Hemodynamic and arterial stiffness differences between African-Americans and Caucasians after maximal exercise

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African-American (AA) men have higher arterial stiffness and augmentation index (AIx) than Caucasian-American (CA) men. Women have greater age-associated increases in arterial stiffness and AIx than men. This study examined racial and sex differences in arterial stiffness and central hemodynamics at rest and after an acute bout of maximal exercise in young healthy individuals. One hundred young, healthy individuals (28 AA men, 24 AA women, 25 CA men, and 23 CA women) underwent measurements of aortic blood pressure (BP) and arterial stiffness at rest and 15 and 30 min after an acute bout of graded maximal aerobic exercise. Aortic BP and AIx were derived from radial artery applanation tonometry. Aortic stiffness (carotid-femoral) was measured via pulse wave velocity. Aortic stiffness was increased in AA subjects but not in CA subjects (P < 0.05) after an acute bout of maximal cycling exercise, after controlling for body mass index. Aortic BP decreased after exercise in CA subjects but not in AA subjects (P < 0.05). Women exhibited greater reductions in AIx after maximal aerobic exercise compared with men (P < 0.05). In conclusion, race and sex impact vascular and central hemodynamic responses to exercise. Young AA and CA subjects exhibited differential responses in central stiffness and central BP after acute maximal exercise. Premenopausal women had greater augmented pressure at rest and after maximal aerobic exercise than men. Future research is needed to examine the potential mechanisms.

Reduced arterial compliance is associated with hypertension and coronary artery disease. Carotid to femoral pulse wave velocity (PWV) has been regarded as the gold standard method (36) because of its accessibility and reliability (16). It is associated with incident CVD, independently of traditional risk factors in various populations (35). Even in young individuals, arterial compliance is a predictor of CVD events (61), and it is associated with functional and structural characteristics of the vasculature (13).

AA men have higher resting aortic blood pressure (BP) and aortic stiffness compared with age- and fitness-matched CA men (20, 21). An acute bout of maximal aerobic exercise also elicits a differential response in peripheral arterial stiffness in AA men compared with CA men (20). However, these studies used relatively small subject numbers and included only men. The differences in arterial stiffness may be attributed to a heightened BP response in AA subjects during exercise (47, 57) and the absence of postexercise hypotension after exercise in AA subjects but not in CA subjects (41). AA subjects also have elevated central BP despite comparable brachial BP compared with CA subjects (21). However, we are not aware of any study examining central BP after exercise in AA subjects. Furthermore, postexercise BP may have important diagnostic and prognostic implications (22). Postexercise hypotension is also associated long-term adaptions in BP with exercise training (19, 30); thus, the BP response to acute exercise can provide important clinical and physiological information.

African-Americans (AA) have a much higher prevalence of hypertension, cardiovascular disease (CVD), and renal disease compared with their Caucasian-American (CA) counterparts (55). Hypertension in AA subjects is characterized by higher incidence, earlier onset, longer duration, higher prevalence, and higher rates of hypertension-related mortality and morbidity (15).
there is a positive association between sympathetic nerve traffic and aortic wave reflection in men but a negative association in women. Thus, considering the large increase in sympathetic nerve activity with exercise, one might expect a differential response in aortic wave reflection between men and women after exercise. Furthermore, enhanced wave reflection is mediated by heart rate (HR) in men but not in women (9), suggesting that exercise may differentially affect wave reflection in women. There is also limited evidence showing that increases in arterial stiffness during exercise are greater in hypertensive women compared with hypertensive men (39). We (46) have shown that endurance-trained men and women exhibit different mechanisms producing postexercise hypotension, with a greater role of total peripheral resistance in women, suggesting sex differences in arterial function after exercise. Nevertheless, little is known regarding the integrated arterial response to maximal exercise in young women or if this response is different compared with young men.

It is also not known whether AA women would have different arterial dynamics at rest and after maximal exercise compared with CA women or if there is a general sex by race interaction for arterial responses after exercise. Limited evidence suggests that AA men (5) and AA women (7) may exhibit differential BP responses to cold pressor tests, and this may be dependent on exercise training status. This suggests there may be a sex by race interaction in BP and possibly arterial function after acute exercise, but this has not been investigated to date.

