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ORIGINAL ARTICLE

Relationship between caffeine consumption and physical activity with arterial elasticity in medical students

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Abstract

Background: Medical students often consume high amounts of caffeine due to heavy academic demands and tend to be less physically active.
Objective: This study aimed to investigate the relationship between caffeine consumption and physical activity with arterial elasticity in medical students at the Faculty of Medicine, Universitas Pembangunan Nasional Veteran Jakarta in 2025.
Methods: This cross-sectional study included 80 undergraduate medical students aged 18–23 years, recruited through consecutive sampling. Data on physical activity, caffeine consumption, and demographics were collected using validated questionnaires. Stress, sleep quality, and eating habits were assessed using the PSS-10, PSQI, and AFHC, respectively. Only healthy participants with low to moderate stress, good sleep quality, and healthy eating habits were included in this study. The SA-3000P Accelerated Photoplethysmography (APG) analyzer was used to assess arterial elasticity, and the results were categorized as suboptimal, normal, or optimal.
Results: Suboptimal arterial elasticity was observed in 51.3% of participants. Caffeine consumption was moderate in most participants (61.3%), while physical activity levels were moderate in 57.5% of the participants. The Chi-square test showed a significant association between caffeine consumption and arterial elasticity ($p = 0.012$), whereas physical activity showed no significant association ($p = 0.093$).
Conclusions: High caffeine consumption was associated with reduced arterial elasticity in medical students, whereas physical activity was not significantly associated. These findings suggest a potential association between caffeine intake and vascular health in young adults.

Keywords: arterial elasticity, caffeine consumption, medical students, physical activity

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Introduction

The World Health Organization (WHO) states that cardiovascular disease is responsible for 17.9 million deaths each year worldwide. In Indonesia, this disease is the leading cause of death.[1] The considerable burden highlights that this condition remains a major health concern and emphasizes the urgent need for stronger preventive strategies.

One important biomarker that reflects vascular health is arterial elasticity.[2] Arterial elasticity is the ability of blood vessels to stretch and then return to their original shape (recoil) during the cardiac cycle. Reduced arterial elasticity results from an imbalance between elastin and collagen fibers in the vascular wall, contributing to elevated blood pressure, hemodynamic disturbances, and an increased risk of cardiovascular disease.[3] Arterial elasticity can be assessed non-invasively using Accelerated Photoplethysmography (APG), a simple and rapid method suitable for vascular evaluation.[2]

Despite various prevention and therapeutic efforts, the incidence of cardiovascular disease remains high. This indicates the need for a deeper understanding of the risk factors leading to vascular dysfunction. Young individuals, especially medical students, tend to have two risk factors for reduced arterial elasticity, namely high consumption of caffeine and lack of physical activity.[4] More than 80% of the world's population is caffeine consumers, with total consumption reaching around 120,000 tons per year. Caffeine consumption is also very common among students, especially medical students, with a reported prevalence of consumption reaching 94%.[5] The caffeine is found in various products, such as coffee, tea, soda, and chocolate.[6] In Indonesia, coffee consumption as the primary source of caffeine shows an increasing trend in the general population, from 8.2% in 2019 to 24% in 2021.[4] Besides its stimulant effects on the central nervous system, caffeine is known to have effects on the cardiovascular system, including through mechanisms of vasoconstriction, increased blood pressure, and decreased arterial elasticity.[7]

On the other hand, physical activity plays an important role in maintaining arterial elasticity. Research shows lower arterial elasticity in individuals with low physical activity levels compared to those with high physical activity levels.[8] However, WHO data from the 2022 Physical Activity Country Profile indicates that 33.5% of the adult population in Indonesia does not meet the minimum physical activity recommendation of 150 minutes of moderate-intensity activity or 75 minutes of vigorous-intensity activity per week.[1]

The high mortality rate due to cardiovascular disease in Indonesia emphasizes the importance of prevention strategies that focus on modifiable risk factors from a young age. In the context of student life, high caffeine consumption and low physical activity are two potential risk factors for decreased vascular function. Medical students tend to adopt a sedentary lifestyle due to high academic demands.[9] In addition, high caffeine consumption, particularly from coffee, is common among medical students to maintain alertness and stamina during academic activities.[10] Although several studies have examined the relationship between caffeine consumption and physical activity on vascular health, studies evaluating both factors, especially in medical students, are still limited. Therefore, this study aims to investigate the relationship between caffeine consumption and physical activity with arterial elasticity in medical students at the Faculty of Medicine, Universitas Pembangunan Nasional Veteran Jakarta. The results of this study are expected to serve as a basis for developing promotive and preventive strategies as well as early detection of cardiovascular disease risk in the young adult population.

