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Atopy, asthma symptoms and eosinophilic airway inflammation in British woodworkers

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ABSTRACT

Objectives Despite reducing exposures to wood dust, woodworkers remain at increased risk of asthma. There have been no recent studies of wood dust exposure, respiratory symptoms or asthma in British woodworkers. This cross-sectional study examined factors associated with asthma in British woodworkers across exposure groups.

Methods Participants answered a reporter-delivered work and respiratory questionnaire, and underwent fractional exhaled nitric oxide (FE_{NO}), spirometry and specific IgE measurements. Wood dust exposure was assigned through a job-exposure matrix. Multiple regression evaluated associations between asthma and factors including exposure, atopy and current asthma symptoms (CAS).

Results A total of 269 woodworkers participated. Median wood dust exposure was 2.00 mg/m^3 (IQR 1.14 mg/m^3). CAS, work-related respiratory symptoms (WRRS) and eosinophilic airway inflammation (FE_{NO}>40 ppb) were common, present in 46%, 11% and 19% of the cohort, respectively. Atopic woodworkers were more likely to have nasal symptoms (OR 2.13, 95% CI 1.18 to 3.85, p<0.05), WRRS (OR 2.78, 95% CI 1.11 to 6.92, p<0.05), asthma (OR 3.40, 95% CI 1.49 to 7.81, p<0.01) and FE_{NO}>40 ppb (OR 2.00, 95% CI 1.03 to 3.88, p<0.05). No effect was seen for airflow obstruction. Symptomatic workers were more likely to have WRRS and asthma (OR 4.29, 95% CI 2.12 to 8.69, p<0.001) but not FE_{NO}>40 ppb or airflow obstruction. A dose-response effect with wood dust exposure was not seen.

Conclusions Asthma symptoms were prevalent among British woodworkers, even at low exposure levels. Atopy was associated with asthma, particularly among symptomatic woodworkers. Further studies should phenotype woodworkers at risk of asthma and inform approaches to reduce risk.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Woodworkers are at increased risk of asthma, and wood dust is a leading cause of occupational asthma in Great Britain.
⇒ No recent studies have described risk factors for asthma in British woodworkers.
⇒ Several factors have been associated with an increased risk of asthma in woodworkers, including atopy and wood dust exposure levels, but these findings have not been consistently replicated.

WHAT THIS STUDY ADDS

⇒ Woodworkers experience a high burden of upper airway, asthma and work-related symptoms, despite a reduction in exposure to wood dust.
⇒ Atopy, but not wood dust exposure, is associated with asthma symptoms and airway inflammation in woodworkers.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Continued surveillance of woodworkers for asthma is needed as symptoms are common.
⇒ The role of different wood species, fractional exhaled nitric oxide as a diagnostic and monitoring tool and asthma mechanisms in woodworkers warrants further exploration.

INTRODUCTION

Around 60 000 workers are employed in the UK woodworking industry.[1] Exposure to wood dust is associated with an increased risk of cough and bronchitis,[2] airway hyper-responsiveness,[3] excess lung function decline[4] as well as an increased risk of asthma.[5] Respiratory symptoms and specific IgE (SIgE) have been reported at wood dust exposures lower than the UK workplace exposure limit (WEL) for softwood of 5 mg/m^3 and mixed/hardwood of 3 mg/m^3,[2] and wood dust has been identified as an important occupational sensitiser.[6] The Health and Occupational Research surveillance scheme shows wood dust is a leading cause of occupational asthma (OA) in Great Britain. The extent to which asthma risk changes across the spectrum of wood dust exposure is not well understood,[7] but recent observational follow-up supports a reduction in symptoms with reducing exposure.[8]

Atopy has been associated with an increased risk of asthma in woodworkers, particularly for those with higher dust exposures.[3] Wood dust exposure appears to have a more significant impact on lung function among smokers, with the effect more pronounced at higher exposures.[4] However, evidence supporting the influence of atopy and smoking on asthma likelihood in woodworkers is inconsistently reported.[2 9 10]

Few recent studies have explored factors associated with asthma in woodworkers, and no recent studies have been conducted in the UK.[2] We aimed to identify factors associated with asthma in British

woodworkers, with an emphasis on asthma symptoms, atopy and wood dust exposure.

