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Impact of Cigarette Smoking on Serum Lipids in Adult Jordanian Men: A Cross-Sectional Comparative Study with Dose–Response Analysis

Saad. AL-Fawaeir

1 Department of Medical Laboratory Science, Faculty of Allied Medical Sciences, Jadara University, Irbid, Jordan.

Corresponding author email address:
s.alfawaeir@jadara.edu.jo

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ABSTRACT

Objective: To examine differences in serum lipid profile between adult male smokers and non-smokers in Jordan and to assess dose–response patterns by smoking intensity.

Methods and Materials: In a cross-sectional comparative study at a private outpatient center in Amman (January–May 2022), 80 adult men were enrolled: 60 smokers and 20 non-smokers matched on age and BMI. Smokers were categorized by daily consumption: less than 10, 10–20, and more than 20 cigarettes. After a 12-hour fast, blood was drawn to measure total cholesterol (TC), triglycerides (TG), HDL-C, and calculated LDL-C using standardized assays on a Cobas 501 analyzer. Group comparisons used t tests/ANOVA with $p<0.05$ considered significant.

Findings: Smokers versus non-smokers showed higher mean LDL-C (137.7±38.2 vs 116.9±25.6 mg/dl; p=0.037), whereas between-group differences for TC (217.8±35.6 vs 180.4±18.3 mg/dl; p=0.087), TG (195.7±42.3 vs 165.8±17.9 mg/dl; p=0.076), and HDL-C (37.8±8.8 vs 41.7±6.4 mg/dl; p=0.056) were not statistically significant. Across smoking-intensity categories, a graded pattern emerged: higher TC, TG, and LDL-C with increasing consumption, and lower HDL-C in heavier smokers (all trend p values ≤0.035). The abstract in the original manuscript incorrectly stated that smokers had higher HDL-C; in the present data, HDL-C is lower among smokers and decreases with higher intensity.

Conclusion: Among adult Jordanian men, smoking is associated with higher LDL-C and an adverse dose–response pattern across lipids, with HDL-C declining as smoking intensity increases. These findings support lipid screening and cessation counseling for smokers. Larger, population-based studies with adjustment for diet, activity, and socioeconomic factors are warranted.

Keywords: Smoking, Lipid profile, Triglycerides, Dose–response, Adult men.

Introduction

Globally, tobacco use continues to be as among the main avoidable causes of fatalities and illnesses, significantly increasing the burden of metabolic and cardiovascular disorders worldwide (Khan Minhas et al., 2024). The World Health Organisation (WHO) claims that smoking kills more than eight million people annually, of which over seven million are directly related to smoking and an additional 1.3 million are due to indirect smoking inhalation (Organization, 2018). The incidence of tobacco use in Jordan is among the highest in the world and the region. According to Jordan's STEPs surveys, the proportion of male tobacco users every day rose from 49.6% in 2007 to 58.0 % in 2019, while the proportion of female tobacco users daily rose from 5.7% in 2007 to 10.8% in 2019 (Al-Sheyab et al., 2024). The incidence of smoking is still high even though its negative effects are widely known, particularly in countries with poor and medium incomes (Sathish et al., 2022). Among its many detrimental effects on health, smoking cigarettes is known to increase the danger of

dyslipidaemia, which is a crucial component in the development of atherosclerosis and the ensuing cardiovascular problems (Ishida et al., 2024).

It is commonly known that the degradation of lipids plays a part in atherosclerosis evolution. The chances of coronary artery disease and cerebrovascular events is greatly increased by changes in lipid and lipoprotein content, such as elevated TG, TC, and LDL-C and reduced HDL-C (Zhang et al., 2022). It has been demonstrated that smoking aggravates these lipid-related disorders, hastening the atherogenic cycle (Kotlyarov, 2023). Knowing how smoking cigarettes specifically affects lipid ratios and blood lipid markers can help develop more focused preventative and treatment plans for CVD (Gallucci et al., 2020).

According to biochemistry, tobacco smoke comprises a variety of free oxidants and radicals that cause systemic inflammation and oxidative stress, two major factors that contribute to endothelial dysfunction and lipid peroxidation (Higashi, 2022). By changing hepatic enzyme production and compromising lipid circulation, nicotine and other harmful substances in tobacco smoke also affect lipid metabolism (Ma et al., 2020). Together, these processes increase the possibility of cardiovascular disease by producing an undesirable lipid profile that is marked by higher atherogenic lipids and lower protective lipoproteins (Fu et al., 2024).

