Chronic Respiratory Disease

Chronic Respiratory Disease
7(2) 113–122

DOI: 10.1177/1479972309354690
crd.sagepub.com
SAGE

Chronic obstructive pulmonary disease and the workplace

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Abstract

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disease associated with increasing morbidity and mortality worldwide. Whilst tobacco smoking is the important cause, other cuases are regognised. This article discusses the contribution that harmful inhaled occupational exposures make to the overall burden of COPD, and goes on to discuss other aspects of the COPD workplace interface. Prevention is key. All healthcare professionals have a resposibility to consider workplace issues when dealing with their COPD patients.

Keywords

COPD, workplace, occupation, emphysema, chronic bronchitis

Introduction

Chronic obstructive pulmonary disease (COPD) is a slowly progressive, potentially highly disabling, respiratory condition with many potential causes. A better understanding of these causes paves the way for effective interventions to reduce the future incidence of this unpleasant condition.

Whilst cigarette smoking is widely recognized as the most important cause, the specific relationship between COPD and the workplace is important, and the perception of this relationship will depend on the experience of each individual health care worker and on the nature of their previous contact with COPD patients and workplaces. This relationship is now better understood, although there is considerable scope for further research.

Whilst those with an interest in historic workplace exposures may largely regard this relationship as causal, implying that certain work exposures can cause COPD, others may see the clear financial and social disadvantages suffered by COPD patients unable to work. As COPD is becoming more common,[1-3] rapidly changing population demographics will inevitably lead to longer working lives for many, including those with chronic respiratory disease. Whilst this change represents an opportunity for workplaces to appropriately adapt to allow COPD patients to work comfortably, this change will also pose new challenges.

The scope of this article is to review the many ways in which workplaces and COPD interact, from historic and current causative factors, through to ways in which COPD patients cope with work, and how they may cope with the increasing demands of future work.

The individual

One of the most commonly asked questions by patients with COPD who have worked in a dusty environment is, "was it my job that left me like this?" Whilst this is a very reasonable question to ask, the answer may be difficult to give with any degree of certainty. Indeed, whilst patients very commonly raise this issue during consultations with health care workers, and ask how their previous exposures to vapours, gases, dusts and fumes (VGDF) have influenced and contributed to their current respiratory diagnosis and disability, the current evidence base does not support an accurate individual assessment of likely causes.

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Even today, evidence to help assess cause(s) of COPD in an established case is generally lacking (for example the relative contributions of smoking, coal dust exposure and family history of COPD in a retired coal miner). Whilst results from studies discussed later in this article are broadly useful in advising patients about the general harm caused by dusty occupations, the results do not normally translate sufficiently well to help inform individual cases. This is probably because most previous studies have been designed to assess the respiratory harm caused by individual workplaces with known exposures, or to measure excesses of COPD in certain population groups. The closest patients may come to such an estimate (in certain countries, including England) may be from the result of deliberations concerning a common law action against previous employers, when apportionment of harm in an individual case may be estimated. An example of such medico-legal estimation was seen relatively recently in respect of compensation for coal miners paid by the UK government for the development of chronic bronchitis and emphysema following legal action in the High Court.[4] This action involved eight test cases on behalf of a collective group of claimants in the hope that some general principles could be agreed to assist compensating future similar cases. The trial and subsequent judgement lasted 17 months, reviewed about 500 medical articles and reports and included approximately 15,000 pages of evidence. Indeed, despite the complexity of the evidence, a relatively simple algorithm was eventually used to calculate the relative amounts of COPD due to smoking and coal dust exposure in each individual miner. This was done in order to apportion damages, so that miners were only compensated for the proportion of their COPD thought to be caused by coal dust exposure.

With relation to how COPD impacts on working life at an individual level, it is interesting that COPD patients seldom spontaneously mention loss of working life although are happy to talk about this once asked directly. Perhaps consultations focus more on the medical harm potentially caused by workplace exposures and less on the financial and other consequences of this harm. As this article will argue, COPD is a potentially important cause of loss of working life, and this potential impact is likely to require further thought as greater demands are placed on ever-ageing workforces.

