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THE INDUSTRIAL INJURIES ADVISORY COUNCIL

POSITION PAPER 45

Chronic obstructive pulmonary disease and coke oven workers

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Industrial Injuries Advisory Council Position Paper 45:

Chronic Obstructive Pulmonary Disease and Coke Oven Work

Summary

This position paper concerns the possible prescription of chronic obstructive pulmonary disease (COPD) in coke oven workers under the Industrial Injuries Scheme (IIS). There is a body of evidence which shows an association between coke oven exposures and non-malignant pulmonary disease, but there is lack of detail in some studies and inconsistency in the nature and magnitude of the effects in others. In the view of the Council the published evidence is insufficient to recommend prescription.

This report contains technical terms, the meanings of which are explained in a concluding glossary.

Background

1. Chronic obstructive pulmonary disease (COPD) is a common condition which is characterised by persistent respiratory symptoms and airflow limitation associated with airway disease and emphysema (GOLD 2019). Cigarette smoking is the commonest cause of COPD but a range of occupational exposures to dusts, gases and fumes can cause the disease or contribute to its severity.

2. IIAC first commented on COPD in 1973 (Cmnd. 5443), which at that time was referred to as 'chronic bronchitis and emphysema'. Due to difficulties in identifying groups of workers likely to meet IIAC's criteria, it was not until 1992 that prescription for COPD was first recommended; in this instance for coal miners (Cmnd. 2091). By that time there was sufficient evidence that coal miners who had worked underground for 20 years or more had at least a doubled risk of developing disabling COPD. This was defined as a forced expiratory volume in one second (FEV_1) in a lung function test of 1 litre or more below the predicted value. Subsequent evidence allowed the initial requirement for an abnormal chest radiograph to be removed and prescription to be extended to surface workers in coal mines.

3. In 2002 IIAC found that there was evidence of an excess of emphysema in workers with prolonged or heavy exposure to cadmium fumes. The Council recommended that the terms of prescription for PD C18 (poisoning by cadmium) be amended to prescribe emphysema for those with exposure to cadmium fumes for twenty years or more. COPD is also considered to be a component of stage 3 byssinosis and is taken into consideration in assessments of disability from that condition (PD D2).

4. IIAC reviewed the potential occupational causes of COPD in 2003 and commissioned independent reviews. The Council considered, in detail, the evidence relating to several occupational groups including:

    a. Cotton textile workers
    b. Surface coal workers
    c. Welders
    d. Grain workers
    e. silica-exposed workers
    f. Isocyanate-exposed workers

5. Of these, the only group which were considered to meet the criteria for prescription were screen workers on the surfaces of coal mines with 40 years or more exposure before 1983. Screen workers who were previously employed underground in mines could aggregate their exposures, with 2 years of surface screen work equivalent to 1 year working underground.

6. IIAC also considered COPD in woodworkers (IIAC Information note 2015). The scientific evidence was limited, of variable quality, and inconsistent in its findings. Prescription was not recommended.

7. The Council recently became aware of civil litigation dealing with COPD in coke oven workers (Pearce and Others v Secretary of State and others) and in workers in a phurnacite plant where there are likely to have been exposures similar to those in a coking plant (Jones and Others v Secretary of State and Others). Whilst recognising that the issues IIAC takes into account when considering prescription under the IIS are not the same as those considered in civil litigation, IIAC has nevertheless undertaken a review of the literature for this group of workers to determine whether they meet the requirements for prescription.

Industrial Injuries Disablement Benefit (IIDB)

8. IIDB provides non-contributory, 'no-fault' benefits for disablement because of accidents or prescribed diseases which arise during the course of employed earners' work. 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.

9. The legal requirements for prescription are set out in The Social Security Contributions and Benefits Act 1992 which 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.

10. Thus, 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.

The Role of the Industrial Injuries Advisory Council (IIAC)

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

12. In considering the question of prescription, the Council 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.

13. Some occupational diseases are relatively simple to verify, as the link with occupation is clear-cut. Some only occur due to particular work or are almost always associated with work, have specific medical tests that prove their link with work, have a rapid link to exposure, or have other clinical features which make it easy to confirm the work connection. Occupational asthma for example falls into that category. COPD and many 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 depends on research evidence that work in the prescribed job or with the prescribed occupational exposure causes the disease on the balance of probabilities. The Council thus looks for evidence that the risk of developing the disease associated with a particular occupational exposure or circumstance is more than doubled. (Previous reports of the Council explain why this threshold was chosen.)

