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## ACUTE EFFECTS OF ENERGY DRINK CONSUMPTION ON LEFT AND RIGHT VENTRICULAR FUNCTION – A 2-DIMENSIONAL SPECKLE TRACKING ECHOCARDIOGRAPHIC STUDY

<i>Aim</i>	Energy drinks (ED) contain high levels of caffeine and taurine and are associated with several cardiovascular effects. We investigated acute effects of consuming low caffeine and taurine content ED on left ventricular (LV) and right ventricular (RV) function assessed by conventional and two-dimensional speckle tracking echocardiography.
<i>Material and methods</i>	In this crossover study, 34 healthy adults, age 19–48 yrs, drank an ED containing 53.25 milligrams of caffeine, 284 mg of taurine, or an equal volume of control drink (CD) on two separate sessions, 7–10 days apart. Standard echocardiographic and speckle tracking imaging were performed before and 60 min after consumption of the study beverages.
<i>Results</i>	Compared to CD, ED caused a significant increase in tricuspid annular plane systolic excursion ( $p=0.04$ ) and RV systolic wave velocity ( $p=0.01$ ) with no effect on global longitudinal strain when compared to CD. LV systolic function was not altered, but mitral early diastolic velocity by tissue Doppler imaging was significantly higher ( $p=0.031$ ), and early diastolic strain rate, as measured by speckle tracking echocardiography, was significantly lower ( $p=0.022$ ).
<i>Conclusion</i>	Reduced caffeine and taurine content ED does not affect LV systolic function, but increases RV longitudinal contractility and improves LV early diastolic filling.
<i>Keywords</i>	Energy drink; caffeine; taurine; speckle-tracking echocardiography; ventricular function
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### Introduction

Energy drink (ED) consumption has been increasing among people of all ages, especially by adolescents and young adults [1]. The main ingredients of these drinks are caffeine, taurine, sugar, and glucuronolactone, so EDs are marketed with claims to increase endurance, physical and mental performance.

Studies on the effects of EDs on blood pressure and heart rate have shown that EDs lead to an increase in blood pressure and heart rate parallel to their caffeine content [2–4]. These acute changes in heart rate, blood pressure, and peripheral vascular resistance would lead to increased cardiac workload [5].

Caffeine is known to increase myocardial contractility through an increase in plasma catecholamines [6–8]. Taurine also has important functions in cardiomyocyte function and contractility [9–11]. However, as a consequence of endothelial dysfunction, the increased cardiac workload cannot be matched by an increase in myocardial blood flow [12–14]. Several cases of cardiomyopathies and myocardial infarction related to ED consumption have been reported

[15–18]. Increased catecholamines, mismatch in myocardial oxygen demand and supply, as well as increased platelet aggregation and hypercoagulability might be reasons underlying the serious adverse events related to EDs [19, 20].

Although effects of EDs on several cardiac hemodynamic and electrocardiographic parameters have been studied extensively, there have been very few studies on the effects of EDs on myocardial function determined by echocardiography. All of the previous studies investigated EDs with high caffeine and taurine content.

In order to minimize the adverse events related to ED consumption, the Turkish Food Codex Statement on Energy Drinks limits the amounts of several ingredients present in EDs. The maximum legal limits of caffeine and taurine in a 250 ml can of ED that may be marketed in Turkey are 37.5 mg of caffeine and 200 mg of taurine. Elsewhere, a similar volume of ED generally contains 80 mg caffeine and 1000 mg of taurine.

In this study, we investigated the acute effects of consuming an ED containing reduced caffeine and taurine on left ventricular (LV) and right ventricular (RV) function

assessed by conventional echocardiography and 2-dimensional speckle tracking echocardiography. To our knowledge the effects of a low caffeine and taurine content ED on myocardial function have not been investigated previously.

## Material and methods

Thirty-four healthy volunteers between the ages of 19 and 48 yrs (mean age, 33 yrs) were included in this single-center, randomized, controlled, crossover study. The study was approved by the Institutional Clinical Trials Ethics Committee (ref:11-484-14) and was conducted according to the Declaration of Helsinki. The trial is registered with [www.isrctn.com](http://www.isrctn.com) (ISRCTN 40313372). All volunteers gave written informed consent for participation. Volunteers were excluded if they were younger than 18 yrs or older than 50 yrs, had any abnormalities in their baseline screening echocardiographic examination, were heavy caffeine consumers (>200 mg/day), smoked >5 cigarettes/day, had low BMI (<18.5 kg/m<sup>2</sup>), were obese (BMI>30 kg/m<sup>2</sup>), or refused to give consent for participation. 79% never consumed EDs and the remaining 21% did not consume EDs on a regular basis.

