

The Briggs group  
100 years in timber  
1908 to 2008

G.L. BRIGGS  
SALES PTY LTD

409 VICTORIA STREET, WETHERILL PARK NSW 2164.  
P.O. BOX 6613, WETHERILL PARK B.C. 1851 NSW AUSTRALIA  
PHONE: (02) 9732 7888 FAX: (02) 9732 7800  
admin@briggs.com.au  
ABN 72 000 098 055

From the FOREST to YOUR FLOOR

### WOOD DUST IS DANGEROUS

AT BRIGGS WE HAVE PREFINISHED MOST OF OUR FLOORING TO  
ELIMINATE FINE WOOD DUST EXPOSURE  
COARSE WOOD DUST FROM SAWING IS UNLIKELY TO BE A PROBLEM

There are many studies and technical reports detailing the dangers of wood dust. Australia has extensive legislation limiting exposure of workers to wood dust. There seems to be no protection for the home.

Wood dust becomes MUCH MORE DANGEROUS when it is below 10 microns in size. This is because the lungs can fairly easily expel particles over about 10 micron. There is still danger for nasal passages and possibly eyes from larger particles.

**Why is wood dust now a problem, after all our ancestors have lived with wood dust and they did not die of it?**

In the last 25 years [or so] the power of floor sanding machines has dramatically increased and there is now a demand for smoother and finer finishes. Gone are the days of hand waxing and floor polishing every few weeks or months. This means floors are sanded with finer sandpaper travelling faster and with much more energy. This results in very small particle sizes: Considerably smaller than the old style of floor finishing.

The normal vacuum cleaner does not remove very well the very fine particles and fine particles cling to surfaces, even the ceilings. When a floor is coated the dust on the floor is 'painted in' and is not a risk. The dust clinging to the walls and ceilings will continue to contaminate the air in a room for months and maybe years. It is not adequate to brush it off as a lot just re-clings and vacuum cleaning spreads it more. It can be 'painted in' by painting the walls and ceilings after the floor is finished.

Below are a number of articles about wood dust that may assist in a better understanding of the problem and risks.

---

The Briggs group – 100 years in timber – 1908 to 2008 ©

24-Apr-09 Information provided is subject to change without notice. Please check for the most up to date documents from: [www.briggs.com.au](http://www.briggs.com.au)

# CONTROLLING WOOD DUST HAZARDS AT WORK

Timber is generally divided into two categories, softwoods like pine and cedar, and hardwoods like oak, teak and jarrah.

The Western Australian occupational exposure standard (OES) for airborne inhalable wood dust is 1mg/m<sup>3</sup> (one milligram per cubic metre) for hardwoods and 5mg/m<sup>3</sup> for softwoods.

The standard for MDF is also 1mg/m<sup>3</sup> because it can contain hardwood.

The average inhalable wood dust in the breathing zone of the worker must not exceed the OES over an eight hour working shift.

Where dust from timber that has been coated with a toxic substance (such as lead paint) is involved, the OES for both the toxic coating and wood dust must be complied with.

Formaldehyde readings should be below 1ppm (one part per million) when averaged over an eight hour day. Short term exposures should not exceed 2ppm.

## **Who is at risk?**

People exposed to wood dust at work are at greater risk of developing nose, sinus, throat, lung, and skin conditions.

Health problems may take a long time to develop, and are most common in people who have spent many years at workplaces exposed to high concentrations of wood dust.

Dusts from hardwoods are usually finer and therefore more easily inhaled.

Those most at risk are at indoor workplaces with inadequate dust extraction systems.

## **What work is hazardous?**

Hazardous amounts of wood dust may be generated by jobs like floor sanding, furniture sanding, wood turning, routing, sawing, sweeping and emptying dust filters.

Dust hazards exist in varying degrees from timber felling in the forest to furniture manufacturing and cabinet making in factories and workshops.

The main hazards occur where there is poor natural or mechanical ventilation.

## **How can wood dust harm you?**

---

Studies in Australia and elsewhere have linked wood dust in workplaces to asthma, bronchitis, lung, sinus and throat irritations, shortness of breath and skin problems.

The IARC (International Agency for Research into Cancer), after researching nasal cancer among woodworkers in Europe, has classified wood dust a human carcinogen.

Wood dust from timbers such as beech and oak which is fine enough to be inhaled are known to cause cancer. Other species such as, birch, mahogany, teak and walnut may also be capable of causing nasal cancer. As this is a rare form of cancer, the risk is small and generally restricted to the finishing trades where the dust is fine.

Freshly cut trees contain large amounts of microbes, mainly moulds. The number of these increase if the logs are stored outside and under moist or humid conditions. The microbes can cause inflammation of the airways during debarking, sawing and transportation.

### **What about different woods?**

While there has been considerable research carried out on European and North American species, relatively little is known about the way dusts from different Australian timbers affect people's health. Each type of timber has its own chemical components and may affect people differently.

Even less is known about dusts from imported woods, for example from Africa, South America and Asia. There is a concern that adverse health effects from some of these wood dusts may not show up for perhaps many years. Protection for workers today is therefore vitally important.

### **What about chemicals?**

Plywoods, fibre boards, particle boards and laminated products contain formaldehyde that can cause irritation of the respiratory system and eyes. Some suppliers provide Material Safety Data Sheets (MSDS) or similar information on .

Small amounts of formaldehyde may be given off during the cutting or machining of particle board, but this is seldom high enough to cause a problem. Higher levels may accumulate if products are stored in plastic or in unventilated enclosed spaces.

