

Effects of Smoking on Hematological Parameters among Baghdad City

Wed Abbas Mohammed

Al-Qasim Green University Babydom,Iraq
wed.abbas@gmail.com

Article Info:

Submitted:	Revised:	Accepted:	Published:
Apr 23, 2025	May 22, 2025	Jun 2, 2025	Jun 7, 2025

Abstract

Cigarette smoking is widely recognized as a major risk factor in the development and progression of various chronic diseases, including atherosclerosis and chronic obstructive pulmonary disease. In Iraq, smoking remains prevalent across both rural and urban populations. It has been associated with significant alterations in inflammatory markers and hematological parameters. This study aimed to examine smoking habits and their effects on key blood parameters, including Total Leukocyte Count (TLC), Differential Leukocyte Count, Total Red Blood Cell Count (TRBC), Hemoglobin (Hb) concentration, and Packed Cell Volume (PCV). A total of 80 healthy adult males aged 20 to 56 years from Baghdad were enrolled, comprising 40 smokers and 40 non-smokers. Participant classification was based on a self-administered questionnaire. The results revealed that TLC, TRBC, Hb concentration, PCV, eosinophils, and lymphocytes were elevated in both light and heavy smokers compared to non-smokers, while neutrophil and monocyte levels were reduced. These findings suggest that cigarette smoking induces measurable changes in hematological profiles, which may contribute to its role in disease pathogenesis.

Keywords: Smoking; Hemoglobin; Packed Cell Volume; Leukocyte Count; Blood parameters

INTRODUCTION

Cigarette smoke is made up of a combination of organic and inorganic compounds that are produced when tobacco is burned. Originally composed of about 4000 complex chemical compounds, 60 of these compounds have been proven to increase the risk of developing cancer, while other harmful compounds can cause heart and lung diseases. Nicotine is a substance found in tobacco that makes quitting smoking challenging. Just a few seconds of inhalation, nicotine enters the brain.. Nicotine promotes the release of neurotransmitters, which are brain chemicals that assist control mood and behavior. and the nightshade family of plants naturally contain nicotine, an alkaloid (most predominantly in tobacco and *Duboisia hopwoodii*) [1].

With the exception of two nicotinic receptor subunits (nAChR-9 and nAChR-10), where it works as a receptor antagonist, it is a receptor agonist at the majority of nicotinic acetylcholine receptors (nAChRs)[2-4]. It is made from tobacco leaves (*Nicotiana tabacum*), and people have enjoyed using it for generations. Posselt and Reimann's *Nicotiana tabacum* was the source of the first known source of nicotine in 1828. It was given that name in honor of Jean Nicot, who in 1560 made the first introduction of *Nicotiana tabacum* to the French court [5]. There are nine primary side effects of nicotine replacement therapy: hiccups, skin responses, lightheadedness, nausea/vomiting, gastrointestinal symptoms, sleep/dream issues, non-ischemic palpitations, and chest discomfort. [6].

when At sufficiently high doses, nicotine may result in nausea, vomiting, diarrhea, salivation, bradyarrhythmia, and possibly seizures, hypoventilation, and death.[7]

The harms of nicotine : It can cause an increase in blood pressure, heart rate, blood flow to the heart, and narrowing of the arteries. Nicotine may also contribute to the hardening of artery walls, which in turn can lead to a heart attack. This chemical can stay in your body for 6-8 hours depending on how often you smoke. Smokers are more likely than non-smokers to develop heart disease, stroke and lung cancer[8].

Smoke from cigarettes is a combination of organic and inorganic chemicals that are produced when tobacco is burned [9]; 60 of these compounds have been shown to increase the risk of developing cancer, while other toxic components are linked to heart and lung illnesses. [10] The following are a some of these hazardous compounds: Nicotine damages

the lining of the arteries and causes significant changes in heart rate and blood circulation. In addition, carbon monoxide in cigarette

smoke binds to hemoglobin in red blood cells, decreasing the blood's ability to transport oxygen as a result [11,12]. Ammonia, benzene, formaldehyde, and cyanide chemicals are all poisonous and detrimental to the liver and kidney cells. Methanol alcohol, which harms the central nervous system and visual nerve.[13-14]

Depending on how much and how it is used (smoked, snuffed, or chewed), tobacco usage has a substantial impact on health. Smoking's harmful effects on health (smoking is the most prevalent Lung cancer and cardiovascular illness are both on the

rise as a result of tobacco use[15-16]. Smoking is a significant risk factor for heart attacks, strokes, cancer, chronic obstructive pulmonary disease (COPD), which includes emphysema and chronic bronchitis, and disorders of the heart, liver, and lungs (particularly lung cancer, cancers of the larynx and mouth, and pancreatic cancer). Hypertension and peripheral vascular disease are also caused by it. The consequences vary depending on the length of time and quantity of smoking. The chance of developing these illnesses is increased by smoking earlier in life and cigarettes with more tar. Moreover, secondhand smoking or environmental tobacco smoke has been linked to harmful health consequences in persons of all ages [17].

Over 5000 compounds, including tars and carbon monoxide (CO), are included in cigarette smoke. Several of these compounds are thought to be harmful to human health. [18,19] Because CO may diffuse quickly and binds to hemoglobin (Hb) strongly in alveolar capillaries (with a binding affinity that is 200–250 times higher than that of O₂), tissue hypoxia is caused by the synthesis of carboxyhemoglobin (HbCO), which raises Hb and red blood cell levels (RBCs). [20].

