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The association between wood dust exposure and respiratory disorders and oxidative stress among furniture workers

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Summary
Background This study was undertaken to determine the effect of wood dust on the respiratory system and oxidative stress in furniture workers and to determine whether any associations exist between respiratory parameters and oxidative stress.
Methods This cross-sectional study was performed on 45 furniture workers and 45 office workers as a reference group in Iran.

The NIOSH method 0600 was used to determine the concentration of particulates. The prevalence of respiratory symptoms was estimated via the European Community Respiratory Health Survey (ECRHS) questionnaire. Oxidative stress biomarkers and respiratory parameters were also measured.
Results The mean concentrations of respirable and non-respirable dust were found to be 1.51 mg/m^3 and 1.23 mg/m^3, respectively. Pulmonary function parameters, including forced vital capacity (FVC), forced expiratory volume in the first second (FEV1), FEV1/FVC, and antioxidant capacity biomarkers such as total antioxidant capacity (TAC) and superoxide dismutase (SOD) were significantly lower, while the prevalence of respiratory symptoms, and malondialdehyde (MDA) levels, were significantly higher in the furniture workers than in the reference group. There were significant positive associations between FVC and FEV1 with SOD and TAC.
Conclusion The present study results indicated that exposure to wood dust significantly increased respiratory disorders and confirmed the association between lung function parameters and oxidative stress.

Keywords Wood workers · Spirometry · Antioxidant defence

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Introduction

It is estimated that 3.6 million workers in the European Union are exposed to wood dust [1–3].

Furniture makers, cabinet makers, carpenters, and veneer/plywood production workers are exposed to wood dust [4, 5].

Wood dust is classified into three categories: total, inhalable and respirable dust [6]. The particles with a mass median aerodynamic diameter of 100 µ are inhalable, and those with a mass median aerodynamic of 4 µ which deposit in the lower respiratory tract and interact with alveolar macrophages, are respirable [7]. It is worth noting that respirable wood dust ranges from 6% to 75% of the total wood aerosol [6, 8].

The American Conference of Governmental Industrial Hygienists (ACGIH) recommends an 8-h threshold limit values-time weighted average (TLV-TWA) of 0.5 mg/m^3 (inhalable fraction) for western red cedar and a TLV-TWA of 1 mg/m^3 (inhalable fraction) for all other types of wood [9].

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The Occupational Safety and Health Administration (OSHA) proposed a permissible exposure limit-timeweighted average (PEL-TWA) of 5 mg/m^3 for respirable dust.

Exposure to wood dust can cause inflammation and irritation of the respiratory system (coughing, wheezing, chronic bronchitis, tightness of the chest, and asthma) [6], dermatitis, urticaria, alveolitis, and decline in lung function, especially FEV1 [10].

Although most of these symptoms occur at concentrations higher than 5 mg/m^3 [10], some studies have shown that exposure to wood dust at concentrations below 5 mg/m^3 can cause sinusitis and other pulmonary diseases [11]. Elavarasi et al. [12] observed a significant increase in genotoxicity among the furniture workers exposed to wood dust at concentrations lower than 0.3 mg/m^3 compared to the reference group. They suggested that lower TLV is required to protect woodworkers.

The first study indicating an increased risk of nasal cancer in woodworkers was published in 1968. This finding was confirmed by a number of subsequent studies [13], and in 1995, wood dust was classified as carcinogenic to humans (Group I) by the International Agency for Research on Cancer (IARC) [14].

While in some studies, exposure to wood dust was associated with impaired lung function [10, 15–17], other studies have failed to show such an association [18–22].

For instance, Cormier et al. [20], Arbak et al. [18], and Borm et al. [19] showed normal respiratory parameters in woodworkers. Also, the studies conducted by Jacobsen et al. [21, 22] in 2008 and 2013 revealed no significant difference in FEV1, FVC, FEV1/FVC values between the workers exposed to wood dust and the reference group.

Oxidative stress is considered as one of the mechanisms by which wood dust exerts its toxicity on the lungs [14, 23, 24].

