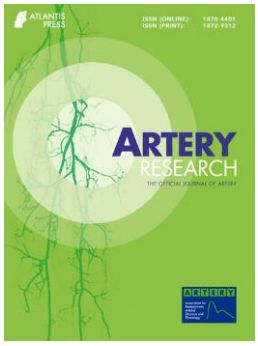

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Short communication

Sex differences in aortic stiffness following acute resistance exercise

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KEYWORDS
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There are established sex differences in vascular structure and function and neurovascular blood pressure (BP) regulation at rest and in response to various stressors.[1,2] Acute resistance exercise (RE) imposes a unique hemodynamic stress on the heart and central vasculature, resulting in functional increases in aortic stiffness. Increases in aortic stiffness in response to acute RE are seen in men[3] and women.[4] Whether there are sex differences in aortic stiffness and central BP in response to acute RE remains poorly examined.[5] The purpose of this study was to examine sex differences in aortic stiffness and central BP in response to acute RE. We hypothesized that women would have an attenuated increase in aortic stiffness and central BP in response to acute RE.

Methods

This study is a secondary analysis of two previously published studies that both used the same methods and study design.[4,6] Briefly, 27 young healthy adults between the ages of 18–35 participated in these studies (n = 13 female). Men and women selected from each study were matched for age. All participants provided written, informed consent. These studies were approved by the University Institutional Review Board and complied with ethical standards put forth in the Declaration of Helsinki.

Height and weight were assessed via wall stadiometer and electronic scale, respectively, and body composition was estimated via air displacement plethysmography (BodPod; COSMED, Concord, CA). Body mass index (BMI) was calculated as weight (kg)/height (m^2). According to health history questionnaires, 5 of the women reported engaging in habitual aerobic exercise and/or endurance sports, 2 reported engaging in habitual resistance exercise and the remainder were sedentary. 6 of the men reported engaging in habitual aerobic exercise and/or endurance sports, 5 identified as engaging in both habitual aerobic and resistance exercise and 2 engaged exclusively in habitual resistance exercise.

Participants were instructed to fast for a minimum of 3 h and avoid exercise and consuming alcohol/caffeine for a minimum 12 h prior to testing. Following a 10-min quiet rest period, participants had hemodynamics assessed using a validated brachial oscillometric cuff (Mobil-O-Graph, I.E.M., Stolberg, Germany).[7] Pulse wave analysis was performed via the cuff clamping down on diastolic pressure for

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Table 1 Descriptive characteristics.

Variable	Males ($n = 14$)	Females ($n = 13$)	p-Value
Age (years)	23 ± 4	24 ± 4	0.48
Body fat (%)	16.4 ± 9.7	21.3 ± 4.9	0.12
Height (cm)	178 ± 6	167 ± 6	0.001
Weight (kg)	77.2 ± 14.0	61.6 ± 9.8	0.001
Body mass index (kg/m^2)	24.2 ± 4.4	22.0 ± 3.1	0.14
5-RM bench press (kg)	76 ± 18	27 ± 7	0.001
Relative 5-RM	0.96 ± 0.23	0.45 ± 0.11	0.001
10-RM biceps curl (kg)	29 ± 7	14 ± 3	0.001
Relative 10-RM	0.37 ± 0.10	0.23 ± 0.49	0.001

RM, repetition maximum.

30 s, enabling acquisition of brachial pressure waveforms. From the brachial pressure waveform, an aortic pressure waveform was derived from a generalized transfer function using the ARCSolver method and used to estimate aortic pulse wave velocity (aPWV), central systolic and diastolic blood pressure, and the augmentation index (AIx).[8] aPWV derived from this method shows acceptable agreement with other methods of assessing PWV such as MRI.[9,10] Participants then completed an acute RE protocol consisting of five sets of five repetitions for bench press and five sets of ten repetitions for biceps curl with exercise being performed at 100% of the participants previously obtained 5-RM and 10-RM. Vascular-hemodynamic measures were measured again 10-min and 20-min after the acute RE bout. Women completed the acute RE bout during the early follicular phase of their menstrual cycle.

A repeated measures analysis of variance (2 groups by 3 time points) was used to examine group effects, time effects and group-by-time interactions with a Bonferroni correction factor applied to adjust for multiple comparisons. Associations of interest were examined using Pearson's correlation coefficients. Significance was determined *a priori* as $p < 0.05$. All data are reported as mean ± standard deviation. All statistical analyses were executed using Statistical Package for the Social Sciences (SPSS, version 21, IBM, Chicago, IL, USA).

Table 2 Central and brachial blood pressure before and after acute resistance exercise.

Variable	Males	Females	Effects		
	($n = 14$)	($n = 13$)	Time	Group	Interaction
Brachial SBP (mmHg)			0.001	0.001	0.001
Baseline	120 ± 10[b]	111 ± 9	*0.20*	*0.17*	*0.08*
P10	145 ± 19[a,b]	115 ± 11			
P20	132 ± 11[a,b]	114 ± 11			
Brachial DBP (mmHg)			0.02	0.12	0.001
Baseline	68 ± 6	68 ± 7	*0.47*	*0.57*	*0.33*
P10	74 ± 7[a,b]	63 ± 8[a]			
P20	66 ± 5	64 ± 8			
Brachial PP (mmHg)			0.001	0.001	0.04
Baseline	52 ± 6[a,b]	45 ± 9	*0.07*	*0.04*	*0.12*
P10	72 ± 17[a,b]	52 ± 10			
P20	67 ± 9[a,b]	50 ± 11			
Mean arterial pressure (mmHg)			0.001	0.001	0.001
Baseline	92 ± 7	88 ± 7	*0.86*	*0.50*	*0.07*
P10	106 ± 11[a,b]	87 ± 8			
P20	96 ± 7[b]	87 ± 9			
Central SBP (mmHg)			0.001	0.001	0.001
Baseline	109 ± 11	107 ± 11	*0.21*	*0.30*	*0.10*
P10	140 ± 20[a,b]	110 ± 11			
P20	127 ± 12[a,b]	108 ± 11			
Central DBP (mmHg)			0.001	0.03	0.002
Baseline	69 ± 6	69 ± 6	*0.36*	*0.74*	*0.37*
P10	75 ± 8[a,b]	64 ± 8[a]			
P20	68 ± 6	64 ± 11			
Central PP (mmHg)			0.001	0.03	0.002
Baseline	40 ± 9	35 ± 7	*0.09*	*0.16*	*0.17*
P10	65 ± 19[a,b]	46 ± 11[a]			
P20	59 ± 11[a,b]	43 ± 12			

SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; P10, 10 min following resistance exercise; P20, 20 min following resistance exercise.
Italicized p-value, adjusted for 5-RM strength.
[a] Significantly different from baseline.
[b] Significantly different than females.

Table 3 Aortic stiffness and augmentation index before and after acute resistance exercise.

Variable	Males	Females	Effects		
	(n = 14)	(n = 13)	Time	Group	Interaction
Aortic PWV (m/s)			0.001	0.002	0.001
Baseline	5.1 ± 0.5	4.9 ± 0.4			
P10	6.1 ± 0.8[a,b]	5.2 ± 0.7[a]			
P20	5.7 ± 0.4[a,b]	5.1 ± 0.3			
Aortic PWV/MAP (m/s/mmHg × 10^{-2})			0.001	0.77	0.27
Baseline	5.6 ± 0.4	5.6 ± 0.3	*0.25*	*0.84*	*0.79*
P10	5.8 ± 0.3[a]	6.0 ± 0.4[a]			
P20	6.0 ± 0.4[a]	6.0 ± 0.4[a]			
Heart rate (bpm)			0.001	0.38	0.001
Baseline	53 ± 6	60 ± 13	*0.32*	*0.91*	*0.07*
P10	74 ± 13[a]	65 ± 13			
P20	72 ± 10[a,b]	62 ± 11			
AIx@ 75 (%)			0.21	0.64	0.38
Baseline	17 ± 15	14 ± 10	*0.01*	*0.41*	*0.02*
P10	27 ± 19[a]	18 ± 15[a]			
P20	16 ± 17	16 ± 13			

PWV, pulse wave velocity; AIx, augmentation index; AIx@ 75, augmentation index at 75 b/min; MAP, mean arterial pressure; P10, 10 min following resistance exercise; P20, 20 min following resistance exercise.
Italicized p-value, adjusted for 5-RM strength.
[a] Significantly different from baseline.
[b] Significantly different than females.

Results

Groups were matched for age. Groups did not differ in body fat % or BMI. Males had higher absolute and relative strength (Table 1). Significant group-by-time interactions were detected for heart rate, brachial and central pressures, and aPWV (Tables 2 and 3, $p < 0.05$). Compared to men, women had an attenuated hemodynamic response (lessened increases in HR and BP) to acute RE. Change in aPWV from baseline to the 10-min time point was associated with change in mean pressure ($r = 0.41$, $p < 0.05$) and 5-RM bench press as a measure of muscular strength ($r = 0.43$, $p < 0.05$). Change in mean pressure from baseline to the 10-min time point was associated with muscular strength ($r = 0.74$, $p < 0.05$). When expressing PWV relative to MAP (PWV/MAP), time effects remained but there were no group-by-time interactions (Table 3, $p > 0.05$). Co-varying for muscular strength abolished all group differences and group-by-time interactions in brachial and central pressures ($p > 0.05$). Interestingly, co-varying for muscular strength abolished time effects for PWV/MAP ($p > 0.05$).

Discussion

Resistance exercise (RE) may be characterized as a series of static muscular contractions performed dynamically that causes a pressure load on the heart and vasculature. In response to acute RE in the present study, women had a markedly attenuated increase in aortic stiffness and central BP compared to men. Reasons for the sex differences in aortic stiffness following acute RE are likely BP mediated. Women had lower brachial and central BP following acute RE. When adjusting for mean distension pressure, sex differences in aPWV no longer remained. Our findings are consistent with previous work that has reported sex differences in ventricular–vascular coupling in response to maximal aerobic exercise[11] with men exhibiting a slight increase in aortic stiffness and women experiencing a decrease in aortic stiffness.[12]

Muscle strength is a significant determinant of the pressor response to static exercise,[13] which in turn is a predictor of increases in aortic stiffness with the perturbation.[14] Women, having lower muscular strength, may have a lower pressor response to acute RE. Indeed, statistically adjusting for strength in our study abolished sex differences in the BP response to acute RE. Our findings differ from recent work from Kingsley et al. that reported no sex differences in aortic stiffness and central hemodynamics following acute RE.[5] Differences in findings are likely due to the training status of participants. Participants in Kingsley et al. were exclusively resistance exercise trained[5] with greater muscular strength and higher BP post RE. Stronger women that are able to generate more force likely experience a greater pressor response during RE and thus a greater increase in aortic stiffness post RE.

In conclusion, women have an attenuated increase in aortic stiffness following acute RE relating to attenuated central hemodynamic reactivity. Underlying sex differences in muscular strength may affect the pressor response to the exercise bout, mediating functional changes in aortic stiffness.

Conflict of interest

We have no conflicts of interest to disclose.

Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.artres.2018.08.002.

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