EFFECT OF SMOKING ON CBC, EXPOSURE OF PAH AND OXIDATIVE STRESS BIOMARKERS IN RURAL POPULATION OF DISTRICT OKARA, PAKISTAN

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ABSTRACT: Smoking is one of the global problems causing different disease. The health effects of smoking are widespread and well known. Smoking cause variation in different parameters of blood profile and exposure to PAHs causing oxidative stress among smokers. The aim of the study is to evaluate the effect of smoking on total blood count, exposure of PAHs and their association with oxidative stress biomarkers in rural population of district Okara. A total of 60 blood sample are collected, 30 males (15 smoker and 15 non-smoker), 30 females (15 smoker and 15 non-smoker); which is further subdivided into three groups based on the number of cigarettes smoked per day (active smoker, passive smoker and non-smoker). Blood samples were collected and subjected for the estimation of hematological parameters. Polycyclic aromatic hydrocarbons using HPLC, oxidative stress biomarkers (lipid peroxide, Superoxide dismutase and Catalase) using spectrophotometer. Among the smokers white blood cell and red blood cell increases and other hematological parameters such as MCV, MCHC, MCH, platelets increase while eosinophils, monocytes, E.S.R decrease. The level of PAHs in smoker increase as compared to non-smoker. The oxidative stress biomarkers in smokers MDA and CAT activity increase while decrease in SOD activity. The comparison between smoker male and smoker female or non-smoker male and non-smoker female shows mostly no significant difference ($p>0.05$). The comparison between smoker male and non-smoker male hemoglobin, T.L.C, E.S.R, neutrophils, R.B.C, platelets, PAHs, MDA , CAT and SOD shows statistically significant difference ($p<0.05$).The comparison between smoker female and non-smoker female hemoglobin, T.L.C, E.S.R, neutrophils, R.B.C, platelets, PAHs, MDA, CAT and SOD shows statistically significant difference ($p<0.05$). Based on the results of present investigation it is concluded that smoking increases the level of free radicals in blood which have deleterious effects in smokers and causing oxidative stress increase level of PAH, which is harmful and causing problems in human body.

Key words: smoking**,** diseases, blood profile, exposure, PAH, oxidative stress, hematological parameters, free radicals, problem.

(Received 23.10.2025 Accepted 15.12.2025)

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs), a large family of global eco-toxins with diverse properties of toxicity and biotic effects, are capable of causing oxidative stress, during its metabolism (Armstrong, 2009; Singh *et al* 2008). PAHs are yields of partial ignition of organic matter, either in nature (open scorching, natural residual, drainage of petroleum-based chemicals or volcanic activities) or anthropogenically. Large man-made sources of PAHs include housing fume gasification, heating and water treatment plants, firwood tar and Blackwood manufacture, coal and aluminum production, oil processing plants and vehicular emissions. (Baklanov *et al*, 2007; Latimer and Zheng, 2003).

In atmosphere, PAHs transpires as particles and vapors which are transported to the human body. The toxicity of compound is mainly dependent on biotransformation, size and phase distribution which may perhaps mature into very compelling mutagens and carcinogens (Zhu *et al*, 2009). Digestive tract of animals readily accumulates PAHs compounds because of their high lipid solubility. (Chomanee 2008) According to a study, bioremediation has proved to be an effective technique for the removal of such contaminants (Abdel and Mansour, 2016).

More than 40 notorious cancer-causing agents including benzo(a)pyrene are identified as a product of tobacco burning (Lannerö *et al*. 2008) Both cigarette smoking and passive exposure to tobacco smoke are important causes of mortality in the United States and other developed countries (Gidding *et al*, 1994) Besides these pathways, induction of oxidative stress has been proposed previously as a possible mechanism of action for carcinogenesis(Gargon *et al* 2001) The hydroxylated PAHs have been used as biomarkers of PAH exposure from tobacco smoke (Hadedorn *et al*, 2009). The metabolism of PAHs initiates through oxidase function of mixed cytochrome P450 forming DNA adducts resulting in the modification lipids and DNA (Penning *et al* 2007;

Palackal *et al*, 2002) As a result of metabolic activation of PAHs, reactive oxygen species and free radicals are produced which covalently form bonds with the DNA causing many physiological changes in human body that may lead to mutations, developmental malformations, tumors and cancer. (Cavalieri and Rogan, 1995). In the proposed study, we aimed to assess the cross-sectional associations of an estimate of PAHs exposure from blood biomarkers of antioxidant capacity among smoker and non-smoker urban population near Okara district, Pakistan.

