prevention, identification and management of occupational COPD. A comprehensive systematic approach was used, adopting a revised method developed by the Scottish Intercollegiate Guidance Network (SIGN) [14]. This same approach was used for the British Occupational Health Research Foundation reviews of occupational asthma and occupational contact dermatitis and urticaria [14–17]. Since much of the published evidence in occupational health is of low 'strength', the Royal College of General Practitioner three star grading system was also used [18,19].

The literature was searched using MEDLINE, EMBASE, Emcare and the British Nursing Index, with searches spanning from 1960 to the end of 2013. A set of search terms were agreed by the study team and an expert working group.

The search strategy provided 8251 titles for consideration, of which 1371 abstracts were reviewed independently by three researchers. A total of 490 papers were obtained and independently and critically assessed, with 187 of these included in the final evidence tables.

Critical appraisal and grading of the strength of the evidence, depending on the likelihood of bias and consideration of confounding factors, were conducted according to the methods described previously. Where reviewers disagreed about the quality rating, the level of the paper or its relevance to this research, discussion was used to resolve disagreement. Where resolution was not achieved, an additional reviewer was involved in the discussion in order to gain a consensus rating.

Graded evidence-based statements were then constructed based around original research questions. To grade these, the views of the project team were assessed using the breadth of evidence available to them.

Where no evidence was available, guidance was based on consensus agreement between an expert working multidisciplinary group, details of which are given in the Acknowledgements.

Results

Prevalence, causation and costs of occupational COPD

Good quality and varied evidence has identified inhaled occupational exposures as a risk factor for the development of COPD. Its contribution is likely to vary depending on the nature and extent of exposure. In some industries, and under some circumstances, this contribution could be equipotent with cigarette smoking, but more generally, cigarette smoking is likely to have the greater effect. Additionally, certain data support occupational exposures in non-smokers as a cause of COPD [20].

Population studies have identified occupational exposures as accounting for approximately 10–15% of the 'burden' of all COPD in working age adults [11,21–24]. This does not imply that 10–15% of individuals have COPD caused by occupational exposures, or even that 10–15% of the disease can be attributed to work in those who have been exposed. The effect is likely to be much more variable than that though on average 10–15% of the harm done will have been caused by occupational exposures assuming the associations to be causal. This amount or proportion is often termed the population attributable risk percentage or fraction (PAR%) and represents the proportion of the total burden of an illness that can be attributed to a particular cause. These figures

also demonstrate the importance of the overall contribution that occupational exposures make to the development of current COPD, given that COPD is a common condition [25–27].

The consistency, strength and plausibility of previously published data have led others to suggest that they support a causal relationship between a wide range of occupational exposures and COPD, although this point is still actively debated [28–30].

It is additionally important to stress that the data used to derive the median PAR% values are largely epidemiological in nature. As a consequence, there are difficulties with interpretation between studies using differing methodologies and studying the effects of different types of workplace exposure. This is a complex area, and new studies and methods of analysis are constantly being developed.

In terms of specific causative exposures at work, most, primarily population-based, evidence supports only the generic effect of exposures to VGDF as being harmful. Indeed, a large number of mixed method publications have identified 'ever' being exposed to VGDF as an important contributor to COPD prevalence and/or severity, even when corrected for the effects of tobacco smoking. A recent study using a job exposure matrix to classify exposures has also identified that occupational biological and mineral dust exposures, gases/fumes and exposures to VGDF were all associated with the incidence of at least moderate severity COPD [31].

Although it is not possible to be definitive from the current evidence base (which is considerable globally), the data support at least an additive effect of VGDF and tobacco smoking [11].

A1 *** SIGN 2++: Occupational exposures are a risk factor for the development of COPD and account for approximately 10–15% of all COPD [11,21–23].

A2 ** SIGN 2+: The prevalence of COPD in working populations varies and can be as high as 30% in the working age population [25–27].

Varying levels of evidence also support specific exposures at work as causative for COPD, although data here are often less reliable [26,27,32–48]. It was not the intent of this process to rate relative potency of agents capable of causing COPD, although previously reported exposures potentially associated with COPD causation include agricultural dusts (from poultry, animal and arable farming products and practices), asbestos, cadmium, carbon black, (refractory) ceramic fibres, coal mine dust, other dusts (from rubber, cotton, wood, iron/steel and smelting), endotoxin, flour, isocyanates and other chemicals, silica and welding fumes.

