Salt Loading Blunts Central and Peripheral Postexercise Hypotension

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ABSTRACT

BABCOCK, M. C., A. T. ROBINSON, J. C. WATSO, K. U. MIGDAL, C. R. MARTENS, D. G. EDWARDS, L. S. PESCATELLO, and W. B. FARQUHAR. Salt Loading Blunts Central and Peripheral Postexercise Hypotension. *Med. Sci. Sports Exerc.*, Vol. 52, No. 4, pp. 935–943, 2020. **Introduction:** High salt intake is a widespread cardiovascular risk factor with systemic effects. These effects include an expansion of plasma volume, which may interfere with postexercise hypotension (PEH). However, the effects of high salt intake on central and peripheral indices of PEH remain unknown. We tested the hypothesis that high salt intake would attenuate central and peripheral PEH. **Methods:** Nineteen healthy adults (7 female/12 male; age, 25 ± 4 yr; body mass index, 23.3 ± 2.2 $kg \cdot m^{-2}$; $\dot{V}O_{2peak}$, 41.6 ± 8.7 $mL \cdot min^{-1} \cdot kg^{-1}$; systolic blood pressure (BP), 112 ± 9 mm Hg; diastolic BP, 65 ± 9 mm Hg) participated in this double-blind, randomized, placebo-controlled crossover study. Participants were asked to maintain a 2300 $mg \cdot d^{-1}$ sodium diet for 10 d on two occasions separated by ≥2 wk. Total salt intake was manipulated via ingestion of capsules containing either table salt (3900 $mg \cdot d^{-1}$) or placebo (dextrose) during each diet. On the 10th day, participants completed 50 min of cycling at 60% $\dot{V}O_{2peak}$. A subset of participants (n = 8) completed 60 min of seated rest (sham trial). Beat-to-beat BP was measured in-laboratory for 60 min after exercise via finger photoplethysmography. Brachial and central BPs were measured for 24 h after exercise via ambulatory BP monitor. **Results:** Ten days of high salt intake increased urinary sodium excretion (134 ± 70 (dextrose) vs 284 ± 74 mmol per 24 h (salt), $P < 0.001$), expanded plasma volume (7.2% ± 10.8%), and abolished PEH during in-laboratory BP monitoring (main effect of diet, $P < 0.001$). Ambulatory systolic BPs were higher for 12 h after exercise during the salt and sham trials compared with the dextrose trial (average change, 3.6 ± 2.1 mm Hg (dextrose), 9.9 ± 1.4 mm Hg (salt), 9.8 ± 2.5 mm Hg (sham); $P = 0.01$). Ambulatory central systolic BP was also higher during the salt trial compared with dextrose trial. **Conclusion:** High salt intake attenuates peripheral and central PEH, potentially reducing the beneficial cardiovascular effects of acute aerobic exercise. **Key Words:** BRACHIAL BLOOD PRESSURE, CENTRAL BLOOD PRESSURE, SALT, AEROBIC EXERCISE, POSTEXERCISE HYPOTENSION

Aerobic exercise training is a commonly prescribed early intervention for individuals with elevated blood pressure (BP) (1) and effectively reduces BP even in individuals with resistant hypertension (2). This benefit of aerobic exercise is of increasing importance, as recent guidelines from the American Heart Association and American College of Cardiology for evaluation of high BP (3) indicate that nearly 50% of US adults have hypertension and recommend antihypertensive lifestyle strategies such as dietary sodium restriction and aerobic exercise (4). Importantly, even a single bout of aerobic exercise has been shown to lower BP by 5–8 mm Hg, a magnitude similar to those reductions observed after long-term exercise training (5). Furthermore, the reductions in BP observed after a single bout of exercise are associated with training-induced reductions in BP (6,7), indicating that at least some of the benefits of aerobic exercise on BP status are the result of acute reductions in BP resulting from a recent bout of exercise (5).

This immediate reduction in BP after a bout of exercise is termed postexercise hypotension (PEH) and is an accepted physiological response to a bout of exercise (8) that provides important prognostic information. For example, BP responses after exercise are linked to future cardiovascular (CV) disease risk (9), such that those with a smaller reduction in BP after aerobic exercise are at greater risk of developing CV disease. In addition, acute submaximal aerobic exercise produces a prolonged period of PEH (up to 24 h), offering the benefit of having lower BP for the majority of the day when BP is at its highest (10–12).

Importantly, acute submaximal aerobic exercise may also produce short-term reductions in arterial stiffness (13). Arterial stiffness plays an important role in modulating central systolic BP (cSBP) via changes in pressure wave magnitude and transmission velocity. End organs including the heart and kidneys are exposed to central, rather than brachial, pressure; therefore,

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cSBP, rather than brachial BP, has been shown to be more closely related to CV events (14). Thus, the benefits of acute submaximal aerobic exercise may also be conferred upon the central circulation, lowering cSBP and reducing the workload of the heart.

Excess dietary salt intake is a widespread CV disease risk factor, with more than 90% of Americans consuming more than is recommended (15). In fact, daily intake averages ~3400 mg $Na^+ \cdot d^{-1}$ (16) despite widespread recommendations that sodium intake be limited to 2300 mg $Na^+ \cdot d^{-1}$ (16). Excess salt intake induces systemic CV consequences including endothelial dysfunction (17–19) and increased arterial stiffness (20,21). In addition, high dietary salt increases plasma volume (22). Because PEH is characterized by a persistent vasodilation that is not matched by increases in cardiac output (CO) (10,23–26), salt-induced changes in vascular function and fluid balance may adversely affect PEH. Little is known about the influence of excess dietary salt on PEH assessed in both the periphery and the central arteries.