Therefore, the purpose of this study was to investigate arterial stiffness and both central and peripheral BPs after a maximal aerobic exercise test in young, healthy AA and CA men and women. We hypothesized that both AA and CA subjects would have unchanged central arterial stiffness, whereas CA subjects, but not AA subjects, would exhibit decreased peripheral arterial stiffness after a maximal exercise bout. We further hypothesized that CA subjects, but not AA subjects, would decrease both brachial and aortic BPs after maximal exercise. We also hypothesized that women would have reduced changes in arterial stiffness compared with men.

METHODS

Participants. This study was approved by the Institutional Review Board of the University of Illinois. One hundred young (age range: 18–37 yr), healthy individuals (28 AA men, 24 AA women, 25 CA men, and 23 CA women) volunteered for this study and provided signed informed consent.

All subjects were free of CVD, metabolic, renal, or respiratory disease and were nonsmokers. Subjects did not take any medications, including over-the-counter pain/anti-inflammatory medication. All subjects were in normal sinus rhythm and had no history arrhythmias. Subjects were self-defined as AA or CA if they reported that both parents were of African descent or both parents were of Caucasian descent. All subjects were recruited from the local community or university population.

Study design. All subjects reported to the laboratory for 2 days of testing (i.e., fasting blood draws on day 1 and vascular assessment on day 2). For vascular measures on day 2, all subjects were at least 3 h postprandial and did not exercise or consume caffeine or alcohol for 24 h before being tested. Female subjects were tested for vascular assessment during the early follicular phase of their menstrual cycle.

On day 2, subjects rested in the supine position for a period of 10 min in a temperature-controlled room before being tested. The sequence of measures was as follows: brachial artery oscillometry, arterial tonometry, peak O₂ uptake (Vo₂peak) exercise testing, and recovery cardiovascular measurements at 15 and 30 min after exercise. These measurement periods were based on prior research showing racial differences in peripheral arterial stiffness after maximal exercise in men (20).

Anthropometrics. Height and weight were recorded as previously described (21).

Brachial artery BP assessment. After 5 min of quiet supine rest in a dimly lit room, resting systolic BP (SBP) and diastolic BP (DBP) were measured with an automated oscillometric cuff following established guidelines (31). All BP measurements were repeated, and the average of the two values was recorded and used for analysis. If values differed by ≥5 mmHg, a third measurement was obtained, and the two closest values were averaged.

Wave reflection and aortic BP. Applanation tonometry was performed using a high-fidelity strain-gauge transducer (SphygmoCor, AtCor Medical, Sydney, NSW, Australia) on the radial artery to obtain pressure waveforms. Aortic SBP, aortic DBP, aortic mean arterial pressure (MAP), augmented pressure, augmentation index (AIx), and normalized AIx at a HR of 75 beats/min were derived from radial pressure waveforms as previously described (20).

Using a generalized validated transfer function (40), a central aortic pressure waveform was reconstructed from the radial artery pressure waveform to obtain aortic SBP and aortic DBP. This transfer function has been validated at rest and during exercise through invasive methods (48). Aortic MAP was derived from integrating the area under the central BP waveform. The reflected wave pressure amplitude (augmented pressure) was defined as the difference between peak SBP and pressure at the inflection point of the aortic waveform. AIx was calculated as the ratio of the amplitude of the pressure wave above its systolic shoulder (i.e., the difference between the early and late systolic peaks of the arterial waveform) to the total pulse pressure (pulse pressure = SBP – DBP). The result was expressed as a percentage and was used as an index of aortic pressure wave-reflection intensity. Because AIx is influenced by HR, AIx values were also normalized to a HR of 75 beats/min as previously described (20).