## Study design

The subjects were randomized by an online randomization tool ([www.randomizer.org](http://www.randomizer.org)) to drink first either 355 ml of an ED (Red Bull®) containing 53.25 milligrams of caffeine, 284 mg of taurine, and 39 mg of sugar or an equal volume of a mixed fruit juice control drink (CD) containing 39 mg of sugar but no caffeine or taurine. The subjects drank the other drink at a different session after a 7–10 day washout period.

The subjects were asked to come for the examination visits after a fast of at least 6 hr and not to have consumed caffeine or cigarettes within the previous 12 hr. At each visit, baseline measurements of heart rate and blood pressure were recorded while lying, and echocardiographic images were recorded. Within 20 min and while in a sitting position, the subjects drank 355 ml of the assigned study drink. They rested for 60 min after consuming the drink before heart rate and blood pressure measurements and echocardiographic imaging were repeated.

## Conventional echocardiography

A conventional, transthoracic echocardiographic examination was performed with a GE Vivid S5 ultrasound machine (GE Vingmed Ultrasound, Horten, Norway) using a 3.6 MHz phased array transducer, according to the guidelines of the American Society of Echocardiography [21]. Images recorded digitally with frame rates of 40–80 fps were analyzed off-line with EchoPac PC software (GE Health Care, Horten, Norway)

by an operator who was blinded to the subject data. Pulsed Wave (PW) Doppler and Tissue Doppler Imaging (TDI) measurements were averaged over 3 cycles. Ejection fraction was calculated by the modified Simpson's method. LV TDI measurements for systolic (S), early diastolic (e') and atrial contraction (a') wave velocities were recorded from both medial and lateral annuli and averaged for final analysis. Tricuspid annular systolic excursion (TAPSE) and RV TDI values were obtained from the lateral tricuspid annulus. A myocardial performance index (MPI) for each ventricle was calculated from the TDI images. The sum of the isovolumetric relaxation time (IVRT) and the isovolumetric contraction time (ICT) was determined by subtracting ejection time from mitral valve closure to opening time. MPI was calculated with the formula:  $MPI = (IVRT + ICT) / ET$ . The medial and lateral annular calculations were averaged for analysis of LV MPI.