Atherosclerotic CVD can be predicted more accurately by lipid ratios, including TC/HDL, TG/HDL, and LDL/HDL ratios, than through single lipid markers (Muscella et al., 2020). These ratios provide a more integrated assessment of the balance between atherogenic and protective lipids in the circulation Gaggini et al., (2022) Smoking-induced disturbances in these ratios have been reported in several studies, with a consistent pattern of increased atherogenic indices among smokers (Aslam et al., 2025; Herath et al., 2022; Qasim et al., 2020). Such alterations not only serve as biomarkers of cardiovascular risk but may also guide clinicians in risk stratification and management (Moosazadeh et al., 2024).

There is a lack of epidemiological research that consistently shows a relationship of dosage to response between the severity of dyslipidaemia and smoking severity (Moosazadeh et al., 2024). Heavier smokers tend to exhibit more pronounced lipid abnormalities compared to light smokers, reflecting a cumulative effect of toxic exposure (Lakshmanan & Saravanan, 2014). Moreover, the duration of smoking has been positively correlated with the extent of lipid derangement, suggesting that chronic exposure amplifies metabolic dysregulation (Qasim et al., 2020). Conversely, lipid profiles have been shown to gradually improve after stopping smoking, particularly in HDL-C levels, highlighting the potential reversibility of smoking-related dyslipidemia (Kawachi et al., 2019).

The present investigation was designed to find out the link between cigarette smoking and serum lipid and lipid ratio parameters in a Jordanian population. In order to give a thorough evaluation of the atherogenic effects of smoking, this investigation examined both traditional lipid indicators and important lipid ratios. The findings of this investigation are anticipated to add to the expanding corpus of research on metabolic disorders brought on by smoking and to guide clinical and public health initiatives to reduce the cardiovascular risks related to tobacco consumption, particularly among Jordanians. The purpose of the study was to thoroughly investigate any potential relationships between cigarette smoking and the concentrations of serum lipid and lipid ratio profile in a sample of adult males from Jordan.

Methods and Materials

Study Setting and Design

This analytical cross-sectional research was executed from December 2024 to July 2025 at the Akadya Medical Center's outpatient internal medicine clinic in Amman, Jordan. The main goal was to assess how smoking cigarettes affected adult males' blood lipid levels and lipid ratio measures. All procedures, including participant recruitment, anthropometric measurements, blood collection, and biochemical analysis, were standardized to ensure methodological accuracy and reproducibility.

Participants

A total of 475 adult male volunteers were recruited consecutively during the study period. Participation was voluntary, and the smokers had been smoking regularly for at least one year. and individuals were enrolled after confirming eligibility based on predefined criteria. Participants were split up into two groups (325 smokers and 150 non-smokers).

The Inclusion and Exclusion Requirements

Subjects could take part if they were adult males between 18 and 60 years of age and were either current smokers with a history of daily smoking for at least one year, or never-smokers in the case of the control group. Exclusion criteria included the use of pharmacological treatments for diabetes mellitus, dyslipidemia, or other metabolic disorders; history of alcohol dependence; or cessation of smoking within the previous year. Additionally, participants with a history of hypertension, CVD (personal or familial), renal or hepatic disorders, or endocrine abnormalities, were excluded. These criteria were carefully applied to minimize confounding variables that could independently affect lipid metabolism or cardiovascular risk.

Anthropometric Measurements

Anthropometric data were collected. A calibrated digital scale was used for determining weight to the closest 0.5 g, and a stadiometer had been utilized for determining height to the closest 0.5 cm. The body mass index (BMI) was calculated by dividing the body's weight in kilograms by the square of the height in meters. Classification of BMI was performed according to WHO guidelines Yoon et al., (2015), with values between 18 and 25 kg/m^2 considered normal.

Blood Sample Collection

Blood collection was performed between 8:00 and 10:00 a.m. after a minimum 12-hour overnight fast to reduce dietary influences on lipid measurements. In sterile vacutainer tubes, around 5 mL of venous blood was extracted from the antecubital veins without anticoagulant. After 15 minutes of clotting at room temperature, the specimens were centrifuged for ten minutes at 4000 × g. The separated serum was analyzed immediately to prevent degradation of lipid components. Any hemolyzed or improperly handled samples were discarded and recollected to ensure data integrity.