Biological plausibility

COPD is characterized by a combination of emphysema and small airway narrowing, both of which are potentially caused by harmful occupational exposures. For example, the roles of sulphur dioxide,[5] vanadium[6] and endotoxin[7] as causes of airway damage are all supported by experimental evidence. The latter exposure is of more general relevance to respiratory harm in the workplace and has been more widely implicated in acute lung injury,[8] asthma,[9] byssinosis[10] and other organic dust–related diseases such as organic dust toxic syndrome.[11]

There are also good experimental data linking certain workplace exposures and the development of emphysema. For example, cadmium is well recognized to cause emphysema in exposed workers,[12] although the prevalence of COPD primarily due to cadmium is difficult to estimate. In this case-referent study, 101 cadmium workers were reviewed with chest x-rays and lung function tests and compared to a group of workers not exposed to cadmium, but matched for age, sex and employment status. Those who had worked with cadmium had greater levels of emphysema, the severity of which became more severe with higher levels of cadmium exposure. Interestingly, cadmium is also a significant constituent of cigarette smoke[13] and also has been shown to cause emphysema in animals.[14,15]

Despite evidence supporting individual occupational exposures causing COPD, however, the majority of epidemiological evidence supports, instead, the more general ‘self-reported’ exposure to VGDF as being a risk factor for the presence of COPD. Interestingly, evidence supporting the biological plausibility of such general exposures causing COPD remains less advanced than for certain specific exposures.

Harmful workplace exposures

The relationship between COPD (or at least chronic respiratory symptoms) and potentially harmful workplace exposures has been known about for many centuries, being cited as early as the 15th century. It was Ramazzini[16] in the 17th century, followed by Greenhow[17] and others in the latter centuries who popularized this long-suspected connection. Although respiratory harm was suspected in certain workers, the actual size of this ‘occupational COPD effect’ was not known (nor was it even assessable at that time).

Despite these very early suggestions of harm associated with exposure to dusty work environments,

a concerted approach to reduce such exposures was not forthcoming. In fact, due to the natural development of technology, and of changes brought about, certainly in the United Kingdom, by the industrial revolution, greater numbers of workers were potentially exposed to harmful indoor working environments.

In parallel with such industrial developments was a greater understanding of epidemiology applied to the workplace, and also a greater appreciation of the difficulties such studies encountered, including factors such as the healthy worker effect and how best to estimate accurate lung function in groups of workers.

Early epidemiological studies (for example Brinkman et al.[18] and Cornwall et al.[19]) began to specifically comment on the relationship between symptoms suggestive of COPD (shortness of breath and regular sputum production) well before accurate physiological measures could be recorded easily in workplaces. In fact, the latter study made a comment about symptoms of bronchitis, and how these affected sickness absence rates in a working group. In other words, by using respiratory symptoms as surrogate markers of airways disease in these early studies, information about causative effects of workplace exposures on lung disease, and the consequences of these diseases in the workplace began to emerge.

The advent of accurate workplace measures of FEV_1 and FVC allowed a much greater understanding of the natural history of occupational COPD, and early studies addressed this issue by studying certain well-defined working groups. For example, Richard Schilling used early portable measures of lung physiology to better define the acute pulmonary response to inhaled cotton dust in groups of textile workers.[20] This study also made measures of dust exposure and was able for the first time to construct dose-response relationships from cross-sectional epidemiological studies. Much of the subsequent understanding of byssinosis and the cotton-related COPD effect is predicated on these original types of approach.

Longitudinal studies

Longitudinal studies offer a unique opportunity to study COPD and work, and allow some account to be taken of the healthy worker effect. Whilst many examples exist, coal and welding exposures are briefly discussed here.

The potential for coal dust exposure to cause lung harm in the form of coal workers pneumoconiosis has been documented for many years,[21] although the ability for coal dust to cause other changes, and particularly those of COPD, has been the subject of previous and current debate. Coggon et al.[22] reviewed the available evidence at the time of the High Court judgement on coal miner's compensation, and concluded that 'the balance of evidence points overwhelmingly to impairment of lung function from exposure to coal mine dust, and this is consistent with the increased mortality from COPD that has been observed in miners.' The actual mechanism of how coal dust causes loss of lung function was however less clear, although the development of centrilobular emphysema, independent to the effects of cigarette smoking, was felt to be the most plausible explanation.

Data from shipyard workers[23] studied in the late 1970s significantly assists the evidence base in relation to both annual lung loss in welders and the potential interaction of welding fume exposure with cigarette fumes. Six hundred and nine shipyard workers were studied, although only 488 of these were available for subsequent follow-up approximately 7 years later. The annual declines in measured FEV_1 were related to increasing age, to being a smoker at the time of the initial assessment and also interestingly to work as a welder or caulker/burner, in comparison to work that did not involve welding or burning. Interestingly, there was evidence of a significant interaction between the effects of smoking and occupational exposures. Specifically, the amount of variance in lung-function decline explained by work exposure was on average half that due to smoking (except when atopy was included in the analysis). There were also significant interactions between these two effects and between them and the effects of age.

