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Assessment of hormone levels and some heavy metals of Iraqi traffic-warden policemen exposed to vehicle exhausts

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Roadside traffic air pollution exposures have been associated with various health deteriorates. In accordance with the WHO's guidelines, the air quality in Iraq is considered unsafe. Automobile pollution, particularly diesel exhaust, produces significant quantities of particulates containing endocrine disruptors, causing negative health impacts. The current study attempted to determine whether Iraqi traffic wardens are vulnerable to traffic air pollution by measuring serum progesterone and testosterone levels, as well as serum lead, copper, manganese, cadmium, and iron concentrations. A total of 72 individuals participated in the current study divided into two main groups: traffic policemen and control. Each group was further subdivided into three subgroups based on age, years of service, and smoking habit. The progesterone levels among police officers exposed to vehicle exhaust were negligibly higher than those of the control group. The study data revealed that there was no statistically significant difference in testosterone levels between the two studied groups. Copper was significantly higher in exposed group than that of the control group. Cadmium levels in the exposed group increased non-significantly, which could contribute to endocrine disruption. While iron and lead levels were lower in police exposed to vehicle exhaust than those of the control group, the manganese showed significant decrease in sera of exposed group. High levels of copper in traffic policemen may be associated with increased oxidative stress. Although non-significant decrease in iron level was recorded in the present work, diesel exhaust exposure might increase the incidence of anemia in study subjects.

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KEYWORDS

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Introduction

Air pollution is an important environmental health challenge across the world [1]. According to the most recent data from the Environmental Statistics Report for Iraq Air Pollutants 2019 [1], the country's annual mean concentration of PM2.5 is 62 g/m^3, which exceeds the World Health Organization

(WHO) Air Quality Guidelines for health–harmful pollution levels of 10 g/m^3. In 2016, it was reported that ambient air pollution in both cities and rural areas caused 4.2 million premature deaths worldwide; this mortality is attributed to PM2.5 exposure [2]. Lower respiratory infections were the main cause of mortality in the communicable disease category in Iraq in 2019, according to the WHO Global Health Estimates database [3].

Many studies have found adverse health effects from exposure to roadside traffic. They have documented many of the illnesses linked to air pollution, including impaired lung function, preterm birth, autism, renal disease, dementia, cardiovascular disease, respiratory disorders, and overall mortality [4].

Throughout life, human is constantly exposed to various stressors, and its ability to respond to these stressors directly affects one's overall health [5]. Stress is proven to be a major cause of illness and reduced productivity, especially in occupational setting. Studies on humans beings suggest that chemical stressors, such as nitrogen oxide (NO_2) carbon monoxide (CO), Sulphur oxides (SO_2), cadmium (Cd), lead (Pb), diesel engine exhaust, toluene, persistent organohalogen pollutants (POPs), methyl tert-butyl ether, nitrophenol derivates, present in urban air could cause alterations in synthesis, secretion, and action of plasma hormones such as progesterone and testosterone, also the development of endocrine reproductive diseases related to altered plasma progesterone and testosterone levels [6].

Progesterone is produced not just in the testicles' Leydig cells, but also in the adrenal glands. It has direct effects upon the process of spermatogenesis and on the Central Neural System (SNC) where it acts as a positive or a negative modulator of Gamma-amino butyric acid GABA A receptor; moreover, it regulates several activities, such as learning, memory and sleep [7]. Furthermore, alterations in plasma progesterone levels could cause mental health disorders in male subjects, for instance: Panic attacks, anxiety, posttraumatic stress disorders, depression and schizophrenia [8].

The synthesis of testosterone occurs in Leydig cells, where there is an adequate supply of cholesterol as a raw material, and it plays an important role in regulating bone growth and puberty in men; it is also important for the development of the male fetus, puberty, and supports sperm production in men. Exposure to air pollutants may alter plasma testosterone and progesterone levels, which are linked to reproductive diseases as well as mental health diseases such as mood disorders, which diminish worker efficiency and performance [9]. However, there has only been little research on the link between air pollution and male reproductive heavy metal contamination. Hazardous metals are one of the most widely dispersed toxins in the world's living environment. They impact cell membrane, mitochondria, lysosomes, endoplasmic reticulum, nuclei, and several enzymes involved in metabolism, detoxification, and damage repair [10]. Several studies have shown that metals like arsenic, cadmium, chromium, lead, and mercury are involved in the overproduction of reactive oxygen species (ROS) and oxidative stress, both of which have a role in toxicity and carcinogenicity. Because of their extreme toxicity, these elements are listed as priority metals of major public health concern [11]. The determination of whether traffic policemen are vulnerable to traffic air pollution was made in this study by comparing plasma progesterone, testosterone, lead, copper, manganese, cadmium, and iron levels in traffic policemen designated as the exposed group and volunteers who designated as the control group. Each group was then separated into three subgroups based on age, service years, and smoking habit.

Materials and methods

Study subjects

A total of 72 individuals participated in the current study divided into two main groups: 39 individuals of traffic policemen with mean age of 31.87 ± 12.9 years and control group consisting of 33 individuals who were apparently healthy with mean age of 33.86 ± 15.9 years and enrolled in the study. They were interviewed for their medical history, duration of the work, smoking habit and drinking alcohol. The study protocol was approved by the Ethics Committee of the College of Sciences/University of Baghdad.

Exclusion Criteria

Exclusion criteria included: alcoholism, type 2 diabetes mellitus, kidney diseases, liver diseases, rheumatoid arthritis, previous bowel infection, hypertension, and the persons who used vitamins or antioxidant supplements.

Blood samples

The blood samples were collected from volunteers from various areas of the province of Baghdad, Iraq. Approximately 5 ml of blood were drawn in plane tube and left for 15 minutes at room temperature for clotting. Then, the serum was separated by centrifugation at 950 xg for 10 minutes. The serum samples were aliquoted and stored at −20◦C until use.

