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Recommendation from the Scientific Committee on Occupational Exposure Limits: Risk assessment for Wood Dust

Substance

Woods are customarily divided into two types: hardwood and softwood. Softwoods are generally conifers (pine, fir, spruce, cedar, etc.) and hardwoods come from deciduous trees (oak, beech, ash, silver birch, etc.) and from certain tropical species (mahogany, teak, etc.). This distinction is purely botanical, softwoods corresponding to the gymnosperms and hardwoods to the angiosperms, and certain characteristics such as the density and hardness of the two types are largely superimposed (Fengel et al., 1989). Wood consists essentially of cellulose, hemicellulose and lignin. The compounds that can be extracted with solvents represent less than 1% of the wood's mass and are mainly aliphatic substances (fatty acids and glycerides), terpenes and phenolic compounds. The biological and physical properties of softwood and hardwood dusts are largely the same (Fengel et al., 1989). Several studies have shown that most of wood dust's mass consists of particles with aerodynamic diameters equal or greater than 10 µm (Chung et al., 2000, IARC., 1995, Pisaniello et al., 1991, Whitehead et al., 1981 b). The size of the particles constituting the wood dust depends on the type of operation carried out on the wood; sawing produces particles with larger volume than does sanding. On the other hand, the particle size does not seem to depend systematically on the type of wood. For a given process, several studies report very similar particle size distributions between pine and oak; others report that dust from hardwood is finer than dust from softwood (IARC, 1995). Depending on the treatments to which the wood has been subjected before machining, the dust can contain additives, preservatives or adhesives.

Individual measurements are made by passing samples through a filter at a known flow rate; the mass of aerosol sampled is calculated from the difference between the weights of the filter after and before sampling, and the concentration is obtained by dividing this mass by the volume of air sampled. These gravimetric determination methods are standardised (INRS, 2000), but the particulate matter suspended in the surroundings of workplaces can come from greatly diverse sources and contains a variable mixture of wood dust, dirt, sand, ash, bio-aerosols, particles from exhaust gases and other suchlike substances. The measurement methods customarily used cannot distinguish between the various elements that constitute inhalable dust and at present no method is known for selectively determining the quantity of wood dust in the total dust. Accordingly, the nature of the dust collected is determined from knowledge about the workplaces in question and about the sampling conditions.

UTILISATION/EXPOSURE

According to data from the European CAREX project, around 2.5 million workers in Europe work in situations where they are exposed to wood dust. This represents between 1% and 3% of the workforce in the European Union countries; the percentage is highest in Spain (3.3%) and in Finland (3.0%). These estimates relate to a fixed time and take no account of occupational history. Considering the exposure frequency among the controls in case-control investigations among the general population for whom a recapitulation of occupational careers was carried out, it is probable that the percentage of workers who have had at least one job involving exposure to wood dust during their working life is close to 15% for men and 5% for women (IARC, 1998).

Until recently, only "total dust" was measured for wood dust exposure. In this document, all figures are expressed as "total dust", unless otherwise stated.

The exposure levels determined vary considerably depending on the branch of industry and the workplace involved (Table 1). These variations are explained by differences in the wood treatment process (cutting method, tool rotation speed, etc.), the type of wood (green or seasoned, softwood or hardwood, panels, etc.) and the ventilation mechanisms. The measurements made in France and collected by the INRS (COLCHIC database) indicate that, although the exposure levels were reduced between 1987 and 2000, they still remain high, with over 50% of the samples having contained more than 1 mg/m^3 in 2000. The highest levels are found in cabinet making, factories producing furniture and seats, and plants manufacturing fibreboard or plywood. In these branches the workplaces exposed to the highest concentrations are those involving scraping or grinding, sanding, and turning on a lathe.

TABLE 1 : Concentrations of wood dust in wood industries

Industry and operation (Country)	No of measur[t]	Mean (mg/m^3)	Range (mg/m^3)	Year
Sawmills (Canada) (Vedal et al., 1986)	78	0,2[c]	nr-6	1982-
Sawmills (Canada) (Demers et al., 2000)	220	1,0[c]	0,03-25,4	1996
Log yard	23	1,5[c]		
Cut-off/debark	26	0,5[c]		
Sawmill	62	0,8[c]		
Lumber yard	24	1,4[c]		
Planer	63	0,9[c]		
Cleanup	16	2,9[c]		
Sawmills (New Zealand) (Douwes et al., 2000)	37	0,3-1,7[c]	0,1-5,8	1998
Sawmills (Australia) (Alwis et al., 1999)	93	4,81[a]/1,59[c]	0,25-74	1998
Joineries (Australie) (Alwis et al., 1999)	66	7,59[a]/3,68[c]	0,21-51	1998
Sawmills (Canada) (Teschke et al., 1994)	224	0,51[a]/0,12[c]	0,08-52	1989
Sawmills (Denmark) (Vinzents et al., 1993)	85	0,54[d]	0,51-0,59[c]	?
Furniture (UK) (Jones et al., 1986)				1983
Conversion	43	2,3[a]	1,0-4,8	
Component making	106	3,4[a]	0,3-53	
Assembly	60	7,0[a]	0,5-27	
Plywood mills (Finland) (Kauppinen, 1986)				
Sawing of veneers	3	1,6[a]	0,6-3,0	1965-1974
Sawing of veneers	4	1,3[a]	1,1-1,5	1975-1984
Sawing of plywood	6	3,3[a]	0,5-12	1965-1974
Sawing of plywood	11	3,7[a]	0,3-19	1975-1984
Sanding of plywood	5	3,0[a]	0,3-6,4	1965-1974
Sanding of plywood	21	3,8[a]	0,8-22	1975-1984

Joineries, furniture factory (Netherlands) (Scheeper et al., 1995)	199	2,95[a]/2,10[c]	nr	1994
Furniture factories (Denmark) (Schlunssen et al., 2001)	54	1.14[c,e]/1.99[c,f]	nr	1997-1998
Manual sanding	143	0.85[c,e]/1.40[c,f]	nr	
Sanding, cutting	53	0.78[c,e]/1.27[c,f]	nr	
Automatic sanding	492	0.70[c,e]/1.12[c,f]	nr	
Cutting	259	0.46[c,e]/0.69[c,f]	nr	
Handling and assembling (ww dep[t])	459	0.46[c,e]/0.70[c,f]	nr	
Handling and assembling (other dep[t])	151	0.69[c,e]/1.09[c,f]	nr	
Mixed work task				

[a] Arithmetic mean, [b] Median, [c] Geometric mean, [d] Mean of geometric means, [e] Total dust, [f] Inhalable dust

Health significance

Animal data

Exposure by instillation

Several experimental studies (Bhattacharjee et al., 1979, Tatrai et al., 1995, Yuan et al., 1990) carried out on various species (guinea pigs, Wistar rats, Sprague-Dawley rats) have indicated that various wood dusts in solution instilled via the nasal or intra-tracheal route have a fibrosing effect on the lungs.

Exposure by inhalation

The results of experimental studies (Drettner et al., 1985, Guney et al., 1987, Holmstrom et al., 1989, Klein et al., 2001, McMichael et al., 1983, Wilhelmsson et al., 1985 b, Wilhelmsson et al., 1985 c), in which animals were exposed to wood dust and various additives via the respiratory route (PCP, formaldehyde, chromates, lindane), did not give clear evidence that wood dust has a carcinogenic or co-carcinogenic effect. These studies, however, have numerous limitations: insufficiently large groups, too short exposure time, and insufficiently clear presentation of the results. Only in one study (Holmstrom et al., 1989) were female Sprague-Dawley rats exposed over a long period (exposure duration 2 years), but the sample was small (total of 64 animals divided into 4 groups: controls, formaldehyde only, wood dust only and formaldehyde + wood dust). A single nasal tumour (squamous cell cancer) was observed and this occurred in the group exposed to formaldehyde alone. One study by Klein *et al.* (2001) was carried out on a sizeable population (471 female Fisher-344 rats divided into 7 groups), but the duration of exposure between 25 and 45 weeks was short and the reporting of data showed some inadequacies.

Exposure via the cutaneous route

One study revealed the carcinogenic role of untreated wood dust extracts administered cutaneously to young NMRI mice (25 to 30 g). In this study a dose-response relationship was shown. This study does not, however, enable the carcinogenicity of wood dust as such to be evaluated (Mohtashamipur et al., 1989).

Genotoxicity tests

According to IARC (1995), constituents of beech that can be extracted with polar organic solvents are genotoxic, as demonstrated by the induction of point mutations in bacteria, DNA single strand breaks in rat hepatocytes *in vitro* and micronuclei in rodent tissues *in vivo*. Extracts of oak wood showed similar activity, but fewer data were

available. Extracts of spruce, the only softwood tested, gave consistently negative results.

