

sampled (except Joinery I, where a randomly selected number of workers from each job title were sampled). For respirable dust sampling, random sampling of each job title was carried out. The sampling was conducted according to the specifications of Standards Australia [1987, 1989]. Polycarbonate filters (25 mm, 0.8 μm , Millipore, Ventura, CA) were used as the collection media since high extractability of endotoxins using such filters has been reported by previous studies [Douwes et al., 1995].

Endotoxin and (1 \rightarrow 3)- β -D-Glucan

After weighing, the sample filters were extracted with endotoxin/glucan-free water [Olenchok, 1990]. The supernatant was analyzed separately for endotoxin and glucan using quantitative endpoint chromogenic limulus assay [Obayashi, 1990] using endotoxin-specific (endospey, standard endotoxin — *E coli* 0111:B4 (Westphal), Seikagaku Co., Tokyo) and glucan-specific lysates (gluspey, standard (1 \rightarrow 3)- β -D-Glucan — pachyman, Seikagaku Co., Tokyo).

Microorganisms

Personal samples of airborne bacteria and fungi were collected using presterilized three-piece cellulose ester membrane filter cassettes (37 mm, 0.45 μm , Millipore) connected to a constant flow personal pump calibrated to 1.5 L/min. The duration of sampling was 4–6 hr. Microorganisms were extracted from the collected filter cassettes using a suspension fluid (0.1% bacteriological peptone with 0.05% Tween 80 and 2% inositol) as described by Eduard et al. [1990]. Serial dilutions of the suspension were then prepared using one-fourth strength Ringer's solution (Oxoid, UK) and 0.1 ml of the dilutions were plated in different media. The plates were incubated at two temperatures (25°C and 40°C). Media used for the isolation of fungi were 2% malt extract agar; for xerophilic fungi, dichloran-glycerol agar (Oxoid, UK); and for Gram-negative bacteria, a selective medium, violet red bile glucose agar (Amyl Media, Australia).

Lung Function Test

Lung function testing followed the guidelines given by the American Thoracic Society [1979] for measuring respiratory function. The Vitalograph Alpha portable spirometer (serial no: AL 06993, Vitalograph Ltd., UK) was used. The measurement of expired air was made on the Vitalograph-Alpha using a Fleisch type pneumotach while the attached microprocessor displayed the data on the screen.

The vital capacity and forced vital capacity tests of workers were conducted before and after a workshift. All the workers at each worksite were tested for lung function and monitored for dust on the same workshift. The spirometer

was calibrated with a 1 L precision syringe (cat. no. 20.408, Vitalograph) before testing. Each worker was requested to perform 3–5 attempts, until it appeared that maximum effort was obtained. The “best test” (highest FVC+FEV₁) was recorded as the lung function capacity of the worker. For each worker, age, height, number of years of exposure to wood dust, ethnic origin, and smoking status were also recorded. The maintenance workers at the woodworking sites were used as controls as their ethnic and social backgrounds were similar to the woodworkers. The job tasks of the maintenance workers did not involve wood dust exposure under normal circumstances. The lung function data of workers having a past history of asthma were not included in the data analyses. The data of the female workers were also not included in the data analyses. The two workers having mild asthma at Joinery I (processing western red cedar) did not participate in the lung function test.

Questionnaire

The Rylander et al. [1990] “Organic Dust Questionnaire” together with appropriate questions on respiratory, eye, and nasal symptoms from the British Medical Research Council's [1960] respiratory questionnaire were used to obtain symptom prevalence data among woodworkers and the control group.

Statistical Analysis

Stepwise multiple regression was used to develop models for prediction of pulmonary function changes from independent variables (using Microsoft Excel, Ver. 5.0, Microsoft Co., Redmond, WA, USA). Cross-shift change in each pulmonary function variable (VC, FVC, FEV₁, FEV₁/FVC, PEF, FEF_{25–75%}) was treated individually as the dependent variable. Independent variables tested in the regression models included age, height, smoking, number of years of exposure to wood dust, and personal exposure data for inhalable dust, respirable dust, inhalable endotoxin, respirable endotoxin, inhalable glucan, and respirable glucan. As the number of exsmokers was few, they were considered nonsmokers. The mean percentage cross-shift changes in lung function were compared with controls (unpaired *t*-test).

