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Effects of regular high-cocoa chocolate intake on arterial stiffness and metabolic characteristics during exercise

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ABSTRACT

Objective: The aim of this study was to examine the effects of regular high-cocoa chocolate consumption on arterial stiffness and fat oxidation during light- to moderate-intensity exercise.
Methods: This randomized, controlled, parallel-group intervention study included 32 Japanese college students (mean age, 20.7 ± 0.3 y; men, n = 24; women, n = 8) who were assigned to either control or intervention groups (n = 16 each). The control group did not alter their habitual diet or physical activity throughout the study period. The intervention group consumed 20 g/d (508 mg of cacao polyphenol) of high-cocoa chocolate for 4 wk. Blood pressure, heart-ankle pulse wave velocity, cardio-ankle vascular index, body composition, and metabolic characteristics during exercise at 50% maximal oxygen uptake level were assessed before and after the intervention.
Results: Four weeks of high-cocoa chocolate ingestion significantly reduced heart-ankle pulse wave velocity and cardio-ankle vascular index (%change, intervention versus control: −2.3 ± 0.9% versus 0.9 ± 0.9%, and −4.8 ± 1.8% versus 0.7 ± 1.3%, respectively; both $P < 0.05$). However, blood pressure, weight, body mass index, body fat, waist circumference, and metabolic characteristics during exercise such as respiratory exchange ratio did not significantly change in either group.
Conclusions: Four weeks of regular high-cocoa chocolate consumption reduced arterial stiffness after considering blood pressure in healthy young men and women. However, the habitual consumption of high-cocoa chocolate for 4 wk did not affect metabolic characteristics during light- to moderate-intensity exercise and body composition.

Introduction

Fruits, wines, and chocolate are polyphenol rich, and their beneficial effects are attracting increasing attention [1–4]. Recent meta-analyses have shown that high-cocoa chocolate or cocoa products favorably affect cardiometabolic health [1–3]. Most individuals can easily intake chocolate, and thus high-cocoa chocolate could be an effective cardiovascular preventive strategy for large populations [5]. Therefore, further scientific evidence of the benefits of high-cocoa chocolate is needed.

Pulse wave velocity (PWV) generally serves as an index of arterial stiffness and increased arterial stiffness has been identified as an independent risk factor for future cardiovascular disease and mortality [6]. A prospective analysis has indicated that weekly chocolate intake benefits arterial stiffness [7]. Indeed, several previous studies have shown that arterial stiffness is reduced by cocoa products and that flow-mediated dilation is increased by the habitual consumption of high-cocoa chocolate intake [2,4,8]. Aside from these studies, however, little is known about whether regular high-cocoa chocolate consumption directly affects arterial stiffness. Pereira et al. found that high-cocoa chocolate simultaneously reduced arterial stiffness, central blood pressure (BP), and brachial BP in young healthy individuals [9]. Many other studies have demonstrated that cocoa consumption reduces BP [2,9,10], but BP is a powerful confounding factor that affects PWV [11,12]. Interventions with high-cocoa chocolate also have indicated that the magnitude of reduction in BP is associated with baseline BP value, and several studies, particularly those that included normotensive persons, found that BP did not improve regardless of intake dose [2,10]. Accordingly, these findings raise the question of whether regular high-cocoa chocolate ingestion directly reduces arterial

MN and YN conceived of, designed, and performed the study and analyzed the data. MN, YN, and NM wrote the paper. MN and NM interpreted the data. All authors approved the final version of the article.

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stiffness or BP and consequently reduces arterial stiffness, especially in healthy young individuals. To the best of our knowledge, the effects of high-cocoa chocolate on arterial stiffness and issues regarding BP have not been comprehensively addressed.

