SMALL PLANTS AND THEIR MEDICAL PROBLEMS— THE FURNITURE INDUSTRY

Health Effects of Wood Dust

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Throughout his history man has had daily contact with plants and he has learned to recognize and avoid the harmful species. The first reference of adverse skin reaction to a plant—probably the pokeberry—was in the 28th century BC in China. Up to 1900 AD, there were almost 300 references in the literature of adverse skin reactions from contact with plants; most of these reactions were caused by flowering plants, not trees or wood. Skin irritation from plants is caused primarily by coarse hairs and spicules—for example, the T-shaped hairs on red-twigged dogwood and the hairs on fig leaves—which produce an erythema when the leaf is rubbed on the skin in the long axis of the hairs.

Although holly (Ilex sp.) has a spiny leaf, it has not been reported as a cause of irritant dermatitis. The leaves of white cedar and juniper, on the other hand, and leaf hairs of the plane tree (a European relative of the sycamore) can produce conjunctival and respiratory irritation. The leaf hairs of the elm (Ulnus) are reported to cause skin irritation (4). Some plants, such as the common English nettle and elm trees (which are in the nettle family), have specialized glandular hairs containing histamine and acetylcholine and can produce skin irritation. Buttercups, on the other hand, contain a lactone that produces blisters, and the essential oils in citrus fruits and the sap of fig trees have photodermatitic effects. Perhaps the most familiar allergic dermatitis is caused by poison ivy in which urushiol is the irritant (1,2).

The adverse health effects of wood and wood dust are less commonly noted than those I have just mentioned. Dermatitis from the Japanese lacquer tree—also caused by urushiol—was known in China as early as the 7th century AD. In the 17th century, the sap of the possumwood was said to cause blindness (2); the sap also contains a chemical substance that has been used as a fish poison (3). French soldiers in Napoleonic time reportedly became ill after eating foods that were cooked on spits of oleander wood; and there are references of similar events among barbecuers in California and Florida (4). All parts of the oleander contain glycosides that resemble digitalis in physiological action (3).

Manzanilla is perhaps the most notorious of all irritant trees. It grows along the seashore in Florida, South America, Central America and the West Indies; the tree has been largely eliminated in southern Florida except for the Everglades National Park. All parts of this tree are toxic, including the fruit. Some of the legends portend death to those who sleep under its shade. Direct contact of the conjunctiva with the wood, contaminated fingers or with water dripping from the tree can produce conjunctivitis, burning pain, photophobia, intense tearing, edema and complete closing of the eyelids. After 1-2 hours of dermal exposure there may be redness with smarting and burning; after 24-hours, vesicles appear that resemble 2nd degree burns. Biting the fruit without

swallowing will result in no more than soreness of the mouth and excessive salivation; if you swallow, the symptoms will include vomiting, difficulty in swallowing, pains in the mouth and abdomen, and bradycardia. Moderate inhalation of the wood dust produces cough, while more severe exposure may cause naso-pharyngitis, laryngitis and bronchitis (2,5).

The first occupational reference to wood dust was in the 18th century when Ramazzini (6) reported nose and eye irritations in pit sawyers. He didn't specify any particular wood except to mention that a cypress odor gave some woodturners headaches. In 1902 there were reports of coryzal symptoms from redwood, and throat and eye irritation from a boxwood substitute (4). Three years later there was an epidemic of such severity among Lancashire shuttle-makers using African boxwood (4,7) that the men refused to work with the wood—37 out of 112 were affected: 34 had difficult breathing; 10 had headaches; 17 had somnolence, laziness or dopiness; 14 had rhinitis; 9 had running of the eyes; 6 had sneezing; 6 had coughing; there were 4 with chest tightness and 3 with slight dyspnea; 2 had loss of appetite; 1 had nausea and vomiting; and 1 became faint. The somnolence, headaches, dyspnea and faintness were thought to be due to an alkaloid in the wood—probably quebrachamine (4), a curare-like compound—that induces a gradual slowing of the heartbeat (7). In 1906, English workmen who became ill from exposure to Gonioma (ie, S. African boxwood) were eligible for workmen's compensation; since then, this species has been replaced largely by other woods.

Another troublesome tropical African wood is Iroko (Chlorophora excelsa), which resembles teak. It is often substituted for teak and can cause epidemics of severe dermatitis, particularly during hot, humid weather or when the wood is wet. Its dust may both irritate and sensitize—the sensitizer is chlorophorin, an oxystilbene—as patients may have both severe dermatitis and/or severe respiratory symptoms (ie, obstructive rhinitis and/or asthma) (2,4,7,8). In 1941 an epidemic was reported (8) in which 50 men were working with wet Iroko logs. All suffered irritation of the exposed skin, while 9 had intense irritation of the covered skin, marked edema of the face with eye irritation, acute coryza with mild headache and mild pharyngitis or chest symptoms (ie, constriction, dry cough and dyspnea that simulated asthma). Iroko is one of the six commonest causes of dermatitis in France to this day.