Smokers' levels of HB and hematocrit may be higher. RBC count and daily cigarette consumption have been found to be directly correlated. [21] White blood cells (WBCs) are also negatively impacted by tobacco use in smokers of both sexes. According to studies, smokers of both sexes have considerably more leukocytes than nonsmokers do, as well as elevated levels of neutrophils, lymphocytes, leukocytes, monocytes, eosinophils, and basophils. [22] Because of the effects of CO on erythrocytes, morphological alterations in complete blood count (CBC) reveal a shift in mean corpuscular volume (MCV) [23]. CO hypoxia is the cause of this, which increases the need for erythrocytes. The requirement

cannot be supplied as a result of vitamin B₁₂ being diverted by cyanide, which changes the shape of RBCs[24]. Both also Many hematological parameters are significantly impacted by smoking. Smoking 10 cigarettes a day results in slightly higher Hb, PCV, and MCV, which is likely due to the accumulation of carboxy hemoglobin in the blood as well as a decrease in plasma volume. Some effects are transient, and the severity of them varies between individuals as well as by the number and type of cigarettes smoked The carboxy hemoglobin level rises by 1% after one cigarette. Heavy smokers' carboxyhemoglobin makes about 4-5% of their total hemoglobin, which might cause polycythemia. In addition to an increase in lymphocyte count in some circumstances (increase in CD4 positive), leucocyte count rises primarily as a result of an increase in neutrophils and neutrophil function , Smokers often have greater platelet counts than non-smokers, but when they stop smoking, their counts drop quickly. Furthermore, smoking raises blood cholesterol levels, which may lead to the buildup of heat tissue in the arteries [25].

The heart receives oxygen and nourishment through the coronary arteries. The coronary arteries may develop plaque, or fatty deposits, over time. This can decrease blood flow to the heart and raise the possibility of a heart attack. The constriction and blockage of coronary arteries are hastened by smoking. When anything, generally a blood clot, blocks the flow of blood to the heart, a heart attack occurs. Heart muscle starts to deteriorate in the absence of oxygen and nutrition. The degree of irreversible cardiac muscle damage determines the heart attack's severity. Other blood arteries are harmed by smoking as well. Peripheral arterial disease, which impairs blood flow, occurs when the arteries supplying the arms and legs with blood are impacted. The complications of peripheral arterial disease include amputation, gangrene, and blood clots. [26].

Respiratory system has been scientifically demonstrated that the lung's absorptive capacity steadily declines with age, with nonsmokers experiencing a volume loss of 20 to 30 mm year. Nevertheless, in smokers, these numbers increase to 60 mm annually. It was also demonstrated that the number drops back to normal levels for non-smokers when smoking is stopped early (before it does any significant harm to the lungs). Some smokers consider the persistent coughing and ongoing sputum discharge brought on by smoking to be normal. It is, however, an indication of the harmful effects of smoking.[27-28].

The color of the sputum may initially be clear, but occasionally, especially during the winter, it may turn yellow, green, or black because of the microorganisms that flourish in

such an environment. Sputum builds up as a result of overactive glands in the lining of the airways of the lungs, which makes it easier for dust, debris, and bacteria to accumulate and enter the airways, where they then constrict and make breathing difficult.

One of the most dangerous diseases for the body is chronic obstructive bronchitis, which is caused by smoking and is sometimes referred to as "smoker's asthma" or "smoker's asthma.". Smoking impairs the ability to breathe regularly and frequently causes enlargement of the alveoli due to the ongoing breakdown of the alveolar walls, which reduces the body's capacity to absorb oxygen from the environment.

One of the most dangerous diseases for the body is chronic obstructive bronchitis, sometimes known as "smoker's asthma," which is caused by persistent smoking.

Smoking impairs the capacity to breathe regularly and frequently causes the alveoli to swell because smoking continuously erodes the walls of the alveoli, which reduces the body's ability to absorb oxygen from the air.

The quantity of cigarettes smoked and the length of time spent smoking are other factors that affect the development of carcinoid tumors. The danger increases with the length of the time. Additional elements include the quantity of smoke inhaled, the cigarette's length, the quantity of dangerous substances and carcinogens, how long they linger in the mouth, smoking before the age of reason, and lastly the absence of a filter tip[26-28].

