HEALTH EFFECTS OF EXPOSURE TO WOOD DUST

A SUMMARY OF THE LITERATURE

U.S. Department of Health and Human Services
Public Health Service
Centers for Disease Control
National Institute for Occupational Safety and Health
Division of Standards Development and Technology Transfer

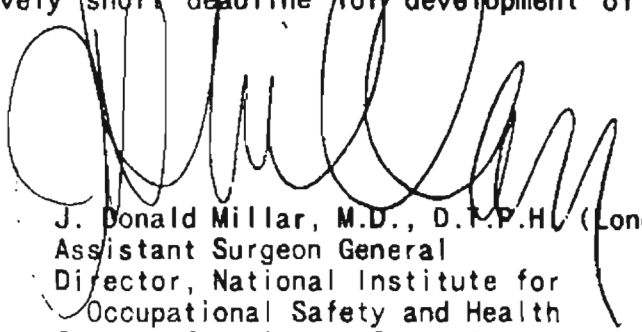
June 1987

DISCLAIMER

Mention of the name of any company or product does not constitute endorsement by the National Institute for Occupational Safety and Health.

FOREWORD

This document identifies and summarizes the literature that describes the potential health effects resulting from exposure to wood dust. It was developed by the National Institute for Occupational Safety and Health (NIOSH) at the request of the U.S. Department of Labor, Occupational Safety and Health Administration (OSHA). This document provides a comprehensive synopsis of the literature related to wood dust. The conclusions described herein are those of the cited authors. The report itself does not contain NIOSH recommendations for regulating occupational exposure to wood dust, nor is it a critical evaluation of the literature. Such recommendations and evaluation were precluded by the volume of published data and the relatively short deadline for development of the document.



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ABSTRACT

This report identifies and summarizes the literature on the potential health effects of exposure to wood dust. This comprehensive literature review defines wood, discusses occupational exposure standards and recommendations for wood dust, describes reports of animal and in vitro testing, and examines human health effects including dermatitis, allergic respiratory effects, mucosal and nonallergic respiratory effects, and carcinogenicity. Also included is a compilation of wood dust concentration ranges reported for various woodworking operations and a brief discussion of wood dust control technology. The report does not contain NIOSH recommendations for regulating occupational exposure to wood dust.

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ACKNOWLEDGMENTS

This document was developed by the Division of Standards Development and Technology Transfer, National Institute for Occupational Safety and Health. Rodger L. Tatken was responsible for preparing the document. Contributions of the following Division staff are gratefully acknowledged: Howard R. Ludwig, for compilation of the industrial hygiene sampling data in Section VII; and Bryan D. Hardin, Ph.D., G. Kent Hatfield, Ph.D., Theodore J. Meinhardt, Ph.D., Richard W. Niemeier, Ph.D., John J. Whalen, Lawrence F. Mazzuckelli, David H. Groth, M.D., and Sheldon H. Rabinovitz, Ph.D. (currently with the U.S. Environmental Protection Agency), for their critical reviews and comments. NIOSH review of this document was provided by Stephen G. Bayer, Raymond E. Biagini, Ph.D., JeAnne R. Burg, Ph.D., John F. Gamble, Ph.D., Charles McCammon, Ph.D., Michael A. McCawley, Ph.D., and William J. Moorman. Carolyn A. Browning performed editorial review.

I. INTRODUCTION

This document identifies and summarizes the literature that describes the potential health effects resulting from exposure to wood dust. For purposes of this document, wood dust is defined in its broadest sense--that is, any wood particles arising from the processing or handling of wood. Because this definition is so broad, every effort has been made to describe as thoroughly as possible the circumstances of the wood dust exposures (e.g., type of wood, dust concentration, and concomitant exposures) as reported by the authors. It should be noted, however, that for much of the literature, this type of information is not available.

The document contains seven sections. This introductory section briefly defines wood and discusses occupational exposure standards and recommendations for wood dust. Section II summarizes reports of animal and in vitro testing. Sections III - VI examine human health data and are arranged by effects, including: dermatitis, allergic respiratory effects, mucosal and nonallergic respiratory effects, and carcinogenicity. Section VII contains a compilation of wood dust concentration ranges reported for various woodworking operations and a brief discussion of wood dust control technology.

Wood is a complex biological and chemical material that consists primarily of cellulose, hemicellulose, and lignin. Cellulose is a linear glucose chain that serves as wood's skeleton. Hemicellulose is a small polysaccharide that may assist in cell growth and maturation. Lignin is a high molecular weight polymer of phenoxyl-propane units that functions in holding wood cells together. Wood may contain a variety of complex organic compounds, including glycosides, quinones, tannins, stilbenes, terpenes, aldehydes, and coumarins. It is beyond the scope of this paper to provide a detailed discussion of the chemistry of wood. The reader is referred to any of numerous texts on the subject (e.g., Esau 1965,1977; Browning 1975; Kollmann and Cote 1968; Sjostrom 1981; Haygreen and Bowyer 1982); reviews are provided in Woods and Calnan (1976), IARC (1981), and Holliday et al. (1986).

Woods have been divided into two different classes, hardwoods and softwoods, each with its own cellular structure and chemical composition. For example, hardwoods have a lower lignin content than softwoods (Holliday et al. 1986). Hardwoods (angiosperms) are the deciduous broad-leaved flowering species. Softwoods (gymnosperms) are the coniferous species that do not normally shed their leaves in winter.

According to Woods and Calnan (1976), the first occupational reference to adverse health effects from wood was made in 1700 by Bernardino Ramazzini,

who reported nose and eye irritation in pitsawyers and headaches in wood turners. In 1902, upper respiratory tract inflammation was described in men working with sequoia wood, and throat and eye irritation was reported in workers who used a boxwood substitute (Woods and Calnan 1976). Three years later, Legge (1905) reported that "African boxwood" caused such severe symptoms among Lancashire shuttlemakers that the men refused to work with it. Following these initial case reports, a substantial body of literature on the health effects of exposure to wood and wood dust has accumulated. Two comprehensive reviews of wood toxicity include Woods and Calnan (1976) and Hausen (1981); briefer treatments are provided in Hanslian and Kadlec (1966), Kadlec and Hanslian (1983), and Jagels (1985).

Until recently, occupational exposure to wood dust was regulated in the U.S. under OSHA's inert or nuisance mineral dust standard (29 CFR* 1910.1000, Table Z-3) at an 8-hr time-weighted average (TWA) concentration of 5 mg/m^3 air for respirable dust and 15 mg/m^3 for total dust. However, several OSHA citations for exceeding nuisance dust levels for grain dust exposures were appealed to the Occupational Safety and Health Review Commission (OSHRC). These cases, Bunge Corporation (OSHRC Docket Nos. 77-1622, 78-838, and 78-2213) and Krause Milling Company (OSHRC Docket No. 78-2307), challenged the applicability of the nuisance dust standard to soybean dust and corn dust, respectively. In both cases, the Review Commission held that the nuisance dust standard did not apply to organic dusts. In a third case that involved wood dust exposure (Bemis Manufacturing Company, OSHRC Docket No. 80-3443), the Administrative Law Judge ruled that wood dust was not an inert mineral dust and therefore not subject to the nuisance dust standard. Although this ruling was not precedent setting, OSHA elected not to appeal it to the OSHRC. No other related cases have since been brought before the Review Commission, and OSHA has not amended Table Z-3 (29 CFR 1910.1000). Therefore, the current law, as stated by the Review Commission's decisions, is that the nuisance dust standard applies to mineral dusts only and not to organic dusts.

Several other countries currently regulate occupational exposure to wood dust. Some countries name specific wood species (e.g., "pine, oak, fir, and beech" in the German Democratic Republic) or a particular class of wood dust (e.g., "hardwood" and "softwood" in Canada and Norway; "nonallergenic" in the United Kingdom and the Netherlands). Other countries, such as Hungary and Poland, regulate "dust of vegetable and animal origin," with varying percentages of free silica. Still others, such as Switzerland, regulate simply "dust." A list of wood dust exposure standards and recommendations is included in Table 1.

In 1980, the American Conference of Governmental Industrial Hygienists (ACGIH) revised its recommended wood dust threshold limit value (TLV®) TWA from 5 mg/m^3 air for nonallergenic dust (ACGIH 1971) to 1 mg/m^3 for hardwood and 5 mg/m^3 for softwood (nonallergenic) dusts, with a short-term (15-min) exposure limit (STEL) of 10 mg/m^3 for softwood only (ACGIH 1980). The revised recommendation does not differentiate between respirable

*Code of Federal Regulations. See CFR in references.

Table 1.--Wood dust exposure standards and recommendations*

Country	Limit** (mg/m^3) TWA	STEL	Comments
Australia			
New South Wales	1		Hardwood
	5		Softwood
Queensland	1		Hardwood
	5		Softwood
Tasmania	1		Hardwood
	5		Softwood
Canada			
Federal	1		Hardwood
	5	20	Softwood
Alberta	5	10	Nonallergenic wood dust
	2.5	5	Allergenic wood dust
British Columbia	5	10	Nonallergenic wood dust
	2.5	5	Allergenic wood dust
	1		Hardwood (guideline)
	5	10	Softwood (guideline)
Manitoba	1		Hardwood (guideline)
	5	10	Softwood (guideline)
New Brunswick	1		Hardwood
	5	10	Softwood
Newfoundland	1		Hardwood
	5	10	Softwood
Nova Scotia	1		Hardwood
	5	10	Softwood
Prince Edward Island	1		Hardwood (guideline)
	5	10	Softwood (guideline)
Quebec	5	10	Nonallergenic wood dust

(Continued)

See footnotes at end of table.

Table 1 (Continued).--Wood dust exposure standards and recommendations*

Country	Limit** (mg/m^3) TWA	STEL	Comments
Canada (continued)			
Saskatchewan	5	10	Nonallergenic wood dust
	1	3	Hardwood
Northwest Territories	1		Hardwood
	5	10	Softwood
Yukon Territory	1		Hardwood (guideline)
	5	10	Softwood (guideline)
Denmark	5		Fine dust
Federal Republic of Germany (West Germany)			No numeric exposure limits; beech and oak dust are listed as having been "shown epidemiologically to be unequivocally carcinogenic;" all other wood dust is "justifiably suspected of having carcinogenic potential" (DFG 1985)
Finland	5		Wood dust
German Democratic Republic (East Germany)	10		Domestic wood dust only (pine, oak, fir, beech)
Hungary	1,000 particles per cc		Other dusts including vegetable and animal origin
India	15		Total dust
Italy	5		Proposed standard
The Netherlands	5		Nonallergenic wood dust

(Continued)

See footnotes at end of table.

Table 1 (Continued).--Wood dust exposure standards and recommendations*

Country	Limit** (mg/m^3) TWA	STEL	Comments
New Zealand	1		Hardwood (guideline)
	5	10	Softwood (guideline)
Norway	1		Hardwood (exotic)
	5		Softwood (Nordic)
Poland	2		Natural, vegetable, and animal dust containing more than 10% free SiO_2
	4		Natural, vegetable, and animal dust containing less than 10% free SiO_2
Sweden	4		Dust from wood containing no impregnating substances
	2		Dust from wood impregnated with substances whose content cannot be determined
			Wood dust classified as carcinogenic (NSBOSH 1984)
Switzerland	20		Total dust
	8		Fine dust
United Kingdom	5	10	Nonallergenic wood dust
United States	5	10	Softwood, nonallergenic (recommendation, ACGIH 1980)
	1		Hardwood (recommendation, ACGIH 1980)
USSR	2		Dust of vegetable or animal origin; greater than 10% free silica
	4		Dust of vegetable or animal origin; less than 10% free silica

(Continued)

See footnotes at end of table.

Table 1 (Continued).--Wood dust exposure standards and recommendations*

Country	Limit** (mg/m^3) TWA	STEL	Comments
Yugoslavia	10		Total dust of vegetable or animal origin, without SiO_2
	3		Respirable dust of vegetable or animal origin, without SiO_2

*This table is adapted from IARC (1981), ILO (1980), Holliday et al. (1986), ACGIH (1980), DFG (1985), and NSBOSH (1984). Note that the first two references list the foreign standards shown under "Limit" as "threshold limit values." It is assumed that this term is used to actually mean "time-weighted average," since "threshold limit value" (TLV®) is a registered ACGIH name that refers specifically to an ACGIH exposure recommendation. A TLV® may be given as either a time-weighted average (TWA) or short-term exposure limit (STEL).

**Except as noted under Comments, all values are for standards.

and total dust. It is based primarily on mucostasis and its role in the development of adenocarcinoma in furniture workers because of prolonged retention of wood dust in the nasal cavity.

Based on their own research, several authors have recommended the adoption of other wood dust exposure limits. Hanslian and Kadlec (1964) proposed maximum allowable concentrations of 1 mg respirable dust/m^3 air for toxic and strongly allergenic woods (e.g., boxwood, cocobolo, mansonia, yew); 5 mg/m^3 for biologically active woods (e.g., pine, larch, ebony, mahogany, teak, cedar); and 10 mg/m^3 for biologically indifferent woods or those with little biological activity (e.g., oak, beech, maple, ash, lime, birch). Ruppe (1973) recommended a concentration of 5 mg/m^3 for "exotic woods" (undefined by author) to avoid allergic respiratory reactions. Brooks et al. (1981) suggested that for Western red cedar dust, a TWA below 3.5 mg/m^3 total dust would protect workers from occupational asthma. Whitehead (1982) proposed a total wood dust concentration of 2 mg/m^3 for both hardwood and softwood dusts for effects other than extreme allergic hypersensitivity. He commented that in the absence of a concentration-response relationship for wood dust and nasal cancer, this limit should reduce the occurrence of mucostasis to background levels and minimize wood dust contact with nasal sinus tissue. However, he also indicated that this limit did not include a margin of safety. Imbus and Dyson (1985) recommended a TLV (assumed to mean TWA) of 5 mg/m^3 for both hardwoods and softwoods to prevent nonallergic, nonneoplastic respiratory disease, and a TLV of 2.5 mg/m^3 total dust for "potentially allergic woods where epidemiologic studies show significant incidence and prevalence of allergic effects such as Western red cedar and mansonia, but not to woods where only incidental case reports are available, such as oak and mahogany." Stuart et al. (1986) proposed a particle-size-selective threshold limit value for wood dust, although their recommendation did not specify quantitative dust concentrations.

II. ANIMAL AND IN VITRO STUDIES

A. Animal Studies

The literature contains relatively few reports of laboratory animal studies on the toxicity of wood dust per se. The earliest such study appears to be that of Bergman et al. (1943), who exposed rabbits for 8 hr/day, 5 1/2 days/week for up to 30 months to red cedar and pine dusts at concentrations of 6 to 10 million particles per cubic foot of air. Sixty-five percent of the particles exceeded 25 um, and only a few (percent unspecified) were below 10 um. Pathological examination revealed lung effects ranging from large abscesses to bronchopneumonia and bronchitis. The nasal passages and upper respiratory tracts of all animals contained wood dust.

Much of the subsequent animal research has dealt with the toxicity, in particular the carcinogenicity, of various chemical substances found in plants and woods. Morton (1968) published the first of a series of papers on the toxic properties of plants used in folk medicine on the island of Curacao. Because esophageal cancer had been common on Curacao (Eibergen 1961), Morton undertook research with O'Gara et al. (1971) at the National Cancer Institute (NCI) on the carcinogenicity of decoctions (extracts prepared by boiling) of selected parts of plants from Curacao in mice and rats, using the screening method devised by O'Gara (1968). After the death of O'Gara in January 1971, the screening program was continued in cooperation with a group of investigators at Howard University and NCI (Kapadia et al. 1976,1978; O'Gara et al. 1974; Pradhan et al. 1974; Dunham et al. 1974). Leaves or leafy twigs, roots or root bark, and fruits were the most commonly examined parts of plants; no samples of wood were extracted for screening. When plants were known to contain carcinogens other than tannic acid (IARC 1976), such as senecio alkaloids (Harris and Chen 1970) and safrole (Long et al. 1963), appropriate steps to free the extracts of these materials were taken. The general tenor of the information developed by this research was that the carcinogenicities of the extracts were functions of their concentrations of tannin; however, tannins did not account for all the carcinogenic activity found.

Gibbard and Schoental (1969) identified several aldehydes, including sinapyl and syringic aldehydes, in Chinese incense and in eight types of wood (eucalyptus, teak, beech, sandlewood, oak, coconut palm, larch, and juniper). Schoental et al. (1971) and Schoental and Gibbard (1972) administered a sinapylaldehyde derivative, 3,4,5-trimethoxycinnamaldehyde, intraperitoneally and subcutaneously to small groups of rats and noted the development of fatty kidney tumors and nasopharyngeal cancer. Although these experiments were too limited in nature to permit firm conclusions, it

was suggested that epoxidation of the aldehyde might be responsible for the tumorigenic activity seen.

The carcinogenicity of other chemical compounds found in wood has also been studied. A detailed description of these studies is beyond the scope of this paper, and the reader is referred to reviews by Tinkler (1986), Sigman et al. (1984), Niemeier (1979), and Gamble (1979) for additional information.

The potential of wood shavings used as animal bedding material to cause tumors was examined in a series of studies reported in the 1970's. Interest was initially raised by Heston and Vlahakis (1968), who had reported on the high incidence of mammary gland and liver tumors in C3H-derived mice strains in their National Institutes of Health (NIH) laboratory. Schoental (1973,1974), based on her previously described work, proposed that spontaneous tumors in laboratory rodents might be due to carcinogenic constituents, including alpha, beta-unsaturated lignin aldehydes and podophyllotoxin, of wood shavings used as animal bedding.

Sabine et al. (1973) reported their observations on C3H-A^{vy} and C3H-A^{vy}fB mice bred and reared in Australia. Historically, those strains had a 90-100% spontaneous incidence of liver and mammary tumors in the U.S. (Heston and Vlahakis 1968, Vlahakis et al. 1970). However, Sabine et al. (1973) reported that colonies of both mouse strains derived from U.S. breeding pairs and raised in Australia on North American Douglas fir sawdust bedding and Australian feed had a significantly ($p<0.001$) reduced incidence of both type of tumors when compared to those previously raised in the U.S. (Heston and Vlahakis 1968, Vlahakis et al. 1970). For mammary tumors, the incidence in Australia declined to almost zero by the second generation. When C3H-A^{vy} mice in Australia were maintained on fir sawdust bedding but given U.S. feed, mammary tumor incidence increased nonsignificantly from 46% (controls) to 50%. However, when they received both U.S. bedding material (Eastern red cedar) and feed, the incidence of mammary tumors increased significantly ($p<0.025$) to 76%. There was also a nonsignificant decrease in mean latency period until tumor appearance from 9.3 months in the controls to 6.4 months in the mice reared on U.S. food and bedding. However, the U.S. feed and bedding significantly ($p<0.001$) increased the average weight of the adult mice. The authors cautioned that these results were preliminary and did not rule out other reasons for differences in tumor incidence, such as genetic variation. However, because the addition of cedar bedding restored the mammary tumor incidence in Australian mice to levels near that historically seen in U.S. mice, they believed that cedar bedding was implicated as a carcinogen.

In response to the above report by Sabine et al. (1973), Heston (1975) examined the occurrence of tumors in the same two strains of mice raised in the U.S. The animals were fed either the Australian diet used by Sabine and coworkers or the same U.S. food on which the colony had formerly been maintained. The animals raised on the softer Australian feed ate more and grew more rapidly than those on the U.S. diet. However, no significant differences in the incidence of mammary or liver tumors due to diet were seen, which agreed with the observations made by Sabine and coworkers. To examine the effect of bedding material, C3H-A^{vy} mice were maintained on a

bedding of either three-fourths pine sawdust plus one-fourth cedar shavings, or on pine sawdust only, and fed the NIH open-formula diet, which was more nutritious than the standard U.S. diet previously used. No significant differences in the growth rate or mammary and liver tumor incidence were seen. Heston (1975) commented that the animals in the Sabine et al. study were in poorer health and more prone to infection. They had heavy mite infestation and significantly lower weight gain. This may have reduced the incidence of tumors by shortening the life span of the animals raised in Australia. Since Heston and Vlahakis (1966) had shown that factors causing decreased animal growth also decreased tumor occurrence, Heston concluded that the reduction in tumor incidence reported by Sabine and coworkers was due to lower weight gain and ectoparasitic infestation.

Sabine (1975) summarized his tumor incidence data in three susceptible strains of mice (C3H-A^{vy}, C3H-A^{vy}fB, and CBA/J) raised in Australia and compared them with reported U.S. data. He reported a significantly ($p<0.05$) increased incidence of spontaneous tumors of the liver and mammary gland, and a reduced mean time to tumor appearance, in all three strains of mice raised on cedar shavings as opposed to those raised on Douglas fir bedding. The hexobarbital sleeping time decreased significantly ($p<0.001$) and hepatic levels of cytochrome P_{450} and associated liver metabolizing enzymes increased significantly (p unspecified) in mice reared on cedar shavings in comparison with those bedded on Douglas fir sawdust. These sleep time changes and enzyme increases were similar to the results reported previously by Ferguson (1966), Vesell (1967), Wade et al. (1968), and Hashimoto et al. (1972). In a "Note Added in Proof" at the conclusion of his paper, Sabine (1975) discussed the previous report of Heston (1975). Sabine indicated that the effect of cedar would have been difficult to assess because the tumor incidence in Heston's noncedar group was virtually 100%. The number of hepatomas per animal was slightly higher in the males raised on cedar, but the difference may not have been significant. Finally, the unstated methods used by Heston to prevent mite infestation may have elevated hepatic enzyme levels. Sabine, therefore, concluded that the question of cedar carcinogenicity in mice remained unresolved.

Vlahakis (1977) studied the effect of bedding material on carcinogenicity in C3H-A^{vy}fB mice. Thirty-seven brother x sister pair matings were made on bedding of mixed North American species of low-resin pine sawdust, and 22 littermate pairs were mated and bedded on a mixture of the same pine sawdust and red cedar shavings. All were fed the NIH open-formula diet. Among the female mice, those on mixed bedding material had a statistically nonsignificant decreased incidence of mammary tumors relative to those on pine sawdust alone (81.8% vs. 86.5%). There was also a nonsignificantly higher average age at mammary tumor appearance in the former group (17.0 vs. 16.0 months). The author concluded that these data were in agreement with his previously reported data (Heston and Vlahakis 1968, Vlahakis et al. 1970) and that the high incidence of tumors in this strain could not be attributed to the cedar shavings in the bedding material. He reiterated Heston's (1975) argument that the reduced tumor incidence reported by Sabine et al. (1973) was due to lower body weight and ectoparasitic infestation.

Two additional studies of the effects of wood bedding material have been reported. Jacobs and Dieter (1978) examined the incidence rate of spontaneous hepatomas in mice inbred for 21-25 generations from noninbred Ha:ICR Swiss stock. They reported no significant difference in hepatoma incidence in male mice raised on pine bedding (31%) versus those raised on a pine-cedar shaving mixture (25%), and concluded that cedar shavings did not contribute to the high incidence of hepatomas in these mice.

DePass et al. (1986) compared the survival, incidence, and latency of dermal neoplasms caused by the carcinogen benzo(a)pyrene (NTP 1985) between male C3H/HeJ mice housed in polycarbonate cages lined with hardwood chips of maple, birch, or beech and mice housed in stainless steel wire mesh cages with no lining. Type of housing had no effect on survival. However, the time-adjusted incidence of dermal neoplasms was significantly ($p<0.001$) higher in the polycarbonate group than in the stainless steel group at all concentrations of benzo(a)pyrene (0.25%, 0.05%, and 0.01% [w/w]). The authors suggested that the increase in tumor incidence in the polycarbonate group might be due to skin contact with urinary metabolites on the bedding or to the slightly lower ambient temperature at which that group was housed. However, they considered it unlikely that the increase was due to substances in the wood-chip bedding, since the chips were made from virgin hardwood containing relatively low levels of tannins, phenols, and other aromatic resins found in higher concentrations in softwood bedding, such as pine shavings.

The fibrogenicity of several wood dusts and plant fibers was studied by Bhattacharjee et al. (1979) and Bhattacharjee and Zaidi (1982). Groups of eight male guinea pigs were injected intratracheally with single saline suspensions containing either 75 mg of sheesham or mango wood dust or hemp or bagasse fibers or 20 mg of jute fiber. The animals were serially sacrificed up to 90 days post-injection and their lungs were examined. By the end of 90 days, Grade I fibrosis was observed in the lungs of the animals given mango and jute, and Grade II fibrosis was seen in those given sheesham and hemp.

McMichael et al. (1983) exposed male and female guinea pigs by inhalation to fir bark dust (average respirable dust concentration 1,143 mg/m^3) for periods of 30 min/day, 5 days/week for 24 weeks. Terminal histopathology revealed moderate to severe lung changes in all the animals, including increased septal connective tissue components and lymphocyte aggregation. Although no pulmonary fibrosis or extensive destruction of the parenchymal tissue was seen following the 24-week exposure period, the authors concluded that fir bark dust may cause inflammatory changes in the lung and should be considered more than simply a nuisance dust.

Wilhelmsson et al. (1985c,1985d) and Drettner et al. (1985) described two studies in which they exposed male Syrian golden hamsters by inhalation to beech wood dust (diameter 5-15 um) both with and without concurrent subcutaneous administration of the known carcinogen diethylnitrosamine (DEN) (NTP 1985). In each study, the animals were divided into four groups (12 per group in Study I, 24 per group in Study II). Two of the groups in each study were exposed to fresh beech wood dust (mean total dust

concentration 15 mg/m^3 [range 10-20 mg/m^3] in Study I; 30 mg/m^3 [range 25-35 mg/m^3] in Study II) for 6 hr/day, 5 days/week for 36 weeks (Study I) or 40 weeks (Study II). One of these two groups was also given 1.5 mg (Study I) or 3.0 mg (Study II) of DEN once a week for the first 12 weeks. The third group in each study was given a corresponding dose of DEN only (positive control), and the fourth group had no exposures (negative control). At the conclusion of the studies, the animals were sacrificed and examined.

In Study I, no nasal or lung tumors were found in any of the groups, nor was any metaplasia found. Four of eight hamsters exposed to both wood dust and DEN, three of eight positive controls, and one of seven negative controls had squamous cell papilloma in the trachea. The reduced number of specimens was due to cannibalism. No significant differences in nonpulmonary organ pathology or weight gain and development were noted. In Study II, with greater amounts of both wood dust and DEN, all sacrificed animals exposed to DEN had nasal lesions ranging from hyperplasia and dysplasia to papillomas. In addition, about half of the DEN-exposed animals had nasal tumors (adenocarcinomas) whether they had been exposed to wood dust (11/21) or not (10/22). Papillomas of the larynx and trachea were also found in over half of this same group (11/18 and 12/19, respectively). In the group exposed to wood dust only, two animals, both of which died spontaneously, had nasal lesions. One had an unclassifiable malignant nasal tumor and the other had focal metaplasia with mild dysplasia. Animals in this group also exhibited mild inflammatory reactions in the respiratory epithelium and submucosal stroma that were not seen in the negative controls. Because cannibalism occurred in both studies, and the affected animals generally had obstructive tumors in the upper airways, the studies had to be terminated earlier than planned. Although the studies were limited in nature and the group sizes inadequate, the authors concluded that wood dust exposure did not increase the tumor incidence of DEN-treated animals, but that it did affect the respiratory mucosa of Syrian golden hamsters.

B. In Vitro Studies

Evans and Nicholls (1974) examined the effects of aqueous extracts of Western red cedar on pig and human lung cell cultures. They found histamine release in both comparable to that of an equivalent amount of cotton dust, and a concentration-response relationship over a narrow range of cedar dust concentrations (5-10 mg/mL). The authors concluded that wood dust may induce the release of histamine in the lungs and upper respiratory tract, and that this might be a significant factor in the development of respiratory symptoms in exposed workers.

As part of their previously described studies, Bhattacharjee et al. (1979) and Bhattacharjee and Zaidi (1982) examined the hemolytic activity and in vitro cytotoxicity of wood dusts (sheesham and mango) and fibers (hemp, bagasse, and jute). They reported that sheesham dust was strongly hemolytic to sheep erythrocytes while mango dust was only weakly hemolytic. Bagasse fibers were strongly hemolytic to rabbit erythrocytes, while hemp and jute fibers were weakly hemolytic. The uptake of erythrosin B by guinea pig

alveolar macrophages exposed to sheesham and mango was significantly ($p<0.005$) less than the uptake by macrophages exposed to chrysotile asbestos, indicating a significantly lesser degree of toxicity for the wood dusts. The uptake of erythrosin B for hemp, bagasse, and jute fibers was equal to or greater than that for chrysotile asbestos. The authors suggested that hemolytic activity may be indicative of the acute response of pulmonary tissues while macrophage cytotoxicity may be indicative of chronic effects (e.g., fibrogenicity).

Liu et al. (1985) examined the in vitro cytotoxicity of wood dusts from three small woodworking establishments in Hong Kong: a plywood and hardwood sawmill, a furniture factory where polish dust was released from belt sanders, and the mixing room of a factory that manufactured sawdust-containing mosquito repellant. Wood species were not identified. The authors sampled for total dust, particle shape and size distributions, and metal concentrations (Cd, Cr, Cu, Fe, Mn, Ni, Pb, and Zn). They found that the percent viability of cultured female albino rat alveolar macrophages incubated with each of the three different dusts decreased with time to 57% (sawmill dust), 38% (sander dust), and 47% (mosquito repellant factory dust) after 24 hr. These levels were significantly ($p<0.05$) different from each other and from the control. Since the dust from the sanding area of the the furniture factory had the highest level of most metals, the authors attributed the difference in macrophage viability to the different metal contents of the dusts.

McGregor (1982) studied the in vitro mutagenicity of ash, oak, beech, elm, and mahogany. Water, water/methanol, and ethyl acetate extracts of sawdusts from these woods were tested using Salmonella typhimurium strains TA98 and TA100, with and without metabolic activation. None of the aqueous extracts was mutagenic. Water/methanol extracts of beech and ash were weakly mutagenic (strains/activation not specified), while elm was found to be toxic. Of the ethyl acetate extracts, beech and ash gave a positive mutagenic response. The former was weakly mutagenic in strain TA98 with activation at low concentrations; at higher levels no mutagenic activity was seen, although there were no obvious signs of toxicity. The ash extract was weakly positive at high concentrations in strain TA100 both with and without activation.

Mohtashamipur et al. (1986) also examined the mutagenicity of beech. Methanol extracts of beech sawdust were fractionated with silica gel using as sequential solvents n-heptane, petrolether, ethyl acetate, methylene chloride, and methanol. Each fraction was tested using Salmonella typhimurium strain TA100 with and without metabolic activation. Concentration-dependent mutagenic activity was observed only with activation from the beech dust extracts after fractionation. The mutagens were found to be concentrated primarily in the ethyl acetate phase and were toxic to the bacteria when tested in a histidine-rich (complete) medium. A nonsignificant increase in the number of revertant colonies was found with the methanol extract (prior to fractionation) at the nontoxic concentration of 2.5 grams equivalent dust per plate. Further tests showed that this methanol extract also inhibited the activity of known mutagens. The authors suggested that the weak activity exhibited by this extract could indicate

the presence of some mutagens in beech that are trapped among other compounds that inhibit mutagenicity.

Specific chemical substances found in wood have also been tested for in vitro mutagenicity. A description of these studies can be found in Tinkler (1986) and Niemeier (1979).

C. Summary

There are relatively little in vitro and in vivo animal data from which to draw firm conclusions regarding the toxicity of wood dust. Certain extracts of beech and ash have been shown to be weak in vitro mutagens. In some studies, the use of cedar bedding material has been associated with an increased incidence of mammary and liver tumors in susceptible strains of mice, although other similar studies have shown no such effect. Only three in vivo inhalation studies were found. In the first, high concentrations of cedar and pine dusts caused lung abscesses and bronchopneumonia in rabbits following chronic inhalation. In the second, high concentrations of fir bark dust caused inflammatory changes in guinea pig lungs following subchronic inhalation. In the third, beech wood dust was administered by inhalation to hamsters concurrently with a known carcinogen. At the concentrations used, no synergistic carcinogenic effects due to wood dust were seen, although there were effects to the respiratory mucosa.

