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Biochemical alterations on occupational stress between smoking and non-smoking iron & steel workers in Egypt

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Superoxide dismutase: SOD, Catalase: CAT, Reduced glutathione: GSH, Glutathione peroxidase: GP_X, Glutathione reductase: GR, Nitric oxide: NO, Malondialdhyde: L-MDA

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Abstract
Many welders have experienced bronchitis, metal fume fever, lung function changes, and an increase in the incidence of lung infection. 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.

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1. Introduction

1.1 Iron and Steel

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 (Allam, 2009).

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) (William and Burgers, 1995).

1.2 Welding Fumes

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 (Antonini et al., 2006).
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 (Li et al., 2004). The excess accumulation of iron in cells produces cellular oxidative stress, leading to cellular damage (Shehata, 2009). Manganese can produce free radicals at cytotoxic levels causing oxidative stress and neurodegeneration (Hamai and Bondy, 2005)

1.3 Smoking and Welding
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 is not related to the life style modification (Yathish et al., 2011).

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 (Sultan et al., 2004)

2. Objective of Research

The main objective 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.

3. Materials and Methods

Welding results in a unique and complex occupational exposure. Recent epidemiological studies have shown an increased risk of diseases following welding fume exposure. Therefore, present study was designed to assessment the possible health hazards due to exposure to welding fumes. The 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 in different region in egypt 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 3 groups was classified according to age into: less than 26 years, 26-35 years, 36-45 years and over 45 years.

3.1 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% ethylenediaminetetraacetic acid (EDTA). Plasma sample were collected after centrifugation and used freshly for determination of nitric oxide (NO) according to Bories and Bories (1995), malondialdhyde (MDA) according to Draper and Hadly (1990), total super oxide dismutase (t.SOD) according to Misra and Fridovich (1972) reduced glutathione(GSH), glutathione reductase (GR) according to Bergmayer (1983) and glutathione peroxidase (GP_x) according to Chiu et al. (1976).

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 (1982) as well as cortisol according to Mullner et al. (1991), IgE according to Plebani et al. (1998), total protein immunoglobulines Igs according to Whicher et al. (1984) and catalase according to Sinha (1972).

3.2 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 $p<0.05$.

4. Results and Discussion

4.1 Levels of heavy metals
The mean value of serum iron level in nonsmokers of iron and steel welders was significantly increased ($P<0.05$) on comparison with healthy control group. The mean value of serum iron level of smokers in iron and steel

welders was significantly increased (P<0.05) on comparison with healthy control and significantly decreased (P<0.05) on comparison with nonsmoking welders group.

The mean value of serum lead, cupper and manganese level in nonsmokers of iron and steel welders was significantly increased (P<0.05) on comparison with healthy control group. The mean value of serum lead, cupper and manganese level of smokers in iron and steel welders was significantly increased (P<0.05) on comparison with healthy control and nonsmoking welders group.

In contrast, the mean value of serum zinc level in nonsmokers of iron and steel welders was significantly decreased (P<0.05) on comparison with healthy control group. The mean value of serum zinc level of smokers in iron and steel welders was significantly decreased (P<0.05)on comparison with healthy control and nonsmoking welders group.

4.2 Activity of antioxidant enzymes

The mean value of erythrocyte SOD, GSH, GR, GP_X activity in nonsmokers of iron and steel welders was significantly decreased (P<0.05) on comparison with healthy control group. The mean value of erythrocyte SOD, GSH, GR, GP_X activity of smokers in iron and steel welders was significantly decreased on comparison with healthy control and nonsmoking welders group.

In contrast, the mean value of erythrocyte catalase activity in nonsmokers of iron and steel welders was significantly increased (P<0.05) on comparison with healthy control group and the mean value of erythrocyte catalase activity of smokers in iron and steel welders was significantly increased (P<0.05) on comparison with healthy control and nonsmoking welders group.

4.3 Serum NO level and L-MDA activity

The mean value of serum nitric oxide level in nonsmokers of iron and steel welders was significantly decreased (P<0.05) on comparison with healthy control group. The mean value of serum nitric oxide level of smokers in iron and steel welders was significantly decreased (P<0.05) on comparison with healthy control and nonsmoking welders group.

The mean value of erythrocyte L-MDA activity in nonsmokers of iron and steel welders was significantly increased (P<0.05) on comparison with healthy control group. The mean value of erythrocyte L-MDA activity of smokers in iron and steel welders was significantly increased (P<0.05) on comparison with healthy control and nonsmoking welders group.

4.4 Level of immunoglobulines and cortisol

The mean value of serum IgG, IgM, IgA, IgE and cortisol level in nonsmokers of iron and steel welders was significantly increased (P<0.05) on comparison with healthy control group.

The mean value of serum IgM level of smokers in iron and steel welders was significantly increased (P<0.05) on comparison with healthy control and nonsmoking welders group.

