INTRODUCTION

Approximately 3.6 million workers in the European Union are exposed to wood dust [45].

Wood is processed in many industries including sawmills with processing of fresh wood, plywood mills producing plywood from fresh wood, other types of mills producing wood composites, and furniture factories or smaller workshops using dry wood only. Studies from recent years indicate different exposure response relationships for dry wood compared to fresh wood [19, 25, 51].

Wood dust is a known inducer of cancer in the nasal cavity and recent reviews have focused on this [14, 40]. Wood dust has also been associated with a variety of respiratory diseases including asthma, chronic bronchitis, nasal symptoms and eye symptoms, as well as chronic impairment in lung function. Although the occurrence of non-malignant respiratory diseases related to wood dust has been reviewed earlier [18, 29, 65], a number of studies have also been performed in recent years. The earlier reviews, however, did not specifically consider the difference between dry and wet wood. Hence, updated reviews concerning non-malignant respiratory diseases divided into dry wood and wet wood is warranted. This review focuses on fresh wood and mixed wood exposure to wood dust. A second review focuses on dry wood exposure [41].

In the reviews, we have not included papers concerning occupational exposure to wood dust and cryptogen fibrosing...
alveolitis, as only a few case control studies have been performed concerning this rare disease and its association to wood dust [7, 30, 39, 54, 58]. Allergic and toxic alveolitis is seen among fresh wood dust exposed workers, especially among sawmill workers, where up to 20% had experienced symptoms consistent with toxic alveolitis [8, 55]. Allergic alveolitis is rare, also among sawmill workers [55], but cases has been reported [32, 60]. Microorganisms are suspected to be by far the most important agent, especially Rhizopus microspores [24]; therefore, all papers on these two diseases are not systematically included in this review, but the importance of allergic and toxic alveolitis with respect to respiratory impairment among fresh wood dust exposed workers are discussed.

METHODS

The literature search for the reviews covered Medline for papers published in English for the period 1969 to June 2009, with the following search conditions: “Wood” [MeSH Terms] AND “Occupational Diseases” [MeSH Terms] NOT “Case Reports” [Publication Type]. This revealed 422 publications. The search was accompanied by a scan of list of references in the identified studies and supplemented with updates until August 2009. Criteria for inclusion were epidemiological studies describing associations between upper or lower respiratory diseases, or symptoms and exposure to fresh or mixed wood dust. Studies not having an internal control group (high or low exposure) or an external control group were excluded. Papers which did not take smoking into consideration, or which did not adjust for age when dealing with lung function were discounted.

In total, 25 original papers were included. To allow for comparison between papers, odds ratios (OR) for symptoms from data provided in the papers, whenever OR’s were stated, were calculated with Chi square test using exact confidence intervals.

Chronic bronchitis was defined as daily coughing and phlegm for at least 3 month during at least 2 consecutive years [13].

RESULTS

Table 1 shows the main results from the reviewed papers. This review focuses on: asthma, asthma symptoms, coughing, chronic bronchitis, rhino-conjunctivitis, and impairment in lung function.

Asthma and Asthma symptom. Seventeen papers including one register-based follow-up study and 15 cross-sectional studies (where one study population was re-investigated after 2 years) have reported on asthma, asthma symptoms or bronchial hyper responsiveness (BHR).

In a register-based population study, Heikillä et al. [33] determined incidence rates of clinically verified asthma for different industries handling both fresh and dry wood. Relative Risk (95% Confidence interval) RR (95% CI) for asthma for all wood exposed males and females compared to administrative control workers were 1.5 (1.2–1.8) and 1.5 (1.2–1.7), respectively. For workers handling primarily fresh wood RR varied between 1.5 (1.2–18) (males forestry and logging) and 1.9 (1.5–2.5) (males sawmilling).

Four studies reported prevalence’s of between 5–14% for physician diagnosed asthma [16], ever asthma [35], current asthma [36] or asthma [34], with OR’s comparing exposed with unexposed workers ranging from 2.5–5.5, being statistically significant in two studies [34, 35]. In addition, 3 studies [16, 19, 25] defined asthma from a combination of symptoms and reported prevalence’s of between 10–73%, with OR 1.5–2.7, significant in 2 studies [16, 19].

Six studies reported significantly increased prevalence’s of wheezing (15–42%) chest tightness (36–43%), shortness of breath (SOB) with wheezing (15%), and chest tightness with wheezing (20%) with OR’s ranging from 1.1–2.7 when comparing exposed to non-exposed [15, 19, 25, 34, 36] or groups with different exposures [15, 20]. In contrast, 2 studies from Thailand and Indonesia did not find any relation between wet wood exposure and asthma symptoms [11, 46].

