



Effect of Smoking on Blood Parameters and Thyroid Hormones in Men with Polycythemia

Marwah A. Majeed

Zainab A. Ridha Al-Ali

Department of Biology/ College of Science/ University of Misan/ Maysan/ Iraq

p-ISSN: 1608-9391

e-ISSN: 2664-2786

Article information

Received: 9/ 6/ 2023

Revised: 25/10/ 2023

Accepted: 5/ 11/ 2023

DOI:

10.33899/rjs.2024.183421

corresponding author:

Zainab A. Ridha Al-Ali

zainab-alali@uomisan.edu.iq

ABSTRACT

Smoking is the primary cause of fatalities, disabling conditions, and avoidable diseases worldwide. Smokers are subject exposed to a variety of dangerous compounds, which stimulate the bone marrow to produce more RBCs. So that, this study aims to know the effects of smoking on men with polycythemia by studying the blood parameters and thyroid hormones. The study is conducted on 100 men (75 with polycythemia and 25 men control), ages (20-59) years, with samples collected from the blood bank at Maysan governorate, from the period November 2022 to March 2023. The results showed an increase significant ($P \leq 0.05$) in the values of red blood cells, hemoglobin and hematocrit, while, the white blood cells count showed no significant ($P \leq 0.05$) differences for both groups of polycythemias (smokers and non-smokers) as compared to the control group. Also, mean corpuscular volume, mean corpuscular hemoglobin and mean corpuscular hemoglobin concentration showed an increase significant ($P \leq 0.05$) in the smokers group. No significant ($P \leq 0.05$) variation was recorded in the values of thyroid stimulating hormone and thyroxine among groups, and triiodothyronine values in polycythemia (smoker and non-smoker) increased significantly ($P \leq 0.05$) in comparison with the control group. According to the above results we conclude the smoking effect in blood parameters and T_3 hormones in smoker polycythemia men.

Keywords: smoking, hemoglobin, polycythemia, thyroid, hormone.

INTRODUCTION

Smoking is the primary cause of fatalities, disabling conditions, and avoidable diseases worldwide. In the United States, it causes more than 480,000 fatalities annually, or about one in every five deaths (Jamal *et al.*, 2018).

Smoking cigarettes increases the chance of developing cardiovascular disorders such as peripheral vascular disease, atherosclerosis, myocardial infarction, stroke, and coronary artery disease (Malenica *et al.*, 2017). The exact process by which smoking causes these disorders in smokers is unknown, but it is believed that abnormalities in blood rheology, infections, inflammation, oxidative stress, changes in the fibrinolytic system, and antithrombotic systems are to blame (Malenica *et al.*, 2017). According to the (Ahmad and Al-Helaly, 2009), the smoking causes oxidative stress in smoker persons when study was conducted at suburbs Mosul City.

Smokers are exposed to a variety of dangerous compounds, such as nicotine, free radicals, carbon monoxide (CO), and other gaseous pollutants (Gitte, 2011).

Hemoglobin (Hb) and Hematocrit (Hct) levels rise as a result of the effects of tobacco and CO on tissue oxygen supply as well as, stimulate bone marrow to produce more RBCs (Roethig *et al.*, 2010), increased production of RBCs, and a very high RBC mass decreases blood flow, and increases the risk of intravascular coagulation, coronary vascular resistance, decreased coronary blood flow, and a propensity to thrombosis (Raval and Paul, 2010).

The thyroid gland is classical endocrine gland located in the anterior neck in front of the trachea. It is essential for the body's healthy development, differentiation, metabolism, and physiological performance. In clinical practice, thyroid dysfunction is one of the most frequent disorders, and it has become more widespread globally in recent years, thus its risk factors have drawn a lot of attention (Garmendia *et al.*, 2014; Taylor *et al.*, 2018).

Thyroid disorders can result in a variety of symptoms, such as the hypoplasia of erythroid cells in the bone marrow, the proliferation of immature erythroid progenitor cells (due to hypothyroidism), or the hyperplasia (related to hyperthyroidism) (Kawa *et al.*, 2010).

Previous research showed that thyroid malfunction can cause anemia, erythrocytosis, leukopenia, thrombocytopenia, and, in rare cases, pancytopenia in blood cells (Davis *et al.*, 1983).

