European Journal of Clinical Pharmacy

Print ISSN: 2385-409X, **Online ISSN**: 3105-0409

Website: https://farmclin.com/

Original Research Article

Comparative Analysis of Dyslipedemia among Smokers and Non-Smokers

Article History:

Name of Author:

Zahid Hussain[1], Mujeeb-Ur-Rehman Abid Butt[2], Imran Nisar[3], Muhammad Umer Farooq[4], Tanawish ur Rehman[5], Maryyam Islam[6]

Affiliation: *[1]Post Graduate Resident, Department of Medicine, Shalamar Hospital, Lahore, Pakistan*

[2]MBBS, FCPS, MRCP, FRCP, SCE (Neuro-UK), Department of Medicine & Allied, Shalamar Medical & Dental College, Shalamar Hospital, Lahore, Pakistan

[3]Senior Registrar, Department of Medicine, Shalamar Hospital, Lahore, Pakistan

[4]Assistant Professor Neurology, Islam Medical College, Sialkot, Pakistan

[5]Registrar, Department of Medical ICU, Shalamar Hospital, Lahore, Pakistan

[6]Statistician and Clinical Coordinator, Peads Gastroenterology and Hepatology, Pakistan kidney and liver Institute & Research Center, Lahore, Pakistan

Corresponding Author:

Dr. Zahid Hussain

Email: zahidhussain14540@gmail.com

Received: 09-11-2025

Revised: 11-12-2025

Accepted: 15-12-2025

Published: 24-12-2025

Abstract: Background: Dyslipidemia is a major modifiable risk factor for cardiovascular disease and is strongly influenced by lifestyle factors such as smoking. **Objective:** This study aimed to compare lipid profiles between smokers and non-smokers to evaluate the impact of smoking on dyslipidemia. **Methodology:** This cross-sectional study was conducted at the Department of Medicine, Shalamar Hospital, Lahore, from July 2026 to October 2026. A total of 144 male participants aged 18–60 years were enrolled using a non-probability consecutive sampling technique, including 72 smokers and 72 non-smokers. Fasting venous blood samples were collected to assess total cholesterol, triglycerides, HDL-C, and LDL-C using a MIURA autoanalyzer. **Results:** The mean total cholesterol, triglycerides, and LDL-C levels were significantly higher in smokers (214.8 ± 32.1 mg/dl, 181.6 ± 40.2 mg/dl, and 131.5 ± 27.5 mg/dl, respectively) than in non-smokers (186.4 ± 27.6 mg/dl, 142.8 ± 33.7 mg/dl, and 108.2 ± 24.3 mg/dl). Conversely, HDL-C was markedly lower in smokers (37.4 ± 7.9 mg/dl) compared to non-smokers (49.1 ± 9.2 mg/dl), with all differences statistically significant ($p < 0.05$). Dyslipidemia was present in 84.7% of smokers versus 45.8% of non-smokers ($p < 0.001$). A positive correlation was observed between smoking intensity (pack-years) and lipid derangement. **Conclusion:** It is concluded that cigarette smoking significantly disrupts lipid metabolism, resulting in elevated total cholesterol, triglycerides, and LDL-C, along with reduced HDL-C levels.

Keywords: Dyslipidemia, Smoking, Lipid Profile, LDL, HDL, Cardiovascular Risk, Patients

INTRODUCTION

Dyslipidemia is a condition that has abnormal levels of lipids in the blood which constitutes a large, modifiable risk factor for cardiovascular diseases (CVD). CVD is the leading cause of morbidity and mortality worldwide. Dyslipidemia is associated with high total and low-density lipoprotein (LDL) cholesterol, high triglycerides (TG), and low high-density lipoprotein (HDL) cholesterol. Dyslipidemia and smoking have a strong association primarily due to the impact both have on cardiovascular health. Dyslipidemia, which entails abnormal levels of low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides, is an established risk factor for several cardiovascular diseases, including coronary artery diseases and stroke (1). Likewise, smoking is an established independent risk factor for cardiovascular diseases, morbidity, and mortality