Therefore, the purpose of this study was to evaluate the influence of high salt intake on PEH. We hypothesized that excess salt intake would expand plasma volume and subsequently attenuate the PEH induced by acute submaximal aerobic exercise. In addition, we sought to determine the effects of high salt intake on central BP; we hypothesized that 10 d of high salt would blunt central measures of PEH.

METHODS

The data reported herein were collected as a part of a registered clinical trial (ClinicalTrials.gov Identifier: NCT03565653). All participants provided written and verbal consent before participating. The institutional review board of the University of Delaware approved all study protocols and procedures, and they conform to the provisions of the Declaration of Helsinki.

Study participants. All participants underwent a medical history screening including a report of habitual physical activity. Height (cm) and body mass (kg) were measured for calculation of body mass index (BMI; $kg \cdot m^{-2}$). Seated BP was measured via oscillometric assessment in triplicate after ≥5 min of seated rest (Dash 2000, GE Medical Systems). The average of the triplicate measures is reported here. Participants also completed the Physical Activity Readiness Questionnaire (27). Participant age ranged from 18 to 34 y, and all participants were recreationally active (self-reported regular exercise participation ≥3 $d \cdot wk^{-1}$). Other exclusion criteria included a history of hypertension diagnosis, CV disease, malignancy, diabetes mellitus, renal dysfunction, current pregnancy, obesity (BMI > 30 $kg \cdot m^{-2}$), and use of nicotine products.

$\dot{V}O_{2peak}$. After the screening visit, participants reported to the Metabolic Stress Testing Laboratory in the Nurse Managed Primary Care Center at the University of Delaware to undergo a maximal cardiopulmonary exercise test. Participants performed cycling exercise to volitional fatigue (Lode CPET; Lode, Groningen, the Netherlands) using a ramped protocol that has been described previously (28). Briefly, participants began cycling at a constant power of 30 W for 3 min. After this 3-min warm-up, power increased by 1 W every 2 s until participants were unable to maintain a pedaling cadence of 60 rpm. Throughout the exercise test, oxygen consumption and carbon dioxide production were measured and averaged in 15-s intervals using indirect calorimetry via an automated open-circuit system (Parvo Medics, Sandy, UT). Before the test, the gas analyzers were calibrated with standardized gasses (16% O_2, 4.05% CO_2), and the pneumotach was calibrated using a standard volume of air (3 L). Heart rate (HR) was monitored via an HR monitor (Polar H7, Polar, USA).

After the $\dot{V}O_{2peak}$ test, participants rested for 10–15 min. During this rest period, participants completed the Automated Self-Administered 24-h dietary recall. After completing the Automated Self-Administered 24-h dietary recall, participants resumed cycling and the workload corresponding to 60% $\dot{V}O_{2peak}$ was determined by $\dot{V}O_2$ measurement. This workload was used on the study day for the exercise bout during experimental visits (described hereinafter).

Sodium intervention. Participants completed a double-blind, placebo-controlled, crossover study. For 10 d, participants were asked to consume a recommended (2300 mg $Na^+ \cdot d^{-1}$) sodium diet. We provided participants with instructions for interpreting nutrition labels and lowering sodium intake. Participants also consumed unmarked capsules each day containing either salt (Morton® table salt (NaCl); 3900 mg $Na^+ \cdot d^{-1}$) or a placebo (dextrose; NOW Foods® dextrose). Total sodium intake during the high-salt condition was designed to be 6200 $mg \cdot d^{-1}$, and that during the dextrose condition was 2300 $mg \cdot d^{-1}$. Each participant completed both conditions in random order separated by ≥2 wk. All female participants were tested during the placebo week of hormonal birth control; visits for female participants were separated by 4 wk. Participants recorded their diet during the first intervention and were asked to match their diet during the second intervention. A copy of their diet log was provided to serve as a menu. On the 10th day of each intervention, participants reported to the Cardiovascular Physiology Laboratory at the University of Delaware for their experimental visit. For 24 h before the experimental visit, participants collected their urine (see hereinafter).

Twenty-four-hour urine collection. Urine was collected during the final 24 h of both diets in a light-protected, sterile 3500-mL container. Participants returned the 3500-mL container upon arriving to the laboratory for the experimental visit. We measured total urine volume, urine specific gravity (Goldberg Brix Refractometer, Reichert Technologies), urinary electrolyte concentrations (EasyElectrolyte Analyzer, Medica), and osmolality (Advanced 3D3 Osmometer, Advanced Instruments) from a mixed aliquot from the 24-h collection container. Urine flow rate was calculated and used to determine 24-h sodium excretion. Participants were instructed to abstain from alcohol, caffeine, antihistamines, and vigorous exercise for the 24-h before and during the 24-h urine collection.

Study visit. On the 10th day of each diet, participants reported to the Cardiovascular Physiology Laboratory at

the University of Delaware. Upon arrival, participants provided a spot urine sample for assessment of hydration status via urine specific gravity (Goldberg Brix Refractometer, Reichert Technologies). Body weight and total body water were measured (Tanita Body Composition Analyzer, Model TBF-300A; Arlington Heights, IL). Samples from female participants were tested using hormonal pregnancy tests (Moore Medical) to ensure that female participants were not pregnant. Participants laid supine and were instrumented for single-lead ECG and oscillometric BP measurements at the upper arm (right arm; Dash 2000, GE Medical Systems). After ≥15 min of supine rest, a venous blood sample was collected (described hereinafter).

