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Research Article

Occupational Wood Dust Exposure and Risk of Chronic Obstructive Pulmonary Disease: A Meta-Analysis

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ABSTRACT

Background: Occupational wood dust exposure has been concerned by several studies, which suggest that wood dust inhalation may be associated with an increased risk of chronic obstructive pulmonary disease (chronic obstructive pulmonary disease, COPD). To assess this association more accurately, this study explored the relationship between occupational exposure to wood dust and COPD.

Method: Retrieve literature from the establishment of the database to January 2024 on wood dust and COPD, including databases such as PubMed, Springer, CNKI (China National Knowledge Infrastructure) and Wanfang. Use the Agency for Healthcare Research and Quality (AHRQ) scale to assess the quality of articles that meet the criteria. Judge the heterogeneity of the articles based on I2 and P values and select the appropriate effect model. Evaluate publication bias through Begg's and Egger's tests.

Results: A total of 437 relevant documents were retrieved and 13 studies, including 3 cohort studies and 6 cross-sectional studies, were included in this meta-analysis. There were 4,367 cases of COPD patients who had not been exposed to wood dust and 1,590 cases of COPD patients who had been exposed. The literature quality scores were all above 5. There was high heterogeneity among the studies (I2 = 94%) and a random effects model was used to analyze the combined odds ratio (OR). The combined OR value indicated that exposure to wood dust increases the risk of developing COPD (OR = 0.58, 95%CI = 0.35 ~ 0.96) and even after controlling for smoking factors, exposure to wood dust still showed an increased risk of developing COPD (OR = 0.53, 95%CI: 0.30 ~ 0.83).The results of subgroup analysis showed that OR (95%CI) was greater than 1, except 6 studies with sample size <1000,3 cross-sectional studies, 4 FEV 1 / FVC <0.7,2 FEV 1 / FV C <LLN and 3 studies before 2020.

Conclusion: Contact with wood dust in the process of labor can increase the risk of COPD for occupational people. We should pay attention to the health monitoring of wood dust staff, control the possible condition as soon as possible and maintain regular follow-up after leaving the post.

Keywords: Occupational contact, Occupational exposure, Wood dust, COPD, Meta

1. Introduction

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable chronic non-communicable disease characterized by persistent respiratory symptoms and airflow limitation[1]. At present, COPD has become the third leading cause of death in China and even in the world, bringing a heavy burden of disease to the society[2]. Occupational exposure plays an important role in obstructive airway disease and is one of the risk factors contributing to COPD[3].Wood is an important raw material in the production of housing construction, household products and daily necessities. Wood dust is known to cause adverse health effects, including pneumoconiosis and fibrosis. Although the harm of wood dust to the lung has been determined, the epidemiological studies of COPD did not determine the quantitative degree of harm to humans and there are many mixed conclusions. Studies[4,5] reported a positive relationship between the two and studies[6,7] reported no significant association. To clarify this issue, a systematic review and meta-analysis of the epidemiological evidence on the relationship between occupational wood dust exposure and COPD risk were conducted and the results are reported below.

2. Method

2.1. Data retrieval

Retrieving the literature on wood dust and COPD from library building to January 2024, of which the English literature is obtained from the Pub Med and Springer databases, The search terms are Wood dust, hardwood dust, cork dust, wood chips and carpentry and chronic obstructive airway diseases, COPD, chronic respiratory diseases. Chinese literature from CNKI and Wan fang database search terms are wood dust, hard wood dust, cork dust, wood chips, woodworking, chronic obstructive pulmonary disease and respiratory disease.

2.2. Selection and extraction of the studies

Inclusion criteria:

- Published literature.
- Case-control study or cohort study.
- Subjects were only exposed to wood dust.
- The relationship between occupational exposure to wood dust and COPD onset or death was evaluated in the paper, calculating the effect value and 95% confidence interval (95%C1).
- Repeat studies with the latest sample size and the latest year of publication.

Elimination criteria:

- Repeat study.
- Outcome, acute bronchitis, acute / chronic bronchitis, asthma or other lung diseases.

2.3. Data extraction

By reading, the duplicate documents were removed. Carefully browsing the title and abstract of the paper, according to the inclusion and exclusion criteria, the control group was not exposed to wood dust or exposed but with low concentration and the contact group was workers exposed to wood dust or high concentration of wood dust. Check whether the outcome complies with the purpose of the study. In order to further improve the research content, the references of the relevant documents that meet the requirements are also compared. The focus and extracted contents included author (length of publication), study type, country, sample size, subject age, source of COPD diagnosis or (95%CI) value and adjusted confounding factors.

