

WOOD DUST SYMPOSIUM

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PROCEEDINGS

Wood dust symposium

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Introduction

On April 15, 2004, at the Eigtveds Pakhus in central Copenhagen, over 50 researchers and occupational health practitioners from three continents gathered, to participate in the Wood Dust Symposium. The symposium was jointly arranged by the National Institute of Occupational Health (NIOH), Denmark, and the collaborators in the European Research Project “Wood Risk”, funded by the European Commission. It is the intent of NIOH that this should be the first symposium in a series of international symposia arranged by the Institute. The symposium has the subtitle: First Danish NIOH Symposium on Occupational Health Risks: Wood Dust. I feel that the symposium was very timely considering the current discussion within the EU administration and the EU countries about suggestions of a lowered occupational threshold limit value for exposure to wood dust, considerations of wood species, and implications for industry and work places.

The symposium was divided into five separate parts. Paul A. Demers, Vancouver, Canada, gave a general introduction to health effects, epidemiology and exposure measurements in his keynote speech.

The first session “Biology and epidemiology of wood dust related sinonasal cancer” was moderated by Kirsti Husgafvel-Pursianen, Helsinki, Finland. Further presentations:

- Michael Dictor, Lund, Sweden, the pathology and etiology of sinonasal cancer
- Eero Pukkala, Helsinki, Finland, epidemiology of sinonasal cancer in Nordic countries.
- Johnni Hansen, Copenhagen, Denmark, epidemiology in the Danish Wood Risk study
- Reetta Holmila, Helsinki, Finland, P53 mutations in sinonasal cancers
- Håkan Wallin, Copenhagen, Denmark, spoke about K-RAS and H-RAS mutations in sinonasal cancers.

The second session “Epidemiology and biology of wood dust-related non-malignant respiratory diseases” was moderated by Fritz Krombach, Munich, Germany. Further presentations:

- Vivi Schlünssen, Århus, Denmark, what do we know about wood dust exposure and non-malignant respiratory diseases - an epidemiological approach
- Paul Borm, Maastricht, the Netherlands, oxidative properties of particles and biological effects. Fritz Krombach, Munich, Germany, ROS-mediated cytokine expression in wood dust-exposed rat alveolar macrophages
- Juha Määttä, Helsinki, Finland, wood dust-induced inflammatory response in mouse macrophages; Harri Alenius, Helsinki, Finland, wood dust-induced pulmonary inflammation in mice.

The third session titled “Exposures and exposure assessment for wood dust” was moderated by Timo Kauppinen, Helsinki, Finland. Further presentations:

- Raymond Vincent, Vandoeuvre, France; company survey on wood dust in four EU countries.
- Tuula Liukkonen, Lappeenranta, Finland exposure measurements on wood dust;
- Timo Kauppinen, Helsinki, Finland, Occupational exposure to wood dust in EU countries (WOODEX).

The last session “Legislation, prevention and risk assessment of wood dust exposures” was moderated by Raymond Vincent, Vandoeuvre, France. Further presentations:

- Kirsti Husgafvel-Pursiainen, Helsinki, Finland; the SCOEL document for TLV for wood dust in the EU,
- Bent Horn Andersen, Copenhagen, Denmark, Danish prevention and legislation strategy,
- Ulrik Spannow, Copenhagen, Denmark, wood dust regulation and prevention in a trade union perspective
- Kim Bøhmert, Copenhagen, Denmark, Wood dust - more than an occupational health problem

The initiative to hold the meeting was taken in April 2003 by participants in the EU commission-funded research project “Wood Risk”. The objectives of the Wood Risk project are to provide accurate, up-to-date data on occupational exposure to wood dust in Europe, to assess associations between such exposure and molecular alterations of sinonasal cancers, and to study in detail biological mechanisms of pulmonary inflammation related to exposure to wood dust. These data will be utilized for assessing health risks of exposure to wood dust. Molecular alterations of sino-nasal cancer, which are known to show a strong association with exposure to wood dust as well as pulmonary inflammation, the latter infrequently found in individuals exposed to wood dust, have been chosen as the main toxicological endpoints of this research. Data are lacking on carcinogenic mechanisms of wood dust-related sino-nasal cancer, and it might be possible that pulmonary inflammation, in addition to genotoxic mechanisms, at some level contributes to the carcinogenic process. Thus, the two endpoints, chosen for the present study, are highly relevant and may even be interrelated.