In general terms, the body of evidence is stronger for coalmine dust, silica, grain and textiles and less strong for the other identified exposures.

Workers in numerous occupations (rather than exposure type) are at increased risk of COPD, supported by varying levels of evidence [8,39,49–59]. These occupations are listed in Table 1.

Many of these cited studies are based on epidemiological techniques and as a consequence may have encountered difficulties differentiating asthma from COPD.

The potential cost of occupational COPD is considerable. US-based data from 2002 attributed the costs of occupational COPD to be $5.0 billion for the USA annually, of which 56% were direct (health care, insurance costs, etc.) and 44% were indirect costs (loss of earnings, taking care of home, etc.) [60]. The authors summarized that the economic burden of occupational COPD is likely to be reduced if workplace exposures are reduced in parallel with a focus on smoking cessation.

A3 *** SIGN 2++: Occupational agents reported to cause COPD with varying degrees of supporting evidence include coal mine dust, silica, asbestos, refractory ceramic fibres, flour, endotoxin, cadmium, carbon black, agricultural dusts (from poultry, animal and arable farming products and practices), dusts from rubber, cotton, wood, iron/steel and smelting, welding fumes, isocyanates and other chemicals [26,27,32–44].

A4 *** SIGN 2++: Workers reported to be at increased risk of developing occupational COPD include farmers, cotton workers, welders, painters, railroad workers, coal miners and underground workers, carpenters, metal workers, construction workers, cement factory workers and gold miners [8,39,49–58].

Table 1. Occupations at increased risk of COPD

Building services and sales workers	Highway and tunnel workers	Repair services/gas station workers
Cement-exposed workers	Iron, steel and ferrochrome workers	Rubber, plastics and leather manufacturing workers
Cleaners	Inorganic dust-exposed workers	Silicon carbide smelter workers
Coke oven workers	Machine operators	Spray painting and welding
Construction and trade workers	Mechanic and repair jobs	The armed forces
Freight, stock and material handlers	Personal services	Textile mill products manufacturing
Farming and agriculture workers	Pottery workers	Transportation and trucking
Food products manufacturing	Railroad workers	Waitresses
Health care workers	Records processing and distribution clerks	Wood workers

Prevention

There are few specific data relating to the primary, secondary or tertiary prevention of occupational COPD; measures that aim to prevent the onset of disease, detect early disease or prevent worsening of established disease, respectively. Initiatives that were generally described in the literature (none relates directly to COPD) included programmes with multiple components, such as exposure control, educational initiatives and health surveillance, making it difficult to distinguish the effect of one single intervention measure.

There is a lack of evidence relating to the use of pre-placement screening (assessing workers medically before defining their job tasks following employment) as a preventative strategy for occupational COPD. Consequently, this section focuses on the individual risk factors identified for the development of occupational COPD that could be targeted specifically in a preventative programme.

Tobacco smoking

As might be expected, there are many studies, mostly cross sectional in nature, that identify smoking as a risk factor for the presence of respiratory symptoms consistent with COPD in a wide range of occupations exposed to VGDF [11,61–79]. Given the, at least, additive effects of tobacco smoking on the development of COPD related to VGDF exposure, this adds further support for smoking cessation approaches to be encouraged amongst workers.

There were no data identified to link passive or environmental tobacco smoke specifically at work and the development of COPD. Recent data do, however, link such exposures and the development of COPD in active smokers, although the effects seen were less significant in never smokers [80].

B1 ** SIGN 2++: Smoking is the main cause of COPD in most occupational groups [11,61–79].

Other personal risk factors

There are data to support a link between α1 antitrypsin deficiency and the presence of increased respiratory symptoms and airways obstruction in groups of workers exposed to VGDF [81]. The evidence, however, does not currently support the exclusion of workers with known deficiency from dusty jobs. Despite the lack of evidence, as those with PiZZ phenotype are at an increased risk of developing COPD, it may be that they are at increased risk from occupational causes of COPD.

