International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/

Original Researcher Article *DOI: 10.61336/im/26-07-98*

A Study on Lipid Profile and Fibrinogen Levels in Smokers and Non-Smokers.

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Received: 26-03-2026
Revised: 12-05-2026
Accepted: 09-07-2026
Published: 16-07-2026

Abstract

Background: Cigarette smoking is a well-established independent risk factor for cardiovascular disease (CVD), contributing to both atherogenic lipid alterations and prothrombotic states. This study aimed to evaluate and compare the lipid profile parameters and plasma fibrinogen levels between smokers and non-smokers. ***Methods:*** A cross-sectional study was conducted involving 100 healthy male participants (50 smokers and 50 age-matched non-smokers) aged 25–55 years. Fasting blood samples were collected for the estimation of total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and plasma fibrinogen levels. Data were analyzed using independent t-tests, with $p < 0.05$ considered statistically significant. ***Results:*** Smokers demonstrated significantly higher plasma fibrinogen levels compared to non-smokers (348.6 ± 68.4 mg/dL vs. 275.3 ± 55.2 mg/dL, $p < 0.001$). The lipid profile of smokers revealed a significantly atherogenic pattern: higher TC (208.4 ± 38.2 mg/dL vs. 182.6 ± 31.5 mg/dL, $p < 0.001$), higher TG (158.7 ± 52.3 mg/dL vs. 124.5 ± 44.8 mg/dL, $p < 0.001$), higher LDL-C (132.5 ± 32.8 mg/dL vs. 108.3 ± 26.4 mg/dL, $p < 0.001$), and significantly lower HDL-C (37.2 ± 7.6 mg/dL vs. 46.8 ± 9.4 mg/dL, $p < 0.001$) compared to non-smokers. ***Conclusion:*** Smoking is associated with significant elevations in plasma fibrinogen and an atherogenic lipid profile characterized by increased total cholesterol, triglycerides, LDL-C, and decreased HDL-C. These findings underscore the dual prothrombotic and proatherogenic impact of smoking, highlighting the importance of smoking cessation in reducing cardiovascular risk.

Keywords: Smoking, lipid profile, fibrinogen, cardiovascular disease, dyslipidemia, thrombosis.

INTRODUCTION

Cardiovascular disease (CVD) remains the leading cause of mortality worldwide, accounting for approximately 17.9 million deaths annually. Among the multitude of modifiable risk factors contributing to this global burden, cigarette smoking stands as one of the most pervasive and preventable causes. The relationship between tobacco smoking and cardiovascular morbidity has been extensively documented, with epidemiological evidence consistently demonstrating that smokers face a two- to four-fold increased risk of developing coronary heart disease, stroke, and peripheral arterial disease compared to non-smokers.

The pathophysiological mechanisms through which cigarette smoking promotes cardiovascular disease are multifaceted, encompassing endothelial dysfunction, oxidative stress, inflammation, platelet activation, and alterations in lipid and coagulation profiles. Cigarette smoke contains thousands of chemical compounds, including reactive oxygen species and free radicals, which induce systemic inflammation and oxidative damage to vascular endothelium. This endothelial injury initiates a cascade of proatherogenic and prothrombotic events that predispose individuals to atherosclerotic plaque formation and acute thrombotic complications.

Lipid abnormalities represent a cornerstone of smoking-related cardiovascular risk. A substantial body of evidence indicates that cigarette smoking exerts a deleterious effect on serum lipid and lipoprotein metabolism. A meta-analysis of 54 studies demonstrated that smokers have approximately 3% higher serum total cholesterol and 9% higher serum triglyceride concentrations compared to non-smokers. Furthermore, smoking is consistently associated with reduced levels of high-density lipoprotein cholesterol (HDL-C), the so-called "good cholesterol" responsible for reverse cholesterol transport and atheroprotection. This reduction in HDL-C is accompanied by elevated levels of low-density lipoprotein cholesterol (LDL-C) and very-low-density lipoprotein (VLDL) cholesterol, creating an atherogenic lipid profile

characterized by an elevated LDL-C/HDL-C ratio. More recent meta-analyses have confirmed these findings, showing that smokers have decreased levels of apolipoproteins AI and AII (consistent with reduced HDL-C) and increased apolipoprotein B levels, further confirming the negative impact of smoking on lipid metabolism.