The main results of the project will be merged into a risk assessment procedure evaluating the new mechanistic data to be generated against the existing literature data. This aims at improving the knowledge for setting occupational exposure levels and increasing safety of workplaces.

To achieve the general objective of the Wood Risk project, the following specific objectives were set:

- (i) To gather information on exposure to wood dust at the European level by utilizing an existing exposure database for carcinogenic compounds, CAREX, and to supplement wood dust exposure information with this database from other exposure registers in the European Union Member States. These data will constitute WOODEX, a new European Union-wide exposure register on wood dust. This database will frequently be updated to allow current exposure assessment in the European Union Member States also in the future.
- (ii) Measurements of actual exposure to wood dust will be carried out in selected occupational settings with quantified exposure either to soft or hard wood dust to supplement register-based exposure data. Species processed will also be documented.
- (iii) To establish and fully characterize a unique European databank on sino-nasal cancer paraffin-embedded tissue samples with relevant information on occupational and exposure history in relation to wood dust.

- (iv) To investigate molecular alterations in p53, K-ras and H-ras genes in sinonasal cancers with and without wood dust exposure selected according to cell type and collected in three member states. In view of the present data, it can be expected that the types of wood dust exposures vary according to the study area, thus providing a setting where contributions from different types of wood/tree species may be evaluated. In particular, associations to softwood and hardwood dust will be investigated.
- (v) To comprehensively characterize wood dust-induced pulmonary inflammation in rats by exposing rats to graded doses of inhalable hard and soft wood dust, by studying morphological pulmonary tissue changes and exploring changes in molecular markers of inflammation on protein and mRNA levels *in vivo*. Production of cytokines, nitric oxide (NO), and reactive oxygen metabolites, activation of transcription factors NF- κ B and AP-1, and other markers of inflammation will be studied to characterize factors important to *in vivo* models.
- (vi) Alveolar epithelial and phagocytic (macrophage) cell lines will be exposed to graded doses of inhalable wood dust to further delineate biochemical and molecular markers of wood dust-induced inflammation, including cytokine and NO production, production of reactive oxygen species, and activation of transcription factors NF- κ B and AP-1. Furthermore, induction of wood dust-induced apoptosis will be studied at cell membrane levels and by exploring the activation of caspases and genotoxicity by the COMET assay. Wood dust-induced specific alterations of gene expression, potentially relevant for human disease, will be explored utilizing molecular biological techniques. These studies also aim at developing *in vitro* models that could supplement *in vivo* animal models in assessing wood dust-induced pulmonary changes.
- (vii) Nasal lavage will be conducted, and lavage fluid and lavaged nasal epithelial and phagocytic cells will be collected from workers exposed to high concentrations of wood at workplaces. The lavages will be conducted right before a vacation, just before the end of the vacation, and two months after the vacation. The aim of these studies is to explore the lavage fluids for markers of inflammation (cytokines, NO), and the lavaged cells for production of inflammatory markers, and for expression of specific genes activated upon exposure to wood dust. The goal is to be able to demonstrate causality between wood dust exposure and increased expression of marker genes associated with harmful health effects.
- (viii) Based on exposure data (WOODEX), mutation analysis of sinonasal cancer cases, *in vitro* studies on wood dust-induced DNA adduct and mutation formation, and characterization of wood dust-induced inflammation in experimental animals, continuous cells lines, and humans, in the WOOD-RISK database will be created.
- (ix) The ultimate goal is to utilize the results obtained through the databases by assessing the levels of acceptable occupational exposure levels for wood dusts to improve safety of work in wood working operations..

The wood risk project will be finalized by the end of 2004.

