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Effects of glucose ingestion on autonomic and cardiovascular measures during rest and mental challenge

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ABSTRACT

Background: High levels of dietary sugar consumption may result in dysregulated glucose metabolism and lead to elevated cardiovascular disease risk via autonomic nervous system and cardiovascular dysfunction. Altered cardiovascular function can be examined using perturbation tasks such as mental challenge. This study examined the effects of controlled glucose intake on cardiovascular measures at rest and in responses to mental challenge in a laboratory setting.

Method: Using a double blind within-subjects design, participants were monitored at baseline, following ingestion of a glucose or taste-control solution, during structured speech (SS), anger recall (AR) and recovery ($N = 24$, 288 repeated measures; age $= 21 \pm 2$ years). Pre-ejection period (PEP), heart rate (HR), stroke index (SI), cardiac index (CI), blood pressure and total peripheral resistance (TPR) were measured throughout the protocol.

Results: Glucose resulted in sustained decreased PEP levels compared to control condition ($\Delta = 11.98 \pm 9.52$ vs. 3.27 ± 7.65 m·s, $P < .001$) and transient increases in resting HR ($P = .011$), CI ($P = .040$) and systolic blood pressure ($P = .009$). Glucose did not result in increased cardiovascular reactivity to mental challenge tasks, but was associated with a delayed HR recovery following AR ($P = .032$).

Conclusion: Glucose intake resulted in a drop in PEP indicating increased sympathetic nervous system activity. No evidence was found for glucose-related exaggerated cardiovascular responses to mental challenge. Dysregulated glucose metabolism may result in elevated cardiovascular disease risk as a result of repeated glucose-induced elevations of sympathetic nervous system activity.

Introduction

The consumption of foods containing high levels of sugar is correlated with cardiovascular disease risk factors, including impaired glucose metabolism, obesity, dyslipidemia, Type 2 diabetes, and hypertension [1–3]. Multiple biological pathways are involved in these adverse outcomes, including glucose related dysregulation of vascular biology and function. Most research addressing sugar intake and cardiovascular risk is cross sectional and based on self-reported usual dietary habits. Little is known about the effects of systematically controlled glucose ingestion on autonomic nervous system and cardiovascular function.

Previous investigations of the effects of acute glucose ingestion on resting cardiovascular function have demonstrated potent hemodynamic effects characterized by increases in cardiac output (CO), heart rate (HR), systolic blood pressure (SBP), and superior mesenteric artery flow and decreases in diastolic blood pressure (DBP) and total peripheral resistance (TPR) [1,4]. These glucose-induced hemodynamic alterations reflect, at least in part, increased demands from the gut for blood for digestive activities.

Evidence suggests that acute ingestion of glucose results in increased mental challenge-induced hypothalamic–pituitary–adrenal (HPA) axis activity [5,6], as well as TPR and attenuated challenge-induced elevation of CO [1]. Other research indicates that ingestion of a gelatin-based drink containing "complex carbohydrates" is associated with increased CO and SBP and decreased TPR at baseline and increased HR reactivity to mental challenge [7]. These studies are clinically important because elevated autonomic nervous system and cardiovascular responses to mental challenge and delayed recovery have been linked to cardiovascular disease risk factors [8,9]. However, these studies did not control for perceived sweetness and the possibility for subsequent cephalic phase insulin release in responses to oral glucose intake. In addition, these investigations have used mental arithmetic and reaction time tasks and such tasks may have limited generalizability to real life stressors [10].

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The present study examined the effects of standardized oral glucose ingestion versus a taste-based control condition (i.e., sucralose) on cardiovascular responses to standardized mental challenge tasks in a laboratory setting. The control solution was used to ensure comparability in the perceived sweetness of the glucose and control solutions. The mental challenge protocol involved personally relevant structured speech tasks to evoke robust emotional and cardiovascular responses [10]. Using a fully counter-balanced within-subject design we tested the hypothesis that: (1) oral glucose results in altered resting levels of cardiovascular measures. Specifically it was expected that glucose would result in a reduction of the pre-ejection period (PEP), an indicator of sympathetic nervous system activity [11–14], (i.e., and increases in other cardiovascular measures associated with increased cardiac demand (HR, CO, SBP, DBP and TPR)). (2) Oral glucose results in exaggerated mental challenge-induced cardiovascular responses. We also explored whether glucose resulted in delayed recovery from the mental challenge tasks.

