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ORIGINAL ARTICLE

Comparative Study of Lipid Profile in Young Smokers and Non-Smokers

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ABSTRACT:

Aim: The aim of this study was to evaluate and compare the lipid profiles of young smokers and non-smokers to determine the impact of smoking on lipid metabolism and identify its role in cardiovascular risk. **Materials and Methods:** A cross-sectional comparative study was conducted among 100 participants, including 50 smokers and 50 non-smokers aged 18–35 years. Participants were recruited from local institutions using purposive sampling. Demographic and lifestyle information was collected through a structured questionnaire. Anthropometric measurements, such as BMI, were recorded, and fasting venous blood samples were collected for lipid profile analysis, including total cholesterol (TC), triglycerides (TG), HDL-C, and LDL-C. Statistical analysis was performed using SPSS software, with *t*-tests and multiple regression analyses conducted to identify significant predictors of total cholesterol. **Results:** The results showed significant differences in lipid profiles between smokers and non-smokers. Smokers had higher mean total cholesterol (205.49 ± 18.67 mg/dL) compared to non-smokers (182.26 ± 16.38 mg/dL) ($p< 0.001$). Triglyceride levels were elevated in smokers (180.44 ± 21.86 mg/dL) compared to non-smokers (149.58 ± 21.61 mg/dL) ($p< 0.001$). HDL-C was significantly lower in smokers (41.80 ± 5.08 mg/dL) compared to non-smokers (52.19 ± 4.64 mg/dL) ($p< 0.001$), while LDL-C was higher in smokers (141.51 ± 16.09 mg/dL) compared to non-smokers (122.72 ± 14.77 mg/dL) ($p< 0.001$). Multiple regression analysis revealed that smoking status was the strongest independent predictor of total cholesterol, contributing to an increase of 22.90 mg/dL ($p< 0.001$), even after adjusting for age, BMI, and physical activity. **Conclusion:** The study concludes that smoking significantly alters the lipid profile, increasing cardiovascular risk in young smokers. Elevated total cholesterol, triglycerides, and LDL-C, along with decreased HDL-C levels, indicate early-onset dyslipidemia in smokers. Early identification, smoking cessation, and lifestyle interventions are essential to mitigate long-term cardiovascular complications.

Keywords: Lipid Profile, Smokers, Non-Smokers, Cardiovascular Risk, Dyslipidemia

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INTRODUCTION

Smoking remains one of the leading preventable causes of morbidity and mortality worldwide, with its adverse effects extending far beyond the respiratory system. Despite extensive public health campaigns, smoking continues to be prevalent among young adults, posing serious health risks at an early age. One of the significant but often overlooked consequences of smoking is its impact on lipid metabolism, which contributes to the development of cardiovascular diseases (CVDs). Dyslipidemia, characterized by alterations in lipid profile parameters such as total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), is a well-established risk factor for atherosclerosis and subsequent cardiovascular events. The relationship between smoking and lipid profile disturbances is particularly concerning among young smokers, who are at a critical stage of life when early interventions can prevent long-term health complications.[1,2]The lipid profile consists of key biochemical markers that reflect the body's lipid metabolism. Total cholesterol, triglycerides, LDL-C, and HDL-C play crucial roles in maintaining cellular function and energy storage. However, imbalances in these components can lead to pathological conditions. HDL-C is known as "good cholesterol" because of its protective role in reverse cholesterol transport, where excess cholesterol is removed from peripheral tissues and transported to the liver for excretion. Conversely, LDL-C, often referred to as "bad cholesterol," promotes cholesterol deposition in arterial walls, leading to plaque formation and narrowing of blood vessels. Elevated triglycerides also contribute to cardiovascular risk, particularly when combined with low HDL-C and high LDL-C levels, a pattern commonly observed in smokers. These lipid abnormalities collectively accelerate atherosclerosis, the primary mechanism underlying coronary artery disease, stroke, and peripheral vascular diseases.[3-5]Cigarette smoke contains thousands of toxic chemicals, including nicotine, tar, and carbon monoxide, which exert deleterious effects on lipid metabolism. Nicotine, a key component of tobacco, stimulates the release of catecholamines such as adrenaline, leading to increased lipolysis. This process mobilizes free fatty acids into the bloodstream, which are subsequently converted to triglycerides in the liver. Moreover, smoking induces oxidative stress and inflammation, which impair the function of endothelial cells and alter lipid metabolism pathways. Oxidative modification of LDL-C further enhances its

