Original
ISSN 2413-0516

Evaluation of DNA Damage in Traffic Cops Exposed to Polycyclic Aromatic Hydrocarbon Pollution in Baghdad City Streets and its Association to Certain Biomarkers

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(Submitted: 03 April 2022 – Revised version received: 14 April 2022 – Accepted: 09 May 2022 – Published online: 26 June 2022)

Abstract
Objectives: To find out the extent to which the genetic material of Baghdad traffic policemen is affected by the oxidative damage caused by pollutants by monitoring the levels of 8-OHdG compared to the levels of antioxidant enzymes and malondialdehyde.
Methods: This study includes 140 participants; they have been divided into two groups (traffic police and office police). Polycyclic aromatic hydrocarbons were analyzed for each participant by GC/FID while 8-OHdG and antioxidant enzymes were measured by the ELISA technique.
Results: The levels of polycyclic aromatic hydrocarbons, 8-Oxo-dG, and malondialdehyde were elevated in the blood of the traffic police compared to the office police, while higher levels of antioxidant enzymes (Catalase and glutathione peroxidase) were observed in the blood of the office police.
Conclusion: Exposure to polycyclic aromatic hydrocarbons can cause oxidative stress through their metabolic derivatives and the resulting active molecules, which lead to the formation of 8-Oxo-dG and the reduction of enzymatic antioxidants, which may lead to the emergence of cancers.
Keywords: DNA Damage, Traffic Cops, Polycyclic Aromatic Hydrocarbons, Pollution, Catalase

Introduction

The impact of air pollution on human health and the wide-ranging effects it has on biodiversity have propelled this problem to the top of the political agenda globally.[1] In 2005, the World Health Organization released air quality standards for outdoor air pollution. These guidelines include allowed values for fine particulate matter (particulate matter with an aerodynamic diameter of less than 2.5 m) on an annual and daily basis, sulfur dioxide, ozone, nitrogen dioxide, and coarse particulate matter (PM2.5) (particulate matter 10 m in aerodynamic diameter).[2]

At the inaugural summit on air pollution and health in 2018, WHO established a target of preventing 7 million deaths worldwide from air pollution by 2030.[3] Air pollution is related to cardiovascular diseases,[4] chronic obstructive pulmonary,[5] and several types of tumors, such as lung, breast, oral, liver, kidney, prostate, bladder and ovarian,[6,7] it is thought to be responsible for 4.2 million premature deaths worldwide.[8]

During the activation of polycyclic aromatic hydrocarbons (PAH) by cytochrome P_{450} (CYP_{450}), a large quantity of reactive oxygen species and numerous electrophiles are produced, which relate covalently to DNA and disrupt cell homeostasis.[9] Polycyclic aromatic hydrocarbons appear to be key risk factors found in automotive exhausts that cause oxidative stress, since exposure to PAHs is linked to an increase in the formation of free radicals.[10] On the other hand, oxidative stress might be one of the reasons driving many of the negative health impacts associated with air pollution, Figure 1.[11] 8-oxo-7,8-dihydro-2′-deoxyguanosine (8-oxodG) is the most common oxidative stress-induced pest that may induce mutations in DNA replication. Also, they are thus important biomarkers of oxidative DNA damage, although they are consistently repaired by base excision repair (BER).[13] It is regarded as a active premutagenic lesion due to its capacity to pair with both cytosine and adenine residues and result in G:C to T:A transversions during DNA replication.[14] The C8 of the imidazole ring in deoxyguanine (dG) can be attacked by the hydroxyl radical (•OH), hydroperoxide radical (•OOH), singlet oxygen ($1O_2$), superoxide (O_2•-), reactive nitrogen species (NO_2), and peroxynitrite anion ($ONOO.^-$) This results in the formation of 8-oxo-dG.[15] But in fact, the interaction of DNA with •OH in Figure 2 is the main source of 8-oxo-dG.[16]

The 8-oxodG has been utilized as a risk factor for several illnesses in addition to being used as a biomarker to assess endogenous oxidative DNA damage.[17]