The presence of atopy has also been reported as a risk factor for the development of airways disease in the occupational setting although again the strength of evidence does not support exclusion of atopic individuals from workplaces with potential for VGDF exposure [82–84]. These studies, for example, did not allow accurate differentiation between asthma and COPD.

Prevention of occupational exposures to VGDF

In general terms, traditional control measures to prevent or reduce exposures to VGDF at work are available, although few identified publications dealt with these issues [77,85,86]. In Great Britain, for example, employers have obligations (COSHH regulations) to prevent or control exposures to hazardous substances at work. This can be achieved by carrying out a risk assessment and applying the principles of good practice to minimize exposure. In order to decide on the most appropriate controls used, a 'hierarchy of control' is usually applied. Methods of control at the top of the hierarchy are generally considered the most effective (e.g. elimination of a particular agent from the workplace), but each method has a role to play depending on the work situation (Box 1).

Box 1. The hierarchy of control

- Elimination: complete removal of an agent from the workplace.
- Substitution: replacing a harmful agent with an alternative less harmful agent (e.g. sand blasting substituted by shot blasting).
- Engineering controls such as total enclosure, partial enclosures with local exhaust ventilation and general ventilation (e.g. ventilated cabs for harvesting/composting vehicles).
- Administrative controls, such as segregation of workers, job rotation to limit potential exposure time, good cleaning and maintenance practices, provision of hygiene facilities, information, instruction and training.
- Personal protective equipment, including respiratory protective equipment.

In Great Britain, employers also have general obligations to ensure that workers receive adequate information, instruction and training about hazards in their workplace, and how these are controlled.

As part of the risk assessment process, workers are expected to be aware of hazards they could potentially become exposed to and how to prevent or reduce these exposures. This should normally be achieved by using the controls provided by their employers, in line with their training. For example, it is important that workers know how and when to use: local exhaust ventilation systems, personal protective equipment, RPE, wet methods to suppress dust or, for example, vacuum cleaners with high-efficiency particulate air filters.

Although RPE is the final option in the hierarchy of control, in practice it is frequently used first in workplaces to reduce exposures to VGDF. Indeed, RPE may be the

most appropriate method available, depending on the nature of the work involved, when other measures cannot reduce the exposures to acceptable levels on their own. If RPE is to be used, it must be fit for purpose [87]. Workers should therefore be involved in its selection and be appropriately trained, as this can only offer protection when worn according to instructions provided by the manufacturer, and following an appropriate individual fit test [88].

It is important that workers are informed that workplace controls are in place to protect their health, by preventing or reducing exposures to hazardous substances, and that the worker uses the control measures appropriately. This is particularly important for new and less experienced workers.

In specific terms relating to COPD, evidence supports a relationship between the increased awareness of respiratory problems within a workforce increasing the use of RPE [88]. Of interest, there is no evidence identifying problems using RPE in workers with COPD, although anecdotally certain workers with COPD do report problems. For example, it is relatively common for workers with airways disease to complain that wearing a face mask makes their breathing more difficult. This may be because unpowered RPE requires that workers with COPD generate inspiratory effort to draw air through the filter system, potentially leading to collapse of their airways. Powered RPE could be one way of overcoming such a problem.

B2 * SIGN 2+: The use of respiratory protective equipment (RPE) can reduce the risk of accelerated lung function decline in certain workers [89,90].

B3 * SIGN 3: Training, intervention and increased awareness are needed to increase the effective use of RPE by workers exposed to agents associated with occupational COPD [88].

Behavioural issues and risk perception

The perception of risk of COPD by workers exposed to potentially harmful VGDF is variable, and often influenced by current health status, and other factors including educational levels and smoking status [91–93]. There is no good quality evidence to assist the risk management process specifically for the development of occupational COPD.

Similarly, there is no published evidence to help develop health and safety policies, or behavioural change strategies, to reduce the individual or group risk relating to the development of occupational COPD.

C1 * SIGN 2+: Risk perceptions of occupational COPD are variable amongst workers, and often influenced by health, educational and smoking status [91–93].

