

Original Paper

Work-related airways disease and risk factors in Mozambiquan wood processing workers

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ABSTRACT

Background: Inhalation of wood dust particulates, bioaerosols and chemical components cause airway disease of a sensitising or irritative nature.

Aims: This study investigated the disease burden and factors associated with work-related airways disease among wood processors.

Methods: A cross-sectional study enrolled 450 workers from 21 Mozambican wood mill and furniture industries. Work-related asthma (WAS) and work-related ocular-nasal (WONS) symptoms were evaluated, using European Community Respiratory Health Survey (ECRHS) questionnaire. Objective assessments included spirometry, fractional exhaled nitric oxide (FeNO) and Phadiatop tests.

Results: The average age was 38 years, three-quarters did not smoke and almost half were atopic. The proportion of workers with WAS was 14% and WONS was 40%. There were 3% that demonstrated bronchial reversibility, 16% fixed airway obstruction and 5% with FeNO > 50 ppb. WONS was positively correlated with female sex [odds ratio (OR) = 2.60, 95% confidence interval (CI) 1.15–5.90] and atopy (OR = 2.04, 95% CI 1.20–3.47). Bronchial reversibility was associated with increasing age (OR = 1.06, 95% CI 1.02–1.10), atopy correlated with high FeNO (OR = 2.85, 95% CI 1.09–7.44). Processing Mutondo wood was positively correlated with WAS (OR_{adj} = 3.68, 95% CI 1.58–8.58), and WONS (OR_{adj} = 9.34, 95% CI 4.54–19.20). Certain wood types, Missanda (OR_{adj} = 6.84, 95% CI 4.17–11.20), Panga-panga (OR_{adj} = 2.31, 95% CI 1.49–3.60) and Mahogany bean (OR_{adj} = 2.52, 95% CI 1.41–4.51) were primarily correlated with WONS.

Conclusions: Mozambiquan wood processors experience significant work-related airway disease attributable to dust particulate. Mutondo wood type, was positively correlated with both lower and upper airway symptoms, whereas other species were linked only to upper airway symptoms.

INTRODUCTION

Work-related wood particulate exposure has historically been acknowledged as an important factor associated with respiratory disease, particularly in industries processing solid wood [1]. In a systematic review [2], processing of timber and furniture production was associated with an elevated risk [Risk Ratio (RR) = 1.53, 95% confidence interval (CI) 1.25–1.87] of asthma. More recently, a synthesis of systematic reviews confirmed strong evidence for this association [3]. Global estimates for asthma prevalence among woodworkers vary geographically, range between 6% and 18%, while estimates for African populations are between 3% and 7% [2, 4]. The occurrence of asthma (4–24%) and rhinitis (39–59%) is higher in newer studies [4, 5].

Studies identified various risk factors associated with airways disease in wood-working populations. Chaiear *et al.* [6] found that women had twice the odds of reporting upper airway symptoms. Furthermore, significant links between atopy and lower as well as upper airway disease has also been shown [7]. Bolund *et al.* observed a dose–response relationship in female smokers, showing a greater likelihood of lung function decline [odds ratio (OR) = 0.847; 95% CI 0.9–82.4] in a 6-year follow-up study [8]. Studies from Ethiopia [9], India [10] and Iran [11] identified that previous wood dust exposure and employment >5 years were associated with respiratory symptoms and/or deterioration in lung function. Positive correlations have also been demonstrated between wood particulate concentration and respiratory

Key learning points

What is already known about this subject:

- Airborne wood dust generated from processing activities is a well-established occupational hazard, contributing to both lower and upper airway symptoms, including asthma.
- Wood mill processors have an elevated risk of experiencing occupational asthma due to exposure to sensitizing agents present in wood dust.
- Despite these known risks, there remains a significant gap in research concerning work-related asthma studies in low- and middle-income countries.

What this study adds:

- The proportion of Mozambican wood mill processors with work-related asthma symptoms was 14%, but lower in comparison to the 40% prevalence of work-related ocular-nasal symptoms.
- Affected workers experienced allergic and non-allergic work-related asthma symptoms in comparable proportions.
- Exposure to wood dust from specific indigenous Mozambican tree species, particularly Mutondo, was strongly correlated with both upper and lower airway symptoms, including asthma.

What impact this may have on practice or policy:

- Healthcare professionals should maintain a high degree of vigilance for occupational asthma in workers that process wood, particularly those involved in wood milling and furniture manufacturing.
- There is a need for preventive measures to reduce work-related rhinitis and asthma in this sector.
- These measures should include, regulatory policies, health-related exposure standards, implementation of workplace control strategies as well as occupational health and safety education and training of both workers and managers.

symptoms [6, 12] while decreased peak expiratory flow was associated with occupation as well as use of powered tools [10].

Although occupational risks among workers in high-income countries is well documented, data from low- and middle-income countries, particularly from Africa, remain limited. The current study investigated the occurrence of work-related symptoms, associated risk factors and objective indicators of airways disease in Mozambican wood-processing workers. Since Mozambique employs a large workforce exposed to various wood species having differing allergenic potential, understanding these risk factors is essential to formulating effective preventive strategies to reduce work-related airways disease [13].

METHODS

This cross-sectional study evaluated 450 of 513 currently employed workers (88% response rate) in 21 of Mozambique's

furniture and joinery factories, with 142 employed in Maputo City (12 factories), 76 in Maputo Province (3 factories) and 232 in Sofala Province (6 factories). The main tasks were carpentry (43%), sawing (29%), joinery, painting and machinery work (each 8%) and maintenance (4%). Common woods processed were Mahogany bean (*Azelaia quanzensis* Welw.) (96%), African teak (*Pterocarpus angolensis* DC) (84%), Panga-panga (*Millettia stuhlmannii* Taub.) (68%) and Pine (*Pinus* sp.) (62%). Over half worked in closed buildings, and only one factory used local exhaust ventilation. Wet cleaning was more common (76%) than cleaning with dry rags (65%) or compressed air (52%).