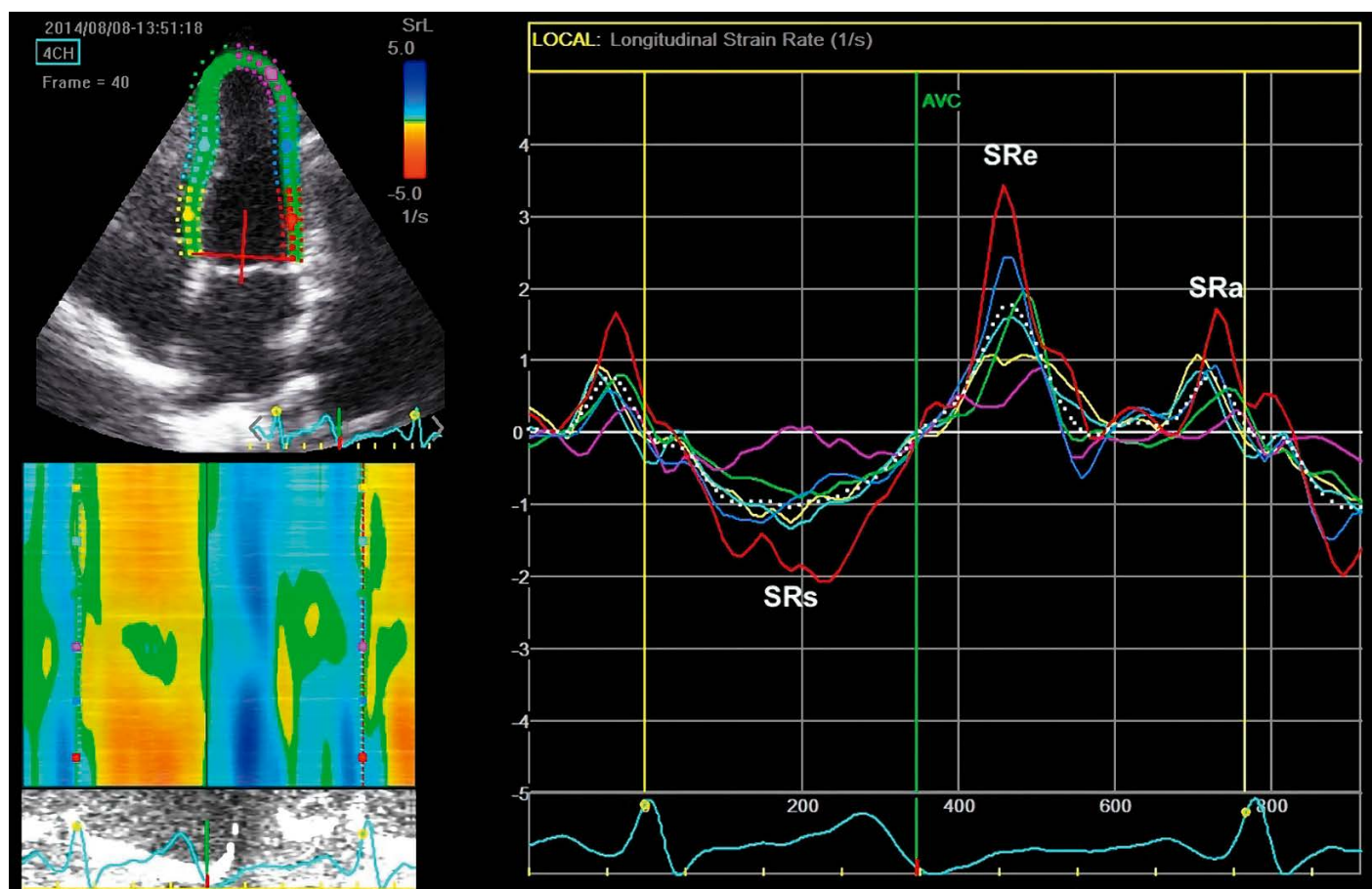
## Dimensional speckle tracking echocardiography

2D Speckle tracking analysis was performed with dedicated EchoPac PC software (GE Health Care, Horten, Norway) by a single operator blinded to the subject data. The operator tracked the endocardium manually in apical 4, 3 and 2 chamber views, and the software tracked the speckle pattern. The region of interest was set to fit the myocardial thickness. Global longitudinal LV strain (GLS) was calculated as the average of the generated 18 segments. Global RV longitudinal strain was calculated from a six segment model, including the RV free wall and interventricular septum. The LV strain rate was measured from LV apical 4, 3 and 2 chamber views in systole (SRs), as well as early (SRe) and late diastole (SRa). The values of 18 segments were averaged for global strain rate analysis (Figure 1).

## Statistical analysis

The sample size was based on previous studies, where consuming a high caffeine-aurine ED caused an absolute 1.5% greater increase in LV global longitudinal strain than the CD [22]. The lowest sample size necessary to determine a 1.5% absolute difference in two drinks, assuming a standard deviation of 2.5% with at least 90% power and 0.05 type 1 error, was 32. Due to the short duration of the study, a dropout rate <5% was anticipated, so 34 patients were included.

Data were evaluated with SPSS v.25 software (IBM Corporation, Armonk, New York, USA) package. The normality of the distribution was evaluated by the Shapiro-Wilk test. Differences between pre and post measurement of each drink was determined by paired samples t-test or Wilcoxon signed rank test depending on the normality of the distribution. Differences between measurements of volunteers after

**Figures 1.** Measurement of systolic (SRs) early diastolic (SRe) and late diastolic (SRa) strain rates by speckle-tracking echocardiography

ED and CD were compared with a two factor, repeated measures ANOVA. Timing of the imaging, i.e., before or after study drink consumption, and the type of drink, i.e., ED or CD, were considered as within subject factors. Study drink- time interactions were investigated to determine the differences between the effects of study beverages on the echocardiographic parameters over time from pre to post consumption measurements. Data are expressed as mean±standard deviation (SD). Significance was set at  $p < 0.05$ .

## Results

All 34 subjects completed the trial according to the protocol. The subjects were 19–48 yrs of age ( $33 \pm 8$  yrs, 18 (53%) were female, 7 (20%) were regular coffee consumers. Mean BMI

was  $24.3 \pm 3.6$  kg/m<sup>2</sup>. None of the subjects consumed EDs on a regular basis. However, 21% had consumed EDs previously.

