WOOD DUSTS

CAS number: None

WESTERN RED CEDAR

TLV–TWA, 0.5 mg/m^3, Inhalable particulate mass

Sensitizer (SEN)

A4 — Not Classifiable as a Human Carcinogen

*All OTHER SPECIES**

TLV–TWA, 1 mg/m^3, Inhalable particulate mass

CARCINOGENICITY

A1 — Confirmed Human Carcinogen: Oak, Beech

A2 — Suspected Human Carcinogen: Birch, Mahogany, Teak, and Walnut

A4 — Not Classifiable as a Human Carcinogen: All other wood dusts

For species suspected to be allergenic, see Table 1.

Summary

This *Documentation* does not address occupational exposures to treated wood dusts.

A TLV–TWA of 0.5 mg/m^3, inhalable particulate mass, is recommended for occupational exposure to Western red cedar dusts to protect exposed workers from developing occupational asthma. This TLV–TWA is based on the extensive Western red cedar data set and from studies of sawmill workers. A sensitization (SEN) notation is appropriate, based on the evidence of both dermatologic and pulmonary sensitizer potential of this dust.

Exposure to wood dust is associated with impaired lung function and both lower and upper respiratory symptoms. These effects have been observed in association with exposure to wood dusts from a wide variety of tree species in furniture and cabinet production, lumber mills, and other settings. A number of studies have observed these effects at levels between approximately 1 and 5 mg/m^3 inhalable particulate mass; therefore, a TLV–TWA of 1 mg/m^3, inhalable particulate mass, is proposed for all species of wood dusts except Western red cedar.

The risk of sino-nasal cancer has been found to be highly elevated among workers exposed to wood dusts. Oak and beech are considered confirmed human A1 carcinogens; birch, mahogany, teak, and walnut are strongly suspected and are assigned the A2 classification. However, the mechanism by which exposure to wood dusts increases the risk of cancer is not clear, and it is possible that other tree species are also carcinogenic. Consequently, all other tree species are assigned the A4 classification, Not Classifiable as a Human Carcinogen, which includes Western red cedar. While very high levels of wood dusts are associated with a greatly increased risk of sino-nasal cancer, it is unclear whether a smaller excess risk exists at lower levels; in particular, recent studies suggest that levels below 1 mg/m^3, *i*nhalable particulate mass, may prevent the development of wood dust-associated sino-nasal cancer. These data support the recommended TLV–TWA of 1 mg/m^3, inhalable particulate mass, based on preventing decreases in pulmonary function.

All TLVs for wood dust are measured as *inhalable* particulate mass because of the evidence of an increased risk of upper and lower respiratory symptoms, and sino-nasal cancer.

Chemical and Physical Characteristics

Tree species can be botanically classified as either gymnosperms, which generally have scale-like or needle-like leaves, or angiosperms, which generally have broad leaves and are deciduous in the temperate regions of the world. For practical purposes, trees are usually classified as softwoods (temperate gymnosperms or conifers), hardwoods (temperate angiosperms), or tropical woods (which are primarily angiosperms but also include some gymnosperms). Commercially important tree species and species specifically mentioned in this review are listed in Table 1 with both their common and Latin names.

Wood is primarily composed of cellulose, hemicellulose, and lignin. In addition to these basic components, wood also contains hundreds of high- and low-molecular-weight organic compounds, collectively known as “wood extractives,” that protect

TABLE 1. Commercially Important Tree Species or Species Cited in the Review

Common Name	Latin Name	Carcinogenity Designation	Potential Allergenic Reference
SOFTWOODS			
Alaska yellow cedar	*Chamaecyparis nootkatensis*	A4	
California redwood	*Sequoia sempervirens*	A4	Chan-Yeung & Abboud, 1976;[1] doPico, 1978[2]
Douglas fir	*Pseudotsuga mensiesii*	A4	
Eastern white cedar	*Thuja occidentalis*	A4	Cartier et al., 1986;[3] Malo et al., 1994[4]
Fir	*Abies*	A4	
Amabilis fir	*Abies amabilis*		
Alpine fir	*Abies lasiocarpa*		
Balsam fir	*Abies balsamea*		
Grand fir	*Abies grandis*		
Silver fir	*Abies alba*		
Hemlock	*Tsuga*	A4	
Western hemlock	*Tsuga heterophylla*		
Mountain hemlock	*Tsuga mertensiana*		
Juniper	*Juniperus*	A4	
Larch	*Larix occidentalis*	A4	
Pine	*Pinus*	A4	Skovsted et al., 2000[5]
Spruce	*Picea*	A4	
Black spruce	*Picea mariana*		
Engleman Spruce	*Picea engrlmannii*		
Norway spruce	*Picea abies*		
Sitka spruce	*Picea sitchemsis*		
White spruce	*Picea glauca*		
Western red cedar	*Thuja plicata*	A4	Chan-Yeung et al., 1973;[6] Paggiaro & Chan-Yeung, 1987[7]
HARDWOODS			
Alder	*Alnus rubra*	A4	
Ash	*Fraxinus americana*	A4	Malo & Cartier, 1989;[8] Szmidt & Gondorowicz, 1994;[9] Fernandez-Rivas et al., 1997[10]
Aspen/Poplar/Cottonwood	*Populus*	A4	
Beech	*Fagus*	A1	Skovsted et al., 2000;[5] Hernandez et al., 1999[11]
Birch	*Betula*	A2	
Cherry	*Prunus*	A4	
Chesnut	*Castanea*	A4	
Elm	*Ulmus*	A4	
Hickory	*Carya*	A4	
Hornbeam/White beech	*Carpinus*	A4	
Lime or basswood	*Tilia*	A4	
Maple	*Acer*	A4	
Oak	*Quercus*	A1	Malo et al., 1995;[12] Sosman et al., 1969[13]
Sycamore	*Platanus*	A4	
Walnut	*Juglans*	A2	
Willow	*Salix*	A4	

continued on pages 3 & 4

TABLE 1. Commercially Important Tree Species or Species Cited in the Review (continued from page 2)

Common Name	Latin Name	Carcinogenity Designation	Potential Allergenic Reference
TROPICAL WOODS			
Abirucana	*Pouteria*	A4	Booth et al., 1976[14]
Andiroba	*Carapa spp.*	A4	
African blackwood	*Dalbergia melanoxyn*	A4	
African zebra	*Microberlinia*	A4	Bush et al., 1978[15]
Afrormosia	*Pericopsis elata*	A4	
Antiaris	*Antiaris Africana, Antiaris toxicara*	A4	Cabanes Higuero et al., 2001[16]
Australian blackwood	*Acacia melanoxyn*	A4	
Balsa	*Ochroma*	A4	
Cabreuva	*Myrocarpus fastigiatus*	A4	Innocenti et al., 1991;[17] Baur et al., 2000[18]
Cedar of Lebanon	*Cedra libani*	A4	Greenberg, 1972[19]
Central American walnut	*Juglans olanchana*	A4	Bush & Clayton, 1983[20]
Cocabolla	*Dalbergia retusa*	A4	Eaton, 1973[21]
East Indian rosewood/ Bombay blackwood	*Dalbergia latifolia*	A4	
African ebony	*Diospryos crassiflora*	A4	Maestrelli et al., 1987[22]
Fernam bouc	*Caesalpinia*	A4	Hausen & Herrmann, 1990[23]
Honduras rosewood	*Dalbergia stevensonii*	A4	
Incense machilus		A4	
Incense cedar	*Calocedrus decurrens*	A4	
Iroko or kambala	*Chlorophora excelsa*	A4	Azofra & Olaguibel, 1989;[24] Pickering et al., 1972;[25] Hernandez et al., 1999[11]
Jarrah	*Eucalyptus marginata*	A4	
Jutelong		A4	
Kauri	*Agathis australis*	A4	
Kejaat	*Pterocarpus angolensis*	A4	Ordman, 1949[26]
Kotibe	*Nesorgordonia papaverifera*	A4	Reques & Fernandez, 1988[27]
Limba	*Terminalia superba*	A4	
Mahogany (African)	*Khaya spp.*	A2	Sosman et al., 1969[13]
Makore	*Tieghemella heckelii*	A4	Ordman, 1949;[26] Obata et al., 2000[28]
Mango	*Mangifera indica*	A4	
Mansonia/Beté	*Mansonia altissima*	A4	Lo Coco et al., 1987;[29] Ordman, 1949[26]
Meranti	*Shorea sp.*	A4	
Motiné		A4	
Nara	*Pterocarpus indicus*	A4	Tochigi et al., 1983[30]
Nyatoh	*Palaquium hexandrum*	A4	
Obeche/African maple/ Samba	*Triplochiton scleroxylon*	A4	Hinojosa et al., 1984;[31] Hinojosa et al., 1986;[32] Innocenti & Angotzi, 1980;[33] Reijula et al., 1994;[34] Weber & Haussinger, 1988;[35] Quirce et al., 2000[36]
Okume	*Aucoumea klaineana*	A4	Ordman, 1949[26]
Palisander/Brazilian rosewood/Tulip wood/ Jakaranda	*Dalbergia nigra*	A4	Colas et al., 1985;[37] Godnic-Cvar & Gomzi, 1990[38]
Pau marfim	*Balfourodendron riedelianum*	A4	Basomba et al., 1991[39]
Ramin	*Gonystylus bancanus*	A4	Howie et al., 1976;[40] Hinojosa et al., 1986[32]

