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Association of Traffic-Related Air Pollution with DNA Damage and Some Other Biological Parameters among Minibuses Drivers in Baghdad City

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Abstract

Fifty blood samples were collected from minibus drivers aged 18-50 years a minimum driving experience of 2 years. The control group involved fifty age matched men who lived in other areas in Baghdad. The results illustrated that the level of both superoxide dismutase and glutathione enzymes were significantly decreased in the drivers group (9.87 ±0.32 µg/ml and 17.73 ±0.41 mg/ml respectively) as compared to the control group (38.00 ±1.78 µg/ml and64.92 ±2.04 mg/ml respectively). The level of catalase enzyme showed a highly significant increase in the drivers group (327.76 ±11.77 µg/ml) as compared to the control group (93.98 ±2.67 µg/ml). The results also showed a highly significant increase in the level of serum tumor necrosis factor alpha in the drivers group (332.17 ±11.84 pg/ml) as compared to the control (52.75 ±2.72 pg/ml). DNA damage showed a significant increase in the drivers group (4.51 ±0.15 pg/ml) as compared to the control group(1.20 ±0.04 pg/ml).

Keywords : Antioxidant, pollution of the air, 8-hydroxy-2'-deoxyguanosine, TNF alpha

العلاقة بين تلوث الهواء المرتبط بالمرور مع تلف الدنا وبعض المؤشرات البيولوجية الاخرى بين سائقي الباصات الصغيرة في بغداد

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الخلاصة

هدفت هذه الدراسة إلى تقييم تأثير تلوث الهواء المصاحب لحركة المرور على بعض المعايير البيولوجية لدى السائقين في مدينة بغداد. تم جمع 50 عينة
دم من السائقين الذين تراوحت أعمارهم بين 18–50 سنة والذين يقودون لمدة سنتين على الأقل. بينما تم جمع مجموعة السيطرة من 50 رجلاً يعيشون
في منطقة أخرى في بغداد ولهم نفس عمر السائقين. أوضحت النتائج انخفاض كل من إنزيم ديسموتاز الفائق أكسيد والجلوتاثيون بشكل ملحوظ في السائقين (9.87 ±0.32 ميكروغرام/مل و17.73 ±0.41 ملغم/مل) مقارنة بمجموعة السيطرة (38.00 ±1.78 ميكروغرام/مل و64.92 ±2.04 ملغم/مل). بينما أظهرت نتيجة إنزيم الكتليز زيادة معنوية عالية في السائقين (327.76 ±11.77 ميكروغرام/مل) مقارنة بمجموعة السيطرة

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(93.98 ±2.67 ميكروغرام/مل). كما أظهرت النتائج زيادة معنوية عالية في مستويات عامل نخر الورم ألفا لدى السائقين (332.17 ±11.84 بيكوغرام/مل) مقارنة بمجموعة السيطرة (52.75 ±2.72 بيكوغرام/مل). أظهر تلف الحمض النووي زيادة معنوية عند التركيز العالي في السائقين (4.51 ±0.15 بيكوغرام/مل) مقارنة بمجموعة السيطرة (1.20 ±0.04 بيكوغرام/مل).

Introduction

Chemical elements involved in the traffic-related air pollution are known to be potent mutagens and carcinogens, causing a growing concern about this problem and exposure to ambient air pollution has been linked to a number of different health effects [1,2]. Urban ambient air pollution is the result of emissions from a multiplicity of sources, mainly stationary, industrial, and domestic fossil fuel combustion and petrol and diesel vehicle emissions [3]. Oxidized guanine bases are present in blood cells at high amounts, as well as in the urine of those who are exposed to vehicle pollutants in city air. According to a body of research, being exposed to air pollution caused by traffic is linked to DNA oxidation, which may raise the chance of developing cancer [4]. Enzyme activities of glutathione peroxidase, superoxide dismutase, and catalase in the blood was recently found to be strongly, but inversely, associated with the risk of developing coronary artery disease. High levels of oxidized DNA in blood cells were linked to exposure to pollution in several cross-sectional investigations[5].

