

WORK-RELATED ASTHMA AMONG WORKERS IN THE WOOD-PROCESSING INDUSTRY: A REVIEW

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ABSTRACT

Various studies, including some in Africa, have reported an association between occupational exposure to wood dust and known probable asthma. Globally, the prevalence of asthma in woodworkers is between 5.6% and 18%; in Africa it is between 3% and 7%. This review focuses on epidemiological evidence from studies performed around the world to interrogate the evidence for the association between the exposure to wood dust and asthma. The review confirms a positive association between occupational exposure to wood dust and asthma. However, it has identified some limitations in the literature that relate to variable diagnostic criteria and epidemiological definitions for asthma: a lack of objective markers of asthma such as non-specific challenge tests and other inflammatory markers as well as markers for allergic sensitisation; understanding the pathophysiological mechanisms involved in the causation of asthma; limited number of studies of other agents present in wood dust such as endotoxins, glucans, terpenes and wood dust allergens; and the limited number of epidemiological studies investigating the dose–response relationships for allergens and other agents causing rhinitis and asthma among workers exposed to wood dust. These issues need to be addressed in future studies contemplating further investigation in this field.

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INTRODUCTION

Wood is among the most important natural resources in the world and annually at least 1 700 million cubic metres are collated for industrial use.¹ Worldwide 12 000 tree species exist, and more than 1 000 of these are used for commercial purposes.² More than 52 types of wood species are grown in Mozambique.³ The National Institute of Statistics (INE) (2011) estimated that at least 5 500 people are employed in 506 companies in the formal sector in Mozambique. The number of workers and companies in the informal sector is not captured by the INE estimates and is unknown, but is likely to be higher than that in the formal sector. This review was undertaken to better understand the respiratory health risks associated with exposure to wood dust in order to develop preventive strategies to reduce these risks.

WOOD PROCESSING AND HIGH-RISK WORK PROCESSES

The major wood-working processes are debarking, sawing, sanding, milling, lathing, drilling, veneer cutting, chipping and mechanical defibrating (Figure 1-4). From the tree-felling stage onwards through the various stages of woodworking and manufacturing processes, workers are exposed to airborne dust of different particle sizes, concentrations and compositions. Sawing and sanding both shatter lignified wood cells and break out whole cells

and groups of cells (chips).⁴ With increased cell shattering finer dust particles are produced. Sawing and milling are mixed cell shattering and chip-forming processes, whereas sanding is almost exclusively cell shattering. In hardwoods, the cells are tightly bound, resulting in more shattering and more dust. Similarly, dry wood leads to more dust formation. Softwood particles are more fibrous and usually larger and as a result less capable of becoming airborne.⁴ Owing to their damp consistency, fungal or bacterial contamination occurs on a large scale when green or fresh wood is processed.

Considerably high heat generation during sawing, machining and sanding may change the chemical composition of wood dust. It has been reported that hardwoods give rise to finer airborne dust at a lower rate during sanding than softwoods, but that the total amount of airborne dust produced depends only on the total mass of wood removed, and not on the type of wood.⁴

Besides exposure to wood dust itself, wood workers are potentially exposed to various chemicals used in the different stages of the production process in the furniture industry, including glues (during assembling), dyes, solvents, dryers (in the finishing stage for painting), varnishes (in the finishing stage for polishing) and hardeners.



Figure 1: Carving and sawing wood



Figure 2: Drilling operations



Figure 3: Planing wood



Figure 4: Machine cleaning

CONSTITUENTS OF WOOD AND WOOD DUST

Wood is the hard fibrous substance composed mostly of the stem and branches of a tree or shrub, and covered by the bark. The inner core of the wood is referred to as 'heartwood' and the outer layer is called 'sapwood'. For industrial purposes, wood is classified into two types: hardwoods and softwoods. Softwoods are derived from coniferous trees (botanically named as *Gymnospermae* with exposed seeds), whereas hardwoods are derived from deciduous trees (botanically named as *Angiospermae* with encapsulated seeds).⁴

The major component (about 95% by weight) of wood is cellulose, hemicellulose and lignin.² Cellulose, which is built up exclusively of D Glucose units joined by β (1 $\rightarrow$ 4) glycosidic linkages, is the major component (40–50%) of both hardwood and softwood. The remaining 5% comprises numerous other high and low molecular weight organic and inorganic compounds, including proteins, which can be extracted ('wood extractives'). These low molecular compounds include terpenes, terpene derivatives such as abietic acid, phenolic compounds, tannins, stilbenes, flavonoids and glycosides, many with known sensitising and irritative properties.²