The effects of personal exposures on percentage cross-shift changes in lung function and percentage predicted lung function were computed by linear correlation analyses (Pearson's R). Predicted normal values were calculated using the formulae of Gibson et al. [1979] for FVC, FEV₁, FEV₁/FVC, and of Lazarus [1982] for VC and FEF_{25–75%}. The percentage predicted values were compared with controls using unpaired *t*-test. Personal exposure data were log-normally distributed and, hence, the natural logarithms of exposure (GM values) were used for regression and

correlation analyses (GraphPad InStat, Ver. V2.04a, San Diego, CA, USA). The SPSS statistical program was used for the questionnaire analysis (SPSS for Windows, Ver. 6.1.3, SPSS Inc., Chicago, IL, USA). Work-related symptoms were adjusted for age and smoking by logistic regression analysis. Relationships among work-related respiratory symptoms and lung function indices were computed by linear regression analyses.

RESULTS

The mean exposure levels of dust, endotoxins, (1 → 3)-β-D-glucans, bacteria, and fungi are given in Table II. Overall, 62% of the personal inhalable dust exposures exceeded the current standards (hardwood: 1 mg/m³, softwood: 5 mg/m³ [Worksafe Australia, 1995]). Among joineries, 95% of the hardwood exposures and 35% of the softwood exposures exceeded the above standards. The wood dust exposures by job titles at different worksites and determinants of wood dust exposure have been described [Alwis et al., 1999b]. The geometric mean dust exposure level at Joinery I was much lower (0.60 mg/m³) than the occupational exposure limit of 5 mg/m³ for softwood. Joinery I processed western red cedar, which is known to cause asthma and rhinitis among such exposed populations [Enarson and Chan-Yeung, 1990].

High levels of endotoxins and (1 → 3)-β-glucans were found in the inhalable fraction compared with the respirable fraction. Some of the personal inhalable exposure levels of endotoxins (Sawmills C, E, F, and Joinery H) exceeded the threshold limit value of 20 ng/m³ [Rylander, 1990].

Effects on Percentage Predicted Lung Function (Chronic Effects)

The woodworkers had low percentage predicted lung functions compared with controls (Table III). The effects of personal exposures on the percentage predicted lung function indices were more pronounced among joinery workers compared with the sawmill and chip mill workers (Table IV). For both joinery workers and sawmill and chip mill workers, the percentage predicted lung function indices were positively correlated with the number of years of exposure to wood dust. The most probable reasons for this might be that some workers developed tolerance to high dust exposure after working for a number of years with wood, possibly leading to the “healthy worker effect,” where those workers who were sensitive to wood dust had left the jobs.

Effects on Cross-Shift Change (Decrease) in Lung Function (Acute Effects)

The mean percentage cross-shift decrease in lung function adjusted for age, height, smoking, number of years