Oxidative stress impairs skeletal muscle contractions leading to the dysfunction of respiratory muscles, particularly of the diaphragm, in patients with severe chronic obstructive pulmonary disease (COPD) [25]. Some studies, such as those conducted by Noertjojo et al. [26] and Fante et al. [27] found obstructive spirometric patterns in the groups exposed to wood dust.

Oxidative stress is an imbalance between the production of free radicals and antioxidant defense. Free radicals have one or more unpaired electrons, and the radicals derived from oxygen and/or nitrogen are the most important free radicals produced in living systems. Direct measurement of oxidative stress biomarkers, such as reactive oxygen species (ROS) and reactive nitrogen species (RNS) is not easy [25]. Therefore, stable molecular products formed via the reaction of ROS and RNS with certain biomolecules such as lipids are measured. The main primary products of lipid peroxidation include isoprostanes, malondialdehyde (MDA), thiobarbituric acid reactive substances (TBARS), lipid hydroperoxides (LOOHs), and conjugated dienes (CDs) [25]. MDA is used in many disorders, such as COPD, as a biomarker of oxidative stress [28]. In 2021, Bhat et al. [29] suggested that serum MDA can be used as an indicator of reduced pulmonary function in cases of difficulty in performing spirometry.

The present study aimed to measure exposure to wood dust among furniture and office workers, to compare lung function parameters, the prevalence of respiratory symptoms, and levels of total antioxidant capacity (TAC) and superoxide dismutase (SOD) as biomarkers of antioxidant status, as well as the mean level of MDA as an indicator of oxidative stress between groups and determine whether any associations exist between lung function parameters and oxidative stress.

Methods

Population and sample of the study

This cross-sectional study was performed on 45 furniture workers exposed to wood dust in Yazd Province, Iran. The reference group included 45 office workers with no history of occupational or nonoccupational exposure to dust or other chemicals which can lead to pulmonary diseases. The participants with a working history of less than 6 months, body mass index greater than 30 kg/m^2, and those with a personal or family history of acute respiratory infections, chronic inflammatory diseases [30], asthma, heart disease, and those who had used vitamins E and C in the last month were excluded from the study.

Data collection

Estimation of the prevalence of respiratory symptoms

The European Community Respiratory Health Survey (ECRHS) questionnaire was used to estimate the prevalence of respiratory symptoms in the exposed and reference groups [31].

Measurement of lung function parameters

Spirometric data were collected before the coronavirus disease 2019 (COVID-19) outbreak in October 2019. Lung function parameters, including forced vital capacity (FVC), forced expiratory volume in the first second (FEV1), forced expiratory flow at 25% and 75% of the pulmonary volume ($FEF_{25–75}$), peak expiratory flow (PEF), and FEV1/FVC ratio, were measured at the end of the working shift using a COSMED Pony Fx desktop spirometer (Metabolic, Rome, Italy).

Exposure assessment

The NIOSH method 0600 [32] was used to determine the concentration of respirable dust with a 50% cut-off point of 4.0 µm at the breathing zone of the stud-

ied groups. The sampling was performed at a flow rate of 2.20 l/min in the morning shift. To determine the respirable dust content, the 10-mm Higgins Dewell cyclone with a 37-mm PVC filter (pore size 5.0 µm) loaded onto a three-piece filter cassette was used. The rapid circulation of air in the cyclone separates particles according to their equivalent aerodynamic diameter. Thus, the respirable dust particles collect on a filter for analysis, while the larger dust particles fall into the grit pot at the bottom of the cyclone. Before and after sampling, the PVC filter and grit pot were weighed with a Sartorius balance (Goettingen, Germany) to measure respirable and non-respirable dust. The same procedures were used to measure wood dust in the reference group.

Relative humidity, air pressure, and temperature were measured at the sampling site. Dust concentration (mg/m^3) was determined after correcting the sampling air volume.

Determination of oxidative stress biomarkers

A 5 ml blood sample was collected from each participant using a syringe and transferred to plain tubes. Serum was separated by centrifugation at 1372 × g for 10 min. The TAC and MDA levels, and SOD activity were measured using a commercially available Zellbio kit (Zellbio Lab, Ulm, Germany) according to the manufacturer's protocol.

