

C6-1. Substances for Which Limits Are Based on Avoidance of Respiratory Effects (continued)

H.S. Number/ Chemical Name	CAS No.	Former PEL	Proposed PEL	Final Rule PEL*
1430C Wood dust, allergenic (Western Red Cedar)	None	--	--	2.5 mg/m ³ TWA

- a For coal dust, respirable fraction less than 5 percent SiO₂.
b For coal dust, respirable fraction more than 5 percent SiO₂.
* OSHA's TWA limits are for 8-hour exposures; its STELs are for 15 minutes unless otherwise specified; and its ceilings are peaks not to be exceeded for any period of time.

BILLING CODE 4810-26-C

H.S. No. 1430A (Hard Wood)
H.S. No. 1430B (Soft Wood)
H.S. No. 1430C (Western Red Cedar)

Before 1980, OSHA regulated wood dust under its nuisance dust standard of 15 mg/m^3 (29 CFR 1910.1000, Table Z-3). However, in a 1985 enforcement proceeding before the Occupational Safety and Health Review Commission, wood dust was held not to be covered by the nuisance dust standard, an inert mineral dust, and the Agency did not regulate this substance after this decision (12 OSHC 1785). Consequently, OSHA had no PEL for wood dust when this generic rulemaking was undertaken. The ACGIH has a TLV-TWA of 1 mg/m^3 for hard wood dust, and a TLV-TWA of 5 mg/m^3 and STEL of 10 mg/m^3 for soft wood dust. OSHA proposed a 1-mg/m^3 8-hour TWA for hard wood dust and a 5-mg/m^3 8-hour TWA for soft wood dust. In the final rule, OSHA is establishing a single 8-hour TWA of 5 mg/m^3 and a STEL of 10 mg/m^3 for all hard wood and soft wood dusts except Western red cedar. For Western red cedar, a highly allergenic species of soft wood, the Agency is establishing an 8-hour TWA limit of 2.5 mg/m^3 . Wood dust is defined as any wood particles arising from the processing or handling of woods. Hard woods derive from the deciduous broad-leaved flowering species of trees, and soft woods include the coniferous species that do not shed their leaves in the winter.

Exposure to wood dust has long been associated with a variety of adverse health effects, including dermatitis, allergic respiratory effects, mucosal and nonallergic respiratory effects, and cancer. The toxicity data in animals are limited, particularly with regard to exposure to wood dust alone; there are, however, a large number of studies in humans. The discussion below first describes some of the relevant toxicological studies and then presents the record evidence on wood dust.

Animal Studies

Groups of male guinea pigs were injected intratracheally with suspensions containing 75 mg of sheesham or mango wood dust or of hemp or bagasse fibers, or 20 mg of jute fiber (Bhattacharjee, Dogra, Lal, and Zaidi 1979/Ex. 1-463; Bhattacharjee and Zaidi 1982/Ex. 1-464). Animals were sacrificed serially at intervals up to 90 days after injection. Lung examination revealed that, at 90 days, Grade I fibrosis of the lungs had occurred in the guinea pigs injected with mango or jute, while those treated with sheesham or hemp had developed Grade II pulmonary fibrosis.

WOOD DUST

CAS: None; Chemical Formula: None

In another experiment involving guinea pigs, animals were exposed by inhalation to average respirable dust concentrations of 1143 mg/m^3 for 30 minutes/day, 5 days/week for 24 weeks (McMichael, DiPalma, Blumenstein et al. 1983, Ex. 1-644). Histopathological examination showed lung changes, described by the authors as moderate to severe, in all exposed guinea pigs. The changes seen included an increase in septal connective tissue components and aggregation of lymphocytes; however, no pulmonary fibrosis or extensive destruction of the parenchymal tissue occurred. The authors of this study concluded that exposure to fir bark dust may cause inflammatory changes in the lung.

Two studies examined the effect of exposing Syrian golden hamsters to beech wood dust by inhalation, with or without concurrent administration of the known carcinogen diethylnitrosamine (DEN) (Wilhelmsson, Hellquist, Olofsson, and Klintenberg 1985/Ex. 1-402; Wilhelmsson, Jernudd, Ripe, and Holmberg 1985/Ex. 1-1042; Drettner, Wilhelmsson, and Lundh 1985/Ex. 1-312). In each study, the animals were divided into four separate groups. In Study I, there were 12 animals per group. Two groups were exposed to fresh beech wood dust (a hard wood dust) at a mean total dust concentration of 15 mg/m^3 for six hours/day, five days/week for 36 weeks, and one of these groups was also given 1.5 mg of DEN once a week for the first 12 weeks. The third group in Study I was given the DEN doses only (positive control), and the fourth group was given no exposure at all (negative control).