Coke oven exposures

14. The process of coking involves heating coal to temperatures of over 1,000°C in the absence of air in a coke oven. Volatile components of the coal are driven off leaving coke which is mainly carbon. In the past coke was widely used in steel production, but with the decline in the steel industry it is no longer produced on a large scale in the UK.

15. Occupational exposures around coke ovens are complex and include airborne coal dust, coke dust, a range of polycyclic aromatic hydrocarbons (PAHs), toxic gases and vapours. Exposures are greatest for those working on the top of the oven (oven top workers) but other workers in coking plants are likely to have some exposure to hazardous dusts and fumes. In the UK, respiratory protection for coke oven top workers was introduced in the late 1970s, but an effective programme was only in place for all oven top workers from around 1982 (Crawford et al 2014).

16. Coke oven workers are recognised to be at increased risk of lung cancer and lung cancer is prescribed for those employed wholly or mainly as a coke oven worker with a cumulative duration of exposure of 15 years or more; or a cumulative duration of top oven work of 5 years or more; or a combination of the two equivalent to a cumulative duration of 5 years on top oven work.

17. Exposures relevant to lung cancer are characterised as the benzene-soluble fraction of the airborne particles (benzene soluble material or BSM, sometimes also referred to as coal tar pitch volatiles or CTPV) and/or one of the main BSM,

benzo(a)pyrene (BaP). Exposures in coke ovens were high during the 1970s in Britain with shift average BSM for oven top workers ranging from around 1,000 to 3,000 g/m^3 and BaP from about 5 to 17 g/m^3. Exposures were slightly lower for oven side workers and lower again for workers employed elsewhere in coke plants (Miller et al 2013).

18. The Phurnacite plant, a specialised coal carbonisation facility similar to a coke oven, operated in South Wales from 1942 until 1991. The plant made smokeless fuel briquettes for domestic heating by compressing crushed coal with a pitch binder prior to coking. In the 1970s total dust concentrations in the areas where coal was crushed and formed into briquettes and on the oven tops were high, typically between 10 and 40 mg/m^3. BSM and BaP concentrations for ovens workers were higher than in most coke oven work around that time, i.e. BSM concentrations were typically around 3,000 g/m^3 and BaP concentrations about 40 g/m^3.

19. The type of exposure which might cause COPD is not likely to be identical to that causing lung cancer. Coal dust, for example, which is present on coking plants can cause COPD but is not a recognised cause of lung cancer. There is little published evidence about exposures on coking plants other than BSM on and around the ovens.

COPD and coke oven exposures

20. IIAC has reviewed the English-language epidemiological evidence relating coke oven exposures to COPD. 12 mortality studies (table 1) and 7 studies which included lung function measurements (table 2) were identified.

Mortality studies of specific working populations

21. There are a number of mortality studies of coke and coal gasification plant workers that were generally designed to investigate malignant disease (Table 1). Several included some information on respiratory mortality. In most studies this was reported as non-malignant respiratory disease; bronchitis; or bronchitis, emphysema and asthma. Only one small study specifically reported mortality for COPD. Not all studies distinguished oven workers who are likely to have had the heaviest exposures from other plant workers.

22. In general, the mortality rates were modestly elevated with a less than doubled risk of death from respiratory disease.

23. Mortality rates are likely to underestimate the overall burden of COPD as the disease is usually non-fatal and can be present for many decades before causing

death. Mortality studies are therefore considered to provide little relevant information on the magnitude of the risks of COPD in working populations.

