

Investigating nasal cytology as a potential tool for diagnosing occupational rhinitis in woodworkers

Claudia Staffieri, MD, PhD¹, Andrea Lovato, MD², Federica Aielli, MD¹, Martina Bortoletto, MD³, Luciano Giacomelli, MD⁴, Mariella Carrieri, MD³, Salvatore Romeo, MD, PhD⁵, Paolo Boscolo-Rizzo, MD¹, Maria Cristina Da Mosto, MD¹, Giovanni Battista Bartolucci, MD³, Gino Marioni, MD² and Maria Luisa Scapellato, MD³

Background: Our primary endpoint was to use nasal cytology to compare woodworkers with unexposed subjects to see if wood dust exposure correlates with specific patterns of inflammatory or infectious rhinitis. A secondary endpoint was to identify any differences in the exposed group's nasal symptoms or nasal cytology by years of exposure or personal exposure levels.

Methods: Ninety-two woodworkers and 90 controls were assessed using a questionnaire and nasal cytology (on nasal mucosa obtained by scraping). Wood dust exposure was investigated using personal sampling methods.

Results: Woodworkers reported significantly more nasal symptoms than controls ($p < 0.00001$). The woodworkers' nasal smears revealed more neutrophils ($p = 0.001$) and significantly higher mean neutrophil scores ($p = 0.001$) than control smears. Lymphocytes were also found more often in the woodworkers' rhinocytograms (statistical trend, $p = 0.06$). Neutrophilic rhinitis was diagnosed more frequently in the exposed workers than in controls (chi-square = 5.97,

$p < 0.05$). Woodworkers with lymphocytes in their nasal smears had been exposed to wood dust for longer periods of time (statistical trend; $p = 0.06$). No differences in nasal symptoms or cell counts emerged when woodworkers were stratified by levels of personal exposure.

Conclusion: Nasal cytology should be further investigated in woodworkers before considering it a screening method for identifying woodworkers with chronic inflammatory rhinitis. © 2015 ARS-AAOA, LLC.

Key Words:

wood dust; woodworkers; occupational rhinitis; nasal cytology; personal exposure

How to Cite this Article:

Staffieri C, Lovato A, Aielli F, et al. Investigating nasal cytology as a potential tool for diagnosing occupational rhinitis in woodworkers. *Int Forum Allergy Rhinol.* 2015;5:814-819.

¹Department of Neurosciences, Otolaryngology Section, Treviso Regional Hospital Branch, University of Padova, Treviso, Italy;

²Department of Neurosciences, Otolaryngology Section, University of Padova, Padova, Italy; ³Department of Cardiology, Thoracic and Vascular Sciences, Preventive Medicine and Risk Assessment Unit, University of Padova, Padova, Italy; ⁴Department of Medicine, University of Padova, Padova, Italy; ⁵Department of Pathology, Treviso Regional Hospital, Treviso, Italy

Correspondence to: Claudia Staffieri, MD, PhD, Department of Neurosciences, Otolaryngology Section, Treviso Branch, Piazzale Ospedale 1, Treviso, Italy; e-mail: c.staffieri@alice.it

C.S. and A.L. contributed equally to the manuscript's preparation. Funding sources for the study: University of Padova (grant 60A07-8485/13 to G.M.); Italian Workers' Compensation Authority (INAIL) (grant 15/2009 to G.B.B.).

Potential conflict of interest: None provided.

Received: 21 February 2015; Revised: 11 April 2015; Accepted: 2 May 2015

DOI: 10.1002/alr.21562

View this article online at wileyonlinelibrary.com.