Ethics

The aim of the study was explained to all participants, and they signed informed consent. The study protocol was approved by Shahid Sadoughi University of Medical Sciences Research Ethics Committee (IR.SSU.SPH.REC.1399.125).

Statistical analysis

Data analysis was performed using SPSS Statistics 21. A χ^2-test was used to assess respiratory symptoms. Student's t-test was used to compare quantitative indices between the exposed and reference groups. A logistic and multiple linear regression analysis was used to model and assess the association between wood dust exposure and prevalence of respiratory symptoms, lung function parameters, and oxidative stress biomarkers after controlling for confounding variables (age, work history, weight, height, smoking status). Pearson's correlation was used to correlate oxidative stress biomarkers with lung function parameters.

Results

Table 1 shows the mean age, work history, height, weight, smoking status, marital status, and level of education in both exposed and reference groups. The results showed no significant differences in age, work history, height, weight, and smoking status between the two groups; however, differences in marital status and level of education were observed between the two groups. 10 exposed group and 4 reference sroups were smokers, and the difference was not statistically significant.

The mean concentrations of respirable and non-respirable particles in furniture workers were found to be 1.51 mg/m^3 and 1.23 mg/m^3, respectively. Particles

Table 1 Demographic variables and levels of exposure to particles

Parameter		Exposed group Mean (SD)	Reference group Mean (SD)	*p*-value
Age (years)		36.60 (8.68)	33.49 (8.68)	0.670 [a]
Work history (years)		8.93 (8.18)	9.00 (5.07)	0.963 [a]
Height (cm)		171.82 (7.83)	174.24 (7.50)	0.138 [a]
Weight (kg)		80.28 (12.55)	74.02 (16.97)	0.051 [a]
Number (Percent)				
Smoking status	Yes	10 (71.40)	4 (28.60)	0.144 [b]
	No	35 (46.10)	41 (53.90)	
Marital status	Single	14 (100.00)	0 (0.00)	< 0.001 [b]
	Married	31 (40.80)	45 (59.20)	
Level of education	No degree	21 (100.00)	0 (0.00)	< 0.001 [b]
	Elementary education	4 (80.00)	1 (20.00)	
	High school diploma	16 (84.20)	3 (15.80)	
	Associate degree	2 (11.10)	16 (88.90)	
	Bachelor degree	2 (13.30)	13 (86.70)	
	Masters degree	0 (0.00)	12 (100.00)	
Respirable dust concentration (mg/m^3)		1.51 (0.47)	ND	–
Non-respirable dust concentration (mg/m^3)		1.23 (0.41)	ND	

Parameters without units are numbers (percent), parameters with units are Mean (SD)
ND not detectable, *SD* Standard deviation
[a]Independent sample t-test
[b]χ^2-test

Table 2 Frequency of respiratory symptoms in the exposed and referent groups

Parameter		Exposed group *Number* (Percent)	Reference group *Number* (Percent)	*p*-value [a]
Wheezing	Yes	16 (80.00)	4 (20.00)	0.002
	No	29 (41.40)	41 (58.60)	
Chest tightness	Yes	11 (78.60)	4 (21.40)	0.020
	No	34 (44.70)	42 (55.30)	
Coughs	Yes	9 (90.00)	1 (10.00)	0.007
	No	36 (45.00)	44 (55.00)	
Chronic coughs	Yes	7 (63.60)	4 (36.40)	0.334
	No	38 (48.10)	41 (51.90)	
Sputum	Yes	13 (81.20)	3 (18.80)	0.006
	No	32 (43.20)	42 (56.80)	
Shortness of breath	Yes	16 (72.70)	6 (27.30)	0.014
	No	29 (42.60)	39 (57.40)	
Asthma	Yes	4 (80.00)	1 (20.00)	0.361
	No	41 (48.20)	44 (51.80)	

Numbers without units are numbers (percent)
[a] χ^2-test

Table 3 Pulmonary function parameters and oxidative stress biomarkers in the exposed and reference groups