Beat-to-beat BP was recorded via finger photoplethysmography (Finometer, Finapres Medical Systems) at the middle finger of the left hand, which was supported at heart level. Systolic BP and diastolic BP were defined as the maximal and minimal value from the arterial BP waveform during each cardiac cycle, respectively. Mean BP was calculated as the integral of the BP waveform. Total peripheral resistance (TPR) and CO were estimated from the arterial BP waveform using the Modelflow method (29). Brachial BP was recorded from the right arm via an automated oscillometric sphygmomanometer (Dash 2000, GE Medical Systems).

Exercise protocol and PEH assessment. After instrumentation, participants rested quietly in a dimly-lit room for 10 min while undergoing continuous recordings of beat-to-beat BP and HR. After this period of supine rest, participants were moved to an upright cycle ergometer (Lode Excaliber; Lode). Seated brachial BP was measured in triplicate via auscultation and used to calibrate the beat-to-beat BP signal.

After seated BP measures, participants began submaximal aerobic exercise with a 5-min warm-up period at self-selected resistance (matched between conditions). The resistance was then increased to the power output determined during the $\dot{V}O_{2peak}$ test (described previously) for 50 min. After 50 min of exercise at 60% $\dot{V}O_{2peak}$, participants completed five additional minutes of aerobic exercise at self-selected resistance as a cool down. Exercise workloads for warm-up, exercise, and cool down were matched between conditions. Participants pedaled at their preferred cadence, and resistance was continuously adjusted to maintain a constant power output. Submaximal aerobic exercise at 60% of $\dot{V}O_{2peak}$ has been reported to produce PEH in studies in male and female participants with normal and high BP of all ages (10–12,30,31). Participants were permitted to drink water *ad libitum* during the first exercise trial, and water intake during exercise was matched for the second exercise trial.

After exercise, beat-to-beat BP and HR were monitored continuously during quiet supine rest for 60 min. In addition, brachial BP (automated oscillometry) was recorded every 10 min. A subset (n = 12) of participants underwent ambulatory BP monitoring for 24 h after exercise (Oscar 2 with SphygmaCor, SunTech Medical). The monitor measured BP on the left arm every 20 min from 0601 to 2200 h and every 30 min from 2201 to 0600 h. This device has been validated for brachial BP measurement (32) and uses the same algorithm as tonometry-based SphygmaCor products and therefore is expected to provide aortic pressure with similar accuracy (33). The SunTech Oscar 2 ambulatory BP monitors also derive the central aortic pressure waveform from pressure waves recorded from the brachial artery and report central BP with each brachial BP recording. On a separate occasion separated from either exercise visit by ≥1 wk, 8 of the 12 participants who underwent ambulatory BP monitoring also completed a sham exercise condition (1 h of quiet seated rest) followed by ambulatory BP monitoring. Before the sham visit, participants consumed their habitual salt intake. The change in BP from preexercising levels was evaluated for 12 h after exercise or seated rest and reported herein.

Biochemical analysis. Serum electrolytes (EasyElectrolyte Analyzer, Medica), plasma osmolality (Advanced 3D3 Osmometer, Advanced Instruments), hemoglobin (Hb 201+, HemoCue), and hematocrit (Sure prep™ capillary tubes, Clay Adams spun in a microcentrifuge at 1950*g* for 5 min, Legend Micro 17, Thermo Sorvall) were measured. The change in plasma volume between conditions was calculated using the following equation, which is based on changes in hemoglobin concentration and hematocrit between conditions (34):

$$\text{plasma volume (\%)} = (100 \times (\text{Hb}_{\text{dextrose}}/\text{Hb}_{\text{salt}})) \times (1 - (\text{Hct}_{\text{salt}}/100))/(1 - \text{Hct}_{\text{dextrose}}/100) - 100$$

Remaining venous blood samples were spun at 750*g* for 15 min at 4°C (Allegra X-22R, Beckman Coulter). Plasma samples were stored at −80°C for future analysis.

Data and statistical analysis. Beat-to-beat BP and ECG signals were recorded continuously (LabChart Pro 8, AD Instruments) at 1000 Hz and stored for offline analysis. During the hour of in-laboratory BP monitoring after exercise, beat-to-beat and brachial BPs were averaged over 10-min intervals. PEH was determined by subtracting resting BP from postexercise BP. Ambulatory BPs were averaged over 60-min intervals and compared with the sham exercise condition. When considering ambulatory BPs, PEH was determined by comparing postexercise BP with the sham exercise condition, as described previously (5,30,35,36). Pulse pressure amplitude ratio was calculated as brachial pulse pressure/central pulse pressure.

Spontaneous cardiovagal baroreflex sensitivity (cBRS) and HR variability (HRV) were calculated as indices of autonomic function. Beat-to-beat series of R-R interval and systolic BP were analyzed using the sequence method for estimating spontaneous cBRS (HemoLab version 8.9, Herald Stauss Scientific), as described in detail previously (37). Sequences of four or more consecutive cardiac cycles in which R-R interval and systolic BP change in the same direction were identified as baroreflex sequences. Changes in R-R interval of >0.5 ms and systolic BP changes of >1 mm Hg were required for detection. A linear regression was applied to individual sequences, and only those sequences in which $R^2 > 0.80$ were accepted. The slopes of individual linear regressions were averaged for a measure of spontaneous cBRS. HRV (Kubios version 3.3, Biosignal Analysis and Medical Imaging Group) was assessed

TABLE 1. Participants.