Whilst longitudinal studies may seem ideal as methods for assessing such risk of COPD in workplaces, they also have significant problems. Such studies are often costly, time consuming and inevitably have difficulties maintaining appropriate levels of follow up within an established cohort. Over time, workplaces tend to concentrate on fitter workers, whereas sicker workers (including presumably workers with respiratory disease) tend to leave; the so-called 'healthy worker effect.' Whilst some of these effects can be controlled for, these complex issues often make interpretation of such studies more problematic.

Population approach

Various population-based approaches have therefore been adopted to estimate what proportion of COPD

relates to workplace exposures. The most straightforward estimate of the size of this problem would be to express the number of work-related cases of COPD as a fraction of the total number of cases in any given population. However, as already discussed, health care workers have very little useful information at their fingertips to define when a case of COPD is or is not (even predominantly or partially) work related, even if an occupational history is documented carefully. This is because although patients with COPD may report occupational exposures, most have also smoked cigarettes, and may have other risk factors, thus making it very difficult, or a matter of opinion, in the individual to decide what is the predominant cause. Counting clinical cases of occupational COPD is therefore not reliable, and as a consequence generating the fraction of all COPD due to work by this method is not possible.

Another possible approach to defining the size of this problem in the context of all COPD would be to use data generated from national reporting schemes for occupational lung disease. The UK based scheme SWORD[24] (Surveillance of Work related and Occupational Respiratory Disease) regularly receives notification of cases of occupational lung diseases from respiratory and occupational physicians. However, cases of occupational COPD reported to this scheme are relatively uncommon in comparison to other diseases reported,[25] and the accuracy of such reporting will also inevitably be affected by the lack of good clinical case definitions of work related COPD. Whilst it is hoped that the relative absence of such reporting seen through such schemes reflects a true absence of such cases, it is equally likely that reporting schemes significantly underestimate the size of this problem, as accurate case identification is problematic.

Consequently, as clinical case-based definitions cannot be relied upon to accurately measure the size of the 'occupational COPD effect,' alternative estimates are used. These are derived normally by using one of two main study designs; first, using workplace-based studies measuring dose of inhaled agent, and the levels of current diagnosed COPD, or second, using population-based estimates. The principle behind the latter is that an estimate can be made of the fraction of COPD that can be reasonably attributed to occupational exposures, termed the population attributable risk (PAR). This can be calculated by knowing the numbers of individuals in any chosen population with and without COPD and the respective levels of workplace exposures within each of these groups. Whilst it is possible to stratify in detail differing levels of exposure, previous studies (due to power constraints) have normally concentrated on whether workers have 'ever' been exposed to general VGDF.

In 2003, the American Thoracic Society (ATS) published an official statement relating to occupation as a cause of airways disease, focussing particularly on asthma and COPD.[26] This statement recognized that whilst traditional occupational lung diseases, such as certain pneumoconioses, were decreasing in prevalence, the issue of airways diseases relating to occupation still required vigilance. Available evidence from two main types of studies was assessed.

First, examples of cross sectional and longitudinal data were summarized from a variety of workplaces. These data were pooled, although not subjected to further post hoc analysis. Second, estimates of PAR for COPD and workplace exposures were compared from multiple population-based studies. In this group, if estimates of PAR had not been calculated by the authors, one of two post hoc analyses was applied to the data in order to derive a median or 'average' PAR estimate.

In this way, various estimates of the size of the contribution of occupation to COPD were considered in the ATS document, based around differing definitions used for the condition in the various studies considered. For example, some studies used only the presence of certain symptoms consistent with COPD as a working definition, whilst others used both symptoms and measures of lung function to define a case. The consensus document concluded that approximately 15% of the total burden of both adult onset asthma and COPD were reasonably attributable to the effects of workplace exposures.

Further data to support these findings have been published since the completion of the ATS statement. Trupin et al.[27] reported their data derived from 2061 US residents randomly selected to complete a telephone-based questionnaire. COPD in this instance was defined by a 'physician diagnosis,' reported by the respondent and used definitions both including and specifically excluding chronic bronchitis. The authors calculated a PAR for self-reported occupational exposures of 20%, with a 95% confidence interval between 13% and 27%. Values of PAR were lower using various exposure estimates derived from a job exposure matrix, but all broadly in line with the estimates derived for the ATS statement.

