Racial Differences in Arterial Stiffness During Recovery Following Repeated Bouts of Maximal Anaerobic Cycling Exercise in Young Women

Noelle Merchant
RACIAL DIFFERENCES IN ARTERIAL STIFFNESS DURING RECOVERY FOLLOWING REPEATED BOUTS OF MAXIMAL ANAEROBIC CYCLING EXERCISE IN YOUNG WOMEN

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ABSTRACT

RACIAL DIFFERENCES IN ARTERIAL STIFFNESS DURING RECOVERY FOLLOWING REPEATED BOUTS OF MAXIMAL ANAEROBIC CYCLING EXERCISE IN YOUNG WOMEN

August 2020

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African Americans (AA) experience a differential cardiovascular response during recovery from aerobic exercise compared to Caucasian Americans (CA). There is also evidence that local and systemic vasculature responds differently to aerobic exercise stimulus. No study has examined racial differences in arterial stiffness following maximal anaerobic exercise in AA and CA women.

**Purpose:** To compare local and systemic arterial stiffness during recovery from repeated bouts of maximal anaerobic exercise in young, healthy AA and CA women.

**Methods:** Twenty (AA=8; CA=12) young, healthy women without hypertension completed two bouts of maximal anaerobic exercise on a cycle ergometer with 30 minutes of
recovery between bouts. Brachial blood pressure and hemodynamic measurements were taken at rest and 5-, 15- and 30- minutes following each bout of exercise. Arterial compliance (AC) was assessed using ultrasound to evaluate local carotid arterial stiffness. Systemic arterial stiffness measured by augmentation index (AIX@75) and segmental arterial stiffness measured by central Pulse Wave Velocity (cPWV) were assessed with SphygmoCor. Two-way repeated measures ANOVA was used to test for possible race (two levels) and time (7 levels) differences between AA and CA.

**Results:** There was a significant race by time interaction for AC and MAP (P ≤ 0.5). There was a main effect of time for AIX@75, which increased immediately following exercise and returned to baseline 30 minutes following exercise, regardless of race (P ≤ 0.05). There was no change in cPWV in response to exercise in either group.

**Conclusion:** Local carotid arterial compliance and brachial blood pressure respond differently in AA women compared to CA women following repeated bouts of maximal anaerobic exercise, despite similar systemic or segmental arterial stiffness. Racial differences in arterial distensibility between AA and CA women in response to maximal anaerobic exercise may be limited to localized changes in the predominantly elastic carotid artery. In addition, while no racial differences were observed in systemic arterial stiffness, repeated maximal anaerobic exercise induces peripheral arterial stiffness but does not induce changes in central arterial stiffness in all participants.
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CHAPTER 1

INTRODUCTION

Background

While heart disease and stroke are the leading causes of death for all Americans, African Americans are at an even greater risk of developing cardiovascular related diseases. (Carnethon et al., 2017). It has been established that African American (AA) women exhibit disparities in the prevalence of cardiovascular disease (CVD) and impaired vascular function compared to Caucasian women (CA) (Vita et al, 2003). AA have a higher incident of CVD and vascular dysfunction, with more severe disease, and earlier onset, compared to their white counterparts (Carnethon et al., 2017). According to the Heart Foundation, 47% of AA women have heart disease compared to 33% of CA women (African Americans and Heart Disease, 2018). The development of coronary artery disease, stroke and other cardiovascular events may be independently associated with impaired vascular function, specifically increased arterial stiffness of large arteries (Ben-Shlomo, 2014). The stiffening of large arteries increases the speed of the ejected blood flow from the left ventricle through the arteries, which results in an earlier return of the reflected pressure wave. The early arrival of the reflected wave during systole increases the afterload on the left ventricle and reduces
coronary artery perfusion pressure during diastole. This increased arterial stiffness is believed to contribute to the development of atherosclerosis (Hodson et al., 2017; Townsend et al., 2017). However, the mechanism responsible for the early onset and severity of hypertension and CVD in AA is unclear. Even young, normotensive AA have been shown to exhibit a heightened vascular response to physical stress despite having normal resting cardiac function (Yan et al., 2014; Zion et al., 2003).

Previous research has shown that resting cardiac and vascular function may be similar in AA and CA adults (Yan et al., 2017). However, to determine if there are underlying physiological differences, exercise can be used as a potent sympathetic stimulus to unmask potential differences not apparent during rest. Previous findings observed racial differences between AA and CA during recovery from acute exercise. Yan et al. observed young AA were found to have greater local carotid arterial stiffness 30 minutes following an acute bout of submaximal aerobic exercise compared to CA, despite similar resting values (2017). Aerobic exercise acutely reduces peripheral artery stiffness, while resistance exercise has been shown to transiently increase arterial stiffness (Naka et al., 2003; Heffernan et al., 2007c; Rossow et al., 2010). Anaerobic sprint exercise has been shown to transiently increase arterial stiffness, similar to that of resistance exercise (Rossow et al., 2010; Sagiv et al., 1999). Despite the transient increase in arterial stiffness, high intensity sprint training may provide cardiovascular benefits similar to aerobic exercise training. With the increasing popularity of high-intensity interval training (HIIT), and the known increased CVD risk in
AA adults, it’s important to examine if there are any racial differences in arterial stiffness after maximal anaerobic exercise.

The steep ramp test (SRT) is a short, maximal anaerobic test performed on a cycle ergometer that doesn’t require the use of breath gas analysis. The use of SRT as a fitness test has recently gained popularity due to the ease of use in clinical and research settings and it requires minimal training to administer (Bongers et al., 2013). The SRT uses a fast, increasing workload of 25W every 10 seconds until the participant reaches volitional exhaustion or cannot keep up with the 50 RPM cadence (Rozenberg et al., 2014). It typically takes between 1 minute to 4 minutes to complete depending on fitness level, and it is generally well tolerated by various populations due to the incremental ramp nature of the test. Prediction equations can be used to estimate an individual’s fitness level (VO2max) and can be used for exercise prescription (Rozenberg et al., 2014).

Several measurements are available to assess arterial stiffness. Arterial stiffness may be caused by arteriosclerosis, a degenerative stiffening of the arterial wall. Arterial stiffness is influenced by the structural and functional components associated with the intrinsic elasticity of the artery (Cho & Kim, 2016; Arnett 2001). Pulse wave analysis, using augmentation index (AIx), is an indication of systemic arterial stiffness. AIx is the ratio of the amplitude of the pressure wave above its systolic shoulder, and pulse pressure. (SphygmoCor XCEL System). The value obtained from AIx indicates the size of the increase or decrease in the pulse height that is formed as a result of the reflected wave. The pulsatility and stiffness of the large artery can also be assessed locally on the common carotid
artery. Echo tracking technology (E-tracking) allows for measurements of arterial stiffness by using ultrasound echo radiofrequency signals to track the changes in the vessel wall diameter (Yu et al., 2012).

This study examined the racial differences in arterial stiffness after repeated bouts of acute maximal anaerobic exercise in African American and Caucasian women. The results of this study would provide important information on the acute vascular response to exercise in this high-risk population. The understanding of potential racial differences may contribute to targeted exercise prescription, and therefore reducing the health disparities between AA and CA women.

Aims and Hypotheses

Aim 1: To examine the racial difference in local carotid arterial stiffness during recovery from repeated bouts of maximal anaerobic cycling exercise in African American and Caucasian women.

Hypothesis: African American women would have a greater increase in local carotid arterial stiffness and reduce arterial compliance after repeated bouts of maximal anaerobic exercise, compared to Caucasian American women.

Aim 2: To examine the racial differences in systemic arterial stiffness during recovery from repeated bouts of maximal anaerobic cycling exercise in African American and Caucasian young women.
**Hypothesis:** African American women would have a greater increase in systemic arterial stiffness after repeated bouts of maximal anaerobic cycling exercise, compared to Caucasian women.

