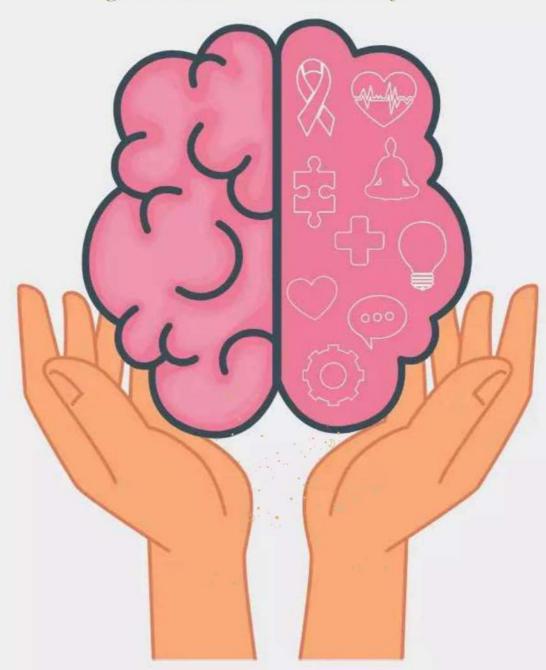
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# STUDY THE EFFECT OF SMOKING ON SOME HEMATOLOGICAL PARAMETERS IN MALES AT AL-NAJAF GOVERNORATE, IRAQ

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### Abstract

In this work, it was studying the effect of cigarette smoking on some blood parameters such as WBC ("LYM" Lymphocyte Percent, "MID" Monocyte Percent, and "GRAN" Granulocyte Percent), RBC ("MCV" Mean Corpuscular Volume, "MCH" Mean Corpuscular Hemoglobin, and "MCHC" Mean Corpuscular Hemoglobin concentration), and PLT ("MPV" Mean Platelet Volume, "PDW" Platelet Distribution Width, and "PCT" Plateleterit) on 125 males from Al-Najaf governorate; (75) smokers and (50) nonsmokers at five ages gropes from 20 to 70 years old. The results showed that the results of LYM, MID, GRAN, MCH, MCHC, MPV, and PDW were non-significant changes in all age groups (p>0.05) in smokers compared with nonsmokers. While, the results MCV and PCT were increasing significant changes in all age groups (p<0.05) for smokers compared with nonsmokers. Also, it is found that there are non-significant changes in MID and MCV comparison between different ages in smokers groups, but other parameters were significant. While in nonsmokers, it is found there are non-significant changes in LYM, GRAN, MPV, and PCT comparison between different ages groups, but other parameters were significant. Therefore, it can be concluded that the cigarette smoking effect to increases for MCV and PCT while no effect on their parameters in the present study such as LYM, MID, GRAN, MCH, MCHC, MPV, and PDW for smokers compared with the same ages of nonsmokers.

**Keywords**: cigarette smoking, hematological parameters, Males, Al-Najaf Governorate, and Iraq.

### Introduction

The evaluation of a complete blood count (CBC) is of utmost importance, as it serves not only as a diagnostic and therapeutic tool for hematologic disorders but also as a means to evaluate an individual's general health status [1]. When establishing a medical diagnosis, choosing a course of treatment, or doing any kind of physiological evaluation, a person's CBC is compared to a reference interval (RI) [2]. Numerous external stimuli, including radiation and smoking, have been identified as contributors to the generation of free radicals, leading to a disruption in the equilibrium between free radicals and the protective mechanisms provided by antioxidants [3]. Cigarette smoking is a serious health problem and most important avoidable causes of death in world [4]. There is substantial evidence linking smoking to several health conditions, including chronic obstructive pulmonary disease, cancer, and atherosclerosis [5-7]. Smoking causes 1.69 million deaths annually due to heart disease, 0.97 million deaths annually due to (COPD), and 0.85 million deaths annually due to lung cancer. [8]. Cigarette smoke has a diverse array of chemical compounds, consisting of around 4000 distinct components. Over the last three to four decades, a substantial amount of