A few recent studies have revealed the genotoxic effect *in vitro* of dust from various types of wood: beech (Mohtashamipur et al., 1990,) and more recently eastern white pine (Mark et al., 1995 b) and southern yellow pine (Mark et al., 1995 a).

All in all, the contribution of experimental studies in animals is limited. There are few such studies, and these were often carried out on small populations and after exposure times which were too short. Overall, these studies do not reveal any carcinogenic or co-carcinogenic role of wood dust in animals, and the rare recent studies do not cast doubt on the conclusions established by the IARC in 1995 concerning the evaluation of animal experimental data (IARC, 1995). The conditions of inhalation and the deposition of particles in the upper respiratory tracts may be very different in animals and in humans; this could result in a different mode of action or in reduced sensitivity in experimental animals. Moreover, no study provides data enabling a dose-effect relationship for sino-nasal cancer in humans to be established.

Human data

Non-carcinogenic effects on the upper respiratory tract

Dahlqvist *et al.* (1996) exposed 19 healthy volunteers to Scotch pine dust for 5 hours through a respirator fitted with a filter in 10 subjects, and with no filter for the other 9. The respective time-weighted averages (TWAs) were 0.04 mg/m^3 and 0.13 mg/m^3. No difference was observed in the functional respiratory parameters after exposure, but the cell and interleukin-6 concentration in nasal lavage was significantly elevated in both groups and more markedly so in the group using respirators without filters.

Wilhelmsson and Lundh (1984) compared the anatomical and pathological characteristics of nasal biopsies in 45 employees (of furniture and flooring factories) and 17 employees of a hospital. The mean exposure level was 2.0 mg/m^3 and the duration ranged from 1 to 39 years (mean = 15 years). Cuboidal metaplasia was more frequent in exposed workers than in the controls (26/45 compared with 4/17; $p < 0.005$). Similar results were published by Boysen *et al.* (Boysen et al., 1986), who carried out biopsies in 44 workers who had been exposed for 10 to 43 years to softwood dust and in 37 controls matched for age. The biopsies of the exposed subjects showed a higher degree of metaplasia (measured by a histological score) than did the controls (mean score: 2.0 compared with 1.4; $p < 0.05$), which increased with the duration of exposure. Four subjects having been exposed (for 20 years in one case and for over 26 years in the other three) showed dysplastic lesions, while there were none in those not exposed (p according to the unilateral Fisher test = 0.08). Furthermore, nasal symptoms were more frequent in exposed (14%) than in control subjects (4%). A few years earlier the same author had published a study (Boysen et al., 1982) involving 103 furniture factory employees in Norway and 54 controls; the frequency of metaplastic lesions was 40% in exposed workers and 17% in the controls. Such anatomical and pathological modifications of the nasal mucosa (metaplasia) were observed some distance away from the tumoral site, in 19 out of 22 patients studied by Wilhelmsson *et al.* (1985 a) who were affected by adenocarcinoma of the ethmoid associated with wood dust.

Holmstrom *et al.* (1988) carried out a study of 100 furniture factory employees in Sweden and 36 controls who were office workers. The mean exposure level of the furniture factory workers to wood dust was 1.65 mg/m^3. Compared with the controls, the exposed subjects showed symptoms of the upper and lower respiratory tracts significantly more often. A reduction of mucociliary clearance was observed in 15% of exposed workers and in 3% of the controls, and the results of an olfactory test were significantly altered in the exposed group in comparison with the control group.

Pisaniello *et al.* (1991) reported the frequency of respiratory nasal and ocular symptoms determined in 168 furniture factory employees in Australia and 46 controls (hospital maintenance employees) in 1989. The mean exposure levels ranged between 3 mg/m^3 and 5 mg/m^3. The employees exposed to wood dust showed significantly more frequent nasal symptoms (obstruction, discharge, nose blowing: between 35% and 51% compared with 11% to 30%) and attacks of ocular irritation (35% compared with 20%) than did the controls. Ahman *et al.* (1995) studied the symptoms reported by 130 woodwork teachers and 112 employees of the Stockholm public schools who were not exposed to wood dust. The exposure levels were not measured. Compared with the controls, the teachers exposed (for the most part to pine and less frequently to linden, juniper or alder wood dusts) complained much more often of ocular disorders (irritation: odds ratio = 3.9; 95% CI = 2.0 - 8.3), nasal disorders (obstruction: odds ratio = 12; 95% CI = 5.7 - 28), pharyngeal disorders (irritation: odds ratio = 5.9; 95% CI = 2.5 - 16) and chronic bronchitis (odds ratio = 12.4; 95% CI = 3.0 - 110).

Numerous other studies have also shown a significant increase in symptoms of the upper respiratory tract in workers exposed to wood dust compared with control workers. The symptoms vary a great deal and include epistaxis, sinusitis, long-lasting colds, nasal obstruction, stasis of the nasal mucus (reduction of nasal clearance), cytological modifications of the mucus and histological modifications of the nasal epithelium. These symptoms occur at relatively low wood dust concentrations (from 1 mg/m^3 upwards), and several studies have demonstrated a dose-effect relationship. Although not very severe, they represent an important functional inconvenience for workers. Two studies (Holmstrom et al., 1997, Rabone et al., 1999) have also shown that daily nasal lavage with an isotonic solution reduces the inconvenience and alleviates these symptoms.

Non-carcinogenic effects on the lower respiratory tract

Several epidemiological studies have investigated the effects of exposure to wood dust on the lower respiratory tract. The signs and symptoms frequently observed in exposed workers are numerous: asthma, coughing, chronic bronchitis, alteration of the respiratory function parameters, idiopathic pulmonary fibrosis, extrinsic allergic alveolitis, etc.

The studies on asthma related to exposure to wood dust, although numerous, are often limited to case descriptions which include neither control groups nor exposure measurements. Nevertheless, they reveal that most wood species can induce occupational asthma. A recent study in New Zealand of 769 employees of five sawmills processing pine wood showed an increased frequency of self-reported asthma among exposed employees (18%, n=704) in comparison to the general population (12.1%, n=592, OR (95% CI): 1.6 (1.1-2.3)) (Douwes et al., 2001). The levels of wood dust were not measured individually, and the employees were classified according to their

work tasks. However, the levels of exposure measured in two of the five sawmills were low: the geometrical means varied between 0.3 and 0.7 mg/m^3, with less than 30% of the measurements higher than 1 mg/m^3 (Douwes et al., 2000). In comparison to the non-exposed workers (9.2%, n=65), asthma was also more common in the low exposure group (15.6%, n=294) and in groups exposed to high levels of 'green dust' (20.4%, n=212) and 'dry dust' (18.8%, n=198). Moreover, eye and nose irritations were significantly more prevalent in the high and low exposure groups.

Schlunssen *et al.* (2001, in press 2001) carried out a study of 2 033 employees of 54 furniture factories processing pine wood and 474 workers from 3 control factories in Denmark. The exposure levels, measured individually for 1 579 employees, were low: the arithmetic mean was 1.19 mg/m^3 (SD: 0.86 mg/m^3). The results did not show a significant increase of prevalence of self-reported asthma among exposed employees (6.3% for non-smokers and 15.1% for smokers) compared to controls (4.7% for non-smokers and 11.7% for smokers). However, the study revealed a significant exposure-response relationship between inhalable dust concentration (classified in 3 exposure categories: lower than 0.74 mg/m^3, 0.74-1.42 mg/m^3 and greater than 1.42 mg/m^3) and asthma symptoms (night chest tightness and night wheeze) among woodworkers. Among female woodworkers, a positive exposure-response relationship was also found for self-reported asthma as well as for physician-diagnosed asthma. Furthermore, woodworkers showed an increased frequency of daily coughing.

These results show that wood dust-induced asthma is not limited only to the dust from western red cedar (*Thuya plicata*). The latter species, however, has been the one most studied and the study by Vedal *et al.* (Vedal et al., 1986), which has been confirmed by other investigations, shows that asthma related to dust from red cedar is frequent: between 5% and 15% of the cases occur at low dust levels (median = 0.21 mg/m^3; mean = 0.46 mg/m^3) and that its frequency increases with the duration of exposure (5% for less than 4 years, 6% for 5-9 years, 10% for 12-19 years and 12% for over 20 years). However, western red cedar, native to the western coastal forests of the United States and Canada, has been planted very little in Europe, except for some experimental plantations in France, Belgium and the UK. It is essentially an imported wood, mainly for outdoor furniture, and its use is limited in Europe, although no precise consumption data were found.

