Occupational exposure to wood dust and the risk of upper respiratory tract diseases

Anna Rapiejko[1], Tomasz R. Sosnowski[2], Krzysztof Sosnowski[3], Dariusz Jurkiewicz[1]
[1] Department of Otolaryngology and Laryngological Oncology with Clinical Department of Cranio-Maxillofacial Surgery, Military Institute of Medicine – National Research Institute, Warsaw, Poland
[2] Faculty of Chemical and Process Engineering, Warsaw University of Technology, Poland
[3] Institute of Control and Industrial Electronics, Faculty of Electrical Engineering, Warsaw University of Technology, Poland

Abstract:
Wood dust is a common occupational exposure factor, particularly in carpentry, furniture manufacturing, sawmilling, and construction. The article reviews current knowledge on the impact of occupational exposure to wood dust on upper respiratory tract diseases. Health risks are influenced by the level and duration of exposure, particle characteristics, deposition sites, and individual host factors. Potential disease pathomechanisms are discussed. Workers exposed to wood dust may develop work-related rhinitis, which may progress to occupational asthma. The documented long-term effect is an increased risk of sinonasal cancer, particularly adenocarcinoma. The key role of technical prevention and medical surveillance is emphasised.

Key words: wood dust, occupational exposure, nasal cavity, rhinitis, sinonasal cancer

Introduction

At the end of the first decade of the XXI century, 2.9 million individuals in Europe were employed in the woodworking and furniture industry, and therefore potentially occupationally exposed to wood dust [1]. In Poland, according to the European WOODEX project conducted between 2000 and 2003, an estimated 310,000 individuals were professionally exposed to inhalable wood dust [2, 3]. Occupational exposure occurs in a wide variety of wood-related industries; with the highest prevalence recorded in construction, followed by the furniture industry, carpentry, sawmills and forestry. The most significant exposure levels occur during mechanical processing of dry wood [4].

The potential adverse health effects associated with exposure to wood dust particles are influenced by factors such as wood particle toxicity and particle size, which determine their penetration and deposition within the respiratory tract [4, 5]. Airborne wood dust exposure can cause a range of health outcomes affecting the upper respiratory tracts, including work-related rhinitis and sinonasal cancer [3, 4]. The European Commission has classified work involving exposure to hardwood and mixed wood dust as a carcinogenic technological process (in accordance with Directive 2017/2398/EC).

The aim of this review is to present the current state of knowledge on the impact of occupational exposure to wood dust on upper respiratory tract diseases, including various forms of rhinitis and nasal and paranasal malignancies.

Methods

The literature search covered PubMed, Scopus and Web of Science databases for articles published between 2010 and 2025. The search strategy used combinations of the following keywords: “wood dust”,

Alergoprofil 2025, Vol. 21, Nr 3, 10-18
DOI: 10.24292/01.AP.213191225

Received: 1.10.2025
Accepted: 17.11.2025
Published: 19.12.2025

"woodworkers", "occupational exposure", "nasal cavity", "rhinitis", and "sinonasal cancer". Preference was given to epidemiological studies and reviews published after 2010, although significant earlier works were also considered. No formal quality assessment was conducted, and studies were selected based on their relevance to occupational exposure to wood dust and effects on the upper respiratory tract.

Wood dust as an occupational exposure

Sources and types of exposure

Wood is situated between the central core of the trunk and the surrounding pulp and bark. Wood dust is produced as a by-product during the processing and conversion of wood and wood-based panels [3, 6]. The most dust-generating processes in the wood industry, especially during automated woodworking are sanding, sawing, and drilling [1].

Wood dust has varying moisture content: dry wood, with a moisture content below 15–20%, is less elastic than moist (green) wood. The processing of dry wood produces more overall dust and a higher proportion of inhalable particles [4, 7, 8].

Botanical classification divides trees into two groups: gymnosperms (conifers) and angiosperms (deciduous trees). The wood of gymnosperms is termed softwood, and the wood of angiosperms is termed hardwood. However, this classification does not necessarily reflect the wood's actual hardness. Hardwoods, such as beech, oak, birch, alder and mahogany, and softwoods such as pine, spruce, and fir, differ in density, extractive content, and allergenic potential [6]. Although only about 800 out of the 12,000 tree species are conifers (softwoods), yet roughly two-thirds of the wood used commercially worldwide comes from this group [4].

Wood is an organic substance composed mainly of cellulose (40–60%), a fibrous and very durable substance, found in wood alongside lignin and hemicelluloses. Hemicelluloses account for 18–35% of wood and are chemically classified as polysaccharides. Lignin is a complex mixture of several substances. The proportion of hemicellulose to lignin determines wood hardness; softwoods generally contain more lignin [3, 6]. Wood extractives, which account for 5–30% of the composition, are low-molecular-weight substances that can affect wood properties. These extractives include non-polar organic compounds (such as fatty acids, resin acids, waxes, alcohols, terpenes, sterols, steryl esters, and glycerides), polar organic compounds (including tannins, flavonoids, quinones, and lignans), and water-soluble substances (such as carbohydrates, alkaloids, proteins, and inorganic material) [9].

