Wasit Journal for Pure Science

Journal Homepage: https://wjps.uowasit.edu.iq/index.php/wjps/index
e-ISSN: 2790-5241 p-ISSN: 2790-5233

Assessment of Testosterone Levels in Tobacco Smokers' Dependence over the Long Term with Some Haemato-Biochemical Parameters

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DOI: https://doi.org/10.31185/wjps.772
Received 20 March 2025; Accepted 01 May 2025; Available online 30 September 2025

ABSTRACT: Smoking is considered a pervasive global concern, negatively affecting health and leading to several diseases that cause millions of fatalities annually. The purpose of this research was to investigate the effects of heavy and chronic tobacco use on testosterone levels, hematological parameters, renal function, cholesterol, triglycerides, and liver enzymes in male smokers. Participants (n = 60) were recruited from a population cohort of men in Al-Ramadi city, aged 20–63 years. The smoker group (n = 30) consisted of individuals who had been addicted to smoking for 10 years or more, consuming 15–40 cigarettes per day. All subjects completed a detailed survey regarding their health status, medication intake, and any other symptoms or diseases. The following parameters were measured: sex hormones, RBC count, Hb, HCT, MCV, MCH, MCHC, WBC count, platelets, renal function markers, lipid profiles, and liver enzymes (ALP, AST, and ALT). Our findings demonstrated a statistically significant decrease in total blood testosterone levels ($p < 0.001$) among chronic smokers compared to non-smokers (1.653 ± 0.097 vs. 5.980 ± 0.195, respectively). Cigarette smoking also adversely affected hematological parameters: RBC count, hemoglobin, hematocrit, MCV, MCHC, and total leukocyte count showed a statistically significant increase ($p < 0.05$). No significant variations were observed in MCH and platelet count ($p > 0.05$). Furthermore, the study revealed a highly significant increase in urea and creatinine levels ($p < 0.001$), indicating impaired renal function. Lipid profiles and liver enzymes (ALP, AST, and ALT) were also significantly elevated ($p < 0.001$) in smokers compared to non-smokers. In conclusion, the present study highlights the detrimental consequences of prolonged smoking, including reduced serum testosterone levels, alterations in hematological parameters, increased risk of kidney dysfunction (as evidenced by elevated urea and creatinine), dyslipidemia (changes in total cholesterol and triglycerides), and abnormal liver enzyme levels. These findings suggest that chronic smoking is a major risk factor for multiple systemic disorders..

Keywords: Tobacco smoking, Testosterone, Blood parameters, Renal function, Cholesterol, liver function

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1. INTRODUCTION

The tobacco epidemic represents one of the most substantial threats to public health in history, accounting for approximately 8 million deaths worldwide each year. Of these, more than 7 million fatalities are directly attributable to tobacco consumption, while about 1.5 million result from non-smokers' exposure to second-hand smoke [1].

Tobacco smoke contains around 8,000 chemicals, many of which have harmful and carcinogenic effects. Free radicals, nicotine, and carbon monoxide are considered the primary contributors to the adverse health impacts of smoking [2]. Smoking is strongly associated with the development of numerous respiratory diseases; for example, more than 80% of Chronic Obstructive Pulmonary Disease (COPD) cases in the United States are linked to smoking [3].

Extensive research has shown that smoking negatively affects health and is a major risk factor for the onset of various pathological conditions and diseases, including cancers, cardiovascular disorders, oral diseases, fetal mortality, impaired vision, reduced bone mass and fractures, diabetes, and certain autoimmune disorders [4].

Some researchers argue that the global decline in male reproductive capacity may be partly attributable to lifestyle factors, particularly harmful behaviors such as smoking, excessive alcohol consumption, sedentary living, and

overeating. These behaviors not only undermine general health but also impair sperm quality, potentially leading to subfertility or infertility in men [5].
Testosterone is regarded as the primary hormone involved in reproductive function as well as metabolic processes. Exposure to cigarette smoke can lead to hormonal imbalances, which in turn may cause DNA damage and exert toxic effects on testicular cells [6].
Moreover, smoking exacerbates endothelial cell dysfunction and promotes thrombus formation in the coronary arteries, with nicotine contributing to reduced vascular function. Elevated levels of carboxyhemoglobin can induce hypoxia and are also responsible for subendothelial edema, as they alter vascular permeability and facilitate lipid deposition [7]. Tobacco exerts both acute and chronic effects on hematological markers, as well as on liver and kidney enzyme activity in the human body [8–10].