PWV. PWV was measured following current guidelines (54). Values from the carotid artery to the femoral artery (cPWV) and from the femoral artery to the superior dorsalis pedis artery (fPWV) were obtained as previously described (SphygmoCor, AtCor Medical) (20). A single high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX) was used to sequentially obtain pressure waveforms between 1) the right common carotid artery and right femoral artery and 2) the right femoral artery and ipsilateral superior dorsalis pedis artery. Consecutive waveforms were captured for a 10-s epoch. Simultaneous ECG gating, as a timing marker, was assessed via a three-lead CM5 configuration and further used to obtain HR. The foot of the pressure wave was identified automatically, removing potential observer bias, using an algorithm that detects the initial upstroke via a line tangent to the initial systolic upstroke point of the pressure tracing and an intersecting horizontal line through the minimum point (12). This algorithm has been shown to be highly reproducible (12). Distances from the suprasternal notch to the femoral artery, carotid artery, and femoral artery sampling site to the superior dorsalis pedis artery were measured as straight lines with a tape measure. The distance from the carotid artery to the sternal notch was then subtracted from the sternal-femoral segment to correct for differences in propagation direction along the arterial path length and taken as a measure of central arterial stiffness. fPWV values were taken as indexes of peripheral arterial stiffness. cPWV values were taken as a measure of central/aortic stiffness. Integral software assessed the pulse wave quality (strength of the pulse wave signal, pulse height variation, pulse length variation, and baseline variation) and SD of mean time differences (SphygmoCor, AtCor Medical). This technique has been shown to be highly reproducible (60).
**Table 1. Subject characteristics**

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th></th>
<th>Women</th>
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<tbody>
<tr>
<td>Age, yr</td>
<td>25 ± 1</td>
<td>25 ± 1</td>
<td>24 ± 1</td>
<td>26 ± 1</td>
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<tr>
<td>Height, cm</td>
<td>178 ± 1</td>
<td>180 ± 1</td>
<td>162 ± 1</td>
<td>165 ± 1</td>
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<tr>
<td>Weight, kg</td>
<td>84 ± 3</td>
<td>83 ± 4</td>
<td>76 ± 4</td>
<td>63 ± 4</td>
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<tr>
<td>Body mass index, kg/m²</td>
<td>26.7 ± 1.1</td>
<td>25.7 ± 1.1</td>
<td>28.9 ± 1.1</td>
<td>23.1 ± 1.2</td>
</tr>
<tr>
<td>Peak O₂ uptake, ml/kg⁻¹·min⁻¹</td>
<td>35.0 ± 1.6</td>
<td>42.1 ± 1.6</td>
<td>27.1 ± 1.7</td>
<td>35.4 ± 1.8</td>
</tr>
<tr>
<td>eGFR, ml/min⁻¹·1.73 m²</td>
<td>105.3 ± 3.3</td>
<td>95.5 ± 3.7</td>
<td>115.4 ± 3.2</td>
<td>88.1 ± 4.2</td>
</tr>
<tr>
<td>Glucose, mg/dl</td>
<td>90.8 ± 4.3</td>
<td>87.8 ± 3.7</td>
<td>93.1 ± 3.2</td>
<td>83.6 ± 4.2</td>
</tr>
<tr>
<td>White blood cell count, mg/dl</td>
<td>5.9 ± 0.4</td>
<td>6.3 ± 3</td>
<td>5.7 ± 3</td>
<td>5.6 ± 0.4</td>
</tr>
<tr>
<td>Triglycerides, mg/dl</td>
<td>96 ± 11</td>
<td>120 ± 9</td>
<td>71 ± 8</td>
<td>77 ± 10</td>
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<tr>
<td>Total cholesterol, mg/dl</td>
<td>160 ± 9</td>
<td>154 ± 7</td>
<td>149 ± 7</td>
<td>147 ± 8</td>
</tr>
<tr>
<td>High-density lipoprotein-cholesterol, mg/dl</td>
<td>44 ± 3</td>
<td>46 ± 2</td>
<td>45 ± 2</td>
<td>57 ± 3</td>
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<td>Very low-density lipoprotein-cholesterol, mg/dl</td>
<td>19 ± 2</td>
<td>24 ± 2</td>
<td>14 ± 2</td>
<td>15 ± 2</td>
</tr>
<tr>
<td>Low-density lipoprotein-cholesterol, mg/dl</td>
<td>97 ± 8</td>
<td>84 ± 7</td>
<td>90 ± 6</td>
<td>74 ± 8</td>
</tr>
</tbody>
</table>

Values are means ± SE. AA, African-American; CA, Caucasian-American; eGFR, estimated glomerular filtration rate. *P < 0.05, sex by race significant interaction; †P < 0.05, significant race differences; ‡P < 0.05, significant sex differences.

