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# Ultra-processed foods consumption and arterial elasticity among male medical students at Universitas Pembangunan Nasional Veteran Jakarta

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## Abstract

**Background :** Medical students commonly consume ultra-processed foods (UPFs) because of demanding academic schedules. Excessive UPF consumption has been associated with vascular dysfunction, but evidence on arterial elasticity in healthy young adults remains limited.

**Objective :** This study investigated the association between UPF consumption and arterial elasticity among male medical students at Universitas Pembangunan Nasional Veteran Jakarta.

**Methods :** A cross-sectional study included 50 male medical students selected by simple random sampling. UPF consumption was assessed using a semi-quantitative food frequency questionnaire and analyzed with NutriSurvey software. Arterial elasticity was measured using an Accelerated Photoplethysmograph Analyzer SA-3000P. Participants were aged ≥18 years, non-smokers, and had no diabetes mellitus, hypertension, or alcohol consumption. Associations were analyzed using a Chi-square test.

**Results :** Sugar intake from UPFs was significantly associated with arterial elasticity (p = 0.041; PR = 2,256; 95% CI = 1,153 – 4.406). In contrast, total energy, salt, and fat intake from UPFs were not significantly associated with arterial elasticity (p = 0.471, p = 0.990, and p = 0.763, respectively).

**Conclusions :** Higher sugar intake from UPFs, rather than total energy, salt, or fat intake from UPFs, was associated with arterial elasticity. Limiting sugar intake from UPFs may help preserve vascular health in young adults.

**Keywords:** arterial elasticity, ultra-processed foods, medical students

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## Introduction

Cardiovascular disease (CVD) remains the leading cause of death worldwide. In 2022, approximately 19.8 million deaths were attributed to CVD.[1] Reduced arterial elasticity is one of the earliest manifestations of vascular dysfunction that precedes the development of hypertension, atherosclerosis, and other cardiovascular diseases.[2] It is also recognized as a biomarker of early vascular aging (EVA), a condition in which vascular age exceeds chronological age.[3] Although vascular aging is typically associated with older adults, early changes in arterial elasticity have been observed in younger populations. Zheng et al.,[4] (2020) reported that 12.9% of adults under 30 years old already exhibited EVA. As these vascular changes occur before the onset of clinical CVD, identifying modifiable risk factors that contribute to reduced arterial elasticity is important for early prevention.

Among the modifiable risk factors, unhealthy diets account for an estimated 7.94 million CVD-related deaths each year.[5] Over the past three decades, the global burden of CVD has increased substantially due to a dietary shift toward high consumption of ultra-processed foods (UPF).[6,7] Ultra-processed foods are highly processed, industrially processed foods containing minimal whole food ingredients. They are typically high in additives, salt, fat, and cholesterol, while being low in dietary fiber and micronutrients.[8,9]

Several biological mechanisms may explain the association between UPF consumption and reduced arterial elasticity. Excess sodium intake promotes hypertension, which induces endothelial dysfunction and structural remodelling of the arterial wall.[10,11] High consumption of added sugars increases inflammation and oxidative stress, whereas high saturated fat levels impair endothelial nitric oxide bioavailability and reduce arterial elasticity.[3] Together, these mechanisms may accelerate vascular aging. High UPF consumption has been shown to accelerate vascular aging by up to 9.9 years among individuals in the highest consumption quintile.[12]

The consumption of UPF continues to increase because of its convenience, affordability, and palatability.[13] Young adults, particularly medical students, are more likely to consume UPF because of demanding academic schedules and limited time for preparing healthy meals.[14] A study conducted at Sam Ratulangi University reported that 36% of college students regularly consumed UPF.[15] Another study at Universitas Indonesia found that 61.2% of medical students had pre-hypertension.[11] These findings indicate that medical students may already be at risk of impaired vascular health.

Arterial elasticity can be assessed using non-invasive methods, including pulse wave velocity (PWV) and accelerated photoplethysmography (APG).[16] However, previous studies have reported inconsistent findings about the association between UPF consumption and arterial elasticity. While one study reported no significant association between UPF consumption and PWV,[17] another study found a significant relationship between weekly UPF consumption and increased PWV or reduced arterial elasticity.[18] These inconsistencies may reflect differences in study populations, dietary assessment methods, and techniques used to evaluate arterial elasticity. In addition, studies examining the association between UPF consumption and arterial elasticity measured using APG, particularly among medical students, remain limited. Previous studies have also reported sex differences in arterial elasticity. Estrogen enhances nitric oxide production, thereby enhancing endothelial function and preserving arterial elasticity.[19] Consequently, young females have higher arterial elasticity than young males.[20] Therefore, only male participants were included in the present study to minimize the potential confounding effects of sex hormones on arterial elasticity. Therefore, this study aimed to investigate

the association between UPF consumption and arterial elasticity among male medical students at Universitas Pembangunan Nasional Veteran Jakarta.

