Occupational Exposure to Airborne Wood Dust during Carpentry Work and Risk of Ischemic Heart Disease: A Comparative Cross-Sectional Study

Reem A Abbas[1*], Hisham S Roshdy[2] and Samar M Sharaf[3]

[1]Community, Occupational and Environmental Medicine Department, Faculty of Medicine, Zagazig University, Egypt.
[2] Cardiology Department, Faculty of Medicine, Zagazig University, Egypt.
[3] Clinical Pathology Department, Faculty of Medicine, Zagazig University, Egypt.
*reemabbasabbas@yahoo.com

Abstract: Occupational exposure to airborne dust is possibly associated with increased cardiovascular morbidity and mortality. The exact mechanism linking the inhalation of airborne particulates to ischemic heart disease is not fully understood yet. This study aimed to investigate the role and the possible mechanism of chronic occupational exposure to airborne wood dust particulates emitted during carpentry work in increasing the risk of ischemic heart disease among a sample of daytime carpenters. Sixty seven male daytime carpenters who met the inclusion criteria and sixty nine non-exposed daytime administrative and health care workers in Ismailia city were recruited in this comparative cross-sectional study. All participants were subjected to a structured questionnaire, clinical examination, and relevant investigations that included fasting blood glucose level, lipid profile, and glycated haemoglobin (HbA1c); inflammatory markers (C-reactive protein, absolute neutrophils count, interleukin-6, and fibrinogen); and selected oxidative stress markers (blood antioxidant enzymes such as glutathione and glutathione peroxidase) and a lipid peroxidation product such as serum malondialdehyde). Serial measurements for respirable wood dust at different worksite areas throughout one working week were conducted and the average of readings in mg/m^3 was calculated. The average concentrations of respirable wood dust particulates at different wood processing machines (sawing machines, planers, and grinding/sanding machines) were (6.39, 44.19, and 131.28 mg/m^3, respectively). The studied markers of systemic inflammation were significantly elevated among carpenters compared with the control group. Serum malondialdehyde was significantly elevated in carpenters compared with the control group. Also, significant reduction of the studied blood antioxidant enzymes in the studied carpenters was observed compared with the control group. It could be concluded that this group of carpenters may be at increased risk for ischemic heart disease and oxidative stress may have an important role in the pro-inflammatory effects of ambient airborne wood particles. Consequently, exposure to airborne wood dust at carpentry workshops should be kept below the permissible exposure limit. Carpenters should be provided with suitable personal protective equipment. Prospective cohort studies are recommended to establish casual relationship between wood dust exposure and the increased risk of ischemic heart disease in carpenters.

[Reem A Abbas, Hisham S Roshdy and Samar M Sharaf. **Occupational Exposure to Airborne Wood Dust during Carpentry Work and Risk of Ischemic Heart Disease: A Comparative Cross-Sectional Study.** *J Am Sci* 2013;9(12):660-668]. (ISSN: 1545-1003). http://www.jofamericanscience.org. 86

Keywords: Airborne wood dust; Ischemic heart disease; Fibrinogen; Particulate air pollution; Inflammatory markers; Oxidative stress.

1. Introduction:

Several studies have found that exposure to ambient air pollution, particularly particulate matter is associated with increased cardiovascular morbidity and mortality (Hoek *et al*., 2001; Peters *et al*., 2001; Pope *et al*., 2002; Pope & Dockery, 2006 and Mills *et al*., 2008). A theory was launched in 1995 by Seaton and coworkers that urban particulate air pollution may provoke alveolar inflammation, with release of mediators capable of increasing blood coagulability and cause cardiovascular deaths in susceptible people e.g. elderly and those with pre-existing cardiovascular diseases or other illnesses such as diabetes mellitus (Sjogren, 1997; Delfino *et al*., 2008 and Raaschou-Nielsen *et al*., 2012).

Till now, understanding of the underlying pathogenic mechanisms remains limited, but it has been proposed that risk of cardiovascular events, including myocardial infarction due to inhalation of small particles is associated with increased blood levels of inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α) and its receptors, adhesion molecules, and acute phase proteins such as C-reactive protein (CRP) and fibrinogen. An increased number of neutrophils in the peripheral blood is another expression of this response and this is also an indicator of ischemic heart disease (IHD) (Sjogren, 1997; Seaton *et al*., 1999; Pai *et al*., 2004 and Mills *et al*., 2008).

Alveolar inflammation induced by inhaled particles may either directly or via oxidative stress leads to systemic inflammation with increased levels of blood coagulability, progression of atherosclerosis, and destabilization or even rupture of vulnerable plaques, resulting in acute ischemic events (Brook *et al*., 2004 and Brook *et al*., 2010).

A substantial body of evidence supports a role for oxidative stress in the pro-inflammatory effects of ambient airborne particles. Reactive oxidant species arise not only from the redox potential of the pollutants themselves but also from the activation of alveolar epithelial cells or resident macrophages and the recruitment and activation of circulating neutrophils (Mills *et al*., 2008).