**Table 2. Central and peripheral hemodynamics**

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th></th>
<th>Women</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Brachial SBP, mmHg</td>
<td>125 ± 2</td>
<td>130 ± 2</td>
<td>115 ± 2</td>
<td>114 ± 2</td>
</tr>
<tr>
<td>Brachial DBP, mmHg</td>
<td>73 ± 2</td>
<td>70 ± 2</td>
<td>72 ± 2</td>
<td>67 ± 2</td>
</tr>
<tr>
<td>Aortic SBP, mmHg</td>
<td>106 ± 2</td>
<td>107 ± 2</td>
<td>101 ± 2</td>
<td>100 ± 2</td>
</tr>
<tr>
<td>Aortic DBP, mmHg</td>
<td>73 ± 2</td>
<td>71 ± 2</td>
<td>73 ± 2</td>
<td>69 ± 2</td>
</tr>
<tr>
<td>Aortic MAP, mmHg</td>
<td>87 ± 2</td>
<td>87 ± 2</td>
<td>87 ± 2</td>
<td>83 ± 2</td>
</tr>
<tr>
<td>HR, beats/min‡</td>
<td>60 ± 2</td>
<td>65 ± 2</td>
<td>69 ± 2</td>
<td>62 ± 2</td>
</tr>
<tr>
<td>AIx@75, %‡</td>
<td>−4.8 ± 2.4</td>
<td>−7.6 ± 2.4</td>
<td>9.2 ± 2.5</td>
<td>4.4 ± 2.7</td>
</tr>
<tr>
<td>AIx, %</td>
<td>1.9 ± 2.3</td>
<td>−2.8 ± 2.4</td>
<td>12.2 ± 2.5</td>
<td>11.0 ± 2.6</td>
</tr>
<tr>
<td>Augmented pressure, mmHg</td>
<td>1 ± 1</td>
<td>−1 ± 1</td>
<td>4 ± 1</td>
<td>3 ± 1</td>
</tr>
<tr>
<td>cPWV, m/s</td>
<td>6.3 ± 0.2</td>
<td>6.1 ± 0.2</td>
<td>5.7 ± 0.2</td>
<td>5.5 ± 0.2</td>
</tr>
<tr>
<td>fPWV, m/s</td>
<td>8.3 ± 0.3</td>
<td>8.7 ± 0.3</td>
<td>8.3 ± 0.3</td>
<td>8.6 ± 0.3</td>
</tr>
</tbody>
</table>

Values are means ± SE. BP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; AIx, augmentation index (AIX) normalized to a heart rate (HR) of 75 beats/min; cPWV, pulse wave velocity from the carotid artery to the femoral artery; fPWV, pulse wave velocity from the femoral artery to the superior dorsalis pedis artery. *P < 0.05, sex by race significant interaction; †P < 0.05, significant race differences; ‡P < 0.05, significant sex differences.
min postexercise, but there were no race or sex differences and no significant interactions. Significant time by race by sex interactions were found in brachial DBP, aortic DBP, and aortic MAP ($P < 0.05$). The change in both brachial DBP and aortic DBP was significantly greater in CA men at 15 min after exercise compared with 30 min after exercise ($P < 0.05$), and it was also significantly different than AA men at 15 min after exercise ($P < 0.05$). There was no significant difference in the change either brachial DBP or aortic DBP between AA and CA women. The change in aortic MAP was greater in CA subjects compared with AA subjects ($P < 0.05$). There was no significant effect and main effect of time, race, and sex were

Fig. 1. A: change from rest to 15 min (P15) and 30 min (P30) after exercise for brachial systolic blood pressure (bSBP). §The change in bSBP was greater at 30 min compared with 15 min after exercise ($P < 0.05$). There were no significant race, sex, or interaction effects. B: change from rest to 15 and 30 min after exercise for brachial diastolic blood pressure (bDBP). There was a significant three-way interaction. *Caucasian-American (CA) men exhibited a different change from African-American (AA) men ($P < 0.05$). §The change in bDBP was different at 30 min compared with 15 min after exercise ($P < 0.05$) for both AA and CA men. There were no significant differences between AA and CA women and no significant differences between 15 and 30 min after exercise for either AA or CA women. Values are means ± SE.