Exposure to wood dust is widespread. It has been estimated that more than 3 million employees in the European Union (EU) are occupationally exposed to wood dust.^{1,2} Wood dust is thought to be a mucous membrane irritant. Although no clear distinction can be drawn between the health risks relating to different species, hardwoods seem to pose a greater health risk than softwoods,³ because occupational exposure to hardwood dust is strongly associated with sinonasal adenocarcinomas. The International Agency for Research on Cancer (IARC) has classed wood dust as carcinogenic for humans.^{4,5} The EU's Scientific Committee for Occupational Exposure Limits (SCOEL) has also stated that exposure to wood dust above 0.5 mg/m³ induces pulmonary effects.⁶ Two quite recent reviews identified a strong association between fresh and dry wood dust exposure and asthma, asthma symptoms, coughing, bronchitis, and acute and chronic lung function impairment.^{3,7}

Exposure to wood dust has also been associated with nasal symptoms (primarily nasal obstruction) in several

studies^{8,9} and wood dust exposure above 1 mg/m³ has been found to coincide with an exposure-effect relationship.⁶ Objective nasal patency measurements obtained using rhinomanometry,¹⁰ nasal peak expiratory flow,¹¹ and acoustic rhinometry¹² have confirmed nasal obstruction in woodworkers. Another nasal function found impaired in woodworkers is nasal mucociliary clearance, which was investigated by Ahman et al.¹³

Respiratory effects induced by wood dust are assumed to involve roughly the same pathophysiological mechanisms as for other organic dusts (such as grain dust and cotton dust), which involve the release of inflammatory mediators by inflammatory and epithelial cells.¹⁴ This assumption is partly sustained by the fact that a significantly higher rate of rhinitis was recorded among workers exposed to wood dust than in unexposed controls.¹⁵ One way to further confirm this hypothesis would be to investigate inflammatory markers (eg, nasal cellularity, neutrophils) in nasal mucosa. A few studies have already been conducted on nasal lavage fluid, with contradictory results.^{14,16} To our knowledge, no other English-language publications have reported on woodworkers assessed using nasal cytology on samples obtained by nasal mucosa scraping.

The primary endpoint of the present investigation was to use nasal cytology to compare a group of woodworkers and a group of unexposed subjects to see whether wood dust exposure correlates with specific patterns of inflammatory or infectious rhinitis. A secondary endpoint was to seek any differences in the nasal symptoms or nasal cytology in the exposed individuals by years of exposure, or personal exposure levels.

Subjects and methods

Subjects

The woodworkers who volunteered for the present study were enrolled during an industrial environment investigation conducted by the Padova University Preventive Medicine and Risk Assessment Service on woodworking factories in the Veneto region (Italy). The study was approved by our institutional research committee. Informed consent was obtained from all individuals taking part in the study.

The inclusion criteria were as follows: (1) male gender; (2) involvement in woodworking processes that entailed exposure to wood dust; (3) wood dust exposure persisting for at least 12 months; (4) a negative medical history for nasal surgery or rhinitis; and (5) anterior rhinoscopy ruling out nasal polyps and obstructive septal deviations. Controls were volunteers who had no history of occupational or other exposure to wood dust or other nasal irritants. Ninety-two woodworkers and 90 controls met the above inclusion criteria and were enrolled in the present study.

Both groups were administered a self-reporting questionnaire investigating nasal symptoms (nasal congestion, rhinorrhea, sneezing fits, nasal itching, anosmia/hyposmia,

epistaxis, ageusia/hypogeusia, snoring, nocturnal oral breathing, cough, headache, and sore throat) in the previous 6 months, and cigarette smoking history. We considered nasal congestion, rhinorrhea, sneezing fits, nasal itching, and anosmia/hyposmia as major symptoms. The group of woodworkers also answered questions on their years of exposure to wood dust and the wood species they worked with (hardwood, softwood, or tropical hardwood).

All procedures performed in the study were in accordance with the ethical standards of our institutional research committee and with the 1964 Helsinki Declaration and its later amendments. Informed consent was obtained from all individual participants included in the study.