Table 1 – Mortality studies of coke oven workers

Reference	Study description	Mortality rates	Comments
Lloyd 1971	Mortality study of 2532 coke oven workers who formed part of a larger cohort of 58,800 Pennsylvianian steelworkers employed up to 1953 and followed-up to 1961.	SMR[1] = 1.03 (7 deaths)	SMR is for all non-malignant respiratory diseases for coke oven workers and not just COPD.
Redmond 1976	Follow up of the same cohort to 1966 by which time there had been 8,600 deaths.	SMR = 1.19 (11 deaths)	SMR again is for all non-malignant respiratory diseases.
Redmond 1972	Mortality study of 4,600 coke oven workers employed in 10 other North American steel plants 1951- 1955	PMR [2]=0.99 (7 deaths, top oven work) PMR =1.78 (5 deaths, partial top)	SMRs are for non-malignant, non-tuberculous respiratory disease
Doll 1972	Report on 547 deaths among 2449 'coal carbonisation workers' in 4 UK gasworks	PMR= 1.57 (77 deaths)	PMR is for 'bronchitis' in process workers. No increase in mortality was seen in a smaller survey of 4 further gasworks.
Davies 1977	Report on 82 deaths amongst 610 coke oven workers employed in 2 South Wales steelworks	SMR = 0.74 (6 deaths)	SMR is for 'bronchitis'. Employment histories were not sufficiently detailed to subdivide the coke plant workers
Collings 1978	Report on 276 deaths over a 9 year period amongst 2854 workers at 13 British Steel coking works	PMR = 1.41 (0.63-2.19)	PMR is for 'bronchitis, emphysema or asthma' amongst coke oven workers
Jacobsen 1977	Report on mortality amongst 4836 workers on 13 UK coking plants	PMR = 1.28 (0.55-2.01)	PMR is for 'bronchitis, emphysema or asthma' amongst coke oven workers
Hurley 1983	Report on 1137 deaths amongst 6746 coke plant workers employed in 14 British Steel (BSC) plants and 13 National Smokeless Fuel (NSF) plants.	SMR = 1.31 (18 deaths, BSC) SMR = 1.17 (19 deaths, NSF)	SMR is for 'bronchitis, emphysema or asthma' amongst coke oven workers
Swaen 1991	Dutch mortality study of 1319 deaths amongst 5659 ex-coke oven workers	SMR = 1.66 (1.24-2.18: all workers) SMR = 1.75 (1.07-2.70: top oven workers	SMRs reported for all non-malignant respiratory disease
Chau 1993	Mortality study of 182 deaths amongst French retired coke plant workers	SMR = 0.68 (6 deaths)	SMR reported for non-malignant respiratory disease in coke plant workers. There were no deaths amongst oven workers and 2 (SMR = 0.77) amongst near-oven workers
Bye 1998	Report on 122 deaths among 888 Norwegian ex-coking works employees.	SMR = 0.57 (0.26-1.09)	SMR reported for COPD for all coke plant workers

[1] SMR = standardised mortality ratio
[2] PMR = proportional mortality ratio

Lung function studies

24. Lung function measurements are a more precise means of identifying COPD. The disease is characterised by a low ratio of the forced expiratory volume in one second (FEV_1) to the forced vital capacity (FVC). There are problems of definition: often a FEV/FVC ratio of <70% is taken to indicate COPD but as FEV/FVC falls with age that underestimates COPD prevalence at younger ages and overestimates it at older ages. An alternative approach is to take the lower limit of normal of FEV/FVC from equations established from an appropriate reference population based on age, height and ethnicity. Choosing appropriate reference population can be difficult as lung function can vary between groups of normal individuals even over relatively short geographic distances.

25. Comparisons of lung function between working groups and control populations are potentially confounded by differences which are unrelated to the exposure under investigation. It is common practice, therefore, to carry out an internal comparison within a working group examining lung function at different levels of exposure. That can be carried out either in a cross-sectional or longitudinal study. Each has its own strengths and weaknesses.

Table 2 – Lung function studies of coke oven workers

Reference	Study description	Mean effect of exposure on FEV_1	Mean effect of smoking on FEV_1	Comments
Walker 1971	Study of symptoms and lung function of 312 coke oven workers and 464 others based at 2 coking plants in Durham and Yorkshire.	-130 ml -50 ml (using analysis of covariance)	-156 ml -80 ml (analysis of covariance for smoking + previous dust exposure)	No information is given about FVC and it is not clear that the reduction in FEV_1 is associated with airflow obstruction/COPD.
Chau 1992	French study of 231 retired but surviving coke oven workers and miners 12 years post retirement.	+140 ml	-131 ml	FEV_1 was 2.46 for oven workers (n= 30); 2.47 for near oven workers (n=67) ; and 2.37 for non-exposed workers (n=46). The study was prone to survivor bias.
Madison 1984	Study of 3,799 workers in 11 US coke plants 1978-1982. Workers were categorised into 6 groups based on estimated exposures to noxious substances	-367 ml	-180 ml	There was a gradient of FEV_1 from presumed least exposed to most exposed category. Regression analysis showed relationships between FEV/FVC<70% and exposure category.