The adverse effects of exotic woods are generally more severe than those of woods that grow in temperate climates; the systemic effects are often toxic and the sensitizers are more potent. One possible explanation is that their extractives or accessory substances—which serve to protect the tree against bacteria, fungi, insects and other animals—are also toxic to man. Except for manzanilla (ie, Hippomane manchinella), no systemic effects have been reported for American woods.

Most of the emphasis in the literature deals with the exotic species, yet most of the wood used in the US is native—less than 5% of the exotic hardwoods are imported into this country. By volume of use, pine and Douglas fir are our major softwoods. These, together with oak (the major hardwood), comprise better than 50% of the wood that is used in the US (Tables 1 and 2).

There is evidence of contact dermatitis, however, from exposure to a number of native American woods (Table 3). Cedar—ie, incense (9), Port Orford (2), Virginia pencil or eastern red, white (2,4) and western red (10)—pine (4,11), Douglas fir (4,12), fir (4), hemlock (13), spruce (4) and poplar (14) are definitely associated. Incense cedar (Calocedrus decurrens) and Virginia pencil cedar, or eastern red cedar (Juniperus

virginiana), are officially recognized causes of dermatitis in the German pencil industry (4). Unconfirmed cases of contact dermatitis have been reported from acacia, alder, beech, birch, chestnut, cypress, dogwood, maple and redwood (15).

TABLE 1 - Volume of Use and Decay-Resistance of Native US Softwoods*

Over 5 billion bd ft:	
Douglas fir (*Pseudotsuga menziesii*)	medium
Pine (Southern yellow) (*Pinus* sp. - 6 species)	medium-low
1.0-4.9 billion bd ft:	
Fir (*Abies* sp. - 8 species)	low
Hemlock (*Tsuga* sp. - 3 species)	low
Ponderosa pine (*Pinus ponderosa*)	low
Redwood (*Sequoia sempervirens*)	high
500-999 million bd ft:	
Western red cedar (*Thuja plicata*)	high
Pine (jack, lodgepole) (*Pinus* sp. - 2 species)	low
Northern white pine (*P. strobus*)	medium
100-499 million bd ft:	
Incense cedar (*Libocedrus decurrens*)	high
Cedar (northern & southern white, Port Orford) (*Thuja occidentalis*, *Chamaecyparis* sp. - 2 species)	high
Cypress (red, yellow, white) (*Taxodium distichum*)	medium
Western larch (*Larix occidentalis*)	medium
Idaho white pine (*Pinus monticola*)	low
Norway pine (*P. resinosa*)	medium
Sugar pine (*P. lambertiana*	low
Spruce (eastern, Engelmannn) (*Picea* sp. - 5 species)	low
Sitka spruce (*P. sitchensis*)	low
50-99 million bd ft:	
Alaska cedar (*Chamaecyparis nootkadensis*)	high
Eastern red cedar (*Juniperus virginian*)	high
Tamarack (*Larix larcina*)	medium

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*Current Industrial Report for Lumber Production and Mill Stocks (1977). Bureau of the Census. (Does not include plywood, posts, pulp, particle board, etc.)

TABLE 2 - Volume of Use and Decay-Resistance of Native US Hardwoods*

Over 1 billion bd ft:	
Red oak (Quercus sp. - 14 species)	medium-low
White oak (Quercus sp. - 16 species)	high-med.
500-999 million bd ft:	
Poplar (Liriodendron tulipfera)	low
100-499 million bd ft:	
Red alder (Alnus rubra)	low
Ash (Fraxinus sp. - 5 species)	low
Aspen (Populus sp. - 2 species)	low
Beech (Fagus grandifola)	low
Rock elm (Ulmus sp. - 4 species)	low
Gum (Liquidamber styraciflua)	low
Hickory (Carya sp. - 4 species)	low
Hard maple (Acer sp. - 2 species)	low
Maple (Oregon, soft) (Acer sp. - 3 species)	low
Tupelo (Nyssa sp. - 3 species)	?
50-99 million bd ft:	
Basswood (Tilia sp. - 2 species)	low
Birch (Betula sp. - 5 species)	low
Cottonwood (Populus sp. - 3 species)	low
Locust (Robinia pseudoacacia, Gleditsia trianthos)	medium
Pecan (Carya sp. - 4 species)	low
Sycamore (Plantanus occidentalis)	?
10-43 million bd ft:	
Walnut (Juglan nigra)	high
Soft elm (Ulmus sp. - 2 species)	low
Cherry (Prunus serotina)	high
Hackberry (Celtis sp. - 2 species)	low
Willow (Salix sp. - 2 species)	low
Butternut (Juglan cinerea)	low
1-3 million bd ft:	
Box elder (Acer negundo)	low
Buckeye (Aesculus sp. - 2 species)	low
Cucumber (Magnolia acuminata)	low
Dogwood (Cornus sp. - 2 species)	low
Magnolia (Magnolia sp. - 2 species)	low
Osage orange (Maclura pomifera)	high
Sassafras (Sassafras albidum)	high