## Methods

This is an analytical observational study with a cross-sectional design. The sample size was calculated using the two-proportion hypothesis formula with $\alpha = 5\%$, $\beta = 80\%$, $P_1 = 19.7\%$,[21] and $P_2 = 39.4\%$.[22] The sample size formula estimates the required number of participants for each comparison group. Therefore, the calculated sample size was doubled, resulting in a total sample size of 50 participants.

Participants were selected using a simple random sampling method. The inclusion criteria were male medical students at Universitas Pembangunan Nasional Veteran Jakarta aged 18–22 years who engaged in moderate physical activity, had either good or poor sleep quality, experienced low-to-moderate stress levels, and provided written informed consent. Exclusion criteria included current smoking, alcohol consumption, and a history of hypertension or diabetes mellitus.

The study was conducted from August to October 2025 at the Physiology and Nutrition Laboratory, Medical Education and Research Center (MERCe), Universitas Pembangunan Nasional Veteran Jakarta. The principal researcher conducted data collection. No enumerator or research assistants were involved in the study.

The independent variable of this study was UPF consumption, which was assessed using a semi-quantitative food frequency questionnaire (SQ-FFQ). This questionnaire was adapted from a previously published SQ-FFQ developed for Indonesian university students and modified to include 40 food and beverage items classified as UPF according to the NOVA classification. This questionnaire assessed dietary intake for the last 30 days.[23] The selected food items represent commonly consumed UPFs among Indonesian young adults, making the instrument culturally appropriate for the study populations. Frequency and household portion sizes were converted to grams using the midpoint method.[24] Ultra-processed foods (UPFs) were defined according to the NOVA classification as industrial formulations made predominantly from processed ingredients and additives, with little or no intact whole foods.[8] Total UPF intake was analyzed using NutriSurvey software to estimate total energy intake (kcal), percentage of energy derived from UPFs, and intake of sugar, salt, and fat from UPFs. The percentage of energy from UPFs was calculated by dividing the energy obtained from UPFs by the recommended daily energy intake for young adult males aged 19-24 years (2650 kcal), then multiplying by 100%. As no universally accepted cut-off for UPF intake is available, participants were categorized into lower and higher UPF intake groups using the sample median percentage of energy derived from UPFs. Sugar, salt, and fat intake from UPFs were categorized according to the Indonesian Ministry of Health Recommendation as ≤50 g/day or >50 g/day, ≤2000 mg/day or >2000 mg/day, and ≤67 g/day or >67 g/day, respectively.[25]

The dependent variable was arterial elasticity, which was measured using the Accelerated Photoplethysmograph SA-3000P (Tokyo Iken Co., Ltd., Japan), a non-invasive arterial assessment device. It performs automatic internal calibration before each measurement. All assessments were performed by the same trained examiner using a standardized protocol to ensure measurement reproducibility.

Arterial elasticity was categorized as suboptimal (<30), normal (30–70), or optimal (>70) according to the manufacturer's recommended classification for the APG device,[26] which has also been used in previous studies.[27] Participants'nutritional status, sleep

quality, and stress level data were collected to describe the study population. Nutritional status was assessed using body mass index (BMI) and classified as underweight (<18.5 kg/m²), normal (18.5–22.9 kg/m²), overweight (23.0–24.9 kg/m²), obese class I (25.0–29.9 kg/m²), or obese class II (≥30 kg/m²). Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI) with Cronbach's α = 0.83, and categorized as good (≤5) or poor (>5). Stress levels were measured using the Perceived Stress Scale-10 (PSS-10) with Cronbach's α = 0.89, and categorized as low (1–14) or moderate (15–26).[25,28–31]

Descriptive analysis was conducted to examine participant characteristics. Associations between independent and dependent variables were analyzed using a Chi-square test. Because only a few participants had optimal arterial elasticity, the normal and optimal arterial elasticity categories were combined into a single category to meet the assumption of the Chi-square test. Statistical significance was set at $p < 0.05$. This study received ethical approval from the Ethics Committee of Universitas Pembangunan Nasional Veteran Jakarta (No. 290/XI/2025/KEP).

