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# NON-MALIGNANT RESPIRATORY DISEASES AND OCCUPATIONAL EXPOSURE TO WOOD DUST. PART II. DRY WOOD INDUSTRY

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**Abstract:** This paper reviews the literature on associations between dry wood dust exposure and non-malignant respiratory diseases. Criteria for inclusion are epidemiological studies in English language journals with an internal or external control group describing relationships between dry wood dust exposure and respiratory diseases or symptoms. Papers took into consideration smoking and when dealing with lung function age. A total of 37 papers forms the basis of this review. The results support an association between dry wood dust exposure and asthma, asthma symptoms, coughing, bronchitis, and acute and chronic impairment of lung function. In addition, an association between wood dust exposure and rhino-conjunctivitis is seen across the studies. Apart from plicatic acid in western red cedar wood, no causal agent has consistently been disclosed. Type 1 allergy is not suspected to be a major cause of wood dust induced asthma.

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**Key words: wood dust, wood industry, epidemiology, lung function, occupational asthma, chronic bronchitis, COPD, occupational rhinitis, respiratory symptoms, review, Denmark.**

## INTRODUCTION

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

Wood is processed in many industries including sawmills processing fresh wood, ply wood mills, 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 [24, 27, 50].

Wood dust is a known inducer of cancer in the nasal cavity and recent reviews have focused on this [19, 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. Alhough the occurrence of non-malignant respiratory diseases related to wood dust has been reviewed earlier [22, 30, 71], a number of studies have been performed in recent years. Furthermore, the earlier reviews 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 are warranted. This review focuses on wood dust exposure associated to dry wood. A second review focuses on fresh wood and mixed wood exposure [41]. In this review, we did not include 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 [11, 31, 38, 57, 65].

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## METHODS

The literature search covered Medline for papers published in English for the period 1969–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 was epidemiological studies describing associations between upper or lower respiratory diseases or symptoms and exposure to wood dust. Studies not having an internal control group (high or low exposure) or an external control group were excluded, as were papers which did not take smoking into consideration, or which did not adjust for age when dealing with lung function.

This review includes 37 original papers with exposure to dry wood. To allow for comparison between papers, odds ratios (OR) for symptoms from data provided in the papers, whenever OR's were not stated, were calculated with Chi square test using exact confidence intervals.

Chronic bronchitis was defined as daily coughing and phlegm for at least 3 months during at least 2 consecutive years [17].

## RESULTS

In Table 1 the main results from the reviewed papers are tabulated. The review will focus on: asthma, asthma symptoms, coughing, chronic bronchitis, rhino-conjunctivitis, and impairment in lung function.

**Asthma and asthma symptoms.** Sixteen studies, including two follow-up, one nested case-control-study and 13 cross-sectional studies have reported on asthma, asthma symptoms or clinical testing for asthma in relation to exposure to dry wood dust.

Jacobsen *et al.* [42] reported OR's of ever asthma and asthma symptoms in a 6 yr follow-up period. They found OR 3.4 (0.9–13) and 1.3 (0.6–2.8) for female woodworkers compared to controls, respectively. When they restricted the follow-up population to subjects with no respiratory symptoms at all at baseline, the OR for asthma symptoms increased to 11 (1.3–97).

In a register-based population study Heikkilä *et al.* [34] 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 dry wood RR (95% CI) varied between 0.9 (0.6–1.4) (females furniture industry) and 2.5 (1.3–4.6) (males manufacturer of wooden containers),

Nine cross-sectional studies from the dry wood industry reported prevalence's of **asthma** based on questionnaire information of **ever asthma** (4.3–11%) [58, 64, 68, 69], **physician diagnosed asthma** (2.6–9.6%) [15, 64], **current asthma attacks** (2.7%) [60], or **work-related asthma (WRA)** (23%) [9]. Two studies did not report a definition of asthma [33, 37].

The reported prevalence's of asthma were increased in wood workers compared to non-exposed controls or groups with lower exposure in all but one [37] of these studies, although only one reported significant differences [68]. One large study reported a dose response relation (DRR) between exposure and self-reported physician diagnosed asthma among female workers with an OR 6.5 (1.1–39) for the highest exposed group, most pronounced for smokers [64].

In 9 studies prevalences of self reported **wheezing** (7–41%) [6, 37, 55, 58, 60, 64, 66, 68, 69], **chest tightness** (9–41%) [55, 58, 64] or shortness of breath (SOB) with wheezing (6–12%) [58, 60, 69] were reported with OR's ranging from (0.7–4.1), 4 studies with significant differences [6, 60, 64, 66]. One study reported non-significant findings for symptoms without providing details [29].

Three studies [9, 55, 60] reported increased frequencies of **WRA symptoms** (7–37%) among woodworkers compared to controls with OR ranging from 2.4–7.1, significant for all but one study [55]. Finally, one study reported increased (WRA) symptoms, OR 6.4 (1.6–26) among highly exposed workers compared to low exposed workers [61].

In 6 studies, **clinical testing for asthma** was included. Norrish *et al.* [55] used PEF variability during work weeks and time off, and diagnosed 4 of 44 woodworkers with occupational asthma (OA). Carosso *et al.* [18] used a non-specific bronchial provocation test to identify 20 with possible asthma among 90 woodworkers. Schlünssen *et al.* [61] in a nested case control study used non-specific bronchial provocation test, bronchodilator induced reversibility (BIR) or increased PEF variability in combination with symptoms in order to define clinical asthma and revealed a positive DRR between current exposure level and clinical asthma or bronchial hyper responsiveness (BHR).

In a study of beech and oak exposed workers, Bohadana *et al.* [15] revealed a positive DRR between BHR and cumulative exposure. In contrast 2 studies [5, 69] did not find any difference in the distribution of BHR in relation to wood dust exposure.

**Chronic bronchitis and cough.** **Chronic bronchitis (CB)**, fulfilling diagnostic criteria, was described in one follow-up study [42] and 4 cross-sectional studies [6, 9, 64, 66].

In the 6 yr follow-up study by Jacobsen *et al.* [42], they for CB found OR 8.9 (1.1–71) for female woodworkers compared to controls, and a DRR between baseline exposure to inhalable wood dust and CB was revealed, with OR 6.0 (1.2–29) in the highest exposed group.

Cross sectional studies reported prevalence's ranging from 1.4–32% and OR's ranging from 1.2–13.8, being

significant in [6, 9]. In [9], they found a significant correlation between current inhalable and respirable wood dust level and CB.

For **coughing** Jacobsen *et al.* (16) found OR 2.8 (1.3–6.1) for female woodworkers compared to controls, and a DRR between baseline exposure level to inhalable wood dust and daily coughing was revealed, with OR 3.8 (1.5–9.7) in the highest exposed group.

Prevalence's of coughing ranging from 10–51% (OR 0.7–7.6) have been reported in 9 cross-sectional studies [15, 37, 55, 58, 60, 64, 66, 68, 69]. For **WR coughing**, the prevalence were 23–74%, with OR between 3.8–∞ [9, 53, 55, 60]. In studies that compared exposed workers to controls [6, 9, 15, 29, 37, 55, 58, 60, 64, 66, 68] and/or compared different exposure levels [53, 58, 64, 69], all but [15, 29, 37, 58, 68, 69] reported significant results. Two studies revealed a positive DRR between current wood dust exposure [3] or duration of employment [66] and frequency of coughing.

**Post-shift decline in lung function.** Thirteen cross-sectional studies have investigated acute obstructive changes in lung function in the dry wood industry. Eleven studies found a significant post-shift decline in $FEV_1$ [7, 12, 37, 50, 53, 62, 64], FVC [7, 37, 50, 53], or $FEV_1$/ FVC [50] among woodworkers. Schlünssen *et al.* found a post-shift decline in $FEV_1$ among woodworkers handling pine [64], and revealed a DRR between post-shift decline in $FEV_1$ and exposure to current wood dust level among smokers, most pronounced for pine workers [62]. Beritic-Stahuljak *et al.* [12] found a DRR between post-shift decline in $FEV_1$ and exposure to current softwood level but not to hardwood.

Mandryk *et al.* [50] found a DRR between post-shift decline in $FEV_1$ and FVC and current inhalable and respirable dust level. Two studies [5, 29] did not find any post-shift changes in $FEV_1$ or FVC.

**COPD.** Seventeen studies including 2 follow-up and 15 cross-sectional studies have reported on baseline lung function parameters and exposure to wood dust.

In the 6 yr follow-up study by Jacobsen *et al.* [43] they found a DRR between baseline as well as cumulative wood dust exposure and decline in $FEV_1$ and FVC. This association was only seen for female workers. In addition, an association between continued employment in the wood industry and decline in lung function was reported, compared to the women who left the wood industry in the follow-up period. In contrast, Glindmeyer *et al.* [28] in a 5 yr follow-up study found no association between dry wood dust of any size fraction and change in lung function indices.