I take the opportunity to thank the organizing committee (Jette Bornholdt, Håkan Wallin, Ingrid B. Lauritsen and Johnni Hansen, Copenhagen, Denmark) and the scientific committee (Håkan Wallin, Jette Bornholdt, Johnni Hansen, Copenhagen, Denmark; Paulo Boffetta, Lyon, France; Paul Borm, Maastricht, the Netherlands; Irma Welling, Kai Savolainen, Kirsti Husgafvel-Pursiainen, Timo Kauppinen, Helsinki, Finland; Fritz Krombach, Munich, Germany; Danièle Luce, Paris, France; Vivi Schlüsen, Århus, Denmark; Inger Schaumburg, Skive, Denmark; and Raymond Vincent, Vandoeuvre, France.) Especially, I would like to thank Jette Bornholdt for her dedication and instrumental role in arranging this meeting.

Håkan Wallin, National Institute of Occupational Health, Copenhagen, Denmark

Wood Dust: Overview & Areas of Uncertainty

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Wood dust is made up of cellulose (β -D-glucose, 40-50%), polyoses (mannose, galactose, xylose, 15-35%), lignin (guaiacyl, syringyl, 20-35%), and “extractives” (low-relative-molecular-mass organic and inorganic compounds, <1-15%). The “extractives” are highly variable between species and include many biologically active agents such as alkaloids (toxins, intoxicants), catechols (strong sensitizers, irritants), flavonoids (cardiac effects), lignans (sensitizers), phenols (irritants, sensitizers), quinones (irritants, sensitizers), stilbenes (sensitizers, irritants, carcinogens?), tannins (carcinogens?), and terpenes (sensitizers, irritants). The variability in health effects observed between species is likely due to these naturally occurring chemicals.

The health effects of wood dust include upper respiratory and eye irritation, asthma, dermatitis, chronic lung disease, and cancer. Wood dust has been most strongly associated with sino-nasal cancer, although other cancer sites such as the naso-pharynx are suspected. The highest risks have been observed among furniture workers which may be due to either high levels of exposure or the tree species used (i.e. oak, beech, teak, mahogany, walnut, mahogany, and birch) or both. Many cross-sectional surveys have observed an elevated prevalence of respiratory symptoms and decreased lung function associated with both conifers (softwood) and deciduous (hardwood) tree species. Occupational asthma and exposure to certain tree species, such as Western red cedar and many exotic tree species (e.g. iroko and obeche), has been well established. However, there is evidence that asthma may also be caused by other more, common tree species such as pine, although the prevalence among exposed workers may be lower. A number of tree species have been associated with allergic and/or irritant dermatitis. Upper respiratory and eye irritation is perhaps the most common health effect of wood dust exposure. Many of the health effects of wood dust are observed in the upper respiratory system and it is most appropriate to measure levels of exposure to the inhalable fraction of wood dust when assessing exposure.

There are several areas of uncertainty remaining. An over-riding issue is that the health effects of wood dust vary by tree species, but it is not always clear which are the most toxic or how to group similar species for risk assessment purposes. It is clear that dose-response relationships exists between exposure to wood dust and the risk of both chronic respiratory disease and sino-nasal cancer, but not yet clear what a safe level of exposure is. Further research regarding the mechanisms by which wood dust causes health effects and more studies using quantitative exposure assessment will help address these issues.

Nasal cancer in the nordic countries

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Sino-nasal cancer is very rare. The age-adjusted incidence among Nordic males varies from $4/10^6$ in Sweden and Finland to $8/10^6$ in Denmark.¹ In women the respective variation is from $2/10^6$ to $5/10^6$. There is no variation in nose cancer mortality between countries. The relative 5-year survival rate among Finnish patients diagnosed with nasal cancer in 1999-2001 is 42%, below the average of all cancer patients.

Both incidence and mortality rates of nasal cancer are decreasing among Nordic males but remain constant among women.¹ Sino-nasal cancer among males is a disease of lower socio-economic strata: the incidence among unskilled labourers is two-fold as compared with higher white-collar employees.² These phenomena roughly fit with smoking patterns but not as strongly as, *e.g.*, in lung cancer.

