# EXPOSURES TO HAZARDOUS AIBORNE SUBSTANCES IN THE WOOD CONVERSION SECTOR

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**Reference**

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**Disclaimer**

Views and/or conclusions in this report are those of Massey University and may not reflect the position of ACC.

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**List of abbreviations used**

| | |
|---|---|
| **AM** | arithmetic mean |
| **ACGIH** | American Conference of Governmental Industrial Hygienists |
| **CCA** | chromated copper arsenate |
| **CFU** | colony forming units |
| **CI** | (95%) confidence interval |
| **DFG** | Deutsche Forschungsgemeinschaft. Federal Republic of Germany Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area. |
| **EU** | endotoxin units |
| **f/cc** | fibres per cubic centimetre of air |
| **$FEV_1$** | forced expiratory volume in one second |
| **$FEV_1$/FVC** | the ratio between forced expiratory volume in one second and forced vital capacity |
| **FVC** | forced vital capacity |
| **GM** | geometric mean |
| **GSD** | geometric standard deviation |
| **HSE** | UK Health and Safety Executive |
| **IARC** | International Agency for Research on Cancer |
| **LEV** | local exhaust ventilation |
| **MAK** | maximum allowable concentrations set by DFG |
| **MDF** | medium density fibreboard |
| **NIOSH** | US National Institute for Occupational Safety and Health |
| **OR** | odds ratio |
| **OSB** | oriented strand board |
| **OSHA** | US Occupational Health and Safety Administration |
| **PB** | particle board |
| **PEFR** | peak expiratory flow rate |
| **PEL** | permissible exposure limit set by OSHA |
| **PPMV** | parts per million per volume |
| **REL** | NIOSH recommended exposure limit |
| **RR** | relative risk |
| **SD** | standard deviation |
| **SE** | standard error |

| | |
|---|---|
| **SIR** | standardised incidence ratio |
| **SMR** | standardised mortality ratio |
| **SPIR** | standardised proportional incidence ratio |
| **TLV** | threshold limit value |
| **TWA** | time-weighted average |
| **VC** | vital capacity |
| **FEF** | forced expiratory flow |

## Executive summary

This report on the key airborne exposures and associated health risks in the wood conversion sector has been produced as part of a study funded by ACC under RFP PL160407. The main components of the study were a literature review (Part I) and an exposure survey (Part II). The literature review has focussed on exposures and related health effects in: 1) plywood, particle board and medium density fibreboard (MDF) manufacturing; 2) the pulp and paper industry; and 3) the joinery and furniture making industry. The exposure survey involved collecting dust and formaldehyde exposure measurements in small, medium and large joineries and furniture makers in New Zealand. Exposure and health effects associated with sawmilling will not be discussed as it was specifically excluded from the RFP. In the following bullet points we will summarise the main findings, followed by a more detailed summary of the literature review and exposure survey.

- The wood conversion sector in New Zealand in 2006 employed 20,099 workers in the category of "sawmills, planing and other wood mills". Within this category, the "plywood and veneer manufacturing" subcategory employed 1,770 people, and the "pulp, paper and paperboard manufacturing" subcategory employed 2,050. 16,929 people were employed as carpenters or joiners and 3,576 as cabinet makers or furniture makers.
- Relatively few studies have assessed exposures or health effects in workers employed in the plywood, particle board and the MDF industry.
- Those studies that have been reported show some evidence of respiratory symptoms, lung function deficit and dermatitis, however, the evidence is mixed.
- Most research has been conducted on the pulp and paper industry.
- Exposure to several irritant agents in pulp and paper mill workers has been shown to cause a range of non-malignant respiratory symptoms, and epidemiological studies have suggested increased mortality from nervous, gastro-intestinal and respiratory system cancers, and certain lymphatic and haematopoietic neoplasms, as well as from cardiovascular disease.
- A number of studies have evaluated both exposures and health effects in the joinery and furniture making industry.

- Average inhalable wood dust levels well in excess of 1 mg/m³ are common.
- Wood dust exposures in this industry have been associated with increased mortality and morbidity from non-malignant respiratory disease.
- Large excesses of sino-nasal cancer risk have been observed in furniture workers processing hardwoods, with a much lower excess observed in those handling softwoods and/or MDF. Associations between wood dust exposure and several other cancers have also been observed.
- A significant proportion of New Zealand joinery and furniture workers (19% and 8% respectively) are exposed to inhalable dust levels in excess of the current New Zealand occupational exposure limit of 5 $mg/m^3$.
- The majority of New Zealand joinery and furniture workers (87% and 63% respectively) are exposed to inhalable dust levels in excess of international standards of 1 $mg/m^3$.
- Formaldehyde exposures are very low in both joinery and furniture workers.
- The relatively large proportion of workers exposed to dust levels in excess of 1 $mg/m^3$ suggests that many New Zealand (and international) joinery and furniture workers are at risk of developing work-related respiratory disease.

***Part I***

The wood conversion sector is a major industrial sector in New Zealand. In 2006 it employed 20,099 workers in the category of "sawmills, planing and other wood mills". Within this category, the "plywood and veneer manufacturing" subcategory employed 1,770 people. The total number employed in the category of "paper and paper products" was 5,000. Within this category the "pulp, paper and paperboard manufacturing" subcategory employed 2,050. A total of 16,929 people were employed as carpenters or joiners and 3,576 as cabinet makers or furniture makers.

In contrast to solid wood products milled from logs, manufactured wood products are composed of wood "pieces" of varying sizes from sliced veneers down to fibres which are held together by either chemical adhesives or natural chemical bonds. The main distinguishing feature of the different products is the size of the wood pieces used; from veneers in *plywood*, wood chips, flakes or strands in *particleboard*, to

wood fibres in *MDF* or *hardboard*. Pulp (for the production of paper) is produced from wood chips and is most commonly produced by a chemical pulping process which involves digesting wood chips with steam and a solution of sodium sulphide and sodium hydroxide. Paper production (from pulp) involves bleaching and subsequent treatments with sodium hydroxide, calcium or sodium hypochlorite, and chlorine dioxide. The joinery and furniture making industries use a combination of solid wood and reconstituted wood products. Occupational hazards in these industries relate predominantly to wood dust and formaldehyde emissions, from either the reconstituted wood products or other glues and resins used, and in some instances to microbial agents.

Formaldehyde is an important component of the wood processing industry, with phenol-, urea-, and melamine- formaldehyde resins being used as adhesives in the manufacture of plywood, and as adhesives and impregnating resins in the manufacture of particle board, oriented strandboard and MDF. Paper mills also produce some specialty products that are coated with formaldehyde-based resins. Formaldehyde has recently been reclassified by the International Agency for Research on Cancer (IARC) and reaffirmed as a Group 1 (proven human) carcinogen, citing sufficient evidence that formaldehyde causes nasopharyngeal cancer and leukaemia (particularly myeloid leukaemia) in humans. Other health effects known to be caused by exposure to formaldehyde include a range of non-malignant respiratory effects including irritation of mucous membranes, asthma, reactive airways dysfunction syndrome (RADS), and also allergic contact dermatitis. The New Zealand Workplace Exposure Standard (ceiling limit of 1 ppm) is currently less stringent than internationally recommended occupational exposure standards, but more stringent than standards in countries such as Australia and the UK.

Wood dust is a common occupational exposure, estimated to affect 5.6% of the working population in New Zealand. There is sufficient evidence that both hardwood and softwood dust cause respiratory cancers, and all wood dust has been recently reclassified by IARC and reaffirmed as a Group 1 carcinogen known to cause sinonasal cancer in humans. Other health effects known to be caused by exposure to wood dust include non-malignant effects such as asthma, airflow obstruction, and both upper and lower respiratory symptoms. These effects have been reported at

levels well below the current New Zealand workplace exposure limit of 5 mg/m³, which is less stringent than the limit of 1 mg/m3 recommended internationally.

Microbial exposures have been shown to be elevated in plywood manufacturing, furniture making and joineries, but levels were generally relatively low. Microbial agents are therefore unlikely to play a major role in occupational respiratory disease in wood processing workers (perhaps with the exception of sawmill workers who have been shown to be exposed to higher levels, but were excluded from this review).

Surprisingly few studies of exposure and/or health effects have been conducted in plywood mills and particle board and MDF production industries. The few studies conducted suggest an increased risk of sarcoma, and respiratory and skin symptom in plywood workers. Only a few studies of health effects have been reported for the particle board manufacturing industry which showed mixed evidence of respiratory symptoms, lung function deficit and dermatitis. The risks for MDF workers are unclear since only one small health study in MDF manufacturing workers has been conducted. The exposure levels for formaldehyde in these industries were generally elevated, but were below the current threshold limit values. Mean inhalable dust levels were below the current standard of 5 mg/$m^3$, but regularly exceeded the more stringent international exposure standard of 1 mg/$m^3$. On the basis that only a few studies are available a valid assessment of current exposure levels and associated health risks can not be made for these industries. Therefore, given the nature of the exposures (wood dust and formaldehyde) and its associated health risks, more studies are needed to further characterise and quantify exposure levels and assess their potential health risks.

The wood conversion industry sector with the most information on exposures and health effects available in the scientific literature is the pulp and paper industry. The complex mix of exposures in the industry, including many known or suspected carcinogens, has been very well characterised and numerous cohort and case-control studies have been conducted. Exposure to several irritant agents in pulp and paper mill workers has been shown to cause a range of non-malignant respiratory symptoms. Epidemiological studies have also suggested increased mortality from nervous, gastro-intestinal and respiratory system cancers, and certain lymphatic and

haematopoietic neoplasms, as well as from cardiovascular disease. Among specific exposures examined, sulphur dioxide and asbestos have been associated with respiratory system cancers, and volatile organochlorine compounds with an increase in all-cancer mortality.

A relatively large number of studies have evaluated both exposures and health effects in the joinery and furniture making industry. The most significant exposures observed have been to inhalable wood dust, with only relatively low exposures to formaldehyde, bacterial endotoxin and monoterpenes reported. Average inhalable wood dust levels were often in excess of 1 mg/m³, but generally below 5 mg/m³. Wood dust exposures in this industry have been associated with increased mortality from non-malignant respiratory disease, and also with the prevalence of a range of non-malignant respiratory diseases such as reduced lung function, asthma and chronic bronchitis. Large excesses of sino-nasal cancer risk have been observed in furniture workers processing hardwoods such as beech and oak, with a much lower although still statistically significant excess observed in those handling softwoods and/or MDF. Associations between wood dust exposure and several other cancers have also been observed.

Only one intervention study which specifically focussed on control measures for wood dust in small wood working shops has been identified. Rather than focusing on technical and engineering solutions, the study applied a health promotion model aimed at changing health behaviours. This study – as well as other studies in other industries - showed that educational intervention measures alone result in only a modest reduction in (wood) dust exposure i.e. a reduction in dust exposure of only 10.4% was achieved.

***Part II***

The exposure survey in the New Zealand joinery and furniture making industry involved measurements of airborne wood dust and formaldehyde and a target of 300 personal inhalable dust samples, 100 personal respirable dust samples, and 300 personal formaldehyde samples was set. We recruited 22 joineries and 8 furniture manufacturing factories. From these 30 factories, a total of 170 employees (96 joiners

and 74 furniture makers) participated in the exposure survey. Repeat samples were taken from 125 employees (74%) for inhalable dust, respirable dust, or formaldehyde measurements, or any combination of these three exposures. Personal inhalable and respirable dust was sampled during a full eight-hour shift (or close to full shift) using PAS-6 and Casella samplers respectively. All formaldehyde samples taken were 15 minute personal samples collected using commercially available (Waters Sep-Pak XPoSure™ Aldehyde Sampler) formaldehyde cartridges. Due to pump faults, filter tampering, and other exclusions, a total of 266 personal inhalable dust samples and 81 personal respirable samples were available for gravimetric analyses. Due to formaldehyde cartridges used for calibration and blanks, and exclusion of cartridges due to pump failures, a total of 274 personal formaldehyde samples were available for analyses.

The mean personal inhalable dust exposure for all joinery workers was 2.48 $mg/m^3$; the mean personal exposure for all furniture makers was approximately half that of the joinery workers (1.22 $mg/m^3$). The overall average of inhalable wood dust exposure for all joinery and furniture workers combined was 1.82 $mg/m^3$. A significant proportion of joinery and furniture workers (19% and 8% respectively) were exposed to levels in excess of the current New Zealand occupational exposure limit of 5 $mg/m^3$. More than 75% of all workers were exposed to levels in excess of current international standards of 1 $mg/m^3$. As was the case with inhalable dust the highest respirable dust levels (i.e. the smaller fraction of inhalable dust that can reach the lower airways) were measured in joinery workers with a mean of 0.27 $mg/m^3$ compared to less than half that (0.12 $mg/m^3$) in furniture makers. The overall mean respirable wood dust exposure for all workers was 0.18 $mg/m^3$. Using the overall dust levels, the respirable dust fraction was determined to be 11% of the total inhalable dust fraction in joinery workers and 10% in furniture workers. Formaldehyde levels were low in both joinery (0.014 ppm) and furniture workers (0.012 ppm).

Given that many of the effects described in Part I occur at dust levels well below 5 $mg/m^3$, and that a relatively large proportion of workers are exposed to dust levels in excess of 1 $mg/m^3$ (an occupational exposure limit recommended in many other countries), many New Zealand joinery and furniture workers may be at risk of developing work-related respiratory disease. Cost effective workplace interventions to

reduce wood dust exposures in joinery and furniture workers are therefore urgently needed. The relatively low dust exposures measured in overseas furniture making factories suggest that average exposure levels of less than 1 mg/$m^3$ are feasible. We hope that this report will contribute to the development of these improved and cost-effective control measures.

## 1. Introduction

This report on the key airborne exposures and associated health risks in the wood conversion sector has been produced as part of a study funded by ACC under RFP PL160407. In the RFP the wood conversion industry was defined by ACC as industries involved in the production of: (i) pulp and paper (including fine chemicals); (ii) veneer and plywood; and (iii) fibre and particle board. The original RFP specifically excluded sawmilling, joinery and furniture making.

The main components of the study were an exposure survey and a literature review. The exposure survey involved collecting dust and formaldehyde exposure measurements in the plywood/particle board and medium density fibreboard (MDF) manufacturing industries, and the literature review as a consequence was intended to be focussed predominantly on these industries. However, during a meeting with representatives of several of the larger plywood and particle board manufacturing plants, on 23 January 2008, concerns were raised by the industry regarding the publication of exposure data. Despite subsequent written and verbal communications between ACC, the Centre for Public Health Research and the industry no agreement could be reached that was satisfactory to all parties involved. As a consequence the original study aims and design have been modified to allow a similar study to instead be conducted in another branch of the wood conversion industry, i.e. joiners and furniture makers that are involved in processing plywood, MDF and particle board (PB). The literature review therefore has been extended to include these industries. Thus the current review will focus on exposures and related health effects in: 1) plywood, PB and MDF manufacturing; 2) the pulp and paper industry; and 3) the joinery and furniture making industry.

The report consists of two parts, with the first part comprising the literature review. The objectives of the literature review were: (i) to provide an overview of the key airborne exposures and associated health risks in the wood conversion sector; (ii) to compare exposure levels reported in the literature with national and international limits; and (iii) to assess which strategies have been most effective in reducing exposure in other countries.

In the first part of the report we also describe some industry statistics as well as the production processes for plywood, PB, MDF, and pulp and paper production, followed by a review of the literature regarding exposures and health effects for each of these industries.

For the literature review we performed a search of PubMed and Web of Science databases from 1968 to 2009. We used a range of keywords: wood conversion exposure; wood health effects; pulp paper exposure; pulp paper health effects; plywood exposure; plywood health effects; particleboard exposure; particleboard health effects; fibre board exposure; fibre board health effects; veneer exposure; veneer health effects; sawmill workers dust; wood dust exposure; pulp paper mortality; organochlorine cancer; organochlorine health effects; organochlorine mortality; organochlorine paper; organochlorine pulp; organochlorine occupational; organochlorine wood industry; organochlorine wood mill; formaldehyde cancer; formaldehyde health effects; formaldehyde mortality; formaldehyde wood industry; formaldehyde occupational; formaldehyde symptoms; joinery; joiner cancer; joinery health effects; joiner mortality; joinery exposure; joinery formaldehyde; furniture makers cancer; furniture makers health effects; furniture makers mortality; furniture makers exposure.

The second part of the report describes the exposure survey conducted in the joinery and furniture making industry. The main aims of the exposure survey were: (i) to assess personal inhalable and respirable dust exposure levels in a representative sample of workers in this industry; (ii) to assess personal airborne formaldehyde exposures in a representative sample of workers in this industry; (iii) to compare these levels with current and proposed exposure limits for wood dust and formaldehyde.

For the exposure survey we recruited 22 joineries and 8 furniture making factories. From these 30 factories, a total of 170 employees (96 joiners and 74 furniture makers) participated in the exposure survey. A target of 300 personal inhalable dust samples, 100 personal respirable dust samples, and 300 personal formaldehyde samples was set.

The work described in this report was overseen by a steering committee which comprised ACC representatives (Dr John Wallaart and Dr Peter Larking), an industry representative (Mr Peter Bodeker), a union representative (Ms Jills Angus Burney), two occupational medicine specialists (A/Professor Bill Glass and Dr Chris Walls), and two of the researchers (Dr Dave McLean and Professor Jeroen Douwes).

# PART I:  Literature Review

## 2. Wood conversion industry in New Zealand

### 2.1 Production

The latest figures available for the production of wood products in New Zealand are from 2007 (MAF, 2008). In this period, production of plywood totalled 421,794 $m^3$, with another 24,972 $m^3$ imported. A total of 80,250 $m^3$ was exported, and total apparent local consumption was 366,516 $m^3$. Veneer production was 688,312 $m^3$, including veneer intended for further production into plywood in New Zealand.

Particleboard production in 2007 (including particleboard, strandboard and triboard from the December 2006 quarter) was 256,239 $m^3$. Prior to this, triboard and strandboard had been included in the "fibreboard" category. A total of 2,614 $m^3$ of particleboard was imported, and 118,167 $m^3$ was exported, and the total apparent consumption in New Zealand was 140,686 $m^3$.

Fibreboard production in 2007 (including hardboard, softboard and MDF from the December 2006 quarter) was 836,755 $m^3$. A total of 19,874 $m^3$ of fibreboard was imported and 607,438 $m^3$ was exported. The total consumption in New Zealand can therefore be estimated to be 249,191 $m^3$.

The production of mechanical and chemical pulp in New Zealand totalled 1,536,301 tonnes in 2007, with 630,003 tonnes of chemical wood pulp and 236,505 tonnes of mechanical wood pulp exported. Total paper and paperboard production was 862,652 tonnes.

### 2.2 Number of workers employed

The number of people employed in the category of "sawmills, planing and other wood mills" totalled 20,099 in 2006 (down 2.7% from 2005, but up from 18,500 workers in 2002). Within this category, the "plywood and veneer manufacturing" subcategory employed 1,770 people in 2006 (down 6.3% from 2005, which follows a decreasing trend in employees since 2002 when there were 2100 workers). The total number employed in the category of "paper and paper products" was 5,000 in 2006 (down

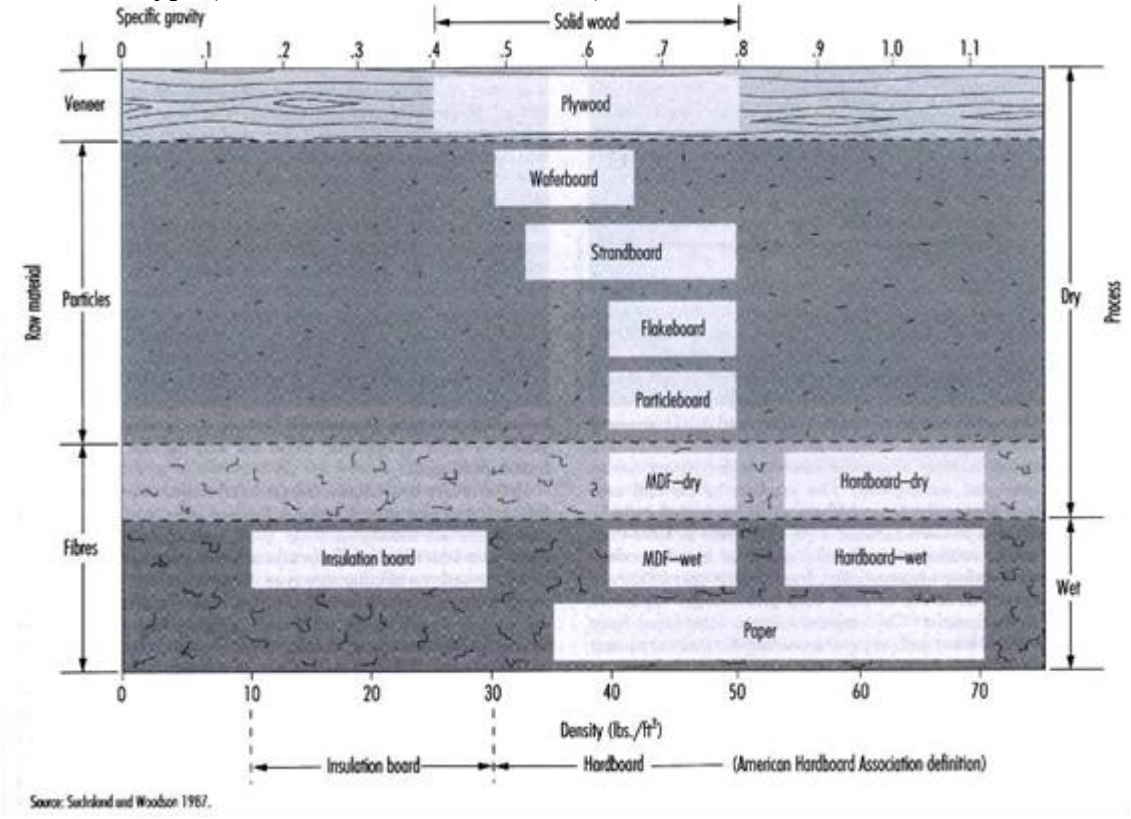
11.3% from 2005, and down 26.1% from the total of 6770 workers in 2002). Within this category the "pulp, paper and paperboard manufacturing" subcategory employed 2,050 people in 2006 (down 6.0% from 2005, which continues a decreasing trend in employees since 2002 when there were 2,990 workers). In 2006, a total of 16,929 people were employed as carpenters or joiners and 3,576 as cabinet makers or furniture makers.

## 2.3 Industry processes

In contrast to solid wood products milled from logs, manufactured wood products are composed of wood "pieces" of varying sizes from sliced veneers down to fibres which are held together by either chemical adhesives or natural chemical bonds. The main distinguishing feature of the different products is the size of the wood pieces used; from veneers in *plywood*, wood chips, flakes or strands in *particleboard* (PB), to wood fibres in *medium density fibreboard* (MDF) or *hardboard*. The relationship between the various types of manufactured wood products is demonstrated in Figure 1 below, reproduced from Suchsland & Woodson (1987).

Plywood (and laminated veneer lumber), strandboard, PB and MDF are produced in New Zealand (see above). Around 850,000 $m^3$ of the 12.3 million $m^3$ total world production of MDF is manufactured in New Zealand.

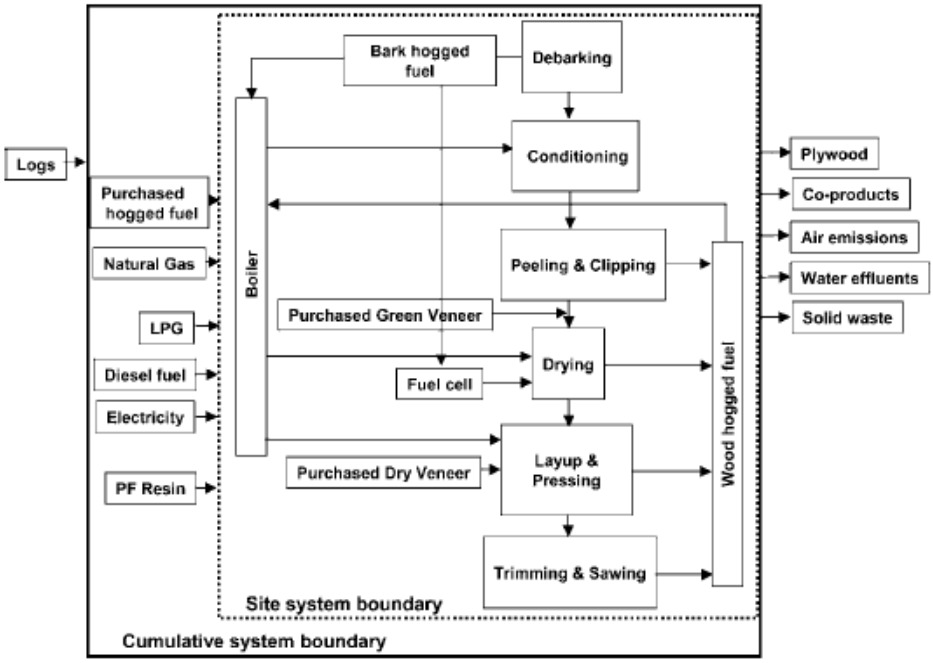
**Figure 1.** Classification of manufactured boards by particle size, density and process type (Suchsland & Woodson, 1987).



### 2.3.1 Plywood processing

The process of plywood production begins by mechanically removing the bark from the logs and cutting them to proper sized wood 'blocks' for peeling (Wilson & Sakimoto, 2005). The blocks are often conditioned by steaming to improve the quality of the veneer. The blocks are rotary peeled into long ribbons of veneer of 2-6mm thickness, clipped to size and sorted by their moisture content. The veneer's moisture content is reduced from 25-100% to 3-5% in veneer driers, after which phenol-, resorcinol- or urea-formaldehyde resin is applied. The sheets are stacked and placed in a hot press, with pressure and heat providing contact and curing, thus bonding the veneers to make plywood. The finishing processes include knothole filling and sanding. The cured panels are removed and sawn to standard sizes, usually 1.22 x 2.44 m (4 x 8 ft)[2]. See Figure 2 below from Wilson & Sakimoto, 2005 for an overview of the process.