Ten cross-sectional studies found associations between baseline lung function parameters and exposure to wood dust. Carosso *et al.* [18] found a DRR between decline in $FEV_1$ as well as DLCO (carbon monoxide diffusion coefficient) and years of employment. Haxhiu *et al.* [33] reported more wood dust exposed workers compared to controls (6 *vs* 3.7%) to have a $FEV_1/FVC < 70\%$. Likewise Shamssain [66] reported 30 *vs* 17% to have a $FEV_1/FVC < 70\%$. In addition, he found FVC, $FEV_1$, $FEV_1$/FVC and PEF decreased among male exposed subjects, and found a DRR between duration of employment and decreased FVC and $FEV_1$/FVC. Hollness *et al.* found a DRR between decreased $FEV_1$ and an exposure-time index, but did not find an association between current dust concentration and $FEV_1$ or FVC, nor was this found by Al-Zuhair *et al.* [7, 37]. Mandryk *et al.* [50] found lower values of $FEV_1$, FVC and $FEV_1$/FVC among furniture workers compared to those non-exposed. They found a DRR between decrease in $FEV_1$ and FVC and the actual dust concentration, both for the respirable and the inhalable dust fraction, but all baseline lung function indices was positively correlated with the number of years of exposure to wood dust.

Whitehead *et al.* [72] found $FEV_1$/FVC decreased among exposed woodworkers, compared to non-exposed controls. Two studies [35, 36] found significantly lower values for FVC and/or $FEV_1$ among subject exposed to dust from MDF and other wood species, compared to controls.

Beritic'-Stahuljak *et al.* [28] found a DRR between duration of exposure and low $FEV_1$ for subgroups of hardwoods (iroko, mahogany and combined hardwood) and low $FEV_1$ respectively for subgroups of softwoods (poplar and mixed wood).

Finally Osman *et al.* [56] reported decreased $FEV_1$, FVC and increased $FEV_1$/FVC among exposed versus non-exposed workers, but found a positive DRR between $FEV_1$ and FVC and dust level, at least for workers employed for less than 10 yrs.

Six studies found no association between wood dust exposure and baseline FVC or $FEV_1$ [5, 15, 29, 56, 64, 69].

**Rhino-conjunctivitis.** Four studies have reported significant increased frequencies of rhinitis (10–52%) among workers exposed to wood dust compared to non-exposed controls [37, 58, 60] or groups with lower exposure [69] with OR's ranging from 2.3–5. Two studies did not find any association between rhinitis and wood dust exposure [29, 64].

Ten papers have reported increased frequencies of nasal symptoms, i.e. **congestion** (24–61%), **rhinorrhea** (20–45%), **sneezing** (39–77%), **nasal itching** (6–21%) and **nasal irritation/discomfort** (26–64%) among workers exposed to wood dust compared to non-exposed controls [6, 35, 36, 54, 55, 56, 58, 59, 66, 68] or groups with lower exposure [73], with OR's ranging from 0.7–∞. Significant raised frequencies of at least one symptom were found in all studies.

Four studies reported prevalence's of **WR rhinitis** (9–26%) [53, 60] or **WR nasal symptoms** (19–63%) [9, 15, 29, 56] of woodworkers *vs* controls with OR's of (3.7–∞) significant in all but one study [15]. One study found an association between duration of exposure and WR nasal symptoms [56]. Two studies found an association between

**Table 1.** Dry wood, characteristics of studies included. If not otherwise stated, symptom risk is given as OR.

| Author, country, year | Type of study/Number | Industry; wood species | Exposure measure mg/m$^3$; unless otherwise stated personal dust in GM (GSD) |
|---|---|---|---|
| Jacobsen, DK, 2009 [42]<br>From same pop. as [43] | FU 6 yr. of [64]<br>E: 1,377<br>C: 297 | Furniture; various, mainly pine, particleboard, fibreboard | Inhal. dust<br>$N_{baseline}$ = 2,217,<br>GM: 0.9 (2.1)<br>$N_{FU}$ = 1,355,<br>GM: 0.6 (1.6)<br>JEM cum. exp. median (range): 3.6 (0–7.6) mg yr./m$^3$<br>Also terpenes and formaldehyde |
| Sripaiboonkij, TA, 2009 [68] | CS<br>$E_{high}$: 42<br>$E_{low}$: 61<br>C: 76 | Furniture; rubber tree | Inhal. dust<br>E: N=14<br>AM range: 0.4–2.9<br>C: N=2<br>AM 0.02<br>JEM: $E_{low,}$ $E_{high}$, exp. chemicals, exp. cyanoacrylat glue |
| Osman, TU, 2009 [56] | CS<br>E: 328<br>C: 328 | Furniture; MDF, beech, pine, fibreboard | Dust total N=?<br>AM 2.04 (1.53)<br>JEM<br>Yr. woodworking |
| Glindmeyer, US, 2008 [28] | FU 5 yr.<br>E: 779 | Furniture, cabinet; various | Dust N=1, 739<br>3 size fractions (<4<10<100) µm<br>377 analyzed<br>% WS and % RPM<br>GM resp: 0.16–0.23<br>%WS mean: 12–34<br>GM inhal: 1.2–2.1<br>%WS mean: 37–69<br>JEM, mean pers. exp. during FU (mg/m$^3$) for 3 size fractions, WS and RPM |
| Jacobsen, DK, 2008 [43] | FU 6 yr. of [64]<br>E: 1,112<br>C: 235 | Furniture; various, mainly pine, particleboard, fibreboard | Inhal. dust<br>$N_{Baseline}$ = 2,217,<br>GM: 0.9 (2.1)<br>$N_{FU}$ =1,355, GM: 0.6 (1.6)<br>JEM, cum. exp., median (range): 3.8 (0–7.6) mg yr./m$^3$ |

| Lower airway symptoms OR (95% CI) * OR calculated | Upper airway symptoms OR (95% CI) * OR calculated | Objective measurements | Confounders included |
|---|---|---|---|
| **$E_{females}$ *vs* C:**<br>AS: 3.4 (0.9–13)<br>$AS_{symp.}$: 1.3 (0.6–2.8)<br>WH: 1.6 (0.7–3.4)<br>CO: 2.8 (1.3–6.1)<br>CB: 8.9 (1.1–71)<br><br>**No baseline resp. sympt.**<br>$AS_{symp.}$: 11 (1.3–97)<br>WH: 5.9 (1.2–30)<br>CO: 5.5 (1.9–16)<br><br>DRR between baseline exp. & incidence of CO & CB<br>No ass. to cum. exp. | | | Sex, age, smoking, atopy |
| **E *vs* C:**<br>AS: 6.1 (0.7–54)<br>WH: 2.7 (0.8–8.7)<br>OB: 1.7 (0.6–4.8)<br>CO: 1.7 (0.6–4.7)<br><br>**$E_{low}$ *vs* C:**<br>AS: 8.4 (1.1–67)<br>WH: 2.5 (0.7–8.8)<br>SOB: 2.0 (0.7–5.8)<br>CO: 2.1 (0.7–6.3)<br><br>**$E_{high}$ *vs* C:**<br>WH: 3.0 (0.8–12)<br>SOB: 1.3 (0.4–4.6)<br>CO: 1.0 (0.3–3.7)<br>No DRR between Symptoms and chemical or glue exp. | **E *vs* C:**<br>NAD: 2.8 (1.2–6.9)<br>EYD: 1.1 (0.5–2.7)<br><br>**$E_{low}$ *vs* C:**<br>NAD: 3.7 (1.5–9.3)<br>EYD: 1.1 (0.4–2.8)<br><br>**$E_{high}$ *vs* C:**<br>NAD: 1.7 (0.6–4.9)<br>EYD: 1.3 (0.4–3.7)<br><br>No DRR chemical or glue exp., except for glue *vs* NAD | NS association between exp. and $FEV_1$, FVC, $FEV_1$% pred., FVC% pred.<br><br>NS DRR between dust or glue and $FEV_1$, FVC, $FEV_1$% pred., FVC% pred. | Age, sex, height, smoking, educational level |
| | **WR E *vs* C:**<br>BN: 54 *vs* 0%, S<br>RN: 24 *vs* 0%, S<br>EYD: 41 *vs* 0%, S<br><br>**E>10 yr. *vs* E<10 yr.:**<br>BN: 3.1* (1.9–5.1)<br>RN: 1.8* (0.97–3.4)<br>EYD: 2.8* (1.5–4.2) | **E *vs* C:**<br>↓ $FEV_1$% pred., ↓FVC% pred., ↑$FEV_1$/FVC% pred.<br><br>**Exp. <10 yr.:**<br>Ehigh *vs* Elow ↑$FEV_1$% pred., ↑FVC% pred.<br><br>**Exp. > 10 yr.:**<br>NS diff $E_{HIGH}$ *vs* $E_{LOW}$ | Age, sex (all males), smoking, height ?? Ref. material for expected lung function not given |
| | | No ass between WS or RPM and change in lung function indices. ($FEV_1$, FVC, $FEV_1$/FVC, $FEF_{25–75}$) for any size fraction | Age, sex, height, weight change, ethnicity, smoking, baseline lung function |
| | | **Females:**<br>DRR between baseline exp., cum. exp., yr. woodworking during FU & ↓$FEV_1$, ↓FVC<br><br>**Males:**<br>No ass between exp. (baseline, cum. or yr.) & lung function | Sex, age, smoking, height, weight, baseline resp. symptoms, atopy |

