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Altered Hematological Parameters and Damaged DNA in Traffic Workers Exposed to Vehicle Emissions

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ABSTRACT

Background: *Vehicle emissions are known for its harmful effect on the environment and public health in term of carrying different pollutants. Traffic police workers are among those who in continues exposure to these emissions and for lung durations.* ***Material and methods:*** *Fifteen police workers were recruited in this study where haematological and DNA alterations were investigated and compared to a control group.* ***Results:*** *The results clearly stated that exposure to vehicular emissions bring alterations to at least one or more of blood parameters such as RBCs, Hgb, WBCs, MCV and MCH. DNA damage; estimated by commit essay, revealed significant abnormalities in the DNA of exposed subjects.* ***Conclusion:*** *Awareness has to be raised among workers to reduce the effect of duty exposure and finding sustainable and clean sources of energy is a priority for people in charge.*

Keywords: Emissions, DNA, Traffic police, and Pollution.

Article Information

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INTRODUCTION

Traffic workers usually work in an environment where they are subjected to high levels of vehicular emissions which are considered major environmental pollutants. The use of fossil fuel for transportation contributes to the release of these emissions which usually carry various solid and gaseous pollutants such as particulate matter (PM), volatile organic compounds (VOCs), heavy metals and polycyclic aromatic hydrocarbons (PAHs). (1) and (2).

Researchers raise the concern about the levels of these toxic substances that potentially dangerous environmentally and to human health. Oxidative stress is one of the implications that play a role in the mechanismof the adverse health effect related to vehicles emissions. Many biomarkers have been recently used to evaluate that effect. For example, urinary 8-OHdG is a product produced from damaged DNA and is easily used to determine any harmful effect of emissions on DNA (3). The damage could include increase in 8-hydroxy-20-deoxyguanosine, tail DNA, micronucleus frequency and a decrease in glutathione (4). Moreover, the emissions were identified to have an effect on platelets activity, blood parameters and stimulating systematic inflammation (5).

The aim of the current study is to investigate the haematological abnormalities and DNA damage in police traffic workers exposed to exhaust emissions. This study can give evidence of linking occupational exposure to emissions with cellular and genetic toxicity. The results of this study could build a protective measurements and mitigating health risks in vulnerable people.

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METHODS

This cross- sectional study was conducted in Najaf city, Iraq which has a population of nearly 1.5 million and more than 100,000 registered vehicles. A research facilitation letter was provided by our institute (Ref. No. 1784 in 09/05/2023) and directed to the Traffic Directorate of Najaf Governorate.

The study included 30 Male participants who were recruited as two groups according to their exposure to traffic related air pollutant (TRAP), fifteen participants were classified as "exposed" who were traffic police workers (Age mean= 31.92±10.05) while the other rest were control (non-exposed indoor workers, age mean= 32.07±9.44), table 1. A Consent was obtained for each participant and a questionnaire was filled in accordingly. The smoking status was determined for each group. 5 mL venous blood was collected; 3 mL was aliquoted into EDTA tubes while the remaining 2 mL was aliquoted in gel serum tube. All samples were kept in wet icebox while transferring to the lab. Complete blood count using haematology autoanalyzer (SWELAB, Boule Diagnostics, Sweden) was run for all EDTA samples then the samples were kept in -80 fridge for comet assay analysis. Lymphocytes were isolated according to the protocols followed by Morres P.J, and Ting A in their paper (6).

DNA damage was estimated in 75 cells of each individual by running comet assay test (OxiSelect™ Comet Assay Kit (3-Well Slides), Cell Biolabs, Inc., USA) following manufacturer's instructions. The tail length of DNA was selected according to the shape and size. Ethidium Bromide was replaced by vista green satin for better results. C-reactive protein (agglutination method, Spinreact, Spain) was run for all gel tube samples following manufacturer's instructions. P-value of <0.05 was considered significant.

Table 1: Demographic characteristics of subjects.

Characteristics	Workers n=15	Control n=15	p-value
Age (year)	31.92±10.05	32.07±9.44	0.734
Weight (Kg)	84.9±12.35	80.8±9.13	0.239
Height (m)	1.73±0.06	1.69±1.55	0.589
BMI (Kg/m^2)	28.2±5.68	26.9±2.69	0.093

RESULTS

The haematological differences among different groups were determined. These included red blood cells count (RBCs), Haemoglobin (Hb), Mean corpuscular Haemoglobin (MCH), mean corpuscular volume (MCV) and white blood cells count (WBCs). The results were interpreted as mean ± standard deviation (SD).

The first comparison was between exposed smoke group (n=9) and control smoke group (n=6) using T-test which showed a significant difference (p.value <0.05) for all parameters except for MCV and MCH which were not affected. The mean value of RBCs for exposed smoke group revealed erythrocytosis (7.89±1.71 $x10^{12}$/L). The same comparison was done for exposed non-smoke group (n=6) and control non-smoke group (n=9), where significant difference was shown in RBCs and MCH only. Here again, erythrocytosis appeared clearly in the exposed non-smoke individuals (6.77 ±1.01). The DNA damage; estimated in 75 cells per individual, of the same groups was significantly high (p. value <0.01) as seen in table 2.