Each participant completed a standardized, interviewer-administered questionnaire, which was adapted from the European Community Respiratory Health Survey (ECRHS), with modifications including current and previous jobs, specific wood species, use of wood preservatives, exposure to glues and other chemicals, as well as symptom onset in relation to workplace exposures. To maintain accuracy, the questionnaire was first translated into Portuguese and subsequently translated back into English.

An *asthma symptom score* (ASS) was determined from answers to five questions, assigning a score of 0 (no) or 1 (yes) to each response. The evaluated symptoms comprised wheezing-associated shortness of breath, awakening with chest tightness, episodes of shortness of breath at rest, shortness of breath following exercise, and nocturnal awakening due to breathlessness, as previously documented [14, 15].

Work-related asthma symptoms (WAS) and *work-related ocular-nasal symptoms* (WONS) were characterized as symptoms occurring during work and improvement away from work.

To evaluate atopy, serum Immunoglobulin E (IgE) responses to common aeroallergens were analysed in 391 blood samples. Samples were analysed at the National Institute for Occupational Health (NIOH) Immunology Laboratory using the Phadia250 instrument (ThermoFisher, Uppsala, Sweden), in accordance with manufacturer's guidelines. Atopic status was positive for results ≥ 0.35 PUA/L.

Lung function was performed using the EasyOne spirometer, following the American Thoracic Society (ATS) and European Respiratory Society (ERS) guidelines [16]. Spirometry classified as F-quality were excluded. The Global Lung Function Initiative (GLI-2012) reference values (Other) were used to establish the lower limit of normal (LLN) at the 95th percentile [17]. Fixed airway obstruction was defined as a post-bronchodilator forced expiratory volume in 1 second (FEV_1)/forced vital capacity (FVC) ratio $< LLN$, using GLI reference values adjusted for age, sex, height and ethnicity. Bronchial responsiveness was classified by the presence of a post-bronchodilator increase in FEV_1 of at least 12% and 200 ml, or an increase in FEV_1 of at least 9% as a percentage of predicted FEV_1 [18].

Fractional exhaled nitric oxide (FeNO) was measured using the NIOX MINO[®] (Aerocrine AB, Sweden) according to manufacturer's instructions and in accordance with ATS/ERS recommendations [19]. Participants inhaled NO-free air close to total lung capacity and exhaled at a controlled flow rate of 50 ml/s for 10 s. Two technically acceptable measurements were conducted. If the outcomes varied by ≥ 10 ppb, a third measurement was performed, and the average of all measurements computed [20]. Testing was conducted before spirometry, during the work shift.

All statistical analyses were conducted using STATA version 12 (StataCorp, College Station, TX, USA). FeNO values were

transformed using the natural logarithm when performing linear regression analysis. Unadjusted linear and logistic regression were used to investigate host-related factors (age, gender and atopy) on outcomes, as well as job-related exposures (employment duration, wood types and chemical exposures). Multivariate linear and logistic regression models controlled for important confounders (age, sex and smoking status).

Ethical approval was obtained from the Human Research Ethics Committee (HREC) of the University of Cape Town (HREC Ref: 543/2016), the National Bioethics Committee of the Ministry of Health of Mozambique (40/CNBS/2015), and the University of Michigan Medical School Institutional Review Board (HUM00070862). Informed consent was obtained from all individual participants prior to their inclusion in the study.

RESULTS

The mean age of the study participants was 38 years, the majority being male (94%), non-smokers (76%) and 48% were classified as atopic (Table 1). The wood types most frequently utilized by 75% of processors were African teak as well as Mahogany bean, whilst Panga-panga was processed by approximately half (55%) the workforce.

Physician-diagnosed asthma was reported by 6% of workers, with 1% diagnosed after age 16 (Table 2). Asthma-related symptoms in the past 12 months included shortness of breath while wheezing, chest tightness at night (15%), shortness of breath after exercise (13%), at rest (8%), with wheezing (2%) and night-time attacks of shortness of breath (6%). In addition, 34% reported hay fever symptoms. WONS were reported by 40% of participants, while 14% experienced asthma symptoms attributed to workplace exposures (WAS) (Table 2). Only 2% of workers changed jobs due to WAS. Wood dust particulate was identified as the primary cause of chest (82%) and ocular-nasal symptoms (90%).

According to the reference values from the GLI-2012, 26% of employees exhibited FEV_1 of <80% predicted, while almost an equivalent proportion (25%) had FEV_1 values that fell below the LLN (Table 3). Spirometry testing identified fixed airflow obstruction in 16% of participants. Bronchodilator reversibility, using the standard ATS/ERS criteria, ranged between 3% and 6% of the workforce.

This group of wood mill and furniture workers had a median FeNO concentration of 18.5 ppb (interquartile range: 12–27). FeNO testing revealed high levels (>50 ppb) in 5% of participants, suggesting the presence of eosinophilic airway inflammation (Table 3). Furthermore, WAS were strongly associated with FeNO ($\beta = 0.30$, GM ratio = 1.35, 95% CI 0.08–0.51, $P = 0.006$) and FEV_1 ($\beta = -0.10$, GM ratio = 0.90, 95% CI -0.17 to -0.02, $P = 0.011$).

Statistical analysis in adjusted models demonstrated that female sex was associated with an increased odds of reporting two or more asthma symptoms ($ASS \geq 2$) and WONS. WONS was positively correlated with atopic status ($OR_{adj} = 2.04$, 95% CI 1.20–3.47) (Table 4). A history of WAS was more commonly noted in processors with hay fever ($OR_{adj} = 1.97$, 95% CI 1.15–3.35) including those that currently smoke ($OR_{adj} = 1.97$, 95% CI 1.02–3.80). Older age was independently associated with increased likelihood of fixed airway obstruction ($OR_{adj} = 1.04$, 95% CI

Table 1. Demographic and occupational characteristics of Mozambican wood processors

