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Low Density Lipoprotein Levels in Male Athletes Smoking and Non-Smokers Ages 17 - 20 Years

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ABSTRACT

Smoking is known to increase oxidative stress, disrupt lipid metabolism, and accelerate the formation of oxidized Low Density Lipoprotein (LDL). Conversely, regular physical exercise enhances antioxidant capacity and improves lipid regulation. The interaction between smoking habits and intense physical activity in young athletes is important, as both may influence lipid balance and LDL levels associated with atherosclerosis risk. This study aimed to determine differences in LDL levels between smoking and non-smoking athletes aged 17–20 years. This study used an analytical observational design with a cross-sectional approach. A total of 23 male athletes were divided into smoking and non-smoking groups. Blood samples were collected via venipuncture to measure LDL levels through laboratory analysis. Data normality was tested using the Shapiro–Wilk test. Since one group was not normally distributed, the Mann–Whitney test was applied with a significance level of 0.05. Smoking athletes had a higher mean LDL level (107.10 ± 29.11 mg/dL) compared to non-smoking athletes (86.02 ± 20.34 mg/dL). However, statistical analysis showed no significant difference between the groups ($p > 0.05$). Descriptively, there was a tendency toward higher LDL levels in smoking athletes, although all values remained within ranges potentially influenced by regular physical activity. moking habits in young athletes did not show a significant effect on LDL levels. This may be due to physiological compensatory mechanisms induced by regular physical training, which can mitigate the negative effects of smoking on lipid metabolism.

Keywords: Low Density Lipoprotein, smoking athletes, physical exercise, oxidative stress

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Introduction

Smoking among young athletes is a serious health issue that is often overlooked. Despite engaging in intensive physical activity, some young athletes continue to smoke, believing that their physical fitness can offset the negative effects of smoking (Beischer et al., 2020). This misconception may lead to underestimation of the long-term health risks associated with smoking. A recent study by Saiphoklang et al.,(2020) reported that the prevalence of smoking among young athletes reaches 25–30%, a concerning figure given its potential impact on both health and athletic performance.

Cigarette smoke contains more than 7,000 chemical compounds, of which nicotine, carbon monoxide (CO), and free radicals are the primary components affecting lipid metabolism (Srinivasa Rao Ch., 2012). Nicotine stimulates the sympathetic nervous system, increasing catecholamine release and leading to excessive lipolysis (Mata et al., 2019). Carbon monoxide has a much higher affinity for hemoglobin than oxygen, resulting in tissue hypoxia that negatively affects lipid metabolism. In addition, free radicals in cigarette smoke induce oxidative stress and trigger systemic inflammatory responses.

The direct impact of smoking on athletes' lipid profiles is substantial. Previous research (Addissouky et al., 2024) indicates that nicotine and other chemicals in cigarettes induce oxidative stress and systemic inflammation, disrupting lipid metabolism. This condition leads to increased hepatic LDL production and reduced clearance of LDL from the bloodstream. Furthermore, Vekic et al., (2023) reported that smoking alters LDL into small dense LDL particles, which are smaller, denser, and more atherogenic than typical LDL. These particles are formed through oxidative modification caused by exposure to cigarette chemicals, making them more likely to contribute to plaque formation in blood vessels.

Furthermore, Zhang et al., (2019) demonstrated that the accumulation of oxidized LDL can trigger early atherosclerosis, reducing oxygen supply to muscles during exercise. This condition may result in decreased endurance, prolonged recovery time, and an increased risk of injury. In addition, Jia et al., (2019) found that adolescent smoking athletes exhibit 15% lower aerobic capacity compared to non-smokers. These findings highlight the negative implications of smoking on athletic performance and physiological function.

According to the Global Sport Institute (2023), effective strategies to address smoking among young athletes include targeted health education programs, routine medical check-ups including lipid profile testing, and smoking cessation programs tailored to training schedules. Coaches and sports organizations also play a critical role in fostering a health-supportive environment. Their involvement is essential in promoting long-term behavioral changes among athletes. These combined efforts are expected to reduce the prevalence of smoking and its associated health risks (Sarzynski et al., 2020).