Several studies have established that other parameters apart from asthma are found to be altered in explorations of the respiratory functions in workers exposed to wood dust, and that they evidence an exposure-effect relationship. After taking age and tobacco into account, the exposure-related alterations variously include a reduction of the Forced Expiratory Volume in one second (FEV_1), a reduction of the Forced Vital Capacity (FVC), a reduction of the FEV_1/FVC ratio and a decrease of the CO diffusion coefficient.

Whitehead *et al.* (1981a) carried out a study of 1 157 employees of several factories using wood in New England. They all answered a questionnaire on their occupational history and their smoking history, and all underwent respiratory function tests. All subjects exposed to finished products, to green wood, to adhesives for plywood and to metals, etc. were excluded. The subjects were divided according to the type of wood to which they had most frequently been exposed (hardwood: mainly maple and a little oak and ash; softwood: white pine) and were classified in three exposure categories

according to the processes applied at their workplaces: low (0 to 2 mg-year/m^3), intermediate (2 to 10 mg-year/m^3) and high ($\geq$ 10 mg-year/m^3). The results showed a significant reduction of the FEV_1/FVC ratio and the MMEFR (*Maximal Mid-Expiratory Flow Rate*) and identified a dose-effect relationship for both types of wood dust. Taking a spirometric parameter lower than the 5^{th} percentile of the distribution of this parameter in a normal population as abnormal, the results were similar and showed odds ratios, associated with the high-exposure category compared with the low-exposure category, of between 2 and 4. Regardless of the type of wood dust (softwood or hardwood), the risk of having a low FEV_1/FVC ratio or MMEFR was 2 to 4 times greater for subjects exposed to more than 10 mg-year/m^3 than for those exposed to less than 2 mg-year/m^3, after adjustment for age and for smoking.

Hessel *et al.* (1995) evaluated the respiratory function of 94 employees of a sawmill in Alberta that only cuts pine and spruce, and 165 employees in the oil industry. The levels of wood dust with diameter smaller than 10 µm (thoracic fraction) were measured at 5 different places (mean = 1.35 mg/m^3; range = 0.1 - 2.2 mg/m^3). Most of the respiratory symptoms were significantly more often reported by those exposed to wood dust than by the controls, and spirometric parameters of those exposed were altered (reduction of the FEV_1 and of the FEV_1/FVC). As regards chronic bronchitis, the odds ratio adjusted for age and smoking associated with exposure for more than three years was 2.14 (95% CI = 1.02 - 4.52).

Noertjojo *et al.* (1996) carried out a study of 243 workers in a sawmill in Vancouver working exclusively with red cedar. These workers, who were followed up for periods of between 4 and 13 years (mean 7.5 years), were compared with office-worker controls investigated under the same conditions. Asthmatic subjects were excluded from the analysis. The FEV_1 and the FVC were measured at each follow-up visit (1982, 1983, 1984, 1988 and 1993 for the subjects exposed, and 1978, 1980, 1982, 1985, 1989 and 1991 for the controls). The sawmill workers were classified in three exposure-level groups according to the geometrical mean of the individual measurements carried out by employment category, which was weighted according to the time spent in this category. The exposure categories low, medium and high corresponded to mean exposure levels of < 0.2 mg/m^3, between 0.2 and 0.4 mg/m^3 and > 0.4 mg/m^3, respectively. The main results showed a significant year-by-year reduction of the FVC accompanied by an exposure-effect relationship (-10.9 ml/year, -15.8 ml/year and -21.3 ml/year respectively for the three exposure categories compared with the controls) after adjustment for age, smoking and initial FVC. Similarly, there was a significant year-by-year reduction of the FEV_1 of the exposed workers compared with the controls, although no dose-effect relationship could be determined. This study shows that chronic exposure to red cedar dust at relatively low levels can induce an alteration of the lung function, even without asthma.

Mandryk *et al.* (1999) carried out a study in Australia of the parameters of the respiratory function tests and respiratory symptoms on 168 employees of sawmills, plants producing shavings and carpentry shops and in 30 controls. The mean exposure levels were measured individually and were respectively 4.8 mg/m^3, 3.2 mg/m^3 and 7.6 mg/m^3. All the parameters studied (FEV_1, FVC, FEV_1/FVC, chronic coughing, chronic bronchitis, blocked nose) were significantly altered in the workers exposed compared with the controls. The dose-response relationships between the individual exposure measurements and the various parameters were significant and most marked in

carpentry shop workers (Pearson's correlation coefficient between -0.34 and -0.65 for the carpenters).

A study (Imbus et al., 1988), carried out on 176 employees of wood fibreboard panels in the USA exposed to low concentrations (between 0.1 and 0.6 mg/m^3) of wood dust, showed no alterations of the respiratory function test parameters. Only 4 subjects (2.3%) showed a FEV_1 reduction greater than 10% between the beginning and end of the working week, and 18 subjects (10.2%) showed a corresponding reduction between 5% and 10%. This is one of the only studies carried out on a relatively large population to have indicated an absence of pulmonary effects due to wood dust exposure. However, no indications as to characteristics of the subjects are given (age, length of service, smoking, etc.) and no control group was used. Moreover, the levels measured were remarkably low and this could explain why no effects were revealed.

Two case-control investigations carried out in England (Hubbard et al., 1996, Scott et al., 1990) established that exposure to wood dust was a risk factor for idiopathic pulmonary fibrosis (also known as cryptogenic fibrosing alveolitis). After adjustment for age and smoking, the odds ratios associated with occupational exposure to wood dust were 2.9 and 3.8 respectively in the two studies. In the second study the fraction attributable to wood dust was 7.2%. No indication of exposure levels was given in these two studies.

Several studies report cases of extrinsic allergic alveolitis (pneumopathy due to hypersensitivity) related to wood dust exposure. This pathology, the best-described form of which is farmer's lung, is related to the inhalation of antigens that induce a hypersensitivity reaction with cellular mediation. It is characterised by the appearance of symptoms such as coughing, fever, discomfort and dyspnoeia that occur a few hours after exposure to the allergen; if exposure ceases, the symptoms regress spontaneously in a few days. Numerous allergens of fungal origin have been incriminated in extrinsic allergic alveolitis among woodworkers: *rhizopus rhizopodiformis* (Belin, 1987), *aspergillus* (Enarson et al., 1990), *thermoactinomyces vulgaris* (Enarson et al., 1990), *penicillinium* (Dykewicz et al., 1988), *cryptostroma corticale* (Baur et al., 2000, etc.). This pathology is fairly frequent among sawmill workers: in Sweden the prevalence is 5 to 10% (Belin, 1987) and in the order of 5% in England (Halpin et al., 1994). No study that relates the risk of this pathology's appearance to exposure level is known.

In general, the published studies clearly indicate that the frequency of allergic and non-allergic respiratory manifestations due to exposure to wood dust, whether to softwood or to hardwood, is significantly elevated in workers exposed to concentrations of 1 mg/m3 and upwards but still well below 5 mg/m^3.

Non-carcinogenic effects on the skin

Dermatoses induced by wood dust show no particular characteristics. They are related to irritant or allergic phenomena or both, and are caused by direct contact of the skin with the dust. In general, they are limited to the most exposed areas (hands, forearms, face, neck).

Numerous studies report the existence of various cutaneous pathologies (contact or allergic eczemas) in workers exposed to wood dust (Cook et al., 1997, de Cock et al., 2000, Estlander et al., 1999, Flechsig et al., 1990, Gan et al., 1987, Goldsmith et al.,

1988, Hinnen et al., 1995, Hjorth, 1979, Jagels, 1985, Lachapelle, 1986, Meding et al., 1996 a, Meding et al., 1996 b, Mitchell et al., 1974, Pires et al., 1999, Stingeni et al., 1998, Thigpen et al., 1989, Watsky, 1997, Woods, 1987, Woods et al., 1976). Most woods seem capable of inducing these cutaneous manifestations of irritant or allergic origin (Woods et al., 1976). A few rare studies report estimates of the prevalence of skin lesions in various groups of workers exposed to wood dust; these range between 4% and 15% of workers, depending on the study and the wood being worked. On the other hand, none of the studies consulted has shown how exposure levels are related to the frequency at which such skin manifestations occur.

Carcinogenic effects - Sino-nasal cancer

Sino-nasal cancer is a rare form of cancer (depending on the country, there are 0.5 to 1.5 new cases per year per 100 000 in men and 0.1 to 0.6/100 000 in women). The incidence varies markedly from one country to the next and, within the same country, from one region to another (IARC, 1977). Although the role of genetic susceptibility cannot be discounted, it can in no way explain this variation of incidence. In particular, the low incidence reported in the United States (0.6/100 000 in men) compared with that in many European countries (e.g. 1.2/100 000 among men in France) cannot be explained by genetic differences, since the populations of the United States and Europe are intimately related at the genetic level. Thus, the main factor that can explain this variation is certainly of environmental origin.