Methods

Participants

Participants aged 18–26 years were enrolled from a university-based community. Exclusion criteria were current use of medications known to alter cardiovascular function, body mass index (BMI) >30 kg/m^2, self-reported history of cardiovascular disease, diabetes, asthma, recent surgery/medical procedure, and any other major medical disorder. The sample was restricted to men only because adequate control for menstrual cycle variations was not feasible given the within-subjects design and the overall scope of the study. Participants with valid data for HR, blood pressure and impedance derived variables were included (N = 24/26; 92%). Demographic and average baseline cardiovascular indices are displayed in Table 1. The protocol was approved by the University of Maryland, Baltimore County's Institutional Review Board. All participants provided written informed consent and were paid $80 for taking part in the study.

Procedures

The protocol involved two testing sessions (one oral glucose administration and one control administration) separated by one week ($\pm$3 days). The glucose vs. control conditions were administered in a double-blind fashion and the order of administration (i.e., whether the first session involved glucose administration and the second session the control condition, or the reverse order) was randomized across participants. Participants were asked to fast overnight and abstain from caffeine and alcohol consumption for 24 h prior to the study. Two mental challenge tasks were used during each session (structured speech and anger recall).

Table 1
Participant characteristics

	Mean $\pm$ S.D. or N (%)
Demographic measures	
Age (years)	20.9 $\pm$ 2.4
Race	
Caucasian	16 (66.7%)
African American	5 (20.8%)
Asian	1 (4.2%)
Latino	2 (8.3%)
BMI (kg/m^2)	24.5 (2.2)
Baseline physiological measures	
PEP (m·s)	129.9 $\pm$ 12.1
HR (bpm)	63.4 $\pm$ 9.6
SI (mL/beat/m^2)	45.7 $\pm$ 15.7
CI (L/min/m^2)	2.7 $\pm$ 0.72
TPR (dyn/cm^2/s)	1300.0 $\pm$ 326.8
SBP (mm Hg)	114.4 $\pm$ 5.8
DBP (mm Hg)	58.5 $\pm$ 6.5
MAP (mm Hg)	80.6 $\pm$ 6.6

All experimental sessions started between 0800 and 1200 h and times of testing were kept consistent within participants ($\pm$1 h). Assessments were obtained in a sound-attenuated, climate controlled room. Participants provided written informed consent prior to being instrumented with the physiological monitoring equipment.

The protocol for each of the two sessions (glucose or control) consisted of six phases: (I) pre-ingestion of glucose or control: "baseline"; (II) post-ingestion resting levels; (III) SS; (IV) recovery SS; (V) AR; and (VI) recovery AR. Thus, data were collected at 12 time-points for each participant during two separate sessions (six glucose ingestions and six control-solution ingestions). During the pre-ingestion baseline period, participants remained quietly seated for 10 min after which participants completed baseline questionnaires. The oral test solution (glucose or control) was then administered. Participants were allowed 1 min to consume all of the test solution. Then, the 30-min post-ingestion resting period was started. The timing of physiological measures was based on prior results indicating that serum glucose and insulin levels are at their highest elevations after 30–60 min post glucose ingestion [1].

Physiological measures were obtained during the last 6 min of the baseline and post-ingestion (glucose or control), resting periods, continuously throughout the mental tasks, and during the 5 min immediately following the tasks (recovery periods). Task evaluation questionnaires were completed prior to and following each mental task.

Glucose and control conditions

A 20% glucose solution was presented at a dose of 1 g glucose per kg of body weight (Now Foods, Bloomingdale, IL). This dose yields significant changes in hemodynamic function [1].

The control condition consisted of a 2.5% sucralose solution (McNeil Nutritionals, Fort Washington, PA) at a volume matching that of the glucose test solution (1 mL of solution per 200 g of body weight). This 2.5% sucralose concentration was chosen based on pilot testing, indicating that participants could not discriminate this control solution from the glucose solution in terms of sweetness or palatability.

The glucose and control solutions were flavored with cherry sugar free Kool-Aid™ (2 g/L) (Kraft Foods, Rye Brook, NY). This flavor concentration was chosen because it was preferred from higher and lower concentrations in pilot testing. The test solutions were prepared by a research technician other than the experimenter prior to each experimental session and served chilled in a 16 oz. plastic cup. The test solution administrations were double blind and coded. Test solution codes were revealed to the experimenter only after all data collections of the project were completed.