Since 8-OHdG is known to represent one of the primary forms of oxidative DNA damage, several studies have evaluated it in tissues or urine as a marker of beneficial oxidative stress [6]. A G-T transversion mutation may occur when 8-OHdG erroneously binds with adenine rather than cytosine during DNA replication. As a result, 8-OHdG is premutagenic[7]. Since previous research demonstrated a significant correlation between short-term exposure to particle matter size 10 and circulating TNF- levels, tumor necrosis factor alpha (TNF-) has a variety of activities, including synergistic effects in inflammatory and immunological responses [8]. Another study [9] revealed that TNF- levels began to rise relatively late, or 24 hours after exposure to diesel exhaust particles. This study aimed to evaluate the impact of air pollution associated with traffic on some biological parameters among minibus drivers in Baghdad city.

Materials and methods

Specimens collection

Blood samples were collected from the drivers and healthy controls via the venipuncture method . A volume of 5ml of blood was taken with disposable syringe sand divided into two aliquots. Sera were separated from the first aliquot (3 ml) by centrifugation for 5 minutes at 3000 revolutions per minute (rpm), placed into Eppendorf tubes, and maintained at -20°C. Two milliliters of the second aliquot were transferred to EDTA tubes for CBC analysis by hematology analyzers. The ethical approval statement, Ref**.:** CSEC/0922/0114, was issued on the 30 September, 2022, by The Ethics Committee/ Department of Biology/ College of Science, University of Baghdad.

Hematological and biochemical analyses

Regarding hematological and biochemical studies serum samples were collected from 50 healthy drivers with 18 to 50 years age range and a minimum of 2 years of driving experience. The control group from uncrowded areas in Baghdad. As antioxidant parameters, we included catalase which is found in all aerobic organisms [10], glutathione which is one of the most extensively investigated antioxidants at the present time [11], and superoxide dismutases since all kingdoms of life contain the group of metalloenzymes known as superoxide dismutases (SODs) [12]. Tumor necrosis factor alpha (TNF-alpha) was investigated since it is known as a

key regulator of inflammatory responses [13]. Hydroxy-2′- deoxyguanosine (8-OhdG) is particularly a ROS-induced DNA base modification followed the attack of hydroxyl radicals (OH) to guanine which causes damage to the DNA [14]. An ELISA device was used to determine the serum levels of these factors using a diagnostic kit from Cloud-coper (USA).

DNA damage assessment

A volume of 50µL of the sample, blank, and standard solution was placed in respective wells of a plate. Then, 50µL of the Detection Reagent A were added to each well immediately. Then, the plate was shaken gently, covered with a plate sealer, and incubated for 1hour at 37 ^{o}C. The solution was warmed to room temperature and mixed gently until appeared uniformed. The solution was aspirated and washed with 350µL of 1X Wash Solution in each well using a multi-channel pipette and kept still for 1-2 minutes. The remaining liquid was completely removed from all wells by snapping the plate onto absorbent paper. The process was repeated 3 times. The remaining Wash Buffer was removed by aspiration or decanting, followed by the inversion of the plate against absorbent paper.

A volume of 100µL of the Detection Reagent B working solution was added to each well, followed by incubation of the plate for 30 minutes at 37 C^{o} after covering it with plate sealer. The aspiration/wash process was then repeated for 5 times. The Substrate Solution (90µL) was added to each well, then the plate was sealed and incubated for 10- 20 minutes at 37 ^{o}C away from light. The solution was observed to turn blue after the addition of the Substrate Solution. Stop Solution (50µL) was then added to each well, leading to a yellow color of the solution, which was mixed by tapping the side of the plate. The microplate reader was immediately used for the measurement at 450 nm.

Statistical analysis

The influence of different groups (drivers and control) on research parameters was discovered using the Statistical Analysis System- SAS (2018) tool. To significantly compare between means, the T-test was utilized. The correlation coefficient between the study's variables was estimated.