Biochemical Analysis

All biochemical tests were carried out at the Akadya Medical Laboratory, a licensed diagnostic facility that complies with global quality standards. TC, TG, HDL-C, and LDL-C serum levels were measured with Roche Diagnostics GmbH's Cobas 501 auto-analyzer in Mannheim, Germany. Reagents, calibrators, and controls supplied by Roche Diagnostics were used to ensure accuracy and consistency of results. Internal quality control checks were performed daily, and external quality assurance protocols were strictly followed throughout the study.

From the measured lipid values, key atherogenic lipid ratios were calculated, including the TC/HDL, TG/HDL, and LDL/HDL ratios. These derived parameters were employed to offer a more thorough evaluation of the balance between atherogenic and protective lipoproteins.

Reference Ranges

For interpretation of lipid and glucose levels, standard clinical reference ranges were applied. The normal range for TC was 50–200 mg/dL, TG 50–200 mg/dL, LDL-C 60–160 mg/dL, and HDL-C 10–60 mg/dL.

Statistical Analysis

Version 26 of the SPSS IBM Statistics software (IBM Corp., Armonk, NY, USA) was utilized to analyze the data. Mean values and standard deviations (Moradinazar et al.) were used for representing continuous parameters, while proportions and frequencies were used to describe categories of variables. Data normality was evaluated using the Shapiro-Wilk test. Considering comparisons between groups, independent samples t-tests were applied when comparing two groups, while several group comparisons were conducted using a one-way ANOVA and post-hoc analysis. To analyze categorical data, the chi-square test was employed. The Pearson's correlation coefficient (r) was computed to investigate the connection between the number of cigarettes smoked daily and lipid parameters, including lipid ratios. To further evaluate the independent associations of lipid parameters and lipid ratios with smoking status, a test of multiple linear regression was performed. Smoking status was treated as the dependent variable, while serum lipid values and lipid ratios were entered as independent predictors. P-values, confidence intervals, and adjusted beta coefficients were presented. P-values below 0.05 were regarded as statistically relevant.

Ethical Considerations

Akadya Medical Center's Institutional Ethics Committee examined and authorized the research procedure. Prior to enrolment, all individuals provided informed and written permission, and study participants' personal information was kept anonymous. Subjects were guaranteed to withdraw from participation at any moment and without repercussions. The Declaration of Helsinki's (2013 edition) ethical guidelines were followed throughout the entire process.

Findings and Results

Smokers had a significantly greater mean age (39.90 ± 8.12 years) compared to non-smokers (36.36 ± 7.95 years), with a substantial effect size (Cohen's d = 0.79) and a highly significant difference ($p < 0.001$). Age distribution showed that only 16.6% of smokers were aged ≤30 years, compared to 31.3% among non-smokers, while a higher proportion of smokers (25.8%) were aged >45 years in contrast to controls (16%). Similarly, the mean BMI was greater for cigarettes users (26.18 ± 1.65 kg/m^2) than non-smokers (25.02 ± 1.43 kg/m^2), with a very large effect size (Cohen's d = 1.15) and a statistically significant difference ($p < 0.001$). Regarding BMI categories, overweight status was more prevalent among smokers (82.8%) compared to non-smokers (66.7%), whereas normal weight was more common among non-smokers (33.3%) than smokers (16.3%), and obesity was observed only among smokers (0.9%) (Table 1).

Table 1

Comparison of Age and BMI Among Groups

Variable	Parameter	Smokers N=325	Non-smokers N=150	Test	d	p-value
Age (years)	Mean ± SD	39.90 ± 8.12	36.36 ± 7.95	19.70	0.79	<0.001
	Min-max	23-61	22-55			
	≤30	54 (16.6%)	47 (31.3%)			
	>30-45	187 (57.5%)	79 (52.7%)			
	>45	84 (25.8%)	24 (16%)			
BMI (kg/m²)	Mean ± SD	26.18 ± 1.65	25.02 ± 1.43	54.37	1.15	<0.001
	Min-max	21-30	21-28			
	Normal weight	53 (16.3%)	50 (33.3%)			
	Overweight	269 (82.8%)	100 (66.7%)			
	Obese	3 (0.9%)	0 (0%)			

BMI = Body Mass Index; d: Cohen's d; SD = Standard Deviation

For continuous parameters, the t-test was employed, and for categorical factors, the chi-square test. The effect magnitude was estimated using Cohen's d.

