

**ARSENIC, METALS, FIBRES,  
AND DUSTS**

VOLUME 100 C  
A REVIEW OF HUMAN CARCINOGENS

This publication represents the views and expert  
opinions of an IARC Working Group on the  
Evaluation of Carcinogenic Risks to Humans,  
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# WOOD DUST

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Wood dust was considered by a previous IARC Working Group in 1994 ([IARC, 1995](#)), although wood-related occupations (i.e. Furniture and Cabinet-making) had been considered by IARC Working Groups earlier, in 1980 and 1987 ([IARC, 1981, 1987](#)). Since that time, new data have become available, these have been incorporated in the *Monograph*, and taken into consideration in the present evaluation.

## 1. Exposure Data

### 1.1 Identification, chemical, and physical properties of the agent

Wood dust, generated in the processing of wood for a wide range of uses, is a complex substance. Its composition varies considerably according to the species of tree being processed. Wood dust is composed mainly of cellulose (approximately 40–50%), polyoses, lignin, and a large and variable number of substances of lower relative molecular mass which may significantly affect the properties of the wood. These include non-polar organic extractives (fatty acids, resin acids, waxes, alcohols, terpenes, sterols, steryl esters, and glycerides), polar organic extractives (tannins, flavonoids, quinones, and lignans) and water-soluble extractives (carbohydrates, alkaloids, proteins, and inorganic material) ([IARC, 1995](#)).

Trees are characterized botanically as gymnosperms (principally conifers, generally referred to as ‘softwoods’), and angiosperms (principally deciduous trees, generally referred to as ‘hardwoods’). Softwood and hardwood are

not botanical concepts, referring to the species of tree and not directly describing the hardness of wood. Out of 12000 different species of trees, only about 800 are coniferous or softwoods, but roughly two-thirds of the wood used commercially worldwide belongs to the group of softwoods. Hardwoods tend to be somewhat more dense, and have a higher content of polar extractives than softwoods ([IARC, 1995](#)). For a comparison of softwoods and hardwoods, see [Table 1.1](#).

For detailed descriptions of the classification and nomenclature, anatomical features, cell-wall structures, distribution of components of wood, and chemical components of wood, see the previous *IARC Monograph* ([IARC, 1995](#)), [Nimz et al. \(2005\)](#), and [Kretschmann et al. \(2007\)](#).

### 1.2 Occupational exposure

The wood species used in wood-related industries vary greatly by region and by type of product. Both hardwoods and softwoods (either domestically grown or imported) are used in the manufacture of furniture. Logging, sawmills, plywood, and particle-board manufacture generally involve the use of trees grown locally ([IARC,](#)

**Table 1.1 Comparison of softwoods and hardwoods**

Characteristic	Gymnosperms/conifers/softwoods	Angiosperms/deciduous wood/hardwoods
Density (g/cm <sup>3</sup> )	White (silver) fir: mean, 0.41 (0.32–0.71) European spruce: mean, 0.43 (0.30–0.64) Scots pine: mean, 0.49 (0.30–0.86)	European beech 0.68 (0.49–0.88) European oak 0.65 (0.39–0.93)
Fibres	Long (1.4–4.4 mm)	Short (0.2–2.4 mm)
Cell type	One (tracheids)	Various
Cellulose	~40–50%	~40–50%
Unit	β-D-Glucose	β-D-Glucose
Fibre pulp	Long	Short
Polyoses	~15–30%	~25–35%
Unit	More mannose More galactose	More xylose
Lignin	~25–35%	~20–30%
Unit	Mainly guaiacyl	Mainly syringyl or guaiacyl
Methoxy group content	~15%	~20%
Extractive content		
Non-polar (e.g. terpenes)	High	Low
Polar (e.g. tannins)	Low	High

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[1995](#)). For detailed descriptions of historical exposures to wood dust and other agents in the workplace, see the previous *IARC Monograph* ([IARC, 1995](#)).

### 1.2.1 Extent of occupational exposure

[Kauppinen et al. \(2006\)](#) used nearly 36000 exposure measurements to estimate the occupational exposure to inhalable wood dust by country, industry, the level of exposure and type of wood dust in 25 Member States of the European Union. In 2000–03, approximately 3.6 million workers in the European Union [and undoubtedly millions more worldwide] were exposed occupationally to inhalable wood dust. The estimated number of workers exposed by industry and the number exposed to a level exceeding 5 mg/m<sup>3</sup> are shown in [Table 1.2](#). The highest exposure levels were estimated to occur in the construction sector and furniture industry.

Due to limited exposure data, there was considerable uncertainty in the estimates concerning construction woodworkers. About 560000 workers (16% of the number of workers exposed) may have been exposed to a level of inhalable wood dust that exceeded 5 mg/m<sup>3</sup>. Mixed exposures to more than one species of wood and dust from wooden boards was very common, but reliable data on exposure to different species of wood could not be retrieved.

The US National Occupational Exposure Survey, carried out during 1981–83 in the United States of America, estimated that about 600000 workers were exposed to wood dust. The largest numbers of exposed workers were employed in the building trades ( $n = 134090$ ), and the lumber/wood product industries ( $n = 153543$ ). Forestry workers (e.g. lumberjacks using chainsaws) were not considered to be exposed in this survey ([NIOSH, 1990](#)).

**Table 1.2 WOODEX: Estimated number of workers exposed to wood dust in the 25 Member States of the European Union, 2000–03**

Industry	Number employed	Number exposed	Exposed (% of employed)	Number exposed > 5 mg/m <sup>3</sup>
Construction	13 million	1.2 million	9	254000
Manufacture of furniture	1.2 million	713000	59	86500
Manufacture of joinery	472000	330000	71	42000
Forestry	445000	148000	33	< 100
Building of ships and boats	294000	31000	11	9600
Sawmilling	259000	196000	76	20000
Manufacture of other wood products	147000	97000	66	15500
Manufacture of wooden boards	124000	92000	74	8400
Manufacture of wooden containers	80000	57000	71	8600
All other employment	163 million	709000	0.4	118000
Total	179 million	3.6 million	2.0	563000

From [Kauppinen et al. \(2006\)](#)

### 1.2.2 Levels of occupational exposure

The highest exposures to wood dust have generally been reported in wood furniture and cabinet manufacture, especially during machine-sanding and similar operations (with wood dust levels frequently above 5 mg/m<sup>3</sup>). Exposure levels above 1 mg/m<sup>3</sup> have also been measured in the finishing departments of plywood and particle-board mills, where wood is sawn and sanded, and in the workroom air of sawmills and planer mills near chippers, saws, and planers. Exposure to wood dust also occurs among workers in joinery shops, window and door manufacture, wooden boat manufacture, installation and refinishing of wood floors, pattern and model making, pulp and paper manufacture, construction carpentry, and logging. Measurements are generally available only since the 1970s, and exposures may have been higher in the past because of less efficient (or non-existent) local exhaust ventilation or other measures to control dust ([IARC, 1995](#)).

Woodworking machines have increased greatly in efficiency since the industrial revolution, and the increased speed of production has resulted in the generation of more dust. The increased efficiency may also result in exposure to finer wood dust particles than in the past,

because smoother surfaces can be produced, and because saws and bits may retain their sharpness for longer. The introduction of engineering controls in some industries in some parts of the world, especially since the 1950s, has, however, reduced the exposure of workers considerably. Unfortunately, engineering controls, even if properly maintained, are not always effective, and the dust generated by hand-held power tools, particularly sanders, is much more difficult to control ([IARC, 1995](#)).

Studies published since the previous *IARC Monograph* reporting wood dust concentrations are presented in [Table 1.3](#).

### 1.2.3 Particle size distribution

[Chung et al. \(2000\)](#) characterized the quantity, particle size distribution and morphology of dust created during the machining of medium-density fibreboard (MDF) in a controlled environment (a 2 × 2 × 2 m<sup>3</sup> dust chamber). In terms of particle size distribution and morphology, the dust generated by machining MDF was generally found to be comparable with the dust generated by similarly machining hardwood or softwood. The quantity of dust generated during sanding

**Table 1.3 Wood dust concentrations in various industries around the world**

Reference, industry and country, period (if reported)	Site, occupation, or exposure circumstance	Concentration of wood dust (mg/m <sup>3</sup> )	Number of samples	Comments
<b>Sawmills and lumber mills</b>				
<a href="#">Demers et al. (2000)</a> , <a href="#">Teschke et al. (1999b)</a> Softwood lumber mill British Columbia, Canada July–August 1996	Sawmill, planer mill, and yard	<i>Geometric mean (GSD)</i> Inhalable particulate: 1.0 (2.7) Estimated wood dust: 0.5 (3.1)	220	Exposure assessment conducted for cross-sectional study of respiratory health among 275 softwood lumber mill workers; mill processed spruce ( <i>Picea engelmannii</i> and <i>glauca</i> ), pine ( <i>Pinus contorta</i> ), and fir ( <i>Abies lasiocarpa</i> ); random sampling strategy; full-shift (7–8 hours) personal inhalable particulate samples collected using seven-hole inhalable dust samplers; wood dust exposure estimated using the resin acid content within dust in combination with observations of job tasks, proximity to dust sources and use of personal protective equipment
<a href="#">Rosenberg et al. (2002)</a> Sawmill, Finland 1997–99	Sawhouse - pine processing - spruce processing	<i>Range of geometric means</i> Inhalable particulate: 0.5–2.2 Inhalable particulate: 0.4–1.9	237 (178 personal)	Measured exposure of 22 sawhouse workers in mills processing pine ( <i>Pinus sylvestris</i> ) and spruce ( <i>Picea abies</i> ); full-shift area and personal inhalable particulate samples collected in breathing zone; exposure measured during evening shift on three consecutive days; IOM samplers to collect inhalable dust; gravimetric determination of inhalable dust; assumption that all or most of inhalable dust originated from wood dust
<a href="#">Hall et al. (2002)</a> , Sawmills British Columbia, Canada 1981–97	Lumber mill	<i>Geometric mean (GSD)</i> 0.72 (3.49)	1237	Analysis of compliance data set (and a nested subset of research data) containing personal exposure measurements to wood dust at 77 lumber mills; 23% of database were research samples, 77% were compliance samples; an empirical “determinants of exposure” model created using multiple linear regression

Table 1.3 (continued)

Reference, industry and country, period (if reported)	Site, occupation, or exposure circumstance	Concentration of wood dust (mg/m <sup>3</sup> )	Number of samples	Comments
<a href="#">Rusca et al. (2008)</a> , Sawmill Switzerland June–October 2002	Sawmill	Mean (range) Inhalable particulate: 1.7 (0.2–8.5)	NR	Cross-sectional survey of male employees at 12 sawmills processing spruce and fir species in the French part of Switzerland; personal measurements of inhalable dust collected using IOM samplers; gravimetric analysis of inhalable dust
<b>Miscellaneous wood-related occupations</b>				
<a href="#">Edman et al. (2003)</a> Wood pellets and briquettes Sweden	Industrial production of wood pellets and briquettes	Geometric mean (range) overall: 1.7 (0.16–19)	24	Personal exposure to wood dust measured gravimetrically and with personal data logging, real-time aerosol monitor; sampling time: 8 hours;
<a href="#">Kalliny et al. (2008)</a> Wood-processing plants USA 1999–2004	Sawmill, plywood assembly plants, secondary wood milling operations, factories producing finished wood products	Geometric mean (GSD) Inhalable: 1.44 (2.67) Thoracic: 0.35 (2.65) Respirable: 0.18 (2.54)		Size-fractionated dust exposure surveyed longitudinally in 10 wood processing plants across the USA; dust exposures measured using the RespiCon Personal Particle Sampler; woods processed included softwoods (e.g. southern yellow pine and Radiata pine), hardwoods (red oak, maple, poplar, birch, rubber tree wood, cherry), engineered woods (medium-density fibreboard, particleboard), and plywood (from southern yellow pine)
<a href="#">Teschke et al. (1999a)</a> Misc. establishments USA 1979–97	Overall Sanders, transportation equipment industry Press operators, wood products industry Lathe operators, furniture industry Sanders, wood cabinet industry	Geometric mean (GSD) 1.86 (6.82) 17.5 (1.79) 12.3 (4.12) 7.46 (4.56) 5.83 (5.19)	1632 personal TWA samples	Analysis of 1632 measurements of airborne wood dust reported to OSHA's Integrated Management Information System and development of an empirical predictive model; measurements collected using OSHA sampling method for "total" particulate (i.e. non-specific gravimetric method)

Table 1.3 (continued)

Reference, industry and country, period (if reported)	Site, occupation, or exposure circumstance	Concentration of wood dust (mg/m <sup>3</sup> )	Number of samples	Comments
<a href="#">Commonwealth of Australia (2008)</a> Wood industries Australia	All wood industries	<i>Arithmetic mean (range)</i> 5.8 (0.06–210)	521	Analysis of existing surveillance data on inhalable wood dust exposure; data gathered via a review of published Australian literature; requests to government agencies, consultants, industry associations, specific industries and researchers; telephone surveys, and new air sampling
<a href="#">Alwis et al. (1999);</a> <a href="#">Mandryk et al. (1999)</a> Wood industries New South Wales, Australia 1996–97	Logging Sawmill Wood chipping Joinery	<i>Geometric mean (GSD)</i> Inhalable dust 0.6 (1.3) 1.6 (3.2) 1.9 (1.7) 3.7 (3.7)  Respirable dust < 0.1 (1.3) 0.3 (2.2) 0.3 (1.7) 0.5 (1.7)	7 93 9 66  4 31 4 39	Personal inhalable and respirable samples collected; sampling time: 6–8 hours
<a href="#">Scarselli et al. (2008)</a> Wood industries Italy 1996–2006	All wood-related	<i>Geometric mean (GSD)</i> 1.0 (1.6)	10837	Analysis of airborne wood dust exposure measurements contained in the SIREP (Italian Information System on Occupational Exposure to Carcinogens) database; 10837 measurements on 10528 workers at 1181 companies; concentration of wood dust (hardwood or mixed wood dust) measured as 8-h TWA; no information about type of sample (personal vs stationary) or sampling strategy (random vs systematic)
<a href="#">Baran &amp; Teul (2007)</a> Wood processing Poland	Sawmill, manufacturing frames for furniture, and manufacturing ready-made furniture	<i>Range</i> 0.59–16.2		Analysis of measurements on 1100 workers employed in 9 wood-processing plants: 2 sawmills, 4 plants manufacturing frames for upholstered furniture, and 3 plants manufacturing ready-made furniture

Table 1.3 (continued)

Reference, industry and country, period (if reported)	Site, occupation, or exposure circumstance	Concentration of wood dust (mg/m <sup>3</sup> )	Number of samples	Comments
<a href="#">HSE (2000)</a> ; <a href="#">Black et al. (2007)</a> Woodworking United Kingdom 1999–2000	Sawmilling, joinery, furniture manufacture, other	Range of medians 1.5–2.8	396	Cross-sectional survey of 46 representative companies in the British woodworking industry; personal samples collected as per MDHS 14/3; sampling time: 3–6 hours during activities judged to be representative of whole shift; gravimetric analysis of inhalable dust
<a href="#">Spee et al. (2007)</a> Building projects the Netherlands 2002	Carpenters - overall Task-based - working indoors - working outdoors - indoors + outdoors	Geometric mean (GSD) 3.3 (2.1) Arithmetic mean 5.2 2.2 16.2	44 29 11 4	Task-based exposure survey of 26 carpenters at 13 building projects from 12 companies; personal and area samples randomly collected as per specially designed protocol for sampling of wood dust in carpentry and furniture industry; gravimetric analysis of wood dust

GSD, geometric standard deviation; NR, not reported; OSHA, Occupational Safety and Health Administration; TWA, time-weighted average

was higher for sanding MDF when compared with sanding either hardwood or softwood. However, there was no significant difference with sanding MDF and natural woods, in terms of the quantity of dust generated.