III. DERMATITIS

Irritation of the skin and eyes resulting from contact with woods or their dusts is relatively common (Senear 1933) and may result from mechanical action (e.g., irritation caused by bristles and splinters), chemical irritation, sensitization (allergic reaction), or by a combination of these. Woods and Calnan (1976) have published a comprehensive review of wood dermatitis, from which much of the following introductory discussion is taken; no attempt will be made to duplicate their compilation of data for over 300 species of trees. In most reports of contact dermatitis, hardwoods of tropical origin have been implicated as the responsible agents; however, a few softwoods and hardwoods from temperate climates, including pine, spruce, Western red cedar, yew, elm, and alder, have also been cited as causal agents. Exotic woods, such as those found in the tropics, generally cause more toxic systemic effects and contain more potent sensitizers. A list of wood species that have been identified as causes of dermatitis is shown in Table 2.

Irritant chemicals are often found in the sap or latex, or in the bark or cracks in the wood. Since these substances exist primarily in the outer part of the tree, loggers and those involved in initial wood processing are most affected. Primary irritant dermatitis caused by wood contact is clinically similar to any irritant dermatitis, consisting of erythema and blistering, which may be accompanied by erosions and secondary infections. Also, the conjunctivas are often affected (Nava 1974; Kubena et al. 1968).

Chemicals causing sensitization are generally found in the heartwood and, therefore, more often affect workers involved in secondary wood processing, such as sawyers, carpenters, joiners, and furniture finishers and polishers exposed to wood dust. The early stages of allergic dermatitis (dermatitis venenata) are redness, scaling, and itching, initially resembling irritant dermatitis. The hands and forearms, eyelids, face and neck, and genitals are generally first affected. Mild cases may exhibit only redness and irritation, which may progress to vesicular dermatitis, and, after repeated exposures, to chronic dermatitis. Allergic dermatitis may appear after several weeks to several years of contact, but in general the eruptions appear after a few days to a few weeks of contact. High humidity, repeated wetting of the skin, and sweating may increase the intensity of the reaction (Suskind 1967, Steiner and Schwartz 1944). Development of tolerance to some woods has been reported (Schwartz 1931).

The sensitizing agents responsible for dermatitis are believed to be chemicals endogenous to the woods. Among agents that have been identified are anthothecol from African mahogany (Morgan et al. 1968), (R)-3,4-dimethoxydalbergione and its quinol from Machaerium scleroxylon (Morgan et al. 1968), and mono- and dimethoxy-substituted quinones or

Table 2.--Woods causing dermatitis*

Botanical name	Common name**
Apocynaceae	
Aspidosperma spp.	Peroba rosa
Betulaceae	
Alnus spp.***	Alder
Bignoniaceae	
Paratecoma peroba	Peroba de Campos, peroba branca, peroba amarella
Tabebuia spp.	Lapacho, suayacan, ipe, mayflower
Burseraceae	
Aucoumea klaineana	Gaboon, okoume
Hernandiaceae	
Hernandia sonora	Topolite
Lauraceae	
Nectandra rodiaei	Antilles greenheart
Phoebe porosa	Brazilian walnut, imbuia
Ocotea spp.	Louro, jigua, pisie
Leguminosae	
Andira inermis	Partridge wood
Brya ebenus	Cocus wood
Cassia siamea	Tagayasan
Dalbergia cearensis	Kingswood, violet wood
Dalbergia granadillo	Costa Rica rosewood
Dalbergia latifolia	East Indian rosewood, Bombay blackwood, sissoo
Dalbergia melanoxylon	African blackwood

(Continued)

See footnotes at end of table.

Table 2 (Continued).--Woods causing dermatitis*

Botanical name	Common name**
Leguminosae (continued)	
Dalbergia nigra	Brazilian rosewood, rio-palisander, grenadilla
Dalbergia retusa	Cocobolo
Dalbergia stevensonii	Honduras rosewood
Distemonanthus benthamianus	Ayan, Nigerian satinwood
Gossweilerodendron balsamiferum	Agba, tola branca
*Prosopis juliflora****	Mesquite
Machaerium scleroxylon	African mahogany
Meliaceae	
Guarea thompsonii	Guarea
Khaya anthotheca	African mahogany
Moraceae	
Chlorophora excelsa	African iroko, kambala, African teak
Pinaceae	
Abies spp.***	Fir
*Juniperus virginiana****	Eastern red cedar
*Libocedrus decurrens****	Incense cedar
Pinus spp.***	Pine
Picea spp.***	Spruce
*Pseudotsuga menziesii****	Douglas fir
*Thuja occidentalis****	Northern white cedar

(Continued)

See footnotes at end of table.

Table 2 (Continued).--Woods causing dermatitis*

Botanical name	Common name**
Pinaceae (continued)	
Thuja plicata***	Western red cedar
Tsuga spp.***	Hemlock
Rutaceae	
Chloroxylon swietenia	East Indian satinwood
Fagara flava (=Zanthoxylum) flavum	West Indian satinwood
Fagara macrophylla	Olon
Sapotaceae	
Bassia latifolia	Moah, mahwa
Mimusops heckelii	Makore, baku
Taxaceae	
Taxus baccata	Yew
Triplochitonaceae	
Mansonia altissima	Mansonia, African black walnut
Ulmaceae	
Ulmus spp.***	Elm
Verbenaceae	
Tectona grandis	Teak

*Adapted from McCord (1958), Fregert and Hjorth (1968), and Gamble (1979).

**There is no accepted international nomenclature.

***North American species.

quinols from cocobolo (Schulz and Dietrichs 1962), iroko (King and Grundon 1949,1950), teak (Vermeer et al. 1949, Schulz 1962), Rio-palisander (Schulz and Dietrichs 1962), and mansonia (Schulz 1962). Additional substances are described in Woods and Calnan (1976) and Gamble (1979).

The earliest modern report of wood dermatitis appears to be that of Stern (1891), who described two flute players who developed a contact dermatitis of the lips from the mouthpieces of their instruments, which were made of cocobolo wood. Jones (1904) described dermatitis among shipyard workers who handled East Indian satinwood. Cash (1911) demonstrated, by patch testing, that an alkaloid isolated from satinwood by Auld (1909) produced dermatitis; the oils from satinwood produced a less dramatic reaction. Matthes and Schreiber (1914) extracted components from six types of wood (East Indian moah, teak, Australian moah, lapacho, tecoma, and greenhart) and tested them for skin irritation. Hoffmann (1928), Senear (1933), Bergquist and Rundberg (1941), and Woods and Calnan (1976) have summarized many similar reports involving a wide variety of wood species, mostly foreign woods. Weber (1953) estimated that 1% of workers develop skin sensitization to sawdust of native American woods. Selected case reports of wood dermatitis are summarized in Table 3.

Another type of dermatitis associated with the wood industry but not caused by contact with wood or wood dust itself is "woodcutters' eczema," also known as "wood poisoning," "cedar poisoning," "pine poisoning," "oak poisoning," "spruce poisoning," and "spruce dermatitis." This condition, which has been described by a number of authors (Mitchell 1965; Mitchell and Armitage 1965; Mitchell et al. 1969; Champion 1965,1971; Suskind 1967; Mitchell and Chan-Yeung 1974), is prevalent among forest workers and is caused by contact with epiphytes, lichens, and liverworts growing on bark or surrounding shrubs. This dermatitis occurs only during work in forest areas, appears within a day or two after starting work, becomes worse in wet weather, and subsides in 2 to 4 weeks after exposure stops. The respiratory system may also be affected.

Fungi growing on bark were first suspected of causing dermatitis in 1921, but it was a quarter of a century before woodcutters' eczema was first attributed to lichens and liverworts (Gamble 1979). One of the most common causes of wood poisoning is the leafy liverwort Frullania, which thrives in high humidity and grows on trunks and branches of trees and shrubs, and on cliffs and rocks. Sesquiterpene lactones have been identified as sensitizers in Frullania (Mitchell and Chan-Yeung 1974). Similarly, usnic acid, a compound related to the photosensitizing furocoumarans, has been identified as the sensitizer in lichens (Mitchell 1965).

Summary

A wide variety of both domestic and foreign wood species have been documented in case reports and epidemiological studies to cause irritant and allergic dermatitis. For some species, chemicals present in the wood, including quinones and quinols, have been isolated and shown to be the sensitizing agent. Another wood-related dermatitis is woodcutters' eczema, which is caused by lichens and liverworts that grow on bark and shrubs.

Table 3.--Selected case reports of wood dermatitis

Reference	Wood types	Comments
Schwartz (1931)	Brazilian walnut	Eleven cases of dermatitis among 100 workers of a cabinetmaking plant were reported. Initial symptoms included burning and itching of the face and eyelids, which spread to the hands, forearms, neck, and other exposed portions of the body. The condition developed 2 days to 2 weeks after the workers began handling this wood. Most of the workers recovered after several weeks, even with continuing exposure. The author contacted other firms that had bought this wood from the same supplier and found that 9 of 10 who replied had experienced dermatitis in their workers. Walnut sawdust patch tests on three volunteers were positive.
Levin (1933)	Unspecified	A case of severe dermatitis of the hands and arms, with itchy eruptions, blisters, and swelling, was reported in a department store cleaner who threw handfuls of moistened sawdust on the floor for sweeping. The female cleaner also reacted to the sawdust in patch testing, and dermatitis recurred whenever attempts were made to resume use of the sawdust.
Stewart (1940)	Mesquite	A male patient developed facial eruptions one day after handling mesquite wood. Patch tests with mesquite sawdust were positive, and no recurrence was noted after the patient stopped using the wood.
Davidson (1941)	African iroko	Dermatitis in more than 50 men working with moist logs in a woodworking shop was described. Nine had severe symptoms (some within an hour of initial contact), including irritation of covered skin, facial edema and eye irritation, headache, pharyngitis, and chest symptoms. Two of these nine had experienced earlier cases of dermatitis attributed to exposure to teak.

(Continued)

Table 3 (Continued).--Selected case reports of wood dermatitis

Reference	Wood types	Comments
Levin (1941); Leider and Schwartzfeld (1950)	Cocobolo	Three cases of allergic dermatitis in women were described. Two had developed itching and characteristic eruptions of the hands, which in one had spread to the face and neck. In both cases, sensitization was traced to a cocobolo knife handle and was confirmed with patch testing. The third patient was a musician who experienced swelling of her lips after she had been playing a wind instrument for 1 month. A patch test for cocobolo was positive, and her symptoms disappeared when she ceased playing the instrument.
Steiner and Schwartz (1944)	Tobasco mahogany	An outbreak of occupational dermatitis among 40 workers in a boatyard woodshop was described. The outbreak occurred when "Tabasco mahogany," principally from Brazil and Honduras, was substituted for "Philippine mahogany," described as a trade name for several types of Phillipine hardwood, none of which actually belongs to the mahogany family. The peak of the outbreak occurred during a hot part of the summer, when 13 workers who wore little clothing and perspired freely were treated for dermatitis. Several of the workers patch tested positively either to the wood itself or to ether extracts.
Vermeer et al. (1949)	Peroba teak	Four cases of occupationally related eczema were reported. Patch tests with this wood dust were positive in all four. The responsible allergen was tentatively identified as lapachonon, a derivative of naphthalene.
Weber (1953)	Poplar	A case of allergic dermatitis in a woodworker who had also suffered weight loss, lymph node enlargement, itching, chills, and sleeplessness was described. Patch tests were positive for poplar. After recovering, the patient returned to work in another part of the plant and symptoms did not recur.

(Continued)

Table 3 (Continued).--Selected case reports of wood dermatitis

Reference	Wood types	Comments
Bourne (1956)	Mansonia	Six cases of dermatitis among 12 sanders in a radio and television cabinet manufacturing plant were described. Initial symptoms were nasal irritation and sneezing, followed by scalp, facial, neck, and shoulder irritation, nasal sores and discharges, and sore throat. In response to patch tests, all six workers developed irritation within 8-12 hr and persisted for as long as 4 days. Symptoms disappeared when use of this wood was stopped.
Morgan and Wilkinson (1965); Morgan et al. (1968)	African mahogany, Machaerium scleroxylon	Several cases of dermatitis at three furniture factories were reported. Six workers from two factories were patch tested with dust from two species of African mahogany (Khaya anthotheca and Khaya ivorensis) and with anthothecol, a constituent of mahogany. Five of the six were positive for K. anthotheca and the sixth was positive for K. ivorensis; two of the six were positive for anthothecol. At the third factory, the first symptoms of dermatitis occurred after M. scleroxylon was substituted for Rio rosewood. Within 3 weeks, 7 out of 24 workers had developed dermatitis. Patients who were patch tested with Rio rosewood had moderate reactions, whereas all of those tested with M. scleroxylon had very strong reactions. Four major compounds were extracted from M. scleroxylon: dalbergin, methyl dalbergin, and (R)-3,4-dimethyloxydalbergione and its quinol derivative. Patch testing with these four compounds and the completely extracted sawdust showed that the activity was caused by the (R)-3,4-dimethyloxydalbergione and its quinol. The authors noted that these two compounds were very similar in structure to the sensitizing constituents of Rio rosewood and cocobolo wood, which are also quinones and quinols.

(Continued)

Table 3 (Continued).--Selected case reports of wood dermatitis

Reference	Wood types	Comments
Findley (1972)	East Indian rosewood	A male patient who had developed a rash on his feet, neck, and trunk over a 6-week period was described. Symptoms were traced to rosewood sawdust vented into the air by an adjacent cutlery handle manufacturer. Patch tests were positive. Of twenty other men working in the area who were examined, only one had a positive patch test.
Bleumink et al. (1973)	Western red cedar	A case of allergic dermatitis of the face, hands, and arms of a sawyer who had been exposed for 2 years was described. Patch tests showed strong delayed cutaneous reaction to cedar extracts as well as to two specific chemical components of the wood, gamma-thujaplicin and 7-hydroxy-4-isopropyltropolone.
Burry (1976)	Radiata pine	Contact dermatitis in nine patients who worked with pine sawdust was described. Patch tests with the sawdust or with substances containing colophony (pine rosin) were positive in all nine patients.
Fowler (1985)	Honduras mahogany	A case of allergic contact dermatitis in a male patient who had been employed as a woodworker for 20 years with no previous skin disease was described. Six months after changing jobs from a well-ventilated to a very dusty work site, he developed a diffuse dermatitis of the hands, lower arms, and face, neck, and upper chest. Patch testing yielded positive results for Honduras mahogany dust. After leaving this job, the individual had no recurrence of dermatitis.

IV. ALLERGIC RESPIRATORY EFFECTS

An allergic respiratory response, like allergic dermatitis, is an immunologically mediated reaction to environmental antigens (allergens) that results in symptoms on exposure to the allergen (Terr 1978). The most commonly reported allergic respiratory effect due to exposure to wood dust is asthma. It may occur alone or in conjunction with dermatitis and may manifest itself as an immediate (within minutes) or delayed (within hours) reaction, or as a combination of the two separated by a recovery period. Case reports have documented asthmatic reactions to several common woods, including oak, mahogany, and redwood, as well as to more exotic woods such as iroko, cocobolo, zebrawood, and abiruana. These reports are summarized in Table 4. The only epidemiologic studies of wood dust-induced asthma are those on Western red cedar, which is discussed in detail later in this section. Reports of respiratory effects not specifically identified as allergic responses are discussed in Section V.

Some reports refer to the observed asthmatic responses of individuals exposed to wood dust as Type I allergic reactions, as originally defined by Gell and Coombs (1963). The characteristics of Type I reactions are shown in Table 5. In this type of reaction, allergen-specific immunoglobulin E (IgE) antibodies are produced within plasma cells embedded in the mucosa. These antibodies diffuse into tissue fluid and attach to mast cells. The reaction of allergen with this cell-bound antibody triggers the release of histamine and other substances that produce typical allergic symptoms of broncho-constriction, vasodilation, smooth muscle contraction, and increased bronchial and nasal secretion.

This definition of asthma as a Type I allergic reaction is a narrow one. Currently, asthma is more universally defined as "reversible obstructive airway disease" (Farr 1987). According to Farr (1987), only a fraction of asthmatic attacks are precipitated solely by IgE reactions. This observation is reflected in the literature on wood-dust-induced asthma. For example, case reports have demonstrated the presence of IgE antibodies in the serum of individuals with asthmatic reactions to African zebrawood (Bush et al. 1978), Quillaja bark (Raghuprasad et al. 1980), and African maple (Hinojosa et al. 1984). However, other reports have failed to show IgE antibodies in persons with asthmatic reactions to Tanganyika aningre (Paggiaro et al. 1981) and Central American walnut (Bush and Clayton 1983).

A similar pattern exists regarding the presence of precipitins (precipitating antibodies) in the serum of individuals following a challenge with wood dusts or extracts. Precipitins have been detected after a challenge with extracts of oak, mahogany, and cedar (Sosman et al. 1969) and iroko (Pickering et al. 1972), but have not been detected following

Table 4.--Selected case reports of allergic respiratory effects caused by wood dust

Reference	Wood types	Comments
Gade (1921)	Unspecified	Respiratory symptoms, including 7 cases of asthma, were reported in 14 of 20 sawmill workers. Asthmatic symptoms first appeared 7 months to 4 years after the men began work at the sawmill. Eosinophilia was present in three of the asthmatic cases.
Bahn (1928)	Pine, fir	A 33-year-old male patient who developed asthma 10 months after beginning work in a sawmill was described. Intracutaneous administration of aqueous wood extracts provoked strong reaction to pine and fir but only slight reaction to oak and beech. Since controls did not react to the pine and fir extracts, Bahn described the worker's condition as allergic and noted that the asthmatic attacks ceased when the patient stopped working at the sawmill.
Markin (1930)	Boxwood	A 34-year-old male patient who had developed asthma over a 5-year period was described. Skin scratch tests were positive both for sawdust used by patient at work and for boxwood. Although desensitization was not successful, the patient developed a gradual tolerance to boxwood.
Ordman (1949a, 1949b)	Kejaat, Congo hardwood, Western red cedar	Two cases of occupational asthma were described. The first was a 29-year-old male cabinetmaker who developed rhinitis and asthma 2 months after the plant he worked in switched to using the listed woods. Skin scratch and intradermal tests using aqueous extracts of each of the woods were positive. The second case was a 39-year-old male whose symptoms began on the first day he handled kejaat. He had previously worked with mahogany, walnut, teak, and pine with no reaction. Skin scratch test was negative; intradermal test was positive to an aqueous extract but negative to the dust itself. In both cases injections for desensitization were successful in alleviating the symptoms.

(Continued)

Table 4 (Continued).--Selected case reports of allergic respiratory effects caused by wood dust

Reference	Wood types	Comments
Sosman et al. (1969)	Oak, mahogany, cedar	Asthmatic symptoms in four woodworkers aged 33-54 were reported. Each had been exposed to wood dust of one of the listed types for 17-25 years; all had experienced sneezing, running nose, cough, wheezing, and difficulty in breathing for 4-8 years. They demonstrated either immediate, delayed, or combined responses to inhalation of the wood dusts and their aqueous or alcohol extracts. Both skin scratch and intradermal tests were negative in all cases. Precipitating antibodies against either the alcohol extract of oak or the aqueous extracts of mahogany or cedar were found in the serum of each individual. The authors suggested that the responses were hypersensitivity reactions.
Greenberg (1972)	Cedar of Lebanon	Symptoms of asthma and rhinitis were described in six joinery-shop workers. Symptoms developed rapidly after initial exposure (within 2 to 96 hr) and included chest tightness, cough, occasional sneezing, and nasal obstruction. No gross deficits were found in pulmonary function tests, nor were precipitating antibodies found. Only one worker had a positive skin patch test.
Pickering et al. (1972)	Iroko, Western red cedar	Two male patients in their mid-thirties, each of whom had developed asthmatic symptoms following exposure to one of the listed woods, were described. Bronchial challenge with either iroko or cedar dust provoked late asthmatic reaction in each. The man who worked with iroko had an immediate reaction to aqueous extracts of the wood. He also had a positive skin scratch test, and his serum contained anti-iroko precipitins. The patient exposed to cedar had no serum precipitins and gave no skin scratch test response.

(Continued)

Table 4 (Continued).--Selected case reports of allergic respiratory effects caused by wood dust

Reference	Wood types	Comments
Eaton (1973)	Cocobolo	Asthma and nasal irritation were reported in three workers who handled exotic hardwoods in a billiard cue manufacturing factory. One subject had a positive skin patch test and the other two had positive skin prick tests with aqueous extracts. Two patients exhibited positive response to bronchial provocation with wood dust, although no quantitative measurements were reported. No immunologic testing was done. Treatments for desensitization with extracts of cocobolo in one patient and of a cocobolo-padauk mixture in the other two produced a reduction in skin reactions and appeared to bring about clinical improvement. The author suggested that dalbergiones were the allergenic factor.
Booth et al. (1976)	Abiruana	Pulmonary hypersensitivity was reported in two workers, both of whom experienced cough, wheezing, and difficulty in breathing when working with this wood, and one of whom had similar symptoms at night. Upon bronchial challenge with an aqueous extract of abiruana, one worker had both immediate and late responses while the second worker had only an immediate response. Both workers had positive intradermal tests with abiruana dust but did not exhibit serum precipitins against aqueous or alcohol extracts. Authors suggested that the reactions were mediated primarily by immunoglobulin E (IgE).

(Continued)

Table 4 (Continued).--Selected case reports of allergic respiratory effects caused by wood dust

Reference	Wood types	Comments
Chan-Yeung and Abboud (1976)	California redwood	Two asthmatic male patients who developed specific immediate and late asthmatic reactions following bronchial challenge with redwood dust were described. Neither had skin reactions or precipitating antibodies against this wood. The authors suggested that low molecular weight compounds found in California redwood extracts, such as hydroxysugiresinol, sugiresinol, and isosequiric acid, may have been responsible for the respiratory symptoms.
doPico (1978)	California redwood	A male patient whose sensitivity was confirmed by a late asthmatic reaction to bronchial challenge of redwood sawdust was described. Skin prick tests were negative and precipitating antibodies were not found.
Bush et al. (1978)	African zebrawood	Asthmatic attacks in a 26-year-old male were reported. He reacted positively to intradermal testing with an aqueous extract of zebrawood dust, to which two atopic and two normal controls showed no response. In inhalation tests with this extract, both immediate and late bronchial responses were noted. The presence of IgE antibodies against the zebrawood extract was demonstrated by the direct radioallergosorbent (RAST) test and the RAST inhibition procedure.
Girard et al. (1980)	Mahogany, teak, okoume, plywood	Asthmatic symptoms were described in four Swiss sanders. Skin patch testing was positive in each, and each exhibited serum antibodies and reduced ventilatory function to bronchial provocation. The authors also reported that in a survey of 96 Swiss woodworkers (mean age 37 years, range 17-69 years; mean exposure duration 11.5 years), 56% had rhinitis, 17% conjunctivitis, 9% dermatitis, and 2% asthma.

(Continued)

Table 4 (Continued).--Selected case reports of allergic respiratory effects caused by wood dust

Reference	Wood types	Comments
Raghuprasad et al. (1980)	Quillaja bark	A case of asthma and rhinitis in a 24-year-old male was described. Allergic reaction was confirmed by positive methacholine challenge and immediate reaction to bronchial challenge with Quillaja bark dust. Presence of IgE antibodies was demonstrated by RAST.
Innocenti and Angotzi (1980)	Sambe	A case of occupational asthma was described in a 57-year-old male. Patient exhibited immediate response to bronchial challenge.
Paggiaro et al. (1981)	Tanganyika aningre	Three cases of occupational asthma in woodworkers were described. Intradermal skin tests in each were positive. Bronchial provocation with the wood dust and aqueous extracts caused immediate response in two of the patients; these two also exhibited bronchial hyperreactivity. No serum precipitins or specific IgE was found in any of the patients.
Bush and Clayton (1983)	Central American walnut	A case of asthma and rhinitis was reported in a 48-year-old male who exhibited immediate and late responses to bronchial challenge with wood dust extract. Intradermal skin testing with walnut wood extract was negative. Specific IgE antibodies and precipitins in the serum were not detected.

(Continued)

Table 4 (Continued).--Selected case reports of allergic respiratory effects caused by wood dust

Reference	Wood types	Comments
Hinojosa et al. (1984)	African maple	Two male patients who developed symptoms of sneezing, rhinorrhea, nasal itching, and shortness of breath several months after beginning exposure to African maple wood dust were described. Type I hypersensitivity was demonstrated by positive skin test reactivity, positive IgE antibodies, and immediate bronchial provocation test response to aqueous extract. No precipitins to aqueous extracts were found in the patients' blood.
Malo et al. (1986)	Spruce, fir, pine	Occupational asthma was reported in 11 male sawmill workers aged 21 to 57 who were exposed to spruce, fir, and pine. Duration of exposure and symptomatology ranged from 1.5 to 40 years and 0.5 to 10 years, respectively. Diagnosis was confirmed by significant changes in serial peak expiratory flow rates as compared to off work and significant changes in bronchial responsiveness to histamine at work as compared to off work. Seven of the workers exhibited immediate skin reactivity to mixed tree pollens. Bronchial provocation tests with sawdust were negative in four of the workers.
Cartier et al. (1986)	Eastern white cedar	A 39-year-old female who developed symptoms of occupational asthma a few weeks after starting work in a shingle-manufacturing sawmill was described. Bronchial challenge with eastern white cedar, Western red cedar, and plicatic acid each caused an isolated late asthmatic reaction. Elevated specific IgE levels to plicatic acid were found. Cross reactivity between Western red cedar and Eastern white cedar was demonstrated. The authors estimated that the latter has half the plicatic acid of the former.

Table 5.--Comparison of Type I and Type III allergic responses*

	Type I (Allergic asthma)	Type III (Extrinsic allergic alveolitis)
Predisposing factors:	Atopy	None known
Region affected:	Airways of the lungs, bronchi to terminal airways	Same as asthma (middle and terminal airways) plus alveolar and interstitial tissue
Onset of symptoms:	Immediate and/or late	Usually after 4-6 hr
Systemic reaction:	None	Usual; accompanied by fever, chills, anorexia, tachycardia, tachypnea
Serological findings:	Raised IgE	IgE normal; IgG precipitins may be present
Pulmonary impairment:	Increased air flow resistance (decrease in FEV_1)	Reduced FVC** and FEV_1**, reduced diffusion, decreased compliance
Eosinophilia:	Common	Transient and uncommon
Skin tests:	Immediate wheal and flare	Edematous reaction 4-6 hr; skin test not of much value because most antigens are locally irritating
Sensitivity:	Extreme sensitivity to bronchoconstrictive action of inhaled histamine and methacholine	
Mechanism:	Antigen: IgE-induced release of pharmacologic mediators from mast cells	Precipitating antigen-antibody complex and complement produce an Arthrus skin reaction but pulmonary lesions of the Arthrus type have not been observed

*From Gamble (1979).

**FVC=forced vital capacity; FEV_1=forced expiratory volume in one second.

challenges with Cedar of Lebanon (Greenberg 1972), California redwood (Chan-Yeung and Abboud 1976; doPico 1978), abiruana (Booth et al. 1976), Tanganyika aningre (Paggiaro et al. 1981), and Central American walnut (Bush and Clayton 1983).

Since antibodies are associated with the skin, the presence of specific antibodies can often be demonstrated by intradermal testing with the suspected allergen. If specific antibodies are present, there is an immediate wheal and flare reaction characteristic of cutaneous anaphylaxis. Such reactions to wood dust have been demonstrated in studies of iroko (Pickering et al. 1972), cocobolo (Eaton 1973), abiruana (Booth et al. 1976), and Tanganyika aningre (Paggiaro et al. 1981), but not in studies of oak, mahogany, and cedar (Sosman et al. 1969), California redwood (Chan-Yeung and Abboud 1976; doPico 1978), and Central American walnut (Bush and Clayton 1983). Carosso et al. (1987) reported that the prevalence of skin reactions to wood dust extracts (walnut, obeche, Douglas fir and other Abies spp., mansonia, chestnut, framire, white poplar, and oak) was significantly ($p<0.0001$) greater in a group of asthmatic woodworkers than in a group of healthy woodworkers.

Some authors have suggested that the sensitizing agents are chemicals endogenous to the wood because bronchoconstriction has been produced in sensitized workers by inhalation challenges with aqueous or alcohol extracts of various woods, including oak, mahogany, and cedar (Sosman et al. 1969); abiruana (Booth et al. 1976); iroko (Pickering et al. 1972); and African zebrawood (Bush et al. 1978).

The most frequently reported and thoroughly studied allergic reaction to wood is that caused by exposure to Western red cedar (WRC), Thuja plicata. The chemistry of WRC has been elucidated by Barton and MacDonald (1971). The first recognition of WRC-induced asthma is generally credited to Doig (1949). WRC was also one of the woods mentioned by Ordman (1949a) and Pickering et al. (1972) in their previously described reports. Other reports of WRC asthma have been published by Milne and Gandevia (1969), Gandevia and Milne (1970), Gandevia (1970), Mitchell (1970), Ishizaki et al. (1973), Mue et al. (1975a,1975b), Hamilton et al. (1979), Cockcroft et al. (1979,1984), and Blainey et al. (1981). The most systematic characterization of WRC asthma has been made by Chan-Yeung and colleagues (Chan Yeung et al. 1971; Chan-Yeung 1973; Chan-Yeung et al. 1973; Chan-Yeung 1977; Chan-Yeung et al. 1978; Ashley et al. 1978; Chan-Yeung et al. 1980; Chan-Yeung 1982; Chan-Yeung et al. 1982; Tse et al. 1982; Lam et al. 1983; Chan-Yeung et al. 1984; Vedal et al. 1986).

The composite clinical picture of WRC asthma described in these studies is as follows:

(a) Proportion of workers affected: approximately 5% of exposed individuals.

(b) Interval between first exposure and symptoms: weeks to years.

(c) Symptoms: the first noticeable signs and symptoms included eye and nasal irritation, watery nasal obstruction, excessively runny nose, rhinorrhea, sneezing, cough, wheeze, and chest tightness. Generally these symptoms were worse at night, with those from bronchi and bronchioles tending to require more exposure to WRC than those from the upper respiratory tract. Relief occurred over the weekend. With continued exposure, the signs, such as paroxysmal cough and wheezing, became progressively worse, occurring in the afternoon, and in some cases immediately upon exposure, as well as in the evening. If exposure continued, the signs and symptoms continued during weekends, so that the syndrome resembled chronic asthma, or asthma with bronchitis.

(d) Pulmonary function: Airway obstruction and hyperinflation were variable in asymptomatic workers with WRC asthma. The bronchial challenge of sensitized individuals is the only method of confirming the diagnosis, and in sensitized individuals, it results in (1) a delayed asthmatic reaction (maximum reduction in forced expiratory volume in one second [FEV_1] and forced vital capacity [FVC] in 4-8 hr lasting 24-48 hr); (2) an immediate asthmatic reaction (diminished FEV_1 and FVC within minutes of exposure, but return to preexposure values within 1 to 2 hr); or (3) a dual asthmatic reaction, a combination of the delayed and the immediate reactions. The delayed response was most frequently observed, but with continued or intensified exposure the early response was evoked in individuals who previously had had only the delayed response. Thus, the pattern of response was concentration-related, at least in the sense that signs and symptoms intensified with increasing length of exposure. Persistence of asthma after cessation of exposure remains in about one-half the individuals.

(e) Chest radiograph: Normal in asymptomatic individuals and upon bronchial challenge.

(f) Skin testing: No apparent relation between skin sensitivity and respiratory changes.

(g) Smcking and history of atopy: There was no positive association between smoking status and history of asthma. Among the cases studied there were more nonsmokers and exsmokers than active smokers, and more individuals with no prior history of asthma than of those with such a history.

(h) No precipitating IgG antibodies in the serum of sensitized individuals. Circulating IgE antibodies were present in about one-third of the individuals.

(i) Variable increases in eosinophils were seen in sensitized individuals.