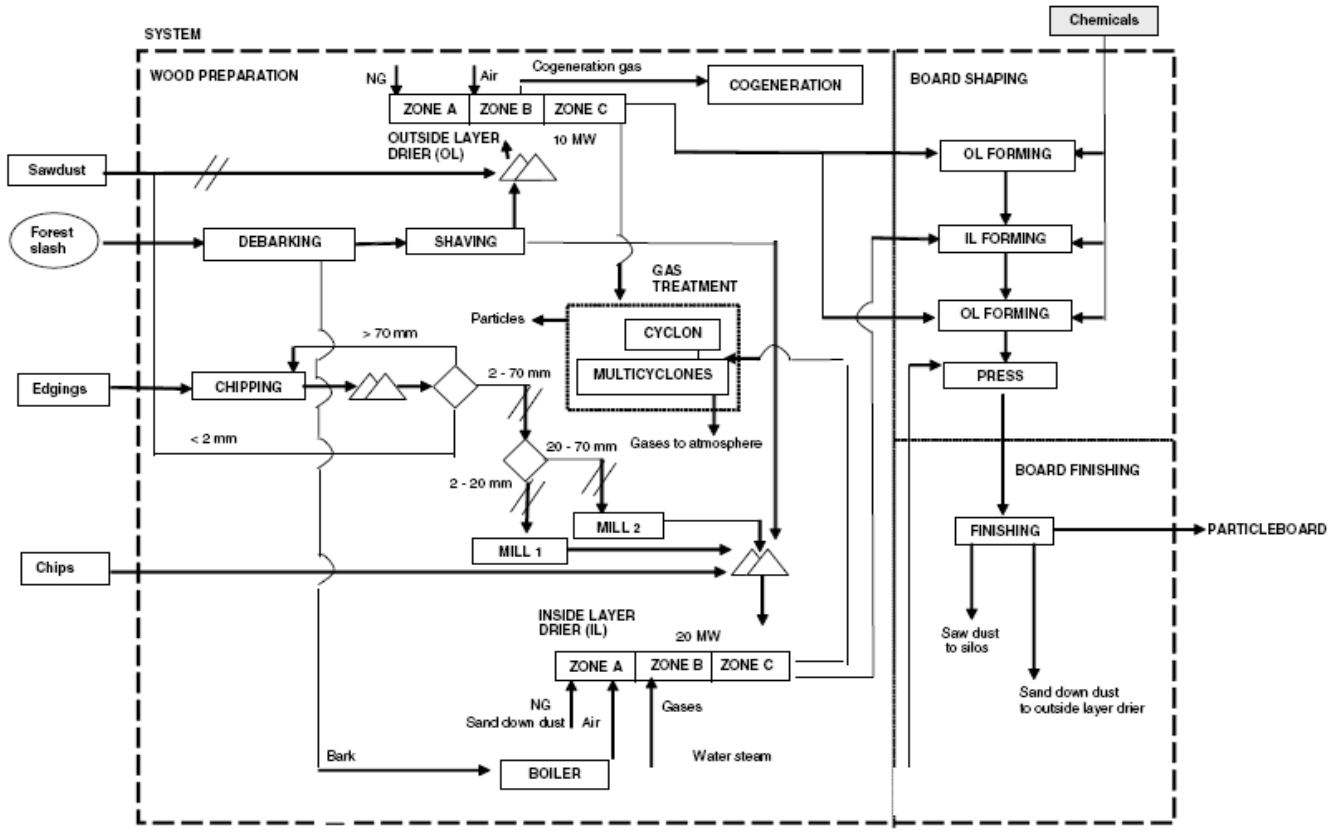
**Figure 2**. System boundaries for both the cumulative and mill site for modelling the plywood manufacturing process. (Wilson & Sakimoto, 2005)



### 2.3.2 Particleboard processing

PB panels are made mostly from branches, chips, shavings, and sawdust (Rivela *et* al., 2006). Bark is removed and the logs are shaved and chipped into flakes of a desired particle size. Particles of different sizes and moisture content are placed in different silos, where they are dried with hot gas, with different combinations of feed silos used to adjust the mass flow entering the units of the process to make up boards consisting of three layers, i.e. fine particles on the outside for smoothness and coarse particles on the inside for strength. The wood raw material is then blended with binding agents, such as urea-formaldehyde, with the resin and other glue additives (including paraffin wax to provide water resistance) sprayed onto the wood particles, after which the mat is formed. The mats are then transferred to the hot press to be pressed and cured. Board finishing consists of cooling the hot boards, cutting the material to size and sanding it into the final product. See Figure 3 below from Rivela *et* al., 2006 for an overview of the process.

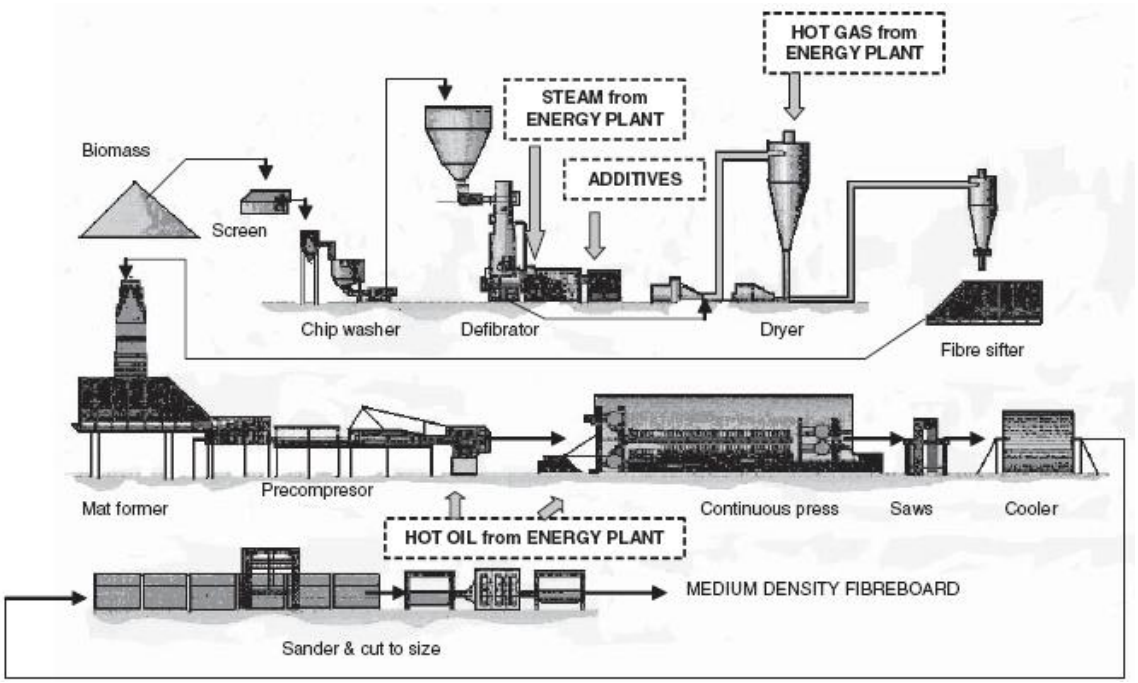
**Figure 3**. Flow sheet of particleboard manufacture. Symbols: NG: natural gas; (Δ): silo; (--//--): screening unit; (◊): classification unit (Rivela *et* al., 2006).



### 2.3.3 Medium density fibreboard processing

Medium density fibreboard (MDF) is made from mainly wood chip, which is refined and softened in a pressurized chamber, and pulped into fibres. The fibres are dried and blended with resin, other additives and sometimes wax to protect against accidental water damage. The resin coated material is formed into a mat and pressure is applied by rollers before loading onto a hot press to activate curing of the resin and bonding of the fibres into a solid panel. The boards are then cooled, sanded, trimmed and sawn to final standard size (Rivela *et al*., 2007). See Figure 4 below from Rivela *et* al., 2007 for an overview of the process.

**Figure 4**. Flow sheet of MDF manufacture (Rivela *et* al., 2006).



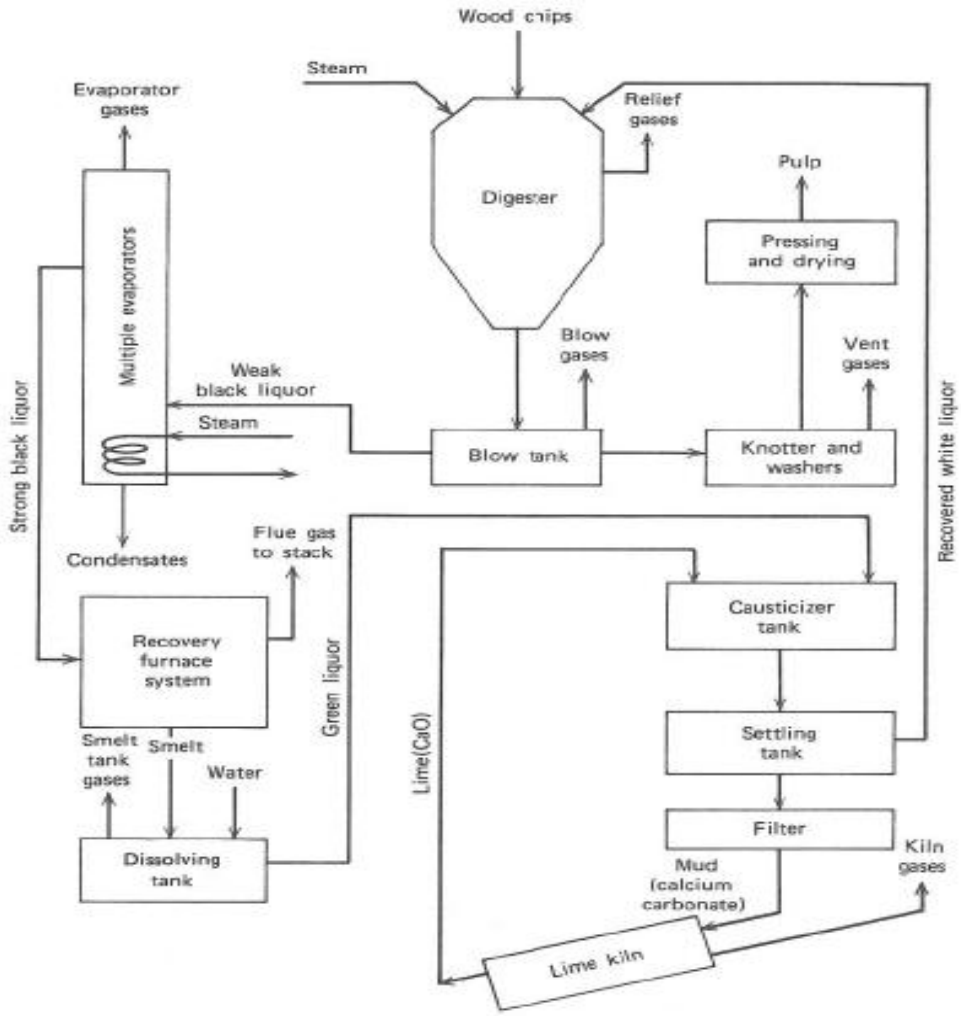
### 2.3.4 Pulp and paper processing

Pulp can be produced by either chemical or mechanical processes, although chemical process methods are the most common (Burgess, 1995).

The most common chemical pulping process is the Kraft or Sulphate process. A digesting tank digests wood chips with steam and a solution of sodium sulphide and sodium hydroxide. The digested wood chips are then moved to the blow tank where gas is blown from the pulp and digestion liquid. The used solution is drained off, following which the pulp is washed, screened and bleached. The drained-off chemicals are recovered by multiple-effect evaporators and dissolved in smelt tanks, where quicklime is added to convert the sodium carbonate to sodium hydroxide. The calcium carbonate thus formed is converted to calcium oxide in the lime kiln, and then slaked with water to produce calcium hydroxide. See Figure 5 below from Burgess 1995 for an overview of the process.

Paper production is usually carried out next to the pulp mill and begins with bleaching of the pulp with chlorine. Elemental chlorine-free bleaching of pulp is now the most popular method accounting for 75% of bleached Kraft pulp globally (ENSIS/CSIRO, 2005). Extraction with sodium hydroxide follows, then calcium or sodium hypochlorite, and lastly, treatment with chlorine dioxide. Beating and refining steps are repeated a number of times, and additives are mixed with the pulp. The consistency of the pulp is adjusted by adding water. Solids content is increased by passing the pulp through a wire sieve and to a heated cylinder. The paper may be coated with clay, mica, talk, casein, soda ash, dyes, plastics, gums, varnishes, linseed oil, organic solvents, and plastics (Burgess, 1995).

**Figure 5**. Flow diagram of a pulp plant (Burgess 1995).



## 3. Specific exposures in the wood conversion industry

The three most significant airborne exposures common to the wood conversion industry are formaldehyde, wood dust and microbial agents. In this chapter each of these exposures will be discussed, including an outline of the known health effects, the relevant occupational exposure standards and where available the levels of exposure encountered in various processes. Studies relevant to each of the specific wood conversion processes will be discussed in more detail in Chapter 4.

### 3.1 Formaldehyde

**Summary**

- Formaldehyde exposure is common in all sectors of the wood conversion industry.
- There is sufficient evidence that formaldehyde causes nasopharyngeal cancer and leukaemia in humans.
- Formaldehyde is classified by the International Agency for Research on Cancer as a Group 1 (proven human) carcinogen.
- Other health effects known to be caused by exposure to formaldehyde include a range of non-malignant respiratory effects including irritation of mucous membranes, asthma, reactive airways dysfunction syndrome (RADS), and also allergic contact dermatitis.
- The New Zealand Workplace Exposure Standard is currently less stringent than internationally recommended occupational exposure standards, but more stringent than standards in countries such as Australia and the UK.

### 3.1.1 Introduction

Formaldehyde is an important component of the wood processing industry, with phenol-, urea-, and melamine- formaldehyde resins being used as adhesives in the manufacture of plywood, and as adhesives and impregnating resins in the manufacture of PB, oriented strandboard (OSB) and MDF. Paper mills also produce some specialty products that are coated with formaldehyde-based resins (IARC, 2006).

Formaldehyde occurs as a natural product in most living systems and in the environment. It is used as a disinfectant and preservative in the form of an aqueous solution (formalin), and is widely used in the production of adhesives and binders for use in the wood, plastics, textiles, leather and related industries (IARC, 1995). Given its widespread use many millions of people worldwide are exposed to formaldehyde environmentally and/or occupationally. Occupational exposure occurs in those industries employed in the direct manufacture of products containing formaldehyde, and also in those industries that use these products such as furniture manufacture or construction. Environmental exposure generally occurs at lower levels in buildings containing manufactured wood products or furniture and textiles that contain formaldehyde, and in the outdoor air from automobile emissions, tobacco smoke and other combustion sources, and from industrial discharges. Formaldehyde is present in all living cells in small amounts, as it is derived from the metabolism from numerous sources, including serine, glycine, sarcosine, choline, and methionine (Nelson *et al*., 1986).

### 3.1.2 Health effects

The upper respiratory tract, including the nasopharynx, mouth, salivary gland, nasal cavity, and larynx, comes into direct contact with formaldehyde upon inhalation, and many processes also expose workers to skin contact. Health effects related to formaldehyde exposure include irritation of mucous membranes and the respiratory system, respiratory effects including asthma and pneumonitis (Graa-Thomsen, *et al*., 1995), and the exacerbation of pre-existing respiratory conditions; chemical burns (Kanerva *et al*., 1994), skin depigmentation, and irritant and allergic contact dermatitis (Bruze *et al*., 1985), and several types of cancer. A comprehensive

summary of the evidence available from human epidemiological data, experimental studies in animals and of the toxicokinetics and metabolism of formaldehyde is available in Volume 88 of the International Agency for Research on Cancer (IARC) Monographs on the Evaluation of Carcinogenic Risks to Humans (IARC, 2006). The carcinogenic potential of formaldehyde was also reviewed by IARC as part of Volume 100 - A Review of Human Carcinogens in October 2009, and the working group concluded that in addition to the recognised risk of nasopharyngeal cancer there is also now sufficient evidence for leukaemia and in particular myeloid leukaemia (Baan *et al,* 2009).

***Non-malignant effects***

Numerous studies have examined the respiratory effects of environmental formaldehyde exposure, from indoor air (often in children) or from outdoor air pollution, or in controlled human exposure studies. These studies have observed effects such as increased prevalence of asthma symptoms (Krzyzanowski *et al*., 1990; Delfino *et al*., 2003; Rumchev *et al*., 2002), increased severity of asthma (Venn *et al*., 2003), increased susceptibility to mite allergen exposure in sensitised asthmatics (Casset *et al*., 2006) although in another study no effect of exposure to formaldehyde at 500 µg/m³ on the response of asthmatic subjects challenged with pollen was observed (Ezratty *et al*., 2007) , irritation of the eyes, nose and throat (Ritchie and Lehnen, 1987; Day *et al*., 1984), reduction in peak expiratory flow rate (Krzyzanowski *et al*., 1990), and increases in exhaled nitric oxide which is a marker of airway inflammation (Franklin *et al*., 2000).

There have been fewer studies of occupational exposure specifically to formaldehyde, partly due to the mixed exposures to other irritants (especially wood dust) that usually occur in the occupational setting. A number of studies have evaluated effects in medical students participating in anatomy courses, exposed to formalin dermally and to formaldehyde gas by inhalation, and these have found increases in IgE specific antibodies to formaldehyde and increased prevalence of eye irritation and headaches over the course of their study (Wantke *et al*., 2000), and skin, eye, nose and throat irritation (Takahashi *et al*., 2007). Case-series reviews have reported on numerous cases who have developed allergic contact dermatitis from occupational exposure to

phenol-formaldehyde resins (Owen & Beck, 2001), and to various formaldehyde containing or releasing agents in the workplace (Aalto-Korte *et al*., 2008).

At high concentrations formaldehyde is a respiratory irritant, while it can act as a sensitiser at lower concentrations (Chan Yeung & Malo, 1994). Accidental high level exposure to formaldehyde in a chemical plant producing urea-formaldehyde resins has been reported as the cause of the development of the persistent non-immunological form of asthma known as 'reactive airways dysfunction syndrome' (RADS) (Vandenplas *et al*., 2004). Several case reports have suggested that urea-formaldehyde from insulating material and particle board was the cause of asthmatic/respiratory symptoms (Frigas *et al*., 1981; Cockcroft *et al*., 1982; Kim *et al*., 2001), and that exposure to formaldehyde gas had been the cause of occupational asthma in three subjects who subsequently responded with significant bronchoconstriction when challenged with formaldehyde gas (Lemière *et al*., 1995). Another case report described a 46 year-old truck driver who developed occupational laryngitis caused by chronic exposure to levels of formaldehyde estimated at 0.03 mg/m³ (Roto & Sala, 1996).

***Malignant effects***

Experimental studies have also shown that formaldehyde is genotoxic, with significant increases in DNA cross-links in fibroblasts, keratinocytes and lymphoblasts in human skin cells seen after exposure to formaldehyde (Emri *et al*., 2004). Similar effects have been observed in humans, with a significant increase in DNA-protein cross-links in peripheral white blood lymphocytes observed in 399 workers with occupational exposure to formaldehyde compared with a reference group with no exposure (Shaham *et al*., 2003). When compared with a non-exposed reference group, workers in a pathology anatomy laboratory exposed to formaldehyde at average levels of 0.44 ppm showed significantly increased DNA and chromosomal damage in peripheral blood lymphocytes (Costa *et al*., 2008).

It has long been known that, when inhaled at high concentrations, formaldehyde can induce lesions on the respiratory epithelial surface area in Rhesus monkeys (Monticello *et al*., 1989), and can also induce a high incidence of squamous-cell

carcinoma of the nasal cavity in rats (Swenberg *et al*., 1980). Since then numerous positive findings from cohort and case-control studies of nasopharyngeal cancer in formaldehyde exposed workers have also been reported, and a recent case-control study nested in a cohort of 7,359 workers employed at a plastics-producing plant (Marsh *et al*., 2007) found a statistically significant increase in risk of nasopharyngeal cancer (SMR 4.43, 95% CI 1.78-9.13) and of cancers of the buccal cavity and pharynx (SMR 1.54, 95% CI 1.08-2.13).

Since 1995, formaldehyde had been classified by the International Agency for Research on Cancer (IARC) as a Group 2A (probable) human carcinogen. However, in a recent reclassification, formaldehyde was upgraded to a proven human carcinogen (Group 1) on the basis of sufficient epidemiological evidence in humans and in animals that formaldehyde causes nasopharyngeal cancer in humans (Cogliano *et al*., 2004). Several studies have suggested associations between formaldehyde exposure of industrial workers and cancer of the lung (Coggon *et al*., 2003; Bertazzi *et al*., 1986, 1989), but this association was most strong (SMR 5.79, 95% CI 1.25-5.51) in workers exposed to a combination of formaldehyde and wood dust (Stellman *et al*., 1998).

That IARC Monograph also noted "strong, but insufficient evidence for a causal association between leukaemia and occupational exposure to formaldehyde", and a recent meta-analysis of 15 studies of leukaemia in formaldehyde exposed workers (Zhang *et al*., 2009) found a summary relative risk of 1.54 (95% CI 1.18 – 2.00) with the highest relative risks seen in six studies of myeloid leukaemia (RR=1.90, 95% CI 1.31 – 2.76). As noted above in Volume 100 - A Review of Human Carcinogens IARC has stated that the working group concluded that in addition to the recognised risk of nasopharyngeal cancer there is also now sufficient evidence for leukaemia, and in particular myeloid leukaemia (Baan *et al*, 2009).

### 3.1.3 Exposure Standards

Occupational exposure limits for formaldehyde differ markedly between countries, and between the various governmental and non-governmental agencies that recommend them. However, none currently reflect the recent reclassification to a

Group 1 carcinogen by IARC. For example, even the stringent ACGIH recommendation dates back to 1987, and it was intended to protect against irritation of the upper respiratory tract and eyes. Formaldehyde exposure standards also come in several forms, i.e. either as 8-hour time weighted averages, short term exposure limits (usually 15 or 30 minutes samples), or ceiling limits that are not to be exceeded. In practice, due to the personal sampling methods that are available, compliance with ceiling limits are assessed using 15 minute samples so they are in effect the same as short term exposure limits. The current occupational exposure standards recommended by various agencies and countries are shown in Table 1 below:

**Table 1. Recommended Occupational Exposure Limits for Formaldehyde**

| | OEL (ppm) | | |
|---|---|---|---|
| | 8-Hour TWA | STEL | Ceiling limit |
| Australia | 1.0 | 2.0 | - |
| ACGIH - TLV | - | - | 0.3 |
| OSHA - PEL | 0.75 | 2.0 | - |
| NIOSH - REL | 0.016 | 0.1 | - |
| DFG - MAK | - | - | 0.3 |
| UK - HSE | 2.0 | 2.0 | - |
| New Zealand - WES | - | - | 1.0 |

OEL: Occupational Exposure Limit; TWA: 8 hour Time Weighted Average; STEL: Short Term Exposure Limit, usually 15 minutes; OSHA-PEL: Permissible Exposure Limit established by Occupational Safety and Health Administration in the US; NIOSH-REL: Recommended Exposure Limit established by the US National Institute for Occupational Safety and Health; DFG-MAK: The maximum allowable concentration established by the DFG; UK-HSE: the UK Health and Safety Executive occupational exposure limit; NZ-WES: the New Zealand Workplace Exposure Standard.

### 3.1.4 Exposure surveys

There is a wealth of data available on formaldehyde exposures in the wood conversion industry, with some of the highest continuous exposures measured in the past in particleboard mills (see Table 6; Chapter 4.2) and in embalming establishments (IARC, 2006) with levels often exceeding 1 mg/m$^3$. The average formaldehyde level measured in plywood mills has generally been below 1 mg/m$^3$

(IARC, 2006). However, exposure levels appear to have dropped in more recent years with the introduction of improved glue formulations containing less formaldehyde. A more complete overview of measured levels in specific industry sectors is provided in Chapter 4 where we discuss relevant health and exposure studies in more detail.

## 3.2 Wood dust

**Summary**

- Wood dust is a common occupational exposure, estimated to affect 5.6% of the working population in New Zealand.
- There is sufficient evidence that both hardwood and softwood dust cause respiratory cancers, and all wood dust has been classified by the International Agency for Research on Cancer as a Group 1 (proven human) carcinogen.
- Other health effects known to be caused by exposure to wood dust include non-malignant effects such as asthma, airflow obstruction, and both upper and lower respiratory symptoms.
- These effects have been reported at levels well below the current New Zealand workplace exposure limit of 5 mg/m³.

### 3.2.1 Introduction

Wood is an important renewable resource with at least 1700 million $m^3$ harvested world wide for industrial use each year (IARC, 1995). Wood is composed primarily of cellulose, hemicellulose and lignin, and hundreds of high and low molecular weight organic compounds collectively known as "wood extractives" (ACGIH, 2005). In a living tree these extractives provide protection against attack by bacteria, fungi and moulds, although once a tree is cut down the wood provides a favourable environment for microbial colonisation and proliferation. In New Zealand, most of the plantation forests used in wood processing are gymnosperms or softwoods, with 90% being the single species *Pinus radiata* plus a small percentage of Douglas fir (*Pseudotsuga menziesii*). The main organic extractives present in these softwood species include the monoterpenes α- and β-pinene, limonene and $\Delta^3$-carene, which are readily volatilised during sawing, veneer slicing or chipping, and the diterpene resin acids (predominantly abietic and pimaric acid) which are less volatile and tend to remain in the wood during processing unless heated sufficiently during kiln-, veneer-, chip- or fibre- drying processes (Demers *et al.*, 1997). The wood conversion processes

described in this report all include stages in which the extractives and/or microbiological components may form part of the overall combined exposure. The extractives are therefore included in the following discussion of the health effects of wood dust, while the microbiological exposures and their potential health effects are described in the following chapter.