Results and discussion

Hematological parameters

Hematological characteristics are thought of as biomarkers of chemical substance exposure, among other biological alterations, since they can cause changes in a variety of hematological components[15]. Table 1 lists the results of the analyses of all hematological parameters adopted in this investigation.

According to the current study, the average red blood cell (RBC) count was 4.94 ±0.06 x10^{6} /mm^{3} in drivers and 4.64 ±0.08 x10^{6} /mm^{3} in controls, white blood cells (WBC) count was 7.71 ±0.10 x10^{3} /mm^{3} in drivers and 6.51 ±0.12x10^{3} /mm^{3} in control, and platelet count was 271.12 ±6.68x10^{3}/mm^{3} in drivers and 252.52 ±4.79 x10^{3}/mm^{3} in control, with non-significant differences between the two groups in all these parameters. Also, non-significant differences were recorded in the parameters of hematocrit percentage, mean corpuscular volume, mean corpuscular hemoglobin, mean corpuscular hemoglobin concentration, and hemoglobin

The reason for the increase of some blood parameters in drivers compared to the control may be due to the fact of their being exposed to airborne particles and other contaminants resulting from crowded traffic in Baghdad streets. The mean value of granulocyte count was significantly (P≤0.01) increased in drivers (65.39 ±0.66 %) compared to control (54.80 ±0.65

%) but the overall lymphocyte and monocyte counts in the two groups did not differ significantly, as shown in table 2. The findings presented here also demonstrate that the circulating granulocyte count increased, probably as a result of the systemic reaction to inhaling air pollutants with high particle sizes. These may have resulted from the bone marrow releasing WBC and their precursors in response to the particle deposition in the lung. These cells comprise between 2 and 6 % of the WBCs that circulate normally, and if their number rises, it means that the bone marrow has been prompted to produce more granulocyte [16].

Table 1: Mean ± standard error of several blood metrics in the drivers as well as the control group

Group	Mean ± SE							
	WBC*10^3 /mm^3	RBC*10^6 /mm^3	HGB (g/dl)	HCT (%)	MCV (μm^3)	MCH (pg)	MCHC (g/dl)	PLT* 10^3/mm^3
Drivers	7.71 ±0.10	4.94 ±0.06	14.53 ±0.05	41.58 ±0.30	91.02 ±0.40	29.85 ±0.19	32.86 ±0.08	271.12 ±6.68
Control	6.51 ±0.12	4.64 ±0.08	14.28 ±0.05	40.98 ±0.42	89.41 ±0.50	28.47 ±0.21	32.49 ±0.10	252.52 ±4.79
T-test	0.343 **	0.217 **	0.181 **	1.056 NS	1.340 *	0.626 **	0.285 **	20.061 NS
P-value	0.0001	0.0075	0.0077	0.261	0.0197	0.0001	0.010	0.0687
* (P≤0.05), ** (P≤0.01).								

Table 2: Mean ± standard error of white blood cells (WBC) of drivers and control group

Group	Mean ± SE		
	Lymphocyte (%)	Monocyte (%)	Granulocyte (%)
Drivers	30.38 ±0.70	4.21 ±0.07	65.39 ±0.66
Control	29.20 ±0.78	4.12 ±0.11	54.80 ±0.65
T-test	2.278 NS	0.262 NS	2.080 **
P-value	0.3038	0.4682	0.0001
** (P≤0.01).			

Biochemical parameters

Enzymes are physiological and biochemical compounds that can serve as indicators to identify potential physiological hazards in certain species that might occur due to environmental pollution [17]. Table 3 contains a list of the results obtained for all biochemical parameters investigated throughout this study. The pollutants known as volatile organic compounds (VOCs), which are connected to gasoline vapour emissions and vehicle exhaust, can cause an oxidant-antioxidant imbalance by increasing intracellular reactive oxygen species (ROS) levels and decreasing antioxidant capacity which is known as oxidative stress (OS) [18].

