Department for Work and Pensions

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Social Security Administration Act 1992

Nasopharyngeal cancer due to exposure to wood dust

Report by the Industrial Injuries Advisory Council in accordance with Section 171 of the Social Security Administration Act 1992 considering prescription for nasopharyngeal cancer due to exposure to wood dust.

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

July 2007

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Industrial Injuries Advisory Council

Secretary of State for Work and Pensions

Dear Secretary of State,

Review of Nasopharyngeal Cancer Due to Wood Dust

This report details the Council's consideration of prescription for nasopharyngeal cancer in relation to wood dust exposure. Concern about this hazard was brought to our attention in 2006 by a former member of the Council. An association between cancer of the nasal sinuses and wood dust is already well-established, and carcinoma of the nasal cavity and associated air sinuses (anatomically distinct sites from the nasopharynx) is currently included in the list of prescribed diseases (PD D6) for which Industrial Injuries Disablement Benefit is payable.

The nasopharynx, which connects the nose with the back of the mouth, is a site where inhaled wood dust can deposit. We have found consistent evidence, across studies from different industries and settings, of an excess risk of cancer of the nasopharynx in workers exposed to wood dust. The link with cancer of the nasopharynx is not clearly limited to particular types of wood or processes. Nor does the evidence suggest that risks are confined to one particular histological sub-type of tumour. Nasopharyngeal cancer is unlikely to occur until exposure to wood dust has exceeded 10 years in aggregate and the disease has a latency of at least 10 years from first exposure.

The Council recommends that cancer of the nasopharynx be prescribed for occupations involving the processing, manufacture or repair of wood or wooden goods, for a period of at least 10 years in aggregate.

Yours sincerely

Professor A J Newman Taylor

Chairman
July 2007

Summary

1. The Industrial Injuries Advisory Council's (IIAC) attention was drawn by a former Council member to the possibility of an association between wood dust exposure and cancer of the nasopharynx. Carcinoma of the nasal cavity and associated air sinuses is already prescribed in occupations involving wood dust exposure. The nasopharynx is also a site of contact when wood dust is inhaled and a number of studies on this subject have been carried out in recent years. The Council therefore reviewed the evidence for an association between nasopharyngeal cancer (NPC) and exposure to wood dust and the case for prescription.

2. A number of cohort and case-control studies were identified together with review papers produced by the World Health Organisation (WHO) International Agency for Research on Cancer, the European Union (EU) Scientific Committee on Occupational Exposure Limits (SCOEL) and the Medical Research Council (MRC) Epidemiology Resource Centre at the University of Southampton.

3. Because NPC is a rare disease the findings of cohort studies were limited by the difficulty of collecting enough cases to mount a statistically adequate investigation.

4. The most important cohort study was a large pooled analysis of five studies (Demers *et a*l. 1995) which included a cohort of British furniture makers. The results of this study indicated a statistically significant excess of NPC (Standardised Mortality Ratio (SMR) 2.4) in wood workers. Other cohort studies had less power to address the study question and their results provided only limited support for these findings.

5. The majority of studies were case-control studies. Although the results were not entirely consistent, most were indicative of a link between exposure to wood dust and NPC. The three separate review papers also reached this conclusion.

6. Studies which considered exposure duration or disease latency indicated that NPC was unlikely to occur until exposure to wood dust had exceeded 10 years and that the disease had a latency of at least 10 years from first exposure.

7. The Council also examined studies which considered the possible role of wood treatment agents, chlorophenols and formaldehyde, in the genesis of NPC. It was concluded that, although there is some evidence to suggest that both agents may be implicated in the development of NPC, this does not preclude an independent effect of wood dust exposure. Thus, the association with NPC is not limited to exposure to treated wood.

8. The evidence did not suggest that any specific occupation, activity, or type of wood was implicated more strongly than others.

9. Although NPC exists in several histopathological types the evidence did not suggest that risks are confined to a particular histological subtype of tumour.

10. Based on a review of the evidence, therefore, IIAC recommends the prescription of cancer of the nasopharynx in those who have been in occupations involving the processing, manufacture or repair of wood or wooden goods, for a period of at least 10 years in aggregate.

Introduction

11. The Council's attention was drawn by a former Council member to the possible association between wood dust exposure and cancer of the nasopharynx. The association between wood dust exposure and cancer of the nasal sinuses is already well-established and carcinoma of the nasal cavity and associated air sinuses is currently included in the list of prescribed diseases (PD D6) under the Industrial Injuries Disablement Benefit (IIDB) Scheme (see Appendix 1).