METHODS

Study population

Worksites were identified through participation in a Health and Safety Executive study and through the Corporate Operational Information System database between 2014 and 2018.[11 12] Worksites ranged from small (<10 employees) to large (>200 employees) and from sectors including furniture manufacture, carpentry, boat building and timber. All workers aged over 16 years and currently exposed to any type of wood dust were eligible.

Health outcomes

Workers underwent an interviewer-administered questionnaire detailing symptoms based on the Medical Research Council and European Community Respiratory Health Survey questionnaires.[13 14] Workers were asked about common ocular, nasal and respiratory symptoms including cough, wheeze, sputum production, breathlessness and chest tightness.

For each symptom, workers were asked whether these were worse at work or improved away from work or on holiday, and work-related symptoms were defined where one or more answers were positive. 'Cough' was defined as usual coughing during the day, 'ever wheeze' was defined as ever wheezing except during colds, 'chronic bronchitis' was defined as a cough with sputum for at least 3 months a year, 'nasal symptoms' as reporting regular itchy, blocked or runny nose and 'ocular symptoms' as symptoms of itchy, gritty, dry eyes or conjunctivitis.[8] Current asthma symptoms (CAS) were defined as wheezing, chest tightness, breathlessness or asthma medication use within the last 12 months.[14] Current asthma was defined as either a current or past physician diagnosis of asthma along with CAS according to European Community Respiratory Health Survey (ECRHS) criteria.[14] Ever asthma was defined either as a current or past self-reported asthma diagnosis. Asthma with latency was defined as adult-onset asthma with onset after first exposure in the woodworking industry.

Fractional exhaled nitric oxide (FE_{NO}) was performed before spirometry using a NOBreath device (Bedfont Scientific, Kent) according to the American Thoracic Society/European Respiratory Society (ATS/ERS) standards.[15] Spirometry was measured using an NDD Easy-On PC spirometer (Zurich, Switzerland) to ATS/ERS standards.[16] Subjects were examined sitting and without a nose clip. Blood samples were analysed for total IgE (TIgE) and specific IgE to hard (oak, mahogany and obeche) and soft (beech, pine, cedar and silver fir) woods using standard ImmunoCAP testing (Phadia, Sweden). Forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC), peak expiratory flow rate and FEV_1/FVC values falling 2 SD below the mean (lower limit of normal (LLN)) were considered abnormal.[17] FE_{NO} values >40 ppb were considered high and values >25 ppb intermediate.[18] Participants were considered sensitised if their IgE to hard or soft wood exceeded 0.35 kU/L.[19] Atopy was defined as a TIgE >100 kU/L.[20]

Exposure assessment

Full details of the hygiene study are published elsewhere.[12] In brief, a passive sampling device was worn on the lapel for the duration of a working shift and standard 8-hour time-weighted averages (8-hour TWA) calculated for inhalable wood dust for the tasks sampled. Wood dust exposures were assigned to each worker via a bespoke job exposure matrix (JEM) generated using a linear-effects model. Each worker was assigned a job code using task-specific codes developed for the woodworking industry.[11] Where workers held more than one current position (eg, a managerial and a manufacturing role), a ratio of time spent in each job was applied. In addition, Standard Occupational Classification 2010 codes were applied to current jobs using a Computer-Assisted Structured Coding Tool.[21 22] Inhalable wood dust exposures for each worker were estimated based on a linear mixed effects model fitted to the logarithm of the measured 8-hour TWA values. The site was treated as a random effect and task as a mixed effect, with between-worker variation treated as normally distributed residual errors. Mean exposure for each task at each site was calculated from the corresponding mean of log-exposure estimated by the model and the residual error SD. For individual workers, model exposures were then weighted by the fraction of time each worker spent undertaking each task.

Statistical analysis

Exposure was explored as both continuous (geometric mean exposure) and categorical (quartiles of exposure: 'lowest', 'low', 'high', 'highest'; 'high' vs 'low' defined as the upper three quartiles versus the lowest quartile; and exposure above or below the 2 mg/m^3, 3 mg/m^3 and 5 mg/m^3 exposure limit) variables.[8]

Normally distributed data were displayed as means and SD and compared using independent t-tests and analysis of variance. Exposure, TIgE and FE_{NO} data were not normally distributed and reported as medians with IQR. Mann-Whitney U tests were used to compare non-normally distributed data, and data were log-transformed for use in regression models, with back-transformed data reported in tables as geometric means. Categorical data were analysed using χ^2 tests.