TABLE 1. Commercially Important Tree Species or Species Cited in the Review (continued from pages 2 & 3)

Common Name	Latin Name	Carcino-genity Designation	Potential Allergenic Reference
TROPICAL WOODS (CON'T)			
Rimu	*Dacrydium cupressinum*	A4	
Sandal wood	*Santalum album*	A4	
Sheesham	*Dalbergia sissoo*	A4	
Soapbark dust	*Quillaja saponaria*	A4	Raghuprasad et al., 1980[41]
Spindle tree wood	*Euonymus europaeus*	A4	Herold et al., 1991[42]
Tanganyike aningre	*?*	A4	Paggiaro et al., 1981[43]
Tasmanian oak	*Eucalyptus delagatensis, E. regnans, E. obliqua*	A4	
Tawa	*Beilschmedia tawa*	A4	
Teak	*Tectona grandis*	A2	

trees from attack by bacteria, fungi, and other potentially harmful agents, as well as provide grain and color to the wood. The extractives represent 5% to 30% of the wood on a mass basis.[44] Softwoods and hardwoods generally differ both in cellular structure and chemical composition. Gymnosperms usually have longer fibers, slightly less polyoses, slightly more lignin, a higher nonpolar (e.g., terpene), and lower polar (e.g., tannin) content than angiosperms, but there is considerable variability between species.[45] Examples of biologically active compounds include terpenes, lignans, and stilbenes, which are primarily found in softwoods; tannins, flavinoids, and quinones, which are primarily found in hardwoods; and phenols, which are found in both.[45–47] Various inorganic compounds have also been found in wood.

Major Sources of Occupational Exposure

Exposure to wood dust occurs among persons employed in a variety of industries. In the European Union alone, Kauppinen et al.[48] estimated that over 2.6 million workers were exposed to wood dust. In general, these industries may be separated into primary wood industries, such as logging, lumber mills, and pulp mills, where relatively fresh wood is used; secondary wood industries, where dried wood is used (e.g., furniture and cabinet manufacturing, wood pattern and model shops, and other manufacturing industries). In addition, substantial amounts of wood are used in construction. Although many of the processes performed within these industries are similar, the levels of exposure may be quite different due to the characteristics of the wood used, the degree to which engineering controls are used to limit exposure, and other factors.

The most important factor that will influence the degree to which a worker will be exposed to wood dust is the type of operation being performed. In general terms, wood dust may be generated during woodworking processes by either shattering the wood cells or by chipping out whole cells or groups of cells.[49] Shattering produces much finer particle sizes than chipping and generally creates more dust. Dust produced from chipped cells is of less concern with regard to human health because the particles are usually so large that they do not remain suspended in air and, therefore, cannot be inhaled under most circumstances. In a general sense, woodworking processes designed to create a smooth surface, such as sanding or grinding, should result in more shattering of cells than rougher woodworking processes. Another factor influencing the generation of wood dust is how the point of operation is oriented relative to the wood surface and grain. Woodworking operations performed parallel to the natural grain of the wood are less likely to shatter cells than processes performed perpendicular to the grain. As one might logically assume, the volume of wood dust generated should also increase with the velocity of the process. For example, machine sanding should generate more dust than hand sanding because a larger area can be sanded during the same period of time.

Particle Size -Selective Sampling

A wide range of mass median aerodynamic diameters (MMAD) for wood dust have been reported, but the majority were generally greater than 10 μm.[49–51] Darcy[52] found bi-modal distributions with a very large diameter mode (20–30 μm) and a small diameter mode (1–2 μm). Generally, larger particles were generated by rough cutting operations and smaller ones by sanding dry wood, although there appears to be great variability in particle sizes within operations as well.[51] Based on the size distribution of wood dust, there is potential for it to be deposited throughout the respiratory system, but the majority will deposit in the head airways (primarily in the nose with nose-breathing)

with high efficiency. Because some of the most important health effects have also been observed in the upper airways (i.e., upper respiratory symptoms and sino-nasal cancer), the TLV recommendations are made based on the inhalable particulate mass (IPM).

Although a number of studies have measured inhalable wood dust concentrations in industry,[50,53–58] most studies that have evaluated the health effects of wood dust have used so-called "total dust" sampling with closed-face 37-mm cassettes (e.g., the NIOSH Method 0500 for "Particulates Not Otherwise Regulated, Total"[59]). Most of the mass is contributed by particles larger than 10 μm, and historical data should be evaluated with great caution.

Several studies have used side-by-side sampling of wood dust to provide a rough approximation for conversion. Vincents and Laursen[58] reported a ratio of 1.6 for the Institute of Occupational Medicine (IOM) (inhalable particulate) to 37-mm closed face (total dust) sampling, based on 40 side-by-side samples collected in 32 large Danish wood factories. Vincents and colleagues[60] reported a ratio of 1.9 for IOM-to-37-mm-closed-face sampling for Norwegian wood industries. Kim and Lee[61] reported a high correlation (Pearson r = 0.91) between the 37-mm closed-face cassette and the IOM sampler for side-by-side wood dust concentrations. The concentrations measured by the IOM sampler were significantly higher; the authors offered the following formula for a conversion between the two data sets:

$$\text{Log(IOM)} = 0.745 + 0.496 \log (\text{total dust}).$$

The ratio of the IOM to the 37-mm cassette was approximately 2.5 for sanding and cutting operations. Perrault et al.[62] conducted side-by-side stationary sampling using a 37-mm closed-face cassette and an IPM sampler and observed an IPM:total dust ratio of 2.8 to 3.7. In another analysis of 66 personal side-by-side samples, a ratio of 1.2 (with a correlation coefficient of 0.90) was observed. Both studies involved the manufacturing of furniture.

Martin and Zalk[63] reported on side-by-side personal sampling conducted with the IOM samplers and the traditional 37-mm closed-face cassettes in a wood shop. While the authors observed IOM:total dust ratios of 1.8 to 4.1 for the seven samples with total dust measurements greater than 0.5 mg/m^3, ratios of 2.1 to 71 were observed for the ten samples between 0.048 and 0.45 mg/m^3 total dust. The authors proposed that large particles of wood projected into the large sample-port opening of the IOM sampler might be responsible for the wide variations at low dust concentrations. Davies and colleagues[64] also observed a ratio of IPM:total of 4.2, based on the results of 34 samples collected using the GSP inhalable and 37-mm closed cassette samplers. Much greater variations in the ratio were observed at low concentrations in this study. The authors also collected 36 side-by-side samples using the SKC seven-hole and GSP samplers and found that the seven-hole collected significantly less particulate.

Davies et al.[64] measured inhalable, thoracic, and total dust exposure in British Columbia lumber mill workers using the GSP and seven-hole (SHS) inhalable samplers, the PEM thoracic sampler, and the 37-mm closed-face cassette total dust sampler. The following were the estimated intersampler measurement ratios: GSP/37-mm sampler = 4.2; GSP/SHS = 1.7; PEM/37-mm = 1.6. The authors noted significant variability at low ambient dust concentrations. The authors postulated that the GSP sampler might be susceptible to projectile particles not normally aspirated (as well as variability in ambient wind speed and loss due to electrostatic effects), and the PEM might be unsuited to the higher concentrations of particles found in occupational settings. Tatum et al.[65] compared the performance of three different personal inhalable dust samplers and a personal *total dust* sampler in a range of facilities found in the wood products industry in the United States (sawmill, plywood mill, oriented strandboard mill, paper mill wood yard, and two furniture manufacturing plants). Reportedly, the conical inhalable dust sampler and the closed-face filter cassette *total dust* sampler may be more precise (i.e., lower coefficients of variation) than the IOM and multi-orifice (7-hole) inhalable dust samplers. As with Davies et al.,[64] the authors postulated that these two inhalable dust samplers may tend to collect projectile particles which would normally be too large to aspirate. In addition, the authors reported that the relative performance of the inhalable samplers might vary by particle-size distribution in the individual workplace when compared to the total dust sampler.

Harper and Muller[66] collected 16 side-by-side personal samples using the IOM sampler and 37-mm closed-face cassette (CFC) in three wood products industries. The IOM/CFC ratios ranged from 1.19 to 19, with a median 3.35.

Schlunssen et al.[67] compared inhalable versus total dust monitoring in a cross-sectional study in the Danish furniture industry using conventional and passive monitors. The passive dust monitor conversion models for equivalent concentrations of total dust and inhalable dust were not significantly different between the current study and the original models based on earlier data collections. They also found that inhalable dust exposure was about 50% higher than total dust.