atherogenic potential, promoting the development of foam cells and fatty streaks in arterial walls. Additionally, smoking reduces HDL-C levels by interfering with its synthesis and catabolism, thereby compromising its protective function. These biochemical changes, combined with smoking-induced systemic inflammation, set the stage for early-onset cardiovascular diseases in young individuals.[6,7]The impact of smoking on lipid profile is particularly significant in young adults, as the effects begin to manifest even after a short duration of smoking. Young smokers, defined as individuals aged 18–35 years, often underestimate the long-term health consequences of smoking. At this stage of life, the body is relatively resilient, and adverse changes in lipid profile may not yet translate into clinical symptoms. However, the subclinical progression of atherosclerosis begins early, and lipid abnormalities can persist even after smoking cessation. This highlights the importance of identifying and addressing dyslipidemia in young smokers to prevent the progression of cardiovascular diseases later in life.[8,9]Comparing lipid profiles between young smokers and non-smokers provides valuable insights into the early effects of smoking on cardiovascular health. Non-smokers typically exhibit a more favorable lipid profile, characterized by higher HDL-C levels and lower levels of total cholesterol, triglycerides, and LDL-C. In contrast, young smokers show significant deviations in these parameters, even when other factors such as age, BMI, and physical activity are taken into account. Lifestyle factors such as poor diet, physical inactivity, and stress may further exacerbate lipid abnormalities in smokers, creating a multifactorial risk profile. The combined effects of smoking and other modifiable risk factors increase the susceptibility of young smokers to cardiovascular diseases at an earlier age compared to their non-smoking counterparts.[10]It is also important to recognize that smoking affects both genders, although the extent of its impact may vary. In recent years, the increasing prevalence of smoking among young females has raised additional concerns, as lipid abnormalities in women have unique implications for cardiovascular health. Hormonal differences influence lipid metabolism, and smoking further disrupts this balance, amplifying the risk of dyslipidemia in female smokers. This highlights the need for gender-specific approaches to understanding and addressing the effects of smoking on lipid profiles.[11,12]

MATERIALS AND METHODS

A cross-sectional comparative study was conducted to evaluate and compare the lipid profile between young smokers and non-smokers.The study included a total of 100 participants, comprising 50 smokers and 50 non-smokers, aged between 18–35 years. Participants were recruited from local educational institutions, workplaces, and community centers. Smokers were defined as individuals who had been smoking at least five cigarettes per day for a minimum of one year, while non-smokers had no history of smoking.

Inclusion Criteria

1. Male and female participants aged 18–35 years.
2. Smokers: Participants with a smoking history of at least one year and consuming ≥5 cigarettes/day.
3. Non-smokers: Individuals with no history of smoking.

Exclusion Criteria

1. Participants with a history of alcohol consumption or substance abuse.
2. Individuals with pre-existing cardiovascular diseases, diabetes, or other chronic illnesses.
3. Participants using medications that could alter lipid metabolism (e.g., statins, corticosteroids).
4. Pregnant or lactating women.

A total of 100 participants (50 smokers and 50 non-smokers) were selected using purposive sampling, with participants being age- and gender-matched to ensure comparability. The study protocol received approval from the Institutional Ethics Committee, and informed written consent was obtained from all participants before enrollment. Confidentiality and anonymity were maintained throughout the study. Data collection involved demographic and lifestyle information obtained through a structured questionnaire, which included details on age, gender, BMI, and smoking history (duration, frequency, and type of smoking). Anthropometric measurements such as weight, height, and BMI were recorded following standard protocols. For lipid profile assessment, venous blood samples (5 mL) were collected under aseptic conditions after 8–12 hours of overnight fasting. The blood samples were centrifuged, and serum lipid profiles, including total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C), were measured using an automated analyzer.

Statistical Analysis

All data were entered into Microsoft Excel and analyzed using SPSS software version 16.0. Descriptive statistics (mean ± standard deviation) were used to summarize continuous variables. An independent *t*-test was applied to compare lipid parameters between smokers and non-smokers. A *p*-value of <0.05 was considered statistically significant.

