

WOOD DUST

1. Exposure Data

1.1 Composition of wood

Wood is one of the world's most important renewable resources and grows in forests all over the world. Forests cover about one-third of the globe's total land area, about 3.4 million km². This area represents more than 1.0 trillion m³ of total tree biomass; of this biomass, about 3.5 thousand million m³ per year are harvested, about half of which is used as fuel, predominantly in developing countries. 'Industrial roundwood' (1.7 thousand million m³/year) is the term applied to all sawn wood (54%), pulpwood (21%), poles, pit props (14%) and wood used for other purposes such as particle-board and fibre-board (11%) (FAO, 1992; Schulz, 1993).

The species of trees that grow and are harvested in different countries vary considerably. Hardwoods dominate, for instance, in Italy, where oak, chestnut and beech are important species (Haden-Guest *et al.*, 1956). In other, primarily colder regions, conifers dominate: for example, pine and spruce in the Nordic countries and pine, spruce, hemlock, cedar and fir in Canada. Table 1 shows trees harvested for industrial use by broad category (conifer versus non-conifer) in some of the countries in which epidemiological studies have been conducted on wood dust. Even within countries, however, there is considerable variation: in western United States of America, conifers, such as Douglas fir and Ponderosa pine, were of primary economic importance in the 1950s, while various non-conifers were important in the mid-west and north-east, and southern yellow pine was the single most important species in the south-east (Haden-Guest *et al.*, 1956).

Many countries with little domestic production of lumber, such as the Netherlands, import wood, and even countries with much domestic production import wood for specific uses: for example, Finland, which produces pine, spruce and birch, imports some tropical woods, such as mahogany and teak, for furniture production (Welling & Kallas, 1991). The data in Table 1 probably represent the species used in the logging, sawmill and pulp and paper industries, which usually consume wood from nearby regions. The species of trees used in different branches of the wood industry are described in sections 1.4.2–1.4.6. About two-thirds of all wood used in the world for industrial purposes is from softwood species (FAO, 1993).

1.1.1 Classification and nomenclature

The Earth has an estimated 12 000 species of tree, each producing a characteristic type of wood. Spermatophytes are subdivided into two classes on the basis of seed type: gymnosperms,

which have exposed seeds, and angiosperms, with encapsulated seeds. These classes are further separated into orders, families, genera and species. As an illustration of the main divisions, the full classification of Scots pine (*Pinus sylvestris* L.) is given in Figure 1.

Table 1. Industrial roundwood^a production in 1980 (thousands of cubic metres) by country

Country	Conifers	Non-conifers
Europe		
Denmark	1 185 (64%)	665 (36%)
Finland	38 010 (88%)	5 010 (12%)
France	14 069 (49%)	14 897 (51%)
Germany (western)	21 670 (74%)	7 657 (26%)
Italy	1 404 (28%)	3 705 (72%)
Netherlands	577 (72%)	228 (28%)
Norway	8 158 (96%)	316 (4%)
Sweden	40 000 (89%)	4 795 (11%)
United Kingdom	2 609 (68%)	1 232 (32%)
Former USSR	246 400 (89%)	31 300 (11%)
Asia		
China ^b	50 016 (63%)	29 186 (37%)
Japan	21 427 (63%)	12 624 (37%)
North America		
Canada	144 100 (93%)	10 124 (7%)
United States	246 525 (75%)	80 570 (25%)
Australia	4 009 (26%)	11 642 (74%)
New Zealand	9 698 (98%)	247 (2%)

From FAO (1993)

^aAll wood used for sawn wood and veneer logs, pulpwood, chips, particles, poles and pit props

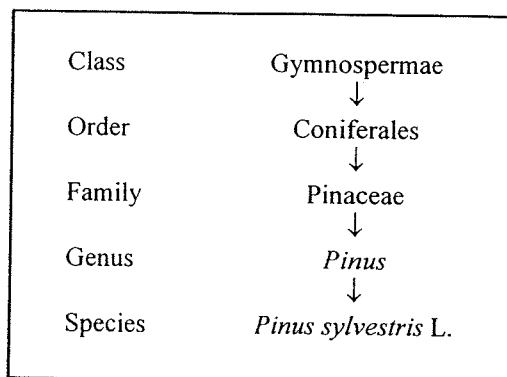
^b Estimates

Most species are deciduous trees or hardwoods, and only about 800 species are coniferous trees or softwoods (Bauch, 1975). Wood-producing tree-like plants, such as bamboo (*Graminaceae*) and palm (*Palmae*), differ from trees in that they lack secondary thickness growth.

The terms 'hardwood' and 'softwood' refer to the species of tree and not necessarily to the hardness of the wood. While hardwoods are generally more dense than softwoods, the density varies considerably within each family and the hardness of the two groups overlaps somewhat (Fengel & Wegener, 1989; see also Table 6). Gymnosperms comprise all trees that yield softwood lumber. Only one order, Coniferales, is important from the point of view of industrial use. The angiosperms are separated into two classes on the basis of initial seed leaf: monocots

(e.g. bamboo and palms) have one initial seed leaf, and dicots (e.g. oak and birch) have two. Dicots comprise all tree-sized plants that yield hardwood lumber and occur mostly in temperate zones.

Figure 1. Classification of Scots pine



From Jane (1970)

The scientific and common names of some softwoods and hardwoods are given in Table 2.

Table 2. Nomenclature of some softwoods and hardwoods

Genus and species	Common name
Softwood	
<i>Abies</i>	Fir
<i>Chamaecyparis</i>	Cedar
<i>Cupressus</i>	Cypress
<i>Larix</i>	Larch
<i>Picea</i>	Spruce
<i>Pinus</i>	Pine
<i>Pseudotsuga menziesii</i>	Douglas fir
<i>Sequoia sempervirens</i>	Redwood
<i>Thuja</i>	Thuja, arbor vitae
<i>Tsuga</i>	Hemlock
Hardwood	
<i>Acer</i>	Maple
<i>Alnus</i>	Alder
<i>Betula</i>	Birch
<i>Carya</i>	Hickory
<i>Carpinus</i>	Hornbeam, white beech
<i>Castanea</i>	Chestnut
<i>Fagus</i>	Beech

Table 2 (contd)

Genus and species	Common name
Hardwood (contd)	
<i>Fraxinus</i>	Ash
<i>Juglans</i>	Walnut
<i>Platanus</i>	Sycamore
<i>Populus</i>	Aspen, poplar
<i>Prunus</i>	Cherry
<i>Salix</i>	Willow
<i>Quercus</i>	Oak
<i>Tilia</i>	Lime, basswood
<i>Ulmus</i>	Elm
Tropical hardwood	
<i>Agathis australis</i>	Kauri pine
<i>Chlorophora excelsa</i>	Iroko
<i>Dacrydium cupressinum</i>	Rimu, red pine
<i>Dalbergia</i>	Palisander
<i>Dalbergia nigra</i>	Brazilian rosewood
<i>Diospyros</i>	Ebony
<i>Khaya</i>	African mahogany
<i>Mansonia</i>	Mansonia, bete
<i>Ochroma</i>	Balsa
<i>Palaquium hexandrum</i>	Nyatoh
<i>Pericopsis elata</i>	Afromosia
<i>Shorea</i>	Meranti
<i>Tectona grandis</i>	Teak
<i>Terminalia superba</i>	Limba, afara
<i>Triplochiton scleroxylon</i>	Obeche

From Vaucher (1986)

1.1.2 Anatomical features

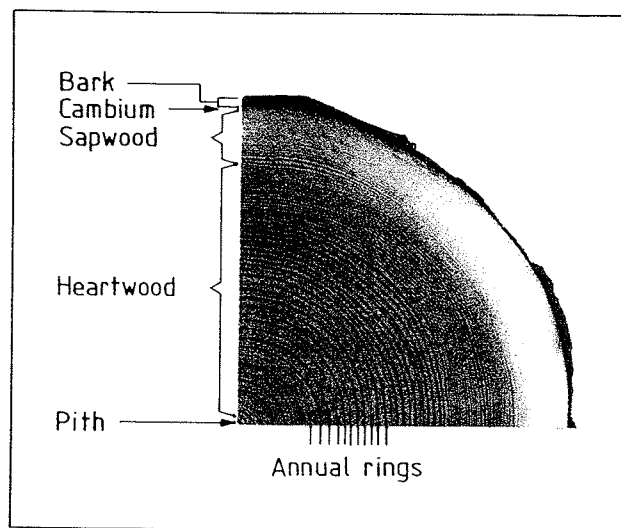
Detailed information on wood anatomy is given by Jane (1970), Panshin and de Zeeuw (1970), Wagenführ and Scheiber (1974), Grosser (1977), Barefoot and Hankins (1982) and Hoadley (1990).

A look at the cross-section of a stem or a stem segment (Fig. 2) reveals a differentiation between bark and wood and, in many species, inner and outer areas with different coloration. Whereas some cells in the outer part (sapwood) are still alive (parenchyma), all cells in the inner part (heartwood) are dead. There is strong biosynthetic activity at the sapwood-heartwood boundary, where stored materials such as starch and other carbohydrates are transformed into low- and medium-relative-molecular-mass substances (extractives) and deposited in the heartwood (Streit & Fengel, 1994). It is assumed that these substances contribute to the conservation and protection of wood. The cells of wood tissue are produced in the cambium, a

cell monolayer between the phloem (inner bark) and xylem (wood) (Fig. 2), where growth in length and thickness of a tree occurs.

The morphology of softwood tissue is simpler than that of hardwood tissue. Softwood consists, in its bulk, of only one cell type, tracheids, which are elongated, fibre-like cells with a square or polygonal cross-view. Less than 10% of the wood consists of short, brick-like parenchymal cells arranged radially. Moreover, some softwoods contain epithelial cells that secrete resin into canals which run horizontally and radially through the wood.

Figure 2. Segment of a stem showing the various macroscopically visible areas of wood

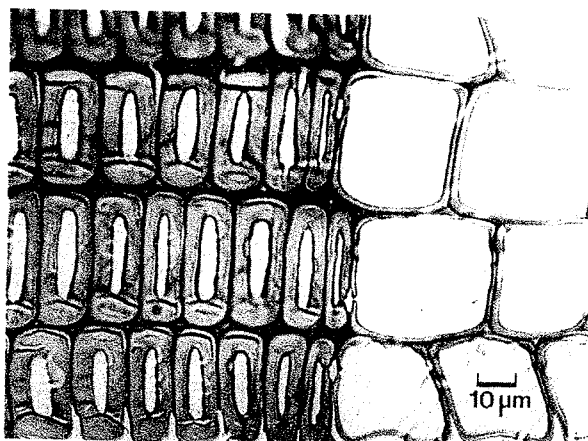


Adapted from Hoadley (1990)

Tissue formed in springtime (in temperate zones) is called ‘earlywood’ and consists of tracheids with wide lumina and thin walls (Fig. 3), which transport water from the roots to the top of the tree. Rows of valve-like openings (pits) at the ends of the tracheids allow exchange of water between adjacent cells. Tracheids produced in summertime (‘latewood’) have thick walls and small lumina; they serve predominantly to stabilize the tree.

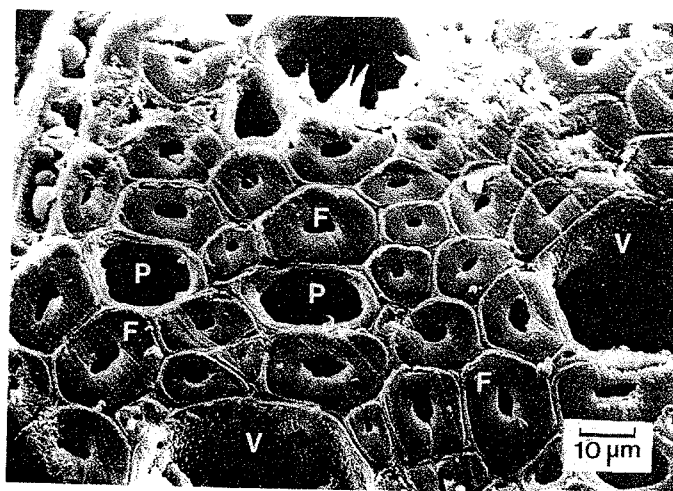
In hardwoods, there is more detailed differentiation between stabilizing, conducting and storage tissue. Stabilizing tissues contain libriform fibres and fibre tracheids, which are elongated cells with thick polygonal walls and small lumina (Fig. 4). The conducting system is composed of vessel elements fitted together to form long tubes of up to several metres. The vessels have thin walls and large diameters. At the junction of two elements, there are stiffening rings or plates with perforations characteristic of different wood species. Storage tissues consist of longitudinally and radially arranged parenchymal cells. Hardwoods that contain resin canals also have a secretory system of epithelial cells.

Figure 3. Border between latewood (left) and earlywood (right) in a softwood (spruce); light micrograph



From Fengel & Wegener (1989)

Figure 4. Hardwood tissue (beech) with vessels (V) and parenchymal cells (P) surrounded by libriform fibres (F); scanning electron micrograph



Adapted from Fengel & Wegener (1989)

1.1.3 Cell wall structure and distribution of components of wood

The walls of wood cells consist of various layers, which differ in structure and chemical composition (Fig. 5; Fengel & Wegener, 1989). The individual cells of wood tissue are glued together in the middle lamella, which consists mainly of lignin, polyoses and pectins. Often there is no exact, visible border between the pure middle lamella and the outer cell wall layer, which is called the primary wall and is formed by a net-like arrangement of cellulose fibrils (Fig. 5a) embedded in a matrix of lignin and polyoses. The middle lamella and primary wall are often called the 'compound middle lamella' (Fig. 5b).

The next layer is the secondary wall, which is subdivided into secondary walls 1 (S1) and 2 (S2). S1 and S2 contain densely packed cellulose fibrils arranged in parallel, which differ in the angle at which the fibrils run: in the S1, the fibrils run at a wide angle in relation to the fibre axis and in the S2, at a small angle. The S2 is the thickest wall layer and accounts for 50% (vessels, parenchymal cells) to 90% (tracheids, libriform fibres) of the whole cell wall. Parenchymal cells are equipped with a third secondary wall (S3) which has a fibril arrangement that is more open than that in the S2.

At the inner border of the cell wall, there is a final thin layer called the tertiary wall (T), in which the cellulose fibrils run at an angle similar to that of the S1. In some species, the tertiary walls of tracheids, fibres and vessels are covered with a wart-bearing amorphous layer (Fig. 5a,b).

The lignin content decreases from the compound middle lamella through the S2, while the cellulose content increases in the same direction (Table 3). The polyose content is highest in the S1, but because of the thickness of the S2, most lignin and polyoses are deposited in this layer.

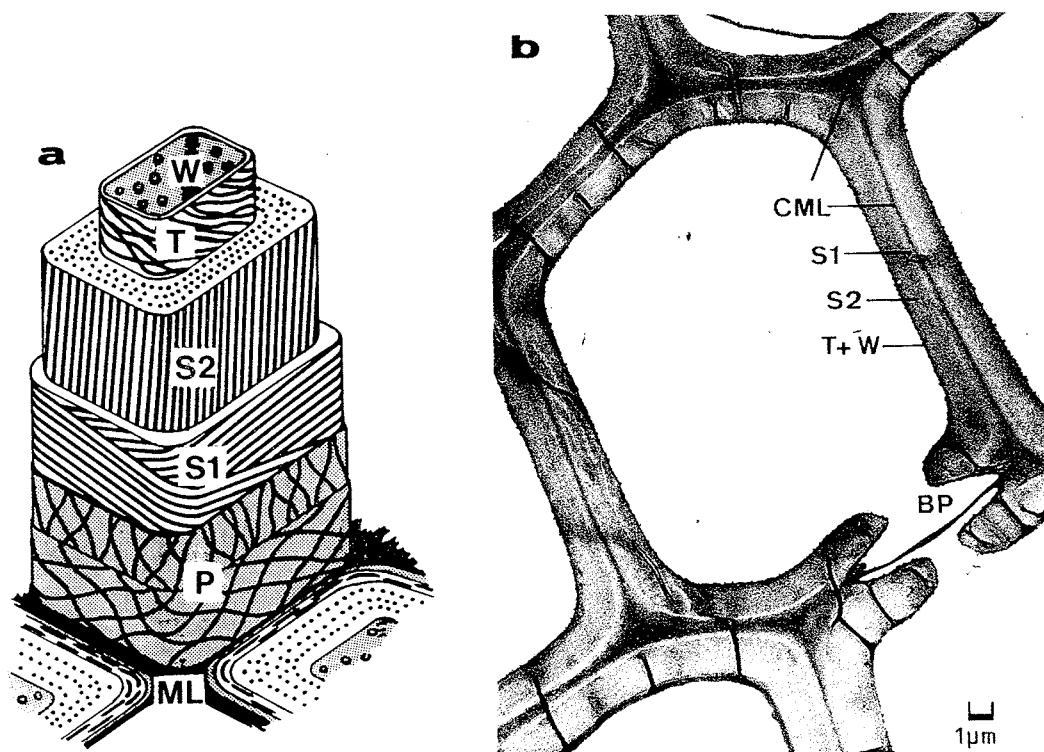
The 'extractives', the organic matter of wood, are found in the resin canals and parenchymal cells. In heartwood, extractives are also deposited in the compound middle lamella and the secondary walls. Trees with a high content also have extractives in the lumina (Fengel, 1989).

In areas where mechanical deformation of stems and branches has occurred, special tissues are found. In such 'compression areas' in softwood tracheids, the S2 is provided with helical cavities and contains a high percentage of lignin (compression wood). In hardwoods, fibres with a thick additional gelatinous layer consisting of relatively pure cellulose are formed in tension areas (tension wood). Compression and tension woods are together referred to as 'reaction wood' and may influence the processing of wood.

1.1.4 Chemical components

In this section, the main constituents of woods are identified and differences between hardwood and softwood are indicated. The formulae of a number of chemical constituents are shown in Figure 6. Chemicals found only in fruit or flowers or in fungi growing on trees are not described. Extensive reviews, with detailed accounts of numerous extractives, have been published (Beecher *et al.*, 1989; Fengel & Wegener, 1989; Swan, 1989). Hillis (1987) specifically reviewed heartwood.

Figure 5. Cell wall structure



From Fengel & Wegener (1989)

(a) Model of a vascular cell (softwood tracheids, hardwood libriform fibres); (b) cross-section of a softwood tracheid (umbrella fir) with a bordered pit (BP); transmission electron micrograph. ML, middle lamella; P, primary wall; S1, secondary wall 1; S2, secondary wall 2; T, tertiary wall; W, warty layer; CML, compound middle lamella (ML + P)

Table 3. Approximate content of lignin, cellulose and polyoses in the cell wall layers of softwood tracheids

Wall layer	Lignin (%)	Cellulose (%)	Polyoses (%)	Main polyoses
Compound middle lamella	60	15	25	Pectins, galactan, xylan
Secondary wall 1	30	35	35	Xylan, mannan
Secondary wall 2 + tertiary wall	25	60	15	Mannan

From Fengel & Wegener (1989)

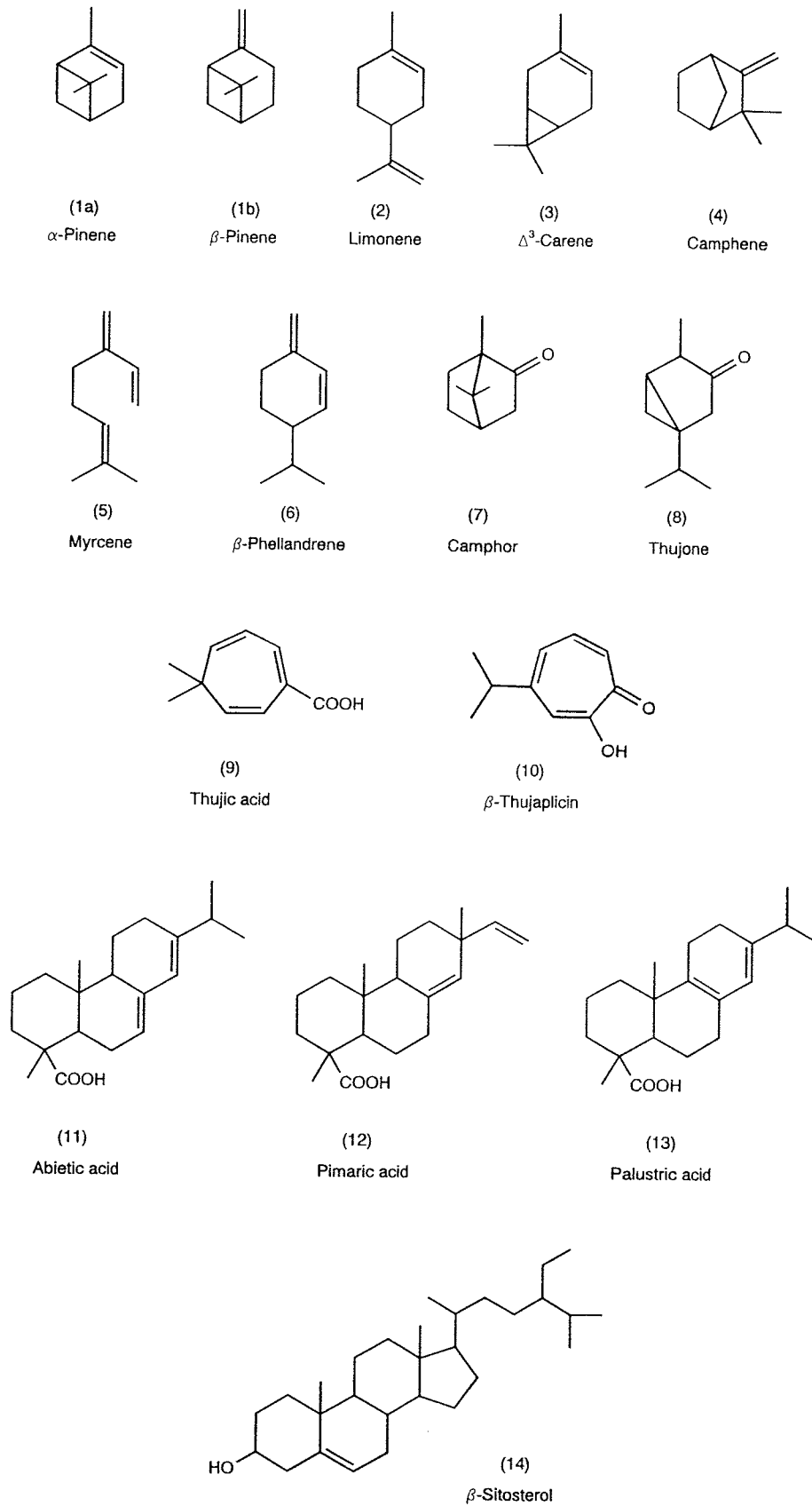
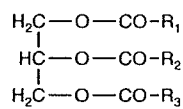
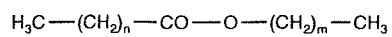
Figure 6. Chemical formulae of certain chemical components of wood

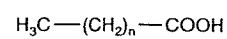
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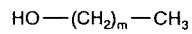
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Fats and Oils



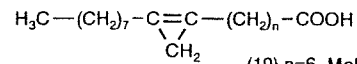
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Waxes



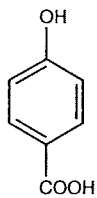
(17)
Fatty acids



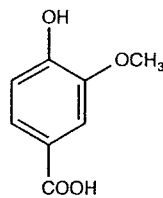
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Fatty alcohols



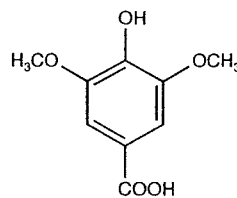
(19) n=6, Malvalic acid
(20) n=7, Sterculic acid



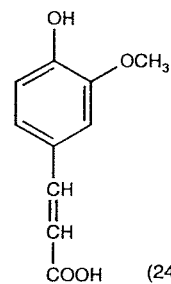
(21)
para-Hydroxy-
benzoic acid



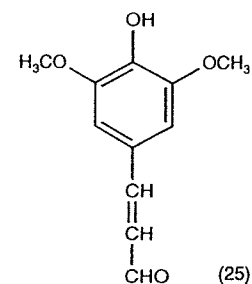
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Vanillic acid



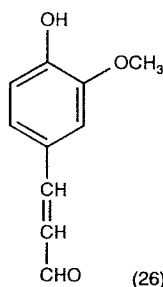
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Syringic acid



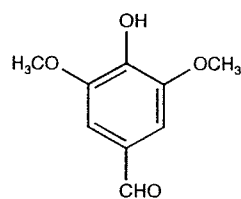
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Ferulic acid



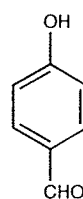
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Sinapaldehyde



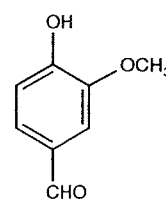
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Coniferyl aldehyde



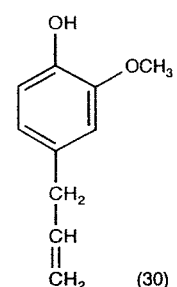
(27)
Syringaldehyde



(28)
para-Hydroxy-
benzaldehyde

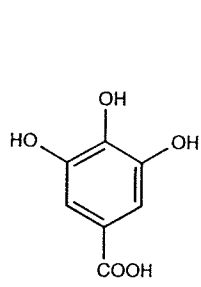


(29)
Vanillin

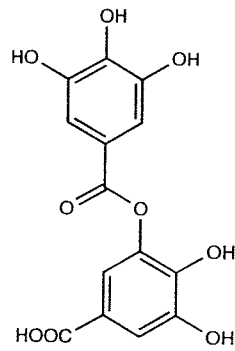


(30)
Eugenol

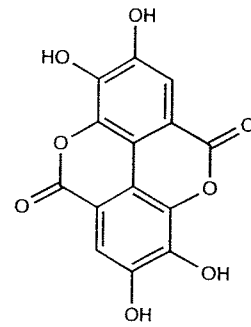
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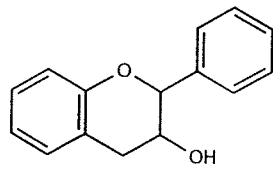
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Gallic acid



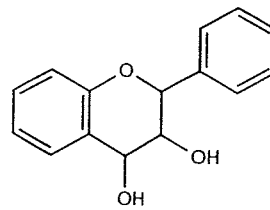
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Digallic acid



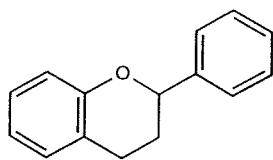
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Ellagic acid



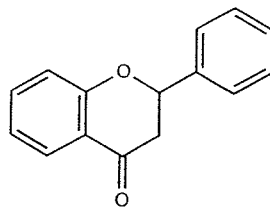
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Flavan-3-ol



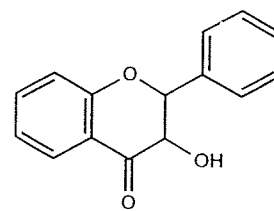
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Flavan-3,4-diol



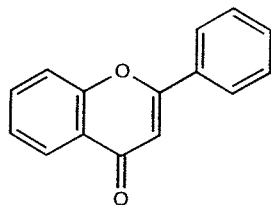
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Flavanes



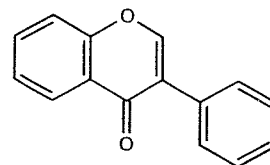
(37)
Flavanones



(38)
Flavanonols



(39)
Flavones



(40)
Isoflavones

Figure 6 (contd)

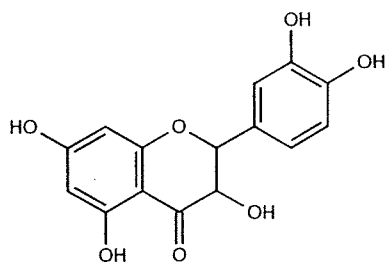
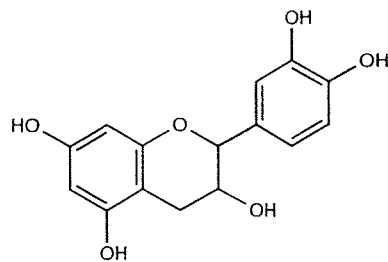
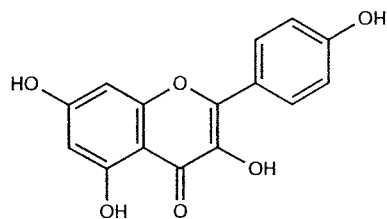
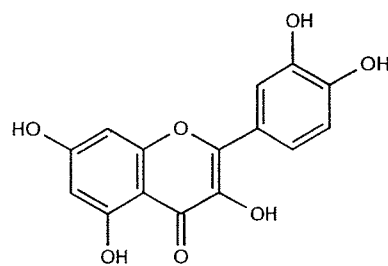
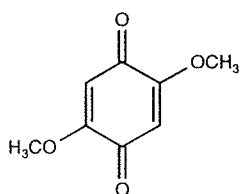
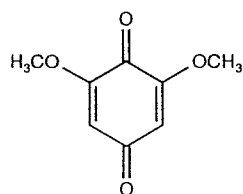
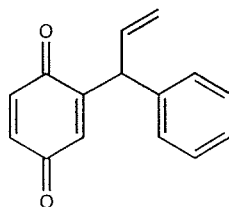
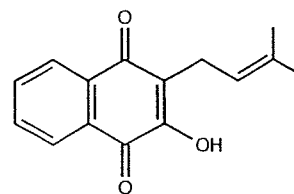
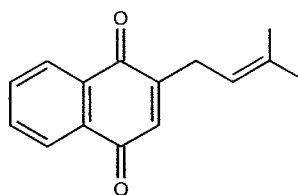
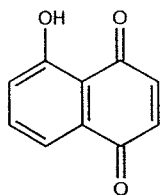
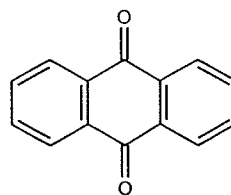
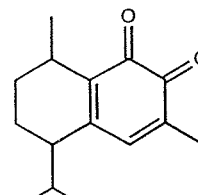
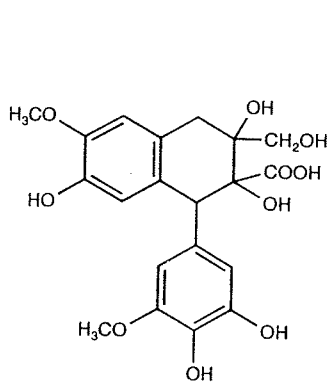
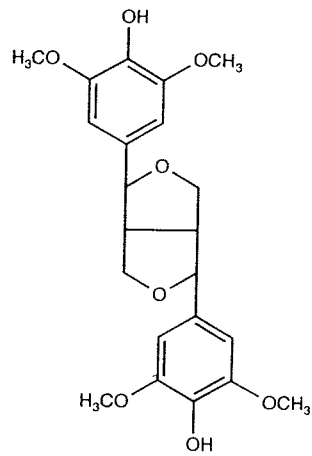
(41)
Taxifolin(42)
Catechin(43)
Kaempferol(44)
Quercetin(45a)
2,5-Dimethoxy-
benzoquinone(45b)
2,6-Dimethoxy-
benzoquinone(46)
Dalbergione(47)
Lapachol(48)
Desoxylapachol(49)
Juglone(50)
Anthraquinones(51)
Mansanone A

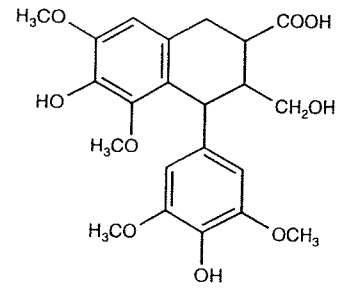
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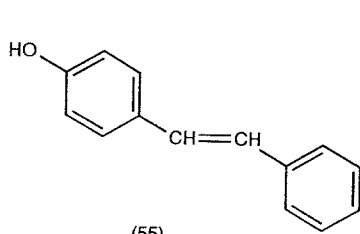
(52)
Plicatic acid



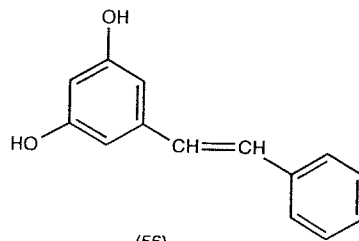
(53)
Syringaresinol



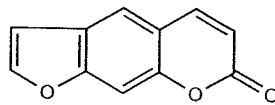
(54)
Thomasic acid



(55)
4-Hydroxystilbene



(56)
Pinosylvin



(57)
Psoralen

(a) Macromolecular components

The essential chemical constituents of wood are cellulose, polyoses (hemicelluloses) and lignin, which has a macromolecular structure. Although cellulose is the uniform structural element of all woods, the proportions and chemical composition of lignin and polyoses differ in softwood and hardwood. Wood generally also contains small amounts of polymeric compounds, such as starch, pectic substances and proteins.

Cellulose is the major component (40–50%) of both softwood and hardwood. It can be briefly characterized as a linear high-relative-molecular-mass polysaccharide built up exclusively of D-glucose units joined by $\beta(1\rightarrow4)$ glycosidic linkages.

Polyoses (hemicelluloses) are present in larger amounts in hardwood than in softwood and differ in their sugar composition. They are composed mainly of five neutral sugar units—the hexoses, glucose, mannose and galactose, and the pentoses, xylose and arabinose. Some polyoses also contain uronic acid units. The molecular chains of polyoses are much shorter than those of cellulose and are branched and/or contain side groups. Softwood has a higher proportion of mannose units and more galactose units than hardwood; hardwood has a higher proportion of xylose units (Table 4).

Table 4. Non-glucosic sugars in polyoses of some woods (%)

Species	Mannose	Xylose	Galactose	Arabinose	Uronic acid	Rhamnose
European spruce	13.6	5.6	2.8	1.2	1.8 ^a	0.3
Scots pine	12.4	7.6	1.9	1.5	5.0	NR
European beech	0.9	19.0	1.4	0.7	4.8 ^a	0.5

From Fengel & Wegener (1989); NR, not reported

^a4-O-Methylglucuronic acid

Lignin, the third macromolecular component of wood, is quite different from the polysaccharides. The monomers of lignin are phenylpropane units joined by various linkages, resulting in complicated three-dimensional macromolecules. The lignin content of softwood is higher than that of hardwood, and softwood and hardwood lignins differ structurally and with regard to their contents of guaiacyl, syringyl and *para*-hydroxyphenyl units. Most softwood lignins are typical guaiacyl lignins containing minor amounts of syringyl and *para*-hydroxyphenyl units. The composition of lignin varies much more in hardwood than in softwood, and hardwood lignins have higher proportions of syringyl units. The syringyl content of typical hardwood lignins varies between 20 and 60%.

(b) Low-relative-molecular-mass components

A heterogeneous mixture of organic and inorganic compounds occurs in different species of wood in various amounts. The organic matter that can be extracted from wood with nonpolar or

polar solvents is commonly called 'extractives'; the inorganic part is reduced mainly to ash in the analysis of wood. The 'extractives' represent 0.1–1% of the wood mass in trees of temperate zones and 15% or more in tropical wood. As some of these compounds protect against injury or attack from fungi, insects and bacteria, they may have toxic, irritant or sensitizing properties.

Organic extractives can have aliphatic, alicyclic or aromatic structures. Non-polar extractives comprise mainly terpenes, fatty acids, resin acids, waxes, alcohols, sterols, steryl esters and glycerides. The polar extractives of wood generally consist of aromatic (phenolic) compounds, i.e. tannins, flavonoids, quinones and lignans. The common water-soluble extractives of wood are carbohydrates and their derivatives, alkaloids, proteins and inorganic material. Hardwood tends to have a higher percentage of polar extractives than softwood (see section 1.3.2 (a) and Table 7).

(i) *Terpenes and terpenoids*

Terpenes are a ubiquitous group of natural compounds, of which over 4000 have been isolated and identified. They can be derived from isoprene (2-methyl-1,3-butadiene) units, which are usually connected to form one or more rings. Two or more isoprene units build up the mono-, sesqui-, di-, tri-, tetra- and polyterpenes.

The extractives of softwood include all classes of terpenes, whereas hardwoods contain mainly higher terpenes. Monoterpenes are found only in some hardwood tropical species, while the volatile oil (turpentine) of softwoods consists mainly of these compounds. The commonest are α - and β -pinene (Fig. 6, **1a**, **1b**) and limonene (**2**) (see IARC, 1993), which are present in all softwoods; but Δ^3 -carene (**3**), camphene (**4**), myrcene (**5**) and β -phellandrene (**6**) are also widespread. α -Pinene has been suggested to be a sensitizing agent, and terpenes with a keto group (e.g. camphor (**7**) and thujone (**8**)) appear to be more toxic than related compounds.

The tropolones, structures with seven-membered rings, are considered to be derivatives of monoterpenes. Compounds such as thujic acid (**9**) and β -thujaplicin (**10**) are found only in species of the *Cypressaceae* family. Sesquiterpenes are found in many tropical woods but are quite rare in hardwood in temperate zones and in softwood.

The diterpenes seem to be restricted to softwood species and occur mainly in the form of resin acids. They are mostly tricyclic compounds, such as abietic (**11**), pimaric (**12**) and palustric acids (**13**). The neutral diterpenes consist of hydrocarbons, oxides, alcohols and aldehydes.

A great variety of triterpenes are present in many hardwoods in tropical and temperate zones and also in softwoods. Most have a steran substructure and must therefore be assigned to the steroids. The main component of the steroid group in both softwood and hardwood is β -sitosterol (**14**). Some saponins, which are glycosides of triterpenes and steroids, cause dermatitis and other diseases.

(ii) *Fats, waxes and their components*

Although saturated and unsaturated higher fatty acids are found in wood mostly as esters with glycerol (fats and oils (**15**)) or with higher alcohols (waxes (**16**)), they also occur in free form (**17**). Free and combined fatty acids typically include linoleic, palmitic (**17**: $n=14$) and stearic acid (**17**: $n=16$) and some cyclopropenic acids, such as malvalic (**19**) and sterculic (**20**)

acid. The corresponding fatty alcohols (18) are also found. Triglycerides dominate the glycerides (fats), as compared with mono- and diglycerides, especially in hardwood species.

(iii) *Phenolic compounds*

Extracts of woods contain low-relative-molecular-mass phenols. Some are probably degradation products of compounds such as lignin, which may be hydrolysed during several extractions or during steam distillation. These simple phenols are represented in hardwood predominantly by acids (including *para*-hydroxybenzoic (21), vanillic (22), syringic (23) and ferulic acids (24)) and in both softwood and hardwood by aldehydes (including sinapaldehyde (25), coniferyl aldehyde (26), syringaldehyde (27), *para*-hydroxybenzaldehyde (28), vanillin (29) and eugenol (30) [see IARC, 1985a]).

The extractives of wood also contain various compounds with phenolic substructures, including tannins, flavonoids, quinones, lignans and stilbenes.

Tannins: Tannins can be separated into hydrolysable and condensed types (phlobaphenes). The hydrolysable tannins are esters of gallic acid (31) and its dimer (digallic acid (32)) and of ellagic acid (33) with monosaccharides, mainly glucose. They may be subdivided into gallotannins, which yield gallic acid, and ellagitannins, which yield ellagic acid upon hydrolysis. The main components of condensed tannins are catechin derivatives (flavan-3-ol (34)) and leucoanthocyanidin derivatives (flavan-3,4-diol (35)). Tannins can precipitate proteins and influence cell metabolism.

In general, tannins are found mainly in hardwoods, although they may also occur in the heartwood of certain softwoods (conifers) that contain condensed tannins. Hydrolysable tannins (gallic acid type) occur less frequently than condensed tannins in hardwoods and are found predominantly in oak, chestnut and eucalyptus. All other woods, particularly tropical woods, contain mainly condensed tannins (catechin type). An overview of tannin chemistry is presented by Hemingway and Karchesy (1989).

Flavonoids: Condensed tannins belong to the large group of extractives known as flavonoids, which comprise the subgroups flavanes (36), flavanones (37), flavanonols (38), flavones (39) and isoflavones (40). They have various numbers of hydroxyl and methoxyl groups on the aromatic rings, either as glycosides or aglycones. The large number of flavonoids is due not only to the degree of saturation of the heterocyclic ring but also to the variation in degree of hydroxylation of the rings. The colour of some heartwoods is a result of the presence of flavonoids and related compounds. These compounds also occur more frequently in hardwood than softwood, although several flavonoids, such as taxifolin (41) and catechin (42), have also been identified in softwood. Other flavonoids found in hardwood are kaempferol (43) (see IARC, 1983a) and quercetin (44) (see IARC, 1983b).

Quinones: A range of aromatic quinones is present in extracts of various woods, including substituted benzoquinones (2,5- (45a) and 2,6-dimethoxybenzoquinone (45b), dalbergione (46)), various naphthoquinones (lapachol (47), desoxylapachol (48), juglone (49)) and anthraquinones (50) and their derivatives, such as quinoid sesquiterpenes (mansonone A (51)). They are responsible for the strong colours, high durability and dermatitic properties of some woods. Dimethoxybenzoquinone has been found in extracts of about 50 wood species.

Lignans: Lignans consist of two β - β -linked phenylpropane units. Some are dimeric structures that are also present in the lignin molecule. Lignans are typical heartwood components and occur in small or negligible amounts in sapwood. Although they are often found in softwood (e.g. plicatic acid (52) in western red cedar), some occur in hardwood, including syringaresinol (53) and thomasic acid (54), particularly in alder, oak and elm species.

Stilbenes: Stilbenes occur especially in the heartwood of both softwood and hardwood. These compounds, e.g. 4-hydroxystilbene (55) and pinosylvin (56), are responsible for the photosensitive reactions that cause darkening of woods.

(iv) *Miscellaneous organic compounds*

Free amino acids and their linked structures (proteins) are also found, but in small amounts. A high protein content encourages the development of wood-destroying organisms. Some hardwood species, particularly many tropical woods, also contain alkaloids of various chemical structures, including very toxic compounds such as berberine and strychnine. Phototoxic compounds belonging to the furocoumarin group, such as psoralen (57) and its derivatives, occur in some tropical wood species. Table 5 includes some biologically active compounds found in wood.

(v) *Inorganic compounds*

The main inorganic components of wood are potassium, calcium and magnesium; silicon is found in some tropical woods (Fengel & Wegener, 1989). These components comprise 0.2–0.5% of wood in temperate zones but often much more in tropical woods. Potentially carcinogenic inorganic elements such as chromium have been found in very small amounts in some wood species (Saka & Goring, 1983).

Table 6 summarizes several characteristics of softwoods and hardwoods; these are generalizations to which there are specific exceptions. Some characteristics are the same for softwoods and hardwoods; for others, the ranges among species overlap; and for a few characteristics, there are marked distinctions.

1.2 Wood-related industries and occupations

Workers in a wide variety of industries may be exposed to wood dust. In this section, the main industries and occupations in which such exposure may occur and the steps in the processes used are described (see also Koch, 1964; Maloney, 1977; FAO, 1981; IARC, 1981; Darcy, 1984; Industrial Accident Prevention Association, 1985; McCammon *et al.*, 1985; Holliday *et al.*, 1986; Suchsland & Woodson, 1986; Clayton Environmental Consultants, 1988; Williston, 1988). Detailed descriptions are generally provided only for processes that result in wood dust. The history of woodworking processes in the sawmill, furniture and construction industries was reviewed previously (IARC, 1981) and is not repeated here; however, major changes in processes that occurred during this century and affect exposure to wood dust are discussed.

Table 5. Examples of biologically active organic compounds found in wood

Substance class	Compound	Wood type
Terpenes	α -Pinene	Softwood
	Δ^3 -Carene	Softwood
	Camphor	Softwood
	Thujone	Softwood
	β -Thujaplicin	Softwood
	Sesquiterpene lactones	Softwood/hardwood
	Abietic/Neobietic acid	Softwood/hardwood
	Saponins	Hardwood
Phenols	Coniferyl aldehyde	Softwood/hardwood
	Sinapaldehyde	Hardwood
	Eugenol	Softwood/hardwood
	3-(Pentadecyl)catechol	Hardwood
	5-(Pentadec-10-enyl)resorcinol	Hardwood
Tannins	Catechin derivatives	Hardwood
	Leucoanthocyanidin derivatives	Hardwood
Flavonoids	Kaempferol	Hardwood
	Quercetin	Hardwood
Quinones	2,5- and 2,6-Dimethoxybenzoquinone	Softwood/hardwood
	3,4-Dimethoxydalbergione	Hardwood (tropical)
	Lapachol	Hardwood
	Desoxylapachol	Hardwood
	Juglone	Hardwood
	Mansonone A	Hardwood (tropical)
Lignans	Plicatic acid	Softwood
Stilbenes	2,3',4',5'-Tetrahydroxystilbene	Softwood
	Chlorophorin	Softwood
	Pinosylvin	Softwood
Miscellaneous	Alkaloids (berberin)	Hardwood
	Furocoumarins (psoralen)	Hardwood (tropical)

From Hausen (1981), Henschler (1983) and Swan (1989)

Table 6. Comparison of softwoods and hardwoods

Characteristic	Gymnosperms/conifers/ softwoods	Angiosperms/deciduous wood/hardwoods
World production of industrial roundwood (1980) ($\times 1000 \text{ m}^3$)	990 000	450 000
Density (g/cm^3)	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%
Units	More mannose More galactose	More xylose
Lignin	~25–35%	~20–30%
Units	Mainly guaiacyl	Mainly syringyl or guaiacyl
Methoxy group content	~15%	~20%
Extractives content		
Non-polar (e.g. terpenes)	High	Low
Polar (e.g. tannins)	Low	High

From Fengel & Wegener (1989)

1.2.1 Major woodworking processes

(a) Debarking

Debarking is the mechanical removal of bark from a log and is performed in sawmills and other mills where logs are first processed. Debarking can be done in a number of ways: in wood-to-wood abrasion, the pounding and friction of logs against each other in a rotating drum removes the bark; in the 'flail' method, chains pound against the log to loosen the bark; in peripheral milling, logs are rotated against knives. Bark can also be removed by pressing tool points against the log to loosen the bark and then using a ring debarker or high-pressure water jets. In general, debarking involves little or no exposure to wood dust, because the wood is 'green' (fresh) and thus has a high moisture content. Furthermore, the main goal of the operation is to leave the wood intact.

(b) Sawing

Saws are used to cut logs or large pieces of wood into appropriate sizes for further modification and use. Sawing is performed by drawing a blade with a series of sharpened teeth through the wood. As with many woodworking machines, the amount of wood dust generated by mechanical sawing operations is influenced by the speed of the sawing action, the angle of cutting relative to the wood grain and the sharpness and width of the blade. Sawing against the wood grain (cross-cutting) is more likely to shatter wood cells than sawing lengthwise (ripping). Sharp, thin blades produce less wood dust by volume because the kerf, the cut made in the wood, is narrower, but the particle sizes are also likely to be smaller.