Corhay 1988	137 Belgian coke oven workers compared with 150 blast furnace workers:	-140 ml (approximately: -3.2% of predicted)		The coke oven workers were slightly older (42 vs 41 yr) and smoked more (82% current/ex-smokers vs 78%). The study was particularly focussed on asthma/ airway responsiveness and coke oven workers had more airway responsiveness than the others (49% vs 19% with measurable PC20).
Wu 2002	Cross-sectional study of 834 Australian coke oven workers. The primary analysis was of years spent on 'operational' work which involved the heaviest exposures	-9 ml/yr (-3 to -16 ml)	-9 ml/pk-yr (-116 ml for smokers with mean 13.5 pk-yr smoking)	Whereas the effect of smoking on FEV was greater than the effect on FVC the opposite was the case for operational work suggesting that the effect demonstrated was not simply COPD
Wu 2004	Longitudinal study based on the same workforce. 580 workers were followed up over approximately 8 years between 1978 and 1990.	-0.5 ml/yr (0.1 to 1.0)	-0.6 ml/yr (0.3 to 0.9)	The effect appeared much less than in the cross sectional study though still on a par with the effect of smoking. As with the cross sectional study the effect on FVC was greater than the effect on FEV. The effect was slightly greater (-0.8 ml/yr) when 111 subjects without detailed exposure histories were excluded.
Hu 2006	A study of 712 Chinese coke oven workers employed on 2 plants, and 211 unexposed control subjects. Exposures were quantified as BSP.	-420 ml (high exposure group)		30% of subjects in the high exposure group had FEV/FVC<70% compared with 7% in the control group. (OR 5.80: 3.13 to 10.76). There was a multiplicative interaction between exposure and smoking with OR 58 (11-305) in the heaviest exposed subgroups.

26. Walker et al (1971) studied symptoms and lung function of 312 oven workers and 464 others based at two UK coking plants (Table 2). The study focused on bronchitis and that was used as an explanatory variable in the analysis rather than being considered as a potential outcome of smoking and occupational

exposures. The mean FEV_1 of the coke oven workers, adjusted for age and height, was 130 ml lower than that of the other workers. The average effect of smoking on FEV was slightly greater than that of occupational exposures at -156 ml. There was a small effect of -32 ml of previous work in dusty jobs, principally coal mining. Bronchitis was more prevalent amongst the coke workers (24% vs 16%) and was associated with lower FEV. An analysis of covariance showed significant adverse effects of bronchitis (mean FEV_1 was 160ml lower in those with bronchitis); work on ovens (mean FEV_1 was 50ml lower in oven top workers); and smoking and previous dust exposure taken together (mean FEV_1 was 80ml lower). The ages of the subjects in the study were not stated and the study did not include any measures of exposure. No data are presented for FVC or for FEV_1/FVC to demonstrate whether the impairments of lung function identified were associated with airflow obstruction and COPD.

27. Chau et a (1992) is a French study of retired but surviving coke oven workers and miners, 12 years post retirement. The response rate was 67% (231/ 354). The average age of the subjects was 65yr and 91% were smokers or ex-smokers. Their average duration of work on coke ovens was 23 yr. 37 men had spent a substantial part of their working life employed as an underground coal miner. The subjects were assigned to one of six exposure categories based on job title. No effect of exposure was demonstrated. The mean FEV_1 was 2.46 L for oven workers (n= 30); 2.47 L for near-oven workers (n=67); and 2.37 for non-exposed workers (n=46). There was a significant effect of smoking on FEV equivalent to -8ml per pack-year for FEV and -6ml/pack-year for FVC.

28. Madison et al (1984) studied 3,799 male workers on 11 US coking plants. Workers were classified into six groups depending on their likely exposure to ‘noxious substances’ in their longest-held job. The job categories were supervisor/ oven bottom/ maintenance/ labourer/ side oven/ top oven worker. Mean FEV_1 data were presented by exposure category, smoking status, and ethnicity. Black workers had FEV_1 measurements which were approximately 500 ml lower than white workers and smokers had FEV_1 s that were approximately 180 ml lower than non-smokers. Within each subgroup there was a gradient of FEV_1 from supervisors to top oven workers of -240 to -400 ml. That was greater than the magnitude of the effects of smoking. A regression analysis of FEV/FVC < 70% showed statistically significant effects of smoking, age, job category and an age-job category interaction but the magnitudes of the effects were not reported. There were additional effects of sputum abnormalities which were assessed as part of the study.

