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Effect of Wood Smoke on Lipid Peroxidation, some antioxidants, Vitamins A, C, E and Some Trace Element Levels in Serum of Iraqi Workers

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Abstract:
Background*: the pollution of wood charcoal smoke may causes many health disorders, among these lipid peroxidation and disturbance of antioxidant levels, in addition to the poisoning with some toxic elements, which in turn affect the level of vital elements in the human body. In this work, the exposure effect of wood smoke on 100 Iraqi workers was studied. The aim of this study is to investigate the effect of charcoal smoke on lipid peroxidation marker malondialdehyde (MDA) , vitamins (A, C, E), albumin, hemoglobin and (Hg, Pb, Mg, Zn, Se) levels in sera of the workers.*
Methods*: One hundred workers were subdivided into two groups according to their exposure period to wood charcoal smoke. Group A includes 50 male who were exposed for 1-10 years, group B includes 50 male who were exposed for 11-25 years and group C which includes 40 healthy male as control group. Levels of MDA, vitamins of A, C, E, albumin, hemoglobin and the elements Pb, Hg, Mg, Zn and Se were estimated in sera of all the studied groups.*
Results*: the results showed that the (MDA) level was elevated in the serum of workers, the highest level was found to be 39.88 ±7.5nmol/dl for workers with longest exposure period compared to the control group, which has a value of 16.5 ±6.1nmol/dl. Vitamins of A, C and E, hemoglobin and albumin levels were found to be lower than normal values in all workers compared to the control group. Levels of Pb, Hg and Mg were increased with the increasing of the exposure period; in contrast, the levels of Zn and Se were decreased in all workers compared to the control group.*
Conclusion*: it concluded that the levels of antioxidants, albumin, hemoglobin and essential elements were disordered due to the oxidative stress and contamination with poisoning elements that occurred as a result to exposure to the wood smoke.*
Keywards: *wood smoke, antioxidant, lipid peroxidation, vitamin A,C,E trace elements.*

I. Introduction

Wood is consided the main source to produce charcoal. Charcoal production process is accompanied by the removal of water and volatile constituents and leaving a light black material composed of carbon and ash [1]. It was reported that wood smoke is capable to produced predominantly OH radicals. In the presence of H_2O_2, wood smoke caused DNA damage. The major species responsible for DNA damage is OH radical [2].

Several early studies were referred to that the emission of particles from residential wood burning and their impact on human health has received much attention lately. They were confirmed that there is adverse health impacts in form of more respiratory symptoms and diseases associated with wood smoke exposure [3-5]. A reverse correlation between lipid peroxidation product (malondialdehyde) and both vitamin C and vitamin E was recorded in many studies[6-7].

It was found that the levels of A, E and C vitamins in rat suffering from stress were decreased in increasing of free radicals amount [8]. In another study which was conducted for estimation the level of A, C and E vitamins as antioxidants versus malondialdehyde in blood sera of patients with urolithiasis, the results of this study confirmed that a reverse correlation between levels of these antioxidant vitamins and malondialdehyde [9].

Bioaccumulation ability of mercury in animals and human beings increase the risk factor of this element as an environmental pollutant. The most poisonous substances of mercury in our environment are organomercury and inorganic mercury compounds. Therefore, mercury toxicity depends on the type of mercury compound and its oxidation state [10]. The toxic metals pose serious human health problems, such as brain damage and mental retardation. Lead effects are generally restricted to especially contaminated areas [11].

The absorption of lead by the body may be occurs through inhalation and ingestion. The absorbed lead either spread to the soft tissues or distributed to the skeleton.

The biological roles of trace elements in protection of health and progress of optimal physiological functions increase the interest to estimate their levels in many clinical studies [12-15], for example, magnesium is essential mineral for life. It requires for over 300 enzymes for their catalytic action, thus it presents in every cell type in every organism [16].