Cigarette Stick Quantities Among Groups

The quantity of cigarettes smoked each day was significantly higher among smokers (16.28 ± 7.51 sticks; range: 5–36) compared to non-smokers, who reported no cigarette consumption (0.00 ± 0.00 sticks; range: 0–0). When categorized, 23.7% of smokers consumed fewer than 10 sticks per day, 48.3% consumed between 10 and 20 sticks, and 28% consumed more than 20 sticks daily. This difference had a substantial effect size (Cohen's d = 16.28) and proved highly significant ($p < 0.001$) (Table2).

Table 2

Comparison of Number of Cigarette Sticks Between Smokers and Non-Smokers

Variable	Parameter	Smokers N=325	Non-smokers N=150	Test	d	p-value
Number of sticks/day	Mean ± SD	16.28 ± 7.51	0.00 ± 0.00	703.41	16.28	<0.001
	Min-max	5-36	0-0			
	• <10	77 (23.7%)	0 (0%)			
	• 10-20	157 (48.3%)	0 (0%)			
	• >20	91 (28%)	0 (0%)			

SD = Standard Deviation, d: Cohen's d. For continuous parameters, the t-test was employed. The effect magnitude was estimated using Cohen's d.

Comparison of Differences in Lipid Profiles Among Groups

Smokers demonstrated significantly higher levels of TC (216.67 ± 12.01 mg/dL vs. 178.11 ± 12.58 mg/dL; Cohen's d = 38.56; $p < 0.001$), TG (202.13 ± 19.31 mg/dL vs. 170.39 ± 14.36 mg/dL; Cohen's d = 31.74; $p < 0.001$), and LDL-C (142.82 ± 11.04 mg/dL vs. 103.48 ± 11.54 mg/dL; Cohen's d = 39.33; $p < 0.001$) compared to non-smokers. Conversely, HDL-C was significantly lower in smokers (34.42 ± 3.42 mg/dL) than in non-smokers

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(42.11 ± 2.27 mg/dL; Cohen's d = −7.68; p < 0.001). Furthermore, atherogenic ratios were markedly elevated among smokers, including the TC/HDL ratio (6.38 ± 0.93 vs. 4.23 ± 0.32; Cohen's d = 2.14; p < 0.001), triglyceride/HDL ratio (5.97 ± 1.10 vs. 4.06 ± 0.41; Cohen's d = 1.91; p < 0.001), and LDL/HDL ratio (4.21 ± 0.69 vs. 2.46 ± 0.29; Cohen's d = 1.75; p < 0.001) (Table 3).

Table 3

Comparison of Differences in Lipid Profiles Among Groups

Variable	Parameter	Smokers N=325	Non-smokers N=150	Test	D	p-value
TC (mg/dL)	Mean ± SD	216.67 ± 12.01	178.11 ± 12.58	1025.84	38.56	<0.001
TG (mg/dL)	Mean ± SD	202.13 ± 19.31	170.39 ± 14.36	322.69	31.74	<0.001
LDL (mg/dL)	Mean ± SD	142.82 ± 11.04	103.48 ± 11.54	1265.18	39.33	<0.001
HDL (mg/dL)	Mean ± SD	34.42 ± 3.42	42.11 ± 2.27	626.61	-7.68	<0.001
TC/HDL ratio	Mean ± SD	6.38 ± 0.93	4.23 ± 0.32	757.72	2.14	<0.001
TG/HDL ratio	Mean ± SD	5.97 ± 1.10	4.06 ± 0.41	424.86	1.91	<0.001
LDL/HDL ratio	Mean ± SD	4.21 ± 0.69	2.46 ± 0.29	870.04	1.75	<0.001

d: Cohen's d; TC stands for total cholesterol, HDL for high-density lipoprotein, LDL for low-density lipoprotein, and TG for triglycerides. SD is for standard deviation. Independent samples t-test was applied for all comparisons, and the effect magnitude was estimated using Cohen's d.