Nasal cytology

Nasal cytology was performed by anterior rhinoscopy, using a nasal speculum. The collection technique consisted of scraping mucosa from the middle portion of the inferior turbinate, using a Rhino-Probe (Arlington Scientific, Springville, UT).¹⁷ The sample was placed on a glass slide, fixed by air drying and then stained using the May-Grunwald Giemsa (MGG) method (Carlo Erba, Milan, Italy). This is the method most widely used in diagnostic nasal cytology because all of the cellular components of the nasal mucosa, from inflammatory cells (neutrophils, eosinophils, mast cells, lymphocytes) to bacteria, spores, fungal hyphae, and mucous secretions, are easily stained. The slide was examined under a Nikon E600 light microscope (Nikon, Mississauga, Ontario, Canada). In each rhinocytogram, 50 microscopic fields were analyzed at $\times 1000$ magnification to ascertain the presence of normal and abnormal cellular elements. Cell counts, and bacterial and fungal analyses were scored by means of a semiquantitative grading method proposed by Meltzer and Jalowayski,¹⁸ as follows: grade 0 (not visible); grade 1 (occasional groups); grade 2 (moderate number); grade 3 (easily visible); and grade 4 (many of which cover the entire field of view). Depending on which cells were predominant,¹⁹ we classified pathological rhinocytograms as follows: (1) eosinophilic rhinitis (eosinophils grading 1 to 4, neutrophils may be present); (2) mast cell rhinitis (mast cells grading 1 to 4, neutrophils may be present); (3) neutrophilic rhinitis (neutrophils grading 1 to 4); (4) bacterial rhinitis (bacterial grading 1 to 4, and neutrophils grading 1 to 4); and (5) fungal rhinitis (fungal grading 1 to 4, and neutrophils grading 1 to 4).

Personal exposure

Personal exposure to inhalable wood dust was assessed using an Institute of Occupational Medicine (IOM) sampler equipped with polyvinyl chloride (PVC) membranes (diameter 25 mm, pore diameter 1.2 μ m) and connected to an air sampling pump (PCXR4-model; SKC Inc., Eighty Four, PA) (Fig. 1) operating at a flow rate of 2 L/minute. Sampling was done during a full 8-hour working day. The PVC membranes were conditioned in the Aquaria Climatic hood



FIGURE 1. Woodworker wearing the IOM sampler connected to the air sampling pump. IOM = Institute of Occupational Medicine.

(Aquaria, Milan, Italy) at $20^{\circ}\text{C} \pm 1^{\circ}\text{C}$ and $50\% \pm 5\%$ relative humidity for 48 hours before and after the sampling period. Dusts were measured gravimetrically on an MC-5 microscale with a detection threshold of 0.001 mg (Sartorius Mechatronics, Milan, Italy).

Statistical analysis

Given the purposes of the investigation, we conducted an intergroup analysis to compare the exposed and unexposed subjects, and an intragroup analysis on the woodworkers. Most of the variables were dichotomized: smoking (no vs yes), symptoms (absent vs present), major symptoms (absent vs present), and personal exposure level ($\leq 1 \text{ mg/m}^3$ vs $> 1 \text{ mg/m}^3$). Furthermore, to avoid any bias due to post hoc considerations, we preliminarily decided to dichotomize also rhinocytograms (normal vs altered) as follows: neutrophil grading (0 vs 1 to 4), eosinophil grading (0 vs 1 to 4), mast cell grading (0 vs 1 to 4), lymphocyte grading (0 vs 1 to 4), bacterial grading (0 vs 1 to 4), and fungal grading (0 vs 1 to 4). Fisher's exact test was used to compare the dichotomized variables. When a significant p value was disclosed, the odds ratio (OR) and the corresponding 95% confidence interval (CI) were calculated.

The Mann-Whitney test was also used to compare the mean neutrophil scores. The t test corrected for unequal variances was used to analyze continuous variables (age, years of exposure). The chi-square test was used to analyze probability distributions.