Occupationally exposed groups often have high exposure to particulate air pollution, such as wood dust, silica dust, asbestos or welding fumes, but whether such dust exposure increases the risk for ischemic heart disease is not clear yet (Tore´n *et al*., 2007 and Delfino *et al*., 2008). Consequently, the specific aim of this study was to investigate the role and the possible mechanism of chronic occupational exposure to airborne wood dust particulates emitted during carpentry work in increasing the risk of ischemic heart disease (IHD) among a sample of daytime carpenters.

2. Methods:

Study design and setting:

This analytical cross-sectional study was conducted from January 2013 to May 2013, at a large carpentry workshop of a private furniture factory and at Suez Canal Authority Hospital in Ismailia city, Egypt.

Study population:

Sixty seven out of 137 male daytime carpenters were recruited in this study. The exclusion criteria were; work duration < 6 months, smoking, obesity (body mass index $\geq$ 30), hypertension, history of ischemic heart disease, diabetes mellitus, cancer, abnormal lipid profile, and surgical procedures (three days before the study). Moreover, workers who had acute infections or chronic inflammatory disorders, or reported taking treatments or diets with high antioxidant levels, lipid lowering drugs, and aspirin intake in the preceding 6 weeks were also excluded from the study.

Age, sex, residence, and socioeconomic status matched non-exposed daytime administrative and health care workers at Suez Canal Authority Hospital in Ismailia city (n = 69) were recruited as a control group with the same exclusion criteria.

Participants were invited by the investigators to an interview where they were asked to give their consent after explaining the purpose and the steps of the study. Questionnaires were administered, clinical examination was carried out, and blood samples were collected by the investigators after clarifying the confidentiality of the data. This step took part in a 30-min interview using a semi-structured interview schedule.

Ethical issues:

Proposal acceptance was obtained from the Research Ethics Committee in Zagazig Faculty of Medicine. Permission was obtained from the owner of the furniture factory and from the manger of Suez Canal Authority Hospital. Moreover, informed consent was obtained from all participants.

Tools of the study:

I) Questionnaire: A structured questionnaire was constructed based on those of other relevant studies (Sjogren, 1997 and Delfino *et al*., 2008). The questionnaire was composed of two main parts:

Part one: included socio-demographic and occupational data such as age, residence, marital status, education, duration in the job, working hours per week, and second or another job.

Part two: included relevant past medical and surgical history as well as family history of ischemic heart disease.

II) Clinical examination:

a) Body mass index (BMI): Measuring the height in meters and weight in kilograms was done to calculate the body mass index (BMI); where the formula for BMI is weight in kilograms divided by height in meters squared (CDC, 2007).

b) Arterial blood pressure: Blood pressure was measured according to the European Society of Hypertension and the European Society of Cardiology guidelines for office or clinic arterial blood pressure measurement. Hypertension was defined as either systolic blood pressure of 140 mm Hg or higher and/or diastolic blood pressure of 90 mm Hg or higher (Giuseppe *et al*., 2003).

III) Laboratory investigations:

Blood sampling: Eight ml venous blood samples were taken from all participants after 12 hours fasting period and were divided into 5 portions: a) 1 ml of whole blood, that was collected into tubes containing Ethylene diamine tetraacetic acid (EDTA), for glycated haemoglobin (HbA1c) and absolute neutrophils count; b) 1 ml of whole blood, that was collected into tubes containing fluoride, for fasting blood glucose; c) 1 ml of whole blood, that was collected into tubes containing heparin, for glutathione (GSH) and glutathione peroxidase; d) 1.8 ml blood was added to sodium citrate for plasma fibrinogen; and e) serum were separated immediately from the remaining part of the samples and stored at −20 °C until analysis for the remaining tests (lipid profile, CRP, IL-6, and serum malondialdehyde).

1) Biochemical measurements:

Fasting blood glucose levels, lipid profile and HbA1c were estimated using Cobas 311 autoanalyzer from Roche Diagnostics, GmbH, Mannheim, USA. Low density lipoprotein (LDL-c) was calculated using the Friedewald formula (Frieldewald *et al*., 1972), providing that triglyceride (TG) not more than 400 mg/dl, if TG was over 400 mg/dl patient was excluded from the study. The reference intervals for serum total cholesterol (TC), LDL-c, and TG were less than 200 mg/dL, 160 mg/dL and 150 mg/dL, respectively, and for high density lipoprotein (HDL-c) was more than 39 mg/dL (Željko *et al*., 2011). The reference interval for fasting blood glucose was from 70 to 110 mg/dL. For assessing HbA1c, total Hb and HbA1c concentrations were determined after haemolysis of the anticoagulated whole blood specimen. Total Hb was measured colorimetrically. HbA1c was determined immunoturbidimetrically. The ratio of both concentrations yields the final percent HbA1c result [HbA1c (%)] (Little *et al*., 1992). The reference value for HbA1c% was less than 5.0%.