The simplest saws are blades with a series of teeth along one edge and a handle on one or both ends; they are powered by a human operator or operators, who move the blade back and forth. Almost all saws used for commercial purposes are mechanically operated. In recent decades, high-energy jets and lasers have been introduced, which generate less wood dust but are not in broad commercial use. The commonest types of mechanical saws are described below.

(i) Band saw

The blade of a band saw is a continuous metal strip with teeth on one or both edges which rotates around two wheels. Band saws are used in many wood industries, from sawmills to wood product manufacturing and can be powered by steam, hydraulic or electric mechanisms.

(ii) Circular saw

The blade of a circular saw is a rigid metal disk with teeth along its circumference which cuts as it rotates. Circular saws are also used in many wood industries. A table saw is a circular saw with the blade protruding through a table; a radial arm saw is a circular saw suspended above the working surface on a movable armature.

(iii) Sash gang saw

In a sash gang saw (frame saw), a series of parallel blades (a gang) fixed between two vertical members (a sash) are drawn up and down to rip boards being moved through the saw on rollers. These saws are used almost exclusively in sawmills to cut large pieces of lumber lengthwise in order to create a set of boards. Circular saws are also commonly used for gang sawing.

(iv) Jig saw

A jig saw has a short, rigid blade attached to a reciprocating mechanism to cut with an up-and-down motion. Jig saws are used in many wood products industries and can be both hand-held and stationary.

(v) Chain saw

A chain saw has a continuous articulated chain with teeth along its outer edge. It is generally powered by a gasoline engine and is used almost exclusively in logging, although it may sometimes be used as a cut-off saw in sawmills.

(c) *Sanding*

Sanding is smoothing the surface of wood, 'an abrasive process in which sharp edges of small, hard, crystalline particles are rapidly drawn across the surface of the wood with pressure being applied perpendicular to the surface' (Holliday *et al.*, 1986). The abrasives used include carborundum, emery, glass and pumice. The smaller the abrasive particles, the finer the dust produced; and the faster the sander, the greater the volume of dust produced. Sanding is done in many wood industries, with small, hand-held sanders or generally larger stationary machines. Sanders can range in size from hand-held to large drums or belts for smoothing a full panel. The commonest types of sander are the belt sander, a continuous strip of sandpaper rotated around two rollers; the disk sander, a circular piece of sandpaper fastened to a rotating disk; the drum sander, a continuous strip of sandpaper rotated on a drum; and the orbital sander, which operates with an elliptical, vibrating motion.

(d) *Planing, jointing, moulding and shaping*

Planing, jointing, moulding and shaping are milling processes. A planer is used to smooth one or more sides of a piece of wood and at the same time to reduce it to a predetermined thickness. The planer head is made up of a series of cutting blades mounted on a cylinder, which revolves at high speed. The operation is generally performed parallel to the wood grain.

A jointer is a machine for squaring and smoothing the edges of lumber or panels. Jointers are used especially in preparation for glueing and in other situations in which a smooth surface is needed. Jointers vary in size from small, hand-held devices to large, stationary machines. A jointer is similar to a planer in its operation, but its blades are generally smaller, and it is designed to smooth or true a surface rather than to change the thickness of a board.

Moulders are used to cut and shape mouldings. They generally consist of a top cutter head, followed by two sideheads and by a bottom cutterhead. The cutterheads are staggered spindles of various designs. Shapers are similar to moulders, but are used to cut and shape the outer sides of wood boards and products. Shapers generally consist of a table through which protrudes a rotating spindle with blades shaped to produce the desired contour. Shapers can be used to cut the edge designs found on furniture and picture frames and in many other applications, such as wooden model making.

(e) *Turning (lathing)*

Turning involves use of a lathe to produce cylindrical shapes in wooden objects. One end of the piece of wood is fixed to a clamp or plate, which is rotated. The point of operation is a tool point or a long knife, which has a cutting, scraping or shearing action (depending on the angle of contact) when applied against the wood. Some lathes rotate the piece quickly while the tool or knife remains stationary and in continuous contact until the desired shape is formed; other lathes rotate the piece slowly and have a peripheral milling cutterhead.

(f) *Boring (drilling), routing and carving*

Boring machines are designed to drill holes for dowel joists, screws and other purposes. They are similar to drills and drill presses used for other materials and contain rotating bits of various designs. Routers are used to shape the edges and corners of wooden objects and to cut

slots of various shapes; a spindle is suspended over the piece, and the process combines aspects of boring and milling. Carving machines are rotating tools mounted on spindles, which are designed for both side and end cutting.

(g) *Mortising and tenoning*

A mortise is a cavity cut into a piece of wood to receive a tenon (a protrusion), which together form a mortise-tenon joint. Mortises are of various shapes and can be formed by different machines. The commonest is the hollow chisel mortiser, which forms a rectangular mortise with a hollow chisel or shell, inside of which are a rotating boring bit or bits. The bits cut the hole, which is squared by the sharp edges of the chisel. Chain mortisers, similar in design to chain saws, and oscillating bit mortisers, which are specialized routers, can also be used. Tenoning machines are also of various designs, involving both milling and sawing actions. End matchers are tenoning machines used to create both the tongue and groove for hardwood flooring.

(h) *Veneer cutting*

A rotary peeler is a lathe-like machine used to cut veneers, thin sheets of wood, from whole logs using a shearing action. The log is rotated against a pressure bar as it hits a cutting knife to produce a thin sheet of 0.25–5 mm in thickness. The logs used in this process may be softened before use by soaking in hot water or steaming. The edges of the sheet are usually trimmed with knives attached to the pressure bar. Veneers are used as decorative laminates or for the manufacture of plywood. Because the moisture content is high, very little wood dust is usually generated by this process.

(i) *Chipping, flaking, hogging and grinding*

Chippers, sometimes known as 'hackers' in Europe, are generally large rotating discs with blades embedded in the face and slots for chips to pass through. The chips are produced when logs or mill wastes are introduced to the blades by inclined gravity feed, horizontal self-feed or controlled power feeding. Generally, the cutting action of the chipper is perpendicular to the blades, and different designs are used for whole logs and for slabs and edgings. Although the term 'chipping' is sometimes used to refer to related processes, such as flaking and hogging, the end product is quite different. Chippers are used in many industries to reduce logs and wood waste to uniform-sized chips for pulp, reconstituted board and other uses.

Flaking machines convert wood into flakes for use as the raw material for particle-board, flake-board and wafer-board. They may be similar in design to a chipper, except that the wood must be fed to the flaker with the grain orientated parallel to the knives. Peripheral milling designs are also used. Wafers are generally made directly from logs that have been stored in a holding pond using a waferizer, a machine containing a series of rotating knives which peel thin wafers (Holliday *et al.*, 1986). Water-saturated wood is best for these processes, and, because the wood must be orientated, short logs are often used. Because the moisture content is high and the wood is orientated, less dust is generated than is commonly the case with chipping.

Hogs are used to reduce pieces of wood and residues into chips for use as fuel or for other purposes in which a uniform size is not required. Hogging machines are of various designs,

including knife-type hogs, with rotating cylinders bearing protruding knives, rotating disks with progressively smaller teeth, and hammer mills, with rapidly revolving 'hammers' that cut the wood by impact. Hogging produces chips of maximal size but not of uniform shape or size.

Grinding is used to reduce wood chips to the consistency of flour. Hammer mills and grinding plates are used to pulverize or grind the wood, and the resulting product is sifted to control the size. Wood flour can be used for a ground cover or animal bedding or as a sweeping compound, filler or extender for composition boards and plastics, depending on the size of the particles. As the wood used for this process is generally dry, exposure to dust in uncontrolled settings is high.

(j) Mechanical defibrating

A mechanical defibrator is a grinding machine used to break wood down into fibres for wood pulp and various types of fibre-board. The wood must have a high moisture content; species such as spruce and fir are preferred because of their light colour, uniform structure and high fibre content. Short logs are forced against a grindstone, made of natural stone or of artificial stone composed of silicon carbide or aluminium oxide. The stone is showered with water to remove the pulp and cool the surface. Little or no exposure to wood dust should occur during this wet process.

1.2.2 Sawmilling

The raw materials for sawmills are supplied by the forestry and logging industries. Workers responsible for cutting down trees (felling), sawing felled trees into log lengths (bucking), trimming off branches and clearing brush are most likely to be exposed to wood dust. Sawing and cutting are usually performed with chain saws, although axes, hand saws and malls (metal wedges) may also be used. Trailer-mounted chipping or hogging machines may be used at logging sites. As wood that is sawn and chipped has a very high moisture content, the particles of dust generated are likely to be large. Trees may also be sheared with hydraulic mechanisms mounted on logging tractors; in general, little dust is produced during the shearing of fresh wood. Although other logging workers, such as those involved in yarding and loading, may be exposed to wood dust, such exposures would probably be low.

Sawmills vary greatly in size, and operations are performed outdoors or indoors depending on the size of the mill and the climate of the region. The smallest sawmills are mobile or portable units consisting of a circular saw, a simple log carriage and a two-saw edger powered by a diesel or gasoline engine and operated by two to four workers (FAO, 1981). The largest mills are permanent structures, have much more elaborate, specialized equipment and may employ more than 1000 workers. A representative production line and various phases of work at a typical Scandinavian sawmill were described previously (IARC, 1981). The equipment in sawmills varies considerably with the age and size of the mill and the type and quality of boards produced.

After transport to a sawmill, logs are stored on land, in bodies of water adjacent to the mill or in ponds constructed for the purposes. The first process is debarking; as the wood is green or has been stored in water, little wood dust is produced. This process has been described as

'messy' rather than dusty, as earth, mould and fungus particles may adhere to the surface of the bark (Holliday *et al.*, 1986).

A cut-off saw, usually a circular saw or a very large chain saw, is used to even up the ends of the trunks before primary breakdown (the first phase of sawmilling) at a headrig. The headrig is a large stationary circular or bandsaw used to cut the log longitudinally. The log is transported to the headrig on a travelling carriage, which can rotate the log 90 or 180° and carries it back and forth through the headrig. Multiple band headrigs may also be used, especially for smaller logs, so that only a single pass may be needed (Williston, 1988). The products of the headrig are a cant (the square centre of the log), a series of slabs (the rounded outer edges of the log) and, in some cases, large boards. In secondary breakdown (or 'resaw'), the cant and large boards are further processed into usable sized boards. In small mills, a circular or band saw may be used; in larger mills, the cant and large boards are generally processed with gang saws of either the sash, circular or band saw type. Boards are cut to the proper width with edgers consisting of at least two parallel saws and to the proper length with trim saws. Edging and trimming can be done with circular or band saws; in some cases, chain saws are used for trimming. Exposure to wood dust may occur in all these operations, but the concentration varies greatly, depending on the distance from the point of operation and whether or not the worker is operating the saw from an enclosed booth (Teschke *et al.*, 1994).

In many mills, the slabs and other waste wood are chipped. Chipping is generally a separate process, but in some cases a chipper may be integrated into the headrig to increase efficiency (Williston, 1988). Wood chips and sawdust may be sold for pulp or used for reconstituted board manufacture, landscaping, fuel and other uses. Exposure to wood dust may occur during chipping.

After breakdown, the boards are sorted according to dimensions and grade and then stacked by hand or machine to await drying, also referred to as seasoning. At this point, fungicides may be applied, either by dipping single boards or bundles or by various spraying procedures, to prevent the growth of fungi on the sap which stain the surface of the wood blue (Kauppinen & Lindroos, 1985).

Cut lumber is either dried in the open air or, more commonly, in various types of kilns, including serial compartment and high-temperature kilns and continuous kilns, in which stacked bundles can move in a perpendicular or parallel position and the air movement is perpendicular or parallel to the boards. Exposure to wood dust is generally low in these operations.

Either before or after drying, the wood is marketable as a green or rough lumber; however, for most industrial uses, it must be processed further. Lumber is cut to its final size and surfaced in a planing mill, usually simultaneously on two sides of the board; planers that operate on all four sides may be called matchers. Moulders are sometimes used to round the edges of the wood. Exposure to wood dust may occur during planing and moulding, because the wood is dry and the aim of the operations is to produce a relatively smooth surface. Dust control systems, such as local exhaust ventilation, may be present in these operations; however, their effectiveness in controlling exposures is not certain (Teschke *et al.*, 1994).

After processing, wood is sorted, stacked and bundled in preparation for shipping. Workers may be exposed to wood dust during these operations, especially if no measures have been taken to remove dust after surfacing operations.

1.2.3 *Manufacture of plywood and other boards*

Plywood, particle-board and other boards consist of wood components of varying sizes, ranging from veneers to fibres, held together by an adhesive bond. The simplest of these boards are created in two steps: generation of the components, directly from whole logs or, for some products, from woodworking waste or non-commercial or low-value tree species; and their recombination into sheets with chemical resins or, in the case of wet process fibre-board, 'natural' bonding. These steps may be carried out at different locations, especially when woodworking waste is used. The manufacture of plywood, particle-board, wafer-board, strand-board, insulation board, fibre-board and hard-board are all relatively new industries which first became commercially important during this century, especially since the 1940s. For example, although techniques for making plywood have existed for many centuries, the term 'plywood' did not enter common usage until the 1920s (Maloney, 1977).

(a) *Plywood manufacture*

The term 'plywood' is used for panels consisting of three or more veneers that have been glued together. Plywood can be made from either softwood or hardwood. Veneers are usually created directly from debarked whole logs by rotary peeling; decorative veneers can be created by slicing a cant with a pressure arm and blade in a manner similar to peeling. The veneers are used either for manufacturing plywood or as decorative laminates for particle-board and other reconstituted boards. After peeling or slicing, the veneers are collected on long, flat trays or rolled onto reels and are then clipped into usable lengths with a guillotine-like machine and dried by artificial heating or natural ventilation. The dried panels are inspected and, if necessary, patched with small pieces or strips of wood and formaldehyde-based resins. If the dried veneers are too small, they can be spliced together by applying a liquid formaldehyde-based adhesive to the edges, pressing the edges together and applying heat to cure the resin. As the wood used to produce veneers is wet and the peeling and clipping operations do not generally produce much dust, relatively little exposure to wood dust occurs during these operations (Holliday *et al.*, 1986).

Plywood panels are produced by placing veneers that are roller- or spray-coated with formaldehyde-based resins between two unglued veneers. The plies are then stacked perpendicular to each other with respect to grain and transferred to a hot press, where they are subjected to pressure and heat in order to cure the resin. They are then cut to the proper dimensions with circular saws and surfaced with large drum or belt sanders. Additional machining may be done to give the plywood special characteristics.

The highest exposures to wood dust during the production of plywood occur in sanding, machining and sawing. Sanding can produce particularly large amounts of dust, since as much as 10–15% of the board may be removed during surfacing (Holliday *et al.*, 1986). These processes are now generally enclosed or done with local exhaust ventilation.

(b) *Manufacture of particle-board and related boards*

Particle-board (chipboard), flake-board, strand-board and wafer-board are made from chips of wood of various sizes and shapes using similar processes. Wafer-board and strand-board are made from very large particles—wood shavings and strands, respectively—and are used primarily for structural applications. Particle-board and flake-board are made from smaller wood chips and are often used to make wood-veneered and plastic-laminated panels for the manufacture of furniture, cabinets and other wood products. Most elements are made directly from logs, branches and mill waste.

The processes used for making reconstituted panels are generally the same. The elements must be sorted by size and grade and then dried, by artificial means, to a closely controlled moisture content. The dried elements are mixed with an adhesive (a phenol-formaldehyde or urea-formaldehyde resin) and laid out in mats. The mats are cut into sections, generally with a circular cut-off saw. The panels are formed into sheets by curing the thermosetting resin in a hot press and are cooled and trimmed to size. If necessary, sanders are used to finish the surface; drum sanders were used earlier, but wide belt sanders are now generally used (Maloney, 1977). Most sanders are enclosed, and large-capacity air systems are necessary to remove the dust generated. Reconstituted boards that are to be covered with a wood veneer or plastic laminate must be sanded, and surface coatings may be applied.

Reconstituted panels are made from either hardwood or softwood. Exposure to wood dust may occur during processing but varies greatly with the moisture content of the wood and the nature of the process. The highest exposures may occur during chipping and grinding of dried wood, and high exposures occur during cutting and finishing of panels, especially in sanding operations, if engineering controls are not in place or functioning properly.

In recent decades, a new industry has emerged to produce reconstituted lumber for various structural uses, such as beams, supports and other weight-bearing elements. While the manufacturing processes used may be similar to those used for making particle-board, isocyanate-based resins are used to add strength.

(c) *Fibre-board manufacture*

Fibre-boards are panels consisting of bonded wood fibres. The fibres are made by reducing (pulping) short logs or wood chips in a manner similar to that used for producing pulp for the paper industry (see IARC, 1981). A mechanical (groundwood) pulping process is usually used, in which chips are soaked in hot water and then ground mechanically. A wet or a dry process may be used to bond the fibres and create the panels. The wet process, based on paper production, was developed first; the dry process, which stems from particle-board techniques, was developed later. In the wet process, a slurry of pulp and water is distributed on a screen to form a mat, which is pressed, dried, cut and surfaced. The boards created by wet processes are held together by natural adhesive-like wood components and the formation of hydrogen bonds (Suchsland & Woodson, 1986). The dry process is similar, except that the fibres are distributed on the mat after addition of a binder (a thermosetting or thermoplastic resin or a drying oil) which forms a bond between the fibres. Fibre-boards vary greatly in density. Hard-board (high-

density fibre-board) and medium-density fibre-board can be produced by wet or dry processes, while insulation board (low-density fibre-board) can be produced only by the wet process.

Fibre-boards can be made from either softwoods or hardwoods. Hardwoods generally make better hard-board, while softwoods make better insulation board (Suchsland & Woodson, 1986). Exposure to wood dust in the fibre-board industry may occur during debarking, cutting of logs to size and chipping (if these are not performed elsewhere) or the handling of wood chips before pulping. The processes involved in pulping have a chemical effect on the groundwood and some of the lignin and extractive materials may be removed; the dust generated during cutting of fibre-board and finishing operations may therefore differ from unprocessed wood dust (Holliday *et al.*, 1986).

1.2.4 *Wooden furniture manufacture and cabinet-making*

Traditionally, furniture is made from solid wood, and many different tree species have been used. Common species include hardwoods, such as sycamore, birch, oak, hickory, cherry, beech, ash and walnut, tropical woods such as mahogany, ebony and teak, and softwoods such as pine, fir, redwood, cedar and larch (Darcy, 1984). In this century, veneer- and plastic-covered chip-board and fibre-board panels have been used increasingly for the manufacture of cabinets, table tops and similar products. Solid hardwoods and hardwood-veneered panels are used for high-quality furniture because of the attractive patterns formed by their grains. In the furniture factories of the High Wycombe area of England, mainly beech, ash and elm are used for making tables and chairs, while elm, ash, veneered chip-board and fibre-board are used for cabinets and similar products (Jones & Smith, 1986).

The wooden furniture industry includes a wide variety of woodworking and non-woodworking operations. Various phases of the production of furniture and cabinets were described previously (IARC, 1981). In order to examine patterns of exposure to wood dust in the furniture industry in High Wycombe, Jones and Smith (1986) identified three stages of furniture production during which exposure may occur: conversion, component making and assembly. Although manufacturing processes vary by country and type of furniture produced, these three stages, sometimes under different names, generally occur in most facilities.

The 'conversion' stage is also referred to as rough milling, rough sizing or breakdown, when rough lumber is cut into the standard sizes needed for further machining. A variety of sawing and planing operations are performed during this stage (Darcy, 1984; Jones & Smith, 1986), usually with stationary machines. The wood used for furniture must generally be well seasoned (dried), and some facilities have kilns to further lower the moisture content before sawing and planing. Wood waste may be reduced with a hogger or chipper. Exposure to wood dust may occur during sawing, planing and chipping. Because of the nature of the operations, exposures should not be high, but if local exhaust ventilation is not used exposure to wood dust could occur.

The second stage, 'component making', is also referred to as machining or machine room operations. The converted pieces of lumber are cut to finished sizes and machined into the components (arms, legs, tops, sides) needed to make furniture. Some sawing and planing is done; in particular, bandsaws are used to shape pieces roughly before machining. To produce a

variety of end-products, different milling machines are used, including jointers, routers, moulders, shapers, tenoners, lathes, boring machines and carving machines. The components may also be sanded, using brush, belt and drum sanders. Exposure to wood dust may occur during sawing, machining and sanding, although use of stationary machines and local exhaust ventilation may reduce exposure. If control measures are not used or are ineffective, however, exposure could be high. While most furniture is produced with machines, some workshops (in Italy and Spain, for example) produce traditional furniture or furniture that resembles antiques using many manual operations (IARC, 1981).

The final stage of production is 'assembly', when the components are put together. The potential source of wood dust at this stage is sanding, usually after assembly, which is often done with hand-held power tools. Dust control is more difficult for such operations. Although sanding was commonly performed at this stage in the English furniture industry, resulting in high exposures (Jones & Smith, 1986), assembled furniture may not require sanding.

Although the operations described above are the main sources of wood dust in the furniture industry, workers with other duties are also potentially exposed. Cleaning and maintenance workers may be exposed while removing dust from a work area or cleaning machinery or ventilation equipment. Their degree of exposure is directly related to the methods used to remove the dust; wet methods and vacuuming produce little dust, while sweeping and brushing may result in exposure, and the use of compressed air to blow off dust can result in high exposures. Assemblers, material handlers and other non-woodworkers, such as glue and upholstery workers, may be exposed if they perform their duties while the wood is still dusty. The furniture industry typically includes many operations in which there is little or no exposure to wood dust because a clean surface is needed for the operation to be effective. These operations include staining, varnishing, lacquering and painting. Generally, workers employed in these operations have little exposure to wood dust. In some situations (e.g. small factories and shops), workers may be employed in multiple phases of production.

French polish is a solution consisting of shellac dissolved in methanol. 'French polishers', however, not only apply this solution but sand down the surface after each coat has dried; the operation may be repeated tens of times. They were classified in the same category as stainers, sprayers and spray polishers in the study of Acheson *et al.* (1984) and were placed in the middle exposure category in the analysis.

Cabinet-making is a skilled trade closely related to furniture making. Cabinet-makers are highly skilled workers who are trained to operate a variety of woodworking machines and use various hand tools to fabricate and repair high-grade furniture. They may also be responsible for finishing surfaces with paints, stains and varnishes and for installing hardware, such as hinges and handles, and for other non-woodworking tasks. Cabinet-makers work in a variety of settings, from large furniture factories to small cabinet-making shops. They may also be employed in the construction industry to build, install and repair cabinets and other furniture and fixtures in both new and existing structures.

1.2.5 Manufacture of other wood products

Other products that may be manufactured from wood are musical instruments, sports equipment, kitchen utensils, wooden boxes, toys, rifle stocks, smoking pipes, coffins, doors and sashes, boats, mobile homes, wooden pallets, flooring, railroad ties, barrels and kegs, prefabricated structures, crates and fences. Exposure to wood dust varies according to the processing operations used, the type of wood used and other factors discussed in sections 1.2.1 and 1.4.1. The type of wood used is related to the use of the product; for example, products such as flooring, parquetry, baseball bats and tool handles are often made from hardwoods for aesthetic reasons and because of their durability. Doors, frames, panels and toys are often made from softwoods because of the ease with which these woods can be cut and machined.

The stages of production in which exposure to wood dust may occur can be categorized in the same way as for the wooden furniture industry in England (Jones & Smith, 1986): an initial phase mainly of machine sawing and planing to convert rough lumber into the sizes needed for further machining; an intermediate phase to cut the pieces of lumber to final size, machine and sand them; and a final stage to assemble the components, which may be sanded as part of the finishing. There is considerable variation between industries. For example, wooden pallets are made from rough lumber and require only sawing and assembly; or the final product of some industries may be a component for another industry, such as the stock for a rifle or the face of a clock. Wooden boats and other products that may be subjected to harsh environmental conditions are sometimes constructed of woods that are naturally resistant to environmental degradation, such as cypress, cedar and teak.

In some of these industries, exposures may be similar to those in the construction industry (see below). For example, the manufacture of prefabricated structures and mobile homes is very similar to construction carpentry, except that the operations are generally performed within an enclosed space. As in the construction industry, softwood is often used for framing and other structural purposes.

1.2.6 Construction, carpentry and other wood-related occupations

(a) The construction industry

Carpenters and joiners are skilled woodworkers employed extensively in the construction industry. Carpenters are responsible for the construction, erection, installation and repair of wooden structures and fixtures. Joiners are usually involved in the finer aspects of construction and the finishing of buildings. Carpenters use various saws, planers and chisels to perform their tasks, while joiners use a wider variety of tools and may perform some of their work away from the building site. The line between these two trades, which had separate guilds during the Middle Ages, is not always clear, however, and they may differ between countries. For example, in the United States, carpenters perform both types of tasks, while in France there is a greater distinction between carpenters and joiners. The term 'finish carpenter' is sometimes used to refer to workers who specialize in installing wooden trim, stairs and floors and other finishing operations.

Woodworking in the construction industry differs from that in the manufacturing industry, in that carpentry and some aspects of joinery are usually carried out at building sites, where conditions are constantly changing and hand tools are still used extensively. These two factors make it difficult to control or monitor exposures. When work is done outdoors, natural ventilation may lower the potential exposure, although operations such as sanding may still result in high exposures to wood dust. Construction-related woodwork carried out in shops with stationary equipment generates exposures to wood dust and other materials that are similar to those in other wood product industries.

Construction involves excavation, building foundations, framing, electrical installation, plumbing, roofing and finishing. Carpenters and other woodworkers may be involved in at least three of these steps: building foundations, framing and finishing. They may be responsible for constructing wooden forms for concrete foundations for both wooden and metal structures, either at the construction site or by prefabrication, from plywood and other reconstituted boards that have been specially treated with light petroleum oils (see IARC, 1989a). The framing of wooden structures involves the preparation, trimming and assembly of the various pieces of wood that comprise the weight-bearing members of a structure, including the roof timbers, beams, floor joists, wall sections, staircases and supports. This work is commonly performed on-site, although the pieces may be pre-cut and, in some situations, pre-assembled off-site. Softwoods, often pine, are most commonly used for beams, trusses, joists and studs, although hardwoods, such as oak, chestnut and elm, may also be used. Reconstituted wood products are often used for walls and underflooring. Wood that has been treated with preservatives, such as chromated copper arsenate (see IARC, 1987a) and chlorophenol derivatives (see IARC, 1986, 1987b), may be used for external walls and in other situations where the wood may be exposed to adverse conditions. A variety of saws and planers may be used, as well as simple hand tools such as chisels and hammers. Exposure to wood dust is rarely high during framing; however, insulation work performed at the same time as framing may involve exposure to insulating materials (see e.g. IARC, 1988).

Carpenters, joiners and other woodworkers, such as cabinet-makers, floor layers and parquetry workers, may be involved in finishing wooden and non-wooden structures, which involves installation of floorboards, staircases, door and window frames and sashes, moulding, cabinets and panelling of structures. Circular saws of various kinds, including table saws and radial arm saws, bandsaws, sanders of various kinds, including hand-held belt and rotary sanders, planers, routers, moulders and tenoners are commonly used. Hardwoods, softwoods, tropical woods and reconstituted panels are all used in finishing. Finishing generates the greatest potential exposure to wood dust, because sanding is done frequently, often in partly or fully enclosed spaces. Sanding, and particularly the sanding of floors, can result in high exposures to wood dust, and such operations are often carried out by workers who do not have adequate protection to avoid respiration of the dust.

(b) Maintenance and repair

Carpenters and joiners may also be responsible for the maintenance and repair of wooden structures and fixtures in industries varying from the services to manufacturing. Their exposures

are similar to those of wooden construction workers, except that the work is more commonly performed indoors and may include other exposures. Although the terms 'carpenter' and 'joiner' are usually associated with the construction and maintenance trades, they are used in many industries to refer to skilled woodworkers. The term 'joiner' is also used in boat building and repair to refer to skilled workers who fabricate, assemble, install or repair wooden furnishings in ships and boats. Carpenters who work on wooden boats and ships are also referred to as 'shipwrights'.

(c) *Pattern and model making*

Wooden pattern makers plan, lay out and construct wooden units or sectional patterns, such as those used in forming sand moulds for casting. Wooden model makers construct precision models of products or parts used in mass production. Both pattern and model makers are highly skilled workers, who are employed by small shops or directly by mass production industries. Pattern and model making are not, however, mass production operations: each piece is made individually, starting from blueprints and ending with a finished product which must often meet very close tolerances. These workers use hand tools, measuring instruments and woodworking machinery such as bandsaws, lathes, planers, routers and shapers and a variety of hard, soft, tropical and laminated woods. The raw material used depends on the intended use; for example, in the United States automobile industry, softwoods are often used for experimental models, while harder and laminated woods are used for models that require more exact, stable dimensions (McCammon *et al.*, 1985).

(d) *Wood shop teachers and artists*

Exposure to wood dust may occur in a number of other occupational settings, including the teaching of woodworking, wood sculpture and design secondary schools, technical schools and universities. In such classes, a variety of woodworking machinery may be used in largely unregulated environments, under health and safety conditions that would not be acceptable in an industrial setting. For example, Lucas and Salisbury (1992) reported that the equipment used by a design materials class in a university art department included a planer, table saw, jointer, lathe, belt sander and band saw and many portable power tools. While the stationary machines had local exhaust ventilation, the fabric bag dust collector was located inside the classroom. Although students may be exposed intermittently, teachers may spend many hours per day in the same setting. Artists who create wooden sculptures and artisans making wooden objects may work under similar conditions.

1.3 Analytical methods

1.3.1 *Characterization and measurement of wood dust*

Wood dust and exposures to wood dust are characterized in several ways that affect the nature of exposures in woodworking industries: by type of wood, as airborne dust concentrations, by particle size distribution and by other parameters.

(a) *Type of wood*

Wood dust is frequently described by wood species or as hardwood or softwood (see section 1.1). Wood dust is also characterized by its moisture content: Dry wood (moisture content less than about 15–20%) is less elastic than moist (green) wood, and woodworking operations with dry wood result in a larger volume of total dust and a higher percentage of inhalable dust particles (Hinds, 1988).

(b) *Airborne dust concentrations*

(i) *Total dust measurements*

The parameter most commonly used to characterize exposures to wood dust in air is total wood dust concentration, in mass per unit volume (usually mg/m^3). Standard gravimetric methods for measuring total dust concentrations, such as NIOSH Method 0500 (Eller, 1984a), have been used routinely. In this general method, a known volume of air is drawn through a special membrane filter contained in a plastic cassette with a sampling pump. The dust concentration is calculated from the change in weight of the filter divided by the volume of air sampled, with a detection limit for personal sampling of wood dust of about $0.1 \text{ mg}/\text{m}^3$. Polyvinyl chloride filters are preferred for sampling wood dust because of its highly variable moisture content. Filters are environmentally equilibrated before and after sampling to avoid spurious effects from differential moisture uptake (Eller, 1984a; Holliday *et al.*, 1986; Sass-Kortsak *et al.*, 1989; Teschke *et al.*, 1994).

The cassette holding the filter is either open- or closed-faced during sampling. In the closed-faced mode, a cap with a 4-mm hole is placed over the 37-mm cassette face to protect the filter. Closed-faced operation is usually recommended when total suspended particulates are being measured (see, for example, United States Occupational Safety and Health Administration, 1993). Beaulieu *et al.* (1980) reported, however, that the open-faced filter collected 30–60% more dust (by weight) than the closed-faced filter, and they suggested that particles larger than about $10 \mu\text{m}$ are collected very inefficiently in the closed-faced configuration, as corroborated subsequently in other studies (Clayton Environmental Consultants, 1988; Hinds, 1988). Other authors have cautioned against the collection of particles 'too large to be inhaled' (Darcy, 1984) which would contribute disproportionately to the total weight of dust (Hounam & Williams, 1974). As an alternative, the United Kingdom Health and Safety Executive recommends a sampling head with seven 4-mm holes, the sampling characteristics of which appear to approximate current definitions of inhalable dust (Jones & Smith, 1986; United Kingdom Health & Safety Executive, 1989; Hamill *et al.*, 1991; Pisaniello *et al.*, 1991).

(ii) *Particle size-selective measurements*

A number of methods have been used to measure, more or less selectively, exposures to wood dust in the respirable particle size range (Hinds, 1988). NIOSH Method 0600 (Eller, 1984b) is intended for measurement of general 'respirable dust' concentrations. The equipment used is the same as that for NIOSH Method 0500, except that air is sampled through a 10-mm nylon cyclone (centrifugal separator) designed to accept 50% of unit density spherical particles of $3.5 \mu\text{m}$ aerodynamic diameter. Another standard technique, the horizontal elutriator

(gravitational separator), is designed to collect a respirable particulate fraction defined by the British Medical Research Council as 50% of particles of 5 μm aerodynamic diameter (Sass-Kortsak *et al.*, 1993).

The performance of the nylon cyclone, the horizontal elutriator and an aluminium cyclone for measuring wood dust were compared directly in an environmentally controlled chamber at various levels of humidity. Higher levels (by about 25%) were consistently measured with the aluminium cyclone than with the elutriator, with which higher levels (by about 40%) were measured than with the nylon cyclone (Sass-Kortsak *et al.*, 1993). It has been suggested that the nylon cyclone does not accurately separate respirable and nonrespirable wood dust particles because of static charge effects with dry wood dust. The Mine Safety Appliances (MSA) respirable dust cassette, which is similar to the standard 37-mm plastic cassette but contains an aluminium inner capsule that is weighed with the filter, is reportedly about twice as efficient for measuring respirable wood dust as the standard plastic cassette (Moore *et al.*, 1990).

Samplers have been developed to measure exposure to the 'inspirable (or inhalable) particulate mass' fraction, which includes large particulates that may deposit and cause adverse effects on the upper airways. As defined by the American Conference of Governmental Industrial Hygienists (Phalen *et al.*, 1986), these samplers must maintain a sampling efficiency $\geq 50\%$ for particles up to 100 μm aerodynamic diameter. A similar definition was proposed by the International Standards Organization (Vincent & Mark, 1990). The development, evaluation and use of specific samplers for the inspirable particulate mass, including wood dust, have been reported (Mark & Vincent, 1986; Hinds, 1988; Vaughan *et al.*, 1990; Vincent & Mark, 1990; Pisaniello *et al.*, 1991).

An early method for characterizing exposure to wood dust is determination of the number, rather than the mass, of particles in a given volume of air. The konimeter has been used extensively to measure respirable dust of 0.5–5.0 μm by drawing random, small volume, short duration spot ('grab') samples through a small orifice where airborne particles impinge on a glass slide coated with adhesive. The particles trapped on the slide are counted electronically or visually with a microscope. The measurements in grab samples are not, however, comparable to longer duration, time-weighted average concentrations (Holliday *et al.*, 1986).

(c) Particle size distribution

When the distribution of particle sizes in an air sample is to be assessed, other methods must be used. The commonest involves a multi-stage cascade impactor (e.g. the Anderson impactor), which separates particles by mass. The impactor consists of a series of perforated plates through which air is drawn at a constant rate. The dynamics of air flow through the holes (which are of different sizes at each stage) result in trapping of particles with a known range of aerodynamic diameters. The dust collected at each stage can be weighed, and a particle size (mass) distribution can be calculated. Results are reported in various ways, for example as percentage of total mass of dust collected at each stage or as mass median aerodynamic diameter (Whitehead *et al.*, 1981a; Carlin *et al.*, 1981; Holliday *et al.*, 1986; Clayton Environmental Consultants, 1988; Pisaniello *et al.*, 1991).

Holliday *et al.* (1986) reported the analysis of wood dust samples by optical microscopy and classification of particles by equivalent circular diameters calculated from their projected areas by an image analysing computer. The result is a particle size frequency distribution, rather than particle mass distribution.

(d) *Other characteristics of wood dust*

Other characteristics of airborne wood dust are occasionally reported. For example, the irregular shapes of wood dust particles are sometimes recorded in photomicrographs (Holliday *et al.*, 1986) or examined by scanning electron microscopy (McCammon *et al.*, 1985). Particle density (specific gravity) has occasionally been reported (Andersen *et al.*, 1977).

The chemical substances that are natural components, additives or adsorbed contaminants are sometimes extracted with water or organic solvents and characterized (see below). Although there is no standard procedure for measuring the extractable fraction of wood dust, the possible role of these substances in the adverse health effects of wood dust has been the subject of considerable research and speculation.

1.3.2 *Chemical analysis of wood constituents*

A survey of several methods for the chemical analysis of wood constituents has been published (Fengel & Wegener, 1989). In general, organic matter (extractives), inorganic matter (ash) and the main cell wall components, polysaccharides and lignin, are determined. It is important to select a sample for analysis that is representative of the wood species. Standardized sampling procedures have been published: e.g. TAPPI Standard T 257 cm-85 (Technical Association of the Pulp and Paper Industry, 1985a). Before chemical analysis, wood must be milled (particle size, 0.05–0.40 mm) to achieve complete penetration of reagents (Fengel & Wegener, 1989).

(a) *Extractives*

As no modern standard method for the extraction of wood exists, every research group has its own strategy for isolating and identifying chemical constituents of wood. The differences in reported data may thus be due to differences in wood composition or the use of different analytical methods. Conventional methods for investigating compounds present in wood involve either steam distillation or extraction with organic solvents in a soxhlet extractor (Mayer *et al.*, 1969, 1971; Nabeta *et al.*, 1987; Christensen *et al.*, 1988; Kubel *et al.*, 1988; Charrier *et al.*, 1992; Weissmann *et al.*, 1992). Table 7 shows the results of soxhlet extraction of four common wood species with a series of organic solvents of increasing polarity (Weissmann *et al.*, 1992). Supercritical fluid extraction has also been used to isolate these compounds (Torul & Olcay, 1984; Demirbaş, 1991), providing much higher yields of some compounds than those achieved with conventional soxhlet extraction. The thermal stability of wood components is not well characterized, and both soxhlet and supercritical fluid extraction may cause molecular changes, such as decomposition and dimerization (Fengel & Wegener, 1989).

Table 7. Yields (%) of successive extractions of four common wood species

Extraction solvents	European spruce	Scots pine		European beech	European oak	
		Sapwood	Heartwood		Sapwood	Heartwood
<i>Non-polar fractions</i>						
Petroleum ether	0.6	2.2	8.6	0.2	0.15	0.15
Diethyl ether	0.2	0.06	0.8	0.1	0.15	0.35
<i>Polar fractions</i>						
Acetone:water 9:1	0.7	0.3	0.7	1.6	3.6	5.8
Ethanol:water 8:2	0.3	0.4	0.4	1.2	0.9	1.8
Totals	1.8	3.0	10.5	3.1	4.8	8.1

From Weissmann *et al.* (1992)

Groups of non-polar and polar substances resulting from extraction with solvents of increasing polarity can be purified further by chromatographic techniques, such as normal and especially reverse-phase high-performance liquid chromatography (Zinkel, 1983; Suckling *et al.*, 1990; Charrier *et al.*, 1992) and thin-layer chromatography (Nabeta *et al.*, 1987; Kubel *et al.*, 1988). Individual substances are identified by infrared and one- or two-dimensional nuclear magnetic resonance spectroscopy and gas chromatography followed by mass spectroscopy (Mayer *et al.*, 1971; Nabeta *et al.*, 1987; Fengel & Wegener, 1989).

(b) *Inorganic compounds in ash*

The inorganic part of wood is analysed as ash after incineration of the organic wood material at 600–850 °C (Fengel & Wegener, 1989). Detailed methods for ash determination are described in TAPPI Standard T 211 om-85 (Technical Association of the Pulp and Paper Industry, 1985b) and ASTM Standard D 1102-56 (American Society for Testing and Materials, 1965). Particular ash constituents can be identified by methods such as energy dispersive X-ray analysis coupled with scanning or transmission electron microscopy, atomic absorption or emission spectroscopy and neutron activation analysis (Fengel & Wegener, 1989).

(c) *Polysaccharides*

Polyoses, the second group of cell wall polysaccharides, differ from cellulose in their solubility in alkali. Only some polyoses are soluble in water. Some can be extracted directly, and others require removal of lignin before extraction, usually by treating pre-extracted wood with an acidified solution of sodium chlorite (pH 4) at 70–80 °C for 3–4 h. A standard procedure for the isolation and determination of polyoses is successive extraction of chlorite holocellulose with 5 and 24% potassium hydroxide. The insoluble residue represents cellulose.

A general procedure for isolating and determining polysaccharides consists of hydrolysis with concentrated acids and subsequent dilution steps to achieve secondary hydrolysis. Sugars can be identified and quantified after hydrolysis by various chromatographic methods, including

thin-layer and high-performance liquid chromatography, gas chromatography partly combined with mass spectroscopy and ion-exchange chromatography via sugar borate complexes.

(d) *Lignin*

All methods for the isolation of lignin have the disadvantage that they fundamentally change the native structure of lignin or release only parts of it relatively unchanged. All lignin samples obtained by acid hydrolysis are changed with regard to structure and properties, predominantly by condensation reactions. These preparations are therefore not suitable for investigating structures but can be used for estimating lignin content. The commonest method for obtaining relatively unchanged lignin is Björkman's procedure of vibratory milling and subsequent extraction of lignin with aqueous dioxane (Björkman, 1956). In one modification of this method, ultrasound is applied during the extraction step to reduce the isolation time (Wegener & Fengel, 1978).

1.4 Exposure to wood dust and other agents in the workplace

1.4.1 General influences on occupational exposure to wood dust

Woodworking operations such as sawing, milling and sanding both shatter lignified wood cells and break out whole cells and groups of cells (chips). The more cell shattering that occurs, the finer the dust particles that are produced. For example, sawing and milling are mixed cell shattering and chip forming processes, whereas sanding is almost exclusively cell shattering. Since wood cells usually measure about 1 mm, airborne dust concentrations depend primarily on the extent of cell shattering rather than on the size or extent of chip formation (Holliday *et al.*, 1986; Hinds, 1988).

In general, the harder the wood, the more tightly bound are the cells; therefore, more shattering occurs with hardwoods, resulting in more dust. Similarly, the cells in dry wood are less plastic and more likely to be shattered, leading to dust formation. While the moisture content of different species of trees varies, it is also influenced by the freshness (greenness) of the wood. Drying and some other processing of wood, such as sawing and machining, may change the chemical composition of wood dust. For example, some of the low-relative-molecular-mass extractives, such as monoterpenes (see section 1.1.4(b)(i)), may be volatilized. Terpenes evaporate from coniferous wood when it warms up during sawing of logs and edging of boards, and concentrations of 100–550 mg/m³ have been measured in these operations in Swedish sawmills (Hedenstierna *et al.*, 1983).

The orientation of the point of operation relative to the wood surface and grain also influences the generation of dust. Woodworking operations performed parallel to the natural grain of the wood are less likely to shatter cells than processes performed perpendicular to the grain. The volume of wood dust generated also depends on how the process is carried out. For example, machine sanding normally generates more dust than manual sanding.

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. Various measures can be taken to control exposure. A simple, effective measure is to remove the worker from the point of operation by placing controls away from the process or by providing an enclosed booth. These measures are not infrequently taken in sawmills, primarily for safety reasons. Another option is to enclose the operation or to provide local exhaust ventilation. For example, Hampl and Johnston (1985) reported on a ventilation system for horizontal belt sanding that can significantly reduce wood dust emissions, and the American Conference of Governmental Industrial Hygienists (1992) has developed design guides for local ventilation in specific woodworking operations. 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.

A number of characteristics of the workplace may also affect the level of wood dust, e.g. the age, density and types of woodworking machinery and the regulatory environment. Regulations with regard to exposure to wood dust and the enforcement of those regulations may vary between countries or even between industries in the same country. Small workplaces are difficult to regulate and, for various reasons, may have fewer engineering controls in place. The use of engineering controls and occupational health regulations have also changed over time.

The quality of and methods used for cleaning are important, because wood dust that has settled on the floor, equipment and other surfaces may become resuspended. In particular, the use of compressed air hoses to clean off surfaces results in high airborne concentrations of wood dust, while wet cleaning methods and use of vacuum systems may result in little or no exposure to dust. Respirators can be used to reduce exposure, and, because of the size of wood dust particles, even simple paper masks can be relatively effective if properly fitted and used. Woodworking operations conducted outdoors or in semi-enclosed buildings may involve lower exposures because of natural ventilation, but higher exposures could result from wind and the lack of local exhaust ventilation and other engineering controls.

1.4.2 *Extent of exposure to wood dust*

The number of workers exposed to wood dust worldwide has not been estimated in the literature; however, estimates are available for some western countries. The National Occupational Exposure Survey, carried out in 1981–83 in the United States, estimated that about 600 000 workers were exposed to wood dust. The largest numbers of exposed workers were employed in the building trades and the lumber/wood product industries. Forestry workers, e.g. lumberjacks using chain saws, were not considered to be exposed in this survey (United States National Institute for Occupational Safety and Health, 1990). The United States produced 24% of all sawn wood in the world in 1990 (FAO, 1992). According to a Finnish survey, about 70 000 workers were exposed to wood dust in logging or in production of sawn wood, wood products and pulp (Anttila *et al.*, 1992); of these, about 12 900 were estimated to have been exposed routinely to more than 1 mg/m^3 of wood dust and 3800 routinely to more than 5 mg/m^3 (Welling & Kallas, 1991). Some occupations, such as construction carpenters, were considered

to have experienced only occasional exposure to wood dust, and they were not included in the Finnish estimates.

[Country-specific estimates and production statistics allow a crude estimate to be made of the number of the workers exposed worldwide. Assuming that the technical level (workforce demand) and the internal structure of the industries that involve exposure to wood dust are approximately the same as in the reference countries, the United States and Finland, the Working Group estimated that the number of workers occupationally exposed to wood dust worldwide is at least two million and probably much higher.]

The industrial hygienic measurements reviewed in sections 1.4.3–1.4.7 and Tables 8–12 include both personal and area sampling. The results probably represent daily average exposures reasonably well because the sampling time required is normally several hours. Many measurements are made for compliance testing, and workers who have moderate or high potential for exposure tend to be monitored more frequently than others. The results, therefore, are considered to be representative for the specific jobs and operations monitored but are not necessarily representative of exposures throughout industry or for all time periods, job categories or sites.

1.4.3 Exposure during sawmilling

Measured concentrations of wood dust in the air of sawmills and planing mills are presented in Table 8. The levels vary widely, ranging from 0.1 to over 100 mg/m³; the mean values are more frequently below than above 1 mg/m³. In the largest study of sawmills and planing mills in the United States, 33% of the measured concentrations exceeded 1 mg/m³ and 8% exceeded 5 mg/m³ (Clayton Environmental Consultants, 1988). The highest exposure often occurs in the vicinity of chippers, saws and planers, but other operations, such as cleaning, grading and maintenance, may sometimes be dusty.