In Study II, there were 24 animals in each of four groups. Two groups of animals were exposed to fresh beech wood dust at a mean total dust concentration of 30 mg/m^3 for six hours/day, five days/week for 40 weeks. The positive and negative control groups were treated as in Study I.

In Study I, none of the hamsters had lung or nasal tumors or metaplasia. Four hamsters exposed to wood dust and DEN exhibited squamous cell papillomas of the trachea, as did three animals in the positive control group and one in the negative control group. No differences in organs other than the respiratory organs were seen between the treated and control groups in Study I.

In Study II, all DEN-exposed hamsters had nasal lesions ranging from hyperplasias and dysplasias to papillomas. In addition, half of all DEN-exposed hamsters developed nasal adenocarcinomas, whether or not they had also been exposed to wood dust.

Half of the DEN-exposed animals also had papillomas of the larynx and trachea. In the wood-dust-exposure-only group, two of the animals had nasal lesions, one of which was an unclassifiable malignant nasal tumor and the other of which consisted of focal metaplasia with mild dysplasia. The authors concluded that exposure to wood dust did not increase the tumor incidence in DEN-exposed animals but did affect the respiratory tract of all exposed animals.

Human Studies

Dermatitis. There are a large number of case reports, epidemiological studies, and other data on the health effects of wood dust exposure in humans. Dermatitis caused by exposure to wood dusts is common, and can be caused either by chemical irritation, sensitization (allergic reaction), or both of these together. As many as 300 species of trees have been implicated in wood-caused dermatitis.

The chemicals associated with allergic reactions are generally found in the inner parts of a tree, e.g., the heartwood, and the workers most prone to these reactions are those involved in secondary wood processing (e.g., carpenters, joiners, and finishers).

The symptoms of sensitization are redness, scaling, and itching, which may progress to vesicular dermatitis and, after repeated exposures, to chronic dermatitis. The parts of the body most often affected are the hands, forearms, eyelids, face, neck, and genitals. This form of dermatitis generally appears after a few days or weeks of contact.

Allergic respiratory effects. Allergic respiratory responses are mediated by the immune system, as is also the case with allergic dermatitis. Many authors have reported cases of allergic reactions in workers exposed to wood dust (Sosman, Schlueter, Fink, and Barboriak 1969/Ex. 1-444; Greenberg 1972/Ex. 1-482; Pickering, Batten, and Pepys 1972/Ex. 1-655; Eaton 1973/Ex. 1-478; Booth, LeFoldt, and Moffitt 1976/Ex. 1-466; Chan-Yeung, Ashley, Corey et al. 1978/Ex. 1-622; Edwards, Brooks, Henderson, and Apol 1978/Ex. 1-950; Innocenti and Angotzi 1980/Ex. 1-1036; Bush and Clayton 1983/Ex. 1-469; Cartier, Chan, Malo et al. 1988/Ex. 1-472). Asthma is the most common response to wood dust exposure, and the allergic nature of such reactions has been demonstrated by the presence of IgE antibodies and positive skin reactions on patch testing. The best-studied of the allergic reactions to wood dust is Western red cedar (WRC) asthma; it is estimated that 5 percent of the workers handling this species are allergic to it. However, only one study is

available that relates exposure level to ventilatory function. In that study, exposure to concentrations of 2 mg/m^3 of WRC dust caused significant decreases in forced vital capacity and forced expiratory volume (Vedal, Chan-Yeung, Enarson et al. 1986/Ex. 1-397). These authors also found that exposures to concentrations above 3 mg/m^3 produced eye irritation.

Mucosal and nonallergic respiratory effects. This section discusses changes in the structure and function of the nasal mucosa and respiratory tract that are caused by exposure to wood dust. These changes include nasal dryness, irritation, bleeding, and obstruction; coughing, wheezing, and sneezing; sinusitis; and prolonged colds. These symptoms have been observed even at wood dust concentrations below 4 mg/m^3 .