Prevalence’s of work-related asthma (WRA) symptoms (wheezing, SOB with wheezing) (6–20%) were reported in 4 papers with OR’s ranging from 0.7–7.0 when comparing exposed to non-exposed [4, 19, 52] or to groups with lower exposure [31], with significantly increased OR’s in [19, 31]. In 5 studies prevalence’s of WRA ranged from 1.1–8.3% with OR’s from 1.5–2.7 when comparing exposed to non-exposed [4, 15, 16, 52], or years of exposure [63], although only one study found significant differences [63]. Another study reported a 1.1% prevalence of red cedar asthma (RCA) based on “a typical history of RCA” in the exposed group, but no information on the control group was available [15]. A 1 year incidence of RCA of 4–5% was estimated based on information on workers having left the work place.

The effect of red cedar (RC) exposure on BHR was explored in 2 studies. In [16] an increased prevalence of BHR among non-atopic RC workers (76%) vs non-atopic controls (4%) and an association between BHR and duration of employment was revealed. In a later follow up [64], persistent BHR was found to be related to exposure levels above 1 mg/m³. Furthermore, BHR was associated to specific IgE for plicatic acid.

Chronic bronchitis and cough. Seven cross-sectional papers reported chronic bronchitis with prevalence’s ranging from 10–69% and OR’s ranging from 1.0–9.6 when woodworkers were compared with controls [4, 31, 34, 36, 46, 52], lower exposure level [46] or lower seniority [57]. Findings were significant in 4 papers [4, 31, 46, 52]. In one study, chronic bronchitis was associated with duration of employment [36].
Coughing was reported in 9 cross-sectional studies with prevalence ranging from 11–46% and OR’s 0.9–26, 4 with significant results [16, 19, 34, 62], in studies comparing exposed to non-exposed [15, 16, 19, 34, 35, 36, 46, 62] or lower exposed groups [11, 46, 63]. Work related coughing was reported in 4 papers with prevalence’s between 14–59% comparing exposed to non-exposed controls [4, 19, 52], or lower exposed [31]. OR’s were ranging from 0.8–18.7, two with significant results [19, 52].

**Post-shift decline in lung function.** A total of 6 cross-sectional studies have investigated acute changes in lung function among workers exposed to fresh wood. Gandevia et al. reported a day to day reversible post-shift decline in FEV₁ among a group of RC workers [27], while Herbert et al. in two studies at oriented strandboard mills showed a significant post-shift decline in FEV₁ and FVC among wood workers [34, 35]. Likewise, Mandryk et al. reported a post-shift decline in FEV₁, FVC, FEV₁/FVC among sawmill workers for both green mill workers and dry mill workers, but they could not confirm a DRR to wood dust exposure [51, 52]. Only one study, Ashley et al., reported no changes in lung function during a work week among woodworkers compared to non-exposed controls [5].

**COPD.** Seventeen studies including 2 industry-based follow-up studies, one register based follow-up study and 14 cross-sectional studies have reported on lung function parameters and exposure to fresh or mixed wood dust.

In an 11 year follow up study, Noertjojo et al. [53] reported a greater decline in FEV₁ and FVC among RC sawmill workers compared to controls, and reported a DRR between mean average exposure during follow up and annual decline in lung function. Friesen et al. in a register-based follow-up study reported a DRR between cumulative wood dust exposure and COPD hospitalisation rate. In contrast, Glindmeyer et al. in a 5-year follow-up study found no association between wood dust of any size fraction and lung function indices.

Twelve cross-sectional studies found associations between baseline lung function parameters and exposure to wood dust. Douwes et al. [20] found high current exposure to wood dust to be associated to reduction in FEV₁, PEF and FVC for green mill and dry mill workers, the latter only significant for workers in green mills. Mandryk et al. revealed a DRR between decreased FEV₁ and current inhalable dust concentration and for green mill workers a DRR between respirable dust and decrease in FVC, but also a positive correlation between baseline lung function indices and years of exposure to wood dust [51, 52]. Likewise, Teschke et al., using different exposure models, reported a DRR between inhalable dust and decreased FEV₁ [61]. Borm et al. [11] found no association between cumulative exposure and lung function indices. They found, however, an association between years of employment and lung function indices for male workers.

Ashley et al. [5] found a borderline significant association between duration of exposure and reduced FEV₁ and FVC among RC workers, while Vedal et al. [63] found a DRR between current wood dust exposure and FEV₁, FVC and FEV₁/FVC, but no association with years of employment.

Liou et al. [46] found a DRR between current exposure level and PEF, mean FEV₁, and mean FVC. A number of studies found decreased PEF, FEV₁ or FEV₁/FVC among exposed workers compared to non-exposed controls [16, 34, 35, 36, 37, 46, 51, 52, 62, 63].

Only two cross-sectional studies found no association between wood dust exposure and baseline FVC or FEV₁ [31, 57].