Fig. 2. A: change from rest to 15 and 30 min after exercise for aortic systolic blood pressure (aoSBP). †There was a significant race difference ($P < 0.05$). The change in aoSBP was greater in CA subjects compared with AA subjects. There were no significant time, sex, or interaction effects. B: change from rest to 15 and 30 min after exercise for aortic diastolic blood pressure (aoDBP). *CA men exhibited a different change from AA men ($P < 0.05$). §The change in bDBP was different at 30 min compared with 15 min after exercise ($P < 0.05$) for both AA and CA men. There were no significant differences between AA and CA women and no significant differences between 15 and 30 min after exercise for either AA or CA women. C: change from rest to 15 and 30 min after exercise for aortic mean arterial pressure (aoMAP). §There was a significant time by race interaction for men, showing that the change in aoMAP from 15 to 30 min after exercise was greater in CA men compared with AA men. †There was an overall race effect ($P < 0.05$) showing a greater change in aoMAP in CA subjects compared with AA subjects. Values are means ± SE.
found in the change in HR. The change in HR in AA men was greater at 15 and 30 min after exercise than AA women (men: 25 ± 2 to 15 ± 2 beats/min; women: 14 ± 2 to 10 ± 2 beats/min). The change in HR was similar in CA men and CA women (men: 26 ± 2 to 18 ± 2 beats/min; women: 23 ± 2 to 16 ± 2 beats/min). CA women also exhibited a greater change in HR compared with AA women ($P < 0.05$). The change in HR was also greater at 15 min compared with 30 min after exercise in all groups ($P < 0.05$). There were no statistically significant effects for the change in cPWV or fPWV. Covariance for VO$_{2\text{peak}}$ did not alter any of these findings. However, after adjustment for BMI, a significant time by race interaction was found for the change cPWV ($P < 0.05$). The change in cPWV was significantly different in CA subjects compared with AA subjects at the 30-min time point after exercise (Fig. 4), with cPWV being below baseline in CA subjects and above baseline in AA subjects, making the difference between the change in cPWV between AA and CA subjects (men and women combined) significant.

The change value of AIx from rest to 15 min after exercise was negatively correlated with the change value of HR ($r =$
0.249, \( P < 0.05 \)) and positively correlated with aortic SBP \((r = 0.266, P < 0.05)\). No correlation was found between the change in AIx and arterial stiffness (cPWV and fPWV) and the change in any of the BP measurements.

**DISCUSSION**

The main finding of the study was that acute maximal exercise induced differential central arterial stiffness and BP responses during exercise recovery in AA and CA subjects. An acute bout of maximal cycling exercise elicited a small increase in central stiffness in AA subjects but a small decrease in central stiffness in CA subjects, making the change in central stiffness significantly different between AA and CA subjects. Another novel finding was that the change in central BP after maximal exercise was reduced in AA subjects compared with CA subjects but that the change in central stiffness was not associated with the change in central BP. These differences cannot be attributed to resting differences in cardiovascular risk factors, as kidney function was normal, resting BP and blood lipids did not differ between CA and AA subjects, and we statistically controlled for differences in BMI. In addition, women exhibited higher aortic wave reflection and a greater change in AIx after maximal aerobic exercise compared with men.

**Racial comparisons.** AA subjects have been shown to exhibit cardiovascular hyperreactivity to stress with an exaggerated BP response to behavioral and physiological (7) sympathoexcitation, which is in accordance with the present study. AA subjects have also been shown to have higher SBP and DBP during submaximal dynamic exercise compared with CA subjects (57). The BP response to exercise is clinically significant since a higher exercise BP response is related to higher CVD risk (52). The exaggerated cardiovascular hyperreactivity may be a result of reduced \( \beta \)-adrenergic sensitivity and augmented \( \alpha_1 \)-adrenergic receptor sensitivity (27), resulting in higher vascular resistance (6). An altered response to sympathetic stimuli might be implicated in the stiffening of large arteries and thus contribute to the development and progression of hypertension and its complications.