There are some sino-nasal cancer cases diagnosed before the age of 40, but the age-incidence curve starts to raise exponentially after that, reaching its maximal value of about $50/10^6$ in males and $30/10^6$ in females in the ages above 75 years.¹

In a joint Nordic study cancer risks for numerous cancer types were calculated for 53 occupational categories based on census occupation.³ In this huge data set, the only significantly elevated incidence rates were observed for shoe and leather workers (incidence 2.9-fold as compared with average population, 95% confidence interval 1.5–5.3) and for wood workers (1.9, 1.6–2.2), both occupations with confirmed risk factors of nasal cancer.

The Finnish system linking occupation-specific cancer risks with occupational exposures through an exceptionally precise matrix (FINJEM) and sophisticated calculation procedure⁴ produced a dose-response pattern of 1.0 for unexposed, 1.4 (95% CI 0.9–3.2) for estimated exposure of $< 5 \text{ mg/m}^3$ -years, and 2.0 (1.1–3.7) for exposure of $\bullet 5 \text{ mg/m}^3$ -years. In an update of this analysis we will separate the adenocarcinoma and squamous cell carcinoma as separate categories and also include co-factors such as smoking.

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P53 mutations in sino-nasal cancers

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The p53 does not function correctly in most cancers. In about half of human malignancies the p53 gene is mutated, which makes the p53 mutation the most common genetic alteration in human cancers. Many types of environmental cancer, lung cancer in particular, show mutations associated with causative exposures. In a framework of an on-going project we are currently studying p53 mutations in sino-nasal cancers (SNCs) from cases with and without occupational exposure to wood dust. For SNC cases from Denmark, Finland and France paraffin-embedded tissue (PET) samples were collected for DNA extraction and analysis in collaboration with national cancer registries. The study population includes all incident cases of cancer of the nose and paranasal sinuses (ICD7: 160) in Denmark for the years 1992-2002 and in Finland for 1989-2002. In France, due to a different study design and a different type of fixation (Bouin) often used in the pathology laboratories, the number of PET samples so far collected is small. Occupational histories for the cases will be obtained by interviews. The preliminary results show a relatively high frequency of p53 mutations in the tumours from Denmark and Finland. In a pilot study, we also used immunohistochemistry for p53 protein expression in tumours from a subset of Finnish and French cases. Based on our preliminary results, there is a good correlation between gene mutations and p53 stabilisation in SNC. Future work will include completion of the mutation analyses, review of tumour histologies by a pathology panel, assessment of occupational history data by industrial hygienists, and statistical data analysis.

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What do we know about wood dust exposure and non-malignant diseases - an epidemiological approach

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Roughly tree million workers in the European Union are regularly exposed to wood dust at work. Well documented and possible non-malignant health effects caused by wood dust exposure include asthma, chronic bronchitis, impairment of lung function, rhino-conjunctivitis, skin problems, allergic alveolitis, and pulmonary fibrosis. Asthma and rhinitis caused by Western red cedar exposure is well characterised. Between 1 and 13 % of western red cedar workers develops asthma, and exposure-response relations has been revealed. The main aetiological agent, plicatic acid, has been identified, and probably non-IgE Immunological mechanisms are most important. Other types of wood dust can cause asthma, rhino-conjunctivitis, chronic bronchitis, skin affection and acute decline in lung function, Unfortunately, our present knowledge is mainly based on case reports, questionnaire data, and lung function from cross sectional studies. Data on mechanisms is scanty, and practical no data on the incidence of non-malignant diseases caused by wood dust is available. Though, recently some evidence for exposure-response relations between wood dust exposure below 1 mg/m^3 and respiratory health effects as asthma, rhinitis, and decline in lung function has appeared.

Wood dust exposure may cause chronic obstructive lung disease, allergic alveolitis and pulmonary fibrosis, but the documentation is very limited.

