12-2021

Acute Consumption of a Sugar-Sweetened Beverage Impairs Microvascular Function in Midwestern Hispanic Males

Emma Hirshman

Anne R. Crecelius
University of Dayton, acrecelius1@udayton.edu

26 (2021) 200129

Contents lists available at ScienceDirect

Human Nutrition & Metabolism

journal homepage: www.sciencedirect.com/journal/human-nutrition-and-metabolism

Acute consumption of a sugar-sweetened beverage impairs microvascular function in Midwestern Hispanic males

Emma Hirshman, Anne R. Crecelius *

Department of Health and Sport Science, University of Dayton, Dayton, OH, USA

ARTICLE INFO

Keywords:
Blood flow
Endothelial function
Latinx
Reactive hyperemia
Fructose
Glucose
Humans

ABSTRACT

Hispanic males are at risk for cardiovascular disease and consume high levels of sugar-sweetened beverages (SSB); yet, their acute vascular response to SSBs is unknown. Ten healthy Hispanic men (18–45 years old) consumed a SSB and reactive hyperemia was performed prior to, immediately following, and 1-hr post-consumption. Both peak and total (area under the curve) forearm blood flow were attenuated immediately following consumption but returned to at or above pre-consumption levels at the 1-hr test.

1. Introduction

Hispanics represent the largest minority group in the United States, comprising about 17.6% of the total population [1]. This population is at a high risk for obesity, diabetes, and many cardiovascular diseases (CVDs) as well as consumes high levels of sugar-sweetened beverages (SSB) [2–6]. Specifically, Hispanic males have about a 1.5 times higher prevalence of diabetes when compared to male non-Hispanic Whites [3,4] and along with non-Hispanic Black men, have the highest consumption of SSBs when examined by ethnicity [6]. Further, across all ethnicities, the 20–39 year old male age-sex group has the highest consumption of SSBs [6].

Recent studies have looked at the acute effects of SSBs on healthy individuals of various ethnicities and found that there is a strong correlation between the increased consumption of sugar drinks and endothelial dysfunction [7–10] an important pre-clinical marker of CVD [11]. In the most comprehensive examination [8], macro and microvascular responses were measured utilizing flow-mediated dilation and acetylcholine iontophoresis. Previous studies in our lab utilized the forearm reactive hyperemia test to examine the acute effect of SSBs on microvascular function in young healthy Caucasian adults and found no significant change in microvascular function [12].

Despite the high consumption rates of SSBs by Hispanic males, the high prevalence of related diseases [4,13,14], and support for a role of vascular function linking these together, this ethnic subgroup has not been directly studied. Thus, we sought to test the hypothesis that acute consumption of SSBs impairs microvascular function in a group of Midwestern Hispanic males.

2. Methods

2.1. Subjects

After approval by the University of Dayton Institutional Review Board and written informed consent, ten healthy, non-obese Hispanic males between the ages of eighteen and forty-five who were non-smokers, and not hypertensive (<140/90 mmHg), separately reported to the lab on one occasion for the 3-h protocol. Inclusion criteria required subjects to either be foreign-born Latinos/Hispanic (no subjects had non-Spanish speaking ethnicities) or have both parents be foreign-born. Recruitment efforts were performed in both English and Spanish language.

2.2. Experimental Environment

Subjects were instructed to fast overnight (12 h), refrain from drinking caffeine for 12 h and abstain from exercising for 24 h. All of the experiments were performed in a temperature controlled Integrative Human Physiology Laboratory (21–22° C) in the supine position. Body composition was assessed via Bioelectric Impedance Analysis technology (Tanita DC-430U Dual Frequency Total Body Composition Analyzer). Subjects rested for 30 min, then completed the pre-

* Corresponding author. Dept. Health and Sport Science, 300 College Park Ave, Dayton, OH, 45469-2968, USA.
E-mail addresses: ehirshman@gmail.com (E. Hirshman), acrecelius1@udayton.edu (A.R. Crecelius).

https://doi.org/10.1016/j.hnm.2021.200129
Received 23 February 2021; Received in revised form 24 May 2021; Accepted 6 August 2021
Available online 12 August 2021

consumption reactive hyperemia (RH) trial. The protocol included three RH trials: pre-consumption, immediate post-consumption, and 1-h post beverage consumption. Each trial was divided into three timepoints: baseline (3 min), occlusion (5 min), and recovery (3 min). After the pre-consumption trial subjects were raised to a seated position and given 5 min to consume the SSB (See Fig. 1). After completion of the beverage, the immediate RH trial occurred. One hour after completion of this trial, the third and final RH test was performed.