Similarly, Hnizdo et al.[28] also recently reported the further analysis of the US based NHANES (National

Health and Nutrition Examination Health Survey) data, and derived a similar conclusion, finding the fraction of COPD attributable to work was estimated as 19.2% overall and 31.1% among never smokers.

In 2007, Blanc and Torén[29] published a review of all studies since the ATS statement that included the PAR (or allowed it to be calculated from the data). This reviewed 14 studies (including the two quoted above) and represented over 400,000 individuals. Again the median PAR% was estimated to be 15.

The same study team, and others, have also assessed these issues on a more global scale, analysing some of the data from the Burden of Obstructive Lung Disease (BOLD) study, the Latin American Project for the Investigation of Obstructive Lung Disease (PLATINO) and the European Community Respiratory Health Survey follow-up (ECRHS II). These data sets in total represented approximately 20,000 individuals. This analysis identified a 0.8% increase in COPD prevalence per 10% increase in occupational exposure prevalence.[30]

Certain epidemiological evidence also supports a possible interactive rather than additive effect occupation and smoking upon COPD causation. In the paper by Trupin,[25] looking at those who self-reported a physician diagnosis of COPD and emphysema, the adjusted odds ratio (OR) for developing COPD was set to 1.0 in never smokers with no self-reported VGDF exposure; by comparison, this rose to 2.4 in those with occupational exposure to VGDF alone, 7.0 in ever smokers alone and 18.4 in those who had both VGDF exposure and had ever smoked. The authors concluded that this demonstrated evidence of an interaction between smoking and exposure to VGDF. In other words, being exposed to VGDF and smoking harm the lung more than would be expected for the addition of each effect separately. This finding has clear implications for advice about smoking cessation to workers who may be continually exposed to VGDF in workplaces.

A similar finding was seen in the FLOW study,[31] based on 2310 interviews with cases of physician-diagnosed COPD, 1202 of whom also completed research clinic visits with spirometry. An excess (OR 14.1 in comparison to never smoking never VGDF exposed workers) and multiplicative risk for the presence of COPD was again identified for the combination of VGDF exposure and smoking.

How do these epidemiological findings apply to the real life situation? Clearly, workplace exposures do not cause 15% of all cases of COPD (approximating to the ATS 15% figure for PAR) but are thought to be responsible for 15% of the total 'burden' of COPD in a given population. Perhaps this is best appreciated by understanding that multiple risk factors are associated with the development of COPD, with cigarette smoking being most prominent. Intervening to stop all harmful VGDF exposure in workplaces now would in time reduce cases of COPD by 15%.

To date, attempts to identify more specifically individual agents that cause COPD using these population-based epidemiological studies have not been particularly useful. This may be due in part to the fact that when workers are grouped into differing exposure types, numbers within each subgroup become smaller, and consequently more difficult to analyse.

Interestingly, whilst certain evidence already exists to support a relationship between exposure to environmental tobacco smoke (ETS) and the development of lung cancer,[32,33] a recent report from the EPIC study[34] also supplies evidence to support the role of ETS in the development of other respiratory diseases.

Estimates of the size of the occupational burden of COPD also allow costs of the COPD 'burden' to be estimated. In terms of costs to individual countries, Tinkelman et al. have recently assessed how countries estimate COPD disability within their pension schemes, and therefore estimate overall costs to their economies.[35] Whilst the derived figures merely serve as estimates, it was concluded that the total cost of COPD in the eight chosen countries (Canada, France, Germany, Italy, Japan, Spain, the United Kingdom and the United States) lay between approximately US$5 billion and US$25 billion per year. The economic argument alone therefore appears reason enough to attempt to solve the various components of this problem, including the proportion of COPD attributable to occupational exposures.

COPD and its effect on work

COPD is now recognized not only to be caused in part by workplace exposures but also to cause significant disruption to working lives of those affected. Eisner[36] used data derived from the Californian Work & Health survey to explore this problem, within which 3805 working-age adults were studied, of whom 172 (4.5%) were categorized as having COPD according to responses during a telephone questionnaire. The advantage of such an approach was inclusion in the study of a range of COPD severity, particularly as clinically based studies tend to concentrate more

severely affected individuals. The diagnosis of respiratory illness was based on questionnaire responses alone as no measures of lung function were made. Health and working statistics were recorded in addition to other relevant demographic factors. As expected, individuals nominating a diagnosis of COPD were significantly disadvantaged by a reduction in general physical health status and depressive symptoms, although appeared to retain certain social functions, such as frequent telephone contact with friends.