It can be utilized as an index in various cancer,[18] neurodegenerative diseases,[19] diabetes,[20] cardiomyopathy[21] and cardiovascular or infectious diseases.[22] As a result, 8-OHdG is helpful for high-risk people' early identification and assessment.[23]

Materials and Methods

One hundred and forty 140 Iraqi policemen affiliated with the traffic police in the city of Baghdad within the Iraqi Ministry of Interior. Their ages range between (25-65 years), during the period from July 15, 2019 to March 25, 2020. These subjects were divided into two groups as the following:
Group 1: 70 policemen of those who were performing their duty in the crowded squares and intersections in Baghdad (Traffic police).
Group 2: 70 policemen of those who were serving inside the buildings of the various traffic directorates (Office police).

Each participant had about 6 ml of blood pulled from a vein, which was then left at room temperature for 15 to 20

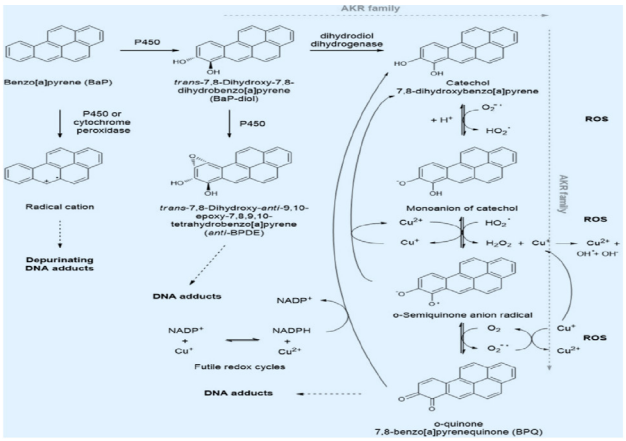

Fig. 1 **The BaP metabolites are powerful reactive compounds that can combine with DNA to generate adducts.**[12]

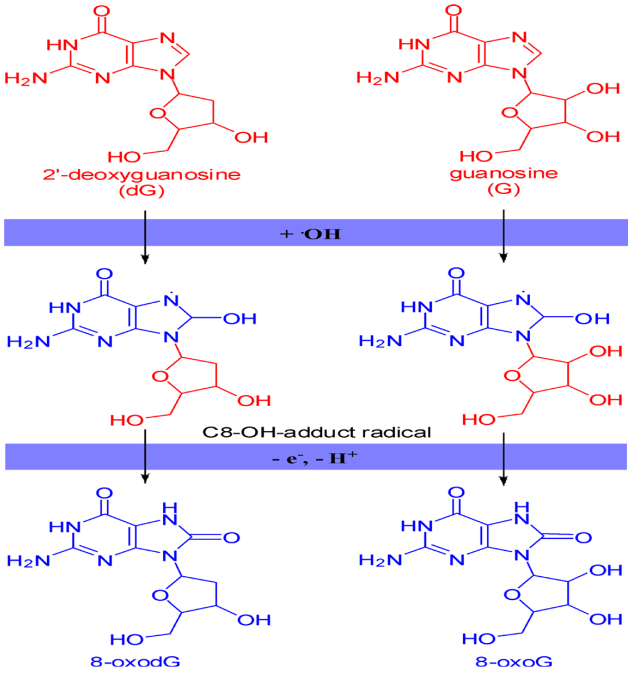

Fig. 2 **The process by which 8-oxodG and 8-oxoG are formed.**[24]

minutes to allow for coagulation, centrifuged for 10 minutes at 3000 rpm to detach the serum, and then the serum was split into small parts and stored at 20°C till it was consumed for PAH determination.

1. Gas chromatography with a Flame Ionization Detector (GC/FID) was used to measure the levels of PAH in serum.
2. 8-OHdG was determined by Enzyme-Linked Immunosorbent assay (ELISA) Kit Catalog Number. ab201734.
3. Determination of malondialdehyde by Buege and Aust method. The MDA was estimated using the thiobarbituric acid, which reacts with malondialdehyde to produce a pink color that can be read at a wavelength of up to 535 nm.
4. Determination of the activity levels of serum glutathione peroxidase, by ELISA Kit Catalog Number. E-EL-H5410.
5. Determination of the serum concentration of catalase, by ELISA Kit Catalog Number. MBS703074.