Thyroid hormones stimulate the growth of erythroid colonies (BFU-E, CFU-E), increase erythrocyte 2, 3 Diphosphoglycerate (DPG) compactness, enhance erythropoiesis through a hyper proliferation of immature erythroid progenitors, increase erythropoietin (EPO) secretion by inducing EPO gene expression, and contribute to hemoglobin production, effect on megakaryocytes through modulation of bone marrow matrix proteins, such as fibronectin, increase the expression of fibronectin gene, alter platelet function and affects hematopoiesis in many ways (Dorgalaleh *et al.*, 2013; Jp and Srikrishna, 2012).

MATERIALS AND METHODS

Subject

The present study was carried out in the blood bank at Maysan governorate. The study population comprised 100 men aged between 20-59 year, (75) men with polycythemia and (25) men who was normally healthy a control from November 2022 to March 2023.

Blood Sample

Blood vein (5 - 6 ml) were taken from all subjects then divided into two and transferred into EDTA tube for complete blood count (CBC) determination and other part was transferred into gel tube for hormonal study. The red blood cells (RBC_s) count, hematocrit (Hct), hemoglobin concentration (Hb), white blood cells (WBC_s) count, and estimation of red blood indices mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH) and mean corpuscular hemoglobin concentration (MCHC) were obtained directly from automatic hematological analyzer (Spin cell 3) apparatus.

The CL-series Thyroid Stimulating Hormone (TSH), Thyroxine Hormone (T_4) and Triiodothyronine Hormone (T_3) assay are a Chemiluminescent Immunoassay Analyzer (CLIA) for the quantitative determination of hormones in serum (Bermudez *et al.*, 1975; Fisher, 1996; Keffer, 1996).

Statistical Analysis

The data was analyzed statistically to know the significance of the different parameters by one way ANOVA, the difference was considered to be significant at $P \leq 0.05$ the values present as means \pm SE (Bryman and Cramer, 2012).

RESULTS

Hematological Parameters

The results of present study showed increase significant ($P \leq 0.05$) in the values of RBC, Hb and Hct ($5.08 \pm 0.05 \times 10^6/\text{mm}^3$, $5.03 \pm 0.05 \times 10^6/\text{mm}^3$, $17.21 \pm 0.10 \text{ g/dL}$, $16.74 \pm 0.10 \text{ g/dL}$ and $55.07 \pm 0.28\%$, $53.6 \pm 0.25\%$ respectively). While, the values of WBCs ($7.63 \pm 0.27 \times 10^3/\text{mm}^3$, $7.5 \pm 0.40 \times 10^3/\text{mm}^3$, respectively) showed no significant ($P \leq 0.05$) differences for both groups of polycythemia (smokers and non-smokers) as compared to the control group ($4.71 \pm 0.08 \times 10^6/\text{mm}^3$, $14.34 \pm 0.06 \text{ g/dL}$, $46.50 \pm 0.33\%$ and $6.86 \pm 0.31 \times 10^3/\text{mm}^3$, respectively) (Table 1).

MCV, MCH and MCHC ($107.90 \pm 0.75 \text{ fL}$, $33.83 \pm 0.29 \text{ pg}$ and $31.36 \pm 0.01 \text{ g/dL}$ respectively) showed increase significant ($P \leq 0.05$) in smokers' group as compared to the control group ($103.76 \pm 2.27 \text{ fL}$, $32.05 \pm 0.82 \text{ pg}$ and $30.83 \pm 0.19 \text{ g/dL}$ respectively). But, MCV and MCH in non-smokers ($105.74 \pm 0.96 \text{ fL}$ and $33.26 \pm 0.37 \text{ pg}$) showed no significant ($P \leq 0.05$) between non-smokers and control group (Table 2).

Table 1: The values of blood parameters in male with polycythemia and control groups

Parameters Groups	RBCs ($10^6/\text{mm}^3$)	Hb (g/dL)	HCT (%)	WBCs ($10^3/\text{mm}^3$)
Control	4.71 ± 0.08 b	14.34 ± 0.06 c	46.50 ± 0.33 c	6.86 ± 0.31 a
Smokers Polycythemia	* 5.08 ± 0.05 a	* 17.21 ± 0.10 a	* 55.07 ± 0.28 a	7.63 ± 0.27 a
Non smokers Polycythemia	* 5.03 ± 0.05 a	* 16.74 ± 0.10 b	* 53.6 ± 0.25 b	7.5 ± 0.40 a

Means \pm SE

*The different letters refer to the significant differences among group at level of ($p < 0.05$)

The similar letters refer to non-significant among group difference at level of ($p < 0.05$).

