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BIOCHEMICAL ALTERATIONS ON OCCUPATIONAL STRESS BETWEEN SMOKING AND NON-SMOKING IRON MEN

Hussein A. Abd El-Maksoud[1], **Lobna M. Salem** [2], **Mohamed G. El-Harrif**[1]
[1]*Department of Biochemistry Fac. Of Vet. Med. Benha University,* [2] *Department of zoonosis Fac. of Vet. Med. Benha University. Egypt.*

A B S T R A C T

The aim of the present study is to clarify the effect of welding fumes as pulmonary oxidative stress on allergic factors, antioxidants, some minerals and immunoglobulines of smoking and non-smoking iron and steel workers. In order to achieve this aim 40 male iron and steel workers of whom 20 were smokers and 20 non-smokers, were recruited. Control subject were 20 healthy volunteer never exposed to welding fumes and non-smoking. The result of the present study showed a significant association between exposure to welding fumes and high level of iron, copper, lead, and manganese, low zinc level, high catalase activity, low super oxide dismutase activity, low glutathione reductase and peroxidase activity, low reduced glutathione level, high level of malondialdhyde, low nitric oxide level, high cortisol level and high immunoglobulines level. These parameters may all be regards as risk factors for exposure to welding fumes. The finding of the present study suggest that oxidative stress, vascular inflammation, allergic reactions and recurrent infections are primary interacting mediators of diseases caused by exposure to welding fumes.

Key words: Oxidative stress, welding fumes, antioxidants, Blood metals, immunoglobulines, cortisol

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1. INTRODUCTION

Iron is the world's most commonly used metal and can usually be found with other elements in the form of steel. It is used primarily in structural engineering applications, maritime purposes, automobiles, and general industrial applications (machinery). Steel can be cast into bars, strips, sheets, nails, spikes, wire, rods or pipes. World production averages one billion metric tons of raw ore annually [3]. In developing countries, the labor is cheap, proper occupational hygiene and pollution control methods are often neglected at worksites. One of the most common cause of injury and illness in the Iron and steel industry is inhalable agent (gases, vapors, and welding fumes) [34]. Welding generates fumes that are a complex mixture of gases (carbon monoxide, carbon dioxide, nitrous oxide, ozone) and metal particulates (iron, manganese, chromium, nickel). These aerosols are comprised of high concentrations of fine and ultrafine metal particles, including manganese, chromium and nickel, which are known to be toxic [4]. Occupational exposure to welding fume among welders leads to alterations of manganese, iron, zinc and lead in body fluids and the oxidative stress status. Welders had altered erythrocytic superoxide dismutase activity and serum malondialdehyde activity. These suggest that occupational exposure to welding fumes among welders disturbs the homeostasis of trace elements in systemic circulation and induces oxidative stress [17]. The excess accumulation of iron in cells produce cellular oxidative stress, leading to cellular damage [28]. Manganese can produce free radicals at cytotoxic levels

causing oxidative stress and neurodegeneration [14]. Cigarette smoking is acknowledged as one of the leading cause of preventable morbidity and mortality, and is one of the largest preventable causes of ill health in the world. Cigarette smoking was responsible for 17%-30% of all deaths from cardiovascular illness. The effect of cigarette smoking are not related to the life style modification [35]. The smoking welders had an increased incidence of abnormal pulmonary function test than the controls who smoked. In other words smoking and welding almost double the percentage of those having abnormal pulmonary function tests [30]. The aim of the present study was to show the biochemical alternation of trace elements, oxidants, antioxidant enzyme, cortisol and immunoglobulines level due to exposure to welding fumes in iron and steel workers.

2. MATRAILS AND METHODS

This study conducted on 40 male of iron and steel workers, of whom 20 were smokers and 20 non-smokers selected from smithy and welding workshops and 20 healthy individuals as control healthy group who was never exposed to welding fumes, non-smokers and without respiratory affection. Any individuals of all groups how had history of diabetes mellitus and kidney and liver disease and atrophy or allergy was excluded. Each group of the three groups was classified according to age into: less than 26 years, 26-35 years, 36-45 years and over 45 years.