Additional information on the particle size distribution of wood dust in workroom air can be found in the previous *IARC Monograph* ([IARC, 1995](#)).

### 1.2.4 Exposure to other agents

Within the furniture-manufacturing industry, exposure may occur to solvents and formaldehyde in glues and surface coatings. Such exposures are usually greatest for workers with low or negligible exposure to wood dust, and are infrequent or low for workers with high exposure to wood dust. Some outdoor furniture has also been manufactured from impregnated wood containing copper–chromium–arsenic compounds. Formaldehyde-based glues and varnishes were introduced in the wood industry after World War II but they became commonly used only in the 1950s and 1960s in most countries.

The manufacture of plywood and particle board may result in exposure to formaldehyde, solvents, phenol, wood preservatives, and engine exhausts. Sawmill workers may also be exposed to wood preservatives and fungal spores. Wood preservatives used include chlorophenol salts in sawmills, and organochlorine pesticides in plywood mills. When coniferous trees are sawn, monoterpenes evaporate into workroom air. In some sawmills, wood is also impregnated with copper–chromium–arsenic salts or creosote. Construction woodworkers may be exposed to asbestos and silica in their work environment. Many of them also varnish wooden floors with solvent- or water-based varnishes, some of which may release formaldehyde. Exposures to chemicals in industries where other wood products are manufactured vary, but are in many cases

similar to those in the furniture-manufacturing industry ([IARC, 1995](#)).

### 1.2.5 Exposure of the general population

Woodworking is a popular hobby and non-occupational exposure may also occur during building and repair operations in homes. Woodworking can encompass a variety of activities that generate wood dust, including sawing, sanding, planing, routing, etc. The woods worked include a variety of particle boards, soft timbers, treated pine, masonite, plywood, and various imported hardwoods and softwoods. The size of the dust particles produced, the amount of dust, and resultant exposure to the person working in these areas depends on several factors including the equipment being used, the ventilation and extraction system in place, the state and type of timber, the general ventilation in the area, and any personal protective equipment that may be used. Exposure levels during non-occupational woodworking may be similar to those at workplaces, but the duration of exposure is usually substantially shorter.

## 2. Cancer in Humans

In the previous *IARC Monograph*, the evidence associated with exposure to wood dust or wood-related occupations or activities and cancer of the nasal cavity and paranasal sinuses (referred to below as ‘sinonasal cancer’), and of the nasopharynx, larynx, lung, stomach, colon, and rectum as well as leukaemia, Hodgkin lymphoma, non-Hodgkin lymphoma, and multiple myeloma was systematically reviewed because excesses had been observed in one or more studies. The Working Group for the previous *IARC Monograph* concluded that there was very strong evidence for sinonasal cancer. In case–control studies, they also consistently observed associations between exposure to wood

dust and cancer of the nasopharynx, but could not rule out confounding; and between wood dust and cancer of the larynx, but noted conflicting evidence from cohort studies. The Working Group concluded that there was “no indication that occupational exposure to wood dust has a causal role in cancers of the oropharynx, hypopharynx, lung, lymphatic and haematopoietic systems, stomach, colon, or rectum” (IARC, 1995).

Since the previous *IARC Monograph*, several studies have been published including selected case series of sinonasal cancer (see Table 2.1), cohort studies (see Table 2.2), registry-based studies (see Table 2.3). For case-control or other studies focused on particular cancer sites, only studies published since the previous volume that reported results for wood dust exposure are summarized here. The results of case-control studies on sinonasal, pharyngeal, and laryngeal cancer are summarized in Tables 2.4, 2.5, and 2.6, respectively. In addition, the results of case-control studies on lung cancer are summarized in Table 2.7, because of the relatively large number of studies that focus on this cancer site. Studies of other cancer sites are summarized in Table 2.8.

## 2.1 Sinonasal cancer

The Working Group for the previous *IARC Monograph* (IARC, 1995) reviewed a large number of case-control studies that consistently observed a strong association between exposure to wood dust or employment in wood-related occupations and sinonasal cancer. Support for this association was found in several large cohort studies of furniture workers (Olsen & Sabroe, 1979; Acheson *et al.*, 1984), but most cohort studies had little power to examine the risks for this cancer site (see Table 18, IARC, 1995). Odds ratios for all or unspecified sinonasal cancers were consistently elevated in case-

control studies conducted in many countries (see Table 20, IARC, 1995).

Very high odds ratios were observed for sinonasal adenocarcinoma and strong evidence of an exposure-response relationship was observed in some studies (Hayes *et al.*, 1986; Olsen & Asnaes, 1986; Luce *et al.*, 1993) (see Table 21, IARC, 1995). In addition, an unusually large proportion of all adenocarcinomas in cases series were woodworkers. Some case-control studies observed an excess risk of sinonasal squamous cell carcinoma associated with wood dust or wood occupations, but the association was much weaker than was observed with adenocarcinoma (see Table 22, IARC, 1995). A pooled re-analysis of 12 case-control studies (including six of the nine above) found strong evidence for an exposure-response relationship among men for adenocarcinoma (OR, 0.6; 95%CI: 0.6–4.7 for low; OR, 3.1; 95%CI: 1.6–6.1 for moderate; and OR, 45.5; 95%CI: 28.3–72.9 for high wood dust), and little evidence for squamous cell carcinoma (OR, 0.9; 95%CI: 0.6–1.2 for low; OR, 1.0; 95%CI: 0.7–1.4 for moderate; and OR, 0.8; 95%CI: 0.4–1.6 for high wood dust) (Demers *et al.*, 1995a). For the three studies with results for squamous cell carcinoma not included in the pooled re-analysis, Fukuda *et al.* (1987) observed an excess among both male (OR, 2.9; 95%CI: 1.5–5.6) and female woodworkers (OR, 2.0; 95%CI: 0.3–14), Shimizu *et al.* (1989) observed an excess of squamous cell carcinoma of the maxillary sinus among male woodworkers (OR, 2.1; 95%CI: 0.8–5.3), and Olsen & Asnaes (1986) observed only a slightly increased risk of carcinoma of the sinonasal cavities among men classified as exposed to wood dust (OR, 1.3; 95%CI: 0.6–2.8).

Among the cohort studies that reported tree species, an excess of sinonasal cancer (SMR, 8.1; 95%CI: 3.7–16) was observed among British furniture workers exposed to hardwood dust (Rang & Acheson, 1981; Acheson *et al.*, 1984). No cases of sinonasal cancer were reported in a much smaller study of German furniture

workers exposed to beech, oak, and pine ([Barthel & Dietrich, 1989](#)) or among two small cohort of workers exposed to softwood—Finnish sawmill workers ([Jäppinen et al., 1989](#)) and American plywood workers ([Robinson et al., 1990](#)). [The Working Group noted that their power to detect an excess was low, and that exposure levels among sawmill and plywood workers were low compared to furniture workers.]

Few case-control studies in the previous *IARC Monograph* reported tree species. Very large excesses of sinonasal adenocarcinoma were associated with hardwood dust exposure in studies from France (OR, 5.30; 95%CI: 1.04–2.70, for highest level of exposure, [Leclerc et al., 1994](#)) and Italy (OR, 0.90; 95%CI: 0.20–4.07, [Battista et al., 1983](#)). Excesses of sinonasal cancer were observed among workers primarily exposed to softwood in case-control studies from Nordic Countries (OR, 3.3; 95%CI: 1.1–9.4, [Hernberg et al., 1983](#)), the USA (OR, 3.1; 95%CI: 1.0–9.0 with 15-year lag, [Vaughan et al., 2000](#)), Canada (OR, 2.5;  $P < 0.03$ , [Elwood, 1981](#)), and France (OR, 1.7, [Leclerc et al., 1994](#)). The results for three of these four studies were restricted to squamous cell carcinoma.

Early case series reported many cases of sinonasal adenocarcinoma that were exposed to hardwoods ([Acheson et al., 1968, 1972](#); [Leroux-Robert, 1974](#); [Luboinski & Marandas, 1975](#); [Andersen et al., 1976, 1977](#); [Engzell et al., 1978](#); [Kleinsasser & Schroeder, 1989](#)). Seven cases of sinonasal squamous cell carcinoma exposed to “softwoods” were reported in a Norwegian case series ([Voss et al., 1985](#)), and three cases of adenocarcinoma were reported among British workers exposed to softwoods ([Acheson et al., 1972](#)). Several new case series have also been published with results relevant for the evaluation of sinonasal cancer ([Table 2.1](#)). Case series of sinonasal adenocarcinoma continue to make up a large proportion of cases with exposure to wood dust, with mean exposure durations ranging from 25 to 37 years. Most case series were restricted

to adenocarcinoma, but in the case series that considered other tumours, the proportion of wood dust exposure was much less in the non-adenocarcinoma cases.

In the period following the previous *IARC Monograph* ([IARC, 1995](#)), five cohort studies ([Table 2.2](#)) were published that are relevant for the evaluation of wood dust, and three present results for sinonasal cancer. In a pooled re-analysis of five previously published cohort studies, [Demers et al. \(1995b\)](#) found an excess risk of sinonasal cancer among men classified as being definitely exposed to wood dust (SMR, 8.4; 95%CI: 3.9–16.0). [Stellman et al. \(1998\)](#) found no evidence of an excess risk associated with self-reported wood dust exposure or longest occupation among participants in the Cancer Prevention Study II. [Innos et al. \(2000\)](#) found an excess risk of sinonasal cancer among Estonian furniture workers highly exposed to wood dust (for men SIR, 2.3; 95%CI: 0.3–8.4,  $n = 2$ ; for women SIR, 3.2; 95%CI: 0.1–18.1,  $n = 1$ ).

Three new case-control studies ([Table 2.4](#)) have published results relevant for the evaluation of sinonasal cancer. [Teschke et al. \(1997\)](#) found no association with softwood or hardwood dust in a small Canadian study. In a pooled re-analysis of European case-control studies [’t Mannetje et al. \(1999\)](#) found a strong association with adenocarcinoma (OR, 12.2; 95%CI: 7.4–20.0), but no association with squamous cell carcinoma (OR, 0.7; 95%CI: 0.5–1.1). [Pesch et al. \(2008\)](#) found a strong association between adenocarcinoma and hardwood dust exposure (OR, 4.0; 95%CI: 1.9–8.3), but not with softwood dust exposure (OR, 0.3; 95%CI: 0.2–0.7). [The Working Group noted that only compensated cases were included, and this may have biased the results towards hardwood dust exposure.]

Table 2.1 Case series of sinonasal cancer according to occupation and exposure to wood dust

Reference, location, name of study	Sex	Origin	Histology	Exposed cases/ total cases	Occupations/exposures	Comments
<a href="#">Svane-Knudsen et al. (1998)</a> Denmark	M, F	Nasal cavity and paranasal sinuses Hospital-based series 1978–95	Adenocarcinomas Epidermoid carcinomas	12/22 3/41	Hardwood dust exposure based on patient records [further details were not provided]	Softwood dust exposure not mentioned
<a href="#">Stoll et al. (2001)</a> France	M, F	Ethmoidal sinuses 1975–2000	Adenocarcinomas	62/76	Exposed to wood dust	Mean duration of wood dust exposure 26 yr
<a href="#">Roux et al. (2002)</a> France		Sinonasal cancer 1985–2001	Adenocarcinomas	134/139	Wood dust exposure from furniture, sawmill, carpentry and wood-product workers	Mean duration of wood dust exposure 30 yr
<a href="#">Barbieri et al. (2005)</a> Italy	M, F	Ethmoidal sinuses 1978–2002	Adenocarcinomas	17/100	Hardwood and softwood dust exposure (5 softwood only)	
<a href="#">Liétin et al. (2006)</a> France	M, F	Ethmoidal sinuses Hospital-based series 1985–2004	Adenocarcinomas	45/60	Wood dust exposure	Mean duration of wood dust exposure 25.6, range 2–44 yr
<a href="#">Fontana et al. (2008)</a> France	M, F	Sinonasal cancer Diagnostic Registry 1981–2000	All	46/76 men 0/24 women	Wood dust exposure	Mean duration of wood dust exposure 37 yr
<a href="#">Llorente et al. (2008)</a> Spain	M, F	Hospital-based series 1986–2002	All	62/79	Wood dust exposure	
<a href="#">Børnholdt et al. (2008)</a> Denmark	M, F	Sinonasal cancer 1991–2001	Adenocarcinomas Squamous cell carcinomas	33/58 7/109	Wood dust exposure as per job title from the Central Person Registry or interview	
<a href="#">Choussy et al. (2008)</a> France	M, F	Ethmoidal sinuses Hospital-based series 1976–2001	Adenocarcinomas	353/418	Wood dust exposure	Mean duration of wood dust exposure 27.7 yr

Table 2.2 Cohort studies of woodworkers exposed to wood dust

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Demers et al. (1995b)</a> Cohort mortality study United Kingdom and USA	Pooled analysis of updated data from 5 studies: British furniture workers ( <a href="#">Acheson et al., 1984</a> ), US furniture workers ( <a href="#">Miller et al., 1994</a> ), two cohorts of plywood workers ( <a href="#">Blair et al., 1990</a> ; <a href="#">Robinson et al., 1995</a> ), and wood model makers ( <a href="#">Roscoe et al., 1992</a> )	Workers classified as exposed to wood dust based on available work history	All cancers (140–208) Pharynx (146–149) Nasopharynx (147)	All woodworkers All woodworkers All woodworkers Possible wood dust Probable wood dust Definite wood dust All woodworkers Possible wood dust Probable wood dust Definite wood dust All woodworkers Possible wood dust Probable wood dust Definite wood dust	1726 20 9 4 0 5 11 1 1 9 18 4 8 6 575 138 136 60 57	0.8 (0.8–0.8) 0.8 (0.5–1.3) 2.4 (1.1–4.5) 2.9 (0.8–7.5) 0.0 (0.0–3.8) 5.3 (1.7–12.4) 3.1 (1.6–5.6) 0.8 (0.0–4.6) 1.2 (0.0–6.5) 8.4 (3.9–16.) 0.7 (0.4–1.0) 0.4 (0.1–1.1) 1.1 (0.5–2.1) 0.8 (0.3–1.8) 0.8 (0.7–0.9) 0.9 (0.8–1.1) 0.8 (0.6–0.9) 0.8 (0.6–1.0) 1.1 (0.8–1.4)	SMRs adjusted for sex, age, & calendar period using national rates	
			Larynx (161)					
			Lung (162) Stomach (151) Intestine (152, 153) Rectum (154) Non-Hodgkin lymphoma (200, 202) Hodgkin disease (201) Multiple myeloma (203)					