The studies cited above found IPM:total dust ratios ranging from 1.2 to 4.2 for total dust concentrations in the range useful for setting a TLV for wood dust. Werner and colleagues[68] suggested 2.5 as a working inhalable-to-total-dust conversion factor, based on the results of studies conducted in different industries with different exposures. This would seem to be a reasonable approximation for

wood. Therefore, a ratio of 2.5 will be used in this document for interpreting studies with exposure measurements based on total dust sampling. However, the reader should recognize that the variability may be higher when the total dust concentrations are less than 0.5 mg/m^3 and lower when the mass is primarily due to relatively small particles (i.e., < 20 μm) as noted by Tatum et al.[65] and Davies et al.[64]

Other Sampling Issues

Recent work in wood dust exposure has focused on the measurement of endotoxin and 1→3-β-D-glucan as possible significant occupational exposures, particularly in the softwood sawmill industry. Monoterpene exposures have also been evaluated, and a separate *TLV® Documentation* is available for Turpentine and Selected Monoterpenes[69] (α-pinene, β-pinene, Δ^3-carene). Douwes et al.[70] evaluated worker exposure to airborne dust, endotoxin, and 1→3-β-D-glucan in two New Zealand sawmills. The authors reported that the measurement of dust exposure is a poor proxy for 1→3-β-D-glucan and endotoxin exposures in these workers. However, these authors did not measure inhalable wood dust, but rather total dust. Alwis et al.[71] noted that high exposures to endotoxins were found in the inhalable fraction (correlation coefficient 0.58; $p<0.05$) compared to the respirable fraction (correlation coefficient 0.41; NS). Dennekamp et al.[72] found a significant correlation between inhalable dust and inhalable endotoxin levels (correlation coefficient 0.69; $p<0.0001$) among softwood lumber mill workers in British Columbia.

Of note, the significant determinants of personal wood dust exposure were found to be local exhaust ventilation ($p<0.001$), job title ($p<0.001$), use of hand-held tools ($p<0.001$), cleaning method ($p<0.011$), use of compressed air ($p<0.045$), and green or dry wood processed ($p<0.001$), with a R^2 (ANCOVA) overall of 0.68. In the same model, the type of wood processed (soft or hardwood) was not found to be significantly related to personal wood dust exposure.[73] Similar results were found by Dennekamp et al.[72]

Demers et al.[74] sampled inhalable particles, monoterpenes (α- and β-pinenes and Δ^3-carene), and resin acids (abietic and primaric acids) in softwood lumber mills of British Columbia. The monoterpene exposures were much lower than those observed in similar studies in Sweden and Finland. The authors stated that these results highlight the importance of considering the content of airborne particulates in lumber mills as well as potential exposure to wood chemicals.

Human Health Effects Studies

The reviewed studies were presented to establish the methodology of exposure measurement (i.e., total dust versus inhalable) and the association with possible human health effects. In addition, a distinction is made between those studies performed among sawmill workers with possible confounding exposures discussed above, compared with other wood dust exposed workers. Finally, a distinction is also made between known allergenic wood dusts (such as Western red cedar) and other wood dust exposures.

Dermatitis

Wood dusts can cause allergic contact dermatitis as a result of Type 1 and Type IV hypersensitivity, as well as irritant dermatitis. The sensitizers in the hardwoods were reportedly the benzo- and naphthaquinones. Although relatively rare, the majority of reported cases of allergic contact dermatitis were occupationally related; these workers often reported respiratory and mucosal symptoms (e.g., conjunctivitis, rhinitis, and asthma), in addition to dermatitis[75–78] (Table 1).

Respiratory Disease

Dozens of studies have examined the risk of respiratory disease among workers exposed to wood dust. This review will be restricted to studies where wood dust was the predominant exposure and where there were no or very low exposure to exogenous chemicals such as formaldehyde, isocyanates, and other manufactured chemicals with known respiratory effects. In addition, only studies where levels of wood dust exposure were reported will be described in detail. For a discussion of other studies, a number of reviews have been published.[46,47,76,79–83] The studies will be presented in chronological order. Studies that deal only with allergenic species (such as Western red cedar) or with allergenic responses will be reviewed in the next section.

Anderson and colleagues[84] compared the respiratory health of 68 Danish furniture workers to 66 unexposed controls. The furniture workers were primarily exposed to teak, oak, chipboard, and palisander, with additional exposure to some mahogany, jakaranda, beech, ramin, motiné, Masonite, and pine. The level of exposure was assessed, based on 68 personal total dust samples. Mucociliary clearance was significantly slower in the exposed group. The occurrence of mucostasis in exposed subjects was related to the concentration of wood dust, with mucostasis in 11% of subjects exposed at 2.2 mg/m^3 and in 63% exposed at 25.5 mg/m^3. An increased prevalence of sinusitis, prolonged colds, asthma, sneezing, and nasal obstruction was also reported among the exposed group, but no differences in lung function were observed.

Whitehead and colleagues[85,86] performed a cross-sectional survey of workers from ten woodworking companies in Vermont to determine the relationship between wood dust exposure and

changes in pulmonary function. A total of 1157 woodworkers participated (74% of eligible); workers with exposures other than to wood dust were excluded from the analyses. Rock maple was the predominant hardwood, with smaller amounts of ash and oak; the softwood was white pine. Approximately 100 stationary dust samples were collected; average levels in different departments ranged from 0.2 to 4.5 mg/m^3 total dust. Workers were divided into three groups, based on cumulative exposure: low (< 2 mg/m^3-years), medium (2–9.9 mg/m^3-years), and high (10+ mg/m^3-years). Low pulmonary function was defined as below the lowest 5th percentile of the normal comparison population. Those in the medium or high hardwood dust exposure categories were 2 to 3 times more likely to have a low ratio of forced expiratory volume in 1 second (FEV_1): forced vital capacity (FVC) and low maximal mid-expiratory flow rate (MMF) compared to workers in the low exposure categories. Low pulmonary flow rates were also 2 to 4 times more likely to occur in those exposed to high levels of pine dust. In a later article using the same data, Whitehead[87] determined average cumulative dust exposure for workers in the high-exposure class was 27.4 mg-yrs/m^3. Exposures above this level were associated with an excess prevalence of decreased pulmonary function. Based on the assumption that personal samples would on average be 3 to 4 times higher than area samples and given a 40-year maximum work lifetime, Whitehead[87] recommended that the average personal exposures should not exceed 2 mg/m^3 total dust.

Al Zuhair and colleagues[53] compared the lung function of 113 workers employed at two English furniture factories to 47 power station workers. The furniture workers were primarily exposed to limba, beech, and ash and to some mahogany, oak, and ramin. Lung function was measured pre- and postshift on Monday and Thursday, and 193 inhalable personal dust samples were collected. Small (approximately 100 ml), but statistically significant ($p<0.001$) cross-shift drops in FEV_1 and FVC were observed among workers from the dustier factory (mean = 5.7 mg/m^3), but not among workers from the less dusty factory (mean = 3.3 mg/m^3) or the controls. No dose–response was observed within the factories.

Wilhelmsson and Drettner[88] conducted a cross-sectional survey of 676 workers (93% participation) from 50 Swedish furniture factories. The prevalence of nasal hypersecretion, nasal obstruction, and colds was significantly higher ($p<0.05$) among workers whose self-reported exposure was heavy or moderate (n=484) compared to those with light or no exposure (n=192). Further examinations were performed on 61 workers exposed to fine wood dust from turning, machining, or sanding. Fifty-four percent of these workers had decreased mucociliary clearance (> 20 minutes). Fifty-nine workers completed pulmonary function testing and had a mean FVC that was approximately 550 ml lower than expected ($p<0.001$). The workers were primarily exposed to dust from birch, beech, oak, mahogany, and teak; the mean wood dust exposure was 2 mg/m^3 total dust (range, 0.30–5.06), based on 28 personal samples from the factories that employed the workers who underwent examinations.

Holness and colleagues[89] compared nasal cytology, lung function, and the prevalence of symptoms among 50 workers employed in four Canadian cabinetmaking shops to 49 hospital workers. The cabinet workers were exposed to dust from a variety of woods including birch, oak, cherry, walnut, cedar, poplar, fir, pine, spruce, chipboard, particleboard, and plywood. Mean levels of exposure were reported as 1.83 mg/m^3 total dust, 0.29 mg/m^3 respirable dust, and 0.06 ppm formaldehyde.[90] The prevalence of eye irritation and rhinitis was significantly higher among wood workers. The cabinetmakers also tended to have more irritated columnar cells and fewer ciliated cells in the nasal tract than the control group. A significant cross-shift drop in FEV_1 and FVC was observed among exposed workers, while no change was observed among controls. Thirty-one percent of exposed workers had a 5% or greater drop in FEV_1 or FVC compared to 13% of controls. The cross-shift drops did not appear to be related to the level of exposure. However, a significant relationship was found between cumulative exposure to both total and respirable dust and to both FEV_1 and FEV_{75} as measured at baseline ($p<0.01$). The relationship with decreased FVC was marginally significant ($p<0.10$). Based on the data provided, a 10% drop in FEV_1 would be predicted, based on 58.8 mg/m^3-years of exposure.

Goldsmith and Shy[91] conducted a cross-sectional study of the respiratory health of North Carolina furniture workers with internal comparison. The study population consisted of 94 furniture workers, of which 55 were exposed to oak, maple, walnut, mahogany, andiroba, poplar, and fiberboard dust. With the exception of the carving area, which had a concentration of 5 mg/m^3, mean exposure was described as approximately 2 mg/m^3 total dust or less. After adjusting for age, sex, and smoking, wood dust exposed workers had a significantly increased risk of work-related sneezing (odds ratio [OR], 4.1) and work-related eye irritation (OR, 4.0). Lung function was measured pre- and postshift. The preshift results were examined in relationship to months of wood dust exposure, and only peak flow rate was significantly associated. In the cross-shift analysis, employment in wood dust-exposed jobs had a borderline association with decreased FEV_1 and forced expiratory flow at 50% of volume ($FEF_{50\%}$).