2.1.Sampling:

From each subject 10 ml of venous blood were taken through a vein puncture using a dry plastic disposable syringe under complete aseptic condition. The blood samples divided into 2 portions the first one poured on 5% ethylenediamine tetraacetic acid (EDTA). Plasma sample were collected after centrifugation and used freshly for determination of nitric oxide (NO) according to Bories and Bories[8], malondialdhyde (MDA) according to Draper and Hadly [12], total super oxide dismutase (t.SOD) according to Misra and Fridovich[19], reduced glutathione(GSH), glutathione reductase (GR) according to Bergmayer[7] and glutathione peroxidase (GP_x) according to Chiu et al. [9]. The remained 5 ml blood poured in tubes without anticoagulants allowed to clot then and centrifuged for isolation of the serum which used freshly for determination of copper, zinc, iron, manganese, and lead according to Bauer [6]as well as cortisol according to Mullner et al. [20], IgE according to Plebani et al.[22], total protein immunoglobulines Igs according to Whicher et al. [33] and catalase according to Sinha[29].

2.2.Statistical analysis:

All values were expressed as mean ± standard error (SE). All statistical analyses were performed using SPSS (version 19). Statistical differences among the experimental groups were assessed by ANOVA. Duncan's test was used as a follow-up test and significance was defined at $p<0.05$.

3. RESULTS

The presented data revealed that exposure to the welding fumes is accompanied by significant decrease ($P<0.05$) in the mean values of serum zinc, plasma total super oxide dismutase, plasma glutathione reductase, plasma glutathione peroxidase, plasma reduced glutathione and plasma nitric oxide. Significant increase ($P<0.05$) in the mean values of serum iron, copper, lead, manganese, serum catalase activity, plasma malondialdhyde, serum cortisol and serum immunoglobulines on compassion with the mean values recorded in the control healthy individuals group

4. DISSCUSION

The increased generation of Reactive oxygen species (ROS) produced by

Table 1: Mean values ± S.E of Serum Zinc (µg/dl), Iron (µg/dl), Lead (µg/dl), Copper (µg/dl), and Manganese (µg/dl).

Parameter	Age	Men groups		
		Control	Non smoking	Smoking
Zinc	Less 26	91.67 ± 0.76^{dC}	87.15 ± 0.44^{dB}	47.93 ± 0.50^{dA}
	26-35	87.17 ± 0.66^{cC}	63.13 ± 0.47^{cB}	39.29 ± 0.39^{cA}
	36-45	80.47 ± 0.64^{bC}	60.27 ± 0.52^{bB}	37.72 ± 0.33^{bA}
	More 45	62.79 ± 0.45^{aC}	57.56 ± 0.28^{aB}	31.78 ± 0.36^{aA}
	Average	80.52 ± 3.32^{C}	67.03 ± 3.56^{B}	39.18 ± 1.75^{A}
Iron	Less 26	100.67 ± 1.45^{aA}	107.33 ± 0.88^{aC}	103.33 ± 0.88
	26-35	107.67 ± 1.45^{bA}	122.33 ± 0.88^{bC}	109.33 ± 1.86^{aB}
	36-45	110.33 ± 0.88^{cA}	131.33 ± 2.33^{cC}	115.67 ± 0.88^{bB}
	More 45	116.00 ± 1.15^{dA}	136.33 ± 1.45^{dC}	119.00 ± 1.53^{cB}
	Average	108.67 ± 1.75^{A}	124.33 ± 3.3^{C}	111.83 ± 1.9^{dB}
Lead	Less 26	22.95 ± 0.35^{aA}	26.21 ± 0.20^{aB}	39.19 ± 0.37^{aC}
	26-35	27.56 ± 0.26^{bA}	30.14 ± 0.16^{bB}	40.71 ± 0.32^{bC}
	36-45	29.19 ± 0.21^{cA}	32.51 ± 0.21^{cB}	44.28 ± 0.42^{cC}
	More 45	32.07 ± 0.21^{dA}	38.05 ± 0.36^{dB}	51.81 ± 0.64^{dC}
	Average	27.94 ± 1.00^{A}	31.73 ± 1.30^{B}	44.00 ± 1.48^{C}
Copper	Less 26	65.95 ± 0.35^{aA}	73.3 ± 1.04^{aB}	138.13 ± 0.94^{aC}
	26-35	77.21 ± 0.49^{bA}	79.96 ± 0.94^{bB}	171.59 ± 1.09^{bC}
	36-45	81.36 ± 1.23^{cA}	84.37 ± 0.91^{cB}	177.01 ± 1.06^{cC}
	More 45	85.65 ± 0.47^{dA}	89.68 ± 2.06^{dB}	208.59 ± 1.76^{dC}
	Average	77.55 ± 2.23^{A}	81.83 ± 1.90^{B}	173.83 ± 7.55^{C}
Manganese	Less 26	1.74 ± 0.03^{aA}	2.15 ± 0.06^{aB}	3.64 ± 0.09^{aC}
	26-35	1.96 ± 0.07^{bA}	2.35 ± 0.05^{bB}	4.35 ± 0.15^{bC}
	36-45	2.19 ± 0.05^{cA}	2.77 ± 0.06^{cB}	4.64 ± 0.06^{cC}
	More 45	2.50 ± 0.05^{dA}	3.46 ± 0.10^{dB}	5.06 ± 0.12^{dC}
	Average	2.10 ± 0.09^{A}	2.68 ± 0.15^{B}	4.42 ± 0.16^{C}