Many wood products have been coated with varnishes, lacquers, polishes and other chemicals. These may cause harm to health under some circumstances, and need to be considered when working with wood.

Dusts from second hand timbers may contain toxic paints, preservatives or lead.

Dust from CCA (copper-chrome-arsenic) treated pine timber is not significantly more toxic than from other timbers, provided dust levels are kept below occupational exposure standards.

---

There are also a number of chemicals used for sealing, coating and polishing timber that may pose a hazard to workers during their application, and the hazards from these substances are described in their respective MSDS.

### **What is the answer?**

The best way of reducing wood dust at a workplace is by using machines that are enclosed or fitted with an effective local exhaust ventilation (LEV).

LEV involves locating an extraction outlet as close as possible to each identified source of dust, and having sufficient air velocity to draw dust away before it becomes airborne.

On some machines with a single cutting, grinding or sanding face, there may be several sources of dust either thrown, drawn or blown from different parts of the machine. Ideally, each dust source should be controlled by the LEV system.

While most wood dust is from machines, hand sanding fine furniture can be one of the dustiest jobs at the workplace. Hand sanding generally requires the use of respiratory protection.

### **What is available?**

LEV systems consist of a hood to capture the dust, duct work to convey the dust and a mechanism such as a filter or cyclone to remove the dust. Energy to move the air and dust through the system is provided by a fan that is driven by an electric motor. Each of these components is important and needs to be matched to the job. Specialised knowledge is needed to design an effective LEV system.

Most modern wood working power tools and machines are fitted with one or more local exhaust ventilation hoods or outlets to capture dust.

Small portable machines such as sanders usually have a filter bag attached. These filters generally do not reduce fine dust exposure sufficiently. Larger machines are connected by a duct to a fixed or portable dust collection unit. The filters on these units are more effective. However in either case respiratory protection may need to be worn if occupational exposure standards are likely to be exceeded.

While most modern wood working machines are designed with dust control in mind, the effectiveness of the systems available can vary considerably. Dust control should be considered when purchasing new machinery.

---

Detailed guidance on the design of ventilation systems for a range of wood working machines is widely available. For example, organisations such as the US National Institute of Occupational Safety and health (NIOSH), the UK Health and Safety Executive (HSE), and the American Conference of Governmental Industrial Hygienists (ACGIH) all publish information on wood working machines. Some of this information is accessible via the Internet.

Maintenance of LEV systems is as important as good design. The performance of LEV systems needs to be monitored and the system maintained in accordance with the manufacturer's specifications.

### **What about older equipment?**

Exhaust ventilation attachments and improvements have also been developed that can be fitted to some earlier machines, for example table saws, band saws, belt sanders and orbital sanders.

Machines and power tools that are difficult to fit with local exhaust ventilation may need to be replaced.

Room exhaust ventilation does not effectively reduce wood dust hazards.

Personal protective equipment, such as dust masks or respirators, should not be used as a substitute for safe exhaust ventilation.

### **What is the Law?**

Section 19 of the Occupational Safety and Health Act 1984 says employers must provide and maintain a work environment in which employees are not exposed to hazards.

Regulations 3.38 and 3.39 of the *Occupational Safety and Health Regulations* require employers, main contractors and self employed persons to:

- identify toxic atmosphere hazards (eg. wood dust);
- assess the risk of injury or harm; and
- consider reducing the risk by:
  - (a) an effective ventilation system;
  - (b) an exhaust system that extracts contaminants; and
  - (c) any other means to prevent a person being exposed.

Section 20 of the *Act* says employees must take reasonable care of their own safety and health and avoid adversely affecting the safety and health of others. An employee must comply "so far as he is reasonably able" with safety instructions, use the protective equipment provided and report hazards and any injury at the workplace.

The law also requires employers and employees to consult in order to resolve safety and health issues in the workplace. If an employee is concerned about his or her

health, they should raise the matter with their employer or safety and health representative if there is one.

### **Identifying hazards**

Hazardous airborne wood dust can be generated by:

- hand sanding;
- machine sanding;
- machining;
- band sawing;
- table sawing;
- routing;
- woodturning;
- copy lathing;
- dry sweeping dusty floors;
- emptying dust filter bags; and
- using compressed air to dislodge dust.

Inspirable (inhalable) dust may not always be visible and, where there is a concern, dust exposure readings need to be taken to determine dust levels.

This can be done by an occupational safety and health service provider.

In addition to being a health hazard, dusty atmospheres or excessive dust accumulating inside equipment or work areas can cause fire or explosion.

### **Assessing risks**

High levels of airborne wood dust at a workplace can place people at risk.

The degree of risk depends on:

- concentrations of airborne dust;
  - size of dust particles;
  - type of wood;
  - additives in the wood;
  - susceptibility of workers;
  - effectiveness of exhaust ventilation; and
  - other safe procedures.
-

## **Control measures**

The preferred order of control measures is:

### **1. Elimination**

This is possible only if wood is not machined, sawn or sanded, or if all work that produces wood dust is outsourced to another workplace with adequate controls.

### **2. Substitution**

Replacing more “hazardous” timbers with less hazardous species may be possible if reliable data on health risks is available. For most species however, there is little data available.

### **3. Isolation**

Enclose plant or keep workers away from dusty areas.

### **4. Engineering**

- Attach efficient local exhaust ventilation to existing plant and hand held power tools.
- Have local exhaust ventilation for hand sanding tasks.