of exposure to wood dust, and personal exposures was significantly high in both joinery workers and sawmill and chip mill workers compared with controls (Table V). Sawmill and chip mill workers had a high mean percentage cross-shift decrement in FEV₁ adjusted for number of years of exposure to wood dust (15%) compared with that of joinery workers (4%). An 11% mean cross-shift change in FEF_{25–75%} adjusted for inhalable endotoxin was also observed among sawmill and chip mill workers. Joinery workers had a high percentage of cross-shift changes in VC (10%) and FEV₁ (15%) adjusted for respirable endotoxin levels compared with those of sawmill and chip mill workers. Effects of inhalable and respirable (1 → 3)-β-D-glucan on the percentage cross-shift decrements in lung function were more prominent among sawmill and chip mill workers compared with those of joinery workers. The total group of woodworkers (joinery workers plus sawmill and chip mill workers) had marked effects of inhalable dust exposures on mean percentage cross-shift change in VC (29%), respirable dust exposure on mean percentage cross-shift change in FEV₁/FVC (15%) and inhalable endotoxin exposures on mean percentage cross-shift change in FVC (38%) compared with those of controls. The effect of all the personal exposures on cross-shift decrements in lung function was more prominent among sawmill and chip mill workers compared with joinery workers. Similar to the effects on percentage predicted lung function, the effects of dust, endotoxin, Gram-negative bacteria, and (1 → 3)-β-D-glucan on cross-shift decreases in lung function were more prominent among the joinery workers compared with the sawmill and chip mill workers (Table VI). The cross-shift decrements in VC and FEF_{25–75%} were positively correlated with the number of years of exposure to wood dust among both joinery workers and sawmill and chip mill workers.

Prevalence of Work-Related Symptoms

Table VII presents the prevalence of work-related respiratory symptoms among woodworkers and controls. Woodworkers had a markedly high prevalence of regular cough, phlegm, and chronic bronchitis (persistent cough and phlegm for more than 3 months per year for more than 2 years) compared with controls. The prevalence of regular phlegm, wheezing, and regular blocked nose was significantly high among joinery workers compared with sawmill and chip mill workers. Particle-size distribution studies have shown that the major portion of wood dust is contributed by particles larger than 10 μm in diameter, which can be easily trapped in the nasal passages [Hinds, 1988; Pisaniello et al., 1991]. The dose–response relationships between personal exposures and work-related symptoms among these woodworkers have been described [Alwis et al., 1999a].

TABLE III. Percentage Predicted Lung Function Among Woodworkers, Australia (1996–1997)

	Joinery workers	Sawmill/chip mill workers	Total	Control
No.	63	105	168	30
Age (yr)	34 ± 13.6	38 ± 12.1	37 ± 12.8	39 ± 11.7
Height (cm)	176 ± 9.3	175 ± 7.8	176 ± 8.2	175 ± 7.8
Years exposed	14 ± 13.1	9 ± 8.3	11 ± 10.6	11 ± 7.7
Smoking status	30%	40%	33%	30%
% Predicted lung function ^a				
VC	83.95 ± 3.02 (0.0001) ^b	83.16 ± 3.15 (0.0001)	83.45 ± 3.12 (0.0001)	95.35 ± 7.93
FVC	84.65 ± 1.43 (0.0001)	84.68 ± 1.05 (0.0001)	84.66 ± 0.72 (0.0001)	94.90 ± 3.85
FEV ₁	84.59 ± 0.82 (0.0001)	84.71 ± 0.65 (0.0001)	84.67 ± 1.20 (0.0001)	93.11 ± 2.81
FEV ₁ /FVC	99.61 ± 0.64 (0.0096)	99.82 ± 0.72 (ns)	99.74 ± 0.70 (ns)	99.90 ± 0.34
FEF _{25–75%}	100.49 ± 8.97 (ns)	101.23 ± 7.00 (ns)	100.96 ± 7.76 (ns)	101.63 ± 5.17

^a% predicted lung function = % observed/predicted (adjusted for age, height, and smoking).

^b(*P* values, ns = not significant).

TABLE IV. Dose–Response Relationships (Pearson's *R*) Between Percentage Predicted Lung Function Indices and Personal Exposures (log transformed), and No. of Years of Exposure (yr), Australia (1996–1997)