Bellion, Mattei, and Treves (1964, as cited in NIOSH 1987a/Ex. 1-1005) found that 97 of 225 workers (carpenters, sawmill workers, woodworkers) exposed from 3 to 24 years to the dust of several different hard woods showed radiologic evidence of pulmonary abnormalities. Black, Evans, Hadfield et al. (1974/Ex. 1-299) studied nine woodworkers from a woodworking factory in England. In all of these workers, mucociliary movement was markedly depressed, leading these authors to conclude that exposure to wood dust in the furniture industry for 10 years or more can impair mucociliary clearance. These findings were confirmed in a Danish study involving furniture makers (Solgaard and Andersen 1975/Ex. 1-443; Andersen, Solgaard, and Andersen 1976/Ex. 1-297; Andersen, Andersen, and Solgaard 1977/Ex. 1-296); compared with controls, the mucociliary transport rate was also significantly impaired in these woodworkers, and dose-response effects were noted.

A respiratory survey conducted by Chan-Yeung, Giclas, and Henson (1980/Ex. 1-474) in pulp and paper mill workers in British Columbia showed that workers exposed to wood dust at a mean total dust concentration of 0.5 mg/m^3 had a slight but statistically significant decrease in pulmonary function values compared with controls. The authors concluded that the chemical preservatives used to treat the wood could also have been responsible for these adverse effects.

In a cross-sectional survey of 1,157 American woodworkers (both hard and soft wood), Whitehead, Ashikaga, and Vacek (1981/Ex. 1-454) found that exposure to higher ($10+ \text{ mg-years/m}^3$), as compared with lower (0 to 2 mg-

years/m³), dust concentrations was associated with a statistically significant and higher incidence of decreased pulmonary function. However, dose-response effects were observed only for soft wood (i.e., pine) dusts. A later study by Beckman, Ashikaga, and Whitehead (1980, as cited in NIOSH 1987a/Ex. 1-1005) examined subgroups of the workers studied by Whitehead and found no correlation between years of exposure to pine wood dust and pulmonary function.

In a pilot study of 55 workers in a North Carolina hardwood furniture plant, Goldsmith (1983, as cited in NIOSH 1987a/Ex. 1-1005) found that, at mean area wood dust concentrations of 2 mg/m³ or below, peak ventilatory flow correlated significantly with cumulative person-years of exposure. Goldsmith interpreted this finding to mean that inhalation of wood dust may impair large-airway function.

A study of Italian woodworkers showed that the number of wood-dust-exposed workers who had developed anosmia (loss of smell) was significantly higher than in a control group of nonexposed workers (Innocenti, Valiani, Vessio et al. 1985/Ex. 1-1037). Amore (1986/Ex. 1-1029) confirmed this finding in other workers exposed to hardwood dusts.

Summary of mucosal and nonallergic respiratory effects. A large number of studies have demonstrated that occupational exposure to wood dust causes both statistically significant and nonsignificant increases in respiratory symptoms at exposure levels as low as 2 mg/m³. These symptoms range from irritation to bleeding, wheezing, sinusitis, and prolonged colds. In addition, chronic wood dust exposure causes mucociliary stasis (i.e., the absence of effective clearance) in the nose and, in some workers, also causes changes in the nasal mucosa. Several studies have demonstrated decreased pulmonary function among wood-dust-exposed workers, although other studies have not confirmed these findings.

Carcinogenicity

The association between occupational exposure to wood dust and various forms of cancer has been explored in many studies and in many countries. In 1987, the International Agency for Research on Cancer (IARC) classified furniture manufacturing in Category I (confirmed human carcinogen) and carpentry in Category 2B (suspected human carcinogen). NIOSH (Ex. 8-47) considers both hard and soft wood dust to be potentially carcinogenic in humans; for soft wood dust, NIOSH recommends a separate 6(b) rulemaking

(Ex. 8-47, Table N6B). NIOSH concurred, however, with the proposed PEL of 1 mg/m³ TWA for hard wood dust (Ex. 8-47, Table N6A).

The discussion below focuses on selected U.S. studies.

Nasal and sinus cavity cancer. The earliest U.S. study of wood dust exposure and nasal cancer was conducted by Brinton, Stone, Blot, and Fraumeni (Ex. 1-468) in 1976. These authors analyzed cancer death rates between 1950 and 1969 in 132 U.S. counties having at least 1 percent of their population employed in furniture and wood-fixture manufacturing. This study revealed that the age-adjusted mortality rate for cancer of the nasal cavity and sinuses among white males in the "furniture" counties was significantly higher than in nonfurniture counties.