2) Markers of systemic inflammation:

2-a) Serum C- reactive protein (CRP) levels were measured by means of immunonephelometric assay (BN ProSpec® System protein analyzer, Siemens Diagnostic Healthcare Inc). The reference value for serum CRP was less than 6 mg/L.

2-b) Plasma fibrinogen level was determined using FibroTEK kit R2 Diagnostics, South Bend, Indiana, USA. It was done using the Clauss technique, which involves measuring the clotting time of dilute plasma after the addition of thrombin. At high thrombin concentrations (>30 NIH units/mL) and low fibrinogen concentrations, the fibrinogen level is inversely proportional to the thrombin clotting time plotted on log - log graph paper (Clauss, 1957). The reference interval for plasma fibrinogen was from 200 to 400 mg/dL.

2-c) Serum interleukin-6 (IL-6) was measured using Sandwich Enzyme Linked Immunosorbent Assay (ELISA) method according to manufacturer's instructions (Human interleukin-6 ELISA, Biosource Europe S.A, Belgium).

2-d) Absolute Neutrophils count was done on KX-21N hematology analyser from Sysmex Corporation, Kobe, Japan. The reference range was form 2000 to 8000 cell/ µL.

3) Oxidative stress markers:

3-a) Serum malondialdehyde (MDA) as a product of lipid peroxidation was measured by colorimetric method using kit provided by BioVision Incorporated. Milpitas Boulevard, Milpitas, USA (Ohkawa *et al*., 1979). The MDA in the sample is reacted with Thiobarbituric Acid (TBA) to generate the MDA-TBA adduct. The MDA-TBA adduct can be easily quantified colorimetrically (λ = 532 nm).

3-b) Glutathione (GSH) activity was determined in fresh heparinized blood by biodiagnostic assay kit (BioChain Institute, Inc. Eureka Drive. Newark, CA USA) (Beutler *et al*., 1963). The improved 5,5'-dithiobis (2-nitrobenzoic acid (DTNB) method combines deproteination and detection into one reagent. DTNB reacts with reduced glutathione to form a yellow product. The optical density, measured at 412 nm, is directly proportional to glutathione concentration in the sample.

3-c) Glutathione peroxidase levels were measured using a commercially available kit (Ransel; Randox Laboratories Ltd., UK) according to the method of Paglia and Valentine, where GPX catalyses the oxidation of glutathione by cumene hydroperoxide. In the presence of glutathione reductase and NADPH, the oxidized glutathione is immediately converted to its reduced form with a concomitant oxidation of NADPH to NADP+. The decrease in absorbance at 340 nm was expressed as units per liter of the hemolysate (Paglia & Valentine, 1967).

IV) Environmental assessment: Fifteen repeated measurements at three different wood processing machines (sawing machines, planers, and grinding/sanding machines) for respirable airborne wood dust throughout one working week were taken during work shift. This was performed using a portable Dust Track Aerosol Monitor 8520. It gives the average of readings in mg/m^3 for the respirable particles (with a cut size of 10 µm) suspended in air within 10 seconds and at flow rate adjusted at 1.7 L/min. The instrument was calibrated to measure wood dust concentration using track pro-software through setting a new calibration factor with a value of 1.45 for wood dust.

Data management:

Data were coded and statistically analyzed using SPSS version 20 (IBM, 2010). Comparison between group means was done using student's t-test; comparison between categorical variables was done by chi-squared test. Correlation coefficient (r) was used for testing the association between two continuous variables. The significance level was considered at *P*-value < 0.05.

3. Results:

Relevant characteristics and family history of participants

The mean of carpenters' age was (41.2±7.2 years) and that of the control group was (43.7±9.3 years). All participants of the study live in Ismailia city. Most of participants were married (82.1% for carpenters and 88.4% for the control group). Those with basic education constituted (47.8% for carpenters and 55.1% for the control group) and those who had

secondary or technical school education constituted (52.2% for carpenters and 44.9% for the control group). The average duration of work for carpenters was (12.7±2.1 years) and for the control group was (13.1±3.7 years). The average working hours per week was (46.7±7.7 hours/week for carpenters and 45.8±6.9 hours/week for the control group). Most of participants did not have second or another job (71.6% for carpenters and 81.2% for the control group). There were no statistical significant differences between both groups regarding the studied demographic and occupational variables as well as family history of coronary heart disease ($P > 0.05$) (Table 1).