The causal role of exposure to wood dust in the genesis of sino-nasal cancer has long been unambiguously established by numerous epidemiological studies carried out in populations of varying geographical origin, which were exposed for different periods and in several fields of activity. The data from the main studies concerning the carcinogenic role of occupational exposure to wood dust have been pooled and reanalysed by Demers *et al.* (Demers et al., 1995 a, Demers et al., 1995 b); the results of these pooled analyses have also been the subject of a technical report by the International Agency for Research on Cancer (IARC, 1998).

Demers *et al.* (1995 a) carried out a pooled analysis of 5 cohorts of workers exposed to wood dust (Acheson et al., 1984, Blair et al., 1990, Miller et al., 1994, Robinson et al., 1986, Roscoe et al., 1992). The first two cohorts consisted of workers employed in the furniture-making industry (England and the United States), the third of employees in the wooden automobile production industry in the United States, and the last two of workers in American plywood factories. After updating of the follow-ups, the regrouped cohort consisted of 28 704 people among whom 7 665 deaths had been observed, including 1 726 resulting from cancer and 11 from sino-nasal cancer. The jobs occupied were classified in three categories according to the probability of exposure to dust: possible, probable and certain. A significant excess number of deaths from sino-nasal cancer (11 cases; Standard Mortality Ratio SMR = 3.1; 95% Confidence Interval 95% CI = 1.6 - 5.6) was found, with a clear increase of the SMR as a function of the exposure probability (possible: 1 case; SMR = 0.8; 95% CI = 0.0 - 4.6, probable: 1 case; SMR = 1.2; 95% CI = 0.0 - 6.5, and certain: 9 cases; SMR = 8.4; 95% CI = 3.9 - 16.0). The excess risk was limited to workers in the furniture industry and no sino-nasal cancer deaths were observed in the plywood industry cohorts. The excess risk was limited to workers who had begun their employment before 1940 (before 1940: 9 cases; SMR = 12.5%; 95% CI = 5.7 - 23.7) and to those whose exposure had begun more than 20 years earlier (20-29 years: 3 cases; SMR = 2.6; 95% CI = 0.5 - 7.6, $\geq$ 30 years: 8 cases;

SMR = 7.6; 95% CI = 3.3 - 15.0). In this pooled analysis the results were strongly influenced by the number of deaths from sino-nasal cancer in the English group of furniture industry workers (10 out of the 11 deaths from sino-nasal cancer). The main limitations of this study, despite the regrouping into 5 cohorts, were: (*i*) the small number of cases, which made it virtually impossible to carry out analyses in sub-groups (exposure duration, latency period, exposure category); (*ii*) the use of death certificates whose information is not very reliable and not very precise (no histological type) for this kind of cancer; (*iii*) the absence of data on measurements of exposure to wood dust, which was only evaluated indirectly by virtue of the job titles.

Demers *et al.* (1995 b) analysed the pooled data from 12 case-control investigations carried out in various countries. The regrouped sample consisted of 930 patients with sino-nasal cancer (680 men and 250 women) and 3 136 controls (2 349 men and 787 women). The occupational histories were coded and seven occupation categories were set up. The exposure levels were determined by a job-exposure matrix and classified in 4 categories (none, low, medium and high) corresponding approximately to estimated concentrations equal to zero, less than 1 mg/m^3, between 1 and 5 mg/m^3 and above 5 mg/m^3. The distribution of histological types was as follows: in men, 169 adenocarcinomas (25%) and 329 squamous cell cancers (48%); in women 26 adenocarcinomas (10%) and 101 squamous cell cancers (40%); this distribution varied markedly from one study to another. The proportion of adenocarcinomas was distinctly higher in the studies carried out in France (49%), Italy (between 22% and 69%) and the Netherlands (25%) than in those carried out in the USA (between 3% and 14%).

Adenocarcinomas: the results showed that there is a sizeable risk of adenocarcinoma in men with a wood-related job (odds ratio = 13.5; 95% CI = 9.0 - 20.0); this risk was particularly high in the case of cabinetmakers and men employed in furniture factories (odds ratio = 41.1; 95% CI = 24.5 - 68.7). No increase in the risk of adenocarcinoma was shown for lumberjacks, foresters and employees in paper pulp plants. The risk for sawmill employees was intermediate (odds ratio = 19.7; 95% CI 11.1 - 35.1) and slightly lower after eliminating those who had worked in furniture factories (odds ratio = 14.9; 95% CI = 8.0 - 28.7). For men, the risk of adenocarcinoma increased with the intensity of exposure (odds ratio = 0.6; 95% CI = 0.1 - 4.7 for low exposures, odds ratio = 3.1; 95% CI = 1.6 - 6.1 for moderate exposures and odds ratio = 45.5; 95% CI = 28.3 - 72.9 for high exposures). The analysis of risk as a function of exposure duration showed a clear relationship, with a large increase of the odds ratios for adenocarcinomas in men employed in a job subject to exposure (odds ratio = 1.08; 95% CI = 1.07 - 1.09 per year, odds ratio = 5.3; 95% CI = 2.5 - 11.1 for duration shorter than 5 years, odds ratio = 10.7; 95% CI = 5.2 - 11.8 for duration of 10 to 19 years, and odds ratio = 36.7; 95% CI = 22.0 - 61.3 for duration of 30 years or more); the risk was high for even short exposure duration (< 5 years). The data also indicated a long latency period, in the order of at least twenty years. The results for women were less conclusive, showing a smaller increase in the risk of adenocarcinoma for women with wood-related jobs (odds ratio = 2.78; 95% CI = 0.75 - 10.3) As with men, the risk was greatest for women employed in furniture factories (odds ratio = 4.6; 95% CI = 1.16 - 18.3). No increase in risk was observed with increase in the intensity of exposure in women, regardless of the histological form. However, the small number of cases precluded detailed analysis.

Squamous cell cancers: The results were more ambiguous for squamous cell cancers than for adenocarcinomas. The risk for women only was approximately doubled,

particularly for women who had worked in moderately or highly exposed jobs; an exposure-effect relationship was evident for the exposure duration. These results, however, were based on only small populations. For men, the risk of squamous cell cancer was not related to being exposed neither at the job, nor to the intensity or the duration of exposure. Overall, the results showed risk estimates for the squamous cell carcinoma forms to be distinctly lower than those for the adenocarcinomas. A review of recent studies, some of which were not included in the re-analysis by Demers *et al.* (1997), shows that exposure to softwood dust results in a higher risk of sino-nasal squamous cell cancers than does exposure to hardwood dust, but the amplitude is lower than that observed between hardwood dust and sino-nasal adenocarcinoma.

The main advantage of this study of data pooled from 12 case-control investigations is that it offers sufficient statistical power to realistically examine the risks according to histological type, sex, work, exposure category and exposure duration. However, there was marked heterogeneity between the studies and the histological types. Thus, the excess risks reported for all the histological types together could be explained largely by the results relating to adenocarcinoma, for which the association with exposure to wood dust is much stronger than in the case of squamous cell forms. Even though the results for adenocarcinoma were on the whole coherent with respect to the studies included, the risk was much higher in Europe (and especially France and Italy) than in North America and China. This difference could be related to the levels of exposure or to the types of wood used, although no data on the type of wood used were available in the pooled analysis to confirm this hypothesis. However, hardwoods are more widely used in Europe, especially in southern countries.

All in all, these two re-analyses indicate extremely high relative risks for sino-nasal cancers (of the order of a few units to a few tens). Thus, the differences of incidence between countries and regions are probably explained by differences in the frequency of exposure and/or by the characteristics of exposure to wood dusts (levels, type of wood).

Adenocarcinoma represents a variable proportion of sino-nasal cancers (between 10% and 50% depending on the country); the link between the onset of this histological form and exposure to wood dust is very clear, more so than in the case of the squamous cell form. The association seems particularly strong with dust from hardwood. A large proportion of the adenocarcinoma cases included in the studies published were related to exposure to hardwood dusts and the case-control investigations in which the type of wood used was noted confirm the stronger association with hardwood dust than with softwood dust. It is virtually impossible, however, to distinguish the respective role of each type of wood in the genesis of sino-nasal cancer. On the one hand, few studies have recorded the necessary information and, on the other, most often both types of wood are used in furniture factories and in carpentry and cabinet-making workshops, the fields of activity in which the risks are highest. The results of some studies with workers exposed solely or mostly to softwood dusts are conflicting, but it seems that such exposure is associated with a smaller increase in the risk of sino-nasal cancer, relating mainly to squamous cell cancers or cancer forms other than adenocarcinoma.