Diagnosis, early identification and health surveillance

Diagnosis of occupational COPD

There is no agreed or standard accepted diagnostic approach for occupational COPD. Although the contribution of occupational exposures may often be suspected to be important as a cause of COPD, often in addition to the effects of other risk factors, quantifying this differential causation is still a matter of individual clinical opinion. One study does show that clinicians are able to attribute differential causation between risk factors in individual COPD cases and have reasonable agreement amongst themselves [94].

Questionnaire

The evidence reviewed did not identify an accepted or validated questionnaire for use in workplace surveillance for occupational COPD. If it is decided to use a questionnaire at work, this should ask about symptoms of bronchitis, wheeze, chest tightness and shortness of breath, body mass index and other relevant information including demographic, smoking habit and specific and general occupational exposures. Ideally, a combination of the Medical Research Council and European Community Respiratory Health Survey respiratory questionnaires should be used, given that many of the questions within these have been validated against clinical endpoints [95,96].

Lung function

Measures of lung function at work are normally restricted to those measures that can be made with portable equipment, most importantly the forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC), peak expiratory flow and relaxed vital capacity. These measures are needed for assessing both the presence and absence of COPD (e.g. a normal FEV_1/FVC ratio excludes COPD) and for assessing progression of known lung disease. The methods used to carry these out are described elsewhere, and include American Thoracic Society/European Respiratory Society and American College of Occupational and Environmental Medicine guidance [97,98].

All lung function tests should be performed by competent operators to ensure that any inaccuracy is minimized, particularly when values over time are being compared [99]. Values should then be recorded, compared over time and be made available to the patient or worker.

In practice, measures taken at work will normally be pre bronchodilator, and certain workers will be receiving inhaled treatments that include long-acting bronchodilators. These issues must be borne in mind, considering most of the evidence base in non-occupational studies will relate to post-bronchodilator values.

Longitudinal measures of lung function

It is recognized that longitudinal measurements of pulmonary function, and in particular of FEV_1, are useful in detecting accelerated or 'excess' decline in lung function and this is also true in the workplace [100]. Although there is no consensus relating to how often these measures should be taken, there is evidence to support regular assessment as helpful for identifying COPD [101].

Adopting a system that allows assessment of measured lung function values over time appears a sensible approach. Hnizdo described a simple, visual, analytical tool, Spirometry Longitudinal Data Analysis software (SPIROLA; freely available) as an aid to spirometry monitoring [102,103]. This software helps to identify spirometry data variability and helps to identify individuals with excessive decline in lung function. This approach has been successfully used in the occupational setting and is recommended [104].

Recent National Institute for Occupational Safety and Health guidance suggests that workers with FEV_1 falls of 10–15% (depending on spirometry quality) from baseline over a year should be medically evaluated [104]. This is a complex area and further information is found in Appendix 1 (available as Supplementary data at *Occupational Medicine* Online). Historic work has also suggested that workers in dusty jobs may be self selected with better lung function, and these issues may also need consideration in research studies and clinical situations [105].

In conjunction with reported symptoms and measures of lung function, assessment of cumulative workplace exposures to harmful agents may contribute to assessing COPD risk in workers [106,107].

D1 * SIGN 2⁻: Accelerated lung function decline is a feature of occupational COPD. This can be identified at work if regular measures of lung function are taken [41,90,94,108–110].

Lung function in the workplace environment

Accurate measures of spirometry are key to the success of any workplace scheme. Achieving such accuracy [104] requires the following:

- documented spirometer accuracy and precision,
- a rigorous and standardized testing technique,
- accurate height measurement (without work shoes or boots),
- standardized measurement of pulmonary function values from the spirometry,
- training of spirometry technicians and
- quality assessment of samples of lung function traces.

Interpretation of lung function results usually includes comparison with predicted values and should also evaluate changes in lung function over time [102].

As there is evidence to suggest that subjects with poor spirometry test performance have lower FEV_1 values, and therefore the exclusion of subjects with test failure may cause selection bias during health surveillance or longitudinal data collection, all attempts should be made to obtain accurate measures from all workers within a lung function assessment programme [103].