Only technically acceptable spirometry and FE_{NO} measurements were used in the final analysis. The outcomes of interest, including respiratory symptoms, work-related respiratory symptoms (WRRS), current or ever asthma, FE_{NO}>40 ppb and FEV_1/FVC<LLN were used as dependent variables in logistic regression models. Wood dust exposure, atopy and CAS were identified as variables associated with asthma in woodworkers, with exposure and atopy identified a priori as predictors of asthma risk.[3] Models were adjusted for smoking, atopy, age, sex and respiratory protective equipment (RPE) use. Stratified analyses were conducted to assess associations between exposure, atopy and CAS on the asthma outcomes of interest.[3] Crude and adjusted ORs were reported with associated 95% CIs. All statistical analyses were performed using SPSS Statistics, V.23.

RESULTS

Two hundred and sixty-nine out of a possible 376 workers participated (participation rate 72%). Reasons for non-participation included inability to capture workers due to shift work, inability of workers to take time away from shift, worker annual leave and worker refusal to participate.

Full results of the hygiene study are reported elsewhere.[12] For worksites participating in the health study, median exposure to inhalable wood dust was 2.00 mg/m^3 (IQR 1.14), and 18 (7%) workers had a current exposure to wood dust >3 mg/m^3. All workers used seasoned (dry) wood. Fourteen (5%) workers self-reported current co-exposure to isocyanate-based paints or resins, epoxies, varnishes or glue.

Table 1 shows key demographic, exposure and clinical characteristics of the study population by presence of CAS. Most participants were male (n=261, 97%), with an average age of 42.4

Table 1 Key demographic, exposure and clinical characteristics of 269 British woodworkers, by presence of current asthma symptoms

	Current asthma symptoms (n=123)	No current asthma symptoms (n=146)	Total (n=269)
Demographics			
Age, years (SD)	42.3 (13.0)	42.5 (12.2)	42.4 (12.6)
Sex, male (%)	118 (96)	143 (98)	261 (97)
Current smoker, n (%)	39 (32)	31 (21)	70 (26)
Ever smoker, n (%)	39 (32)	41 (28)	80 (30)
BMI, kg/m^2 (SD)	27.83 (5.92)	26.17 (3.81)	26.93 (4.9
Exposure			
Hardwood exposure, n	35 (28)	41 (28)	76 (28)
Softwood exposure n (%)	14 (11)	11 (8)	25 (9)
Mixed exposure, n (%)	74 (60)	94 (64)	168 (62)
Uses RPE, n (%)	84 (68)	123 (84)†	207 (77)
Years exposed in woodworking industry (SD)	17.33 (12.50)	20.21 (12.94)*	18.89 (12.80)
Current exposure, mg/m^3 (IQR)	2.0 (1.3)	2.0 (0.9)	2.0 (1.1)
Exposure >5 mg/m^3, n (%)	2 (1)	1 (1)	3 (1)
Exposure >3 mg/m^3, n (%)	10 (8)	8 (6)	18 (7)
Exposure >2 mg/m^3, n (%)	65 (53)	95 (65)*	160 (60)
Exposure 'high' versus 'low', n (%)	89 (72)	113 (77)	202 (75)
Symptoms			
Ever wheeze, n (%)	66 (54)†	18 (12)	84 (31)
Cough, n (%)	58 (47)†	24 (16)	82 (31)
Chronic bronchitis, n (%)	17 (14)	4 (3)	21 (8)
Nasal symptoms, n (%)	36 (29)	35 (24)	71 (26)
Ocular symptoms, n (%)	42 (34)†	25 (17)	67 (25)
Any WRRS, n (%)	27 (22)†	2 (1)	29 (11)
WRNS, n (%)	16 (13)	19 (13)	35 (13)
WROS, n (%)	34 (28)	27 (19)	61 (23)
Asthma, atopy and sensitisation			
Current asthma, n (%)	40 (33)†	0 (0)	40 (15)
Ever asthma, n (%)	40 (33)†	15 (10)	55 (21)
Asthma with latency, n (%)	10 (8)	3 (2)	13 (5)
Asthma inhalers, n (%)	23 (19)†	0 (0)	23 (9)
Atopic symptoms, n (%)	67 (55)	62 (43)	129 (48)
Atopic, n (%)	80 (65)*	74 (51)	154 (57)
Total IgE, kU/L (IQR), n=246	46.0 (129.0)†	27.0 (62.0)	36.0 (69.8)
Positive SIgE to hard or soft wood, n (%)	0 (0)	1 (1)	1 (1)
FE_{NO} and spirometry			
$FE_{NO,}$ ppb (IQR)	19.3 (28.7)	18.7 (19.8)	18.7 (24.1)
FE_{NO}>25 ppb, n (%)	47 (40)	45 (32)	92 (36)
FE_{NO}>40 ppb, n (%)	27 (23)	23 (17)	50 (19)
FEV_1, L (SD)	3.7 (0.6)	4.0 (0.7)†	3.84 (0.72)
FEV_1<LLN, n (%)	2 (2)	1 (1)	3 (1)
FEV_1/FVC<LLN, n (%)	8 (7)	4 (3)	12 (4)