**Racial Disparities in Cardiovascular Disease Risk**

Cardiovascular disease accounts for nearly 1 of 3 deaths in the United States each year, resulting in more than $316 billion in direct and indirect costs (Heart Disease and Stroke Statistics, 2017). The highest percentage of individuals suffering from CVD are non-Hispanic African Americans (Levenstein et al, 2001). With the numbers reaching nearly half, 48% of AA women and 46% of AA males have known CV, compared to 32% of white women and 35% of white men (Heart Disease and Stroke Statistics, 2017). Additionally, African Americans have the highest prevalence of hypertension in the world, with 40% of all adult AA being diagnosed with hypertension (Levenstein et al, 2001). In addition to greater frequency of developing hypertension, AA develop it earlier and have increased complications of CVD (Zion et al., 2003). When those numbers are broken down by sex, AA women have the highest prevalence of hypertension at 47% compared to men, at 43% (Zhao et al., 2015). While high blood pressure and aging have been shown to increase arterial stiffness, AA men have shown signs of increased arterial stiffness as early as 21 years of age (Zion et al., 2003; Heffernan et al., 2007). Chronically high blood pressure can result in the development of arterial stiffness, and central arterial stiffness has been reported to be higher in AA than non-Hispanic whites (Buie et al., 2019).
Despite the staggering statistics, the physiological mechanism for these disparities are unclear. Therefore, it’s important to investigate possible mechanisms that may explain the racial disparities in regard to CVD.

**Local Carotid Arterial Stiffness**

In addition to traditional CVD risk factors, such as hypertension and obesity, increased arterial stiffness and alterations to arterial wall compliance has been shown to be a predictor of future CVD, prior to onset of disease (Zion et al., 2003). In normal, healthy arteries, elastin and collagen fibers in the artery wall allow for optimal oxygen delivery to organs and tissue. However, chronically elevated blood pressure causes changes to the elastic properties in the wall which results in stiff, non-compliant arteries (Buie et al., 2019). The disruption of elastin and collagen fibers disturbs normal vessel dilation and constriction in response to systolic and diastolic cardiac cycles. The carotid artery is the most susceptible artery to the effects of hypertension in relation to increased wall stiffness. Arterial stiffening is a general phenomenon in large central elastic arteries, and the change in carotid artery stiffness with age has been shown to be similar to that of the abdominal aorta in normotensive individuals (Kawazaki et al., 2005). Therefore, because the carotid artery is more accessible than the abdominal aorta, arterial stiffness measurements of carotid artery is an appropriate alternative to aorta measurements for determining arterial stiffness.

Stiffer arteries include calcifications, large amounts of collagen, and ruptured pieces of elastic tissue (Matthew et al., 2011). Plaque formation in large elastic arteries such as the carotid artery are positively correlated with risk factors such as obesity and hypertension (Jin
et al., 2012). Increased arterial stiffness leads to changes in the stress-strain relationship, which results in accelerated hypertension and eventually the development of atherosclerosis (Zion et al., 2003). This damage to the arterial wall increases pressure, which may further break down the elastin and collagen properties, increasing stiffness and reducing compliance. Researchers have found that reduced arterial compliance and increased arterial stiffness is associated with increased cardiovascular and all-cause mortality (DeLoach and Townsend, 2008). Since arterial stiffness can appear before any other apparent CVD risk factors, early detection is vital to reduce morbidity and mortality related to CVD. This is particularly important in high-risk populations.

Pressure-strain elasticity (EP), arterial compliance (AC) and beta stiffness index ($\beta$) are commonly used as non-invasive indicators of local arterial stiffness. The buffering capacity of elastic arteries, such as the carotid artery, is affected by the structure and the diameter of the vessel. During the cardiac cycle, changes in the diameter of the artery occur with the blood flow, by increasing in diameter during systole and decreasing during diastole (Jin et al., 2012). However, when the artery becomes stiffer, the degree of changes to the diameter of the vessel decreases. This can result in the augmentation of blood flow through the arterial tree. Echo tracking technology allows for measurements of arterial stiffness by using ultrasound echo radiofrequency signals to track the changes in the vessel wall diameter (Yu et al., 2012). EP, which is associated with the elastic modulus, is used to measure the stress applied to the vessel wall and is affected by pulse pressure. EP is calculated as $EP = (P_{\text{max}} - P_{\text{min}}) / [(D_{\text{max}} - D_{\text{min}}) / D_{\text{min}}]$. 


Beta-stiffness, which is independent of pressure, is calculated by the natural logarithm of the systolic and diastolic blood pressure ratio. \( \beta \) is calculated as \( \beta = \ln(P_{\text{max}}/P_{\text{min}})/[(D_{\text{max}} - D_{\text{min}})/D_{\text{min}}] \) with \( D_{\text{max}} \) and \( D_{\text{min}} \) representing the maximum (systolic) and minimum (diastolic) diameters, and \( P_{\text{max}} \) and \( P_{\text{min}} \) are the highest (systolic) and lowest (diastolic) carotid pressures, respectively. AC is an indicator of vessel compliance that looks at the ratio of the area of change of the artery to the pulse pressure. AC is calculated as: \( AC = \pi (DD_2 - SD_2)/4(SBP - DBP) \), with \( DD \) representing diastolic (or minimum) arterial diameter, \( SD \) representing systolic (or maximum) arterial diameter, \( DBP \) representing lowest (diastolic) carotid pressures, and \( SBP \) representing highest (systolic) carotid pressure. These measurements are important tools that have allowed researchers to discover possible mechanisms for hypertension and CVD before symptoms appear. This is particularly important in AA populations due to the early development and accelerated progression of hypertension and CVD.

**Local Arterial Stiffness Post-exercise**

Aerobic exercise training has traditionally been used to reduce CVD risk in adults. An acute bout of moderate aerobic exercise has been shown to transiently increase arterial compliance in young, healthy men (Kim et al., 2017; Kingwell et al., 1997). However, high-intensity interval training (HIIT) has recently gained popularity due to the short time frame necessary to complete the exercise and the additional benefits it may provide compared to continuous aerobic exercise (Rossow et al., 2010).
Chronic HIIT has been shown to have an overall positive effect on cardiovascular health and may improve cardiovascular fitness, however acute bouts of anaerobic exercise may result in transiently increased arterial stiffness (Burgomaster et al. 2005; Larsen et al. 2014). Limited research has shown HIIT elicits a similar arterial response to what is observed with resistance exercise, such as increased AC and $\beta$ (Rosso et al., 2010; DeVan et al., 2005). Babcock et al. found that a single bout of anaerobic exercise using the Wingate protocol significantly increased $\beta$ and Ep in healthy adults (2015). These finding are consistent with previous research showing that repeated bouts of acute supramaximal exercise reduced AC and increased $\beta$ in healthy adults (Rossow et al., 2010). Following supramaximal exercise, $\beta$ was not increased after the first bout, despite observing a decrease in AC. However, a second bout of supramaximal anaerobic exercise resulted in a further reduction in AC and significant increases in $\beta$ compared to resting levels (Rossow et al., 2010). These results suggest there may be a cumulative effect of anaerobic exercise on arterial compliance and stiffness.

Therefore, based on current research, anaerobic exercise has been shown to increase local carotid artery stiffness and reduces arterial compliance in young healthy adults.

