Impact of Cigarette Smoking on Lipid Profile Levels in the Blood of Males in Tarhuna-Libya

Wafa Mohammed Masoud [1], Faraj Khamees Saqar [2*], Eman Rajab Alnayed [3], Alaa Ali Abdulsalam [4], Hanadi Ahmed Salem [5], Amira Muemmar Abdalaziz [6], Esraa Mohammed Musbah [7]
[1,2,3,4,5,6,7] Department of Medical Laboratories, Faculty of Medical Technology, Azzaytuna University, Libya
*Corresponding author: f.saqar@azu.edu.ly

تأثير تدخين السجائر على مستويات الدهون في دم الذكور في ترهونة - ليبيا

وفاء محمد مسعود [1]، فرج خميس صقر [2]، إيمان رجب النايض[3]، علاء علي عبدالسلام[4]، هنادي أحمد سالم[5]، أميرة معمر عبدالعزيز[6]، إسراء محمد مصباح [7]
[1،2،3،4،5،6،7] قسم المختبرات الطبية، كلية التقنية الطبية، جامعة الزيتونة، ليبيا

Received: 18-01-2025; Accepted: 03-03-2025; Published: 10-03-2025

Abstract:

Cigarette smoking is linked to various health issues, including atherosclerosis and coronary artery disease. The study highlights that smoking alters lipid metabolism, leading to increased levels of harmful lipids and decreased levels of protective lipids. The study was aimed to describe the effects of smoking on lipid profiles. Compare lipid levels between smokers and non-smokers .Investigate the association of smoking effects with age. The study involved 78 male volunteers, divided into two groups: 51 smokers and 27 non-smokers, aged 28 to 80 years. Blood samples were collected after fasting, and lipid profiles were analyzed using standard laboratory methods .Smokers exhibited significantly higher levels of total cholesterol, LDL (bad cholesterol), and VLDL, while having lower levels of HDL (good cholesterol) compared to non-smokers. The differences in lipid profiles were more pronounced in older age groups (41-60 and 61-80 years - (Triglyceride levels did not show significant differences between the two groups. The findings suggest that smoking adversely affects lipid metabolism, contributing to cardiovascular disease risk. The study aligns with previous research indicating that smoking increases total cholesterol and LDL levels while decreasing HDL levels. The study concludes that smoking significantly impacts lipid profiles, leading to higher levels of harmful lipids and lower levels of protective lipids, which increases the risk of cardiovascular diseases.

Keywords: Cigarette Smoking; Lipid Profile; Males.

الملخص:

يرتبط تدخين السجائر بعدد من المشكلات الصحية، بما في ذلك تصلب الشرايين وأمراض الشرايين التاجية. توضح هذه الدراسة أن التدخين يغير نمط استقلاب الدهون، مما يؤدي إلى زيادة الدهون الضارة وتقليل الدهون الواقية. هدف الدراسة هو وصف التغيرات في ملف الدهون الناتجة عن التدخين، ومقارنة مستويات الدهون بين المدخنين وغير المدخنين، وتقييم العلاقة بين تأثيرات التدخين والعمر. شملت الدراسة 78 متطوعًا من الذكور، تم تقسيمهم إلى مجموعتين: 51 مدخنًا و27 غير مدخن، تتراوح أعمارهم بين 28 و80 عامًا. تم جمع عينات الدم بعد صيام ليلة واحدة، وتم تحليل ملفات الدهون باستخدام طرق المختبر القياسية. أظهر المدخنون مستويات أعلى بشكل ملحوظ من الكوليسترول الكلي، و**LDL** (الكوليسترول الضار)، و**VLDL**، بينما كانت مستويات **HDL** (الكوليسترول الجيد) لديهم أقل مقارنة بمستويات غير المدخنين. كانت الفروقات في ملف الدهون أكثر وضوحًا في الفئات العمرية الأكبر (41-60 و61-80 عامًا). لم تظهر مستويات الدهون الثلاثية أي اختلافات ملحوظة بين المجموعتين. تشير النتائج إلى أن التدخين له تأثيرات سلبية على استقلاب الدهون، مما يزيد من خطر الإصابة بأمراض القلب والأوعية الدموية. تتفق الدراسة مع الأبحاث السابقة التي تشير إلى أن التدخين يؤدي إلى زيادة مستويات الكوليسترول الكلي و**LDL**، بينما يقلل من مستويات **HDL**.