Additives and wood preservatives have been referred to as playing a role in the genesis of sino-nasal cancer. This is difficult to establish, and has not been taken into account in

most studies. However, since such studies have been carried out over different periods and in different places with respect to different industrial processes, it is quite improbable that wood treatment with chemicals would play a major role in the larger number of sino-nasal cancer cases having been observed in workers exposed to wood dust.

As is usual in carcinogenesis problems related to environmental exposure, it is impossible to be certain about the existence of a mechanism, with or without a threshold, in the genesis of sino-nasal cancer related to wood dust.

Carcinogenic effects - Other cancers

Nasopharyngeal cancer: several studies (Hardell et al., 1982, Kawachi et al., 1989, Ng, 1986, Olsen et al., 1984, Sriamporn et al., 1992, Vaughan et al., 1991, West et al., 1993), described in the CIRC monograph (IARC, 1995), have reported a link with wood-related jobs. This association has also been suggested by the pooled analysis of 5 cohorts of woodworkers cited earlier (IARC, 1998), The SMR was determined to be of 2.37 (9 cases; 95% CI = 1.09 - 4.51), and its value is greater in subjects whose probability of exposure to wood dust is highest (SMR = 5.3; 95% CI = 1.7 - 12.4). The excess risk would seem to be limited to workers who started their jobs at least 30 years before the onset of the cancer, and before 1950. However, possible confusion factors (in particular, exposure to formaldehyde and to chlorophenols) have not been taken into account and the largest study, in which exposure to wood dust and to formaldehyde was estimated (Olsen et al., 1984), did not confirm this association (odds ratio = 0.4; 95% CI = 0.2 - 1.0).

More recently, Vaughan *et al.* (2000) carried out a case-control investigation in 196 incident cases of cancer of the nasopharynx that occurred between 1987 and 1993 in the USA. The exposure of the subjects to formaldehyde and wood dust was determined, for each job, by two industrial hygienists who were “blind” concerning the status (case or control) of the subjects. For wood dust the odds ratio associated with having had at least one job subject to exposure was non-significant when adjusted for the non-occupational confounding factors (odds ratio = 1.5; 95% CI = 0.7 - 3.3) and the adjustment for exposure to formaldehyde reduced the odds ratio (odds ratio = 1.2; 95% CI = 0.5 - 2.7). No significant trend of the odds ratio was found to be a function of either the wood dust exposure level, the exposure duration or the cumulative exposure.

The IARC monograph made the overall conclusion that the studies published suggested a causal link between occupational exposure to wood dust and nasopharyngeal cancer, but that this had not been definitely established. The studies published since then have not changed this conclusion. Be that as it may, the relative risks or odds ratios seem lower than those that quantitate the association with sino-nasal cancer, and can be explained by factors other than wood dust.

Lung cancer: the pooled analysis of the 5 cohorts (IARC, 1998) did not suggest any increase in the risk of lung cancer related to wood dust (575 deaths from lung cancer observed, compared with 721 expected; SMR = 0.80; 95% CI = 0.73 - 0.87); smoking was not taken into account and it is possible that smoking plays the part of a negative confounding factor (owing to the fire risk involved, the proportion of smokers among workers exposed to wood dust could be lower than in the general population). Recent studies (Bruske-Hohlfeld et al., 2000, Robinson et al., 1996, Stellman et al., 1998,

Wang et al., 1995) are not very conclusive and suffer from important limitations. The positive studies indicate small risks (relative risks always lower than 2), which may be explained by a failure to fully control for confounding factors.

Laryngeal cancer: several case-control studies related to laryngeal cancer (Bravo et al., 1990, Flanders et al., 1982, Maier et al., 1992, Muscat et al., 1992, Pollan et al., 1995, Vaughan et al., 1991, Wortley et al., 1992, Wynder et al., 1976, Zagraniski et al., 1986, Zheng et al., 1992) have demonstrated an association of this cancer with exposure to wood dust or with wood-related occupations. On the other hand, the findings of cohort studies on the same subject yield consistently non-significant results, and the result of the pooled analysis of 5 cohorts (IARC, 1998) was also negative Other more recent studies (Gustavsson et al., 1998, Laforest et al., 2000) have also not found such a link.

Cancers in other locations: various epidemiological studies have considered the relationship between exposure to wood dust and cancers in other locations. In those which indicate a significant association, the excess risk is most often low, with odds ratios or SMRs of the order of 2. These low risk levels can be explained by confounding factors not having been taken into account, or incompletely so.

In its monograph (IARC, 1995) the IARC concluded that there was insufficient proof of a causal relation between occupational exposure to dust and the genesis of cancers of the nasopharynx, the oropharynx, the hypopharynx, the lungs, the stomach, the colon, the rectum, the lymphatic organs and the haematopoietic system. More recent studies published subsequent to the IARC monograph do not call its conclusions into question.

DOSE-RESPONSE RELATIONSHIP - QUANTITATIVE RISK ASSESSMENT

Sino-nasal cancer

As regards the risk of sino-nasal cancer, a quantitative risk assessment cannot be considered to be realistic because of the lack of good-quality quantitative data on exposure levels associated with increased risks. The most reliable data concerning the association between exposure to wood dust and the risk of sino-nasal cancer come from case-control studies among the general population, in which the individual exposures of the subjects included were evaluated retrospectively by experts (review of the questionnaires case-by-case by industrial hygienists or by job-exposure matrices). This cannot provide the quantified cumulative exposure data required for a quantitative risk assessment.

Respiratory tract effects

Published studies at best indicate the average levels of exposure (with sometimes an indicator of dispersal and the range), and health effects are rarely reported in relation to the exposure levels. It is thus impossible to know to what precise levels of wood dust workers presenting health impairments were exposed. Moreover, exposure varies widely according to time, protective equipment and work tasks, whereas most of the measurements are not representative of these circumstances. Because of this lack of sufficient exposure data, a formal quantitative evaluation of the risks is not feasible. Instead, we classified expected health effects according to three different thresholds: 0.5, 1 and 5 mg/m^3, and considered only studies reporting average levels of exposure lower than these values. Tables 2, 3, and 4 were constructed according to the level of

the central indicator of exposure (arithmetic mean, geometric mean or median) in the published data.

0.5 mg/m^3 threshold (TABLE 2)

Very few studies have been conducted for workers exposed to average concentrations of wood dust lower than 0.5 mg/m^3. The main reason is that such low levels are rarely observed in the wood industry (see Table 1). At these levels, dust of western red cedar induces effects on the lower respiratory tract (asthma, bronchial hyperreactivity and lung function impairment). The effects observed in respect of other species of wood demonstrate an action of wood dust on the lower and upper respiratory tracts; however, the clinical importance of these health effects (local increase of inflammation markers) is minor. Only two studies demonstrated a significant association between low exposure level and health effects having clinical importance: these two studies were both conducted among workers exposed to western red cedar dust, and no studies were available to assess health effects of dust from other wood species at these exposure levels.

1 mg/m^3 threshold (TABLE 3)

Studies reporting average exposures of between 0.5 and 1 mg/m^3 are also rare, since exposure is usually higher than at these levels. Several studies evidenced an increase in the frequency of sino-nasal symptoms and effects on the lower respiratory tracts (cough, chest tightness, lung function impairment and asthma). Overall, workers exposed to wood dust concentrations of between 0.5 and 1 mg/m^3 exhibited significant health impairments; this was demonstrated by several studies conducted among workers exposed to dust from various species of wood.

5 mg/m^3 threshold (TABLE 4)

For exposures higher than or equal to 1 mg/m^3, the effects on health are clear and include various symptoms of the disturbances of the upper respiratory tract, a significant alteration of respiratory function parameters and asthma. Moreover, several studies clearly showed a dose-effect relationship between exposure levels and the frequency of respiratory problems, and between the duration of exposure and a decrease of the respiratory function.

Recommendation

Exposure to wood dust was shown to be associated with an increase of sino-nasal cancers, though this is a rare cancer. Impairment of respiratory function and increased prevalence of pulmonary symptoms were also observed in humans after exposure to wood dust.

Distinction between softwoods and hardwoods

As regards the risk of **sino-nasal cancer,** it seems that hardwood dusts are particularly dangerous, as they are probably also more dangerous than softwood dusts with respect to adenocarcinomas. However, as has already been stressed, it is impossible at the

moment for two essential reasons to clearly identify the particular role of each type of wood in the genesis of cancer: (*i*) too few studies have addressed this problem and (*ii*) both types of wood are usually used in most wood-related fields of activity and workers have been exposed to both of them.