Comparison of lipid parameters across different smoking intensities

The comparison of lipid parameters across different smoking intensities revealed a clear dose-dependent deterioration in lipid profiles with increasing daily cigarette consumption. Total cholesterol rose progressively from 203.17 ± 9.18 mg/dL in individuals smoking fewer than 10 sticks per day to 216.28 ± 6.66 mg/dL in those smoking 10–20 sticks, and further to 228.77 ± 8.21 mg/dL among heavy smokers (>20 sticks/day), with the difference being highly significant (p < 0.001). A similar trend was observed for TG, which increased from 184.61 ± 13.68 mg/dL in light smokers to 197.49 ± 10.11 mg/dL in moderate smokers and reached 224.97 ± 13.43 mg/dL in heavy smokers (p < 0.001). LDL-C followed the same pattern, increasing from 130.14 ± 8.92 mg/dL to 143.23 ± 6.35 mg/dL and 152.84 ± 7.92 mg/dL across the three groups (p < 0.001). Conversely, HDL-C decreased markedly with smoking intensity, dropping from 38.83 ± 2.14 mg/dL in light smokers to 34.18 ± 1.77 mg/dL in moderate smokers and further to 31.10 ± 2.24 mg/dL in heavy smokers (p < 0.001). Moreover, atherogenic lipid ratios showed a significant stepwise worsening: TC/HDL increased from 5.24 ± 0.37 to 6.34 ± 0.43 and 7.40 ± 0.70, TG/HDL rose from 4.77 ± 0.49 to 5.79 ± 0.47 and 7.29 ± 0.85, while LDL/HDL escalated from 3.36 ± 0.30 to 4.20 ± 0.35 and 4.95 ± 0.54 (all p < 0.001) (Table 4).

Table 4

Comparison of lipid parameters across different smoking intensities

Variable	Parameter	<10 sticks/day N=77	10-20 sticks/day N=157	>20 sticks/day N=91	Test	p-value
TC (mg/dL)	Mean ± SD	203.17 ± 9.18	216.28 ± 6.66	228.77 ± 8.21	227.20	<0.001
TG (mg/dL)	Mean ± SD	184.61 ± 13.68	197.49 ± 10.11	224.97 ± 13.43	258.47	<0.001
LDL (mg/dL)	Mean ± SD	130.14 ± 8.92	143.23 ± 6.35	152.84 ± 7.92	192.50	<0.001
HDL (mg/dL)	Mean ± SD	38.83 ± 2.14	34.18 ± 1.77	31.10 ± 2.24	312.24	<0.001
TC/HDL ratio	Mean ± SD	5.24 ± 0.37	6.34 ± 0.43	7.40 ± 0.70	367.71	<0.001
TG/HDL ratio	Mean ± SD	4.77 ± 0.49	5.79 ± 0.47	7.29 ± 0.85	370.24	<0.001
LDL/HDL ratio	Mean ± SD	3.36 ± 0.30	4.20 ± 0.35	4.95 ± 0.54	317.51	<0.001

TC stands for total cholesterol, HDL for high-density lipoprotein, LDL for low-density lipoprotein, and TG for triglycerides. SD is for standard deviation. Independent samples t-test was applied for all comparisons

Smoking Status as a Predictor for Lipid Profile Abnormalities

HDL-C exhibited a strong negative association with smoking status (B = −0.114, β = −1.165, t = −12.428, p < 0.001, 95% CI: −0.133 to −0.096), indicating that lower HDL-C levels were linked to being a smoker. Conversely, LDL-C showed a significant positive association (B = 0.036, β = 1.681, t = 5.980, p < 0.001, 95% CI: 0.024 to 0.048), as did TG (B = 0.011, β = 0.570, t = 2.738, p = 0.006, 95% CI: 0.003 to 0.020). The LDL/HDL ratio also had a significant negative association with smoking status (B = −0.673, β = −1.465, t = −3.130, p = 0.002, 95% CI: −1.095 to −0.251). The TG/HDL ratio showed a borderline non-significant trend (B = −0.278, p = 0.065), while TC was not a significant predictor (B = −0.004, p = 0.133) (Table 5).