Dust generated from wood processing is a heterogeneous mixture of inorganic and organic particles – wood fragments, viable and non-viable microorganisms,

endotoxins, glucans, allergens, or mycotoxins – which are all potentially hazardous upon inhalation.¹

EXPOSURE AND CAUSATIVE AGENTS

Furniture workers are exposed to several sensitisers or irritants including wood dust, 'wood extracts' (terpenes, terpene derivatives such as abietic acid and plicatic acid), endotoxins, glucans, isocyanate vapours and other chemicals (including glues, dyes, varnishes, paints, solvents and hardeners).^{5,6}

Exposure assessment studies have demonstrated that workers in the wood-processing industry are exposed to variable concentrations of hazardous agents (microorganisms, endotoxins, glucans and terpenes) associated with wood dust. The exposure summary estimates reported in these studies have wide ranges, namely, wood dust particulate (0.05–12.0 mg/m³, microorganisms (1.696–2,000 cfu/m³ $\times$ 10³), endotoxins (0.4–6.93 ng/m³), glucans (3.5–18.9 ng/m³) and terpenes (0.23–158 mg m³).

As expected, dry mills had higher dust particulate levels than wet mills. In six of the 11 exposure studies evaluated^{1,7,8,9,10,11} the threshold limit values for wood dust particulate recommended by ACGIH (2013) of 0.5 mg/m³ for Western red cedar and 1 mg/m³ for all other species was exceeded. There are currently no international exposure

standards for other agents such as the glucans, endotoxins, terpenes and wood allergens found in wood dust.

AIRWAY DISEASE AND PATHOPHYSIOLOGICAL MECHANISMS

Exposure to wood dust and other causative agents associated with wood dust has long been associated with a variety of adverse health effects of the upper and lower airways. Health effects associated with exposure to wood dust and associated agents present in the upper airways include rhinitis, whereas in the lower airways it results in occupational asthma and general lung function deficits.¹²

The mechanisms for wood dust-causing asthma are far from fully understood.¹³ For Western red cedar, a unique constituent, plicatic acid, has been shown to be a causal factor, and both immunological and non-immunological mechanisms are involved. For other types of wood no clear causal agent has consistently been found. Specific IgE sensitisation has been reported to be responsible for asthma in woodworkers; however, Type 1 allergy is not suspected to be a major cause of wood dust-induced asthma. Apart from IgE-mediated sensitisation, several other mechanisms, including irritative mechanisms, have been reported.

Animal studies have shown that wood components, such as the major constituent in pine resin – abietic acid – causes direct toxicity via lytic damage to alveolar, tracheal and bronchial epithelial cells. Wood-dust extracts from both hard and soft woods are able to induce the release of pro-inflammatory mediators from macrophages, express and induce the release of inflammatory mediators in human epithelial cell line, and modulate the expression of cytokines and chemokines.^{13,24}

The oxidative products of monoterpenes or abietic acid can also cause airway inflammation through immune reactions. This was supported in an experimental study, which showed increased alveolar cell concentration, mainly macrophages in bronchoalveolar lavage (BAL) after exposure to concentrations of 450 ng/m³.¹³ Pylkkänen et al¹⁴ studied the capacity of wood dusts to induce the production of reactive oxygen species (ROS) and caspase-3 activity in human bronchial epithelial cells. Dust from three wood species – pine, birch and oak – induced ROS production. The study concluded that exposure to external particles may induce cellular apoptosis and necrosis of epithelial cells, which again may lead to shedding of the bronchial mucosa, to bronchial hyper-reactivity, and the exacerbation of asthma.

EPIDEMIOLOGY OF ALLERGIC SENSITISATION AND ASTHMA ASSOCIATED WITH WOOD DUST

The association between exposure to wood dust and asthmatic symptoms has been documented previously.¹⁵

A meta-analysis of wood-dust exposure and risk of asthma showed that the pooled relative risk (RR) of asthma among workers exposed to wood dust was 1.53 (95% CI 1.25–1.87). When the analysis was restricted to studies carried out on Caucasian populations, the pooled RR was 1.59 (95% CI 1.26–2.00), whereas the pooled RR of studies on Asian populations was 1.15 (95% CI 0.92–1.44).¹⁶

Table I reports on various epidemiological studies of asthma and other respiratory symptoms and the definitions used in these studies. While the prevalence of asthma in woodworkers globally based on various different definitions is between 5.6% and 18%, the prevalence in Africa appears to be lower: between 3% and 7%.^{4,17,18,19} The prevalence of rhinitis appears to be much higher ranging, at between 16% and 33%.