As regards the risk of **non-carcinogenic effects on the respiratory tracts**, practically all types of wood can cause various pulmonary symptoms in exposed workers, although only some, red cedar in particular, have been studied in any detail. However, the large number of studies involving red cedar should not be taken to imply that the dust of this species is the only one responsible for asthma and non-allergic affections of pulmonary function. It is very probable that similar results for respiratory effects due to the dusts of other wood species would be found if they had been studied in sufficient detail. This is suggested by the numerous descriptions of asthma cases related to occupational exposure to dust from many types of wood.

Irrespective of their already emphasised inadequacies, the results of experimental studies in animals provide no conclusive argument to justify a distinction between effects specific to softwood dusts and other effects specific to hardwood dusts.

With regard to currently available data and with a view to protecting the health of workers, all in all it does not seem pertinent to distinguish between softwood and hardwood dusts.

Particle size considerations

In the last decade, the rationale for sampling particle sizes relevant to expected health effects has gained recognition with the adoption of health-related aerosol size fraction definitions by the *International Organization for Standardization* (IOS, 1991), the *American Conference of Governmental Industrial Hygienists* (ACGIH, 2000) and the *Comité Européen de Normalisation* (CEN). Three particle size distributions relevant to different capture areas of the human respiratory tract are distinguished: inhalable (any particles which enter the nose and mouth; 50% capture of 100 μm aerodynamic diameter particles); thoracic (particles which pass the larynx; 50% capture of 10 μm particles) and respirable (or alveolar: particles which enter the alveolar region; 50% capture of 4 μm particles). Occupational exposure standards specifically indicating these dust fractions should have two major advantages: reducing the variability due to fluctuations in the size distributions sampled and targeting the appropriate risk factors for wood dust-related disease.

However, most epidemiological studies have not assessed exposure-response relationships using particle size-selective measurements.

Considerable uncertainties exist to establish valid conversion ratios from total dust (as measured by the equipment used for most epidemiological studies), to an inhalable dust level, which is the most appropriate size fraction for the mass effects of exposure to wood dust. However, the available data suggest that a numerical value of an OEL expressed as "inhalable dust" may be set at approximately twice the numerical value for the corresponding limit value for "total dust" (see Annex).

The mechanism underlying carcinogenesis by wood dust has not yet been elucidated. The few positive results in genotoxicity tests were obtained mainly with extracts of woods. The hypothesis of physico-mechanical cancer induction has not been clearly demonstrated by experiments.

The studies available do not provide adequate information for setting a health-based limit value for the protection of workers exposed to wood dust.

Taking into account the uncertainties and limitations of the available studies, it can be stated that exposure above 0.5 mg/m^3 induces pulmonary effects and should be avoided. Exposure levels lower than 0.5 mg/m^3 were associated with the induction of bronchial asthma only when the exposure was to western red cedar dust. The level of 0.5 mg/m^3 (total dust) and 1 mg/m^3 (inhalable dust) is probably below the levels to which the cases of sino-nasal cancers had been exposed.

TABLE 2: Studies reporting mean exposure levels lower than 0.5 mg/m^3

STUDY	STUDY POPULATION	WOOD TYPE	DUST* CONC. (MG/M^3)	RESULTS
			SINO-NASAL SYMPTOMS	
(Dahlqvist et al., 1996)	E: 19 healthy volunteers	Pine	0.04[a] (n=10) 0.13[a] (n=9)	Increase of concentration of interleukin-6 in nasal lavage fluid (0.5 to 5.9 pg/ml)
			PULMONARY SYMPTOMS	
(Johard et al., 1992)	E: 19 C: 25	-	0.25[b] (IQR: 0.22-0.34)	Increase of concentration of albumin (53 vs. 34 mg/l, p<0.001), fibronectin (80 vs. 41 µg/l, p<0.001) and hyaluronan (42 vs. 27µg/l, p<0.001) in bronchoalveolar lavage No difference in lung function (VC, FEV_1, TLC, RV, TL_{CO})
(Vedal et al., 1986) (Chan-Yeung et al., 1984)	E: 652 C: 440	WRC	0.46 (range: 0-6) (median = 0.21) 10%>1 mg/m^3	Decline of FEV_1 and FVC Bronchial hyperreactivity in nonatopic subgroup: 19.6% vs. 4.4%, p<0.001 Asthma: 10.4% vs. 4.3%, OR=2.7, p<0.001 Work-related asthma: 4.1% vs. 1.6%, OR=2.7, p<0.05
(Noertjojo et al., 1996)	E: 243 C: 140	WRC	low: <0.2 medium: 0.2-0.4 high: >0.4	Decreased FVC: compared to controls: -10.9, -15.8 and –21.3 ml/yr for low, medium and high exposure

*: total dust concentration unless otherwise specified
a: controlled exposure
IQR: Inter Quartile Range
FEV_1: forced expiratory volume in one second
TL_{CO}: diffusion capacity
E: Exposed ; C: Controls
b: median
VC: vital capacity
TLC: total lung function
WRC: Western Red Cedar
c: geometric mean
FVC: forced vital capacity
RV: residual volume

TABLE 3: Studies reporting mean exposure levels lower than 1 mg/m^3

STUDY	STUDY POPULATION	WOOD TYPE	DUST* CONC. (mg/m^3)	RESULTS
			SINO-NASAL SYMPTOMS	
(Ahman et al., 1996)	E: 39 C: 32	Various	0.57 (range: 0.12-1.18) Resp. fract.: 0.10 (0.02-0.21)	Increased frequency of nasal symptoms Decline of mucociliary clearance
(Douwes et al., 2001) (Douwes et al., 2000)	E: 704 C: 65	Pine	between 0.3 and 0.7 30% > 1 mg/m^3	Eye and nose irritations significantly more prevalent in high and low exposure groups
			PULMONARY SYMPTOMS	
(Eriksson et al., 1997)	E: 38 C: 217	Pine	0.6 (range: 0.1-4.6)	Reduced preshift lung function values
(Schlunssen, 2001) (Schlunssen et al., In Press 2001)	E: 2381 C: 619	Pine	0.72 (range:0.1-3.9) 1.19# (range:0.2-9.8)	No significant increase of declared asthma prevalence: 6.3% and 15.1% for non-smoking and smoking exposed employees vs. 4.7% and 11.7% for non-smoking and smoking controls Night chest tightness: 0-0.7 mg/m^3: OR=1 (Ref.); 0.7-1.4 mg/m^3: ORa=1.4 (0.7-3.0); >1.4 mg/m^3: ORa=2.2 (1.0-4.9) Night wheeze: 0-0.7 mg/m^3: OR=1 (Ref.); 0.7-1.4 mg/m^3: ORa=2.2 (1.2-4.0); >1.4 mg/m^3: ORa=2.2 (1.1-4.4)
(Douwes et al., 2001) (Douwes et al., 2000)	E: 704 C: 65	Pine	between 0.3 and 0.7 30% > 1 mg/m^3	Asthma: 18% vs. 9.2%, OR adjusted (95% CI): 1.6 (1.1-2.3) Cough: non-exposed: 12.3%; low: 25.9% (OR adjusted (95% CI)): 2.7 (1.2-6.5); high green: 46.2% (ORa=5.2 (2.1-13.0)); high dry: 32.3% (ORa=3.3 (1.4-7.9))

*: total dust concentration unless otherwise specified
#: inhalable dust
E : Exposed ; C : Controls
a: controlled exposure
b: median
c: geometric mean
IQR: Inter Quartile Range
VC: vital capacity
FVC: forced vital capacity
FEV_1: forced expiratory volume in one second
TLC: total lung function
RV: residual volume
TL_{CO}: diffusion capacity

TABLE 4a: Studies reporting mean exposure levels lower than 5 mg/m^3 – Sino-nasal symptoms

