Journal of Health and Rehabilitation Research (2791-156X)
Volume 4, Issue 3
Double Blind Peer Reviewed.
https://jhrlmc.com/
DOI: https://doi.org/10.61919/jhrr.v4i3.1462
www.lmi.education/

Examining Dyslipidemia Disparities Between Smokers and Non-Smokers: A Comparative Study

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Keywords
Dyslipidemia, smoking, lipid profiles, cardiovascular risk, total cholesterol, LDL-C, HDL-C, triglycerides, smoking cessation, public health.

Disclaimers

Authors' Contributions	All authors contributed to the study design, data collection, analysis, and manuscript preparation.
Conflict of Interest	None declared
Data/supplements	Available on request.
Funding	None
Ethical Approval	Respective Ethical Review Board
Study Registration	N/A
Acknowledgments	N/A

ABSTRACT

Background: Dyslipidemia, characterized by elevated total cholesterol, LDL-C, triglycerides, and low HDL-C, is a significant risk factor for cardiovascular disease. Smoking exacerbates these lipid abnormalities, increasing cardiovascular risk, yet the disparities in lipid profiles between smokers and non-smokers are not well defined.

Objective: To compare the lipid profiles of smokers and non-smokers and evaluate the extent of dyslipidemia associated with smoking.

Methods: A cross-sectional study was conducted with 500 participants (250 smokers, 250 non-smokers) from an urban hospital. Fasting serum samples were collected and analyzed for total cholesterol, LDL-C, HDL-C, and triglycerides. Smoking status was self-reported and validated by cotinine levels. Statistical analysis included ANOVA and multiple regression to assess lipid differences between groups, adjusting for confounders.

Results: Smokers had significantly higher total cholesterol (213.4 ± 42.1 mg/dL vs. 197.2 ± 38.7 mg/dL, $p < 0.001$) and LDL-C (142.7 ± 35.4 mg/dL vs. 126.3 ± 31.6 mg/dL, $p < 0.001$) and lower HDL-C (44.3 ± 11.2 mg/dL vs. 53.7 ± 12.9 mg/dL, $p < 0.001$) compared to non-smokers.

Conclusion: Smoking significantly worsens dyslipidemia, highlighting the need for targeted smoking cessation interventions to reduce cardiovascular risk

INTRODUCTION

Dyslipidemia, characterized by abnormalities in lipid levels such as elevated total cholesterol, low-density lipoprotein cholesterol (LDL-C), triglycerides, and reduced high-density lipoprotein cholesterol (HDL-C), poses a significant threat to cardiovascular health globally. These lipid irregularities are strongly linked to the pathogenesis of atherosclerotic cardiovascular diseases (ASCVD), including coronary artery disease, stroke, and peripheral artery disease, and contribute substantially to morbidity and mortality worldwide. The pathophysiology of dyslipidemia involves complex interactions between genetic predispositions, dietary habits, physical inactivity, and other modifiable risk factors, highlighting the need for targeted prevention strategies aimed at reducing the burden of ASCVD (1). Despite extensive research on dyslipidemia and its association with cardiovascular risk, the impact of lifestyle factors such as smoking on lipid profiles remains a critical area requiring further exploration.

Smoking is one of the most potent reversible risk factors for cardiovascular diseases and is widely recognized for its detrimental effects on overall health, including its influence on lipid metabolism. The toxic constituents of cigarette smoke, such as nicotine and carbon monoxide, induce oxidative stress and inflammatory responses, leading to lipid abnormalities that are characterized by increased levels of LDL-C and triglycerides and decreased levels of HDL-C. These alterations contribute to the development of atherogenic dyslipidemia, which exacerbates the risk of cardiovascular events in smokers compared to non-smokers. The effects of smoking on lipid metabolism are mediated through multiple pathways, including the oxidative modification of lipoproteins, dysregulation of lipid-regulating enzymes such as lipoprotein lipase and hepatic lipase, and inflammatory processes that promote the synthesis of atherogenic lipoproteins (2).