Parameter	Exposed group Mean (SD)	Reference group Mean (SD)	*p*-value [a]
FVC (%)	93.37 (7.62)	98.17 (12.09)	0.027
FEV1 (%)	88.08 (10.05)	94.24 (11.43)	0.008
FEV1/FVC (%)	83.64 (12.10)	89.62 (8.44)	0.008
FEF 25–75 (%)	80.17 (13.40)	82.26 (10.38)	0.411
PEF (%)	81.84 (9.08)	84.35 (10.52)	0.260
SOD (U/ml)	9.22 (2.42)	10.59 (2.20)	0.006
TAC (mM)	1.16 (0.13)	1.48 (0.29)	<0.001
MDA (μM)	3.85 (2.11)	2.65 (0.68)	<0.001

[a] Independent sample t-test

were not detectable in the work environments of the reference group.

The prevalence of respiratory symptoms in the studied groups is presented in Table 2.

As can be seen, the prevalence rates of all symptoms (except for chronic cough and asthma) were significantly higher in the exposed group than in the reference group ($p<0.05$).

As shown in Table 3, some pulmonary function parameters such as FEV1/FVC, FVC, and FEV1were significantly lower in the exposed group than in the reference group.

Table 3 also shows the mean levels of TAC, SOD, and MDA.

The mean values of TAC and SOD were significantly lower in the exposed group than in the reference group (TAC: 1.16 ± 0.13 mM vs. 1.48 ± 0.29 mM; SOD: 9.22 ± 2.42 U/ml vs. 10.59 ± 2.20 U/ml).

Conversely, the MDA level was significantly higher in the exposed group (3.85 ± 2.11 μM) compared with the reference group (2.65 ± 0.68 μM).

Of the exposed subjects, 34 had a normal spirogram, while 13 and 2 exposed subjects had obstructive and restrictive lung disease, respectively. The corresponding values in the reference group were 39, 5, and 2, respectively. There was a significant difference in the prevalence of obstructive pattern between the two groups ($p=0.035$). No mixed lung pattern was observed.

Table 4 The association between exposure to wood dust and prevalence of respiratory symptoms before and after controlling for confounding variables

Parameter	OR crude (95% CI)	*p*-value [a]	OR adjusted (95% CI)	*p*-value [b]
Wheezing	5.65 (1.71, 18.67)	0.004	5.89 (1.68, 20.66)	0.006
Chest tightness	4.53 (1.17, 17.54)	0.029	4.38 (1.07, 17.86)	0.04
Coughs	11 (1.33, 90.95)	0.026	9.76 (1.14, 83.99)	0.04
Chronic coughs	1.88 (0.51, 6.96)	0.340	1.64 (0.41, 6.50)	0.48
Sputum	5.68 (1.49, 21.65)	0.011	5.83 (1.69, 23.27)	0.01
Shortness of breath	3.58 (1.25, 10.29)	0.018	3.47 (1.15, 10.45)	0.03
Asthma	4.29 (0.46, 40.01)	0.201	1.27 (0.09, 18.62)	0.86

OR Odds ratio, *95% CI* 95% Confidence interval
[a] χ^2-test
[b] Binary logistic regression analysis

Table 5 The association between wood dust exposure and lung function and oxidative stress parameters before and after controlling for confounders

Parameter	Crude regression coefficient (95% CI)	*p*-value[a]	Adjusted regression coefficient (95% CI)	*p*-value[b]
FVC (%)	–4.80 (–9.03, –0.54)	0.027	–4.35 (–8.81, 0.10)	0.05
FEV1 (%)	–6.15 (–10.66, –1.64)	0.008	–5.44 (–10.05, –0.83)	0.02
FEV1/FVC (%)	–5.97 (–10.34, –1.60)	0.008	–5.80 (–10.35, –1.24)	0.01
FEF 25–75 (%)	–2.09 (–7.11, 2.93)	0.411	–3.64 (–8.66, 1.38)	0.15
PEF (%)	–2.51 (–6.91, 1.89)	0.260	–3.39 (–7.83, 1.05)	0.13
SOD (U/ml)	–1.37 (–2.34, –0.39)	0.006	–1.43 (–2.45, –0.40)	0.007
TAC (mM)	–0.32 (–0.42, –0.23)	<0.001	–0.32 (–0.42, –0.22)	<0.001
MDA (μM)	1.19 (0.54, 1.85)	<0.001	1.23 (0.53, 1.93)	0.001