Table 2.2 (continued)

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Demers <i>et al.</i> (1995b)</a> (contd.)				Possible wood dust Probable wood dust Definite wood dust All woodworkers	9 8 11 47	1.0 (0.5–1.9) 1.3 (0.6–2.5) 1.6 (0.8–2.8) 0.7 (0.5–0.9)		
<a href="#">Stellman <i>et al.</i> (1998)</a> Prospective cohort USA	Prospective study of 362823 men enrolled in the American Cancer Society Cancer Prevention Study II in 1982 and followed up for 6 yr	Self-reported wood dust exposure or wood-related occupation	Leukaemia (204–208) All causes (0–999)  All cancers (140–208)  Pharynx (146–149)  Nasopharynx (147)	Wood dust exposure Wood occupation Wood dust exposure Wood occupation	2995 1271 961 381 7 2 1 1 1 0 8 2	1.1 (1.0–1.1) 1.2 (1.1–1.2) 1.1 (1.0–1.2) 1.2 (1.1–1.3) 0.9 (0.4–2.0) 0.8 (0.2–3.4) 0.4 (0.1–3.3) 1.4 (0.4–1.8) 1.1 (0.1–8.4) 0 1.6 (0.8–3.4) 1.2 (0.3–4.9)	RRs adjusted for age and smoking status	

Table 2.2 (continued)

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">(Stellman <i>et al.</i>, 1998)</a> contd.)			Lung (162)	Wood dust exposure	317	1.2 (1.0–1.3)		
				Wood occupation	111	1.1 (0.9–1.4)		
			Stomach (151)	Wood dust exposure	40	1.3 (1.0–1.9)		
				Wood occupation	11	1.1 (0.6–1.9)		
			Colon (153)	Wood dust exposure	100	1.0 (0.8–1.3)		
				Wood occupation	37	1.0 (0.8–1.5)		
			Rectum (154)	Wood dust exposure	23	1.3 (0.8–2.0)		
				Wood occupation	9	1.5 (0.8–2.9)		
			Non-Hodgkin lymphoma (200, 202)	Wood dust exposure	39	1.1 (0.8–1.5)		
				Wood occupation	12	1.0 (0.6–1.7)		
			Hodgkin disease (201)	Wood dust exposure	4	1.2 (0.4–3.4)		
				Wood occupation	1	1.0 (0.1–7.7)		
			Multiple myeloma (203)	Wood dust exposure	16	1.0 (0.6–1.8)		
				Wood occupation	4	0.7 (0.3–1.9)		
		Leukaemia (204–208)	Wood dust exposure	32	0.9 (0.6–1.3)			
			Wood occupation	14	1.1 (0.6–1.9)			

Table 2.2 (continued)

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Innos <i>et al.</i> (2000)</a>	Retrospective study of incident cancers in all furniture workers employed in Tallinn, Estonia, for at least six months between 1 January 1946 and 31 December 1988 and living in Estonia on 1 January 1968.	Exposure based on industrial hygiene surveys and work history	All cancers (140–208)	Med. exposure men	55	1.2 (0.9–1.6)	SIRs adjusted	
Retrospective cancer incidence cohort study Estonia				High exposure men	265	1.0 (0.9–1.1)	for age and calendar period	
				Med. exposure women	98	1.0 (0.8–1.2)		
				High exposure women	171	1.1 (0.9–1.3)		
			Buccal cavity (140–145)	Med. exposure men	5	3.7 (1.2–8.6)		
				High exposure Men	6	0.8 (0.3–1.7)		
				Med. exposure women	2	2.5 (0.3–8.9)		
				High exposure women	2	1.6 (0.2–5.8)		
			Pharynx (146–149)	Med. exposure men	3	4.0 (0.8–11.8)		
				High exposure men	6	1.5 (0.6–3.3)		
				Med. exposure women	0	0.0		
				High exposure women	0	0.0		
			Paranasal sinus (160)	Med. exposure men	0	0.0		
				High exposure men	2	2.3 (0.3–8.4)		
				Med exposure women	0	0.0		
				High exposure women	1	3.2 (0.1–18.1)		

**Table 2.2 (continued)**

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Innos <i>et al.</i> (2000)</a> (contd.)			Larynx	Men	7	0.7 (0.3–1.4)		
				Women	1	1.7 (0.0–9.4)		
			Bronchi and lung (162)	Med. exposure men	9	0.8 (0.4–1.5)		
				High exposure men	70	1.0 (0.8–1.3)		
				Med. exposure women	5	1.1 (0.4–2.6)		
			Stomach (151)	High exposure women	11	1.6 (0.8–2.9)		
				Med. exposure men	11	1.7 (0.9–3.0)		
				High exposure men	36	0.9 (0.6–1.2)		
				Med. exposure women	13	1.2 (0.7–2.1)		
				High exposure women	23	1.4 (0.9–2.1)		
			Colon (153)	Med. exposure men	6	3.0 (1.1–6.7)		
				High exposure men	18	1.5 (0.9–2.4)		
				Med. exposure women	8	1.4 (0.6–2.7)		
				High exposure women	16	1.8 (1.0–2.9)		
Rectum (154)	Med. exposure men	1	0.6 (0.0–3.2)					
	High exposure men	13	1.2 (0.7–2.1)					
	Med. exposure women	7	1.6 (0.6–3.2)					
	High exposure women	11	1.6 (0.8–2.9)					

Table 2.2 (continued)

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Innos <i>et al.</i> (2000)</a> (contd.)			Hodgkin disease (201)	Med. exposure men High exposure men Med. exposure Women High exposure women	1 3 0 1	2.6 (0.1–14.3) 1.4 (0.3–4.2) 0.0 1.3 (0.0–7.5)		
			Haematopoietic and lymphatic (200–208)	Med. exposure men High exposure men Med. exposure women High exposure women	2 14 5 3	0.8 (0.1–2.8) 0.9 (0.5–1.5) 1.0 (0.3–2.3) 0.4 (0.1–1.2)		
<a href="#">Szadkowska-Stańczyk &amp; Szymczak (2001)</a>	79 deceased lung cancer cases from a cohort of 10575 Polish pulp and paper mill workers (7084 men, 3491 women), 1+ yr, 1968–90, observed through 1995	Employment history obtained from the mills; occupational exposure was assessed by experts and a cumulative dose index	Lung (162)	Wood dust exposure Low Moderate & high 1–4 yr of exposure 5+ yr of exposure	10 4 6 4 6	2.1 (0.9–4.9) 2.1 (0.6–7.4) 2.1 (0.7–6.3) 1.7 (0.5–6.2) 2.4 (0.8–7.7)	ORs adjusted for smoking. Matched on sex, birth year ( $\pm 1$ yr), hire year ( $\pm 3$ yr), and vital status	
				Low cumulative dose High cumulative dose	4 6	2.1 (0.5–9.2) 2.0 (0.7–5.4)		

**Table 2.2 (continued)**

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Dement <i>et al.</i> (2003)</a>	13354 male carpenter members of the United Brotherhood of Carpenters and Joiners of America matched to the New Jersey State Cancer registry, who had participated in the New Jersey Carpenters fund before 1 July 2000 and matched to the New Jersey Carpenters Pension Fund	Employment as a carpenter	All cancers (140–208)	All	592	1.1 (1.0–1.2)	SIRs adjusted for age and calendar period	The lowest duration of carpenter work (< 10 yr) was used as the comparison group for expected cases
Cohort mortality study USA			Pharynx (146–149)		11	1.4 (0.7–2.4)		
			Oesophagus (150)		8	1.1 (0.5–2.2)		
			Stomach (151)		12	1.0 (0.5–1.8)		
			Rectum (154)		35	1.5 (1.1–2.1)		
			Liver and gallbladder (155, 156)		12	1.6 (1.1–2.1)		
			Larynx (161)		14	1.2 (0.7–2.0)		
			Trachea, bronchus, and lung (160, 162)		137	1.5 (1.2–1.7)		
			Other respiratory (163–165)		15	4.2 (2.4–6.9)		
			Leukaemia (204–208)		12	0.8 (0.4–1.4)		
			Myeloma (203)		9	1.5 (0.7–2.8)		

Table 2.2 (continued)

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Lee et al. (2003)</a> Cohort cancer incidence study Sweden	365424 male construction workers screened by the Organization for Working Environment, Occupational Safety and Health during 1971–93 and followed during 1971–99 through the Swedish National Cancer Registry	Wood dust exposure assessment based on a job-exposure matrix	Multiple myeloma (203)	Never exposed <sup>1</sup> Ever exposed <sup>1</sup> Never exposed <sup>2</sup> Ever exposed <sup>2</sup>	376 20 376 20	1.0 (reference) 0.8 (0.49–1.20) 1.0 (reference) 0.8 (0.49–1.23)	RR <sup>1</sup> adjusted for BMI at entry to cohort RR <sup>2</sup> adjusted for age, BMI, and other occupational co-exposures	
<a href="#">Jansson et al. (2005)</a> Cohort cancer incidence study Sweden	Male construction workers, same population as <a href="#">Lee et al. (2003)</a> 260052 workers in cohort after excluding those missing smoking and BMI	Wood dust exposure assessment based on a job-exposure matrix	Oesophagus (adenocarcinoma)  Gastric (cardia; adenocarcinoma)  Oesophagus (squamous cell carcinoma)	No exposure Moderate exposure High exposure No exposure Moderate exposure High exposure No exposure Moderate exposure High exposure	61 3 0 152 11 2 170 8 1	1.0 (reference) 0.8 (0.2–2.5) 0 1.0 (reference) 1.1 (0.6–2.0) 4.8 (1.2–19.4) 1.0 (reference) 0.7 (0.4–1.5) 2.2 (0.3–15.9)	IRRs adjusted for attained age, calendar year at entry into cohort, tobacco smoking at entry to cohort and BMI at entry to cohort	

Table 2.2 (continued)

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases/deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Purdue <i>et al.</i> (2006)</a> Cohort cancer incidence study Sweden	Male construction workers, same population as <a href="#">Lee <i>et al.</i> (2003)</a> , 307779 workers in cohort after excluding those missing smoking	Wood dust exposure assessment based on a job-exposure matrix	All sites (140–208)  Oral cavity (140–145)  Pharynx (146–149)  Larynx (161)	Never exposed Ever exposed Never exposed Ever exposed Never exposed Ever exposed Never exposed Ever exposed	490 20 166 5 108 4 216 11	1.0 0.7 (0.4–1.0) 1.0 0.5 (0.2–1.2) 1.0 0.6 (0.2–1.6) 1.0 0.8 (0.5–1.5)	RRs adjusted for age, smoking status and snuff use	
<a href="#">Sjödahl <i>et al.</i> (2007)</a> Cohort cancer incidence study Sweden	Male construction workers, same population as <a href="#">Lee <i>et al.</i> (2003)</a> , 256357 workers in cohort after excluding those missing smoking and BMI	Wood dust exposure assessment based on a job-exposure matrix	Gastric (non-cardia) (151)	Wood dust No exposure Moderate exposure High exposure	892 53 3	1.0 (reference) 0.9 (0.7–1.2) 1.2 (0.4–3.6)	RRs adjusted for age, smoking status and BMI	

BMI, body mass index; CI, confidence interval; IRR, incidence rate ratio; RR, relative risk; SIR, standardized incidence ratio; standardized mortality ratio; yr, year or years



**Table 2.3 (continued)**

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases /deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Weiderpass et al. (2001)</a> Census linkage study Finland	2833 cases of endometrial cancers and 1101 cervical cancers diagnosed since 1971 in a cohort of 413877 skilled and specialized workers in Finland excluding farming occupations	Occupations were coded into job titles and a national job-exposure matrix (FINJEM) converted each job title into a probability and mean level of exposure	Endometrium  Cervix	Wood surface finisher Low wood dust exposure High wood dust exposure	8 368 70	1.8 (0.8–3.5) 1.0 (0.9–1.2) 1.1 (0.8–1.4)	SIRs adjusted for birth cohort, follow-up period, and social class	
				Woodworker, NEC Plywood and fibreboard worker	7 24	2.5 (1.0–5.1) 1.6 (1.0–2.3)		
				Low wood dust exposure High wood dust exposure	249 34	1.2 (1.0–1.4) 1.2 (0.9–1.7)		



**Table 2.3 (continued)**

Reference, location, name of study	Cohort description	Exposure assessment	Organ site (ICD code)	Exposure categories	No. of cases /deaths	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Laakkonen et al. (2006)</a> Census linkage study Finland	Incident cancer cases in all economically active Finns born during 1906–45 who participated in the national population census on 31 December 1970 (667121 men; 513110 women)	Exposure to wood dust: None (0) Low (< 3 mg/m <sup>3</sup> -yr) Med (3–50 mg/m <sup>3</sup> -yr) High (> 50 mg/m <sup>3</sup> -yr)	Nasal cavity (160)	None men women Low men women Med men women High men women	259 118 15 1 17 1 1 0	1.0 (0.9–1.1) 1.0 (0.8–1.2) 1.6 (0.9–2.6) 1.7 (0.0–9.7) 1.3 (0.8–2.1) 0.8 (0.0–4.4) 1.2 (0.0–6.9) 0.0	SIRs adjusted for age and social class	Exposure lag period 20 yr
			Larynx (161)	None men women Low men women Med men women High men women	1965 128 76 1 77 3	1.0 (1.0–1.1) 1.0 (0.8–1.2) 1.1 (0.8–1.3) 1.2 (0.0–6.8) 0.7 (0.6–0.9) 2.1 (0.4–6.1) 0.1 (0.0–0.7) 0.0		
			Lung (162)	None men women Low men women Med men women High men women	27309 3446 936 21 1784 48 108 12	1.0 (1.0–1.0) 1.0 (1.0–1.0) 1.1 (1.0–1.2) 0.9 (0.6–1.4) 1.0 (1.0–1.1) 1.0 (0.8–1.4) 0.9 (0.7–1.0) 1.0 (0.5–1.7)		

CI, confidence interval; CMORs, cancer mortality odds ratios; PCIRs, proportional cancer incidence ratios; RR, relative risk; SIRs, standardized incidence ratio; yr, year or years

## 2.2 Cancer of the nasopharynx

The previous *IARC Monograph* reviewed nine community-based case-control studies of cancer of the nasopharynx (see Table 25, [IARC, 1995](#)). The majority indicated an excess risk associated with either wood dust exposure (4/5 studies) or wood-related occupations (3/4 studies). Many of these studies had positive results based on very small numbers, and did not control for confounding. The studies were conducted in many different countries and odds ratios were generally in the range of 1.5–2.5. Among the studies that adjusted for the effects of smoking and alcohol, [Vaughan \(1989\)](#) and [Vaughan & Davis \(1991\)](#) observed an excess risk among carpenters (OR, 4.5; 95%CI: 1.1–19), and all woodworkers employed for 10 years or longer (OR, 4.2; 95%CI: 0.4–27). [Sriamporn et al. \(1992\)](#) observed an excess risk among wood cutters (OR, 4.1; 95%CI: 0.8–22).