With regard to employment status, however, individuals with COPD faired worse for current employment (46.5%), contrasting with individuals reporting a diagnosis of asthma, who did not report a deleterious effect on employment. More specifically, the presence of COPD was associated with prolonged non-participation in work (OR 2.92 for more than 5 years since regular employment) and clear problems with perceiving limitation in their own capacity to work. Whilst there was a suggestion that duration of work was lower than those with 'no chronic condition,' these effects were not significant. Wang[37] has also studied the effects of various chronic medical conditions on work performance, using a WHO workplace questionnaire. COPD was again found to have significant effects on absenteeism.

Tinkelman[38] reinforced these findings in a retrospective study of over 2000 established cases of COPD, approximately half of which were of working age. Perhaps contrary to the typical perception of this disease, this group missed a mean of 4.6 days in the 6 months prior to the study analysis. Comment was made that the typical stereotype of a COPD patient (elderly, unemployed) may not be applicable to a significant proportion of those already with such a diagnosis.

Halpern[39] progressed this idea to study not only the short-term sickness absence associated with exacerbations of chronic bronchitis but also estimated the impact on productivity in the workplace. This literature review not only identified that patients with chronic bronchitis had more days off work but also that appropriate treatment of these exacerbations led to a reduction in further exacerbations and comparatively less work loss. It was concluded that clinical outcomes and workplace costs are related. While this relationship was identified to be clearer in terms of work loss, further exploration was suggested to assess decreased productivity and to evaluate this relationship using objective indicators of absenteeism and productivity, rather than relying on recall from patients alone.

A Dutch study subsequently explored this issue further, identifying that patients' (and in certain cases, workers') knowledge of their COPD improved adaptation to the condition, but paradoxically reduced their ability to control shortness of breath at work. This finding is clearly worthy of further investigation, although suggests at least in part that being in possession of more information about the origins of breathlessness may interfere with coping with this symptoms at work.[40] The same group also showed a significant decrement in quality of life for COPD patients disabled from work, in comparison to those in work, for a similar degree of airways obstruction.[41]

Adverse COPD outcomes

Having established that COPD is in part potentially caused by occupational exposures to VGDF and that workers with COPD suffer personal, financial and work disability along with social costs, recent data also suggests that COPD associated with work exposure may carry with it an adverse prognosis in comparison to COPD unrelated to work, for a given level of airflow obstruction. Recent data from San Francisco helps to clarify this.[42] A total of 234 patients with COPD were included in a study to assess various aspects of COPD including quality of life, activity limitation and the potential relationship between these factors and the workplace. This group was a sub-population of a much larger, US based, random digit dial epidemiological study. Of these, more than half (55%) reported exposure to VGDF during their longest-held job, and a quarter of all COPD patients reported 'respiratory related work disability,' defined as not working at the time of the interview due, at least in part, to a 'lung or breathing condition.' Furthermore, this study showed that combined exposure to harmful workplace agents and 'respiratory related work disability' was associated with the greatest risk at follow up of frequent restricted activity days. This restriction was attributed to many factors including 'a breathing or lung condition' (OR 3.8; 95% CI 1.4 to 10.1), emergency department visit (OR 3.9; 95% CI 1.4 to 10.5) and hospitalization (OR 7.6; 95% CI 1.8 to 32).

How to identify COPD at work

Workplaces are ideal arenas within which to focus on lung health and to potentially identify workers with

abnormal lung function suggestive of COPD. This is a complex area, and although it is not within the scope of this article to deal with this issue in detail, the identification of COPD in cross-sectional studies of lung function and the identification of workers with accelerated annual decline in FEV_1 will be briefly discussed and have both been the subject of considerable recent debate.[43,44]

Cross-sectional measures of lung function (such as those taken as part of health surveillance programmes in workplaces) may identify workers with established COPD, by comparing measured values from workers with a predicted value, derived from a set of predictive equations. Those found to have a reduced ratio between the FEV_1 and the FVC (below 70%) by definition have evidence of airways obstruction and may have early or more advanced COPD. Reference to, for example, the GOLD guidance[45] will help identify the level of COPD severity.