Results

Table 1 displays the average age for the two groups.

Mean concentration of PAHs, 8-Oxo-dG, MDA, and two antioxidant enzymes (Catalase and G-px) for the two groups are shown in Table 2, the effect of the exposure period to pollutants from car exhaust and diesel engines on the traffic police who are in the offices as well as the traffic police deployed in the crowded intersections of the Baghdad city are summarized in the (Table 3) and (Table 4) respectively.

Discussion

Polycyclic aromatic hydrocarbons are hazardous pollutants, as exposure to these harmful substances increases the chance of

Table 1. **The groups' average ages**

Group	*N.*	Age (mean ± SD)
Office police	70	38.54 ± 5.49
Traffic police	70	40.15 ± 5.7

Table 2. **The average levels of PAHs, 8-Oxo-dG, MDA, Catalase, and Gpx were within the two groups**

Parameter	Group	Mean ± S.E.	*P*-value
PAHs (ppm)	Office police	6.91 ± 0.12	0.0001
	Traffic police	8.94 ± 0.09	
8-Oxo-dG conc. (ng/ml)	Office police	96.88 ± 9.35	0.0001
	Traffic police	148.17 ± 8.47	
MDA conc. (µmol/l)	Office police	2.1 ± 0.22	0.025
	Traffic police	2.84 ± 0.26	
Catalase activity (pg/ml)	Office police	648.15 ± 15.3	0.0001
	Traffic police	398.52 ± 16.58	
G-px activity (pg/ml)	Office police	55.74 ± 2.99	0.0001
	Traffic police	34.34 ± 2.2	

P-value <0.05 is significant.

Table 3. **Comparison of the analyzed parameters for the office police group according to the length of exposure**

Parameter	Duration of exposure	Mean ± S.E.	*P*-value
PAHs (ppm)	less than 10 years	6.7 ± 0.31	0.599
	more than 10 years	6.97 ± 0.12	
8-Oxo-dG (ng/ml)	less than 10 years	101.55 ± 21.14	0.780
	more than 10 years	95.39 ± 10.44	
MDA (µmol/l)	less than 10 years	1.97 ± 0.54	0.880
	more than 10 years	2.14 ± 0.23	
Catalase (pg/ml)	less than 10 years	652.12 ± 43.34	0.995
	more than 10 years	646.88 ± 15	
G-px (pg/ml)	less than 10 years	59.89 ± 6.18	0.703
	more than 10 years	54.41 ± 3.44	

P-value >0.05 is non-significant.

Table 4. **Comparison of the analyzed parameters for the traffic police group according to the length of exposure**

Parameter	Duration of exposure	Mean ± S.E.	*P*-value
PAHs (ppm)	less than 10 years	8.80 ± 0.21	0.487
	more than 10 years	9.01 ± 0.09	
8-Oxo-dG (ng/ml)	less than 10 years	145.05 ± 12.62	0.782
	more than 10 years	149.60 ± 10.99	
MDA (µmol/l)	less than 10 years	2.63 ± 0.38	0.974
	more than 10 years	2.93 ± 0.34	
Catalase (pg/ml)	less than 10 years	394.49 ± 33.74	0.947
	more than 10 years	400.36 ± 18.85	
G-px (pg/ml)	less than 10 years	35.27 ± 5.00	0.781
	more than 10 years	33.91 ± 2.28	

P-value >0.05 is non-significant.

developing cancer,[25] due to the harmful effects that these toxins and their reactive metabolites, such as dihydrodiols and epoxides, can have when they join to DNA and cellular proteins[26] like mutations, problems in development, and cancers that occur from the cell damage.[27]

The results of this study showed the presence of high concentrations of polycyclic aromatic hydrocarbons in the blood serum of the traffic police who are at the busy street intersections of the city of Baghdad, relative to the levels in the blood serum of the office police group. Since 2003, the number of passenger automobiles, buses, trucks, and household generators has skyrocketed in Iraq, posing serious environmental concerns. Near the Technology University in Baghdad, on and surrounding the Muhammad Al-Qasim highway, a study was conducted by[28] to assess the connection between the level of activity, the movement of cars with various engines, and the pollution that comes from exhaust pipes, and it was discovered that pollutants such as sulfur and polycyclic aromatic hydrocarbons, increase during the start and end periods of the working hours of state departments. Moreover, Iraqi inhabitants employ hundreds of thousands of tiny electric generators in their houses,[29] which use heavy oil or gasoline to create electricity, resulting in large quantities of soot, carbon deposits, and sulfur oxides.[30]