D2 ** SIGN 2++: Workers at risk of occupational COPD should be assessed through a health surveillance programme including lung function measured by spirometry [90,111].

Prognosis

There are very few data relating to the prognosis of occupational COPD in comparison to non-occupational COPD. It is likely that continued harmful exposure to inhaled agents with the potential to cause COPD, continued smoking and the severity of COPD are all important markers of an adverse outcome.

The presence of COPD irrespective of whether it was caused by workplace agents is associated in many with a reduced quality of life and is a significant cause of sickness absence. As the disease progresses, the personal impact of this disease can increase, with deterioration of health-related quality of life, with greater impairment of ability to work and declining participation in social and physical activities [112,113]. Other financial adverse outcomes are described, including effects on household income, uncertainty for the future and restricted earning abilities. There is additional evidence to suggest that workers with COPD have increased mental health problems leading to depression and anxiety, which may influence fitness or desire to work in individual cases [114,115].

In terms of ability to work with COPD, there is a limited number of previous studies that address this issue [115,116]. These, not surprisingly, conclude that severe lung impairment can cause an inability to work, work inactivity and unemployment. It was concluded that a sizeable (1 in 17 individuals) proportion of non-participation in the overall workforce could be attributed to the presence of COPD [117].

Limited data support adults with asthma or COPD having greater risk of self-reported reduced general health [115]. Furthermore, those with COPD or asthma also had a worse mental health status, and COPD had reduced current employment.

E1 * SIGN 2+: The social and economic burden of occupational COPD may be reduced if workplace exposures are reduced in parallel with a focus on smoking cessation [60].

E2 ** SIGN 2++: COPD in workers is associated with diminished physical and mental health and an increase in depression and anxiety, which can lead to early retirement and subsequent financial impact on workers and their families [112–115].

Clinical management

The medical management of occupational COPD is no different from that already accepted for non-occupational COPD (e.g. National Institute for Clinical Excellence guidance in the UK), although certain evidence does support targeted interventions to improve knowledge,

attitudes and behaviour of all workers at risk of developing occupational COPD [92,118].

Primary and secondary health care providers

Case identification and occupational history

It is possible to assess the occupational contribution to individual cases of diagnosed COPD, although such characterization remains a matter of clinical judgement. Taking an occupational history will help identify possible relevant exposures, guided by the listed relevant occupations and exposures highlighted in this document. Patients and workers should be allowed to talk freely relating to their previous work, and a chronological list of jobs and job tasks should be recorded, where possible including details of known exposures. Knowledge of historic and current exposures may also help make or exclude a differential or coexisting diagnosis (e.g. silicosis, siderosis, coal worker's pneumoconiosis, extrinsic allergic alveolitis, bronchiolitis).

Lung function measurements

Occupational COPD does not differ from non-occupational COPD due to other causes in respect of diagnostic physiology.

Patients with accelerated annual decline in FEV_1 should have an occupational history taken as above, in an attempt to exclude an occupational cause. This is true not only for those with established COPD but also for those without an established respiratory diagnosis.

In general, falls of FEV_1 of 10–15% from baseline over 1 year should be deemed clinically significant and require further investigation. This also applies to those with significant smaller falls year on year (e.g. 100 mls decline each year for 5 years). This may involve advice in relation to smoking cessation and gaining further information about the nature of, and potential to reduce, workplace exposures to VGDF.

Compensation

Patients may ask about compensation issues. It is outside the scope of this article to discuss this in more detail, but information relating to the Industrial Injuries Disablement Benefit process in Great Britain, for example, can be found on the relevant website listed in Box 3. Currently compensation is, however, available in certain circumstances, including workers with a previous history of 20 plus years of coalface work or cadmium fume exposure.

Occupational health management

Workplace approach

A workplace-based risk assessment is central to managing exposures to VGDF that are associated with occupational COPD. The hierarchy of control should be used to plan strategies to prevent or reduce harmful exposures where possible. RPE is the last line of defence in the hierarchy of control, but it may be useful if the combination of other approaches does not reduce exposures effectively.

Workers should be informed about the hazards and associated risks of their work, and also given information about procedures and equipment in place to help reduce the risk to their health. Advice about smoking cessation is important, given the, at least, additive effects of tobacco smoking on the development of COPD related to VGDF exposure.