The low caffeine, low-taurine ED did not cause any significant change in heart rate, blood pressure, or double product (systolic blood pressure  $\times$  heart rate) as compared to the CD. Effects of the study beverages on hemodynamic parameters are summarized in Table 1.

## Effects of the ED on LV function

ED did not cause a significant change in LV TDI S velocity, global longitudinal peak systolic strain or myocardial performance index. Both energy drink and control drink caused an increase in left ventricular ejection fraction (EF) over time ( $p$  for main effect time=0.002), but there were no

**Table 1.** Acute effects of study beverages on hemodynamic variables

Variable	Before ED	After ED	p, value	Before CD	After CD	p, value	p, interaction*
SBP (mmHg)	112.0±10.8	113.2±9.6	0.417	111.9±10.7	112.5±10.0	0.537	0.644
DBP (mmHg)	71.2±8.4	72.4±8.0	0.448	71.0±6.2	72.5±6.9	0.169	0.797
MAP (mmHg)	84.8±8.5	86.0±7.6	0.353	84.7±6.3	85.8±6.4	0.183	0.991
Heart rate (bpm)	72.2±9.3	71.4±8.0	0.437	71.6±7.7	70.2±7.5	0.246	0.677
Double Product (mmHg $\times$ bpm)	8150.3±1668.4	8101.5±1327.7	0.799	8032.0±1305.5	7911.1±1210.9	0.392	0.664

Data are mean±SD. CD, control drink; ED, energy drink; DBP, diastolic blood pressure; Double product = SBP  $\times$  heart rate; MAP, mean arterial pressure; \*p, drink  $\times$  time interaction in repeated measures ANOVA; SBP, systolic blood pressure.



**Table 2. Acute effects of study beverage consumption on LV function**

Variable	Before ED	After ED	p, value	Before CD	After CD	p, value	p, interaction*
Mitral E (m/s)	0.75±0.13	0.78±0.15	0.082	0.76±0.12	0.78±0.14	0.333	0.274
Mitral A (m/s)	0.48±0.12	0.49±0.14	0.729	0.48±0.11	0.49±0.13	0.740	0.942
LV S (cm/s)	9.6±1.4	9.9±1.2	0.210	9.6±1.3	9.9±1.1	0.253	0.782
LVE' (cm/s)	13.8±2.5	14.4±2.8	0.122	14.6±2.7	14.2±2.4	0.292	0.031
LV a' (cm/s)	8.5±1.5	8.8±2.0	0.508	9.0±2.5	9.0±2.5	0.880	0.577
LV MPI	0.38±0.07	0.38±0.08	0.884	0.39±0.05	0.39±0.07	0.909	0.985
LV GLS (%)	-20.1±3.0	-20.4±2.1	0.300	-19.9±2.3	-20.1±2.0	0.727	0.213
LV SRs (s <sup>-1</sup> )	-1.4±0.3	-1.3±0.7	0.208	-1.4±0.3	-1.3±0.3	0.611	0.096
LVEF (%)	58.8±6.4	60.2±6.1	0.013	58.0±5.9	59.1±6.4	0.012	0.771
E/e'	5.5±1.1	5.6±1.1	0.925	5.6±1.2	5.4±1.1	0.264	0.510
LV Sre (s <sup>-1</sup> )	1.8±0.4	1.6±0.6	0.009	1.7±0.5	1.8±0.5	0.288	0.022
LV Sra (s <sup>-1</sup> )	0.7±0.2	0.9±0.4	0.061	0.7±0.2	0.8±0.2	0.568	0.181
E/SRE (m)	0.4±0.1	0.6±0.3	<0.001	0.5±0.2	0.5±0.2	0.911	0.051

Data are mean±SD. CD, control drink; ED, energy drink; LVEF, left ventricular ejection fraction; LV a', late diastolic mitral annular velocity; LV e', early diastolic mitral annular velocity; LV GLS, left ventricular global longitudinal strain; LV MPI, left ventricular myocardial performance index; LV S, systolic mitral annular velocity; LV Sra, left ventricular late diastolic strain rate; LV Sre, left ventricular early diastolic strain rate; LV SRs, left ventricular systolic strain rate; Mitral A, late diastolic transmitral flow velocity; Mitral E, early transmitral flow velocity.