Participant Characteristics	
n (F/M)	19 (7/12)
Race (AA/A/H/C)	1/4/2/12
Age, yr	25 ± 4
Height, cm	153 ± 26
Body mass, kg	70 ± 13
BMI, $kg \cdot m^{-2}$	23.3 ± 2.2
Systolic BP, mm Hg	112 ± 9
Diastolic BP, mm Hg	65 ± 9
Mean BP, mm Hg	81 ± 9
Sodium intake, mmol per 24 h	136 ± 43

Data are mean ± SD.
A, Asian; AA, African American; C, Caucasian; F, female; H, Hispanic; M, male.

using R-R intervals from the ECG recordings. Power spectral analysis using fast Fourier transformation was performed, and HRV was evaluated in the frequency domain using high-frequency/low-frequency (HF/LF) ratio (38).

The statistical approaches reported here were informed by recent guidelines for statistical reporting of CV research (39). The differences in Δsystolic BP, Δmean BP, and Δdiastolic BP after submaximal cycling exercise were examined using a generalized linear mixed-model analysis with repeated measures for diet and time. In the case of a significant effect (time, diet, or interaction), Tukey's *post hoc* analysis was performed. The effects of salt pills on blood and urine measures were tested using two-tailed paired *t*-tests. Plasma volume expansion was tested using a one-sample *t*-test. Statistical significance was set at $P < 0.05$, and results are expressed as mean ± SD. Statistical analyses were completed using GraphPad Prism version 8.0.

RESULTS

Participant characteristics are reported in Table 1. Participants were recreationally active ($\dot{V}O_{2peak}$, 41.6 ± 8.7 $mL \cdot min^{-1} \cdot kg^{-1}$), were nonobese, and had nonhypertensive BP. Table 2 demonstrates the effects of the salt pill intervention. Although participants were asked to eat a recommended (2300 $mg \cdot d^{-1}$) sodium diet, 24-h urinary sodium excretion during the dextrose condition suggested that they consume ~3400 $mg \cdot d^{-1}$, which is more similar to average sodium intake among Americans. Ten days of salt pills increased urinary sodium excretion and expanded the plasma volume by 7.2% ± 10.8% versus dextrose ($P = 0.01$; estimated from changes in hemoglobin and hematocrit). Total body water (assessed via bioelectrical impedance analysis) and weight were increased after salt compared with dextrose. Serum sodium concentrations and plasma osmolality were not different between conditions. Overall cBRS at baseline was not different between conditions (25.0 ± 11.6 $ms \cdot mm\ Hg^{-1}$ (dextrose) vs 29.0 ± 13.3 $ms \cdot mm\ Hg^{-1}$ (salt); $P = 0.48$). Similarly, the LF/HF ratio was not different during the preexercise baseline (0.65 ± 0.46 (dextrose) vs 0.67 ± 0.38 (salt); $P = 0.96$). Although the LF/HF ratio was increased after exercise (main effect of time, $P < 0.01$), this increase was not different between dietary conditions (main effect of diet, $P = 0.49$).

Average exercise workload was 122 ± 35 W. Baseline HR was similar between dietary conditions (dextrose, 57 ± 9 bpm; salt, 55 ± 9 bpm; $P = 0.42$) and increased similarly during exercise (dextrose, 143 ± 22 bpm; salt, 141 ± 17 bpm; main effect of exercise, $P < 0.001$; main effect of diet, $P = 0.57$). This increase represented, on average, 61% ± 4% of HR reserve and was consistent with the target workload.

Figure 1 demonstrates the changes in beat-to-beat systolic, mean, and diastolic BP measured in-laboratory over 60 min after submaximal aerobic exercise. Systolic BP was reduced (mean change, −4.8 ± 2.4 mm Hg) during in-laboratory BP monitoring after exercise on day 10 of dextrose; however, this effect was abolished after 10 d of salt capsules (mean change, 5.3 ± 3.9). Mean (−0.2 ± 1.6 mm Hg (dextrose) vs 2.0 ± 2.4 mm Hg (salt)) and diastolic (−0.1 ± 1.5 mm Hg (dextrose) vs −0.4 ± 1.4 mm Hg (salt)) BP were not different between dietary conditions. Similar to beat-to-beat BP, brachial systolic BP was reduced after exercise after dextrose but not salt (main effect of diet, $P = 0.02$), whereas mean (main effect of diet, $P = 0.24$) and diastolic (main effect of diet, $P = 0.68$) BPs were not different between conditions (data not shown). HR was increased for 60 min after exercise compared with preexercising levels after both dextrose (mean change, 12.2 ± 8.5 bpm) and salt (mean change, 9.9 ± 8.3 bpm); however, there were no differences between dietary conditions (main effect of time, $P < 0.001$; main effect of diet, $P = 0.43$). Under both dietary conditions, Modelflow-derived estimates of CO remained elevated after exercise compared with preexercising values; however, CO was modestly higher after salt (mean change from preexercise, 1.2 ± 0.4 l per minute) compared with dextrose (mean change from preexercise, 0.9 ± 0.4) capsules (Fig. 2A). Modelflow-derived estimates of TPR were reduced similarly in both dietary conditions after exercise compared with dextrose (Fig. 2B).

Brachial ambulatory BP measures of participants who underwent 24-h BP monitoring are presented in Figure 3. After a nonexercise sham condition, ambulatory systolic BP increased throughout the day. One hour of submaximal aerobic exercise after habitual salt intake attenuated the rise in systolic BP; however, 10 d of high salt intake prevented the BP-lowering effects of exercise (mean change in systolic BP, 9.8 ± 2.5 mm Hg (sham), 9.9 ± 1.4 mm Hg (salt), 3.6 ± 2.1 mm Hg (dextrose); $P < 0.01$). There was a trend ($P = 0.10$) for ambulatory diastolic

TABLE 2. Effects of salt capsules.