Table 5

Multiple Linear Regression Analysis for Predictors of Smoking Status

Model		Non-standard Coefficients		Standardized Coefficients	Test	Significance value	95.0% Confidence Interval for B	
		B	Std. Error	Beta			Low	Upper
1	(Constant)	2.741	0.401		6.827	0.000	1.952	3.530
	TC	-0.004	0.003	-0.191	-1.504	0.133	-0.009	0.001
	TG	0.011	0.004	0.570	2.738	0.006	0.003	0.020
	LDL	0.036	0.006	1.681	5.980	0.000	0.024	0.048
	HDL	-0.114	0.009	-1.165	-12.428	0.000	-0.133	-0.096
	TG ratio	-0.278	0.150	-0.773	-1.849	0.065	-0.573	0.018
	LDL ratio	-0.673	0.215	-1.465	-3.130	0.002	-1.095	-0.251

TC stands for total cholesterol, HDL for high-density lipoprotein, LDL for low-density lipoprotein, and TG for triglycerides.
Multiple linear regression analysis was performed. The dependent variable was smoking status.

Correlation of Lipid and Lipid Ratio Profile Parameters with Age, BMI, and Number of Cigarette Sticks

Correlation analysis demonstrated that the amount of cigarette sticks smoked per day was strongly and positively correlated with TC (r = 0.797, p < 0.001), TG (r = 0.836, p < 0.001), LDL-C (r = 0.761, p < 0.001), TC/HDL ratio (r = 0.909, p < 0.001), TG/HDL ratio (r = 0.920, p < 0.001), and LDL/HDL ratio (r = 0.884, p < 0.001), while it was strongly and negatively correlated with HDL-C (r = −0.861, p < 0.001). BMI showed weak but significant negative correlations with LDL (r = −0.112, p = 0.044), TC/HDL ratio (r = −0.168, p = 0.002), TG/HDL ratio (r = −0.156, p = 0.005), and LDL/HDL ratio (r = −0.165, p = 0.003), but a positive association with HDL-C (r = 0.168, p = 0.002). Age, however, did not show significant correlations with any lipid parameters (all p > 0.05) (Table 6).

Table 6

Correlation of Lipid and Lipid Ratio Parameters with Age, BMI, and Number of Cigarette Sticks Among Smokers

Variable	Parameter	Age	BMI	No of stick
TC	Correlation value	0.018	-0.107	0.797**
	Significance	0.749	0.053	0.000
TG	Correlation value	-0.016	-0.098	0.836**
	Significance	0.778	0.079	0.000
LDL	Correlation value	-0.001	-0.112*	0.761**
	Significance	0.988	0.044	0.000
HDL-C	Correlation value	0.025	0.168**	-0.861**
	Significance	0.659	0.002	0.000
TC/HDL ratio	Correlation value	0.005	-0.168**	0.909**
	Significance	0.934	0.002	0.000
TG/HDL ratio	Correlation value	-0.003	-0.156**	0.920**

	Significance	0.958	0.005	0.000
LDL/HDL ratio	Correlation value	0.000	-0.165**	0.884**
	Significance	0.994	0.003	0.000

TC stands for total cholesterol, HDL for high-density lipoprotein, LDL for low-density lipoprotein, and TG for triglycerides. Pearson's correlation coefficient (r) was used. Significance levels: p < 0.01, p < 0.05 (2-tailed).

Discussion and Conclusion

Globally, cigarette smoking continues to be among the top modifiable risk factors for fatalities and morbidity (Fu et al., 2024). Smoking promotes thickening and narrowing of blood vessels, elevates heart rate and blood pressure, and increases the likelihood of blood clot formation, all of which significantly contribute to cardiovascular risk (Gaggini et al., 2022).

The current study provides further evidence that cigarette smoking has a profound negative implication on lipids metabolic rate. Smokers showed noticeably greater values of TC, TG, and LDL-C. On the other hand, smokers had significantly lower levels of HDL-C, which is renowned for its protective function in cardiovascular health. Furthermore, smokers exhibited noticeably greater amounts of atherogenic lipid ratios (TG/HDL-C ratio, LDL/HDL ratio, and the TC/HDL ratio), suggesting a higher risk of cardiovascular problems and an increased atherogenic lipid composition. These results are in line with earlier research done on a variety of ethnicities. In Portugal, for instance, Sousa et al., (2024) showed substantial variations in cholesterol measurements, with smokers having greater quantities of TC, LDL-C, and TG and non-smokers having increased rates of HDL-C (Moradinazar et al., 2020; Sousa et al., 2024). Similar patterns were noted by Mouhamed et al., (2013) in a Tunisian sample, who found that smokers had reduced HDL-C scores and elevated TC, LDL-C, and TG values. Similarly, smokers in Japan had lower HDL-C levels and increased LDL-C, TC, and TG amounts, according to Nakamura et al., (2021), indicating that smoking's negative effects on lipid profiles are universal across geographic and ethnic groups. Furthermore, in an Iranian sample, Moradinazar et al., (2020) found that smokers had greater TG and decreased HDL-C levels, but no discernible changes in LDL-C and TC . In a Persian community, Momayyezi et al., (2024) discovered that smokers had increased TG levels and a higher frequency of dyslipidaemia; however, they also found that smokers exhibited higher HDL-C amounts and that there were no appreciable changes in LDL-C and TC between cigarette users and non-users (Momayyezi et al., 2024). The complex relationship between smoking and lipid metabolism is highlighted by these variances between research, which could be caused by variances in dietary practices, analytical techniques, and genetic susceptibility.