A p value < 0.05 was considered significant, whereas values in the range of $0.07 \geq p \geq 0.05$ were assumed to indicate a statistical trend. The STATATM 8.1 (Stata Corp, College Station, TX) statistical package was used for all analyses.

Results

Intergroup analyses (woodworkers vs controls)

In the present study, 92 woodworkers and 90 controls met our inclusion criteria and were enrolled. The mean age of the 2 groups was 39.5 years for the woodworkers (standard deviation [SD] 11.4 years) and 35.2 years for the controls (SD 13.4 years); it differed significantly between the 2 groups (t test corrected for unequal variances; $p = 0.018$). Twenty-five woodworkers and 27 controls reported a history of cigarette smoking (Fisher's exact test; $p = 0.74$). Nasal symptoms and major symptoms were reported by 57 (62%) and 27 (29%) woodworkers, and by 37 (41%) and 22 (24%) controls, respectively. Fisher's exact test disclosed a significant difference between the 2 groups in the distribution of nasal symptoms ($p < 0.00001$; OR = 3.8; 95% CI, 1.96 to 7.39), and major symptoms ($p = 0.017$; OR = 2.07; 95% CI, 1.05 to 4.14).

The cell counts on nasal cytology (Fig. 2), and the presence of bacteria and fungi in the woodworkers and controls are shown in Table 1. The rhinocytograms were pathological for 58 woodworkers and 43 controls, with a trend toward a significant difference (Fisher's exact test; $p = 0.05$; OR = 1.86; 95% CI, 0.98 to 3.52). Neutrophils were seen more frequently in the woodworkers' nasal smears (Fisher's exact test; $p = 0.001$; OR = 2.86; 95% CI, 1.46 to 5.65) and this group's mean neutrophil score was significantly higher (Mann-Whitney test; $p = 0.001$) than in the control group. Lymphocytes were also more common in the woodworkers' rhinocytograms (Fisher's exact test; statistical trend, $p = 0.06$). No other significant differences emerged between the 2 groups in terms of the other cell counts (Fisher's exact test; eosinophils, $p = 0.12$; mast cells, $p = 0.59$), or the presence of bacteria (Fisher's exact test; $p = 0.31$), or fungi (Fisher's exact test; $p = 0.3$). A neutrophilic rhinitis was diagnosed more frequently among the woodworkers (40%) than in the control group (23%) (chi-square = 5.97, $p < 0.05$). Eosinophilic rhinitis was diagnosed in 2 woodworkers and 1 control, and mast cell rhinitis in 1 woodworker and 2 controls.

Intragroup analyses (woodworkers)

There were no significant differences in their nasal symptoms when woodworkers were stratified by cell counts (Fisher's exact test; neutrophils, $p = 0.46$; lymphocytes, $p = 0.39$; eosinophils, $p = 1.00$; mast cells, $p = 0.47$), or presence of bacteria or fungi (Fisher's exact test; $p = 0.78$ and $p = 1.00$, respectively).

Woodworkers who were cigarette smokers did not report nasal symptoms (Fisher's exact test; $p = 0.33$) or major symptoms (Fisher's exact test; $p = 0.44$) more frequently than nonsmokers, nor did they have pathological rhinocytograms more frequently (Fisher's exact test; $p = 0.55$).

The mean period of wood dust exposure was 14.7 ± 12.3 years. The t test corrected for unequal variances disclosed a trend toward a significantly longer period of exposure when lymphocytes were identified in woodworkers'