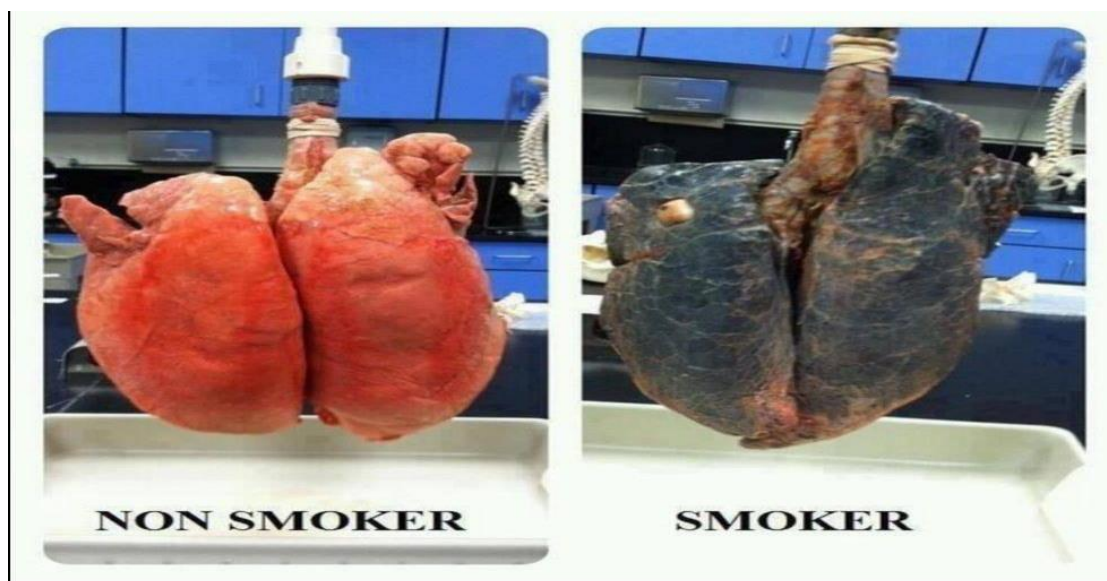


Figure 1. The lung for the smoker and non smoker

MATERIALS AND METHODS

The study was conducted during the period from October to April 2025, forty people smoked (cigarettes, hookahs, electronic cigarettes). The effect of smoking on some blood parameters was studied, including red and white blood cell counts, hemoglobin, blood groups, and a control sample. 40 healthy blood donors .

Appendices provide a list of the apparatus, chemicals, biological materials, and kits that were utilized to complete the current investigation.

Materials and tools are (Westergren glass tubes , EDTA tube , Pipette , Westergren tube rack , tube contain the Sodium Citrate , Slides ,Anti-A, Anti-B, Anti-D drops)

Firstly: Erythrocyte Sedimentation Rate (ESR) Test:

withdraw blood from the smoker . put the blood in EDTA tube , transfer 1 cc the blood from EDTA tube to sodium citrate tube , mix so that the blood mixes with Sodium citrate in tube ,Fill westergren pipette up to zero mark ,place westergren pipette exactly in vertical position for 1 hr and we record the result.

Second: Blood Group Test

We draw blood from a smoker , put the blood in an EDTA tube , took three drops of blood and put it on the slide , put anti-A, anti-B and anti-D drops on each drop of blood ,Move the slide a bit and record the results

Third: Complete Blood Count (CBC) Test

Draw 1 cc blood sample , Place the blood sample in an EDTA tube and shake the tube slowly until the blood mixes with the anticoagulant. , put the sample on a roller mixer for two minutes, so that the blood mixes well with EDTA , make sure that there is a paper in the printer connected to the CBC device , make the pipette of the device reach the end of the tube so that it absorbs the blood properly, while pressing the large button located behind the pipette , hear the sound of the device informing us that the blood collection process is completed in the pipette,quickly withdraw the tube, the device continues to count blood components for a minute , After that, the reading and printing process takes place.