Many processes are involved in the mechanistic changes in lipid metabolism caused by cigarette smoking. Excessive catecholamine release from nicotine's stimulation of the sympathetic-adrenal system promotes lipolysis and raises plasma levels of free fatty acids (Mouhamed et al.). Hypertriglyceridemia is a result of the liver receiving these higher FFAs, which stimulate the production of more hepatic TG and very low-density lipoproteins (VLDL-C) Mithun et al., (2019). Additionally, smoking lowers HDL-C levels via lowering oestrogen amounts. By reducing lipoprotein lipase production, hyperinsulinemia—which is frequently seen in smokers—exacerbates lipid abnormalities and raises TC, LDL-C, VLDL-C, and TG values. Furthermore, as opposed to non-smokers, smokers frequently eat diets higher in fat and cholesterol but lower in fibre Howard et al., (2000), which could exacerbate these lipid changes even more.

Additionally, the current investigation identified a substantial dose-response association between changes in lipid markers and daily cigarette consumption. The comparison of lipid parameters across different smoking intensities revealed a clear dose-dependent deterioration in lipid profiles with increasing daily cigarette consumption. Increased cigarette consumption was strongly and positively correlated with TC, TG, LDL-C, and lipid ratios, while showing a strong negative correlation with HDL-C. These results mirror findings from earlier studies. Mithun et al., (2019) reported that smokers averaged approximately 11.6 ± 4.5 cigarettes per day and demonstrated similar lipid abnormalities. Additionally, in Rastogi et al., (1989) reported that smokers who smoked over ten cigarettes a day had far decreased HDL-C amounts than smokers who smoked fewer cigarettes Rastogi et al., (1989), proving a direct

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dose-responsive link between lipid dysregulation and smoking severity.

The current results also support scientific proof that smoking cigarettes damages important enzymes that regulate metabolism and lipid circulation. Reduced lecithin-cholesterol acyl-transferase (LCAT) production, changes in cholesterol ester transfer protein (CETP), and hepatic lipase production are some of the factors contributing to HDL-C decline, which all impair HDL-C activity and metabolism. Neki, (2002) similarly demonstrated that HDL-C levels were significantly lower among smokers, findings that parallel those observed in the present study. The low level of HDL-C has important clinical implications, as low HDL-C is strongly associated with the development of atherosclerosis and increased cardiovascular risk. Moreover, smokers in the current investigation also experienced significantly higher serum TC, LDL-C, and LDL-C levels, consistent with the results of Mithun et al., (2019), who reported significant increases in these lipid fractions among smokers. Oxidative stress plays a pivotal role in these processes. Smoking accelerates oxidative modification of LDL particles, generating potent pro-atherogenic mediators that promote endothelial dysfunction and plaque formation (Khurana et al., 2021; Khurana et al., 2000).

The regression model in the current study further substantiated these associations, showing that being a smoker was independently linked to lower HDL-C levels and higher LDL-C and TG levels. The LDL/HDL ratio also exhibited a significant positive association with smoking, indicating a higher atherogenic risk, while the TG/HDL ratio showed a borderline but non-significant trend. These results support earlier research by Al-Jaf & Al-Jaf, (2020), who found that smokers had a considerably greater incidence of aberrant TG and LDL-C values than non-smokers, and Jain & Ducatman, (2018), who showed that smokers were far more likely to have low HDL-C and aberrant TG. These results have been supported by meta-analyses and systematic reviews, which consistently demonstrate that smoking cigarettes lowers TG and HDL-C values (Al-Jaf & Al-Jaf, 2020; Mouhamed et al., 2013; Rashan et al., 2016; Taiwo & Thanni, 2021).