Clinical findings of participants

The average body mass index for carpenters was (27.7±4.1 Kg/m^2) and for the control group was (28.2±3.3 Kg/m^2) with no statistical significant difference ($P > 0.05$). The average systolic blood pressure for carpenters was (130.7±7.3 mmHg) and for the control group was (132.3±4.1 mmHg) with no statistical significant difference ($P > 0.05$). Moreover, the average diastolic blood pressure for carpenters was (85.9±4.1 mmHg) and for the control group was (86.1±2.3 mmHg) with no statistical significant difference ($P > 0.05$) (Table 2).

Results of laboratory investigations of participants

The average fasting blood glucose level for carpenters was (88.9±10.3 mg/dL) and for the control group was (86.7±8.7 mg/dL) with no statistical significant difference ($P > 0.05$). The average value of HbA1c % for carpenters was (4.2±0.5) and for the control group was (4.1±0.1) with no statistical significant difference ($P > 0.05$). The mean values of total cholesterol, low density lipoprotein, triglyceride, and high density lipoprotein for the carpenters and the control group were within the normal reference range with no statistical significant differences. Markers of systemic inflammation (C-reactive protein, absolute neutrophils count, interleukin-6, and fibrinogen) were significantly elevated among carpenters compared with the control group. A significant elevation of serum malondialdehyde was observed among carpenters compared with the control group ($P<0.001$). Also, significant reduction of blood antioxidant enzymes, glutathione and glutathione peroxidase in carpenters was observed compared with the control group ($P<0.001$) (Table 3).

On studying the association between some systemic inflammatory markers and oxidative stress markers of the studied carpenters, the results showed significant positive association between serum malondialdehyde and the studied inflammatory markers (CRP, IL- 6, and plasma fibrinogen) [r (P); 0.71 (0.02), 0.79 (0.006), and 0.85 (0.002)]. While, significant negative association was found between blood glutathione and blood glutathione peroxidase and the studied inflammatory markers (Table 4).

Environmental assessment

The average concentrations of respirable wood dust at different wood processing machines (sawing machines, planers, and grinding/sanding machines) were (6.39, 44.19, and 131.28 mg/m^3, respectively) (Table 5).

Table (1): Relevant characteristics and family history of participants.

Demographic and occupational variables and relevant family history of participants	*Carpenters (N = 67)*	*Control group (N = 69)*	*Test of significance*	*P-value*
	N (%)	*N (%)*		
Age (years) ($\overline{X} \pm SD$) *Min – Max*	(41.2 ± 7.2) 30 – 53	(43.7 ± 9.3) 32 - 58	t-test (1.75)	0.08
Marital status				
Married	55 (82.1%)	61 (88.4%)	χ^2 (1.08)	0.29
Single/divorced/widowed	12 (17.9%)	8 (11.6%)		
Education				
Basic education	32 (47.8%)	38 (55.1%)	χ^2 (0.73)	0.39
Secondary/Technical school	35 (52.2%)	31 (44.9%)		
Duration in job (Years) ($\overline{X} \pm SD$) *Min – Max*	(12.7 ± 2.1) 2 – 15	(13.1 ± 3.7) 4 – 18	t-test (0.77)	0.44
Working hours/week ($\overline{X} \pm SD$)	(46.7± 7.7)	(45.8 ± 6.9)	t-test (0.72)	0.47
Second/another job				
Yes	19 (28.4%)	13 (18.8%)	χ^2 (1.71)	0.19
No	48 (71.6%)	56 (81.2%)		
Family history of coronary heart disease				
Yes	11 (16.4%)	9 (13.1%)	χ^2 (0.31)	0.58
No	56 (83.6%)	60 (86.9%)		

Table (2): Clinical examination findings of participants.

Clinical findings	*Carpenters (N = 67)*	*Control group (N = 69)*	*t-test*	*P-value*
	($\overline{X} \pm SD$)	($\overline{X} \pm SD$)		
Body mass index (BMI) (Kg/m^2)	27.7 ± 4.1	28.2 ± 3.3	0.79	0.43
Min - Max	26 - 28	26 - 29		
Arterial blood pressure (mmHg)				
Systolic blood pressure	130.7 ± 7.3	132.3 ± 4.1	1.58	0.12
Min - Max	120 - 135	120 - 138		
Diastolic blood pressure	85.9 ± 4.1	86.1 ± 2.3	0.35	0.73
Min - Max	80 - 86	80 - 89		

Table (3): Results of laboratory investigations of participants.