29. Corhay et al (1998) compared 137 Belgian coke oven workers with 150 blast furnace workers. The primary purpose of the study was to investigate the prevalence of airway hyper-responsiveness, but some lung function data were presented. The coke oven workers were slightly older (42 vs 41 yr) and smoked

more (82% current/ex-smokers vs 78%). Their mean FEV_1 was 96.8% of the predicted value and the blast furnace workers' was 100% of predicted after an average of 16 years' work. [For men of average height a FEV of 3.2% below the predicted value is equivalent to -141ml or 9 ml/yr for each of 16 years work]. There was no clear excess of airflow obstruction with mean FEV/FVC 80.4% for the coke oven workers and 80.8 for the others.

30. Corhay et al (1991) published a similar study of 'pre-retired' coke workers in abstract form. The results were similar with coke workers' mean FEV_1 being 91% of predicted and blast furnace workers' 95% of predicted after an average of 26 years' work.

31. Wu et al (2002) reported a cross-sectional study of 834 Australian coke oven workers with at least one year of exposure. Their mean exposure was 190 g/m^3 BSM with the compulsory wearing of respiratory protective equipment with a nominal protection factor of 10 for oven top workers from the early 1980s. The primary analysis was of years spent on 'operational work' which involved the heaviest exposures. There was a relationship between years spent on operational work and FEV (-9 ml/yr) and FVC (-14 ml/yr). The estimated effects were greater than those for smoking (-3.4 ml/yr for FEV_1 and -2.0 ml/yr for FVC). In the case of smoking the effect on FEV was greater than the effect on FVC (as expected with COPD) whereas that was not the case in relation to occupational exposure, suggesting that the effect of exposure was not simply to cause COPD.

32. Wu et al (2004) then carried out a longitudinal study based on the same workforce. 580 workers with at least two lung function measures were followed up for an average of 6.8 years between 1978 and 1990. Regression analysis showed an excess rate of decline of FEV_1 of 0.5 ml/yr for each year spent on operational work and an excess decline of FVC of 0.7 ml/yr. The rates of decline were slightly greater when 111 subjects without detailed exposure histories were excluded from the analysis (0.8 ml/yr for FEV_1 and 1.0 ml/yr for FVC). Smoking was associated with an excess rate of decline of 0.6 ml/pk-yr for FEV and 0.5ml/pk-yr for FVC. This was of similar magnitude to the effect of operational work. As with the cross-sectional study, the effect of exposure on FVC appeared greater than the effect on FEV (which is atypical of COPD), but in the longitudinal study the magnitude of the effects appeared to have been only one tenth of those in the cross-sectional study.

33. Hu et al (2004) is a study of 712 Chinese coke oven workers based on two plants, and 211 control subjects who worked in a calibration equipment factory. The participation rate was 90%. Individuals' exposures were estimated from BSM measurements taken from the bottom, middle and tops of the ovens, and the duration of work in each area. Conditions in the two coke ovens differed, with the mean BSM concentrations on the top of plants being 744 and 190 g/m^3,

respectively. There was an exposure-response relationship with mean FEV_1 420ml lower in the highest exposure group and FVC 320ml lower compared with the control group. 30% of subjects in the high exposure group were considered to have COPD defined as FEV/FVC<70% compared with 7% in the control group (OR 5.80: 3.13 to 10.76). The effects were on a par with those of smoking (9% COPD in the never-smoking group and 36% in the heavy smokers (OR 5.26 :2.90 to 9.53). There was a suggestion of a multiplicative interaction between exposure and smoking with OR 58 (11-305) in the heaviest-exposed, heaviest-smoking subgroup but the data are consistent with an additive effect as is more generally seen in studies of occupational COPD. The overall prevalence of COPD was high (7% in the non-exposed group at average age 36) and there is an unexplained adjustment of the data that progressively decreases the FEV/FVC relative to the FEV and FVC with increasing exposure, and so increases the apparent prevalence of COPD. COPD severity was determined with reference to prediction equations derived from a reference population which had a different age and sex distribution from the study population.

Discussion

34. There is a body of evidence evaluating the risk of COPD in coke oven workers which derives from both mortality and from lung function studies. There are, however, weaknesses in the studies that limit their interpretation and their application to IIAC's criteria for prescription under the IIS.

35. Most of the mortality studies suggest an increased risk of death from chronic respiratory disease in coke plant or oven workers. The magnitude of the effect was modest with less than a doubling of risk. However, the nature of COPD is such that mortality studies have little ability to identify relationships between work and COPD and are likely to underestimate any effect of work. Most individuals with COPD die of some other condition and it is that condition which is recorded on death certificates. The mortality studies are therefore relatively uninformative and are not considered further.