Wood dust from various tree species differs in hardness, specific gravity, particle morphology, and the composition of secondary substances, including terpenes, phenolic compounds such as tannins, flavonoids, and non-protein nitrogen compounds [3]. Although flavonoids are present in both hardwood and softwood, hardwood contains significantly more. The specific factors or combination of factors responsible for the varying toxic effects of hardwood and softwood dusts have not yet been clearly identified [6].

In occupational settings, exposure often extends beyond pure wood dust to include substances used in technological processes, such as resins, adhesives (including formaldehyde-based glues), paints, impregnating agents, and varnishes. Furthermore, the processing of moist or stored wood can expose workers to naturally occurring substances that may be pathogenic, allergenic, or toxic, including microbiological contaminants such as bacteria, actinomycetes, and moulds that produce mycotoxins [4, 6, 10].

Particle size and pattern of nasal deposition

The size of wood dust particles depends on the processing method; for example, sawing produces larger particles than sanding [1]. The relationship between particle size and wood type remains uncertain. For a specific process, several studies report similar particle-size distributions for pine and oak, whereas others indicate that hardwood dust is finer than softwood dust [1, 9].

An international convention defines health-related aerosol fractions for workplace particle measurement: the inhalable fraction, which can enter the respiratory tract; the thoracic fraction, which can penetrate the thoracic region; and the respirable fraction, which can reach the alveoli [11]. Wood dusts are generally categorized as containing 'coarse' and 'very coarse' particles. The majority of wood dust particles have an aerodynamic diameter ≥10 µm, which classifies them primarily as extra-thoracic and, to a lesser extent, thoracic [6, 9].

Exposure to wood dust results in the inhalation of particles through the nasal cavity. A significant proportion of these particles is deposited in the nasal cavities, while some enter the bronchi. Fine dust particles with diameters of 5 µm or less, known as the respirable fraction, can reach the alveoli [1]. The transport and deposition of particles are strongly influenced by par-

ticle morphology, including size, shape, and density, as well as by airflow dynamics and nasal cavity morphology, such as airway size and shape [12–14].

Inertial deposition plays the predominant role in the deposition of wood dust particles within the nasal cavities, and its efficiency can be characterised by the Stokes number (Stk):

$$Stk = \frac{u\rho d^2}{18\mu L}$$

where u (m/s) stands for the velocity of the particle, ρ (kg/m^3) represents particle density, d (m) is the effective particle diameter, μ represents air viscosity (kg/[m × s]), and L is the characteristic size of the air channel.

A higher Stk results in stronger particle inertia effects, that is, increased deposition. As airflow increases, both particle velocity (u) and Stk value rise, causing particles to deviate from streamlines at curvatures and abrupt turns, particularly in the nasal vestibule and anterior septal region. Inertial deposition is influenced not only by particle velocity (u) and diameter (d), but also by particle density (ρ). Less porous, heavier particles are more likely to undergo inertial deposition than more porous particles of the same size. Simulations by Tian et al. demonstrated that heavy oak dust settled in the nasal cavity to a greater extent than lighter dust of similar size [13]. However, particle size (d) has a greater influence on deposition than either velocity or density. Supporting this, Yu et al. found that large particles ≥20 µm were deposited in the upper respiratory tract regardless of their density [15]. Similarly, Sun et al. showed that wood dust particles exhibit higher nasal deposition and lower deposition in the lower respiratory tract [16]. The dimension of the air channel (L) is also an important parameter that affects the Stk. Lower values of L increase both the Stk and inertial deposition, which explains the elevated particle loads near the nasal valve and nasal regions characterised by narrowing [14].

Additional physical phenomena that influence the deposition of large aerosol particles include gravitational settling, electrostatic interactions, and direct interception. Gravitational settling contributes minimally because wood dust particles have little residence time in the nasal cavity during airflow. Electrostatic effects can be largely neglected due to the wet, charge-conductive surfaces of the nasal cavities. Consequently, direct interception remains an important deposition mechanism for large, non-spherical particles during their flow through narrow channels.