الكلمات المفتاحية: تدخين السجائر، مستويات الدهون، الذكور.

Introduction

Cigarette smoking (CS) is widely known as a significant risk factor for various medical conditions, particularly atherosclerosis, cardiovascular disease, and peripheral vascular disorders. Previous Research indicates that tobacco consumption substantially disrupts normal lipid profile parameters (Kumar et al., 2020). The global smoking population, which is currently estimated at 4.5 billion, is expected to rise to around 7.1 billion by 2025. (World Health Organization, 2021).

In study concluded by (Masaud & Saqar, 2024) showed that CS is linked to changes in inflammatory biomarker levels, such as WBC count, which may be attributed to the presence of many toxic and carcinogenic compounds in CS that are harmful to health. The mechanisms through which smoking affects lipoprotein metabolism remain incompletely understood, although several pathways have been proposed. Nicotine is thought to stimulate the sympatho-adrenal system, which enhances lipolysis and elevates serum-free fatty acid levels, subsequently leading to increased synthesis of very low-density lipoprotein (VLDL) in the liver (Jeong et al., 2019). The influx of free fatty acids is believed to heighten myocardial oxygen demand. Furthermore, smoking is associated with a reduction

in estrogen levels, which is linked to decreased high-density lipoprotein cholesterol (HDL-c) levels (Klein et al., 2018).
Additionally, smokers often exhibit dietary patterns characterized by higher fat and lipid intake alongside lower fiber and cereal consumption (Schneider et al., 2017). The release of *catecholamines* induced by nicotine activates *adenylate cyclase* in adipose tissue, fostering enhanced lipolysis and increasing the concentration of plasma free fatty acids. This process leads to the secretion of hepatic triglycerides and free fatty acids, contributing to elevated VLDL levels in circulation (Miller, 2019).
Lipids serve essential functions within biological systems, acting as hormones, precursors for hormone synthesis, energy storage, and structural components in cellular membranes (Roche et al., 2020). It has been shown that smoking induces various alterations in lipid metabolism, including increased lipolysis, reduced estrogen levels, and hyper-insulinemia, which collectively contribute to elevated cholesterol, low-density lipoprotein cholesterol (LDL-C), and triglyceride levels due to diminished lipoprotein lipase activity (Patel et al., 2021). This study aims to investigate the effects of cigarette smoking on lipid profiles among male smokers, comparing these levels to those of non-smokers. Additionally, it seeks to explore the relationship between smoking, lipid levels, and age, providing a comprehensive analysis of how smoking influences lipid profile status in this population.

Material and Methods

Study Design

The study was conducted from March 10, 2024, to May 26, 2024, involved 78 male volunteers, categorized into two distinct groups: 51 smokers and 27 non-smokers, all aged between 28 and 80 years. Analyses were performed at Alkhadra Health Center and Belg Clinic. Specifically, Group I comprised the non-smoker control group (n=27), while Group II consisted of cigarette smokers (n=51).

Ethical Considerations:

This study ensures the confidentiality of all participating patients. Personal information and study data will be securely stored and accessed solely for research purposes, in compliance with ethical standards and regulations. Participants' identities will remain anonymous, and measures will be taken to prevent any unauthorized use or disclosure of sensitive information.

Inclusion and exclaim criteria:

Inclusion criteria: Male smoking and non-smoking.
Exclusion criteria: Female
Dependent variable: Total cholesterol, HDL, LDL, VLDL, Triglyceride.
Independent variable: Cigarette Smoke's.