However, it is more difficult to interpret other situations such as low normal values of lung function or lung function in workers heavily exposed to VGDF. In these situations, repeated measures of lung function over time may assist the interpretation of individual worker's lung function. As a consequence, workplaces that wish to identify workers with an accelerated level of FEV_1 decline over time are required to make serial and accurate measures over a relatively long period of time.

Measuring longer-term measures of lung function in workers raises certain issues of accuracy and interpretation that do not apply to the individual, or 'one off' measure. For example, changes year on year in the FEV_1 of an individual worker may not just reflect true or actual change in FEV_1 but may also reflect changes due to the inaccuracy of the spirometer or inaccuracies of measurement caused by poor measurement technique. For example, as ATS guidance stipulates that spirometers are required to be accurate to $\pm 3\%$, relatively large changes in FEV_1 over a year are required before this is thought to be clinically significant, and in excess of what might be expected from the variation of the reading due to the spirometer alone.

As a consequence, the ATS suggests that a 15% reduction in FEV_1 over 1 year is the minimum fall that should arouse suspicion of a clinical problem.[46] A 300-500 mL drop is also considered a significant fall over 1 year (depending on the accuracy of the spirometry programme in the workplace), or 100 mL a year for 5 sequential years.

It may be difficult to justify, however, waiting for many years observing decline in lung function in certain workers. A decision to intervene in the workplace and referral a worker for further assessment must be based on all relevant factors, not only lung function change.

To address the issue of spirometry accuracy, Hnizdo et al.[47] performed a study using data from four different workplace-monitoring programmes, measuring data precision using a pair-wise within-person variation statistic. They concluded that a 10% fall in FEV_1 over one year in a worker within a good quality workplace-monitoring programme should be of concern and suggest intervention and further evaluation, whereas a fall of about 15% appeared appropriate for clinical evaluation of those with COPD or asthma.

Harber et al.[48] also recently evaluated 5724 people in the Lung Health Study, a multicentre study addressing smoking cessation and anticholinergic bronchodilator administration in smokers with early COPD. Participants had a baseline evaluation followed by five annual assessments, which included questionnaires and spirometry. The effect of ongoing occupational exposure to dust or fumes on FEV_1 was studied. In males with early COPD, each year of continued occupational exposure was associated with a 0.25% reduction in post-bronchodilator predicted FEV_1%. Whilst this was smaller than the effect seen in ongoing smokers (1.2%-1.9%), the effect was significant. The authors concluded that there is a need for secondary prevention of COPD by controlling occupational fume exposures.

Future approaches and legislation

As population smoking rates are likely to fall further with time, other causative factors associated with COPD will become proportionally more important. As occupational exposures to VGDF have been identified as a significant risk factor for the development of this condition, there is much scope for future research to define how best to intervene in the workplace to reduce the subsequent risk of this condition. This research by necessity must include epidemiological, clinical and exposure measurement approaches, using a combination of quantitative and qualitative techniques.

Modern workplaces are ideal environments to study the effects of airborne exposure and lung response as many workforces are relatively stable, easily defined, well characterized and traceable.

Measures of complex current exposure levels can also be made. Newer techniques (such as mathematical modelling and more accurate exposure characterization) may help us better understand the process of developing COPD in the occupational context and provide a more sound evidence base to guide future public health policy and legislation for the workplace.

In summary, there is a substantial evidence base to support a significant workplace contribution to the causation of COPD. The size of this 'occupational effect' on COPD is currently thought to account for 15% of the total disease burden. The harmful effects of workplace exposures to VGDF are thought to interact with cigarette smoking to produce lung harm, although the relative effects will vary from person to person.

Multiple interventions are likely to be needed to address these issues. It seems reasonable that at the individual COPD patient level, simple advice concerning 'dirty' workplaces, and the need to reduce harmful exposures, should be included along with smoking cessation advice. At the workplace level, there are opportunities to develop and improve health surveillance strategies to identify early the workers who are developing respiratory problems.

At the societal level, it is increasingly realized that workers with COPD can suffer both financially and socially as a result of lost work, and these problems may be compounded by future changes in population age. The recent health, work and well-being agenda[49] highlights many issues pertinent to COPD, including the need to ensure that workplaces are able to allow workers with COPD to continue to work efficiently, and to devise approaches to improve well-being and to prevent prolonged sickness absence. This new way of thinking offers further specific research opportunities.

The power of all health care professionals to influence this process should never be underestimated. All cases of COPD should be assessed with these occupational issues in mind. Think COPD, think work!

Acknowledgement

The authors thank Ann Collins for help and advice on manuscript preparation.

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