



The Impact of Energy Drink Consumption on Vascular Endothelial Function

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Abstract

Vascular endothelial dysfunction contributes to a variety of cardiovascular and metabolic diseases. Despite anecdotal reports of adverse cardiovascular and hemodynamic effects of energy drink consumption there has been very little scientific research conducted on this important topic. Furthermore, the limited research available has not yielded consistent results. This study tested the hypothesis that consumption of a commercially available energy drink would have no effect on endothelial function as assessed by the post-occlusive hyperemic response (reactive hyperemia index, RHI). Sixteen normotensive, non-smokers (10 men) participated in this study. Vascular endothelial function was assessed, as the RHI, via peripheral artery tonometry using the EndoPAT™ (Itamar Medical Ltd, Caesarea, Israel). Measures were made in the semi-recumbent position at the following time points: 45-min following quiet rest (baseline) as well as 2 hr and 4 hr post consumption of 24 oz of Monster energy drink. The RHI was similar during the three different measurement time points; baseline: 1.7 ± 0.6 units, 2 hr post: 1.8 ± 0.6 units, and 4 hr post: 1.8 ± 0.5 units (main effect of time: $P = 0.82$). These findings suggest that acute consumption of a commercially available energy drink has no effect on vascular endothelial function.

Keywords

Blood flow, Cardiovascular disease, Caffeine, Nitric oxide, Vascular function

Introduction

Energy drink consumption has grown in popularity and remains a rapidly growing segment of the beverage industry [1]. A number of regulatory bodies and anecdotal reports have suggested adverse cardiovascular and hemodynamic effects of energy drink consumption [2,3]. However, mechanistic scientific research investigating this important issue is limited and does not always yield consistent results [4,5]. Cardiovascular disease (CVD) affects millions of people in the United States alone and is currently the number one cause of mortality [6,7]. Therefore, given the popularity of energy drinks combined with the high prevalence of CVD it is critical to investigate the impact of energy drink consumption on indices of risk factors known to contribute to CVD.

The mechanisms resulting in CVD are multifactorial. However, one of the primary underlying pathologies of CVD is atherosclerosis, a disease process that develops sub-clinically in childhood and then presents with clinical symptoms such as angina, myocardial infarction, stroke or death during middle age or beyond [6,8]. One of the initiating events in the development of atherosclerosis is endothelial dysfunction. Endothelial dysfunction is also a complex process and is believed to be a global systemic disease that is associated with a reduction in endothelial derived vasodilation and subsequent increase in endothelial derived vasoconstriction as well as micro vascular remodeling [9-11]. To date there is very limited information regarding the effect of energy drinks on peripheral vascular endothelial function. Some studies report improved indices of endothelial function following consumption of either energy drinks or specific isolated ingredients contained within most commercially available energy drinks [12-16] while others have reported a negative effect [17,18]. Accordingly, this study was designed to test the hypothesis that acute energy drink consumption would have no effect on vascular endothelial function.

Material and Methods

Ethical approval

All experimental protocols and techniques were approved by the Institutional Review Board at the University of Texas at Austin. Participants were given a verbal description of all procedures and were informed of the purpose and risks involved before providing their informed, written consent. All trials were conducted in the morning following an overnight fast (at least 12 hr). Participants refrained from caffeinated or alcoholic beverages for a minimum of 72 hr prior to data collection and all trials were conducted in a temperature controlled laboratory (~24°C and 40% relative humidity).

Participants

Sixteen normotensive, non-smokers (10 men) participated in this protocol. Participant characteristics were: age, 25 ± 3 years; height, 176 ± 8 cm; weight, 73 ± 10 kg; and body mass index, 23 ± 3 kg·m². None of the participants were taking medications and all were free of any known cardiovascular, cerebrovascular, or metabolic disease. The phase of the menstrual cycle was recorded, but not controlled.