Determination of hormones concentration

The levels of testosterone and progesterone hormones in the blood were measured quantitatively in exposed and control groups using a competitive ELISA kit (Monobind, U.S.A). Competitive Enzyme Immunoassay is the underlying principle of this approach. In men, normal testosterone and progesterone levels is (2.5-10 ng/ml) and (0.13-1.22 ng/ml), respectively.

Determination of the concentration of heavy metals

The level of Pb, Cu, Fe, Mn and Cd in study samples were measured using Atomic Absorption spectrophotometer Shimadizu Model (AA7000).

Statistical analysis

Descriptive analysis was used to investigate exposed and control demographic data. Statistical analyses were performed using SPSS release IBM Statistic 25 for Windows. Continuous variables were tested for normality by the Kolmogorov–Smirnov test. Values were presented as mean ± standard deviation. A p value < 0.05 was considered statistically significant. Mean of each variable was compared with age, years of services and smoking habit categories. We used linear regression analysis to correlate the biochemical variables in the study group. The profile plots (interaction plots) of these means were plotted to visualize the relationships easily. The post hoc multiple comparison tests were performed for each dependent variable separately. Multivariate regression analyses were applied to access the associations among biochemical parameters (testosterone, progesterone, heavy metals (Cu, Cd, Fe, Mn, Pb) and various groupings of dependent variables. Three models were created: Model 1, adjusted for age (≤30, 31 - 44 and ≥45 years), model 2, adjusted with years of service (≤10, 11 - 23 and ≥24 years) and model 3 adjusted to smoking habit (yes or no), as shown in Figure 1.

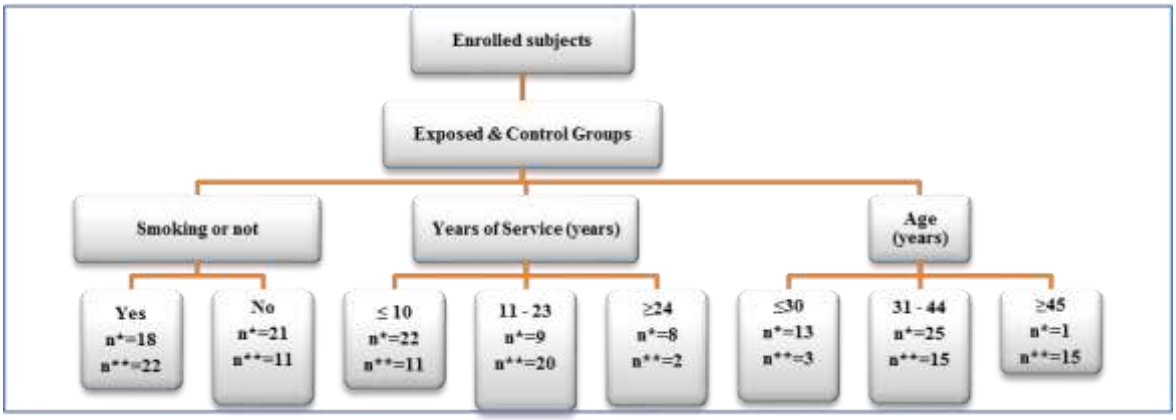


FIGURE 1 Flowchart of subjects' enrollment categorized into three models
Note: n*(control) and n** (exposed)

TABLE 1 Comparison of biochemical parameters in studied groups

Dependent Variable	ID	Mean	Std. Error	95% Confidence Interval		Sig.
				Lower Bound	Upper Bound	
BMI	Exposed	27.684	.699	26.288	29.079	.112
	Control	26.148	.650	24.850	27.445	.112
Testosterone (ng/ml) ↓	Exposed	2.532	.236	2.060	3.004	.211
	Control	2.940	.220	2.501	3.379	.211
Progesterone (ng/ml) ↑	Exposed	.439	.034	.371	.507	.098
	Control	.361	.031	.299	.424	.098
Lead (ng/L) ↓	Exposed	205.316	16.069	172.952	237.680	.814
	Control	211.146	18.670	173.542	248.750	.814
Manganese (ng/L) ↓*	Exposed	253.677	11.524	230.467	276.888	.002*
	Control	313.178	13.390	286.210	340.146	.002*
Cupper (µg/L) ↑*	Exposed	499.283	36.291	426.189	572.378	.006*
	Control	339.729	42.167	254.801	424.657	.006*
Iron (µg/L) ↓	Exposed	126.395	6.364	113.577	139.212	.199
	Control	139.124	7.394	124.231	154.017	.199
Cadmium (ng/L) ↑	Exposed	83.199	5.410	72.303	94.096	.056
	Control	66.923	6.286	54.262	79.584	.056

** The mean difference is significant at the .05 level.*

Result and discussion

Testosterone

In the exposed and control groups, the mean testosterone serum levels (ng/mL) were 2.532 ng/mL (95% CI 2.06-3.004) and 2.940 ng/mL (95% CI 2.501-3.379) respectively, as shown in Table 1. These data revealed that there was no statistically significant difference in testosterone levels between the two studied groups (exposed and control).

