



Department for Work and Pensions

Department for Work and Pensions Social
Security Administration Act 1992

Chronic Obstructive Pulmonary Disease (COPD) – Chronic Bronchitis and Emphysema

**Report by the Industrial Injuries Advisory Council
in accordance with Section 171 of the Social Security
Administration Act 1992 reviewing the prescription
of chronic obstructive pulmonary disease
(COPD) – chronic bronchitis and emphysema.**

Presented to Parliament by the Secretary of State for Work and Pensions

By Command of Her Majesty

November 2007

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INDUSTRIAL INJURIES ADVISORY COUNCIL

Secretary of State for Work and Pensions

Dear Secretary of State,

REVIEW OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD) – CHRONIC BRONCHITIS AND EMPHYSEMA

In 2003, we announced that the Industrial Injuries Advisory Council (IIAC) would be conducting a review of occupational coverage for chronic bronchitis and emphysema. The decision to review these diseases arose as a consequence of representations by delegates at IIAC's first public meeting in Sheffield in March 2003 to consider prescription for surface coal workers. The Council had last reviewed occupational coverage for chronic bronchitis and emphysema in May 1996 (Cm. 3240). After an initial review of the literature we felt it was an opportune time to undertake a formal review of the available evidence.

Chronic bronchitis and emphysema are now usually considered as causes of Chronic Obstructive Pulmonary Disease (COPD). COPD is an umbrella term which includes a number of diseases which have in common obstruction to airflow in the bronchial airways, which is predominantly irreversible. In addition to chronic bronchitis and emphysema it includes other respiratory conditions, such as chronic severe asthma. This report only reviews COPD caused by chronic bronchitis and emphysema.

In considering the prescription of COPD the Council has focused on:

- (1) The level of lung function loss – measured as forced expiratory volume in one second (FEV₁) – which is associated with important respiratory disability.
- (2) Evidence for exposures or occupations where the risk of this level of lung function loss was at least twice as frequent as in the general population.
- (3) Whether the circumstances of exposure associated with this level of lung function loss could be readily defined.

There is a considerable scientific literature which addresses the question of whether exposures experienced at work can cause COPD. To assist the Council in its evaluation of this literature we commissioned two reports, the first on COPD and occupation, the second on COPD and silica. These reports identified a number of exposures at work where the evidence was sufficiently suggestive for the Council to undertake further investigation. These included surface coal work, welding, cotton textile manufacture and exposure to grain dust and silica. One of our members also requested consideration be given for workers exposed to isocyanates. For none of these however, did the scientific literature provide evidence for a doubling of risk of an FEV₁ loss of 1 litre (L) in those working in well defined circumstances of exposure. We therefore met with a number of the authors of relevant studies and where appropriate commissioned further analysis of their findings, appropriate for our purpose.

However, the results of surveys conducted by the Institute of Occupational Medicine in Edinburgh indicated that the levels of dust to which surface coal workers on the screens were exposed were sufficient to double the risk of disabling loss of lung function if experienced for a 40 year working life.

On the basis of our extensive review we can confirm the current prescription of chronic bronchitis and emphysema (COPD) in underground coal miners, in workers exposed to cadmium fume and in cases of byssinosis.

We recommend that the prescription for Prescribed Disease (PD) D12 be extended to exposure to coal dust in screen workers employed at the surface of coal mines in those employed for 40 years for exposures occurring before 1983. A number of workers are likely to have transferred from underground coal work to less dusty surface screen work. For those workers, we recommend that time spent underground and at the surface as a screen worker be aggregated such that 2 years of surface work would be equivalent to 1 year of underground work.

For the other occupations which were considered in detail, including textile workers without a history of byssinosis, workers exposed to silica dust and welders, there was a lack of evidence of sufficient lung function loss or exposure data, or both, to recommend prescription or amendments to prescription. We recommend research is conducted to address these questions. We will continue to monitor emerging evidence with a view to reconsidering prescription for other occupations in the future.

Yours sincerely,

Professor A J Newman Taylor

Chairman

21 November 2007

Summary

1. Chronic bronchitis and emphysema are common diseases in the general population and are now encompassed by the term Chronic Obstructive Pulmonary Disease (COPD). The predominant cause of COPD in the general population is cigarette smoking.
2. The Industrial Injuries Advisory Council (IIAC) has completed its review of occupational coverage for the prescribed disease (PD) chronic bronchitis and emphysema (PD D12). Following reviews of the literature, consultation with experts in several areas, consideration of reviews commissioned by independent academic bodies and public consultation the Council has made the following recommendations.
3. The Council recommends that the current terms of prescription remain appropriate for chronic bronchitis and emphysema in underground coal workers (PD D12), for byssinosis in cotton workers (PD D2) and for emphysema in workers exposed to cadmium fume (PD C13). The Council further recommends that PD D12 (chronic bronchitis and emphysema) be extended to include exposure to coal dust in screen workers employed at the surface of coal mines. Workers should have been employed for the equivalent of 40 years and qualifying exposures should have occurred before 1983.
4. As a number of workers transferred from underground coal work to less dusty surface screen work for various reasons, which include respiratory illness, the Council recommends that time spent underground can be aggregated with time spent at the surface as a screen worker, such that 2 years of surface work as a screen worker before 1983 is equivalent to 1 year working underground in a coal mine.
5. While this extension to PD D12 will now benefit a small number of potential claimants who have spent their working life as surface coal workers, it also extends the prescription to those who have worked less than 20 years underground by allowing aggregation of their time underground with their time as a surface coal worker.
6. IIAC also considered prescription for other occupational groups including cotton textile workers, welders, grain workers and workers exposed to silica or isocyanates. There was a lack of suitable evidence to recommend prescription or amendment to prescription for any other occupational categories. However, IIAC will continue to monitor emerging evidence and requests that further research of a level specified in paragraph 80 of this report be undertaken to investigate the relationship of lung function decrements in COPD to levels of exposure from specific agents in these and other occupational categories.

INTRODUCTION

The Lungs

7. The lungs can be considered as two adjoining compartments: the conducting airways; and the alveoli, which are the site of gas exchange. During inspiration, air inhaled through the nose or mouth is conducted through the branching bronchial airways (the larger bronchi and smaller more peripheral bronchioles) to the alveoli. In expiration, air flows from the alveoli through the conducting airways to the nose or mouth. Alveoli are blind ending sacs in the respiratory zone of the lungs, which allow oxygen to diffuse from the air into the blood and carbon dioxide to diffuse from the blood into the air. A diagram of the lungs can be seen in Figure 1.

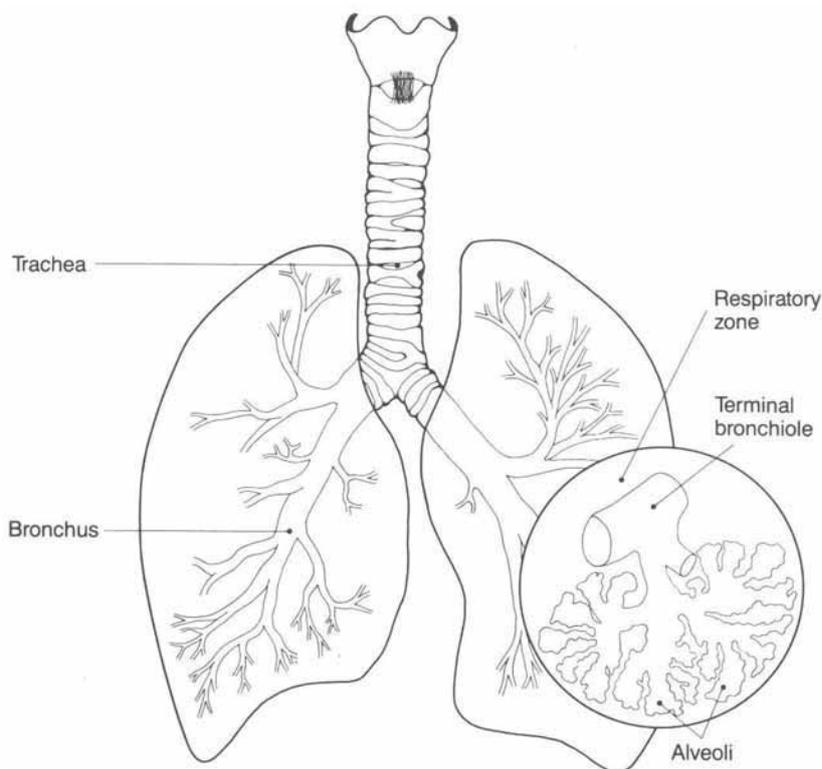


Figure1. Diagram of the lungs showing the airways and respiratory zone. Air passes through the trachea, bronchi and bronchioles (too small to be represented in the diagram) to alveoli which arise from terminal bronchioles. The exchange of oxygen and carbon dioxide occurs in the alveoli. (Diagram taken from Cotes and Steel, 'Work related lung disorders' and reproduced with kind permission of Blackwell Scientific Publications Ltd.)