The aim of this study is to investigate the effect of wood charcoal smoke on lipid peroxidation(MDA) , vitamins (A, C, E), albumin, hemoglobin and (Hg, Pb, Mg, Zn, Se) levels in blood sera of workers and the effect of the exposure period.

II. Subjects and Methods

Subjects

This study was conducted from December 2014to April 2015; blood samples were collected from 100 Iraqi workers who worked at some wood charcoal preparation positions in Bob al-Sham area, Baghdad – Iraq. Workers were divided into two groups according to their exposure period to wood smoke. Group A included 50 male who were exposed for 1-10 years, group B included 50 male who were exposed for 11-25 years, and the control group included 40 apparently healthy men.

Methods

a. Determination of trace element levels in blood serum

The elements Mg, Zn, Se. Hg and Pb were estimated by the atomic absorption method. The process was conducted according to the following conditions [15].

Element	**Wave length (nm)**	**Air flow (L/min)**	**Acetylene flow (L/min)**	**Burner height (mm)**
Hg	353.65	10	2.4	4
Pb	271	10	2.3	4
Mg	285.21	10	2.6	4
Zn	231.86	10	2.4	4
Se	196	10	2.3	4

b. Determination the level of malondialdehyde in blood serum:

Malondialdehyde is a product of oxidation of fat, the measurement of this substance gives the impression of the level of peroxidation. The interaction between thiobarbituric acid and malondialdehyde gives a colored product. Level of malondialdehyde was estimated by measure the absorbance of the colored product at a wavelength of 532 nm. [17].

c- Determination of vitamin (A, E, C) in serum:

The level of vitamin E was measured using the modified method of De Leenheer. Standard solutions were prepared, mobile phase consists of 99% methanol and 1% water, the measurement of vitamin E level was conducted at wavelength of 278 nm using ODS C-18 column. Level of vitamin A was estimated using the same mobile phase and column type used in the estimation of vitamin E, and at wavelength of 330 nm. Vitamin C level was determined by using a solution of sodium ethylenediaminetetraacetate with water (pH 6), wavelength 254 nm, using ODS C-18 column [18].

d- Determination of serum albumin :

This method depends on the quantitative binding of albumin to the indicator 3,3',5,5'-tetrabromo-m cresol sulphonephthalein (bromocresol green, BCG). The complex of albumin –BCG was maximally absorbed at 578 nm, the absorbance being directly proportional to the concentration of albumin in the sample [15]. The concentration of albumin in the sample was calculated by applying the following formula:-

$$\text{Albumin concentration (g/100 ml)} = \frac{\text{A sample}}{\text{A standard}} \text{ x concentration of standrd}$$

e- Measurement of hemoglobin (Hb)

The amount of hemoglobin was measured by cyanmethemoglobin method**.** In this method, blood is mixed with Drabkin's solution, a solution that contains ferricyanide and cyanide. The ferricyanide oxidizes the iron in the hemoglobin; thereby changing hemoglobin to methemoglobin, then methemoglobin unites with the cyanide to form cyanmethemoglobin, which produces a color that can be measured in a colorimeter at wavelength of 540 nm. The color relates to the concentration of hemoglobin in the blood sample [19]. The concentration of hemoglobin in the sample was calculated by applying the following formula:-

$$\text{Hemoglobin concentration} = \frac{\text{absorbance of blood sample}}{\text{absorbance of standard}} \text{ x standard concentration}$$

Statistical analysis

The data of all parameters were expressed as mean ±SD. Statistical evaluation of data was performed using SPSS program version 17. $P < 0.05$ was taken to indicate the significance values [20].

III. Results and Discussion

The results of mercury and lead levels (mean±SD) in blood sera of worker groups (A and B groups) and the control group (group C) were recorded in table (1). The results showed that levels of mercury and lead are significantly higher in serum of both worker groups compared to the control group. The highest levels of both these toxic elements were found in group B due to the increasing of exposure period to the wood smoke.

