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GENOTOXICITY AND OXIDATIVE STRESS DUE TO EXPOSURE TO WOOD DUST AMONG CARPENTERS

By

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Abstract:

Background: Genetic material is known to be one of the sensitive targets of wood dust. **Objectives:** This study aimed at measurement of the concentration of the respirable fraction of wood dust generated by different wood operating processes. In addition, the study investigated DNA damage among wood dust exposed carpenters and its relation to oxidative stress (OS) induced by exposure to wood dust. **Methods:** Several air samples were collected from 4 carpentry workshops dealing with soft and hard types of unpolished wood and the respirable wood dust concentration was calculated. The study population consisted of an exposed group (n=28) and a matched control group (n=26). Every participant underwent occupational questionnaire, assessment of chromosomal aberrations (CAs) (as an index for DNA damage) and serum glutathione peroxidase enzyme (GPX) as one of the antioxidant enzymes. **Results:** The highest respirable wood dust concentration was that generated by sawing operations. Laboratory investigations revealed statistically significantly higher frequency of CA which was associated with significant decrement in serum GPX among the exposed workers. Increased CA was positively correlated with the duration and the degree of exposure. **Conclusion:** Occupational exposure to wood dust is associated with genotoxicity and oxidative stress. This could be due to lack of protective measures, so there is a need to educate the carpenters about the potential hazard of occupational exposure and the importance of using protective measures.

Key Words: wood dust, DNA damage, chromosomal aberrations, oxidative stress, glutathione peroxidase

Introduction

Wood is one of the world's most important resources and its wide use makes it one of the most commonly seen occupational exposures. The term wood industry covers industries, which predominately carry out mechanized processing of wood (Teschke et al.,1999). Some of the most dusty work tasks are manual sanding in the furniture making, sawing and drilling. However, the dust is often reduced by adding filters or local ventilation on the machines. Despite this effort a part of the wood dust will always escape the filtering systems causing an exposure of the personnel working in the facility (Spee et al., 2007).

Wood dust is a complex mixture. Its chemical composition depends on the species of tree and consists mainly of cellulose, polyoses, and lignin, with a large and variable number of substances with lower relative molecular mass. Cellulose is the major component of both softwood and hardwood. Polyoses (hemicelluloses) are present in larger amounts in hardwood than in softwood while the lignin content of softwood is higher than that of hardwood (Palus et al.,1999).

Exposure to wood dust is associated with a large variety of health effects. The non-carcinogenic ones include irritation and inflammation of the respiratory epithelium causing coughing, wheezing, chronic bronchitis, and asthma. In addition, one of the main concerns is the observation that occupational exposure to wood dust – especially to hardwood dust – is related to considerably elevated risk of cancer (Fransman et al., 2003). Numerous epidemiological studies have consistently demonstrated a strong causal association between exposure to hardwood dust and sino-nasal adenocarcinoma (Demers et al., 1995). Accordingly, the International Agency for Research on Cancer (IARC) in 1995 have classified wood dust as group 1 carcinogen (carcinogenic to humans).

Genetic effects of wood dust have important implications for the induction of cancer. Wood dust particles are believed to induce genotoxicity in two ways; primary and secondary. Primary genotoxicity is proposed to be through generation of oxidants by the dust particles themselves while the secondary mechanism is linked to excessive and persistent formation of reactive oxygen radical species (ROS) from inflammatory cells during particle induced inflammation (Määttä et al., 2006).

So far, most of the Egyptian studies concerned with occupational health effects of wood dust, have focused on respiratory

adverse effects. In our study, we assessed the concentrations of the respirable fraction of the wood dust during different mechanical processing of unpolished wood in some of carpentry workshops. Then we investigated the genotoxic effects of wood dust exposure through assessment of chromosomal aberrations (CA). Glutathione peroxidase (GPX), one of the free radical scavenging enzymes, was measured to give information on oxidative stress that may be a potential indicator of DNA damage and carcinogenic mechanism.

Subjects & Methods

Study Population:

The study was carried out on 33 male carpenters working in carpentry workshops located in El-Basateen district, Cairo. After applying selection criteria (working in carpentry workshops for at least the preceding 2 years) and exclusion of workers who were taking regular medications or were exposed to radiation during the last 12 months before sampling, 5 carpenters were excluded from the beginning of the study. Accordingly, there were only 28 workers who participated in the study. The workers were exposed to a mixture of soft and hard unpolished wood dust.