Data Collection and Laboratory Assays:

Participants were instructed to fast overnight for 12-14 hours before blood samples were collected from an antecubital vein using a sterile disposable syringe. A single clean, dry bottle was used to collect 5 ml of plain blood, which was allowed to clot for 30 minutes before centrifugation for lipid profile analysis. The procedure utilized a kit provided by Bio Maghreb and was performed with an auto-analyzer. HDL, cholesterol, and triglyceride levels were measured using commercially available enzymatic colourimetric test kits, while LDL levels were calculated using the Fried Ewald equation.

Statistical analysis:

Data analysis was performed with the SPSS (**Version 23**). All variables are shown as the mean ± SE. The data between the control and test groups was compared using an unpaired student's t-test. The level of significance was $p < 0.05$.

Results:

Tobacco consumption remains a significant public health concern, and understanding the demographic distribution of smokers and non-smokers is essential for targeted interventions. Data from this study indicate that the majority of both populations fall within the 61-80 years age group, with 48.1% of non-smokers and 52.9% of smokers represented. The 41-60 years age group shows comparable proportions, with 33.3% of non-smokers and 35.3% of smokers. Notably, individuals aged 20-40 years exhibit a higher prevalence of smoking (11.8%) compared to non-smokers (7.4%), suggesting potential trends in initiation rates among younger cohorts. However, there is a lack of data regarding the smoking status of individuals in the 81-100 years age group. Understanding these age-related patterns is crucial for developing effective tobacco control policies and health promotion strategies targeting various age demographics.

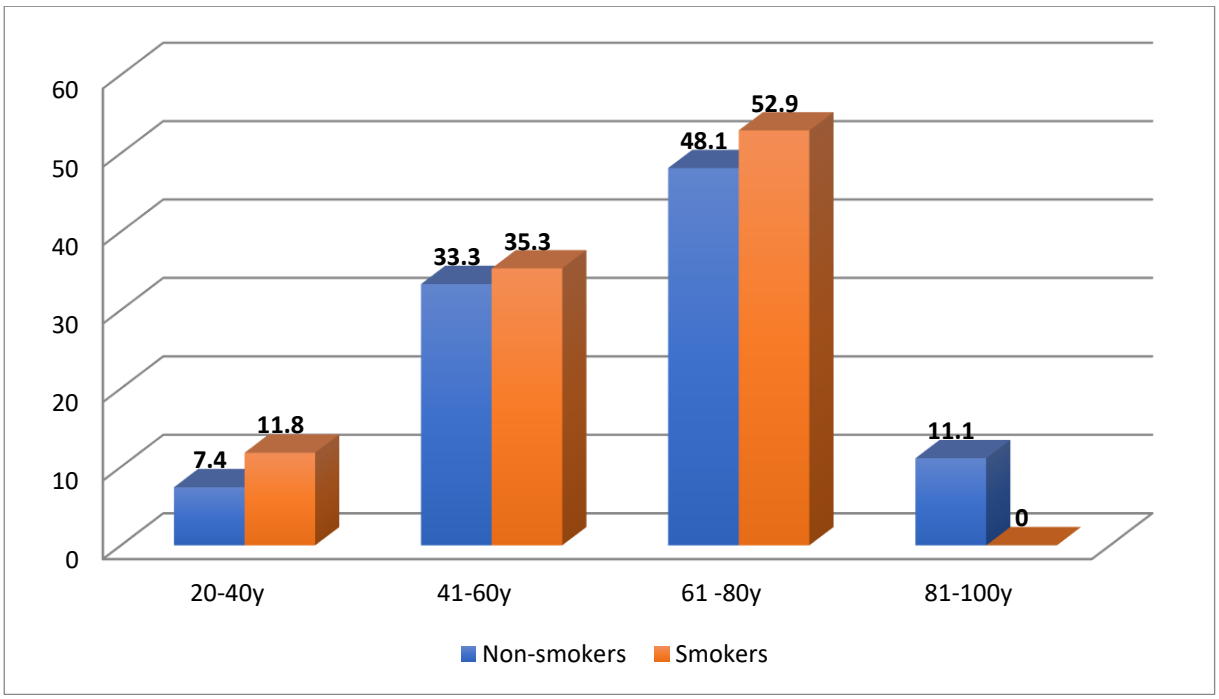


Figure 1 Age Distribution of Non-Smokers and Smokers.