### 3.2.2 Health effects

The health effects known to be caused by exposure to softwood dust include cancer and non-malignant effects including asthma, non-asthmatic airflow obstruction, and both upper and lower respiratory symptoms. These are discussed below.

***Non-malignant effects***

Wood dust has been found to be associated with a wide range of both acute and chronic respiratory symptoms that have been reviewed and described elsewhere (Enarson & Chan-Yeung, 1990; Demers *et al*., 1997). Effects of exposure to both hardwood and softwood dust have been shown to be associated with upper and lower respiratory symptoms, reduced lung function, increased bronchial responsiveness, occupational asthma, chronic (non-asthmatic) airflow obstruction, and eye and nose irritation. Although considerable research into these effects has been conducted in sawmill workers, these will not be described here since sawmilling is outside the scope of this report. More recent studies, and in particular those relevant to softwoods such as pine will be discussed briefly below.

A recent Swedish case-control study compared patients with idiopathic pulmonary fibrosis (n=140) with a reference group (n=577) from the general population of Sweden (Gustafson *et al*., 2007). The main findings in this study were increased risk of idiopathic pulmonary fibrosis among men exposed to birch dust (OR 2.4, 95% CI 1.18-4.92) and other hardwood dust (OR 2.5, 95% CI 1.06-5.89).

In a recent cross-sectional survey the prevalence of occupational asthma and rhinitis among 591woodworkers in south-eastern Nigeria was investigated (Aguwa *et al*., 2007). Rhinitis and/or asthma symptom prevalence was assessed by questionnaire,

and for each subject the peak expiratory flow rate (PEFR) was assessed. There was a significant increase in prevalence of rhinitis and asthma with increasing length of wood dust exposure. There was also a significant increase in the prevalence of abnormal PEFR with increasing length of wood dust exposure. The OR of having an abnormal PEFR (for those who worked >5 years compared to those who worked for <5 years) was 3.26 (95% CI 2.25 – 4.72). The authors concluded that the prevalence of occupational rhinitis and asthma was high, and that the risk increased with the number of years employed as a woodworker.

A questionnaire based survey of work-related respiratory symptoms in industrial arts teachers (n=130) from the Stockholm public school system, exposed almost daily to pine, found that the arts teachers had a higher frequency of eye, nose, throat, skin, and lower airway symptoms – including chronic bronchitis (OR 12.4, 95% CI 2.95-111) when compared with 112 control subjects (Åhman *et al*., 1995). Nasal irritation was associated with cleaning with a broom (OR 4.71, 95% CI 1.64-13.5), but nasal dripping was decreased when cleaning with a broom (OR 0.29, 95% CI 0.09-0.97).

In a registry-based population study in Finland, a country in which the predominant tree species processed are softwoods, all woodworkers were followed for asthma incidence from 1986 to 1998 (Heikkilä *et al*., 2008). Approximately 158,000 blue-collar workers and about 13,000 administrative workers were employed in the wood processing industry, and the proportion of blue collar workers classified as being exposed to wood dust was 34% in men and 26% in women. The relative risk for asthma was increased for all woodworkers among both genders (Men: RR 1.5, 95% CI 1.2-1.8; and Women: RR 1.5, 95% CI 1.2-1.7). No clear dose-response relationship was found between exposure levels of wood dust and relative risk of asthma, i.e. groups of low and medium exposure in both men (RR 1.4, 95% CI 1.1-1.7; and RR 1.7, 95% CI 1.4-2.2, respectively) and women (RR 1.4, 95% CI 1.2-1.8; and RR 1.6, 95% CI 1.3-2.0, respectively) had significantly increased risk for asthma, but the high exposure group did not (Men: RR 1.2, 95% CI 0.9-1.6; and Women: RR 1.2, 95% CI 0.8-1.6).

***Malignant effects***

Wood dust was classified as a Group 1 carcinogen by IARC in 1995, and this classification was reaffirmed by IARC in 2009 (Straif *et al*, 2009) on the basis of sufficient evidence of cancer in humans. In the IARC Monograph no explicit distinction was made between hardwoods and softwoods, although the evaluation was qualified by reference to the significant excess risk of sino-nasal cancer observed in workers exposed primarily to hardwood dusts (IARC, 1995). The association between exposure to hardwood dust and sinonasal adenocarcinoma is well documented and reviewed elsewhere (IARC, 1995; Demers *et al*., 1995). Since that time additional evidence has accumulated on the proportion of cases attributal to occupational exposures, and of the extent of the increased risk.

A pooled analysis of four European case-control studies of sino-nasal cancer ('t Mannetje *et al*., 1999) confirmed that wood dust was associated with excess risk in men (OR 2.36, 95% CI 1.75-3.20, 168 cases), and that the excess risk was particularly high for adenocarcinoma (OR 12.20, 95% CI 7.43-20.0, 115 cases). In this study, the risk of sino-nasal cancer in the general population attributable to occupational exposures to wood dust was also estimated at 18% of all cases. Further confirmation of the extent of the excess risk of sino-nasal cancer due to occupational exposure to wood dust also comes from a recent Italian case-control study (d'Errico *et al*., 2009) in which a significant increase in risk (OR 58.6, 95% CI 23.74-144.8, 41 cases) with "ever-exposure" to wood dust was observed. A clear dose-response relationship between estimated cumulative exposure (adjusted for co-exposures) and adenocarcinoma was also observed, as is shown in Table 2 below:

**Table 2. Odds ratios for adenocarcinoma with cumulative exposure to wood dust (d'Errico *et al*., 2009)**

| Cumulative exposure (years) | 5 yrs | 10 yrs | 15 yrs | 20 yrs | 25 yrs | 30 yrs | 35 yrs |
|---|---|---|---|---|---|---|---|
| OR (95% CI) | 2.24 (1.76-2.85) | 5.0 (3.09-8.10) | 11.2 (5.42-23.04) | 25.0 (9.53-65.55) | 55.9 (16.74-186.5) | 124.9 (29.4-530.7) | 55.9 (16.74-186.5) |

Most studies reported in the scientific literature, however, have involved exposure to mixed or unspecified wood dust types, although in some instances the predominant species processed in the study region provide clues to the likely type of dust. For example early studies conducted in British Columbia (Elwood, 1981), Norway (Voss *et al*., 1985; Boysen *et al*., 1986), Nordic countries (Hernberg *et al*., 1983) and Western Washington, USA (Vaughan & Davis, 1991) all observed relatively modest, but still statistically significant, excess risks of sino-nasal cancer in workers exposed to wood dust. In these regions the predominant tree species processed were softwoods like spruce and pine. Subsequent studies in France (Leclerc *et al*., 1994) and British Columbia (Teschke, 1994) of workers exposed to softwood dust both failed to detect statistically significant increases in risk, although in both studies there was a suggestion of increased risk with increased exposure intensity or duration. The evidence for the carcinogenicity of softwood dusts is, therefore, still weaker than that for hardwoods although sufficient for all wood dust to be classified as carcinogenic.

Recent *in vitro* studies have used both animal (Määttä *et al*., 2006) and human cell (Bornholdt *et al*., 2007) lines to assess inflammatory responses and genotoxicity of dust from both hardwood and softwood species (including pine and MDF dust). It is interesting to note that these studies have shown that all wood species are capable of inducing increased expression of inflammatory cytokines and chemokines, and of inducing increased DNA strand breaks, and that there was no significant difference in the effects of either hardwood and softwood dust.

The evidence for an association between wood dust exposure and other forms of cancer has also accumulated in recent years, and in some cases the research has differentiated between exposure to hardwoods and softwoods. For example, a cohort of 362,823 men from the American Cancer Society's Cancer Prevention Study – II was followed for six years (Stellman *et al*., 1998). Of those, 45,399 men were either exposed to wood dust, or worked in wood-related industries, or both. This study found a small, but statistically significant increase in all-cause mortality for wood dust-exposed men (RR 1.07, 95% CI 1.03-1.11) and wood-workers (RR 1.17, 95% CI 1.11-1.24), as well as increases in all-cancer mortality in both the wood dust-exposed men (RR 1.08, 95% CI 1.01-1.15) and wood-workers (RR 1.17, 95% CI 1.05-1.30). There was also a small excess risk in lung cancer for wood dust-exposed men (RR

1.17, 95% CI 1.04-1.31), and a significant trend of increased lung cancer risk with increased wood dust exposure (trend P=0.02) in wood workers.

In a more recent case-control study that compared 1,368 lung cancer cases with 1,192 cancer free controls, after adjustment for age, gender, ethnicity, smoking and place of residence, an increased risk of lung cancer (OR 3.15, 95% CI 1.45 – 6.86) was observed for combined wood dust related occupations and industries (Barcenas *et al*., 2005). Lung cancer risk was also associated (OR 1.60, 95% CI 1.19 – 2.14) with an increased level of an overall summary measure of wood dust exposure; among those exposed to cigarette smoke and wood dust there was a cumulative effect resulting in an increase in risk of lung cancer greater than for cigarette smoke or wood dust alone, with 21% of cases being attributed to their interaction.

In a similar case-control study that examined the relationship between wood dust exposure and upper aero-digestive and respiratory cancers (Jayaprakash *et al*., 2008), regular exposure to wood dust was found to be associated with an increased risk of upper aero-digestive and respiratory cancers (OR 1.32, 95% CI 1.01-1.77) and of lung and tracheal cancers (OR 1.69, 95% CI 1.20-2.36). Regular exposure to high levels of wood dust was associated with increased risk of all upper aero-digestive cancers (OR 1.69, 95% CI 1.09-2.64), and regular exposure over a 20 year period was associated with more than double the risk of laryngeal and lung cancers in the high exposure group (OR 2.1, 95% CI 1.31-3.56). Regular exposure to wood dust was also associated with more than twice the risk of squamous cell carcinoma of the nasal cavity, nasopharynx and hypopharynx, with a significant dose-response relationship (OR 2.15, 95% CI 0.98-4.72). In addition, wood dust exposure was associated with adenocarcinoma of the lung (OR 1.85, 95% CI 1.15-2.97), and it was also suggested that smoking and wood dust exposure had a co-carcinogenic effect.

A recent German population-based case-control study, conducted to evaluate the effect of wood dust exposure on the risk of laryngeal cancer, included separate analyses for those exposed to hardwood and softwood dust (Ramroth *et al*., 2008). When exposure history was determined by questions on the use of specific species such as softwoods (fir and spruce etc) and hardwoods (beech and oak etc), excess

risks were observed in those with high exposure to both hardwood dust (OR 2.6, 95% CI 1.3-5.2) and to softwood dust (OR 2.2, 95% CI 1.1-4.2).

### 3.2.3 Exposure standards

As is the case with formaldehyde, there has been much debate about the appropriate levels at which to set exposure standards for wood dust, particularly for dust from the softwoods (Demers *et al*., 1997). Until recently the most common standard internationally has been a TWA of 1 mg/m³ for certain hardwoods (due to the well recognised risk of nasal cancer) and 5 mg/m³ for softwoods such as pine as these were regarded as relatively benign. In some jurisdictions a STEL of 10 mg/m³ is also applied for softwoods. This has changed significantly, however, in the last few years as knowledge of the potential for adverse health effects has increased.

These changes include the reclassification of all wood dust as a Group 1 (or "proven human") carcinogen by IARC in 1994, removing the distinction that had previously been made between hardwoods and softwoods. In addition, numerous studies, including some New Zealand studies (Douwes *et al*., 2001, 2006), have shown a range of non-malignant respiratory effects including asthma and (non-asthma related) airflow obstruction occurring at levels well below 5 mg/m³. In light of this information a number of standard setting agencies have developed revised exposure standards or recommendations.

In 1992 the Dutch Expert Committee on Occupational Standards (DECOS) evaluated the health effects of exposure to wood dust, and recommended an exposure limit of 0.2 mg/m³ total dust (DECOS, 1992). The EU Scientific Committee on Occupational Exposure Limits (SCOEL) has recommended an OEL of 1 mg/$m^3$ inhalable dust. The ACGIH also signalled an intention in 2003 to change their occupational exposure limit for wood dust by removing the distinction between hardwoods and softwoods, and instead distinguishing between allergenic and non-allergenic tree species, with a proposed standard of an 8-hour TWA 0.5 mg/m³ inhalable dust for allergenic species and 1 mg/m³ for non-allergenic species. *Pinus radiata* (the species processed almost exclusively in New Zealand) was identified in a proposed appendix as a commercially important tree species known to induce sensitisation, and would therefore have been

regarded as an allergenic species and have attracted the 8-hour TWA of 0.5 mg/m³. This change has yet to be adopted by the ACGIH, and instead a compromise standard of 1.0 mg/m³ for all species except Western Red Cedar was adopted in the 2005 (and subsequent) versions of their recommended standards (with a notification that the wood dust standard is still under review).

NIOSH has also recommended an exposure limit of a TWA of 1 mg/m³ for up to a 10 hour work shift for all wood types other than Western Red Cedar. The New Zealand Workplace Exposure Standard (WES) remains at 1 mg/m³ for hardwoods such as beech and oak, and 5 mg/m³ (with a STEL of 10 mg/m³) for softwoods. The current Australian standard is the same as New Zealand, while the UK Health and Safety Executive standard is an 8-hour TWA of 5 mg/m³ inhalable dust for both hardwoods and softwoods. The New Zealand Department of Labour has recently proposed amendments to a number of Workplace Exposure Standards, including a reduction of the WES for all wood dust to 1 mg/m³ inhalable dust.

The recommended exposure limits for wood dust in various jurisdictions are listed below in Table 3.

**Table 3. Recommended Occupational Exposure Limits for Wood Dust**

| | | OEL (mg/m³) | | |
|---|---|---|---|---|
| Country/Agency | Tree species | TWA | STEL | Notations |
| ACGIH | Western Red Cedar (WRC) | 0.5 | - | Sensitiser |
| | All other species | 1 | - | Oak and beech A1 carcinogen |
| Australia | Hardwoods | 1 | - | - |
| | Softwoods | 5 | 10 | Sensitiser, standard under review |
| DFG | All species | 1 | - | 3B carcinogen |
| European Union | All species | 1 | - | - |
| The Netherlands | All species | 0.2 | - | - |
| New Zealand | Hardwoods (beech and oak) | 1 | - | Sensitiser, A1 carcinogen |
| | Softwoods | 5 | 10 | Sensitiser |
| NIOSH | All species (except WRC) | 1 | - | Known carcinogen |

### 3.2.4 Exposure surveys

Although levels of exposure have generally declined with the introduction of improved control methods, wood dust remains a common occupational exposure estimated to affect 5.6% of the working population in New Zealand including most of the 14,000 employed in the wood conversion sector (‘t Mannetje *et* al., 2004). The results of a number of recent exposure surveys focussed specifically on wood dust are described below.

Several large surveys of exposure in wood processing occupations have been conducted in other countries. For example, 1,632 TWA wood dust measurements from the US Occupational Safety and Health Administration (OSHA) Integrated Management Information System (IMIS) database from 1979 to 1997 were collected and analysed (Teschke *et al*., 1999). The overall average exposure was 7.93 mg/m$^3$ (AM) or 1.86 mg/m$^3$ (GM) for exposure to all types of wood. Based on univariate analyses, exposure levels decreased over time from GM 4.59 mg/m$^3$ in 1979 to GM 0.14 mg/m$^3$ in 1997. The highest wood dust exposures were found in occupations such as sanders in the transportation equipment industry (GM 17.5 mg/m$^3$), press operators in the wood products industry (GM 12.3 mg/m$^3$), lathe operators in the furniture industry (GM 7.46 mg/m$^3$), and sanders in the wood cabinet industry (GM 5.83 mg/m$^3$).

Occupational exposure to inhalable wood dust has also been reported for 25 member states of the European Union (EU-25) (Kauppinen *et al*., 2006). In 2000-2003, an estimated 3.6 million workers (2.0% of the employed population) was exposed to inhalable wood dust in the EU. Of note is 700,000 workers in the furniture industry and a further 700,000 workers employed as joiners, carpenters and other woodworkers. A total estimate of 563,000 workers (16%) overall were exposed to >5 mg/m$^3$ of inhalable wood dust, with workers in furniture-manufacturing and construction estimated to have the highest levels of exposure. About 897,000 (25%) of all workers were exposed to 2-5 mg/m$^3$ inhalable wood dust, 763,000 (21%) were exposed to 1-2 mg/m$^3$, 597,000 (17%) were exposed to 0.5-1 mg/m$^3$, and 747,000 (21%) were exposed to <0.5 mg/m$^3$.

In a more recent study, data was collected from the SIREP (Italian Information System on Occupational Exposure to Carcinogens) database from 1996 up to 2006 to examine levels of occupational exposure to wood dust in Italy (Scarselli *et al*., 2008). An analysis of variance was performed to determine which factors contributed most to exposure to wood dust. A total of 10,837 exposure measurements were reported, referring to 10,528 workers and 1,181 companies. The overall arithmetic mean was 1.44 mg/m$^3$, the overall geometric mean was 0.97 mg/m$^3$, with a geometric standard deviation of 1.61 and a 95% CI of 0.15-3.80. About 74% of all exposure measurements had a value of <2 mg/m$^3$. For both men and women the most frequently exposed workers were woodworking machine setters and setter-operators (AM 1.59, GM 1.11, GSD 1.70), wood-products machine operators (AM 1.24, GM 0.83, GSD 1.27) and wood and related products assemblers (AM 1.33, GM 0.80, GSD=1.70). The job titles with the highest exposures within these categories were machine-operators that shave wood (AM 2.04, GM 1.43, GSD 2.62) and wood sawyers (AM 1.82, GM 1.37, GSD 1.57). The geometric mean wood dust exposures for men in all wood-related jobs was 0.99 mg/m$^3$ (GSD 1.66) compared to 0.87 mg/m$^3$ (GSD 1.26) in women. Building and repairing of ships and boats generated the highest dust exposures (GM 1.41 mg/m$^3$). The analysis of variance determined that factors such as “company size”, “job category”, “activity sector” and “geographical area” all significantly affected wood dust exposures.

### 3.3 Microbial agents

**Summary**

- Studies show that exposure to microbial agents in the wood conversion industry (excluding sawmills) are elevated, but levels are generally relatively low.
- Exposure to microbial agents are unlikely to play a major role in occupational respiratory disease in wood processing workers

### 3.3.1 Introduction

Microorganisms are small living organisms such as fungi yeasts and bacteria. They are ubiquitous and can be present at very high concentrations in particular occupational (and non-occupational) environments. Microbial agents include agents excreted by microorganisms or fragments or cell wall components of dead microorganisms. Levels of microorganism and microbial agents in the wood conversion industry have been reported to be elevated, but workers' exposure levels are generally (with the exception of sawmill workers) relatively modest. Since the evidence that microbial exposures are involved in respiratory health effects in wood workers is limited we will only briefly review health effects of the main microbial agents measured in the wood conversion industry i.e. fungi, fungal (1→3)-ß-glucan, and bacterial endotoxin.

### 3.3.2 Fungi

Fungi are ubiquitous eukaryotic organisms comprising an abundance of species, many of which can grow on untreated (wet/moist) timber. Most replicate by production of numerous spores that are well adapted to airborne dispersal. Spore sizes range typically from 2 to 10 µm and spores can stay airborne for long periods and may deposit in the respiratory system with some of the smaller spores reaching the alveoli (Eduard, 2006). Many fungal species have been described as producers of Type I allergens, and IgE sensitization to the most common outdoor and indoor fungal species like *Alternaria*, *Penicillium*, *Aspergillus* and *Cladosporium spp*. is strongly associated with allergic respiratory disease, especially asthma. Allergens can be found in spores, hyphae and fungal fragments. At high concentrations fungi may also be involved in Type III and IV allergic reactions including extrinsic allergic alveolitis (or hypersensitivity pneumonitis). As far as we are aware, there is no evidence (apart from the sawmill environment) that these fungal-related health effects occur in the wood processing industry. Milder respiratory health effects due to lower exposure levels can not, however, be excluded.

### 3.3.3 Fungal (1→3)-ß-glucan

(1→3)-β-glucans are *non-allergenic* water-insoluble structural cell wall components of most fungi, some bacteria, most higher plants and many lower plants (Stone & Clarke, 1992). They consist of glucose polymers with variable molecular weight and degree of branching (Williams, 1997). Elevated levels of (1→3)-β-glucans have been demonstrated in several occupational settings including sawmills and joineries (Mandryk *et al*., 1999).

The first reports suggesting a potential role for (1→3)-β-*D*-glucans in the development of respiratory health effects appeared in the late 1980s and early 1990s (Rylander *et al*., 1992); since then, a limited number of population studies have been conducted both in the indoor and occupational environment. These studies reported associations between (1→3)-β-D-glucan exposure and upper airway irritations and fatigue/tiredness (Wan & Li, 1999b; Mandryk *et al*., 2000; Heldal *et al*., 2003; Gladding *et al*., 2003), however, these associations were not confirmed in other studies (Rylander *et al*., 1992; Rylander, 1997; Thorn & Rylander, 1998a). Also, no clear associations with lung function were found i.e. some studies reported adverse effects on lung function (Rylander, 1996; Douwes *et al*., 2000a; Mandryk *et al*., 2000) whereas others found no (Thorn & Rylander., 1998a; 1998b) or even an opposite association (Thorn *et al*., 2001). One study also suggested that (1→3)-β-*D*-glucan was associated with an increased risk of atopy (Thorne & Rylander 1998b), similar to that observed in some animal studies (Wan *et al*., 1999; Ormstad *et al*., 2000; Instanes *et al*., 2004), but this was not confirmed in a smaller study (Rylander *et al*., 1998). Thus, the currently available epidemiological data do not permit conclusions to be drawn regarding the presence (or absence) of an association between environmental glucan exposure and specific adverse health effects (Douwes, 2005). The evidence that (1→3)-β-*D*-glucans play a role in airway symptoms in woodworkers is limited.

### 3.3.4 Bacterial endotoxin

Endotoxins are composed of lipopolysaccharides and lipooligosaccharides and are non-allergenic cell wall components of Gram-negative bacteria with strong pro-

inflammatory properties. Endotoxin is commonly present in many occupational environments (Douwes *et al*., 2002) including furniture factories, joineries, plywood manufacturing and sawmills (Mandryk *et al*., 1999, 2000; Douwes *et al*., 2000b; Fransman *et al*., 2003; Rongo *et al*., 2004; Harper & Andrew, 2006).

Endotoxin has long been recognised as an important factor in the aetiology of occupational lung diseases (Douwes & Heederik, 1997; Douwes *et al*., 2002) and organic dust toxic syndrome (Anonymous, 1998). Subjects exposed to high levels of endotoxin in inhalation experiments experience clinical effects such as fever, shivering, arthralgia, influenza-like symptoms (malaise), blood leukocytosis, neutrophilic airway inflammation, asthma symptoms such as dry cough, dyspnoea and chest tightness, bronchial obstruction, as well as dose-dependent lung function impairment (FVC, $FEV_1$, and flow-volume variables) and decreased lung diffusion capacity (Pernis *et al*., 1961; Castellan *et al*., 1987; Clapp *et al*., 1994; Jagielo *et al*., 1996; Michel *et al*., 1992, 1997a,b). Many occupational studies have shown positive associations between endotoxin exposure and health effects including both reversible (asthma) and chronic airway obstruction, respiratory symptoms (symptoms of asthma, bronchitis and byssinosis) and increased airway responsiveness. Several of these studies reported clear exposure-response relationships (Smid *et al*., 1992; Vogelzang *et al*., 1998). One study in the potato processing industry showed that acute airway obstruction was already apparent at levels of around 50 endotoxin units (EU)/$m^3$ (~ 5ng/$m^3$) (Zock *et al*., 1998). There is only limited evidence that endotoxin plays a role in airway symptoms in woodworkers.

### 3.3.5 Do microbial agents cause health effects in the wood conversion industry?

There are a small number of studies that have assessed microbial exposures in the wood conversion industry most of which were conducted in the sawmill industry (which are excluded from this review). Only a few studies have found a direct association between microbial exposures in wood processing occupations (other than sawmilling) and health effects. This may have several reasons: 1) these associations have not been extensively studied; and 2) exposure levels to micro-organisms and microbial agents in the wood processing industry are generally modest and may therefore not be high enough to cause adverse health effects.

Although levels are relatively low they are generally elevated compared to non-occupational environments, and they have been shown to exceed suggested occupational exposure limits in some cases. For example, several studies (Mandryk *et al*., 1999; Fransman *et al*., 2003; Rongo *et al*., 2004) in plywood manufacturing, furniture making and joineries have measured personal endotoxin levels in excess of 50 EU/$m^3$ – the recommended occupational exposure limit for endotoxin in The Netherlands (Heederik & Douwes, 1997; Dutch Expert Committee on Occupational Standards, 1998). One of these studies (Mandryk *et al*., 1999) also found an association between microbial exposure and respiratory health effects.

Thus, although exposures to microbial agents are elevated in the wood conversion industry, the levels are relatively low and no clear association with respiratory health effects have been established. The authors therefore consider that exposures to microbial agents are unlikely to play a major role in occupational respiratory disease in wood processing workers (apart from sawmill workers who are excluded from this review).

# 4. Exposure and health effect studies by industry sector

In this chapter the studies of both exposures and health effects that have been conducted in the specific wood conversion industry sectors covered in this report will be reviewed.

## 4.1 Plywood industry


**Summary**

- Relatively few studies have assessed exposures or health effects in workers employed in the plywood industry.
- The primary airborne exposures experienced by plywood mill workers are to wood dust and formaldehyde.
- Dermal exposure to formaldehyde occurs mainly in the arms and chest.
- An excess risk of sarcoma has been observed in plywood mill workers.
- Workers in plywood mills who were exposed to phenol-formaldehyde and/or melamine-formaldehyde had work-related allergic dermatitis.
- A New Zealand study showed an increased risk of respiratory symptoms as well as nose, eye and skin symptoms.
- An Indonesian study found no clear association between exposure to wood dust and nasal inflammation or lung function.