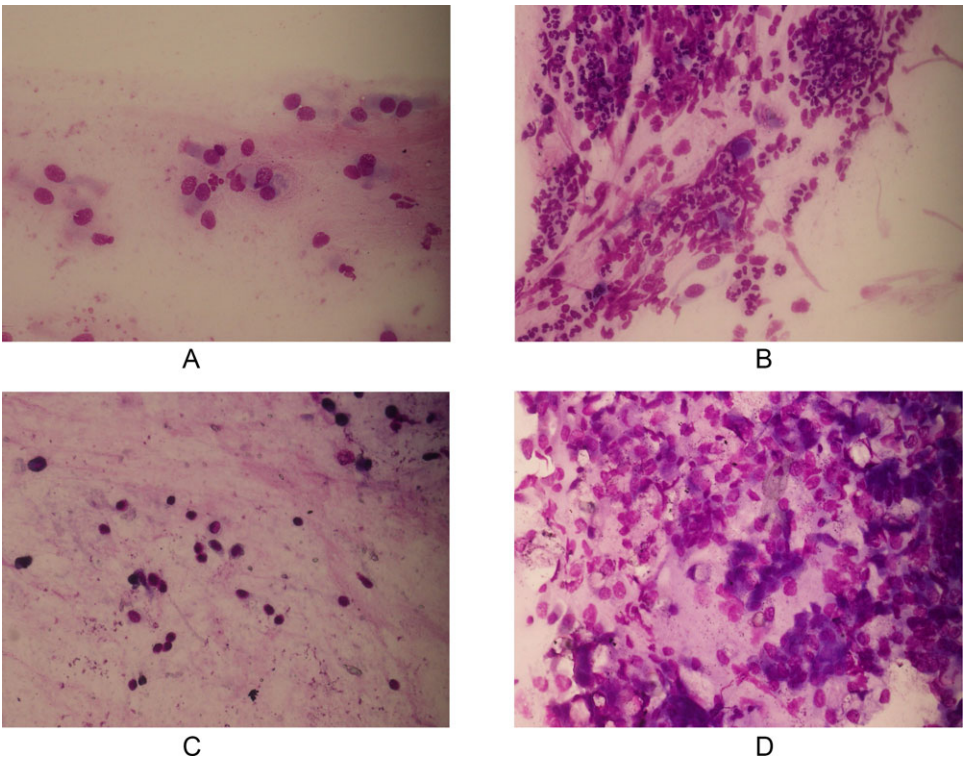


FIGURE 2. MGG staining showing (A) normal rhinocytogram; (B) neutrophils and lymphocytes; (C) eosinophils; and (D) bacteria and goblet cells. Original magnification $\times 400$. MGG = May-Grunwald Giemsa.

TABLE 1. Participants found positive on nasal cytology among 92 WWs and 90 controls

	Neutrophils		Lymphocytes		Eosinophils		Mast cells		Bacteria		Fungi	
Grades ^a	WWs	Controls	WWs	Controls	WWs	Controls	WWs	Controls	WWs	Controls	WWs	Controls
0	23	44	77	84	84	87	83	84	74	78	90	85
1–4	69	56	15	6	8	3	9	6	18	12	2	5

^aGrades were assigned by means of a semiquantitative grading method proposed by Meltzer and Jalowayski¹⁸ as follows: grade 0 (not visible); grade 1 (occasional groups); grade 2 (moderate number); grade 3 (easily visible); and grade 4 (many of which cover the entire field of view). WW = woodworker.

rhinocytograms ($p = 0.06$). There were no other significant differences relating to the mean period of exposure when woodworkers were stratified by the other cell counts (t test corrected for unequal variances; neutrophils, $p = 0.62$; eosinophils, $p = 0.63$; mast cells, $p = 0.26$), by the presence of bacteria or fungi (t test corrected for unequal variances; $p = 0.58$ and $p = 0.61$, respectively), or by nasal symptoms (Fisher's exact test; $p = 0.77$).

Personal exposure levels were $\leq 1 \text{ mg/m}^3$ in 68 woodworkers and $>1.0 \text{ mg/m}^3$ in 24. None had levels of exposure to wood dust in excess of 5.0 mg/m^3 . There were no significant differences in personal exposure levels when woodworkers were assessed according to their nasal symptoms (Fisher's exact test; $p = 0.12$), cell counts (Fisher's exact test; neutrophils, $p = 0.11$; lymphocytes, $p = 0.33$; eosinophils, $p = 0.67$; mast cells, $p = 1.00$), or presence of bacteria or fungi (Fisher's exact test; $p = 0.13$ and $p = 1.00$, respectively).