### **5. Administration**

Some examples:

- Use suction cleaners – rather than compressed air – to remove accumulated dust from ledges, corners, pits and floors.
- Empty filter bags outdoors, away from work areas, and where the dust will not blow back into the workplace.
- Rotate dusty tasks to reduce workers’ exposure times.
- Provide education, supervision and training on wood dust hazards for both employers and employees.
- Monitor risks to ensure they remain as low as possible.
- Workers exposed to wood dust should wash or shower before eating or smoking. This is of particular importance in the case of treated wood.

### **6. Protective equipment**

- Protective equipment such as face masks and respirators are a last line of protection. In some circumstances they may be needed to supplement other measures in order to reduce exposure to levels below the occupational exposure standard.
  - Respiratory equipment should meet Australian Standards AS/NZ 1715 and 1716.
  - People with skin sensitive to certain wood dusts should wear suitable protective clothing, eg. coveralls, long sleeves, and properly fitted industrial gloves.
-

## **Further information**

Further information relating to many of the matters referred to above may be found in National Association of Forest Industries publications or obtained from:

Chamber of Commerce and Industry  
180 Hay Street  
EAST PERTH WA 6000  
Tel.: 9365 7555  
Email: [info@cciwa.asn.au](mailto:info@cciwa.asn.au)

UnionsWA  
Level 4  
79 Stirling Street  
PERTH WA 6000  
Tel: 9328 7877  
Email: [unions@tlc.wa.org.au](mailto:unions@tlc.wa.org.au)

WorkSafe Western Australia  
1260 Hay Street  
WEST PERTH WA 6005  
Tel: 9327 8777  
Email: [safety@worksafe.wa.gov.au](mailto:safety@worksafe.wa.gov.au)

---

## Wood dust - Health hazards and control

- **Document Type:** Guidance Note

**Keycode:** web only

**Industry:** [Wood and wood products manufacturing](#)

**Category:** [Hazardous Substances](#)

**Division Author:** Manufacturing & Agriculture

**Publication Date:** 06 June 2005

**Date First Published:** 23 October 2003

**Summary:** This guidance note provides information on reducing the risks associated with exposure to wood dust.

### PURPOSE

This guidance note provides advice on minimising exposure to dust from handling and working with wood.

For the purpose of this guidance note, wood means natural timbers, particleboard (sometimes called chipboard) and fibreboard (also known as Medium Density Fibreboard or MDF).

The Guidance Note does not cover hazards associated with manufacturing particleboard or fibreboard.

### BACKGROUND

The manufacture of wood products such as architrave and skirting mouldings, furniture, doors and windows often results in the generation of fine airborne wood particles and dust. Typical wood-working activities that produce dust are machining operations (e.g. sawing, routing, turning) and sanding (hand or machine).

Other sources of breathable wood dust are the bagging of dust from dust extraction systems, using compressed air to blow dust off articles and dry sweeping of factory floors, etc.

### MDF VS OTHER FORMS OF WOOD

This guidance note makes no distinction between dust generated from wood and fibreboard or particleboard such as MDF. This decision is based on a comprehensive

study conducted in the United Kingdom by the Health and Safety Executive (HSE). It concluded that the ill-health effects associated with dust exposure arising from the machining of MDF are no different from those effects arising from machining other forms of wood (see Further Information for details of this report).

It is also important to note that when working with particleboard and fibreboard, there is a low risk of exposure to formaldehyde. Formaldehyde is used in the production of manufactured wood, such as MDF. When first made the unsealed surface of the boards may release some formaldehyde gas, but this quickly dissipates during initial storage.

Information provided by Australian manufacturers of the boards indicates that the release of formaldehyde gas from unsealed boards supplied to workplaces is well below the accepted exposure limits (see Further Information for a reference to National Occupational Health and Safety Commission (NOHSC) Exposure Standards).

#### WHAT ARE THE HEALTH HAZARDS?

Reported health effects associated with exposure to dust from wood products include: skin disorders such as allergic dermatitis. Certain timbers are known to produce adverse health effects and sensitisation (see Further Information for a reference to a HSE information sheet on toxic woods)

asthma and impairment of lung function

irritation of the nose, rhinitis (runny nose), violent sneezing, blocked nose and nose bleeds

throat irritation, and sore and watering eyes.

A rare type of nasal cancer has also been reported in people who have worked in very dusty wood-working environments with little or no dust control in place. **MANAGING THE RISKS**

#### Controlling the build up of wood dust

The nature of wood-working is such that total elimination of wood dust from the work environment is not usually practicable. However, the health risk associated with exposure to dust from wood products can be minimised through:

using a process or method of work that reduces the generation of dust to a minimum; e.g. using a plane instead of a sander to shape the wood

providing dust capturing equipment to all dust-producing processes; e.g. local exhaust ventilation at wood working machines and dust bags on tools

maintaining plant and equipment in good condition; e.g. inspect local exhaust ventilation systems regularly to ensure they are working efficiently and check for holes and leakages in duct work.

#### Using alternative woods

The supplier of wood and specialty timbers can provide information, e.g. a material safety data sheet, about any potential health effects of the wood being used. Employers should consider using woods that have similar strength or decorative effects but are less hazardous.

### Monitoring dust levels

Even with the use of recommended dust control techniques, it may not be practicable to prevent exposure to wood dust. If there is uncertainty about whether there is a risk to health from exposure to dust from wood products, air monitoring may need to be carried out.