### 4.1.1 Introduction

Relatively few studies of exposure and/or health effects have been conducted specifically in plywood mills, and most studies have been unable to differentiate between the effects of the major exposures of wood dust and formaldehyde due to the mixed and highly correlated exposures that occur in the industry. Those that have been reported in the literature have identified significant exposures to wood dust and wood extractives, formaldehyde and microbial agents, as well as respiratory effects including asthma and nasopharyngeal cancer and skin effects.

### 4.1.2 Exposure studies

A study of total exposure (respiratory and dermal) to glue components in a Finnish plywood mill (n = 300) conducted during 1996 and 1997 focused on phenol-formaldehyde (Mäkinen *et al*. 1999). Using stationary sampling, the mean values for respiratory exposure to formaldehyde ranged from 0.03 mg/m$^3$ to 0.31 mg/m$^3$. In the breathing zone of workers, the mean values for respiratory exposure to formaldehyde ranged from 0.05 mg/m$^3$ to 0.30 mg/m$^3$. Dermal exposure measurements (n=15) showed that the main contaminated parts of the body were the arms and chest. Exposure of the chest was measured at 470 µg/h (range from 118 – 1,274 µg/h), and for the lower arms at 328 µg/h (range from 35 – 1,750 µg/h), with the total body exposure estimated at 1,060 µg/h (range from 308 – 2,663 µg/h). Phenol-formaldehyde and/or melamine-formaldehyde were found to have caused allergic dermatitis in 9 (10.2%) workers.

In 2003, the Centre for Public Health Research studied respiratory symptoms and occupational exposures in New Zealand plywood mill workers producing plywood from *Pinus radiata*. The mean exposure level of inhalable dust was 0.7 mg/m$^3$ with a geometric standard deviation (GSD) of 1.9, compared with the New Zealand workplace exposure standard for inspirable dust of 5 mg/m$^3$. The mean exposure level for endotoxin was 23.0 Endotoxin Units (EU)/m$^3$ (GSD 2.8), although 7 of the 20 samples had levels higher than an exposure limit of 50 EU/m$^3$ recommended in The Netherlands (Heederik & Douwes, 1997; DECOS, 1998). The mean (GM) exposure levels for wood extractives included abietic acid 0.7 µg/m$^3$ (GSD 1.8), α-pinene 1.0 mg/m$^3$ (GSD 2.7), β-pinene 1.5 mg/m$^3$ (GSD 2.8) and $\Delta^3$-carene 0.10 mg/m$^3$ (GSD 1.4). The mean (GM) exposure level measured for formaldehyde was 0.08 mg/m$^3$ (GSD 3.0) compared with the New Zealand workplace exposure standard of 1.25 mg/m$^3$ (or 1 ppm).

Another cross sectional study investigated inhalable wood dust exposure levels, respiratory symptoms, lung function, and nasal inflammation in Indonesian workers producing plywood from *Meranti* species logs (Borm *et al*., 2002). The average (GM) exposure levels measured in veneer peeling was 1.52 mg/m$^3$ (GSD 1.83, n=14), in assembly workers 1.70 mg/m$^3$ (GSD 1.16, n = 6), in veneer drier workers 1.71 mg/m$^3$

(GSD 1.57, n= 12), in graders 1.86 mg/$m^3$ (GSD 1.97, n = 10), in veneer preparation 1.90 mg/$m^3$ (GSD 1.47, n=16), in composers 2.84 mg/$m^3$ (GSD 1.36, n=10), and in finishing 3.27 mg/$m^3$ (GSD 2.31, n=15).

The levels of exposure to wood dust and formaldehyde reported in the literature are shown below in Tables 4 (updated from IARC, 1995) and 5 (taken from IARC, 2006) respectively.

**Table 4. Concentrations of wood dust* measured in plywood mills**

| Country and work area | No. of samples | Mean[#] ($mg/m^3$) | Range or (GSD) | Reference |
|---|---|---|---|---|
| **United States - softwood plywood mills** | | | | |
| Edge sawing, sanding, machining | 12 | 1.7[a] | 0.7-3.2 | Whitehead *et al*., 1981 |
| Veneer lathe, clipper, dryer, gluing, pressing | 13 | 0.4 | 0.1-0.7 | " |
| Softwood and veneer and plywood mills | 56 | 0.6[b] | <0.1-6.4 | Clayton Environmental Consultants, 1988 |
| **Finland - softwood plywood mills** | | | | Kauppinen, 1986 |
| Log debarking/cutting | 4 | 0.4 | 0.2-0.7 | " |
| Peeling | 2 | - | 0.2-0.3 | " |
| Sawing of veneers | 3 | 1.6 | 0.6-3.0 | " |
| Sawing of veneers | 4 | 1.3 | 1.1-1.5 | " |
| Sawing of plywood | 6 | 3.3 | 0.5-12.0 | " |
| Sawing of plywood | 11 | 3.7 | 0.3-19.0 | " |
| Sanding of plywood | 5 | 3.0 | 0.3-6.4 | " |
| Sanding of plywood | 21 | 3.8 | 0.8-22.0 | " |
| Chipping in finishing department | 11 | 2.6 | 0.7-7.1 | " |
| Finishing department | 18 | 0.7 | 0.3-2.4 | " |
| Sawing | 24 | 2.1 | 0.4-5.0 | Welling and Kallas, 1991 |
| Sorting, cleaning, glue mixing, hogger | 4 | 11.1 | 7.1-15.0 | " |

| | | | | |
|---|---|---|---|---|
| **Indonesian – plywood mill** | | | | |
| Veneer peeling | 14 | 1.52 [b] | (1.83) | Borm *et al*., 2002 |
| Assembly | 6 | 1.7 [b] | (1.16) | " |
| Veneer dryers | 12 | 1.71 [b] | (1.57) | " |
| Graders | 10 | 1.86 [b] | (1.97) | " |
| Veneer preparation | 16 | 1.90 [b] | (1.47) | " |
| Composers | 10 | 2.84 [b] | (1.36) | " |
| Finishing | 15 | 3.27 [b] | (2.31) | " |
| **New Zealand - plywood mill** | | | | |
| Green end | 7 | 0.8 [b] | (2.7) | Fransman *et al*., 2003 |
| Dryers | 20 | 0.6 [b] | (1.7) | " |
| Composers | 6 | 1.6 [b] | (2.1) | " |
| Pressing | 9 | 0.6 [b] | (1.4) | " |
| Finishing department | 12 | 0.8 [b] | (1.9) | " |

* Dust fractions were not given in IARC report; [#]Arithmetic mean unless otherwise specified, [a] Area samples, [b] Geometric mean.

**Table 5. Concentrations of formaldehyde in plywood mills**

| Country and work area | No. of samples | Mean* ($mg/m^3$) | Range (GSD) | Reference |
|---|---|---|---|---|
| **Sweden** | 47 | 0.4 | - | Rosén *et al*., 1984 |
| **Finland** | | | | Kauppinen 1986 |
| Glue preparation, short term | 15 | 2.7 | 0.7-6.2 | |
| Glue preparation, short term | 19 | 0.9 | 0.1-2.8 | |
| Assembling | 32 | 1.9 | <0.1-5.4 | |
| Assembling | 55 | 0.7 | 0.03-8.3 | |
| Hot pressing | 41 | 2.5 | <0.1-9.5 | |
| Hot pressing | 43 | 0.6 | 0.07-2.6 | |
| Sawing plywood | 5 | 0.6 | 0.4-1.0 | |
| Sawing plywood | 12 | 0.1 | 0.03-0.3 | |
| Coating plywood | 7 | 1.2 | 0.6-2.2 | |
| Coating plywood | 28 | 0.4 | 0.03-0.7 | |
| **USA** | | | | Stewart *et al.,* 1987 |
| Plywood manufacture | 27 | 0.3 [a] | 0.1-0.5 | |
| Plywood manufacture | 26 | 0.1 [a] | 0.01-0.6 | |

| | | | | |
|---|---|---|---|---|
| **Indonesia** | | | | Malaka and Kodama, 1990 |
| Plywood mill | 40 | 0.8 | 0.3-2.8 | |
| **Finland** | | | | Mäkinen *et al*., 1999 |
| Patching | 6 | 0.07 | 0.03-0.10 | |
| Dryer infeed | 6 | 0.06 | 0.01-0.15 | |
| Forklift driving | 6 | 0.07 | 0.02-0.20 | |
| Assembly | 4 | 0.30 | 0.10-0.81 | |
| Assembly | 6 | 0.15 | 0.10-0.27 | |
| Hot pressing | 5 | 0.13 | 0.08-0.23 | |
| Glue preparation | 2 | 0.15 | 0.07-0.23 | |
| Finishing (puttying) | 4 | 0.09 | 0.07-0.14 | |
| Finishing (sanding) | 2 | 0.05 | 0.01-0.07 | |
| **New Zealand** | | | | Fransman *et al*., 2003 |
| Dryers | 14 | 0.07 [a] | (3.2) | |
| Composers | 2 | 0.03 [a] | (1.0) | |
| Pressing | 5 | 0.16 [a] | (2.7) | |
| Finishing end | 1 | 0.04 [a] | - | |

* Arithmetic mean unless otherwise specified

[a] Geometric mean

### 4.1.3 Health effects

Only three health effects studies in plywood mill workers were identified. In a US population-based case-control study that included 251 sarcoma cases and 1,908 general population controls, occupational risk factors for five sarcoma subtypes were evaluated in men born from 1929 to 1953 who were first diagnosed with sarcoma between December 1984 and November 1988 (Hoppin *et al*., 1999). Wood dust and plywood were found to be associated with leiomyosarcoma (OR 2.21, 95% CI 1.25 – 3.86).

The New Zealand cross-sectional study (Fransman *et al*., 2003) reported significantly more "attacks of shortness of breath with wheezing in the last 12 months" in plywood mill workers (n=112) than in the general population (n=415), with an OR of 1.8 (95% CI 1.0 – 3.1). Other asthma symptoms were also increased, when compared to the general population, but these did not reach statistical significance. Workers also experienced more nose, eye and skin symptoms. By contrast the Indonesian cross-sectional study (Borm *et al*., 2002) did not show a clear association between exposure to wood dust and nasal inflammation or lung function.

## 4.2 Particleboard and oriented strandboard industry

**Summary**

- There have been a number of studies of formaldehyde exposures in the particle board industry.
- Relatively few wood dust exposure measurements have been reported.
- Only one study of microbiological contamination in particleboard manufacture has been reported.
- The majority of formaldehyde measurements in oriented strandboard plants are below current threshold limit values.
- Formaldehyde levels in excess of current threshold limit values occur more frequently in particleboard manufacture.

- Wood dust levels measured in both types of processes are within current threshold limit values.
- Relatively few studies of health effects have been reported.
- Those studies that have been reported show some evidence of respiratory symptoms, lung function deficit and dermatitis, however, the evidence is mixed and more studies are therefore needed.
- Skin effects are believed to be of a non-allergic nature.

### 4.2.1 Introduction

Considerably more exposure studies conducted in PB and OSB plants have been reported in the literature than for all other wood conversion industries (apart from the pulp and paper and sawmill industries), although only five health effects studies have been reported.

### 4.2.2 Exposure studies

Formaldehyde exposure levels measured in Finnish PB manufacturers, using area sampling methods, ranged from 0.4 to 2.3 ppm depending on location in the plant (Kauppinen & Niemelä, 1985). The Occupational Safety and Health Administration conducted personal sampling of formaldehyde in three PB manufacturing plants in the United States, and reported average (GM) levels for different jobs ranging from 0.10 to 0.32 ppm (Zimowski, 1986). The 1995 IARC monograph on formaldehyde cited the results of a Swedish study which found average (AM) levels of 0.3 ppm in PB manufacturing plants (IARC, 1995).

A number of studies have been conducted primarily to assess the effect of formaldehyde (and in some cases wood dust) exposure on mucous membranes and the respiratory system, and these studies have included exposure measurements. For example, in a study of histopathological changes in the nasal mucosa in men with occupational exposure to formaldehyde and wood dust in two Swedish PB plants (Edling *et al*., 1988) exposure data was reported from measurements made by

company industrial hygienists on different occasions between 1975 and 1983. The time weighted average (TWA) levels of formaldehyde in air were reported to range from 0.1 to 1.1 mg/m³, with peaks of up to 5 mg/m³, and the concentrations of wood dust were about 0.6-1.1 mg/m³. Formaldehyde measurements (n=15) taken in a US OSB plant that used phenol formaldehyde resin were reported to all be below 0.05 ppm (Imbus & Tochilin, 1988). Area and personal formaldehyde measurements taken in a PB manufacturer in the US were reported to have a median value of 0.62 ppm (Horvath *et al*., 1988).

In a similar study in a Canadian OSB plant (Herbert *et al*., 1995), 10 area samples of formaldehyde were all below 0.2 ppm. Formaldehyde and wood dust exposure and their effect of on pulmonary function were studied in 35 workers in a Turkish PB plant (Erdem *et al*., 1996). Airborne concentrations of formaldehyde and wood dust were reported to be 1.70 ± 0.24 ppm and 0.41 ± 0.02 mg/m$^3$ respectively, but neither formaldehyde nor wood dust exposure were found to be associated with lung function.

A more recent exposure survey conducted in Quebec, Canada included 100 personal samples and 103 area samples of formaldehyde in three PB and six OSB plants (Lavoué *et al*., 2005). In the OSB plants the personal samples (n=55) in different jobs ranged from an average (GM) of 0.03 to 0.06 ppm with GSDs ranging from 1.3 to 1.7, and the area samples (n=52) ranged from GM 0.02 to 0.09 ppm with GSDs of 1.3 to 2.5. In the PB plants the personal samples (n=45) ranged from GM 0.08 to 0.23 ppm with GSDs of 1.3 to 2.3, and the area samples (n=51) ranged from GM 0.08 to 0.75 ppm with GSDs of 2.0 to 3.9.

As certain industries in Quebec are classified as regulated, they are regularly monitored by government occupational health teams who conduct exposure measurements as well as surveys of health outcomes. The results of these exposure measurements dating back to 1984 have been recently compiled and reported (Lavoué *et al*., 2005). The sampling strategies used for these government measurements were not recorded, but the formaldehyde levels measured and the variability as expressed by the GSD were in all cases considerably higher than those measured by the research teams. In 48 samples in OSB plants the GM ranged from 0.04 to 0.54 mg/m³ with

GSDs ranging from 1.1 to 2.9, and in 104 samples in PB plants the GM ranged from 0.17 to 0.45 mg/m³ with GSDs ranging from 1.4 to 3.0. The authors considered the consistently higher government measurements were most likely to be due to a "worst case" sampling bias in compliance monitoring programmes.

Air sampling for microbiological contamination was performed in a fibreboard factory and two chipboard factories in south-eastern Poland (Dutkiewicz *et al*., 2001). They found that the concentration of microorganisms in the fibreboard factory was high at the initial stages of production, which involved shredding waste wood and storing chips (ranging from 71.8 – 95.2 × $10^3$ colony forming units (CFU)/$m^3$), with lower concentrations in the later stages of forming and formatting of fibreboards (8.4 – 17.5 × $10^3$ CFU/$m^3$). The concentration of microorganisms in the chipboard factories were lower compared to the fibreboard factory (5.3 – 34.8 × $10^3$ cfu/$m^3$) except for one site which had a high concentration of airborne microorganisms at 101.5 x $10^3$ cfu/$m^3$.

*Penicillium* species were found growing on waste wood by chippers (46.2 – 75.2% of total fungal strains) and at tanks. *Aspergillus fumigatus* (79.7% of total fungal isolates) was found on stored chips. *Penicillium* spp. and *Aspergillus fumigatus* possess immunotoxic and allergenic properties and are associated with occupational respiratory disease (Lacey *et al*., 1988; Lacey & Dutkiewicz, 1994). *Aspergillus fumigatus* may cause allergic alveolitis, asthma, pulmonary aspergillosis, and possibly mycotoxicoses (Di Salvo, 1983; Krysińska-Traczyk, 1973; Land *et al*., 1987).

The results of formaldehyde and wood dust exposure measurement surveys in PB and OSB plants are summarised in Table 6 below.

**Table 6. Levels of formaldehyde and wood dust reported in studies of particle board and oriented strandboard manufacturing plants**

| | Formaldehyde | | Wood dust | | |
|---|---|---|---|---|---|
| | GM or range ($mg/m^3$) | GSD (or range) | GM or range ($mg/m^3$) | Sampling method | Reference |
| **Particleboard** | | | | | |
| Sweden | 0.4 (AM) | - | | | Rosén *et al*., 1984 |
| Finland | 0.5 – 2.9 | - | - | Area | Kauppinen and Niemelä, 1985 |
| Glue preparation | 2.7 | (0.4-6.0) | | | |
| Blending | 1.2 | (0.1-2.5) | | | |
| Blending | 0.9 | (<0.1-1.7) | | | |
| Forming | 2.1 | (<0.6-5.7) | | | |
| Forming | 1.7 | (0.1-5.9) | | | |
| Hot pressing | 4.2 | (1.4-11.7) | | | |
| Hot pressing | 2.1 | (0.25-5.7) | | | |
| Sawing | 5.9 | (0.9-11.3) | | | |
| sawing | 1.2 | (<0.1-4.1) | | | |
| US | 0.1 – 0.4 | - | - | Personal | Zimowski, 1986 |
| Sweden | 0.4 (AM) | - | - | - | IARC, 1995 |
| Sweden | 0.15 – 1.6 | (peak 7.7 $mg/m^3$) | 0.60 – 1.10 | - | Edling *et al*., 1988 |
| US | 0.7 | - | - | - | Horvath *et al*., 1988 |
| Indonesia | 3.0 | (1.5-4.3) | | | Malaka and Kodama, 1990 |
| Turkey | 2.1 | (± 0.3 $mg/m^3$) | 0.41 ± 0.02 | - | Erdem *et al*., 1996 |
| Quebec | 0.1 – 0.3 | 1.6 – 2.8 | - | Personal – research | Lavoué *et al*., 2005 |
| Quebec | 0.1 – 1.0 | 2.5 – 4.9 | - | Area – research | Lavoué *et al*., 2005 |
| Quebec | 0.2 – 0.6 | 1.7 – 3.8 | - | Personal – compliance | Lavoué *et al*., 2005 |
| Quebec | 0.04 – 2.5 | 1.9 – 5.0 | - | Area – compliance | Lavoué *et al*., 2005 |

| | | | | | |
|---|---|---|---|---|---|
| **Oriented strandboard** | | | | | |
| US | < 0.06 | - | - | - | Imbus and Tochilin, 1988 |
| Canada | ≤ 0.3 | - | 0.27 | - | Herbert *et al*., 1994 |
| Canada | ≤ 0.06 | - | 0.05 – 0.50 ® | - | Herbert *et al*., 1995 |
| Quebec | 0.04 – 0.07 | 1.6 – 2.1 | - | Personal – research | Lavoué *et al*., 2005 |
| Quebec | 0.03 – 0.12 | 1.6 – 3.0 | - | Area – research | Lavoué *et al*., 2005 |
| Quebec | 0.04 – 0.7 | 1.4 – 3.6 | - | Personal – compliance | Lavoué *et al*., 2005 |
| Quebec | 0.01 – 0.17 | 1.3 – 3.9 | - | Area – compliance | Lavoué *et al*., 2005 |

GM, geometric mean; GSD, geometric standard deviation; AM, arithmetic mean; ®, respirable fraction of dust

### 4.2.3 Health effects

Only five studies of health effects in PB or OSB workers have been reported, with four evaluating respiratory health and one evaluating dermatitis.

A cross-sectional study evaluated respiratory health in 176 workers from two OSB plants in the US (Imbus & Tochilin, 1988). Each participant filled in a respiratory questionnaire and underwent spirometry before and during their work shift, and the authors reported no evidence of an acute effect on pulmonary function. A similar cross-sectional study used respiratory questionnaires and spirometry to assess respiratory health among workers in a PB plant in Turkey (Erdem *et al*., 1996). This study found insignificant cross-shift changes in spirometric values and found no clear association between exposed and non-exposed workers, or any association with duration of exposure.

In another cross-sectional study in a Canadian OSB plant, 127 workers completed respiratory questionnaires, and underwent spirometry and skin prick tests against common allergens (Herbert *et al*., 1994, 1995). An unexposed comparison group of 165 oil workers from the same region was also tested. The ratio of $FEV_1$/FVC was significantly reduced in the OSB plant workers when compared to the oil field workers. OSB plant workers who were ex-smokers and current smokers were also three times as likely as current or ex smoking oil field workers to have an $FEV_1$/FVC ratio of <75%. Significant cross shift reductions in $FEV_1$ ($p=0.04$) and FVC ($p=0.02$) were observed, and a number of respiratory symptoms of airway reactivity were also significantly more common, in the OSB plant workers.

A study on dermatitis in a Canadian PB manufacturing facility surveyed approximately 250 people, conducting a cutaneous examination and patch testing and using a questionnaire assessing symptoms (including asthma, allergies, rashes, willingness to seek medical consultation and undergo patch testing), past and family medical history, workplace exposures and the use of protective equipment (Saary *et al*., 2001). Skin rashes were reported by 52% of workers, with an average duration of 3 years, and 53% of those workers reported that symptoms were worse at work. Eye symptoms were reported by 48% of workers, with an average duration of three years,

and 70% of those workers reported that symptoms were worse at work. Nasal symptoms were reported by 24% of workers, with an average duration of nine years, and 60% of those workers reported that symptoms were worse at work. Cough symptoms were reported by 19% of workers, with an average duration of seven years, with 75% of those workers reporting that symptoms were worse at work. Dust was a factor employees believed was associated with each of the symptoms. Although 52% reported rash, upon clinical examination, only 19% had their dermatitis confirmed.

The authors considered that many of the symptoms were due to irritant rather than allergic effects, as the skin symptoms experienced were heterogeneous and not associated with any particular job title or job location, and those with self-reported rashes were more likely to have dry skin in the past year. Upon examination they had dry hands and those with symptoms were more likely to wear gloves, which support irritation rather than an allergic reaction. Five employees (24%) had cracking of the skin on very dry hands which suggested chronic irritation. Patch testing showed 4 workers (19%) to be skin-allergic for quaternium-15, a substance used to purify water-soluble coolants and found in cutting fluids, although quaternium-15 was not used at the plant during the time of the study. There were other allergens which produced positive responses in patch tests, but none of the volatile organic compounds (VOCs) typically found in wood products such as "$\alpha$ and $\beta$ pinene, ethyl benzene, methylene chloride, and xylene" induced a positive response. Therefore, it was concluded that dermatitis was most likely due to irritant exposures, particularly from wood dust, rather than exposures to allergens.

## 4.3 Medium density fibreboard

**Summary**

- The literature contains only limited information on levels of exposure or health effects in MDF production.
- Several studies report levels of dust and formaldehyde generated during the subsequent processing of MDF.
- High levels of wood dust are generated in the absence of extraction ventilation, but these are reduced markedly with extraction ventilation.
- The evidence regarding differences in dust levels produced when processing MDF or solid timber is conflicting.

### 4.3.1 Introduction

Very few studies of exposures and/or health effects of workers in the medium density fibreboard (MDF) production industry were found, with most studies focusing on the subsequent use of MDF by joiners and furniture makers.

### 4.3.2 Exposure and health effects studies

Formaldehyde levels were measured in MDF mills in Sweden in the 1980s (Rosén *et al*., 1984), with an average (AM) of 0.3 mg/m³ (n=19). No range of values or SD was reported.

A study in Finland investigated workers' exposure to MDF dust, wood dust and formaldehyde and potential inflammatory nasal responses and symptoms (Priha *et al*., 2004). The study included 22 workers in the MDF group, 23 workers in the wood dust group and 15 unexposed controls. The average (GM) level of inhalable dust measured was 1.2 mg/m$^3$, while the highest exposures to MDF dust occurred among workers involved in sanding (2.1 mg/m$^3$) or cutting boards (1.6 mg/m$^3$). Average (GM) formaldehyde concentrations were 0.17 mg/m$^3$. Nasal irritation was reported by 36% (8/22) of the workers, eye and throat irritation by 5% (1/22), and skin irritation by

14% (3/22). Formaldehyde levels of the workers with nasal symptoms were lower than those of the workers without symptoms (GM 0.12 vs. 0.19 mg/$m^3$) suggesting that formaldehyde may not be the main cause of the symptoms. Eosinophil levels in the nasal cavities in the MDF group were increased suggesting a Type I allergic reaction.

In the United Kingdom it was shown that levels of inhalable dust were higher when sanding MDF compared with solid timbers (hardwood and softwood), but showed little difference between MDF and solid timber when sawing (Chung *et al*., 2000). Formaldehyde levels ranged from 0.01 to 0.17 mg/$m^3$ in this study. A pilot study of inhalable and respirable wood dust production generated from MDF and softwoods in a cabinet-making shop (Hursthouse *et al*., 2004), found exposure levels of inhalable dust ranging from 6.9 to 91 mg/$m^3$ from circular sawing and hand sanding of MDF compared with 2.5 to 45 mg/$m^3$ when processing softwood solid timbers. The level of exposure to the respirable fraction of the dust was also significantly higher when processing MDF compared to softwoods.

Two recent Australian reports have also compared the levels of dust and formaldehyde generated when processing MDF and solid timbers, and when using local exhaust ventilation or relying on natural ventilation (Golec, 2006; ASCC, 2008). Golec reported formaldehyde levels ranging from <0.01 to 0.6 ppm during routing, panel sawing, circular sawing and belt sanding of MDF, with little variation on emission levels with ventilation on or off. Inhalable wood dust levels differed markedly for the different processes when extraction was on or off, as shown in Table 7. Inhalable MDF (and PB) dust generated during cutting and routing operations were similar or lower than that produced by solid timbers with local extraction ventilation on. However, routing of MDF (and PB) without the use of local extraction ventilation produced significantly higher dust levels than solid woods.