*Current Industrial Report for Lumber Production and Mill Stocks (1977). Bureau of the Census. (Does not include plywood, posts, pulp, particle board, etc.)

TABLE 3 - American Species Causing Contact Dermatitis

Thuja plicata - Western red cedar
One case of allergic contact dermatitis (10)

Juniperus - Juniper, eastern red cedar
Cause of dermatitis in German pencil industry (9)

Libocedrus - Incense cedar
Two cases reacted to thymoquinone and hydrothymoquinone; could also be irritant; cause of dermatitis in German pencil industry (9)

Chamaecyparis lawsoniaries - Port Orford cedar
Unconvincing evidence (4)

Thuja occidentalis - White cedar
A 1926 description of dermatitis (4)

Pinus sp. - Pine
Relatively uncommon; sensitization mostly in non-American species (4,13)

Picea sp. - Spruce
Possible sensitization to hydrostilbenes as cross-reaction with stilbesterol (2,4)

Pseudotsuga mensiesii - Douglas fir
Three cases with + patch test; two had prior skin disease, two had no dust exposure (4,12)

Abies sp. - Fir
8/125 persons had + patch test, significant in 4/8; needles are common irritant (13)

Tsuga sp. - Hemlock
+ patch test in 1/125 foresters with dermatitis (13)

Populus - Poplar
One atypical case of allergic contact dermatitis; + patch test may be irritation (14)

Prosopis sp. - Mesquite
Not commonly used, except as fuel (14)

Irritant dermatitis primarily affects exposed surfaces; however, the dust may lodge on the eyelids, face, neck and moist skin folds (such as genital areas) and irritate them as well. On the other hand, high humidity in the Pacific Northwest is thought to increase the susceptibility to low-level irritants (eg, in wood, bark and needles) among forest workers, loggers, pond men and lumber handlers and is believed to be the critical factor in the induction of dermatitis among these workers (12). Splinter wounds from Douglas fir and Port Orford cedar heal slowly (2,4). Dermatitis does not seem to be as much of a problem with western red cedar as does asthma—only one case of allergic contact dematitis has been reported.

Up to the present time, asthma and rhinitis have been confirmed in over a dozen species of wood, most of which are exotic types (Table 4). The best known and most studied—including epidemiologic investigations—is an American species called western red cedar (Thuja plicata). It was reported in Japan as early as 1923, but has been well described only within the last 20 years. beta-Thujaplicin is the sensitizer in western red cedar and incense cedar. It's interesting that there are two different sensitizers in western red cedar, one for the skin (beta-thujaplicin) and one for the respiratory system (plicatic acid). Plicatic acid is also a strong organic acid, so it can act as a primary irritant. Except for some of the cancer studies, this is the only wood for which there are specific epidemiological studies.

Some of the symptoms of western red cedar exposure may take weeks or months to develop (Table 5). These symptoms—which usually appear before pulmonary function is affected—include eye and nasal irritation, nasal obstruction, sneezing, coughing, excessive rhinorrhea and chest tightness; symptomatic people generally maintain a normal baseline pulmonary function—at least in the early stages. Symptoms tend to worsen at night, but as exposure increases the symptoms move closer to the onset of exposure. Thus, if one is making a case study, he may get some individuals who have a late response, some who have an early response and some with both early and late responses. Long exposures more often give a dual response, while minor exposures usually tend toward the late response. In those individuals having western cedar asthma, the chest radiograph is normal and there is usually no relationship between skin testing and respiratory sensitivity. (A few studies in Japan have noticed some relationship between skin testing and respiratory effects, but in this country that has not been noted.) There are usually no precipitating antibodies in sensitized individuals and no apparent relationship between smoking and atopic status. In fact, there seem to be more non-smokers and ex-smokers in the case reports. Whether these people are more susceptible or this is a matter of selection, we do not know. The effects of the exposures appear to be reversible if there is no immediate reaction. If both late and early responses are evident, reversibility is less likely.