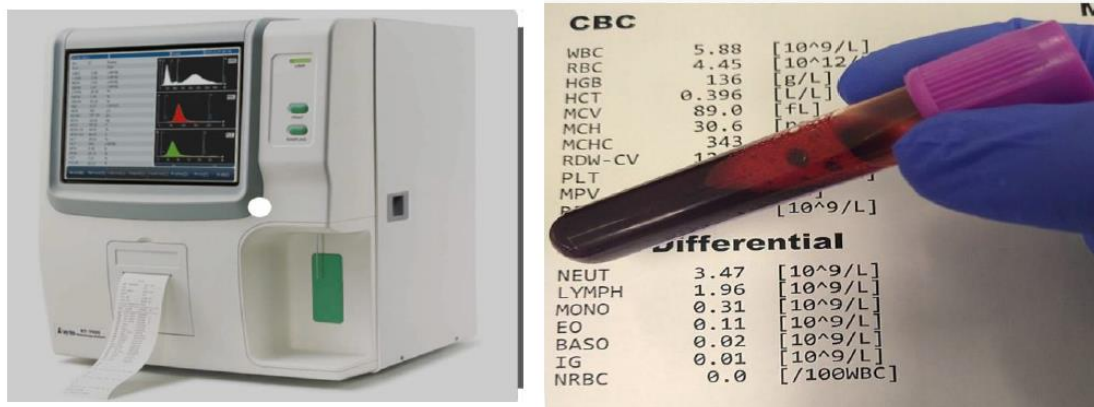


Figure 2. Complete Blood count (CBC) test

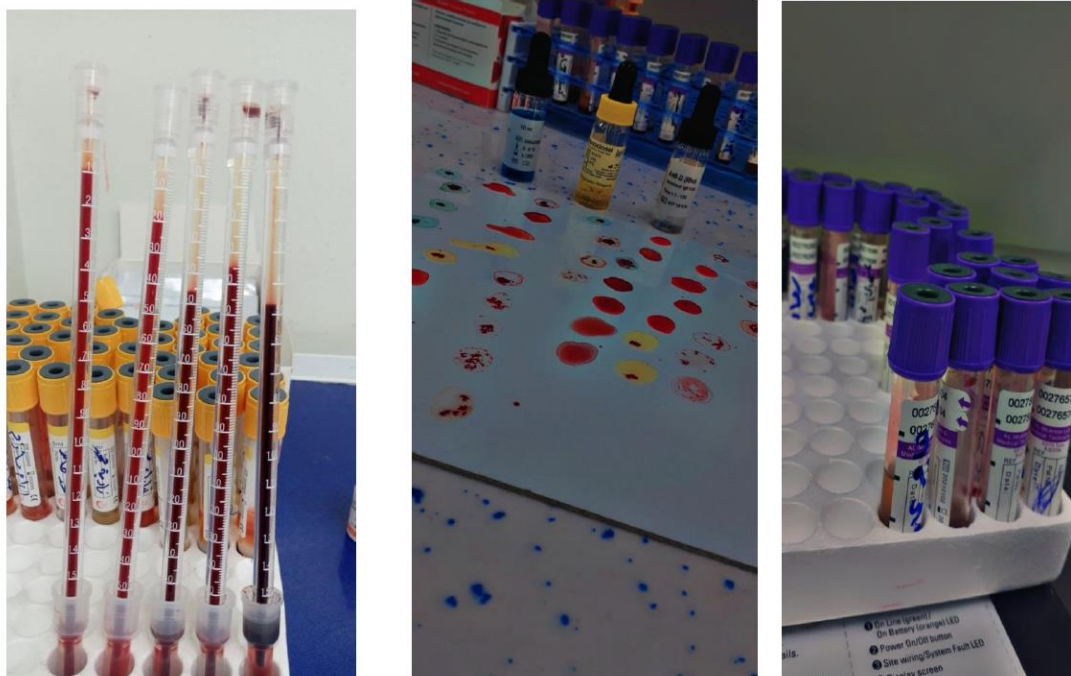


Figure 3. ESR TEST, Blood Group Test, EDTA tube.

RESULTS AND DISCUSSION

Distributing age group in smoker revealed that < 30 year age group had the highest frequency (42.0%), while the age group 30-40 year recorded the lowest frequency (20.0%). Among control, the age group < 30years had the highest frequency (45.0%), followed by the age group 30-40 year (40.0%).

Table 1. Distribution of age group based on numbers of smokers and non-smokers

Age Group (year)	Smoking (N =40)		Control (N = 40)	
	N	%	N	%
< 30	17	42.0	18	45.0
30 – 40	8	20.0	16	40.0
> 40	15	38.0	6	15.0

Distributing of Period of smoking (Years), Smoking (**arghile**) revealed that <10 and >20 year age group had the highest frequency (38.0%), while the age group 10-20 year recorded the lowest frequency (24.0%). Among smoking(**cigarettes**), the age group < 10years had the highest frequency (47.0%), followed by the age group 10-20 year (32.0%).

Table 2. Distribution of Period of smoking (Years) based on numbers of smokers (arghile) and smokers(cigarettes)

Period of smoking (Years)	Smoking (arghile)		smoking(cigarettes)	
	21	%	19	%
< 10	8	38.0	9	45.0
10 – 20	5	24.0	6	32.0
> 20	8	38.0	4	21.0

Hematological Parameters

A complete blood type would describe each of the 40 blood groups, and an individual's blood type is one of many possible combinations of blood-group antigens[29]. Almost always, an individual has the same blood group for life, but very rarely an individual's blood type changes through addition or suppression of an antigen in infection, malignancy, or autoimmune disease.[20][30-33].

Table 3. Distribution of Hematological Parameters

Blood group and Rh	Smoking (arghile)	percentage (%)	smoking(cigarettes)	percentage (%)
A	5		4	
B	6		5	
AB	5		5	

Blood group and Rh	Smoking (arghile)	percentage (%)	smoking(cigarettes)	percentage (%)
O	5		5	
Rh positive	16		15	80
Rh negative	5		4	20
Total	21	100%	19	20

Forty samples of smokers were studied and compared with forty others who did not smoke for comparison with the variable of blood groups. Among our results, the difference in blood groups was similar, with no variation or specific effect.

Tobacco smoking has been correlated to cause several major morphological and biochemical problems in individuals. In this study hematological parameters. WBC count is perhaps the most useful, inexpensive and simple biomarker for endothelial damage[34].

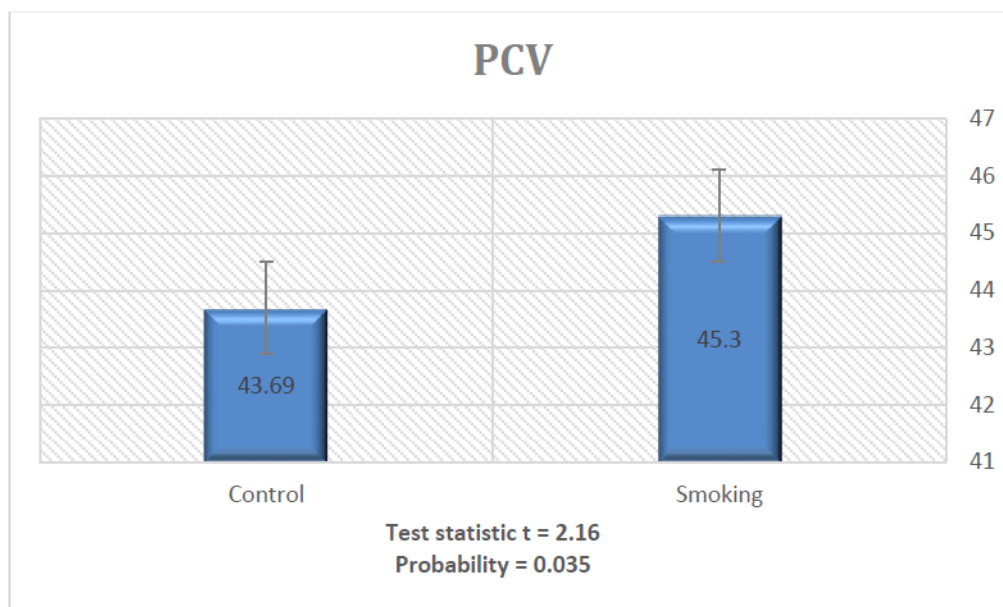
Table 4. comparison of smokers & controls with-Value

