Asthma in furniture and wood processing workers:  
a systematic review

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Background  Wood dust is a common cause of occupational asthma. There is potential for high exposure to wood dust during furniture and wood manufacturing processes.

Aims  To evaluate the evidence for non-neoplastic respiratory ill health associated with work in the furniture and wood manufacturing sector.

Methods  A systematic review was performed according to PRISMA guidelines. Articles were graded using SIGN (Scottish Intercollegiate Guideline Network) and MERGE (Methods for Evaluating Research Guidelines and Evidence) criteria, with data grouped by study outcome.

Results  Initial searches identified 1328 references, from which 55 articles were included in the review. Fourteen studies were graded A using MERGE or >2++ using SIGN. All but one paper describing airway symptoms reported an increased risk in higher wood dust exposed workers in comparison to lower or non-exposed groups. Five studies reporting asthma examined dose response; three found a positive effect. The relative risk for asthma in exposed workers in the single meta-analysis was 1.5 (95% CI 1.25–1.87). Two studies reported more obstructive lung function (forced expiratory volume in 1 s [FEV1]/forced vital capacity < 0.7) in exposed populations. Excess longitudinal FEV1 decline was reported in female smokers with high wood dust exposures in one study population. Where measured, work-related respiratory symptoms did not clearly relate to specific wood immunoglobulin E positivity.

Conclusions  Work in this sector was associated with a significantly increased risk of respiratory symptoms and asthma. The evidence for wood dust exposure causing impaired lung function is less clearly established. Further study is required to better understand the prevalence, and causes, of respiratory problems within this sector.

Key words  Furniture; occupational asthma; systematic review; wood manufacturing.

Introduction  

In Great Britain, an estimated 380,000 people were occupationally exposed to wood between 2000 and 2003 [1]. A number of respiratory diseases have been associated with exposure to wood dust. Chronic obstructive pulmonary disease has been described in epidemiological and cross-sectional studies of construction and woodworkers [2,3]. Extrinsic allergic alveolitis has also been reported, mainly attributed to exposure to microbial contaminants [4,5]. A higher incidence of idiopathic pulmonary fibrosis has also been found in woodworkers than in matched controls and an increased risk of lung cancer has been reported recently in wood dust-exposed groups [6,7]. Finally, exposure to wood dust is associated with an increased risk of occupational asthma (OA). Prevalence of OA in some populations of wood workers has been estimated at 5%, and a significant number of workers developing the condition continue to suffer symptoms long after their exposure has ceased [8,9].

Occupational exposure to wood dust in the UK may have been declining over time, but data from the Surveillance of Work-related and Occupational Respiratory Disease (SWORD) database implicate wood dust as an increasingly common cause of OA [10,11]. However, the mechanism by which wood dust causes
asthma remains poorly understood [12]. Possible causative agents, including plicatic acid, monoterpenes and endotoxin exposure, have been suggested [13–15]. Plicatic acid is a low-molecular-weight agent shown to be directly toxic to pulmonary epithelium and causes histamine release from the basophils of workers with western red cedar asthma [13,14]. Terpenes are found in softwoods such as pine and have been linked with both irritant and allergic respiratory disease, whereas endotoxin exposure has been described predominantly where wood is wet and therefore more likely to be colonized with mould (though endotoxin exposure in dry wood environments has also been reported) [15–17]. Formaldehyde-based stabilizers used in wood composite manufacture have also been implicated as respiratory sensitizers [18]. Some authors have identified allergic disease processes for specific woods [19,20]. However, a single unifying immunological mechanism has yet to be described.

A variety of wood types including hardwoods, softwoods and wood composites are in frequent industrial use. Hard and soft wood can be used both wet (unseasoned or recently cut, for example, in the sawmill and timber industries) and dry (air or kiln-dried, as in furniture manufacture). A large number of UK workers occupationally exposed to wood are employed in furniture and wood manufacturing [1]. However, much of the data on the short- and long-term asthmagenic effects of wood has come from studies of wet wood industries with an emphasis on western red cedar workers and the actions of plicatic acid [8,9,21]. Western red cedar is uncommon in the UK, where a variety of other hard and soft wood species are used more frequently [1]. Consequently, the aim of this review was to evaluate the relationship between respiratory ill health, and particularly asthma, with working in the furniture and wood manufacturing industries.