Measure	Dextrose	Salt	*P*
Body mass, kg	**69.6 ± 12.2**	**70.1 ± 12.6**	**0.03**
Total body water, kg	**40.9 ± 7.8**	**41.5 ± 2.3**	**0.02**
Systolic BP, mm Hg	109 ± 9	108 ± 9	0.97
Diastolic BP, mm Hg	61 ± 9	61 ± 4	0.79
Mean BP, mm Hg	77 ± 9	77 ± 4	0.82
Urinary Na^+ excretion, mmol per 24 h	**135 ± 70**	**284 ± 74**	**<0.001**
Plasma Osm, $mOsm \cdot kg\ H_2O^{-1}$	294 ± 4	294 ± 4	0.58
Serum Na^+, $mmol \cdot L^{-1}$	141.4 ± 2.2	141.4 ± 1.7	0.68
Serum K^+, $mmol \cdot L^{-1}$	4.14 ± 0.48	4.09 ± 0.48	0.64
Serum Cl^-, $mmol \cdot L^{-1}$	**104.8 ± 1.7**	**106.2 ± 1.7**	**0.02**
Hemoglobin, $mg \cdot dL^{-1}$	**13.5 ± 1.0**	**12.8 ± 1.4**	**<0.01**
Hematocrit, %	42.4 ± 2.6	41.5 ± 3.9	0.31

Data are presented as mean ± SD. Bold font indicates significant difference.
Cl^-, chloride; K^+, potassium; Na^+, sodium; Osm, osmolality.

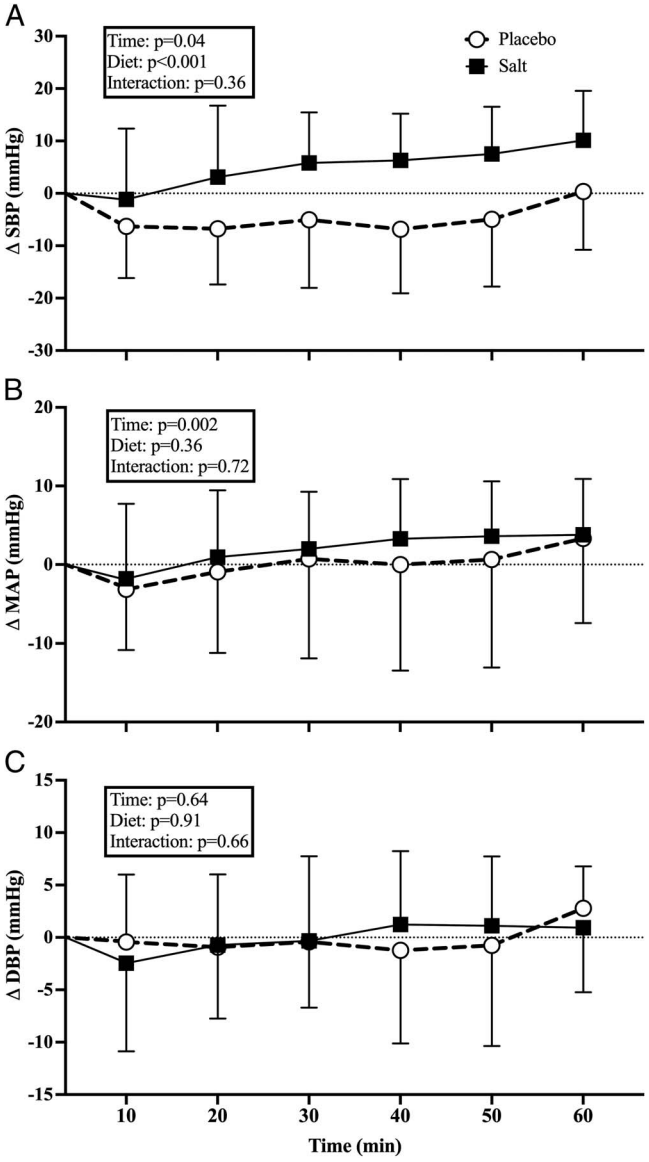

FIGURE 1—Changes in systolic (A), mean (B), and diastolic (C) BP from baseline during recovery from submaximal aerobic exercise. After 10 d of habitual salt intake plus dextrose capsules (*open circles*), systolic BP is reduced compared with preexercising levels. However, after 10 d of high dietary salt intake (*closed squares*), this effect of exercise is abolished. Data are presented as mean ± SD. *$P < 0.05$ between diets.

BP to increase throughout the day; however, this did not reach statistical significance (Fig. 3). There was no difference in the average change in diastolic pressure between conditions (sham, 7.4 ± 3.3 mm Hg; salt, 7.4 ± 1.2 mm Hg; dextrose, 6.1 ± 2.2 mm Hg; $P = 0.34$). Similarly, Figure 4 demonstrates that 1 h of aerobic exercise reduced the increase in cSBP that occurred after an hour of seated rest; an effect that was attenuated when exercise was performed after 10 d of salt capsules (mean change in cSBP, 4.0 ± 2.4 mm Hg (sham), 2.4 ± 2.8 mm Hg (salt), 0.7 ± 2.4 mm Hg (dextrose); $P = 0.02$). There was a trend for the same effect on cDBP; however, these values did not reach statistical significance (mean change in cDBP, 3.5 ± 1.8 mm Hg (sham), 3.0 ± 2.5 mm Hg (salt), 1.3 ± 3.7 mm Hg (dextrose); $P = 0.10$). Brachial (sham, 50 ± 3 mm Hg; salt, 45 ± 7 mm Hg; dextrose, 46 ± 6 mm Hg; $P = 0.29$) and central (sham, 36 ± 2 mm Hg; salt, 33 ± 5 mm Hg; dextrose, 32 ± 5 mm Hg; $P = 0.15$) pulse pressures were not different between conditions. Pulse pressure amplification ratio was increased after a bout of acute submaximal exercise on the dextrose condition (1.47 ± 0.09) compared with sham exercise (1.37 ± 0.07), but not following the salt condition (1.39 ± 0.07; main effect of diet, $P = 0.01$).