Chronic bronchitis and emphysema – the disease

8. Chronic obstructive pulmonary disease (COPD) is an umbrella term for a number of respiratory diseases which include chronic bronchitis and emphysema as well as chronic severe asthma. This report is concerned only with the chronic bronchitis and emphysema component of COPD. The British Thoracic Society has defined COPD as a slowly progressive disorder, characterised by airways obstruction (reduced FEV_1 and FEV_1/FVC ratio – see paragraph 12), which does not change markedly over several months and where most of the impairment of lung function is irreversible.

9. Bronchitis is inflammation of the mucosal lining of the bronchial tubes which causes excessive mucus secretion and sputum production. Chronic bronchitis is commonly defined as a cough productive of sputum on most days during at least three consecutive months, for not less than two consecutive years. The inflammation with over-secretion of mucus in the bronchi is often a response to inhaled irritant particles such as cigarette smoke, coal dust or fumes. Irritant particles can also cause an obstructive bronchiolitis with permanent narrowing of the bronchi and bronchioles and a consequent reduction in the rate at which air can be exhaled from the lungs.

10. Emphysema is a pathological change in lung structure characterised by destruction with dilatation of the alveolar walls and the elastic tissues supporting the structure of the lungs. This destruction results in an overall loss in the alveolar surface area where gas exchange takes place, reducing the uptake of oxygen from inspired air. The accompanying loss of elasticity of the surrounding tissue results in airway narrowing, with obstruction to airflow, due to premature closure of the bronchioles when breathing out. This can lead to severely disabling breathlessness.

11. Chronic bronchitis and emphysema often occur together. Both conditions impair the function of the lungs by limiting airflow particularly during expiration. In addition, emphysema reduces the ability of the lungs to take up oxygen from inspired air. COPD as the outcome of chronic bronchitis and emphysema can cause severe respiratory disability, as a consequence of the airways narrowing and alveolar wall destruction, and shorten life. Tobacco smoking is the main risk factor for developing these conditions whose effects are predominantly irreversible. Other risk factors include dusts, gases, vapours and fumes inhaled at work. The American Thoracic Society has estimated that the proportion of COPD in the population attributable to occupational factors is 15%.

Lung function tests

12. Lung function is most commonly assessed by measuring the volume of air that can be exhaled in a specific time period. The forced vital capacity (FVC) is the total volume of air (often expressed in litres) that can be forcibly exhaled after a maximal inspiration. The volume of air exhaled in the first second of a maximal forced expiratory manoeuvre is the forced expiratory volume in one second (FEV_1). FEV_1 is dependent upon an individual's sex, age and stature and is normally some two-thirds to three-quarters of the FVC. This ratio can be substantially reduced by chronic airflow limitation in COPD. Figure 2 shows an example of FEV_1 and FVC recorded by a spirometer in a normal individual and in an individual with airflow limitation. Whereas airflow limitation is predominantly reversible in asthma, in COPD it is predominantly irreversible.

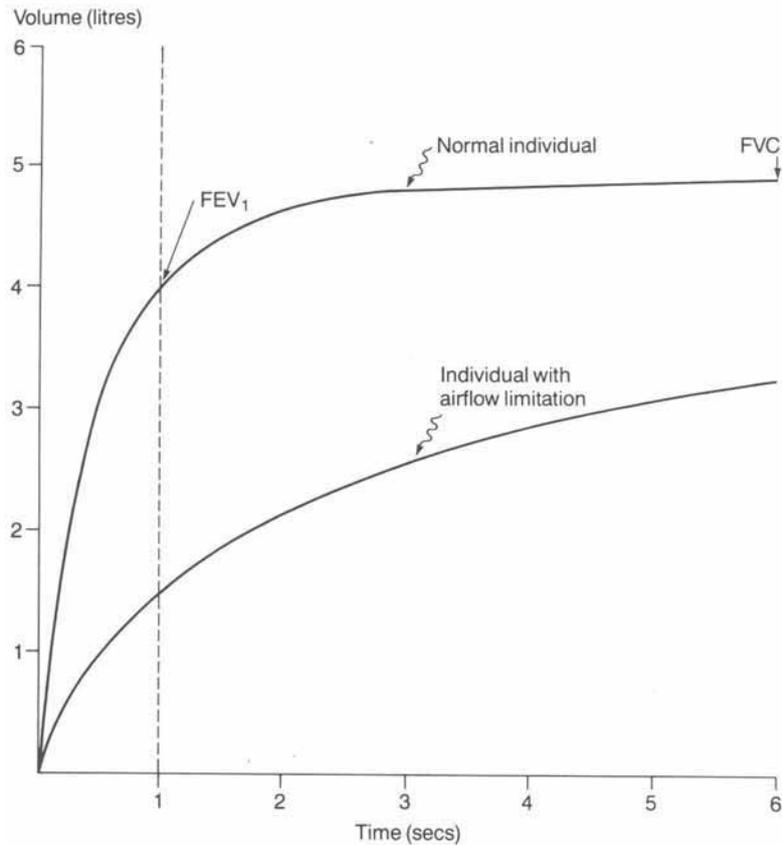


Figure 2. Forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) in a normal individual and an individual with airflow limitation. The normal individual expels between $\frac{2}{3}$ and $\frac{3}{4}$ of his FVC in the first second (i.e. the FEV_1/FVC ratio is between $\frac{2}{3}$ and $\frac{3}{4}$). The individual with airflow limitation takes longer to expel the air from their lungs; FEV_1 is reduced and FEV_1/FVC ratio is less than $\frac{1}{2}$.

13. FEV_1 and FVC are measures of lung volume which increase during childhood and adolescence, reach a peak and plateau during the third decade of life and subsequently slowly decline during adult life, because of the gradual loss of lung elasticity with age. The rate of decline of FEV_1 is accelerated in so-called “susceptible” smokers by some 3-fold from about 30ml per annum to about 90ml per annum. This increase in the rate of FEV_1 decline (about 60ml p.a.) can cause the loss of an excess of more than 1 litre (L) (some 1200ml) in lung function as compared to normal over about 20 years. These effects are illustrated in Figure 3. By inference, a similarly increased rate of decline of FEV_1 also occurs in those exposed in the course of their work to a sufficient concentration of cadmium fume or coal dust. FEV_1 in those who have experienced sufficient exposure to these agents over 20 years will be 1L or more less than would be otherwise expected.

14. FEV_1 in the population is determined primarily by age, sex and height. Having taken these factors into account FEV_1 is distributed normally (in statistical terms) around an average (or “predicted”) value with one standard deviation of some 0.5L in men and some 0.45L in women. Figure 4 shows a normal distribution for FEV_1 . Men who are more than 2 standard deviations below the average FEV_1 for their age, sex and height will therefore have an FEV_1 of 1L or more below their

average (“predicted” value). While this will include the lower 2.5% of the “normal” distribution, it will also include those whose FEV₁ has been reduced by disease e.g. COPD caused by tobacco smoke or occupational exposures or both. Figure 5 illustrates the effect of smoking in a population whose FEV₁ previously had a “normal” distribution. Similar effects occur with some occupational exposures, such as underground coal mining and inhalation of cadmium fume, and could potentially arise from the exposures considered in this report.