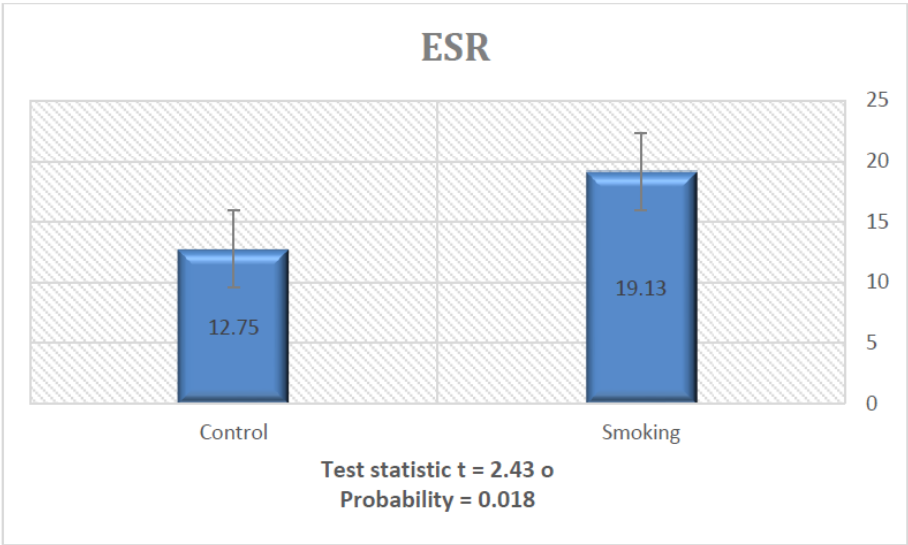
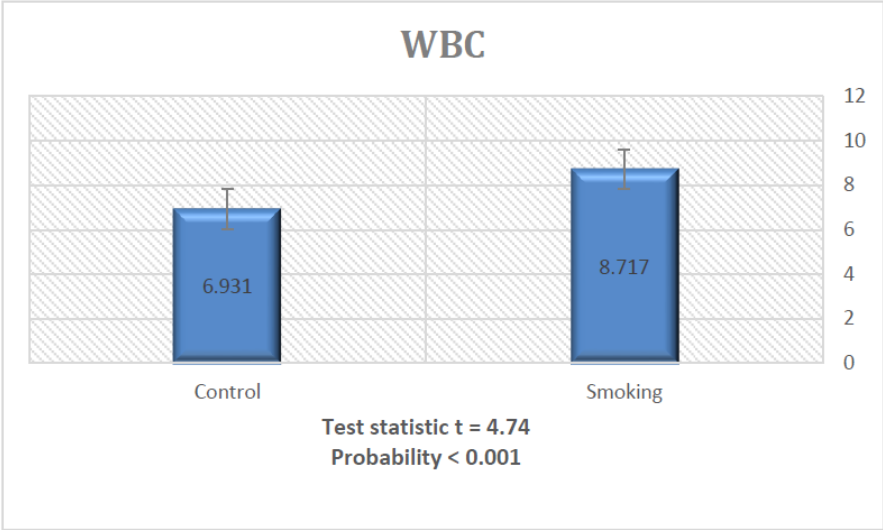
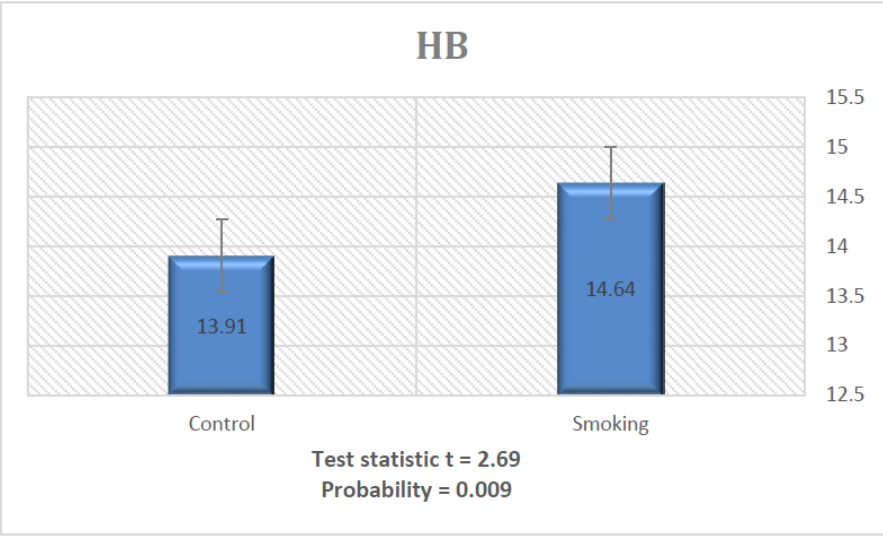
Parameters	Smokers	Controls	P-values
PCV%	45.30±4.035	43.69±2.472	0.035
Hb%	14.64±1.400	13.91±1.007	0.009
WBC (10 ³ /μL)	8.717± 1.887	6.931±1.454	< 0.001
RBC (million cells/ μL)	5.206±0.5116	5.037±0.5116	0.094
ESR (mm/hour)	19.13±14.43	12.75± 8.24	0.018
PLT (10 ³ /μ)	242.25 ±72.53	260.28 ±60.24	0.1066