TABLE 4 - Asthma and/or Rhinitis Due to Wood Dust

Thuja (native US species) - Western red cedar, arborvitae
Immediate, late and dual reactions; confirmed by bronchial challenge (16-23)

Quercus (native US species) - Oak
Confirmed by bronchial challenge (24)

Chamaecyparis (native US species) - Port Orford cedar
Old report of asthma in woodworkers (2,4)

Hippomane (native US species) - Manzanilla, beach apple
Rhinitis; not widely used (2,4,5)

Sequoia (native US species) - Redwood
Two case reports with dual reaction; confirmed by bronchial challenge (25)

Chlorophora - Iroko, African teak
Chlorophorin is sensitizer for dermatitis; cause of industrial asthma in Belgium (2,4)

Pericopsis - Afromosia
Can produce skin and respiratory irritation, asthma and systemic symptoms (2,4)

Pterocarpus - Kejaat, African teak
Dust causes dermatitis and respiratory symptoms (26)

Dalbergia - Rosewood, cocobolo
Many members of this genus cause allergic contact dermatitis (27)

Gossweilerodendron - Nigerian cedar, Agba
Possible case of asthma (4)

Citrus - Orangewood
One unconfirmed case (2,4)

Khaya - African mahogany
Confirmed by bronchial challenge and precipitating antibody; genus could be Swietenia (4,24,27,28)

Triplochiton - Obeche, African whitewood
Confirmed by skin and inhalation test (4)

Cedra - Cedar of Lebanon
Six case reports of asthma and rhinitis (29)

Gonystylus - Ramin
Reports of asthma and dermatitis; case of extrinsic allergic alveolitis syndrome, but challenge gave reduced FEV_1 and transfer factor in 6-8 hrs (30)

Microberlinia - African zebrawood
One case of asthma with dual reaction, confirmed by challenge and immediate skin test reactivity (31)

Pouteria - Abiruana
One case with immediate reaction and one with dual reaction on challenge; both + skin tests (32)

Buxus - Boxwood
One case of asthma and cough from dust; dual response on challenge; + skin test (33)

TABLE 5 - Characteristics of Western Red Cedar Asthma

Latency - Weeks to months

Symptoms - Tearing and nasal irritation; nasal obstruction; excessive rhinorrhea; sneezing; cough; wheeze; chest tightness

Pulmonary function - Acute airways obstruction (reduced FEV_1 and FEF_{50}), baseline PFT not reduced in early stages

Onset of symptoms & reduced PFT - Symptoms worsen at night, occurring closer to onset of exposure with increasing exposure; same pattern of change in PFT on bronchial challenge, though some persons have immediate response and some have a dual response

Chest radiograph - Normal

Skin test - No relation between skin and respiratory sensitivity

Serology - No precipitating antibodies in sensitized persons

Eosinophilia - Variable increases in sensitized persons

Smoking and atopy - No apparent relation in most studies

Reversibility - May be reversible if no immediate reaction; relief from symptoms over a weekend without exposure

An allergic reaction—by definition—requires a period of previous exposure during which sensitization to the antigen takes place. Continued or additional exposure increases one's sensitivity; fortunately, only a small proportion of the exposed population is affected. The lung and skin contact the antigenic substances most often, but the allergic reaction usually is expressed by the skin. Asthma and/or rhinitis may occur alone or concomitantly with dermatitis. The early stages of allergic dermatitis (dermatitis venenata) are redness, scaling and itching, which signs initially resemble airborne contact dermatitis (4). The hands, forearms, eyelids, face, neck and genitals are affected first. Erythema and irritation may then progress to vesicular dermatitis and on to chronic dermatitis after repeated exposures.

Native American species reported to be sensitizers include: acacia, alder, ash, beech, birch, chestnut, cedar, creosote bush, elm, maple, mesquite, oak, pine, poplar, prune and spruce (14). Allergic contact dermatitis has been confirmed in several varieties of cedar (2,4,9,10), mesquite (2), pine (11), spruce (4), hemlock (13), fir (4), Douglas fir (12) and poplar (14). Skin allergens thus far identified are:

poplar: a labile quinone in the heartwood and a sesquiterpene lactone
pine: pinosylvine, delta-3-carene and coniferylbenzoate
western red cedar: beta-thujaplicin
incense cedar: thymoquinone
liverworts: sesquiterpene lactones
lichens: d-usnic acid

Creosote bush (Dictamnus) has not been confirmed as a skin sensitizer, nor is it a native of North America.