Criteria for prescription

15. COPD (chronic bronchitis and emphysema) attributable to occupational cause does not have unique clinical features. The diagnosis alone does not, therefore, allow occupational attribution. In this situation, IIAC looks to the scientific literature to examine whether there is evidence which shows that those in a particular occupation or exposed to a specific agent are twice or more as likely to develop the disease as compared with the general population (see Appendix 1).

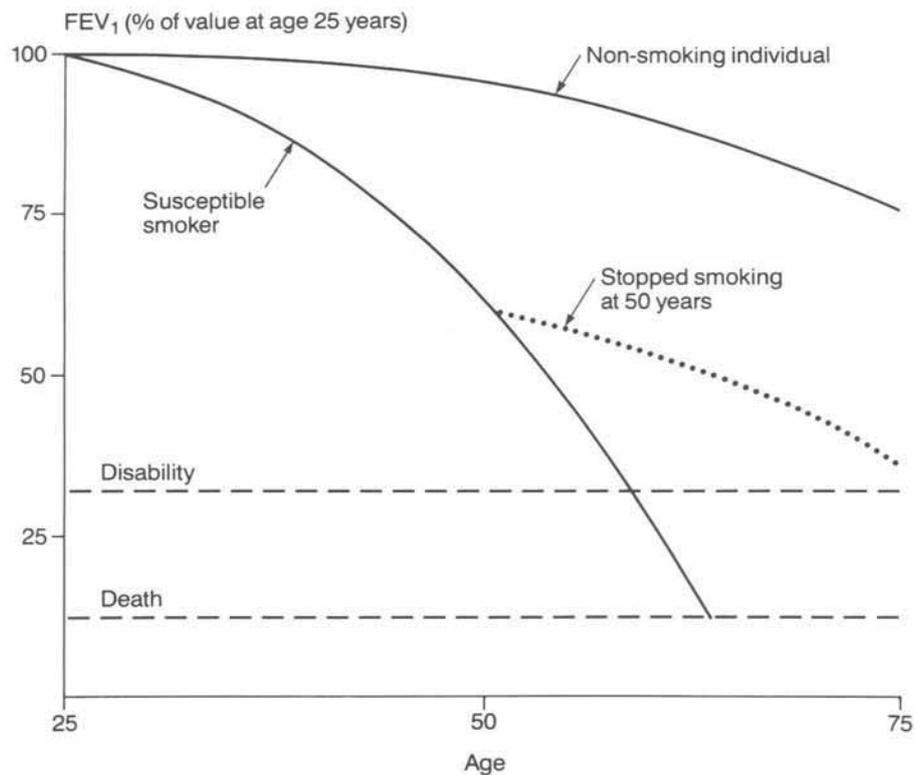


Figure 3. Diagrammatic representation of FEV₁ decline with age in non-smokers and “susceptible” smokers. FEV₁ declines from the age of about 25 years at a rate which, in a normal individual, does not lead to disability during a usual lifespan. The accelerated rate of decline in FEV₁ in the “susceptible” smokers is sufficient for this to cause disability in later life and premature death. If the susceptible smoker stops smoking sufficiently early in life (shown here at age 50 years) lost lung function is not regained, but the rate of decline returns to normal and disability later in life will be avoided.

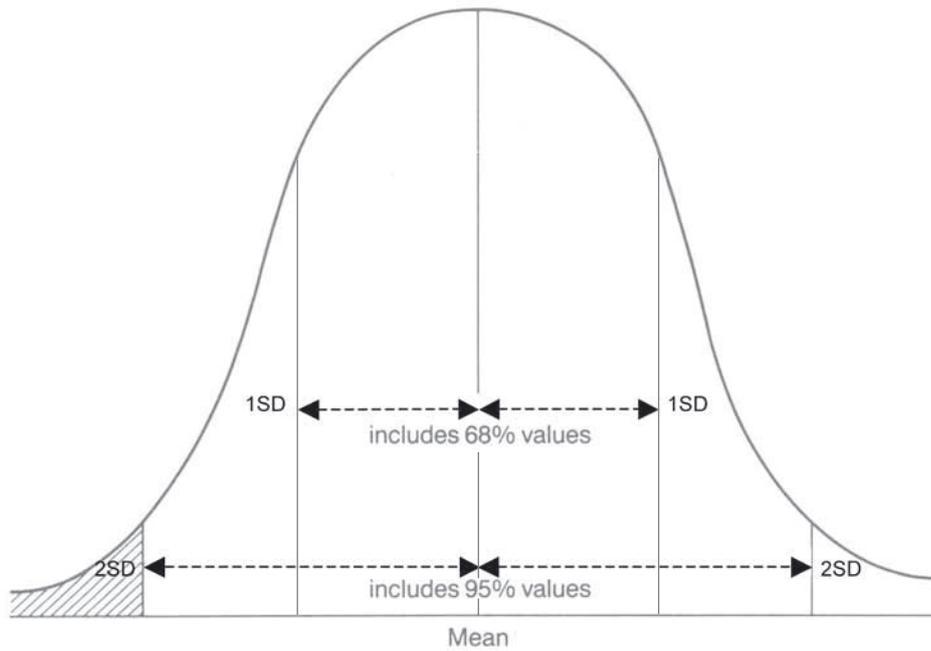


Figure 4. This illustrates the important characteristics of a “normal” distribution: the values are spread symmetrically around the mean value; 1 standard deviation above and below the mean value includes 68% of the values and 2 standard deviations above and below the mean value include 95% of the values.

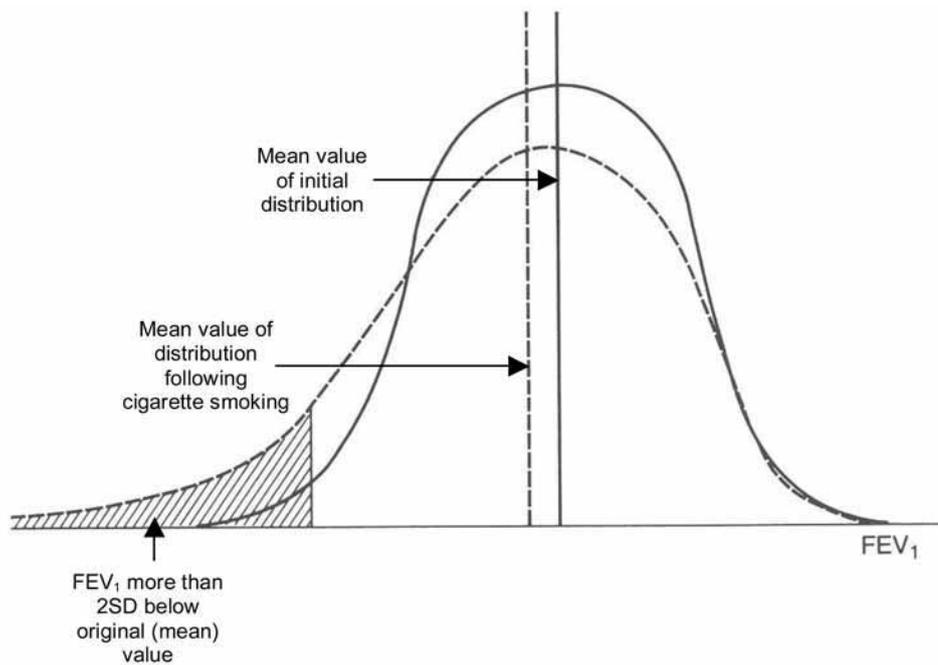


Figure 5. This demonstrates the consequences of the variable effect of cigarette smoking on a population whose lung function previously had a normal distribution of FEV₁. The average (mean) effect is relatively small but the effect on the “susceptible” minority is marked and a considerable number of persons will have values of FEV₁ more than 2 standard deviations below their anticipated (from the initial distribution) value.