information has been gathered, elucidating the precise chemical makeup of cigarette smoke in terms of both qualitative and quantitative aspects. Several compounds have been identified, including various pyridine alkaloids like nicotine, ammonia, acrolein, phenols, acetaldehyde, and N-nitrosamine. Additionally, polycyclic aromatic hydrocarbons such as benzopyrine, combustion gases like carbon monoxide, nitrogen oxides, and hydrogen cyanide, as well as trace metals and α-emitter radioactive elements such as polonium, radium, and thorium have been detected [8,9]. The act of smoking cigarettes results in the introduction of several harmful substances into the body. These chemicals or their metabolites have the potential to exhibit electrophilic properties, allowing them to interact with biological macromolecules. Alternatively, they may induce oxidative stress by the generation of reactive species or the activation of radical chain reactions [10]. Cigarette smoke contains a significant amount of Reactive Oxygen and Nitrogen Species (ROS and RNS), including nitrogen, alkoxyl, and peroxyl radicals. These factors may induce the generation of additional free radicals, which subsequently trigger lipid peroxidation on the lowdensity lipoprotein (LDL) particle and result in dysfunction of endothelial cells [11]. Numerous highly oxidative and carcinogenic compounds, including polynuclear aromatic hydrocarbons, tobacco-specific N-nitrosamines, and aromatic amines, are present in cigarette smoke [12]. Last but not least, several studies have shown that smoking negatively affects white cell count, differential leukocyte percentages, and indicators of platelet activity [13]. Based on statistics provided by the World Health Organization (WHO), it is estimated that the annual worldwide mortality rate resulting from smoking-related illnesses now stands at around 5 million individuals. Projections indicate that if this prevailing pattern persists, the number of deaths attributable to smoking-related ailments is anticipated to double, reaching 10 million by the year 2025 [14]. Multiple studies have demonstrated that smoking has detrimental effects on human health and serves as a contributing factor for the onset of various pathological conditions and diseases, including chronic obstructive pulmonary disease, cancer, pancreatitis, gastrointestinal disorders, periodontal disease, metabolic syndrome, and certain autoimmune diseases [14,15]. Extensive research has been conducted on the impact of smoking on changes in the hemostatic and fibrinolytic system, antioxidant status, and hematological parameters. However, the findings of these studies have been conflicting. The objective of the current investigation was to assess the influence of secondhand smoke exposure on indicators of complete blood count. Consequently, the current investigation was undertaken to assess the impact of cigarette smoking on some hematological parameters in individuals who smoke compared to a control group of non-smokers of similar age.

### Methology

A case-control study with an analytical approach was undertaken to assess the levels of various white blood cells (specifically Lymphocyte Percent, Monocyte Percent, and Granulocyte Percent), red blood cells (including Mean Corpuscular Volume, Mean Corpuscular Hemoglobin, and Mean Corpuscular Hemoglobin concentration), and platelets (such as Mean Platelet Volume, Platelet Distribution Width, and Plateletcrit) among individuals who smoke cigarettes and hookahs in the Najaf governorate of Iraq. A study was undertaken throughout the first half of the year 2023. The present research used a cross-sectional design and included a sample of 125 male participants who were between the age range of 21 to 70 years and exhibited good health. For the aim of this research, a total of 75 individuals who smoke cigarettes and hookah were

selected as the study group, whereas 50 individuals who do not smoke were chosen as the control group.

Method of data collection: A pre-designed and pre-tested questionnaire was used to gather biosocial data from participants, including information on age, smoking dosage, duration of smoking, and any associated illnesses. The present research aimed to examine the impact of cigarette smoking on haematological markers in a cohort of individuals who were in good health. The research consisted of a cohort of 125 participants, with 75 individuals classified as smokers and 50 individuals classified as non-smokers. The age range of the participants spanned from 21 to 70 years. Informed permission was obtained from each participant in the research, and the study methodology received approval from the Ethical Review Committee. The collection of data pertaining to smoking behaviors and tobacco consumption was facilitated by the use of a self-administered questionnaire, which was completed by the study participants. The participants included in this trial exhibited no signs of current liver and renal illness, chronic pancreatitis, gastrointestinal disease, inflammatory bowel disease, history of ischemic heart disease or diastolic blood pressure, endocrine problems, infection, or hormonal medication.