The habitual intake of high-cocoa chocolate/cocoa products confers beneficial effects on metabolism-related biomarkers [1,3]. Acute high-cocoa chocolate ingestion can increase the mobilization of free fatty acids or triacylglycerols during light- to moderate-intensity exercise [13,14], suggesting that high-cocoa chocolate consumption has beneficial effects on exercise as part of weight loss programs. In general, plasma free fatty acid flux rapidly peaks at light to moderate intensity of 45% to 50% maximal oxygen uptake (VO_2 max), and lipids are thought to provide slightly more than half the energy consumed at rest and during light- to moderate-intensity exercise [15]. If regular high-cocoa chocolate consumption actually increases fat oxidation, changes in metabolic parameters could be determined by measuring respiratory gas exchange during exercise.

Based on this background, we tested the hypothesis that regular high-cocoa chocolate consumption induces a reduction in arterial stiffness or an increase in fat oxidation during light- to moderate-intensity exercise in healthy young individuals.

Methods

Participants

Thirty-two healthy Japanese college students (mean age, 20.7 ± 0.3 y; men, n = 24; women, n = 8) at our institution participated in this randomized, controlled, parallel-group intervention study. None of the participants were taking medication nor had any of them participated in a regular exercise program for ≥1 y. The participants were matched for age, sex, BP, and arterial stiffness as closely as possible and randomly assigned to either a control (n = 16) or an intervention (n = 16) group (Fig. 1). The purpose, procedures, and risks of the study were explained to all of the students and they provided written informed consent before participating. The Human Ethics Committee at the Osaka Institute of Technology approved the study (approval number: 2016-47), which proceeded according to the guidelines of the Declaration of Helsinki. The study was also registered in the UMIN Clinical Trials Registry UMIN000026486.

Sample size and experimental procedures

We determined the sample size for each group by power calculations using G*Power 3 and assumed that arterial stiffness would be reduced by ~5% according to previous findings [2,9,16]. To detect this difference at 80% power with a two-tailed α of 5%, an intervention group comprising 10 to 16 participants was needed. Thus, we assigned 16 participants each to the control and intervention groups.

Both groups were assessed before and after the study. All tests proceeded in a quiet airconditioned room (22°C–24°C) at the same time of day and at the same number of hours after the last meal to avoid potential diurnal variations. The participants were required to abstain from caffeine and to fast for ≥4 h before each test.

Body composition

Weight, body fat, lean body mass (LBM), and body mass index were determined by bioelectrical impedance using a TBF-410 instrument (Tanita Co., Tokyo, Japan) as described in a previous study [12]. Waist circumference was measured around the abdomen at the level of the navel at the late expiratory phase using a tape measure. Day-to-day coefficients of variation (CV) for each parameter were all <10% under our experimental conditions [12,16,17].

Arterial stiffness, BP, and heart rate

We measured PWV, BP, and heart rate (HR) with the participants in the supine position using a semiautomated device (VS-1500 AE/AN; Fukuda Denshi, Tokyo, Japan) [12,16,17]. Heart-ankle PWV (haPWV) and the cardio-ankle vascular index (CAVI) were calculated as indicators of arterial stiffness [12,16,17]. The carotid-femoral PWV (cfPWV), which is an index of central arterial stiffness, was measured using the same device in the control (n = 15) and intervention (n = 16) groups [18]. The intraobserver CVs for haPWV, CAVI, and cfPWV determined in the laboratory on two separate days were 2.6 ± 0.6%, 3.6 ± 0.6%, and 7.5 ± 1.2%, respectively [12,16–18].