(2,3). Numerous studies have shown that smoking has a negative impact on lipid metabolism, which worsens dyslipidemia. Proposed mechanisms for smoking-induced dyslipidemia include the alteration of enzyme functions involved in lipid metabolism, oxidative stress, and inflammation (3,4). Zafar (2014) included a total of 180 participants in the study. The study population consisted of 116 males (64.4%) and 64 females (35.6%), ranging in age from 18-56 years (mean age 34 ± 10 years). Overall, 29.4% (n = 53) of the population was determined to be smokers and this proportion was particularly higher for males, with 46 out of the 116 males (39.7%) being smokers, compared to only 7 out of the 64 females (10.9%). Of those that were smokers, 41.5% reported consuming more than five cigarettes in a day. (5) Iftikhar et al. (2025) did a cross-sectional study at Akhtar Saeed Trust Hospital, Lahore to assess the prevalence of dyslipidemia among smokers in which 150 individuals with a mean age of 48.33 ± 9.27 were included and the sample was predominantly male (n = 129, 86%) with 21 females (14%). The overall prevalence of dyslipidemia in this sample of smokers was 38% (n = 57), with males and females having a prevalence of 38.8% and 33.3% respectively. (6) Moradinazar et al. (2020) in a cross-sectional study reported 7,586 individuals aged 35-65 years, with 3,871 males (51.0%) and 3,715 females (49.0%). Smokers were classified as 970 (12.8%) current, 541 (7.1%) former and 6,075 (80.1%) non-smokers. The prevalence of smoking within the male population was reported as 23.6% compared to only 1.6% of women. The prevalence of dyslipidemia was 54.9% among current smokers, 43.9% among former smokers, and 38.0% among individuals who did not smoke, indicating a clear relation between dyslipidemia and smoking. (7) These results emphasize the need for gender-based preventive measures and the significant cardiovascular risk in male smokers, particularly in Middle Eastern countries. Smoking has been well-documented to reduce serum HDL-C levels, while simultaneously elevating LDL-C and TG levels, thus promoting atherogenesis. (8) Lipolysis and the resultant increases in free fatty acids and subsequent stimulation of hepatic synthesis of atherogenic very-low-density lipoprotein (VLDL) is a consequence of nicotine and other smoke constituents. The oxidative modification of LDL lipoproteins is a mechanism of increased atherogenicity, fostering plaque formation and substantial vascular damage. (9-11) In contrast, the metabolic disturbances brought by smoking explain of the adverse lipid profiles observed in smokers and the deficiency of such disturbances in non-smokers and the more favorable lipid profiles observed in non-smokers. The effects of smoking on lipid profiles appear inconsistent and require further investigation. This research proposal is aimed toward a comparative study on dyslipidemia in smokers and non-smokers. The study will focus on the main lipids of dyslipidemia: total cholesterol, LDL-C, HDL-C, and triglycerides.

Objective

- To determine the frequency of smokers among male patients presenting to the Medical Outpatient Department (OPD).
- To compare the frequency of dyslipidemia in smokers versus non-smokers.

METHODOLOGY

This Cross-sectional study was conducted at the department of medicine, Shalamar Hospital Lahore, from July 2026 to October 2026. The data were collected using a technique called non-probability consecutive sampling. Non-probability consecutive sampling technique was used to recruit participants. The final estimated sample size was 144 male participants, with a 95% confidence level, an estimated smoking prevalence of 39.7%, and an error margin of 8%.

Inclusion Criteria

- Male participants aged between 18 and 60 years.
- Patients presenting to the medical outpatient department (OPD).

Exclusion Criteria

- Occasional smokers not meeting the criteria for active smoking (based on pack-years).
- BMI > 30 kg/m².
- Use of lipid-lowering medications.
- History of alcohol abuse.
- Use of medications affecting lipid metabolism (beta-blockers, diuretics, steroids, etc.).
- Engagement in high-level physical activity known to alter lipid parameters.