Pisaniello and colleagues[56] performed a survey of dust exposure and respiratory symptoms among 168 men employed in 15 Australian furniture factories with no exposure to solvents. The prevalence of respiratory symptoms was compared to 46 hospital

workers. For the analysis, workers were divided into two groups, either predominantly exposed to hardwoods (Tasmanian oak, teak, nyatoh) or reconstituted softwood boards. The mean levels of personal inhalable dust exposure were 3.8 mg/m^3 for hardwood workers and 3.3 mg/m^3 for softwood workers. Formaldehyde was also measured for some reconstituted softwood workers with a mean concentration of 0.06 ppm. A significantly increased prevalence of nasal obstruction, runny nose, sneezing, and multiple nasal symptoms was observed among all furniture workers combined and hardwood workers, but only runny nose was significantly elevated among the softwood board workers. When dust was classified as none (controls), low (< 2 mg/m^3), medium (2–5 mg/m^3), and high (> 5 mg/m^3), evidence for a dose–response was observed for runny nose, sneezing, and multiple nasal symptoms among all furniture workers combined (statistical tests were not performed). No association with lower respiratory symptoms was observed.

Norrish and colleagues[55] performed a cross-sectional study of 41 New Zealand furniture workers compared to 38 office workers. The furniture workers were exposed to a rimu, kauri, tawa, fiberboard, and California redwood. Inhalable wood dust levels ranged from 1.0 to 25.4 mg/m^3 (median 3.6 mg/m^3), with 32% of samples exceeding 5 mg/m^3. The median formaldehyde exposure was 0.06 mg/m^3 (range 0.01 to 0.27 mg/m^3) based on area sampling. Significantly higher prevalence rates for nasal obstruction, nasal discharge, sneezing, persistent cough, and breathlessness were reported among the furniture workers compared to the office workers. Upper respiratory and eye symptoms were reported more commonly in association with exposure to dust from rimu.

?hmans and colleagues[92] performed a cross-sectional survey of respiratory symptoms and exposures among 130 Swedish woodwork teachers (94% participation), primarily exposed to Scandinavian wood (pine, birch, juniper, alder, lime) and reconstituted wood (plywood and chipboard), with occasional exposures to exotic woods (teak, mahogany, jutelong). Their results were compared to 103 unexposed teachers and 9 other school workers who were similar in regards to sex, age, height, and smoking. Shops were classified as good or poor, based on work environment factors (general and local exhaust ventilation, housekeeping) and type of woodworking machines present. The woodwork teachers had a significantly higher prevalence of chronic bronchitis, dry cough, dyspnea, phlegm, nasal obstruction, nasal irritation, eye irritation, and many other symptoms, which persisted after adjustment for smoking, sex, and atopy. The prevalence of bronchial irritation, chronic bronchitis, dry cough, dyspnea, nasal irritation, sneezing, burning throat, and throat irritation were higher among teachers working in poor shops than in good shops. A significant association between bronchial irritation, nasal obstruction, and throat irritation and exposure to Scandinavian woods was observed while only sneezing was associated with exposure to exotic woods. Although the primary exposure was to wood dust, most of the teachers also taught other industrial arts classes to a lesser extent and may have also been exposed to solvents and other potential respiratory irritants.

?hmans and colleagues[93] further studied 39 woodwork teachers employed full time for at least 3 years and compared their results to 32 unexposed school workers. The mean levels of wood dust exposure were 0.57 mg/m^3 total dust (range, 0.18–1.12) and 0.10 mg/m^3 respirable dust (range, 0.02–0.21), based on personal sampling. The mean terpene concentration was 0.68 mg/m^3, based on area sampling. Participants were examined on Monday morning and Thursday afternoon, and the prevalence of nasal obstruction, itchy nose, and nasal irritation increased significantly over the week among the exposed, but not the controls. Mucociliary clearance slowed significantly ($p<0.001$) over the week among the exposed, but not the controls. The level of wood dust exposure was correlated with nasal obstruction ($p<0.05$), runny nose ($p<0.01$), and itchy nose ($p<0.01$), while no significant association was observed with terpene exposure.

Liou and colleagues[94] examined pulmonary function and the prevalence of respiratory symptoms among 82 workers from 12 Taiwanese wood milling operations and compared them to 262 office workers. The milling workers were exposed to a mix of incense machilus, sandalwood, Taiwan incense cedar, and various hardwood species. Seven stationary total dust samples were collected using a six-stage cascade impactor; the respirable fraction ranged from 2.4% to 50.2%. The six samples collected for grinding and screening had a mean of 12 mg/m^3 (range, 4.4–22.4 mg/m^3), while the single sample collected for a sawyer had a concentration of 2.9 mg/m^3. Exposure was classified as none (control group), low (sawyers), and high (grinding and screening) in order to examine dose–response relationships. For nonsmokers, the prevalence of chronic phlegm and chronic bronchitis among high exposure workers was significantly higher than in the controls. FEV_1, MMF, peak expiratory flow rate (PEFR), and $FEF_{25\%}$ were found to be significantly lower in the exposed workers than in the controls, and significant trends by level of exposure were observed for FVC, FEV_1, MMF, PEFR, $FEF_{25\%}$, and $FEF_{50\%}$ among both smokers and nonsmokers. After adjusting for age, sex, height, and smoking status, all parameters of pulmonary function were significantly lower in the exposed population than in the controls and a significant association with level of exposure was also observed.

Several population-based studies have also observed an excess risk of respiratory disease among wood workers. An excess incidence of chronic lung disease, defined as either doctor-

diagnosed chronic bronchitis or emphysema or episodes of respiratory symptoms lasting 3 or more months, was observed among 868 Dutch men followed for 25 years who reported employment as wood and paper workers (relative risk [RR], 1.72; 95% confidence interval [CI], 1.10–3.62, after adjustment for age, time period, and smoking).[95] Exposure to wood dust in the same population was assessed using a job exposure matrix.[96] After adjustment for age and smoking, smaller excesses of both chronic lung disease incidence (RR, 1.31; 95% CI, 0.85–2.02) and mortality (RR, 1.37; 95% CI, 0.97–2.93) were observed. An excess of respiratory disease was also observed in a Norwegian study of 1512 people.[97] After adjustment for age, sex, and smoking, wood dust exposure was associated with a nonsignificant excess of both obstructive lung disease, defined as physician-diagnosed asthma or emphysema (OR, 1.8; 95% CI, 0.8–3.5), and spirometric airflow limitation, defined as FEV_1 < 80% or FEV_1/FVC < 70% of predicted (OR, 1.5; 95% CI, 0.7–4.6). A significant excess of obstructive lung disease mortality was observed (RR, 1.45; 95% CI, 1.09–1.92), after adjustment for age and smoking, among 11,541 men who were employed in wood-related occupations in a follow-up of Cancer Prevention II survey participants.[98] However, the relative risk among those men who reported regular exposure to wood dust was no higher than among those who did not.

Saw Mill Workers and Respiratory Disease

WOOD DUST EXPOSURE

As part of a study of a large Canadian pulp and paper mill, Chan-Yeung and colleagues[99] compared the lung function of 319 sawmill workers exposed to Douglas fir, Western hemlock, fir, and spruce to 496 unexposed log pond and office workers. Seventy-one personal total dust samples were collected (mean, 0.5; range, < 0.1–2.7 mg/m^3). No significant differences in the prevalence of symptoms were reported. However, wood dust-exposed workers had slightly lower FEV_1 and FVC than unexposed workers. This difference was statistically significant after adjustment for smoking and other potential confounders.

Halpin and colleagues[54] compared respiratory symptoms and lung function of 103 Welsh sawmill workers, who were exposed to spruce, Douglas fir, and pine, to 58 workers from a nearby metal products factory. The mean exposure to inhalable particles in the "low" exposed areas was 0.7 mg/m^3, while the mean exposure in the "high" exposed areas was 3.0 mg/m^3. Controls were also exposed to dust (mean 2.5 mg/m^3), but not to wood dust. Workers in the high dust areas were over twice as likely to report work-related breathlessness and nasal, eye, and flu-like symptoms compared to workers in low dust areas and controls. Chronic bronchitis and symptomatic bronchial reactivity were twice as prevalent among both low and high wood dust-exposed workers compared to controls. No significant differences in FEV_1 or FVC were noted among the groups. Exposure to molds was noted in many areas of the mill, with visible mold more commonly reported in the low dust areas. Symptoms consistent with extrinsic allergic alveolitis were observed in two workers (4.4%) in the high exposure group and in none of the low exposure group. Of note, Baur et al.[18] and Halpin et al.[100] reported individual cases of extrinsic allergic alveolitis in wood workers (a parquet floor layer exposed to cabreuva (*Myrocarpus fastigiatus*) and a saw mill worker exposed predominantly to spruce and Douglas fir, respectively) with IgG binding against wood dust and multiple fungi. Halpin et al.[100] reviewed the literature with respect to reported cases of extrinsic allergic alveolitis and wood dust exposure. Although fungi serve a clear etiologic role in this disease, Halpin and colleagues opined that wood dust itself might be an important etiology.