Although there are no specific studies addressing access to other treatment modalities at work, it seems sensible to stress the importance of access to appropriate medication and workplace smoking cessation initiatives where they are available. European guidance exists to assist employers and workers [119].

There were no studies addressing the effectiveness of workplace-based pulmonary rehabilitation programmes for occupational COPD, although this would appear a sensible area for further investigation, given that many workers with COPD will be required to exert themselves as part of their work.

Respiratory health monitoring programme

Health surveillance may not be a legal requirement for identifying COPD in every workplace; more information can be found on the Health & Safety Executive (HSE) website [120]. However, carrying out periodic measures of lung function for workers exposed to VGDF at work would be regarded as good practice. This needs a planning phase, in order to explain to workers why respiratory health monitoring is needed and what will happen if early lung damage is found. Likely constituents of a health programme include the following:

(i) Assessment of workers' respiratory health before they start a relevant job to provide a baseline, using a questionnaire and lung function assessment. Lung testing should measure the FEV_1 and the FVC.
(ii) Introduction of regular assessment as advised by a health professional with knowledge of the workplace. This should involve further questionnaires and lung function assessments. An explanation must be given of the results to individual workers and a view taken on fitness to work where appropriate. Workers with early COPD are often able to work normally.
(iii) Use of suitably qualified, competent advisers: health professionals involved in a respiratory health monitoring programme should be suitably qualified and follow appropriate spirometry protocols (e.g. American College of Occupational and Environmental Medicine, Association for Respiratory Technology and Physiology. It is important to ensure that all measurements taken are as accurate as possible. There is good guidance available to ensure this.
(iv) Accurate interpretation of information: health professionals should interpret the result trends for groups of workers and individuals, and identify any need to revise the risk assessment. Suitable software is available free, see Box 3.

(v) A responsible person should be appointed at work, to whom any symptoms that occur between assessments should be reported.
(vi) A medical record should be kept and workers should be encouraged to keep a copy of their results in case they change jobs.
(vii) Attendance records can be useful to identify any patterns in sickness absence in the workplace.

Conclusion

This review has highlighted a broad range of literature relevant to COPD and work. Certain areas are well represented in the literature and include causation of COPD by VGDF, and to a lesser degree, the utility of lung function in the workplace. Areas where there are gaps in knowledge include the diagnostic process, sensitivity and specificity of diagnostic investigations, the use of RPE in addition to other control measures to reduce VGDF exposures, the benefits or otherwise of health surveillance for COPD and aspects of workplace-based rehabilitation programmes. These appear to represent good areas for further study.

The hierarchy of control is illustrated in Box 1, a summary of the key points and recommendations is given in Box 2, and useful links are listed in Box 3. Occupations at increased risk of COPD are shown in Table 1. The evidence-based statements are shown in Tables 2 and 3. Certain evidence-based statements are replicated within the appropriate text where relevant.

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Table 2. Evidence-based statements for occupational COPD relating to prevention, causation, behavioural issues and risk perception

Evidence grade	Evidence-based statement	Reference
A1 *** SIGN 2++	Occupational exposures are a risk factor for the development of COPD and account for approximately 10–15% of all COPD	11,21–23
A2 ** SIGN 2+	The prevalence of COPD in working populations varies and can be as high as 30% in the working age population	25–27
A3 *** SIGN 2++	Occupational agents reported to cause COPD with varying degrees of supporting evidence include coal mine dust, silica, asbestos, refractory ceramic fibres, flour, endotoxin, cadmium, carbon black, agricultural dusts (from poultry, animal and arable farming products and practices), dusts from rubber, cotton, wood, iron/steel and smelting, welding fumes, isocyanates and other chemicals	26,27,32–44
A4 *** SIGN 2++	Workers reported to be at increased risk of developing occupational COPD include farmers, cotton workers, welders, painters, railroad workers, coal miners and underground workers, carpenters, metal workers, construction workers, and cement factory workers and gold miners	8,39,49–58
B1 ** SIGN 2++	Smoking is the main cause of COPD in most occupational groups	11,61–79
B2 ** SIGN 2+	The use of RPE can reduce the risk of accelerated lung function decline in certain workers	89,90
B3 * SIGN 3	Training, intervention and increased awareness are needed to increase the effective use of RPE by workers exposed to agents associated with occupational COPD	88
C1 * SIGN 2+	Risk perceptions of occupational COPD are variable amongst workers and often influenced by health, educational and smoking status	90–93