2.3. Blood glucose and cholesterol

Aseptic technique and a lancet were used to obtain blood samples (~40 μl) for the cholesterol, HDL, triglycerides, and blood glucose panel (PTS diagnostics lipid panel) prior to the pre-consumption RH trial. These were measured via a CardioChek device (Polymer Technology Systems, Indianapolis, Ind). Glucose measurements were repeated immediately post consumption and 1-h post consumption via a Glucometer (Contour Next EZ Blood Glucose Meter) using a 1.8 mm spring loaded lancet (Unistik 3 Normal) for the blood draw.

2.4. Systemic hemodynamics

Systemic hemodynamics were measured throughout the experiment. Heart rate was obtained using a 3 lead ECG (Power lab, AD Instruments). Blood pressure was measured on the finger using beat-to-beat photoplethysmography (Nova, Finapres, Netherlands). Calibration via upper arm cuff occurred during each trial.

2.5. Beverage

Subjects were given 5 min to consume a 24-ounce water beverage that was sweetened with 65g of a 55% fructose, 45% glucose mixture (to mimic the concentration of HFCS in Coca-Cola products [15]. The beverage was also flavored with one serving (~4 drops) of lemon juice, a beverage solution used in previous investigations [16].

2.6. Forearm blood flow

Forearm blood flow (FBF) was measured using venous occlusion plethysmography with mercury-insalistic strain gauges. A blood pressure cuff was placed around the subject's wrist and inflated to a suprasystolic value of ~200mmHg to occlude inward and outward flow of the hand in order to isolate the forearm vasculature for measurement. Another cuff was placed superior to the antecubital fossa on the same arm. This cuff cycled from inflated at ~50 mmHg for a span of 7 s to deflated for 8 s. Forearm vascular conductance was calculated by FBF/MAP x 100 and was used to account for mean arterial pressure [17,18].

2.7. Reactive Hyperemia

After measurement of baseline FBF, the cuff on the upper arm was rapidly inflated to 200 mmHg for 5 min of transient ischemia. Blood flow was measured immediately prior and immediately following cuff deflation. This location and duration of ischemia is chosen to mimic the reactive hyperemia (RH) protocol utilized in previous studies [12,17,18]. After 5 min, the cuff was rapidly deflated and flow measures commenced with an initial cycle of 4 s of inflation and 3 s of deflation for 56 s before returning to the baseline FBF procedure of 7:8 s for ~2 min. Previously, this design has been utilized to investigate various endothelial–derived vasodilator pathways [17,18]. Peak forearm blood flow (FBF) and area under the curve (AUC) (Fig. 3) were calculated from the dynamic reactive hyperemic response over a span of 150s after the 5-min occlusion (Fig. 2).

2.8. Statistical analysis

Repeated measures ANOVA was used to investigate changes over time during the experiment. Tukey's post-hoc testing was used to determine specific pairwise comparisons via Prism 8 software. Significance was set *a priori* at $P < 0.05$. Data are presented as mean ± SD.

3. Results

3.1. Subjects characteristics

Subjects (age: 22 ± 2 years; height: 180 ± 9 cm; weight: 84 ± 11 kg) were first or second generation from a variety of Hispanic cultures including: Chile, Colombia, Cuba, Dominican Republic, Mexico, Nicaragua, Puerto Rico, and Spain. All subjects chose to complete study-related paperwork in English. Blood lipids were within normal range and additional subject characteristics are presented in Table 1.