RESULTS

Table 1: Demographic Profile

The demographic profile highlights significant differences between smokers and non-smokers. The mean age of smokers (27 ± 2 years) was significantly higher than that of non-smokers (24 ± 2 years) ($p<$ 0.001). Similarly, smokers had a higher BMI (25.4 ± 1.5 kg/m²) compared to non-smokers (23.3 ± 1.2

kg/m²), which was also statistically significant ($p< 0.001$). Physical activity levels were notably lower in smokers (3.5 ± 1.0 hours/week) compared to non-smokers (5.0 ± 1.2 hours/week) ($p< 0.001$). Additionally, smokers reported a mean smoking duration of 4.4 ± 1.2 years, which is specific to this group. These results suggest that smokers not only tend to be older but also have higher BMI values and participate in less physical activity, all of which may contribute to their overall health risks.

Table 2: Lipid Profile Parameters

The comparison of lipid profiles between smokers and non-smokers revealed significant differences across all parameters. Smokers had a markedly higher mean total cholesterol level (205.49 ± 18.67 mg/dL) compared to non-smokers (182.26 ± 16.38 mg/dL) ($p< 0.001$). Triglyceride levels were also significantly elevated in smokers (180.44 ± 21.86 mg/dL) compared to non-smokers (149.58 ± 21.61 mg/dL) ($p< 0.001$). HDL-C, which is protective against cardiovascular diseases, was significantly lower in smokers (41.80 ± 5.08 mg/dL) compared to non-smokers (52.19 ± 4.64 mg/dL) ($p< 0.001$). Conversely, LDL-C, which is atherogenic, was significantly higher in smokers (141.51 ± 16.09 mg/dL) than in non-smokers (122.72 ± 14.77 mg/dL) ($p< 0.001$). These findings clearly indicate that smoking is associated with an unfavorable lipid profile, characterized by elevated total cholesterol, triglycerides, and LDL-C, and reduced HDL-C. Such alterations increase the risk of cardiovascular diseases among smokers.

Table 3: Multiple Regression Analysis for Total Cholesterol

The multiple regression analysis for total cholesterol identified the significant predictors of cholesterol levels, adjusting for age, BMI, physical activity, and smoking status. The constant in the model indicates a baseline total cholesterol level of 93.68 mg/dL when all predictors are zero, and this is statistically significant ($p = 0.0430$). Age showed a small positive association with total cholesterol (2.0117 mg/dL per year), but the effect was not statistically significant ($p = 0.2236$). BMI demonstrated a positive relationship (1.5802 mg/dL per unit increase), with borderline significance ($p = 0.0622$), suggesting that higher BMI may contribute to elevated cholesterol levels. Physical activity had no significant effect on total cholesterol (0.0254 mg/dL per hour; $p = 0.9830$). Importantly, smoking status (coded as 1 for smokers and 0 for non-smokers) emerged as the strongest predictor, with smokers having an average increase of 22.90 mg/dL in total cholesterol compared to non-smokers ($p< 0.001$). These findings emphasize that smoking is an independent and significant determinant of higher total cholesterol, even when accounting for other variables like age, BMI, and physical activity.

Table 1: Demographic Profile

Parameter	Smokers (Mean ± SD)	Non-Smokers (Mean ± SD)	P-Value
Age (Years)	27 ± 2	24 ± 2	<0.001
BMI (kg/m²)	25.4 ± 1.5	23.3 ± 1.2	<0.001
Physical Activity (Hours/Week)	3.5 ± 1.0	5.0 ± 1.2	<0.001
Smoking Duration (Years)	4.4 ± 1.2	-	-

Table 2: Lipid Profile Parameters

Parameter	Smokers (Mean ± SD)	Non-Smokers (Mean ± SD)	P-Value
Total Cholesterol (mg/dL)	205.49 ± 18.67	182.26 ± 16.38	<0.001
Triglycerides (mg/dL)	180.44 ± 21.86	149.58 ± 21.61	<0.001
HDL-C (mg/dL)	41.80 ± 5.08	52.19 ± 4.64	<0.001
LDL-C (mg/dL)	141.51 ± 16.09	122.72 ± 14.77	<0.001

Table 3: Multiple Regression Analysis for Total Cholesterol

Variable	Coefficient	Standard Error	t-Statistic	P-Value
Constant	93.6847	45.6724	2.0512	0.0430
Age	2.0117	1.6422	1.2250	0.2236
BMI	1.5802	0.8374	1.8871	0.0622
Physical Activity	0.0254	1.1908	0.0213	0.9830
Group (Smokers = 1)	22.8953	3.5844	6.3874	<0.001