The present study aims to evaluate the impact of cigarette smoking on testosterone levels, selected hematological parameters, and biochemical indicators in individuals with long-term smoking addiction, compared to non-smokers.

2. MATERIALS AND METHODS

2.1. Research methodology and participants

Sixty male volunteers were recruited, excluding individuals with chronic conditions such as hypertension, type 2 diabetes, cardiovascular disease, a history of infertility, or those undergoing hormonal therapy. Participants were divided into two groups: 30 smokers (with a smoking history of at least 10 years and a daily consumption of 15–40 cigarettes) and 30 non-smokers as the control group. The ages of participants in both groups ranged from 20 to 63 years. All participants were residents of Al-Ramadi city during the study period, which extended from July to November 2024.

2.2. Managements

Venous blood samples (approximately 10 ml) were collected from each participant in appropriate containers in the morning after an overnight fast. Of this, 7 ml were used for the evaluation of serum testosterone, estradiol, and biochemical parameters. After allowing the samples to clot, they were centrifuged at 3000 rpm for 15 minutes. Hormone levels were measured using electrochemiluminescence immunoassay technology (Roche, Cobas, Germany).

A 3 ml blood sample was collected in vacuum tubes containing ethylenediaminetetraacetic acid (EDTA) and gently mixed. The samples were then analyzed using a hematology analyzer (Dymind DH76, China) to perform a complete blood count (CBC). The hematological parameters assessed included red blood cell count (RBC), hemoglobin concentration (HGB), hematocrit (HCT or PCV), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), white blood cell count (WBC), and platelet count (PLT).

Furthermore, participants underwent the following biochemical assessments: serum levels of renal function markers (urea and creatinine), lipid profile parameters (total cholesterol and triglycerides), total bilirubin, and liver function enzymes (alkaline phosphatase [ALP], alanine aminotransferase [ALT], and aspartate aminotransferase [AST]). These measurements were performed using the Diasys Response 920 analyzer (Diasys Co., Germany) with high-performance diagnostic reagent kits. The assays were based on biochemical techniques employing spectrophotometry and colorimetry to determine the concentrations of chemical compounds in biological samples.

2.3. Statistical examination

An independent samples t-test was used to compare the smoking group with the control group, with statistical significance set at $p < 0.05$. Data were analyzed using SPSS software, and Microsoft Excel was employed for data presentation [11].

3. RESULTS AND DISCUSSION

Tobacco Products: Smoking is recognized as a major contributor to preventable illness and death worldwide [12]. The mean age of smokers and non-smokers was 42.5 ± 1.58 and 41.27 ± 1.92 years, respectively. The average duration of smoking among smokers was 17.03 ± 1.34 years.

Testosterone levels were significantly lower in smokers (1.653 ± 0.097 ng/ml) compared to the control group (5.980 ± 0.195 ng/ml), showing a highly significant difference ($p < 0.001$). In contrast, no significant differences were observed in estradiol levels between smokers (34.987 ± 2.222 pg/ml) and the control group (30.980 ± 1.495 pg/ml; $p = 0.141$) (Table 1).

Both the free form of testosterone and the fraction bound to sex hormone-binding globulins (SHBG) are present in circulation. The free form accounts for approximately 65–80% of total testosterone. Consequently, total testosterone concentrations may be influenced by fluctuations in SHBG and other plasma proteins [13].

Our findings showed that serum testosterone levels in smokers were significantly lower than in non-smokers, which is consistent with the results reported by El Salam et al. [14].

It has been hypothesized that smoking lowers testosterone levels by causing Leydig cell dysfunction through multiple mechanisms, including chronic hypoxia and oxidative stress (ROS) induced by smoking. Several explanations have been proposed for this phenomenon. Nicotine exerts neuroendocrine effects on the hypothalamo-pituitary-gonadal axis and also directly affects Leydig cells by downregulating key steroidogenic enzymes (NR5A1, CYP11A1, and 3β-HSD1) or by inducing cell apoptosis, which involves upregulating pro-apoptotic proteins (Bax and caspase-3) and downregulating the anti-apoptotic protein Bcl-2 [15].