Demographic characteristic	Prevalence (%) <i>n</i> = 450
Age (years)	38 ± 13
Gender (% male versus female)	94:6
BMI	23 ± 4
Smoking status, <i>n</i> (%)	
Current smokers	69 (15)
Ex-smokers	41 (9)
Never smokers	340 (76)
Job title	
Wood supply—pre-processing (front end) (wood transport to factory, wood unloading and seasoning, wood treatment)	39 (9)
Wood processing	288 (64)
Sawyer/helper/supervisor	32 (7)
Machine operator/helper/drilling/cutting time controller	84 (19)
Carving	4 (1)
Planning	4 (1)
Sanding	18 (4)
Assembling/Skeleton maker	125 (28)
Upholstering	4 (1)
Painting/varnishing	17 (4)
Post processing (back end) (inspection before finishing, furniture to store or warehouse)	123 (27)
Total years in industry	8.6 ± 8.2
Total years in the current job	7.3 ± 7.6
Past history of lung disease (self-reported)	
Previous treatment for tuberculosis	27 (6)
Repeated childhood chest infections	17 (4)
Previous treatment for chronic bronchitis	4 (1)
Family history of allergy	157 (35)
Presence of atopy (positive Phadiatop test/ family history of allergy)	217 (48)
Types of wood used	
African teak (<i>Pterocarpus angolensis</i> DC)	338 (75)
Mahogany bean (<i>Azelia quanzensis</i> Welw)	337 (75)
Panga-panga (<i>Millettia Stuhlmannii</i> Taub)	248 (55)
Pine (<i>Pinussp</i>)	189 (42)
Missanda (<i>Erythrophleum suaveolens</i> Brenan)	129 (29)
Lebombo ironwood (<i>Androstacys johnsonii</i> Prain)	126 (28)
African mahogany (<i>Khaya nyasica</i> Stapf)	98 (22)
Mutondo (<i>Cordyla Africana</i>)	68 (15)
African sandalwood (<i>Spirostachys Africana</i> Sonder)	35 (8)
Zebrawood (<i>Microberliniabrazzavillensis</i>)	14 (3)
Other (e.g. Messassa, Ncula, Mipepe) (<i>Brachystegiaspiciformis</i> , <i>Pterocarpustinctiorius</i> , <i>Albiziaadanthifolia</i>)	77 (17)

1.02–1.06) and bronchial reversibility ($OR_{adj} = 1.06$, 95% CI 1.02–1.10). In addition, high FeNO levels (≥ 50 ppb) was positively correlated with childhood-onset asthma ($OR_{adj} = 4.98$, 95% CI 1.53–16.22) and atopy ($OR_{adj} = 2.85$, 95% CI 1.09–7.44).

In the multivariate adjusted models (Table 5), specific wood species were significantly associated with WONS. Exposure to Mutondo wood dust was strongly linked to the presence of both WONS ($OR_{adj} = 9.34$) and WAS ($OR_{adj} = 3.68$). Other wood types, such as Missanda ($OR_{adj} = 6.84$, 95% CI 4.17–11.20), Panga-panga ($OR_{adj} = 2.31$, 95% CI 1.49–3.60) and Mahogany bean ($OR_{adj} = 2.52$,

Table 2. Asthma history and respiratory symptoms reported by Mozambiquan wood processors

Symptom	Prevalence (%) <i>n</i> = 450
Asthma history	
Ever asthma	48 (11)
Doctor-diagnosed asthma	28 (6)
Current use of asthma medication	6 (1)
Asthma attack in the past 12 months	8 (2)
Current use of asthma medication OR	9 (2)
Asthma attack in the past 12 months	
Asthma-related symptoms	
Ever attacks of wheezing (wheezing in the past)	66 (15)
Wheezing in the past 12 months	29 (6)
Asthma symptom score (ASS \geq 2 symptoms)*	45 (10)
ASS < symptoms	405 (90)
Work-related asthma symptoms	
Episode of high exposure at work causing tight chest, shortness of breath, wheeze or cough	3 (1)
Work-related asthma symptoms ^a	65 (14)
Work-related asthma symptoms ^b	36 (8)
Job change due to work-related asthma symptoms	7 (2)
Work-related ocular-nasal symptoms	
Work-related ocular-nasal symptoms ^a	182 (40)
Work-related ocular-nasal symptoms ^b	88 (20)
Causes of work-related symptoms in the symptomatic group	
Asthma symptoms	
Wood dust	53 (82)
Other (machinery, heavy lifting and varnish)	5 (1)
Ocular-nasal symptoms	
Wood dust	164 (90)
Other (smoke, varnish and glue)	9 (2)

*Symptoms (chest/ocular-nasal) experienced at work ever.

^bSymptoms (chest/ocular-nasal) experienced at work that improves when away from work.

^aAsthma symptom score: An asthma symptom score was computed based on the sum of answers (0 = no, 1 = yes) to five questions on asthma-like symptoms in the past 12 months (short of breath while wheezing, woken up with chest tightness, attack of shortness of breath at rest, attack of shortness of breath after exercise, woken up by attack of shortness of breath).

95% CI 1.41–4.51), were positively linked to WONS, but showed no correlation with lower airway outcomes.

Workers who had undergone health and safety training were more likely to report both WONS (OR = 3.77, 95% CI 2.34–6.06) and WAS (OR = 1.94, 95% CI 1.02–3.67).

Airway obstruction showed a positive relationship with dye exposure (OR = 3.03, 95% CI 1.31–7.00), but was inversely correlated with length of employment (OR = 0.96, 95% CI 0.93–0.99) (Supplementary Tables).

DISCUSSION

This study offers new perspectives into the burden of respiratory symptoms attributable to work and asthma phenotypes among wood processors in Mozambique, highlighting the significance of specific indigenous wood species associated with these clinical endpoints. These findings support the growing body of evidence linking exposure to airborne wood particulate with both lower and upper respiratory tract morbidity.