12. Since the nasopharynx is also a site of contact when wood dust is inhaled it is biologically plausible that wood dust exposure may also be associated with an increased risk of nasopharyngeal cancer (NPC). It was noted that a number of studies on this subject have been carried out in recent years and that a growing body of evidence now exists.

13. The Council has therefore reviewed the evidence for an association between NPC and exposure to wood dust and the case for prescription.

The Industrial Injuries Disablement Benefit Scheme

14. The Industrial Injuries Advisory Council (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 major part 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.

15. The IIDB Scheme provides a benefit that can be paid to an employed earner because of an industrial accident or Prescribed Disease (PD).

The legal requirements for prescription

16. 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.

17. 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.

18. In seeking to address the question of prescription for any particular condition, the Council first looks for a workable definition of the disease. Then it 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 available scientific evidence. 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

19. 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 dermatitis is caused by his/her 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

20. 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. 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 cannot 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 the Council’s decision on prescription.

Nasopharyngeal Cancer

21. The nasopharynx is an airspace lying at the back of the nose and above the soft part of the palate (roof of the mouth). It connects the nose to the back of the mouth (oropharynx), allowing breathing through the nose and the swallowing of mucous produced by the lining membranes of the nose.

22. Cancer of the nasopharynx is a rare disease with a low background rate in the general population. The disease exists in several histopathological types (squamous cell carcinomas, adenocarcinomas and undifferentiated tumours). Some 230 new cases overall are diagnosed in the UK each year. NPC can occur at any age, but is more likely to be seen in people aged between 40 and 60 years, and affects more men than women. There is marked geographical variation in frequency and a significantly elevated risk has been observed amongst the populations of South East Asia.

23. A number of factors, in addition to wood dust exposure, have been suggested as contributory or causal factors. In some areas of the world, such as China and North Africa, dietary factors (such as the cooking of salt-cured fish and meat, resulting in the release of nitrosamines) are thought to increase a person's risk of developing the disease. Other factors which have been shown to be associated with NPC are prior exposure to the Epstein-Barr virus, a prior history of other ear, nose and throat disease, smoking, alcohol consumption and exposure to certain chemical compounds.

24. It is clear, therefore, that NPC cannot be considered a uniquely occupational disease and that the case for prescription requires robust research evidence indicating a doubling of risk at certain levels of exposure to wood dust.

25. Wood is normally classified as either hardwood (usually from deciduous trees such as oak and beech, and from certain tropical species such as mahogany and teak) or softwood (usually from conifers such as pine and cedar). Hardwood and softwood may have different roles in the genesis of cancer. In particular, hardwood dusts are more strongly implicated in the development of sino-nasal cancer than are softwood dusts. Wood dust also varies in terms of particle size, (depending largely on the nature of operation being carried out rather than on the type of wood being worked) and such factors may modify disease risk. Questions arise, therefore, as to whether any association of NPC with wood dust is restricted to certain types of wood or patterns of woodworking, or to particular hisotopathological categories of disease.

26. Research findings also suggest the possibility that formaldehyde and chlorophenols may be linked to the development of NPC. Both substances are commonly used as wood treatment agents, and so may be concomitant exposures in those exposed to wood dust. As well as assessing the overall relation between NPC and woodworking, the Council considered whether elevated risks were restricted to those exposed to wood treated with these agents.

Method of investigation

27. A call for evidence and information was made via the Council web site and a literature search was undertaken by IIAC's scientific secretariat.

28. A number of original studies which have directly considered the association between wood dust exposure and NPC were identified. In addition the following scientific reviews containing relevant information were considered:

a) Two monographs published by the World Health Organisation (WHO) International Agency for Research on Cancer (IARC) (i) Volume 25, 1981: *Wood, Leather and some Associated Industries*. (ii) *Volume 62, 1995: Wood Dust and Formaldehyde*.

b) A review produced by the European Union (EU) Scientific Committee on Occupational Exposure Limits (SCOEL) *Risk Assessment for Wood Dust* 2003.

c) A legal report by Professor David Coggon, Medical Research Council (MRC) Epidemiology Resource Centre, University of Southampton, submitted as evidence to the Council.

29. Further papers relating to the possible role of the wood treatment agents, formaldehyde and chlorophenols were also considered, including a further monograph by IARC, Volume 88, 2006, *Formaldehyde*.