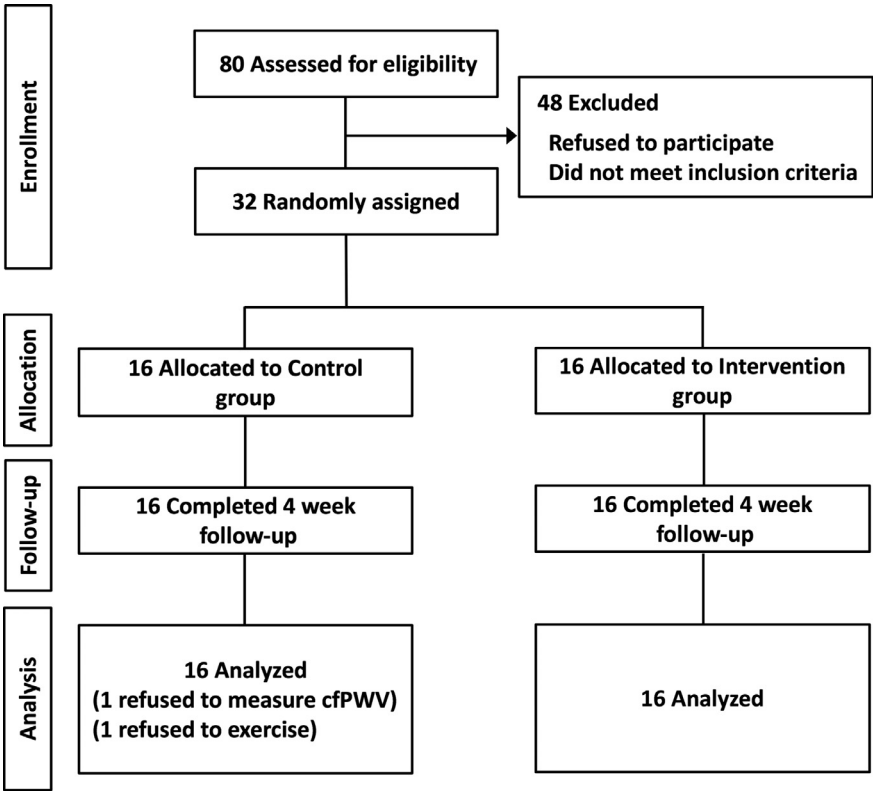

Fig. 1. Flow diagram of study participants.

Metabolic parameters at rest and during submaximal exercise

Rest and exercise proceeded on an electromagnetically braked cycling ergometer (75 XL III; Konami, Tokyo, Japan) in the upright position. After a 5-min rest, 6 min of exercise proceeded at a constant submaximal intensity according to previous protocols of 45, 70, and 100 W for men and 20, 35, and 50 W for women. Pedaling rates remained constant at 60 rpm [19,20].

Minute expired ventilation (VE), oxygen uptake (VO_2), and carbon dioxide output (VCO_2) V were collected and determined every 15s using an automatic gas analyzer with a mixing chamber (AR10; Arco System, Chiba, Japan). Heart rate was monitored using an HR monitor (V800; Polar Japan, Tokyo, Japan). The data were averaged during the last 2 min of the 5-min rest and the 6-min bout of exercise at each intensity. Respiratory exchange ratios (RER), rates of substrate oxidation of carbohydrate (CHO) and fat, and the relative contribution of CHO and fat oxidation to the total energy production were determined [21]. Circulating levels of glucose and lactate were assessed at rest and before the end of 6 min of exercise. Glucose and lactate were measured using glucose (Medisafe Mini GR-102; Terumo Co., Tokyo, Japan) and lactate (Lactate Pro2; Arkray Inc., Kyoto, Japan) analyzers. Ratings of perceived exertion were determined using the Borg 15-point rating scale [22].

We established relationships between submaximal intensity and VO_2 as well as HR. Maximal work rates at estimated maximal HR and VO_2 at maximal work rates (estimated VO_2 max) were individually calculated by extrapolating these linear relationships. Maximal HR was estimated as 220 minus age [23]. Exercise intensity at 50% of the estimated VO_2 max was individually assumed by interpolating the linear relationship between work rate and VO_2. Exercise intensity at 50% VO_2 max was individually determined from the work rates nearest to 50% of estimated VO_2 max at the three levels of intensity, and then parameters during exercise were subsequently analyzed. Data from one participant in the control group who declined to exercise were excluded. The day-to-day CV for VO_2 during exercise was 5.3 ± 1.5% [17,20].

Daily dietary intake and steps

Dietary intake before and after the study period was assessed using a brief-type self-administered diet history questionnaire [24]. Daily steps were obtained from the health care app on Apple iPhone [25]. According to our studies [26], a valid day was defined as wearing the device for >2000 steps, and days when the equipment was not worn were excluded from analysis. We subsequently analyzed data about steps from 15 of the participants (control: n = 7, intervention: n = 8) who had four or more valid days per week, and data were compared between the first (pre) and last (post) 2 wk of the intervention period.