3.2. Blood glucose

The pre-consumption, immediate post-consumption, 1-hr post consumption results were 102 ± 8 mg/dL to 102 ± 8 mg/dL to 95 ± 12 mg/dL respectively. Mean blood glucose was not significant (p = 0.3101) across all trials.

3.3. Systemic hemodynamics

Systemic hemodynamics are presented in Table 2. Immediately following SSB consumption there were slight, but significant elevations in both heart rate (HR; 57 ± 12 vs 59 ± 11 bpm; p = 0.0015) and mean arterial pressure (MAP; 74 ± 16 vs 79 ± 8 mmHg; p=<0.0001).

3.4. Reactive hyperemic response

Dynamic mean reactive hyperemic response (all flows acquired) is presented in Fig. 2 while the peak (greatest magnitude) and total FBF (area under the curve) responses are presented in Fig. 3. Peak FBF immediately after consumption (19.9 ± 10.3 mL/100 mL/min) was lower, although not statistically significantly so, than pre-consumption (26.3 ± 10.1 mL/100 mL/min; ANOVA p = 0.13). The 1-hr post-

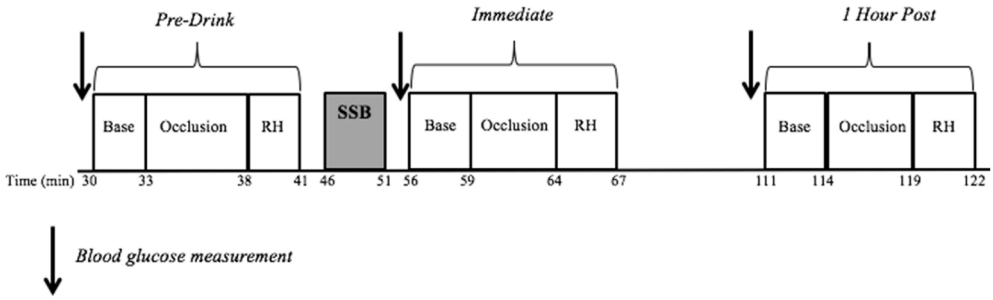

Fig. 1. Experimental timeline. SSB= sugar-sweetened beverage, RH = reactive hyperemia.

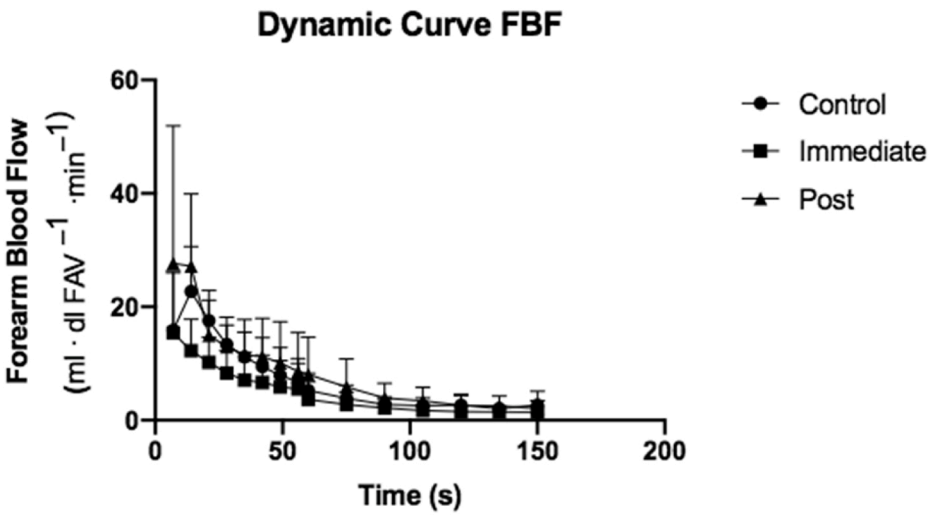

Fig. 2. Mean ± SD forearm blood flow over time following release of 5 min of upper arm occlusion, as measured by venous occlusion plethysmography. Reactive hyperemia trials occurred pre, immediate post-, and 1-h post consumption of a sugar-sweetened beverage in 10 young, healthy, Hispanic males.