Figure 1. *Scanning electron microscope image of pine wood sanding dust at 1000× magnification (from authors' collection).*

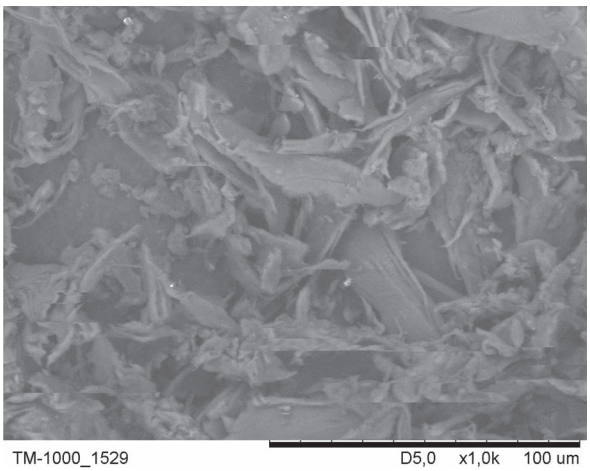

Figure 1 presents an electron microscope image of pine wood sanding dust with high-aspect-ratio fibres and plate-like fragments that are several dozen micrometres wide and significantly longer. These particles exhibit irregular, jagged edges and a rough, fibrous surface, which are characteristic features of fractured coniferous wood cell walls. The dimensions of pine wood sanding dust in the samples analysed by the authors were characterised by a volumetric diameter Dv10 of 15.52 µm, a median Dv50 of 43.35 µm, and a Dv90 of 116.3 µm. The fibres, driven mainly by inertial forces, cause the sediment to accumulate in the anterior region of the vestibule and the anterior part of the septum. Studies comparing carbon and glass fibres in nasal cavity replicas have confirmed that high-inertia fibres settle mainly in the anterior regions of the nasal cavity, whereas low-inertia fibres are capable of penetrating deeper into the respiratory tract [17]. Additionally, increasing fibre length or the aspect ratio limits penetration beyond the valve and turbinate areas. Higher flow velocities also raise the impact on the anterior regions, leading to deposition mainly in the vestibule and anterior part of the nasal septum [18, 19].

Wood dust is often non-spherical with a fibrous shape, as shown in figure 1. Therefore, in the analysis of inertial deposition, the effective particle diameter should be considered, which can be defined as the diameter of a sphere with the same mass as the fiber, i.e.

$$d = \sqrt[3]{\frac{6m}{\pi\rho}}$$

where m is the mass of the fibre. However, this assumption does not account for the possibility of fibre

reorientation (rotation) during flow, which promotes deeper penetration of the fibrous particle compared to a spherical particle of the same mass (fig. 2).

Figure 2. *The difference in behaviour between a fibre-shaped particle and a spherical particle of the same mass (an elongated particle can bypass an obstacle due to rotation).*

The physiological response to the removal of deposited wood dust particles is determined by the deposition sites within the nasal cavity. Two primary mechanisms facilitate the removal of wood particles: mechanical and mucociliary. Specifically, particles that do not pass through the nasal valve and settle on the squamous epithelium of the nasal vestibule, which lacks a mucous coating, are eliminated mechanically through sneezing and nose blowing. In contrast, particles deposited on the ciliated epithelium of the nasal cavity are cleared by the mucociliary mechanism, which transports mucus towards the pharynx [13]. However, limited information is available regarding the transport, deposition, and elimination of particles in the maxillary and frontal sinuses [4].

High levels of wood dust exposure can reduce mucociliary clearance and, in some cases, cause mucostasis [9, 20]. Normally, the mucociliary transit time in humans is approximately 12–15 min. However, in the study by Özler and Akoğlu, the average nasal mucociliary clearance time among wood industry workers, as measured by the saccharin test, was significantly longer: 16.72 (±2.71) min compared to the control group: 12.28 (±1.98) min ($p < 0.0001$). Furthermore, a positive correlation was observed between the duration of wood dust exposure and the prolongation of mucociliary clearance time [20]. As a result, impaired nasal mucociliary function increases the duration of contact between wood dust particles and the nasal mucosa, which can, in turn, result in adverse health effects in the nasal region [21, 22].

Key factors influencing upper respiratory tract hazards include the level and duration of wood dust exposure, particle properties, airflow rate, deposition sites, residence time within the respiratory tract, and host-specific risk factors [14]. Host factors include genetic susceptibility, including atopy, and exposure to tobacco smoke [23].

Work-related rhinitis

Jacobsen et al. report that rhinitis and upper respiratory tract symptoms are among the most prevalent complaints among workers in the wood industry, regardless of exposure to fresh, mixed or dry wood [7, 8]. A review of 37 epidemiological studies on dry wood exposure demonstrated a higher prevalence of work-related rhinitis (9–26%) among workers exposed to dry wood dust compared to unexposed individuals [8]. Zamora & Poole indicate that occupational rhinitis is diagnosed at least 3 times more frequently than occupational asthma, and that more than half of occupational asthma cases involve coexisting rhinitis [24]. Siracusa's et al. review identified occupational rhinitis in nearly 60 different professions, with 'dusty' industries, including the wood industry, representing the highest risk groups [25].