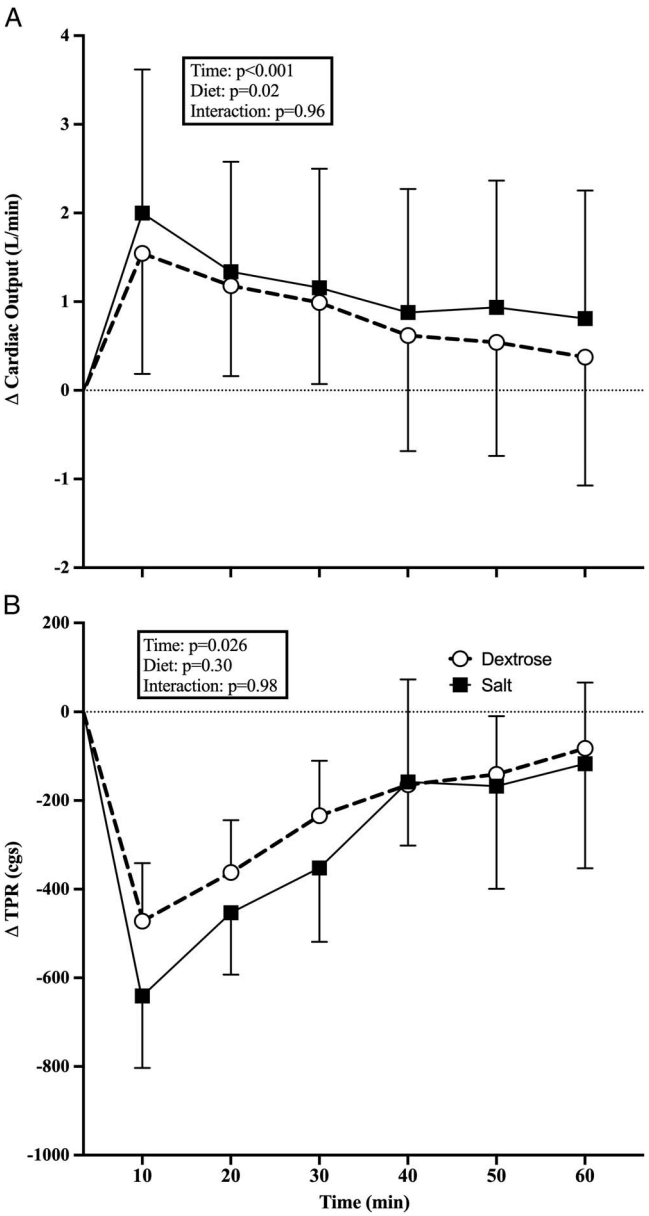

FIGURE 2—Changes in Modelflow-derived estimates of CO (A) and TPR (B) from baseline during recovery from submaximal aerobic exercise. CO remains elevated after submaximal aerobic exercise; however, CO after exercise is modestly but significantly higher after 10 d of salt capsules (*filled squares*) compared with dextrose (*open circles*). Changes in TPR were similar regardless of dietary condition. Data are presented as mean ± SD.

APPLIED SCIENCES

DISCUSSION

The novel findings of the present study were that 1) PEH generated via 60 min of submaximal aerobic exercise is also observed in cSBP, 2) high dietary salt intake abolishes the peripheral PEH generated via 60 min of submaximal aerobic exercise, and 3) high dietary sodium intake reduced central PEH. To our knowledge, this is the first study to investigate the influence of submaximal aerobic exercise on central indices of BP. The findings presented herein are similar to previous findings, in that submaximal aerobic exercise produced a prolonged period of reduced systolic BP measured at the brachial artery; however, we have now extended these observations to include central BP.

The findings of the present study are noteworthy in light of the current estimates that nearly half of American adults have high BP (3) and should engage in lifestyle modifications such as exercise to improve CV health (4). The data presented herein suggest that aerobic exercise may be rendered ineffective in acutely lowering systolic BP in the presence of a high-salt diet. These data highlight the need for comprehensive lifestyle (i.e., both diet and exercise) modifications to optimize CV health and reduce the burden of hypertension.

PEH is characterized by a reduction in TPR that is not completely offset by an increased CO (10,23–26). High dietary sodium intake increases plasma volume (22), which may subsequently augment CO and offset the reduction in TPR that follows acute aerobic exercise. Changes in hemoglobin and hematocrit in the present study suggest that plasma volume is expanded after high sodium intake and Modelflow-derived estimates of CO were concurrently increased compared with the habitual sodium condition. These findings are similar to studies that have used either water drinking (40) or hypertonic saline infusions (41) to replace the fluid loss that occurs during exercise and have demonstrated attenuated PEH. In addition, neural mechanisms have