## Results

A total of 50 participants were included in this study. Most participants were 20 years old. As shown in **Table 1**, most participants had normal BMI (38%), moderate stress levels (64%), and poor sleep quality (58%).

**Table 1**. Participant characteristics

| No. | Charactheristics | Total (n = 50) |
|---|---|---|
| 1. | Age, years | 20 (18 – 22) |
| 2. | Body Mass Index, n (%) | |
| | Underweight | 3 (6.0) |
| | Normal | 19 (38.0) |
| | Overweight | 16 (32.0) |
| | Obese 1 | 7 (14.0) |
| | Obese 2 | 5 (10) |
| 3. | Stress Level, n (%) | |
| | Moderate | 32 (64.0) |
| | Low | 18 (36.0) |
| 4. | Sleep Quality, n (%) | |
| | Poor | 29 (58.0) |
| | Good | 21 (42) |

**Figure 1** illustrates that most participants exhibited suboptimal arterial elasticity (64%), followed by normal and optimal arterial elasticity. These findings indicate that reduced arterial elasticity was common in this young adult population.

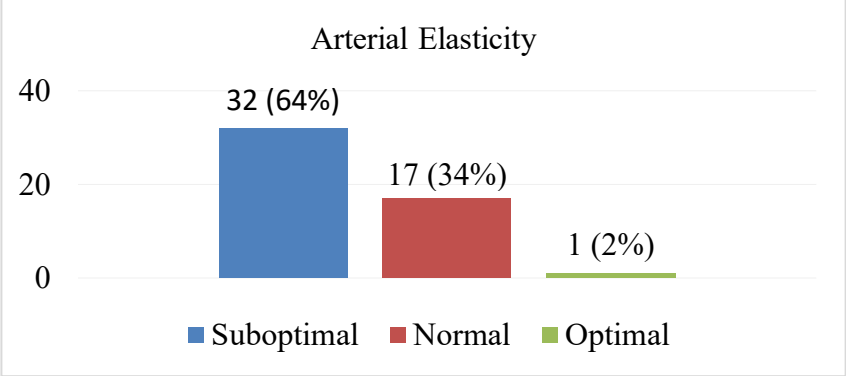


**Figure 1.** Arterial elasticity distribution among participants

**Table 2** presents UPF intake among participants. Overall, participants consumed an average of 1327 ± 529.1 kcal/day from UPFs, accounting for approximately 50 ± 20% of total energy intake. The most consumed UPF foods were instant noodles and fried chicken. Meanwhile, the most-consumed UPF beverages were fruit-flavored, carbonated, and chocolate malt beverages.

**Table 2**. Energy and Nutrient Intake from UPF among Participants

| No. | Energy and Nutrient Intake | Mean ± SD / Median (Min -Max) |
|---|---|---|
| 1. | Energy (kcal) | 1327 ± 529.1 |
| 2. | % UPF Consumption | 50 ± 20 |
| 3. | Gram of UPF (g) | 490.3 ± 206.7 |
| 4. | Sugar (g) | 24.5 (0.9 – 137.4) |
| 5. | Salt (mg) | 1746.1 (560.6 – 5516.1) |
| 6. | Fat (g) | 58.7 (19.5 – 180.7) |
| 7. | Carbohydrate (g) | 116.6 (30 – 331.4) |
| 8. | Protein (g) | 73 ± 36.1 |
| 9. | Cholesterol (mg) | 83.7 (35.9 – 1653.5) |

Note: Normally distributed data are presented as mean ± SD and non-normally distributed data are presented as Median (Min -Max)

The median sugar intake derived from UPF was 24.5 g, with a wide range from 0.9 to 137.4 g, shown in Table 2. Similarly, the median salt intake from UPF was 1746.1 mg (range 560-5516.1 mg), corresponding to approximately 75.9 mmol of sodium. In addition, the median fat intake from UPF was 58.7 g, ranging from 19.5 to 180.7 g.