The prevalence of asthma appears to be higher in dry sawmills (11.8%) compared to green sawmills (7.5%); however, the prevalence of symptoms appears to be higher in the green sawmills.¹⁷ Woods species such as pine, mahogany, African teak, red cedar and imbuia are associated with a higher prevalence of asthma and rhinitis. Furthermore, fibre- and chipboards and mansononia are also associated with a high prevalence of rhinitis.

HOST RISK FACTORS

A. GENETICS

A meta-analysis of wood-dust exposure and risk of asthma conducted by Perez-Rios¹⁶ demonstrated a higher risk of asthma among European and American studies than among Asian studies, indicating possible genetic factors in the development of asthma. This suggests the existence of interactions between genetic susceptibility to asthma and environmental factors. Several genetic factors are probably responsible for allergen sensitisation, which is determined by HLA genotype, including sensitisation to occupational agents.²⁷

B. ATOPY

Schlünssen et al,¹⁵ in their Danish study, demonstrated that asthma was associated with atopy, using clinical endpoints such as asthma symptoms; OR = 4.2 (95% CI = 2.4–7.7) as well as asymptomatic bronchial hyper-responsiveness; OR = 7.4 (95% CI = 2.8–19.7).

C. UPPER AIRWAY SYMPTOMS

Occupational rhino-conjunctivitis often precedes the onset of IgE-mediated occupational asthma and it should be considered an important risk factor for the future development of occupational asthma, especially when high molecular weight agents are involved.²⁷ Occupational rhinitis is associated with an increased risk of asthma, although the proportion of subjects with occupational rhinitis who will develop occupational asthma remains uncertain.²⁸

TABLE I: EPIDEMIOLOGICAL STUDIES OF WOOD WORKERS

TYPE OF WORKFORCE (AUTHOR, COUNTRY)	DEPARTMENTS	N	OUTCOME MEASURE	WOODS IMPLICATED	PREVALENCE	DEFINITION OF ASTHMA AND RHINITIS
Sawmill plants (Ugheoke, Nigeria) ¹⁹	Not stated	228	Respiratory symptoms	NR	Symptoms: Cough 35.1% Breathlessness 8.3% Wheezing 3.1%	NR
Furniture industry (Públio, Brazil) ²⁰	Furniture, painting and finishing	499	Respiratory symptoms	NR	Asthma 5.6% Rhinitis 16%	Asthma: Wheeze and attack of shortness of breath. Rhinitis: Nasal congestion, rhinorrhoea and sneezing.
Carpentry, sawmill plant (Aguwa et al, Nigeria) ²¹	Carpentry, sawmill, carving and timber marketing	591	Occupational asthma and rhinitis	Mahogany, iroko, cedar and mansonia	Asthma 6.3% Rhinitis 78%	Asthma: Wheezing and breathlessness. Rhinitis: One or more of the following – sneezing, rhinorrhoea and/or nasal/pharyngeal itching.
Sawmill (Fox, South Africa) ⁸	Dry mill – stress grading Wet mill – green chain, stacking and kilns	392	Asthma	Pine	Asthma: Definite 0.5% Probable 2.6% Possible 10.2%	Definite: Positive questionnaire and $\geq 15\%$ improvement in FEV ₁ post-salbutamol or on return from leave. Probable: Positive questionnaire and $> 10\%$ and $< 15\%$ improvement in FEV ₁ post-salbutamol or on return from leave or doctor diagnosis of asthma. Possible: Positive questionnaire (except doctor-diagnosed asthma).
Furniture plant (Arbak et al, Turkey) ²²	Furniture decoration	64	Respiratory symptoms and peak expiratory flow rate	Fir tree (<i>Aberia mulleriana</i>)	Symptoms: Nasal 34.4% Cough 23.4% Shortness of breath 18.8% Chest tightness 15.6% Wheezing 14.1%	NR
Plywood and wood-working plant (Borm et al, Indonesia) ²³	Veneer preparation, veneer peeling, veneer drying, assembly, grading, composer and finishing	982	Respiratory symptoms, lung function, and nasal cellularity	Meranti	Symptoms: Cough 23.5% Shortness of breath 4.1% Wheezing 1.6%	NR
Furniture factory (Milanowski et al, Poland) ⁹	Sawing, trimming, sanding, planing, and varnishing workshop	48	Work-related symptoms	Fibre- and chipboards	Symptoms: Cough 52.1% Shortness of breath 12.5% Asthma 6.2% Rhinitis 33.3%	Asthma: Specific inhalation challenge test. Rhinitis: Rhinorrhoea and nasal congestion.
Furniture factory (Schlünssen et al, Denmark) ²⁵	Sanding, cutting, assembling and packing	2507	Respiratory symptoms and lung function	Pinewood, particle board, hardwood, beech medium-density fibreboard, and mixed.	Asthma 6.3% Rhinitis 19.2%	'Yes' to: • At least one group A question (Have you been told by a doctor that you have asthma? Do you have asthma? Have you ever had asthma? Do you ever had wheeze?) AND • Two or more group B questions (Do you ever have chest tightness? Do you wake in the night with chest tightness? Do you wake in the night wheezing?).