| Author, country, year | Type of study/Number | Industry; wood species | Exposure measure mg/m$^3$; unless otherwise stated personal dust in GM (GSD) |
|---|---|---|---|
| Heikkilå, FI, 2008 [34] | R-FU Registers: Wood prossing industries.<br>Incident AS reimbursement register<br><br>Sub-cohort<br>Dry wood<br>$E_{furniture}$: 5,036<br>$E_{container}$: 387<br>C: 12, 839 | Furniture, wooden containers; pine, spruce, birch. | For whole study incl. fresh wood: JEM 5 exp. levels based on industrial meas. |
| Priha, FI, 2004 [59] | CS<br>$E_{MDF}$: 22<br>$E_{Wood}$: 23<br>C: 15 | Furniture; MDF, birch, pine | Inhal. dust N=45<br>$GM_{MDF}$ 1.2 (2.0)<br>$GM_{Wood}$ 1.3 (2.7)<br>Formaldehyde, VOC |
| Schlünssen, DK, 2004 [61] | Nested CC<br>E: 302<br>4 exp. levels<br><br>C: 71 | as [64] | Inhal. dust N=347<br>GM: 1.0 (2.0) |
| Schlünssen, DK, 2004 [62] | CS<br>E: 1,560 | as [64] | Inhal. dust N=2,217<br>GM: 1.0 (2.1) |
| Schlünssen, DK, 2002 [63] | CS<br>E: 161<br>3 exp. levels<br>C: 19 | as [64] | Inhal. dust N=140<br>AM 1.2 (0.6) |
| Schlünssen, DK, 2002 [64] | CS<br>E: 2,033<br>3 exp. levels<br><br>C: 474 | Furniture; various, mainly pine, particleboard, fibreboard | Inhal. dust N=1,579 + JEM for 382<br>GM: 0.9 (2.1) |
| Milanowski, PO, 2002 [53] | CS<br>E: 27<br>$C_1$: 21-varnish<br>$C_2$: 41 | Furniture; fibreboard, chipboard | Yr. of exp.<br>Department |
| Rongo; TA, 2002 [60] | CS<br>E: 546<br>2 exp. levels<br>C: 565 | Workshops; various hard and softwood | Inhal. dust N=106<br>GM: 3.9 (2.3)<br>JEM |

| Lower airway symptoms OR (95% CI) * OR calculated | Upper airway symptoms OR (95% CI) * OR calculated | Objective measurements | Confounders included |
|---|---|---|---|
| **RR: $AS_{men}$:**<br>$E_{furniture}$ *vs* C:1.1 (0.8–1.5)<br>$E_{container}$ *vs* C: 2.5 (1.3–4.6)<br>**RR: $AS_{female}$:**<br>$E_{furniture}$ *vs* C: 0.9 (0.6–1.4) | | | Sex, age. No adj. for smoking, but has been considered |
| | NAD:<br>$E_{MDF}$ : 36%<br>$E_{Wood}$: 26%<br>C: 0% | **NAL pre and post shift $E_{MDF}$ & $E_{Wood}$ post shift *vs* C:** ↑protein.<br>**Post *vs* pre-shift:**<br>$E_{MDF}$ & $E_{wood}$ ↑Nitric oxide<br>$E_{wood}$ ↓TNF-α<br>No diff. cytokines or cell counts | Not adjusted for smoking, but has been considered |
| **E *vs* C:**<br>$AS_{symp.}$: 1.5 (0.8–2.8)<br>WR $AS_{symp.}$: 1.1 (0.5–2.6)<br>**4 exp. levels**<br>DRR WR $AS_{symp.}$<br>**$E_{high}$ *vs* $E_{low}$:**<br>$AS_{sympt.}$ 2.3 (1.0–5.6)<br>WR $AS_{symp.}$: 6.4 (1.6–27) | | **E *vs* C:**<br>$AS_{symp.}$ + BHR: 2.2 (0.8–6.5) Clinical AS: 2.1 (0.7–6.4)<br>**4 exp. levels**<br>DRR for $AS_{symp.}$ + BHR & for Clinical AS<br>**$E_{high}$ *vs* $E_{low}$:**<br>$AS_{symp.}$ +BHR: 18.3 (2.0–171)<br>Clinical AS: 3.3 (1.1–10) | Smoking, age, sex, atopy |
| | | DRR between exp. and post-shift $FEV_1$ for non-smokers | Smoking, age, sex, height, weight, atopy |
| | Self-rated BN↓<br>during work shift related exp. | Acoustic rhinometry: DRR between exp. and mucosal swelling during work shift | Smoking, age, sex, height, weight, atopy |
| **E *vs* C:**<br>NS diff<br>**$E_{med}$ *vs* $E_{low}$:**<br>AS: 1.3 (0.8–2.3)<br>WH: 1.6 (1.1–2.2)<br>CT: 1.5 (0.7–3.0)<br>CO: 1.4 (1.1–1.8)<br>CB: 1.2 (0.8–1.9)<br>**$E_{high}$ *vs* $E_{low}$**<br>AS: 1.4 (0.8–2.7)<br>$AS_{female}$: 6.5 (1.1–39)<br>WH: 1.4 (0.9–2.1)<br>CT: 2.2 (1.0–4.9)<br>CO: 1.4 (1.0–1.9)<br>CB: 1.4 (0.8–2.3)<br>Neg. ass CO and yr. exposed | **E *vs* C:**<br>NS for RH, RN, IN, CJ | No relation between exp. and $FEV_1$, FVC: Sig. Post shift decline in $FEV_1$ among E using pine *vs* other E. | Smoking, sex, age, height, atopy |
| **WR E *vs* $C_2$:**<br>CO: 74 *vs* 0%, S<br>SOB: 18 *vs* 0%, S<br>**WR E *vs* $C_1$:**<br>CO: 9.1* (2.1–43)<br>SOB: 4.6 (0.4–226) | **WR E *vs* $C_2$:**<br>RH: 26 *vs* 0%, S<br>CJ: 26 *vs* 0%, S<br>**WR E *vs* $C_1$:**<br>RH: 0.5 (0.1–1.9)<br>CJ: 0.5 (0.1–1.9) | | Smoking, sex, age.<br>Symptoms not adjusted |
| **E *vs* C:**<br>AS: 0.8 NS. WH: 1.3 (0.8–.2.0)<br>SOB+WH: 1.9 (1.1–3.4)<br>CO: 1.6 (1.2–2.0)<br>**WR E *vs* C:**<br>WH/SOB+WH/CT: 5.4 (3.4–8.5)<br>CO: 3.8 (2.7–5.9) | **E *vs* C:**<br>RH: 2.3 (1.4–3.6)<br>CJ: 2.3 (1.4–4.6)<br>**WR E *vs* C:**<br>RH: 3.7 (2.0–6.6)<br>CJ: 3.1 (1.3–9.8) | | Smoking, age, sex (all males) |

| Author, country, year | Type of study/Number | Industry; wood species | Exposure measure mg/m$^3$; unless otherwise stated personal dust in GM (GSD) |
|---|---|---|---|
| Bohadana, FR, 2000 [15] | CS<br>E: 114<br>4 exp. levels<br>C1: 13<br>C2: 200 (historical) | Furniture; beech, oak | Inhal. dust N=443 (other worksites, 10 yr. prior to study)<br>JEM<br>Cum. exp. index |
| Alwis, AU, 1999 [9] | CS<br>E: 82<br>C: 34 | As [50] | As [50] |
| Mandryk, AU, 1999 [50] | CS<br>E: 82<br>C: 34 | Joinery; mixed species including red cedar | Inhal. dust N = 66<br>GM 3.7 (3.7)<br>Yr. of exp.<br>Also resp. dust, endotoxins, glucans, bacteria, |
| Talini, IT, 1998 [69] | CS<br>E: 143<br>C: 63 (assemblers) | Furniture; mainly pine and beech | Total dust. N = 17<br>GM (range) 2.7 (0.1–9.0) |
| Åhman, SW, 1996 [5]<br>From same pop. as [6] | CS<br>E: 40<br>C: 39 | as [6] | as [3] |
| Åhman, SW, 1996 [1] | CS<br>E: 39<br>C: 31 | as [6] | as [3] |
| Åhman, SW, 1996 [3]<br>From same pop. as [6] | CS<br>E: 39<br>C: 32 | as [6] | Total dust N=39<br>AM (range): 0.6 (0.1–1.2)<br>Also resp. dust, terpenes |
| Åhman, SW, 1995 [4] | CS<br>E: 24<br>C: 24 | as [6] | Classes pr. week (teachers) |
| Åhman, SW, 1995 [6] | CS<br>E: 130<br>C: 112 | Wood shops; various, mostly pine | Assessment of hygienic parameters |
| Norrish, NZ, 1992 [55] | CS<br>E: 44<br>C: 38 | Furniture; rimu | Inhal. dust N = 78<br>median (range)<br>3.6 (1.0–25.4)<br>Also formaldehyde |
| Shamssain, SA, 1992 [66] | CS<br>E: 145<br>C: 152 | Furniture; pine, MDF | Area dust N =?<br>AM (SD): 3.8 (1.3)<br>Yr. of exp. (employment range: 1–20) |