Mental challenge tasks

Structured speech (SS)

The speech task involved presenting a speech in front of the experimenter. Speeches involved providing a convincing defense in a hypothetical scenario where the participant was falsely accused of a crime. Because of the repeated measures design, two scenarios were used (one scenario during each session and the presentation order of the scenarios was random and counterbalanced). One scenario involved being falsely accused of shoplifting by a plain-clothed policeman. The second scenario involved being stopped for speeding after running a stop sign that was not visible due to vegetation overgrowth. Participants were instructed to read the scenario, then prepare and recite a speech defending themselves to an imaginary

judge (the experimenter). The participants had 1 min to prepare the speech and 3 min to present it. SS tasks are well established as challenge tasks to elicit psychological distress and cardiovascular responses [15,16].

Anger recall (AR)

Participants described in detail a personally relevant anger-provoking event that occurred in the past two weeks. Participants were instructed to describe how they felt, what they said, did, and how others responded during the event. Consistent with the SS task, a unique event was recalled at each visit. One minute was given to identify the event and the verbal task lasted 3 min. This AR task was developed by Ironson et al. [17] and has been used in several studies examining mental-challenge induced cardiovascular reactivity [18–20].

Validation of experimental manipulations

Sweetness and hunger ratings were obtained before and after ingestion of glucose and control solutions using a 5-point rating scale in order to examine whether the tasks produced the anticipated effects on perceived sweetness and reduced hunger.

To evaluate the effectiveness of the mental challenge tasks in evoking affective responses, participants completed the Positive and Negative Affect Schedule (PANAS) [21]. The PANAS consists of 20 adjectives with the following 5-point response options: "very slightly or not at all", "a little", "moderately", "quite a bit", and "extremely". The PANAS is a well established tool for the evaluation of affect responses. For purposes of this study the Negative Affect scale was used (score range = 10–50). The PANAS Negative Affect subscale has good internal consistency (Cronbach's α ranging from 0.84 to 0.87) [21].

Physiological measures

Impedance cardiography

The electrical impedance of the thorax, ECG and heart sound data were collected continuously throughout the baseline and task periods using the Minnesota Impedance Cardiograph model 304B (Greenwich, CT). A tetrapolar band electrode configuration was used [11]. Experimental and pharmacological blockade studies have shown that the PEP can be used as a valid index of sympathetic nervous system activity [13,14] although substantial individual differences exist [11,14]. The participants' position was kept constant during the tasks to minimize artifacts of posture [22]. Placement of ECG electrodes was chosen to avoid interference with the impedance cardiograph electrodes. ECG and heart sound data were amplified and filtered via a Grass Biological Amplifier (Warwick, RI). The ECG and impedance wave data were sampled at 1000 samples per second in an analog format and then digitized prior to analysis.

The following measures were derived from the impedance cardiogram: PEP, the interval from the onset of ventricular depolarization to the beginning of mechanical contraction (expressed in m·s). HR was measured from the R–R interval of the ECG and presented in beats/minute (bpm). The stroke index (SI) was calculated as stroke volume divided by body surface area (BSA) and measured in milliliters per beat per m^2 ($mL/beat/m^2$). The cardiac index (CI) was defined as the blood volume ejected into the systemic circulation per minute divided by BSA (in $L/min/m^2$). TPR was used as an index of peripheral vascular resistance and calculated as ($TPR = MAP/CO \times 80$) expressed in $dynes/cm^2/s$ [11].

Blood pressure assessments

SBP, DBP, and mean arterial pressure (MAP) were measured oscillometrically using a Critikon Dinamap Vital Signs Monitor Model 8100 (Critikon, Tampa, FL). All blood pressure measurements were obtained from the participant's non-dominant arm. Blood pressure data were collected at 90 s intervals during the rest periods and at 60 s intervals during the task and recovery periods.