Sample processing: Participants who identified as smokers and non-smokers were recruited for the laboratory study. A 5 ml syringe was used to extract a blood sample from the participants' veins, which was then transferred into EDTA tubes. Each participant was assigned a unique code, which was then recorded on the respective tubes for both smokers and non-smokers. Subsequently, a manual preparation process was conducted for each tube, followed by their placement on a device known as a (shaker) for a duration of 3-5 minutes. This (shaker) facilitates the movement and rotation of the tubes, ensuring the thorough mixing of the blood samples with the anticoagulant substance, specifically EDTA, contained within the tubes. The purpose of this step is to prevent the coagulation of the samples, thereby preparing them for analysis using the CBC device.

CBC: (complete blood count): The blood cell count evaluations were conducted using the Sysmex automated hematological analyzer, a highly accurate and precise instrument capable of measuring 18 hematological parameters. The Sysmex analyzer primarily utilizes the electronic resistance (impedance) detection technology to accurately count and size the leukocytes, erythrocytes, and platelets. By using three initial hydraulic systems for white blood cells (WBCs), red blood cells (RBCs), and platelets (PLTs), this study aims to demonstrate the method of determining the blood count results of these cells. The findings will be shown on a liquid crystal display (LCD) in the form of a histogram and will also be printed on thermal paper [16].

The parameters examined in the complete blood count (CBC) assay encompassed Lymphocyte Percent (LYM), Monocyte Percent (MID), and Granulocyte Percent (GRAN), as well as Mean Corpuscular Volume (MCV), Mean Corpuscular Hemoglobin (MCH), Mean Corpuscular Hemoglobin concentration (MCHC), Mean Platelet Volume (MPV), Platelet Distribution Width (PDW), and Plateletcrit (PCT).

**Data analysis:** The current research aimed to investigate the disparities in blood parameters between individuals who smoke and those who do not. The Mann-Whitney U test, independent t-test, ANOVA, and Kruskal-Wallis tests were used to assess both

quantitative and qualitative data. The analysis of the association between quantitative variables was conducted by using the Pearson correlation coefficient. A p-value below 0.05 was deemed to be statistically significant. The statistical analysis was performed using IBM SPSS Statistics (version 27 for Windows, USA).

### Results

The results were divided into two groups for non-smokers and smokers. Criteria related to white blood cells were found: (LYM, MID, and GRAN), as well as criteria for red blood cells: (MCV, MCH, and MCHC), in addition to platelet parameters: (MPV, PDW, and PCT), where the results are tabulated in table (1), and table (2). Table 1 showed a significant increase (p<0.05) in MCV and PCT in smokers compared with non-smoker for all age groups. The values of LYM, MID, GRAN, MCH, MCHC, MPV, and PDW were non-significantly decreased (p>0.05) in smoker compared with non- smoker in all age groups. Table 2 showed a significant (p<0.05) in MCH, MCHC, and PDW in smokers and non-smoker with all age group in the present study. Significant changes were observed in LYM, GRAN, MPV, and PCT for smoker with all age group, while no significant changes were observed in MID and MCV for smoker with all age group. But, for non- smoker, it was noted MID, MCV, MCH, MCHC, and PDW significant with all age groups, while non-significant in LYM, GRAN, MPV, and PCT in all age groups. Figures from (1) to (9) show the relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50 y, 51-60y, and 61-70y) for eight parameters that studied in the present study.

Table 1. Comparison between smokers and non-smokers groups for all the parameters.