Significantly higher WBC count compared to non-smokers < 0.001 The high WBC count in male smokers in this study is consistent with other published reports [35,36].

The mechanism for smoking-induced increase in WBC count is not clear. It has been suggested that inflammatory stimulation of the bronchial tract induces an increase in inflammatory markers in the blood but it has also been The high WBC count in our male smoking subjects may also suggest that they might be at greater risk for developing atherosclerosis and cardiovascular diseases than non-smokers. Suggested that nicotine may induce an increase in blood lymphocyte counts [37,38]. Observed that hemoglobin values were significantly high in smokers (P=0.009). Elevated levels of hemoglobin are correlated with increased numbers or sizes of RBCs. RBC values were significantly high in smokers than those of non-smokers ,and are consistent with other investigations [39-41]. It is reported that high level RBC, WBC and Hematocrit are associated with blood viscosity and

clotting in smokers [42,43]. High level of RBC is termed as polycythemia and very high RBC mass slows blood velocity and increase the risk of intravascular clotting, coronary vascular resistance, decreased coronary blood flow, and a predisposition to thrombosis [43]. PCT were observed significantly low in smokers than non-smokers ($P=0.017$). Low PCT value indicates the platelet abnormality resulted by absence of a bone marrow response to a peripheral demand for platelets. We did not find any significant changes in platelets (PLT), mean platelet volume (MPV) or platelet distribution width (PDW) between smokers and non-smokers. The previous reports have shown that chronic smoking causes platelet activation and smoking cessation improves platelet function [44,45]. However, Butkiewicz et al. studied the effect of smoking on platelet activation and some morphological parameters including MPV and they did not find any effect of smoking on MPV [46]. Similarly, Arslan et al. investigated the effects of smoking on MPV in young healthy male population (smokers 56 and non-smokers 46) and they found no significant difference in MPV between the smoking and non-smoking healthy male participant [47]. The ESR was somewhat higher in smokers than the average ESR of nonsmokers, but there was no discernible increase. Another report discovered that smoking induces an increase in the mean ESR; this followed a progressive pattern as the number of cigarettes smoked increased [48].





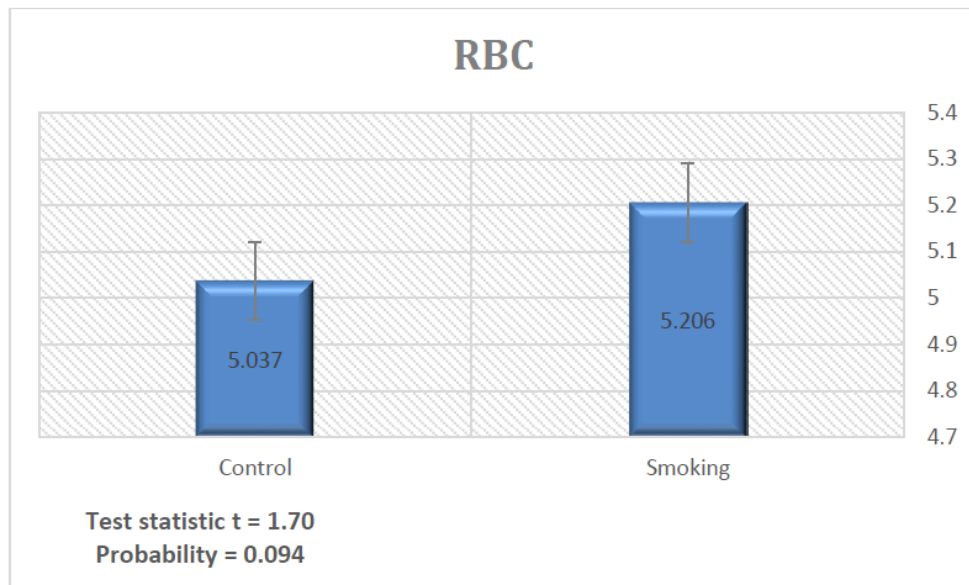


Figure 4. shows comparison between Control & Smoking of Hematological Parameters

CONCLUSION

From the present study, we can concluded that continuous cigarette smoking increases erythrocyte count, hemoglobin concentration, hematocrit, leukocyte count, mean corpuscular volume and mean corpuscular hemoglobin concentration and these alterations might be associated with a greater risk for developing atherosclerosis.

Acknowledgment

I would like to express my deepest gratitude and thanks to Dr. Mohamed Obeid supervisors for his suggestion and planning of the scheme of this study and the inspiring guidance, counseling, encouragement and support. I would also like to thank him deeply for all the help and thoughtfulness; He showed me through the difficult times for my business. Do not forget the laboratory Department-Baghdad hospital and all the working staff.