\*p, drink x time interaction in repeated measures ANOVA.

significant drink-time interactions. Mitral inflow early (E) and late diastolic velocities (A) and TDI a' were not different for the ED and CD. However, TDI early diastolic velocity (e') was increased to a greater extent with energy drink than control drink (p=0.031) and early diastolic strain rate (SRe) decreased with the consumption of the ED (p=0.009), and this effect was significantly greater when compared to the CD (p=0.022). The ED did not cause a change in the E/e' ratio, but the E/SRe ratio, which is considered to be a more accurate marker of LV filling pressures [23], increased after energy drink consumption (p<0.001) but did not change after CD consumption. There was a tendency to statistical significance regarding drink × time interaction (p=0.051). Table 2 summarizes the acute effects of the study beverages on LV function.

### Effects of the ED on RV function

The ED caused a significant increase in TAPSE and RV TDI S velocity as compared to the CD (p=0.004). TDI derived RV e' and a' velocities were also similar after both

drinks. RV performance index was reduced (p=0.011), and global longitudinal RV strain was increased (p<0.001) from baseline after ED, but these changes were not significantly different from those caused by the CD when tested by drink-time interaction (p interaction =0.150 for MPI, and =0.244 for GLS). TDI derived right ventricular e' was different among study drinks (p for main effect of drink=0.046) without a significant interaction with time. TDI a' velocities were similar after both drinks. Table 3 summarizes the acute effects of the study beverages on RV function.

### Discussion

In this study, a low-caffeine-low taurine ED caused a change in RV systolic function, but it did not affect LV systolic function. LV early diastolic variables were improved with the ED when compared to the CD, without a difference in late diastolic function.

Previous studies of effects of EDs on the cardiovascular system showed that EDs caused an increase in both heart

**Table 3. Acute effects of study beverage consumption on RV function**

Variable	Before ED	After ED	p, value	Before CD	After CD	p, value	p, interaction*
RV S (cm/s)	14.1±1.6	15.1±1.9	0.020	14.4±1.8	14.3±1.7	0.942	0.001
RV e' (cm/s)	14.4±2.7	15.0±3.0	0.201	14.2±2.8	15.5±2.8	0.023	0.083
RV a' (cm/s)	12.4±3.5	12.2±3.7	0.807	11.9±3.0	12.9±3.7	0.175	0.062
RV MPI	0.34±0.08	0.26±0.19	0.011	0.33±0.06	0.31±0.06	0.219	0.150
TAPSE (mm)	26.1±2.9	28.9±3.3	<0.001	26.2±3.6	27.2±3.3	0.102	0.004
RV GLS (%)	-22.1±1.6	-23.8±2.3	<0.001	-22.4±3.2	-23.0±3.0	0.433	0.244

Data are mean±SD. CD, control drink; ED, energy drink; \*p, drink x time interaction in repeated measures ANOVA; RV a', late diastolic tricuspid lateral annular velocity; RV e', early diastolic tricuspid lateral annular velocity; RV GLS, right ventricular global longitudinal strain; RV MPI, right ventricular myocardial performance index; RV S, systolic tricuspid lateral annular velocity; TAPSE, tricuspid annular plane systolic excursion.

rate and blood pressures, as well as cardiac output, which might reflect increased sympathetic activation [4, 24–27]. Our study failed to find a difference between a low caffeine ED and the CD with regard to blood pressure, heart rate, or the double product, which is thought to be an index of myocardial oxygen consumption [28].

Caffeine and taurine are the main active ingredients of EDs, and they are believed to be responsible for the cardiovascular effects. The major, acute effects of caffeine on myocardium are likely due to adenosine receptor inhibition, phosphodiesterase inhibition leading to an increase in cytosolic calcium, and increased norepinephrine release from sympathetic nerve endings [29].

Taurine has modulatory effects on intracellular calcium and exerts a positive inotropic effect [9, 30]. A supplement that contains taurine, carnitine, and coenzyme Q10 was shown to improve LV end diastolic volume in patients with LV failure [31]. When given together, caffeine and taurine were found to increase myocardial isometric force [32].