Statistical analysis

Data are presented as means ± standard deviations or percentages as appropriate. Effects of glucose on resting levels were examined using 2×2 repeated measures analysis of variance (ANOVA), with condition (glucose vs. placebo) and pre- vs. post-ingestion as within-subject factors. To evaluate the effects of acute glucose vs. control ingestion on cardiovascular reactivity to mental challenge, a 2×6 repeated measures multivariate ANOVA was used with two within-subjects factors: condition (glucose vs. control) and a 6-level experimental period (baseline, post-glucose/control ingestion, SS, recovery SS, AR, and recovery AR). Dependent measures were: PEP, HR, SI, CI, TPR, SBP and DBP (MAP was not included in the multivariate model to avoid singularity as a consequence of mathematically related variables). Missing values occurred in 4% of the total number of data points and were imputed using individualized substitution for the multivariate models only, to avoid listwise elimination of cases with missing values. Significant multivariate main effects and interaction terms were subsequently examined for 2×2 interaction terms (glucose vs. control × baseline vs. task) and simple effects to further explore differences between glucose vs. control on task responses for the dependent variables separately. Imputed values were only used for the multivariable models and the post hoc analyses were based on the actual values without imputation. Because of the within-subject design, standard adjustments for co-variates such as age and race were not used as subjects serve as their own control. Because BMI may change over time, associations with BMI at the time of glucose condition were analyzed. A P-value of .05 was considered statistically significant.

Results

Sample characteristics

Table 1 displays the participant characteristics and baseline values for the physiological data. Manipulation checks were consistent with the purposes of the protocol. Glucose resulted lower hunger ratings at 30 min following ingestion compared to participants receiving the control solution (glucose: 2.04 ± 1.42 vs. control: 2.58 ± 1.14, P = .039). The control condition was also well matched to the glucose condition in terms of perceived sweetness (sweetness glucose: 3.67 ± .92 vs. control: 3.42 ± 0.83; t (23) = 0.97, P = .34). BMI did not change during the course of the study (glucose session BMI = 24.54 ± 2.20 kg/m^2, control session BMI = 24.50 ± 2.29 kg/m^2; P = .66).

The mental challenge tasks induced the anticipated emotional responses, with increases in negative affect (from baseline = 13.8 ± 4.4 to SS = 15.2 ± 4.9 and AR = 17.6 ± 5.1; F (2,22) = 21.42 P<.001; aggregated across glucose and control sessions). No differential emotional responses were found for glucose vs. control (P value > .2).

The cardiovascular response patterns following glucose were significantly different from the control condition, as indicated by the multivariate repeated measures ANOVA for all dependent measures combined (glucose vs. control: F (7,17) = 3.08, P = .027; experimental period: F (35,565) = 6.13, P<.001; and glucose × experimental period interaction: F (35,565) = 1.74, P = .006). No effects of order of the condition (glucose vs. control) or mental challenge task (AR vs. SS) on baseline or task-induced cardiovascular measures were observed.

Effects of glucose on resting physiological measures

As shown in Fig. 1, PEP decreased following glucose ingestion (Δ = 11.98 ± 9.52 m·s; t (23) = 6.16 P<.001). The glucose-induced decrease in PEP was larger than the decrease following the control solution (Δ = 3.27 ± 7.65 m·s; t (23) = 2.09, P = .048; F interaction (1,23) = 20.17, P<.001). Although the baseline PEP levels were higher in the glucose than in the control condition (130.2 ± 11.9 vs. 125.3 ± 11.9 m·s, t (23) = 2.65, P = .014), the stronger effects of glucose vs. control on PEP were not attributable to these differences in baseline as reflected by the significant interaction term.

Glucose also resulted in an increase in CI, which did not occur in response to the control solution (Δ = 0.25 ± 0.41 $L/min/m^2$; t (23) = 2.97, P = .007 vs. Δ = 0.03 ± 0.29 $L/min/m^2$; t (23) = 0.44, P = .67; F interaction (1,23) = 4.72, P = .040). HR responses were also different across conditions, showing no response to glucose and a decline in the control condition (Δ = 0.69 ± 3.91 bpm; t (23) = 0.86, P = .40 vs. Δ = 1.73 ± 3.02 bpm; t (23) = 2.80, P = .010; F interaction (1,23) = 7.63, P = .011). Glucose effects on SI were non-significant.