Earlier work from our laboratory (21) has shown that resting central BP is elevated in AA subjects despite comparable brachial BP at rest. Here, we extend our previous work by showing that central SBP after exercise changed differently in AA and CA subjects despite comparable changes in brachial BP. However, we did not note increased central arterial stiffness in AA participants compared with their CA counterparts at rest, nor did we see differential responses of leg peripheral arterial stiffness after maximal aerobic exercise, as in the earlier studies (20, 21). It is possible that the discrepancy was a result of different study subjects since our subjects were all sedentary and both men and women were included in the present study. The AA population in the present study was also more overweight and less fit compared with the CA population, whereas BMI and fitness were similar between AA and CA populations in previous studies. However, since we statistically controlled for fitness and BMI, this should not influence our present findings, but this may explain differences compared with earlier investigations.

The mismatch between the changes in central and peripheral arterial stiffness may have been due to differences in BPs. Changes in central SBP were greater in CA subjects than in AA subjects, whereas the changes in brachial SBP were comparable in both groups. Previous work (20) has shown that changes in peripheral PWV match changes in vascular tone, but these were not necessarily associated with changes in peripheral BP. However, it is possible that central arterial compliance is more dependent on changes in central BP than peripheral muscular arteries since we observed that the change in central BP was in the same direction as the change in cPWV, although these changes were in opposite directions for AA subjects compared with CA subjects. Nevertheless, the change in central BP was not associated with changes in cPWV, suggesting that the change in arterial stiffness was independent of changes in central BP. Peripheral arterial stiffness is likely less dependent on BP because it may be subject to other mediators, possibly local vasoactive substances, such as nitric oxide (38), prostaglandin (14), and endothelin (53).

The AA group in our study was less fit and had higher BMI than the CA group. After adjustment for cardiorespiratory fitness levels, none of the results were altered. When we statistically adjusted for BMI, only cPWV was affected. Thirty minutes after maximal exercise, the change in cPWV was in opposite directions for CA versus AA subjects, and the difference in this change was significant. Therefore, central stiffness, but none of the other measured variables, was affected by BMI, but not fitness in our study. Differential arterial stiffness responses in AA and CA subjects may be affected by structural factors such as the composition of the arterial wall, including the contents of the extracellular matrix (37). Arteries become wider and less elastic with a reduction in arterial elastin and an increase in collagen content (25). The wall thickness of large arteries correlates well with the burden of generalized atherosclerosis and is a reliable predictor of coronary events (44). AA subjects had greater mean aortic wall thickness than CA subjects and higher age-related mean maximal aortic wall thickness (28, 45). In addition, AA subjects tend to have higher relative collagen content in the aorta than CA subjects between 30 and 69 yr of age (32). AA subjects also have a tendency to overgrow connective tissue in response to diseases (42). Although we did not measure arterial wall composition in our study, the higher content of collagen in the aortic arterial wall in AA subjects may affect elastic properties of the large arteries after exercise. It should be noted the studies revealing structural differences between AA and CA groups were population-based studies with a wide age range from teenagers to older individuals. The fact that we did not observe differences in central stiffness between AA and CA subjects at rest confirmed the use of acute exercise as physiological perturbation to evoke racial difference in elastic properties in the central arteries in young individuals free of overt CVD.

**Sex comparisons.** In agreement with previous studies (10, 29), women demonstrated higher baseline levels of aortic wave reflection compared with men. When normalized for HR, AIx remained higher in women. Augmented pressure was also increased in women. The majority of wave reflection as seen by the heart is attributable to the branching of the aorta at the celiac trunk and renal arteries and its terminal tapering (33). These sex differences have been attributed to women with shorter body height and an associated shorter aorta, which, in turn, results in earlier wave reflections (49). Even in height-matched elderly hypertensive subjects, women had earlier
arterial wave reflection, although of similar magnitude, compared with men, possibly explained by a reduced aortic arterial diameter of ~5% and increased aortic stiffness (17). Both factors are likely to lead to an increased cPWV; however, in our study as well as another study in young healthy individuals (43), cPWV was higher in men at rest, suggesting there may be other factors modulating central arterial stiffness. It should also be noted that the traditional way of interpreting wave reflection was based on the assumption that the central arterial was a uniform tube with single reflection site and that forward and backward waves maintain their shape while traveling to the end of the tube and back (50, 51). The disassociation between resting cPWV and AIx in men and women in our study may be explained by recent evidence suggesting the traditional model may be oversimplified and that backward waves are likely compound waves consisting of echo from many reflection sites (58, 59). In an elastic aorta, AIx is more likely to be related to the intensity of the reflected wave rather than to its velocity. Also, in healthy men, vasoactive drugs can change aortic AIx independently from changes in cPWV, suggesting that AIx and cPWV are not necessarily associated (23).