Table (1): mercury and lead levels (mean ± SD) in blood sera of worker and control groups.

Groups	No.	Mercury level (mean ± SD) µg/dl	lead level (mean ± SD) µg/dl	T
C	40	0.128 ±0.413	20.1 ±7.6	-
A	50	1.023 ±0.786	117.22 ±9.1	P<0.001
B	50	2.169 ±0.0895	182.24 ±21.3	P<0.001

A = group of workers (exposure period: 1-10 years).
B = group of workers (exposure period: 11-25 years).
C = control group.

Lead is a poisonous metal that can damage nervous connections (especially in young children) and cause blood and brain disorders. Long term exposure to lead or its salts can cause nephropathy and colic-like abdominal pains. The effects of lead are the same whether it enters the body through breathing or swallowing. Lead can affect almost every organ and system in the body. The main target for lead toxicity is the nervous system, both in adults and children. Long-term exposure of adults can result in decreased performance of the nervous system functions. It may also cause weakness in fingers, wrists, or ankles. Lead exposure also causes small increases in blood pressure, particularly in middle-aged and older people and can cause anemia. Exposure to high lead levels can severely damage the brain and kidneys in adults or children and ultimately cause death. In pregnant women, high levels of exposure to lead may cause miscarriage. Chronic, high-level exposure in men can damage the organs responsible for sperm production [21-24].

The lipid peroxidation in term of malondialdehyde (MDA) was recorded in table (2). The results showed that the level of MDA was raised in serum of workers compared to control group.

Table (2): MDA (mean ± SD) in blood sera of workers and control groups

Groups	No.	MDA level (mean ± SD) n mol/dl	T
C	40	16.5 ±6.1	-
A	50	25.7 ±3.7	P<0.05
B	50	39.88 ±7.5	P<0.001

A,B and C as in table 1

The reason of this result may be attributed to the increase of reactive oxygen species (ROS) which lead to damage the cell membrane and increase the level of lipid peroxidation. This result is in agreement with other studies [25]. Table (3) shows the levels of A, E and C vitamins in serum of workers and control groups. The values of these vitamins in both A and B groups were less than their values in control group. These results can be attributed to the effect of the generated oxidants due to the exposure periods of wood smoke. Vitamin A is belonged to the carotenoid compounds that known to be effective antioxidants toward ROS [26]. Moreover, vitamin A has many biological functions including participating in the process of vision by interact with the opsin protein forming visual purple which is also necessary in the formation of mucous carbohydrates which constitute mucosa material to secrete layer epithelium that provides protection for respiratory, urinary, and reproductive channels. It also recommended for taking food rich with vitamin (A) to protect of ocular conjunctiva from damage as well as to raise the level of HDLC in the blood [18, 27].

The lack in vitamin E level as shown in (table 3) can be interpreted within stress concept that results from environmental effects. Vitamin E is an antioxidant fat soluble vitamin; it works to protect the fats in cell membranes from oxidation damage. Vitamin E affects the cellular immune response, where it works to strengthen the immune status. Vitamin E stimulates T cells. Helper Lymphocytes, which works to transform the production of antibodies IgM type to IgG type. Our results in this study are consistent with the results of many researchers [28].

The decreasing in vitamin C level in workers groups in comparison to control group as shown in table 3 was attributed to the same reasons of both A and E vitamins, especially vitamin C is another form of antioxidant micronutrients. It is a water soluble vitamin and it protects cells against the degenerative processes of cataract and age-related macular degeneration (AMD). Moreover, vitamin C intake was shown to be positively associated with bone mineral status at various sites in adolescent girls and young women and with markers of bone formation in adolescent girls [29].