**Table 3** shows there was no association between the percentage of energy derived from UPFs and arterial elasticity (PR = 0.803; p = 0.471). In contrast, sugar intake from UPFs demonstrated a significant association with arterial elasticity. Participants with sugar intake >50 g/day had a higher proportion of suboptimal arterial elasticity (63.6%) compared to those within the recommended intake or ≤50 g/day (28.2%), with a prevalence ratio indicating more than a twofold increased risk (PR = 2.256; 95% CI = 1.153-4.406; p = 0.041). Meanwhile, salt and fat intake from UPFs were not statistically significant (salt: PR = 1.131; p = 0.990; fat: PR 1.166; p =0.763).

**Table 3.** Association of energy, sugar, salt, and fat intake from ultra-processed foods with arterial elasticity

| Variables | Arterial Elasticity | | | | Total | | PR (95% CI) | p-value |
|---|---|---|---|---|---|---|---|---|
| | Suboptimal | | Normal + Optimal | | | | | |
| | N | % | N | % | N | % | | |
| **Percentage of energy derived from UPFs** | | | | | | | | |
| Higher (> median) | 13 | 56.6 | 10 | 43.5 | 23 | 100 | 0.803 | 0.471 |
| Lower (≤ median) | 19 | 70.4 | 8 | 29.6 | 27 | 100 | | |
| **Sugar intake from UPFs** | | | | | | | | |
| >50 g/day | 7 | 63.6 | 4 | 34.4 | 11 | 100 | 2.256 (1.153 – 4.406) | 0.041* |
| ≤50 g/day | 11 | 28.2 | 28 | 71.8 | 39 | 100 | | |
| **Salt intake from UPFs** | | | | | | | | |
| >2,000 mg/day | 7 | 38.9 | 11 | 61.1 | 18 | 100 | 1.131 | 0.990 |
| ≤2,000 mg/day | 11 | 34.4 | 21 | 65.6 | 32 | 100 | | |
| **Fat intake from UPFs** | | | | | | | | |
| >67 g/day | 6 | 42.9 | 8 | 57.1 | 14 | 100 | 1.166 | 0.763 |
| ≤67 g/day | 12 | 33.3 | 24 | 66.7 | 36 | 100 | | |

Note: significant p<0.05, Chi-square test

## Discussion

This study involved 50 male medical students aged 18 – 22 years, representing a young adult population generally considered to have a low risk of cardiovascular disease. Although arterial aging is typically present in older age, early alterations in arterial elasticity may occur in young adults due to risk factors such as hyperglycemia, adiposity, smoking, and lifestyle behaviors.[32] Decreased arterial elasticity is closely related to structural changes, particularly increased tunica intima thickness and endothelial dysfunction.[33]

The participants' characteristics in this study indicate the presence of several risk factors that may affect vascular health. Regarding nutritional status, more than half (56.0%) were classified as overweight or obese. This high proportion of excess body weight is consistent with findings among Indonesian university students, whereas overweight and obesity rates exceed 30%.[34] Increased adiposity has been associated with chronic inflammation, which contributes to reduced arterial elasticity.[35]

The majority of participants experienced moderate stress levels (64%), consistent with findings among medical students at Universitas Nusa Cendana (46,8%).[36] Medical students experienced heightened stress levels due to long study hours and high academic workload. Chronic stress may elevate cortisol levels and alter arterial tone, thereby decreasing arterial elasticity.[37]

Poor sleep quality was also common, affecting 58% of participants, which is comparable to the prevalence among medical students at Universitas Yarsi (57%).[38] The high prevalence of poor sleep quality is caused by high academic pressure, fear of failure, and hectic exam schedules. Inadequate sleep has been linked to increased sympathetic nervous system activity and oxidative stress, thereby impairing arterial elasticity.[39]

The participants' risk profile in this study was reflected in arterial elasticity findings, with 66% showing suboptimal elasticity despite their young age. This indicates the

presence of early vascular aging, likely influenced by dietary patterns, lifestyle, excess adiposity, psychological stress, and poor sleep quality.[27,40]Participants consumed an average of 1327 ± 529.1 kcal/day from UPFs. The majority of participants (54%) were classified as having lower total energy derived from UPF intake. This finding is consistent with a study reporting that UPFs contributed an average of 15.7% total energy intake among residents in Jakarta.[41]