2.4. Quality evaluation of literature

The quality of the literature was assessed against the Healthcare Research and Quality Scale (AHRQ)[8]. There are 11 items in the self-rating scale (with “yes”, “no” and “unclear” respectively[9]:

- Is the source of the data clearly identified (survey, literature review)?
- Are the inclusion and exclusion criteria for the exposed and non-exposed groups (or cases and controls) listed or are previous publications cited?
- Is the time stage of identifying the patients given?
- Are the subjects continuous if it is not of population origin?
- Does the subjective factors of the evaluator obscure other aspects of the research subjects?
- Describes any assessment to ensure quality (e. g. testing of primary outcome measures).
- Explains any patient excluding analysis.
- Describes how to evaluate and (or) measures to control confounders.
- Explains how, if possible, missing data was processed in the analysis.
- Summarizes the patient response rate and the completeness of data collection.
- Identifying the percentage of expected incomplete data or follow-up results if, follow-up is available.

Each item is worth 1 point. The literature was scored by 2 researchers following the assessment protocol and any inconsistent scores were resolved by group discussion. The literature quality is divided into the following categories: low quality = 0~3 points; medium quality = 4~7 points; high quality = 8~11 points.

2.5. Statistical treatment

RevMan 5.4.1 software was used to analyze the extracted data, standardized mean difference (standardized mean difference, SMD) was used to describe the effect analysis statistics and 95%CI, combined with the Q test and I^2 to determine whether there is heterogeneity and its size, I2> 50% considered the random effect model; otherwise, the fixed effect model was used. The funnel plot and Egger’s test were combined to analyze for publication bias.

3. Results

3.1. Basic information of literature

The retrieval procedure is shown in **(Figure 1)**. The retrieval procedure is shown in **Figure 1**. A total of 437 related documents were retrieved (including Pub Med234, Springer 106,65 on CNKI and 32 on Wanfang). According to the criteria of literature inclusion and elimination, duplicate documents were excluded and after the exposure group information or the exposure group information was excluded, a total of 8 articles were included in the meta-analysis[10-18].

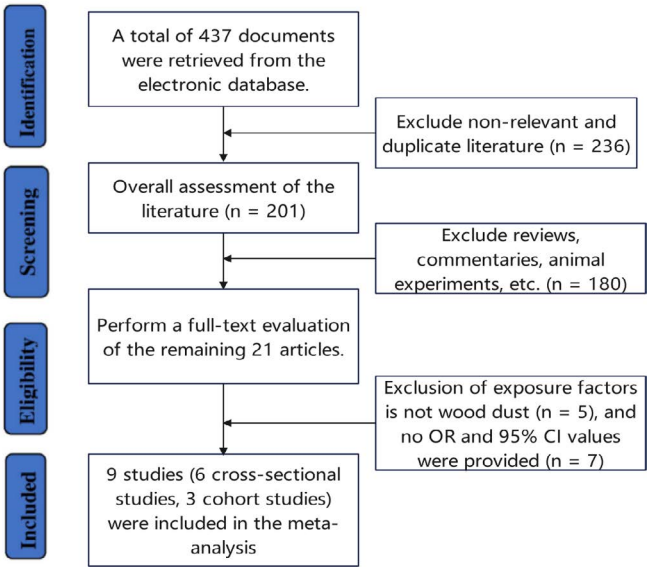

Figure 1: A meta-analysis of the literature inclusion flow.

Nine articles were retrieved in this study, including two from cohort studies[14,16], Cross-sectional studies in 7 articles[9-13,15,17], Results from a total of 13 studies, there were 4367 COPD patients without wood dust exposure and 1590 COPD patients without wood dust exposure. The exposed population are wood processing or carpentry. According to the study area, 3[15-17] were obtained from Denmark, 1 for in Sweden[9], Norway[10], New Zealand[11], Italy, Congo[13] and USA[14].The basic characteristics of the included literature are shown in **(Table 1)**.

3.2. Meta analyses

The 13 included studies had low heterogeneity (I2 = 94%, P = 0.03) and were analyzed using the random effects model. The combined OR values suggested that exposure to wood dust increased the risk of COPD (OR = 0.58, 95%CI = 0.35 ~ 0.96) **(Figure 2)**. While adjusting for smoking among confounding factors, it showed that exposure to wood dust increased the risk of COPD (OR = 0.53, 95%CI: 0.30 ~ 0.83) **(Figure 2)**.