In order effectively to prevent diseases caused by wood dust exposure, we have to expand our knowledge. Future research should focus on:

- Exploring the temporal relation between wood dust exposure and diseases with latency time (e.g. asthma, chronic obstructive lung diseases)
- Revealing dose-response relations between wood dust exposure and health effects
- The impact of concurrent exposures, e.g. biohazards, terpenes
- The impact of different wood species
- The disease mechanisms
- Documentation of the health impact of preventive initiatives

Ros-mediated cytokine expression in pine-dust exposed rat alveolar macrophages*

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Respiratory symptoms, reduced lung function, and asthma have been reported in workers exposed to wood dust in a number of epidemiological studies. The underlying pathomechanisms, however, are not well understood. Pine is one of the most widely used wood species in the wood processing industry. In this study, the effects of dust from untreated pine (PD) and heat-treated pine (HPD) on the release of inflammatory mediators were analyzed in rat alveolar macrophages. After 4 h incubation, both PD and HPD induced a significantly ($p < 0.05$) increased tumor necrosis factor- α (TNF- α) and macrophage inflammatory protein-2 (MIP-2) mRNA expression as well as a concentration-dependent (5 - 200 $\mu\text{g/ml}$) TNF- α and MIP-2 protein production. Interestingly, PD induced a significantly higher TNF- α and MIP-2 production than HPD. Polystyrene microspheres used as negative particulate control did not elicit a cytokine response. Moreover, a significantly increased reactive oxygen species (ROS) production, measured by dichlorofluorescein fluorescence, was observed in alveolar macrophages exposed to PD and HPD. In the presence of the antioxidants glutathione and N-acetyl-L-cysteine, the PD- and HPD-induced production of ROS, TNF- α , and MIP-2 was significantly reduced. Finally, electron spin resonance analyses demonstrated a significantly higher endogenous antioxidative capacity of HPD compared to PD. These results indicate that pine dust is able to induce an inflammatory response in rat alveolar macrophages and that ROS mediate the pine dust-induced TNF- α - and MIP-2 expression. Moreover, heat treatment of pine may improve the antioxidant ability of pine dust and thus reduce pine dust-induced inflammatory responses.

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Wood dust-induced inflammatory response in mouse macrophages

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In addition to nasal and sino-nasal adenocarcinomas, wood dust exposure can induce several nonmalignant, mainly respiratory diseases such as allergic rhinitis, chronic bronchitis, and asthma. To find out whether wood dust is able to influence the development of inflammatory process through macrophages, we have elucidated the effects of wood dust exposure on the cytokine and chemokine expression of mouse macrophage cell line cells (RAW 264.7). The cells were exposed to graded doses of selected hardwood (birch, beech, oak, and teak) and softwood dusts (pine and spruce). TiO₂ and LPS were used as controls. The mRNA expression of major proinflammatory cytokines (IL-1 β , TNF- α , and IL-6), an anti-inflammatory cytokine (IL-10), and several chemokines (CCL2, CCL3, CCL4, CCL5, and CCL24) were assessed by real time PCR at several time points after wood dust exposure. TNF- α , IL-6, and CCL2 expression was studied also at the protein level using the ELISA method. Wood dust had in general more effects on cyto- and chemokine expression than inorganic dust TiO₂. All wood dusts induced TNF- α , IL-6, CCL3, and CCL4 expression and inhibited IL-1 β and CCL24 expression. However, many differences were detected in the strength of induction or inhibition between different wood dusts. In the case of CCL2, birch, beech, pine, and spruce induced CCL2 production, but oak and teak dusts had no effect. Oak dust, that has been previously shown to be carcinogenic, appears to be a weaker inducer of inflammatory response than the other wood dusts. Our results show that exposure to different wood dusts elicits dose-dependent changes in the levels of inflammatory mediators in mouse macrophage cells. These findings suggest that exposure to wood dust may significantly influence development of inflammatory process in the airways by modulating the expression of proinflammatory cytokines and chemokines.

Measurement of wood dust exposure

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The Finnish Institute of Occupational Health is coordinating the EU project 'Risk Assessment of Wood Dust: Assessment of Exposure, Health Effects and Biological Mechanisms'. The exposure assessment part of the project aims at assessing current exposure (prevalence and level) to wood dust by industry in the member states of EU. For this purpose the following measurement data were received from six EU countries:

Country	Source of data	Total number of measurements	Number of measurements used in exposure estimation *
Denmark	Danish National Institute of Occupational Health and Dr Vivi Schlünssen	2 970	1 900
Finland	Finnish Institute of Occupational Health (FIOH)	2 300	590
France	COLCHIC data base, Institut National de Recherche et de Sécurité (INRS) and CRAM	7 880	5 000
Germany	Holz-Berufsgenossenschaft (Holz-BG) Munich	20 870	8 930
the Netherlands	studies of Wageningen Agricultural University and Utrecht University	530	360
the UK	Health and Safety (HSE)	2 260	400

* measurements older than 10 years, background measurements, measurements with sampling time less than 2 h were excluded

All concentrations were presented as inhalable dust; conversion factors (between 1 - 2) were therefore used to convert "total" dust concentrations to inhalable dust.