Comparative analyses of lipid profiles between smokers and non-smokers provide valuable insights into the extent of lipid dysfunction attributable to smoking and underscore the heightened cardiovascular risk among smokers. Understanding these differences is essential for developing targeted interventions, such as smoking cessation programs, that not only address nicotine addiction but also mitigate the adverse effects of smoking on lipid profiles. Moreover, such comparisons can inform clinical guidelines and public health policies by providing objective data on the impact of smoking on cardiovascular risk and the potential benefits of smoking cessation in improving lipid profiles (3). Although existing studies have highlighted the association between smoking and dyslipidemia, the strength and consistency of these associations vary across different populations and methodologies, underscoring the need for further research to elucidate these relationships (4).

This study aims to compare the lipid profiles of smokers and non-smokers to determine the extent of dyslipidemia associated with smoking and to identify the specific lipid alterations that arise from smoking. By examining the

concentrations of key lipid components such as total cholesterol, LDL-C, HDL-C, and triglycerides, this research seeks to establish whether smoking itself is an independent risk factor for dyslipidemia or whether the observed associations are influenced by other factors, such as age, sex, or comorbidities. Such an approach not only enhances our understanding of the link between smoking and lipid metabolism but also provides evidence-based recommendations for the management of dyslipidemia in smokers (5). The hypothesis driving this study is that smokers are more likely to exhibit a dyslipidemic profile compared to non-smokers, characterized by elevated LDL-C and triglycerides and reduced HDL-C levels. This hypothesis aligns with existing literature suggesting that the toxic components of cigarette smoke adversely affect lipid regulation through oxidative stress, inflammation, and enzyme dysregulation (6).

Ultimately, the purpose of this research is to advance knowledge on the interplay between smoking and dyslipidemia, thereby informing clinical practices and public health strategies aimed at reducing cardiovascular risk through smoking cessation and lipid management. The findings from this study are expected to provide concrete evidence supporting the implementation of smoking cessation as a critical component of dyslipidemia and cardiovascular disease prevention efforts. By elucidating the specific lipid alterations associated with smoking, this study aims to contribute to the development of targeted therapeutic interventions that can effectively reduce the cardiovascular burden in smokers (7).

MATERIAL AND METHODS

The study employed a cross-sectional comparative design to evaluate the lipid profiles between smokers and non-smokers, aiming to determine the extent of dyslipidemia associated with smoking. Data were collected from January to June 2024 at a large urban teaching hospital with a diverse, multiethnic patient population. This setting was chosen to enhance the generalizability of the findings and to provide a standardized environment for the collection of clinically relevant data, including blood samples for lipid analysis. Participants were recruited through voluntary response and purposive sampling methods, using posters and flyers distributed across various hospital departments, particularly the cardiology and pulmonology clinics. Additionally, attending physicians were encouraged to invite eligible patients to participate in the study.

Eligible participants included adults aged 18 to 65 years, with an equal distribution of male and female participants, who were classified into two groups: smokers and non-smokers. Smokers were defined as individuals who had consumed at least one cigarette per day for the past six months or more. Ex-smokers, defined as those who had quit smoking for at least five years, and non-smokers, defined as individuals who had never smoked in their lifetime, were also included to provide a comprehensive comparison. Exclusion criteria included individuals with secondary dyslipidemias due to conditions such as hypothyroidism, nephrotic syndrome, or chronic kidney disease, those on lipid-lowering medications or hormone replacement therapy, and those with a history of major cardiac events within the past year to eliminate acute conditions that could influence lipid parameters (8).

Participants provided written informed consent after being fully briefed on the study's objectives, procedures, and their rights, including the right to withdraw at any time. Ethical approval for the study was obtained from the hospital's ethics committee, and the study was conducted in accordance with the principles outlined in the Declaration of Helsinki. Initial clinical interviews were conducted, followed by the administration of a standardized questionnaire that gathered information on smoking habits, including the number of cigarettes smoked daily, years of smoking, and any prior smoking cessation attempts. Non-smokers were queried about passive smoking exposure to account for it as a potential confounding factor. Additional data on potential confounders such as dietary habits, physical activity levels, alcohol consumption, medical history, medication use, and family history of cardiovascular disease were also collected.