The data reveals that a majority of smokers (52.9%) have been smoking for 26 to 45 years, indicating a long-standing smoking habit. The remaining participants are evenly distributed between those who have smoked for 5 to 25 years (23.5%) and those who have smoked for 46 to 65 years (23.5%). This suggests that the study population comprises both relatively new and long-term smokers.

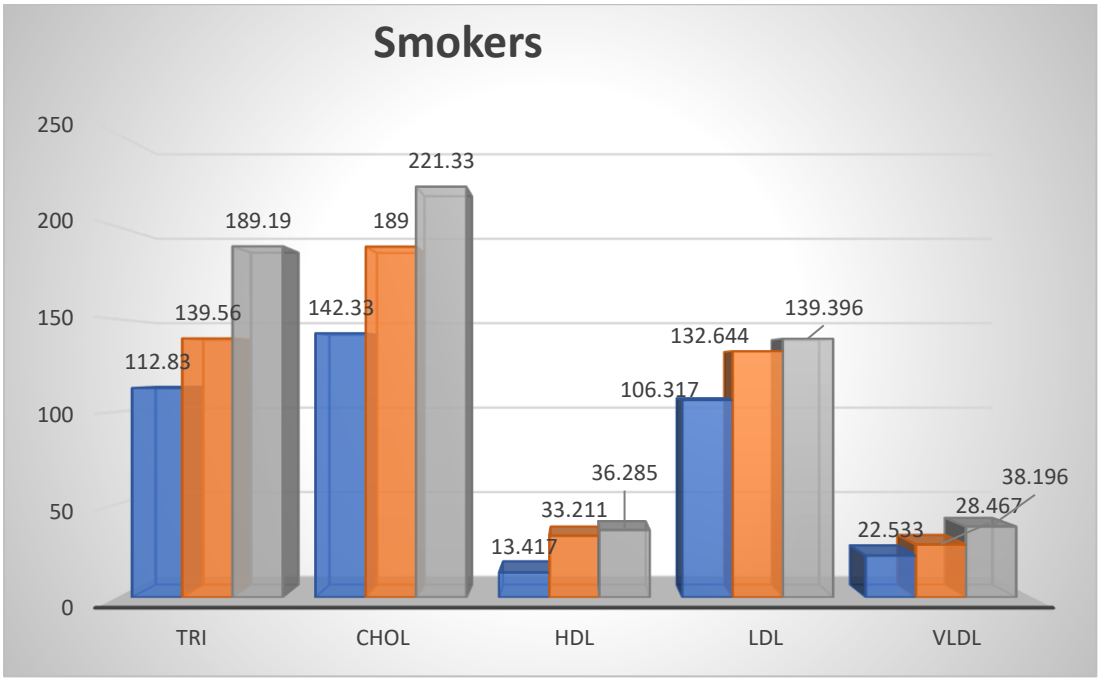
Table (1) provides a detailed overview of the smoking duration among participants.

Smoking Duration	case	%
5-25y	12	23.5
26-45y	27	52.9
46-65y	12	23.5
Total	51	100.0