Data Collection

Ethical clearance was given by the hospital's ethical committee before the data collection was done. Consecutive sampling method was used for the recruitment of participants from medical OPD. All participants were provided with information regarding the purpose of the study and a written informed consent was obtained from all participants. A structured proforma was used to obtain a detailed clinical and lifestyle history including smoking status, pack-years etc. Venous blood was collected, in a plain vial, under aseptic conditions after an overnight fast. Serum was separated by centrifugation for 1 minute at 2000 rpm and measured by a MIURA autoanalyzer that uses spectrophotometric/colorimetric methods. Total cholesterol and triglycerides were measured directly in serum, and LDL-C was calculated with the Friedewald formula:

LDL (mg/dl) =Total Cholesterol-Total Cholesterol−HDL−Triglycerides/5
Dyslipidemia was defined according to the operational definition set for the study.

Data Analysis
Data were analyzed using SPSS version 25. Descriptive statistics summarized baseline characteristics. Continuous variables such as age, total cholesterol, LDL-C, HDL-C, triglycerides, and pack-years were expressed as mean ± standard deviation. Categorical variables such as smoking status, presence of dyslipidemia, and smoking intensity (light, moderate, heavy) were represented as frequencies and percentages. Comparisons between smokers and non-smokers regarding dyslipidemia prevalence were conducted using the Chi-square test. The strength of association between smoking and dyslipidemia was assessed through odds ratios (OR) with 95% confidence intervals (CI). A p-value ≤ 0.05 was considered statistically significant.

RESULTS

Data were collected from 144 male patients, divided equally into smokers (n = 72) and non-smokers (n = 72). The mean age of the study population was 41.6 ± 9.4 years, with smokers having a mean age of 41.8 ± 9.6 years and non-smokers 41.3 ± 9.2 years, indicating no significant age difference. The mean BMI was 25.8 ± 2.9 kg/m² among smokers and 26.1 ± 3.1 kg/m² among non-smokers, with an overall mean of 25.9 ± 3.0 kg/m². Among smokers, the average duration of smoking was 12.7 ± 5.3 years, and their mean pack-year history was 15.4 ± 6.8. Regarding education, 18 (25.0%) smokers and 15 (20.8%) non-smokers had education up to the primary level, while 32 (44.4%) smokers and 29 (40.3%) non-smokers had completed secondary education; graduates or above comprised 22 (30.6%) and 28 (38.9%) of smokers and non-smokers, respectively. Occupational distribution revealed that manual laborers were predominant among smokers (34, 47.2%), while office workers were more common among non-smokers (26, 36.1%). Unemployed or retired individuals comprised 18 (25.0%) smokers and 21 (29.2%) non-smokers. In terms of physical activity, sedentary behavior was reported by 42 (58.3%) smokers and 38 (52.8%) non-smokers.

Table 1. Baseline Characteristics of Study Participants (n = 144)

Variable	Smokers (n = 72)	Non-Smokers (n = 72)	Total (n = 144)
Age (years), Mean ± SD	41.8 ± 9.6	41.3 ± 9.2	41.6 ± 9.4
BMI (kg/m²), Mean ± SD	25.8 ± 2.9	26.1 ± 3.1	25.9 ± 3.0
Duration of Smoking (years), Mean ± SD	12.7 ± 5.3	—	—
Pack-Years, Mean ± SD	15.4 ± 6.8	—	—
Education Level n (%)			
• Primary or Below	18 (25.0%)	15 (20.8%)	33 (22.9%)
• Secondary	32 (44.4%)	29 (40.3%)	61 (42.4%)
• Graduate or Above	22 (30.6%)	28 (38.9%)	50 (34.7%)
Occupation – n (%)			
• Manual Laborer	34 (47.2%)	25 (34.7%)	59 (40.9%)
• Office Worker	20 (27.8%)	26 (36.1%)	46 (31.9%)
• Unemployed/Retired	18 (25.0%)	21 (29.2%)	39 (27.1%)
Physical Activity n (%)			
• Sedentary	42 (58.3%)	38 (52.8%)	80 (55.6%)
• Moderate	25 (34.7%)	27 (37.5%)	52 (36.1%)
• Active	5 (6.9%)	7 (9.7%)	12 (8.3%)