In a later case-control study, these authors (Brinton, Blot, Becker et al. 1984/Ex. 1-467) analyzed cases of nasal and sinus cancers occurring in North Carolina and Virginia between 1970 and 1980. This study identified a significantly elevated risk of adenocarcinomas in males working in the furniture manufacturing industry, but no increased risk among lumber, carpentry, or construction workers. There was no significant increase in the risk of squamous cell carcinoma in workers from any other wood-related industry.

In a study sponsored by the Inter-Industry Wood Dust Task Force, Viren, Vogt, and Dixon (1982, as cited in NIOSH 1987a/Ex. 1-1005) described a death certificate case-control study of nasal cancer deaths for 1963 to 1977 in North Carolina, Mississippi, Washington, and Oregon. Findings of this study included a relative nasal cancer risk of 1.95 for industries involving lumber and wood products; however, no significant relative risk of nasal cancer was seen for workers in the furniture-manufacturing industry.

Imbus and Dyson conducted a study of nasal cancer and North Carolina furniture workers (1985, as cited in NIOSH 1987a/Ex. 1-1005). This study found: (1) that there was a statistically significant increase of nasal cancer among furniture workers; (2) that the nasal cancer rates among North Carolina furniture workers were much lower than those reported for English furniture workers; (3) that the number of nasal cancer deaths among North Carolina furniture workers decreased between 1956 and 1977; and (4) that a slight excess in nasal cancer may have existed among North Carolina furniture workers but is currently either declining or nonexistent.

At present, the National Cancer Institute is conducting a cohort mortality study of 36,622 workers employed in the wood, metal, and plastic furniture manufacturing industries (Miller et al. 1988, as cited in NIOSH 1987a/Ex. 1-1005). Results are too preliminary to be described at this time.

Summary of evidence for nasal and sinus cavity cancers. NIOSH (1987a/Ex. 1-1005) concluded that the literature clearly demonstrates an association between occupational wood dust exposure and nasal cancer. English studies first identified this link by showing a 10- to 20-times-greater incidence of nasal adenocarcinoma among woodworkers in the furniture industry than among other woodworkers and 100 times greater than in the general population. In the United States, three studies have reported a fourfold risk of nasal cancer or adenocarcinoma in furniture workers, and another study noted a similar relationship between nasal cancer and wood dust exposure. One other study failed to find such an association for furniture workers, but did find an increase among logging and timber industry workers.

Pulmonary cancer. A number of studies investigating the association between wood dust exposure and the development of lung cancer have been conducted. Milham (1974/Ex. 1-943) found a significant excess of malignant tumors of the bronchus and lung in workers who had belonged to the AFL-CIO United Brotherhood of Carpenters and Joiners of America. Only construction workers showed a statistically significant increase in lung cancer rate.

In a study of lung cancer in Florida residents, Blot, Davies, Brown et al. (1982/Ex. 1-465) found that an elevated risk of lung cancer that was statistically significant existed among workers in the lumber and wood industry and in construction; however, smoking may have been a confounding factor in these results.

Summary of evidence for pulmonary cancer. The association between lung cancer and occupational wood dust exposure is inconclusive, although several epidemiological studies have reported increases in lung cancer among wood-dust-exposed workers.

Hodgkin's disease. The data on the relationship between exposure to wood dust and the development of Hodgkin's disease are conflicting. Milham (1987/Ex. 1-750) and Milham and Hesser (1967/Ex. 1-645) concluded, on the basis of a case-cohort study of 1,549 white males dying of this disease between 1940-1953 and 1957-1964, that there was

an association between Hodgkin's disease and exposure to wood dust.

Another study (Spiers 1969/Ex. 1-445) concluded that men working in the wood industries in the eastern United States were at special risk for Hodgkin's disease, and suggested that pine pollen exposure might be responsible for the increase.

A Washington State epidemiological study (Petersen and Milham 1974/Ex. 1-654) also found that woodworkers had an increased risk of Hodgkin's disease, and the work of these authors was supported by the results of another study (Grufferman, Duong, and Cole 1976/Ex. 1-484), which showed a nonsignificant increase in the relative risk for Hodgkin's disease among woodworkers.

Summary of evidence for Hodgkin's disease. Although the data are conflicting, several epidemiological studies of U.S. workers do report increases in the incidence of Hodgkin's disease among woodworkers. This excess is particularly apparent among carpenters.

Other cancers. NIOSH (1987a/Ex. 1-1005) concluded that the data on the relationship between occupational exposure to wood dust and the development of cancers other than nasal, Hodgkin's disease, or lung cancers are insufficient and inconclusive.