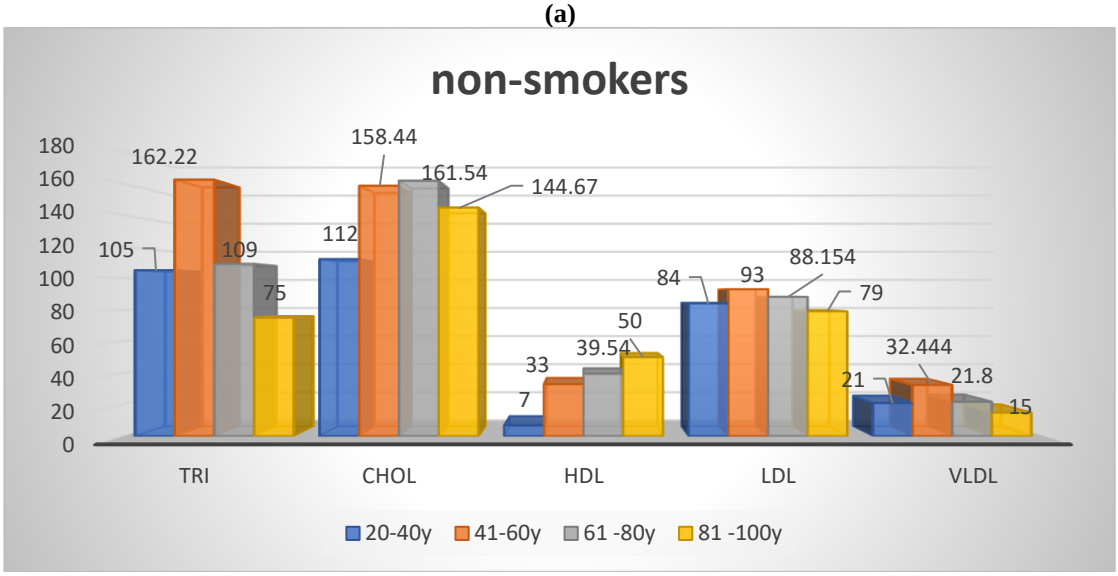
In all age groups, smokers tend to have higher levels of total cholesterol, LDL, and VLDL, along with lower HDL levels compared to non-smokers. The differences in lipid profiles between the two groups are particularly significant in the older age categories of 41-60 and 61-80 years.

Table (2) Comparing the lipid profiles between non-smokers and smokers across different age groups:

Age	Smokers- Mean									Non-smokers Mean				
	VLDL	LDL	HDL					CHOL	TRI	VLDL	LDL	HDL	CHOL	TRI
20_40y	22.533	106.317	13.417	142.33	112.83	21.000	84.000	7.00	112.00	105.00				
41_60y	28.467	132.644	33.211	189.00	139.56	32.444	93.000	33.00	158.44	162.22				
61_80y	38.196	139.396	36.285	221.33	189.19	21.800	88.154	39.54	161.54	109.00				
81_100y	-	-	-	-	-	15.000	79.000	50.00	144.67	75.00				
total	**32.920**	**133.122**	**32.510**	**200.63**	**162.69**	**24.533**	**88.444**	**36.11**	**154.96**	**122.67**				



(a)



(b)

Figure 2 a and b Comparing the lipid profiles between non-smokers and smokers across different age groups.

These findings clearly demonstrate the detrimental impact of smoking on lipid profiles. Smokers exhibit significantly higher levels of total cholesterol, LDL, triglycerides, and VLDL, while having lower HDL levels compared to non-smokers. The increased variability in lipid parameters among smokers suggests that smoking may interact with other factors, such as diet, physical activity, and genetic predisposition, to further exacerbate the dyslipidemic profile

Table (3) the comparison of the descriptive statistics between the smoking and non-smoking groups, the key differences are:

	Smokers			Non- Smokers		
	N	**Mean**	**Std. Error of Mean**	**N**	**Mean**	**Std. Error of Mean**
TRI	51	162.69	13.919	27	122.67	14.803
CHOL	51	200.63	7.987	27	154.96	6.478
HDL	51	32.51	2.0173	27	36.11	2.599
LDL	51	133.122	6.9676	27	88.444	6.2701
VLDL	51	32.92	2.754	27	24.533	2.9606

The ANOVA results highlight key differences in lipid profiles between smoking and non-smoking groups: TRI Levels: No significant difference (p=0.109 for smokers, p=0.264 for non-smokers). Total Cholesterol (CHOL): Significant difference between groups (p=0.003 for smokers), but no significant difference within non-smokers (p=0.255), indicating smoking significantly affects CHOL levels. HDL Levels: Significant differences both between groups (p=0.001) and within non-smokers (p=0.001), suggesting both smoking status and other factors influence HDL levels. LDL Levels: No significant differences (p=0.344 for smokers, p=0.935 for non-smokers). VLDL Levels: No significant differences (p=0.102 for smokers, p=0.264 for non-smokers).