Previous studies involving adults aged 18–45 years found no association between UPF consumption, accounting for approximately 50% of total daily energy intake, and arterial elasticity.[17] In the present study, although a higher proportion of participants had suboptimal arterial elasticity in the higher UPF consumption group than in the lower UPF consumption group, the difference was not statistically significant (56.5% vs. 43.5%; p = 0.471). Nevertheless, evidence from cohort studies has shown that each 10% increase in UPF intake raises CVD risk by 12%.[42] Ultra-processed foods are designed to be hyperpalatable, with high sugar, salt, and fat content, thereby stimulating the brain's reward system and food-regulation pathways.[43] This leads to increased appetite and reduced satiety responses, causing the body to consume more calories. The high content of processed carbohydrates, added sugars, salt, and saturated fat in UPF can cause metabolic and inflammatory disorders that can decrease arterial elasticity.[44] Consistent with these mechanisms, a cohort study found that high UPF consumption was significantly associated with cardiovascular disease even after adjusting for total energy intake.[45] Similarly, a meta-analysis study found that the adverse effects of UPF consumption were related to its high content of added sugar, salt, saturated fat, and low content of protective nutrients compared to the high total calories from UPF consumption.[46] These findings imply that the nutritional quality of UPFs may have a greater influence on cardiovascular health than energy intake per se.

The median sugar intake from UPFs in this study was 24.5 g (range: 0.9–137.4 g). Most participants (78%) consumed sugar within the recommended daily limit (≤50 g/day) established by the Indonesian Ministry of Health. Despite this, sugar intake from UPFs was significantly associated with arterial elasticity (p = 0.041). Participants with sugar intake >50 g/day had a 2.256 times higher prevalence of developing suboptimal arterial elasticity than those consuming ≤50 g/day (PR = 2.256; 95% CI: 1.153 – 4.406). This finding is consistent with a study in healthy adults showing that consumption of 20–25 g of glucose decreases brachial–ankle pulse wave velocity (baPWV), a marker of arterial elasticity, whereas no decrease in baPWV occurs after consuming 15 g of glucose.[47]

High sugar intake may induce postprandial hyperglycemia, which can reduce nitric oxide bioavailability. This effect is mediated by increased arginine catabolism, leading to the accumulation of asymmetric dimethylarginine (ADMA), a competitive inhibitor of endothelial nitric oxide synthase (eNOS).[48] Decreased nitric oxide bioavailability may enhance arterial matrix metalloproteinase (MMP) activity. Activation of MMP-2 and MMP-9 promotes elastin degradation, collagen deposition, and arterial smooth muscle cell migration within the arterial wall. These structural arterial alterations contribute to decreased arterial elasticity.[49] These biological pathways were not directly assessed in the present study. Therefore, further longitudinal and mechanistic studies are needed to confirm this association. The proposed mechanism by which glucose affects arterial elasticity is illustrated in **Figure 2.**

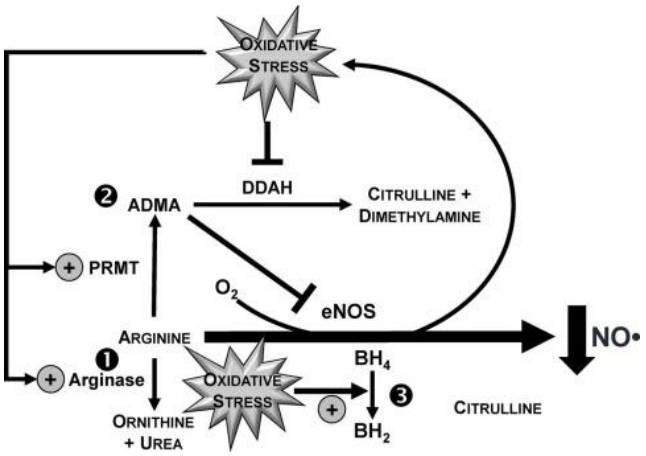


**Figure 2**. Mechanism of the Effect of Glucose on Arterial Elasticity[48]

These findings suggest the importance of limiting sugar intake from UPFs, even among apparently healthy young adults. Intervention through nutrition education and healthy food policies in universities may help preserve vascular health and reduce future cardiovascular risk.

The median salt intake from UPFs was 1746.1 mg (range: 560 – 5516.1 mg), equivalent to 75.9 mmol of sodium. Most participants (64%) consumed salt within the Indonesian Ministry of Health recommendation of ≤2,000 mg/day. In contrast, a national survey found that the average daily salt intake of Indonesians was 2,746 mg/day.[50]

A study conducted in healthy adults reported that an average salt intake of 1498 mg was positively associated with increased peripheral blood pressure but showed no association with PWV, a marker of arterial elasticity.[17] In the present study, the proportion of suboptimal arterial elasticity in the group with salt intake >2,000 mg/day was higher than that in those with salt intake ≤2,000 mg/day; however, the difference was not statistically significant (38.9% vs. 34.4%; p = 0.990).