Parameters	Groups	N	Mean ± SD	P value
LYM	Smoker	75	31.304±7.094	0.903
LINI	Non-smoker	50	31.460±6.793	NS
MID	Smoker	75	5.872± 1.355	0.185
MID	Non-smoker	50	6.220± 1.533	NS
GRAN	Smoker	75	62.824± 7.744	0.718
GRAN	Non-smoker	50	62.320± 7.412	NS
MCV	Smoker	75	83.417± 4.346	0.036
MCV	Non-smoker	50	81.480± 5.844	S
мсн	Smoker	75	28.277± 1.816	0.143
мсн	Non-smoker	50	27.712± 2.469	NS
МСНС	Smoker	75	34.145±1.92187	0.713
MCHC	Non-smoker	50	34.032±1.92187	NS
MPV	Smoker	75	7.252±0.688	0.270
WIFV	Non-smoker	50	7.124±0.535	NS
PDW	Smoker	75	9.796±1.297	0.254
ruw	Non-smoker	50	9.558±0.839	NS
рст	Smoker	75	0.1936±0.158	0.023
PCT	Non-smoker	50	0.1412±0.027	S

HS: High significant difference between groups (p value <0.01)

S: Significant difference between groups (p value <0.05)

NS: Non-significant difference between groups.

Table 2. Comparison between different ages for all parameters in both smokers and non-smokers groups

		Smokers Non-smoker		ers			
Parameters	Age (years)	N	Mean	LSD <sub>0.05</sub> P value	N	Mean	LSD <sub>0.05</sub> P value
	(21-30)	15	32.160 b		10	35.900 a	
	(31-40)	15	33.380 a	0.001	10	30.100 b	0.161
LYM	(41-50)	15	33.400 a	HS	10	29.720 с	NS
	(51-60)	15	33.460 a	113	10	32.300 b	NS NS
	(61-70)	15	24.120 c		10	29.280 c	
	(21-30)	15	5.700 a		10	4.920 d	
	(31-40)	15	5.980 a	0.235	10	6.100 b	0.001
MID	(41-50)	15	5.380 a	NS	10	6.800 b	0.001 HS
	(51-60)	15	6.500 a	l NS	10	7.540 a	
	(61-70)	15	5.800 a		10	5.740 c	
	(21-30)	15	62.140 b		10	59.180 a	
	(31-40)	15	60.640 c	0.001	10	63.800 a	0.342
GRAN	(41-50)	15	61.220 c	0.001	10	63.480 a	380000000000000000000000000000000000000
	(51-60)	15	60.040 c	HS	10	60.160 a	NS
	(61-70)	15	70.080 a		10	64.980 a	
	(21-30)	15	82.460 a		10	82.360 b	
	(31-40)	15	83.387 a	0.072	10	81.240 с	0.0001
MCV	(41-50)	15	82.800 a	0.073	10	82.620 b	0.0001
	(51-60)	15	86.220 a	NS	10	75.020 d	5.020 d 5.160 a 3.700 a
	(61-70)	15	82.220 a		10	86.160 a	
	(21-30)	15	28.093 b		10	28.700 a	
	(31-40)	15	28.480 b		10	27.680 b	
MOT	(41-50)	15	28.833 b	0.009	10	28.420 a	0.001
MCH	(51-60)	15	29.060 a	HS	10	25.020 с	HS
	(61-70)	15	26.920 с	İ	10	28.740 a	
	(21-30)	15	35.120 a		10	34.820 a	
	(31-40)	15	34.187 b		10	34.120 a	1
MCHC	(41-50)	15	34.860 b	0.006	10	34.440 a	0.025
	(51-60)	15	33.740 b	HS	10	33.360 b	S
	(61-70)	15	32.820 c		10	33.420 b	1
	(21-30)	15	6.593 b		10	6.920 a	
	(31-40)	15	7.413 a		10	6.900 a	
MPV	(41-50)	15	7.333 a	0.001	10	7.500 a	0.060
	(51-60)	15	7.440 a	HS	10	7.240 a	NS
	(61-70)	15	7.480 a		10	7.060 a	1
	(21-30)	15	8.627 b		10	9.320 a	
	(31-40)	15	10.247 a		10	9.060 b	1
PDW	(41-50)	15	9.927 a	0.001 10 9.870.a	0.038		
os misteres (utile)	(51-60)	15	9.740 a	HS	10	10.080 a	S
	(61-70)	15	10.440 a	1	10	9.460 b	
	(21-30)	15	0.182 b		10	0.151 a	1000 100000
PCT	(31-40)	15	0.169 b	0.005	10	0.144 a	0.405
	(41-50)	15	0.154 b	HS	10	0.144 a	NS

(51-60)	15	0.135 b	10	0.140 a
(61-70)	15	0.328 a	10	0.127 a

HS: High significant difference between groups (p value <0.01)

<sup>.</sup>groups with different letters are significant difference.