Data presented as (Mean ± S.E). S.E = Standard error. Mean values with different superscript letters in the same column are significantly different at ($P<0.05$). Mean values with different superscript letters in the same row are significantly different at ($P<0.05$).

chromium (Cr), lead (Pb), iron (Fe) and manganese (Mn), has been shown to disrupt biochemical homeostasis, resulting in lipid peroxidation, DNA damage, depletion of sulfhydryls, and altered calcium homeostasis [36]. These biochemical alternations have the potential to produce adverse health effects in welders [17]. Welding workers are more prone to impaired pulmonary function, chronic bronchitis, asthma, lung cancer, photokeratitis, eye burns, cataract, chronic damage of external parts of the eye, maculopathy impaired humeral immunity, erythema, non-melanocytic skin cancer, malignant melanoma, reduced fertility, decreased volume of semen and sperm count, direct toxicity to sperm production and decreased sperm motility [30]. The present study showed, a significant increase in the mean value of serum iron, lead, copper, and manganese and significant decrease in the mean value of serum zinc was observed in welders when compared with control group. In the study done by Guojun [13] who showed that the serum concentrations of manganese and iron as well as the blood lead concentration were significantly higher in welders than in control subjects.

Table 2: Changes of Plasma GSH (mmol/L), GR (U/L), GP_X (U/L), SOD (U/ml)), Serum CAT (U/ml).