STUDY	STUDY POPULATION	WOOD TYPE	DUST* CONC. (MG/M^3)	RESULTS
			SINO-NASAL SYMPTOMS	
(Holmstrom et al., 1988)	E: 100 C: 36	Particle-board	1.65 (SD: 1.06)	Nasal discomfort: 53% vs. 25%, p<0.001 Eye discomfort: 21% vs. 6%, p<0.05 Reduction of mucociliary clearance (>20min): 15% vs. 3%, p=0.07
(Holness et al., 1985)	E: 50 C: 49	Various	1.83 (SD: 1.51) Resp. fraction: 0.29	Rhinitis: 32% vs. 10%, p<0.05 Eye irritation: 20% vs. 6%, p<0.05
(Wilhelmsson et al., 1984)	$E_{heavy/mod.}$: 484 $E_{light/no}$: 192	Various	2.0 (range: 0.30-5.06)	Nasal hypersecretion: 20% vs. 12%, p<0.05 Nasal obstruction: 40% vs. 30%, p<0.05 More than two colds/yr: 21% vs. 9%, p<0.05 Reduction of mucociliary clearance (>20 min): 54% (n=61)
(Halpin et al., 1994)	E_{HIGH}: 58 E_{LOW}: 45 C: 52	Softwood	Low: 0.74[c] High: 2.87[c]	No difference between low exposure group and control group Work-related nasal and eye symptoms greater in the high exposure group
(Pisaniello et al., 1991)	E: 168 C: 46	Various	Hardwood: 3.8[#] Softwood: 3.3[#]	Regular blocked nose: 51% vs. 30%, p<0.05 Three or more nasal symptoms: 35% vs. 15%, p<0.05 Eye irritation due to work in the past year: 35% vs. 20%, p<0.05 Regular ear inflammation or ear infections: 15% vs. 4%, p<0.05
(Norrish et al., 1992)	E: 44 C: 38	Various	3.6[b#] (range: 1-25) 32% > 5 mg/m^3	Nasal obstruction: 61% vs. 21%, OR=6.0 (2.0-18.2), p<0.01 Nasal discharge: 41% vs. 13%, OR=4.6 (1.4-16.4), p<0.01
(Andersen et al., 1977, Andersen et al., 1976)	E: 68	Various	63% > 5 mg/m^3 28% > 10 mg/m^3	Mucostasis (>40 min): 1.0-2.9 mg/m^3: 11%; 3.0-4.9 mg/m^3: 25%; 5.0-6.9 mg/m^3: 31%; 7.0-9.9 mg/m^3: 45%; > 10 mg/^{m3}: 63%

*: total dust concentration unless otherwise specified
#: inhalable dust
E : Exposed ; C : Controls
WRC: Western Red Cedar
OTH: Other types of wood
a: controlled exposure
b: median
c: geometric mean
IQR: Inter Quartile Range
VC: vital capacity
FVC: forced vital capacity
FEV_1: forced expiratory volume in one second
TLC: total lung function
RV: residual volume
TL_{CO}: diffusion capacity

TABLE 4b: Studies reporting mean exposure levels lower than 5 mg/m^3 – Pulmonary symptoms

STUDY	STUDY POPULATION	WOOD TYPE	DUST* CONC. (mg/m^3)	RESULTS
			PULMONARY SYMPTOMS	
(Brooks et al., 1981)	E_{WRC}: 74 E_{OTH}: 58 C: 22	WRC Various	4.7 (resp. fract.:0.2) 1.3 (resp. fract.:0.16)	Occupational asthma: E_{WRC}: 13.5%; E_{OTH}: 5.2%; C:0% Any pulmonary disease: E_{WRC}: 33.7%; E_{OTH}: 34.4%; C:16.0%
(Hessel et al., 1995)	E: 94 C: 165	Spruce and pine	Resp. fraction: 1.35 (range: 0.1-2.2)	Reduced FEV_1, FEV1/FVC Pulmonary symptoms more prevalent: +shortness of breath: 28.7% vs. 12.2%, age- and smoking-adjusted OR=2.8; 95% CI: 1.5-5.5 +bronchitis: OR_a for sawmill workers exposed > 3 yr = 2.14 (1.02-4.52) +asthma : OR_a for sawmill workers exposed > 3 yr = 3.67 (1.00-13.5)
(Holmstrom et al., 1988)	E: 100 C: 36	Particle-board	1.65 (SD: 1.06)	Deep airway discomfort: 39% vs. 14%, p<0.01 Reduced FVC, no decline of FEV_1
(Holness et al., 1985)	E: 50 C: 49	Various	1.83 (SD: 1.51) Resp. fraction: 0.29	Fall in FVC over the work shift: -2.4% vs. –0.15, p<0.01 Fall in FEV_1 over the work shift: -2.5% vs. +0.15, p<0.01 Inverse relationship between cumulative dust exposure index and FEV_1: r=0.51, p<0.001
(Halpin et al., 1994)	E_{HIGH}: 58 E_{LOW}: 45 C: 52	Softwood	Low: 0.74[c] High: 2.87[c]	No difference between low exposure group and control group Work-related cough and wheeze greater in the high exposure group
(Al Zuhair et al., 1981)	E: 204 C: 47	Various	between 0.46 and 8.3 (resp. fract.: 0.08-0.44)	Significant decline during the work shift of FEV_1 and FVC
(Mandryk et al., 1999)	E: 168 C: 30	Various	Sawmills: 4.8[#] (Resp. fract.: 0.37) Joineries: 7.6[#] (Resp. fract.: 0.67)	FVC (% pred. adj. for age, height and smoking): 84.7% (SD=0.7) vs. 94.9% (3.8), p<0.0001 FEV_1 (% pred. adj. for age, height and smoking): 84.7% (1.2) vs. 93.1% (2.8), p<0.0001 Significant cross-shift decrements for FVC (E: 3.5% vs. C: 2.1%), FEV_1 (5.7% vs. 1.8%), for FEV_1/FVC (2.9% vs. 0.1%) adjusted for age, height,

				smoking, duration of exposure and inhalable dust Regular cough: 61.0% vs. 23.5%, p<0.001 Chronic bronchitis: 30.3% vs. 11.8%, p<0.05 Work-related cough: 32% vs. 0%, p<0.01
(Norrish et al., 1992)	E: 44 C: 38	Various	3.6[b#] (range: 1-25) 32% > 5 mg/m^3	

*: total dust concentration unless otherwise specified
#: inhalable dust
E : Exposed ; C : Controls
WRC: Western Red Cedar
OTH: Other types of wood
a: controlled exposure
b: median
c: geometric mean
IQR: Inter Quartile Range
VC: vital capacity
FVC: forced vital capacity
FEV_1: forced expiratory volume in one second
TLC: total lung function
RV: residual volume
TL_{CO}: diffusion capacity

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Annex I

CONSIDERATIONS ON PARTICULATE MATTER IN ORDER TO ESTABLISH SIZE-SELECTIVE OEL VALUES FOR WOOD DUST

There now exist harmonised, internationally accepted standards. In recent years, studies of the application of particle size-selective sampling criteria for occupational aerosol exposure assessment. have yielded an extensive body of new knowledge that is now beginning to influence occupational hygiene practice in many industries. It all began in the late 1970s and took root in the early 1990s, culminating in a wide degree of international acceptance. These are reflected in the harmonised recommendations of the International Standards Organisation (ISO), the Comité Européen de Normalisation (CEN) and the American Conference of Governmental Industrial Hygienists (ACGIH) that currently prevail (1-3). These criteria identify target performance curves for samplers for collecting the 'inhalable', and the progressively finer thoracic and respirable, particle size fractions. Each such curve prescribes the target sampler efficiency as a function of particle aerodynamic diameter for representative worker breathing and external wind conditions.

The CEN document EN-481 defines conventions for particle size fractions which are also to be used in assessing the possible health effects resulting from inhalation of airborne particles in the workplace. These conventions are defined for the inhalable, thoracic and respirable fractions; which one is most appropriate and their choice will depend on the region of effect of the component of interest in the airborne material particles.

The convention definitions are:

Inhalable fraction: The mass fraction of total airborne particles which is inhaled through the nose and mouth. The inhalable fraction depends on the speed and direction of the air movement, on breathing rate and on other factors.

Thoracic fraction: The mass fraction of inhaled particles penetrating beyond the larynx.

Respirable fraction: The mass fraction of inhaled particles penetrating to the unciliated airways.

The graphic representation of these conventions is shown in Figure 1.