In the present investigation, 32 woodworkers (34%) reported using only 1 species of wood (softwood in 7 cases, hardwood in 25), whereas the other 60 (66%) used more than 1 species. No significant differences in nasal symptoms, cell counts, or presence of bacteria or fungi emerged between these 2 subgroups of woodworkers.

Discussion

Occupational exposure to wood dust may cause various diseases, the most common being chronic bronchitis and nonasthmatic chronic airflow obstruction. The incidence of these conditions seems to vary with the type of wood, the manner in which it is handled, and climatic conditions.²⁰ Nasal complaints have frequently been reported in occupational groups exposed to wood dust, which appears to modify nasal physiology in terms of mucociliary activity and airflow resistance on rhinomanometry.²¹ Evidence

from nasal lavage fluid analyses in woodworkers has suggested that wood dust may act as a mucous membrane irritant, causing a chronic inflammatory response.²²

The primary endpoint of the present investigation was to use nasal cytology to compare a group of woodworkers with a group of controls to see if wood dust exposure correlated with specific patterns of inflammatory or infectious rhinitis. Nasal cytology on nasal mucosa obtained by scraping is a low-cost technique: slides can easily be stained with MGG and examined under the light microscope in an outpatient setting. The main strengths of our study lie in the numerosity and homogeneity of the series considered (all males with no medical history of rhinitis or nasal surgery, who underwent personal sampling of inhalable wood dust). The main weakness relates to the cross-sectional design that is not ideal for studying chronic wood dust exposure.

The woodworkers in our sample reported significantly more nasal symptoms (62%) than controls (41%). The distribution of symptoms found in our study is comparable with other reports: approximately 50% of workers in wooden furniture industries complain of nasal discomfort.^{9,10}

Considered woodworkers also had pathological rhinocytograms (see definition in Subjects and Methods) significantly more frequently than controls. Even though this difference indicated a trend toward a significant difference, it has to be noted that a considerable part of our controls (48%) showed an altered rhinocytogram. On the other hand, a neutrophilic rhinitis was diagnosed definitely more often in the woodworkers than in the control group. The mean neutrophil score was significantly higher in the nasal smears of woodworkers than in controls, and the woodworkers' lymphocyte score was higher too, though the difference did not reach statistical significance. These findings are consistent with a picture of chronic inflammatory rhinitis disclosed by nasal cytology in woodworkers. In agreement with our results, Holness et al.²³ found more inflammatory cells in cabinetmakers' nasal smears than in those of control workers. In an article published in Italian, Porro et al.²⁴ also investigated cabinetmakers' nasal cytology (on nasal mucosa obtained by scraping), but found no significant differences in the cytograms of the exposed and unexposed subjects. Other studies on the nasal cellularity of woodworkers analyzed nasal lavage fluids: (1) Ahman et al.¹⁶ studied Arts teachers exposed to wood dust and found that their neutrophil counts did not differ significantly from those of controls; (2) Priha et al.²⁵ studied the nasal lavage obtained from woodworkers before and after a working shift, and found no differences in their cell counts. Such contradictory results may be due to the different sampling methods used (we scraped nasal mucosa from the inferior turbinate, not nasal lavage). It is also worth mentioning that a marked variability in nasal lavage parameters has been reported in repeated collections.²⁶ We opted for nasal scraping rather than nasal lavage because we collected samples at the workplace and samples are easier to handle.²⁷

Our secondary endpoint was to identify any differences in our woodworkers' nasal symptoms or nasal cytology by period of exposure (in years) or personal exposure level. Most (66%) of the woodworkers handled more than 1 species of wood. Woodworkers with lymphocytes in their nasal smears had been exposed to wood dust for longer periods (with a trend toward a significant difference): this finding suggests that wood dust may act as a chronic irritant in the nasal mucosa.