Methods

The study used a cross-sectional design and was conducted from July to December 2025 at the Physiology and Nutrition Laboratory Unit, Medical Education and Research Center, Universitas Pembangunan Nasional Veteran Jakarta (UPNVJ). The study population consisted of students from the Faculty of Medicine UPNVJ.

The research participants were UPNVJ Faculty of Medicine students in the 2024/2025 academic year, aged 18-23 years, with mild or moderate stress levels, good sleep quality, good eating habits, non-smokers, no history of diabetes mellitus or cardiovascular disease, and no consumption of alcohol or medications that could affect vascular function. Eligibility screening was carried out using structured self-administered questionnaires and validated assessment instruments. All information was obtained directly from participants through self-report to ensure they met the inclusion criteria before enrollment.

The sample size was calculated using the difference of two proportions formula with $\alpha = 5\%$, $\beta = 80\%$, $P_1 = 0.565$ (proportion of participants with reduced arterial elasticity in the high caffeine group of the general population)[11], and $P_2 = 0.258$ (proportion of participants with reduced arterial elasticity in the healthy participants group of the general population).[12] The calculation resulted in 74 participants. The sample size was increased by 10% to 81 participants to account for possible non-response. Participants were selected using consecutive sampling. Arterial elasticity was measured using the Accelerate Photoplethysmography (APG) Analyzer SA-3000P with a sensitivity of 71.4% and specificity of 90%. Arterial elasticity was categorized as suboptimal (<30), normal (30-70), and optimal (>70).[13] The APG examination was performed by inserting the index finger into a finger probe for 2-3 minutes. Participants were asked to remove all metal accessories and then sit comfortably, and were instructed not to talk, move, or fall asleep during the measurement process. Before the examination, participants were asked not to consume caffeine for at least 4-6 hours or engage in strenuous exercise beforehand. The examination was conducted in a comfortable room with adequate temperature and lighting.

Caffeine consumption was assessed using the Caffeine Consumption Questionnaire-Revised (CCQ-R). The average accuracy of CCQ-R reporting reached 83.5%, and more than 60% of participants achieved an accuracy level of ≥ 85%, meeting the standards for inter-rater agreement. The reliability test results for this questionnaire yielded a Cronbach's alpha > 0.70.[14] Construct validity was reinforced by the absence of demographic factors influencing reporting accuracy. These results make the CCQ-R suitable for use in research and clinical practice.[15] The Modified Caffeine Consumption Questionnaire-Revised (mCCQ-R) is a modification of the Indonesian version of the CCQ-R, tailored to foods and beverages containing caffeine in Indonesia. The validity test results in the research population showed an average accuracy of 89%, with 73% of participants having a score >85%. Furthermore, caffeine consumption was categorized as low <100 mg, moderate 100-300 mg, and high >300 mg.[16]

Physical activity was measured using the Global Physical Activity Questionnaire (GPAQ) in Metabolic Equivalent of Task (MET) units for a week. This questionnaire has moderate to good reliability with Spearman's rho values of 0.67-0.73. Physical activity levels were categorized as high (MET > 3000), moderate (600≤MET≤3000), and low (MET < 600).[17]

Stress levels were measured using the Perceived Stress Scale (PSS)-10 questionnaire. Stress scores were categorized as low (0-13), moderate (14-26), and high (27-40).[18] The Indonesian version of the PSS-10 is known to be valid and reliable, with a Cronbach's alpha of 0.83.[19]

Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) with a Cronbach's alpha of 0.79.[20] A total PSQI score > 7 indicates poor sleep quality.[21] Eating habits were assessed using the Adolescent Food Habits Checklist (AFHC), which includes measurement of fat intake, high-energy foods, fruit and vegetable consumption, snack consumption, and nutritional knowledge. Scores of AFHC were categorized as good eating habits (score ≥12) and poor eating habits (score < 12). This questionnaire has been translated into Indonesian and demonstrated good reliability, with a Cronbach's alpha of 0.86.[22]

Univariate analysis was performed to describe the characteristics of the participants, physical activity, caffeine consumption habits, and arterial elasticity. Categorical variables were presented as frequencies and percentages. Continuous variables were first tested for normality using the Shapiro-Wilk test and then presented as mean ± standard deviation (SD) if normally distributed or median and interquartile range (IQR) if non-normally distributed. The Chi-square test was used to determine the relationship between caffeine consumption and physical activity with arterial elasticity. If the assumptions of the Chi-square test were not met, an alternative method, including cell merging, was applied. A p-value less than 0.05 was considered statistically significant. All data were analyzed using SPSS software version 24.