[a]Independent sample t-test
[b]Linear regression analysis

Table 6 The correlation between respiratory parameters and oxidative stress biomarkers in the studied groups

Parameter	FVC	FEV1	FEV1/FVC	SOD	TAC	MDA
FVC (%)	1	–	–	–	–	–
FEV1 (%)	0.60**	1	–	–	–	–
FEV1/FVC (%)	0.14	0.15	1	–	–	–
SOD (U/ml)	0.24*	0.43**	0.07	1	–	–
TAC (mM)	0.21*	0.30**	0.21	0.23*	1	–
MDA (μM)	–0.14	–0.13	–0.13	–0.16	–0.26*	1

Pearson's correlation *$P<0.05$; **$P<0.01$

To control the effects of confounders such as age, weight, height, work history, and cigarette smoking on the prevalence of respiratory symptoms, binary logistic regression analysis was used.

As shown in Table 4, before and after controlling for confounding variables, there was a significant association between exposure to wood dust and the prevalence of respiratory symptoms (except for chronic cough and asthma) ($p<0.05$).

After controlling for confounding variables, the risk of wheezing, chest tightness, coughs, sputum, and shortness of breath increased by 5.89, 4.38, 9.76, 5.83 and 3.47 times, respectively, as a result of exposure to wood dust.

Table 5 shows the association between exposure to wood dust and changes in lung function parameters and oxidative stress biomarkers before and after controlling for confounding variables. As the results show in both before and after controlling for confounding variables, a significant association was observed between wood dust exposure and reduction of FVC, FEV1, FEV1/FVC, SOD, and TAC values, so that these parameters would be reduced by 4.35, 5.44, 5.80, 1.43, and 0.32 units, respectively, after controlling for confounding variables. Exposure to wood dust significantly increased MDA levels.

Table 6 shows the correlation between respiratory parameters and oxidative stress in the studied groups. As shown in Table 6, a significant positive correlation was observed between FVC with SOD (r = 0.24) and TAC (r = 0.21). Also, FEV1 was positively correlated with TAC (r = 0.30) and SOD (r = 0.43).

Discussion

There was no statistically significant difference between the two groups in terms of age, height, weight, work history, and smoking status.

In the present study, the mean concentration of respirable dust (1.51 mg/m^3) was higher than non-respirable dust (1.23 mg/m^3). This could be explained by the fact that the rotational speed of machines, ambient temperature and humidity, and type of wood (hardwood or softwood) affect the size of particles. More heat generated by the increased speed of the tool during machining, in turn leads to a greater number of respirable dust compared to non-respirable dust [33].

Although mean concentration of respirable dust in this study was below occupational exposure limit set by OSHA, both upper and lower respiratory tract infections and genotoxicity as a result of exposure to wood dust at concentrations less than 0.5 mg/m^3 and 0.3 mg/m^3, respectively, have also been reported [11, 12].

Thepaksorn et al. [34] reported the mean concentration of respirable dust of 1.11 mg/m^3 in 704 wooden toy workers in Thailand. In Mandryk's study [35] the mean concentrations of inhalable dust in the green mills and dry mills were 1.52 mg/m^3 and 1.371 mg/m^3, respectively. About 70% of the exposures at the dry mills and 50% of exposures at the green mills were higher than the threshold limit value of 1 mg/m^3. The authors suggested that the use of old equipment, poor maintenance of the local ventilation system, leaking

ventilation ducts, and the shortage of respirators have caused high dust exposure levels among the workers.

A study in 25 EU countries on 3.6 million workers exposed to wood dust, including carpenters, sawmill workers, furniture workers, forestry workers, and other wood industry workers, demonstrated that 16% of the workers were exposed to respirable dust above 5 mg/m^3 [36]. In 2009, Osman et al. [10], in a study on 328 workers exposed to wood dust and 328 control group reported that the mean concentration of respirable wood dust was 2.04 mg/m^3, which is lower than the ACGIH TLV (5 mg/m^3). Of the workers 9.5% were exposed to wood dust at concentrations above 5 mg/m^3, and none of the workplaces had ventilation systems.