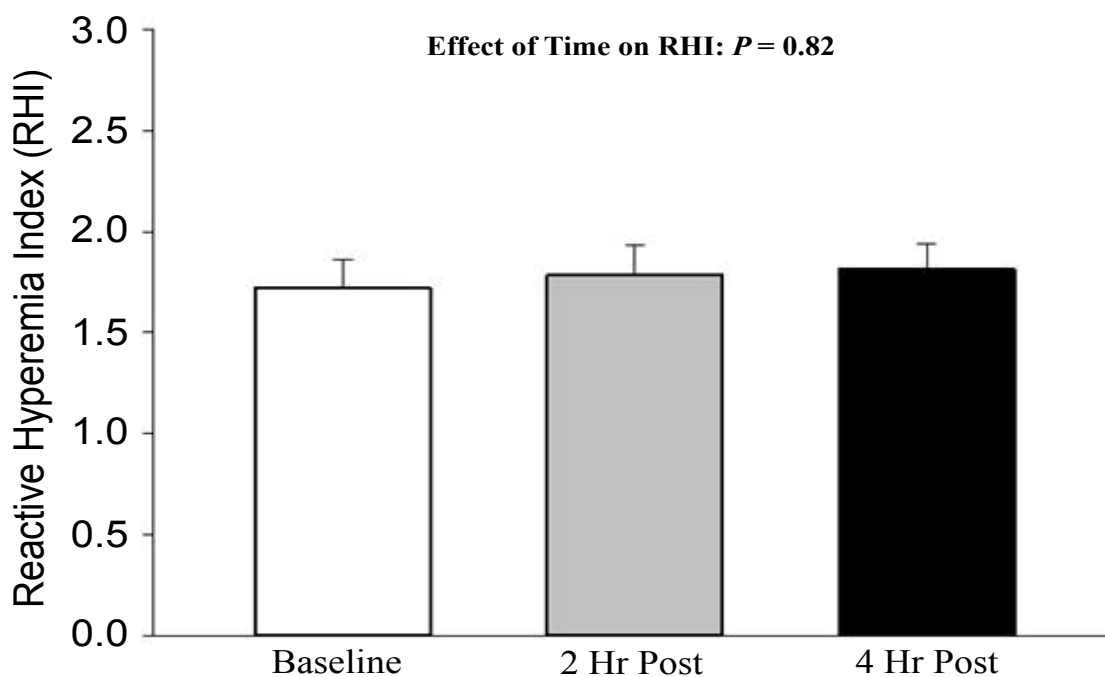


Figure 1: Endothelial function expressed as the reactive hyperemia index at baseline, 2 hr, and 4 hr post-energy drink consumption. Energy drink consumption had no effect on endothelial function (main effect of time; $P = 0.82$).

Instrumentation and measurements

All data was collected in a dimly lit room with the participant in a semi-recumbent position on a patient bed. Body weight (to the nearest 10th of a kg) was determined from the average of two consecutive measurements using a digital medical scale with platform stadiometer (Seca 763). Participants were then instrumented with a blood pressure cuff on an upper arm for assessment of arterial blood pressure via auscultation of the brachial artery (Sun Tech Medical, Inc.). An additional blood pressure cuff was placed on the other arm for arterial occlusion during the data collection period (see below).

Vascular endothelial function was assessed via peripheral artery tonometry using the EndoPAT™ (Itamar Medical Ltd, Caesarea, Israel). This is a well validated approach to assess endothelial function that is utilized in both research and clinical settings. This method involves a cuff placed around the right and left index finger and a blood pressure cuff placed around the upper portion of the dominant arm. The finger cuffs measure pulse amplitude tonometry prior to and during reactive hyperemia induced by release of a blood pressure cuff, thus providing an index of blood flow and vascular function. Baseline amplitude tonometry was assessed for 5 min after which the blood pressure cuff around the dominant arm was inflated to ~250 - 280 mmHg for 5 min and tonometry measures occurred throughout cuff inflation and for 5 min post cuff deflation. After each cuff inflation/deflation period an arterial tonometry reactive hyperemic index (RHI) is calculated which provides a validated measure of endothelial function [17,19,20].

Experimental protocol

Following instrumentation participants rested quietly in the semi-recumbent position for a minimum of 45 min prior to data collection. After this 45 min resting period peripheral vascular endothelial function was assessed (as described above). Following this baseline measurement the participants consumed a 24 ounce can of Monster Energy drink. Participants finished the beverage within 20 min. Peripheral vascular endothelial function was repeated, as previously described, at 2 hr and 4 hr post-beverage consumption.

Statistical analysis

The effect of energy drink consumption on the RHI was analyzed using a commercially available statistical package (Sigma Plot 11.0).

Data was analyzed using a 1 Way Repeated Measures analysis of variance (ANOVA) with main effect of time; baseline (pre-beverage consumption), 2 hr and 4 hr post beverage consumption. Significant differences were accepted at $P < 0.05$ and data are presented as means \pm SE unless otherwise noted.

Results

An endothelial function, indexed as RHI, was not affected following consumption of the energy drink (Figure 1). The RHI at baseline was 1.7 ± 0.1 units, whereas the RHI was 1.8 ± 0.2 units and 1.8 ± 0.1 units at 2 and 4 hr post consumption respectively (main effect of time: $P = 0.82$).

Discussion

This study investigated the effect of energy drink consumption on peripheral vasculature endothelial function. The primary finding is that 24 oz of a commercially available energy drink had no effect of endothelial function.