Figure (1): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on LYM.

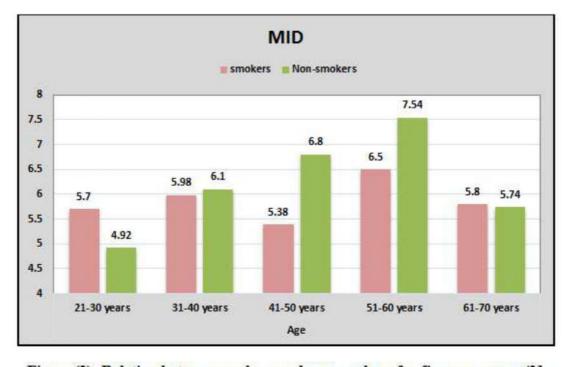


Figure (2): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on MID.

S: Significant difference between groups (p value <0.05)

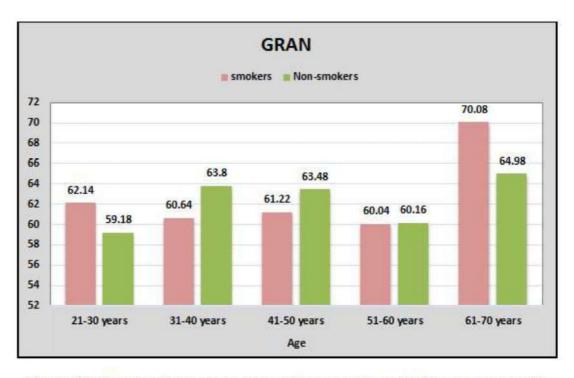


Figure (3): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on GRAN.

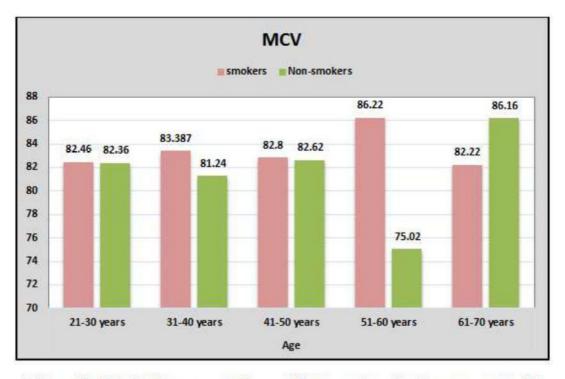


Figure (4): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on MCV.



Figure (5): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on MCH.



Figure (6): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on MCHC.

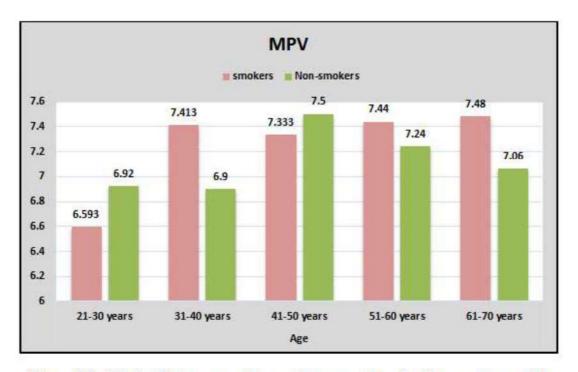


Figure (7): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on MPV.

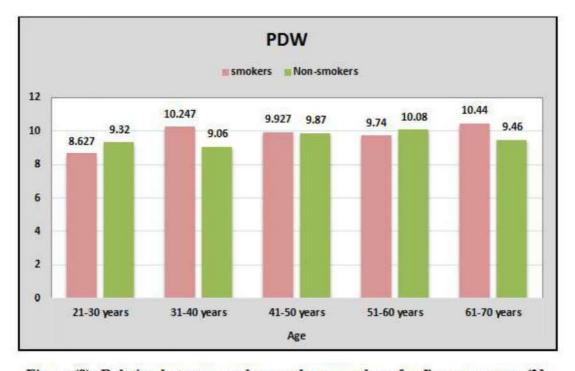


Figure (8): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on PDW.