**Methods**

A systematic review was performed in accordance with PRISMA guidelines [22]. Search terms were agreed by three team members (Table 1), in partnership with a Health and Safety Executive (HSE) librarian. The major (or, in PubMed, MeSH) heading from Column 1 in Table 1 was combined consecutively, and in descending order, with terms from Column 2 followed by Column 3 until no new references were generated. This process was repeated keeping the major term for Columns 2 and 3. A free text search using the same terms was then conducted to ensure no references were missed. Web of Science, PubMed, Embase, ProQuest, OSHUPDATE (including ‘grey literature’ from HSELINE, NIOSHTIC, RILOSH and CISDOC) and the HSE e-library were searched from January 1970 to December 2014. OmniVis software (Instem Scientific v. 6.1.12), a reference management tool, was used to cluster references using the same terms from Table 1. This software groups references using Booleans and keywords. The clusters were then manually assessed for relevance using the following inclusion and exclusion criteria in order to generate final abstracts for review.

Abstracts were reviewed independently and then agreement was assessed. Where the relevance of the abstract was not clear, or there was disagreement, the full paper was reviewed. Studies were considered eligible for inclusion where they met the following criteria: (i) adult workers from wood processing or furniture manufacturing industries; (ii) meta-analyses, controlled trials,

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The ‘*’ wild card was used to find plurals and word variants. MDF, medium density fibreboard.
longitudinal studies or cross-sectional analyses; (iii) respiratory or nasal symptoms, asthma, lung physiology or sensitization identified as a study outcome and (iv) English language papers. Studies containing data from both the wet and dry wood industries were included.

Papers were specifically excluded where they (i) were case reports or letters, (ii) contained data only from the timber industry, (iii) contained hygiene data alone or (iv) solely examined dermatitis, cancer or immunological mechanisms.

As a high degree of heterogeneity was expected between the studies, articles were assessed for quality using both the Scottish Intercollegiate Guideline Network (SIGN) guidance and the Methods for Evaluating Research Guidelines and Evidence (MERGE) [23–25]. MERGE was developed specifically for observational research and was employed as a second evaluation tool to ensure consistency, and that quality of papers was not under or overestimated.

Papers were grouped by common themes identified as specific study endpoints. These were (i) ocular, nasal and respiratory symptoms; (ii) asthma; (iii) lung function and (iv) sensitization (including atopy, specific skin prick testing and specific immunoglobulin E [IgE]). Where wood exposures had been measured in the study population, evidence for a dose–response relationship was sought.

Data pertaining to each study endpoint were extracted and recorded on an agreed pro forma (Appendix 1, available as Supplementary data at Occupational Medicine Online), and then tabulated for comparison. The specific challenges associated with meta-analysis in observational studies have been reported [23,26]. The wide search criteria employed here retrieved studies with substantial differences and, therefore, did not permit further statistical analysis. Consequently, data were expressed as ranges, mean prevalence or incidence according to the type of data collected. Confidence intervals (CIs) are referred to in the text or in Table S1 (available as Supplementary data at Occupational Medicine Online), where appropriate. Common confounders are included in Table S1 (available as Supplementary data at Occupational Medicine Online).

**Results**

**Evidence quality and characteristics**

Initial searching generated 1328 references, of which 446 abstracts were independently reviewed for relevance by the study team and 55 papers were included in the final review. Figure 1 shows the number of papers excluded at each stage.

Using SIGN criteria, one of the 55 studies was rated 1−, and four were rated 2++ (as shown in Table 2). The remaining 50 were rated 2+ or below. Using MERGE, 14 of the 55 papers were graded A. Summary details for these 14 studies, grouped by study endpoint, are presented in Table S1 (available as Supplementary data at Occupational Medicine Online); details of the remaining studies are available in Table S2 (available as Supplementary data at Occupational Medicine Online).

**Table 2.** Studies by SIGN or MERGE rating

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All articles were systematically graded using both tools and a predefined pro forma. Data for all but the methodology paper are presented here.