MATERIALS AND METHODS

Study Population: The study population consisted of 60 healthy subjects (30 women/30 men) divided into two groups i.e., smokers (consuming more than 10 cigarettes in a day) and non-smokers, belonged to the areas near district Okara, Pakistan. The blood donors were particularly adults with age ranging from 18 to 65 years (mean32.7±9.9). The blood samples were collected during a four-month duration (January 2019 - April 2019). More than 100 samples were collected initially which were scrutinized down to 60 samples; in accordance with the requirements of proposed study. The research was approved by the Ethics and written, well-versed consent was taken from the participants before signing them up for the study.

Sample Extraction and Preparation: A specific amount of intravenous blood was drawn from each selected individual and was kept in dipotassium ethylene diamine tetra acid (EDTA) anticoagulant tubes. All biological samples were labeled with specific lab number and names in order to keep in good condition for further analysis. Generally, EDTA is suggested as the anticoagulant for admirable hematological testing as it permits the finest maintenance of cellular components and morphology of blood cells *(Canetti, 2016).* In 3ml blood sample, 4-5mg EDTA was added per sample in test tubes in order to cease blood clotting. Ethylene diamine tetra acetic acid (EDTA) strongly and permanently binds calcium ions, which evades blood from clotting. *(Hennø et al., 2017)*

Complete blood count: A comprehensive blood test of selected individuals was carried out and red blood cells, white blood cells, and platelets were measured, which was achieved by using a MYTHIC 22; a fully automated (Microprocessor controlled) hematology analyzer used for in vitro analytical testing of blood samples. MYTHIC 22, a visual measurement system for the examination of more than 22 hematological parameters was used for complete blood count (CBC), stating overall health of selected subjects which measured hemoglobin, T.L.C, E.S.R, neutrophils, lymphocytes, monocytes, eosinophils, R.B.C, MCV, HCT (PCV), platelets, MCH and MCHC *(Okpogba et al, 2019).*

Polycyclic aromatic hydrocarbons (PAHs): Initially stock solutions of PAHs were prepared which includes anthracene-3,4,8, 9-dibenzo(a)pyrene and 1-hydroxypyrene using HPLC grade DCM. The pole which examines the levels was of the dimensions 25 cm x 10 mm C18 (Waters). The moving solvent was HPLC grade acetonitrile which was run isostatically at 1 ml/min. The volume of the injection was 10 pL. The detector was photodiode array detector with a complete spectrum of 190-600 nm and detection range of 1.2 nm. To establish a standard linear curve, standards were run in several dilutions. Plasma (~500 pi) was pipetted into 13x100 mm borosilicate tubes. The supernatant was shifted to a clean tube and the removal process was repeated. The supernatants were combined and dehydrated under nitrogen at room temperature in the chemical hood. When the solvent had vaporized, tubes were covered and kept in refrigerator at 4°C until examined. The filtrate was warmed at room temperature and refabricated with an amount of dichloromethane equal to unusual plasma volume. Tubes were sonicated for 1 minute and the refabricated extract was transferred to labelled sample vials before inserting them in auto-sampler for analysis.

Oxidative Stress Biomarkers: The blood was centrifuged at 3000 g for 10 minutes. The plasma was composed cautiously and used for the analysis of lipid peroxidation and SOD (Superoxide Dismutase) and CAT(Catalase) activity using standard essays.

Statistical Analysis: All parameters showed obtainable normal or marginally normal distribution using SPSS (IBM 20). It was used for the parametric comparisons across groups (smokers and non-smokers) matched for age, expressed as means ± standard deviation (SD). Statistical significance was well-defined at $p < 0.05$, with p values being 2-sided. Correlation was applied between 3PAHs and oxidative stress markers.

RESULTS AND DISCUSSION

Complete Blood Count Profile in Smoker and Non-smoker: The mean and SD values of smoker and non-smoker shows an increased value in smoker of CBC parameters hemoglobin, T.L.C, neutrophils, R.B.C, platelets, MCV, MCHC, MCHC and HCT and decrease value of E.S.R, lymphocyte, monocytes, eosinophils. The results of our conclusions showed that cigarette smoking has many adverse effects on hematological parameters (e.g., hemoglobin, T.L.C, neutrophils, R.B.C, platelets, MCV, MCHC, MCHC, HCT, E.S.R, lymphocyte, monocytes and eosinophils).

