

EFFECTS OF DURATION OF EXPOSURE TO WOOD DUST ON PEAK EXPIRATORY FLOW RATE AMONG WORKERS IN SMALL SCALE WOOD INDUSTRIES

SULTAN A. MEO

Department of Physiology
College of Medicine
King Saud University Riyadh
Riyadh, K.S.A.

Abstract

Objectives: Occupational and environmental lung diseases are one of major problems of clinical medicine. Several occupations are associated with adverse health effects, and the lung is one of the parts of the body most vulnerable to airborne hazards. Exposure to gas, fume, and dust can lead to occupational lung diseases. The objective of the study was to assess the effects of wood dust and the duration of exposure on peak expiratory flow rate (PEFR) and additionally, to minimize possible health risks for wood workers by providing them with information about wood dust related hazards. **Methods:** The present study was conducted under the supervision of the Department of Physiology, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia during the year 2002. It was designed as a matched case-control cross-sectional study of spirometry in forty six non-smoking wood workers, aged 20–60 years, who worked without the benefit of wood dust control ventilation or respiratory protective devices. PEFR measurements were performed using an electronic spirometer. **Results:** The present study results demonstrated that in wood workers exposed for longer periods than 8 years, PEFR was significantly reduced as compared with their matched controls. **Conclusion:** Based on the results of the present study, we conclude that PEFR in wood workers is impaired and the stratification of results shows a dose-response effect of years of wood dust exposure on its value.

Key words:

Occupational hazards, Wood dust, Pulmonary function test, Peak expiratory flow rate

INTRODUCTION

Occupational lung diseases are the major work-related illnesses. They are usually induced by extended exposure to irritating or toxic substances that may cause acute or chronic respiratory ailments, however, severe single exposures can also generate chronic lung diseases. Occupational exposures play a role in the onset of several respiratory diseases and the lung function deficit. The lung function impairment is the most common occupational respiratory problem in industrial plants especially in the welding [1] cement [2] and wood industrial sectors [3].

Wood is one of the worldwide most important renewable resources and extends over approximately one-third of the earth's total landmass: about 3.4 million km². It is estimated that there are 12 000 species of trees, each producing a characteristic type of wood. In the plant kingdom, trees belong to the division of spermatophytes and are subdivided into gymnosperms and angiosperms; both types of wood generate wood dust. Wood dust is produced when machines or tools are used to cut or shape wood materials. Industries in which large amount of wood dust is produced

Received: October 18, 2004. Accepted: December 6, 2004.

Address reprint requests to Prof. S.A. Meo, Department of Physiology, College of Medicine, King Saud University, P.O. Box 2925, Riyadh 11461, K.S.A. (e-mail: sultanmeo@hotmail.com or smeo@edu.ksu.sa).

include sawmills, dimension mills, furniture industries, cabinetmaking, and carpentry [4].

Wood dust is a light brown or tan fibrous powder-like substance generated when timber is processed: chipped, sawed, turned, drilled, or polished. Its composition varies considerably according to species of tree and consists mainly of cellulose, polyoses, and lignin, with a large and variable number of substances of lower relative molecular mass, which may significantly affect the properties of the wood. These include polar organic extractives (tannins, flavonoids, quinones and lignans), non-polar organic extractives (fatty acids, resin acids, waxes, alcohols, terpenes, sterols, steryl esters and glycerols), and water-soluble extractives (carbohydrates, alkaloids, proteins and inorganic material). Wood dust is used to prepare charcoal as an absorbent for nitroglycerin, filler in plastics, linoleum, and paperboard. Another commercial use of wood dust is wood compost [5].

Exposure to wood dust has long been associated with a variety of adverse health effects, including dry cough, malaise, chronic bronchitis, shortness of breath, chest pain, conjunctivitis, rhinitis, dermatitis, occupational asthma, allergic alveoli, headache [6], nasal sinus carcinoma [7–9] and lung function deficits. In view of the available information, it is worthwhile to investigate the dose-response effects of wood dust on peak expiratory flow rate (PEFR) in wood workers, working in small scale wood industries without the benefit of self-protective measures and to compare the results with those observed in their matched controls. The two other aspects of this investigation, deserving to be stressed are that all the participants were non-smokers, and that matched case controls were used in the study.

MATERIALS AND METHODS

The present study was conducted under the supervision of the Department of Physiology, College of Medicine, King Saud University, Riyadh, Kingdom of Saudi Arabia during the year 2002.