The control group consisted of 26 men who were employed in 2 primary public schools and had no history of exposure to wood dust. It was assured that the exposed workers and the controls were matched in age, smoking habits and socioeconomic standard.

Both exposed and control groups were subdivided according to smoking habit into smokers (n= 17, 17, respectively) and non smokers (n= 11, 9, respectively).

Methods

A) Measurements of wood dust levels

The measurement of wood dust level was carried out in 4 carpentry workshops located in the El-Basateen district. The 4 workshops were working in sawing, sanding, and drilling of unpolished soft and hard wood using mechanical equipment. The surface areas of the workshops ranged from (72- 100 m^2). All the workshops were moderately ventilated by extract system except the 1^{st} workshop which was naturally ventilated.

Unfortunately all the machine operators refused to use the personal air sampler so we had to fix the personal air sampler on the machine itself in a certain place so as it was as much as possible in the breathing zone of the machine operators thus the dust collected was representative of his actual exposure.

Air samples were collected at 4 sites 1. Sawing machines 2. sanding machines 3. Drilling machines 4. General indoor concentrations. Each air sample was continued for 8 hours and sampling was repeated 10 times to make a total number of air samples collected inside the 4 carpentry workshops, 160 air samples.

The dust was collected on pre-weighed Whatman GF A (glass fiber) filters. It was mounted on an open filter holder. The air was aspirated by a vacuum pump (air flow was 2L/ min) and the reading of the gas meter was recorded before and after the sampling period. After sampling, the filters were dried over silica gel for at least 24 hours before being re-weighed. According to Wright's method (1954), we could measure the total dust concentration and the respirable dust (<5 μm) concentration expressed in mg/m3.

Laboratory investigations

1] Study of chromosomal aberration (CA) in PBL

The CA analysis was conducted following a standard protocol. A total of 1 ml aliquot of whole blood was cultured in F-10 medium supplemented with 20% fetal bovine serum, 0.5 ml PHA, 5000 IU/ ml penicillin and 1000 IU/ml streptomycin. Each culture was incubated at 37 ˚C for 72 h. Metaphases were obtained by adding 0.2 μg/ml colchicine to the cultures 3 h before harvesting. The cells were collected by centrifugation, re-suspended in a pre-warmed hypotonic solution (0.075 M KCI) for 15 min at 37˚C and fixed in acetic acid: methanol (1:3 v/v). Chromosome preparations were stained with 3.3% Giemsa. The slides were analyzed at 1000 magnification using a light microscope and 100 metaphases cells were screened per each individual. Cells with 46 chromosomes were scored for CA. The analysis of CA included chromatid and chromosome breaks, chromatid deletions, chromatid rings and dicentrics chromosomes.

2] Glutathione peroxidase (GPX)

The activity of GPx in serum was measured spectrophotometrically as described by Paglia and Valentine (32). The enzyme reaction was initiated by the addition of H2O2 to the reaction medium and the rate of NADPH oxidation was followed at 340 nm. The amount of enzyme that oxidizes 1 mol NADPH per minute was considered to be one unit.

Statistical Analysis

Results were evaluated for each group. Data were compared using Student's t-test. Qualitative data were compared using chi square test. ANOVA test was used to compare exposed subgroups and control

subjects. Pearson correlation test was used to test the correlation between different variables among the exposed groups. The statistical significance was defined as P value <0.05. Computer based statistical package for social sciences (SPSS) for windows 9.1 program was used.

Results

The mean values of the measured respirable wood dust concentrations at different locations in the 4 carpentry workshops were summarized in table (1). Sawing was found to generate the highest concentrations of respirable wood dust in the 4 workshops.

The study population of this study consisted of 28 male carpenters exposed to unpolished soft and hard wood dust. Their age range was (17-50 years) with A mean age of 33.7±9.97 years showing no statistically significant difference when compared with the control group (range= 16-50 years, mean = 31.76±10.85). The mean duration of working years of the exposed personnel was 15.5±8.9 years. The percentage of smokers among the exposed workers was 60.7% (n=17) with no statistical difference of significance when compared with that of the control subjects (65%) (n=17) (P>0.05).