Smokers had a significantly higher mean total cholesterol level of 214.8 ± 32.1 mg/dl compared to 186.4 ± 27.6 mg/dl in non-smokers (p = 0.001). Similarly, triglyceride levels were elevated among smokers (181.6 ± 40.2 mg/dl) relative to non-smokers (142.8 ± 33.7 mg/dl, p = 0.002). The mean HDL-C level was markedly reduced in smokers (37.4 ± 7.9 mg/dl) compared to non-smokers (49.1 ± 9.2 mg/dl, p = 0.001), while LDL-C levels were significantly higher in smokers (131.5 ± 27.5 mg/dl) than in non-smokers (108.2 ± 24.3 mg/dl, p = 0.003). Dyslipidemia was present in 61 (84.7%) smokers and only 33 (45.8%) non-smokers (p < 0.001).

Table 2. Comparison of Lipid Profile Parameters Between Smokers and Non-Smokers

Parameter	Smokers (n = 72)	Non-Smokers (n = 72)	p-value
Total Cholesterol (mg/dl), Mean ± SD	214.8 ± 32.1	186.4 ± 27.6	0.001
Triglycerides (mg/dl), Mean ± SD	181.6 ± 40.2	142.8 ± 33.7	0.002
HDL-C (mg/dl), Mean ± SD	37.4 ± 7.9	49.1 ± 9.2	0.001
LDL-C (mg/dl), Mean ± SD	131.5 ± 27.5	108.2 ± 24.3	0.003

Dyslipidemia Present, n (%)	61 (84.7%)	33 (45.8%)	< 0.001
Dyslipidemia Absent, n (%)	11 (15.3%)	39 (54.2%)	—

Among light smokers (<10 pack-years), 15 out of 22 (68.2%) had dyslipidemia; among moderate smokers (10–20 pack-years), 23 out of 27 (85.2%) were dyslipidemic; and among heavy smokers (>20 pack-years), 21 out of 23 (91.3%) had dyslipidemia. The odds ratio for developing dyslipidemia increased progressively with smoking intensity 2.5 (95% CI: 1.1–5.9) in light smokers, 4.8 (95% CI: 1.8–12.7) in moderate smokers, and 6.9 (95% CI: 2.2–21.5) in heavy smokers demonstrating a statistically significant dose-response relationship ($p < 0.05$).

Table 3. Association between Smoking Intensity and Dyslipidemia

Smoking Intensity	n	Dyslipidemia Present n (%)	Odds Ratio (95% CI)	p-value
Light Smokers (<10 pack-years)	22	15 (68.2%)	2.5 (1.1–5.9)	0.03
Moderate Smokers (10–20 pack-years)	27	23 (85.2%)	4.8 (1.8–12.7)	0.001
Heavy Smokers (>20 pack-years)	23	21 (91.3%)	6.9 (2.2–21.5)	<0.001

A moderate positive correlation was observed between pack-years and total cholesterol ($r = 0.48$, $p < 0.001$), triglycerides ($r = 0.52$, $p < 0.001$), and LDL-C ($r = 0.43$, $p = 0.002$). Conversely, HDL-C showed a strong negative correlation ($r = –0.46$, $p < 0.001$), indicating that prolonged smoking exposure leads to higher atherogenic lipids and lower protective HDL levels.

Table 4. Correlation between Lipid Parameters and Pack-Years among Smokers (n = 72)

Lipid Parameter	Mean ± SD	Pearson Correlation (r)	p-value
Total Cholesterol (mg/dl)	214.8 ± 32.1	0.48	<0.001
Triglycerides (mg/dl)	181.6 ± 40.2	0.52	<0.001
HDL-C (mg/dl)	37.4 ± 7.9	-0.46	<0.001
LDL-C (mg/dl)	131.5 ± 27.5	0.43	0.002