Lipid profiles were assessed through blood samples collected after a 12-hour fast to minimize variations in lipid levels due to dietary intake. Serum samples were analyzed in the hospital’s enzymatic laboratory using standardized methods. Total cholesterol, LDL-C, and triglycerides were measured quantitatively by enzymatic assays, while HDL-C levels were determined using the method described by Warnick et al., involving dextran sulfate magnesium chloride. LDL-C was calculated using the Friedewald equation for triglyceride levels below 400 mg/dL. All laboratory procedures included quality control measures, such as the use of control samples and calibration of instruments, to ensure the accuracy and reliability of the lipid measurements (9).

Data analysis was performed using SPSS version 25.0. Descriptive statistics, including means and standard deviations for continuous variables and frequencies and percentages for categorical variables, were calculated. Normality of the data was assessed using the Shapiro-Wilk test and histograms. Comparative analyses between smokers and non-smokers were conducted using independent samples t-tests for normally distributed continuous variables, while the Mann-Whitney U test was used for non-normally distributed variables. Chi-square tests were used for categorical data. Multiple logistic regression analysis was performed to adjust for potential confounders, including age, gender, BMI, physical activity, dietary patterns, alcohol use, and family history of cardiovascular disease, with smoking status as the primary predictor variable. Interaction terms were included in the regression models to assess whether the effect of smoking on lipid profiles was moderated by factors such as gender or age. Statistical significance was set at $p<0.05$ for all analyses, and results were reported with 95% confidence intervals where applicable (10).

The study design and methodologies were carefully selected to minimize biases and ensure robust comparisons between the lipid profiles of smokers and non-

smokers. The comprehensive approach, including standardized participant selection, data acquisition methods, and rigorous statistical analyses, aimed to provide valuable insights into the impact of smoking on dyslipidemia and contribute to the broader understanding of modifiable cardiovascular risk factors associated with smoking.

RESULTS

The study included 500 participants, with 250 smokers and 250 non-smokers, matched on demographic variables such as age and gender to ensure comparability between groups. The mean age of smokers was 45 years (SD = 10.1), while the mean age of non-smokers was 44 years (SD = 9.8), with no significant difference between the two groups (p = 0.612). Gender distribution was also similar, with the smoker group comprising 150 males and 100 females, and the non-smoker group comprising 145 males and 105 females (p = 0.713).

The mean BMI for smokers was 26.7 kg/m^2 (SD = 4.5), and for non-smokers, it was 26.5 kg/m^2 (SD = 4.3), showing no significant difference (p = 0.485). These demographic similarities ensured that age, gender, and BMI were not confounding factors in the analysis of lipid profiles.

Table 1: Demographic and Baseline Characteristics of Study Participants

Parameter	Smokers (Mean ± SD)	Non-Smokers (Mean ± SD)	p-value
Age (years)	45 ± 10.1	44 ± 9.8	0.612
BMI (kg/m²)	26.7 ± 4.5	26.5 ± 4.3	0.485
Gender (Males/Females)	150/100	145/105	0.713

The primary outcome measures were the lipid profiles of smokers and non-smokers, which included total cholesterol, LDL-C, HDL-C, and triglycerides. Smokers exhibited significantly higher levels of total cholesterol and LDL-C compared to non-smokers (p < 0.001). Specifically, the mean total cholesterol level among smokers was 213.4 mg/dL (SD = 42.1), whereas non-smokers had a mean total cholesterol level of 197.2 mg/dL (SD = 38.7). LDL-C levels were also elevated in smokers at 142.7 mg/dL (SD = 35.4) compared to 126.3 mg/dL (SD = 31.6) in non-smokers. Conversely, HDL-C levels were significantly lower in smokers, averaging 44.3 mg/dL (SD = 11.2) compared to 53.7 mg/dL (SD = 12.9) in non-smokers (p < 0.001). Triglyceride levels were also higher in smokers, with a mean of 178.5 mg/dL (SD = 59.3), compared to 148.9 mg/dL (SD = 52.4) in non-smokers (p < 0.001).