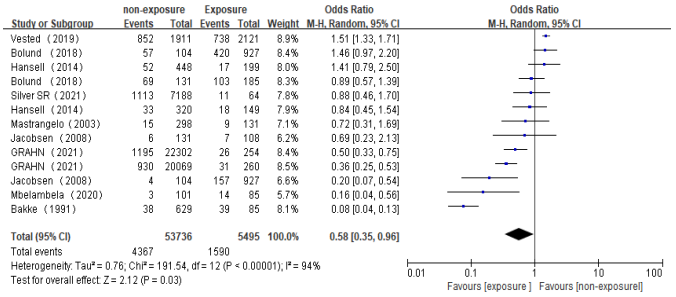

Figure 2: Forest plot of wood dust and COPD disease conditions.

3.3. Published bias test

Publication bias tests were conducted on the literature and data included in the analysis and both Begg's rank correlation test and Egger's linear regression method indicated the presence of publication bias in the study (P = 0.32), as shown in **(Figure 3)**. Due to the small number of studies, the trim and fill method was used to further assess the stability of the publication outcomes. Four hypothetical studies were added in the preliminary meta-analysis to meet the requirements for no publication bias and the results after adding these four hypothetical studies were OR = 1.13 (95%CI = 1.07 ~ 1.43), consistent with the original range of results, still supporting the conclusion that exposure to wood dust increases the risk of developing COPD.

3.4. Subgroup analysis

Subgroup analysis of different sources of COPD diagnosis, study region and study type. Using sample size, study type, source of COPD diagnosis and year of publication, the results showed that except for six studies with sample size <1000, three cross-sectional studies, four FEV 1 / FVC <0.7, the other sub combinations and OR (95%CI) were greater than 1 **(Table 2)**.

Table 1: Basic characteristics of the literature included.

Author (year of publication)	The type of research	country	Case / non-exposure	Case / exposure	Age / year	COPD, the confirmed source	Crude OR (95%CI) value	Adjust the OR (95%CI) value	Adjusted for confounding factors	quality grade
GRAHN[9] (2021)	cohort study	Sweden	930/20069 1 195/22302	31/5706/254	25 ~ 70	Doctor diagnosis	man:1.26 (0.88–1.81) woman:0.41 (0.18–0.9)	man:0.36 (0.25~0.53) woman:0.50 (0.33~0.75)	Age, smoking	8
Bakke[10](1991)	cross-sectional study	Norway	38/629	9/85	15~70	FEV1/FVC<0.7	1.8 (0-8 -3-5)	0.08 (0.04~ 0.13)	Gender, age, smoking	6
Hansell[11] (2014)	cross-sectional study	New Zealand	52/448 33/320	17/199 18/149	25~75	Doctor diagnosis/ FEV1/FVC< LLN	man:0.78 (0.40–1.53) woman:0.78 (0.35–1.74)	man:1.41 (0.79~2.50) woman:0.84 (0.45~1.54)	Gender, age, smoking, ethnicity and household income	6
Mastrangelo[12] (2003)	cross-sectional study	Italy	15/298	9/131	44~65	FEV1/FVC<0.7	1.16 (1.08–1.24)	0.72 (0.31~0.69)	Age, smoking	7
Mbelambela[13] (2020)	cross-sectional study	Congo	3/101	14/85	40~70	FEV1/FVC<0.7	2.6 (1.7-5.9)	0.16 (0.04~0.56)	Age, height, education level and passive smoking	6
Silver SR[14] (2021)	cohort study	USA	1113/7188	11/64	50 ~ 62	Doctor diagnosis	0.86 (0.501.56)	0.88 (0.46~1.70)	Gender, nationality, culture, family income, smoking	8
Jacobsen[15] (2008)	cross-sectional study	Denmark	4/104 6/131	157/927 7/108	>40	FEV1/FVC<0.7	man:1.39 (0.61–3.15) woman: 3.86 (0.62–23.70)	man:0.20 (0.07~0.54) woman:0.69 (0.23~2.13)	Gender	6
Bolund[16] (2018)	cohort study	Denmark	69/131 57/104	103/185 420/927	16~67	FEV1/FVC<LLN	man:0.72 (0.2–2.4) woman:12.00 (1.3–11.10)	man:0.89 (0.57~1.39) woman:1.46 (0.97~2.20)	Age, height and gender	6
Vested[17] (2019)	cross-sectional study	Denmark	852/1911	738/2121	19~63	Doctor diagnosis	0.73 (0.33-1.59)	1.51 (1.33~ 1.71)	Gender, age and cigarette smoking	6

Table 2: Results of the Meta subgroup analysis of the relationship between wood dust occupational exposure and copd.