None of the cohort studies reviewed by the previous *IARC Monograph* provided results for cancer of the nasopharynx, a rare tumour with an incidence rate of approximately 1/100000 in European countries.

In the period following the previous *IARC Monograph* ([IARC, 1995](#)), five new or updated cohort studies ([Table 2.2](#)) were published including a pooled re-analysis of five previously published cohort studies. [Demers et al. \(1995b\)](#) found an excess risk of cancer of the nasopharynx among workers classified as definitely exposed to wood dust (SMR, 5.3; 95%CI: 1.7–12.4,  $n = 5$ ) and, overall, excesses were observed among both furniture (SMR, 2.4; 95%CI: 1.2–5.9,  $n = 7$ ) and plywood workers (SMR, 4.6; 95%CI: 0.6–16.4,  $n = 2$ ). [Stellman et al. \(1998\)](#) found no evidence of an excess risk associated with self-reported wood dust exposure or longest occupation among participants in the Cancer Prevention Study II. The remaining cohort studies did not present results for this organ site.

Three new case-control studies ([Table 2.5](#)) have published results relevant for the evaluation of cancer of the nasopharynx. [Armstrong et al. \(2000\)](#) observed an increased risk associated with wood dust among Malaysian Chinese workers (OR, 2.4; 95%CI: 1.3–4.2). [Vaughan et al. \(2000\)](#) in a population-based study observed no increased risk overall (OR, 1.2; 95%CI: 0.5–2.7), and no evidence of an exposure-response relationship in analyses by maximum or cumulative exposure in a multicentre study in the USA. In another population-based study [Hildesheim et al. \(2001\)](#) found an increased risk overall (OR, 1.7; 95%CI: 1.0–3.0), which increased with both duration and cumulative exposure in Taiwan, China. It was also reported that these results were not affected by further adjustment for formaldehyde. One further hospital-based study reported an excess for nasopharyngeal and sino-nasal cancer combined ([Jayaprakash et al., 2008](#)).

## 2.3 Cancer of the pharynx

The previous *IARC Monograph* reviewed four case-control studies of cancer of the pharynx other than the nasopharynx (see Table 26, [IARC, 1995](#)). Two indicated an excess risk associated with wood-related occupations, although one was based on very small numbers. Another found mixed evidence. None of the cohort studies reviewed by that Working Group provided relevant results.

Four new case-control studies ([Table 2.5](#)) published since the previous *IARC Monograph* have results relevant for the evaluation of cancer of the pharynx other than the nasopharynx. [Gustavsson et al. \(1998\)](#) observed a decreased risk for cancer of the hypopharynx associated with wood dust in Sweden (OR, 0.5; 95%CI: 0.3–1.0). [Laforest et al. \(2000\)](#) observed no increased risk overall (OR, 0.9; 95%CI: 0.5–1.7), and only a slightly increased risk in the highest categories of cumulative exposure (OR, 1.5; 95%CI: 0.6–3.9). [Berrino et al. \(2003\)](#) found an increased risk of

**Table 2.4 Case-control studies of sinonasal cancer and exposure to wood dust**

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Teschke <i>et al.</i> (1997)</a> Population-based case-control study Canada	Nasal cavity and paranasal sinus (160)	All incident cases with histologically confirmed primary malignant tumours age $\geq$ 19 yr; 1990–92	Controls were selected randomly from 5-yr age and sex strata of the provincial voters list; frequency-matched for age and sex	Occupational histories obtained by interview and occupational exposures assessed by job classification	Hardwood dust Softwood dust	0.6 (0.1–3.0) 0.7 (0.3–1.6)	Sex, age (< 60, 60–69, $\geq$ 70), cigarette smoking (0–19, $\geq$ 20 pack-years)	
<a href="#">Mannetje <i>et al.</i> (1999)</a> Pooled population-based case-control study Italy, France, Netherlands, Germany, Sweden	Nasal cavity and paranasal sinus (160)	555 cases (104 women, 451 men) from 4 studies in Italy and 1 each from the Netherlands, France, Germany, and Sweden	1705 controls (241 women, 1464 men) from the same studies	Occupational history and job-exposure matrices were applied for wood dust	Wood dust exposure: Women Men Adenocarcinoma Squamous cell carcinoma	1.2 (0.3–4.5) 2.4 (1.8–3.2) 12.2 (7.4–20.0) 0.7 (0.5–1.1)	Age group and study centre. Sex and smoking where applicable	
<a href="#">Pesch <i>et al.</i> (2008)</a> Industry-based case-control study Germany	Nasal cavity and paranasal sinus (160)	86 male cases of adenocarcinoma of the nasal cavity and paranasal sinuses identified among workers with a recognized occupational disease during 1994–2003	204 controls randomly recruited from recognized accidents and falls frequency-matched to controls for age with 60 yr cut-off. Controls were also employed in the woodworking industries	Cumulative and average wood dust exposure quantified with a job-exposure matrix based on wood dust measurements at recent and historical workplaces	High exposure to: Hardwood Softwood Particle board Medium-density fibreboard	4.0 (1.9–8.3) 0.3 (0.2–0.7) 0.5 (0.3–1.0) 0.3 (0.1–1.1)	Smoking, age, region, ever exposed to varnishes or stains	Only cases with successful compensation claims were used

Table 2.5 Case-control studies of cancer of the pharynx and exposure to wood dust

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Gustavsson <i>et al.</i> (1998)</a> Population-based case-control study Sweden	Pharynx (140-149)	401 incident squamous cell carcinomas, men aged 40-79 yr living in Stockholm or Southern health care region, 1988-91	Randomly selected from the base population, frequency-matched on region and age group	Occupational history, exposure assessment based on literature survey of exposure	Ever exposed	0.5 (0.3-1.0)	Region, age, alcohol consumption, smoking habits	
<a href="#">Armstrong <i>et al.</i> (2000)</a> Population-based case-control study Malaysia	Nasopharynx (147)	282 Chinese cases identified between July 1990 and June 1992 through diagnosis records and/or treatment at centres with radiotherapy in the study area of Selangor & the Federal Territory	Matched by age ( $\pm$ 3yr) to 1 control in good health with no history of cancer of the head, neck or respiratory system, selected from Chinese population	Occupational histories were obtained by interview, exposure based on job	Any history of occupational exposure to wood dust	2.4 (1.3-4.2)	Diet and cigarette smoke indices, and matched on age	
<a href="#">Vaughan <i>et al.</i> (2000)</a> Multicentred population-based case-control study USA 1987-93	Nasopharynx (147)	196 newly diagnosed cases in men & women, age 18-74 yr, from 5 registries (Connecticut, Detroit, Iowa, Utah and western Washington)	244 controls from the general population through random-digit dialling and frequency-matched to the cases by age ( $\pm$ 5yr), sex and cancer registry	Lifetime histories of occupational and chemical exposures taken by interview; estimates of exposures assessed on a job-by-job basis	Ever exposed Max exposure (mg/m <sup>3</sup> ): > 0.0-0.55 > 0.55-1.50 > 1.50 Cumulative (mg/m <sup>3</sup> -yr): > 0.0-2.75 > 2.75-15.70 > 15.70	1.2 (0.5-2.7) 1.3 (0.5-3.6) 2.0 (0.5-8.1) 0.2 (0.0-2.1) 0.7 (0.2-2.5) 3.0 (0.9-9.8) 0.4 (0.1-2.3)	Age, sex, race, SEER site, cigarette use, proxy status, education and cumulative exposure to formaldehyde	

Table 2.5 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Laforest et al. (2000)</a> Hospital-based case-control study France 1989–91	Hypopharynx (148)	296 men, incident squamous cell carcinomas, histologically confirmed from 15 hospitals	Controls were patients with primary cancers of different sites requiring the same medical environment as case cancers, frequency-matched on age, same hospital or similar hospitals nearby, 1987–91	Detailed lifetime occupational history taken, occupational exposures were assessed through a job-exposure matrix	Ever exposed Probability: ≤ 70% > 70% Duration: < 6 yr 6–10 yr > 10 yr Cumulative: Low (< 10) Medium (10–42) High (> 42)	0.9 (0.2–4.1) 0.7 (0.3–1.8) 1.1 (0.5–2.3) 0.5 (0.2–1.5) 1.0 (0.3–2.9) 1.2 (0.5–3.0) 0.6 (0.2–1.6) 0.7 (0.3–2.3) 1.5 (0.6–3.9)	Age, smoking, alcohol, exposure to formaldehyde (yes/no), and mineral fibres (yes/no)	
<a href="#">Hildesheim et al. (2001)</a> Population-based case-control study Taiwan, China July 1991 to December 1994	Nasopharynx (147)	375 newly diagnosed, histologically confirmed cases identified through two tertiary care hospitals in Taipei, Taiwan, China; < 75 yr of age, residents of Taipei city for 6+ mo	325 community controls matched to cases on sex, age and geographic residence by use of listings available through the National Household Registration System	Occupational history via interview, blindly assessed by an industrial hygienist for intensity and probability of exposure	Ever exposed Duration: ≤ 10 yr > 10 yr Cumulative: < 25 ≥ 25	1.7 (1.0–3.0) 1.2 (0.6–2.5) 2.4 (1.1–5.0) 1.2 (0.6–2.5) 2.4 (1.2–5.1)	Age, sex, education, ethnicity, and HLA	

Table 2.5 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Berrino <i>et al.</i> (2003)</a> Population-based case-control study Italy, France, Spain, Switzerland 1979–82	Hypopharynx (148)	304 male incident cases from Calvados, France; Turin and Varese, Italy; Pamplona and Zaragoza, Spain; and Geneva, Switzerland	2176 male population controls	Occupational history through specialist interview; exposures assessed using a job-exposure matrix	< 55 yr of age: Possible exposure Probable exposure > 55 yr of age: Wood dust exposure	0.3 (0.1–1.0) 0.4 (0.2–1.2) 2.1 (1.2–3.7)	Age, centre, tobacco, alcohol, diet, socioeconomic status, and exposure to other agents	
<a href="#">Vlajinac <i>et al.</i> (2006)</a> Hospital-based case-control study Serbia & Montenegro 1998–2000	Oropharynx (146)	100 consecutive incident cases at the Clinical Centre of Serbia	100 controls among patients treated during the same period for non-malignant diseases of the head/neck, matched on age ( $\pm 2$ yr), sex and place of residence	Occupational exposure to various chemicals, dust and other agents	Exposure to wood dust <sup>1</sup> Occupational exposure to wood dust <sup>2</sup>	2.3 (1.0–5.7) 4.2 (1.5–11.9)	<sup>1</sup> Education, BMI, smoking, alcohol, family history of cancers <sup>2</sup> Smoking, dental diseases, HSV infection, smoking, alcohol	
<a href="#">Jayaprakash <i>et al.</i> (2008)</a> Hospital-based case-control study Buffalo, NY, USA and Germany 1982–98	Sinonasal & nasopharynx & hypopharynx (160, 147, 148)	90 incident cases in men diagnosed at Roswell Park Cancer Institute	1522 controls	Self reported exposures about prior exposure to wood dust at work	Moderate exposure High exposure Occasionally exposed Regularly exposed	1.5 (0.9–1.5) 1.35 (0.4–4.6) 1.45 (0.85–2.5) 1.6 (0.75–3.3)	Age, sex, tobacco, education, year of enrollment	

BMI, body mass index; CI, confidence interval; HLA, human leukocyte antigen; HSV, herpes simplex virus; mo, month or months; yr, year or years

cancer of the hypopharynx among men over the age of 55 years (OR, 2.1; 95%CI: 1.2–3.7), and a decreased risk among men under 55 years (OR, 0.4; 95%CI: 0.2–1.2). [Vlajinac et al. \(2006\)](#) observed an increased risk of cancer of the oropharynx (OR, 2.3; 95%CI: 1.0–5.7) associated with wood dust in Serbia and Montenegro.

All five cohort studies published in the period following the previous *IARC Monograph* provided results for cancer of the pharynx, although none provided results for subsites of other than the nasopharynx. The pooled re-analysis of five previously published cohort studies ([Demers et al., 1995b](#)) observed slightly fewer cases of cancer of the pharynx than expected (SMR, 0.8; 95%CI: 0.5–1.3). [Stellman et al. \(1998\)](#) also found no evidence of an excess risk associated with self-reported wood dust exposure or longest occupation among participants in the Cancer Prevention Study II (RR, 0.9; 95%CI: 0.4–2.0). [Innos et al. \(2000\)](#) found an excess risk of cancer of the pharynx among Estonian furniture workers exposed to both medium levels (SIR, 4.0; 95%CI: 0.8–11.8) and high levels of exposure (SIR, 1.5; 95%CI: 0.6–3.3). [Dement et al. \(2003\)](#) observed slightly more cases of cancer of the pharynx than expected among members of the US carpenters union (SMR, 1.4; 95%CI: 0.7–2.4). [Purdue et al. \(2006\)](#) observed a somewhat reduced risk among Swedish construction workers exposed to wood dust versus those who were not (RR, 0.6; 95%CI: 0.2–1.6,  $n = 4$ ).

## 2.4 Cancer of the larynx

The previous *IARC Monograph* reviewed ten case-control studies of cancer of the larynx (see Table 27, [IARC, 1995](#)). The majority had some indication of an excess risk associated with either wood dust exposure (1/2 studies) or wood-related occupations (7/8 studies), although sometimes based on small numbers. The studies were conducted in the USA ( $n = 7$ ), Europe ( $n = 2$ ), and the People's Republic of China ( $n = 1$ ), and the

majority of these studies adjusted for the effects of smoking. No support for this association was found in the cohort studies (see Table 18, [IARC, 1995](#)). The five cohort studies that reported results for cancer of the larynx observed fewer cancers than expected.

Seven new case-control studies ([Table 2.6](#)) have published results relevant for the evaluation of cancer of the larynx. [Pollán & López-Abente \(1995\)](#) in a Spanish study observed an excess risk among woodworkers (OR, 2.7; 95%CI: 0.9–7.7) that increased with duration of employment. [Gustavsson et al. \(1998\)](#) observed a decreased risk for cancer of the larynx associated with wood dust in Sweden (OR, 0.5; 95%CI: 0.3–0.9). [Laforest et al. \(2000\)](#) observed no increased risk overall and no evidence of an association with duration or cumulative exposure in a french study (OR, 1.0; 95%CI: 0.6–1.7). [Elci et al. \(2002\)](#) also found no association with wood dust in a Turkish study (OR, 1.1; 95%CI: 0.8–1.4). [Berrino et al. \(2003\)](#) found an increased risk of cancer of the larynx among men over the age of 55 (OR, 1.7; 95%CI: 1.2–2.6), and a decreased risk among men under 55 (OR, 0.6; 95%CI: 0.3–1.1). [Ramroth et al. \(2008\)](#) reported an excess based on a checklist (OR, 2.1; 95%CI: 1.2–3.9), but a weaker association based on a method using a job-specific questionnaire (OR, 1.4; 95%CI: 0.8–2.5). [Jayaprakash et al. \(2008\)](#) reported an excess among men based on self-reported exposure (OR, 2.1; 95%CI: 0.9–5.0). Six of the seven studies adjusted for the potential effects of smoking and alcohol consumption, but the last only adjusted for smoking.