The level of wood dust concentrations has decreased in many countries from the late 1980s to the beginning of the 2000s. According to the latest measurement results (10 past years) the mean (GM) wood dust concentrations varied between 1.0 - 1.5 mg/m³ in sawmilling and planing, and the manufacture of wooden boards. The average concentrations were between 0.5 - 3.5 mg/m³ in the manufacture of wooden products, and between 1.0 - 3.0 mg/m³ in furniture manufacturing.

The average wood dust concentrations were below the occupational exposure limit (OEL) of the EU, i.e. 5 mg/m^3 , but in Finland about 5 - 10%, and in the UK 15 - 30% of the concentrations exceeded this value. Typically about 30% of the measured concentrations exceeded the value of 2 mg/m^3 , which is the OEL e.g. in Germany.

However, it seems that it is possible to reduce wood dust levels with efficient dust control measures, e.g. only 0.4% of the Danish measurement results in the furniture industry exceeded the limit value of 5 mg/m^3 . When the furniture factories with good exhaust ventilation were selected from the German data, 99% of the concentrations were below 5 mg/m^3 and 89% below 2 mg/m^3 , according to the calculations by Holz-BG.

Occupational exposure to wood dust in the European Union: Preliminary results

Timo Kauppinen, Finnish Institute of Occupational Health, Helsinki, Finland

Objectives: The aim of this study was to estimate current exposure to wood dust in the member states of European Union (EU) for the hazard control, exposure surveillance and risk assessment purposes as a part of the EU supported project 'Risk assessment of wood dust: Assessment of exposure, health effects and biological mechanisms (WOOD-RISK)'.

Material and methods: The assessment procedure was designed to provide the numbers of exposed workers by country (15 'old' member states of EU, crude estimates for 10 'new' member states), industry (6 wood industries, 4 other sectors), major species of wood (pine, spruce, oak, beech, wooden boards etc), and level of exposure (5 classes). Company survey on the use of species of wood and on the distribution of workers into similar exposure groups was carried out in Finland, France, Germany, and Spain. The country questionnaires were filled in by 15 national experts providing data on labor force, use of different species of wood, and some other factors needed in exposure assessment. Detailed exposure data were collected from industrial hygiene measurement databases and some large surveys in Denmark, Finland, France, Germany and the United Kingdom. The calculation algorithms of the constructed WOODDEX database were used to generate preliminary estimates of exposure. These estimates will be reviewed, modified, and finalized by national experts. The basic data and final estimates will be included in WOODDEX database, whose data will be freely available through the Internet by the end of 2004.

Results: It was estimated that there were about 3 million workers occupationally exposed to inhalable wood dust (2% of the employed population) in 15 EU countries in 2000-2002. Construction employed about 1 million exposed workers, most of which were carpenters. The numbers of exposed workers were 600,000 in furniture industry, 300,000 in builders' carpentry industry, 200,000 in sawmilling, 100,000 in wooden board industry, and 800,000 in all other industries. The highest exposure levels occurred in furniture and builders' carpentry mills. Variable exposure levels were typical to construction woodworkers. Lower levels occurred in other wood-industries and forestry. According to preliminary estimates, about 200,000 workers (<10% of the exposed) may be exposed to a level exceeding 5 mg/m³, which is the occupational exposure limit set by EU. About 1.5 million workers (50% of the exposed) were exposed to low levels below 0.5 mg/m³ of inhalable wood dust. Mixed exposure to more than one species of wood was very common.

Conclusions: Exposure to wood dust is common, but the exposure levels are usually below the exposure limit of EU. High exposures requiring efficient dust control occur mainly in mills producing wooden furniture and builders' carpentry, as well as in some construction woodworks.