| Lower airway symptoms OR (95% CI) * OR calculated | Upper airway symptoms OR (95% CI) * OR calculated | Objective measurements | Confounders included |
|---|---|---|---|
| **E *vs* $C_2$:**<br>AS: 2.2* (0.5–9.2)<br>CO/PH: 1.2* (0.5–2.6)<br>CB: 2.4* (0.4–17) | **WR E *vs* $C_1$:**<br>RN: 19 *vs* 0%, NS<br>EYD; 14 *vs* 0%, NS<br>E: DRR sore throat and exp. | FVC, $FEV_1$, $FEV_1$/FVC: NS<br>DRR between BHR & cum. exp. | Smoking, sex, age, height |
| **WR E *vs* C:**<br>AS: 2.5* (0.5–24.1)<br>WH: 3.7 * (1.1–16)<br>CT: 2.7 * (0.9–8.8)<br>CO: 5.3 * (2.0–16)<br>CB: 3.5* (1.1–15) | **WR E *vs* C:**<br>BN: 4.2* (1.6–11)<br>RN: 4.9* (1.7–16)<br>IN: 1.3* (0.4–4.5)<br>SN: 4.0* (1.4–13)<br>CJ: 2.6* (0.3–123)<br>EYD: 1.9* (0.8–5.3) | | Smoking, sex (all males), age, |
| | | **E *vs* C:**<br>FVC↓, $FEV_1$↓, $FEV_1$/FVC↓<br>Post-shift decline FVC, $FEV_1$, $FEV_1$/FVC, PEF;<br>**E:** DRR dust & ↓ in $FEV_1$, FVC, DRR dust & postshift decline $FEV_1$, FVC, PEF | Smoking, sex (all males), age, height |
| **E *vs* C:**<br>AS: 3.7 (0.5–36)<br>WH: 3.4 (0.9–13)<br>SOB+WH: 3.1(0.4–29)<br>CO: 1.5 (0.4–6.6) | **E *vs* C:**<br>RH 5.0* (1.1–45) | **E *vs* C:**<br>FVC, $FEV_1$, BHR: NS | Smoking, sex (all males), age, atopy |
| **E *vs* C start of week:**<br>NS diff. LAD<br>**E *vs* C end of week:**<br>LAD: 13% *vs* 5%<br>2.5* (0.8–8.0) | | **E *vs* C:** BHR: NS<br>$FEV_1$/FVC↓ during week E and C<br>**E:** positive DRR between dust and ↑CV% end of week<br>Positive DRR between terpene exp. end of week and $FEV_1$, FVC, DLCO. | Smoking, sex, age, height |
| | | **E *vs* C:**<br>↓Serial nasal peak expiratory flow rate during work week | Smoking, sex, age, |
| | **E *vs* C end of week:**<br>↑VAS nasal symptoms<br>**E:** ↑VAS during week | **E *vs* C:**<br>No diff rhinomanometry.<br>↓Nasal MCC end of week<br>↑Anosmia in E during week. | Smoking, sex, age, atopy (more atopy E) |
| | | **E *vs* C:** No diff. in NAL of inflammatory markers<br>**E:** Corr. between change in NAL neutrophils during week and exp. time | Smoking, age, sex, atopy |
| **E *vs* C:**<br>WH: 4.1 (1.3–13)<br>CO: 5.4 (2.5–12)<br>CB: 13.8 (3.1–61) | **E *vs* C:**<br>BN: 13.0 (6.2–28)<br>RN: 4.2 (1.9–11)<br>SN: 5.6 (2.7–11)<br>AN: 6.4 (2.3–17) | | Smoking, age, sex, height, atopy |
| **E *vs* C:**<br>WH/CT: 1.7 (0.6–4.8)<br>CO: 7.6 (1.4–52.7)<br>**WR E *vs* C:**<br>WH/CT: 2.8 (0.7–12)<br>CO: 32 *vs* 0%, S | **E *vs* C:**<br>BN: 6.0 (2.0–18)<br>RN: 4.6 (1.4–16)<br>SN: 7.4 (2.5–22)<br>EYD: 2.0 (0.7–5.7) | **E:** WR asthma (PEF + symptoms) found 4/44 | E and C matched smoking, age, sex, atopy |
| **E *vs* C:**<br>WH: 3.1* (1.2–9.1)<br>CO: 1.9* (1.2–3.2)<br>CB: 1.7* (0.8–3.9)<br>**E:** DRR yr. exp. CO | **E *vs* C:**<br>BN: 4.4* (2.5–7.7)<br>**E:** DRR yr. exp. BN | **E *vs* C:**<br>FVC, $FEV_1$, $FEV_1$/FVC, PEF ↓ in $E_{Male}$. $FEV_1$/FVC <70%: 30 *vs* 17%, p<0.01<br>**E:** DRR yr. exp. & ↓$FEV_1$/FVC, ↓ FVC ($E_{Male}$) | Smoking (all non-smokers), sex, age, ethnicity, height<br>Symptoms not adjusted |

| Author, country, year | Type of study/Number | Industry; wood species | Exposure measure mg/m$^3$; unless otherwise stated personal dust in GM (GSD) |
|---|---|---|---|
| Pisanello, AU, 1991 [58] | CS<br>E: 168<br>3 exp. levels<br>C: 46 | Furniture; various | Inhal. dust N = 171<br>GM (range): 2.9 (0.4–24.0)<br>Yr. of exp. |
| Holmström, SW, 1991 [35] | CS<br>$E_{MDF}$: 16<br>$E_{wood}$: 29<br>C: 36 | Industry?; MDF, various | Dust meas. N = ?<br>AM 1.4 |
| Holmström, SW, 1988 [36] | CS<br>E: 100<br>C: 36 | Furniture; MDF | Dust meas. N = ?<br>AM (SD): 1.7 (1.1) |
| Beritic-Stahuljak, YU, 1988 [12] | CS<br>$E_{softwood}$: 356<br>$E_{hardwood}$: 42 | Industry?<br>Softwood: pine,<br>Poplar. Mixed Hardwood: iroko, okoume, mahogany | Dust meas. N =?<br>$AM_{total}$: 0.8–40.1<br>Also respirable<br>Exp. index using yr. exp. |
| Goldsmidt, USA, 1988 [29] | CS<br>E: 55<br>C: 16 | Furniture; oak, maple, walnut, poplar, mahogany | Area dust meas.<br>N =?<br>Cum. month exp. (employed)<br>$Mean_{male}$: 222<br>$Mean_{female}$:108 |
| Carosso, IT, 1987 [18] | CS<br>E: 90<br>C: 53 | Furniture; mainly oak, aspen, pine | Yr. of exp. (mean employment 26 yr.) |
| Holness, CA, 1985 [37] | CS<br>E: 50<br>C: 49 | Furniture; | Inhal. dust N = 50<br>AM (SD): 1.8 (1.5)<br>Also resp. dust<br>Cum. exp. index: conc. x yr. in the company |
| Innocenti, IT, 1985 [39] | CS<br>E: 13<br>C: 24 | Furniture; chestnut | Dust. N=10<br>GM (range) 2.5–9.0 (1.9–26) |
| Haxhiu, YU, 1982 [33] | CS<br>E: 201<br>C: 109 | Furniture; beech | |
| Al-Zuhair, UK, 1981 [7] | CS<br>E: 124<br>C: 52 | Furniture; softwood, hardwood | Total dust; N=193<br>AM (range): 4.7 (0.5–8.3). |