Vascular endothelial dysfunction is a common risk factor for a variety of cardiovascular, cerebral vascular, and metabolic diseases [11]. In addition recent research has suggested that endothelial dysfunction is also a contributor to dementia, Alzheimer's disease, as well as cognitive dysfunction [21]. To date the impact of energy drink consumption on endothelial function has been the focus of limited scientific research, with some reports suggesting a positive [12-16] and others suggesting a negative effect [17,18]. Given the popularity of energy drinks and the elevated disease risk associated with endothelial dysfunction this is a critical topic that warrants further research. Grasser et al. assessed the cutaneous blood flow response, using laser-Doppler flowmetry, during administration of the endothelial-dependent vasodilator acetylcholine [13]. They observed a significantly elevated blood flow response to ACh 2 hr following consumption or 355 ml of red bull relative to consumption of 355 ml of water [13]. These findings are supported by a study which observed significant increases in flow mediated dilation (FMD) in the brachial artery in a group of young smokers and non-smokers following oral ingestion of 1.5 grams of taurine [14]. While this dose of taurine is common to what is found in many energy drinks it is difficult to extrapolate these findings to energy drink consumption per se. Tsutsui et al. recently reported an increase in post-occlusive

blood flow in the finger following consumption of caffeinated vs. decaffeinated coffee [15]. Lastly, Umemura et al. demonstrated that the forearm blood flow response (assessed using plethysmography) during incrementally higher doses of ACh was elevated following consumption of 300 mg of caffeine relative to placebo [16]. In addition there are limited studies that indicate a negative effect of energy drinks on peripheral endothelial function. Worthley et al. demonstrated a reduction in pulse wave amplitude in response to a flow-mediated stimulus (assessed using the EndoPAT™) following consumption of 250 ml of a sugar-free energy drink relative to 250 ml of water [17]. Another case report suggests that energy drink consumption reduces endothelial function as assessed by FMD. However, this finding needs to be interpreted cautiously as it was based on one subject [18]. It is unclear why the results differ between the limited studies which have investigated this topic but the discrepancies are likely related to a number of methodological considerations. These include; subject cohort and sample size, type and volume of beverage consumed, time course of measurements before and after consumption etc. The findings of the current study add additional information to the literature and suggest that there is no effect, either positive or negative, of energy drink consumption on endothelial function. Furthermore, the RHI values obtained in this study, at all time points were approximately 1.8 units which are similar to values reported in other studies that did not involve any form of perturbation [20].

Methodological Considerations

There are several methodological considerations that are worth mentioning. This study was designed to assess the impact of a commercially available energy drink and thus was practical in nature. Therefore, these findings are not able to isolate out the role of the various ingredients contained within the beverage. The increase in blood flow that occurs following cuff release is predominately due to shear stress against the blood vessel wall which results in a robust increase in bioavailability of the potent vasodilator Nitric Oxide. Therefore, the methodology utilized in the current study specifically assessed endothelial-dependent vasodilation and a separate approach to target endothelial-independent vasodilation (i.e., sublingual nitroglycerin) was not performed. However, the findings of similar endothelial-dependent responses before and after beverage consumption strongly suggest that there was no effect of the energy drink on any facet of the vasodilator pathway. Furthermore, a previous study reported that energy drink consumption had no effect of endothelial-independent vasodilation at 1 hr post-consumption [17]. As previously mentioned this study was designed to be practical in nature; therefore, the participants consumed a volume of energy drink (24 oz) that is commonly purchased in a real life setting. As a result the amount of caffeine and other potentially vasoactive ingredients consumed was not normalized to body mass index. However, we do not feel as if this approach impacted the results because separate correlation analyses, at each measurement time point, revealed no relationship between body mass index and endothelial function (Baseline: $r^2 = 0.04$, $P = 0.41$; 2 hr post: $r^2 = 0.02$, $P = 0.63$; 4 hr post: $r^2 = 0.07$, $P = 0.31$, data not shown). There was not a time control condition in this study. However, RHI, assessed using the EndoPAT™, remained constant when measurements were obtained at 2 hr intervals over a 12 hr time period (total of 7 measurements) or at 1 hr intervals over a 5 hr time period (total of 5 measurements) [22]. Therefore, we do not feel that a lack of a time control condition impacted the findings. Lastly, all of the participants were relatively young and healthy individuals and thus it remains unknown if these findings translate to older individuals or those with disease risk and/or overt disease.

Conclusion

The findings of the current study suggest that a commercially available energy drink has no impact of endothelial vascular function for up to 4 hr post consumption.

Conflict of Interest

The author has no conflict(s)-of interest/disclosures to report.

Author Contribution

R. Matthew Brothers contributed to all aspects of this work.

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