Most of the cells of exposed smoke group showed medium damage (20.78±8.18) and low damage (19±6.32) comparing to (4.83±1.94) and (6.83±3.06) in control smoke group, respectively. Almost five cells (4.89±1.9) comparing to nearly two cells (1.83±0.75) revealed high DNA damage in both groups, respectively. About 30 cells (30.33±13.26) of

exposed smoke group showed no damage compared to almost 62 cells (61.5±4.09) in control smoke group. The median tail length of exposed smoke group was (18.02±5.36 µm) compared to (4±0.8µm) in control smoke group. The difference in the severity of damage between these two groups was significantly high (p. value <0.01), as seen in Table 2. For exposed non-smoke and control non-smoke groups, most of the damage was low (25.33±7.2) and (7.89±5.01) in the two groups, respectively.

About 20 cells (20±2.53) of exposed non-smoke individuals showed medium damage compared to almost 5 cells (5.33±1.86) with high damage in the same group. The same pattern was seen in control non-smoke group where about four cells (3.56±1.94) showed medium damage, while nearly just one cell (0.89±0.6) showed high damage in the same group. The median tail length of exposed non-smoke group was (19.67±2.96 µm) compared to (4.54±1.54µm) in control non-smoke group. Clearly seen that most of the cells of the control non-smoke participants showed no damage (62.67±6.96), while the damage was seen in almost 24 cells (24.33±7.12) in exposed non-smoke group. The difference in the severity of damage between these two groups was significantly high (p. value <0.01), as seen in Table 3.

Generally, the control groups (regardless smoking status) revealed no damage of about 83%, followed by 10%, 5% and 2% with low, medium and high damage, respectively. On the other hand, and comparing to all exposed individuals (regardless smoking status), the percentage of no damage was about 37%, while 29% of the cells revealed low damage, followed by 27% and 7% showed medium and high damage, respectively, figure 1.

Pearson correlation revealed strong positive correlation between DNA damage and the duration of exposure. The correlation was positive (0.718, 0.732, 0.531) for high, medium and low DNA damage, respectively. The tail length had a strong positive correlation with the hours of exposure (0.835).

Table 2: Differences in DNA damage between exposed smoke and control smoke groups.

Indicators	Exposed Smoke 9		Control smoke 6		T test	P value (Sig.)
	Mean	SD	Mean	SD		
No damage	30.3333	13.25707	61.5000	4.08656	-5.525	.000
Low Damage (number of cells from 75 cells)	19.0000	6.32456	6.8333	3.06050	4.346	.001
Medium Damage (number of cells from 75 cells)	20.7778	8.18196	4.8333	1.94079	4.633	0.000
High Damage (number of cells from 75 cells)	4.8889	1.90029	1.8333	.75277	3.711	0.003
Tail Length (µm)	18.0222	5.36217	4.0027	.80080	6.280	.000

Table 3: Differences in DNA damage between exposed nonsmoke and control nonsmoke groups SD : Standard Deviation ; HS : High Significant at P value <0.01.

Indicators	Exposed Nonsmoke 6		Control Nonsmoke 9		T test	P value (Sig.)
	Mean	SD	Mean	SD		
No damage	24.3333	7.11805	62.6667	6.96419	-10.355	.000
Low Damage (number of cells from 75 cells)	25.3333	7.20185	7.8889	5.01110	5.563	.000
Medium Damage (number of cells from 75 cells)	20.0000	2.52982	3.5556	1.94365	14.262	**0.000**
High Damage (number of cells from 75 cells)	5.3333	1.86190	.8889	.60093	6.761	**0.000**
Tail Length (µm)	19.6667	2.95544	4.5413	1.54759	13.054	.000