Chan-Yeung et al. (1973) identified a low molecular weight (ca. 400 daltons) organic acid that makes up about 40% of the nonvolatile components of WRC as the causative agent for WRC asthma, although another study (Mue et al. 1975a) implicated protein found in WRC. Because Chan-Yeung et al. (1973)

considered the organic acid unique to WRC, they named it plicatic acid after the species name. Plicatic acid has since been isolated from and identified as the causal agent of asthma caused by eastern white cedar (Thuja occidentalis) (Cartier et al. 1986). Chan-Yeung et al. (1980) showed that plicatic acid activates complement and generates chemotactic activity in pooled normal human serum. The authors proposed that the acid could activate complement in vivo, thereby inducing an inflammatory response in the lungs. Chan-Yeung (1982) and Lam et al. (1983) have also suggested that nonspecific bronchial hyperreactivity is significant in the pathogenesis of WRC asthma, particularly in the characteristic late asthmatic reaction.

Several surveys of WRC workers have been reported. NIOSH conducted a health hazard evaluation at a wood products facility in Washington State (Edwards et al. 1978). Results were subsequently reported by Brooks et al. (1981). Seventy-four shake mill workers exposed to WRC, 58 planer mill workers exposed primarily to fir, hemlock, and alder, and 22 clerical and technical workers not exposed to wood dust were surveyed. A medical questionnaire, limited physical examination, and pulmonary function testing were administered; personal air sampling was also conducted. WRC-exposed workers exhibited a significant ($p<0.001$) decrease in FEV_1 between Monday preshift baseline and postshift tests. The planer mill and office worker groups did not exhibit a similar reduction. No significant relationship between duration of employment and FEV_1 was noted in any of the three groups. Occupational asthma (defined as an observed decline in FEV_1 of greater than 10% from the Monday preshift baseline compared to one or more subsequent tests during any of the following three days and a positive clinical history suggesting asthma) was diagnosed in 10 of 73 (13.5%) WRC workers, 3 of 58 (5.2%) planer mill workers, and none of the controls.

Chan-Yeung et al. (1978) and Ashley et al. (1978) reported on a two-part respiratory survey of WRC workers. In the first part of the survey (Chan-Yeung et al. 1978), 405 cedar-exposed workers and 252 controls exposed to wood dust other than cedar (primarily hemlock, fir, and spruce) were examined. A medical and occupational questionnaire was administered and spirometric measurements (FEV_1, FVC, and maximum expiratory flow rate) were taken. The cedar workers had a higher prevalence of respiratory signs and symptoms, including cough, phlegm, wheezing, and breathlessness, as well as more rhinitis and conjunctivitis. However, there was no difference in lung function between cedar workers and controls. Although quantitative data on smoking habits were not available, a correlation between respiratory symptoms and cigarette smoking was shown and a synergistic effect between cedar dust exposure and smoking was suggested. The controls who had previously been exposed to WRC (65 out of 252) had higher prevalence of respiratory symptoms than the other controls. In the companion report, Ashley et al. (1978) reported that the prevalence of chest symptoms in cedar workers increased with duration of exposure and that the decline in ventilatory function was greater, but in both groups smoking was a more significant determinant. A large number of cedar workers and controls noted improvement when away from work.

In a companion report to their survey, Ashley et al. (1980) reported higher serum alpha$_1$-antitrypsin (A_1-AT) concentrations in the noncedar

woodworkers than in the cedar workers, although dust concentrations to which both groups were exposed were similar. Differences were independent of smoking habits. In addition, there was a strong correlation between serum A_1-AT levels and duration of employment in the noncedar workers. The authors suggested that their results might be due to either an industry-specific factor, the characteristics of the dust, or dust concentration, but they could offer no explanation of the underlying mechanism.

Chan-Yeung et al. (1984) compared a group of 652 cedar mill workers to a group of 440 male office workers not exposed to air contaminants. A medical and occupational questionnaire was administered, and spirometric measurements, allergy skin prick tests, and methacholine inhalation challenge tests were conducted. When controlled for differences in age, race, and smoking, cedar workers had significantly ($p<0.001$) higher prevalence of chronic cough, phlegm, and dyspnea. Physician-diagnosed asthma was not significantly greater among the cedar workers, but worker-reported asthma was greater. Cedar sawmill employment was strongly associated ($p<0.001$) with lower levels of lung function as measured by FEV_1 and FVC. The effect of cedar exposure was consistently greater than the effect of current smoking. Among nonatopic workers, bronchial hyperreactivity (defined as a PC_{20} [methacholine provocation concentration causing a 20% decrease in FEV_1] of 8 mg/mL or less) was more common in cedar workers than in office workers. However, no difference between cedar and office workers was seen in the atopic individuals. Bronchial hyperreactivity increased with duration of employment in cedar workers but not in office workers. The authors calculated that the estimated odds of bronchial hyperreactivity for cedar workers increased by a factor of 1.30 for each 10 years of employment ($p=0.006$). Chan-Yeung et al.'s (1978) earlier survey did not show lower levels of lung function in cedar workers versus those exposed to other wood dusts. The authors suggested that the results of their latest survey meant that exposure to other wood dust may not be harmless or that an uncontrolled factor associated with wood mill employment may also be associated with lower levels of lung function.

Vedal et al. (1986) evaluated the association between pulmonary function and dust exposure in WRC workers. Their survey of 652 WRC sawmill workers included a respiratory and occupational questionnaire and spirometry test. Each worker was assigned an exposure level based on area and personal sampling for total dust. Total dust concentrations ranged from undetectable to 6.0 mg/m^3. Of the 334 workers assigned dust levels, only 33 were exposed to concentrations greater than 1 mg/m^3, and of those only 13 were greater than 2 mg/m^3. Work-related asthma, defined as asthmatic symptoms that improved on days off, was seen in 52 (8%) workers and was more prevalent with 10 or more years of employment (estimated odds ratio 2.1). When height, age, race, and cigarette smoking were controlled for, levels of ventilatory function were related to dust exposure but not to duration of employment. FEV_1 and FVC were significantly ($p<0.05$) lower in workers exposed to dust concentrations of 2 mg/m^3 or more, but no relationship was seen between $FEF_{25-75\%}$ or FEV_1/FVC and dust concentrations. The authors indicated that this association was based on only 13 workers and must therefore be interpreted cautiously. However, if correct, it would indicate

a pattern of restriction rather than airflow limitation. Exposures above 3.0 mg/m^3 were also associated with eye irritation (p=0.02).

Not all reactions resembling allergic responses that have been reported in woodworkers have resulted from chemicals inherently present in the wood. Allergy-like effects on the lower respiratory tract have also been attributed to the inhalation of mold and fungal spores during the processing of contaminated wood products. These effects include maple bark disease caused by inhalation of Cryptostroma corticale (Towey et al. 1932; Wenzel and Emanuel 1967), wood pulp worker's disease caused by Alternaria (Schlueter et al. 1972), sequoiosis caused by inhalation of redwood sawdust contaminated with Pullularia (Cohen et al. 1967), and suberosis caused by Penicillium frequentans, a contaminant of cork (Pimentel and Avila 1973; Avila and Lacy 1974). Similar reactions have been attributed to wood dust contaminated with Bacillus subtilis (Johnson et al. 1980) and Trichoderma and Scopulariopsis (Halprin et al. 1973). These disorders are often referred to as extrinsic allergic alveolitis (EAA) or as hypersensitivity pneumonitis because of the marked interstitial inflammatory changes that accompany them (Kaltreider 1973). Table 5 lists some of the characteristics of EAA. A review of EAA has been made by Molina (1984). The allergic responses produced by these microorganisms resemble the reactions caused by the inhalation of organic dust, such as moldy hay, in farmer's lung disease. The allergic response in farmer's lung disease is a Type III reaction in which the subject develops antibodies that react with antigens to form immune complexes, damaging vascular endothelial and other tissues either at the site of the reaction or at some remote site. Common signs and symptoms in acutely ill patients include malaise, sweating, chills, fever, loss of appetite, breathlessness, and a sense of tightness in the chest. Dyspnea and cough are the prominent signs in chronic cases where there is irreversible lung damage.

Signs and symptoms of extrinsic allergic alveolitis were described by Howie et al. (1976) in a patient exposed to ramin wood dust. The 34-year-old man was a nonsmoker who had experienced episodes of breathlessness, coughing, shivering, sweating, and fatigue for 2 years after heavy exposure to ramin dust. He was asymptomatic on weekends and holidays. His physical examination, chest x-rays, blood tests, and pulmonary function tests were normal. He showed no hypersensitivity in prick tests with common allergens or ramin dust. Precipitins to saline extracts of fresh ramin dust were found in his serum. Lung function tests showed a significant ($p<0.0025$) reduction in FEV_1 4-6 hr after he was exposed to ramin dust at high concentrations, with return to baseline values after 24 hr. The authors attributed the reduction in FEV_1 to a true impairment of gas flow, since there was no significant rise in carboxyhemoglobin levels or alterations in computed alveolar volumes. They concluded that ramin dust was capable of producing a clinical syndrome similar to that of extrinsic allergic alveolitis.

As part of their survey of Swedish furniture workers (see also Section V), Wilhelmsson et al. (1984,1985b) conducted a survey of nasal allergies among 268 workers at 6 wood furniture factories in southern Sweden. Sixteen percent of the workers (mean exposure duration 12 years) had a history

compatible with hypersensitivity. Based on skin prick and bronchial provocation tests, the authors reported that 2% of the workers were allergic to wood dust and 3% to molds found in the workplace. The most common mold was Paecilomyces spp. Although providing no quantitative data, the authors estimated that both these figures were higher than would be found in the general population.

Alexandersson et al. (1986) reported declines in FVC and FEV_1 averaging 0.3-0.4 L in Swedish wood trimmers exposed to high concentrations of molds (10^5-10^6 colony-forming units/m^3), but not in trimmers exposed to mold levels 10 to 100 times lower.

A case of acute respiratory illness attributed to inhalation of wood chip dust contaminated with toxins from unspecified microbial species has also been reported (Centers for Disease Control 1986). The illness, which affected 5 of 11 exposed workers at a municipal golf course, was classified as "organic dust toxic syndrome" (ODTS). None of the workers had positive reactions to a standard battery of wood chip and wood chip microbe antigens.

Summary

Asthmatic reactions (reversible airway obstruction) to a variety of both domestic and foreign woods have been reported. Immunological findings in individuals with wood-dust-induced asthma vary. In some cases, a Type I allergic reaction was confirmed by the presence of IgE antibodies. Positive skin reactions and the presence of precipitating antibodies to wood dust or extracts have also been reported, but not universally.

The most thoroughly studied allergic respiratory reaction to wood is WRC asthma. It is estimated that 5% of the exposed population is sensitive to this wood. Workers exposed to WRC have exhibited an increased prevalence of respiratory signs and symptoms when compared to workers exposed to no or other types of wood dust. These symptoms increased with exposure duration. Decreased ventilatory function, as measured by FVC and FEV_1, has been demonstrated in workers exposed to WRC compared to those not exposed. However, no difference was seen between workers exposed to WRC and those exposed to other wood dusts. Only a single study relating WRC dust concentrations to ventilatory function was found; in that study concentrations of 2 mg/m^3 or more were associated with significantly lower FVC and FEV_1 levels.

Another allergic respiratory effect related to wood dust is extrinsic allergic alveolitis, which is a Type III allergic reaction. This reaction is associated not with pure wood dust, but rather with exposure to wood contaminated by microbial species.

V. MUCOSAL AND NONALLERGIC RESPIRATORY EFFECTS

This section summarizes studies that have examined changes in the structure and function of the nasal mucosa and respiratory tract due to exposure to wood dust. Respiratory effects that have been identified as allergic reactions (such as asthma and hypersensitivity pneumonitis) are discussed in Section IV.

Bellion et al. (1964) reported pulmonary abnormalities among carpenters, sawmill workers, and woodworkers exposed to chestnut, teak, rosewood, mahogany, birch, and kejaat. Exposure duration ranged from 3 to 24 years. Among 225 workers examined, 97 were found to have radiologic evidences of pulmonary abnormality. The most frequent finding was an accentuation of bronchovascular markings. In 9 of 79 workers studied in some detail, the most apparent radiologic change was micronodular shadows, resembling those found in miliary tuberculosis, scattered throughout the entire pulmonary field.

Similar findings were reported by d'Agostino et al. (1965), who described a case of diffuse pulmonary fibrosis secondary to pneumoconiosis in a woman who had worked in a sawmill for 15 years. The patient complained of coughing and dyspnea, with moderate expectoration, for about 3 years. Radiographs of her lungs contained shadows indicating that small nodules were disseminated throughout the lungs and that they were accompanied by diffuse reticulation.

Michaels (1967) described pathological changes in the lungs of two deceased Canadian workers who had been exposed to wood dust for 10 and 12 years, respectively. The cause of death in both cases was unrelated to the lung conditions. Both workers had developed centrilobular fibrosis and emphysema. Michaels discovered within the alveoli of the lungs microscopic basophilic particles, thought to be grains of wood dust that had caused a foreign body and histiocytic reaction. Sections of lungs from 21 other patients who had worked in the Ontario timber industry for unknown lengths of time and at unknown concentrations of wood dust were also examined. No particles similar to those found in the lungs of the first two cases were seen.

As part of their research on nasal cancer among British woodworkers, Hadfield (1969) and Hadfield and Macbeth (1971) examined the nasal passages of several thousand workers in furniture factories. They documented various mucosal changes, including metaplasia. Because of the relationship of these changes to carcinogenicity, they are discussed in more detail in Section VI.

Ruppe (1973) examined 140 East German woodworkers (average age 36.8 years) exposed to exotic woods (species not named) for periods ranging from 4 to more than 10 years. Ruppe found an increased incidence of coughing and expectoration, colds and sneezing, sinusitis, nasal mucosal irritation, and reduced ventilatory function after acetylcholine challenge compared to a control group of 73 workers (average age 42.5 years) not exposed to wood dust. All of the above symptoms, except for the positive acetylcholine challenge, were seen even among the 14 workers exposed to wood dust concentrations below 5 mg/m^3. However, this number of subjects was too small to permit statistical analysis.

Black et al. (1974) studied nine woodworkers (average age 56 years, range 48-66) from a single factory in High Wycombe, England. Three were nonsmokers; four were light-to-moderate smokers. Mucociliary transport rates were measured by the movement of polystyrene spheres irreversibly labeled with ^{99m}technetium that had been placed at the center of the anterior end of the middle turbinate. Transport rates in 12 controls (average age 48 years, range 31-69; 8 nonsmokers, 3 moderate smokers, and 1 pipe smoker), none of whom had been exposed occupationally to wood dust, ranged from 1.9 to 18.5 mm/min, with a mean of 6.8 mm/min. Additional information on normal mucociliary transport rates were obtained from four other studies cited by Black et al. (1974). In these four studies, a total of 126 normal subjects had exhibited a mean rate of 7.8 mm/min.

Mucociliary movement in the group of nine woodworkers was severely depressed. During a 30-min observation period, three of the woodworkers had no discernible movement and four others had only slight transport of the labeled spheres. Only the remaining two woodworkers had sufficient movement to yield estimates of the transport rate; these were calculated to be 2.7 and 1.0 mm/min, respectively. Only the former rate was within the range of values measured in the controls. This worker had been exposed to wood dust for the shortest time, 6 years. The investigators concluded that exposure to wood dust in the furniture industry for 10 years or more can impair mucociliary clearance at the anterior end of the middle turbinate.

Solgaard and Andersen (1975) and Andersen et al. (1976,1977) have described a study of 68 men (average age 41 years, range 17-66) in 8 large Danish factories producing wooden furniture and fixtures. Workers engaged in varnishing, painting, etc., and those who had had colds within the preceding 2 weeks were not used in the investigation. The wood dust exposure periods ranged from 1 to 51 years, with an average of 16 years. A questionnaire on working conditions, symptoms, and smoking habits was answered by each worker. Examinations of the selected workers consisted of oral and nasal inspections, recording of FEV_1 and $FEF_{25-75\%}$ (forced expiratory flow during the middle half of the FVC), taking of a moist nasal swab, and determining the mucus transport rate using the saccharine/sky blue method. A control group of 66 healthy persons (age range 18-47 years), 19 of whom were women, was studied by the same techniques as were the woodworkers.

Wood dust concentrations were determined gravimetrically based on samples taken from the general work areas as well as from the breathing zones of the individual workers. For the latter, 43 of 68 measurements (63%) exceeded 5 mg/m^3 and 19 (28%) exceeded 10 mg/m^3. For the general work area samples, 58% exceeded 5 mg/m^3 and 23% exceeded 10 mg/m^3. Measurement of particle size yielded a maximum in the 6-10 um range. During the measurements, 41 workers were engaged in sanding by hand or by machine and 27 in sawing, planing, or drilling. The average concentrations of dust for these two groups were 14.3 and 5.2 mg/m^3, respectively. Teak was the primary wood used on the day of examination, followed by oak, chipboard, palisander, mahogany, jakaranda, beech, and pine. The most frequent complaints by the workers were dryness in the nose, irritation of the eyes, nasal obstruction, protracted colds, and frequent headaches. For comparison purposes, the workers were placed into two groups, those exposed to wood dust concentrations below 5 mg/m^3 (Group I; 25 workers) and those exposed above 5 mg/m^3 (Group II; 43 workers). Both groups had similar age distributions, length of wood dust exposure, and smoking habits. Middle-ear inflammation and common colds occurred significantly ($p<0.05$) more often in Group II. Sinusitis, prolonged colds, asthma, itching or bleeding nose, sneezing, and nasal obstruction were more prevalent, but nonsignificant, in Group II. No relationship was found between symptoms and the type of work performed, age, or length of employment. FEV_1 and $FEF_{25-75\%}$ values measured for the workers did not differ significantly from normal calculated control values, nor were there any spirometric differences between Groups I and II.

The mucociliary transport rate was significantly ($p<0.05$) lower among the 68 woodworkers than among the 66 controls. Among the latter, only 9 (14%) had times longer than 40 min (defined as mucostasis) for transport of a particle of saccharin from the superior surface of the inferior turbinate to the pharynx whereas 26 (38%) of the woodworkers had transport times greater than 40 min. The fraction of workers with mucostasis increased with increasing concentration of wood dust: 63% of those exposed to the highest mean wood dust concentration of 25.5 mg/m^3 had mucostasis whereas only 11% of those exposed to the lowest mean concentration (2.2 mg/m^3) had mucostasis. In Groups I and II, 20% and 49%, respectively, had mucostasis. Nine subjects who had mucostasis during the study were reexamined in their homes after a 48-hr weekend without exposure to wood dust. Six of these individuals then had normal mucus transport times, but three still had mucostasis. Inert plastic dust particles (25 mg/m^3) with a size distribution similar to that of the beech dust did not change the mucociliary transport rate. Andersen et al. (1977) therefore concluded that the mechanical effect of the dust was not the cause for the mucostatic effect of the wood dust.

Andersen (1986) subsequently reported on a controlled exposure chamber study in which healthy volunteers (prior wood dust exposure, if any, unspecified) were exposed to dust from pine, beech, oak, mahogany, or chipboard at a concentration of 10 mg/m^3. The size distribution ranged from 1.6 um to 14.9 um with a maximum of approximately 30% from 4.9 um to 8.0 um. No effect on mucociliary clearance was seen following a 5-hr exposure. However, based on qualitative discomfort responses made by the

exposed volunteers, wood dust was reported to be almost four times more irritating than plastic dust at the same 10 mg/m^3 exposure concentration for 4 hr. Referring to his earlier nasal clearance study (Andersen et al. 1976,1977), Andersen (1986) indicated that controlled exposure chamber studies of acute effects on healthy subjects cannot always be used to predict chronic workplace effects.

Werner (1979) reported the results of rhinolaryngologic examinations of 226 workers (103 men, mean age 37.1 years; 123 women, mean age 36.7 years) at a furniture pressboard manufacturing plant in Leipzig. Eleven percent of the subjects had worked for less than 2 years and over 75% had worked for more than 5 years. The woods used included pine, fir, beech, oak, ash, birch, alder, mahogany, mansonia, and okoume. Wood dust concentrations were reported only as being below the 1979 German MAC values, and there was concomitant exposure to formaldehyde at concentrations averaging 3 to 4 times the MAC value. Chronic inflammatory changes in the nasal mucous membranes were seen in 143 (63.3%) workers. These findings were significant (p value unspecified) when compared to a control group of 104 individuals (60 men, mean age 41.4 years; 44 women, mean age 35.5. years) not exposed to either wood dust or formaldehyde. Fifteen (6%) workers had a decreased sense of smell and five (2.2%) were anosmic. Fifteen of the 20 cases of decreased or absent sense of smell were associated with chronic inflammatory changes in the nasal mucosa. This indicated to the author that these effects were due primarily to changes in the upper respiratory tract rather than to the nervous system. Cytological and histological examination demonstrated severe metaplasia in an unspecified number of workers.

Chan-Yeung et al. (1980) conducted a respiratory survey that included medical-occupational questionnaires, limited physical examinations, spirometry, and chest x-rays of 1,932 workers in a pulp and paper mill in British Columbia, Canada. The results obtained from 1,826 white male workers were analyzed. The authors reported that workers exposed to wood dust (mean total dust concentration of 0.5 mg/m^3, range from less than 0.1 to 2.7 mg/m^3) had a slight but statistically significant ($p<0.05$) decrease in pulmonary function values (FVC and FEV_1) compared to controls after adjustments were made for differences in age, height, duration of employment, smoking habits, and alcohol intake. Because the workers were also exposed to small amounts of the chemical wood preservatives pentachlorophenol and tetrachlorophenol (maximum concentrations 0.118 mg/m^3 and 0.08 mg/m^3, respectively), the authors concluded that wood dust or the chemical preservatives were responsible for the deleterious effects seen.

Al Zuhair et al. (1981) examined ventilatory function among 124 British workers exposed to wood dust. At one of two furniture factories, where mean total dust and mean respirable dust concentrations ranged from 1.36 to 8.29 mg/m^3 and 0.19 to 0.44 mg/m^3, respectively, a statistically significant ($p<0.05$) decrease in FVC and FEV_1 was seen during the workshift. Other ventilatory measurements were too variable to discern a pattern. At the other, less dusty furniture factory (mean total dust concentrations of 0.46 to 4.16 mg/m^3) and in an inoperational power

station, no such change was observed. No concentration-response relationship was seen at any of the work sites between exposure concentrations and changes in FVC and FEV_1, although those workers exposed to the highest wood dust concentrations in the first factory tended to show the largest drop.

Whitehead et al. (1981a) presented the results of a cross-sectional survey of 1,157 American woodworkers at ten companies exposed to either hardwood (predominantly rock maple, with some ash and oak) or softwood (white pine). The survey included basic spirometric pulmonary function tests (FVC, FEV_1, and maximal mid-expiratory flow rate [MMEFR]), pulmonary symptom and smoking history questionnaire, and a work history. Approximately 100 dust measurements were taken. These were used to construct a personal relative dust exposure index by multiplying average departmental dust concentrations times the number of years the worker spent in each department. Workers were grouped by indices as follows: low (0-2 mg-years/m^3), medium (2-10 mg-years/m^3), and high (10+ mg-years/m^3). After elimination of workers in certain departments where exposure to chemicals and dusts other than wood occurred, the results from 574 workers were analyzed. To control for age, height, and sex, the authors used Knudson prediction equations. For both hardwood and softwood dusts, the authors found that exposure to higher versus lower dust concentrations was associated with statistically significant ($p<0.05$) higher odds for the occurrence of low pulmonary flow rates as measured by FEV_1/FVC and MMEFR. Concentration-response effects were demonstrated for softwoods but only suggested for hardwood exposures.

Beckman et al. (1980) examined a subgroup of 238 of the 1157 pine furniture workers studied by Whitehead et al. (1981). Beckman and coworkers determined respiratory dysfunction (defined either as decreased pulmonary function below the fifth percentile of normals or as chronic cough or phlegm from questionnaire responses). They found a correlation between dysfunction and sex, age, and smoking, but no effect was seen for years of exposure to pine wood dust. This finding contradicted Whitehead et al.'s (1981) conclusions for pine workers, which Goldsmith (1983) suggested may be due to the heterogeneous definition of pulmonary dysfunction used by Beckman and coworkers.

Goldsmith (1983) reported the results of a pilot study of 94 workers, 55 of whom had been exposed to wood dust, in a North Carolina hardwood furniture plant. The woods used were primarily oak, maple, walnut, mahogany, andiroba, poplar, and fiberboard. For a workshift, Goldsmith measured ventilatory function, both baseline performance and changes in respiratory flow. All mean work area wood dust concentrations except for carving operations were 2.0 mg/m^3 or less. Mass mean aerodynamic diameters ranged from 2.4 um to 23 um; the fraction of dust less than 10 um in diameter ranged from 30% to 82%, depending on the operation. After adjusting for age, sex, height, and smoking, Goldsmith found no relationships between either current or cumulative wood dust exposures and FEV_1, FVC, FEF_{50}, FEF_{75}, or FEV_1/FVC. However, peak flow did correlate significantly ($p=0.0345$) with cumulative person-months of exposure, suggesting that inhalation of wood dust may impair large airway

function. Changes in ventilatory function over a workshift did not correlate with either job classification or mean cross-sectional particulate concentrations. Only a difference in FEV_1 and FVC for finishing jobs suggested a weak correlation with finishing jobs that might implicate finishing fume or vapor exposure.

Lukjan and Pregowski (1984) reported finding chronic obstructive lung disease (COPD) in 72 of 804 Polish woodworkers (332 men, 472 women). Among nonsmokers, the incidence rate (4.4%) was independent of formaldehyde concentration. Among smokers, the incidence rate was 3 to 4 times higher and was also independent of formaldehyde.

Wilhelmsson and Drettner (1984) examined 753 workers from 50 furniture factories in southern Sweden. The woods used were primarily hardwoods, including birch, beech, oak, mahogany, and teak. Wood dust concentrations were measured at six of the plants. The mean concentration was 2.0 mg/m^3 (range 0.30-5.06 mg/m^3). The authors divided the 676 respondents (mean age 53 years, mean exposure duration 27 years) into two groups, those with no or only slight exposure to wood dust (192) and those with moderate or heavy exposure to wood dust (484). In the group with slight exposure, 12% had perennial hypersecretion from the nose, 30% had perennial obstruction, and 9% had more than two common colds per year. The corresponding figures for the heavily exposed group were 20%, 40%, and 21%, respectively. All three differences between the two groups were statistically significant ($p<0.05$). However, no significant difference was noted in the incidence of sinusitis.

The authors also performed the following examinations on selected subgroups of the woodworkers: rhinomanometry, air-conditioning capacity of the nose, mucociliary clearance, and spirometry. Fifty workers underwent rhinomanometry. Of these, 36 were experiencing nasal discomfort. The difference between measured resistance value without decongestant and calculated normal value with decongestant was significantly ($p<0.05$) higher in the group with nasal problems. Also, the difference in nasal mucosal swelling measured before and after application of a decongestant indicated a nonsignificant increase in the group with nasal complaints. Fifty-seven workers exposed to fine wood dust underwent air-conditioning capacity tests. Thirty-nine of these were experiencing nasal discomfort. No difference between the two groups was seen; values measured were comparable to those of a normal group of subjects. There was also no difference seen in nasal clearance between a group of 42 workers with nasal discomfort and a group of 19 without. For the previous three tests, the authors made no comparisons between wood-exposed and nonexposed workers. However, in the fourth test (spirometry), workers exposed to wood dust had a significantly ($p<0.001$) lower FVC than would be expected in a normal reference group.

Additional reports of these Swedish furniture workers (Wilhelmsson and Lundh 1984, Wilhelmsson et al. 1985a) discuss changes in the workers' nasal epithelium. Two other Scandinavian studies of the nasal mucosa of woodworkers have also been published (Drettner and Stenkvist 1979; Boysen

and Solberg 1982). Because of their significance to nasal cancer, these four studies are discussed in Section VI.

Holness et al. (1985) examined the effects of wood dust on 50 Canadian cabinet makers. Fifty hospital support staff were selected as controls. The study protocol included work history, smoking habits, and a respiratory symptoms questionnaire; environmental sampling; nasal cytology; and pulmonary function testing. Mean total dust concentrations were 1.83 mg/m^3 for the cabinet makers and 0.43 mg/m^3 for the controls. The woodworkers reported more nasal and eye symptoms and more cough, sputum, and wheezing (32%) than did the controls (10%). Although no significant differences were seen between nasal smears of the woodworkers and the controls, the former had more irritated columnar cells in their cytological smears. The woodworkers had a significant ($p=0.001$) decrease in ventilatory function (FVC and FEV_1) over the workshift. The workers also showed significantly ($p=0.005$) larger changes in FVC and FEV_1 than the controls. Thirty-one percent of the woodworkers had a greater than 5% decrease in FVC or FEV_1 over the work shift as compared to only 13% of the controls (significant at $p=0.039$). Using a cumulative dust exposure index similar to that of Whitehead et al. (1981), Holness et al. (1985) demonstrated a significant ($p=0.008$) inverse relationship between FEV_1 and FEF_{75} and this index for both total and respirable dust. FVC showed a similar but nonsignificant trend. The authors suggested that increasing exposure to wood dust may be associated with a progressive deterioration in lung function. Decreases in ventilatory function over the workshift were not related to measured dust concentrations (those with larger decreases tended to be exposed to lower concentrations). The authors suggested that this was due either to the "healthy worker effect" (where there is a selection of workers who are not sensitive to the dust) or to the development of a dust tolerance by workers with higher exposure that causes them to have a less pronounced ventilatory response. Since this was an observational rather than experimental investigation, the authors recommended additional study to clarify this question.

Innocenti et al. (1985) examined 13 male woodworkers (mean age 46.2 years; 8 smokers, 5 nonsmokers) from 2 Italian furniture factories where European chestnut was currently being used exclusively. The mean length of chestnut wood exposure was 4.8 years and of other wood dust exposure was 21 years. Twenty-four male hospital workers (mean age 44.4 years; 11 smokers, 13 nonsmokers) who had never been exposed to wood dust were used as controls. All subjects underwent rhinoscopy and an evaluation of their sense of smell. Trigeminal nerve impairment was not seen in any of the subjects; however, four workers and none of the controls were diagnosed with anosmia. This difference was significant ($p=0.0108$). There was a positive correlation between olfactory scores and previous exposure to wood dust, but not between olfactory scores and either age or previous exposure to chestnut wood dust. Two other reports of anosmia in workers chronically exposed to hardwood dusts were cited by Amoore (1986) in his review of the effects of chemical exposure on human olfaction.

Summary

Exposure to wood dust has been reported to cause both significant and nonsignificant increases in respiratory symptoms, including nasal dryness, irritation, bleeding, and obstruction; coughing, wheezing, and sneezing; sinusitis; and prolonged colds. These increases have been seen even at mean total dust concentrations below 4 mg/m^3. Significantly reduced mucociliary transport rates have also been reported in workers chronically exposed to wood dust, and in one study the reduced rates were related to increasing dust concentrations. These effects were not seen following acute inhalation exposure, nor were they seen following inhalation of high concentrations of inert plastic spheres with a size distribution similar to that of beech dust. This implies that mucostasis results from long-term exposure and is not caused simply by the mechanical action of the dust, although such irritation may be a contributing factor. Nasal mucosal changes have also been reported in woodworkers. Several studies have reported decreased ventilatory function, as measured by FVC and FEV_1, among workers exposed to wood dust, although two other studies were negative. Two studies have also shown a significant decrease in woodworkers' lung function over the workshift, although no concentration-response relationship was seen.

VI. CARCINOGENICITY

This section summarizes human case and epidemiologic reports on wood dust carcinogenicity. The section is divided into four parts, each concluded by a brief summary. Part A contains a discussion of preneoplastic nasal mucosa changes and nasal and nasopharyngeal cancer.* Pulmonary cancer is discussed in Part B, Hodgkin's disease in Part C, and other cancers in Part D. Part A is further subdivided by country in which the studies were conducted, beginning with Great Britain, where the relationship between wood dust and nasal cancer was first systematically examined. Studies conducted in the United States are discussed next, followed by those in Canada, Denmark, Sweden, Finland, Norway, Italy, France, East and West Germany, the Netherlands, Belgium, Austria, Switzerland, and Australia. Organization by country was used to present the large number of studies reported in the literature because it allows one to follow chronologically the work of individual researchers in their respective countries. It also segregates differences that might result from exposure conditions unique to a particular country (e.g., type of wood). Parts B, C, and D are not divided into subparts, although within each, studies conducted in the United States are discussed first.