High-cocoa chocolate intervention

Participants in the intervention group consumed 20 g/d (10 g at ~1000 and 10 g at 1500 daily; cacao polyphenol, 508 mg) of high-cocoa chocolate (Chocolate Kouka Cacao 72%, Meiji Co. Ltd., Tokyo, Japan) for 4 wk. Nutrition facts indicate that 5 g of the chocolate included comprised 28 kcal of energy, 0.5 g of protein, 2.1 g of fat, 1.7 g of CHO, 0.6 g of dietary fiber, and 0.3 mg of sodium. Observers provided the same type of chocolate to all participants in the intervention group weekly. Daily compliance with chocolate intake was confirmed using the Line social network service app (LINE Co., Tokyo, Japan) via mobile phone. Participants in the control group had no concomitant intervention. All participants were advised to maintain their usual diet and to refrain from any other specific exercise throughout the study period.

Statistical analysis

The results are presented as means ± standard error of the mean. Parameters before the study were compared between the two groups using independent Student's *t* tests. Changes in parameters were evaluated using a two-way repeated-measures analysis of variance (ANOVA). When the *F*-value was significant, the Bonferroni method was applied for post hoc multiple comparisons. Changes in arterial stiffness between pre- and postintervention were assessed using independent Student's *t* tests and analyses of covariance (ANCOVA) that included age, sex, height, weight, baseline values, and mean BP as covariates. Relationships were assessed using Pearson correlations. All data were statistically analyzed using SPSS 14.0 J (IBM SPSS Japan, Tokyo, Japan) and Excel Statistics 2015 (Social Survey Research Information, Tokyo, Japan). Effect size (ES; using Cohen's d) and statistical power (1-β) were calculated using G*Power 3. Values were regarded as statistically significant when $P < 0.05$.

Results

Baseline data

Table 1 shows the physical characteristics of all the participants. None of the parameters significantly differed between the control and intervention groups before the study. Estimated HR max in the control and intervention groups was 196 ± 22 and 191 ± 15 beats/min, respectively. Estimated VO_2 max did not significantly differ in either absolute

Table 1
Physical characteristics of the participants pre- and postintervention (data are expressed as means ± SE)

Variables	Control group (n = 16)						Intervention group (n = 16)						*t* test or ANOVA		
	Pre			Post			Pre			Post			Group *P*-value	Time *P*-value	Interaction *P*-value
Participants (men/women)	(12/4)						(12/4)								
Age, y	20.7	±	0.3		–		20.8	±	0.3		–		0.870	–	–
Height, cm	166.7	±	2.3		–		164.3	±	1.8		–		0.396	–	–
Body mass, kg	62.2	±	2.7	62.4	±	2.6	57.8	±	2.6	57.7	±	2.7	0.233	0.851	0.453
Body mass index, kg/m^2	22.3	±	0.7	22.4	±	0.7	21.3	±	0.7	21.2	±	0.7	0.296	0.831	0.523
Body fat, %	25.1	±	2.0	24.8	±	2.2	21.0	±	1.7	20.1	±	1.6	0.107	0.079	0.336
Lean body mass, kg	46.4	±	2.1	46.8	±	2.1	45.5	±	1.9	46.0	±	2.0	0.763	0.156	0.917
Waist circumference, cm	77.8	±	1.8	78.0	±	1.9	74.2	±	1.8	74.1	±	2.0	0.174	0.882	0.632
HR, beats/min	64	±	7	61	±	5	64	±	9	62	±	8	0.847	0.061	0.608
Systolic BP, mmHg	120	±	3	122	±	4	120	±	2	120	±	2	0.865	0.264	0.487
Diastolic BP, mmHg	70	±	1	71	±	1	70	±	1	70	±	1	0.913	0.727	0.647
Mean BP, mmHg	88	±	2	90	±	2	88	±	1	88	±	1	0.708	0.307	0.473
Pulse pressure, mmHg	50	±	2	51	±	3	50	±	2	50	±	2	0.870	0.343	0.653
Resting VE, L/min	7.8	±	0.4	7.8	±	0.4	7.8	±	0.3	7.4	±	0.4	0.671	0.395	0.489
Resting VO_2, L/min	0.27	±	0.01	0.26	±	0.01	0.25	±	0.01	0.25	±	0.01	0.303	0.985	0.663
Resting VCO_2, L/min	0.22	±	0.01	0.21	±	0.01	0.20	±	0.01	0.20	±	0.01	0.281	0.521	0.606
Resting RER	0.83	±	0.01	0.81	±	0.01	0.82	±	0.01	0.81	±	0.02	0.655	0.227	0.983
Glucose, mg/dL	74	±	3	79	±	2	75	±	3	82	±	3*	0.646	0.008	0.364
Lactate, mmol/L	1.1	±	0.1	1.0	±	0.1	1.1	±	0.1	1.0	±	0.1	0.889	0.008	0.686
Total caloric intake, kcal/d	1604	±	137	1603	±	109	1719	±	108	1666	±	141	0.588	0.699	0.712
Carbohydrate intake, g/d	216.6	±	20.8	217.9	±	17.2	246.5	±	20.2	235.7	±	23.1	0.376	0.681	0.600
Fat intake, g/d	51.7	±	5.2	50.6	±	5.1	48.7	±	3.4	47.0	±	4.7	0.590	0.605	0.914
Protein intake, g/d	56.6	±	5.0	54.9	±	4.0	57.2	±	4.6	57.1	±	5.9	0.835	0.690	0.718