Table (3): levels of vitamins A, E and C (mean ± SD) in blood sera of workers and control groups

Groups	No.	Vitamin A level (mean ± SD) µg/dl	Vitamin E level (mean ± SD) mg/dl	Vitamin C level (mean ± SD) mg/dl	T
C	40	61.4 ±8.2	1.18 ±0.11	1.62 ±0.88	
A	50	30.6 ±10.6	0.760 ±0.73	0.69 ±0.38	P<0.05
B	50	24.6 ±12.5	0.531 ±0.51	0.49 ±0.21	P<0.001*

It was known that the hemoglobin range value for men is 13.6-17.7 mg/dl. The results shown in table (4) referred that the hemoglobin level in worker groups was less than its level in control group. This result may be attributed to the oxidative stress which forms ROS. The excess in ROS relative to GSH and glutathione peroxidase lead to formation of H_2O_2, which lead to damage the red blood cell membranes and result in decrease of hemoglobin level [30]. Table (4) shows that the level of albumin in workers groups are significantly low compared to the control group because the albumin is one of antioxidant that works by connecting the copper and iron with albumin to clearance of free radicals. The lack of albumin leads to decrease the clearance of free radicals, and therefore, increases the lipid peroxidation. In another hand, the low level of albumin in the blood serum leads to many disorders, the most important ones are pressure and blood viscosity because the albumin form about (60%) of the proportion of total protein in the blood. Thus, the decline in the level of albumin leads to disturbances in important functions of the body and this agrees with the results of study [31].

Table (4): Hemoglobin and albumin level (mean ± SD) in blood of workers and control groups

Groups	No.	Hb level (mean ± SD) g/dl	albumin level (mean ± SD) g/dl	T
C	40	16.03 ±0.30	4.34 ±0.12	
A	50	13.25 ±0.38	3.21 ±0.21	P<0.05
B	50	11.49 ±0.46	2.12 ±0.47	P<0.001

The results of Mg, Se and Zn levels in blood sera of the workers and control groups were recorded as shown in table (5), values of Mg were significantly higher in both worker groups compared with control group.. , the sequence of Mg level in the studied groups may be attributed to the exposure period of wood charcoal smoke which leads to oxidative damage of red blood cell membrane. Especially if we take into consideration that the magnesium level inside the cell fluids be higher than its level in extracellular fluids [32]. The results also showed reduction of selenium level in blood sera of worker groups compared to control group. The decrease in Se level cause a disturbance in glutathione peroxidase function, which leads to more oxidative damage in cell membranes, it should be noted that all the Se levels are within normal ranges and be decreasing according to duration of exposure to wood smoke [33].

Similar to Se, Zn level in both worker groups was decreased in comparison to control group. This result can be attributed to the oxidative stress which results from exposure to wood smoke for a long period; especially this trace element has important biological roles such as growth and cell division, cofactor for many metalloenzymes as in alcohol dehydrogenase and DNA polymerization [34].

Table (5): levels of Mg, Se and Zn (mean ± SD) in blood sera of workers and control groups

Groups	No.	Mg level (mean ± SD) µg/dl	Se level (mean ± SD) ng /dl	Zn level (mean ± SD) µg/dl	T
C	40	1528.70 ±100.3	91 ±0.14	61.72 ±5.21	-
A	50	2282.18 ±561 .6	78 ±0.78	40.28 ±7.80	P<0.001
B	50	2984 .62 ±389.9	69± 0.58	29.68 ±5.90	P<0.001

IV. Conclusion

According to the results of this study, we can conclude the following

1- The lipid peroxidation increased with increasing of the exposure period to the wood charcoal smoke.
2- The disorders of antioxidants (vitamins of A, E and C), albumin, hemoglobin and essential element levels (Zn, Se and Mg) are confirmed the effects of the oxidative stress and poisoning elements (Hg and Pb) that occurred due to exposure of wood smoke.
3- Moreover, the damage of many cell membranes in the body and the nerve ending in the brain can be attributed to the increasing of reactive oxygen species.