Sixty per cent (33) of papers reported any data on prevalence or incidence of respiratory or nasal symptoms. Fifty-eight per cent (32) measured lung physiology, including peak expiratory flow rate (PEFR), forced expiratory volume in 1 s (FEV1), forced vital capacity (FVC), ratio of FEV1 to FVC (FEV1/FVC), forced expiratory flow rate (FEF), mid-expiratory flow rate (MEF),
bronchodilator-induced reversibility (BDIR), bronchial hyperresponsiveness (BHR), transfer factor (TL\textsubscript{co}) and specific inhalation challenge (SIC). Asthma was recognized as a study endpoint in 15\% (8) of studies, with 13\% (7) of studies reporting sensitization data.

**Symptoms**

Most studies reported either mean symptom frequency or odds ratios (ORs), the latter measuring the increased risk of a particular health outcome as a function of exposure group, normally comparing exposed woodworkers with either lesser or non-exposed populations (Tables S1 and S2, available as Supplementary data at Occupational Medicine Online).

The most frequently reported respiratory symptom was cough, affecting between 6 and 80\% of exposed workers (ORs ranged between 1.2 and 5.5). Wheeze and chest tightness were also commonly seen in exposed workers, with frequencies of between 9 and 40\% (ORs between 1.3 and 5.9).

Shortness of breath was also excessively reported in woodworkers, with a range of frequencies of between 10 and 39\% (ORs ranged between 1.7 and 10.6). Sputum production and bronchitis (or chronic bronchitis) were described in fewer studies, but in generally high levels (frequencies reported between 12 and 67\% of exposed individuals, ORs ranged between 0.9 and 20).

Nasal symptoms in woodworkers were reported in approximately half of the 55 studies. The prevalence in exposed workers ranged between 25 and 64\% in the 26 relevant studies (ORs ranged between 0.8 and 16.4). Similarly, ocular or throat symptoms were common (ranging from 20 to 51\% in the included studies; ORs of between 1.1 and 13.5).

Work-related respiratory symptoms (WRRS), suggestive but not diagnostic of OA, were inconsistently defined between differing studies and were reported in fewer studies. Where WRRS were described, the prevalence among exposed workers ranged from 25\% for work-related wheeze to 52\% for work-related cough [17,28]. Two studies reported increased ORs for WRRS or work-related nasal symptoms, ranging from 1.8 to 6.0 [29,30].

Possible relationships between wood dust exposure and the presence of symptoms (or dose–response relationships) were explored in eight studies. For example, Jacobsen \textit{et al.} reported significantly increased risk for both cough and chronic bronchitis in female workers in high versus low-exposure categories [31]. The same group showed a dose–response relationship for nasal symptoms in two earlier studies [32,33]. In contrast, three studies found no increase in symptoms when stratified by exposure [34–36]. In one study of Tanzanian woodworkers, risks for wheeze and shortness of breath were reported to be higher in workers with lower wood dust exposures [30]. The authors highlighted the increased use of respiratory protective equipment among workers with higher exposures as a possible explanation for this.

**Asthma**

Asthma was defined in varying ways across studies. For example, five papers identified asthma cases using worker questionnaire responses [28,31,33,36,37], and one study used insurance data and ICD-10 codes to identify population-based asthma incidence [38]. A further study included a nested case–control analysis and performed objective measures of asthma including PEFR variability, BHR and BDIR [39]. A single meta-analysis was also identified; that included asthma studies where the diagnosis was made with objective measures or was self-reported through interview [40].

Asthma was reported in an additional nine studies where the method for identifying cases was unclear. These papers reported asthma prevalence of between 5 and 30\%, representing ORs between 3.7 and 5.5 in comparison to lesser or non-exposed populations.

Studies using serial PEF recordings to support a diagnosis of asthma included Norrish \textit{et al.}, who identified 12\% of exposed furniture workers with that condition [28]. Lipscomb \textit{et al.} reported similar prevalence, with 10.6\% of their exposed population reporting ‘ever asthma’, and 8\% reporting an asthma attack in the last 12 months [37].

In a large Finnish epidemiological study, Heikkilä \textit{et al.} identified a relative risk (RR) of asthma of 1.5 for both male and female woodworkers compared with the general Finnish population, similar to that reported in other non-exposed blue-collar workers (RR 1.4) [38]. Pérez-Rios \textit{et al.} found comparable results in their meta-analysis of 19 studies, with RR of 1.5 for asthma in woodworkers [40].

