National Institute for Public Health
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Ministry of Health, Welfare and Sport

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A systematic review of the literature

RIVM report 2021-0146

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Synopsis

Occupational exposure to wood dust
A systematic review of the literature

Employees can be exposed to wood dust while performing woodworking processes such as sawing and sanding. Such exposure is known to be associated with health problems. RIVM has carried out a review of the scientific literature on wood dust and occupational exposure to wood dust.

The data has not been compared or interpreted by RIVM. The data was collected at the request of the Health Council. The Health Council will use this data to provide a recommendation to the Minister of Social Affairs and Employment on whether or not the health-based occupational exposure limit for wood dust needs to be adjusted.

The review focused on various aspects including characteristics of wood dust as a result of wood processing and the mechanism and degree of exposure, as well as what is known about what happens in the body after exposure and what diseases and conditions people can develop as a result.

Long-term exposure to wood dust can cause nasal cancer. It can also impair lung function, leading to difficulty breathing, and cause irritation of the eyes, nose, lungs, and skin.

Keywords: wood dust, nasal cancer, lung function, wood working, occupational health and safety, exposure, health-based occupational exposure limit, workplace exposure, respiratory diseases, wood

Publiekssamenvatting

Beroepsmatige blootstelling aan houtstof.
Een systematisch literatuuronderzoek

Werknemers die hout bewerken, zoals zagen en schuren, kunnen blootstaan aan houtstof. Het is bekend dat deze blootstelling schadelijk kan zijn voor de gezondheid. Het RIVM heeft alle kennis in de wetenschappelijke literatuur over houtstof en blootstelling aan houtstof op de werkvloer verzameld.

Het RIVM heeft de informatie niet vergeleken of geïnterpreteerd. De informatie is verzameld in opdracht van de Gezondheidsraad. De Gezondheidsraad zal met deze informatie de minister van Sociale Zaken en Werkgelegenheid (SZW) adviseren of de gezondheidskundige advieswaarde voor blootstelling van werknemers aan houtstof moet worden aangepast.

Het gaat onder andere om kenmerken van het stof door houtbewerking, hoe mensen eraan blootstellen en in welke mate. Ook is gekeken wat bekend is wat er in het lichaam gebeurt na blootstelling, en welke ziektes en aandoeningen mensen ervan kunnen krijgen.

Het is bekend dat werknemers neuskanker kunnen krijgen als zij langdurig aan houtstof blootstaan. Ook kan het de functie van de longen verminderen, waardoor mensen onder andere moeilijker kunnen ademhalen. Verder kan het irritatie aan de ogen, neus, longen en huid veroorzaken.

Kernwoorden: houtstof, neuskanker, longfunctie, houtbewerking, arbo, blootstelling, gezondheidskundige advieswaarde, beroepsmatige blootstelling, luchtwegaandoeningen, hout

Contents

Summary

A systematic review was performed on characteristics of the substance wood dust and occupational exposure to wood dust. Relevant studies published before 11 May 2021 were reviewed. The search was performed by using the scientific search engines Embase and Scopus. In addition, relevant information summarized by (inter)national organizations such as the International Agency for Research on Cancer (IARC), the Health Council of the Netherlands, the American Conference of Governmental Industrial Hygienists (ACGIH) and the Scientific Committee on Occupational Exposure Limits (SCOEL) was used in the current review.

The characteristics of the substance wood dust (Chapter 3) and its occurrence, production and use (Chapter 4) were described. The methods used to measure and analyze occupational exposure to wood dust were summarized in Chapter 5. The route of exposure and the information regarding occupational exposure to wood dust in terms of the concentrations in the air are described in Chapter 6. The kinetics (absorption, distribution, metabolism and excretion) of exposure to wood dust are subsequently described in Chapter 7. All relevant *in vivo* studies on wood dust (including wood dust extracts, condensates and wood drying fumes) (Chapter 8) and relevant longitudinal epidemiological studies on the association between occupational wood dust exposure and health (Chapter 9) that were found in the current literature search were summarized. In Chapter 10, mode of action and pathogenesis are described. Finally, existing guidelines, standards and evaluations are described in Chapter 11.

The data found has been summarized but has not been weighed, compared or interpreted by RIVM. At the request of the Dutch Minister of Social Affairs and Employment, the Health Council of the Netherlands, in cooperation with the Nordic Expert Group, will use the summaries to provide a recommendation for a health-based occupational exposure limit.

1 Introduction

RIVM has carried out a review of literature on occupational exposure to wood dust. Employees can be exposed to wood dust while performing woodworking processes such as sawing, drilling, planing, sanding and shaving. Exposure to wood dust may cause carcinogenic effects (e.g. sinonasal cancer), non-carcinogenic effects on the respiratory tract (e.g. alterations in respiratory function) and dermatosis.

The data found has been summarised but has not been weighed, compared or interpreted by RIVM. The review covered literature on substance identification, occurrence, production and use, measurement and analysis of workplace exposure, occupational exposure data, kinetics, effects in animals, observations in humans, mode of action and pathogenesis, and existing guidelines, standards and evaluations.

At the request of the Dutch Minister of Social Affairs and Employment, the Health Council of the Netherlands, in cooperation with the Nordic Expert Group, will use the summaries to assess adverse health effects, and to provide a recommendation for a health-based occupational exposure limit.

2 Method

Literature was retrieved from the databases Embase and Scopus. The search was performed in March 2020 and updates were carried out on 1 October 2020 and 11 May 2021. Relevant search terms were agreed upon by the research team and the library of RIVM (see Appendix 1). In addition, relevant information summarised by (inter)national organisations (i.e. 'grey' literature) such as the International Agency for Research on Cancer (IARC), the Health Council of the Netherlands, the American Conference of Governmental Industrial Hygienists (ACGIH) and the Scientific Committee on Occupational Exposure Limits (SCOEL) was included in the current review. Appendix 2 contains a list of abbreviations used in the current report.

The review covered literature focused on occupational exposure to softwood and hardwood dust. In general, for Chapters 3–5, 6.1, 7 and 11, reviews of the grey literature were used, and supplemented where relevant by literature retrieved from the Embase and Scopus databases.

In Chapters 6.2 and 8–10, relevant literature retrieved from Embase and Scopus were used. Studies summarised in Chapter 9 are not summerised in Chapter 6. In Chapter 9, only literature focused on *occupational* exposure to soft- or hardwood dust was reviewed, whereas in other chapters (e.g. Chapters 7, 8 and 10) the search was not limited to literature on occupational exposure.

Additional selection criteria were applied to Chapter 9 because the number of epidemiological studies performed was too large to review in the time available:

- Health outcomes reported by IARC (1) and SCOEL (2) to potentially be associated with wood dust exposure:
  - carcinogenic effects: sinonasal cancer, nasopharynx cancer, pharynx cancer, larynx cancer and lung cancer (1);
  - irritation and sensitisation (2):
    - non-carcinogenic effects in the upper respiratory tract: increased incidence of metaplasia in the nasal mucosa, reduced mucociliary clearance, altered olfactory function, increased frequency of nasal symptoms (obstruction, discharge, nose blowing), episodes of ocular irritation, pharyngeal disorders and chronic bronchitis;
    - non-carcinogenic effects in the lower respiratory tract: asthma and rhinitis, coughing, chronic bronchitis, alteration of the respiratory function parameters, idiopathic pulmonary fibrosis and extrinsic allergic alveolitis.
  - non-carcinogenic effects on skin: dermatosis.
- Only literature in which an exposure–response relationship is examined with estimated or measured exposure information (mg/m^3) is summarised in Chapter 9 because this is one of the requirements for determining a health-based recommended occupational exposure limit. Therefore, studies without any

(semi-)quantitative information on wood dust exposure are not included.
- Case reports and studies not written in English or Dutch were excluded.

Depending on the treatments to which wood has been subjected before processing, the dust can contain additives, preservatives or adhesives (2) (additional information on other agents to which workers may be exposed during the processing of wood may be found, for instance, in a review performed by IARC (see paragraph 1.2.4 of IARC 2012 (1)). In the current study, only exposure to wood dust is of interest. Therefore, literature in which untreated wood was examined is most relevant. The *in vivo* and *in vitro* studies described in Chapters 8 and 10 were limited to untreated wood only (unless otherwise specified). However, very few epidemiological studies have been performed in which only untreated wood was examined. Therefore, studies in which untreated and/or treated wood exposure was examined were summarised in Chapter 9.

3 Substance identification

3.1 Name and other identifiers of the substance

Table 1 Name and other identifiers of the substance

Name(s) in the IUPAC nomenclature or other international chemical name(s)	Hardwood and softwood dust
Other names (usual name, trade name, abbreviation)	Sawdust
ISO common name (if available and appropriate)	Not applicable
EC/EINECS number (if available and appropriate)	Not applicable
EC name (if available and appropriate)	Not applicable
CAS number	Not applicable
Other identity code (if available)	Not applicable
Molecular formula	Not applicable
Structural formula	Not applicable
SMILES notation (if available)	Not applicable
Molecular weight or molecular weight range	Varies
Information on optical activity and typical ratio of (stereo) isomers (if applicable and appropriate)	Not applicable
Description of the manufacturing process and identity of the source (for UVBC substances only)	Not applicable
Degree of purity (%)	Not applicable

Detailed information on substance identification is given by several international organisations, including IARC (1, 3), the Health Council of the Netherlands (4), ACGIH (2002) (5) and SCOEL (2003) (2). The most extensive description of wood dust identification and chemical composition is given in a review performed by IARC (3). Below is a summary mainly of reviews performed by IARC (1, 3), supplemented by information taken from earlier evaluations by other international organisations.

Wood dust is any wood particle generated in the processing of wood for a wide range of uses. Wood dust is frequently described by wood species or as hardwood or softwood. The terms ‘hardwood’ and ‘softwood’ refer to angiosperm trees with encapsulated seeds (principally deciduous and porous) and gymnosperm trees with exposed seeds (principally evergreen and nonporous), respectively, and not necessarily to the hardness of the wood. See Table 2 for differences between softwoods and hardwoods as summarised by IARC (3). Out of 12,000 different species of trees, only about 800 are coniferous or softwoods, but roughly two-thirds of the wood used commercially worldwide belongs to

the group of softwoods. Hardwoods tend to be somewhat more dense, and have a higher content of polar extractives than softwoods (3).

Table 2 Comparison of softwoods and hardwoods from IARC (3)

Characteristic	**Gymnosperms/conifers/ softwoods**	**Angiosperms/deciduous wood/hardwoods**
World production of industrial round wood (1980) (× 1,000 m^3)	990,000	450,000
Density (g/cm^3)	White (silver) fir: mean 0.41 (0.32–0.71)	European beech: mean 0.68 (0.49–0.88)
	European spruce: mean 0.43 (0.30–0.64)	European oak: mean 0.65 (0.39–0.93)
	Scots pine: mean 0.49 (0.30–0.86)	
Fibres	Long (1.4–4.4 mm)	Short (0.2–2.4 mm)
Cell type	One (tracheids)	Various
Cellulose	~40–50%	~40–50%
Unit	β-d-Glucose	β-d-Glucose
Fibre pulp	Long	Short
Polyoses	~15–30%	~25–35%
Units	More mannose	More xylose
	More galactose	
Lignin	~25–35%	~20–30%
Units	Mainly guaiacyl	Mainly syringyl or guaiacyl
Methoxy group content	~15%	~20%
Extractives content		
Non-polar (e.g. terpenes)	High	Low
Polar (e.g. tannins)	Low	High

Wood dust is also characterised by its moisture content: dry wood (moisture content less than about 15–20%) is less elastic than moist (green) wood. Woodworking operations with dry wood result in a larger volume of total dust and a higher percentage of inhalable dust particles. Wood dust stems from material that in many instances has undergone several kinds of treatment, such as disinfection, conservation, chemical staining and gluing (3, 4). However, the scope of the current literature search is on the raw material and not on substances added during the treatment of wood.

Wood dust is a complex substance. Its composition varies considerably according to the species of tree being processed. The main components of wood dust are:

- cellulose (approximately 40–50%);
- hemicellulose (also known as polyose);
- lignin;

- wood extractives (5–30%) – substances of lower relative molecular mass which may significantly affect the properties of the wood:
  - non-polar organic extractives (fatty acids, resin acids, waxes, alcohols, terpenes, sterols, steryl esters and glycerides);
  - polar organic extractives (tannins, flavonoids, quinones and lignans) and;
  - water-soluble extractives (carbohydrates, alkaloids, proteins and inorganic material) (3).

3.2 Physical-chemical properties

Table 3 Physical-chemical properties (NIOSH Pocket Guide to Chemical Hazards - Wood dust[1])

Properties	Value
State of the substance at normal temperature and pressure	Dust from various types of wood[1]
Melting/freezing point	Not applicable[1]
Boiling point	Not applicable[1]
Relative density	No information available
Vapour pressure	0 mmHg (approximately)[1]
Surface tension	Not applicable
Water solubility	Not applicable[1]
Partition coefficient n-octanol/water	Not applicable
Flash point	Not applicable[1]
Flammability	No information available
Explosive properties	No information available[1,2]
Self-ignition temperature	No information available
Oxidising properties	No information available
Granulometry	No information available
Stability in organic solvents and identity of relevant degradation products	No information available
Dissociation constant (pKa)	Not applicable
Viscosity	Not applicable

1 NIOSH Pocket Guide to Chemical Hazards – Wood dust. https://www.cdc.gov/niosh/npg/npgd0667.html (last viewed October 2019).
2 It is noted that sufficient care should be taken to avoid wood dust explosions: https://www.arboportaal.nl/onderwerpen/houtstof/welke-maatregelen-zijn-belangrijk-bij-het-werken-met-houtstof. https://connect.nen.nl/Standard/PopUpHtml?RNR=148301&search=&Native=1&tokeN=42f99b4e-8389-4922-a4a2-272020859c86#4.3Minimaleontstekingsenergievaneenstofwolk

The wood dust particle size depends on the type of process carried out on the wood; sawing produces larger particles than sanding. There is conflicting information whether the particle size also depends on the type of wood. For a given process, several studies report very similar particle size distributions between pine (softwood) and oak (hardwood), whereas others report that dust from hardwood is finer than dust from softwood (3). Several studies have shown that most of wood dust's mass consists of particles with aerodynamic diameters equal to or greater than 10 µm (2).

4 Occurrence, production and use

Occupational exposure to wood dust occurs all over the world, in a wide variety of wood-related industries. In the WOODEX study, Kauppinen et al. (2006) (6) estimated that the number of workers exposed to inhalable wood dust (from both hard- and softwood and including dust from chemically treated wood) in 2000–2003 in the European Union was highest in the construction industry (1.2 million), followed by the furniture industry (700,000), builders' carpentry industry (300,000), sawmilling (200,000) and forestry (150,000). The exposure assessment procedure integrated labour force data, company survey data, country questionnaire data and exposure measurement data. Dermal exposure was excluded from their study. There were an additional 700,000 exposed workers in miscellaneous industries in which carpenters, joiners and other woodworkers were employed. Using data from WOODEX, the highest exposure levels (>5mg/m^3) were found in the construction and furniture industries in Europe (for more information on the measurements performed in WOODEX, see Chapter 6 'Occupational exposure data', Table 5).

Tiessink et al. (2009) (7) further report that in the Netherlands approximately 360,000 workers are exposed to wood dust. Industries in which exposure to wood dust constitutes an important risk for employees (approximately 100,000 workers) in the Netherlands are furniture, carpentry, timber trading and wood processing. In addition, in the construction industry approximately 135,000 workers are potentially exposed to wood dust. It is not described in Tiessink et al. (2009) (7) in which industries the remaining approximately 125,000 workers are exposed to wood dust.

5 Measurement and analysis of workplace exposure

5.1 Environmental exposure monitoring

The potential adverse health effects of exposure to airborne wood particles depends both on the toxicity of the wood particles and on the size of the inhaled particles (particle aerodynamic diameter[1]). The latter determines the place of penetration in the respiratory tract. There is an international convention on three health-related aerosol fractions for measurement of dust particles in the workplace: inhalable, thoracic and respirable, which is presented in figure 1 (8, 9). The smaller the particles the deeper they can penetrate the respiratory tract.

Internationally, in the case of wood dust the *inhalable* fraction is sampled (e.g. in the UK, Netherlands, Germany and elsewhere in the EU (10–13)). This fraction covers all particles that may enter the nose and mouth. This is important since wood dust is known to cause health effects in the upper airways, which are related to particles smaller than 100 µm that end up in this part of the respiratory tract. See also figure 1.

The *respirable* fraction is the portion of inhalable particles that enter the deepest part of the lung, the nonciliated alveoli (D50[2] = 4 µm) (see Figure 1).

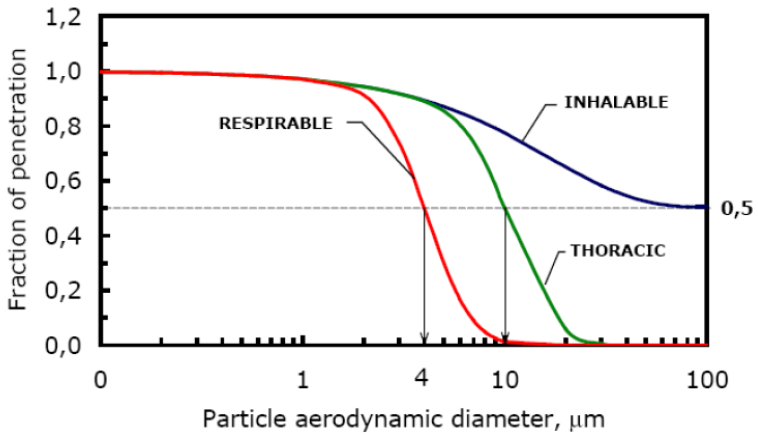

Figure 1 Probability of aerosol penetration as a function of aerodynamic diameter, internationally agreed by CEN/ISO/ACGHI (13)

5.2 Inhalable dust sampling

The inhalable dust concentration is usually measured using active sampling, meaning that the air is actively drawn over a sampler containing a preconditioned filter via tubing using a pump. The sampling pump must be calibrated before and after sampling, and the sampling flow must meet the requirements set by the sampler so that the inlet air speed meets the conditions of the sampler. The sampling volume is the

1 Particle aerodynamic diameter: the diameter of a sphere of density 1 g cm-3 with the same terminal velocity due to gravitational force in calm air as the particle, under the prevailing conditions of temperature, pressure and relative humidity (NEN-EN 481).
2 D50: = particle diameter corresponding to 50% sampling efficiency.

product of the sampling flow and the duration of the measurement (13). Passive sampling of wood dust was reported by Vinzents (1996) (14) and Schlünssen et al. (2001) (15). This method collects wood dust on sticky transparent foils by diffusion. The amount of dust is determined by light extinction. The concentration of dust cannot be calculated (since the sampling volume is not known), and therefore this method must always be calibrated with the conventional active sampling method (14).

Personal air sampling is the standard for comparison with an occupational exposure limit (OEL) (16). Dust samplers for personal monitoring should comply with the internationally agreed convention on inhalable dust to enable the comparison of results of different studies and to check compliance with an OEL (8).

Collecting inhalable wood dust is common in many countries. However, many different inhalable dust samplers are used. Sánchez Jiménez et al. (2011) (13) published a review of monitoring methods for inhalable wood dust. Table 4 presents an overview of inhalable dust samplers and whether or not they meet the international inhalable dust convention. Table 4 was largely based on Sánchez Jiménez et al. (2011) (13). In the US, a method for sampling inhalable dust was published by NIOSH (17). All tested inhalable dust samplers meet the inhalable dust convention with the exception of the 37-mm cassette (both open-face and closed). The 37-mm cassette samplers measure the so-called ‘total dust fraction’, which is defined by the sampler itself and not by the inhalable dust convention (18). However, there are critical aspects to be mentioned on all samplers; e.g. external wind may result in under-sampling using PAS-6 and PERSPEC, and particles larger than 100 µm are over-sampled when using the IOM sampler. Both the PAS-6 and IOM samplers are used in the Netherlands for collecting wood dust (13).

Table 4 Overview of dust samplers in use in mainly EU Member States

Type of sampler	Sampling flow	Particles collected	Meets CEN/ISO/ ACGIH curve (see figure 1) for inhalable dust?	Comments	Countries
37-mm cassette (closed)	2.0 L/min	Only on filter	No	Lower dust concentra-tions than 37-mm cassette (open). Large particles not effectively collected	Present: US: NIOSH Past: France, Sweden, Denmark
37-mm cassette (open-face)	2.0 L/min	Only on filter	No	Higher dust concentra-tions compared with 37-mm cassette (closed)	Present: unknown Past: Spain, Finland
Multi-orifice (seven-hole) sampler	2.0 L/min	Only on filter	Yes	May exhibit bias in some workplace conditions	UK, HSE
IOM Inhalable sampling head	2.0 L/min	On both filter and internal surface of the sampler	Yes	Over-samples large particles (>100 um)	UK, HSE Finland Nether-lands
Button*	4.0 L/min	Filter close to inlet, so minimal trans-mission losses	Yes	Low level Particulate Matter (PM) sampling	Unknown
GSP Conical Inhalable Sampler (CIS)	3.5 L/min	?	Yes	May exhibit bias in some workplace conditions	Germany, UK, HSE
CIP10 sampler version 2	10.0 L/min	No inner wall losses	Yes	CIP 10 version 1 showed some inner wall losses	France
PAS-6	2.0 L/min	Only on filter	Yes	External winds of 1 m/s result in under-sampling	The Netherlands
PERSPEC sampler	2.0 L/min	On both filter and internal walls of the inlet nozzle	Yes	External winds of ≥ 0,5 m/s result in under-sampling	Italy (no longer commer-cially available)
RespiCon multi-stage particle sampler **	3.11 L/min	Only on filter	Yes	Collects inhalable, thoracic and respirable dust at the same time	Unknown

* Button: SKC: https://www.skcltd.com/products2/sampling-heads/button-sampler.html
** RespiCon: TSI: http://www.a-a.co.kr/pdf/TSI-8522.pdf

Comparison between different samplers
A few studies were performed on the interchangeability of different samplers in the case of wood dust. SCOEL presented a table with conversion factors for the most common personal sampler instruments used in the EU to fit with the inhalable curve (2). This table was based on wind tunnel experiments (19).

Although total dust was examined in the past, it is no longer considered to be a reliable method of measuring most relevant particles regarding health effects (20). Different types of filter cassettes are used for measuring total dust and inhalable dust. Calibration studies have shown that inhalable wood dust concentrations are on average 1.6 to 4 times higher than total dust concentrations due to the use of different filter cassettes (20).

Respirable dust sampling
The respirable wood dust fraction is regularly sampled as part of research. Just as for measuring the inhalable fraction, measuring the respirable fraction must comply with the respirable convention (see Figure 1). Respirable dust is measured by active sampling using a pump connected to a wearable cyclone (i.e. the sampler). Many different cyclones are available for respirable dust measurements (21).

Analysis of inhalable dust samples
After sampling, the filter is reconditioned to remove excess moisture, after which the mass of inhalable dust is determined gravimetrically. The concentration of inhalable dust is determined by dividing the mass of inhalable dust by the sampling volume (16, 17, 22).

Dust samplers collect airborne particles in the workplace atmosphere independent of their composition. Since the method used to determine the inhalable dust concentration is based on a gravimetric method, this may lead to overestimation of the inhalable wood dust concentration. A method based on spectroscopic analysis of the cellulose content has been developed that is very specific since cellulose is present only in the wood dust particles. The method uses diffuse reflectance infrared Fourier-transform spectroscopy (DRIFTS) (23, 24).

To date there is no standard analytical method for distinguishing between hardwood or softwood dust. Identification of hardwood dust may be based on tannin content as hardwood dusts such as oak or mahogany have a higher tannin concentration than softwoods (e.g. pine, spruce, fir) (13). The total tannin content cannot be used to identify the hardwood type since the tannin content also differs within wood species (13).

Gallic acid (3,4,5-trihydroxybenzoic acid) and ellagic acid (2,3,7,8-Tetrahydroxychromeno[5,4,3-cde]chromene-5,10-dione) have been reported as good indicators for oak dust. Oak, chestnut and acacia have also shown high peaks of gallic, vanillic, ellagic and syringaldehyde acid, but not softwood species such as fir and spruce (13, 25). Carrieri et al. (2014) (26) developed a method for identifying teak wood dust via analysis of lapachol and deoxylapachol.

5.3 Biological exposure monitoring

Gallic acid in nasal lavage can be used as a marker to estimate oak dust exposure (27).

6 Occupational exposure data

6.1 Factors determining wood dust exposure

Routes of occupational exposure are inhalation, dermal contact and eye contact. The most important route of exposure is considered to be inhalation.

The inhaled concentration of wood dust during work activities depends on various factors. The **type of wood** may affect the amount of wood dust generated and thus the level of exposure (28). For instance, sanding of medium-density fibreboard (MDF) generates more wood dust than sanding of natural wood (1, 7, 29). In contrast to sanding, sawing MDF and sawing natural wood did not result in different inhalable wood dust concentrations (29). Thermally modified wood creates a larger amount of fine dust than similar but unmodified wood (30).

The **type of processing** also affects the amount of dust generated during work activities. For instance, large amounts of wood dust are generated when applying high-energy processes such as sanding, sawing, milling, drilling and shaving (7). In addition, handheld machines for sawing, sanding and milling as well as manual sanding result in relatively high wood dust concentrations (7).

Cleaning of the workplace, machines and products with compressed air is also considered to be an important cause of wood dust exposure. Concentrations of >20 mg/m^3 in the air can occur. Dry sweeping is also known to result in high concentrations of wood dust in the air (7). In addition, bagging dust from dust extraction systems can result in high wood dust exposure (31).

The availability and enforcement of **personal protective equipment and collective control measurements** also affect exposure to wood dust (3). Exposure to wood dust may have been higher in the past because of less efficient (or non-existent) local exhaust ventilation or other measures to control dust (3). In general, as a result of better collective control measures, lower exposure to wood dust is found in large compared to small factories (e.g. 32).

6.2 Occupational wood dust exposure levels in the Netherlands and Nordic countries

Although the focus in the current section is on the Netherlands and Nordic countries, studies on Europe as a whole are also summarised, as these may include data from the Netherlands and from Nordic countries. IARC (1, 3) and SCOEL (2003) (2) summarised exposure measurement data from a wide range of studies on both soft- and hardwood dust exposure in occupational settings. Since IARC published its findings in 2012, only relevant literature published in 2010 or later is summarised in this section, assuming IARC searched for literature published until at least 2010.

Two studies published after 2010 using data from Nordic countries, and two studies using European data, are summarised in Table 5. No Dutch study published in 2010 or later was found. Note, however, that two Dutch studies published before 2010 (36, 37), also included in the WOODEX study, had already been included in previous reviews; therefore, only limited data from these studies are summarised here (see Table 6). Another five studies published in 2010 or later in which occupational exposure data were reported for European countries other than The Netherlands or for Nordic countries are briefly summarised in Table 7.

Table 5 Occupational wood dust exposure concentration levels in Nordic countries or Europe reported in studies published ≥ 2010

General information	Exposure measurement data
Reference: Straumfors et al. (2020) (33), Straumfors et al. (2018) (34) **Country:** Norway **Type of industry:** sawmill industry (10 departments in 11 industrial sawmills, sorting, and planing companies) **Type of wood:** predominantly spruce or pine **Type of process:** sawing, sorting of green and dry timber, kiln drying, planing **Years:** 2013–2016	**Exposure measurement data:** GM (GSD) **thoracic** dust fraction: 0.09 mg/m^3 dust (2.6) (0.6%>2mg/m^3) GM (GSD) **inhalable** dust fraction: 0.72 mg/m^3 dust (2.6) (10%>2mg/m^3) Thoracic dust exposure was highest for maintenance workers (0.18 (2.8)) and workers sorting dry timber (0.14 (2.0)). Inhalable dust exposure was highest for workers involved in kiln drying (8.49 (11.5)) and sorting dry timber (0.69 (0.2)). Limit of detection: 0.023 mg for thoracic dust and 0.011 mg for inhalable dust. **Personal or stationary sampling and number of measurements:** In total, 2305 full shift (duration 170–642 min, median 513 min) personal samples were collected involving 1–6 repeated measurements (of 205 workers in 10 different departments). **Methods of exposure measurement and analysis:** The authors report that thoracic dust fraction samples were collected using BGI GK2.69 cyclones (BGI Inc., Waltham, MA, US) mounted with Millipore 37-mm sampling cassettes (Merck Life Sciences, Darmstadt, Germany) at a flow rate of 1.6 l/min^{-1}. Inhalable samples were collected using 37-mm conical inhalable sampling cassettes (CIS; Casella Solutions, Kempston, UK) at a flow rate of 3.5 l/min^{-1}. Samples for dust and resin acid analysis were collected using polyvinylchloride filters (PVC, pore size 5 µm, Merck). PC filters with a pore size of 0.8 µm were used for the thoracic fraction, whilst for inhalable samples the authors used a pore size of 1.0 µm to reduce the resistance across the filter and maintain the required high flow throughout the work shift. Gravimetric measurements were performed using a microbalance (Sartorius AG, MC 210p, Göttingen, Germany).
Reference: Eriksson et al. (2017) (32) **Country:** Sweden **Type of industry:** wood pellet industry (14 small, medium or large production units	**Exposure measurement data:** Inhalable wood dust fractions in: Personal samples: GM (GSD) = 0.62 (3.20) mg/m^3; Range = ≤0.10–47.2 mg/m^3 Stationary samples: GM (GSD) = 0.37 (4.40) mg/m^3; Range = ≤0.06–85.3 mg/m^3 In 2013, 37 samples out of 110 (34%) exceeded 0.5 mg/m^3 (N=13 exceeded 2mg/m^3)

General information	Exposure measurement data
based on annual production in tonnes) **Type of wood:** softwood **Type of process:** maintenance workers, production workers, bagging operators and loaders (details in column 2) **Years:** 2001, 2004, 2005, 2011, 2012, 2013	Inhalable wood dust concentration per occupation based on both personal and stationary sampling (In total, 617 inhalable dust measurements were available from 2001 to 2013, and all were used in the statistical analysis): Maintenance workers (various tasks, not specified): GM (GSD) = 0.90 (2.89) mg/m^3; Range = ≤0.10–32 mg/m^3 Production workers (oversaw the production process via computers in a control room, performed walk-through surveys in the different production areas within the premises, or cleaned areas or equipment contaminated by wood dust): GM (GSD) = 0.49 (4.26) mg/m^3; Range = ≤0.10–85 mg/m^3 Bagging operators (filled plastic sacks with wood pellets): GM (GSD) = 0.39 (2.41) mg/m^3; Range = ≤0.10–5.6 mg/m^3 Loaders (loaded sawdust or wood shavings into the production process or wood pellets onto trucks): GM (GSD) = 0.17 (1.68) mg/m^3; Range = ≤0.10–32 mg/m^3 No limit of detection reported. **Personal or stationary sampling and number of measurements:** 328 personal samples and 289 area samples were included in the analysis. **Methods of exposure measurement and analysis:** An open-faced 37-mm filter cassette was used in 2001 for personal sampling, and in 2001, 2004 and 2005 for area sampling. An IOM sampler was used for personal sampling in 2004 and 2005, while a GSP sampler was used for personal and area sampling in 2011, 2012 and in 2013. The result from the 37-mm open-faced cassette was multiplied by 2.5, as recommended by the Swedish Work Environment Authority, in order to make the exposure assessment comparable with the IOM and the GSP samplers. **Additional information:** Ventilation: Local exhaust ventilation was more common in 2011, 2012 and 2013 during sacking and in other parts of the premises where emission of wood dust occurred than in 2001, 2004 or 2005. General ventilation: considered satisfactory by employees and employers in all years. Cleaning: Sweeping with a hand-held brush, blowing with compressed air or sucking up with a vacuum cleaner were the most common ways of performing the cleaning task at all of the premises taking part in the study.

General information	Exposure measurement data
Reference: Vangronsveld et al. (2010) (35) **Country:** European countries (unknown which countries) **Type of industry:** composite wood products (CWP) plants **Type of wood:** CWP: oriented strand board (OSB), MDF, particle board (PB) and wood fibre insulation board (WFI) **Type of process:** production of CWPs (e.g. sawing) **Years:** 2004–2009	**Exposure measurement data:** Total inhalable particulates (TIP; assumed to comprise 100% wood dust), GM (GSD): A wide variety of functions were examined. For instance, the four **highest** GM exposures were found in: Dryer operators processing OSB (4 measurements): 0.93 mg/m^3 (not enough samples to calculate GSD) Cleaning OSB (40 measurements): 0.88 (3.26) mg/m^3 QC operator processing OSB (5 measurements): 0.89 (4.01) mg/m^3 The four **lowest** GM exposures were found in: Supervisor working with MDF (4 measurements): 0.15 mg/m^3 (not enough samples to calculate GSD) Press operator processing MDF (10 measurements): 0.25 (1.88) mg/m^3 Dryer operator processing MDF (1 measurement): 0.25 mg/m^3 (not enough samples to calculate GSD) Press operator processing OSB (33 measurements): 0.27 (6.82) mg/m^3 No limit of detection reported. **Personal or stationary sampling and number of measurements:** Personal and stationary measurements were performed at 9 CWP plants. Of the 446 pairs of samples collected for MDI and TIP, 283 pairs were personal samples measured over a five-year period (2004–2009) during 37 Industrial Hygiene (IH) surveys and the remaining 163 pairs were background area samples collected at key locations along the production line, measured in 2008/2009 during 15 IH surveys. **Methods of exposure measurement and analysis:** A known volume of workplace air was drawn through the sampling device (IOM sampler equipped with preconditioned, preweighed 25mm 0.8µm membrane filter) at a fixed sampling rate (2 L/min) and duration (according to UK HSE method MDHS 14/3) over a half or full work shift. Gravimetric analyses.
Reference: Kauppinen et al. (2006) (6) (description of WOODEX data) **Country:**	**Exposure measurement data:** In total, 3,600,000 workers were exposed to wood dust in the 15 European countries examined in 2000–2003. Level of exposure was estimated in total, by country and industry. 8-hour time-weighted GM concentration of inhalable fraction of wood dust: >5 mg/m^3: 560,000 workers (16% of all exposed workers examined)

General information	Exposure measurement data
15 European countries **Type of industry:** wood industries (industries in which predominantly mechanised processing of wood is carried out, e.g. sawmilling and planing of wood, impregnation of wood, manufacture of panels and boards and manufacture of furniture) and non-wood industries (industries in which workers hold typical woodworking occupations e.g. construction carpenter, woodworking machine operator and cabinet maker). **Type of wood:** pine, spruce, oak, beech, birch and other softwoods and hardwoods; includes chemically treated wood. **Type of process:** various (see 'Type of industry') **Years:** 2000–2003	>2 mg/m^3: 1.5 million workers (41% of all exposed workers examined) >1 mg/m^3: 2.2 million workers (62% of all exposed workers examined) <0.5 mg/m3: 750,000 workers (21% of all exposed workers examined) The total number of exposed workers varied by country, ranging from <3,000 in Luxembourg and Malta to 700,000 in Germany. Numbers of workers exposed to >2mg/m^3 in Denmark, Finland, The Netherlands and Sweden were: 21,000, 17,000, 70,000 and 18,000, respectively. The highest numbers of workers exposed to wood dust were found in the construction industry (1.2 million), furniture industry (700,000), builders' carpentry industry (300,000), sawmilling (200,000) and forestry (150,000). The highest numbers of workers exposed to 2–5 mg/m^3 were found in construction (388,000 workers), sawmilling (35,000) and forestry (34,000). The highest numbers of workers exposed to >5 mg/m^3 were found in construction (254,000 workers) and the furniture industry (87,000). No limit of detection reported. **Personal or stationary sampling and number of measurements:** There were 2,704 measurement results from Denmark, 1,230 from Finland, 7,881 from France, 20,872 from Germany, 389 from The Netherlands and 2,665 from the UK, totalling 35,760. For other countries a suitable proxy country was selected by the assessment team with the help of the national expert. Direct national data covering all exposure groups were not available for any country. The assessment team estimated the missing values based on their own experience in wood industry workplaces. **Methods of exposure measurement and analysis:** 8-hour time-weighted GM concentration of inhalable fraction of wood dust was estimated for a wide variety of jobs, industries and countries. The exposure assessment procedure integrated labour force data, company survey data, country questionnaire data and exposure measurement data.

Table 6 Occupational exposure concentration levels in The Netherlands (studies published before 2010)

Reference and country	(Geometric) mean wood dust concentration
Reference: Scheeper et al. (1995) (36) **Country:** The Netherlands **Type of wood:** unknown	GM inhalable wood dust concentration of 2.1 mg/m^3
Reference: Spee et al. (2007) (37) **Country:** The Netherlands (Dutch construction workers) **Type of wood:** unknown	GM inhalable wood dust concentration of 3.3 mg/m^3 (range 0.8–11.6 mg/m^3)

Table 7 Occupational exposure concentration levels in other European countries (studies published in 2010 or later)

Reference and country	(Geometric) mean wood dust concentration
Reference: Vanadziņš et al. (2010) (38) **Country:** Latvia **Type of wood:** pine, birch, oak	Mean occupational wood dust concentration: 1.0 to 9.6 mg/m^3 (38% exceeded the national permissible exposure limit of 6 mg/m^3)
Reference: Gioffré et al. (2012) (39) **Country:** Italy **Type of wood:** hardwood	Mean inhalable wood dust: 0.05 to 12.0 mg/m^3 in wood processing factories (sawmills and carpentries)
Reference: Magagnotti et al. (2013) (40) **Country:** Italy **Type of wood:** hard- and softwood	Mean exposure to inhalable wood dust of 1.75 mg/m^3 outside the cab of powerful chippers and of 0.57 mg/m^3 inside the cab. When using smaller machines without an enclosed cab, a mean wood dust exposure was found of 1.08 mg/m^3.
Reference: Reinhold et al. (2014) (41) **Country:** Estonia **Type of wood:** mainly birch, pine, spruce, juniper	Mean occupational wood dust exposure: 2.0 to 10.5 mg/m^3
Reference: Mater et al. (2016) (42) **Country:** France **Type of wood:** unknown	Median GM occupational wood dust exposure: 0.42 mg/m^3

7 Kinetics

This chapter describes the kinetics (absorption, distribution, metabolism and excretion) of exposure to wood dust. The most extensive description on the kinetics of wood dust is given in an evaluation performed by DECOS (1992) (4).
The summary below is based mainly on DECOS (1992) (4), supplemented by information taken from the evaluations by IARC (1, 3, 43) and more recently published data retrieved during the current literature search.

The focus of the kinetics of wood dust exposure is on its deposition and clearance in the respiratory tract. Little or no information is available on potential systemic absorption (1, 3, 4, 10, 43). There are indications of mutations in lymphocytes in peripheral blood in humans (see Chapter 8 'Effects in animals'), which may indicate systemic uptake. However, the cause of these effects is unclear (1, 44). In addition, it has been reported that ultrafine particles (particles with a diameter smaller than 100 nm) can penetrate the alveolar region and enter the blood circulation system (45). A significant fraction of inhaled (nano)particles can also become available for the oral route via the mucociliary escalator. Systemic findings in inhalation studies may therefore also be caused by uptake from the gastro-intestinal tract (45).

7.1 Humans

7.1.1 *Deposition*

For wood dust the process of deposition of the substance on the surface of the respiratory tract must be differentiated from the process of clearance (see Section 7.1.2). Deposition patterns for dusts depend not only on the particle size of the dust, but also on: the hygroscopicity, electrostatic properties and shape of the particles; the respiratory dynamics of the individual (e.g. oral or nasal breathing, frequency, depth, flow rate); and the anatomy of the respiratory tract. However, the factors of the respiratory dynamics are relatively constant in an individual (except for differences in nose / mouth breathing); thus the size and shape of the particles largely determine their deposition in the different areas of the respiratory tract. Different woodworking processes lead to different wood dust particle sizes, which will affect penetration and deposition in the respiratory tract.

The rather large variation in particle diameters causes wood particles to be deposited to a varying extent in airways and lungs, where they can elicit local responses, and from where they can be transported to the following tissues: maxillary and frontal sinuses, lung interstitium, hilar lymph nodes, and – via mucociliary transport and subsequent ingestion – the gastro-intestinal tract (4).

The filter capacity of mouth breathing is less effective than that of nasal breathing. For particle diameters of <5 µm the amount entering the trachea and the bronchial tree via mouth breathing was estimated to be about four times as large as the amount entering via nose breathing (4). In humans, wood particles with a diameter of >10 µm inhaled via the

nose are almost completely deposited in the nose. About 50% of particles with an aerodynamic diameter of about 2 µm are retained in the nose, and their deposition in the ciliated tracheobronchial region amounts to about 10% of the mass entering the trachea. Almost all particles below 1 µm diameter pass through the nose to the lower airways (4).

Hadfield (46) observed that in most of the examined subjects, wood dust is deposited in two areas of the nasal mucosa. The first is an ellipsoidal area measuring 1 x 0.5 cm on the anterior lower part of the nasal septum, and the second is on the anterior end of the middle turbinates, also measuring about 1 cm in length. In a few subjects – those with an absolutely straight septum – the dust is distributed equally on both sides. For all other subjects there was a greater deposition of dust on the side of the nose where the airway was more open (4).

7.1.2 *Clearance*

Elimination of dust particles is largely effected by the mucociliary system of the respiratory tract, which causes mucous secretion to move towards the larynx, aided by physiological reactions like coughing. The same mechanism acts in the extrathoracic region of the mouth, the pharynx and the sagittal part of the nose. Particles deposited in the anterior part of the nose are eliminated exteriorly. Little is known about the transportation, deposition and elimination of particles in the maxillary and frontal sinuses. Heavy exposure to wood dust may result in decreased mucociliary clearance and, sometimes, in mucostasis (3). This was confirmed in a recent study by Özler and Akoğlu (2020) (47), who found that the mean nasal mucociliary transit time (measured with the saccharin test) in 25 male wood industry workers was significantly longer (16.72 min ± 2.71) than in 25 age-matched male controls (12.28 min ± 1.98). It is, however, noted that this study report does not provide further details.

Particles deposited in the alveoli are phagocytised and transported into the lung interstitium or via the lymph vessels towards the hilar lymph nodes, where they are stored (4). No reports have been found on the fate of wood dust particles which remain on the mucosa for a long time (4). Coarse particles (2.5–10 µm) deposited in the nose are rapidly removed by sneezing, sniffing and mucociliary clearance. However, some areas in the nasopharynx lack cilia, and particles deposited in these regions have longer retention times, i.e. up to several days (4).

7.2 Animals

The number of animal experiments is limited and the deposition of wood dust in the respiratory tract was generally not measured (1). In one study, of one-year old female rats, clearance time and retention mass were measured (48). However, experimental procedures and results in this study were described in very limited detail (for further information, see Chapter 8 ‘Effects in animals’).

Overall, the relevance to humans of inhalation studies in animals with exposure to particles is unclear. The conditions of inhalation and the deposition of particles in the upper respiratory tract may be very different in animals and in humans.

8 Effects in animals

Summaries of all relevant *in vivo* studies on wood dust (including wood dust extracts, condensates and wood drying fumes) found in the current literature search and published in English before 11 May 2021 are presented in Tables 8–15.

