

## Evaluating the Acute Effects of Energy Drink Consumption on Arterial Stiffness in Healthy Young Adults

### ABSTRACT

**Background:** Energy drinks are popular among adolescents and young adults for their perceived benefits in enhancing energy, alertness, and performance. These beverages often contain high levels of caffeine, sugar, and various stimulants, which may have acute effects on cardiovascular health. Arterial stiffness, assessed by augmentation index (Alx), is a key indicator of cardiovascular health and can be influenced by these beverages. This study aimed to evaluate the acute effects of energy drink consumption on arterial stiffness in healthy young adults.

**Methods:** In this cross-sectional study, 64 healthy subjects with normal blood pressure (BP) and no cardiovascular or metabolic disorders participated. After a baseline measurement of vascular and hemodynamic parameters, participants consumed a can of energy drink. Measurements of arterial stiffness, including Alx, pulse wave velocity (PWV), and central BP, were taken at baseline and then 30 minutes and 2 hours post consumption using a validated non-invasive system.

**Results:** Significant decreases in diastolic BP at both peripheral and central were observed at 2 hours compared to baseline ( $P = .011$  and  $P = .020$ , respectively). Total vascular resistance decreased notably over time ( $P = .044$ ). While stroke volume, cardiac output, cardiac index, heart rate, PWV, augmentation pressure, and coefficient of reflection remained stable, significant transient increases in Alx and Alx@75 were recorded at 30 minutes after energy drink consumption ( $P = .016$  and  $P = .005$ , respectively), returning to baseline values by 2 hours, indicating a transient but notable effect on arterial stiffness.

**Conclusion:** This study underscores the need for caution regarding energy drink consumption, particularly among young people, due to its acute effects on arterial stiffness. Further research is warranted to explore the long-term effects and underlying mechanisms of energy drinks on cardiovascular health.

**Keywords:** Energy drinks, arterial stiffness, augmentation index, cardiovascular health, pulse wave velocity

### INTRODUCTION

Energy drinks are widely consumed by adolescents and young adults, often as a quick pick-me-up during busy schedules, late-night studying, or before sports activities. They are marketed as beverages that can boost energy levels, increase alertness, and enhance physical and mental performance. The convenience and perceived benefits make them attractive options. Most energy drinks contain caffeine, often in high amounts compared to other beverages. They may also include sugars, vitamins, amino acids (like taurine), and herbal extracts (such as guarana).<sup>1</sup> The combination of these ingredients can have acute physiological effects on the cardiovascular system.<sup>2</sup> Also, excessive consumption or mixing with alcohol can pose even greater risks.

Arterial stiffness is associated with aging and various cardiovascular risk factors.<sup>3</sup> The augmentation index (Alx) is a new parameter used in the field of cardiovascular medicine to assess arterial stiffness and wave reflection in the arterial system. The Alx is a measure of the extent to which the central arterial pressure wave is augmented by wave reflections from the periphery. It is influenced by the

### ORIGINAL INVESTIGATION

Salim Yaşar<sup>1</sup> 

Muhammed Geneş<sup>2</sup> 

Özkan Eravcı 

Ahmet Arslan<sup>3</sup> 

Hülya Şirin<sup>3</sup> 

Suat Görmel<sup>1</sup> 

Serdar Fırtına<sup>1</sup> 

Mehmet Sadık Karpat<sup>1</sup> 

Cem Barçın 

Murat Çelik<sup>1</sup> 

<sup>1</sup>Department of Cardiology, Gülhane Training and Research Hospital, University of Health Sciences, Ankara, Türkiye

<sup>2</sup>Department of Cardiology, Sincan Training and Research Hospital, Ankara, Türkiye

<sup>3</sup>Department of Public Health, University of Health Sciences, Gülhane School of Medicine, Ankara, Türkiye

**Corresponding author:**

Salim Yaşar

✉ dr.salimyasar@hotmail.com

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interaction between forward and reflected pressure waves in the arterial system. It is commonly calculated using arterial pressure waveforms obtained from non-invasive methods. A higher Alx indicates increased arterial stiffness and enhanced wave reflections, which are often associated with aging, hypertension, and other cardiovascular risk factors. Conversely, a lower Alx is generally associated with better arterial compliance and reduced wave reflections, which are indicative of a healthier cardiovascular system.