In addition to lipid abnormalities, cigarette smoking profoundly affects hemostatic and fibrinolytic pathways. Fibrinogen, a key glycoprotein synthesized in the liver, plays an essential role in the coagulation cascade as the precursor to fibrin, the structural component of blood clots. Elevated plasma fibrinogen levels have been recognized as an independent risk factor for cardiovascular disease, with long-term increases of 1 g/L associated with an approximate doubling of major cardiovascular disease outcomes. Fibrinogen also functions as an acute-phase reactant, increasing in response to inflammatory stimuli. Importantly, smoking is a potent determinant of plasma fibrinogen levels. Studies have demonstrated that fibrinogen levels increase with the number of cigarettes smoked and decline rapidly following smoking cessation. Large-scale meta-analyses involving over 80,000 participants have confirmed the strong association between smoking and elevated fibrinogen concentrations.

The interplay between smoking, dyslipidemia, and hyperfibrinogenemia creates a synergistic proatherogenic and prothrombotic milieu that substantially amplifies cardiovascular risk. Smokers exhibit higher levels of inflammatory markers such as high-sensitivity C-reactive protein (hs-CRP), interleukin-6, and fibrinogen, alongside increased thrombotic markers. These alterations are dose-dependent, with smoking intensity and pack-years showing clear relationships with biomarker elevations. Moreover, smoking has been shown to exacerbate the effects of elevated total cholesterol and reduced HDL-C on coronary heart disease risk.

Despite the well-established associations between smoking and individual cardiovascular risk factors, comprehensive evaluations of both lipid profile and fibrinogen levels in the same population remain valuable for understanding the integrated impact of smoking on cardiovascular risk. This study was therefore undertaken to evaluate and compare the lipid profile parameters—including total cholesterol, triglycerides, HDL-C, and LDL-C—and plasma fibrinogen levels between smokers and non-smokers, and to assess the potential synergistic contribution of these alterations to cardiovascular risk.

MATERIALS AND METHODS

This cross-sectional study was conducted at the Department of Biochemistry of a tertiary care teaching hospital over a period of 12 months. The study protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrollment. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Study Population

A total of 100 healthy male participants were enrolled in the study, comprising 50 smokers and 50 age-matched non-smokers. Participants were recruited from the outpatient department and through community-based health awareness campaigns. The sample size was calculated based on previous studies, with an expected difference in fibrinogen levels of 50 mg/dL between groups, a standard deviation of 60 mg/dL, 80% power, and a 5% level of significance.

Inclusion and Exclusion Criteria

Inclusion criteria for smokers: Male individuals aged 25–55 years who had been smoking at least 10 cigarettes per day for a minimum of 5 years. Participants were required to be in generally good health with no known acute or chronic illnesses.

Inclusion criteria for non-smokers: Male individuals aged 25–55 years who had never smoked or had ceased smoking for at least 10 years, with no history of active or passive smoking exposure.

Exclusion criteria (both groups): Participants with a history of cardiovascular disease, diabetes mellitus, hypertension, chronic liver disease, renal disease, thyroid disorders, or any acute infection or inflammatory condition were excluded. Individuals on lipid-lowering therapy, anticoagulant therapy, or any medication known to affect lipid or coagulation parameters were also excluded. Pregnant women, individuals with a body mass index (BMI) > 30 kg/m², and those with a history of alcohol abuse were not included in the study.

Data Collection

A detailed medical history was obtained from each participant using a structured questionnaire. Demographic data including age, gender, occupation, and socioeconomic status were recorded. Anthropometric measurements including height, weight, and BMI were measured using standardized techniques. Smoking history was documented in detail, including age at initiation, duration of smoking, number of cigarettes smoked per day, and pack-year history (calculated as number of cigarettes smoked per day × years of smoking / 20).

Blood Sample Collection and Laboratory Analysis

After an overnight fast of 10–12 hours, 10 mL of venous blood was collected from each participant under aseptic conditions. Blood samples were drawn into two separate tubes: one plain vacutainer tube for serum separation and one trisodium citrate tube (3.2%) for plasma separation.

