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Platelet Count as a Marker of Thrombosis Risk in Male Athlete Smokers Aged 17–20 Years

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ABSTRACT

Platelets play a crucial role in hemostasis and blood clot formation, contributing to vascular stability. Cigarette smoking is known to increase oxidative stress, which may impair endothelial function and promote platelet activation. In athletes, regular physical training induces physiological adaptations that help regulate hematological balance, whereas smoking exposure introduces reactive oxygen species that may increase thrombotic potential. This study aimed to compare platelet counts among three groups of young males: athlete smokers, athlete non-smokers, and non-athlete controls aged 17–20 years. A total of 33 participants were selected using purposive sampling, with 11 individuals in each group. Venous blood samples were collected from the cubital vein and analyzed using a hematology analyzer. Data normality was assessed using the Shapiro–Wilk test, and group differences were analyzed using one-way ANOVA with a significance level of 0.05. The highest mean platelet count was observed in athlete non-smokers (380.65 ± 138.31 $\times 10^3/\mu L$), followed by the control group (328.48 ± 65.11 $\times 10^3/\mu L$) and athlete smokers (276.20 ± 64.61 $\times 10^3/\mu L$). Statistical analysis revealed no significant differences among groups ($p = 0.096$). These findings suggest that regular physical activity may help maintain platelet stability in young athletes despite smoking habits; however, the potential long-term risk of thrombosis associated with smoking should be considered.

Keywords: Platelets, Smoking, Athletes

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Introduction

Hemostasis is a tightly regulated physiological process in which platelets play a central role in preventing bleeding and restoring damaged vascular tissue (Mandel et al., 2022). Normal platelet counts range from 150,000 to 450,000/μL, with an average lifespan of approximately 8–10 days, reflecting their dynamic turnover in circulation (Chaudhary et al., 2022). Deviations from this physiological range may compromise vascular integrity and disrupt blood flow, thereby increasing the risk of thrombotic or hemorrhagic events (Repsold & Joubert, 2021). During vascular injury, platelets adhere to the damaged site and release procoagulant mediators that accelerate clot formation and maintain hemostatic stability (Azzahra, 2020).

Platelet counts and activity are known to vary in response to physiological stress, including physical exercise. Acute and chronic exercise stimuli can trigger adaptive responses such as splenic contraction, resulting in the release of platelet reserves into systemic circulation (Mochizuki et al., 2025). During high-intensity exercise, increased oxygen consumption promotes the production of reactive oxygen species (ROS), which is typically counterbalanced by enhanced antioxidant enzyme activity, including superoxide dismutase (Dahlia, 2023). However, when ROS production exceeds endogenous antioxidant capacity, platelet activation may increase, thereby elevating the risk of excessive coagulation (Wang & Zennadi, 2020). Consequently, appropriate regulation of exercise intensity

is essential for maintaining platelet homeostasis in athletes.

A contrasting physiological condition is observed in individuals exposed to cigarette smoke. Smoking induces excessive free radical production, accelerating oxidative stress and impairing endothelial function, which subsequently influences platelet behavior (Klein et al., 2023). In athlete non-smokers, platelet counts may increase transiently following exercise while remaining within normal physiological limits (Jia et al., 2024). Conversely, exposure to nicotine and carbon monoxide in athlete smokers has been shown to enhance platelet aggregation and promote a prothrombotic state (Khudhur et al., 2025). Similar endothelial dysfunction has also been reported with the use of electronic cigarettes, indicating that alternative nicotine delivery systems may exert comparable vascular effects (Lyytinen et al., 2023). These findings suggest that platelet responses to exercise-induced stress may be exaggerated in athletes who smoke compared to their non-smoking counterparts (Barale et al., 2023).

Excessive platelet activation or elevated platelet counts can increase blood viscosity and promote clot formation, thereby heightening the risk of thrombosis (Firdayanti et al., 2023). Thrombus formation within blood vessels may obstruct blood flow and lead to serious cardiovascular complications, particularly in physically active individuals (Miele et al., 2024). Active smokers exhibit increased platelet activity and fibrinogen levels, contributing to a hypercoagulable state through mechanisms described in Virchow's triad (Dzikrinina & Zahra, 2024). These alterations indicate that smoking not only compromises athletic performance but also increases thrombotic risk through endothelial dysfunction and arterial stiffness (Mohammadi et al., 2022).