In mice and rats exposed to diesel exhaust, toxicological investigations found a connection between total air pollution and lower daily sperm production [12]. Some biological mechanisms have been proposed to link ambient air pollution to decreased sperm quality, including the promotion of circulating hazardous species generation through oxidative stress and inflammatory reactions [13]. Furthermore, particulate matter (PM2.5), which can easily enter the respiratory system, can carry a variety of

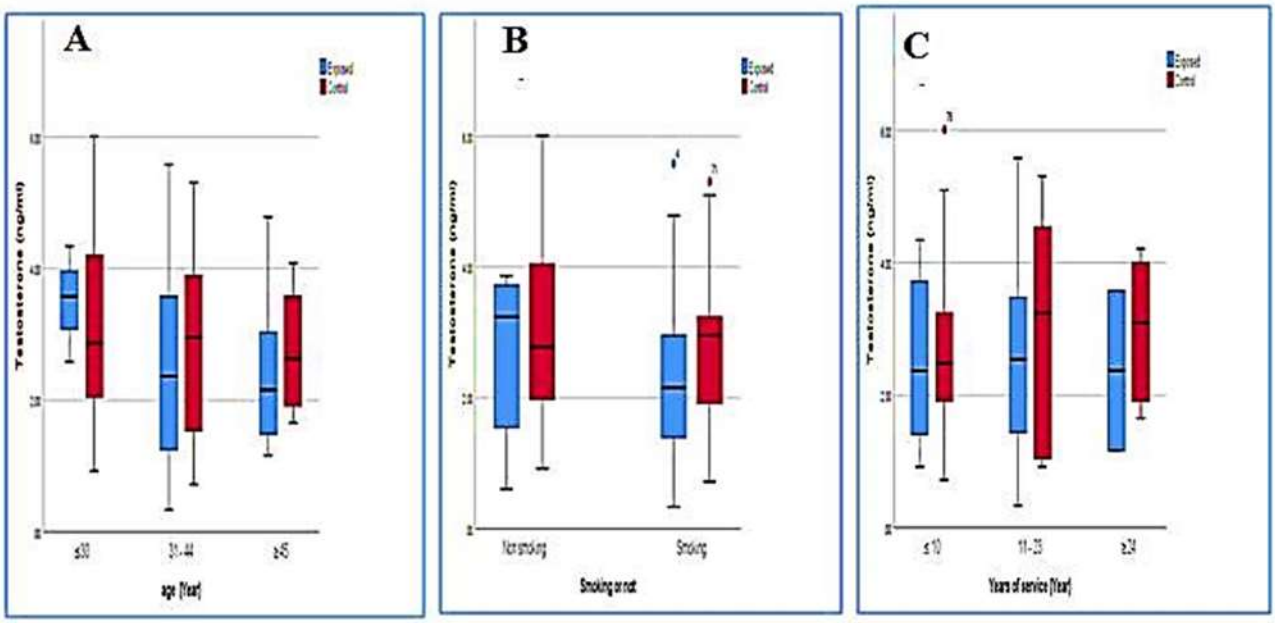
trace elements as well as polycyclic aromatic hydrocarbons (PAHs). These chemicals are endocrine disruptors that can influence the hypothalamic-pituitary-adrenal axis as well as testicular spermatogenesis, potentially causing sperm changes. Research in the Czech Republic's Teplice district looked at the impact of air pollution on male reproductive outcomes [14].

Figure 2A shows the findings of stratified analyses based on age for a population study, showing a non-significant drop in testosterone concentration in exposure categories when compared with the equivalent control group, with the exception of age less than 30 years. During adolescence and early adulthood, testosterone levels are at their highest, and the first physiological manifestations of testosterone in the body emerge during puberty. The levels of testosterone in men's bodies start to diminish as they become older [15].

When compared with the non-smoker subgroup, smoking habit effects manifest in a reduction in testosterone level; however, the drop is non-significant as can be seen in Figure 2B. Chronic smoking can disrupt endocrine homeostasis and decrease fertility in both men and women. Although the male reproductive system is more resilient, research on the effects of smoking on steroid hormone levels has produced mixed results. The impact of smoking on the male reproductive system in terms of hormonal balance has been observed to be insignificant [16]. Cigarette smoking has been linked to men's early impotence. Cigarette smoking causes sperm cell destruction, impaired sperm motility, and infertility, with smoking being the most significant risk factor [17]. The association between smoking and infertility could be related to lower testosterone levels, which could lead to a reduction in fertility.

Air pollution is one of the world's most important environmental issues. As a result, many studies have focused their efforts not only on determining the long-term health impacts of air pollution exposure [18,19], but also on determining the short-term impact of air pollution on human health [20,21]. So, our findings are applied to the years of service of traffic policemen, and it has been seen that the decline in testosterone level continues in all three groupings when compared with control groups, as shown in Figure 2C.

Short-term exposure to air pollution can result in a variety of health problems, including inflammation, coagulation abnormalities, high blood pressure, and lung damage [22]. Long-term exposure to PM10, PM10–2.5, and PM2.5 has been linked to a disruption in male reproductive hormone levels. It has been revealed that the pathophysiological routes for the effects of air pollution on male reproductive health are inflammation, oxidative stress, and epigenetic alteration [23]. According to Zhang *et al*. (2017), sulfur dioxide (SO2), particulate matter (PM), and ozone (O_3) can build in the testes and cause rat testis cell death [24].



FIHURE 2 Profile plots for marginal means of testosterone level (ng/ml) subjected to age (A), smoking habit (B) and years of service (C)

Progesterone

As indicated in Table 1, total progesterone levels among police officers exposed to vehicle exhaust were negligibly higher than that of the control group, with values of 0.439 (95% CI 0.371-0.507) and 0.361 (95% CI 0.299-0.424), respectively. Figure 3A depicts a nonsignificant drop in progesterone levels in the exposed group based on age less than 30 years, an increase in the range of 31-44 years, and more than 45 years groups. Nonetheless, a smoker's exposure subgroup shows a decrease in progesterone levels, as illustrated in Figure 3B. Figure 3C shows that the group subjected to years of service had a nonsignificant reduction in progesterone levels across all exposure categories.