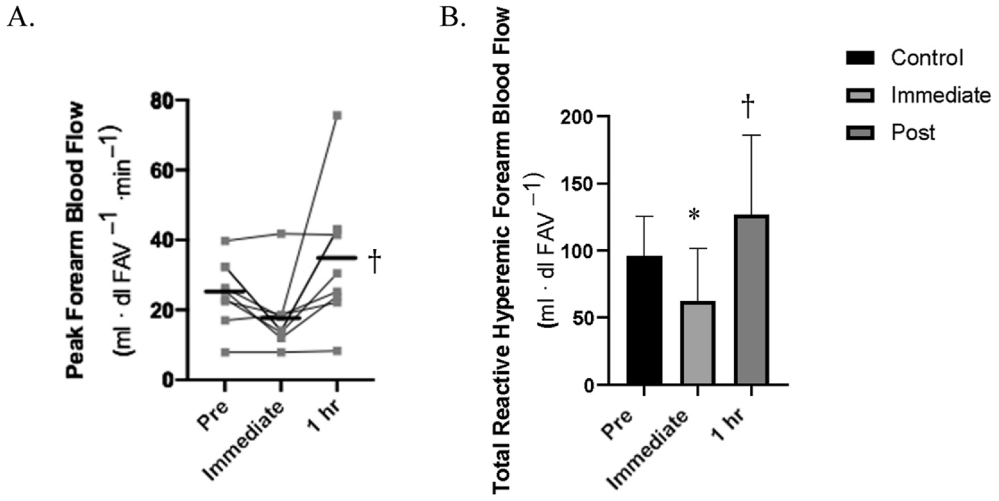

Fig. 3. Peak (individual in grey lines, mean in black bars; A.) and Total (area under the curve; mean ± SD; B.) forearm reactive hyperemia in response to 5-min occlusion pre sugar-sweetened beverage consumption, immediately following consumption, and 1-hr post consumption in Hispanic males (n = 10). *P = 0.01 vs pre-consumption, †P = 0.04 vs immediate.

Table 1
Demographic characteristics of study participants (N = 10).

Characteristic	Mean (SD)
Age (years)	22 (2)
Height (cm)	180 (9)
Weight (kg)	84 (11)
BMI	26 (4)
% Body Fat	16 (8)
Cholesterol (mg/dl)	135 (30)
HDL (mg/dl)	49 (9)
Triglyceride (mg/dl)	104 (41)

Table 2
Systemic hemodynamics.

Time	Rest HR (bpm) (SD)	Rest MAP (mmHg) (SD)	Rest Systolic Pressure (mmHg) (SD)	Rest Diastolic Pressure (mmHg) (SD)
Pre-Consumption	57 (12)	85 (7)	123 (8)	67 (8)
Immediate Post Consumption	59 (11)	88 (6)	125 (6)	69 (8)
1-Hour Post Consumption	62 (11)	88 (7)	123 (7)	70 (7)

consumption peak RH FBF (34.3 ± 18.2 mL/100 mL/min) was non-significantly greater than the immediate post-consumption trial (p = 0.07). Changes were similar for Peak FVC and are shown in Fig. 4. Total FBF AUC for the pre-consumption trial (100.4 ± 31.1 mL/100 mL) was significantly different from the immediate post-consumption trial (66.2 ± 37.2 mL/100 mL; p = 0.04) but not the 1-h post consumption trial (127.8 ± 58.9 mL/100 mL; p = 0.21). immediate post-consumption trial was significantly lower than 1-h post-consumption trial (Fig. 3B; p = 0.009).