**Table 7. Influence of extraction ventilation on dust exposures when processing MDF**

| Process | Inhalable dust (mg/m³) | |
|---|---|---|
| | Ventilation on | Ventilation off |
| Routing | <0.1 | 14 |
| Panel sawing | 0.2 | 1.9 |
| Circular sawing | 2.2 | 21 |
| Belt sanding | 1.1 | 113 |

## 4.4 Pulp and paper industry


**Summary**

- Of all the wood conversion industries, most research has been conducted on the pulp and paper industry.
- Pulp and paper production workers have been exposed to a complex mixture of hazardous substances, including many known or suspected carcinogens.
- The patterns of exposure in the industry have changed over time due to the range of processes used.
- Exposure to several irritant agents in pulp and paper mill workers has been shown to cause a range of non-malignant respiratory symptoms.
- Epidemiological studies have suggested increased mortality from nervous, gastro-intestinal and respiratory system cancers, and certain lymphatic and haematopoietic neoplasms, as well as from cardiovascular disease.
- Among specific exposures examined, sulphur dioxide and asbestos have been associated with respiratory system cancers, and volatile organochlorine compounds with an increase in all-cancer mortality.


### 4.4.1 Introduction

The wood conversion industry sector with the most information on exposures and health effects available in the scientific literature is the pulp and paper industry, due largely to the multi-centre collaborative study conducted by the International Agency

for Research on Cancer. The complex mix of exposures in the industry, which have changed over time due to the introduction of different technologies, has been very well characterised and numerous cohort and case-control studies have been conducted.

### 4.4.2 Exposure studies

Pulp and paper production workers are exposed to a complex mixture of hazardous substances, including known or suspected carcinogens such as wood dust, various wood extracts and associated bioaerosols, reduced sulphur compounds, talc, formaldehyde, combustion products, epichlorohydrin, acid mists, auramine and other benzidine-based dyes, and a range of chlorinated organic compounds. The patterns of exposure in the industry, however, are complicated because of the range of different processes that have been used over time in the various stages of pulp and paper manufacture (McLean *et al*., 2006).

Exposures in this industry have been well characterised through the development of a Pulp and Paper Department Exposure Matrix (PAPDEM) for an international collaborative cohort study of mortality and cancer incidence in 15 countries coordinated by the International Agency for Research on Cancer (Kauppinen *et al*., 1997). PAPDEM incorporated over 31,000 previously unpublished measurements of 246 different chemical agents, characterised by country, mill type, department and job, and duration and date of sampling. Most (>80%) of the measurements available were from the 1980s and early 1990s, and for 33 of the 246 agents sampled the relevant threshold limit value was exceeded more than 10% of the time.

The list of the agents measured in the IARC exposure measurement database with the proportion of measurements exceeding the relevant threshold limit value (adapted from Kauppinen *et al*., 1997) is shown in Table 8 below, with relevant New Zealand Workplace Exposure Standards and 2009 ACGIH Threshold Limit Values, to indicate the range of exposures identified in this industry.

## Table 8. Chemical agents included in the IARC PAPDEM

| Agent | % > TLV | 2009 ACGIH TLV (In ppm unless stated otherwise) | NZ WES |
|---|---|---|---|
| **Inorganic gases** | | | |
| Sulphur dioxide | 38 | 0.25 (STEL) | 2 ppm |
| Hydrogen sulphide | 2 | 10 (NIC) | 10 |
| Chlorine | 2 | 0.5 | 0.5 |
| Carbon monoxide | 13 | 25 | 3.5% of haemoglobin in blood |
| Chlorine dioxide | 19 | 0.1 | 0.1 |
| Ammonia | 15 | 25 | 25 |
| Nitrogen dioxide | 26 | 3 | 3 |
| Carbon dioxide | 0 | 5000 | 5000 |
| Ozone | 25 | Heavy work 0.05<br>Moderate work 0.08<br>Light work 0.1<br>< 2 hours 0.20 | 0.1 (C) |
| Sulphur oxides, calculated as SO2 | 0 | 0.25 (STEL) | 2 |
| Nitrogen oxide | 0 | 50 | 25 |
| Hydrochloric acid | 28 | 2 (C) | 5 (C) |
| **Mineral and unspecified dusts** | | | |
| Asbestos | 5 | 0.1 f/cc – all forms | 6 f/ml – chrysotile<br>0.1 f/ml - amphiboles |
| Silica | 3 | 0.025 mg/m³ | 0.1 mg/m³ - cristobalite<br>0.2 mg/m³ - quartz |
| Talc | 6 | 2 mg/m³ | 2 mg/m³ |
| Kaolin | 7 | 2 mg/m³ - respirable | 10 mg/m³ - inhalable<br>2 mg/m³ - respirable |
| Coal | 0 | 0.4 mg/m³ anthracite<br>0.9 mg/m³ bituminous | 3 mg/m³ - respirable |
| **Metals and their compounds** | | | |
| Lead | 14 | 0.05 mg/m³ | 0.1 mg/m³ |
| Welding fumes | 27 | 5 mg/m³ | 5 mg/m³ |
| Calcium oxide | 42 | 2 mg/m³ | 2 mg/m³ |
| Nickel | 2 | Elemental 1.5 mg/m³<br>Soluble inorganic 0.1 mg/m³<br>Insoluble inorganic 0.2 mg/m³<br>Nickel subsulfide 0.1 mg/m³ | 1 mg/m³ - metal<br>0.1 mg/m³ - soluble |
| Chromium | 0 | Metal & CrIII cpds 0.5 mg/m³<br>Water-soluble Cr VI cpds 0.05 mg/m³<br>Insoluble Cr VI cpds 0.01 mg/m³ | 0.5 mg/m³ - metal<br>0.05 mg/m³ - Cr(VI) |
| Iron | - | 5 mg/m³ | 5 mg/m³ |
| Manganese | 5 | 0.2 mg/m³ | 1 mg/m³ |
| Mercury | 5 | Alkyl cpds 0.01 mg/m³<br>Aryl cpds 0.1 mg/m³<br>Elemental and inorganic 0.025 mg/m³ | 0.025 mg/m³ - inorganic<br>0.01 mg/m³ - alkyl cpds |
| Sodium hydroxide | 8 | 2 mg/m³ (C) | 2 mg/m³ (C) |
| Copper | 20 | 0.2 mg/m³ (fume) | 0.2 mg/m³ (fume) |
| Cobalt | 5 | 0.02 mg/m³ | 0.05 mg/m³ |
| Iron oxide fumes | 0 | 5 mg/m³ | 5 mg/m³ |
| Zinc oxide | 0 | 2 mg/m³ | 5 mg/m³ - fume |
| Aluminium | 0 | 1 mg/m³ | 10 mg/m³ (dust) |
| Cadmium | 0 | 0.01 mg/m³ (inspirable)<br>0.002 mg/m³ (respirable) | 0.01 mg/m³ (inspirable)<br>0.002 mg/m³ (respirable) |
| Titanium dioxide | 20 | 10 mg/m³ | 10 mg/m³ |
| **Organic solvents** | | | |
| Toluene | 6 | 20 | 50 |
| Turpentine | 3 | 20 | 100 |
| Methanol | 11 | 200 | 200 |
| Phenol | 2 | 5 | 5 |
| Isopropanol | 1 | 200 | 400 |
| Styrene | 8 | 20 | 50 |
| Ethyl acetate | 2 | 400 | 200 |
| Ethanol | 5 | 1000 (STEL) | 1000 |
| Acetone | 4 | 500 | 500 |
| Methyl ethyl ketone | 6 | 200 | 150 |

| | | | |
|---|---|---|---|
| Butanol | 8 | 20 | 50 (C) |
| Chloroform | 9 | 10 | 2 |
| Terpenes | - | - | - |
| Carbon disulphide | 14 | 1 | 10 |
| Xylene | 0 | 100 | 50 |
| Pinene, alpha- | | - | - |
| Butyl acetate | 0 | 150 | 150 |
| Pinene, beta- | | - | - |
| Vinyl acetate | 5 | 10 | 10 |
| Perchloroethylene, | 30 | 25 | 50 |
| Tetrachloroethylene | | 25 | 50 |
| White spirits | - | 100 | 100 |
| Isopropyl benzene | - | 50 | 25 |
| Methyl isobutyl ketone | 0 | 50 | 50 |
| Ethoxyethanol, 2- | 80 | 2 | 5 |
| Propylene glycol monomethyl ether | 17 | 100 | 100 |
| Gasoline | 0 | 300 | 300 |
| Methylene chloride | 12 | 50 | 50 |
| Trimethyl benzene | 4 | 25 | 25 |
| Ethyl benzene | 0 | 100 (NIC to 50) | 100 |
| Hexane, *n*- | 4 | 50 | 20 |
| Butoxyethanol, 2- | 0 | 20 | 25 |
| Butyl acrylate | 0 | 2 | 10 |
| Propanol, *n*- | 0 | 100 | 200 |
| Cyclohexane | 0 | 100 | 100 |
| Ethoxyethyl acetate, 2- | 0 | 5 | 5 |
| Trichloroethylene | 26 | 10 | 50 |
| Isopropyl acetate | 0 | 100 | 250 |
| Butylglycol acetate | - | 20 | - |
| Methyl acrylate | 0 | 2 | 10 |
| Heptane, *n*- | 0 | 400 | 400 |
| Methyl methacrylate | 0 | 50 | 50 |
| Methyl acetate | 0 | 200 | 200 |
| Octane | 0 | 300 | 300 |
| Ethyl acrylate | 0 | 5 | 5 (C) |
| Methyl cyclohexane | 0 | 400 | 400 |
| Cyclohexanol | 0 | 50 | 50 |
| **Other organic compounds** | | | |
| Dimethylsulphide | - | 10 | - |
| Formaldehyde | 40 | 0.3 (C) | 0.3 (C) |
| Methyl mercaptan | 9 | 0.5 | 0.5 |
| Diphenyl | 42 | 0.2 | 0.2 |
| Furfuryl aldehyde | 11 | 2 | 2 |
| Ethyl mercaptan | 1 | 0.5 | 0.5 |
| Paper dust | - | - | - |
| Diethylsulphide | - | - | - |
| Wood dust | 23 | 1 mg/m³ | 5 mg/m³ (softwoods)<br>1 mg/m³ (hardwoods) |
| Polychlorinated biphenyls | 2 | 0.5 mg/m³ | 0.1 mg/m³ |
| Benzo(a)pyrene | - | (L) | - |
| Acetic acid | 3 | 10 | 10 |
| Carene, delta- | - | - | - |
| Epichlorohydrin | 7 | 0.5 | 0.5 |
| Formic acid | 0 | 5 | 5 |
| Hydrazine | 49 | 0.01 | 0.01 |
| Acrolein | 40 | 0.1 (C) | 0.1 |
| Paperboard dust | - | - | - |
| Pulp dust | - | - | - |
| Oil mist | 0 | 5 mg/m³ | 5 mg/m³ |
| Diphenylmethane-diisocyanate(MDI) | 22 | 0.005 | 0.02 mg/m³ |
| Flax dust | - | - | - |
| Mercaptans | - | 0.5 | - |
| Toluenediisocyanate (TDI) | 0 | 0.005 | 0.02 mg/m³ |
| Acrylonitrile | 0 | 2 | 2 |
| *N*-Methyl-2-pyrrolidine (vapour) | - | - | 25 |
| Naphthalene | 14 | 10 | 10 |
| Isobutyl methacrylate | - | - | - |

| | | | |
|---|---|---|---|
| Dimethyltrisulphide | - | - | - |
| Acrylamide | 9 | 0.03 mg/m³ | 0.03 mg/m³ |
| Fluorine | - | 0.03 mg/m³ | 1.6 mg/m³ |
| Trimellitic anhydride | 50 | 0.0005 mg/m³ | 0.039 mg/m³ |
| **Bioaerosols** | | | |
| Fungal spores | - | - | - |
| Bacteria | - | - | - |
| Endotoxins | - | - | - |
| **Other exposures** | | | |
| Dyes | - | - | - |
| Chlorides | - | - | - |
| Sulphuric acid | 13 | 0.2 mg/m³ | 1 mg/m³ |
| Sulphur compounds | - | - | - |
| Hydrogen peroxide | 0 | 1 | 1 |
| Antimony | 0 | 0.5 mg/m³ | 0.5 mg/m³ |

### 4.4.3 Health effects

As in most industrial cohorts, epidemiological studies of pulp and paper workers have found lower overall mortality and all-cancer mortality due to the well known phenomenon of the "healthy worker effect', but a number of studies have suggested increased risks of specific causes of mortality including cardiovascular diseases (Langseth & Kjærheim, 2006; Persson *et al*., 2007), and cancers of the gastrointestinal (Milham & Demers, 1984;Henneberger *et al*., 1989; Sala-Serra *et al*., 1996), and respiratory systems (Milham & Demers, 1984; Siemiatycki *et al*., 1986; Toren *et al*., 1991; Band *et al*., 1997), certain lymphatic and haematopoietic neoplasms (Coggon *et al*., 1997; Band *et al*., 1997; Matanoski *et al*., 1998), brain cancer (Band *et al*., 1997; Andersson *et al*., 2002), ovarian cancer (Langseth & Andersen, 1999), and kidney cancer (Band *et al*., 1997). Non-malignant respiratory effects of specific exposures have also been reported.

#### *Non-malignant effects*

In numerous studies an association between exposures in the pulp and paper industry and non-malignant respiratory effects have been observed, including wheezing symptoms (Andersson *et al*., 2003; Henneberger *et al*., 2005), asthma (Glindmeyer *et al*., 2003; Henneberger *et* al., 2005; Andersson *et al*., 2006), bronchial hyperresponsiveness (Malo *et al*., 1994; Andrae *et* al., 1988), chronic bronchitis (Henneberger *et* al., 2005), decline in pulmonary function parameters including $FEV_1$, FVC and $FEV_1$/FVC ratio (Heederik *et* al., 1987; Mehta *et* al., 2005) and rhinitis

(Hoffman *et al*., 2004). The exposures implicated in these studies include soft-paper dust, ozone, chlorine dioxide and sulphur dioxide.

Mortality from non-malignant disease in female pulp and paper workers in Norway has also been investigated (Langseth & Kjærheim, 2006), and significantly elevated mortality (SMR 1.14, 95% CI 1.05-1.24) was observed. This was mainly attributed to increased risk of ischaemic heart disease (SMR 1.22, 95% CI 1.03-1.24) and cerebrovascular diseases (SMR 1.16, 95% CI 0.94-1.42). Short term workers in paper departments showed the highest mortality risk compared to other departments. Women ever employed in paper departments had a 5% and 9% increased risk of dying from ischaemic heart disease and respiratory diseases respectively, compared to women never employed in paper departments.

In another cohort study conducted to investigate cardiovascular mortality among Swedish pulp and paper mill workers (Persson *et al*., 2007), there was an increased mortality risk in males from diseases of the circulatory system in those with more than 20 years employment (SMR 113, 95% CI 101 – 126). In those with more than one year of employment there was increased risk from ischaemic heart disease (SMR 109, 95% CI 99-120) and from cerebrovascular diseases (SMR = 104, 95% CI = 84-127). Work with steam and power generation, sulphate digestion, and maintenance were all related to significant increases of risk of death from diseases of the circulatory system.

***Malignant effects***

As well as the excess risk of specific cancers found in pulp and paper worker cohorts from several countries mentioned above, the overall dataset from the IARC study has also been used to evaluate cancer risk associated with three specific exposures in the industry. An investigation of asbestos exposure and lung and pleural cancer mortality found a deficit in overall mortality and in mortality from malignant neoplasms (Carel *et al*., 2002). Mortality from pleural cancer, however, was significantly increased among exposed workers compared to unexposed workers (RR 2.53, 95% CI 1.03-6.23), and a trend in both pleural and lung cancer mortality was suggested according to weighted cumulative exposure and duration of exposure. The results suggested an association between estimated exposure to asbestos, as found in most industrial

plants, and an elevated mortality from pleural and lung cancer. A (non-significant) increase in risk of lung cancer (SMR 1.33, 95% CI 0.94-1.83, 37 deaths) was also found in the New Zealand pulp and paper workers cohort (McLean *et al*., 2002), with a significant increase (SMR 1.45, 95% CI 1.00-2.04, 33 deaths) observed in those workers with exposure to pulp and paper dust.

Another study evaluated risk associated with sulphur dioxide exposure in the pulp and paper industry (Lee *et al*., 2002). After adjustment for occupational co-exposures, an increased risk of lung cancer among exposed workers compared with non-exposed (rate ratio 1.49, 95% CI 1.14-1.96) was suggested. In another evaluation of the risk associated with exposure to organochlorine compounds a weak but statistically significant association between all-cancer mortality and weighted cumulative exposure to volatile organochlorine compounds (including trichloroethylene, perchloroethylene, dichloromethane and trichloromethane) was observed (McLean *et al*., 2006).

### 4.5 Joinery and furniture-making industry

**Summary**

- A number of studies have evaluated both exposures and health effects in the joinery and furniture making industry.
- The most significant exposures observed have been to inhalable wood dust, with only relatively low exposures to formaldehyde, bacterial endotoxin and monoterpenes reported.
- Average inhalable wood dust levels well in excess of 1 mg/m³ are common, although levels consistently below 1 mg/m³ have been reported from the Danish furniture making industry.
- Cleaning and sanding processes appear to produce the highest wood dust exposures.
- Extraction systems have been shown to be highly effective at reducing wood dust levels.

- Wood dust exposures in this industry have been associated with increased mortality from non-malignant respiratory disease, and also with the prevalence of a range of non-malignant respiratory diseases such as reduced lung function, asthma and chronic bronchitis.
- Large excesses of sino-nasal cancer risk have been observed in furniture workers processing hardwoods such as beech and oak, with a much lower although still statistically significant excess observed in those handling softwoods and/or MDF.
- Associations between wood dust exposure and several other cancers have also been observed.

### 4.5.1 Introduction

The joinery and furniture making industries use a combination of solid wood and reconstituted wood products. Occupational hazards in these industries relate predominantly to wood dust and formaldehyde emissions, from either the reconstituted wood products or other glues and resins used. This chapter describes studies that have focused on exposure levels (mostly to wood dust) and associated health effects. Since these studies are of direct relevance to the exposure survey conducted in the New Zealand joinery and furniture making industry (Chapter 6) they will be discussed in more detail than studies cited in the previous sections.

### 4.5.2 Exposure studies

Time weighted average total and respirable personal dust and formaldehyde exposures were measured in 48 workers in four cabinet-making plants in Toronto, Canada (Sass-Kortsak *et al*., 1986). The mean exposure for total dust was 1.83 mg/m$^3$ (SE 0.22), for respirable dust 0.29 mg/m$^3$ (SE 0.05), and for formaldehyde 0.06 ppm (SE 0.01). Five work categories were investigated: sawing, sanding, assembly, lamination graphics and gluing, plus a miscellaneous group. The respirable dust concentration for sanding (0.63 mg/m$^3$ ± 0.20) was significantly higher than for the other categories. Sanding operations also produced a higher percentage of respirable dust (22%) than sawing (6%) and assembly (13.5%). Total dust concentration levels were highest for sanding

(2.91 mg/m$^3$ ± 0.70) followed by assembly (1.85 mg/m$^3$ ± 0.22) and lowest for sawing (1.72 mg/m$^3$ ± 0.61), and the other categories.

Personal inhalable dust exposures in 182 workers were measured in two logging sites, four sawmills, one major wood chipping operation, and five joineries in New South Wales, Australia (Alwis *et al*., 1999). The overall geometric mean personal inhalable dust exposure in 66 samples in joineries was 3.7 mg/m$^3$ (GSD 3.67). A one-way analysis of variance (ANOVA) revealed that joinery operations job title (P=0.0001), local exhaust ventilation (P<0.001), use of compressed air (P<0.001), type of wood processed (P=0,001), and cleaning method used (P<0.001) were the main determinants of wood dust exposure, while the use of handheld tools was not found to be a significant determinant (P=0.22).

In a methodological study, airborne endotoxin was measured in three woodworking (joinery) shops in the south-eastern US using three different samplers tested side-by-side including the 37 mm closed face plastic cassette (CFC), the IOM inhalable sampler, and the Button sampler developed by the University of Cincinnati (Harper & Andrew, 2006). No long-term TWA samples exceeded the recommended limit value of 50 EU/m$^3$ used in the Netherlands. The geometric mean concentration from the IOM samples was 11 EU/m$^3$, from CFC samples it was 3.6 EU/m$^3$ and from the Button samples it was 2.1 EU/m$^3$.

Dermal exposure to monoterpenes α-pinene, β-pinene and $\Delta^3$-carene has been assessed in thirty joiners (Eriksson & Wiklund 2004). Sampling was carried out during sawing of pine in joinery shops. The total potential body exposure during sawing was 238 mg/h and for collecting it was 100 mg/h. For the hands, the total exposure was 9.24 mg/h during sawing and 3.25 mg/h during collecting. Monoterpenes were detected at patches which were placed on the skin underneath the clothes which indicated contamination of the skin. However, the levels of monoterpenes on the inside of the clothes were significantly lower than the levels on the outside. Exposure to the inner chest and inner lower right leg were statistically lower (P<0.001) compared with the exposure at the outer chest and outer lower right leg during sawing.

In a study on exposure to wood dust and endotoxin in small-scale wood industries in Tanzania (Rongo *et al*., 2004), dust (n=281) and endotoxin (n=157) samples were collected. The overall average (GM) wood dust exposure was 3.32 mg/m$^3$ (GSD 2.47), and the overall average (GM) endotoxin exposure was 91 EU/m$^3$ (GSD 3.74). Wood dust levels were highest in carving (GM 15 mg/m$^3$) and cleaning operations (GM 10 mg/m$^3$) and the lowest in machine planing (GM 2 mg/m$^3$). Also, on average, wood dust levels were 1.69 times higher in the dry season than in the wet season. The highest endotoxin exposure was recorded when workers were sewing seat covers (GM 384 EU/m$^3$, GSD 4.43). On average, endotoxin levels were 2.6 times higher in the dry season than in the wet season. Wood dust and endotoxin levels were correlated, but the correlation was relatively weak (r=0.44; P=0.001; n=159).

In a recent Danish study, the determinants of wood dust exposure in the furniture manufacturing industry were investigated (Mikkelsen *et al.,* 2002). During 1997 and 1998 they took 2,362 inhalable dust samples from 54 factories, finding an overall average (GM) dust level of 0.95 mg/m$^3$ (GSD 2.08). Several determinants of increased dust exposure were identified (see Chapter 5.2). There were increased exposures to inhalable dust for woodworking (1.20 mg/m$^3$), manual sanding (1.99 mg/m$^3$), sanding and cutting (1.35 mg/m$^3$), automatic sanding (1.27 mg/m$^3$) and cutting (1.10 mg/m$^3$). The use of compressed air increased the dust exposures but this was only significant for cutting (1.13mg/m$^3$, P<0.05) and general woodworking (1.24 mg/m$^3$, P<0.05). In addition, having dust on the work piece produced increased dust exposure for manual sanding (2.40 mg/m$^3$), sanding and cutting (1.45 mg/m$^3$), automatic sanding (1.41 mg/m$^3$), handling and assembling (0.77 mg/m$^3$).

As part of the same research project, a cross-sectional study measuring wood dust exposure was performed in the Danish furniture-making industry (Schlunssen *et al*., 2001). From the 54 furniture factories they carried out 1,685 personal inhalable dust measurements. The types of wood the workers were exposed to were broken down to: pine (42%), MDF or particle board (15%), hardwood (6%), mixed wood types (34%), and no information available (4%). The investigators assessed inhalable wood dust exposure to specific work tasks and found manual sanding produced the highest exposures (GM 1.99, GSD 1.88), while handling and assembling in wood working departments and other departments produced the lowest exposures (GM 0.69, GSD

2.02; and GM 0.70, GSD 2.04, respectively). The size of the factory (judged by the number of employees) was also found to correlate with wood dust exposure. It was found that in smaller factories there was higher inhalable wood dust exposure (GM 1.81, GSD 2.39 in factories with 5-9 employees) compared with larger factories (GM 0.63, GSD 2.15 in factories with 200-499 employees). The results of this study were compared with a 1988 cross-sectional Danish study of the wood and furniture industry in order to observe time-dependent changes in exposure levels, and inhalable wood dust exposure in this study was found to be approximately 50% less than in the 1988 study.

In a wood dust exposure survey conducted in the British woodworking industry in 1999/2000, a total of 406 personal inhalable samples were taken from 46 sites (Black *et al*., 2007). Most companies (80%) employed fewer than 25 workers. The effects of woodworking machines or activity on exposure was analysed and it was found that using a circular saw, sanding, and cleaning created the highest levels of exposure (median ranged from 3.2 to 10.8 $mg/m^3$). The influence of local exhaust ventilation (LEV) on exposure was also assessed and it was found that LEV provided inadequate control for circular saws (44% of samples $<5mg/m^3$), assembly (50% of samples $<5mg/m^3$), and the 'other' category (63% of samples $<5mg/m^3$). The influence of weekly checks or thorough 14-monthly checks on LEV was assessed and it was found that median TWA levels increased if either weekly or 14-monthly checks were not conducted (3.2 $mg/m^3$). Recirculated filtered air was not found to be a significant source of exposure i.e. whether circulated or not in both cases 73-74% of exposures were $<5mg/m^3$.

### 4.5.3 Health effects studies

A large number of studies of health effects in joinery and furniture manufacturing workers have been published, as summarised below.