Work-related rhinitis is triggered or worsened by workplace exposures and may involve both immunological and non-immunological mechanisms. It includes 2 primary forms: occupational rhinitis and work-exacerbated rhinitis [26]. Occupational rhinitis is characterized by symptoms such as rhinorrhoea, nasal congestion, sneezing, itching, nasal discomfort, and post-nasal drip, which are directly attributable to occupational exposure in individuals without prior rhinitis. These symptoms typically resolve upon cessation of exposure [24, 26, 27]. Occupational rhinitis can be classified as allergic (IgE-dependent or non-IgE-dependent) or non-allergic/irritant [27]. Low-molecular-weight agents (less than 10 kDa), including wood dust, primarily induce rhinitis symptoms through non-IgE-mediated mechanisms, often functioning as haptens or irritants [27]. Occupational conjunctivitis frequently co-occurs. Differentiating between irritant and allergic mechanisms can be clinically challenging, as both processes may coexist in some individuals [27, 28]. In work-exacerbated rhinitis, pre-existing rhinitis is aggravated by workplace stimuli, with aggravating factors typically acting through non-immune mechanisms [24, 26].

Histological examinations in exposed workers have revealed mucosal hyperplasia, goblet cell metaplasia, and chronic inflammatory infiltrates in the nasal mucosa. Analysed by Jacobsen et al. and cytological studies by Staffieri et al. and Lovato et al. indicate that in most cases of occupational rhinitis caused by wood dusts, neutrophilic components and chronic, non-specific inflammation predominate, while IgE-dependent sensitisation is relatively uncommon [7, 8, 29, 30]. Chronic neutrophilic rhinitis is diagnosed more frequently in exposed workers

than in controls, with an allergic component present in some individuals [29, 30]. Woodworkers exhibiting lymphocytes in nasal smears typically have longer durations of wood dust exposure, and hardwood dust elicits a stronger inflammatory response [30]. Allergic mechanisms are implicated in workers who develop specific IgE-mediated sensitisation to allergenic proteins found in several hardwood and softwood species. Sensitisation to moulds and actinomycetes present on stored wood has also been associated with upper airway symptoms. Schlünssen et al. demonstrated that IgE-mediated sensitisation to pine and beech dust occurs in only a small proportion of workers, primarily at high exposure levels, and does not account for most nasal symptoms [31]. This finding supports the conclusion that the majority of rhinitis cases in the carpenters are non-allergic or irritant in nature. Additional evidence from analyses of woodworkers' nasal lavage fluids suggests that wood dust acts as a mucous membrane irritant, provoking a chronic inflammatory response. Objective assessment using rhinomanometry and acoustic rhinometry have confirmed nasal obstruction in workers [32]. A reduction in nasal cavity volume and an increase in perceived nasal congestion have been objectively documented. Furthermore, exposure to wood dust is associated with acute nasal obstruction in a dose-dependent manner, as measured by acoustic rhinometry, with intensified effects observed at wood dust concentrations >1 mg/m³ [32].

Impairment of mucociliary clearance, mucosal oedema and chronic inflammation in the upper respiratory tract serve as important pathophysiological mechanism linking these changes to chronic diseases such as rhinosinusitis and potentially to nasal and sinus cancers. Additional occupational non-malignant upper airway conditions include nasal erosions, laryngitis, olfactory dysfunction, and vocal cord dysfunction [33].

Zamora et al., Moscato et al. and Zhao et al. highlight the strong association between rhinitis and occupational asthma [24, 26, 33]. Analyses of occupational asthma cases indicate that rhinitis frequently precedes asthma development and is present in more than half of affected individuals [24, 28]. Moscato et al. demonstrated that the coexistence of occupational rhinitis with occupational asthma correlates with increased severity and chronicity of asthma, which has direct prognostic and therapeutic implications [26]. Early recognition of occupational rhinitis is essential for the prevention of occupational asthma [24, 27].

Upper respiratory tract cancers

Nasal and paranasal sinus cancer

The most robustly documented association between wood dust exposure and cancer involves malignancies of the nasal cavity and paranasal sinuses, particularly adenocarcinoma [4]. Sinonasal cancer is rare, representing less than 1% of all cancers and under 4% of head and neck tumours. Its incidence is approximately 1 case per 100,000 person-years in most developed countries [34, 35].

The International Agency for Research on Cancer assessment (IARC), which synthesised data from multiple cohort and case-control studies, identified a markedly elevated risk of nasal cavity and paranasal sinus cancer, particularly adenocarcinoma, among workers exposed to hardwood dust (beech, oak). Evidence for squamous cell carcinoma of the sinuses and nasal cavity was weaker and less consistent. Wood dust was classified as a group 1 human carcinogen [9, 36]. A meta-analysis of 28 studies by Binazzi et al. demonstrated a several-fold increase in the risk of all sinonasal cancer following wood dust exposure, with the strongest association observed for adenocarcinomas (an increase of several dozen times). A statistically significant dose-response trend was also confirmed [21]. It is estimated that the latency period between exposure to wood dust and the onset of wood dust-induced nasal cancer is at least 20 years [10]. Soćko et al. conducted risk assessments based on epidemiological data and estimated that, at typical exposure levels in the wood industry, risk increases significantly after 30–35 years of work, especially at higher wood dust concentrations [6]. Simultaneous exposure to hardwood dust and formaldehyde increases the risk of nasopharyngeal cancer [10, 37].