Hessel and colleagues[101] compared respiratory symptoms and lung function of 94 Alberta sawmill workers, who were exposed primarily to pine and spruce, to a control population of 165 oil field workers. Workplace exposure levels were measured as particles less than 10 μm (PM10) and levels were between 0.1 and 2.2 mg/m^3, with a mean of 1.4 mg/m^3. The sawmill workers had significantly lower average values for FEV_1 and FEV_1/FVC after adjusting for age, height, and smoking. Sawmill workers were 2.5 times as likely as the oil field workers to report asthma (CI, 0.76–8.32) and had a higher prevalence of shortness of breath (OR, 2.8; 95% CI, 1.5–5.5) and wheeze with chest tightness (OR, 2.6; 95% CI, 1.2–5.6). The risk of asthma and bronchitis increased with duration of employment.

Wood Dust, Endotoxin, and Other Exposures

Mandryk and colleagues[102] conducted a cross-sectional study of respiratory symptoms, lung function, and exposure to wood dust and related biohazards among workers from four sawmills, one chipping mill, and five joineries in Australia. The sawmills and chipping mill all processed eucalyptus, a hardwood, while the joineries processed a mixture of hard and softwoods, including radiata pine, Western red cedar (one joinery), meranti, oak, and jarrah. The mean exposures in the sawmills were 4.8 mg/m^3 inhalable dust, 0.4 mg/m^3 *respirable* dust, 13.0 ng/m^3 endotoxin, and 3.3 ng/m^3 1→3-β-D-glucan. The mean exposures in the chipping mill were 3.2 mg/m^3 inhalable dust, 0.3 mg/m^3 respirable dust, 3.5 ng/m^3 endotoxin, and 4.6 ng/m^3 1→3-β-D-glucan. The mean exposures in the joineries were 7.6 mg/m^3 inhalable dust, 0.7 mg/m^3 respirable dust, 4.7 ng/m^3 endotoxin, and 0.6 ng/m^3 1→3-β-D-glucan. Workers from sawmills and chipping mill (n=105) and joinery workers (n=63) were grouped for analysis. Both groups had significantly lower FVC

and FEV_1; increased prevalence of regular cough and phlegm; and greater cross-shift drops in FVC, FEV_1, FEV_1/FVC, $FEF_{25–75}$, and PEFR than maintenance workers (n=30) from the same sites. In dose–response analyses, decreased lung function was most consistently associated with duration of exposure and inhalable dust levels, but it was also associated with respirable dust and endotoxin levels.

The same researchers[71] reported on exposure to the same biohazards in wood dust among the above listed worker groups in addition to two logging sites as a cross-sectional study with maintenance staff controls. There were highly significant associations between mean personal inhalable endotoxin exposures and gram negative bacterial levels ($p<0.0001$) and between mean personal inhalable 1→3-β-D-glucan and fungi levels (0.0003). The prevalence of reported cough, phlegm, chronic bronchitis, nasal symptoms, frequent headaches, and eye and throat irritation was significantly higher among wood exposed workers (195) compared with controls (34). A dose–response relationship was found between personal exposures and work-related symptoms (adjusted for age and smoking) among all the wood workers except the loggers. Of interest, there were high reports of chest tightness (37% joinery workers, 21% saw and chip mill workers), and among those reporting chest tightness, 33% joinery and 52% chip and saw mill workers reported symptoms recurred on their first day back from the weekend or vacation. The authors[71] postulated that some of the symptoms and illness reported in wood exposed workers were due to exposure to these biohazards, suggestive of organic dust disorders reported among workers exposed to swine confinement, poultry, and contaminated humidifiers.

Further investigation by Mandryk et al.[103] compared the exposure levels and effects of personal exposure on cross-shift lung function and work-related symptoms among two groups of Australian hardwood sawmill workers from three green mills and two dry mills compared to controls. Measurement of wood dust biohazards (e.g., endotoxins, 1→3-β-D-glucan, gram-negative bacteria, and fungi), lung function, and reported symptoms were made. The geometric mean of the inhalable dust was 1.53 mg/m^3 for the green mills and 1.71 mg/m^3 for the dry mills; however, there was wide variation, with 70% and 50% of the inhalable dust exposures greater than the Australian occupational exposure limit of 1 mg/m^3 TWA for the green and dry mills respectively. The endotoxin and glucan exposures were consistently higher for the green mills; there were significant and high correlations between all of the biohazards measured. The prevalence of symptoms reported, adjusted for age and smoking, were significantly higher among the green mill workers than the dry mill workers or controls, particularly cough, phlegm, chronic bronchitis, eye and throat irritation, and flu-like symptoms; dry mill workers reported significantly more nasal discharge and headache. With regards to lung function, green mill workers were slightly lower than dry mill, while all wood dust exposed workers compared to controls had significantly lower mean percentage predicted lung function. Although the majority of the endotoxin and glucan levels were below 10 ng/m^3, they were significantly correlated with work related symptoms and lung function. The green mill workers had a high and statistically significant correlation between inhalable dust exposure and cross-shift decrement in $FEF_{25–75}$ ($r=0.95$; $p<0.0001$); significant associations with lung function were also noted for respirable as well as inhalable wood dust levels.

Asthma

Asthma has been attributed to the dust from many different tree species, based on case reports or epidemiologic studies, as discussed in several reviews.[76,77,79,80] Some North American softwoods and hardwoods, as well as many exotic species, have been identified as allergenic (see Table 1). The proportion of all occupational asthma due to wood dust has been reported to be 6% in the United Kingdom[104] and 11% in Quebec, Canada.[105] The proportion of occupational asthma cases may be higher in regions where allergenic tree species are common, such as in British Columbia, Canada where 47% of cases have been reported to be associated with wood dust, primarily Western red cedar.[106] While many case reports and epidemiologic studies on asthma associated with wood dust have been published, only Western red cedar has been studied extensively and has significant dose–response information available.

Borm et al.[107] performed a cross-sectional study in 1997 during the shift in an Indonesian woodworking plant using mostly meranti work of 982 workers. Personal sampling divided the workers into three exposure groups: < 2.0 mg/m^3; 2–5 mg/m^3; and > 5 mg/m^3 inhalable. A questionnaire, lung function (flow volume and forced oscillation) tests, and nasal lavage were used to assess upper respiratory tract inflammation. Some reported symptoms (e.g., wheezing) showed a positive dose–response, particularly among the women workers. Male workers showed a significant association between increasing years of exposure and decreasing lung function; women workers, less exposed historically and currently, did not show this association. No consistent differences in nasal inflammation were found for the workers relative to exposure level, pulmonary function, or reported symptoms.

Bohadana et al.[108] evaluated 114 male woodworkers (predominantly oak and beech) from five furniture factories in France compared to 13 unexposed male controls and 200 historic controls (from the early 1990s) in a cross-sectional study. Personal dust sampling with calculation of cumula-

tive dust exposure, as well as methacholine challenge was performed. Geometric mean total dust exposures depended on job and local exhaust ventilation: 3.5 ± 2.47 mg/m^3 nonsanding and 7.86 ± 2.01 mg/m^3 sanding with ventilation versus 2.43 ± 2.80 mg/m^3 nonsanding and 4.48 ± 3.47 mg/m^3 sanding without ventilation. The median cumulative exposure to dust was 110 years × mg/m^3 (range, 70–160 years × mg/m^3). Increased exposure was not associated with decrease in FEV_1 or FVC. However, increasing cumulative exposure was significantly associated with reported sore throat and with positive methacholine challenge test. The authors[108] concluded that workers exposed to oak and beech were at increased risk for sore throat and bronchial hyperresponsiveness. Longitudinal studies on the same worker population are ongoing.

Schlunssen et al.[77] conducted a cross-sectional study of 2303 woodworkers from 54 furniture factories and 576 controls from nonwood-exposed factories using questionnaire and lung function (including a sub-sample of 1508 persons performing pre/post shift) with personal dust monitoring; acoustic rhinometry, skin-prick testing, bronchial responsiveness, and 2 weeks of peak flow monitoring were performed on a subpopulation. The geometric mean exposures were 0.93 mg/m^3 inhalable (geometric standard deviation [GSD], 2.10)(0.60 mg/m^3 total dust [GSD, 1.96]). No association was found between dust concentrations and lung function. Workers with >1 mg/m^3 exposure to wood dust had an increased frequency of morning and daily cough and throat symptoms compared to controls. Workers with >1.42 mg/m^3 exposure had an increased prevalence of daily cough and chest tightness and wheeze at night relative to low exposed workers (<0.74 mg/m^3). Pine-exposed workers were more likely to be associated with reported wheezing and decrease in cross-shift FEV_1, while beech-exposed workers reported an increased frequency of throat symptoms. As part of the larger study, Schlunnsen and colleagues[83] performed nasal rhinometry on 161 wood workers and 19 controls. Exposure to inhalable dust among the furniture workers ranged from 0.17 to 3.44 mg/m^3 (mean, 1.17). Increased mucosal swelling was observed after 4 and 7 hours of work and was correlated with wood dust exposure levels. Self-rated nasal obstruction was not correlated with obstruction as measured with nasal rhinometry but was increased postwork shift for exposed workers.