**Racial Differences in Local Arterial Stiffness at Rest and Post-exercise**

The exact mechanism for the racial differences in hypertension and CVD between AA and CA adults is unclear, however some research has suggested it may be associated with reduced arterial compliance and increased arterial stiffness, even in young healthy individuals (Yan et al., 2014). AA adults were shown to have 9% greater increase in resting $\beta$ when compared to CA counterparts (Din-Dzietham et al., 2004).
Young, apparently healthy and normotensive AA women have been shown to have similar resting brachial blood pressure as CA, however they exhibit higher central blood pressure, vascular dysfunction and increased arterial stiffness (Yan et al., 2014). AA adults, in particular, are more likely to experience stiffness in the elastic arteries, such as the aorta and carotid arteries, than CA adults. This may contribute to the increased CVD risk factors in AA (Buie et al., 2019). Additionally, normotensive, apparently healthy AA men with no family history of hypertension were found to have differences in resting arterial compliance and autonomic modulation, when compared to matched non-AA males (Zion et al., 2003).

In a large, community-based study of older adults, Markert et al. found that AA had higher carotid artery stiffness when compared to non-Hispanic whites at rest (2011). Arterial diameter was also measured and there were no racial differences found between AA and non-Hispanic white adults, despite the difference found in carotid artery stiffness. These findings suggest that age may be associated with carotid artery stiffness in AA but not in CA individuals. They also determined that age may not be associated with carotid artery diameter when comparing AA and non-Hispanic whites. Vessel diameter typically increases with age related arteriosclerosis, which is due to thickening of the intima-media layer and an increase in collagen fibers in the arterial wall, which increases stiffness (Market et al., 2011; van Popele et al., 2001). These findings may explain the racial disparity in incidents of stroke in older individuals.

Contrary to the previous findings in older adults, no differences were found in arterial stiffness at rest in young healthy men and women (Yan et al., 2017; Ranadive et al., 2015).
Since exercise has been shown to have an effect on local arterial stiffness, the researchers sought to determine if there would be any racial differences after an acute bout of submaximal exercise. Interestingly, Ep and \( \beta \) were increased in both groups 30 minutes post-exercise, similar to what has been observed with HIIT and resistance exercise (Yan et al., 2017). Additionally, AA participants exhibited greater Ep and \( \beta \) when compared to CA participants (Yan et al., 2017). These results suggest that AA adults have an exaggerated hemodynamic response to an acute bout of submaximal aerobic exercise when compared to CA adults. However, there is no known research on racial differences in local arterial stiffness in women following dynamic anaerobic exercise.

**Systemic Arterial Stiffness**

In healthy populations, the cardiovascular system works in conjunction to prevent wide fluctuations in pressure. The arterial tree starts with the aorta, a compliant artery, and as the arteries branch throughout the periphery, they become stiffer. When the wave travels from the compliant arteries to the stiffer arteries, a reflected wave is generated backwards. This prevents high pulsatility of blood flow to travel to the delicate microvasculature of end-organs. The magnitude and speed of the wave is a representation of systemic arterial stiffness (Mitchell et al., 2011). Increased arterial stiffness results in a higher magnitude of the reflected wave, increases in left ventricular afterload and decreases coronary perfusion. This is highly correlated to hypertensive target-organ damage (Heffernan et al., 2007).
Pulse wave analysis, using augmentation index (AIx), is a measure of systemic arterial stiffness (Figure 1). AIx, expressed as a percentage, is the ratio of the difference between the early and late systolic peaks of the arterial waveform and pulse pressure. AIx is calculated as the augmentation pressure (AP) divided by pulse pressure (PP) x 100. Due to the influence of heart rate on AIx, it is typically adjusted to a heart rate of 75 beats per minute (AIx@75). The value obtained from AIx indicates the size of the increase or decrease in the pulse height that is formed as a result of the reflected wave. If the peak of reflected wave is greater than the peak of the forward wave, AIx has a positive value. Conversely, if the peak of the reflected wave is smaller than the peak of the forward wave, the AIx will have a negative value. Therefore, a higher AIx is associated with increased systemic arterial stiffness (Mitchell et al., 2011; Townsend et al., 2015; Yan et al., 2014).

Figure 1. Schematic of augmentation index (AIx). Augmentation is calculated at the ratio of pulse pressure and the difference between systolic and diastolic blood pressure
Systemic Arterial Stiffness Response to Exercise

Aerobic exercise training has been used to reduce CVD risk factors in adults, however, the effect of resistance/anaerobic exercise training on arterial stiffness remains controversial (Bond et al., 2015; Gottschall et al, 2014). Understanding the acute arterial response to exercise is important when determining the appropriate exercise mode and intensity. Aerobic exercise has been consistently shown to decrease arterial stiffness following an acute bout of maximal exercise (Kingwell et al., 1997; Naka et al, 2002). AIx was significantly lower 20 minutes after recovery from maximal aerobic exercise when compared to resting measurements in a group of young, healthy men (Heffernan et al, 2007b). In addition, compared to rest, women had a greater reduction in AIx@75 at 15 and 30 minutes after an acute bout of maximal aerobic cycling exercise than was observed in men (Yan et al., 2014). While the research on aerobic exercise suggests arterial stiffness is reduced after an acute bout of exercise, few studies have examined the effect of anaerobic exercise on systemic arterial stiffness. High intensity interval training (HIIT) has recently gained popularity for its short duration (Bartlett et al., 2011). While some research has shown chronic HIIT has positive cardiovascular benefits, similar to aerobic exercise training, it may transiently have negative effects on the arterial system following acute exercise bouts (Kemi, O., 2012). This is due to increased arterial stiffness and reduced arterial compliance, similar to what has been observed with resistance exercise (Burgomaster et al. 2005; Larsen et al. 2014; Rossow et al., 2010).
Kingsly et al. found repeated bouts of maximal anaerobic exercise, utilizing the Wingate protocol, resulted in increased systemic arterial stiffness, as indicated by increased AIx@75, in young, healthy men (2016). These results were observed after the third bout of exercise, at 1-minute following exercise. However, since this study only focused on men, it unclear if these observations would appear in healthy, normotensive women after an acute bout of anaerobic exercise.

**Racial Differences in Systemic Arterial Stiffness at Rest and Post-exercise**

African Americans not only have a higher rate of developing hypertension, they also develop hypertension earlier in life, which results in hypertension for a longer duration and higher rates of hypertension-related mortality and morbidity (Yan et al., 2014; Flack, 1995). Previous studies have used AIx to examine the differences in systemic arterial stiffness in different racial groups, including AA and CA. Race was found to be an independent predictor of increased resting AIx when comparing AA and CA adults (Morris et al., 2012; Heffernan et al., 2008). Young, healthy, normotensive AA were found to have significantly greater AIx, AIx@75, augmentation pressure (AP), and faster arterial reflection time when compared to CA. These results suggest the AA participants had greater systemic arterial stiffness when compared to the CA participants, even after controlling for body fat and cardiorespiratory fitness, and despite having similar resting brachial blood pressures (Heffernan et al., 2008). These findings suggest that race may be an independent predictor of greater AIx in AA adults when compared to CA.
In addition to resting measures, acute exercise may also result in a differential arterial response. An acute bout of maximal aerobic cycling exercise was shown to induce small increases in systemic arterial stiffness in AA participants, but resulted in small decreases in arterial stiffness in CA participants. This resulted in significant differences between the groups, despite no racial differences in resting AIx@75 (Yan et al., 2014). These results support the idea that exercise can unmask physiological differences not apparent at rest in apparently healthy adults. However, to our knowledge, there is no research that assesses racial differences in systemic arterial stiffness in women after an acute bout of anaerobic exercise.