| Lower airway symptoms OR (95% CI) * OR calculated | Upper airway symptoms OR (95% CI) * OR calculated | Objective measurements | Confounders included |
|---|---|---|---|
| **E *vs* C:**<br>AS: 1.3* (0.4–3.9)<br>WH: 0.7* (0.4–1.4)<br>CT: 0.6* (0.3–1.4)<br>CO: 0.7* (0.3–1.3)<br>**E:** No DRS yr. exp. or current exp. | **E *vs* C:**<br>RH: 2.7* (1.3–6.0)<br>BN: 2.4* (1.2–5.2)<br>RN: 6.8* (2.5–22)<br>SN: 3.3* (1.4–8.7)<br>IN: 0.9* (0.4–2.4)<br>**E *vs* C WR:**<br>EYD: 2.2* (1.0–5.6) | | Smoking, age, sex (all males) |
| **$E_{all}$ *vs* C:**<br>LAD: 2.5* (0.7–10)<br>**$E_{MDF}$ *vs* C**<br>LAD: 6.2* (1.6–30) | **$E_{all}$ *vs* C:**<br>BN: 2.6* (0.7–12)<br>RN: 4.3* (0.8–43)<br>IN: 0.8* (0.1–6.3)<br>AN: 4.3* (0.8–43)<br>EYD: 4.3* (0.8–43)<br>**$E_{MDF}$ *vs* C:**<br>BN: 4.8* (0.9–27)<br>RN: 5.7* (0.7–68)<br>IN: 2.5* (0.3–21)<br>AN: 13.2* (1.9–142)<br>EYD: 13.2* (1.9–142) | **E *vs* C:**<br>↓FVC, ↓ $FEV_1$ in $E_{MDF}$ & $E_{wood}$<br>Impaired MCC 15% *vs* 3% (NS)<br>Nasal mucosal swelling: NS | Smoking, sex, age, height, weight, |
| **E *vs* C:**<br>LAD: 4.0* (1.4–11) | **E vs C:**<br>NAD: 3.4* (1.4–7.9)<br>EYD: 4.5* (1.0–20) | **E *vs* C:**<br>↓FVC<br>Impaired nasal MCC<br>15 *vs* 3% (NS)<br>↑Mucosal swelling (NS)<br>↑Anosmia | Smoking, sex, age, height, weight, |
| | | **E:** post shift ↓$FEV_1$<br>$E_{softwood}$: DRR cum. yr. exp. & low $FEV_1$, FVC poplar & mixed pine<br>$E_{hardwood}$: DRR cum. yr. exp. & low $FEV_1$ | Smoking, sex, age, height |
| **E *vs* C:**<br>CO, WH, CT<br>(All NS, OR not given) | **E *vs* C:**<br>RH (NS OR not given)<br>**WR E *vs* C:**<br>SN: 4.1 (1.1–15)<br>EYE: 4.0 (1.0–16.6) | **E *vs* C:** No diff FVC, $FEV_1$,<br>**E:** Corr. ↓PEF during work shift & cum. month exp.<br>DRR between ↓PEF during work shift and fraction of particles <10 µm | Smoking, age, sex, height, atopy, ethnicity |
| | | **E *vs* C:** E ↓DLCO<br>DRR DLCO, $FEV_1$ & yr. of exp. (neg. correlation) | Smoking, age, height, weight |
| **E *vs* CV**<br>AS: 1.0* (0.1–14)<br>WH: 2.0* (0.5–8.1)<br>CO: 1.5* (0.6–3.9) | **E *vs* C:**<br>RH: 4.1* (1.3–15.7)<br>EYD: 3.8* (0.9–23) | **E *vs* C:** Greater post shift ↓ in FVC & $FEV_1$. No DRR.<br>Neg. correlation baseline $FEV_1$ & cum. exp. index | Smoking, age, height, sex? |
| | | **E *vs* C**<br>Turbinate Hypertrophy:<br>69 *vs* 50%, p=0.22<br>Anosmia:<br>30 *vs* 0%, p=0.01 | Sex (all males), age |
| **E *vs* C:**<br>AS: 2.8* (0.6–27) | | **E *vs* C:**<br>$FEV_1$/FVC% < 70%: 6 *vs* 4%<br>1.7* (0.5–7.3) | Smoking, sex (all males), age |
| | | **High exposed factory vs C:**<br>Post shift ↓FVC and ↓$FEV_1$<br>No DRR | Smoking, age, height, weight |

| Author, country, year | Type of study/Number | Industry; wood species | Exposure measure mg/m$^3$; unless otherwise stated personal dust in GM (GSD) |
|---|---|---|---|
| Wilhelmsson, SW, 1984 [73] | CS<br>$E_{Low}$ 192<br>$E_{High}$ 484 | Furniture; | Area dust. N=28<br>AM (range): 2.0 (0.3–5.1)<br>Self-assessment (little, moderate, high) |
| Whitehead, US, 1981 [72] | CS<br>$E_{softwood}$: 220<br>$E_{hardwood}$: 354 | Various. Hardwood: mostly Maple. Softwood: pine | Area dust. N = 100<br>Exp. index: conc. x yr. in the company<br>$E_{low}$: 0–2 mg yr./m$^3$<br>$E_{med}$: 2–10 mg yr./m$^3$<br>$E_{high}$: 10+ mg yr./m$^3$ |
| Black, UK, 1974 [14] | CS<br>E: 9<br>C: 12 | Furniture | |

**Countries:** AU: Australia. CA: Canada. DK: Denmark; FI: Finland; FR: France; IT: Italy; NZ: New Zealand; PO: Poland; SA: South Africa; SW: Sweden; TA: Tanzania; TU: Turkey; UK: United Kingdom; US: United States of America; YU: Yugoslavia

**Type of study/Number:** C: controls; CS: Cross sectional study; CC: Case control study; E: exposed; FU: follow-up study; R-FU: Register follow-up study; W: Wood dust

**Exposure measure and statistics:** AM: arimetric 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; MDF: medium density fibreboard; NS: non-significant; OR: odds ratio; Pred: predicted; RR: relative risk; Resp: respirable; RPM: residual particulate matter; SD: standard deviation; S: significant; VOC: volatile organic compounds; WS: wood solids

**Symbols symptoms and objective measurements:** AS: asthma, BN: blocked Nose; CB: chronic bronchitis; CJ: conjunctivitis; CO: cough; CT: chest tightness; CV%: closing volume% VC; DLCO: carbon monoxide diffusion coefficient; EYD: eye irritation; $FEV_1$: forced expiratory volume in one second; FVC: forced vital capacity; IN: itchy nose; LAD: Lower Airway Discomfort; MCC: Mucociliary clearance; NAD: nasal discomfort; NAL: nasal lavage; PH: phlegm; RH: rhinitis; RN: runny nose; SOB: shortness breath; SN: sneezing; WR: work related; WH: wheeze.

change in nasal symptoms during work shift [63] or during work week [3] and exposure to wood dust.

Two studies have reported a significantly increased prevalence of **conjunctivitis** (5%) or **WR conjunctivitis** (3–26%), OR 2–∞ [53, 60] in wood workers. Two studies did not find increased prevalence of conjunctivitis [64] or WR conjunctivitis [9].

Five studies have reported increased prevalence's of eye irritation/discomfort (20–44%) with OR's from 2.0–4.5 when comparing exposed to non-exposed [35, 36, 37, 55, 68], significant in all but 2 studies [55, 68]. Increased prevalence of **WR eye irritation** was reported in 5 studies (14–41%) [9, 15, 29, 56, 58] with OR's of 1.9–∞, significant in [29, 56, 58].

**Objective measurements of nasal obstruction** and mucosal swelling have been performed in 6 studies in the dry wood industry [1, 3, 35, 36, 63, 73]. Schlünssen *et al.*, using **acoustic rhinometry**, revealed a DRR between current exposure level and increased mucosal swelling during work shift [63] while Åhman *et al.* showed **nasal PEF** deterioration during work week [1]. None of the studies including rhinomanometry have found any association to wood dust exposure [3, 35, 36], but Wilhelmsson *et al.* confirmed the sense of obstruction among 50 woodworkers [73].

Four papers have studied **nasal mucociliary clearance** (MCC) among wood workers, and all showed a higher percentage of woodworkers having reduced MCC compared to non-exposed controls [14, 35, 36] or during work week [3], although only significantly so in 2 studies [3, 14].

Furthermore, **nasal lavage fluid (NAL)** investigations have been carried out in 2 studies.

Åhman *et al.* found a DRR between number of classes per week and percentage neutrophils in NAL in exposed art teachers, but otherwise no significant differences in inflammatory markers compared to non-exposed [4]. Priha *et al.* investigated NAL pre- and post-shift, and found decreased TNF-α, an increased nitric oxide and increased protein content among exposed workers compared to controls. No difference in cell count was revealed [59].

In addition, sense of smell has been reported as being decreased in some studies among woodworkers [6, 35], and a few studies have objectively found a decrease in smell perception [3, 36, 39].