Excessive salt intake may impair vascular function through several mechanisms. High salt intake can increase plasma volume and systemic vascular resistance, leading to increased blood pressure and decreased arterial wall elasticity.[51] In addition, excess salt can also increase superoxide ($O^{2-}$), which reacts directly with nitric oxide and inhibits its diffusion into vascular smooth muscle, preventing vasodilation and increasing blood pressure. Salt also disrupts the endothelial glycocalyx, which can interfere with vascular shear stress mechanotransduction and cause endothelial dysfunction. Intake of more than 150 mmol (3450 mg) of sodium can also increase epithelial sodium channel (eNaC) access, leading to increased intracellular sodium and decreased arterial elasticity.[52] In the present studies, these biological pathways were not assessed, so they should be considered as potential mechanisms.

However, these adverse vascular effects are more pronounced at substantially higher sodium intake. Consistent with this, a meta-analysis study found no significant association between usual salt intake and vascular structure as measured by carotid-femoral pulse wave velocity (cf-PWV).[51] In contrast, another meta-analysis reported that salt intake > 5000 mg per day was associated with reduced arterial elasticity.[53]

The median fat intake from UPF was 58.7 g (range: 19.5 – 180.7 g). One study demonstrated that consumption of high-fat meals (50 – 105 g of fat) could reduce arterial reactivity and flow-mediated dilation (FMD) due to reduced nitric oxide bioavailability.[54]

The majority of participants (72%) had fat intake categories in line with Ministry of Health recommendations (≤67 g/day). The proportion of suboptimal arterial elasticity was higher in the group with fat intake >67 g/day compared to the group with fat intake ≤67 g/day, although this difference was not statistically significant (42.9% vs. 33.3%; p = 0.763). Previous studies have suggested that high-fat foods are rich in saturated fatty acids (SFA) and trans fatty acids (TFA), which increase inflammation and oxidative stress. Fat consumption can increase postprandial triglyceride and lipoprotein levels, which decrease nitric oxide concentrations. Increased oxidative stress and decreased nitric oxide can disrupt the vascular endothelium through increased matrix metalloproteinase (MMP) activation. Matrix metalloproteinases increase elastin degradation and vascular smooth muscle cell migration, thereby decreasing arterial elasticity.[54]

Previous studies have reported inconsistent findings regarding the relationship between total fat intake and cardiovascular health. The Prospective Urban Rural Epidemiology (PURE) study, which included adults aged 35-70 years from 18 countries, found that total fat consumption and fat types such as SFA and TFA were not significantly associated with cardiovascular disease and mortality.[55] Similarly, a meta-analysis of 43 prospective cohort studies reported no significant associations between total fat, SFA, monounsaturated fatty acids (MUFA), or polyunsaturated fatty acids (PUFA) and cardiovascular disease.[56] In addition, the Tehran Lipid and Glucose Study found no significant association between total fat or individual fatty acids and incident cardiovascular disease after adjustment for potential confounders.[57] These findings should be interpreted cautiously because of methodological differences across studies. Previous studies primarily involved middle-aged and older adults and evaluated long-term cardiovascular outcomes, whereas the present study included healthy young adults and assessed arterial elasticity as an early marker of vascular dysfunction. Therefore, the absence of a significant association between total fat intake from UPFs and arterial elasticity does not necessarily contradict previous evidence.

Several limitations of this study should be acknowledged. First, overall dietary patterns were not assessed. There, the contribution of overall dietary quality to arterial elasticity could not be determined. Second, lipid profiles were not assessed, so the potential influence of undiagnosed dyslipidemia on arterial elasticity could not be excluded. Third, the small sample size limited the use of multivariable regression analysis to adjust for potential confounders, such as BMI and stress level. Fourth, the small sample size, involving only male medical students from a single university, may limit the external validity of the findings.

## Conclusions

This study shows that higher sugar intake from UPFs was significantly associated with suboptimal arterial elasticity. However, no significant associations were observed between total energy, salt, or fat intake from UPFs and arterial elasticity. These findings suggest that limiting sugar intake from UPFs may support vascular health.

## Conflict of interest

All authors declare no conflicts of interest

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## Author Contributions

ASM: 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; EH: design of the study, interpretation of data; MD: design of the study, interpretation of data

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