Although most reference books refer to progesterone as a feminine hormone, its importance in the male endocrine system has been overlooked. Progesterone from the testes and adrenals has long been thought to be a physiologically insignificant by-product of steroidogenesis that is not converted to testosterone. However, the serum progesterone/androgen ratio rises in several conditions, including aging. The importance of progesterone as a regulator of the male endocrine system has only become more apparent in recent years [25].

Exposure to Endocrine-Disrupting Chemicals (EDCs) has been suggested to cause minor but significant changes in circulating steroid hormone levels [26].

Wang *et al*. (2021) found substantial relationships between air pollutants and male blood sex hormones, particularly serum estradiol, progesterone, and the testosterone to estradiol ratio [27] in cohort research done in China. Higher level of PM10, PM2.5, and NO_2 were linked to higher levels of estradiol and progesterone, as well as a drop in the testosterone to estradiol ratio. Previous research has revealed that air pollutants such fine particulate matter and ozone may activate the hypothalamus pituitary adrenal (HPA) axis, causing stress hormone release [28].

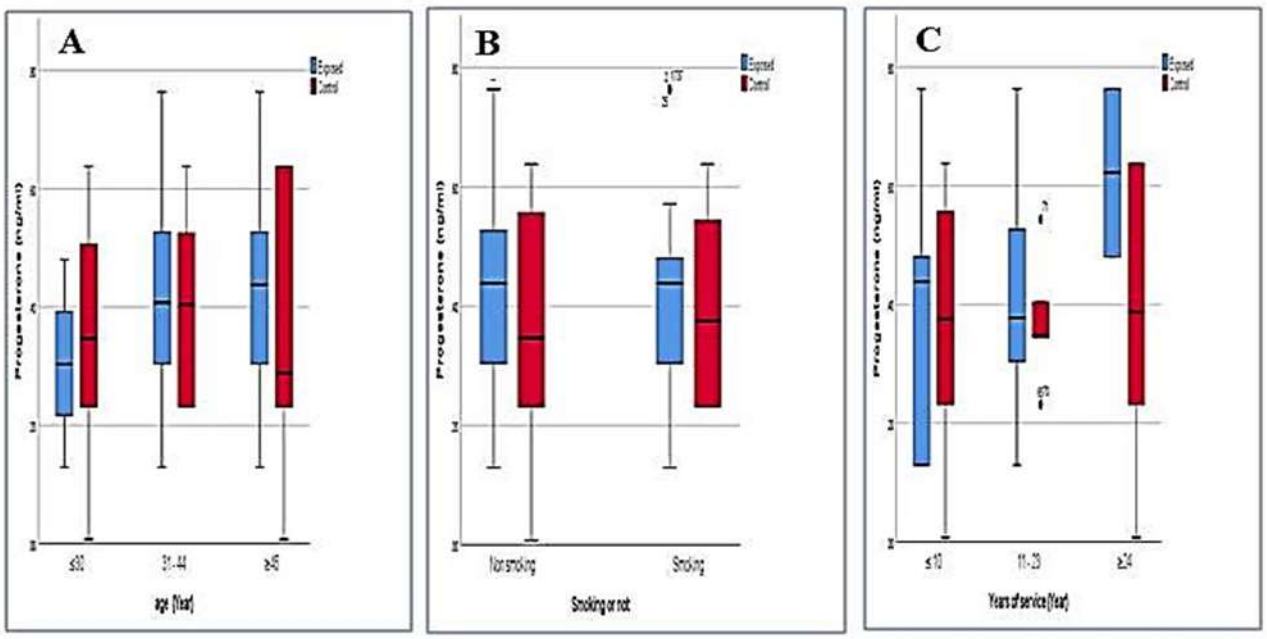


FIGURE 3 Estimated Marginal Means of progesterone level (mg/dL) subjected to age (A), smoking habit (B) and years of service (C)

Manganese

Manganese levels in traffic wardens were found to be significantly lower than those of the control group, with values of 253.67 ng/L (95% CI 230.46-276.88) and 313.17 ng/L (95% CI 286.21-340.14) respectively, as shown in Table 1.

Figure 4A shows that exposure subgroups based on age (less than 30 and 31-44) had statistically significant lower manganese concentrations than those of the control group. As seen in Figure 4B, the significant drop in smoking and non-smoking exposed groups continues in comparison to the control group. As indicated in Figure 4C, a statistically significant decrease in manganese levels was detected among traffic wardens with less than 10 years of service.

Mn(III) binds to transferrin in the plasma and enters neurons, where it dissociates and travels to axon terminals. Mn (II) has been found to penetrate into other brain regions and the cerebrospinal fluid through passive diffusion, where it then accumulates in the mitochondria of neurons, oligodendrocytes, and astrocyte cells.

A cohort Canadian study discovered a link between ambient levels of exposure to vehicle exhaust and industrial manganese emissions and the incidence of Parkinson's disease. The effects of combustion engine exhaust on bus garage employees were investigated [29]. In the short term exposure, pulmonary function reduction was thought to have little effect on individual work abilities. Furthermore, no influence on pulmonary function was detected in the occupationally exposed workers [30].