Many other tree species, including hardwoods (ash and oak), softwoods (California redwood, white cedar), and many tropical woods, have been identified as causing asthma (Table 1). These identifications were based on epidemiologic studies and case reports of occupational asthma where wood dust was identified as the causative agent, based further on evidence that wood dust exposure preceded the onset of asthma and one or more of the following:

1. A laboratory or workplace challenge using dust from that tree species
2. An immediate wheal and flare reaction on skin testing using aqueous extracts from the tree species
3. The presence of specific IgE antibodies demonstrated using the RAST (radioallergosorbent) test or precipitating antibodies to the tree species detected in the sera of affected patients.

Sawmill Workers and Asthma

WOOD DUST EXPOSURE

Chan-Yeung and colleagues[6] examined 22 woodworkers with respiratory symptoms and previous exposure to Western red cedar dust. Eighteen of the 22 subjects reacted to inhaled provocation with Western red cedar extract. Responses included immediate, delayed, and dual asthmatic responses. Ashley et al.[109] and Chan-Yeung and colleagues[110] compared cedar sawmill workers to other sawmill workers exposed to noncedar wood dust and found increased cough, phlegm, wheeze, breathlessness, rhinitis, and conjunctivitis in the cedar mill workers, but no differences in pulmonary function were found between the two groups of sawmill workers. Unfortunately, no unexposed controls were included in this study.

Brooks and colleagues[111] compared the questionnaire and spirometry results from 74 Western red cedar shake mill workers to 22 control subjects. Lung function was measured pre- and postshift for 3 consecutive days. Mean wood dust levels were 4.7 mg/m^3 total dust. Occupational asthma, defined as a 10% or greater drop in FEV_1 from Monday preshift to any subsequent test during the next 3 work shifts and a positive clinical history, was observed in 14% of the cedar workers and in none of the control subjects. Chronic bronchitis, defined as chronic cough or phlegm on most days, was observed in 34% of the cedar workers and in 16% of the controls. The prevalence of asthma was related to level of exposure, ranging from no asthma among splitters (0.5 mg/m^3), 5% prevalence among splitters (3.6 mg/m^3), 11% prevalence among packers (4.8 mg/m^3), to 24% prevalence among sawyers (6.8 mg/m^3).

Vedal and colleagues[112] studied 93% of all employees in one cedar sawmill (n=652 tested) and collected 104 *total dust* measurements which were used to assign estimates of personal wood dust exposure for 334 workers, based on job title. Estimated wood dust levels were 0.45 mg/m^3, and 301 were exposed, on average, to less than 1 mg/m^3, 20 were exposed to between 1 and 2 mg/m^3, and 13 were exposed at levels over 2 mg/m^3 (with a maximum value of 6 mg/m^3). Occupational asthma was defined as the presence of four or five symptom complexes consistent with asthma with a temporal relation to work, and no asthma prior to employment

at the sawmill. The prevalence of occupational asthma was 6% in the low, 5% in the medium, and 15% in the high exposure groups. Vedal et al.[113] also studied bronchial hyper-responsiveness in sawmill workers where Western red cedar was primarily cut. Eighteen percent of the workers showed bronchial hyper-responsiveness. In general, those with bronchial hyper-responsiveness also had lower levels of pulmonary function than those without bronchial hyper-responsiveness.

Chan-Yeung and Desjardins[114] evaluated four workers from the same sawmill reported by Vedal who developed occupational asthma after the initial survey. Three of these four developed asthma symptoms after being transferred from low exposure (outside) jobs to high exposure (inside) jobs. The mean total dust exposure levels for outside jobs ranged from 0.01 to 0.27 mg/m^3 (with no samples measuring over 2.5 mg/m^3). For inside jobs, the mean total dust exposure levels ranged from 0.18 to 0.57 mg/m^3 (with up to 4% of samples over 2.5 mg/m^3).

Noertjojo and colleagues[115] conducted a longitudinal study of 243 Western red cedar sawmill workers compared to 140 office workers. None of the participants had been previously diagnosed as asthmatic by a physician, but they had participated in at least two cross-sectional surveys conducted by Chan-Yeung et al. and Vedal et al. Exposure was estimated, based on 916 personal total dust samples. Lifetime average exposures (geometric mean) were classified as low (0.13 mg/m^3), medium (0.30 mg/m^3), and high (0.61 mg/m^3). After adjustment for age, height, race, and smoking, high-exposed sawmill workers experienced a decline in FVC of 21 ml/year ($p<0.05$), while the medium exposed group experienced a decline of 16 ml/year ($p<0.05$), and the low -exposed group experienced a decline of 11 ml/year relative to control subjects. Although the sawmill workers experienced annual declines in FEV_1 compared to the control subjects, a consistent dose-response pattern was not observed.

Wood Dust, Endotoxin, and Other Exposures

Cormier et al.[116] performed a cross-sectional study in 17 sawmills (predominantly pine) in Eastern Canada, measuring respirable dust, bacteria, endotoxins, and molds. A total of 1205 sawmill workers participated in the respiratory health questionnaire, lung function testing, skin-prick tests, and venous blood for specific serum IgG against molds. Nonworker controls (4 cases: 1 control) underwent questionnaire and serum testing. Respirable dust levels ranged from 0.039 to 5.147 mg/m^3. When adjusted for age, height, and weight, all workers had reportedly normal lung function with no effect of length of employment; respiratory symptoms were reportedly associated predominantly with smoking, and the prevalence of asthma was reportedly similar to that of the general Canadian population (7%). Workers in pine sawmills had a greater prevalence of positive skin-prick tests to pine; however, this was not associated with abnormal lung function. Significantly more workers had highly positive antibody scores to molds ($p=0.001$) compared to controls, although this response was not associated with abnormal lung function. The authors acknowledged the possibility of the healthy worker effect and indicated that a possible increased risk for asthma and other allergic lung diseases existed among these wood-exposed workers, based on the antigen results.

Cancer

Cancer-associated exposure to wood dust has been extensively reviewed in the International Agency for Research on Cancer (IARC) Monograph on wood dust,[45] as well as by other authors.[117–121] This review will be restricted to key epidemiologic studies and those with results for sino-nasal cancer that report levels of wood dust exposure or estimate dose–response relationships and to studies that identify tree species. While some studies have observed an association between wood dust and cancers other than sino-nasal (e.g., lip, lung, nasopharyngeal, pleural, gastric, colorectal, and cervical cancers, Hodgkin's disease and multiple myeloma, and even possibly neuroblastoma in the offspring), the results have been less consistent, the relative risks observed have not been as high, and there have been confounding issues such as tobacco use and socioeconomic class.[45,118,121–124]

An excess of sino-nasal cancer among woodworkers was first recognized in the 1960s in England.[125,126][Workers in the furniture manufacturing and cabinetmaking industries were found to have a 10- to 20-fold increased risk of nasal cancer[127] and a 100- to 500-fold increased risk of nasal adenocarcinoma.[128,129] Subsequently, many case–control studies conducted in different countries have confirmed the initial findings, with extremely high relative risks being observed in European studies. For example, a Dutch study[130] observed a 140-fold excess risk for furniture and cabinet makers, a French study[131] observed an 35-fold excess among cabinet makers, and an Italian study[132] observed a 90-fold excess among woodworkers and cabinetmakers. However, the only U.S. study to look at sino-nasal adenocarcinoma observed a more modest but significant risk among furniture workers (OR, 5.7).[132]Smaller, but consistently elevated risks were still observed when the risk among workers employed in all wood-related jobs were considered together or when all sino-nasal cancers (regardless of histology) were considered together. Although the highest risks have been observed among workers in the wood furniture industry, excesses have also been observed in other wood-related industries, such as sawmills, cabinet-making, and carpentry.[122,125,134]

The pooled data from 12 studies conducted in 7

different countries were used to examine the relationship between wood dust and sino-nasal cancer in detail.[134] The combined data set included 680 male cases, 2349 male controls, 250 female cases, and 787 female controls. An elevated risk of adenocarcinoma among the men that was associated with employment in wood-related occupations (OR, 13.5; 95% CI, 9.0–20.0). Dose–response was examined using a job-exposure matrix developed for the study. There was no excess among the men in the lowest exposure category, a smaller but significant excess was observed in the moderate exposure category (OR, 3.1), and a large excess was observed among men in the highest exposure category (OR, 46) after adjustment for study, sex, and age. Further adjustment for smoking did not alter the relationship. Very few women had been employed in wood related occupations. Little evidence of a relationship with squamous cell carcinomas was observed; an increased risk of squamous cell carcinoma was found only among those who worked for 30 or more years in occupations with fresh wood dust exposure. Workers were exposed to a mixture of both hardwoods and softwoods.

Several other case–control studies have also observed a dose–response relationship using semi-quantitative estimates of wood dust exposure based on job title and industry.[130,135] Because of the long latency of sino-nasal cancer, it is largely assumed that the effective period of exposure for most studies was 20 to 30 years prior to diagnosis, which is equivalent to the 1950s and 1960s for most studies. Unfortunately, there are very few exposure measurements from that period.