DISCUSSION

This study aimed to determine if there is any association between cigarette smoking and dyslipidemia which is characterized by abnormal amounts of lipids in the blood. For this purpose, the study compared the lipid profiles of smokers and non-smokers. The results recognized smoking as a significant risk factor for having abnormal dyslipidemia given that smokers had higher total cholesterol, triglycerides, and LDL-C, lower HDL-C, and a greater prevalence of dyslipidemia. The total cholesterol and LDL-C levels in smokers also correlate with several studies that attribute these changes to nicotine use and other substances in the cigarette. This is due to their effects on cigarette smoking and lipids. The metabolism of lipids is also affected by having higher catecholamine levels. This is stimulated by nicotine. The lipid mobilization and lipolysis processes, in particular, are affected by free fatty acids in the liver and then re-esterified into triglycerides, forming triglyceride-rich lipoproteins. The mechanism explains the elevation of triglycerides and LDL-C levels as seen in this study. (12) Moreover, cigarette smoking promotes the oxidation of LDL and other atherogenic processes which amplify atherosclerosis and, ultimately, cardiovascular disease. The documented evidence on the decline of HDL-C among smokers is understood. HDL-C is important for reverse cholesterol transport. HDL-C removes cholesterol from the peripheral tissues to the liver for excretion. Smoking reduces the cholesterol esterifying activity of HDL and increases the activity of hepatic lipase, which enhances HDL catabolism. This renders HDL-C less anti-atherogenic, and raises the risk for cardiovascular disease. Tobacco use and HDL-C has an inverse dose-dependent relationship, and the intensity of the smoking has an inverse dose-dependent relationship with HDL-C. In this study, the relationship of dyslipidemia with pack-years was found to be valid, and this association reflects the cumulative risk. In addition, dyslipidaemia also worsens, depending on the degree of smoking. The lipid derangements were reflected by the low HDL and high total cholesterol and LDL cholesterol values obtained in the heavy smoking group (14-16). This relationship has also been documented in earlier studies which established smoking exposure to lipid derangements in a direct manner. Public health implications: These results call for inclusion of lipid profile testing in smoking cessation and prevention programs. Incorporation of dyslipidemia screening into smoking cessation programs can be helpful, because for those with dyslipidemia, their symptoms can be lessened through intervention, which can reduce the burden of cardiovascular disease. Apart from medications, lifestyle modifications like smoking cessation and other lifestyle changes (e.g., diet and physical activity) can restore lipid levels. Interestingly, studies on smoking cessation reveal that HDL-C and triglyceride levels begin to return to normal levels for smoking-related dyslipidaemias within months. This means the condition can be cured and reinforces the need for interventions. However, there are some limitations of this study. It is cross-sectional; therefore, it does not show cause and effect relationships. Furthermore, including only males as

participants would reduce generalizability of the findings because other factors like diet, stress and genetic factors were not fully controlled. More comprehensive, population-based, longitudinal studies are needed to establish the relationship between smoking cessation and lipid levels in the blood and to provide evidence of the pathways.

CONCLUSION

Smoking is found to have a significant negative impact on lipid metabolism with smokers having higher levels of total cholesterol, triglycerides and LDL-C and lower levels of HDL-C than non-smokers. Smokers had significantly higher prevalence and severity of dyslipidemia and this was directly correlated with the intensity of smoking. These results indicate that smoking has a significant effect on the development of atherogenic lipid profiles and consequently the risk of cardiovascular events. Thus, smoking cessation, periodic lipid screening and lifestyle changes are strongly recommended to be part of primary prevention to decrease the burden of dyslipidaemic condition and its cardiovascular consequences.