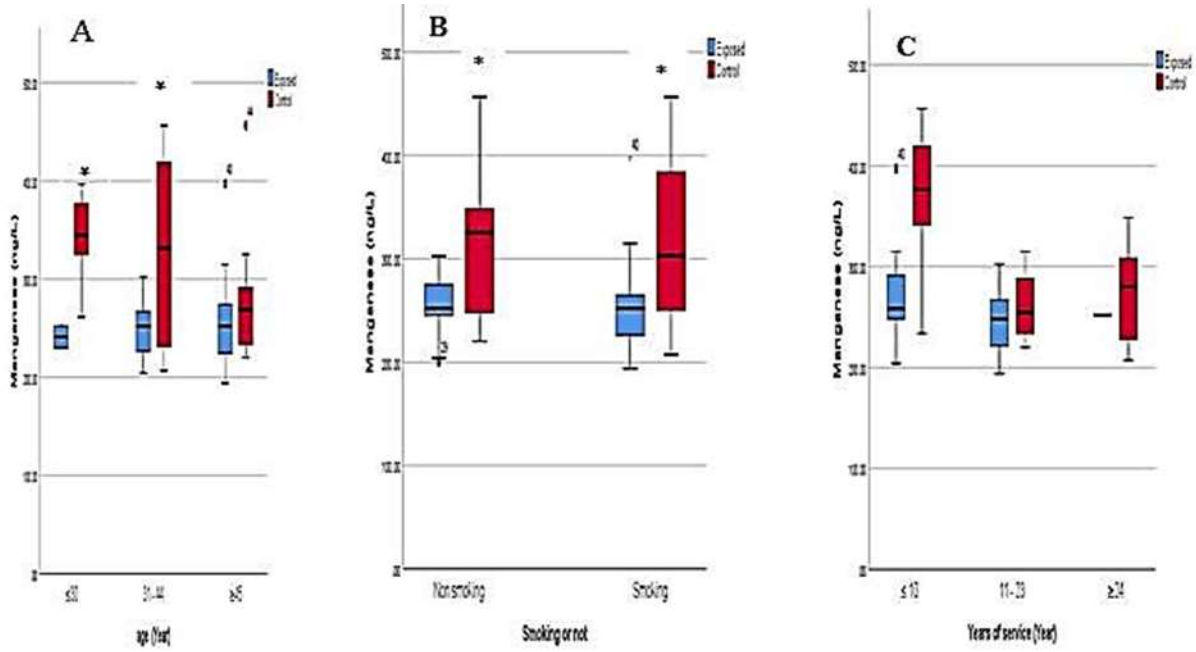


FIGURE 4 Profile plots for marginal means of Manganese levels subjected to age (A), smoking habit (B) and years of service (C)
** The mean difference is significant at the 0.05 level*

Iron

As shown in Table 1, iron levels were negligibly lower in police officers exposed to vehicle exhaust than those of the control group, with values of 126.395 µg/L (95% CI 113.57-139.21) and 139.124 µg/L (95% CI 124.23-154.01), respectively.

Figure 5 A shows a nonsignificant decrease in iron levels in the exposed groups based on age (31-44 and over 30 years), with an increase in young people (less than 30 years). Yet, as shown in Figure 5A, a smoker's exposed subgroup has a decrease in iron levels 5B. Years of service reveals no statistical differences in the exposed group compared with the control group, as shown in Figure 5C.

Diesel exhaust exposure has been shown in animal experiments to generate substantial increases in red blood cell count and hemoglobin due to hypoxia produced by high carbon monoxide levels and impaired lung function [31]. On the contrary, according to Askoy (1985) [32] and Rinsky *et al*. (1987) [33], high levels of exposure increase the incidence of anemia and leukemia in people.

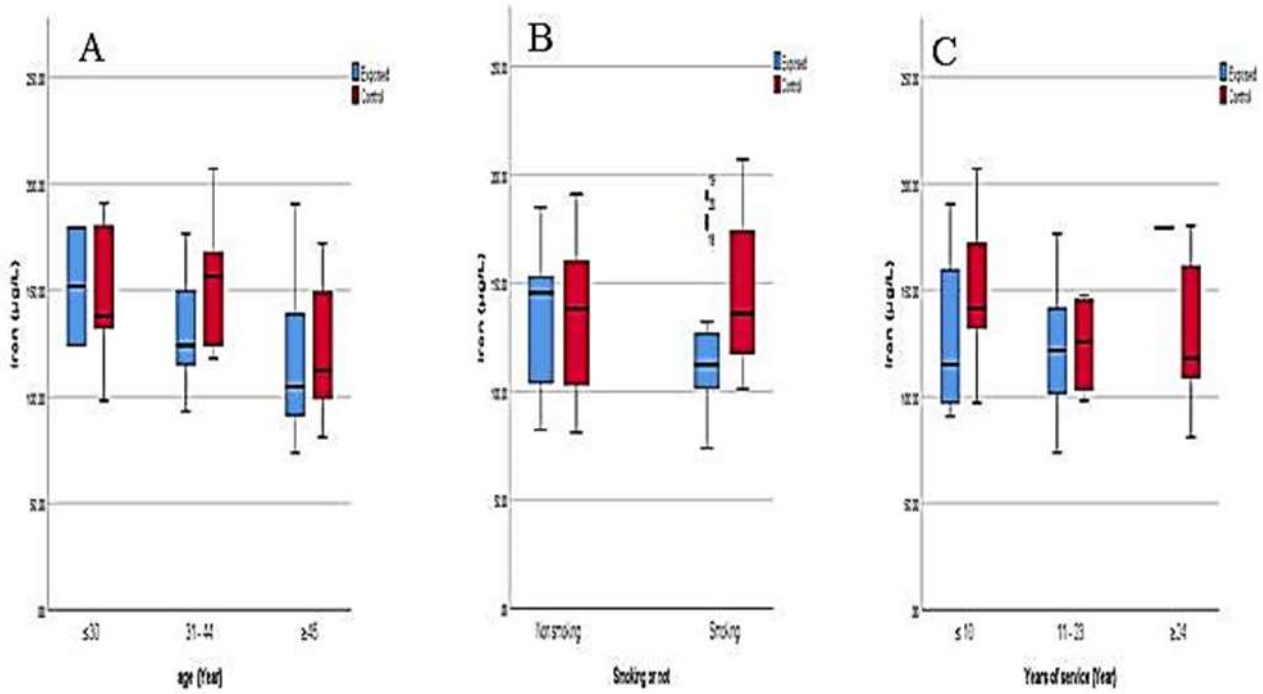


FIGURE 5 Profile plots for marginal means of Iron levels subjected to age (A), smoking habit (B) and years of service (C)