Laboratory investigation results	*Carpenters (N = 67)*	*Control group (N = 69)*	*t-test*	*P-value*
	$\overline{X} \pm SD$	$\overline{X} \pm SD$		
Biochemical measurements				
Fasting blood glucose (mg/dL)	88.9 ± 10.3	86.7 ± 8.7	1.34	0.18
HbA1c (%)	4.2 ± 0.5	4.1 ± 0.1	1.63	0.11
Lipid profile (mg/dL)				
Total cholesterol (TC)	190.2 ± 8.6	192.3 ± 7.5	1.52	0.13
Low density lipoprotein (LDL-c)	139.1 ± 7.6	141.2 ± 7.2	1.66	0.10
Triglyceride (TG)	135.8 ± 5.2	137.2 ± 4.1	1.75	0.08
High density lipoprotein (HDL-c)	45.9 ± 7.4	47.1 ± 6.9	0.98	0.32
Markers of systemic inflammation				
C-Reactive protein (CRP) (mg/L)	3.6 ± 0.8	0.9 ± 0.1	27.8	0.000
Absolute Neutrophils count (cell/µL)	6200.7 ± 550.8	4500.9 ± 550.3	18.0	0.000
Interleukin – 6 (IL-6) (Pg/mL)	18.1 ± 5.3	4.5 ± 1.1	20.9	0.000
Plasma fibrinogen (mg/dL)	465.6 ± 60.3	260.1 ± 35.2	24.4	0.000
Oxidative stress markers				
Serum malondialdehyde (nmol/mL)	4.7 ± 0.9	1.9 ± 0.3	24.5	0.000
Blood glutathione (µM /L)	53.5 ± 7.9	76.9 ± 4.7	21.1	0.000
Blood glutathione peroxidase (U/L)	146.8 ± 26.2	210.8 ± 33.1	12.5	0.000

Table (4): Association between some systemic inflammatory markers and oxidative stress markers of the studied carpenters.

systemic inflammatory markers in carpenters	**Serum malondialdehyde (nmol/mL)**	**Blood glutathione (µM /L)**	**Blood glutathione peroxidase (U/L)**
C-Reactive protein (mg/L)	r (*P*) 0.71 (0.02)	r (*P*) -0.73 (0.02)	r (*P*) -0.65 (0.04)
Interleukin - 6 (IL-6) (Pg/mL)	r (*P*) 0.79 (0.006)	r (*P*) -0.69 (0.03)	r (*P*) -0.68 (0.03)
Plasma fibrinogen (mg/dL)	r (*P*) 0.85 (0.002)	r (*P*) -0.86 (0.002)	r (*P*) -0.75 (0.01)

Table (5): The average concentration of respirable wood dust at the carpentry workshop.

Average concentration of respirable wood dust at different wood processing machines (mg/m^3)		
Sawing machines	**Planers**	**Grinding/Sanding machines**
6.39	44.19	131.28

4. Discussion

Evidence accumulated over more than 50 years of epidemiologic and clinical research has established a strong association between airborne particulates exposure during several environmental pollution episodes and cardio-respiratory morbidity and mortality. However, whether occupational exposure increases this risk is not clear yet (Pope & Dockery, 2006; Rückerl *et al*., 2007 and Feng & Yang, 2012). Previous epidemiological studies found that occupational exposure to wood dust was associated with an increased risk for ischemic heart

disease [RR (95% CI): 1.7 (1.0 to 3.0) and 1.12 (1.04 to 1.20), respectively] (Hammar *et al*., 1992 and Torén *et al*., 2007).

The primary strength of this study is that exposure in the carpenters' microenvironment (carpentry workshop) was measured where workers spend most of their time, and we conducted serial measurements of airborne wood dust and the average concentrations was calculated. The results of this study revealed that the average concentrations of respirable wood dust particulates at different wood processing machines (sawing machines, planers, and grinding/sanding machines) were (6.39, 44.19, and 131.28 mg/m^3, respectively) which exceed the permissible exposure limit (PEL) for respirable wood dust [8 - hour Time Weighted Average (8 h TWA) = 5 mg/m^3] (OSHA, 2012).

During the past decade there was a growing interest about the idea that atherosclerosis is an inflammatory disease, and about the finding that serum levels of markers of inflammation can be used to predict the risk of cardiovascular events. Elevated concentrations of acute-phase reactants, such as C-reactive protein, soluble intercellular adhesion molecule-1, TNF-α, and fibrinogen, were found in patients with acute coronary syndromes and were used as predictors of future risk in apparently healthy individuals. The inflammatory cytokine interleukin-6 (IL-6) is a powerful inducer of the hepatic acute-phase response, and it has been proposed to be a central mediator in the pathogenesis of coronary heart disease through a combination of autocrine, paracrine, and endocrine mechanisms (Targher *et al*., 2001; Delfino *et al*., 2005; Mills *et al.,* 2005 and Mills *et al.,* 2007). It has been shown that interleukin-6 is released from the bronchial mucosa and stimulates the production of fibrinogen in the liver which induces an increase in blood viscosity and promotes thrombus formation (Hilt *et al*., 2002 and Rückerl *et al*., 2007). Consequently, previous studies revealed significant association between IL-6 and the risk of myocardial infarction in apparently healthy individuals (Ridker *et al*., 2000 and Targher *et al*., 2001). Moreover, occupational exposure to airborne wood dust among sawyers was associated with an increased risk of IHD; where increased concentrations of IL-6 in some biological fluids were revealed. These concentrations might hypothetically stimulate the liver cells to secrete more fibrinogen (Sjogren, 1997).