The prevalence of current asthma in this study was 7%, while 10% of workers had current asthma symptoms with an ASS \geq

Table 3. Pulmonary function indices of wood processing workers of Mozambiquan wood processors

Pulmonary function indices ^{a,b}	Prevalence (%) <i>n</i> = 435
FEV ₁ L [median (IQR)]	3.03 (2.59–3.44)
FVC L [median (IQR)]	3.80 (3.36–4.39)
FEV ₁ % pred. [median (IQR)]	89 (79–96)
FVC % pred. [median (IQR)]	92 (84–104)
FEV ₁ < LLN	102 (23)
FEV ₁ < 80% pred.	115 (26)
FVC < LLN	89 (20)
FVC < 80% pred.	78 (18)
FEV ₁ /FVC [median (IQR)]	80 (73–86)
FEV ₁ /FVC < LLN (pre-bronchodilator)	107 (25)
FEV ₁ /FVC < 70% (pre-bronchodilator)	83 (19)
Fixed airway obstruction	
Post-bronchodilator: FEV ₁ /FVC < LLN	<i>n</i> = 423 67 (16)
Bronchial reversibility (PBD)**	
\geq 200 ml FEV ₁ increase post-bronchodilator	<i>n</i> = 423 71 (17)
\geq 12% FEV ₁ increase post-bronchodilator	14 (3)
\geq 12% and \geq 200 ml FEV ₁ increase post-bronchodilator	14 (3)
Post-bronchodilator Δ FEV ₁ \geq 9% of predicted FEV ₁	24 (6)
Fractional exhaled nitric oxide (FeNO)	
FeNO (ppb) [median (IQR)]	<i>n</i> = 443 18.5 (12–27)
Low < 25 ppb	317 (72)
Elevated 25–50 ppb	104 (23)
High > 50 ppb	22 (5)

FEV₁: forced expiratory volume in 1 second; FVC: forced vital capacity; L: litres; LLN: Lower limit of normal; IQR: interquartile range; % pred: % predicted.

^aPre-bronchodilator values, unless stated otherwise.

^bGlobal lung function initiative (GLI) reference values used.

**Post-bronchodilator (PBD)

[21, 22]. In addition, notable bronchial reversibility was observed in 3–6% of participants. These results indicate that the proportion of asthma in Mozambiquan wood processors appears to be at the upper end of estimates documented for various African nations (3–7%) [4], yet at the lower end of the proportions (4–24%) documented in more recent global studies [5]. The variability in asthma prevalence across these different studies may be attributable to regional factors, variations in wood species processed and differences in workplace environments.

Obstructive lung disease was present in 19% of subjects, with 16% exhibiting fixed airway obstruction, suggesting the presence of chronic obstructive pulmonary disease. These results are comparable to findings from an Ethiopian study, in which the prevalence was 17% [23]. Interestingly, the proportion of current or former smokers in our study was low (24%). Among workers with fixed airway obstruction, 21% reported currently smoking, while 10% had tuberculosis (TB) in the past. These results indicate that, in addition to workplace exposures, other environmental determinants, including biomass fuel usage and exposure to ambient air contaminants, are probably significant contributors to obstructive lung disease in this and other African communities [24].

Table 4. Association between work-related respiratory symptoms and individual host risk factors among Mozambiquan wood processors based on unadjusted regression models

	Asthma symptom score (≥ 2 versus 0–1)	Work-related ocular-nasal symptoms (ever) ^a	Work-related ocular-nasal symptoms ^b	Work-related asthma symptoms (ever) ^a	Work-related asthma symptoms ^b	PBD $\geq 12\%$ and ≥ 200 ml FEV ₁ increase	FeNO ≥ 50 ppb
Prevalence, n (%)	45 (10)	182 (40)	88 (20)	65 (14)	36 (8)	14 (3)	22 (5)
Demographic characteristics							
Age	1.00 (0.97–1.02)	0.98 (0.96–0.99)**	0.99 (0.97–1.00)	1.00 (0.99–1.03)	1.00 (0.97–1.02)	1.06 (1.02–1.10)**	0.97 (0.94–1.01)
Gender (% male versus female)	0.36 (0.14–0.93)*	0.44 (0.20–0.98)*	0.38 (0.17–0.81)*	1.37 (0.40–4.70)	1.09 (0.25–4.81)	NC	1.38 (0.18–10.68)
BMI	0.97 (0.88–1.06)	0.94 (0.88–0.99)*	0.96 (0.90–1.03)	0.99 (0.92–1.06)	1.01 (0.93–1.10)	1.05 (0.96–1.16)	1.00 (0.89–1.11)
Smoking history^c							
Ex-smokers	1.04 (0.35–3.11)	0.68 (0.34–1.36)	0.75 (0.30–1.87)	1.72 (0.74–3.97)	1.25 (0.42–3.78)	1.61 (0.34–7.56)	0.40 (0.05–3.03)
Current smokers	1.44 (0.66–3.18)	1.19 (0.71–2.01)	1.67 (0.92–3.03)	1.97 (1.02–3.80)*	0.91 (0.34–2.44)	0.44 (0.06–3.47)	NC
Allergy history							
Family history of allergy	2.86 (1.53–5.36)**	1.35 (0.91–2.01)	1.46 (0.91–2.56)	1.39 (0.81–2.38)	1.98 (0.99–3.92)	0.50 (0.14–1.83)	1.62 (0.68–3.84)
Atopy (positive Phadiatop)	1.71 (0.87–3.37)	1.24 (0.83–1.87)	2.04 (1.20–3.47)**	0.86 (0.49–1.52)	1.26 (0.60–2.62)	1.17 (0.35–3.91)	2.85 (1.09–7.44)*
Medical history							
Hay fever	1.64 (0.88–3.05)	2.93 (1.95–4.38)***	NC	1.97 (1.15–3.35)*	1.26 (0.62–2.54)	0.14 (0.18–1.06)	1.35 (0.56–3.22)
Childhood-onset (≤ 16 years) asthma	1.98 (0.64–6.11)	1.65 (0.71–3.82)	1.48 (0.57–3.88)	1.70 (0.61–4.75)	CS	1.58 (0.20–12.71)	4.98 (1.53–16.22)***
Adult-onset (> 16 years) asthma	CS	0.98 (0.16–5.93)	1.03 (0.11–9.32)	CS	CS	NC	4.96 (0.53–46.39)
Repeated childhood chest infections	4.09 (1.37–12.21)*	1.03 (0.38–2.76)	0.88 (0.25–3.12)	2.59 (0.88–7.61)	3.86 (1.19–12.51)*	2.17 (0.27–17.77)	1.29 (0.16–10.23)
Chronic bronchitis	9.37 (1.29–68.22)*	4.47 (0.46–43.36)	4.19 (0.58–30.14)	1.99 (0.20–19.42)	3.91 (0.40–38.63)	NC	NC
Pulmonary tuberculosis	2.81 (1.07–7.39)*	1.01 (0.46–2.24)	0.50 (0.15–1.69)	1.76 (0.68–4.55)	2.13 (0.69–6.52)	1.35 (0.17–10.82)	0.75 (0.10–5.84)