16. COPD is characterised by airways obstruction of increasing severity with loss of FEV₁. In considering the prescription of COPD, IAC considered:

- (1) The level of FEV₁ loss associated with important respiratory disability;
- (2) Whether there were occupations or exposures at work where this level of FEV₁ loss was at least twice as frequent as in the general population; and
- (3) Whether the circumstances of exposure associated with this level of FEV₁ loss could be readily defined.

Historical background to the prescription of chronic bronchitis and emphysema

17. Prescription for chronic bronchitis and emphysema has had a long and complex history, due to difficulties in prescribing a disease which is common in the general population with a single dominant cause, cigarette smoking, and thus in distinguishing occupational from other non-occupational causes of the disease. IAC first considered the prescription of chronic bronchitis and emphysema in its review 'Pneumoconiosis and Byssinosis' (Cmnd. 5443), published in 1973. At that time, there was insufficient evidence to support prescription for chronic bronchitis and emphysema independently from pneumoconiosis but the Council agreed it would monitor emerging research.

18. The Council reconsidered the evidence in its 1988 report, 'Bronchitis and Emphysema' (Cm. 379). Despite new evidence published since the previous review, there remained significant barriers to prescription and IAC was unable to recommend that chronic bronchitis and emphysema be compensated under the Industrial Injuries scheme. The Council concluded that coal dust did adversely affect lung function in exposed workers independently of pneumoconiosis but there was a lack of evidence on the magnitude of the effect on lung function, in both smokers and non-smokers, in relation to the levels of exposure to coal dust in underground coal mines. IAC called for additional epidemiological research to be conducted to address these questions.

19. Following IAC's request, research was published which provided the necessary evidence to enable the prescription of chronic bronchitis and emphysema in the Command paper 'Chronic bronchitis and emphysema' (Cm. 2091), published in 1992. Studies showed that the risk of a reduction in FEV₁ to <65% predicted (to about 1L less than the predicted value) was more than doubled in underground coal miners, both smokers and non smokers, who had experienced a cumulative exposure equivalent on average to working underground for 20 years in UK mines in 1960's and 1970's. A level of FEV₁ of 1L below the predicted value was on average associated with shortness of breath when walking with others on the level, i.e. a clinically significant level of respiratory disability.

20. Taking this evidence together, the Council recommended the prescription of chronic bronchitis and emphysema in miners whose FEV₁ was 1L or more below their predicted value and who had worked underground in a coal mine for 20 years or more. The time requirement was set as the evidence indicated that it would take 20 years of underground coal work at 3-6 mg m⁻³ in order to accumulate the 60-120 mg m⁻³ yr⁻¹ exposure to coal dust necessary to double the risk of a disabling loss of lung function. Prescription of chronic bronchitis and emphysema in underground coal miners was based on evidence of: 1) an FEV₁ of at least 1L below the predicted value for a person of similar age, height and sex, based on the Cotes formula; 2) Evidence of dust retention in the lungs which was interpreted from nodules seen on a chest radiograph (Category 1 pneumoconiosis); 3) 20 years work underground in a coal mine.

21. The Council reviewed the terms of prescription for chronic bronchitis and emphysema again in 1996 in 'Chronic Bronchitis and Emphysema' (Cm. 3240). This review focussed on the diagnostic criteria, in particular the FEV₁ test. Representations were made to the Council that the terms of prescription were disadvantageous to shorter or older claimants, since they have a smaller lung volume than taller or younger people, irrespective of health status. After considering the evidence, the Council recommended that the measurement of lung impairment should still be made by using the test of FEV₁ as compared to the predicted values for those of similar age, sex and height. However, to remove any unfairness of the test to older, shorter men who may have smaller predicted values for FEV₁, any person presenting with an absolute FEV₁ measurement of 1L or below would fulfil the medical criterion of the prescription. The Council also recommended that the requirements for a chest radiograph should be discontinued as new evidence indicated that mortality from chronic bronchitis and emphysema in coal miners was not correlated with mortality from pneumoconiosis.

22. In 2000 the Council published its position paper 11 'Lung Function Assessment, Industrial Injuries Disablement Benefit, Prescribed Disease D12 (Chronic Bronchitis and Emphysema in Underground Coal workers)' laying out the scientific basis for the use of the Cotes formula for calculating the predicted value for lung function for claimants. The Cotes formula was based on robust studies of healthy, working age subjects, including some smokers. Other formulae, such as the European Community for Coal and Steel (ECCS) formula, were based upon amalgamations of various studies of different designs, from various European countries, which might not be relevant to the British workforce. The Council concluded that the Cotes formula remained the most appropriate calculation for predicting lung function for the purposes of the prescribed diseases

23. In its report 'Conditions due to Chemical Agents' (Cm. 5395, 2002), the Council clarified the terms of prescription for exposure to cadmium fumes. Evidence indicated that there was an excess of emphysema in workers exposed to prolonged or heavy exposure (> 20 years) to cadmium fumes. IAC recommended that the terms of prescription for PD C18 be amended to prescribe emphysema for exposure to cadmium fumes for twenty years or more.

Current terms of prescription

24. The current terms of prescription for chronic bronchitis and emphysema (PD D12) are described in Appendix 2. Emphysema is prescribed for workers exposed to cadmium fumes (PD C18). COPD is also taken into consideration in assessments for the effects of byssinosis (PD D2).

Claims activity

25. In 2006, there were 980 claims for PD D12 (chronic bronchitis and emphysema) and 130 new assessments for disablement. In the same year, there were no new claims or assessments for PD C18 (cadmium-related emphysema). For PD D2 (byssinosis) [where COPD is taken into account during assessments for disablement], there were 20 new claims and no new assessments in 2006.¹

Background to this review

26. IAC holds regular public meetings throughout the United Kingdom. In March 2003, at the Sheffield public meeting, representations were made to IAC by participants to consider extending the prescription for chronic bronchitis and emphysema to surface coal workers. Following a preliminary review of the literature IAC decided to reconsider occupational coverage for chronic bronchitis and emphysema (PD D12) in surface coal workers and other occupations.

Method of investigation

27. In 2003, IAC announced its review of the occupational coverage for COPD (chronic bronchitis and emphysema) and commissioned an independent academic review of the scientific literature from the Institute for Environment and Health at the University of Leicester. This review specifically focused on research which quantified: a) the risk of developing COPD (chronic bronchitis and emphysema) in terms of lung function decrement and b) the exposures associated with this risk in different occupations.

28. The commissioned review examined the case for prescription for COPD (chronic bronchitis and emphysema) for a wide range of occupational groups. It recommended that IAC should consider the evidence in more detail for several occupational groups including:

- i) Cotton textile workers
- ii) Surface coal workers
- iii) Welders
- iv) Grain workers

¹ Claims and assessments in any particular year provide an indication of Industrial Injuries Disablement Benefit activity. It should be noted that individual claims may not be assessed in the same year as they are filed, likewise assessments may relate to a previous year's claim.

29. It also concluded there was limited literature available which addressed IIAC's questions about the level of FEV₁ loss in relation to detailed exposure data (i.e. the levels and durations of exposure). It was recommended that IIAC seek further evidence about lung function and exposure data directly from the experts who had conducted the published research (see Appendix 3). The commissioned review was published on the IIAC website for a three month period of public consultation. A call for evidence was also made requesting any evidence about occupations at risk of COPD (chronic bronchitis and emphysema) to be sent to IIAC. A second review was commissioned by IIAC from the Department of Epidemiology and Public Health at Imperial College, London to investigate the risk of COPD in silica-exposed workers. This commissioned review was also placed on the IIAC website for a period of public consultation. One of our members also asked consideration to be given to COPD due to exposure to isocyanates, and a targeted extensive review was conducted.