Record Evidence

Many participants submitted comments to the record pertaining to wood dust (see, for example, Exs. 8-34, 3-748, 3-233, 3-349, 3-362, 3-626, 3-682, 3-824, 3-836, 3-859, 3-899, 3-955, 3-1160, 3-917, 115, 127, 131, 141, 155, 168, 183, 191, 194, 3-1453, 195, 196, 189, 82, 80, and 3-911; Tr. 12, pp. 144 to 455). These commenters described their facilities and woodworking processes, employee safety and health programs, and concerns about the impact of the proposed rule's limits for wood dust on their industries. The issues raised by these participants concerned the following topics:

- (1) The technological and economic feasibility of the proposed limits;
- (2) The justification for a separate standard for soft wood and hard wood;
- (3) The health effects evidence;
- (4) The appropriate levels for the final rule's PELs; and
- (5) The evidence for a separate limit for allergenic wood dusts.

The discussions below deal with each of these points in turn.

Representatives from many affected segments of the wood industry stated that achieving the proposed limits of 1 mg/m³ for hard wood and 5 mg/m³ for

soft wood would be technologically or economically infeasible or extremely difficult (Exs. 8-34, 3-917, 168, 183, 191, 80, and 3-911). OSHA has determined that, at the present time, the health evidence suggests that a single PEL of 5 mg/m³ is appropriate for both hard and soft wood dust, with the exception of Western red cedar, for which a PEL of 2.5 mg/m³ is being set. These revised PELs have been determined to be feasible (see the detailed discussion of these issues in the Technological Feasibility and Economic Impact sections of this preamble).

OSHA proposed separate permissible exposure limits for soft wood (5 mg/m³) and hard wood (1 mg/m³). The Agency received comments on this topic from many participants; these commenters were unanimously opposed to the setting of separate limits for these two types of wood dust (Exs. 8-34, 3-748, 3-682, 3-859, 3-899, 3-917, 191, 196, 80G, 80L, 80N, and 3-911; Tr. XII, pp. 12-290, 12-326, and 12-331). These participants stated that there was no health basis for making a distinction between hard wood and soft wood dusts (Exs. 33-899, 3-955, 3-917, and 191; Tr. 12, pp. 326-331; Tr. 12, p. 290). According to Dr. Harold Imbus, speaking for the Inter-Industry Wood Dust Coordinating Committee (Tr. pp. 12-58, 12-60), the distinction between the two woods derived from the fact that the early studies showing an increased cancer incidence in woodworking employees involved British furniture makers, who predominantly used hard wood; this association caused investigators to attribute greater toxicity to hard wood dust.

Commenters were of the opinion that this distinction was no longer warranted by the evidence; in fact, Dr. Lawrence Whitehead, certified industrial hygienist and a professor at the University of Texas School of Public Health (Tr. p. 12-331), stated that his own work suggested that some soft wood dust exposures might actually produce stronger adverse effects than equivalent exposures to some hard wood dusts.

Other commenters reported that it is not possible to distinguish soft wood from hard wood dust except by chemical analysis (Ex. 8-34, p. 28), that most facilities in the wood industries use both hard and soft woods (Exs. 3-682, 3-859, and 3-899), and that the distinction between the two types of woods is inappropriate (Ex. 3-917). For example, Joseph Gerard, Vice President of the American Furniture Manufacturers Association (Ex. 3-917) stated:

The distinction between hard woods and soft woods is purely botanical. Many so-

called "softwoods" are actually hard (i.e., Douglas fir as a softwood is harder than the hardwood birch) and one of the softest woods in existence (balsa) is botanically a hardwood (Ex. 3-917, p. 2).

Jamie Cohen, speaking for the United Petitioners, a coalition of labor unions (Tr. 12, p. 294), believes that a bifurcated standard for the two types of dusts would place an undue burden on employers and could lead to compliance problems. The posthearing brief submitted by the United Brotherhood of Carpenters and Joiners of America (Ex. 196) reiterated these points by stating: "Given the frequent intermixture of wood types in the workplace, this [setting two separate standards] would render OSHA's compliance efforts virtually worthless" (Ex. 196, p. 7).