Recommendations

One of the most important recommendations about smoking is the need to avoid smoking of all kinds, quantities and forms Avoid smoking near pregnant women, children and people with various diseases The harm of smoking in the long run and that the effect of side smoking is more difficult and greater than the effect of direct smoking .

REFERENCES

1. Fagerström, K. (2014, December). Nicotine: Pharmacology, toxicity and therapeutic use. *Journal of Smoking Cessation*, 9(2), 53–59. <https://doi.org/10.1017/jsc.2014.27>
2. International Union of Basic and Clinical Pharmacology (IUPHAR). (n.d.). Nicotinic acetylcholine receptors: Introduction. *IUPHAR Database*. Retrieved September 1, 2014, from <https://www.guidetopharmacology.org>
3. Malenka, R. C., Nestler, E. J., & Hyman, S. E. (2009). Autonomic nervous system. In A. Sydor & R. Y. Brown (Eds.), *Molecular neuropharmacology: A foundation for clinical neuroscience* (2nd ed., p. 234). McGraw-Hill Medical.
4. Kishioka, S., Kiguchi, N., Kobayashi, Y., & Saika, F. (2014). Nicotine effects and the endogenous opioid system. *Journal of Pharmacological Sciences*, 125(2), 117–124. <https://doi.org/10.1254/jphs.14R03CP>
5. Cunha, O. T., Rego, A. C., & Oliveira, C. R. (2008). Cellular and molecular mechanisms involved in the neurotoxicity of opioid and psychostimulant drugs. *Brain Research Reviews*, 58(1), 192–208. <https://doi.org/10.1016/j.brainresrev.2008.03.002>
6. Hartmann-Boyce, J., Chepkin, S. C., Ye, W., Bullen, C., & Lancaster, T. (2018, May). Nicotine replacement therapy versus control for smoking cessation. *Cochrane Database of Systematic Reviews*, 5, CD000146. <https://doi.org/10.1002/14651858.CD000146.pub5>
7. England, L. J., Bunnell, R. E., Pechacek, T. F., Tong, V. T., & McAfee, T. A. (2015, August). Nicotine and the developing human: A neglected element in the electronic cigarette debate. *American Journal of Preventive Medicine*, 49(2), 286–293. <https://doi.org/10.1016/j.amepre.2015.01.015>
8. Centers for Disease Control and Prevention. (n.d.). *Health effects of cigarette smoking*. Retrieved from https://www.cdc.gov/tobacco/data_statistics/fact_sheets/health_effects/effects_cig_smoking/
9. Gn, C. R., & Rogman, A. (1996). Tobacco methods and instrumentation: A half century forum. *Recent Advances in Tobacco Science*, 22, 131–304.
10. Janson, C., Chinn, S., Jarvis, D., Zock, J. P., Torén, K., & Burney, P. (2001). Effect of passive smoking on respiratory symptoms, lung function, and serum IgE: A European study. *The Lancet*, 358(9299), 2103–2109.
11. Hussein, M. U., & Ali, A. H. (2019). The effect of mean period and ejection time in patients with hypertension. *Research Journal of Biotechnology*, 14(Special Issue I), 297–302.
12. Sheps, D. S., Herbst, M. C., Hinderliter, A. L., Adams, K. F., Ekelund, L. G., & Neil, J. J. (1990). Arrhythmias produced by elevated carboxyhemoglobin in coronary disease. *Annals of Internal Medicine*, 113(5), 343–351.
13. Ariens, E., Simonis, A. M., & Offermeir, J. (1976). *Introduction to general toxicology*. Academic Press.
14. Sodhi, P. K., Goyal, J. L., & Mehta, D. K. (2001). Methanol-induced optic neuropathy: Treatment with high-dose IV steroids. *International Journal of Clinical Practice*, 55, 599–602.
15. Haldane, J. (1985). The action of carbonic oxide on man. *The Journal of Physiology*, 18(5–6), 430–462.
16. Narkiewicz, K., Kjeldsen, S. E., & Hedner, T. (2005). Is smoking a causative factor of hypertension? *PubMed*, 14(2), 69–71.