Table 2: Lipid Profile Comparison Between Smokers and Non-Smokers

Lipid Parameter	Smokers (Mean ± SD)	Non-Smokers (Mean ± SD)	p-value
Total Cholesterol (mg/dL)	213.4 ± 42.1	197.2 ± 38.7	<0.001
LDL-C (mg/dL)	142.7 ± 35.4	126.3 ± 31.6	<0.001
HDL-C (mg/dL)	44.3 ± 11.2	53.7 ± 12.9	<0.001
Triglycerides (mg/dL)	178.5 ± 59.3	148.9 ± 52.4	<0.001

Further analysis was conducted to assess the impact of smoking intensity on lipid profiles among smokers. Heavy smokers, defined as those consuming more than 20 cigarettes per day, exhibited higher total cholesterol and LDL-C levels compared to light smokers, who consumed 20 cigarettes or fewer per day. Specifically, heavy smokers had a mean total cholesterol level of 225.6 mg/dL (SD = 43.7) compared to 202.3 mg/dL (SD = 39.1) in light smokers (p < 0.001). Similarly, LDL-C levels were 153.9 mg/dL (SD = 36.2) in heavy smokers, versus 131.6 mg/dL (SD = 32.8) in light smokers. HDL-C levels were lower in heavy smokers at 39.2 mg/dL (SD = 10.5), compared to 47.1 mg/dL (SD = 11.6) in light smokers (p < 0.001). Triglycerides were also higher among heavy smokers, averaging 189.7 mg/dL (SD = 61.2) compared to 164.8 mg/dL (SD = 57.1) in light smokers.

Table 3: Lipid Profile Comparison Between Heavy Smokers and Light Smokers

Parameter	Heavy Smokers (>20 cigarettes/day) (Mean ± SD)	Light Smokers (≤20 cigarettes/day) (Mean ± SD)	p-value
Total Cholesterol (mg/dL)	225.6 ± 43.7	202.3 ± 39.1	<0.001
LDL-C (mg/dL)	153.9 ± 36.2	131.6 ± 32.8	<0.001
HDL-C (mg/dL)	39.2 ± 10.5	47.1 ± 11.6	<0.001
Triglycerides (mg/dL)	189.7 ± 61.2	164.8 ± 57.1	<0.001

Additionally, the duration of smoking was found to have a significant impact on lipid profiles. Participants who had been smoking for more than 10 years showed higher levels of total cholesterol and LDL-C compared to those who had smoked for 10 years or less. Specifically, those with a smoking duration of more than 10 years had a mean total cholesterol level of 228.3 mg/dL (SD = 45.1) and LDL-C levels of 155.4 mg/dL (SD = 37.2). In contrast, participants who had smoked for 10 years or less had mean total cholesterol levels of 199.5 mg/dL (SD = 38.6) and LDL-C levels of 130.8 mg/dL (SD = 32.5). HDL-C levels were lower in the longer-duration smokers at 38.7 mg/dL (SD = 10.3) compared to 46.5 mg/dL (SD = 11.8) in the shorter-duration group (p < 0.001). Triglycerides were similarly higher in the

Table 4: Lipid Profile Comparison Based on Smoking Duration

Parameter	Smoking Duration >10 years (Mean ± SD)	Smoking Duration ≤10 years (Mean ± SD)	p-value
Total Cholesterol (mg/dL)	228.3 ± 45.1	199.5 ± 38.6	<0.001
LDL-C (mg/dL)	155.4 ± 37.2	130.8 ± 32.5	<0.001
HDL-C (mg/dL)	38.7 ± 10.3	46.5 ± 11.8	<0.001
Triglycerides (mg/dL)	195.2 ± 62.7	161.9 ± 56.9	<0.001

longer-duration smokers, averaging 195.2 mg/dL (SD = 62.7) compared to 161.9 mg/dL (SD = 56.9). These findings clearly indicate that smoking, particularly at higher intensities and longer durations, is associated with adverse alterations in lipid profiles, thereby increasing cardiovascular risk. The results underscore the importance of smoking cessation as a primary intervention for improving lipid profiles and reducing the risk of cardiovascular diseases associated with dyslipidaemia.

DISCUSSION

The findings of this study demonstrated a clear association between smoking and dyslipidemia, with smokers exhibiting significantly higher levels of total cholesterol, LDL-C, and triglycerides, and lower levels of HDL-C compared to non-smokers. These results are consistent with previous research that has established smoking as a major risk factor for adverse lipid alterations, which in turn increase the risk of cardiovascular diseases. The study supported the hypothesis that smoking intensifies dyslipidemic patterns, contributing to atherogenic profiles that elevate the risk of coronary artery disease, stroke, and other cardiovascular events (1). The observed lipid abnormalities in smokers can be attributed to the toxic effects of cigarette smoke, which include oxidative stress, inflammation, and direct interference with lipid metabolism, as previously documented in the literature (2).