The results presented in Table 8 are not directly comparable across studies and countries. For example, the low concentrations reported from Canada (Teschke *et al.*, 1994) are partially due to representative sampling. This strategy tends to provide lower results than selective sampling from sites and operations involving high exposure, which is the procedure used in many other studies. The method of measurement and the use of the geometric rather than the arithmetic mean in reporting may also affect the results (see section 1.3.1).

Most measurements reported in the literature are from the 1980s. There are no comprehensive data available to indicate any clear changes over time in the level of wood dust in sawmills or planing mills.

The species of wood processed in sawmills vary. In the Canadian study (Teschke *et al.*, 1994), coniferous (soft) wood, such as hemlock and fir, was processed. Coniferous species (pine and spruce) are also the main raw materials in Finnish sawmills (Welling & Kallas, 1991). Locally and in countries where coniferous trees are rare, deciduous trees may be the main wood used in sawmills. For example, a sawmill in West Virginia, United States, processed white oak,

Table 8. Concentrations of wood dust in sawmills and planer mills

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Sawmills (Canada)	18		0.3–6.1	1985	Holliday <i>et al.</i> (1986)
Sawmills (Canada)	78	0.2 ^b	ND–6	1982–	Vedal <i>et al.</i> (1986)
Sawmilling (Canada)	191 ^c			1989	Teschke <i>et al.</i> (1994)
Sawmills		0.1 ^d			
Yard		0.1 ^d			
Maintenance		0.2 ^d			
Powerhouse		0.2 ^d			
Log boom, kiln, other		0.1 ^d			
Sawmills (USA)	55	2.6	0.7–10.6	1981–82	Morey (1982)
Sawmills and planing mills (USA)	193	0.7 ^d	0.10–410	1987–88	Clayton Environmental Consultants (1988)
Sawmills (Finland)				1980–85	Welling & Kallas (1991)
Sawing	18	1.6	0.1–4.9		
Stacking	3	0.2	0.1–0.3		
Trimming	33	2.8	0.1–28.0		
Packaging	14	1.4	0.23–3.3		
Chipper, hogger	7	1.8	0.6–3.0		
Sawmills (Denmark)	85	0.5 ^e	0.5–0.6 ^f	NR	Vinzents & Laursen (1993)
Sawmills (Germany)	6	2.7 ^b	0.2–50	NR	Scheidt <i>et al.</i> (1989)
Planer mill (Canada)	NR	0.2 ^d		1989	Teschke <i>et al.</i> (1994)
Planer mills (Finland)				1980–85	
Sawing	8	7.8	0.6–35.2		
Planing	11	2.0	0.1–8.4		

ND, not detectable; NR, not reported

^a Arithmetic mean unless otherwise specified; time-weighted average personal and/or area samples

^b Median

^c Including planer mill

^d Geometric mean

^e Mean of geometric means

red oak, poplar, soft maple, basswood and cherry (Morey, P., 1982, cited in United States National Institute for Occupational Safety and Health, 1987).

Sawmill workers may be exposed to chemical agents other than wood dust. Chlorophenols have been used widely in sawmills since the 1940s to prevent staining of freshly cut timber. The chlorophenols used most commonly were pentachlorophenol, tetrachlorophenols and trichlorophenols, which were usually applied to wood as water-soluble salts by dipping or spraying. Although the levels of chlorophenols reported in the air are usually below 0.1 mg/m^3 , heavy exposure may occur through the skin when boards are handled manually immediately after treatment (Kalliokoski & Kauppinen, 1990). Some impurities of chlorophenols—chlorinated phenoxyphenols and polychlorinated dibenzofurans—have also been identified in the sawdust of trimming-grading plants (Levin *et al.*, 1976). Because of concern about the health effects of chlorophenates and their possible contamination with polychlorinated dibenzo-*para*-dioxins (IARC, 1987c), fungicides and other substitutes have been introduced. In Canada and the United States, a mixture of didecyldimethyl ammonium chloride and 3-iodo-2-propynyl butyl carbamate is used (Teschke *et al.*, 1995). Exposure to fungicides may occur if the boards are handled while still wet during grading, sorting and other operations. Many woods, especially those that have been kiln dried, may not need to be treated with fungicides, and some species, such as red cedar, are not susceptible to sapstain fungus.

The numbers of natural fungi and bacteria in wood increase during storage and drying and become suspended in air when wood is processed or handled. For example, the average concentration measured with an Andersen sampler in sawing departments and stacking sites of Finnish sawmills was about 14 000 colony-forming units (cfu)/ m^3 of mesophilic bacteria, 130 cfu/ m^3 of actinomycetes, 660 cfu/ m^3 of xerophilic fungi, 6500 cfu/ m^3 of mesophilic fungi and 3000 cfu/ m^3 of thermotolerant fungi (Kotimaa, 1990).

In specialized mills, wood may be further treated with preservatives, fire retardants or chemicals that protect the surface from mechanical wear or weathering. For example, railroad ties, pilings, fence posts, telephone poles and other wood expected to be in contact with soil or water may be treated with creosote oils (see IARC, 1985b), pentachlorophenol solutions or salts containing copper, chromium (see IARC, 1990) and arsenic (see IARC, 1987a). Stains and colourants may also be used, and paint may be applied to seal the ends of boards or to add company marks.

1.4.4 *Exposure during the manufacture of plywood and other boards*

Concentrations of wood dust in the air of plywood, particle-board and other wood-based panel mills are presented in Table 9. The mean levels in plywood mills are often close to 1 mg/m^3 . In a study in the United States, 27% of the measured values exceeded 1 mg/m^3 in hardwood veneer/plywood mills and 11% in softwood veneer/plywood mills (Clayton Environmental Consultants, 1988). The heaviest exposures usually occur in finishing departments where plywood is sawn and sanded. Some operations, such as drying, assembly and hot pressing, entail hardly any exposure to wood dust.

Table 9. Concentrations of wood dust in plywood, particle-board and related industries

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Plywood mills (USA)					
Edge sawing, sanding, plywood machining	12	1.7	0.7-3.2	1978	Whitehead <i>et al.</i> (1981a)
Veneer lathe, clipper, dryer, dry veneer handling, gluing and pressing	13	0.4	0.1-0.7		
Hardwood veneer and plywood mills (USA)	48	0.8 ^b	0.1-21	1987-88	Clayton Environmental Consultants (1988)
Softwood veneer and plywood mills (USA)	56	0.6 ^b	< 0.1-6.4	1987-88	Clayton Environmental Consultants (1988)
Veneer and plywood mill (Canada)	7		0.1-2.6	1985	Holliday <i>et al.</i> (1986)
Plywood mills (Finland)					
Log debarking/cutting	4	0.4	0.2-0.7	1975-84	Kauppinen (1986)
Peeling	2	NR	0.2-0.3	1975-84	
Sawing of veneers	3	1.6	0.6-3.0	1965-74	
Sawing of veneers	4	1.3	1.1-1.5	1975-84	
Sawing of plywood	6	3.3	0.5-12	1965-74	
Sawing of plywood	11	3.7	0.3-19	1975-84	
Sanding of plywood	5	3.0	0.3-6.4	1965-74	
Sanding of plywood	21	3.8	0.8-22	1975-84	
Chipping in finishing department	11	2.6	0.7-7.1	1975-84	
Finishing department	18	0.7	0.3-2.4	1975-84	
Plywood mills (Finland)				1980-85	Welling & Kallas (1991)
Sawing	24	2.1	0.4-5.0		
Sorting, cleaning, glue mixing, hogger	4	11.1	7.1-15.0		
Particle-board mills (Finland)					Kauppinen & Niemelä (1985)
Hogging	3	11	0.1-29	1975-84	
Chipping	3	1.1	0.7-1.7	1975-84	
Drying of chips	2	NR	24-29	1965-74	
Blending	3	5.3	1.0-8.0	1965-74	
Blending	3	0.8	0.6-0.9	1975-84	
Forming	9	13	4.0-26	1965-74	

Table 9 (contd)

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Particle-board mills (Finland) (contd)					Kauppinen & Niemelä (1985)
Forming	4	0.4	< 0.1–0.5	1975–84	
Hot pressing	6	4.1	1.0–6.1	1965–74	
Hot pressing	5	0.8	< 0.1–2.1	1975–83	
Sawing	4	14	10–20	1965–74	
Sawing	9	1.1	< 0.1–2.3	1975–84	
Reconstituted-board mills (USA)	112	0.7 ^b	0.1–205	1987–88	Clayton Environmental Consultants (1988)
Reconstituted-board mill (Canada)	5		1.5–5.1	1985	Holliday <i>et al.</i> (1986)
Process hard-board mills (USA)	116	0.6 ^b	< 0.1–45	1987–88	Clayton Environmental Consultants (1988)
Fibre-board mill (Finland)					Welling & Kallas (1991)
Piling of boards	2	2.6	1.8–3.3	1980–85	
Sawing of boards	2	3.2	1.8–4.6	1980–85	

NR, not reported

^a Arithmetic mean unless otherwise specified; time-weighted average personal and/or area samples^b Geometric mean

The levels of wood dust in various reconstituted-board (particle-board, fibre-board, hardboard, strand-board) mills exceeded 1 mg/m^3 in 22% of measurements in a large study in the United States (Clayton Environmental Consultants, 1988); however, much higher concentrations have been reported, e.g. in forming and sawing of particle-board, especially before the 1980s (Kauppinen & Niemelä, 1985).

Some data on changes in exposure levels over time are presented in the Table. The level of exposure during sawing and sanding in Finnish plywood mills did not change significantly in two consecutive 10-year periods (Kauppinen, 1986). A substantial decrease in exposure since the mid-1970s has been seen, however, in dusty operations in particle-board mills (Kauppinen & Niemelä, 1985).

Phenol-formaldehyde resin adhesives are widely used to produce softwood plywood for use under severe service conditions, such as for construction and boat building. Urea-formaldehyde resin adhesives are used extensively in producing hardwood plywood for furniture and interior panelling and can be fortified with melamine resin to increase their strength. Before the introduction of formaldehyde-based resins in the 1940s, soya bean and blood-albumin adhesives were used, and cold pressing of panels was common. These operations are still used, but are increasingly rare.

Other agents to which some plywood workers may be exposed include formaldehyde (see monograph, p. 217) and phenol (see IARC, 1989b) emitted from glues, pesticides, heating emissions from coniferous veneers, solvents from coating materials and engine exhaust from forklift trucks. Pesticides that have been used in plywood glues include lindane (see IARC, 1987d), aldrin (see IARC, 1974), heptachlor (see IARC, 1991), chloronaphthalenes, chlorophenols and tributyltin oxide. The mean level of formaldehyde in most operations is now below 1 ppm (1.23 mg/m^3), and exposure to phenol is usually well below that concentration. Most pesticides mixed in glues are only slightly volatile and are not detectable in workroom air; the exception is chloronaphthalenes, which are more volatile. Exposure to pesticides may also occur through the skin. The levels of solvents during painting and other surface treatment of plywood are 1–50 ppm. Levels of terpenes in plywood mills are not detectable ($< 1.5 \text{ ppm}$) in most operations, and the levels were only 1–6 ppm during debarking of pine logs and peeling, drying and sorting of pine veneers, in spite of the obvious presence of a blue haze during processing of pine (Kauppinen, 1986).

Formation of polycyclic aromatic hydrocarbons due to heating during sawing and sanding of plywood could not be detected in measurements carried out in a Finnish plywood mill; however, these compounds may occur in glueing and finishing departments of plywood mills, from exhausts of forklift trucks (Kauppinen, 1986).

Exposures in reconstituted-board mills are similar to those in plywood mills. Formaldehyde-based resins, and especially urea-formaldehyde resin (Kauppinen & Niemelä, 1985), are commonly used in glueing particle-board and other wood-based panels, and the level of formaldehyde in particle-board mills may exceed 1 ppm (1.23 mg/m^3). Urea-formaldehyde resins release formaldehyde during curing more readily than phenol-formaldehyde resins; however, improvements in resin formulation have reduced exposures (Holliday *et al.*, 1986). Exposure may also occur to pesticides, such as heptachlor, and solvents in surface coatings

(Kauppinen & Niemelä, 1985). Workers in the area of stockpiles of untreated wood chips or conveyors used to transport the chips may be exposed to moulds, bacteria and fungi (Cohn *et al.*, 1984).

1.4.5 Exposure during wooden furniture manufacture and cabinet-making

Table 10 summarizes the levels of wood dust in the wooden furniture industry, including cabinet-making. The reported mean levels are higher than in sawmilling and wood-based panel manufacture: a concentration of 1 mg/m^3 was exceeded in 41% of measurements in household furniture manufacture, in 22% in office furniture manufacture and in 52% in kitchen cabinet manufacture in the United States (Clayton Environmental Consultants, 1988). The mean levels shown in Table 10 are frequently between 1 and 10 mg/m^3 . The highest exposures occur in wood machining jobs, such as sanding, and in cabinet-making. Wood is usually machined in separate departments of large furniture plants, but some dusty jobs, such as sanding between applications of varnish layers (e.g. French polishing), may be done in surface coating departments (Welling & Kallas, 1991).

Both hardwood and softwood are commonly used in the manufacture of furniture. The proportions of different species of wood used depends on many factors, such as the country, product, plant and period considered. For example, in the British plants surveyed in 1983 (Jones & Smith, 1986), beech, ash, elm, mahogany, walnut, veneered particle-board and medium density fibre-board were used. In the Finnish furniture industry in 1986, mainly pine and birch were used, but spruce, oak, mahogany, teak and other wood species were employed to some extent (Welling & Kallas, 1991). Case reports and epidemiological studies provide some additional information on the species of wood used in the past in furniture factories of different countries (see section 2).

The mean level of wood dust in seven British furniture factories decreased from 7.8 mg/m^3 (138 samples) in 1976–77 to 4.2 mg/m^3 (209 samples) in 1983, probably due mostly to improvements in local exhaust ventilation of machines (Jones & Smith, 1986).

Other agents than wood dust to which workers in furniture and cabinet-making may be exposed include formaldehyde and solvents from varnishes, paints and glues. In spray-varnishing and -painting and in sanding of surface-coated furniture, workers may also be exposed to nonvolatile components of surface coatings, such as pigments and resins. The level of formaldehyde in the air of surface coating departments of furniture plants varies from 0.1 to over 5 ppm ($0.12 \rightarrow 6.15 \text{ mg/m}^3$), often averaging close to 1 ppm (1.23 mg/m^3). Workers who machine wood may occasionally be exposed to formaldehyde if, for example, formaldehyde-based glues are used in veneering and the hot press is situated close to wood processing machines. In addition, formaldehyde may be released from reconstituted panels during machining (Sass-Kortsak *et al.*, 1986) or may be bound in wood dust aerosol (Stumpf *et al.*, 1986).

Glueing, staining and varnishing are generally done at a distance from woodworking operations, however, so that machine operators and cabinet-makers, who are usually heavily

Table 10. Concentrations of wood dust during furniture and cabinet-making

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Furniture manufacture (United Kingdom)				1973	Hounam & Williams (1974)
Turning	2	8.6	4.6–12.5		
Band sawing	6	4.3	1.0–7.3		
Routing	6	4.1	1.8–8.6		
Assembly	9	5.5	2.1–9.8		
Planing	9	5.0	1.8–10.9		
Sanding	9	7.2	2.0–22.6		
Spindle moulding	8	5.1	1.5–8.4		
Furniture manufacture (United Kingdom)				1983	Jones & Smith (1986)
Conversion	43	2.3	1.0–4.8		
Component making	106	3.4	0.3–53		
Assembly	60	7	0.5–27		
Furniture manufacture (Denmark)				1974–75	Andersen <i>et al.</i> (1977)
Drilling, planing, sawing	27	5.2			
Machine and hand sanding	41	14.3			
Furniture manufacture (USA)				1978	Whitehead <i>et al.</i> (1981a)
Rough mill (softwood)	5	0.6	0.2–1.1		
Rough mill (hardwood)	7	0.8	0.2–2.6		
Assembly (softwood)	2	2.8	2.5–3.1		
Assembly (hardwood)	3	1.5	1.1–2.1		
Lathe, planer, router (hardwood)	9	1.8	0.2–6.3		
Lathe, planer, router (softwood)	9	1.6	0.3–4.3		
Sanding (hardwood)	12	4.5	1.4–11.4		
Sanding (softwood)	13	3.2	0.6–14.3		
Manufacture of household furniture (Canada)				1985	Holliday <i>et al.</i> (1986)
Processing of hardwood	11		0.3–5.2		
Processing of particle-board	6		0.5–6.8		
Processing of softwood, particle-board	5		1.7–15.6		
Manufacture of office furniture (Canada)				1985	Holliday <i>et al.</i> (1986)
Processing of hardwood	7		0.5–1.7		
Processing of particle-board	9		0.4–5.6		

Table 10 (contd)

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Manufacture of household furniture (USA)	112	1.3 ^b	0.2–240	1987–88	Clayton Environmental Consultants (1988)
Manufacture of office furniture (USA)	23	0.8 ^b	0.2–3.8	1987–88	Clayton Environmental Consultants (1988)
Furniture factories (Germany)				NR	Scheidt <i>et al.</i> (1989)
Routing, planing	10	6.0 ^c	3–59		
Sanding	8	2.8 ^c	1.2–9.1		
Sanding (manual)	8	6.1 ^c	2.7–17		
Sawing	19	2.9 ^c	0.4–123		
Furniture manufacture (Australia)				1989–90	Pisaniello <i>et al.</i> (1991)
Wood machinists	99	3.2	0.4–24		
Cabinet-makers	57	5.2	0.4–19		
Chair framemakers	15	3.5	2.0–7		
Manufacture of furniture and fixtures (Finland)				1980–85	Welling & Kallas (1991)
Boring	3	8.7	0.9–22		
Lathing	6	14	1.8–64		
Machine sanding	47	18	0.6–320		
Planing	9	0.8	0.1–2.5		
Routing	10	6.4	0.7–15		
Sanding between varnishing operations	17	16	0.4–81		
Sawing	44	6.8	0.3–73		
Spindle moulding	5	12	0.6–45		
Trimming	2	0.8	0.3–1.3		
Hand sanding	8	20	0.5–60		
Furniture factories (Sweden)	28	2.0	0.3–5.1	NR	Wilhelmsson & Drettner (1984)
Furniture manufacture (Denmark)	396	1.1 ^d	1.1–1.2 ^c	NR	Vinzents & Laursen (1993)
Cabinet-making (Czechoslovakia)			1961–62		Kubiš (1963)
Belt sander	10	24	3.6–65		
Cabinet-making shop (United Kingdom)	71	8.1		NR	Al Zuhair <i>et al.</i> (1981)

Table 10 (contd)

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Cabinet-making (Canada)		NR	1984		Sass-Kortsak <i>et al.</i> (1986)
Assembly	19	1.9			
Laminating, graphics, glueing	3	1.1			
Sanding	7	2.9			
Sawing	12	1.7			
Miscellaneous work	7	1.2			
Manufacture of kitchen cabinets (Canada)			1985		Holliday <i>et al.</i> (1986)
From hardwood	12	0.3–5.1			
From particle-board	5	0.7–3.7			
Manufacture of kitchen cabinets (USA)	42	1.6 ^b	0.3–13	1987–88	Clayton Environmental Consultants (1988)

NR, not reported

^a Arithmetic mean unless otherwise specified; time-weighted average personal and/or area sample

^b Geometric mean

^c Median

^d Mean of geometric means

^e Range of geometric means

exposed to wood dust, are not exposed regularly to other chemicals: The mean exposure of cabinet-makers to formaldehyde was usually low (< 0.1 ppm [< 0.23 mg/m³]) in a Canadian study (Sass-Kortsak *et al.*, 1986). The mean level of solvents in finishing departments was about 20% of the national exposure limit of a mixture in Danish measurements in the early 1990s (Vinzens & Laursen, 1993) and about 40% in Finnish measurements in 1975–84 (Priha *et al.*, 1986). The solvents used are typically mixtures of several chemicals, such as aliphatic hydrocarbons (solvent naphtha, white spirit [see IARC, 1989c]), aromatic hydrocarbons (toluene [see IARC, 1989d], xylene [see IARC, 1989e]; less often benzene [see IARC, 1987e] and styrene [see IARC, 1994a]), esters, alcohols, ketones and glycol ethers (Partanen *et al.*, 1993). The constituents of solvent mixtures can vary, e.g. by country, period and facility. Pigments and other agents that may be used in furniture factories have been listed elsewhere (IARC, 1981).

1.4.6 Exposure during the manufacture of other wood products

The concentrations of wood dust measured during the manufacture of wooden doors, windows, prefabricated buildings, boats and other wood products are presented in Table 11. Measurements made during unspecified woodworking, which may be related to production of furniture or other wood products, are also included in the Table. The mean concentrations are similar to those in furniture manufacture because largely similar machining operations are used. The highest exposures occur in wood machining operations, where the mean levels are usually 1–10 mg/m³.

No studies on changes over time in the levels of wood dust in these industries were available to the Working Group, but in some countries levels may have declined during the last few decades, as in the furniture industry (see section 1.4.5). The main reason is use of local exhaust systems for woodworking machines. Comparative measurements made in woodworking shops in Germany with the exhaust on and off indicate that the concentration of wood dust is very high when the exhaust system is out of operation (see Table 11; Wolf *et al.*, 1986).

As in the furniture industry, many species of softwoods and hardwoods are used. For example, in the study in Germany mentioned above, mainly oak and beech were used but many workers had also been exposed to pine, spruce and other species (Wolf *et al.*, 1986). Coniferous wood species are frequently used in the manufacture of window frames, doors and prefabricated buildings, and about 90% of all wood used in this way in Finland in 1986 was pine or spruce (Welling & Kallas, 1991).

Other agents that occur in workroom air depend on the products and processing methods used; they may include surface coatings (solvents, resins, pigments), glues (formaldehyde, phenol, epoxy compounds, polyurethanes) and engine exhaust, and the levels may be comparable to those found in the furniture industry (see section 1.4.5). Wood is usually coated and treated away from dust-generating operations, because dust may interfere with the application of chemicals; however, solvents, formaldehyde and other vapours may spread to areas where wood processing operations are being performed, and exposure may also occur through dermal contact from handling treated wood, through the release of chemicals into the air when treated wood is heated during wood processing operations, or through inhalation of dust from treated wood.

Table 11. Concentrations of wood dust in other wood product industries

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Woodworking shops (Germany)				NR	Wolf <i>et al.</i> (1986)
Sawing with exhaust	91	[5.9]	0.2–47		
Sawing without exhaust	22	[34.4]	1.5–184		
Moulding with exhaust	64	[5.6]	0.1–60		
Moulding without exhaust	12	[17.3]	1.2–113		
Sanding with exhaust	69	[8.3]	0.3–55		
Sanding without exhaust	13	[56.7]	3.7–500		
Assembly with exhaust	6	[5.2]	1.0–11		
Assembly without exhaust	19	[9.3]	0.7–40		
Woodworking shops (Germany)				1987–88	Albracht <i>et al.</i> (1989)
Sanding	84	3.6 ^b			
Sawing	88	2.4 ^b			
Moulding	38	1.0 ^b			
Planing	27	1.1 ^b			
All-round woodworkers	42	2.0 ^b			
Woodworking shops (Germany)				NR	Scheidt <i>et al.</i> (1989)
Sawing, routing, sanding	6	5.1 ^b	2.9–6.6		
Woodworking (USA)				1987–88	Clayton Environmental Consultants (1988)
Saw operators	191	0.8 ^c	< 0.1–240		
Sander operators	85	1.2 ^c	0.1–41		
Milling machine operators	111	1.2 ^c	0.1–250		
Woodworking machine shops (United Kingdom)				NR	Hamill <i>et al.</i> (1991)
Hard- and softwood processing	7		0.5–5.1		
Softwood processing	37		0.3–55		
Hard- and softwood processing	51		0.5–33		
Woodworking factories (Denmark)	153	0.9 ^d	0.4–1.3 ^e	NR	Vinzents & Laursen (1993)
Joinery workshops (France)	6	22	2.4–73	NR	IARC (1981)

Table 11 (contd)

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Joinery shops (Sweden)				NR	Nygren <i>et al.</i> (1992)
Circular sawing	13	0.5 ^c			
Sanding	15	1.2 ^c			
Cutting	20	0.3 ^c			
Manufacture of doors and windows (Finland)				1980–85	Welling & Kallas (1991)
Machine sanding	5	3.4	1.4–6.7		
Packaging	2	1.5	1.4–1.5		
Sawing	6	2.0	1.2–3.3		
Spindle moulding	2	1.4	1.3–1.4		
Manufacture of doors and windows (Denmark)	118	0.6 ^d	0.6–0.8 ^e	NR	Vinzents & Laursen (1993)
Manufacture of prefabricated buildings (Canada)	8		0.4–2.5	1985	Holliday <i>et al.</i> (1986)
Manufacture of prefabricated houses (Finland)				1980–85	Welling & Kallas (1991)
Sawing	5	1.8	0.6–4.6		
Spindle moulding	2	2.9	0.6–5.1		
Manufacture of signs and plaques (USA)				1983	McCawley, M. (1983; cited in United States National Institute for Occupational Safety and Health, 1987)
Router, sander	18	3.2	1.0–8.1		
Wood component fabrication (USA)				1975	Kominsky & Anstadt (1976)
Router/groover operator	5	21.8	1.4–51.0		
Saw operator (total particulate)	19	68.8	0.7–688		

Table 11 (contd)

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Boat building (USA) Carpenters in assembly	27	2.4	0.3–16.2	1983	Crandall, M.S. & Hartle, R.W. (1984; cited in United States National Institute for Occupational Safety and Health, 1987)
Manufacture and repair of wooden boats (Finland)	4	1.2	0.8–1.8	1980–85	
					Welling & Kallas (1991)

NR, not reported

^a Arithmetic mean unless otherwise specified; time-weighted average personal and/or area samples

^b Median

^c Geometric mean

^d Mean of geometric means

^e Range of geometric means

Potential exposure to pesticides is high in the building of wooden boats because the wood must be protected from decay and marine borers (Jagels, 1985). Manufacture of windows, garden furniture, balcony decks, railroad ties, piers and other wooden structures for outdoor use may entail exposure to wood preservatives, such as chlorophenols, creosote, chromated copper arsenate and ammoniacal copper arsenate. The concentration of arsenic around various types of joinery machines was 0.5–3.1 $\mu\text{g}/\text{m}^3$ in six Swedish joinery shops using wood impregnated with copper–chromium–arsenic salt. The concentration of chromium was 0.4–2.3 $\mu\text{g}/\text{m}^3$ and that of copper was 0.4–1.9 $\mu\text{g}/\text{m}^3$. No hexavalent chromium was found (Nygren *et al.*, 1992). Exposure to low levels of arsenic has also been reported in factories where wood is impregnated with arsenic-containing preservatives (Rosenberg *et al.*, 1980). Insulation materials used in the manufacture of prefabricated houses often contain man-made mineral fibre products, such as glasswool and stonewool (Rockwool[®]) (see IARC, 1988). A number of other chemicals may be used as additives, including inorganic salts as fire retardants and chlorophenates as preservatives (Suchsland & Woodson, 1986).

1.4.7 Exposures in other wood-related occupations

Table 12 summarizes the concentrations of wood dust in some other wood-related occupations and operations, including flooring and parquet laying, pattern and model making, wood handling in pulp mills and teaching art and vocational skills.

The level of exposure in wooden model making in the automotive industry and in metal foundries averaged about 1 mg/m^3 in a study in the United States (McCammon *et al.*, 1985). Model makers use a wide variety of woodworking machines and hand tools in preparing models. Prototypes are made of softwoods, such as pine, bass, jelutong, plywood lavan and mahogany. Mahogany has been used for die models, but cativo wood impregnated with phenol–formaldehyde resin is now commonly used. Paints, sealers and lacquers that release various solvents are used to coat models. Model-making may also involve use of adhesive systems, such as white glues and epoxy resins, and plastics like carvable putties, fibre glass and poly-foams. Model-making also requires the use of glues that contain epoxy compounds and amines. The highest solvent concentration measured in the United States was 10% of the exposure limit of the mixture; no formaldehyde or amines were detected in air (McCammon *et al.*, 1985).

Some building trades entail exposure to wood dust. Sanding of parquet before varnishing is a dusty operation, which is usually carried out by specialized workers. Varnishes applied to parquets and wooden floors often contain formaldehyde-based resins and organic solvents, and the level of exposure to formaldehyde during varnishing may be over 1 ppm (1.23 mg/m^3). Construction carpenters use handsaws and circular saws both indoors and outdoors; however, since no measurements of exposure to wood dust were available to the Working Group, the mean level is probably low. Construction carpenters may also be exposed occasionally to other agents in the wide variety of activities carried out at construction sites. Most tasks on a construction site are performed by specialized workers, and it is unlikely that carpenters would be involved in e.g. painting, but different trades often work side-by-side, resulting in potential cross-exposure to e.g. other dusts and insulation materials. In addition, small construction

Table 12. Concentrations of wood dust in other wood-related occupations

Industry and operation (country)	No. of measurements	Mean ^a (mg/m ³)	Range (mg/m ³)	Year	Reference
Wooden model making (USA)					
Research and safety model shop	10	0.9	0.2–3.4	1980	Enright, J.C. (1980; cited in United States National Institute for Occupational Safety and Health, 1987)
Wood mill	10	4.7	1.2–10.2		
Pattern making (Canada)	5		1.0–2.6	1985	Holliday <i>et al.</i> (1986)
Automotive wood model shop (USA)				NR	McCammon <i>et al.</i> (1985)
Model makers/hardwood	12	0.6	0.2–0.3		
Model makers/soft- and hardwood	23	0.8	0.2–8.3		
Model makers/softwood	4	0.3	0.2–0.5		
Multi-axis machine operators	4	0.5	0.2–1.0		
Sweepers	5	1.6	0.1–6.1		
Shapers	7	2.7	0.3–13.9		
Wood mill (general)	10	0.3	0.05–0.5		
Parquet sanding (Germany)	5	6.6 ^b		1987–88	Albracht <i>et al.</i> (1989)
Flooring/hard- and softwood (Canada)	7		0.3–1.7	1985	Holliday <i>et al.</i> (1986)
Parquet sanding (Germany)	2	9.3	4.4–14	NR	Scheidt <i>et al.</i> (1989)
Pulp/paper mill (USA)				1987–88	Clayton Environmental Consultants (1988)
Chipping, debarking, screening, loading	19	0.3 ^c	< 0.1–18		
Art school (USA)					
Sawing, sanding, planing	8	6.0	0.9–24.2	1976	Levy, B.S.B. (1976; cited in United States National Institute for Occupational Safety and Health, 1987)
University art department (USA)	4	3.5	1.6–5.7	1980	Lucas & Salisbury (1992)

^a Arithmetic mean unless otherwise specified; time-weighted average personal and/or area samples

^b Median

^c Geometric mean

companies and those using non-union labour may not make clear distinctions between the responsibilities of different trades.

Pulp making and some papermaking processes start with retrieval of logs from storage, debarking and then chipping, and workers handling wood are exposed to wood dust, although the level of exposure is usually below 1 mg/m^3 (Table 12). High levels of fungal spores and bacteria have been found occasionally at wood and chip handling sites of pulp and paper mills (Kotimaa, 1990). Teachers and other personnel working in vocational and art schools may also have occupational exposure to wood dust (Table 12). Exposure may be high, owing to poor ventilation, but it is generally not continuous.

Forestry workers are a large occupational group who process and handle wood regularly. Lumberjacks have cut trees for centuries, first with axes and handsaws and, since about 1950, with chain saws. No measurements were available of their level of exposure to wood dust, but it is probably lower than those usually found in wood industries. Other exposures of forestry workers include engine exhaust (see IARC, 1989f) from chain saws and forest vehicles, chain oils and gasoline (see IARC, 1989g) used as fuel for chain saws.

1.4.8 Particle size distribution of wood dust in workroom air

Exposures to wood dust can be characterized not only by the mass (or number of particles) per unit volume of air but also by the distribution of particle sizes. Wood dust particles are typically irregular in shape and have rough surfaces, as observed by scanning electron microscopy; however, no differences in morphological pattern have been noted among samples from different operations (Liu *et al.*, 1985).

Several investigators have reported particle size distributions for wood dust in workplace air in various industries. Representative studies are summarized in Tables 13 and 14. In most studies, the major portion of the wood dust mass is contributed by particles larger than $10 \mu\text{m}$ in aerodynamic diameter (Whitehead *et al.*, 1981a; Darcy, 1984; Lehmann & Fröhlich, 1987, 1988; Hinds, 1988; Pisaniello *et al.*, 1991). This is attributable, in part, to the fact that larger particles are also heavier. Holliday *et al.* (1986) used an optical microscopy method (see section 1.3.1) to count particles in various size ranges and found that 61–65% (as calculated by the United States National Institute for Occupational Safety and Health, 1987) of the particles measured $1\text{--}5 \mu\text{m}$.

Some investigators have reported that the particle size distribution varies substantially according to woodworking operation, sanding producing more small particles and sawing producing more large particles (Hounam & Williams, 1974; Darcy, 1984; Liu *et al.*, 1985). Other investigators, however, have found no consistent differences (Holliday *et al.*, 1986; Lehmann & Fröhlich, 1988; Pisaniello *et al.*, 1991).

There is also some evidence that processing (especially sanding) of hardwoods can generate a higher percentage of small particulates than processing of softwoods, although again the evidence is by no means consistent and other studies have shown no differences. Whitehead *et al.* (1981a) suggested that processing of hardwoods may lead to higher concentrations of respirable dust than processing of softwoods, on the basis of a comparison of 15 samples taken

Table 13. Particle size distribution of hardwood dust (%)

Wood, operation	Total dust (mg/m ³)	Stage ^a								
		0	1	2	3	4	5	6	7	Back-up filter
Oak, hand sanding	6.9	72.6	9.6	5.1	3.3	2.3	1.7	1.7	1.5	2.2
Oak, machine sanding	2.7	65.0	12.2	3.9	4.2	3.0	3.4	3.0	2.1	3.3
Oak, sanding (hand portable machine)	2.7	47.2	14.6	7.2	9.1	7.0	4.8	2.6	2.3	5.2
Oak and beech, sawing and machine sanding	5.4	44.4	21.9	7.0	7.2	2.9	2.4	1.1	2.4	10.7
Particle-board and beech, sawing and planing	9.4	65.1	15.9	6.1	8.2	2.8	0.9	0.5	0.5	0.0
Ash, hand sanding	1.9	49.5	16.7	14.3	10.1	4.3	2.2	1.2	0.0	1.7
Beech, sawing	4.1	62.7	12.7	9.5	3.5	2.9	2.8	2.4	1.9	1.6

From Lehmann & Fröhlich (1988)

^a Stage 0, > 9.0 mm; stage 1, 5.8–9.0; stage 2, 4.7–5.8; stage 3, 3.3–4.7; stage 4, 2.1–3.3; stage 5, 1.1–2.1; stage 6, 0.65–1.1; stage 7, 0.43–0.65

Table 14. Wood dust sizes measured in the workplace in various studies

Study description	Equipment/operation	Sampling device	Mass median aerodynamic diameter (mm)	Reference
Cabinet-making (Czechoslovakia), 1 plant, 1961–62; area samples/total dust	Belt sander	NR	Up to 95%, < 5 Most, 2–3	Kubiš (1963)
Furniture (England), 5 plants, 1973; personal samples/total dust	Band sawing, turning	Four-stage cascade centripeter	11.5	Hounam & Williams (1974)
	Planing		9.2	
	Routing, moulding		10.0	
	Sanding		8.4	
	Assembly		7.6	
			(< 25%, < 5)	
Furniture (Denmark), 8 plants, 1974–75; personal samples/total dust	Sanding, drilling, planing, sawing	NR	33% (mass), < 5 41% (mass), 6–10 11% (mass), 11–15 15% (mass), > 16	Andersen <i>et al.</i> (1977)
Wooden component fabrication (USA), 1 plant, 1975; personal samples	Saw operator, router/groover operator	Six-stage cascade impactor	> 10	Kominsky & Anstadt (1976)
Wooden products (USA), 2 plants, 1976; personal samples	Shake mill (western red cedar)	Cyclone unit	39%, < 10 23%, 10–20 38%, > 20	Edwards <i>et al.</i> (1978)
	New planer mill (Douglas fir/hemlock)	Cyclone unit	47%, < 10 25%, 10–20 28%, > 20	
Plywood/furniture (USA), 12 plants, 1978; area samples/total dust	Veneer lathe/clipper, dryer, dry veneer handling, edge sawing/sanding, machining, assembly, milling, sanding	Six-stage cascade impactor	[1.3] mg/m ³ < 5.5 ^a [3.3] mg/m ³ < 14.1 ^a	Whitehead <i>et al.</i> (1981a)

Table 14 contd)

Study description	Equipment/operation	Sampling device	Mass median aerodynamic diameter (mm)	Reference
Furniture (England), 2 plants, 1981; personal samples/total dust	Sawmill	Seven-stage cascade impactor	17.3	Al Zuhair <i>et al.</i> (1981)
	Assembly		18.0	
	Machine floor		9.3	
	Cabinet shop		12.5	
Wooden model making (USA), 3 shops, 1981-82; personal samples/total dust	Model maker, sweeper, shaper operator, plastic shop worker, multi-axis machine operator	Nine-stage cascade impactor	7.7 (range, 5.2-10); 18-61% respirable dust ^b	McCammon <i>et al.</i> (1985)
Furniture (England), 7 plants, 1983; personal samples/total dust	Machine sanding, hand sanding, sawing, other cutting	Impactor	9 (54% (mass), 4-10)	Jones & Smith (1986)
Signs/plaques (USA), 1 shop, 1983; area samples/ total dust	Router, sander	Four-stage cascade impactor	46-60% (mass), < 3.5 30-35% (mass), 3.5-20 5-20% (mass), > 20	McCawley, M. (1983; cited in United States National Institute for Occupational Safety and Health, 1987)
Woodworking (Finland), 1 shop, 1983; personal samples/total dust	Unloading wood, sawing, other machines, planing	Optical microscopy	97.8%, < 5	Lindroos (1983)
Plywood (Finland), 6 mills, 1984; personal and area samples/total dust	Sawing Finishing General workroom	MSA cyclone	40% respirable dust 29% respirable dust 65% respirable dust	Kauppinen <i>et al.</i> (1984)
Particle-board (USA), 1 plant, 1986; area samples/ total dust	Sanding	Six-stage cascade impactor	8.26	Stumpf <i>et al.</i> (1986)
Various industries (Canada), 23 plants, 1985; personal samples/total dust	Sawing	Optical microscopy	62%, 1-5	Holliday <i>et al.</i> (1986)
	Sanding		61%, 1-5	
	Planing/routing/shaping		65%, 1-5	

Table 14 contd)

Study description	Equipment/operation	Sampling device	Mass median aerodynamic diameter (mm)	Reference
Various industries (Germany), 17 factories, 2 training shops, 1983-85; personal and area samples	Sanding, sawing, routing/planing	Anderson impactor	44.4-72.6%, > 9	Lehmann & Fröhlich (1987)
			9.6-21.9%, 5.8-9	
			3.9-14.3%, 4.7-5.8	
			3.3-10.1%, 3.3-4.7	
			2.3-7.0%, 2.1-3.3	
			0.9-4.8%, 1.1-2.1	
			0.5-3.0%, 0.7-1.1	
Various industries (Hong Kong), 3 factories; personal samples	Sawmill, sanding (furniture factory), mixing (mosquito-coil factory)	Scanning electron microscopy	9-12.8%, > 10	Liu <i>et al.</i> (1985)
			18-26.1%, 5-10	
			61.7-73%, 0-5	
Furniture (Australia), 15 factories, 1989; personal samples	Sanding Sawing Mixed woodworking	IOM/7-hole; cascade impactor	16-19	Pisaniello <i>et al.</i> (1991)
			17-22	
			15-23	

NR, not reported; MSA, Mine Safety Appliances Co. (Pittsburgh, PA); IOM, Institute of Occupational Medicine

^a Gravimetric concentrations are given, rather than percentages. Of the 15 samples reported, only two contained dust of a mass median aerodynamic diameter < 5.5 µm at concentrations ≥ 1 mg/m³, and only three contained dust < 14.1 µm at > 2 mg/m³.

^b Scanning electron microscopy indicated that length-to-width ratios were 2.3:1 and 1.9:1 in two air samples collected with a 0.5-in [1.3-cm] stainless-steel cyclone.

during furniture and plywood manufacture. Darcy (1982), however, found that the distribution of particle sizes from sanding pine and oak were very similar (see distribution curves reproduced by Hinds, 1988). Pisaniello *et al.* (1991) reported only a very slight difference in the average mass median aerodynamic diameter of dust from hardwood (18.7 μm ; geometric standard deviation (GSD), 2.0) and from softwood/reconstituted wood (19.6 μm ; GSD, 2.1).

1.5 Regulations and guidelines

Several countries have set standards or guidelines for occupational exposures to wood dust, with 8-h time-weighted average (TWA) exposure limits ranging from 1 to 10 mg/m^3 (United States National Institute for Occupational Safety and Health, 1987, 1992). In the regulations of some countries, a particular class of wood dust is named (e.g. 'hardwood' and 'softwood'). For example, in the United Kingdom, the long-term exposure limit (8-h TWA) for hardwood and softwood is 5 mg/m^3 ; for hardwood, there is a notation that the substance can cause respiratory sensitization, and the limit for softwood is noted for intended change (United Kingdom Health and Safety Executive, 1992). In Canada, the limits are 1 mg/m^3 for hardwood and 5 mg/m^3 for softwood (United States National Institute for Occupational Safety and Health, 1987). In Germany, dusts of oak and beech have been classified as human carcinogens (group III A1) since 1985, and other wood species are suspected of having carcinogenic potential (group III B). The technical exposure limit for total wood dust was set at 2 mg/m^3 for all industrial plants in 1993. The limit of 5 mg/m^3 set up for old industrial plants in 1987 will no longer be allowed, with a few exceptions, by 31 December 1995 (Deutsche Forschungsgemeinschaft, 1993). In Sweden also, wood dust is considered potentially carcinogenic (United States Occupational Safety and Health Administration, 1987).

In other countries, wood dust is regulated under more general categories of particulate matter. In Hungary and Poland, for example, dust of vegetable and animal origin is regulated; dust containing various percentages of free silica are regulated in Poland (United States Occupational Safety and Health Administration, 1987). Standards for organic dusts are used for wood dust in Finland (8-h threshold limit value [TLV], 5 mg/m^3 ; maximum for 15 min, 10 mg/m^3) (Työministeriö, 1993). Switzerland has no specific standards for wood dust but controls 'total dust' and 'fine dust' (United States National Institute for Occupational Safety and Health, 1987).

The American Conference of Governmental Industrial Hygienists (1993) recommended the following TLVs: 1 mg/m^3 for an 8-h TWA for wood dust (certain hardwoods such as beech and oak); and 5 mg/m^3 TWA and 10 mg/m^3 for the short-term exposure limit to softwoods, with the notation that the substance has been identified elsewhere as a suspected human carcinogen. Similar exposure limits have been adopted by several other countries (e.g. Australia, New Zealand and Norway) as regulations or guidelines (United States National Institute for Occupational Safety and Health, 1987). 'Particulates not otherwise regulated' are covered in the United States (United States Occupational Safety and Health Administration, 1993).

2. Studies of Cancer in Humans

2.1 Case reports

Many cases of cancer of the sinonasal cavities and paranasal sinuses (referred to below as 'sinonasal cancer') have been reported among woodworkers. The earliest reports of cases of cancer of the upper respiratory tract in association with woodworking were published in Germany (reviewed by Schroeder, 1989).

Macbeth (1965) reported 20 patients (17 men) from High Wycombe, United Kingdom, presenting with a malignant disease of the paranasal sinuses; 15 of the cases, all in men, were associated with the making of wooden chairs. Macbeth noted later that the tumours were all adenocarcinomas (Acheson, 1976). Several furniture factories are located in High Wycombe which specialize in chair-making from a variety of domestic and imported hardwoods. The cross-sectional prevalence of woodworkers in the local male population at the time of the study was 23.5%.

Following these observations, a survey was carried out in the Oxford area, including High Wycombe, of 148 cases (98 males) of nasal cancer diagnosed in 1951–65 (Acheson *et al.*, 1968; Acheson, 1976). Cases were classified according to sex and histological type of cancer. The results for men indicated a strong relationship between adenocarcinoma and present or past work in the furniture industry. Of the 33 cases of adenocarcinoma in men, 24 (73%) were in woodworkers, 22 of whom (67%) worked in furniture manufacture. Among the 65 remaining male cases of nasal tumour, the corresponding numbers were five (8%) and three (5%). For the subgroup of men who were employed at the onset of their illness, which was diagnosed in 1956 or later, a detailed occupational history was obtained and was compared with the findings of the 1961 census. The estimated rate for adenocarcinoma in cabinet- and chair makers and wood machinists in High Wycombe was similar, namely 0.7 ± 0.2 per 1000 men per annum during the decade 1956–65, which was at least 500 times the rate in adult males in southern England. The risk was extended to workers making products other than chairs. The results suggested that carpenters and joiners in High Wycombe had no increase in risk. The species of wood used by the 16 patients for whom information was available before the Second World War were oak (14/16), beech (11/16) and mahogany (13/16). Walnut was also frequently used.

Acheson *et al.* (1972) performed a survey of nasal adenocarcinoma in England excluding the Oxford area. Cases of nasal adenocarcinoma were collected from cancer registries (for the period 1961–66 for most registries) and were compared with cases of nasal cancer other than adenocarcinomas. The study comprised 107 cases of adenocarcinoma (80 male) and 110 cases of nasal cancer other than adenocarcinoma (85 male) when restricted to cases 'accepted' mainly on the basis of confirmation of the histological classification. Thirty-three men (41%) and one woman (3.7%) with nasal adenocarcinoma had at some time worked as woodworkers; of these, 24 (73%) men had worked in the furniture industry. The ratio of observed cases:expected cases was 95 for furniture workers and 5 for other woodworkers (principally, carpenters and joiners) on the basis of the distribution at the 1961 census. Among nasal cancer cases other than adenocarcinoma, a significant excess in woodworkers was also observed. The main types of

wood dusts were known for some of the woodworkers (with both adenocarcinoma and other histological types): most were exposed to more than one species. The species most often indicated in the furniture industry were oak (eight adenocarcinoma patients), mahogany (six adenocarcinoma patients) and beech, birch and walnut (four adenocarcinoma patients for each of the species). Four patients had used mainly softwoods; three of those with adenocarcinomas were joiners or joiners and carpenters, and the last had worked as a packing case maker and had a squamous-cell carcinoma.