This study obtained ethical approval from the Health Research Ethics Committee of Universitas Pembangunan Nasional Veteran Jakarta, with number 317/XII/2025/KEP.

Results

One of the 81 study participants was excluded for being 17 years old, which did not meet the eligibility criteria (18-23 years). Therefore, a total of 80 participants were included in the final analysis. The number of male and female participants was equal, with a median age of 21 years. Most participants had a normal body mass index (BMI) (43.8%) and low stress levels (68.8%). As shown in **Table 1**, there were no significant differences in age (p = 0.598), sex (p = 0.371), BMI (p = 0.915), or stress level (p = 0.264) across arterial elasticity groups.

Table 1. Participants' characteristics according to arterial status

Characteristics	Arterial Elasticity: Suboptimal n = 41	Arterial Elasticity: Normal+Optimal n = 39	p-value
Age, median (IQR)	20 (2)	21 (2)	0.598[a]
Sex, n (%)			
Male	23 (57.5)	17 (42.5)	0.371[b]
Female	18 (45.0)	22 (55.0)	
BMI, n (%)			
Underweight	1 (33.3)	2 (66.7)	0.915[b]
Normoweight	18 (51.4)	17 (48.6)	
Overweight	11 (50.0)	11 (50.0)	

Obese	11 (55.0)	9 (45.0)	
Stress level, n (%)			
Low	31 (56.4)	24 (43.6)	0.264[b]
Moderate	10 (40.0)	15 (60.0)	

Notes: [a]Kruskal-Wallis test, [b]Chi-square test, Arterial elasticity: suboptimal (<30), normal (30–70), optimal (>70).

As presented in **Table 2**, most participants reported moderate caffeine consumption (61.3%) and moderate levels of physical activity (57.5%).

Table 2. Distribution of caffeine consumption and physical activity among the participants

Characteristics	Arterial Elasticity	
	Suboptimal n = 41	Normal+Optimal n = 39
Caffeine consumption, n (%)		
High	7 (100)	0 (0,0)
Moderate	23 (46.9)	26 (53.1)
Low	11 (45.8)	13 (54.2)
Physical Activity, n (%)		
High	14 (70.0)	6 (30.0)
Moderate	21 (45.7)	25 (54.3)
Low	6 (42.9)	8 (57.1)

Notes: Caffeine consumption: low (<100 mg/day), moderate (100–300 mg/day), high (>300 mg/day). Physical activity: high (MET > 3000), moderate (600–3000), low (<600). Arterial elasticity: suboptimal (<30), normal (30–70), optimal (>70).

The mean daily caffeine consumption was 166.79 mg, ranging from 0 to 619.91 mg/day. Based on caffeine sources, participants primarily consume caffeine from coffee (65%) and tea (63.8%) (**Figure 1**).

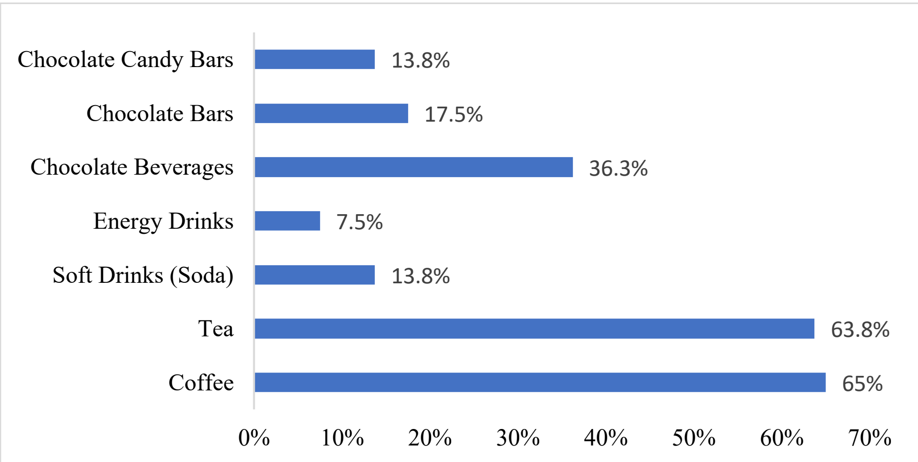

Figure 1. Distribution of caffeine consumption sources

In this study, 41 participants (51.2%) were classified as having suboptimal arterial elasticity (**Figure 2**).