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

Parameter	Age	Men groups		
		Control	Non smoking	Smoking
	Less 26	91.67 ± 0.76^{dC}	87.15 ± 0.44^{dB}	47.93 ± 0.50^{dA}
Zinc	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 are 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 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}

Table 3: Changes of Serum 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}

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 ± 61.79^{C}
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 are 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 increased generation of Reactive oxygen species (ROS) produced by 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 (Yerer et al.,2004). These biochemical alternation have the potential to produce adverse health effects in welders (Li et al., 2004).

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 (Sultan et al.,2004)

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.

Study done by Guojun (2004) 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. 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.

Also, 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 (Li et al., 2004).

The results were in agreement with Shehata (2009) 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, (Rim et al., 2010) 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. (2004) who reported that exposure to welding

fumes because enhanced production of (ROS) with concomitant depletion of antioxidants enzymes.

In the study done by Zhu et al. (2004) 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. These 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. (2005) 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 assumed to be destruction of H_2O_2 formed by action of oxidase (Abdel-Maksoud et al., 2004).

Also, 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 non smoking control subjects. The decrease in plasma antioxidant capacity in smokers may be due to a profound depletion of plasma protein sulfhydryls (Rahman et al., 1996).

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 (Imamoglu et al., 2010).

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 (Imamoglu et al., 2010).

MDA increases under heavy metal stress, and an increasing amount of MDA represents the formation of free radicals under heavy metal stress (Choudhary et al., 2007).

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 (Pasupathi et al., 2009).

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 (Rutgers et al., 1999).

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 (Rodrigues et al., 2010).

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 (Kataria et al., 2010). These results exhibited a bidirectional feedback between the immune system and hypothalamus-pituitary-adrenal

axis. The levels of cortisol can regulate IgE levels (Toda et al., 2007).

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 (Antonini et al., 2007).

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 (Schwiebert et al., 1996).

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 (Dearman et al., 2000).

Furthermore, immune defence 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 (Abd Allah, 1998)

Conclusion

From the results we concluded that 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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References

Abd Allah H.M., 1998. A study on the health hazards of manganese exposure with emphasis on its immunological effect. M.S. thesis, Faculty of Medicine, Cairo University.

Abdel-Maksoud H., Azab M., Farara K., 2004. Biochemical alternation of hepatic effects of magnesium in antioxidant enzyme. Menofia Veterinary Medical Journal, 3, 23-33.

Allam H.K., 2009. Auditory and respiratory health disorders among worker in iron and steel factory. M.S. thesis, Faculty of Medicine, Menofia University

Antonini J.M., Afshari A.A., Stone S., Chen B., Schwegler-Berry D., Fletcher W.G., Goldsmith W.T., Vandestouwe K.H., McKinney W., Castranova V. and Frazer D.G., 2006. Design, construction, and characterization of a novel robotic welding fume generator and inhalation exposure system for laboratory animals. Journal of Occupational and Environmental Hygie ,3, 194–203.

Antonini J.M., Roberts J.R., 2007. Chromium in stainless steel welding fume suppresses lung defense responses against bacterial infection in rats. Journal of Immunotoxicology, 4, 117-127.

Bauer J.D., 1982. Clinical laboratory methods 9th Ed, the C.V. Mosby company ,11830 , waistline industrial Missouri: 63146 U.S.A p: 112.

Bergmayer H.V., 1983. Methods of enzymatic analysis 3rd Ed. Varlag Chem. Weiheim. Academic Press Inc. 210-212.

Bories P.N., Bories C., 1995. Nitrate determination in biological fluids by enzymatic one step assay with nitrate reductase. Journal of Clinical Chemistry, 41, 904-7.

Chiu D., Staults F., Tappal L., 1976. Purification and properties of rat lung soluble glutathione peroxidase. Biochimica et Biophysica Acta, 445, 558-566

Choudhary M., Jetley U.K., Abash Khan M., Zutshi S., Fatma T., 2007. Effect of heavy metal stress on proline, malondialdehyde, and superoxide dismutase activity in the cyanobacterium Spirulina platensis-S5. Ecotoxicology and Environmental Safety Journal,66, 204–209.

Dearman R.J., Warbrick E.V., Humphreys I.R., Kimber I., 2000. Characterization in mice of the immunological properties of five allergenic acid anhydrides. Journal of Applied Toxicology, 20, 221–230.

Draper H.H., Hadly N., 1990. Malondialdhyde determination as an index of lipid peroxidation. Journal for Methods in Enzymology,186, 421-431.

Guojun J.L., Long L.Z., Ling L., Ping W., Wei Z., 2004. Occupational Exposure to Welding Fume

among Welders. Journal of Occupational & Environmental Medicine, 46, 241-248.

Hamai D., Bondy S., 2005. Oxidative basis of manganese neurotoxicity. Annals New York Academy of Sciences, 1012, 129-140.

Imamoglu N., Mukerrem B.Y., hamiyet D., Recep S., 2010. Erythrocyte antioxidant enzyme activities and lipid peroxidation in the erythrocyte membrane of stainless-steel welder exposed to welding fumes and gases. International Journal of Hygiene and Environmental Health, 211, 63-68.