This study was conducted due to the limited number of studies specifically examining the relationship between LDL levels and smoking habits in adolescent athletes. Most previous research has focused on the general population or adolescents with obesity, leaving a gap in athlete specific data (DiPietro et al., 2020).

Therefore, this study aims to provide a scientific basis for early prevention strategies, safer training program development, and informed health decision-making. The findings are expected to contribute to improving health outcomes among young athletes aged 17–20 years who engage in smoking (López-Bueno et al., 2020).

Methodology

This study employed an analytical observational design with a cross-sectional approach to compare Low-Density Lipoprotein (LDL) levels between smoking and non-smoking athletes aged 17–20 years. The study subjects consisted of 26 male athletes who were actively engaged in regular training and met the inclusion criteria, including having a normal body mass index (18.5–24.9 kg/m^2) and no history of metabolic or cardiovascular diseases. The smoking group was defined as athletes who consumed at least one cigarette per day consistently for a minimum of six months, while the non-smoking group consisted of athletes who had never smoked and were not regularly exposed to cigarette smoke.

Blood sampling was conducted in the morning between 07:00 and 09:00 after approximately 10 hours of fasting. A total of 5 mL of venous blood was collected from the cubital vein. LDL levels were analyzed at the Central Research & Diagnostic Laboratory of Satwa Sehat Malang using blood chemistry analysis methods, with results expressed in mg/dL. The reference value for LDL used in this study was <130 mg/dL for males, and values exceeding this threshold were categorized as elevated LDL levels.

The measurement data were analyzed descriptively to describe the baseline characteristics of the subjects in both groups. Normality testing was performed using the Shapiro–Wilk test to determine the distribution of LDL data in each group. Since one group was not normally distributed, the difference in LDL levels was analyzed using the Mann–Whitney U test with a significance level of 0.05. All statistical analyses were performed using SPSS version 26.0.

Result

This study presents the findings on the characteristics of the subjects, LDL levels, and statistical analyses comparing smoking and non-smoking athletes.

Table 1. Characteristics of Study Participants

		G1	G2
Variable	**n**	**Mean ± SD**	**Mean ± SD**
Age (years old)	26	18.85 ± 0.89	18.85 ± 1.06
Height (cm)	26	172.80 ± 4.75	168.38 ± 4.55
Weight (kg)	26	64.05 ± 11.90	61.36 ± 9.77
SYS (mmHg)	26	118.38 ± 11.26	123.31 ± 4.97
DYS (mmHg)	26	76.15 ± 8.97	81.15 ± 9.07
BMI (kg/m2)	26	21.35 ± 3.29	21.60 ± 3.14

Table 1 shows that each study group consisted of 26 subjects (n = 26). The mean age of both groups was identical at 18.85 years (G1 SD = 0.89; G2 SD = 1.06), indicating that the groups were demographically comparable in terms of age. The mean height in G1 was 172.80 ± 4.75 cm, while in G2 it was 168.38 ± 4.55 cm. The mean body weight was higher in G1 (64.05 ± 11.90 kg) compared to G2 (61.36 ± 9.77 kg).

Body Mass Index (BMI) was relatively similar between the groups (G1 = 21.35 ± 3.23 kg/m^2; G2 = 21.60 ± 3.14 kg/m^2), indicating comparable nutritional status. In terms of blood pressure, the G2 group (SYS = 123.31 ± 4.97 mmHg; DYS = 81.15 ± 9.07 mmHg) showed higher mean systolic and diastolic values compared to the G1 group (SYS = 118.38 ± 11.26 mmHg; DYS = 76.15 ± 8.97 mmHg).

Table 2. Analysis of LDL Levels

		Low-Density Lipoprotein (LDL) Levels
Groups	**n**	**Mean ± SD**
K_1	13	107.10 **±** 29.11
K_2	13	86.02 **±** 20.34

Based on the descriptive analysis presented in Table 2, the mean Low-Density Lipoprotein (LDL) levels in the smoking athlete group (107.10 ± 29.11 mg/dL) were higher than those in the non-smoking athlete group (86.02 ± 20.34 mg/dL). This finding indicates a tendency toward increased LDL levels among smoking athletes.