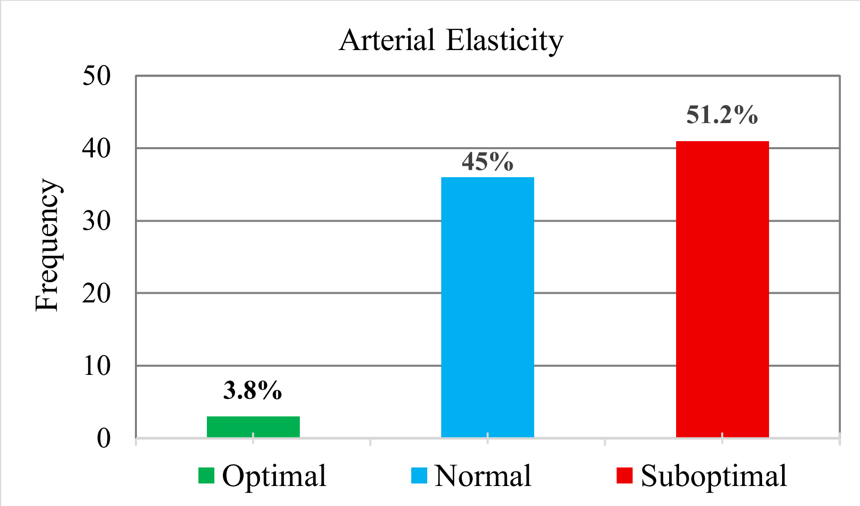

Figure 2. Distribution of arterial elasticity

Chi-square analysis demonstrated a significant association between caffeine consumption and arterial elasticity (p = 0.012) (**Table 3**).

Table 3. Association between caffeine consumption and arterial elasticity

Caffeine Consumption	Arterial Elasticity				Total		p-value
	Suboptimal		Optimal + Normal				
	N	%	N	%	N	%	
High	7	100	0	0	7	100	0.012
Low + Moderate	34	46.6	39	53.4	73	100	

In contrast, physical activity was not significantly associated with arterial elasticity (p = 0.093) (**Table 4**).

Table 4. Association between physical activity and arterial elasticity

Physical Activity	Arterial Elasticity				Total		p-value
	Suboptimal		Optimal + Normal				
	N	%	N	%	N	%	
Low + Moderate	27	45.0	33	55.0	60	100	0.093
High	14	70.0	6	30.0	20	100	

Discussion

The distribution of participant characteristics was comparable between the suboptimal and normal+optimal arterial elasticity groups. No significant differences were observed in age, sex, BMI, or stress level between the two groups (p > 0.05) (**Table 1**). These findings indicate that the participants were relatively homogeneous across arterial elasticity categories.

The study results found that 41 (51.2%) participants had suboptimal arterial elasticity. Vascular aging can occur at a young age, a condition known as early vascular aging (EVA), caused by the accumulation of risk factors.[23] The results of studies in the 20-30 age group show that metabolite levels, such as histidine and beta-alanine, are positively associated with arterial elasticity as measured by carotid-femoral Pulse Wave Velocity (cfPWV). This indicates that metabolic changes can contribute to EVA and confirms that arterial elasticity can decrease subclinically from a young age.[24]

The majority of caffeine consumption among participants was in the moderate category, with an average of 166.79 mg/day, ranging from 0 to 619.91 mg/day. This finding is in line with a study of 103 first-year medical students in Perak, Malaysia, which showed that 86% of participants consumed caffeine to stay alert during lectures or when they had to study at night.[25] Similarly, a study among college undergraduates reported an average caffeine intake of approximately 159 mg/day, with most students consuming caffeine to enhance mood and cognitive performance. In that study, coffee and tea were identified as the primary sources of caffeine, while energy drink consumption was less common. In the present study, coffee was the primary source of caffeine consumption. This data is in line with data from the Indonesian Ministry of Agriculture, which shows a 15.8% increase in coffee consumption in Indonesia from 2019 to 2021.[4]

This study shows that the majority of participants engaged in moderate-intensity physical activity. These results are in line with the Indonesian Health Survey (SKI), which recorded that the prevalence of physical activity among individuals aged 15-24 in Indonesia was 54.8% in the moderate intensity category.[26]