References

[1]. Wondwosen Bogale. **2009**. Preparation of Charcoal Using Agricultural Wastes. Ethip. J. Educ. & Sci. Vol. 5 (1) p.80-94.

[2]. Stephen S. Leonard, Suwei Wang, Xianglin Shi, Bryan S. Jordan, Vince Castranovaa, Michael A. Dubick. **2000**. Wood smoke particles generate free radicals and cause lipid peroxidation, DNA damage, NFkB activation and TNF-a release in macrophages. Toxicology 150 147 – 157.

[3]. Elsa Nielsen, Marianne Dybdahl. Health effects assessment of exposure to particles from wood smoke. Danish environmental protection agency, Environmental project. No. 1235. **2008**.

[4]. Wei Y. L and Adeline S. 2012. Biomass fuels and lung cancer. Respirology. 17, 20–31.

[5]. Orozco L.M, Garcia J, Villar A, Ramirez S, Anto J.M and Jea J. 2006. Wood smoke exposure and risk of chronic obstructive pulmonary disease. Eur Respir J; 27: 542–546.

[6]. Krajcovicova M, Paukova V. and Dusinska M. 2004. Lipid peroxidation in relation to vitamin C and vitamin E levels. Cent. Eur. J. Publ. Health; 12 (1): 46-48.

[7]. Emma A. M, Orla P. B, John A. L, Joshua R and Garret A. F. 2001. Effects of vitamin E on lipid peroxidation in healthy persons. JAMA. Vol 285, No. 9: 1178-1182.

[8]. Karatas F, Kara H, Servi S, Tug T, Erulas F.A and Koca M. 2005. Investigation of Antioxidant Vitamins (A, E, C) and Lipid Peroxidation Levels in Rats Injected N-(1,3-Benzothiazol-2-yl)-N-(4,5-dihydro-1H-imidazol-2-yl) amine. Molecules, 10, 922-928

[9]. Kato J, Alice A. R, Sekharjit S, Bilasini D.S, Th Ibetombi D and Gyaneshwar S.W. 2007. Lipid peroxidation and antioxidant vitamins in urolthiasis. Indian Journal of Clinical Biochemistry, vol. 22 (1): 128-130.

[10]. Ghinwa M. N and Bohumil V. Toxicity and Sources of Pb, Cd, Hg, Cr, As, and Radionuclides in the Environment (part 2). Heavy Metals in the Environment. 2009. pp.1-50.

[11]. Bieby V.T, Siti Rozaimah S.A, Hassan B, Mushrifah I, Nurina A. and Muhammed M. A Review on Heavy Metals (As, Pb, and Hg) Uptake by Plants through Phytoremediation. Inter. J Chem. Engin. Vol. 2011:1-32.

[12]. Saraymen R, Kilic E,Yazar S, Saraymen B. Magnesium, copper, zinc, iron and chromium levels in sweat boxers. JIUMF. 2003, 10(3): 121 -125.

[13]. Osama M. AL. sabaawy. 2012. The relationship between lipid profile and selected trace elements for adult in Mosul city. Oman Med. J. Vol. 27(4):300-303.

[14]. Arash R, Afsaneh T, Safoura K and Abdol JM. 2014. Serum copper, zinc and lipid peroxidation in pregnant women with preeclampsia in Gorgan. Open Biochem. J. 8:83-88.

[15]. C.A. Burtis, E.R. Ashwood, Tietz. Fundamentals of Clinical Chemistry, 4th ed., Elsevier India Pvt. Ltd., India, 1996, pp. 341-349.

[16]. Martineau J, Barthelemy C, Garreau B, Lelord G. 1985. Vitamin B6, magnesium, and combined B6-Mg: therapeutic effects in childhood autism. Biological Psychiatry. 20 (5):467–478.

[17]. Power L.W. 1989. Diagnostic hematology clinical and technical principles, Clin. Chim. Acta. Vol. 11, p. 101-121.