The pathomechanism underlying these cancers involves prolonged, high-dose exposure of the nasal cavity and sinus epithelium to fine wood dust particles. This exposure results in chronic inflammation, impaired mucociliary transport, metaplasia, and epithelial dysplasia [4, 9, 21, 36, 38]. Özler et al. found that wood industry workers exhibited significantly prolonged mucociliary clearance time compared to controls, which supports the proposed mechanism [20]. Additionally, IARC demonstrated that beech and oak extracts possess genotoxic properties, such as inducing mutations in bacteria and causing DNA damage in hepatocytes, while spruce extracts did not exhibit such effects [9, 36]. These cancers typically have a long latency period.

The clinical presentation is generally non-specific, frequently resulting in diagnostic delays. Reported symptoms include chronic, unilateral nasal obstruction, epistaxis, rhinorrhea, facial pain or a sensation of facial fullness, toothache, possibly anosmia and orbital symptoms [39].

Nasopharyngeal cancer

Nasopharyngeal cancer is rare, with approximately 133,000 new cases reported globally, representing 0.7% of all malignant cancers [40]. The association between wood dust exposure and nasopharyngeal cancer is less well documented. A meta-analysis by Meng et al. reported a twofold increase in risk with high wood dust exposure, with higher concentrations and longer duration further elevating risk [41]. Beigzadeh et al. confirmed this association, noting a lower risk in Europe compared to Asia and America, potentially due to genetic factors, differing exposures, and varying health and safety standards. The risk was particularly elevated for squamous cell nasopharyngeal carcinoma [42]. A case-control study in Thailand identified a significant risk increase for types 2 and 3 cancers (non-keratinising and undifferentiated nasopharyngeal cancer), with higher risk associated with exposure exceeding 10 years, initial exposure after 25, and high cumulative exposure [43]. Proposed pathomechanisms include interactions between Epstein–Barr virus and other environmental factors such as tobacco smoke and pollution, as well as the irritant effect of wood dust on the nasopharyngeal epithelium [42–44].

Nasopharyngeal cancer often remains asymptomatic for extended periods and typically presents with non-specific symptoms. The most common manifestations include neck swelling, unilateral nasal obstruction, recurrent epistaxis, and ear symptoms resulting from middle ear inflammation. In advanced stages, patients may experience headaches and symptoms of cranial nerve neuropathy [45].

Laryngeal cancer

Laryngeal cancer is a moderately common cancer, with approximately 185,000 new cases diagnosed globally in 2020, accounting for about 1% of all new cancer cases [40]. The evidence for a link between wood dust and laryngeal cancer is much weaker than for the cancers discussed above. A meta-analysis by Meng et al. did not show a clear, statistically significant increase in laryngeal cancer risk across the entire sample. However, some subgroups showed a moderately increased odds ratio [46]. Dutch National Institute for Public Health and the Environment report also stated that the evidence for laryngeal cancer is limited [4].

The pathomechanism of the tumour is postulated to be, as is generally the case for the respiratory tract, chronic accumulation of dust particles in the larynx. This leads to prolonged irritation, reactive oxygen species and DNA damage, potentially exacerbated by concomitant exposure to tobacco smoke, which is the dominant risk factor for laryngeal cancer [4, 41].

The clinical presentation of laryngeal cancer typically includes hoarseness, sore throat, dysphagia, otalgia, dyspnoea, and oral symptoms [47].

Prevention and occupational health

Due to the well-documented link between exposure to wood dust and cancer of the nasal cavity and paranasal sinuses, as well as numerous non-malignant respiratory diseases, wood dust has been classified as a carcinogen and allergen in the European Union. A binding limit value of 2 mg/m³ has been introduced for the inhalable fraction, and Poland has adopted the same value as the threshold limit value (NDS) for all types of wood dust, lowering the previous limit of 3 mg/m³ [4, 6]. At the same time, expert analyses (Scientific Committee on Occupational Exposure Limits, Dutch Expert Committee on Occupational Standards), cited in Rijs's et al. review, suggest that significant respiratory effects may occur above 0.5 mg/m³ (total dust) and 1 mg/m³ (inhalable fraction). This argues for keeping exposure as low as possible below the legal limit [4].

Szewczyńska & Pośniak conducted an environmental study of the Polish furniture industry and found that the actual concentrations of the respirable fraction of wood dust often significantly exceed expected values. The concentrations of the inhalable fraction ranged from 1.34 to 22.13 mg/m³, and the respirable fraction accounted for approximately 25% of the inhalable fraction, which emphasises the risk to the lower respiratory tract. The highest concentrations of both fractions were observed at grinding stations [48].