As shown in Fig. 2, the glucose condition resulted in increased SBP whereas no effect of the control solution was observed (Δ = 3.22 mm Hg; t (23) = 3.17, P = .004 vs. Δ =

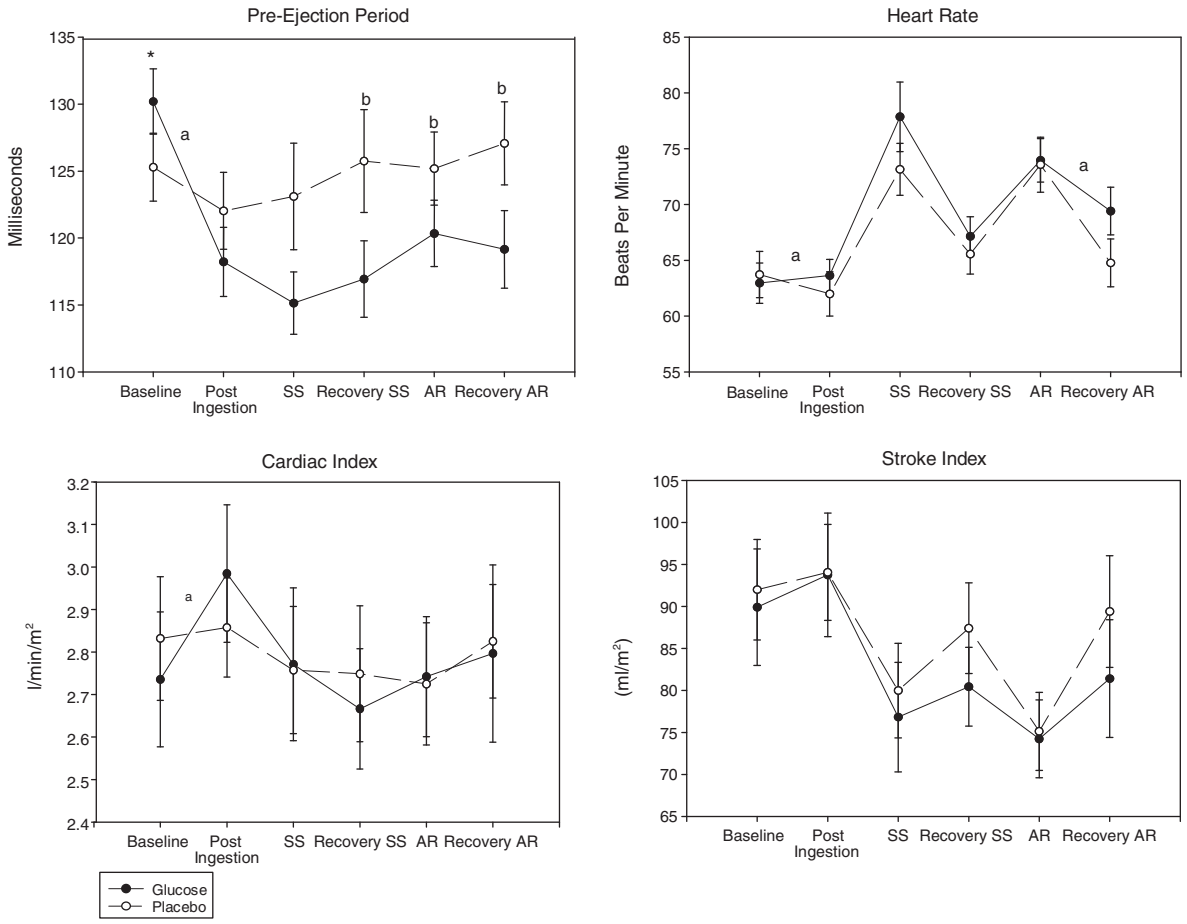

Fig. 1. Effects of glucose on measures of impedance cardiography-based measures of cardiac function. Responses to glucose (solid lines) and control (dashed lines) are shown for: pre-ejection period (PEP), heart rate (HR), cardiac index (CI), and stroke index (SI). PEP is an index of autonomic nervous system activity, with shifts toward lower values indicating sympathetic nervous system activation. Study phases included pre-ingestion baseline; resting levels 30 min post ingestion of glucose or control; structured speech (SS); post-SS recovery; anger recall (AR); post-AR recovery. All tasks resulted in a significant change from baseline values. *=Significant difference at baseline. a=Significant interaction between task and the preceding task. b=Significant interaction between task and baseline.

0.29 mm Hg; t (23)=0.32, P=.75; F interaction (1,23)=8.05, P=.009). No differential effects of glucose on DBP, MAP or TPR levels were found (P interaction>.10).

The differential effects of glucose vs. control on PEP, CI, HR and SBP were not related to the order of the conditions (i.e., glucose in the first session and control in the second session). BMI was also not associated with the different response in glucose vs. control in PEP (r=0.04, P=.87), CI (r=−0.09, P=.67), HR (r=0.15, P=.48) or SBP (r=0.12, P=.57).