30. This body of evidence was subsequently reviewed by members of the IIAC Research Working Group.

Consideration of the evidence

31. Two general approaches have been adopted in research investigations. In cohort studies, people with exposure to wood dust have been identified, and their subsequent incidence of disease or mortality compared with that of an unexposed control population, allowing a direct estimate of relative risk (RR) or relative mortality (Standardised Mortality Ratio, SMR). In case-control studies, patients who have developed NPC have been identified, and their past exposure to suspected causal agents including wood dust compared with that of controls who do not have the disease. From this, an odds ratio (OR) can be estimated.

32. Cohort studies have the advantage that the subjects studied can be chosen on the basis of established comparatively high levels of exposure to wood dust; but findings are limited by the rarity of NPC and the difficulty of collecting enough cases to mount a statistically adequate investigation. Case-control studies often begin with the assembly of NPC cases through hospital services and tend, instead, to be limited by less certainty over exposures, assessed in retrospect and often at a lower level in the general population than in special occupational cohorts.

33. Studies of both types are susceptible to a number of other potential sources of bias, as well as variations in disease and exposure definition. Thus, some reports carry less information than others in a challenging area of research inquiry.

34. The most important cohort findings in the field derive from the pooled analysis of the data from five studies carried out by Demers *et al*. (1995). Pooling of the data enables an analysis which is based on a greater number of exposed subjects and a larger number of cases. These studies produced a combined cohort of 28,704 furniture makers and woodworkers, including a cohort of British furniture makers studied between 1941 and 1991. The overall analysis indicated a statistically significant excess of NPC (SMR 2.4, 95% CI[1] 1.6-5.6, based on 9 observed cases versus 3.8 expected[2]). For furniture workers separately the SMR was 2.9 (CI 1.2-5.9, based on 7 observed cases, 2.4 expected) and for plywood workers 4.6 (CI 0.6-16.4, based on 2 observed cases, 0.4 expected). Cancer often has a long latency (interval between exposure and development of the disease). It is noteworthy that seven of the nine cases occurred in those whose first exposure had occurred more than 30 years previously, while for the remaining two cases first exposure had occurred between 10 and 19 years previously.

35. Other cohort studies have had less statistical power to address the study question and their results provide only limited support for these findings. An incidence study (Malker *et al*. 1990) carried out in Sweden reported a statistically significant increased risk of NPC in fibreboard workers, Standardised Incidence Ratio (SIR) 3.9, based on 4 cases, but no increased risk in either furniture makers (SIR 0.8 based on 3 cases) or plywood workers (no cases). A mortality study in Hong Kong (Lam and Tan 1984) reported a statistically non-significant risk for cabinet makers, although based on only 2 cases.

36. Two further cohort studies were similarly unable to identify a significantly increased risk. Stellman *et al*. (1998) reported an RR of 1.44 (CI 0.19-10.9) for wood dust exposure, but based on only one case over the six years of study. Innos *et al*. (2000) found no cases among a cohort of furniture makers.

37. Because NPC is uncommon, most studies of NPC have had a case-control design. Their results, although not entirely consistent, are suggestive of a link between exposure to wood dust and NPC. The majority of studies indicate an increased risk, although there is variation in terms of the particular occupations or type of work involved. For example, Armstrong *et al*. (2000) investigated a range of possible contributory factors in Malaysian Chinese patients and found that wood dust exposure was a significant risk factor (OR 2.36, CI 1.33-4.19). An earlier study in a similar population (Armstrong *et al*. 1983) identified an RR of 2.2 (p=0 .08), for wood dust and sawdust exposure.

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[1] A 95% confidence interval (CI) represents a plausible range in which the true population value lies, given the extent of statistical uncertainty in the data; a lower confidence limit >1 suggests a positive association that is unlikely to arise simply by chance (less than 1 chance in 20).

[2] Expected on the basis of background rates by age, sex and sometimes other characteristics.

38. Zheng *et al*. (1992), studying a Shanghai population, reported a significantly increased risk for pattern makers and cutters (SIR 2.8) based on 6 cases, although no increased risk in wood product makers. Ng (1986), in Hong Kong, focussing on sino-nasal cancer and using cases of NPC and cases of other cancers as two referent groups, reported that five of the cases of NPC were furniture makers and wood workers, compared with one in the other cancer referent group. The highest risk (OR 8.0, CI 2.3-28.2) was reported by Sriamporn *et al*. (1992) in Thailand, for woodcutters working in agriculture; non-agricultural woodcutters were also at increased risk (OR 4.1, CI 0.8-22.1).