Based on these considerations, smoking status may be associated with variations in platelet count and thrombotic risk among young athletes. However, evidence regarding platelet quantity in adolescent athlete smokers remains limited. Therefore, this study aimed to compare platelet counts as an indicator of thrombosis risk among athlete smokers, athlete non-smokers, and non-athlete control participants aged 17–20 years.

Methodology

This study employed a cross-sectional comparative analytical design to examine differences in platelet counts as an indicator of thrombosis risk among three groups, namely athlete smokers, athlete non-smokers, and non-athlete controls aged 17–20 years. Participants were recruited using purposive sampling, resulting in a total of 33 male participants with equal distribution across groups (n = 11 per group). Smoking status was determined using the World Health Organization Global Adult Tobacco Survey (WHO GATS/TQS), while individuals with hematological disorders or those consuming medications known to affect platelet function were excluded to reduce potential confounding factors.

Blood sampling was conducted under standardized conditions in the morning to minimize the influence of circadian variation. Approximately 5 mL of venous blood was collected from the cubital vein using sterile procedures. Platelet counts were measured as part of a Complete Blood Count (CBC) examination using an automated hematology analyzer at the Central Research and Diagnostic Laboratory (Central Riset & Diagnostik Satwa Sehat), Malang. The results were expressed in units of $\times 10^3/\mu L$.

All collected data were analyzed using statistical software to ensure the validity and reliability of the findings. The normality of platelet count distribution was assessed using the Shapiro–Wilk test to determine whether the data met the assumptions for parametric analysis. Differences in mean platelet counts among the three groups were examined using one-way analysis of variance (ANOVA). When necessary, further analysis was conducted to identify specific group differences, with the level of statistical significance set at $p < 0.05$. This analytical approach was designed to provide an objective assessment of platelet count variation as an early indicator of thrombosis risk among athletes with different smoking statuses.

Result

Tabel 1. Description of Participant Characteristics

Variable	n	x̄ ± SD
Weight (kg)	33	62,68 ± 9,925
Height (cm)	33	169,56 ± 4,759
Age (years old)	33	18,75 ± 0,936
BMI (kg/m^2)	33	21,75 ± 2,986
SYS (mmHg)	33	120,03 ± 7,43
DYS (mmHg)	33	78,63 ± 8,557

The descriptive characteristics of the participants are presented in Table 1. The mean age of the 33 participants was 18,75 ± 0,936 years. The average body weight and height were 62,68 ± 9,925 kg and 169,56 ± 4,759 cm, respectively. The mean body mass index was 21,75 ± 2,986 kg/m^2, indicating that most participants were within the normal range. Resting systolic and diastolic blood pressure values were 120,03 ± 7,43 mmHg and 78,63 ± 8,557 mmHg, suggesting that all participants were in normal physiological condition at the time of measurement.

Tabel 2. Platelet Levels and Normality Test

Group	n	x̄ ± SD	p-value
Smokers	11	276,2 ± 64,61	0,855
Athletes	11	380,65 ± 138,31	0,767
Control	11	328,48 ± 65,11	0,315

The Shapiro–Wilk test was conducted to examine the distribution of platelet count data across the three groups. The results of the normality test are presented in Table 2. Platelet count data for athlete smokers showed a significance value of 0,855, athlete non-smokers 0,767, and control participants 0,315. Since all significance values exceeded 0,05, the platelet count data were normally distributed. These results indicate that the data met the assumption of normality and were appropriate for parametric statistical analysis.

Tabel 3. ANOVA Test

Variable	*p*-value	Description
Trombosit	0,096	Not Significant

Differences in mean platelet counts among athlete smokers, athlete non-smokers, and control participants were analyzed using one-way ANOVA. The results of the ANOVA are shown in Table 3. The analysis produced a significance value of 0,096, which is greater than the threshold of 0,05. This finding indicates that there were no statistically significant differences in platelet counts among the three groups.