TYPE OF WORKFORCE (AUTHOR, COUNTRY)	DEPARTMENTS	N	OUTCOME MEASURE	WOODS IMPLICATED	PREVALENCE	DEFINITION OF ASTHMA AND RHINITIS
Small-scale wood industries (Rongo et al, Tanzania) ⁹	Planing, sawing, carving, and drilling	546	Respiratory symptoms and dust exposure	African teak, mahogany, bloodwood, East African camphor, East African afzella.	Asthma 5–7%	Asthma: Asthmatic symptoms of shortness of breath with wheezing.
Sawmill (Douwes et al, New Zealand) ¹⁸	Administration, green mill, planer mill, remanufacturing and moulding	772	Asthma and other respiratory symptoms	Pine	Symptoms: Wheezing 29.6% Asthma: 18%	Asthma: Awoken by shortness of breath in past 12 months or asthma attack in past 12 months or current asthma medication.
Sawmill (Mandryk et al, Australia) ¹⁷	Three green mills and two dry mills	87	Work-related symptoms	Eucalyptus	Symptoms in dry mill: Asthma 11.8% Cough 29.4% Breathlessness 8.8% Wheezing 8.8% Chest tightness 11.8% Symptoms in green mill: Asthma 7.5 % Cough 50.9% Breathlessness 24.5 % Wheezing 24.5% Chest tightness 28.3%	Asthma-like symptoms.
Furniture industry (Talini et al, Italy) ⁵	Furniture manufacturing, spray painting and assembling	296	Asthma-like symptoms, bronchial hyper-responsiveness	Pine and beech	Asthma 7.7%	Asthma: Symptoms such as chronic cough, wheeze, attack of shortness of breath; Non-specific bronchial hyper-reactivity: PD20 <0.8 mg/ml.
Wood-processing factories (Alwis, Australia) ⁴	Two logging sites, four sawmills, one major wood-chipping operation and five joineries	195	Symptoms	Eucalyptus, Pinus radiata, meranti (Shorea spp), Western red cedar (Thuja plicata), brushbox (Tristania conferta)	Symptoms: Breathlessness 17.4% Wheezing 22.6% Chest tightness 27.2% Asthma 10.8%	NR
Furniture plant (Shamssain, South Africa) ²⁶	Not stated	145	Pulmonary function and symptoms	Pine and medium-density board fibre wood	Symptoms: Nasal 49% Cough 43% Phlegm 15%	NR

*NR: Not reported.

D. SMOKING

The relationship between smoking and occupational asthma is complex and contradictory. However, various studies have shown that smokers are at increased risk of developing sensitisation, although not necessarily asthma, due to agents that cause asthma through an IgE-mediated mechanism. Schlünssen,¹⁵ in her study of asthma among atopic and non-atopic woodworkers, found that smokers were more often asthmatic, with OR = 3.6 (95% CI = 1.7–7.6) for work-related asthma symptoms and OR = 2.3 (95% CI = 1.0–5.4) for symptomatic bronchial hyper-responsiveness compared to non-smokers.

ENVIRONMENTAL RISK FACTORS

A. JOB DURATION

Aguwa et al,²¹ in their study among woodworkers in south-eastern Nigeria, reported that the prevalence of occupational rhinitis and asthma among woodworkers increased with the duration of exposure. Among workers with less than two years' exposure, the prevalence of rhinitis was 8.7% and of asthma was 1.9%, whereas in workers with more than 10 years of exposure the prevalence of the rhinitis was 36.5% and that of asthma 6.5%.