8.1 Animal experiments, single exposure

Table 8 Respiratory tract effects after single exposure

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
Intratracheal instillation				
Bhattacharjee et al. (1979) (49)	Male guinea pigs N=8/group Intratracheal instillation 3 groups: - sheesham wood dust - mango wood dust - vehicle control	**Sheesham and mango wood dust** (Particle size: 71–75% <9.1 µm; 28% <4.5 µm) **Exposure levels:** 75 mg/animal (in 1.5 ml saline); average BW 300 g **Sacrifice:** 60 days (N=4/grp) or 90 days (N=4/grp) after instillation **Effect parameters:** histopathology of the lungs	**Lung histopathology:** Disintegration of giant cells, centrilobular emphysema and slight fibrosis in the lungs at both time points. Results described in text only.	Study focused on lung pathology and fibrosis. No guideline followed. Limited reporting of experimental conditions, procedures and results. Besides pathology results, no other data reported (on e.g. general toxicity). No incidences reported, no data presented on statistical significance.
Yuan et al. (1990) (50)	Male Wistar rats N=15/group Intratracheal instillation	**Hard wood dust** (no data on wood type or particle size) **Exposure levels:** 0 (saline) or 50 mg/animal (in 1 ml saline); BW range = 180–220 g (no average BW given) **Sacrifice:** 1, 3 or 6 months after instillation (number of animals/time point not given) **Effect parameters:**	**BAL cell differentials:** no significant exposure-related changes **BAL fibronectin** (ng/ml)**:** 1 month: 0: 34 (± 25) 50: 54 (± 28)* 3 months: not reported 6 months: 0: 14 (± 10) 50: 29 (± 15)* **Lung collagen** (mg/total lung)**:**	Study focused on inflammatory and fibrogenic changes in the lungs. No guideline followed. Limited reporting of experimental conditions, procedures and results. Pathology data reported only in wording, no data on controls. No data on wood type or particle size.

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
		cell differentials and fibronectin in BAL; lung collagen levels and histopathology	1 month: 0: 43 (± 7.6) 50: 67 (± 6.2)* 3 months: not reported 6 months: 0: 64 (± 12) 50: 121 (± 31)* * p<0.05 compared with controls **Lung histopathology:** (results described in text only, no incidences reported, time points not specified) 50: wood dust, cellular nodules, dust-laden giant cells and epithelioid cells found in alveolar space; negligible quantity of fine collagen fibres in or around the nodules	Statistical methods not specified.
Tátrai et al. (1995) (51)	Male Sprague-Dawley rats N=5/group Intratracheal instillation 4 groups: - pine wood dust - cellulose	**Pine wood dust** (respirable; no data on particle size), **cellulose**, and **fibre-free pine wood extract** **Exposure levels:** 0 (saline) or 15 mg/animal (in 1 ml saline) BW range = 230–260 g (no average BW given) **Sacrifice:**	**Histopathology:** (results shown in pictures or described in text only; no incidences or statistics reported) **Control animals:** no pathological changes in the lungs and lymph nodes **Pine wood extract:** no pathological changes in the lungs and lymph nodes	Study focused on lung pathology. No guideline followed. Limited reporting of experimental conditions, procedures and results. Pathology data reported only in wording or pictures, no incidences given. No

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
	- fibre-free pine wood extract - saline	1 week or 1 month after instillation (number of animals per time point not reported) **Effect parameters:** histopathology of the lung and regional lymph nodes	**Pine wood dust:** 1 week after exposure: - Lung: alveolar and interstitial edema with infiltration of neutrophils, lymphocytes, plasma cells and multinucleated giant cells which had fibre-like foreign bodies in the cytoplasm - Lymph nodes: mild sinus histiocytosis 1 month after exposure: - Lung: granulomatous inflammation, thickened interstitium with lymphocytic foci, increase of argyrophilic fibres in interalveolar septa and lumina of bronchioli and alveoli - Lymph nodes: non-specific sinus histiocytosis **Cellulose:** Similar effects as pine wood dust	statistical analysis reported. No data on particle size. Authors noted uptake of fibre-like material by multinucleated giant cells after exposure to pine wood dust and cellulose.
Oropharyngeal aspiration				
Sisler et al. (2019) (52)	Male C57BL/6 J mice N=10–12/group Oropharyngeal aspiration	**Yellow pine dust** ($PM_{2.5}$ fraction) suspended in saline **Exposure levels:**	**Histopathology (0 and 280):** - Acute bronchiolitis and alveolitis (day 1; incidence): 0: 0/10	Study focused on inflammatory changes in the lungs. No guideline followed.

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
		0 (saline), 28, 140 or 280 µg/animal; average BW 22.65 g **Sacrifice:** 1 and 7 days after treatment for BALF analysis (all doses), or 1 and 84 days after treatment for histopathology (0 and 280 µg) **Effect parameters:** inflammatory parameters in BAL, histopathology of the lung, EDM (enhanced-darkfield microscopy) and microscopic analysis of wood dust in lung (0 and 280 µg) **Statistical analysis:** ANOVA (histopathology results were not statistically analysed)	280: 9/11 (mild to moderate) - Inflammatory changes in the lung (day 84): 0: 0/12 280: 1/12 (minimal) - Lung fibrosis: no effects **Deposition and clearance (280 µg):** Wood dust particles found widely distributed in the alveolar region at day 1. At day 84, the majority of particles were cleared from the lungs (no quantitative analysis performed). **BAL:** no significant exposure-related changes	The results are described in limited detail; the presentation of statistical significance in figures is unclear. Authors noted uptake of wood dust particles by alveolar macrophages.

8.2 Animal experiments, short-term and subchronic exposure

Table 9 Respiratory tract effects after repeated exposure

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
Inhalation exposure				
Creutzenberg et al. (1994) (48)	Female F-344 Fischer rats (1-year old; no BW data given) Number of animals/group: unclear Intranasal instillation or inhalation exposure (nose-only) Single and repeated (9–10 times) exposure	Single exposure to (^{51}Cr)Fe_2O_3-labelled **beech dust** **Exposure levels:** Inhalation: 600 mg/m^3 for 10–15 min (particle size 4–5 µm MMAD), or **Intranasal instillation:** 50 µl 10% wood dust in saline into the right nasal cavity (particle size unknown) **Pretreatment:** with unlabelled wood dust (some of the animals, numbers not given): 9 intranasal doses of ~2.5 mg; or 10 1-hour inhalation exposures to 600 mg/m^3 wood dust (~1.5 mg per exposure) **Sacrifice:** no details reported **Effect parameters:** nasal retention and clearance, histopathology (not described in methods section)	**Histopathology:** inflammation of the nasal mucosa in all repeatedly exposed animals (no further details reported) **Nasal clearance half time** (average ±SD)**:** Instillation: 216 ± 180 min (single exposure); 415 ± 180 min (pretreated animals) Inhalation: 233 ± 52 min (single exposure); 276 ± 50 min (pretreated animals) **Retained mass** after single exposure**:** Instillation: 2.1 ± 0.7 mg Inhalation: 0.21 ± 0.09 mg	Study focused on nasal retention and clearance. No guideline followed. Limited reporting of experimental conditions, procedures and results – e.g. number of animals/group is unclear, details of the exposure regimen are missing, treatment of controls is unclear, histopathology only briefly mentioned in results (not in methods section). No statistical analysis performed.
Güney et al. (1987) (53)	Swiss Albino rats (sex not specified)	**Wood dust** (particle size 7–10 µm) from chairmaking industry **Exposure levels:**	Data reported in wording or shown in pictures only	Study focused on pathology of nasal and paranasal tissues.

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
	Wood dust: N=16 Controls: N=10 Inhalation exposure: 12 h/d, 7 d/wk for 4 months	not reported **Sacrifice:** after 4 months **Effect parameters:** Histopathology of the respiratory tract	**Wood dust:** - macroscopy: congestions and edema in all animals (affected tissues not specified) - microscopy: Nasal tissues: epithelial desquamation (40%)*, loss of cilia (50%), mucosal and submucosal congestion (87.5%), edema (75%), inflammation (~100%). Paranasal sinuses: similar, though less severe, changes as in nasal tissues Lower respiratory tract: not reported * % of animals showing lesions **Controls:** no (histo)pathological changes	No guideline followed. Limited reporting of experimental conditions, procedures and results – e.g. exposure not characterised (concentrations unknown, possible co-exposure to other substances; mode of exposure [whole body or nose only] not specified), no information on animal sex or husbandry, aerosol particle size was rather large. No statistical analysis performed.
Intranasal administration				
Määttä et al. (2006) (54)	Female BALB/c mice N=8/group Intranasal instillation	**Hard wood dust** (birch and oak; particle size ≤5 µm) **Exposure levels:** 0.5 or 50 µg per animal suspended in 50 µl PBS (no BW data reported)	Data shown in pictures or figures only. **Bronchial hyperreactivity:**	Mechanistic study to unravel wood dust induced pulmonary inflammation.

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
	2x/wk for 3 weeks	**Controls:** PBS (sham), LPS (20 pg/50 µl; the same LPS level as in birch dust), and titanium dioxide (TiO_2) **Sacrifice:** 1 day after last exposure **Effect parameters:** cytokine, chemokine and chemokine receptor gene expression in the lungs (mRNA induction), bronchial hyperreactivity to methacholine (at day 19), serum IgE and IgG2a, differential cell counts in BAL, histopathology of the lungs (at day 21) **Statistical analysis:** Mann-Whitney test, GraphPadPrism Software (p-values expressed relative to PBS-treated animals)	no significant change in response to wood dust exposure **Inflammatory cells:** 50 µg: increased influx of inflammatory cells in lung tissue and in BAL fluid (oak and birch dust) 0.5 µg: no response Sham control: no response **Cytokine expression:** 50 µg: increase in IL-1β, TNFα and TGF-β1 ($p<0.05$), effects strongest for oak dust 0.5 µg: no response Sham control: no response No response on Th1 and Th2 cytokines at any exposure **Chemokines:** 50 µg: increased (oak dust, $p\leq0.05$); variable outcome for birch dust depending on type of tested chemokine 0.5 µg: no response Control (TiO_2): variable outcome depending on type of tested chemokine	Authors noted uptake of wood dust particles by alveolar macrophages.
Määttä et al. (2007) (55)	Female BALB/c mice N=8-9/group	**Oak wood dust** (particle size ≤5 µm)	Data shown in pictures or figures only.	Study focused on immunomodulatory effects.

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
	Intranasal instillation 2x/wk for 3.5 weeks, in an ovalbumin (OVA)-asthma model	**Exposure levels:** 50 µg per animal suspended in 50 µl PBS (no BW data reported) **Controls:** PBS (sham), TiO_2 **OVA treatment:** i.p. sensitisation on days 1 and 11 (20 µg OVA with alum in 100 µl PBS), inhalation challenge with 1% OVA for 20 min on days 22–24. **Sacrifice:** day 25 **Effect parameters:** bronchial hyperreactivity to methacholine (at day 25), cytokine and chemokine expression in the lungs (mRNA induction), BAL analysis (cell differentials, IL-13, TNF-α, CCL3), histopathology of the lung, sereum OVA-specific IgG2a and IgE **Statistical analysis:** Mann-Whitney test, GraphPadPrism Software	**Bronchial hyperreactivity:** Decreased OVA-induced airway hyperreactivity after oak dust exposure ($p<0.05$, compared with OVA-treatment only). **Inflammatory cells**: Oak dust (- OVA)[$]: influx of inflammatory cells in lung tissue and in BAL fluid ($p<0.001$ compared with sham) Oak dust + OVA[$]: decreased influx of neutrophils in BAL ($p<0.01$ compared with oak dust only) **Cytokines and chemokines:** Oak dust (- OVA): increase in TNF-α ($p<0.001$), IL-1β ($p<0.05$), CCL3 ($p<0.001$ compared with sham); inhibition of OVA-induced IL-13 upregulation ($p<0.01$) Oak dust + OVA: increase in TNF-α ($p<0.01$ compared with OVA only) [$] '- OVA': nonallergic mice '+ OVA': allergic mice	

Table 10 Dermal effects after repeated exposure

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
Meding et al. (1996) (56)	Female Dunkin-Hartley guinea pigs N=15/group Dermal sensitisation study **Induction:** 4 closed epidermal applications on days 0, 2, 7 and 9 (and 2 injections of Freund's complete adjuvant on day 7) **Challenge:** Patches were removed after 24 h; reactions were assessed at 48 h and 72 h after application. Rechallenge after 4 weeks	**Jelutong dust** (hardwood grown in south-east Asia) extracted in acetone and dried **Exposure levels:** Induction: 20% jelutong in petrolatum (pet) Challenge: 0.2, 1 and 6 mg jelutong in acetone in empty Finn chambers; acetone was evaporated just before application. Animals were simultaneously challenged with 12.5%, 4%, 1% and 0.3% jelutong in pet, and rechallenged with 12.5%, 8%, 4%, 2%, 1%, 0.3% and 0.1% in pet. **Controls:** vehicle **Effect parameters:** The minimal criterion for a positive test reaction was confluent erythema. **Statistical analysis:** Fisher's exact test	Number of positive guinea pigs at 72 h after challenge (jelutong vs controls): 12.5% pet: 14/15*** vs 0/15 4% pet: 12/15*** vs 0/15 1% pet: 5/15 vs 1/15 0.3% pet: 3/15 vs 0/15 6 mg: 15/15*** vs 0/15 1 mg: 10/15*** vs 1/15 0.2 mg: 4/15* vs 0/15 Number of positive guinea pigs at 72 h after rechallenge (jelutong vs controls): 12.5% pet: 10/15*** vs 1/15 8% pet: 7/15* vs 1/15 4% pet: 7/15** vs 0/15 2% pet: 5/15* vs 0/15 1%: 3/15 vs 0/15 0.3%: 1/15 vs 0/15 0.1%: 1/15 vs 0/15 Fewer positives were observed 48 h after (re)challenge * p<0.05; ** p<0.01; *** p<0.001 compared with controls P for trend: not reported	Cumulative contact enhancement test, as described by Tsuchiya et al. (1985) (57). Study meets generally accepted scientific principles. Deficiencies: results reported in limited detail (e.g. no grading of responses reported, no individual animal data) no data on endpoints other than sensitisation

8.3 Animal experiments, chronic exposure, nonneoplastic effects

Table 11 Respiratory tract effects after chronic exposure

.	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
McMichael et al. (1983) (58)	Guinea pigs N=5/sex/group Inhalation exposure (whole-body) 0.5 h/d, 5 d/wk for 24 weeks 3 groups: one untreated control group, one sham-exposed control group, one group exposed to fir bark dust	**Fir bark dust** (particle size: 1.5 µm MMAD) **Exposure levels:** 0 (sham) and 1143 mg/m^3 **Sacrifice:** after 24 weeks **Effect parameters:** lung function, food and water intake, BW, (histo)pathology of the respiratory tract **Statistical analysis:** not reported	Data described in wording or shown in figures only. **BW, food and water intake:** no exposure-related changes **Lung function:** Slightly increased tidal volume and decreased breathing frequency during wood dust exposure, signs of respiratory distress (most evident after the exposure-free weekend) **Pathology:** Wood dust exposure: marked edema on the apex of the lungs, vascular granulation tissue on the pleural surface, lymphoid aggregates and perivascular inflammation in the lungs, thickened interalveolar septa (no incidences reported). Dust particles were found in alveolar spaces, phagocytised by septal cells, and isolated in connective tissue. **Controls:** no data reported	No guideline followed. Deficiencies: limited reporting of experimental conditions and results; small number of animals/sex/group; only one dose tested (no exposure-response data); -unusual exposure regimen (short daily exposure at extremely high concentration); -no statistical analysis performed.

8.4 Animal experiments, carcinogenicity

Table 12 Carcinogenicity – respiratory tract effects

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
Holmström et al. (1989) (59)	Female Sprague-Dawley rats N=16/group Inhalation exposure (whole-body) 6 h/d, 5 d/wk for 104 weeks	**Beech wood dust** (particle size: 70% ≤10 µm; 10–20% ≤5 µm) **Exposure levels:** 0 (control) and 25 mg/m^3 **Sacrifice:** after 104 weeks **Effect parameters:** mortality, BW, histopathology (nose, lung) **Statistical analysis:** Mann Whitney U-test and Fischer's exact test	**Mortality, BW:** no differences between exposed group and controls. Two animals died intercurrently (1 exposed, 1 control; not examined histologically). **Histopathology:** no tumours; no squamous metaplastic or dysplastic lesions in the respiratory tract Pulmonary emphysema (incidence): 0 mg/m^3: 0/15 25 mg/m^3: 5/15 ($p<0.05$ compared with controls) Non-respiratory tract tumours were found in 46-53% of the animals; the incidence did not differ between groups (details not reported).	No guideline followed. Deficiencies: • aerosol particle size was rather large; • -only one sex and one dose level tested (no exposure–response data); • small number of animals/group; • limited reporting of results; • inadequate reporting of tumours outside the respiratory tract.
Tanaka et al. (1991) (60)	Male Wistar rats N=15/group Inhalation exposure (whole-body)	**Beech wood dust** (MMAD: 7.2 µm; GSD: 2.2) **Exposure levels:**	**Growth, organ weight:** no significant differences between exposed group and controls **Histopathology:** no exposure-related neoplastic or other lesions observed	No guideline followed. Deficiencies: • aerosol particle size was rather large;

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
	6 h/d, 5 d/wk for 6 months	0 (control) or 15.3 (± 13.1) mg/m^3 **Sacrifice:** 18 months after the start of exposure **Effect parameters:** growth, organ weight (lung, liver, spleen, kidneys), histopathology (lung, liver, spleen, kidneys, larynx, trachea, bronchus, nasal tissues) **Statistical analysis:** not reported		• short exposure period; • small number of .animals/group • only one dose level tested (no exposure–response data) • only one sex tested • no details on statistical methods
Klein et al. (2001) (61), Klein et al. (2003) (62)	Female Fischer 344 rats Groups: Oak dust (N=60) Negative control (sham; N=115) Positive control (N=58) Inhalation exposure (whole-body) 4–5 h/d, 5 d/wk for 25 weeks (oak dust 24/60; sham 48/115; positive	**Oak wood dust** (particle size range 0.4–10 µm, with the majority 2-7 µm) **Exposure levels:** 0 (sham) or 18 mg/m^3 Positive control: 72 µg/m^3 N-nitrosodimethylamine (NDMA) **Sacrifice:** Wood dust: 9 rats (exposed for 25 weeks) were sacrificed after 26, 34 or 45 weeks (N=3 per time point), remaining	**Survival:** not significantly affected by wood dust exposure **BW:** no exposure-related changes **Tumour incidence:** Respiratory tract (without oral cavity): Adenocarcinoma in the lung: 0: 0/96 18: 1/51 NDMA: 0/46 Bronchial carcinoma: 0: 0/96 18: 1/51	No guideline followed. Deficiencies: • limited characterisation of exposure • aerosol particle size rather large • only one dose level tested (no exposure–response data) • only one sex tested • oak wood dust also contained up to 5 µg/m^3 of chromate (the total chromium

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
	control 23/58 animals) or lifespan (oak dust 36/60; sham 67/115; positive control 35/58 animals)	51 rats lived until their natural death. Sham: 19 sham-exposed rats were sacrificed after 26, 34 or 45 weeks (N=4–5 per time point), remaining 96 rats lived until their natural death. **Effect parameters:** survival, BW, histopathology, tumour incidence **Statistical analysis:** no details reported. Prematurely killed rats were excluded from evaluation (9 oak dust, 19 sham, 12 NDMA exposed rats)	NDMA: 0/46 Nasal cavity tumours: 0: 0/96 18: 0/51 NDMA: 14/46 (1 adenocarcinoma; 13 mucoepidermoid tumours) **Tumours outside respiratory tract:** no difference in incidence between oak dust- and sham-exposed animals	concentration was later determined to be 4 ng total Cr/m^3, Klein et al., 2003) • no details on statistical methods
Drettner et al. (1985) (63), Wilhelmsson et al. (1985a) (64), Wilhelmsson et al. (1985b) (65)	Male Syrian golden hamsters N=12/group (0 and 15 mg/m^3; study 1) or N=24 /group (0 and 30 mg/m^3; study 2)	**Beech wood dust** (particle size: 70% ≤10 µm; 10–20% ≤5 µm) **Exposure levels:** 0, 15 or 30 mg/m^3 **Sacrifice:**	**Histopathology** (incidence)**:** Study 1 (0 and 15 mg/m^3): no exposure-related neoplastic or other lesions observed Study 2 (0 and 30 mg/m^3): Nasal tumours: 0: 0/21	No guideline followed. Deficiencies: • short exposure period • small number of animals/group; • only one sex tested;

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
	Inhalation exposure (whole-body) 6 h/d, 5 d/wk for 36 weeks (0 and 15 mg/m^3) or for 40 weeks (0 and 30 mg/m^3)	after last exposure (36 or 40 weeks) **Effect parameters:** Histopathology (larynx, trachea, lungs, liver, kidneys, nasal tissues) **Statistical analysis:** chi-square and Fisher's exact test	30: 1/22 (malignant, but unclassifiable due to autolysis) Metaplasia/dysplasia of the nasal epithelium: 0: 0/19 30: 1/22 Nasal respiratory epithelium: mild inflammation of and stroma observed in 30 mg/m^3 group, not in controls (no incidences reported) Lower respiratory tract: no exposure-related lesions	• no data on endpoints other than pathology (e.g. survival, organ weight); • autolytic alterations complicated histopathological examination of (unspecified number of) animals that died spontaneously; • aerosol particle size rather large.

Table 13 Carcinogenicity – dermal effects

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
Mohtashamipur et al. (1989) (66)	Female NMRI mice N=70/group Skin application 2x/wk for 3 months	**Beech wood dust extract*** **Exposure levels:** 2.5, 5, 7.5 or 10 g equivalent dust/mouse (total dose per week; in 30 µl acetone on a 1-1.5 cm shaven area of the lower back) Negative controls (3 groups): acetone; shaved skin only; untreated (and unshaven) Positive controls (2 groups): 5 and 10 µg benzo[a]pyrene **BW:** 25–30 g (at purchase; no group averages reported) **Sacrifice:** Mice lived till their natural death, or were killed to prevent suffering. **Effect parameters:** histopathology of treated skin area **Statistical analysis:** Mann-Whitney U-test (distribution of life span and tumours) and chi-square test * Dust samples were extracted in methanol. Dried extracts were purified and eluted sequentially. Each fraction was tested for mutagenicity	**Survival:** not significantly affected by wood dust treatment (no further details reported) **Tumours:** A significant overall carcinogenic effect was observed in mice treated with wood dust extract when compared with negative controls ($p < 0.01$). Incidence and types of tumours are given in Table 14 below.	No guideline followed. Deficiencies: • only one sex tested; • short exposure period. The IARC Working Group also noted a dose-dependent increase in the incidence of skin squamous cell papillomas and carcinomas combined or papillomas alone (IARC 1995; IARC 2012).

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
		(see Mohtashamipur et al. 1986). The only fraction found to be mutagenic was the ethyl acetate phase, which was subsequently used for this carcinogenicity assay.		

Table 14 Tumour incidence in mice study reported by Mohtashamipur et al. (1989) (66), adapted from IARC (2012)

Tumour	**Extract (g)**				**Negative controls**			**Benzo[a]pyrene (µg)**	
	2.5 (N=43)	5 (N=50)	7.5 (N=46)	10 (N=49)	Acetone, shaven (N=42)	Shaven (N=44)	Untreated (N=43)	5 (N=43)	10 (N=42)
Skin squamous cell carcinoma	1	-	-	1**	-	-	-	1	15
Skin squamous cell papilloma	1	1	6	5**	-	-	-	2	5
Skin keratoacanthoma	-	-	1	-	-	-	-	-	2
Skin papillary cystadenoma	-	1	-	-	-	-	-	-	-
Sebaceous gland adenoma	-	-	-	-	-	-	-	2	-
Mammary gland adenocarcinoma	-	4	3	2	-	-	-	1	1
Mammary gland adenoacanthoma	-	-	-	1	-	-	-	-	-
Mammary gland mixed tumour	-	-	-	2	-	-	-	-	-
Fibrosacroma	-	-	1	-	-	-	-	-	-
Haemangiosarcoma	-	1	-	-	-	-	-	-	-
Neurofibrosarcoma	-	1	-	-	-	-	-	-	-
Lymphoma	-	-	-	1	-	-	-	-	-
Anaplastic carcinoma	-	1	-	-	-	-	-	-	-
Precancerous skin lesion[a]	2	4	8	6	2	1	-	13	18

Number of animals are effective numbers

[a] epithelial hyperplasia/hyperkeratosis

** P<0.01 for trend (Cochran-Armitage test), where comparisons are made for 0 (acetone, shaven), 2.5, 5, 7.5 and 10 g extract groups, including squamous cell carcinomas and papillomas combined, or papillomas alone

Table 15 Carcinogenicity – other effects

Reference	Animal species and exposure design	Data on exposure and effect endpoints	Results	Remarks
Pott et al. (1989) (67)	Female Wistar rats N = not reported i.p. injection, 3 times (timing of injections not specified) 52 rats were examined; this included surviving animals and animals that died 'relatively early, but excluding those lost through cannibalism'.	**Beech wood dust** (particle size not specified) **Exposure levels:** Total dose: ambiguously reported as 250 or 300 mg, suspended in saline (50 mg wood dust/ml; no BW data reported) **Controls:** saline only **Sacrifice:** 140 weeks after the first treatment **Effect parameters:** post mortem examination of the abdominal cavity and histopathology (extent of the investigation is unclear)	**Tumour incidence:** no mesothelioma or sarcoma in the abdominal cavity after treatment with wood dust (no further details reported)	No guideline followed. Deficiencies: • very limited reporting of experimental details, procedures and results • unclear how many animals were initially treated, how many died or were killed in bad health • exposure route not relevant to human exposure to wood dust

8.5 Animal experiments, reproductive and developmental effects

No data available.

9 Observations in humans

All relevant longitudinal epidemiological studies on the association between occupational wood dust exposure and health are summarised in Tables 16–20. Tables 17 and 18 each present several studies that used data from the same cohort. Within the tables, the studies are arranged by year of publication (from oldest to newest).

Other publications, mostly cross-sectional studies, are summarised in less detail in Appendix 3.

In Table 21 meta-analyses are summarised. No meta-analyses were performed in which exposure to wood dust was quantified by estimated or measured exposure information (mg/m^3).

9.1 Cohort studies, respiratory effects

Table 16 Prospective cohort studies – respiratory effects

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
Noertjojo et al. (1996) (68) Prospective cohort study Canada, Vancouver Sawmill industry **Follow-up:** 4–13 years (mean 7.5 years) **Inclusion criteria:** • participated in at least two surveys (surveys among exposed took place in 1982, 1983, 1984, 1988, 1993; surveys among controls took place at three-yearly intervals between 1978 and 1991); • had no asthma at either survey; • provided job history;	Wood dust 8-hour personal and area time-weighted average (TWA) in mg/m^3, Western red cedar (softwood), unknown whether wood was treated. Measured fraction not reported. **Exposure concentrations:** Geometric mean (SD) (mg/m^3): Office workers: 0 Sawmill workers: • low (<0.2): 0.13 (0.04); • medium (0.2-0.4): 0.30 (0.05); • high (>0.4): 0.61 (0.41). No significant differences in dust concentration across time for three exposure groups (low, medium and high).	**Health outcome:** lung function (FEV and FVC1) decline **Health assessment:** spirometry on the work site, according to American Thoracic Society. Unknown if performed before or after shift. **Statistical analyses:** multiple regression analyses **Covariates:** age (years), height (cm), baseline lung function, smoking status (current, ex- or non-smokers) and race (white, non-white)	Lung function change (ml/year), adjusted for covariates; regression coefficient (p-value): • $FEV_{1:}$ -12.1 ($p<0.01$); • FVC: -14.6 ($p<0.05$); • unadjusted data not reported. Same analyses, stratified according to level of wood dust exposure (low, medium, high); regression coefficient (p-value): $FEV_{1:}$ • high: -8.7 ($p\geq0.05$); • medium: -16.9 ($p<0.05$); • low: -9.7 ($p\geq0.05$); FVC:	**Selection bias:** Respondents who did not participate in ≥2 surveys had more respiratory symptoms and lower lung function. A **healthy-worker survivor effect** may therefore have affected the results (potentially resulting in a weaker association). A **healthy-smoker effect** was observed: current smokers in the high-exposure group had better lung function than non-smokers and ex-smokers at the onset of the study, suggesting that these smokers were healthier and tolerated both high exposure and smoking.	• Low dust concentrations are examined. • Western red cedar is examined. May result in asthma at lower concentrations than dust from other wood species. • Objective information on exposure to wood dust was used. • Exposure based partly on area sampling, not personal sampling. • Measured fraction not reported. Loss-to-follow-up: approximately 80% response at beginning of study and approximately 70% response at end of study

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
• smoking behaviour did not change during the study. **Study population:** 243 sawmill workers; 140 office workers from City Hall in Vancouver **Earlier publications on the same cohort:** Chan-Yeung et al. (1984) (69)	**Exposure assessment:** • cassettes with polyvinyl chloride membrane filters 37-mm in diameter with a pore size of 0.8 µm were used at a flow rate of 2L/min. Gravimetric analyses; • 916 personal and 216area samples, performed in 1982, 1983, 1984, 1988, 1993. **Daily exposure estimate for each job:** Each job since 1980 was assigned an estimated daily dust exposure based on the GM value of all dust measurements for that job. Area sampling was used only if personal sampling was not performed.		• high: -21.3 ($p<0.05$); • medium: -15.8 ($p<0.05$); • low: -10.9 ($p\geq0.05$).	Adjustments were, however, made for smoking. **Information bias:** Potentially imprecise exposure assessment and (nondifferential) misclassification bias because of formula used to calculate cumulative exposure (mean dust concentration job x duration job) **Recall bias:** Potential because of retrospective determination of job history through participants' self-reporting, potentially more so in individuals with low lung function **Confounding:** Co-exposures unknown	

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
	Daily cumulative dust exposure: multiplying the duration in each job by the mean dust concentration of each job over the period of study **Job history:** questionnaire administered by a trained interviewer				
Glindmeyer et al. (2008) (70) Prospective cohort study US: across all states **Woodworking processes:** • sawing, milling and sanding; • 10 large (work-force >300) wood processing facilities: 1 sawmill-planing-plywood, 1 plywood, 1 milling,	Both hardwood and softwood (% distribution assessed by type of facility) In several of the facilities treated wood was also processed. **Exposure assessment:** • 2,363 personal dust measurements using Respicon impactors; • Sampling time 4-8 hours; • 3 particle size fractions, plus determination of WS in dust (diffuse reflectance infrared	**Health outcome:** • lung function decline (FEV_1, FVC, FEV_1,/FVC and $FEF_{25–75}$); • respiratory symptoms (using repeated question-naires). **Health assessment:** For each participant at least 3 forced spirometry tests performed over the follow-up period. Only pre-broncho-dilator testing was done	Mean annual lung function decline per exposure level unit was obtained, broken down by: • 4 lung function parameters (FEV_1, FVC, FEV_1,/FVC and $FEF_{25–75}$); • 4 facility types (milling, sawmill-planing-plywood, plywood, furniture-cabinet); • 2 exposure variables (RPM) and WS; • 3 size fractions (extra-thoracic,	• Assessment of wood dust exposure using a JEM may have led to Berkson-type error (due to assignment of exposure at group level) but will probably not have resulted in a biased exposure–response slope. • (Uncontrolled) **confounding** is possible since no other workplace exposures than	• Inclusion and exclusion criteria for study participants unclear. • Date of study not provided. • An average follow-up period of ± 4 years may be short to detect major effects. • Inhalable dust exposure as such was not associated with lung function decline (results not presented).

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
3 cabinet and 4 furniture. **Follow-up:** 5 years **Inclusion criteria:** not further specified **Study population:** N=1,164 • no reference population; • total-amount of person-years approx. 3,000.	Fourier-transform spectroscopy); • 1,052 individual plant-department-job combinations collapsed into 184 groups with similar mean exposure; • wood dust differentiated into <u>extra-thoracic</u> (inhalable–thoracic), <u>tracheo-bronchial</u> (thoracic–respirable) and <u>respirable</u> size fractions. **JEM:** • Concentration estimates of WS and RPM form the basis for an industry-specific JEM. • For each individual the job history was linked to the JEM and mean exposure was calculated, weighted for duration of working in each similar exposure group.	**Statistical analyses:** Multivariable (two-stage) linear regression analysis with forward selection was done on annual decline of lung function parameters. **Covariates:** age, baseline lung function, sex, height, body-weight change and pack years of cigarettes smoked	tracheo-bronchial and respirable); • 2 adjustment strategies for pack-years smoked (lifetime cumulative pack-years and study packs per day). From these **192** analyses, details of <u>only the 14 statistically significant</u> ($p<0.05$) associations were quantitatively provided: • For <u>milling</u>: -21.4/-22.0 ml/year **FEV_1** per 0.1 mg/m^3 **respirable RPM** • For <u>sawmill planing plywood</u>: -23 ml/year **FEV_1** per 0.1 mg/m^3 **respirable RPM** • For <u>sawmill planing plywood</u>: -40 ml/year **FVC** per 0.1 mg/m^3 **respirable RPM**	wood dust were considered.	• Findings seem difficult (if not impossible) to extrapolate to inhalable dust levels. • Only significant associations were reported. • Multiple testing was done and apparently not accounted for in analyses and in interpretation. This may have resulted in spurious results, in particular since 'negative' findings were not presented. • Associations between exposures and respiratory symptoms were not reported.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
	Exposure concentrations: • Geometric mean across 10 facilities range = 0.8–2.5 mg/m^3 for inhalable, 0.2–0.5 mg/m^3 for thoracic and 0.1–0.2 mg/m^3 for respirable dust • AM concentration (mg/m^3) of WS per facility type: *Extra-thoracic* Furniture-cabinet: 0.90 Plywood: 0.02 Milling: 0.52 Sawmill-planing-plywood: 0.08 *Tracheo-bronchial* Furn.-cabinet: 0.45 Plywood: 0.03 Milling: 0.11 Sawmill-planing-plywood: 0.02 *Respirable* Furn.-cabinet: 0.072 Plywood: 0.005 Milling: 0.039		• For milling: -0.33 %/year **FEV_1/FVC** per 0.1 mg/m^3 **respirable RPM** • For sawmill planing plywood: +0.28/+0.29 %/year **FEV_1/FVC** per 0.1 mg/m^3 **respirable RPM** • For sawmill planing plywood: +11% /year **FEV_1/FVC** per 0.1 mg/m^3 **respirable WS** • For milling: -71/-73 ml/year **FEF_{25-75}** per 0.1 mg/m^3 **respirable RPM**		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
	Sawmill-planing-plywood: 0.005 • AM concentration (mg/m^3) of RPM per facility type: *Extra-thoracic* Furn.-cabinet: 1.12 Plywood: 0.58 Milling: 1.05 Sawmill-planing-plywood: 0.87 *Tracheo-bronchial* Furn.-cabinet: 0.02 Plywood: 0.24 Milling: 0.02 Sawmill-planing-plywood: 0.03 *Respirable* Furn.-cabinet: 0.23 Plywood: 0.16 Milling: 0.15 Sawmill-planing-plywood: 0.26				

Table 17 Prospective cohort studies – respiratory effects: summary of publications using the same cohort study data (71–73), starting with general information regarding the cohort study

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
General information cohort study used in Bolund et al. (2018) (71), Jacobsen et al. (2009) (72), Jacobsen et al. (2008) (73) • Prospective cohort study • Denmark • Furniture industry (54 factories) • Woodworking processes: cutting, sanding and assembly **Follow-up:** 6 years (study performed 1997–2005) **Inclusion criteria:** individuals participating at baseline and follow-up **Reference population:** workers from 3	• Personal 8-hour TWA measurements using passive dust monitors, previously calibrated against active sampling for inhalable dust with filters and gravimetric analysis • 2,217 measurements at baseline and 1,355 at the end of follow-up • Type of wood not specified, likely to be both hardwood and softwood; might involve treated wood **Exposure concentrations:** GM (GSD) overall exposure: • baseline: 0.9 (2.1) mg/m^3 • follow-up: 0.6 (1.6) mg/m^3 Median (range) cumulative exposure: • Men: 3.8 (0–7.6) mg/m^3-years	See study summaries below.	See study summaries below.	**Selection bias:** Those who left this industry were less likely to participate in the follow-up study (thus, potential healthy worker survivor bias). Women who left the industry had [i] more respiratory symptoms at baseline and [ii] a higher asthma incidence at follow-up. **Confounding:** Concurrent exposures in the furniture industry were not accounted for.	• Passive personal dust monitors were used. • Measurement results were converted into equivalent inhalable dust concentrations and used in all analyses.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
factories producing refrigerators or hearing aids (low organic dust exposure) **Earlier publications on the same cohort:** • Schlünssen et al. (2001) (15) • Schlünssen et al. (2004) (74) • Schlünssen et al. (2002) (75) • Schlünssen et al. (2004) (76) • Schlünssen et al. (2011) (77) • Jacobsen et al. (2013) (78)	• Women: 3.3 (0–6.9) mg/m^3-years **Exposure assessment:** Assignment of exposure level using an internal JEM, based on factory size and work tasks **Cumulative exposure:** assessed using job history (questionnaire), working duration and internal JEM				
Jacobsen et al. (2008) (73) See general information above. **Study population:** 1,112 woodworkers (927 men and 185 women) **Reference population:**	See general information above.	**Health outcome:** lung function **Health assessment:** • Pre-shift spirometric lung function testing (without bronchodilator testing) was	• Quartiles of cumulative dust exposure in mg/m^3 × year: Q1: 0–2.96 (ref) Q2: 2.97–3.74 Q3: 3.75–4.71 Q4: >4.71 • Regression coefficients for	See general information above. **Confounding:** Concurrent exposures in the furniture industry were not accounted for.	• Multiple testing was done and apparently not accounted for in analyses or interpretation. • A re-analysis of these data was published in 2018 (see

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
235 workers (104 men and 131 women) **Inclusion criteria:** acceptable lung function in both surveys		done, both at baseline and at the end of follow-up. • According to guidelines of European Respiratory Society • Absolute and relative changes in FEV_1 and FVC • Cumulative incidence of COPD: FEV_1/FVC<70 % **Statistical analyses:** • Associations between cumulative exposure (categories) and lung function changes were assessed by multiple linear	absolute change in ml/year: • FEV_1 decline in men Q2: -4.6 (p 0.2) Q3: -5.2 (p 0.2) Q4: -0.3 (p 0.9) • FEV_1 decline in women Q2: -1.8 (p 0.08) Q3: -14.5 (p 0.04) Q4: -25.0 (p 0.01) • FVC decline in men Q2: -8.2 (p 0.05) Q3: -4.3 (p 0.3) Q4: -2.1 (p 0.6) • FVC decline in women Q2: -3.4 (p 0.6) Q3: -18.3 (p 0.01) Q4: -14.8 (p 0.14) • OR of COPD incidence among men: Q2: 1.3 (0.6–3.0) Q3: 1.5 (0.7–3.2)		Bolund et al. (2018) (71)).

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
		regression models. • Associations between cumulative exposure (categories) and incidence of airways obstruction (FEV_1/FVC< 0.7) were assessed by multivariable logistic regression models. **Covariates:** Models were stratified by sex and smoking status and adjusted for age, height, and weight change.	Q4: 1.4 (0.6–3.2) • OR of COPD incidence among women: Q2: 1.5 (0.2–8.4) Q3: 3.3 (0.7–16) Q4: 3.9 (0.6–24) (p=0.08 for trend)		
Jacobsen et al. (2009) (72) See general information above. **Study population:** 1,377 woodworkers	See general information above.	**Health outcome:** asthma and lower respiratory tract symptoms **Health assessment:**	Associations between being a woodworker (vs. reference group) and cumulative incidence of 7	See general information above. **Reporting bias:** Self-reported symptoms may be overreported by	Difficult to interpret observed differences in risk estimates between men and women; authors speculate

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks

(1,137 men and 240 women)
Reference population:
297 workers (137 men and 160 women)
Inclusion criteria: symptom questionnaires completed in both surveys (numbers in specific analyses varied; depending on the number who were free of symptoms at baseline)
		• questionnaire, both at baseline and at the end of follow-up
		• Cumulative incidence of symptoms was studied.
		Statistical analyses:
		Associations between [i] woodworker vs reference or [ii] baseline exposure categories and incidence of each respiratory symptom were assessed by multivariable logistic regression analysis.
		