Tabel 4. Uji Post Hoc Dunnet T3

Group	p-value
Smoker–Athlete	0,176
Smoker–Control	0,305
Athlete–Control	0,684

Post hoc analysis using the Dunnett T3 test was performed to further examine pairwise differences between groups. The results are presented in Table 4. Comparisons between athlete smokers and athlete non-smokers yielded a significance value of 0,176, between athlete smokers and control participants 0,305, and between athlete non-smokers and control participants 0,684. All p-values were above 0,05, indicating that no significant differences were observed in platelet counts between any pair of groups.

Dicussion

The present study found no statistically significant differences in platelet counts among athlete smokers, athlete non-smokers, and non-athlete controls aged 17–20 years. This finding supports previous evidence suggesting that smoking does not always directly influence platelet quantity, but rather affects platelet function and activation during the coagulation process (Pamungkas, Tri, Ferlian & Widyantara, Bagus, 2021). Exposure to nicotine and oxidative compounds increases the production of reactive oxygen species (ROS), leading to oxidative stress and endothelial cell damage. This endothelial dysfunction reduces nitric oxide (NO) bioavailability, a key molecule that normally inhibits platelet

aggregation, thereby increasing platelet activity without necessarily altering platelet count (Handayani, Dwi, 2022). Hematological indicators such as mean platelet volume (MPV) and elevated P-selectin expression in smokers further reflect the presence of larger and more reactive platelets with greater coagulation potential (Handayani, Dwi, 2022).

However, these smoking-related effects appear to be less pronounced in athletes. Regular physical training provides compensatory physiological adaptations, including enhanced antioxidant capacity, improved blood flow, and preserved endothelial function, which collectively help maintain platelet counts within normal physiological limits. Exercise-induced activation of the sympathetic nervous system stimulates catecholamine release and splenic contraction, resulting in the transient mobilization of platelet reserves into circulation (Biernat et al., 2024). Over time, repeated exposure to physical training promotes long-term adaptations such as improved endothelial responsiveness, increased antioxidant defense, and reduced chronic inflammation. As a result, platelets become more stable, more sensitive to prostacyclin, and less prone to activation during resting conditions.

The absence of significant differences in platelet counts among groups may also be influenced by biological and lifestyle-related factors. The relatively young age of the participants, combined with intermittent smoking patterns, allows for efficient nicotine metabolism, thereby limiting toxin accumulation that could disrupt hemostatic processes. Consequently, platelet counts in smokers aged 17–20 years may remain within normal ranges (Rustiah et al., 2025). Variations in smoking frequency, number of cigarettes consumed, and filter usage further contribute to heterogeneous nicotine exposure among individuals. In addition, unregulated biological factors such as dietary intake, hydration status, psychological stress, and sleep quality may influence platelet levels and contribute to interindividual variability (Wibowo & Pangemanan, 2017). The use of certain medications or bioactive substances, including anticoagulants, cardiovascular agents, β-lactam antibiotics, as well as alcohol, caffeine, garlic, ginger, ginseng, and tobacco products, has also been reported to affect platelet activity (Pematasari & Zulkiefly, 2020).

Furthermore, the overall physical condition of the participants may have provided an additional protective effect. Adaptations resulting from regular physical exercise enhance endothelial cell function and antioxidant capacity, enabling the body to better counteract oxidative stress induced by intense physical activity and cigarette smoke exposure. Therefore, the lack of significant differences in platelet counts observed in this study is likely attributable to the combined effects of exercise-induced physiological adaptation, young age, variability in smoking intensity, and the influence of dietary and pharmacological factors.

Conclusions

This study demonstrates that platelet counts in athlete smokers, athlete non-smokers, and non-athlete controls aged 17–20 years remain within normal physiological ranges, with the highest mean values observed in athletes, followed by the control group and smokers. These findings indicate that cigarette exposure during late adolescence has not yet produced a measurable quantitative effect on platelet levels. Regular physical training appears to provide a protective effect by improving endothelial function, enhancing antioxidant capacity, and maintaining hemostatic stability, thereby attenuating the hematological impact of smoking. Although no statistically significant differences were identified among groups, the absence of quantitative changes in platelet count does not exclude potential long-term thrombotic risk, as smoking-related oxidative stress may primarily influence platelet activation rather than platelet number over prolonged exposure.

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