39. Outside South East Asia, Kawachi *et al*. (1989) in New Zealand, investigated associations between different occupations and various cancers including NPC. An increased risk was found for wood workers as a whole (OR 2.46, CI 0.86-6.60) and for carpenters, (OR 2.51, CI 0.62-8.53), although neither of these estimates were statistically significant at the 5% level[3]. By contrast a significantly increased risk was found for carpenters and loggers (OR 6.02, CI 1.01-28.41).

40. Three studies report no significantly increased risk associated with woodworking and wood dust exposure. Hardell *et al*. (1982) found a RR of 1.1 for woodworkers, which included sawmill workers, carpenters and cabinet makers, and 1.5 for cabinet makers alone, based on cases reported to the Swedish Cancer registry between 1970 and 1979. For wood dust exposure, Olsen *et al*. (1984) in Denmark, reported a RR of 0.4 (CI 0.2-1.0). Vaughan *et al*. (2000), using cases derived from US Cancer Registries in five states, reported an OR of 1.3 (CI 0.6-2.6), although there was some suggestion in these data of a significantly increased risk at higher levels of cumulative exposure.

41. In an earlier study, Vaughan (1989) compiled cases from a smaller population area. Although a non-significant risk of NPC for those with previous employment as a carpenter was identified (OR 3.3, CI 0.8-13.2), the risk for carpenters employed in the construction industry was significantly elevated (OR 4.8, CI 1.2-19.4). Furthermore, the risk increased markedly in both groups when an induction period of more than 15 years was taken into account (for carpenters OR 4.5 and for construction carpenters OR 6.8).

42. A number of researchers have attempted to estimate the duration of exposure required for the development of NPC and to take into account the latency period of the disease. The results of these studies tend to strengthen the case for an association between NPC and wood dust exposure in that they indicate a stronger association with NPC with a longer duration of wood dust exposure and a higher risk assuming a disease latency period of 10 years or more.

[3] Indicating a less than 1 in 20 probability that the findings arose by chance (assuming no true effect of wood dust on risk of NPC).

43. Hildesheim *et al*. (2001) found that the overall adjusted RR for workers exposed to wood dust (based on occupational category) was 1.7 (CI 1.0-3.0) and that this rose to 2.4 (CI 1.1-5.0) in those with more than 10 years exposure. In addition, the relative risk for those exposed before the age of 25 years was 2.3 (CI 1.2-4.4), as opposed to only 1.1 (CI 0.47-2.5) for those first exposed after this age. Compared with the unexposed, those exposed before the age of 25 for more than 10 years had an RR of 2.8 (CI 1.2-6.9). There was also a significant positive trend for years of exposure (p=0.03), where the first 10 years of exposure before diagnosis was excluded. In this study an attempt was made to classify the intensity of exposure. In an analysis which included only those judged to have had a high intensity exposure for at least 10 years the relative risk was 3.4 (CI 1.2-9.4).

44. West *et al*. (1993) investigating risk factors for NPC in the Philippines reported a RR of 3.6 (CI 1.8-7.2) for those whose first exposure occurred less than 35 years ago and 5.5 (CI-1.9-16) for those whose first exposure occurred more than 35 years previously. Unfortunately, exposure in this case was to "dust and/or exhaust fumes" thus limiting the relevance of the data. However, Yang *et al*. (2005) reported a significantly increased risk with wood dust exposure of more than 25 years (OR 2.29 CI 1.04-5.07), while for those with less than 25 years exposure the OR was reduced (OR 0.54, CI 0.22-1.36).

45. Vaughan's 1989 study reported an OR of 1.2 for carpenters employed for less than 10 years, rising significantly to 8.8 for those employed for more than 10 years. When a 15 year latency period was taken into account the ORs were 1.6 and 12.4 respectively. In an extension of this study (Vaughan and Davis 1991), which included data for a further five years, an OR of 4.2 (CI 0.4-26.6) was reported for woodworking of at least 10 years, taking place at least 15 years before diagnosis. All cases had worked as carpenters. (A table listing the occupations at risk of NPC and the evidence relating to them can be found in Appendix 2.)