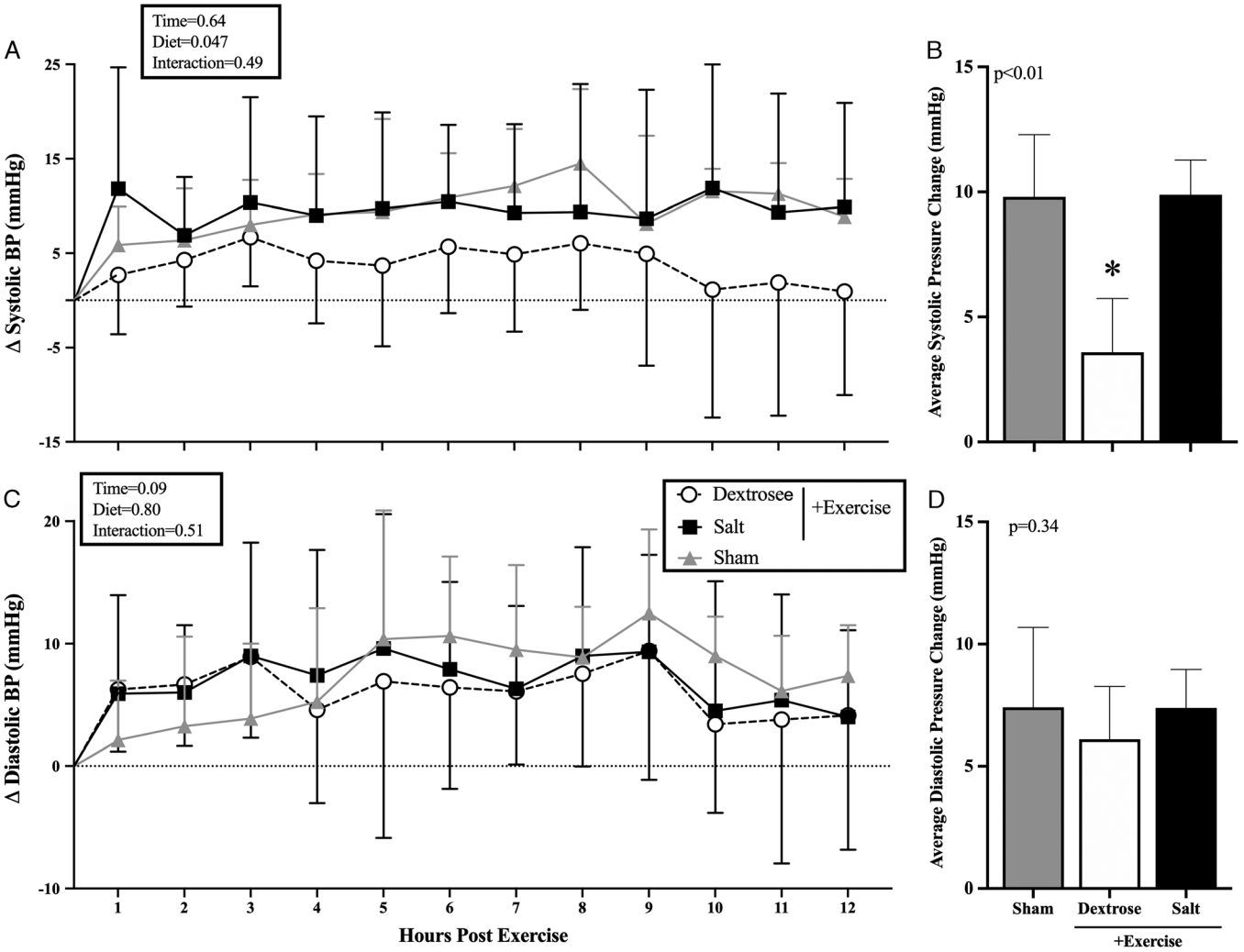

FIGURE 3—Hourly (A) and average (B) changes in systolic BP over 12 h after a bout of submaximal aerobic exercise or 1 h of quiet rest. After quiet rest (sham, gray triangles, and bars), ambulatory systolic BP increased over the following 12 h. One hour of submaximal aerobic exercise after habitual salt intake (dextrose, *open circles*, and *bars*) attenuated the rise in systolic BP; however, 10 d of high dietary salt intake (salt, *filled squares*, and *bars*) prevented the BP-lowering effects of exercise. Hourly (C) and average (D) changes in diastolic BP were not different between conditions. Data are presented as mean ± SD. *Significantly different from sham.

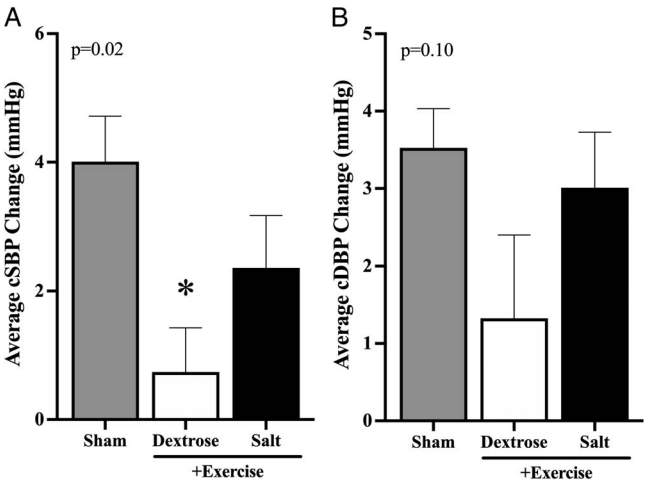

FIGURE 4—A, Average ambulatory central systolic BP is reduced over 12 h after a bout of submaximal aerobic exercise compared with sham exercise (*gray bars*) on a habitual salt diet (dextrose, *open bars*); however, 10 d of high dietary salt intake (salt, *black bars*) attenuates the BP-lowering effects of cSBP. B, High dietary salt intake did not significantly affect central diastolic BP. Data are presented as mean ± SD. *Significantly different from sham.

been implicated in contributing to PEH (42–44), albeit to a lesser extent than the reduction in TPR (24,25). Although there is evidence to suggest that dietary salt intake affects autonomic control of BP (22,45–47), the cBRS and HRV data presented in the present study do not support speculation that high dietary salt intake blunted PEH via neural mechanisms. However, future studies focused on the mechanisms by which dietary salt affects PEH should utilize more direct assessments of autonomic function (e.g., microneurography).