Group of workers ^a	% Predicted lung function	Inhalable dust	Respirable dust	Inhalable endotoxin	Respirable endotoxin	Gram-negative bacteria	Respirable glucan	No. of years of exposure
Joinery	FEV ₁	−0.65***	−0.41***	−0.60***	−0.55***	−0.66***	0.28*	0.77***
Sawmill/chipping		−0.26**	ns	ns	ns	ns	ns	0.49***
Total		−0.30***	−0.27***	−0.33***	ns	−0.24**	0.18*	0.59***
Joinery	FVC	−0.57***	−0.36**	−0.52***	−0.48***	−0.58***	ns	0.59***
Sawmill/chipping		ns	ns	ns	ns	ns	ns	0.38***
Total		−0.27***	−0.19*	−0.30***	ns	−0.26***	ns	0.48***
Joinery	FEV ₁ /FVC	−0.34**	ns	−0.31*	−0.28*	−0.64***	ns	0.56***
Sawmill/chipping		0.22*	ns	ns	ns	ns	0.19*	0.27**
Total		ns	−0.20**	ns	ns	−0.24**	0.24**	0.34***
Joinery	FEF _{25–75%}	−0.63***	−0.40**	−0.58***	−0.53***	−0.32*	0.27*	0.73***
Sawmill/chipping		ns	ns	ns	ns	ns	ns	0.48***
Total		−0.29***	−0.24**	−0.32***	−0.16*	0.26***	ns	0.58***

^aJoinery: *n* = 63, sawmill/chip mill: *n* = 105, total: *n* = 168.

**P* < 0.05.

***P* < 0.01.

****P* < 0.001; ns, not significant.

Significant positive correlations were found between respiratory symptoms and percentage cross-shift change (decrease) in lung function among total woodworkers (Table VIII). Regular phlegm and blocked nose were positively correlated with the percentage cross-shift decrease in FVC among sawmill and chip mill workers. Both joinery workers and sawmill and chip mill workers showed significant inverse relationships between respiratory symptoms and percentage predicted lung function (FVC, FEV₁, FEV₁/FVC, FEF_{25–75%}) (Table IX). The total group of woodworkers showed significant correlations between percentage

predicted lung function (FVC, FEV₁, FEV₁/FVC, FEF_{25–75%}) and phlegm and bronchitis.

DISCUSSION

The correlations found between lung function indices and personal exposures indicated that airborne wood dust and biohazards associated with wood dust (endotoxins, (1 → 3)-β-D-glucans, fungi, and Gram-negative bacteria) have negative effects on the pulmonary function of woodworkers. The cross-shift decrements in lung function were

TABLE VI. Dose–Response Relationships (Pearson’s R) Between Percentage Cross-Shift Change (Decrease) in Lung Function Indices and Personal Exposures (log transformed) and No. of Years of Exposure to Wood Dust (yr), Australia (1996–1997)

Group of workers ^a	% Decrease in lung function ^b	% Decrease						No. of years of exposure
		Inhalable dust	Respirable dust	Inhalable endotoxin	Respirable endotoxin	Gram-negative bacteria	Respirable glucan	
Joinery	VC	−0.66***	−0.42***	−0.61***	−0.56***	−0.31*	0.28*	0.81***
Sawmill/chipping		0.26**	ns	ns	ns	ns	0.20*	0.48***
Total		−0.29***	−0.28***	−0.32***	ns	ns	0.23**	0.59***
Joinery	FEV ₁	0.55***	0.35***	0.51***	0.47***	0.54***	ns	−0.77***
Sawmill/chipping		−0.26**	ns	ns	ns	ns	−0.21*	−0.39***
Total		0.21**	0.25***	0.24**	ns	ns	−0.24**	−0.50***
Joinery	FVC	0.67***	0.42***	0.61***	0.56***	0.67***	−0.28*	−0.80***
Sawmill/chipping		−0.25**	ns	ns	ns	ns	−0.20*	−0.48***
Total		0.29***	0.28***	0.33***	ns	0.23**	−0.22**	−0.60***
Joinery	FEF _{25–75%}	−0.33**	ns	−0.31*	−0.28*	−0.66***	ns	0.57***
Sawmill/chipping		0.22*	ns	ns	ns	ns	0.19*	0.28**
Total		ns	−0.20*	ns	ns	−0.22**	0.23**	0.35***
Joinery	PEF	0.43***	0.27*	0.39**	0.36***	0.41***	ns	−0.32***
Sawmill/chipping		−0.25*	ns	ns	ns	ns	−0.21*	−0.65**
Total		0.16**	0.22**	0.19*	ns	ns	−0.24**	−0.40**

^aJoinery: n = 63, sawmill/chip mill: n = 105, total: n = 168.