Study population

Over several days, the principal investigator visited the timber market in the Godhra camp, North Karachi, Paki-

stan, during the summer vacation (July–August 2002) and interviewed approximately 72 wood mill workers. A detailed history of each worker was taken to determine whether they would be included in the study or not on the basis of the exclusion criteria. They were questioned about smoking cigarettes or other tobacco products, chewing tobacco or betel nut products. After initial interviews, 46 apparently healthy male wood workers, mean age, 32.13 ± 1.87 years (mean \pm SEM; range 20–60 years) with mean duration of exposure 5.52 ± 0.60 years (mean \pm SEM; range 1–14 years), were included in the study group and 26 persons were excluded from the study. The wood workers worked for at least 8–10 h/day for six days/week, without using any self-protective measures, whereas the extent of exposure to dust was significant. The control group, selected in a similar way from approximately 70 interviewed persons, comprised finally 46 matched healthy men, mean age, 33.28 ± 1.72 years (mean \pm SEM; range 20–60 years). The control group was composed primarily of shopkeepers and salesmen. All subjects were matched for age, height, and weight.

Exclusion criteria

Subjects with clinical abnormalities of the vertebral column, thoracic cage, neuromuscular diseases, known cases of gross anemia, diabetes mellitus, pulmonary tuberculosis, bronchial asthma, chronic bronchitis, bronchiectasis, emphysema, malignancy, drug addicts, cigarette smokers, tobacco chewers and subjects who had undergone vigorous exercise, abdominal or chest surgery and subjects exposed in any industry other than wood industry were also excluded from the study.

METHODS

Small scale wood industries

The small scale wood industries were located at Godhra camp, New Karachi, Pakistan, near the main road to attract the customers. Small scale wood industries in Pakistan differ from large scale industries in developing countries. They are located in temporary shelters with 6–8 poles supporting a roof that was made up of old wooden

material, and in a few industries of old iron sheets, with their walls also made up of wooden boards. No exposure control equipment, e.g., exhaust ventilation, was fitted in any of the small-scale wood industry; teak was the most frequent type of wood used in these industries.

Spirometry

Peak expiratory flow test was performed on an electronic spirometer (Compact Vitalograph, UK). The test was carried out at a fixed time of the day (9:00–13:00) to minimize the diurnal variation [10]. The apparatus was calibrated daily and operated within the ambient temperature range of 20–25°C. The precise technique of performing various lung function tests in the present study was based on the operation manual of the instrument with special reference to the official statement of the American Thoracic Society of Standardization of Spirometry [11]. After taking a detailed history and anthropometric data, the subjects were informed about the whole maneuver. The subjects were encouraged to practice this maneuver before the peak expiratory flow test. The test was performed on the subject in standing position without using a nose clip. The test was repeated three times after adequate rest, and the results were available in the spirometer.

Statistical analysis

Statistical analysis was conducted using a paired t-test (two-tailed). A level of statistical significance was established at a value of $p < 0.025$. The PEFR data were correlated against the duration of exposure. Linear regression was applied to determine the correlation coefficient: the PEFR data against the duration of exposure to wood dust.

RESULTS

Overall anthropometric data: age, height and weight did not significantly differ between the study and control groups. The PEFR values in the wood dust-exposed workers were lower than in controls, but the significant difference was noted in the workers exposed for more than 8 years (range 10–14 years) (Table 1, Fig. 1). The percent-

Table 1. Peak expiratory flow rate (PEFR) in the study and control groups

Years of exposure	PEFR (liters/min)		Percentage changes	P value
	Study group	Control group		
<4	312.28 ± 23.62 (n = 21)	357.95 ± 24.89 (n = 21)	12.75	NS
4–6	279.71 ± 24.56 (n = 14)	355.57 ± 30.75 (n = 14)	21.33	NS
8	175.00 ± 22.29 (n = 11)	373.45 ± 40.50 (n = 11)	53.13	$p < 0.001$
Total 1–14	269.54 ± 16.10 (n = 46)	360.93 ± 17.23 (n = 46)	25.32	$p < 0.001$

The table gives mean values of PEFR ± standard error; the number of subjects are given in brackets.
NS – not significant.

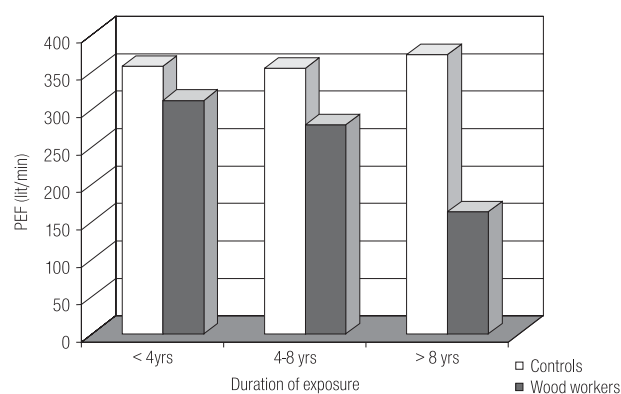


Fig. 1. Graphical representation of the peak expiratory flow rate (PEFR) in the wood workers and matched controls.