BP, blood pressure; HR, heart rate; RER, respiratory exchange ratio; SE, standard error; VCO_2, carbon dioxide output; VE, minute expired ventilation; VO_2, oxygen uptake.
*$P < 0.05$ vs each preintrvention.

(control versus intervention: 2.35 ± 0.22 versus 2.43 ± 0.18 L/min) or relative (control versus intervention: 37.8 ± 2.8 versus 41.6 ± 2.0 mL·kg·min^{-1}) values per body mass. The work rate of exercise at estimated 50% VO_2 max did not significantly differ between the groups (control versus intervention: 64 ± 6 versus 72 ± 6 W).

Effects of high-cocoa chocolate on body composition and hemodynamics

Body composition, such as body mass, body mass index, body fat, LBM, and waist circumference, did not significantly change in either group after 4 wk (Table 1). Similarly, neither BP nor HR in the supine position significantly differed after the intervention in either group (Table 1). However, two-way repeated-measures ANOVA indicated significant interactions of haPWV and CAVI (both $P < 0.05$). Mean haPWV (ES, 0.67; 1-β, 0.70) and CAVI (ES, 0.6; 1-β, 0.61) were significantly reduced (both $P < 0.05$) in the intervention group (Fig. 2 A, C) compared with the control group and even more so after adjusting haPWV or CAVI for age, sex, height, weight, baseline values, and mean BP when analyzed by ANCOVA (Fig. 2 B, D). In addition, cfPWV was significantly reduced in the intervention (pre versus post: 6.03 ± 0.13 versus 5.78 ± 0.13 m/s, $P < 0.01$; ES, 0.81;1-β, 0.85), but not in the control group (pre versus post: 5.97 ± 0.12 versus 5.89 ± 0.13 m/s). The changes in arterial stiffness did not significantly correlate with the amount of chocolate consumed per body mass (haPWV: $R = -0.144$, $P = 0.297$; CAVI: $R = -0.025$, $P = 0.928$; cfPWV: $R = -0.167$, $P = 0.537$).