***Non-malignant respiratory effects***

Associations have been observed between wood dust exposure in furniture workers and a range of both acute and chronic non-malignant respiratory diseases such as

decreased pulmonary function, asthma(-like) symptoms and allergies. Increased mortality from non-malignant respiratory disease has also been observed. For example, participants from the American Cancer Society's Cancer Prevention Study – II were prospectively followed for mortality for 6 years from 1982, with occupation and exposure information obtained by questionnaires (Demers *et al.,* 1998). The study population included men who worked in a wood-related occupation (including carpenters, furniture makers and woodworkers) and a reference population who had not worked in wood-related jobs or had no exposure to wood dust. The study group consisted of 11,541 men with 1,271 deaths observed, whereas the reference group consisted of 317,424 men with 22,994 deaths observed. Among wood exposed workers there was a small excess risk of death from all causes (RR 1.17, 95% CI 1.11-1.24), and an excess for deaths from non-malignant respiratory disease (RR 1.42, 95% CI 1.15-1.74).

In a cross-sectional study of 296 furniture workers, participants were examined using a questionnaire, a spirometry test, a bronchial provocation test with methacholine, and a skin prick test (Talini *et al.,* 1998). The workers were divided into spray painters (exposed to low concentrations of diisocyanates and solvents), woodworkers (exposed to wood dusts), and assemblers (control group). The prevalence of any shortness of breath with wheeze and dyspnoea were higher in spray painters (13.5% and 11.5%, respectively) and woodworkers (7.7% and 6.4%) compared to assemblers (1.6% and 1.6%). There was no significant difference between the groups for bronchial hyperreactivity. Asthma-like symptoms combined with non-specific bronchial hyperreactivity was more common in woodworkers (10%) and spray painters (13.3%) than in assemblers (4%), but this did not reach statistical significance.

In a study of respiratory effects among 38 workers in Swedish joinery shops (Eriksson *et al.,* 1997), wood dust exposure was measured by personal air sampling and monoterpene exposure using personal diffusive sampling with charcoal. Urine samples taken before and after the work shift were also analysed to determine the amount of verbenols in the urine. A respiratory questionnaire was administered in all workers, and lung function tests were carried out before and after the work shift. The average (GM) exposures measured were 43 mg/m$^3$ for terpenes, 0.4 mg/m$^3$ for wood dust and 9.12 μmol/mmol for verbenols. Post-shift urine samples generally contained

higher concentrations of verbenols than the pre-shift samples, and mean personal exposures to α-pinene during a workday were significantly correlated with the concentration of verbenols in urine after the end of the work shift (r=0.69). In pre-shift measurements in joinery workers several lung function parameters were found to be significantly lower than in the reference group (P=0.07-<0.001), and the absence of major changes in cross-shift lung function were considered to be indicative of chronic rather than acute reactions in the airways.

In a study conducted in four sawmills, a wood chipping mill, and five joineries (including two which processed only MDF) in New South Wales, Australia, the effects of personal exposure to wood dust, bacterial endotoxins, (1→3)-β-D-glucans, Gram-negative bacteria, and fungi on lung function among woodworkers were examined (Mandryk *et al*. 1999). Average (GM) levels of inhalable dust exposure in the two joineries using MDF were 11.47 mg/m$^3$ (GSD 2.02) and 7.32 mg/m$^3$ (GSD 2.86); of inhalable endotoxin 5.98 ng/m$^3$ (GSD 3.20) and 5.22 ng/m$^3$ (GSD 1.88); and of inhalable (1→3)-β-D-glucan 0.50 ng/m$^3$ (GSD 1.22) and 0.30 ng/m$^3$ (GSD 1.73). The mean levels of exposure to respirable dust were 0.80 mg/m$^3$ (GSD 2.06) and 0.54 mg/m$^3$ (GSD 2.02), of respirable endotoxin 0.71 ng/m$^3$ (GSD 2.09) and 0.36 ng/m$^3$ (GSD 1.92), of respirable (1→3)-β-D-glucan 0.12 ng/m$^3$ (GSD 1.19) and 0.12 ng/m$^3$ (GSD 1.12).

When compared with the reference population the woodworkers were found to have a higher prevalence of regular cough, phlegm, and chronic bronchitis, and also a lower percentage predicted lung function i.e. the percent predicted FVC in wood workers was 84.7% (± 0.72%) versus 94.9 (± 3.85) in the referents; the percent predicted $FEV_1$ in wood workers was 84.7% (± 1.20%) versus 93.1% (± 2.81) in the referents. The correlations found between personal exposures and lung function indices suggested that airborne wood dust and biohazards associated with wood dust (endotoxins, (1→3)-β-D-glucans, fungi, and Gram-negative bacteria) had negative effects on the pulmonary function of woodworkers.

In another cross-sectional survey of woodworkers (Carosso *et al.*, 1987), subjects completed a questionnaire, underwent skin prick, serological, and lung function tests.

A significant negative correlation was detected between the lung function parameters $FEV_1$, $MEF_{50}$, $TL_{CO}$, and $K_{CO}$, and duration of exposure to wood dusts.

Another study focussing on respiratory symptoms was conducted among male workers exposed to wood dust (n=546) in small-scale wood industries in Tanzania, and non-exposed workers (n=565) (Rongo *et al.,* 2002). A sample of the exposed workers (n=106) were also monitored to assess wood dust exposure. Wood dust exposures ranged from 2.9 mg/m$^3$ to 22.8 mg/m$^3$, and the overall average (GM) was 3.86 mg/m$^3$ (GSD=2.33). "Shortness of breath with wheezing" (OR=1.9, 95% CI=1.1-3.4) was significantly more prevalent in the exposed compared to the unexposed workers, as was "woken by shortness of breath" (OR=2.1, 95% CI=1.4-3.1). Runny nose and sneezing for more than once a week was also strongly associated with exposure to dust (OR=2.3, 95% CI=1.4-3.6, $P \leq 0.01$) as was sensitivity to house dust, food, animal, or grasses/plants (OR=2.4, 95% CI=1.9-3.1, $P \leq 0.01$).

Pulmonary function was examined in furniture workers (n=145) and a reference population from a bottling factory (n=152) in the republic of Transkei (Shamssain, 1992). Among the exposed workers 20% were working with pine, and 80% with MDF. The mean personal total dust concentration in the factory was 3.82 mg/m$^3$ (SD 1.34). Exposed male workers had significantly lower forced expiratory indices compared to the reference group ($P<0.001$) with a forced expiratory flow between the first 200 ml and 1200 ml of FVC (FEF 200-1200) and peak expiratory flow (PEF) of 81.3% and 89.4% of predicted respectively. The percentage of workers with an $FEV_1$/FVC less than 70% was significantly higher ($P<0.01$) in the exposed group (30.3%) compared with the reference group (17.4%), and this percentage was significantly higher ($P<0.01$) in exposed workers with longer duration of employment. FVC showed a significant ($P<0.01$) inverse association with exposure, equating to an approximately 26 ml decline per year of exposure. Exposed workers had a significantly higher prevalence of respiratory symptoms, such as nasal symptoms (50% vs. 19%), wheezing (13% vs. 5%), cough (43% vs. 29%) and phlegm (15% vs. 9%; not statistically significant); cough and nasal symptoms increased with years of employment.

Personal inhalable dust exposures and respiratory symptoms in employees (n=193) from 15 Australian furniture factories were examined in a cross-sectional study, and compared with a reference group of 46 male hospital maintenance staff (Pisaniello *et al.,* 1991). All participants answered a health questionnaire, and their personal exposure to wood dust was measured each day over the two-day study period. Average wood dust exposures measured in wood machinists were 3.2 mg/m$^3$, in cabinetmakers 5.2 mg/m$^3$, and in chair makers 3.5 mg/m$^3$. A limited number of formaldehyde samples were also collected, which showed a mean concentration of 0.05 ppm (range 0.029-0.091) in situations where reconstituted wood was being machined or sanded. The mean value for hardwood exposure was 3.8 mg/m$^3$ (GSD 2.8), and for reconstituted wood exposures the mean value was 3.3 mg/m$^3$ (GSD 2.7). There were only minor differences in particle size distributions for "sanding, sawing, and mixed woodworking operations with mass median aerodynamic diameter ranges of 16-19, 17-22, and 15-23 μm, respectively.

Significant differences were observed between the woodworkers and the reference group for eye irritation (35% vs. 20%), ear inflammation/infection (15% vs. 4%) and several nasal symptoms, such as regular blocked nose (51% vs. 30%), frequent sneezing (41% vs. 17%), regular runny nose or excess nasal secretion (45% vs. 11%), reporting 2 or more nasal symptoms (52% vs. 28%), and reporting 3 or more nasal symptoms (35% vs. 15%). Users of hardwoods were most likely to experience nasal problems with 57% reporting regular blocked nose, 49% reporting frequent sneezing, 54% reporting regular runny nose or excess nasal secretion, 60% reporting 2 or more nasal symptoms, and 43% reporting 3 or more nasal symptoms. The authors found that exposure-effect gradients were not discernible in this cross-sectional study, except for some nasal symptoms.

Ventilatory capacity in workers exposed to wood dust in two furniture factories was compared with a reference population in a power station (Al Zuhair *et al.,* 1981). The concentrations of dust measured in various work zones in the furniture factories ranged from 0.01 to 4.40 mg/m$^3$, and in the breathing zones of workers it ranged from 0.09 to 8.29 mg/m$^3$. The concentration of airborne respirable dust was measured in only one of the furniture factories and ranged from 0.08 to 0.44 mg/m$^3$. A significant fall in FVC and $FEV_1$ across the work shift was observed in workers of both furniture

factories. No dose-response relationship was found in any factory between the levels of exposure to airborne dust and changes in $FEV_1$ and FVC, although those exposed to the highest dust concentrations in one of the furniture factories showed the largest drop in $FEV_1$ and FVC.

In another study, furniture-design students (n=64) producing furniture using mainly fir were compared with students (n=62) from different departments in the same schools (Arbak *et al.,* 2004). Symptoms of cough (23.4% vs. 8.1%, P=0.016) and shortness of breath (18.8% vs. 6.5%, P=0.034) were higher in furniture-design students compared with the reference group. Furniture-design students also had a higher prevalence of conjunctivitis (34.4% vs. 9.7%, P=0.001) and of rhinitis (34.4% vs. 19.4%, P=0.044). Respiratory function and chest radiographs were normal in both groups. PEF recordings were performed for approximately one month, and diurnal variability greater than 20% was observed in 12 of the 64 (18.7%) design students, whereas it was detected in only 4 of the 62 (6.4%) controls (P=0.034). No difference in occurrence of diurnal variability greater than 20% was observed at weekends between both groups (P=0.457).

A series of cross-sectional exposure and respiratory health studies have been conducted in woodworkers from 54 Danish furniture factories who worked with pine or reconstituted wood (54%), hardwood (8%) and mixed wood types (39%), and a reference population from three factories with no wood dust exposure (Schlünssen *et al.*, 2002a, 2002b, 2004a, 2004b, 2008). In the first study, furniture workers (n=161) and controls (n=19) were tested for mucosal swelling and acute nasal obstruction using acoustic rhinometry (Schlunnsen *et al.*, 2002a). Personal passive dust monitoring was performed on 140 of the woodworkers, and a positive wood dust dose-dependent relationship was found with mucosal swelling and acute nasal obstruction. This was most marked after four hours of work.

Spirometry was performed on 2,423 of the furniture workers, and questionnaires about respiratory symptoms and wood dust exposure were administered to 2,033 furniture workers and 474 workers from the reference population (Schlunssen *et al.*, 2002b). Personal passive inhalable dust samples were collected from 1,579 of the furniture workers. The wood dust levels measured were relatively low (GM 0.93

mg/m$^3$, GSD 2.10), and within the 0.17 – 9.78 mg/m$^3$ range. Despite the relatively low dust levels the authors observed a dose-response relationship between dust exposure and asthma symptoms, a higher prevalence of wheezing and a cross-shift decrease in $FEV_1$ in the workers exposed to pine dust. In a study published two years later (Schlunssen *et al.*, 2004a) in which asthma was assessed by symptom questionnaires and pulmonary function tests, the authors found an association between wood dust exposure and asthma symptoms and bronchial hyperresponsiveness (BHR) in atopic workers. No clear pattern was seen in non-atopic workers or for other indices of asthma.

In a subgroup of workers (n=365) and controls (n=88), the authors explored the associations between wood dust exposure and sensitisation to wood dust, and the relationship between wood dust sensitisation and asthma (Schlunssen *et al.*, 2004b). This study indicated that although the level of exposure to wood dust had an impact on the rate of sensitisation, IgE mediated allergies did not play a major role in occupational asthma in woodworkers.

It should be noted that the measured effects in the above studies were relatively modest and often were not statistically significant; this may be due to the relatively low level of wood dust exposure and/or to the common bias in cross-sectional studies known as the healthy worker effect (Checkoway *et al.*, 2004).

In a (longitudinal) follow-up study conducted 6 years later (Schlunssen *et al*., 2008), a study design which is less prone to bias from the healthy worker effect, the authors found a significant decline in lung function (i.e. -27.97 ml/year $FEV_1$ in the highest exposure category, and in the cumulative incidence proportion of $FEV_1$/FVC <75%) associated with cumulative dust exposures, and these were most apparent in female workers.

Another five-year longitudinal respiratory health study was conducted in the wood processing industry, involving workers from one plywood plant, one combined sawmill/planing/plywood plant, one milling plant, three cabinet making plants and four furniture-making plants (Glindmeyer *et al.*, 2008). A total of 2,363 personal samples were collected, divided into three size fractions (extrathoracic,

tracheobronchial, and respirable). They were then further apportioned into: wood solids (the major non-volatile constituents of wood) and residual particulate matter which was composed of some materials derived from wood (water and wood volatiles; contaminants related to its storage and/or processing; and background particulate contaminants generally present in industrial facilities). These were used to calculate each individual's 8-hour TWA exposure for 65 exposure types. Spirometric tests and health questionnaires were conducted on 1,164 subjects. The overall mean exposure levels were 0.66 mg/m$^3$ for extrathoracic fractions, 0.32 mg/m$^3$ for tracheobronchial fractions, and 0.05 mg/m$^3$ for respirable fractions. Statistically significant effects were observed only for respirable residual particle matter in the milling facility (i.e. the mean exposure of 0.147 mg/m$^3$ was associated with a change in $FEV_1$ of -32 ml/year, a change in $FEV_1$/FVC of -0.48 %/year, and a change in $FEV_{25-75}$ of -0.11 l/s/year), and in the combined sawmill/planning/plywood facility (i.e. the mean exposure of 0.256 mg/m$^3$ was associated with a change in $FEV_1$ of -59 ml/year and a change in FVC of -103 ml/year). No significant adverse effects of wood solids exposures were found.

In addition to these international studies one (small) study was conducted in New Zealand furniture makers (Norrish *et al.*, 1992). The study involved 44 wood workers and 38 controls with no dust exposures. Inhalable dust exposure levels ranged from 1.0 to 25.4 mg/m$^3$ with a median of 3.6 mg/m$^3$. Full shift formaldehyde levels ranged from less than 0.01 to 0.27 mg/m$^3$ with a median of 0.06 mg/m$^3$. Both lower and upper respiratory symptoms were significantly more common in wood workers than in the controls (e.g. persistent cough in winter, OR 7.6, CI 1.4-52.7; persistent cough in summer, OR 9.5 CI 1.1-210.9; breathlessness, OR 3.4, CI 1.1-11.2; nasal obstruction, OR 6.0, CI 2.0-18.2). Five workers were identified as suspected cases of occupational asthma based on symptoms and repeated peak flow measurements. Exposure to (Rimu) dust was frequently cited by workers as being associated with respiratory symptoms.

***Sino-nasal cancer***

In the literature reviewed in this section, we begin with the well known association between sino-nasal cancer and exposure of furniture workers to hardwood dusts such

as beech and oak; we then describe studies in which softwood dust is implicated – which in some cases is stated and in others can be assumed due to the region in which the study was conducted.

A large excess of sino-nasal cancer (500-fold for sino-nasal adenocarcinoma) was seen in furniture and other workers exposed to wood dust in the High-Wycombe area of England (Acheson *et al*., 1968), with the highest risks apparent in workers exposed to dusts from hardwoods such as beech and oak although in subsequent studies people with sino-nasal cancer exposed to many other species including softwoods have also been identified. Follow up studies of nasal cancer in wood workers in England and Wales (Acheson *et al*., 1981) have shown that while the standardised incidence ratio (SIR 1.49) for carpenters and joiners did not reach the conventional limit of significance ($P>0.05$), significant elevations were observed for cabinet and chair-makers (SIR 9.66, $P<0.01$), for machinists (SIR 6.16, $P<0.05$), and for other woodworkers (SIR 2.93, $P<0.05$). In another follow-up study (Acheson *et al*., 1984), no relationship was found between increasing levels of dust in the workplace and increasing mortality for any other cancer site apart from nasal cancer (RR=8.1, 95% CI 3.7-15.4, $P<0.05$).

Since the original finding of excess risk of sino-nasal cancer among furniture workers exposed to hardwoods, an excess has been observed in working populations for which the predominant or only exposure has been to softwood dusts. A number of case-control studies have shown increased risk of cancers in joinery and furniture manufacture. Seven cases of nasal cancer in furniture-makers in Denmark were investigated using the Danish death certificates for the years 1956-1966 (Mosbech & Acheson, 1971). An interview with the relatives of the deceased patients was carried out to form a detailed occupational history of the patient. Five of the cases were male woodworkers and the other two cases were wives of carpenters. Four furniture-makers died of nasal adenocarcinoma during 1956-1966, and since it is a rare type of tumour, which appears in other countries in the same occupation, the authors concluded that there was an increased risk of nasal adenocarcinoma in the Danish furniture-making industry.

In a registry based study in Denmark (Olsen *et al*., 1988), there was an increased risk of sino-nasal cancer evident in those engaged in wood manufacturing occupations (standardised prevalence incidence ratio or SPIR 2.07, $p<0.05$), with a higher risk evident in the manufacture of wooden furniture (SPIR 3.60, $P<0.05$). In a recent Swedish census linkage study (Hemelt *et al.*, 2004), sino-nasal cancer risk for a number of occupations including wood workers was assessed. In this study, it was assumed that woodworkers who were furniture makers were exposed to both indigenous softwoods and imported hardwoods, while construction workers were only exposed to softwood. Wood workers overall had a significantly elevated risk for total nasal cancer (SIR 1.19, 95% CI 1.53 – 2.36), and particularly for adenocarcinoma (SIR 5.36, 95% CI 4.02 – 6.99). Those with the longest duration of exposure (i.e. over 4 census periods) to softwood dust only had a significant excess risk (SIR 7.31, 95% CI 1.38-21.7). For those with longest duration of exposure (i.e. over three census periods) to combined softwood and hardwood dust (i.e. furniture makers) there was significant excess risk of total nasal cancer (SIR 10.0, 95% CI 4.76-18.5) and particularly for adenocarcinoma (SIR 28.6, 95% CI 13.6-52.7).

A Japanese study found that cancers of the nasal sinus were significantly elevated (RR 2.9, $P<0.05$) in male carpenters, joiners and furniture makers (Fukuda *et al*., 1987). In a related study (Fukuda & Shibata, 1988), men who were carpenters, joiners, furniture makers, or other woodworkers were found to be at significant risk of developing maxillary sinus cancer (RR 2.9, 95% CI 1.45-5.64, $P<0.05$). In a case-control study conducted in The Netherlands (Hayes *et al*., 1986), adenocarcinoma was very strongly associated with furniture and cabinet making (OR 139.8, 90% CI 31.6-999.4), and factory joinery and carpentry work (OR 16.3, 90% CI 2.8-85.3). In another Japanese study (Shimizu *et al*., 1989), men who had a history of doing woodwork involving only sanding/lathe work had a significantly increased risk of squamous cell carcinoma of the maxillary sinus (RR 7.5, 95% CI 1.5-38.5, $P=0.02$).

A pooled reanalysis of cancer mortality of workers in wood-related industries combined five cohort studies (Demers *et al.*, 1995). The combined cohort included 28,704 people, of whom 7,665 had died. While overall mortality was significantly lower than expected when compared with the general population, sino-nasal cancer (SMR 3.1, 95% CI 1.6-5.6) and nasopharyngeal cancer (SMR 2.4, 95% CI 1.1-4.5)

were in significant excesses. The excess of nasal cancer was seen only in British furniture workers, while the nasopharyngeal cancers were observed in both furniture and plywood mill workers. Cancers of the buccal cavity and pharynx, intestines, liver, lung, skin and leukaemia were significantly lower than compared with the general population. In furniture workers, sinonasal cancer (SMR 4.3, 95% CI 2.2-7.8) and nasopharyngeal cancer (SMR 2.9, 95% CI 1.2-5.9) were again, the only statistically significant excesses.

These results were stratified by the likelihood of wood dust exposure, and it was shown that the risks of sinonasal cancer (SMR 8.4, 95% CI 3.9-16.0) and nasopharyngeal cancer (SMR 5.3, 95% CI 1.7-12.4) was elevated in the group with definite exposure to wood dust. When stratified by decade of first employment, the highest risk of sinonasal cancer SMR (SMR 12.5, 95% CI 5.7-23.7) was found in those first employed prior to 1940. For workers employed from 1940 to 1949 elevated SMRs were shown for nasopharyngeal cancer (SMR 5.3, 95 CI 1.7-12.3) and for multiple myeloma (SMR 1.9, 95% CI 1.0-3.3). For workers at least 30 years after their employment, elevated SMRs were shown for sinonasal cancer (SMR 7.6, 95% CI 3.3-15.0) and nasopharyngeal cancer (SMR 8.9, 95% CI 3.6-18.3). In summary, this pooled reanalysis found that wood workers have an excess of nasopharyngeal cancer, multiple myeloma and sinonasal cancer.

***Other cancers***

Associations with other respiratory cancers have also been observed in joinery and furniture manufacture workers. For example, a non-significant increase in risk of laryngeal cancer (RR 8.1, 95% CI 0.95-68.78) has been observed in woodworkers and furniture makers in a case-control study on the Texas Gulf Coast (Brown *et al*., 1988). In a hospital-based case-control study on occupational exposures and risk of oesophageal cancer conducted in eastern Spain (Santibanez *et al*., 2008), there was a statistically significant increase in adenocarcinoma (OR 9.69, 95% CI 1.32-70.81). Cabinet makers and related workers as well as wood-processing and papermaking-plant operators had increased risks for squamous cell carcinoma of the oesophagus but these were not statistically significant.

Case-control studies conducted in Germany have shown elevated risk for testicular cancer and lung cancer in cabinet makers. The case-control study of testicular germ cell cancer (Stang *et al*., 2005) found that cabinet makers had an increased risk (OR 2.52, 95% CI 0.94-6.76) compared with carpenters (OR 0.59, 95% CI 0.16-2.14), and that there was a greater risk (OR 3.58, 95% CI 1.08-11.86) in people who worked as a cabinet maker for longer than 5 years. A pooled analysis of two case-control studies on occupational lung cancer risk for men in Germany showed that the paper, wood, and printing occupations combined had an increased risk for lung cancer (OR 1.31, 95% CI 1.10-1.56), primarily in cabinet makers or wood processing workers (OR 1.36, 95% CI 1.11-1.66) (Bruske-Hohfield *et al*., 2000).

In a study of the incidence of non-Hodgkin's lymphoma in different employment categories, using data from the Swedish Cancer-Environment Registry, which links cancer incidence during 1961 to 1979 with occupational information from the 1960 census (Linet *et al*., 1993), it was found that workers in the furniture making and furnishing industry had increased risk (SIR 1.3, P<0.05, 55 cases). Other woodworkers in sawmills and planing mills also had increased risk (SIR 1.8, P<0.05, 17 cases). The authors noted that care must be taken in drawing causal inferences from this registry based analysis because information on exposure and duration of employment was not available, however the possible misclassification of exposure in a study such as this is likely to be random and therefore more likely to obscure relationships rather than to create false positive findings.

While all-cause mortality was lower than expected in a cohort mortality study among men and women first employed between 1946 and 1952 in union-organized furniture-making shops (Miller *et al.* 1994), there was an increased risk of pleural cancer (SMR 3.0, 95% CI 1.0-7.1), particularly in white male furniture workers (SMR 3.7, 95% CI 1.2-8.7). Subsequent analyses on those employed in wood furniture plants and followed for at least 20 years showed that white male furniture workers had a (non-significant) greater than 2-fold increase in Hodgkin's disease, myeloid leukaemia and chronic nephritis. Other male workers (a category comprising black and other non-white workers) also had a (non-significant) nearly 2-fold increased risk of mortality from cancers of the buccal cavity and pharynx, colon and rectum, pancreas, and prostate, and an SMR of 2.1 for infective and parasitic disease. For white female

workers who were followed up for at least 20 years, there were (non-significant) increased risks of pancreatic cancer (SMR 2.2) and lung cancer (SMR 1.4). In black and other female workers there was a 3.4-fold excess in breast cancer, although this too, was not statistically significant.

An excess of colon and rectal cancer has been observed in a retrospective cohort of wood dust exposure and cancer in furniture workers in Estonia (Innos *et al.,* 2000). The risk of sinonasal cancer was elevated (but not significantly) and there were no cases of nasopharyngeal cancers, but nine cases of other pharyngeal cancer occurred in men compared with 4.94 expected.

A cohort of 13,354 male carpenters in New Jersey who belonged to the United Brotherhood of Carpenters and Joiners in America were matched with the New Jersey State Cancer Registry from 1979 to 2000 (Dement *et al.,* 2003). All-cancer incidence in this cohort was increased (SIR 1.07, 95% CI 0.99-1.16), as were cancers of the digestive organs and peritoneum (SIR 1.24, 95% CI 1.04-1.47) with rectal cancer reaching statistical significance (SIR 1.51, 95% CI 1.05-2.10). There was also significant excess risk observed for cancers of the respiratory system (SIR 1.52, 95% CI 1.29-1.76), and in particular cancer of the trachea, bronchus and lung (SIR 1.45, 95% CI 1.22-1.72). Union members who have worked for more than 30 years had excesses in cancers of digestive organs and peritoneum (SIR 3.98, $P<0.05$), rectum (SIR 4.85, $P<0.05$), trachea, bronchus and lung (SIR 4.56, $P<0.05$), and other respiratory system areas (SIR 11.00, $P<0.05$). Analyses that lagged results 15 years since starting work in the carpenter union also revealed an excess in testicular cancer (SIR 2.48, $P<0.05$).