**Rhino-conjunctivitis.** Two studies reported significantly increased prevalence’s of rhinitis (17–31%) with OR’s 1.6–2.6, when comparing woodworkers to non-exposed controls [15, 19] or groups with lower exposure [19]. One study did not find exposure to wood dust associated with rhinitis [25]. Three papers reported WR nasal symptoms (9–49%) [4, 31, 52], and 2 [4, 52] found significantly increased prevalence’s of runny nose and sneezing with OR between 3.5–7.0 in green and dry mill workers compared to controls. Significantly increased occurrence of conjunctivitis was reported in one study with OR 9.9 (1.7–400) between exposed and non-exposed workers [19]. One study did not find current inhalable wood dust exposure associated to conjunctivitis [25]. Three papers comparing woodworkers and controls reported increased prevalence of WR conjunctivitis [4, 19, 52], significant in [19], while the others reported significantly increased prevalence of WR eye irritation, especially among green mill workers [4, 52]. In addition, one study reported a non-significant increase in WR eye irritation among sawmills workers [31].

One paper reported a significant negative association between exposure duration and an irritation syndrome including nasal or conjunctiviral irritation [57].

NAL (nasal lavage) performed in one study revealed a higher cell count among females in the highest exposure category (>5 mg/m³) [11].

**DISCUSSION**

When estimating respiratory health effects of occupational exposure to wood dust it is crucial to have valid exposure estimates. In the presented papers, wood dust exposure was assessed in different ways. Some studies estimated exposure solely on employment status [16, 19, 62], but most studies included dust measurements at least on a limited number of workers. Group exposure estimates were based on additional information about work area, job title, etc. Some studies based exposure assessment on a substantial amount of measurements [11, 20, 26, 28, 52, 53, 61]. Exposure misclassification in many of the studies is likely. When comparisons are made between groups of more
Table I. Characteristics of studies included. Unless otherwise stated, symptom risk is given as OR.

<table>
<thead>
<tr>
<th>Author, country, year</th>
<th>Type of study/ Number</th>
<th>Industry; wood species</th>
<th>Exposure measure mg/m³; unless otherwise stated personal dust in GM (GSD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glindmeyer, US, 2008 [28]</td>
<td>FU 5 yr. E: 385</td>
<td>Sawmill, planning, plywood, milling Various wood types</td>
<td>Dust N=647  3 size fractions (&lt;4&lt;10&lt;100) μm  150 analysed for % WS and % RPM  GM resp: 0.10–0.19  %WS mean: 2–28  GM inhal: 0.77–1.07  %WS mean: 8–39  JEM: Mean individual exp. during FU, mg/m³; for 3 size fractions, WS, RPM</td>
</tr>
<tr>
<td>Heikkilä, FI, 2008 [33]</td>
<td>R-FU Registers: Wood processing industries. Incident AS reimbursement register E: 56,721 Other blue-collar W: 101,413 C: 12,839</td>
<td>Wood processing industries various, 10 industries wet and dry pine, spruce, birch</td>
<td>JEM 5 exp levels based on industrial meas. total dust  Woodworkers (Ew):  ( E_{low} \leq 0.02 )  ( E_{med} = 0.5 )–(&lt;1.5 )  ( E_{high} \geq 1.5 )  ( E_{b} ): Other bluecollar workers, wood exp. unknown C (administrative)  Also divided into types of work</td>
</tr>
<tr>
<td>Rusca, SW, 2008 [57]</td>
<td>CS E: 111</td>
<td>Sawmills; spruce, fir</td>
<td>Area inhal. dust N=?  AM: 1.7 (range 0.2–8.5)  Also bacteria, fungi</td>
</tr>
<tr>
<td>Friesen, CA, 2007 [26]</td>
<td>R-FU E: 11,273</td>
<td>Sawmills; softwood</td>
<td>Inhal. dust N=1399; JEM non-spec. particulate and wood dust  Cum. exp. particulate: mean (max): 9.8 (220) mg yr/m³  Cum. wood dust: mean (max.): 6.8 (89) mg yr/m³</td>
</tr>
<tr>
<td>Douwes, NZ, 2006 [20]</td>
<td>CS E: 167 3 exp. levels</td>
<td>Sawmills; pine</td>
<td>Inhal. dust N=183  GM: 0.5 (2.7)  JEM: 3 exp. catagories  ( E_{low} \leq 0.4 ) (2.8)  ( E_{high-dry} ) ≤ 0.6 (2.2)  ( E_{high-green} ) ≤ 0.8 (2.3)</td>
</tr>
<tr>
<td>Ugheoke, NI, 2006 [62]</td>
<td>CS E: 150 C: 150</td>
<td>Sawmills; mansonia, iroko, walnut</td>
<td>E vs C</td>
</tr>
<tr>
<td>Teschke, CA, 2004 [61]</td>
<td>CS E: 105 C: 483</td>
<td>Sawmills; pine, spruce</td>
<td>Inhal. dust N=103  GM: 0.54 (2.9)</td>
</tr>
<tr>
<td>Lower airway symptoms OR (95% CI) * OR calculated</td>
<td>Upper airway symptoms OR (95% CI) * OR calculated</td>
<td>Objective measurements</td>
<td>Confounders included</td>
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</tr>
<tr>
<td>No ass. between WS and lung function indices for any size fraction</td>
<td>Neg. ass. between resp. RPM and annual change in FEV₁, FEV₁/FVC, or FEF₂⁰₋₇⁵ in milling,</td>
<td>Age, sex, height, weight change, ethnicity, smoking, baseline lung function</td>
<td></td>
</tr>
<tr>
<td>Neg. ass. between resp. RPM and annual change in FEV₁ and FVC in sawmill-planning-plywood</td>
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**RR: AS men E vs C:**  
\[ E_{\text{w}}: 1.5 (1.2–1.8) \]  
\[ E_{\text{low}}: 1.4 (1.1–1.7) \]  
\[ E_{\text{med}}: 1.7 (1.4–2.2) \]  
\[ E_{\text{high}}: 1.2 (0.9–1.6) \]  
\[ E_{\text{rand}}: 1.4 (1.1–1.8) \]  
\[ E_{\text{med}}: 1.9 (1.5–2.5) \]  