**Maximal Anaerobic Exercise**

Acute anaerobic exercise has been shown to result in transiently increased arterial stiffness and decreased arterial compliance, similar to what is observed in resistance exercises. However, there is limited research on the arterial response in AA adults and no research on the effect of high intensity cycling on AA women. The steep ramp test (SRT) is a short, maximal anaerobic test performed on a cycle ergometer that doesn’t require the use of breath gas analysis. It has recently gain popularity due the ease of use in clinical and research settings and the minimal training necessary to administer. The SRT typically takes between 1 minute to 4 minutes to complete, depending on fitness level, and is generally well tolerated by various populations due to its incremental ramp nature. It has been validated in special populations such as type 2 diabetes, children, chronic heart failure patients and chemotherapy patients and (Rozenberg et al., 2014).
Prediction equations can be used to estimate an individual’s fitness level (VO2max) and can be used for exercise prescription. The SRT has been shown to be more accurate in estimating cardiorespiratory fitness than submaximal exercise protocols (Rozenberg et al., 2014). The SRT uses a fast, increasing workload of 25W every 10 seconds until the participant reaches volitional exhaustion or cannot keep up with the 50 RPM cadence. For the purposes of this project, the SRT would be used as a maximal, high intensity cycling test that may transiently increase arterial stiffness. The repeated bouts may also result in a compounding effect by further increasing arterial stiffness from baseline after the 2nd bout of exercise.
CHAPTER 2

DESIGN AND METHODS

Design Overview

A total of 20 CA (n=12) and AA (n=8) women between the ages of 18-35 years of age participated in this study. The recruited population were free of any major disease, not taking any medications that may alter the cardiovascular response to exercise and safely able to exercise. Details of the inclusion and exclusion criteria are explained below under Participant Recruitment and Screening. The eligible participants participated in two bouts of anaerobic cycling exercise using the SRT protocol. Vascular and hemodynamic measurements were taken at rest, and 5 (P5-post-SRT1), 15 (P15 post-SRT1), and 30 minutes (P30 post-SRT1) following a maximal anaerobic exercise test on cycle ergometer, and 5 (P5 post-SRT2), 15 (P15 post-SRT2), and 30 (P30 post-SRT2) minutes following the second maximal anaerobic exercise test (Figure 2).
**Participant Recruitment and Screening**

Participants were recruited from within the University of Massachusetts, Boston (UMB), through flyers posted across the UMass Boston campus, mass emails to the UMB campus community, tabling events in heavily trafficked areas of UMass Boston, and word of mouth. Participants were included if they met the following criteria:

- Females aged 18-35 years,
- Individuals who are premenopausal women, who can be on combined estrogen/progestin hormonal contraceptive therapy (oral pill, transdermal patch, or vaginal ring),
- Individuals that are able to exercise and are free of any major disease as determined by answering “NO” on the Physical Activity Readiness Questionnaire for Everyone (PAR-Q Plus),

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**Figure 2. Schematic of Testing and Measurement Protocol.** Measurements for all blood pressure and arterial stiffness variables were taken following a 5-minute rest and then during recovery from exercise at 5, 15- and 30- minutes following each SRT exercise.
• Individuals who are self-defined African American or Caucasian as indicated in the demographic questionnaire.

Individuals unable to consent for themselves were excluded from the study. Furthermore, individuals who met any one of the following exclusion criteria were excluded from the study:

• Individuals less than 18 years of age or greater than 35 years of age were not included due to age related changes in vascular function;
• Individuals who were pregnant or lactating;
• Individuals taking medications that may affect central or peripheral circulation or blood pressure, were on nonsteroidal anti-inflammatory agents or serotonin reuptake inhibitors;
• Individuals who smoke or chew tobacco, including e-cigarettes or have in the last 6 months;
• Individuals who have diabetes, hypertension with blood pressure >140/90 mmHg, congestive heart failure, angina, or peripheral vascular disease;
• Individuals with history of serious arrhythmias and/or acute myocardial ischemia at rest or during exercise as indicated on the screening questionnaires.

Participants completed a PAR-Q Plus, and health history questionnaire which included medical history, pregnancy status, blood pressure, medication list, family history and exercise history. A demographic questionnaire was also completed prior to their initial visit to ensure all inclusion criteria are met. Participants were included in this study if
they were African American, having origins in any of the black ethnic groups (not of Hispanic origin) or Caucasian, having origins in any of the original peoples of Europe, North Africa or the Middle East.

**Study Visit**

Participants reported to the Cardiovascular Exercise Physiology Lab at UMass Boston on the day of their study visit. Upon arrival, research assistants reviewed all documentation to ensure pre-participation screening documents were completed and inclusion criteria was met. Participants were asked to be fasted for at least 3 hours, to refrain from consuming caffeine on the day of the study visit, and to avoid exercise and alcohol for 12 hours prior to the study visit. A review of the informed consent document was provided by the graduate research assistant to the participant and once consent was given, resting measurements were taken. Height and weight were measured using a stadiometer (to the nearest 0.5 cm) and a beam balance platform scale, respectively. Participants then rested in the supine position for 5 minutes to obtain baseline resting measurements. (SphygmoCor XCEL System; AtCor Medical Pty Ltd, Sydney, Australia). All blood pressure and arterial stiffness measurements were assessed at the beginning of the visit before the initiation of the first maximal anaerobic exercise test.
Measurements

Brachial Blood Pressure and Aortic Blood Pressure

Blood pressure measurements were recorded using an automated cuff (SphygmoCor XCEL System, AtCor Medical Pty Ltd, Sydney, Australia). A brachial BP cuff was placed on the upper right arm to obtain brachial systolic and brachial diastolic blood pressure.

Mean arterial pressure (MAP) was calculated using the equation:

\[
\text{MAP (mmHg)} = \frac{1}{3} (\text{SBP} - \text{DBP}) + \text{DBP}
\]

Maximal Work on Cycle Ergometer

Maximal wattage obtained during the SRT test was determined by the following equation

\[
\text{Watt_max} = \text{kg} \times (25/0.5)
\]

Systemic Arterial Stiffness Measurements

AIX was used as an indication of peripheral systemic arterial stiffness. To assess AIX, brachial pressure waveforms were obtained, and generalized transfer functions were used to construct central aortic waveforms (SphygmoCor XCEL System; AtCor Medical Pty Ltd, Sydney, Australia). Aortic parameters are derived from the average central pressure waveforms (SphygmoCor User Manual, 2019). AIX is the ratio of the amplitude of the pressure wave above its systolic shoulder and pulse pressure.
It is typically expressed as a percentage and calculated as the augmentation pressure (AP) divided by pulse pressure (PP) x 100. AP is derived from the difference between the two aortic pressure peaks during systole and PP is the difference between the maximum and minimum of the aortic PP. Due to the influence of heart rate on AIx, it is adjusted to a heart rate of 75 beats per minute (AIx@75). The value obtained from AIx indicates the size of the increase or decrease in the pulse height that is formed as a result of the reflected wave.

**Local Carotid Arterial Stiffness Measurements**

Local carotid arterial stiffness was assessed using echo-tracking (E-tracking) software on an Ultrasound machine (Hitachi Aloka Medical, Ltd). The Ultrasound technique requires a simultaneous heart rate assessment. To determine the heart rate, three electrodes were applied to the skin (one each on the left and right collarbone and one on the right hip). An ultrasound probe was placed on the right common carotid and E-tracking software will calculate maximum diastolic diameter (D_max) and minimum diastolic diameter (D_min), elastic modulus (Ep), arterial compliance (AC) and beta stiffness parameters (β). D_max and D_min represent the maximum (systolic) and minimum (diastolic) diameters, respectively. EP, which is associated with the elastic modulus, is used to measure the stress applied to the vessel wall and is affected by pulse pressure. EP is calculated as: \( \frac{P_{\text{max}} - P_{\text{min}}}{[(D_{\text{max}} - D_{\text{min}}) / D_{\text{min}}]} \). β-stiffness, which is independent of pressure, is calculated by the natural logarithm of the systolic and diastolic blood pressure ratio. β stiffness is calculated as \( \beta = \ln\left(\frac{P_{\text{max}}}{P_{\text{min}}}\right)/\left[\frac{(D_{\text{max}} - D_{\text{min}})}{D_{\text{min}}}\right] \) with P_max and P_min are the highest (systolic) and lowest (diastolic) carotid pressures, respectively.
AC is an indicator of vessel compliance that looks at the ratio of the area of change of the artery to the pulse pressure. AC is calculated as: 

$$AC = \pi \left( DD^2 - SD^2\right)/ 4(SBP – DBP)$$

with DD representing diastolic (or minimum) arterial diameter, SD representing systolic (or maximum) arterial diameter, DBP representing lowest (diastolic) carotid pressures, and SBP representing highest (systolic) carotid pressure.