Inhalation of organic particles produces two distinct types of allergic reactions: asthma (and/or rhinitis) and extrinsic allergic alveolitis (or hypersensitivity pneumonitis) (34,35). The most common characteristics of each are summarized in Table 6.

Asthma and rhinitis have been confirmed by bronchial challenge for western red cedar (Thuja plicata) (16-22), redwood (Sequoia sempervirens) (25), and oak (species unknown) (24). Asthma has been reported in woodworkers exposed to Port Orford cedar, but it has not been confirmed by bronchial challenge (36).

Plicatic acid has been identified as the allergen in western red cedar (21). The sensitizers have not been identified for other woods. Skin tests and precipitins were negative for redwood (25) and generally negative for western red cedar (21). The characteristics of western red cedar asthma (summarized in Table 5) are similar to those for redwood asthma. One case of asthma from oak dust produced an immediate reaction (reduced FEV_1, FVC, FEF_{25-75}) on challenge to oak dust and its alcoholic extract, while other wood dusts and a water extract of oak dust were negative. Precipitating antibody against the alcoholic oak extract was found in the serum (24).

The adverse health effects of native American woods—primarily dermatitis and asthma—are summarized in Tables 7 and 8. Most species have no case reports; only western red cedar has been studied in any detail.

There are no reports of asthma or rhinitis from Douglas fir; there are reports of dermatitis, probably by contact rather than by dust. Hemlock is responsible for one case. There are no reports of dermatitis from California redwood, but there are two case reports of asthma. A couple of instances of dermatitis and over 90 cases of asthma have occurred from western red cedar.

TABLE 6 - Comparison Between Occupational Asthma and Extrinsic Allergic Alveolitis (modified from references 34, 35, 37-41)

Asthma (type I)	Extrinsic Allerg. Alv. (III, IV)
Predisposing factors	
Atopy	None known
Region affected	
Airways of lung & bronchi to terminal airways	Mid & terminal airways, also alveolar & interstitial tissue
Onset of symptoms	
Immediate (and/or late)	Usually after 4-6 hours
Systemic reaction	
None	Usual, accompanied by fever, chills, anorexia, tachycardia, tachypnea
Serology	
Elevated IgE antibodies	IgE normal; IgG precipitating antibodies may be present
Eosinophilia	
Common	Transient and uncommon
Skin tests	
Immediate wheal and flare	Edematous reaction after 4-6 hours; skin test of little value--most antigens are local irritants
Sensitivity	
Extreme sensitivity to bronchoconstrictors	
Pulmonary impairment	
Increased air flow resistance (reduced FEV_1)	Reduced FVC and FEV_1, reduced diffusion and compliance
Etiology	
Platinum salts, grain & wood dusts	Fungi, animal proteins (some induce Type I reaction) and thermophylic actinomycetes
Mechanism	
Antigenic: IgE-induced release of pharmacologic mediators from mast cells	Precipitating antigen-antibody complex and complement produce Arthrus skin reaction; Arthrus-type pulmonary lesions have not been observed
Treatment	
Reversed with adrenergic drugs; blocked by cromolyn sodium	May be reversed with steroids, not with bronchodilators

TABLE 7 - Summary of Health Effects of Native US Softwoods

Variety	Dermatitis(n)	Asthma(n)	Comments (ref.)
Douglas fir (*Pseudotsuga*)	+ (3)		Probably by contact rather than by dust (4,12)
Pine (*Pinus*)	+ (2)		Sensitizer is stilbene in turpentine (13)
Fir (*Abies*)	+ (4)		Contact, not dust (13)
Hemlock (*Tsuga*)	+ (1)		(13)
Redwood (*Sequoia*)		+ (2)	Rhinorrhea sneezing, cough, tight chest, dyspnea, dual reaction on challenge. Sequoiosis (allergic alveolitis) also described (25,42)
Incense cedar and eastern red cedar (*Libocedrus* and *Juniperus*)	+ (2)		Sensitizer is thymoquinone; also in coleus, lavender and horsemint (9)
Port Orford cedar (*Chamaecyparis*)	?	?	(4)
Western red cedar (*Thuja*)	+ (1)	+ (90^{+})	"Cedar poisoning" from lichens and liverworts. Plicatic acid causes asthma and rhinitis; beta-thujaplicin is skin sensitizer (16-22,10)
Northern white cedar (*Thuja*)	?		(4)
Larch and tamarack (*Larix*)			"Woodcutters' eczema" in Europe; urticaria in European species (2,4)
Sitka spruce (*Picea*)	+		(2,4)