Difficulties in interpreting the research evidence

30. Because the dominant cause of COPD in the general population is cigarette smoking, it is important in evaluating studies of COPD in workforces to ensure that the potential confounding effect of cigarette smoking has been taken into account. In other words, to ensure that where the frequency of COPD is increased in a workforce as compared with a reference population, this is not due to an increased prevalence of cigarette smoking in the workforce.

31. For the purposes of recommending an occupation (e.g. underground coal mining) or exposure to a specific agent (e.g. cadmium fume) for prescription, IIAC looks for evidence that the proportion of those with an FEV₁ of 1L or more below the average is more than doubled in the exposed as compared with an unexposed population, having taken cigarette smoking into account. This approach, while necessary for IIAC, differs from that taken in the analysis of the majority of studies which have investigated the effects on lung function of occupational exposures. The results of such studies are often reported as the difference in average FEV₁ between the exposed and unexposed populations, without information about the distribution of FEV₁ in the two groups, which does not therefore allow the pattern of severe loss of lung function (illustrated by Figure 5) to be determined. The results of prospective (cohort) studies tend to be expressed as differences in the rates of decline in FEV₁, which although indicative of a relevant effect on FEV₁ of the occupational exposure, only allow an inference to be made by extrapolation, which may not be reliable, of the eventual magnitude of FEV₁ loss sufficient to meet the criteria for prescription. The scientific literature on COPD and occupation, while reporting wholly valid results of the findings, often does not provide direct evidence of an effect on lung function of an occupational exposure of a magnitude sufficient to allow recommendation of prescription.

32. Those employed in the occupations considered in this report will tend, at least during the first 10 to 15 years of employment, to be healthier than the average. The jobs are arduous and require a high level of physical fitness. This is called

the “healthy worker” effect. In addition, those who remain in such employment (“survivors”) are on average often healthier than employees who leave such work, leaving a healthier population at work, who are often those seen and investigated in research studies. In one study of coal miners lung function in those who had left work before retirement age was (on average) less than those who remained in employment. The implications are that, on average, lung function in those who are in employment is likely to be better than those of the same sex and age in the general population, and studies which compare lung function of employed groups with the general population can underestimate the magnitude of lung function loss in the occupational group. In addition, single measurements of lung function only provide information about the current level of lung function (say FEV₁), not about previous levels of FEV₁. As the normal population range in males varies by some 2L it is therefore possible for an individual (such as a worker in employment) to experience a significant loss of lung function, while remaining within the normal population range.

EVIDENCE RELATING TO SPECIFIC OCCUPATIONAL GROUPS

Surface coal workers

33. The current terms of prescription for chronic bronchitis and emphysema are restricted to underground coal workers who have worked for twenty years or more. IAC considered extending prescription for PD D12 to surface coal work in its 1996 report (Cm. 3240). The Council concluded there was a lack of evidence available to suggest that the respirable coal dust levels were sufficiently high to prescribe chronic bronchitis and emphysema for surface coal work.

34. The Council received representations about the dusty nature of certain occupational sub-categories of surface coal work during the IAC public meeting in Sheffield in 2003. The Institute of Occupational Medicine in Edinburgh has measured airborne dust levels in several British collieries as part of the Pneumoconiosis Field Research studies. In order to determine the dust levels associated with various occupational sub-groups at the surface of typical pits in the UK, IAC commissioned a review of these data. The Institute re-analysed the environmental data to address IAC’s questions about dust levels in surface occupations.

35. In the last IAC report on chronic bronchitis and emphysema (Cm. 3240) the risk of developing the diseases was described as a function of the cumulative exposure to respirable coal dust. A coal worker in any given occupation in the colliery is exposed to a level of respirable dust which can be expressed as the total mass of coal dust in a given volume of air. For example, a miner working at the coal face would, on average, have been exposed to 3-6 milligrams of dust per cubic metre of air (mg m⁻³) during the relevant time period. These data were used to estimate the average amount of dust that a miner would be exposed to over a typical working lifetime. In the case of the underground coal

worker a lifetime cumulative dust exposure in the order of 60-120 mg year m⁻³ would be reached after working for twenty years with a respiratory dust level of 3-6 mg m⁻³. From the evidence published on dust exposures the likelihood of developing a disabling loss of lung function is doubled at these levels of exposure.

36. With this information in mind, IAC asked the Institute of Occupational Medicine to provide details of any surface occupation which had an average respirable dust concentration of greater than 1.5 mg m⁻³, the minimum level at which an individual engaged in the occupation for a working lifetime of forty years would accumulate the qualifying level of coal dust exposure of 60-120 mg yr m⁻³.

37. Analysis of records from the Pneumoconiosis Field Research studies focused on several surface occupational job titles: screens surface, pickers screens surface, special bankers surface, rubbish flight tamber surface, smith and electric welders surface, welders surface and dry cleaner attendants surface. Among these, only work on the screens was consistently associated with a mean concentration of coal dust greater than 1.5 mg m⁻³.

38. Further analysis of the data established the average length of time a worker spent in these occupational sub-categories; for each pit, most workers spent little or no time at the surface but those workers who had surface occupations spent the majority of their time there. Screens work seemed particularly dusty and people were employed for greater than 90% of their time in this work. Screen workers handled and sorted coal manually. There was a high degree of variability in the exposure data for screen workers which seems likely to reflect the different times when the wetting process was introduced at the screens. Wetting served to reduce dust levels at the screens, so that screen workers were probably not exposed to the levels of dust associated with hazardous levels of dust after the introduction of wetting. Collieries had implemented effective wetting processes by 1983.

39. The Council recommends that the prescription for PD D12 be extended to include work at the screens on the surface of coal mines for 40 years or more with the qualifying exposure to have occurred before 1983. There is insufficient evidence that other sub-categories of surface work were exposed to sufficiently dusty conditions to be eligible for prescription.

40. The Council is aware that a number of underground coal workers may have transferred to surface work. The Council recommends that screen workers who were previously occupied underground in the mine can aggregate their duration of dust exposure, such that 2 years of surface screen work is equivalent to 1 year working underground. For example, a coal worker whose FEV₁ is 1L or more below the predicted value with 4 years working at the screens on the surface before 1983 and 18 years underground would be eligible for benefit.

41. IAC recognises that benefit will be restricted to workers in older age groups. The recommended date (i.e. 1983) is based on evidence of changes in work practices that is confirmed by trade unions. While relatively few people may qualify from surface work alone, more people will be eligible to claim by aggregating time as a surface coal worker with time as an underground coal worker.

Cotton textile workers

42. Work in the preparation of cotton prior to spinning, particularly in those employed in carding, stripping and grinding, has been associated with a high prevalence of byssinosis. Byssinosis is a disease of the airways caused by the inhalation of dust, of cotton and other organic fibres such as flax. It is characterised by episodes of chest tightness associated with acute airway narrowing which, in the early stages of the disease, occurs characteristically on the first day back at work after an absence, such as a weekend or holiday (Stage 1 byssinosis). With continuing exposure the symptoms and airway narrowing persist beyond the first day after an absence (Stage 2 byssinosis). With further exposure, persistent and irreversible airway narrowing, or COPD, can develop (Stage 3 byssinosis). Byssinosis is a prescribed disease in cotton and flax workers and COPD is therefore considered in these workers with a history of byssinosis.

43. However, it is unclear whether textile workers develop COPD in the absence of byssinosis, and independent of smoking, and whether there is sufficient evidence of increased risk to recommend a change to the prescription for PD D12.

44. Several studies have found that, on average, lung function is lower in cotton workers than in the general population and in general in those with a history of byssinosis. A mortality study reported in 1985 of women aged between 15 and 74 years found a marked excess in the proportional mortality ratio (PMR) from all causes of respiratory disease, including byssinosis, in textile workers, particularly in those employed as labourers and in fibre preparation, whose PMR was more than 200 (i.e. more than twice expected; a PMR of 100 is equivalent to no excess mortality). The Occupational Health Decennial Supplement published in 1995 reported a PMR for chronic bronchitis and emphysema in female textile workers of 119 and of byssinosis of 1140.