**Regional Arterial Stiffness Measurements**

Central pulse wave velocity (cPWV) is a measure of regional arterial stiffness and is considered the gold standard in the evaluation of arterial stiffness. Measurements were obtained from the right common carotid artery (CCA), obtained using applanation tonometry, to the right femoral artery, obtained from pulsations through a cuff applied around the thigh. Values were obtained from pressure wave forms over a 10-second epoch (SphygmoCor XCEL System; AtCor Medical Pty Ltd, Sydney, Australia). Distances from the CCA measurement site and the femoral artery, the carotid artery to the suprasternal notch, and femoral artery to the top of the thigh cuff were measured in a straight line with a tape measure. The distance from the CCA to the sternal notch was subtracted from the carotid-femoral segment length to account for differences in the pulse-wave propagation (SphygmoCor XCEL System). cPWV between the carotid and femoral arteries is considered a representation of central PWV. cPWV evaluates the speed of the pressure wave traveling through the arteries and is calculated as distance (m)/transit time (s). A stiff aorta will result in a high PWV, while a compliant aorta will have a low PWV value.
Steep Ramp Test Exercise Protocol

After resting measurements were completed, participants performed two bouts of an SRT. The SRT is a maximal anaerobic cycling test to fatigue, and was performed on the cycle ergometer. The SRT requires participants to maintain 50 revolutions per minute (RPM) on the cycle ergometer while 0.5 kg of weight (25 watts) is added every 10 seconds. Adding weight to the ergometer increases the resistance felt by the participant (Rozenberg, Bussmann, Lesaffre, Stam, & Praet, 2015; Stuiver et al., 2017; Bongers, De Vries, Helders, & Takken, 2013). Participants warmed-up for 2 minutes with 0.5 kg of resistance and a cadence of 50 RPM’s. After the 2-minute warm up, resistance was added in accordance with the protocol above.

Participants were asked to provide a maximal effort during the test, with verbal encouragement provided by trained research staff. The test could be stopped by either a trained research assistant if participant pedaling cadence fell below 50 revolutions per minute, or when the participant requested to end the test. The steep ramp test typically lasts 1-4 minutes in young, healthy individuals and depending on individual fitness levels. Participants returned to the supine position for resting measurements within 1-2 minutes following exercise. The participants repeated the above protocol immediately after the 30 minutes vascular measurements were obtained.

Data Collection

Data was collected over a 3-year period. For the purpose of this thesis, I collected data on 16 participants and previous research assistants collected data on 5 participants.
While data on those 5 participants were collected by previous research assistants, all analysis on arterial stiffness measurements and statistical analysis were performed by me.

**Statistical Analyses**

Data are presented as mean ± standard error. The primary outcome variables included 1.) brachial blood pressure, 2.) local carotid arterial stiffness and 3.) systemic arterial stiffness. Descriptive variables and baseline variables were analyzed with independent t-tests for possible racial differences. Paired t-test were used to determine if there was a main effect of time for all blood pressure and stiffness variables.

Due to multiple timepoints, various two-way repeated-measures analysis of variance (ANOVA) were performed to determine which timepoints were most suitable to be used in the final analysis to test for possible race and time interaction effects. The final analysis was determined using a 2x7 (race x time) ANOVA for brachial blood pressure, local carotid arterial stiffness and systemic arterial stiffness variables. Post hoc t-tests were performed when a significant main effect or interaction was detected by the initial ANOVA with SDIK correction for multiple comparisons. A normality check was performed for all variables to ensure data was normally distributed and 3.0 Interquartile Range (IQR) was used to determine extreme outliers. P ≤ 0.05 was used for statistical significance. Statistical analyses were performed using IBM SPSS Statistics 26 software (SPSS, Inc., Chicago, IL, USA).
Power Analysis

Because there is no preliminary data or existing data for aim 1, we used data from a study examining AIx@75 in AA and CA women. Heffernan et al. have shown an average difference of 6.6% with a pooled standard deviation of 14 between AA and CA (Heffernan et al., 2008). We will need to study 72 participants in each group to be able to reject the null hypothesis that the population means of the two groups are equal with probability (power) 0.8. The Type I error probability associated with this test of this null hypothesis is 0.05.

Similarly, for aim 2, we have based our power analyses on a previous study on beta-stiffness index, a marker for local carotid arterial stiffness, in AA and CA men. Heffernan et al., have shown an average 0.6 difference a pooled standard deviation of 0.7, between AA and CA men (Heffernan et al., 2009). Power analysis yielded an estimated total sample size of 48 with 24 participants per group that would provide at least 80% power at the 5% level of significance. Power calculations were performed using PS-Power and Sample Size Calculations software.

Confidentiality and HIPAA Compliance

All study data was collected directly from study participants and health and other sensitive information was kept confidential in accordance with the Health Insurance Portability and Accountability Act (HIPAA). To maintain confidentiality, all participant data was de-identified and referenced by a study identification number. Participant data was stored on a password-protected computer and in locked filing cabinets in a secure room.
Participation in this study was voluntary and all enrolled participants were required to sign an informed consent form. This study was approved by the University of Massachusetts, Boston Institutional Review Board (IRB) and was conducted in accordance with the Declaration of Helsinki.
CHAPTER 3
RESULTS

Participant Characteristics and SRT Exercise Performance

Twenty-one women were recruited for this study and 20 participants were included in the final analysis (8 AA and 12 CA). While the blood pressure exclusion criteria was >140/90, we excluded one participant due to higher diastolic blood pressure, which resulted in differences in blood pressure between groups. We decided to exclude this participant from the final analysis so our results could be based on healthy, normotensive women based on current blood pressure guidelines. Participant characteristics, hemodynamics and SRT exercise performance are presented in Table 1 and Table 2. There were no significant differences in age, height, weight, or BMI between CA and AA at baseline, as previously described in methods. There were no differences in resting brachial systolic blood pressure (BraSBP), brachial diastolic blood pressure (BraDBP) or brachial mean arterial pressure (BraMAP) between groups at baseline. Exercise time on cycle ergometer, $W_{\text{max}}$, and estimated absolute VO$_2$ peak was not different between CA and AA for both bouts (Table 2). AA had lower estimated relative VO$_2$ peak (mL/kg/min) following both bouts of exercise compared to CA.
There was no difference in exercise time on cycle ergometer between the first bout and second bout for either AA or CA. Hemodynamic variables in AA and CA at rest and following exercise are presented in Figures 2-11, and the values for all participants regardless of race are presented in Table 3.