The present results showed that the mean serum level of glutathione (GSH) was significantly ($P \leq 0.01$) decreased in drivers (17.73 ±0.41mg/ml) as compared to control (64.92 ±2.04 mg/ml). The conclusion that the decrease in GSH is due to traffic-related air pollution is supported by Dianat *et al*. [19], who discovered that after being exposed to particulate matter of size 10, the levels of antioxidant enzymes such as superoxide dismutase, catalase, and glutathione peroxide significantly lowered. Serum level of catalase had a highly significant increase in drivers (327.76 ±11.77 µg/ml) compared to control (93.98 ±2.67 µg/ml). The association between the rise in CAT in drivers and air pollution caused by traffic was also

reported by Wenten et al. [20], who reported that youngsters exposed to high levels of ambient air pollution showed clear evidence of the interaction between the genes for catalase and myeloperoxidase. Serum level of SOD was significantly ($P \leq 0.01$) decreased in drivers (9.87 ±0.32 µg/ml) compared to control (38.00 ±1.78 µg/ml). These results agree with those reported by Sreenivasan *et al*. [21] who found that the levels of superoxide dismutase and glutathione peroxidase, two antioxidant enzymes, were drastically reduced, while that of catalase increased in Indian workers engaged in construction. The decrease in SOD in the drivers group was possibly caused by the accumulation of ROS in the blood.

Immunological and molecular parameters

TNF-alpha, according to Bradley J.R. et al. [13], is recognized as an important regulator of autoimmune responses and is connected to the genesis of a number of inflammatory and autoimmune illnesses. TNF- is primarily generated by natural killer cells, T-lymphocytes, and activated macrophages and is a homotrimer protein with 157 amino acids, according to its structural composition [22]. The present results showed that the mean serum level of TNF-α was highly significantly ($P \leq 0.01$) increased in drivers (332.17 ±11.84 pg/ml) compared to control (52.75 ±2.72 pg/ml). When exposed to air pollution from traffic, immune cells in drivers' lungs, such as alveolar macrophages, produce TNF. This finding was supported by El-Sharkawy and Ahmed's [23] investigation, which discovered that particle exposure to the pulmonary bronchial tree may cause a local inflammatory response with the production of certain cytokines by macrophages, neutrophils, and T-cells. Weidong et al.'s [24] study found that exposure to particle material, whether short-term or long-term, raises the concentrations of cellular and inflammatory mediators in the blood, including IL-1b, IL-6, IL-8, IFN-g, C-reactive protein, TNF-a, and fibrinogen, along with higher white blood cells count.

Several traffic-related air pollution (TRAP) components, including particulate matter (PM), black carbon (BC), ozone (O3), nitrogen oxides (NOx), and polyaromatic hydrocarbons (PAHs), have been associated with changes in DNAm; typically lowering DNAm after exposure. Effects of air pollution on DNAm have been observed across the human lifespan [25]. The damage caused to DNA by ROS has sparked profound interest in the medical world due to their involvement in various pathological conditions. 8-OHdG is an oxidized nucleoside excreted in the body as a reparative consequence of damage to DNA [26]. According to the National Center for Biotechnology Information [27], 8-hydroxy-2'-deoxyguanosine, the main spontaneously oxidized byproduct of 2'-deoxyguanosine, is recognized as an indicator for DNA oxidative damage. Guanine and reactive oxygen species react to generate 8-hydroxy-2'-deoxyguanosine. Additionally, 8-hydroxy-2'-deoxyguanosine (8-OHdG), which has been widely used as a biomarker for oxidative stress and carcinogenesis [28], is one of the most prevalent forms of free radical-induced oxidative lesions.

According to our study's findings, there has been a considerable rise ($p<0.01$) in the serum levels of 8-OHdG in drivers (4.51 ±0.15 pg/ml) when compared to the controls (1.20 ±0.04 pg/ml). According to Alessandro Di Minno *et al*. [29], who discovered that the measurement of stable metabolites of oxidative reactions provides a more accurate measure of the degree of oxidative stress due to the significant instability of reactive oxygen species, the number of drivers has increased due to exposure to air pollution and higher levels of reactive oxygen species. Systemic oxidative stress that occurs in vivo is shown by the biomarker 8-OHdG, which is produced by oxidative DNA damage, as showing in Figure 1.