46. The range of different occupations covered by these studies makes it difficult to identify particular wood-related occupations which are at risk. Furthermore, it is not possible from the data to determine whether the risk is specifically associated with a particular type of wood (hardwood or softwood), since in many studies wood type was either not specified or the occupations studied suggest potential exposure to both types. In a recent report *Risk Assessment for Wood Dust* the EU SCOEL carried out a detailed review of the evidence in relation to this question and concluded that, given the currently available data, it did not seem pertinent to distinguish between the two types. They reached similar conclusions regarding the particle size generated by different woodworking operations, noting that most epidemiological studies have not assessed exposure-response relationships using particle size measurements.

47. The inclusion of different histopathological types of tumour in different studies similarly makes it difficult to distinguish a particular type of tumour which may be associated with wood dust exposure. Although the majority of tumours reported in the various studies were squamous cell carcinomas, a number of other histopathological types were also included in several of the investigations.

48. The relative importance of concurrent exposure to wood treatment agents, (chlorophenols and formaldehyde), has been investigated in a few studies. A study conducted amongst US saw mill workers (Hertzman *et al*. 1997) found no increased risk of NPC (SIR 0.34, CI 0.06-1.06) associated with chlorophenol. However, results of more recent studies are more strongly suggestive of a role for chlorophenol exposure. For example, Zhu *et al*. (2002) reported a significantly elevated risk (OR 2.2, CI 1.1-4.3) and Mirabelli *et al*. (2000) reported an elevated risk which increased with duration of exposure. The OR for more than 10 years of exposure to chlorophenol was 5.68, (CI 1.72-16.1). In both these studies findings were not significantly altered by controlling for the effects of wood dust exposure, but the difficulty of separating the effects of the two exposures was noted. An independent effect of wood dust exposure is not therefore precluded. The results of the study by Hardell *et al*. (1982) suggest a possible interaction between wood dust and chlorophenol exposure, with a RR for nasal cancer and NPC together of 8.4 in woodworkers exposed to chlorophenols, versus 2.7 for other types of workers similarly exposed to these substances.

49. The association between NPC and formaldehyde exposure is less well supported. In the study by Vaughan *et al*. (2000) a significant increasing risk was identified with increasing duration of exposure to formaldehyde and the non-significant OR of 1.3 for wood dust exposure was reduced to 1.1 following adjustment for formaldehyde exposure. However, West *et al*. (1993) identified a significant association between NPC and formaldehyde exposure in Filipino workers exposed to wood dust (RR 4.0).

50. Studies which have focussed specifically on formaldehyde producers or users (i.e. in workers not exposed to wood dust) have similarly produced conflicting results, as exemplified in two recent studies. Coggon *et al*. (2003) found only one death from NPC (2 expected) in a long-term follow-up of a cohort of highly exposed British formaldehyde producers and users. By contrast, Hauptmann *et al*. (2004) reported a significant increase in deaths from NPC in US workers employed in formaldehyde production and use, (SMR 2.10, CI 1.05-4.21), noting that none of the eight cases had exposure to wood dust.

51. A recent review by IARC (2006) of the association between formaldehyde and different cancers placed strong emphasis on the results of Hauptmann *et al*. and concluded that there was sufficient evidence that formaldehyde exposure was a cause of NPC. However, Marsh *et al*. (2007) subsequently challenged IARC's conclusions arguing that re-analysis of certain subsets of

Hauptmann's data cast doubt on this conclusion. Overall, therefore, there would appear to be suggestive, but not conclusive evidence of an association between formaldehyde and NPC and, for present purposes, the strength of this association appears insufficient to suggest a doubling of risk in its own right or to rule out an independent effect of wood dust exposure.

Conclusions

52. There is a relatively large literature on wood dust exposure and NPC and the results of different studies, although not entirely consistent, are suggestive of an association. Some of the inconsistencies in the data may be explained by the low incidence of the disease and the difficulty of designing studies with sufficient power to identify an increased risk in a particular group. In addition, it is clear that a range of non-occupational factors (environmental, lifestyle and possibly genetic) have also been associated with the disease and account needs to be taken of confounding when assessing the role of wood dust exposure.

53. Having said this, a doubling of risk has been demonstrated in the majority of studies involving woodworking occupations.

54. There is a body of evidence to suggest that concurrent exposure to wood treatment agents, notably chlorophenols and possibly also formaldehyde, has a role to play in the development of NPC. However, this does not preclude an independent effect of wood dust exposure, or the possibility of an interactive effect between these substances and wood dust. Thus, the risk of NPC does not appear to be restricted to those exposed to treated wood products.

55. The results of those studies which have investigated the relationship between duration of exposure to wood dust and NPC suggest that a doubling of risk is unlikely to occur until exposure duration has exceeded 10 years.