The association between occupational exposure to wood dust and cancer has been reviewed in several references, which are cited herein. In 1981, the International Agency for Research on Cancer evaluated the available data and concluded the following:

> There is sufficient evidence that nasal adenocarcinomas have been caused by employment in the furniture-making industry. The excess risk occurs mainly among those exposed to wood dust. Although adenocarcinomas

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*The term "nasal cancer" refers to ICD (1980) code 160, malignant neoplasm of the nasal cavities, middle ear, and accessory sinuses. "Nasopharyngeal cancer" is ICD code 147. Included within ICD code 160 are codes 160.0 (nasal cavities); 160.1 (auditory tube, middle ear, and mastoid air cells); 160.2 (maxillary sinus); 160.3 (ethmoidal sinus); 160.4 (frontal sinus); 160.5 (sphenoidal sinus); 160.8 (other); and 160.9 (accessory sinus, unspecified). Histological classes of nasal cancer tumors include squamous cell carcinoma, adenocarcinoma, transitional cell carcinoma, esthesioneurocytoma, other carcinomas, melanoma, sarcoma (excluding lymphoma), lymphoma, and other malignant neoplasms (Redmond et al. 1982). For a discussion of the epidemiology of nasal and nasopharyngeal cancer, see Redmond et al. (1982) and Shanmugaratnam (1982), respectively. The terms used to describe the cancers discussed in this section are those used by the cited authors.

predominate, an increased risk of other nasal cancers among furniture workers is also suggested. . . .No evaluation of the risk of lung cancer is possible. . . .The epidemiological data are not sufficient to make a definitive assessment of the carcinogenic risks of employment as a carpenter or joiner. A number of studies, however, raise the possibility of an increased risk of Hodgkin's disease. There is conflicting evidence about an association between nasal adenocarcinoma and work as a carpenter. The highest level of relative risk reported is much lower than that for cabinet-makers and other woodworkers in the furniture industry, and much of the evidence is anecdotal; the possibility that the reported cases of nasal cancer had worked in these industries could not be ruled out. The evidence suggesting increased risks of lung, bladder and stomach cancer comes from large population-based occupational mortality statistical studies and is inadequate to allow an evaluation of risks for these tumors. (IARC 1981)

Wills (1982) analyzed data from 12 countries and calculated that more than three-fourths of the adenocarcinomas observed in areas where furniture making or other woodworking was a significant occupation had occurred in woodworkers. He concluded that machine sanding of hardwoods may entail special risk and that his review of the data did not contradict the conclusions of IARC (1981).

Mohtashamipur and Norpoth (1983) reviewed data from ten countries and concluded that "epidemiological findings. . .are in agreement that the woodworkers are at the most risk for [adenocarcinoma of the ethmoids and nasal cavities]."

A review published by the Health and Safety Executive of Great Britain concluded the following:

The association between adenocarcinoma of the nasal cavity and sinuses and work with hard wood has been demonstrated repeatedly. In a few studies, exposure to soft wood dust has also been associated with a cancer of the nasal cavity, but on these occasions the tumours have differed histologically. . . .[I]t is not possible to conclude whether this is due to the different nature of the softwood dust or to the presence of added wood preservatives. . . .There are no dose response data for this condition. . . .[I]t is probable that the excesses [of Hodgkin's disease] observed were not artifacts. Dose data are not available. . . .Gastro-intestinal cancer, lymphoma, leukaemia, myeloma, malignant melanoma, bladder cancer and non-melanoma skin cancer, have all been found to excess in various studies of woodworkers. The observations however, have been far from consistent and require to be confirmed. There are no dose data. . . . (Greenberg 1986)

A review prepared by the Ontario, Canada Ministry of Labour concluded the following:

Exposure to wood dusts is convincingly associated with the development of cancer of the nasal cavity and paranasal sinuses. The association has been observed in populations with exposure to either hardwoods or

softwoods and the offending agent(s) have not yet been identified. . . . There is no convincing evidence that exposure to wood dust increases the risk for any other type of cancer. No dose-response information is available for wood dust and sino-nasal cancer. (Ministry of Labour 1986)

A. Nasal and Nasopharyngeal Cancer

Great Britain--

According to Catalina (1981), the earliest report linking woodworking and cancer was published in France by Moure and Portman in 1925. However, 40 years passed before interest in the occupational significance of this association was rekindled by a series of reports from Great Britain. Macbeth (1965) cited the work of laryngologist Esme Hadfield, who had noticed that the frequency of nasal and sinus cancer among woodworkers in the Buckinghamshire furniture industry was unusually high. Of a total of 20 cases of nasal cancer from High Wycombe, the center of the Buckinghamshire furniture-making industry, 17 were males and 3 were females, but none of the females worked with wood. Among the males, 15/17 (88.2%) were woodworkers; however, the proportion of woodworkers in the local male population was only 23.5%.

Stimulated by this observation of Hadfield, Acheson et al. (1967,1968) reviewed the hospital and cancer registry records from Oxfordshire and those parts of Buckinghamshire (including High Wycombe) and Berkshire within the Oxford Regional Hospital Board. This region, which is located in south-central England, had a population of just over 1 million (as of April 1961), of which approximately 14,000 were employed as woodworkers. The initial report (Acheson et al. 1967) identified 85 cases (59 males, 26 females) of carcinoma of the nose, nasal cavities, middle ear, and accessory sinuses first diagnosed during the decade 1956-1965. Twenty-three of these cases, all males, were adenocarcinomas. Of those 23, 13 were from High Wycombe. When the data were analyzed by occupation, it was found that 17 of the 59 men were woodworkers. Thirteen of the 17 had adenocarcinomas, which was significant ($p<0.001$) compared to the 10 adenocarcinomas in the remaining 42 nonwoodworkers. The authors estimated that the crude average annual incidence rate of nasal adenocarcinoma in all types of male woodworkers in High Wycombe during 1956-1965 was 0.6 per 1,000, which was nearly the same as that for the usually much more common carcinoma of the bronchus. They concluded that the most probable explanation for the development of these adenocarcinomas was the existence of a hazardous exposure in the furniture industry in High Wycombe prior to the decade studied.

In the following year, Acheson et al. (1968) updated their preliminary findings. The study population had been expanded to include two groups, both from the Oxford Hospital Region. Group 1 consisted of those patients diagnosed between 1956 and 1965. This was the same population described in the initial report. Group 2 included patients diagnosed prior to 1956 or after 1965. Reevaluation of the hospital records had reduced the number of nasal cancer cases in Group 1 from 85 to 83 (56 males, 27 females).

However, there were still 23 adenocarcinomas, all in males. Group 2 contained 65 cases (42 males, 23 females), of which 13 (10 males, 3 females) were adenocarcinomas. In Group 1, the high male/female ratio for adenocarcinoma was significantly ($p<0.001$) different from the ratio for other tumor types. In Group 2, a similar difference was noted, but it was not significant. The age distribution of adenocarcinoma in Group 1 was also significantly ($p<0.001$) different than for other nasal cancers. Only 1 out of 23 adenocarcinomas occurred in patients over age 65, while half the other types occurred in those over 65. Similar differences were noted in Group 2, although they too were not significant. In Group 1 were 17 woodworkers, 15 of whom were in the furniture industry. Of these 17, 14 (all furniture workers) had adenocarcinoma. This association between adenocarcinoma and working in the furniture industry was significant ($p<0.001$). One nonwoodworker in the furniture industry also had adenocarcinoma. In Group 2 were seven woodworkers, five in the furniture industry. Of these seven, five (three furniture workers and two other woodworkers) had adenocarcinoma. This was not statistically significant. Nineteen of the adenocarcinomas occurred within Buckinghamshire (15 were in High Wycombe furniture workers), whereas all other types of nasal tumor were distributed fairly equally among the three "shires" in the study area. The authors recalculated an average incidence rate of adenocarcinoma among furniture workers in High Wycombe for 1956-1965 of 0.7 ± 0.2 per 1,000 per year. Among 16 patients for whom employment information was available, most had been exposed to the hardwoods beech, oak, and mahogany. The latency period between entry in the furniture industry and diagnosis of adenocarcinoma was approximately 40 years. An analysis of case histories showed that adenocarcinomas may develop in persons working in the furniture industry for only 5 years. Also, they may develop as long as 34 years after leaving the industry. The authors concluded that the hazard was present at least at the start of World War II and that it was not due to substances introduced since the war. On this basis, they eliminated exotic hardwoods, synthetic glues, stains, and varnishes as causative agents. They hypothesized that adenocarcinoma was caused by exposure to wood dust itself, and not to polish, varnish, or wood-treatment chemicals. They did not consider snuff use to be a significant factor.

In a subsequent discussion of nasal cancer among workers of the boot and shoe industry in Northamptonshire, Acheson et al. (1970) pointed out that 8 of 26 (30.8%) of these patients with adenocarcinoma had used snuff. This compares to 3 of 11 (27.3%) of the patients with nasal adenocarcinomas in the furniture-making industry of Buckinghamshire who were known to have indulged in this habit. The authors reversed their earlier judgment (Acheson et al. 1968) and suggested that the use of snuff might contribute to the development of nasal cancer.

Hadfield (1969) summarized the 83 cases of nasal cancer in the Group 1 portion of the Oxford Region study and reported on a survey of 131 workers (128 males, 3 females) in one furniture factory who had worked in the industry for more than 5 years. Although no premalignant condition of the nasal mucosa was identified, pathological changes (dry and atrophic mucosa, nasal polyps, chronic rhinitis, and chronic hypertrophic rhinitis) were

found twice as often in the group exposed to wood dust (25/73; 34%) than in the group rarely or never exposed (9/58; 15%).

In the following year, Hadfield (1970) enlarged Group 1 to include nasal cancer cases newly diagnosed during 1966-1970. The study population now included 92 cases diagnosed in the Oxford Hospital Region from 1956 to 1970. These included 28 cases of nasal adenocarcinoma in Buckinghamshire, all men, 23 of whom were from High Wycombe. Seven additional cases of adenocarcinoma were found in Oxfordshire, 3 of which were women not exposed to wood dust. Of the 32 men with adenocarcinoma, 24 were currently employed as woodworkers in the furniture industry (primarily wood machiners, cabinetmakers, and chairmakers); and 5 others had previously been similarly employed. The author calculated the crude annual incidence rate of adenocarcinoma for men in High Wycombe to be ten times greater than for men in the rest of Buckinghamshire. Because only a few of the cases of adenocarcinoma arose among men exposed to glues, stains, and varnishes rather than to wood dusts per se, Hadfield concluded that the former materials could be excluded as major contributors to the development of the disease. She also stated that the ubiquitousness of smoking among patients made it difficult to assess its significance. However, snuff use might have played a contributory role, since 3 of 17 men with adenocarcinoma for whom information was available used it. In all 35 adenocarcinoma patients, the disease originated in the ethmoid sinuses. Thirty-one of the 35 patients were 65 years or older at the time of diagnosis, and the mean latency period was 38 years. In concluding her report, Hadfield described the initiation of a survey of men employed as woodworkers in the High Wycombe furniture industry.

The results of this survey were reported in the following year (Hadfield and Macbeth 1971). The noses of 3,100 workers in furniture factories were examined during the year from mid-June 1969 to mid-June 1970. Only one malignancy was found. Deposits of dust were commonly found on the nasal septum, particularly at the anterior ends of the middle turbinates. Smears taken from the middle turbinates of the noses of 55 workers and examined microscopically were normal in 9 cases (20%) and gave evidence of squamous metaplasia in 35 cases (77.8%); 1 revealed the adenocarcinoma cited above. Ten smears were unsatisfactory and could not be used. By contrast, smears from 28 control personnel (office and maintenance personnel, and upholsterers) were normal in 20 cases (80%) and indicated squamous metaplasia in only 5 cases (20%) (3 smears were unsatisfactory). A group of 22 snuff users examined similarly yielded mid-turbinate smears indicating normal conditions in 5 (23.8%) and squamous metaplasia in 16 (76.2%) (1 smear was unsatisfactory). The authors hypothesized that squamous metaplasia, by reducing mucociliary clearance, could allow deposition and retention of fine wood dust that acted as a chronic irritant. In turn, this irritation might lead to adenocarcinoma.

Additional information on the nasal mucosa of furniture workers was provided as part of the previously described study of mucociliary clearance in woodworkers (see Section V) by Black et al. (1974). The authors took nasal smears from nine furniture workers from a single factory in High Wycombe. Only the three nonsmokers had normal mucosa. Squamous epithelial cells were

found in one smear and metaplastic cells were found in three other smears; all were from smokers or snuff users. The authors suggested that in workers with impaired clearance, certain mucosal regions would be subject to higher concentrations of soluble toxic constituents, including carcinogens. This might account for the relatively high incidence of squamous metaplasia in the nasal mucosa of woodworkers.

To examine whether the increased risk of nasal adenocarcinoma in the Buckinghamshire and Oxfordshire furniture industry existed in other parts of the country, Acheson et al. (1972) undertook a case-control study of adenocarcinoma among all the cancer registry regions in England and Wales (except for the previously studied Oxford Region). Most registries covered the period 1961-1966, although some covered a longer (unspecified) span. A total of 145 cases and 133 controls were initially identified. Controls were patients with types of nasal cancer other than adenocarcinoma. They were matched by sex, age (within 5 years), and region and year of cancer registry. Occupational histories were obtained from the patient or relative, or from a treating physician. After the elimination of records for which the histological classification of adenocarcinoma could not be confirmed or for which occupational histories were not available, 107 cases (80 males, 27 females) and 110 controls (85 males, 25 females) remained. For males, the authors found a significant ($p<0.001$) relationship between nasal adenocarcinoma and employment as a woodworker. When the woodworkers were further divided between those in the furniture industry and those not in that industry (primarily carpenters and joiners), the ratio of observed to expected cases was much greater among the furniture workers (95:1) than the latter (5:1). A significant ($p<0.001$) excess of nasal cancer other than adenocarcinomas among woodworkers was also seen, and the greater relative risk was again to furniture workers. Because of problems of classification and the small numbers involved, no statistically valid conclusions could be drawn regarding adenocarcinomas in females.

Acheson (1976) summarized the two surveys of nasal adenocarcinoma done in Great Britain during the previous decade, the Oxford Hospital Region survey (Acheson et al. 1967,1968) and the national survey (Acheson et al. 1972). The author pointed out that the former study had led to nasal adenocarcinoma being listed in 1969 as a recognized and compensable industrial disease for workers in the furniture industry in the United Kingdom. Combining the two surveys, Acheson (1976) recalculated an average latency period of 42.8 years. He noted a decline in the number of cases of adenocarcinoma diagnosed since 1970, but indicated that there had been a concomitant decline in the number of woodworkers in the Buckinghamshire furniture industry. He did not consider glues or varnishes to be causative agents, since wood dust was the only common factor in all the cases.

Five years later, Acheson et al. (1981) updated their national survey of the incidence of nasal cancer in England and Wales. A total of 1,935 cases for the period 1963-1967 were initially ascertained from information provided by the Office of Population Censuses and Surveys. Of those, 1,602 were verified as being malignant disease of the nasal cavity and accessory sinuses in residents aged 16 and older registered with the National Cancer Register of England and Wales. Occupational histories were obtained from

the patient or relative, or from the hospital or death certificate. The average annual incidence rates per million for nasal cancer of all histological types were 74.0 for men and 57.8 for women (male/female ratio 1.4:1). For men classified as woodworkers, there were 59 observed cases of nasal cancer versus 20.8 expected cases, for a significant ($p<0.01$) standardized incidence ratio (SIR) of 284. Specific occupational SIR's (all significant at $p<0.05$) were 966 (cabinetmakers and chairmakers), 616 (machinists), and 293 (other woodworkers). The SIR for carpenters and joiners (149) was not significant. The histological tumor type was available for 808 (92.3%) of the men and 339 (89.9%) of the women for whom an occupational classification could be made. There were 97 cases of nasal adenocarcinoma in men and 33 in women. There was a significant (p value not provided) excess of adenocarcinomas in male woodworkers (29 observed, 6.4 expected).

In the following year, Acheson et al. (1982) updated their data on the incidence of nasal adenocarcinoma in the Buckinghamshire furniture industry. There were now 48 cases of nasal carcinoma registered with the Oxford Regional Cancer Register between 1945 and 1981. The authors indicated that skilled furniture makers such as wood machinists, cabinetmakers, and chairmakers had experienced a cumulative lifetime risk of having nasal adenocarcinoma of at least 1 in 120 during the period studied. In addition, using a birth cohort analysis, they reported that the incidence ratio for nasal adenocarcinoma increased to a maximum for the men born during 1900-1909, whose median year of entry into the furniture industry was about 1920, and declined thereafter. However, the decline in incidence of nasal adenocarcinoma in men was not statistically significant. The incidence of nasal carcinoma among skilled woodworkers in the furniture industry was more than 12 times that among other workers in the industry. Comparing those who had worked less than 20 years in the furniture industry with those who had worked 20 years or more, the authors reported a significantly ($p<0.05$) greater risk for nasal cancer in the latter group.

Rang and Acheson (1981) and Acheson et al. (1984b) reported on a group of more than 5,000 men who were born before January 1, 1940, and who had worked prior to December 31, 1968, for any period of time in at least 1 of 9 furniture factories in Buckinghamshire. The initial report (Rang and Acheson 1981) examined 5,371 men through the end of 1968. The authors found a hundredfold increase in nasal adenocarcinoma in furniture workers as compared to the local population, and a significant relationship between increasing incidence of both nasal cancer (all histological types) and adenocarcinoma and qualitatively increasing dustiness of work within the cohort. The follow-up study (Acheson et al. 1984b) described 5,108 men (1,638 of whom had died) who had been followed to the end of 1982. There was a significant ($p<0.05$) deficiency of deaths observed from all neoplasms combined (standardized mortality ratio [SMR] 88). However, the nine deaths due to nasal adenocarcinoma were significantly ($p<0.05$) greater than expected. These nine cases were all recorded in occupations qualitatively classified as "very dusty." The mortality patterns among skilled workers were also examined by presumed duration of work in and by time since entry into the furniture industry. In both cases there were significantly ($p<0.05$) increasing trends for nasal adenocarcinoma.

Sarvesvaran and Bowen (1985) described two fatal cases of nasal adenocarcinoma in British woodworkers. The first was an 81-year-old man who had been diagnosed at age 70 as having adenocarcinoma of the ethmoid sinuses. The second, a 63-year-old male who had been a cabinet worker for 20 years, was diagnosed as having adenocarcinoma of the nasal sinuses.

Gardner and Winter (1984) described a recently developed statistical technique to examine the relationship between mortality and environment. As an example of the method, the authors reviewed nasal cancer mortality in British industries. Mortality data came from the Atlas of Cancer Mortality in England and Wales for 1968-1978, and industrial data came from the 1971 census, which contained information on 1,366 geographic areas and 207 industrial classifications. There were 1,556 male deaths from all types of nasal cancer. The furniture and upholstery industry showed the strongest relationship with nasal cancer (standardized slope = 0.1164, $p<0.01$).

Baxter and McDowall (1986) investigated the excess of male mortality from pleural mesothelioma and nasal and bladder cancer also reported in the Atlas of Cancer Mortality in England and Wales. The authors conducted a case-control study of four London boroughs that had significant woodworking industries. Fifty-four deaths were verified for the period 1968-1978, inclusive, for which cancer of the nose, nasal cavities, or sinuses were certified. Each was matched against two controls. The first control was randomly selected from male deaths from all other cancers, and matched for borough of residence, year of death, and age (within 5 years). The second control was randomly selected from male deaths from all causes including cancer (except nasal cancer) and matched as above. In the case group, eight deaths were among occupations known to be associated with high wood dust exposure: 4 cabinetmakers, 1 wood machinist, and 3 carpenters. In the combined control groups, there were three French polishers, a woodworking occupation which has lower wood dust exposure than the above three occupations. If the control group excluded the three polishers, then there was a significantly ($p<0.05$) increased risk for nasal cancer with woodworking. However, if the three polishers were included in the analysis, the association between nasal cancer and woodworking was not statistically significant (relative risk 2.5, 95% confidence interval [CI] 0.7-8.8).

In a compilation of occupational mortality statistics (Registrar General 1986) for men in Great Britain who died aged 16-74 in the 4 years 1979, 1980, 1982, and 1983, there was no increase in nasal cancer deaths among all woodworkers and pattern makers aged 20-64. However, the increase among men aged 65-74 was significant (proportional mortality ratio [PMR] 267 based on 8 cases, $p<0.05$). This difference between age groups was attributed to the long latency period for the development of nasal cancer. An increase in nasal cancer deaths among cabinetmakers aged 20-64 (PMR 1,020 based on 2 cases, $p<0.05$) was also seen. Carpenters and joiners had a nonsignificantly increased PMR (244 based on 6 cases) for ages 65-74. The report cautioned that one of its shortcomings was the fact that the occupation coded was the deceased's last occupation and might not represent the primary occupation or the one responsible for the risk leading to death.

United States--

The first report linking wood dust exposure in the U.S. to nasal cancer was that of Brinton et al. (1976). They described an ecological study in which the cancer death rates from 1950 to 1969 among inhabitants of 132 counties in the United States having at least 1% of their population employed in furniture and wood fixture manufacturing were compared with 264 control counties. Counties were matched for regional location, population, nonwhite proportion, median family income, and level of education. The ratio of the age-adjusted mortality rate for cancer of the nasal cavity and sinuses among white males in the furniture counties to the corresponding rate in the control counties was significantly (p value unspecified) greater than one.

In the following year, Brinton et al. (1977) described a death certificate case-control study of nasal cancer among workers in 19 North Carolina counties where at least 1% of the total population was employed in wood furniture and fixture manufacturing. Thirty-seven deaths attributed to cancer of the nasal cavity and sinuses for the period 1956-1974 were compared with 73 controls dying from other causes. The groups were matched by sex, race, county of death, age at death (within 2 years), and year of death. Comparison of death certificates showed that 8 of 37 people (21.6%) dying from nasal cancer had been employed in the furniture industry, while only 5 of the 73 controls (6.8%) had been so employed. An additional 5 cases (13.5%) were persons with wood-related jobs, as compared with 7 (9.6%) of the controls. This yielded an odds ratio of 4.4 (95% CI 1.3-15.4) for employment in the furniture industry as compared to 1.5 (95% CI 0.4-4.3) for other woodworking operations. The excess risk among furniture workers was seen both in those who died at or above age 65 and those who died younger. Among the 13 cases for which histology was recorded, 4 were adenocarcinomas (3 of which were furniture workers), 4 were squamous cell carcinomas, 3 were undifferentiated carcinomas, 1 was lymphoepithelioma, and 1 was myxosarcoma.

Brinton et al. (1984) followed up their previous work with a case-control study of primary malignancies of the nasal cavity and sinuses diagnosed at four hospitals in North Carolina and Virginia between 1970 and 1980. For cases alive at the time of interview (1980-1982), two controls were selected and matched by hospital, year of admission (within 2 years), age (within 5 years), sex, race, and state economic area (or county) of residence. These controls were required to be living at the time of interview. Excluded were patients with malignant neoplasms of the buccal cavity and pharynx, esophagus, nasal cavity, middle ear, accessory sinuses, larynx, or secondary neoplasms; benign neoplasms of the respiratory system; mental disorders; acute sinusitis; chronic pharyngitis and nasopharyngitis; chronic sinusitis; deflected nasal septum; or nasal polyps. For cases deceased at the time of interview, two different controls were selected. The first was a hospital control matched as above, but the control was not required to be living. The second came from a list of deceased individuals identified through state vital statistics offices. Matching criteria were similar to those used for living cases and included age (within 5 years), sex, race, county of residence, and year of death. A total of 193 cases, 232 hospital controls, and 140 death certificate controls were identified, from which successful interviews were completed for 160 cases, 178 hospital controls,

and next of kin for 112 death certificate controls. Risks were calculated relative to those who never worked in each industry and adjusted for sex of study subjects when possible. Among occupations involving potential exposure to wood dust, nonsignificant elevations in risk for males were associated with lumbering (relative risk 1.45), carpentry (1.60), and construction (1.23). There were no females in any of these categories. Among furniture manufacturers, the relative risk was 0.74 in males and 0.91 in females. Additional examination according to tumor histology showed a significantly ($p<0.05$) elevated risk of adenocarcinoma for males in the furniture manufacturing industry (relative risk 5.68) but not in other industries involving wood dust exposure (lumbering, carpentry, construction). There was no significantly increased risk of squamous cell carcinoma in any wood-related industry. The authors noted that of the ten woodworkers who developed adenocarcinoma (four of whom were furniture workers), seven were first employed 20 or more years prior to the development of the cancer, indicating a long latency period. Cigarette and pipe smoking and snuff use were shown to nonsignificantly increase the risk of cancers of the nasal cavity and sinuses. These three tobacco habits were moet strongly related (still nonsignificantly) with squamous cell tumors.

Roush (1978) and Roush et al. (1980) reported on a case-control death certificate study of cancer of the nose and paranasal sinuses in Connecticut. A total of 216 verified cases of sinonasal cancer were compared with 662 male deaths from 1935-1975 at age 35 or older, using random sampling without stratification or matching of any kind. However, a comparison of cases and controls revealed a similarity for certain demographic characteristics, including year of death, age at death, percentage of foreign born, percentage in two or more different industries, and percentage in two or more different jobs. The authors calculated an odds ratio of association between sinonasal cancer and wood dust of 4.0, which was statistically significant ($p<0.011$). Carpenters, most of whom were in the construction trade, showed no elevated risk. Although numerical relative risks were not calculated, cabinet workers and other woodworkers were considered high risk groups. There were eight cases of sinonasal cancer in the occupational groups associated with wood dust exposure, two of which were adenocarcinoma and three of which were squamous cell carcinoma.

Lingeman et al. (1982) reported on 72 patients (45 men, 27 women) with glandular neoplasia of the sinonasal tract whose biopsies had been examined at the Armed Forces Institute of Pathology. Ages ranged from 7 to 90 years (median 58). Ten of the adult males had worked in occupations with potential wood dust exposure; an eleventh reported working with wood in a home workshop. The neoplasms of all but one of these men were mucin-producing adenocarcinomas. No histologic tumor type predominated among male and female patients who had no known exposure to wood dust.

Viren et al. (1982), in a report prepared for the Inter-Industry Wood Dust Task Force, described a death certificate case-control study of nasal cancer deaths occurring from 1963 through 1977 in the states of North Carolina, Mississippi, Washington, and Oregon. These states were selected because of their diverse wood-related industries and wood species. The final study population consisted of 332 male cases, 204 female cases, and 1,072 controls

(2 per case) dying of other causes matched by sex, race, year of death, age of death (within two years), and county of death. Within the same sex, cases and controls reported similar cigarette smoking habits. However, the percentage of males who had smoked (77%) was higher than that of females (31%), and the males had smoked for a longer time. Solvent exposures were also similar in both cases and controls. Histological information about tumor type was not obtained. The authors calculated relative risks of 1.95 (significant at $p<0.05$) for industries involving lumber and wood products. When this category was divided into those industries where timber and logging were clearly involved and those where logging was not specified, a relative risk of 3.0 (significant at $p<0.01$) was calculated for the former. Other wood-related industries had a relative risk of 1.65, which was not statistically significant. No association between employment in the furniture industry and nasal cancer was seen. When the data were analyzed by occupation, only those in forestry and logging demonstrated a significant ($p<0.01$) relative risk for nasal cancer.

Based on information provided by the North Carolina Department of Vital Statistics, Imbus and Dyson (1985) analyzed nasal cancer deaths in the North Carolina furniture industry. There were 196 cases of nasal cancer between 1964 and 1977, of which 6 were in furniture industry workers. Extrapolating U.S. Census figures for the North Carolina population between 1960-1980, the authors calculated an average incidence rate of nasal cancer in North Carolina of 0.28 per million and in the North Carolina furniture industry of 0.94 per million. This excess of nasal cancer in furniture workers was statistically significant ($p<0.05$).

To demonstrate the difference in nasal cancer rates reported by Acheson et al. (1967,1968) in England versus those in North Carolina, Imbus and Dyson (1985) calculated that 626 and 816 nasal cancer deaths would have been seen in North Carolina furniture workers and in the rest of the North Carolina population, respectively, if the English occurrence rates were used. This compares to the 6 and 190 cases, respectively, actually seen in North Carolina. The authors hypothesized a number of reasons for the much greater excess of nasal cancer in England versus that seen in North Carolina. These include: (1) exposure to higher concentrations of dust, (2) different types of woods processed, (3) presence of chemicals or other additives in or on the wood (4) partial pyrolysis caused by different manufacturing methods, and (5) different particle size distribution.

In addition, from the work of Brinton et al. (1984) and statistics from North Carolina, Imbus and Dyson (1985) showed a decreasing number of nasal cancer deaths among furniture workers during 1956-1966 (5 deaths) and 1966-1977 (3 deaths). According to the American Furniture Manufacturers Association, no cases of cancer were seen during 1974-1984. From this, the authors concluded that a slight excess of nasal cancer may have existed in the North Carolina furniture industry, but that the excess appeared to reflect earlier manufacturing conditions and is currently either declining or nonexistent.

Barnes (1986) described the clinical and pathologic features of 17 cases (9 men, 8 women) of intestinal-type adenocarcinoma (ITAC) of the sinonasal

tract. These cases were selected from the files of the Presbyterian-University Hospital and Eye and Ear Hospital of Pittsburgh for the years 1952 to 1984. The occupation of 14 of the patients were available; only one of those, a cabinetmaker, seemed to indicate occupational exposure to wood dust. Eight tumors originated in the maxillary sinus, seven in the nasal cavity, and two in the ethmoid sinus. The author also summarized the clinical and pathologic features of 213 cases of ITAC. At least 19% of these occurred in woodworkers. Based on this review, Barnes commented on the differences between ITAC that occurs in woodworkers and ITAC that occurs sporadically in the general population. The former occurs primarily in men, originates almost exclusively in the nasal cavity or ethmoid sinus, and has a better prognosis.

The National Cancer Institute (NCI) is currently conducting a cohort mortality study of 36,622 workers first employed between 1946 and 1962 in the manufacture of wood, metal, and plastic furniture and identified from membership records of the United Furniture Workers of America insurance fund (Miller 1987). Preliminary results revealed two nasal cancer deaths among white males in the study cohort (3.6 expected). One was a 75-year-old chrome furniture worker and the other was a 46-year-old upholstered wood furniture worker. The authors have cautioned that the study was limited by its use of broad industrial classifications and single job titles, by incomplete work histories and lack of detailed exposure information, and by a lack of power to detect risks for rare causes of death such as nasal cancer.

Canada--

Ball (1967) published a death certificate analysis of nasal cancer in Canadians during the decade 1956-1965. Occupations were grouped into 10 categories; woodworkers included the logging industry, bushworkers, carpenters, cabinetmakers, and timber workers. Death certificates on which the underlying cause of death was stated to be cancer of the nose and nasal cavities were matched with the nearest succeeding certificates that corresponded in sex, year of death, and province of habitation; ages were matched as closely as possible, and in 96% of the cases were within 2 years. A total of 508 cases of cancer of the nose and sinuses were obtained: 340 in men and 168 in women. These numbers yielded a male/female ratio of 2.02. Slightly over 58% of these cancers involved the maxillary sinuses; the remaining number of tumors were equally divided between the other sinuses and the nose and nasal cavities. There were 28 cases of nasal cancer among woodworkers: 16 in furniture makers and carpenters; 11 in lumberers, loggers, and sawyers; and 1 in a wood merchant. The corresponding numbers of nasal cancer cases among the controls were 14, 9, and 1. The author concluded that woodworkers had no significant excess of deaths due to nasal cancer.

In a subsequent communication based on the same collection of data, Ball (1968) reported no evidence for a localization in any Canadian province of an excess of deaths due to nasal cancer among woodworkers. He also separated furniture makers from the carpenter category in his initial

analysis, and he found no difference between either category and the controls.