After a review of this record evidence, OSHA has determined that the health evidence for the toxicity of wood dust cannot be separately distinguished for soft wood and hard wood. In addition, the Agency is convinced by the many comments from wood industry employers that most operations involve both kinds of wood and are performed on the same machines and equipment and in the same facility. Thus, any controls installed to reduce exposures would of necessity need to be sufficient to reduce airborne dust levels to the lower of the two limits (i.e., to the proposed wood dust limit of 1 mg/m³). According to the Inter-Industry Wood Dust Coordinating Committee:

[I]mposition of a limit of 1 mg/m³ for hardwood dust and 5 mg/m³ for softwood dust effectively imposes a limit of 1 mg/m³ on a large number of plants, including those where only small amounts of hardwood are used (Ex. 3-748, p. 3).

Many commenters took exception to the review of the health effects evidence for wood dust presented by OSHA in the preamble to the proposed rule. Objections were raised by the Inter-Industry Wood Dust Coordinating Committee (Exs. 8-34, 3-748, and 168), the Appalachian Hardwood Manufacturers (Ex. 3-626), the American Furniture Manufacturers Association (Exs. 3-917 and 191), the Georgia-Pacific Corporation (Exs. 3-955 and 183), the Hardwood Plywood Manufacturing Association (Ex. 3-911), and others.

These participants criticized many of the individual studies described by OSHA; some commenters found fault with several of these studies on the grounds that they involved British or other non-U.S. woodworkers (see, for example, Exs. 8-34, 191, 3-626, and 3-917), involved only a small number of subjects (see, for example, Exs. 8-34, 168, and 191), had inconsistent results

(see, for example, Ex. 8-34), or failed to demonstrate a dose-response relationship between wood dust exposure and the health effect of concern (see, for example, Exs. 8-34, 3-626, 3-917, and 191). The Inter-Industry Wood Dust Coordinating Committee (IIWDCC) stated:

[T]he observations in the European studies are not representative of conditions in U.S. workplaces, especially under modern conditions. . . .

The English and other European experience does not provide an accurate predictive model for the incidence of nasal cancer. . . . The excesses of nasal cancer observed in the European studies simply have not been observed in the United States at any time. . . . (Ex. 3-748, pp. 2, 52).

OSHA agrees with the IIWDCC that the incidence of nasal cancer seen in the United States is substantially lower than that seen in other countries, particularly in Great Britain. However, the Agency does not agree that excesses in nasal cancers, and particularly of nasal adenocarcinomas, have not been observed in American woodworkers. Several U.S. studies have reported excesses in nasal cancer risks among employees in the wood industries (Brinton, Stone, Blot, and Fraumeni 1976/Ex. 1-468; Brinton, Blot, Becker et al. 1984/Ex. 1-467; Viren, Vogt, and Dixon 1982, and Imbus and Dyson 1985, both as cited in NIOSH 1987a/Ex. 1-1005).

In response to those commenters who argued that none of the studies described by OSHA presented sufficient dose-response data to be used as a basis for establishing a limit, the Agency emphasizes that it is not relying on any single study to determine that wood dust presents a significant risk of material health impairment. Instead, OSHA is making this determination on the basis of the findings in the dozens of studies reporting on the respiratory, irritant, allergic, and carcinogenic properties of wood dust. The Agency finds the results of these studies biologically plausible and their findings reproducible and consistent. It is true that some of these studies, like all human studies, have limitations of sample size, involve confounding exposures, have exposure measurement problems, and often do not produce the kind of dose-response data that can be obtained when experimental animals are subjected to controlled laboratory conditions. What the large group of studies being relied upon by OSHA to establish the significance of the risk associated with exposure to wood dust do show is that the overall weight of evidence that such exposures are harmful and cause loss of functional capacity and material

impairment of health is convincing beyond a reasonable doubt.

The industry strongly supported a single 5-mg/m³ standard for both hard wood and soft wood dusts (Exs. 8-34, 3-626, 3-682, 3-824, 3-899, 3-1180, 3-917, 168], 183, 191, 80 and attachments, and 3-911); some commenters (Exs. 3-859, 194, and 196) argued for a 1-mg/m³ limit for all wood dust, while others (Exs. 3-955, 155, and 183) were of the opinion that the nuisance dust limit of 10 mg/m³ was appropriate for wood dust. The United Petitioners (Tr. p. 12-294) strongly endorsed a 1-mg/m³ standard for wood dust of all types on the grounds that the available health evidence clearly supports this limit.