### Table 1. Participant Characteristics

<table>
<thead>
<tr>
<th></th>
<th>CA (n=12)</th>
<th>AA (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>22 ± 0.9</td>
<td>22 ± 0.8</td>
</tr>
<tr>
<td>Brachial SBP (mmHg)</td>
<td>113 ± 1</td>
<td>116 ± 3</td>
</tr>
<tr>
<td>Brachial DBP (mmHg)</td>
<td>67 ± 2</td>
<td>72 ± 1</td>
</tr>
<tr>
<td>Brachial MAP (mmHg)</td>
<td>86 ± 2</td>
<td>82 ± 1</td>
</tr>
<tr>
<td>HRrest (BPM)</td>
<td>64 ± 1</td>
<td>64 ± 2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>63 ± 3</td>
<td>69 ± 4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165 ± 2</td>
<td>167 ± 2</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23 ± 1</td>
<td>25 ± 1</td>
</tr>
<tr>
<td>MVPA volume (min/week)</td>
<td>187 ± 45</td>
<td>126 ± 36</td>
</tr>
</tbody>
</table>

Values are mean ± SE; AA, African American; CA, Caucasian American; 1st, first SRT; 2nd, second SRT; n, No. of participants; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate; BMI, body mass index; MVPA, moderate-vigorous physical activity; VO₂ Peak, peak oxygen consumption. # P≤ 0.05, significant baseline differences between AA and CA. No significant differences were found in baseline characteristics.
Table 2. Exercise Performance

<table>
<thead>
<tr>
<th></th>
<th>CA (n=12)</th>
<th>AA (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time_1st (mins)</td>
<td>106 ± 15</td>
<td>99 ± 12</td>
</tr>
<tr>
<td>Resistance_1st (kg)</td>
<td>5.8 ± 0.7</td>
<td>5.6 ± 0.6</td>
</tr>
<tr>
<td>Wmax_1st (Watt)</td>
<td>278 ± 10</td>
<td>290 ± 10</td>
</tr>
<tr>
<td>Absolute VO\textsubscript{2} Peak_1st (L/min)</td>
<td>2.2 ± 0.1</td>
<td>2.3 ± 0.1</td>
</tr>
<tr>
<td>Relative VO\textsubscript{2}_1st (mL/kg/min)</td>
<td>37.2 ± 1.4#</td>
<td>32.4 6± 1.2#</td>
</tr>
<tr>
<td>Time_2nd (mins)</td>
<td>107 ± 17</td>
<td>100 ± 11</td>
</tr>
<tr>
<td>Resistance_2nd (kg)</td>
<td>5.9 ± 0.9</td>
<td>5.7 ± 0.5</td>
</tr>
<tr>
<td>Wmax_2nd (Watt)</td>
<td>284 ± 9</td>
<td>294 ± 13</td>
</tr>
<tr>
<td>Absolute VO\textsubscript{2} Peak_2nd (L/min)</td>
<td>2.3 ± 0.1</td>
<td>2.3 ± 0.1</td>
</tr>
<tr>
<td>Relative VO\textsubscript{2}_2nd (mL/kg/min)</td>
<td>37.5 ±1.3#</td>
<td>33.08 ± 1.1#</td>
</tr>
</tbody>
</table>

Values are mean ± SE; CA, Caucasian American; AA, African American; 1st, first SRT; 2nd, second SRT; n, No. of participants; SRT, steep ramp test; Time, time on cycle ergometer; Wmax, maximal work; VO\textsubscript{2} Peak, peak oxygen consumption. # P≤ 0.05, significant baseline differences between AA and CA.
Table 3. Blood pressure and local, systemic and regional arterial stiffness for all participants

<table>
<thead>
<tr>
<th></th>
<th>Resting</th>
<th>P5 post-</th>
<th>P15 post-</th>
<th>P30 post-</th>
<th>P5 post-</th>
<th>P15 post-</th>
<th>P30 post-</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>SRT1</td>
<td>SRT1</td>
<td>SRT1</td>
<td>SRT2</td>
<td>SRT2</td>
<td>SRT2</td>
</tr>
<tr>
<td>BraSBP*†</td>
<td>114 ± 1</td>
<td>128 ± 2</td>
<td>113 ± 1</td>
<td>113 ± 1</td>
<td>130 ± 2</td>
<td>118 ± 2</td>
<td>114 ± 1</td>
</tr>
<tr>
<td>BraDBP*</td>
<td>70 ± 1</td>
<td>68 ± 1</td>
<td>66 ± 1</td>
<td>69 ± 1</td>
<td>67 ± 1</td>
<td>67 ± 1</td>
<td>68 ± 1</td>
</tr>
<tr>
<td>BraMAP*†</td>
<td>85 ± 1</td>
<td>87 ± 1</td>
<td>81 ± 1</td>
<td>83 ± 1</td>
<td>88 ± 1</td>
<td>84 ± 1</td>
<td>84 ± 1</td>
</tr>
<tr>
<td>D_min*</td>
<td>6.4 ± 0.2</td>
<td>6.3 ± 0.2</td>
<td>6.3 ± 0.1</td>
<td>6.5 ± 0.2</td>
<td>6.1 ± 0.1</td>
<td>6.3 ± 0.1</td>
<td>6.6 ± 0.1</td>
</tr>
<tr>
<td>AC*†</td>
<td>1.2 ± 0.0</td>
<td>0.9 ± 0.1</td>
<td>1.00 ± 0.1</td>
<td>1.1 ± 0.0</td>
<td>0.8 ± 0.0</td>
<td>1.0 ± 0.1</td>
<td>1.0 ± 0.0</td>
</tr>
<tr>
<td>β- Stiffness*</td>
<td>5.0 ± 0.4</td>
<td>6.2 ± 0.3</td>
<td>6.0 ± 0.3</td>
<td>5.4 ± 0.3</td>
<td>6.2 ± 0.4</td>
<td>6.0 ± 0.4</td>
<td>6.2 ± 0.3</td>
</tr>
<tr>
<td>EP*</td>
<td>61.2 ± 4.3</td>
<td>78.2 ± 3.9</td>
<td>70.8 ± 3.5</td>
<td>64.1 ± 3.0</td>
<td>78.5 ± 5.1</td>
<td>69.9 ± 3.7</td>
<td>73.6 ± 4.2</td>
</tr>
<tr>
<td>PWV</td>
<td>5.0 ± 0.1</td>
<td>4.9 ± 0.1</td>
<td>5.2 ± 0.2</td>
<td>5.0 ± 0.2</td>
<td>4.9 ± 0.2</td>
<td>5.3 ± 0.2</td>
<td>5.1 ± 0.2</td>
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<tr>
<td>Alx*</td>
<td>8.3 ± 2.5</td>
<td>6.8 ± 2.1</td>
<td>4.8 ± 2.7</td>
<td>1.6 ± 2.5</td>
<td>6.7 ± 2.4</td>
<td>3.2 ± 2.6</td>
<td>-0.73 ± 3.4</td>
</tr>
<tr>
<td>Alx75*</td>
<td>3.1 ± 2.6</td>
<td>11.4 ± 2.0</td>
<td>6.2 ± 2.0</td>
<td>0.7 ± 2.7</td>
<td>14.0 ± 2.3</td>
<td>7.9 ± 2.7</td>
<td>1.8 ± 3.2</td>
</tr>
</tbody>
</table>

Values are mean ± SE. BraSBP, brachial diastolic blood pressure; BraMAP, brachial mean arterial pressure; D_min, diastolic diameter; AC, arterial compliance; β-stiffness, beta-stiffness index; EP, elastic modulus; PWV, pulse wave velocity; Alx, augmentation index; Alx75, augmentation index at 75. *Significant main effect of time; P ≤ 0.05. † Significant race x time interaction; P ≤ 0.05.
**Figure 3.** Brachial SBP at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. Brachial SBP at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. Brachial SBP at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout. * Significant main effect of time (P ≤ 0.05). † Significant race by time interaction (P ≤ 0.05).
Figure 4. Brachial DBP at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. Brachial DBP at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout. * Significant main effect of time (P ≤ 0.05)
Figure 5. Brachial MAP at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. Brachial MAP at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout. * Significant main effect of time (P ≤ 0.05). † Significant race by time interaction (P ≤ 0.05)
Figure 6. AC at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. AC at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout. * Significant main effect of time (P ≤ 0.05). † Significant race by time interaction (P ≤ 0.05)
Figure 7. Beta-stiffness index at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. Beta-stiffness at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout. * Significant main effect of time ($P \leq 0.05$).
Figure 8. EP at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. EP at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout.
* Significant main effect of time (P ≤ 0.05).
Figure 9. D_min at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. D_min at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout.