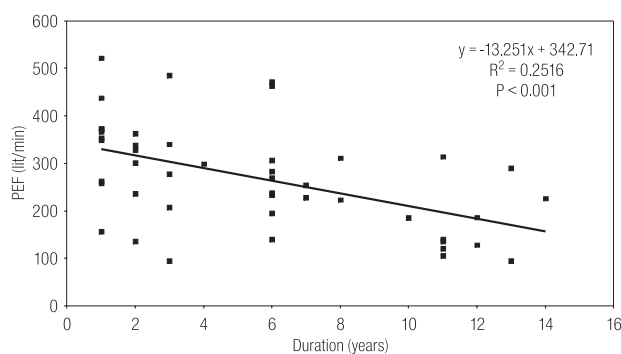


Fig. 2. Regression analysis of peak expiratory flow rate (PEFR) against the duration of exposure of the wood workers, a negative correlation ($r = -0.50$; $p < 0.001$) was found, indicating that the increased duration of wood dust exposure decreased the PEFR values.

age of the PEFR reduction in the wood workers was related to the period of exposure. Salient is more than 50% reduction of PEFR in workers exposed to wood dust for a period of more than 8 years. Regression analysis of the

PEFR values against the duration of exposure revealed the significant negative correlation $r = -0.50$ ($p < 0.001$) (Fig. 2). This data indicated that the increased duration of exposure to wood dust decreased the PEF values.

DISCUSSION

The present study shows that the dose-effect relationship between wood dust exposure and decreased PEF values in non-smoking wood workers is directly proportional to the duration of exposure. This association has not been previously controlled by age, height or weight. The other aspects of this study deserving to be stressed are that all the participants were non-smokers, none of the worker used respiratory protective measures, and that the matched case controls were used in the study.

Milanowski et al. [6] reported lower FVC and FEV₁ values in wood workers compared to controls and also demonstrated a significant pre-shift/post-shift decline in FVC, FEV₁, FEV₁/FVC, and PEF among wood workers. Our results based on PEF correlate with the results observed by these authors [6], however, in the present study we did not observe values based on the pre-shift and post-shift changes, but we found in wood workers the overall decline in the PEF values.

Milanowski et al. [12] showed a more significant reduction of PEF in furniture workers than in controls. A higher drop in the spirometric parameters occurred in younger workers. Mandryk et al. [13] reported that the mean percentage of cross-shift in lung function parameters (FVC, FEV₁, FEV₁/FVC, FEF_{25-75%}) were markedly lower in wood workers compared with controls.

Pontier et al. [7] showed an airway obstructive pattern in spirometry in workers exposed to wood dust.

Liou et al. [3] demonstrated that the pulmonary function parameters, PEF and FEF_{25%}, were significantly lower in the exposed workers than in controls for both smokers and non-smokers and also a declined trend with increasing exposure levels. Similarly, Schlunssen et al. [14] showed a cross-shift decrease in FEV₁ among workers using pine wood.

In addition, Noertjojo et al. [15] reported that sawmill workers had a significantly higher decline in FEV₁ and

FVC values compared with office workers adjusted for age, smoking, and initial lung function. A dose-response relationship was also observed between the level of exposure and the annual decline in FVC.

Dahlqvist et al. [16] showed signs of a mild obstructive impairment with a decreased FEV₁ and the FEV₁ decline was more evident during the working days.

Hedenstierna et al. [17] studied the pulmonary function in wood trimmers and reported that FVC and FEV₁ were reduced in this group of workers.

The present study confirms the findings of other authors and suggests that wood dust adversely affects the peak expiratory flow rate. An analysis of pathophysiological aspects of the drop in the PEF values provides an objective assessment of functional changes relative to environmental and occupational exposures and determines acute or chronic morbid processes [18]. In patients with severe chronic obstructive pulmonary disease (COPD), peak expiratory flow is persistently low and represents collapsed large airways [19]. In view of the pathophysiological aspects and the PEF decrease, our results suggest that the wood dust adversely affects the lung function and this impairment is associated with the duration of exposure to wood dust. The findings are of importance in that they demonstrate the need to reduce exposure and show the magnitude of the effect in a surviving population. Therefore, it is advisable that wood workers, their employers, and health officials work together to adopt technical preventive measures such as ventilated work areas and to use appropriate respiratory protective devices. Pre-employment and periodic medical surveillance tests in this occupational group are strongly recommended. These measures will help to identify susceptible workers so that they could take further preventive measures and appropriate medication.