Table 2: The values of Red Blood Cells (RBCs) indices in men with polycythemia and control group

Parameters Groups	MCV (fL)	MCH (pg)	MCHC (g/dL)
Control	103.76 ± 2.27 b	32.05 ± 0.82 b	30.83 ± 0.19 b
Smokers Polycythemia	* 107.90 ± 0.75 a	* 33.83 ± 0.29 a	* 31.36 ± 0.01 a
Non smokers Polycythemia	105.74 ± 0.96 ab	33.26 ± 0.37 ab	* 31.48 ± 0.21 a

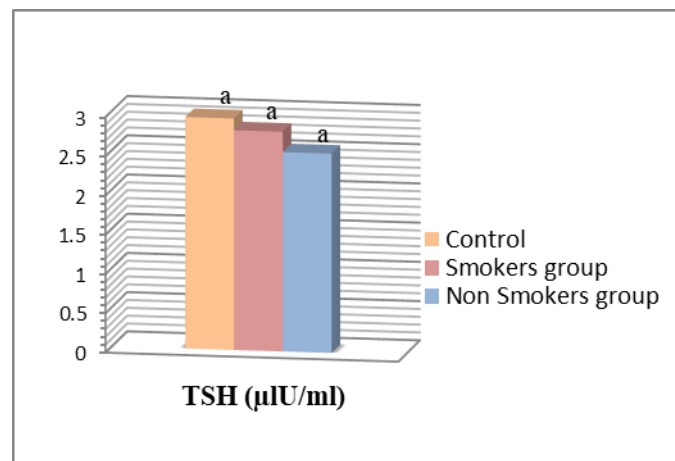
Means ±SE

*The different letters refer to significant difference among group at level of ($p \leq 0.05$)The similar letters refer to non-significant difference among group at level of ($p \leq 0.05$).**Hormonal Parameters**

Non-significant ($p \leq 0.05$) variation was recorded in the levels of TSH in the smoker and non-smoker groups (2.75 ± 0.23 , 2.54 ± 0.21 $\mu\text{IU/ml}$, respectively) in comparison with the control group (2.95 ± 0.22 $\mu\text{IU/ml}$) as shown in Fig. (1).

Non-significant ($p \leq 0.05$) variation was recorded in the levels of T_4 in the smoker and non-smoker groups (8.05 ± 0.21 , 7.74 ± 0.33 Ug/dl , respectively) in comparison with the control group (8.36 ± 0.27 $\mu\text{g/dl}$) as shown in Fig. (2).

The T_3 levels in smoker (0.86 ± 0.02 ng/dl) and non-smoker groups (0.88 ± 0.02 ng/dl) were increased significantly ($P \leq 0.05$) in comparison with control group (0.80 ± 0.02 ng/dl). While, did not differ significantly ($p \leq 0.05$) between smoker and non-smoker group as shown in Fig. (3).

**Fig. 1: The levels of Thyroid Stimulating hormone (TSH) in men with polycythemia (smokers and non-smokers) and control**

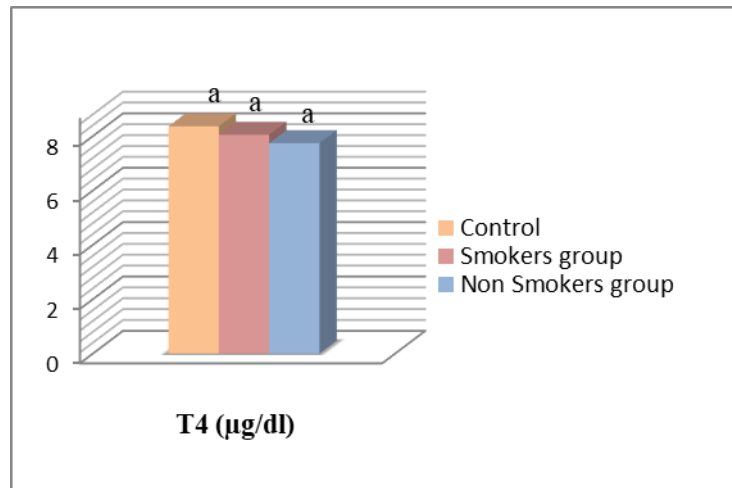


Fig. 2: The levels of Thyroxine (T₄) in men with polycythemia (smokers and non-smokers) and control