After the results from the United Kingdom were published, cases were reported from many countries, including Belgium (Debois, 1969), the Netherlands (Delemarre & Themans, 1971), Denmark (Andersen, 1975; Andersen *et al.*, 1976, 1977), France (Trotel, 1976), Australia (Ironsides & Matthews, 1975), western (Gülzow, 1975; Kleinsasser & Schroeder, 1989) and eastern Germany (Löbe & Ehrhardt, 1983; Wolf *et al.*, 1986), Sweden (Engzell *et al.*, 1978; Klintonberg *et al.*, 1984), Austria (Smetana & Horak, 1983), Norway (Voss *et al.*, 1985), Switzerland (Rüttner & Makek, 1985) and Spain (López *et al.*, 1990). Subsequently, many more case reports were published in different countries (for an extensive review, see for instance Mohtashamipur *et al.*, 1989a). A systematic analysis of these studies is not included, as many analytical studies were available.

The studies that presented data on occupational exposures to specific species of wood are summarized in Table 15.

2.2 Descriptive studies

The studies placed under this heading were mainly designed for generating hypotheses, especially by use of record linkage with routinely collected data.

A number of descriptive studies (Table 16) have dealt with cancer mortality or incidence among woodworkers, defined on the basis of occupational title and/or industrial branch reported on death certificates (Menck & Henderson, 1976; Milham, 1976; Gallagher *et al.*, 1985), in hospital files (Grufferman *et al.*, 1976; Menck & Henderson, 1976; Bross *et al.*, 1978), in cancer registries (Acheson, 1967; Nandakumar *et al.*, 1988) or in the records of pension funds (Olsen & Jensen, 1987; Olsen *et al.*, 1988), in union files (Milham, 1978) or declared at censuses (Pukkala *et al.*, 1983; Gerhardsson *et al.*, 1985; Pearce & Howard, 1986; Linet *et al.*, 1988, 1993). None of these studies provides quantitative or semi-quantitative information on exposure to wood dust.

Some studies based on occupational titles specifically address nasal cancer in woodworkers (Table 17), both in terms of mortality (Minder & Vader, 1987) and morbidity (Malker *et al.*, 1986; Vetrugno & Comba, 1987; Olsen, 1988). These studies corroborate the well-established indication of an increased risk for nasal cancer in woodworkers. The same observation applies to incidence studies of nasal cancer in which information on occupation was provided by questionnaires and/or interviews with patients (Ghezzi *et al.*, 1983; Petronio *et al.*, 1983).

Table 15. Case reports of sinonasal cancer according to occupation and type of wood

Study, year, country	Sex	Origin	Histological type	Exposed cases/ total cases	Occupations	Main types of wood
Acheson <i>et al.</i> (1968); Acheson (1976); Oxfordshire, United Kingdom	M	Registry	Adenocarcinoma	24/33	22 in the furniture industry (mainly wood machinists and furniture makers)	Oak, beech, mahogany, walnut
Acheson <i>et al.</i> (1972), United Kingdom	M	Registries	Adenocarcinoma	33/80	24 in the furniture industry (mainly cabinet- and chair makers, wood machinists and turners) 6 not in the furniture industry (mainly joiners and carpenters)	Oak, mahogany, beech, birch, walnut Softwoods in 3 cases
Leroux-Robert (1974), France	M	Hospital	Adenocarcinoma, ethmoid sinus	26/92	Not reported	All patients had used European hardwoods, some exclusively, and mainly oak (22/26), exclusively oak in one case
Luboiniski & Marandas (1975), Paris, France	M	Hospital	Adenocarcinoma, ethmoid sinus	21/43	8 joiners, 4 joiners and cabinet-makers, 7 cabinet- makers, 1 cooper, 1 coffin maker	European hardwoods (oak, chestnut, wild cherry, walnut, beech, poplar); tropical species, including mahogany
Ironside & Matthews (1975), Victoria, Australia	M	Hospital	Adenocarcinoma	10/18	3 carpenters, 2 woodturners/ wood machinists, 2 builders, 1 sawmill owner, 1 timber worker, 1 joiner	Native timber

Table 15 (contd)

Study, year, country	Sex	Origin	Histological type	Exposed cases/ total cases	Occupations	Main types of wood
Andersen <i>et al.</i> (1976, 1977), Aarhus, Denmark	MF	Hospital	Adenocarcinoma	12/17	10 cabinet- and chair makers, 1 turner, 1 coach builder	Several kinds of wood used by each cabinet- and chair maker; primarily beech, oak, walnut; periodically mahogany and teak
	MF	Hospital	Squamous-cell carcinoma	5/71	1 carpenter, 1 turner, 1 sawyer, 1 forestry worker, 1 brushmaker	
Engzell <i>et al.</i> (1978), Sweden	M	Registry	Adenocarcinoma	19/36	19 joiners or cabinet-makers (at least 12 cabinet-makers)	Hardwoods such as oak, teak, mahogany and birch; never exclusively softwood
Voss <i>et al.</i> (1985), Norway	M	Hospital	Adenocarcinoma	1/2	Cabinet-maker	Birch, pine, spruce, teak, mahogany
	M	Hospital	Squamous-cell carcinoma and undifferentiated carcinoma	8/30	3 joiners/carpenters, 3 loggers, 1 sawmill/carpenter, 1 cabinet-maker	Pine and spruce (7 cases), pine and lime (1 case)
Kleinsasser & Schroeder (1989), Germany	M	National recruitment	Adenocarcinoma, intestinal type	77/85	55 joiners and cabinet-makers, 11 wheelwrights, 6 coopers, 5 parquet floor layers, 5 carpenters (non-exclusive categories)	Oak, beech No case patient had handled softwood or exotic wood exclusively.

Table 16. Studies of mortality and morbidity in woodworkers

Reference	Method	Results
Milham & Hesser (1967)	Analysis of occupations reported on death certificates (New York State, USA, 1940–53 and 1957–64). Comparison with all other causes of death	Hodgkin's disease: significant association with woodworking (χ^2 , 14.59; $p < 0.001$)
Acheson (1967)	Analysis of occupations of patients in the Oxford area, United Kingdom, 1956–65	Hodgkin's disease: three cases among woodworkers versus four expected
Grufferman <i>et al.</i> (1976)	Incidence by occupation in Boston, USA, 1959–73. Reference rates: whole Boston population	Hodgkin's disease: woodworkers: RR, 1.6 (95% CI, 0.9–2.6); 15 observed
Menck & Henderson (1976)	Mortality by occupation in Los Angeles County, USA, 1968–70. Reference rates: mortality in all occupations	Lung cancer: lumber, wood, furniture: SMR, 1.1 [0.7–1.8]; 20 observed
Milham (1976)	Proportionate analysis of mortality by occupation from death certificates, Washington State, USA, 1950–71	Plywood mill workers, stomach cancer: PMR, 1.5 [1.0–2.2]; 32 observed; leukaemia: PMR, 1.9 [1.2–2.9]; 23 observed Sawmill workers, cancer of the testis: PMR, 1.7 [0.9–2.7]; 15 observed Carpenters, stomach cancer: PMR, 1.3 [1.1–1.4]; 271 observed; Hodgkin's disease: PMR, 1.6 [1.1–2.2]; 38 observed
Bross <i>et al.</i> (1978)	Analysis of occupations of cancer patients attending Roswell Park Memorial Institute, USA, 1956–65. Comparison groups: noncancer patients	Oesophageal cancer: lumber workers: significantly increased risk
Milham (1978)	Analysis of mortality of woodworkers, USA, 1969–73. Reference rates from US population	Malignant neoplasm of stomach: SMR, 1.1 [1.0–1.2]; 407 observed
Pukkala <i>et al.</i> (1983)	Incidence by occupation declared at 1970 census in Finland, 1971–75. Reference rates from total economically active population	Lung cancer: woodworking: SIR, 1.3 [1.2–1.4]; 366 observed Joiners: SIR, 1.4 [1.3–1.6]; 264 observed
Gallagher <i>et al.</i> (1985)	Proportionate cancer mortality analysis of woodworkers in British Columbia, Canada, 1950–78	Loggers: nasal sinus: PCMR, 3.6 (1.2–8.5); 5 observed Woodworkers: stomach: PCMR, 1.3 (1.1–1.5); 116 observed; non-Hodgkin's lymphoma, PCMR, 1.4 (1.0–1.9); 42 observed

Table 16 (contd)

Reference	Method	Results
Pearce & Howard (1986)	Analysis of cancer mortality rates by occupation in New Zealand, 1974–78. Reference: mortality rates of all employed people	Large-bowel cancer in cabinet-makers and woodworkers: RR, 2.6 (1.3–4.6); 11 observed
Olsen & Jensen (1987)	Proportionate analysis of cancer incidence by occupation in Denmark, 1970–79	Among men: woodworking: stomach cancer: SPIR, 1.8 (1.3–2.5), 41 observed; breast cancer: SPIR, 3.9 (1.2–12) Among men: manufacture of wooden furniture: cancer of nasal cavities and sinuses: SPIR, 5.9 (2.5–14), 5 observed
Nandekumar <i>et al.</i> (1988)	Incidence by occupation in Western Australia, 1960–84. Reference rates: other occupations except farming	Multiple myeloma: woodworking: RR, 1.7 (0.78–3.9); 3 cases
Linnet <i>et al.</i> (1988)	Incidence by industry through record linkage between 1960 census and National Cancer Registry in Sweden, 1961–79. Reference: national incidence rates	Acute nonlymphocytic leukaemia: wood (men): SIR, 1.3 [1.0–1.7]; 67 observed
Linnet <i>et al.</i> (1993)	Incidence by industry through record linkage between 1960 census and National Cancer Registry in Sweden, 1961–79. Reference: national incidence rates	Non-Hodgkin's lymphoma: furniture and furnishings (men): SIR, 1.3 [1.0–1.7]; 55 observed

CI, confidence interval; RR, relative risk; SMR, standardized mortality ratio; PMR, proportionate mortality ratio; SIR, standardized incidence ratio; PCMR, proportionate cancer mortality ratio; SPIR, standardized proportionate incidence ratio

Table 17. Mortality and morbidity studies on nasal cancer in wood workers

Reference	Method	Results
Malker <i>et al.</i> (1986)	Incidence in Swedish subjects who reported their occupation as woodworker at 1960 census. Follow-up through 1979. Comparison with population.	Woodworkers (males) - All nasal cancers: SIR, 1.3 [0.8–1.9], 24 observed - Adenocarcinomas: SIR, 2.2 [0.8–4.8], 6 observed Furniture workers (males) - All nasal cancers: SIR, 4.1 [2.7–6.1], 25 observed - Adenocarcinoma: SIR, 17 [10–26], 19 observed
Minder & Vader (1987)	Mortality of Swiss subjects who reported their occupation as woodworker at 1980 census. Follow-up through 1985. Comparison with all workers.	SMR: 6.2 (3.6–10), 16 observed
Vetrugno & Comba (1987)	Analysis of the occupations reported by 189 cases diagnosed and/or treated in 1983–85 at 61 Italian ear-nose-and-throat clinics and hospital departments.	Among males, woodworkers account for 11% of the case series and 22% of adenocarcinoma cases.
Olsen (1988)	Analysis of employment histories reported by cases diagnosed in Denmark 1970–84.	Among males: wooden furniture production: SIR, 3.6 (1.3–8.0), 5 observed
Ghezzi <i>et al.</i> (1983)	Incidence among woodworkers in Brianza (Italy), 1976–80. Comparison with incidence in other occupations.	Rate ratio: 4.4 (1.8–9.1), 7 observed
Petronio <i>et al.</i> (1983)	Incidence in woodworkers in Trieste (Italy), 1968–80. Comparison with incidence in all occupations.	Woodworkers: incidence rate: 6.4×10^{-5} All occupations: incidence rate: 0.54×10^{-5}
Gerhardsson <i>et al.</i> (1985)	Record linkage between 1960 census and 1961–79 cancer registry in Sweden for morbidity from respiratory cancers in furniture workers. Reference rates: all other employed men.	Sinonasal carcinoma: SMR, 7.1; 90% CI, 4.4–11, 15 observed Sinonasal adenocarcinoma: SMR, 44; 90% CI, 27–69, 14 observed

CI, confidence interval; SMR, standardized mortality ratio; SIR, standardized incidence ratio

2.3 Cohort studies

The only cohort study that addressed the issue of exposure to wood dust was that conducted in High Wycombe, United Kingdom (Acheson *et al.*, 1984); the others assessed exposure by occupational title.

One cohort study was conducted in Finland of 1223 sawmill workers followed-up during 1945–80 (Jäppinen *et al.*, 1989). Cancer incidence was not in excess overall, and no cases of nasal cancer were found (0.3 expected). The only cancer for which an increased incidence was seen was non-melanocytic skin cancer (excluding basal-cell carcinoma), with six cases in men (standardized incidence ratio [SIR], 3.1; 95% confidence interval [CI], 1.2–6.8) and two cases in women [SIR, 1.8; 95% CI, 0.2–6.6]; however, four of the six male patients were first employed after 1945, when chlorophenols were used.

Four cohort studies of furniture workers are available, from the United Kingdom, Denmark, Germany and the United States. In the Danish study, 40 428 members of the carpenters' and cabinet-makers' union in 1971 were followed up to 1976 (Olsen & Sabroe, 1979). The overall mortality of both active and retired workers was below that expected; the only cancer for which increased mortality was seen was that of the nose and sinuses (standardized mortality ratio [SMR], 4.7; 95%CI, 2.5–6.8; four deaths).

In the study of 5108 furniture workers in High Wycombe, United Kingdom, followed through 1982, overall mortality and mortality from all cancers were below expectation; the only cancer site for which there was increased mortality was that of the nose and sinuses ([SMR, 8.2; 95%CI, 3.7–16] nine deaths) (Rang & Acheson, 1981; Acheson *et al.*, 1984). When workers were divided into three groups according to dustiness of workplace, all nasal cancer deaths were found in the group exposed to the most dust [SMR, 16; 95% CI, 7.2–30].

In a cohort of 759 cabinet-makers or joiners studied during 1973–84 in Germany, no cases of nasal cancer were found [expected number not reported]; the only cancer for which the incidence was increased was malignant melanoma (SIR, 9.5; 95%CI, 2.4–28; two cases) (Barthel & Dietrich, 1989).

A cohort of furniture makers in the United States, which included 34 801 subjects (of whom 12 158 were employed in wooden furniture facilities), was studied between 1946 and 1983 (Miller *et al.*, 1989, 1994). Overall mortality and mortality from all cancers were below expectation for the wooden furniture workers; the only neoplasm for which mortality was increased was myeloid leukaemia (SMR, 1.9; 95%CI, 1.0–3.5; 11 deaths); seven deaths were from acute leukaemia. A significant increase in mortality from chronic nephritis was also found (SMR, 2.5; 95% CI, 1.1–5.0; eight deaths). Two cohort members died from nasal cancer (2.5 expected); one case occurred in the cohort of wooden furniture workers [expected number not reported].

A cohort of 2283 plywood workers from four mills in Washington and Oregon, United States, was studied between 1945 and 1977 (Robinson *et al.*, 1990). Overall mortality and mortality from all cancers were below expectation, and no significantly increased mortality was seen. No deaths from nasal cancer were found (0.4 expected).

A total of 10 322 American Cancer Society volunteers enrolled in a large prospective study conducted between 1959 and 1972 reported wood-related occupations, and their mortality was compared with that of over 400 000 volunteers with other occupations (Stellman & Garfinkel, 1984). Overall mortality and mortality from all cancers were close to expectation; a significant increase in mortality was found from cancers of the stomach (SMR, 1.5; 44 deaths) and urinary bladder (SMR, 1.4; 29 deaths); non-significant increases in mortality were found for lung cancer (SMR, 1.1; 135 deaths) and nasal cancer (SMR, 2.0; two deaths); the two deaths from nasal cancer occurred among carpenters and joiners (SMR, 3.3).

Cohort studies on woodworkers are summarized in Table 18.

During the last decade, several papers raised the possibility that the risk for colorectal cancer was increased among wooden pattern and model makers exposed to wood dust in the automobile industry (Swanson & Belle, 1982; Swanson *et al.*, 1985; Tilley *et al.*, 1990; Becker *et al.*, 1992; Roscoe *et al.*, 1992). These studies are summarized in Table 19. Although various study designs were used, leading to different risk estimates (SMRs, proportionate mortality ratios [PMRs] and SIRs), the first three reported excess risks for colorectal cancer. The suggested association has been the object of some debate in the scientific literature (Chovil, 1982; Davies, 1983; Kurt, 1986). [The studies that gave positive results had several methodological problems, namely short duration of observation, high proportion of loss to follow-up and inadequate assessment of exposure; the study that was of more adequate design with respect to ascertainment of exposure and control of confounding (Roscoe *et al.*, 1992) did not reach positive conclusions.]

2.4 Case-control studies

Information on exposure to wood dust or employment in wood-related occupations has been reported in studies dealing with many cancer sites. The Working Group reviewed in particular case-control studies of organs in the respiratory, the digestive and the lymphohaematopoietic systems. The case-control studies are grouped according to whether exposure to wood dust was addressed specifically or whether the results are based on job titles or industrial branch. The term 'relative risk' is used to cover all estimated risk ratios, which are usually given as odds ratios.

2.4.1 Cancer of the nasal cavity and paranasal sinuses

(a) Exposure to wood dust

Hernberg *et al.* (1983) reported the results of a joint Danish-Finnish-Swedish case-control study of 167 patients with primary malignant tumours of the nasal cavity and paranasal sinuses diagnosed in Denmark, Finland and Sweden between July 1977 and December 1980. Ninety-five cases were epidermoid carcinoma, 18 were adenocarcinoma, 17 were anaplastic carcinoma and 37 were other histological types. Controls were 167 patients with tumours of the colon and rectum, who were matched to patients on country, sex and age at diagnosis. Cases and controls were identified through the national cancer registries of Finland and Sweden and four of the five oncological centres in Denmark. Subjects were interviewed extensively by telephone to

Table 18. Cohort studies of workers in wood-related industries

Industry	Reference	Methods	Results	Notes
Sawmill workers	Jäppinen <i>et al.</i> (1989)	1223 sawmill workers employed between 1945 and 1961; follow-up till 1980; cancer incidence in the cohort contrasted to local incidence rates. Lost to follow-up: 0.2%	<p>SIR: All cancers: men, 1.1 (0.9–1.3), 90 observed women, 1.2 (0.9–1.6), 55 observed Skin cancer: men, 3.1 (1.2–6.8), 6 observed women, [1.8; 0.2–6.6], 2 observed No other significantly increased SIR. No case of nasal cancer, 0.3 expected.</p> <p><i>Women</i> Lip, mouth, pharynx (1 observed/0.9 expected) Stomach, 1.1 (0.4–2.5), 5 observed Colon (2 observed/2.3 expected) Rectum, 2.3 (0.6–5.8), 4 observed Larynx (0 observed/0.1 expected) Lung, 3.3 (0.9–8.3), 4 observed Lymphoma (0 observed/0.9 expected) Hodgkin's disease (0 observed/0.2 expected) Leukaemia, 2.7 (0.6–8.0), 3 observed</p> <p><i>Men</i> Lip, mouth, pharynx, 1.8 (0.6–3.8), 6 observed Stomach, 0.8 (0.4–1.5), 11 observed Colon, 1.7 (0.6–4.1), 5 observed Rectum, 1.3 (0.4–3.3), 4 observed Larynx (2 observed/2.1 expected) Lung, 1.0 (0.6–1.4), 24 observed Lymphoma, 2.0 (0.6–5.2), 4 observed Hodgkin's disease (2 observed/0.8 expected) Leukaemia, 2.2 (0.6–5.5), 4 observed</p>	Sawn timber, mainly pine and spruce; dust levels in sawmills generally below 1 mg/m ³

Table 18 (contd)

Industry	Reference	Methods	Results	Notes
Furniture workers	Olsen & Sabroe (1979)	40 428 members of the Danish carpenters'/cabinet-makers' trade union active or retired in 1971; follow-up through 1976; mortality in the cohort contrasted with national mortality rates	SMR: All causes: Active workers, 0.82 (0.76–0.88), 692 observed Retired workers, 0.70 (0.67–0.74), 1483 observed Nasal cancer: All workers, 4.7 (2.5–6.8), 4 observed (3 cases in cabinet-makers, 1 in a carpenter); no other significantly increased SMR <i>Active</i> Intestine, 0.75 (0.29–1.2) Stomach, 1.1 (0.53–1.6) Lung, 0.96 (0.68–1.1) Leukaemia, 1.3 (0.55–2.0) <i>Retired</i> Intestine, 0.94 (0.65–1.2) Stomach, 0.84 (0.59–1.1) Lung, 1.1 (0.92–1.3) Leukaemia, 0.71 (0.29–1.1)	Type of wood not reported
Furniture workers	Rang & Acheson (1981); Acheson <i>et al.</i> (1984)	5108 workers born before 1940 and active before 1969 in at least one of nine furniture workshops in High Wycombe (United Kingdom) Categorization of exposures: - class I (less dusty): office workers, upholsterers and yardmen - class II (dusty): polishers, veneerers and maintenance men - class III (very dusty): cabinet- and chair makers, sanders and wood machinists Mortality studied for 1941–82; rates for England and Wales used as reference. Lost to follow-up: 1.2%	SMR: All causes: 0.68 (0.62–0.76), 1638 observed All cancers: 0.88 (0.80–0.97), 435 observed Nasal cancer: 8.1 (3.7–16), 9 observed; all cases in people with very dusty occupations (0.57 expected). No other significantly increased SMR or trend with level of exposure. Mouth, pharynx, 1.2 (0.45–2.7) Stomach, 1.2 (0.92–1.5) Colon, 0.68 (0.42–1.0) Rectum, 1.1 (0.7–1.7) Larynx, 0.58 (0.12–1.7) Lung, 0.80 (0.68–0.93) All lymphatic/haematopoietic, 0.92 (0.61–1.3)	Chairs traditionally made from beech; wide range of imported hardwood used in furniture (Acheson <i>et al.</i> , 1968)

Table 18 (contd)

Industry	Reference	Methods	Results	Notes
Furniture workers	Barthel & Dietrich (1989)	759 cabinet-makers or joiners from 170 enterprises located in Neubrandenburg district (Germany), followed from 1973 to 1984; cancer incidence in the cohort compared with incidence rates of the population of the district	SIR: All tumours, 1.1, 40 observed Malignant melanoma, 9.5 (2.4–28), 2 observed No other significantly increased SIR; no case of nasal cancer Stomach, 1.3, 7 observed Appendix, colon, sigmoid, 1.5, 3 observed Rectum, 2.1, 6 observed Lung, 0.68, 9 observed Lymphoma, 3.9, 1 observed Myeloma, 5.2, 1 observed	Species of wood most frequently worked with: pine, beech and oak
Furniture workers	Miller <i>et al.</i> (1989, 1994)	12 158 members of the United Furniture Workers of America first employed between 1946 and 1962 at factories producing wooden furniture. Mortality studied from 1946 to 1983; US rates used as reference. SMRs computed for subcohort followed for at least 20 years (10 497 subjects).	SMR: All causes, 0.9 (0.8–0.9), 1427 observed All malignant neoplasms, 0.9 (0.8–1.0), 342 observed Buccal cavity and pharynx, 0.7 (0.3–1.4), 7 observed Stomach, 1.0 (0.5–1.6), 14 observed Colon and rectum, 0.8 (0.6–1.1), 36 observed Nose, 1 observed Larynx, 0.6 (0.1–1.8), 3 observed Lung, 1.0 (0.8–1.1), 116 observed Hodgkin's disease, 2.2 (0.6–5.5), 4 observed Non-Hodgkin's lymphoma, 1.0 (0.5–1.9), 11 observed Multiple myeloma, 1.6 (0.7–3.1), 9 observed Leukaemia, 1.4 (0.8–2.2), 17 observed	

Table 18 (contd)

Industry	Reference	Methods	Results	Notes
Plywood workers	Robinson <i>et al.</i> (1990)	2283 white males who worked for at least one year between 1945 and 1955 in any of four mills located in Washington and Oregon (USA); mortality studied through March 1977; US rates used as reference; 2% lost to follow-up	SMR: All causes, 0.7 [0.7–0.8], 570 observed All malignant neoplasms, 0.7 [0.6–0.9], 100 observed Lymphatic/haematopoietic, 1.6 [0.8–2.7], 12 observed Lymphosarcoma and reticulosarcoma, 1.0 [0.3–2.6], 4 observed Hodgkin's disease, 1.1 [0.1–4.0], 2 observed Multiple myeloma, 3.3 [0.7–9.7], 3 observed Other lymphatic, 2.7 [0.6–8.0], 3 observed Leukaemia, 0.9 [0.3–2.0], 5 observed Buccal cavity and pharynx, 0.6 [0.1–1.9], 3 observed Stomach, 0.4 [0.1–1.1], 4 observed Intestine, 0.6 [0.3–1.2], 8 observed Nose, 0 observed, 0.4 expected Larynx, 0.5 [0.01–2.5], 1 observed Lung, 0.8 [0.5–1.1], 33 observed	Plywood manufactured from softwood (mainly Douglas fir, but also cedar, pine, spruce, hemlock, larch, true firs and redwood)
Woodworkers	Stellman & Garfinkel (1984)	10 322 volunteers enrolled in the American Cancer Society prospective study, whose occupation was in a wood-related industry, followed 1959–72; mortality compared with that of over 400 000 non-woodworkers in the study	SMR: All causes, 0.98, 2503 observed All cancers, 1.0, 513 observed Stomach, 1.5, $p < 0.01$, 44 observed Urinary bladder, 1.4, $p < 0.05$, 29 observed Lung, 1.1, $p > 0.05$, 135 observed Nasal cavity, 2.0, $p > 0.05$, 2 observed Colon and rectum, 0.75, $p < 0.05$, 57 observed Larynx, 0.68, $p > 0.05$, 3 observed Leukaemia, 1.3, $p > 0.05$, 32 observed Hodgkin's disease, 0.67, $p > 0.05$, 3 observed Other lymphatic, 0.70, $p > 0.05$, 17 observed	Stomach: among carpenters and joiners, SMR 1.7, $p < 0.01$, 36 observed Lung: among carpenters and joiners, SMR 1.2, $p < 0.05$, 101 observed Nasal cavity: both cases occurred among carpenters and joiners (SMR, 3.3)

SIR, standardized incidence ratio; CI, confidence interval; SMR, standardized mortality ratio

Table 19. Cohort studies of wooden pattern and model makers

Reference	Methods	Results	Notes
Swanson & Belle (1982)	1070 wooden model and pattern makers active in 1970 in seven automobile manufacturing plants located in Detroit (USA); cancer incidence in the cohort studied 1970–78 and compared with that of the population of metropolitan Detroit. 24.1% lost to follow-up	SIR: All cancers, 1.5 [1.1–2.0], 40 observed Colon and rectum, 2.9 [1.4–5.1], 11 observed Salivary glands, 21 [2.4–72], 2 observed No other significantly increased SIR	
Swanson <i>et al.</i> (1985)	316 wooden model or pattern makers employed by one US automobile manufacturing company in 1976 and followed through 1982. Colon cancer incidence in the cohort compared with that of the population of metropolitan Detroit. 36.4% lost to follow-up	SIR: Colon cancer, 4.9 [1.3–13], 4 observed	
Tilley <i>et al.</i> (1990)	7062 white male pattern and model makers active or retired in 1980 in the USA and Canada followed through 1985 in order to study cause-specific mortality and incidence of colorectal cancer; expected figures derived from incidence rates of Detroit SEER registry and from US death rates; 6% lost to follow-up by 1984	SMR: All causes, 0.7 (0.6–0.8), 335 observed All malignant neoplasms, 0.9 (0.7–1.1), 108 observed Large intestine, 2.0 (1.3–3.0), 22 observed No other significantly increased SMR SIR: Colorectal cancer, 1.1 (0.8–1.5), 39 observed	Incidence study limited to subcohort involved in screening programme
Becker <i>et al.</i> (1992)	528 model and pattern makers employed by a German automobile company between 1960 and 1985, followed through 1985; mortality compared with that of a cohort of tool makers in the same company. 2% of model makers and 6% of tool makers lost to follow-up	RR: All causes, 1.1 (0.5–2.2), 28 observed All malignant neoplasms, 1.8 (1.3–2.7), 11 observed Stomach, 6.9 (1.4–32), 3 observed Genitourinary organs, 5.7 (1.4–23), 3 observed Brain, 9.8 (1.4–70), 2 observed	

Table 19 (contd)

Reference	Methods	Results	Notes
Roscoe <i>et al.</i> (1992)	2294 white male wooden model makers employed for any time between 1940 and 1980 by three US automobile companies in metropolitan Detroit; vital status ascertained through 1984. US mortality rates used as reference.. 1% lost to follow-up	SMR: All causes, 0.8 (0.7–0.8), 706 observed All cancers, 1.0 (0.8–1.2), 173 observed No significantly increased SMRs; no association between colon or stomach cancer and exposure to wood in nested case-control study	Automotive model makers in the USA worked with mahogany, maple, birch, cherry and South American hardwoods; since the mid-1950s, mainly South American hardwoods, such as cativo, for dies

SIR, standardized incidence ratio; SMR, standardized mortality ratio; RR, rate ratio

establish occupational and exposure histories. Excess risks were observed for exposure to hardwood dust alone (odds ratio, 2.0; 95% CI, 0.2–21), softwood dust alone (3.3; 1.1–9.4) and mixed hard and softwood dusts (12; 2.4–59) on the basis of a matched analysis. Both of the subjects who had been exposed to hardwood dust alone, and none of the 13 subjects exposed to softwood dust alone, had adenocarcinomas. Subjects who had been exposed to mixed hardwood/softwood dusts had cancers of mixed histological types.

Olsen *et al.* (1984) studied the relationship between occupational factors and sinonasal and nasopharyngeal cancer in Denmark. A total of 488 cases of sinonasal carcinoma (excluding sarcomas) were diagnosed in Denmark between 1970 and 1982 and identified by the Danish Cancer Registry. The 2465 controls were patients with cancers of the colon, rectum, prostate or breast, diagnosed during the same period. Employment histories (industry only) back to 1964 were collected through linkage with national pension fund records, and current occupation was established through linkage with the Central Population Registry. A group of industrial hygienists reviewed the employment histories and assigned an exposure category (unexposed, probably exposed, certainly exposed, insufficient information) to a list of predetermined compounds, which included wood dust (relative risk, 2.5; 95% CI, 1.7–3.7 for men).

Olsen and Asnaes (1986) further evaluated this data set by histologically confirmed sub-groups (squamous-cell and adenocarcinoma). The odds ratio for adenocarcinoma among men with definite exposure to wood dust was 16 (5.2–51), which increased to 30 (8.9–104) when only exposures 10 or more years before diagnosis were considered. The odds ratio for squamous-cell carcinoma associated with definite exposure to wood dust was 1.3 (0.6–2.8) and did not change when only exposure 10 or more years before diagnosis was considered.

Hayes *et al.* (1986a) conducted a case-control study of sinonasal cancer in the Netherlands. Cases were histologically confirmed primary epithelial sinonasal cancers newly diagnosed in men aged 35–79 years between 1978 and 1981 who were identified by the six major institutions in the Netherlands which treat head and neck tumours. The controls were a random sample of living and dead males in the Netherlands in 1980, who were selected from municipal resident registries and the records of the Central Bureau of Genealogy, respectively. A total of 91 case patients or their survivors and 195 controls or their survivors were interviewed. Detailed occupational and exposure histories were collected. An excess risk for all sinonasal cancers (odds ratio, 2.5 [95% CI, 1.2–5.1]), especially adenocarcinoma (18 [5.5–57]), was observed for employment in wood-related occupations, after adjustment for age. Exposure to wood dust was assessed by an industrial hygienist who was unaware of the case or control status of the person. The risk of adenocarcinoma was higher when only high-exposure jobs were considered (26 [7.0–99]), but no excess of squamous-cell carcinoma was observed (0.5 [0.1–2.9]) among men in the highest exposure category. The excess of adenocarcinoma was seen among workers first employed before 1942. The authors stated that the woodworkers had been exposed to both hard- and softwood dusts.

Merler *et al.* (1986) conducted a case-control study of nasal cancer in Vigevano, Italy, with the primary goal of examining the risk in the leather industry. Cases were malignancies of the sinonasal cavity newly diagnosed in residents of Vigevano between 1968 and 1982 and identified through the otolaryngology departments of local hospitals, the cancer registry of the

National Cancer Institute of Milan and the mortality records of the city. Two controls per case, matched on age, sex, vital status and, if dead, on year of death, were chosen from electoral rolls (living controls) and mortality records (dead controls). Interviews, which included occupational histories, were conducted with 21 case patients and 39 controls. No case patient and two controls had been exposed to wood dust.

Bolm-Audorff *et al.* (1989, 1990) conducted a case-control study of histologically confirmed cases of nasal and nasopharyngeal cancer diagnosed in hospitals in Hesse, Germany, between 1 January 1983 and 31 December 1985. Fifty-four of the 66 cases were sinonasal cancers. A single control was matched to each case on age, sex and residence and was chosen from among patients with non-occupational bone fractures. Information on exposure was collected through interviews. The relative risk for all cases (sinonasal and nasopharyngeal cancer) was 3.8 (95% CI, 0.8–17), which increased to 7.8 (1.3–48) for an exposure of five years or more. Six of the seven exposed case patients had sinonasal cancer. The two exposed cases of sinonasal adenocarcinoma were both associated with exposure to hardwood (beech and oak), while the two other carcinomas were associated with exposure to softwoods; the two remaining exposed cases, a lymphoma and a neuroblastoma, were associated with exposure to mixed woods and hardwoods, respectively. Two controls had been exposed to wood dust. [The Working Group noted that the tree species were not identified for controls.]

Vaughan (1989) performed a case-control study of squamous-cell cancers of the sinonasal cavity in western Washington State, United States. Living patients in whom the cancers were diagnosed between 1979 and 1983 were identified from a population-based tumour registry, and 27 people with sinonasal cancer were interviewed. Random-digit dialling was used to obtain 552 controls who were similar in age and sex to the cases. Interviews were used to collect life-time work histories. The analyses were adjusted for age, sex, smoking and alcohol consumption. Excesses of squamous-cell cancers of the sinonasal cavity were reported for forestry and logging workers (odds ratio, 1.8; 95% CI, 0.4–7.2) and woodworking machine operators (7.5; 1.5–37). Vaughan and Davis (1991) later categorized these cases according to exposure to wood dust. The odds ratio for employment in any wood-related occupation was 2.4 (0.8–6.7), which increased to 7.3 (1.4–34) when only exposure for 10 or more years after an induction period of 15 years was considered. The authors stated that the cases were associated with exposure predominantly to dust from softwood. Information on exposure was obtained from surrogates for half of the cases but none of the controls; however, exclusion of cases for which information was obtained from surrogates did not greatly affect the risk estimates.

Luce *et al.* (1991, 1992, 1993) conducted a case-control study of patients with sinonasal cancer diagnosed between January 1986 and February 1988 in 27 participating hospitals in France. A total of 207 patients out of 303 were alive at the time of interview and agreed to participate in the study; 57 died before being interviewed, and 39 could not be located. Controls of similar age and sex to the patients were recruited from two sources: patients with a cancer diagnosed at another site and at the same or a nearby hospital; and neighbourhood controls selected from lists provided by the patients. Of these, 323 hospital and 86 neighbourhood controls were eligible and agreed to participate in the study. Detailed occupational and exposure histories were collected by personal interviews. The degree of occupational exposure to wood

dust was assessed by an industrial hygienist who was unaware of the case or control status of the person. Among men, an elevated risk for sinonasal adenocarcinoma (odds ratio, 2.89; 95% CI, 1.36–6.15, based on 77 exposed cases and 29 exposed controls) was associated with probable or definite, medium–high exposure to wood dust, but no relationship was observed for squamous-cell carcinoma (1.0; 0.4–2.6). The results for specific occupational groups are presented in Tables 21 and 22.

Lerclerc *et al.* (1994) reported the results of further analyses of this study in respect of species of wood. Eighty of the 82 male patients with adenocarcinoma had been exposed to hardwood dusts, but only seven of these to hardwood alone. The odds ratio for adenocarcinoma among men exposed to hardwood or mixed wood dust was 1.68 (95% CI, 0.78–3.62). Positive trends were observed for duration and intensity of exposure. The relative risk was higher among men exposed before 1946 (2.54) than those first exposed afterwards (1.37). [The odds ratio for adenocarcinoma among men exposed exclusively to hardwood was 5.7.] Seventeen of the 59 male squamous-cell cases were associated with exposure to wood dust—three to hardwood only, three to softwood only and the remainder to a mixture of woods. The authors stated that because few subjects were exposed to one wood type alone, the relative risks for squamous-cell carcinoma could not be calculated for exposure to each type of wood alone. The odds ratios for squamous-cell carcinoma associated with exposure to hardwood (or hardwood plus other woods) and softwood (or softwood plus other woods) were 1.4 and 1.7 (not significant), respectively. Duration and intensity of exposure to either hard- or softwoods were not clearly associated with the risk for squamous-cell carcinoma, although some evidence for an excess risk was observed for subjects exposed before 1946. [The Working Group was concerned that the procedures for selecting non-hospital controls may have artificially biased the proportion of woodworking controls downward. For non-hospital controls, cases were asked to provide ‘the names of several persons (colleagues excluded)’ to serve as referents. The proportion of non-hospital controls exposed to hardwoods (18.8%) and softwoods (20.3%), however, was similar to the proportion among hospital controls (18.3% and 16.3%, respectively), indicating that bias was unlikely.]

Zheng *et al.* (1992a) conducted a case–control study of nasal cancer in Shanghai, China. Patients with newly diagnosed sinonasal cancer between January 1988 and February 1990 were identified in the population-based cancer registry of Shanghai. Controls were randomly selected in the general population from the records of the Shanghai Resident Registry. Personal interviews were conducted with 60 cases and 414 controls, and information was collected on occupational history and exposures. Of the cases, 25 were squamous-cell carcinomas, six were adenocarcinomas, 22 were tumours of other histological types and the remainder were not evaluated histologically. The relative risks for all sinonasal cancers combined were calculated for self-reported exposure to wood dust (odds ratio, 1.9; 95% CI, 0.7–5.0) and employment in wood-related occupations (1.7; 0.5–6.3).

(b) Occupational group

Brinton *et al.* (1977) conducted a case–control study among people who died in North Carolina (United States) counties in which at least 1% of the population was employed in furniture manufacture according to the 1963 census. Death certificates were used to identify 37

cases of cancer of the nasal cavity and sinuses between 1956 and 1974. Two controls for each case ($n = 73$) were randomly selected from among people who had died and were of the same sex, race, county of death, age at death and year of death. Information on occupation and industry was also obtained from the death certificates, and a matched analysis was performed. Elevated risks were observed for people employed in the furniture industry (odds ratio, 4.4; 95% CI, 1.3–15) and in other woodworking occupations (sawmill workers and carpenters) (1.5; 0.4–4.3).

Cecchi *et al.* (1980) performed a case–control study of sinonasal adenocarcinoma in Florence, Italy. Cases diagnosed between 1963 and 1977 were identified from the records of the otorhinolaryngology clinic or the radiology institute of the University of Florence. Eleven of 13 patients or their survivors [numbers of patients and survivors not given] were interviewed in order to obtain information on occupation and smoking habits. Two controls per case, matched on sex, age, place of residence, smoking and year of admission to hospital, were selected from among non-cancer internal medicine patients and received the same interview. Three case patients and two controls had been employed as woodworkers. Of the exposed patients, two had worked in small woodworking shops, and the third had worked with both wood and leather.

Roush *et al.* (1980) conducted a case–control study of sinonasal cancer in Connecticut (United States) based on the tumour register. Cases were sinonasal cancers in 216 men 35 years of age or older who had died between 1935 and 1975. The 691 controls were a random sample of men aged 35 years or older who had died in Connecticut between 1935 and 1975. Occupational information was collected from death certificates and city directories, in which information is based on interviews conducted during door-to-door surveys. Job titles were classified for exposure to wood dust on the basis of a review of the literature. The odds ratio associated with wood-related occupations was 4.0 (95% CI, 1.5–11) when information from both sources was considered. The odds ratio was somewhat lower (2.8) when only information from death certificates was considered and somewhat higher (5.9) when only information from city directories was considered.

Tola *et al.* (1980) performed a case–control study of patients with malignant tumours of the nose and paranasal sinuses reported to the Finnish Cancer Registry between 1970 and 1973. For each case a single control of similar age and sex was chosen from among cancer patients (other than respiratory) from the same geographical area. Questionnaires on occupational history and exposures were completed by 45 case subjects and 45 controls. Of the cases, 20 were squamous-cell carcinomas, 10 were transitional-cell carcinomas and two were malignancies classified as adenocarcinoma. One patient with an adenocarcinoma had been employed as a joiner and had been exposed mainly to oak dust. One control had been employed as a carpenter. No other results related to exposure to wood were reported.

Elwood (1981) reported the results of a case–control study of 121 men with primary epithelial tumours of the sinonasal cavity seen at the main cancer treatment centre in British Columbia, Canada, between 1939 and 1977. Of the cases, 61 were squamous-cell carcinomas, 20 were anaplastic carcinomas, 16 were transitional-cell carcinomas, 11 were adenocarcinomas, six were sarcomas, and seven were of unknown histological type. A control group of 120 patients with cancer that was considered to be unrelated to smoking or outdoor work, matched

on age and year of diagnosis, was chosen. Information on occupation and smoking was retrieved from medical records, and relative risks were calculated using conditional logistic regression after adjustment for smoking and ethnicity. An elevated risk was observed for employment in wood-related occupations (odds ratio, 2.5; $p < 0.03$). Of the 28 exposed patients, 10 were loggers, seven were carpenters, four were forestry workers, four were construction workers, two were log scalers and one was a cabinet-maker. The authors reported that the predominant exposure of all but the cabinet-maker would have been to native softwoods.

Hardell *et al.* (1982) conducted a case-control study of nasal and nasopharyngeal cancers in northern Sweden to examine their relationship with exposure to phenoxy acids or chlorophenol, which included 44 male patients with sinonasal cancer who had been reported to the Swedish Cancer Registry between 1970 and 1979. Thirty-one of the cases were squamous-cell carcinomas, four were anaplastic carcinomas and three were adenocarcinomas; six were tumours of other histological types. The 541 controls had initially been identified and interviewed for a study of soft-tissue sarcoma and lymphoma. Information on exposure and employment history were collected using postal questionnaires and supplemental telephone interviews. A crude relative risk of 2.0 [95% CI, 1.1–3.6] was observed for previous employment as a carpenter, cabinet-maker or sawmill worker (19 exposed cases, 151 exposed controls). The authors noted that little hardwood is used for furniture making in northern Sweden.

Battista *et al.* (1983) performed a case-control study of sinonasal cancer in the province of Siena, Italy, where 4–7% of the active male population is employed in wood-related industries. They studied 36 male patients in whom sinonasal cancers were diagnosed at the Ear, Nose and Throat Clinic or the Radiotherapy Unit in Siena between 1963 and 1981. Seventeen (47%) of the cases were squamous-cell carcinomas and five (14%) were adenocarcinomas. For each case, five referents were selected from among men of the same age (within one year) who were admitted to the medical clinic of Siena for other diseases at the same time. All case patients and 164 of the 180 referents or their next-of-kin completed a postal questionnaire, and occupational histories were collected. Exposure to wood dust was defined as employment as a woodworker or cabinet-maker. An elevated risk for all sinonasal cancers was reported for exposed men (odds ratio, 4.7; 95% CI, 1.7–13), and the risk was especially increased for adenocarcinoma (90; 20–407). The seven case patients with exposure to wood dust had used a wide variety of species, the commonest being chestnut (four cases), oak (four cases), poplar (three cases) and fir (two cases).

Brinton *et al.* (1984) conducted a case-control study of patients with sinonasal cancer admitted to four hospitals in North Carolina and Virginia, United States, between 1970 and 1980. Two controls for each living case patient, matched on year of admission, age, sex, race and area of residence, were selected from living hospital patients. For deceased cases, similar matching criteria were used, but one hospital patient (not required to be living) and one patient with a death certificate were chosen. Potential controls were excluded if an upper aerodigestive cancer, oesophageal cancer, benign respiratory neoplasm, mental disorder or chronic sinonasal disease had been diagnosed. Telephone interviews were conducted with 160 case patients and 290 controls or their survivors. An elevated relative risk for sinonasal adenocarcinoma was associated with previous employment in any wood-related job (odds ratio, 3.7; $p < 0.05$), but no excess of squamous-cell carcinoma was observed (odds ratio, 0.8).

Ng (1986) conducted a case-control study of cancer of the nasal cavity and sinuses among Chinese people in Hong Kong. Two series of controls were used: people with nasopharyngeal cancer and people with other malignancies, all of which were diagnosed between 1974 and 1981 at the Institute of Radiology and Oncology in Hong Kong. There were 225 cases of nasal cancer (119 squamous-cell, 50 anaplastic, four adenocarcinomas, 37 of other histological types and 15 of unknown histological type), 224 cases of nasopharyngeal cancer (112 squamous-cell, 102 anaplastic and 10 of unknown histological type) and 226 controls with other malignancies. Occupational histories were collected from medical records. Two wood-related occupational categories were considered: furniture makers and woodworkers (comprising two cancers of the nasal cavity and sinuses, five nasopharyngeal cancers and one other malignancy) and construction carpenters (comprising two cancers of the nasal cavity and sinuses and three nasopharyngeal carcinomas). None of the four nasal cavity and sinus adenocarcinomas were in wood-related workers. No odds ratios or other estimates of relative risk were presented for wood-related occupations.

Fukuda *et al.* (1987) and Fukuda and Shibata (1988, 1990) reported the results of a case-control study on Hokkaido Island in Japan of cases of squamous-cell carcinoma of the maxillary sinus, newly diagnosed in 1982-86 in people between 40 and 79 years of age, at the two university and the two medical college hospitals on Hokkaido Island. Two controls per case, matched on sex, age and residence, were chosen from among a pool of potential controls selected from telephone directories. A questionnaire was posted to all potential cases and controls, who were later telephoned to obtain their permission to participate in the study and to confirm the responses to the questions. The matched analysis in the latest published results included 169 eligible cases and 338 eligible controls. The exposure category of woodworkers consisted of people employed as carpenters, joiners, furniture makers and other woodworkers. Excess risks for squamous-cell carcinoma of the maxillary sinus were observed among both men (relative risk, 2.9; 95% CI, 1.5-5.6) and women (2.0; 0.3-14). A significant trend ($p < 0.05$) of increasing risk with increasing duration of employment was also seen.