<table>
<thead>
<tr>
<th>RR: AS women E vs C:</th>
<th></th>
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</thead>
</table>
| \[ E_{\text{w}}: 1.5 (1.2–1.7) \]  
\[ E_{\text{low}}: 1.4 (1.2–1.8) \]  
\[ E_{\text{med}}: 1.6 (1.3–2.0) \]  
\[ E_{\text{high}}: 1.2 (0.8–1.6) \]  
\[ E_{\text{rand}}: 1.4 (1.1–1.6) \]  

**COPD hospitalisation rate:**  
No ass. cum. non-spec. dust  
DRR cum. wood dust  
RR 1.93 \( \frac{E_{\text{cum-high}}}{E_{\text{cum-low}}} \)  

<table>
<thead>
<tr>
<th>COPD hospitalisation rate:</th>
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<tbody>
<tr>
<td>Sex (all males) age, ethnicity</td>
<td>No adj. for smoking, but has been considered</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>E\text{high-dry} vs E\text{low}:</th>
<th>E\text{high-green} vs E\text{low}:</th>
</tr>
</thead>
</table>
| \[ AS: 2.1 (1.0–4.4) \]  
\[ \downarrow \text{FEV}\text{_1}, \downarrow \text{PEF}, \downarrow \text{FVC} \]  
\[ \text{Borderline} \downarrow \text{FVC} \]  
\[ \downarrow \text{FEV}\text{_1}, \downarrow \text{FVC}, \downarrow \text{PEF} \]  

<table>
<thead>
<tr>
<th>Non-Smokers E vs C:</th>
<th>Smokers E vs C:</th>
</tr>
</thead>
</table>
| WH: 5 vs 0%, \( p<0.01 \)  
SOB+WH: 1 vs 0%, NS  
CO*: 25.5 (9.7–77) | E vs C:  
| lover PEF for both smokers and non-smokers | Sex (all males), smoking, age, height |

Smokers E vs C:  
WH: 4 vs 0%, NS  
SOB+WH: 2 vs 0%, NS  
CO*: 6.4 (0.8–289)  
Using diff. models of exp.:  
DRR Exp. & \downarrow \text{FEV}\text{_1}  

Smoking, sex, age, ethnicity
<table>
<thead>
<tr>
<th>Author, country, year</th>
<th>Type of study/ Number</th>
<th>Industry; wood species</th>
<th>Exposure measure mg/m³; unless otherwise stated personal dust in GM (GSD)</th>
</tr>
</thead>
</table>
| Fransman, NZ, 2003 [25]     | CS                    | Plywood mill; pine     | Inhal. dust N=57
GM 0.7 (1.9)
Job titles: low/high
Yr. of exp.
Also endotoxins, abietic acid, terpenes, formaldehyde |
JEM: E<sub>low</sub>: <2
E<sub>med</sub>: 2–5
E<sub>high</sub>: >5
Yr. of exposure, cum. exp. |
| Douwes, NZ, 2001 [19]       | CS                    | Sawmills; pine         | JEM (work area, job title)
4 exp. categories:
C<sub>low</sub> (non-exp.)
E<sub>low</sub> (non/low)
E<sub>high-green</sub>
E<sub>high-dry</sub> |
| Mandryk, AU, 2000 [52]      | CS                    | Sawmills (green mills, dry mills); eucalypt | Inhal. dust N=93
GM<sub>green</sub>: 1.5 (3.7)
GM<sub>dry</sub>: 1.7 (2.5)
Also resp. dust, endotoxins, glucans, bacteria |