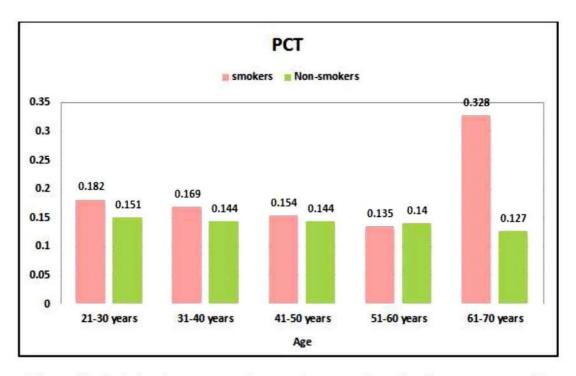


Figure (9): Relation between smokers and non-smokers for five age groups (21-30y, 31-40y, 41-50y, 51-60y, and 61-70y) on PCT.

### Discussion:

The present investigation revealed notable disparities in hematological parameters between individuals who smoke and those who do not smoke. Specifically, the mean corpuscular volume (MCV) and plateletcrit (PCT) were found to be considerably elevated in smokers compared to non-smokers (p<0.05). Additionally, the analysis reveals that there is no discernible statistically significant difference in the levels of LYM, MID, GRAN, MCH, MCHC, MPV, and PDW. MCV, MCH, and MCHC are three primary hematological parameters used to assess the mean cellular volume and hemoglobin content of erythrocytes. An elevation in mean corpuscular volume (MCV) levels was seen among those who smoke compared to those who do not smoke (p<0.05). This finding aligns with prior research conducted in this area [17,18]. The mean corpuscular volume (MCV) serves as an indicator of red blood cell size. Deviations from the normal size can suggest the presence of anemia, with smaller or larger red cells indicating such a condition. Elevated MCV levels may be indicative of megaloblastic, hemolytic, pernicious, or macrocytic anemia, typically associated with deficiencies in iron and folic acid [19]. According to our data, smoking has a serious negative impact on haematological variables including hemoglobin and hematocrit, particularly the mcv parameter. The overall erythrocyte count was almost the same in smokers and non-smokers. There was a statistically significant difference in erythrocyte values between male smokers and female smokers, with the former group exhibiting greater values. In the present research, it was observed that the levels of hemoglobin were found to be substantially higher in those who smoke compared to those who do not smoke, irrespective of gender. However, no statistically significant variation was seen in the levels of hematocrit between these two groups of participants. In contrast, it was shown that male smokers had considerably higher hematocrit levels compared to female smokers. The observed