Meo's²⁹ study among workers in small-scale wood industries found that the peak expiratory flow (PEF) in exposed workers was negatively associated with years of exposure. In workers with less than four years' exposure the PEF was 312.28 ± 23.62 l/min compared to those with more than eight years, having a PEF of 177.00 ± 22.29 l/min.

Shamssain et al²⁶ found that the prevalence of respiratory symptoms increases with the increase in the number of years of employment. Cough and nasal symptoms showed a pronounced increase with years of employment. The prevalence of cough in workers with 1–4 years of employment was 35%, whereas in workers with 9–12 years this was 66%. The prevalence of nasal symptoms in workers with 1–4 years of employment was 49% compared to workers with 9–12 years employment was 55%. The study also found that the proportion of subjects with an FEV₁/FVC ratio below 70% in the exposed group employed for 10–19 years was 56.2% compared to the 26.7% in the exposed group with less than 10 years of employment.

B. EXPOSURE LEVELS

Schlünssen and Schaumburg¹⁵ found that symptomatic bronchial hyper-responsiveness was related to dust in a dose-dependent manner, with OR = 18.3 (2.0–170.8) when comparing the group with the highest (4.10 mg/m³) to the lowest (0.17 mg/m³) exposure level. The same tendency was seen for work-related asthma symptoms with OR = 6.4 (95% CI = 1.6–26.4).

Schlünssen and Schaumburg,²⁵ in their earlier study, reported that the prevalence of asthma and rhinitis increased with increasing of level of exposure. The prevalence of asthma in male workers exposed to wood-dust levels ranging between 0.0 mg/m³ and 0.74 mg/m³ was 3.9% compared to workers exposed to dust ranging between 0.74 mg/m³ and 1.42 mg/m³ which was 7.8%. Similarly, the prevalence of rhinitis in workers exposed to wood dust ranging between 0.0 mg/m³ and 0.74 mg/m³ was 19.1% compared to workers exposed to wood dust ranging between 0.74 mg/m³ and 1.42 mg/m³, which was 22.3%.

Mandryk et al¹⁷ demonstrated a relationship between exposure to endotoxins and glucans and work-related symptoms in workers at three green mills and two dry mills. The mean (GM) personal inhalable endotoxins (6.61 ng/m³) and inhalable glucans (2.63 ng/m³) exposures were high for the green mills (1.58 ± 1.52 ng/m³) compared with those of dry mills (0.38 ± 1.73 ng/m³), respectively. Green-mill workers showed a strong association between personal exposure and work-related symptoms. Significant relationships were found between glucan exposure and nasal congestion (OR = 62; 95% CI: 30.79–124.85), endotoxins and nasal congestion (OR = 49; 95% CI: 24.29–98.67), when the same logistic model was adjusted for endotoxins and glucans, respectively. Similarly, glucan exposure was associated with chronic bronchitis (OR 163; 95% CI: 71.52–368.71), as were endotoxins associated with chronic bronchitis (OR = 17; 95% CI: 7.92–36.23).

In a study of respiratory symptoms and dust exposure among male workers in small-scale wood industries in Tanzania, allergic symptoms were reported more commonly in the low-exposure group with OR = 2.4 (95% CI = 1.8–3.1) and the high-exposure group with OR = 2.7 (95% CI = 1.8–4.0), compared to control groups.⁹

A study of asthma and other respiratory symptoms in workers at a New Zealand pine-processing sawmill showed that working in sawmilling was associated with an increased prevalence of asthma and cough symptoms. Asthma symptoms in the exposed workers (18%) were more common than in the general population (12.1%), with adjusted OR = 1.6 (95% CI = 1.1–2.3). Asthma was also more common in the low-exposure group (15.6%) and high-exposure groups (high exposure to 'green dust' = 20.4% and 'dry dust' = 18.8%) than in non-exposed workers (9.2%). The respective adjusted odds ratios were OR = 1.9 (95% CI = 0.7–4.9), 2.7 (95% CI = 0.9–7.6), and 2.1 (95% CI = 0.8–5.7), respectively, but these were not significant. Adjusted odds ratios for symptoms of cough were OR = 2.7 (95% CI = 1.2–6.5) for the low, OR = 5.2 (95% CI = 2.1–13.0) for the high 'green dust' and OR = 3.3 (95% CI = 1.4–7.9) for the high 'dry dust' exposure groups compared to the non-exposed group.¹⁸