Table (1) Comparison between the levels of Testosterone and Estradiol expressed as mean ± the standard error of smokers group versus the non-smokers group

Parameters Parameters	T	N	Mean	Std. Error	t- test	p- value
Age/ years	Smokers	30	42.5	±1.58	0.496	0.622
	Non smokers	30	41.27	± 1.92		
Testosterone ng/ml	Smokers	30	1.653	±0.097	19.92	0.000
	Non smokers	30	5.980	±0.195		
Estradiol (E2)pg/ml	Smokers	30	34.987	±2.222	1.49	0.141
	Non smokers	30	30.980	±1.495		

Concerning the haematological parameters for the participants .The mean value of RBC, Hb, HCT, MCV, MCH,MCHC, WBC and Platelets were higher in smokers who were dependence on chronic tobacco smoking (5.540 × 106/µL ± 0.066 , 15.853g/dl ±0.103 , 45.783% ±0.387,88.373fL ±0.521, 29.863pg ±0.239, 34.340 g/dL ±0.157, 8.828 103/µL ±0.655, 245.267 103/µL ±20.995 respectively) compared to non-smoker (5.087× 106/µL ±0.051, 14.773 g/dl ±0.142, 44.360%±0.587, 86.000 fL ±0.537, 29.373 pg±0.194, 33.660 g/dL±0.155, 6.955 103/µL ± 0.223 , 226.333103/µL ±7.080 respectively) , (table 2). The findings revealed that individuals in the smoking cohort demonstrated significant disparities relative to non-smokers in the following metrics: RBC count ($p < 0.001$), Hb level ($p < 0.001$), HCT % ($p = 0.048$), MCV ($p = 0.002$), MCHC ($p = 0.003$), and WBC ($p = 0.010$). In contrast, there is no notable difference in MCH ($p=0.117$) and platelets ($p=0.399$) among smokers when compared with the group of control.

To provide context for these results, our analysis revealed a significant elevation in RBC count among individuals with long-term smoking habits compared to non-smokers. These findings are consistent with those of Khalid et al. [16], who conducted a similar study involving 100 male participants. In their study, smokers who consumed 7–10 cigarettes daily showed a significantly higher RBC count compared to non-smokers (5.1304×10^6/µL vs. 4.3852×10^6/µL). In addition to the increase in RBC count, smokers also exhibited higher hemoglobin (HGB) and hematocrit (HCT) levels compared to non-smokers. These results align with the conclusions of numerous other studies [17, 18].

Several researchers have hypothesized that the elevated hemoglobin levels observed in smokers may serve as a compensatory mechanism. This increase in hemoglobin is thought to result from exposure to carbon monoxide. When carbon monoxide binds to hemoglobin (Hb), it forms carboxyhemoglobin, an inactive form of hemoglobin that cannot transport oxygen. Moreover, carboxyhemoglobin shifts the hemoglobin-oxygen dissociation curve to the left, further reducing hemoglobin's ability to deliver oxygen to tissues.

To compensate for this diminished oxygen-carrying capacity, smokers maintain higher hemoglobin levels compared to non-smokers. The increased erythrocyte and hematocrit levels in male smokers can be attributed to tissue hypoxia induced by elevated carboxyhemoglobin, which stimulates erythropoietin release and enhances erythropoiesis. Additionally, carbon monoxide in tobacco smoke increases capillary permeability, leading to a reduction in plasma

volume. This effect resembles polycythemia, characterized by an increased proportion of red blood cells in blood volume, as reflected in elevated hematocrit levels [19].

MCV, MCH, and MCHC are the three principal indices of red blood cells employed to ascertain the average size of red blood cells and their hemoglobin content. The findings of our research indicated that smokers exhibited significantly elevated mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration (MCHC), whereas the disparities in mean corpuscular hemoglobin (MCH) values among smokers and those who did not smoke were not o statistical significant. Ahmed et al. [20] corroborated these findings, revealing that smokers exhibited significantly elevated MCV and MCHC values in comparison to non-smokers. In the investigation of red cell indices, including MCH, MCV, and MCHC conducted by Srivastava et al. [21], The WBC count was found to be significantly elevated in smokers. Several studies have reported higher total leukocyte counts in smokers [22, 23]. Multiple mechanisms may contribute to smoking-induced leukocytosis. Nicotine can stimulate the adrenal glands to release catecholamines and steroid hormones, which in turn can increase WBC levels. The association between elevated endogenous hormones—such as adrenaline and cortisol—and increased leukocyte counts is well established [24].