rise in hemoglobin (Hb) levels within the group of smokers is consistent with earlier research findings, which have shown considerably higher hematocrit and Hb levels among smokers. Furthermore, among smokers, there is a notable increase in red blood cell (RBC) count as the intensity of smoking escalates. In their investigation, Whitehead et al. (year) noticed a substantial rise in hemoglobin concentration and hematocrit among those who smoked more than 10 cigarettes per day [20]. The rise in hemoglobin concentration is thought to be facilitated by the presence of carbon monoxide, and several researchers have proposed that the elevation in hemoglobin levels seen in the blood of smokers may serve as a compensatory mechanism [21]. The binding of carbon monoxide to hemoglobin results in the formation of carboxyhemoglobin, which is an inert variant of hemoglobin that lacks the ability to transport oxygen. Carboxyhemoglobin furthermore induces a leftward shift in the dissociation curve of hemoglobin, leading to a diminished capacity of hemoglobin to transport oxygen to the tissue [22]. In order to counterbalance the reduced capacity for oxygen delivery, those who smoke have an elevated amount of hemoglobin compared to those who do not engage in smoking behavior. The elevated quantities of red blood cells and hematocrit levels seen in male individuals who smoke may be attributed to the occurrence of tissue hypoxia resulting from heightened production of carboxyhemoglobin. This, in turn, triggers an augmented release of erythropoietin, so promoting erythropoiesis [15]. The presence of carbon monoxide in tobacco smoke contributes to an elevation in capillary permeability, resulting in a reduction in plasma volume. This effect resembles the characteristics of polycythemia, a condition marked by a higher proportion of erythrocytes in the total blood volume. Consequently, elevated hematocrit values are observed as well. MCV, MCH, and MCHC are three primary hematological parameters used to assess the mean cellular volume and hemoglobin content of erythrocytes. The present research found that smokers exhibited considerably higher mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) values compared to non-smokers. However, there were no significant differences seen in mean corpuscular hemoglobin concentration (MCHC) and red cell distribution width (RDW) values between smokers and nonsmokers. Other studies have also confirmed that smokers tend to have higher values of mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) compared to non-smokers. These values surpass the reference interval and are indicative of certain diseases such as kidney dysfunction, hyperuricemia, hypertension, and hypercholesterolemia. The findings of this research are in opposition to those of a previous study that failed to observe any statistically significant alterations in mean corpuscular volume (MCV) between smokers and nonsmokers. A considerably low value was seen among those who smoke. In contrast to our findings, Salamzadeh conducted a study in which the mean corpuscular volume (MCV) was used to assess the size of red blood cells. The presence of red cells that are smaller or larger than the normal size in Salamzadeh's study indicated the presence of anemia. In our study, elevated levels of MCV were observed, suggesting that the subjects may be experiencing megaloblastic, haemolytic, pernicious, or macrocytic anemia. These types of anemia are typically caused by deficiencies in iron and folic acid. The mean corpuscular hemoglobin (MCH) refers to the average weight of hemoglobin contained inside an individual red blood cell. On the other hand, the mean corpuscular hemoglobin concentration (MCHC) represents the quantity of hemoglobin in a given volume of densely packed red blood cells. Our research found a statistically significant increase in the number of leukocytes in male smokers

compared to non-smokers. Moreover, the leukocyte count values exhibited a statistically significant increase among male individuals who smoke [23].

### Conclusion

We analyzed pooled data of smoking and non-smoking health examiners obtained from 125 subjects across Najaf governorate who used Sysmex XE-2100 to determine blood parameters using (CBC) and also evaluated patterns of changes in CBC parameters according to age and smoking habit. it can be concluded that the cigarette smoking causes significant higher increases in MCV and PCT, while no significant in LYM, MID, GRAN, MCH, MCHC, MPV, and PDW.

### References

- [1] Nah, E. H., Kim, S., Cho, S., & Cho, H. I. (2018). Complete blood count reference intervals and patterns of changes across pediatric, adult, and geriatric ages in Korea. Annals of Laboratory Medicine, 38(6), 503.
- [2] Friedberg, R. C., Souers, R., Wagar, E. A., Stankovic, A. K., & Valenstein, P. N. (2007). College of American Pathologists The origin of reference intervals. Arch Pathol Lab Med, 131, 348-57.
- [3] Ogawa, K., Tanaka, T., Nagoshi, T., Sekiyama, H., Arase, S., Minai, K., ... & Yoshimura, M. (2015). Increase in the oxidised low-density lipoprotein level by smoking and the possible inhibitory effect of statin therapy in patients with cardiovascular disease: a retrospective study. BMJ open, 5(1), e005455.
- [4] Jha, P., Jacob, B., Gajalakshmi, V., Gupta, P. C., Dhingra, N., Kumar, R., ... & Peto, R. (2008). A nationally representative case—control study of smoking and death in India. New England journal of medicine, 358(11), 1137-1147.
- [5] Pasupathi, P., Saravanan, G., & Farook, J. (2009). Oxidative stress bio markers and antioxidant status in cigarette smokers compared to nonsmokers. Journal of Pharmaceutical Sciences and Research, 1(3), 55.
- [6] Padmavati, S. (2002). Prevention of heart disease in India in the 21st century: need for a concerted effort. Indian Heart J.
- [7] Rani, M., Bonu, S., Jha, P., Nguyen, S. N., & Jamjoum, L. (2003). Tobacco use in India: prevalence and predictors of smoking and chewing in a national cross sectional household survey. Tobacco control, 12(4), e4-e4.
- [8] Pasupathi, P., Saravanan, G., & Farook, J. (2009). Oxidative stress bio markers and antioxidant status in cigarette smokers compared to nonsmokers. Journal of Pharmaceutical Sciences and Research, 1(3), 55.
- [9] Anazawa, T., Dimayuga, P. C., Li, H., Tani, S., Bradfield, J., Chyu, K. Y., ... & Cercek, B. (2004). Effect of exposure to cigarette smoke on carotid artery intimal thickening: the role of inducible NO synthase. Arteriosclerosis, thrombosis, and vascular biology, 24(9), 1652-1658.
- [10] Orhan, H., Evelo, C. T., & Şahin, G. (2005). Erythrocyte antioxidant defense response against cigarette smoking in humans—the glutathione S-transferase vulnerability. Journal of biochemical and molecular toxicology, 19(4), 226-233.