* Significant main effect of time ($P \leq 0.05$).
Figure 10. Aix@75 at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. Aix@75 at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout. * Significant main effect of time (P ≤ 0.05).
Figure 11. cPWV at rest and during recovery from repeated bouts of maximal anaerobic exercise in AA and CA. cPWV at rest and 5-, 15-, and 30 minutes following an SRT bout and 5-, 15-, and 30 minutes following a second SRT bout.

Brachial Blood Pressure

Brachial Systolic Blood Pressure. There was a significant race by time interaction for BraSBP (race by time interaction p = 0.026, Figure 3). BraSBP was significantly higher in AA compared to CA at P30 post-SRT2 (p = 0.053). Compared to resting, both CA and AA experience significant increases in BraSBP at P5 post-SRT1 and 5 post-SRT2 (P ≤ 0.05). For both CA (p = 0.000) and AA (p = 0.003) BraSBP was significantly lower at P15 post-SRT1 and P30 post-SRT1 compared to P5 post-SRT1 (CA, p = 0.000; AA, p = 0.003). In CA,
BraSBP at P15 post-SRT2 (p = 0.009) and P30 post-SRT2 (p = 0.000) was significantly lower than P5 post-SRT. BraSBP at P15 post-SRT1 and P30 post-SRT1 were significantly lower than P5 post-SRT2 (p = 0.000), and P15 post-SRT2 (p = 0.006) and P30 post-SRT2 (0.000) were lower than P5 post-SRT2 in CA.

There was a significant main effect of time for BraSBP (time effect p = 0.000, Table 3). Compared to resting, BraSBP was significantly higher at P5 post-SRT1 and P5 post-SRT2, regardless of race (P ≤ 0.05). BraSBP at P15 post-SRT2 and P30 post-SRT2 was significantly lower than P5 post-SRT1 (P ≤ 0.05). BraSBP at P15 post-SRT1 and P30 post-SRT1 were significantly lower than P5 post-SRT2 and, P15 post-SRT2 and P30 post-SRT2 were lower than P5 post-SRT2 (P ≤ 0.05).

**Brachial Diastolic Blood Pressure.** There was a significant main effect of time for BraDBP (time effect p = 0.023, Table 3).

**Brachial Mean Arterial Pressure.** There was a significant time by race interaction for BraMAP (time by race p = 0.015, Figure 5). BraMAP was significantly higher in AA at resting (p = 0.054) and P30 post-SRT2 compared to CA (p = 0.044). In CA, BraMAP at P15 post-SRT2 (p = 0.032) and P30 post-SRT2 (p = 0.010) was significantly lower than P5 post-SRT1. BraMAP at P15 post-SRT1(p = 0.001) and P30 post-SRT1(p = 0.002) were significantly lower than P5 post-SRT2, and P15 post-SRT2 (p = 0.010) and P30 post-SRT2 (p = 0.001) were lower than P5 post-SRT2 in CA (P ≤ 0.05).

There was a main effect of time for BraMAP (time effect p = 0.000, Table 3). Compared to resting, BraMAP was significantly higher at P5 post-SRT2, regardless of race (p = 0.012).
BraMAP was significantly lower at P15 post-SRT1 (p = 0.000), P30 post-SRT1 (p = 0.006) and P30 post-SRT2 compared to P5 post-SRT1 (p = 0.035). BraMAP at P5 post-SRT2 was significantly higher than P15 post-SRT1 (p = 0.003) and P30 post-SRT (p = 0.017), and P30 post-SRT2 was significantly lower than P5 post-SRT2 (p = 0.035).

**Local Carotid Arterial Stiffness**

**Carotid Arterial Compliance (AC).** There was a significant race by time interaction for carotid AC (time by race p = 0.040, Figure 6). AC was significantly higher in CA compared to AA at P15 post-SRT1 (p = 0.027). AC decreased significantly in CA from resting to P5 post-SRT1 (p = 0.020), resting to P5 post-SRT2 (p = 0.000), and P30 post-SRT1 to P5 post-SRT2 (p = 0.000). AC was lower in CA at P5 post-SRT1 compared to P30 post-SRT1 (p = 0.000) and P30 post-SRT2 (p = 0.039, and lower at P5 post-SRT2 compared to P15 post-SRT2 (p = 0.000) and P30 post-SRT2 (p = 0.000). In AA, AC was significantly decreased from resting to P5 post-SRT2 (p ≤ 0.040).

There was a significant main effect of time for carotid AC (time effect p = 0.000, Table 3). Compared to resting, AC was significantly decreased at P5 post-SRT1 (p = 0.000) and P5 post-SRT2 (p = 0.000), regardless of race. AC at P5 post-SRT1 is significantly lower than P30 post-SRT1 (p = 0.000), and P5 post-SRT2 is significantly lower than P30 post-SRT2 (p = 0.000). AC was significantly lower at P5 post-SRT1 and P15 post-SRT1 compared to P30 post-SRT1 (p = 0.000 and p = 0.002, respectively). AC was significantly lower at P5 post-SRT2 compared to P30 post-SRT1 (p = 0.000) and compared to P15 post-SRT1 (p = 0.002).
AC was significantly lower at P5 post-SRT2 compared to P15 post-SRT2 (p = 0.001) and P30 post-SRT2 (p = 0.000).

**β-stiffness (β).** There was a significant main effect of time for β (time effect p = 0.001, Table 3). Compared to resting, β was significantly increased at P5 post-SRT1 (p = 0.021) and decreased significantly from P5 post-SRT1 to P30 post-SRT1, regardless of race (p = 0.034). β was significantly increased at P15 post-SRT1 compared to P30 post-SRT1 (p = 0.012). β was significantly increased at P30 post-SRT2 compared to P30 post-SRT1 (p = 0.052).

**Elastic Modulus (EP).** There was a significant main effect of time for EP (time effect P = 0.000, Table 3). Compared to resting, EP was significantly increased at P5 post-SRT1 and significantly decreased at P30 post-SRT1, regardless of race (p = 0.004). EP is significantly increased at P5 post-SRT2 compared to resting (p = 0.008).

**Diastolic Diameter (D_Min).** There was a significant main effect of time for D_Min (time effect (p = 0.014, Table 3). D-min was significantly lower at P5 post-SRT2 compared to P30 post-SRT2, regardless of race (p = 0.045).

**Systemic Arterial Stiffness**

**Augmentation Index (AIx).** There was a significant main effect of time for AIx (time effect p = 0.001, Table 3). Compared to resting, AIx was significantly lower at P30 post-SRT1, regardless of race (p = 0.051).

**Augmentation Index at 75 (AIx@75).** There was a significant main effect of time for AIx@75 (time effect p = 0.000, Table 3). Compared to resting, AIx@75 was significantly
increased at P5 post-SRT1 (p = 0.001 and P5 post-SRT2 (p = 0.009), regardless of race. AIX is significantly lower at P30 post-SRT1 compared to P5 post SRT1 (p = 0.009), and P30 post-SRT2 compared to P5 post-SRT2 (p = 0.014). At P5 post-SRT2, AIX@75 was significantly higher compared to P30 post-SRT1 (p = 0.001). AIX@75 was significantly higher at P5 post-SRT1 compared to P30 post-SRT2 (p = 0.047), and was significantly higher at P5 post-SRT2 when compared to P15 post-SRT1 (p = 0.037).

Regional Arterial Stiffness

Pulse Wave Velocity (cPWV). There was no significant main effect of time, main effect of race, or interaction for cPWV (Table 3).
CHAPTER 4

DISCUSSION

The major findings of this study are as follows. First, CA women experienced disparate changes in local carotid arterial compliance and brachial blood pressure during recovery from repeated bouts of maximal anaerobic exercise compared to AA women. Second, no racial disparities were observed in systematic or regional arterial stiffness following acute bouts of maximal anaerobic exercise, suggesting the racial differences are localized in the carotid artery. Third, maximal anaerobic exercise elicits overall changes in local carotid and systematic arterial stiffness, but not in central regional arterial stiffness for all participants.