## DISCUSSION

When estimating respiratory health effects of occupational exposure to wood dust it is crucial to have valid exposure estimates. Wood dust exposure in the presented papers has been assessed in different ways. Some studies, mainly older ones, estimated exposure solely on employment status [14, 33] or parameters including years of exposure/employment [3, 6, 18, 53]. Most studies included dust measurements but mostly on a limited number of workers. Group exposure estimates have been based on additional

| Lower airway symptoms OR (95% CI) * OR calculated | Upper airway symptoms OR (95% CI) * OR calculated | Objective measurements | Confounders included |
|---|---|---|---|
| | **E *vs* C:**<br>BN: 1.5* (1.1–2.3)<br>RN: 1.8* (1.1–3.1) | **50 E:** Corr. mucosal swelling & nasal symps. (rhinoma-nometry), no corr. clearance & symptoms.<br>Clearance ↓54%.<br>**59 E:** ↓FVC compared to pred. values | Lung function: Smoking, age?, sex?, height? |
| | | **$E_{high}$ *vs* $E_{low}$:**<br>↓$FEV_1$/ FVC for both $E_{softwood}$ and $E_{hardwood}$<br>**E:** DRR cum. exp. and ↓$FEV_1$/ FVC NS ass exp. and ↓$FEV_1$ & ↓FVC | Smoking, sex, age, height |
| | | **E *vs* C:**<br>Nasal MCC:<br>8/9 *vs* 0/12 ($p<0.01$) | E more smokers, older |

information about work area, job title, etc. Some studies based exposure assessment on a substantial number of measurements [7, 15, 28, 42, 43, 51, 62, 64].

Misclassification of exposure in many of the studies is likely. When comparisons are made between groups of exposed wood workers this misclassification might attenuate the exposure-effect relation. There are large differences in exposure levels in the papers reviewed. Mean dust levels ranged from 0.4–9.0 mg/m$^3$ (AM or GM). In general, the older studies reported larger dust concentrations, while in the more recent studies a cross country gradient with higher dust concentrations in third world countries seems to exist. This is probably caused by considerable differences in production facilities and dust controlling systems, for example, ventilation and enclosure. Compared to exposure to fresh wood and mixed wood reviewed in [41], dry wood workers on average seem to be higher exposed, which is confirmed in the European wood dust exposure survey from 2006 [45].

All but 4 follow-up studies and one nested case control study are 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 bias the results, possibly by underestimating the true effect.

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 hospital staff, laboratory and office workers), making interpretation difficult [18, 35, 36, 37, 53, 55, 58, 59, 60].

Smoking is strongly causally related to the development of respiratory symptoms and decline in lung function, including COPD and chronic bronchitis, and therefore studies without information on smoking were excluded. The expected lung function depends on age, sex and height, and in general these factors have been taken into consideration in the included studies. Atopy is also a known risk factor for asthma and rhino-conjunctivitis, but only some studies have taken atopic status into consideration [3, 4, 42, 43, 55, 61, 62, 63, 64, 69].

Though only few studies revealed significant associations between exposure to wood dust 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 in the dry wood industry. The positive findings are confirmed in the few follow-up studies [34], at least for subgroups, i.e. females [42]. No clear pattern between exposure level and prevalence of asthma is seen across studies. As an example, Norrish *et al.* [55] found no DRR for asthma symptoms when comparing current low, medium and high exposed workers, while other studies on low exposed cohorts have revealed significantly increased prevalences of asthma symptoms, WR asthma symptoms, and in one study a DRR between dust exposure and clinical asthma [61, 64]. Very heterogeneous methodologies across a wide range of countries make it difficult directly to compare the different studies.

The studies reported in this review suggest that exposure to dry wood dust may cause CB. Most studies reported OR's above 1.0 when comparing woodworkers to controls, although only 2 cross-sectional studies report significant OR's above 2.0. Increased development of CB was also found in the only follow-up study dealing with CB, at least for females [42]. This is in accordance with findings from studies on fresh wood, where a review also suggest an association between wood dust exposure and CB, although the symptoms here might also be related to co-exposures of moulds and endotoxins [41].

Coughing is an unspecific symptom, which may reflect acute, benign irritation of the airways, as well as diseases like asthma, bronchitis or COPD. Coughing and WR coughing in relation to wood dust exposure seems to be a consistent finding across studies confirmed in the only

follow-up study dealing with coughing, at least for females [42]. In addition, studies revealing DRR between wood dust exposures and coughing support a causal effect [3, 42, 66].

An acute obstructive effect of wood dust exposure during workdays or during work weeks seems likely, as most studies measuring lung function have shown a post-shift decline in lung function. Also, several studies revealed DRR's between exposure and post-shift decline in $FEV_1$, strongly supporting an acute effect of wood dust on the lower airways.

When studying the effect of exposure on 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 ($FEV_1$, FVC, or $FEV_1$/FVC) among wood workers, and some studies revealed an association to current exposure or to years of exposure.

The 2 follow-up studies [28, 43] investigating trends in lung function show conflicting results, at least for females. Both studies were conducted on low exposed cohorts, where it is possible that effects on lung function may only be evident for the most susceptible groups, i.e. females, as reported by Jacobsen *et al.* Individual exposure assignments in both studies were based on JEMs and time of exposure and possible misclassifications of exposure were present, which would tend to underestimate the effect of exposure on lung function changes. In the study by Glindmeyer *et al.*, it is suggested that the follow-up cohort might represent a survivor group of workers without accelerated decline in lung function, as participants in the follow-up study in general had a longer employment status than non-participants.

There seem to be a consistent trend across studies on rhinitis, nasal symptoms, conjunctivitis, and eye irritation, supporting an effect of wood dust on nasal mucosa and conjunctiva. A few studies involving either nasal PEF or acoustic rhinometry have confirmed nasal obstruction in relation to wood dust exposure. In addition, studies on MCC have consistently revealed a reduced clearance among high exposed workers.

The mechanisms for wood dust inducing respiratory impairment are not 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 [13]. 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 [2, 20, 67, 74].

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 [10]. Wood dust extracts from both hard and soft wood are able to induce the release of pro-inflammatory mediators from macrophages [46, 48], express and induce the release of inflammatory mediators in human epithelial cell line [16], and modulate the expression of cytokines and chemokines [47].

In this review we focus on dry wood. Biohazards, mostly endotoxins and mould are mainly a concern when exposed to fresh wood [41], but exposure have been found at lower concentrations in the dry wood industry, where positive correlations between exposure to endotoxins and (1→3)-β-glucan and work-related bronchitis have been reported [9], and an association between cross shift decrease in lung function and exposure to endotoxins has been shown [50]. A recent study [32] of airborne endotoxins in joineries found endotoxins to be highly correlated to wood dust exposure, and it was concluded that endotoxins were only likely to be a problem when associated with very high dust levels in the dry wood industry.

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 to cause irritation of mucous membranes, and are suspected of causing impairment of lung function and BHR at levels of 100–450 mg/m$^3$ [8, 44]. Though terpenes are mainly found in fresh wood, lower levels have also been documented in the dry wood industry. In one study of joineries, terpene levels ranging from 9–214 mg/m$^3$ were revealed, together with chronic impairment in lung function among wood workers compared to controls, and this was mainly ascribed to terpenes [26]. Of the reviewed studies, Åhman *et al.* [5] found levels ranging from 0.02 –6.8 mg/m$^3$, but did not find respiratory symptoms associated to these low levels, while Jacobsen *et al.* reported GM (GSD) of 7.0 (2.8) mg/m$^3$, without reporting on health effects [42].

Processing of plywood and fibreboard may cause exposure to formaldehyde [49, 59] and asthma symptoms among woodworkers exposed to formaldehyde alone or in combination with wood dust have been documented [36]. A number of the reviewed papers included evaluations of the formaldehyde concentration [35, 36, 55, 59] and found formaldehyde levels ranging from 0.01–0.27 mg/m$^3$. A health- based recommended 8-hour time-weighted occupational exposure limit (OEL) of 0.15 mg/m$^3$ have been recommended in the Netherlands [25]. In the reviewed papers, it is in general not possible to distinguish the effects of wood dust and formaldehyde. However, in one recent study [27] at ply mills, an association between asthma symptoms and formaldehyde was revealed, with the highest level GM 0.16 mg/m$^3$. 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 and MDF.

Some studies indicate females to be more susceptible to wood dust exposure than men with regards to accelerated decline in lung function, bronchitis, coughing, and possi-

bly asthma [42, 43, 64]. Matheson *et al.* [52] found significant associations between exposure to biological dust and chronic bronchitis, and COPD in women, but not in men. Several population studies have reported a higher incidence of asthma among females [21, 70], and a greater susceptibility to develop lung function impairment in relation to tobacco exposure. The causes of females being more prone to develop asthma, respiratory symptoms, and impairment in lung function are not fully understood. Different explanations have been presented, e.g. airway size, hormonal factors, and social factors [21, 70]. A gender-related difference of the cough reflex has also been suggested, with women having a more sensitive cough reflex than men when tested with capsain. The proposed mechanism has been a higher sensitivity of the sensory receptors of the respiratory tract [23].