Number of workers in each group is reported in parentheses.
*Statistically significant at p<0.05.
†Statistically significant at p<0.01.
BMI, body mass index; FE_{NO}, fractional exhaled nitric oxide; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; LLN, lower limit of normal; RPE, respiratory protective equipment; SIgE, specific IgE; WRNS, work-related nasal symptoms; WROS, work-related ocular symptoms; WRRS, work-related respiratory symptoms.

(SD 12.6) years. Seventy (26%) workers were current smokers. One hundred and fifty-four workers (57%) were atopic and one-sixth of the study population (n=40, 15%) had current asthma. A significant proportion of the study population reported CAS (n=123, 46%). WRRS were less frequent at 11% (n=29). Thirteen workers (5%) had asthma with latency, with no difference between groups. Rates of SIgE sensitisation to wood dust were very low overall and not significantly different between groups.

Symptomatic workers had worked fewer years in the woodworking industry, were less likely to use RPE and fewer were exposed at levels >2 mg/m^3. Wheeze, cough, ocular symptoms and WRRS were more common among workers with CAS. Atopy and asthma were significantly more common in those with CAS: 33% of workers with CAS reported ever asthma vs 10% without (p<0.001). FEV_1 was significantly lower in those with CAS.

In logistic regression models, no positive dose-response was seen between increasing exposure to wood dust (either in quartiles of exposure, 'high' vs 'low' or above the 2 mg/m^3 threshold) and respiratory symptoms, CAS, asthma, airway inflammation or airflow obstruction (table 2 and online supplemental tables 1–3). Work at exposures >2 mg/m^3 was associated with significantly increased work-related nasal symptoms (WRNS) compared with below the 2 mg/m^3 threshold (adjusted OR 3.97, 95% CI 1.53 to 10.31, p=0.005, table 2). Conversely, workers in the 'high' exposure group and workers with exposures >2 mg/m^3 had lower odds for respiratory symptoms, CAS and WRRS compared with those in the lower exposure groups (tables 1 and 2, online supplemental tables 1–3).

Atopic workers had significantly higher odds of nasal and ocular symptoms, WRRS, CAS and current asthma (table 3). However, no associations were seen between atopy and cough, ever wheeze or chronic bronchitis (online supplemental table 4). Atopic woodworkers had increased odds of airway inflammation defined by an FE_{NO}>40 ppb (unadjusted OR 2.00, 95% CI 1.03 to 3.88, p=0.04), but no effect was seen for airflow obstruction (adjusted OR 0.8, 95% CI 0.24 to 2.69, online supplemental table 4).

Symptomatic woodworkers had increased odds of ocular symptoms and asthma (table 4). In controlled analyses, symptomatic workers were at increased odds of ocular symptoms (OR 2.94, 95% CI 1.59 to 5.43, p<0.001) and tended towards increased odds of WRNS and work-related ocular symptoms (WROS), although this did not reach significance. Workers with CAS were significantly more likely to self-report ever asthma (OR 4.29, 95% CI 2.12 to 8.69, p<0.001). No clear associations were seen for CAS and FE_{NO} or airflow obstruction.