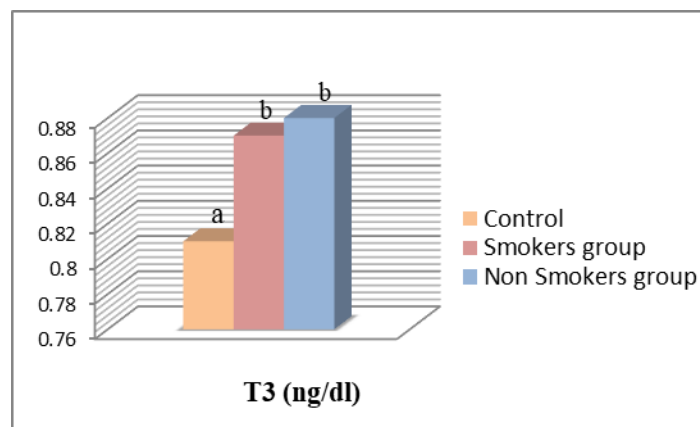


Fig. 3: The levels of Triiodothyronine (T₃) in men with polycythemia (smokers and non-smokers) and control

DISCUSSION

The results of the present study agree with a study done by (Raval and Paul, 2010), they showed very high RBC mass and a high quantity of RBCs in smokers polycythemia. Also, observed that smokers had significantly higher Hb the concentration. In fact, higher Hb concentrations are linked to higher RBCs counts or sizes. RBCs levels were noticeably higher in smokers compared to non-smokers (Tarazi *et al.*, 2008). According to another study, higher RBCs and Hct levels are associated with higher blood viscosity and coagulation in smokers (Ho, 2004).

In comparison to non-smokers, smokers' Hb values were noticeably higher (Kume *et al.*, 2009). Increased Hct levels have been linked to polycythemia and a higher risk of atherosclerosis progression and cardiovascular disease (Ferro *et al.*, 2004). The important experimental disparities revealed in the blood parameters (RBCs, Hb, and HCT) in subject's smokers in comparison with non-smokers, according to the study by (Asif *et al.*, 2013). Other studies agree with the results of our study, they show that Hb and Hct were considerably greater in smokers than in non-smokers, and that polycythemia was observed in 7.02% of all individuals (8.42% of smokers versus 6.54% of non-smokers) (Kung *et al.*, 2008).

The effect of smoking on the blood parameter may be due to CO released from smoke binds with hemoglobin to form carboxyhemoglobin (COHb), which affects tissue hypoxia and increases erythropoietin production and erythropoiesis. Plasma volume decreases in response to CO elevation of capillary permeability, similar to relative polycythemia (Pankaj *et al.*, 2014).

Smokers have higher (COHb) concentrations, which cause progressive hypoxia and changes to the hematological parameters (Lakshmi *et al.*, 2014). CO is produced by the inefficient use of carbon-containing materials, as in the state of cigarette smoking in comparison to oxygen, CO is thought to have an affinity for hemoglobin that is around 200 times higher (Carallo *et al.*, 1998). As a result, COHb complex which inhibits the release of Oxygen from hemoglobin to tissue, forms when CO combines with hemoglobin rather than Oxygen in the erythrocyte (Cronenberger *et al.*, 2008).

The number of cigarettes smoked per day increases, the mean Hb concentration and HbCO levels gradually rise. Additionally, the duration of chronic exposure to HbCO is linked to the development of polycythemia (Leifert, 2008).

Smoking has been observed to generate a unique state of linked polycythemia to chronic hypoxia, increasing the release of erythrocytes due to an increase in HbCO, and causing a decrease in plasma volume (Raval and Paul, 2010), this causes increase in Hct and Hb levels, which are thought to be a compensation strategy for CO exposure (Roethig *et al.*, 2010). When exposed the animals to the passive smoking exposure for different periods, they found a significant decrease ($p \leq 0.05$) in the total number of white blood cells and a significant increase ($p \leq 0.05$) in each of packed cell volume and Hb concentration in groups exposed to cigarettes smoking as compared with control group (Al- Attar and Jihad, 2013).

On the contrary, they showed that the WBCs count in smokers with polycythemia increased significantly than non-smokers (Al Dayyeni *et al.*, 2023).

Another study suggested that the elevated leukocyte count may be caused by nicotine's catabolic release of catecholamine, which raises blood lymphocyte numbers, the irritating effects of cigarette smoke on the respiratory system and the ensuing inflammation may also be contributing factors to a higher WBCs count, additionally, it has been proposed that inflammation-stimulating the bronchial tract causes blood circulation to produce more inflammatory markers (Calapai *et al.*, 2009).