Effects of high-cocoa chocolate on metabolic parameters at rest and during exercise

None of VE, VO_2, VCO_2, RER, HR, or ratings of perceived exertion at rest and during exercise at 50%VO_2 max significantly changed in either group (Tables 1 and 2). Rates of substrate oxidation of CHO

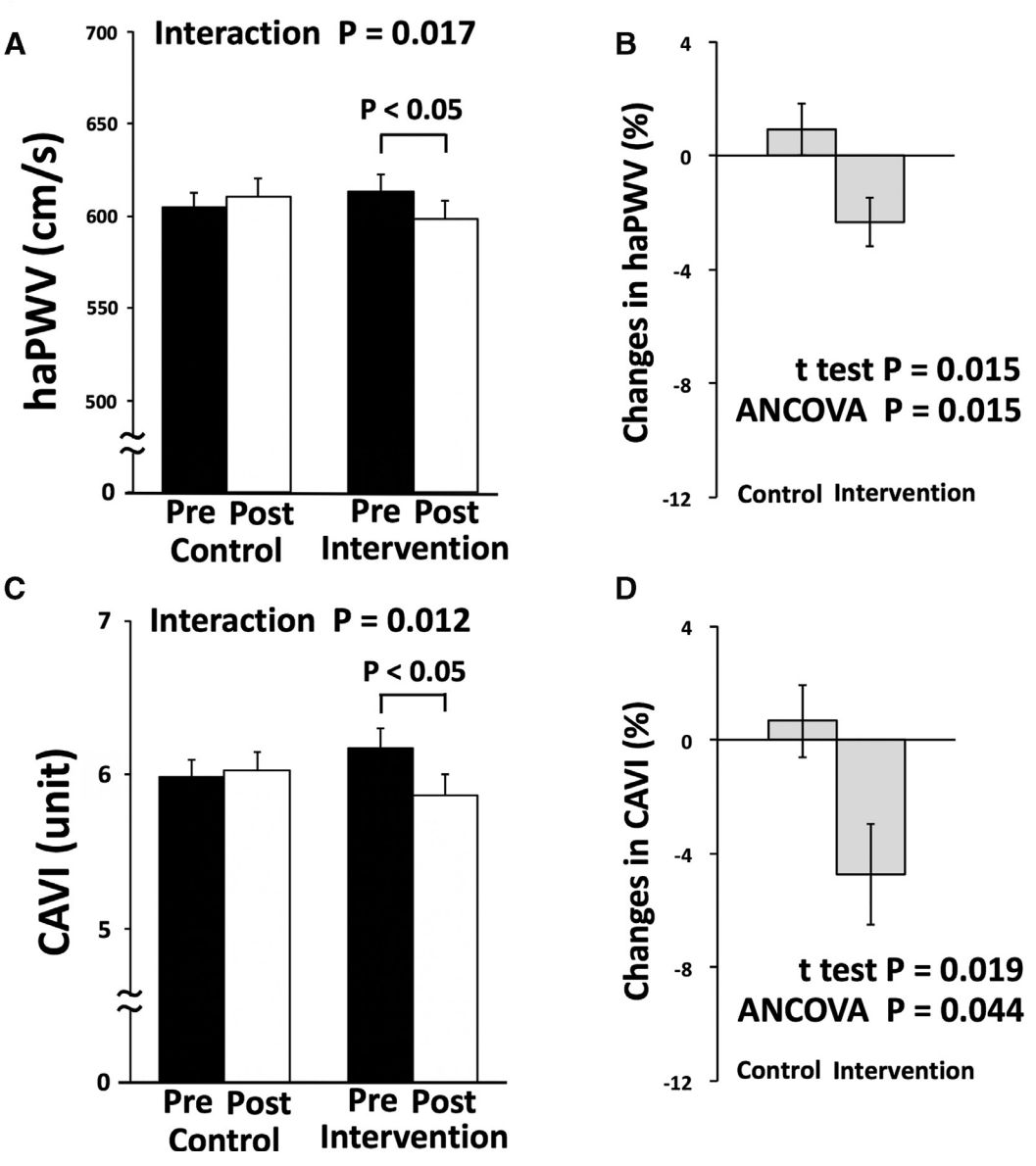

Fig. 2. Changes in haPWV (A, B) and CAVI (C, D) before and after the intervention. ANCOVA, analysis of covariance; CAVI, cardio-ankle vascular index; haPWV, heart-ankle pulse wave velocity; pre, before intervention; post, after intervention. Data are expressed as means ± standard error.