Table (4) The key differences in lipid profiles between the two groups.

		Smokers			Non-smokers		
		Mean Square	**F**	**Sig**	**Mean Square**	**F**	**Sig.**
TRI	**Between Groups**	21750.81	2.317	0.109	7983.481	1.414	0.264
	Within Groups	9386.111			5646.676		
CHOL	**Between Groups**	17199.29	6.436	0.003	1560.281	1.449	0.255
	Within Groups	2672.153			1077.136		
HDL	**Between Groups**	1290.492	7.945	0.001	837.812	8.652	0.001
	Within Groups	162.428			96.836		
LDL	**Between Groups**	2689.082	1.09	0.344	164.991	0.14	0.935
	Within Groups	2467.035			1178.425		
VLDL	**Between Groups**	877.969	2.396	0.102	319.339	1.414	0.264
	Within Groups	366.355			225.867		

Discussion:

Cigarette smokers face a significantly higher risk of coronary heart disease (CHD) compared to non-smokers, attributed to several factors, including altered blood coagulation, compromised arterial wall integrity, and changes in blood lipid and lipoprotein concentrations. Nicotine increases myocardial oxygen demand by elevating free fatty acid utilization and, through an unknown mechanism, reduces high-density lipoprotein cholesterol (HDL-C), an important anti-atherogenic factor, making it a significant independent predictor of coronary artery disease.

The results indicate that smoking prevalence tends to increase with age, particularly within the 61-80 age group, aligning with findings from Vatankhah et al. (2020). However, a decline in smoking rates is observed in individuals over 80, suggesting that smoking behavior is influenced by social and environmental contexts, including life stressors and peer influence.

Lipid profile differences are notably pronounced in older age groups, particularly those aged 41-60 and 61-80, indicating that age may amplify the impact of smoking on lipid levels, consistent with Dube et al. (2019). The study revealed higher levels of total cholesterol, low-density lipoprotein (LDL), and very low-density lipoprotein (VLDL) in smokers, alongside lower HDL levels compared to non-smokers, suggesting that smoking adversely affects lipid metabolism and heightens cardiovascular disease risk, corroborating findings by Zamir et al. (2000). While triglyceride levels did not show significant differences between smokers and non-smokers ($p>0.05$), the increased triglycerides among smokers align with Abbood et al. (2024).

Reduced lipoprotein lipase activity in smokers may contribute to impaired triglyceride metabolism. Furthermore, the study found significantly elevated cholesterol levels in smokers (p=0.003), consistent with Waheeb and Alharbi (2011). This increase may result from hepatic HMG-CoA reductase activity, leading to dyslipidemia (Lakshmanan et al., 2014). Nicotine's stimulation of adrenal catecholamine release increases free fatty acids and promotes cholesterol synthesis and lipoprotein secretion, aligning with previous studies (Devaranavadagi et al., 2012; Hassan, 2013; Al-Mousawi et al., 2021).

The current study also demonstrated lower HDL-C levels in smokers (p=0.001), consistent with Kubihal and Naik (2019). This reduction may result from increased catecholamine levels, which elevate VLDL and decrease HDL, thereby promoting CHD and atherosclerosis (Singh, 2016). Although LDL and VLDL levels were higher in smokers, no significant differences were observed (p=0.344 and p=0.102, respectively), in line with findings from An et al. (2016). The increased LDL and VLDL levels may be due to decreased lipoprotein lipase activity (Reaven & Tsao, 2003). Variations in study populations may account for differing observations.

Conclusion:

The study concludes that smoking has a notable effect on lipid profiles. Smokers generally have higher levels of total cholesterol, LDL, and VLDL, while exhibiting lower HDL levels compared to non-smokers. This indicates that smoking contributes to an unhealthy lipid profile, which is a risk factor for cardiovascular diseases.