In an investigation into the increased incidence of respiratory symptoms among female woodworkers exposed to dry wood, the cumulative incidence proportion of daily coughing and chronic bronchitis was found to be associated with baseline wood-dust exposure in a dose-dependent manner. The risk estimates for daily coughing (with reference to the lowest exposure quartile) was OR = 1.6 (95% CI = 0.6–4.3), OR = 3.2 (95% CI = 0.9–6.8) and OR = 3.8 (95% CI = 1.5–9.7), respectively, in the second and third lowest and the highest quartile. The figures for chronic bronchitis were, accordingly, OR = 2.3 (95% CI = 0.4–14.5), OR = 3.0 (95% CI = 0.5–18.7) and OR = 6.0 (95% CI = 1.2–28.8).³⁰

Kespohl et al found that obeche wood from Cameroon (called *ayous*) had less allergen content compared to obeche wood from Ghana (called *wawa*).³¹ Further analysis showed that the reduced allergen content in *ayous* wood could be ascribed to a reduced amount of major obeche wood allergen Trip s 1. This emphasises the importance of using the content of wood dust allergens, and not only the concentration of airborne wood dust, in estimating dose–response relationships.²

PREVENTION STRATEGIES

Primary prevention of occupational asthma includes reducing exposure in order to prevent the development of respiratory disease in susceptible workers. Several studies support the view that reduction is likely to be followed by a concomitant reduction in disease burden and that use of personal protective equipment is the less effective measure in the hierarchy of controls against health hazards.³² Control of exposure can be achieved by different control measures and the hierarchical strategy commonly applied is elimination, reduction, isolation, ventilation, avoidance of exposure and personal protection.³² The preferred measure is substitution of an agent in the work process, for instance, the substitution of woods with strong sensitising potential with less strong sensitising woods. When substitution is not possible, exposure reduction, which is achieved in practice through a combination of different interventions, is the next best approach. It is important to define a desirable reduction in exposure and exposure standards can play an important role in this process.

Several organisations and countries have set standards or drawn up recommendations for limiting wood-dust exposure. The ACGIH has proposed a level of 0.5 mg/m³ for Western red cedar and 1 mg/m³ for all species [American Conference of Governmental Industrial Hygienists (ACGIH), 2013]. The Swedish exposure limit is 2 mg/m³ for all types of wood.³³ In South Africa the occupational exposure limit for wood dust is 5 mg/m³ (<http://www.labour.gov.za/DOL/legislation/regulations/occupational-health-and-safety/regulation-ohs-hazardous-chemical-substances>).

The intervention study conducted in Minnesota, United

States, which included a combination of engineering, administrative, and behavioural components, has demonstrated that a combination of these measures can contribute to a reduction in the level of exposure. The median dust concentration at baseline in the original study was 5.87 mg/m³ among intervention businesses and 6.23 mg/m³ among comparison businesses. One year later, the median dust concentrations were reduced to 4.71 mg/m³ (a reduction of 19.8%) and 5.58 mg/m³ (a reduction of 10.4%) from baseline in the intervention and comparison businesses, respectively.³⁴

Hasle et al³⁵ have suggested that small enterprises have special problems that need to be considered when designing interventions for the work environment. The risk is higher and the ability to control lower due to limited human and financial resources. Tools and methods aimed at small enterprises may include different types of checklist (including both risk assessment and more action-oriented types), comprehensive health and safety management systems, and accident prevention. The most successful methods appear to be action-oriented, combining health and safety with other management goals, and based on trust and dialogue.³⁵

RESEARCH GAPS

While this review confirms a positive association between occupational exposure to wood dust and asthma, it has identified some deficiencies in the literature that relate to methodological issues, including a lack of

- power due to small sample sizes, variable diagnostic criteria and epidemiological definitions for asthma;
- objective markers of asthma, including non-specific challenge tests and inflammatory markers;
- information on the actual wood species used in the production processes;
- detailed information about the concentrations of other agents present in wood dust such as endotoxins, glucans, terpenes and wood dust allergens, and
- epidemiological studies investigating the dose–response relationships for rhinitis and asthma among workers exposed to wood dust.

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DECLARATION OF CONFLICT OF INTEREST

The authors declare no conflict of interests.

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