Table 2
Changes in metabolic characteristics during exercise at 50%VO_2 max level pre- and postintervention (data are expressed as means ± SE)

Variables	Control group						Intervention group						ANOVA		
	Pre			Post			Pre			Post			Group *P*-value	Time *P*-value	Interaction *P*-value
VE, L/min	24.4	±	1.6	24.6	±	1.6	27.5	±	1.8	26.6	±	1.7	0.289	0.499	0.332
VO_2, L/min	1.10	±	0.08	1.09	±	0.08	1.18	±	0.08	1.14	±	0.08	0.584	0.112	0.180
VCO_2, L/min	0.99	±	0.07	0.97	±	0.07	1.07	±	0.08	1.02	±	0.07	0.507	0.053	0.416
RER	0.90	±	0.01	0.88	±	0.01	0.91	±	0.01	0.90	±	0.01	0.177	0.090	0.379
%VO_2 max, %	48.1	±	1.3	48.0	±	1.5	49.0	±	1.1	47.4	±	1.3	0.927	0.164	0.224
CHO oxidation, g/min	0.99	±	0.09	0.91	±	0.10	1.12	±	0.11	1.04	±	0.10	0.356	0.138	0.965
Fat oxidation, g/min	0.18	±	0.02	0.21	±	0.03	0.17	±	0.02	0.18	±	0.02	0.485	0.333	0.668
%CHO contribution, %	65.5	±	3.6	57.1	±	4.2	68.3	±	3.6	65.5	±	3.2	0.177	0.090	0.379
%Fat contribution, %	34.5	±	3.6	42.9	±	4.2	31.7	±	3.6	34.5	±	3.2	0.177	0.090	0.379
HR, beats/min	117	±	2	113	±	3	117	±	2	116	±	3	0.540	0.112	0.395
RPE	12.5	±	0.4	12.0	±	0.5	12.2	±	0.4	12.1	±	0.5	0.856	0.218	0.473
Glucose, mg/dL	69	±	3	80	±	3*	74	±	3	81	±	2*	0.332	<0.001	0.345
Lactate, mmol/L	1.5	±	0.1	1.6	±	0.1	1.6	±	0.1	1.5	±	0.1	0.913	0.709	0.395

%CHO and %fat contribution, relative contribution of carbohydrate and fat oxidation to the total energy production; %VO_2 max, VO_2 corresponds to estimated VO_2 max at each preintervention; CHO, carbohydrate; CHO and fat oxidation, rates of substrate oxidation of carbohydrate and fat; HR, heart rate; SE, standard error; RER, respiratory exchange ratio;VCO_2, carbon dioxide output; VE, minute expired ventilation; VO_2, oxygen uptake; RPE, ratings of perceived exertion.
*$P < 0.05$ vs each preintervention.

and fat and the relative contributions of CHO and fat oxidation to total energy production during exercise also did not significantly change in both groups (Table 2). Circulating glucose levels at rest tended to increase in the control groups and significantly increased in the intervention group. Moreover, glucose during exercise significantly increased in both groups after 4 wk (Table 2). Lactate during exercise also did not change significantly in either group.

Dietary and exercise habits

Dietary habits did not significantly change in either group with the exception of high-cocoa chocolate ingestion. Both groups also stated that their amounts of physical activity had not changed between before and during the intervention period. Daily step counts did not significantly change in both groups (control pre versus post: 7474 ± 672 versus 7556 ± 797 steps/d; intervention: pre versus post: 7833 ± 463 versus 8403 ± 638 steps/d).

Discussion

The salient findings of the present study were as follows. Four wk of high-cocoa chocolate consumption reduced haPWV, CAVI, and cfPWV in healthy young individuals, but did not significantly change BP, body composition, RER, and glucose at rest or during exercise.