The basis for prevention is therefore the elimination or reduction of exposure at source. Documentation by Soćko et al. and Central Institute for Labour Protection (CIOP-PIB) guidelines indicate the need to use local extraction systems on all machines and sealed dust extraction systems. Effective general ventilation is also needed. Automation and sealing of the most dust-producing processes (grinding, cutting) are recommended [3, 6]. From an occupational health per-

spective, it is also crucial to eliminate dry sweeping and compressed air blowing and use industrial vacuum cleaners and central extraction systems, proper storage of dust in closed containers, and provision of appropriate work clothing, washing without beating, and access to changing rooms with showers [3]. Personal protective equipment, such as filtering half masks, is recommended as a supplement, not as the basis for protection [6].

As work involving exposure to wood dust has been classified as carcinogenic, this entails specific legal requirements. This includes keeping a register of exposed work and employees to store exposure documentation and test results, for long-term storage, and to conduct systematic occupational risk assessment [3, 6].

From the point of view of occupational medicine, it is important to strengthen preventive supervision, systematically monitor symptoms of the upper and lower respiratory tract, conduct periodic functional tests, maintain oncological vigilance, adjust workstations based on identified symptoms, educate, and document the course of exposure and disease.

Available data show that prevention is indeed effective. Jacobsen et al. conducted a study in the Danish furniture industry that compared 2 cross-sectional cohorts of furniture workers. They showed that a reduction in average dust concentrations of approximately 40% over 6 years was associated with a decrease in the prevalence of ever wheezing (2.4%), coughing (4.9%), chronic bronchitis (2%), and nasal symptoms (6%). [49] This confirms that a decrease in wood dust exposure may result in fewer workers experiencing respiratory symptoms.

Conclusions

Occupational exposure to wood dust is a significant public health issue, particularly in carpentry, furniture manufacturing, sawmilling, and construction. The available literature shows that workers are most exposed during the mechanical processing of dry wood, such as sanding, cutting, and drilling. The health risk is also influenced by particle characteristics, such as size, shape, and density, as well as airflow conditions and the anatomy of the nasal cavity. These factors determine where the dust is deposited and the duration of contact with the mucous membrane.

The most common effects of exposure are upper respiratory tract complaints, mainly work-related rhinitis. The pathomechanism of these problems is mainly based on non-allergic/irritant mechanisms and chronic, non-specific inflammation and possible impairment of mucociliary clearance. This affects the occurrence of symptoms such as nasal obstruction, sneezing, and nasal discharge. In addition, prolonged dust exposure promotes chronic inflammation and may contribute to more serious health consequences.

Clinically, the most serious long-term effect of exposure is an increased risk of cancer of the nasal cavity and paranasal sinuses, particularly adenocarcinoma in people exposed to hardwood dust. Wood dust is classified as a carcinogen in the European Union. It is therefore important to keep concentrations as low as possible and to implement preventive measures. Prevention is based on reducing emissions at source, proper technological cleaning, and the use of personal protective equipment. Occupational health surveillance is also important, including monitoring symptoms, periodic examinations, documenting exposure, and maintaining oncological vigilance. Available studies show that reducing dust concentrations indeed decreases the frequency of respiratory symptoms, which emphasises the real effectiveness of workplace prevention.

References

1. *European Federation of Building and Woodworkers EFBWW, CEI-Bois and Usl7. Raport: Less Dust. Brussels, Belgium 2010. http://www.efbww. eu publications-and-downloads/ reports-and-studies/less- dust/514-a PL.pdf.*
2. *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: 549-61.*
3. *Bujak-Pietrek S, Soćko R, Konieczko K et al. Pyły drewna, wytyczne dla pracodawców, pracowników i służb związanych z bezpieczeństwem i higieną pracy. Instytut Medycyny Pracy im. Prof. J. Noffera, Łódź 2021.*
4. *Rijs K, van Triel J, Bos P et al. Occupational exposure to wood dust. A systematic review of the literature. RIVM report 2021-0146. RIVM, 2021.*
5. *Rapiejko A, Sosnowski T, Sosnowski K et al. Experimental analysis of allergenic tree pollen deposition in a three-dimensional anatomical model of the adult nasal cavity. Otolaryngol Pol. 2025; 79(5): 1-20.*
6. *Socko R, Pakulska D, Szymczak W. Wood dust – inhalable fraction. Documentation of proposed values of occupational exposure limits (OELs). Podstawy i Metody Oceny Środowiska Pracy. 2021; 2(108): 27-138.*
7. *Jacobsen G, Schaumburg I, Sigsgaard T et al. Non-malignant respiratory diseases and occupational exposure to wood dust. Part I. Fresh wood and mixed wood industry. Ann Agric Environ Med. 2010; 17(1): 15-28.*