Parameter	Age	Men groups		
		Control	Non smoking	Smoking
GSH	Less 26	18.93 ± 0.13^{dC}	10.59 ± 0.09^{dB}	7.56 ± 0.06^{dA}
	26-35	14.31 ± 0.21^{cC}	9.64 ± 0.20^{cB}	6.82 ± 0.06^{cA}
	36-45	13.40 ± 0.46^{bC}	8.55 ± 0.07^{bB}	6.33 ± 0.09^{bA}
	More 45	11.69 ± 0.19^{aC}	8.06 ± 0.15^{aB}	5.83 ± 0.11^{aA}
	Average	14.58 ± 0.82^{C}	9.21 ± 0.30^{B}	6.63 ± 0.20^{A}
GR	Less 26	11.91 ± 0.07^{bC}	8.49 ± 0.12^{cB}	6.22 ± 0.07^{cA}
	26-35	11.39 ± 0.08^{bC}	7.68 ± 0.08^{bB}	5.51 ± 0.07^{bA}
	36-45	10.98 ± 0.09^{aC}	6.84 ± 0.06^{aB}	5.10 ± 0.08^{bA}
	More 45	10.57 ± 0.09^{aC}	6.55 ± 0.05^{aB}	4.16 ± 0.05^{aA}
	Average	11.21 ± 0.15^{C}	7.39 ± 0.23^{B}	5.25 ± 0.23^{A}
GP_X	Less 26	8.12 ± 0.09^{dC}	5.47 ± 0.08^{dB}	3.50 ± 0.07^{dA}
	26-35	7.20 ± 0.08^{cC}	4.69 ± 0.07^{cB}	3.14 ± 0.04^{cA}
	36-45	6.22 ± 0.06^{bC}	3.99 ± 0.19^{bB}	2.46 ± 0.15^{bA}
	More 45	5.73 ± 0.06^{aC}	3.45 ± 0.08^{aB}	1.96 ± 0.10^{aA}
	Average	6.82 ± 0.28^{C}	4.40 ± 0.23^{B}	2.77 ± 0.18^{A}
CAT	Less 26	29.62 ± 0.50^{bA}	38.68 ± 0.46^{aB}	60.22 ± 1.08^{aC}
	26-35	23.07 ± 10.03^{aA}	45.19 ± 0.49^{bB}	70.33 ± 0.80^{bC}
	36-45	34.90 ± 0.45^{cA}	47.95 ± 0.52^{cB}	74.35 ± 0.81^{cC}
	More 45	41.14 ± 0.78^{dA}	51.77 ± 0.65^{dB}	86.91 ± 1.01^{dC}
	Average	32.18 ± 2.94^{A}	45.9 ± 1.46^{B}	72.95 ± 2.91^{C}
SOD	Less 26	15.83 ± 0.15^{dC}	11.71 ± 0.11^{dB}	7.90 ± 0.14^{dA}
	26-35	13.24 ± 0.12^{cC}	10.72 ± 0.18^{cB}	7.33 ± 0.06^{cA}
	36-45	12.27 ± 0.12^{bC}	9.94 ± 0.05^{bB}	6.91 ± 0.09^{bA}
	More 45	11.75 ± 0.10^{aC}	9.10 ± 0.10^{aB}	5.46 ± 0.11^{aA}
	Average	13.27 ± 0.48^{C}	10.37 ± 0.3^{B}	6.9 ± 0.28^{A}

Data presented as (Mean ± S.E). S.E = Standard error. Mean values with different superscript letters in the same column are significantly different at ($P<0.05$). Mean values with different superscript letters in the same row are significantly different at ($P<0.05$).

Table 3: Changes of Plasma NO (µmol/l), L-MDA (nmol/ml)

Parameter	Age	Men groups		
		Control	Non smoking	Smoking
NO	Less 25	59.26 ± 0.70^{dC}	41.24 ± 0.44^{dB}	33.59 ± 0.34^{dA}
	26-35	55.40 ± 0.66^{cC}	33.61 ± 0.35^{cB}	29.92 ± 0.31^{cA}
	36-45	52.56 ± 0.47^{bC}	30.66 ± 0.30^{bB}	27.70 ± 0.51^{bA}
	More 45	49.64 ± 0.50^{aC}	27.48 ± 0.30^{aB}	19.78 ± 0.16^{aA}
	Average	54.21 ± 1.1^{C}	33.25 ± 1.54^{B}	27.75 ± 1.53^{A}
L-MDA	Less 26	11.35 ± 0.23^{aA}	21.12 ± 0.40^{aB}	44.18 ± 0.32^{aC}
	26-35	11.79 ± 0.11^{bA}	24.8 ± 0.38^{bB}	53.00 ± 0.49^{bC}
	36-45	12.51 ± 0.23^{cA}	26.54 ± 0.47^{cB}	55.31 ± 0.48^{cC}
	More 45	13.72 ± 0.39^{dA}	33.28 ± 1.01^{dB}	62.23 ± 0.81^{dC}
	Average	12.34 ± 0.29^{A}	26.44 ± 1.36^{B}	53.68 ± 1.96^{C}

Data presented as (Mean ± S.E). S.E = Standard error. Mean values with different superscript letters in the same column are significantly different at ($P<0.05$). Mean values with different superscript letters in the same raw are significantly different at ($P<0.05$).