4. Discussion

In the present study, acute SSB consumption acutely impaired microvascular function in Hispanic males, as measured by forearm reactive hyperemia. Specifically, total FBF (area under the curve) was significantly lower than pre-consumption, and the impairment in peak

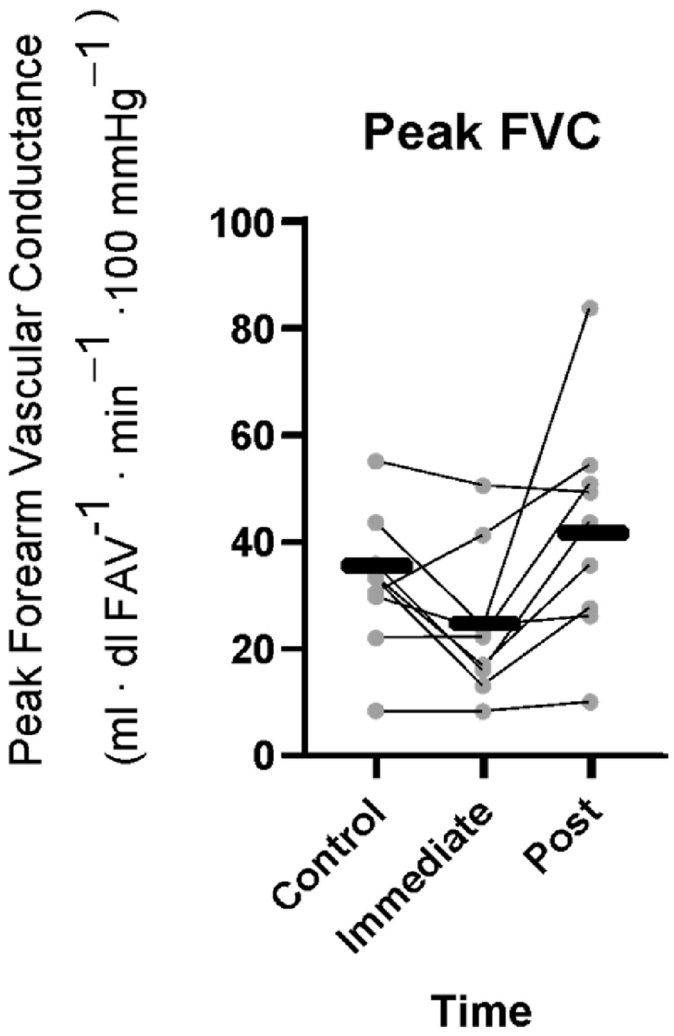

Fig. 4. Peak individual (grey circles and lines) and mean (black bars) forearm vascular conductance following release of 5 min of upper arm occlusion, as measured by venous occlusion plethysmography. Reactive hyperemia trials occurred pre, immediate post-, and 1-h post consumption of a sugar-sweetened beverage in 10 young, healthy, Hispanic males.

FBF approached statistical significance. Interestingly, 1 h following consumption, function was normalized and the attenuation in response observed immediately following consumption did not coincide with an observable acute hyperglycemic response.

The ethnicity-specific findings in the present student align with those of Loader et al. in a mixed population [8] yet differ from our previous work in a Caucasian-only population [12]. In our previous work [12], there was no significant impact of acute SSB consumption on microvascular blood flow. While a limitation of the present study is that we did not direct compare responses to a non-Hispanic population, it is possible that there is an increased sensitivity to SSBs in the presently studied group. Regarding systemic responses, HR and MAP increased slightly, but statistically significant, immediately following SSB consumption similar to previous reports [19,20].

Observable hyperglycemia was not present in the current study, despite a significant fructose-glucose load. It is possible this is attributable to the use of whole blood measures versus plasma or serum [21]. Another possible explanation is that despite instructions to maintain minimal movement, contraction mediated glucose uptake occurred [22]. Given a lack of significant change in measured glucose levels, we did not include any multivariate or correlational analysis of glucose levels and vascular responses. Had we been able to measure glucose levels continuously, this approach could have been warranted. However, if anything, impairments in vascular function despite observable hyperglycemia indicate we may be underestimating the detrimental effect of SSBs consumption on vascular function.

We did not collect past and current intake of SSBs to explore whether long term consumption habits have an impact on the acute response of the vessels. Given the prevalence of high SSB consumption in this population, future studies should address whether this impacts the acute response and whether interventions to decrease consumption would improve vascular function.