Elwood (1981) described a retrospective case-control study of 121 men with cancer of the nasal cavity or paranasal sinuses who were seen between 1939 and 1977 at the A. Maxwell Evans Clinic, the main cancer treatment center in British Columbia. Each control was the next patient (after the case patient) admitted to the Clinic who was in the same 5-year age group as the case patient and whose condition had been diagnosed within a 2-year period of the case patient's diagnosis. This control group contained patients with tumors showing no strong relation to outdoor exposure or smoking (stomach, large bowel, prostate, bone, connective tissue, lymphatic, and hematopoietic). Information on occupation, smoking habits, and ethnic origin were taken from each patient's standardized medical record. Twenty-eight cases were in occupations involving wood exposure: 10 loggers, 7 carpenters, 4 laborers in forest industries, 4 construction workers, 2 log scalers, and 1 cabinetmaker. In the first control group, 15 patients were in a wood-related occupation. The author calculated a significant ($p<0.03$) relative risk of 2.5 for sinonasal cancer associated with wood exposure, when adjusted for smoking and ethnic origin. The relative risk for smoking, when adjusted for wood exposure and ethnic origin, was also significant (4.9, $p<0.0003$). The unmatched estimate of risk associated with both wood exposure and smoking was 7.1. There were 11 adenocarcinomas, 61 squamous cell carcinomas, 20 anaplastic carcinomas, and 6 sarcomas among the cases. An increased risk with wood exposure was seen for nasal cancer of all these tumor types. The authors concluded that both wood exposure and smoking were associated with increased risk of tumors of the nasal cavities and nasal sinuses. Using Canadian census figures they calculated an annual incidence of nasal cancers of 15.7 per million for workers exposed to wood compared to 6.5 per million for other workers. According to these estimates, a worker exposed to wood annually from age 20 would have a risk of developing the disease by age 70 of almost 1 in 1,000.

Finkelstein (1985) described a death certificate case-control study among Ontario workers who had died during 1973-1983 from malignant neoplasia of the nose, nasal cavities, middle ear, and accessory sinuses. A control was randomly selected from deaths in the same year and matched by age and sex. There were eight deaths identified among men with definite wood dust exposure (three furniture workers and five carpenters) and three among the controls. The resultant odds ratio was not statistically significant, but the author cautioned that the sample size was too small to permit meaningful analysis.

Gallagher and Threlfall (1985) conducted a proportionate mortality study of 420,814 deaths in individuals aged 20 and older occurring in British Columbia, Canada from 1950 to 1978. The study included 12,115 deaths in male woodworkers (carpenters, cabinetmakers, furniture makers, sawmill workers, lumber graders and scalers, and woodworkers not elsewhere classified); 9,931 in loggers; and 85 in pulp and paper workers. No deaths due to sinonasal cancer were reported.

Denmark--

Mosbech and Acheson (1971) reviewed the 123 Danish deaths due to malignant neoplasms of the nasal cavity, middle ear, and accessory sinuses during 1956-1966. Seventy-five of these were in men and 48 in women, giving a male/female ratio of nearly 1.6. In eight cases, woodworking was listed as the occupation of either the patient (six cases) or the husband of the patient (two cases). One of these cases was discarded because the tumor was in the nasopharynx rather than in the nasal cavities. A detailed occupational history was obtained for the remaining seven. Four of these seven had nasal adenocarcinoma, and all four had worked in the furniture making industry. A fifth man had been a woodworker for 3 years but had spent most of his life as a farm worker; he died from squamous carcinoma of the nasal cavity. Two women in the group had had no occupational contact with wood dust but their husbands were carpenters; both women died of undifferentiated carcinoma of the nasal cavity. The authors concluded that an increased risk of nasal adenocarcinoma had existed in the Danish furniture industry in recent years. An addendum to the paper mentioned that two other Danish woodworkers, neither of whom had worked in the furniture making industry, had died, in one case of squamous cell carcinoma of the nose and, in the other, of melanoma of the nasal septum.

Andersen (1975) and Andersen et al. (1976, 1977) described 186 patients with malignant and semimalignant tumors of the nose and sinuses treated at the ear, nose, and throat department of the Aarhus Community Hospital during 1965-1974. Among them were 17 patients (15 men and 2 women) with nasal adenocarcinoma. Twelve of these 17 (70.6%) were woodworkers: 10 woodcutters, 1 wood turner, and 1 coach maker. The latency period from first wood dust exposure until tumor development ranged from 28 to 57 years; the duration of employment ranged from 14 to 49 years. Ten of the 12 woodworkers with adenocarcinoma were interviewed and were found to have worked for years in the production of furniture, principally from beech, oak, or walnut. Several had also worked with mahogany, teak, and palisander. When the group of adenocarcinomas was eliminated from the totals, the proportion of woodworkers among the patients with other types of malignancies of nose and sinuses was only 7.1%. Ten of these other patients had also been employed in occupations in which they were exposed to wood dust: three carpenters, two woodcutters, and one each of wood turners, coopers, sawyers, lumberjacks, and brush binders. The most common tumor among these 10 people was squamous cell carcinoma; there were 2 malignant melanomas and 1 each of anaplastic carcinoma, adenocystic carcinoma, and papilloma. Based on this sample, Andersen (1975) calculated a yearly incidence rate of 0.5 cases of nasal cancer per thousand for Danish woodcutters, which was similar to that reported in England by Acheson et al. (1967). No Danish national rates were provided for comparison. The author concluded that adenocarcinoma of the nasal cavity and/or paranasal sinus in a person who had worked at the grinding and polishing of wood should be accepted in Denmark as an occupational disease.

Olsen and Sabroe (1979) reported on a 6-year follow-up study of mortality among Danish carpenters, joiners, and cabinetmakers. The cohort was defined as all members of the Danish Carpenter/Cabinet Makers' Trade Union on

January 1, 1971 aged 20-84. It included both active and retired workers. Mortality was determined from union and insurance company reports and the National Board of Health for the period January 1, 1971, to December 31, 1976. Of all causes of death, only the incidence of nasal cancer (histological tumor type unspecified) was above the national average (SMR 467, 95% CI 253-679). However, this statistic was based on only four cases, three among cabinetmakers and one among carpenters. The age-adjusted proportional relative risk of nasal cancer for cabinetmakers compared to carpenters was 2.33 (95% CI 0.32-16.65).

Olsen et al. (1984) described a case-control study of sinonasal cancer and occupational exposures. Using the Danish Cancer Registry and computerized employment histories from the National Supplemental Pension Fund dating to 1964, 839 cases of cancer of the nasal cavity, sinuses, and nasopharynx and 2,465 cancer (colon, rectum, prostate, and breast) controls diagnosed during 1970-1982 were examined. The controls were matched by sex, age (within 5 years), and year of diagnosis (within 5 years). A statistically significant excess risk for nasal cancer (histological tumor type unspecified) among males with exposure to wood dust was found (relative risk 2.5, 95% CI 1.7-3.7). No such association was seen for nasopharyngeal cancer. When a latency period of 10 or more years between first exposure and tumor diagnosis was introduced, the relative risk of sinonasal cancer increased to 2.9 (95% CI 1.8-5.3). When adjusted for formaldehyde exposure, the relative risk associated with wood dust remained significantly ($p<0.05$) elevated whether no latency (relative risk 2.1) or a 10 year latency (relative risk 3.8) period was considered. The authors indicated that persons with exposures to both formaldehyde and wood dust were at a higher risk of developing nasal cancer than were persons with exposure to only one.

Olsen and Asnaes (1986) further analyzed the Danish Cancer Registry data described in the preceding report to investigate the relationship between formaldehyde exposure and nasal cancer. After exclusion of cases for which histopathologic classification could not be confirmed, there remained for analysis a total of 759 (509 males, 250 females) nasal cancers, including 466 (310 males, 156 females) cancers of the nasal cavity and paranasal sinuses and 293 (199 males, 94 females) cancers of the nasopharynx. The same 2,465 controls previously selected were used. Among men, there was no significant ($p>0.05$) association of squamous cell carcinoma of the nasal cavity and paranasal sinuses or of cancer of the nasopharynx with either wood dust or formaldehyde exposure. However, for adenocarcinoma, the authors calculated a significant excess risk of 16.3 (95% CI 5.2-50.9) for wood dust after adjusting for formaldehyde among men ever-exposed. The relative risk for joint wood dust-formaldehyde exposure was 39.5 (95% CI 22.0-70.8). When a latency period of 10 years since first exposure was included in the analysis, the relative risk for adenocarcinoma with wood dust exposure increased to 30.4 (95% CI 8.9-103.9) and the joint risk increased to 44.1 (95% CI 22.2-87.8). For women, no risk estimates for any nasal cancers could be made because the exposure rates were too low.

Sweden--

Drettner and Stenkvist (1979) described a cytologic study of cells either rinsed or directly sampled by probe from the noses of woodworkers. In about 2% of the 715 woodworkers examined by direct sampling, metaplastic changes in mucosal cells were observed. However, no definitively precancerous or cancerous cells were identified. The authors stated that the frequency of ethmoidal cancer in the Swedish furniture industry was approximately 4-5 cases per year among 17,000 workers (0.26 per thousand).

Engzell et al. (1978) used the cancer registry of the Swedish National Board of Health and Welfare to locate cases of nasal adenocarcinoma reported during the decade 1961-1971. This registry covered approximately 95% of all new cancer cases in Sweden. There were 46 adenocarcinoma cases (36 males, 10 females). The occupations of the 10 women could not be determined. Of the 36 men, 19 (53%) had been joiners, 1 had been a building worker, 6 had been fitters, 3 had worked in flour mills, 3 had been transport workers, and 4 had had miscellaneous occupations. The authors calculated relative risk ratios for adenocarcinoma of 165 for joiners and 109 for furniture workers. They stated that there was a convincing association between adenocarcinoma and the joinery occupation, although no statistical analysis was provided. They did not consider smoking or snuff use to be a significant factor. Most joiners had worked with multiple species of wood, both hard and soft. The authors also found 212 cases of squamous cell or poorly differentiated carcinoma of the nose in 127 men (including 5 joiners and 8 other woodworkers) and 85 women. Among the men, exposure to a variety of organic dusts was common, but no conclusions about their association with nasal cancer could be made. Very little information about female occupations was available.

Engzell (1979) reported the initiation by the Nordic Council of a joint case-control study in Denmark, Norway, Sweden, and Finland to identify occupations and exposures associated with nasal cancer. He also discussed an enlargement of the Swedish study described in his preceding paper (Engzell et al. 1978). The author now had information on 44 men who had developed nasal adenocarcinoma. Twenty-two of these men were joiners and one other was a building worker. The latency period from first exposure to wood dust to the diagnosis of nasal adenocarcinoma for 21 of the joiners varied from 22 to 70 years, with a mean of 44.7 years. The exposure period ranged from 9 to more than 30 years. One man had worked in the industry for only 9 years 5 months and was diagnosed as having a nasal adenocarcinoma 39.6 years after ceasing his work as a joiner. The author noted the similarity between these cases and those reported in England and Denmark.

Malker et al. (1986) updated Engzell's (1979) occupational nasal cancer survey to include incidence data for 1961-1979. Among males employed in Sweden in 1960, 648 cases of nasal cancer were identified. Seventy-seven (12%) were adenocarcinomas, 306 (47%) were squamous cell carcinomas, and 265 (41%) were other histologic types. A standardized cumulative (19-year) incidence ratio (SIR), adjusted for age and geographic region, was used to estimate cancer risk. Significantly ($p<0.01$) increased incidences of adenocarcinoma (SIR 16.7) and total nasal cancers (SIR 4.1) were found for

the furniture industry. A nonsignificantly elevated adenocarcinoma SIR of 2.2 was also observed for work in the wood industry. When the data were examined by trade, significantly ($p<0.01$) increased SIR's were found among woodworkers for both adenocarcinoma (4.7) and total nasal cancers (1.5). Further categorization revealed that most of the adenocarcinoma increase was among furniture makers and cabinetmakers (SIR 9.8) and to a lesser degree among carpenters and joiners (SIR 2.3). However, the incidence of total nasal cancer in carpenters and joiners was not elevated (SIR 1.1). No increases were noted in any of the industries or trades for squamous cell carcinoma.

The results of the Nordic Council joint nasal cancer case-control study described above by Engzell (1979) were described in two papers by Hernberg et al. (1983a,1983b). Cases of cancer of the nasal cavity and paranasal sinuses came from Finnish and Swedish national cancer registries and from Danish hospital records for 1977-1980. One hundred and sixty-seven patients (110 men, 57 women) answered a standardized telephone interview, and they were individually matched by country, sex, and age at diagnosis (within 3 years) with living patients having colon or rectal cancer. The mean age of the interviewed cases was 64.1 for males and 62.5 for females. Eighteen of the cases were adenocarcinomas. The analysis was confounded because of mixed exposures to wood dust as well as paints, lacquers, and wood preservatives. Analysis of smoking habits suggested a synergistic effect, although the authors indicated that the numbers were too small to permit any definite conclusion. The authors reported that exposure to hardwood dust, either alone or in combination with softwood dust, was mainly associated with nasal adenocarcinoma. This association was significant ($p<0.05$) for the mixed dusts. No such relationship was found for softwood dust alone. The results also suggested an association between exposure to mixed wood dust and increased risk of epidermoid and anaplastic carcinoma.

Hardell et al. (1982,1983) reported the results of a case-control study designed to examine the relationship between nasal and nasopharyngeal cancer and exposure to phenoxy acid or chlorophenols. Cases consisted of male patients with nasopharyngeal cancer or cancer of the nasal cavity or paranasal sinuses, aged 25-85, who were reported to the Swedish Cancer Register in 1970-1979 and who were residents of three northern Swedish counties at the time of diagnosis. These counties had a heavy concentration of forestry work and timber, pulp, and paper production. The group of 541 controls came from a pool of controls used in earlier cancer studies in northern Sweden. The controls came from the same counties as soft tissue sarcoma and lymphoma cases and were considered to accurately represent the general population. Smoking and snuff habits were similar in both cases and controls. A total of 44 cases of nasal cancer, including 3 adenocarcinomas, and 27 cases of nasopharyngeal cancer were seen. High-grade exposure (defined as more than 1 month total or more than 1 week continuously) to chlorophenols was related to a significant ($p<0.05$) sevenfold increase in both types of cancer. However, there was no significant association between either type of cancer and either phenoxyacid or low-grade chlorophenol exposure. Additionally, in woodworkers (sawmill workers, carpenters) not exposed to chlorophenols, no increased cancer risk was seen. Cabinetmakers

not exposed to chlorophenols had a doubled (but statistically not significant) risk of nasal cancer.

The above study by Hardell and coworkers elicited responses from researchers in Great Britain, Denmark, and Canada. Acheson et al. (1984a) indicated that chlorophenols were unlikely to have been used to treat hardwoods used in the British furniture industry. In addition, nasal adenocarcinoma occurred in British furniture workers who had left the industry before chlorophenols were introduced as a timber treatment. Acheson et al. (1984a) therefore concluded that chlorophenol exposure was not a likely cause of the occupational nasal cancers seen among British furniture workers. Olsen and Jensen (1984) reviewed 839 cases of cancer of the nasal cavity, sinuses, and nasopharynx selected from the 1972-1982 Danish Cancer Registry files. These cases were compared to 2,465 controls with cancer of the colon, rectum, prostate, and breast (matched by age, sex, and year of diagnosis). When adjusted for occupational wood dust exposure, there was no increased risk of sinonasal cancer among males with former exposure to chlorophenols. The authors concluded that their findings did not support the hypothesis that chlorophenols were significant in the etiology of nasal cancer in wood workers. Gallagher and Threlfall (1984) analyzed their British Columbia mortality data specifically for cancer deaths in male workers with presumed exposure to chlorophenols and phenoxy herbicides, including farmers, gardners and nursery workers, sawmill workers, pulp workers, and railroad track workers. The authors reported 14 deaths due to sinonasal cancer among farmers, which was statistically significant ($p<0.05$). They also reported one sinonasal cancer death in sawmill workers and one in pulp workers. The latter two figures differed from those published in their complete death record analysis (Gallagher and Threlfall 1985), in which they reported no deaths from sinonasal cancer among woodworkers. However, even the single deaths in sawmill and pulp workers were not statistically significant.

Klintenberg et al. (1984) described the treatment and clinical outcome for 28 patients (24 men, 4 women; mean age 66 years) with adenocarcinoma of the ethmoid sinuses seen at the University Hospital in Linkoping, Sweden since 1964. The region served by the hospital had approximately 900,000 residents and a large amount of furniture industries. Twenty (83%) of the men had been exposed to dust from hardwood for 20 to 55 years (mean, 40 years). Data on Swedish national rates of nasal cancer were not provided.

In the following year, Wilhelmsson et al. (1985a) examined the nontumorous part of nasal and ethmoidal mucosa adjacent to the tumors from the 20 wood dust-exposed men described above by Klintenberg et al. (1984) and an additional two cases, both of whom had been exposed to wood dust. Fifteen of the 22 cases presented papillary adenocarcinomas; the remaining 7 were alveolar adenocarcinomas, of which 3 were mucous-producing. In 19 cases cuboidal metaplasia was present, and 16 of those also showed dysplasia. A transitional zone with dyplastic cuboidal epithelium adjacent to the tumor was observed in 10 cases, and squamous metaplasia was seen in 5 cases. The authors proposed that nasal adenocarcinomas are preceded by metaplasia and dysplasia in cells having the ability to produce mucous. Such cells could then transform into either mucous-producing epithelium or metaplastic squamous epithelium. The predominance of observed cuboidal metaplasia might

imply that it is a precursor of ethmoidal adenocarcinoma. This hypothesis was further supported by the fact that in 16 cases cuboidal dysplasia was present, and in 10 cases dyplastic cuboidal epithelium was found contiguous with the tumor. Squamous dysplasia was not present in any of the cases.

Wilhelmsson and Lundh (1984) examined a group of 45 workers (mean age 40 years; mean exposure time 15 years, range 1-39 years) from 5 furniture factories in Sweden. Wood dust concentrations ranged from 0.35-5.1 mg/m^3 (mean 2.0 mg/m^3). A control group of 17 hospital staff (mean age 38 years) was used. Rhinoscopy was performed in all workers and controls, and smears were collected from the middle turbinate in the widest nasal cavity. Biopsies were taken from the same area at least 5 mm behind the anterior curvature of the middle turbinate. Thirty-nine (87%) of the 45 workers had normal nasal anatomy. However, 22 (49%) of the 45 had an abnormal mucous lining in the middle turbinate area. There was no correlation between histological and rhinoscopic findings. Histological examination of the biopsies revealed significant ($p<0.05$) increases in the workers relative to the controls in the occurrence of columnar epithelium, ciliated columnar epithelium, and metaplastic cuboidal epithelium. Nonsignificant increases in goblet cell hyperplasia, metaplastic epithelium, fibrotic submucosal connective tissue, and infiltration of round cells (lymphocytes and plasma cells) and eosinophilic leucocytes were seen in the exposed workers. The authors suggested that nasal mucosa changes caused by wood dust exposure occurred in steps. The first was loss of cilia and hyperplasia of the goblet cells, followed by cuboidal cell metaplasia.

Gerhardsson et al. (1985) presented the results of a 19-year cohort study among 8,141 Swedish men, aged 20-64 in 1960 and classified as furniture workers in the 1960 census. The reference group consisted of 1.4 million male blue collar workers, except those classified as furniture workers, aged 20-64 in 1960. Fourteen cases of adenocarcinoma were seen in furniture workers, 11 of the nasal cavity and 3 of the maxillary sinus. Seventeen cases of adenocarcinoma of the nasal cavity were seen in the reference population. Of these, five were in woodworkers (other than furniture workers). Therefore, of the 28 cases of adenocarcinomas of the nasal cavity that occurred between 1961 and 1979, 16 had been classified as woodworkers in the 1960 census. The authors calculated standardized morbidity ratios among the furniture workers of 7.1 (90% CI 4.4-10.9) for sinonasal cancer, 44.1 (90% CI 26.6-68.9) for sinonasal adenocarcinoma, and 63.4 (90% CI 35.5-104.9) for adenocarcinoma of the nasal cavity. The authors suggested that the excess risk of sinonasal cancer in woodworkers was confined to adenocarcinoma and that there had been no decrease in the incidence of sinonasal cancer in furniture workers in Sweden after 1965.

Finland--

Tola et al. (1980) described a case-control study of 45 patients over 35 years of age with malignant tumors of the nose and paranasal sinuses reported to the Finnish Cancer Registry during 1970-1973 from southern-southwestern Finland. This area includes most of the Finnish pulp and paper manufacturing, wood, and furniture industries. Controls were selected from among other cancer patients (excluding respiratory cancer)

living in the same geographical area. Two of the cases, one of whom was a joiner who had been exposed primarily to oak dust, had adenocarcinoma. Only one control, a carpenter, had wood dust exposure. Because of the small numbers, the authors concluded that their study was noninformative since no occupational group with an excess risk of developing nasal cancer was found. Because wood dust exposure was rare in both the cases and controls, they concluded that a case-control comparison for Finnish data was ineffective, and they emphasized that the absence of incidence should not be interpreted as proof of no effect. A similar inability to detect increases in nasal cancers among Finnish woodworkers was subsequently reported in a case-control study by Partanen et al. (1985).

Norway--

Boysen and Solberg (1982) examined nasal biopsies from 103 current (mean age 48 years, mean employment 34 years) and 10 retired (mean age 72 years, mean employment 44 years) Norwegian furniture workers and 57 controls (mean age 51) to determine if precancerous lesions could be detected in the nasal mucosa of furniture workers. Dysplasia was found in 14 of the workers (12%) and in 1 of the controls (2%); squamous metaplasia was found in 45 of the workers (40%) and 9 of the controls (17%). The biopsies were scored histologically on a scale of 0 (pseudostratified columnar epithelium) to 5 (dysplasia). The mean histological score was significantly higher ($p<0.05$) for workers than controls. The mean score was nonsignificantly higher for smokers than nonsmokers in both workers and controls. Nasal stenosis and mechanical processing of wood were both significantly related to the histological score, while age, length of exposure, and type of wood (hard or soft) were not. The authors suggested that dysplasia might represent a preneoplastic lesion leading to nasal adenocarcinoma.

Voss et al. (1985) presented the results of a study of 70 cases of sinonasal cancer (40 males, mean age 66; 24 females mean age 60) treated at the Department of Otorhinolaryngology, National Hospital of Norway during 1972-1976. Forty-nine of the cases were classified as nasopharyngeal carcinoma, three were adenocarcinoma, and eight were non-Hodgkin's lymphoma. Occupational histories were obtained by telephone interviews. Twelve (30%) of the 40 male patients had worked as woodworkers (including cabinetmakers, joiners, and carpenters), in saw and planing-mills, or in forestry and were considered to have been exposed to wood dust. The mean latency period from first exposure to diagnosis was 44 years; the mean exposure period was 32 years. Only one of the 12, a cabinetmaker, had adenocarcinoma. Standard proportional morbidity ratios were significant ($p<0.01$) for sinonasal carcinomas for the general classification of woodworkers, for cabinetmakers, and for sawmill and planing-mill workers, and highly significant ($p<0.001$) for all workers exposed to wood dust. With the exception of one cabinetmaker, who had also processed hardwood, all patients exposed to wood dust had been exposed exclusively to native softwoods (primarily pine and spruce).

Italy--

Cecchi et al. (1980) reported a case-control study of 69 patients (47 men, 22 women) with primary cancer of the nasal cavity and paranasal sinuses seen at the Otorhinolaryngology Clinic and the Radiology Institute of the University of Florence from 1963 to 1967. Of the 62 cases for which biopsy results were available and patient/relative interviews conducted, 11 were adenocarcinomas. Three of these were woodworkers (average age 62) with 17 years average employment. When matched to either of two control groups (noncancer hospital patients and nonadenocarcinoma of the nose or paranasal sinus cancer patients), the association with woodworking was statistically significant ($p<0.001$). The authors indicated that smoking habits were not likely to be a source of bias.

In the following year, Merler et al. (1981) described an epidemiological survey of patients with tumors of the nasal cavity and paranasal sinuses seen in the Ear, Nose, and Throat Departments of five Verona provincial hospitals from 1969 to 1979. Of 25 patients (13 males, 12 females) with tumors, there were 16 carcinomas and 9 nonepithelial tumors. Among the carcinomas were three adenocarcinomas, all in male woodworkers. Two other workers with epithelial tumors (one squamous and one undifferentiated) had been employed in wood processing industries.

Battista et al. (1983) described a case-control study of 36 male patients seen at the Ear, Nose and Throat Clinic and the Radiotherapy Unit of Siena and diagnosed as having cancer of the nasal cavities or paranasal sinuses between 1963 and 1981. The controls, who were male patients admitted to the Medical Clinic of Siena for all causes except nasal neoplasia, were matched 5:1 to the patients for age (within 1 year) and time of admission (within 3 months). Information was obtained from all 36 patients and 164 controls by mail and telephone questionnaires. A total of 7 carcinomas, 4 of which were mucinous adenocarcinoma and 1 of which was squamous cell carcinoma, were seen in male patients employed in the wood and furniture industries. The median exposure duration was 40 years. The woods used by the patients were primarily oak, chestnut, poplar, and fir. The odds ratio associated with exposure to wood dust was 5.4 (95% CI 1.7-17.2) for all carcinomas and 89.7 (95% CI 19.8-407.3) for mucinous adenocarcinomas.

Ghezzi et al. (1983) described the incidence of malignant tumors of the nasal cavity and paranasal sinuses occurring in 1976-1980 in Como and Milan provinces, a geographic area where there is a high concentration of furniture industries. Information was obtained from hospital records. For all types of nasal cancer, the ratio of incidence rates among woodworkers to others was 4.42, which was similar to that in Siena (Battista et al. 1983). For adenocarcinoma, the rate ratio was 56.5, but the reliability of this value was questioned because of problems in evaluating certain census figures. When the data were examined using a case-control approach with the controls being nonadenocarcinoma nasal tumors, an odds ratio of 60 was calculated, which was consistent with the rate ratio estimate.

Petronio et al. (1983) reported on a retrospective study of cancer of the nasal cavity and paranasal sinuses in the province of Trieste during

1968-1980. Information came from the biopsy register of the Department of Pathology. Twenty-one cases (10 males, 11 females) were ascertained, and occupational histories and smoking habits were obtained by interview for 18 of the cases. Two cases of adenocarcinoma (one in a cabinetmaker) and seven cases of squamous cell tumor (one in a joiner) were seen. The authors calculated a nationwide annual incidence rate of nasal cancer of 5.4×10^{-6} with no sex difference, which was one of the lowest in Europe, and an incidence rate of 6.4×10^{-5} among woodworkers.

Loi et al. (1986) reported on the preliminary results of a case-control study of 49 male patients with nasal cancer admitted to hospitals in the vicinity of Pisa. For each, five controls were selected from among those hospital patients with other than nasal or respiratory cancers or lymphomas. They were matched on sex, age, residence, and time of hospital admission. Information on occupational exposures and smoking habits was obtained by a mail questionnaire. At the time of this report, responses had been received from 45 cases and 139 controls. Histological information was available on 38 nasal tumors, indicating that 12 were adenocarcinomas (6 of which were mucinous adenocarcinomas) and 21 were squamous cell carcinomas. Of the cases and controls, 76.3% and 82%, respectively, were smokers. When all tumor types were considered, a significant ($p<0.05$) relative risk of 6.16 was found for woodworkers. Wood exposure showed a significant ($p<0.05$) relative risk of 13.83 for adenocarcinomas. However, no significant risks were observed for mucinous adenocarcinomas or squamous cell carcinomas. The authors suggested that adenocarcinomas were associated with wood dust exposure and squamous cell carcinomas with smoking.

France--

Gignoux et al. (1968) reported on 52 cases of malignant tumors of the ethmoid and maxillary sinuses seen between 1952 and 1967. Sixteen of these patients were woodworkers (11 cabinetmakers, 3 carpenters, 1 sawyer, and 1 barrel maker). Fifteen of these 16 had adenocarcinomas. The mean age of the woodworkers was 58 years. All had worked with wood since the age of 16, indicating a latency period of over 40 years from first exposure to wood dust until recognition of nasal cancer. In the following year Gignoux and Bernard (1969) added one more case of nasopharyngeal cancer in a cabinetmaker to their series. There was now a total of 53 nasal cancers, of which 16 were adenocarcinomas in woodworkers. Two years later, Gignoux et al. (1971) wrote again about the same group of 53 patients, indicating that no additional cases of cancers of the ethmoid had come to their attention during the 2 years since their last report.

Fombeur (1972) described five cases of tumors of the ethmoid and maxillary sinuses among joiners and cabinetmakers. Three of the patients had ethmoidomaxillary epitheliomas, two of which were identified as adenocarcinomas, the fourth had Wegener's disease, and the fifth had a metaplastic nasal polyp that seemed to be undergoing malignant transformation in a part of its periphery. Fombeur suggested that the irritant effect of wood dust resulted in hyperplastic, or even metaplastic, lesions of the sinonasal mucosa, and that such changes preceded the appearance of any malignant tumor.

Adenis et al. (1973) reported on 125 cases of nasal cancer from among 41,613 French hospital records (300 per 100,000 admissions) collected through December 1970. Of 115 tumors that were subjected to biopsy, 103 were carcinomas and 12 were sarcomas. Of the former group, 21 were adenocarcinomas (18 of the ethmoid, 1 of the nasal fossa, and 2 of the sphenoid and maxillary sinuses). Fourteen of the 18 patients with ethmoid adenocarcinomas were men, 10 of whom worked with wood: 6 as cabinetmakers, 1 as a lumberjack, 1 as a sander, 1 as a cooper, and 1 as a broom maker. The occupations of the 4 women with ethmoid adenocarcinomas were not known. The male patients had started work between the ages of 13 and 16 years; the average age at which the first symptoms of nasal cancer appeared was 60 years, giving a latency period from the first exposure to wood dust of 44-47 years. The authors considered adenocarcinoma of the ethmoid in woodworkers to be an occupational cancer.

Leroux-Robert (1974) summarized French, English, and Dutch references and noted that among 450 patients with cancer of the ethmoid, 124 (27.6%) were woodworkers. To these he added 100 new patients with cancer of the nasal cavity (particularly the ethmoid), 26 of whom were male woodworkers. The average age of these 26 men was 60 years (range 37-75 years) and the average duration of their exposure to wood dust was 40 years (range 22-54 years). The patients named oak, fir, poplar, walnut, chestnut, wild cherry, sipo, niangon, okoume, and iroko as the woods with which they worked. Four of the 26 patients had never used tropical woods, 7 had used them very little, and 6 had used them for only a few years. Leroux-Robert did not consider that agents added to wood or coming from the other materials used by the woodworker were causes of the carcinomas because they were too varied and too inconsistently used. The author suggested that tannin present in the wood might be responsible for ethmoid cancers in woodworkers. He concluded that exotic woods should not be considered to be the only ones involved in the induction of nasal cancers among woodworkers, which should be classified as an occupational disease.

Luboinski and Marandas (1975) observed 43 cases (41 males, 2 females) of adenocarcinoma among 67 cases of primary malignant epithelial tumors of the ethmoid seen at the Gustave Roussy Institute at Villejuif during the period 1957-1974. Twenty-one of the 43 patients (49%) with adenocarcinomas had been woodworkers: 11 cabinetmakers, 8 carpenters, 1 barrel maker, and 1 coffin maker. None of the other 24 cases of nasal cancer was among woodworkers. The woods used by these patients included both European woods, such as oak, chestnut, wild cherry, walnut, beech, and poplar; and exotic woods, such as mahogany, sipo, okoume, niangon, teak, makore, African walnut, and bosse. Among the 21 patients identified as woodworkers, 11 were still working, 7 had retired, and 3 had changed professions. The mean latency period between initial wood exposure and the appearance of cancer of the ethmoid in these three groups was 42, 52, and 40 years. The mean duration of exposure to wood was 42, 45, and 6 years, respectively. The authors also suggested that the frequency with which cancer of the ethmoid affects woodworkers justifies its inclusion as an indemnifiable occupational disease for workers exposed to wood dust, along with erythematous and eczematous dermatitis, conjunctivitis, and asthma. The last three

conditions had been indemnifiable for woodworkers in France since February 1967.