ACKNOWLEDGMENTS

Thanks are due to Bashir Ahmed and Imran Khan Meo (medical students, Hamdard College of Medicine and Dentistry, Karachi) for their help in the collection of data. We also extend our thanks to Mr. Amir S Marzouk for his

assistance in the data analysis as well as to Mr. Ibraheem and Miss. Nada A, Aziz A Salam for typing the manuscript.

REFERENCES

1. Meo SA, Muhammad A Azeem, Mirza MF Subhan. *Lung function in Pakistani welding workers*. J Occup Environ Med 2003; 45: 1068–73.
2. Meo SA, Azeem MA, Ghori MG, Subhan MMF. *Lung function and surface electromyography of intercostal muscles in cement mill workers*. Int J Occup Med Environ Health 2002; 15(3): 279–87.
3. Liou SH, Cheng SY, Lai FM, Yang JL. *Respiratory symptoms and pulmonary function in mill workers exposed to wood dust*. Am J Ind Med 1996; 30(3): 293–9.
4. IARC. *Wood Dust. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans*. Vol. 62. Lyon, France: International Agency for Research on Cancer; 1995. p. 35–215.
5. Weber SG, Kullman E, Petsonk WG, Jones S, Olenchock W, Sorenson J, et al. *Organic dust exposures from compost handling: case presentation and respiratory exposure assessment*. Am J Ind Med 1993; 24: 365–74.
6. Milanowski J, Gora A, Skorska C, Krysinska-Traczyk E, Mackiewicz B, Sitkowska J, et al. *Work-related symptoms among furniture factory workers in Lublin region (eastern Poland)*. Ann Agric Environ Med 2002; 9(1): 99–103.
7. Bussi M, Gervasio CF, Riontino E, Valente G, Ferrari L, Pira E, et al. *Study of ethmoidal mucosa in a population at occupational high risk of sinonasal adenocarcinoma*. Acta Otolaryngol 2002; 122(2): 197–201.
8. Hildesheim A, Dosemeci M, Chan CC, Chen CJ, Cheng YJ, Hsu MM, et al. *Occupational exposure to wood, formaldehyde, and solvents and risk of nasopharyngeal carcinoma*. Cancer Epidemiol Biomarkers Prev 2001; 10(11): 1145–53.
9. Morales Angulo C, Megia Lopez R, Del Valle Zapico A, Acinas O, Rama J. *Nasal sinus adenocarcinoma in patients exposed to wood dust in the Community of Cantabria, Spain*. Acta Otorrinolaringol Esp 1997; 48(8): 620–4.
10. Glindmeyer HW, Lefante JJ, Jones RN, Rando RJ, Weill H. *Cotton dust and across shift change in FEV₁ as predictors of annual change in FEV₁*. Am J Resp Crit Care Med 1994; 149: 584–90.
11. American Thoracic Society. *Statement on standardization of spirometry*. Am Rev Res Dis 1987; 136: 1286–96.
12. Milanowski J, Krysinska-Traczyk E, Skorska G, Cholewa G, Sitkowska J, Dutkiewicz J, et al. *The effect of wood dust on the respiratory system. Medical examination of furniture factory workers*. Pneumonol Alergol Pol 1996; 64(1): 32–7.
13. Mandryk J, Alwis KU, Hocking AD. *Work-related symptoms and dose-response relationships for personal exposures and pulmonary function among woodworkers*. Am J Ind Med 1999; 35(5): 481–90.
14. Schlunssen V, Schaumburg I, Taudorf E, Mikkelsen AB, Sigsgaard T. *Respiratory symptoms and lung function among Danish woodworkers*. J Occup Environ Med 2002; 44(1): 82–98.
15. Noertjojo HK, Dimich-Ward H, Peelen S, Ditttrick M, Kennedy SM, Chan-Yeung M. *Western red cedar dust exposure and lung function: a dose-response relationship*. Am J Respir Crit Care Med 1996; 154 Pt 1): 968–73.
16. Dahlqvist M, Johard U, Alexandersson R, Bergstrom B, Ekholm U, Eklund A, et al. *Lung function and precipitating antibodies in low exposed wood trimmers in Sweden*. Am J Ind Med 1992; 21(4): 549–59.
17. Hedenstierna G, Alexandersson R, Belin L, Wimander K, Rosen G. *Lung function and rhizopus antibodies in wood trimmers. A cross-sectional and longitudinal study*. Int Arch Occup Environ Health 1986; 58(3):167–77.
18. Quackenboss JJ, Lebowitz MD, Krzyzanowski M. *The normal range of diurnal changes in peak expiratory flow rate*. Am Rev Resp Dis 1991; 143: 323–30.
19. Fallat R, Snow M. *Cardiopulmonary bedside monitoring*. In: Eubanks DH, Bone RC, editors. *Principles and Applications of Cardiorespiratory Care Equipment*. Philadelphia: Mosby; 1994. p. 283–7.