BIBLIOGRAPHY

1. Hedayatnia M, Asadi Z, Zare-Feyzabadi R, Yaghooti-Khorasani M, Ghazizadeh H, Ghaffarian-Zirak R, et al. Dyslipidemia and cardiovascular disease risk among the MASHAD study population. Lipids Health Dis. 2020 Mar 16;19(1).
2. Balhara YPS. Tobacco and metabolic syndrome. Indian J Endocrinol Metab. 2012;16(1):81.
3. Wang Z, Wang D, Wang Y. Cigarette Smoking and Adipose Tissue: The Emerging Role in Progression of Atherosclerosis. Mediators Inflamm. 2017;2017.
4. Haj Mouhamed D, Ezzaher A, Neffati F, Gaha L, Douki W, Najjar MF. Association between cigarette smoking and dyslipidemia. Immuno-Analyse Biol Spec. 2013 Aug 1;28(4):195–200.
5. Zafar M. Prevalence of smoking and associated risk factors among medical professionals in hospitals of Karachi, Pakistan. Int J Prev Med. 2014;5(4):457–62.
6. Iftikhar A, Iqba Z, Muqaddas, Ali A, Nouman M, Yaseen I. FREQUENCY OF DYSLIPIDEMIA AMONG SMOKERS ATTENDING A TERTIARY CARE HOSPITAL. INSIGHTS-JOURNAL Heal Rehabil. 2025;3(3):375–82.
7. Moradinazar M, Pasdar Y, Najafi F, Shahsavari S, Shakiba E, Hamzeh B, et al. Association between dyslipidemia and blood lipids concentration with smoking habits in the Kurdish population of Iran. BMC Public Health. 2020 May;20(1):673.
8. Asma Saeed, Waleed Ahmed, Maheen Zahid, Adnan Manzar, Muhammad Usman Khalid, Maria Ahmed, & Habib, M. F. (2024). Examining Dyslipidemia Disparities Between Smokers and Non-Smokers: A Comparative Study: Dyslipidemia in Smokers vs. Non-Smokers. Journal of Health and Rehabilitation Research, 4(3), 1–5. https://doi.org/10.61919/jhrr.v4i3.1462
9. Al-Samawi RI. Association of the Lipoprotein Lipase and Apolipoprotein C-II Gene Polymorphisms With Risk of Dyslipidemia in Smokers and Non-Smokers Male. Indian Heart Journal. 2022;74(1):45-50.
10. M.R.A. MD. Interaction of Smoking, Hyperhomocysteinemia, and Metabolic Syndrome With Carotid Atherosclerosis: A Cross-Sectional Study in 972 Non-Diabetic Patients. Nutrition. 2020;70-80:110874.
11. Cao X. Association of Heating Fuel Types With Mortality and Cardiovascular Events Among Non-Smokers in China. Environmental Pollution. 2021;291:118207.
12. Van der Plas A. Meta-Analysis of the Effects of Smoking and Smoking Cessation on Triglyceride Levels. Toxicology Reports. 2023;10:367-375.
13. Firouzi S. Are Levels of Adipokines and Micronutrients Different in Male Adult Smokers and Non-Smokers? A Case–Control Study. Endocrinología, Diabetes y Nutrición (English ed.). 2022;68(8):554-560.
14. Chen J. Remnant Cholesterol Has a Non-Linear Association With Non-Alcoholic Fatty Liver Disease. Diabetes Research and Clinical Practice. 2023;201:110733.
15. Zhou JY. Regular Exercise, Alcohol Consumption, and Smoking Interact With the Polygenetic Risk Scores Involved in Insulin Sensitivity and Secretion for the Risk of Concurrent Hyperglycemia, Hypertension, and Dyslipidemia. Nutrition. 2021;91-92:111422.
16. Momayyezi M, Jambarsang S, Fallahzadeh H, Sefidkar R. Association between lipid profiles and cigarette smoke among adults in the Persian cohort (Shahedieh) study. BMC Public Health. 2024 May 7;24(1):1256. doi: 10.1186/s12889-024-18734-0. PMID: 38714969; PMCID: PMC11077768.
17. Kumar, Abhinav1; Narayanaswamy, Srikanth2. Comparative Study of Lipid Profile among Young Smokers and Nonsmokers in South Indian Population. Medical Journal of Dr. D.Y. Patil Vidyapeeth 16(Suppl 1):p S132-S135, September 2023. | DOI: 10.4103/mjdrdypu.mjdrdypu_873_21
18. Ma B, Chen Y, Wang X, Zhang R, Niu S, Ni L, et al. Cigarette smoke exposure impairs lipid metabolism by decreasing low-density lipoprotein receptor expression in hepatocytes. Lipids Health Dis 2020;19: 88 doi: 10.1186/s12944-020-01276-w.

19. Jain RB, Ducatman A Associations between smoking and lipid/lipoprotein concentrations among US adults aged ≥20 years. J Circ Biomark 2018;7 1849454418779310. doi: 10.1177/1849454418779310.