Table 4: Changes of Serum IgA (mg/dl), IgG (mg/dl), IgM (mg/dl), IgE (µg/ml), Cortisol (µg/dl)

Parameter	Age	Men groups		
		Control	Non smoking	Smoking
IgA	Less 26	74.00±1.73^{aA}	178.00±4.58^{aB}	233.67±7.51^{aC}
	26-35	95.67±2.03^{bA}	212.67±2.73^{bB}	263.33±2.96^{bC}
	36-45	105.33±2.03^{cA}	234.00±3.79^{cB}	281.33±2.60^{cC}
	More 45	127.67±3.76^{dA}	274.33±6.01^{dB}	323.00±7.37^{dC}
	Average	100.67±5.91^{A}	224.75±10.7^{B}	275.33±10.05^{C}
IgG	Less 26	687.33±17.61^{aA}	861.00±7.00^{aB}	1127.33±21.22^{aC}
	26-35	756.67±7.69^{bA}	944.00±7.37^{bB}	1213.00±11.93^{bC}
	36-45	780.67±4.63^{cA}	971.00±5.51^{cB}	1356.33±12.81^{cC}
	More 45	814.33±7.31^{dA}	1022.67±9.53^{dB}	1663.67±6.98^{dC}
	Average	759.75±14.74^{A}	949.67±17.92^{B}	1340.08±61.79C
IgM	Less 26	75.67±2.03^{aA}	103.67±1.45^{aB}	133.67±1.45^{aC}
	26-35	85.33±1.45^{bA}	116.33±1.45^{bB}	171.67±2.03^{bC}
	36-45	92.67±1.45^{cA}	128.00±2.08^{cB}	188.00±2.65^{cC}
	More 45	104.67±2.03^{dA}	151.33±11.39^{dB}	216.67±3.48^{dC}
	Average	89.58±3.28^{A}	124.83±5.86^{B}	177.5±9.11^{C}
IgE	Less 26	19.67±0.88^{aA}	76.33±1.2^{aB}	110.00±1.53^{aC}
	26-35	28.67±0.88^{bA}	88.00±1.15^{bB}	124.00±1.73^{bC}
	36-45	35.33±0.88^{cA}	92.33±1.20^{cB}	130.67±1.45^{cC}
	More 45	41.67±1.45^{dA}	102.33±0.88^{dB}	139.33±0.88^{dC}
	Average	31.33±2.50^{A}	89.75±2.85^{B}	126±3.29^{C}
Cortisol	Less 26	2.17±0.05^{aA}	6.48±0.15^{aB}	8.34±0.19^{aC}
	26-35	2.30±0.05^{aA}	7.12±0.12^{bB}	9.91±0.17^{bC}
	36-45	2.74±0.09^{bA}	7.60±0.11^{cB}	10.65±0.19^{cC}
	More 45	3.86±0.09^{cA}	9.57±0.24^{dB}	13.74±0.30^{dC}
	Average	2.77±0.2^{A}	7.69±0.35^{B}	10.66±0.6^{C}

Data presented as (Mean ± S.E). S.E = Standard error. Mean values with different superscript letters in the same column are significantly different at (P<0.05). Mean values with different superscript letters in the same raw are significantly different at (P<0.05).

The serum manganese concentrations in welders showed approximately four-fold increases as compared with those of control subjects, whereas the increases in levels of serum iron and blood lead were 1.9-fold and 2.6-fold, respectively. In addition, Long-term, low-level exposure to the welding fume cause significantly increase in serum concentrations of manganese and iron and the blood concentration of lead among the welders [17]. The results were in agreement with Shehata [28] who found that there was significant differences between the smokers of the exposed group and the control group as regards the levels of lead, cadmium, manganese, iron, chromium. Moreover, [24] demonstrated that the smoking is dangerous to health and can increase the total body burden of heavy metals for metals workers. The present study revealed that a significant decrease in the mean value of serum total superoxide dismutase, glutathione reductase, glutathione peroxidase and reduced glutathione and significant increase in the mean value of serum catalase was observed in welders when compared with control group. Which were in agreement with McNeilly et al. [18] who reported that exposure to welding fumes cause enhanced production of (ROS) with concomitant depletion of antioxidants enzymes. In the study done by Zhu et al. [37] who demonstrated that the average values of SOD, CAT and GP_X erythrocytes in the welders group were significantly decreased and suggested that with a prolonged duration of exposure to photochemical smog of welding the values of SOD, and GP_X, except for CAT, in the welders were significantly decreased. This