Analysis of the percentage of chromosomal aberrations (CA%) among the study participants showed marked significant elevations of total CA % among the wood dust exposed personnel compared to their controls (P<0.05 table 2). Subtypes of CA namely, chromatid breaks, chromosome break, dicentric chromosomes, ring chromosomes and deletions were found to be significantly higher among the exposed group compared to their controls. Moreover, significant decrease in the serum level of glutathione peroxidase (GPX) enzyme was found among the exposed group versus their controls (P<0.01 table 2).

The effect of the degree of exposure to wood dust on genotoxicity and oxidative stress was shown in table 3 where the highest CA and lowest GPX were detected in the first workshop which had higher concentrations of wood dust.

Cigarette smoking was found to have additional genotoxic effect as shown in table (4). There was higher frequency of CA and lower GPX among exposed smokers compared to exposed non-smokers although the differences were statistically insignificant (P>0.05). However, both exposed smokers and non-smokers had higher statistically significant CA and markedly lower GPX compared to their controls (P<0.001).

On correlating different parameters, the study revealed statistically significant positive correlation between duration of exposure and CA % (r=0.42 P<0.05). On the other hand, GPX was significantly correlated in a negative pattern with both duration of exposure and CA% (P<0.05 table 5).

Table (1): Mean ±SD of concentrations of 8 hours TWA of respirable wood dust (mg/ m3) measured in different locations in 4 carpentry workshops.

	Sawing	Sanding	Drilling	Indoor
1st workshop	8.15±0.83**	3.16±0.88*	2.08±0.28*	4.46± 0.66
2nd workshop	4.87±2.12*	3.05±1.34*	1.83±0.84*	3.25±1.42
3rd workshop	3.89±1.18*	1.7±0.59*	0.44±0.19	2.04±0.56
4th workshop	3.32±1.47*	1.48±0.47*	0.92±0.42	1.9±0.87

*exceeds the permissible exposure limits stated by NIOSH (1 mg/m^3)

** exceeds the permissible exposure limits stated by OSHA and Egyptian Environmental law # 4 (1994) (5 mg/m^3)

Table (2): Mean ±SD of chromosomal aberrations (CA) % and glutathione peroxidase (GPX) (U/mg protein) in wood dust exposed and the control groups.

	Exposed N=28	Control N=26	p
Total CA%	23.78±9.87	9.61±3.98	<0.01*
CB	5.53±3.68	5.03±1.90	<0.01*
DB	7.10±3.34	4.07±1.71	<0.01*
Del.	5.28±2.20	1.42±0.80	<0.01*
Dic.	4.64±1.76	1.38±1.02	<0.01*
Ring	1.21±1.20	0.30±0.4	<0.01*
GPX	15.52±5.97	28.88±5.39	<0.01*

CB (chromatid break), DB (chromosome break), Dic (dicentric chromosomes), del (chromosomal deletion).

*statistically significant P<0.01

Table (3): Mean ± SD of frequency of CA % and GPX (U/mg protein) concentration in wood dust exposed workers (with respect to ambient concentrations in different workshops) and control subjects.

	1st workshop N=8	2nd workshop N=6	3rd workshop N=7	4th workshop N=7	Control N=26	P
Wood dust (mg/m^3)	4.46± 0.66	3.25±1.42	2.04±0.56	1.9±0.87	------	-----
CA %	30.5±5.50[b]	28.33±7.52[b]	24.57±8.7	11.42±4.5[a]	9.61±3.98[a]	<0.05*
GPX	12.97±6.24[c]	12.36±4.08[c]	16.32±5.98	19.72±4.07	28.88±5.39	n.s

*statistically significant

no statistical difference of significance between values having the same letter.

Table (4): Mean ± SD of frequency of CA % and GPX (U/mg protein) concentration with respect to smoking in both wood dust exposed workers and control subjects.

		Exposed N=28		Control N=26	P
CA%					
Smoking	No		No		
Yes	17	25.29±10.19[1]	17	10.11±4.42[2]	<0.001*
No	11	21.45±9.34[1]	9	8.66±2.95[2]	<0.001*
GPX					
Smoking	No		No		
Yes	17	14.23±6.09[1]	17	27.50±5.89[2]	<0.001*
No	11	17.12±5.09[1]	9	30.5±5.47[2]	<0.001*

*statistically significant

Values with the same numbers are statistically insignificant (P>0.05)

Table (5): correlation coefficient between duration of exposure, CA % and GPX (U/mg protein) concentration in wood dust exposed workers.