Curtes et al. (1977) described 21 adenocarcinomas among 36 primary malignant tumors of the ethmoid seen at the otorhinolaryngological clinic at Rennes between 1964 and 1973. Thirteen of the adenocarcinomas were in woodworkers. One other woodworker had a squamous cell carcinoma. All the woodworkers were men. Three of the fourteen no longer worked with wood. Their exposure duration was 9.6 years and latency period from first exposure to diagnosis was 27 years. Exposure duration for the remaining 11 averaged 46.4 years. Woods used by the woodworkers included chestnut, oak, beech, fir, ash, walnut, cherry, mahogany, gaboon, citron, and iroko. The authors summarized from the literature and their own cases a total of 557 ethmoid cancers, of which 157 (28.2%) were in woodworkers. These cancers included 213 adenocarcinomas, 111 (52.1%) of which were in woodworkers. The authors concluded that ethmoid adenocarcinoma in woodworkers should be recognized as an occupational disease.

As part of a series of articles published on workers in the French wood industry, Petiet et al. (1981) reported on 25 ethmoid cancer patients in Nancy diagnosed between 1975 and 1979. Nine of the 25 (36%) were woodworkers (average age 58 years). Six of the nine had adenocarcinoma. The authors also summarized eight French studies (including their own) between 1972 and 1979 and reported that of 458 ethmoid cancer patients, 150 (32.75%) were woodworkers. Of these 150, 139 (92.66%) had adenocarcinoma. Cabal and Teyssier (1981) described eight cases of ethmoid cancer diagnosed in the Loire province between 1975 and 1980. Seven of the patients were woodworkers. The average length of wood dust exposure for the five cases for which a detailed history was provided was 39 years. Brugere et al. (1981) reported on 53 ethmoid adenocarcinomas (48 males, 5 females) diagnosed at the Curie Institute in Paris between 1961 and 1980. Of the 44 males for which occupational history was available, 35 (80%) were woodworkers. Marandas et al. (1981) reported that between 1961 and 1979, there were 112 cases of ethmoid cancer out of a total of 15,712 cancers diagnosed at the Gustave-Roussy Institute. Of these 112, 61 were adenocarcinomas, and 35 of those were in woodworkers.

East and West Germany--

Gulzow (1975) described a case of ethmoid adenocarcinoma in a 59-year-old patient who had worked as a carpenter for 44 years. The author considered the possibility that this was a case of occupationally related adenocarcinoma. As part of his report, Gulzow also obtained thin layer chromatograms from methanol extracts of wood shavings, wood powder, and wood tar prepared from beech and oak, two types of wood used frequently by carpenters. The powders from both woods yielded greater amounts of methanol-soluble compounds, which were attributed to the powders' greater surface area. The author proposed that cases of adenocarcinoma of the paranasal sinuses among woodworkers exposed to fine hardwood dusts be compiled to determine whether this disease should be considered an occupational one in German workers.

Lobe and Ehrhardt (1978) published a study of malignant disease of the nose and paranasal sinuses among 420 patients (252 males, 168 females) admitted for treatment to the otorhinolaryngological clinic at the University of Jena in the German Democratic Republic (GDR) between 1931 and June 1, 1977. The occupations of 179 patients (111 males, 68 females) could be determined; 37 (20.8%) were woodworkers. Adenocarcinomas accounted for 18 (10.1%) of the malignancies, of which 13 (72.2%) were in woodworkers. This association between woodworking and adenocarcinoma was significant ($p<0.001$). All of the affected woodworkers were over age 40. Woodworkers had 19 other carcinomas, 3 sarcomas, and 2 other malignancies of the nose and paranasal sinuses. The woodworkers did not differ from the general population in smoking habits.

In a retrospective epidemiological study, Smetana and Horak (1983) examined 127 patients with malignant tumors of the nose and paranasal sinuses. Adenocarcinomas accounted for 22.8% of the total number of malignancies. Although only 2.14% of the total number of occupationally active patients in the authors' area of examination were woodworkers, 17.2% of the patients with adenocarcinoma and adenocystic carcinoma had been exposed to wood dust. Exposure duration ranged from 15 to 35 years. The correlation between wood dust exposure and these types of tumors was significant ($p<0.001$).

In an empirical case study, Grimm et al. (1984) reported the results of a survey of hospitals with otorhinolaryngology departments in the Federal Republic of Germany and West Berlin. Occupational histories were determined by questionnaires to both the patient or his survivors and to his supervisor or colleagues at work. When possible, the workplace was also inspected. Of the 66 cases of adenocarcinomas identified, 40 (36 males, 4 females) diagnosed between 1964 and 1983 were evaluated. Twenty-two of the 36 male cases and none of the females were in occupations involving wood exposure. Similar proportions of woodworkers (15/22; 68%) and nonwoodworkers (10/14; 71%) were smokers. Exposure to hardwood (oak and beech) was more frequently mentioned. The median latency period from time of first exposure to wood dust until diagnosis of adenocarcinoma was 40 years. No correlation between increasing period of exposure or latency period with frequency of adenocarcinoma was seen, but the authors cautioned that the small number of cases analyzed made it impossible to draw statistically significant conclusions.

Thurauf and Hartung (1984) described six cases of nasal adenocarcinoma reported for indemnification as an occupational disease in the Federal Republic of Germany following exposure to wood dust.

Gibel et al. (1985) described the results of a cross-sectional study of cancer of the nasal cavity and paranasal sinuses carried out in seven districts in the GDR and East Berlin. A total of 628 patients (404 males, 224 females) were identified from cancer registries for the period 1958-1979. Sixty-two (15.3%) of the men and 5 (2.2%) of the women were woodworkers. Among men, the most frequent tumor location was the ethmoid sinus (35.5%); adenocarcinomas were the most frequent histological type (35.5%).

Ernst and Ambrosch (1986) evaluated 40 patients with nasal adenocarcinoma and reported that they had been exposed to oak and beech dusts. The authors believed that this proved a causal connection between these types of dust and the disease, and recommended that worker compensation be permitted in individual cases following occupational exposure to oak or beech dust.

The Netherlands--

Delemarre and Themans (1971) described 16 patients who had been treated for nasal adenocarcinoma at the Anthony van Leeuwenhoek Hospital between 1944 and 1967. Eleven of the 16 (68.8%) were woodworkers: 6 furniture makers, 2 carpenters, 1 wagon maker, 1 mechanical woodworker, and 1 ship's carpenter working in a sawmill. These patients were compared to a control group of 33 patients, 3 of whom were woodworkers, treated at the hospital from 1956 to 1968 for carcinoma of the nose of a histological type other than adenocarcinoma. The relationship between woodworker occupation and nasal adenocarcinoma was significant ($p<0.001$).

In a case-control study designed to examine the relationship between nasal cancer and wood dust (Hayes et al. 1986a) and formaldehyde (Hayes et al. 1986b) exposure, the authors identified 116 Dutch males with histologically confirmed cancer of the nasal cavities or accessory sinuses newly diagnosed between 1978-1981. Of the 116 cases, 67 were squamous cell carcinomas, 28 were adenocarcinomas, and 21 were other types. The control group consisted of age-stratified random samples of living male residents of the Netherlands in 1982 (223 controls) and of deceased males in the Netherlands in 1980 (36 controls). Interviews, including smoking and alcohol consumption information, were completed for 91 cases and 195 controls. Exposure to wood dust and formaldehyde was rated subjectively by one of the authors on a scale from 0 (none) to 9 (highest) based on job histories. When all types of nasal cancer were combined, only employment in the paper and wood industry was associated with a significantly ($p<0.05$) increased risk of nasal cancer. This was due exclusively to the strong association with adenocarcinoma (age-adjusted odds ratio [OR]=11.3). For wood-related occupations, there was a significant risk for adenocarcinomas associated with ever being employed in furniture and cabinetmaking (OR=139.8) and in factory joinery and carpentry work (OR=16.3). No increased risk was seen for other types of nasal cancer. When wood dust exposure concentrations were stratified as low (levels 1-2), medium (levels 3-4), or high (levels 5-9), a significant risk of squamous cell carcinoma was associated with low levels of exposure only (smoking-adjusted OR=2.5). There was also a significantly elevated relative risk for adenocarcinoma associated with high levels of exposure (smoking-adjusted OR=26.0). The association between wood dust and adenocarcinoma was strongest for those employed in wood dust-related occupations between 1930 and 1941 (OR=86.0), particularly with first employment during that period. There were no cases of adenocarcinoma among those newly employed in wood-related occupations after 1941. The authors suggested that this might mean that the risk factors associated with adenocarcinoma had changed since that time. However, they also cautioned that the long latency period generally associated with this disease may make such a conclusion premature. There was also a significant relationship between duration of employment at high exposure levels and adenocarcinoma.

The odds ratios were 10.1 for less than 10 years exposure, 38.4 for more than 10 years exposure, and 33.0 for more than 30 years exposure. There was no evidence that the relative risk of adenocarcinoma was lower among those whose exposure ceased in 1966.

Belgium--

Debois (1969) reported that of 29 patients from Mechelse with tumors of the nasal cavity and the ethmoid sinus seen between 1958 and 1968, 16 were woodworkers or apprentices and 2 others (a furniture worker and a wood industry freight worker) had some contact with woods. Among these 18 people, 15 had adenocarcinomas, 2 had epidermoid carcinomas, and 1 had an undifferentiated cancer. Among the 11 patients without any relation to woodworking, 5 had adenocarcinomas, 4 had epidermoid carcinomas, and 2 had undifferentiated cancers. The author suggested that further epidemiological study of cancer in woodworkers be conducted.

Austria--

Loffler (1972) published a summary of 64 patients (34 men, 30 women) with malignant tumors of the nose or the paranasal sinuses treated from 1960 to 1970 at the First Otorhinolaryngological Clinic of the University of Vienna. Thirty-seven of these patients had carcinomas, 2 of which were adenocarcinomas of the ethmoid and 2 others were adenocarcinomas of the maxillary sinus. Two of the patients with adenocarcinomas had worked as or with a cabinetmaker; the other 2 were women without any known connection to a wood-processing occupation. Loffler concluded that the clinic did not have sufficient industry of that sort to furnish a statistically valid sample for testing the hypothesis that occupational exposure to wood dust resulted in the development of adenocarcinoma of the nasal mucosa.

Switzerland--

Ruttner and Makek (1985) described a case series of 31 Swiss patients with adenocarcinoma of the nose and paranasal sinuses observed between 1953 and 1984. Thirteen of the patients had occupational exposure to wood dust. The authors suggested an association of adenocarcinoma with wood dust, although the nature of the carcinogen was unknown, and suggested further study.

Vader and Minder (1987) examined the relative risk of mortality from cancer of the nasal cavity and paranasal sinuses among Swiss furniture workers aged 15-79. Analyzing death certificates for the years 1979-1982 and national census figures for 1980, they observed 9 cases of sinonasal cancer among 41,667 furniture workers and 59 cases among 1,813,798 male workers in the general population. They calculated an SMR of 6.6 ($p<0.0001$, 95% CI 3.0-11.6) for death due to sinonasal cancer and an odds ratio of 230 (p value not specified) for death from adenocarcinoma. The authors concluded that sinonasal cancer in furniture workers was one of the most clearly defined occupational cancers in Switzerland.

Australia--

Ironside and Matthews (1975) discussed 19 cases of adenocarcinomas found among 99 cases of malignant tumor of the nose and paranasal sinuses at the Cancer Institute of Victoria. Eighteen of the cases were in men and only one in a woman, who was listed as a homemaker. This difference in sex distribution was significant ($p<0.0001$). Information regarding most recent occupation was available for 13 of the men. These included three carpenters, one timber worker, and one sawmill proprietor. When compared to either the 1966 male Victorian workforce or the total number of male patients registered at the Cancer Institute in 1973, the occurrence of nasal adenocarcinoma in 5 woodworkers out of a sample of 13 patients was significant ($p<0.00012$).

Summary--

The association between nasal cancer and occupations involving exposure to wood dust has been clearly established in the literature. This relationship was first noted in the late 1960's in Great Britain, where the incidence of nasal adenocarcinoma, a rare type of nasal cancer, among woodworkers in the furniture industry was found to be 10 to 20 times greater than among other woodworkers and 100 times greater than the general population. The association has been confirmed to varying degrees in Canada, Denmark, Sweden, Norway, Italy, France, East and West Germany, the Netherlands, Belgium, Switzerland, and Australia. Negative studies and insufficient data have also been reported in Canada, Finland, and Austria. In the United States, three studies have reported an approximately fourfold increased risk of either nasal cancer or adenocarcinoma in furniture workers, and a fourth has reported a similar relationship between nasal cancer and wood dust exposure. A fifth study failed to show an increased risk of nasal cancer among furniture workers, but did indicate such an increase among workers in logging and timber industries.

As noted above, the association between nasal adenocarcinoma and wood dust exposure is particularly strong among furniture industry workers, although other woodworkers have also been shown to be at risk. Similarly, exposure to hardwoods has been implicated most often, but there are several reports implicating softwoods as well. There is no conclusive evidence linking other histologic types of nasal cancers to wood dust exposure. There are also no quantitative concentration-response data, although there is some indication that adenocarcinoma is linked with qualitatively higher levels of wood dust exposure and that the risk increases with a latency period from first exposure. Studies in England and Scandinavia have also detected metaplasia and dysplasia in the nasal mucosa of workers exposed to wood dust.

B. Pulmonary Cancer

In his analysis of the 1969-1971 death records of the AFL-CIO United Brotherhood of Carpenters and Joiners of America, Milham (1974) found a significant ($p<0.05$) excess of malignant neoplasms of the bronchus and lung (SMR=106.7) and a slight but statistically nonsignificant excess for

malignant neoplasms of the more inclusive respiratory system category (SMR=102.9). When examined by trade, furniture workers, cabinetmakers, and millmen, lumber, and sawmill workers showed no increase in lung cancer incidence. Only construction workers showed a statistically significant elevated lung cancer rate (SMR=118).

Blot and Fraumeni (1976) conducted an ecological survey of lung cancer mortality in industrial counties of the United States during the period 1950-1969. These counties were defined as ones in which at least 0.1% of the population was employed in one or more of 18 manufacturing industries. No excess lung cancer mortality was seen in counties with lumber or furniture industries. However, lung cancer mortality among white males was significantly ($p<0.05$) elevated in counties with paper manufacturing industries.

Harrington et al. (1978) followed up the work of Blot and Fraumeni (1976) with a death certificate case-control study of the 858 white males in 12 coastal counties of Georgia who died of primary lung cancer during 1961-1974. Each of these decedents was matched for age at death (within 1 year), year of death (within 6 years), and county of usual residence with a white male who died of conditions other than lung or bladder cancer or chronic respiratory disease. No information on smoking habits was provided. There were 97 cases employed in the wood and paper industry versus 78 in the controls. This difference was not statistically significant. When examined by county, there was an excess risk associated with the wood and paper industry (relative risk=3.3, $p<0.05$) in the combined nine smaller counties but not in any of the three largest counties. The nine smaller, less populated, counties had 20.1% of the people dying of pulmonary cancer employed in the wood and paper industry whereas only 7.2% of the control population in these counties was employed in that industry. Harrington et al. (1978) stated that the risk was greatest among sawmill, lumber, and forestry workers, but provided no data to support this assertion.

Blot et al. (1982) conducted a two-part case-control study to examine the high lung cancer mortality along the northeast coast of Florida. The first part of the study was hospital-based and included all male residents in Duval County newly diagnosed with primary lung cancer at one of 13 Jacksonville hospitals during the period April 10, 1978, through April 9, 1979. Two controls were matched by age (within 2 years), race, and hospital. The second component used Florida State mortality files for 1976 for both cases and controls. The cases were all deaths attributed to primary lung cancer among male residents of four counties, matched by race, age, and county of residence to an equal number of randomly selected controls. In both parts of the study, controls excluded cases of lung cancer and chronic respiratory disease. Occupations were coded based on SIC codes. There were 181 cases and 342 controls in the first part of the study and 217 cases and 217 controls in the second. A total of 807 interviews were completed; after exclusions, the final study group included 755 subjects (321 cases and 434 controls). When adjustments were made for cigarette smoking, a statistically significant risk for lung cancer was seen for the lumber and wood industry (RR=1.7, $p<0.04$) and construction (RR=1.4, $p<0.04$). The excess risk in the former category, which included primarily

sawmill laborers, was primarily due to those with exposure to wood dust (RR=1.9) rather than those without (RR=1.2). The authors commented that the excess risk in this category was limited to smokers, raising the possibility of a co-carcinogenic effect.

Milne et al. (1983) conducted a death certificate case-control study of lung cancer in Alameda County, California between 1958 and 1962. The study group included 925 lung cancer deaths (826 as primary cause and 99 as significant condition present at death) and 6,420 deaths from other cancers. Deaths occurring under the age of 18 were eliminated because they were not considered to contribute to an analysis of occupational factors. No information on tobacco use was available. Occupation and industry information were coded by Bureau of Census Industrial and Occupational Classification System. Analyses were stratified on age at death and analyzed separately for males and females. Two overlapping control groups were utilized, one consisting of all other 6,420 cancer deaths and the other consisting of a "reduced" group of 4,880 other cancers not known to be strongly associated with occupational risk factors. Combined risk estimates were computed as the maximum likelihood estimate of the odds ratio. Males employed in the furniture industry had a significant ($p<0.01$) risk of lung cancer when compared to either the complete or reduced control group (OR=4.2 and 3.6 respectively). Those employed in construction also had a significant ($p<0.01$) risk of lung cancer (OR=1.6). No similar risk was shown for sawmills, or for the combined classification lumber/furniture manufacturing. When stratified by occupations, there was no significant lung cancer risk to male carpenters or cabinetmakers/furniture finishers.

Dubrow and Wegman (1983) identified occupations with potentially high cancer risk by combining the results of 12 major occupational disease surveillance studies covering the years 1950-1973 conducted in the United States and Great Britain. The analysis was limited to white males. Observed-to-expected ratios and observed numbers of deaths or cases were abstracted from these 12 studies, and comparison tables were constructed for occupational categories comparable across two or more studies. Carpenters and cabinetmakers were listed as having increased risk of cancer of the trachea, bronchus, and lung.

Stellman and Garfinkel (1984) analyzed data compiled from 1959 through 1972 in the American Cancer Society's prospective epidemiologic study of more than 1 million people in 25 states. The analysis was limited to only males alive on July 1, 1960. There were 10,322 woodworkers (carpenters/joiners, lumber and sawmill workers, and furniture/cabinetmakers) and 406,798 nonwoodworkers in the study population. Smoking habits, use of over-the-counter medications, and beer consumption were similar in both groups. Neither the woodworker subgroups nor the woodworkers as a whole had an excess combined cancer death rate when compared to the nonwoodworkers. However, carpenters and joiners did show a significant excess of lung cancer (age and smoking adjusted SMR=122, $p<0.03$). The authors hypothesized that men in this subgroup were most likely to be exposed to asbestos, which might have contributed to the rate. As an aside, the only nasal cancers observed in this study were in carpenters and joiners (2 cases). This was too small a number to permit meaningful statistical analysis, but was twice the number

expected in woodworkers and three times that expected based on carpenters and joiners only.

Digiesi (1972) described a case of diffuse nodular bronchiolar (alveolar cell) carcinoma with hydropneumothorax in a 40-year-old man who had been exposed to wood dust in an Italian woodworking factory since the age of 8 years. The patient was not a smoker. The author hypothesized that there was a causal relationship between the patient's exposure to wood dust and the onset of bronchiolar-alveolar carcinoma.

In the Rang and Acheson (1981) study of nasal cancer in English furniture workers described in part A of this section, no excess cancer risk at any other sites, including bronchial cancer, was found. However, there was a significant trend ($p<0.05$) of increasing bronchial cancer mortality with increasing dustiness of work (subjectively determined by job classification). When SMR's were estimated for skilled furniture workers (those who could reasonably be assumed to have started working before the age of 20), no excess of bronchial cancer was observed. However, again there was a trend of increasing mortality with increasing dustiness of work, although it was not quite statistically significant ($p=0.06$).

Esping and Axelson (1980) described a case-control study that examined the occurrence of respiratory and digestive tract cancer among woodworkers in Sweden. The study included 95 cases (25 respiratory cancers, none of which were nasal cancer, and 70 of the digestive tract, including mouth) selected from the local death register in the small Swedish town of Mjolby during 1963-1977. Only males over 50 years of age were included. These were matched against 370 male controls from the register in the preceding and two succeeding positions for each case. For woodworkers, a fourfold excess of respiratory cancer was found ($p<0.05$). Subdividing the woodworking category into "furniture makers" and "other woodworkers" suggested that the risk was primarily to the former. The authors concluded that there was excess mortality from respiratory cancer among woodworkers, especially furniture makers, that could not be explained by smoking or other confounding factors.

In their study of Swedish furniture workers described in part A of this section, Gerhardsson et al. (1985) found no increased risk for laryngeal or lung cancer.

Gallagher and Threlfall (1985), in their proportionate mortality study of deaths in British Columbia, Canada from 1950 to 1978, reported a statistically significant increase in deaths due to lung cancer among woodworkers and the subgroup carpenters. The PMR's were 119 (95% CI 105-134, $p<0.004$) and 122 (95% CI 106-140, $p<0.03$), respectively. Sawmill workers had lower lung cancer PMR's than expected but the lowered risks were not statistically significant. Lumber grader and scalers had normal lung cancer risk, while loggers showed a reduced risk. The authors commented that this might be the result of the large number of deaths due to accidents to loggers, which depresses the proportion of observed deaths due to cancer.

In a case-control study, Siemiatycki et al. (1986) reviewed 19 potential cancer sites among 2,180 male hospital patients aged 35-70 in Montreal,

Canada from September 1979 to June 1983. Information regarding the patients' life-styles and work histories was obtained via interviews or questionnaires. The control groups consisted of individuals drawn from those with other cancer sites in this study. Lung cancer was excluded from all control series. Five hundred and twenty-five (24.1%) of the subjects were considered to have had any exposure to wood dust, and 345 (15.8%) had substantial exposure. The main occupations in which wood dust exposure was coded were construction (carpenters, painters, pipefitters, plumbers, etc.), forestry and logging, and fabricating and assembling wood products. The odds ratio for the association of lung cancer with substantial exposure to wood dust was of borderline significance (OR=1.3, 95% CI 1.0-1.8), but was significant ($p<0.05$) when all wood dust exposure (doubtful plus substantial) was considered. Additionally, when the substantially exposed category was divided into two segments (less than or equal to 15 years' exposure and greater than 15 years' exposure), those with the longer exposure were shown to have a significantly ($p<0.05$) increased risk of lung cancer. Because this study controlled for smoking and other occupational exposures (e.g., asbestos), the authors claimed that their positive findings were persuasive.

Kauppinen et al. (1986) described a Finnish case-control study that examined a cohort of 3,805 men born after 1904 who had worked for at least 1 year in the particle board, plywood, sawmill, or formaldehyde glue industries between 1944 and 1965. One hundred and fifty-one primary malignant "respiratory" tumors were identified; there were no tumors of the nose or sinuses. From these, 57 cases were selected for which histological or cytological specimens or necropsy and hospital protocols were available. Three control subjects for each case were selected from the cohort and matched by birth year. The comparison of exposure was based on work histories and job exposure matrices. These matrices were constructed by one of the authors based on general and plant-specific hygiene data on exposures, information on ventilation, work procedures, and other relevant factors at the plants. At least 1 month of exposure time to wood dust was required to be considered "exposed." Exposure concentrations were classified as follows: low level (0.1-1mg/m^3), moderate level (1-5 mg/m^3) and high level (over 5 mg/m^3). The estimated average wood dust exposure concentration was 1-2 mg/m^3 and the mean duration of exposure was 10 years. When adjusted for smoking, the odds ratios for respiratory cancer for wood dust exposure were 1.60 with no provision for a latency period and 1.68 with a 10-year latency period. These were not statistically significant at the 5% level. There was no significant exposure-response relationship observed for either exposure concentration to or cumulative amount of wood dust. However, the odds ratio for cumulative amount over 5 mg/m^3-years was higher than that for under 5 mg/m^3-years.

Summary--

Data on the association between lung cancer and occupations involving exposure to wood dust are conflicting, and the body of literature is considerably smaller than that for nasal cancer. Negative studies have been reported in England, Sweden, and Finland. However, several case-control and cohort mortality studies in the United States, Canada, and Sweden have reported increases in lung cancer among wood-exposed workers, but there is

not unanimity as to which particular occupational subgroup of woodworkers is affected. In the United States, construction workers, sawmill workers, furniture workers, and carpenters and joiners have each been singled out in individual studies. One explanation for increased lung cancer in construction workers and carpenters and joiners has been potential asbestos exposure to individuals in those occupational classifications. One study implicated smoking as a potential co-carcinogenic factor. There are no concentration-response data.

C. Hodgkin's Disease

Milham (1967) and Milham and Hesser (1967) published a case-control study of 1,549 white males, ages 25 years or more, who had died of Hodgkin's disease (malignant lymphoreticuloadenoma) in New York State during the years 1940-1953 and 1957-1964. Each was matched to a control who died of other causes by age (within 5 years), sex, race, county of residence, and date of death (within 4 weeks). Seventy of the cases were categorized as woodworkers, whereas only 31 controls were similarly classified. This difference was statistically significant ($p<0.001$). The investigators concluded that their findings suggested that death from Hodgkin's disease could be associated with occupational exposure to wood dust.

In an ecological study based on data published by the U.S. Public Health Service, Bureau of the Census, and Department of Agriculture, Spiers (1969) analyzed the occurrence of Hodgkin's disease among whites from 1956-1965 in areas of the United States with various percentages of commercial pine forest land. He calculated ratios for both men and women of the number of deaths due to Hodgkin's disease to the number of combined deaths from Hodgkin's disease, lymphosarcoma, and reticulosarcoma. These ratios served as indices of mortality. Among males in the 37 states east of the Rocky Mountains, Spiers found a significant ($p<0.05$) relationship between this ratio and both the percent of employed white population in the wood, lumber, and furniture industries and the percent of area of commercial forest land covered by pine. He concluded that males in the wood industry in the eastern states were at special risk of developing Hodgkin's disease, conceivably depending upon the extent of exposure to pine pollen, and pointed out that this conclusion would be strengthened greatly if it could be corroborated by data from primary sources.

In Milham's (1974) study of union carpenters and joiners previously described in part B of this section, the overall SMR for Hodgkin's disease was 90, but for the over 60 age group, the SMR was 177 ($p<0.05$). Milham stated that since Hodgkin's disease shows a bimodal age incidence curve, this would indicate that the second mode is etiologically different from the first and may be occupational in origin.

Petersen and Milham (1974) examined Washington State death records for all males 20 years and older for the period 1950-1971. Both case-control and proportional mortality ratio analyses were conducted. For the former, each of the 707 Hodgkin's disease deaths was matched against a control selected randomly from all deaths except those due to Hodgkin's disease or to

accidents and violence. A subset of 158 case-control pairs was also selected from the Washington death file for the period 1965-1970 for a separate interview study. In the main case-control study, 56 of the 707 matched pairs included a case that had a wood-related occupation (carpenters, cabinetmakers, sawyers, and papermakers) when its control did not. Thirty-two pairs had a wood-related control when its case did not. This difference was statistically significant ($p<0.05$). In the interview study, 23 matched pairs had a wood-related case and not a control and 10 pairs had a wood-related control and not a case. This was also significant ($p<0.05$). In the age-specific proportional mortality ratio analysis, there were 56 deaths observed and 34.34 expected. The PMR was 163, which was significant at the $p<0.001$ level. Petersen and Milham concluded that their study within Washington State provided additional support for the hypothesis that woodworking and Hodgkin's disease mortality are associated.

Milham (1976a,1976b) presented an additional age and death-specific proportionate mortality analysis of nearly 300,000 Washington State death records for white males over age 20 for the period 1950-1971. Loggers, plywood workers, and sawmill and other mill workers were not found to have increased PMR's for Hodgkin's disease, but pulp and paper makers (12 cases observed) and carpenters (38 cases observed) had significantly ($p\leq0.05$) increased PMR's of 192 and 162, respectively.

Milham (1983) subsequently updated his Washington State study to include nearly 430,000 male deaths for the period 1950 to 1979 and 25,000 female deaths for 1974-1979. Among wood-exposed occupations, significantly ($p<0.01$) elevated PMR's for Hodgkin's disease were noted among carpenters (PMR 150), millwrights (PMR 324), and paper and pulp mill workers (PMR 194).

Grufferman et al. (1976) conducted an incidence survey of Hodgkin's disease in the Boston Standard Metropolitan Statistical Area during the period 1959-1973. The average annual incidence rate of Hodgkin's disease during the study period was 5.1 per 100,000 for males and 3.2 per 100,000 for females. These figures gave a male/female ratio of 1.6. During the 3 successive 5-year periods that comprised the study period, the average annual incidence rates for men were relatively constant at 4.9, 4.6, and 5.4 per 100,000. For women, the corresponding values were 2.8, 3.4, and 3.5 per 100,000. The percentage increase from the first to the third value for women was 2.5 times that for men. When the age-specific incidence rates for Hodgkin's disease were plotted, the curve for males was generally above that for females. Both curves reached early peaks, of about 7.0 per 100,000 for men, at about 23 years of age. The curves then fell gradually to minima that were fairly constant between ages of about 30 to 50 years. Thereafter, the curves rose to peaks, higher than the early ones, at 80-90 years. Of seven urban occupations, the two with the largest relative risks of Hodgkin's disease were (1) lawyers and judges and (2) woodworkers, although neither of these were significant at the $p=0.05$ level. However, the authors concluded that their findings supported those of Milham and Hesser (1967) and Petersen and Milham (1974) and suggested that woodworkers have a small increased risk of developing Hodgkin's disease.

Greene et al. (1978) examined death certificates in North Carolina counties having a significant proportion of the population employed in furniture making and lumbering. They found 167 white male deaths from Hodgkin's disease. Each of these decedents was matched with two white males who had died of other causes by county of death, age, and year of death. Matched triplet analysis revealed a nonsignificant relative risk of developing Hodgkin's disease of 1.4 (95% CI 0.5-2.3) among occupational groups with wood and paper exposure. The major difference occurred for carpentry and lumbering, which was significant (relative risk 4.2, 95% CI 1.4-12.5). The authors also reported a case of familial occurrence of Hodgkin's disease in three siblings and a first cousin, two of whom had worked for over 12 years with cedar wood products immersed in pentachlorophenol. The authors hypothesized that the association between occupational exposure to wood and Hodgkin's disease may be related to the use of chemical preservatives.

Following the paper of Milham and Hesser (1967), Acheson (1967) studied the occurrence of Hodgkin's disease during the period 1956-1965 among residents served by the Oxford Regional Board. This was the same population studied for cancer of the nasal cavities and paranasal sinuses in furniture makers. High Wycombe had a crude average annual Hodgkin's disease incidence rate of 4.0 per 100,000. The rest of Buckinghamshire had a rate of 2.9 per 100,000 and the remainder of the study area in Oxfordshire, Northamptonshire, and Berkshire had a rate of 2.4 per 100,000. Acheson stated that the differences between the incidences of Hodgkin's disease in High Wycombe and in other parts of the study area were not significant. Using the age distributions of all woodworkers in England and Wales as the basis, Acheson calculated that four woodworkers in the study area would have been expected to develop Hodgkin's disease during the period of the study. Since only three cases of the disease were observed, Acheson concluded that exposure to wood dust in the Oxford area did not contribute appreciably to the incidence of Hodgkin's disease there.

Fonte et al. (1982) reported a case-control study of 207 males and 180 females (mean age 47.3 years, range 10-70) admitted to the University of Pavia Medical Clinic in Italy from January 1972 to December 1979 with a diagnosis of Hodgkin's disease. The controls were 441 males and 330 females (mean age 48.8 years, range 11-78) admitted to an internal medicine unit in Pavia from June 1977 to December 1979. Occupations were coded according to Italian job classification systems. The authors found a statistically significant risk of Hodgkin's disease for workers classified in the wood industry (relative risk 7.2, $p<0.005$).

No increased risk of Hodgkin's disease or cancers of the reticuloendothelial system were reported in the previously described studies by Rang and Acheson (1981), Acheson et al. (1984), Stellman and Garfinkel (1984), and Gallagher and Threlfall (1985).

Summary--

As was the case for lung cancer, there are conflicting data regarding the relationship of Hodgkin's disease to occupational exposure to wood dust. Negative studies have been reported in England, Canada, and the United

States. However, several other U.S. case-control and cohort mortality studies have reported increases in Hodgkin's disease among woodworkers; this excess occurrence seems to be concentrated among carpenters. There are no concentration-response data.