There is growing concern about the potential health risks associated with energy drink consumption. High caffeine and sugar intake may have adverse health effects such as increased heart rate, anxiety, weight gain, and sleep disturbances. While some studies have suggested potential acute increases in blood pressure associated with energy drink consumption, there is not a well-established and widely recognized direct relationship between the consumption of energy drinks and the Alx.

The aim of this study was to evaluate the acute effects of energy drink consumption on Alx as a measure of arterial stiffness in healthy young adults.

## METHODS

### Study Population

This is a cross-sectional pilot study, with the participation of 64 healthy subjects. All had normal blood pressure (BP) and did not have established any cardiovascular disease, diabetes, thyroid disease and hyperlipidemia. Subjects who experienced an increase in BP as a "stressful" response during the visit were excluded from the study to avoid a white coat effect. Furthermore, subjects with allergies to beverage ingredients and pregnancy and breast-feeding excluded female participants. Based on self-reporting, none of the participants used caffeine-containing energy drinks or caffeine-containing medications.

The study protocol conforms to the ethical guidelines of the Declaration of Helsinki and was approved by the authors' Institutional Research Ethics Committee (Decision date: September 14, 2022, Decision number: 2022/126). All subjects gave written informed consent. In this study, methods such as artificial intelligence-supported technologies (large

language models, etc.), chatbots, or image generators were not used.

### Study Design

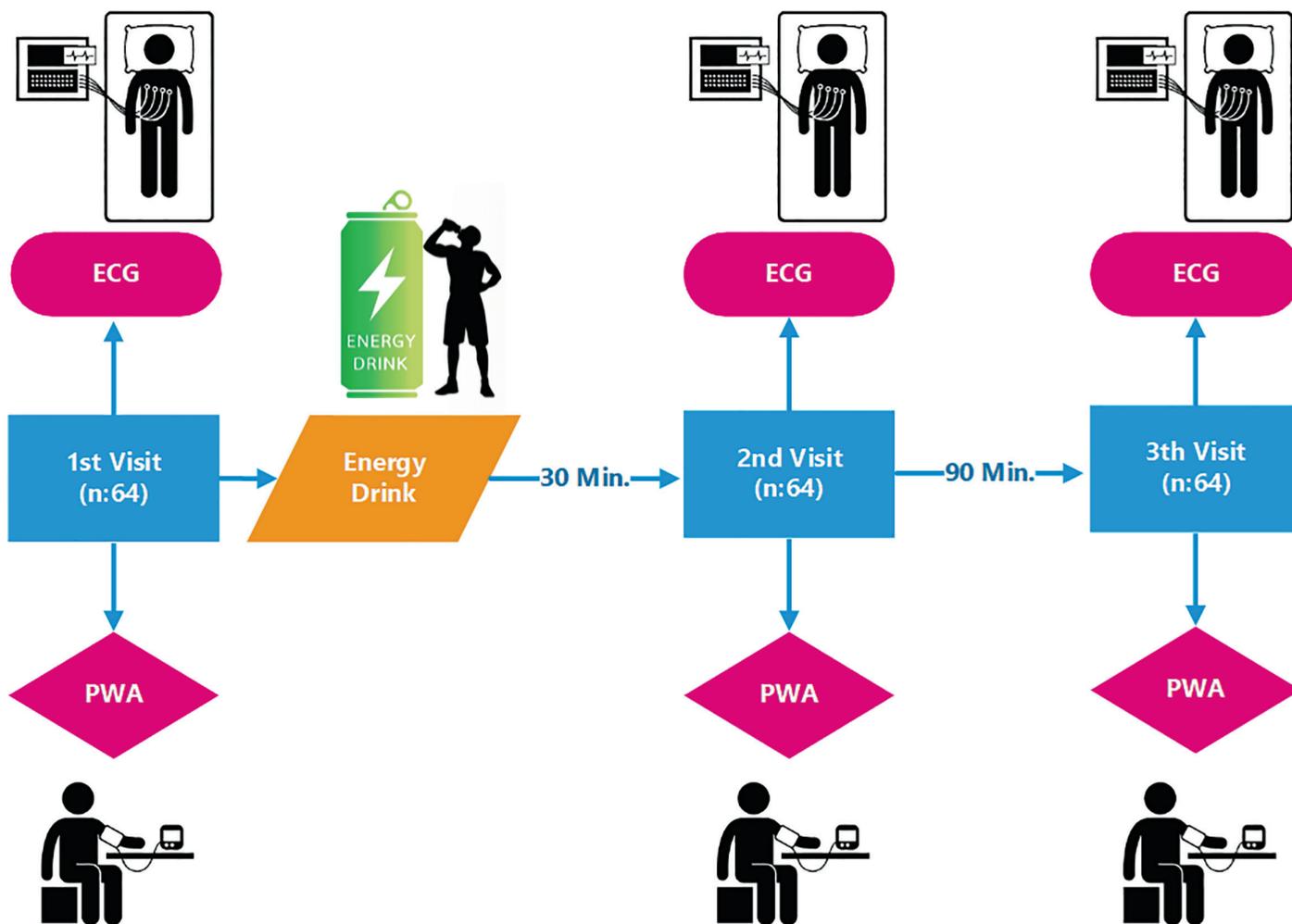
To avoid circadian-related BP differences, all measurements were performed in the morning after a light breakfast between 08:00 and 09:00 AM, in a quiet, temperature-controlled room at 20–23°C. It was recommended that all participants to avoid caffeinated products such as tea, chocolate, or cola drinks, and alcohol for at least 48 hours prior to the study date and on the day of the study. After the subjects were informed about the protocol, they underwent physical examinations. The variables investigated were anthropometric data, vascular and hemodynamic parameters, and arterial stiffness indices. A baseline measurement was obtained while the subject sitting after a minimum rest of 5 minutes. After baseline measurements, all subjects were assigned to consume 250 mL of a commercially available energy drink (containing water, 27.5 g of sugar, vitamin B3, vitamin B5, vitamin B6, and vitamin B12, and a proprietary energy blend of taurine (200 mg), sodium bicarbonate, magnesium carbonate, caffeine (37.5 mg), and glucuronolactone). The contents listed were obtained from the manufacturer's ingredient list and were not verified through independent analysis. All measurements were repeated 30 minutes, and 2 hours after energy drink consumption (Figure 1).