56. Similarly, studies which have analysed risks according to different assumed latencies of disease point to a latency period of at least 10 years. With sufficient allowance for exposure duration and latency, associations with NPC have been more clear-cut.

57. No specific occupation, woodworking activities, or type of wood is implicated more clearly than others. Nor does the evidence suggest that risks are confined to one particular histological subtype of tumour.

Recommendations

58. IIAC recommends prescription for cancer of the nasopharynx in those who have been in occupations involving the processing, manufacture or repair of wood or wooden goods, for a period of at least 10 years in aggregate (see Appendix 3; PD D13).

Prevention

59. Cancer and other respiratory diseases caused by wood dust can be prevented by ensuring workers do not inhale the dust either by preventing the dust entering the workers' breathing zone, or, if that cannot be achieved, by the use of personal protective equipment.

60. The Control of Substances Hazardous to Health Regulations 2002 (COSHH) apply to work with hazardous substances such as wood dust. Hardwood dusts fall within the definition of a "carcinogen".

61. The COSHH 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 to the substance as far as is reasonably practicable.

62. Where it is not reasonably practicable to prevent exposure to wood dust, the levels of dust in the air must be adequately controlled by the use of appropriate work processes, systems and engineering controls and measures, including ventilation systems, to control dust at source. Suitable respiratory protective equipment may be used in addition, where adequate control cannot otherwise be achieved.

Appendix 1: Prescription for PD D6

Disease number	Name of disease or injury	Type of job
D6	Carcinoma of the nasal cavity or associated air sinuses (nasal carcinoma)	a) Attendance for work in or about a building where wooden goods are manufactured or repaired; or b) attendance for work in a building used for the manufacture of footwear or components of footwear made wholly or partly of leather or fibreboard; or c) attendance at work at a place used wholly or mainly for the repair of footwear made wholly or partly of leather or fibreboard.

Appendix 2: List of occupations at risk of NPC and the evidence relating to them.

Occupational exposure	Risk: SMR, SIR, OR, RR (Probability: CI or p)	Reference
Furniture makers	SMR 2.9 (CI 1.2-5.9)	Demers *et al*. (1995)
Plywood workers	SMR 4.6 (CI 0.6-16.4)	Demers *et al*. (1995)
Fibre board workers	SIR 3.9 (p<0.05)	Malker *et al*. (1990)
Pattern makers and cutters	SIR 2.8 p<0.05)	Zheng *et al*. (1992)
Wood cutters in agriculture	OR 8.0 (CI 2.3-28.2)	Sriamporn *et al*. (1992)
Wood cutters, non-agricultural	OR 4.1 (CI 0.8-22.1)	Sriamporn *et al*. (1992)
Woodworkers, various	OR 2.46 (CI 0.86-6.60)	Kawachi *et al*. (1989)
Carpenters	OR 2.51 (CI 0.62-8.53)	Kawachi *et al*. (1989)
Carpenters and loggers	OR 6.02 (CI 1.01-28.41)	Kawachi *et al*. (1989)
Construction carpenters	OR 4.8 (CI 1.2-19.4)	Vaughan (1989)
Carpenters, taking account of 15 years latency	OR 4.5 (CI 1.1-18.7)	Vaughan (1989)
Carpenters > 10 years exposure, taking account of 15 years latency	OR 4.2 (CI 0.4-26.6)	Vaughan (1989)
Construction carpenters, taking account of 15 years latency	OR 6.8 (CI 1.6-28.2)	Vaughan (1989)
Wood dust exposure	OR 2.36 (CI 1.33-4.19)	Armstrong *et al*. (2000)
Wood dust exposure >10 years (2001)	RR 2.4 (CI 1.1-5.0)	Hildesheim *et al*.
Wood dust exposure >25 years	OR 2.29 (CI 1.04-5.07)	Yang *et al*. (2005)
Wood dust and saw dust exposure	OR 2.2 (p 0.08)	Armstrong *et al*. (1983)

Abbreviations used in the table: SMR, standard mortality ratio; SIR, standard incidence ratio; OR, odds ratio; RR, relative risk; CI, confidence interval; p, probability.

Appendix 3: Recommended prescription for PD D13

Disease number	Name of disease or injury	Type of job
D13	Carcinoma of the nasopharynx	Work involving the processing, manufacture or repair of wood or wooden goods, for a period of at least 10 years in aggregate

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