36. There are five moderately large lung function studies which suggest an effect of coke oven work on lung function (Walker et al, Maddison et al, Wu et al 2002, Wu et al 2004, and Hu et al). Only one relatively small study (Chau et al 1992) failed to report an effect. It had only a modest (67%) response rate and as a study of retired, but surviving workers 12 years after retirement, it was prone to a survivor bias that had the potential to mask an effect of work. This study was also not considered further. For the other studies there were issues in relation to the nature of the lung function abnormalities and the magnitude of the effects of coke oven exposures which needed to be considered.

37. With regard to the nature of the lung function abnormalities, only two of the studies (Maddison et al, Hu et al) demonstrated a COPD-like effect with exposure-related

reductions in FEV which were greater than the reductions in FVC. A third smaller study (Corhay 1991) showed FEV/FVC to be marginally lower in coke oven workers, consistent with the effects of COPD.

38. Two related studies (Wu et al 2002 and 2004) demonstrated effects on lung function which were atypical of COPD with exposure-related reductions in FVC that were greater than the effects on FEV. The explanation for that was not clear but it suggests that some other process such as lung fibrosis contributed to the impairment of lung function. The effect of smoking in the same study was more typical of COPD with a greater effect on FEV than FVC. In one study, (Walker et al) only FEV_1 was reported and so the nature of the lung function abnormality is uncertain.

39. With regard to the magnitude of the effects, in all five studies which demonstrated adverse effects of coke oven exposures on lung function, these were of similar magnitude to the effects of cigarette smoking. In the two earlier studies (Walker et al, Maddison et al) the equivalence of effect was to typical coke oven exposures in the 1960s, 1970s, and early 1980s. In the later studies, the equivalence of effect appeared to have been to much lower-level exposures. In the Australian studies (Wu et al) the magnitude of the effect of exposure on lung function appeared much larger in the cross-sectional study than in the longitudinal study. These differences and other methodological and data inconsistencies made it difficult to draw firm conclusions about the relationship between the effects of smoking and coke oven emissions in the development of COPD.

40. Only one study quantified the risk of COPD in relation to any measure of exposure. The exposure measure used was BSM and none of the studies quantified coal dust which would have been present in coke works with the potential to contribute to COPD. The other studies either compared coke oven workers with other groups of workers or used job title as a surrogate for exposure.

Conclusion

41. For the Council to recommend a disease for prescription under the IIS, there should be epidemiological evidence that ideally is drawn from several independent studies and is sufficiently robust that further research at a later date would be unlikely to overturn it. Given the inconsistent evidence about the nature of the lung function abnormalities and the magnitude of the effects in the published studies and the paucity of exposure data on which to base attribution in individual cases, IIAC concluded that the current body of evidence relating COPD to occupational exposures in coke oven workers is not sufficient to recommend prescription.

Prevention

42. The Control of Substances Hazardous to Health Regulations 2002 (COSHH), (as amended), apply across the workplace and thus include work carried out in or around the vicinity of a coke oven. The Regulations require that work is not carried out if it is liable to expose any employees to any substance hazardous to health unless a suitable and sufficient assessment has been made of the risks created by the work and appropriate measures are taken to prevent exposure as far as is reasonably practicable. Where it is not reasonably practicable to prevent exposure by elimination or substitution with a safer substance or total enclosure, exposure must be adequately controlled by the use of appropriate work processes, systems and engineering controls and measures to control exposures at source. Suitable respiratory protective equipment should be used in addition to engineering measures where adequate control cannot be otherwise achieved. Those working in areas of the workplace where exposure is likely to happen, such as on or around the coke oven, should be informed of the hazards/risks and be provided with the appropriate training. Additionally, the COSHH regulations require employers to arrange appropriate health surveillance.

References

Bye T., Romundstad P., Rønneberg A., Hilt B. Health Survey of Former Workers in a Norwegian Coke Plant: Part 2. Cancer Incidence and Cause Specific Mortality. Occup. Environ. Med. 1998;55:622–626.

Chau N, Bertrand JP, Guenzi M, et al. Lung function in retired coke oven plant workers. Br J Ind Med 1992;49:316–25.