[52] Mandryk, AU, 2000

Part of study [51]
### Fresh wood dust and respiratory diseases – a review

<table>
<thead>
<tr>
<th>Lower airway symptoms OR (95% CI) * OR calculated</th>
<th>Upper airway symptoms OR (95% CI) * OR calculated</th>
<th>Objective measurements</th>
<th>Confounders included</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Yr. exp. vs C:</strong></td>
<td><strong>RH:</strong> 35.7% E</td>
<td><strong>Sex, age, ethnicity</strong></td>
<td><strong>Sex, age, ethnicity</strong></td>
</tr>
<tr>
<td><strong>&lt;2yr.; 2–6.5 yr.; 6.5 yr.</strong></td>
<td><strong>CJ:</strong> 25% E</td>
<td><strong>Smoking, sex, age</strong></td>
<td><strong>No smoking information for C</strong></td>
</tr>
<tr>
<td><strong>AS:</strong> 0.5 (0.2–1.7); 1.0 (0.3–2.7); 3.1 (1.3–7.2)</td>
<td><strong>NS increase of nasal and eye symp. in rel. to exp. level</strong></td>
<td><strong>Smoking, sex, age, ethnicity, mill</strong></td>
<td><strong>No smoking information for C</strong></td>
</tr>
<tr>
<td><strong>WH:</strong> 0.4 (0.1–1.6); 1.4 (0.6–3.6); 1.8 (0.7–4.3)</td>
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<tr>
<td><strong>SOB+WH:</strong> 1.1 (0.4–3.0) 1.0 (0.4–2.7); 2.6 (1.1–5.8)</td>
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</table>

#### Males:

- WH 1.6%; CO: 23.5%
- **Females:**
  - WH: 1.8%, CO: 22.3%,
  - No ass. exposure level, cum. exp. or yr. exp.

#### Objective measurements

<table>
<thead>
<tr>
<th><strong>E</strong> vs <strong>C</strong></th>
<th><strong>RH:</strong> 2.1* (1.0–4.9)</th>
<th><strong>Sex, age, ethnicity</strong></th>
<th><strong>Smoking, sex, age, ethnicity, mill</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>E</strong> vs <strong>C</strong></td>
<td><strong>CJ:</strong> 9.9* (1.7–400)</td>
<td><strong>Smoking, sex, age</strong></td>
<td><strong>No smoking information for C</strong></td>
</tr>
<tr>
<td><strong>WR</strong></td>
<td><strong>WR-RH:</strong> 5.3 (1.4–45)</td>
<td><strong>Smoking, sex, age</strong></td>
<td><strong>No smoking information for C</strong></td>
</tr>
<tr>
<td><strong>WH</strong></td>
<td><strong>WR-CJ:</strong> 47 vs 0% (p&lt;0.05)</td>
<td><strong>Smoking, sex, age</strong></td>
<td><strong>No smoking information for C</strong></td>
</tr>
</tbody>
</table>

#### Smoking, sex, age

**E** vs **C**

- WH: 2.4* (0.7–11)
- CO: 3.4* (1.2–10)
- CB: 4.5* (1.3–20)

**WR**

<table>
<thead>
<tr>
<th>Green mills vs C</th>
<th>BN: 2.3* (0.9–6.5)</th>
<th><strong>FEV1, FVC decreased in both green and dry mill FEV1/FVC decreased in dry mill</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>RN: 3.6* (1.2–12)</td>
<td><strong>FVC↓, FEV1↓</strong></td>
<td><strong>FEV1, FVC decreased in both green and dry mill FEV1/FVC decreased in dry mill</strong></td>
</tr>
<tr>
<td>IN: 2.7* (0.8–11)</td>
<td><strong>FEV1/FVC↓</strong></td>
<td><strong>Post shift decline FEV1, FVC,</strong></td>
</tr>
<tr>
<td>SN: 7.0* (2.2–26)</td>
<td><strong>FEV1↓,</strong></td>
<td><strong>FEV1/FVC, Pos. corr. between inhal. dust and post shift decline in VC, FEV1</strong></td>
</tr>
<tr>
<td>CJ: 1.3* (0.1–79)</td>
<td></td>
<td><strong>25–75%</strong></td>
</tr>
<tr>
<td>EVD: 4.5* (1.5–15)</td>
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</table>

**Dry mills vs C**

| BN: 1.7* (0.6–4.6) | **FEV1, FVC decreased in both green and dry mill FEV1/FVC decreased in dry mill** |
| RN: 3.7* (1.2–11.2) | **FVC↓, FEV1↓** |
| IN: 1.5* (0.4–6.7) | **FEV1/FVC↓** |
| SN: 5.2* (1.4–21) | **Post shift decline FEV1, FVC,** |
| CJ: 1.0* (0.0–81) | **FEV1↓,** |
| EVD: 1.9* (0.5–7.5) | **FEV1/FVC, Pos. corr. between inhal. dust and post shift decline in VC, FEV1** |