Moreover, the workers' exposure to respirable dust exceeded 5 mg/m^3 in the studies conducted by Magagnotti et al. [37] on 60 chipper operators in Italy, Abbas et al. [30] on 67 carpenters in Egypt, and Mohammadian et al. [38] on 10 wood workers in Iran.

Neghab et al. [39] in the sawmill workers in Iran, reported the mean concentrations of inhalable and respirable dust of 2.44 mg/m^3 and 6.76 mg/m^3, respectively, which is higher than ACGIH TLV-TWA. The authors suggested that the use of air jets and dry sweeping, lack of awareness of health effects of wood dust among the workers, and lack of a proper ventilation system might explain the reasons of these results.

The FVC, FEV1, and FEV1/FVC values were significantly lower in the exposed group than in the reference group.

The results are consistent with the findings of other studies on workers exposed to wood dust [10, 15–17, 40]. For example, Shamssain et al. [41] in a study of furniture makers, reported significant decreases in all spirometric indices, including FVC, FEV1, FEV1/FVC, FMF, FEF, and PEF in the wood exposed group in comparison with the non-exposed group. In Osman's study, the FVC values in the exposed group were significantly lower than that of the control group [10].

Beydon et al. [36] also showed that the respiratory parameters, including FVC, FEV1, and FEV1/FVC were significantly lower in sawmill workers exposed to wood dust in comparison to the non-exposed group.

Additionally, in the study conducted by Hosseini et al. [42] on 276 carpentry workers in Iran, the FEV1 and FEV1/FVC in the wood exposed group were significantly lower than those of the reference group. Similar findings have been reported by others [39, 43, 44]. After controlling for the important confounders, significant associations remained between exposure to wood dust and a decrease in FVC, FEV1, FEV1/ FVC values.

Similarly, the findings of Hessel et al. [45], Noertjojo et al. [26], and Neghab et al. [39] in the sawmill workers showed a significant association between exposure to wood dust and decreased lung function parameters after controlling the confounding variables.

In contrast, some studies have failed to find such an effect on wood dust. For example, surveys such as those conducted by Cormier et al. [20], Arbak et al. [18], and Borm et al. [19] showed that the pulmonary function of woodworkers was normal.

In both studies conducted by Jacobsen et al. [21, 22] in Denmark in 2008 and 2013, no significant difference in the FEV1, FVC, and FEV1/FVC values was observed between the wood exposed workers and the reference group.

The difference between the results of different studies may be due to differences in the workers' age, exposure duration [18], wood dust concentrations [22], the type of wood and processing method, used technology, the geographical area, the mill size or climatic conditions, and ventilation system [19, 20]. Wood dust exposure can be reduced by 3–10 times via good housekeeping and ventilation [6].

In the present study, the group exposed to wood dust had a significantly higher prevalence of respiratory symptoms, including coughs (90%), sputum (81.2%), chest tightness (78.6%), wheezing (80%), and shortness of breath (72.7%) when compared with reference group (coughs: 10%, sputum: 18.8%, chest tightness: 21.4%, wheezing: 20%, and shortness of breath: 27.3%). The risk of wheezing, chest tightness, cough, sputum and shortness of breath were 5.65, 4.53, 11, 5.68, and 3.58 times higher, respectively in the exposed group in comparison to the reference group.

In accordance with the present results, in Schlunssen's study, in Denmark, a significant increase in the cough and wheezing frequency in 54 furniture factories (2033 carpenters and 474 control members) in comparison to a control group was observed [16].

In the study conducted by Osman et al. [10] 53.7%, 43%, 41.2%, and 23.8% of the woodworkers had nasal congestion, red eyes, itchy eyes, and runny nose, respectively.

Similarly, Bislimovska et al. [43] in a study on 37 parquet manufacturing workers and 37 reference group, reported the prevalence of cough (29.7%) and sputum (16.2%) in the exposed group was significantly higher than that of the reference group.