### Vascular and Hemodynamic Parameters and Arterial Stiffness Indices

Brachial Cuff-Based Method was performed using the validated, commercially available system "Mobil-O-Graph® device – Pulse Wave Analysis Monitor" (Mobil-O-Graph, IEM, Germany) with the ARCSolver method (Austrian Institute of Technology, Vienna, Austria) and HMS CS 4.2; I.E.M (Hypertension Management System Client Server) software, which allows recording of central pressures and arterial stiffness parameters as transmitted.<sup>4</sup> It is approved by The European Society of Cardiology (ESC) and Food and Drug Administration (FDA).<sup>5</sup> Briefly, Mobil-O-Graph uses the Oscillometric technique with a standard BP cuff in the brachial artery. Peripheral arterial and central arterial pressure and mean arterial pressure (MAP) were evaluated. Central systolic BP was determined from brachial waveforms, recorded with the cuff at the level of diastolic BP ( $\pm 5$  mmHg) for approximately 10 seconds using a high-accuracy pressure sensor.<sup>4,6</sup> Using the derived central waveforms, pulse waveform analysis was performed and indices of arterial stiffness such as pulse wave velocity (PWV), Alx, and augmentation pressure were estimated. Pulse wave velocity is a direct measure of arterial stiffness of the large arteries. The Alx75 was calculated by means of the pressure difference between the reflection wave peak (P2) and the incident wave peak (P1), expressed as the pulse pressure (PP) percentage [ $Alx75 = (P2 - P1) / PP \times 100$ ]. As the Alx is influenced by the heart rate, this index is normalized for a heart rate at 75 bpm (Alx@75) in accordance with Wilkinson et al.<sup>7</sup> In addition to the indices of arterial stiffness, the hemodynamic parameters evaluated were systolic volume (SV), cardiac output (CO), cardiac index (CI), and total vascular resistance (TVR). Two measurements 5 minutes apart were made and the average between them

## HIGHLIGHTS

- It is important for individuals, especially young people, to be informed about the potential impact of energy drinks on their cardiovascular health.
- The importance of understanding the long-term effects of energy drink consumption on cardiovascular health, beyond just acute increases in BP.
- Exploring the specific mechanisms through which caffeine, taurine, and glucuronolactone may impact arterial stiffness and endothelial dysfunction.
- Considering public health implications and policy recommendations based on the findings of studies investigating energy drink effects on arterial stiffness and BP.