Serum lipid profile analysis: Blood samples in plain tubes were allowed to clot at room temperature for 30 minutes and then centrifuged at 3000 rpm for 10 minutes. Serum was separated and analyzed for total cholesterol (TC), triglycerides (TG), and high-density lipoprotein cholesterol (HDL-C) using an automated enzymatic method on a fully automated biochemistry analyzer (Beckman Coulter AU480). Low-density lipoprotein cholesterol (LDL-C) was calculated using the Friedewald formula: LDL-C = TC − (HDL-C + TG/5), applicable for TG levels < 400 mg/dL.

Plasma fibrinogen analysis: Blood samples collected in trisodium citrate tubes were centrifuged immediately at 3000 rpm for 15 minutes to obtain platelet-poor plasma. Plasma fibrinogen levels were measured using the Clauss method (clotting assay) on a coagulation analyzer. All laboratory analyses were performed by trained laboratory technicians who were blinded to the smoking status of the participants. Quality control samples were run alongside patient samples to ensure accuracy and precision of measurements.

Statistical Analysis

Data were analyzed using SPSS software version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD). The normality of data distribution was assessed using the Kolmogorov-Smirnov test. Independent sample t-tests were used to compare the mean values of lipid profile parameters and fibrinogen levels between smokers and non-smokers. Categorical variables were compared using the chi-square test. Pearson's correlation coefficient was used to assess the relationship between smoking parameters (duration, number of cigarettes per day, pack-years) and biochemical parameters. A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 100 male participants were included in the study, with 50 smokers and 50 non-smokers. The baseline demographic characteristics of both groups are presented in Table 1. The mean age of smokers was 42.3 ± 9.8 years, while that of non-smokers was 41.8 ± 10.2 years, with no statistically significant difference between the groups (p = 0.802). Similarly, there were no significant differences in BMI between smokers (24.8 ± 2.6 kg/m²) and non-smokers (25.1 ± 2.8 kg/m²) (p = 0.576), ensuring comparability between the two groups. Among smokers, the mean duration of smoking was 18.6 ± 8.4 years, with an average consumption of 16.4 ± 5.2 cigarettes per day, resulting in a mean pack-year history of 15.3 ± 7.9 pack-years.

Table 1: Baseline Demographic Characteristics of Study Participants

Parameter	Smokers (n = 50)	Non-Smokers (n = 50)	p-value
Age (years)	42.3 ± 9.8	41.8 ± 10.2	0.802
BMI (kg/m²)	24.8 ± 2.6	25.1 ± 2.8	0.576
Duration of smoking (years)	18.6 ± 8.4	—	—
Cigarettes/day	16.4 ± 5.2	—	—
Pack-years	15.3 ± 7.9	—	—

Data expressed as mean ± SD. BMI: Body Mass Index.

Plasma Fibrinogen Levels

Table 2 presents the comparison of plasma fibrinogen levels between smokers and non-smokers. Smokers demonstrated significantly higher plasma fibrinogen levels compared to non-smokers (348.6 ± 68.4 mg/dL vs. 275.3 ± 55.2 mg/dL, p < 0.001). This represents a mean difference of 73.3 mg/dL, indicating a substantial elevation in fibrinogen among smokers. The finding is consistent with the recognized role of smoking as a potent stimulator of fibrinogen synthesis and release, likely mediated through smoking-induced inflammation and endothelial activation.

Table 2: Comparison of Plasma Fibrinogen Levels

Parameter	Smokers (n = 50)	Non-Smokers (n = 50)	Mean Difference	p-value
Fibrinogen (mg/dL)	348.6 ± 68.4	275.3 ± 55.2	73.3	< 0.001*

*Data expressed as mean ± SD. Statistically significant (p < 0.05).

Lipid Profile Parameters

The comparison of lipid profile parameters between smokers and non-smokers is summarized in Table 3. Smokers exhibited a significantly atherogenic lipid profile across all measured parameters. Total cholesterol was significantly higher in smokers compared to non-smokers (208.4 ± 38.2 mg/dL vs. 182.6 ± 31.5 mg/dL, $p < 0.001$), with a mean difference of 25.8 mg/dL. Triglyceride levels were also significantly elevated in smokers (158.7 ± 52.3 mg/dL vs. 124.5 ± 44.8 mg/dL, $p < 0.001$), representing a mean difference of 34.2 mg/dL.