Male cancer incidence by occupation during the period 1972 to 1984 has also been investigated in New Zealand (Firth *et al.,* 1996). For the occupational code of "bricklayers, carpenters" there was elevated risk for all cancers combined (SIR 127, 95% CI=121-134). For specific cancer sites, carpenters had increased risk for cancers of the buccal cavity (SIR 1.84, 95% CI 1.30-2.53), stomach (SIR 1.61, 95% CI 1.13-2.22), lung (SIR 165, 95% CI 1.41-1.93), bladder (SIR 1.84, 95% CI 1.27-2.57) and multiple myeloma (SIR 2.65, 95% CI 1.45-4.46). For all woodworkers, the cancer sites with increased incidence were: lip (SIR 1.68, 95% CI 1.04-2.58), stomach (SIR

1.44, 95% CI 1.10-1.86), colon (SIR 1.36, 95% CI 1.11-1.65), rectum (SIR 1.46, 95% CI 1.16-1.81), nose and sinuses (SIR 1.30, 95% CI 0.34-3.36), lung (SIR 1.51, 95% CI 1.33-1.69), prostate (SIR 1.53, 95% CI 1.14-2.00), bladder (SIR 1.57, 95% CI 1.17-2.06), and multiple myeloma (SIR 2.30, 95% CI 1.44-3.49).

## 5. Control measures to reduce wood dust exposures

**Summary**

- The respiratory health effects associated with wood dust and the relatively high proportion of workers exposed in New Zealand to levels above international and proposed New Zealand exposure limits require effective control measures to be developed and implemented.
- Studies on determinants of wood dust exposure provide clues for control measures, but may on their own be insufficient for developing an effective control strategy.
- Only one intervention study which specifically focussed on control measures for wood dust has been identified.
- Educational intervention measures alone, such as risk education and providing information on good work practice, result in only a modest reduction in (wood) dust exposure.
- For more effective control measures to be developed – directed at the conditions and tasks that contribute most to these exposures - more detailed information on peak exposures is essential.

### 5.1 Introduction

As described in chapter three, most international standards have reduced the exposure limit for wood dust from 5 mg/m$^3$ to 1 mg/m$^3$ (or in some cases even lower). In response to these international developments, the Department of Labour has recently proposed a reduction of the New Zealand Workplace Exposure Standard for all wood dust to 1 mg/m$^3$. Given that dust exposure levels in the New Zealand joinery and furniture industries often exceed this level (see the results of our exposure survey in Chapter six), effective control measures are needed.

## 5.2 Determinants of wood dust exposure

A number of studies have focused on determinants of exposure in the wood conversion industry (Scheeper *et al*., 1995; Schneider *et al*., 2002; Mikkelsen *et al*., 2002; Schlunssen *et al*., 2001, 2008). These have indentified a number of activities and/or work processes that were associated with significantly increased or decreased dust exposures, and therefore provide clues for intervention strategies. For example, in a large study in Denmark involving 2,358 dust measurements in 54 furniture factories it was shown that sanding, use of compressed air, use of fully-automated machines, manual work, cleaning with compressed air, kitchen manufacturing, and small size (<20 employees) were associated with significantly increased inhalable dust levels, whereas manual assembling/packing, sanding with adequate exhaust ventilation, vacuum cleaning of machines, and professional cleaning staff were associated with significant decreases in dust exposures (Schlunssen *et al*., 2008). Smaller studies in countries with higher exposure levels have found similar results, including particularly strong reductions in exposures associated with improved exhaust ventilation (Scheeper *et al*., 1995; Alwis *et al*., 1999; Rongo *et al*., 2002).

Experimental studies have shown that changes to equipment such as using thinner saw blades, regularly sharpening saw blades, using saw blades with more and differently shaped teeth, and using lower drilling and sawing speeds can also reduce exposure levels. However, these data are often based on a small number of measurements and they are typically reported in the 'grey literature', most of which is not available in English.

There are also studies that have developed predictive exposure models for wood dust in sawmills (Friesen *et al*., 2006). Although these models explain a relatively high proportion (up to 54%) of the variability in exposure levels, they are of limited use in identifying effective control measures. They do, however, provide information about high and low exposed jobs and high and low exposure areas within each plant, and can therefore be helpful in guiding the development of control measures.

Thus, although studies such as those described above provide important clues for interventions, more detailed information on the occurrence and determinants of (peak)

exposures is required to develop a truly effective control strategy as will be explained below.

### 5.3 Peak exposures and control measures

For effective control measures directed at the conditions and tasks that contribute most to these exposures to be developed, more detailed information on peak exposures is helpful and in most cases essential. In most jobs in the wood conversion industry (and other industries), workers experience a series of short-term peak exposures associated with particular work activities that disproportionally influence the average 8 hour-time weighted average (8hr-TWA) exposure. As a consequence, 8hr-TWA exposures are not sensitive enough to identify (task-) specific determinants of exposure and/or to evaluate the effect of particular control measures.

This is also demonstrated by the fact that in most studies, the variability in 8hr-TWA dust exposures can only be explained to a limited extent by job, task and other work variables (Friesen *et al*., 2006; Meijster *et al*., 2007). As a result, control measures that are developed on the basis of the results of those studies have generally only had limited success in reducing exposure levels (Burstyn *et al*., 1997; Elms *et al*., 2005), although specific examples for the wood processing industry are currently not available. In contrast, studies targeting peak dust exposures have proven to be significantly more effective. This is demonstrated by a recent study in bakery workers which showed that specific task-based interventions targeted towards these peak exposures led to an overall reduction of peak exposures of more than 50%, between 22-100% depending on the task/intervention combination (Meijster *et al*., 2008). Of the 17 control measures applied only one showed no reduction in exposure, while 13 showed reduction in peak exposure levels of >50%. Because 75% of the time-weighted average exposure was directly associated with peak exposures that occurred with specific tasks and activities, a substantial reduction in the overall average daily exposures was feasible. To identify peak exposures and evaluate the effects of various interventions the investigators used real-time dust monitors. Others have used “video exposure monitoring” (VEM) which allows a graphical representation of a worker's exposure (as measured by a direct reading monitor) to be displayed on a video recording of the worker's activities (Rosén *et al*., 2005). Similar studies have to our

knowledge not been conducted in the wood conversion industry, and are therefore needed.

### 5.4 Intervention study in wood workers

The only intervention study that we are aware of in this industry is a study conducted in 48 small woodworking shops in Minnesota, USA (Lazovich *et al*, 2002). Rather than focusing on technical and engineering determinants of exposure, the authors applied a health promotion model aimed at changing health behaviours. The investigators evaluated behavioural and organizational aspects using planning committees, focus groups and a pilot study to gain information on the barriers and incentives surrounding the availability and use of dust controls. The intervention consisted of general written recommendations, technical assistance to enhance engineering, instructions on administrative methods to control wood dust, and worker training sessions to modify work practices associated with high dust production.

The outcome measures evaluated included work practice surveys, dust measurements and task observations by the investigators. A smaller than expected reduction in wood dust levels (10.4%) was achieved using these methods. While the investigators attributed the low level of improvement achieved to the difficulties inherent in engaging small business owners and workers, it is not clear to what extent they had engaged the study participants in the identification of the true determinants of exposure. The study reports the provision of instructions on administrative methods to control wood dust, and worker training to modify practices associated with high dust generation, but the evidence on which these recommendations were based is not clear. Given that the inhalable fraction of wood dust is not visible to the naked eye, the use of visual observations (even by trained investigators) as a method of demonstrating the elevated wood dust production associated with specific tasks to worker participants in the study is clearly less than optimal. A recent study in bakery workers confirmed that educational intervention measures alone, such as risk education and providing information on good work practice, result in only a modest reduction in dust exposure (Meijster *et al*., 2009), particularly when compared to the more effective approach that targeted specific peak exposures (Meijster *et al*., 2008). We

are not aware of intervention studies that have assessed the efficacy of measures to control other (non-wood dust) exposures in the wood processing industry.

Thus, no effective control strategies have been developed for the wood processing industry despite a large number of workers being exposed to levels well above international (and proposed New Zealand) standards. Based on current knowledge, control measures are likely be most effective when targeting activities such as sanding, cleaning, use of compressed air, etc., and could include changes in work behaviours, use of different tools, conducting tasks in a different location, use of vacuum cleaning rather than dry sweeping, use of alternatives to compressed air, and introducing or improving local exhaust ventilation, etc. However, more rigorous exposure surveys focussed on peak exposures are required to develop a truly effective control strategy for this industry.

# PART II: New Zealand Exposure Survey in Joineries and Furniture Factories

## 6. Exposure survey of airborne dust and formaldehyde in the New Zealand joinery and furniture making industry


**Summary**

- A significant proportion of New Zealand joinery and furniture workers (19% and 8% respectively) are exposed to inhalable dust levels in excess of the current New Zealand occupational exposure limit of 5 mg/m$^3$.
- The majority of New Zealand joinery and furniture workers (87% and 63% respectively) are exposed to inhalable dust levels in excess of current international standards of 1 mg/m$^3$.
- Dust exposure levels in furniture makers were half those of joinery workers.
- Approximately 10% of the inhalable dust mass that joinery and furniture workers are exposed to was respirable dust.
- Dust exposure levels in New Zealand furniture makers appear to have reduced significantly in the last 10-20 years;
- Formaldehyde exposures are very low in both joinery and furniture workers and are unlikely to play a major role in causing adverse health effects in these occupations.
- The relatively large proportion of workers exposed to dust levels in excess of 1 mg/m$^3$ suggests that many New Zealand (and international) joinery and furniture workers are at risk of developing work-related respiratory disease.
- Cost effective workplace interventions to reduce wood dust exposures in joinery and furniture workers are feasible and are urgently needed.


### 6.1 Introduction

As indicated earlier the original aim of this project was to collect dust and formaldehyde exposure measurements in the plywood/particle board and MDF manufacturing industry. However, because of problems of access, we have had to change the focus from plywood/particle board and MDF manufacturing to plywood/particle board and MDF processing industries. In particular, the study has targeted small, medium and large joineries and furniture makers. The exposure survey

involved measurements of airborne wood dust and formaldehyde and a target of 300 personal inhalable dust samples, 100 personal respirable dust samples, and 300 personal formaldehyde samples was set.

## 6.2 Materials and Methods

### *Recruitment of participants*

In total, 237 factories were identified through websites such as nkba.org.nz, fanzweb.org, masterjoiners.co.nz, tradefinder.co.nz and the yellow and white pages. Of these 237 factories/workshops, 122 factories were not eligible for inclusion because they: 1) were no longer in business; 2) were not joineries or furniture makers which used wood; 3) were retailers/importers/wholesalers; 3) were not in the targeted study areas (greater Wellington area, Hastings and Auckland); or 4) could not be contacted. Of the remaining 115 factories/workshops, 30 agreed to participate consisting of 22 joineries and 8 furniture makers. From these 30 factories, a total of 170 employees (96 joiners and 74 furniture makers) participated in the exposure survey. Repeat samples were taken from 125 employees (74%) for inhalable dust, respirable dust, or formaldehyde measurements, or any combination of these three exposures.

At the start of the work shift, as many workers as possible were asked to wear sampling pumps in the small factories; workers from medium-to-large factories were selected only if they were involved in woodwork (a proportion of workers in these medium-to-large factories do not work with wood, in contrast to smaller workshops where everyone is exposed to wood dust). All of the joineries and furniture makers were from the greater Wellington region except for one large furniture maker from Auckland and one medium-sized furniture maker from Hastings. The factories were visited between August 2008 and May 2009.

### *Dust measurements*

All samples were taken in accordance with the method described in the Australian Standard (AS 3640-2004 Workplace Atmospheres – Method for sampling and

gravimetric determination of inspirable dust). This standard specifies the sampling of the range of particle sizes that are taken in through the nose or mouth during breathing, referred to as the "inhalable" or "inspirable" fraction.

All inhalable dust samples taken were full shift (or close to full shift) personal samples collected using a pump set at an airflow of 2.0 ± 0.1 litres per minute connected with flexible tubing to an inhalable dust sampling head (PAS-6). All respirable dust samples taken were full shift (or close to full shift) personal samples collected using a pump set an airflow of 2.2 ± 0.1 litres per minute connected with flexible tubing to a respirable dust cyclone sampling head (Casella). The sampling head was located in the breathing zone of the worker being sampled. As is standard, the breathing zone was defined as a hemisphere of 300 mm radius extending in front of the face and measured from the mid-point of a line joining the ears.

Each inhalable and respirable sampling head contained a pre-weighed 25 mm Whatman glass fibre filter with a nominal pore size of 5 µm. Pumps were calibrated before and after each sampling session to check the flow rate, and the total sampling time was recorded to calculate the air volume sampled. For inhalable dust samples, 56 field blanks and 38 laboratory blanks were used for quality control. For respirable dust samples, 14 field blanks and 11 laboratory blanks were used for quality control.

Due to pump faults, filter tampering, and other exclusions, a total of 266 personal inhalable dust samples and 81 personal respirable samples were available for gravimetric analyses.

***Analysis of inhalable and respirable dust***

All sample heads were transported intact to the Centre for Public Health Research laboratory for analysis, and were weighed after allowing the filters to come to equilibrium with the balance room atmosphere.
Samples were weighed using a Mettler Toledo AX105 microbalance with a resolution of one microgram. Results are expressed as milligrams of dust per cubic metre of air ($mg/m^3$). Dust concentrations were adjusted for field blanks and those samples with

levels below the detection limit (3 respirable dust samples) were assigned a value of 0.01 mg/$m^3$.

***Formaldehyde measurements***

All formaldehyde samples taken were 15 minute personal samples collected using a pump set at an airflow of 1.5 ± 0.1 litres per minute connected with flexible tubing to a formaldehyde cartridge (Waters Sep-Pak XPoSure™ Aldehyde Sampler) containing acidified 2,4-dinitrophenylhydrazine-coated silica. The formaldehyde cartridge was located in the breathing zone (as defined above) of the worker being sampled.

Pumps were calibrated before and after each sampling session to check the flow rate, and the total sampling time was recorded to calculate the air volume sampled. For formaldehyde samples, 6 blank cartridges (and 2 blank SKC tubes) were used for quality control. Due to formaldehyde cartridges used for calibration and blanks, and exclusion of cartridges due to pump failures, a total of 274 personal formaldehyde samples were available for analyses.

***Analysis of formaldehyde cartridges***

All formaldehyde cartridges were sent to AsureQuality laboratory in Auckland for analysis. The samples were analyzed for formaldehyde by derivatisation with 2,4-di-nitrophenylhydrazine (DNPH) and subsequent high performance liquid chromatography (HPLC) with UV detection. The samples were kept refrigerated at 4°C and derivatised and extracted within three days of sample collection. Derivatised samples were analysed within three days after preparation. The limit of detection for this assay was 0.1 µg, and 23 samples had a concentration below this. These samples were assigned a value of two-thirds of the lowest measured concentration above the limit of detection i.e. 0.002 ppmv.

***Statistical analyses***

Exposures approximated to a lognormal distribution; hence exposure measurements were logarithmically transformed and presented as geometric means with geometric

standard deviations. Outlier samples with dust concentration of 50 mg/m$^3$ or higher were excluded as it was considered that these may have been tampered with. Including these samples did not significantly change the overall results.

### 6.3 Results

Table 9 shows inhalable dust exposure levels for all joinery and furniture factories separately as well as the combined exposure for all joinery workers and all furniture makers together. The geometric mean (GM) for all joineries was 2.48 mg/m$^3$ (GSD 2.57); the mean exposure for all furniture makers was approximately half that of the joinery workers (GM 1.22 mg/m$^3$; GSD 2.84). The overall average (GM) of inhalable wood dust exposure for all workers combined was 1.82 mg/m$^3$ (GSD 2.85). The relatively high geometric standard deviations (GSDs) indicate a relatively large variation of exposure levels both within individual operations and between operations. This variation in exposure levels between operations is visualised in Figure 6 which shows the GMs of each joinery and furniture maker, as well as the GM for joineries combined, furniture makers combined, and the total GM exposure for all workers. Due to the relatively low number of workers in many joineries, and in some furniture factories, the average exposures and standard deviations are often based on only a small number of measurements per operation. However, the average and geometric standard deviations for these smaller operations appear to be very similar to those reported for operations with larger number of workers (table 9).

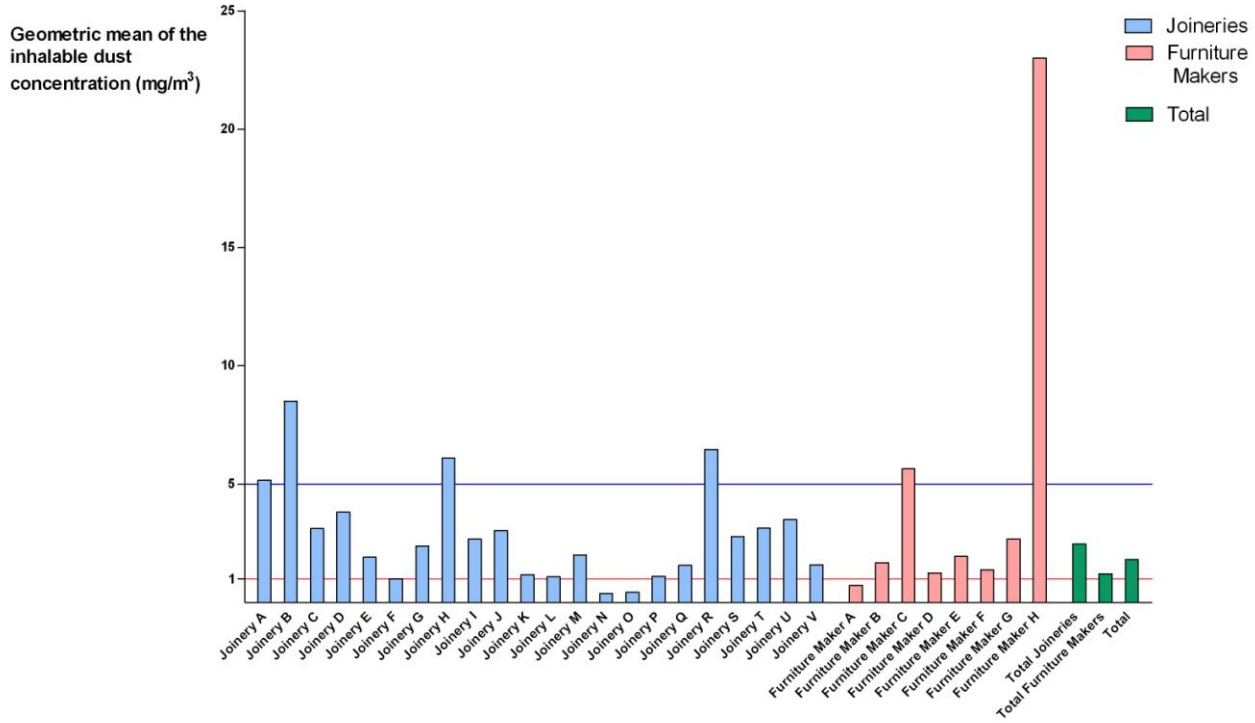
Approximately one in five (19%) joinery workers were exposed to levels exceeding the current New Zealand exposure standard of 5 mg/m$^3$ compared to approximately one in thirteen (8%) workers in furniture making factories (Table 9). Overall, 14% of all joinery and furniture workers exceeded the exposure standard of 5 mg/m$^3$. When compared to the more stringent exposure limit of 1 mg/m$^3$ recommended by the ACGIH and proposed by the Department of Labour our data show that 86% of all joinery workers and 63% of all furniture makers exceeded this exposure level, which equated to 76% of all workers measured in this study (table 9).

**Table 9. Inhalable dust concentration for joineries and furniture makers in Wellington, Hastings and Auckland**

| Type of industry | N | GM ($mg/m^3$) | GSD | Min | Max | % above 5 $mg/m^3$ | % above 1 $mg/m^3$ |
|---|---|---|---|---|---|---|---|
| Joineries | | | | | | | |
| A | 3 | 5.16 | 1.76 | 2.74 | 8.09 | 67% | 100% |
| B | 2 | 8.51 | 1.79 | 5.63 | 12.87 | 100% | 100% |
| C | 6 | 3.12 | 2.12 | 1.09 | 7.49 | 17% | 100% |
| D | 9 | 3.82 | 1.54 | 2.03 | 7.63 | 33% | 100% |
| E | 6 | 1.92 | 1.23 | 1.50 | 2.71 | 0% | 100% |
| F | 3 | 1.01 | 1.89 | 0.54 | 1.92 | 0% | 33% |
| G | 22 | 2.38 | 2.51 | 0.30 | 16.27 | 14% | 86% |
| H | 12 | 6.10 | 2.63 | 1.70 | 35.94 | 58% | 100% |
| I | 16 | 2.68 | 2.77 | 1.05 | 48.95 | 19% | 100% |
| J | 6 | 3.04 | 3.57 | 0.90 | 30.51 | 33% | 83% |
| K | 8 | 1.17 | 2.29 | 0.51 | 4.43 | 0% | 50% |
| L | 2 | 1.09 | 1.76 | 0.73 | 1.62 | 0% | 50% |
| M | 1 | 2.01 | - | - | - | 0% | 100% |
| N | 1 | 0.38 | - | - | - | 0% | 0% |
| O | 2 | 0.43 | 1.99 | 0.27 | 0.71 | 0% | 0% |
| P | 6 | 1.11 | 1.27 | 0.76 | 1.55 | 0% | 83% |
| Q | 6 | 1.57 | 1.73 | 0.78 | 2.93 | 0% | 67% |
| R | 4 | 6.47 | 2.29 | 2.53 | 15.06 | 50% | 100% |
| S | 5 | 2.78 | 1.63 | 1.63 | 4.49 | 0% | 100% |
| T | 14 | 3.14 | 2.40 | 1.53 | 47.66 | 15% | 100% |
| U | 3 | 3.50 | 1.34 | 2.69 | 4.81 | 0% | 100% |
| V | 13 | 1.59 | 2.03 | 0.36 | 6.63 | 8% | 77% |
| Furniture factories | | | | | | | |
| A | 43 | 0.72 | 3.09 | 0.05 | 7.32 | 5% | 37% |
| B | 39 | 1.68 | 2.00 | 0.36 | 8.85 | 10% | 79% |
| C | 3 | 5.65 | 2.41 | 2.57 | 14.61 | 33% | 100% |
| D | 24 | 1.25 | 2.44 | 0.15 | 7.17 | 4% | 67% |
| E | 3 | 1.96 | 1.59 | 1.29 | 3.24 | 0% | 100% |
| F | 2 | 1.38 | 1.38 | 1.10 | 1.73 | 0% | 100% |
| G | 1 | 2.69 | - | - | - | 0% | 100% |
| H | 1 | 23.00 | - | - | - | 100% | 100% |
| All joineries | 150 | 2.48 | 2.57 | 0.27 | 48.95 | 19% | 86% |
| All furniture makers | 116 | 1.22 | 2.84 | 0.05 | 23.00 | 8% | 63% |
| All workers | 266 | 1.82 | 2.85 | 0.05 | 48.95 | 14% | 76% |

GM, geometric mean; GSD, geometric standard deviation

**Figure 6. Average inhalable dust concentrations (mg/m$^3$) of joineries and furniture makers.**




OEL 1 = ACGIH OEL for wood dust is 1 mg/m$^3$; OEL 5 = Current New Zealand OEL for wood dust is 5 mg/m$^3$

Table 10 shows respirable dust exposure levels for joineries and furniture makers. As was the case with inhalable dust the highest respirable dust levels were measured in joinery workers with a geometric mean of 0.27 mg/m$^3$ (GSD 2.85) compared to less than half that (GM 0.12 mg/m$^3$ ;GSD 5.13) in furniture makers. The overall GM of respirable wood dust exposure for all workers was 0.18 mg/m$^3$. The variation in respirable dust exposure among furniture makers was considerably higher than for inhalable dust (GSD 5.13 versus GSD 2.85). The variation in respirable dust levels between operations is visualised in Figure 7 which shows the GMs of each joinery and furniture maker, as well as the GM for joineries combined, furniture makers combined, and total GM.

Using the overall dust levels, the respirable dust fraction was determined to be 11% of the total inhalable dust fraction in joinery workers and 10% in furniture workers. No occupational exposure standards have been defined for respirable wood dust, i.e. all existing national and international standards are defined based on inhalable dust as described above.