8. *Jacobsen G, Schaumburg I, Sigsgaard T et al. Non-malignant respiratory diseases and occupational exposure to wood dust. Part II. Dry wood industry. Ann Agric Environ Med. 2010;17(1): 29-44.*
9. *International Agency for Research on Cancer, World Health Organization: IARC Monogr Eval Carcinog Risks Hum. Vol. 62. Wood dust and Formaldehyde. Agency, Lyon 1995; 62: 35-215.*
10. *S.T.O.P. Carcinogens at Work. https://stopcarcinogensatwork.eu/fact/hardwood-dust.*
11. *European Committee for Standardization (CEN). Workplace atmospheres – Size fraction definitions for measurement of airborne particles. Report No. BS EN 481. CEN, London, UK 1993.*
12. *Shen Z, Tolu Mekonne T, Cai X et al. Experimental measurements of particle deposition in the human nasal airway. Int J Pharm. 2025; 672: 125280.*
13. *Tian ZF, Inthavong K, Tu JY. Deposition of inhaled wood dust in the nasal cavity. Inhal Toxicol. 2007; 19: 1155-65.*
14. *Guo Y, Tang Y, Su Y et al. Influencing factors of particle deposition in the human nasal cavity. Laryngoscope Investig Otolaryngol. 2024; 9(4): e1308.*
15. *Yu H, Xie Y, Yang X et al. Migration and deposition characteristics of micro-dust in the human upper airway in dust-polluted tunnel environments. J Hazard Mater. 2025; 488: 137371.*
16. *Sun B, Jia J, Zhou G. Impact of inhalable dust with different material properties and particle morphologies on respiratory deposition in occupational exposure. Process Saf Environ Prot. 2025; 199: 107220.*
17. *Su WC, Wu J, Marijnissen JCM et al. Deposition of Man-Made Fibers in a Human Nasal Airway. Aerosol Science and Technology. 2008; 42: 173-81.*
18. *Ma R, Yang F, Wang Y et al. Numerical analysis of Platanus orientalis L. microfiber inhalation exposure in human sinonasal cavities. J Aerosol Sci. 2025; 188: 106617.*
19. *Dastan A, Abouali O, Ahmadi G. CFD simulation of total and regional fiber deposition in human nasal cavities. J Aerosol Sci. 2014; 69: 132-49.*
20. *Özler GS, Akoğlu E. Impairment of nasal mucociliary clearance time in wood industry workers. Eur Arch Otorhinolaryngol. 2020; 277(2): 493-6.*
21. *Binazzi A, Ferrante P, Marinaccio A. Occupational exposure and sinonasal cancer: a systematic review and meta-analysis. BMC Cancer. 2015; 15: 49.*
22. *Sun SS, Hsieh JF, Tsai SC et al. The role of rhinoscintigraphy in the evaluation of nasal mucociliary clearance function in patients with sinusitis. Nucl Med Commun. 2000; 21: 1029-32.*
23. *Palczynski C, Łacwik P, Kuprys-Lipinska I et al. Nieżyt nosa związany z pracą zawodową. Alergia. 2019; 2: 6-9.*
24. *Zamora-Sifuentes J, Poole JA. Occupational Rhinitis: An Update. Curr Allergy Asthma Rep. 2023; 23(10): 579-87.*
25. *Siracusa A, Desrosiers M, Marabini A. Epidemiology of occupational rhinitis: prevalence, aetiology and determinants. Clin Exp Allergy. 2000; 30(11): 1519-34.*
26. *Moscato G, Vandenplas O, Van Wijk RG et al.; European Academy of Allergology and Clinical Immunolgy. EAACI position paper on occupational rhinitis. Respir Res. 2009; 10(1): 16.*
27. *Hox V, Steelant B, Fokkens W et al. Occupational upper airway disease: how work affects the nose. Allergy. 2014; 69: 282-91.*
28. *Shusterman D. Occupational irritant and allergic rhinitis. Curr Allergy Asthma Rep. 2014; 14(4): 425.*
29. *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(9): 814-9.*
30. *Lovato A, Staffieri C, Ottaviano G et al. Woodworkers and the inflammatory effects of softwood/hardwood dust: evidence from nasal cytology. Eur Arch Otorhinolaryngol. 2016; 273(10): 3195-200.*
31. *Schlünssen V, Kespohl S, Jacobsen G et al. Immunoglobulin E-mediated sensitization to pine and beech dust in relation to wood dust exposure levels and respiratory symptoms in the furniture industry. Scand J Work Environ Health. 2011; 37(2): 159-67.*
32. *Schlünssen V, Schaumburg I, Andersen NT et al. Nasal patency is related to dust exposure in woodworkers. Occup Environ Med. 2002; 59(1): 23-9.*
33. *Zhao YA, Shusterman D. Occupational rhinitis and other work-related upper respiratory tract conditions. Clin Chest Med. 2012; 33(4): 637-47.*
34. *Franchi A, Miligi L, Palomba A et al. Sinonasal carcinomas: recent advances in molecular and phenotypic characterization and their clinical implications. Crit Rev Oncol Hematol. 2011; 79(3): 265-77.*
35. *Mensi C, Consonni D, Sieno C et al. Sinonasal cancer and occupational exposure in a population-based registry. Int J Otolaryngol. 2013; 2013: 672621.*
36. *International Agency for Research on Cancer, World Health Organization. IARC Monographs on the evaluation of carcinogenic risk to humans. Vol. 100 C. Arsenic, metals, fibres and dusts. Agency, Lyon 2012.*
37. *Siew SS, Martinsen JI, Kjaerheim K et al. Occupational exposure to wood dust and risk of nasal and nasopharyngeal cancer: A case-control study among men in four Nordic countries, with an emphasis on nasal adenocarcinoma. Int J Canc. 2017; 141: 2430-6.*
38. *Alonso-Sardón M, Chamorro AJ, Hernández-García I et al. Association between occupational exposure to wood dust and cancer: a systematic review and meta-analysis. PLoS One. 2015; 10(7): e0133024.*
39. *Resteghini C, Baujat B, Bossi P et al.; ESMO Guidelines Committee. Sinonasal malignancy: ESMO-EURACAN Cli-*