Factors influencing in smoker and non-smoker CBC profile: Similar result was obtained by different authors in previous study. But in our current study of selected area Okara show slightly change in values. The present

study reveals that this might be difference in values because of industries, smog, burning of wood, low standard cigarette, local drugs, light and menthol cigarettes, cigars and Pipes, little cigars, cigarillos, hookahs, bidis, clove cigarettes, dissolvable products, electronic cigarettes (also referred to as: vape pen, e-hookah, hookah pen), traditional smokeless tobacco products, waterpipes (also referred to as: hookah, shisha, narghile, argileh).

Table 1: Compares of the hematological parameters between smokers and non-smokers

CBC Parameters	Sex M=30 F=30	Smoker		Non Smoker	
		Mean	SD	Mean	SD
Hemoglobin	Male	14.59	0.94	11.79	4.24
g/dl	Female	13.62	0.89	10.26	2.40
T.L.C	Male	6300	282.84	5500	694.74
1/cmm	Female	6253.33	368.54	5473	589.31
E.S.R	Male	12.6	6.58	29.46	15.98
m/1st hour	Female	12.2	4.70	21.06	9.82
Neutrophils	Male	66.47	1.14	65.27	1.43
%	Female	65.8	1.46	64.26	1.84
Lymphocyte	Male	28.47	0.71	31.2	6.26
%	Female	28	4.96	28.86	2.09
Monocytes	Male	3.13	0.80	3.3	0.59
%	Female	2.53	0.49	3.06	0.67
Eosinophils	Male	1.73	0.44	1.8	0.4
%	Female	1.67	0.46	1.6	0.48
R.B.C	Male	4.7	0.40	3.65	0.92
Million/cmm	Female	4.61	0.38	3.63	0.55
Platelets	Male	262000	10795.06	249666.7	10749.68
1/cmm	Female	253733.33	7469.64	245333.3	9491.51
HCT	Male	43.94	3.33	35.32	2.91
%	Female	39.19	3.70	34.97	2.80
MCV	Male	94.73	4.78	76.02	12.36
Fl	Female	82.27	5.38	68.79	17.92
MCH	Male	28.47	1.94	23.81	2.64
pg	Female	27.28	4.58	21.72	1.66
MCHC	Male	40.03	2.19	33.15	3.42
%	Female	35.51	3.52	31.92	2.83

Polycyclic Aromatic Hydrocarbon Level in Smoker and Non-smoker: The results showed that the 3PAHs level in blood demonstrated an increasing trend in experimental group among the experimental groups, smoker male recorded the maximum value. The comparison between the smoker male and smoker female shows p=0.815 ($p<0.05$), which revealed that no significant difference. The comparison between the non-smoker male and non-smoker female shows p=0.636 ($p<0.05$), which proves there is no significant difference. Comparison between smoker male and non-smoker male, p=0.005 ($p<0.05$) which proves significant difference. When smoker female is compared with non-smoker female, p value is 0.017 ($p<0.05$) which proves significant difference.

Polycyclic aromatic hydrocarbons do not present in the plant of tobacco, but are developed mainly by incomplete combustion of tobacco and further organic components during smoking. Polycyclic aromatic hydrocarbons are contemporary in nearly unburned products of tobacco, mostly which containing tobacco fire-cured variations. Throughout fire curing, polycyclic aromatic hydrocarbons in combustion vapors produced by flaming wood are left on the leaves of tobacco.

Factor influencing in smoker and non-smoker PAHs exposure: The current study of selected area Okara show might be slightly change in values with other authors. This reveals that this might be difference in values because of industries, smog, burning of wood, different sources from where PAHs generated, low standard cigarette, local drugs, light and menthol cigarettes, cigars and Pipes, little cigars, cigarillos, hookahs, bidis, clove cigarettes, dissolvable products, electronic cigarettes (also referred to as: vape pen, e-hookah, hookah pen), traditional smokeless tobacco products, waterpipes (also referred to as: hookah, shisha, narghile, argileh).

Table 2: Level of Polycyclic aromatic hydrocarbon in smoker and non-smoker

PAH	Sex M=30 F=30	Smoker		Non Smoker	
		Mean	SD	Mean	SD
Anthracene	Male	7.24	0.40	6.80	0.61
pmol/ml	Female	7.04	0.52	6.84	0.69
Benzo(a)pyrene	Male	5.03	0.57	4.82	0.49
pmol/ml	Female	5.1	0.51	4.76	0.59
1-hydroxypyrene	Male	267.26	11.15	252.6	11.14
pmol/ml	Female	266.13	16.62	256.06	9.89
3 PAH	Male	279.55	11.41	266.52	11.43
pmol/ml	Female	278.27	16.63	264.47	10.80

Table 3: Mean and SD of Oxidative Stress and Enzyme Activity in Smoker and Non-Smoke