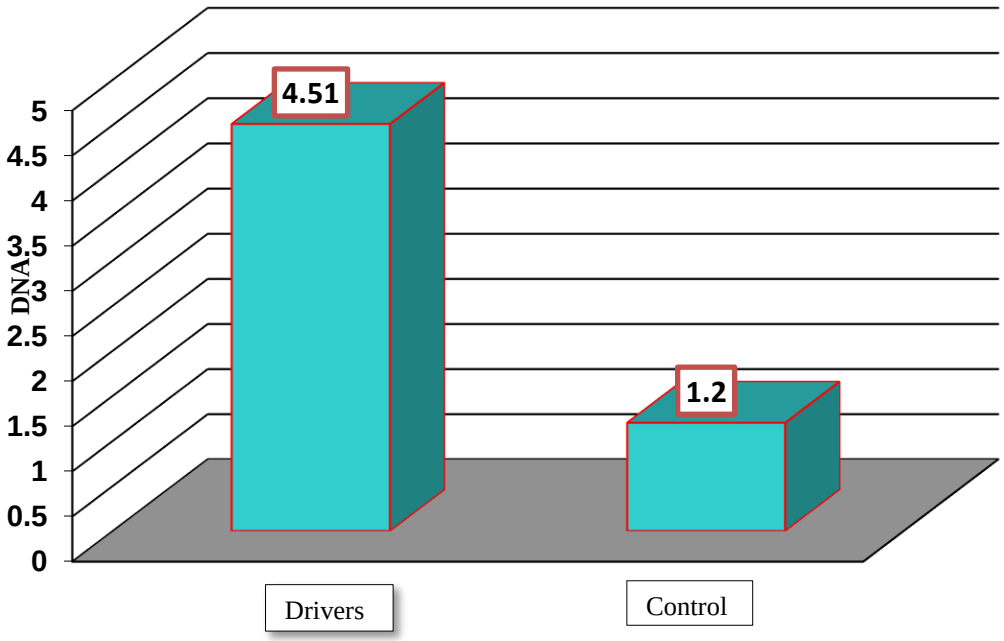

Figure 1: Comparison between drivers and control groups in DNA

Table 3: Mean ± standard Err values of immunological, biochemical and molecular parameters in the driver and control groups.

Group	Mean ± SE				
	GSH (mg/ml)	CAT (µg/ml)	TNF-α (pg/ml)	SOD ((µg/ml)	8-OHdG (pg/ml)
Drivers	17.73 ±0.41	327.76 ±11.77	332.17 ±11.84	9.87 ±0.32	4.51 ±0.15
Control	64.92 ±2.04	93.98 ±2.67	52.75 ±2.72	38.00 ±1.78	1.20 ±0.04
T-test	3.100 **	33.491 **	33.721 **	2662 **	0.439 **
P-value	0.0001	0.0001	0.0001	0.0001	0.0001
** (P≤0.01).					

Conclusions

The present study concludes that air pollution from traffic exposure leads to significant alterations in biological responses in human body. The information gathered here suggests that drivers' presence in areas with air pollution from traffic may combat hazardous impacts to their health. The hematological profiles of the drivers showed a non-significant increase in the values of WBC, MCV, PCV, MCH, RBC, and Hb, as well as an unremarkable decrease in MCHC. The statistical analysis indicated that the biochemical parameters like glutathione and superoxide dismutase had a significant decrease in the drivers as compared to control. However, the levels of catalase, 8-hydroxy-2'-deoxyguanosine (which is regarded as an indicator to DNA oxidation damage), and tumor necrosis factor-*a* revealed a significant increase in the drivers when compared to the control group. Thus, the traffic-related air pollution has a remarkable effect on human life and activity, especially for those who use driving as a source of livelihood. Hence, it is necessary to reduce its effects by increasing plant cultivation and decreasing the number of cars.

Conflicts of Interest: Authors have no conflicts of interest to declare.

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