**E** vs **C:**

- **FVC↓, FEV1↓**
- **Post shift decline FEV1, FVC,**
- **FEV1/FVC, PEF**
- **No DRR**
<table>
<thead>
<tr>
<th>Author, country, year</th>
<th>Type of study/ Number</th>
<th>Industry; wood species</th>
<th>Exposure measure mg/m³; unless otherwise stated personal dust in GM (GSD)</th>
</tr>
</thead>
</table>
GM sawmill: 1.6 (3.2)  
GM chip mill: 2.9 (1.7)  
Yr. of exp, resp. dust, endotoxins, glucans, bacteria |
| Liou, TA, 1996 [46]  | CS  
E<sub>High</sub>: 34  
E<sub>Low</sub>: 38  
C: 262 | Wood mill; powder | Total dust  
E<sub>High</sub> AM (N=6): 12.0  
E<sub>Low</sub> (N=1): 2.9 |
E: 243  
C: 140 | Sawmill; red cedar | Dust meas. N=1,132 (during 12 years)  
JEM, cum. exp.  
Mean daily: <0.2, 0.2 to 0.4, >0.4 |
AM (range): 1.35 (0.1–2.2) |
| Herbert, CA, 1995 [35] | CS E: 127 C: 165 | OSB-production; aspen, balsam | Area dust resp. N = 4  
AM 0.05–0.5  
Also formaldehyde, MDI |
| Herbert, CA, 1994 [34] | CS E: 99 C: 165 | OSB-production; aspen | Area dust total sawline N=1 (0.27)  
Also formaldehyde |
| Halpin, UK, 1994 [31] | CS E: 103  
2 exp. levels  
C: 52  
(incl. paint sprayer & welders) | Sawmill; spruce, pine | Total dust N=62  
GM low range: 0.2–1.1  
GM high range: 1.3–6.3  
GM control: 2.3  
Fungal spores |
| Vedal, CA, 1988 [64]  
FU of [16] | FU E: 227  
2 exp. levels | Sawmill; red cedar | Dust meas. N=? (personal, area)  
job, location |
| Vedal, CA, 1986 [63], Same study as [16] | CS E: 652  
2 exp. levels | Sawmill; red cedar | Dust meas. N=78 (personal, area)  
JEM, 334 assign.  
AM: 0.46 (range 0–6)  
Yr. of exp. |
### Lower airway symptoms OR (95% CI) * OR calculated

<table>
<thead>
<tr>
<th>WR E vs C:</th>
<th>Upper airway symptoms OR (95% CI) * OR calculated</th>
<th>Objective measurements</th>
<th>Confounders included</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>WR:</strong></td>
<td><strong>E</strong></td>
<td><strong>BN:</strong> 2.1* (0.8–5.3)</td>
<td>Smoking, sex (all males), age, height</td>
</tr>
<tr>
<td><strong>WH:</strong> 0.5* (0.2–1.4)</td>
<td><strong>RN:</strong> 3.5* (1.3–11)</td>
<td><strong>E:</strong> Decreased FEV₁, PEF.</td>
<td><strong>E:</strong> DRR exp. decrease in, FEV₁, FVC, PEF</td>
</tr>
<tr>
<td><strong>CO:</strong> 1.0* (0.3–3.5)</td>
<td><strong>IN:</strong> 2.0* (0.7–7.3)</td>
<td>Smoking, sex, age, height</td>
<td></td>
</tr>
<tr>
<td><strong>CT:</strong> 3.5* (1.1–14)</td>
<td><strong>SN:</strong> 4.6* (1.6–16)</td>
<td><strong>E:</strong> FEV₁, FVC reduced in E.</td>
<td>Smoking, sex (all males), height, ethnicity, atopy</td>
</tr>
<tr>
<td><strong>CB:</strong> 2.0* (1.0–4.5)</td>
<td><strong>CO:</strong> 1.5 (0.7–2.9)</td>
<td><strong>E vs C:</strong></td>
<td><strong>E:</strong> Post shift decline in FVC, FEV₁</td>
</tr>
<tr>
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<td><strong>E:</strong> Post shift decline in FVC, FEV₁</td>
</tr>
</tbody>
</table>

### FEV₁, FVC: Larger decline in E. DRR between exp. and annual decline in FVC

#### smoking, sex (all males), height, ethnicity, atopy

### No differences in FEV₁ and FVC between E and C

### Smoking, sex (all males), age, height, atopy
<table>
<thead>
<tr>
<th>Author, country, year</th>
<th>Type of study/ Number</th>
<th>Industry; wood species</th>
<th>Exposure measure mg/m³; unless otherwise stated personal dust in GM (GSD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gandevia, AU, 1970 [27]</td>
<td>CS E&lt;sub&gt;high&lt;/sub&gt;: 30 E&lt;sub&gt;low&lt;/sub&gt;: 17</td>
<td>Saw Mill; red cedar</td>
<td>Area dust meas. N=? 250–270 particles/m³</td>
</tr>
</tbody>
</table>

**Countries:** AU: Australia; CA: Canada; FI: Finland; NZ: New Zealand; SW: Sweden; TA: Taiwan; UK: United Kingdom; US: United States of America

**Type of study/Number:** C: controls; CS: Cross sectional study; E: exposed; FU: follow-up study; R-FU: Register follow-up study

**Exposure measure and statistics:** AM: arithmetic mean; Ass: associated; CI: confidence interval; Conc: concentration; Corr: correlation; Cum: cumulative; Diff: difference; DRR: dose response relationship; Exp: exposure; GM: geometric mean; GSD: geometric standard deviation; JEM: job exposure matrix; Inhal: inhalable; MDI: methylene diisocyanate; NS: non-significant; OR: odds ratio; OSB: oriented strand board; P: population; Pred: predicted; RR: relative risk; Resp: respirable; RPM: residual particulate matter; SD: standard deviation; S: significant; W: worker; WS: wood solids