The present population of Hispanic men was slightly overweight, based upon BMI. However, in our previous work [15] the subject population was also on the upper range of normal adiposity (25 ± 1) and did not demonstrate impaired vascular responses post consumption. Thus, we do believe that the present findings are representative of the Hispanic male population, rather than a confounding effect of obesity.

The present study demonstrates an impaired vascular responsiveness in Hispanic males following consumption of an SSB. Given the prevalence of CVDs and the high consumption rates in this population, the findings of this study provide an initial mechanistic understanding of the relationship between SSBs and CVD in this at-risk population and should spur future interventional research.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

The authors would like to thank the volunteer participants for this study and Jenna Sorensen for her assistance in data collection.

References

[1] U.C. Bureau, FFF: hispanic heritage month 2016, Available from: https://census.gov/newsroom/facts-for-features/2016/cb16-ff16.html, 2018. accessed 5/24/20.
[2] A.A. Bremer, R.S. Byrd, P. Auinger, Racial trends in sugar-sweetened beverage consumption among US adolescents: 1988-2004, Int. J. Adolesc. Med. Health 23 (3) (2011) 279–286, https://doi.org/10.1515/ijamh.2011.056 [accessed 5/24/20].
[3] C.L. Ogden, M.D. Carroll, B.K. Kit, K.M. Flegal, Prevalence of childhood and adult obesity in the United States, 2011-2012, Jama 311 (8) (2014) 806–814, https://doi.org/10.1001/jama.2014.732.
[4] L.P. Pabon-Nau, A. Cohen, J.B. Meigs, R.W. Grant, Hypertension and diabetes prevalence among U.S. Hispanics by country of origin: the National Health Interview Survey 2000-2005, J. Gen. Intern. Med. 25 (8) (2010) 847–852, https://doi.org/10.1007/s11606-010-1335-8.
[5] S. Park, L. Pan, B. Sherry, H.M. Blanck, Consumption of sugar-sweetened beverages among US adults in 6 states: behavioral risk factor surveillance system, 2011, Prev. Chronic Dis. 11 (2014) E65, https://doi.org/10.5888/pcd11.130304.
[6] A. Rosinger, K. Herrick, J. Gahche, S. Park, Sugar-sweetened beverage consumption among U.S. Adults, 2011-2014. NCHS Data Brief 270, 2017, pp. 1–8. Available from: https://www.ncbi.nlm.nih.gov/pubmed/28135185. accessed 5/24/20.
[7] J. Loader, D. Montero, C. Lorenzen, R. Watts, C. Meziat, C. Reboul, et al., Acute hyperglycemia impairs vascular function in healthy and cardiometabolic diseased subjects: systematic Review and meta-analysis, Arterioscler. Thromb. Vasc. Biol. 35 (9) (2015) 2060–2072, https://doi.org/10.1161/atvbaha.115.305530.
[8] J. Loader, C. Meziat, R. Watts, C. Lorenzen, D. Sigaudo-Roussel, S. Stewart, et al., Effects of sugar-sweetened beverage consumption on microvascular and macrovascular function in a healthy population, Arterioscler. Thromb. Vasc. Biol. 37 (6) (2017) 1250–1260, https://doi.org/10.1161/ATVBAHA.116.308010.
[9] E. Mah, R.S. Bruno, Postprandial hyperglycemia on vascular endothelial function: mechanisms and consequences, Nutr. Res. (N.Y.) 32 (10) (2012) 727–740, https://doi.org/10.1016/j.nutres.2012.08.002.
[10] P. Varsamis, G. Walther, B. Share, F. Taylor, S. Stewart, C. Lorenzen, et al., Transient endothelial dysfunction induced by sugar-sweetened beverage consumption may be attenuated by a single bout of aerobic exercise, Microvasc. Res. 115 (2018) 8–11, https://doi.org/10.1016/j.mvr.2017.07.003.
[11] T.J. Anderson, F. Charbonneau, L.M. Title, J. Buithieu, M.S. Rose, H. Conradson, et al., Microvascular function predicts cardiovascular events in primary prevention: long-term results from the Firefighters and Their Endothelium (FATE) study,