Fibrinogen, an acute-phase protein, plays a crucial role in the coagulation cascade. It has emerged as an important risk factor for ischemic heart disease (IHD). In fact, fibrinogen may possibly be the major risk factor, exceeding the "contributions" of homocysteine, cholesterol and other lipids in the pathogenesis of coronary heart disease (Delfino *et al*., 2008). However, studies regarding its association with air pollution are inconclusive. In a recent study, fibrinogen has been shown to increase in association with high levels of ambient particles such as in air pollution episodes (Rückerl *et al*., 2007). Also, a previous study found that exposure to wood dust was significantly associated with increased plasma concentrations of fibrinogen (Langley *et al*., 1991).

C-reactive protein (CRP), a well-known biomarker of systemic inflammation, has been one of the first acute phase reactants to be examined in association with air pollution in several studies. Increased concentrations have been shown during several known air pollution episodes all over the world (Rückerl *et al*., 2007). Increased concentrations of CRP are known to predict cardiovascular events in healthy subjects (Danesh *et al*., 2000). Neutrophils count also is considered as an independent predictor of cardiovascular morbidity (Anneli *et al*., 2010).

In the present study blood markers that indicate an inflammatory response (C-reactive protein, absolute neutrophils count, interleukin-6, and fibrinogen) were measured in a group of carpenters exposed to wood dust and a control group not occupationally exposed to wood dust. The studied markers of systemic inflammation were significantly elevated among carpenters compared with the control group.

Alternatively, some previous epidemiological studies conducted on workers exposed to dust have shown a decreased risk for IHD when these groups have been compared with national death rates. This decreased risk is well known as the healthy worker effect and can be explained by the lack of comparability between the particular group of workers and the general population which will include sick and disabled people (Sjogren, 1997 and Mills *et al*., 2008).

While epidemiological evidence strongly supports the association between particulate air pollution and cardiopulmonary effects, the mechanism is not fully understood. It has been suggested that inhaled particles may lead to systemic inflammatory responses through the release of IL-6, TNF-α or histamine and oxidative stress within the lungs and/or systemically. For the last several years, there has been mounting experimental evidence that ultrafine particles (UFPs; tentatively defined as PM < 0.1 μm in diameter) can induce the greatest amount of oxidative stress and inflammation per unit of PM mass (Cho *et al*., 2005 and Ntziachristos *et al*., 2007).

A novel finding in the present study is the significant elevation of serum malondialdehyde that was observed among the studied carpenters compared

with the control group. Also, significant reduction of blood antioxidant enzymes (glutathione and glutathione peroxidase) in the studied carpenters was observed compared with the control group. Moreover, there was significant positive association between serum malondialdehyde and the studied inflammatory markers among the studied carpenters; while, significant negative association was found between blood glutathione and blood glutathione peroxidase and the studied inflammatory markers.

It was hypothesized that diminished antioxidant response could lead to oxidative stress and then inflammation, with increased susceptibility to adverse cardiovascular events. This is important because oxidative stress may be a mechanism whereby air pollutants induce inflammation in the airways and systemically, leading to acute adverse cardio-respiratory responses (Delfino *et al*., 2008).

However, evidence from other experimental studies indicated that exposure to air pollution is associated with rapid changes in autonomic nervous system balance, favoring sympathetic nervous system activation and parasympathetic withdrawal. Other lines of evidence also suggest that inhaled nano-particulates can rapidly cross the alveolar–capillary barrier and directly affect the cardiovascular system (Brook *et al*., 2010).

It is well established that fibrinogen is positively correlated with several of the traditional risk factors for IHD such as age, cigarette smoking, obesity, diabetes, hypertension, chronic inflammations, plasma low density lipoprotein, lower social classes, and living in urban areas (Sjogren, 1997 and Tore´n *et al*., 2007). So, one of the main strengths of the present study is that the influence of these important confounders was controlled.

Moreover, the influence of important confounding factors involved in inflammation and infection that known to play a pro-atherogenic role and consequently are considered as potent risk factors for acute ischemic syndromes (Rückerl *et al.*, 2007) was also eliminated.

Also, confounding factors such as lipid-lowering drugs intake and health-related events (acute infection or surgery) which have been shown to reduce CRP through inhibition of its hepatic synthesis (Rückerl *et al*., 2007) have been controlled in this study.

However, there are several limitations. Although the data were collected at the worksites, we were not able to correct for exposures in other environments. The source of exposure to particles could be environmental as well as occupational (Mills *et al*., 2007). Moreover, it has also been shown that manual workers have higher concentrations of fibrinogen than non-manual workers (Sjogren, 1997 and Folsom *et al*., 2006).