**Symbols symptoms and objective measurements:** AS: asthma, BN: blocked Nose; CB: chronic bronchitis; CJ: conjunctivitis; CO: cough; CT: chest tightness; EYD: eye irritation; FEV<sub>1</sub>: forced expiratory volume in one second; FVC: forced vital capacity; IN: itchy nose; NAD: nasal discomfort; NAL: nasal lavage; RH: rhinitis; RN: runny nose; SOB: shortness breath; SN: sneezing; WR: work related; WH: wheeze.

or less exposed wood workers this misclassification might attenuate the dose-response relation. For example, Douwes *et al.* [19] ascribed misclassification of exposure as the reason for finding associations between being exposed and non-exposed workers for WR asthma symptoms, without being able to show any association to exposure level.

There are large differences in exposure level in the papers reviewed. When dust measurements were performed, low exposure levels of total or inhalable dust ranged from < 0.05–2.9 mg/m³ (AM or GM) and high exposure levels from 0.6–12 mg/m³. Compared to the dry wood industry reviewed in [41], there is a tendency towards lower exposure levels in the fresh wood industry, which is also clear from the European wood dust exposure survey from 2006 [45].

All but 5 follow-up studies were cross-sectional studies. A cross-sectional design hampers the possibilities to study associations between exposure and chronic diseases with latency time, for example asthma, chronic bronchitis and chronic impairment of lung function. In addition, a “healthy worker effect”, i.e. a tendency of workers experiencing respiratory complaints to leave a dusty job or to transfer to less dusty jobs, can cause skewing of risk estimates due to selection bias.

Ideally, cases and controls should be identical, apart from contrasts in exposure, and in most studies other industrial workers were selected as controls, while in some studies groups that probably differed markedly from the workers in the wood industry had been chosen (including office workers, general population) making interpretation difficult [16, 19, 25, 31, 46, 53].

Smoking is strongly causally related to the development of respiratory symptoms and decline in lung function, including COPD and chronic bronchitis, and therefore we have excluded studies without information on smoking. The
expected lung function depends on age, sex and height, and these factors have generally been included in the studies. Atopy is a known risk factor for asthma and rhinoconjunctivitis, but only some studies have taken atopic status into consideration [5, 31, 34, 35, 57, 64]. Although only a few studies revealed significant associations between wood dust exposure and occurrence of asthma and WRA, it is evident when looking across studies, that a consistent pattern of elevated prevalence’s and OR’s of asthma and asthmatic symptoms is revealed. The positive findings were confirmed in the only follow-up study [33]. No clear pattern between exposure level or duration and prevalence of asthma is seen across studies, i.e. very heterogeneous methodologies across a wide range of countries make it difficult directly to compare the different studies.

From the studies reported in this review it seems evident that exposure to fresh wood dust may cause CB. All but one study reported OR’s above 1.0 and several studies reported significant OR’s above 2.0 when comparing woodworkers to controls. CB could be related to exposures mostly or only present when handling wet wood, for example, moulds and endotoxin, and therefore one might argue it is not an effect of wood dust exposure per se. On the other hand, studies in the dry wood industry with little or no exposure to these have reported high OR’s [41].

Coughing is an unspecific symptom, which may reflect acute irritation of the airways and toxic alveolitis, as well as diseases like asthma, bronchitis, COPD or allergic alveolitis. Coughing and work-related coughing in relation to wood dust exposure seems to be a consistent finding across studies. Eduard et al. found DRR between prevalence of coughing and the exposure level of mould spores among wood trimmers [24], which suggests the microbial exposure to be of importance. However, increased prevalence of coughing has also been found in the dry wood industry, where studies revealing DRR between wood dust exposure per se and coughing support an inherent wood dust effect [41].

An acute obstructive effect of fresh wood dust exposure during workdays or during work weeks seems likely, as most studies measuring lung function showed a post-shift decline in lung function, although DRR did not support the finding for fresh wood, as opposed to a number of studies in the dry wood industry [41].

When studying lung function, a cross-sectional design as used in most of the reviewed papers is at best suboptimal. Even so, a number of studies revealed reduced baseline lung function (FEV1, FVC, or FEV1/FVC) among wood workers, and some studies revealed an association to current exposure or to years of exposure. The two follow-up studies [28, 53] investigating trends in lung function showed conflicting results. Both studies were performed among low exposed workers, but wood types differed as Noertjojo et al. studied a cohort exposed to RC, a known
asthmagen while RC exposed workers were excluded by Glindmeyer et al. Individual exposure assignments in both studies were based on JEMs and exposure duration. Nortjojo et al. based JEM on measurements of total dust, whereas Glindmeyer et al. divided measurements into wood solids (WS), residual particulate matter (RPM) and 3 size fractions. While Glindmeyer et al. found no association between any size fraction of WS and change in lung function, they reported significant effects of inhalable and thoracic dust on excess annual decline in lung function in the pooled population, including workers exposed to dry wood, but ascribed the findings to the RPM component of the respirable fraction. In the dry wood industry, one follow-up study of equally low exposed workers showed a DRR between wood dust exposure (baseline and cumulative) and decline in FEV₁ and FVC among female workers, supporting a chronic effect of wood dust, suggested as being caused by a greater susceptibility for females [41, 42].