Effects of glucose on mental challenge-induced physiological reactivity

The effects of glucose on cardiovascular measures were primarily observed in the initial 30-min responses to glucose vs. control and no additional mental challenge-induced differences were noted (P values from 30-min post-ingestion to SS or AR levels>.10 for all measures). The strongest effect was observed for HR during SS, which tended to increase more in the glucose condition compared to control (F (1,23)=3.28. P=.083).

Recovery pattern analyses revealed a delayed HR recovery following AR following glucose compared to control (Δ=6.29±6.86; t (20)=4.10, P=.001 vs. Δ=10.00±4.80; t (20)=9.54, P<.001; F interaction (1,17)=5.48, P=.032). This difference in HR recovery was not significantly associated with BMI (r=0.26, P=.31).

Discussion

The present investigation suggests that glucose ingestion results in decreased PEP, increased CI, HR, and SBP during resting conditions compared to a control condition. The PEP responses are likely to reflect increased sympathetic nervous system activation and the other cardiovascular responses may reflect a combination of sympathetic activation and parasympathetic withdrawal. Both mental challenge tasks resulted in significant cardiovascular responses from baseline values but the magnitude of reactivity did not differ between glucose versus control conditions. Results further suggested that glucose resulted in a delayed post-challenge HR recovery.

The effect of carbohydrates on SBP, HR, and CO is well documented and reflects increased demands for digestive processes [1,23–25]. Elevations in CO and BP in response to carbohydrates have been attributed, in part, to vasodilatation associated with insulin secretion and sympathetically mediated increases in HR [1]. Further, Kopp [2] has speculated that excessive sympathetic activation in response to glucose loading may promote the development of essential hypertension via vascular remodeling ultimately resulting in increased peripheral vascular resistance. The present study did not find effects of glucose on TPR, which may partially reflect the relatively young age and good physical health of the study participants. Additional research is needed to examine responses to glucose in the presence of sustained abnormalities in glucose metabolism, which may reveal stronger associations between perturbation tasks and cardiovascular dysregulation.

No differential effects of glucose versus control were found on cardiovascular responses to the mental challenge tasks. However, HR recovery following the AR task was delayed after glucose ingestion. Prolonged cardiovascular recovery from mental challenge has been associated with risk for the development of cardiovascular disease, as well as future subclinical alterations in cardiovascular function. For example, delayed recovery of cardiovascular responses to mental