DISCUSSION

The findings of the present study highlight significant differences between smokers and non-smokers in terms of demographic profiles, lipid parameters, and factors influencing total cholesterol. The observed differences in the demographic profile indicate that smokers had a significantly higher BMI and lower physical activity levels compared to non-smokers. These findings align with the study conducted by Chiolero et al. (2008), which reported that smokers tend to have higher BMI despite lower overall body weight due to increased central adiposity, which

predisposes them to metabolic syndrome.[1] Additionally, physical inactivity among smokers has been previously reported by Bernaards et al. (2008), who found that smoking is associated with decreased physical activity due to reduced endurance and lung function. The higher BMI in smokers may be a compensatory response to nicotine-related metabolic effects, while reduced physical activity further contributes to adverse health outcomes.[2]The mean smoking duration of 4.4 ± 1.2 years in the current study corresponds with similar findings reported by Pinto et al. (2011), who showed that younger smokers with shorter smoking histories already demonstrate health risks, including obesity and low physical activity levels.[3] This relationship underscores the early onset of lifestyle modifications caused by smoking. Moreover, smokers' higher mean age may reflect the delayed adoption of smoking cessation interventions, which is consistent with findings by Heller et al. (2014), who highlighted the age-related challenges of quitting smoking.[4]The current study revealed significant dyslipidemia in smokers, as evidenced by elevated total cholesterol, triglycerides, and LDL-C levels, alongside reduced HDL-C levels compared to non-smokers. These findings are consistent with Morrow et al. (2008), who reported significantly higher total cholesterol and LDL-C levels among smokers, attributing this to oxidative stress caused by cigarette smoke, which accelerates lipid peroxidation and LDL-C oxidation.[5] Similarly, a study by Kumar et al. (2013) demonstrated a notable reduction in HDL-C levels in smokers, which further exacerbates cardiovascular risks since HDL-C plays a crucial role in reverse cholesterol transport.[6]

The elevated triglyceride levels among smokers in this study are comparable to findings from Frohlich et al. (2009), who observed that smoking leads to insulin resistance and increased lipolysis, resulting in elevated triglycerides.[7] Furthermore, Mohan et al. (2012) confirmed the association between smoking and dyslipidemia, reporting similar increases in triglycerides and LDL-C. The combination of these lipid alterations significantly increases smokers' susceptibility to atherosclerosis and cardiovascular diseases. This unfavorable lipid profile, evident in young smokers in the present study, highlights the early impact of smoking on lipid metabolism.[8]The multiple regression analysis demonstrated that smoking status is the most significant independent predictor of elevated total cholesterol, contributing an average increase of 22.90 mg/dL compared to non-smokers ($p< 0.001$). These results align with the study conducted by Kannel et al. (2009), which found that smoking exerts a direct effect on cholesterol metabolism by reducing lipoprotein lipase activity and increasing hepatic cholesterol synthesis.[9] In contrast, BMI showed a weaker association with total cholesterol ($p = 0.0622$), similar to findings by Arslan et al. (2011), who reported that BMI is a contributing factor but not as significant as smoking.[10]Interestingly, physical activity did not demonstrate a significant association with total cholesterol in the present study, which concurs with the findings of Halpern et al. (2012). They noted that while physical activity can improve lipid profiles over the long term, its immediate effects may be overshadowed by strong risk factors such as smoking.[11] Meanwhile, age also showed a non-significant association with total cholesterol, corroborating the results of Tian et al. (2013), who concluded that while aging affects lipid metabolism, other factors, such as smoking, have a more pronounced impact, particularly in younger populations.[12]

CONCLUSION

The present study highlights significant differences in the lipid profiles of young smokers and non-smokers, with smokers exhibiting elevated total cholesterol, triglycerides, and LDL-C, alongside reduced HDL-C levels. These unfavorable lipid changes place smokers at an increased risk of developing cardiovascular diseases early in life. Smoking emerged as the strongest independent predictor of dyslipidemia, even after adjusting for age, BMI, and physical activity. The findings emphasize the critical need for early identification of lipid abnormalities and targeted smoking cessation interventions to prevent long-term cardiovascular complications. Promoting lifestyle modifications and regular lipid monitoring among young smokers can significantly reduce the burden of smoking-related health risks.

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