Data are presented as OR (95% CI), unless otherwise indicated. Asthma symptom score: short of breath while wheezing, woken up with chest tightness, attack of shortness of breath at rest, attack of shortness of breath after exercise, woken up by attack of shortness of breath. Each OR represents a separate unadjusted regression model.

^aSymptoms (chest/ocular-nasal) ever experienced at work.

^bSymptoms (chest/ocular-nasal) experienced at work that improves when away from work.

^cCurrent or ex-smokers versus never smokers

*P-value < 0.05;

**P-value < 0.01;

***P-value < 0.001.

OR: odds ratio; CI: confidence interval; NC: not calculable; CS: chi-square test significant $P < 0.05$.

Table 5. Self-reported environmental risk factors associated with work-related respiratory symptoms among Mozambiquan wood processors in multivariate regression models

	Asthma symptom score (≥ 2 versus 0–1)	Work-related nasal symptoms (ever) ^a	Work-related ocular symptoms ^b	Work-related asthma symptoms (ever) ^a	Work-related asthma symptoms ^b	PBD $\geq 12\%$ and ≥ 200 ml FEV ₁ increase	FeNO ≥ 50 ppb
Prevalence, n (%)	45 (10)	182 (40)	88 (20)	65 (14)	36 (8)	14 (3)	22 (5)
Environmental factors							
Employment duration in factory	1.00 (0.95–1.05)	1.00 (0.97–1.03)	1.03 (0.99–1.07)	1.04 (0.99–1.08)	1.02 (0.97–1.08)	1.01 (0.95–1.08)	1.04 (0.97–1.12)
Employment duration in current job	0.99 (0.94–1.05)	1.00 (0.97–1.04)	1.02 (0.97–1.06)	1.00 (0.95–1.04)	0.99 (0.93–1.05)	1.04 (0.97–1.12)	0.99 (0.91–1.07)
Working with chemicals							
Glues	0.97 (0.46–2.02)	1.12 (0.72–1.74)	0.56 (0.31–1.03)	1.28 (0.70–2.32)	1.39 (0.64–3.00)	2.41 (0.66–8.75)	2.46 (0.98–6.21)
Dyes	1.20 (0.34–4.23)	0.84 (0.37–1.88)	0.77 (0.25–2.33)	0.44 (0.10–1.91)	0.38 (0.05–2.94)	2.15 (0.38–12.30)	1.81 (0.48–6.80)
Solvents	2.17 (0.76–6.17)	0.50 (0.22–1.18)	0.14 (0.02–1.04)	0.64 (0.18–2.20)	1.85 (0.59–5.75)	1.22 (0.13–11.02)	1.11 (0.24–5.17)
Varnishes	1.07 (0.47–2.43)	0.58 (0.35–0.96)	0.50 (0.24–1.02)	0.84 (0.42–1.67)	1.42 (0.63–3.23)	0.68 (0.14–3.44)	1.36 (0.52–3.58)
Hardeners	1.07 (0.30–3.78)	0.76 (0.35–1.67)	0.44 (0.13–1.53)	0.94 (0.31–2.85)	1.16 (0.33–4.12)	1.02 (0.11–9.35)	0.90 (0.20–4.18)
Type of woods							
Panga-panga	0.73 (0.37–1.44)	2.31 (1.49–3.60)***	0.74 (0.43–1.27)	0.59 (0.33–1.06)	1.01 (0.48–2.16)	0.39 (0.11–1.41)	0.49 (0.20–1.18)
Mahogany bean	1.16 (0.50–2.71)	2.52 (1.41–4.51)**	1.02 (0.53–1.97)	0.64 (0.33–1.25)	1.97 (0.65–5.98)	1.04 (0.21–5.18)	0.47 (0.18–1.24)
African teak	1.36 (0.56–3.30)	1.53 (0.89–2.63)	0.98 (0.51–1.89)	0.78 (0.39–1.54)	1.93 (0.64–5.85)	0.52 (0.13–2.13)	0.75 (0.26–2.19)
Leombo ironwood	1.77 (0.89–3.52)	0.91 (0.58–1.44)	0.64 (0.35–1.18)	1.01 (0.54–1.88)	1.99 (0.94–4.19)	1.31 (0.36–4.74)	1.02 (0.40–2.61)
African mahogany	1.92 (0.92–4.01)	1.65 (1.01–2.70)*	1.00 (0.53–1.89)	1.24 (0.64–2.40)	1.69 (0.75–3.77)	0.56 (0.12–2.75)	0.72 (0.23–2.24)
Pine	1.20 (0.59–2.44)	0.68 (0.44–1.05)	0.81 (0.47–1.41)	1.17 (0.65–2.10)	0.92 (0.43–1.96)	0.87 (0.25–2.99)	1.31 (0.53–3.21)
African sandalwood	1.13 (0.32–3.98)	0.88 (0.39–1.96)	0.76 (0.25–2.31)	0.92 (0.30–2.80)	1.42 (0.40–5.04)	NC	0.58 (0.07–4.63)
Mutondo	1.10 (0.42–2.86)	9.34 (4.54–19.20)***	2.43 (1.23–4.80)*	1.90 (0.92–3.94)	3.68 (1.58–8.58)**	1.36 (0.26–7.08)	0.35 (0.05–2.74)
Zebrawood	1.71 (0.36–8.26)	1.41 (0.46–4.39)	0.75 (0.16–3.63)	0.42 (0.05–3.38)	2.25 (0.47–10.84)	NC	NC
Missanda	0.74 (0.34–1.60)	6.84 (4.17–11.20)***	2.29 (1.33–3.95)**	0.55 (0.27–1.11)	2.09 (0.99–4.41)	0.62 (0.13–2.98)	0.35 (0.10–1.24)
Cleaning activities							
General cleaning	1.02 (0.29–3.60)	0.84 (0.39–1.79)	0.70 (0.28–1.76)	1.19 (0.40–3.60)	1.49 (0.33–6.62)	0.15 (0.02–1.04)	0.96 (0.21–4.47)
Cleaning equipment	0.29 (0.10–0.83)*	0.55 (0.23–1.33)	1.19 (0.38–3.77)	0.47 (0.17–1.28)	2.29 (0.29–17.91)	NC	0.57 (0.12–2.76)
Cleaning PPE	0.96 (0.30–3.11)	0.58 (0.27–1.24)	0.80 (0.32–1.97)	0.50 (0.20–1.22)	3.36 (0.43–26.13)	NC	1.83 (0.22–14.88)
Use of PPE	1.36 (0.50–3.70)	2.09 (1.11–3.94)*	1.68 (0.74–3.82)	1.27 (0.54–2.98)	2.80 (0.65–12.13)	0.49 (0.12–2.04)	1.31 (0.37–4.68)
Mask use	1.84 (0.52–6.48)	1.18 (0.59–2.35)	0.60 (0.28–1.30)	1.11 (0.43–2.86)	2.10 (0.47–9.40)	1.00 (0.12–8.59)	NC
Training	1.39 (0.68–2.84)	3.77 (2.34–6.06)***	2.10 (1.17–3.78)*	1.94 (1.02–3.67)*	0.96 (0.45–2.06)	0.46 (0.12–1.71)	0.63 (0.26–1.54)