Importantly, the effects of high dietary salt intake on PEH were observed via ambulatory BP monitoring as well as in the laboratory. In the present study, ambulatory systolic BP increased throughout the day after the sham condition (i.e., when participants did not exercise). As previously described, submaximal exercise attenuated the increase in ambulatory BP (11,12,30). Ten days of high dietary sodium intake, however, reduced the beneficial effects of submaximal aerobic exercise on ambulatory BP. These data are concerning, as the PEH that occurs after a single bout of aerobic exercise is considered a critical CV benefit for patients with elevated BP (8,10,12,30). The increased prevalence of hypertension (from 31.9% to 45.6% (4)) following the 2017 American College of Cardiology/American Heart Association guidelines for the prevention, detection, evaluation, and management of high blood pressure in adults (3) highlights the need to further investigate lifestyle factors that may reduce CV risk. The present data provide support for the idea that excess dietary sodium intake may increase CV disease risk by attenuating the BP benefits that result from exercise.

Another important and novel finding of the present study was that submaximal aerobic exercise also lowers cSBP by ~3 mm Hg. High dietary sodium intake has been associated with increased arterial stiffness (20,21), which can contribute to higher cSBP. Although we did not measure arterial stiffness directly in this study, it is unlikely that increased arterial stiffness contributed to the attenuation in central PEH, as structural changes involving the extracellular matrix proteins collagen and elastin that determine arterial stiffness would take considerable time (months, if not years). It is therefore unlikely that the 10-d intervention used here would contribute significantly to increased arterial stiffness. Previous reports have indicated that changes in dietary sodium can produce rapid (i.e., 1–2 wk, similar to the time frame of the present intervention) changes in arterial stiffness (20,48). In both cases, however, these changes occurred concomitantly with changes in arterial BP, which is likely responsible for the difference in arterial stiffness (20). In the present study, however, mean BP was not different after 10 d of high sodium intake, so it remains unclear if differences in central PEH simply reflect the pressures observed in the periphery, or are the results of sodium-induced alterations in arterial load. Without a comprehensive assessment of arterial load and arterial stiffness in the present study, it is difficult to identify the underlying mechanism by which central PEH was attenuated after high sodium intake.

Finally, it is possible that high dietary sodium led to endothelial dysfunction, as has been previously reported (17–19), which may have reduced the vasodilatory capacity of resistance arteries, thereby attenuating the reduction in TPR that is typically observed after exercise. Although we did not examine this possible underlying mechanism directly, this explanation is unlikely for 2 reasons: 1) Modelflow-derived indices of TPR were not different after exercise in either condition, and 2) previous work from Halliwill and colleagues (49) indicates that PEH is not dependent on the production of nitric oxide. In this previous study, nitric oxide synthase was inhibited via intravenous infusion of N^G-mono-methyl-L-arginine after exercise. When compared with a control condition (sham exercise), PEH was still present despite inhibition of nitric oxide synthase, indicating that PEH occurs independent of nitric oxide production after exercise. However, the present study did not directly assess endothelial function; therefore, the contribution of sodium-induced endothelial dysfunction on PEH warrants further investigation.

Limitations. There are several limitations of the present study that must be addressed. The study population for this study was limited to recreationally active young adults with nonhypertensive BP. Individuals with high BP tend to have larger changes in their BP after exercise (8) and are more predisposed to have salt-sensitive BP (i.e., BP that is affected by sodium intake). Therefore, the observations from the present study may not be true for adults with hypertension.

Consistent with previous reports (35,36), the magnitude of changes in diastolic BP was smaller than that observed in systolic BP. Although we did not observe significant differences in ambulatory diastolic BP, the mean values followed the same pattern as systolic BP. Because the present sample size estimates were designed to observe differences in systolic BP, it is likely that we were underpowered to observe effects on diastolic BP. Effect size estimates from the current data indicate

that 26 individuals would be required to detect a difference in diastolic BP. Future studies should include larger sample sizes to examine the effects of dietary sodium intake on ambulatory diastolic BP after submaximal aerobic exercise.

In addition, this study relied on estimates of plasma volume expansion to provide insight into how sodium may attenuate PEH. More direct measures of plasma volume such as carbon monoxide rebreathing (50) may provide more reliable measures of the plasma volume expansion induced by high-sodium diets and may have strengthened the study. Furthermore, CO and TPR were assessed using Modelflow and therefore should be interpreted cautiously, especially considering the inaccuracy of these measures when core temperature is elevated (i.e., recovery from exercise) (51).

Finally, we did not measure arterial stiffness in the present study. Although we observed an attenuation of central PEH after high sodium intake, it is possible that this finding reflects the systemic attenuation in PEH that was observed in brachial BP measures. However, the differences observed in pulse pressure amplification suggest that high dietary sodium differentially affects the central compared with peripheral BP.

CONCLUSIONS

In conclusion, the current data indicate that excess dietary sodium intake interferes with the postexercise reductions in BP known as PEH. These data also indicate that the BP-lowering effects of acute aerobic exercise extend to central BP as well. The widespread overconsumption of dietary sodium in America, coupled with the increasing epidemic of hypertension, demonstrates the importance of these findings. Comprehensive lifestyle modifications including aerobic exercise participation should also include dietary sodium restriction to maximize the BP benefits that result from exercise and overall CV health.

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