Biochemical Parameters	Sex M=30 F=30	Smoker		Non Smoker	
		Mean	SD	Mean	SD
MDA	Male	2.59	0.35	1.52	0.23
nmol/ml	Female	2.47	0.31	1.54	0.24
CAT	Male	91.45	7.32	67.01	1.86
nmol/ml	Female	92.05	3.59	67.38	0.93
SOD	Male	4.54	0.22	19.11	0.40
units/ml	Female	4.5	0.30	18.03	2.53

Factors influencing in smoker and non-smoker oxidative stress biomarkers: The current study of selected was similar to previous study but slightly change in values with other authors because of small area of samples Okara. This reveals that this might be difference in values because of industries, smog, burning of wood, no of cigarettes per day, low standard cigarette, local drugs, light and menthol cigarettes, cigars and Pipes, little cigars, cigarillos, hookahs, bidis, clove cigarettes, dissolvable products, electronic cigarettes (also referred to as: vape pen, e-hookah, hookah pen), traditional smokeless tobacco products, waterpipes (also referred to as: hookah, shisha, narghile, argileh).

Correlation between CBC and Oxidative Stress in Smokers and Non-smokers: Oxidative stress and the free radical damage which is a reason for cellular structures which is an actual problem in this poisonous world. Extraordinary intensities of free radicals are related with exposure to environmental pollutants, inflammatory diseases, and low antioxidant activity. Free radicals are actually unsteady and reactive with further molecules. Once free radical starts reactions, they incline to increase by chain reactions with cellular material. The chain reactions incline to shows long-term effects and the possible to formation of cellular structure damage. It is significant to think that body requires a convinced quantity of OS to contract with microbes, toxins etc. Equally too much and too little OS is a problem for humans.

Table 4: Correlation between Oxidative Stress and PAH

	Correlated Biomarkers	Male Smoker	Female Smoker	Male Non Smoker	Female Non Smoker
3PAH	MDA	0.43	-0.44	0.14	0.28
	CAT	-0.13	0.05	-0.01	0.39
	SOD	0.09	-0.10	-0 .34	0.04

A blood CBC profile interaction may specify OS exposure to smoking cigarette which interrupts hematological parameters and OS biomarkers adversely, smokers. The consequence appears to be even lower in smokers concerning OS biomarker and antioxidant enzymes activity. Removal of cigarette smoking could break the contrasting effects for smokers, as well as for fit non-smokers in their vicinity. Further elements on a blood CBC profile that may specify OS are a reduced lymphocyte count. The neutrophil/lymphocyte ratio at

approximately point in the future, but a lower lymphocyte count is not a worthy thing. Platelets concentration might also be reduced along with a rise in OS. R.B.C are mainly sensitive to oxidative stress, which can be reason to red blood cell hemolysis.

Correlation between Oxidative Stress and PAHs in Smokers and Non-smokers: PAHs exposure is connected with a rise in oxidative stress in the body and characteristics with small TAC and higher oxidative DNA injury have an expressively advanced risk of lung cancer linked to people with higher TAC and lower oxidative DNA injury. Conversely, the research did not propose a clear essential association because used biological samples collected. Meanwhile in the current study, an approaching investigation was designed to use the estimate of PAHs exposure and OS, no progressive doubt among the exposures to risk factors. Our results expression that relationship among total antioxidant capacity and OS levels in the smoker and non-smoker blood. In never-smokers among, oxidative stress considerably decreased. It is supposed that great amounts of ROS are formed in smokers and the antioxidant capacity as a self-protective mechanism also rises by smoking, thus for the positive correlation among the oxidative stress biomarkers. Though, non-smokers have a lesser antioxidant capacity, inadequate detoxification of ROS may have caused in high levels of OS.

Conclusion: This study has shown that smoking increases white blood cell counts. This study concluded that smoking effected blood cells, specifically WBC and RBC. Same effect was observed in passive smokers. Both carcinogenic and non-carcinogenic PAHs is present in cigarette Benzo(a)pyrene (BaP) is one of the major PAHs present in cigarette and other are anthracene and 1-hydropyrene PAHs originate in the tar fraction of cigarette smoke, these PAHs rise the level of polycyclic aromatic hydrocarbons in smokers. Free radicals are produced by smoking which rise the ROS activity and effect in the rise of levels of TBARS, and CAT. Free radicals rise that consume more of antioxidants such as SOD, which might more effect the decrease in the level of these antioxidants in smokers. Smoking cigarette may have several antagonistic consequences in smokers, with respect to OS and antioxidant biomarkers markers.

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