The MCV, MCH, and MCHC levels were increased significantly in men smokers compared to control, these results are in agreement with study of (Inal *et al.*, 2014) about the effects of smoking on hematological parameters, smokers' MCV values were higher than MCV in nonsmokers. Also, they noted that smokers' MCV levels of RBCs macrocytosis were higher than those of non-smokers. Moreover, it is shown that smokers have higher levels of MCV, MCH, MCHC, and COHb, which can increase blood viscosity and vascular loading, resulting in RBC macrocytosis and hyperchromia (Kung *et al.*, 2008).

While, observed no significant differences in MCV and MCH between smokers and non-smokers, but they found a significant decline in MCHC values among smokers (Pankaj *et al.*, 2014).

In contrast, the study of (Salamzadeh, 2004) showed the amounts of MCH and MCHC values in the smoker group were markedly decreased in comparison to those of the non-smoker group.

Our study shown no significance for the levels of TSH and T_4 and increase significance in men smokers and non-smokers compared to the control group, this result is consistent with the previous study, which indicates that smoking has an impact on the pituitary gland's operation and increases the levels of several of its hormones, such as growth hormone (GH) and adrenocorticotrophic (ACTH), but has no impact on TSH levels (Kapoor and Jones, 2005). While, in another study the level of TSH in smokers' serum is lower than those of controls (Jorde and Sundsfjord, 2006).

These results agree with a study performed by Fisher and his group (1997) that they found no significant effect of smoking on T_4 levels. Contrary to findings from another study, they noted that smokers' T_4 levels were higher than those of non-smokers' (Kadkhodazadeh *et al.*, 2020).

They found higher in serum level of T_3 in smokers when compared to the control (Ahmadi *et al.*, 2012). Another study investigating the influence of smoking on thyroid hormone

levels have observed an increase in T_3 levels in smokers compared with non-smokers (Gruppen *et al.*, 2020). The results of previous studies are agreed with our study.

The people who smoke heavily, the serum level of T_3 compared to non-smoking, it has been significant decreased according to study by (Pradhan *et al.*, 2020). As a thyroid stimulant, nicotine has been used. The hypothalamic-pituitary-thyroid axis is another highly active system that nicotine can stimulate. It imitates acetylcholine's effects at specific central nicotine acetylcholinergic receptors, activating the sympathetic nervous system, which then stimulates the thyroid gland and increases T_3 output. Smokers' lower serum levels of thyroid stimulating hormone are due to its suppression by the elevated levels of serum T_3 because of negative feedback regulation (Balhara and Deb, 2014).

The mechanism through which cigarette smoking affects TSH and thyroid hormone levels is still unclear. This is not surprising since there are more than 4000 components in tobacco. Several studies have suggested that smoking lessens thyroid autoimmune processes (Wiersinga, 2013), causing changes in TSH and T_3 levels. Also proposed is a relationship between rising T_3 levels and falling TSH levels (Cryer *et al.*, 1976) a result of smokers' elevated sympathetic nerve activity (Melander *et al.*, 1977).

CONCLUSION

The current study concluded that smoking causes an increase in hematological parameters (red blood cells, hemoglobin and hematocrit) in men with polycythemia smoker and non-smoker due to an increase in the production of red blood cells from the bone marrow. In other hand the changes in the values of red blood cells indices in men with polycythemia smoker and non-smoker was higher specially in mean corpuscular hemoglobin concentration. Also, the results observed that the T_3 hormone increased in men with polycythemia smoker and non-smoker, while the T_4 and TSH hormones were not affected in smoker Polycythemia men, this maybe explain the role of smoking on hypothalamus pituitary thyroid axis.