Stratified analyses were conducted to explore associations between symptoms and atopy and controlled for exposure and other confounders (table 5). Woodworkers with both CAS and atopy were more likely to have WRRS (OR 8.61, 95% CI 3.55 to 20.84, p<0.001), current asthma (OR 15.86, 95% CI 6.82 to 36.89, p<0.001) and a high FE_{NO}>40 ppb (OR 2.44, 95% CI 1.27 to 4.67, p<0.01). No associations were seen for asthma with latency or airflow obstruction. In workers with CAS and no atopy, no significant associations were seen with variables of interest. In the absence of CAS, atopic woodworkers were significantly less likely to have WRRS (OR 0.08, 95% CI 0.01 to 0.59, p<0.05) and no relationship was seen with current asthma or FE_{NO}.

DISCUSSION

Asthma symptoms were prevalent among British woodworkers, even at low exposure levels. Symptoms of cough and wheeze were present in around 30% of the study population, and asthma was present in 15%, levels higher than would be expected in adults in the general population,[23] and in similar proportion to those reported in a recent study of Scandinavian woodworkers.[8] Atopy was a significant modifying factor in associations between asthma symptoms, WRRS, asthma and airway inflammation.

Table 2 Adjusted logistic regression models showing associations between wood dust exposure, respiratory symptoms, upper airway symptoms and asthma-related outcomes

	Ever wheeze	CB	WRNS or WROS	WRNS	WROS	Any WRRS	Current asthma symptoms	Current asthma
GM current exposure, mg/m^3 (95% CI)	1.01 (0.62 to 2.43)	1.33 (0.60 to 44.87)	1.41 (0.73 to 3.67)	1.14 (0.55 to 3.69)	0.96 (0.43 to 3.05)	0.76 (0.18 to 2.16)	1.07 (0.66 to 2.48)	1.67 (0.71 to 5.43)
'High' versus 'low'	1.17 (0.61 to 2.25)	0.21 (0.03 to 1.56)	1.83 (0.83 to 4.08)	2.44 (0.77 to 7.79)	2.15 (0.68 to 6.84)	0.16 (0.03 to 0.81)*	0.83 (0.38 to 1.82)	0.55 (0.16 to 1.93)
Current exposure >2 mg/m^3 (yes vs no)	0.49 (0.23 to 1.08)	0.16 (0.01 to 1.79)	2.81 (1.39 to 5.66)†	3.97 (1.53 to 10.31)†	1.37 (0.60 to 3.11)	0.39 (0.17 to 0.89)*	0.66 (0.39 to 1.10)	0.69 (0.34 to 1.39)

ORs and 95% CIs are shown in parentheses.
Models controlled for age, sex, smoking, atopy and RPE use.
'High' versus 'low' defined as the upper three quartiles versus the lowest quartile.
*Statistically significant at p<0.05.
†Statistically significant at p<0.01.
CB, chronic bronchitis; GM, geometric mean; WRNS, work-related nasal symptoms; WROS, work-related ocular symptoms; WRRS, work-related respiratory symptoms.

When stratified by the presence or absence of atopy, woodworkers with CAS had significantly higher odds of WRRS, current asthma and airway inflammation only when atopy was also present.

Asthma symptoms were prevalent in the current study, with rates comparable to recent systematic reviews.[2 9] Woodworkers have been demonstrated to be at increased risk of asthma symptoms in several workplace studies.[24 25] Female, but not male, woodworkers with higher exposures have been shown to have an increased risk for chronic bronchitis, although data from other studies are conflicting.[24] Since female representation in the current study was very low (3%), analysis on the effects of gender was not possible. It is important for individuals, employers, occupational health practitioners and respiratory physicians to be aware of the risk of asthma in woodworkers of both genders and support measures to reduce this.

The presence of atopy was associated with increased nasal and ocular symptoms, WRRS, CAS and asthma. Symptomatic and atopic woodworkers were significantly more likely to have WRRS suggesting that these individuals may be at increased risk of work-related asthma. Similarly, the same group of workers had higher odds of asthma. This is in contrast to previous research suggesting non-atopic (rather than atopic) woodworkers are at increased risk of WRRS.[3] The current study differs through a smaller proportion of female workers (3% in the current study vs 26%), wood species exposure (mixed vs solely soft wood) and dust exposure levels (median 2 mg/m^3 vs 0.96 mg/m^3). Higher dust exposures or mixed species exposures (to wood composites) may explain these differences in findings. Atopy is well known to be a risk factor for asthma, in particular T-helper 2 high disease, and atopy is common among the UK population. Introducing pre-employment screening based on atopy may cause ethical challenges as this is unlikely to offer advantages to individual workers, since identifying those who will develop disease is difficult. Preselection of workers away from woodworking based on atopic status is not recommended.[26]