+ = confirmed by patch test and/or bronchial challenge
(n) = number of cases
? = unconvincing report, unconfirmed by patch test or challenge
(ref.) = reference number(s)

TABLE 8 - Summary of Health Effects of Native US Hardwoods

Variety	Dermatitis(n)	Asthma(n)	Comment (ref.)
Oak (*Quercus*)	?	+ (1)	Sneezing, rhinorrhea, cough, precipitating antibody; immediate reaction on challenge; "woodcutters' eczema" in Europe; adenocarcinoma in European sp. (4,24)
Poplar (*Liriodendron*)	+ (2)		Sensitizer is sesquiterpene lactones (2,14)
Red alder (*Alnus*)	?		Dermatitis from European sp. (14)
Ash (*Fraxinus*)	?		Dermatitis from European sp. (14)
Aspen (*Populus*)	?		"Woodcutters' eczema" in France (36)
Beech (*Fagus*)	?	?	Vasomotor rhinitis; may be adenocarcinoma from European sp. (2,4,14)
Elm (*Ulmus*)	?		"Woodcutters' eczema" in Europe (14)
Maple (*Acer*)	?		"Maple bark strippers' disease", extrinsic allergic alveolitis from fungus (43,15,14)
Basswood (*Tilia*)	?		(2)
Birch (*Betula*)			Irritant and dermatitis in non-native sp. (14)
Locust (*Robinia* and *Gleditsia*)			"Woodcutters' eczema" in Europe (2)
Sycamore (*Platanus*)			"Woodcutters' eczema" in Europe (44)
Walnut and butternut (*Juglans*)			Contact dermatitis and contact sensitivity from European sp. (2)

Cherry (Prunus)	Prune wood may be a skin sensitizer (44)
Hackberry (Celtis)	European sp. are skin, mucosal and respiratory irritants (2,4)
Cucumber and magnolia (Magnolia)	Cross-sensitivity with compositae and sesquiterpene lactones (2)
Holly (Ilex)	"Woodcutters' eczema" in Italy (2)
Oregon myrtle and sassafras (Lauracea)	Sneezing from inhalation of pungent oils (2)

\+ = confirmed by patch test and/or bronchial challenge
(n) = number of cases
? = unconvincing report, unconfirmed by patch test or challenge
(ref.) = reference number(s)

There are many more species of hardwoods than softwoods, although their consumption is less than that of the softwoods. The only confirmed case of asthma from native American hardwoods has involved oak; the species was not identified (24).

Two other diseases that are associated with the wood industry are not caused by either wood or its dust; these are "woodcutters' eczema" and hypersensitivity pneumonitis. Fungi growing on wood bark was suspected of causing dermatitis in 1921; it wasn't until over a quarter of a century later that lichens and liverworts were shown to be the etiologic agents of "woodcutters' eczema" (4). This disease—also called "oak poisoning", "cedar poisoning", "pine poisoning", "spruce dermatitis", "spruce poisoning" and "wood poisoning" (45–49)—occurs only after one has been in a forested area for a day or two; the condition is worse in wet weather and subsides 2–4 weeks after the cessation of exposure (12,45). The respiratory system may be affected as well as the skin. The sensitizers have been identified in the liverworts (sesquiterpene lactones) and lichens (d-usnic acid). The most common cause of dermatitis may be attributed to the liverworts. The lactones in liverworts also cross-react with lactone allergens in the compositae family, among which yellow poplar and magnolia are included. Compositae is also one of the largest families of flowering plants, which may explain the reason for the large amount of "weed dermatitis" that we see. Lichens, which may consist of both fungi and algae, have a wide distribution and grow on trees. Algae too can grow on trees; some algae can cause sensitization, but the specific allergen has not been identified.

Hypersensitivity pneumonitis, or extrinsic allergic alveolitis, is similar to "woodcutters' eczema" in that it is caused by microorganisms growing on the wood. Hence, we have "maple bark strippers disease", "sequoiosis" and "woodpulp workers' disease" (34,37,43).

(See Table 6 for a comparison of occupational asthma and extrinsic allergic alveolitis.) While extrinsic allergic alveolitis may come from fungi and lichens on the wood, it also is associated with fungi on moldy hay, cheese, mushrooms and compost.

Nasal cancer is another disease that has become linked with exposure to wood or wood dust. As much as a 12-fold increase in risk has been found among woodworkers. First reported among furniture makers in the High Wycombe district of England in 1965 (50,51), the association has been confirmed by the authors (52-55) as well as by investigators in other countries—ie, Belgium (56,57,58), France (59,60,57,61), Denmark (62,63), Australia (64), Italy (65) and the United States (53)—more locally, in North Carolina (66). Originally noted in furniture makers, the disease also has been observed among woodworkers outside of the furniture industry (55,67); for example, in the boot and shoe industry (68). A reduced risk was observed in the United States (69,70) and Canada (71,72).