Additionally, the irritant effects of smoke on the respiratory tract induce inflammation and cytokine production, both of which can influence leukocyte numbers [25]. These effects are believed to be dose-dependent. Notably, smoking cessation has been associated with a reduction in leukocyte counts [26].

In contrast, platelet count (PLT) was the only hematological parameter that showed no significant increase in smokers compared to non-smokers. These findings are consistent with those of Bilto [27], who studied the effects of smoking on blood rheology and biochemistry in 606 subjects, including 302 smokers (smoking history: 5–20+ years; 10–40+ cigarettes/day) and 304 non-smoking controls. Similarly, Hasan et al. [28] reported no significant difference in PLT counts between smokers (242.74 × 10^3/µL) and non-smokers (245.74 × 10^3/µL).

However, some studies have recorded a significant increase in platelet count, suggesting that both short- and long-term smoking can damage the endothelium. Moreover, platelet production is regulated by hormonal metabolism, which may be disrupted by smoking, resulting in elevated platelet numbers [29].

Table (2) Comparison between the levels of haematological parameters as mean ± the standard error of smokers group versus the non-smokers group

Parameters	T	N	Mean	Std. Error	t- test	p- value
RBC 10^6 /µL	**Smokers**	30	5.540	± 0.066	5.46	0.000
	Non smokers	30	5.087	± 0.051		
Hb g/dl	**Smokers**	30	15.853	± 0.103	6.15	0.000
	Non smokers	30	14.773	± 0.142		
HCT %	**Smokers**	30	45.783	± 0.387	2.025	0.048
	Non smokers	30	44.360	± 0.587		
MCV Fl	**Smokers**	30	88.373	± 0.521	3.170	0.002
	Non smokers	30	86.000	± 0.537		
MCH pg	**Smokers**	30	29.863	± 0.239	1.592	0.117
	Non smokers	30	29.373	± 0.194		
MCHC g/dl	**Smokers**	30	34.340	± 0.157	3.09	0.003
	Non smokers	30	33.660	± 0.155		
WBC 10^3/ µL	**Smokers**	30	8.828	± 0.655	2.71	0.010
	Non smokers	30	6.955	± 0.223		
Platelets 10^3/ µL	**Smokers**	30	245.267	±20.995	0.855	0.399
	Non smokers	30	226.333	± 7.080		

Regarding the Biochemical parameters of the participants. The mean value of Urea, Creatinin, Cholesterol, Triglyceride, ALP, ALT and AST were higher in smokers who were consuming chronic tobacco smoking (43.500 mg/dl ± 1.612, 0.914 mg / dl ± 0.049 , 203.000 mg/dl ± 6.725 , 189.600 mg/dl ± 21.499 , 80.133 U/L ± 3.430 , 37.600 U/L ± 1.499 , 29.467 U/L ± 0.872 respectively) compared to non-smoker (27.533 mg/dl ± 2.306 , 0.414 mg/dl ± 0.039 , 124.333 mg/dl ±4.015 , 90.500 mg/dl ± 3.923 , 57.600 U/L ± 1.307 , 23.500 U/L ± 1.415 , 22.400 U/L ± 1.249 respectively), (table 3).

Table (3) Comparison between the levels of Biochemical parameters as mean ± the standard error of smokers group versus the non-smokers group

Parameters	T	N	Mean	Std. Error	t- test	p- value
Urea mg/dl	**Smokers**	30	43.500	± 1.612	5.675	0.000
	Non smokers	30	27.533	± 2.306		
Creatinin mg/dl	**Smokers**	30	0.914	± 0.049	7.99	0.000
	Non smokers	30	0.414	± 0.039		
Cholesterol mg / dl	**Smokers**	30	203.000	± 6.725	10.04	0.000
	Non smokers	30	124.333	± 4.015		
Triglyceride mg/dl	**Smokers**	30	189.600	± 21.499	4.54	0.000
	Non smokers	30	90.500	± 3.923		
ALP U/L	**Smokers**	30	80.133	± 3.430	6.14	0.000
	Non smokers	30	57.600	± 1.307		
ALT U/L	**Smokers**	30	37.600	± 1.499	6.84	0.000
	Non smokers	30	23.500	± 1.415		
AST U/L	**Smokers**	30	29.467	± 0.872	4.64	0.000
	Non smokers	30	22.400	± 1.249		

This study examined the effect of cigarette smoking on blood lipid profiles, particularly total cholesterol and triglyceride levels. The results demonstrated that smokers had significantly higher total cholesterol and triglyceride levels compared to non-smokers ($p < 0.001$). Lipids are essential structural components of cells and participate in numerous metabolic processes; therefore, imbalances in lipid fractions can compromise human health [32].