Table 3. Evidence-based statements for occupational COPD relating to early identification, health surveillance, management and prognosis

Evidence grade	Evidence-based statement	Reference
D1 * SIGN 2^-	Accelerated lung function decline is a feature of occupational COPD. This can be identified at work if regular measures of lung function are taken	41,90,94,108–110
D2 ** SIGN 2++	Workers at risk of occupational COPD should be assessed through a health surveillance programme including lung function measured by spirometry	90,111
E1 * SIGN 2+	The social and economic burden of occupational COPD may be reduced if workplace exposures are reduced in parallel with a focus on smoking cessation	60
E2 ** SIGN 2++	COPD in workers is associated with diminished physical and mental health and an increase in depression and anxiety, which can lead to early retirement and subsequent financial impact on workers and their families.	112–115

Box 2. Key points: Summary and recommendations

- There is consistent evidence from population studies that a median of 10–15% of the total burden of chronic obstructive pulmonary disease is associated with exposure to inhaled vapours, gases, dusts and fumes in the workplace. There is wider variation in this value across the entire evidence base. This is the proportion of chronic obstructive pulmonary disease that could be eliminated in the future if such harmful exposures were controlled adequately.
- Tobacco smoking also increases the harm produced by vapours, gases, dusts and fumes at work. All available support should be given to such workers to help them quit.
- There are a number of specific workplace exposures that are established causes of chronic obstructive pulmonary disease. Taking an occupational history in patients or workers with possible or established chronic obstructive pulmonary disease will identify these.
- Traditional control measures to prevent or reduce exposures to vapours, gases, dusts and fumes at work are likely to be the most effective methods of reducing occupational chronic obstructive pulmonary disease. Such reduction in exposure may also reduce other health risks including, for example, the risk of occupational asthma, extrinsic allergic alveolitis and pneumoconiosis.
- Identification of workers with rapidly declining lung function, irrespective of their specific exposure, is important as they are likely to need further medical and exposure assessment. These individuals can be identified at work by accurate annual measures of lung function. This may be possible to achieve either by existing lung function programmes or by existing health surveillance schemes.
- Early identification of cases with chronic obstructive pulmonary disease is important so that thought can be given to cause and action taken to reduce causative exposures. This can be achieved in the workplace using a combination of a respiratory questionnaire and accurate lung function measurements.
- Occupational chronic obstructive pulmonary disease is associated with adverse mental health, financial and social outcomes.

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Conflicts of interest

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Disclaimer

The contents of this paper, including any opinions and/or conclusions expressed, are those of the authors alone and do not necessarily reflect HSE policy.

Box 3. Useful links

HSE COPD guidance: www.hse.gov.uk/pubns/guidance/ and http://www.hse.gov.uk/copd/index.htm (5 March 2014, date last accessed).

COSHH essentials: www.hse.gov.uk/coshh/essentials/ (5 March 2014, date last accessed).

British Lung Foundation website COPD section: http://www.lunguk.org/you-and-your-lungs/conditions-and-diseases/copd (5 March 2014, date last accessed).

Department of Health website: an outcomes strategy for people with COPD and asthma in England. This document contains relevant comments about the workplace. http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_127974 (5 March 2014, date last accessed).

Association of Respiratory Technologists and Physiologists: for guidance on physiology testing http://www.artp.co.uk/ (5 March 2014, date last accessed).

SPIROLA: a longitudinal spirometry data monitoring software http://spirola.com/.

Department of Work and Pensions: industrial injuries disablement benefit information is available from http://www.dwp.gov.uk/index.shtml (5 March 2014, date last accessed).

European Agency for Safety and Health at Work: E-facts sheets http://osha.europa.eu/en/publications/e-facts/ (5 March 2014, date last accessed).

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