In our study, personal sampling of the inhalable fractions of wood dust disclosed no cases in which values exceeded the limit of 5 mg/m³ imposed by European and Italian legislation. The European Directive 2004/37 (European Union²⁸) establishing this exposure limit of 5 mg/m³ for hardwood and mixed wood dust emphasizes that it is the inhalable fraction that must be sampled. On the other hand, 26% of our woodworkers exposed had personal levels of exposure to wood dust beyond the limit of 1 mg/m³ permitted by the American Conference of Governmental Industrial Hygienists (ACGIH)²⁹ and the National Institute of Occupational Safety and Health (NIOSH). We found no differences in nasal symptoms when woodworkers were stratified by levels of personal exposure. This is consistent with the reports from most authors who found that subjects exposed to wood dust had more nasal complaints than unexposed controls, but the more heavily exposed of the former did not have more severe nasal complaints than the less heavily exposed.^{3,7} Ahman et al.¹³ found that nasal symptoms correlated weakly with the percentage of inhalable dust in a series of 39 woodwork teachers. Schlünssen et al.¹² reported a significantly more severe self-rated nasal obstruction in their 2 most heavily exposed subcohorts among a series of 161 woodworkers.

In the present series, we found no significant differences in the cell counts when the woodworkers were stratified by level of exposure to wood dust. Similar results were reported by Borm et al.,¹⁴ who assessed the nasal lavage of 930 woodworkers and found no significant differences in nasal cellularity between the exposure classes identified. This poor correlation with exposure levels may be due to a selection bias intrinsic in cross-sectional studies.⁸ Clearly, a personal sampling procedure lasting one 8-hour working day may not be representative of the wood dust levels experienced previously. Exposure may vary significantly day by day, also depending on the type of wood, the manner in which it is handled, and the climatic conditions at the time.

Conclusion

A significant proportion of the woodworkers considered in the present investigation revealed a chronic form of inflammatory rhinitis on nasal cytology, with higher neutrophil and lymphocyte counts than controls. However, an important part of our controls (48%) showed altered rhinocytograms according to our preliminary definitions. Nasal cytology should be further investigated in

woodworkers before considering it as a routine screening method for identifying woodworkers with chronic inflammatory rhinitis. 

Acknowledgement

We thank Frances Coburn for correcting the English version of the manuscript.