In a study by Nath et al. [33], non-smokers exhibited significantly lower mean total cholesterol levels compared to smokers (172.05 ± 29.76 vs. 205.05 ± 51.58, $p < 0.001$). In contrast, Gupta V et al. [34] reported no statistically significant differences in cholesterol levels between smokers and non-smokers. This discrepancy may be explained by differences in sample selection criteria, participants' dietary habits, the younger age of the study group, and the shorter duration of smoking in their study.

In this investigation, elevated levels of triglycerides were found to correlate with an increase in the levels of total cholesterol. These results are consistent with what Sousa et al [35]. Chandran et al [36], who did studies that were quite similar to this one, demonstrated that smoking causes a change in the lipid profile, which in turn leads to dyslipidaemia in smokers. Furthermore, the effects of this shift grow more pronounced with the number of cigarettes that are smoked.

In this investigation, elevated triglyceride levels were found to correlate with increased total cholesterol levels. These results are consistent with the findings of Sousa et al. [35]. Chandran et al. [36], in similar studies, demonstrated that smoking alters the lipid profile, leading to dyslipidemia in smokers. Moreover, the effects of this alteration become more pronounced with the number of cigarettes smoked.

Compared to non-smokers, individuals with a smoking history of more than 10 years had significantly higher levels of the liver enzymes ALT, AST, and ALP ($p < 0.001$). This suggests that chronic smoking may influence liver enzyme activity. These findings are consistent with those reported by Baled and Mohamed [37], who also observed similar results. However, according to Suriyaprom et al. [38] and several earlier studies, smoking may not directly damage hepatocytes, implying that the observed changes in enzyme levels could be attributed to indirect mechanisms.

4. CONCLUSION

This study concluded that middle-aged men with heavy and long-term smoking habits tend to have markedly lower testosterone levels. Moreover, several hematological parameters—including white blood cell count, mean corpuscular hemoglobin concentration, mean corpuscular volume, hematocrit, hemoglobin level, and red blood cell count—differ significantly between smokers and non-smokers. By contrast, no substantial differences were observed in mean

corpuscular hemoglobin (MCH) or platelet counts. In addition, kidney function appears to be negatively affected in smokers, as indicated by elevated serum creatinine and urea levels. Smoking was also clearly associated with increased levels of total cholesterol and triglycerides, as well as significantly higher liver function markers (ALP, AST, and ALT) in chronic tobacco users. Therefore, prompt smoking cessation may help reverse these adverse biochemical and physiological alterations, ultimately reducing the risk of developing severe health complications in the future.

REFERENCES

[1] Institute for Health Metrics and Evaluation. (2019). Global Burden of Disease database. Retrieved July 17, 2023, from http://www.healthdata.org

[2] S. Hong, P. Teesdale-Spittle, R. Page, B. Ellenbroek. and P. Truman "Active Compounds Present in Tobacco Smoke: Potential Interactions Between Smoking and Mental Health". Neurosci., 16. 2022. https://doi.org/10.3389/fnins.2022.885489

[3] NL. Benowitz, and PG. Brunetta. "Smoking hazards and cessation. In: Broaddus VC, Mason RJ, Ernst JD, King TE Jr, Lazarus SC, Murray JF, et al., editors. Murray & Nadel's textbook of respiratory medicine. 6th ed. Philadelphia: Elsevier Saunders; 2016. p. 807-13

[4] World Health Organization." The Tobacco Body ", Geneva, Switzerland: WHO ,2019. [Online].Available: https://iris.who.int/bitstream/handle/10665/324846/WHO-NMH-PND-19.1-eng.pdf [Accessed : Apr.11,2025]

[5] Lv. Osadchuk, and AV. Osadchuk. "Individual lifestyle and male fertility". Hum Physiol., 49:196–207,2023. doi: 10.1134/S0362119722600527

[6] Granata S, Morosini C, Valerii M C, Fagiolino I, Sangiorgi S, Ghini S, Spisni E, Vivarelli F, Fairclough L C, Paolini M and Canistro D.(2024). Heat-not-burn technology affects plasma testosterone levels and markers of inflammation, oxidative stress in the testes of rats.