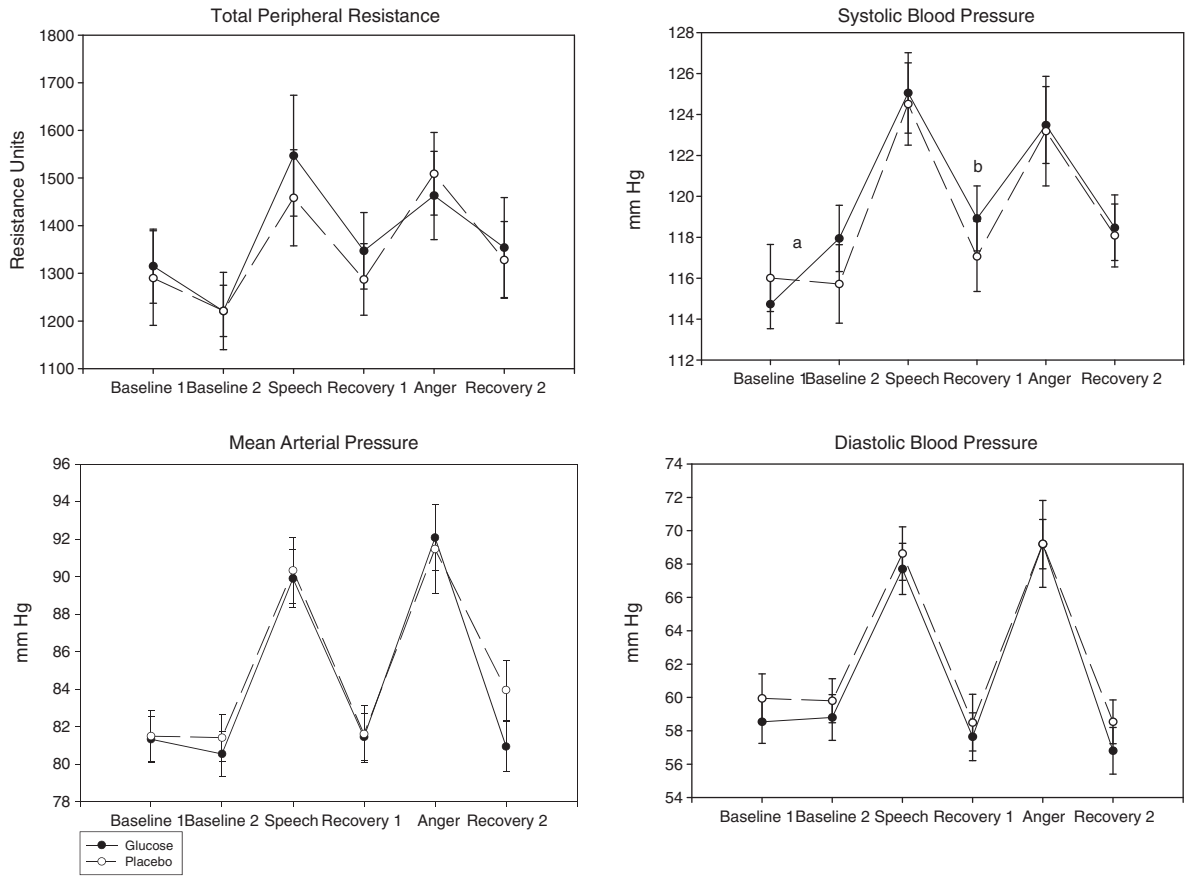

Fig. 2. Effects of glucose on total peripheral resistance and blood pressure. Responses to glucose (solid lines) and control (dashed lines) are displayed for TPR, SBP, DBP and MAP. Study phases include pre-ingestion baseline; resting levels 30 min post ingestion of glucose or control; structured speech (SS); post-SS recovery; anger recall (AR); post-AR recovery. All tasks resulted in a significant change from baseline values. a = Significant interaction between task and the preceding task. t = Trend for group difference ($P<.10$).

challenge is associated with family history of cardiovascular disease [26,27]. Other results indicate that slowed cardiovascular recovery to mental challenge predicts increased blood pressure three years later [28,29]. Poor HR recovery has also been associated with low HR variability (HRV), decreased vagal tone and relative sympathetic dominance in cardiac control which is characteristic of the early stages of essential hypertension [30] as well as diabetes mellitus [31–33]. Future studies examining both sympathetic and parasympathetic responses to glucose administration are important because autonomic dysregulation is associated with increased fasting glucose and elevated HbA1c levels [34].

The present study has several strengths and limitations. The strengths include the matching of glucose and control test solutions for sweetness and the fully within subjects design as well as the counterbalancing of test solution and speech scenario presentation order. One of the limitations concerns the restriction of the study sample to male participants. Another limitation is the lack of measurement of circulating glucose and insulin levels. Participants experienced more hunger in response to glucose and despite the successful matching for perceived sweetness it is possible that cephalic phase insulin release has influenced the results. In addition, the small sample size may further limit generalizability of the findings. We conducted an a priori power analyses based on effect sizes reported in previous studies [1], which indicated that the sample of the present study was more than sufficient to detect the expected effects at $\alpha = 0.05$ with a power of $>80\%$.

In summary, the results of this study suggest that acute glucose ingestion has potent effects on resting cardiac function as reflected in decreased PEP, and increased SBP, HR, and CI. This study did not reveal effects of glucose on stress-induced cardiovascular responses to mental-challenge laboratory tasks. Glucose ingestion may result in delayed HR recovery from mental challenge. Sweet tasting foods and liquids affect insulin release [35], but how these effects relate to cardiovascular reactivity and recovery is still unknown. Future investigations are needed to establish the effects of acute carbohydrate ingestion on cardiovascular function and cardiovascular reactivity and recovery by contrasting the effects of sugars vs. carbohydrates that contain fiber and/or saturated and unsaturated fat. Elevated sympathetic nervous system-mediated increases in resting cardiovascular parameters following glucose intake may contribute to dysregulated vascular biology and the development of essential hypertension and other adverse cardiovascular health outcomes.

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Conflict of interest

The authors have no conflicts of interest to disclose.

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