REFERENCES

- Ahmad, T.Y.; Al-Helaly, L.A. (2009). Oxidative stress for smoking persons in suburbs Mosul City. *Raf. J. Sci.*, **20**(1), 22-32. Doi:10.33899/rjs.2009.41320
- Ahmadi, R.; Asgary, V.; Abedi, G.R. (2012). The comparison between the effects of cigarette and waterpipe smoke on serum level of TSH, T_3 and T_4 in male rats. *Razi J. Med. Sci.*, **19**(102), 12-17.
- Al Dayyeni, A.M.; Al-Gailani, B.T.; Mahdi, M.G. (2023). The role of erythropoietin levels and other hematological factors in the diagnosis of polycythemia vera in Iraqi patients. *Iraqi J. Hemat.*, **12**(1), 50-56. Doi:10.4103/ijh.ijh_8_23
- Al-Attar, H.; Jihad, T. (2013). Effect of passive smoking on some physiological and biochemical parameters in male Swiss albino mice (Mus Musculus). *Raf. J. Sci.*, **24**(6), 1-15. Doi: 10.33899/RJS.2013.74558
- Asif, M.; Karim, S.; Umar, Z.; Malik, A.; Ismail, T.; Chaudhary, A.; Rasool, M. (2013). Effect of cigarette smoking based on hematological parameters: Comparison between male smokers and non-smokers. *Turkish J. Bioch. / Turk Biyok. Derg.*, **38**(1). Doi:10.5505/tjb.2013.68077
- Balhara, Y.P.S.; Deb, K.S. (2014). Impact of tobacco on thyroid function. *Thyr. Res. Pract.*, **11**(1), 6-16. Doi:10.4103/0973-0354.124187
- Bermudez, F.; Surks, M.I.; Oppenheimer, J.H. (1975). High incidence of decreased serum triiodothyronine concentration in patients with nonthyroidal disease. *J. Clin. Endocrinol. Metab.*, **41**(1), 27-40. Doi: 10.1210/jcem-41-1-27
- Bryman, A.; Cramer, D. (2012). "Quantitative Data Analysis with IBM SPSS 17, 18 and 19: A Guide for Social Scientists". Routledge.
- Calapai, G.; Caputi, A.P.; Mannucci, C.; Russo, G.A.; Gregg, E.; Puntoni, R.; Nunziata, A. (2009). Cardiovascular biomarkers in groups of established smokers after a decade of

- smoking. *Basic Clin. Pharm. Toxicol.*, **104**(4), 322-328. Doi: 10.1111/j.1742-7843.2008.00361.x
- Carallo, C.; Pujia, A.; De Franceschi, M. S.; Motti, C.; Gnasso, A. (1998). Whole blood viscosity and haematocrit are associated with internal carotid atherosclerosis in men. *Coron. Art. Dis.*, **9**(2), 113-117.
- Cronenberger, C.; Mould, D.R.; Roethig, H.J.; Sarkar, M. (2008). Population pharmacokinetic analysis of carboxyhaemoglobin concentrations in adult cigarette smokers. *British J. Clin. Pharmacol.*, **65**(1), 30-39. Doi: 10.1111/j.1365-2125.2007.02974.x
- Cryer, P. E.; Haymond, M.W.; Santiago, J.V.; Shah, S.D. (1976). Norepinephrine and epinephrine release and adrenergic mediation of smoking-associated hemodynamic and metabolic events. *New England J. Med.*, **295**(11), 573-577. Doi: 10.1056/NEJM197609092951101
- Davis, F.B.; Cody, V.; Davis, P.J.; Borzynski, L.J.; Blas, S.D. (1983). Stimulation by thyroid hormone analogues of red blood cell Ca²⁺-ATPase activity *in vitro*. Correlations between hormone structure and biological activity in a human cell system. *J. Biol. Chem.*, **258**(20), 12373-12377.
- Dorgalaleh, A.; Mahmoodi, M.; Varmaghani, B.; Kia, O.S.; Alizadeh, S.; Tabibian, S.; Khatib, Z. K. (2013). Effect of thyroid dysfunctions on blood cell count and red blood cell indice. *Iranian J. Pedi. Hemat. Oncol.*, **3**(2), 73.
- Ferro, J.M.; Canhão, P.; Stam, J.; Bousser, M.G.; Barinagarrementeria, F. (2004). Prognosis of cerebral vein and dural sinus thrombosis: Results of the international study on cerebral vein and dural sinus thrombosis (ISCVT). *Stroke*, **35**(3), 664-670. Doi: 10.1161/01.STR.0000117571.76197.26.
- Fisher, C. L.; Mannino, D. M.; Herman, W. H.; Frumkin, H. (1997). Cigarette smoking and thyroid hormone levels in males. *Intern. J. Epidemiol.*, **26**(5), 972-977. Doi.org/10.1093/ije/26.5.972
- Fisher, D.A. (1996). Physiological variations in thyroid hormones: Physiological and pathophysiological considerations. *Clin. Chem.*, **42**(1), 135-139.
- Garmendia Madariaga, A.; Santos Palacios, S.; Guillén-Grima, F.; Galofré, J.C. (2014). The incidence and prevalence of thyroid dysfunction in Europe: A meta-analysis. *J. Clin. Endocrinol. Metab.*, **99**(3), 923-931. Doi: 10.1210/jc.2013-2409
- Gitte, R.N. (2011). Effect of cigarette smoking on plasma fibrinogen and platelet count. *Asian J. Med. Sci.*, **2**(3), 181-184. Doi:10.3126/ajms.v2i3.4261
- Gruppen, E. G.; Kootstra-Ros, J.; Kobold, A. M.; Connelly, M.A.; Touw, D.; Bos, J. H.; Dullaart, R. P. (2020). Cigarette smoking is associated with higher thyroid hormone and lower TSH levels: the PREVEND study. *Endocr.*, **67**, 613-622. Doi: 10.1007/s12020-019-02125-2
- Ho, C.H. (2004). White blood cell and platelet counts could affect whole blood viscosity. *J. Chinese Med. Assoc.*, **67**(8), 394-397.
- Inal, B.; Hacıbekiroglu, T.; Cavus, B.; Musaoglu, Z.; Demir, H.; Karadag, B. (2014). Effects of smoking on healthy young men's hematologic parameters. *North. Clin. Istanbul*, **1**(1), 19. Doi: 10.14744/nci.2014.39974
- Jamal, A.; Phillips, E.; Gentzke, A.S.; Homa, D.M.; Babb, S.D.; King, B.A.; Neff, L.J. (2018). Current cigarette smoking among adults—United States, 2016. *Morbid. Mortal. Weekly Rep.*, **67**(2), 53. Doi: 10.15585/mmwr.mm6702a1
- Jorde, R.; Sundsfjord, J. (2006). Serum TSH levels in smokers and non-smokers. The 5th Tromsø study. *Experim. Clin. Endoc. Diab.*, **114**(07), 343-347. Doi: 10.1055/s-2006-924264.
- Jp, G.; Srikrishna, R. (2012). Role of red blood cell distribution width (rdw) in thyroid dysfunction. *Int. J. Biol. Med. Res.*, **3**(2), 1476-1478.
- Kadkhodazadeh, H.; Amouzegar, A.; Mehran, L.; Gharibzadeh, S.; Azizi, F.; Tohidi, M. (2020). Smoking status and changes in thyroid-stimulating hormone and free thyroxine levels during a decade of follow-up: The Tehran thyroid study. *Caspian J. Internal Med.*, **11**(1), 47. Doi: 10.22088/cjim.11.1.47.