A recent study that measured air pollutants around Iraq, showed that the highest number was recorded in Al-Diwaniyah, followed by Baghdad, specifically the Dora area.[31] Our results are in agreement with several previous studies, such as research[32] on PAHs released by vehicles, which found that street cops have high exposures, much higher than chefs, and their exposure levels are comparable to coke factory workers. Another study in China found that traffic cops have a higher risk of PM2.5 pollution in the workplace than office cops who are considered a control group.[33]

In our current study, high levels of 8-Oxo-dG were found in the blood serum of the traffic police group at the intersections of the capital Baghdad compared with the levels recorded for the vital sign in the police group offices (representing the control group), and it was found that the increase in the levels of 8-Oxo-dG was in conjunction with the increase in the levels of PAHs in the traffic police group in the streets of Baghdad city who are directly exposed to the dangers of pollutants emitted from car exhaust and diesel engines. Many studies have utilized the 8-OHdG not only as a biomarker to estimate the impact of endogenous oxidative DNA damage but also as a health risk for a variety of disorders, including cancer.[34]

Various studies regarding the role of malondialdehyde, catalase and reduced glutathione in oxidative status studies have been reported in different clinical conditions.[35,36] Our study also added an MDA biomarker to assess its levels in the blood of traffic police personnel and compare it with levels of PAHs in the two study groups. This study found an important correlation between the level of pollutants in the blood and the level of MDA, where the relationship between the level of PAHs and the level of MDA was positive and highly statistically significant as shown In the shape (after completion).

Our findings are consistent with the research conducted by[37] on the workers of filling stations in Nanjing, China. This study demonstrated that refueling workers' blood GSH levels were much lower than those of office workers. In contrast, refueling workers' serum MDA and 8-OHdG levels were substantially greater than those of office employees. In research on traffic cops,[38] reported a positive relationship between PM2.5 exposure and 8-Oxo-dG. Another study found that smokers' plasma MDA levels were larger than non-smokers.[39] Another significant study[34] was conducted on coke plant workers in China, in which workers in a coke plant were classified into a group exposed to relatively less particulate matter and a second group highly exposed to particulate matter exposed. As an internal dosage, urine concentrations of PAHs metabolites and minerals were evaluated. The study reported higher levels of urinary 8-Oxo-dG and MDA were substantially linked to higher levels of PM2.5 and overall PAHs. By redox cycling, Polycyclic aromatic hydrocarbons intermediates generate ROS and trigger oxidative stress through many metabolic processes. Exposure to Polycyclic a romantic hydrocarbon from environmental pollution was found to be positively related to urinary 8-OHdG amounts in epidemiological studies. The generation of ROS in normal metabolic processes, however, often does not result in oxidative stress because it is exactly balanced by the natural antioxidant system. As a result, toxins, ionizing radiations, and other external factors, as well as consumer habits and lifestyle factors such as alcohol consumption, tobacco, lack of physical activity, a poor diet, and certain genetic factors, could all contribute to an increase in ROS production that overwhelms antioxidant defenses, resulting in oxidative stress. Higher levels of 8-OHdG may operate as a biomarker for oxidative stress on DNA, although it is not a particular diagnostic for PAH exposure.[39] The rate of DNA repair processes, which involves the removal of damaged bases or complete nucleotides, is determined by the analyzed persons' health state, age, diet, metabolic activity, and behavior.[40] Additionally, biological membranes' shape and fluidity can be altered by ROS-induced membrane lipid peroxidation, which ultimately affects how well the membranes operate. Malondialdehyde (MDA) and hydroxynonenal are two of the most well studied indicators of lipid peroxidation (HNE). MDA is a highly reactive nuclear factor produced both by lipid peroxidation and as a byproduct of the synthesis of prostaglandins and thromboxanes that can attack large molecules, including the group of proteins from amino acids or sulfhydryls resulting in changes in their functions. HNE is a

significant toxin produced when polyunsaturated fatty acids are attacked by ROS. It interacts with proteins to produce advanced end products of lipid oxidation. Both HNE and MDA approaches have been fined in atherosclerotic lesions.[41]