		Covariates:
		Models were stratified by sex and adjusted for smoking, age and
			respiratory symptoms:
			• Men: OR ranged between 0.73 and 1.90, not statistically significant.
			• Women: OR ranged between 0.36 and 8.85, statistically significant for (OR(CI)):
			  • Daily coughing: 2.8 (1.3–6.1)
			  • Hay fever: 0.36 (0.16–0.83)
			Analyses were repeated, re-defining the population at risk as those without all symptoms at baseline (instead of just the specific symptom under study).
			• Men: no major differences in OR
			• Women: risk estimates
				those with higher exposure at follow-up.	a higher susceptibility among women.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
		baseline hay fever (marker of atopy).	increased (OR(CI)): • Asthma symptoms: 11 (1.3–97) • Ever wheeze: 5.9 (1.2–30) • Daily coughing: 5.5 (1.9–16) Dose–response relationship with baseline dust exposure was found only for women: • Q1: <0.70 mg/m^3 (reference category) • Q2: 0.70–0.94 mg/m^3 • Q3: 0.95–1.32 mg/m^3 • Q4: >1.32 mg/m^3 Women (OR(CI)): • Daily coughing: Q2: 1.6 (0.6–4.3) Q3: 3.2 (0.9–6.8)		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
			Q4: 3.8 (1.5–9.7) • Chronic bronchitis: Q2: 2.3 (0.4–15) Q3: 3.0 (0.5–19) • Q4: 6.0 (1.2–29)		
Bolund et al. (2018) (71) See general information above. **Study population:** 1,112 woodworkers (927 men and 185 women) **Reference population:** 235 workers (104 men and 131 women) **Inclusion criteria:** acceptable lung function in both surveys	See general information above.	**Health outcome:** lung function (FEV_1, FVC) and COPD **Health assessment:** • Pre-shift spirometric lung function testing (without bronchodilator testing) was done, both at baseline and at the end of follow-up. • According to guidelines of European	Baseline exposure categories for COPD analysis (mg/m^3): • No (ref): 0 • Low: >0–≤0.972 • High: >0.972–≤1.61 OR of COPD incidence among male smokers: • Low: 0.94 (CI 0.3–3.0) • High: 0.66 (CI 0.2–2.2) OR of COPD incidence among female smokers: • Low: 5.49 (CI 0.6–49) • High: 8.47 (CI 0.9–82)	See general information above.	Please note that this is a re-analysis of previously published data (73). The most important differences are: • use of z-scores lung function based on GLI2012, instead of crude differences • definition of COPD using LLN instead of <70% fixed • both cumulative and

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
		Respiratory Society • Z-scores and LLN (the lower 5th percentile of a healthy non-smoking population) were calculated using GLI2012 (The Global Lung Function Initiative 2012 (GLI2012)) equations on the basis of age, height, sex and ethnicity. • Z-score changes of FEV_1, FVC and FEV_1/FVC at follow-up • Cumulative incidence of COPD: FEV_1/FVC<LLN **Statistical analyses:**	Cumulative exposure categories for COPD analysis (mg/m^3 × year): • No (ref): 0 • Low: >0–≤3.75 • High: >3.75–≤7.55 OR of COPD incidence among male smokers: • Low: 0.82 (CI 0.3–2.7) • High: 0.72 (CI 0.2–2.4) OR of COPD incidence among female smokers: • Low: 5.57 (CI 0.6–52) • High: 12.0 (CI 1.3–111) Baseline exposure categories for lung function analysis (mg/m^3): • No (ref): 0 • Low: >0–≤0.822		baseline wood dust exposure analysed • models additionally adjusted for amount of smoking and asthma at baseline

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
		• associations between cumulative exposure (categories) and longitudinal change in z-score for lung function: multiple linear regression models • associations between either baseline or cumulative exposure (categories) and incidence of new-onset COPD (FEV_1/FVC<LLN): multivariable logistic regression models **Covariates:**	• Medium: >0.822–≤1.144 • High: >1.144–≤1.61 Beta for ΔzFEV_1 among men in ml (p-value): • Low: 0.05 (0.42) • Med.: 0.08 (0.18) • High: 0.14 (0.04) Beta for ΔzFEV_1 among women in ml (p-value): • Low: -0.07 (0.40) • Med.: -0.21 (0.08) • High: -0.01 (0.97) Cumulative exposure categories for lung function analysis (mg/m^3 × year): • No (ref): 0 • Q1: >0–≤2.97 • Q2: >2.97–≤3.75 • Q3: >3.75–≤4.71 • Q4: >4.71–≤7.55 Beta for ΔzFEV_1 among men in ml (p-value):		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
		• stratified by sex and smoking status • adjusted for asthma at baseline, weight change and amount of smoking	• Q1: 0.09 (0.16) • Q2: 0.03 (0.67) • Q3: 0.05 (0.46) • Q4: 0.08 (0.17) Beta for $\Delta zFEV_1$ among women in ml (p-value): • Q1: 0.02 (0.82) • Q2: -0.05 (0.67) • Q3: -0.32 (0.009) • Q4: -0.31 (0.049)		

Table 18 Retrospective cohort studies – respiratory effects: summary of publications using the same cohort study data (79, 80), starting with general information regarding the cohort study

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
General information cohort study used in Vested et al. (2019) (79), Vested et al. (2021) (80) • Retrospective cohort study • Denmark, nationwide • Woodworking and farming industry **Inclusion criteria:** ever employed in farming or wood industry between Jan 1964 and Dec 2007	• Type of wood unknown • Inhalable wood dust 8-hour TWA in mg/m^3 • Includes chemically treated wood **Exposure information:** • wood dust exposure: WOODEX exposure database (quantitative organic dust IEM) (Kauppinen et al. (2006) (6); see also Table 5, Chapter 6 of the current report). (Estimates for the furniture industry were based on Danish estimates, whereas remaining estimates were established from measurements from several countries with	**Vital status:** based on the Civil Registration System	See study summaries below.	See study summaries below.	See study summaries below.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
	similar production circumstances.) • A 6% annual decline in wood dust exposure was assumed and calculated from 1975 and onwards. **Job history:** employment histories (also outside of farming or wood industry) from Danish Supplementary Pension Fund Register (SPF)				
Vested et al. (2019) (79) See general information above. **Follow-up:** • 1964–2013 • ever employed in farming or wood industry between Jan 1964 and Dec 2007 • COPD follow-up start: Jan 1997 when occupational	Occupational exposure to organic dust in farming or wood industry (see general information above) **Exposure concentrations:** Based on quartiles (mg/m^3-year): • Low (0.02–1.32) • Low-intermediate (>1.32–3.82) • Intermediate-high (>3.82–10.45) • High (>10.45–193.9)	**Health outcome:** COPD **Health assessment:** diagnosis of COPD derived from the Danish National Patient Register 1977–2013 (based on information from hospital records and not general practitioners)	**Exposure–response relationship:** Unadjusted association wood dust (quartiles; low=ref) and COPD, RR (95% CI): • Low-intermediate: 0.90 (0.80–1.02) • Intermediate-high: 0.86 (0.76–0.98) • High: 0.65 (0.57–0.74) Association wood dust (quartiles; low=ref) and COPD, adjusted for age, calendar year	• Lag of exposure (*healthy- worker survivor effect*) and left-truncation bias (by including only the inception population in analyses) were explored (see results section). • Smoking information was available only for a small	• The subanalyses were not performed separately for wood dust exposure only. • Observed inverse association may point to a healthy-worker bias. • Lagging of exposure and employment

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
wood or farming dust exposure <1997, otherwise Jan of the year following the year of first employment in wood or farming industry **Censoring:** Follow-up occurred until death, disappearance, emigration, first diagnosis of COPD or 31 December 2013. **Inclusion criteria:** born 1950 onwards **Exclusion criteria:** white-collar worker **Study population:**		One or more of the following diagnoses: • (ICD-10): emphysema (J43, J43.0, J43.1, J43.2, J43.8, J43.9) • other COPD (J44, J44.0, J44.1, J44.8, J44.9) • (ICD-8): bronchitis, unspecified (490), chronic bronchitis (491), emphysema (492) **Statistical analyses:** logistic regression analysis performed as a discrete survival function with person-years as the unit of analysis **Covariates:**	and gender, RR (95% CI): • Low-intermediate: 0.84 (0.75–0.95) • Intermediate -high: 0.75 (0.65–0.85) • High: 0.46 (0.40–0.53) Unadjusted association wood dust (continuous; mg/m^3-year) and COPD, RR (95% CI): 0.99 (0.99–0.99) Association wood dust (continuous; mg/m^3-year) and COPD, adjusted for age, calendar year and gender, RR (95% CI): 0.98 (0.98-0.99) **Subanalyses:** among workers in the farming or wood industry **Asthma censoring:** Censoring asthma (a potential precursor for COPD) cases did not alter the association	proportion of the total population and confounding by smoking cannot therefore be ruled out. • No adjustments were made for the use of treated wood. • Possible exposure misclassifica-tion: work outside of the farming or wood industry was assigned to zero exposure to organic dust.	duration were explored and did not appear to affect the observed association. • Smoking was more prevalent among those with lower organic dust exposure, suggesting a less healthy lifestyle and potential residual confounding. • COPD cases based on hospital records, so COPD may be underreported.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
• N=175,409 workers (N=2,716,631 person-years) • Reference category 'Low' (0.02–1.32 mg/m^3-year): N=679,130 person-years; 61% individuals solely exposed to wood dust • Total 3,162 COPD cases (incidence of 116 cases per 100,000 person-years) **Earlier publications on the same cohort:** • Kauppinen et al. (2006) (6) • Schlünssen et al. (2004) (81)		age, sex and calendar year. Smoking data were available for only a subpopulation, i.e. 2,121 exposed to wood dust and 1,915 exposed to farming organic dust).	(data not shown in article). **Analysis on left-truncation bias:** subset first employment in the farming or wood industry 1997–2007 (the 'inception population'): RRadj (95% CI), (quartiles; low (0.02–0.41 mg/m^3-year)=ref): 1.01 (0.81–1.27), 0.73 (0.57–0.93) and 0.56 (0.43–0.73) **Stratification by duration of employment:** RRadj (95% CI); quartiles; ref=no exposure (0 mg/m^3-year)): • 1 year (709,199 person-years): 1.00 (0.83–1.20); 0.81 (0.67–0.98); 0.90 (0.75–1.09) • 2 years (505,356 person-years): 1.19 (0.92–1.52);		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
			1.18 (0.92–1.51); 0.94 (0.73–1.21) • 3–6 years (793,201 person-years): 1.54 (1.22–1.93); 1.37 (1.09–1.72); 1.03 (0.81–1.30) • 7–14 years (505,925 person-years): 1.03 (0.78–1.36); 1.03 (0.78–1.36); 0.92 (0.70–1.20) • 15–40 years (202,950 person-years): 0.92 (0.61–1.36); 0.88 (0.60–1.31); 0.64 (0.43–0.95) **Stratification by duration of employment:** RRadj (95% CI) continuous organic dust exposure per mg/m^3-year: • 1 year (709,199 person-years): 0.97 (0.92–1.01)		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
			• 2 years (505,356 person-years): 0.98 (0.95–1.01) • 3–6 years (793,201 person-years): 0.98 (0.97–0.99) • 7–14 years (505,925 person-years): 0.99 (0.98–1.00) • 15–40 years (202,950 person-years): 0.99 (0.98–1.00) **Smoking adjustments:** • quartiles; ref=low (0.06–6.05 mg/m^3-year): RRadj (95% CI): 0.90 (0.43–1.89), 0.34 (0.11–1.02) and 0.73 (0.33–1.59) • continuous organic dust exposure per mg/m^3-year: RRadj (95% CI): 0.99 (0.97–1.02) **Lagged analyses:** (10 years); quartiles (reference category:		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
			No (0 mg/m^3-year)); RRadj (95% CI): 1.05 (0.94–1.16), 0.92 (0.83–1.02) and 0.63 (0.56–0.70) Adjustment for a lifetime history of **chronic disease-related hospitalisations** did not change the direction or magnitude of the observed inverse association (data not shown in article).		
Vested et al. (2021) (80) See general information above. **Follow-up:** after the year of initial asthma or COPD diagnosis (including A&E (accident & emergency) and other hospital visits) **Censoring:** censoring for	Previous year exposure to wood dust (see general information above) **Exposure concentrations:** median previous year exposure level (mg/m^3-year): • Low (>0–0.7) • High (>0.7)	**Health outcome:** hospital readmission for asthma and COPD **Health assessment:** • derived from the Danish National Patient Register 1997–2007. • COPD was defined according to	**Exposure–response relationship:** **Asthma** readmission; RR (95% CI); 0 mg/m^3=ref: >0–0.7 mg/m^3: • crude: 2.85 (1.60–5.09) • adjusted: 2.38 (1.23–4.60) >0.7 mg/m^3: • crude: 2.97 (1.66–5.32) • adjusted: 2.67 (1.35–5.26)	• Severity of COPD may have a large impact on work ability, possibly explaining the lack of association with COPD readmission (healthy-worker bias). • Analyses were not adjusted for smoking.	• Associations were observed for asthma readmission but not COPD readmission. • Job task information cannot be accounted for in IEM. • Possible exposure misclassifica-tion: work

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
death, emigration, loss to follow-up, 65 years of age, retirement or end of follow-up **Inclusion criteria:** • first hospital diagnosis of either asthma or COPD between 1997 and 2007 • after first diagnosis: blue-collar workers in wood industry • the majority of the reference group worked in other industries and a minority was unemployed. **Exclusion criteria:** white-collar worker **Study population:**		the International Classification of Diseases, 10th revision (ICD-10) as emphysema (J43, J43.0, J43.1, J43.2, J43.8, J43.9) or other chronic obstructive pulmonary disease (J44, J44.0, J44.1 J44.8, J44.9). • Asthma was defined by ICD-10 codes for asthma (J45, J45.0, J45.1, J45.8, J45.9) or status asthmaticus (J46, J46.9). **Statistical analyses:** logistic regression analyses with	**COPD** readmission; RR (95% CI); 0 mg/m^3=ref: >0–0.7 mg/m^3: • crude: 2.13 (0.92–4.94) • adjusted: 1.49 (0.52–4.28) >0.7 mg/m^3: • crude: 1.42 (0.59–3.41) • adjusted: 0.90 (0.30–2.75)	• No adjustments were made for the use of treated wood.	outside of the farming or wood industry was assigned to zero exposure to organic dust.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
• 769 asthma patients (3,777 person-years) • 342 COPD patients (1,369 person-years) **Earlier publications on the same cohort:** Vested et al. (2019): Vested et al. (2021) examined part of the cohort examined in Vested et al. (2019) but for a different health outcome.		person-years as the unit of analysis **Covariates:** • age (10-year categories), sex, years since first diagnosis, socioeconomic status and labour force participation* (yes/no) * Labour force participation was defined as a period, minimally 25% of the year, with no public benefit payment. All independent variables were lagged one year.			

9.2 Cohort studies, carcinogenic effects

Table 19 Retrospective cohort studies – carcinogenic effects

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
Siew et al. (2012) (82) • retrospective longitudinal cohort study • Finland (nationwide) • all industries **Follow-up:** 1971–1995 **Censoring:** person-years at date of emigration, death or until 31 December 2005 (see Pukkala et al. (2009) (83)) **Inclusion criteria:** all male participants in national population census on 31 December 1970 born between 1906 and 1945 and employed during	• estimated inhalable wood dust exposure • any tree species; solid wood, including bark; fresh and dried wood; wooden boards; chemically treated wood; unspecified wood dust. Cellulose pulp and paper dust not included. **Cumulative exposure estimates:** • Unexposed; • Low: 0.1–9.9 mg/m^3-years • High: ≥10 mg/m^3-years **Exposure assessment:** Exposure determined by Finnish JEM (FINJEM) (85) and quantified as 'inhalable concentration (mg/m^3) in workroom air': • based on exposure measurements,	**Health outcome:** nasal cancer, nasal squamous cell cancer, nasopharynx cancer, lung cancer **Health assessment:** • incident cases diagnosed between 1971 and 1995 obtained from Finnish Cancer Registry (FCR; coverage 99% of all malignant solid tumours diagnosed in Finland) • topography according to ICD-7 and for morphology to MOTNAC 1951 (83)	RR (95% CI) nasal cancer: • 0.1–9.9 mg/m^3-year 1.63 (0.85–3.11) • ≥10 mg/m^3-year 1.57 (0.98–2.52) • any exposure 1.59 (1.06–2.38) RR (95% CI) nasal squamous cell carcinoma: • 0.1–9.9 mg/m^3-year 1.94 (1.08–3.51) • ≥10 mg/m^3-year 2.06 (0.91–4.68) • any exposure 1.98 (1.19–3.31) RR (95% CI) lung cancer: • 0.1–9.9 mg/m^3-year 0.95 (0.87–1.03) • ≥10 mg/m^3-year 0.91 (0.84–0.98) • any exposure 0.93 (0.87–0.98) RR (95% CI) nasopharynx: • any exposure 0.66 (0.30–1.45)	• misclassifica-tion because of use of group-level data on exposure and confounders • exposure misclassifica-tion because jobs held in 1970 were used for whole follow-up period instead of complete job histories	• 20-year latency assumption, whereas 10 years in Siew et al. (2017) (84); no explanation provided • number of person-years for exposed and non-exposed not reported

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
1970 (for at least half of full working hours (see also (83)) **Study population:** 1.2 million men **Number of cases:** newly diagnosed nasal cancer 292, nasal squamous cell cancer 167, nasopharynx cancer 149, lung cancer 30,137 **Earlier publications on the same cohort:** • Pukkala et al. (2009) (83) provides more information on the cohort study. • Siew et al. (2017) (84) also used Finnish national population	hazard surveys, and assessments by industrial hygienists • 13 occupational groups potentially exposed to wood dust **Cumulative exposure:** • job held in 1970 (source: population census), assuming same job for 20–65 years of age • calculated for every 5-year birth cohort (from 1906–1910 until 1941–1945) and every 5-year calendar period of observation (from 1971–1975 until 1991–1995) • Exposure of each birth cohort was assumed to start in the year when the average age of the birth cohort was 20 and end in the year of the midpoint	**Statistical analyses:** • SIR per occupation group • RR per exposure group using Poisson regression • 20-year latency assumption **Covariates:** • adjustment using aggregated data for SES, age, period of follow-up, smoking, formaldehyde exposure • model for lung cancer further adjusted for occupational co-exposures to asbestos and silica dust	• exposure groups not reported SIRs (95% CI) nasal cancer: • timber workers (1 case): 2.03 (0.05–11.3) • sawmill workers (6 cases): 2.23 (0.82–4.85) • plywood, wooden board makers (2 cases): 2.55 (0.31–9.20) • construction carpenters (16 cases): 1.28 (0.73–2.07) • boat builders (2 cases): 4.25 (0.52–15.4) • bench carpenters (1 case): 0.85 (0.02–4.75) • cabinetmakers, joiners (1 case): 1.25 (0.03–6.98) • woodworking machine operators (1 case): 0.80 (0.02–4.47)		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
census, but Siew et al. (2012) (82): examined only a selection of the workers and applied a different design.	during the observation period, minus 20 years (latency). For instance, to calculate the cancer risk for the observation period 1981–1985 (midpoint 1983), only the exposures until 1963 were taken into account. • Proportion of exposed persons and mean exposure level for each occupation were used to calculate annual average exposure for each calendar year.		• woodworkers, not included elsewhere (2 cases): 7.34 (0.89–26.5) SIRs (95% CI) nasopharynx: • sawmill workers (1 case): 0.80 (0.02–4.48) • construction carpenters (5 cases): 0.91 (0.30–2.13) • bench carpenters (1 case): 1.65 (0.04–9.17) • woodworking machine operators (1 case): 1.69 (0.04–9.43) SIRs (95% CI) lung cancer: • upholsterers (38 cases): 1.14 (0.81–1.57) • timber workers (69cases): 1.21 (0.94–1.53) • sawmill workers (279 cases): 0.89 (0.79–1.00)		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
			• plywood, wooden board makers (82 cases): 0.96 (0.76–1.19) • floor layers (1 case): 0.42 (0.01–2.34) • construction carpenters (1,885 cases): 1.19 (1.14–1.25) • boat builders (53 cases): 0.94 (0.71–1.23) • bench carpenters (110 cases): 0.80 (0.66–0.96) • cabinetmakers, joiners (76 cases): 0.84 (0.66–1.05) • woodworking machine operators (122 cases): 0.86 (0.71–1.02) • wooden surface finishers (21 cases): 1.33 (0.83–2.04) • woodworkers not included elsewhere (30 cases): 0.96 (0.65–1.37)		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
			SIRs not reported for following occupational groups because no cases were observed: • Nasal cancer: upholsterers, floor layers, wooden surface finishers and char workers in wood industry • Nasopharynx: upholsterers, timber workers, plywood wooden board makers, floor layers, boat builders, cabinetmakers/ joiners, wooden surface finishers, woodworkers not included elsewhere and char workers wood industry • Lung cancer: char workers wood industry SIRs nasal squamous cell carcinoma not reported		

9.3 Case-control studies, carcinogenic effects

Table 20 Case-control studies – carcinogenic effects

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
Kauppinen et al. (1993) (86) • nested case–control study based on woodworkers cohort • Finland (nationwide) • woodworkers N=35 particleboard, plywood, sawmill, and formaldehyde glue plants **Follow-up:** 25 years **Study population:** Cohort of 7,307 male woodworkers (in analyses adjusted for smoking 35% were disregarded due to missing data on smoking) who worked ≥1	• wood dust exposure determined from JEM (estimated fraction (e.g. inhalable, respirable) not reported) • treated wood • mainly softwood such as pine and spruce, some hardwood **Exposure concentrations:** estimated mean level of exposure about 1 mg/m^3 (range not reported) **Exposure assessment:** Based on data from the cohort study used in the current study (86), a JEM was made specifically for wood dust per calendar year (88). The JEM was based on:	**Health outcome:** respiratory cancer: primary malignant neoplasms at sites with a possibility of direct epithelial contact with inhaled agents namely lungs and trachea (N=113), larynx, epiglottis (N=12), tongue (N=3), pharynx (N=2), mouth (N=1), nose and sinuses (N=1) **Health assessment:** FCR (1957–1982) (practically complete coverage across Finland) **Statistical analyses:** multiple conditional logistic regression analyses (in some	**Respiratory cancers (all types pooled)** (OR (90%CI)): Any wood dust exposure (at least 0.1mg/m^3 >1 month): • 1.05 (0.74–1.50) • Adjusted for smoking: 0.98 (0.60–1.61) Level of wood dust exposure: 0.1–1 mg/m^3: • 1.19 (0.74–1.89) • Adjusted for smoking: 0.86 (0.46–1.59) >1 mg/m^3 (unconditional logistic regression): • 0.64 (0.30–1.37) • Adjusted for smoking: 0.74 (0.22–2.56) Cumulative exposure: 0.01–5 mg/m^3-years: • 1.09 (0.69–1.74)	**Bias:** • Job histories outside of the plants were not considered, so possible underestima-tion of exposure and misclassifica-tion. • Less complete job information and exposures for cases (mostly deceased) compared with controls (mostly alive), possibly leading to differential misclassifica-tion. The bias was corrected by adjusting the ORs by survival status. **Confounding:** • Adjustments for co-exposures	• Analyses unadjusted for smoking were performed on a larger population than analyses adjusted for smoking. • Unknown whether characteristics such as exposure for the 35% without smoking information differed from those with smoking information. • Little information is available on how exposure measurements were performed (in

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
year between 1944 and 1965 **Number of cases and controls:** 136 cases diagnosed between 1957 and 1982 and 408 controls **Selection of controls:** alive and free from respiratory cancer at date of diagnosis of the corresponding case (3 controls per case were selected from within the cohort and matched by birth year) **Earlier publications on the same cohort:** Kauppinen et al. (1986) (87) (Kauppinen et al. (1993) (86) is an update of Kauppinen et al	• industrial hygiene measurements • walk-throughs in the plants • interviews of persons with long experience of circumstances of exposure in the plants • documents provided by the plants • job history from plant registers and in some cases through interviews **Cumulative exposure:** product of mean level and duration of exposure	analyses unconditional logistic regression analyses performed) **Covariates:** smoking status (<35 vs ≥35 years; available for 65% of the population), vital status **Additional analyses:** latency (induction period of 10 years of the disease) taken into account	• adjusted for smoking: 0.96 (0.48–1.92) >5mg/m^3-years: • 1.12 (0.75–1.68) • adjusted for smoking: 1.00 (0.58–1.71) **Upper respiratory cancer** (OR (90%CI)): Any wood dust exposure: • 1.18 (0.39–3.53) • Adjusted for smoking: 1.56 (0.26–9.33) **Lung cancer** (OR (90%CI)): Any wood dust exposure: • 0.68 (0.39–1.18) • Adjusted for smoking: 1.31 (0.82–2.10) (unconditional logistic regression) **Adenocarcinoma of the lung:** number of exposed cases too small (<4).	(treated wood) were not made. • Smoking data were available only for part of the study population (65%).	this or previous publications). • It is unknown which fraction (e.g. inhalable, respirable) is estimated. • A relatively low mean level of exposure to wood dust is estimated (range is, however, unknown).

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
(1986) with extended follow-up (from N=3,805 workers and 19 plants to N=7,307 workers and 35 plants))			Analyses for level of wood dust and cumulative wood dust were performed only for respiratory cancers (pooled). **Latency period taken into account:** **Respiratory cancers (all types pooled)** (OR (90%CI)): Any wood dust exposure: • 0.91 (0.65–1.28) • Adjusted for smoking: 0.97 (0.60–1.56) Level of wood dust exposure: 0.1–1 mg/m^3: • 0.99 (0.68–1.44) • Adjusted for smoking: 0.98 (0.57–1.68) >1 mg/m^3: • 0.87 (0.53–1.43) • Adjusted for smoking: • 0.83 (0.44–1.57) Cumulative exposure: 0.01–5 mg/m^3-years:		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
			• 0.84 (0.56–1.26) • Adjusted for smoking: 0.94 (0.52–1.69) >5mg/m^3-years: • 1.05 (0.69–1.60) • Adjusted for smoking: 1.09 (0.60–1.99) **Upper respiratory cancer** (OR (90%CI)): Any wood dust exposure: • 1.14 (0.38–3.37) • Adjusted for smoking: 0.60 (0.04–8.27) **Lung cancer** (OR (90%CI)): Any wood dust exposure: • 0.59 (0.35–0.99) • Adjusted for smoking: 0.44 (0.18–1.08)		
Vaughan et al. (2000) (89) • Population-based case–control study	• estimated personal exposure to total wood dust 8-hour TWA in mg/m^3	**Health outcome:** epithelial nasopharyngeal cancer into three histological ICD-O groups:	**All nasopharyngeal cancer subtypes**, ever exposed, OR (95% CI), without and with adjustment for formaldehyde:	Relatively low number of cases and controls in the highest two exposure categories.	• According to the authors, analyses were focused on differentiated squamous cell

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
• US, 5 regions: Connecticut, metropolitan Detroit, Iowa, Utah, western Washington **Follow-up:** 1987–1991 (1987–1993 in one region) **Inclusion criteria cases:** incident cases of epithelial nasopharyngeal cancer between 1 April 1987 and 30 June 1991 (at one registry up to 30 June 1993); men and women aged 18–74 **Inclusion criteria controls:** general population controls identified by random digit dialling, frequency matched to cases by age, sex and	• type of wood and treatment not reported **Exposure concentrations:** Cumulative exposure (mg/m^3-years) per category: • Unexposed: 125 cases; 220 controls • >0.0–2.75: 5 cases; 13 controls • >2.75–15.70: 10 cases; 5 controls • >15.70: 2 cases; 6 controls. Maximum (mg/m^3) lifetime exposure per category: • Unexposed: 125 cases; 220 controls • 0.0–0.55: 10 cases; 15 controls • >0.55–1.50: 6 cases; 4 controls • >1.50: 1 cases; 5 controls **Job history:** • assessed by structured telephone interviews	• epithelial NOS (not otherwise specified) (801x-804x) (N=24) • undifferentia-ted (8020, 8021, 8082) or non-keratinising (8072, 8073) (N=54) • squamous cell (805x-808x, except 8072, 8073) (N=118) **Health assessment:** Data on incidence and histological type from 5 population-based cancer registries (completeness of registry not reported) in the US participating in the National Cancer Institute's SEER programme.	• 1.3 (0.6–2.6) • 1.1 (0.5–2.3) **Squamous cell and epithelial NOS**, maximum exposure (mg/m^3), OR (95% CI), without and with adjustment for formaldehyde: • >0.0–0.55: 1.7 (0.7–4.4), 1.3 (0.5–3.6) • >0.55–1.50: 2.5 (0.6–10.2), 2.0 (0.5–8.1) • >1.50: 0.3 (0.0–3.2), 0.2 (0.0-2.1) • trend test p=0.76 and p=0.68 (without and with adjustment for formaldehyde, resp.) Cumulative exposure (mg/m^3-years), OR (95% CI) without and with adjustment for formaldehyde:	**Potential exposure misclassifica-tion:** • recall bias in self-reported occupational histories • telephone interviews to determine job history • lack of information on specific working conditions • proxy interviews (although exclusion of proxy interviews and adjustment for proxy status did not change the results substantially (data not presented)) **Potential residual confounding:** e.g. for uncontrolled lifestyle factors (diet), or infection	and epithelial NOS carcinomas because of differences in aetiology from undifferentia-ted and non-keratinising carcinomas of the nasopharynx.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
cancer registry area **Number of cases and controls:** 196 cases, 244 controls **Response rate:** cases 82%, controls 76% **Relevant references:** Description of data collection in Vaughan et al. (1996) (90)	• proxy interviews performed (usually with spouse) for 19% of cases and 1.2% of controls • questions referred to the period before the reference date (1 year before diagnosis for cases, 1 year before ascertainment for controls) **Exposure assessment:** evaluation and classification by industrial hygienist • based on occupation and industry code, self-report of occupational exposure by participants, and frequency and type of dust-generating tasks • jobs classified according to assigned 8-hour TWA levels: no or	**Statistical analyses:** logistic regression analyses focused on the group of 142 differentiated squamous cell and epithelial NOS carcinomas **Covariates:** adjusted for age, sex, race, SEER site, cigarette use (non-smokers, former/current smokers), alcohol intake, education, proxy status of interviews – with and without adjustment for formaldehyde exposure	• >0.0–2.75: 0.9 (0.3–3.0), 0.7 (0.2–2.5); • >2.75–15.70: 3.9 (1.2–12.5), 3.0 (0.9-9.8) • >15.70: 0.6 (0.1–3.7), 0.4 (0.1–2.3) • trend test p=0.42 p=0.99 (without and with adjustment for formaldehyde, resp.) Duration (years), OR (95%), without and with adjustment for formaldehyde: • 1–4: 1.5 (0.5–4.2), 1.4 (0.5-4.4) • 5–13: 1.4 (0.3–5.6), 1.3 (0.3–5.4) • ≥14: 1.8 (0.5–7.3), 0.8 (0.2–3.7) • trend test p=0.27 p=0.74 (without and with adjustment for formaldehyde, resp.)	with Epstein-Barr virus (which the authors state plays a more important part in non-keratinising and undifferentiated carcinomas) or occupational co-exposures.	

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks

	minimal exposure (<0.10 mg/m^3), low (>0.10 and <1.0 mg/m^3), moderate (>1.0 and <2.0 mg/m^3), and high (>2.0 mg/m^3)
	• individual exposure expressed as
	  • ever exposed
	  • maximum exposure over the lifetime
	  • duration (years)
	  • cumulative exposure: sum across all jobs of exposure concentration (using category midpoints) x duration of job
	• cut-off points cumulative exposure based on the 50th and 75th percentile among exposed controls				

Laforest et al. (2000) (91)	• Estimated (by JEM) 8-hour TWA wood dust exposure	**Health outcome:** incident primary laryngeal and	• Unadjusted data were not reported in article.	**Non-differential misclassifica-tion:**	• Power issue: most examined individuals

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
• case–control study • hospital-based • France (national) **Follow-up:** 25 years **Study population:** male patients with newly diagnosed larynx cancer cases selected from hospitals **Number of cases and controls:** • cases: 296 larynx and 201 hypopharynx cancer • controls, hospital-based: 296 men with newly diagnosed primary cancers requiring the same medical environment as the cases (e.g.	• Type of wood not reported. • Not reported whether wood was treated. **Number of cases and controls per cumulative exposure concentration category:** (larynx cancer / hypopharynx cancer / controls): • never exposed: 249/168/260 • low (<10 mg/m^3-years): 20/10/14 • medium (10-42 mg/m^3-years): 15/8/13 • high (>42 mg/m^3-years): 12/15/9 **Job history:** • job title and tasks held for ≥6 months, as determined by interviews performed by occupational physicians	hypopharyngeal squamous cell cancers diagnosed between 1 January 1989 and 30 April 1991 Because of the paucity of other histological types (less than 1%), only squamous cell cancers were considered. **Health assessment:** diagnosed and histologically confirmed in 15 French hospitals between 1 January 1989 and 30 April 1991. **Statistical analyses:** logistic regression **Covariates:** • adjusted for age (<60; ≥60), smoking (<30 pack-years; ≥30	• never exposed = reference category in all analyses **Cancer of the larynx**, OR (95% CI): Wood dust exposure: • Ever exposed: 1.00 (0.58–1.72) Probability of exposure: • ≤70%: 0.92 (0.44–1.95) • >70%: 1.08 (0.53–2.21) Duration of exposure (years): • <6: 1.41 (0.62–3.20) • 6-10: 0.49 (0.17-1.48) • >10: 1.03 (0.46–2.30) Cumulative wood dust exposure: • Low (<10 mg/m^3-years): 0.97 (0.44–2.14) • Medium (10–42 mg/m^3-years): 1.16 (0.49–2.75)	• JEM not based on measurements but on expert judgement of exposure to wood dust in the 1970s • JEM originally considered to be ordinal in nature and therefore not suitable to determine wood dust exposure quantitatively (93) **Bias:** Interviewers were aware of case–control status (possible interviewer bias) but unaware of study hypotheses. **Confounding:** Authors did not consider substances known or suspected to be carcinogenic to the larynx and pharynx, such as	were never exposed to wood dust. • The study initially identified 664 patients, meeting the inclusion criteria, but of these, 136 (20.5%) could not be interviewed (due to health problems, death, refusal to participate or because they could not be located). • Of the 355 controls identified, 50 (14%) could not be interviewed (due to medical conditions, refusal, or

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
gall bladder, prostate, brain and nervous system) **Selection of controls:** • by frequency matching on age • recruited between 1987 and 1991 in the same hospitals as the cases or in similar hospitals nearby **Earlier publications on the same study population:** Goldberg et al. (1997) (92)	• jobs coded with standard classification for occupation (ISCO) and industry (ISIC) **Exposure assessment:** • JEM developed by one of the investigators (93) by expert judgement, considering: type of woodworking operation, proximity to woodworking processes, workplace setting, freshness/dryness of wood • no industrial hygiene sampling data used in this JEM • jobs classified on basis of assumed 8-hour TWA exposures that might be expected to have occurred in the 1970s	pack-years), alcohol (0–4 glasses/day; ≥5 glasses/day), formaldehyde (same JEM), mineral fibres • ORs for hypopharynx were additionally adjusted for asbestos. • Level of education was not included as a potential confounder as it did not appear to be relevant.	• High (>42 mg/m^3-years): 0.86 (0.33–2.28) **Cancer of the hypopharynx**, additionally adjusted for asbestos, OR (95% CI)**:** Wood dust exposure: • Ever exposed: 0.89 (0.47–1.68) Probability of exposure: • ≤70%: 0.69 (0.28–1.76) • >70%: 1.06 (0.48–2.31) Duration of exposure (years): • <6: 0.53 (0.19–1.51) • 6–10: 1.00 (0.34–2.91) • >10: 1.19 (0.48–2.95) Cumulative wood dust exposure (mg/m^3-years): • <10: 0.56 (0.20–1.56) • 10–42: 0.74 (0.25–2.25)	sulphuric acid and polycyclic aromatic hydrocarbons.	because they could not be located).

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
	• classification into four categories: none, low (<1 mg/m^3), moderate (1–5 mg/m^3) and high (>5 mg/m^3) • For each ISCO-ISIC combination, the JEM gives the probability of exposure, and the level of exposure in categories. • To determine cumulative exposure, midpoints of the intervals were used.		• >42: 1.52 (0.59–3.94) **Additional analyses** according to laryngeal subsite (endo- and epilarynx), OR (95% CI)**:** Endolarynx, ever wood dust exposure: • 1.12 (0.62–2.03) Epilarynx, ever wood dust exposure: • 0.81 (0.38–1.75) Introducing an induction time (5, 10, 15 years) did not change results, as most exposures had started at the beginning of the working life (results not tabulated in article).		
Pesch et al. (2008) (94) • case–control study • Germany (nationwide) • woodworking industry **Follow-up:**	• inhalable wood dust from soft- and hardwoods (beech and oak) • treated wood **Exposure estimates:** • median of average over all years of	**Health outcome:** adenocarcinoma of the nasal cavity and paranasal sinuses (ADCN) **Health assessment:** • histopathologi-cally confirmed	OR (95% CI) high vs low (cut-off defined as 5 years based on duration x percentage of use of respective type of wood): • hardwood (i.e. beech and oak) 4.0 (1.9–8.3)	**Potential exposure misclassifica-tion:** • lack of information on specific working conditions • next of kin interviews (34%	• Low number of cases in the range <3.5 mg/m^3 (N=6) limits refining exposure categories and leads to

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks

• selection of cases from 1994 to 2003
• controls randomly recruited from a database of recognised cases with accidents on their way between home and workplace or fall accidents during shift
• controls individually matched to cases by year of birth (±2 years)
Inclusion criteria cases:
• Males
• histopathologi-cally confirmed adenocarcino-ma of the nasal cavity and paranasal
	employment exposure (IQR): cases 4.9 (4.4–5.7) mg/m^3; controls 3.8 (2.4–4.6) mg/m^3
	• median cumulative exposure over all years of employment (IQR):
	  • cases 159 (98–198) mg/m^3-years
	  • controls 109 (45–185) mg/m^3-years
	Job history:
	Occupational and exposure history assessed with structured questionnaire during interviews:
	• specific job activities in the wood industries
	• type of wood (hardwood, softwood, particle boards)
	• wood additives
		on basis of WHO classification of head and neck tumours
		• health data from nationwide insurance database of Holz-BG; complete coverage of eligible cases for those insured
		Statistical analyses:
		logistic regression analyses, conditional on age (<60 years, ≥60 years), adjusted for smoking (pack-years), age, 3 regions, interviewee (subject or next of kin), ever exposed to varnishes or stains (only in
			• softwood 0.3 (0.2–0.7)
			• particle board 0.5 (0.3–1.0)
			• MDF 0.3 (0.1–1.1)
			• similar ORs without adjustment for varnishes or stains
			OR (95% CI) average exposure:
			• <3.5 mg/m^3 (ref)
			• >3.5–<5 mg/m^3 10.54 (3.34–33.27)
			• >5 mg/m^3 48.47 (13.30–176.63)
			• Trend per mg/m^3 3.40 (2.19–5.28), p=0.0001
			OR (95% CI) cumulative exposure:
			• <140 mg/m^3-years (ref)
			• >140–<200 mg/m^3-years 1.72 (0.77–3.87)
			• >200 mg/m^3-years 4.20 (1.69–10.43)
			• trend per mg/m^3-years 1.01 (1.01–1.02), p=0.0001
			OR (95% CI) duration:
				for both cases and controls)
				• limitations inherent to use of a JEM
				Potential selection bias:
				cases who worked in the woodworking industries but were not insured or did not request compensation by Holz-BG not identified
					imprecision of risk estimates.
					• Inclusion of relatively highly exposed controls from the woodworking industries may lead to a conservative risk estimate compared with population-based studies.
					• Stratification of measurements (used for development JEM) by industry and year resulted in small numbers for calculating exposure in some years and industries in the JEM.
					• Not clear if year of

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks

sinuses (ADCN)
• ever employed in the wood industries
• recognised occupational disease between 1994 and 2003
• insured by Holz-Berufsgenos-senschaft (Holz-BG)
Inclusion criteria controls:
• men
• ever employed in the wood industries
Number of cases and controls:
86 cases,
204 controls
Response rate cases:
67% (57 cases and 29 next of kin from 129 identified cases)
	Exposure assessment:
	• cumulative exposure in woodworking industry calculated with five-year lag
	• JEM by type of industry based on:
	  • time-weighted shift averages of personal measurements of inhalable dust (in period 1986–2002)
	  • personal measurements of total dust in reconstructed historical workplaces
	• Exposure assessment refined by an expert-rated factor for each job title. This factor considered:
	  • The dustiness of the job activity
		analyses of quantitative exposure categories)
		Covariates:
		all covariates with 5-year lag
			• <20 years (ref)
			• 20–<40 years 1.38 (0.64–2.96)
			• ≥40 years 0.83 (0.35–1.97)
			• trend per year 1.00 (0.98–1.03), p=0.80
			OR (95% CI) time since first exposure:
			• <40 years (ref)
			• 50–<50 years 1.81 (0.69–4.70)
			• ≥50 years 2.81 (0.95–8.32)
			• trend per year 1.11 (1.04–1.19), p=0.002
			OR (95% CI) time since last exposure:
			• <1 year (ref)
			• 1–<8 years 3.13 (1.47–6.63)
			• ≥8 years 2.69 (1.25–5.79)
			• trend per year 1.04 (1.01–1.07), p=0.006
			ORs also reported but not shown here for raw wood, intermediary and
					diagnosis of disease corresponds to year of registration in the database of Holz-BG.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
Response rate controls: 75% (204 controls including 69 next of kin)	• dust extraction measures • working far or near field • maintenance operations • duration of exposure during a working shift		final processing, and separately for cabinet makers/joiners and saw millers		
Bhatti et al. (2011) (95) • case–control study • US (Washington state) • working activities in 7 wood-related industries • included woodworking as a hobby **Follow-up:** newly diagnosed lung cancer cases between May 1993 and July 1996 aged 18–74 and residing in	• Unknown but due to wide variety of occupations likely both hardwood and softwood • also likely that in several of the facilities treated wood was also processed **Exposure concentrations:** external database (levels not reported) **Exposure assessment:** algorithm based on personal TWA <u>total</u> wood dust measurements including determinants (e.g. duration spent in	**Health outcome:** newly diagnosed lung cancer, 6 histological groups: • carcinoma NOS • large cell • small cell • squamous • adeno-carcinoma • other **Health assessment:** identified through surveillance system (1993–1996) **Statistical analyses:** • multivariable unconditional	• OR (95% CI) for ever vs never having: • worked in logging, forestry, or tree farming industry: 0.9 (0.7–1.2) • worked in sawmill, planing mill or shingle mill: 1.5 (1.1–2.1) • worked in manufacturing plywood: 0.9 (0.6–1.4) • worked in manufacturing particle board: 0.8 (0.2–2.8)	**Bias:** • healthy-worker survivor bias unlikely (lifetime occupational history was considered) • Differential recall about wood-related exposures for cases and controls may have biased results (recall bias). **Confounding:** • endotoxin exposure (having a potential protective effect)	• Overall findings: Trend of lower odds of lung cancer among wood dust exposed; dose–response analyses did not reveal clear patterns. • Defining exposure groups based on quartiles resulted in relatively small numbers for some of the tasks.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
western Washington **Selection of controls:** Population-based controls identified by random dialling, matched to cases by 5-year age group **Numbers of cases and controls:** 440 cases and 845 controls (men only)	activity) and modifiers (e.g. distance to blade or type of tool) of exposure **Job history:** telephone interviews **Cumulative lifetime exposure (months×mg/m^3):** calculated for each participant based on woodworking jobs/activities and specific durations of employment	logistic regression analyses using exposure as ever vs never worked in a specific type of facility or as a hobby • using quartiles of cumulative exposure (based on controls only) and never woodworking (cumulative exposure 0) as reference; total and for 5 specific activities • Analyses were repeated for the specific histological subtypes. **Covariates:** Models were adjusted for age	• worked in pulp or paper mill: 0.8 (0.5–1.2) • worked as finish carpenter: 1.2 (0.8–1.7) • worked as general carpenter: 0.7 (0.5–1.0) • woodworking as a hobby: 0.8 (0.6–1.0) • main findings for quartiles of cumulative exposure for all woodworking activities (OR (95%): Q1: 0.9 (0.6–1.3) Q2: 0.7 (0.5–1.1) Q3: 0.8 (0.6–1.2) Q4: 0.9 (0.6–1.3) as compared with non-exposed • findings consistent (OR around 1; p>0.05) for dose–response analysis-specific activities	• other concurrent exposures in the workplace (but unlikely to explain inverse associations) • Residual confounding due to smoking amount is possible (but unlikely to explain the inverse associations when assuming that exposed participants smoked more).	