Wood dust-related health effects and occupational limit values

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Exposure to dusts from both hardwood (deciduous) and softwood (coniferous) tree species is associated with a large variety of health effects. The non-carcinogenic ones include irritation and other symptoms in the nose and the eyes, irritation and inflammation of the respiratory epithelium, coughing, wheezing, chronic bronchitis, and asthma. In addition, one of the main concerns is the observation that occupational exposure to wood dust – especially to hardwood dust – is related to considerably elevated risk of cancer. Numerous epidemiological studies have consistently demonstrated a strong causal association between exposure to hardwood dust and sino-nasal adenocarcinoma (ICD10: C31), a rare type of human cancer. The overall evaluation of the working group of the International Agency for Research on Cancer (1995) notifies that wood dust is carcinogenic to humans (Group 1). The evaluation is based on the marked increase in the occurrence of cancers of the nasal cavities and paranasal sinuses among workers exposed predominantly to hardwood dusts. Accordingly, Council Directive 1999/38/EC, enforced in April 1999, states that hardwood dusts, exposure to oak and beech in particular, are to be considered human carcinogens. The Directive gives a limit value of 5 mg/m³ (inhalable fraction) for occupational exposure to hardwood dust. However, it seems evident that to protect woodworkers from effects on respiratory health, a lower limit value is needed. It appears very well documented in the scientific literature that exposure to wood dust at levels below 5 mg/m³, including 1 mg/m³ and lower, is causing sino-nasal as well as pulmonary symptoms. In fact, symptoms in the upper and lower respiratory tracts have been observed in some studies at exposure levels as low as 0.5 mg/m³ and lower. The Scientific Committee of Occupational Limits (SCOEL), giving expert advice to the European Commission, is currently preparing documentation on the existing scientific evidence on wood dust for establishment of occupational exposure limits in the EU.

Financial support from EU, 5th FP (contract no. QLK4-2000-00573), and the Academy of Finland (project no. 53631) is acknowledged.

Wood dust regulation and prevention – a trade union perspective

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The scientific evaluation on wood dust indicates, that wood dust exposure provides risks for cancer, allergic and non-allergic dermal and airway responses, irritation etc.

According to the Carcinogen Directive (1999/38 amending 90/394 Directive) workers must be effectively protected from the risks of developing cancer as a result of occupational exposure to hardwood dusts.

Currently, the limit value is of great interest. The present European wood dust limit value (5 mg/m^3) is a technical value not based on scientific evidence. A Dutch recommendation ($0,2 \text{ mg/m}^3$) shows a kind of long perspective. And SCOEL seems to point at $0,5 \text{ mg/m}^3$. Now we are waiting for the Commission's proposal.

For the employers the established limit value of 5 mg/m^3 seems to be adequate: The employers of CEI-Bois have asked their national affiliates to request the national authorities to instruct SCOEL to withdraw its proposal for reducing the demand on wood dust in the working atmosphere to $0,5 \text{ mg/m}^3$ and to keep the demand on 5 mg/m^3 , according to Nytt fra RAMIT, 1-2003.

In a trade union perspective any doubts of health effects should lead to regulation in favour of the workers (principle of precaution). For this reason, the limit value of wood dust shall be $0,5 \text{ mg/m}^3$. This is reasonable according to scientific evidence on health effects, the technical measures of prevention and the actual levels of exposure.

Prevention starts by conducting a risk assessment focusing on any risk of wood dust in the work place; including the hazardous properties; the level, type and duration of exposure; the effect of preventive measures taken or to be taken etc. This is European law.

In fact, it is possible to reduce the exposure to all most zero.

Swedish example, late 1990es:

Goal for not exceeding $0,3 \text{ mg/m}^3$ was reached. The important factors for success were the commitment from personnel, support from management and the trade union and competent technical advice.

Lesson learned from the Swedish case: Reducing wood dust provides added value, better work environment, reducing cleaning time, better quality and more effective and profitable production.

Summing up: Worker participation is the determinant of successful occupational health and safety management and a major contributing factor in the reduction of occupational diseases and injuries - Also with respect to wood dust.