[18]. Jaffer Hashim Mohsen, Hanan Fadel Abbas and Kasim Kadhim Alasedi. 2013. The effect of mercury on lipid peroxidation and its relation with vitamin (A,E) and essential elements in dentals serum. J. Life Sci. Vol.7 (4), p. 370-376.

[19]. Rice, E.W.1967. Rapid determination of total hemoglobin as hemoglobin cyanide in blood containing carboxyhemoglobin. Clin. Chim. Acta, 18, 89 -91.

[20]. Annaduria, B.2007. A Textbook of Biostatistics.1st ed. New Age International Publishers (formerly Wiley Eastern Limited). New Delhi. P. 201-213.

[21]. Bryson, P. D., Wholers, M. D., (1996) "Comprehensive Review in Toxicology for Emergency Clinicians", 3rd ed., New York, San Francisco., pp. 600-613. Burtis, C. A. & Ashood, E. R., (1999)"Lead, TextBook of Clinical Chemistry", New York. pp. 989-91.

[22]. Mazzetti, B. Grigolo, R.M. Borzoi, R. Meliconi, A. Facchini, Serum copper/zinc superoxide dismutase levels in patients with rheumatoid arthritis, Int. J. Clin. Lab. Res. 26 (4) (1996) 245-249.

[23]. Bruninga, D. (2000) Vitamins C and E fight side effect of pelvic radiation for cancer. Am. J. Gastroenterology, 86:1-5.

[24]. J.H. Muhsen, A.M. Ahmood, W.F. AL-Taie, K.A. AL-Zubaidi, Mercury pollution and its relation with essential and trace elements (Zn, Mg, Cr, Fe) in occupational workers, Ibn Al-Haitham J. For Pure & Appl. Scl. A 19 (4) (2006) 90-100.

[25]. A.M. Ahmood, J.H. Muhsen, W.F. AL-Taie, K.A. AL-Zubaidi, Xanthine oxidase activity in relation with some essential and trace elements in atherosclerosis sera, Ibn Al-Haitham J. For Pure & Appl. Sci. 20 (2) (2007) 73-81.

[26]. John E. J. 2000. The role of carotenoids in human health. Nutr. Clin. Care. Vol. 5(2):56-65.

[27]. Yue Z, Wan-Rong H, Shize and Ying-Ping L. 2008. Effects of vitamin A on conjunctival goblet cells of rat after corneal transplantation. Int. J. Ophthalmolo. Vol.1 (2): 109-113.

[28]. Pekmezci D. 2011. Vitamin E and immunity. Vitam. Horm. Vol. 86:179-215.

[29]. D. Bruninga, (2000) Vitamins C and E fight side effect of pelvic radiation for cancer, Am. Gastroenterology 86:1-5.

[30]. Kaket TP. and Ahmad R. 2011. Biochemical studies on hemoglobin modified with reactive oxygen species (ROS). Appl. Biochem. Biotech.Vol. 164 (8):1422-1430.

[31]. Barry H. 2008. The antioxidants properties of serum albumin. FEBS Let. Vol. 582.

[32]. R.T. Fowler, M.H. Abernethy, T.A. Walmsley, H.W. Taylor, Measurement of magnesium by continuous-flow colorimetry, Clin. Chem. 28 (1982) 523.

[33]. H.E. Ganther, D.G. Hafeman, R.A. Lawrence, R.E. Serfass, W.G. Hoekstra, Selenium and glutathione peroxidase in health and disease a review, in: A.S. Prasad, D. Oberleas (Eds.), Trace Elements in Human Health and Disease, Vol. 2, Essential and Toxic Elements, Academic Press, New York, 1976, pp. 165-234.

[34]. Indranil C, Sanjoy K, Mousumi B and Anindya D. 2007. Evaluation of srun zinc level and plasma SOD activity in senile cataract patients under oxidative sress. Indian J. Clin. Biochem. Vol 22 (2) :109-113.