ECG: Electrocardiography, PWA: Pulse Wave Analyses

Figure 1. Study design flow chart

was considered for the final analysis. These mean values were used as references for statistical analyses.

### Statistical Analysis

After all data were collected, they were statistically analyzed using the Statistical Package for Social Sciences 25.0 for Windows (SPSS Inc., Chicago, Ill, USA). The Kolmogorov-Smirnov test was used to analyze the normality of the data. Continuous data are expressed as means  $\pm$  SD. A repeated-measures analysis of variance was used to assess differences between variables before and after energy drink consumption. Categorical variables were described as percentages and analyzed by Fisher's exact test. Correlations of indices of arterial stiffness with the different variables studied were performed by the Pearson or Spearman correlation coefficient, when indicated. Statistical significance was assumed to be present at a 2-sided  $P$  value of  $<.05$ .

## RESULTS

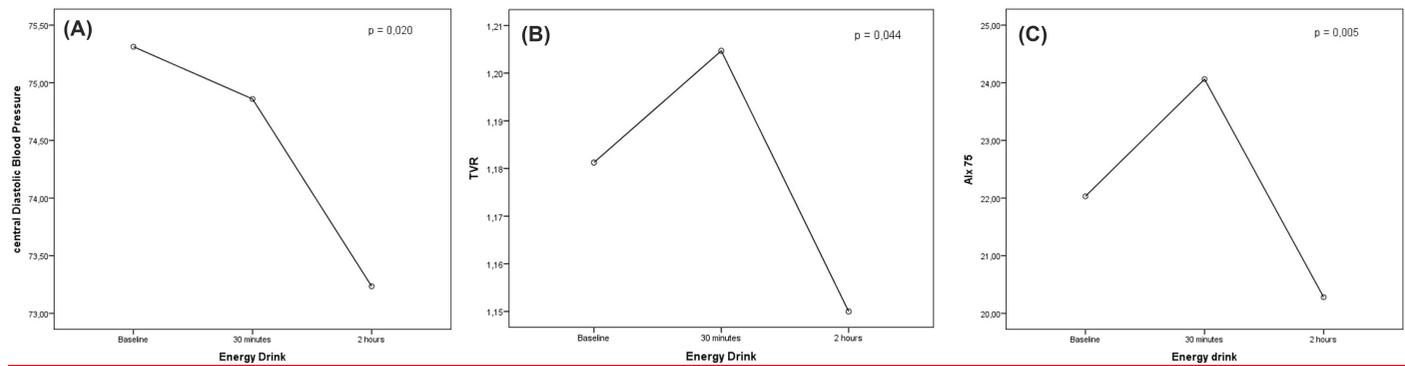
### Baseline Characteristics

Sixty-four participants (mean age  $23.40 \pm 2.58$  years, 32 (50%) males; mean body mass index  $22.78 \pm 3.27$  kg/m<sup>2</sup>) were enrolled in the study. All participants were healthy, had no

remarkable medical history, and no abnormalities were found in baseline physical examination. No unexpected side effect occurred during the study and all 64 volunteers successfully completed the study.

### Vascular, Hemodynamic, and Arterial Stiffness Indices

Regarding the vascular variables, it was observed that DBPp and DBPc were significantly lower over time, with the 2-hour measurement showing a notable drop compared to baseline ( $P = .011$  and  $P = .020$ , respectively) (Figure 2A). The other vascular parameters did not differ between groups. No statistically significant change was observed in other vascular parameters after energy drink consumption. Regarding hemodynamic data, the authors observed a significant TVR decrease over time, with the 2-hour measurement showing a notable drop compared to baseline ( $P = .044$ ) (Figure 2B). The other hemodynamic parameter did not change over time. Augmentation index and  $AIx@75$  showed significant changes, particularly at the 30-minute after energy drink consumption, indicating transient changes in arterial stiffness ( $P = .016$  and  $P = .005$ , respectively) (Figure 2C). The transient changes in arterial stiffness metrics suggest dynamic adjustments in arterial function, with a notable effect at 30



**Figure 2. The change in central aortic blood pressure parameters after energy drink consumption, (A) central diastolic blood pressure, (B) total vascular resistance, (C) AIX@75.**

minutes but returning to baseline levels by 2 hours. However, PWV, augmentation pressure, and coefficient of reflection did not change significantly, indicating that other measures of arterial stiffness and reflection were stable (Table 1).