Three studies found atopic woodworkers to have higher risks for both asthma and airway responsiveness, again compared with non-exposed controls [28,31,39]. Schlünssen \textit{et al.} also found that atopic woodworkers in the highest exposure category had an increased risk for asthma and the presence of BHR (OR 22.9). Non-atopic woodworkers in the highest exposure category had an OR of 20.3 for asthma and work-related symptoms [39]. However, no dose–response effect was seen in the study of Heikkilä \textit{et al.} or in that of Sripaiboonkij \textit{et al.} [36].

Female gender was also identified to be a risk factor for asthma in one particular study (OR of 11.3 reported for asthma symptoms in women with no baseline symptoms in the longitudinal cohort), although no other studies specifically identified gender as an independent predictor for either the presence or development of asthma in woodworkers [31].

**Lung function**

All but two studies reported some lung function measures in woodworkers; including FEV\textsubscript{1}, FVC, FEV\textsubscript{1}/FVC,
FEF and MEF. In addition, two papers presented additional information on airway reactivity, and a further paper reported TLECOx.

It was evident that spirometry was performed using differing protocols, suggesting that overall quality and reproducibility may not be homogeneous, and control populations differed (Tables S1 and S2, available as Supplementary data at Occupational Medicine Online). Eighteen studies measured lung function at a single time point (STP), 11 measured cross-shift lung function changes and a further three assessed longitudinal decline.

Seventy-eight per cent (14) of studies reported significantly lower STP FEV1 or FVC in their exposed populations, with two studies reporting more obstructive spirometry (as defined by an FEV1/FVC < 0.7) among exposed versus control populations [3,41]. Shamssain also identified that 56% of woodworkers employed between 10 and 19 years had an FEV1/FVC of <70%, compared with 27% employed between 1 and 9 years. By contrast, four studies reported no difference in STP lung function between exposed and control populations (Table S2, available as Supplementary data at Occupational Medicine Online).

Bohadana et al. reported higher than predicted FEV1 and FVC in exposed woodworkers but did show a significant increase in BHR across exposure categories, with only 8% of workers in the lowest exposure category, versus 27% of workers in the highest exposure category, having BHR [34]. However, an earlier Italian study did not demonstrate any increase in BHR between exposure populations, with OR 0.6 (95% CI 0.3–1.6) in woodworkers not significantly increased from controls [42].

Ninety-one per cent (10) of studies that measured cross-shift changes in lung function reported lower FEV1 or FVC values in their exposed populations. Mean cross-shift loss in FEV1 ranged between 0.11 and 14.9%, in FVC between 0.12 and 5.85% and in MEF and FEF values between 4.8 and 22.2%. Mandryk et al. found cross-shift FEV1, and FVC decrements in joiners were significantly associated with number of years wood dust exposed, with correlation coefficients of −0.77 and −0.8, respectively [16]. Conversely, however, Jacobsen et al. found no evidence that cross-shift change correlated with longitudinal lung function decline [43].

Two further studies (including >1000 workers) examined longitudinal lung function decline in woodworkers. In their North American cohort, Glindmeyer et al. measured mean inhalable dust concentrations of 1.45 mg/m³ (range 0.77–2.51 mg/m³), and mean respirable dust concentrations of 0.18 mg/m³ (range 0.1–0.21 mg/m³) [44]. In this context, they reported a negative effect of cumulative exposure to respirable dust in the milling and in the sawmill/planing/plywood industries on FEV1, FEV1/FVC and FEF levels, and in the sawmill/planing/plywood industry for FVC. No effect was observed in furniture and cabinet workers. Conversely, Jacobsen et al. found accelerated lung function decline in female furniture workers who smoked, this effect being more marked in those workers with higher wood dust exposures [45]. They also found small, but significant, excess longitudinal FEV1 decline in both male and female workers still employed in the wood industry at follow-up.

Sensitization

Seven papers reported data on sensitization, using: skin prick tests (SPTs) to common aeroallergens or to specific woods and moulds; total serum IgE; and serum IgE to specific wood species.