The incidence of these tumors was highest from about 1920-1940, during the advent of power machinery but prior to the use of exhaust ventilation.

In their ecological study of cancer rates in 132 US counties—in which 1% or more of the total population employed in furniture and fixtures manufacturing—Brinton et al (67) found excesses of melanoma (SMR=109) and multiple myeloma (SMR=109) in addition to an excess of nasal cavity and sinus cancers (SMR=119). Out of 19 sites, these were the only SMR's greater than 100. Other mortality ratios were less than 100.

The same investigators—using death certificates—conducted a case-control study (66) of nasal cancer in North Carolina counties, again based on at least 1% of the total population being employed in furniture and fixtures manufacturing. Of the 37 persons who had died from cancer of the nasal cavity and sinuses, there were 22% involved in furniture manufacturing compared to 6.8% of the controls. Those having other exposure to wood (carpenters, sawyers, lumbermen and loggers) were 13.5% vs 9.6% for the controls. The odds ratio in the matched triplet analysis was 4.4 (1.3-5.4 at the 95% confidence interval) for furniture workers and 1.5 (1.4-4.3, 95% confidence interval) for other woodworkers. The histogenesis of 13/37 indicated that: Four tumors were adenocarcinomas, three of which were associated with the furniture industry. Because of the limited amount of occupational data on death certificates, "further study is needed to clarify the risk of nasal cancer in US furniture workers and to identify the specific carcinogens involved" (66).

Milham (69) conducted a proportionate mortality study of men in the forest products industry of Washington State who had died from cancer between 1950 and 1971. He found no excess mortality from either nasal or nasopharyngeal cancer among any of the occupational groups within the industry; the PMR for all malignant neoplasms among loggers and carpenters was 93 and 106, resp., and was no more than 100 for the other groups. The PMR for stomach cancer was slightly increased in all groups. PMR's for Hodgkin's disease, multiple myeloma and leukemia were generally higher in all occupations except loggers, sawmill and other mill workers. Milham has suggested that ingested wood particles may cause stomach cancers and that the "physical and chemical breakdown products of wood may be carcinogenic." This conclusion is similar to that of Morton concerning the association between tannins and cancer (73).

Milham (70) also examined the mortality patterns of the AFL-CIO United Brotherhood of Carpenters and Joiners of America; the members of this union were largely construction workers and had been exposed to asbestos. He found high SMR's for

pleural mesothelioma (182—due to asbestos exposure), Hodgkin's disease in men over age 60 (160); lymphosarcoma (168) and lymphatic leukemias in men less than 50 years of age (375). Small elevations were observed for cancer of the lung (100), stomach (112), small intestine (102), and prostate (103). The SMR for all causes was 81, 95 for all malignant neoplasms and 76 for neoplasm of the nasopharynx.

At least 28 compounds of known structure have been identified in 454 species of Spermatophyta and Pteriodophyta (75). These compounds include alkaloids, tannins, nitroso compounds, triterpene glycosides, parascorbic acid, podophyllotoxin, rotenone, safrole and shikimic acid. Several possible human carcinogens from wood have been discovered since 1976; the most likely agents are summarized in Table 9.

TABLE 9 - Possible Carcinogens Found in Wood

Tannins (hydrolyzable and condensed)
In oak (hydrolyzable form occurs primarily in wood, while the condensed form is mainly in the bark), maple bark, birch, beech, sweetgum, horse chestnut, hickory, pecan, butternut, sassafras, hackberry, sycamore, willow, aspen and cottonwood; not in walnut, poplar, box elder, elm, ash and basswood (77)

Sinapaldehyde and coniferaldehyde
3,4,5-trimethoxycinnamaldehyde (TCMA), may be a derivative of sinapaldehyde and coniferaldehyde; has produced nasal squamous carcinomas in rat; sinapaldehyde is found primarily in hardwoods, coniferaldehyde in softwoods; both occur in maple, oak, sweetgum, walnut and butternut (77,78,79,80)

2,6-Dimethoxy-1,4-benzoquinone
Oxidation product of sinapaldehyde; produces carcinomas in animals (79)

Podophyllotoxin
In Juniperus sp. (81)

Quercetin
Mutagenic by Ames test; in oak (heartwood and sapwood), sweetgum and Osage orange (heartwood) (77,82)