^bLung function indices were adjusted to age, height, smoking by multiple linear regression analyses. % Change (decrease) in FEV₁ = [FEV_{1(morning)} − FEV_{1(afternoon)}/FEV_{1(morning)}] × 100.

*P < 0.05; **P < 0.01; ***P < 0.001; ns = not significant.

also positively correlated with the number of years of exposure to wood dust. The lung function changes observed among woodworkers were obstructive in nature.

Woodworkers had significantly high prevalence of regular cough, phlegm, chronic bronchitis, and regular blocked nose compared with controls. The high personal airborne dust exposure levels observed, and workers not wearing respirators, might have contributed to this high prevalence of respiratory and nasal symptoms. Overall, only 10% of the workers used appropriate respirators and, among them, respirators were worn on average less than 50% of the time during a workshift. The South Australian Study [Pisanello et al., 1991] also reported a high prevalence of nasal symptoms among furniture workers, where the geometric mean exposure level was 2.9 mg/m³ (range: 0.4–24 mg/m³). The prevalence of bronchitis was high among smokers (20%) compared with nonsmokers (10%). Previous studies also reported high prevalence of chronic bronchitis among woodworkers [Enarson and Chan-Yeung, 1990; Li et al., 1990; Liou et al., 1996].

Significant positive correlations were found among percentage cross-shift decreases in lung function and respiratory symptoms. Respiratory symptoms were inversely correlated with percentage predicted lung function among joinery workers and sawmill and chip mill workers.

Wood dust and biohazards associated with wood dust are potential health hazards in the wood processing industry.

TABLE VII. Percentage Prevalence of Work-Related Respiratory Symptoms^a Among Woodworkers and Controls, Australia (1996–1997)

	Sawmill and chipmill	Joinery	Woodworkers ^b	Controls
No.	108	82	195	34
Age (yr) ^c	38 ± 12.8	36 ± 14.7	37 ± 13.6	40 ± 11.3
Years exposed (yr) ^c	8 ± 8.7	12 ± 12.1	10 ± 10.5	9 ± 7.7
Smoking	40%	23%	33%	30%
Symptoms				
Asthma	8.3	13.4	10.8	5.9
Dry cough	24.1	23.2	23.1	23.5
Regular cough	58.6***	62.2***	61.0***	23.5
Regular phlegm	46.0*	67.1***	61.5***	23.5
Chronic bronchitis	29.6*	31.7	30.3*	11.8
Breathlessness	18.5	18.3	17.4	14.7
Wheezing	15.7	32.9*	22.6	11.8
Chest tightness	21.3	36.6	27.2	17.6
Dyspnea	2.8	7.3	4.6	0.0
Regular blocked nose	46.3*	63.4***	42.6	29.4

^aAdjusted for age and smoking by logistic regression.

^bIncluding workers at logging sites.

^cMean ± SD.

*P < 0.05, **P < 0.01, ***P < 0.001 (chi-square analysis; compared with controls).

TABLE VIII. Significant Correlations Between Percentage Cross-Shift Change (Decrease) in Lung Function and Respiratory Symptoms Among Joinery Workers and Sawmill and Chip Mill Workers, Australia (1996–1997)

Group of workers ^a	% Cross-shift decrease in lung function ^b	Respiratory symptom	(r) ^c	P-value
Total	FVC	Regular phlegm	0.18	0.012
		Chronic bronchitis	0.14	0.046
Sawmill/chip mill		Regular phlegm	0.19	0.049
		Blocked nose	0.19	0.046
Total	FEV ₁	Chronic bronchitis	0.14	0.047
		Regular phlegm	0.17	0.017
Total	FEV ₁ /FVC	Chest tightness	0.15	0.045
		Dyspnea	0.15	0.034
Total	PEF	Regular phlegm	0.15	0.037

^aTotal: n = 168, sawmill/chip mill: n = 105.