	GPX	CA%
Duration (years)	r= -0.57 P<0.01	r= 0.42 P<0.01
GPX (U/mg protein)	----------	r= -0.69 P<0.01

Discussion

Wood dust is created when machines or tools are used to cut or shape wood materials. Exposure to wood dust is defined by various methods; total dust, inhalable dust, and respirable dust. Total dust is collected by dust collector and is considered less relevant with respect to health effects, because it is without size specifications. The inhalable dust is defined as those particles captured by samplers regardless to orientation with an aerodynamic diameter between 0 and 100 μm. Particles 0.5 μm to 5 μm (respirable particles) are deposited in the lower airways (ACGIH,1997). It is worth noting that respirable wood dust ranges from 6% to 75% of the total wood aerosol (Demer et al., 1997)

In 1977, the National Institute of Occupational Safety and Health (NIOSH, 1977) recommended that wood dust (soft, hard, and western red cedar) be considered a potential occupational carcinogen and that exposure be limited to 1 mg/m^3 as a TWA exposure up to a 10-hour workday during a 40-hour workweek. Higher permissible exposure limit (PEL) was established by OSHA and the Egyptian Law for Environment # 4 (1994) (5 mg/m^3). However, on reviewing the scientific literature, exposure to wood dust at levels below 5 mg/m3, including 1 mg/m^3 and lower, was found to cause sino-nasal as well as pulmonary symptoms. In fact, symptoms in the upper and lower respiratory tracts have been observed in some studies at exposure levels as low as 0.5 mg/m^3 and lower (Husgafvel-Pursiainen,2004). Similarly, Elavarasi et al., (2002) detected significant increase in the genetic damage in the form sister chromatid exchanges (SCEs) and micronuclei (MN) among wood dust exposed workers at environmental levels as low as 0.3mg/m^3 . Consequently, it seems evident that to protect woodworkers, a lower limit value is needed.

In our study, air samplers were placed to be as near as possible to the respiratory zone of the machine operators. Measurements revealed generation of respirable fraction of wood dust that was in excess of the recommended TWA levels by NIOSH (1 mg/m3) particularly in workshop 1 which also exceeded the (PEL) stated by OSHA (5mg/m3) and the Egyptian Law for the environment # 4 (1994) (5mg/m3) (Table 1). The highest generation of dust was during sawing procedures compared to sanding and drilling. This goes in accordance with Hursthouse et al. (2004) who reported high exposures of 2.5– 45 mg /m^3 when sawing softwood. In another study conducted by Spee et al., (2007), indoor sawing of wood sheets yielded the highest exposure (25.8–34.9 mg /m3 during 2 h).

In our study, general indoor concentrations of respirable wood dust were within the permissible limits stated by OSHA but exceeded NIOSH's in all workshops (table 1). This can be attributed to sweeping of the fallen dust and different activities in the workshops. However, dust of other types than wood dust could have been present on the floor which makes it unlikely that the respirable fraction consisted entirely of wood dust (Spee et al., 2007).

The current work revealed differences in respirable wood dust concentrations between the 4 carpentry workshops where the 1st workshop showed the highest concentrations. This can be explained by the poor ventilation in that workshop. Some authors emphasize the importance of good housekeeping and ventilation for reduction of exposure (Cecala et al., 2000 and Vermeulen et al., 2000). It was reported that, local exhaust ventilation reduces the exposure to wood dust with a factor 3 to 10 (Thrope and Brown, 1994).

The genotoxicity related to an occupational exposure can be evaluated using different genetic endpoints, e.g., DNA damage, chromosomal aberrations (CA), and micronuclei (Celik and Akbas ¸ 2005). In our study, CA analysis was utilized to evaluate the extent of genome damage in carpenters. CA are particularly dangerous to the cell because the physical discontinuity of the chromosome may cause loss of genetic information and even cell death if a housekeeping gene is involved (Pasquini et al., 2001).