significant decrease may be due to possible oxidative stress resulted from increased production of reactive oxygen species. In contrast with our finding Sung et al. [31]revealed that the antioxidant enzyme glutathione peroxidase (GP_X) and Superoxide dismutase(SOD) showed significant increase in welders compared with unexposed subjects. They explained this increase by the fact that increased oxidative stress by welding fumes might stimulate the formation of more antioxidant enzymes to compensate the consumed SOD and other antioxidant enzymes, so it is possible that prolonged oxidant stress leads to an increase in these antioxidant enzymes. The elevation in catalase activity indicate adaptation changes in response to large amount of hydrogen peroxide which decomposed by catalase, due to catalase enzyme which is found in blood, bone marrow, mucus membranes, kidney and liver. Its function is destruction of H_2O_2 formed by action of oxidase [2]. In addition, it reported that the plasma antioxidant capacity was significantly decreased in welders who smoke 1 h after smoking, when compared with plasma from age-matched nonsmoking control subjects. The decrease in plasma antioxidant capacity in smokers may be due to a profound depletion of plasma protein sulfhydryls [23]. Smoking can trigger the oxidative stress in various tissues. The SOD levels in the smoking welders were significantly higher when compared to non-smoking welders and controls. Furthermore, the increase in CAT activity was significant in smoking welders compared to the controls. These findings have revealed that smoking triggers antioxidant enzyme activities between welders[15]. The present study showed a significant increase of the mean value of plasma activity of MDA and significant decrease in the mean value of plasma level of nitric oxide was observed in welders when compared with control group. It was reported that the difference in the range of MDA activity in the welder subjects was much higher than in controls due to excessive formation of free radical and activation of lipid peroxidation [15]. MDA increases under heavy metal stress, and an increasing amount of MDA represents the formation of free radicals under heavy metal stress [10]. Tobacco smoke contains large numbers of free radicals that are capable of initiating or promoting oxidative injury. Cigarette smokers have higher lipid peroxidation products in their blood compared to non-smoking and smoking increase the concentration of serum MDA activity [21]. The decrease in nitric oxide level may be explained by; perhaps the nitric oxides formed are reacting with other free radicals, such as superoxide, produced by neutrophil or undergoing denitrification by bacteria thereby decreasing the observed levels. There may also be up regulation of NO synthase isoform which might serve to overcome the worsing air way construction [26]. In addition, this decreased production of NO may be related to diminished L-arginine transport capacity, leading to the decreased basal NOS activity. An additional factor that could limit L-arginine transport is its low plasma L-arginine concentration [25]. The present study revealed that a significant increase in the mean value of serum level of cortisol, IgE and total protein immunoglobulines was observed in welders when compared with control group. The significant increase in serum cortisol indicated that the workers were stressed. Simultaneous increase in serum IgE was correlated well with increased cortisol concentrations. The changes in the levels of serum cortisol affected the IgE levels[16]. These results exhibited a bidirectional feedback between the immune system and hypothalamus-pituitary-adrenal axis. The levels of cortisol can regulate IgE levels [32]. In this respect, Cytokines and chemokines that are important in inflammatory responses were all significantly increased in the stainless steel welding fume group after infection compared to the infected air control. This elevation in inflammatory signaling is