^bAdjusted for age, height, and smoking.

^cCorrelation coefficient.

TABLE IX. Significant Correlations Between Percentage Predicted Lung Function and Respiratory Symptoms, the Study of Woodworkers in Australia (1996–1997)

Group of workers ^a	% Predicted lung function ^b	Respiratory symptom	(r) ^c	P-value
Joinery	FVC	Regular phlegm	-0.22	0.045
		Blocked nose	-0.32	<0.001
Sawmill/chip mill		Regular phlegm	-0.17	0.018
		Chronic bronchitis	-0.14	0.046
Total	FEV ₁	Regular phlegm	-0.23	0.038
		Blocked nose	-0.26	0.007
Joinery	FEV ₁ /FVC	Regular phlegm	-0.18	0.011
		Chronic bronchitis	-0.16	0.024
Sawmill/chip mill		Regular phlegm	-0.26	0.020
		Dyspnea	-0.20	0.038
Total	FEF _{25-75%}	Regular phlegm	-0.20	0.040
		Regular phlegm	-0.15	0.036
Joinery		Chronic bronchitis	-0.15	0.039
		Regular phlegm	-0.22	0.050
Sawmill/chip mill		Blocked nose	-0.20	0.037
		Dry cough	-0.20	0.043
Total		Regular phlegm	-0.20	0.039
		Regular phlegm	-0.19	0.008
Total		Chronic bronchitis	-0.15	0.043

^aJoinery: n = 63, sawmill/chip mill: n = 105, total: n = 168.

^bAdjusted for age, height, and smoking by multiple regression.

^cCorrelation coefficient.

However, using proper dust extraction systems, occupational exposure to wood dust and the biohazards associated with wood dust can be prevented or minimized. When the exposure cannot be fully controlled, workers should be provided with appropriate personal protective equipment. Workers should also be educated on the potential health effects of wood dust exposure.

ACKNOWLEDGMENTS

The authors are grateful to the management and the employees of the companies for participating in this study. We thank Mr. Jim Morton, the Timber Trade Industrial Association (TTIA, NSW), and Mr. Warren Baker, the Construction, Forestry, Mining and Energy Union (CFMEU, NSW) for facilitating access to companies. We also thank Mr. John Lee and Mr. Trevor Mayhew, the WorkCover Authority, NSW, for their continuous support, assistance, and advice.

REFERENCES

- Acheson ED, Cowdell RH, Hadfeld E, Macbeth RG. 1968. Nasal cancer in woodworkers in the furniture industry. *Br Med J* 2:587–596.
- Al Zuhair YS, Whitaker CJ, Cinkotai FF. 1981. Ventilatory function in workers exposed to tea and wood dust. *Br J Ind Med* 38:339–345.
- Alwis KU, Mandryk J, Hocking AD. 1999a. Exposure to biohazards in wood dust — bacteria, fungi, endotoxins and (1 → 3)-β-D-glucans. *Appl Occup Environ Hyg* (in press).
- Alwis KU, Mandryk J, Hocking AD, Lee J, Mayhew T, Baker W. 1999b. Dust exposures in the wood processing industry. *Am Ind Hyg Assoc J* (in press).
- American Thoracic Society. 1979. ATS statement—snowbird workshop on standardization of spirometry. *Am Rev Respir Dis* 119:831–838.
- Belin L. 1987. Sawmill alveolitis in Sweden. *Int Arch Allergy Appl Immunol* 82:440–443.
- British Medical Research Council. 1960. Standardized questionnaire on respiratory symptoms. *Br Med J* 2:1665.
- Carroso A, Ruffino C, Bugiani M. 1987. Respiratory diseases in wood workers. *Br J Ind Med* 44:53–56.
- Castellan RM, Olenchock SA, Kinsley KB, Hankinson JL. 1987. Inhaled endotoxin and decreased spirometric values. An exposure-response relation for cotton dust. *N Engl J Med* 317:605–610.
- Cohen HI, Merigan TC, Kosek JC, Eldridge F. 1967. A granulomatous pneumonitis associated with redwood sawdust inhalation. *Am J Med* 43:785–794.
- Dahlqvist M, Johard U, Alexandersson R, Bergstrom B, Ekholm U, Eklund A, Milosevich B, Tornling G, Ulfvarson U. 1992. Lung function and precipitating antibodies in low exposed wood trimmers in Sweden. *Am J Ind Med* 21:549–559.
- Donham KJ, Reynolds SJ, Whitten P, Merchant JA, Burmeister L, Popendorf WJ. 1995. Respiratory dysfunction in swine production facility workers: dose-response relationships of environmental exposures and pulmonary function. *Am J Ind Med* 27:405–418.