Chau N., Bertrand J., Mur J., Figueredo A., Patris A., Moulin J., Pham Q. Mortality in Retired Coke Oven Plant Workers. Br. J. Ind. Med. 1993;50:127–135.

Collings PL. Coke workers' mortality, a nine year follow-up in the British Steel Industry. Report prepared by the Statistics Branch, Institute of Occupational Medicine, Edinburgh, Scotland for the European Coal and Steel Community and the British Steel Corporation Edinburgh, 1978. (Report No TM/78/1.)

Corhay JL, Bury T, Louis R, et al. Bronchial responsiveness in active steelworkers. Eur Respir J 1998;11:272–7.

Corhay JL, Mariz S, Bury Th, Tulippe Ch, Delavignette JP, Radermecker MF. Bronchial hyperresponsiveness in healthy preretired coke-oven workers. Eur Respir J 1991; 4: 251

Crawford, J. O., Dixon, K., Miller, B. G., & Cherrie, J. W. (2014). A review of the effectiveness of respirators in reducing exposure to polycyclic aromatic hydrocarbons for coke oven workers. The Annals of Occupational Hygiene, 58(8), 943–954.

Davies GM. A mortality study of coke oven workers in two South Sales integrated steelworks. Br J Ind Med 1977;34: 291-7.

Doll R, Vessey MP, Beasley RWR, et al. Mortality of gasworkers. Final report of a prospective study. Br J Ind Med 1972;29:394-406

Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease. 2019.

Hu Y, Chen B, Yin Z, Jia L, Zhou Y, Jin T. Increased risk of chronic obstructive pulmonary diseases in coke oven workers: interaction between occupational exposure and smoking. Thorax. 2006;61(4):290–5.

Hurley JF, Archibald R, Collings PL, Fanning DM, Jacobsen M, Steele RC. The mortality of coke workers in Britain. Am J Ind Med 1983;4:691-704.

Jacobsen M, Hurley JF. Mortality at British factories engaged in coal carbonisation and tar distillation. Institute of Occupational Medicine. Research Report TM/77/13 1977.

Jones and Others v the Secretary of State for Energy and Climate Change and Others. London, Royal Courts of Justice 2012. Case No: Case No: HQ09X03547

Lloyd JW. Long term mortality study of steelworkers. V. Respiratory cancer in coke plant workers. J Occup Med 1971;13:53-68.

Madison R, Afifi AA, Mittman C. Respiratory impairment in coke oven workers: relationship to work exposure and bronchial inflammation detected by sputum cytology. J Chron Dis 1984;37:167–76.

Miller, B. G., Doust, E., Cherrie, J. W., & Hurley, J. F. (2013). Lung cancer mortality and exposure to polycyclic aromatic hydrocarbons in British coke oven workers. *BMC Public Health*, *13*(1), 962–1.

Pearce and Others v the Secretary of State for Business, Energy and Industrial Strategy and Others. London, Royal Courts of Justice 2018. Case No: HQ15A03468.

Redmond CK, Ciocco A, Lloyd JW, et al. Long-term mortality study of steelworkers. VI. Mortality from malignant neoplasms among coke oven workers. J Occup Med 1972;14:621–9.

Redmond CK, Strobino BR, Cypess RH. Cancer experience among coke by-product worker. Ann N Y Acad Sci 1976;271:102–15.

Swaen GMH, Slangen JJM, Volovics A, Hayes RB, Scheffers T, Sturmans F. Mortality of coke plant workers in the Netherlands. Br J Ind Med 1991;48:130-5.

Walker DD, Archibald RM, Attfield MD. Bronchitis in men employed in the coke industry. Br J Ind Med 1971;28:358–63.

Wu J, Kreis IA, Griffiths D, et al. Cross sectional study on lung function of coke oven workers: a lung function surveillance system from 1978 to 1990. Occup Environ Med 2002;59:816–23

Wu J, Kreis IA, Griffiths D, et al. Respiratory symptoms and lung function of coke oven workers: a lung function surveillance system from 1990–2000. J Occup Environ Med, 2004;46:906–15.