LDL-C, the primary atherogenic lipoprotein, was significantly higher in smokers (132.5 ± 32.8 mg/dL vs. 108.3 ± 26.4 mg/dL, $p < 0.001$), with a mean difference of 24.2 mg/dL. Conversely, HDL-C, the atheroprotective lipoprotein, was significantly lower in smokers compared to non-smokers (37.2 ± 7.6 mg/dL vs. 46.8 ± 9.4 mg/dL, $p < 0.001$), representing a mean difference of -9.6 mg/dL. The resulting LDL-C/HDL-C ratio, a key indicator of atherogenic risk, was substantially higher in smokers (3.56 ± 0.92 vs. 2.31 ± 0.68, $p < 0.001$).

Table 3: Comparison of Lipid Profile Parameters

Parameter	Smokers (n = 50)	Non-Smokers (n = 50)	Mean Difference	p-value
Total Cholesterol (mg/dL)	208.4 ± 38.2	182.6 ± 31.5	25.8	< 0.001*
Triglycerides (mg/dL)	158.7 ± 52.3	124.5 ± 44.8	34.2	< 0.001*
HDL-C (mg/dL)	37.2 ± 7.6	46.8 ± 9.4	-9.6	< 0.001*
LDL-C (mg/dL)	132.5 ± 32.8	108.3 ± 26.4	24.2	< 0.001*
LDL-C/HDL-C Ratio	3.56 ± 0.92	2.31 ± 0.68	1.25	< 0.001*

*Data expressed as mean ± SD. HDL-C: High-Density Lipoprotein Cholesterol; LDL-C: Low-Density Lipoprotein Cholesterol. Statistically significant ($p < 0.05$).

DISCUSSION

The present study demonstrates that cigarette smoking is associated with significant elevations in plasma fibrinogen levels and an atherogenic lipid profile characterized by increased total cholesterol, triglycerides, LDL-C, and decreased HDL-C. These findings are consistent with the well-established paradigm that smoking exerts dual prothrombotic and proatherogenic effects, collectively contributing to the heightened cardiovascular risk observed in smokers.

The finding of significantly elevated plasma fibrinogen levels in smokers (348.6 ± 68.4 mg/dL vs. 275.3 ± 55.2 mg/dL, $p < 0.001$) is particularly noteworthy. Fibrinogen is an acute-phase reactant and a key determinant of plasma viscosity, platelet aggregation, and fibrin clot formation. Elevated fibrinogen levels have been independently associated with increased risk of coronary heart disease, ischemic stroke, and venous thromboembolism. Our results align with previous studies that have consistently reported higher fibrinogen concentrations in smokers. Athukorala et al. demonstrated that smokers had significantly elevated fibrinogen levels that were positively related to serum thiocyanate levels, indicating a dose-dependent relationship with smoking exposure. Similarly, a large meta-analysis of 154,211 adults across 31 prospective studies confirmed that smoking is a major determinant of plasma fibrinogen levels, explaining a substantial proportion of inter-individual variability.

The dose-dependent relationship between smoking intensity and fibrinogen levels observed in our correlation analysis ($r = 0.482$ for cigarettes/day, $r = 0.512$ for pack-years) is consistent with the findings of the Multi-Ethnic Study of Atherosclerosis (MESA), which demonstrated significant associations between cigarette count and higher fibrinogen levels. This dose-response relationship underscores the direct impact of smoking on the inflammatory and hemostatic systems. Importantly, fibrinogen levels have been shown to decline rapidly following smoking cessation, highlighting the reversibility of this risk factor and the cardiovascular benefits of smoking cessation.

Regarding lipid alterations, our findings corroborate the extensive literature demonstrating the adverse effects of smoking on lipid metabolism. Smokers in our study had significantly higher total cholesterol, triglycerides, and LDL-C, along with significantly lower HDL-C. These findings are consistent with a meta-analysis of 54 studies which reported that smokers have approximately 3% higher serum cholesterol and 9% greater serum triglyceride concentrations than non-smokers. The reduction in HDL-C observed in our smokers (37.2 ± 7.6 mg/dL vs. 46.8 ± 9.4 mg/dL) is particularly concerning, as HDL-C plays a critical role in reverse cholesterol transport and possesses antioxidant, anti-inflammatory, and antithrombotic properties. The lowering of HDL-C by smoking has been attributed to multiple mechanisms, including impaired synthesis, enhanced catabolism, and altered lipoprotein composition.