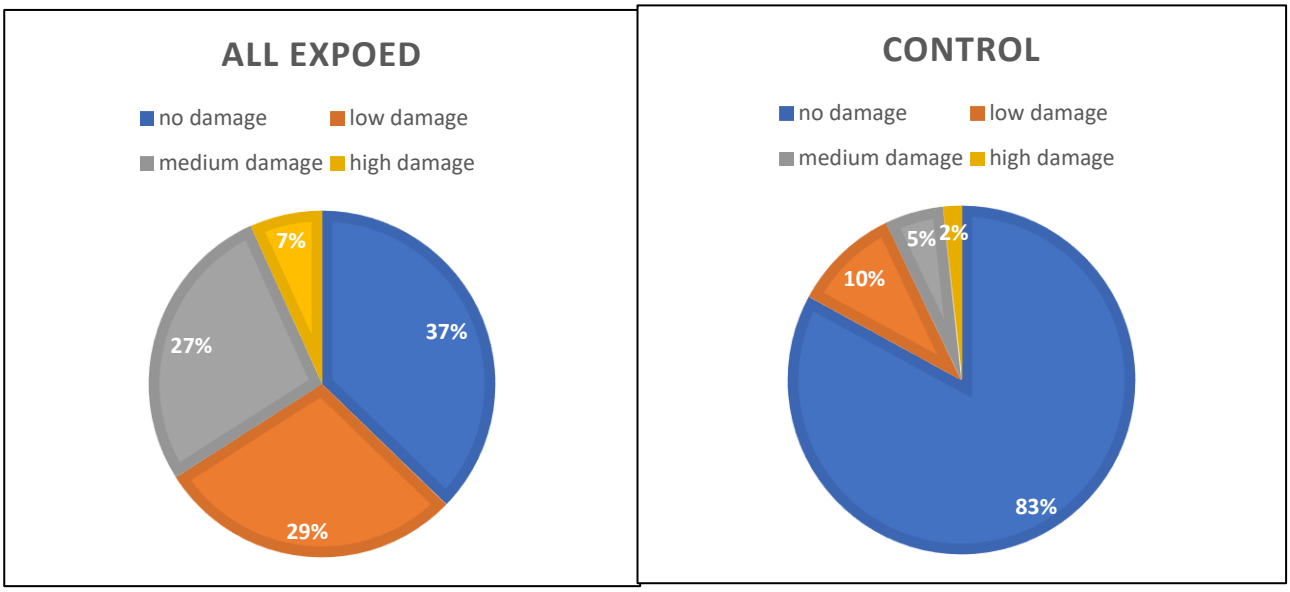

Figure 1: Percentages of DNA damages in Exposed and control groups.

DISCUSSION

Exposure to vehicle emissions, which are the primary source of urban air pollution has been associated with abnormalities in the blood parameters and DNA integrity. These emissions are found to be filled with fine particulate matter (PM 2.5), carbon monoxide (CO), nitrogen dioxide (NO2) and other toxic substances which trigger oxidative stress and inflammatory responses, that alter haematological parameters leading to erythrocytosis, elevated haemoglobin levels, changes in some red blood cells indices such as MCV and MCH (7) and (8). The increasing number of residents and vehicles, accordingly; in a small city like Najaf, has drawn our attention to study the effect of pollutants emitted from these vehicles on occupationally exposed subjects specifically traffic police workers, in a way to increase the awareness towards this health and environmental issue and set up some regulations in urbanized areas.

The study was designed to evaluate the effect of vehicle emissions on traffic police workers comparing that to a control group of

non-exposed participants, and taking into account the smoking status of both groups. This allowed us to study the synergetic effect of both smoking and emissions as well as emissions only. The comparison between exposed smoke and control smoke groups using (one way anova) T-test revealed a significant difference (P.value <0.05) regarding RBCs, Hb, HCT and WBCs while MCV and MCH were not affected. On the other hand, and while comparing the other two groups when the effect of smoking was excluded, difference raised in RBCs and MCH only. This is in contrast to what have been stated by Maffei et al. where no significant difference were found while comparing police traffic workers to control group regarding these haematological parameters (9). However, another study conducted by Odozi EB. and Ajayi O., found that there was increase in RBCs, Hb and HCT with alterations in RBCs indices (10). Moreover, another recent study revealed leukocytosis in 15% of exposed participants compared to control group (11) and (12).

Regarding DNA, the damage was estimated in 75 cells per individual for all the groups. The damage was highly in exposed individuals both smokers and non-smokers. The DNA damage correlated with the duration of exposure. A study conducted in Baghdad on traffic police men stated that exposing to air pollutants specifically polycyclic aromatic hydrocar- bons could reduce the levels of antioxidant enzymes with in turn increasing DNA damage that can finally ending up with cancers (13). Another study revealed that traffic police officers could be at danger of developing DNA damage due to increasing the levels of urinary 8-OHdG level, a biomarker of oxidative DNA damage comparing to control group (1) and (3). This was also revealed by another study where there was increase in the level of 8-OHdG level by 0.329% and tail DNA by 0.051% and that was positively correlated with intersection duty time in traffic policemen compared to office policemen (4).

Limitations

Small sample size was the first limitation which will be increased during the subsequent related research. The type of emissions has to be determined and the level of the oxidative DNA damage biomarkers has to be estimated accordingly. Our participants distributed over the whole city, and they were rotated in different locations as a part of their duties, so we could not locate which location contributes more to adverse health effect. Finally, there were small number of published papers which focus on vehicle emission contamination and its consequences in the middle east or neighbor countries, which if were available; comparison of our results would be more logic.

CONCLUSION

Exposure to air pollutants were found to have an effect on blood parameters as well as DNA integrity in traffic police officers. The degree of effect positively correlates with the exposure time. This brings a high priority concern that needs to be taking into consideration by the local government as it raises a public health issue. Sustainable sources of energy; which almost with zero emissions, is a reasonable option to think about especially in cities with high traffic-related air pollution.

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