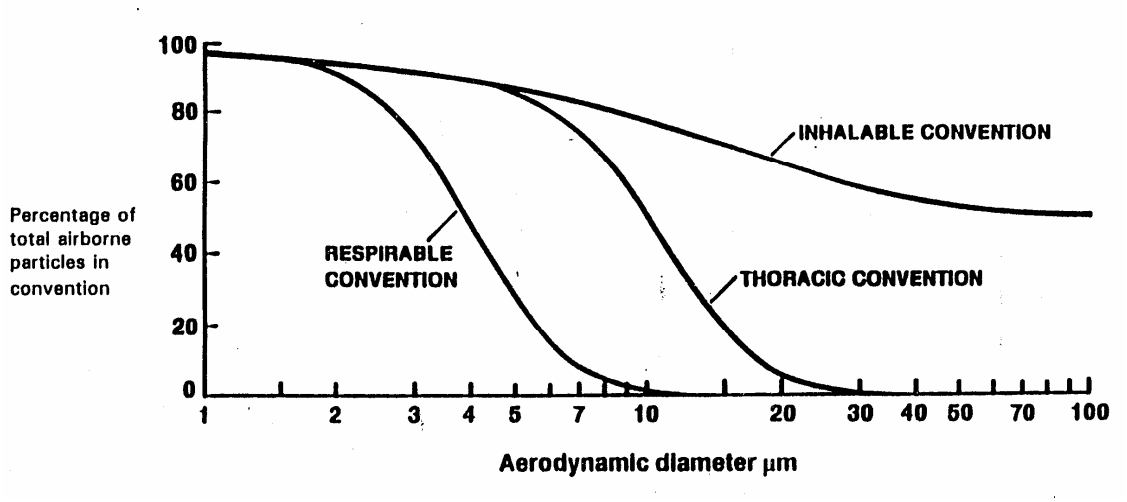

Figure 1: The inhalable, thoracic and respirable conventions as percentages of total airborne particles

In order to examine whether the most representative personal sampler instruments used by the Member States of the European Union fit with the inhalable curve (see Figure 1), a collaborative European study (4) of different personal inhalable aerosol samplers was conducted in a wind tunnel in 1997 and the results are shown in Table 1.

Table 1. Correction factors recommended to obtain satisfactory performance

Sampler type	Correction factor 0.5 ms^{-1}	Correction factor 1.0 ms^{-1}
IOM	0.9 (filter+cassette) 1.0 (filter only)	1.0 (filter*cassette)
Seven-hole	1.0	1.2
GSP	1.0	1.0
PAS-6	1.0	1.25
PERSPEC	1.0*	†
CIP10-I	1.15	1.15
37-mm open face	1.15	1.15
37-mm closed face	1.0	1.2

* Inlet losses recovered and included in sample; † Inlet losses not recovered

On the basis of the results of this wind tunnel test, a standard has been published by CEN (5).

The sampler types chosen for testing were all commercially available instruments, and the majority are either statutory or recommended methods within the member countries represented in the project consortium, i.e. instruments used in workplaces for testing compliance with the OELs.

The IOM (Institute of Occupational Medicine) personal inhalable sampler and the seven-hole personal sampler are both used in Great Britain. The GSP (Gesamtstaubprobenahme an der Person) personal sampler is used in Germany. The PAS-6 personal sampler is used in the Netherlands. The PERSPEC personal sampler is used in Italy. The CIP10-I personal sampler is used in France. The open-face 37 mm polystyrene cassette and the closed-face 37 mm polystyrene cassette are used in Sweden and Spain, respectively.

The test showed that the IOM and GSP samplers were the two which best followed the sampling convention for inhalable dust. The open-face and closed-face filter cassettes were found to have similar sampling efficiencies, but for particle sizes large than ≅ 45μm the sampling efficiencies were very low, of the order of 10 percent of the total airborne particle concentration. In this study it was also observed that the closed-face 37 mm cassette fitted well with the thoracic convention curve for a particle size of around 30μm.

One of the conclusions arising from this study was that these personal samplers and filter cassettes had to be tested in the field.

During the 1990s several workplace comparisons between personal sampling of "total dust" (as defined by filter cassettes) and inhalable dust were published for various industrial environments: borate processing plants, bakeries, the woodworking industry, aluminium welding in repair shops, aluminium foundries, lead battery production plants, electrode paste plants, nickel mines and processing plants, metal machining shops, and lead smelters. All the results, except those pertaining to fumes, showed that the inhalable concentrations were greater than the corresponding "total dust" concentrations by a factor in the range of 1.5 to 3.0. The conversion factors between the two fractions' concentrations were dependent on the size distribution of the dust sampled, and it would therefore be variable and would not be well described by a simple linear regression. Nevertheless, in seeking to control the amount of airborne material to a particular level, the numerical value of an inhalable dust OEL would be higher than the corresponding "total dust" OEL.

Today, six years after the international adoption of the sampling convention for inhalable dust, no widely accepted method for converting "total dust" OELs into inhalable dust OELs has emerged, and the issue therefore remains important but unresolved.

Some examples of health effects depending on type of dust and airborne particle fraction were published by WHO (6), as shown in the following table:

Type of dust	**Main health effect**	**Target organ**	**Fraction of interest**
Free crystalline silica	Silicosis (lung fibrosis); Progressive and irreversible Restrictive lung disease Also carcinogenic	Lungs, gas-exchange region, alveoli	Respirable fraction
Coal dust	Coal workers' pneumoconiosis Restrictive lung disease	Lungs, gas-exchange region, alveoli	Respirable fraction
Asbestos	Asbestosis; lung cancer: Mesothelioma	Lungs; bronchial and gas-exchange region	Thoracic and respirable fractions
Lead dust	Systemic intoxication (Blood and central nervous system)	Through respiratory system into the bloodstream	Inhalable fraction
Manganese	Systemic intoxication (Blood and central nervous system)	Through respiratory system into the bloodstream	Inhalable fraction
Wood dusts	Certain hard woods cause nasal cancer	Nasal airways	Inhalable fraction
Cotton dust	Byssinosis;	Lungs	Thoracic

	obstructive lung disease		fraction
Dried sugar cane dust	Bagassosis (extrinsic allergic alveolitis)	Lungs	Respirable fraction
Cement dust	Dermatoses	Skin	Any particle size
Pentachlorophenol	Systemic poisoning	Through skin into bloodstream	Any particle size

As can be seen, the appropriate selected size fraction should be most closely associated with the health effect(s) of concern for each substance.

Potential health effects from exposure to wood dust have been summarised and include pulmonary function changes, allergic respiratory responses (asthma) and cancer of the nasal cavity and paranasal sinuses (7-10). A sampling method that accurately measures the amount of **inhalable** wood dust, including particulate matter deposited in the nose, is therefore desirable for the evaluation of worker exposure to airborne wood dust.

Traditionally in the United States and in some European countries, wood dust sampling has been performed by means of the total dust sampling method, which uses a 37 mm diameter filter in a closed-face cassette.

Some measurements using personal inhalable particulate mass (IPM) sampling in woodworking environments have been reported (10-12), usually referred to as the IOM sampler; and a study involving side-by-side comparison sampling studies of "total dust"/inhalable dust for the measurement evaluation of airborne wood dust has been published (13). Results from this study indicate that the inhalable dust/"total dust" ratio for wood dust is generally in the range of 2 to 4 at relatively high wood dust concentrations (≥ 0.5 mg/m^3). However, when "total dust" concentrations were below 0.5 mg/m^3, the corresponding inhalable ratio for personal samplers was erratic (range of 2.1 to 71). Overall, the IPM sampling method can be expected to collect more particulate mass than the total dust method.

In unpublished sampling data from industrial wood dust monitoring presented at a congress (14, 15), inhalable dust/"total dust" ratios of 1.9 to 2.8 and 0.2 to 11.3 were found(14, 15). One of these studies reported inhalable dust/"total dust" ratios of 3.4 to 10.8 when total dust concentrations were less than 0.5 mg/m^3 (15).

In summary, based on the wood dust health-related disease information and on the results obtained in the field studies to compare total dust/inhalable dust sampling methods (filter cassette and IOM sampler) for the evaluation of airborne wood dust, two principal points can be made:

1. The inhalable fraction is the best convention to explain the critical effect(s) of wood dust in the upper airways and it would therefore be the most appropriate fraction to sample.

2nd. Taking into account the difference in sampling efficiency for large particles between the IOM and the filter cassette samplers, it can be concluded that the numerical value of the OEL for

"inhalable dust" may be set at approximately twice the numerical value of what would have been the corresponding limit value for "total dust".

Assuming these two points, the threshold concentrations (OELs) for wood dust considered in the document SCOEL/SUM/102 B are expressed as the total dust fraction. Applying a ratio of inhalable dust/total dust of 2 or 3 to 1, the new threshold concentrations (OELs) expressed as inhalable dust would be as follows :

Threshold concentration (mg / m^3) in Doc. SCOEL/SUM/102 B (total dust)	Range of the new corrected Concentration Equivalent, expressed as the inhalable fraction (mg/m^3)
0.5	1.0 to 1.5 Inhalable fraction
1.0	2.0 to 3.0 Inhalable fraction
5.0	10.0 to 15.0 Inhalable fraction

A new sampling method may therefore imply a new OEL and any change in sampling/analytical methods raises a host of questions concerning the relationship between sampling for "total dust" and for inhalable dust.

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