OSHA finds that the health evidence in the record as a whole does not support a PEL of 1 mg/m³ for all wood dusts. In addition, the Agency believes that a 1-mg/m³ limit would present serious problems of feasibility for affected parties (see Section VII, Summary Economic Impact and Regulatory Flexibility Analysis). The Agency also finds that the health evidence clearly indicates that occupational exposure to wood dust poses a significant risk of material health impairment at the 10-mg/m³ (or particulate) level. OSHA concludes that establishing an 8-hour PEL of 5 mg/m³ and a 15-minute STEL of 10 mg/m³ for all wood dusts (except Western red cedar) will substantially reduce this significant risk.

The final rule establishes an 8-hour TWA PEL of 2.5 mg/m³ for Western red cedar wood dust, based on its widely recognized ability to cause immune-system-mediated allergic sensitization. Evidence in the record demonstrates the seriousness of this effect. A study by Brooks, Edwards, Apol, and Edwards (1980) that was submitted by the United Petitioners (Ex. 82D) reports that a high prevalence of occupational asthma was observed among workers exposed to WRC wood dust (Ex. 82D, p. 315).

At the hearing, Dr. Brooks described occupational asthma as follows:

[T]here are spasms of the bronchial tubes, there is reduced air flow on expiration . . . [the extent of which depends] on the extent of the exposure, and also . . . on the duration of the exposure . . . as a consequence of this sensitization and airway injury from the sensitization and the asthmatic reaction and the various biochemical and cellular changes that occur, there develops an associated process . . . the airways develop an increased sensitivity and an increased bronchospastic responsiveness to many different non-specific stimuli. So such things as cold air, dust, fumes, gases that are non-specific and wouldn't normally . . . [affect]

most individuals [will affect] the individual with occupational asthma. And it's [such] hyper-reactive airways that cause individuals to continue to have disability and to continue to have symptoms once they leave the work place. . . . They develop this non-specific bronchial hyper-reactivity which may last the rest of their life (Tr. pp. 12-339 to 12-343).

Some commenters (Exs. 8-34, 183, and 191) opposed the establishment of a separate PEL for Western red cedar. These participants argued that a lower PEL "for wood dust generally would be necessary or appropriate to address allergic symptoms" (Ex. 8-34, Health Effects Comments, p. 8, footnote 6). According to the Inter-Industry Wood Dust Coordinating Committee (IIWDCC):

[P]revention of allergic reactions is best achieved by good housekeeping measures directed specifically at the allergenic species (Ex. 8-34, p. 8).

Among the work practices recommended by these commenters were maintaining clean work spaces, wearing protective clothing, and avoiding skin contact with the allergenic species (Ex. 8-34, Health Effects Comments, p. 17).

Although OSHA endorses training, good work practices, and the use of appropriate protective clothing, the Agency does not agree that a reduced PEL for Western red cedar (WRC) is unnecessary. The health effects associated with occupational exposure to WRC are too severe not to be cause for concern. In addition, there is good evidence in the record of the dose-response relationship between occupational exposure to WRC dust and woodworkers' asthma. A study by Vedal, Chan-Yeung, Enarson et al. (1986/Ex. 1-397) shows such a relationship, with asthma beginning at a WRC level of 3.4 mg/m³ and a statistically significant reduction in forced respiratory capacity noted in workers exposed to 2 mg/m³ WRC dust or more. Harold Imbus, a physician representing the IIWDCC, stated:

This study, small though it may be, tends to support dose response, and a threshold level between 2 and 3.4 mg/m³ for the protection of effects of WRC (Ex. 8-34, p. 7).

The 1980 study by Brooks, Edwards, Apol, and Edwards found a dose-related relationship between total WRC dust level and prevalence of asthma in employees in jobs with the greatest dust exposures. The Brooks et al. study found asthma in zero percent of WRC workers exposed at 0.5 mg/m³; however, at 3.56 mg/m³, this percentage rose to 5 percent.

The United Petitioners submitted a 1988 paper by Goldsmith and Shy that

found that there is a clearly defined asthma syndrome produced by WRC (Ex. 3-362). OSHA finds these studies convincing evidence of WRC's allergenic potential; in addition, the Agency believes that a threshold for occupational asthma exists and lies between 2 and 3.4 mg/m³. Based on this evidence, OSHA concludes that an 8-hour PEL of 2.5 mg/m³ is necessary to protect workers from the significant and often permanent effects of immune-mediated occupational asthma associated with exposure to WRC dust at levels above this limit. Several record comments agree that a separate PEL for WRC dust is warranted and that the threshold level is as described above (see, for example, Exs. 8-34 (Imbus review, p. 6), 168, and 191; Tr. p. 12-292; Tr. pp. 12-317, 12-318, and 12-320).