All five cohort studies published in the period following the previous *IARC Monograph* provided results for cancer of the larynx. The pooled re-analysis of five previously published cohort studies ([Demers et al., 1995b](#)) observed slightly fewer cases of cancer of the larynx than expected (SMR, 0.7; 95%CI: 0.4–1.0), and no association with probability of exposure. [Stellman et al. \(1998\)](#) observed a potential excess risk associated with self-reported wood

Table 2.6 Case-control studies of cancer of the larynx and exposure to wood dust

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Pollán &amp; López-Abente (1995)</a> Hospital-based case-control study Spain January 1982 to August 1985	Larynx (161)	50 male residents of Madrid with histologically confirmed squamous cell carcinomas diagnosed at Ramon y Cajal Hospital	1 hospital control (matched by sex, age, admission date excluding alcohol or tobacco-related conditions) and 1 population control (matched on sex, age, residential census sections at diagnosis)	Extensive job history up to 1 yr before diagnosis; subject was considered exposed at $\geq 1$ yr of employment	All woodworkers 1–20 yr > 20 yr	2.7 (0.9–7.7) 1.6 (0.4–5.9) 5.6 (1.2–27.6)	Age, tobacco and alcohol consumption, and other occupational groups	
<a href="#">Gustavsson <i>et al.</i> (1998)</a> Community-based case-control study Sweden 1 January 1988 to 31 January 1991	Larynx (161)	401 incident squamous cell carcinomas in all Swedish men aged 40–79 yr living in Stockholm or the southern health care region	Referents randomly selected from the base population and frequency-matched to the cases for region and age group (40–54, 55–64, 65–79 yr)	Exhaustive occupational history taken and exposure assessments were based on a literature survey of exposure data for different occupations	Ever exposed	0.5 (0.3–0.9)	Adjusted for region, age, alcohol consumption and smoking habits	

Table 2.6 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Laforest et al. (2000)</a> Hospital-based case-control study France 1 January 1989 to 30 April 1991;	Larynx (161)	296 primary incident squamous cell cancers diagnosed and histologically confirmed in 15 French hospitals; only men were included in the study	Controls were patients with primary cancers of different sites requiring the same medical environment as case cancers, selected by frequency matching on age and recruited between 1987 and 1991 in the same hospitals as the cases or similar hospitals nearby	Detailed lifetime occupational history taken and occupational exposures were assessed through a job-exposure matrix	<b>Wood dust</b> Ever exposed Probability of exposure: ≤ 70% > 70% Duration of exposure: < 6 yr 6–10 yr > 10 yr Cumulative level: Low (< 10) Medium (10–42) High (> 42)	1.0 (0.6–1.7) 0.9 (0.4–2.0) 1.1 (0.5–2.2) 1.4 (0.6–3.2) 0.5 (0.2–1.5) 1.0 (0.5–2.3)	Age, smoking, alcohol, exposure to formaldehyde, and mineral fibres	
<a href="#">Elci et al. (2002)</a> Hospital-based case-control study Turkey 1979–84	Larynx (161)	940 cases among men identified from patients admitted to the Oncology Treatment Center of the Social Security Agency Okmeydani Hospital in Istanbul	1519 referent patients with Hodgkin disease, soft tissue sarcoma, non-melanoma skin cancer, testis, bone and male breast cancer as well as a series of non-cancer subjects	Occupational history taken using a questionnaire, occupations coded and assessed using a job-exposure matrix developed for occupational dusts	Wood dust exposure Low intensity Med intensity High intensity Low probability Med probability High probability	1.1 (0.8–1.4) 0.8 (0.5–1.4) 1.4 (1.0–1.9) 0.8 (0.4–1.4) 1.3 (1.0–1.7) 1.4 (0.7–2.5) 0.4 (0.2–0.9)	Age, smoking, and alcohol consumption	

Table 2.6 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)*	Adjustment for potential confounders	Comments
<a href="#">Berrino et al. (2003)</a> Population-based case-control study Italy, France, Spain, Switzerland 1979–82	Endolarynx (161)	696 male incident endolarynx cases diagnosed in Calvados, France; Turin & Varese, Italy; Pamplona & Zaragoza, Spain; Geneva, Switzerland	2176 male population controls	Occupational history taken through specialist interview; occupational exposures assessed using a job-exposure matrix	< 55 yr of age: Possible exposure Probably exposure > 55 yr of age Wood dust exposure	0.5 (0.2–1.1) 0.6 (0.3–1.1) 1.7 (1.2–2.6)	Age, centre, tobacco, alcohol, diet, socioeconomic status, and exposure to other agents	
<a href="#">Jayaprakash et al. (2008)</a> Hospital-based case-control study Buffalo, NY, USA and Germany 1982–98	Larynx (161)	124 incident male cases diagnosed at Roswell Park Cancer Institute	1522 controls	Self reported exposures about prior exposure to wood dust at work	Moderate exposure High exposure Occasionally exposed Regularly exposed	0.8 (0.5–1.3) 2.1 (0.9–4.96) 0.8 (0.4–1.4) 1.5 (0.8–2.8)	Age, sex, tobacco, education, year of enrollment	
<a href="#">Ramroth et al. (2008)</a> Population-based case-control study South-western Germany 1998–2000	Larynx (161)	257 histologically confirmed incident larynx cancer cases in men and women diagnosed in Rhein-Neckar-Odenwald region	769 population controls	Occupational history taken through specialist interview; occupational exposures assessed using exposure substance check-list (SCL), detailed occupational history, supplementary job-specific questionnaires (JSQ)	SCL: Wood dust Hardwood dust Softwood dust JSQ: Wood dust Hardwood dust Softwood dust	2.1 (1.2–3.9) 2.6 (1.3–5.2) 2.2 (1.1–4.2) 1.4 (0.8–2.5) 1.2 (0.6–2.5) 1.5 (0.7–2.8)	Adjusted for age, sex, tobacco, alcohol, education	

CI, confidence interval; RR, relative risk; yr, year or years

dust exposure (RR, 1.6; 95%CI: 0.8–3.4,  $n = 8$ ), but not for wood occupations (RR, 1.2; 95%CI: 0.3–4.9,  $n = 2$ ) among participants in the Cancer Prevention Study II. [Innos et al. \(2000\)](#) observed fewer cases of cancer of the larynx than expected (SIR, 0.7; 95%CI: 0.3–1.4) among male Estonian furniture workers. [Dement et al. \(2003\)](#) observed slightly more cases of cancer of the larynx than expected among members of the US carpenters union (SMR, 1.2; 95%CI: 0.7–2.0). [Purdue et al. \(2006\)](#) observed a somewhat reduced risk among Swedish construction workers exposed to wood dust versus those who were not (RR, 0.8; 95%CI: 0.5–1.5).

Recent registry-based studies also presented results for wood dust and cancer of the larynx. No excess was observed among woodworkers in a large Nordic census-based cancer incidence linkage study ([Pukkala et al., 2009](#)). [Arias Bahia et al. \(2005\)](#) observed a slight excess of cancer of the larynx in a Brazilian cancer registry and mortality study. [Laakkonen et al. \(2006\)](#) found no relationship with wood dust exposure in a Finnish cancer registry study.

## 2.5 Cancer of the lung

The Working Group for the previous *IARC Monograph* reviewed 24 case-control studies of cancer of the lung (see Table 28, [IARC, 1995](#)). Roughly half had some indication of an excess risk associated with either wood dust exposure or wood-related occupations. The studies were conducted in North America ( $n = 11$ ), Europe ( $n = 9$ ), Asia ( $n = 3$ ), and New Zealand ( $n = 1$ ). No support for this association was found in the cohort studies (see Table 18, [IARC, 1995](#)). The seven cohort studies that reported results for cancer of the lung observed a similar number of cancers to that expected.

Three new case-control studies ([Table 2.7](#)) have published results relevant for the evaluation of cancer of the lung. [Wu et al. \(1995\)](#) observed an increased risk of non-small cell lung cancers

among African- and Mexican-American men. [Matos et al. \(2000\)](#) observed an increased risk for lung cancer among sawmill workers, but not other woodworkers in Argentina. [Barcenas et al. \(2005\)](#) observed an excess of lung cancer associated with wood-related occupations or self-reported exposure in an American case-control study. All results were adjusted for smoking.

Four of the five cohort studies published in the period following the previous *IARC Monograph* provided results for cancer of the lung. The pooled re-analysis of five previously published cohort studies ([Demers et al., 1995b](#)) observed slightly fewer cases of cancer of the lung than expected (SMR, 0.8; 95%CI: 0.7–0.9). [Stellman et al. \(1998\)](#) observed a slight excess risk associated with self-reported wood dust exposure (RR, 1.2; 95%CI: 1.0–1.3), but not for wood occupations (RR, 1.1; 95%CI: 0.9–1.4) among participants in the Cancer Prevention Study II. [Innos et al. \(2000\)](#) observed an increased risk among highly exposed female (SIR, 1.6; 95%CI: 0.8–2.9), but not among male Estonian furniture workers (SIR, 1.0; 95%CI: 0.8–1.3). [Dement et al. \(2003\)](#) observed an excess among members of the US carpenters union (SMR, 1.5; 95%CI: 1.2–1.7). In a nested case-control study of Polish pulp and paper mill workers, [Szadkowska-Stańczyk & Szymczak \(2001\)](#) observed an excess of lung cancer associated with wood dust exposure (OR, 2.1; 95%CI: 0.9–4.9), but no evidence of an exposure-response relationship.

Recent registry studies also presented results for wood dust and lung cancer. A small excess was observed among women (SIR, 1.2; 95%CI: 1.1–1.4) but not among men (SIR, 0.96; 95%CI: 0.94–0.97) in a large Nordic census-based cancer incidence linkage study ([Pukkala et al., 2009](#)). [Arias-Bahia et al. \(2005\)](#) observed mixed results in Brazil; a slight excess in the cancer registry study and a decreased risk in the mortality study. [Laakkonen et al. \(2006\)](#) found no relationship with wood dust exposure in a Finnish cancer registry study.

Table 2.7 Case-control studies of cancer of the lung and exposure to wood dust

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
<a href="#">Wu <i>et al.</i> (1995)</a> Hospital-based case-control study USA	Lung (162)	113 African-American and 67 Mexican-American cases with newly diagnosed lung cancer recruited from the hospitals in Houston and San Antonio, Texas	270 healthy controls without prior histories of cancer from community centres, cancer screening programmes, churches and employee groups frequency-matched on age, ethnicity and sex	Occupational histories collected by interview, self-reported occupational exposure to wood dust	Wood dust exposure African-American: Non-small cell lung cancer Small cell lung cancer Mexican-American: Non-small cell lung cancer Small cell lung cancer	3.5 (1.4–8.6) 4.8 (1.2–18.5) 0.7 (0.0–12.4) 3.8 (0.8–17.4) 0.3 (0.0–6.2)	Age, sex, mutagen sensitivity, and pack-yr (smoking)	.
<a href="#">Matos <i>et al.</i> (2000)</a> Hospital-based case-control study Argentina 1994–96	Lung (162)	199 male patients residents in the city or in the province of Buenos Aires and admitted for treatment in any of 4 hospitals of Buenos Aires city	393 controls; 2 male control subjects hospitalized for conditions unrelated to tobacco use during the same period, and residents in the same area, matched by hospital and age ( $\pm$ 5yr)	Occupational history obtained by interview; occupational exposure assessed by job-exposure matrix	Occupation in: Sawmills or wood mills Furniture Woodworkers (carpenters, cabinet-makers, machine operators)	4.8 (1.2–19.0) 1.0 (0.4–2.2) 0.7 (0.3–1.5)	Age group, hospital, pack-yr and industries with $P < 0.05$	

Table 2.7 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	Relative risk (95% CI)*	Adjustment for potential confounders	Comments
<a href="#">Barcenas <i>et al.</i> (2005)</a> Hospital-based case-control study USA July 1995 to October 2000	Lung (162)	1368 men and women with incident histologically confirmed lung cancer diagnosed at the University of Texas Cancer Center	1192 cancer-free enrollees of private multispecialty clinics; matched on gender and ethnic groups	Longest held occupation or industry, self-reported wood dust exposure obtained by interview; minimum of 1 yr	Wood related occupation/industry Self-reported wood dust exposure Occupation, industry, or self-reported exposure: Lung (adenocarcinoma) Non-small cell lung carcinoma (excluding adenocarcinoma) Small cell lung carcinoma	3.2 (1.5–6.9) 1.5 (1.2–2.1) 1.5 (1.0–2.1) 1.9 (1.3–2.7) 1.1 (0.5–2.3)	Adjusted for age, gender, ethnicity, smoking status, and place of residence	
<a href="#">Jayaprakash <i>et al.</i> (2008)</a> Hospital-based case-control study Buffalo, NY, USA and Germany 1982–98	Lung (162)	809 incident male cases diagnosed at Roswell Park Cancer Institute	1522 controls	Self reported exposures about prior exposure to wood dust at work	Moderate exposure High exposure Occasionally exposed Regularly exposed	1.1 (0.9–1.4) 2.15 (1.3–3.6) 1.1 (0.8–1.4) 1.7 (1.2–2.4)	Age, sex, tobacco, education, year of enrollment	

## 2.6 Other cancer sites

The results for other cancer sites were reviewed, but were less consistent than for the respiratory tract. The results for case–control studies for wood dust that were published subsequent to the previous *IARC Monograph* are presented in [Table 2.8](#).

## 2.7 Furniture and cabinet-making industry

The Working Group also addressed the carcinogenic risk associated with the furniture and cabinet-making industry that was evaluated in the previous *IARC Monograph* Volume 25 ([IARC, 1981](#)), and reassessed in Supplement 7 ([IARC, 1987](#)) when it was classified as *carcinogenic to humans (Group 1)*. Since then, new studies and pooled analyses have strengthened the association between working in this industry and sinonasal and nasopharyngeal cancers, including [Fukuda & Shibata \(1988\)](#), [Minder & Vader \(1988\)](#), [Magnani \*et al.\* \(1993\)](#), [Demers \*et al.\* \(1995a, b\)](#) and [Bouchardy \*et al.\* \(2002\)](#). Such studies are listed among others published since 1980 in [Table 2.9](#) available at <http://monographs.iarc.fr/ENG/Monographs/vol100C/100C-10-Table2.9.pdf>; [Table 2.10](#) available at <http://monographs.iarc.fr/ENG/Monographs/vol100C/100C-10-Table2.10.pdf>; [Table 2.11](#) available at <http://monographs.iarc.fr/ENG/Monographs/vol100C/100C-10-Table2.11.pdf>; [Table 2.12](#) available at <http://monographs.iarc.fr/ENG/Monographs/vol100C/100C-10-Table2.12.pdf>; [Table 2.13](#) available at <http://monographs.iarc.fr/ENG/Monographs/vol100C/100C-10-Table2.13.pdf>; [Table 2.14](#) available at <http://monographs.iarc.fr/ENG/Monographs/vol100C/100C-10-Table2.14.pdf>; [Table 2.15](#) available at <http://monographs.iarc.fr/ENG/Monographs/vol100C/100C-10-Table2.15.pdf>; and [Table 2.16](#) available at <http://monographs.iarc.fr/ENG/Monographs/vol100C/100C-10-Table2.16.pdf>.