Subgroup analysis		Study the number	OR (95%CI)	P	I^2 / %
Sample size					
	≥1000	3	1.26(1.12~1.41)	<0. 001	96
	<1000	6	0.66(0.55~0.81)	<0. 001	92
The type of research					
	cross-sectional study	6	1.20(1.07~1.33)	<0. 001	94
	cohort study	3	0.59(0.47~ 0.74)	0.008	74
COPD, the confirmed source					
	Doctor diagnosis	4	1.25(1.12~1.40)	<0. 001	93
	FEV1/FVC<0.7	4	0.18(0.12~0.28)	<0. 001	90
	FEV1/FVC<LLN	2	1.00(0.74~1.36)	0.38	0
The year of publication					
	Before 2020	6	1.20(1.08~1.34)	<0. 001	94
	After 2020	3	0.45(0.35~ 0.58)	0.04	64

4. Discussion

The results of 13 studies selected in this study showed that exposure to wood dust during labor is a risk factor for COPD (OR = 0.58, 95%CI = 0.35 ~ 0.96) and the results were relatively reliable.

COPD is a common, preventable and treatable condition characterized by persistent respiratory symptoms and airflow limitation, usually due to airway and (or) alveolar abnormalities caused by apparent exposure to toxic particles or gases[18]. COPD is due to lung structural changes, small airway stenosis and destruction of lung parenchyma, which destroys the attachment of alveoli and small airways, resulting in the decline of the elastic retraction ability of lung[19]. Workers in different occupational categories who are exposed to harmful gases, dust and smoke during their work activities can increase the risk of developing COPD, such as farmers, miners, construction workers and operators in other industries (such as those exposed to metal fumes like cadmium and aluminum)[20], Exposure to occupational hazards and smoking significantly increases the risk of developing COPD. Smoking is the most studied risk factor associated with COPD and many studies consistently identify smoking as the primary risk factor for the development of the disease. However, there is sufficient evidence available to suggest that nonsmokers may also have COPD. Nearly one-quarter of confirmed COPD cases in Japan, the United Kingdom and the US occur among non-smokers[21].Therefore, in this meta-analysis, the factor of smoking was adjusted and the adjusted summary OR was 0.53 (95%CI: 0.30 ~ 0.83), indicating that non-smokers exposed to wood dust still have an increased risk of developing COPD. The pathogenic mechanism of COPD caused by exposure to wood dust is not yet clear, but some studies have found that after workers are exposed to productive dust, the bronchial mucosa is repeatedly stimulated, thus reducing its ability to clear foreign bodies, making it easier for bacteria to invade the respiratory tract and cause infections, leading to irreversible airflow limitation[22];Long-term inhalation of dust can cause the shortening of respiratory cilia and induce the occurrence of COPD[23]. The particle size, composition and exposure time of wood dust also affect the risk of developing COPD. Fine dust particles are more likely to penetrate deep into the lungs, causing more severe inflammatory responses and tissue damage[24].Different types of wood dust may have varying biological activities, leading to different levels of damage to the respiratory system. COPD is a disease caused by multiple factors and occupational exposure is a hazard factor that is not widely noticed. Wood dust is deposited in the alveoli after inhalation in wood processing and contains a variety of substances and microorganisms, which can cause inflammation and airway obstruction and pose a threat to the health of occupational people[25]. In addition, wood dust contains lignin, cellulose, saponin, glycoside, benzene, phenol, silicon, ene, acid quinone and other substances and various microorganisms attached to them, which can cause body inflammatory reaction, airway obstruction and other lung acquired diseases and cause the health harm of occupational people. Therefore, the implementation of occupational protective measures and regular health check-ups are particularly important for high-risk occupational groups[26]. Future research should further explore the specific pathogenic mechanisms of wood dust to develop more effective prevention and intervention measures, reducing the incidence of occupational COPD.

This meta-analysis has some limitations: First, although both Chinese and English literature were searched, no Chinese literature that met the criteria was included. Second, the analysis was not stratified by the length of employment in jobs with exposure to wood dust, as most of the included studies only assessed whether participants had been exposed to wood dust. Third, the concentration of wood dust exposure was not further considered. Fourth, there is a certain degree of publication bias in this study, indicating that there may be some gray literature that has not been searched for.

In conclusion, this study explored the heterogeneity between inter-studies by systematic search, meta regression, analyzed the combined OR by random effects model and assessed publication bias by Begg's and Egger's tests and thus concluded that exposure to wood dust during labor process could increase the risk of COPD in occupational groups.

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