The data indicate that while systolic pressures and some hemodynamic parameters remained stable over time, there were some dynamic changes in BP and vascular resistance over time, with specific changes in diastolic pressures and arterial stiffness parameters.

**DISCUSSION**

Despite their popularity, multiple studies have shown that there are health concerns because of reported cardiovascular side effects of energy drink such as arterial hypertension

and arrhythmia.<sup>8,9</sup> These adverse effects have been mostly attributed to the caffeine, taurine, and glucuronolactone content.<sup>10</sup> However, the effects of ED consumption on arterial stiffness and endothelial dysfunction, which could indicate subclinical end-organ damage, remain inconclusive.<sup>9,11</sup> On the other hand, Javidan Akhundova et al<sup>12</sup> have shown that energy drinks containing low doses of caffeine do not affect BP, heart rate, and endothelial function in healthy individuals. Because of conflicting results, this study aimed to assess the transient effects of energy drink consumption on BP on arterial stiffness.

Healthy large arteries, such as the aorta, carotid arteries, and cervical arteries, have a strong cushioning function. Arterial stiffness describes the rigidity of the walls of large

**Table 1. Effects of Energy Drink Consumption on Peripheral and Central Arterial Pressure, Hemodynamics, and Arterial Stiffness Over Time**

|                                     | Baseline       | 30 minutes     | 2 hours        | P           |
|-------------------------------------|----------------|----------------|----------------|-------------|
| <b>Peripheral arterial pressure</b> |                |                |                |             |
| SBP, mm Hg                          | 119.15 ± 11.54 | 118.89 ± 12.55 | 119.18 ± 11.58 | .973        |
| DBP, mm Hg                          | 73.71 ± 9.07   | 73.09 ± 7.84   | 70.95 ± 8.63   | <b>.011</b> |
| MAP, mm Hg                          | 94.51 ± 9.05   | 94.06 ± 8.53   | 93.04 ± 8.49   | .203        |
| PP, mm Hg                           | 45.40 ± 9.76   | 45.79 ± 11.38  | 47.84 ± 10.83  | .249        |
| <b>Central blood pressure</b>       |                |                |                |             |
| SBPc, mm Hg                         | 106.45 ± 10.11 | 106.37 ± 10.52 | 105.78 ± 9.40  | .807        |
| DBPc, mm Hg                         | 75.31 ± 8.88   | 74.85 ± 8.01   | 73.23 ± 8.59   | <b>.020</b> |
| PPc, mm Hg                          | 30.98 ± 7.19   | 31.51 ± 8.95   | 32.56 ± 7.55   | .389        |
| <b>Hemodynamics</b>                 |                |                |                |             |
| Stroke volume                       | 60.85 ± 14.05  | 59.78 ± 12.00  | 62.77 ± 14.89  | .157        |
| CO                                  | 4.87 ± 0.72    | 4.78 ± 0.75    | 4.96 ± 0.83    | .150        |
| CI                                  | 2.75 ± 0.36    | 2.70 ± 0.35    | 2.79 ± 0.36    | .169        |
| Heart rate, bpm                     | 82.42 ± 12.61  | 81.51 ± 11.98  | 80.90 ± 11.19  | .513        |
| TVR                                 | 1.18 ± 0.16    | 1.20 ± 0.16    | 1.15 ± 0.15    | <b>.044</b> |
| <b>Arterial stiffness</b>           |                |                |                |             |
| Alx                                 | 17.76 ± 7.61   | 20.25 ± 8.14   | 17.21 ± 8.27   | <b>.016</b> |
| Alx@75                              | 22.03 ± 9.74   | 24.06 ± 7.80   | 20.28 ± 10.23  | <b>.005</b> |
| PWV                                 | 4.99 ± 0.39    | 4.95 ± 0.45    | 4.97 ± 0.39    | .750        |
| Augmentation pressure (mm Hg)       | 7.20 ± 3.89    | 7.98 ± 5.48    | 7.29 ± 4.24    | .480        |
| Coefficient of reflection, (%)      | 53.57 ± 8.70   | 55.20 ± 9.36   | 55.35 ± 8.51   | .222        |