Data are presented as OR (95% CI), unless otherwise indicated. Asthma symptom score: short of breath while wheezing, woken up with chest tightness, attack of shortness of breath at rest, attack of shortness of breath after exercise, woken up by attack of shortness of breath. Each OR adjusted for age, gender, atopy and smoking.

*Symptoms (chest/ocular-nasal) ever experienced at work.

^bSymptoms (chest/ocular-nasal) experienced at work that improves when away from work.

^cP-value < 0.05;

**P-value < 0.01;

***P-value < 0.001.

OR: odds ratio; CI: confidence interval; PPE: personal protective equipment.

Alternatively, this could be indicative of chronic untreated asthma, since only 1% used asthma medication while 14% reported WAS.

To our knowledge, this is the first study, from Africa, to identify elevated FeNO levels (>50 ppb) in individuals working with wood. Nonetheless, the proportion of employees with elevated FeNO (5%) found in this study is considerably lower than the 18% reported in British woodworkers (FeNO >40 ppb) [25]. Furthermore, the significant correlation between high FeNO (≥ 50 ppb) and post-bronchodilator responsiveness indicate that individuals with elevated FeNO may be more prone to exhibit airway hyper-responsiveness, a key characteristic of asthma. Comparable links between FeNO and airway hyper-responsiveness have been observed in healthcare workers as well as apprentices in the bakery, pastry and hairdressing industries [26, 27], suggesting that WAS in woodworkers is probably characterized by a combination of non-allergic and allergic pathways. Further research is required to elucidate the underlying pathophysiological processes involved in asthma triggered by different wood species [28]. While allergic sensitization has been demonstrated for certain wood species [2], TH2 hypersensitivity is not considered a major mechanism, since only 20% of affected individuals demonstrate specific IgE sensitization [28]. In addition, sensitization rates appear to be considerably more elevated for tropical wood types (30%) versus non-tropical wood types (3%) [29]. Other studies suggest that individuals working with low molecular weight (LMW) agents such as wood particulate, are more prone to experience a tight chest at work, daily sputum production and delayed asthmatic responses, suggesting the possible role of non-IgE-mediated mechanisms as well [30].

In this study, the prevalence of WONS (40%) was notably higher than WAS (14%), a pattern consistent with previous reports, in which work-related chest symptoms have been reported in 6–14% of workers [25, 31]. Exposure to wood particulate was reported as the primary cause of work-related lower (82%) and upper respiratory symptoms (90%), further suggesting that irritative mechanisms may play a significant role in the respiratory effects of wood dust exposure. Furthermore, since the study demonstrated that WAS were strongly associated with both FeNO and FEV₁, it is probable that this was indicative of underlying undiagnosed asthma. The elevated levels of airborne wood particulate ($GM = 3.29 \text{ mg/m}^3$) observed in these workers is therefore likely to contribute to the predominance of irritative respiratory symptoms [28].

Among host-related risk factors, atopy showed a positive correlation with WONS and high FeNO, aligning with other research, which has shown robust relationships with atopy and ocular-nasal symptoms in workers of furniture and sawmill factories [7, 8]. In addition, women tended to report elevated ASSs and WONS. This is consistent with recent studies demonstrating that women have twice the odds of experiencing upper airway symptoms [7]. Other investigators have also proposed that this higher preponderance of occupational allergic reactions in women may be influenced by sociocultural roles and variations in allergen exposure stemming from particular job distribution trends based on gender [32]. Although current smokers were more likely to have WAS, the impact of smoking on asthma attributed to occupations is still unclear, with contradictory results reported across different wood-working and industrial settings [33].

With regard to occupational risk factors, the study identified a notable disparity in the correlation between employment duration and airborne wood particulate levels. The average employment duration (7–8 years) in this study falls within the mid-range observed for other African studies (4–12 years) [9, 34, 35] but it is significantly shorter than that reported in European studies (8–19 years) [25, 36, 37]. However, the airborne wood exposure levels recorded in this study as well as other African studies ($3.29\text{--}10.14 \text{ mg/m}^3$) [9, 13, 38] were markedly higher than European studies ($0.09\text{--}1.9 \text{ mg/m}^3$) [25, 39–41]. This suggests that African woodworkers experience higher exposures over a shorter duration, potentially leading to earlier onset of respiratory symptoms, which is likely to be due to poorer working conditions and inadequate control measures. Furthermore, while employment duration was consistently associated with increased odds of WAS and lung function impairment, these associations did not reach statistical significance.