Kataria A.K., Kataria N., Maan R., 2010. Correlation of serum IgE with stress in Indian dromedaries affected with skin wounds. Journal of Stress Physiology & Biochemistry, 6, 17-24.

Li G.J., Zhang L.L., Lu L., Wu P., Zheng W., 2004. Occupational exposure to welding fume among welders, alteration of manganese, iron, zinc, copper and lead in body fluids and oxidative stress status. Journal of Occupational & Environmental Medicine, 46, 241-248.

McNeilly J.D., Heal M.R., Beverl I.J., Howe A., Gibson M.D., Hibbs L.R., MacNee W., Donaldson K., 2004. Soluble transition metals cause the pro-inflammatory effects of welding fumes in vitro. Toxicology and Applied Pharmacology Journal, 196, 95-07

Misra H.P., Fridovich I., 1972. The role of super oxide anion in the auto oxidation of epinephrine and simple super oxide dismutase. Journal of Biological Chemistry, 247, 3170-3175

Mullner S., Naubauer H., Koning W., 1991. A radioimmunoassay for the determination of insulin in several animal species. Insulin derivatives. Journal of Immunological Methods, 140, 211-198.

Pasupathi P., Rao Y.Y., Farook J., Saravanan G., Bakthavathsalam G., 2009. Effect of smoking on lipid and oxidative stress biomarkers in patient with acute myocardial infarction. Research Journal of Medical Sciences, 4, 151-159.

Plebani M., Bernardi D., Basso D., Faggian D., Borghesan F., 1998. Measurement of specific immunoglobulin E. method comparison and standardization. Journal of Clinical Chemistry, 44, 1974-1979.

Rahman I., Morrison D., Donaldson K., 1996. Systemic oxidative stress in asthma, COPD and smokers. American Journal of Respiratory and Critical Care Medicine, 154, 1055–1060.

Rim K.A., Hamza A.C., 2010. Head and neck cancer due to heavy metal exposure via tobacco smoking and professional exposure. Toxicology and Applied Pharmacology Journal, 248, 71–88.

Rodrigues N.P., Monique B.M., Carmen R.A., Claudia B.C., Giovanni E. M., Tatiana M.C., Antonio C., Mendes R., 2010. Oxidative stress, L-arginine-nitric oxide and arginase pathways in platelets from adolescents with anorexia nervosa. Blood Cells, Molecules, and Diseases Journal, 44, 164-168

Rutgers S.R., van der Mark T.W., Coers W., Moshage H., Timens W., Kauffman H.F., Koeter G.H., Postama D.S., 1999. Markers of nitric oxide metabolism in sputum and exhaled air are not increase in chronic obstructive pulmonary disease. International Journal of Respiratory medicine, 54, 576-80.

Schwiebert A., Lisa A.B., Cristiana S., Carol A.B., Bruce S.B., Robert P.S., 1996. Glucocorticosteroid inhibition of cytokine production: Relevance to anti-allergic actions Journal of Allergy and Clinical Immunology, 97,143-125.

Shehata R.A.S., 2009. oxidative stress status due to metal exposure in welding M.S. thesis, Faculty of Med., Cairo Univ.

Sinha A.K., 1972. Colorimetric assay of catalase. Annals of Clinical Biochemistry Journal, 47, 389-394.

Sultan A.M., Thamir A., 2004. Health hazard of welding fumes. M.S thesis, College of Medicine, King Khalid Univ.

Sung G., Yangho K., Michael L., Donna L., Vincent C., Val V., 2005. Correlates of Oxidative stress and Free radical activity in serum from Asymptomatic Shipyard Welders. American Journal of Respiratory and Critical Care Medicine, 172, 1541-1548.

Toda M., Sakaguchi Y., Morimoto K., 2007 Correlation between serum IgE and salivary cortisol levels in subjects with type I allergic disorders. International Journal of Immunopathology and Pharmacology , 20, 203-205.

Whicher J.T., warren C., Chambers R.E., 1984: Immunochemical assays for immunoglobulines. Annals of Clinical Biochemistry Journal, 21 , 78-91.

William and Burgers S., 1995. Recognition of health hazards in industry: a review of material and prosses. 2nd Ed John Wiley and Sons, New York pp. 303-17

Yathish T.R., Manjula C.G., Deshpande S.R., Gayathree L., 2011. A study on the association of coronary artery disease and smoking by a questionnaire method. Journal of Clinical and Diagnostic Research , 5, 264-268.

Yerer M.B., Yapislar H., Aydogan S., Yalcin O., Baskurt O.K., 2004. Lipid peroxidation and deformability of red blood cells in experimental sepsis in rats: the protective effects of melatonin. Clinical hemorheology and microcirculation Journal, 30, 77-82.

Zhu Y.G., Zhou J.F., Shan W.Y., Zhou P.S., Tong G.Z., 2004. Potential oxidative stress in the bodies

of electric arc welding operators: effect of photochemical smog. Biomedical and Environmental Sciences Journal, 17, 381-9.