- Kapoor, D.; Jones, T.H. (2005). Smoking and hormones in health and endocrine disorders. *European J. Endoc.*, **152**(4), 491-499. Doi: 10.1530/eje.1.01867.
- Kawa, M. P.; Grymula, K.; Paczkowska, E.; Bańkiewicz-Masiuk, M.; Dąbkowska, E.; Koziółek, M.; Machaliński, B. (2010). Clinical relevance of thyroid dysfunction in human haematopoiesis: Biochemical and molecular studies. *European J. Endoc.*, **162**(2), 295-305. Doi: 10.1530/EJE-09-0875.
- Keffer, J.H. (1996). Preanalytical considerations in testing thyroid function. *Clin. Chem.*, **42**(1), 125-134. Doi.org/10.1093/clinchem/42.1.125
- Kume, A.; Kume, T.; Masuda, K.; Shibuya, F.; Yamazaki, H. (2009). Dose-dependent effects of cigarette smoke on blood biomarkers in healthy Japanese volunteers: Observations from smoking and non-smoking. *J. Health Sci.*, **55**(2), 259-264. Doi:10.1248/jhs.55.259
- Kung, C.M.; Wang, H.L.; Tseng, Z.L. (2008). Cigarette smoking exacerbates health problems in young men. *Clin. Invest. Med.*, E138-E149. Doi: 10.25011/cim.v31i3.3471.
- Lakshmanan, A.; Saravanan, A. (2014). Effect of intensity of cigarette smoking on haematological and lipid parameters. *J. Clin. Diagn. Res., JCDR*, **8**(7), BC11. Doi: 10.7860/JCDR/2014/9545.4612
- Leifert, J.A. (2008). Anemia and cigarette smoking. *Intern. J. Lab. Hematol.*, **30**(3), 177-184.
- 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. *Med. Arch.*, **71**(2), 132. Doi: 10.5455/medarh.2017.71.132-136.
- Melander, A.; Westgren, U.; Ericson, L. E.; Sundler, F. (1977). Influence of the sympathetic nervous system on the secretion and metabolism of thyroid hormone. *Endocr.*, **101**(4), 1228-1237. Doi.org/10.1210/endo-101-4- 1228
- Pankaj, J.; Reena, J.; Mal, K.L.; Ketan, M. (2014). Effect of cigarette smoking on hematological parameters: comparison between male smokers and non-smokers. *IJSN*, **5**(4), 740-3. Doi: 10.5455/medarh.2017.71.132-136
- Pradhan, A.; Ganguly, A.; Naik, M.N.; Nair, A.G.; Desai, S.; Rath, S. (2020). Thyroid eye disease survey: An anonymous web-based survey in the Indian subcontinent. *Indian J. Ophthalm.*, **68**(8), 1609. Doi: 10.4103/ijo.IJO_1918_19
- Raval, M.; Paul, A. (2010). Cerebral venous thrombosis and venous infarction: Case report of a rare initial presentation of smoker's polycythemia. *Case Rep. Neurol.*, **2**(3), 150-156. Doi.org/10.1159/000322571
- Roethig, H. J.; Koval, T.; Muhammad-Kah, R.; Jin, Y.; Mendes, P.; Unverdorben, M. (2010). Short term effects of reduced exposure to cigarette smoke on white blood cells, platelets and red blood cells in adult cigarette smokers. *Reg. Toxic. Pharm.*, **57**(2-3), 333-337. Doi: 10.1016/j.yrtph.2010.04.005.
- Salamzadeh, J. (2004). The hematologic effects of cigarette smoking in healthy men volunteers.
- Tarazi, I.S.; Sirdah, M.M.; El Jead, H.; Al Haddad, R.M. (2008). Does cigarette smoking affect the diagnostic reliability of hemoglobin $\alpha_2\delta_2$ (HbA2)? *J. Clin. Lab. Anal.*, **22**(2), 119-122. Doi: 10.1002/jcla.20228
- Taylor, P.N.; Albrecht, D.; Scholz, A.; Gutierrez-Buey, G.; Lazarus, J.H.; Dayan, C.M.; Okosieme, O.E. (2018). Global epidemiology of hyperthyroidism and hypothyroidism. *Nat. Rev. Endocr.*, **14**(5), 301-316. Doi: 10.1038/nrendo.2018.18.
- Wiersinga, W.M. (2013). Smoking and thyroid. *Clin. Endocr.*, **79**(2), 145-151. Doi: 10.1111/cen.12222.
-