The statistically significant values in the table are indicated in bold.

arteries and disruption of this cushioning function.<sup>3</sup> In brief, the aortic pulse wave is a combination of the forward pressure wave from left ventricular ejection and the reflected wave caused by impedance mismatches in the arterial tree. The forward wave depends on the mechanical properties of large central arteries, while the reflected wave is influenced by the overall elasticity of the arterial tree, wave velocity, and distance to reflection points. In young or elastic vessels, the reflected wave occurs during diastole, aiding coronary perfusion and having little impact on cardiac afterload during systole. Arterial stiffness is increased wave reflections and their pathophysiological manifestations are increased systolic BP, decreased diastolic BP, and therefore increased PP (especially central pulse pressure (PPc)).<sup>13,14</sup>

Pulse wave velocity, central BP, and Alx are markers of arterial stiffness, which is associated with cardiovascular morbidity and mortality.<sup>15</sup> Central BP is more relevant to cardiovascular outcomes than the peripheral BP.<sup>16</sup> The hemodynamic parameters evaluated were stroke volume (SV), cardiac output (CO), and TVR.

The central arterial BP was derived with the use of a generalized transfer function, which is a validated estimate of the central arterial pressure waveform. Central pulse wave analysis allows measurement of the Alx (Alx@75), the central systolic BP (SBPc), and the PPc (PPc as the difference between SBPc and SBPp). The stiffness-induced increase in PPc was shown to be an independent predictor of cardiovascular risk.<sup>17</sup> The peripheral PP (PPp) also considered a marker of arterial stiffness. The PPp/PPc ratio, also known as pulse pressure amplification (PPA), is an important physiological function to protect the heart against afterload and microcirculation from pulsatile pressure stress.<sup>18</sup>

The Alx and PWV are the most important indicators of arterial stiffness obtained from non-invasive methods and have been currently considered independent predictors of major CV events and all-cause mortality.<sup>15,19</sup> The Alx and PWV provide insights into the wave reflections in the arterial system, which can impact BP and cardiac workload. While PWV is a direct measure of arterial distensibility, the Alx is a more complex parameter depending on vascular elasticity and peripheral resistance.<sup>20</sup> The Alx is calculated by measuring the augmentation pressure, which is the difference between the second and first systolic peaks of the arterial pressure waveform and dividing it by the PP. It is often expressed as a percentage. The Alx at 75 beats per minute (Alx@75) is a specific value calculated under standardized conditions, usually representing the Alx normalized to a heart rate of 75 beats per minute. This normalization provides valuable information about arterial health and cardiovascular risk and allows for better comparability between individuals and studies. Augmentation index at 75 beats per minute is often used in clinical research and medical practice. Elevated Alx and Alx@75 values are markers of increased cardiovascular risk, indicating compromised arterial function, higher arterial stiffness, and increased wave reflections, which are commonly linked

to aging, hypertension, and other cardiovascular risk factors.<sup>21</sup> Conditions such as hypertension, atherosclerosis, and aging are known to contribute to arterial stiffening, thereby affecting the augmentation index. Conversely, interventions that enhance arterial elasticity, such as lifestyle changes and specific medications, can lower Alx@75, reflecting improved arterial compliance and reduced wave reflections, which signify a healthier cardiovascular system. Furthermore, thresholds for Alx may vary based on the measurement method and the population being studied. The Alx@75 is a useful measure of arterial function but should be interpreted alongside an individual's overall health, medical history, and other cardiovascular risk factors for a complete assessment of cardiovascular health.