Safrole
Weak hepatocarcinogen; converts to proximate carcinogen, 1-hydroxysafrole; found primarily in root bark of sassafras (77,78)

Tannins are generally found in heartwood and outer bark. Being polyphenols, they are capable of forming multiple hydrogen bonds with proteins, which property makes them useful in the tanning industry (77). Structurally, they are of two types:

Hydrolyzable - As esters of a sugar (usually glucose) with one or more gallic acid groups, they are readily hydrolyzed by acids or enzymes to the sugar and the phenolic acid moiety (eg, gallic or ellagic acid). These tannins are known as tannic acid and are usually obtained commercially from nutgall. Tannic acid has been topically applied to burns; however, patients so treated often develop liver necrosis. Animal studies have since shown that tannic acid is an hepatocarcinogen, as well as an oncogenic promoter of 2-acetylaminofluorene (77,81).

Condensed - These flavanoid polymers contain only phenolic moieties and polymerize rather than hydrolyze in acid solution. The flavanoid precursors account for most of the yellow, red and blue colors in flowers and fruits. Condensed tannins and related polyflavanoids are quite common extractives, particularly in bark (77).

Morton (83) has suggested that the excessive incidence of esophageal cancer in Curacao and South Carolina may be due to the unusually large dietary intake of condensed tannins and anthocyanins from tea, herbal remedies, wine, peanuts, and the like. Plant extracts rich in phenols (especially, the catechin type of condensed tannins and related anthocyanins) have produced tumors in rats. No tumors were evident from a tannin-free extract (74,75,83). Tannin-containing extracts from chestnut (Castonea), valonea (Quercus) and mimosa (Acacia) produced either sarcomas or liver tumors in mice. Hydrolyzable tannins gave only liver tumors, whereas condensed tannins produced both liver tumors and sarcomas at the injection site (75).

TMCA (3,4,5-trimethoxycinnamaldehyde), a p-O-methyl derivative of sinapaldehyde—a lignin found mainly in hardwoods and hardwood smoke—induces a variety of tumors in the rat, including nasal squamous carcinomas. The oxidation product of sinapaldehyde (2,6-dimethoxy-1,4-benzoquinone), also found in wood, induces sarcomas at the site of its subcutaneous injection in rats and mice (79). Coniferaldehyde, along with sinapaldehyde—a normal constituent of lignin—is predominant in softwoods (Juniperus and Larix) but is also present in hardwoods (76,80). TMCA as such is not present in wood lignins, but it may form metabolically and should, therefore, be considered as a wood carcinogen (76,79).

Podophyllotoxin and related lignins have been found in Juniperus virginiana and certain other Juniperus species. It is the active constituent of podophyllin, which has been used as a cathartic, a remedy for condyloma acuminata and as an antitumor agent. Applied to the mouse uterus or given in the diet, it can produce hyperplastic lesions and occasional tumors. It is also the agent in cedar bedding that is likely to cause the increased incidence of spontaneous tumors in mice (78,84).

Quercetin, a flavanoid related to tannins and one of the most common phenolic compounds in vascular plants, has been shown to be mutagenic in Salmonella typhimurium; its mutagenic activity is about tripled in the presence of liver microsomes. Several related flavanoids and flavanoid metabolites, however, show no mutagenic activity either with or without metabolic activation (82).

Safrole is a major constituent of the oil of sassafrass and a very minor constituent of

many other wood species. It was long used in soft drinks (root beer and sarsaparilla), candy and baked goods; tea made from the oil was used as a stimulant, diuretic, carminative, diaphoretic, a treatment for bronchitis, a sudorific for colds and as a home remedy or spring tonic. Oil from the root bark of sassafrass is 80% safrole; it was banned by the FDA for use as a food additive when 0.5% safrole was found to produce liver tumors in the rat. The actual carcinogen is probably a metabolic intermediate, such as 1-hydroxysafrole (77,78,85-87).

* * *

In summary, four types of adverse health effects have been shown to be associated with wood or wood dust:

systemic effects (ie, headache, nausea and sleepiness);

irritation, leading primarily to dermatitis;

sensitization (ie, allergic contact dermatitis, rhinitis and asthma) and

cancer (confirmed nasopharyngeal and suspected GI cancers).

Systemic effects are not linked to any commercial North American species. Asthma has been confirmed in three North American species (oak, redwood and western red cedar), dermatitis is certain for 7 species (Douglas fir, pine, fir, hemlock, eastern red cedar, western red cedar and poplar). The prevalence of asthma and dermatitis is unknown; the only epidemiologic studies thus far are those with western red cedar. The association between wood dust and cancer needs further study.

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