Takasaka *et al.* (1987) performed a case-control study of male patients with nasal or paranasal cancer who were admitted to Tohoku University Hospital, Japan, between 1971 and 1982. Three to five controls of the same sex, age and date of admission were selected from among patients with other otorhinolaryngological diseases admitted to the same hospital. Mailed questionnaires requesting occupational history and information on exposures were completed by 107 case patients and 413 controls. Eighty-five of the 98 cases for which histological information was available were squamous-cell carcinomas and six were adenocarcinomas. Excess risks were associated with longest-held occupation as a forester (odds ratio, 2.0; 95% CI, 0.5-7.3), woodworker (1.6; 0.4-7.1) or carpenter (2.1; 0.8-5.8).

Bimbi *et al.* (1988) conducted a hospital-based case-control study in Milan, Italy, of 53 patients with malignant epithelial cancers admitted between 1982 and 1985 to the Head and Neck Oncology Department of the National Institute for the Study and Treatment of Cancer. Controls were 217 patients admitted to the same department during the same years, mainly for cancers of the nasopharynx, thyroid and salivary gland. Information on occupational history was collected from medical records. Three cases and no control had been employed as woodworkers.

Finkelstein (1989) reported the results of a study based on information from death certificates of 124 men, 35 years of age or older, who had died of cancer of the nasal cavity or paranasal cavity in Ontario, Canada, between 1973 and 1983. One control per case, matched on age and year of death, was chosen from among people who had died of other causes. Information on usual job and industry was collected from death certificates. Nine cases and six controls had been employed in wood-related occupations (odds ratio, 1.9 [95% CI, 0.6–6.4]). Workers who had been employed in nickel refining (10 cases and no control) were excluded from the unmatched analyses.

Kawachi *et al.* (1989) reported the results of an exploratory study to examine the risk for cancer among woodworkers. Case patients and controls were men in the New Zealand Cancer Registry in whom cancer had been diagnosed between 1980 and 1984 and whose occupation was noted in Registry records. The case patients were 46 registrants in whom cancer of the nasal cavity and sinuses had been diagnosed, while the controls were 19 858 registrants with cancers at all other sites. The only information available on exposure was the current or most recent occupation. No excess risk was observed (odds ratio, 1.0; 95% CI, 0.2–4.0; based on two exposed cases) after adjustment for age.

Loi *et al.* (1989) conducted a case-control study of nasal cancer in the Pisa area of Italy, a region where there are many factories manufacturing wooden products, especially furniture. Case patients were 38 male nasal cancer patients admitted to Pisa University Hospital between October 1972 and October 1983; five controls per case, matched on sex, age, province of usual residence and admission date, were chosen from among patients admitted to the same hospital for diseases other than respiratory cancer or lymphoma. A postal questionnaire was used to obtain information on occupational history and smoking habits. Workers who had been employed in wood-related occupations for six or more months at least five years before diagnosis were considered to have been exposed. Subjects who had been employed in leather-related occupations, their matched controls and other controls who had been employed in leather-related occupations were excluded from the analyses of wood-related risks. A relative risk of 9.7 (95% CI, 3.2–29) was observed for employment in wood industries. The relative risk for adenocarcinoma alone tended to infinity, as all case subjects had been exposed. Individual information on exposure to different wood species was not available, but the authors stated that chestnut, walnut and fir were the most commonly used in the region. [The Working Group noted that some of the study subjects might have died before the study was conducted, and, consequently, next-of-kin may have been interviewed.]

Shimizu *et al.* (1989) conducted a case-control study of 45 men and 21 women with newly diagnosed squamous-cell carcinoma of the maxillary sinus at six university hospitals in north-eastern Japan, between October 1983 and October 1985. Two controls, matched on age and sex, were selected from a random sample of residents in the same area from telephone directories. Each patient was asked to complete a questionnaire during initial hospitalization, which requested information about previous occupations and other potential risk factors; controls completed the same questionnaire by post. A matched analysis was performed. The relative risk among men for woodworking or joinery was 2.1 (95% CI, 0.8–5.3); the risk was 7.5 (1.5–39) when only jobs involving sanding or turning were considered.

Viren and Imbus (1989) conducted a study based on information on death certificates of 536 people in the United States who had died of nasal cancer in the states of Washington and Oregon between 1963 and 1977, Mississippi between 1962 and 1977 and North Carolina between 1964 and 1977. Two controls per case ($n = 1072$) were chosen from among people in the same states who had died from causes other than cancer, non-malignant respiratory disease or accidents; they were matched to the case patient on sex, age, race and year of death. Usual occupation and industry were obtained from death certificates. A matched analysis was performed only for men (332 cases, 664 controls). Relative risks of 3.3 ($p < 0.01$) for forestry and logging workers, 1.3 for woodworkers and woodworking machine operators and 1.6 for carpenters were observed. [The Working Group noted that the subjects from North Carolina may also have been included by Brinton *et al.*, 1977.]

Haguenoer *et al.* (1990) conducted a case-control study to investigate occupational risk factors for cancers of the upper respiratory and digestive tracts (nose, lips, buccal cavity, pharynx and larynx) in northern France. An occupational history, which included only jobs held for at least 15 years, was established by interview; people who did not have at least one job that met this criterion (one-half the subjects) were excluded from the study. There were 14 histologically confirmed sinonasal cancers among men treated in the first semester of 1983. Two controls per case, matched for sex, age, ethnic group, area of residence and histories of smoking and alcohol drinking, were chosen from among non-cancer hospital patients in the same region. Four patients with sinonasal cancer and no matched control reported previous employment as a woodworker.

Comba *et al.* (1992a) reported the results of a collaborative case-control study in north-eastern and central Italy of cases of sinonasal cancer diagnosed between 1982 and 1987 at hospitals providing services to the provinces of Verona, Vicenza and Siena. Four controls per case were selected from among patients admitted for diseases other than chronic rhino-sinonasal diseases or nasal bleeding to the same hospital at the same time, who were similar to the case patient with regard to sex, age and area of residence. Personal interviews were conducted by telephone or post with 78 of 96 case patients and 254 of 378 controls or their next-of-kin, to collect information on occupational history and exposures. An elevated relative risk associated with woodworking was observed for both men (odds ratio, 5.8 [95% CI, 1.8–18]) and women (3.2 [0.2–50]). The relative risks for sinonasal adenocarcinoma (14 [2.3–83]) and squamous-cell carcinoma (1.7 [0.3–9.2]) were presented for men only. The authors noted that the types of wood used by the case patients were both hard- and softwoods of many species, including birch, fir, poplar, beech, maple, cherry, oak, mahogany, walnut and chestnut.

Comba *et al.* (1992b) conducted another case-control study among the residents of Brescia Province in north-eastern Italy, with the primary aim of examining the relative risk for sinonasal cancer associated with employment in the metal industry. Cases were malignant epithelial sinonasal tumours treated at the ear, nose and throat department or the radiotherapy unit of Brescia Hospital between 1980 and 1989. Four controls per case were chosen from among patients treated at the same centres for benign and malignant tumours of the head and neck, excluding epidermoid carcinomas of the tongue, oral cavity, oro- and hypopharynx and larynx, who were of the same sex and age. A total of 34 case patients (23 men) and 102 controls

(70 men) or their survivors [numbers of cases and survivors not given] were interviewed, and detailed occupational histories were obtained. The age-adjusted odds ratio for male woodworkers was 11 [95% CI, 0.5–229].

Zheng *et al.* (1993) conducted a case-control study of 147 white men, 45 years of age or older, who had died from sinonasal cancer in 1985 and were identified in a national survey of mortality in the United States in 1986. Controls were 449 white men who had died during the same period from causes not related to smoking or alcohol consumption. The next-of-kin of cases and controls received a structured questionnaire by post requesting information on demographic factors, histories of smoking and alcohol consumption, occupational history and dietary habits. After adjustment for age and cigarette smoking, a relative risk of 1.7 (95% CI, 0.6–4.3) was observed for previous employment as a carpenter or other wood-related worker, relative to professional, managerial, technical and sales workers.

Magnani *et al.* (1993) conducted a case-control study of sinonasal cancer in the district of Biella in north-western Italy with the primary goal of examining risks in the woollen textile industry. Cases were epithelial or histologically unspecified sinonasal cancers diagnosed among residents of the local health areas of Biella and Cossato between 1976 and 1988. Four controls per case were chosen from among patients of the same sex and age, who were admitted to the same hospital in the same year with diagnoses other than respiratory cancer. Mailed questionnaires or telephone interviews, which included an occupational history, were completed by 26 cases and 111 controls or their relatives. An elevated risk for sinonasal cancer was associated with employment as a wood or furniture worker (odds ratio, 4.4; 95% CI, 1.4–13); the risk was much higher when only adenocarcinomas were considered (22; 4.4–124).

The studies on sinonasal cancer are summarized in Tables 20, 21 and 22.

Demers *et al.* (1995) performed a pooled analysis of case-control studies of sinonasal cancer and exposure to wood dust, in which the following criteria had been met: the histological types of the cases were identified; occupational histories had been collected from patients (or their survivors) and controls by interview or questionnaire; and data on age, sex and tobacco smoking were available. The authors of the 15 studies that met these criteria were asked to participate in the pooled analysis; 12 were both able and willing to do so. The studies were those conducted in Shanghai, China (Zheng *et al.*, 1992a); France (Luce *et al.*, 1991, 1992, 1993); Hesse, Germany (Bolm-Audorff *et al.*, 1989, 1990); Siena, Verona and Vicenza, Italy (Comba *et al.*, 1992a); Brescia, Italy (Comba *et al.*, 1992b); Biella, Italy (Magnani *et al.*, 1989, 1993); Vigevano, Italy (Merler *et al.*, 1986); the Netherlands (Hayes *et al.*, 1986a,b, 1987); northern Sweden (Hardell *et al.*, 1982); North Carolina and Virginia, United States (Brinton *et al.*, 1984); Los Angeles, United States (Mack & Preston-Martin, unpublished data); and Seattle, United States (Vaughan, 1989; Vaughan & Davis, 1991). The aggregated data consisted of 680 male cases (169 adenocarcinomas, 329 squamous-cell cancers, 157 of other histology and 25 of unknown histology), 2349 male controls, 250 female cases (26 adenocarcinomas, 101 squamous-cell cancers, 105 of other histology and 18 of unknown histology) and 787 female controls. Seven categories of jobs with potential exposure to wood dust were defined by combining occupation and industry title: forestry workers, loggers, pulp and paper workers, sawmill

Table 20. Results of community-based case-control studies of sinonasal cancer: all histological types and unspecified

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/ RR	95% CI or <i>p</i>	Comments
Exposure to wood dust								
Hernberg <i>et al.</i> (1983)	Denmark/ Finland/ Sweden	MF	167/167	Interviews	Hardwood only	2.0	0.2–21	
					Softwood only; primarily pine and spruce, also birch and aspen	3.3	1.1–9.4	
					Mixed hard- and softwood	12	2.4–59	
Olsen <i>et al.</i> (1984)	Denmark	MF	488/2465	Linkage with national pension fund records	Exposure to wood dust (men only)			Exposure based on expert assessment of work history
					Probable exposure	1.2	0.7–2.1	
					Definite exposure ≥ 10 years since first exposure	2.5 2.9	1.7–3.7 1.8–4.7	
Merler <i>et al.</i> (1986)	Italy	MF	21/39	Interview	Exposure to wood dust			0/2 exposed case/controls
Bolm-Audorff <i>et al.</i> (1989, 1990)	Germany	MF	66/66	Interviews	Exposure to wood dust; oak, beech and softwood	3.8	0.8–17	Cases include 12 naso- pharyngeal cancers. Six of seven exposed cases had nasal cancer. Controls matched on sex, age and residence
					Duration ≥ 5 years	7.8	1.3–48	
Zheng <i>et al.</i> (1992a)	China	MF	60/414	Interviews	Exposure to wood dust	1.9	0.7–5.0	Self-reported exposure; adjusted for age
					Wood-related occupations	1.7	0.5–6.3	
Occupational group								
Brinton <i>et al.</i> (1977)	USA	M	37/73	Death certificates	Furniture industry	4.4	1.3–15	Controls matched on sex, race, age, county and year of death
					Sawmill workers, carpenters and other woodworking occupations	1.5	0.4–4.3	
Roush <i>et al.</i> (1980)	USA	M	216/691	Death certificates and city directories	Wood-related occupation			
					Death certificate only	2.8		
					City directories only	5.9		
					Either source of information	4.0	1.5–11	

Table 20 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/ RR	95% CI or <i>p</i>	Comments
Occupational group (contd)								
Tola <i>et al.</i> (1980)	Finland	MF	45/45	Questionnaires	Wood-related occupations			1/1 exposed case/control
Elwood (1981)	Canada	M	121/120	Medical records	Wood-related occupations; primarily softwood	2.5	<i>p</i> < 0.03	Matched on age and year of diagnosis and adjusted for smoking and ethnicity
Hardell <i>et al.</i> (1982)	Sweden	M	44/541	Questionnaires	Carpenter, cabinet-maker or sawmill worker	2.0	[1.1-3.6]	Crude relative risk; 19/151 exposed cases/controls
Battista <i>et al.</i> (1983)	Italy	M	36/164	Questionnaires	Woodworker or cabinet-maker; exposure to chestnut, oak, poplar, fir, alder, walnut, beech and acacia	4.7	1.7-13	Cases of carcinoma only; matched on age
Brinton <i>et al.</i> (1984)	USA	MF	160/290	Interviews	Furniture industry Lumber industry Carpentry	0.8 1.4 1.5	0.3-2.0 0.7-2.6 0.6-3.4	Adjusted for year of admission, age, sex, race and area of residence
Hayes <i>et al.</i> (1986a)	Netherlands	M	91/195	Interviews	Wood-related occupations Furniture and cabinet-making Factory joinery/carpentry House carpentry Other wood-related occupations	2.5 13 2.1 0.6 1.1	[1.2-5.1] [2.7-59] [0.4-11] [0.1-4.3] [0.3-4.8]	Adjusted for age Adjusted for age
Ng (1986)	Hong Kong	MF	225/226	Medical records	Furniture makers, woodworkers Construction carpenters			2/1 exposed cases/control 2/0 exposed cases/control
Takasaka <i>et al.</i> (1987)	Japan	M	107/337	Questionnaires	Longest-held occupation Foresters Woodworkers Carpenters	2.0 1.6 2.1	0.5-7.3 0.4-7.1 0.8-5.8	

Table 20 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/ RR	95% CI or <i>p</i>	Comments
Occupational group (contd)								
Bimbi <i>et al.</i> (1988)	Italy	MF	53/217	Medical records	Wood industry			3/0 exposed cases/control
Finkelstein (1989)	Canada	M	124/124	Death certificates	Wood-related occupations	1.9	[0.6–6.4]	
Kawachi <i>et al.</i> (1989)	New Zealand	M	46/19 858	Tumour registry records	Woodworkers	1.0	0.2–4.0	Adjusted for age
Loi <i>et al.</i> (1989)	Italy	M	38/153	Questionnaires	Wood industries. Individual exposure by wood species not known, but walnut, chestnut and fir commonly used in region	9.7	3.2–29	Calculated after excluding cases and controls who were leather workers
Viren & Imbus (1989)	USA	M	332/664	Death certificates	Forestry and logging Woodworking and woodworking machine operators Carpenters	3.3 1.3 1.6	<i>p</i> < 0.01	Matched on sex, age, race, state and year of death 5/8 exposed cases/ controls 14/18 exposed cases/ controls
Haguenoer <i>et al.</i> (1990)	France	M	14/28	Interviews	Woodworkers			Only jobs held for at least 15 years were evaluated; 4/0 exposed cases/control
Comba <i>et al.</i> (1992a)	Italy	MF	78/254	Interviews and questionnaires	Woodworkers (men) Furniture makers, joiners or carpenters Lumberjack Woodworkers (women)	5.8 6.5 4.1 3.2	[1.8–18] [1.7–25] [0.9–19] [0.2–50]	Results for men adjusted for age. Exposure to both hard- and softwoods
Comba <i>et al.</i> (1992b)	Italy	M	23/70	Interviews	Woodworkers	11	[0.5–229]	Adjusted for age

Table 20 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/ RR	95% CI or <i>p</i>	Comments
Occupational group (contd)								
Zheng <i>et al.</i> (1993)	USA	M	147/449	Questionnaire	Carpenters and other woodworkers	1.7	0.6–4.3	Adjusted for age and smoking
Magnani <i>et al.</i> (1993)	Italy	MF	26/111	Questionnaires	Wood and furniture workers	4.4	1.3–13	
					Duration, 1–9 years	3.5	0.6–19	
					Duration, ≥ 20 years	5.8	1.4–24	

OR, odds ratio; RR, relative risk; CI, confidence interval; M, male; F, female

Table 21. Results of community based case-control studies of sinonasal cancer: adenocarcinoma

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/ RR	95% CI or <i>p</i>	Comments	
Exposure to wood dust									
Hayes <i>et al.</i> (1986a)	Netherlands	M	23/195	Interviews	Wood-related occupations	18	[5.5-57]	Adjusted for age	
					Furniture and cabinet-making	140	[18-1094]	Adjusted for age	
					Factory joinery/carpentry	16	[2.1-125]		
					Housing carpentry and other wood-related occupations	0.0		0/9 exposed case/controls	
					Exposure to wood dust	8.5	[2.6-28]	Exposure based on expert assessment of work history	
				High exposure	26	[7.0-99]			
Olsen & Asnaes (1986)	Denmark	M	39/2465	Linkage with national pension fund records	Definite wood dust exposure ≥ 10 years since first exposure	16 30	5.2-51 8.9-104	Exposure based on expert assessment of work history records; adjusted for exposure to formaldehyde	
Luce <i>et al.</i> (1991, 1992, 1993); Leclerc <i>et al.</i> (1994)	France	M	82/320	Interviews	Loggers	0.6	0.1-4.6	All results adjusted for age	
					duration >15 years	0.0			
					Cabinet-makers	35	18-69		
					duration >15 years	33	14-76		
					Woodworking machine operators	7.4	3.5-16		
					duration >15 years	48	8.8-260		
					Carpenters, joiners	25	15-44		
					duration >15 years	45	22-50		
					Exposure to wood dust				Exposure based on expert assessment of work history
					Probable or definite, medium-high exposure	289	136-615		
Hardwood dust (incl. mixed)	168	78-362							
> 35 years of exposure	303	64-1427	80 of 82 cases exposed to hardwood or mixed dusts						
Highest level of exposure	530	104-2696							
First exposed before 1946	254	55-1185							

Table 21 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/ RR	95% CI or <i>p</i>	Comments
Occupational group								
Cecchi <i>et al.</i> (1980)	Italy	MF	11/22	Interviews	Wood-related occupation	[3.7]	[0.5-27]	Crude odds ratio; 3/2 exposed cases/controls
Battista <i>et al.</i> (1983)	Italy	M	5/NR	Questionnaires	Woodworker or cabinet-maker Chesnut, oak, poplar, alder, walnut and acacia	90	20-407	
Brinton <i>et al.</i> (1984)	USA	M	13/183	Interviews	Furniture industry Lumber industry Carpentry Any wood-related job	5.7 1.6 2.9 3.7	1.7-19 <i>p</i> < 0.05	4 exposed cases 3 exposed cases 10 exposed cases;
Comba <i>et al.</i> (1992)	Italy	M	13/184	Interviews and questionnaires	Wood workers (men)	14	[2.3-83]	
Magnani <i>et al.</i> (1993)	Italy	MF	14/111	Questionnaires	Wood and furniture workers	22	4.4-124	

OR, odds ratio; RR, relative risk; CI, confidence interval; M, male; F, female; NR, not reported

Table 22. Results of community-based case-control studies of sinonasal cancer: squamous-cell carcinoma

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/ RR	95% CI or <i>p</i>	Comments
Exposure to wood dust								
Hayes <i>et al.</i> (1986a)	Netherlands	M	50/195	Interviews	Exposure to wood dust High exposure	1.3* 0.5	[0.6–2.7] [0.1–2.9]	*Adjusted for age; exposure based on expert assessment of work history
Olsen & Asnaes (1986)	Denmark	M	215/2465	Linkage with national pension fund records	Definite exposure to wood dust ≥ 10 years since first exposure	1.3 1.3	0.6–2.8 0.5–3.6	Exposure based on expert assessment of work history; adjusted for exposure to formaldehyde
Vaughan (1989); Vaughan & Davis (1991)	USA	MF	27/552	Interviews	Forestry/logging Duration ≥ 10 years Woodworking machine operator Duration ≥ 10 years Any wood-related occupation Exposures lagged by 15 years Duration ≥ 10 years after lagging by 15 years	1.8 11 7.5 29 2.4 3.1 7.3	0.4–7.2 1.5–37 0.8–6.7 1.0–9.0 1.4–34	Primary exposure to softwood; all results adjusted for age, sex, smoking and alcohol intake
Luce <i>et al.</i> (1991, 1992,1993); Leclerc <i>et al.</i> (1994)	France	M	59/320	Interviews	Loggers Duration >15 years Cabinet-makers Duration >15 years Woodworking machine operators Duration >15 years Carpenters, joiners Duration >15 years Carpenter, joiner employed in wood manufacture Duration >15 years Exposure to wood dust Probable or definite low exposure Probable or definite medium-high exposure	2.9 3.9 1.6 3.4 1.2 0.0 1.6 1.9 2.3 8.1 1.4 1.0	0.8–10 0.3–56 0.3–8.9 0.5–22 0.2–6.6 0.5–5.1 0.4–9.0 0.6–9.0 1.3–50 0.6–3.0 0.4–2.6	All results age-adjusted Exposure based on expert assessment of work history

Table 22 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/ RR	95% CI or <i>p</i>	Comments
	France (contd)				Hardwood dust (incl. mixed) First exposed < 1946	1.4 2.2	1.0-4.8	14/58 exposed cases/controls No trend with duration/intensity
					Softwood dust (incl. mixed) First exposed < 1946	1.7 2.5	1.1-6.0	13/54 exposed cases/controls No trend with duration/intensity
Occupational group								
Brinton <i>et al.</i> (1984)	USA	M	53/183	Interviews	Furniture industry Lumber industry Carpentry Any wood-related job	0.3 1.1 1.0 0.8		1 exposed case 12 exposed cases 5 exposed cases 22 exposed cases
Fukuda <i>et al.</i> (1987); Fukuda & Shibata (1988, 1990)	Japan	MF	169/338	Questionnaires	Woodworkers (men) Woodworkers (women) Duration of woodworking (men) 2-11 years 12-29 years 29-55 years	2.9 2.0 1.1 2.5 4.2	1.5-5.6 0.3-14	Cases were maxillary sinus only; matched on age, sex and residence Significant trend (<i>p</i> < 0.05): 8/16 exposed cases/controls 10/9 exposed cases/controls 11/6 exposed cases/controls
Shimizu <i>et al.</i> (1989)	Japan	M	45/90	Questionnaires	Woodworking or joinery Sanding or lathing	2.1 7.5	0.8-5.3 1.5-39	Matched on age and sex
Comba <i>et al.</i> (1992a)	Italy	M	25/184	Interviews and questionnaires	Woodworkers (men)	1.7	[0.3-9.2]	
Magnani <i>et al.</i> (1993)	Italy	MF	11/111	Questionnaires	Wood and furniture workers	0.9	0.4-8.3	

OR, odds ratio; RR, relative risk; C I, confidence interval; M, male; F, female

workers, furniture makers, other wood product workers and carpenters. The jobs were classified with regard to the level of exposure to wood dust on the basis of an ad-hoc job-exposure matrix. Logistic regression was applied, with control for age and study. No association was seen between tobacco smoking and exposure to wood dust. A high risk for adenocarcinoma in men was associated with employment in wood-related occupations (odds ratio, 14; 95% CI, 9.0–20); no excess risk was found for squamous-cell carcinoma in men (0.8; 0.6–1.1). The corresponding odds ratios for women were 2.8 (0.8–10) and 1.2 (0.5–3.1). When subjects were categorized according to exposure to wood dust, a clear exposure-response relationship was found for adenocarcinoma in men, but not for squamous-cell carcinoma in men or for either histological type in women (Table 23). Similarly, an association with duration of employment in wood-related jobs or duration of moderate or high exposure to wood dust was found only with adenocarcinoma in men. The overall relative risks for adenocarcinoma showed some heterogeneity among the studies included in the re-analysis, with particularly high risks for adenocarcinoma in European countries other than Sweden (Table 24). [This finding may suggest variability in the type or intensity of exposure in different countries. The Working Group noted that elevated risks were found for highly exposed individuals in Sweden and the United States, however, although these were based on small numbers.]

Table 23. Results of pooled analysis of studies on exposure to wood dust: cases of adeno- and squamous-cell carcinoma

Sex	Exposure to wood dust	No. of exposed controls	Adenocarcinoma		Squamous-cell carcinoma	
			No. of exposed cases	Odds ratio	No. of exposed cases	Odds ratio
Men	Low	83	1	0.6	6	0.5
	Moderate	402	14	3.1*	42	1.0
	High	82	104	46*	11	0.8
Women	Low	11	2	7.7*	2	1.5
	Moderate	10	0	0	2	4.5
	High	6	0	0	2	1.6

From Demers *et al.* (1995)

* $p < 0.05$

Table 24. Odds ratios for adenocarcinoma by individual study included in a pooled re-analysis of studies of exposure to wood dust

Reference	Any exposure		High exposure	
	No. of exposed cases/controls	Odds ratio	No. of exposed cases/controls	Odds ratio
Zheng <i>et al.</i> (1992)	0/12	0.0	0/1	0.0
Luce <i>et al.</i> (1991, 1992, 1993)	79/45	161	69/15	516
Bolm-Audorff <i>et al.</i> (1989, 1990)	2/1	64	1/0	∞
Comba <i>et al.</i> (1992a)	8/23	12	7/11	23
Comba <i>et al.</i> (1992b)	2/2	32	2/1	50
Magnani <i>et al.</i> (1989, 1993)	5/8	15	5/2	55
Merler <i>et al.</i> (1986)	1/4	0.5	1/0	∞
Hayes <i>et al.</i> (1986a,b, 1987)	17/35	13	16/13	36
Hardell <i>et al.</i> (1982)	1/277	0.5	1/22	6.1
Brinton <i>et al.</i> (1984)	4/58	0.9	2/9	3.0
Mack & Preston-Martin (1995)	0/12	0.0	0/2	0.0
Vaughan (1989); Vaughan & Davis (1991)	0/98	0.0	0/6	0.0

From Demers *et al.* (1995); odds ratios adjusted for age and study

2.4.2 Cancers of other parts of the respiratory system

(a) Nasopharyngeal cancer

(i) Exposure to wood dust

Armstrong *et al.* (1983) conducted a study of 100 Chinese patients (65 men and 35 women) diagnosed with nasopharyngeal cancer between 1973 and 1980 and treated at the Institute for Radiotherapy at the General Hospital of Kuala Lumpur (the only hospital offering this treatment for nasopharyngeal cancer in Malaysia). A matched neighbourhood control of the same sex and of similar age was selected for each case. Both cases and controls had lived in the study region for at least five years. Interviews were used to collect information on occupational and other exposure. A matched analysis was performed. The relative risk reported for exposure to wood and sawdust was 2.2 ($p < 0.08$, one-sided).

In the study of Olsen *et al.* (1984) described on p. 109, there were 266 cases of nasopharyngeal carcinoma (excluding sarcomas). A relative risk of 0.4 (95% CI, 0.2–1.0) was observed among men with definite exposure to wood dust. Olsen and Asnaes (1986) further evaluated this data set by histologically confirmed sub-group; results were not presented for nasopharyngeal cancer, but the authors stated that there was no association with exposure to wood dust.

In the study of Vaughan (1989), described on p. 110, 21 people with nasopharyngeal cancer were interviewed. An excess risk for nasopharyngeal cancer was reported for carpenters (odds ratio, 3.3; 95% CI, 0.8–13) in any employment; the risk increased to 4.5 (1.1–19) after exclusion of the last 15 years. Vaughan and Davis (1991) later categorized these cases according to exposure to wood dust. The odds ratio for nasopharyngeal cancer associated with employment in any wood-related occupation was 1.2 (0.2–4.6) and increased to 4.2 (0.4–27) when only exposure for 10 or more years after an induction period of 15 years was considered. The authors stated that the case patients were predominantly exposed to softwood dust.

In the study of Bolm-Audorff *et al.* (1989, 1990), described on p. 110, 12 of the 66 cases were nasopharyngeal cancers. One case of undifferentiated carcinoma of the nasopharynx was associated with exposure to wood dust (oak and beech) for 24 years, while two controls had been exposed to wood dust (species not identified), one of them for fewer than five years.

The etiology of nasopharyngeal carcinoma was studied in the Philippines, in investigations addressing both viral (Hildesheim *et al.*, 1992) and non-viral (West *et al.*, 1993) risk factors. There were 104 people with histologically confirmed nasopharyngeal carcinoma in the Philippines General Hospital and 104 hospital controls (matched on sex, age and private versus public ward) and 101 community controls (matched on sex, age and neighbourhood). The occupational history of each subject was collected by personal interview. The occupations of carpenter, lumberman, raftsman, woodchopper, farm manager and farmer were considered to entail exposure to wood dust on the basis of an assessment by an industrial hygienist who was unaware of the case or control status of the subject. A matched analysis was conducted. The relative risk for exposure fewer than 35 years before diagnosis was 1.3, and that for exposure 35 or more years before first diagnosis was 2.1. [The Working Group noted that the authors did not control for the presence of Epstein–Barr virus antibodies, which showed a strong association with nasopharyngeal cancer (odds ratio, 21) in the study of Hildesheim *et al.* (1992).]

(ii) *Occupational group*

In the study of Hardell *et al.* (1982) described on p. 113, there were 27 male patients with nasopharyngeal cancer; five were squamous-cell carcinoma, 20 were anaplastic carcinoma and two were adenoid cystic carcinoma. A crude relative risk of 1.3 [95% CI, 0.6–2.9] was reported for employment as a carpenter, cabinet-maker or sawmill worker, with nine exposed cases and 151 exposed controls. [The comparability of the ages of cases and controls was unknown.]. The authors noted that little hardwood is used for furniture making in northern Sweden.

In the study of Ng (1986), described on p. 114, there were 224 cases of nasopharyngeal cancer: 112 squamous-cell, 102 anaplastic and 10 of unknown histology; 226 controls had other malignancies. Among people in the two wood-related occupational categories, furniture makers and woodworkers had five nasopharyngeal cancers and one other malignancy, and construction carpenters had three nasopharyngeal cancers and no other malignancies. No odds ratios or other estimates of relative risk were presented.

In the study of Kawachi *et al.* (1989), described on p. 115, excess risks were observed for nasopharyngeal cancer among woodworkers (odds ratio, 2.5; 95% CI, 0.9–6.6), forestry and

logging workers (6.0; 1.0–28) and carpenters (2.5; 0.6–8.5), after adjustment for age. No cases were observed among sawmill workers, pulp and paper workers or cabinet-makers.

Sriamporn *et al.* (1992) performed a case-control study in North-east Thailand of 120 patients with histologically confirmed nasopharyngeal cancer diagnosed between 1987 and 1990, who were undergoing radiation therapy at the only hospital offering such therapy in the area. Sixty-nine (57.5%) of the cases were squamous-cell carcinoma, and the remaining 51 were undifferentiated carcinomas. The 120 controls were patients admitted to the same hospital for diseases other than cancer and respiratory disease, who were matched to the cases on sex and age. Occupational histories were collected by questionnaire. The results were adjusted for age, sex, smoking, consumption of alcohol and salted fish, education and area of residence. Excess risks were reported for wood cutting, excluding agriculture (odds ratio, 4.1; 95% CI, 0.8–22) and for wood cutting and farming combined (8.0; 2.3–28).

Studies on nasopharyngeal cancer are summarized in Table 25.

(b) *Pharyngeal cancer other than cancer of the nasopharynx*

Elwood *et al.* (1984) reported the results of a case-control study of 374 patients with primary epithelial cancers of the oral cavity, pharynx (excluding nasopharynx) and larynx in British Columbia, Canada. The study included 44 oropharyngeal cancers, 38 hypopharyngeal cancers and five pharyngeal cancers at 'other' subsites. Controls were 374 patients with selected other cancers, who were matched to the cases on age and sex. Lifetime occupational histories were collected by interview in 1977–80. No quantitative results for wood-related exposures were reported, but the authors stated that 'exposure to wood dust was assessed and analysed in more detail, examining nature, intensity, and duration of exposure, but no regular or significant trends were seen'.

In the study of Vaughan (1989), described on p. 110, 183 people with cancer of the oro- or hypopharynx and 552 controls were interviewed. Excess risks were reported for carpenters, construction carpenters and machine operators in the wood industry after exclusion of exposure during the previous 15 years. Vaughan and Davis (1991) later categorized these cases according to exposure to wood dust. The odds ratio for pharyngeal cancer associated with employment in any wood-related occupation was 0.5 (95% CI, 0.2–1.2); it was 1.5 (0.4–5.5) when only exposure for 10 or more years after an induction period of 15 years was considered.

In the study of Haguenoer *et al.* (1990), described on p. 116, there were 114 histologically confirmed cases of oro- and hypopharyngeal cancers (no nasopharyngeal cancers were included) among men treated during the first semester of 1983. A matched analysis was conducted. A history of employment in woodworking occupations was associated with an increased risk for all cancers of the upper respiratory tract combined (odds ratio, 3.5; 95% CI, 1.2–10). Four case patients with oro- and hypopharyngeal cancers and one matched control had been employed as woodworkers, but no odds ratio was presented.

Maier *et al.* (1991) conducted a case-control study in the Heidelberg and Giessen areas of Germany of 200 male patients with squamous-cell carcinomas of the mouth, oropharynx, hypopharynx and larynx that had been diagnosed or treated at an eye, nose and throat clinic during 1987 and 1988. For each case, four age-matched male controls were selected from among

Table 25. Community-based case-control studies on cancer of the nasopharynx

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Exposure to wood dust								
Armstrong <i>et al.</i> (1983)	Malaysia	MF	100/100	Interviews	Exposed to wood/saw dust	2.2	<i>p</i> (one-sided) = 0.08	Self-reported exposure; matched on age and sex; ethnic Chinese
Olsen <i>et al.</i> (1984)	Denmark	MF	266/2465	Linkage with national pension fund records	Definite exposure to wood dust (men)	0.4	0.2–1.0	Exposure based on expert assessment of work history
Vaughan (1989); Vaughan & Davis (1991)	USA	MF	21/552	Interviews	Carpenters Construction carpenters Any wood-related occupation Duration ≥ 10 years	4.5 6.8 1.5 4.2	1.1–19 1.6–28 0.2–6.1 0.4–27	Only squamous-cell cancers; exposure primarily to softwood. Exposures lagged by 15 years. Results adjusted for age, sex, smoking and alcohol intake
Bolm-Audorff <i>et al.</i> (1990)	Germany	MF	12/66	Interviews	Exposed to wood dust			1/2 exposed case/controls
West <i>et al.</i> (1993)	Philippines	MF	104/205	Interviews	Wood dust-exposed occupation < 35 years since first employment ≥ 35 years since first employment	1.3 2.1		Exposed occupations were carpenters, farm managers, farmers, lumbermen, raftsmen and wood-choppers. Matched on sex and age
Occupational group								
Hardell <i>et al.</i> (1982)	Sweden	M	27/541	Questionnaires	Carpenter, cabinet-maker or sawmill worker	1.3	[0.6–2.9]	9/151 exposed cases/controls
Ng <i>et al.</i> (1986)	Hong Kong	MF	224/226	Medical records	Furniture makers, woodworkers Construction carpenters	[5.0]		5/1 exposed cases/control 3/0 exposed cases/control

Table 25 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Occupational group (contd)								
Kawachi <i>et al.</i> (1989)	New Zealand	M	NR/ ~19 000	Tumour registry records	Woodworkers	2.5	0.9–6.6	Adjusted for age 0/108 exposed case/controls 0/91 exposed case/controls
					Foresters, loggers	6.0	1.0–28	
					Sawmill, pulp and paper workers			
					Cabinet-makers			
				Carpenters	2.5	0.6–8.5		
Sriamporn <i>et al.</i> (1992)	Thailand	MF	120/120	Interview	Wood cutting, not agriculture	4.1	0.8–22	Adjusted for age, sex, smoking, consumption of alcohol and salted fish, education and area of residence
					Wood cutting and agriculture	8.0	2.3–28	

NR, not reported; OR, odds ratio; RR, relative risk; CI, confidence interval; M, male; F, female

non-cancer patients attending the same clinic and a university clinic. Information on occupational and other exposures was collected from questionnaires. An elevated relative risk associated with self-reported exposure to wood dust was found for cancers at all upper aerodigestive tract sites combined (2.2; 95% CI, 1.0–4.9), but no site-specific results were reported.

Merletti *et al.* (1991) reported the results of a case-control study on occupation and cancer of the oral cavity and oropharynx in Turin, Italy. The cases were cancers of the oropharynx ($n = 12$) and oral cavity ($n = 74$) diagnosed among male residents of Turin between 1982 and 1984. The 385 controls were selected from a random sample of city residents interviewed as part of a study on laryngeal cancer. Occupational histories were collected by interview. Results were presented only for all cases combined (oropharynx and oral cavity). The odds ratios were adjusted for age, education, area of birth, tobacco smoking and alcohol drinking. The odds ratio for previous employment as a cabinet-maker or related woodworker was 1.2 (95% CI, 0.4–3.9); that for employment in any wood industry was 0.9 (0.3–3.0), and that for employment in the wood furniture industry was 1.4 (0.4–5.5).

Huebner *et al.* (1992) performed a case-control study of oral and pharyngeal (excluding nasopharyngeal) cancer among residents of four areas of the United States: Los Angeles, metropolitan Atlanta, two counties south of San Francisco, and New Jersey. Cases were identified from population-based tumour registries, to give 1114 cases diagnosed between 1 January 1984 and 31 March 1985. Controls (1268) were obtained through random-digit dialling (aged 18–64 years) and Health Care Financing Administration files (aged 65–79 years) and were frequency matched to controls on sex, race, age and study area. Information on occupation and exposure was collected at interviews. The results were adjusted for age, race, smoking, alcohol consumption and study location. The relative risks for pharyngeal cancers in men were 2.2 (95% CI, 1.0–4.7) for previous employment in the furniture/fixture industry and 2.3 (0.7–7.4) for work with woodworking machines.

Studies on oropharyngeal and hypopharyngeal cancers are summarized in Table 26.

(c) *Laryngeal cancer*

(i) *Exposure to wood dust*

Maier *et al.* (1992) reported on a case-control study of laryngeal cancer in Germany. The cases were histologically confirmed squamous-cell carcinomas of the larynx in 164 male patients who had attended the department of Otorhinolaryngology-Head and Neck Surgery, University of Heidelberg, for treatment or follow-up examinations during 1988–89. Controls were 656 males with no known cancer, who were selected randomly from two out-patient clinics in Heidelberg and matched to the cases 4:1 by age and residential area. All subjects were interviewed on life style, education and occupational history and exposures. The percentages of cases and controls, respectively, who were exposed at least once in a week during 10 years or more to different wood dusts were as follows: wood dust in general, 12.6% and 8.3% ($p < 0.08$); beech, oak, 5.8% and 6.1% ($p < 0.7$); pine, 12.6% and 7.5% ($p < 0.06$); 'precious wood', 0.8% and 3.5% ($p < 0.3$); and exotic wood, 0% and 2.1% ($p < 0.3$). For exposure to pinewood dust, the relative risks, p values and undefined CIs, adjusted for alcohol consumption and tobacco

Table 26. Community-based case-control studies on cancer of the oro- and hypopharynx in occupational groups

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Elwood <i>et al.</i> (1984)	Canada	MF	87/374	Interviews	Unspecified exposure to wood			'No regular or significant trends'
Vaughan (1989), Vaughan & Davis (1991)	USA	MF	183/552	Interviews	All carpenters	1.3	0.5-3.4	Only squamous-cell cancers; all exposures lagged by 15 years
					Construction carpenters	1.8	0.7-4.8	
					Wood machine operator	2.8	0.3-2.4	
					Any wood-related occupation Duration ≥ 10 years	0.5 1.5	0.2-1.2 0.4-5.5	
Haguenoer <i>et al.</i> (1990)	France	M	114/228	Interviews	Woodworkers			4/1 exposed cases/control. Only jobs held for at least 15 years evaluated; matched on sex, age, ethnic group, area of residence, smoking and alcohol drinking
Huebner <i>et al.</i> (1992)	USA	MF	1114/1268	Interviews	Furniture/fixture industry	2.2	1.0-4.7	For subset of male cases of pharyngeal cancer only
					Woodworking machines	2.8	0.7-7.4	

OR, odds ratio; RR, relative risk; CI, confidence interval; M, male; F, female

smoking, were as follows: all laryngeal cancers, 1.9 ($p = 0.05$; CI, 0.9–3.7); glottal cancers, 3.2 ($p = 0.03$; CI, 1.1–9.0); supraglottal cancers, 1.3 ($p = 0.6$; CI, 0.4–3.5). The mean time between the beginning of exposure to pinewood dust and the expression of laryngeal cancer was 39.7 years. [The Working Group noted the incomplete documentation of the composition of the control group, statistical methods and results.]

Zheng *et al.* (1992b) reported on a population-based case-control study of laryngeal cancer in Shanghai, China. A total of 263 residents of urban Shanghai, aged 20–75, in whom laryngeal cancer was newly diagnosed in 1988–90, were identified from a population-based cancer registry in Shanghai; 201 (76.4%) were interviewed. Controls were randomly selected from the urban Shanghai population and frequency matched by sex and age to all cases of oral, pharyngeal, laryngeal and nasal cancers reported to the Shanghai Cancer Registry during 1985–86. Of the 414 controls interviewed, 12% were second controls. The interview covered demographic data, tobacco smoking, alcohol drinking, dietary habits and occupational history and exposures. Adjusted odds ratios were calculated by stratification and unconditional logistic regression. Self-reported occupational exposure to wood dust among males was associated with an odds ratio of 1.4 (95% CI, 0.6–3.2), adjusted for age and smoking.

(ii) *Occupational group*

A case-control study of laryngeal cancer was reported by Wynder *et al.* (1976) in the United States which included 258 men and 56 women with a histologically confirmed laryngeal cancer (ICD 161.0,1), who had been admitted to hospitals in New York City, Los Angeles, Houston, Birmingham, Miami and New Orleans during 1970–73. Controls were 516 hospitalized men and 168 women (without a current diagnosis of tobacco- or alcohol-related disease and with no history of cirrhosis, stroke, gastric ulcers or myocardial infarct), who were individually matched to the cases by year of interview, hospital status and age at diagnosis. Occupational exposure to wood dust was reported for 22% of the male cases and 1.2% of controls ($p < 0.05$). Of the current smokers, equal proportions of cases and controls had been exposed to wood dust.

Occupational risks for laryngeal cancer were examined in the Third National Cancer Survey data (Flanders & Rothman, 1982). Cancer cases occurring during 1969–71 were identified in records from seven cities and two states in the United States, and a 10% probability sample of the subjects was interviewed. Ninety men with laryngeal cancer represented the cases, and 933 men with cancers at other sites, excluding oesophagus, stomach, small intestine, colon, pancreas, liver, bladder, kidney, lung, bronchus, oral cavity and pharynx, constituted the control group. Information on age, alcohol use, tobacco use and industrial and occupational categories (longest and second-longest held job) was obtained at interview and used in the analysis. The relative risks, adjusted for age, alcohol use, tobacco smoking and race, were 3.5 [95% CI, 0.6–19] for work in the lumber industry and 1.3 [0.3–6.6] for carpenters.

A hospital-based case-control study in New Haven, Connecticut (United States) (Zagraniski *et al.*, 1986), addressed occupational risk factors for laryngeal cancer. The cases were histologically confirmed primary squamous-cell carcinomas of the larynx (ICD 8 US adapted: 161) diagnosed in 1975–80 among white male residents of Connecticut alive in 1980–

81 and treated in one of two New Haven hospitals. Controls were white male general surgery patients with no prior diagnosis of cancer or respiratory disease, who were individually matched to the cases by hospital, calendar year of admission, decade of birth, county of residence, smoking status and type of tobacco used. The interview covered occupational history, including specific exposures, medical history, tobacco and alcohol use and demographic and home environmental data. A total of 148 cases were identified; 22 were not invited for interview because of death, move out of the state, loss to follow-up and other reasons. No proxy data were sought. Of the remaining 126 cases, 14.3% refused to be interviewed. Thus, 12.7% completed a telephone interview and 73.0% completed a personal interview. Of the 317 controls, 57.1% were interviewed personally. Any employment as a woodworker was associated with an odds ratio of 2.5 (95% CI, 0.5–13); the odds ratio for carpenters was 1.1 (0.6–2.0). These figures were adjusted for lifetime exposure to tobacco and alcohol and were based on a conditional logistic model with 87 cases and 153 controls. [The Working Group noted the low response rate among the controls.]

Morris Brown *et al.* (1988) reported on a case-control study of laryngeal cancer conducted in six counties of the Gulf Coast of Texas, United States. Cases were primary laryngeal cancers (ICD9: 161.X, 231.0) diagnosed in white males aged 30–79, identified through tumour registers and the records of 56 hospitals. During 1975–80, 220 living and 83 dead case patients were identified. Controls were a sample of white males resident in the six-county area, who were frequency matched to the cases by age, vital status, ethnic group and county of residence; they were identified from Texas Department of Health mortality tapes, drivers' licence records and Medicare records. Interviews including job histories were completed for 153 living case patients (69.5%) and close relatives of 56 dead case patients (67.5%). Exclusions on histological grounds resulted in 183 case patients (136 living, 47 dead). There were 250 controls (179 alive, 71 dead); the response rates were 62.8% for the dead controls, 60.9% for those identified from drivers' licence lists and 85.7% for those identified from Medicare records. The odds ratio for any employment as a woodworker or furniture maker, adjusted for tobacco smoking and alcohol drinking in the logistic model, was 8.1 (95% CI, 0.95–69; 7 exposed cases); that for employment as a carpenter was 1.7 (0.8–3.5; 19 exposed cases).

Bravo *et al.* (1990) reported on a case-control study of laryngeal cancer conducted in Spain. The cases were histologically confirmed epidermoid carcinomas of the larynx diagnosed in 85 patients (83 men) at La Paz Hospital, Madrid, during 1985–87. Twenty-six eligible case subjects were not included in the study because of death, having moved out of the country, refusal or loss. Controls were a random sample of 170 patients from the same hospital, excluding those with respiratory diseases or alcoholic cirrhosis; they were 'stratified' with respect to the cases according to sex, age and admission month. Personal interviews were conducted with all subjects, except for 15 case patients for whom a relative was interviewed. Occupational exposure to wood dust was associated with a crude odds ratio of 0.70. The mean duration of exposure to wood dust was 25 years for the exposed cases and 26 years for the exposed controls. [The Working Group noted the incomplete description of the exposure assessment and the crude statistical analysis].