- [11] Seet, R. C., Lee, C. Y. J., Loke, W. M., Huang, S. H., Huang, H., Looi, W. F., ... & Halliwell, B. (2011). Biomarkers of oxidative damage in cigarette smokers: which biomarkers might reflect acute versus chronic oxidative stress? Free Radical Biology and Medicine, 50(12), 1787-1793.
- [12] IARC Working Group on the Evaluation of Carcinogenic Risks to Humans, World Health Organization, & International Agency for Research on Cancer. (2004). Tobacco smoke and involuntary smoking (Vol. 83). Iarc.
- [13] Lymperaki, E., Makedou, K., Iliadis, S., & Vagdatli, E. (2015). Effects of acute cigarette smoking on total blood count and markers of oxidative stress in active and passive smokers. Hippokratia, 19(4), 293.
- [14] Buist, A. S., Vollmer, W. M., & McBurnie, M. A. (2008). Worldwide burden of COPD in high-and low-income countries. Part I. The Burden of Obstructive Lung Disease (BOLD) Initiative [State of the Art Series. Chronic obstructive pulmonary disease in high-and low-income countries. Edited by G. Marks and M. Chan-Yeung. Number 6 in the series]. The international journal of tuberculosis and lung disease, 12(7), 703-708.
- [15] Malenica, M., Prnjavorac, B., Bego, T., Dujic, T., Semiz, S., Skrbo, S., ... & Causevic, A. (2017). Effect of cigarette smoking on haematological parameters in healthy population. Medical Archives, 71(2), 132.
- [16] Ahmed, O. A. (2016). Effects of smoking cigarette on white blood cell and platelet parameter on a sample of normal subject in Rania city. Imperial Journal of Interdisciplinary Research, 2(9), 887-92.
- [17] Q AL-temimi, S. (2017). The effect of cigarette smoking on some blood parameters, blood pressure and renal function test. journal of kerbala university, 13(1), 1-10.
- [18] Muhammad, A.; Sajjad, K. .; Zubaida, U.and Arif, M (2013). Effect of cigarette smoking based on hematological parameters: comparison between male smokers and nonsmokers. .turkey J biochem. 38 (1): 75–80.
- [19] Yousif Y. (2015). Effects of Cigarette Smoking on Blood Rheology and Biochemistry. I.J.S.R. 4: 3.
- [20] Whitehead, T. P., Robinson, D., Allaway, S. L., & Hale, A. C. (1995). The effects of cigarette smoking and alcohol consumption on blood haemoglobin, erythrocytes and leucocytes: a dose related study on male subjects. Clinical & Laboratory Haematology, 17(2), 131-138.
- [21] Raub, J. A., & Benignus, V. A. (2002). Carbon monoxide and the nervous system. Neuroscience & Biobehavioral Reviews, 26(8), 925-940.
- [22] Belanger, A. (2015). Biophysical studies aimed at potential therapies of hemoglobin associated diseases. Wake Forest University.
- [23] Barreto SG. How does cigarette smoking cause acute pancreatitis? Pancreatology: official journal of the International Association of Pancreatology. 2016;16(2):157–63.