[7] Frontiers in Toxicology .6 , https://doi.org/10.3389/ftox.2024.1515850

[8] D. Singh, "Effect of cigarette smoking on serum lipid profile in male population of Udaipur (Rajasthan)". Int J Clin Biochem., Res 3: 368-70, 2016.

[9] M. Al- Mufarah, N. Alharbi, A. Aqeeli, R. Bokhedher, and Z. Al Mohsen, "Consequence of smoking on hematological parameters in apparently healthy medical students". International Journal of Medical Microbiology and Tropical Diseases8(4):288–293, 2022. https://doi.org/10.18231/j.ijmmtd.2022.056.

[10] Djabali Nacira, Berrezig Wiem, Ghai Djamila, "Effects of Active Smoking on Renal Function". AENSI J., 9(24): 191-195, 2015.

[11] P. Pannuru, D.Vaddi, and V. Nallanchakravarthula ," Influence of Chronic Cigarette Smoking on Serum Biochemical Profile in Male Human Volunteers". Journal of Health Science., 55(2) 265–70, 2009.

[12] P. Haaland, "Experimental design in biotechnology". (2020). CRC press. https://doi.org/10.1201/9781003065968

[13] Tobacco [Internet]. World Health Organization. World Health Organiza tion. Available from: https://www.who.int/news-room/fact-sheets/detail/ tobacco. Accessed 21 Feb 2023.

[14] K. Chin, I. Soelaiman, I. Mohamed, W. Ngah, "Serum testosterone, sex hormone-binding globulin and total calcium levels predict the calcaneal speed of sound in men". Clinics (Sao Paulo)., Aug;67(8):911-6, 2012.

[15] M. El Salam, S. Zaki, M. Mousa, and A. Motawi, "Effect of cigarette smoking on serum testosterone level among male smokers: a cross-sectional study". The Egyptian Journal of Chest Diseases and Tuberculosis., 70 (1). p 124-127, 2021. DOI: 10.4103/ejcdt.ejcdt_61_20

[16] X. Guo, H. Wang, X. Wu, X. Chen, Y. Chen, J. Guo, et al. "Nicotine affects rat Leydig cell function in vivo and vitro via down-regulating some key steroidogenic enzyme expressions". Food Chem Toxicol., 110:13–24. 24, 2017.

[17] T. Khalid, S. Raza, F. Kalsoom, I. Nawaz, M. Hayat, and R. Yasmin, "Assessment of hematological parameters in young smokers: a case study from faisalabad, Pakistan". Journal of Population Therapeutics & Clinical Pharmacology., 31(9). (253-261), 2024. DOI: 10.53555/0hrmca61

[18] S. Nargish, M. Kabir, K. Akhter, K. Nahid, M. Hossain, P. Karmakar, M. Ahmed, and M. Islam , "Effect of smoking on the red blood cell count, haemoglobin concentration, haeematocrit and red cell indices in adult male smokers". Eastern Mediterranean Health Journal., 7(1):1-5, 2022.

[19] D. Açık, E. Suyani, B. Aygün, M. Bankir , “The effect of smoking on hematological parameters”. J Appl Psychol., 32(2):150–8, 2020.

[20] R. Aitchison, and N. Russell, “Smoking- a major cause of polycythaemia”. J R Soc Med., 81(2):89–91, 1988.

[21] I. Ahmed, M. Mohammed, H. Hassan, and I. Ali I, “Relationship between tobacco smoking and hematological indices among Sudanese smokers”. Journal of Health, Population and Nutrition ., 43:5, 2024 .https://doi.org/10.1186/s41043-023-00493-0

[22] A. Srivastava, K. Tiwari, S. Irfan, N. Zaidi, S. Ahmad, S. Mehdi, “Cigarette smoking and its effect on coagulation profile, hematological parameters, and oxygen saturation in healthy blood donor”. Asian journal of medical sciences.,15(8), 2024. DOI: 10.3126/ajms.v15i8.65345

[23] M. Malenica, B. Prnjavorac, T. Bego, T. Dujic, S. Semiz, S. Skrbo, A. Gusic, A. Hadzic, A. Causevic , “Effect of Cigarette Smoking on Haematological Parameters in Healthy Population”. ORIGINAL PAPER., 71(2): 132-136, 2017. doi: 10.5455/medarh.2017.71.132-136

[24] R. Çiftçiler, A. Guven, I. Haznedaroğlu, and S. Aksu, “Effects of smoking on hema tological parameters and ferritin levels”. Medical Bulletin of Haseki. , 57(4), 2019.