From reviewing the studies in [Tables 2.9](#) to [2.16](#) together with the data on exposure to wood dust in [Tables 2.1](#) to [2.8](#), the Working Group attributed the causal association between working in the furniture and cabinet-making industry and sinonasal and nasopharyngeal cancers to wood dust.

Another possible association observed in the industry included excesses of pleural malignant mesothelioma, which is most likely the result of asbestos exposure. Another possible excess of haematopoietic malignancies may be the result to other exposures such as solvents. Relevant results are presented in [Tables 2.11](#), [2.13](#) and [2.15](#) (online), but the Working Group considered data for these sites to be inconsistent and inadequate for evaluation.

## 2.8 Synthesis

There is consistent and strong evidence from both case–control studies and large cohort studies that wood dust causes sinonasal cancer. Most of these studies do not specify the histology of the tumours. Among the case–control that specified histology, very large excess risks were observed for sinonasal adenocarcinoma and wood dust exposure. Case series have found a large proportion of adenocarcinoma cases to be woodworkers.

There is also weaker evidence that wood dust causes cancer of the nasopharynx. The majority of case–control studies observed an increased risk of cancer of the nasopharynx associated with wood dust exposure or with employment in wood-related occupations, although often based on small numbers. This is supported by the pooled re-analysis of cohort studies where a strong association was observed with probability of wood dust exposure. The primary confounder of concern was formaldehyde exposure, but in the pooled cohort study the probability of wood dust exposure, which would likely be inversely correlated with formaldehyde exposure, was

**Table 2.8 Case-control studies of other cancers and exposure to wood dust**

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)* OR	Adjustment for potential confounders	Comments
<a href="#">Fritschi &amp; Siemiatycki (1996)</a> Population-based case-control study Canada 1979–85	Non-Hodgkin lymphoma (200, 202)	3730 male cases aged 35–70 yr, resident in Montreal, histologically confirmed non-Hodgkin lymphoma, Hodgkin disease, or myeloma	533 colorectal, bladder, prostate, stomach, kidney, melanoma, pancreas and oesophageal cancer controls. 533 population controls selected by electoral lists or random-digit dialling	Occupational history obtained by interview or questionnaire	Wood dust exposure: Non-substantial Substantial	0.5 (0.3–0.8) 0.8 (0.5–1.3)	Age, proxy status, income (quintiles), ethnicity	Results for wood dust only presented for non-Hodgkin lymphoma
<a href="#">Cocco et al. (1998)</a> Census-linked case-control study USA 1984–92	Gastric cardia (151.1)	1056 cases of gastric cardia cancer were identified in men aged 25 yr or more using death certificates from 24 states	5280 control subjects were identified the same way but who died of non-malignant disease; 5:1 match on region, sex, race, and age	Usual occupation obtained from death certificates, exposure was assessed using a job-exposure matrix	Wood dust exposure: Unexposed All exposed Low level exposure Med level exposure High level exposure	1.0 (reference) 0.8 (0.6–1.1) 0.9 (0.6–1.4) 0.7 (0.5–1.2) 1.0 (0.5–2.2)	Matched on region, sex, race, and age	

Table 2.8 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)* OR	Adjustment for potential confounders	Comments
<a href="#">Cocco et al. (1999)</a> Census-linked case-control study USA 1984-96	Stomach (151)	41957 deaths of stomach cancer aged 25+ yr using death certificates from 24 states	83914 controls who died of non-malignant disease; 2:1 match on region, sex, race, and age ( $\pm 5$ yr)	Usual occupation obtained from death certificates, exposure was assessed using a job-exposure matrix	White men: Med probability High probability Med intensity High intensity African-American men: Med probability High probability Med intensity High intensity White women: Med probability High probability Med intensity High intensity African-American women: Medium probability Medium intensity High intensity	0.9 (0.8-1.1) 1.0 (0.9-1.1) 1.0 (0.9-1.1) 0.9 (0.7-1.1)  1.0 (0.7-1.3) 0.9 (0.8-1.2) 1.1 (0.9-1.3) 0.8 (0.6-1.0)  0.7 (0.4-1.2) 0.8 (0.3-2.2) 0.9 (0.7-1.2) 0.7 (0.3-1.6)  1.3 (0.5-3.8) 0.9 (0.5-1.6) 0.4 (0.1-3.4)	Matched on region, sex, race, and age	

Table 2.8 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)* OR	Adjustment for potential confounders	Comments
<a href="#">Mao <i>et al.</i> (2000)</a> Registry-linked case-control study Canada 1994–97	Non-Hodgkin lymphoma (200, 202)	1469 histologically confirmed incident cases (764 men, 705 women) of non-Hodgkin lymphoma diagnosed in 8 Canadian provinces who were 20–74 yr of age	5073 controls frequency matched on age and sex randomly selected from within the same provinces via Provincial Health Insurance Plans, Property Assessment databases, or random-digit dialling	Home or work exposure to 17 chemicals was obtained through questionnaires or interviews	Wood dust exposure: Men Women Never exposed 1–6 yr exposure ≥ 7 yr exposure	0.9 (0.8–1.1) 1.4 (1.0–2.0) 1.0 (reference) 1.2 (0.7–1.9) 1.7 (1.1–2.6)	10-yr age groups, province, BMI (< 20, 20–27, > 27), consumption of milk	
<a href="#">De Roos <i>et al.</i> (2001)</a> Population-based case-control study Canada & USA 1992–94	Neuroblastoma	538 incident cases under 19 yr of age at 139 participating hospitals	504 cases were identified through random-digit dialling individually caliper-matched to cases on date of birth	Telephone interviews with parents for maternal and paternal occupational history. Self-reported exposure assessed by an industrial hygienist (IH)	Wood dust exposure: Paternal occupational exposure Self-reported exposure IH-reviewed exposure	1.4 (0.8–2.3) 1.5 (0.8–2.8)	Child's age, maternal race, maternal age, and maternal education	

Table 2.8 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)* OR	Adjustment for potential confounders	Comments
<a href="#">Briggs et al. (2003)</a> Population-based case-control study USA 1984-88	Non-Hodgkin lymphoma (200, 202) Hodgkin disease (201)	1511 non-Hodgkin lymphoma, 343 Hodgkin disease cases diagnosed among African-American and white men born 1929-53, from Atlanta, Detroit, Connecticut, Iowa, Kansas, Miami, San Francisco, Seattle	1910 controls with no history of the selected cancer identified by random-digit dialling and frequency-matched by birth year, and geographic region of cancer registry	Occupational history collected by professional interviewers	Wood dust exposure: Non-Hodgkin lymphoma: African-American White Hodgkin disease: African-American White	1.4 (0.7-2.8) 1.1 (0.9-1.3) 4.6 (1.6-13.3) 0.9 (0.7-1.3)	Age and cancer registry.	
<a href="#">Fritschi et al. (2005)</a> Population-based case-control study Australia January 2000-August 2001	Non-Hodgkin lymphoma (200, 202)	Incident cases of non-Hodgkin lymphoma diagnosed in New South Wales or the Australian Capital Territory; aged 20-74 yr	Controls were randomly selected from the New South Wales and Australian Capital Territory Electoral Rolls, frequency matched on age, sex and region of residence	Lifetime occupational history obtained by telephone interview & mailed questionnaire. Exposure assessment done blindly by an occupational hygienist and a job-exposure matrix	Hardwood dust exposure: Non-substantial Substantial Softwood dust exposure: Non-substantial Substantial	1.5 (0.9-2.4) 1.7 (1.0-2.9) 1.2 (0.6-2.7) 1.6 (1.1-2.6) 1.7 (1.0-2.8) 1.6 (0.7-3.8)	Adjusted for age, sex, state and ethnic origin	

Table 2.8 (continued)

Reference, study location and period	Organ site (ICD code)	Characteristics of cases	Characteristics of controls	Exposure assessment	Exposure categories	RR (95%CI)* OR	Adjustment for potential confounders	Comments
<a href="#">Pan <i>et al.</i> (2005)</a> Registry-linked case-control study Canada 1994–97	Brain (191)	1009 incident cases of histologically confirmed primary brain cancer from 8 provinces	5039 population control subjects aged 20–76 yr collected in the same study area	Occupational history obtained through questionnaires. Self-reported exposure	Wood dust exposure: Men Women Both sexes	1.3 (1.9–1.4) 1.1 (0.8–1.7) 1.2 (1.0–1.4)	Age, province of residence, education, alcohol intake, total energy intake, smoking pack-yr, and sex	
<a href="#">Fritschi <i>et al.</i> (2007)</a> Population-based case-control study Australia January 2001–August 2002	Prostate (185)	606 histologically confirmed cases in Western Australia, aged 40–75 yr. 402 cases of benign prostatic hyperplasia identified from hospital records	471 controls aged 45–75 yr randomly selected from the Western Australia electoral roll August 2001–October 2002; frequency-matched on 5 yr age groups	Occupational history obtained by questionnaires and interviews. Exposure was assessed for each occupation by an occupational hygienist for probability, frequency and total dose.	Wood dust exposure: Prostatic cancer– Not exposed Non-substantial Substantial Benign prostatic hyperplasia– Not exposed Non-substantial Substantial	1.0 (reference) 1.1 (0.8–1.4) 1.2 (0.5–2.6) 1.0 (reference) 1.1 (0.8–1.4) 0.8 (0.4–1.4)	Adjusted for age	

BMI, body mass index; yr, year or years

associated with nasopharyngeal cancer risk, and an excess was observed among both the furniture workers and plywood workers subcohorts.

There was weaker evidence for other sites such as the pharynx, larynx, and lung. Although positive associations were observed in some case-control studies, the pattern was not as consistent and not supported by positive findings in cohort studies.

The great majority of studies did not report on the specific tree species to which workers were exposed or whether exposure was due primarily to hardwoods or softwoods. The few studies that did address tree species were relevant only for the evaluation of sinonasal cancer. There is strong evidence for an association between sinonasal cancer and exposure to hardwood dusts, based on the results of the few studies that specifically assessed exposure to hardwoods and on the results of case series that identified specific tree species. Among the few case-control studies that assessed the relationship with softwoods, there was a consistent excess risk, but the magnitude of the excess was small in comparison to hardwoods, and the association was primarily with squamous cell carcinoma.

### 3. Cancer in Experimental Animals

Only a limited number of studies in experimental animals have been published on the carcinogenicity of wood dust. Studies described below include those summarized in the previous *IARC Monograph* ([IARC, 1995](#)) as well as studies published since.

#### 3.1 Inhalation

##### 3.1.1 Rat

An inhalation study to determine the carcinogenicity of inhaled oak wood dust with and without wood preservatives was conducted in

rats. Six groups of 58–61 female F344 rats were exposed to: 1) 18 mg/m<sup>3</sup> of untreated oak wood dust; 2) wood preservatives containing 1 µg/m<sup>3</sup> lindane and 0.2 µg/m<sup>3</sup> pentachlorophenol (PCP); 3) oak wood dust treated with lindane and PCP; 4) 21 µg/m<sup>3</sup> of sodium dichromate; 5) oak wood dust treated with chromate (wood contained the equivalent of 39 µg/m<sup>3</sup> chromate); and, 6) 72 µg/m<sup>3</sup> of *N*-nitrosodimethylamine (positive control). A group of 115 rats were sham-exposed (negative control). Approximately 24 rats/group were exposed for 25 weeks and approximately 36 rats/group were exposed for their lifespan. The particle size was reported as 2–7 µm. The untreated wood dust contained up to 5 µg/m<sup>3</sup> of chromate. No respiratory tract tumours were observed in the negative controls. The positive control group of animals exposed for their lifespan had an incidence of 12/35 nasal cavity tumours. Respiratory tract tumours occurred less frequently in animals exposed for only 25 weeks than in those exposed for their lifespan. The only significant finding in rats exposed for 25 weeks was an increased incidence over controls of benign tumours of organs other than the respiratory tract in the group exposed to chromate aerosol alone. In the rats exposed to untreated oak wood for their lifespan, 2/36 rats developed malignant tumours of the respiratory tract (one in the oral cavity, one bronchial carcinoma, but none in the nasal cavity); there were no benign tumours of the respiratory tract. There was 1/37 animals exposed to wood dust treated with chromate stain, and 1/34 animals exposed to chromate aerosol for their lifespan that had a nasal cavity tumour, but none were found in the rats exposed to untreated wood dust ([Klein et al., 2001](#)).

Sixteen female Sprague Dawley rats were exposed to 25 mg/m<sup>3</sup> of untreated beech wood dust (70%, ≤ 10 µm; 10–20%, ≤ 5 µm) for 6 hours/day, 5 days/week for 104 weeks. There were 16 untreated controls. In the 15 surviving exposed rats and 15 control rats, no respiratory tract

tumours were observed. Incidences of non-respiratory tract tumours did not differ between untreated and exposed rats ([Holmström \*et al.\*, 1989](#)).

Fifteen female Wistar rats were exposed to 15.3 mg/m<sup>3</sup> of beech wood dust (mass median aerodynamic diameter [MMAD], 7.2 µm; geometric standard deviation [GSD], 2.2) for 6 hours/day, 5 days/week, for 6 months, and were observed for up to 18 months. No respiratory tract tumours were found in exposed rats or in 15 untreated controls. The incidence of non-respiratory tract tumours did not differ between exposed rats and untreated controls ([Tanaka \*et al.\*, 1991](#)).

### 3.1.2 Hamster

One group of 12 and one group of 24 male Syrian golden hamsters were exposed to either 15 or 30 mg/m<sup>3</sup> beech wood dust (70%, ≤ 10 µm; 10–20%, ≤ 5 µm) for 6 hours/day, 5 days/week for either 36 or 40 weeks, respectively. One group of 12 and one group of 24 animals served as untreated controls. No respiratory tract tumours were observed in the 12 animals exposed to 15 mg/m<sup>3</sup>, but 1/22 animals exposed to 30 mg/m<sup>3</sup> had an unclassifiable infiltrating malignant nasal tumour (not significantly different from controls) ([Wilhelmsson \*et al.\*, 1985a, b](#)). [The Working Group noted that in the above inhalation studies, the size of the dusts was quite large, which might allow some deposition in the upper respiratory tract, but very little deposition in the lower respiratory tract. No measurement of deposition was made, so the actual exposure is unknown.]

## 3.2 Intraperitoneal injection

### 3.2.1 Rat

Female Wistar rats received three weekly intraperitoneal injections of beech wood dust [total dose reported as 250 or 300 mg/animal]

suspended in saline, and were held for 140 weeks. No mesotheliomas or sarcomas were reported in the 52 rats examined ([Pott \*et al.\*, 1989](#)). [The Working Group noted the limited reporting of the study. No details on the number of starting animals or on particle size were given.]