Copper

Copper levels in traffic wardens were significantly higher than in the control group, with values of 499.283 µg/L (95% CI 426.18-572.37) and 339.729 µg/L (95% CI 254.80-424.65), respectively, as shown in Table 1. Figure 6A shows that exposure subgroups based on age (less than 30 and more than 45) had statistically significant elevation in copper concentrations than that of the control group. When compared with the control group, the rise in copper levels was significant in non-smoking exposed groups and non-significant in smoking exposed groups, as shown in Figure 6B. A statistically significant increase in copper levels was found among traffic wardens with more than 24 years of service, as depicted in Figure 6C.

Copper is an essential element since it is required for the regular activity of several enzymes. Cuproenzymes, which are engaged in redox processes, can change its oxidation state, which can lead to the formation of harmful chemicals known as free radicals [34].

Many investigations have shown that oxygen free radicals and hydroxyl radicals cause DNA strand fragmentation and nitrogenous base oxidation [35].

A study conducted by Alkhatib *et. al* (2014) addressed the occupational exposure to copper in Jordanian jewelry manufacturing workers. They recorded no significant variation in the metal concentration measured in urine sample of among these workers [36].

Tobacco smoking has long been known to hasten the decrease of lung function over time, and the effect becomes more pronounced as the number of cigarettes smoked each day increases. However, given the no-smoking group's increase in copper level was comparable to the non-smoking group's increase, the potential impact on the results appears to be minor. Furthermore, a link between smoking and copper exposure (or the effect of smoking on copper exposure) is implausible [37].

Copper plasma levels were significantly higher in zinc and lead workers in Poland than those of the control group, according to Kasperczyk A. *et al*. 2012, and they were related positively with lead concentrations. They expected that as Cu levels rose, CuZn-

SOD activity would rise as well. Lead-induced oxidative stress may enhance the activity of this enzyme, which is part of the antioxidant defense system. They hypothesized that the presence of metals like Se, Zn, Cu, and Fe alters lead toxicity. A lower level of lead exposure boosts the immune system, whilst a higher level of exposure may suppress it [38].

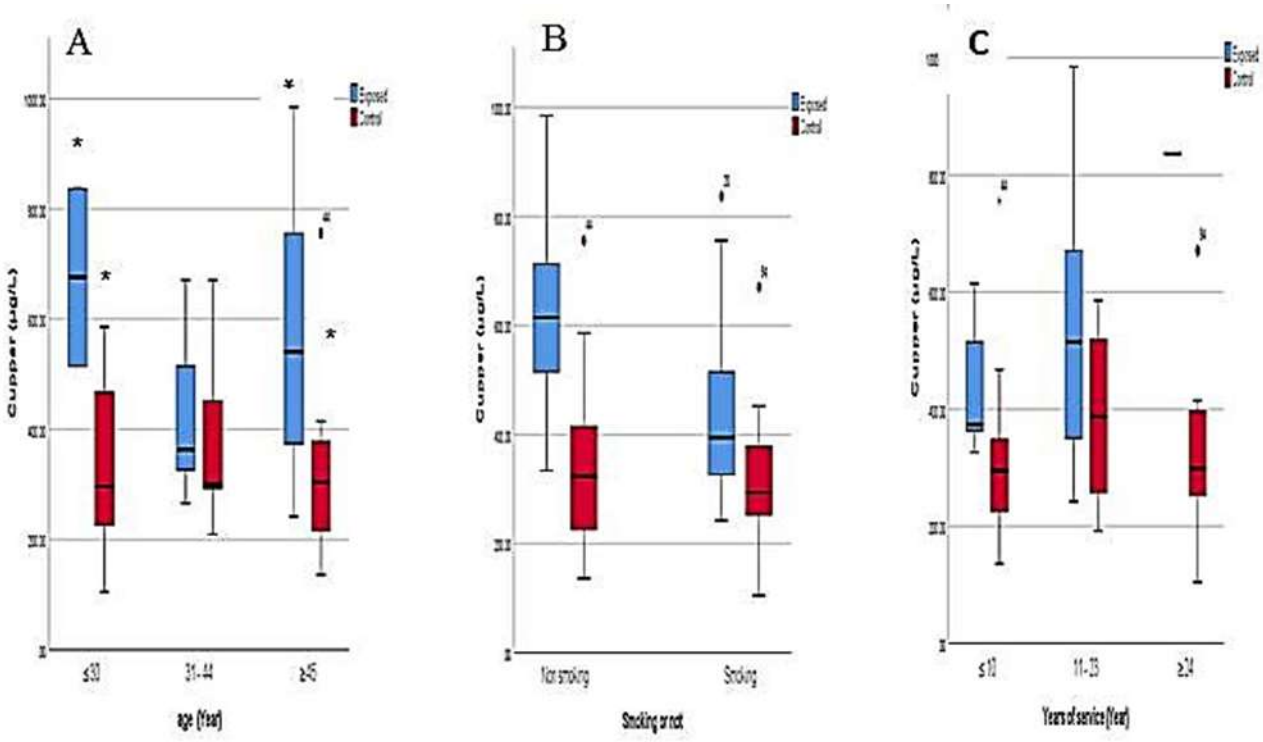


FIGURE 6 Profile plots for marginal means of Copper levels subjected to age (A), smoking habit (B) and years of service (C)
** The mean difference is significant at the 0.05 level*

Cadmium

The mean cadmium levels in the exposed group were 83.199 ng/L (95% CI 72.303-94.096) and 66.923 ng/L (95% CI 54.26-79.58) in the control groups, as indicated in Table 1. Figure 7A depicts the results of age-based stratified analyses for a population study, demonstrating a significant increase in cadmium concentration in the exposure category (31-44 year) when compared with the equivalent control group, whereas the increase was nonsignificant in the other two subsets (less than 30 and more than 45). Smoking habits cause an increase in cadmium content, but the increase is nonsignificant, as shown in Figure 7B. Figure 7C shows that the increase in cadmium level continues in groups subdivided by years of service.