The risk to health needs to be assessed taking into account the nature of the work, duration of exposure and control measures in place. NOHSC occupational exposure standards have been determined for hard woods and soft woods (see also Further Information).

Note: Both the assessment and any subsequent consideration of control options are best carried out in consultation with relevant employees and any health and safety representatives.

### Improving housekeeping to minimise dust

Simple changes to work practices can minimise the level of wood dust in the workplace; e.g.

- prevent accumulation of dust and wood chips by cleaning/emptying dust collection equipment regularly

- use dustless methods for cleaning up such as wet clean up, damping down before sweeping, or using an industrial vacuum cleaner fitted with a HEPA filter. Do not use compressed air to clear work benches or to blow dust off wood products.

- implement a 'clean up as you go' policy.

### Providing respiratory protective equipment

When other dust control measures are not practicable, a respiratory protective device (RPD) suitable for particulates should be worn. Australian / New Zealand Standard AS/NZS 1715 Selection, use and maintenance of respiratory protective devices provides comprehensive guidance on how to select the correct type of RPD. When selecting a RPD, ensure that the equipment meets an appropriate standard. Look for Australian Standard markings (see AS/NZS 1716 Respiratory protective devices) or equivalent on the respirator or its container.

## OTHER SAFETY MEASURES

- Provide information, instruction and training; e.g.

  - obtain health and safety information from the wood supplier or manufacturer and have this readily accessible

  - inform employees on the hazards and risks associated with exposure to wood dust

  - train employees on the correct use of control measures adopted at the workplace

  - supervise employees to ensure that the adopted control measures are being utilised correctly.

- Reduce the chance of dust explosion by keeping ignition sources such as flame and sparks away from locations where dust is being generated.

## LEGAL REQUIREMENTS

All employers have a general duty under the Occupational Health and Safety Act 2004 (OHS Act) to provide and maintain so far as is reasonably practicable a working environment that is safe and without risks to health.

Manufacturers, importers and suppliers of wood have an obligation under the OHS Act to ensure information about their products is available so that they can be used safely and without risks to health. Such information may be provided in the form of a material safety data sheet.

#### FURTHER INFORMATION:

##### Acts & Regulations

Acts and regulations are available from Information Victoria on 1300 366 356 or order online at [www.bookshop.vic.gov.au/](http://www.bookshop.vic.gov.au/)

If you only want to view the legislation you can use the Parliament of Victoria web site; go to [www.dms.dpc.vic.gov.au/](http://www.dms.dpc.vic.gov.au/) , click on "Victorian Law Today" and scroll down to the "Search" window.

##### Australian Standards

AS 3640 -- 1989: Workplace atmospheres -- Method for sampling and gravimetric determination of inspirable dust

Australian Standards are available from Standards Australia on 1300 654 646, or on-line at [www.standards.com.au](http://www.standards.com.au)

##### WorkSafe Victoria

Useful health and safety information is available on WorkSafe Victoria's web site; go to [www.worksafe.vic.gov.au](http://www.worksafe.vic.gov.au) and click on the WorkSafe Victoria logo. Or contact our Advisory Service on 9641 1444 or toll free 1800 136 089.

Copies of publications, including Codes of Practice, can be obtained by contacting WorkSafe Victoria on 03 9641 1333 or your local WorkSafe Victoria office.

##### National Occupational Health and Safety Commission

The National Occupational Health and Safety Commission (NOHSC) web site provides access to technical data and occupational health and safety information; go to [www.ascc.gov.au/OHSLegalObligations/](http://www.ascc.gov.au/OHSLegalObligations/)

If you can't find guidance material that gives the solutions or advice you're after, try the national database of guidance material on the NOHSC web page; go to <http://natindex.ascc.gov.au/>

##### Health and Safety Executive (UK)

Wordworking Sheet No. 30 -- Toxic Woods

This information sheet can be downloaded from the HSE web site. Go to

[www.hse.gov.uk/pubns/woodindx.htm](http://www.hse.gov.uk/pubns/woodindx.htm)

and click on the subject title Toxic Wood. Other useful information relating to woodworking and wood dust can also be obtained at this web address.

Hazard Assessment Document EH75/1 -- Medium density fibreboard

A copy of this document is held at the WorkSafe Victoria Bourke Street Library and can be viewed by contacting the library on 9941 0550 or 9941 0552.

Special Note on Codes of Practice: Codes of Practice made under the Occupational Health and Safety Act 1985 provide practical guidance to people who have duties or obligations under Victoria's OHS laws. The Occupational Health and Safety Act 2004 allows the Minister for Workcover to make Compliance Codes which will provide greater certainty about what constitutes compliance with the OHS laws.

Codes of Practice will continue to be a practical guide for those who have OHS duties and WorkSafe will continue to regard those who comply with the topics covered in the Codes of Practice as complying with OHS laws. WorkSafe will progressively review all Codes of Practice and replace them with guidance material and in appropriate cases, with Compliance Codes.

Note: This guidance material has been prepared using the best information available to WorkSafe Victoria. Any information about legislative obligations or responsibilities included in this material is only applicable to the circumstances described in the material. You should always check the legislation referred to in this material and make your own judgement about what action you may need to take to ensure you have complied with the law. Accordingly, the Victorian WorkCover Authority extends no warranties as to the suitability of the information for your specific circumstances.

## Acts and Regulations

Acts and regulations are available from Information Victoria on 1300 366 356 or order online at [www.bookshop.vic.gov.au](http://www.bookshop.vic.gov.au).