Neghab et al. [39] reported the risk of developing wheezing, chest tightness, sputum, chronic sputum, coughs, chronic coughs, and shortness of breath in sawmill workers to be 4.75, 2.47, 12. 57, 2.76, 4.94, 2.02, and 4.64 times higher than a reference group, respectively.

Similar findings have been reported by others [18, 35, 42, 44]. Additionally, after controlling for confounders, significant associations remained between exposure to wood dust and increased prevalence of respiratory symptoms.

Similarly, Hessel's study showed that the risk of shortness of breath and wheezing increased by 2.83 and 2.58 times, respectively, as a result of exposure to wood dust [45].

In the present study, 13 exposed workers and 5 reference group subjects had obstructive spirometric patterns, and the observed difference was statistically significant. Similarly, Fante et al. [27], Neghab et al. [39], and Noertjojo et al. [26] reported an increased risk for obstructive pulmonary diseases in woodworkers.

In the present study, the exposed group showed significantly higher levels of MDA and lower levels of SOD and TAC in comparison with the reference group. Additionally, after controlling for confounders, significant associations remained between exposure to wood dust and reduced antioxidant capacity and increased oxidative stress biomarker.

Exposure to wood dust resulted in 0.32, and 1.43 unit decreases in TAC and SOD levels and 1.23 unit increases in MDA in furniture workers in comparison to the reference group.

It is difficult to explain this result because antioxidant capacities may fluctuate due to many changes, such as inflammation, stress, diet, and genetics [46].

Studies in the workers exposed to other agents known to induce oxidative stress revealed inhibition of the antioxidant enzyme, i.e., SOD and glutathione peroxidase (GPx) or depletion of substrate molecules, i.e., glutathione (GSH) in exposed workers in comparison to controls [47, 48].

For example, Tope et al. [49] showed that organophosphates can inhibit the activity of SOD and the concentration of GSH in the farmworkers.

Farahat et al. [6] reported a significant reduction in GPx levels among the wood-exposed carpenters (15.52 ± 5.97 U/mg protein) in comparison with the reference group (28.88 ± 5.39 U/mg protein) and the lowest serum GP_X concentration was observed where the highest concentration of respirable wood dust was reported.

Abbas et al. [30] found a significantly higher MDA and lower GSH and GPx in woodworkers in comparison with the control group. The authors suggested that a decrease in antioxidant capacity could lead to oxidative stress and inflammation, in other words, oxidative stress may be the mechanism by which air pollutants cause airway inflammation and lead to acute cardiorespiratory complications.

In Wultsch's study, woodworkers had a significantly greater mean of MDA when compared with the control group [5]. Gaballah et al. [50] showed that exposure to wood dust was associated with a significant reduction in SOD and GPx levels. These authors suggested that occupational exposure to wood dust is associated with oxidative stress.

There was a significant positive correlation between FVC and FEV1 with SOD and TAC.

Similarly, in 2021, Bhat et al. [29] showed significantly decreased FEV1, FVC, and FEV1/FVC, as well as significantly increased MDA levels in the sawmill workers as compared to a control group. The authors suggested that serum MDA can be used as an indicator of decreased pulmonary function, especially in patients in whom spirometry performing is difficult.

A limitation of this study is that the possible role of marital status and level of education in antioxidant capacities outcomes have not been investigated. Therefore, further studies regarding the role of marital status and level of education would be necessary.

Conclusion

In the present study, there was no statistically significant difference between the two groups in terms of demographic data and the number of cigarette smokers, and smoking intensity. Therefore, these cannot be the cause of the differences in results between the two groups; hence, the changes in pulmonary function parameters and oxidative stress biomarkers can be attributed to exposure to wood dust.

The observed correlation between FVC and FEV1 with SOD and TAC supports the theory that oxidative stress is more likely a mechanism of impaired lung function. According to the results of this study, a diet high in antioxidants (such as fruits and vegetables), as well as appropriate personal protective equipment and workplace safety and training are recommended for the prevention and control of acute and chronic lung diseases in wood workers.

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Conflict of interest F. Kargar-Shouroki, M.R. Dehghan Banadkuki, S. Jambarsang and A. Emami declare that they have no competing interests.

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