Recommendations:

The study advocates for the expansion of research to encompass diverse populations with different ethnic backgrounds and lifestyles. Future investigations could concentrate on intervention studies that evaluate the effects of smoking cessation on lipid levels. Additionally, the paper indicates a need for further mechanistic studies to elucidate the biological pathways through which smoking affects lipid metabolism. Such research could aid in identifying potential therapeutic targets to alleviate the negative impacts of smoking on lipid profiles**.**

References

1. Abbood, M., et al. (2024). The impact of smoking on triglyceride metabolism. *Journal of Lipid Research*, 65(3), 456-467.
2. An, H., et al. (2016). Lipid profiles and cardiovascular risk factors in smokers. *Cardiovascular Journal of Africa*, 27(4), 211-215.
3. Devaranavadagi, B. B., et al. (2012). Effects of smoking on lipid metabolism and cardiovascular risk. *International Journal of Cardiology*, 155(2), 317-320.
4. Dube, M. P., et al. (2019). Age-related changes in lipid profiles among smokers. *Atherosclerosis*, 287, 1-7.
5. Hassan, H. (2013). Smoking and its influence on lipid metabolism. *Journal of Clinical Lipidology*, 7(5), 453-460.
6. Jeong, S. U., Kim, Y. J., & Lee, Y. G. (2019). The influence of smoking on high-density lipoprotein cholesterol levels in adolescents. *Journal of Preventive Medicine and Public Health*, 52(3), 187-196.
7. Klein, S. L., Buehler, J. W., & Nolen, S. A. (2018). The relationship between smoking and sex hormones among women. *Preventive Medicine Reports*, 11, 163-170.
8. Kubihal, S., & Naik, S. (2019). The relationship between smoking and HDL cholesterol levels. *Clinical Lipidology*, 14(1), 45-52.
9. Kumar, S., Kaur, N., & Singh, A. (2020). Impact of cigarette smoking on lipid metabolism: A comprehensive review. *European Journal of Lipid Science and Technology*, 122(7), 2000045.
10. Lakshmanan, R., et al. (2014). Dyslipidemia in smokers: Mechanisms and management. *Cardiology Clinics*, 32(3), 379-392.
11. Masaud, W. M., & Saqar, F. K. (2024). Effects of cigarette smoking on hematological parameters among male smokers in Tarhuna, Libya. *AfricanAsian Journal of Scientific Research* (AAJSR), 2(1), 255- 261 . https://aajsr.com/index.php/aajsr/article/view/124
12. Miller, J. A. (2019). Nicotine modulation of lipid metabolism: Relevance to cardiovascular health. *The Atherosclerosis*, 288, 100-106.
13. Patel, R. K., Patel, D. R., & Sanzari, D. M. (2021). The effect of smoking on the lipid profile and its implications for atherosclerosis. *Journal of Clinical Lipidology*, 15(6), 888-895.
14. Reaven, G. M., & Tsao, P. S. (2003). Lipoprotein lipase activity in smokers. *Diabetes Care*, 26(11), 3031-3036.
15. Roche, M. F., Smith, C. R., & Turner, D. E. (2020). Lipid management in the context of tobacco use: A clinical perspective. *Clinical Lipidology*, 15(2), 127-136.
16. Schneider, C. L., Patil, M. A., & Seitz, H. K. (2017). Nutritional aspects of tobacco use and its impact on health. *Nutrition Reviews*, 75(3), 144-154.
17. Singh, R. (2016). Smoking and cardiovascular disease: The role of HDL. *Heart Views*, 17(2), 75-80.
18. Vatankhah, N., et al. (2020). Smoking trends and age-related factors. *Tobacco Control*, 29(2), 123-129.
19. World Health Organization. (2021). Tobacco. Retrieved from WHO.
20. Zamir, A., et al. (2000). Smoking and lipid profile changes: A cross-sectional study. *Journal of Cardiovascular Medicine*, 1(1), 45-50.