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
		(groups) and smoking status.	• no exposure-related trend found		
Siew et al. (2017) (84) • case–control study, register-based • countries: Norway (NO), Sweden (SE), Finland (FI), Iceland (IS) • study nested in NOCCA cohort (83) **Follow-up:** from the first available census (1960 in NO, SE, 1970 in FI, 1981 in IS) until the date of emigration, death or 31 December of 2003 (NO), 2004 (IS), 2005 (FI, SE) **Study population cases:**	• Occupational inhalable exposure to wood dust • freshly cut, dried, chemically treated wood • including bark from pine, spruce, birch, other softwoods and hardwoods (in Nordic countries mainly softwoods) **Exposure concentrations:** cumulative exposure categorised into low, moderate and high corresponding to the 50% and 90% percentiles of the cumulative exposures distribution among all exposed cases and controls: • high ≥ 28.82 mg/m^3 x years • moderate 6.71–28.81 mg/m^3 x years	**Health outcome:** cancer of the nose and sinuses (C30–31), nasopharynx (C11), adenocarcinoma (identified with histology codes used in the cancer registries in the four countries) **Health assessment:** linkage to cancer registries and national population registries **Statistical analyses:** HR estimated with conditional logistic regression, adjusted for formaldehyde exposure (reported only for adenocarcinoma)	HR (95% CI) nasal adenocarcinoma, unadjusted: • High: 28.86 (9.81–84.91) • Moderate: 11.69 (7.71–17.73) • Low: 3.16 (2.08–4.81) • None: ref HR (95% CI) nasal adenocarcinoma, adjusted for formaldehyde exposure: • High: 16.53 (5.05–54.08) • Moderate: 7.59 (4.38–13.13) • Low: 3.11 (2.04–4.75) • None: ref HR (95% CI) nasal cancer other than adenocarcinoma, unadjusted: • High: 1.00 (0.58–1.72)	**Misclassifica-tion:** • within occupational group because of the use of a JEM • job histories based on jobs at time of censuses **Confounding:** Potential residual confounding because analyses are unadjusted or adjusted only for formaldehyde exposure	Results for sensitivity analyses with 20-year latency assumption are not tabulated or reported in the article.

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
men diagnosed with specified cancers, minimum age 20 years at diagnosis and at least one census record prior to diagnosis **Study population controls:** randomly selected among persons who were alive and free from the cancers studied on the date of diagnosis of the cases, matched with cases for year of birth and country **Number of cases and controls:** • cases: nasal adenocarcino-ma 393, other nasal cancer 2,446, nasopharyn-	• low ≤6.70 mg/m^3 x years Median cumulative exposure: 6.71 mg/m^3 x years **Exposure assessment:** NOCCA JEMs (96): • For each country, a national JEM was constructed by a team of experts on the basis of the Finnish matrix (FINJEM, based on exposure measurements, hazard surveys, and assessments by industrial hygienists). • included jobs from woodworking and non-wood industry such as forestry, construction and building and repairing of ships and boats • >300 specific job titles, 29 exposure		• Moderate: 1.26 (0.96–1.65) • Low: 1.13 (0.92–1.38) • None: ref HR (95% CI) nasopharyngeal cancer, unadjusted: • High: 1.08 (0.52–2.24) • Moderate: 1.13 (0.82–1.57) • Low: 1.01 (0.84–1.38) • None: ref HR (95% CI) all nasal cancers, unadjusted: • High: 2.27 (1.54–3.35) • Moderate: 2.42 (1.97–2.97) • Low: 1.34 (1.12–1.60) • None: ref		

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
geal cancer 1,747 • controls: 5 male controls per case **Earlier publications on the same cohort:** • Pukkala et al. (2009) (83) • Siew et al. (2012) (82): also used Finnish national population census, but examined only a selection of the workers and applied a different design.	agents, 4 exposure periods: 1945–59, 1960–74, 1975–84, 1985–94 • proportion of exposed (P) and mean level of exposure (L) among the exposed persons in each specific occupation and time period characterised for each job title **Job history:** Occupational information from census records: • NO: 1960, 1970, 1980 • SE: 1960, 1970, 1980 and 1990 • FI: 1970, 1980, 1990 • IS: 1981 **Cumulative exposure:** for each case and control quantified as P x L x employment period in exposed occupation. • Cases and controls were assumed to				

Study design and population	Exposure assessment	Health assessment	Results	Bias/confounding	Remarks
	have worked from age 20 to 65 years. • If occupation changed between census records, change was assumed to have occurred in the middle of the period between census years. • 10-year latency period: exposures <10 years before diagnosis exempted				

9.4 Meta-analyses

Table 21 Meta-analyses: wood dust and health

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
Taskar and Coultas (2006) (97) includes 5 studies (98–102)	**Inclusion criteria:** not reported	**Overall analyses:** • idiopathic pulmonary disease, environmental and occupational wood dust exposure (5 studies), OR (95% CI): 1.94 (1.34–2.81) • unclear whether fixed or random-effects model was used	No test for heterogeneity and publication bias reported
Pérez-Ríos et al. (2010) (103) includes 19 studies: 12 case–control studies (74, 104–113), 3 cohort studies (114–116) and 4 mortality studies (117–120)	**Inclusion criteria:** • original data from case–control, cohort or standardised or proportional morbidity or mortality rate studies (SMR or PMR) • clear diagnostic criteria for asthma (either clinically diagnosed or self-reported through interview); asthma-like disorders not considered as asthma • explicitly described occupational exposure to wood • provided ORs or RRs and 95% CIs or enough data to calculate these figures • If data were duplicated in more than one study, only the most recent or detailed publication was included. • When a study provided data from multiple countries, the average risk of all countries was used.	**Overall analyses:** asthma, random-effects model, occupational wood dust exposure, RR (95% CI): 1.53, 1.25–1.87 **Subgroup analyses:** • asthma, random-effects model, occupational wood dust exposure, RR (95% CI): • cohort studies: 1.34 (1.01–1.78) • case–control studies: 1.74 (1.19–2.56) • population based: 1.37 (1.05–1.80) • hospital based: 4.56 (1.66–12.51) • PMR studies: 1.39 (0.91–2.13) • case–control + PMR studies: 1.63 (1.22–2.17) • incidence studies: 1.61 (1.26–2.05) • male only: 1.30 (1.12–1.51) • female only: 1.51 (1.33–1.71) • smoking adjusted: 1.19 (1.02–1.39)	**Test for heterogeneity, overall analyses:** • R (proportion of total variance due to between-study variance): 0.76 • Q test p-value: 0.0001 **Egger's test for publication bias:** • p=0.63

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
	Exclusion criteria: • cross-sectional studies, as such studies cannot provide useful information on a temporal relationship between the exposure and outcome • mortality studies analysing asthma as a cause of death, without specifying its ICD were excluded • studies that used asthma-like symptoms or other manifestations of respiratory impairment, such as wheezing and acute decline of lung function, as an outcome and did not establish a diagnosis of asthma • studies that dealt with sensitisation when it was used by the authors either as a proxy for exposure or as an outcome	• smoking non adjusted: 2.05 (1.41–2.98) • Caucasians: 1.59 (1.26–2.00) • Asians: 1.15 (0.92–1.44) • quality score <2.5: 1.63 (1.24–2.15) • quality score ‡2.5: 1.41 (1.13–1.76)	
Paget-Bailly et al. (2012) (121) includes 22 studies: 18 case–control studies (91, 122–138) and 4 cohort studies (93, 139–141)	**Inclusion criteria:** • occupational case–control and cohort studies • published in French and English • Overlapping studies were checked and the most recently published studies were included.	larynx cancer, random-effects model, occupational wood dust exposure, 22 studies, RR (95% CI): • wood dust (high)*: 0.95 (0.80–1.14) • wood dust (low)*: 0.95 (0.81–1.12) * The 'ever exposed' category was not available and impossible to infer from the reported data. For these studies, the authors used the extreme categories (low and high) of the exposure score and combined each in turn with the 'ever' category from the other studies.	**Test for heterogeneity:** • wood dust high: P <0.006, I^2 = 48.8%. • wood dust low: P <0.033, I^2 = 38.9%. **Egger's test for publication bias:** • wood dust high: p=0.08

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
			• wood dust low: p=0.082
Bolund et al. (2017) (142) includes 14 studies examining occupational organic dust exposure, of which there are 3 studies on wood dust exposure (68, 70, 73)	**Inclusion criteria:** • longitudinal/prospective cohort studies with a minimum of 1 year follow-up time • exposure: organic dust, endotoxin and/or other dust-associated exposures in different work settings (cotton dust, grain dust, farm dust, paper dust, wood dust) • outcome: longitudinal change in the lung function indices of forced expiratory volume in the 1st s (FEV1), forced vital capacity (FVC) and/or FEV1/FVC • study subjects: humans • language: English • full-length, original publications in peer-reviewed journals **Exclusion criteria:** • cross-sectional or cross-shift studies and follow-up time under 1 year • exposure: inorganic dust exposure or 'mixed' dust exposure without opportunity to separate organic and inorganic dust • lack of exposure gradient, such as exposed vs controls or without different levels of exposure among the exposed subjects • prognostic studies of specific patient groups • studies with <50 exposed subjects	**Overall analyses:** • long-term change in FEV_1, random-effects model, occupational organic dust exposure (i.e. cotton, grain, farm, paper and wood dust) (14 studies), OR (95% CI): -4.92 (-9.69 to -0.14) • long-term change in FVC, random-effects model, occupational organic dust exposure (i.e. cotton, grain, farm, paper and wood dust) (14 studies), OR (95% CI): -1.47 (-6.30 to 3.35) • No subgroup analyses were performed.	No test for heterogeneity or publication bias reported

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
	• lack of a quantitative estimate of association between lung function change and organic dust exposure • lack of adjustment for smoking • In order to avoid repeated cohorts inflating their effect on the review, only one of the articles was included.		
Alonso-Sardón et al. (2015) (143) includes 5 case–control studies (83, 144–147)	**Inclusion criteria:** • case–control studies published in peer-reviewed journals • description of occupational exposure to wood dust among cases and controls • diagnosis of sinonasal adenocarcinoma by biopsy	**Overall analyses:** • sinonasal adenocarcinoma, random-effects model, occupational wood dust exposure (no exposure = reference) (5 studies): OR=10.28, 95% CI=5.92–17.85 • no subgroup analyses	**Test for heterogeneity:** P <0.0001, I^2 = 85%
Binazzi et al. (2015) (148) includes 14 studies: 11 case–control studies (145, 147, 149–157) and 3 cohort studies (82, 139, 158)	**Inclusion criteria:** • articles published in peer reviewed journals • English language • epidemiologic studies published after 1985 • case–control or cohort design • studies involving humans (men or/and women) • including the sinonasal cancer subtypes adenocarcinomas and squamous cell carcinoma • referring to occupations and/or occupational settings with a possible risk of sinonasal cancer • (potential) exposure to specific risk factors stated explicitly, or an industrial/economic	**Overall analyses:** • sinonasal cancer, random-effects model, occupational wood dust exposure, including 11 case–control studies: RR=5.91, 95% CI=4.31–8.11 • sinonasal cancer, random-effects model, occupational wood dust exposure, including 3 cohort studies: RR=1.61, 95% CI=1.10–2.37 **Subgroup analyses:** • sinonasal cancer, random-effects model, occupational wood dust exposure by duration of exposure: • <15 years: RR=2.40, 95% CI=1.34–4.31	**Test for heterogeneity, overall analyses:** I-squared = 90.4%, p = 0.000 **Egger's test for publication bias:** =0.009

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
	activity recognised as having exposure to the risk factor (e.g. exposure to hexavalent chrome includes chromate production, stainless-steel welding, chrome pigment production, chrome plating, and ferrochrome production) • providing effect estimates with the corresponding measures of variability, or data permitting their calculation • exposure to wood dust including logging and sawmill working, pulp and paper industry, furniture industry, cabinetmaking, joinery and carpentry, woodworking machine operating, wood manufacturing, forestry **Exclusion criteria:** • no original results (reviews, letters, comments) • insufficient data (e.g. lack of information about the number of cases and controls or about the method used)	• ≥15 years: RR=9.19, 95% CI=5.84–14.46 According to histological type: • sinonasal adenocarcinomas, random-effects model, occupational wood dust exposure: RR=29.43, 95% CI=16.46–52.61 • squamous cell carcinoma, random-effects model, occupational wood dust exposure: RR=1.46, 95% CI=1.01–2.1	
Hancock et al. (2015) (159) includes 36 studies (82, 86, 95, 127, 134, 160–190)	**Inclusion criteria:** • contained an estimate of relative risk for lung cancer or data allowing such estimates to be calculated • contained a risk estimate related to a dichotomous index of exposure (ever vs never) or data allowing such estimates to be calculated	**Overall analyses:** lung cancer, random-effects model, occupational wood dust exposure (36 studies): RR=1.25, 95% CI=1.11–1.41 **Subgroup analyses:** lung cancer, random-effects model, occupational wood dust exposure:	**Test for heterogeneity, overall analyses:** P <0.01, I^2 = 82.1%.

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
	• contained an explicit analysis of wood dust as an exposure category at an individual, not occupational, level or contained an analysis of a wood dust-related occupation (e.g. woodworking, carpentry, furniture/cabinet making) • in English or Chinese	• outside of Nordic countries (mix of soft- and hardwood; N=33 studies): RR=1.34, 95% CI=1.19–1.50 • Nordic countries (predominantly softwood; 5 studies): RR=0.63, 95% CI=0.39–0.99 • studies adjusting for smoking: RR=1.31, 95% CI=1.10–1.56 • studies adjusting for smoking, and outside Nordic countries (19 studies): RR=1.48, 95% CI=1.29–1.69 • studies adjusting for smoking, and in Nordic countries (4 studies): RR=1.48, 95% CI=1.29–1.69 • self-reported data on exposure (19 studies): RR=1.29, 95% CI=1.15–1.45 • combination of methods to determine exposure (4 studies): RR=1.47, 95% CI=1.11–1.96 • job-exposure matrix to determine exposure (15 studies): RR=1.18, 95% CI=0.93–1.48 • self-reported data on exposure (12 studies), outside of Nordic countries and controlling for smoking status: RR=1.33, 95% CI=1.16–1.51 • Combination of methods to determine exposure, outside of Nordic countries and controlling for smoking status (5 studies): RR=1.76, 95% CI=1.29–2.42	**Egger's test for publication bias:** p=0.456

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
		• job-exposure matrix to determine exposure, outside of Nordic countries and controlling for smoking status (2 studies): RR=2.09, 95% CI=1.01–4.34 Histological subtypes: • squamous cell carcinomas (4 studies): RR= 1.54, 95% CI=1.24–1.92 • adenocarcinomas (7 studies): RR=1.32, 95% CI=1.08–1.60 • small-cell lung carcinomas (7 studies): RR=1.32, 95% CI=1.05–1.66 **Additional analysis:** wood dust-related occupations (59 studies): RR=1.15, 95% CI=1.07–1.23	

Beigzadeh et al. (2019) (191) includes 7 case–control studies (84, 89, 123, 192–195)	**Inclusion criteria:**
	• cohort or case–control studies
	• studies that reported the role of occupational exposure to wood dust in risk of nasopharyngeal cancer
	• studies that contained an estimate of OR, RR, ES, CI and information from which to derive these
	• original reports
	Exclusion criteria:
	• information on the studies not accessible or could not be extracted
	• reviews, case reports, conference, letters and animal studies
		Overall analyses:
		nasopharyngeal cancer (NPC), random effects model, occupational wood dust exposure (no=reference category), OR (95% CI): 1.5 (1.09–2.07)
		Subgroup analyses:
		• all fixed effects models, except for squamous cell carcinoma and adjusted analyses
		• Region (number of studies): OR (95% CI)
		  • Asia (N=4): 1.87 (1.39–2.53)
		  • Europe (N=1): 1.02 (0.85–1.23)
		  • US (N=2): 1.2 (0.57–2.52)
		• Histological subtype (number of studies): OR (95% CI):
			Test for heterogeneity: I^2=50.4%, P=0.06
			Egger's test for publication bias: P=0.073

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
	• exposure to wood dust was not occupational • studies not in English • studies that did not report the risk of outcome/s • abstract of studies	• squamous cell carcinoma (N=4): 1.68 (1.03–2.74) • nonkeratinizing carcinoma (N=1): 1.21 (0.51–2.85) • Adjusted (sex, age, smoking, diet, family history and formaldehyde exposure) vs crude results (number of studies): OR (95% CI): • adjusted (N=7): 1.5 (1.09–2.07) • crude (N=2): 2.14 (1.50–3.04) • Exposure assessment (number of studies): OR (95% CI): • qualitative exposure assessment (N=6): 1.5 (1.07–2.09) • a job-exposure matrix (N=1): 1.05 (0.87–1.28) • Year of exposure (number of studies): OR (95% CI): • >10 (N=3): 1.93 (1.42–2.62) • <10 (N=4): 1.35 (1.02–1.79) • Quality score of studies (number of studies): OR (95% CI): • >8 (N=3): 1.12 (0.84–1.48) • <8 (N=4): 2.00 (1.38–2.92)	
Zhang et al. (2019) (196) includes 14 case–control studies: 9	**Inclusion criteria:** • case–control studies • studies published in English	**Overall analyses:** asthma, fixed effects model, paper and/or wood dust exposure (no exposure = ref), OR (95% CI): 1.62 (1.38–1.90)	**Test for heterogeneity:** I^2 = 35%, P = 0.08

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
examined occupational wood dust exposure (108, 109, 112, 113, 197–201), 4 examined a combination of wood and paper dust exposure (104, 106, 107, 110) and 1 examined paper dust exposure (202).	• studies reporting the odds of asthma related to organic dust exposure with corresponding 95% CI • control group: obtained from either healthy community members (population-based) or inpatients without asthma (hospital-based)		No results for a test for publication bias reported

Meng et al. (2020) (203) includes 10 studies: 9 case–control studies (84, 122, 192, 194, 195, 204–207) and 1 cohort study (93)	**Inclusion criteria:**
	• randomised controlled trial, cohort studies and case–control studies
	• participants aged over 18 years
	• control group without any prior exposure to wood dust
	• definite diagnosis of NPC made by history and ICD-O
	
	Exclusion criteria:
	• missing information on exposure and outcome
		Overall analyses:
		nasopharyngeal cancer (NPC), random effects model, occupational wood dust exposure (no exposure = ref), OR (95% CI):
		2.18 (1.62–2.93)
		Subgroup analyses:
		• Design (number of studies), OR (95% CI):
		  • case–control (N=10): 2.04 (1.54–2.72)
		  • cohort study (N=1): 5.3 (1.70–12.4)
		• Exposure assessment (number of studies), OR (95% CI):
		  • questionnaire (N=8): 2.28 (1.77–2.93)
		  • JEM (N=3): 1.73 (1.15–2.61)
			Test for heterogeneity:
			I^2=43.0%, P=0.001
			Begg's and Egger's tests for publication bias:
			P=0.477, P=0.235

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias

	• studies using same dataset	• Geographic regions (number of studies), OR (95% CI):
		  • Asia (N=7): 2.24 (1.75–2.87)
		  • US (N=2): 2.24 (1.17–4.25)
		• Adjusted (gender, age, smoke, diet, family history and formaldehyde exposure) vs crude results (number of studies), OR (95% CI):
		  • adjusted (N=8): 2.17 (1.70–2.77)
		  • crude (N=3): 1.76 (1.11–2.78)	

Park et al. (2021) (208) included 6 case–control studies (209–214)	**Inclusion criteria:** • study on adult population over 18 years of age • IPF diagnosis criteria based on the clinician's judgement of the patient's symptoms, clinical findings and imaging findings (histological diagnosis may not have been included in the diagnosis) • categorisation of the surveyed jobs or occupational and environmental exposure factors that could lead to risk of IPF • survey methods for occupational and environmental exposure factors included any method that is systematic and planned, ranging from self-reporting by post or telephone to face-to-face surveys with experts **Exclusion criteria:**	**Overall analyses:** • IPF (not reported whether random or fixed model), occupational wood dust exposure (no exposure = reference category) (4 studies): OR=1.62, 95% CI=1.04–2.53 • IPF (not reported whether random or fixed model), carpentry and woodworkers (no exposure = ref) (4 studies): OR=1.56, 95% CI=0.87–2.82	**Tests for heterogeneity:** • occupational wood dust exposure: I^2=5.0%, P=0.37 • carpentry and wood workers: I^2=38%, P=0.19 Egger's test not performed

Reference of meta-analysis and included studies	Inclusion and exclusion criteria	Results	Heterogeneity score and publication bias
	• reviews, letters, editorials, case reports, studies on animals or children • theoretical studies on the medical system itself, revisions after the medical system were introduced • papers or papers not related to the research purpose • papers in languages other than English • studies that focused on known risk factors, such as asbestosis, coal worker's pneumoconiosis and silicosis		

10 Mode of action and pathogenesis

10.1 Genotoxicity and cancer

10.1.1 *Findings by IARC*

IARC (2012) (1) concluded that wood dust exposure can cause cancer of the nasal cavity, paranasal sinuses and nasopharynx in humans; there was weaker evidence for other cancer sites such as the pharynx, larynx and lung. Although IARC concluded that the mechanism responsible for the carcinogenicity of wood dust is unknown (1), it evaluated potential mechanisms, including: tissue injury induced by the deposition of wood dust particles in the sinonasal region; impaired ciliary clearance; and direct genotoxicity and indirect genotoxicity secondary to chronic inflammation. Wood dust may also act as a carrier for other genotoxic agents (e.g. chromate). Overall, IARC noted that there was weak evidence for these mechanisms in cellular assays, short-term animal assays, or assays for genotoxicity using peripheral blood cells or buccal epithelial cells obtained from workers exposed to wood dust.

Workers exposed to wood dust have increased frequencies of metaplasia and hyperplasia in nasal epithelial biopsies, although these alterations were not considered to be precursor lesions of neoplasia at this organ site (1).

With respect to the type of wood, IARC noted that the great majority of epidemiological studies did not report on the specific tree species to which workers were exposed or whether exposure was due primarily to hardwoods or softwoods. In an evaluation by the Health Council of the Netherlands, hardwood and softwood extracts and condensates were considered to be genotoxic *in vitro*, as indicated by the ability to cause gene mutations, chromosomal aberrations and/or SCEs (215). However, the Health Council of the Netherlands concluded that the *in vitro* genotoxicity was weak, that it was not confirmed by adequate *in vivo* testing, and that it is unknown to what extent the properties of these surrogates (i.e. extracts and condensates) represent those of the original material (215). Genotoxicity studies and other mechanistic (non-genotoxicity) *in vitro* studies are summarised in Tables 22–25.

10.1.2 *More recently published studies*

Since the publication of the IARC (2012) (1) report, two new *in vitro* studies have appeared that studied the mechanism of carcinogenicity.

Staffolani et al. (2015) (216) investigated the effects of hardwood (oak and padouk) and softwood (pine and fir) dusts (particle size: >60% were <1 µm) on DNA damage and repair in an *in vitro* model using BEAS-2B human bronchial epithelial cells. Wood dust exposure induced reactive oxygen species (ROS) formation and accumulation of oxidised DNA bases, which was associated with a delay in DNA repair activity. Long-term exposure resulted in soft-agar colony formation and induction of blood vessel formation. These initiated cells showed extensive autophagy and reduced DNA repair, which was associated with reduced OGG1 (8-oxoguanine glycosylase) expression and oxidised DNA base

accumulation. These events were found to be related to the activation of EGFR/AKT/mTOR pathway, through phosphorylation and subsequent inactivation of tuberin. Cellular uptake of wood dust was not investigated. The authors concluded that the persistence in the tissue of wood dusts, and their repetitious binding with EGFR (epidermal growth factor receptor), may continually trigger the activation switch, leading to chronic down-regulation of genes involved in DNA repair, leading to cell transformation and proliferation.

Wilson et al. (2015) (217) investigated the effects of extracts of hardwood (teak, walnut, mahogany, poplar and red oak) and softwood (yellow pine, cypress, spruce and cedar) dusts on human aryl hydrocarbon receptor (AhR) activation in a transgenic yeast strain. AhR is a ligand-activated transcription factor linked to a (non-)genotoxic mode of action of specific classes of chemicals. Teak dust extract was found to be the most potent inducer of AhR signalling, followed by walnut, mahogany and poplar; oak and softwood dust extracts were neither potent nor efficacious with respect to activation of AhR signalling. The authors identified 2-methylanthraquinone (2-MAQ), primarily present in teak dust, as a potent inducer of AhR signalling and concluded that 2-MAQ may mediate toxic effects through activation of AhR signalling in exposed woodworkers.

A number of tests with human material have also been published in recent years which may be helpful in elucidating (part of) the carcinogenic mechanism of wood dust.

Cellai et al. (2019) (218) investigated oxidative DNA damage by analysing M_1dG (3-(2-deoxy-β-D-erythro-pentafuranosyl)pyrimido[1,2-α]purin-10(3H)-one deoxyguanosine) DNA adducts in nasal epithelial cells of 136 woodworkers and 87 controls in Tuscany, Italy. The results showed a significant excess of M_1dG in woodworkers exposed to average levels of 1.48 mg/m^3 wood dust (8-hour TWA) relative to controls. The overall MR of M_1dG adduct levels between woodworkers and controls was 1.28 (95% CI 1.03–1.58). After stratification for smoking habits and occupational status (exposure to wood dust alone and co-exposure to VOCs), the association of M_1dG with wood dust (alone) was even greater in non-smoking workers, with an MR of 1.43 (95% CI 1.09–1.87). M_1dG was significantly associated with co-exposure to VOCs (MR of 1.95; 95% CI 1.46–2.61) and occupational history (i.e. years of exposure; MR of 2.47; 95% CI 1.67–3.62). The frequency of M_1dG was significantly correlated to the urinary excretion of 15-F_{2t} isoprostane, a biomarker for oxidant status. The authors concluded that their observation of an enhanced frequency of M_1dG adducts in cells of the respiratory tract of workers occupationally exposed to wood dust was consistent with a genotoxic mechanism, though other components could have contributed to the total adduct formation.

Wultsch et al. (2015) (219) investigated the number of micronuclei (indicative of genomic damage), nuclear buds (reflecting gene amplification), binucleated cells (caused by mitotic disturbances) and acute cytotoxicity parameters (pyknosis, karyorrhexis, condensed chromatin and karyolysis) in buccal and nasal cells of workers of a veneer factory (N=51) who were exposed to wood dust and VOCs, in

carpenters of a furniture factory which used no synthetic chemicals (N=38) and in a control group (N=65). Additionally, markers were measured in blood plasma which reflect inflammation (C-reactive protein, CRP) and redox status (malondialdehyde, MDA; oxidised low-density lipoproteins, oxLDL). Workers at the veneer factory (veneer contained 55% softwood and 45% hardwood) were exposed to an average level of 0.39 mg/m^3 inhalable wood dust (GM of measurements over 1 week) and were co-exposed to VOCs; furniture workers were exposed to an average level of 0.66 mg/m^3 inhalable wood dust (mainly spruce) and were co-exposed to organic glue. No induction of micronucleated cells was observed in either epithelium in the two exposure groups while all other nuclear anomalies except pyknosis were increased. MDA was also significantly elevated in workers from both factories. According to the authors, the results show that exposure to low levels of wood dust causes cytotoxic anomalies, but not the formation of micronuclei, whereas positive effects with respect to genotoxicity were obtained in a number of studies (several were also described in IARC (2012) (1) and IARC (1995) (3), where workers were exposed to higher wood dust levels (e.g. (220–225)).

Bruschweiler et al. (2014) (220) analysed the micronucleus (MN) frequency in nasal and buccal epithelial cells of 31 workers with a history of exposure to wood dust (≥5 years), and 19 non-exposed workers from Switzerland. The exposed group consisted of parquet layers, installers, carpenters and furniture workers (non- or ex-smokers) who were exposed to wood dust mainly from fir, spruce, beech, oak and wooden boards (which may have contained glue, formaldehyde and other chemicals) such as MDF and melamine. The mean inhalable dust concentration, determined for two consecutive 8-hour work shifts, was 2.9 mg/m^3. MN frequencies in nasal (OR of 3.1; 95% CI 1.8–5.1) and buccal cells (OR of 1.8; 95% CI 1.3–2.4) were significantly higher in the exposed group than in the non-exposed group, and also increased with exposure duration. The exposed group had higher frequencies of cells with nuclear buds, karyorrhectic, pyknotic and karyolytic cells and a decrease in the frequency of basal, binucleated and condensed cells compared with controls.

Bruschweiler et al. (2016) (44) used the comet assay to investigate DNA damage in peripheral blood samples in the same population of woodworkers and controls as studied by Bruschweiler et al. (2014) (220). In the 2016 study (44), however, the group of 31 exposed workers was regrouped according to the predominant wood type used: natural wood (such as untreated fir, spruce, beech and oak: 12 workers) and composite wood (such as MDF and other composites, which contain binders that can potentially emit carcinogenic substances: 19 workers)[3]. Gravimetrically determined average wood dust concentrations of the inhalable fraction were 3.6 mg/m^3 and 1.7 mg/m^3 for groups exposed to natural and composite wood, respectively. DNA damage was greater in the composite wood group than in both the group exposed to natural woods ($p<0.001$) and the unexposed controls ($p<0.001$). No difference in DNA damage was observed between workers exposed to natural

3 These two types of wood dust exposure (natural vs composite wood) were not specified in Bruschweiler et al. (2014) (220) (nor is it possible to reconstruct them from the available data).

woods and controls. No correlations were found between DNA damage and wood dust concentration or duration of exposure.

10.1.3 *Intestinal-type sinonasal adenocarcinoma*

Intestinal-type sinonasal adenocarcinoma (ITAC) is a relatively rare tumour associated with exposure to wood dust (though other types of sinonasal cancers have also been related to wood dust exposure). ITACs have a unique histological appearance, are mostly localised in the superior nasal cavity and ethmoid sinus with rare distant metastases, and develop after a long latency period of 20–30 years' exposure to wood dust. There are no identified precursor lesions leading to the development of ITACs, although hyperplasia, squamous metaplasia, and dysplasia occur frequently in areas adjacent to sinonasal carcinomas (1). In addition to the literature reviewed by IARC, a number of new mechanistic studies on ITACs in humans were identified in the current literature search.

Pérez-Escuredo et al. (2012) (226) investigated the spectrum of *TP53* gene mutations, as well as p53 protein expression, in 95 cases of primary ITACs of the (para)nasal tissues. Forty-four ITACs were analysed for *TP53* mutations; 92 ITACs were analysed for p53 expression. Eighty-three patients had occupational exposure to wood dust, with a mean of 32 years (range 1–60 years; no quantitative exposure data or information on possible co-exposure to other substances was reported), and 50 were habitual smokers. A frequency of 41% (18/44) *TP53* mutations and 72% (66/92) p53 immunopositivity was found in ITACs. All 18 patients with *TP53* mutations had professional exposure to wood dust, and most cases with p53 positive staining had wood dust aetiology (but because of the low number of cases without wood dust exposure [12/95], these associations were not statistically significant). With respect to clinicopathologic parameters, no association was found between *TP53* mutation and histologic type, tumour stage, intracranial invasion, recurrence, metastasis or survival. *TP53* mutations, or p53 immunopositivity, were not significantly correlated with tobacco smoking. G→A transition (50%, 9/18 cases) was the most common alteration detected, almost exclusively in non-smokers, whereas G→T (27%, 5/18 cases) was detected in smokers only. According to the authors, these data point to wood dust exposure as the causal factor in the mutagenesis of *TP53*.

Perez-Escuredo et al. (2016) (227) analysed chromosomal aberrations in ITACs of 37 patients. Thirty-four patients had occupational exposure to wood dust with a mean of 33 years (range 5–60 years; no quantitative exposure data or information on possible co-exposure to other substances was reported), and 18 were tobacco and alcohol users. Microarray comparative genomic hybridisation identified the following recurrent aberrations: gains at 5p15 (22 cases, 60%), 8q24 (21 cases, 57%), 20q13 (20 cases, 54%), 20q11 and 8q21 (19 cases, 51%), and 20p13 and 7p11 (16 cases, 43%), and losses at 5q11-qter, 8p12-pter and 18q12-23 (15 cases, 40%), and 17p13 and 19p13 (13 cases, 35%). Chromosomal loss at 4q32-ter and gains at 1q22, 6p22 and 3q29, as well as deletion of TIMP2 and CRK, correlated with an unfavourable clinical outcome. The authors did not attempt to relate specific aberrations to wood dust exposure or other factors (e.g. smoking). It

was concluded that ITACs have a unique pattern of chromosomal abnormalities. The four different histological subtypes of ITAC (papillary, colonic, solid and mucinous) appeared genetically similar. Four chromosomal gains and losses and two specific genes showed prognostic value and may be involved in tumour progression.

Holmila et al. (2010) (228) analysed *TP53* gene mutations in 358 sinonasal cancer cases from Denmark, Finland and France. Cases were not limited to ITACs; histological examination showed 59% squamous cell carcinomas, 34% adenocarcinomas, and 6% carcinoma NOS. Exposure to wood dust (100 cases in total) and other risk factors (e.g. occupational exposure to formaldehyde, chromium (VI) compounds, nickel and its organic compounds, textile and leather dust) was assessed. In Finland (109 cases) and France (79 cases), wood dust concentrations were assessed by industrial hygienists into 6 categories (unexposed; <0.3 mg/m^3; 0.3–<1 mg/m^3; 1–<2 mg/m^3; 2–<5 mg/m^3; ≥5 mg/m^3). A significant association between wood dust exposure and adenocarcinoma histology was observed (OR for adenocarcinoma 12.6, 95% CI 5.0–31.6; exposed vs unexposed, adjusted for smoking). With regard to types of wood dust, those cases who had had the most exposure to hardwood dust (>50% of total wood dust exposure) exhibited almost exclusively adenocarcinoma (19 of 20), whereas those mainly exposed to softwood dust also presented squamous cell carcinoma (23 of 43). *TP53* mutations occurred in all histologies, with an overall frequency of 77%. *TP53* mutation positive status was most common in adenocarcinoma (OR 2.0, 95% CI 1.1–3.7; compared with squamous cell carcinoma), and mutation positivity showed an overall, nonsignificant association with wood dust exposure (OR 1.6, 95% CI 0.8–3.1). Risk of *TP53* mutation was significantly increased in association with duration (≥24 years, OR 5.1, 95% CI 1.5–17.1; compared with ≤24 years), average level (>2 mg/m^3; OR 3.6, 95% CI 1.2–10.8; compared with ≤2 mg/m^3) and cumulative level (≥30 mg/m^3 x years; OR 3.5, 95% CI 1.2–10.7; compared with ≤30 mg/m^3 x years) of wood dust exposure; adjustment for formaldehyde affected the ORs only slightly. Smoking did not influence the occurrence of *TP53* mutation. According to the authors, the results indicate that mutational mechanisms, in particular *TP53* mutations, are associated with work-related exposure to wood dust in sinonasal cancer.

Hoeben et al. (2016) (229) reviewed the literature on general ITAC histopathology, clinical presentation and prognosis, treatment options, and molecular profile (including gene mutation and/or altered expression of *B-Raf*, *K-Ras*, *N-Ras*, *H-Ras*, *EGFR*, *ERBB2* and *TP53*). The authors argue that ITAC probably develops through intestinal metaplasia induced by hardwood and leather dust. They hypothesise that normal respiratory mucosa first undergoes cuboidal metaplasia, followed by intestinal metaplasia, and then possibly through dysplasia develops into ITAC. Inhaled wood dust particles larger than 5 µm become trapped in the mucosa of the middle turbinate and ethmoid and weaken the ciliary function of the nasal cells, which prolongs the contact between the particles and the mucosa. They suggest that wood dust does not have direct mutagenic properties of its own, and hypothesise that prolonged exposure to and irritation by wood dust particles stimulate cellular turnover by inflammatory pathways. Chronic inflammation seems to

play a role in the tumorigenesis and the phenotypical switch to ITACs. Inflammation is known to be able to cause genetic and epigenetic changes that promote proliferation and growth. It is not yet known which cytokines are most important in the inflammation induced by wood dust.

10.2 Respiratory tract effects

In the assessment performed by SCOEL (2003) (2), non-carcinogenic effects, signs and symptoms in the respiratory tract after occupational exposure to wood dust were reported to include: increased incidence of metaplasia in the nasal mucosa, reduced mucociliary clearance, altered olfactory function, increased frequency of nasal symptoms (obstruction, discharge, nose blowing) and episodes of ocular irritation, pharyngeal disorders, asthma, coughing, chronic bronchitis, altered respiratory function parameters (FEV_1; FVC; FEV_1/FVC; MMEFR), idiopathic pulmonary fibrosis and extrinsic allergic alveolitis.

Generally, limited data are available with respect to the mechanisms of toxicity of the abovementioned wood dust-related non-carcinogenic effects in the respiratory tract. Some mechanistic data are available from studies in non-human experimental systems (e.g. on ROS production, expression of cytokines and chemokines, inflammation and enzyme induction); these studies are summarised in Chapter 8 'Effects in animals' (*in vivo* studies) and Table 25 (*in vitro* studies). The current literature search revealed the following studies in humans published in recent years.

Bono et al. (2019) (230) investigated the prevalence of urinary 15-F_{2t} isoprostane (15-F_{2t}-IsoP), a biomarker for oxidative stress and peroxidation of lipids, in 123 woodworkers (from 44 companies) compared with 57 unexposed controls living in the Tuscany region of Italy. Data on wood dust exposure (as well as smoking habits and possible occupational co-exposure to carcinogens) were derived from Italian national exposure registries; the mean level of wood dust exposure was 1.48 mg/m^3 (8-hour TWA). The results showed a statistically significant excess of urinary 15-F_{2t}-IsoP in woodworkers: the overall MR between workers exposed to wood dust and controls was 1.36 (95% CI 1.18–1.57) after correction for age and smoking habits. After stratification for occupational co-exposure to carcinogens, the MR (woodworkers vs controls) was 1.34 (95% CI 1.15–1.56; p=0.0001) for workers exposed to wood dust only, and 1.41 (95% CI 1.17–1.70; p=0.0002) for workers exposed to wood dust with organic solvents. Increased urinary 15-F_{2t}-IsoP levels also correlated with occupational history (i.e. exposure duration).

Saad-Hussein et al. (2016) (231) investigated the association between *ADAM33* gene polymorphisms, arginase activity and lung function impairment in 82 non-smoking woodworkers from an Egyptian furniture factory and 81 controls. ADAM33 is a proteinase which cleaves α2-macroglobulin, and is supposed to play a role in lung function impairment. Genomic DNA from peripheral blood leukocytes was analysed for *ADAM33* single nucleotide polymorphisms (T1, T2, S1 and Q1); arginase activity was determined in blood, and ventilatory function tests (FEV_1, FVC and PEF) were performed in all workers. No information

on the level of wood dust exposure or possible co-exposure to other substances was reported. Significant reductions in all lung function parameters ($P<0.0001$) and a significant increase in arginase activity ($p<0.05$) were observed in woodworkers compared with controls. The authors concluded that an association between *ADAM33* gene polymorphisms and impaired lung function was apparent in wood dust-exposed workers; arginase activity may play an associated important role in increasing this impairment in woodworkers.

Ahman and Holmstrom (2000) (232) performed nasal histamine challenges in 14 woodwork teachers with work-related rhinitis and 14 healthy non-allergic controls to investigate whether nasal complaints were related to nasal hyperreactivity. Symptoms (nasal blockage, runny nose, nasal itching, sneezing) were scored (0–3) and acoustic rhinometry was performed before and after each histamine challenge (histamine concentrations were stepwise increased over subsequent challenges, from 0 to 16 mg/ml histamine in saline). No information on the level of wood dust exposure or possible co-exposure to other substances was reported. There was no significant difference between groups regarding symptom scores or acoustic rhinometry. According to the authors, the results indicate that nasal hyperreactivity is not a prominent factor in wood dust-related rhinitis.

Gripenbäck et al. (2005) (233) exposed 11 healthy non-smoking volunteers to pinewood dust for 1 hour in a whole-body exposure chamber. Average concentrations of total dust, inhalable dust and respirable dust were 5.5, 6.8 and 1.0 mg/m^3, respectively[4]. Determination of aerodynamic particle size showed two particle modes, at ~8 and ~25 µm. FEV_1 was measured pre- and post-exposure, BAL fluid and blood cells were differentially counted, and expression of activation, adhesion and subset markers on alveolar macrophages and T-lymphocytes was determined 2–6 weeks before and 20 hours after exposure. A statistically significant increase in total cell numbers in BAL fluid was observed after exposure ($p<0.001$); the percentage of lymphocytes ($p=0.019$) and eosinophils ($p<0.001$) in BAL was increased, and the percentage of macrophages was decreased correspondingly ($p=0.003$). The absolute number of mast cells in BAL fluid was also increased post-exposure ($p=0.004$). In peripheral blood, there was a significant decrease in relative lymphocyte and eosinophil numbers ($p<0.05$), and an increase in neutrophil numbers ($p=0.007$). Exposure resulted in an altered macrophage phenotype in BAL fluid: expression of CD14 was increased ($p<0.001$), and expression of CD11a, CD86 and HLA-DR was decreased ($p<0.001$, $p=0.019$ and $p<0.001$, respectively). In addition, the percentage of CD4+ T-cells in BAL expressing CD69 was decreased ($p=0.0078$). No changes in marker expression were found in peripheral blood lymphocytes, and the CD4/CD8 ratio remained unchanged in BAL fluid and blood. The authors concluded that inhalation of pinewood dust leads to the recruitment of inflammatory cells to the airways of healthy individuals, and that the increase in numbers of eosinophils, T-lymphocytes and mast cells (i.e. cells of crucial importance

4 It is noted that the average concentration of inhalable dust was higher than the average concentration of total dust. This may be related to differences in sampling efficiency of the devices used for sampling total vs inhalable dust.

to airway inflammation) in the lungs may be related to the increased risk of developing respiratory disorders among woodworkers.

Jacobsen et al. (2010) (234) reviewed the literature on non-malignant respiratory diseases and occupational exposure to wood dust. They state that the mechanisms by which wood dust induces respiratory impairment are far from being fully understood. For red cedar, a low molecular compound, plicatic acid, has been shown to be a causal factor for asthma, and both immunological and non-immunological mechanisms are involved. For other types of wood, no clear causal agent has consistently been found. Specific IgE sensitisation has been reported to be responsible for asthma in woodworkers; however, Type 1 allergy is not suspected to be a major cause of wood dust-induced asthma. Apart from IgE-mediated sensitisation, several other mechanisms, including irritative mechanisms, have been reported. Animal studies have shown that wood components, such as the major constituent in pine resin – abietic acid – causes direct toxicity via lytic damage to alveolar, tracheal and bronchial epithelial cells. Wood dust extracts from both hard- and softwoods are able to induce the release of pro-inflammatory mediators from macrophages, express and induce the release of inflammatory mediators in human epithelial cell line, and modulate the expression of cytokines and chemokines.

The conclusions from animal and *in vitro* studies formulated by Jacobsen et al. (2010) (234) were based on studies which are also tabulated in this report. One additional study was found which was not reviewed by Jacobsen et al. (2010) (234): Pylkkänen et al. (2009) (235) studied the capacity of wood dust to induce the production of ROS and apoptosis (caspase-3 activity) in human bronchial epithelial cells. Dust from three wood species – pine, birch and oak – induced ROS production and apoptosis. The study concluded that oxidative stress by ROS may be an important mechanism in wood dust-related pulmonary toxicity.

10.3 Dermal effects

As described in the evaluation by SCOEL (2003) (2), numerous studies report the occurrence of various cutaneous pathologies (contact or allergic eczemas) in workers exposed to wood dust. Most woods seem capable of inducing these cutaneous manifestations. Dermatoses induced by wood dust show no particular characteristics. They are related to irritant or allergic phenomena or both, and are caused by direct contact of the skin with the dust. In general, they are limited to the most exposed areas (hands, forearms, face, neck).