Participants with high caffeine consumption had a significantly higher proportion of suboptimal arterial elasticity than those with low or moderate caffeine consumption ($p = 0.012$). This finding indicates an association between high caffeine intake and arterial elasticity status. One possible explanation is that irregular consumption of high doses of caffeine may be associated with vasoconstriction, increased blood pressure, and sympathetic nervous system activation, thereby increasing hemodynamic stress on arterial walls. These physiological responses have been suggested to be associated with reduced arterial elasticity.[27]

In contrast, several previous studies have reported that long-term regular caffeine consumption may be associated with improved endothelial function and reduced inflammation markers, such as lower CRP and E-Selectin.[28] Other studies have shown that regular consumption of 400-600 mg/day of caffeine for one week or 400 mg/day for two weeks can increase adenosine A2A receptor activity and cAMP accumulation, which contributes to improved vascular function and increased arterial elasticity.[29] Additionally, regular moderate caffeine consumption (>3 cups/day) has been reported to be associated with higher arterial elasticity compared with lower consumption (≤3 cups/day) in individuals aged 17 years.[27] These findings suggest that long-term regular (habitual) caffeine consumption, whether low, moderate, or high dose, has a positive effect on arterial elasticity by preventing inflammation and increasing adenosine A2A and cAMP activity.

However, those findings differ from the results of the present study, which showed that higher caffeine consumption was associated with a greater proportion of suboptimal arterial elasticity. This discrepancy may be explained by differences in consumption patterns. Participants with irregular caffeine consumption patterns may experience less physiological adaptation to caffeine exposure. In this condition, caffeine may act as an acute trigger through increased sympathetic nerve activity, blood pressure, and vascular response, likely because the endothelium has not yet adapted to caffeine exposure.[30] Long-term, irregular consumption of high-dose caffeine has the potential to cause persistent increases in blood pressure, oxidative stress, and endothelial dysfunction. The combination of these factors can worsen the quality of the arterial walls and reduce arterial elasticity.[27,30] These mechanisms may contribute to reduced arterial elasticity, although causal relationships cannot be confirmed due to the observational nature of this present study.

Short-term caffeine consumption appears to produce vascular responses that differ from habitual long-term regular consumption, but may resemble the effects observed in individuals without physiological adaptation to caffeine. Research by Kadek et al.,[31] shows that short-term consumption of moderate doses of caffeine (2-3 cups) can increase heart rate and blood pressure for 2 to 5 hours afterward due to caffeine's direct stimulation of the adrenal medulla to release the hormone epinephrine and block adenosine receptors. Other analyses have demonstrated an increase in systolic blood pressure of 10-20 mmHg after 60 minutes of consuming a moderate dose of caffeine (150 mg) due to the inhibitory mechanism on phosphodiesterase.[7] This exposure to high blood pressure can put mechanical stress on the vascular wall, which can cause endothelial dysfunction. This condition can further trigger the vascular remodeling process and lead to a decrease in arterial elasticity.[32] These findings suggest that short-term caffeine consumption can cause a decrease in arterial elasticity.

In this study, participants with high caffeine consumption (>300 mg/day) had a higher proportion of suboptimal arterial elasticity than those with low or moderate caffeine consumption. These results are consistent with similar studies showing that irregular exposure to high doses of caffeine can trigger a sympathetic response, increased blood pressure, and changes in vascular function that contribute to decreased arterial elasticity.[33] However, due to the cross-sectional design, these findings reflect an association rather than a causal relationship. Therefore, moderating caffeine consumption may be considered as a potential strategy to support arterial elasticity in young adults. In this study, participants with lower caffeine intake (≤ 300 mg/day) tended to exhibit normal or optimal arterial elasticity. This finding aligns with existing literature suggesting that caffeine intake up to approximately 400 mg/day is generally considered safe in healthy adults (19 years or older).[34] This restriction is important to prevent a decrease in arterial elasticity that can occur due to excessive caffeine consumption, as well as to help maintain endothelial function and overall cardiovascular health.