No studies have thus far examined the risk of sino-nasal cancer in relationship to quantitative estimates of wood dust exposure. Analyses of both cancer mortality[136] and incidence[129] among a cohort of 5108 High-Wycombe furniture workers found an association between "dustiness" and the risk of sino-nasal cancer. No sino-nasal adenocarcinomas were observed among men in the "less dusty" jobs, while one was observed (0.01 expected) among "dusty" jobs (polishers, veneerers, and maintenance workers), and seven were observed among the "very dusty" jobs (Cabinet and chair makers, sanders, wood machinists). Sampling conducted in the High-Wycombe furniture industry in 1983 observed mean inhalable dust concentrations in different factories ranging from 2.1 to 8.1 mg/m^3 with the highest exposures across the factories among hand sanders (mean=6.9 mg/m^3) and machine sanders (mean, 5.7 mg/m^3).[137] A comparison with *inhalable* dust sampling results from the same factories conducted in 1976/77 indicated that exposures were significantly higher in the earlier period. An exposure survey of five factories in the same area published in 1974 reported an overall mean dust level of 5.9 mg/m^3.[50] The type of sampler was not reported but assumed to be for total dust; however, it is possible that inhalable samplers were used.

While some case–control studies have identified tree species anecdotally, only one large case–control study has examined the risk of sino-nasal cancer associated with hardwoods and softwoods separately.[135] Based on interviews with study participants, all 80 male adenocarcinoma cases had been exposed to hardwood dusts, either alone (n=7) or in combination with soft, tropical, or composite woods, but none had been exposed to softwoods, tropical woods, or composite woods alone. Of the 17 male squamous cell cases who had been exposed to wood dust, 3 had been exposed to hardwood only, 3 to softwood only, and the remainder to a mixture of woods. Of the 76 exposed controls, 15 had been exposed to hardwoods alone, 15 to softwoods alone, 2 to composite woods alone, and 1 to tropical woods alone. One smaller Nordic case–control study[138] also reported results based on type of tree and observed an excess among workers exposed primarily to softwoods (OR=3.3, pine and spruce, but also birch and aspen), hardwood (OR=2.0), and mixed wood (OR=12.0), but the results were not reported for specific histologic types. Tree species was assumed based on industry and regional practices.

Information regarding the species of tree was primarily available from sino-nasal adenocarcinoma case series. Acheson and colleagues[139] reported that 24 cases in the British furniture industry had been exposed to oak, mahogany, beech, birch, and walnut. Leroux-Robert[140] reported that 26 French cases had used European hardwoods and 22 had used oak, either mainly or exclusively. Luboinski and Marandas[141] reported that 21 French cases had been exposed to oak, chestnut, cherry, walnut, beech, poplar, and mahogany. Andersen and colleagues[84] reported that 12 Danish cases had primarily used beech, oak, and walnut and, periodically, mahogany and teak. Engzell and colleagues[142] reported that 19 Swedish cases employed as joiners and cabinet makers had been exposed to oak, beech, mahogany, and birch and never exclusively to softwoods. Finally, Kleinsasser and Schroeder[143] reported that 77 German cases had been exposed to oak and beech, and never exclusively to softwoods or tropical woods. One Norwegian study[144] reported on seven patients with sino-nasal squamous cell carcinoma who had only been exposed to pine and spruce.

In general, the relative risks observed in North American studies have been considerably lower than those observed in European studies. In the pooled analysis of 12 case–control studies, the excess of sino-nasal adenocarcinoma among wood dust-exposed workers was limited to the 8 European studies and 1 U.S. study[133] with the remaining 2 U.S. studies and 1 Chinese study having no exposed cases.[134] Other U.S. and Canadian studies have examined the risk for all types of sino-nasal cancer together and observed excess risks,

but these have ranged from 1.5 to 4.4.[145–152] When a pooled analysis of the data from four cohorts of U.S. woodworkers and the British furniture workers was performed, the sino-nasal cancer mortality excess appeared to be restricted to the British cohort.[153] However, the U.S. studies had relatively low power for detecting a two- or threefold excess risk as might be expected, based on the case–control studies, and had little exposure data available. One cohort study of Canadian softwood sawmill observed an excess of sino-nasal cancer (standardized incidence ratio [SIR], 1.9; 7 observed versus 3.6 expected) that was not related to chlorophenol fungicide exposure.[154] However, no analysis in relation to wood dust exposure was presented.

Teschke et al.[155] reported on surveillance of incident nasal cancers in British Columbia from 1990 to 1992 using a case–control methodology with follow up interview to investigate occupational exposure history. As opposed to earlier investigations in British Columbia and nearby Washington State, there was no association with occupations exposed to wood or wood dust and an increased risk of nasal cancer. The authors suggested that this might reflect the decrease of occupational wood dust exposure in British Columbia to < 1 mg/m^3.

Several studies have compared the nasal histology of workers who were exposed to wood dust to unexposed controls. Boysen and Solberg[156] collected nasal biopsies from 103 workers from 5 Norwegian furniture factories and 54 controls. Metaplastic squamous epithelium were observed in 40% of the furniture workers and 17% of controls while dysplasia were observed in 12% and 2%, respectively. Boysen and colleagues[157] also collected nasal biopsies from 44 men who had only been exposed to softwoods. Four wood workers, who had been exposed for 20 or more years, had dysplasia versus zero in the controls. Wilhelmsen and Lundh[158] collected nasal biopsies from 45 Swedish furniture workers and 17 controls. Metaplastic cuboidal epithelium was significantly more frequent and columnar epithelium significantly was less frequent, while no difference in the prevalence of metaplastic squamous epithelium was observed.

Genotoxicity

Nelson et al.[159] and others have shown dose dependent genotoxicity in animals from hardwood dust exposure per se as measured by *in vivo* induction of micronuclei in rat nasal epithelium.[45,117–121] Some studies have examined genetic and related effects among humans exposed to wood dust. Spitz et al.[160] found a significantly increased odds ratio (OR, 2.8) for reported wood dust exposure among 165 cases of lung cancer versus 239 controls associated with the risk of mutagen sensitivity (based on an *in vitro* assay quantitating mutagen induced chromatid breaks), despite controlling for smoking and mutagen sensitivity. Stratified analysis revealed a greater than multiplicative interaction between wood dust and both smoking and mutagen sensitivity. Kurttio and colleagues[161] examined the prevalence of chromosomal aberrations among 13 nonsmoking male plywood workers compared to 15 nonsmoking, age-matched controls. Significantly ($p<0.01$) more chromatid breaks were observed among the exposed. Jiang and colleagues[162] studied the micronucleus frequency in the peripheral lymphocytes of 298 match factory workers exposed to poplar and linden dust compared to 45 waiters. Exposed workers had significantly ($p<0.01$) more micronuclei than the controls, although no evidence of a dose–response was apparent. In additional studies by Jiang et al.,[163] 83 workers in a wood processing factory with birchen dust (1.26 ± 0.41 mg/m^3) had significantly higher micronucleus frequency in peripheral blood lymphocytes ($p<0.01$) compared to controls. Of note, the micronucleus tests in mice using steamed or baked birchen dust significantly lowered the inducing effect in the micronucleus test. Palus and colleagues[164] examined the prevalence of DNA single-strand breaks in peripheral lymphocytes of 24 wooden furniture workers and 28 controls. Significantly ($p<0.05$) more DNA single-strand breaks were observed among the exposed. Additional studies by Palus et al.[165] evaluated the effect of cigarette smoking on single strand breaks and DNA repair versus occupational exposure among wood furniture workers. Occupational exposure had a significant effect on DNA repair in nonstimulated lymphocytes regardless of smoking status compared with controls ($p<0.05$). Furthermore, Palus et al.[166] found similar results for occupational wood exposure compared to controls in DNA damage detected by the comet assay in white blood cells, regardless of smoking ($p<0.005$).

Recently researchers using *in vivo* and *in vitro* assays have argued that the carcinogenicity of wood dust is entirely attributable to contamination by other known carcinogen used in wood treatment and preservation.[121,167–169] However, this research is not considered relevant to the discussion of the carcinogenicity of wood dust exposure.

TLV Recommendation

Western Red Cedar

There have been many studies showing the strong association between occupational exposure to Western red cedar dust as an allergen and occupational asthma. Brooks and colleagues[111] observed no asthma cases among splitters exposed at 0.5 mg/m^3 total dust. Vedal and colleagues[112] observed a 5% asthma prevalence among workers exposed to less than 1 mg/m^3 total dust (of note, this is a relatively low reported prevalence of asthma even for the general population), but they observed a 15% prevalence in the group exposed to over 2

mg/m^3 total dust (with a maximum value of 6 mg/m^3) of Western red cedar dust. Chan-Yeung and Desjardins[114] reported that four workers from the same facility first developed asthma after being transferred from jobs with exposure below 0.3 mg/m^3 total dust to jobs with exposures ranging from 0.2 to 0.6 mg/m^3 *total dust*. Noertjojo and colleagues[115] observed significant declines in lung function among workers whose mean exposure was 0.3 mg/m^3 total dust, but not among those whose mean exposure was 0.13 mg/m^3 total dust. Asthma has been attributed to occupational exposure to allergenic dusts from many different tree species in addition to Western red cedar, based on case reports or epidemiologic studies (Table 1). Unfortunately, very little data regarding the actual levels of exposure necessary to cause asthma are available.

Based on the studies of Western red cedar, a TLV–TWA of 0.5 mg/m^3, inhalable particulate mass, is recommended. A sensitization (SEN) notation is also appropriate.