Sensitisation may precede or coincide with the development of asthma. Wood dust contains both high-molecular weight (HMW) and low-molecular weight (LMW) allergens.[27] A minority of wood species are associated with immunological proteins and production of SIgE that is more often seen in HMW OA.[2] In contrast, mechanisms in western red cedar OA are not associated with SIgE and are associated with plicatic acid.[2] Wood dust has been associated with early, late and dual specific inhalation challenge (SIC) responses in OA.[28 29] Atopy is associated with an increased risk of OA, particularly in HMW but not LMW-exposed workers.[30] The low levels of specific sensitisation observed in the current study support the lack of a measurable IgE response. Characterisation of SIgE responses to wood dust, and the underlying mechanisms associated with developing allergic disease, has recently been highlighted as important areas of further study.[5]

Atopy was also associated with an increased risk of eosinophilic airway inflammation defined by a FE_{NO}>40 ppb. Atopic

Table 3 Crude and adjusted logistic regression comparing associations between atopy and nasal and ocular symptoms, current asthma symptoms, work-related symptoms, airway inflammation and airflow obstruction

	NS	OS	CAS	WRNS/WROS	WRRS	Current asthma	FE_{NO}>40 ppb
Crude OR Atopy (yes vs no)	1.97 (1.11 to 3.51)*	2.09 (1.15 to 3.77)*	1.81 (1.11 to 2.96)*	1.57 (0.87 to 2.85)	2.57 (1.06 to 6.25)*	3.51 (1.55 to 7.94)†	2.00 (1.03 to 3.88)*
Adjusted for exposure 'high' versus 'low'	1.99 (1.12 to 3.55)*	2.10 (1.16 to 3.79)*	1.81 (1.10 to 2.96)*	1.59 (0.88 to 2.90)	2.58 (1.06 to 6.28)*	3.51 (1.55 to 7.94)†	2.02 (1.04 – 3.94)*
Adjusted for age, sex, smoking	2.12 (1.18 to 3.83)*	2.19 (1.20 – 4.01)*	1.98 (1.19 – 3.28)†	1.68 (0.91 to 3.08)	2.76 (1.11 – 6.85)*	3.38 (1.48 – 7.73)†	1.86 (0.94 to 3.66)
Adjusted for above plus RPE use	2.13 (1.18 – 3.85)*	2.22 (1.21 – 4.07)†	2.02 (1.20 – 3.38)†	1.68 (0.91 to 3.10)	2.78 (1.11 – 6.92)*	3.40 (1.49 – 7.81)†	1.86 (0.94 to 3.68)

Workers with no atopy were the control group. ORs and 95% CIs are shown in parentheses.
'High' versus 'low' defined as the upper three quartiles versus the lowest quartile.
*Statistically significant at p<0.05.
†Statistically significant at p<0.01.
CAS, current asthma symptoms; CB, chronic bronchitis; FE_{NO}, fractional exhaled nitric oxide; NS, nasal symptoms; OS, ocular symptoms; RPE, respiratory protective equipment; WRNS, work-related nasal symptoms; WROS, work-related ocular symptoms; WRRS, work-related respiratory symptoms.

Table 4 Crude and adjusted logistic regression models between current asthma symptoms, work-related symptoms, airway inflammation and airflow obstruction