View the legislation at Victorian Law Today: [www.legislation.vic.gov.au](http://www.legislation.vic.gov.au)

## Standards Australia

**Copies of standards can be obtained by contacting Standards Australia on 1300 654 646 or by visiting the web site at [www.standards.com.au](http://www.standards.com.au).**



Call us on 1800 136 089

Email us on [info@worksafe.vic.gov.au](mailto:info@worksafe.vic.gov.au)





WORLD HEALTH ORGANIZATION  
INTERNATIONAL AGENCY FOR RESEARCH ON CANCER

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans

# Volume 62

## Wood Dust and Formaldehyde

Summary of Data Reported and Evaluation

---

[Wood dust](#)

[Formaldehyde](#)

---

Last Updated 08/13/97

# WOOD DUST (Group 1)

For definition of Groups, see [Preamble Evaluation](#).

**VOL.:** 62 (1995) (p. 35)

## 5. Summary of Data Reported and Evaluation

### 5.1 Exposure data

Wood is one of the world's most important renewable resources. At least 1700 million m<sup>3</sup> are harvested for industrial use each year. Wood dust, generated in the processing of wood for a wide range of uses, is a complex substance. Its composition varies considerably according to species of tree. Wood dust is composed mainly of cellulose, polyoses and lignin and a large and variable number of substances of lower relative molecular mass which may significantly affect the properties of the wood. These include non-polar organic extractives (fatty acids, resin acids, waxes, alcohols, terpenes, sterols, steryl esters and glycerols), polar organic extractives (tannins, flavonoids, quinones and lignans) and water-soluble extractives (carbohydrates, alkaloids, proteins and inorganic material).

Trees are characterized botanically as gymnosperms (principally conifers, generally referred to as softwoods) and angiosperms (principally deciduous trees, generally referred to as hardwoods). Roughly two-thirds of the wood used commercially worldwide belongs to the group of softwoods. Hardwoods tend to be somewhat more dense and have a higher content of polar extractives than softwoods.

It is estimated that at least two million people are routinely exposed occupationally to wood dust worldwide. Nonoccupational exposure also occurs. The highest exposures have generally been reported in wood furniture and cabinet manufacture, especially during machine sanding and similar operations (with wood dust levels frequently above 5 mg/m<sup>3</sup>). Exposure levels above 1 mg/m<sup>3</sup> have also been measured in the finishing departments of plywood and particle-board mills, where wood is sawn and sanded, and in the workroom air of sawmills and planer mills near chippers, saws and planers. Exposure to wood dust also occurs among workers in joinery shops, window and door manufacture, wooden boat manufacture, installation and refinishing of wood floors, pattern and model making, pulp and paper manufacture, construction carpentry and logging. Measurements are generally available only since the 1970s, and exposures may have been higher in the past because of less efficient (or non-existent) local exhaust ventilation and other measures to control dust.

The wood species used in wood-related industries vary greatly by region and by type of product. Both hardwoods and softwoods (either domestically grown or imported) are used in furniture manufacture. Logging, sawmills and plywood and particle-board manufacture generally involve use of trees grown locally. Most of the wood dust (by mass) found in work environments has a mean aerodynamic diameter of more than 5 µ. Some investigators have reported that the dust generated in operations such as sanding and during the processing of hardwoods results in a higher proportion of smaller particle sizes, but the evidence is not consistent.

Within the furniture manufacturing industry, exposure may occur to solvents and formaldehyde in glues and surface coatings. Such exposures are usually greatest for workers with low or negligible exposure to wood dust and are infrequent or low for workers with high exposure to wood dust. The manufacture of plywood and particle-board may entail exposure to formaldehyde, solvents, phenol, wood preservatives and engine exhausts. Sawmill workers may also be exposed to wood preservatives and fungal spores. Exposures to chemicals in industries where other wood products are manufactured vary but are in many cases similar to those in the furniture manufacturing industry.

### 5.2 Human carcinogenicity data

The risk for cancer, and particularly cancer of the nasal cavities and paranasal sinuses, among woodworkers has been investigated in many epidemiological studies. Some of the studies provided specific information on cancer risk associated with exposure to wood dust, and those studies were given greatest weight in the evaluation.

Most of the available cohort and case-control studies of cancer of the nasal cavities and paranasal sinuses have shown increased risks associated with exposure to wood dust. These findings are supported by numerous case reports. Very high relative risks for adenocarcinoma at this site, associated with exposure to wood dust, have been observed in many countries, particularly in Europe. The lower risks observed in the studies in the United States may be due to differences in concentration or type of wood dust, but in one of these studies the more heavily exposed groups had significantly increased risks. A pooled analysis of 12 case-control studies revealed a clearly increasing risk with increasing estimated levels of exposure to wood dust, overall and in most individual studies. The excess appears to be attributable to wood dust *per se*, rather than to other exposures in the workplace, since the excess was observed in various countries during different periods and among different occupational groups, and because direct exposures to other chemicals do not produce relative risks of the magnitude associated with exposure to wood dust.

Adenocarcinoma of the nasal cavities and paranasal sinuses is clearly associated with exposure to hardwood dust; in several series of cases of adenocarcinoma from different countries, a high proportion of cases had been exposed to hardwood, and these findings were confirmed in several case-control studies as well. There were too few studies of any type to evaluate cancer risks attributable to exposure to softwood alone. In the few studies in which exposure was primarily to softwood, the risk for cancer of the nasal cavities and paranasal sinuses was elevated but considerably lower than that in studies of exposure to hardwood or to mixed wood types; furthermore, in the studies of exposure to softwood, exposure to hardwood could not clearly be ruled out. It is more difficult to attribute excess risk to any particular species of wood. The concentration of wood dust and the duration of exposure may also contribute to differences in the risks of workers exposed to different types of wood. These studies consistently indicate that occupational exposure to wood dust is causally related to adenocarcinoma of the nasal cavities and paranasal sinuses.