The subgroup analyses further revealed that the intensity and duration of smoking had a dose-dependent effect on lipid profiles, with heavy smokers and those with a longer smoking history showing worse dyslipidemic patterns. This dose-response relationship aligns with findings from other studies that have reported similar trends, suggesting that the adverse impact of smoking on lipid metabolism escalates with the number of cigarettes smoked and the duration of smoking (3). The higher levels of LDL-C and triglycerides and the reduced HDL-C levels observed in heavy and long-term smokers underscore the need for targeted smoking cessation interventions, particularly for individuals with prolonged and intensive smoking habits. Previous studies have also highlighted that smoking cessation can lead to significant improvements in lipid profiles, thereby reducing cardiovascular risk, further emphasizing the critical role of smoking cessation in cardiovascular disease prevention (4).

The strengths of this study included its robust cross-sectional design and the use of standardized lipid measurements, which allowed for a reliable comparison of lipid profiles between smokers and non-smokers. The inclusion of a diverse, multiethnic population from an urban teaching hospital enhanced the generalizability of the findings. Additionally, the study controlled for several potential confounders, including age, gender, BMI, physical activity, dietary habits, and alcohol consumption, through the use of multiple logistic regression analyses, thereby strengthening the validity of the observed associations between smoking and dyslipidemia. However, the study also had limitations. As a cross-sectional study, it could not establish causality, and the reliance on self-reported smoking status, despite the use of cotinine validation, introduced the possibility of reporting bias. The exclusion of individuals with certain secondary dyslipidemias or recent major cardiac events may have further limited the generalizability of the results to all populations (5).

The study's findings are in agreement with the broader body of evidence linking smoking to adverse lipid profiles. The mechanisms underlying these associations include the oxidative modification of lipoproteins, inhibition of lipoprotein lipase activity, and increased hepatic lipase activity, all of which contribute to the dysregulation of lipid metabolism in smokers. Inflammatory processes associated with smoking also play a critical role, as increased levels of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha have been shown to alter lipid synthesis and metabolism, further exacerbating dyslipidemia (6). These mechanisms highlight the complex interplay between smoking, inflammation, and lipid metabolism, which collectively contribute to the elevated cardiovascular risk observed in smokers.

Given the strong association between smoking and dyslipidemia, public health interventions should prioritize smoking cessation as a key strategy for cardiovascular disease prevention. Campaigns aimed at educating smokers about the harmful effects of smoking on lipid profiles and cardiovascular health could be instrumental in promoting smoking cessation. Healthcare providers should routinely assess smoking status and provide counseling and support for smoking cessation, particularly for individuals at high risk of dyslipidemia and cardiovascular diseases. The integration of smoking cessation interventions into broader cardiovascular risk reduction programs could enhance their effectiveness, as suggested by the significant improvements in lipid profiles observed in individuals who quit smoking (7).

Future research should aim to address the limitations of this study by employing longitudinal designs to better establish causal relationships between smoking and dyslipidemia. Additionally, studies should explore the molecular and genetic factors that mediate the effects of smoking on lipid metabolism, which could provide insights into targeted therapies for smokers who are unable or unwilling to quit. Investigating the impact of smoking cessation on lipid

profiles and cardiovascular outcomes over time could further reinforce the benefits of quitting smoking and inform public health strategies aimed at reducing the burden of cardiovascular diseases associated with smoking (8). Furthermore, considering the role of potential confounders such as diet, physical activity, and genetic predispositions in future studies would provide a more comprehensive understanding of the interactions between smoking and lipid metabolism.

CONCLUSION

In conclusion, this study confirmed that smoking is significantly associated with adverse lipid profiles, which contribute to an increased risk of cardiovascular diseases. The findings underscore the critical need for effective smoking cessation interventions as part of comprehensive dyslipidemia management and cardiovascular risk reduction strategies. Public health policies should continue to promote smoking cessation and provide resources to support individuals in quitting smoking, thereby improving lipid profiles and reducing the incidence of smoking-related cardiovascular diseases.

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