Cadmium (Cd) is a very poisonous and carcinogenic metal that is extensively distributed in nature. Human exposure to Cd can come from a variety of sources, including nutrition, drinking water, occupational exposures, and cigarette use [39]. Smoking is a substantial source of Cd exposure, and it has been shown that smoking can dramatically raise Cd levels in the human body [40]. A study by Kresovich *et. al.* (2015) aimed to analyze the blood levels of cadmium and lead in occupationally exposed individuals aged 15–40 years, they found that the blood levels of cadmium and lead were significantly higher compared with the control group, which may increase the exposed individuals' health risk [41].

A cross-sectional study by Ciarrocca *et al.,* 2013 [42] was conducted to assess the relationship between exposure to cadmium and circulating reproductive hormone levels in urban and rural male workers. The researcher discovered that the mean value of

cadmium exposure was higher among traffic policemen than that of roadmen [43].

Cadmium has been linked to the promotion of apoptosis, oxidative stress, DNA methylation, and DNA damage, all of which have been linked to malignancies of the kidney, lung, pancreatic, and prostate [44].

Cadmium is a highly hazardous metal that can disrupt a variety of biological systems at concentrations that are typically much lower than those found in most toxic metals [42]. Because of its capacity to bind to androgen and estrogen receptors, Cd has been classified as an endocrine disruptor because of its negative effects on reproduction, disruption of steroidogenesis, and spermatogenesis in vivo and in laboratory animals. Cd exposure has been linked in clinical investigations to prostate toxicity, testicular toxicity, and infertility. Cd may have a negative impact on the male reproductive system by lowering the quality of sperm. Several human investigations have found a favorable relationship between Cd and testosterone, depending on the route, dose, and period of exposure [45].

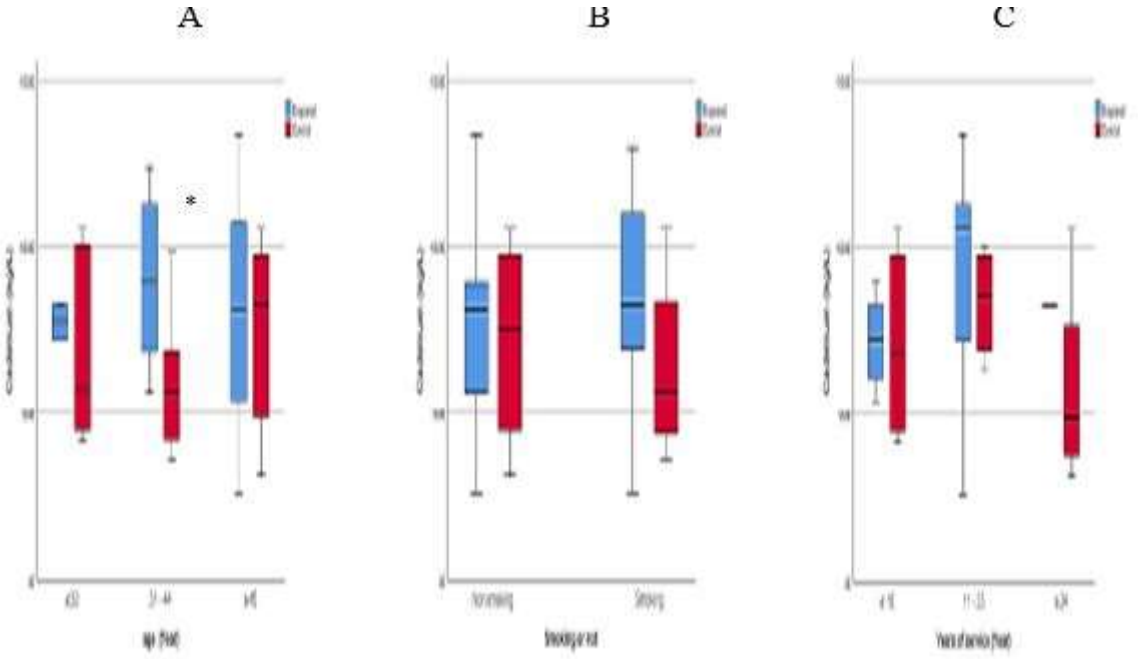


FIGURE 7 Profile plots for marginal means of Cadmium levels subjected to age (A), smoking habit (B) and years of service (C)

** The mean difference is significant at the 0.05 level*

Lead

Lead levels among police officers were found to be nonsignificant lower than those in the control group, with values of 205.316 ng/L (95% CI 172.95-237.68) and 211.146 ng/L (95% CI 173.54-248.75) respectively, as shown in Table (3-2). When compared with the control subgroup, exposure subgroups in relation to age and years of service had statistically nonsignificant differences in lead concentrations, according to Figures 8 A & C. There was a nonsignificant variation in smoking exposed groups in comparison to control subgroup, as shown in Figure 8B.

Chronic lead poisoning elevates blood pressure and has hormonal and neurological consequences. Lead interacts with a variety of proteins in renal cells, causing lead nephrotoxicity. Exposure to lead also produces oxidative stress, which results in metabolic dysfunction, cellular degeneration, and transport impairments [46]. According to an Egyptian study conducted by Kamal *et al.* (1990), the levels of lead in traffic policemen were greater than those of the control group.