This investigation is the first to identify an association between Missanda, Panga-panga and Mahogany bean wood dust particulate and WONS, while exposure to Mutondo wood was linked to both WONS and WAS. Earlier studies have shown that wood types of Pine, Mahogany, African Teak, Red Cedar and Imbuia are commonly linked to increased rates of asthma and rhinitis [5].

The current study also demonstrated that workers engaged in equipment cleaning were less likely to have $ASS \geq 2$. However, personal protective equipment (PPE) use and participation in health and safety training correlated with greater likelihood of airway symptoms attributed to work. The negative correlation between equipment cleaning and asthma symptoms aligns with previous research findings, which showed that wet cleaning methods and implementation of local exhaust ventilation are correlated with reduced dust levels [13]. However, the positive association between PPE use, health and safety training and work-related symptoms is at variance with other studies that have demonstrated protective effects of PPE [9, 42]. Since this study could not establish temporality due to its cross-sectional design, the findings suggest that workers experiencing higher dust exposures may have adopted PPE use as a result of these conditions, or that PPE may have been used inconsistently or improperly. Additional studies are required to assess how effective these interventions are within the scope of wood processing operations.

Overall, the strength of this study includes its comprehensive assessment of risk factors using multiple asthma indices, including bronchial reversibility and FeNO levels to assess airway inflammation. However, the cross-sectional design limited its ability to investigate temporal relationships between exposure and respiratory outcomes. Moreover, the healthy worker effect, potentially resulted in underestimating the actual impact of wood dust on airways disease in this group, since the adverse health outcomes of non-respondents were not known. Furthermore, due to resource constraints in remote rural settings no formal diagnosis of WAS was made in affected workers, the study relied on epidemiological definitions instead.

This study confirms that respiratory symptoms and airway disease are common among Mozambiquan wood-processing workers and are strongly correlated with occupational exposures. The results reveal that the asthma disease burden among these

processors is at the higher end estimates observed in African studies and that IgE and non-IgE mediated asthma phenotypes occurred in comparable proportions. Furthermore, exposure to wood dust from specific indigenous species emerged as a key determinant of work-related symptoms. Gaining a deeper insight into the mechanisms of asthma, in relationship to other host and occupational factors, could aid in creating more effective preventive strategies to reduce the impact of obstructive lung disease in this sector. These findings further support the implementation of workplace controls to reduce exposures based on international standards, medical surveillance of those at high risk of airway disease and improved education and training of both workers and managers. Additional studies are needed to enhance our insights into the allergenic potential of indigenous hardwood species so as to inform evidence-based policy interventions [5].

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COMPETING INTERESTS

None declared.

REFERENCES

1. Dias M, Gomes B, Cervantes R *et al.* Microbial occupational exposure assessments in sawmills—a review. *Atmosphere* 2022;13:266.
2. Wiggans RE, Evans G, Fishwick D, Barber CM. Asthma in furniture and wood processing workers: a systematic review. *Occup Med (Lond)* 2016;66:193–201.
3. Dalbøge A, Albert Kolstad H, Ulrik CS *et al.* The relationship between potential occupational sensitizing exposures and asthma: an overview of systematic reviews. *Ann Work Expo Health* 2023;67:163–181.
4. Chamba P, Nunes E. Work-related asthma among workers in the wood-processing industry: a review. *Curr Allergy Clin Immunol* 2016;29:110–117.
5. Baatjies R, Chamba P, Jeebhay MF. Wood dust and asthma. *Curr Opin Allergy Clin Immunol* 2023;23:76–84.
6. Chaiear N, Ngoencharee J, Saejiw N. Respiratory symptoms and pulmonary function among workers in a rubber wood sawmill factory in Thailand. *Am J Public Heal Res* 2018;6:65–71.
7. Jacobsen G, Schaumburg I, Sigsgaard T, Schlünssen V. Wood dust exposure levels and respiratory symptoms 6 years apart: an observational intervention study within the Danish furniture industry. *Ann Work Expo Health* 2021;65:1029–1039.