45. A study of a random sample of the population of Oldham and Bolton in Lancashire, which included ex-cotton workers, was undertaken in the mid 1980's to investigate the frequency of permanent effects of cotton dust on lung function. The average FEV₁ in ex-cotton workers was lower than in those in the population who had not been exposed to dust, by an amount similar to the effect of light smoking (less than 15 cigarettes/day), and FVC was lower by an amount equivalent to the effect of moderate (15-25 cigarettes/day) or heavy smoking (more than 25 cigarettes/day). The study did not distinguish cotton workers with a history of byssinosis from those without and did not provide information on the proportion of ex-cotton workers, as compared with those not exposed to dust, whose FEV₁ was 1L or more below their predicted average values.

46. A recent 20 year follow up study from China comparing the rate of lung function decline in cotton and silk workers found an excess rate of decline in FEV₁ in cotton workers, primarily in those with a history of byssinosis.

47. IAC commissioned a study by the University of Manchester to analyse existing data from a longitudinal study comparing lung function in textile workers and man-made fibre textile workers. The group analysed contained few subjects with byssinosis and account was taken of smoking habits. The odds of a disabling loss of lung function were doubled in those working for more than 15 years in cotton mills, but the estimate was subject to statistical uncertainty, and no clear pattern was found by work area or cumulative level of exposure in the analyses presented. The Council is unaware of any comparable data from other investigations.

48. Whilst valid evidence exists suggesting loss of lung function due to exposure to cotton dust, it is not possible either to distinguish an effect due to cotton dust which is independent of cigarette smoking and byssinosis or to establish which jobs incur sufficient risk. The Council has concluded that there is insufficient evidence to recommend prescription of COPD in cotton or other textile workers without a history of byssinosis.

49. The Council recommends that the current terms of prescription for byssinosis should stay unchanged.

Welders

50. Welding is the process of joining metals together by the application of heat or pressure or both, with or without the use of a filler metal. Depending on the type of welding process and the metals being welded, welding can generate exposures to a wide variety of fumes, gases and dusts, which are associated with well described adverse effects such as “metal fume fever”, pulmonary oedema and pneumococcal pneumonia.

51. Several studies have investigated the question of whether the frequency of COPD is increased in welders, with conflicting results. An excess prevalence of cough and sputum among welders has been found in several studies, but a reduction in FEV₁ has been found inconsistently. Studies of shipyard workers in North East England have reported abnormalities in lung function in those exposed to welding fumes in comparison with other shipyard workers. In a study of 607 shipyard workers whose age ranged between 17 and 69 years, FEV₁ in those exposed to welding fume was on average 250ml less than those in other trades. However, this effect was found only in cigarette smokers. A follow up study was undertaken some 7 years later which included 487 of those seen in the first study, many of whom were no longer employed in the shipyard. The findings suggested that the rate of decline of FEV₁ in a 50 year old non-smoker not exposed to welding fume was on average 16.2ml per year. The additional loss associated with cigarette smoking was an estimated 17.7ml per year and with welding 16.4ml per year. The report of the study did not include an estimate

of the prevalence of important FEV₁ loss in those exposed to welding fume as compared with other shipyard workers. We met with the senior author of the report to enquire whether further analysis of the data was feasible to address this question, but unfortunately this did not prove possible.

52. Other studies of welders in Royal Naval dockyards undertaken in 1980's did not find evidence for an adverse effect on lung function in welders. We also met with the senior investigator of these studies, who confirmed these findings to us.

53. Despite extensive investigation the Council has not found evidence that welders have a greater than doubling of risk of developing an FEV₁ reduction of 1 L or more as compared to the general population. We are therefore not able to recommend extension of prescription for PD D12 to welders.

Grain silo workers

54. Several studies have shown an increased prevalence of cough and sputum in grain workers. A series of studies undertaken in Canada in 1970's and 1980's comparing grain elevator workers with office workers found a lower average level of FEV₁ and an increased rate of decline of FEV₁. Average FEV₁ and the rate of decline of FEV₁ was related to the measured level of exposure to grain dust. On average exposure to grain dust had a similar effect on FEV₁ as 6 mg m⁻³ smoking 1 pack of cigarettes a day. The results of these studies however, do not allow an estimate of the prevalence of important FEV₁ loss in grain as compared with the office workers.

55. There have been no comparable studies of grain workers in UK which would allow an understanding of the risk of COPD in grain workers in this country.

56. In the absence of evidence to indicate a doubling of risk of a disabling loss of FEV₁ the Council is unable to recommend the prescription of COPD in grain workers.

Silica-related COPD

57. The literature on silica and COPD is sufficiently large to have been the subject of a separate review commissioned by IIAC from the Department of Epidemiology and Public Health at Imperial College, London.

58. Silica (or silicon dioxide) is abundant in the earth's surface; occupational exposure to it is widespread in several industries, which include mining, tunnelling, quarrying, construction and work in quarries, potteries and foundries. Inhalation of respirable silica can cause silicosis, which is characterised by fibrous nodules in the lungs; these can coalesce to cause conglomerate silicosis, which causes severe loss of lung function and early death. Silicosis is also associated with a markedly increased risk of tuberculosis. Silicosis and silicosis complicated by tuberculosis are already covered by the prescribed disease provisions of the IIDB scheme [PD D1 (pneumoconiosis – silicosis)].

59. Silica presents particular difficulties in considering prescription in relation to COPD. Inhaled silica is fibrogenic, causing silicosis. The question to be answered for which IIAC has sought evidence, is whether a disabling loss of lung function due to COPD is caused by inhaled silica dust, independently of silicosis or other confounding factors, such as tobacco smoking. While a large scientific literature exists about silica and COPD, few studies have provided clear evidence of an effect of silica in causing COPD, independent of smoking and silicosis, or of the magnitude of the loss of lung function caused by such an effect.

60. Distinguishing the separate effects of silicosis and COPD is inherently problematic. Silicosis causes a loss of lung volume with a reduction in FEV₁ that may mimic loss of lung function arising from airways obstruction (COPD). Researchers have attempted to address this problem in several different ways. Some studies have considered the FEV₁/FVC ratio, which will be reduced in airways obstruction and unchanged in lung fibrosis in which a parallel loss of FEV₁ and FVC occurs. Other studies have excluded patients with evidence of silicosis on the chest radiograph. However, chest radiographs do not detect small silicotic nodules. A few studies have therefore used computed tomography (CT) scans of the lungs, which have a higher resolution than the chest radiograph and can distinguish silicotic nodules from emphysema. Such studies however, are limited by the size of the population in which CT scans can be undertaken. Investigations have also been undertaken of the relationship between exposure to silica during life to the presence of silicosis and emphysema at post mortem. A separate problem in interpreting the findings of the reported studies, in groups such as miners and tunnellers, is the mixed exposures experienced and the difficulty in distinguishing the effects of inhaled silica from exposure to other airborne pollutants, such as nitrogen dioxide. Finally, there is the need to understand the effects of silica on COPD independent of smoking. The question is whether a disabling loss of lung function from COPD, caused by silica exposure, occurs independently of silicosis or other confounding factors such as tobacco smoking or other occupational exposures. As the paragraphs below highlight, no study provides a complete solution to all of these difficulties.

61. The commissioned review identified several studies, particularly in gold miners and construction workers, which suggested an association between silica exposure and the development of COPD. However, the studies reviewed also identified an important and consistent effect of cigarette smoking on lung function in the populations studied. Several studies were of respiratory disease in South African gold miners. These have found a relationship between increasing severity of silicosis on the chest radiograph and magnitude of loss of FEV₁ and FVC. In addition, they found evidence for associated airways obstruction (reduced FEV₁ and FEV₁/FVC ratio), which was particularly marked in cigarette smokers. One study of South African gold miners found that COPD occurred predominantly in cigarette smokers; the independent effects of silica inhalation were relatively small in comparison with the effects of cigarette smoking.