D. Other Cancers

A causal relationship between multiple myeloma and wood dust exposure was first suggested by Brinton et al. (1976). In Milham's (1983) updated survey of occupational mortality in Washington State, a significantly ($p<0.01$) increased risk for multiple myeloma was seen in pulp and paper mill workers (PMR 182); nonsignificant increases were seen in cabinetmakers (PMR 104), carpenters (PMR 118), and plywood workers (PMR 144). In an ecological study of Texas State economic areas, Agu et al. (1980) found a significant ($p<0.05$) positive association between the age-adjusted mortality rate from multiple myeloma and percentage of population employed in carpentry, when control was made for race. Tollerud et al. (1985), in a 1956-1980 death certificate case-control study in North Carolina counties in which at least 1% of the population was employed in furniture and fixture manufacturing, found a nonsignificant excess risk of multiple myeloma, especially among those who died before age 65 and among those born before 1905. A significantly elevated risk was seen in furniture workers born before 1905 who died prior to age 65. Robinson et al. (1986a), in a mortality study of 2,283 plywood mill workers employed for at least 1 year between 1945 and 1955, reported a nonsignificant excess in mortality due to lymphatic and hematopoietic cancer excluding leukemia (SMR 156). Within this classification, the subgroup multiple myeloma showed the greatest excess (SMR 333), but the data were too sparse to distinguish clearly among subgroups.

Decoufle et al. (1977) and Bross et al. (1978) reported the results of a detailed survey of 6,434 white males and 7,815 white females, each having 1 of 22 different kinds of cancer, from among 25,416 patients admitted to the Roswell Park Memorial Institute for Cancer Research, Buffalo, New York between 1956 and 1965. Demographic, social (including smoking habits), medical, and occupational information was obtained prior to diagnosis of disease. Fifty-four occupational/industrial groups were studied for men and eighteen for women. Among males ever-employed, the authors reported a nonsignificant increased age-adjusted relative risk for stomach cancer in carpenters, and for testicular and kidney cancer and myeloma in lumber laborers, and a significant ($p<0.05$) relative risk for esophageal cancer in lumber laborers. Among men employed for at least 5 years, similar increased relative risks were not seen in carpenters and lumber laborers.

Budy and Rashad (1977) studied mortality rates among two groups of carpenters in Hawaii: 227 (Group A) who died during 1949-1951, inclusive, before arsenate-treated wood came into wide use, and 293 (Group B) who died during 1970-1973, inclusive, after the use of arsenate-treated wood became widespread. For both groups, the highest numbers of deaths from all causes and from all cancers occurred during the age decade of 65-74 years. Carpenters were matched with noncarpenters on the next record in sequence

that matched the carpenter in sex, age (within 5 years), and year of death. Age-specific death rates were computed from U.S. census data from 1950 for the first period and from 1970 for the second period. In Group A, there were 60 malignant neoplasms among the carpenters and 30 in the controls; in Group B the corresponding numbers were 67 and 63. This difference was statistically significant ($p<0.05$) in Group A but not in Group B. No significant difference between carpenters and controls in either Group A or B were seen for cancer of the trachea, bronchus, and lung or for cancer of the lymphatic and hematopoietic tissue. The overall relative risk for cancer death was 3.58 for Group A and 2.72 for Group B. For Group A carpenters, the mean annual death rate for ages 16 to 65+ was 1,203 per 100,000, whereas the corresponding figure for Group B was 1,259 per 100,000. For comparison, the mean annual death rates in the general population for the same two periods of time were 559 per 100,000 and 549 per 100,000. This is a consistent and significant ($p<0.05$) increase in total deaths among carpenters. The authors commented that the excess of cancer death was consistent before and during a period of substantial exposure to arsenate-treated wood.

Burkart et al. (1981) described a study designed to investigate the use of hospital records for occupational health surveillance. The authors reviewed the records of 9612 male patients aged 18 and older discharged during 1980 from 4 hospitals in 3 southern Oregon counties. The 3 counties had approximately 12,000 workers employed in the sawmill, millwork, logging, and other wood products industries. Those workers represented 29% of the total labor force. After the elimination of invalid records and patients for whom occupational histories could not be obtained, a total of 7,906 discharges were included in the final study population. The control group consisted of 1,245 of the discharged patients chosen to represent a wide variety of infrequently occurring conditions that would not all be affected to the same degree by a particular exposure. No statistically significant ($p<0.05$) increased risks from any types of cancer were seen, although a nonsignificantly increased risk for leukemia, primarily in workers with 10 or more years employment in the sawmill industry, was noted. To further examine this potential relationship, the authors rechecked the records for secondary diagnoses, since the initial analysis included only primary diagnoses. Eleven new cases of leukemia were discovered, four of whom had worked 10 or more years in the sawmill industry. Combining all the leukemia data resulted in a significant ($p<0.05$) concentration-response relationship between years of sawmill exposure and leukemia. The bulk of the leukemia cases were over age 65.

In their cancer morbidity study among 1,070 white male model and pattern makers employed as of January 1, 1970, in seven Detroit automobile plants, Swanson and Belle (1982) reported significantly ($p<0.01$) increased age-adjusted SMR's for all types of cancer combined (SMR 150) and for colorectal (SMR 286) and salivary gland cancer (SMR 2100). Nonsignificant increases were observed for lymphoma (SMR 301) and melanoma (SMR 323). In subsequent communications, Chovil (1982) and Davies (1983) questioned some of the methodology of the study. The authors responded to each of the questions raised, reemphasized the limitations of their descriptive study, and reiterated their findings.

Swanson et al. (1985) subsequently analyzed 316 white male modelmakers and patternmakers employed by one U.S. automobile manufacturer as of January 1, 1976. Ages of the workers ranged from 18 to 68 years, with 58.5% between 35 and 54 years. There were 10 incident cancers diagnosed between 1976 and 1982, of which 4 (40%) were colon cancer (SMR 487.0, $p<0.01$). The authors indicated that this finding was consistent with earlier studies and recommended cohort and case-control studies to provide more definitive information.

Kurt (1986) questioned whether the increased rate of colon cancer reported by Swanson et al. (1985) could be due to either acrylonitrile exposure from plastics used in modelmaking or the workers' sedentary lifestyle. Both factors had previously been reported to be associated with an increase in colon cancer. Swanson and Burrows (1986) replied that their study was a descriptive analysis for which exposure data and levels of worker activity were not available, and that future studies should incorporate these potential risk factors.

In their 1972-1978 proportional mortality study of 1,257 pattern and model makers, Robinson et al. (1980) reported statistically significant excess proportions of deaths due to both colon (PMR 167, $p<0.002$) and brain cancer (PMR 211, $p<0.03$). Among members of the predominantly wood shop locals, significant excesses of colon cancer (PMR 163, $p<0.01$) and leukemia (PMR 200, $p<0.03$) were found.

In a cohort of 3,572 pulp and paper mill workers employed for at least one year between 1945 and 1955, Robinson et al. (1986b) reported statistically nonsignificant excesses of death due to lymphosarcoma and reticulosarcoma (SMR 169, 90% CI 92-287) and to stomach cancer (SMR 123, 90% CI 78-185).

Franco and Fonte (1983) expanded the previously described case-control study of Fonte et al. (1982) to include 347 males and 322 females admitted to the University of Pavia Medical Clinic, Italy, between January 1969 and December 1980 and diagnosed as malignant lymphomas (387 cases of Hodgkin's disease and 282 cases of non-Hodgkin's lymphoma). Among those employed in the wood industry, there were 14 cases and 8 controls with malignant lymphoma. This difference was significant at the $p<0.001$ level.

Stellman and Garfinkel (1984), in their previously reported review of woodworkers enrolled in the American Cancer Society's prospective epidemiologic study, reported significantly increased mortality ratios for stomach cancer (SMR 149, $p<0.01$) and bladder cancer (SMR 142, $p<0.05$) among all woodworkers, and for stomach cancer (SMR 170, $p<0.01$) in carpenters and joiners. They also noted a significantly decreased mortality ratio for colorectal cancer (SMR 75, $p<0.05$) in all woodworkers.

In their analysis of British Columbia death statistics, Gallagher and Threlfall (1985) and Svirchev et al. (1986) reported a significant excess of several types of cancer in male woodworkers. These included stomach cancer (PMR 144, $p=0.0001$), lymphosarcoma and reticulum cell sarcoma combined (PMR 159, $p=0.006$), and all types of cancer (PMR 112, $p=0.0007$) for the general woodworking category; stomach cancer (PMR 155, $p<0.001$), lymphosarcoma and

reticulum cell sarcoma combined (PMR 165, $p<0.05$), and all cancers combined (PMR 114, $p=0.0006$) in carpenters; pleural cavity (PMR 6553, $p=0.0009$), prostate (PMR 569, $p=0.0042$), and all cancers combined (PMR 194, $p<0.0001$) in cabinetmakers and furniture makers; and lymphosarcoma and reticulum cell sarcoma combined (PMR 275, $p<0.05$) in sawmill workers. Siemiatycki et al. (1986), in their case-control study of cancer patients in Montreal, reported an increased odds ratio of 1.7 (95% CI 1.1-2.8) for stomach cancer and wood dust exposure.

Preliminary results of the NCI cohort mortality study of members of the United Furniture Workers of America (Miller 1987) indicated reduced overall cancer mortality among white males in the wood furniture and other wood products industries, although a slight excess (p value not provided) of bone cancer was found.

Summary--

There are insufficient and inconclusive data regarding the relationship between occupational exposure to wood dust and cancers other than nasal, lung, and Hodgkin's disease. Multiple myeloma, stomach and colorectal cancer, and lymphosarcoma are among the cancers most often mentioned in the available cohort mortality and morbidity studies as occurring in occupations in which wood dust exposure occurs.

VII. INDUSTRIAL HYGIENE MEASUREMENTS AND CONTROLS

This section contains a compilation of wood dust concentrations measured in the workplace (Table 6 and Summary Tables 7-12) and a brief discussion of wood dust control technology. Operations that generate wood dust can be classified into three general categories: sawing, sanding, and milling. The latter includes such operations as planing, chipping, routing, moulding, jointing, shaping, and boring. Descriptions of these operations are provided in Holliday et al. (1986), NSC (1980), and Hjorth and Holtrop (1966). It should be noted that the amount of wood dust generated and the particle size distribution will vary depending on the particular operation (e.g., sanders, shapers, and routers have been shown to give off more dust than other types of woodworking machinery [Hampl 1982]) as well as on the type of wood (Huebener 1987) and the water content of the wood (Holliday et al. 1986).

The various values in Tables 7-12 are presented only to provide the reader with a general idea of the ranges of wood dust concentrations that have been encountered in the workplace. They should not be generalized for epidemiological analysis of health effects related to wood dust exposure except for cases in which effects have been documented at specific dust concentrations. Unless otherwise noted, sampling and analyses were done by the gravimetric measurement of dust collected on a filter. Where data are missing, they were not given by the investigator. Wood type and the mention of local exhaust ventilation (where provided) are noted.

Good work practices, process substitution, and properly designed engineering controls can be used to minimize worker exposure to wood dust. A survey of lumber mills and furniture manufacturing plants (Anonymous 1979) revealed that work practices included (1) administrative controls, such as training programs and labeling, and standard operating procedures (e.g., housekeeping and cleanup programs); (2) the use of personal protective equipment, such as eye and face protection (e.g., safety glasses, goggles, and face shields), respirators, and protective clothing (e.g., gloves, aprons, long sleeved shirts, and regular trousers); and (3) proper use of woodworking machinery. NIOSH has published general health and safety work practice guidelines for sawmills and planing mills (NIOSH 1978), plywood and veneer mills (NIOSH 1977), wooden furniture manufacturing (NIOSH 1975), prefabricated wooden building manufacturers (NIOSH 1976b), and millwork shops (NIOSH 1976a). The National Safety Council (NSC 1970-1986; NSC 1980) and the American National Standards Institute (ANSI 1975) have also published safety guidelines for various types of woodworking machinery.

Substitution of processes that eliminate or reduce wood dust generation has also been reported (Holliday et al. 1986). These processes include peeling

and slicing to produce veneer sheets, high-energy water jets for cutting and machining, and laser cutting. The first two processes are limited in their application; the third is still experimental.

The primary engineering control for wood dust is local exhaust ventilation (Hampl and Johnston 1985). In addition to reducing worker exposure to wood dust, ventilation also reduces potential safety hazards due to fire and explosion (NFPA 1980). ACGIH (1986) has made general recommendations for local exhaust system configurations and ventilation rates for various types of woodworking equipment, including band, table, swing, and rip saws; disc, belt, drum, and swing arm sanders; single and double planers; routers; jointers; molders, matchers, and sizers; and lathes. ACGIH recommended exhaust volumes are shown in Table 13.

Local exhaust systems have been specifically designed for belt sanders (Hampl and Johnston 1985), shapers (Huebener 1987), and circular table saws (WCBBC 1982). The belt sander exhaust ventilation system utilizes a narrow low-volume, high-velocity slot hood located between the belt surface and the worktable, and a jet stripper consisting of a series of small-diameter nozzles located inside a driven pulley hood opposite the operator station. By creating high velocity air streams, the jet stripper destabilizes the wood-dust-containing boundary air layer surrounding the belt, and the hood collects the dust from the turbulent air before the dust can become reentrained in the boundary layer. With either the slot hood or the jet stripper operating, reductions in breathing zone wood dust concentrations averaging 50% were achieved. With both parts of the ventilation system operating, the average reduction increased to 80%. The shaper exhaust system consists of a flexible hood that permits the wood to pass through but prevents the escape of the wood dust. Dust reductions ranging from 16:1 to 120:1 were observed in laboratory tests. The circular table saw exhaust system consists of a conventional exhaust hood beneath the saw table and a second, narrower hood covering the saw blade.

An example of the use of area ventilation to control wood dust levels is provided by Bullock (1979). As part of a study examining the recirculation of industrial exhaust air, the author evaluated the ventilation system of a furniture manufacturing plant. The system consisted of five large blowers (43,850 cubic feet per minute [cfm]) connected to a dust collection system of 21 modular bag filter compartments. The performance of the dust collector, which was determined by measuring the dust concentrations in the inlets and return streams, exceeded 99.5% during the survey. There was virtually no difference in total dust concentrations before and after recirculation of the exhaust air.

Table 6.--Wood dust concentrations measured in the workplace in various studies

Study description	Miscellaneous details	Wood dust concentrations			
			TWA's, mg/m^3		
	Equipment	Number of Samples	Range	Mean	Median
Kubis (1963)	Belt sander (1961)	5	3.6-65.0	27.9	23.0
Cabinetmaking	Belt sander (1962)	5	4.8-34.2	19.2	18.4
Czechoslovakia (Nitra)					
1 plant		10	3.6-65.0	23.6	20.7
1961-1962					
Area samples/total dust					

NOTE: In 1961, five sanders with local exhaust ventilation rates ranging from 0-285 m/min were studied; dust concentrations were inversely proportional to ventilation rates. In 1962, hard oak dust concentrations were determined as Kubis varied the local exhaust ventilation rate (0-372 m/min), grain size of the abrasive (30-80), dimensions of the push-lever, and the belt width (12-15 cm) on a single sander. Up to 95% of the dust particles were <5 um with most 2-3 um in size. Sampling and analysis methodologies were not provided.

		Number of	TWA's, mg/m^3		
	Operation	Samples	Range	Mean	Median
Hanslian and Kadlec (1964)	Sanding (machine)		?-235		
Furniture	Sanding (hand)		?-220		
Czechoslovakia	Sawing		?-175		
1 plant	Turning		?-135		
1964	Boring		?-125		
Area samples/total dust					

NOTE: Timber species used were ebony, beech, monsonia, mahogany, and larch. Dust concentrations during a full shift in a sanding shop (2-4 belt sanders) reached on the average 40 mg/m^3. Analysis by optical microscopy indicated that 5-70% of the dust particles collected were <5 um in size.

(Continued)

	Department	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Paustovskaya et al. (1968)	Sanding	2	16.7-25.5	21.1	21.1
Furniture (mahogany)	Hand finishing	1	23.5	23.5	23.5
U.S.S.R. (Kiev)					
1 plant		3	16.7-25.5	21.9	23.5
1968					
Personal samples/total dust					

NOTE: A total dust concentration of 18.3 mg/m^3 was found 1 m from the dust-producing processes; concentrations of 5-6 mg/m^3 were measured in remote areas of the shop. The industrial hygiene sampling was by the Chernovsty Municipal Medical-Epidemiological Station Laboratory; sampling and analysis methodologies were not provided.

	Facility	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Imbus (1978)	Plant 1 (1971)	4	0.6-3.3	1.6	1.2
Furniture	Plant 2 (1971)	7	0.9-8.9	3.5	2.5
U.S.A. (North Carolina)	Plant 3 (1971)	6	1.1-5.3	2.5	2.4
6 plants (1 company)	Plant 4 (1971)	3	2.2-7.9	4.4	3.1
1971-1973	Plant 5 (1971)	4	2.8-15.4	6.6	4.1
Area samples/total dust	Plant 6 (1973)	12	0.1-4.9	1.3	0.9
		36	0.1-15.4	2.8	2.2

NOTE: These data summarize wood dust sampling conducted by a large furniture company. The highest total wood dust concentration (15.4 mg/m^3) was found at an automatic polisher where compressed air-blow off was used extensively; it was recommended that this practice be eliminated. The concentration of respirable dust collected at a sanding operation in Plant 6 was 0.7 mg/m^3.

(Continued)

Soderqvist and Ager (1975) Sawmill Sweden 1972-1973	NOTE: Based on 36 personal samples, it was estimated that 80% of 689 workers had TWA total dust exposures <2 mg/m^3, 18% to 2.1-5.0 mg/m^3, and 2% to >5.0 mg/m^3. This data was part of a multidisciplinary survey of Swedish sawmills. Concentrations over 5 mg/m^3 were measured in sawing, packaging, and trimming operations. The chipper controllers, cleaning personnel, and slat controllers had the highest wood dust exposures. [Summary details from IARC 1981]
Otto (1973) Woodworking German Democratic Republic 27 shops 1973 Area samples/total dust	NOTE: This is a summary of wood dust sampling conducted by the Erfurt District Inspectorate for Occupational Health. The following types of wood were being processed: mansonia, makore, obeche, limba, sapeli, afromosia, okume, and zebrano. Of the 102 total dust samples collected, 14% were <5 mg/m^3, 24% were from 5-9 mg/m^3, 24% were from 10-19 mg/m^3, and 38% were >20 mg/m^3. The samples were collected mainly during the use of belt sanders on wood veneers.

(Continued)

	Operation	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Hounam and Williams (1974)	Band sawing				
Furniture	(MMAD=11.5 um)	6	1.0-7.3	4.3	5.3
England (High Wycombe)	Planing (MMAD=9.2 um)	9	1.8-10.9	5.0	3.1
5 plants	Routing (MMAD=10.0 um)	6	1.8-8.6	4.1	3.6
1973	Spindle moulding				
Personal samples/total dust	(MMAD=10.0 um)	8	1.5-8.4	5.1	5.1
	Sanding (MMAD=8.4 um)	9	2.0-22.6	7.2	3.6
	Assembly (MMAD=7.6 um)	10	2.1-25.5	7.5	5.2
	Turning (MMAD=11.5 um)	2	4.6-12.5	8.6	8.6
		50	1.0-25.5	5.9	4.4

NOTE: These results are among the first to be reported for England. Chairs and other types of furniture were made of beech, elm, mahogany, walnut, and veneered chipboard at four of the five plants; cupboards or cabinets were made of veneered chipboard at the fifth plant. The machining operations at all the plants surveyed were considered to be well ventilated by local exhaust systems. The size distributions, including the mass median aerodynamic diameter (MMAD), of airborne dust particles near these operations were measured using a 4-stage cascade centripeter to collect 37 samples. Most of the dust was found on the second stage of the centripeter, where dust of 4.15-13.65 um was collected. Less than 25% of the mass of the dust collected was from particles <5 um in diameter.

(Continued)

	Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Rosensteel (1974)	Roundtable shaper operator:				
Cabinets	Total dust	2	8.6-8.9	8.8	8.8
U.S.A. (North Carolina)	Respirable dust	2	0.02-0.4	0.2	0.2
1 plant					
1974					
Personal samples					

NOTE: This is a NIOSH health hazard evaluation (HHE) conducted at a plant that produced television and stereo cabinets. The roundtable shapers inscribed a predetermined pattern in particle board. The local exhaust ventilation on the shapers was of only limited effectiveness and improvement was recommended. A 10-mm nylon cyclone was used as a preselector in the respirable dust sampling.

	Operation	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Andersen et al. (1977)	Sanding	41		14.3	
Furniture	Drilling/planing/sawing	27		5.2	
Denmark (Aarhus county)					
8 plants		68	1.3-77.5	10.7	
1974-1975					
Personal samples/total dust					

NOTE: These data are part of a study of nasal mucociliary clearance rates. Teak was the most frequently processed wood, followed by oak, chipboard, palisander, mahogany, jakaranda, beech, ramin, motine, Masonite, and pine. Of the dust samples collected, 63% were >5 mg/m^3 and 28% were >10 mg/m^3. It was also determined that 33% of the mass of the dust was from particles <5 um, 41% was from 6-10 um, 11% was from 11-15 um, and 15% was from particles >16 um in size; methodology for particle sizing was not given.

(Continued)

	Exposure:Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Kominsky and Anstadt (1976)	Total dust:				
Wood component fabrication	Saw operator	17	0.7-416	38.1	1.7
U.S.A. (Ohio)	Router/groover operator	5	1.2-48.5	12.5	1.7
1 plant					
1975		22	0.7-416	32.3	1.7
Personal samples	Respirable dust:				
	Saw operator	13	0.07-4.4	0.7	0.4
	Router/groover operator	3	0.3-0.8	0.5	0.4
		16	0.07-4.4	0.6	0.4

NOTE: This is a NIOSH HHE conducted at a plant involved in the fabrication of component parts (e.g., book case back panels, clock faces, and television cabinets). Component parts were fabricated mainly from Masonite hardboard; however, particle board, fiberboard, and flake board were also used. Most of the boards used were comprised of redwood, oak, elm, and Douglas fir; secondary woods included ash, cottonwood, hickory, magnolia, birch, beech, pecan, holly, maple, sweet gum, sycamore, walnut, willow, and poplar. The highest dust concentrations found were for a multi-blade rip saw helper who had an average exposure over 4 days of 153 mg/m^3 (range 1.4-416 mg/m^3) and for a router operator who had a 1-day exposure of 48.5 mg/m^3; it was recommended that the existing local exhaust ventilation for these two operations be improved. All other full-shift total dust samples were <7 mg/m^3. Four total dust samples of 10-18 min duration collected during blowdown of dust bins averaged 48.2 mg/m^3 (range 11.2-118 mg/m^3). A 10-mm nylon cyclone was used as a preselector in the respirable dust sampling. The MMAD of particles collected using a 6-stage cascade impactor was >10 um.

(Continued)

	Operation	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Levy (1976)	Sawing	2	1.1-3.4	2.2	2.2
Woodworking	Sanding	2	4.3-6.8	5.5	5.5
U.S.A. (New York)	Sawing/sanding	2	0.9-5.0	3.0	3.0
1 shop	Sawing/sanding/planing	2	2.3-24.2	13.2	13.2
1976					
Personal samples/total dust		8	0.9-24.2	6.0	3.9

NOTE: This is a NIOSH HHE conducted at the woodworking area of an art school. No local exhaust ventilation was provided on any of the woodworking machines. Sampling times varied from 45-250 min in duration. A 6-hr general area sample for total dust indicated a concentration of 2.5 mg/m^3.

	Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Gunter (1977a)	Mill worker	1	4	4	4
Furniture	Sander in specialty department	1	90	90	90
U.S.A. (Utah)					
1 plant		2	4-90	47	47
1976					
Personal samples/total dust					

NOTE: This is a NIOSH technical assistance study conducted at a small plant that produced office furniture such as desks, tables, and file cabinets. It was recommended that engineering controls be utilized to eliminate the excessive wood dust exposure to the sander in the specialty department.

(Continued)

Edwards et al. (1978)
Wood products
U.S.A. (Washington)
1 company
1976
Personal samples

Exposure:Department	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Total dust:				
Shake Mill (western red cedar)	85	0.06-31.9	4.7	1.6
New Planer Mill (fir/hemlock)	59	0.1-16.7	1.3	0.3
Old Planer Mill (fir/hemlock)	6	0.2-54.8	18.1	1.0
Bark Plant (fir/alder)	5	1.2-8.3	4.0	2.4
Presto-log Plant (fir/alder)	5	0.6-4.6	1.7	0.9
Silvacel Plant (alder)	5	2.3-21.5	13.5	15.6
Plywood Plant (fir/cedar)	4	0.3-1.3	0.6	0.5
	169	0.06-54.8	4.0	
Respirable dust:				
Shake mill (western red cedar)	39	0.01-1.2	0.2	0.1
New planer mill (fir/hemlock)	23	0.07-0.3	0.2	0.2
Old planer mill (fir/hemlock)	1	0.3	0.3	0.3
Bark plant (fir/alder)	1	0.3	0.3	0.3
Presto-log plant (fir/alder)	2	0.2-0.2	0.2	0.2
Silvacel plant (fir/cedar)	2	0.5-0.6	0.5	0.5
	68	0.01-1.2	0.2	

NOTE: This is a NIOSH HHE conducted at a diversified wood products company. In the Old Planer Mill, a chipper operator had a total dust exposure of 51.4 mg/m^3 and the set-up man 54.8 mg/m^3; based on lower results obtained on similar jobs in the New Planer Mill (which has environmental controls) it was judged possible to also reduce these dust concentrations. In the Presto-log plant, sawdust was compressed into fireplace logs. In Silvacel production, wood chips were ground into fibers, passed through a dryer, and bagged; due to the high concentration of total dust found in this area, it was recommended that engineering controls be installed. A 10-mm nylon cyclone was used as a preselector in the respirable dust sampling. The following average particle size distribution was found in the Shake Mill from 4 samples collected: 39% <10 um, 23% from 10-20 um, and 38% >20 um in size. In seven samples collected in the New Planer Mill, 47% of the particles were <10 um, 25% were 10-20 um, and 28% were >20 um in size. Methodology for particle sizing was not given.

(Continued)

	Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Gunter (1977b) Wood pattern making U.S.A. (Colorado) 1 shop 1977 Personal samples/total dust	Pattern maker	6	0.6-8.0	3.1	2.4

NOTE: This is a NIOSH HHE conducted at a plant that made wood patterns for a foundry. Local exhaust ventilation was available at each work station in the pattern shop.

	Operation	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Friar (1978) Furniture England 7 plants 1978 Total dust	Sanding (machine)	45	1-75	6	
	Assembly	35	2-41	8	
	Band sawing	13	2-127	18	
	Other sawing	21	2-35	8	
		114	1-127	8	

NOTE: These data summarize wood dust sampling (personal and area) conducted by the Factory Inspectorate of the Health and Safety Executive in a traditional furniture manufacturing area. Out of 114 samples collected, 48% were >5 mg/m^3; belt sanding and sanding during assembly were the dustiest operations. Almost all the sanding machines and saws were provided with local exhaust ventilation.

(Continued)

	Department	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Apol (1979a)	Sawmill (includes chipper)	23	0.03-0.8	0.4	0.3
Sawmill/planer mill	Planer mill	15	0.2-0.5	0.3	0.3
U.S.A. (South Dakota)					
1 plant		38	0.03-0.8	0.3	0.3
1978					
Area samples/total dust					

NOTE: This is a NIOSH HHE conducted at a facility processing pine. Area samples were fairly representative of the workers' exposures to wood dust.

	Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Apol (1979b)	Cabinetmaker	4	2.0-3.0	2.4	2.3
Cabinet shop	Panel saw operator	2	0.9-1.6	1.3	1.3
U.S.A. (Colorado)	Rip saw/planer operator	3	2.5-36.1	17.4	13.7
1 shop	Sander/shaper/rip saw/planer	3	1.9-11.9	6.4	5.2
1978					
Personal samples/total dust		12	0.9-36.1	6.9	2.4

NOTE: This is a NIOSH HHE conducted at a custom cabinet shop using oak, plywood veneer, and particle board. A worker who operated a power rip saw most of the shift had the highest exposure (i.e., 36.1 mg/m^3); it was recommended that a new hood be installed under this saw.

(Continued)

	Facility:Operation	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Weaver (1979)	Plant 1: Rip/cutoff sawing #1		8.4-77.2		
Furniture	Rip/cutoff sawing #2		16.1-60.1		
U.S.A. (North Carolina)	Boring		4.8	4.8	
7 plants	Drum sanding		2.5	2.5	
1978	Plant 2: Belt sanding		27.4-56.6		
Personal samples/total dust	Plant 3: Routing #1		3.1-12.3		
	Routing #2		5.8-15.5		
	Plant 4: Double trim sawing		6.0	6.0	
	Plant 5: Routing		5.7	5.7	
	Plant 6: Belt sanding		1.6-3.6		
	Plant 7: Hand shaping		15.3-21.1		
	Edge sanding		2.4-20.7		
			2.4-77.2		

NOTE: These data summarize wood dust sampling conducted by the North Carolina Department of Labor. Area samples for total dust collected near the sawing operations in Plant 1 ranged from 1.6-5.6 mg/m^3. Area samples indicated a respirable dust concentration of 2.1 mg/m^3 near cutoff sawing in Plant 1 and 1.5 mg/m^3 for a router operator at Plant 5; a 10-mm nylon cyclone was used as a preselector for these samples. In many instances, little or no local exhaust ventilation was available in the facilities surveyed.

(Continued)

Whitehead et al. (1981b)
Plywood/furniture
U.S.A. (Vermont)
1978
12 plants
Area samples/total dust

Facility:Area	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Plywood (hardwood):				
Veneer lathe/clipper	3	0.2-0.6	0.5	0.6
Dryer	4	0.1-0.4	0.3	
Dry veneer handling	4	0.2-0.5	0.3	
Edge sawing/sanding	6	0.9-2.3	1.4	
Machining	6	0.7-3.2	1.9	
Assembly	2	0.3-0.6	0.5	0.5
Plywood (hardwood)	25	0.1-3.2	1.0	
Furniture (hardwood):				
Rough milling	7	0.2-2.6	0.8	
Detailed milling (finishing)	9	0.2-6.3	1.8	
Sanding	12	1.4-11.4	4.5	
Assembly	3	1.1-2.1	1.5	1.3
Furniture (hardwood)	31	0.2-11.4	2.6	
Furniture (softwood):				
Rough milling	5	0.2-1.1	0.6	
Detailed milling (finishing)	9	0.3-4.3	1.6	
Sanding	13	0.6-14.3	3.2	
Assembly	2	2.5-3.1	2.8	2.8
Furniture (softwood)	29	0.2-14.3	2.2	
Furniture (hardwood & softwood)	60	0.2-14.3	2.4	

NOTE: These data are part of a study of the respiratory health of woodworkers. Fifteen samples collected with a 6-stage cascade impactor indicated that relatively little dust on a mass basis was present in the smallest size ranges.

(Continued)

	Department	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Chan-Yeung et al. (1980) Sawmill Canada (British Columbia) 1 mill 1979 Personal samples/total dust	Sawmill (including permanently assigned maintenance staff)	71	<0.1-2.7	0.5	

NOTE: These data are part of a respiratory health effects study at a pulp and paper mill.

	Department	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Enright (1980) Wood model making U.S.A. (Michigan) 1 shop 1980 Personal samples/total dust	Research & Safety Model Shop	10	0.2-3.4	0.9	0.4
	Wood Mill	10	1.2-10.2	4.7	4.3
	Model Shop/Body Shop	20	0.1-22.4	2.4	0.8
	Hardware & Styling Studio	12	0.1-2.0	0.5	0.2
	Die Engineering Model Shop	15	0.2-8.3	1.7	0.4
	Crating and Omni-milling	8	0.1-0.6	0.2	0.2
	Plastics Shop	6	0.1-0.9	0.4	0.1
		81	0.1-22.4	1.7	0.5

NOTE: These data are a summary of wood dust sampling conducted by an automobile producer. Three of the workers monitored had exposures greater than 10 mg/m^3, i.e., a shaper operator in the Wood Mill (10.2 mg/m^3), a model maker performing sanding by hand (11.0 mg/m^3), and a model maker using a powered orbital sander (22.4 mg/m^3). Based on sampling with a 10-mm nylon cyclone, only 2-5% of the mass of the particulate in the two samples collected was respirable.