8. Bolund ACS, Miller MR, Jacobsen GH, Sigsgaard T, Schlünssen V. New-onset COPD and decline in lung function among wood dust-exposed workers: re-analysis of a 6-year follow-up study. *Ann Work Expo Health* 2018;20:1–13.
9. Awoke TY, Takele AK, Mekonnen WT *et al.* Assessment of dust exposure and chronic respiratory symptoms among workers in medium scale woodwork factories in Ethiopia; a cross sectional study. *BMC Public Health* 2021;21:309–312.
10. Bose AK, Kadam DD. Respiratory morbidities among wood workers: an epidemiological study from the informal sector. *Int J Acad Med* 2021;7:206–211.
11. Hosseini DK, Nejad MVS, Sun H, Hosseini HK, Adeli SH, Wang T. Prevalence of respiratory symptoms and spirometric changes among non-smoker male wood workers. *PLoS One* 2020;15:e0224860.
12. Nafisa YW, Dawud FA, Sulaiman I, Salisu AI, Tukur MA. Influence of varying degree of wood dust exposure on pulmonary function and respiratory symptoms among wood workers in Kano, North Western Nigeria. *Niger J Physiol Sci* 2020;35:161–165.
13. Chamba PS, Baatjies R, Singh TS, Cumbane AJ, Jeebhay MF. Exposure characterisation of wood dust particulate, endotoxins and (1–3)- β -D-glucans, and their determinants in Mozambique wood processing workers. *Ann Work Expo Health* 2023;67:485–495.
14. Pekkanen J, Sunyer J, Anto JM, Burney P; European Community Respiratory Health Study. Operational definitions of asthma in studies on its aetiology. *Eur Respir J* 2005;26:28–35.
15. Le Moual N, Varraso R, Siroux V, *et al.* Domestic use of cleaning sprays and asthma activity in females. *Eur Respir J* 2012;40:1381–1389.
16. Graham BL, Steenbruggen I, Barjaktarevic IZ *et al.* Standardization of spirometry 2019 update an official American Thoracic Society and European Respiratory Society technical statement. *Am J Respir Crit Care Med* 2019;200:E70–E88.
17. Stanojevic S, Kaminsky DA, Miller MR *et al.* ERS/ATS technical standard on interpretive strategies for routine lung function tests. *Eur Respir J* 2022;60:2101499.
18. Tuomisto LE, Ilmarinen P, Lehtimäki L, Tammola M, Kankaanranta H. Immediate bronchodilator response in FEV₁ as a diagnostic criterion for adult asthma. *Eur Respir J* 2019;53:1–7.
19. ATS and ERS. ATS/ERS recommendations for standardized procedures for the online and offline measurement of exhaled lower respiratory nitric oxide and nasal nitric oxide, 2005. *Am J Respir Crit Care Med* 2005;171:912–930.
20. Dweik RA, Boggs PB, Erzurum SC *et al.* An official ATS clinical practice guideline: interpretation of exhaled nitric oxide levels (FENO) for clinical applications. *Am J Respir Crit Care Med* 2011;184:602–615.
21. Sunyer J, Pekkanen J, Garcia-Esteban R *et al.* Asthma score: predictive ability and risk factors. *Allergy* 2007;62:142–148.
22. Sá-Sousa A, Pereira AM, Almeida R *et al.* Adult asthma scores—development and validation of multivariable scores to identify asthma in surveys. *J Allergy Clin Immunol Pract* 2019;7:183–190.e6.
23. Fante D, Mariam TG, Mulat E, Demissie WR; Jimma University. Prevalence of respiratory disorders among woodworkers in Jimma Town, Southwest Ethiopia. *PMRR* 2019;5:1–6.
24. Jones R. The scale of the problem of obstructive lung disease in Africa becomes clearer, but where are the solutions? *Eur Respir J* 2018;51:1702562.
25. Wiggans RE, Sumner J, Robinson E. Respiratory symptoms, lung function and sensitisation across different exposure groups of British woodworkers. *Thorax* 2017;72:A1–278.
26. Mwanga HH, Baatjies R, Singh T, Jeebhay MF. Work-related allergy and asthma associated with cleaning agents in health workers in Southern African tertiary hospitals. *Am J Ind Med* 2022;65:382–395.
27. Tossa P, Paris C, Zmirou-Navier D *et al.* Increase in exhaled nitric oxide is associated with bronchial hyperresponsiveness among apprentices. *Am J Respir Crit Care Med* 2010;182:738–744.
28. Schlunssen V, Sigsgaard T, Raulf-Heimsoth M, Kespohl S. Workplace exposure to wood dust and the prevalence of wood-specific sensitization. *Allergologie* 2012;35:402–412.

29. Kespohl S, Ochmann U, Maryska S *et al.* Diagnostic algorithm for IgE mediated wood dust allergy. *AL* 2016;39:168–174.
30. Vandenplas O, Godet J, Hurdubaea L *et al.* Are high- and low-molecular weight sensitizing agents associated with different clinical phenotypes of occupational asthma? *Allergy* 2019;74:261–272.
31. Jacobsen G, Schlünssen V, Schaumburg I, Sigsgaard T. Increased incidence of respiratory symptoms among female woodworkers exposed to dry wood. *Eur Respir J* 2009;33:1268–1276.
32. Moscato G, Apfelbacher C, Brockow K *et al.* Gender and occupational allergy: report from the task force of the EAACI Environmental and Occupational Allergy Interest Group. *Allergy Eur J Allergy Clin Immunol* 2020;75:2753–2763.
33. Jeebhay MF, Ngajilo D, Le Moual N. Risk factors for nonwork-related adult-onset asthma and occupational asthma: a comparative review. *Curr Opin Allergy Clin Immunol* 2014;14:84–94.
34. Asgedom AA, Bråtveit M, Moen BE. High prevalence of respiratory symptoms among particleboard workers in Ethiopia: a cross-sectional study. *Int J Environ Res Public Health* 2019;16:10.
35. Jabur B, Ashuro Z, Abaya SW. Chronic respiratory symptoms and lung function parameters in large-scale wood factory workers in Addis Ababa, Ethiopia: a comparative cross-sectional study. *Int Arch Occup Environ Health* 2022;95:1221–1230.
36. Douwes J, McLean D, Slater T, Pearce N. Asthma and other respiratory symptoms in New Zealand pine processing sawmill workers. *Am J Ind Med* 2001;39:608–615.
37. Paraskevaidou K, Porpodis K, Kontakiotis T, Kioumis I, Spyrtos D, Papakosta D. Asthma and rhinitis in Greek furniture workers. *J Asthma* 2021;58:170–179.
38. Asgedom A, Bråtveit M, Schlünssen V, Moen EB. Exposure to inhalable dust, endotoxin and formaldehyde in factories processing particleboards from eucalyptus trees in Ethiopia. *Environ Occup Health Pract* 2020;2:1–10.
39. Straumfors A, Olsen R, Daae HL *et al.* Exposure to wood dust, microbial components, and terpenes in the Norwegian sawmill industry. *Ann Work Expo Health* 2018;62:674–688.
40. Straumfors A, Corbin M, McLean D *et al.* Exposure determinants of wood dust, microbial components, resin acids and terpenes in the saw- and planer mill industry. *Ann Work Expo Health* 2020;64:282–296.
41. Hagström K, Jacobsen G, Sigsgaard T, Schaumburg I, Erlandsen M, Schlunssen V. Predictors of monoterpene exposure in the Danish furniture industry. *Ann Occup Hyg* 2012;56:253–263.
42. Soongkhang I, Laohasiriwong W. Respiratory tract problems among wood furniture manufacturing factory workers in the northeast of Thailand. *Kathmandu Univ Med J* 2015;13:125–129.