nical Practice Guideline for diagnosis, treatment and follow-up. ESMO Open. 2025; 10(2): 104121.

40. *Sung H, Ferlay J, Siegel RL et al. Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA Cancer J Clin. 2021; 71(3): 209-49.*
41. *Meng E, Yin J, Jin W et al. Wood dust exposure and risks of nasopharyngeal carcinoma: a meta-analysis. Eur J Public Health. 2020; 30(4): 817-22.*
42. *Beigzadeh Z, Pourhassan B, Kalantary S et al. Occupational exposure to wood dust and risk of nasopharyngeal cancer: A systematic review and meta-analysis. Environ Res. 2019; 171: 170-6.*
43. *Ekpanyaskul C, Sangrajrang S, Ekburanawat W et al. Semi-quantitative exposure assessment of occupational exposure to wood dust and nasopharyngeal cancer risk. Asian Pac J Cancer Prev. 2015; 16(10): 4339-45.*
44. *Zhang JX, Xu H, Shen T et al. Wood dust exposure and risk of sinonasal and nasopharyngeal cancer: a meta-analysis. Austin J Derm. 2014; 1(2): 1-5.*
45. *Hasnaoui M, Lahmar R, Ben Mabrouk A et al. Predictive epidemiological and clinical factors of nasopharyngeal carcinoma diagnosis: Adult versus pediatric population. Int J Pediatr Otorhinolaryngol. 2020; 137: 110203.*
46. *Meng E, Xin Z, Jianrui D et al. Meta-Analysis of the Relationship Between Occupational/Environmental Exposure to Wood Dust and Laryngeal Cancer. Cancer Med. 2024; 13(20): e70330.*
47. *Shephard EA, Parkinson MA, Hamilton WT. Recognising laryngeal cancer in primary care: a large case-control study using electronic records. Br J Gen Pract. 2019; 69(679): e127-e133.*
48. *Szewczyńska M, Pośniak M. Ocena narażenia zawodowego na pyły drewna pracowników przemysłu meblarskiego w Polsce. Medycyna Pracy. 2017; 68(1): 45-60.*
49. *Jacobsen G, Schaumburg I, Sigsgaard T et al. Wood Dust Exposure Levels and Respiratory Symptoms 6 Years Apart: An Observational Intervention Study Within the Danish Furniture Industry. Ann Work Expo Health. 2021; 65(9): 1029-39.*

Wkład pracy/Author's contributions:
Conceptualization: A.R., T.S., D.J.
Literature search: A.R., K.S.
Writing – Original Draft: A.R., T.S.
Writing – Review & Editing: A.R., T.S., K.S., D.J.
Supervision: T.S., D.J.
Konflikt interesów/Conflict of interests:
Nie występuje.
Finansowanie/Financial support:
Nie występuje.
Etyka/Ethics:
Treści przedstawione w artykule są zgodne z zasadami Deklaracji Helsińskiej, dyrektywami EU oraz ujednoliconymi wymaganiami dla czasopism biomedycznych.

ORCID
Anna Rapiejko – ID – https://orcid.org/0000-0002-8906-2405
Tomasz R Sosnowski – ID – https://orcid.org/0000-0002-6775-3766
Krzysztof Sosnowski – ID – https://orcid.org/0009-0004-7304-4642
Dariusz Jurkiewicz – ID – https://orcid.org/0000-0003-3729-2679

Correspondence
Anna Rapiejko, MD
Department of Otolaryngology and Laryngological Oncology with Clinical Department of Cranio-Maxillofacial Surgery, Military Institute of Medicine – National Research Institute, Warsaw
04-141 Warszawa, ul. Szaserów 128
e-mail: ania.rapiejko@gmail.com