## 3.3 Administration with known carcinogens or other modifying factors

### 3.3.1 Rat

Four groups of 16 female Sprague-Dawley rats were exposed 6 hours/day, 5 days/week for 104 weeks by inhalation to: air (control); 25 mg/m<sup>3</sup> untreated beech wood dust (70%, ≤ 10 µm; 10–20%, ≤ 5 µm); 14.9 mg/m<sup>3</sup> formaldehyde; or wood dust plus formaldehyde. Metaplastic or dysplastic lesions were observed in rats exposed to formaldehyde with or without wood dust, but the incidences between both groups were not statistically different. No such lesions were observed in control rats or in rats exposed to wood dust alone. No respiratory tract tumours were observed in rats exposed to wood dust or to wood dust plus formaldehyde ([Holmström \*et al.\*, 1989](#)).

Two groups of 20 male Wistar rats were exposed by inhalation to air (control); or 15 mg/m<sup>3</sup> of beech wood dust (MMAD, 7.2 µm; GSD, 2.2) for 6 hours/day, 5 days/week for 6 months. Thereafter, five rats per groups were exposed to 10.2 mg/m<sup>3</sup> of sidestream cigarette smoke for 2 hours/day, 5 days/week for 1 month. The experiment was terminated 18 months after the start of the exposures. No tumours of the respiratory tract were observed ([Tanaka \*et al.\*, 1991](#)).

### 3.3.2 Hamster

Two groups of 12 male Syrian golden hamsters were exposed by inhalation to air (control) or 15 mg/m<sup>3</sup> beech wood dust (70%, ≤ 10 µm; 10–20%, ≤ 5 µm) for 6 hours/day, 5 days/week for 36 weeks. Another two groups of hamsters were treated similarly but also received weekly subcutaneous injections of 1.5 mg *N*-nitrosodiethylamine (NDEA) for the first 12 consecutive weeks. No nasal tumours were observed in the four groups. Tracheal squamous cell papilloma incidences were: 1/7, controls; 0/8, wood dust; 3/8, NDEA; 4/8, NDEA plus wood dust ([Wilhelmsson et al., 1985a, b](#)).

Two groups of 24 male Syrian golden hamsters were exposed by inhalation to air or 30 mg/m<sup>3</sup> beech wood dust (70%, ≤ 10 µm; 10–20%, ≤ 5 µm) for 6 hours/day, 5 days/week for 40 weeks. Another two groups of hamsters were treated similarly but received weekly subcutaneous injections of 3 mg NDEA for the first 12 consecutive weeks. No respiratory tract tumours were found in control animals. The incidence of these tumours did not differ between the groups treated with NDEA or NDEA plus wood dust ([Wilhelmsson et al., 1985a, b](#)).

### 3.4 Exposure to wood dust extracts

In a lifetime experiment, four groups of 70 female NMRI mice weighing 25–30 g [age unspecified] received skin applications of a mutagenic fraction of a methanol extract of beech wood dust in 30 µL acetone twice a week for 3 months. Positive and negative controls were included in the study ([Table 3.1](#)). No effect on survival was observed between the treated groups and the negative control groups. A comparison between mice treated with wood dust extract and mice serving as negative controls indicated an overall carcinogenic effect ( $P < 0.01$ ,  $\chi^2$  test) ([Mohtashamipur et al., 1989](#)). [The Working Group also noted a dose-dependent increase in

the incidence of skin squamous cell papillomas and carcinomas combined or papillomas alone.]

Four groups of 50 male and female Kunming mice were intragastrically administered 0, 1, 2 or 4 g/kg body weight of a water extract of birch wood dust, once a week for 5 weeks. Thereafter, mice were given 0.5% butylated hydroxytoluene for 3 weeks in the diet. The experiment was terminated at experimental Week 15. There was a dose-dependent increase ( $P < 0.05$ ) in lung tumour incidence (0/50, 2/49, 4/48, 7/49, respectively), and multiplicity (0, 0.04, 0.15, 0.24 tumour/mouse, respectively). No significant increase was observed in a similar experiment using an organic extract of birch wood dust ([He et al., 2002](#)).

### 3.5 Exposure to wood shavings

Studies directed at testing the potential carcinogenicity of cedar shavings were inadequate in that they did not have control groups ([Vlahakis, 1977](#); [Jacobs & Dieter, 1978](#)).

### 3.6 Synthesis

Several of the studies investigating the carcinogenicity of inhaled wood dust in rats and hamsters used particles with relatively large MMADs, a design that would enhance deposition in the upper respiratory tract, including the nasal cavity. Despite this design, the results of the animal studies do not confirm the nasal carcinogenicity of wood dust observed in humans. No measurement of the actual deposition of wood dust in the respiratory tract was made, and therefore the amount of the exposure is unknown.

In one study in mice, a methanol extract of beech wood dust was tested by skin application. Although a dose-dependent increase in the incidence of skin tumours was observed, this result cannot be used in the evaluation of the carcinogenicity in experimental animals of wood dust per se.

**Table 3.1 Study in mice exposed to mutagenic fractions of methanolic extracts of dust<sup>a</sup>**

Tumour	Negative controls			Extract (g)						Benzo[ <i>a</i> ]pyrene (µg)		
	Untreated (n = 43)	Shaven (n = 44)	Shaven, acetone-treated (n = 42)	2.5 (n = 43)	5.0 (n = 50)	7.5 (n = 46)	10.0 (n = 49)	5 (n = 43)	10 (n = 42)			
Skin squamous cell carcinomas	-	-	-	1	-	-	1 <sup>b</sup>	1	15			
Skin squamous cell papillomas	-	-	-	1	1	6	5 <sup>b</sup>	2	5			
Skin keratoacanthomas	-	-	-	-	-	1	-	-	2			
Skin papillary cystadenomas	-	-	-	-	1	-	-	-	-			
Sebaceous gland adenomas	-	-	-	-	-	-	-	2	-			
Mammary gland adenocarcinomas	-	-	-	-	4	3	2	1	1			
Mammary gland adenoacanthomas	-	-	-	-	-	-	1	-	-			
Mammary gland mixed tumours	-	-	-	-	-	-	2	-	-			
Fibrosarcomas	-	-	-	-	-	1	-	-	-			
Haemangiosarcomas	-	-	-	-	1	-	-	-	-			
Neurofibrosarcomas	-	-	-	-	1	-	-	-	-			
Lymphomas	-	-	-	-	-	-	1	-	-			
Anaplastic carcinomas	-	-	-	-	1	-	-	-	-			
Precancerous skin lesions	-	1	2	2	4	8	6	13	18			

<sup>a</sup> Dust from untreated, semidry beech wood

<sup>b</sup> [ $P < 0.01$ ; Cochran-Armitage test for trend] where comparisons are made for 0 (acetone-treated controls), 2.5, 5.0, 7.5 and 10 g extract groups, including squamous cell carcinomas and papillomas combined, or papillomas alone

Adapted from [Mohhtashamipur et al. \(1989\)](#), numbers of animals given are effective numbers

## 4. Other Relevant Data

### 4.1 Deposition and clearance of particulates in the nasal region

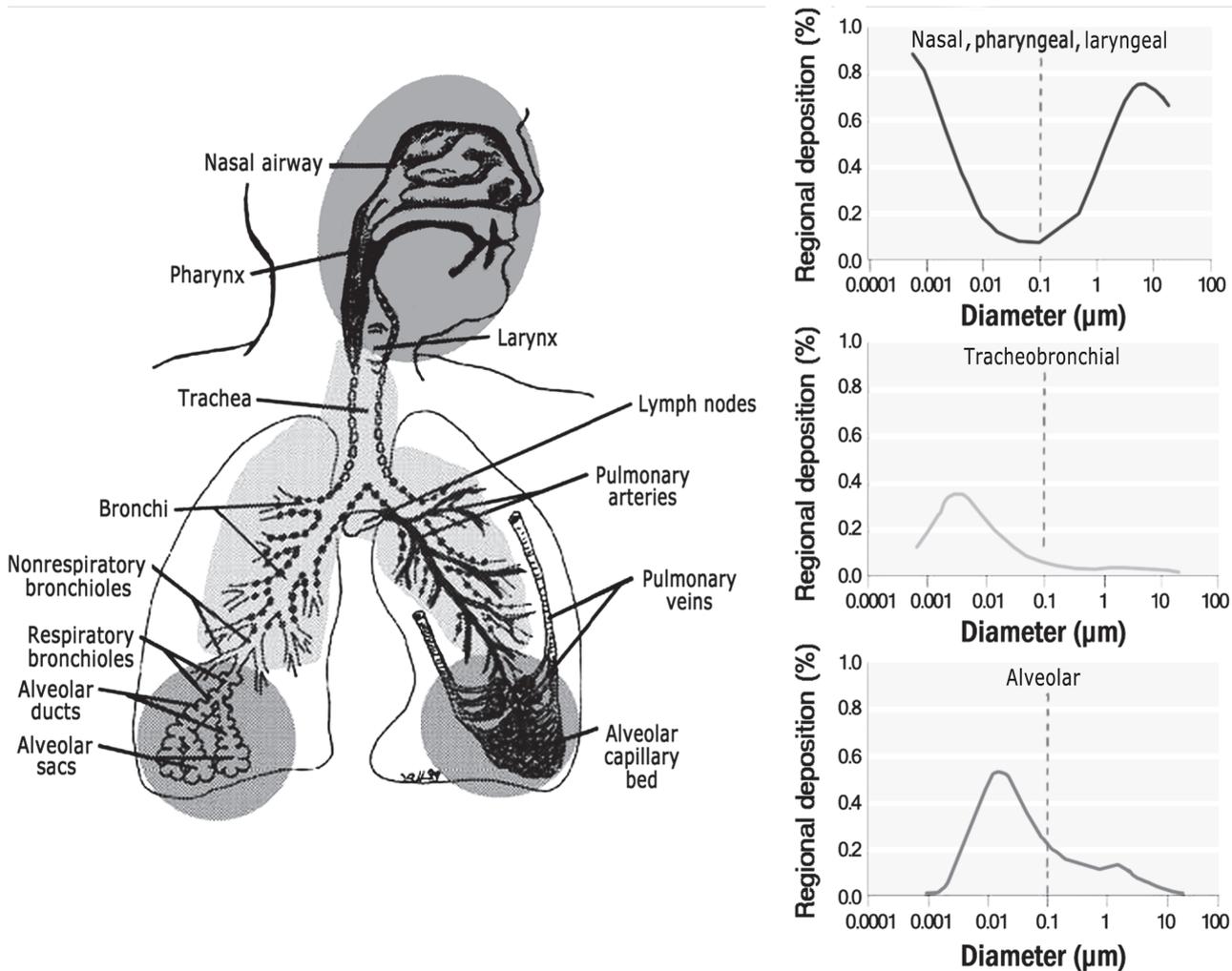
The anatomy and physiology of the upper respiratory tract is complex, and there are significant differences between rodents, non-human primates, and humans (reviewed by [Stuart, 1984](#); [Harkema, 1991](#)). Wood dust, leather dust, and metal-containing dusts are complex mixtures that have been associated with the development of sinonasal and nasopharyngeal cancers in humans ([IARC, 1995](#)). The nasal region is a primary target of inhaled toxicants. In humans, the particulate fraction of wood and leather dusts is considered to be responsible for carcinogenesis ([Fu et al., 1996](#); [Feron et al., 2001](#)). Particulate dosimetry in the upper respiratory tract depends on anatomy, airflow dynamics, and histology. Three-dimensional models have been developed to facilitate interspecies comparisons ([Anjilvel & Asgharian, 1995](#)). Humans vary in their breathing patterns at rest and at work; these patterns have an impact on the extent of nasal deposition of particles. In humans, coarse particles (2.5–10 µm) deposit by impaction in the nasal region; very fine particles (less than 0.01 µm in diameter) deposit in the nasopharynx by diffusion (Fig. 4.1; reviewed in [Oberdörster et al., 2005](#)). Coarse particles deposited in the nose are rapidly removed by sneezing, sniffing, and mucociliary clearance. However, some areas in the nasopharynx lack cilia, and particles deposited in these regions have longer retention times that can be up to several days ([Feron et al., 2001](#)).

### 4.2 Molecular pathogenesis

The histopathological classification of cancers arising in the sinonasal region (nasal cavity and paranasal sinuses) and in the nasopharynx varies with the anatomical location

and associated risk factors ([Rosai, 2004](#)). In the sinonasal region, benign tumours or sinonasal papillomas occur; the inverted papilloma subtypes may progress to malignant squamous cell carcinomas in 3–13% of cases ([Littman & Vaughan, 2006](#)). The most common malignant tumour in the sinonasal region is squamous cell carcinoma, which is usually associated with cigarette smoking (['t Mannetje et al., 1999](#)), and rarely following exposure to wood dust (see Section 2). Adenocarcinomas are strongly associated with exposure to wood and leather dusts ([Fu et al., 1996](#); [d'Errico et al., 2009](#)). Wood dust exposure was associated with a 21-fold [95%CI: 8.0–55.0] increase in the risk of having a sinonasal adenocarcinoma or a squamous cell carcinoma compared to not being exposed ([Bornholdt et al., 2008](#)). These occupationally related carcinomas have a unique histological appearance described as intestinal-type sinonasal adenocarcinoma (ITAC). The majority of ITACs are localized in the superior nasal cavity and ethmoid sinus. This cancer develops after a long latent period of 20–30 years of exposure to wood dust, and is locally invasive with rare distant metastases ([Llorente et al., 2009](#)). Other malignant sinonasal cancers include cylindrical (transitional) cell carcinoma, small cell neuroendocrine carcinoma, and undifferentiated (anaplastic) carcinoma ([Rosai, 2004](#)).

In the nasopharynx, the histopathological classification varies with age and associated risk factors ([Yu & Yuan, 2006](#)). Keratinizing squamous cell carcinomas occur at older ages, and the majority of nasopharyngeal carcinomas are non-keratinizing carcinomas, either differentiated or undifferentiated ([Rosai, 2004](#)). Non-keratinizing nasopharyngeal carcinomas are more common in high-risk populations in association with Epstein-Barr virus infection and other risk factors as discussed in Section 4.4.

**Fig. 4.1 Deposition of inhaled particles in the human respiratory tract during nasal breathing**

From [Oberdörster et al., \(2005\)](#). Drawing courtesy of J Harkema. Reproduced with permission from Environmental Health Perspectives.