All Other Wood Dust Species

Studies of workers exposed to wood dust have observed decreased lung function compared to unexposed controls[81,91,94,99,101–103,115] or across a workshift.[53,89,102] These effects have been observed among workers exposed to wood dust from a variety of tree species in a variety of settings (e.g., lumber mills, furniture manufacturing, cabinet making) and countries.

Some studies examined lung function or symptoms by level of exposure and observed patterns consistent with a dose–response relationship,[54,56,84–86,89,102,103] although only a few of these studies have supplied the information necessary to determine a TLV.[54,67,84–87,89,100] Andersen et al.[84] observed a dose–response relationship between the level of exposure and prevalence of mucostasis, with an 11% prevalence of mucostasis among workers with a mean exposure of 2.2 mg/m^3 total dust. Halpin and colleagues[54,100] observed an increased prevalence of symptoms among workers with a mean exposure of 3.0 mg/m^3 inhalable dust, compared to both workers with a mean exposure of 0.7 mg/m^3 inhalable dust and controls. Based on the study by Holness and colleagues,[89] a 10% drop in FEV_1 would be predicted, based on 1.5 mg/m^3 total dust exposure over a 40-year working life. Whitehead[87] estimated that chronic personal exposures above 2 mg/m^3 total dust from both hardwoods and softwoods were associated with decreased pulmonary function among sawmill and furniture workers Furthermore, pine-exposed workers had significantly decreased postshift FEV_1 and increased reported wheezing at these relatively low levels. This study was a cross-sectional design associated with the underestimation of risk associated with the healthy-worker effect (i.e., that ill workers will drop out of the workforce and are less likely to be represented in cross-sectional studies).

While there was some variability in the results, many other studies have observed increased symptoms or decreased lung function among workers with mean exposures to wood dust that were similar to those reported above. The highest reported level at which no adverse effects were observed was 3.3 mg/m^3 inhalable dust;[53] however, that was not a recent study, and measurement of inhalable dust has become more precise. Subsequently, Wilhelmsson and Drettner[88] observed decreased mucociliary clearance and FVC among furniture workers with a mean exposure of 2 mg/m^3 total dust. Goldsmith and Shy[81,91] observed decreased air flow among workers whose mean exposure was described as approximately 2 mg/m^3 total dust or less. Although there were biohazardous, concomitant compounds such as endotoxin, Mandryk and colleagues[102] observed decreased pulmonary function and increased symptoms among sawmill workers with a mean exposure to 4.8 mg/m^3 inhalable dust; additional studies by Mandryk et al.[63] showed significantly increased respiratory symptoms and lung function changes (including cross-shift decrements) with geometric mean of 1.53 mg/m^3 inhalable dust for green mills and 1.71 mg/m^3 for dry mills. ?hmans and colleagues[92,93] observed an elevated prevalence of respiratory symptoms among industrial arts teachers with a mean exposure of 0.6 mg/m^3 total dust, but participants were also potentially exposed to other respiratory irritants. Chan-Yeung and colleagues[99] observed decreased lung function among sawmill workers with a mean exposure of 0.5 mg/m^3 total dust, but the authors described the changes as slight. Most recently in a cross-sectional study, Schlunssen[67] found increased reported asthma and asthma symptoms in Danish woodworkers (including decreased postshift FEV_1 among pine exposed workers) exposed to predominantly soft woods of 0.94 $\pm$ 2.10 mg/m^3 inhalable and 0.60 $\pm$ 1.96 mg/m^3 total dust, but with a relatively wide deviation of dust measurement. There is much inconsistency in the studies with regard to the specific nature of the health effects; nevertheless, to protect workers from the developing decreases in pulmonary function and other occupational lung diseases, a TLV–TWA of 1 mg/m^3, inhalable particulate mass, is recommended.

Carcinogenicity of Wood Dusts

Many studies have observed large excess risks of sino-nasal cancer, particularly adenocarcinoma, among workers employed in wood dust-exposed jobs.[45] Based on interviews with cancer patients, exposure to oak and beech was clearly associated with an excess risk of cancer, while birch, mahogany, teak, and walnut were strongly suspected.[45,84,135,139–143] However, the mechanism by which exposure to wood dust increases the risk of cancer is not clear, and it is possible that other tree

species are also carcinogenic. Based on the evidence currently available, an A1, Confirmed Human Carcinogen, notation is assigned to beech and oak, while an A2, Suspected Human Carcinogen, notation is assigned to birch, mahogany, teak, and walnut. The remaining tree species are assigned a designation of A4, Not Classifiable as a Human Carcinogen.

No studies have thus far examined the risk of sino-nasal cancer in relationship to quantitative estimates of wood dust exposure, although a number of studies have observed a dose–response relationship using semi-quantitative estimates of wood dust exposure based on job title and industry.[130,134–136] Because of the long latency of sino-nasal cancer, it is largely assumed that effective period of exposure for most studies was 20 to 30 years prior to diagnosis, which equates to the 1950s and 1960s for most studies. Unfortunately, there are very few measurements from that period. Sampling in the British furniture industry in the early 1980s indicated exposure levels ranging from 2.1 to 8.1 mg/m^3 inhalable dust, while the mean exposure in a 1974 survey was 5.9 mg/m^3 total dust, and it can be safely assumed that earlier exposures were at least as high. While the very high risks of sino-nasal cancer may have been associated with exposure levels greater than 2 mg/m^3 inhalable dust, it is not clear whether relative risks of 2 to 5, which have been observed in more recent studies, may be associated with lower exposures. Recent data suggest that occupational wood dust levels < 1 mg/m^3 (inhalable particulate mass) may substantially decrease the risk of nasal cancer.[155] These data further support the TLV Committee recommendation of a TLV–TWA of 1 mg/m^3, inhalable particulate mass, for nonallergenic wood dusts discussed above, based on prevention of occupational asthma.

TLV Basis — Critical Effect(s)

Western red cedar: Asthma
All other species: Pulmonary function.

TLV Chronology

1972: TLV–TWA, 5 mg/m^3, Nonallergenic
1981–present: TLV–TWA, 1 mg/m^3, Certain hardwoods (e.g., beech, oak)
TLV–TWA, 5 mg/m^3; TLV–STEL, 10 mg/m^3, Softwoods
1998 *proposed*: TLV–TWA: 5 mg/m^3, inhalable particulate; Sensitizer (SEN) — Hardwoods and Softwoods (nonallergenic); Certain hardwoods (beech, birch, mahogany, oak, and walnut); Softwoods (nonallergenic); and Hardwoods and Softwoods (mixture)
TLV–TWA: 0.5 mg/m^3, inhalable particulate; Sensitizer (SEN) — Western red cedar
Carcinogenicity: A1, Confirmed Human Carcinogen — Certain hardwoods (beech, birch, mahogany, oak, and walnut); and Hardwoods and Softwoods (mixture); A4, Not Classifiable as a Human Carcinogen — Softwoods (nonallergenic); and Western red cedar
1999 *proposed*: TLV–TWA: 5 mg/m^3, inhalable particulate — Hardwoods and Softwoods (nonallergenic)
TLV–TWA: 5 mg/m^3, inhalable particulate; SEN — Beech, Birch, Mahogany, Oak, Teak, and Walnut; and Softwoods and other Hardwoods (allergenic)
TLV–TWA: 0.5 mg/m^3, inhalable particulate; SEN — Western red cedar
Carcinogenicity: A1, Confirmed Human Carcinogen — Beech and Oak; A2, Suspected Human Carcinogen — Birch, Mahogany, Teak, and Walnut; A4, Not Classifiable as a Human Carcinogen — Softwoods and Other Hardwoods (allergenic); and Western red cedar
2001 *proposed*: TLV–TWA: 2 mg/m^3, inhalable particulate; A4, Not Classifiable as a Human Carcinogen — Nonallergenic and Noncarcinogenic Wood Dust
TLV–TWA: 0.5 mg/m^3, inhalable particulate; SEN; A4 — Western red cedar
TLV–TWA: 1 mg/m^3, inhalable particulate; SEN; A4 — Other Respiratory Allergic Wood Dust
TLV–TWA: 1 mg/m^3, inhalable particulate — Confirmed or Suspected Carcinogenic Wood Dust
Carcinogenicity: A1, Confirmed Human Carcinogen — Beech and Oak; A2, Suspected Human Carcinogen — Birch, Mahogany, Teak, and Walnut
2003 *proposed*: Wood Dusts

Nonallergenic species

TLV–TWA, 1 mg/m^3, inhalable particulate mass

Western red cedar

TLV–TWA, 0.5 mg/m^3, inhalable particulate mass; SEN

Carcinogenicity

A1, Confirmed Human Carcinogen: Oak and Beech
A2, Suspected Human Carcinogen: Birch, Mahogany, Teak, and Walnut
A4, Not Classifiable as a Human Carcinogen: All other wood dust species

2005*:* Wood Dusts

Western Red Cedar

TLV–TWA, 0.5 mg/m^3, inhalable particulate mass; SEN; A4

All Other Species

TLV–TWA, 1 mg/m^3, inhalable particulate mass

Carcinogenicity

A1, Confirmed Human Carcinogen: Oak and Beech
A2, Suspected Human Carcinogen: Birch, Mahogany, Teak, and Walnut
A4, Not Classifiable as a Human Carcinogen: All other wood dust species

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