5. Conclusions and recommendations:

The results of the present study are consistent with the hypothesis that exposure to particulate air pollution increases the risk of ischemic heart disease, not only when the exposure is environmental but also when it is occupational. It was concluded from this study that the studied blood inflammatory markers can be used as indicators for the increased risk of cardiovascular events among carpenters exposed to wood dust. Oxidative stress may be an important mechanism whereby airborne wood dust induces inflammation in the airways and systemically, leading to adverse cardio-respiratory responses.

So, the exposure to airborne wood dust particulates at carpentry workshops should be kept as low as possible. The association between different sizes of airborne wood dust and biomarkers of inflammation and oxidative stress should be studied. Exposure assessments of UFPs which have a high pro-inflammatory potential resulting from their ability to transloctae systemically from pulmonary sites and their potential to promote oxidative stress near the workers require special attention in future epidemiologic research.

Prospective cohort studies are recommended to establish casual relationship between wood dust exposure and the increased risk of nonfatal myocardial infarction or death from cardiovascular causes in carpenters.

It is likely that biomarkers are not all equivalent in their sensitivity to pollutant-induced responses and that the exposures are not all equivalent in their representation of causal pollutant components. In this sense, the exploratory nature of this research can help provide data for the selection of the best biomarkers (e.g., C-reactive protein, absolute neutrophils count, interleukin-6, and fibrinogen) to the selected exposures (airborne wood dust) for future research.

Providing antioxidants and diet high in antioxidant-rich foods (such as fruits and vegetables) for carpenters may help protect the adverse cardiovascular effects associated with chronic exposure to wood dust.

Suitable personal protective measures should be available for carpenters. In addition sufficient training of workers for safe work practice is crucial.

Competing interests:

The authors declare that there are no competing interests. Moreover, this research was financed by the authors of the study.

Acknowledgements:

We would like to thank the owner of the furniture factory and the manger of Suez Canal Authority Hospital for facilitating our work. Also, we would like to thank all participants for making this study possible.

Corresponding author:
Reem A Abbas
Community, Occupational and Environmental Medicine Department, Faculty of Medicine, Zagazig University, Egypt.
Mailing address: reemabbasabbas@yahoo.com

References:

1. Anneli F., Anpeli E., Ambrosio O., *et al.* (2010): Neutrophils count for the identification of postmenopausal hypertensive women at increased cardiovascular risk. *Obstet Gynecol*; 115 (4):695-703.
2. Beutler E., Duron O. and Kelly B.M. (1963): Improved method for the determination of blood glutathione. *J Lab Clin Med*; 61:882-888.
3. Brook R.D., Franklin B., Cascio W., *et al.* (2004): Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*; 109:2655-2671.
4. Brook R.D., Rajagopalan S., Pope C.A., *et al.* (2010): Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. *Circulation*; 121: 2331-2378.
5. Centers for Disease Control and Prevention (CDC) (2007): Body mass index [http://www.cdc.gov/nccdphp/dnpa/bmi/adult_bmi/about_adult_bmi.htm].
6. Cho A.K., Sioutas C., Schmitz D.A., *et al.* (2005): Redox activity of airborne particulate matter at different sites in the Los Angeles Basin. *Environ Res*; 99:40-47.
7. Clauss A. (1957): Gerinnungsphysiologische Schnellmethode zurBestimmung des Fibrinogens. *Acta Haemat*; 17: 237-246.
8. Danesh J., Whincup P., Walker M., *et al.* (2000): Low grade inflammation and coronary heart disease: prospective study and updated metaanalyses. *BMJ*; 321:199-204.
9. Delfino R.J., Sioutas C. and Malik S. (2005): Potential role of ultrafine particles in associations between airborne particle mass and cardiovascular health. *Environmental Health Perspectives*; 113 (8): 934-946.
10. Delfino R.J., Staimer N., Tjoa T., *et al.* (2008): Circulating biomarkers of inflammation, antioxidant activity, and platelet activation are associated with primary combustion aerosols in subjects with coronary artery disease. *Environ Health Perspect*; 116 (71): 898-906.
11. Feng J. and Yang W. (2012): Effects of particulate air pollution on cardiovascular health: A population health risk assessment; *PLoS ONE*; 7(3): e33385 [http://www.plosone.org/article/info:doi/10.1371/journal.pone.0033385].
12. Folsom A.R., Chambless L.E., Ballantyne C.M., et al. *et al.* (2006): An assessment of incremental coronary risk prediction using C-reactive protein and other novel risk markers: the atherosclerosis risk in communities study. *Arch Intern Med*; 166: 1368-1373.
13. Friedewald W.T., Levy R.I. and Fredrickson D.S. (1972): Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem*; 18:499-502.
14. Giuseppe M., Guy D., Anna D., *et al.* (2003): European Society of Hypertension -European Society of Cardiology guidelines for the management of arterial hypertension. *Journal of Hypertension*; 21:1011-1053.
15. Hammar N., Alfredsson L., Smedberg M., *et al.* (1992): Large differences in the incidence of myocardial infarction among occupational groups. *Scand J Work Environ Health*; 18:178-185.
16. Hilt B., Qvenild T., Holme J., *et al.* (2002): Increase in interleukin-6 and fibrinogen after exposure to dust in tunnel construction workers. *Occup Environ Med*; 59:9-12.
17. Hoek G., Brunekreef B., Fischer P., *et al.* (2001): The association between air pollution and heart failure, arrhythmia, embolism, thrombosis, and other cardiovascular causes of death in a time series study. *Epidemiology*; 12 (3):355-357.
18. IBM (2010): IBM SPSS Statistics 19 Core System Users' Guide. SPSS Inc, Chicago [http://www.IBM-SPSS-Statistics-User_s-Guide.pdf].
19. Langley P.G., Hughes R.D., Rolando N., *et al.* (1991): Increased elastase-al-antitrypsin complex in fulminant hepatic failure: relationship to bacterial infection and activation of coagulation. *Clin Chim Acta*; 200:211-220.
20. Little R., Wiedmeyer H., England J., *et al.* (1992): Inter-laboratory standardization of measurements of glycohemoglobins. *Clin Chem*; 38:2472-2478.
21. Mills N.L., Tornqvist H., Robinson S.D., *et al.* (2005): Diesel exhaust inhalation causes