likely due to an enhanced innate immune response to the elevated bacterial burden in the lungs of the stainless steel welding fume group [5]. During a severe inflammatory response, inflammatory cells release a number of mediators, including interleukin 1 (IL-1), tumor necrosis factor (TNF-a), amines, and so forth. At elevated levels, these mediators will stimulate centers in the brain, which in turn activate the hypothalamic pituitary-adrenal axis; these results in an increase of cortisol in the circulation, thereby attenuating the inflammatory response [27]. In addition, studies in mice have demonstrated that known human chemical respiratory allergens provoke selective type 2 immune responses associated with specific IgE antibody production, increases in the total serum concentration of IgE and the induced or elevated expression of cytokines that favour the elicitation of immediate type allergic reactions [11]. Furthermore, immune defense mechanism such as secretory immunoglobulin and interferon may be also adversely affected and this explain increase mortality among metals exposed mice challenged with influenza virus [1]. In conclusion, exposure to welding fumes accompanied by high levels of Fe, Cu, Pb, Mn, MDA, catalase activity, cortisol, and immunoglobulines and low activity of Zn, SOD, GR, GP_X , GSH and NO. These may all be regarded as risk factors due to exposure to welding fumes in iron and steel workers.

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مجلة بنها للعلوم الطبية البيطرية
BENHA UNIVERSITY
FACULTY OF VETERINARY MEDICINE

التغيرات الكيميائية الحيوية وأثرها في الاجهاد الوظيفي بين الحدادين المدخنين وغير المدخنين

حسين عبد المقصود على[1]-لبني محمد علي سالم[2] -محمد جمال الحريف [1]

[1]قسم الكيمياء الحيوية -كلية الطب البيطري بمشتهر-جامعة بنها.
[2] قسم الامراض المشتركة-كليه الطب البيطري بمشتهر-جامعه بنها.

الملخص العربي

تم اجراء هذا البحث لدراسة التأثير الكيميائي الحيوي للتعرض لأدخنة اللحام في عمال الحدادة واللحام المدخنين وغير المدخنين. فلقد اجريت هذه الدراسة على 60 شخصا متطوعا تم تقسيمهم كالاتي: المجموعة الاولي (المجموعة الضابطة): وتشمل 20شخصا من غير المعرضين لا دخنه اللحام ومن غير المدخنين. المجموعة الثانية (المجموعة الضابطة الموجبة): تحتوي على 20عاملا من المعرضين لأدخنة اللحام من غير المدخنين. المجموعة الثالثة: تحتوي على 20 عاملا من المعرضين لأدخنة اللحام ومن المدخنين. وقد تم تقسيم المجموعات الثلاث حسب مراحلهم العمرية الي: اقل من 25 عام–من 25الي 35عام–من 36–45عام–أكبر من 45عام. هذا وقد استخدم مصل الدم لقياس مستويات الحديد والنحاس والزنك والرصاص والمنجنيز وانزيم الكتاليز والكورتيزول والاميونوجلوبيولينات. واستخدمت الخلايا الدموية الحمراء لقياس انزيمات سوبر اوكسيد ديسميوتاز وجلوتاثيون ريدكتاز وجلوتاثيون بيرواكسيداز والجلوتاثيون المختزل واكسيد النيتريك ومالون داي الدهيد. هذا وقد اسفرت الدراسة عن وجود تغيرات كيميائية حيوية واضحة لدي عمال الحدادة واللحام تمثلت في زيادة معنويه في مستويات كل من الحديد والرصاص والنحاس والمنجنيز ونشاط انزيم الكتاليز ومالون داي الدهيد وكذلك مستوي الكورتيزول والاميونوجلوبيولينات. كما اسفرت عن وجود نقص معنوي في نشاط كل من انزيم سوبر اوكسيد ديسميوتاز وجلوتاثيون ريدكتاز وجلوتاثيون بيرواكسيداز والجلوتاثيون المختزل وكذلك اكسيد النيتريك ومستوي الزنك في الدم. ولقد بينت الدراسة ان عمال الحدادة واللحام لديهم زيادة في حالة التأكسد وكذلك هم أكثر عرضه من غيرهم لأمراض الحساسية والالتهابات كما ان للتدخين دور مهم في ذلك. لذا تنصح الدراسة عمال الحدادة اللحام بالابتعاد عن التدخين تماما والعمل في اجواء ملائمه واستعمال الادوات الواقية وكذلك اجراء فحوصات دوريه للتأكد من سلامتهم.

(مجلة بنها للعلوم الطبية البيطرية: عدد 24 (1)، يونيو 2013: 118-127)