Some commenters (Tr. p. 12-316) were of the opinion that many other woods, such as Douglas fir, pine, red and white oak, redwood, walnut, spruce, boxwood, cocobolo, teak, mahogany, and others, should also be designated by OSHA as allergenic in this rulemaking. However, OSHA finds that, as Dr. Imbus of the IIWDCC notes, "it is unlikely that species other than WRC are responsible for large numbers of cases of respiratory allergies" (Ex. 8-34, Imbus review, p. 6). The authors of the Goldsmith and Shy (1988) paper concur:

Other commonly used woods such as oak, birch, redwood, pine, teak, alder, and hemlock, produce pulmonary effects that are less well described than the asthma responses to Western red cedar (Ex. 3-362, p. 13).

The IIWDCC contends that, at the present time, there is "no consensus even as to which species should be considered allergenic" (Ex. 168). OSHA concludes that other species are somewhat allergenic. The evidence in the literature does not indicate that any other species is nearly as allergenic as

WRC or would cause nearly as high a proportion of allergic reactions among exposed workers. However, the Agency will monitor the literature on these other potentially allergenic species so that other woods with demonstrably allergenic properties can be identified in the future.

Based on the evidence presented above, OSHA is establishing a PEL of 5 mg/m³ as an 8-hour TWA and 10 mg/m³ as a 15-minute STEL for hard and soft wood dust, with the exception of Western red cedar, for which a PEL of 2.5 mg/m³ (8-hour TWA) is being established. OSHA concludes that promulgation of these exposure limits will substantially reduce the significant risk of material impairment in the form of pulmonary dysfunction (including changes in peak flow, interference with mucociliary clearance, respiratory symptoms, and chronic effects) that is associated with exposure to wood dust at the higher levels that would be permitted in the absence of any limit.

Conclusions For All Respiratory Toxicants

As Table C6-2 and the discussions above show, limits for the respiratory toxins have been established to control employee exposures to or below the airborne concentrations of these substances that have been associated with the development of acute or chronic respiratory effects. For most of these substances, the evidence is sufficient to identify the NOE or low-effect levels that are related to these effects in humans or animals.

Accordingly, OSHA concludes that maintaining employee exposures at or below these limits will greatly decrease the likelihood that employees will be at significant risk of respiratory effects when they are exposed to these substances in the workplace. Because the chronic pulmonary disease caused by exposure to toxic dusts is often

incapacitating, such exposures can effectively end the working life of severely affected individuals. Less-serious pulmonary disease can result in lost workdays, both as a result of the associated symptoms themselves and as a consequence of increased susceptibility to respiratory infections. The effects of exposure to acute pulmonary toxins, such as ozone or trimellitic anhydride, range from reduced lung function to life-threatening pulmonary edema. OSHA has determined that these adverse pulmonary effects constitute material impairments of health. Lowering the Agency's former limits or establishing limits where none previously existed will substantially reduce these significant occupational risks.

ALLERGIC RESPIRATORY EFFECTS:

Clinical condition:

Type of wood:

Hypersensitivity pneumonitis

Acute airway obstruction

Allergy of upper respiratory tract

Asthma

Mahogany, teak, okume, cedar, sequoia, cork,
maple-bark, plywood, oak
Ablruana
Arbor-vitae

Rhinitis

Iroko, red cedar, abluana, domestic sawdust,
mahogany, ramie, oak; also formaldehyde
(see dermatitis below)
Cedar, abachi, makore, iroko, okume,
manzonia, rosewood

Particle Size Distribution of Airborne Dust from the Danish Study⁽²⁰⁾

Range (μ)	Percent of Particles
< 6	33
6-10	41
11-15	11
16 >	15

Major Types of Wood Preservatives	Comments
1. Creosote	**Avoid Use if Possible
2. Pentachlorophenol (PCP)	**Avoid Use if Possible
3. Arsenicals	
Chromated copper arsenate (CCA) Ammoniacal copper arsenate (ACA) Fluor-chrome-arsenate phenol (FCAP)	*Use CCA instead
4. Acid copper chromate (ACC)	
5. Chromated zinc chloride (CZC)	
6. Bis,tri-n-butyltin oxide (TBTO)	
7. Copper naphthenate; zinc naphthenate	**Low Human Toxicity
8. Copper-8-quinolinolate	**Low Human Toxicity
9. 3-Iodo-2-propanyl butyl carbanmate	
10. Nidex-10 (Boric acid & paraffin wax)	**Low Human Toxicity