vascular dysfunction and impaired endogenous fibrinolysis. *Circulation*; 112(25):3930-3936.
22. Mills N.L., Törnqvist H., Robinson S.D., *et al.* (2007): Air pollution and atherothrombosis. *Inhal Toxicol*; 19 (suppl 1):81-89.
23. Mills N.L., Robinson S.D., Fokkens P.H.B., *et al.* (2008): Exposure to concentrated ambient particles does not affect vascular function in patients with coronary heart disease. *Environmental Health Perspectives*; 116 (6):709-715.
24. Ntziachristos L., Froines J.R., Cho A.K., *et al.* (2007): Relationship between redox activity and chemical speciation of size-fractionated particulate matter. *Part Fibre Toxicol*; 4:5 [http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1899517/].
25. Occupational Safety and Health Administration (OSHA) (2012): Wood dust. United States Department of Labor [http://www.osha.gov/sltc/wooddust/].
26. Ohkawa H., Ohishi N. and Yagi K. (1979): Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. *Anal Biochem*; 95351-95358.
27. Paglia D. and Valentine W. (1967): Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *J Lab Clin Med*; 70:158-169.
28. Pai J.K., Pischon T., Ma J., *et al.* (2004): Inflammatory markers and the risk of coronary heart disease in men and women. *N Eng J Med*; 351: 2599-2610.
29. Peters A., Dockery D.W., Muller J.E., *et al.* (2001): Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*; 103:2810-2815.
30. Pope C.A., Burnett R.T., Thun M.J., *et al.* (2002): Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution. *J Am Med Assoc*; 287:1132-1141.
31. Pope C.A. and Dockery D.W. (2006): Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manag Assoc*; 56:709-742.
32. Raaschou-Nielsen O., Andersen Z., Jensen S., *et al.* (2012): Traffic air pollution and mortality from cardiovascular disease and all causes: a Danish cohort study. *Environmental Health*; 11:60 [http://www.ehjournal.net/content/11/1/60].
33. Ridker P.M., Rifai N., Stampfer M.J., *et al.* (2000): Plasma concentration of interleukin-6 and the risk of future myocardial infarction among apparently healthy men. *Circulation*; 101:1767-1772.
34. Rückerl R., Greven S. and Ljungman P. (2007): Air Pollution and inflammation (Interleukin-6, C-Reactive Protein, Fibrinogen) in myocardial infarction survivors. *Environmental Health Perspectives*; 115(7): 1072-1080.
35. Seaton A., Soutar A., Crawford V., *et al.* (1999): Particulate air pollution and the blood. *Thorax*; 54:1027-1032.
36. Sjogren B. (1997): Occupational exposure to dust: inflammation and ischemic heart disease. *Occupational and Environmental Medicine*; 54:466-469.
37. Targher G., Zenari L., Bertolini L., *et al.* (2001): Elevated levels of interleukin-6 in young adults with type 1 diabetes without clinical evidence of microvascular and macrovascular complications. *Diabetes Care*; 24 (5): 956-957.
38. Torén K., Bergdahl I.A., Nilsson T., *et al.* (2007): Occupational exposure to particulate air pollution and mortality due to ischemic heart disease and cerebro-vascular disease. *Occup Environ Med*; 64(8):515-519.
39. Željko R., Alberico L., Guy D., *et al.* (2011): ESC/EAS Guidelines for the management of dyslipidemias. *European Heart Journal*; 32:1769-1818.

12/11/2013