The present study revealed statistically significant increase in the frequencies of CA in exposed subjects when compared to their controls (23.78 % and 9.61%, respectively, $P<0.01$) (table 2). Our results are greatly supported by a comparable

study carried out by Rekhadevi et al., (2009) among a group of Indian carpentry workers where frequency of CAs was about 2 times that in their controls. The same study revealed significant increase in comet tail length through using comet assay technique. Moreover, in the context of the genotoxic effects of wood dust, detectable DNA damage was reported by Bornholdt et al.,(2007) when they incubated human epithelial cell line A549 with extracts of wood dust. In 2006, Celik and Kanik reported increased apoptosis as judged by an increase in the frequency of karyolysis in exfoliated epithelial buccal cells among wood dust exposed personnel. DNA damage was presented in a study made by Palus et al., (1999) in the form of significant increase in single strand break and DNA repair in peripheral lymphocytes. However, the chemicals used to confer resistance and durability to wood such as formaldehyde and arsenic compounds may also be a cause for the genetic effect (Bahia et al., 2005).

Smoking, degree and duration of exposure had a considerable effect on DNA damage in the current study. As shown in table (4), there was higher frequency of CAs among both exposed and control smokers in comparison to non- smokers in both groups although the difference was not statistically significant ($P>0.05$). Some studies pointed to the very complex interaction between smoking and occupational exposure to genotoxic agents as cigarette smoking itself is a well- known risk factor for several types of cancer (Palus et al., 1998). Consequently, cigarette smoking is a known confounding factor that may influence the frequency of cytogenetic damage, such as chromosomal aberration, SCE, and MN formation in humans (Celik et al., 2005). This may explain the presence of relatively higher frequency of CAs in workers of the 3rd workshop although they were exposed to lower ambient concentrations of respirable wood dust (2.04±1.56 mg/m3), as they were all smokers.

A significant effect of degree and duration of exposure to wood dust on DNA damage was evident by the higher frequency of CAs detected among workers employed in the workshops with higher ambient concentrations (table 3) and the positive correlation between duration of exposure and CA frequency ($r= 0.42$ $P<0.05$) (table 5).

The mechanism behind the DNA-damaging effect of wood dust may possibly be the production of reactive oxygen species (ROS) as detected by Long et al.,(2004). Recently, dusts from different tree species

widely used in wood industry were found to have significant stimulating effect on the production of ROS (PylkkÃnen et al., 2009). In spite of numerous biodefense systems, excessive production of these free radicals overwhelms the cells' intrinsic antioxidant defenses which leads to development of a state of oxidative stress (OS). This imbalance between body's defense mechanisms and free radical generation promotes cellular injury and tissue damage particularly proteins, lipids and DNA (Karago¨zler et al., 2002).

In the present study, serum glutathione peroxidase(GPX) which is a selenoenzyme responsible for elimination of ROS, was significantly lowered in exposed carpentry workers in comparison to their controls (15.52±5.97, 28.88±5.39, respectively P<0.01) (table 2). Similar low levels were seen in wood workers exposed to bass wood (Wu et al.,2002). Moreover, studies in other groups of workers exposed to agents known to induce ROS generation, revealed decreased levels of antioxidant enzyme levels in exposed workers in comparison to controls (Tope et al., 2006).

Decrements in serum GPX was remarkable in the 1st workshop where the highest concentration of respirable wood dust was demonstrated (table 3) which supports the claims of induction of ROS by the wood dust exposure with depletion of substrate molecules.

The intimate relation between OS and DNA damage was evident by the negative correlation between CA frequency and the GPX level (r= -0.69 P<0.01) (table 5) which is greatly supported by a similar association between diminished superoxide dismutase and GPX serum levels and increased frequency of CA, micronuclei and abnormal comet assay (Rekhadevi et al., 2009).

The negative effect of either passive or active smoking on the level of serum GPX was discussed in many studies. Rajpurkar et al.,(2000) reported an increase in the level of oxidants with a simultaneous decrease in the level of antioxidant enzymes in association with smoking. This goes in accordance with the slight decrement in GPX level in exposed and control smokers (table 4; P>0.05). Lack of statistically significant differences between smoker and non smokers exposed persons may be due to the fact that the non-smokers were actually passive smokers.

In conclusion, occupational exposure to wood dust was associated with increased frequency of CAs. The positive genotoxicity may be due to lack of protective measures as none of the exposed

personnel used facemasks or gloves. So there is a need to educate the carpenters about the potential hazard of occupational exposure and the importance of using protective measures. It is suggested that biomonitoring of genotoxic effects in wood industry with furthermore comprehensive controlled studies are needed to support our observations.

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