In an ACGIH (2015) (236) review it is further noted that wood dust can cause allergic contact dermatitis as a result of Type I and Type IV hypersensitivity, as well as irritant dermatitis. Although allergic contact dermatitis is relatively rare, the majority of the reported cases were occupationally related; these workers often reported respiratory and mucosal symptoms (e.g. conjunctivitis, rhinitis, and asthma), in addition to dermatitis ACGIH (2015) (236).

Table 22 Mutagenicity

Reference	Test system	Exposure conditions	Results	Remarks
Mohtashamipur et al. (1986) (237)	**Reverse mutation assay (Ames test; plate incorporation method):** *Salmonella typhimurium* TA100 -/+ S9 -/+ NADPH-generating system	**Beech wood dust extract:** Methanol extracts of untreated beech wood dust were dried and dissolved in DMSO (for mutagenicity testing) or in methanol for fractionation on silica gel using various eluents; fractions were dried and dissolved in DMSO. **Exposure levels:** 0, 2.5, 5, 10 g per plate (at least in triplicate; exposure duration not reported). **Positive controls:** benzo(a)pyrene, aflatoxin B_1, N-methyl-N'-nitro-N-nitrosoguanidine	**Non-fractionated extracts:** no induction of reverse mutant frequency Cytotoxicity: up to 76% cell death at the top dose **Fractionated extracts:** Mutagenicity was mostly found in response to treatment with the ethyl acetate phase (other phases were not reported). *+S9*: Dose-dependent, up to 2.6-fold increase of mutant frequency relative to solvent control Cytotoxicity: up to 69% cell death at the top dose. *-S9*: no effect on mutant frequency Statistical analysis not reported.	No guideline followed. Deficiencies: • limited reporting of experimental procedures and results • no information on statistics • only one strain tested

Reference	Test system	Exposure conditions	Results	Remarks

Mohtashamipur and Norpoth (1990) (238)	**Reverse mutation assay (Ames test; plate incorporation method):**
	Salmonella typhimurium TA97, TA98, TA100, TA1535 and TA1537
	
	-/+ S9
		Chemically or microbially degraded untreated beech wood dust*
		
		Exposure levels:
		0, 0.25, 0.5, 1, 5 and 10 g dust in acetone per plate (triplicate experiments per dose; exposure duration not reported)
		Positive controls:
		benzo(a)pyrene, aflatoxin B_1, ethyl methane sulfonate, sodium azide
		
		* Chemical degradation: acetone/water (9:1) wood dust extracts were dried and fractionated into 3 fractions (phases I, II and III) on Sephadex using various organic solvents. Microbial degradation (TA100 with S9 only): 3- or 30-day incubation of wood dust with the fungi *Phanerochaete chrysosporium* or *Chaetomium globosum* followed by ethyl acetate extraction.
			Chemical degradation:
			Cytotoxicity: observed for all 3 phases, up to ~100% cell death at 1 g/plate
			
			Mutagenicity:
			Phase I and III: no induction of reverse mutant frequency
			Phase II: dose-dependent increase in mean revertants per plate in TA 100 (+S9), TA1535 (-S9) and TA1537 (no statistics reported):
			• TA100 (+S9, -S9)
			  • 0: 98.7 (± 5.9), 75.2 (± 7.4)
			  • 0.25: 203.3 (±21.3), 85.0 (±1.0)
			  • 0.5: 424.0 (±39.8), 84.0 (±7.1)
			  • 1: 942.0 (±61.6), 0.0 (±0.0)
				No guideline followed.
				
				Deficiencies:
				• limited reporting of experimental procedures and results (e.g. quantitative data on cytotoxicity were given for only a few conditions
				• no data on positive controls reported)
				• some exposure conditions tested in only one strain
				• no information on statistics

Reference	Test system	Exposure conditions	Results	Remarks

			• TA1535 (+S9, -S9)
			  • 0: 25.6 (± 1.0), 22.6 (± 2.7)
			  • 0.25: 12.3 (±0.9), 116.0 (±1.0)
			  • 0.5: 13.7 (±2.5), 175.0 (±32)
			  • 1: 12.0 (±0.8), 1.0 (±0.0)
			• TA1537 (+S9, -S9)
			  • 0: 18.2 (± 1.6), 7.2 (± 1.9)
			  • 0.25: 40.7 (±3.8), 29.0 (±2.7)
			  • 0.5: 82.7 (±14.4), 40.7 (±3.8)
			  • 1: 531.0 (±14.7), 9.7 (±5.2)
			• TA 897, TA98: no induction of reverse mutant frequency
			• After repurification of the mutagenic fraction on silica-gel (ethylacetate phase, tested in TA100 +S9 only):	

Reference	Test system	Exposure conditions	Results	Remarks
			• 0: 86.0 (± 4.8) • 1: 1893.0 (± 653) • 5: 3206.0 (± 196) • 10: 4901.0 (± 192) • (no toxicity reported) **Microbial degradation** (TA100 +S9)**:** • *Ch. globosum*: no induction of reverse mutant frequency • *Ph. chrysosporium*: dose-dependent increase in mean revertants/plate after 30 days; no increase after 3 days incubation (results reported in figures only; no statistics reported)	
Kurttio et al. (1990) (239)	**Reverse mutation assay (Ames test; plate incorporation method):** *Salmonella*	**Wood-chip drying fumes from spruce and birch:** • Direct exposure: fumes of heated (80 or 170°C), freshly planed wood chips	**Toxicity:** reduced survival in all strains after direct exposure (no statistics reported; no data reported of extracts or indirect exposure)	no guideline followed **Deficiencies:** • limited reporting of experimental procedures and results

Reference	Test system	Exposure conditions	Results	Remarks
	typhimurium TA97, TA100 TA102 -/+ S9	• Indirect exposure: fumes were filtered, condensed, and collected on XAD-2, followed by a series of extraction steps • Extract exposure: wood chips extracted using DMSO, acetone, L-α-phosphatidyl-choline, dipalmitoyl and distilled water **Exposure levels:** one concentration of fumes (not characterised) for 10, 20 or 30 min, followed by 48 h (TA98, TA100) or 72 h (TA102) incubation at 37°C (3–6 plates per strain) **Negative controls:** DMSO, acetone, lecithin, water, XAD-2 filter extract **Positive controls:** 4-nitro-*o*-phenylenedi-amine, sodium azide, cumene hydroxyperoxide, benzo[a]pyrene, methyl-ene methanesulfonate	**Mutagenicity:** no increase in mean revertants/plate after direct exposure, indirect exposure, or exposure to extracts	• exposure to fumes was not characterised • only 3 strains tested • no information on statistics • The authors concluded that some mutagenicity was observed upon exposure to wood chips heated at 170°C, but the IARC Working Group noted the inappropriate correction for cell survival and concluded that the results were negative for mutagenicity (IARC 1995).
Singer et al. (1995) (240)	**Reverse mutation assay (Ames test):**	**Wood drying condensates from Douglas fir, red oak,**	Results reported in wording ('toxic', 'mutagenic', or 'no	No guideline followed.

Reference	Test system	Exposure conditions	Results	Remarks

	• *Salmonella typhimurium* TA97a, TA98, TA100 TA102
	• *Escherichia coli PQ37* (SOS Chromotest*)
	
	-/+ S9
	
	* performed in microtiters plates (classical protocol) and in agar plates (modified protocol)
		southern yellow pine, yellow poplar and eastern white pine:
		• Undiluted condensate ('1x'): collected from wood chips dried at 120°C in a closed laboratory extraction-drying system
		'1000x' condensate:
		• 1000-fold concentrated (by lyophilization at -52°C) condensate
		Exposure levels:
		• Ames test: 10-fold serial dilutions of 1x condensate, ranging from 1 µl of a 10E-6 dilution to 1 ml undiluted condensate, for 48 h (in triplicate)
		• SOS chromotest: unclear (dose levels ambiguously reported), for 3.5 h (in quadruplicate; classical protocol) or for 18–24 h (in duplicate; modified protocol)
			effect') or figures only; no quantitative data provided, no statistical analysis reported
			Ames test:
			• Yellow poplar (-S9; +S9):
			  • TA97a: no effect; mutagenic
			  • TA98: no effect; no effect
			  • TA100: toxic; no effect
			  • TA102: toxic; no effect
			• Douglas fir (-S9; +S9):
			  • TA97a: no effect; mutagenic, toxic
			  • TA98: no effect; no effect
			  • TA100: no effect; mutagenic
			  • TA102: toxic; mutagenic
			• Red oak (-S9; +S9):
			  • TA97a: no effect; toxic
				Deficiencies:
				• limited reporting of experimental procedures and results
				• no quantitative data provided
				• ambiguous reporting of exposure levels and conditions
				• condensates not chemically analysed
				• no information on statistics

Reference	Test system	Exposure conditions	Results	Remarks
		Negative controls: 10% DMSO-saline **Positive controls:** Methyl methanesulfonate, 4-Nitroquinoline 1-oxide, sodium azide, 2-amino anthracene, 2-aminofluorene	• TA98: no effect; no effect • TA100: toxic; toxic • TA102: toxic; toxic • White pine (-S9; +S9): • TA97a: toxic; mutagenic, toxic • TA98: no effect; no effect • TA100: toxic; mutagenic, toxic • TA102: toxic; mutagenic, toxic • Southern yellow pine (-S9; +S9): • TA97a: no effect; mutagenic • TA98: no effect; no effect • TA100: no effect; toxic • TA102: toxic; mutagenic, toxic **SOS Chromotest:** • Classical protocol: no cytotoxicity, no mutagenicity observed after	

Reference	Test system	Exposure conditions	Results	Remarks
			treatment with wood drying condensate • Modified protocol: 1x condensates: no cytotoxicity, no mutagenicity • 1000x condensates: cytotoxicity observed for all condensates; mutagenicity observed for Douglas fir (+S9) only	

Table 23 Cytogenicity

Reference	Test system	Exposure conditions	Results	Remarks
***In vivo* studies**				

Nelson et al. (1993) (241)	**Micronucleus test** and **DNA adduct formation** in nasal tissue
	
	Male Wistar rats
	
	Intranasal instillation 1x/d on 3 consecutive days
		Beech wood dust (particle size not reported) extracted in saline, ethanol, methanol, acidic methanol (pH 3), or acidic methanol followed by purification (as described by Mothashamipur et al. (1986))
		Exposure levels:
		0.5, 1 and 2 g/kg BW (BW range 220–250 g; no group averages reported) of dried extract in 50 µl saline or 1:1 mixture of saline and alcohol
		Negative controls:
		saline, ethanol and methanol
		Positive controls:
		1, 2 and 4 mg nitrosodimethylamine; 0.5, 1 and 2 mg formaldehyde
		Sacrifice:
		24 h after last treatment
		
		Micronucleï were assessed in nasal epithelial cells (duplicate slides per animal); total adduct levels
			Micronucleï (#/1000 cells):
			• Negative controls:
			  • saline: 1.6 (± 1.14)
			  • ethanol: 1.2 (± 0.84)
			  • methanol: 1.6 (± 1.14)
			• Wood dust:
			  • saline extracts:
			  0.5: 1.8 (± 1.10)
			  1: 2.0 (± 1.41)
			  2: 1.6 (± 1.14)
			  • ethanol extracts:
			  0.5: 2.0 (± 0.71)
			  1: 3.8 (± 1.30)*
			  2: 4.2 (± 1.30)*
			  • methanol extracts:
			  0.5: 1.8 (± 1.30)
			  1: 3.8 (± 0.84)*
			  2: 4.4 (± 1.82)*
			  • methanol extracts, acidic:
			  0.5: 1.8 (± 1.64)
			  1: 3.0 (± 0.71)
			  2: 1.8 (± 1.30)
			  • methanol extracts, acidic, purified:
			  0.5: 2.4 (± 1.52)
			  1: 4.0 (± 1.22)*
			  2: 2.2 (± 1.10)
			• Positive controls:
			  • nitrosodimethylamine:
			  1: 2.4 (± 0.89)
				No guideline followed.
				
				Deficiencies:
				• limited reporting of experimental details, procedures and results
				• number of animals/group not specified
				• statistical analysis unclear
				• no data on general toxicity reported
				• number of scored cells unclear
				• validity of ^{32}P-postlabelling test unclear, since no positive control data are reported

Reference	Test system	Exposure conditions	Results	Remarks
		were determined by liquid scintillation counting after ^{32}P-postlabelling of DNA extracted from the nose.	2: 4.0 (± 0.71)* 4: 5.0 (± 1.00)** • formaldehyde: 0.5: 3.2 (± 1.30)* 1: 4.2 (± 1.30)* 2: 5.2 (± 1.48)** * p<0.01; ** p<0.001 (unclear which control is used for comparison) P for trend: not reported **DNA adducts:** no significant changes after treatment with wood dust extracts (no data on positive controls reported) no data on general toxicity reported	
***In vitro* studies**				
Zhou et al. (1995) (242)	**CA** in MRC-2 human embryonic lung cells -/+ S9	**Beech, oak and pine wood dust extracts:** Methanol extracts of untreated wood dust were semi-purified on a silica gel and dried. **Exposure levels:**	**CA:** ***without S9*:** • Abnormal cells (%) • negative control: 2 • EMS (0.1; 0.2 mg/ml): 14**; 24** • beech; oak: 0.033: 2; 3 0.1: 4;4 0.33:7*; 7*	Deficiencies: • limited reporting of experimental procedures and results • limited number of cells evaluated

Reference	Test system	Exposure conditions	Results	Remarks
		0.033, 0.1, 0.33, 1, 3.3*, and 10* mg/ml (beech, oak) or 1, 3.3, 10, 33 and 100* mg/ml in culture medium (pine), for 36 h (in triplicate) **Negative control:** culture medium **Positive controls:** benzo(a)pyrene (B(a)P), ethylmethanesulfonate (EMS) * CA not evaluated because of high cytotoxicity	1: 8*; 10** P trend <0.05 (beech and oak) • pine: 1:2 3.3:3 10: 2 33.3: 3 * p<0.05; ** p<0.001 compared with controls • Chromatid breaks • negative control: 1 • EMS (0.1; 0.2 mg/ml): 10; 17 • beech; oak: 0.033: 2; 3 0.1: 4; 3 0.33: 5; 6 1: 9; 10 • P trend <0.05 (beech and oak) • pine: 1:1 3.3:2 10: 2 33.3: 3 ***With S9:***	• no data or statement on historical controls

Reference	Test system	Exposure conditions	Results	Remarks

			• Abnormal cells (%)
			  • negative control: 1
			  • B(a)P (0.003; 0.01 mg/ml): 7*; 17**
			  • beech; oak:
			  0.033: 1; 2
			  0.1: 3; 2
			  0.33:2; 4
			  1: 4; 5*
			  • pine:
			  1: 2
			  3.3: 3
			  10:1
			  33.3:2
			• Chromosome breaks and gaps, chromatid gaps, chromosome and chromatid exchange, MI: no significant changes after exposure to wood dust extracts
			Cytotoxicity:
			dose-dependent decrease in cell viability from >90% at the lowest dose to 0% at the highest dose (with and without S9)	

Mark et al. (1995) (243)	**CA** and **SCE** test in human PBL and CHO cells no metabolic activation	**Wood drying condensate from Southern Yellow Pine**, collected from fresh wood chips, dried at 121°C **Exposure levels:**	**CA:** % aberrant cells (0, 0.01, 0.1, 1, 10 µl/ml): CHO: 2, 6*, 10*, 16*, 3 (exp. 1) CHO: 2, 7*, 12*, 15*, 34* (exp. 2) PBL: 1, 2, 5*, 9*, 16* (exp. 1)	Deficiencies: • limited reporting of experimental procedures and results

Reference	Test system	Exposure conditions	Results	Remarks
		• 5 concentrations, range 0.01–100 µl condensate/ml culture medium (in duplicate), for 24 h • 2 experiments **Controls:** medium only (no positive control included)	PBL: 2, 2, 4, 7*, 9* (exp. 2) * p<0.05 compared with controls P for trend: not reported **SCE:** no significant changes in PRI or SCE **Cytotoxicity:** dose-dependent decrease in viability of CHO cells, from 100% (lowest dose) to 31% (highest dose; no statistics reported, not reported for PBL). Dose-dependent decrease in MI in CHO and PBL, p<0.05 compared with controls at 10 and 100 µl/ml	• procedure for preparation and characterisation of dosing solutions unclear • no positive controls included • no metabolic activation included
Mark et al. (1995) (244)	**CA** and **SCE** test in human peripheral blood lymphocytes (PBL) no metabolic activation	**Wood drying condensate from Eastern White Pine**, collected from fresh wood chips, dried at 121°C **Exposure levels:** • 5 concentrations, range 0.01–100 µl condensate/ml culture medium (in duplicate), for 24 h • 2 experiments (CA only) **Controls:** medium only (no positive control included)	**CA:** % aberrant cells (0, 0.01, 0.1, 1, 10, 100 µl/ml): Exp 1: 2, 6*, 11*, 13*, 18*, 33* Exp 2: 2, 9*, 11*, 13*, 16*, 27* **SCE:** average SCE/cell (± SE) (0, 0.01, 0.1, 1, 10, 100 µl/ml): 8.44 (± 0.21), 8.96 (± 0.18)*, 9.18 (± 0.25)*, 9.28 (± 0.21)*, 10.10 (± 0.35)*, 10.50 (± 0.24)* no significant changes in PRI **Cytotoxicity:** MI (%): (0, 0.01, 0.1, 1, 10, 100 µl/ml):	Deficiencies: • limited reporting of experimental procedures and results • procedure for preparation and characterization of dosing solutions unclear • no positive controls included

Reference	Test system	Exposure conditions	Results	Remarks
			Exp 1: 6.3, 5.4, 4.0*, 3.8*, 3.3*, 2.1* Exp 2: 6.6, 4.9*, 4.1*, 3.3*, 3.0*, 2.0* * p<0.05 compared with controls P for trend: not reported	• no metabolic activation included
Mark et al. (1996) (245)[a]	**CA** and **SCE** test in CHO cells no metabolic activation	**Wood drying condensate from Eastern White Pine**, collected from fresh wood chips, dried at 121°C **Exposure levels:** • 5 concentrations, range 0.01–100 µl condensate/ml culture medium (in duplicate), for 24 h • 2 experiments (CA only) **Controls:** medium only (no positive control included)	**CA:** % aberrant cells (0, 0.01, 0.1, 1, 10, 100 µl/ml): Exp 1: 2, 11*, 15*, 24*, 28*, 32* Exp 2: 4, 15*, 17*, 18*, 21*, 28* **SCE:** average SCE/cell (± SE) (0, 0.01, 0.1, 1, 10, 100 µl/ml): 6.32 (± 0.17), 7.02 (± 0.25)*, 8.52 (± 0.32)*, 9.58 (± 0.33)*, 10.78 (± 0.29)*, 11.22 (± 0.35)* no significant changes in PRI **Cytotoxicity:** MI (%) (0, 0.01, 0.1, 1, 10, 100 µl/ml): Exp 1: 8.50, 7.35, 6.35*, 5.50*, 3.70*, 2.75* Exp 2: 9.20, 8.00, 6.80*, 5.25*, 3.60*, 2.35*	Deficiencies: • limited reporting of experimental procedures and results • procedure for preparation and characterisation of dosing solutions unclear • no positive controls included • no metabolic activation included

Reference	Test system	Exposure conditions	Results	Remarks
			Cell viability: dose-dependent decrease from 91% (lowest dose) to 58% (highest dose) * p<0.05 compared with controls P for trend: not reported	
Mark et al. (1995) (246)	**CA** test in human PBL no metabolic activation	**Wood drying condensate from Red Oak**, collected from fresh wood chips, dried at 121°C. **Exposure levels:** • 5 concentrations, range 0.01–100 µl condensate/ml culture medium (in duplicate), for 24 h • 2 experiments (CA only) **Controls:** medium only (no positive control included)	**CA:** no significant change in percentage of aberrant cells after treatment with the condensate **Cytotoxicity:** no significant change in MI after treatment with the condensate	Deficiencies: • Due to the lack of a positive control (given the negative results) the validity of the assay cannot be confirmed. • limited reporting of experimental procedures and results • procedure for preparation and characterisation of dosing solutions unclear • no metabolic activation included

Reference	Test system	Exposure conditions	Results	Remarks
Mark et al. (1996) (245)	**CA** and **SCE** test in CHO cells no metabolic activation	**Wood drying condensate from Douglas-fir**, collected from fresh wood chips, dried at 121°C **Exposure levels:** • 5 concentrations, range 0.01–100 µl condensate/ml culture medium (in duplicate), for 24 h • 2 experiments (CA only) **Controls:** medium only (no positive control included)	**CA:** % aberrant cells (0, 0.01, 0.1, 1, 10, 100 µl/ml): Exp 1: 1, 2, 5*, 7*, 10*, 13* Exp 2: 3, 5, 7*, 9*, 13*, 15* **SCE:** average SCE/cell (± SE) (0, 0.01, 0.1, 1, 10, 100 µl/ml): 5.24 (± 0.139), 6.16 (± 0.173)*, 6.38 (± 0.168)*, 6.92 (± 0.210)*, 7.22 (± 0.221)*, 8.88 (± 0.309)* no significant changes in PRI **Cytotoxicity:** MI (%) (0, 0.01, 0.1, 1, 10, 100 µl/ml): Exp 1: 7.05, 5.28*, 3.75*, 3.66*, 3.55*, 3.16* Exp 2: 6.90, 6.18, 5.10*, 5.35*, 4.28*, 3.60* **Cell viability:** dose-dependent decrease from 86% (lowest dose) to 32% (highest dose) * p<0.05 compared with controls P for trend: not reported	Deficiencies: • limited reporting of experimental procedures and results • procedure for preparation and characterisation of dosing solutions unclear • no positive controls included • no metabolic activation included

Table 24 DNA damage (in vitro studies)

Reference	Test system	Exposure conditions	Results	Remarks
Bornholdt et al. (2007) (247)	**Comet assay**, **cytotoxicity** (LDH release), **cytokine expression** (IL-6, IL-8) A549 human lung carcinoma cell line	**Hard (beech, birch, oak, teak) and soft (pine, spruce) wood dust** (>90% of particles <5 µm); **MDF dust** (<100 µm, mainly <10 µm) **Exposure levels:** 0, 10, 30, 100, 300 µg dust/ml culture medium (in triplicate) for 3, 6, 24* and 48 h* **Positive control:** H_2O_2 (comet assay) * 24 and 48 h: strong cytotoxicity; not further investigated	**Comet assay** (DNA strand breaks): • 3 h: significant dose–response for beech, teak, pine and MDF ($p<0.01$); 1.2–1.4-fold increase at the top dose compared with controls; no difference between hard- and softwoods • 6 h: significant dose–response for MDF only ($p<0.01$) **Cytotoxicity:** no increase in LDH release after 3 and 6 h exposure to wood dust; strong cytotoxicity after 24 and 48 h (data not shown) **Cytokines:** increased IL-6 and IL-8 mRNA expression after 3 and 6 h exposure to all wood dusts; statistically significant dose-response in the range of 0–100 µg/ml for all wood dusts ($p<0.05$ or smaller; linear regression), except for spruce after 3 h	• The IARC Working Group noted that the use of a malignant lung carcinoma cell line as a surrogate for sinonasal epithelial cells is questionable, and no particular control group was included (IARC 2012). • data shown only in pictures or figures

Table 25 Miscellaneous: ex vivo / in vitro non-genotoxicity studies

Reference	Test system	Exposure conditions	Results	Remarks
Evans and Nicholls (1974) (248)	**Histamine release** pig and human lung	**Western red cedar extracts:** aqueous extracts of dust collected from workshop benches and floor **Exposure levels:** • pig lung: 5, 10, 25, 50, 80, 100 mg/ml in Tyrode solution (in triplicate) • human lung: 80 mg/ml in Tyrode solution (in duplicate) • incubation: for 25 min with 100 mg fresh, chopped lung tissue **Positive control:** compound 48/80	**Histamine release:** • Pig lung: dose-dependent increase in histamine release, inhibited by addition of potassium cyanide (metabolic inhibitor) (no statistics reported) • Human lung: no relevant results reported (only one dose tested; no negative control)	Investigation of respiratory allergenic potential. Deficiencies: • validity of the method unclear • limited reporting of experimental procedures and results • no negative control included • exposure insufficiently characterised • no statistics
Bhattacharjee et al. (1979) (49)	**Macrophage cytotoxicity:** guinea pig alveolar macrophages	**Sheesham and mango wood dust** (particle size: 71–75% <9.1 µm; 28% <4.5 µm) **Exposure levels:** 0.1 mg wood dust in 1 ml HBSS (Hanks' basal salt solution) for 3 h (N=5 per dust type; 3 replicates) **Negative control:**	**Cytotoxicity:** • Sheesham dust: 2.4-fold increase in percentage of erythrosin-stained cells compared with negative control ($p<0.0005$) • Mango dust: 2.2-fold increase in percentage of erythrosin-stained cells	Study performed in parallel to *in vivo* study (see Table 8) to investigate a supposed relationship between *in vivo* fibrogenicity and *in vitro*

Reference	Test system	Exposure conditions	Results	Remarks
		cells only **Positive control:** chrysotile dust	compared with negative control ($p<0.005$)	cytotoxicity and hemolytic activity. Deficiencies: • limited reporting of experimental procedures and results • only one dose tested in macrophage assay

	Hemolytic activity:
	sheep red blood cells
		Sheesham and Mango wood dust (particle size: 71–75% <9.1 µm; 28% <4.5 µm)
		Exposure levels:
		5, 10, 20 or 40 mg dust suspended in 10 ml cell suspension, for 2 h (number of replicates not reported)
		Negative control:
		wood dust in veronal-buffered saline only; red blood cells only
		Positive control:
		quartz
			Hemolysis:
			• Sheesham dust: dose-dependent increase
			  • 20 mg: 10% hemolysis ($p<0.005$)
			  • 40 mg: 55% hemolysis ($p<0.0005$)
			• Sheesham dust induced hemolysis at doses of 20 mg ($p<0.005$) and 40 mg ($p<0.0005$).
			• Mango dust:
			  • 40 mg: 4.5% hemolysis ($p<0.005$ compared with controls)
			• Mango dust was weakly hemolytic (4.5% hemolysis at a dose of 40 mg; $p<0.005$)	

Torronen et al. (1989) (249)	**Enzyme induction:** • aryl hydrocarbon hydroxylase (AHH) • aldehyde dehydrogenase (ALDH)	**Extract of hard- (alder; aspen) and softwood (pine; mixture of pine and spruce*):** Acetone extracts were filtered, dried and redissolved in acetone.	**Enzyme induction:** dose-dependent increase in AHH and ALDH activity after exposure to wood extracts. Softwoods, especially pine, seemed more potent than hardwoods.	Study aimed to compare different types of wood used as animal bedding material. Deficiencies: • data shown in figures

Reference	Test system	Exposure conditions	Results	Remarks
	• Hepa-1 mouse hepatoma cell line	**Exposure levels:** 0.5–5 mg equivalent wood dust/ml culture medium, for 24 h **Negative control:** vehicle * mixture ratio not reported	**Cytotoxicity:** dose-dependent decrease in cell survival after exposure to wood extracts	only (no statistics reported) • limited reporting of experimental procedures and results • only one replicate performed • no statistical analysis • legends of figures seem mixed up
Ayars et al. (1989) (250)	**Cell lysis** in: • type II pneumocytes* • A549 human lung carcinoma cell line **Epithelial desquamation** in tracheal explants* **Histology** in: • tracheal explants* • lung (*ex vivo*)* * from Sprague-Dawley rats	**Plicatic** (PA) **and abietic acid** (AA) (constituents of red cedar and pine wood, respectively) **Exposure levels:** 0.001–5 mg/ml for up to 24 h (lysis assay, tracheal explants); 1 mg plicatic acid/ml or 0.1 mg abietic acid/ml for 4 h (rat lungs, infused *in situ* immediately after sacrifice) **Negative control:** medium only **Positive control:**	**Cell lysis:** time- and dose-dependent increase, up to ~100% lysis after 24 h (pneumocytes and A549 cells). **Epithelial desquamation:** Average (± SEM) number of desquamated cells (x 10E5) at 24 h: • PA (0, 0.1, 1, 2.5, 5 mg/ml): 2.6 (± 0.8), 3.7 (± 1.6), 4.0 (± 0.7), 13.7 (± 1.3)*, 19.7 (± 1.7)* • AA (0, 0.01, 0.1, 1 mg/ml): 6.5 (± 1.0), 29.5 (± 2.5),	Study to investigate if these softwood resin constituents can directly damage lung cells

Reference	Test system	Exposure conditions	Results	Remarks
		Triton-X-100 (lysis assay)	87.5 (± 5.0)**, 895.0 (± 150.0)** **Histology:** • PA: desquamation of bronchial epithelium • AA: desquamation of bronchial and alveolar epithelium • (no incidences reported) * p<0.05; ** p<0.01 compared with negative controls P for trend: not reported	
Gordon et al. (2002) (251)	**Cytotoxicity**, **metallothionein (MT) mRNA expression** Chinese hamster lung fibroblast (V79) cells	**Southern yellow pine dust** (particle size <3.4 µm; untreated) **Exposure levels:** 62.5, 125, 250, 500, 1000 and 1250 µg wood dust/ml (suspended in F12 culture medium) for 24 or 48 h (in duplicate or triplicate) **Negative control:** medium only	**Cytotoxicity:** • dose-dependent decrease in colony survival after treatment with wood dust • average LC_{50}: 883 ± 91 µg/ml **MT expression:** no significant changes after exposure to wood dust	The aim of the study was to compare the effects of CCA (chromium, copper, arsenic) pressure-treated wood* with untreated wood * not described here
Naarala et al. (2003) (252)	**ROS production, apoptosis**, **cytotoxicity** mouse macrophages (RAW 264.7),	**Pine, birch and beech dusts** (endotoxin concentration <50 pg/mg) • 'total dust': prepared by sanding (particle size not reported)	Quantitative results reported for total dust only: **ROS production** (average % increase over controls, ± SEM): • Pine (RAW 264.7; PMNL)	Mechanistic study to investigate the role of oxidative stress in wood

Reference	Test system	Exposure conditions	Results	Remarks
	human polymorphonuclear leukocytes (PMNL)	• 'fine dust': >95% of particles <5 µm **Exposure levels:** 1–1000 µg wood dust/ml (suspended in HBSS buffer) for 60 min (ROS production, cell viability) or 3 h (apoptosis) **Negative control:** vehicle **Positive control:** etoposide (for apoptosis)	• 1: 28.9 ± 7.4; 20.3 ± 2.4 • 10: 176.5 ± 21.7*; 75.6 ± 2.4* • 25: 421.2 ± 74.5*; 152.0 ± 7.0* • 50: 587.9 ± 61.5*; 247.4 ± 10.5* • 100: 696.0 ± 73.9*; 369.3 ± 15.3* • 500: 551.7 ± 61.3*; 525.0 ± 26.4* • 1000: 308.6±38.4*; 427.8 ± 28.1* • Birch (RAW 264.7; PMNL) • 1: 1.8 ± 2.0; 10.3 ± 2.8 • 10: 3.7 ± 2.1; 19.2 ± 5.6 • 25: 24.9 ± 2.1; 36.0 ± 9.9 • 50: 49.1 ± 4.8*; 52.1 ± 14.8* • 100: 69.0 ± 5.8*; 73.6 ± 19.5* • 500: 96.4 ± 8.3*; 104.5 ± 25.3* • 1000: 78.5 ± 7.0*; 85.8 ± 21.9* • Beech (RAW 264.7) • 1: 4.0 ± 1.7 • 10: 0.1 ± 1.3 • 25: 2.4 ± 1.3	dust induced (cyto)toxicity

Reference	Test system	Exposure conditions	Results	Remarks

			• 50: 3.8 ± 2.3
			• 100: 13.5 ± 4.2
			• 500: 29.1 ± 6.4
			• 1000: 30.4 ± 6.7
			PMNL:
			no significant changes (no data reported)
			Cell viability:
			• dose-dependent decrease (all wood types)
			• average cell viability (%) at highest dose (pine, birch, beech):
			  • RAW 264.7: 36.1*, 73.8*, 56.0*
			  • PMNL: -7.1*, 31.3*, 30.9*
			
			* $p<0.05$ compared with controls
			P for trend: not reported
			
			Apoptosis (caspase-3 activity):
			• RAW 264.7: 38.1% increase after pine dust exposure (100 µg/ml only; no statistics reported); no changes after birch or beech dust exposure
			• PMNL: unreliable (no increase after exposure to positive control)	

Reference	Test system	Exposure conditions	Results	Remarks
			• fine dusts: similar effects on ROS production and cell viability as total dusts (no details reported)	
Long et al. (2004) (253)	**ROS production, cytokine expression, cell viability** alveolar macrophages from male Sprague-Dawley rats	**Pine dust** (PD), **heat-treated pine dust** (HPD) (>95% of particles <5 µm) **Exposure levels:** 5–200 µg/ml (suspended in culture medium) for 4 h **Negative controls:** untreated cells; medium only; 3-µm polystyrene microspheres **Positive control:** LPS	**Cell viability** (LDH release): no effect of wood dust treatment **Cytokines:** • mRNA: 100 µg/ml (PD and HPD): increased TNF-α and MIP-2 mRNA expression ($p<0.05$ compared to untreated controls) • protein: 5 µg/ml and above (PD and HPD): dose-dependent increase in TNF-α and MIP-2 release ($p<0.05$ compared to medium only); PD higher than HPD at all dose levels ($p<0.05$) **ROS:** 200 µg/ml (PD and HPD): increased ($p<0.05$ compared with untreated controls) **Co-incubation** with antioxidants glutathione or N-acetyl-cysteine: significant decrease ($p<0.05$) of PD- and HPD-induced release of ROS, TNF-α, and MIP-2	Mechanistic study to investigate the role of oxidative stress and inflammatory mediators in wood dust-induced (cyto)toxicity Data shown in pictures or figures only

Reference	Test system	Exposure conditions	Results	Remarks
Määttä et al. (2005) (254)	**Cytokine and chemokine expression** RAW 264.7 mouse macrophages	**Oak** and **birch dust** (>93% of particles <5 µm) **Exposure levels:** 10, 30, 100 or 300 µg/ml (in 5 ml PBS) for 2, 6, 24 or 48 h **Positive controls:** TiO_2, LPS **Negative control:** PBS only	**Birch dust:** • dose-dependent increase in: TNF-α*** (R+P) [$], IL-6* (R+P), CCL2*** (R+P), CCL3*** (R), CCL4*** (R), CXCL2/3*** (R) • dose-dependent decrease in: IL-1β** (R), CCL24** (R) **Oak dust:** • dose-dependent increase in: TNF-α*** (P), CCL2*** (P), CCL3*** (R), CCL4*** (R), CXCL2/3** (R) dose-dependent decrease in: • IL-1β*** (R), CCL24** (R) [$] R: mRNA; P: protein * p<0.05, ** p<0.01, *** p<0.001 for dose-dependency (ANOVA)	Mechanistic study to investigate the effect of wood dust exposure on the expression of inflammatory mediators Data shown in pictures or figures only
Määttä et al. (2006) (255)	**Cytokine and chemokine expression** RAW 264.7 mouse macrophages	**Hardwood (teak, beech) and softwood (spruce, pine) dust** (>90% of particles <5 µm) **Exposure levels:** 10, 30, 100 or 300 µg/ml (in 5 ml PBS) for 2, 6, 24 or 48 h **Negative control:** PBS only	**Teak dust:** • dose-dependent increase in: TNF-α*** (P) [$], IL-6* (R), CCL2*** (R+P), CCL3*** (R), CCL4*** (R), CXCL2/3** (R) • dose-dependent decrease in: IL-1β*** (R), CCL24*** (R) **Beech dust:** • dose-dependent increase in: TNF-α* (R+P), CCL2*** (P),	Mechanistic study to investigate the effect of wood dust exposure on the expression of inflammatory mediators

Reference	Test system	Exposure conditions	Results	Remarks
			CCL3*** (R), CCL4*** (R), CXCL2/3*** (R) • dose-dependent decrease in: IL-1β* (R), CCL24** (R) **Pine dust:** • dose-dependent increase in: TNF-α*** (P), IL-6** (P), CCL2** (R+P), CCL3*** (R), CCL4*** (R), CXCL2/3*** (R) • dose-dependent decrease in: IL-1β*** (R), CCL24* (R) **Spruce dust:** • dose-dependent increase in: TNF-α*** (P), IL-6** (P), CCL2*** (R+P), CCL3*** (R), CCL4*** (R), CXCL2/3*** (R) • Dose-dependent decrease in: IL-1β** (R) $ R: mRNA; P: protein * p<0.05, ** p<0.01, *** p<0.001 for dose-dependency (ANOVA)	Data shown in pictures or figures only
Pylkkänen et al. (2009) (235)	**ROS production**, **apoptosis, cytotoxicity** human bronchial epithelial cells (BEAS-2B)	**Hard- (birch, oak) and soft- (pine) wood dust** (>90% of particles <5 µm), endotoxin concentrations 50 (pine),	**Cytotoxicity:** dose-dependent decrease in cell viability after 2 and 6 h: 500 µg/ml: 14%*** (pine), 11%*** (birch), 16%** (oak) decrease in viability	Mechanistic study to investigate the role of oxidative stress in wood

Reference	Test system	Exposure conditions	Results	Remarks
		220 (birch) and 70 (oak) pg/mg **Exposure levels:** 10, 50 and 500 µg/ml, suspended in culture medium, for 0.5, 2, 6, 12 and 24 h **Controls:** untreated cells	**ROS:** maximum increase: • pine: 3.3-fold*** (50 µg/ml; 0.5 h) • birch: 2.1-fold*** (500 µg/ml; 2 h) • oak: 2.3-fold*** (50 µg/ml; 2 h) **Apoptosis** (caspase-3 activity): increased after 2 and 6 h. Maximum increase after 2 h at 500 µg/ml: • pine: 8.9-fold*** • birch: 3.8-fold*** • oak: 16.4-fold*** ** p<0.01; *** p<0.001 compared with controls P for trend: not reported	dust-induced (cyto)toxicity
Staffolani et al. (2015) (216)	**Cell transformation assay**; **comet assay** (DNA strand breaks) human bronchial epithelial cells (BEAS-2B)	**Hard- (oak, padouk)** and **soft- (pine, European Silver fir) wood dust** (>60% of particles <1 µm), not impregnated, endotoxin concentration <100 pg/ml **Exposure levels:** 5–500 µg/cm^2 (suspended in PBS and diluted in culture medium) for up to 24 h	**Cell viability:** ≥ 100 µg/cm^2 for 24 h: 50% decrease **Cell transformation (PDL):** increased PDL (no statistics reported) **ROS and oxidative DNA damage:** 50 µg/cm^2 for 16–24 h: increased (p<0.05 compared with controls)	Mechanistic study to unravel pathway of wood dust-induced cell transformation Data shown in pictures or figures only

Reference	Test system	Exposure conditions	Results	Remarks
		Tests: cell viability, cytotoxicity, PDL, Comet assay, ROS production, DNA repair activity, cell morphology		
Wilson et al. (2015) (217)	**Aryl hydrocarbon receptor (AhR) activation assay** transgenic yeast strain YCM3 (which expresses human AhR, with lacZ reporter plasmid)	**Hard- (teak, walnut, mahogany, poplar, red oak**) **and soft-** (**yellow pine, cypress, spruce, cedar**) **wood dust extracts:** filter-sterilised methanol extracts **Exposure levels:** 0.01–600 µg/ml **Positive control:** β-naphthoflavone (β-NF) **EC_{25}:** Concentration at which activation of AhR signalling is 25% of the positive control.	**EC_{25}** (µg/ml; 95% CI): • Teak: 0.09 (0.07–1.11) • Walnut: 2.99 (2.41–3.72) • Mahogany: 12.07 (8.54–17.45) • Poplar: 38.09 (31.85–47.65) • Other wood dusts: EC_{25} not reached Further experiments showed that 2-methylanthraquinone accounted for the AhR ligand activity in teak dust.	Mechanistic study to investigate the potential role of AhR (a ligand-activated transcription factor linked to the carcinogenic action of specific classes of chemicals) in wood dust toxicity

11 Existing guidelines, standards and evaluations

11.1 General population

There are no standards set for wood dust, derived for the general population by international organisations such as the World Health Organization (WHO), Agency for Toxic Substances and Disease Registry (ATSDR), the US Environmental Protection Agency (US EPA) and the European Chemical Agency (ECHA).

11.2 Working population

The GESTIS Substance Database[5] of the Institut für Arbeitsschutz der Deutschen Gesetzlichen Unfallversicherung (IFA) is used to gather occupational limit values of wood dust within Europe. The GESTIS Substance Database contains four entries under Occupational exposure limits of wood dust:

- dust, hardwood;
- dust, wood;
- dust, wood, total dust;
- softwood dust.

The references for the OELs for the US are given in the notes below Table 26. Abbreviations are listed in Appendix 2.

5 https://www.dguv.de/ifa/gestis/gestis-stoffdatenbank/index-2.jsp

Table 26 Occupational exposure limits for wood dust

Country (organisation)	OEL (ppm)	OEL (mg/m³)	TWA	Type of exposure limit	Type of wood dust (entry in GESTIS Substance Database where applicable)
The Netherlands	-	2	8 h	OEL	Dust, hardwood
European Commission	-	**2**[a,b]	8 h	BOELV	Dust, hardwood
	-	**3**[a,b,c]	8 h		Dust, hardwood
Germany (DFG)	-	-	-	-	-
Germany (AGS)	-	**5**[b]	8 h	MAK	Dust, hardwood
	-	2[d]	8 h	MAK	Dust, hardwood
United Kingdom (HSE)	-	3[e,f]	8 h	WEL	Dust, hardwood
		5	8 h		Softwood dust
Denmark	-	2	8 h	OEL	Dust, wood, total dust
	-	4	15 min	OEL	Dust, wood, total dust
	-	1	8 h		Softwood dust
	-	2	15 min		Softwood dust
Sweden (SWEA)	-	2	8 h	OEL	Dust, hardwood
		2	8 h		Dust, wood, total dust
		2	8 h		Softwood dust
US (ACGIH)[h]	-	0.5[g]	8 h	TLV	Western red cedar
	-	1[g]	8 h	TLV	All other species
US (NIOSH)[i]	-	1	8 h	REL	Hard wood dust, Soft wood dust, Western red cedar dust
US (OSHA)[j]	-	2.5	8 h	PEL	Wood dust (Western red cedar)
		5	8 h	PEL	Wood dust (all wood dusts except Western red cedar)
		10	15 min		Wood dust (all wood dusts except Western red cedar)

a Inhalable fraction: If hardwood dusts are mixed with other wood dusts, the limit value shall apply to all wood dusts contained in the mixture.
b Bold type = BOELV
c Limit value until 17.01.2023
d Reference value that represents the state of the art. Individual measures are related to this value.
e Inhalable fraction

f If hardwood dusts are mixed with other wood dusts, the WEL shall apply to all the wood dusts present in that mixture.
g Inhalable particulate matter
h ACGIH (2015) (236)
i NIOSH Pocket Guide to Chemical Hazards - Wood dust6
j https://www.cdc.gov/niosh/pel88/wooddust.html

6 https://www.cdc.gov/niosh/npg/npgd0667.html – last reviewed October 2019

11.3 Available background information on the derived occupational exposure limits

The Netherlands
The current OEL for hardwood dust in the Netherlands is 2 mg/m^3 TWA for 8 hours. This limit also applies if hardwood is mixed with other types of wood dust.[7]

In 1992, the Dutch Expert Committee on Occupational Safety (DECOS) advised a health-based recommended OEL for wood dust. Two considerations formed the basis of this: first, irritation of eyes and respiratory tract should be prevented, as well as mucostasis and impaired clearance of the nasal cavities; and second, the risk of asthma and hyperreactivity of the upper respiratory tract. DECOS advised an occupational exposure limit of 0.2 mg total wood dust per m^3 8-hour TWA. It was expected that prevention of eye and nose irritation would also prevent the induction of nasal metaplasia. A clear distinction in health risks between different wood species could not be made. However, dust of Western red cedar may cause occupational asthma at lower concentrations than dust of other wood species. In general, hardwood species seemed to constitute a greater health risk than softwood species (4).