62. Studies of the lungs of South African gold miners at post mortem found the presence of emphysema to be associated with shortness of breath and reduced FEV₁ during life. The major factor associated with the presence of emphysema in these studies was cigarette smoking during life. Some studies reported an association between exposure to silica in life and the degree of emphysema at post mortem. However, this association was generally limited to cigarette smokers and does not provide convincing evidence of an effect of silica independent of smoking in causing emphysema. Few studies have considered miners who have never smoked, but one such investigation found no relationship between the level of exposure to silica dust and a clinically important degree of emphysema. One study of the relationship of emphysema to silica dust exposure in South African gold miners concluded, that as compared with the effect of tobacco smoke, the effect of silica dust on emphysema appeared very small; tobacco smoking potentiated the effects of silica dust on emphysema, but silica dust exposure in the absence of tobacco smoke was seldom associated with a significant degree of emphysema².

63. Studies in construction workers, particularly tunnellers, have found evidence for an increased rate of decline of FEV₁ in tunnel workers, as compared with a control group consisting of outdoor concrete workers, firemen and engineers. One study found a prevalence of COPD, as defined by respiratory symptoms and an FEV₁/FVC ratio of less than 70%, of 14% in tunnel workers and of 8% in the comparison group. A subsequent study of the relationship of lung function loss with the different exposures to which the tunnellers were exposed, identified cumulative exposure to nitrogen dioxide as the most probable cause. No significant relationship was found between cumulative exposure to quartz (a crystalline form of silica) and the rate of decline of FEV₁, in those who had never smoked, although the rate of decline in FEV₁ in those who had ever smoked was associated with exposure to all agents, including quartz. However, the strongest relationship overall between FEV₁ decline in both smokers and non-smokers was with cumulative exposure to nitrogen dioxide.

64. Fibrotic nodules in the lungs of cases of silicosis can be seen on the chest radiograph, when above a minimum size. In order to distinguish the fibrogenic effect of silica from an effect on the airways, several studies have excluded cases with evidence of silicosis on the chest radiograph. In general, these studies found evidence for a greater rate of decline of FEV₁ and of reduced FEV₁/FVC ratio, in both smokers and non-smokers, in relation to cumulative silica exposure. However, it is unclear the extent to which these changes might be related to silicotic nodules, which are undetectable on a chest radiograph and whether such decrements of loss eventually give rise to a more than doubled risk of disabling lung function. To overcome the first of these limitations, a few studies have investigated lung function in silica workers using CT scans, which have a higher resolution than chest radiographs of the lung, to identify and distinguish the changes of silicosis and emphysema.

² (Hnizdo E, Sluis-Cremer GK, Abramowitz JA. Emphysema type in relation to silica dust exposure in South African gold miners. *Am Rev Respir Dis* 1991;143:1241-1247).

65. These studies have in general found a stronger association of lung function loss with changes of emphysema than with silicotic nodules on CT scans. In one study of 111 silica exposed workers, non-smokers without silicosis had no evidence of emphysema, whereas all smokers with silicosis had emphysema. In smokers, the prevalence and severity of emphysema increased with the severity of silicosis. Emphysema and severe airways obstruction were particularly prevalent in cases of conglomerate silicosis³. The results of these studies suggest that the major cause of lung function loss, particularly in cigarette smokers, is the severity of emphysema associated with silicosis, rather than the direct effects of the silicotic nodules.

66. The inference from these studies, as from the studies of South African gold miners, is that, as compared to tobacco smoke, the contribution of inhaled silica to the development of emphysema is small. Furthermore relationships between cumulative silica exposure and the risk of emphysema have been found primarily in cigarette smokers.

67. Thus, while a large scientific literature exists about silica and COPD, the studies reviewed do not provide clear evidence of a doubling of risk of a disabling loss of FEV₁ attributable to COPD, independent of silicosis and of cigarette smoking, in silica exposed workers as compared to the general population.

68. The Council is therefore unable to recommend prescription of COPD in workers exposed to inhaled silica dust.

69. Pneumoconiosis due to exposure to silica (i.e. silicosis) is a prescribed disease (PD D1) and lung function loss attributable to emphysema in cases of silicosis is taken into account in the assessment of disability.

Isocyanate exposed workers

70. Isocyanates are a group of highly reactive, low molecular weight chemicals. In the UK approximately 60,000 tonnes of isocyanates are used every year in polyurethane foam manufacture and other substances including paints, inks, lacquers and synthetic rubbers.

71. Inhalation of isocyanates in high concentrations is toxic, causing inflammatory changes in mucous membranes of the respiratory tract, eyes and stomach. At lower concentrations, exposure to isocyanates may cause asthma due to the development of a specific hypersensitivity reaction. In fact, isocyanates are the single most frequent cause of occupational asthma in the UK. Occupational asthma due to isocyanates is a prescribed disease (PD D7a).

72. IIAC conducted a literature search and analysed the evidence relating to isocyanates and COPD. A key study followed up a cohort of workers exposed to isocyanates for 5 years with serial measurements of lung function. In this

³ Conglomerate silicosis is a more severe form of silicosis, where as the disease progresses the small nodules (characteristic of simple silicosis) coalesce to form conglomerate masses observed on chest radiographs.

study a reduction in FEV₁ was observed in non-smokers exposed to the higher levels of isocyanates. There was no additional loss of FEV₁ among smokers with similarly high levels of exposure. Results of other studies have not been consistent with the findings in this study and none have reported levels of FEV₁ reduction sufficiently disabling to recommend prescription.

73. IIAC concludes that, at present, there is insufficient evidence to be able to recommend prescription of COPD due to isocyanates in any occupational category. However, the Council will continue to monitor future evidence.

Prevention

74. The contribution of work to the overall burden of COPD can be prevented by ensuring that workers are not exposed to causative dusts, gases and fumes.

75. The Control of Substances Hazardous to Health Regulations 2002 (COSHH) apply to work with hazardous substances. These regulations require that work is not carried out with any substance liable to be hazardous to health unless a suitable and sufficient assessment has been made of the risks created by the work and measures are taken to prevent exposure as far as is reasonably practicable. Where it is not reasonably practicable to prevent exposures they must be adequately controlled by the use of appropriate work processes, systems and engineering controls and measures, including ventilation systems, to control exposures at source. Suitable respiratory protective equipment may be used in addition, where adequate control cannot otherwise be achieved.

Recommendations

76. The Council considers that the current prescriptions for PD D12 (chronic bronchitis and emphysema) relating to underground coal workers, for emphysema for cadmium exposed workers (PD C13) and byssinosis for cotton textile workers (PD D2) remain appropriate.

77. The Council further recommends that the prescription for PD D12 (chronic bronchitis and emphysema) should be extended to include exposure to coal dust during screen work at the surface of a coal mine, for a period of at least 40 years in aggregate, for exposures before 1983 (see box below).