Atopy (as defined as SPT positivity to common aeroallergens) was generally common in exposed populations (ranging between a prevalence of 9 and 80%). Skovsted et al. reported no difference in prevalence of specific IgE to pine in atopic compared with non-atopic workers [46]. In their follow-up study, however, Schlünnsen et al. reported high levels of atopy among pine and beech workers, with a significant correlation between atopy and reported respiratory symptoms [47].

Where studies reported specific IgE to wood types, positive results were uncommon. Ricciardi et al. found no specific IgE to iroko wood in a group of asthmatics, all of whom had a sustained fall in PEF on SIC to iroko extract [20]. Notably, this group also demonstrated a significant increase in blood eosinophils and positive methacholine challenge post-SIC, suggesting these individuals had mounted a significant allergic response to iroko inhalation.

Furthermore, Skovsted et al. found no difference in prevalence of specific IgE between exposed and non-exposed workers (both 3%), and Schlünnsen et al. reported only 1.7% of exposed pine workers having pine-specific IgE, and 3.1% of exposed beech workers having beech-specific IgE [46,47]. The latter study did not firmly establish a relationship between specific sensitization and respiratory symptoms but did find an association with increasing wood exposures and higher rates of specific IgE; 7.8% of workers in the highest exposure category were sensitized to pine, with 9.8% of beech workers in the highest exposure category sensitized.

Discussion

A number of high-quality observational studies were identified for, and included in this review, although the majority of papers were of a lower evidence rating. Only five studies were graded SIGN 2++ or above (Table 2), although MERGE identified more highly rated studies. This finding emphasizes the importance of using tools specifically designed for reviewing occupational research, where the validity and applicability of studies may otherwise be underestimated [23].
Exposure to wood dust in the furniture and wood manufacturing industries was associated with an increased risk of a variety of reported respiratory symptoms, and particularly cough. This was based on evidence from both cross-sectional and longitudinal studies. Indeed, reported symptoms were more common in higher exposed woodworkers in all but one study reporting airway symptoms. It is at least plausible that some of this excess relates to recall bias, but there is evidence to suggest that though respiratory illness may alter symptom reporting, workers continue to self-report with high sensitivity [48].

There was conflicting evidence to support a dose–response relationship for respiratory symptoms in woodworkers. Neither Rongo et al. nor Sripaiboonkij et al. found increasing levels of wheeze or chest tightness with increasing dust exposure, despite large study numbers and relatively high wood dust exposures; inhalable dust ranging from 0.02 to 2.93 mg/m$^3$ and 1.43 to 22.76 mg/m$^3$, respectively [30,36].

Woodworkers in these industries were also found to be at a greater risk of asthma, despite measured exposure to dust levels being lower than may be expected in certain European countries [49]. Schlünsen et al. found an excess of self-reported and physician-diagnosed asthma in atopic female wood workers [39]. They also reported that atopic workers in the highest exposure category had significantly more asthma symptoms and BHR. Conversely, non-atopic workers in the same exposure category had less BHR and more work-related asthma symptoms. Though CIs were wide, this finding suggested that non-atopic and atopic wood workers may manifest different clinical outcomes as a result of wood dust exposure. More conclusive work is needed to better understand these responses.

The only meta-analysis included in the review reported an increased RR for asthma in woodworkers of 1.5 compared with the general population [40]. Heikilä et al. found similar RRs (1.5 in both female and male woodworkers) comparable to blue-collar workers in the same industry, although did not identify a dose effect [38]. Inclusion in the study depended on inclusion in a national registry, potentially missing workers with a past, or missed, asthma diagnosis. Jacobsen et al. demonstrated an increased risk of asthma for female woodworkers in the highest exposure category [31]. Interestingly, they also reported significantly more asthma in female workers who had left the furniture industry at follow-up, suggesting, at least in part, a healthy worker effect.

Cross-shift changes in lung function have been reported in other industries including dairy workers and mussel pickers [50,51]. Their utility is uncertain when diagnosing occupational respiratory problems. Whilst these measures evidently document an acute respiratory response to the work environment, they may be affected by the normal diurnal variation in lung function, which is classically exaggerated in asthma. They may be further confounded by shift work and poor spirometry measurement, and cross-shift change has been shown to have a high specificity but low sensitivity for identifying cases of OA [52].