In studies of squamous-cell carcinoma of the nasal cavities and paranasal sinuses, smaller excesses were generally reported than for adenocarcinomas, and a pooled analysis of 12 case-control studies found no association with exposure to wood dust.

A number of case-control studies on nasopharyngeal cancer have reported an association with employment in wood-related occupations; however, confounding was not ruled out from these studies, and the largest study, from Denmark, in which exposure to wood dust was estimated, did not confirm the association. Case-control studies of laryngeal cancer consistently showed an association with exposure to wood dust or woodworking; however, cohort studies of woodworkers gave consistently negative results. Overall, these studies provide suggestive but inconclusive evidence for a causal role of occupational exposure to wood dust in cancers of the nasopharynx.

Studies of the association between exposure to wood dust and cancers of the oropharynx, hypopharynx, lung, lymphatic and haematopoietic systems, stomach, colon or rectum individually gave null or low risk estimates, gave inconsistent results across studies, and did not analyse exposure-response relationships. The evidence for an association between exposure to wood dust and Hodgkin's disease was somewhat more suggestive, in that some case-control studies showed moderately high risks, but these results were not substantiated by the results of cohort studies or some of the well-designed case-control studies. In view of the overall lack of consistent findings, there is no indication that occupational exposure to wood dust has a causal role in cancers of the oropharynx, hypopharynx, lung, lymphatic and haematopoietic systems, stomach, colon or rectum.

### **5.3 Animal carcinogenicity data**

Dust from beech wood was tested for carcinogenicity by inhalation and for enhancement of carcinogenicity when administered with sidestream cigarette smoke or formaldehyde in two studies in rats, or with *N*-nitrosodiethylamine administered by subcutaneous injection in two studies in hamsters. The studies did not show any significant carcinogenic or co-carcinogenic potential of beech wood dust, but each of the studies

suffered from various kinds of limitations and had some inadequacies in reporting of data.

The mutagenic fraction of a methanol extract of beech wood dust was tested for carcinogenicity by skin application in one study in mice. Although a significant, dose-dependent increase in the incidence of skin tumours and a marginally significant, dose-dependent increase in the incidence of mammary tumours were observed, these results cannot be used in an evaluation of the carcinogenicity of wood dust *per se*.

In a preliminary study, beech wood dust was tested for local carcinogenicity by intraperitoneal injection in rats; no peritoneal tumours were reported.

#### **5.4 Other relevant data**

General knowledge of particle size indicates that wood dust can be deposited in human upper and lower airways, the deposition pattern depending partly on particle size. Heavy exposure to wood dust may result in decreased mucociliary clearance and, sometimes, in mucostasis. No data were available on clearance of wood dust from the lower airways.

Exposure to wood dust may cause cellular changes in the nasal epithelium. Increased frequencies of cuboidal metaplasia and dysplasia were found in some studies of workers exposed to dust from both hardwood and softwood. These changes can potentially progress to nasal carcinoma.

Impaired respiratory function and increased prevalences of pulmonary symptoms and asthma occur in workers exposed to wood dust, especially that from western red cedar.

There is little reliable information on the effects of wood dusts on the respiratory tract of rodents. One study *in vitro* showed that various wood dusts are cytotoxic and can induce drug metabolizing enzymes.

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.

#### **5.5 Evaluation**

There is *sufficient evidence* in humans for the carcinogenicity of wood dust.

There is *inadequate evidence* in experimental animals for the carcinogenicity of wood dust.

#### **Overall evaluation**

Wood dust *is carcinogenic to humans (Group 1)*.

For definition of the italicized terms, see [Preamble Evaluation](#)

# FORMALDEHYDE (Group 2A)

For definition of Groups, see [Preamble Evaluation](#).

**Vol.:** 62 (1995) (p. 217)

**CAS No.:** 50-00-0

**Chem. Abstr. Name:** Formaldehyde

## 5. Summary of Data Reported and Evaluation

### 5.1 Exposure data

Formaldehyde is produced worldwide on a large scale by catalytic, vapour phase oxidation of methanol. Annual world production is about 12 million tonnes. It is used mainly in the production of phenolic, urea, melamine and acetal resins, which have wide use in the production of adhesives and binders for the wood, plastics, textiles, leather and related industries. Formaldehyde is also used extensively as an intermediate in the manufacture of industrial chemicals, such as 1,4-butanediol and 4,4'-diphenylmethane diisocyanate (for polyurethanes and particle-board), pentaerythritol (for surface coatings and explosives) and hexamethylene tetramine (for phenol-formaldehyde resins and explosives). Formaldehyde is used as such in aqueous solution (formalin) as a disinfectant and preservative in many applications.

Formaldehyde occurs as a natural product in most living systems and in the environment. Common nonoccupational sources of exposure include vehicle emissions, some building materials, food, tobacco smoke and its use as a disinfectant. Levels of formaldehyde in outdoor air are generally below 0.001 mg/m<sup>3</sup> in remote areas and below 0.02 mg/m<sup>3</sup> in urban settings. The levels of formaldehyde in the indoor air of houses are typically 0.02-0.06 mg/m<sup>3</sup>; average levels of 0.5 mg/m<sup>3</sup> or more have been measured in 'mobile homes' constructed with particle-board or in houses with urea-formaldehyde insulation, but the levels have declined in recent years as a result of changes in building materials.