[25] V. Deutsch, L. Lerner-Geva, A. Reches, V. Boyko, R. Limor, D. Grisaru, et al. “Sustained leukocyte count during rising cortisol level”. Acta Haematologica. ,118(2):73–6, 2007.

[26] D. Granger, and E. Senchenkova , “Inflammation and the Microcirculation”. San Rafael (CA); 2010.

[27] G. Abel, J. Hays, P. Decker, G. Croghan, D. Kuter, and N. Rigotti, “Effects of biochemically confirmed smoking cessation on white blood cell count”. Mayo Clin Proc., 80(8):1022–8, 2005.

[28] YY. Bilto, “Effects of cigarette smoking on blood rheology and biochemistry”. Int J Sci Res., 3(4):107-112, 2015.

[29] HM. Hasan, T. Arhouma, M. Khanfar, and M. Azzam, “Study the Relationship between the Nicotine and Lipid Profile with Some Hematology Parameters in Serum of Smoker and Non-Smoker Blood Samples”. Journal of Biosciences and Medicines. , 10 (6) 20–36, 2022. DOI: 10.4236/jbm.2022.106003

[30] F. Mobarrez, L. Antoniewicz, J. Bosson, J. Kuhl, D. Pisetsky , and M. Lundback ,”The effects of smoking on levels of endothelial progenitor cells and microparticles in the blood of healthy volunteers”. PLoS One; 9(2): e90314, 2014.

[31] H. Eid , E. Moazen, M. Elhussini, H. Shoman, A. Hassan , A. Elsheikh, A. Rezk, A. Moursi, M. Atef, A. Kabil , “The Influence of Smoking on Renal Functions Among Apparently Healthy Smokers”. Journal of Multidisciplinary Healthcare. , 15 2969–2978, 2022. https://doi.org/10.2147/JMDH.S392848

[32] E. Ritz, U. Benck, E. Franek, C.Keller, M. Seyfarth, and J. Clorius, “Effects of smoking on renal hemodynamics in healthy volunteers and in patients with glomerular disease”. J Am Soc Nephrol., 9(10).1798-804, 1998.

[33] GT. Russo, A. Giandalia, EL Romeo, et al. “Gender differences in lipoprotein metabolism”. Ital J Gend Specif Med., 1(2): 58-65, 2015.

[34] MC. Nath, AS. Rahman, MC. Nath , A. Dutta, Z. Khan, E. Ghosh , S. Akhter, S. Islam, S. Sultana, R. Begum, M. Rahman , “The Effect of Cigarette Smoking on Fasting Lipid Profile: A Single Center Study”. Fortune J Health Sci., 5 (2): 363-373, 2022. DOI: 10.26502/fjhs.067

[35] V. Gupta, S. Tiwari, CG. Agarwal, P. Shukla, H. Chandra, and P. Sharma, “Effect of short term cigarette smoking on insulin resistance and lipid profile in asymptomatic adults”. Indian journal of physiology and pharmacology., 50: 285, 2006.

[36] IR. Sousa, M. Miranda, H. Gomes, A. Figueiredo, J. Silva, J. Campos, “Relationship Between Smoking and Lipid Profile in Four Primary Health Care Units: A Research Study”. Cureus., 16(9): e69172, 2024. DOI 10.7759/cureus.69172

[37] E. Baled, and I. Mohamed, “Cigarette smoking effects on biochemical and hematological parameter in Brack city in south Libya”. Sebha university journal of journal of medical sciences., 18(1), 2023. DOI: 10.51984/JOMS.V18I1.2637

[38] K. Suriyaprom, T. Hamrnroongroj, P. Namjuntra, Y. Chantaranipapong, R. Tungtrongchitr, “Effects of tobacco smoking on alph-2-macroglobulin and some biochemical parameters in Thaimales”. Southeast Asian J. Trop. Med. Public Health., 38(5):918-926, 2007.