تأثير التدخين على معايير الدم وهرمونات الغدة الدرقية عند الرجال المصابين بفرط كريات الدم

مروة علي مجيد زينب عبد الجبار رضا العلي

قسم علوم الحياة/ كلية العلوم/ جامعة ميسان/ ميسان/ العراق

الملخص

يعد التدخين السبب الرئيسي للوفيات، حالات الإعاقة والأمراض التي يمكن الوقاية منها في جميع أنحاء العالم، يتعرض المدخنون لمجموعة متنوعة من المركبات الخطرة والتي تحفز نخاع العظم لإنتاج المزيد من كرات الدم الحمراء، لذا هدفت هذه الدراسة الى معرفة تأثير التدخين على الرجال المصابين بكثرة الحمر من خلال دراسة معايير الدم وهرمونات الغدة الدرقية، أجريت الدراسة على 100 رجل (75 مصابين بكثرة الحمر و 25 رجلاً سليمين) تتراوح أعمارهم بين (20-59) سنة، تم جمع العينات من بنك الدم في محافظة ميسان، للفترة من تشرين الثاني 2022 إلى آذار 2023.

اظهرت النتائج زيادة معنوية في قيم كريات الدم الحمراء وخضاب الدم وحجم خلايا الدم المرصوصة لكلا مجموعتي كثرة الحمر (المدخنين وغير المدخنين) مقارنة بمجموعة السيطرة، لم تظهر كريات الدم البيضاء فروقات معنوية مقارنة بمجموعة السيطرة. كما اظهرت دلائل الكرية زيادة معنوية مقارنة مع مجموعة السيطرة. لم تسجل اختلافات معنوية في قيم الهرمون المحفز للدرقية وهرمون التايروكسين بين مجموعتي الرجال المصابين بكثرة الحمر (مدخنين وغير مدخنين)، اما بالنسبة الى هرمون ثلاثي يود الثيرونين ازداد معنوياً لكلا مجموعتي كثرة الحمر (المدخنين وغير المدخنين) مقارنة بمجموعة السيطرة. من النتائج اعلاه يمكن الاستنتاج بان التدخين له تأثير على معايير الدم وهرمونات الدرقية عند الرجال الذين يعانون من كثرة كريات الدم الحمراء.

الكلمات الدالة: التدخين، الهيموغلوبين، كثرة الحمر، الدرقية، هرمونات.