In conclusion, this review, despite the limitations in study design and exposure assessment, supports 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 like moulds, endotoxin and terpenes might contribute to the health effect, though these exposures are most likely to be present in the fresh wood industry. Formaldehyde may contribute to the health effects, especially in the plywood and MDF processing industry.

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## REFERENCES

1. Åhman M, Soderman E: Serial nasal peak expiratory flow measurements in woodwork teachers. *Int Arch Occup Environ Health* 1996, **68**, 177–182.

2. Åhman M, Hage-Hamsten M, Johansson SGO: IgE-mediated allergy to wood dusts probably doers not explain the high prevalence of respiratory symptoms among Swedish woodwork teachers. *Allergy* 1995, **50**, 559–562.

3. Åhman M, Holmstrom M, Cynkier I, Soderman E: Work related impairment of nasal function in Swedish woodwork teachers. *Occup Environ Med* 1996, **53**, 112–117.

4. Åhman M, Holmstrom M, Ingelman-Sundberg H: Inflammatory markers in nasal lavage fluid from Industrial Arts teachers. *Am J Ind Med* 1995, **28**, 541–550.

5. Åhman M, Persson L, Lagerstrand L, Soderman E, Cynkier I: Lung Function in Woodworking Teachers in Sweden. *Int J Occup Environ Health* 1996, **2**, 204–210.

6. Åhman M, Soderman E, Cynkier I, Kolmodin-Hedman B: Work-related respiratory problems in industrial arts teachers. *Int Arch Occup Environ Health* 1995, **67**, 111–118.

7. Al-Zuhair YS, Whitaker CJ, Cinkotai FF: Ventilatory function in workers exposed to tea and wood dust. *Br J Ind Med* 1981, **38**, 339–45.

8. Alexandersson R: A longitudinal study of respiratory health hazards of exposure to terpenes in saw-mills. *Arh Hig Rada Toksikol* 1988, **39**, 417–420.

9. Alwis KU, Mandryk J, Hocking AD: Exposure to Biohazards and wood dust: Bacteria, Fungi, Endotoxins, and (1→3) beta-D-Glucans. *Appl Occup Environ Hyg* 1999, **14**, 598–608.

10. Ayars GH, Altman LC, Frazier CA, Chi EY: The toxicity of constituents of cedar and pine woods to pulmonary epithelium. *J Allergy Clin Immunol* 1989, **83**, 610–618.

11. Baumgartner KB, Samet JM, Coultas DB, Stidley CA, Hunt WC, Colby TV, Waldron JA: Occupational and environmental risk factors for idiopathic pulmonary fibrosis: a multicenter case-control study. Collaborating Centers. *Am J Epidemiol* 2000, **152**, 307–315.

12. Beritic-Stahuljak D, Valic F, Cigula M, Ivankovic D, Prijatelj A: Aerosol analysis, respiratory function and skin sensitivity testing in workers exposed to soft and hardwood dust. *Arh Hig Rada Toksikol* 1988, **39**, 387–403.

13. Bernstein IL, Chan-Yeung M, Malo J-L, Bernstein DI: *Asthma in the Workplace, and Related Conditions*. 3rd ed. Taylor & Francis, New York 2006.

14. Black A, Evans JC, Hadfield EH, Macbeth RG, Morgan A, Walsh M: Impairment of nasal mucociliary clearance in woodworkers in the furniture industry. *Br J Ind Med* 1974, **31**, 10–17.

15. Bohadana AB, Massin N, Wild P, Toamain JP, Engel S, Goutet P: Symptoms, airway responsiveness, and exposure to dust in beech and oak wood workers. *Occup Environ Med* 2000, **57**, 268–273.

16. Bornholdt J, Saber AT, Sharma AK, Savolainen K, Vogel U, Wallin H: Inflammatory response and genotoxicity of seven wood dusts in the human epithelial cell line A549. *Mutat Res* 2007, **632**, 78–88.

17. British Medical Research Council: Definition and classification of chronic bronchitis for clinical and epidemiological purposes. A report to the Medical Research Council by their Committee on the Aetiology of Chronic Bronchitis. *Lancet* 1965, **1**, 775–779.

18. Carosso A, Ruffino C, Bugiani M: Respiratory diseases in wood workers. *Br J Ind Med* 1987, **44**, 53–56.

19. Carton M, Goldberg M, Luce D: [Occupational exposure to wood dust. Health effects and exposure limit values]. *Rev Epidemiol Sante Publique* 2002, **50**, 159–178.

20. Cormier Y, Merlaux A, Duchaine C: Respiratory Health Impact of Working in Sawmills in Eastern Canada. *Arch Environ Health* 2000, **55**, 424–430.

21. de Marco R, Locatelli F, Sunyer J, Burney P: Differences in incidence of reported asthma related to age in men and women. A retrospective analysis of the data of the European Respiratory Health Survey. *Am J Respir Crit Care Med* 2000, **162**, 68–74.

22. Demers PA, Teschke K, Kennedy SM: What to do about softwood? A review of respiratory effects and recommendations regarding exposure limits. *Am J Ind Med* 1997, **31**, 385–398.

23. Dicpinigaitis PV, Rauf K: The influence of gender on cough reflex sensitivity. *Chest* 1998, **113**, 1319–1321.

24. Douwes J, McLean D, Slater T, Pearce N: Asthma and Other Respiratory Symptoms in New Zealand Pine Processing Sawmill Workers. *Am J Ind Med* 2001, **39**, 608–615.

25. Dutch Expert Committee on Occupational Standards: *Formaldehyde: Health-based Recommended Occupational Exposure Limit.* Report No.: 2003/02OSH. Health Council of the Netherlands, Netherland 2003.

26. Eriksson KA, Levin JO, Sandstrom T, Lindstrom Espeling K, Linden G, Stjernberg NL: Terpene exposure and respiratory effects among workers in Swedish joinery shops. *Scand J Work Environ Health* 1997, **23**, 114–120.

27. Fransman W, McLean D, Douwes J, Demers PA, Leung V, Pearce N: Respiratory symptoms and occupational exposures in New Zealand plywood mill workers. *Ann Occup Hyg* 2003, **47**, 287–295.

28. Glindmeyer HW, Rando RJ, Lefante JJ, Freyder L, Brisolara JA, Jones RN: Longitudinal respiratory health study of the wood processing industry. *Am J Ind Med* 2008, **51**, 595–609.

29. Goldsmith DF, Shy CM: An epidemiologic study of respiratory health effects in a group of North Carolina furniture workers. *J Occup Med* 1988, **30**, 959–965.

30. Goldsmith DF, Shy CM: Respiratory health effects from occupational exposure to wood dusts. *Scand J Work Environ Health* 1988, **14**, 1–15.

31. Gustafson T, Dahlman-Hoglund A, Nilsson K, Strom K, Tornling G, Toren K: Occupational exposure and severe pulmonary fibrosis. *Respir Med* 2007, **101**, 2207–2212.

32. Harper M, Andrew ME: Airborne endotoxin in woodworking (joinery) shops. *J Environ Monit* 2006, **8**, 73–78.

33. Haxhiu MA, Djokic TD, Fehmiu E, Begraca M, Ukmata H: Occupational asthma in wood workers. *Arbete och Hälse* 1982, **19**, 35–44.

34. Heikkilä P, Martikainen R, Kurppa K, Husgafvel-Pursiainen K, Karjalainen A: Asthma incidence in wood-processing industries in Finland in a registerbased population study. *Scand J Work Environ Health* 2008, **34**, 66–72.

35. Holmstrom M, Rosen G, Wilhelmsson B: Symptoms, airway physiology and histology of workers exposed to medium-density fiber board. *Scand J Work Environ Health* 1991, **17**, 409–413.

36. Holmstrom M, Wilhelmsson B: Respiratory symptoms and pathophysiological effects of occupational exposure to formaldehyde and wood dust. *Scand J Work Environ Health* 1988, **14**, 306–311.

37. Holness DL, Sass-Kortsak AM, Pilger CW, Nethercott JR: Respiratory function and exposure-effect relationships in wood dust-exposed and control workers. *J Occup Med* 1985, **27**, 501–506.

38. Hubbard R, Lewis S, Richards K, Johnston I, Britton J: Occupational exposure to metal or wood dust and aetiology of cryptogenic fibrosing alveolitis [see comments]. *Lancet* 1996, **347**, 284–289.

39. Innocenti A, Valiani M, Vessio G, Tassini M, Giannelli M, Fusi S: Wood dust and nasal diseases: exposure to chestnut wood dust and loss of smell (pilot study). *Med Lav* 1985, **76**, 317–320.