- Douwes J, Versloot P, Hollander A, Heederik D, Doekes G. 1995. Influence of various dust sampling and extraction methods on the measurement of airborne endotoxin. *Appl Environ Microbiol* 61:1763–1769.
- Dutkiewicz J, Jabinski L, Olenchock SA. 1988. Occupational biohazards: a review. *Am J Ind Med* 14:605–623.
- Dykewicz MS, Laufer P, Patterson R, Roberts M, Sommers HM. 1988. Woodman's disease: hypersensitivity pneumonitis from cutting live trees. *J Allergy Clin Immunol* 81:455–460.
- Eduard W, Lacey J, Karlsson K, Palmgren U, Strom G, Blomquist G. 1990. Evaluation of methods for enumerating microorganisms in filter samples from highly contaminated occupational environments. *Am Ind Hyg Assoc J* 51:427–436.
- Enarson DA, Chan-Yeung M. 1990. Characterization of health effects of wood dust exposures. *Am J Ind Med* 17:33–38.
- Fogelmark B, Rylander R. 1993. Lung inflammatory cells after exposure to mouldy hay. *Agents Action* 39:25–30.
- Forest Industries. 1992. Forest facts, overview. Canberra: Forest Industries.
- Franklin CIV. 1982. Adenocarcinoma of the paranasal sinuses in Tasmania. *Australas Radiol* 26:49–52.
- Gandevia B, Milne J. 1970. Occupational asthma and rhinitis due to western red cedar (*Thuja plicata*), with special reference to bronchial reactivity. *Br J Ind Med* 27:235–244.
- Gibson J, Gallagher H, Johansen A, Webster I. 1979. Lung function in an Australian population. *Med J Aust* 1:292–295.
- Goldsmith DF, Shy CM. 1988. An epidemiologic study of respiratory health effects in a group of North Carolina furniture workers. *J Occup Med* 30:959–965.
- Hedenstierna G, Alexandersson R, Belin L, Wimander K, Rosen G. 1986. Lung function and *Rhizopus* antibodies in wood trimmers. *Int Arch Occup Environ Health* 58:167–177.
- Hinds WC. 1988. Basis for particle size-selective sampling for wood dust. *Appl Ind Hyg* 3:67–72.
- Holness DL, Sass-Kortsak AM, Pilger CW, Nethercott JR. 1985. Respiratory function and exposure-effect relationships in wood dust exposed and control workers. *J Occup Med* 27:501–506.
- IARC. 1995. Wood dust and formaldehyde (IARC monographs on the evaluation of carcinogenic risks to humans, vol. 62). Lyon, France: International Agency for Research on Cancer.
- Ironside P, Matthews J. 1975. Adenocarcinoma of the nose and paranasal sinuses in woodworkers in the state of Victoria, Australia. *Cancer* 36:1115–1121.
- Lazarus R. 1982. Lung-function reference values from Victorian power-industry workmen. *Med J Aust* 2:121–124.
- Leclerc A, Cortes MM, Gerin M, Luce D. 1994. Sinonasal cancer and wood dust exposure: results from a case-control study. *Am J Epidemiol* 140:340–349.
- Li D, Yuan L, Yi S, Jiang Z. 1990. Effects of wood dust exposure on respiratory health: cross-sectional study among farmers exposed to wood dust. *Am J Ind Med* 17:84–85.
- Liou SH, Cheng SY, Lai FM, Yang JL. 1996. Respiratory symptoms and pulmonary function in mill workers exposed to wood dust. *Am J Ind Med* 30:293–299.