References

- Kauppinen T, Vincent R, Liukkonen T, et al. Occupational exposure to inhalable wood dust in the member states of the European Union. *Ann Occup Hyg*. 2006;50:549–561.
- Kauffer E, Wrobel R, Görner P, et al. Site comparison of selected aerosol samplers in the wood industry. *Ann Occup Hyg*. 2010;54:188–203.
- Jacobsen G, Schaumburg I, Sigsgaard T, Schlunssen 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.
- International Agency for Research on Cancer (IARC). *Wood Dust and Formaldehyde*. Vol 62. Lyon, France: IARC; 1995. IARC Monographs.
- International Agency for Research on Cancer (IARC). *A Review of Human Carcinogens: Arsenic, Metals, Fibres, and Dusts*. Vol 100C. Lyon, France: IARC; 2012. IARC Monographs.
- Scientific Committee on Occupational Exposure Limits (SCOEL). European Commission. Employment, Social Affairs & Inclusion. Recommendation from the Scientific Committee on Occupational Exposure Limits: Risk Assessment for Wood Dust. SCOEL/SUM/102 Final. December 2003. <http://ec.europa.eu/social/BlobServlet?docId=3876&langId=en>. Accessed May 24, 2015.
- Jacobsen G, Schaumburg I, Sigsgaard T, Schlunssen V. Non-malignant respiratory diseases and occupational exposure to wood dust. Part II. Dry wood industry. *Ann Agric Environ Med*. 2010;17:29–44.
- 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.
- Holmström M, Rosén G, Wählander L. Effect of nasal lavage on nasal symptoms and physiology in wood industry workers. *Rhinology*. 1997;35:108–112.
- Wilhelmsson B, Drettner B. Nasal problems in wood furniture workers. A study of symptoms and physiological variables. *Acta Otolaryngol*. 1984;98:548–555.
- Ahman M, Söderman E. Serial nasal peak expiratory flow measurements in woodwork teachers. *Int Arch Occup Environ Health*. 1996;68:177–182.
- Schlunssen V, Schaumburg I, Andersen NT, et al. Nasal patency is related to dust exposure in woodworkers. *Occup Environ Med*. 2002;59:23–29.
- Ahman M, Holmström M, Cynkier I, Söderman E. Work-related impairment of nasal function in Swedish woodwork teachers. *Occup Environ Med*. 1996;53:112–117.
- Borm PJ, Jetten M, Hidayat S, et al. Respiratory symptoms, lung function, and nasal cellularity in Indonesian wood workers: a dose-response analysis. *Occup Environ Med*. 2002;59:338–344.
- Rongo LM, Besselink A, Douwes J, et al. Respiratory symptoms and dust exposure among male workers in small-scale wood industries in Tanzania. *J Occup Environ Med*. 2002;44:1153–1160.
- Ahman M, Holmström M, Ingelman-Sundberg H. Inflammatory markers in nasal lavage fluid from Industrial Arts teachers. *Am J Ind Med*. 1995;28:541–550.
- Gelardi M, Passalacqua G, Fiorella ML, et al. Nasal cytology: the “infectious spot”, an expression of a morphological-chromatic biofilm. *Eur J Clin Microbiol Infect Dis*. 2011;30:1105–1109.
- Meltzer EO, Jalowayski AA. Nasal cytology in clinical practice. *Am J Rhinol*. 1988;2:47–54.
- Meltzer EO, Orgel Ha, Jalowayski AA. Nasal cytology. In: Naclerio R, Durham SR, Mygind N, eds. *Rhinitis: Mechanisms and Management*. New York: Marcel Dekker Inc.; 1999:175–202.
- Enarson DA, Chan-Yeung M. Characterization of health effects of wood dust exposures. *Am J Ind Med*. 1990;17:33–38.
- Holmström M, Rosén 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.
- Granstrand P, Nylander-French L, Holmström M. Biomarkers of nasal inflammation in wood-surface coating industry workers. *Am J Ind Med*. 1998;33:392–399.
- 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.
- Porro S, Mascagni P, Ferraioli E, Toffoletto F. [Health surveillance on workers exposed to wood dust: usefulness of the assessment of the nasal mucosa]. *G Ital Med Lav Ergon*. 2007;29:248–250. Italian.
- Priha E, Pennanen S, Rantio T, et al. Exposure to and acute effects of medium-density fiber board dust. *J Occup Environ Hyg*. 2004;1:738–744.
- Keman S, Jetten M, Douwes J, Borm PJ. Longitudinal changes in inflammatory markers in nasal lavage of cotton workers. Relation to endotoxin exposure and lung function changes. *Int Arch Occup Environ Health*. 1998;71:131–137.
- Howarth PH, Persson CG, Meltzer EO, et al. Objective monitoring of nasal airway inflammation in rhinitis. *J Allergy Clin Immunol*. 2005;115:414–441.
- European Union. Council Directive 2004/37/EC of 29 April 2004 on the protection of workers from the risk related to exposure to carcinogens or mutagens at work. Official Journal of the European Community. <http://eurlex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2004:229:0023:0034:EN:PDF>. Accessed May 24, 2015.
- American Conference of Governmental Industrial Hygienists (ACGIH). *Documentation of the Threshold Limit Values and Biological Exposure Indices*. 7th ed. 2012 Supplement. Cincinnati, OH: ACGIH; 2012.