It is estimated that several million people are exposed occupationally to formaldehyde in industrialized countries alone. The highest continuous exposures (frequently > 1 mg/m<sup>3</sup>) have been measured in particle-board mills, during the varnishing of furniture and wooden floors, in foundries, during the finishing of textiles and in fur processing. Short-term exposures to much higher levels have been reported occasionally. Exposure to more than 1 mg/m<sup>3</sup> also occurs in some facilities where resins, plastics and special papers are produced. The average formaldehyde level measured in plywood mills and in embalming establishments is about 1 mg/m<sup>3</sup>. Lower levels are encountered, for example, during the manufacture of garments, man-made mineral fibres, abrasives and rubber. Periodic occupational exposure occurs e.g. during disinfection in hospitals and in food processing plants, in some agricultural operations and during firefighting. The development of resins that release less formaldehyde and improved ventilation have resulted in decreased exposure levels in many occupational settings, such as particle-board, plywood and textile mills and foundries.

The exposures that may occur concomitantly with formaldehyde in occupational settings vary by industry, facility and period. They include other components of formaldehyde-based glues and varnishes, solvents, wood dust, wood preservatives and textile finishing agents.

### 5.2 Human carcinogenicity data

Excess numbers of nasopharyngeal cancers were associated with occupational exposure to formaldehyde in two of six cohort studies of industrial or professional groups, in three of four case-control studies and in meta-analyses. In one cohort study performed in 10 plants in the United States, the risk increased with category of increasing cumulative exposure. In the cohort studies that found no excess risk, no deaths were observed from nasopharyngeal cancer. In three of the case-control studies, the risk was highest in people in the highest

category of exposure and among people exposed 20-25 years before death. The meta-analyses found a significantly higher risk among people estimated to have had substantial exposure than among those with low/medium or no exposure. The observed associations between exposure to formaldehyde and risk for cancer cannot reasonably be attributed to other occupational agents, including wood dust, or to tobacco smoking. Limitations of the studies include misclassification of exposure and disease and loss to follow-up, but these would tend to diminish the estimated relative risks and dilute exposure-response gradients. Taken together, the epidemiological studies suggest a causal relationship between exposure to formaldehyde and nasopharyngeal cancer, although the conclusion is tempered by the small numbers of observed and expected cases in the cohort studies.

Of the six case-control studies in which the risk for cancer of the nasal cavities and paranasal sinuses in relation to occupational exposure to formaldehyde was evaluated, three provided data on squamous-cell tumours and three on unspecified cell types. Of the three studies of squamous-cell carcinomas, two (from Denmark and the Netherlands) showed a positive association, after adjustment for exposure to wood dust, and one (from France) showed no association. Of the three studies of unspecified cell types, one (from Connecticut, United States) gave weakly positive results and two (also from the United States) reported no excess risk. The two case-control studies that considered squamous-cell tumours and gave positive results involved more exposed cases than the other case-control studies combined. In the studies of occupational cohorts overall, however, fewer cases of cancer of the nasal cavities and paranasal sinuses were observed than were expected. Because of the lack of consistency between the cohort and case-control studies, the epidemiological studies can do no more than suggest a causal role of occupational exposure to formaldehyde in squamous-cell carcinoma of the nasal cavities and paranasal sinuses.

Less information was available to evaluate the association of formaldehyde with adenocarcinoma of the nasal cavities and paranasal sinuses, and the small excess observed in one case-control study in Denmark may have been confounded by exposure to wood dust.

Neither cohort nor case-control studies showed excess risks for oropharyngeal, laryngeal or lung cancer among workers exposed to formaldehyde. The studies of industrial cohorts also showed low or no risk for lymphatic or haematopoietic cancers; however, the cohort studies of embalmers, anatomists and other professionals who use formaldehyde tended to show excess risks for cancers of the brain, although they were based on small numbers. These findings are countered by a consistent lack of excess risk for brain cancer in the studies of industrial cohorts, which generally included more direct and quantitative estimates of exposure to formaldehyde than did the cohort studies of embalmers and anatomists.

### 5.3 Animal carcinogenicity data

Formaldehyde was tested for carcinogenicity by inhalation in mice, rats and hamsters, by oral administration in drinking-water in rats, by skin application in mice, and by subcutaneous injection in rats. In additional studies in mice, rats and hamsters, modification of the carcinogenicity of known carcinogens was tested by administration of formaldehyde in drinking-water, by application on the skin or by inhalation.

Several studies in which formaldehyde was administered to rats by inhalation showed evidence of carcinogenicity, particularly induction of squamous-cell carcinomas of the nasal cavities, usually only at the highest exposure. Similar studies in hamsters showed no evidence of carcinogenicity. Studies in mice either showed no effect or were inadequate for evaluation. In rats administered formaldehyde in the drinking-water, increased incidences were seen of forestomach papillomas in one study and of leukaemias and gastrointestinal tract tumours in another; two other studies in which rats were treated in the drinking-water gave negative results. Studies in which formaldehyde was applied to the skin or injected subcutaneously were inadequate for evaluation.