Table 3. Normality Test Results of LDL Levels

Groups	*p*-value	Description
G_1	0.040	Not normally distributed
G_2	0.987	Normally distributed

The normality test results (Table 3) showed that LDL data in the smoking group (G1) were not normally distributed (p = 0.040), whereas the non-smoking group (G2) showed a normal distribution (p = 0.987). As one group violated the normality assumption, the overall data were considered non-normally distributed. Therefore, differences in LDL levels between groups were analyzed using the non-parametric Mann–Whitney U test instead of a parametric test.

Table 4. Results of the Mann–Whitney U Test

Variable	P-Value	Description
LDL	0.091	Not Significant

The Mann–Whitney U test results (Table 4) showed no statistically significant difference in LDL levels between smoking (G1) and non-smoking athletes (G2) (p = 0.091). Although higher LDL values were observed in the smoking group, the

difference was not sufficient to reach statistical significance, indicating that smoking did not have a measurable effect on LDL levels in this study population.

Dicussion

Physiological Effects of Smoking on LDL

Smoking influences lipid metabolism through oxidative stress, enzymatic disruption, and hormonal changes. Exposure to cigarette smoke introduces toxic compounds such as nicotine, carbon monoxide, and free radicals, which promote oxidative stress and systemic inflammation (Vicol et al., 2022). This condition increases the susceptibility of LDL particles to oxidation, reducing their clearance from circulation and potentially elevating LDL levels (Mitra et al., 2011). Additionally, smoking may decrease lipoprotein lipase (LPL) activity and increase lipolysis via catecholamine release, further contributing to increased very low-density lipoprotein (VLDL) production and LDL formation (Behl & Stamford, 2022). However, these effects are typically more pronounced in individuals with long-term and heavy smoking exposure.

Physiological Effects of Exercise on LDL

Regular physical exercise plays a protective role in lipid metabolism through multiple physiological adaptations (Łakomy et al., 2025). Exercise enhances LPL activity, improves insulin sensitivity, and increases mitochondrial fat utilization, leading to reduced VLDL production and improved lipid profiles (Logan et al., 2023). Furthermore, exercise increases endogenous antioxidant capacity, which helps counteract oxidative stress and prevents LDL oxidation (Ghafouri et al., 2019). It also improves endothelial function and promotes high-density lipoprotein (HDL)-mediated reverse cholesterol transport, indirectly maintaining LDL within normal ranges (García-Giménez et al., 2024)

LDL Levels in Smoking Athletes

The present study found no statistically significant difference in LDL levels between smoking and non-smoking athletes, although a higher mean LDL was observed in the smoking group. This finding highlights an important aspect: regular physical training may attenuate the negative effects of smoking on lipid metabolism (Bhale et al., 2024). Physiological adaptations in athletes, including enhanced antioxidant defense and improved lipid utilization, may act as compensatory mechanisms that stabilize LDL levels despite exposure to cigarette smoke (Harrison et al., 2019).

In addition, the relatively young age of participants and the likely moderate smoking intensity may contribute to the absence of significant differences (Ishida et al., 2024). Younger individuals generally have better metabolic and cardiovascular resilience, allowing for greater physiological compensation against risk factors such as smoking (Healy et al., 2024)

Conclusions

This study found no statistically significant difference in Low-Density Lipoprotein (LDL) levels between smoking and non-smoking athletes aged 17–20 years. Although smoking athletes exhibited higher mean LDL levels, the difference was not statistically significant. These findings suggest that regular physical activity may attenuate the negative effects of smoking on lipid metabolism through physiological adaptations. However, smoking should not be considered safe, as its long-term effects may still pose significant health risks. Further studies with larger sample sizes and longer exposure duration are recommended to better understand the relationship between smoking and lipid profiles in athletes.

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