**Table 10. Respirable dust concentration for joineries and furniture makers in Wellington, Hastings and Auckland**

| Type of industry | N | GM ($mg/m^3$) | GSD | Min | Max |
|---|---|---|---|---|---|
| Joineries | | | | | |
| A | 2 | 0.27 | 1.20 | 0.23 | 0.30 |
| D | 2 | 0.15 | 1.21 | 0.13 | 0.17 |
| E | 1 | 0.29 | - | - | - |
| G | 7 | 0.33 | 1.44 | 0.23 | 0.66 |
| H | 2 | 0.30 | 1.26 | 0.25 | 0.35 |
| I | 3 | 0.86 | 12.59 | 0.16 | 15.81 |
| J | 2 | 0.92 | 3.69 | 0.37 | 2.32 |
| K | 3 | 0.53 | 1.90 | 0.30 | 1.07 |
| L | 1 | 0.31 | - | - | - |
| P | 2 | 0.21 | 1.05 | 0.20 | 0.21 |
| Q | 2 | 0.17 | 1.81 | 0.11 | 0.26 |
| T | 3 | 0.18 | 1.42 | 0.13 | 0.25 |
| U | 6 | 0.26 | 1.80 | 0.10 | 0.54 |
| V | 4 | 0.20 | 1.65 | 0.10 | 0.30 |
| Furniture factories | | | | | |
| A | 16 | 0.05 | 3.05 | 0.01 | 0.28 |
| B | 15 | 0.46 | 3.09 | 0.13 | 6.31 |
| D | 8 | 0.07 | 5.16 | 0.01 | 0.48 |
| G | 1 | 0.01 | - | - | - |
| All joineries | 41 | 0.27 | 2.85 | 0.10 | 15.81 |
| All furniture maker | 40 | 0.12 | 5.13 | 0.01 | 6.31 |
| All workers | 81 | 0.18 | 3.25 | 0.01 | 15.81 |

GM, geometric mean; GSD, geometric standard deviation

**Figure 7. Average respirable dust concentrations (mg/m$^3$) in Joineries and Furniture Makers**

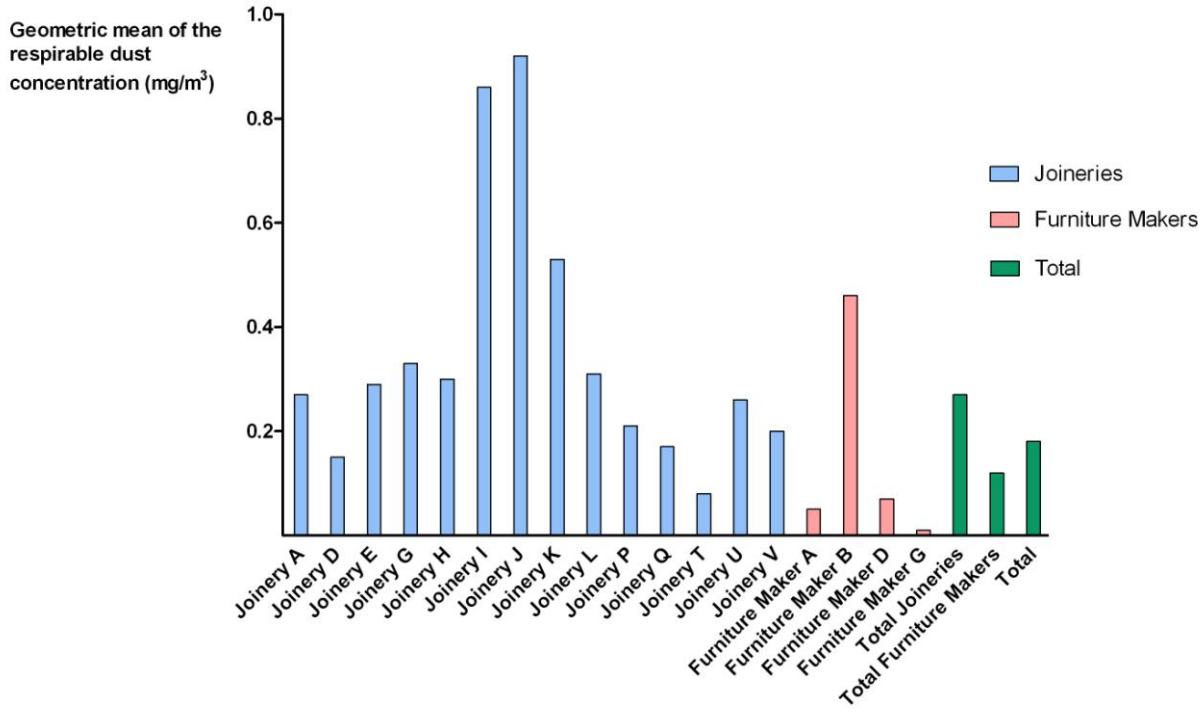


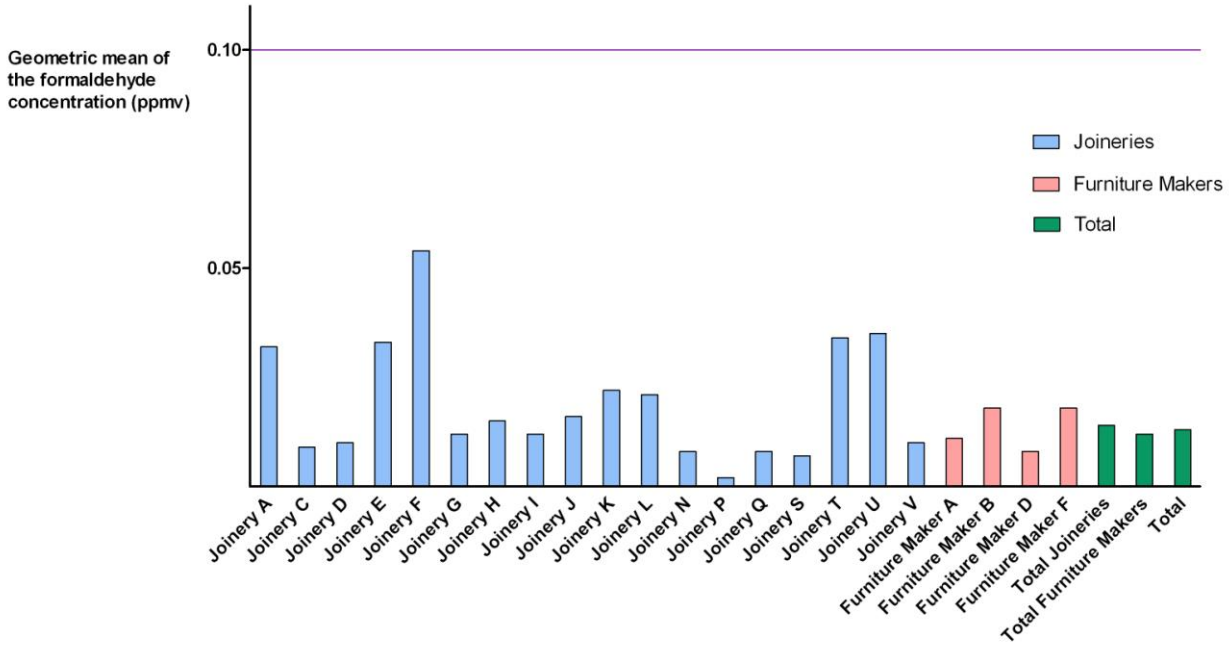

Table 11 shows formaldehyde exposure levels for joineries and furniture makers. The geometric mean formaldehyde exposures were very similar for furniture makers (0.012 parts per million per volume (ppmv); GSD 2.45) and joinery workers (0.014 ppmv; GSD 0.014). The total GM of formaldehyde exposure for all workers was 0.011 ppmv. These levels are very low, even when compared to the short term exposure limit (STEL) of 0.1 ppm recommended by NIOSH. In fact, only two samples (<1% of all samples) exceeded the level of 0.1 ppm (one furniture making and one joinery sample). Figure 8 shows the GMs of each joinery and furniture maker, as well as the GM for joineries combined, furniture makers combined, and total GM. There was no correlation between formaldehyde and dust exposures (Figure 9).

**Table 11. Formaldehyde levels (ppmv) for joineries and furniture makers in Wellington, Hastings and Auckland**

| Type of industry | N | GM (ppmv) | GSD | Min | Max |
|---|---|---|---|---|---|
| Joinery | | | | | |
| A | 6 | 0.032 | 2.20 | 0.007 | 0.071 |
| C | 11 | 0.009 | 1.77 | 0.004 | 0.024 |
| D | 6 | 0.010 | 1.70 | 0.004 | 0.018 |
| E | 3 | 0.033 | 1.31 | 0.026 | 0.044 |
| F | 6 | 0.054 | 1.11 | 0.047 | 0.061 |
| G | 23 | 0.012 | 2.21 | 0.002 | 0.040 |
| H | 5 | 0.015 | 1.23 | 0.011 | 0.018 |
| I | 8 | 0.012 | 1.32 | 0.007 | 0.016 |
| J | 3 | 0.016 | 1.14 | 0.014 | 0.018 |
| K | 10 | 0.022 | 1.83 | 0.010 | 0.050 |
| L | 3 | 0.021 | 1.36 | 0.015 | 0.026 |
| N | 3 | 0.008 | 1.26 | 0.007 | 0.011 |
| P | 6 | 0.002 | 1.23 | 0.002 | 0.004 |
| Q | 6 | 0.008 | 1.23 | 0.007 | 0.011 |
| S | 3 | 0.007 | 1.74 | 0.004 | 0.011 |
| T | 6 | 0.034 | 1.24 | 0.026 | 0.043 |
| U | 5 | 0.035 | 1.38 | 0.022 | 0.047 |
| V | 13 | 0.010 | 2.87 | 0.003 | 0.124 |
| Furniture Maker | | | | | |
| A | 40 | 0.011 | 2.08 | 0.002 | 0.120 |
| B | 49 | 0.018 | 1.95 | 0.002 | 0.048 |
| D | 52 | 0.008 | 2.69 | 0.002 | 0.069 |
| F | 7 | 0.018 | 3.01 | 0.004 | 0.084 |
| All joineries | 126 | 0.014 | 2.43 | 0.002 | 0.124 |
| All furniture maker | 148 | 0.012 | 2.45 | 0.002 | 0.120 |
| All workers | 274 | 0.013 | 2.45 | 0.002 | 0.124 |

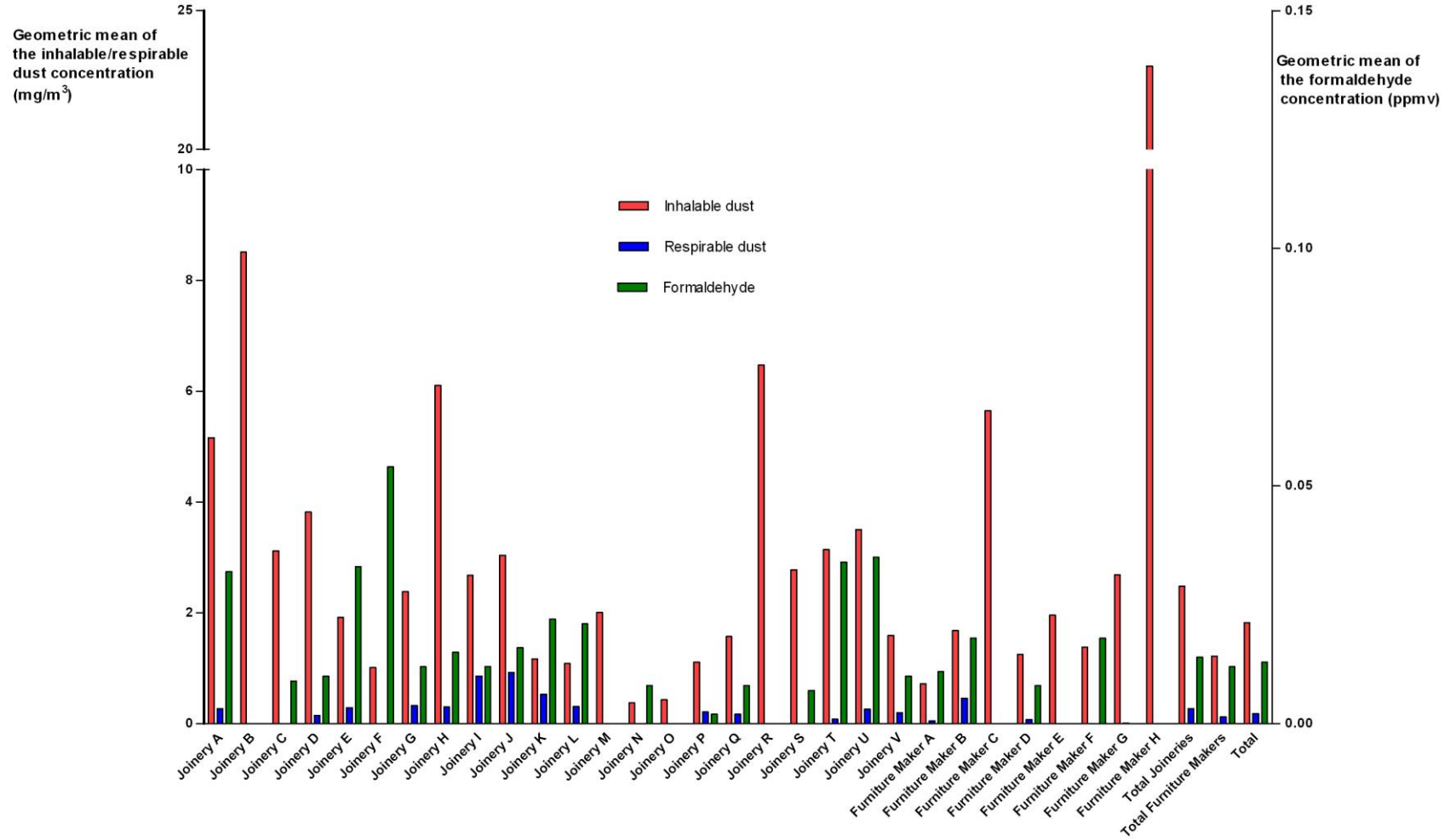
GM, geometric mean; GSD, geometric standard deviation

**Figure 8. Average formaldehyde concentration (ppmv) in joineries and furniture makers**




NIOSH: Recommended short term exposure limit (STEL) of 0.1 ppm; OSHA-PEL: Permissible Exposure Limit established by Occupational Safety and Health Administration in the US of 2.0 ppm (not shown in the graph); New Zealand WES for formaldehyde is a ceiling limit of 1.0 ppm (not shown in the graph).

**Figure 9. Average dust (inhalable and respirable) and formaldehyde concentration in joineries and furniture makers.**



## 6.4 Discussion and interpretation of study results

Our exposure study showed that a significant proportion of joinery and furniture workers (19% and 8% respectively) are exposed to levels in excess of the current New Zealand occupational exposure limit of 5 mg/m$^3$. More than 75% of all workers were exposed to levels in excess of current international standards of 1 mg/m$^3$. Exposure levels in furniture makers were about half those of joinery workers. Our study also showed that exposures to formaldehyde were low in both joinery and furniture workers.

### *Inhalable dust*

In our study, exposure levels for joinery workers were substantially higher than those reported in a large EU study (Kauppinen *et al*., 2006) which estimated occupational exposure to inhalable wood dust by industry categories in 25 European Union (EU) member states. In our study, 87% of workers were exposed to 1 mg/m$^3$ compared to 59% in the EU, and 19% exposed to 5mg/m$^3$ versus 12.6% in the EU. For furniture workers our data showed a higher proportion of workers exposed to >1 mg/m$^3$ compared to workers in the EU (63% versus 52%), whereas at the higher exposure level (>5 mg/m$^3$) this was reversed with a lower proportion of workers exposed to >5 mg/m$^3$ in our study compared to the EU study (i.e. 8% versus 12.2%). Mean inhalable dust exposures in Australian joinery workers (n=66) (Mandryk *et al.,* 1999) were higher than those measured in our study (3.68 mg/m$^3$ versus 2.49 mg/m$^3$). However, it should be noted that the study in Australia was conducted more than 10 years ago and similar to trends in other countries (e.g. Schlunssen *et al.,* 2008) exposure levels may have reduced in more recent years. Some evidence for reduced exposures in New Zealand is shown by our data. In particular, exposure levels measured in furniture makers in the current study were significantly lower (1.22 mg/m$^3$) than levels measured in the only other study in New Zealand furniture makers which reported a mean exposure level of 3.6 mg/m$^3$ based on 64 personal measurements in 11 furniture factories (Norrish *et al.,* 1992). This suggests that exposure levels may have dropped almost three-fold in the furniture making industry over the past 15-20 years, possibly due to improved measures to reduce airborne exposures. Although this is plausible, given international trends for reduced wood dust exposures, these differences may

also in part be attributable to other factors including differences between factories that were surveyed in 1992 and those surveyed more recently.

Despite the potential reduction in dust exposure levels in more recent years, levels are generally still high. In particular, mean exposure levels to inhalable wood dust measured in joinery and furniture workers were considerably higher (2.49 mg/m$^3$ and 1.22 mg/m$^3$, respectively) than those previously measured in other parts of the wood conversion industry. For example, in sawmill workers, using identical methods, we have previously (measurements were taken in 2000) shown geometric mean wood dust exposure of 0.52 mg/m$^3$ (GSD 2.66; n=183) (Douwes *et al.*, 2006). Also, international studies have shown that geometric mean levels of less than 1 mg/m$^3$ are achievable for the furniture making industry as demonstrated in a recent study from Denmark which showed a geometric mean exposure level of 0.60 mg/m$^3$ based on 1,355 dust measurements collected from 1,044 workers (Schlunssen *et al.*, 2008).

The same authors also identified a number of activities and/or work processes that were associated with significantly increased or decreased dust exposures. Sanding, use of compressed air, use of fully-automated machines, manual work, cleaning with compressed air, kitchen manufacturing, and small size (<20 employees) were found to significantly increase inhalable dust levels, whereas manual assembling/packing, sanding with adequate exhaust ventilation, vacuum cleaning of machines, and professional cleaning staff were associated with significant decreases in dust exposures. The fact that most of New Zealand's joinery and furniture making industry is small scale (<20 employees) may have contributed to the relatively high exposure levels measured in our study compared to those measured in Denmark.

***Respirable dust***

We have measured respirable dust to assess the fraction of dust that is able to penetrate deep into the respiratory system (as opposed to the non-respirable fraction of inhalable dust which is predominantly trapped in the nose, throat and upper respiratory tract). We found that the respirable fraction accounted for 9.9% of the inhalable dust mass. This is very similar to what other studies have shown. For example, a study in the North American wood processing industry (predominantly

furniture and cabinet makers) measured geometric mean exposure levels (n=2363) for inhalable and respirable dust of 1.45 mg/m$^3$ (GSD 2.7) and 0.18 mg/m$^3$ (GSD 2.6) respectively (Glindmeyer *et al.,* 2008). Not only was the mean exposure to respirable dust comparable (0.18 mg/m$^3$ in both studies), but the proportion respirable versus inhalable dust was also very similar i.e. 9.9% versus 12.4%.

Similarly, in Australian joinery workers respirable dust accounted for 13% of the inhalable dust mass (Mandryk et al., 1999). Respirable dust levels in Australian joinery workers (0.48 mg/m$^3$; GSD 1.70) were also higher than those measured in our survey of New Zealand joinery workers (0.27 mg/m$^3$; GSD 2.85). Although respirable dust can penetrate into the deeper airways - where it has the potential to cause more severe health effects - there is currently no indication that malignant or non-malignant health risks should be evaluated on the basis of respirable rather than inhalable dust exposures. Hence, no occupational exposure limit exists for respirable dust. Also, most studies showing dose-response associations between wood dust exposure and health effects have measured inhalable dust rather than respirable dust. Therefore, any conclusions as to the potential risks associated with measured dust levels (see below) will have to be based on the inhalable dust fraction (which includes the respirable dust fraction).

***Formaldehyde***

Formaldehyde levels in our study were very low, with a geometric mean of 0.012 ppm for all joinery and furniture workers combined. Similarly low mean levels (0.05 ppm) have been reported in Australian furniture makers (Pisaniello *et al.,* 1991) and in Canadian cabinet makers (0.06 ppm) (Sass-Kortsak *et al.,* 1986). These studies suggest that formaldehyde exposures are not a major issue in the joinery and furniture making industry when using reconstituted wood products.

***Health risks***

As discussed extensively in the first section of this report, the most serious health effect of exposure to wood dust is the risk of nasal and sino-nasal cancers. There is also evidence of increased risks of lung cancer in workers with high exposure to wood

dust, and preliminary analyses of data from our study of "Occupational Lung Cancer in Adult New Zealanders" confirm this (unpublished data). Of more immediate concern are the non-malignant respiratory effects of wood dust exposure, and these have been shown to occur at levels of exposure well below those considered to increase the risk of malignant effects**.** In particular, a large number of studies in a wide range of wood working occupations have shown strong associations between wood dust exposure and respiratory disease including upper and lower respiratory tract symptoms and inflammation, impaired lung function, increased bronchial responsiveness and occupational asthma.

Studies in joinery and furniture workers confirm this picture (see Section 1), including one small study conducted in New Zealand (Norrish *et al*., 1992). These effects have been shown at levels well below the current New Zealand occupational exposure level of 5 $mg/m^3$, and effects have even been shown to occur at levels below international standards of 1 $mg/m^3$. Thus, given that the majority (>75%) of New Zealand joinery workers and furniture makers are exposed to levels in excess of 1 $mg/m^3$ with a significant proportion (14%) being exposed in excess of 5$mg/m^3$ we believe that a large proportion of these workers are at risk of developing respiratory disease. Although it is encouraging that exposure levels appear to have reduced in the past 10-20 years, they remain unacceptably high. In light of the potential risk of respiratory disease associated with these exposures, and given that the ACGIH maximum exposure level of 1$mg/m^3$ has been proposed to be adopted by New Zealand in the near future, cost effective workplace interventions to reduce wood dust exposures to below this level should be developed urgently.

### 6.5 Determinants of exposure

In addition to the descriptive statistics of the distribution of exposures in the joinery and furniture manufacturing sectors, we applied further statistical analyses to examine associations between a number of workplace and work process variables and levels of exposure and also tested interaction effects between these variables. These analyses were conducted separately for the joineries and the furniture manufacturing plants.

At the time that sampling was conducted information on a number of exposure variables related to the material being processed, specific job tasks being performed and the use of specific control measures was recorded, as was the total time spent on each variable. The specific exposure variables recorded were:

Material being processed:

- MDF
- Timber
- Steel and laminate

Job tasks being performed:

- Assembly
- Cleaning
- Sanding
- Sawing
- Machining
- Finishing

Control measures used:

- Local exhaust ventilation
- Extraction to a bag filter
- Use of personal protective equipment

Using log-transformed exposure data we first conducted univariate analyses to find which variables were associated with exposure. We then conducted stepwise model building using mixed effects models starting with the variable that explained the most variance, then adding other significant variables until no further improvement in the model fit could be achieved. The statistical significance of each model was established using the Likelihood Ratio test. In a final analysis we tested for various interaction effects between exposure variables.

The results of the mixed effects model in furniture manufacturing plants is shown in Table 12 below:

| Mixed Effects Model – Furniture Manufacturing | | | |
|---|---|---|---|
| **Model variables (fixed effects** | **Estimate (β)** | **SE** | **p>(t)** |
| **Intercept** | **0.47** | **0.43** | **0.28** |
| **Cleaning** | **-0.12** | **0.22** | **0.57** |
| **Assembly** | **0.31** | **0.24** | **0.19** |
| **Sanding** | **0.76** | **0.25** | **<0.01** |
| **Sawing** | **0.21** | **0.21** | **0.29** |
| **Machining** | **-0.33** | **0.23** | **0.15** |
| **Finishing** | **0.51** | **0.23** | **0.03** |
| **Use of LEV** | **0.57** | **0.22** | **0.01** |
| **Use of Extract2Bag Filter** | **0.99** | **0.45** | **0.03** |
| **Use of Extract2Bag Filter*Sawing** | **3.79** | **1.01** | **<0.001** |
| | | | |
| **Between-workers variance** | | **0.68** | |
| **Within-workers variance** | | **0.16** | |
| **Total explained variance** | | **36.59%** | |

In these analyses the intercept represents the level of exposure without performing any of these tasks. The model explained 37% of the variance observed, and shows that the variance between workers performing different tasks is much greater that the variance within individual workers from day to day. It is clear from this table that in the furniture manufacturing plants sampled, the processes of sanding and finishing were the most consistent contributors to exposure by 2.14 mg/m³ with lower and upper confidence limits of 1.31 and 3.49) and 1.67 (1.06-2.61) mg/m³ respectively. Cleaning and machining actually reduced exposure by 0.89 mg/m³ and 0.72 mg/m³ respectively, although in both cases the reduction was not statistically significant. Interestingly the use of both local exhaust ventilation and extraction to a bag filter were both significant contributors to the level of exposure, by 1.77 (1.15 – 2.72) mg/m³ and 2.69 (1.11 – 6.50) mg/m³ respectively, although this probably represents a selection effect as they are both used in operations that increase dust emissions and

the increase in exposure would be far greater if these control measures were not applied. The most significant interaction effect observed was for sawing while using extraction to a bag filter, which resulted in a very high contribution of 44.26 (6.11 – 320.41) mg/m³.

These analyses indicate that interventions targeted at sanding and finishing processes are likely to be most effective at reducing current levels of exposure, as would interventions aimed at controlling the release of dust from bag filters while sawing.

The results from the sampling of joineries, which on average had double the levels of exposure measured in furniture manufacture, are less reassuring with respect to the information provided to guide the targeting of control measures. The results of the mixed effects model in joineries is shown in Table 13 below:

| **Mixed Effects Model – Joineries** | | | |
|---|---|---|---|
| **Model variables (fixed effects** | **Estimate (β)** | **SE** | **p>(t)** |
| **Intercept** | **0.39** | **0.31** | **0.21** |
| **Cleaning** | **0.38** | **0.20** | **0.05** |
| **Assembly** | **0.30** | **0.19** | **0.88** |
| **Sanding** | **0.33** | **0.18** | **0.07** |
| **Sawing** | **0.27** | **0.19** | **0.17** |
| **Machining** | **0.20** | **0.18** | **0.28** |
| **Finishing** | **-0.02** | **0.19** | **0.94** |
| **Use of LEV** | **-0.22** | **0.19** | **0.23** |
| **Use of Extract2Bag Filter** | **-0.36** | **0.34** | **0.29** |
| | | | |
| **Between-workers variance** | | **0.31** | |
| **Within-workers variance** | | **0.74** | |
| **Total explained variance** | | **8.05%** | |

For joineries the model fit was much poorer, explaining only 8% of the variance observed, and no clear pattern emerged with cleaning and sanding being the only marginally significant contributors to exposure at 1.46 (0.99 – 2.16) mg/m³ and 1.39 (0.98 – 1.98) mg/m³ respectively. Both the use of local exhaust ventilation and

extraction to a bag filter reduced exposure, but this was not statistically significant. This indicates that the working environment in joineries was less well controlled than that in furniture making, and further evaluation using video exposure monitoring to identify the peaks of dust emission that are likely to constitute the main exposure source will be required before interventions can be trialled.

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