In their study of exposure to wood dust and cancer of the upper respiratory tract, described on p. 110, Vaughan and Davis (1991) compared 234 cases of squamous-cell cancer of the larynx with 547 controls. They observed no excess risk for ever having been employed in a wood-exposed occupation (odds ratio, 1.0; 95% CI, 0.5–1.9), after adjustment for potential confounders. An elevated risk was observed for employment for 10 or more years, when allowing for a 15-year induction period (2.5; 0.6–10).

In a hospital-based case-control study in the United States (Muscat & Wynder, 1992), the associations between laryngeal cancer and exposure to tobacco, alcohol and occupational factors were examined in 194 white men with histologically confirmed primary laryngeal cancer selected from the records of the Memorial Sloan-Kettering Cancer Center and seven other hospitals in New York, Illinois, Michigan and Pennsylvania and interviewed in 1985–90. Controls were 184 men matched by hospital, age and year of interview, who included patients with gastrointestinal cancers, prostate cancers or lymphomas and bone, spinal and other 'non-neoplastic conditions'. Eighty-nine percent of the eligible case patients and controls agreed to be interviewed; no proxies were used. Self-reported occupational or recreational exposure to wood dust for at least 8 h per week for at least one year was associated with an odds ratio of 1.7 (95% CI, 0.7–4.6), adjusted for age, education, tobacco smoking, alcohol drinking and relative body weight.

A population-based case-control study in western Washington State (United States) (Wortley *et al.*, 1992) addressed occupational risk factors for laryngeal cancer. Cases were laryngeal cancers (ICD0: 161.0–161.9) diagnosed in 1983–87 in 291 patients who were identified through the cancer surveillance system (population-based cancer registry covering 13 counties in western Washington) of the Fred Hutchinson Cancer Research Center, Seattle; 235 (80.8%) were successfully interviewed. The closest next-of-kin was interviewed if the case was dead (17 surrogate interviews). Controls were men identified by random-digit dialling, who were frequency matched to the cases in categories of age and sex; 547 (80%) of the eligible controls were successfully interviewed. Lifetime occupational histories were coded according to the 1980 United States census codes for occupation and industry. Odds ratios were derived from a multiple logistic regression model and adjusted for smoking, alcohol drinking, age and education. The odds ratio for any employment as a woodworking machine operator, lagged by 10 years, was 0.4 (95% CI, 0.1–1.3); for fewer than 10 years, the odds ratio was 0.4, and for at least 10 years, it was 2.3 (CIs not given; trend $p = 0.36$). Five cases and 18 controls had been employed as woodworking machine operators. [The Working Group noted that although the number of controls in this study (547) was similar to that in the study of Vaughan and Davis (1991), they used separate control series.]

The studies on laryngeal cancer are summarized in Table 27.

(d) *Lung cancer*

(i) *Exposure to wood dust*

Blot *et al.* (1982) reported on occupational determinants of lung cancer in an area of northern Florida (United States) with exceptionally high rates of lung cancer. Interviews were conducted with 181 patients with lung cancer, 342 hospital controls (1978–79), 217 next-of-kin

Table 27. Community-based case-control studies on cancer of the larynx

Reference	Country	Sex	Cases/ Controls	Source of controls on exposure	Exposure to which relative information	OR/RR risk applies	95% CI or <i>p</i>	Comments
Exposure to wood dust								
Maier <i>et al.</i> (1992)	Germany	M	164/656	Interview	Wood dust		<i>p</i> < 0.08	12.6% of cases, 8.3% of controls; incomplete documentation
					Pinewood dust	1.9	<i>p</i> = 0.05	
					Pinewood dust, glottal cancers only	3.2	<i>p</i> = 0.03	
Zheng <i>et al.</i> (1992b)	China	M	177/269	Interview	Wood dust (self-reported)	1.4	0.6–3.2	Adjusted for age, smoking
Occupational group								
Wynder <i>et al.</i> (1976)	USA	M	258/516	Interview	Wood dust		<i>p</i> < 0.05	Crude analysis; 22% of cases, 1.2% of controls
Flanders & Rothman (1982)	USA	M	90/933	Interview	Lumber industry as major employer	3.5	[0.6–19]	Adjusted for age, race, smoking, alcohol consumption
					Carpenter	1.3	[0.3–6.6]	
Zagraniski <i>et al.</i> (1986)	USA	M	87/153	Interview	Woodworker (ever)	2.5	0.5–13	Adjusted for smoking, alcohol consumption; low response in controls
					Carpenter (ever)	1.1	0.6–2.0	
Morris Brown <i>et al.</i> (1988)	USA	M	183/250	Interview	Woodworker or furniture maker (ever)	8.1	1.0–69	Adjusted for smoking, alcohol consumption
					Carpenter	1.7	0.8–3.5	
Bravo <i>et al.</i> (1990)	Spain	MF	85/170	Interview	Wood dust	0.7		Incomplete documentation

WOOD DUST

Table 27 (contd)

Reference	Country	Sex	Cases/	Source of controls on exposure	Exposure to which relative information	OR/RR risk applies	95% CI or <i>p</i>	Comments
Occupational group (contd)								
Vaughan & Davis (1991)	USA	MF	234/547	Interview	Any wood dust >10 years with 15-year induction	1.0 2.5	0.5-1.9 0.6-10	Squamous-cell only; exposure predominantly to softwood
Muscat & Wynder (1992)	USA	M	194/184	Interview	Wood dust	1.7	0.7-4.6	Adjusted for age, education, smoking, alcohol consumption, relative body weight
Wortley <i>et al.</i> (1992)	USA	MF	235/547	Interview	Woodworking machine operator > 10 years	2.3	<i>p</i> (trend) = 0.36	Adjusted for age, education, smoking, alcohol consumption

OR, odds ratio; RR, relative risk; CI, confidence interval; M, male; F, female

of dead lung cancer patients and 217 dead control subjects. The controls were selected from among hospital patients with diagnoses of or deaths from diseases other than lung cancer or chronic respiratory disease. The response rates were 86% for cases and 83% for controls. The final study group consisted of 321 cases and 434 controls. An excess relative risk for lung cancer (1.7; 95% CI, 1.0–2.7) was reported among people ever employed in the lumber or wood industries, after adjustment for tobacco smoking. A gradient was suggested with duration of employment in the lumber or wood industry: the relative risk was 1.3 among those employed 1–9 years and 1.6 among those employed for more than 10 years; both relative risks were calculated in comparison with those for people never employed in the wood industry. The excess was concentrated among those exposed to wood dust mainly in sawmills (1.9); the excess associated with exposure to wood dust was higher (3.4; 10 exposed cases) for small-cell carcinoma than for other cell types.

A hospital-based case-control study was conducted in an area of Louisiana (United States) with a high rate of lung cancer (Correa *et al.*, 1984). 'Current' primary lung cancers were identified from admission and pathology records of all the major hospitals in southern Louisiana, in one central Louisiana parish and two northern Louisiana parishes, except for the city of New Orleans, where the study was limited to four large hospitals. [The period of case ascertainment was not given.] A control subject was selected for each case from the same hospital and individually matched by race, sex and age. Patients whose main diagnosis was emphysema, chronic bronchitis, chronic obstructive pulmonary disease or cancer of the larynx, oral cavity, oesophagus or urinary bladder were excluded. Acceptable personal interviews were conducted with 1338 (76%) cases and 1393 (89%) controls. The questions covered occupation, residence, diet, smoking and drinking habits, health, water supply and other related items. The odds ratios (unconditional) were adjusted for smoking. For all occupations, only white male workers who had ever been employed in the 'forestry' category (most of them sawmill workers) had a significantly elevated odds ratio (1.7). Exposure to wood dust was associated with an odds ratio for lung cancer of 1.4 ($p < 0.05$); of 45 other suspected occupational exposures, only mineral oil mist was also significantly associated with lung cancer.

A nested case-control study of lung cancer in 19 608 male employees of a chemical plant in Texas (United States) (Bond *et al.*, 1986), was designed to examine the associations between lung cancer and a number of occupational exposures. A total of 308 lung cancer deaths were recorded during 1944–80 on death certificates as cancer of the bronchus, lung or site unspecified within the respiratory system as the underlying cause, as a contributing cause or as 'other significant conditions'. Two control groups, one a dead and the other a living series, were individually matched to the lung cancer patients. Work histories were grouped into 50 work areas of homogeneous exposures. Chemical and physical exposure profiles were developed by an industrial hygienist for each case and each control. In comparison with pooled controls, the odds ratio for any (as opposed to no) exposure to wood dust was 1.1 (95% CI, 0.72–1.8). With a 15-year lag, it was 1.3 (0.78–2.2); for low exposure to wood dust, 3.9 (1.1–14); for moderate exposure, 0.91 (0.39–2.1); and for high exposure, 0.99 (0.56–1.8). [The Working Group noted that this was a study of a multi-product chemical plant where there were numerous exposures

and where exposure to wood dust would be minimal in comparison with that in wood-related industries.]

Kjuus *et al.* (1986) used data from interviews with 176 men admitted to two hospitals in Telemark and Vestfold, Norway, for lung cancer during 1979–83 and with 176 age-matched control subjects admitted during the same period to the same hospitals. Patients with conditions that would have precluded employment in heavy industry, poor general health, obvious mental conditions or chronic obstructive lung disease were excluded as controls. One case and two potential controls refused the interview. Woodwork as the main lifetime occupation was associated with an odds ratio of 0.7 (95% CI, 0.2–2.3), adjusted for cigarette smoking. The odds ratio for exposure to wood dust, adjusted for a number of confounders, was 0.5, representing a significant deficit.

A population-based multi-site case–control study of cancer addressed associations between occupational factors and cancer (Siemiatycki *et al.*, 1986; Siemiatycki, 1991). A total of 3730 histologically confirmed cases of cancer at 19 sites, newly diagnosed among male residents, aged 35–70, of Montréal, Canada, in 19 major hospitals during 1979–85 were identified, and the patients were interviewed for detailed lifetime job histories and potential confounders. The response rate was 81.5%. A team of chemists and hygienists examined the questionnaires and translated each job into a list of potential exposures with the help of a checklist of some 300 occupational exposures. Odds ratios for each cancer site were calculated in comparison with other cancers and adjusted for smoking and other factors, including other occupational exposures, and were presented for any exposure and for substantial exposure (at least 10 years' duration after the first five years). There were 1082 patients with lung cancer, and 857 (79%) responded. The odds ratio for any exposure to wood dust was 1.2 [95% CI, 1.0–1.5]; for substantial exposure, it was 1.3 [0.9–1.8]. When the analysis was restricted to French-Canadian subjects, the odds ratios for oat-cell lung cancer were 1.3 [0.9–2.0] for any exposure and 1.6 [0.9–2.8] for substantial exposure.

A population-based case–control study was conducted in New Mexico, United States (Lerchen *et al.*, 1987) of 506 white residents of New Mexico (333 males and 173 females), aged 25–84 years, with primary lung cancer other than bronchioalveolar carcinoma, which was diagnosed in 1980–82 and registered by the New Mexico Tumor Registry. A total of 771 controls (499 males and 272 females) were identified through randomly selected residential telephone numbers and, for people aged 65 or older, from a roster of Medicare participants. The controls were frequency matched to cases for sex, ethnic group and 10-year age category. The case subjects and controls or their next-of-kin were interviewed about smoking and occupational history, and a self-reported history of exposures to 18 specific agents was obtained. The interview rate was 89% for cases and 83% for controls. The odds ratio for ever having been employed for at least one year as a woodworker was 0.8 (95% CI, 0.3–1.7), after adjustment for age and ethnic group. [The Working Group noted that the numbers of case subjects and controls exposed to wood dust were exactly the same as those of people employed one or more years as a woodworker and assumed that these were the same individuals.]

Twenty-five major industrial titles were evaluated as risk factors for lung cancer in a population-based case–control study conducted in Shanghai, China (Levin *et al.*, 1988). The

case series consisted of all lung cancers newly diagnosed during 1984–85 among men aged 35–64 who resided in the urban areas of Shanghai. Population controls were randomly selected from the same areas from specified age categories in sampling fractions that produced similar age distributions for the cases and controls. Personal interviews were conducted with 733 surviving case subjects (88% of the total incident cases and 99% of survivors) and 760 controls. The interview concerned lifetime occupational history, smoking and other information; detailed data were obtained on every job the subject had held for at least one year since the age of 16. The employment data were classified by the industrial and occupational headings defined for the 1982 Chinese population census. Any employment in furniture manufacture was associated with an odds ratio of 1.3 (95% CI, 0.5–3.4), adjusted for smoking and age. The adjusted odds ratio for any employment in timber processing or as a wood, bamboo, hemp, rattan, palm or straw products maker was 1.2 (0.7–2.2). Self-reported exposure to wood dust was associated with a significantly increased odds ratio of 1.7 (1.0–2.7), in contrast to people who reported no exposure to dust, smoke or fumes in the workplace. Most of the subjects who reported exposure to wood dust worked in furniture manufacture or timber production. A total of 672 female case subjects and 735 female controls were also interviewed, but the small numbers in many industrial and occupational categories precluded a detailed analysis. An increased risk, similar to that observed among men, was associated with self-reported exposure to wood dust [no odds ratio given]. The authors found only slight differences in risk by cell type for most occupational or industrial categories but did not document this statement.

(ii) *Occupational group*

Harrington *et al.* (1978) analysed data from death certificates on the occupations of 858 white men who died of lung cancer in coastal Georgia, United States, during 1961–74, and of 858 controls who died of conditions other than lung cancer, chronic respiratory disease or bladder cancer during the same period. The controls were individually matched to the cases by age at death, year of death, sex, race and county of usual residence, and matched-pairs analyses were conducted. The usual occupations were coded into major occupational and industrial categories. The relative risk for work in the wood and paper industry was 1.3 ($p > 0.05$). A significant excess relative risk for work in the wood and paper industry (3.3; $p < 0.01$) was found in small rural counties but not in the largest counties. The excess was greater among sawmill, lumber and forestry workers than among pulp and paper workers and carpenters, but no relative risks were presented. No data were available on tobacco smoking and possible industrial confounders.

In a pilot study, Esping and Axelson (1980) used data from death certificates on the occupations associated with 25 deaths from respiratory cancer (ICD [1965]: 160–163) and those of 370 controls who had died from diseases other than respiratory and digestive cancers in the small town of Mjölby, Sweden, where there was a comparatively large woodworking industry. The deaths had occurred among men 50 years of age or older during the period 1963–77. The age-adjusted rate ratio for 'exposure to woodwork' was 4.1 (95% CI, 1.6–11). The crude relative risks were 6.0 for furniture makers and 2.3 for other woodworkers. The smoking habits were not known.

A case-control study in Alameda County, California, United States (Milne *et al.*, 1983), covered 925 deaths from lung cancer (747 men) and 6420 deaths from other cancers (3130 men) that occurred among county residents over 18 years of age between 1958 and 1962. The study examined associations between lung cancer and usual occupation and industry, as recorded on the death certificate and coded by the US Bureau of Census Industrial and Occupational Classification System. The odds ratio were 0.8 for men employed in sawmills, 4.2 ($p < 0.01$) for men in furniture manufacture, 1.0 for male cabinet-makers and furniture finishers and 1.2 for carpenters.

In a hospital-based case-control study in metropolitan Florence, Italy (Buiatti *et al.*, 1985), frequency matching on smoking status was used to compare 376 people with histologically confirmed primary lung cancer (340 men) with 892 control subjects with discharge diagnoses other than lung cancer and attempted suicide (817 men). The case patients and controls had been admitted in the period 1981-83. Occupational histories were collected for all cases and controls in person, and the response rate was 100%. The odds ratio for men ever employed in woodwork, adjusted for age, smoking and place of birth, was 0.6 (95% CI, 0.3-1.1).

Coggon *et al.* (1986) reported on a case-control study of cancer of the bronchus among middle-aged men in Cleveland, Humberside and Cheshire, United Kingdom, diagnosed during 1975-80. Controls were patients with other cancers. Occupational and smoking histories were obtained from a postal questionnaire, addressed either to the patients or their next-of-kin. The overall response rate was 52% (738 cases, 2204 controls). For patients who reported ever having been employed as a woodworker, the relative risk was 1.7 (95% CI, 1.0-3.0), adjusted for age, residence, source of occupational history and smoking. The risk ratio for ever having been employed in the industrial order of timber and furniture was 1.6 ($n = 17$). The authors compared the distribution of occupations among respondents and nonrespondents, using information from hospital records, and found no evidence of bias in reporting by response category.

In a population-based case-control study in northern Sweden (Damber & Larsson, 1987), data on 589 dead male cases and two series of matched control subjects drawn from population registries (582 deceased, 453 living) were used to examine associations between the risk for lung cancer and occupation. The cases represented deaths during 1972-79 among people in whom lung cancer was diagnosed. The occupations were ascertained from a postal questionnaire addressed to living controls or to close relatives of the cases and dead controls. The response rates were 98% for cases, 96% for dead controls and 97% for living controls. At least one year of employment as a carpenter was associated with an odds ratio of 0.8 (95% CI, 0.5-1.3), adjusted for lifetime tobacco consumption, when compared with dead controls, and with an odds ratio of 0.7 (0.5-1.2) when live controls were used.

The association between occupation and the risk for lung cancer was examined in a case-control study conducted in six areas of New Jersey, United States (Schoenberg *et al.*, 1987). The cases were histologically confirmed primary cancers of the trachea, bronchus and lung diagnosed in 763 white males in 1980-81. Nine hundred white male population controls were selected from files of drivers' licences and death certificates. Interviews were completed with 429 case patients and 564 controls or their next-of-kin (334 and 336, respectively), in order to obtain demographic data and information on personal and environmental risk factors, including

smoking, diet and occupation. The response rate was 70% for the case patients and 64% for the controls. Information on industry and job title was coded by the index system used in the 1970 United States census. The risk for lung cancer was analysed for 42 job title categories and 34 job titles in specified industries, after adjustment for smoking. The risk among men who had ever been employed as furniture or fixture workers was 1.5 (95% CI, 0.76–3.0). Smoking-adjusted odds ratios for carpenters (46 cases, 55 controls) and lumber and wood products workers (16 cases, 17 controls) were 0.90–0.99.

A population-based case-control study of lung cancer and occupation was conducted in two industrialized areas of northern Italy (Ronco *et al.*, 1988) involving 126 men who had died from lung cancer between 1976 and 1980. Controls were a random sample of 384 men who had died from other causes (except chronic lung conditions and smoking-related cancers) during the same period and who were individually matched to the cases by year of death and 10-year age class. Next-of-kin were interviewed at home or by telephone with regard to the lifelong tobacco consumption and occupational histories of the cases and controls. The response rate was 77% for cases and 78% for controls. Job titles were coded by the ILO classification, and industrial activities according to the United Nations international classification of industries. While no excess risk was associated with carpentry or joinery (6 cases, 28 controls), increased odds ratios were observed for woodworkers employed in furniture and cabinet-making, with an aggregated odds ratio of 2.8 (0.93–8.4), adjusted for age, smoking and having been engaged in an occupation known or suspected to be associated with increased lung cancer risk.

Hoar Zahm *et al.* (1989) examined the associations between different histological types of lung cancer and occupations in 4431 white Missouri (United States) residents with histologically confirmed lung cancer diagnosed in 1980–85 and reported to the Missouri Cancer Registry. The 11 326 controls were all white Missouri residents with a diagnosis of any cancer except those of the lip, oral cavity, oesophagus, lung, bladder, ill-defined sites and unknown sites, during the same period. The occupation at the time of diagnosis of cancer was abstracted from the Registry files, which obtained this information from medical records. Occupation was coded according to the index system of the United States Bureau of Census, and codable information was available for 52% of the cases and 45% of the controls. The smoking history was unknown for 15% of the cases with known occupation and for 37% of the controls. Odds ratios were calculated for a number of occupations, with adjustment for age and smoking. For all lung cancers, the odds ratio associated with cabinet- and furniture making was 1.3 (95% CI, 0.5–3.3), and that for carpenters was 1.3 (1.0–1.7). Cabinet- and furniture makers had increased risks for adenocarcinoma of the lung (2.0; 0.4–8.1), small-cell carcinoma (1.6; 0.2–7.9) and tumours of 'other' or mixed-cell types (1.9; 0.4–7.4), but not for squamous-cell carcinoma (0.7; 0.1–3.5). In carpenters, the odds ratio for adenocarcinoma was 1.6 (1.0–2.5); that for small-cell carcinoma, 1.1 (0.6–2.0); that for squamous-cell carcinoma, 1.2 (0.8–1.8); and that for other or mixed type, 1.3 (0.8–2.2).

The studies of cancer risk in wood-related occupations, using the New Zealand Cancer Registry (Kawachi *et al.*, 1989), described on p. 115, showed age-adjusted odds ratios of 1.3 (95% CI, 1.2–1.6) for all woodworkers, 1.3 (0.85–1.9) for foresters and loggers, 1.8 (1.2–2.5) for sawmill workers, 1.2 (0.77–1.8) for cabinet-makers 1.3 (1.1–1.5) for carpenters. Although

52% of the pulp and paper mill and sawmill workers were regular smokers at the time of the 1981 census, compared with a 38% smoking prevalence in the total labour force of New Zealand, sawmill workers did not have an excess risk of other cancers (those of the larynx, oesophagus and bladder) associated with tobacco smoking. In carpenters, the prevalence of smoking was 36%.

A case-control study was conducted in France to examine the relationship between bronchial adenocarcinoma and exposure to wood dust (Schraub *et al.*, 1989). All histologically confirmed male cases of adenocarcinoma of the lung reported to the cancer registry of the Doubs region during 1978-85 formed the case series: 22 living and 40 dead cases were identified; nine case patients could not be located, and the remaining case patients or their next-of-kin were interviewed. A population sample (three controls randomly selected from among males within five years of the ages of the cases at the time of diagnosis) of 160 men formed the control group, representing an 86% participation rate. The controls were on average five years older than the cases. Occupational exposures and consumption of tobacco and alcoholic beverages were documented by interviews with live case patients and 160 controls and with families or physicians of the dead case patients. The crude odds ratio for exposure to wood dust was 1.1 (95% CI, 0.38-2.7). Adjustments for cigarette smoking, age and urban-rural residence resulted in 'only trivial, nonsignificant increases' in the odds ratio associated with exposure to wood dust. The mean duration of exposure was 6.8 years for the cases and 17.3 years for the controls. [The Working Group noted that, although the controls were older than the cases, the authors did not indicate whether the work histories of the controls were truncated to match the time-frame of those of the cases.]

A community-based case-referent study of occupational risk factors for lung cancer was conducted in the Detroit metropolitan area, United States (Burns & Swanson, 1991). Histories of occupation and tobacco use were obtained by telephone interview for 5935 incident lung cancer case patients and 3956 controls with colorectal cancer. The cases and controls were identified through the metropolitan Detroit cancer surveillance system, in which patients are enrolled within two to six weeks after diagnosis; the overall response rate was about 93%. The odds ratio for woodworkers was 1.1 (95% CI, 0.70-1.8), after adjustment for age at diagnosis, cigarette smoking history, race and sex. For workers in wood manufacture, which included many fewer subjects than the occupational category 'woodworkers', the odds ratio was 2.3 (0.81-6.4).

In a hospital-based case-control study conducted in five German cities (Jöckel *et al.*, 1992), 194 patients with primary lung cancer, 194 hospital controls with an admission diagnosis unrelated to tobacco smoking and 194 population controls identified from residential registries were interviewed about smoking, occupational and residential histories. Controls were individually matched to the cases by sex and age. The response rate of the population controls was 40.7%. The smoking-adjusted odds ratio was 0.9 (95% CI, 0.46-2.0) for males in the paper, wood and printing industries, 0.7 (0.28-1.9) for wood processing workers and 0.8 (0.36-1.6) for carpenters and brick masons. [The Working Group noted the low participation rate in the controls.]

In a hospital-based case-control study in nine metropolitan areas of the United States (Morabia *et al.*, 1992), 1793 male lung cancer cases were matched by race, age, hospital, year of

interview and cigarette smoking with 2230 cancer and 998 non-cancer hospital controls, some of whom had tobacco-related diagnoses. Usual occupation, exposure to potential carcinogens and cigarette smoking were addressed during interviews conducted in 1980–89. Carpenters and cabinet-makers had a nonsignificant excess risk (odds ratio, 1.4), adjusted for age and tobacco smoking.

An updating (Kauppinen *et al.*, 1993) of a Finnish case-control study nested in a cohort of male woodworkers (Kauppinen *et al.*, 1986) was based on a cohort of 7307 workers from 35 industrial facilities (sawmills and furniture, construction carpentry, plywood and particle-board factories). A total of 136 incident respiratory cancers (cancers of the lung, trachea, larynx, epiglottis, tongue, pharynx, mouth, nose and nasal sinuses) was identified in the cohort during 1957–82. Three control subjects in the cohort who had not contracted respiratory cancer were matched to each case by year of birth. Plant- and time-specific job exposure matrices were constructed for 12 major agents in the wood industry. Job histories were based on plant records and complemented by responses to questionnaires from the case patients and controls or their next-of-kin; the questionnaires also provided data on tobacco smoking. The smoking-adjusted odds ratio for lung cancer and exposure to wood dust was 1.3 [95% CI, 0.8–2.3] with no exposure lagging; the ratio dropped to 0.44 [0.2–1.3] after lagging by 10 years. No trend by level or cumulative exposure was observed.

Associations with occupation were examined in a case-control study conducted in India (Notani *et al.*, 1993) of 246 male residents of Maharashtra State with a diagnosis of lung cancer. A total of 212 sex- and age-matched hospital controls (patients with cancers of the mouth and pharynx or non-neoplastic oral diseases) were selected, such that the community distribution was similar to that of the cases. Interviews were conducted with the case patients and controls to obtain lifetime occupational history, self-reported history of specific exposures, demographic variables, tobacco use, alcohol consumption and medical history. Each job was coded according to the International Standard Classification of Occupations. The odds ratios for ever having been employed as a woodworker were 3.0 (95% CI, 1.0–9.3; adjusted for age) and 3.2 (0.9–12; adjusted for age and smoking). [The Working Group noted that the participation rates of the case patients and controls were not documented and that the inclusion of controls with cancer of the pharynx biased the odds ratio towards the null.]

The associations between lung cancer and occupation were examined in a case-control study of 965 women aged 29–70 in whom primary lung cancer was diagnosed in the cities of Shenyang and Harbin, China, and notified to the cancer registries of these cities during 1985–87 (Wu-Williams *et al.*, 1993). They represented 92% of the eligible cases. Controls were women randomly selected from the populations of the same cities and frequency matched to the cases by age. Personal interviews were conducted with the cases and controls to obtain demographic data and information on lifetime smoking habits, sources of pollution, histories of occupation and specific exposures, and medical and dietary histories. The employment data were classified by industry and occupation according to the classification of the 1982 Chinese population census. The odds ratio for workers in the manufacture of wooden products, adjusted for smoking, study area, age and education, was 0.9 (95% CI, 0.5–1.7). For nonsmokers, the odds ratio, adjusted for study area, age and education, was 1.5 ($p > 0.05$). Timber processing was associated with an

odds ratio of 1.1 (0.6–2.0), adjusted for smoking, study area, age and education. For nonsmokers, the odds ratio, adjusted for study area, age and education, was 1.5 ($p > 0.05$). Self-reported exposure to wood dust was associated with an odds ratio of 1.1 (0.8–1.7; adjusted for smoking, study area, age and education). The odds ratio was 1.3 ($p > 0.05$) among nonsmokers, after adjustment for study area, age and education.

Studies on lung cancer are summarized in Table 28.

2.4.3 Cancers of the lymphatic and haematopoietic system

(a) Non-Hodgkin's lymphoma

(i) Exposure to wood dust

In the study of Siemiatycki (1991), described on p. 140, the total number of eligible cases of non-Hodgkin's lymphoma was 258; 215 responded, for a response rate of 83%. The odds ratio for any exposure to wood dust was 0.8 [95% CI, 0.6–1.2]; for substantial exposure, it was 1.0 [0.6–1.6].

Partanen *et al.* (1993) reported on a small industry-based case-control study of malignant lymphoma and exposures in the wood industry. In a retrospective cohort of male woodworkers, eight cases of non-Hodgkin's lymphoma were notified to the Finnish Cancer Registry in 1957–82. Fifty-two controls from the cohort were individually matched to the cases by year of birth and survival in 1983. Individual employment histories in woodworking facilities were abstracted from factory records, and a number of exposures were reconstructed with an ad-hoc plant- and period-specific job-exposure matrix. For the cases, this information was completed by interview of selected people at the factories and from questionnaires sent to the case subjects or their next-of-kin. [The Working Group noted that the data on exposure were more detailed for cases than for controls and that this may have induced a positive bias in the results.] The unadjusted odds ratios associated with exposure to wood dust were 2.1 (95% CI, 0.43–11) for all lymphomas and 2.1 (0.23–20) for non-Hodgkin's lymphoma.

(ii) Occupational group

Cartwright *et al.* (1988) reported on risk factors for non-Hodgkin's lymphoma in a case-control study in the United Kingdom. Case patients were identified in 1979–84 in hospitals, the cancer registry and the lymphoma panel in Yorkshire; additional cases during the period were sought in the area. Only cases confirmed histologically were accepted. The controls were in-patients with a variety of nonmalignant conditions. Attempts were made to match two hospital controls to each case by residential health district, sex and age. Case patients and control patients were interviewed with regard to past medical history, drug use, family medical history, hobbies, occupation, smoking and alcohol consumption. Of a total of 1407 patients with non-Hodgkin's lymphoma who had been notified, 437 (244 with low-grade tumours, 177 with high-grade tumours and 36 with unspecified subtypes) were interviewed; the commonest reasons for failure to be interviewed were insufficient information on the patient's age, sex or address, lack of histopathological confirmation or death before interview. An interview was completed with 724 controls. For woodworkers, a 'nonsignificant risk ratio under 2.0' was reported, with 28 exposed cases and 35 exposed controls. Occupational or private (more than three months) exposure to

Table 28. Case-control studies of lung cancer

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative	OR/RR risk applies	95% CI or <i>p</i>	Comments
Exposure to wood dust								
Blot <i>et al.</i> (1982)	USA	M	321/434	Interview	Wood dust	1.9		All lung cancer
					Wood dust	3.4		
Correa <i>et al.</i> (1984)	USA	MF	1338/1393	Interview	Wood dust	1.4	<i>p</i> < 0.05	Adjusted for smoking; white men
Bond <i>et al.</i> (1986)	USA	M	308/588	Company records	Any wood dust (15-year lag)	1.3	0.8–2.2 0.6–1.8	Nested study
					High exposure	1.0		
Kjuus <i>et al.</i> (1986)	Norway	M	176/176	Interview	Woodworking Wood dust	0.7 0.5	0.2–2.3 <i>p</i> < 0.05	Matching by age; adjusted for cigarette smoking
Siemiatycki <i>et al.</i> (1986), Siemiatycki (1991)	Canada	M	857/1360	Interview	Wood dust, any exposure	1.2	[1.0–1.5] [0.9–1.8]	Adjusted for a number of confounders
					Wood dust, substantial exposure	1.3		
Lerchen <i>et al.</i> (1987)	USA	M	333/499	Interview	Wood dust (ever)	0.8	0.3–1.7	Adjusted for age, ethnic group, smoking
Levin <i>et al.</i> (1988)	China	M	733/760	Interview	Wood dust (self-reported)	1.7	1.0–2.7	Category matching by age; adjusted for smoking and age
Occupational group								
Harrington <i>et al.</i> (1978)	USA	M	858/858	Death certificate	Wood and paper industry (usual job)	1.3	<i>p</i> > 0.05	Matching by age, year of death, race and residence
Esping & Axelson (1980)	Sweden	M	25/370	Death register	Woodworking Furniture maker Other woodworker	4.1 6.0 (crude) 2.3 (crude)	1.6–11	Rough adjustment for age

WOOD DUST

Table 28 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative	OR/RR risk applies	95% CI or <i>p</i>	Comments
Occupational group (contd)								
Milne <i>et al.</i> (1983)	USA	M	747/3130	Death certificate	Sawmills Furniture manufacture Cabinet-maker, furniture finisher (usual job)	0.8 4.2 1.0	<i>p</i> > 0.05 <i>p</i> < 0.01 <i>p</i> > 0.05	
Buiatti <i>et al.</i> (1985)	Italy	M	340/817	Interview	Woodworking (ever)	0.6	0.3–1.1	Adjusted for age, smoking, place of birth
Coggon <i>et al.</i> (1986)	United Kingdom	M	738/2204	Postal questionnaire	Woodworking (ever)	1.7	1.0–3.0	Adjusted for age, residence, source of history (patient or relative), smoking
Damber & Larsson (1987)	Sweden	M	589/1035	Postal questionnaire	Carpenter (at least 1 year)	0.8 (dead controls) 0.7 (living controls)	0.5–1.3 0.5–1.2	Matching by sex, birth year. residence; adjusted for smoking
Schoenberg <i>et al.</i> (1987)	USA	M	763/900	Interview	Furniture and fixture worker (ever)	1.5	0.8–3.0	Adjusted for smoking
Ronco <i>et al.</i> (1988)	Italy	M	126/384	Interview	Furniture or cabinet-maker (ever)	2.8	0.9–8.4	Adjusted for age, smoking, other occupations
Hoar Zahm <i>et al.</i> (1989)	USA	M	4431/11 326	Cancer register (medical record)	Cabinet- and furniture makers (at time of diagnosis)	1.3 (all lung cancer) 2.0 (adenocarcinoma)	0.5–3.3 0.4–8.1	Adjusted for age, cigarette smoking
Kawachi <i>et al.</i> (1989)	New Zealand	M	4224/15 680	Cancer registry	Any woodworking Sawmill worker Cabinet-maker	1.3 1.8 1.2	1.2–1.6 1.2–2.5 0.8–1.8	Adjusted for age

Table 28 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative	OR/RR risk applies	95% CI or <i>p</i>	Comments
Occupational group (contd)								
Schraub <i>et al.</i> (1989)	France	M	53/160	Interview	Wood dust	1.1	0.4–2.7	
Burns & Swanson (1991)	USA	MF	5935/3956	Interview	Woodworker Wood manufacture	1.1 2.3 (usual job)	0.7–1.8 0.8–6.4	Adjusted for age, race, sex, tobacco smoking
Jöckel <i>et al.</i> (1992)	Germany	M	146/292	Interview	Wood processing worker (ever)	0.7	0.3–1.9	Matching by sex and age; adjusted for tobacco smoking
Morabia <i>et al.</i> (1992)	USA	M	1793/3228	Interview	Carpenter and cabinet-maker (usual job)	1.4	<i>p</i> > 0.05	Adjusted for age and smoking
Kauppinen <i>et al.</i> (1993)	Finland	M	136/408	Company records	Wood dust	0.4	[0.2–1.3]	Adjusted for smoking; 10-year lagging of exposures
Notani <i>et al.</i> (1993)	India	M	246/212	Interview	Woodworker (ever)	3.2	0.9–12	Adjusted for age and smoking
Wu-Williams <i>et al.</i> (1993)	China	F	965/959	Interview	Timber processing (> 1 year) Wood dust (self-reported)	1.5 1.1	<i>p</i> > 0.05 0.8–1.7	In nonsmokers Adjusted for smoking, study area, age, education

OR, odds ratio; RR, relative risk; CI, confidence interval; M, male; F, female

wood dust was associated with a significantly increased risk ratio of 1.5 (95% CI, 1.0–2.1). Exposure to wood dust was associated more strongly with the low-grade subtype of non-Hodgkin's lymphoma (odds ratio, 2.0; $p < 0.05$) than with the high-grade type (odds ratio, 1.1). [The Working Group noted that the controls were insufficiently described; it is not clear whether the 'risk ratios' reported are crude or adjusted for sex and age; a source of possible bias is the fact that a large proportion of cases could not be interviewed.]

A case-control study of non-Hodgkin's lymphoma and occupation based on information from death certificates was reported by Schumacher and Delzell (1988), involving 501 male residents of North Carolina (United States), who died at 35–75 years of age from non-Hodgkin's lymphoma (ICD 8,9 200.0–200.9, 202.0–202.9) during 1968–70, 1975–77 and 1980–82. The controls were 569 male residents of North Carolina who died from causes other than cancer in the same periods and who were frequency matched to the cases by age, year of death and race. The usual occupation and industry were abstracted from death certificates and coded according to a system that combined industry and occupation. Work in paper and wood industries was associated with odds ratios of 0.79 [95% CI, 0.5–1.3] in whites and 1.3 [0.3–4.8] in blacks. For work in the furniture industry, which was a subcategory of paper and wood, the odds ratios were 0.74 [0.4–1.4] for whites and 1.9 [0.1–30] for blacks.

Franceschi *et al.* (1989) reported on a hospital-based case-control study of non-Hodgkin's lymphoma conducted in the province of Pordenone, north-eastern Italy. The cases were histologically confirmed non-Hodgkin's lymphomas diagnosed in 1984–88 in men and women under the age of 80, who had been admitted as in-patients or referred for follow-up at the out-patient clinics at Aviano Cancer Centre and general hospitals in the province. Of the 232 eligible case patients with lymphosarcoma and reticulosarcoma (ICD: 200) and other non-Hodgkin's lymphoma (ICD: 202), 18 died before they could be interviewed and there was no histological confirmation for six. None of the living case patients refused interview. The case series thus comprised 208 non-Hodgkin's lymphoma patients—110 men and 98 women. Controls were 401 interviewed in-patients at the same hospitals (215 men, 186 women), who were under the age of 80. Patients with admission diagnoses of malignant disorders, conditions related to alcohol and tobacco consumption, haematological, allergic and autoimmune conditions, and diseases that might have resulted in diet modifications, such as diseases of the respiratory and digestive tracts, cardiovascular disease and diabetes, were excluded from the control series. The interview covered sociodemographic characteristics, smoking, consumption of alcohol and coffee, intake of selected food items, medical history, vaccinations, tonsillectomy, medical radiation exposure, occupational history and self-reported exposure to 20 potentially carcinogenic agents, including wood dust. The job category 'wood and furniture workers', not further specified, was associated with an odds ratio of 0.66 (95% CI, 0.37–1.2), adjusted for age and sex. The number of subjects exposed to wood dust was too small for analysis.

Persson *et al.* (1989) examined associations between occupational exposures and Hodgkin's disease and non-Hodgkin's lymphoma in Sweden. The 106 non-Hodgkin's lymphoma cases (66 in men) were identified at the register of the Department of Oncology, Örebro Medical Centre Hospital in people who were alive in 1986 and whose cancers were diagnosed when they were 20–80 years of age, during 1984–86. Six eligible case patients were either unwilling to parti-

cipate or could not be contacted. Population controls representing the catchment area of the hospital comprised 275 people aged 20–80, after replacement of 17% who were unable or unwilling to participate. 'Exposure' was defined as that occurring 5–45 years before diagnosis of a non-Hodgkin's lymphoma and lasting at least one year. An elevated risk was suggested in carpenters and cabinet-makers (crude odds ratio, 3.1). The odds ratio for exposure to fresh wood, as defined by employment as a sawmill worker, lumberjack or paper pulp worker, was 1.3. The odds ratio for work as a carpenter or cabinet-maker, adjusted for age at diagnosis, sex and farming, was 2.8 [95% CI, 0.9–8.5]; that for exposure to fresh wood was 1.0 [0.3–3.5].

A subsequent study (Persson *et al.*, 1993) was conducted with similar methods in an adjacent region of Sweden (Östergötland, Jönköping, Kalmar). The cases were non-Hodgkin's lymphomas (ICD8: 200.0–2) diagnosed in 1975–84 among men who were alive in 1986, who were aged 20 years or more, who were living in the catchment area and were identified at the cancer registry covering the region. After 14 refusals and lack of diagnostic confirmation, 93 cases of non-Hodgkin's lymphoma remained. The 204 controls had also been used in other case-control studies (Flodin *et al.*, 1986, 1987, 1988) and resided in the catchment area from which the cases were drawn. A postal questionnaire requested information about occupational exposures, medical care and leisure exposures. 'Exposure' was defined as occupation 5–45 years before the diagnosis of a non-Hodgkin's lymphoma and lasting at least one year. The crude odds ratio for non-Hodgkin's lymphoma in subjects exposed to fresh wood (sawmill workers, lumberjacks and paper pulp workers) was 2.6. The odds ratio for carpenters and cabinet-makers was 0.9 [95% CI, 0.3–2.4], adjusted for age at diagnosis and occupational confounders. Among the workers exposed to fresh wood, the subcategory 'lumberjacks' was associated with an adjusted odds ratio of 6.0 [95% CI, 0.8–44].

Reif *et al.* (1989) conducted a series of case-control studies on a number of cancer sites in New Zealand to assess associations between forestry work and cancer. All cancer cases in men aged 20 years and over were identified in 1980–84 at the New Zealand Cancer Registry. Occupation was recorded for 19 904 men (80%); 535 eligible cases of non-Hodgkin's lymphoma (ICD: 200,202) were available. Cases of other cancers formed the control group. The age-adjusted odds ratio for non-Hodgkin's lymphoma among foresters and loggers was 1.8 (95% CI, 0.85–4.0); the odds ratio for sawmill workers was 1.2 (0.43–3.2; four exposed cases).

Whittemore *et al.* (1989) conducted a case-control study on mycosis fungoides, a cutaneous T-cell lymphoma. They interviewed 174 people over 20 years of age in northern California, Los Angeles County and the Seattle-Puget Sound area (United States) in whom mycosis fungoides had been diagnosed in 1981–86 and identified at tumour registries and hospitals. The controls were 294 people selected by random-digit dialling who were also interviewed about potential risk factors for mycosis fungoides, and a lifetime employment history was taken. The response rate among the case subjects was 60% (23% were dead), and that among controls was 76%. Relatively fewer case subjects than controls reported previous employment in the paper and wood industry (relative risk, 0.5; $p = 0.02$). The risk was also reduced among people exposed to chromium and its salts, mercury and its salts, halogenated and aromatic hydrocarbons and uncured plastic. [The Working Group noted that employment in the wood and paper industry

was insufficiently focused to provide a reasonable proxy for exposure to wood dust; in addition, the response rate for the cases was low.]

Scherr *et al.* (1992) conducted a hospital-based case-control study on occupational exposures and risk for non-Hodgkin's lymphoma. Interviews were conducted with 303 residents of the Boston, Massachusetts (United States), metropolitan area (80% participation rate) with confirmed non-Hodgkin's lymphoma diagnosed in 1980-82, or with their next-of-kin, and with 303 population controls (72% participation rate) matched by age, sex, town and precinct of residence. The case patients were identified at nine hospitals and the controls from town resident lists. Interviews were completed with 202 patients, 101 proxies of patients and 303 controls, and job histories were obtained. The odds ratio for non-Hodgkin's lymphoma associated with exposure to particles (dust, sawdust and fibres) was 1.4 (95% CI, 0.9-1.8); that for employment in the paper and wood industry was 1.7 (0.7-4.2). [The Working Group noted that the categories 'particles' and 'paper and wood industry' were remote proxies for exposure to wood dust; furthermore, a high proportion of next-of-kin of patients were interviewed]

A population-based case-control study of 622 white men with non-Hodgkin's lymphoma diagnosed in 1980-83 and 1245 white male population controls without haematopoietic or lymphatic malignancies in Iowa and Minnesota, United States, was conducted to examine associations between occupation and risk for non-Hodgkin's lymphoma (Blair *et al.*, 1993). Case patients and controls who resided in the cities of St Paul, Duluth, Minneapolis and Rochester were excluded because agricultural exposures were the primary focus of the study. Case coverage was almost complete; interviews were conducted with 87% of the case subjects or their next-of-kin and with 77% of the controls. The interview covered sociodemographic characteristics, agricultural activities and exposures, exposures to chemicals in hobbies, residential history, medical history, familial history of cancer and occupational history. A job-exposure matrix was developed for job title-industry combinations and a number of exposures. Exposure to wood dust was associated with an odds ratio of 0.9 (95% CI, 0.7-1.2), adjusted for age, state, smoking, family history of malignant lymphoproliferative disease, agricultural exposure to pesticides, use of hair dyes and direct or surrogate responder. In a category of lower intensity of exposure to wood dust, the odds ratio was 0.9 (0.7-1.2). In the category of higher intensity, there were no cases and two controls.

(b) *Hodgkin's disease*

(i) *Exposure to wood dust*

In the study of Partanen *et al.* (1993), described on p. 146, four cases of Hodgkin's disease were notified. In comparison with 21 controls from the cohort individually matched to the cases, the unadjusted odds ratio for Hodgkin's lymphoma and exposure to wood dust, based on three exposed cases, was 2.1 (95% CI, 0.21-22).

(ii) *Occupational group*

Milham and Hesser (1967) reported an association between occupational exposure to wood and Hodgkin's disease in upstate New York, United States. They analysed the occupations of 1549 white men, aged 25 years or more, who had died of Hodgkin's disease during 1940-53 and 1957-64, and 1549 dead controls individually matched to the cases by age, sex, race (white

only), residence and date of death. 'Exposure to wood' was defined as notification of a wood-related occupation (the commonest were carpenter and cabinet-maker) on the death certificate. The analysis revealed 69 pairs in which the case was exposed and the control unexposed, and 30 pairs in which the case was unexposed and the control exposed, yielding an odds ratio of [2.3] ($p < 0.001$). No other occupational group showed a significant excess.

Petersen and Milham (1974) evaluated the risk for Hodgkin's disease in occupations related to woodworking in Washington State, United States. The study had three phases: (i) a case-control study of deaths from Hodgkin's disease in 1950-71, with controls consisting of all residual, nonaccidental and nonviolent deaths, individually matched to each case by year of death, age at death and county of residence (707 matched pairs), in which wood-related occupations were ascertained from death certificates; (ii) a study of dead cases of Hodgkin's disease and dead controls during 1965-70 (158 matched pairs), in which occupational histories were obtained from interviews with relatives; and (iii) a proportionate mortality study of deaths from Hodgkin's disease in 1950-71. In the case-control study based on death certificates, there were 56 discordant pairs in which the case was in a woodworker and 32 discordant pairs in which the control was a woodworker, yielding an odds ratio of [1.8] ($p < 0.05$). In the study based on interviews, the frequencies of discordant pairs were 23 and 10, respectively ([odds ratio, 2.3] $p < 0.05$). The proportionate mortality ratio in woodworkers was 1.6 (56 deaths from Hodgkin's disease observed; $p < 0.001$).