European Commission
The European BOELV is 2 mg/m^3 TWA for 8 hours (Directive 2019/130). Transitional period: Limit value 3 mg/m^3 until 17 January 2023.

The former European Union SCOEL concluded in 2003 that exposure to wood dust was shown to be associated with an increase of sinonasal cancers (2), but a quantitative risk assessment was not feasible because of the lack of good-quality quantitative data on exposure levels associated with increased risks.

SCOEL recommended exposure limits according to different thresholds: 0.5, 1 and 5 mg/m^3 based on impairment of respiratory function and increased prevalence of pulmonary symptoms (2). The studies available did not provide adequate information for setting a health-based limit value for the protection of workers exposed to wood dust. Taking into account the uncertainties and limitations of the available studies, it could be stated that exposure above 0.5 mg/m^3 induces pulmonary effects and should be avoided. Exposure levels lower than 0.5 mg/m^3 were associated with the induction of bronchial asthma only when the exposure was to Western red cedar dust. The levels of 0.5 mg/m^3 (total dust) and 1 mg/m^3 (inhalable dust) were considered likely to be below the levels to which the cases of sinonasal cancers had been exposed.

Germany (DFG)
The DFG does not specify OELs for wood dust (256). Oak and beech were assigned a special position, as wood dust from these species appears to be particularly carcinogenic. Oak and beech wood dusts are classified as Carcinogen category 1 in the MAK List, not as established carcinogens but as established carriers of a carcinogenic principle which is associated with these dusts either necessarily or with great regularity.

7 https://wetten.overheid.nl/BWBR0008587/2020-08-01#BijlageXIII

Dusts from woods other than oak and beech are classified as Carcinogen category 3.

Germany (AGS)
Background information on the derived OEL by the AGS is not publicly available. Reference is made to the BOELV.

United Kingdom (HSE)
From January 2020, the HSE lowered the WEL for hardwood dust (inhalable fraction) from 5 mg/m^3 to 3 mg/m^3 (257). The HSE followed the limits set by the European Commission under the Chemical Agents Directive (98/24/EC) and the Carcinogens and Mutagens Directive (2004/37/EC).

Denmark
In 1987, Göteburgs Universitet published a document in Danish on wood dust[8]. This document may be used to derive an OEL. However, since the document was in Danish, it has not been included in this report.

Sweden (SWEA)
No background information was found.

US (ACGIH)
In 1986, the ACGIH (236) recommended a TLV-TWA of 0.5 mg/m^3 inhalable particulate matter for occupational exposure to Western red cedar dust to prevent asthma. For all other wood species, a TLV-TWA of 1 mg/m^3 was recommended to prevent upper and lower respiratory tract irritation and effects on pulmonary function.

US (NIOSH)
In 1987, NIOSH provided a comprehensive synopsis of the literature related to wood dust (258). This document does not contain recommendations for regulating occupational exposure to wood dust and no other information on NIOSH recommendations was not found during this project.

US (OSHA)
In 1994, OSHA established a permissible exposure limit of 5 mg/m^3 and an STEL of 10 mg/m^3 for all hardwood and softwood dusts except Western red cedar (259). For Western red cedar, a highly allergenic species of softwood, OSHA established an 8-hour TWA limit of 2.5 mg/m^3. Wood dust is defined as any wood particles arising from the processing or handling of woods. Hardwoods derive from the deciduous broad-leaved flowering species of trees, and softwoods include the coniferous species that do not shed their leaves in the winter.

No biological exposure limits are available for wood dust.

8 https://gupea.ub.gu.se/handle/2077/4074?locale=en

11.4 Classifications

Table 27 Classifications for carcinogenicity and sensitisation

Evaluating country (organisation)	Category and Carcinogenicity/ mutagen	Skin sensitisation	Reference
The Netherlands	1A – known to be carcinogenic to humans (hardwood dust); 2 – suspected human carcinogen (softwood dust)	-	CMR-list (260)
European Commission	Annex I of Directive 2004/37/EC - work involving exposure to hardwood dusts	-	Directive 2004/37/EC
IARC/WHO	1 – carcinogenic to humans (hard- and softwood dust)	Not evaluated	IARC 1995
Germany (DFG)	1 - carcinogenic in man (beech and oak wood dust) 3 - suspected carcinogens (wood dust (except beech and oak wood dust)	Various species[1]	DFG 2015
Germany (AGS)	NA	NA	NA
United Kingdom (HSE)	Hardwood dust[2] Capable of causing cancer and/or heritable genetic damage. Capable of causing occupational asthma.	Hard- and softwood dust[2]	EH40/2005
Denmark	NA	NA	NA
Sweden (SWEA)	NA	NA	NA
US (ACGIH)	A1 – confirmed human carcinogen (oak and beech) A2 – suspected human carcinogen (birch, mahogany teak, walnut) A4 – not classifiable as a human carcinogen (all other woods)	Western red cedar classified as a respiratory and dermal sensitiser. Insufficient data to recommend a notation for other types of wood.	ACGIH 2015
US (NIOSH)	NA	NA	NA
US (OSHA)	NA	NA	NA

NA – not available

1 The DFG distinguishes three levels of risk: danger of sensitisation of the skin (Sh); danger of sensitisation of the airways (Sa); danger of sensitization of the airways and the skin (Sah). Tree species are classified as follows:

- Acacia melanoxylon R.Br., Australian blackwood (Sh)
- Brya ebenus DC., cocus wood (Sh)
- Chlorophora excelsa (Welw.) Benth. & Hook, iroko, kambala (Sh)
- Dalbergia latifolia Roxb., East Indian rosewood, Bombay blackwood (Sh)
- Dalbergia melanoxylon Guill. et Perr., African blackwood (Sh)

- Dalbergia nigra Allem., Brazilian rosewood (Sh)
- Dalbergia retusa Hemsl., cocobolo, rosewood (Sh)
- Dalbergia stevensonii Standley, Honduras rosewood (Sh)
- Distemonanthus benthamianus Baill., ayan (Nigerian satinwood) (Sh)
- Grevillea robusta A. Cunn., Australian silky oak (Sh)
- Khaya anthotheca C.DC., African mahogany (Sh)
- Machaerium scleroxylon Tul., pao ferro, Santos rosewood (Sh)
- Mansonia altissima A.Chev., mansonia, pruno, bété, African black walnut (Sh)
- Paratecoma peroba (Record) Kuhlm., ipe peroba (Sh)
- Tectona grandis L.f., teak (Sh)
- Terminalia superba Engl. & Diels, fraké, limba, afara, white afara (Sa)
- Thuja plicata (D.Don.) Donn., western red cedar, giant arborvitae,
- shinglewood (Sah)
- Triplochiton scleroxylon K.Schum., obeche, wawa, African whitewood (Sah)

2 Category not further specified

Wood dust is not classified for reproductive toxicity by the WHO, European countries or the US.

Appendix 1: Search terms

The search was performed in March 2020 and updates were carried out on 1 October 2020 and 11 May 2021. The number of references found over the complete period is reported below.

11.1 Embase

Set #24: Chapters 4, 6, and 9 (#24 combines search terms for wood dust and occupational exposure).
Set #40: Chapter 5 (#40 combines search terms for wood dust, occupational exposure and measurement and analysis)
Set #139: Chapters 7, 8 and 10:

- #53: Chapter 7 (#53 combines search terms for wood dust and toxicokinetics);
- #58: Chapter 10 (#58 combines search terms for wood dust and mechanisms of toxicity);
- #76: Chapter 8 (#76 combines search terms for wood dust and effects in animals);
- #137: Chapter 10 (additional search terms for mechanisms of toxicity);
- #138: Chapter 8 (additional search terms for effects in animals).

Set(#)	Search terms	References
#139	#53 OR #58 OR #76 OR #137 OR #138	664
#138	#136 NOT #76	92
#137	#119 NOT #58	157
#136	#133 OR #135	267
#135	#132 NOT #134	216
#134	(#121 OR #123 OR #125 OR #127 OR #129 OR #131) AND [humans]/lim	425
#133	(#121 OR #123 OR #125 OR #127 OR #129 OR #131) AND [animals]/lim	196
#132	#121 OR #123 OR #125 OR #127 OR #129 OR #131	641
#131	#130 AND 'Review'/it	52
#130	#114 AND #128	1,648
#129	#107 AND #128	541
#128	'in vitro study'/exp OR 'in vivo study'/exp OR 'vitro*':ti OR 'vivo*':ti	16,547,159
#127	#114 AND #126	13
#126	'disease model'/exp OR 'disease model*':ti	187,383
#125	#124 AND 'Review'/it	5
#124	#114 AND #122	204
#123	#107 AND #122	81
#122	'experimental animal'/exp OR cat:ti OR cats:ti OR cattle:ti OR dog:ti OR dogs:ti OR 'fish*':ti OR goat:ti OR goats:ti OR 'guinea pig*':ti OR 'hamster*':ti OR 'horse*':ti OR 'monkey*':ti OR pig:ti OR pigs:ti OR 'rabbit*':ti OR rat:ti OR rats:ti OR sheep:ti	2,258,330
#121	#107 AND #120 OR (#114 AND #120 AND 'review'/it)	77
#120	'animal experiment'/exp OR 'animal experiment*':ti OR 'animal model*':ti OR	2,553,598

	'mechanism of action*':ti OR 'mode of action*':ti	
#119	#116 OR #118	409
#118	#117 AND 'Review'/it	83
#117	#114 AND #115	859
#116	#107 AND #115	343
#115	'toxicity and intoxication'/exp OR 'toxicology'/exp OR 'toxic*':ti OR 'adverse effect*':ti OR 'side effect*':ti OR (('mode*' NEAR/3 'action*'):ti,ab) OR 'mechanism*':ti,ab OR 'lavag*':ti,ab OR 'mutagenicity'/exp OR 'carcinogen'/exp OR 'genotoxicity'/exp OR 'cytotoxicity'/exp OR 'mutagen testing'/exp OR 'carcinogen testing'/exp OR 'chromosome aberration'/exp	4,306,161
#114	#108 OR #113	5,540
#113	(#109 OR #110) AND (#111 OR #112)	3,542
#112	'dust exposure'/exp	4,192
#111	'dust'/exp OR 'dust*':ti,ab OR condensate:ti,ab OR condensates:ti,ab OR 'constituent*':ti OR 'wood based material*':ti OR 'drying fume*':ti OR 'wood processing*':ti,ab OR 'wood component*':ti,ab	107,490
#110	'wood'/exp OR 'wood*':ti OR 'tree'/exp OR oak:ti OR beech:ti OR ash:ti OR birch:ti OR teak:ti OR maple:ti OR chestnut:ti OR mahogany:ti OR ebony:ti OR elm:ti OR hickory:ti OR walnut:ti OR aspen:ti OR poplar:ti OR willow:ti OR acacia:ti OR eucalyptus:ti OR pine:ti OR fir:ti OR spruce:ti OR sequoia:ti OR cypress:ti OR larch:ti OR conifer:ti OR hemlock:ti OR 'sawmill*':ti OR 'plywood*':ti	110,943
#109	'hardwood'/exp OR 'hardwood*':ti OR 'hard wood*':ti OR 'softwood*':ti OR 'soft wood*':ti	726
#108	'wood dust'/exp OR 'wooddust*':ti OR 'wood dust*':ti OR 'wood process*':ti OR 'sawdust'/exp OR 'sawdust*':ti OR 'saw dust':ti	2,754
#107	#101 OR #106	2,073
#106	(#102 OR #103) AND (#104 OR #105)	1,294
#105	'dust exposure'/exp/mj	1,664
#104	'dust'/exp/mj OR dust:ti OR condensate:ti OR condensates:ti OR 'constituent*':ti OR 'wood based material*':ti OR 'drying fume*':ti OR 'wood processing*':ti OR 'wood component*':ti	56,134
#103	'wood'/exp/mj OR wood*:ti OR 'tree'/exp/mj OR oak:ti OR beech:ti OR ash:ti OR birch:ti OR teak:ti OR maple:ti OR chestnut:ti OR mahogany:ti OR ebony:ti OR elm:ti OR hickory:ti OR walnut:ti OR aspen:ti OR poplar:ti OR willow:ti OR acacia:ti OR eucalyptus:ti OR pine:ti OR fir:ti OR spruce:ti OR sequoia:ti OR cypress:ti OR larch:ti OR conifer:ti OR hemlock:ti OR 'sawmill*':ti OR 'plywood*':ti	67,319
#102	'hardwood'/exp/mj OR 'hardwood*':ti OR 'hard wood*':ti OR 'softwood*':ti OR 'soft wood*':ti	719

#101	'wood dust'/exp/mj OR 'wooddust*':ti OR 'wood dust*':ti OR 'wood process*':ti OR 'sawdust'/exp/mj OR 'sawdust*':ti OR 'saw dust':ti	1,379
#84	#10 NOT #82	705
#83	#22 NOT #82	2,829
#82	#31 OR #40 OR #45 OR #53 OR #58 OR #76 OR #81	1,004
#81	#78 OR #80	613
#80	#79 AND 'review'/it	104
#79	#24 AND #77	1,112
#78	#12 AND #77	540
#77	'diseases'/exp/mj OR 'health*':ti OR 'health hazard'/exp OR 'aetiology'/exp	20,192,608
#76	#73 OR #75	175
#75	#72 NOT #74	140
#74	(#60 OR #62 OR #64 OR #67 OR #69 OR #71) AND [humans]/lim	356
#73	(#60 OR #62 OR #64 OR #67 OR #69 OR #71) AND [animals]/lim	130
#72	#60 OR #62 OR #64 OR #67 OR #69 OR #71	496
#71	#70 AND 'Review'/it	41
#70	#22 AND #68	1,096
#69	#10 AND #68	417
#68	'in vitro study'/exp OR 'in vivo study'/exp OR 'vitro*':ti OR 'vivo*':ti	16,547,159
#67	#22 AND #65	9
#66	#10 AND #65	2
#65	'disease model'/exp OR 'disease model*':ti	187,383
#64	#63 AND 'Review'/it	5
#63	#22 AND #61	145
#62	#10 AND #61	57
#61	'experimental animal'/exp OR cat:ti OR cats:ti OR cattle:ti OR dog:ti OR dogs:ti OR 'fish*':ti OR goat:ti OR goats:ti OR 'guinea pig*':ti OR 'hamster*':ti OR 'horse*':ti OR 'monkey*':ti OR pig:ti OR pigs:ti OR 'rabbit*':ti OR rat:ti OR rats:ti OR sheep:ti	2,258,330
#60	#10 AND #59 OR (#22 AND #59 AND 'review'/it)	43
#59	'animal experiment'/exp OR 'animal experiment*':ti OR 'animal model*':ti OR 'mechanism of action*':ti OR 'mode of action*':ti	2,647,397
#58	#55 OR #57	252
#57	#56 AND 'Review'/it	37
#56	#22 AND #54	452
#55	#10 AND #54	223
#54	'toxicity and intoxication'/exp OR 'toxicology'/exp OR 'toxic*':ti OR 'adverse effect*':ti OR 'side effect*':ti OR (('mode*' NEAR/3 'action*'):ti,ab) OR 'mechanism*':ti,ab OR 'lavag*':ti,ab	3,840,913
#53	#47 OR #51 OR #52	79
#52	#22 AND #50 AND 'review'/it	5
#51	#10 AND #50	53
#50	#48 AND #49	2,487,243

#49	'toxicokinetic*':ti,ab OR 'absorp*':ti,ab OR 'distribut*':ti,ab OR 'metaboli*':ti,ab OR 'excret*':ti,ab OR 'clearanc*':ti,ab	3,333,171
#48	'biological phenomena and functions concerning the entire organism'/exp	21,257,199
#47	#10 AND #46	29
#46	'toxicokinetic*':ti OR 'absorp*':ti OR 'distribut*':ti OR 'metaboli*':ti OR 'excret*':ti	770,708
#45	#42 OR #44	205
#44	#43 AND 'Review'/it	22
#43	#24 AND #41	348
#42	#12 AND #41	193
#41	'occupational exposure'/exp AND 'expos*':ti	29,765
#40	#37 OR #39	218
#39	#38 AND 'Review'/it	26
#38	#24 AND #36	458
#37	#12 AND #36	196
#36	#32 OR #33 OR #34 OR #35	11,088,734
#35	'monitoring'/exp OR 'monitor*':ti	685,461
#34	'analysis'/exp OR 'quantit*':ti	9,390,626
#33	'measurement'/exp OR 'measure*':ti	2,212,296
#32	'sampling'/exp OR 'sampling*':ti OR 'sample*':ti	570,420
#31	#29 OR #30	192
#30	#24 AND #28 AND 'review'/it	31
#29	#12 AND #28	168
#28	#25 OR #26 OR #27	979,968
#27	'industr*':ti OR 'machine*':ti OR 'manufactor*':ti OR 'enterpris*':ti OR 'process*':ti OR 'production*':ti OR 'produce*':ti OR factory:ti OR 'factorie*':ti OR 'occurenc*':ti OR 'occuri*':ti	664,972
#26	'industry and industrial phenomena'/exp/mj	170,992
#25	'industry'/exp	342,923
#24	#22 AND #23	1,369
#23	#19 OR #20 OR #21	1,059,553
#22	#13 OR #18	3,833
#21	'occupational disease'/exp OR 'occupant* diseas*':ti OR 'work related diseas*':ti OR 'work related illness*':ti OR 'work-related diseas*':ti OR 'work-related illness*':ti OR 'occupat* illness*':ti	160,588
#20	'carpenter'/exp OR 'woodwork*':ti OR 'wood work*':ti OR 'employment'/exp OR 'employ*':ti OR 'work'/exp OR 'worker'/exp OR 'worker*':ti OR 'job exposure matrix'/exp OR 'job exposure matrix*':ti OR jem:ti OR 'furniture'/exp OR 'sawmill'/exp OR 'pulp and paper industry'/exp OR 'wood furniture*':ti OR 'cabinet manufactur*':ti OR 'plywood finish*':ti OR 'particle board*':ti OR 'sawmill*':ti OR 'planer mill*':ti OR 'joiner*':ti OR 'door manufactur*':ti OR 'wooden boat*':ti OR 'wood* floor*':ti OR 'model making*':ti OR 'carpent*':ti OR 'logging':ti OR 'construct*':ti OR 'forest*':ti OR 'timber*':ti	653,789

#19	'occupational exposure'/exp OR 'occupational*':ti OR 'expos*':ti	358,793
#18	(#14 OR #15) AND (#16 OR #17)	1,726
#17	'dust exposure'/exp	4,192
#16	'dust'/exp OR dust:ti	43,176
#15	'wood'/exp OR wood:ti OR 'tree'/exp OR oak:ti OR beech:ti OR ash:ti OR birch:ti OR teak:ti OR maple:ti OR chestnut:ti OR mahogany:ti OR ebony:ti OR elm:ti OR hickory:ti OR walnut:ti OR aspen:ti OR poplar:ti OR willow:ti OR acacia:ti OR eucalyptus:ti OR pine:ti OR fir:ti OR spruce:ti OR sequoia:ti OR cypress:ti OR larch:ti OR conifer:ti OR hemlock:ti	106,244
#14	'hardwood'/exp OR 'hardwood*':ti OR 'hard wood*':ti OR 'softwood*':ti OR 'soft wood*':ti	726
#13	'wood dust'/exp OR 'wooddust*':ti OR 'wood dust*':ti OR 'wood process*':ti OR 'sawdust'/exp OR 'sawdust*':ti OR 'saw dust':ti	2,754
#12	#10 AND #11	657
#11	#7 OR #8 OR #9	731,180
#10	#1 OR #6	1,607
#9	'occupational disease'/exp/mj OR 'occupat* diseas*':ti OR 'work related diseas*':ti OR 'work related illness*':ti OR 'work-related diseas*':ti OR 'work-related illness*':ti OR 'occupat* illness*':ti	110,432
#8	'carpenter'/exp/mj OR 'woodwork*':ti OR 'wood work*':ti OR 'employment'/exp/mj OR 'employ*':ti OR 'work'/exp/mj OR 'worker'/exp/mj OR 'worker*':ti OR 'job exposure matrix'/exp OR 'job exposure matrix*':ti OR jem:ti OR 'furniture'/exp/mj OR 'sawmill'/exp/mj OR 'pulp and paper industry'/exp/mj OR 'wood furniture*':ti OR 'cabinet manufactur*':ti OR 'plywood finish*':ti OR 'particle board*':ti OR 'sawmill*':ti OR 'planer mill*':ti OR 'joiner*':ti OR 'door manufactur*':ti OR 'wooden boat*':ti OR 'wood* floor*':ti OR 'model making*':ti OR 'carpent*':ti OR 'logging':ti OR 'construct*':ti OR 'forest*':ti OR 'timber*':ti	361,549
#7	'occupational exposure'/exp/mj OR 'occupational*':ti OR 'expos*':ti	326,741
#6	(#2 OR #3) AND (#4 OR #5)	749
#5	'dust exposure'/exp/mj	1,664
#4	'dust'/exp/mj OR dust:ti	26,899
#3	'wood'/exp/mj OR wood:ti OR 'tree'/exp/mj OR oak:ti OR beech:ti OR ash:ti OR birch:ti OR teak:ti OR maple:ti OR chestnut:ti OR mahogany:ti OR ebony:ti OR elm:ti OR hickory:ti OR walnut:ti OR aspen:ti OR poplar:ti OR willow:ti OR acacia:ti OR eucalyptus:ti OR pine:ti OR fir:ti OR spruce:ti OR sequoia:ti OR cypress:ti OR larch:ti OR conifer:ti OR hemlock:ti	61,959
#2	'hardwood'/exp/mj OR 'hardwood*':ti OR 'hard	719

#1	wood*’:ti OR ‘softwood*’:ti OR ‘soft wood*’:ti ‘wood dust’/exp/mj OR ‘wooddust*’:ti OR ‘wood dust*’:ti OR ‘wood process*’:ti OR ‘sawdust’/exp/mj OR ‘sawdust*’:ti OR ‘saw dust’:ti	1,379

11.2 Scopus

Chapter 4 – Occurrence, production and use / Chapter 6 – Occupational exposure data / Chapter 9 – Observations in humans (629 references):

(((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) or (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) or (TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*" OR "sawmill*"))) and (TITLE-ABS ("dust*" OR "dust expos*" OR "dustexpos*")))) and (TITLE("occupat* diseas*" OR "work relat* diseas*" OR "work relat* illness*" OR "work relat* sick*" OR "occupat* sick*" OR "occupat* ill*" or "occupat* expos*" OR "occupat*" OR "expos*" OR "carpenter*" OR "woodwork*" OR "wood work*" OR "employ*" OR "work*" OR "job expos*" OR "job expos* matri*" OR "jem" OR "furnitur*" OR "cabinet manufactur*" OR "plywood finish*" OR "particle board*" OR "sawmill*" OR "saw mill*" OR "planer mill*" OR "planermill*" OR "joiner*" OR "door manufact*" OR "wooden boat*" OR "wood* floor*" OR "model making*" OR "carpent*" OR "logging" OR "construct*" OR "forest*" OR "timber*" or "expos*" or "occupat* expos*" or "occupat*")))

Chapter 5 – Measurement and analysis of workplace exposure (43 references):

(((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) or (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) or (TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*"))) and (TITLE ("dust*" OR "dust expos*" OR "dustexpos*")))) and (TITLE("occupat* diseas*" OR "work relat* diseas*" OR "work relat* illness*" OR "work relat* sick*" OR "occupat* sick*" OR "occupat* ill*" or "occupat* expos*" OR "occupat*" OR "expos*" or "carpenter*" OR "woodwork*" OR "wood work*" OR "employ*" OR "work*" OR "job expos*" OR "job expos* matri*" OR "jem" OR "furnitur*" OR "cabinet manufactur*" OR "plywood finish*" OR "particle board*" OR "sawmill*" OR "saw mill*" OR "planer mill*" OR "planermill*" OR "joiner*" OR "door manufact*" OR "wooden boat*" OR "wood* floor*" OR "model making*" OR "carpent*" OR "logging" OR "construct*" OR "forest*" OR "timber*" or "expos*" or "occupat*

expos*" or "occupat*"))) and (title("sampl*" or "measur*" or "analysis*" or "analyz*" or "quantit*" or "monitor*"))

Chapter 7 – Kinetics (341 references):

(((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) OR (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) OR (TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*"))) AND (TITLE ("dust*" OR "dust expos*" OR "dustexpos*")))) AND (TITLE ("adapt*" OR "anhydrob*" OR "behavi*" OR "biologic* activ*" OR "biologic* vari*" OR "biomagnetis*" OR "chemoreact*" OR "chronobiolog*" OR "colon* collaps*" OR "cryobiolog*" OR "dying" OR "death" OR "ecolog*" OR "electrophys*" OR "evolut*" OR "evolv*" OR "excret*" OR "heredi*" OR "homeostas*" OR "life" OR "live" OR "metaboli*" OR "morphol*" OR "nutrit*" OR "pharmacol*" OR "physica*" OR "secret*" OR "thermogen*" OR "clearanc*" OR "distribut*" OR "toxicokin*" OR "absorp*"))) OR (((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) OR (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) OR (TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*"))) AND (TITLE ("dust*" OR "dust expos*" OR "dustexpos*")))) AND (TITLE ("toxicokinetic*" OR "absorp*" OR "distribut*" OR "metaboli*" OR "excret*")))

Chapter 8 – Effects in animals (107 references):

(((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) or (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) or (TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*"))) and (TITLE ("dust*" OR "dust expos*" OR "dustexpos*")))) and (title("animal experim*" or "animal test*" or "animal model*" or "mechanism of action*" or "mode of action*"))) or (((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) or (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) or

(TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*"))) and (TITLE ("dust*" OR "dust expos*" OR "dustexpos*")))) and (title("experiment* animal" or "cat" or "cats" or "cattle" or "dog" or "dogs" or "fish*" or "goat" or "goats" or "guinea pig*" or "hamster*" or "horse*" or "monkey*" or "primate*" or "pig" or "pigs" or "rabbit*" or "rat" or "rats" or "sheep"))) or (((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) or (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) or (TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*"))) and (TITLE ("dust*" OR "dust expos*" OR "dustexpos*")))) and (title("diseas* model*" or "sick* model*" or "ill* model*"))) or (((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) or (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) or (TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*"))) and (TITLE ("dust*" OR "dust expos*" OR "dustexpos*")))) and (title("in vitro" or "in vivo" or "vitro*" or "vivo*")))

Chapter 10 – Mode of action and pathogenesis (108 references):
((TITLE ("wood dust*" OR "wooddust*" OR "wood process*" OR "woodprocess*" OR "sawdust*" OR "saw dust*")) OR (((TITLE ("hardwood*" OR "hard wood*" OR "softwood*" OR "soft wood*")) OR (TITLE ("wood*" OR "tree*" OR "oak*" OR "beech*" OR "ash*" OR "birch*" OR "teak*" OR " maple* " OR "chestnut*" OR "mahogany*" OR "ebony*" OR "elm*" OR "walnut*" OR "hickory*" OR "aspen*" OR "poplar*" OR "willow*" OR "acacia*" OR "eucalyptus*" OR "pine*" OR "fir*" OR "spruce*" OR "sequoia*" OR "cypress*" OR "larch*" OR "conifer*" OR "hemlock*"))) AND (TITLE ("dust*" OR "dust expos*" OR "dustexpos*")))) AND (TITLE ("toxic*" OR "intoxic*" OR "overload*" OR "overdos*" OR "poison*" OR "venom*" OR "withdraw*" OR "sensitivit*" OR "advers* effect*" OR "side effect*" OR ("mode" W/2 "action*") OR "mechanis*" OR "lavag*"))

Appendix 2: Abbreviations

ACGIH: American Conference of Governmental Industrial Hygienists
ADCN: adenocarcinoma of the nasal cavity and paranasal sinuses
AGS: Ausschuss für Gefahrstoffe (Committee on Hazardous Substances)
AhR: aryl hydrocarbon receptor
AM: arithmetic mean
ATSDR: Agency for Toxic Substances and Disease Registry
BAL(F): bronchoalveolar lavage (fluid)
BHR: bronchial hyperreactivity
BOELV: binding occupational exposure limit value
BW: body weight
CA: chromosome aberration
CHO: Chinese hamster ovary
CI: confidence interval
COPD: chronic obstructive pulmonary disease
CWP: composite wood products
DECOS: Dutch Expert Committee on Occupational Standards
DFG: Deutsche Forschungsgemeinschaft (German Research Foundation)
DMSO: dimethyl sulfoxide
ECHA: European Chemical Agency
ES: effect size
$FEF_{25–75}$: Maximal Mid-Expiratory Flow Rate
FEF_{50}: forced expiratory flow at 50% of the vital capacity
FEF_{75}: forced expiratory flow at 75% of the vital capacity
FEV_1: forced expiratory flow volume in 1 second
FEV_1/FVC ratio: measurement of the amount of air forcefully exhaled
FVC: forced vital capacity
GM: geometric mean
GSD: geometric standard deviation
HSE: Health and Safety Executive (UK)
IARC: International Agency for Research on Cancer
ICD: International Code of Disease
ICD-O: International Classification of Diseases for Oncology
IEM: industry exposure matrix
IFA: Institut für Arbeitsschutz der Deutschen Gesetzlichen Unfallversicherung
IOM: Institute of Occupational Medicine
i.p.: intraperitoneal
IPF: idiopathic pulmonary fibrosis
IQR: interquartile range
ITAC: intestinal-type sinonasal adenocarcinoma
JEM: job–exposure matrix
LDH: lactate dehydrogenase
LLN: lower limit of normal
MDF: medium density fibreboard
MI: mitotic index
MMAD: mass median aerodynamic diameter
MMEFR: maximum mid-expiratory flow rate
MR: mean ratio
NADPH: nicotinamide adenine dinucleotide phosphate
NIOSH: National Institute of Occupational Safety and Health (US)

NOS: not otherwise specified
OECD: Organisation for Economic Co-operation and Development
OEL: Occupational exposure limit
OR: odds ratio
OSB: oriented strand board
OSHA: Occupational Safety and Health Administration (US)
p: p-value
PB: particle board
PBL: peripheral blood lymphocyte
PBS: phosphate buffered saline
PDL: population doubling level
PEF: peak expiratory flow rate
PEL: permissible exposure limit
PMR: proportional morbidity rate
ppm: parts per million
PRI: proliferative rate index
Ref: reference category
REL: recommended exposure limit
ROS: reactive oxygen species
RPM: residual particulate matter
RR: relative risk
SCE: sister chromatid exchange
SCOEL: Scientific Committee on Occupational Exposure Limits
SD: standard deviation
SIR: standardized incidence ratio
SMR: standardized mortality ratio
STEL: Short-term Exposure Limit
SWEA: Swedish Work Environment Authority
TLV: threshold limit value
TWA: time-weighted average
US EPA: US Environmental Protection Agency
VC: vital capacity
VOCs: volatile organic compounds
WEL: workplace exposure limit
WFI: wood fibre insulation board
WHO: World Health Organization
WS: wood solids

Appendix 3: Summary of epidemiological studies not included in Chapter 9

This appendix includes brief summaries of (mainly cross-sectional) studies that were not included in Chapter 9.

Within the tables, the studies are arranged by year of publication (from old to new). Separate tables are presented for studies in which the association of short-term (Table 28) and long-term (Table 29) wood dust exposure with irritation and sensitisation was examined. Seven studies are presented separately in Table 30 because they all used data from the same cohort in a Danish furniture industry. Note that other papers from the same study were published that did not meet the inclusion criteria in the current literature review (15, 77, 78, 261, 262). Table 31 presents three longitudinal studies examining the association between long-term wood dust exposure and carcinogenic effects. No studies were found in which short-term occupational wood dust exposure was examined in relation to carcinogenic effects. The remarks in Tables 28–31 contain only the most important limitations.

Table 28 Short-term occupational wood dust exposure and irritation and sensitisation effects

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
Andersen et al. (1977) (263)	**Type of study:** Cross-sectional **Country (region):** Denmark **Type of industry:** Furniture **Follow-up period:** Same day (i.e. exposure and health effects are measured on the same day) **Study period:** Nov 1974–Jan 1975 **Number of participants:** 68 workers **Number and origin of controls:** None **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	**Exposure measurement data:** Wood dust concentration >5mg/m^3 in 63% (25 participants) of all measurements. Range 0–80 mg/m^3. It is not reported which fraction was measured (most likely thoracic fraction). It is not reported for how many hours wood dust exposure was measured. During the measurements 41 workers were occupied with machine- and hand-sanding and 27 with work such as drilling, planing or sawing. The **average** dust concentrations in these two groups were 14.3 and 5.2 mg/m^3, respectively. The difference between these two groups was statistically significant. **Personal or stationary sampling of exposure levels:** Personal sampling. The investigation was always carried out in the afternoon, and was thus preceded by at least five hours' normal work. Distribution of the particles according to size in the categories <5, 6–10, 11–15 and above 16 µm in diameter was 33, 41, 11 and 15%, respectively. Although stationary sampling was done, results are not reported or included in analyses. **Number of measurements:** 68	**Types of health effect observed:** Airway symptoms, mucostasis and lung function **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** See stratified results. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Workers exposed to dust levels above 5 mg/m^3 (high) more frequently experienced inflammation of the middle ear and common colds. No significant difference in sinusitis, prolonged	Information on (non-)response (i.e. proportion of the people approached for this study that agreed to participate) is lacking. The measured fraction is not reported and it is unknown for how many hours wood dust exposure was measured. Analyses were not separated for treated (veneering) or untreated wood.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Methods of exposure measurement and analysis: The dust concentration was measured with a personal sampler (MSA model G) with a sampling head consisting of a filter holder (Sartorius SM 16517) and a filter (Sartorius SM 11304025) with a 0–8 µm pore size, placed in the breathing zone of the worker. Similar measurements with a high-volume sampler placed one metre from the work station were also taken, as well as temperature and humidity measurements. The filters were weighed after they had been acclimatised for 18 hours in the weighing room. **Job and exposure history (JEM, questionnaires, census data, etc):** 8 furniture plants (with at least 12 workers) The workers were initially questioned about their present and former working conditions. **Job classification:** Excluded workers engaged in varnishing, painting or similar occupations and those with a cold within the previous two weeks **Woodworking processes performed (e.g. sawing, drilling):** Sanding, veneering and assembling, among others. The different processes were often carried out in the same room.	colds, asthma or troubles such as itching or bleeding nose, frequent sneezing and nasal obstruction. Prevalence of mucostasis: 1.0–2.9 mg/m^3: 11% 3.0–4.9 mg/m^3: 25% 5.0–6.9 mg/m^3: 31% 7.0–9.9 mg/m^3: 46% ≥10 mg/m^3: 63% No difference was found in FEV_1 and $FEF_{25–75\%}$ between the high and low dust concentration exposure groups. **Did authors find a dose–response relationship (yes/no)?:** Yes.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Types of wood: Hard- and softwood: teak (hardwood) was most frequently processed, followed by oak (hardwood), chipboard and palisander, but mahogany, jakaranda, beech, ramin, motine, masonite and pine (softwood) were also processed. 37 workers processed only one material on the day of investigation, but 21 processed 2–4 different materials that day. Veneering took place. **Types of tool used:** During the measurements 41 workers were occupied with machine- and hand-sanders and 27 with work such as drills, planes or saws. **Types of medical examination:** The workers were initially questioned about their airway symptoms and smoking habits. The mouth and nose were then inspected using a head lamp and speculum, a forced expiratory spirogram was obtained, and finally nasal mucociliary clearance was measured with the saccharine/sky-blue technique. (The nasal mucociliary transit time for the 68 subjects is shown in Figure 3 in Andersen et al. (1977). The distribution curve had two maxima, one with 26 subjects (38%) at a transit time exceeding 40 minutes (defined as mucostasis) and one at		

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		a transit time of 14 minutes. The fastest transit time recorded was 6 minutes.) **Information on incidence:** unknown **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Prevalence using non-parametric tests (poorly specified) **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: smoking was examined.		
Holness et al. (1985) (264)	**Type of study:** Cross-sectional (cross-shift) study and retrospective longitudinal cohort study. **Country (region):** Canada **Type of industry:** Woodworking **Follow-up period:** One work shift **Study period:** The study took place on consecutive	**Exposure measurement data:** Woodworkers total dust, mean (SD): 1.83 (1.51) mg/m^3 Controls total dust, mean (SD): 0.43 (0.38) mg/m^3 Woodworkers respirable dust, mean (SD): 0.29 (0.31) mg/m^3 Controls respirable dust, mean (SD): 0.25 (0.33) mg/m^3 **Personal or stationary sampling of exposure levels:** Personal sampling of total dust and respirable dust (particulates less than 10 in aerodynamic diameter) (and formaldehyde) **Number of measurements:**	**Types of health effect observed:** Nasal cytology, respiratory symptoms (e.g. cough, rhinitis) and lung function **Single, short-term exposure or long-term exposure:** Short- and long-term (cross-shift and cumulative exposure) **Data on SIR/SMR/RR (no. observed/no. expected cases) and**	No information on response. Unclear whether all relevant confounders are included in the multiple regression analyses (whilst information on e.g. smoking and height is available). Unknown which type of wood was processed and

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Mondays in January and February 1984. **Number of participants:** 50 woodworkers **Number and origin of controls:** 50 hospital workers (34 housekeeping and 14 maintenance) **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	99 (one per study participant) **Methods of exposure measurement and analysis:** Gravimetry. Total dust: sampling head unclear; 37 mm PVC membrane filters, flow rate 2 L/min. Respirable dust: 10 mm nylon cyclone with PVC filters, flow rate 1.7 L/min. **Job and exposure history (JEM, questionnaires, census data, etc):** Symptom questionnaire, lung function, nasal cytology **Job classification:** Four companies located within the same geographical area of Toronto, Canada and under contract with the union local were randomly selected. Within each plant, all available workers in the sawing, sanding and assembly areas were studied. **Woodworking processes performed (e.g. sawing, drilling):** Sawing, sanding, assembly, laminating and gluing and miscellaneous, which included foremen and those in the shipping and receiving areas. **Types of wood:** Unknown and thus unknown whether untreated or treated wood was processed **Types of tool used:** Not specified **Types of medical examination:**	**95% CI or p-values:** No significant differences were seen between the nasal smears of the woodworkers and the controls. Nasal complaints were significantly increased. No significant association was found (results not reported) between exposure and change in lung function over the work shift. Association between cumulative dust exposure index and baseline lung function: Respirable Dust Index (average respirable dust level for area multiplied by years of exposure) adjusted for pack-years smoked: FVC (% predicted) -0.59 (p-value: 0.098 FEV_1 (% predicted): -0.91 (p-value: 0.001) FEF_{50} (% predicted): -1.47 (p-value:0.162)	whether it was treated or not. Results are potentially affected by healthy-worker effect (those who became sensitive to wood no longer work there). 38% of woodworkers used respirators, which was not accounted for in the analyses. Results on total dust are also presented. Unclear whether these results are comparable to inhalable dust (details on type of total dust sampler are missing).