- Michaels L. 1967. Lung changes in wood workers. *Can Med Assoc J* 96:1150–1155.
- Obayashi T. 1990. A new endotoxin-specific assay. *Adv Exp Med Biol* 256:215–223.
- Olenchock SA. 1990. Endotoxins. In: Morey PR, Freeley JC Sr, Otten JA, editors. *Biological contaminants in indoor environments*. Philadelphia: American Society for Testing and Materials. p 190–200.
- Olenchock SA. 1994. Health effects of biological agents: the role of endotoxins. *Appl Occup Environ Hyg* 9:62–64.
- Pisaniello DL, Connell KE, Muriale L. 1991. Wood dust exposure during furniture manufacture — results from an Australian survey and considerations for threshold limit value development. *Am Ind Hyg Assoc J* 52:485–492.
- Rastogi SK, Gupta BN, Husain T, Mathur N. 1989. Respiratory health effects from occupational exposure to wood dust in sawmills. *Am Ind Hyg Assoc J* 50:574–578.
- Reynolds SJ, Donham KJ, Whitten P, Merchant JA, Burmeister LF, Pependorf WJ. 1996. Longitudinal evaluation of dose-response relationships for environmental exposures and pulmonary function in swine production workers. *Am J Ind Med* 29:33–40.
- Rylander R. 1985. Organic dusts and lung reactions — exposure characteristics and mechanisms for disease. *Scand J Work Environ Health* 11:199–206.
- Rylander R. 1990. Health effects of cotton dust exposures. *Am J Ind Med* 17:39–45.
- Rylander R. 1996. Airway responsiveness and chest symptoms after inhalation of endotoxin or (1 → 3)- β -glucan. *Indoor Built Environ* 5:106–111.
- Rylander R, Haglund P, Lundholm M. 1985. Endotoxin in cotton dust and respiratory function decrement among cotton workers in an experimental cardroom. *Am Rev Respir Dis* 131:209–213.
- Rylander R, Peterson Y, Donham KJ. 1990. Questionnaire evaluating organic dust exposure. *Am J Ind Med* 17:121–126.
- Shamssain MH. 1992. Pulmonary function and symptoms in workers exposed to wood dust. *Thorax* 47:84–87.
- Standards Australia. 1987. Workplace atmospheres—methods for sampling and gravimetric determination of respirable dust. Australian Standard 2985–1987. New South Wales: Standards Australia.
- Standards Australia. 1989. Workplace atmospheres—methods for sampling and gravimetric determination of inspirable dust. Australian Standard 3640–1989. New South Wales: Standards Australia.
- Vaughan NP, Chalmers CP, Botham RA. 1990. Field comparison of personal samplers for inhalable dust. *Ann Occup Hyg* 34:553–573.
- Whitehead LW, Ashikaga T, Vacek P. 1981. Pulmonary function status of workers exposed to hardwood or pine dust. *Am Ind Hyg Assoc J* 42:178–186.
- Wilhelmsson B, Drettner B. 1984. Nasal problems in wood furniture workers. *Acta Otolaryngol* 98:548–555.
- Woods B, Calnan CD. 1976. Toxic woods. *Br J Dermatol* 95(suppl. 13):1–97.
- Worksafe Australia. 1995. Exposure standards for atmospheric contaminants in the occupational environment. Canberra: Australian Government Publishing Service.