Elevated concentrations of urinary 8-Oxo-2′-deoxyguanosine were found to be strongly related to an increased risk of lung cancer in never-smokers in an early prospective investigation. A recent, nested case-control study of lung cancer and automotive pollution found that employees in highly polluted areas had a lifelong risk of developing lung cancer of at minimum 50%.[42] As a result, we can conclude that a deficiency of ability to regenerate mitochondrial and nuclear DNA injury is associated with a variety of neurological illnesses and tumors. Whereas, the high formation of 8-oxodG is a potential mutagenic lesion in DNA that leads to the conversion of G: C to T: A (G → T) during DNA replication. Therefore, it is potentially a powerful cancer prediction weapon, if mutation-prone DNA lesions such as 8-oxo dG can be identified in genome scales.[43] MDA reacts with DNA to yield harmful adducts of deoxyadenosine and deoxyguanosine.[44] Additionally, the shape and fluidity of biological membranes are altered during the membrane lipid peroxidation process brought on by ROS, which ultimately affects how well they function.[41]

Two types of antioxidant enzymes (catalase and glutathione peroxidase) were selected to study their levels in the blood serum of the two study groups and their relationship to PAH level for both groups.

Our current study, found low levels in the levels of both catalase and glutathione peroxidase enzymes in the blood serum of traffic police personnel deployed in Baghdad city intersections compared to the levels of the two enzymes were in the control group represented by the offices police, and the levels of these enzymes were inversely proportional to the levels of pollutants in the blood serum represented by polycyclic aromatic hydrocarbons. Also, some studies conducted to evaluate the effects of occupational exposure to pollutants on oxidative stress in the body have indicated a decrease in the levels of antioxidant enzymes. In a study of pollution-exposed taxi drivers,[45] reported a decrease in CAT and G-Px activities compared to the occupationally unexposed group. Cohort research done before, during, and after the Beijing Olympics has reported that when air pollution levels increased, indicators of total antioxidant status-declined.[46] The production of a sizable quantity of ROS by particulate matter in traffic exhausts is one theory for the reported negative health impacts. The smaller particles of the aerosols in the environment contain smaller, more ROS-rich particles. Polycyclic aromatic hydrocarbons are also present in fine particles from vehicle exhaust (PAHs). It has been demonstrated that antioxidants play a critical role in the catalysis of the conversion of (O_2) to H_2O_2 and the breakdown of H_2O_2 into H_2O, respectively. Oxidative stress occurs when reactive oxygen species exceed the capacity of the antioxidants to act to defend the cell. Once antioxidant enzymes are depleted, the cell is more vulnerable to the harmful effects of a xenobiotic that can lead to cell harm or death. As a result, the repeated inhalation of gasoline vapors possesses potential to cause oxidative stress by lowering the body's antioxidant defenses and cellular functions.[47] Poor nutritional status can also contribute to oxidative stress, for example, selenium deficiency, which is associated with increased oxidative stress; higher GPx activity may arise from optimizing Se's nutritional status. A higher risk of cancer in epidemiological research studies has been linked to low levels of antioxidant intake. G-Px loss resulted in loss of endothelial function, decreased angiogenesis, and increased infarction severity and vascular permeability in experimental animals,[48] as seen the activity of catalase, catalase, SOD and GPx to have a substantial negative relationship with the risk of coronary artery disease in patients.[49] In response to oxidative stress, prolonged antioxidant enzyme deficiency enhances tissue sensitivity and severity.[50]

Conclusion

Exposure to air pollutants like polycyclic aromatic hydrocarbons can reduce levels of antioxidant enzymes and thus create a state of oxidative stress, manifested in the presence of high levels of 8-oxodG and MDA, which can lead to DNA damage that can lead to many types of cancer in the traffic policemen.

Conflict of Interest

None.

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