There is not a well-established and widely recognized direct relationship between the consumption of energy drinks and the Alx@75. However, some general insights into the potential cardiovascular effects of energy drinks and how they might influence parameters such as arterial stiffness, including the Alx can be suggested. Energy drinks typically contain caffeine, sugar, and other ingredients like taurine and B-vitamins. Caffeine, a central nervous system stimulant, is a primary component of many energy drinks and has been associated with changes in BP and heart rate. Caffeine is known to have acute effects on BP and heart rate, which are factors that can influence the Alx. Some studies have suggested that acute caffeine intake may lead to a temporary increase in arterial stiffness, but the overall relationship is complex and can vary among individuals. Consumption of energy drink can lead to increased sympathetic nervous system activity, potentially impacting blood vessel function. Moreover, the other components of energy drinks, such as sugar and certain additives, may also have cardiovascular effects. Excessive sugar content, for example, may contribute to metabolic issues, including insulin resistance, which is associated with cardiovascular risk factors. Also, individual responses to energy drinks can vary based on factors such as age, overall health, and tolerance to caffeine with all this, energy drinks lead to an increase in heart rate and BP, it might the timing and interaction of arterial waves, potentially altering the Alx value.

Despite the perception among young people that these energy drinks are safe and beneficial, surrogate markers of arterial stiffness reveal the acute impact of energy drinks on arterial stiffness and can offer valuable prognostic insights beyond traditional cardiovascular risk factors. However, it's crucial to recognize that the impact of energy drinks on cardiovascular health may depend on various factors, including an individual's overall health status, pre-existing cardiovascular conditions, the amount and frequency of energy drink consumption, and individual sensitivity to caffeine. Additionally, research on these topics is ongoing, and new research in this area might provide more insights into the relationship between energy drinks and cardiovascular parameters. Most studies on the cardiovascular effects of energy drinks focus on short-term responses. Long-term consumption patterns and their potential impact on arterial health, including the Alx, may need further investigation.

The findings suggest that energy drink consumption can acutely increase arterial stiffness, as reflected by elevated Alx values, in young healthy adults. This effect is consistent with the known cardiovascular impacts of high caffeine and stimulant intake. Although the clinical significance of these transient changes in Alx is not fully understood, they highlight the potential cardiovascular risks associated with energy drink consumption.

### Study Limitations

This study has several limitations. First, the small sample size, single-center design and lack of long-term follow-up are the main limitations of this study. Therefore, it can be considered a pilot study. Future studies with larger sample sizes will include power analysis to determine the appropriate sample size. Also quantitative data on exact consumption time was not collected, no analysis was performed on this variable. These might ambiguous the effect of energy drink consumption on surrogate markers of arterial stiffness obtained non-invasively. This study highlights the acute effects of energy drink consumption on arterial stiffness in healthy young adults, showing significant transient increases in Alx and Alx@75 after 30 minutes. However, these effects return to baseline within 2 hours. So, further research with larger and more diverse populations is needed to evaluate the chronic cardiovascular effects of energy drinks.

Second, although young healthy adult individuals were included in the study, there was no echocardiographic data. Third, there is no specific threshold value for Alx. It may vary depending on the measurement method and the population studied. But the authors believe that their findings may inspire for further studies to clarify the acute effect of energy drinks consumption on Alx. Finally, compared to previous studies, the study was conducted using relatively lower doses of energy drinks.

### CONCLUSION

In summary, in this study, the authors observed an increase in Alx and Alx@75 values, an indicator of arterial stiffness, in the early period after energy drink consumption in young adult healthy individuals. These observations could be reflective of physiological responses to energy drink consumption or adjustments in vascular tone and resistance. Understanding these changes can be crucial for evaluating cardiovascular health and the effects of energy drink consumption on BP and arterial function. While energy drinks offer a temporary energy boost, their potential risks to arterial stiffness parameters among young people warrant caution, as changes in arterial stiffness from energy drink consumption can affect cardiovascular health. Awareness, education, and responsible consumption are key in addressing this trend.

**Ethics Committee Approval:** This study was approved by Ethics Committee of University of Health Sciences Türkiye, Gülhane Training and Research Hospital (Approval No: 2022&272, Date: 12.09.2022).

**Informed Consent:** Written informed consent was obtained from the patients who agreed to take part in the study.

**Peer-review:** Externally peer reviewed.

**Author Contributions:** Concept – S.Y., M.Ç.; Design – M.Ç., M.G.; Supervision – C.B., M.Ç.; Resources – Ö.E., S.Y.; Materials – M.S.K., S.G.; Data Collection and/or Processing – H.Ş., A.A.; Analysis and/or Interpretation – S.G., S.F.; Literature Search – S.Y., M.G.; Writing – S.Y., M.Ç.; Critical Review – M.Ç., C.B.

**Declaration of Interests:** The authors have no conflicts of interest to declare.

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