17. Vainio, H. (1987). Is passive smoking increasing cancer risk? *Scandinavian Journal of Work, Environment & Health*, 13(3), 193–196.
18. Blumenthal, I. (2001). Carbon monoxide poisoning. *Journal of the Royal Society of Medicine*, 94, 270–272.
19. Lakshmi, A. S., Lakshmanan, A., Kumar, G. P., & Saravanan, A. (2014). Effect of cigarette smoking intensity on hematological and lipid parameters. *Journal of Clinical and Diagnostic Research*, 8(7), 11–13.
20. Yu, G., Phillips, S., Gail, M. H., et al. (2017). The effect of cigarette smoking on oral and nasal microbiota. *Microbiome*, 5, 3.
21. Asif, M., Karim, S., Umar, Z., et al. (2013). Effect of cigarette smoking on hematological parameters. *Turkish Journal of Biochemistry*, 38(1), 75–80.
22. Helman, N., & Rubenstein, L. S. (1975). Age, sex, and smoking effects on erythrocytes and leukocytes. *American Journal of Clinical Pathology*, 63(1), 35–44.
23. Tsiara, S., Elisaf, M., & Mikhailidis, D. P. (2003). Influence of smoking on predictors of vascular disease. *Angiology*, 54, 507–530.
24. Geffken, D., Cushman, M., Burke, G. L., Polak, J. F., Sakkinen, P. A., & Tracy, R. P. (2001). Physical activity and inflammation in the elderly. *American Journal of Epidemiology*, 153, 242–250.
25. Lewis, S. M., Bain, B. J., & Bates, I. (2006). *Practical hematology* (10th ed., pp. 1–40). Elsevier.
26. National Health and Medical Research Council. (2018). *Nutrient reference values for Australia and New Zealand*.
27. Brigden, M. L. (1999). Clinical utility of the erythrocyte sedimentation rate. *American Family Physician*, 60(5), 1443–1450.
28. Böttiger, L. E., & Svedberg, C. A. (1967). Normal erythrocyte sedimentation rate and age. *British Medical Journal*, 2(5544), 85–87.
29. Varol, E., Icli, A., Kocyigit, S., Erdogan, D., Ozaydin, M., & Dogan, A. (2012). Effect of smoking cessation on mean platelet volume. *Clinical and Applied Thrombosis/Hemostasis*. Advance online publication.
30. Caponnetto, P., Russo, C., Di Maria, A., et al. (2011). Circulating endothelial coagulative markers after smoking cessation: A 12-month study. *European Journal of Clinical Investigation*, 41(6), 616–626.
31. Butkiewicz, A. M., Kemonia-Chetnik, I., Dymicka-Pickarska, V., Matowicka-Karna, J., Kemonia, H., & Radziwon, P. (2006). Smoking and thrombocytopoiesis in men and women. *Advances in Medical Sciences*, 51, 123–126.
32. Arslan, E., Yakar, T., & Yavasoglu, I. (2008). Effect of smoking on mean platelet volume and lipid profile. *Anadolu Kardiyoloji Dergisi*, 8(6), 422–425.
33. Ghosh, A., Chowdhury, S. D., & Ghosh, T. (2012). Undernutrition in Nepalese children: Biochemical and hematological study. *Acta Paediatrica*, 101(6), 671–676.
34. Can, C., Malkoç, H., Coşkun, H., Küçüköğlü, B., & Özmen, E. (2007). Evaluation of reticulocyte parameters in anemia types. *International Journal of Laboratory Hematology*, 29(5), 327–334.
35. Freedman, D. S., Flanders, W. D., Barboriak, J. J., Malarcher, A. M., Gates, L. (1996). Smoking and leukocyte subpopulations in men. *Annals of Epidemiology*, 6(4), 299–306.
36. Kawada, T. (2004). Smoking-induced leukocytosis after cessation. *Archives of Medical Research*, 35, 246–250.

37. Calapai, G., Caputi, A. P., Mannucci, C., Russo, A. G., Gregg, E., Puntoni, R., et al. (2009). Cardiovascular biomarkers in long-term smokers. *Basic & Clinical Pharmacology & Toxicology*, 104, 322–328.
38. Geffken, D., Cushman, M., Burke, G. L., et al. (2001). Physical activity and markers of inflammation. *American Journal of Epidemiology*, 153, 242–250.
39. Kume, A., Kume, T., Masuda, K., Shibuya, F., & Yamazaki, H. (2009). Dose-dependent effects of cigarette smoke on biomarkers. *Journal of Health Sciences*, 55(2), 259–264.
40. Islam, M. M., Amin, M. R., Begum, S., Akther, D., & Rahman, A. (2007). Total WBC count in adult male smokers. *Journal of Bangladesh Society of Physiology*, 2, 49–53.
41. Bain, B. J., Rothwell, M., Feher, M. D., Robinson, R., Brown, J., et al. (1992). Hematological changes on smoking cessation. *Journal of the Royal Society of Medicine*, 85, 80–82.
42. Ho, C. H. (2004). WBC and platelet counts influence whole blood viscosity. *Journal of the Chinese Medical Association*, 67(8), 394–397.
43. Levenson, A. C., Simon, F. A., Cambien, F., & Beretti, C. (1987). Smoking, hypertension, and blood hyperviscosity. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 7, 572–577.
44. Varol, E., Icli, A., Kocyigit, S., Erdogan, D., Ozaydin, M., & Dogan, A. (2012). Effect of smoking cessation on MPV. *Clinical and Applied Thrombosis/Hemostasis*. Advance online publication.
45. Caponnetto, P., Russo, C., Di Maria, A., et al. (2011). Circulating coagulative activation markers after smoking cessation. *European Journal of Clinical Investigation*, 41(6), 616–626.
46. Butkiewicz, A. M., Kemonia-Chetnik, I., Dymicka-Piekarska, V., Matowicka-Karna, J., Kemonia, H., & Radziwon, P. (2006). Smoking and platelet activation. *Advances in Medical Sciences*, 51, 123–126.
47. Arslan, E., Yakar, T., & Yavasoglu, I. (2008). Smoking and lipid profile in young men. *Anadolu Kardiyoloji Dergisi*, 8(6), 422–425.
48. Calapai, G., Caputi, A. P., Mannucci, C., Russo, A. G., Gregg, E., Puntoni, R., et al. (2009). Cardiovascular biomarkers in smokers after a decade. *Basic & Clinical Pharmacology & Toxicology*, 104, 322–328.