Jacobsen et al. found no relationship between acute, cross-shift changes, and longer term lung function loss in their study of woodworkers [43]. This may have reflected exposures other than wood in the workplace; for example, Mandryk et al. demonstrated significant cross-shift lung function change in a workplace where substantial endotoxin, bacterial and fungal exposures were measured [16].

This review identified conflicting evidence for impairment of lung function among exposed woodworkers, with certain cross-sectional studies reporting a difference in measured values between exposed and control populations, but evidence for excess longitudinal lung function decline was only demonstrated in certain subgroups. Two studies reported annual decline in lung function, but with mixed conclusions. Jacobsen et al. found female smokers exposed to wood dust had significantly accelerated lung function decline [45]. In contrast, Glindmeyer et al. found no relationship between longitudinal lung function decline and employment in the furniture or cabinet making industry [44]. Studies in western red cedar workers have demonstrated irreversible longitudinal lung function decline, findings that have been replicated with other forms of OA [8,53].

It was not the aim of this study to review all the evidence relating to mechanisms by which wood dust causes respiratory disease. Whilst the exact causative agent (or agents) responsible is not clearly identified, one study suggested that high levels of endotoxin were linked to the reporting of cough in furniture workers [17]. In addition, the relationship between chronic bronchitis and wood processing was particularly strong, even when the effects of smoking had been taken into consideration. Jacobsen et al. reported increased risks for chronic bronchitis in a longitudinal study of female woodworkers and showed that increasing exposures in this group increased the likelihood of bronchitis [31]. Whether this finding represented an ‘irritant’ effect is not clear, but symptoms may have reflected underlying disease mechanisms not typical of allergy (or Type I hypersensitivity) [12]. In addition to intensity of exposure, the propensity of wood to cause asthma may be influenced by the chemical properties of specific species, the particle size generated, the route of exposure and individual susceptibility to disease [44,54].

However, whilst certain studies supported the development of sensitization to wood dust as being a potentially important process in the development of respiratory symptoms and asthma, the mechanism remains unclear. Skovsted et al. identified a potentially pathogenic protein band in beech and pine but reported very low levels of pine- and beech-specific IgE in exposed
Work-related respiratory symptoms do not clearly...

To our knowledge, this review is the only systematic review of asthma in workers employed in the furniture or wood manufacturing industries. Its findings are comparable to a previous review of the dry wood industry [55]. Since a greater number of papers were included in the current review, and a wide range of search terms employed over a long period of interest, it is unlikely that a significant number of relevant studies were missed. Although there was heterogeneity between studies, consideration of a range of endpoints allowed us to extract data relevant to several areas of interest and raise points for further investigation.

There are, however, certain downsides to consider. Our review findings were difficult to compare for a number of reasons. Exposed populations were drawn from a variety of sources including worksites, outbreaks or from hospital cohorts. Population size, country, exposure level and design also varied between papers. A number of studies were published prior to the year 2000, and before mandatory changes to exposure limits in both North America and Europe. As such, some data may not accurately reflect current practice; although recent international data suggests that exposure to wood dust continues to differ both within and between countries [49].

In addition, exposure misclassification represents an important potential source of bias within this review. Some studies reported data on inhalable or respirable dust, whereas others reported only total or non-specific dust readings. Variation in measurement techniques could also have influenced clinical risk attributed to exposure and bias results, potentially underestimating (or overestimating) the magnitude of past exposures especially where workers have subsequently moved to lower exposed tasks [27].

In summary, this review found an increased risk of respiratory symptoms and asthma in people working in the wood processing and furniture manufacturing industries. Accelerated lung function decline due to ongoing exposure was evident for specific study groups. It is important to note that although much of the more robust research included was conducted in lower exposure environments, ill health occurred across all exposure groups. Further study is required considering other ways of assessing airway inflammation in the workplace, to explore the mechanisms through which wood dust causes respiratory disease and asthma and most importantly how to define interventions to reduce wood-related respiratory ill health.

Key points

- Work in the furniture and wood processing industries is associated with an increased prevalence of respiratory symptoms and asthma.
- Evidence for a greater risk of impaired lung function with increasing exposure to wood dust is conflicting.
- Work-related respiratory symptoms do not clearly relate to IgE sensitization in exposed woodworkers and mechanisms by which woodworkers are sensitized to wood dust remain unclear.

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Conflicts of interest

None declared.

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