To our knowledge, the present study is the first study examining sex differences in arterial wave reflection after an acute bout of maximal aerobic exercise. The change in wave reflection after maximal exercise was greater in women than in men. Submaximal exercise at a HR of 100 beats/min has been shown to decrease AIx in young men and women similarly during exercise (3). Sex differences during recovery from high-intensity exercise may be due to differential levels of catecholamines, as higher levels of catecholamines in men result in increased AIx and greater HR during recovery (8). Thus, the difference in AIx could be a function of differences in HR, as higher HR is independently associated with a reduction in aortic AIx during β-adrenergic stimulation (26).

However, the difference in HR between men and women is unlikely to explain the sex difference in AIx, since there was still a sex difference in the change in wave reflection when we controlled for HR (normalized AIx at a HR of 75 beats/min). Muscle sympathetic nerve activity may play an important role in modulating aortic waveforms in men and women, as positive correlations have been found between sympathetic activity and wave reflection in men but not an association was found in women (10). Considering the larger increase in sympathetic nerve activity with exercise, it may not be surprising to find differential changes in aortic wave reflection during recovery. However, the mechanism underlying the inverse relationship remains unknown; therefore, we can only speculate the potential mechanisms. An increased sensitivity to β-adrenergic receptor stimulation in women may be partly responsible for the observed differential responses (24). Greater β-adrenoceptor-mediated vasodilatation in resistance arteries in women during recovery from exercise may contribute to differences in central wave patterns compared with men.

Female sex hormones may also be responsible for this differential response in women. Follicle-stimulating hormone (FSH) is highly correlated with arterial mechanical properties and may be the most sensitive marker of declining arterial function with aging (56). Our female subjects were all tested during the early follicular phase of the menstrual cycle, which is characterized by a rise in FSH during the first days of the cycle. It is possible that the differences between men and women were increased due to FSH. Finally, estrogen has direct vasodilatation effects by stimulating endothelial nitric oxide synthase activity via activation of estrogen receptor-α (11, 18) and potentially decreases wave reflection. However, the contribution of estrogen is probably limited because the surge of estrogen and the associated decline in AIx occurs only during late follicular phase of the menstruation cycle (1). Future research is warranted to examine the hormone levels and other potential mechanisms responsible for the observed differential responses in men and women.

**Strength and limitations.** The strength of the present study is a relatively large sample size. In addition, women were tested during the early follicular phase of their menstrual cycle to minimize the effect of female hormones. The design of the study also allowed us to examine baseline differences in our four groups as well as cardiovascular responses after an acute bout of exercise. Standard maximal aerobic exercise was used as a stressor to elicit cardiovascular responses that may not be evident at rest.

We did not control for socioeconomic status; however, we recruited most of our subjects from university students, and, therefore, they had a similar level of education. Furthermore, this is a cross-sectional study, and cause-effect cannot be inferred.

We did not directly measure aortic stiffness or central wave reflection but used noninvasive validated and well-accepted techniques. cPWV is an accepted standard measure of arterial stiffness and has been used in most large-scale studies. Also, central wave reflection, although derived from a transfer function, is valid both at rest and after exercise, as shown by validation compared with invasive techniques (48). Furthermore, although we screened subjects based on health history, we do not know if any of the subjects had latent coronary artery disease, diastolic dysfunction, or left ventricular hypertrophy, which might have affected our results. However, given the young age and general health status of our subject population, it is unlikely that these factors exhibited a significant influence on our results. Also, none of the subjects experienced any signs or symptoms of coronary artery disease during the maximal exercise test.

**Conclusions.** Young AA and CA subjects exhibited differential responses in central stiffness and central BP after acute maximal exercise. Premenopausal women had greater AIx and augmented pressure at rest and exhibited greater reduction in aortic wave reflection after maximal aerobic exercise.

**GRANTS**

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**DISCLOSURES**

No conflicts of interest, financial or otherwise, are declared by the author(s).

**AUTHOR CONTRIBUTIONS**


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