	Nasal symptoms	Ocular symptoms	WRNS/WROS	Ever asthma	FE_{NO}>40 ppb	FEV_1/FVC<LLN
Crude OR CAS (yes vs no)	1.31 (0.76 to 2.26)	2.51 (1.42 to 4.44)*	1.68 (0.95 to 2.99)	4.18 (2.17 to 8.03)*	1.48 (0.80 to 2.75)	2.60 (0.76 to 8.88)
Adjusted for exposure	1.35 (0.78 to 2.33)	2.57 (1.45 to 4.55)*	1.77 (0.99 to 3.16)	4.19 (2.17 to 8.07)*	1.53 (0.82 to 2.85)	2.54 (0.74 to 8.72)
Adjusted for age, sex, smoking, BMI	1.36 (0.78 to 2.36)	2.75 (1.53 to 4.94)*	1.77 (0.98 to 3.19)	4.84 (2.45 to 9.56)*	1.79 (0.94 to 3.40)	2.04 (0.58 to 7.26)
Adjusted for atopy	1.22 (0.69 to 2.15)	2.56 (1.40 to 4.57)*	1.66 (0.91 to 3.01)	4.31 (2.15 to 8.64)*	1.65 (0.86 to 3.18)	2.22 (0.61 to 8.09)
Adjusted for above plus RPE use	1.28 (0.72 to 2.30)	2.94 (1.59 to 5.43)*	1.82 (0.99 to 3.36)	4.29 (2.12 to 8.69)*	1.82 (0.93 to 3.59)	1.84 (0.49 to 6.93)

Workers with no current asthma symptoms were the control group. ORs and 95% CIs are shown in parentheses.
*Statistically significant at p<0.01.
BMI, body mass index; CAS, current asthma symptoms; FE_{NO}, fractional exhaled nitric oxide; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; LLN, lower limit of normal; RPE, respiratory protective equipment; WRNS, work-related nasal symptoms; WROS, work-related ocular symptoms.

individuals are well-documented to have a higher FE_{NO}.[15] Airway inflammation was common among the study population and almost a fifth of workers had an FE_{NO}>40 ppb. A FE_{NO}>40 ppb has been shown to have a sensitivity of 78.6%–88.3% and specificity of 82.6%–89.5% for predicting asthma in symptomatic individuals.[31] In the current study, 23% of workers with CAS had a FE_{NO}>40 ppb and 40% had a FE_{NO}>25 ppb. Workers with CAS and atopy were significantly more likely to have a FE_{NO}>40 ppb in adjusted analyses (OR 2.44, 95% CI 1.27 to 4.67, p<0.01). In contrast, no significant relationships were observed with airflow obstruction. Spirometry is more commonly used as both a diagnostic and surveillance tool in workers at risk of asthma. FE_{NO} has been demonstrated to identify early bronchial hyperresponsiveness in groups of occupationally exposed apprentices, even when spirometry is normal,[32] and it is recommended as a first-line objective test for asthma in recently updated UK guidance.[23] The development of obstructive lung function is a late sign in asthma and, when present, may be irreversible.[33] The utility of FE_{NO} in occupational settings is not established, but this research suggests it could be useful in the identification of early airway inflammation and should be further explored.

Most participants had neither a raised FE_{NO} nor obstructive airways disease. Although these measurements were made cross-sectionally, the absence of airway inflammation or airflow obstruction may suggest an explanation other than airways disease for respiratory symptoms.[10] Respiratory irritants, alternative respiratory diagnoses or alternative exposures may account for the presence of symptoms and longitudinal studies are needed to examine whether workers will develop lung disease.[5]

Exposure to wood dust, either as a continuous or dichotomised variable, was not clearly associated with increased odds of asthma symptoms or asthma but was associated with increased odds of WRNS and WROS and reduced odds of WRRS. In the current study, workers were also exposed to other irritants or allergens including glues, solvents, paints and resins, and a small number also worked with isocyanates. Exposure attribution may therefore explain the lack of dose response. Current dust exposures were used in analyses, but no data were available on previous dust exposures. The intensity of exposure to wood dust may be as important as overall exposure when assessing asthma risk and longitudinal exposures would enhance future exposure attribution and assessment.[34]

The lack of a clear dose-response relationship may also be explained by the healthy worker effect (HWE).[35] Workers with CAS had significantly less time exposed in the woodworking industry, suggesting attrition of workers with respiratory symptoms. Furthermore, there were few people within the study with airflow obstruction, suggesting these people have left the workplace. Although asthma can occur after many years' exposure to an occupational allergen, it is more common in the first years of exposure.[36] Workers had spent an average of 19 years within the woodworking industry, increasing the influence of the HWE. The lower likelihood of WRRS and CAS in those with higher exposures may point to early exit from the workforce for those with symptoms, and cohort studies are required to further examine the HWE phenomenon among woodworkers, especially at lower exposure levels.