#### 4.2.1 Cancer of the nasal cavity and paranasal sinuses

These cancers are extremely rare with only an overall annual incidence of approximately 1/100000 in Europe ([Muir et al., 1987](#)). There have been few studies of molecular and genetic alterations associated with the development of sinonasal cancers, and no link to chemical carcinogens has been established ([Saber et al., 1998](#)). Inverted papilloma is recognized as a preneoplastic lesion, and mutations in the *p53* tumour-suppressor gene have been associated with progression to squamous cell carcinoma. Epigenetic alterations

characterized by promoter hypermethylation have also been identified in sinonasal papilloma ([Stephen et al., 2007](#)). In ITACs of patients with known long-term exposure to wood or leather dust, *p14<sup>ARF</sup>* and *p16<sup>INK4a</sup>* promoter methylation was detected in 80% and 67% of cases, respectively ([Perrone et al., 2003](#)). In the same study, *p53* mutations were present in 44% (7/16 cases) of the ITACs, and in all but one case the mutations were G:C→A:T transitions in 86% of the cases, and involved the CpG dinucleotides in 50% of the cases. Loss of heterozygosity at chromosomal loci encoding the *p53* (locus 17p13), *p14<sup>ARF</sup>* and

*p16<sup>INK4a</sup>* (locus 9p21) genes were also reported in 58% and 45% of the cases, respectively ([Perrone et al., 2003](#)). *p53* Mutations were previously reported in only 18% (2/11) of sinonasal adenocarcinomas from patients with unknown exposure ([Wu et al., 1996](#)). *K-RAS* mutations were also reported in ITACs with a frequency of 13%, whereas the frequency was very low (1%) in squamous cell carcinoma ([Saber et al., 1998](#); [Bornholdt et al., 2008](#)). Strikingly, among the five mutations located in codon 12 of the *K-RAS* gene, the G→A transition was the most common, and was present in tumour tissue (adenocarcinoma) from two wood-dust-exposed patients and from one patient with unknown exposure ([Bornholdt et al., 2008](#)). [The Working Group noted that a clear link between exposure to wood or leather dust and specific G:C→A:T transitions in ITACs remains to be demonstrated.] Although ITACs resemble colonic adenocarcinomas histologically, alterations in *E-cadherin* and *β-catenin* genes characteristic of the APC pathway and alterations in mismatch-repair genes are rare in sinonasal adenocarcinomas ([Perez-Ordóñez et al., 2004](#)).

Unique patterns of chromosomal gains and losses have been associated with wood-dust-related ITACs ([Korinth et al., 2005](#); [Llorente et al., 2009](#)). Overexpression of c-erbB2 protein was found in one-third of cases ([Gallo et al., 1998](#)).

There are no identified precursor lesions leading to the development of ITACs, although hyperplasia, squamous metaplasia, and dysplasia occur frequently in areas adjacent to sinonasal carcinomas ([Llorente et al., 2009](#)). A morphological study of nasal biopsies from 139 leather workers employed for a median of 29 years revealed squamous metaplasia in 65% of cases, dysplasia in 41% of cases, and goblet cell hyperplasia in 22% of cases. The presence of goblet cell hyperplasia was associated with longer occupational exposures in leather-tanning activities ([Palomba et al., 2008](#)).

#### 4.2.2 Cancer of the nasopharynx

There are few studies of molecular alterations in cancer of the nasopharynx. Many genetic alterations (chromosomal gains and losses) have been described in endemic nasopharyngeal carcinomas, but none of these changes have been specifically linked to wood or leather dust exposure ([Hui et al., 1999](#); [Chan et al., 2002](#)).

### 4.3 Mechanisms of toxicity and carcinogenicity

#### 4.3.1 Tissue injury

Histopathological changes associated with tissue injury and repair (metaplasia, hyperplasia) are extremely common in the upper respiratory tract of experimental animals and humans. In rats, the inhalation of a wide range of volatile and semi-volatile industrial chemicals induces tissue injury, inflammation, and hyperplasia; however, there is no consistent association with subsequent development of nasal cancer. Inflammation, IgE-mediated allergic rhinitis associated with the inhalation of particulate antigens, and inflammatory sinonasal polyps are very common in humans, yet sinonasal cancers are rare as discussed in Section 4.2. Common histopathological changes found in the nasal epithelium include cuboidal and squamous metaplasia and hyperplasia of goblet cells and cylindrical cells. These reactive changes are not considered to be precursors for the development of neoplasia. It is possible that wood dust particles incite tissue injury by direct mechanical damage, although there are no experimental data to support this mechanism ([Feron et al., 2001](#)).

### 4.3.2 Impaired ciliary clearance and mucostasis

Heavy occupational exposure to wood dust has been reported to impair ciliary clearance, and to contribute to mucostasis (IARC, 1995). Theoretically, the impaired clearance of wood dust particles could lead to prolonged contact with the upper respiratory epithelium (Littman & Vaughan, 2006). Impaired mucociliary clearance may also allow particulate antigens to gain entry to nasal-associated lymphoid tissues, and enhance allergic sensitization (Feron *et al.*, 2001).

### 4.3.3 Direct genotoxicity

Direct genotoxic effects of wood dust extracts were summarized in IARC (1995). Overall, the mutagenic activity of beech and oak wood extracts was detected in bacterial systems and in rat hepatocytes *in vitro*. Several chemicals were isolated from wood extracts, but only quercetin and  $\Delta^3$ -carene were shown to be mutagenic (IARC, 1995). Exposure to hexavalent chromium has been associated with the development of sinonasal cancers (Sunderman, 2001).

Dust particles may act as carriers for genotoxic agents. Chromium compounds are often present in oak and beech dusts as they are frequently used in the wood-processing industry, particularly as potassium dichromate in stains as well as fixing agents in wood preservatives. Stained furniture is made largely from oak and beech as they contain enough tannic acid to allow for chemical staining (Klein *et al.*, 2001). Nasal tumours were produced in rats following the inhalation of chromate-stained oak wood dust Klein *et al.* (2001). It was hypothesized that chromate trapped in dust particles is slowly released as hexavalent chromium in the nasal mucosa. Leather workers and tanners are also exposed to hexavalent chromium (Stern *et al.*, 1987). Hexavalent chromium is genotoxic and

**Table 4.1 Other risk factors for cancers of the nasal cavity and paranasal sinuses<sup>a</sup>**

Exposure	Reference
Boot and shoe manufacture and repair	IARC (1987, 2012b)
Formaldehyde	IARC (1995, 2012d)
Hexavalent chromium	IARC (1990, 2012b)
Mineral oils	IARC (1987, 2012d)
Mustard gas	IARC (1987, 2012d)
Selected nickel compounds	IARC (1990, 2012b)
Tobacco smoking	IARC (2002, 2012c)

<sup>a</sup> All classified as Group 1 carcinogens by IARC

has been linked with the development of sinonasal cancers in humans (Table 4.1; IARC, 1990).

DNA damage (detected by comet assay) in peripheral blood leukocytes was studied in 35 furniture workers and in 41 control office workers. Approximately 20% of woodworkers had elevated levels of DNA damage that did not depend on smoking status compared to 13% of control smokers and 7% of control non-smokers (Palus *et al.*, 1999). [The Working Group noted that the significance of this study is difficult to assess because DNA damage in the sinonasal mucosa was not studied.]

Another group of 60 male furniture workers occupationally exposed for more than 5 years to a mixture of softwood and hardwood dusts (7.4–25.8 mg/m<sup>3</sup>) was studied for markers of genotoxicity using peripheral blood lymphocytes and buccal epithelial cells. Controls were 60 healthy male government workers with no history of wood dust exposure. Statistically significant elevations in DNA damage in peripheral blood lymphocytes were detected in workers using the Comet assay. Increased frequencies of micronuclei and chromosomal aberrations were also detected in the peripheral blood lymphocytes of workers. An increased frequency of micronuclei was also detected in buccal epithelial cells obtained from workers. Micronucleus frequency was increased in both workers and controls who were smokers and consumed alcohol. Serum

levels of superoxide dismutase activity and glutathione peroxidase activity, but not catalase activity, were reduced in the workers ([Rekhadevi et al., 2009](#)). [The Working Group noted that the authors of this study could not eliminate a potential effect of exposure to chemical adhesives and wood polish in these workers.]

[Çelik & Kanik \(2006\)](#) studied the frequency of micronuclei and other nuclear alterations in exfoliated buccal mucosal cells from 20 workers occupationally exposed to wood dust and 20 healthy controls. Dust levels in the workplace were 4.7–28.9 mg/m<sup>3</sup>. In the controls, the micronucleus frequency was 1.5 ± 1.2% compared to 6.6 ± 1.6% in the workers. Evidence of nuclear injury (karyolysis, karyorhexis) and binucleated cells was also increased in the workers. Smokers in both groups showed increased micronucleus frequency and evidence of nuclear injury. [The Working Group noted that the use of buccal epithelial cells as a surrogate for sinonasal mucosa had not been validated.]

The genotoxicity of six wood dusts and dust from MDF coated with oak was compared in the A549 human lung carcinoma cell line ([Bornholdt et al., 2007](#)). As determined by a comet assay, beech, birch, teak, pine, and MDF dusts increased DNA strand breaks 1.2–1.6-fold after 3 hours of exposure. [The Working Group noted that the use of a malignant lung carcinoma cell line as a surrogate for sinonasal epithelial cells is questionable, and that no particulate control group was included.]

No data based on genotoxic assays were available to the Working Group for workers exposed to leather dusts.

#### 4.3.4 Indirect genotoxicity

The most likely mechanism proposed for the carcinogenicity of wood dust is a combination of reduced clearance of large particles from the middle turbinate and ethmoid regions of the sinonasal cavity, leading to mechanical

irritation, inflammation, and increased cell proliferation ([Llorente et al., 2009](#)). In support of the association between chronic inflammation and sinonasal cancer, [Holmila et al. \(2008\)](#) analysed COX-2 and p53 protein expression in 23 cases of adenocarcinoma; 17 were exposed to wood dust and 19 were smokers. Elevated COX-2 expression was found in 13 cases including eight cases who were non-smokers; ten of these cases had a history of wood dust exposure. In 50% of the cases with elevated COX-2 expression, there was elevated p53 protein expression in the same histological pattern as COX-2. COX-2 protein expression was confirmed at the mRNA level.

In a murine model of lung inflammation induced by intranasal instillation of birch or oak dusts two times a week for 3 weeks, oak dust induced more inflammation with an influx of neutrophils and lymphocytes compared with birch dust that elicited an influx of eosinophils ([Määttä et al., 2006](#)).

These dusts were also tested for induction of pro-inflammatory mediators from murine RAW 264.7 macrophage cell lines. Birch dust increased the release of the pro-inflammatory cytokines IL-6 and TNF- $\alpha$ , and oak dust caused a smaller release of TNF- $\alpha$ . Birch dust also elicited a stronger chemokine response than oak dust ([Määttä et al., 2005](#)).

A panel of six wood dusts and MDF dust was assessed for expression of IL-6 and IL-8 pro-inflammatory cytokines using the human A549 lung carcinoma cell line. Based on expression of IL-8 mRNA, teak dust was more potent than MDF, birch, spruce, or pine dust; with beech and oak dust showing the weakest activity in this assay ([Bornholdt et al., 2007](#)).

Human alveolar macrophages obtained from healthy volunteers were exposed to endotoxin-free pine dust for 2 hours. This exposure induced a dose-dependent release of the pro-inflammatory mediators, TNF- $\alpha$  and MIP-2, that was associated with increased production of reactive oxygen species ([Long et al., 2004](#)).

No experimental data were available to the Working Group on the release of inflammatory mediators from animals or cell cultures following exposure to leather dusts.

Overall, these experimental studies provide evidence that wood dust from a variety of hardwoods and softwoods can elicit the release of pro-inflammatory mediators after short-term exposures, and suggest a possible association between inflammation and the development of cancer.

In summary, the mechanism responsible for the carcinogenicity of wood or leather dusts is unknown as concluded previously by [IARC \(1995\)](#). In 2000, the Health Council of the Netherlands concluded that wood dust cannot be classified as a non-genotoxic carcinogen or as a direct or indirect genotoxic carcinogen due to insufficient mechanistic data ([Feron et al., 2001](#)).

#### 4.4 Other risk factors for sinonasal and nasopharyngeal cancers

The most important exposures associated with the development of sinonasal cancers are occupational exposures in furniture and wood-working industries, leather and shoe manufacturing, and in nickel workers ([Table 4.1](#); [IARC, 2012b](#)).

Exposures to other agents classified by IARC as *carcinogenic to humans (Group 1)* have also been associated with cancers of the nasal cavity and paranasal sinuses ([Table 4.1](#)).

Other occupations that have been suggested to be linked with the development of sinonasal cancers include agricultural workers, workers in food manufacturing and preserving, and workers in the textile industry, and in the manufacturing of rubber and plastic products ([Leclerc et al., 1997](#); [Luce et al., 2002](#)).

Nasopharyngeal cancers occurring in low-risk populations, including Europe and the US, peak in adolescents and young adults and are

**Table 4.2 Other risk factors for nasopharyngeal cancer<sup>a</sup>**

Exposure	Reference
Chlorophenol	<a href="#">Zhu et al., (2002)</a> , <a href="#">IARC (1999)</a>
Epstein-Barr virus (EBV)	<a href="#">IARC (1997, 2012a)</a>
Ingestion of salted fish and preserved foods during childhood	<a href="#">IARC (2002, 2012c)</a>
Formaldehyde	<a href="#">IARC (1995, 2012d)</a>
Mustard gas	<a href="#">IARC (1987, 2012d)</a>
Tobacco smoking	<a href="#">IARC (2002, 2012c)</a>

<sup>a</sup> All classified as Group 1 carcinogens except for chlorophenol (2B)

associated with Epstein-Barr virus (EBV) infection. The highest risk populations are in the Cantonese region of Southern China and Hong Kong Special Administrative Region, followed by Taiwan, China, the Arctic region, South-eastern Asia, and North Africa. In these high-risk populations, peak incidence is at 50–59 years and the most important risk factors are dietary in association with EBV infection ([Yu & Yuan, 2002](#); [IARC, 2012a](#)).

Tobacco smoking is a risk factor for both sinonasal cancer ([t Mannetje et al., 1999](#); [IARC, 2002](#)) and nasopharyngeal cancer, in addition to occupational exposure to formaldehyde or mustard gas ([Table 4.2](#)). Squamous cell carcinomas in the nasopharynx have also been linked with exposure to a wood preservative, chlorophenol ([Table 4.2](#); [IARC, 1999](#); [Zhu et al., 2002](#)).

It is worth noting that EBV infects almost everyone worldwide but the infection is usually kept dormant by the immune system. Exposure to agents that deregulate the immune system may potentially activate this oncogenic virus ([IARC, 2012a](#)).

No published reports were available to the Working Group on genetic susceptibility to development of sinonasal cancers or nasopharyngeal carcinoma associated with exposure to wood or leather dusts.

## 4.5 Synthesis

Potential mechanisms responsible for the carcinogenicity of wood dust include tissue injury induced by the deposition of wood dust particles in the sinonasal region, impaired ciliary clearance, direct genotoxicity and indirect genotoxicity secondary to chronic inflammation. Wood or leather dusts may also act as carrier for other genotoxic agents (e.g. chromate). There is weak evidence for these mechanisms in cellular assays, short-term animal assays, or assays for genotoxicity using peripheral blood cells or buccal epithelial cells obtained from workers exposed to wood dust.

Workers exposed to wood or leather dusts have increased frequencies of metaplasia and hyperplasia in nasal epithelial biopsies, although these alterations are not considered to be precursor lesions of neoplasia at this organ site. In one study, leather workers also showed increased evidence of dysplasia in nasal biopsies. No mechanistic data were available to the Working Group for leather dust exposure.

## 5. Evaluation

There is *sufficient evidence* in humans for the carcinogenicity of wood dust. Wood dust causes cancer of the nasal cavity and paranasal sinuses and of the nasopharynx.

There is *inadequate evidence* in experimental animals for the carcinogenicity of wood dust.

Wood dust is *carcinogenic to humans* (Group 1).

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