A previous study found that regular high-cocoa chocolate ingestion simultaneously reduced cfPWV and BP among healthy young individuals [9]. Although PWV is widely identified as the gold standard among methods of assessing arterial stiffness [6,27], BP is a major confounding factor that has powerful effects on PWV [11,12]. Thus, we investigated whether regular high-cocoa chocolate consumption directly reduces arterial stiffness in healthy young individuals. Our results indicated that the CAVI and PWV became significantly reduced after chocolate ingestion. In general, CAVI is an index of arterial stiffness from the aorta to the ankle, after adjustment for BP [28]. In addition, BP and HR did not significantly change after intervention. Throughout the study period, neither dietary habits nor physical activity significantly differed. Therefore, these findings indicated that regularly consuming high-cocoa chocolate reduces arterial stiffness after adjusting BP in healthy young individuals.

The physiological mechanisms through which the regular consumption of chocolate with a high cocoa content reduces arterial stiffness remain obscure. However, high-cocoa chocolate can enhance endothelium-dependent vasodilation, primarily by increasing nitric oxide (NO) production or through bioavailability [2,8], which could contribute to a reduction in arterial stiffness [27]. Indeed, one study has demonstrated that polyphenols can increase NO synthase activity and NO release in endothelial cells in vitro [29]. Thus, cocoa polyphenol might increase the production or bioavailability of NO, and thus induce arterial functional changes such as reducing arterial stiffness. However, we did not obtain direct evidence in vivo to support this notion and further studies are required.

Acute ingestion of high-cocoa chocolate can increase fat oxidation during exercise [13,14]. Although an acute study is important, it was unclear whether regular ingestion of high-cocoa chocolate affects metabolic characteristics at rest or during exercise. After 4 wk of ingestion, there were no significant changes to VO_2, HR, lactate, RER, indexes of fat oxidation, body weight, fat, or LBM. In addition to the present reduction in arterial stiffness, the daily amount of ingested polyphenol in this study was the same or slightly higher than that in previous studies [1–3,13,14]. Thus, the results might not have been due to an insufficient dose of polyphenol or chocolate. Plasma levels of polyphenol or epicatechin, which are components of high-cocoa chocolate, generally reach a maximum at 2 h after intake, and then gradually disappear [30]. Thus, one explanation for the conflicting findings between this and previous acute studies might be different circulating amounts of polyphenol or epicatechin at the start of exercise. Therefore, the present findings indicate that regular ingestion of high-cocoa chocolate does not affect metabolic characteristics either at rest or during light- to moderate-intensity exercise.

Contrary to previous findings [2,10], we found that resting BP and glucose did not significantly improve in young participants. The magnitude of BP reduction relates to baseline BP levels [10], and young individuals with normal BP are unlikely to have a redundant reduction in BP. On the other hand, we also found increased resting glucose levels, especially among those in the intervention group who consumed regular normal diets plus 20 g/d of high-cocoa chocolate. Thus, although body mass and fat did not significantly increase, adding high-cocoa chocolate to the diet might have induced a small increase in blood glucose levels, meaning that high-cocoa does not always positively affect metabolic-related health.

The study limitations are as follows. The magnitude of the reduction in arterial stiffness was relatively small. However, an increase in PWV of 1 m/s generally corresponded to a ~10% increase in risk for cardiovascular events or mortality [6]. Therefore, the effects are clinically noteworthy. The intervention group was given absolute amounts of chocolate, and the control group did not receive a placebo. Alternatively, the changes in arterial stiffness did not significantly correlate with relative amounts per body mass. Moreover, previous studies have not found any significant improvements in BP or arterial stiffness after placebo control chocolate ingestion [4,10]. Accordingly, the reduction in arterial stiffness was probably not profoundly affected by minor mass-based differences relative to ingested amounts or placebo effects. Finally, we did not measure circulating lipid profiles. However, all participants were healthy young individuals with no chronic diseases, and thus were unlikely to have risk factors for atherosclerosis or a need to improve circulating lipid profiles.

Conclusions

Results from the present study demonstrated that 4 wk of regular high-cocoa chocolate ingestion reduced arterial stiffness after adjusting for BP in a group of healthy young individuals. However, habitual high-cocoa chocolate intake probably does not affect body composition or metabolic characteristics at rest or during light- to moderate-intensity exercise.

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