(Continued)

	Operation	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Salisbury (1981) Woodworking U.S.A. (Kentucky) 1 shop 1980 Personal samples/total dust	Sanding/grinding/lathe	4	15.6-45.8	29.1	27.5

NOTE: This is a NIOSH technical assistance study conducted to help evaluate ventilation at the art studios of a university. The dusts generated were from maple and pine. Typical exposures per day (and sampling times) were 40-67 min. Computed as 8-hr TWA's, and assuming zero exposure for the time not sampled, the highest exposure was 5.7 mg/m^3. Most of the dust was generated by portable disk and belt sanders.

	Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Boiano (1981) Wood model making U.S.A. (Indiana) 1 shop 1981 Personal samples/total dust	Pattern maker	2	0.3-0.8	0.6	0.6

NOTE: This is a NIOSH HHE conducted at a truck wood model making shop.

(Continued)

	Facility:Department	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Al Zuhair et al. (1981) Furniture England 2 plants 1981 Personal samples/total dust	Plant 1:				
	Sawmill (MMAD=17.3 um)	75		3.5	
	Assembly (MMAD=18.0 um)	4		0.7	
	Plant 1	79		3.4	
	Plant 2:				
	Machine Floor (MMAD=9.3 um)	43		1.7	
	Cabinet Shop (MMAD=12.5 um)	71		8.1	
	Plant 2	114		5.7	
	Plants 1 and 2	193		4.1	

NOTE: These data are part of a study of ventilatory function in workers exposed to wood dust. Plant 1 made cabinets and cupboards from veneered chipboard; Plant 2 produced chairs, tables, and cabinets from limba, beech, ash, and to a lesser extent, from mahogany, oak, and ramin. Average concentrations of total dust determined from area samples were 1.4 mg/m^3 in the Saw Mill (6 samples) and 0.8 mg/m^3 in Assembly (2 samples) in Plant 1; and 0.7 mg/m^3 on the Machine Floor (16 samples) and 4.4 mg/m^3 in the Cabinet Shop (13 samples) in Plant 2. Respirable dust sampling (as measured by a Hexhlet) indicated average concentrations in Plant 2 of 0.19 mg/m^3 (4 samples) and 0.44 mg/m^3 (4 samples) on the Machine Floor and in the Cabinet Shop, respectively. MMAD's were estimated from 24 samples collected with a 7-stage cascade impactor.

(Continued)

	Type of Wood Processed	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Morey (1982)	Basswood	6	0.7-2.1	1.3	1.1
Sawmill (West Virginia)	Poplar	18	0.7-3.1	1.4	1.1
U.S.A.	Soft maple	9	0.8-4.9	2.2	2.2
1 mill	Red oak	5	1.0-5.5	3.2	2.7
1981-1982	White oak	14	1.0-10.6	3.7	2.7
Area samples/total dust	Cherry	3	6.4-8.1	7.2	6.9
		55	0.7-10.6	2.6	2.0

NOTE: These data are from a NIOSH survey conducted at a small sawmill where different species of hardwood (as available in the yard) were processed. Samples were collected at three locations (the booth where the headsaw was operated; and at two sites where workers were observed transiently, across from the headsaw booth and on a catwalk over the edger). Moisture content of the wood being processed was significantly related to the dust concentrations at the three plant locations monitored.

(Continued)

	Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
McCammon et al. (1985)	Model maker (soft/hardwood)	23	0.2-8.3	0.8	
Wood model making	Model maker (softwood)	4	0.2-0.5	0.3	
U.S.A. (Michigan)	Model maker (hardwood)	12	0.2-1.3	0.6	
3 shops	Sweeper	5	0.1-6.1	1.6	
1981-1982	Shaper operator	7	0.3-13.9	2.7	
Personal samples/total dust	Plastic shop worker	3	0.03-0.7	0.4	0.6
	Multi-axis machine operator	4	0.2-1.0	0.5	
		58	0.03-13.9	1.0	

NOTE: This is a summary of the NIOSH characterization of exposures in automotive wood model shops. Control of dust emissions from shapers was deemed to be very difficult. The average total dust concentration of 29 area samples was 0.9 mg/m^3 (range 0.05-17.5 mg/m^3). Inconsistent results were found (i.e., 0.1-180% respirable dust) when sampling with a 10-mm nylon cyclone. It was hypothesized that since wood dust can acquire a static charge it may "stick" to a nylon cyclone. MMAD averaged 7.7 um (range 5.2-10 um) in samples collected in nine different areas with a 9-stage cascade impactor, with 18-61% of the dust being respirable. 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-inch stainless steel cyclone.

	Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Ellenbecker and Wegman (1983)	Sander	3	0.5-0.9	0.6	0.5
Woodworking					
1982					
1 shop					
Personal samples/respirable dust					

NOTE: This is a NIOSH HHE conducted at a grandfather clock factory. A 10-mm nylon cyclone was used as a preselector in the respirable wood dust sampling. Three area samples for total dust collected near the woodworking operations averaged 2.0 mg/m^3 (range 0.2-4.6 mg/m^3). Local exhaust ventilation appeared to be in good operating condition.

(Continued)

	Facility	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Jones and Smith (1986)	Plant 1 (1983)	36	1.0-6.2	2.6	
Furniture	Plant 2 (1983)	15	0.7-4.1	2.1	
England (High Wycombe)	Plant 3 (1983)	22	0.9-13.7	2.9	
7 plants	Plant 4 (1983)	9	1.4-8.8	4.6	
1976-1983	Plant 5 (1983)	39	1.1-6.8	2.8	
Personal samples/total dust	Plant 6 (1983)	29	0.3-18.3	2.2	
	Plant 7 (1983)	60	0.5-53	8.1	
	1983 Results	210	0.3-53	4.3	
	Comparison by operation				
	Machine sanding: 1976-1977	36	2.1-6.3*	3.7	
	1983	49	0.7-53	5.7	
	Hand sanding: 1976-1977	33	5.8-32*	16.9	
	1983	42	0.5-27.2	6.9	
	Sawing: 1976-1977	29	4.7-13.1*	7.4	
	1983	49	1.0-30.3	3.4	
	Other cutting: 1976-1977	44	2.0-10.8*	6.9	
	1983	70	0.3-8.8	2.3	
	1976-1977 Results	142	2.0-32*	8.5	
	1983 Results	210	0.3-53	4.3	

*Individual plant averages

NOTE: These data are a summary of wood dust sampling conducted by the Health and Safety Executive. Tables and chairs were produced mainly from beech, ash, and elm at six of the plants; sideboards, wardrobes, and loudspeaker cabinets were manufactured at three of the plants from elm, ash, veneered chipboard, or medium density fiberboard. Almost all woodworking machines in the plants surveyed in 1983 were fitted with some form of local exhaust ventilation; most of the hand

(Continued)

sanding operations were carried out in the open with no exhaust ventilation for dust control. The high average dust concentration in Plant 7 (i.e., 8.1 mg/m^3) reflected heavy and prolonged sanding. Overall, narrow belt sanders created the greatest dust concentrations (8.5 mg/m^3, range 1.2-20.3 mg/m^3), followed by hand sanding (6.9 mg/m^3, range 0.5-27.2 mg/m^3) and drum, bobbin, and brush sanders (4.4 mg/m^3, range 0.5-51.4 mg/m^3). The MMAD of the dust collected with an impactor during the machine sanding of mahogany was 9 um with about 54% of the mass of the dust in the range 4-10 um. A comparison between the results of the 1983 survey and an earlier one (1976-1977) indicated that there had been a significant decline in personal exposures to wood dust in the furniture plants surveyed.

	Equipment	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
McCawley (1983)	Router	5	1.7-8.1	4.0	3.4
Signs/plaques	Sander (large)	8	1.0-5.6	2.9	2.6
U.S.A. (New York)	Sander (small)	5	1.7-4.5	2.9	2.0
1 shop					
1983		18	1.0-8.1	3.2	2.6
Area samples/total dust					

NOTE: These data are from a NIOSH research study that evaluated different wood dust sampling methods at a sign shop. Wood processed included western red cedar and laminated pressed wood. All airborne dust samples were collected using a 4-stage cascade impactor. It was determined that 45-60% of the mass of the dust was from particles <3.5 um, 30-35% from 3.5-20 um, and that 5-20% of the dust was from particles >20 um in size.

(Continued)

	Department:Operation	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Lindroos (1983)	Pretreatment:				
Woodworking	Unloading wood	10	0.5-28.0	4.3	4.3
Finland	Sawing	11	0.3-2.0	0.8	0.8
1 shop	Other machines	6	0.4-1.0	0.8	0.9
1983					
Personal samples/total dust	Pretreatment Department	27	0.3-28.0	2.1	
	Woodworking:				
	Planing	20	0.02-1.8	0.4	0.3
	Other machines	11	0.2-4.7	1.1	0.5
	Woodworking Department	31	0.02-4.7	0.7	
	Pretreatment and Woodworking	58	0.02-28.0	1.4	

NOTE: These data are part of a study that assessed working conditions when impregnated wood was used to produce windows and doors. Concentrations of total dust determined from area samples were 0.6 mg/m^3 (range 0.2-1.0 mg/m^3) in the Pretreatment Department (9 samples) and 0.3 mg/m^3 (range 0.1-0.4 mg/m^3) in the Woodworking Department (13 samples). Seven respirable dust samples collected in the Pretreatment Department averaged 0.16 mg/m^3 (range 0.14-0.19 mg/m^3); 6 respirable dust samples collected in the Woodworking Department averaged 0.14 mg/m^3 (range 0.11-0.20 mg/m^3). A cyclone was used as a preselector in the respirable dust sampling. The number percentage of dust particles <5 um in size in 15 samples collected in processing areas averaged 97.8% (range 93.6-100.0%). Particle sizing was done by optical microscopy at 500X magnification.

(Continued)

	Job Title	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Daniels and Anderson (1983)	Wood shop operator	3	2.0-3.3	2.6	2.5
Wood model making	Model maker	4	0.5-4.0	1.7	1.2
U.S.A. (Michigan)					
1 shop		7	0.5-4.0	2.1	2.0
1983					
Personal samples/total dust					

NOTE: This is a NIOSH HHE conducted at an automotive wood model shop. The majority of the woodworking machines were equipped with local exhaust ventilation.

	Job Title:Operation	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Crandall and Hartle (1984)	Carpenters in assembly:				
Boat building	Prefab	8	0.8-16.2	4.6	2.9
U.S.A. (Maine)	Main line	19	0.3-2.8	1.5	1.3
1 plant					
1983		27	0.3-16.2	2.4	1.7
Personal samples/total dust					

NOTE: This is a NIOSH technical assistance study conducted in the assembly building of a fibrous glass-reinforced boat plant. Two area total dust samples collected in prefab were 0.4 and 1.8 mg/m^3. All large woodworking machines had local exhaust ventilation.

(Continued)

	Facility	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Wilhelmsson and Drettner (1984)	Plant 1		0.3-3.0	1.4	
Furniture	Plant 2		1.9-5.1	4.0	
Sweden (Smaland county)	Plant 3		0.4-3.2	1.3	
5 plants	Plant 4		0.8-2.8	1.7	
1984	Plant 5		0.4-3.0	1.3	
Personal samples/total dust		28	0.3-5.1	2.0	

NOTE: These data are part of a study of the occurrence of upper respiratory symptoms in the furniture industry. Most of the woodworkers were exposed to hardwood such as birch, beech, oak, mahogany, and teak.

(Continued)

	Facility:Department	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Kauppinen et al. (1984) Sawmill/plywood Finland 6 mills/6 plants 1984 Personal and area samples/ total dust	Sawmills:				
	Sawing Department				
	Framing saws	6	0.4-3.8	1.2	0.8
	Edging	3	0.8-3.4	2.1	2.1
	Trimming	4	0.1-0.7	0.4	0.3
	Sawing surface plates	1	5.1	5.1	5.1
	Stapling Department	3	0.4-2.7	1.0	0.8
	Finishing Department	38	0.1-9.6	2.3	
	Sorting Department	7	0.9-6.3	2.2	
	Planing Department				
	Ripping saws	5	0.5-2.0	1.2	1.4
	Planing	5	0.6-0.9	0.7	0.7
	Packaging	2	0.2-0.4	0.3	0.3
	Other Operations				
	Cleaning	4	3.1-15.0	7.6	6.2
	Control of chip makers	2	3.5-5.0	4.3	4.3
		80	0.1-15	2.2	
	Plywood Plants:				
	Sawing	23	0.4-12	2.5	1.7
	Finishing	6	0.8-9.5	3.2	2.1
	Other operations	2	1.4-2.1	1.8	1.8
	Plywood Plants	31	0.4-9.5	2.6	

NOTE: This was an industrial hygiene assessment. The sawmills and plywood plants processed pine, spruce, and birch. Two of the plywood plants also produced particle board and strip board. The average concentration of total dust in the Finishing areas of the plywood plants was 0.9 mg/m^3 (18 samples, range 0.3-2.4 mg/m^3) which reflected the exposure to which most of the workers were subjected. In the Finishing Departments of the sawmills, the respirable dust concentration averaged 26% (range 2-78%) of the mass of total dust in 16 samples collected. The average percentages of respirable dust in the finishing departments of the plywood plants were as follows: 40% in sawing operations (21 samples, range 1-90%), 29% in finishing operations (6 samples, range 8-64%), and 65% in the general workroom air (15 samples, range 16-100%). A 10-mm nylon cyclone was used as a preselector in the respirable dust sampling.

(Continued)

	Operation		Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Kauppinen and Niemela (1985)	Hogging*	1975-1984	3	0.1-29	11	3.9
Particle board production	Chipping*	1965-1974	1	1.1	1.1	1.1
Finland		1975-1984	3	0.7-1.7	1.1	1.0
8 plants	Chip drying*	1965-1974	2	24-29	26.5	26.5
1965-1984	Blending*	1965-1974	3	1.0-8.0	5.3	7.0
Area samples/total dust		1975-1984	3	0.6-0.9	0.8	0.8
*Short-term exposures were determined.	Forming	1965-1974	9	4.0-26	13	12
		1975-1984	4	<0.1-0.5	0.4	0.4
	Prepressing	1965-1974	2	6.0-7.0	6.5	6.5
	Hot pressing	1965-1974	6	1.0-6.1	4.1	4.6
		1975-1984	5	<0.1-2.1	0.8	0.5
	Planing	1965-1974	1	0.6	0.6	0.6
	Sawing	1965-1974	4	10-20	14	14
		1975-1984	9	<0.1-2.3	1.1	1.1
	Grinding	1975-1984	2	0.6-0.8	0.7	0.7
	Grading	1975-1984	1	1.1	1.1	1.1
	Packing	1975-1984	2	<0.6-0.6	0.4	0.4
	Matching	1975-1984	1	1.2	1.2	1.2
	1965-1974 Results		28	0.6-29	10.0	
	1975-1984 Results		33	<0.1-29	1.8	
	Particle board produced from sawdust:					
	Forming machines		4	26-360		
	Sawing machines		3	23-58		
	Prepressing area		2	15-17	16	16
	Hot pressing area		1	13	13	13
			10	13-360		

NOTE: These data are part of an epidemiological case-control study. Wood used was a mixture of spruce, pine, aspen, and birch. The main reason for the decrease in exposure to wood dust over time was likely the improved local exhaust ventilation at the forming and sawing machines.

(Continued)

	Department		Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Kauppinen (1986)	Log barking/cutting	1975-1984	4	0.2-0.7	0.4	0.4
Plywood	Peeling	1975-1984	2	0.2-0.3	0.3	0.2
Finland	Veneer sorting	1965-1974	1	0.4	0.4	0.4
19 plants	Veneer sawing	1965-1974	3	0.6-3.0	1.6	1.2
1965-1984		1975-1984	4	1.1-1.5	1.3	1.3
Personal samples/total dust	Plywood sawing	1965-1974	6	0.5-12	3.3	1.9
		1975-1984	11	0.3-19	3.7	2.8
	Plywood sanding	1965-1974	5	0.3-6.4	3.0	3.1
		1975-1984	21	0.8-22	3.8	2.6
	Plywood sorting	1975-1984	2	1.6-1.8	1.7	1.7
	Plywood finishing	1975-1984	18	0.3-2.4	0.7	0.8
	Chipping	1975-1984	11	0.7-7.1	2.6	2.4
	1965-1974 Results		15	0.3-12	2.7	
	1975-1984 Results		73	0.2-22	2.4	

NOTE: These data are part of an epidemiological case-control study. Wood used was a mixture of birch, spruce, and pine. Concentrations exceeding 5 mg/m^3 were occasionally measured in sanding and sawing at sites where the local exhaust systems were insufficient. In Plywood Sanding, higher concentrations were measured in samples which included the work phase of cleaning a sanding belt with compressed air. The concentration of dust in the general air in Plywood Sanding was about 1 mg/m^3. The respirable dust comprised 15-40% of the total dust in sanders' and sawyers' breathing zones. General area samples in Plywood Finishing contained about 65% respirable dust. A cyclone was used as a preselector in the respirable dust sampling.

(Continued)

	Facility	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Innocenti et al. (1985) Furniture Italy 1985 2 plants Personal samples/total dust *GM=geometric mean	Plant 1	6	2.5-25.3	9.1(GM*)	
	Plant 2	4	1.9-4.4	2.5(GM)	
		10	1.9-25.3	5.4(GM)	

NOTE: This was part of a preliminary investigation of anosmia in chestnut woodworkers.

Holness et al. (1985)
Furniture
Canada (Toronto)
4 plants
1984

NOTE: Total and respirable personal wood dust samples were collected for 50 woodworkers involved in sawing, sanding, and assemblying who were part of a health study of respiratory effects. The average total dust exposure was 1.8 mg/m^3. The average respirable dust exposure was 0.3 mg/m^3; a 10-mm nylon cyclone was used as a preselector.

(Continued)

	Sample Type: Job Category	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Sass-Kortsak et al. (1986)					
Cabinetmaking	Total Dust:				
Canada (Ontario)	Sawing	12		1.7	
4 plants	Sanding	7		2.9	
1984	Assembly	19		1.9	
Personal samples	Laminating/Gluing	3		1.1	
	Miscellaneous	7		1.2	
	Total Dust	48		1.8	
	Respirable Dust:				
	Sawing	12		0.1	
	Sanding	7		0.6	
	Assembly	18		0.3	
	Laminating	3		0.3	
	Miscellaneous	7		0.3	
	Respirable Dust	47		0.3	
	Sample Type: Wood Type				
	Total Dust:				
	Softwood	18		2.2	
	Hardwood	14		0.3	
	Respirable Dust				
	Softwood	18		2.0	
	Hardwood	14		0.3	

NOTE: Softwoods processed included cedar, poplar, fir, pine, spruce, chipboard, particle board, and fir plywood; hardwoods included birch, cherry, oak, and walnut. Personal total dust exposures were significantly higher ($p=0.003$) than those that would have been estimated from 12 area wood dust samples collected. All table saws and stationary sanders were equipped with local exhaust ventilation in 3 of the 4 plants; the mean total dust exposure (3.8 mg/m^3) in the 4th plant (without local exhaust ventilation) was significantly higher than the other three.

(Continued)

Holliday et al. (1986)
Various industries
Canada
23 plants
1985
Personal samples/total dust
* S=softwood
H=hardwood
PB=particle board
GM=geometric mean, mg/m^3

Industry:Product	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Primary Industries:				
Sawmill (S*)	18	0.3-4.1		
Flooring (S/H*)	7	0.3-1.7		
Veneer/plywood (H)	7	0.1-2.6		
Reconstituted board (H)	5	1.5-5.1		
Primary Industries	37	0.1-5.1	1.4 (GM*=0.7)	
Secondary Industries:				
Millwork (S)	15	0.3-6.5		
Millwork (S/H)	5	0.3-15.2		
Prefabricated buildings (S)	8	0.4-2.5		
Misc. wood products (S/H)	23	0.4-15.0		
Secondary Industries	51	0.3-15.2	2.2 (GM=0.9)	
Tertiary Industries:				
Kitchen cabinets (H)	12	0.3-5.1		
Kitchen cabinets (PB*)	5	0.7-3.7		
Household furniture (S/PB)	5	1.7-15.6		
Household furniture (H)	11	0.3-5.2		
Household furniture (PB)	6	0.5-6.8		
Office furniture (H)	7	0.5-1.7		
Office furniture (PB)	9	0.4-5.6		
Pattern making (H)	5	1.0-2.6		
Tertiary Industries	60	0.3-15.6	2.4 (GM=1.6)	
All Industries	148	0.1-15.6	2.0 (GM=1.1)	

(Continued)

	Operations in All Industries:	Number of Samples	TWA's, mg/m^3 Range	Mean	Median
Holliday et al. (1986) (continued)	Planing			3.3	(GM=2.6)
	Sanding			2.3	(GM=1.4)
	Sawing/cutting			2.2	(GM=1.2)
	Misc. operations in Primary Industries			1.9	(GM=1.3)
	Shaping/routing			1.9	(GM=1.1)
	Misc. operations not covered elsewhere			1.6	(GM=1.0)
	Sawing/cutting in Primary Industries			1.2	(GM=0.7)
		96	0.1-15.6	2.2	

NOTE: These data are part of a background overview of the occupational health aspects of wood dust exposure in Ontario industry. Median total dust concentrations in the general workplace air determined from area samples were as follows: 0.3 mg/m^3 in the Primary Industries (10 samples), 0.8 mg/m^3 in the Secondary Industries (12 samples), 1.2 mg/m^3 in the Tertiary Industries (28 samples), and 0.6 mg/m^3 overall. Particle sizing by optical microscopy indicated that for the 19 samples sized, particles in the 1-5 um range were the most numerous across all operations; for sawing, sanding, and planing/routing/shaping, the average fractions of particles in this size range were roughly 62, 61, and 65%, respectively.

Vedal et al. (1986) Sawmill Canada (British Columbia) 1 mill 1982-1985	NOTE: These data are part of a 3-year longitudinal health study of workers in a western red cedar sawmill. Exposures were assigned for 334 workers from 46 personal and 32 area total wood dust samples collected that ranged from undetectable to 6.0 mg/m^3. The average total dust exposure of the workers was 0.5 mg/m^3; the median exposure was 0.2 mg/m^3. It was determined that 10% of the workers were exposed to more than 1.0 mg/m^3 of total wood dust and that 3.9% were exposed to more than 2.0 mg/m^3.

Table 7.--Summary of total dust concentrations in sanding operations

Reference	Description	Number of Samples	TWA's, mg/m^3 Range	Mean
Kubis (1963)	Belt sanding	10	3.6-65.0	23.6
Hanslian and Kadlec (1964)	Belt sanding			40
Paustovskaya (1968)	Sanding	2	16.7-25.5	21.1
Imbus (1978)	Sanding	17	0.2-15.4	3.7
Otto (1973)	Mainly belt sanding:	14	<5	
		25	5-9	
		25	10-19	
		38	>20	
Hounam and Williams (1974)	Sanding	9	2.0-22.6	7.2
Andersen et al. (1977)	Sanding	41		14.3
Levy (1976)	Sanding	2	4.3-6.8	5.5
Gunter (1977a)	Sanding	1	90	90
Friar (1978)	Machine sanding	45	1-75	6
Weaver (1979)	Drum sanding		2.5	2.5
	Belt sanding		1.6-56.6	
	Edge sanding		2.4-20.7	
Whitehead et al. (1981b)	Sanding on hardwood	12	1.4-11.4	4.5
	Sanding on softwood	13	0.6-14.3	3.2
Jones and Smith (1986)	Machine sanding: 1976-1977	36	2.1-6.3	3.7
	1983	49	0.7-53	5.7
	Hand sanding: 1976-1977	33	5.8-32	16.9
	1983	42	0.5-27.2	6.9
McCawley (1983)	Large sander	8	1.0-5.6	2.9
	Small sander	5	1.7-4.5	2.9
Sass-Kortsak et al. (1986)	Sanding	7		2.9
Holliday et al. (1986)	Sanding			2.3

Table 8.--Summary of total dust concentrations in sawing operations

Reference	Description	Number of Samples	TWA's, mg/m^3 Range	Mean
Imbus (1978)	Sawing	6	0.6-4.7	1.9
Hounam and Williams (1974)	Band sawing	6	1.0-7.3	4.3
Kominsky and Anstadt (1976)	Sawing	17	0.7-416	38.1
Levy (1976)	Sawing	2	1.1-3.4	2.2
Friar (1978)	Band sawing	13	2-127	18
	Other sawing	21	2-35	8
Apol (1979b)	Panel sawing	2	0.9-1.6	1.3
Weaver (1979)	Rip/cutoff sawing		8.4-77.2	
	Double trim sawing		6.0	6.0
Al Zuhair et al. (1981)	Sawmill department	75		3.5
Jones and Smith (1986)	Sawing: 1976-1977	29	4.7-13.1	7.4
	1983	49	1.0-30.3	3.4
Lindroos (1983)	Sawing	11	0.3-2.0	0.8
Sass-Kortsak et al. (1986)	Sawing	12		1.7
Holliday et al. (1986)	Sawing/cutting			2.2

Table 9.--Summary of total dust concentrations in woodworking operations (excluding sanding/sawing)

Reference	Description	Number of Samples	TWA's, mg/m^3 Range	Mean
Paustovskaya (1968)	Hand finishing	1	23.5	23.5
Imbus (1978)	Grinding	2	1.3-4.9	3.1
	Routing	2	1.0-2.2	1.6
	Molding	2	1.1-2.7	1.9
	Lathe/tenoner/shaper	3	3.3-4.5	3.7
Hounam and Williams (1974)	Planing	9	1.8-10.9	5.0
	Routing	6	1.8-8.6	4.1
	Spindle moulding	8	1.5-8.4	5.1
	Assembly	10	2.1-25.5	7.5
	Turning	2	4.6-12.5	8.6
Rosensteel (1974)	Roundtable shaper	2	8.6-8.9	8.8
Kominsky and Anstadt (1976)	Routing/grooving	5	1.2-48.5	12.5
Gunter (1977a)	Mill worker	1	4	4
Friar (1978)	Assembly	35	2-41	8
Weaver (1979)	Boring		4.8	4.8
	Routing		3.1-15.5	
	Hand shaping		15.3-21.1	
Whitehead et al. (1981b)	Rough milling	12	0.2-2.6	0.7
	Detailed milling	18	0.2-6.3	1.7
	Assembly	5	1.1-3.1	2.0
Enright (1980)	Routing	1	2.4	2.4
	Shaping	4	2.0-10.2	5.2
	Omni milling	3	0.1-0.4	0.2
McCawley (1983)	Routing	5	1.7-8.1	4.0
Lindroos (1983)	Unloading wood	10	0.5-28.0	4.3
	Planing	20	0.02-1.8	0.4

(Continued)

Table 9 (Continued).--Summary of total dust concentrations in woodworking operations (excluding sanding/sawing)

Reference	Description	Number of Samples	TWA's, mg/m^3 Range	Mean
McCammon et al. (1985)	Sweeper	5	0.1-6.1	1.6
	Shaping	7	0.3-13.9	2.7
	Multi-axis machining	4	0.2-1.0	0.5
Jones and Smith (1986)	Cutting (excluding sawing):			
	1976-1977	44	2.0-10.8	6.9
	1983	70	0.3-8.8	2.3
Sass-Kortsak et al. (1986)	Assembly	19		1.9
	Laminating/gluing	3		1.1
	Miscellaneous	7		1.2
Holliday et al. (1986)	Planing			3.3
	Shaping/routing			1.9

Table 10.--Summary of general area samples for total dust collected in woodworking operations

Reference	Description	Number of Samples	TWA's, mg/m^3 Range	Mean
Paustovskaya (1968)	Remote areas			5-6
Levy (1976)	Sawing/sanding/planing	1		2.5
Al Zuhair et al. (1981)	Sawmill Department	6		1.4
	Assembly Department	2		0.8
	Machine floor	16		0.7
	Cabinet shop	14		4.4
McCammon et al. (1985)	Wood mill	10	0.05-0.5	0.3
	Mini mill	4	0.3-0.6	0.4
Lindroos (1983)	Pretreatment Department	9	0.2-1.0	0.6
	Woodworking Department	14	0.1-0.4	0.3
Holliday et al. (1986)	Secondary Industries	12		0.8
	Tertiary Industries	28		1.2

Table 11.--Summary of respirable dust concentrations in woodworking operations

Reference	Description	Number of Samples	TWA's, mg/m^3 Range	Mean
Imbus (1978)	Sanding	1	0.7	0.7
Rosensteel (1974)	Roundtable shaper	2	0.02-0.4	0.2
Kominsky and Anstadt (1976)	Saw	13	0.07-4.4	0.7
	Router/groover	3	0.3-0.8	0.5
Weaver (1979)	Cutoff sawing			2.1
	Router			1.5
Enright (1980)	Orbital sander	1	0.5	0.5
			(2.2% of total dust)	
	Hand sanding	1	0.4	0.4
			(4.8% of total dust)	
Al Zuhair et al. (1981)	Machine floor	4		0.19
	Cabinet shop	4		0.44
McCammon et al. (1985)	18-61% of dust (mass) is respirable			
Ellenbecker and Wegman (1985)	Sander	3	0.5-0.9	0.6
Lindroos (1983)	Pretreatment Dept.	7	0.14-0.19	0.16
	Woodworking Dept.	6	0.11-0.20	0.14
Holness et al. (1985)	Sawing/sanding			0.3
Sass-Kortsak et al. (1986)	Sawing	12		0.1
	Sanding	7		0.6
	Assembly	18		0.3
	Laminating/gluing	3		0.3
	Miscellaneous	7		0.3

Table 12.--Summary of particle size data in woodworking operations

Reference	Description	MMAD, um
Hounam and Williams (1974)	Sanding	8.4
NOTE: More than 75% of the dust (mass) was >5 um in size.	Band sawing	11.5
	Planing	9.2
	Routing	10.0
	Spindle moulding	10.0
	Assembly	7.6
	Turning	11.5
Andersen et al. (1977)	33% (mass) was in particles <5 m	
	41% (mass) was in range 6-10 um	
	11% (mass) was in range 11-15 um	
	15% (mass) was in particles >16 um	
Kominsky (1976)	Saw and router/groover	>10
Whitehead et al. (1981b)	Relatively little dust was in smallest size ranges	
Al Zuhair et al. (1981)	Machine floor	9.3
	Cabinet shop	12.5
	Sawmill	17.3
	Assembly	18.0
McCammon et al. (1985)	Wood Mill A (9/25/80)	10
	Wood Mill A (1/9/80)	9.8
	Wood Mill A (3/4/80)	6.6
	Wood Mill A (9/4/80)	8.4
	Wood Mill C (3/4/80)	6.1
	Mini Mill A (1/9/80)	9.0
	Mini Mill A (1/10/80)	5.2
	Shaper Room C (3/5/80)	6.1
	Shaper Room C (3/5/80)	7.8

(Continued)

Table 12 (Continued).--Summary of particle size data in woodworking operations

Reference	Description	MMAD, um
Jones and Smith (1986)	Sanding 54% (mass) was in the range 4-10 um	9
McCawley (1983)	45-60% (mass) was in particles <3.5 um 30-35% (mass) was in range 3.5-20 um 5-20% (mass) was in particles >20 um	

Table 13.--Recommended exhaust volumes for woodworking machinery*

Type of machinery	Exhaust volume (cfm)
Saws	
Band	700-1,950
Table (including rip, mitre, and variety saws)	350-550
Swing	350-440
Gang rip 900-2,060	
Self-feed table rip	790-1,350
Sanders	
Disc	350-1,250
Horizontal Belt	790-1,650
Vertical belt	440-1,100
Drum (single)	350-1,400
Drum (multiple)	550-1,400
Swing arm	440
Planers	
Single	785-2,200
Double	1,335-3,600
Routers	350-800
Jointers	350-800
Molders, matchers, sizers	1,690-5,370
Lathes	
Automatic	800-5,000
Forming	350-1,400

*Adapted from ACGIH (1986)

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