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Respiratory symptoms by questionnaire. Nasal cytology by nose swabs. Maximal expiratory flow volume curves were obtained using a wedge spirometer. **Information on incidence:** The woodworkers did not have significantly lower baseline lung function than the hospital workers. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Differences between the exposed and control groups were compared using chi square, logit analysis, unpaired t tests and multiple regression analysis. Exposure–effect relationships were examined with unpaired t tests and regression analysis. **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: information was available on age, height, gender, smoking, exposure to formaldehyde, use of respirators and pack-years smoked.	FEF_{75} (% predicted): -2.89 (p-value: 0.001) Total Dust Index (average total dust level for area multiplied by years of exposure): FVC (% predicted): -0.12 (p-value:0.082) FEV_1 (% predicted): -0.17 (p-value: 0.008) FEF_{50} (% predicted): -0.17 (p-value: 0.396) FEF_{75} (% predicted): -0.60 (p-value: 0.003) Adjusted for pack-years smoked. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Mean (±SD) change in lung function during work shift (% change between afternoon value and morning value) in workers in exposure category low or high:	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
			Low (<0.2mg/m^3 respirable dust): FVC: -2.51 ± 4.64 FEV_1: -3.82 ± 4.95 FEF_{50}: -0.51 ± 11.46 FEF_{75}: -7.94 ± 18.68 High (≥0.2mg/m^3 respirable dust): FVC: -2.21 ± 2.98 FEV_1: -1.11 ± 2.98 FEF_{50}: 0.09 ± 6.84 FEF_{75}: 4.89 ± 14.58 **Did authors find a dose–response relationship (yes/no)?:** No.	
Vedal et al. (1986) (265)	**Type of study:** Cross-sectional **Country (region):** Canada **Type of industry:** Sawmill **Follow-up period:** One work shift **Study period:** 1982–1989 **Number of participants:** 334 (N=33 exposed)	**Exposure measurement data:** Total dust concentration (a 37-mm filter cassette was used): Range: undetectable to 6.0 mg/m^3 Mean: 0.46 mg/m^3 >2.0 mg/m^3: 13 workers **Personal or stationary sampling of exposure levels:** Personal sampling and area sampling only when worker was relatively immobile in job or if the weight and bulk of the sampler might have hindered a worker's safety **Number of measurements:**	**Types of health effect observed:** Lung function and respiratory complaints **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** None	Total dust was examined. Unclear whether the processed wood was untreated or whether workers were exposed to other occupational substances. Due to cross-sectional nature

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Number and origin of controls: N=301 (exposure level of <1.0mg/m^3) **Name of study / cohort:** Unknown **Earlier publications on the same cohort:** None known	78 (46 by area and 32 by personal sampling). Workers who had the same job title and job location were considered to have the same level of dust exposure. **Methods of exposure measurement and analysis:** Samplers consisted of filter cassettes 37-mm in diameter with a 0.8 µm pore size through which air was pumped at 2 L/min. Gravimetric analyses: Dust concentration was calculated by change in weight of the filter divided by the volume of air sampled. **Job and exposure history:** 334 workers were assigned a level of exposure based on the 78 measurements. **Job classification:** See above. **Woodworking processes performed:** Unknown **Types of wood:** Mainly western red cedar (softwood). Unknown whether treated. **Types of tool used:** Unknown **Types of medical examination:** Questionnaire and spirometric testing. Spirometry was performed at the work site using a 13.5 L Collins waterseal spirometer. **Information on incidence:** Unknown	**Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Association between categorical total dust levels and FEV_1 (ml) (adjusted for height, age, race, cigarette smoking): 1.0–1.9mg/m^3: -148 (not significant, i.e. p≥0.05) (N=20) ≥2.0mg/m^3: -337 (p<0.05) (N=13) (reference category: <1.0mg/ m^3; N=301) The association between categorical total dust levels and FVC; ml): 1.0–1.9mg/m^3: -187 (p≥0.05) (N=20) ≥2.0mg/m^3: -412 (p<0.05) (N=13) (reference category: <1.0mg/ m^3; N=301) No associations were found with $FEF_{25–75\%}$	unclear whether healthy-worker effect affected the results.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Mortality prevalence/percentage of the control group or general population (background data): Not reported **Type of statistical analyses:** Multiple linear regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: adjusted for height, age, race, cigarette smoking.	(maximum midexpiratory flow rate; ml/sec) or FEV_1/FVC (%)). Chronic cough, dyspnoea, persistent wheeze and asthma were not related to dust exposure (15% of those exposed to ≥2.0mg/m^3 had asthma). **Did authors find a dose–response relationship (yes/no)?:** Yes.	
Pisaniello et al. (1991) (266)	**Type of study:** Cross-sectional **Country (region):** Australia **Type of industry:** Furniture manufacture (15 furniture factories) **Follow-up period:** Same day exposure and health measurements **Study period:**	**Exposure measurement data:** Inhalable wood dust, GM: 2.9 mg/m^3, range 0.4–24 mg/m^3 **Personal or stationary sampling of exposure levels:** Personal sampling **Number of measurements:** Two separate samples of inhalable dust were taken, one in the morning and one in the afternoon. Typically these were each about 3 hours in duration and were combined on a time-weighted basis to give a TWA concentration for each worker. Most workers	**Types of health effect observed:** Respiratory symptoms **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Results from the logistic regression analysis were	Results of the logistic regression analyses are not shown in the article.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	May and September 1989 **Number of participants:** 168 furniture factory employees **Number and origin of controls:** 46 hospital maintenance workers **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	spent their whole day engaged in repetitive or similar woodworking tasks. **Methods of exposure measurement and analysis:** Personal exposure to inhalable wood dust was evaluated by using IOM or 7-hole sampling heads located on the workers collar. Sampling heads contained 25 mm Gelman A/E glass fibre filters or DM-800 membrane filters (25 mm, 0.8um). Gravimetric analyses were performed. **Job and exposure history:** Determined by questionnaire. **Job classification:** Low exposure: TWA inhalable dust exposure ≤2mg/m^3 Medium exposure: 2–5 mg/m^3 High exposure (N=51): ≥5mg/m^3 **Woodworking processes performed:** Wood machining and assembly/cabinetmaking; to a lesser extent cleaning, wood gluing, stitching of veneer **Types of wood:** Both hardwood (Tasmanian oak, teak, nyatoh) and softwood (radiata pine), including plain or veneered reconstituted softwood timber panels (both treated and untreated) **Types of tool used:**	not reported in the article. Authors concluded: 'When allowance was made for factors such as age, smoking, previous asthma, and childhood nasal problems by the use of logistic regression, there was no substantial alteration to the trends that are evident from Table II'. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** A wide range of respiratory symptoms were examined. Dose–response relationship was found only for certain nasal symptoms (whether differences were significant was not examined).	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Unknown **Types of medical examination:** British Medical Research Council's 1976 respiratory questionnaire. In addition, there were questions on skin, eye and nasal effects. **Information on incidence:** A wide range of health symptoms were examined. For instance, of all examined woodworkers 32% usually had a cough during the day, 25% regularly had a dry nose and 51% regularly had a blocked nose. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported. **Type of statistical analyses:** Logistic regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: adjusted for age, smoking, previous asthma, and childhood nasal problems.	**Did authors find a dose–response relationship (yes/no)?:** No.	
Pisaniello et al. (1995) (267)	**Type of study:** Cross-sectional **Country (region):** Australia (Adelaide) **Type of industry:** Furniture	**Exposure measurement data:** 8-hour TWA exposure to inhalable wood dust. The daily TWA exposure to inhalable wood dust ranged from 0.5 mg/m^3 to 15 mg/m^3, with an overall AM of 3 mg/m^3.	**Types of health effect observed:** Metaplasia **Single, short-term exposure or long-term exposure:**	Results of the logistic regression analyses are not shown in the article.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Follow-up period: Same day exposure and health measurements **Study period:** May and September 1989 **Number of participants:** 50 furniture workers with 10 years or more of woodworking experience **Number and origin of controls:** 50 controls selected from maintenance workers from the hospital and university staff (and matched according to age, sex and smoking status) **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:**	**Personal or stationary sampling of exposure levels:** Personal and stationary sampling **Number of measurements:** 50 **Methods of exposure measurement and analysis:** IOM or 7-hole sampling heads were located on the workers' collars. The sampling heads contained 25 mm Gelman A/E glass fibre filters or DM-800 membrane filters (25 mm, 0.8 µm). The authors noted that although glass fibre filters are recommended for high dust loadings and were used for the great majority of measurements, the DM-800 filters gave equivalent results. However, more care in handling was required with the latter type of filter, which is more suitable for the measurement of respirable wood dust. Filters were weighed before and after sampling with a Mettler ME30 microbalance. IPM sampling was undertaken at a flow rate of 2 L/min using SKC. Supplementary measurements of airborne wood dust included area IPM sampling and personal and area RPM monitoring. Casella Higgins cyclones, fitted with Gelman DM-800 filters and operating at a flow rate of 1.9 L/mm, were used for the RPM work.	Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Results from the logistic regression analysis were not reported in the article. Authors concluded: 'no statistically significant relationship was demonstrated between the occurrence of metaplasia and current wood dust exposure, smoking status, age, years of experience or the type of wood used during the subject's working life'. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Prevalence rate ratio >2/≤2 mg/m^3	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Pisaniello et al. (1991) (266)	**Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaires **Job classification:** furniture workers **Woodworking processes performed (e.g. sawing, drilling):** Sawing, sanding, assembly/cabinetmaking, cleaning **Types of wood:** Tasmanian oak, teak, nyatoh and the softwood radiata pine Wood species use was mixed on certain days, but the furniture workers usually worked with only one category of wood material – reconstituted softwood panels (plain or veneered), solid hardwood or solid softwood. **Types of tool used:** Unknown. **Types of medical examination:** Nasal examination (anterior rhinoscopy) was performed and brush cytological specimens were obtained from the anterior region of the middle turbinate. **Information on incidence:** 23 woodworkers and 10 controls had cuboidal metaplasia. **Mortality prevalence/percentage of the control group or general population (background data):**	(unadjusted for confounders): Cuboidal metaplasia: 1.3 (not significant) Squamous metaplasia: 1.0 (not significant) **Did the authors find a dose–response relationship (yes/no)?:** No.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Not reported **Type of statistical analyses:** Prevalence ratio and logistic regression analyses **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Logistic regression analyses were adjusted for confounders. Prevalence ratios were not. With very few exceptions, respiratory protection was not worn when woodworking.		
Åhman et al. (1996) (268)	**Type of study:** Cross-sectional **Country (region):** Sweden **Type of industry:** Woodwork teachers **Follow-up period:** One work week **Study period:** Unknown **Number of participants:** 39 **Number and origin of controls:** 32 (other school personnel) **Name of study/ cohort:**	**Exposure measurement data:** Personal sampling (mg/m^3): Mean (range) total dust: 0.57 (0.12–1.18) Mean (range) respirable dust: 0.10 (0.02–0.21) Area sampling (mg/m^3): Mean (range) total dust: 0.38 (0.06–1.24) (respirable dust not measured by area sampling). **Personal or stationary sampling of exposure levels:** Personal and area sampling **Number of measurements:** 39 **Methods of exposure measurement and analysis:** Airborne dust (total dust and respirable dust) was sampled during a whole working day with personal equipment. Area sampling of	**Types of health effect observed:** Nasal function **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** See below. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):**	Small population is examined. Unclear how high the non-response was in the control group. Healthy-worker effect cannot be excluded.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Unknown **Earlier publications on the same cohort:** Ahman et al. (1995) (269)	total dust was also performed with the equipment on a stand in the middle of the room 1–5 m above the floor. Total dust was collected with a membrane filter (Millipore, cellulose ester) mounted in a 37-mm cassette, and respirable dust was sampled with a size-selective particle cyclone sampler (Casella). For all dust sampling, the flow through the filters was about 2 L/min. Gravimetric analysis of the dust was carried out according to Swedish standards. While these dust measurements were going on, short-term exposure (10 minutes) to dust was repeatedly examined with a direct reading instrument (Sibata). **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire. **Job classification:** 39 selected woodwork teachers employed full time and for at least 3 years and 32 control subjects (other school personnel) were examined at the beginning and end of a working week. **Woodworking processes performed (e.g. sawing, drilling):** Various (sawing, cleaning, operating boring machine, grinding machine, etc.), see Ahman et al. (1995) (269)	There were significant but rather weak correlations between the percentage of respirable dust in the total dust and the change from Monday to Thursday in nasal obstruction (Spearman's p = -0.32, p <0.055), runny nose (Spearman's p = +0.45, p <0.01), and itching nose (Spearman's p = +0.41, p <0.01). Otherwise, there was no significant correlation between symptoms and test results and measured levels of total or respirable dust (and terpenes). **Did authors find a dose–response relationship (yes/no)?:** Yes.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Types of wood: Pine was the most frequently used wood, being used daily by almost all teachers. The teachers often also used lime, birch, juniper, alder, plywood and chipboard. Exotic woods such as teak, mahogany and jelutong were used infrequently. **Types of tool used:** Various **Types of medical examination:** The tests of nasal physiology were performed at Huddinge Hospital on the Monday morning after two days without exposure to wood dust, and the same tests were repeated on the Thursday afternoon after work. **Information on incidence:** In contrast to the control subjects, the woodwork teachers had more nasal symptoms on the Thursday afternoon than on the Monday morning, especially those working in rooms without mechanical ventilation. Their mucociliary clearance worsened during the week (mean increase 4 min, P <0.001). A small impairment of olfactory function was also found, but their rhinomanometric values did not change significantly. **Mortality prevalence/percentage of the control group or general population (background data):**		

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Not reported **Type of statistical analyses:** Differences in test results between the groups were evaluated with Student's t test, and the correlations between tests and measured work environmental factors were evaluated from linear regression. **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** The control subjects were matched to the woodwork teachers for school, sex, age, height and smoking habits. School personnel with frequent exposure to irritants or previous work as woodwork teachers were excluded.		
Dahlqvist et al. (1996) (270)	**Type of study:** Cross-sectional (experimental) **Country (region):** Sweden **Type of industry:** sawmill sawing Scots pine (Pinus sylvestris) **Follow-up period:** Same day (dust and lung function measurement before	**Exposure measurement data:** Sampling duration 5 hours. TWA. Median (interquartile range) total dust concentration (mg/m^3) with (N=10) and without particle filter (N=9): 0.04 (0.04–0.06) mg/m^3 and 0.13 (0.08–0.16) mg/m^3, resp. **Personal or stationary sampling of exposure levels:** Personal sampling; sampling point was located inside the respirator and on the shoulder of the subject. **Number of measurements:** 19	**Type of health effects observed:** Nasal lavage, spirometry, measurements of the transfer factors of the lung, and bronchial provocation with methacholine **Single, short-term exposure or long-term exposure:** Short-term	No information on response of individuals to the questionnaire. Note that the wood dust concentration was collected inside the respirator.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	and after 5 hours of exposure). **Study period:** Unknown. Participants were sourced close to the area where timber was sawn 2–3 days before spending 5 hours in the sawmill. **Number of participants:** 19 volunteers – men who gave negative answers to questions about airway or lung disease, atopy and being non-smokers during the past six months, and had never worked as a sawyer or with wood processing. **Number and origin of controls:** No controls	**Methods of exposure measurement and analysis:** Total dust was collected on cellulose acetate filters mounted in a 25 mm sampling head. Endotoxin was sampled on IOM samplers (SKC, PA, USA). The samplers were connected to battery-powered pumps carried by the men, with a flow rate of about 2 L/min. During sampling for dust, the sum of terpenes and endotoxin was measured for every subject for the whole exposure time (5 hours). The filters were weighed before and several days after sampling. The detection limit for total dust was 0–1 mg, corresponding to 0.01–0.02 mg/m^3. Baseline unexposed values were obtained two to three days before the exposure. **Job and exposure history (JEM, questionnaires, census data, etc):** Not applicable (experimental) **Job classification:** Not applicable (experimental) **Woodworking processes performed (e.g. sawing, drilling):** Sawing **Types of wood:** Scots pine (Pinus sylvestris) **Types of tool used:** Not reported **Types of medical examination:**	**Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** No difference was observed in the functional respiratory parameters after exposure, but the cell and interleukin-6 concentration in nasal lavage was significantly elevated in both groups and more markedly so in the group using respirators without filters. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** No stratified results were reported. **Did authors find a dose–response relationship (yes/no)?:** No.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Name of study/ cohort: Unknown **Earlier publications on the same cohort:** None known	Physical examinations were performed. **Information on incidence:** Median (interquartile range) number of cells/ml nasal savage fluid and the concentration of IL-6 in the nasal savage fluid before and after 5 hours of exposure in the sawmill are reported. Median (interquartile range) of lung function and bronchial reactivity after exposure (as % of values before exposure) for subjects with and without a particle filter are reported. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Mann-Whitney test and Spearman's rank correlation analysis **Whether authors took into account for other risk factors, such as smoking and mixed exposure to other compounds:** Non-smokers were examined. There were no significant differences in age, height, or weight between the group with and without a particle filter.		
Mandryk et al. (1999) (271)	**Type of study:** Cross-sectional (cross-shift) **Country (region):** Australia	**Exposure measurement data:** Sampling duration 6–8 hours. *Inhalable dust* concentration (mg/m^3): In the four sawmills (N=93):	**Types of health effect observed:** Lung function	Information on response is lacking and selection of control group is poorly described.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Type of industry: Four sawmills, a wood chipping mill, and five joineries **Follow-up period:** Same day (dust and lung function measurement during the same work shift) **Study period:** 1996–1997 **Number of participants:** 168 woodworkers (only men) **Number and origin of controls:** 30 maintenance workers **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	AM = 4.81; GM (GSD) = 1.59 (3.19); Range = 0.25–74.05 In the woodchipping mill (N=4): AM = 3.17; GM (GSD) = 2.86 (1.66); Range = 1.80–5.66 In the five joineries (N=66): AM = 7.59; GM (GSD) = 3.68 (3.67); Range = 0.21–50.65 *Respirable dust* concentration (mg/m^3) in the four sawmills (N=31): AM = 0.37; GM (GSD) = 0.29 (2.17); Range = 0.05–1.05 In the woodchipping mill (N=4): AM = 0.28; GM (GSD) = 0.26 (1.71); Range = 0.12–0.40 In the five joineries (N=39): AM = 0.67; GM (GSD) = 0.48 (1.70); Range = 0.03–2.55 Overall, 62% of the personal inhalable dust exposures exceeded the standards at the time of the study in Australia (hardwood: 1 mg/m^3, softwood: 5 mg/m^3). **Personal or stationary sampling of exposure levels:** Only personal inhalable and respirable dust sampling was carried out. Inhalable dust sampling: all job titles (woodworking) and all workers at each worksite were sampled except in Joinery 1,	**Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Pearson's R between percentage *predicted* lung function indices and personal *inhalable* dust exposures (log transformed) (unadjusted): Joinery (N=63): FEV_1: -0.65 (p<0.001) FVC: -0.57 (p<0.001) FEV_1/FVC: -0.34 (p<0.01) FEF25–75%: -0.63 (p<0.001) Sawmill/chip mill (N=105): FEV_1: -0.26 (p<0.01) FVC: not significant FEV_1/FVC: 0.22 (p<0.05) FEF25–75%: not significant	Analyses are poorly explained. Healthy-worker effect cannot be excluded.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		where a random selection of workers were sampled. Respirable dust sampling: only a random selection of each job title **Number of measurements:** 168 **Methods of exposure measurement and analysis:** Casella 7-hole samplers (modified UKAEA) and Higgins-cyclone samplers were used for inhalable dust and respirable dust sampling, respectively. Polycarbonate filters (25 mm, 0.8 µm, Millipore, Ventura, CA) were used. The sampling was conducted according to the specifications of Standards Australia. Gravimetric analyses. **Job and exposure history (JEM, questionnaires, census data, etc):** Unknown **Job classification:** Unknown **Woodworking processes performed (e.g. sawing, drilling):** Not specifically reported (products produced by sawmills: green timber, woodchips, kiln dried timber for flooring. Joineries: staircases, window frames, mouldings, handrails, pantry cupboard doors). **Types of wood:**	Total (N=63+105): FEV_1: -0.30 (p<0.001) FVC: -0.27 p<0.001) FEV_1/FVC: not significant FEF25–75%: -0.29 (p<0.001). Pearson's R between percentage *cross-shift change in* lung function indices and personal *inhalable* dust exposures (log transformed) (adjusted for age, height, smoking): Joinery (N=63): VC: -0.66 (p<0.001) FEV_1: 0.55 (p<0.001) FVC: 0.67 (p<0.001) FEF25–75%: -0.33 (p<0.01) PEF: 0.43 (p<0.001) Sawmill/chip mill (N=105): VC: 0.26 (p<0.01) FEV_1: -0.26 (p<0.01) FVC: -0.25 (p<0.01) FEF25–75%: 0.22 (p<0.05) PEF: -0.25 (p<0.05)	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		None of the worksites used chemical preservatives during wood processing (untreated). All sawmills and the woodchipping mill used hardwood (eucalypt). Most joineries used both hard- and softwood, including MDF. One used only softwood (western red cedar) and one used mostly hardwood (e.g. Tasmanian oak, brush oak). Results for the different work sites are reported in the results section. **Types of tool used:** Not reported **Types of medical examination:** Spirometry before and after a work shift; questionnaire to obtain respiratory, eye and nasal symptom prevalence data. **Information on incidence:** Not reported. The mean percentage cross-shift decrease in lung function was higher for woodworkers than controls. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Pearson's R and multiple regression analyses **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: age, height and smoking	Total (N=63+105): VC: -0.29 ($p<0.001$) FEV_1: 0.21 ($p<0.01$) FVC: 0.29 ($p<0.001$) FEF25–75%: not significant PEF: 0.16 ($p<0.01$) The association between respirable dust exposure and cross-shift change in lung function is not included here but is reported in the study. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** No stratified results reported **Did authors find a dose–response relationship (yes/no)?:** Yes.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
Mandryk et al. (2000) (272)	**Type of study:** Cross-sectional (cross-shift) **Country (region):** Australia **Type of industry:** Four sawmills (including two groups of sawmill workers: green mill and dry mill workers) **Follow-up period:** same day (dust and lung function measurement during the same work shift) **Study period:** 1996–1997 **Number of participants:** 93 sawmill workers **Number and origin of controls:** 30 maintenance workers **Name of study/ cohort:** Not known	**Exposure measurement data:** Sampling duration 6–8 hours. *Inhalable dust* concentration (mg/m^3) in the three green mills (N=56): AM = 6.26; GM (GSD) = 1.52 (3.71); Range = 0.25–74.05 In the two dry mills (N=37): AM = 2.62; GM (GSD) = 1.71 (2.46); Range = 0.55–11.22 *Respirable dust* concentration (mg/m^3) in the three green mills (N=20): AM = 0.27; GM (GSD) = 0.19 (2.26); Range = 0.05–0.98 n the two dry mills (N=11): AM = 0.49; GM (GSD) = 0.46 (1.43); Range = 0.28–1.05 About 70% and 50% of the inhalable dust exposures at the dry mills and the green mills, respectively, exceeded the occupational exposure limit of 1 mg/m^3 (8-hour TWA) for hardwood dust in Australia at that time. High dust exposures were observed at one of the examined sawmill, most likely due to ageing equipment, poor maintenance of the local exhaust ventilation systems, and leakage of dust from the joints of the central exhaust ventilation system into the working area.	**Types of health effect observed:** Respiratory complaints and lung function **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Association between inhalable dust and frequent headache (adjusted for age and smoking) (OR (95% CI)): 5.72 (1.55–20.90). Pearson's R between percentage *cross-shift change* in lung function indices and personal *inhalable* dust exposures (log transformed) (adjusted for age, height, smoking) in green mill workers (N=53) compared with	Healthy-worker effect cannot be excluded. Information on response is lacking and selection of control group is poorly described. Analyses are poorly explained.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Earlier publications on the same cohort: Mandryk et al. (1999) (271) (a subpopulation was examined in Mandryk et al. (2000) (272)).	None of the woodworkers at sawmills wore respirators. **Personal or stationary sampling of exposure levels:** Only personal inhalable and respirable dust sampling was carried out. Inhalable dust sampling: all job titles (woodworking) and all workers at each worksite were sampled. Respirable dust sampling: only a random selection of each job title **Number of measurements:** 93 **Methods of exposure measurement and analysis:** Casella 7-hole samplers (modified UKAEA) and Higgins-cyclone samplers were used for inhalable dust and respirable dust sampling, respectively. Polycarbonate filters (25 mm, 0.8 µm, Millipore, Ventura, CA) were used. The sampling was conducted according to the specifications of Standards Australia. Gravimetric analyses. **Job and exposure history (JEM, questionnaires, census data, etc):** Unknown **Job classification:** Unknown	controls: VC: 0.97 ($p<0.001$) FEV_{25-75}: 0.95 ($p<0.001$). Results of the association between wood dust exposure and change in lung function for *dry mill workers* are not included in the article because no significant associations were found. Association between respirable dust exposure and change in lung function was not examined. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** None reported **Did authors find a dose–response relationship (yes/no)?:** Yes.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Woodworking processes performed (e.g. sawing, drilling): Not specifically reported (products produced were green timber, woodchips and kiln-dried timber; debarking was normally carried out at logging sites in the forest) **Types of wood:** None of the worksites used chemical preservatives during wood processing. All sawmills used hardwood (eucalypt). Products processed: green timber, woodchips and kiln-dried timber **Types of tool used:** Not reported **Types of medical examination:** Spirometry before and after a work shift; questionnaire to obtain respiratory, eye and nasal symptom prevalence data **Information on incidence:** Green mill workers more often had work-related symptoms than dry mill workers. Green mill workers had slightly lower FEV1/FVC, the percentage predicted VC, FVC, FEV1 and FEF25±75% compared with dry mill workers. When compared with the controls, both green mill and dry mill workers showed a significantly low mean percentage predicted lung function.		

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Mortality prevalence/percentage of the control group or general population (background data): Not reported **Type of statistical analyses:** Pearson's R and multiple regression analyses **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: age, height, smoking		
Thetkathuek et al. (2010) (273)	**Type of study:** Cross-sectional **Country (region):** Thailand **Type of industry:** 8 factories that used natural rubber tree wood **Follow-up period:** Same day **Study period:** April–October 2007 **Number of participants:** N=533 working in departments that processed rubber wood **Number and origin of controls:**	**Exposure measurement data:** TWA (a full-shift single sample of wood dust was collected at the workstations of selected workers in each department (92.3% worked ≤8 hours)). Examined fraction is not reported. 201 workers and 0 controls were exposed to >5mg/m^3. **Personal or stationary sampling of exposure levels:** Stationary samples only (it was assumed that airborne wood dust was dispersed homogeneously across each department): Highest mean was observed in department 'Sanding': 5.08 (2.95) mg/m^3 Lowest mean was observed in department 'Cutting': 3.01 (0.69) mg/m^3 Mean in department 'Office/clerical': 2.11 (1.13) mg/m^3 **Number of measurements:**	**Types of health effect observed:** Lung function **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** A negative association was found between mean dust exposure level and FVC (-0.72 to 0.011) but not for FEV_1 (p=0.071). In addition, dust level was negatively correlated with FEV_1/FVC (-0.584	Response is not reported. Only stationary measurements are reported. Results of regression analyses are not fully reported and it is not reported which fraction is measured.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	N=152. Office workers served as controls. **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	58 samples were collected from the 8 examined factories (3–12 samples per department type). **Methods of exposure measurement and analysis:** Sampling device had low-flow pumps (SKC model 224-PCXR4), IOM Samplers (SKC Model 225-70A), and 25 mm PVC filters (SKC, Inc). The device was assembled on a stand, 1.6 metres high. The flow rate of the pump was 2L/min. Gravimetric analysis of the sample filters was performed using NIOSH Method No. 0500. **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire **Job classification:** Only departments that had rubber wood dust were included. Office workers served as controls. **Woodworking processes performed (e.g. sawing, drilling):** Department types were: transfer of raw material (wood), cutting, drilling, planing, assembly, finishing and office/clerical. **Types of wood:** Untreated (workers who used glue or solvents were excluded) rubber wood (hardwood) **Types of tool used:**	to -0.360). Reported duration of mask use was not correlated with FVC and FEV_1 and FEV_1/FVC. Adjusted for gender, age, height, duration of smoking and mask use. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Not available **Did authors find a dose–response relationship (yes/no)?:** No.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Unknown **Types of medical examination:** Spirometry **Information on incidence:** Lung function capacity: mean (SD): 84.33% (13.41); minimum–maximum: 49.8–132.56% **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Multiple linear regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Workers who used glue or solvents were excluded. In analyses, adjustments were made for the duration of use of mask.		
Górny and Gołofit-Szymczak (2020) (274) (information regarding applied methods was in part	**Type of study:** Cross-sectional **Country (region):** Poland **Type of industry:** 10 wood pellet production facilities **Follow-up period:** Unknown when questionnaire was completed	**Exposure measurement data:** Inhalable wood dust concentration (workplace measurements and outdoor measurements were performed), AM (±SD): Workplace: 8.52 (±13.67) Outdoor: 0.02 (±0.01) **Personal or stationary sampling of exposure levels:** Stationary samples at workplace during typical occupational activities and outside each studied plant building to determine the	**Types of health effect observed:** Respiratory (dry cough, cough with phlegm and wheezing) and irritative complaints (runny nose, throat irritation, nose irritation, eye irritation and skin irritation)	(Non-)response is not reported. Other information is also missing, such as the date of study and follow-up period. A large number of analyses were performed.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
derived from (275))	**Study period:** May–September (year unknown) **Number of participants:** 28 workers (22 out of 28 were under 35 years of age) **Number and origin of controls:** No controls **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** Górny et al. (2019) (275)	so-called outdoor (atmospheric) background contamination. During the measurements, all sampling instruments were placed at a height of 1–1.5 m above the floor or ground level to simulate aspiration from the human breathing zone. **Number of measurements:** 40 (workplace) and 20 (background) samples **Methods of exposure measurement and analysis:** Particulate (wood dust) measurements were performed using conical inhalable sampler (CIS) equipped with APEX pumps (Casella Measurements Inc., Bedford, Great Britain) operated at a flow rate of 1.2 L min^{-1} for 8 h as well as 3.5 L min^{-1} for 6 h, respectively. All collected samples were analysed in duplicate. The particulates were collected on 37 mm Teflon filters with 2 µm pore size (SKC Ltd, Eighty-four, PA, USA). The wood dust mass was gravimetrically determined by weighting the filters before and after sampling with an ultra-microbalance (model XP2U, Mettler Toledo, Zürich, Switzerland), following a 24-hour equilibration period at a constant air temperature and humidity (22 ± 3 °C and 45 ± 5%, respectively). In addition, particulate wood dust measurements were performed using Grimm	**Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** See below. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** No significant association was observed between workers exposed to wood dust (i.e. ≤3mg/m^3 vs >3mg/m^3) and respiratory and irritative complaints. Results are poorly presented. **Did authors find a dose–response relationship (yes/no)?:** No.	Only stationary measures were examined. Only a possible association between two occupational wood dust exposure categories (i.e. ≤3mg/m^3 vs >3mg/m^3) was examined (no linear relationship was examined and no other wood dust exposure categories were examined). Results are not clearly reported (the meaning of the term 'appearance probability' is unclear). Few covariates are examined. There is a wide variety of covariables available in their data, but they are not included

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		aerosol spectrometer (275). It assumed that these measurement data were not used in the current analyses and they are therefore not reported in the current report. **Job and exposure history (JEM, questionnaires, census data, etc):** Unknown **Job classification:** Job titles not incorporated in analyses **Woodworking processes performed (e.g. sawing, drilling, etc):** During an 8-h shift, the main tasks of employees were: supplying sawdust or shavings to the production hall, manual or mechanical loading of same onto belt conveyors, pelletiser work inspection, hand-held wood dust removal from the mechanical elements of the production line, product quality control, pellet packaging, and preparation of the pallets with sorted products for transport outside the plant. **Types of wood:** Sawdust and shavings from both coniferous (softwood) and deciduous (hardwood) trees **Types of tool used:** See 'Woodworking processes performed'. **Types of medical examination:** Questionnaire **Information on incidence:**		in one model (most likely explained by the fact that the number of respondents is small).

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Lung function capacity: mean (SD): 84.33% (13.41). minimum–maximum: 49.8–132.56% **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Logistic regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Information on smoking was available but not incorporated in the analyses.		
Asri et al. (2020) (276)	**Type of study:** Cross-sectional **Country (region):** Peninsular Malaysia (Kelantan and Perak states) **Type of industry:** Sawmill, furniture factory, rice mill **Follow-up period:** Same day **Study period:** Not reported **Number of participants:**	**Exposure measurement data:** Total inhalable dust concentration: Sawmill workers (N=17): 2.4×10^3 µg/m^3 (IQR: 1.1×10^3 – 5.8×10^3) Furniture factory workers (N=24): 1.0 µg/m^3 (IQR: 0.3–6.7) Rice mill workers (N=36): 1.1×10^3 (0.9×10^3 – 2.1×10^3 Non-exposed: 0.5 (0.1–2.8×10^2) **Personal or stationary sampling of exposure levels:** Personal sampling **Number of measurements:** 117	**Types of health effect observed:** Lung function (FVC measured and predicted, FEV_1 measured and predicted, % FEV1/FVC measured and predicted) **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:**	Small samples. 'Exposed' workers were exposed to organic dust, including wood dust. Analyses were performed only for 'exposed' individuals taken together, not separately for wood dust-exposed individuals.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	77 exposed and 39 non-exposed **Number and origin of controls:** N=39; administrative staff **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	**Methods of exposure measurement and analysis:** Health and Safety Executive's Methods for the Determination of Hazardous Substances (MDHS) 14/4 used as guidance on general methods for sampling and gravimetric analysis. The total inhalable dust concentration was collected using an IOM personal airborne sampler loaded with glass microfibre filter connected to a sampling pump via tygon tubing, which was attached to the workers. **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire **Job classification:** Questionnaire. Rice mill, sawmill, furniture factory, non-exposed. **Woodworking processes performed (e.g. sawing, drilling):** Unknown **Types of wood:** Unknown **Types of tool used:** Unknown **Types of medical examination:** Post-shift spirometry **Information on incidence:** Not applicable **Type of statistical analyses:**	See stratified results below. **Results of a survival analysis:** Not performed. **Stratified results (dose–response relationship):** Exposed: FVC predicted: r = -0.282, p = 0.013 FEV1 predicted: r = -0.241, p = 0.035 FEV1/FVC predicted: r = 0.879, p = 0.018 Non-exposed: FVC predicted: r = -0.514, p = 0.02 FEV1/FVC measured: r = 0.394, p = 0.021 FEV1/FVC predicted: r = 0.545, p = 0.001 No other significant association was reported. **Did authors find a dose–response relationship (yes/no)?:** Yes.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		The correlation of exposure with total inhalable dust and lung function was determined using a Spearman's rank correlation test. **Whether authors took into account for other risk factors, such as smoking and mixed exposure to other compounds:** Yes: smoking habit, date of birth, height, weight, gender and race were keyed into the spirometer system.		

Table 29 Long-term or both short- and long-term occupational wood dust exposure and irritation and sensitisation effects

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
Whitehead et al. (1981) (277)	**Type of study:** Cross-sectional **Country (region):** America (New England) **Type of industry:** Woodworking **Follow-up period:** Same day **Study period:** June–November 1978 **Number of participants:** 1157 **Number and origin of controls:** Workers in non-dusty environments in the same plants **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	**Exposure measurement data:** Average dust levels obtained ranged from 0.2 mg/m^3 for plywood shipping/receiving areas to 4.5 mg/m^3 for hardwood sanding areas. Number of hours measured is unknown. **Personal or stationary sampling of exposure levels:** Stationary total dust levels. The majority of samples were taken in stationary positions on machines as close as possible to worker breathing zones. Some samples were collected in the centre of a workroom. The average dust exposure measured for the department type in which the employee worked is multiplied by each employee's years in the company to obtain a personal relative dust exposure index. Three categories were examined: Low (0–<2 mg-years/m^3) Medium (2–<10 mg-years/m^3) High (10+ mg-years/m^3) **Number of measurements:** 100 total suspended dust samples **Methods of exposure measurement and analysis:** Unknown	**Types of health effect observed:** Lung function (FVC, FEV_1, $FEF_{25–75\%}$) (low vs normal) **Single, short-term exposure or long-term exposure:** Long-term (cumulative) **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** See stratified results below. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Analyses were adjusted for smoking. Association between hardwood dust exposure and FEV_1/FVC (OR (p-value)): Medium (ref: low): 2.61 (0.003)	Information on hours of exposure assessed is lacking. Only total dust levels are examined. Only stationary measures are examined. Results are potentially affected by healthy-worker effect.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Job and exposure history (JEM, questionnaires, census data, etc): Questionnaire **Job classification:** Questionnaire **Woodworking processes performed (e.g. sawing, drilling):** Sanding, machining and assembly in rough lumber mills and finish mills, and non-dusty departments including shipping/receiving and administration. Excluded were those exposed to finishes, green (undried) wood dust (as in sawmill and bark-stripping operations), engaged in plywood glue-up and press operations, in metal machine shop situations and using dusty materials other than the major wood used (for example, masonite, plastic laminates or upholstery materials). **Types of wood:** Softwood (pine) and hardwood (rock maple, ash and oak) **Types of tool used:** Unknown **Types of medical examination:** Spirometric pulmonary function status and questionnaire to determine pulmonary symptoms. Pulmonary impairment was examined by comparing results of the	High (ref: low): 3.12 (0.001) High (ref: medium): 1.24 (0.52) Med+high (ref: low): 2.80 (<0.001) Association between hardwood dust exposure and FEF25–75% (OR (p-value)): Medium (ref: low): 2.17 (0.01) High (ref: low): 2.14 (0.02) High (ref: med): 1.03 (0.93) Med+high (ref: low): 2.12 (.005) No association between hardwood dust exposure and FVC and FEV_1. Association between pinewood dust exposure and FEV_1/FVC (OR (p-value)): Medium (ref: low): 2.40 (0.08) High (ref: low): 4.03 (0.001)	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		spirometric tests with external reference values. **Information on incidence:** e.g. hardwood workers never-smokers **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Logistic regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: smoking was examined.	High (ref: medium): 2.06 (0.17) Med+high (ref: low): 2.63 (0.01) Association between pinewood dust exposure and FEF25–75% (OR (p-value)): Medium (ref: low): 1.77 (0.14) High (ref: low): 2.45 (0.02) High (ref: med): 1.59 (0.30) Med+high (ref: low): 2.00 (0.03) No association between pinewood dust exposure and FVC and FEV_1. **Did authors find a dose–response relationship (yes/no)?:** Yes.	
Bohadana et al. (2000) (278)	**Type of study:** Cross-sectional **Country (region):** France	**Exposure measurement data:** Modelled using algorithm; 8-hour TWA and cumulative exposure. GM ranging from 2.4 to 7.9 mg/m^3 depending on sanding tasks (higher) and local exhaust ventilation (lower).	**Types of health effect observed:** Respiratory symptoms, lung function and non-specific bronchial responsiveness (non-	Exposure levels were estimated on the basis of a large database, but a very simplified

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Type of industry: Wooden furniture factories processing mainly beech and oak **Follow-up period:** Not applicable **Study period:** 1995–1996 **Number of participants:** 127 (men only) **Number and origin of controls:** 13 unexposed woodworkers plus 200 workers from other industries (all men) **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	**Personal or stationary sampling of exposure levels:** Presumably personal, but not clearly stated **Number of measurements:** 443 in 28 similar wooden furniture factories (3 in this study) **Methods of exposure measurement and analysis:** Millipore filter holders with 37-mm glass microfibre filters, flow 1 L/min. Inhalable dust, gravimetrical analysis **Job and exposure history (JEM, questionnaires, census data, etc):** Working duration and job history (questionnaire) **Job classification:** Based on sanding task, with(out) LEV; thus 4 categories; work history (years) in this industry. **Woodworking processes performed (e.g. sawing, drilling):** sanding and other Includes handling various types of saws, planing machines, mortising, tenoning, drilling machines, spindle moulding, slotting and milling machines. **Types of wood:** Hardwood (beech and oak) (most likely including wood that was treated) **Types of tool used:**	carcinogenic effects lower respiratory tract) **Single, short-term exposure or long-term exposure:** Long-term average exposure over working life **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Crude numbers [n(%) for symptoms and BHR; mean (SD) lung function parameters and BHR slope and p-values from adjusted models are reported in the article. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Cumulative exposure [year-mg/m^3] in categories (0, ≤70, 70–	algorithm (deterministic model) was used to assign an exposure concentration to each individual. Smoking was not well controlled for in BHR analyses (but maybe it was checked).