They also found no link between blood lead levels and age or period of employment. A group of researchers in Sudan's Khartoum State discovered no significant differences in lead contents between the exposed (traffic policemen) and control groups depending on age or time of employment [47].

Pala *et al*., (2002) revealed that there was no significant difference in blood lead levels between indoor and outdoor working policemen, and that the differences were also non-significant when smoking habits were taken into account. These findings back up our findings [48].

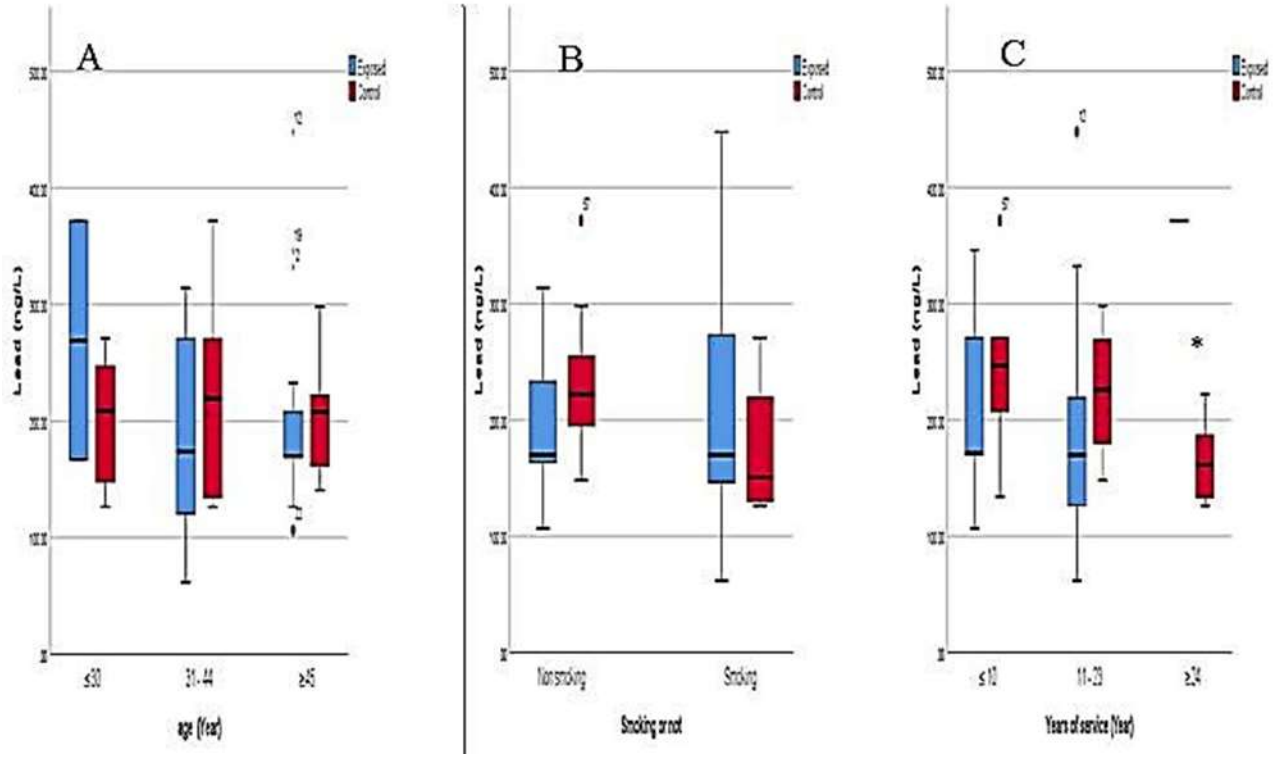


FIGURE 8 Profile plots for marginal means of lead levels (ng/L) subjected to age (A), smoking habit (B) and years of service (C)

Conclusion

One potential source of health damage appears to be exposure to dangerous compounds in the environment, notably exposure to car exhaust emissions, as experienced by traffic wardens. Although the current investigation found no significant changes in sex hormone levels (testosterone and progesterone) in groups exposed to diesel exhaust, a decline in testosterone level was observed. According to existing statistics, exposure to heavy metals and other contaminants in major urban centers' transportation infrastructure can result in endocrine disrupting chemicals that can impede hormone production, metabolism, and transport. Long-term exposure to particulate matter (PM) has been associated with changes in male reproductive hormone levels. Furthermore, particulate matter (PM2.5), which is easily inhaled, can carry a range of trace elements as well as polycyclic aromatic hydrocarbons.

Heavy metals cause direct structural changes in biomolecules or cause indirect damage by the production of reactive oxygen and nitrogen species such as hydroxyl and superoxide radicals, hydrogen peroxide, nitric oxide, and other endogenous oxidants. Cadmium levels in the exposed group increased significantly, which could contribute to endocrine disruption. Cadmium may have deleterious effects on the male reproductive system due to its ability to bind to androgen and estrogen receptors. Because

of its capacity to drive DNA strands to break and for bases to oxidize via oxygen free radicals and hydroxyl radicals, high levels of copper in the sera of traffic policemen may be associated with increased oxidative stress. Although non-significant decrease in iron level was recorded in the present work, diesel exhaust exposure might increase the incidence of anemia in study subjects. According to the findings, traffic policemen are particularly sensitive to general health problems as a result of automotive emissions in the workplace. The study's ability to identify extremely minor changes in the given biomarkers' measures associated with exposure could have been hampered by the study's small sample size, which would diminish the study's capacity to detect very small changes in the given biomarkers' measures associated with exposure.

Optimizing the architecture and power of urban transportation networks, promoting clean energy automobiles, reducing vehicle emissions, and managing the accumulation of certain materials and heavy metal elements may all require further work.

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