Additionally, there is a proof that smoking-induced negative lipid changes may continue even after quitting. While studies such as Attard et al., (2017) have demonstrated that quitting smoking can reduce TG levels and improve overall lipid profiles, a meta-analysis by van der Plas et al., (2023) showed that some detrimental effects on lipid metabolism may continue post-cessation. This emphasizes how crucial early detection and preventative measures are to lowering long-term cardiovascular risks. Because smokers generally consume greater amounts of fat and less grain and fibre than non-smokers, diet may potentially be a confounding factor Rastogi et al., (1989), which might explain why the two groups' lipid profiles were found to vary.

Numerous experimental and human research further demonstrate the detrimental effects of smoking on HDL-C levels. According to Jain & Ducatman, (2018), current smokers had 1.6 times the adjusted odds of developing aberrant HDL-C when contrasted with non-smokers. Aryanpur et al., (2018) highlighted the widespread effects of tobacco use on lipid metabolism by showing in a meta-analysis that passive as well as active smoking significantly decreased HDL-C amounts. It has also been demonstrated that increased homocysteine levels, which are frequently detected in smokers, negatively impact HDL-C (Shih et al., 2023). Additionally, smokers' decreased oestrogen levels worsen HDL-C inhibition, increasing the possibility of atherogenesis (Nagasawa et al., 2012).

Cigarette smoking significantly contributes to the development of an cardiovascular-promoting lipid composition, which is characterized by excessive TC, TG, LDL-C, and lipid ratios, as well as decreased HDL-C, according to the combined data from the current study and earlier studies. Such lipid imbalances lead to plaque formation, dysfunction of endothelial cells, and eventually higher cardiac morbidity and death. Comprehending such processes highlights the urgent necessity of vigorous public health initiatives meant to curb tobacco use and encourage quitting smoking. Such initiatives could significantly lessen the impact of smoking-related dyslipidaemia and its cardiovascular effects when paired with lifestyle changes like better eating habits and more exercise.

This study's comparatively large sample size, which improves the findings' statistical power and dependability, is one of its main advantages. The detailed biochemical analysis performed using standardized, high-precision laboratory techniques ensures the accuracy and consistency of lipid and lipid-ratio measurements. Furthermore, detection of daily cigarette consumption allowed for the assessment of dose-

dependent effects, providing a more comprehensive understanding of smoking's impact on lipid metabolism.

However, there are several restrictions on the study. Because of its cross-sectional layout, it is unable to prove a link between smoking and changes in lipid levels throughout a period of time. Additionally, lifestyle characteristics including stress levels, physical exercise, and eating habits, were unmanaged, which may have influenced lipid levels and could confound the observed associations.

Implement smoking cessation programs as a key strategy to improve lipid profiles and reduce cardiovascular risk among smokers. Incorporate routine lipid profile screening for individuals who smoke, especially heavy smokers, to allow early detection and intervention. Perform longitudinal cohort studies to prove the causal associations between smoking intensity, duration, and progressive alterations in lipid parameters. Evaluate the combined effect of smoking with other cardiovascular risk factors, such as obesity, hypertension, and diabetes, on lipid profiles and lipid ratios.

This study demonstrates that cigarette smoking significantly worsens lipid metabolism and increases cardiovascular risk. Smokers showed elevated TC, TG, and LDL-C, along with markedly lower HDL-C levels and more atherogenic lipid ratios compared to non-smokers. The comparison of lipid parameters across different smoking intensities revealed a clear dose-dependent deterioration in lipid profiles with increasing daily cigarette consumption. The number of cigarettes smoked daily showed strong positive correlations with adverse lipid changes, while regression analysis identified low HDL-C and high LDL and TG levels as key smoking-induced side effects. Such outcomes highlight the harmful, dose-dependent smoking's impact on lipid parameters and reinforce the importance of smoking cessation and early cardiovascular risk management among this population.

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Declaration of Interest

The authors of this article declared no conflict of interest.

Ethical Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants. Ethical considerations in this study were that participation was entirely optional.

Transparency of Data

In accordance with the principles of transparency and open research, we declare that all data and materials used in this study are available upon request.

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Authors' Contributions

All authors equally contribute to this study.

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