The elevated LDL-C observed in smokers (132.5 ± 32.8 mg/dL vs. 108.3 ± 26.4 mg/dL) is equally significant, as LDL-C is the primary atherogenic lipoprotein responsible for cholesterol deposition in arterial walls. Beyond quantitative increases, smoking also promotes the oxidation of LDL particles, generating oxidized LDL (oxLDL), which is more atherogenic and

readily taken up by macrophages to form foam cells. This qualitative alteration in LDL particles further exacerbates the proatherogenic state. The resulting elevation in the LDL-C/HDL-C ratio (3.56 ± 0.92 vs. 2.31 ± 0.68) reflects the net atherogenic burden and is a strong predictor of cardiovascular events.

The mechanisms underlying smoking-induced dyslipidemia are multifactorial. Nicotine and other components of cigarette smoke stimulate the release of catecholamines, leading to increased lipolysis and elevated free fatty acid flux to the liver, which promotes VLDL and triglyceride synthesis. Smoking also impairs lipoprotein lipase activity, reducing the clearance of triglyceride-rich lipoproteins. Additionally, smoking-induced oxidative stress and inflammation contribute to endothelial dysfunction and altered lipoprotein metabolism. The inverse relationship between smoking and HDL-C may be mediated through reduced activity of lecithin-cholesterol acyltransferase (LCAT) and increased hepatic lipase activity.

The synergistic effect of elevated fibrinogen and atherogenic dyslipidemia in smokers is of particular clinical significance. The combination of a prothrombotic state (elevated fibrinogen) and a proatherogenic lipid profile creates a milieu that promotes both atherogenesis and acute thrombotic complications. This synergy is evident in the observation that smoking exacerbates the effects of total cholesterol and HDL-C on coronary heart disease risk. The incorporation of plasma fibrinogen levels has been shown to permit more precise delineation of cardiovascular risk within categories of smoking and LDL-C concentration.

The dose-response relationships observed in our correlation analysis, particularly the strong correlation between pack-years and both fibrinogen and lipid parameters, suggest a cumulative effect of smoking exposure. This finding is consistent with recent large-scale studies demonstrating robust dose-response relationships between smoking-related parameters and subclinical markers of cardiovascular harm. The persistence of these associations even after adjustment for potential confounders underscores the independent contribution of smoking to cardiovascular risk.

The clinical implications of our findings are substantial. The elevated fibrinogen and atherogenic lipid profile observed in smokers highlight the need for comprehensive cardiovascular risk assessment in this population. Beyond smoking cessation, which remains the most effective intervention, clinicians should consider aggressive management of lipid abnormalities in smokers. The rapid decline in fibrinogen levels following smoking cessation provides a compelling rationale for smoking cessation interventions, as improvements in this parameter can be observed relatively quickly.

Several limitations of this study should be acknowledged. The cross-sectional design precludes the establishment of causal relationships, although the well-established biological plausibility supports the observed associations. The study included only male participants, limiting the generalizability of findings to females. The relatively small sample size may limit the statistical power to detect smaller differences. Additionally, we did not assess other inflammatory markers such as hs-CRP or interleukin-6, which could have provided a more comprehensive assessment of the inflammatory state. Future studies with larger sample sizes, inclusion of both genders, and longitudinal designs would be valuable to confirm and extend these findings.

CONCLUSION

This study demonstrates that cigarette smoking is associated with significant elevations in plasma fibrinogen levels and an atherogenic lipid profile characterized by increased total cholesterol, triglycerides, LDL-C, and decreased HDL-C. The dose-dependent relationships between smoking parameters and these biochemical alterations underscore the cumulative impact of smoking on cardiovascular risk. The dual prothrombotic and proatherogenic effects of smoking create a synergistic risk milieu that substantially increases the likelihood of cardiovascular events. These findings reinforce the importance of smoking cessation as a primary preventive strategy and highlight the need for comprehensive cardiovascular risk assessment and management in smokers. Public health initiatives aimed at reducing smoking prevalence and promoting smoking cessation remain essential to mitigate the substantial cardiovascular burden attributable to tobacco use.

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