Circulation 123 (2) (2011) 163–169, https://doi.org/10.1161/CIRCULATIONAHA.110.953653.
[12] J.L. Keller, E.K. Kelsch, A.R. Crecelius, Acute effects of sugar-sweetened beverage consumption on hemodynamics and reactive hyperemia in young, Healthy Humans 4 (2) (2018), 2018, http://www.jnsci.org/index.php?journal=nsci&page=article&op=view&path%5B%5D=486. accessed 5/24/20.
[13] L. Chen, B. Caballero, D.C. Mitchell, C. Loria, P.H. Lin, C.M. Champagne, et al., Reducing consumption of sugar-sweetened beverages is associated with reduced blood pressure: a prospective study among United States adults, Circulation 121 (22) (2010) 2398–2406, https://doi.org/10.1161/CIRCULATIONAHA.109.911164.
[14] M.L. Wang, S.C. Lemon, B. Olendzki, M.C. Rosal, Beverage-consumption patterns and associations with metabolic risk factors among low-income Latinos with uncontrolled type 2 diabetes, J. Acad. Nutr. Diet. 113 (12) (2013) 1695–1703, https://doi.org/10.1016/j.jand.2013.06.351.
[15] J.S. White, L.J. Hobbs, S. Fernandez, Fructose content and composition of commercial HFCS-sweetened carbonated beverages, Int. J. Obes. 39 (1) (2015) 176–182, https://doi.org/10.1038/ijo.2014.73.
[16] J. Keller, J. Kahlhofer, A. Peter, A. Bosy-Westphal, Effects of low versus high glycemic index sugar-sweetened beverages on postprandial vasodilatation and inactivity-induced impairment of glucose metabolism in healthy men, Nutrients 8 (12) (2016), https://doi.org/10.3390/nu8120802.
[17] A.R. Crecelius, J.C. Richards, G.J. Luckasen, D.G. Larson, F.A. Dinenno, Reactive hyperemia occurs via activation of inwardly rectifying potassium channels and Na+/K+-ATPase in humans, Circ. Res. 113 (8) (2013) 1023–1032, https://doi.org/10.1161/CIRCRESAHA.113.301675.
[18] A.R. Crecelius, B.S. Kirby, J.C. Richards, L.J. Garcia, W.F. Voyles, D.G. Larson, et al., Mechanisms of ATP-mediated vasodilation in humans: modest role for nitric oxide and vasodilating prostaglandins, Am. J. Physiol. Heart Circ. Physiol. 301 (4) (2011) H1302–H1310, https://doi.org/10.1152/ajpheart.00469.2011.
[19] C.L. Chapman, T. Grigoryan, N.T. Vargas, E.L. Reed, P.J. Kueck, L.D. Pietrafesa, et al., High-fructose corn syrup-sweetened soft drink consumption increases vascular resistance in the kidneys at rest and during sympathetic activation, Am. J. Physiol. Ren. Physiol. 318 (4) (2020) F1053–F1065, https://doi.org/10.1152/ajprenal.00374.2019.
[20] A.H. Malik, Y. Akram, S. Shetty, S.S. Malik, V. Yanchou Njike, Impact of sugar-sweetened beverages on blood pressure, Am. J. Cardiol. 113 (9) (2014) 1574–1580, https://doi.org/10.1016/j.amjcard.2014.01.437.
[21] H.S. Kim, Blood glucose measurement: is serum equal to plasma? Diabetes Metab. J 40 (5) (2016) 365–366, https://doi.org/10.4093/dmj.2016.40.5.365.
[22] A. Bergouignan, C. Latouche, S. Heywood, M.S. Grace, M. Reddy-Luthmoodoo, A. K. Natoli, B.A. Kingwell, Frequent interruptions of sedentary time modulates contraction- and insulin-stimulated glucose uptake pathways in muscle: ancillary analysis from randomized clinical trials, Sci. Rep. 6 (2016) 32044, https://doi.org/10.1038/srep32044.