In experiments to test the effect of formaldehyde on the carcinogenicity of known carcinogens, oral administration of formaldehyde concomitantly with *N*-nitrosodimethylamine to mice increased the incidence of tumours at various sites; skin application in addition to 7,12-dimethylbenz[*a*]anthracene reduced the latency of skin tumours. In rats, concomitant administration of formaldehyde and *N*-methyl-*N'*-nitro-*N*-nitrosoguanidine in the drinking-water increased the incidence of adenocarcinoma of the glandular stomach. Exposure of

hamsters by inhalation to formaldehyde increased the multiplicity of tracheal tumours induced by subcutaneous injections of *N*-nitrosodiethylamine.

#### 5.4 Other relevant data

The concentration of endogenous formaldehyde in human blood is about 2-3 mg/L; similar concentrations are found in the blood of monkeys and rats. Exposure of humans, monkeys or rats to formaldehyde by inhalation does not alter the concentration of formaldehyde in the blood.

Occupational exposure to formaldehyde results in damage to nasal tissues; however, these findings may have been confounded by concomitant exposures. No data were available on the induction of cell proliferation in humans. There are no conclusive data showing that formaldehyde is toxic to the immune system, to the reproductive system or to developing fetuses in humans.

More than 90% of inhaled formaldehyde gas is absorbed in the upper respiratory tract of rats and monkeys. In rats, it is absorbed in the nasal passages; in monkeys, it is also absorbed in the nasopharynx, trachea and proximal regions of the major bronchi. In mice exposed to high concentrations of formaldehyde, minute ventilation is decreased by 50% throughout exposure, resulting in a lower effective dose. This occurs only transiently in rats, as the minute ventilation is rapidly restored. Formaldehyde is rapidly oxidized to formate, which is incorporated into biological macromolecules, excreted in the urine or oxidized to carbon dioxide.

Acute or subacute exposure of rats to a concentration of 2.5 mg/m<sup>3</sup> appears to cause no detectable damage to the nasal epithelium and does not significantly increase rates of cell turnover. Cell turnover rates in rat nose during subchronic or chronic exposures to formaldehyde do not increase at 2.5 mg/m<sup>3</sup>, increase marginally at concentrations of 3.7-7.4 mg/m<sup>3</sup> and increase substantially at concentrations of 12.3-18.4 mg/m<sup>3</sup>. Concentration is more important than length of exposure in determining the cytotoxicity of formaldehyde.

Inhalation of formaldehyde leads to the formation of DNA-protein cross-links in the nasal respiratory mucosa of rats and monkeys. Much lower levels of DNA-protein cross-links were found in the nasopharynx, trachea and carina of some monkeys, in decreasing concentrations with passage through the respiratory tract, but none were found in the maxillary sinus. The formation of DNA-protein cross-links is a sublinear function of the formaldehyde concentration in inhaled air from 0.86 to 18.4 mg/m<sup>3</sup>, and the yield of DNA-protein cross-links at a given inhaled concentration is approximately an order of magnitude lower in monkeys than in rats. Yields of DNA-protein cross-links are higher in the lateral meatus of the rat nose and lower in the medial and posterior meatuses. There is no detectable accumulation of DNA-protein cross-links during repeated exposure.

About 50% of formaldehyde-induced tumours in the nasal mucosa of rats have a point mutation in the *p53* tumour suppressor gene.

No adequate data were available on genetic effects of formaldehyde in humans. It is comprehensively genotoxic in a variety of experimental systems, ranging from bacteria to rodents, *in vivo*. Formaldehyde given by inhalation or gavage to rats *in vivo* induced chromosomal anomalies in lung cells, micronuclei in the gastrointestinal tract and spermhead anomalies.

Formaldehyde induced DNA-protein crosslinks, DNA single-strand breaks, chromosomal aberrations, sister chromatid exchange and gene mutation in human cells *in vitro*. It induced cell transformation, chromosomal aberrations, sister chromatid exchange, DNA strand breaks, DNA-protein crosslinks and gene mutation in rodent cells *in vitro*.

Administration of formaldehyde in the diet to *Drosophila melanogaster* induced lethal and visible mutations, deficiencies, duplications, inversions, translocations and crossing-over in spermatogonia. Formaldehyde induced mutation, gene conversion, DNA strand breaks and DNA-protein crosslinks in fungi and mutation and DNA damage in bacteria.

In rodents and monkeys, there is a no-observable-effect level (2.5 mg/m<sup>3</sup>) of inhaled formaldehyde with respect to cell proliferation and tissue damage in otherwise undamaged nasal mucosa. These effects are considered to contribute to subsequent development of cancer. Although these findings provide a basis for extrapolation to humans, conclusive data demonstrating that such cellular and biochemical changes occur in humans exposed to formaldehyde are not available.

## 5.5 Evaluation

There is *limited evidence* in humans for the carcinogenicity of formaldehyde.

There is *sufficient evidence* in experimental animals for the carcinogenicity of formaldehyde.

### Overall evaluation

Formaldehyde *is probably carcinogenic to humans (Group 2A)*.

For definition of the italicized terms, see [Preamble Evaluation](#)

**Previous evaluation:** Suppl. 7 (1987) (p. 211)

### Synonyms

- BFV
- FA
- Fannoform
- Floguard 1015
- FM 282
- Formaldehyde, gas
- Formalin
- Formalin 40
- Formalith
- Formic aldehyde
- Formol
- Fyde
- Hoch
- Ivalon
- Karsan
- Lysoform
- Methaldehyde
- Methyl aldehyde
- Methyl oxide
- Methylene oxide
- Morbicid
- Oxomethane
- Oxymethylene
- Paraform
- Superlysoform