The results of this study show that there is no relationship between physical activity and arterial elasticity ($p = 0.093$). The lack of a significant association in this study may be explained by the cross-sectional design, which does not capture long-term exposure to physical activity. Regular physical activity is known to improve endothelial function and enhance arterial elasticity by reducing inflammatory and oxidative stress pathways in vascular tissue, as well as increasing antioxidant enzyme levels and nitric oxide bioavailability.[35] Evidence from the literature suggests that the vascular effects of physical activity are dependent on the intensity and duration of the activity. A meta-analysis demonstrated that acute exercise does not immediately increase arterial elasticity but may produce transient improvements within 30 minutes, which return to baseline after 24 hours. The studies also show that only moderate-to-vigorous physical activity of sufficient duration (30-59 minutes) produces measurable improvements in arterial elasticity.[36] These findings indicate that short-term or sporadic physical activity may not lead to sustained vascular changes, particularly when measured outside the acute response window. Consistently, short-duration physical activity does not produce significant changes in arterial elasticity.[37] Acutely, physical activity can decrease smooth muscle tone and increase arterial compliance and arterial elasticity, as measured by PWV. These effects are temporary because they do not alter the structure of the arterial wall. However, in the long term, repeated hemodynamic exposure from regular exercise induces endothelial adaptation, increased vasodilation capacity, and increased vascular density, which permanently improves arterial elasticity.[36] This concept is supported by a longitudinal study involving male firefighters aged 20–49 years, in which participants were classified into light-, moderate-, and vigorous-intensity physical activity groups and followed over one year. Body composition, arterial elasticity, VO_2 max, and handgrip strength were assessed at baseline and follow-up. Physical activity was evaluated using the International Physical Activity Questionnaire (IPAQ). All participants were guided by the same trained examiner using standardized instructions to minimize self-reported bias. The study demonstrated improvements in arterial elasticity across all groups, with the greatest enhancement observed among those engaging in the vigorous-intensity group. Additionally, arterial elasticity was inversely associated with age, BMI, and relative fat mass, and positively associated with VO_2 max and muscle mass.[38]

Further supporting evidence comes from a study in young adults in Brazil, which found that waist circumference partially mediated the relationship between moderate-to-vigorous physical activity and arterial elasticity. This suggests that the beneficial vascular effects of physical activity may be partly driven by its role in reducing body weight and visceral fat, both of which are closely linked to reduced arterial elasticity.[39] In line with this, a study among medical students found that those with the lowest muscle mass-to-visceral fat (MVF) ratio had a 6.54-fold higher risk of suboptimal arterial elasticity than those with low or high MVF ratios.[40]

Another important consideration is the method used to assess physical activity. In this study, physical activity was measured using self-reported questionnaires, which are prone to recall bias and may not accurately capture the intensity and duration of the activity. This limitation may have contributed to the non-significant findings. In contrast, studies employing objective measurements provide stronger evidence of an association. Accelerometer-based assessments have shown a significant relationship between physical activity and arterial elasticity.[39]

Additionally, the relatively homogenous characteristics of the study population may have contributed to the lack of a significant association between physical activity and arterial elasticity. Variables such as age and BMI did not differ significantly between arterial elasticity groups, indicating limited variability in key vascular determinants.

This study has several strengths. First, restricting caffeine consumption before the measurement helped minimize its acute effects, allowing the result to better reflect participants' baseline vascular condition. Second, the relatively homogeneous characteristics of the participants reduced the influence of potential confounding factors such as age, sex, BMI, and stress level. Furthermore, focusing on young adults provides valuable insight into early vascular changes and highlights the role of modifiable lifestyle factors in vascular health.

This study also has several limitations that should be considered when interpreting the results. Physical activity was measured using a questionnaire, so the data obtained are subjective and potentially to reporting bias. This study also did not include lipid profile testing, so the possible influence of dyslipidemia on arterial elasticity could not be evaluated.

Conclusions

There is a significant relationship between caffeine consumption and arterial elasticity. Participants with high caffeine consumption had a significantly higher proportion of suboptimal arterial elasticity than those with low or moderate caffeine consumption ($p = 0.012$).

These findings suggest that excessive caffeine consumption may be associated with reduced arterial elasticity. Therefore, limitation of caffeine consumption to no more than 300 mg per day is recommended to maintain arterial elasticity and cardiovascular function. In contrast, physical activity did not show a significant relationship ($p = 0.093$). Further research is recommended involving a larger population and using more objective physical activity measurement tools, such as accelerometers or pedometers.

Conflict of Interest

The authors declare no conflicts of interest related to this article.

Ethic Statement

Written informed consent was obtained from the participant prior to data collection.

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Author Contributions

CKA: design of the study, data acquisition, interpretation of data, drafting the manuscript; NB: conceptualization, design of the study, interpretation of data, critical revision of the manuscript for important intellectual content, and final approval of the version to be published; IM: design of the study, interpretation of data; MC: design of the study, interpretation of data

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