There seem to be a consistent trend across studies on rhinitis, nasal symptoms, conjunctivitis, and eye irritation supporting an effect on exposure to fresh wood dust on nasal mucosa and conjunctiva. This is in accordance with findings from studies of exposure to dry wood [41].

The mechanisms for wood dust inducing respiratory impairment are far from being fully understood. For RC, a low molecular compound, plicatic acid has been revealed to be a causal factor, and both immunological and non-immunological mechanisms are involved [10]. Apart from RC no causal agent has consistently been disclosed. Specific sensitization has been reported, but type 1 allergy is not suspected to be a major cause of wood dust induced asthma [1, 17, 59, 66].

Apart from IgE mediated sensitization several other mechanisms are possible. Animal studies have shown that wood components, for example the major constituent in pine resin abietic acid, causes direct toxicity via lytic damage to alveolar, tracheal and bronchial epithelial cells [6]. Wood dust extracts from both hard and soft wood are able to induce the release of pro-inflammatory mediators from macrophages [47, 49], express and induce the release of inflammatory mediators in human epithelial cell line [12], and modulate the expression of cytokines and chemokines [48].

Workers handling fresh wood are concurrently exposed to inflammatory components like moulds, bacteria and natural volatile components of fresh wood. Mould exposure may, apart from asthma, lead to allergic or toxic alveolitis, which has been described in sawmill workers and wood trimmers [9, 23, 32, 55]. Symptoms consistent with alveolitis, for example, coughing, wheezing, dyspnoea, are also associated to asthma and bronchitis. Hence, the different diseases, as well as the exposure of relevance (wood dust, microorganisms), can hardly be disentangled in an epidemiological setting. Biohazards, mostly endotoxins and mould exposure, have mainly been studied at sawmills processing fresh wood [22, 51], but have also been found at lower concentrations in the dry wood industry associated to chronic bronchitis [4], and cross-shift decrease in lung function [51]. Thus, respiratory effects caused by work in the fresh wood industry is probably a combination of exposure to wood dust per se and other exposures, such as endotoxins, glucans and mould spores.

Monoterpenes are volatile substances naturally occurring in pine and other coniferous trees and may be liberated mainly during handling of fresh wood. Terpenes have been documented as causing irritation of the mucous membranes, and are suspected of causing impairment of lung function and BHR at levels of 100–450 mg/m³ [2, 44]. Only one of the reviewed studies explored terpene exposure. Fransman et al. [25] found low levels in sawmills ranging from GM 0.5–4.4 mg/m³ and did not find respiratory symptoms associated to these low levels. On the contrary, studies solely focusing on terpene exposure [3, 50, 56] have shown considerably higher levels of terpenes with GM’s ranging from 35–250 mg/m³. In one Swedish study of sawyers exposed to levels above 150 mg/m³, more BHR was revealed compared to lower exposed sawmill workers [50]. It was suggested that oxidative products of monoterpenes or abietic acid could cause airway inflammation through immune reactions. This was supported in an experimental study, which showed increased alveolar cell concentration, mainly macrophages in BAL after exposure to 450 mg/m³ of terpenes [43].

It has been documented that processing of plywood may cause exposure to formaldehyde [50] and asthma symptoms among woodworkers exposed to formaldehyde alone or in combination with wood dust [38]. A number of the reviewed papers in fact included evaluations of the formaldehyde concentration [25, 34, 35] and found formaldehyde levels ranging from 0.04–0.33 mg/m³. A health-based recommendation 8-hour time-weighted occupational exposure limit (OEL) of 0.15 mg/m³ has been recommended in the Nederland’s [21]. In the reviewed papers, it is generally not possible to distinguish the effects of wood dust and formaldehyde, although in one recent study [25] at ply mills, an association between asthma symptoms and formaldehyde was revealed, with the highest level of GM 0.16 mg/m³. Thus, it cannot be rejected that formaldehyde alone or in combination with wood dust may have influenced results, especially from the part of the industry processing plywood.

In conclusion, this review supports, despite the limitations in study design and exposure assessments, that wood dust exposure is a risk factor for development of asthma, chronic bronchitis, rhino-conjunctivitis and chronic impairment in lung function. The mechanisms are mostly unknown. Concurrent exposures, such as moulds, endotoxin and terpenes, contribute to the health effects in the wet wood industry.

Acknowledgements

The study was supported by the Danish Work Environment Foundation; Viborg County; Danish Medical Research Council; Health Insurance Fund; and Danish Lung Association.
REFERENCES