Abramson *et al.* (1978) conducted a case-control study of Hodgkin's disease in Israel. All cases histologically diagnosed among Jewish residents of Israel in 1960-72 were eligible, giving 527 patients (454 definite, 37 probable and 36 possible). Jewish controls were drawn from the national population register, individually matched to the cases by sex, birth year, country of birth, father's region of birth (for subjects born in Israel) and year of immigration. Interviews were conducted with patients or proxies; proxy information was obtained for 68% of cases and 28% of controls. The response rate was 96%, and suitable controls were interviewed for 473 cases. The interview yielded information on occupation. Separate comparisons were made for the main histological subtypes, nodular sclerosis and mixed cellularity. Work with wood or trees (predominantly carpentry) was associated with a relative risk of 1.1 ($p > 0.05$). The risk for nodular sclerosis subtype was 0.6, but that for mixed cellularity was 5.2 ($p = 0.0005$).

Greene *et al.* (1978) identified 167 deaths from Hodgkin's disease among white men in North Carolina, United States, in 1956-74, and two controls for each case, matched by sex, race, county of death and age and year of death. A risk ratio of 1.4 (95% CI, 0.8-2.3) was associated with occupational exposure to wood and paper and a risk of 4.2 (1.4-13) with carpentry and lumbering.

Fonte *et al.* (1982) reported on a case-control study in Italy on Hodgkin's disease diagnosed in 207 men and 180 women admitted to the university hospital of Pavia during 1972-79. The controls were 441 men and 330 women admitted to an internal medical unit in Pavia in 1977-79. The occupations appearing in medical records were classified. Nine case patients worked in the wood industry, resulting in a relative risk of 7.2 (95% CI, 2.3-22). [The Working Group noted that the methods used were not well described.]

Bernard *et al.* (1987) reported on risk factors for Hodgkin's disease in a case-control study in the United Kingdom. All cases identified between October 1979 and December 1984 in hospitals in Yorkshire were eligible for inclusion; only those histopathologically confirmed were accepted. The controls represented in-patients with a variety of nonmalignant conditions and were matched to the cases by health district, sex and age in a ratio of 2:1. Case patients and controls were interviewed about past medical history, drug use, family medical history, hobbies, occupation, smoking and alcohol consumption. The study comprised 297 interviewed patients, who represented 70% of all histologically confirmed cases. For both woodworkers and contact with wood dust, a nonsignificant risk ratio 'under 2.0' was reported (woodworkers: 16 cases, 35 controls; wood dust: 24 cases, 46 controls). [The Working Group noted that a large proportion of case patients could not be interviewed and the results for woodworkers and exposure to wood dust were stated only as 'under 2.0'.]

Brownson and Reif (1988) evaluated occupational risks for Hodgkin's disease, mainly in farming, by identifying cases and controls through the cancer registry in Missouri (United States). Hodgkin's disease (ICD 9: 201) was diagnosed in 475 white male Missouri residents over 20 years of age in 1984 and 1985. The 1425 controls represented other cancers, excluding those of the oral cavity, pharynx, oesophagus, larynx, lung, bladder and prostate, and were individually matched 3:1 to the cases by age. Usual occupation and industry, as obtained from the routine records of the registry, were coded by the codes of the 1980 United States census. The registry also provided data on the smoking habits of the subjects. Carpenters had an increased risk for Hodgkin's disease: odds ratio, 3.1; 95% CI, 1.0-9.8, adjusted for age and smoking. [The Working Group noted that the control group included certain cancers that are potentially etiologically related to exposure to wood dust, which would have biased the odds ratio towards the null.]

In the study of Persson *et al.* (1989), described on p. 150, 54 cases of Hodgkin's disease were identified. No excess risk for Hodgkin's disease was associated with carpentry or cabinet-making; the odds ratio, adjusted for age at diagnosis, sex and farming, was 0.2 [95% CI, 0.01-2.8]. The adjusted ratio for exposure to fresh wood, as defined by employment as sawmill worker, lumberjack or paper pulp worker, was 0.4 [0.1-1.5].

In the study of Persson *et al.* (1993), reported on p. 151, 31 cases of Hodgkin's disease were identified. The odds ratio for Hodgkin's disease associated with the job title 'carpenters and cabinet-makers' was 0.2 (one exposed case, 25 exposed controls). The odds ratio for Hodgkin's disease in subjects exposed to fresh wood, adjusted for age at diagnosis and occupational confounders, was 3.8 [95% CI, 0.9-17].

(c) *Multiple myeloma*

(i) *Exposure to wood dust*

A hospital-based case-control study of multiple myeloma was reported in the United Kingdom (Cuzik & De Stavola, 1988). The cases were identified at major referral centres in six areas of England and Wales between 1978 and 1984. Two controls were sought for each case and matched by age and sex: one from the same hospital as the case and one from the list of the general practitioner of the case patient. Interviews were conducted with 409 case subjects, 399

hospital controls and 260 general practitioner controls to obtain occupational histories and information on exposures to chemicals and radiation, diseases, immunizations, family history, chronic infections and defects in immune regulation. The results were given as percentages of employed or exposed cases and controls: 1.5% of the case patients and 2.5% of the hospital controls had been employed in the production of furniture or upholstery [crude odds ratio, 0.6; 95% CI, 0.2–1.7]; 2.8% of the case patients and 1.3% in the controls had been exposed to wood dust for 1–10 years [crude odds ratio, 2.2; 0.8–6.5]; and 3.0% of the cases and 4.3% of the controls had been exposed for more than 10 years [crude odds ratio, 0.7; 0.3–1.5].

In 1982, more than 77 000 American Cancer Society members enrolled over 1.2 million friends, neighbours and relatives in a prospective mortality study, which included the completion of an initial questionnaire on medical, occupational and lifestyle factors and exposure history. In a case–control study of multiple myeloma based on these data (Boffetta *et al.*, 1989), 282 people who had died during the first four years and for whom multiple myeloma was reported on the death certificate as the underlying or contributing cause of death were identified, after successful tracing of 98.5% of subjects and 84% coverage of death certificates. Four randomly selected controls were matched to the cases by sex, American Cancer Society division, year of birth and ethnic group, for a total of 1128. A further subdivision was made between incident cases ($n = 128$) and prevalent cases ($n = 154$) during the case ascertainment period, since a cancer detected before this period might have affected habits and occupations and the reporting of them. Self-reported exposure to wood dust was associated with an odds ratio of 1.2 (95% CI, 0.5–3.2; logistic model, with adjustment for age, sex, ethnic group, division of the American Cancer Society, education, history of diabetes, X-ray treatment, pesticide and herbicide exposure and farming) for incident cases.

A population-based case–control study (Heineman *et al.*, 1992) was carried out of the occupational exposures of 1098 Danish men in whom multiple myeloma was diagnosed in 1970–84 and recorded at the Danish Cancer Registry, and of 4169 male population controls matched on birth year who were alive at the time of diagnosis of the case. Histological confirmation was available for 92% of the cases. Job histories from 1964 on were abstracted from the records of the nationwide Supplementary Pension Fund. A job–exposure matrix was constructed for 47 substances. The age-adjusted odds ratios related to exposure to wood dust were 1.2 (95% CI, 0.7–2.1) for furniture maker as the most recent occupation, 1.1 (0.4–2.5) for sawmill and other woodwork, 1.0 (0.6–1.4) for wood and wood products, 0.7 (0.3–1.3) for lumber and 1.6 (0.7–4.0) for miscellaneous wood products. Employment for fewer than five years in the wood and wood product industry was associated with an age-adjusted odds ratio of 1.1 (0.6–1.9); in those employed for five years or more, it was 0.9 (0.4–1.8). The results for women were reported in another article (Potterm *et al.*, 1992). There were 1010 cases and 4040 matched controls. The industrial category ‘wood/products’ was associated with an age-adjusted odds ratio of 1.1 (95% CI, 0.3–3.4); the odds ratio for probable exposure to wood dust was 1.9 (0.4–8.1).

(ii) *Occupational group*

The risk for multiple myeloma among furniture workers was evaluated in a population-based case–control study in 20 counties of North Carolina, United States (Tollerud *et al.*, 1985).

Listings of deaths during 1956–80 showed that 301 men were recorded with multiple myeloma or another immunoproliferative neoplasm (ICD9: 203.0,1,8; 238.6). These men were matched with one to three male controls by race, county of usual residence, age at death and year of death, to give 858 controls. The principal industry of employment, as recorded in the death certificate, was analysed: furniture manufacture was associated with an odds ratio of 1.3 ($p = 0.25$) and other woodworking with an odds ratio of 1.1 ($p = 0.69$). For furniture workers born before 1905 and who died before the age of 65, the odds ratio was 5.4 ($p = 0.05$). The odds ratios were lower for other combinations of birth year, age at death and race; none reached statistical significance. The authors noted that no information was available on time or duration of employment or specific occupational activities or exposures, including potential confounders. Underreporting of occupation in older individuals was a further concern.

Potential risk factors for multiple myeloma (ICD1965: 203.99) were evaluated in a case-control study of 131 patients and 431 referents, all of whom were alive (Flodin *et al.*, 1987). The cases were identified from the registers of the cytological departments of three hospitals and the medical clinics of three further hospitals, all in central and south-eastern Sweden. They were diagnosed in 1973–83 and included patients who survived until 1981–83. About one-third of all incident cases in the catchment area were estimated to have been identified. Referents were a random sample from the population registers of the catchment area. A postal questionnaire sought information on potential risk factors; the response rate was 96% for cases and 80% for controls. At least one year of exposure to fresh wood (i.e. lumberjacks, paper pulp workers and sawmill workers), lagged by five years from the diagnosis of multiple myeloma, was associated with a crude rate ratio of 3.9 (95% CI 1.9–7.6; 17 cases). Another crude rate ratio was, however, presented for the same association: 2.6 [no CI given]. The rate ratio for exposure to fresh wood, adjusted for age, was 3.2 (1.5–6.5); after adjustment for age, exposure to exhaust fumes, creosote, concrete and brickwork, sulfonyleurea, γ -radiation, ex-smoking and farming, the rate ratio (men only) was 2.6 (1.1–5.7). Working with dried timber was not associated with a significant excess risk.

In the study of Reif *et al.* (1989), in New Zealand, described on p. 151, the number of eligible cases of multiple myeloma (ICD: 203) was 295. The age-adjusted odds ratio associated with forestry and logging was 0.53 (95% CI, 0.08–3.7).

(d) *Leukaemia*

(i) *Exposure to wood dust*

In the study of Partanen *et al.* (1993) reported on p. 150, 12 cases of leukaemia were diagnosed, and 79 controls from the cohort were individually matched to the cases. Exposure to wood dust was not associated with an increased risk (crude odds ratio, 0.56; 95% CI, 0.2–2.2).

(ii) *Occupational group*

In a short communication, Burkart (1982) reported an excess risk for leukaemia among long-term workers in sawmills. Male cases (ICD9 clinically modified: 204–208) and noncancer controls were identified in four hospitals in Oregon, United States, during 1980 and administered an occupational questionnaire. With a 90% response rate, 26 leukaemia cases and 836 controls were evaluated for exposure in sawmills. The age-adjusted summary relative risk was

1.1 for < 10 years of exposure and 3.2 for > 10 years of exposure, 'with a Mantel-Haenszel summary χ^2 for dose-response significant at $p = 0.017$.' Industrial hygiene surveys in the plants indicated use of chlorophenols.

Oleske *et al.* (1985) reported on a case-control study of hairy-cell leukaemia, a rare, usually chronic form of leukaemia. In 1975-81, 53 patients with this cancer who were residents of Illinois and northern Indiana, United States, were identified at the Hairy Cell Tumor Registry and Treatment Center at the University of Chicago and through inquiries to 1100 haematologists, pathologists and medical oncologists. Interview responses were obtained from 36 patients and nine proxies. Three neighbourhood controls were matched to each case by age, sex and race. In the process of identifying eligible controls, 19% of those eligible refused interviews, so that 134 controls were interviewed. Working for a minimum of 20 h per week during at least six months in woodwork was associated in men with an odds ratio of 4.0 (95% CI, 0.90-18), after control for age, sex and race.

Pearce *et al.* (1986) reported on leukaemia among New Zealand agricultural workers. The cases were those classified as ICD: 204-208 and registered at the New Zealand Cancer Registry in 1979-83 among 546 men who were aged 20 years or more at registration. Four controls were matched to each case by age and year of registration; those with malignant lymphoma, multiple myeloma or soft-tissue sarcoma were excluded. The occupation of carpenter, as recorded on the cancer registration form (current or most recent job title), was associated with an odds ratio of 1.5 (95% CI, 1.0-2.3).

Potential risk factors for chronic lymphatic leukaemia were evaluated in a case-control study of 111 cases (ICD[1965]: 204.15) and 431 controls, all of whom were alive (Flodin *et al.*, 1988). Cases were identified from the registers of the cytological departments of three hospitals and the medical clinics of two further hospitals, all in central and south-eastern Sweden. Most of the cases were diagnosed in 1975-84, but some as early as 1964, and included those in which the patient survived until 1981-83. Controls were a random sample from the population registers of the catchment area. A postal questionnaire sought information on potential risk factors, with a response rate of 91% for case patients and 85% for controls (non-responders were replaced by other controls). At least one year of exposure to fresh wood (i.e. lumberjacks, paper pulp workers and sawmill workers), lagged by five years from the diagnosis of chronic lymphatic leukaemia, was associated with a crude rate ratio of 3.2 (95% CI, 1.5-6.6; 13 exposed cases). The risk ratio for exposure to fresh wood (men only), adjusted for age, solvents, farming, exhausts and contact with horses, was 2.4 (1.0-5.0). Working with dried timber was not associated with a significant excess risk.

In the study of Reif *et al.* (1989), reported on p. 151, there were 534 eligible cases of leukaemia (ICD: 204-208). The age-adjusted odds ratio for all leukaemias associated with forestry and logging was 0.96 (95% CI, 0.36-2.6; four exposed cases); that for sawmill workers was 0.52 (0.13-2.1; two exposed cases).

Loomis and Savitz (1991) reported on a case-control study of occupation and leukaemia (ICD9: 204-208) among 5147 men in 16 states of the United States on the basis of information from death certificates. The controls were 51 470 men who had died of other causes, excluding brain cancer, during 1985-87. The results were given for usual occupation or industry, as

abstracted from the death certificates. Woodworking was associated with an age- and race-adjusted odds ratio of 0.9 (95% CI, 0.7–1.0). The odds ratio for occupations in wood products industries was 0.7 (0.5–0.9) and that for carpenters was 0.9 (0.7–1.1).

Fincham *et al.* (1993) reported on a case–control study of cancers at several sites, using data from the Alberta Cancer Registry, Canada. On the basis of undocumented numbers of cases of leukaemia and controls with all other cancers, a crude odds ratio of 1.8 (95% CI, 1.2–2.8; 23 exposed cases) was reported for exposure to wood dust. [The Working Group noted the lack of detail in the description of the study and the crudeness of the statistical analysis.]

Studies on lymphohaematopoietic cancers are summarized in Table 29.

2.4.4 Cancers of the digestive tract

(a) Exposure to wood dust

Spiegelman and Wegman (1985) examined the relationship between occupational risk factors and colon and rectal cancer in a case–control study based on the Third National Cancer Survey, in which data were collected on all incident cancers occurring in 1969–71 in seven metropolitan areas and two states of the United States (a region containing 10.3% of the national population). A 10% random sample was interviewed to collect information on primary and secondary occupation and industry and duration of time in these jobs. The cases were colon or rectal cancers in 343 men and 208 women. The controls were 626 men and 1235 women with other cancers classified by the authors as not commonly associated with occupational exposures (cancers of the soft tissue, eye, brain, endocrine glands, breast, male and female reproductive tracts and lymphomas). Occupational exposure to wood was estimated from a job–exposure matrix based on the National Occupational Hazards Survey. For colon and rectal cancer combined, the odds ratios, adjusted for age, race, marital status, income, weight and nutritional scores, were 1.1 ($p = 0.69$) for men and 1.5 ($p = 0.04$) for women. For colon cancer alone, the odds ratios were 1.3 ($p = 0.24$) for men and 1.5 ($p = 0.07$) for women.

In the study of Siemiatycki (1991), described on p. 140, 251 cases of stomach cancer were identified. The odds ratios were 1.4 [95% CI, 1.0–1.9] for any exposure and 1.1 [0.7–1.7] for substantial exposure. For the 497 cases of colon cancer, the odds ratios were 1.0 [0.8–1.3] for any exposure and 0.9 [0.7–1.2] for substantial exposure; for the 257 cases of rectal cancer, the odds ratios were 1.0 [0.7–1.4] for any exposure and 1.3 [0.8–2.0] for substantial exposure.

Peters *et al.* (1989) performed a case–control study of colorectal cancer among 147 white men in Los Angeles county (United States) in whom colorectal adenocarcinoma was first diagnosed in 1974–82 when they were 25–44 years of age. A matched series of 147 neighbourhood controls of the same sex, race, date of birth and neighbourhood of residence were selected. Occupational and exposure histories were collected by interview. The odds ratio for exposure to wood dust was 3.6 (95% CI, 1.2–11) after adjustment for age and education. The results by sub-site were 2.1 (0.5–8.5) for the right side of the colon, 1.5 (0.3–6.6) for the transverse and descending colon, 3.6 (0.6–21) for the sigmoid colon and 9.4 (2.0–45) for the rectum.

Table 29. Case-control studies on lymphatic and haematopoietic cancers

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Non-Hodgkin's lymphoma								
<i>(i) Exposure to wood dust</i>								
Siemiatycki <i>et al.</i> (1986); Siemiatycki (1991)	Canada	M	117/1563	Interview	Wood dust, substantial, >15 years' exposure	0.5	0.2-0.9	Adjusted for a number of confounders
			215/2357		Wood dust, substantial (update)	1.0	[0.7-1.5]	
Partanen <i>et al.</i> (1993)	Finland	M	8/52	Company records	Wood dust	2.1	0.2-20	Nested study; matched by age and survival; low power
<i>(ii) Occupational group</i>								
Cartwright <i>et al.</i> (1988)	United Kingdom	MF	437/724	Interview	Wood dust > 3 months	1.5	1.0-2.1	Incomplete documentation. Selection and information biases possible
Schumacher & Delzell (1988)	USA	M	501/569	Death certificate	Furniture industry (usual job):			
					Whites	0.7	0.4-1.4	
					Blacks	1.9	0.1-30	
Franceschi <i>et al.</i> (1989)	Italy	MF	208/401	Interview	Wood and furniture worker	0.7	0.4-1.2	
Persson <i>et al.</i> (1989)	Sweden	MF	106/275	Postal questionnaire	Carpenter, cabinet-maker	2.8	[0.9-8.5]	Adjusted for age, sex, farming
					> 1 year; 5-45 years' latency Fresh wood	1.0	[0.3-3.5]	
Reif <i>et al.</i> (1989)	New Zealand	M	535/19 369	Cancer register	Sawmill worker	1.2	0.4-3.2	Four exposed cases
Whittemore <i>et al.</i> (1989)	USA	MF (Cases: mycosis fungoides)	174/294	Interview	Employment in paper and wood industry	0.5	<i>p</i> = 0.02	Paper and wood a remote proxy for exposure to wood dust. Low response in cases

WOOD DUST

Table 29 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Non-Hodgkin's lymphoma (contd)								
<i>Occupational group (contd)</i>								
Scherr <i>et al.</i> (1992)	USA	MF	303/303	Interview	Employment in paper and wood industry	1.7	0.7–4.2	Paper and wood a remote proxy for wood dust
Blair <i>et al.</i> (1993)	USA	M	622/1245	Interview	Wood dust	0.9	0.7–1.2	Adjusted for age, state, smoking, family cancers, pesticides, hair dyes, responder
Persson <i>et al.</i> (1993)	Sweden	M	93/204	Postal questionnaire	Fresh wood: lumberjack	6.0	[0.8–44]	Adjusted for age, occupational confounders
Hodgkin's disease								
<i>(i) Exposure to wood dust</i>								
Partanen <i>et al.</i> (1993)	Finland	M	4/21	Company records	Wood dust	2.1	0.2–22	Nested study; matched by age and survival; low power
<i>(ii) Occupational group</i>								
Milham & Hesser (1967)	USA	M	1549/1549	Death certificate	Exposure to wood		<i>p</i> < 0.001	Discordant pairs 69/30
Petersen & Milham (1974)	USA	M	707/707	Death certificate	Woodworker	[1.8]	<i>p</i> < 0.05	Discordant pairs 56/32
Petersen & Milham (1974)	USA	M	158/158	Interview	Woodworker	[2.3]	<i>p</i> < 0.05	Discordant pairs 23/10
Abramson <i>et al.</i> (1978)	Israel	MF	506/473	Interview	Work with wood/trees	1.1 5.2	<i>p</i> > 0.05 <i>p</i> < 0.0005	All Hodgkin's disease Mixed cellularity subtype
Greene <i>et al.</i> (1978)	USA	M	167/334	Death certificate	Carpentry and lumbering	4.2	1.4–13	Matched by sex, race, county, age and year of death

Table 29 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Hodgkin's disease (contd)								
<i>Occupational group (contd)</i>								
Fonte <i>et al.</i> (1982)	Italy	MF	387/771	Clinic charts	Wood industry	7.2	2.3–22	Methods not well described
Bernard <i>et al.</i> (1987)	United Kingdom	MF	297/489	Interview	Wood dust	'Under 2.0'	<i>p</i> < 0.05	Large non-response of cases; incomplete documentation of results for wood dust
Brownson & Reif (1988)	USA	M	475/1425	Cancer register	Carpenter	3.1	1.0–9.8	Adjusted for age and smoking
Persson <i>et al.</i> (1989)	Sweden	MF	54/275	Postal questionnaire	Fresh wood	0.4	[0.1–1.5]	Adjusted for age, sex and farming
Persson <i>et al.</i> (1993)	Sweden	M	31/204	Postal questionnaire	Fresh wood	3.8	[0.9–17]	Adjusted for age, occupational confounders
Multiple myeloma								
<i>(i) Exposure to wood dust</i>								
Cuzik & De Stavola (1988)	United Kingdom	MF	399/399	Interview	Wood dust > 10 years	[0.7]	[0.3–1.5]	Crude analysis
Boffetta <i>et al.</i> (1989)	USA	MF	128/512	Questionnaire	Wood dust (self-reported)	1.2	0.5–3.2	Adjusted for age, sex, ethnic group, residence and farming
Heineman <i>et al.</i> (1992)	Denmark	M	1098/4169	Pension fund records	Probable exposure to wood dust	0.8	0.6–1.2	
Pottern <i>et al.</i> (1992)	Denmark	F	1010/4040	Pension fund records	Probable exposure to wood dust	1.9	0.4–8.1	

WOOD DUST

Table 29 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Multiple myeloma (contd)								
<i>(ii) Occupational group</i>								
Tollerud <i>et al.</i> (1985)	USA	M	301/858	Death certificate	Furniture manufacture	1.3	<i>p</i> = 0.25	
					Furniture manufacture, born < 1905 and died < 65 years of age	5.4	<i>p</i> = 0.05	
Flodin <i>et al.</i> (1987)	Sweden	MF	131/431	Questionnaire	Fresh wood > 1 year; lagged by 5 years (men only)	2.6	1.1–5.7	Adjusted for a number of potential confounders. Possible selection of cases
Reif <i>et al.</i> (1989)	New Zealand	M	295/19 609	Cancer register	Forestry worker and logger	0.5	0.1–3.7	One exposed case
Leukaemia								
<i>(i) Exposure to wood dust</i>								
Partanen <i>et al.</i> (1993)	Finland	M	12/79	Company records	Wood dust	0.6 (crude)	0.2–2.2	Nested study; matched by age and survival; low power
<i>(ii) Occupational group</i>								
Burkart (1982)	USA	M	26/836	Questionnaire	Sawmill exposure > 10 years	3.2	<i>p</i> = 0.017 for dose–response	Adjusted for age
Oleske <i>et al.</i> (1985)	USA	M	35/104	Interview	Woodworking > 20 h/week, > 6 months	4.0	0.9–18	Hairy-cell leukaemia
Pearce <i>et al.</i> (1986)	New Zealand	M	546/2184	Cancer register	Carpenter	1.5	1.0–2.3	

Table 29 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Leukaemia (contd)								
<i>Occupational group (contd)</i>								
Flodin <i>et al.</i> (1988)	Sweden	M	71/200	Postal questionnaire	Fresh wood	2.4	1.0–5.0	Adjusted for age, exposure to solvents, horses, farming, exhausts. Possible selection of cases
Reif <i>et al.</i> (1989)	New Zealand	M	534/19 370	Cancer register	Sawmill worker	0.5	0.1–2.1	Two exposed cases
Loomis & Savitz (1991)	USA	M	5147/51 470	Death certificates	Woodworker	0.9	0.7–1.0	Adjusted for age and race
Fincham <i>et al.</i> (1993)	Canada	NR	NR	Cancer register	Wood dust	1.8 (crude)	1.2–2.8	Incomplete documentation

OR, odds ratio; RR, relative risk; CI, confidence interval; M, male; F, female; NR, not reported

(b) *Occupational group*

Brownson *et al.* (1989) conducted a cancer registry-based case-control study of colon cancer involving white males with histologically confirmed colon cancer diagnosed between 1984 and 1987 who were reported to the Missouri Cancer Registry in the United States. Five controls for each case were randomly selected from among other white male cancer patients reported to the Registry. Data on occupation and industry from registry records (originally abstracted from medical records) were available for 1993 cases and 9965 controls. The odds ratio for carpenters was 0.9 (95% CI, 0.6–1.4) after adjustment for age. No other results related to exposure to wood were presented.

Fredriksson *et al.* (1989) reported the results of a case-control study of all people living in the region of Umeå aged 30–75 in whom adenocarcinoma of the colon had been diagnosed between 1980 and 1983 and reported to the Swedish Cancer Registry, who were alive at the time of data collection. For each case, two controls of similar age, sex and residence were identified from the National Population Register. A postal questionnaire, which included an occupational history, was completed by 312 case patients (156 men) and 623 controls (306 men). Decreased risks were observed for men who were previously employed as lumberers (odds ratio, 0.7; 95% CI, 0.4–1.0), pulp workers (0.7; 0.3–1.6) and sawmill workers (0.5; 0.3–0.9) after adjustment for age and physical activity.

In the study of Kawachi *et al.* (1989), reported on p. 115, 1014 cases of stomach cancer, 2043 of colon cancer, 1376 of rectal cancer, 184 of liver cancer, 120 of gall-bladder cancer and 571 of pancreatic cancer were available for analysis. The odds ratios for employment as a woodworker were 1.2 (95% CI, 0.9–1.6) for stomach cancer, 0.7 (0.5–0.9) for colon cancer and 1.1 (0.8–1.4) for rectal cancer.

In a further analysis, Dockerty *et al.* (1991) examined the risk for stomach cancer in a cancer registry-based case-control study in New Zealand. The study base and methods were the same as those described by Kawachi *et al.* (1989). There were 1016 men with stomach cancer available for analysis. The 19 042 controls consisted of registrants with cancer at all other sites. The only information available on exposure was the current or most recent occupation in the register. After adjustment for age, socioeconomic level, ethnic group and smoking, excess risks were observed for foresters and loggers (odds ratio, 1.8; 95% CI, 1.0–3.3) and cabinet-makers (1.4; 0.7–2.8), while decreased risks were observed for wood preparation and pulp and paper workers (0.8; 0.4–1.7) and carpenters (0.8; 0.5–1.2).

González *et al.* (1991) examined the association between occupation and gastric cancer in Spain. The cases were gastric adenocarcinomas diagnosed between 1987 and 1989 at 15 hospitals in Barcelona province, Zaragoza city, Soria province, Lugo province and the north of La Coruña province. Controls, matched on age, sex and area of residence, were selected from among patients at the same hospitals, excluding those with respiratory or gastric cancer, chronic respiratory disease, diabetes or chronic diseases that require a special therapeutic diet. Occupational histories were collected for 354 cases (235 men) and 354 controls by interviewers who were unaware of the case or control status of the patients. Odds ratios were calculated by logistic regression in order to adjust for socioeconomic status and diet. Relative risks were

calculated for people ever employed in forestry (odds ratio, 1.0; 95% CI, 0.3–3.6), wood and paper production (0.5; 0.2–1.7) and furniture and wood manufacture (1.8; 0.5–6.9). The odds ratio for employment in any job with exposure to wood dust was 1.0 (0.4–2.3).

Arbman *et al.* (1993) performed a case–control study of colon and rectal cancer in Sweden, among patients under the age of 75 with histologically confirmed adenocarcinoma of the colon or rectum, who were identified in hospitals in the county of Östergötland in south-eastern Sweden. Two control groups were selected: hospital patients with hernias and anal disorders and a random sample of the general population. A questionnaire, which included information on occupational history, was completed by 98 patients (51 men) with colon cancer, 79 (48 men) with rectal cancer, 371 (309 men) hospital controls and 430 (203 men) general population controls. The odds ratios for men employed as carpenters were 0.5 [95% CI, 0.1–2.7] for colon cancer and 0.9 [0.3–3.2] for rectal cancer. The odds ratios for men employed as forestry workers were 0.9 [0.4–2.0] for colon cancer and 0.5 [0.2–1.5] for rectal cancer. The odds ratios for men employed as sawmill workers were 1.2 [0.4–3.3] for colon cancer and 0.4 [0.1–1.9] for rectal cancer. The prevalence of exposure was very low among women.

Studies on cancers of the digestive tract are summarized in Table 30.

3. Studies of Cancer in Experimental Animals¹

3.1 Inhalation

3.1.1 Rat

Sixteen female Sprague-Dawley rats, 11 weeks of age, were exposed to untreated beech wood dust (approximately 70% of the dust particles with a maximal diameter of about 10 μm and 10–20% of the particles with a diameter of about $\leq 5 \mu\text{m}$) at 25 mg/m^3 for 6 h per day on five days per week for 104 weeks. Surviving animals were killed and autopsied; only the nasal cavities and respiratory tract were examined histologically. No respiratory tract tumour and no squamous metaplastic or dysplastic lesions were found among the 15 surviving animals. About 50% of the animals were reported to have tumours outside the respiratory tract, but the incidence was said not to differ from that in untreated controls (Holmström *et al.*, 1989a). [The Working Group noted the small number of animals and the inadequate reporting of the tumours outside the respiratory tract.]

Fifteen female Wistar rats, four weeks of age, were exposed to beech wood dust (mass median aerodynamic diameter, 7.2 μm ; geometric standard deviation, 2.2 μm) at $15.3 \pm 13.1 \text{ mg}/\text{m}^3$ for 6 h per day on five days per week for six months and were observed for up to 18 months, when survivors were killed. At autopsy, the animals were examined grossly, and

¹The Working Group was aware of studies in progress in which rats are exposed to oak wood dust by inhalation (IARC, 1994b).

Table 30. Community-based case-control studies of cancer of the digestive tract

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Stomach cancer								
<i>(i) Exposure to wood dust</i>								
Siemiatycki <i>et al.</i> (1986); Siemiatycki (1991)	Canada	M	156/1524	Interviews evaluated by panel of chemists and industrial hygienists	'Substantial' exposure < 16 years	1.2	[0.6–2.6]	
			251/2397		≥ 16 years	1.9	[1.0–3.7]	
					Update	1.1	[0.7–1.7]	
<i>(ii) Occupational group</i>								
Dockerty <i>et al.</i> (1991)	New Zealand	M	1016/19 042	Tumour register	Foresters and loggers	1.8	1.0–3.3	Current or most recent occupation; adjusted for age, socioeconomic level and smoking
					Cabinet-makers	1.4	0.7–2.8	
					Wood preparation, pulp and paper workers	0.8	0.4–1.7	
					Carpenters	0.8	0.5–1.2	
Kawachi <i>et al.</i> (1989)	New Zealand	M	1014/18 890	Tumour register	All woodworkers	1.2	0.9–1.6	Same population and methods as Dockerty <i>et al.</i> (1991)
González <i>et al.</i> (1991)	Spain	MF	354/354	Interviews	Any wood dust-exposed job	1.0	0.4–2.3	Cases were adenocarcinomas; controls matched on age, sex and residence; adjusted for diet and socioeconomic status
					Forestry	1.0	0.3–3.6	
					Wood and paper production	0.5	0.2–1.7	
					Furniture/wood manufacture	1.8	0.5–6.9	
Colon cancer								
<i>(i) Exposure to wood dust</i>								
Spiegelman & Wegman (1985)	USA	MF	370/1861	Interviews Job-exposure matrix	Wood (men)	1.3	<i>p</i> = 0.24	Adjusted for age
					Wood (women)	1.5	<i>p</i> = 0.07	
Siemiatycki (1991)	Canada	M	497/2056	Interviews evaluated by panel of chemists and industrial hygienists	Any exposure to wood dust 'Substantial' exposure	1.0 0.9	[0.8–1.2] [0.7–1.2]	

Table 30 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Colon cancer (contd)								
<i>Exposure to wood dust (contd)</i>								
Peters <i>et al.</i> (1989)	USA	M	106/106	Interviews	Wood dust	2.1 1.5 3.6	0.5–8.5 0.3–3.6 0.6–21	Right side of colon Transverse and descending colon Sigmoid colon Subjects aged 25–44; adjusted for age and education
<i>(ii) Occupational group</i>								
Brownson <i>et al.</i> (1989)	USA	M	1993/9965	Tumour register	Carpenters	0.9	0.6–1.4	Adjusted for age
Fredriksson <i>et al.</i> (1989)	Sweden	M	156/306	Questionnaire	Lumbermen Pulp workers Sawmill workers	0.7 0.7 0.5	0.4–1.0 0.3–1.6 0.3–0.9	Adjusted for age and physical activity. Cases were adenocarcinomas.
Kawachi <i>et al.</i> (1989)	New Zealand	M	2043/17 861	Tumour register	All woodworkers	0.7	0.5–0.9	Same population and methods as Dockerty <i>et al.</i> (1991)
Arbman <i>et al.</i> (1993)	Sweden	M	51/512	Questionnaire	Carpenters Forestry workers Sawmill workers	0.5 0.9 1.2	[0.1–2.7] [0.4–2.0] [0.4–3.3]	Cases were adenocarcinomas
Rectal cancer								
<i>(i) Exposure to wood dust</i>								
Spiegelman & Wegman (1985)	USA	MF	551/1861	Interviews and a job–exposure matrix	Wood (men) Wood (women)	1.1 1.5	<i>p</i> = 0.69 <i>p</i> = 0.04	Colon and rectum combined; adjusted for age

Table 30 (contd)

Reference	Country	Sex	Cases/ controls	Source of information on exposure	Exposure to which relative risk applies	OR/RR	95% CI or <i>p</i>	Comments
Rectal cancer (contd)								
<i>Exposure to wood dust (contd)</i>								
Siemietycki (1991)	Canada	M	257/1299	Interviews evaluated by panel of chemists and industrial hygienists	Any exposure to wood dust	1.0	[0.7-1.4]	
					'Substantial' exposure	1.3	[0.8-2.0]	
Peters <i>et al.</i> (1989)	USA	M	41/41	Interviews	Wood dust	9.4	2.0-45	Subjects aged 25-44; adjusted for age and education
<i>(ii) Occupational group</i>								
Kawachi <i>et al.</i> (1989)	New Zealand	M	1376/18 528	Tumour register	All woodworkers	1.1	0.8-1.4	Same population and methods as Dockerty <i>et al.</i> (1991)
Arbman <i>et al.</i> (1993)	Sweden	M	48/512	Questionnaire	Carpenters	0.9	[0.3-3.2]	Cases were adenocarcinomas
					Forestry workers	0.5	[0.2-1.5]	
					Sawmill workers	0.4	[0.1-1.9]	

OR, odds ratio; RR, relative risk; CI, confidence interval; M, male; F, female

lungs, nasal cavities, livers, spleens and kidneys were examined histologically. No respiratory tract tumour was found, and the incidence of tumours outside the respiratory tract did not differ significantly from that in untreated controls (Tanaka *et al.*, 1991). [The Working Group noted the small number of animals in each group and the short exposure.]

3.1.2 Hamster

One group of 12 and one group of 24 male Syrian golden hamsters, 10 weeks of age, were exposed to beech wood dust (approximately 70% of the dust particles with a maximal diameter of about 10 μm and 10–20% of the particles with a diameter of about $\leq 5\text{mm}$) at 15 and 30 mg/m^3 for 6 h per day on five days per week for 36 and 40 weeks, respectively. At these times, the survivors were killed and autopsied; nasal cavities, respiratory tracts, livers and kidneys were examined histologically. No respiratory tract tumour was reported in the 12 animals exposed to 15 mg/m^3 , but 1/22 hamsters exposed to 30 mg/m^3 had an unclassifiable infiltrating malignant nasal tumour [not significant], and one other animal in this group had cuboidal metaplasia with mild dysplasia of the nasal epithelium (Wilhelmsson *et al.*, 1985a,b). [The Working Group noted the short duration of the experiment.]

3.2 Intraperitoneal injection

Rat: In a preliminary report on a study of the carcinogenic activity of various fibrous and granular dusts, one group of female Wistar rats [initial number unspecified], eight weeks of age, received three weekly intraperitoneal injections of beech wood dust [size of dust particles unspecified and total dose ambiguously reported as 250 or 300 mg/animal] suspended in 0.9% sodium chloride solution (50 mg wood dust/ml). The surviving animals were killed 140 weeks after the first treatment [survival time not clearly specified]. At post-mortem examination of the abdominal cavity, no mesothelioma or sarcoma was found in the 52 rats examined (Pott *et al.*, 1989). [The Working Group noted the limited reporting of the experimental details and that UICC chrysotile asbestos induced mesotheliomas when similarly administered in parallel groups.]

3.3 Administration with known carcinogens or other modifying factors

3.3.1 Rat

Four groups of 16 female Sprague-Dawley rats, 11 weeks old, were exposed by inhalation in chambers to (i) air (controls); (ii) about 25 mg/m^3 untreated beech wood dust (approximately 70% of the dust particles with a maximal diameter of about 10 μm and 10–20% of the particles with a diameter of about $\leq 5\text{mm}$); (iii) 12.4 ± 1.1 ppm [14.9 ± 1.3 mg/m^3] formaldehyde; or (iv) beech wood dust (as above) plus 12.7 ± 1.0 ppm [15.2 ± 1.2 mg/m^3] formaldehyde for 6 h per day on five days per week for 104 weeks. No difference in the mortality rates was reported between the groups at any time during the study [mortality rates and statistical test unspecified]. Surviving animals were killed and autopsied; only nasal cavities and respiratory tracts were examined histologically. One respiratory tract tumour, a nasal squamous-cell carcinoma, was

found in the group exposed to formaldehyde alone. Pronounced squamous metaplasia, with or without keratinization of the nasal epithelium at the level of the naso- and maxillary turbinates, was found in 9/16 rats exposed to formaldehyde and in 8/15 rats exposed to wood dust plus formaldehyde. In addition, pronounced squamous metaplasia accompanied by dysplasia of the nasal epithelium occurred in 1/16 rats exposed to formaldehyde and in 4/15 rats exposed to wood dust plus formaldehyde. No such metaplastic or dysplastic nasal cavity lesions were encountered in any of the controls or in rats exposed to wood dust alone. Tumours outside the respiratory tract were reported to affect about 50% of the animals, but this incidence was said not to differ from that in controls (Holmström *et al.*, 1989a). [The Working Group noted the small number of animals in each group and the inadequate reporting of tumours outside the respiratory tract.]

Two groups of 20 male Wistar rats, four weeks old, were exposed by inhalation in chambers to clean air (controls) or to beech wood dust (mass median aerodynamic diameter, 7.2 μm ; geometric standard deviation, 2.2 μm) at $15.3 \pm 13.1 \text{ mg/m}^3$ for 6 h per day on five days per week for six months (total exposure, 666 h). Immediately thereafter, five rats from each group were exposed to sidestream cigarette smoke (from 10 cigarettes per day) at 10.2 mg/m^3 [standard deviation unspecified] for 2 h per day on five days per week for one month (total exposure, 40 h). After clearance periods of 12 months for rats exposed only to wood dust and 11 months for rats exposed to wood dust plus cigarette smoke (i.e. 18 months after the start of the experiment), all rats, including the controls, were killed. At autopsy, animals were examined grossly, and lungs, nasal cavities, livers, spleens and kidneys were examined histologically. No intercurrent mortality occurred, and no tumours of the nose or of other segments of the respiratory tract were observed. The incidence of tumours outside the respiratory tract did not differ significantly from that in untreated controls (Tanaka *et al.*, 1991). [The Working Group noted the small number of animals in each group and the relatively short treatment and observation periods.]

3.3.2 Hamster

Two groups of 12 male Syrian golden hamsters, about 10 weeks old, were exposed by inhalation in chambers to air (controls) or to untreated beech wood dust (about 70% of the particles had a maximal diameter of about 10 μm , and 10–20% of the particles had a diameter of about $\leq 5 \mu\text{m}$) at 15 mg/m^3 (range, 10–20 mg/m^3) for 6 h per day on five days per week for 36 weeks. A further two groups of hamsters were treated similarly but were also given *N*-nitrosodiethylamine (NDEA) at 1.5 mg/animal by subcutaneous injection, weekly for the first 12 consecutive weeks. All survivors were killed at week 36. No tumours of the nose were observed in 12 hamsters exposed to wood dust alone. Tracheal squamous-cell papillomas occurred in 1/7 controls, 0/8 hamsters exposed to wood dust alone, 3/8 hamsters treated with NDEA alone and 4/8 hamsters exposed to wood dust and NDEA (Wilhelmsson *et al.*, 1985a,b). [The Working Group noted the short duration of the experiment, the small numbers of animals in each group, the absence of data on mortality rates and the high losses of animals and tissues due to cannibalism.]

Two groups of 24 male Syrian golden hamsters, about 10 weeks old, were exposed by inhalation in chambers to air (controls) or to untreated beech wood dust (about 70% of the particles with a maximal diameter of about 10 μm , and 10–20% of the particles with a diameter of about $\leq 5 \mu\text{m}$) at 30 mg/m^3 (range, 25–35 mg/m^3) for 6 h per day on five days per week for 40 weeks. A further two groups of hamsters were treated similarly but were also given 3.0 mg/animal NDEA by subcutaneous injection, weekly for the first 12 consecutive weeks. The survivors were killed at week 40. The death rate was very high in all groups and significantly higher in the two NDEA-treated groups than in the two other groups ($p < 0.05$; Fisher's exact test) [death rates not further specified]. One of the 22 hamsters exposed to wood dust alone had an unclassifiable, malignant, infiltrating nasal tumour, and another hamster in this group had focal cuboidal metaplasia with mild dysplasia of the nasal epithelium. No neoplastic, dysplastic or metaplastic changes occurred in the respiratory tracts of controls. The types and incidences of respiratory tract neoplasms and dysplasia in the groups exposed to NDEA and to wood dust plus NDEA were as follows: nasal tumours (papillomas and adenocarcinomas), 10/22 (46%) and 11/21 (52%); laryngeal and/or tracheal tumours (papillomas), 10/19 (53%) and 11/18 (61%); lung tumours (adenocarcinoma), 0/19 and 1/18 (6%); and nasal dysplasia, 8/18 (44%) and 4/17 (24%). The incidences of these respiratory tract lesions did not differ significantly between these two groups [Fisher exact test] (Wilhelmsson *et al.*, 1985a,b).

3.4 Skin application of wood dust extracts

Mouse: Four groups of 70 young female NMRI mice [age unspecified], weighing 25–30 g, received skin applications of a mutagenic fraction of a methanol extract of dust from untreated, semi-dry beech wood in 30 μl acetone on a 1–2- cm^2 shaven area of the lower back twice a week for three months. The freshly prepared, weekly doses of the fraction were equivalent to 2.5, 5, 7.5 and 10 g wood dust per mouse. Five similar groups of mice served as controls: one was treated with acetone on the shaven skin, one was shaved only and one was neither shaved nor treated with acetone; two positive control groups were treated with 5 and 10 μg benzo[*a*]pyrene, respectively. All mice were observed until they died naturally or were killed to avoid severe suffering. The survival of treated mice was not significantly different from that of untreated mice ($p = 0.571$; Mann-Whitney U test). The positive controls and mice treated with the mutagenic wood dust extract developed precancerous skin lesions (epithelial hyperplasia and hyperkeratosis) and benign and malignant tumours of the skin and mammary glands just beneath the treated skin area (see Table 31). Comparison of the mice treated with wood dust with the negative controls was reported to show a significant overall carcinogenic effect ($p < 0.01$; χ^2 test) (Mohtashamipur *et al.*, 1989b). [The Working Group noted that a significant trend is observed for skin tumours, whether or not the analysis includes the keratoacanthoma and the papillary cystadenoma. The trend test for mammary tumours is significant when mammary gland adenocarcinomas, the adenoacanthoma and the mixed mammary tumours are grouped, and it is marginally significant when only the adenocarcinomas and the adenoacanthoma are considered.]

Table 31. Results of application to the skin of mice of mutagenic fractions of a methanol extract of dust from untreated, semi-dry beech wood, with negative and positive controls

Tumour	Negative controls			Extract (g)				Benzo[a]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 carcinoma	-	-	-	1	-	-	1 ^a	1	15
Skin squamous-cell papilloma	-	-	-	1	1	6	5 ^a	2	5
Skin keratoacanthoma	-	-	-	-	-	1	-	-	2
Skin papillary cystadenoma	-	-	-	-	1	-	-	-	-
Sebaceous gland adenoma	-	-	-	-	-	-	-	2	-
Mammary gland adenocarcinoma	-	-	-	-	4	3	2 ^{b,c}	1	1
Mammary gland adenoacanthoma	-	-	-	-	-	-	1 ^{b,c}	-	-
Mammary gland mixed tumours	-	-	-	-	-	-	2 ^b	-	-
Fibrosarcoma	-	-	-	-	-	1	-	-	-
Haemangiosarcoma	-	-	-	-	1	-	-	-	-
Neurofibrosarcoma	-	-	-	-	1	-	-	-	-
Lymphoma	-	-	-	-	-	-	1	-	-
Anaplastic carcinoma	-	-	-	-	1	-	-	-	-
Precancerous skin lesions	-	1	2	2	4	8	6	13	18

Adapted from Mohtashamipur *et al.* (1989b), numbers of animals given are effective numbers

^a[$p < 0.01$; 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 both squamous-cell carcinomas and papillomas, or papillomas alone

^b[$p < 0.02$; Cochran-Armitage test for trend if included in the analysis] where comparisons are made for 0, 2.5, 5.0, 7.5 and 10 g extract groups, including mammary gland adenocarcinoma, adenoacanthoma and mixed mammary gland tumours

^c[$p < 0.06$; Cochran-Armitage test for trend] where comparisons are made for 0, 2.5, 5.0, 7.5 and 10 g extract groups and only mammary gland adenocarcinoma and adenoacanthoma are considered

3.5 Experimental data on wood shavings

It has been suggested in several studies that cedar wood shavings, used as bedding for animals, are implicated in the prominent differences in the incidences of spontaneous liver and mammary tumours in mice, mainly of the C3H strain, maintained in different laboratories (Sabine *et al.*, 1973; Sabine, 1975). Others (Heston, 1975) have attributed these variations in incidence to different conditions of animal maintenance, such as food consumption, infestation with ectoparasites and general condition of health, rather than to use of cedar shavings as bedding. Additional attempts to demonstrate carcinogenic properties of cedar shavings used as bedding material for mice of the C3H (Vlahakis, 1977) and SWJ/Jac (Jacobs & Dieter, 1978) strains were not successful. In none of these studies were there control groups not exposed to cedar shavings.