Disease number	Name of disease or injury	Type of job Any job involving
D12	<p>Chronic bronchitis or emphysema (or both) where, with maximum effort there is evidence of forced expiratory volume in one second (measured from the position of maximum inspiration with the claimant making maximum effort) which is:</p> <p>(i) at least one litre below the appropriate mean value predicted, obtained from the following predictions formulae which give the mean values predicted in litres:</p> <ul style="list-style-type: none"> • For a man, where the measurement is made without back-extrapolation, $(3.62 \times \text{Height in metres})$ minus $(0.031 \times \text{Age in years})$ minus 1.41; or, where the measurement is made with back-extrapolation, $(3.71 \times \text{Height in metres})$ minus $(0.032 \times \text{Age in years})$ minus 1.44 • For a woman, where the measurement is made without back-extrapolation, $(3.29 \times \text{Height in metres})$ minus $(0.029 \times \text{Age in years})$ minus 1.42; or, where the measurement is made with back-extrapolation, $(3.37 \times \text{Height in metres})$ minus $(0.030 \times \text{Age in years})$ minus 1.46 or <p>(ii) less than one litre</p>	<p>Exposure to coal dust (whether before or after 5 July 1948) by reason of working:</p> <p>a) underground in a coal mine for a period or periods amounting in the aggregate to at least 20 years</p> <p>or</p> <p>b) on the surface of a coal mine as a screen worker for a period or periods amounting in the aggregate to at least 40 years before 1983</p> <p>or</p> <p>c) a combination of underground and screen working, such that 2 years working as a surface screen worker before 1983 is equivalent to 1 year working underground, amounting in the aggregate to at least the equivalent of 20 years working underground.</p> <p>Any such period or periods shall include a period or periods of incapacity while engaged in such an occupation.</p>

78. IIAC recommends that time spent as a screen worker can be aggregated with previous time spent underground in coal mines to qualify for prescription, such that two years spent working on the surface as a screen worker is equivalent to one year working underground in a coal mine.

79. The Council is unable to recommend prescription for any other occupational category but will continue to monitor research in this area.

Call for research studies to be undertaken

80. Prescription for occupational COPD has been hampered due to a lack of suitable evidence of a disabling loss of lung function in exposed workers, where exposure data have also been collected. The Council does not have a budget to fund primary research itself, but requests that such studies are undertaken in order that it can revisit the question of prescription in the future. Many studies report the mean loss of lung function per unit time, but the Council requires to know the distribution of lung function, and in particular the proportion of workers with an FEV₁ 1L below predicted as compared with the proportion in a suitable comparator group (see paragraphs 16 and 19).

APPENDIX 1: The legal requirements for prescription

The Industrial Injuries Disablement Benefit Scheme

81. The Industrial Injuries Disablement Benefit (IIDB) scheme provides non-contributory, 'no-fault' benefits for disablement because of accidents or prescribed diseases which arise during the course of employed earners' employment. The benefit is paid in addition to other incapacity and disability benefits. It is tax-free and administered by the Department for Work and Pensions.

The role of the Industrial Injuries Advisory Council

82. IIAC is an independent statutory body established in 1946 to advise the Secretary of State for Social Security on matters relating to the IIDB scheme. The majority of the Council's time is spent considering whether the list of prescribed diseases for which benefit may be paid should be enlarged or amended.

The legal requirements for prescription

83. The Social Security Contributions and Benefits Act 1992 states that the Secretary of State may prescribe a disease where he is satisfied that the disease:

- a) ought to be treated, having regard to its causes and incidence and any other relevant considerations, as a risk of the occupation and not as a risk common to all persons; and
- b) is such that, in the absence of special circumstances, the attribution of particular cases to the nature of the employment can be established or presumed with reasonable certainty.

84. In other words, a disease may only be prescribed if there is a recognised risk to workers in an occupation, and the link between disease and occupation can be established or reasonably presumed in individual cases.

85. In seeking to address the question of prescription for any particular condition, the Council first looks for a workable definition of the disease. The Council then searches for a practical way to demonstrate in the individual case that the disease can be attributed to occupational exposure with reasonable confidence. For this purpose, reasonable confidence is interpreted as being based on the balance of probabilities according to the available evidence in the scientific literature. An accident at work is specifically catered for within the IIDB scheme. However, if the condition might result from occupational exposure in the absence of an identifiable accident, the Council must consider whether it should be included in the list of diseases that are prescribed for benefit purposes. In these circumstances, it may be possible to ascribe a disease to a particular occupational exposure in two ways – from specific clinical features of the disease or from epidemiological evidence that the risk of disease is at least doubled by the relevant occupational exposure.

Clinical features

86. For some diseases attribution to occupation may be possible from specific clinical features of the individual case. For example, the proof that an individual's asthma is caused by his occupation may lie in its improvement when s/he is on holiday and regression when s/he returns to work, and in the demonstration that s/he is allergic to a specific substance with which s/he comes into contact only at work. It can be that the disease only occurs as a result of an occupational hazard (e.g. coal workers' pneumoconiosis).

Doubling of risk

87. Other diseases are not uniquely occupational and, when caused by occupation, are indistinguishable from the same disease occurring in someone who has not been exposed to a hazard at work. In these circumstances attribution to occupation on the balance of probabilities depends on epidemiological evidence that work in the prescribed job, or with the prescribed occupational exposure, increases the risk of developing the disease by a factor of two or more. In the case of chronic bronchitis and emphysema the criteria for prescription were that there needed to be evidence of a greater than doubled risk of a disabling loss of lung function in exposed compared with unexposed workers. The requirement for, at least, a doubling of risk is not arbitrary. It follows from the fact that if a hazardous exposure doubles risk, for every 50 cases that would normally occur in an unexposed population, an additional 50 would be expected if the population were exposed to the hazard. Thus, out of every 100 cases that occurred in an exposed population, 50 would do so only as a consequence of their exposure while the other 50 would have been expected to develop the disease, even in the absence of the exposure. Therefore, for any individual case occurring in the exposed population, there would be a 50% chance that the disease resulted from exposure to the hazard, and a 50% chance that it would have occurred even without the exposure. Below the threshold of a doubling of risk only a minority of cases in an exposed population would be caused by the hazard and individual cases therefore could not be attributed to exposure on the balance of probabilities. The epidemiological evidence required should ideally be drawn from several independent studies, and be sufficiently robust that further research at a later date would be unlikely to overturn it.

APPENDIX 2: Current terms of prescription for COPD – chronic bronchitis and emphysema

Disease number	Name of disease or injury	Type of job Any job involving
D12	<p>Chronic bronchitis or emphysema; or both where, with maximum effort, where there is accompanying evidence of forced expiratory volume in one second (measured from the position of maximum inspiration with the claimant making maximum effort) which is:</p> <p>(i) at least one litre below the appropriate mean value predicted, obtained from the following predictions formulae which give the mean values predicted in litres:</p> <ul style="list-style-type: none"> • For a man, where the measurement is made without back-extrapolation, $(3.62 \times \text{Height in metres})$ minus $(0.031 \times \text{Age in years})$ minus 1.41; or, where the measurement is made with back-extrapolation, $(3.71 \times \text{Height in metres})$ minus $(0.032 \times \text{Age in years})$ minus 1.44 • For a woman, where the measurement is made without back-extrapolation, $(3.29 \times \text{Height in metres})$ minus $(0.029 \times \text{Age in years})$ minus 1.42; or, where the measurement is made with back-extrapolation, $(3.37 \times \text{Height in metres})$ minus $(0.030 \times \text{Age in years})$ minus 1.46 or <p>(ii) less than one litre</p>	<p>Exposure to coal dust by reason of working underground in a coal mine for a period or periods amounting in the aggregate to at least 20 years (whether before or after 5 July 1948) and any such period or periods shall include a period or periods of incapacity while engaged in such an occupation.</p>

Disease number	Name of disease or injury	Type of job Any job involving
C18	Emphysema	Inhalation of cadmium fumes for a period of, or periods which amount in aggregate to, 20 years or more.
D2	Byssinosis ⁴	Work in any room where any process up to and including the weaving process is performed in a factory in which the spinning or manipulation of raw or waste cotton or of flax, or the weaving of cotton or flax, is carried on.

⁴ The effects of COPD are taken into consideration when assessing benefit for claimants for PD D2.

APPENDIX 3: Consultations with experts

Professor John Cotes	Formerly of the University of Newcastle-upon-Tyne
Professor David Hendrick	University of Newcastle-upon-Tyne
Dr Rob Niven	University of Manchester
Dr Brian Miller	Institute of Occupational Medicine, Edinburgh
Surgeon Commander Grant McMillan	Institute of Occupational and Environmental Medicine, University of Birmingham
Dr David Chinn	University of Edinburgh
Dr Lesley Rushton	Imperial College, London