The acute effects of consuming high caffeine-taurine EDs on myocardial function, as determined by cardiac imaging, were investigated in very few studies. In a study by Baum [27], EDs increased fractional shortening and late diastolic mitral inflow velocity in response to exercise, as compared to placebo. Menci et al [22] found a significant increase in LV ejection fraction, global longitudinal strain of both left and right ventricles, as well as mitral and tricuspid annular plane systolic excursions after a high caffeine and taurine ED. Diastolic function was not changed. On the contrary, the CD did not cause any change in any of the studied variables. We have found an increase in ejection fraction with both energy drink and control drink over time without a significant difference between the two drinks. As the control drink also increased the ejection fraction the effect might be attributed to volume and sugar content rather than caffeine and taurine. In a cardiac magnetic resonance imaging (MRI) study, a similar ingredient ED caused an increase in MRI-derived LV peak systolic strain and strain rate without a change in diastolic function. The CD, containing caffeine but not taurine, did not cause a significant difference in the studied variables. Thus, the authors attributed the effect of the ED to its taurine content [33]. In a pilot study, Stopa et al did not find any change in the conventional echocardiographic and speckle tracking variables in response to an ED containing 160 mg of caffeine and 2000 mg of taurine [34].

We failed to demonstrate a significant change in LV systolic function with a limited caffeine-taurine content ED, but RV systolic RV S and TAPSE were significantly higher after consumption of an ED as compared to the CD. The right ventricle has several physiological and anatomical differences compared to the left ventricle. First of all, the right ventricle

contracts against a low resistance, pulmonary circulation, so it only needs one fifth of the energy that the LV needs to maintain the same cardiac output [35]. LV myocardial mass is six times that of the RV mass [36]. The lower amounts of the active ingredients, caffeine and taurine, in the ED consumed in our study could still have had some effect on the thin RV myocardium with its lesser energy need, whereas higher concentrations of caffeine and taurine might be necessary to increase the contractility of the thicker LV. Additionally, RV myocardial fibers have a longitudinal orientation, whereas LV fibers have a longitudinal alignment at the subendocardium and a circumferential alignment at midwall. Thus, RV contraction is mostly longitudinal, whereas LV contraction is more circumferential and radial [37]. We have investigated only global longitudinal strain and other parameters of longitudinal function such as TAPSE and LV and RV systolic (S) waves. Circumferential or radial strain of the left ventricle might be influenced more by the ED, so the GLS value might not reflect the true effect of ED consumption on myocardial function in different directions.

Although an acute increase in LV contractility after ED consumption seems to be a favorable effect, chronic increases in catecholamines and contractility could be counterproductive. Several cases of ED-related Takotsubo-like cardiomyopathy have been reported, mostly related to excessive ED consumption [17, 18, 38]. Chronic coffee consumption and heart failure are known to have a J-shaped curve relationship [39]. The low caffeine-taurine ED used in the current study did not affect LV systolic function. Thus, our study demonstrated that legal restriction of caffeine and taurine in EDs might be effective in preventing adverse cardiovascular events.

LV early diastolic wave velocity ( $e'$ ), as measured by tissue Doppler imaging, increased, and early diastolic strain rate decreased significantly after consumption of ED when compared to the CD. The ED did not cause a change in the  $E/e'$  ratio, but the  $E/SRe$  ratio, which is considered to be a more accurate marker of LV filling pressure, increased, with a tendency toward statistical significance ( $p=0.051$ ). Early diastolic relaxation is an active process that requires ATP [40]. Taurine might have a function in phosphorylation of phospholamban and lead to an increase in calcium uptake by sarcoplasmic reticulum, and, in that manner, enhance ventricular relaxation [9].

## Study limitations

Due to legal restriction of the ED ingredients in Turkey, a high caffeine-taurine ED was unavailable, so we were not able to make a head-to-head comparison between low caffeine-taurine EDs with higher caffeine-taurine versions. The subjects in our study were young, healthy adults, so



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findings might not be generalized to the elderly in whom the distribution and metabolism of the active ingredients might differ. The post-ED echocardiographic investigations were recorded at a single time point, at 60 min, based on previous studies showing that peak concentration of caffeine is reached at 60 min, and that significant increases in heart rate and blood pressure occur after 60 min [29, 41]. Peak effects on contractility might be earlier or later than 60 min due to the presence of other active ingredients. Prolonged use of EDs might have cumulative effects, so serial measurements would have been more beneficial. Lastly, the study only investigated the longitudinal strain of the left ventricle, GLS would not have reflected the effects of ED on circumferential and radial strain.

## Conclusion

Acute ingestion of an ED with limited caffeine and taurine content increased RV contractility and LV early diastolic filling when compared to a CD lacking caffeine and taurine. LV longitudinal systolic function was not changed. Limitation of the caffeine-aurine content of EDs might be beneficial in preventing long term adverse health effects.

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