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Mixed **Types of medical examination:** Questionnaire (symptoms), lung function, non-specific bronchial [hyper]responsiveness (methacholine challenge testing) **Information on incidence:** Overall, the prevalence of chronic respiratory symptoms tended to be low (e.g. 5 individuals had dyspnoea in the group with 110–160 mg/m^3-years). The prevalence of irritant symptoms tended to be somewhat higher than that of chronic symptoms, with maximal values of 32% for red (burning) eyes in the group with 70–110 mg/m^3-years. However, only the prevalence of sore throat increased significantly with increasing exposure. The pulmonary function variables of historical controls and woodworkers according to the various classes of cumulative exposure are included in the report. Overall, the measured values tended to exceed the predicted ones for most variables. Two unexposed individuals had one of the chronic respiratory symptoms. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:**	110, 110–160 and >160 years.mg/m^3). Dose–response trend for bronchial responsiveness: 1/13 (8%); 4/28 (14%); 6/28 (21%); 6/25 (21%); 9/33 (27%), p-value 0.002 adjusted for baseline FEV_1 and age **Did authors find a dose–response relationship (yes/no)?:** Yes, for bronchial responsiveness and prevalence of specific symptom sore throat.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Multiple linear regression, including mixed models **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Smoking considered but not (always) taken into account. Other exposures not included.		
Borm et al. (2002) (279)	**Type of study:** Cross-sectional **Country (region):** Indonesia **Type of industry:** Plywood and woodworking plant **Follow-up period:** Not applicable **Study period:** 1996–1997 **Number of participants:** 982 **Number and origin of controls:** Administrative workers (N=57) from the same plant included in low exposure category	**Exposure measurement data:** 6-hour TWA **Personal or stationary sampling of exposure levels:** Personal; 52% GM (GM) <2 mg/m^3; 30% GM 2–5 mg/m^3; 18% GM >5 mg/m^3. A wide range of GMs (6-hour TWA) of inhalable wood dust exposures are given for various job titles. Only the lowest and highest GMs (GSD) are shown here: Category 1 (0–2 mg/m^3): 0.35 (1.98) (administration) - 1.93 (1.61) mg/m^3 (moulding) Category 2 (2–5 mg/m^3): 2.11 (1.35) (grading) - 4.87 (1.37) mg/m^3 (veneer-handcut) Category 3 (>5 mg/m^3): 5.21 (1.64) (finishing) - 12.23 (1.65) mg/m^3 (sanding) Mean (SEM) cumulative exposure (mg/m^3 x years) in men: Category 1 (0–2 mg/m^3): 15.65 (0.53)	**Types of health effect observed:** Lung function, respiratory symptoms and nasal inflammation **Single, short-term exposure or long-term exposure:** Long-term (cumulative exposure) **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Beta estimates with p-values **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):**	Response is unknown and possible healthy-worker/smoker effect. This study was restricted to meranti wood. The aim was to investigate the inflammatory response in the nose in workers exposed to softwood dust. Cumulative exposure was determined by using current wood dust

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Name of study/ cohort: Unknown **Earlier publications on the same cohort:** None known	Category 2 (2–5 mg/m^3): 35.45 (1.43) Category 3 (>5 mg/m^3): 35.57 (3.22) Total: 26.09 (0.89) Mean (SEM) cumulative exposure (mg/m^3 x years) in women: Category 1 (0–2 mg/m^3): 11.48 (0.45) Category 2 (2–5 mg/m^3): 25.45 (1.66) Category 3 (>5 mg/m^3): 34.87 (4.78) Total: 15.47 (0.63) **Number of measurements:** 243 **Methods of exposure measurement and analysis:** Inhalable dust; active sampling using PAS-6 heads with glass fibre filters at a flow rate of 2 L/min **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire. An internal JEM was used to assign an exposure level (not measured) to study participants on the basis of their (sub)function. **Job classification:** 37 categories according to unit and (sub)function; subdivision into 3 larger exposure groups (GM <2; 2-5; >5 mg/m^3). **Woodworking processes performed (e.g. sawing, drilling):**	Association between 3 categories of current wood dust exposure (<2.0 mg/m^3; 2–5 mg/m^3 and >5 mg/m^3) and lung function (* p<0.05 ** p<0.01), adjusted for smoking, glue and age: FEV_1 (l): Men (M) (β): 0.079; 0.177**; 0.177* Women (W) (β): NS; NS; NS FVC (l): M (β): 0.05; 0.134; 0.127 W (β): 0.153; 0.132; 0.076 FEV_1/FVC (%): M (β): 0.07; 0.138; 0.119 W (β): -0.09; -0.01; -0.03 MEF (l/s): M (β): 0.028; 0.095; 0.050 W (β): 0.167; 0.195; 0.170	exposure. Results on cumulative exposure were not reported.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Wide range of processes, e.g. veneering preparation, moulding, assembly, boring, cross-cutting **Types of wood:** Meranti (hardwood) (including treated wood; adjustments for formaldehyde were made, see below) **Types of tool used:** Unclear **Types of medical examination:** Symptom questionnaire, lung function (both impedance and spirometry), and cellularity in nasal lavage fluid **Information on incidence:** Respiratory complaints were in general most prevalent in the highest exposure category (>5mg/m^3). No difference was found in recovery of nasal lavage between men and women or exposure categories. However, a significantly lower cell count was detected in women in the highest exposure category ($p<0.05$, ANOVA, least SD test). FEV_1 in men (% of predicted on the basis of age and height) was 102%, 106% and 107% for exposure categories of <2, 2–5 and >5 mg/m^3, respectively. Approximately 50% of both male and female workers were exposed to glue.	PEF (l/s): M (β): 0.219**; 0.191; 0.143 W (β): 0.290**; 0.256**; 0.234* Resonance frequency: M (β): -0.505*; -0.538*; -0.416* W (β): -0.300**; -0.294**; -0.147 Average resistance (R): M (β): -0.220; -0.250; 0.220** W (β): 0.202; 0.176; 0.030 Reactance (X): M (β): 0.386*; 0.453*; 0.295* W (β): 0.308**; 0.256; 0.177** Frequency dependence: M (β): 0.340**; 0.330**; 0.250** W (β): 0.020; 0.030; 0.040 No association was found between: - cumulative wood dust exposure and lung	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Mortality prevalence/percentage of the control group or general population (background data): Not reported **Type of statistical analyses:** Multiple linear regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: sex, age and smoking. Glue (possible exposure to formaldehyde) qualitatively considered but not quantified.	function (data not reported); - current or cumulative wood dust exposure and respiratory symptoms; - current wood dust exposure and nasal cellularity (adjusted for age, weight, duration of employment, and recovered volume). Within workers in exposure category I (<2mg/m^3): those with coexposure to formaldehyde (N=286) versus those only exposed to wood dust (N=280) showed no increased symptoms or decreased lung function (data not shown). **Did authors find a dose–response relationship (yes/no)?:** No.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
Rongo et al. (2002) (280)	**Type of study:** Cross-sectional **Country (region):** Tanzania **Type of industry:** Small-scale wood industries (SSWI) **Follow-up period:** Not applicable **Study period:** Unknown **Number of participants:** 546 men **Number and origin of controls:** 565 male office workers **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	**Exposure measurement data:** 8-hour TWA (mg/m^3) **Personal or stationary sampling of exposure levels:** Personal, overall GM 3.9 mg/m^3; lowest GM (GSD) in individuals working in manual carpentry: 2.9 (1.8) mg/m^3; highest GM (GSD) observed in individuals working with carving machine: 22.8 (2.8) mg/m^3 **Number of measurements:** 106 **Methods of exposure measurement and analysis:** Inhalable dust; active sampling using IOM sampling heads with glass fibre filters, flow rate 2 L/min; gravimetric analysis **Job and exposure history:** Questionnaire on work history and characteristics **Job classification:** On the basis of work tasks and dust measurement results: 1) non-exposed; 2) planing and sawing manually; 3) machine drilling, planing and sawing; 4) carving using wood machines, cleaning the workshops and repairing the wood machines; 5) others **Woodworking processes performed (e.g. sawing, drilling):** Planing, sawing, carving, drilling	**Types of health effect observed:** Respiratory symptoms **Single, short-term exposure or long-term exposure:** Short- and long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** % of prevalence, OR with 95% confidence interval **Results of a survival analysis:** Not performed. **Stratified results (dose–response relationship):** Yes, low exposure group and high (combined medium-high exposure groups). An increased prevalence of respiratory symptoms in the exposed small-scale wood industry workers compared with non-exposed office workers. A	Symptoms analysed one by one without a priori grouping. No clear cut-off point of exposure level between low and medium/high exposure groups. Groups were based on job title, not wood dust exposure.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Types of wood: Both softwood and hardwood (including treated wood: 'Chemicals used in the SSWI were lubricant oils for wood machines, wood glues with thinners, polish and varnish.') **Types of tool used:** Manual tools; machines for drilling, planing, sawing and carving **Types of medical examination:** Symptom questionnaire (both chronic and work-related/temporal symptoms) **Information on incidence:** Asthma symptoms during the last year were significantly more prevalent in the exposed compared with non-exposed workers. Symptoms of cough (last year) were more common in the low- and high-exposure workers than in the non-exposed group. Nose, eye, and skin symptoms were more prevalent in the high exposure group. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Multivariable logistic regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:**	significant increase in symptoms such as cough; coughing up phlegm; awakened regularly because of cough; shortness of breath with wheezing; awakened by shortness of breath, runny nose, and sneezing more than once a week; itching and watering eyes; and allergy/sensitivity to house dust, food, animals, or grasses/plants was demonstrated. High-exposure workers seemed to have a slightly higher prevalence of symptoms compared with the low(er)-exposure workers, although this difference was not statistically significant – for instance, cough OR (95% CI) for low and high exposure 1.6 (1.2–2.0) and 1.6 (1.1–2.4),	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Yes: age and smoking (no concurrent exposures in the workplace).	respectively. Adjusted for age, height, weight, duration of employment in the current job, hours per week of work, daily use of protective equipment and smoking status. **Did authors find a dose–response relationship (yes/no)?:** Not clearly; only for selected symptoms and not significant.	
Douwes et al. (2006) (281)	**Type of study:** Cross-sectional/ case–control study including cross-shift changes **Country (region):** New Zealand **Type of industry:** Sawmills **Follow-up period:** 1 year **Study period:** Unclear	**Exposure measurement data:** TWA (mg/m^3) **Personal or stationary sampling of exposure levels:** Personal, GM per category 0.4–0.8 mg/m^3. Total GM (GSD): 0.52 (2.66) mg/m^3 (N=183) **Number of measurements:** 183 **Methods of exposure measurement and analysis:** Inhalable dust, IOM sampling heads with glass fibre filters. Gravimetric analysis. **Job and exposure history:** Only current job considered	**Types of health effect observed:** Lung function and atopy **Single, short-term exposure or long-term exposure:** Short- and long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Beta estimates with 95% CI for differences in lung	The design is a case–control study, but cases and controls were pooled to study associations between dust exposure, lung function and atopy. Rather nebulous, but case/control status was taken into account in

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Number of participants: 59 asthmatics and 167 non-symptomatics **Number and origin of controls:** No controls **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** Douwes et al. (2001) (282)	**Job classification:** Job titles classified into 4 categories: non-exposed; low or intermittent exposure; high exposure to green dust; high exposure to dry dust **Woodworking processes performed (e.g. sawing, drilling):** Sawing logs to produce green timber and subsequently sorting, grading, trimming and stacking; kiln-drying the green timber; processing kiln-dried timber in the planer mill, remanufacturing and moulding **Types of wood:** Pine (softwood) (treated wood: including chemicals such as resin acids and monoterpenes). **Types of tool used:** Not specified **Types of medical examination:** Skin prick testing (8 allergens); lung function spirometry **Information on incidence:** N=59 had asthma symptoms. Subjects with asthma symptoms exhibited a significantly lower FEV_1 and PEF than those without asthma symptoms, whereas their FVC was very similar.	function, stratified by symptom status: For high dry dust exposure FEV_1 -0.19 L (CI -0.37–0.00); for high green dust exposure FEV_1 -0.26 L (CI -0.46–-0.06) vs no/low/intermittent exposure, adjusted for symptom status, sex, age, ethnicity, smoking and height **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** No, but results were stratified according to high green dust and high dry dust exposure. **Did authors find a dose–response relationship (yes/no)?:** No.	those analyses (by adjustment or stratification). Adjusted for current and previous smoking, but unclear whether models were adjusted for pack-years.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Mortality prevalence/percentage of the control group or general population (background data): Not reported **Type of statistical analyses:** Multivariable logistic (atopy) and linear (lung function) regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: symptom status, sex, age, height, ethnicity and smoking (no other occupational exposures than wood dust).		
Rusca et al. (2008) (283)	**Type of study:** Cross-sectional **Country (region):** Switzerland **Type of industry:** 12 sawmills **Follow-up period:** Not applicable **Study period:** 2002 **Number of participants:** 111 male woodworkers **Number and origin of controls:**	**Exposure measurement data:** TWA (mg/m^3) **Personal or stationary sampling of exposure levels:** Personal (presumably); mean 1.7 mg/m^3 and range 0.2–8.5 mg/m^3 (more details on the applied methods can be found in Oppliger et al. (2005) (284)) **Number of measurements:** 50 **Methods of exposure measurement and analysis:** Inhalable dust; active sampling using IOM heads with glass fibre filters at a flow rate of 2 L/min	**Types of health effect observed:** Medical respiratory and general symptoms **Single, short-term exposure or long-term exposure:** Long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Partial regression coefficients from generalised linear models	Exposure–response analysis with dust exposure levels not performed. Generalised linear models using dust concentration not sufficiently elaborated.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	None **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	**Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire on occupational history and seniority (duration of occupational exposure to wood dust) **Job classification:** On the basis of measurements performed in the 12 sawmills; unclear whether the mean exposure level of a sawmill was assigned to all (non-sampled) workers from the respective mill **Woodworking processes performed (e.g. sawing, drilling):** Sawing and planing **Types of wood:** Predominantly softwood (spruce and fir) (untreated: 'The study was designed for workers exposed to the same species of fresh wood before any use of biocide or chemical agents.') **Types of tool used:** Not detailed **Types of medical examination:** Symptom questionnaire, lung function **Information on incidence:** Group 1 workers (<5 years) complained twice as much of ocular, nasal and upper airways tract irritations when exposed to wood dust. Junior workers showed significantly more	**Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Only according to seniority (years of occupational wood dust exposure). Results suggest that occupational exposure to wood dust in a Swiss sawmill does not promote a clinically relevant decline in lung function. **Did authors find a dose–response relationship (yes/no)?:** No.	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		irritation syndrome (defined by itching/running nose, snoring and itching/red eyes) than senior workers. Lung function tests were not influenced by bioaerosol levels nor dust exposure levels. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Generalised linear models **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: seniority, smoking, atopy. Other quantified exposures: viable fungi and bacteria, endotoxin.		
Osman and Pala (2009) (285)	**Type of study:** Cross-sectional **Country (region):** Turkey **Type of industry:** Furniture manufacturing **Follow-up period:** Not applicable **Study period:** 2006–2007	**Exposure measurement data:** 8-hour TWA (mg/m^3) **Personal or stationary sampling of exposure levels:** Personal, mean level 2 mg/m^3 (10% had ≥5 mg/m^3) **Number of measurements:** Unclear (non-measured workers were assigned the measured exposure level of others in the same workplace) **Methods of exposure measurement and analysis:**	**Types of health effect observed:** Lung function and respiratory symptoms **Single, short-term exposure or long-term exposure:** Short- and long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:**	Smoking not taken into account in analyses. No association presented between exposure level and symptoms. Assignment of exposure level to

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Number of participants: 328 **Number and origin of controls:** 328 employed in stores **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	Active sampling, flow rate 2 L/min, unknown sampling head with 37-mm PVC filter, gravimetric analysis. Unclear whether inhalable dust is measured through this method. **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire for occupational history **Job classification:** Not done (analyses based on exposure level and worked years) **Woodworking processes performed (e.g. sawing, drilling):** Unknown **Types of wood:** Mixed softwood and hardwood (including treated wood): MDF, beech, pine and fibreboard **Types of tool used:** Not specified **Types of medical examination:** Questionnaires and lung function testing **Information on incidence:** Mean values of the$FEV_1$and FVC in the furniture workers were significantly lower than those in the control group; however, FEV1/FVC values were higher.	n(%) for symptoms; mean and 95% CI of %predicted lung function value; p-values. For instance, for those with working time <10 years, FEV1 (%) was 87.0 and 96.7 for <4 and ≥4 mg/m^3, respectively. For those with working time ≥10 years, FEV_1 (%) was 89.9 and 92.4 for <4 and ≥4 mg/m^3, respectively. Not further adjusted. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Only for lung function: ≥4 vs <4 mg/m^3; ≥5 vs <5 mg/m^3, further stratified by working time (≥10 vs <10 years) **Did authors find a dose–response**	non-measured individuals unclear.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Mortality prevalence/percentage of the control group or general population (background data): Not reported **Type of statistical analyses:** Unadjusted comparisons using χ^2 and Student's t-tests **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** No.	**relationship (yes/no)?:** No (indications for reverse association).	
Zaman (2017a) (286)	**Type of study:** Retrospective longitudinal study **Country (region):** Indonesia (Ogan Ilir District, South Sumatra Province) **Type of industry:** Plywood **Follow-up period:** Same day **Study period:** Unknown **Number of participants:** 87 workers (of total 381 employed at factory)	**Exposure measurement data:** Personal dust (unknown number of hours): mean (SD): 0.3 (0.4) mg/m^3 min–max: 0.06–0.85 mg/m^3 Area dust (unknown number of hours): mean (SD): 0.7 (0.4) mg/m^3 min–max: 0.30–1.25 mg/m^3 **Personal or stationary sampling of exposure levels:** Inhalable dust (size of particles not reported) measured by personal and stationary sampling **Number of measurements:** Unknown **Method of exposure measurement:** Unknown **Method of analyses:**	**Types of health effect observed:** Lung function **Single, short-term exposure or long-term exposure:** Short- and long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Work duration x personal dust (years/mg/m^3; Pearson correlation (p-value)): FVC: -.098 (0.367) Work duration x area dust (years/mg/m^3;	The article is poorly written, which makes it difficult to extract information or determine its quality. Information seems to be lacking – e.g. it is unclear how participants were selected and the number of hours of exposure measured is unknown.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Number and origin of controls: No controls **Name of study/ cohort:** Not known **Earlier publications on the same cohort:** The authors do not refer to it, but based on the location of factories, it is possible that the same cohort has been examined in Zaman et al. (2015) (287) (no association with health was examined in that study). In addition, very similar methods were used in Zaman (2017b) (288)	Gravimetric **Job and exposure history (JEM, questionnaires, census data, etc):** Unknown **Job classification:** Unknown **Woodworking processes performed (e.g. sawing, drilling):** Cutting and sanding **Types of wood:** rubber wood (hardwood) (unknown whether treated or not) **Types of tool used:** Unknown **Types of medical examination:** Spirometry **Information on incidence:** Prevalence: the spirometry values of FVC, FEV_1, and FEV_1/FVC were 3.67(0.92) litres, 3.20(0.86) litres, and 87.40(11.57)%, respectively. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported. **Type of statistical analyses:** Correlation and multiple linear regression	Pearson correlation (p-value)): FVC: 0.051 (0.636) Correlations between cumulative exposure and the outcomes $FEV_1$and FEV_1/FVC were not significant. The results of the multiple linear regression analyses were not shown in the publication. The authors report that the results showed non-significant associations (and were adjusted for age, gender, smoking and height and both exposure measurements, i.e. cumulative area and personal measurements, in the model). **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Not available	The exposure measurement method is not described. Multiple linear regression analyses are not shown and it seems that both cumulative area and personal measurements are included in the same model, which is not desirable.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds: Yes: age, gender, smoking and height	**Did authors find a dose–response relationship (yes/no)?:** No.	
Zaman (2017b) (288)	**Type of study:** Cross-sectional **Country (region):** Indonesia (South Sumatra Province) **Type of industry:** Pencil slat **Follow-up period:** Same day **Study period:** Unknown **Number of participants:** 90 workers (of total 271 employed at factory) **Number and origin of controls:** No controls **Name of study/ cohort:** Unknown	**Exposure measurement data:** Personal dust (unknown number of hours): mean (SD): 0.1 (0.3) mg/m^3 min–max: 0.002–2.490 mg/m^3 Area dust (unknown number of hours): mean (SD): 0.1 (0.2) mg/m^3. min–max:0.060–0.845 mg/m^3 **Personal or stationary sampling of exposure levels:** Both **Number of measurements:** 90 **Method of exposure measurement:** Unknown **Method of analyses:** Gravimetric **Job and exposure history (JEM, questionnaires, census data, etc):** Unknown **Job classification:** Unknown **Woodworking processes performed (e.g. sawing, drilling):** Sanding and cutting	**Types of health effect observed:** Lung function **Single, short-term exposure or long-term exposure:** Short- and long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Work duration x personal dust (years/mg/m^3; Pearson correlation (p-value)): FVC: -0.181 (0.016) FEV1: -0.182 (0.086) FEV1/FVC: 0.011 (0.917) Work duration x area dust (years/mg/m^3; Pearson correlation (p-value)): FVC: -0.199 (0.060) FEV_1: -0.233 (0.035)	Identical methods were used in Zaman et al. (2017) (rubber) and identical limitations were applicable.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Earlier publications on the same cohort: The authors do not refer to it, but based on the location of factories, it is possible that the same cohort has been examined in Zaman et al. (2015) (287) (no association with health was examined in that study). In addition, very similar methods were used in Zaman (2017a) (286).	**Types of wood:** Pulai wood (hardwood) (coloured, dried, sanded and cut) **Types of tool used:** Unknown **Types of medical examination:** Spirometry **Information on incidence:** Prevalence: the spirometry values of FVC, FEV_1, and FEV_1/FVC were 3.5(1.0) L, 3.0(0.9) L, and 85.0(11.9)%, respectively, for 90 subjects. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Correlation and multiple linear regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: age, gender, smoking and height	FEV_1/FVC: -0.157 (0.140) All multiple linear regression analyses showed to be non-significant (results not reported in the article) **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Not performed **Did authors find a dose–response relationship (yes/no)?:** No.	
Badirdast et al. (2017) (289)	**Type of study:** Cross-sectional **Country (region):** Iran **Type of industry:** Chipboard	**Exposure measurement data:** TWA (mg/m^3) and cumulative exposure **Personal or stationary sampling of exposure levels:** Personal. Overall GM = 19 mg/m^3; across tasks ranging from 10 to 70 mg/m^3. Cumulative ranging up to 1700mg/m^3 x years.	**Types of health effect observed:** Alteration of the respiratory function parameters (non-carcinogenic effects lower respiratory tract)	Chipboard is treated wood. Exposure levels are very high, in particular in the 'shredding' group.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Follow-up period: Not applicable **Study period:** Unknown **Number of participants:** 100 woodworkers (chipboard) **Number and origin of controls:** 50 guards **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	**Number of measurements:** Done for each worker, thus at least 100 (and 50 in controls). The number of measurements per worker is not specified, but is likely to be 1. **Methods of exposure measurement and analysis:** Active sampling using SKC personal samplers with 25 mm ester cellulose mixed filter with a pore size of 0.8 µm; flow and sampling time not stated. Inhalable dust, gravimetrical analysis **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire on work history provided information on the number of years with exposure to wood dust **Job classification:** 11 occupational groups: bark-stripping, shredding, fine shredding, milling, drying, adhesive mixing, pressing, sawing, sanding, transportation, sanitation **Woodworking processes performed (e.g. sawing, drilling):** Different chipboard manufacturing task groups, such as disembarkation, shredding, fine shredding, milling, drying, adhesive mixing, pressing, sawing, sanding, sanitation, and transportation.	**Single, short-term exposure or long-term exposure:** Long-term average exposure over working life **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** None reported **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Associations between lung function parameters (standardised for age, height, and weight) and cumulative exposure to wood dust are presented as scatterplots. Regression equations are provided – for instance: FEV_1=3.55–0.0000929×cumulative exposure.	Cumulative exposures of more than 1500 mg/m^3 x years are hard to believe. Linear regression models were not adjusted for smoking. Lower lung function at higher levels of cumulative exposure may be explained by the older study participants. They have worked longer in this industry and on average have smoked more. Controls (guards) are included in exposure–

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Types of wood: Chipboard (presumably softwood but not specified by the authors), which is treated wood **Types of tool used:** Not specified, but likely to be various given the variety of tasks **Types of medical examination:** Lung function (Micro lab II spirometer) **Information on incidence:** Not reported **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Linear regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** No: most notably adjustment for smoking not mentioned.	**Did authors find a dose–response relationship (yes/no)?:** Yes: linear model suggests a trend.	response analyses.
Yusof et al. (2019) (290)	**Type of study:** Cross-sectional **Country (region):** Malaysia **Type of industry:** Furniture	**Exposure measurement data:** Continuous measurements for 8 hours **Personal or stationary sampling of exposure levels:** Stationary, inhalable dust. Concentrations between 0.5 and 1.1 mg/m^3 **Number of measurements:**	**Typed of health effect observed:** Respiratory and skin health effects **Single, short-term exposure or long-term exposure:**	Static (area) dust measurements are likely underestimate true worker exposure.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Follow-up period: Not applicable **Study period:** Unknown **Number of participants:** 241 **Number and origin of controls:** No controls **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	Unknown **Methods of exposure measurement and analysis:** Dust Track Aerosol Monitor (Model 8520) **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire on working information and past work history. **Job classification:** According to work area: high exposure in material supply area and machinery area; low exposure in store, spray and sanding areas **Woodworking processes performed (e.g. sawing, drilling):** Sanding, sawing, drilling **Types of wood:** Rubber wood (hardwood). There is a probability of exposure to rubber wood dust alone or in combination with other chemicals, especially in spraying and assembly areas (not adjusted for in analyses). **Types of tool used:** Unknown **Types of medical examination:** Symptom questionnaire and lung function testing **Information on incidence:** High exposure (N=59): cough 14 (23.7%), chest tightness 8 (13.6%), phlegm 6	Long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Symptoms: n(%), OR with 95% CI; lung function parameters: mean and SD, beta estimates (adjusted differences) with 95% CI. For instance: FEV_1 high vs low exposure -0.51 (-0.69–-0.34) L, adjusted for sex, age, height, education and smoking. Nasal symptoms high vs low exposure OR = 3.9 (CI 2.0–7.5), sex, age, education and smoking. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** No, only high vs low exposure	Only two categories of worker exposure with 1 mg/m^3 as cut-off according to work area. Smoking amount and years not taken into account.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		(10.2%), breathlessness 4 (6.8%), wheezing 3 (5.1%), nasal symptoms 43 (72.9%), eye symptoms 39 (66.1%), skin symptoms 17 (28.8%). Low exposure (N=182): cough 33 (18.1%), chest tightness 27 (14.8%), phlegm 15 (8.2%), breathlessness 16 (8.8%), wheezing 6 (3.3%), nasal symptoms 79 (43.4%), eye symptoms 86 (47.3%), skin symptoms 39 (21.4%) **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Multivariable logistic (for symptoms) and linear (lung function parameters) regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: sex, age, height, education and smoking (only yes/no; not ex-smoking and pack-years). No concurrent exposures.	**Did authors find a dose–response relationship (yes/no)?:** Not applicable	

Table 30 Summary of epidemiological studies using the same cohort in a Danish furniture industry: short-term occupational wood dust exposure and irritation and sensitisation effects

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
Schlünssen et al. (2002) (291)	**Type of study:** Cross-sectional **Country (region):** Denmark **Type of industry:** Furniture **Follow-up period:** Not applicable **Study period:** 1997–1998 **Number of participants:** 161 **Number and origin of controls:** 19 workers from factories producing refrigerators or hearing aids **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** (15, 75)	**Exposure measurement data:** 8-hour TWA **Personal or stationary sampling of exposure levels:** Personal; AM 1.17 mg/m^3; range 0.17–3.44 mg/m^3 **Number of measurements:** 140 **Methods of exposure measurement and analysis:** Passive dust monitors, previously calibrated against active sampling for inhalable dust with filters and gravimetric analysis **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire on work history; internal JEM created to assign dust exposure levels to 5 workers without measurements **Job classification:** Into three groups on the basis of IQR of inhalable dust exposure (<0.74; 0.74–1.42; >1.42 mg/m^3) **Woodworking processes performed (e.g. sawing, drilling):** Not specified here; see other publication of this cohort. **Types of wood:**	**Types of health effect observed:** Subjective and objective nasal obstruction **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Coefficients for dust exposure category from regression on change in nasal congestion. For example, beta for $DA_{4.1}$ change was 0.215 (p=0.001) per unit of exposure. Significant unadjusted increase in nasal obstruction over the work day found in medium (0.74-1.42 mg/m^3) and high (>1.42 mg/m^3) exposure groups, but not in	Passive personal dust monitors were used. Measurement results were converted into equivalent inhalable dust concentrations. Unclear how dust exposure category was applied in multiple linear regression models; looks like 0/1/2/3 for controls and <0.74; 0.74–1.42 and >1.42 mg/m^3, respectively.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Pine (softwood), composite wood (particle board, veneer, MDF), hard wood, mixed types. In part treated wood. Not adjusted for in analyses **Types of tool used:** Not specified here; see other publication of this cohort **Types of medical examination:** Acoustic rhinometry and nasal symptoms questionnaire **Information on incidence:** For the two highest exposure concentrations, a significant increase in congestion – decreased nasal cavity volume and cross sectional areas – was found after 4 and 7 hours of work, compared with before work. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Multiple linear regression models **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: age, sex, height, weight, allergy, hay fever, temperature, use of compressed air,	the low exposure group or the controls. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Temporal patterns of unadjusted mucosal swelling variables are presented for controls and inhalable dust exposure (<0.74; 0.74–1.42; >1.42 mg/m^3). In addition, scatterplot of one of the crude mucosal swelling variables vs wood dust exposure is presented. A positive correlation between concentration of dust and change in mucosal swelling was observed. A significant increase in self-rated nasal obstruction was found after work compared with before work for the two highest exposure groups. No correlation between objective nasal variables	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		use of pine wood, and heart rate before measurement by acoustic rhinometry.	and self-rated nasal obstruction was found. **Did authors find a dose–response relationship (yes/no)?:** Yes: for some of the crude mucosal swelling variables. Exposure to wood dust was related by dose to acute nasal obstruction measured by acoustic rhinometry and by self-reported obstruction.	
Schlünssen et al. (2002) (75)	**Type of study:** Cross-sectional **Country (region):** Denmark **Type of industry:** Furniture **Follow-up period:** Not applicable **Study period:** 1997–1998 **Number of participants:** 2033 **Number and origin of controls:** 474 mechanical assembly workers	**Exposure measurement data:** 8-hour TWA **Personal or stationary sampling of exposure levels:** Personal, GM 0.93 mg/m^3; range 0.17–9.78 mg/m^3. **Number of measurements:** 1579 **Methods of exposure measurement and analysis:** Passive dust monitors, previously calibrated against active sampling for inhalable dust with filters and gravimetric analysis **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire on work history; internal JEM	**Types of health effect observed:** Respiratory symptoms and lung function **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Symptoms: For instance, OR for asthma 0.99 (CI 0.67–1.46) and 1.32 (0.92–1.93) for wood dust exposure of <1 and >1 mg/m^3,	Passive personal dust monitors were used. Measurement results were converted into equivalent inhalable dust concentrations. Large number of analyses are performed, including analyses of a variety of

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	from factories producing refrigerators or hearing aids **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** (15, 291)	created to assign dust exposure levels to workers without measurements. **Job classification:** Into three groups on the basis of IQR of inhalable dust exposure (<0.74; 0.74–1.42; >1.42 mg/m^3), and into two groups in separate analyses (<1; >1 mg/m^3) **Woodworking processes performed (e.g. sawing, drilling):** Cutting, sanding, assembly **Types of wood:** Pine (softwood), particleboard, hardwood, beech, MDF, mixed. In part treated wood, which was not adjusted for in analyses **Types of tool used:** Not specified, but likely to be various given the variety of tasks **Types of medical examination:** Symptom questionnaire, lung function **Information on incidence:** Frequencies resporiatory symptoms are given for woodworkers, controls with previous wood dust exposure and for controls, stratified by smoking status. Significant differences: compared with non-smoking male controls (15%), a higher percentage of non-smoking woodworkers experienced daily coughing (25.7%). In	respectively, vs controls adjusted for sex, smoking status, atopy and work skills. Within woodworkers, e.g. OR (CI) for chronic bronchitis 1.21 (0.78–1.90) and 1.35 (0.81–2.25) for wood dust exposure of 0.74–1.42 and >1.42 mg/m^3, respectively, vs <0.74 mg/m^3, adjusted for seniority, sex, smoking status and atopy. Lung function: mean % predicted given for exposure groups (<0.74; 0.74–1.42; >1.42 mg/m^3), stratified for sex and smoking status (never, ex, current). Results adjusted for pack-years not presented **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** See above.	symptoms, exposure categories, stratified by sex and smoking status.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		addition, 8.9% of non-smoking woodworkers had physician-diagnosed hay fever and 11.9% had at least one eye symptom, which was significantly higher compared to non-smoking male controls (17.4% and 23.5%, respectively). **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** ANOVA. Stratification by sex and smoking status **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: sex, age, smoking status, previous exposure to dust (excluding wood dust), sideline occupation, education, self-reported job satisfaction. For lung function analyses also pack-years smoked, body mass index and height.	**Did authors find a dose–response relationship (yes/no)?:** Yes, in some (potentially spurious) results. Tendency for inverse trend for lung function parameters	
Schlünssen et al. (2004) (74)	**Type of study:** Cross-sectional (nested case-referent design) **Country (region):** Denmark	**Exposure measurement data:** 8-hour TWA **Personal or stationary sampling of exposure levels:** Personal; GM 0.97 mg/m^3, range 0.17–4.10 mg/m^3 **Number of measurements:**	**Types of health effect observed:** Different indices of asthma **Single, short-term exposure or long-term exposure:** Short-term	Passive personal dust monitors were used. Measurement results were converted into

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Type of industry: Furniture **Follow-up period:** Not applicable **Study period:** Unknown **Number of participants:** 302 **Number and origin of controls:** 71 workers from factories producing refrigerators or hearing aids **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** (15, 75, 76, 291)	347 **Methods of exposure measurement and analysis:** Passive dust monitors, previously calibrated against active sampling for inhalable dust with filters and gravimetric analysis **Job and exposure history (JEM, questionnaires, census data, etc):** Questionnaire. **Job classification:** Questionnaire on work history; internal JEM created to assign dust exposure levels to 61 workers without measurements **Woodworking processes performed (e.g. sawing, drilling):** Sanding, cutting, handling, assembling **Types of wood:** Not specified here; see other publication on this cohort **Types of tool used:** Not specified here; see other publication on this cohort **Types of medical examination:** Symptom questionnaire, lung function, non-specific bronchial responsiveness or bronchodilator induced reversibility testing, repeated peak flow measurements, skin prick testing to common allergens **Information on incidence:**	**Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** Four definitions of asthma; additional analyses stratified by atopy. ORs and 95% CI for wood dust exposure (yes/no) and exposure categories **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Results for four groups of inhalable dust exposure (<0.80; 0.80–0.99; 1.0–1.39; >1.39 mg/m^3), four definitions of asthma and stratification by atopy are included in the article. See most relevant results below. **Did authors find a dose–response relationship (yes/no)?:** Yes: for asthma symptoms with BHR: OR 5.7 / 3.8 / 18.7 for 0.80–0.99, 1.0–	equivalent inhalable dust concentrations.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		244 had asthma symptoms, of which 54 had BHR, 130 had atopy and 78 had increased PEF variation **Mortality prevalence/percentage of the control group or general population (background data):** Not reported. **Type of statistical analyses:** General Additive Models with linear regression and a log link function **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: smoking status, age, previous dusty non-woodworking jobs, sideline occupation, education level, structural lung diseases and FEV_1 (for BHR analyses).	1.39 and >1.39 mg/m^3, respectively, vs <0.80 mg/m^3. Adjustment for sex, age, sideline occupation, use of beech, baseline FEV_1, smoking, atopy	
Schlünssen et al. (2004) (76)	**Type of study:** Short longitudinal study on cross-shift changes **Country (region):** Denmark **Type of industry:** Furniture **Follow-up period:** Not applicable **Study period:** 1997–1998	**Exposure measurement data:** 8-hour TWA **Personal or stationary sampling of exposure levels:** Personal; GM 0.96 mg/m^3 **Number of measurements:** 2217 **Methods of exposure measurement and analysis:** Passive dust monitors, previously calibrated against active sampling for inhalable dust with filters and gravimetric analysis.	**Types of health effect observed:** Cross-shift decline in FEV_1 **Single, short-term exposure or long-term exposure:** Short-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:**	No associations were found among smokers, challenging generalisability. No analysis in exposure categories.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Number of participants: 1560 **Number and origin of controls:** Not applicable **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** (15, 75, 291)	**Job and exposure history (JEM, questionnaires, census data, etc):** **Job classification:** Eight different individual or group-based (statistical) strategies for exposure assessment **Woodworking processes performed (e.g. sawing, drilling):** Not specified here; see other publication on this cohort **Types of wood:** Pine (softwood), particle board or fibre board, hard wood (mainly beech), mixture. In part treated wood. Not adjusted for in analyses **Types of tool used:** Not specified here; see other publication on this cohort **Types of medical examination:** Repeated lung function measurements **Information on incidence:** None reported **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:**	Coefficients, standard errors with p-values for decline in FEV_1 per mg/m^3 dust exposure Only indication for an association in non-smokers (approx. 1 ml decline per mg/m^3), and appeared to be most pronounced among pine workers. Adjusted for age, sex, height, body mass index, use of beech **Results of a survival analysis:** Not performed. **Stratified results (dose–response relationship):** No; all models used the estimated level of wood dust exposure in mg/m^3 continuously, not in categories **Did authors find a dose–response relationship (yes/no)?:** Only in the sense that a higher dust exposure was related to a steeper cross-	Passive personal dust monitors were used. Measurement results were converted into equivalent inhalable dust concentrations.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Least square regression, with modelling the adjusted residuals of cross-shift FEV1 with the wood dust exposure estimate in mg/m^3. **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: age, sex and height; smoking, body mass index, wood species.	shift lung function decline, assuming linearity.	

Table 31 Long-term occupational wood dust exposure and carcinogenicity

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
Kauppinen et al. (1986) (87)	**Type of study:** Retrospective longitudinal nested case–control study **Country (region):** Finland **Type of industry:** 19 particleboard, plywood, sawmill, and formaldehyde glue factories **Follow-up period:** 25 years **Study period:** 1944–1980 **Number of participants:** The numbers of particleboard, plywood, sawmill, and glue production workers in the cohort were 619, 1780, 1394 and 12 (3805), respectively. **Number and origin of controls:**	**Exposure measurement data:** Exposure was estimated using a JEM specifically for wood dust per calendar year. The JEM was based on industrial hygiene measurements, walk-throughs in the plants, interviews of persons with long experience of circumstances of exposure in the plants, and documents provided by the plants. The estimated mean level of exposure to wood dust was about 1.0 mg/m^3 among the exposed cases and about 1.5 mg/m^3 among the exposed controls when calculated according to the work histories in the plants and the respective job exposure matrices. The mean duration of exposure was 9 years for the exposed cases and 11 years for the exposed controls. Individuals were considered to be exposed if they were exposed to wood dust for at least one month. Three levels of exposure to wood dust were classified: low (0.1–1 mg/m^3), moderate (1–5 mg/m^3) and high (>5 mg/m^3). **Personal or stationary sampling of exposure levels:** Although some measurements were performed, very little information is provided regarding measurements (‘In two	**Types of health effect observed:** ‘Respiratory cancers’ were examined (including tongue, pharynx, larynx, epiglottis, lung, trachea cancer). **Single, short-term exposure or long-term exposure:** Long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** See stratified results below. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** Association between wood dust exposure and respiratory cancers, OR (90% CI):	Little information is available on how exposure assessment took place (measurement data are not shared; unknown whether inhalable or respirable exposure data are used). No information on response. Although latency and smoking status were examined, they were not included in the analyses of the relationship between estimated wood dust concentrations and health.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	117 (3 controls per case were selected from within the cohort and matched by birth year). Each control had to be alive and free from respiratory cancer at the date of diagnosis of the corresponding case. **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	plywood plants, one particleboard plant, and one sawmill, measurements were made of some air contaminants for which earlier quantitative data were lacking.'). **Number of measurements:** Not applicable **Methods of exposure measurement and analysis:** Unknown **Job and exposure history (JEM, questionnaires, census data, etc):** JEM to determine exposure. Job history from plant registers and in some cases through interviews **Job classification:** See 'Job and exposure history'. A minimum of one year of employment in at least one of the 19 plants under study was required. **Woodworking processes performed (e.g. sawing, drilling):** Not reported **Types of wood:** Unknown whether untreated or treated wood was examined. Unknown which types of wood were examined in the sawmill and glue industries. **Types of tool used:** Not reported **Types of medical examination:**	0.1 to 1 mg/m^3 (N=26 exposed): 1.18 (0.58–2.39) >1mg/m^3 (N=6 exposed): 0.67 (0.20–2.23) Association between *cumulative* wood dust exposure and respiratory cancers: ≤5mg/m^3-years (N=13): 0.98 (0.37–2.59) >5 mg/m^3-years (N=19): 1.22 (0.57–2.58) Latent periods: excluding exposures during the 10 years immediately preceding the diagnosis of the cases **Did authors find a dose–response relationship (yes/no)?:** Yes (not statistically significant).	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
		Finnish Cancer Registry (1957–1980). **Information on incidence:** 60 respiratory cancers were found. The following respiratory cancers were considered: malignant tumours of the following primary sites (number of cases found in the current study): tongue (N=1), other mouth (N=0), pharynx (N=1), nose/sinuses (N=0), larynx/epiglottis (N=4) and lung/trachea (N=54). Note that none of the examined individuals had malignant tumours in the mouth, nose or sinuses. **Mortality prevalence/percentage of the control group or general population (background data):** 67% of controls and 5% of cases were still alive **Type of statistical analyses:** ORs were calculated by maximum likelihood method. **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Smoking status and latency were considered, but not in the analyses examining (cumulative) wood dust exposure.		

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
Demers et al. (1995) (93)	**Type of study:** Pooled case–control study, retrospective **Country (region):** Pooled data from seven countries (China, France, Germany, Italy, Netherlands, Sweden, US) **Type of industry:** Wood-related industries **Follow-up period:** Not reported: cases identified in '70s and '80s. **Study period:** Not reported **Number of participants:** Men: 680 cases (169 adenocarcinomas, 329 squamous cell cancers, 157 other histologies, 25 unknown histology). Women: 250 cases (26	**Exposure measurement data:** Assumed 8-hour TWA average exposure categories: none, low (<1 mg/m^3), moderate (1–5 mg/m^3), high (>5 mg/m^3). Means and distribution not reported. **Personal or stationary sampling of exposure levels:** Not applicable **Number of measurements:** Not applicable **Methods of exposure measurement and analysis:** Not applicable **Job and exposure history (JEM, questionnaires, census data, etc):** JEM developed by one of the investigators. Work history data from the original studies (for some studies recoded). Classification into categories based on highest level of wood dust exposure during total work history **Job classification:** 7 job categories **Woodworking processes performed (e.g. sawing, drilling):** All woodworking operations **Types of wood:** Information not available for all 12 studies **Types of tool used:**	**Types of health effect observed:** Sinonasal cancer (all histologies, adenocarcinoma, squamous) **Single, short-term exposure or long-term exposure:** Long-term (highest level of wood dust exposure during total work history) **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** OR men All histologies: None = ref Low: 0.8 (0.4–1.5) Moderate: 1.2 (0.9–1.6) High: 5.8 (4.2–8.0) Adenocarcinoma: None = ref Low: 0.6 (0.1– 4.7) Moderate: 3.1 (1.6–6.1) High: 45.5 (28.3–72.9)	Exposure categories indicate highest level of wood dust exposure during total work history and not cumulative exposure. Pattern of results for adenocarcinomas were consistent between most studies, but there was heterogeneity in effect size. For squamous cell carcinomas, there was considerable heterogeneity between studies.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	adenocarcinomas, 101 squamous cell cancers, 105 other histologies, 18 unknown histology) **Number and origin of controls:** 2349 men, 787 women. Origin differed between studies (population-based registries, hospitals, friends or neighbours, random digit dialling) **Name of study/ cohort:** Unknown **Earlier publications on the same cohort:** None known	Not reported. **Types of medical examination:** All cases identified by histology. Source of cases: hospital or tumour registries **Information on incidence:** Not reported **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Logistic regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: analyses adjusted for study and age category; adjustment by cigarette smoking (not reported) resulted in OR <10% different from OR without this adjustment.	Squamous: not significant for all exposure categories Women: not significant except for adenocarcinoma and low exposure: OR 7.7 (1.3–44.8); no cases in moderate and high exposure groups. **Results of a survival analysis:** Not performed **Stratified results (dose–response relationship):** See above. **Did authors find a dose–response relationship (yes/no)?:** Yes.	
Ekpanyaskul et al. (2015) (193)	**Type of study:** Case–control study **Country (region):** Thailand **Type of industry:** Not applicable	**Exposure measurement data:** Low intensity (score 1) 0.02–1 mg/m^3, moderate intensity (score 2) >1–10 mg/m^3, high intensity (score 3) >10 mg/m^3 of total dust. Percentage none, low, moderate, high in cases: 81, 8.9, 9.5, 0.6; in controls: 88.7, 4.3, 6.7, 0.3.	**Type of health effects observed:** NPC, 3 types: keratinising squamous cell carcinoma (type 1) and non-keratinising carcinoma, which is further	No remarks.

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Follow-up period: Not applicable **Study period:** 2007–2009 **Number of participants:** 327: new incident NPC patients, recruited at their first visit to the centre and histologically diagnosed during the period 2007–2009 **Number and origin of controls:** 327: randomly selected from healthy persons who visited non NPC patient admitted to the same centre, recruited in the same study period and matched by age, gender and centre **Name of study/ cohort:** Unknown	Median cumulative exposure cases + controls = 70 (intensity x duration (years)) **Personal or stationary sampling of exposure levels:** Not applicable **Number of measurements:** Not applicable **Methods of exposure measurement and analysis:** Not applicable **Job and exposure history (JEM, questionnaires, census data, etc):** JEM. Complete detailed occupational histories via interviews. All occupations assessed with respect to probability, frequency and intensity of exposure to wood dust by 3 experts. **Job classification:** By probability, frequency and intensity of exposure to wood dust. Top 3 wood-related occupations were carpenter, construction worker and cabinet maker. **Woodworking processes performed (e.g. sawing, drilling):** Not reported **Types of wood:** Mainly hardwood (most likely also including treated wood) **Types of tool used:**	characterised as differentiated (type 2) or undifferentiated (type 3). **Single, short-term exposure or long-term exposure:** Long-term **Data on SIR/SMR/RR (no. observed/no. expected cases) and 95% CI or p-values:** NPC2&3, adjusted OR, Cumulative exposure: None: ref. <70: 1.39 (0.75–2.60) ≥70: 2.17 (1.03–4.58) NPC2&3, adjusted OR, Intensity: None: ref. 0.02–1 mg/m^3: 2.09 (0.99–4.37) >1-10 mg/m^3: 1.45 (0.78–2.69) >10 mg/m^3: 1.61 (0.13–20.14) No dose–response for all NPC types **Results of a survival analysis:**	

Reference	Study design and population	Data on exposure and health assessment	Results	Remarks (conclusion regarding quality)
	Earlier publications on the same cohort: Ekburanawat et al. (2010) (205)	Not reported **Types of medical examination:** Histology to define three histologic types: keratinising squamous cell carcinoma (type 1) and non-keratinising carcinoma, which is further characterised as differentiated (type 2) or undifferentiated (type 3). **Information on incidence:** New incident NPC patients recruited at their first visit to the National Cancer Institute in Bangkok and 5 regional cancer centres and histologically diagnosed during 2007–2009. **Mortality prevalence/percentage of the control group or general population (background data):** Not reported **Type of statistical analyses:** Conditional logistic regression **Whether authors took into account other risk factors, such as smoking and mixed exposure to other compounds:** Yes: adjustment for educational level, smoking status, and alcohol intake. Epstein-Barr virus (EBV) seropositivity was considered but not adjusted for because >90% were seropositive.	Not performed **Stratified results (dose–response relationship):** See above. **Did the authors find a dose-response relationship (yes/no)?:** Yes: in WHO types 2 and 3 with cumulative exposure.	

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