40. International Agency for Research on Cancer: *Wood Dust and Formaldehyd.* IARC monographs on the evaluation of carcinogenic risks to humans. Vol. 62. IARC, Lyon 1995.

41. Jacobsen G, Schaumburg I, Sigsgaard T, Schlünssen V: Non-malignant respiratory diseases and occupational exposure to wood dust. Part I. Fresh wood and mixed wood industry. *Ann Agric Environ Med* 2010, **17**, 15–28.

42. Jacobsen G, Schlünssen V, Schaumburg I, Sigsgaard T: Increased incidence of respiratory symptoms among female woodworkers exposed to dry wood. *Eur Respir J* 2009, **33**, 1268–1276.

43. Jacobsen G, Schlünssen V, Schaumburg I, Taudorf E, Sigsgaard T: Longitudinal lung function decline and wood dust exposure in the furniture industry. *Eur Respir J* 2008, **31**, 334–342.

44. Kasanen JP, Pasanen AL, Pasanen P, Liesivuori J, Kosma VM, Alarie Y: Evaluation of sensory irritation of delta3-carene and turpentine, and acceptable levels of monoterpenes in occupational and indoor environment. *J Toxicol Environ Health A* 1999, **57**, 89–114.

45. Kauppinen T, Vincent R, Liukkonen T, Grzebyk M, Kauppinen A, Welling I, Arezes P, Black N, Bochmann F, Campelo F, Costa M, Elsigan G, Goerens R, Kikemenis A, Kromhout H, Miguel S, Mirabelli D, McEneany R, Pesch B, Plato N, Schlünssen V, Schulze J, Sonntag R, Verougstraete V, De Vicente MA, Wolf J, Zimmermann M, Husgafvel-Pursiainen K, Savolainen K: Occupational exposure to inhalable wood dust in the member states of the European Union. *Ann Occup Hyg* 2006, **50**, 549–561.

46. Long H, Shi T, Borm PJ, Määttä J, Husgafvel-Pursiainen K, Savolainen K, Krombach F: ROS-mediated TNF-alpha and MIP-2 gene expression in alveolar macrophages exposed to pine dust. *Part Fibre Toxicol* 2004, **1**, 3.

47. Maatta J, Luukkonen R, Husgafvel-Pursiainen K, Alenius H, Savolainen K: Comparison of hardwood and softwood dust-induced expression of cytokines and chemokines in mouse macrophage RAW 264.7 cells. *Toxicology* 2006, **218**, 13–21.

48. Määttä J, Majuri ML, Luukkonen R, Lauerma A, Husgafvel-Pursiainen K, Alenius H, Savolainen K: Characterization of oak and birch dust-induced expression of cytokines and chemokines in mouse macrophage RAW 264.7 cells. *Toxicology* 2005, **215**, 25–36.

49. Malmberg PO, Rask-Andersen A, Larsson KA, Stjernberg N, Sundblad BM, Eriksson K: Increased bronchial responsiveness in workers sawing Scots pine. *Am J Respir Crit Care Med* 1996, **153**, 948–952.

50. Mandryk J, Alwais KU, Hocking AD: Work-related symptoms and dose-response relationships for personal exposures and pulmonary function among woodworkers. *Am J Ind Med* 1999, **35**, 481–490.

51. Mandryk J, Alwis U, Hocking AD: Effects of personal exposures on pulmonary function and work-related symptoms among sawmill workers. *Ann Occup Hyg* 2000, **44**, 281–289.

52. Matheson MC, Benke G, Raven J, Sim MR, Kromhout H, Vermeulen R, Johns DP, Walters EH, Abramson MJ: Biological dust exposure in the workplace is a risk factor for chronic obstructive pulmonary disease. *Thorax* 2005, **60**, 645–651.

53. Milanowski J, Góra A, Skórska C, Krysińska-Traczyk E, Mackiewicz B, Sitkowska J, Cholewa G, Dutkiewicz J: Work-related symptoms among furniture factory workers in Lublin region (eastern Poland). *Ann Agric Environ Med* 2002, **9**, 99–103.

54. Noertjojo HK, Dimich-Ward H, Peelen S, Dittrick M, Kennedy SM, Chan-Yeung M: Western red cedar dust exposure and lung function: a dose-response relationship. *Am J Respir Crit Care Med* 1996, **154**, 968–973.

55. Norrish AE, Beasley R, Hodgkinson EJ, Pearce N: A study of New Zealand wood workers: exposure to wood dust, respiratory symptoms, and suspected cases of occupational asthma. *N Z Med J* 1992, **105**, 185–187.

56. Osman E, Pala K: Occupational exposure to wood dust and health effects on the respiratory system in a minor industrial estate in Bursa/Turkey. *Int J Occup Med Environ Health* 2009, **22**, 43–50.

57. Pinheiro GA, Antao VC, Wood JM, Wassell JT: Occupational risks for idiopathic pulmonary fibrosis mortality in the United States. *Int J Occup Environ Health* 2008, **14**, 117–123.

58. Pisaniello DL, Connell KE, Muriale L: Wood dust exposure during furniture manufacture – results from an Australian survey and considerations for threshold limit value development. *Am Ind Hyg Assoc J* 1991, **52**, 485–492.

59. Priha E, Pennanen S, Rantio T, Uitti J, Liesivuori J: Exposure to and acute effects of medium-density fiber board dust. *J Occup Environ Hyg* 2004, **1**, 738–744.

60. Rongo LM, Besselink A, Douwes J, Barten F, Msamanga GI, Dolmans WM, Demers PA, Heederik D: Respiratory symptoms and dust exposure among male workers in small-scale wood industries in Tanzania. *J Occup Environ Med* 2002, **44**, 1153–1160.

61. Schlünssen V, Schaumburg I, Heederik D, Taudorf E, Sigsgaard T: Indices of asthma among atopic and non-atopic woodworkers. *Occup Environ Med* 2004, **61**, 504–511.

62. Schlünssen V, Sigsgaard T, Schaumburg I, Kromhout H: Cross-shift changes in $FEV_1$ in relation to wood dust exposure: the implications of different exposure assessment methods. *Occup Environ Med* 2004, **61**, 824–830.

63. Schlünssen V, Schaumburg I, Andersen NT, Sigsgaard T, Pedersen OF: Nasal Patency is related to dust exposure in woodworkers. *Occup Environ Med* 2002, **59**, 23–29.

64. Schlünssen V, Schaumburg I, Taudorf E, Mikkelsen AB, Sigsgaard T: Respiratory symptoms and lung function among Danish woodworkers. *J Occup Environ Med* 2002, **44**, 82–98.

65. Scott J, Johnston I, Britton J: What causes cryptogenic fibrosing alveolitis? A case-control study of environmental exposure to dust. *Br Med J* 1990, **301**, 1015–107.

66. Shamssain MH: Pulmonary function and symptoms in workers exposed to wood dust. *Thorax* 1992, **47**, 84–87.

67. Skovsted TA, Schlünssen V, Schaumburg I, Wang P, Staun-Olsen P, Skov PS: Only few workers exposed to wood dust are detected with specific IgE against pine wood. *Allergy* 2003, **58**, 772–779.

68. Sripaiboonkij P, Phanprasit W, Jaakkola MS: Respiratory and skin effects of exposure to wood dust from the rubber tree Hevea brasiliensis. *Occup Environ Med* 2009, **66**, 442–447.

69. Talini D, Monteverdi A, Benvenuti A, Petrozzino M, Di Pede F, Lemmi M, Carletti A, Macchioni P, Serretti N, Viegi G, Paggiaro P: Asthma-like symptoms, atopy, and bronchial responsiveness in furniture workers. *Occup Environ Med* 1998, **55**, 786–791.

70. Torén K, Gislason T, Omenaas E, Jögi R, Forsberg B, Nyström L, Olin AC, Svanes C, Janson C; RHINE Group: A prospective study of asthma incidence and its predictors: the RHINE study. *Eur Respir J* 2004, **24**, 942–946.

71. Whitehead LW: Health effects of wood dust--relevance for an occupational standard. *Am Ind Hyg Assoc J* 1982, **43**, 674–8.

72. Whitehead LW, Ashikaga T, Vacek P: Pulmonary function status of workers exposed to hardwood or pine dust. *Am Ind Hyg Assoc J* 1981, **42**, 178–186.

73. Wilhelmsson B, Drettner B: Nasal problems in wood furniture workers. A study of symptoms and physiological variables. *Acta Otolaryngol Stockh* 1984, **98**, 548–555.

74. Wilhelmsson B, Jernudd Y, Ripe E, Holmberg K: Nasal hypersensitivity in wood furniture workers. An allergological and immunological investigation with special reference to mould and wood. *Allergy* 1984, **39**, 586–595.