Glossary

Types of study

Case control study: A study which compares people who have a given disease (cases) with people who do not (non-cases, also called controls) in terms of exposure to one or more risk factors of interest. Have cases been exposed more than non-cases? The outcome is expressed as an **Odds Ratio**, a form of **Relative Risk.**

Cohort study: A study which follows those with an exposure of interest (usually over a period of years), and compares their incidence of disease or mortality with a second group, who are unexposed or exposed at a lower level. Is the incidence rate higher in the exposed/more exposed workers than the unexposed/less exposed group? Sometimes the cohort is followed forwards in time ('prospective' cohort study), but sometimes the experience of the cohort is reconstructed from historic records ('retrospective' or 'historic' cohort study). The ratio of risk in the exposed relative to the unexposed can be expressed in various ways, such as a **Relative Risk**, or **Standardised Mortality Ratio.**

Cross-sectional study: A study which classified people at a point in time as having a given disease (or characteristic) or not (controls), and then compares them in terms of exposure to one or more risk factors of interest. Is disease more frequent in those with exposure than in those without? The outcome can be expressed as an **Odds Ratio**, **Prevalence Ratio** or **Relative Risk**.

Measures of association

Statistical significance and P values: Statistical significance refers to the probability that a result as large as that observed, or more extreme still, could have arisen simply by chance. The smaller the probability, the less likely it is that the findings arise by chance and the more likely they are to be 'true'. A 'statistically significant' result is one for which the chance alone probability is suitably small, as judged by reference to a pre-defined cut-point. (Conventionally, this is often less than 5% ($P<0.05$)).

Regression analysis: a set of statistical processes for estimating the relationships between a dependent variable (often called the 'outcome variable') and one or more independent variables (often called 'predictors', 'covariates', or 'features').

Analysis of covariance: a method for comparing sets of data that consist of two variables (treatment and effect, with the effect variable being called the variate), when a third variable (called the covariate) exists that can be measured but not controlled and that has a definite effect on the variable of interest.

Relative Risk (RR): A measure of the strength of association between exposure and disease. RR is the ratio of the risk of disease in one group to that in another. Often the first group is exposed and the second unexposed or less exposed. *A value greater than 1.0 indicates a positive association between exposure and disease*. (This may be causal, or have other explanations, such as bias, chance or **confounding**.)

Odds Ratio (OR): A measure of the strength of association between exposure and disease. It is the odds of exposure in those with disease relative to the odds of exposure in those without disease, expressed as a ratio. For rare exposures, odds

and risks are numerically very similar, so the OR can be thought of as a **Relative Risk**. A value greater than 1.0 indicates a positive association between exposure and disease. (This may be causal, or have other explanations, such as bias, chance or **confounding**.)

Standardised Mortality Ratio (SMR): A measure of the strength of association between exposure and mortality; a form of **Relative Risk** in which the outcome is death. The SMR is the ratio of the number of deaths (due to a given disease arising from exposure to a specific risk factor) that occurs within the study population to the number of deaths that would be expected if the study population had the same rate of mortality as the general population (the standard).
By convention, SMRs (and **proportional mortality ratios**, as described below) are usually multiplied by 100. Thus, an SMR (or PMR) of 200 corresponds to a RR of 2.0. For ease of understanding in this report, SMRs (or PMRs) are quoted as if RRs, and are not multiplied by 100. Thus, a value greater than 1.0 indicates a positive association between exposure and disease. (This may be causal, or have other explanations, such as **bias**, chance or **confounding**.)

Proportional Mortality Ratio (PMR): A PMR is the proportion of observed deaths from a given cause in a given population divided by the proportion of deaths from that cause expected (in a standard population). The value is often expressed on an age-specific basis or after age adjustment. It is a form of **Relative Risk**.

Other epidemiological terms

Confidence Interval (CI): The **Relative Risk** reported in a study is only an *estimate* of the true value in the underlying population; a different sample may give a somewhat different estimate. The CI defines a plausible range in which the true population value lies, given the extent of statistical uncertainty in the data. The commonly chosen 95% CIs give a range in which there is a 95% chance that the true value will be found (in the absence of bias and confounding). *Small studies generate much uncertainty and a wide range, whereas very large studies provide a narrower band of compatible values*.

Confounding: Arises when the association between exposure and disease is explained in whole or part by a third factor (confounder), itself a cause of the disease, that occurs to a different extent in the groups being compared.

For example, smoking is a cause of lung cancer and tends to be more common in blue-collar jobs. An apparent association between work in the job and lung cancer could arise because of differences in smoking habit, rather than a noxious work agent.

Studies often try to mitigate the effects of (‘control for’) confounding in various ways such as: restriction (e.g. only studying smokers); matching (analyzing groups with similar smoking habits); stratification (considering the findings separately for smokers and non-smokers); and mathematical modelling (statistical adjustment).

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