The Control of Substances Hazardous to Health 2002 regulations require exposure to occupational asthmagens to be as low as reasonably practicable. We found median wood dust exposures of 2 mg/m^3, lower than both the 3 mg/m^3 mixed/hardwood and 5 mg/m^3 softwood UK WELs.[12 37] Our findings support data from the UK and across Europe suggesting there has been a reduction in wood dust exposure over time, reflecting changes in processing, employment and safety measures.[1] Data suggest the UK has higher average wood dust exposures compared with other European countries, which may explain the high prevalence of OA caused by exposure to wood dust.[1]

Table 5 Adjusted logistic regression models showing odds of work-related respiratory symptoms, asthma, airway inflammation and airflow obstruction stratified by presence of atopy and/or current asthma symptoms

	WRRS	Current asthma	Asthma with latency	FE_{NO}>40 ppb	FEV_1/FVC<LLN
CAS and atopy	8.61 (3.55 to 20.84)*	15.86 (6.82 to 36.89)*	1.06 (0.29 to 3.80)	2.44 (1.27 to 4.67)*	1.72 (0.51 to 5.76)
CAS no atopy	1.43 (0.52 to 3.88)	1.36 (0.57 to 3.26)	1.01 (0.14 to 7.12)	0.52 (0.17 to 1.58)	1.38 (0.37 to 5.81)
Atopy no CAS	0.08 (0.01 to 0.59)	No data	1.16 (0.27 to 5.22)	0.76 (0.36 to 1.58)	0.37 (0.04 to 3.13)

ORs and 95% CIs are shown in parentheses.
Models controlled for age, sex, smoking, RPE use and wood dust exposure ('high' vs 'low').
*Statistically significant at p<0.01.
CAS, current asthma symptoms; FE_{NO}, fractional exhaled nitric oxide; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; LLN, lower limit of normal; RPE, respiratory protective equipment; WRNS, work-related nasal symptoms; WRRS, work-related respiratory symptoms.

Limitations

Cross-sectional studies of this kind are liable to exposure misclassification and are influenced by other factors such as workforce, factory size and health and safety practices.[38] Exposure misclassification on polytomous scales may dilute effect sizes at higher exposures, and thus lead to error biases towards the null.[39] We used a detailed JEM based on empirical measurements to reduce exposure misclassification and included health and safety practices in models to reduce confounding. Both current and cumulative wood dust exposure have been associated with increased respiratory symptoms and asthma, and exposure-response relationships have been reported to vary with different wood species.[5] In addition to wood dust, workers in the current study may have been exposed to glues, solvents, paints, resins or isocyanates. These substances are both irritant and allergenic and may explain the high rates of respiratory symptoms across exposure groups and the lack of a dose-dependent relationship. Measures of airway inflammation and lung function were only collected at a single time point, compared with symptom data which spanned 12 months or more. Cross-sectional studies may be influenced by selection bias, where symptomatic workers are more likely to participate. Longitudinal studies are needed to explore whether symptoms are related to the development of abnormal airways physiology and to better understand the mechanisms of wood dust OA.

Definitions for atopy and sensitisation vary across studies and may impact comparability. Atopy is commonly defined through the presence of skin-prick positivity, which was not performed in this study. However, a TIgE >100 kU/L has been shown to have good positive predictive value for at least one positive allergy test in individuals with asthma symptoms.[20] Compared with skin-prick tests, total IgE correlates better with the total allergic component of asthma and is more easily comparable between populations.[40] Since rates of SIgE sensitisation were low in the study, it is unlikely they interfered with TIgE results. Even where specific wood allergens have been used in patients with confirmed wood dust OA, specific sensitisation rates have been low, suggesting an IgE response is not significant.[19] Where positive SIgE results are present, they may be helpful in identifying sensitisation to wood species, but the current findings suggest that a negative IgE to wood dust is unlikely to be helpful in excluding asthma in exposed woodworkers.

CONCLUSION

CAS are prevalent at levels of wood dust exposure lower than the current UK WEL. Atopy was a significant modifier for asthma and airway inflammation among woodworkers, particularly among symptomatic workers. Further studies to phenotype and endotype populations of woodworkers at risk of, and suffering from, asthma will inform future approaches to screening and diagnosis in these populations. Longitudinal studies are required to monitor wood dust exposures on a local, regional, national and international scale to understand how this influences respiratory symptoms and disease in exposed populations.

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