4. Other Data Relevant to an Evaluation of Carcinogenicity and its Mechanisms

4.1 Deposition and clearance

4.1.1 Humans

No studies of the deposition of wood dust in human airways were available to the Working Group. Particle deposition in the airways has been the object of several studies (for reviews, see Brain & Valberg, 1979; Warheit, 1989). Large particles ($> 10 \mu\text{m}$) are almost entirely deposited in the nose; the deposition of smaller particles depends on size but also on flow rates and type of breathing (mouth or nose); there is also inter-individual variation (Technical Committee of the Inhalation Specialty Section, Society of Toxicology, 1987). Particles deposited in the nasal airways are removed by mucociliary transport (for reviews, see Proctor, 1982; Warheit, 1989).

4.1.2 Experimental systems

No data on the deposition or clearance of wood dusts in animals were available to the Working Group.

4.2 Toxic effects

4.2.1 Humans

(a) Effects on the nose

In a cross-sectional study in eight furniture factories in Denmark, 68 workers were exposed to total dust at concentrations $> 5 \text{ mg/m}^3$ in 63% of the measurements (Solgaard & Andersen, 1975; Andersen *et al.*, 1977). The workers were exposed to a variety of hardwoods, including teak, and to pine and composites, including chipboard and Masonite. Analysis of particle size

showed that 33% of the particles were $< 5 \mu\text{m}$. These workers had significantly lower nasal mucociliary transport rates than a group not exposed to dust; there was also a concentration-dependent decrease in the rates of the exposed workers: mucostasis was found in 63% of workers exposed to an average of 25.5 mg/m^3 and in 11% of those exposed to 2.2 mg/m^3 (mean concentration). Of nine subjects with mucostasis re-examined after 48 h with no exposure to wood, three still had mucostasis, while the six others had clearance rates within normal limits. Middle-ear inflammation and common colds were more frequently reported by people exposed to concentrations $> 5 \text{ mg/m}^3$ than among those exposed to lower levels.

The nasal mucociliary transport-rate was investigated in nine woodworkers, 48–66 years of age, with 6–27 years of employment in the furniture industry in England (Black *et al.*, 1974). They had slower rates than 12 people not exposed to wood dust. Only the worker with the shortest length of employment (six years) had a clearance rate within normal limits. Three workers had complete stasis. The results of cytological examination of nasal smears were reported only for the exposed workers: squamous cells were found in four workers, cuboidal cells in one and 'less mature basal cells' in another.

Boysen and Solberg (1982) studied 103 workers in five Norwegian furniture factories in a cross-sectional study. The subjects constituted about 60% of workers who had been employed for at least 16 years. Ten retired workers and 54 people without nasal disease or an occupation associated with nasal cancer, who were not employed in woodworking industries, were examined. Nasal biopsy samples taken from the middle turbinate showed metaplastic squamous epithelium in 40% of the furniture workers and 17% of controls; the corresponding figures for dysplasia were 12 and 2%, respectively. Mechanical processing of wood was associated with histological changes of the nasal mucosa. Dysplasia occurred in four of 15 furniture workers with exposure mainly to birch, spruce and pine and in nine of 84 exposed mainly to hardwoods.

Nasal biopsy samples taken from the middle turbinate of 44 workers who had been exposed for 10–43 years to softwood dust but not to hardwood dust showed more changes than biopsy samples taken from age-matched men without nasal disease or an occupation associated with nasal cancer (mean score, 2.0 versus 1.4; $p < 0.05$) (Boysen *et al.*, 1986). Four woodworkers and no control had dysplasia; these four men had been exposed for 20 years (one man) and more than 26 years (three men). Nasal symptoms were more frequent among the furniture workers (14% versus 4%; $p < 0.05$).

Biopsy samples were taken from the nasal mucosa of the middle turbinate, at least 5 mm behind the anterior curvature, of 45 randomly selected workers in five furniture factories and one parquet flooring factory and 17 hospital workers in Sweden (Wilhemsson & Lundh, 1984). The mean length of exposure was 15 years (range, 1–39 years). Metaplastic cuboidal epithelium was significantly more prevalent among the woodworkers (26/45 versus 4/17; $p < 0.05$), and columnar epithelium was significantly less frequent (34/45 versus 17/17; $p < 0.05$). The prevalence of metaplastic squamous epithelium was not significantly increased (9/45 versus 4/17), and that of goblet-cell hyperplasia was somewhat more frequent (10/45 versus 1/17).

Cuboidal metaplasia of the nasal mucosa was found in 19 of 22 cases of ethmoidal adenocarcinoma associated with exposure to wood dust in Sweden (Wilhemsson *et al.*, 1985c). Histological examination of non-tumour nasal mucosa from 22 woodworkers with ethmoidal

adenocarcinoma, who had been exposed to wood dust for an average of 38 years (range, 18–55 years), showed cuboidal metaplasia in 19; 16 also had dysplasia. A transitional zone with dysplastic cuboidal epithelium in continuity with the tumour was observed in 10 cases. Squamous metaplasia was also seen in five cases, but there were no cases of squamous dysplasia.

A cross-sectional study in Germany involved 149 male workers with at least 15 years' exposure to wood dust in different industries and 33 workers with no exposure to dust or chemicals (controls); people who had worked as farmers, welders or metal workers or were exposed to cement dust were excluded. Current exposure to wood dust varied between $< 1 \text{ mg/m}^3$ and $> 5 \text{ mg/m}^3$. Mucociliary clearance was not significantly different in workers exposed to unprocessed woods (oak, beech and softwood) and controls. Woodworkers with concomitant exposure to formaldehyde or chromium had decreased clearances ($p = 0.04$ and 0.01 , respectively), and workers exposed to particle-board had slower mucociliary clearance. The findings in nasal biopsy samples taken from the middle turbinate were reported for various single cell types (columnar-cell hyperplasia, squamous-cell metaplasia, cuboid metaplasia) and mixed cell types (Wolf *et al.*, 1994). [The Working Group noted the inadequate reporting of the histological classification and the high prevalence of squamous epithelial metaplasia in the control group. The Group analysed the data according to hyperplasia and metaplasia in single cell types and found no significant differences between woodworkers and controls (Table 32). The odds ratios for woodworkers exposed to softwood or hardwood, but no additives, were 2.2 (95% CI, 0.81–6.2) for cuboid metaplasia, 0.40 (0.16–1.0) for squamous-cell metaplasia and 1.3 (0.47–3.5) for columnar-cell hyperplasia. Cuboid metaplasia was commoner in workers exposed to hardwood (3.5; 1.1–12), softwood (3.1; 0.77–12) or particle-board (2.5; 0.70–8.8) without additives than in controls but was significant only for workers exposed to hardwood.]

In a cross-sectional study of workers in furniture factories in Sweden who were exposed to formaldehyde alone and to formaldehyde plus wood dust, nasal discomfort was more frequent than in clerks (Holmström & Wilhemsson, 1988). The mean combined exposure to wood dust was 1.7 mg/m^3 and that to formaldehyde was 0.25 mg/m^3 ; however, the prevalence of symptoms was similar in workers exposed to formaldehyde alone (mean concentration, 0.26 mg/m^3). Impaired mucociliary clearance in the nose was seen in 15% of the group exposed to wood dust plus formaldehyde, in 3% of controls and in 20% of those exposed to formaldehyde alone exposure ($p < 0.05$). Workers exposed to formaldehyde alone or to formaldehyde plus wood dust had significantly ($p < 0.01$) decreased sensitivity in an olfactory test in comparison with controls. Marked histological changes were seen in the nasal mucosa of 25% of people exposed only to formaldehyde (score, 2.2; $p < 0.05$), but the difference between those exposed to both formaldehyde and wood dust (64% ($p < 0.01$); score, 2.1) and the controls (53% ($p < 0.01$); score, 1.6) was not significant. No correlation was found between histological score and either duration or concentration of exposure (Holmström *et al.*, 1989b).

A total of 676 workers in 50 Swedish furniture factories were classified according to exposure to wood dust as 'heavily/moderately' or 'slightly/non-exposed' (Wilhemsson & Drettner, 1984) [the details of the classification were not reported]. Nasal hypersecretion (20%

Table 32. Frequency of histological findings in nasal biopsy samples from German woodworkers according to exposure

Type of wood	Additives ^a	No. ^b	Histological finding										
			Columnar-cell hyperplasia (1)	Squamous metaplasia (2)	Cuboid metaplasia (3)	Columnar hyperplasia, squamous (4)	Columnar hyperplasia, cuboid (5)	Squamous metaplasia, cuboid (6)	Any columnar hyperplasia (1+4+5) ^c	Any squamous metaplasia (2+4+6) ^c	Any cuboid metaplasia (3+5+6) ^c	Normal	Dysplasia
Hardwood	No	31	6	1	5	3	1	6	10	10	12	9	3
Softwood	No	17	2	1	4	2	1	1	8	4	6	6	0
Particle-board	No	26	5	5	4	4	3	1	13	10	8	4	0
Softwood	Yes	19	3	9	3	2	1	0	6	11	4	1	1
Hardwood	Yes	21	7	4	2	2	1	1	10	7	4	4	1
Mixed	Yes	30	9	5	3	7	0	3	16	15	6	3	2
Controls	-	33	2	12	3	3	3	1	6	14	5	9	1

From Wolf *et al.* (1994)

^a Glues, solvents, etc.

^b The Working Group noted that the total numbers of men and findings were different, indicating that some people were not biopsied.

^c Calculated by the Working Group

versus 12%; $p < 0.05$), obstruction (40% versus 30%; $p < 0.05$) and more than two common colds per year (21% versus 9%; $p < 0.05$) were reported more often in subjects with heavy/moderate exposure than in the other group.

A cross-sectional study was conducted of 101 woodworkers and 73 people not exposed to dust in Germany. The concentrations of dust were measured for each of the men [method of sampling was not reported]: 14 were exposed to $< 5 \text{ mg/m}^3$, 15 to $5\text{--}9 \text{ mg/m}^3$, 36 to $10\text{--}19 \text{ mg/m}^3$ and 36 to $\geq 20 \text{ mg/m}^3$. An increased frequency of hyperplasia and reddening of the nasal mucosa was seen in the exposed workers (50–86% versus 7% in controls) (Ruppe, 1973). Radiographic signs of sinusitis were found in 25% of the woodworkers and 5% of controls. Cough, with or without phlegm (50% versus 11%), and conjunctivitis (15% versus 0%) were also reported more frequently among the exposed workers [significance values not reported].

In a cross-sectional study of the frequency of pulmonary and nasal symptoms in 168 woodworkers and 298 workers with no significant exposure to wood dust in furniture factories in South Australia (Pisaniello *et al.*, 1992), the mean concentration of hardwood dust was 3.8 mg/m^3 , and the mean concentration of softwood dust produced by machining particle-board and medium-density fibre-board was 3.3 mg/m^3 . There was a significant association (odds ratio, 2.2; 95% CI, 1.2–4.2) between exposure to hardwood dust and two or more nasal symptoms, after adjustment for smoking and age.

In a cross-sectional study, Goldsmith and Shy (1988) examined 55 people exposed to hardwood dust in the furniture industry in the United States. The mean length of employment in this industry was 16.6 years, and the current concentration of dust was $\leq 2 \text{ mg/m}^3$. Frequent sneezing and eye irritation were commoner in these workers than in workers with no exposure to wood dust or finishes (prevalence odds ratios, 4.1 and 4.0; $p < 0.05$) in an analysis with adjustment for age, sex and smoking habits. Significant differences were reported for nasal obstruction (61% versus 21%), nasal discharge (41% versus 13%) and sneezing (77% versus 32%).

Symptoms in the upper and lower airways were reported more frequently among 44 randomly selected woodworkers, exposed to concentrations of $1.0\text{--}24.5 \text{ mg/m}^3$ dust, than among 38 office workers examined in a cross-sectional study in New Zealand (Norrish *et al.*, 1992).

The effects of exposure to wood dust on the nose are summarized in Table 33.

(b) *Effects on the lung*

There are several case reports of asthma due to exposure to wood dust (for reviews, see Kadlec & Hanslian, 1983; Goldsmith & Shy, 1988). The asthmatic responses to western red cedar (Chan-Yeung, 1982, 1994) and eastern white cedar (Cartier *et al.*, 1986) are elicited by plicatic acid.

Cough (odds ratio, 2.2; $p < 0.001$), dyspnoea (2.5; $p < 0.001$) and asthma (2.7; $p < 0.001$) were reported more frequently among 652 western red cedar mill workers than among 440 office workers in a cross-sectional study in Canada (Chan-Yeung *et al.*, 1984). Impairment of pulmonary function, as measured by forced expiratory volume in 1 sec (FEV_1), forced vital capacity (FVC), forced mid-expiratory flow between 25 and 75% of FVC ($\text{FEF}_{25\text{--}75\%}$) and

Table 33. Effects (other than cancer) of exposure to wood dust on the nose

Study population		Geographical area	Industry	Wood type	Dust concentration in air (mg/m ³)	Particle size, characteristic	Period of exposure (years)	Nasal effects		Reference																					
Exposed (age, years)	Controls (age, years)							Nasal region	Effect																						
68 men (17-66)	66 men	Denmark, Aarhus county	Eight wood-working factories	Teak, oak, chipboard, palisander and other woods	> 5 in 63% of measurements	5-10 µm maximum	1-51; mean, 16		Mucostasis: 15% in controls, 38% in exposed, 63% in exposed with dust ≥ 10 mg/m ³ (n = 17)	Solgaard & Andersen (1975); Andersen <i>et al.</i> (1977)																					
9 (48-66)	12 (31-69)	England, High Wycombe area	One wood-working factory				6-27		Mucociliary clearance of polystyrene particles in controls, 6.8 (1.9-18.5) mm/min. Mucostasis in 7/9 exposed. In workers, nasal mucosa was normal columnar (3); normal + squamous cells (1); normal + cuboidal (1); normal + squamous metaplasia (3); normal + less mature 'basal' cells (1). Results for controls not given	Black <i>et al.</i> (1974)																					
103 active (32-69); 10 retired (68-81)	54 (35-79)	Norway, western	Five furniture factories	Birch, beech, oak, pine, mahogany, teak, chipboard (made of pine and spruce)			Active, 16-57; mean, 34; retired, 28-57; mean, 44	Anterior curvature of middle turbinate	Rhinascopy: hyperplastic rhinitis: 5 controls, 37 workers (p < 0.05); mucosal polyps: 1 controls, 3 workers Histological score: controls, 1.5; all workers, 2.4 (p < 0.05); active workers, 2.4; retired workers, 2.9	Boysen & Solberg (1982)																					
44 (29-64)	37 men (35-66)	Norway	Six furniture factories	Exclusively softwood			10-43; mean, 24	Anterior curvature of middle turbinate	Histological score <table border="1"> <thead> <tr> <th></th> <th>Controls</th> <th>Workers</th> </tr> </thead> <tbody> <tr> <td>All</td> <td>1.4</td> <td>2.0 (p < 0.05)</td> </tr> <tr> <td>Age ≤ 44</td> <td>1.2</td> <td>1.6</td> </tr> <tr> <td>Age 44-54</td> <td>1.4</td> <td>2.4</td> </tr> <tr> <td>Age ≥ 55</td> <td>1.6</td> <td>1.9</td> </tr> <tr> <td>Smokers</td> <td>1.6</td> <td>2.4 (p < 0.05)</td> </tr> <tr> <td>Non-smokers</td> <td>1.3</td> <td>1.6</td> </tr> </tbody> </table>		Controls	Workers	All	1.4	2.0 (p < 0.05)	Age ≤ 44	1.2	1.6	Age 44-54	1.4	2.4	Age ≥ 55	1.6	1.9	Smokers	1.6	2.4 (p < 0.05)	Non-smokers	1.3	1.6	Boysen <i>et al.</i> (1986)
	Controls	Workers																													
All	1.4	2.0 (p < 0.05)																													
Age ≤ 44	1.2	1.6																													
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Age ≥ 55	1.6	1.9																													
Smokers	1.6	2.4 (p < 0.05)																													
Non-smokers	1.3	1.6																													
45 (mean, 40)	17 (mean, 39)	Sweden, Småland county	Five furniture factories		0.3-5.1; mean, 2.0		1-39; mean, 15	Middle turbinate in widest nasal cavity	Columnar epithelium: 14/17 controls, 23/45* workers ciliated: 12/17 controls, 16/45 workers (p < 0.05); unciliated, 2/17 controls, 7/45 workers; Cuboidal epithelial metaplasia, 1/17 controls, 16/45 workers (p < 0.05) Squamous epithelial metaplasia, 2/17 controls; 6/45 workers	Wilhelmsson & Lundh (1984)																					

Table 33 (contd)

Study population		Geographical area	Industry	Wood type	Dust concentration in air (mg/m ³)	Particle size, characteristic	Period of exposure (years)	Nasal effects		Reference
Exposed (age, years)	Controls (age, years)							Nasal region	Effect	
22 men with ethmoidal adenocarcinoma (57-86)		Sweden	14 furniture makers, 3 french polishers, 2 boat builders, 2 wood machinists, 1 woodwork teacher				18-55; mean, 38		19 cases of cuboidal metaplasia, 16 with dysplasia 10 cases of transitional zone with dysplastic cuboidal epithelium in continuity with the tumour 5 cases of squamous metaplasia	Wilhelmsson <i>et al.</i> (1985c)
100 exposed to wood dust and HCHO (mean, 40.5)	36 (mean, 39.7)	Sweden	Furniture workers	Particle-board	Wood, 1.65±1.06; HCHO, 0.25±0.05	82% < 5 µm	1-30; mean, 9		Nasal discomfort: 6 controls, 53 wood + HCHO, 45 HCHO; eye discomfort: 2 controls, 21 wood + HCHO, 17 HCHO; deep airway discomfort: 5 control, 39 wood + HCHO, 31 HCHO; frequent headache: 2 control, 17 wood + HCHO, 17 HCHO	Holmström & Wilhelmsson (1988)
70 exposed to HCHO (mean, 36.0)			Chemical plant		0.05-0.5		1-36; mean, 10.4			
89 exposed to wood dust and HCHO	32	Sweden	Furniture workers	Particle-board	Wood, 1.65±1.06; HCHO, 0.25±0.05	82% < 5 µm	1-30; mean, 9	Median or inferior aspect of middle turbinate	Nasal biopsy scores: control, 1.56; wood + HCHO, 2.07; HCHO, 2.16 (<i>p</i> < 0.05)	Holmström <i>et al.</i> (1989b)
62 exposed to HCHO			Chemical plant		0.05-0.5		1-36; mean, 10.4			
484 with heavy or moderate exposure	192 with light or no exposure	Sweden, Småland county	50 furniture factories		Mean, 2.0; range, 0.30-5.06		1-60; mean, 27		Nasal hypersecretion: 12% light or no exposure 20% moderate/heavy exposure Nasal obstruction: 30% light or no exposure 40% moderate/heavy exposure	Wilhelmsson & Drettner (1984)

Table 33 (contd)

Study population		Geographical area	Industry	Wood type	Dust concentration in air (mg/m ³)	Particle size, characteristic	Period of exposure (years)	Nasal effects		Reference
Exposed (age, years)	Controls (age, years)							Nasal region	Effect	
101 (18-65)	73 (18-65)	Germany			≤ 5-≥ 20				Ruppe (1973)	
								Sneezing: 0/73 controls 7/14 < 5 mg/m ³ 11/15 5-9 mg/m ³ 32/36 10-19 mg/m ³ 30/36 ≥ 20 mg/m ³ Mucosal changes: 5/73 controls 7/14 < 5 mg/m ³ 8/15 5-9 mg/m ³ 31/36 10-19 mg/m ³ 25/36 ≥ 20 mg/m ³		
134 (mean, 33.6)	298 (mean, 40.1)	South Australia	15 furniture factories	Oak, teak, nyardoh, radiase pine, particle-board, fibre-board	Hardwood dust, 3.2; particle-board, fibre-board, 3.3			Two or more nasal symptoms (out of five): exposure to hardwood dust, odds ratio, 2.2 (1.2-4.2)	Pisaniello <i>et al.</i> (1992)	
149 men (> 35)	33	Germany	Woodworkers	Oak, beech, softwood, particle-board	< 1-→ 5	≥ 15	Middle turbinate	Mucociliary clearance longer in workers exposed to particle-board dust Columnar-cell hyperplasia: all wood workers versus controls, odds ratio, 4.4 (<i>p</i> = 0.05) Squamous-cell metaplasia: woodworkers versus controls, odds ratio, 0.37 (<i>p</i> = 0.02) Cuboid metaplasia: all woodworkers versus controls, odds ratio, 2.9 (<i>p</i> = 0.3)	Wolf <i>et al.</i> (1993)	

Table 33 (contd)

Study population		Geographical area	Industry	Wood type	Dust concentration in air (mg/m ³)	Particle size, characteristic	Period of exposure (years)	Nasal effects		Reference
Exposed (age, years)	Controls (age, years)							Nasal region	Effect	
44 men (mean, 47.2) 11 women (mean, 41.3)	12 men (mean, 42.2) 4 women (mean, 44.5) and 7 men and 14 women in a finishing department	USA, North Carolina	One furniture factory	Hardwoods, fibre-board			Men, mean, 18.5; women, mean, 9	Frequent sneezing; odds ratio, 4.1 (1.1-15)	Goldsmith & Shy (1988)	
44 men (mean, 33)	38 men (mean, 33)	New Zealand	11 furniture and joinery facilities	Rimu wood, kauri, tawa, medium-density fibre, Californian red wood	1-25.4			Nasal obstruction: 27/44 versus 8/38, $p < 0.01$ Nasal discharge: 12/44 versus 5/38, $p < 0.01$ Sneezing: 34/44 versus 12/38, $p < 0.01$	Norrish <i>et al.</i> (1992)	

* $p < 0.05$

FEV₁/FVC, was significantly correlated ($p < 0.001$) with increasing length of employment in cedar mills. The odds ratios were adjusted for smoking, race and age.

Occupational asthma was diagnosed in 10 of 73 workers exposed to red cedar dust in a cross-sectional investigation in the United States, which also included 132 mill workers and 22 clerks and engineers not exposed to wood dust (Brooks *et al.*, 1981). The mean concentration of total dust was 4.7 mg/m³. Pulmonary diseases (chronic bronchitis, occupational asthma, chronic nonspecific airways disease and non-occupational asthma) were commoner among the workers than among controls (34% versus 16%) [p value not reported]. The prevalence of chronic bronchitis in workers exposed to a mixture of woods, mainly Douglas fir, West Coast hemlock and red alder, was similar to that of the workers exposed to western red cedar.

In the study of Norrish *et al.* (1992), described on p. 177, differences were reported for persistent cough in winter (30% versus 5%; $p < 0.01$) and work-related cough (32% versus 0%; $p < 0.01$). Five woodworkers were identified as having occupational asthma. The authors stated that adjustment for smoking did not alter the results.

In a cohort study based on census data on occupational title in Sweden, the rate of mortality from asthma was greater in woodworking machine operators (SMR, 2.3; 95% CI, 1.1–3.4), after adjustment for smoking (Torén *et al.*, 1991).

Ávila (1972) reported on 23 Portuguese cork workers with bronchial asthma, all of whom gave positive responses in inhalation tests for immediate and late reactions to a skin prick with cork. A further 12 cork workers with diseases affecting mainly peripheral gas-exchange tissues all gave positive responses in skin tests for late (type III, arthris) reaction to cork; they showed diffuse, fine miliary mottling on chest radiographs, which disappeared within five weeks, except in a few cases where lesions attributable to fibrosis were reported. [The Working Group noted the lack of information on examination procedures].

The exposure of 334 workers to total dust was determined from job title and job location in a cross-sectional study in Canada (Vedal *et al.*, 1986). The workers were exposed mainly to wood dust from western red cedar. In 78 samples, the total dust concentration ranged from undetectable to 6.0 mg/m³, with a mean of 0.46 mg/m³; 33 workers were considered to be exposed to > 1.0 mg/m³. Spirometric measurements (FVC and FEV₁) gave lower values ($p < 0.05$) for 13 men exposed to concentrations > 2.0 mg/m³; chronic cough, dyspnoea, persistent wheeze and asthma were not related to duration of work or dust concentration.

Al Zuhair *et al.* (1981) studied workers in two furniture factories in the United Kingdom. In the first factory, 53 workers in a sawmill and an assembly department were exposed to dust concentrations of 2.9 and 0.5 mg/m³, respectively. In the second factory, 60 workers on a machine floor and in a cabinet shop were exposed to mean total dust concentrations of 1.4 and 8.3 mg/m³, respectively. These workers had significantly decreased FEV₁ and FVC over the workshift period (0.08–0.12 L; $p < 0.001$), while there was no consistent decrease in lung function over the workshift period among workers in the first factory.

Pulmonary function (FVC, FEV₁, FEV₁/FVC and maximal mid-expiratory flow [MMEF; identical to FEF_{25–75%}]) was determined in 1151 subjects exposed to maple or pine wood dust in a cross-sectional survey in the United States (Whitehead *et al.*, 1981b). Suspended dust concen-

trations were measured in area samples, and a cumulative index of the dose was constructed for each person by multiplying the concentration in the job area by the working time. The workers were classified as having low (0–2 mg-years/m³), medium (2–10 mg-years/m³) or high (10 or more mg-years/m³) exposure to wood dust. The authors classified the results of the spirometric tests as 'normal' or 'impaired' on the basis of external reference values and calculated the odds ratios between different categories of exposure. The ratio for FVC or FEV₁ was not significantly increased with increasing levels of exposure in the groups exposed to maple or pine wood, but FEV₁/FVC and MMEF were lower in people with high exposure. In a comparison of high and low exposure categories, the odds ratios for FEV₁/FVC and MMEF were 3.1 ($p = 0.01$) and 2.1 ($p = 0.02$) for workers exposed to maple dust and 4.0 ($p = 0.01$) and 2.5 ($p = 0.02$) for workers exposed to pine dust, after adjustment for smoking.

In a study of 145 nonsmoking furniture workers and 152 nonsmoking workers in a bottling firm with no exposure to dust in South Africa, cough (40.6% versus 23.7%; $p < 0.01$), phlegm (4.1% versus 10.5%; $p < 0.05$), dyspnoea (18.7% versus 5.7%; $p < 0.05$), wheezing (12.8% versus 4.8%; $p < 0.05$) and nasal symptoms (49.5% versus 18.7%; $p < 0.01$) were two to three times commoner in exposed than unexposed workers (Shamssain, 1992). Spirometric measurements were significantly lower for exposed men than for male controls (FVC: 3.64 versus 4.14 L, $p < 0.001$; FEV₁: 2.65 versus 3.20 L, $p < 0.001$; FEV₁/FVC: 73.2 versus 77.6%, $p < 0.01$; forced mid-expiratory flow between 25 and 75% of FVC [FMF_{25–75%}]: 3.09 versus 3.68 L/s, $p < 0.01$; forced expiratory flow between the first 200 and 1200 ml of FVC [FEF_{200–1200}]: 4.94 versus 7.06 L/s, $p < 0.001$; peak expiratory flow [PEF], 6.14 versus 7.92 L/s, $p < 0.001$); there was no significant difference in these measurements between exposed and unexposed women. The frequency of an FEV₁/FVC below 70% was significantly higher among the woodworkers than the controls (30% versus 17%, $p < 0.01$), and the proportion was higher in men with 10–19 years of employment than in men with 1–9 years of employment (56% versus 27%, $p < 0.01$); 20% of the workers handled pine wood and 80% medium-density fibre-board. The mean total dust concentration in the factory was 3.8 mg/m³.

In the study of Goldsmith and Shy (1988), described on p. 177, peak flow (but no other test of pulmonary function) was correlated with duration in jobs with exposure to wood dust.

The decrease in lung function over a work shift was greater in 50 carpenters and joiners than in 49 hospital workers (Holness *et al.*, 1985). The decreases in FVC were 2.4 ($p = 0.001$) and 0.15% ($p = 0.77$), respectively. The mean total dust concentration was 1.8 mg/m³.

(c) Other effects

Exposure to wood may cause irritant dermatitis, contact urticaria and allergic contact dermatitis (for reviews, see Woods & Calnan, 1976; Hausen, 1986). The contact allergens in a number of woods have been identified, e.g. R-3,4-dimethoxydalbergione was found in a tropical hardwood, *Machaerium scleroxylum* (Beck *et al.*, 1984). Allergic conjunctivitis was reported in a worker exposed to spindle tree dust (Herold *et al.*, 1991).

Of 162 patients with a positive response in a skin prick test to one of 14 woods, 107 had no allergic symptoms (Oehling, 1963).

Inhalation fever and extrinsic allergic alveolitis have been observed in studies of workers exposed to wood contaminated with moulds (Emanuel *et al.*, 1962; Belin, 1987; Dykewicz *et al.*, 1988).

4.2.2 Experimental systems

A mouse hepatoma cell line, Hepa-1, was used to study cytotoxicity (effect on cell growth) and induction of enzymes (cytochrome P450IA1 and aldehyde dehydrogenase). The cells were exposed for 24 h to acetone extracts (final concentration of acetone, 0.5%) of bleached cellulose materials, softwoods (pine and a mixture of pine and spruce) and hardwoods (alder and aspen), all of which are used as bedding materials in cages for small laboratory animals. The softwood and alder extracts (final concentrations corresponding to 1.25–5 mg bedding material/ml cell culture medium) were more cytotoxic to the hepatoma cells than the aspen extract, whereas the bleached cellulose materials were found to be nontoxic at doses up to and including 20 mg/ml. Both softwood and hardwood extracts induced the activity of cytochrome P450IA1 and aldehyde dehydrogenase at concentrations which caused little toxicity (Törrönen *et al.*, 1989).

In order to investigate the toxicity of plicatic and abietic acids, which are constituents of Western red cedar and pine woods, respectively, primary cultures of rat type II cells (isolated from Sprague-Dawley rats [sex unspecified]) and of human lung carcinoma cell line A549 were exposed to solutions of up to 1 mg/ml abietic acid and 5 mg/ml plicatic acid for 2–24 h. A time- and dose-dependent induction of cell lysis was seen with both cell types. Abietic acid was significantly more toxic (first observable effect after 2 h at 0.1 mg/ml) than plicatic acid (first effect after 4 h at 2.5 mg/ml). In studies with cultured tracheal explants from Sprague-Dawley rats [sex unspecified], both compounds produced dose-dependent desquamation of epithelial cells, abietic acid again having a higher toxic potential than plicatic acid (Ayars *et al.*, 1989).

In order to assess the tumorigenic effect of the combination of beech wood dust and formaldehyde (see also section 3.1), groups of 16 female Sprague-Dawley rats (11 weeks old) were exposed by inhalation in whole-body exposure chambers to freshly prepared wood dust (70% with a longest dimension of about 10 μm , 10–20% $\leq 5 \mu\text{m}$) at 25 mg/m³ for 6 h per day on five days per week for 104 weeks, with or without formaldehyde. There was also an untreated control group. Animals were exposed in an inversed 24-h cycle, which ensured that they were as active as possible during exposure. Apart from neoplastic and preneoplastic lesions (see section 3.1), histopathological evaluation showed a greater prevalence of pulmonary emphysema in rats exposed to wood dust than in the control animals ($p < 0.05$), but no differences in mortality rates and no significant difference in the histological appearance of pulmonary epithelium were observed (Holmström, *et al.*, 1989a).

In a study to assess the tumorigenic effects of a combination of beech wood dust and NDEA (see also section 3.1), groups of 19–23 male Syrian hamsters, weighing 90–120 g, received either wood dust or NDEA alone or the combination. The animals were exposed by inhalation in whole-body exposure chambers to particles of fresh beech wood dust (30 mg/m³) for 6 h per day on five days per week over a period of 40 weeks. In the group of hamsters exposed to wood dust alone, slight inflammatory reactions of the respiratory epithelium and

submucosal stroma were detected, which were not observed in the respective control animals (Wilhemsson *et al.*, 1985a,b).

Sixteen male guinea-pigs weighing about 300 g were given a single intratracheal instillation of 75 mg of sheesham or mango wood dust as an autoclaved suspension. Animals were killed 60 and 90 days after treatment. Treatment induced disintegration of giant cells, centrilobular emphysema and slight fibrosis in the lungs at both times (Bhattacharjee *et al.*, 1979).

The enzyme induction activity of shavings from Eastern red cedar and oil of cedarwood was studied indirectly in groups of 6–18 C3H-A, CBA/J and Swiss albino mice [sex unspecified] as barbiturate sleeping time, the time between loss and restoration of the righting reflex after intraperitoneal injection of hexobarbital at 125 mg/kg bw). In five separate experiments in which mice were reared and/or housed with cedar bedding material for at least three weeks, a reduction in sleeping time ($p < 0.01$) was seen, which was attributed to the induction of enzymes responsible for hexobarbital oxidation (Sabine, 1975).

4.3 Genetic and related effects

4.3.1 Humans

Chromosomal aberrations in peripheral lymphocytes were studied in 13 male nonsmokers employed in three plywood factories in Finland, who were reported to be exposed to fumes emitted from heated wood. The controls were 15 male nonsmokers matched for age but not employed in wood industries. The frequency of chromatid breaks was 2.1% in the exposed group and 1.0% in the controls ($p < 0.01$) (Kurttio *et al.*, 1993). [The Working Group noted that exposure to wood dust was not mentioned.]

4.3.2 Experimental systems

Extracts of certain woods prepared by a variety of methods (see Table 34 and section 1.3.2) gave weak positive or borderline effects for reverse mutation in *Salmonella typhimurium*. Unequivocal positive results have been obtained only with beech wood (Mohtashamipur *et al.*, 1986); however, other woods have not been examined to the same extent. [The Working Group noted that wood contains constituents that can reduce the activity of mutagens such as benzo[*a*]pyrene, aflatoxin B₁ and methylmethane sulfonate.] Chemically and bacterially degraded beech wood lignin significantly induced reverse mutation in *S. typhimurium* (Mohtashamipur & Norpoth, 1990), but fumes produced during the drying of birch and spruce wood were not mutagenic to *S. typhimurium* (Kurttio *et al.*, 1990). [The Working Group noted the inappropriate correction for cell survival applied by the authors, which resulted in a different conclusion.]

Cyclohexane–ethanol extracts of beech, oak and particle-board increased the number of DNA single-strand breaks per fragile sites in rat hepatocytes *in vitro* (Schmezer *et al.*, 1994).

Alcoholic extracts of beech wood increased the frequency of micronuclei in the crypts of the small intestine of mice treated by gavage and in the nasal epithelium of rats after topical application (Nelson *et al.*, 1993).

Of several compounds isolated from wood, only quercetin and Δ^3 -carene showed mutagenic activity (Table 35).

Table 34. Genetic and related effects of wood dusts

Test system	Result ^a		Extraction medium	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Ash				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	(+)	(+)	Methanol	McGregor (1982)
Beech				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	(+)	0	Methanol; cyclohexane/water	Brockmeier & Norpoth (1981)
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	0	(+)	Methanol	Mohtashamipur <i>et al.</i> (1984)
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Acetone/water	Kubel <i>et al.</i> (1988)
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	0	+	Methanol/ethyl acetate	Mohtashamipur <i>et al.</i> (1986)
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	0	+	Acetone/water; lignin degradation	Mohtashamipur & Norpoth (1990)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	(+)	Methanol	McGregor (1982)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Acetone/water	Kubel <i>et al.</i> (1988)
DIA, DNA strand breaks, rat hepatocytes <i>in vitro</i>	(+)		Cyclohexane/ethanol	Schmezer <i>et al.</i> (1994)
MVM, Micronucleus induction, mouse duodenal crypts <i>in vivo</i>	+		Methanol/ethyl acetate	Mohtashamipur & Norpoth (1989)
MVR, Micronucleus induction, rat nasal epithelial cells <i>in vivo</i>	+		Methanol/ethyl acetate	Nelson <i>et al.</i> (1993)
Birch				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Drying fumes	Kurttio <i>et al.</i> (1990)
Chestnut				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Acetone/water	Weissmann <i>et al.</i> (1989)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Acetone/water	Weissmann <i>et al.</i> (1989)
Elm				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Methanol	McGregor (1982)

Table 34 (contd)

Test system	Result ^a		Extraction medium	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Limba, obeche and walnut				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	(+)	0	Methanol; cyclohexane/water	Brockmeier & Norpoth (1981)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	(+)	0	Methanol; cyclohexane/water	Brockmeier & Norpoth (1981)
Mahogany				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Methanol; cyclohexane/water	Brockmeier & Norpoth (1981)
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Methanol	McGregor (1982)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Methanol; cyclohexane/water	Brockmeier & Norpoth (1981)
Oak				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	(+)	0	Methanol; cyclohexane/water	Brockmeier & Norpoth (1981)
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Methanol	McGregor (1982)
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Acetone/water	Weissmann <i>et al.</i> (1989)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	(+)	0	Methanol; cyclohexane/water	Brockmeier & Norpoth (1981)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Acetone/water	Weissmann <i>et al.</i> (1989)
DIA, DNA strand breaks, rat hepatocytes <i>in vitro</i>	+		Cyclohexane/ethanol	Schmezer <i>et al.</i> (1994)
Spruce				
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Acetone/water	Kubel <i>et al.</i> (1988)
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Drying fumes	Kurtio <i>et al.</i> (1990)
SA2, <i>Salmonella typhimurium</i> TA102, reverse mutation	-	-	Drying fumes	Kurtio <i>et al.</i> (1990)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Acetone/water	Kubel <i>et al.</i> (1988)

WOOD DUST

Table 34 (contd)

Test system	Result ^a		Extraction medium	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
Spruce (contd)				
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Drying fumes	Kurttio <i>et al.</i> (1990)
DIA, DNA strand breaks, rat hepatocytes <i>in vitro</i>	-	-	Cyclohexane/ethanol	Schmezer <i>et al.</i> (1994)
Particle-board				
DIA, DNA strand breaks, rat hepatocytes <i>in vitro</i>	+	+	Cyclohexane/ethanol	Schmezer <i>et al.</i> (1994)

^a+, considered to be positive; (+), considered to be weakly positive in an adequate study; -, considered to be negative; ?, considered to be inconclusive (variable responses in several experiments within an adequate study); 0, not tested

Table 35. Genetic and related effects of wood-related compounds

Test system	Result ^a		Reference
	Without exogenous metabolic system	With exogenous metabolic system	
Δ^3-Carene			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	(+)	-	Kurttio <i>et al.</i> (1990)
SA2, <i>Salmonella typhimurium</i> TA102, reverse mutation	-	-	Kurttio <i>et al.</i> (1990)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	(+)	-	Kurttio <i>et al.</i> (1990)
Coniferyl alcohol			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
Deoxydopodophyllotoxin			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
2,6-Dimethoxybenzoquinone			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
Eugenol			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	IARC (1985a)
Quercetin			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	0	+	Bjeldanes & Chang (1977)
SA8, <i>Salmonella typhimurium</i> TA1538, reverse mutation	0	(+)	Bjeldanes & Chang (1977)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	0	+	Bjeldanes & Chang (1977)
Scopoletin			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)

Table 35 (contd)

Test system	Result ^a		Reference
	Without exogenous metabolic system	With exogenous metabolic system	
3,4,5-Trimethoxycinnamic acid			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
Vanillic acid			
SA0, <i>Salmonella typhimurium</i> TA100, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)
SA9, <i>Salmonella typhimurium</i> TA98, reverse mutation	-	-	Mohtashamipur & Norpoth (1984)

^a+, considered to be positive; (+), considered to be weakly positive in an adequate study; -, considered to be negative; ?, considered to be inconclusive (variable responses in several experiments within an adequate study); 0, not tested

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Wood is one of the world's most important renewable resources. At least 1700 million m³ are harvested for industrial use each year. Wood dust, generated in the processing of wood for a wide range of uses, is a complex substance. Its composition varies considerably according to species of tree. Wood dust is composed mainly of cellulose, polyoses and 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 glycerols), polar organic extractives (tannins, flavonoids, quinones and lignans) and water-soluble extractives (carbohydrates, alkaloids, proteins and inorganic material).

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

It is estimated that at least two million people are routinely exposed occupationally to wood dust worldwide. Nonoccupational exposure also occurs. The highest exposures 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/m³). Exposure levels above 1 mg/m³ 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 and other measures to control dust.

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 furniture manufacture. Logging, sawmills and plywood and particle-board manufacture generally involve use of trees grown locally. Most of the wood dust (by mass) found in work environments has a mean aerodynamic diameter of more than 5 µm. Some investigators have reported that the dust generated in operations such as sanding and during the processing of hardwoods results in a higher proportion of smaller particle sizes, but the evidence is not consistent.

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. The manufacture of plywood and particle-board may entail exposure to formaldehyde, solvents, phenol, wood preservatives and engine exhausts. Sawmill workers may also be exposed to wood preservatives and fungal spores. 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.

5.2 Human carcinogenicity data

The risk for cancer, and particularly cancer of the nasal cavities and paranasal sinuses, among woodworkers has been investigated in many epidemiological studies. Some of the studies provided specific information on cancer risk associated with exposure to wood dust, and those studies were given greatest weight in the evaluation.

Most of the available cohort and case-control studies of cancer of the nasal cavities and paranasal sinuses have shown increased risks associated with exposure to wood dust. These findings are supported by numerous case reports. Very high relative risks for adenocarcinoma at this site, associated with exposure to wood dust, have been observed in many countries, particularly in Europe. The lower risks observed in the studies in the United States may be due to differences in concentration or type of wood dust, but in one of these studies the more heavily exposed groups had significantly increased risks. A pooled analysis of 12 case-control studies revealed a clearly increasing risk with increasing estimated levels of exposure to wood dust, overall and in most individual studies. The excess appears to be attributable to wood dust *per se*, rather than to other exposures in the workplace, since the excess was observed in various countries during different periods and among different occupational groups, and because direct exposures to other chemicals do not produce relative risks of the magnitude associated with exposure to wood dust.

Adenocarcinoma of the nasal cavities and paranasal sinuses is clearly associated with exposure to hardwood dust; in several series of cases of adenocarcinoma from different countries, a high proportion of cases had been exposed to hardwood, and these findings were confirmed in several case-control studies as well. There were too few studies of any type to evaluate cancer risks attributable to exposure to softwood alone. In the few studies in which exposure was primarily to softwood, the risk for cancer of the nasal cavities and paranasal sinuses was elevated but considerably lower than that in studies of exposure to hardwood or to mixed wood types; furthermore, in the studies of exposure to softwood, exposure to hardwood could not clearly be ruled out. It is more difficult to attribute excess risk to any particular species of wood. The concentration of wood dust and the duration of exposure may also contribute to differences in the risks of workers exposed to different types of wood. These studies consistently indicate that occupational exposure to wood dust is causally related to adenocarcinoma of the nasal cavities and paranasal sinuses.

In studies of squamous-cell carcinoma of the nasal cavities and paranasal sinuses, smaller excesses were generally reported than for adenocarcinomas, and a pooled analysis of 12 case-control studies found no association with exposure to wood dust.

A number of case-control studies on nasopharyngeal cancer have reported an association with employment in wood-related occupations; however, confounding was not ruled out from these studies, and the largest study, from Denmark, in which exposure to wood dust was estimated, did not confirm the association. Case-control studies of laryngeal cancer consistently showed an association with exposure to wood dust or woodworking; however, cohort studies of woodworkers gave consistently negative results. Overall, these studies provide suggestive but inconclusive evidence for a causal role of occupational exposure to wood dust in cancers of the nasopharynx.

Studies of the association between exposure to wood dust and cancers of the oropharynx, hypopharynx, lung, lymphatic and haematopoietic systems, stomach, colon or rectum individually gave null or low risk estimates, gave inconsistent results across studies, and did not analyse exposure-response relationships. The evidence for an association between exposure to wood dust and Hodgkin's disease was somewhat more suggestive, in that some case-control studies showed moderately high risks, but these results were not substantiated by the results of cohort studies or some of the well-designed case-control studies. In view of the overall lack of consistent findings, there is 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.

5.3 Animal carcinogenicity data

Dust from beech wood was tested for carcinogenicity by inhalation and for enhancement of carcinogenicity when administered with sidestream cigarette smoke or formaldehyde in two studies in rats, or with *N*-nitrosodiethylamine administered by subcutaneous injection in two studies in hamsters. The studies did not show any significant carcinogenic or co-carcinogenic potential of beech wood dust, but each of the studies suffered from various kinds of limitations and had some inadequacies in reporting of data.

The mutagenic fraction of a methanol extract of beech wood dust was tested for carcinogenicity by skin application in one study in mice. Although a significant, dose-dependent increase in the incidence of skin tumours and a marginally significant, dose-dependent increase in the incidence of mammary tumours were observed, these results cannot be used in an evaluation of the carcinogenicity of wood dust *per se*.

In a preliminary study, beech wood dust was tested for local carcinogenicity by intraperitoneal injection in rats; no peritoneal tumours were reported.

5.4 Other relevant data

General knowledge of particle size indicates that wood dust can be deposited in human upper and lower airways, the deposition pattern depending partly on particle size. Heavy exposure to wood dust may result in decreased mucociliary clearance and, sometimes, in mucostasis. No data were available on clearance of wood dust from the lower airways.

Exposure to wood dust may cause cellular changes in the nasal epithelium. Increased frequencies of cuboidal metaplasia and dysplasia were found in some studies of workers exposed to dust from both hardwood and softwood. These changes can potentially progress to nasal carcinoma.

Impaired respiratory function and increased prevalences of pulmonary symptoms and asthma occur in workers exposed to wood dust, especially that from western red cedar.

There is little reliable information on the effects of wood dusts on the respiratory tract of rodents. One study *in vitro* showed that various wood dusts are cytotoxic and can induce drug metabolizing enzymes.

Constituents of beech that can be extracted with polar organic solvents are genotoxic, as demonstrated by the induction of point mutations in bacteria, DNA single-strand breaks in rat hepatocytes *in vitro* and micronuclei in rodent tissues *in vivo*. Extracts of oak wood showed similar activity, but fewer data were available. Extracts of spruce, the only softwood tested, gave consistently negative results.

5.5 Evaluation¹

There is *sufficient evidence* in humans for the carcinogenicity of wood dust.

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

Overall evaluation²

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

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¹ For definitions of the italicized terms, see Preamble, pp. 23-27.

² This evaluation is based on the observation of a marked increase in the occurrence of cancers of the nasal cavities and paranasal sinuses among workers exposed predominantly to hardwood dusts.

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