Local Carotid Arterial Compliance and Stiffness

To our knowledge, this is the first study examining the effect of consecutive maximal anaerobic cycling exercise on arterial compliance, which is inversely related to arterial stiffness, in young, healthy AA and CA women. The novel finding from our study demonstrates that AA women experience less variation in local carotid arterial compliance during recovery from consecutive bouts of maximal anaerobic exercise compared to CA. CA women experienced a reduction in arterial compliance immediately following both bouts of exercise and recovered in 30 minutes while arterial compliance was maintained in AA during
recovery, except an immediate increase following the second bout of exercise. In addition, at 15 minutes during recovery from the first SRT, carotid AC was lower in AA compared to CA. These findings build on previous studies examining recovery of carotid stiffness following acute aerobic exercise (Yan et al., 2017, Schroeder et al., 2019). Carotid arterial stiffness was examined in AA and CA men and women during 90 minutes of recovery from 45 minutes of a single bout of submaximal aerobic exercise (Yan et al., 2017). The AA cohort had higher local carotid arterial beta-stiffness and EP overall, which are inversely related to arterial compliance, but there were no differences in their recovery patterns during 90 following exercise (Yan et al., 2017). It is possible exercise intensity and recovery timepoints may explain the differences in our findings.

The observations from the current study show CA and AA experience a differential response to maximal anaerobic exercise during the early stages of recovery from a single SRT. However, the previous report only examined arterial stiffness at 30, 60 and 90 minutes following acute aerobic exercise (Yan et al., 2017). These findings may suggest the disparate response to maximal anaerobic exercise occurs during early recovery phase, within 30 minutes following acute anaerobic exercise between AA and CA women. The immediate response to maximal anaerobic exercise in CA women in the current study is similar to findings from consecutive bouts of supramaximal exercise in Caucasians (Rossow et al, 2010). Rossow et al. found local arterial carotid compliance decreased immediately following supramaximal anaerobic exercise and returned to baseline after 25 minutes of recovery in young, healthy men and women (2010). These findings suggest AA women
experience a blunted response to maximal anaerobic exercise following the first exercise bout, compared to CA women.

Although the present study didn’t examine the mechanism for the differential changes in arterial compliance between AA and CA, several possible contributors were explored. Previous studies have suggested changes in compliance following exercise are inversely related to resting arterial compliance (Rossow et al., 2010). Our findings suggest no differences in resting compliance, therefore the racial differences in local carotid arterial compliance during recovery is likely not due to resting values (Figure 7). Another potential contributor for the blunted vascular response in AA women is blood pressure during recovery from exercise. Arterial stiffness increases transiently as BP increases because of the nonlinear distensibility characteristics of the artery (Arnett et al., 2001). Our findings of higher brachial SBP and brachial MAP in AA 30 minutes following the second SRT, agree with previous findings suggesting higher carotid SBP during recovery from maximal sprint exercise (Rossow et al., 2010) and higher MAP following moderate-intensity cycling (Kingwell et al., 1997) is related to lower arterial compliance.

The present study found racial disparities in brachial blood pressure, with higher brachial MAP in AA following the second bout of maximal anaerobic exercise compared to CA women. Yan et al. found a greater change in brachial SBP 30 minutes following a single bout of maximal aerobic cycling exercise compared to 15 minutes, but no racial differences between AA and CA men and women (2014). The discrepancies between our study and the previous study may be due to differences in intensities of exercise stimulus. The higher
exercise intensities associated with anaerobic exercise may elicit greater release of stress hormone and disturbance to the sympathoadrenal system, compared to aerobic exercise (Hackney, AC., 2006). We observed changes in brachial blood pressure during recovery from consecutive bouts of maximal anaerobic exercise in CA women but not in AA, suggesting AA women experienced less variation in blood pressure changes during exercise recovery compared to CA women.

The blunted BP and carotid arterial stiffness responses following anaerobic exercise in AA women is in contrast with previous reports of heightened hemodynamic responses to physiological and psychological stimulus in AA compared to CA. AA men have been shown to have reduced vasodilation and increased vasoconstriction in response to an adrenergic stimulus (Lemogour et al., 2004; Lang et al., 1999). AA women have been shown to experience an exaggerated blood pressure response to physical stimulus (Bond et al., 1999), and Yan et al. (2016) observed higher DBP in AA during 90 minutes of recovery from acute aerobic exercise than CA. Maximal anaerobic exercise may exert blunted hemodynamic effects in AA, while aerobic exercise might elicit heightened responses. While post-exercise hypotension was not assessed in the current study, our results show brachial SBP in both CA and AA women was decreased at 15 and 30 minutes following a single bout of exercise when compared to 5 minutes following exercise. However, AA women didn’t experience any changes in brachial SBP following the second bout of exercise, suggesting AA may not benefit from reduced blood pressure in response to repeated bouts of maximal anaerobic exercise. Future studies are warranted to compare BP and arterial stiffness responses both during and following different modes and intensities of exercise in AA. This may hold
clinical significance to develop targeted exercise prescription for individuals who have concerns for exercise-associated hypo/hypertension or arterial complications.

Surprisingly, there was no racial differences in local carotid arterial stiffness between AA and CA women following acute maximal anaerobic cycling exercise. While the exact reason for the disparate findings is not clear, it would appear that changes in BP may explain the differential responses between AA and CA women for pressure dependent AC, but not for pressure independent $\beta$. Exercise has been shown to induce transient stiffening of the carotid artery immediately following one bout of supramaximal anaerobic exercise (Babcock et al., 2015) and recover at 25 minutes (Rossow et al., 2010). This is consistent with our findings of increased arterial stiffness in all participants immediately following exercise and recovering at 30 minutes. We also observed the repeated bout of SRT further increased arterial stiffness at 30 minutes following the second bout of exercise as compared to 30 minutes following the first bout. These findings suggest local carotid arterial stiffness may be sustained following consecutive SRTs. The previous research by Rossow et al. observed a cumulative effect at 5 minutes following repeated bout of supramaximal cycling exercise and returned to baseline at 25 minutes, despite no significant changes following the first bout of 30-second supramaximal sprint exercise (2010). Our study adds to the literature by showing repeated SRTs elicit a sustained response, with stiffness increasing following both bouts of exercise. When examining the denominator of beta-stiffness, the observed cumulative effect in local carotid arterial stiffness may be due to changes in diastolic diameter during recovery. Carotid artery diameter during diastole, a measure of carotid artery vasodilation, was higher 30 minutes following the second SRT when compared to immediately following the first
SRT. This change in diastolic carotid artery diameter following the second bout of exercise may be due to elevated sympathetic nervous system activity and may have contributed to the sustained stiffness observed in $\beta$ following the second SRT.

**Systemic and Regional Arterial Stiffness**

To our knowledge, this study is the first to examine the effects of repeated bouts of maximal anaerobic exercise on aortic wave reflection, a marker for systemic arterial stiffness, during both the immediate recovery and up to 30 minutes after exercise. Contrary to our hypothesis, the current study found no racial differences in systemic arterial stiffness between AA and CA women at rest or following repeated bouts of maximal anaerobic exercise. While previous findings show AA men have higher resting aortic wave reflection compared to their CA counterparts (Heffernan et al., 2008), Yan et al. found no differences in systemic arterial stiffness between AA and CA after a single bout of maximal aerobic exercise (2014). Adding to these findings, the current study suggests AA and CA women exhibit a similar systemic arterial response to repeated bouts of maximal anaerobic exercise.

Overall, aerobic exercise has consistently been shown to decrease aortic wave reflection during recovery (Heffernan et al., 2007b; Yan et al., 2014). Our study suggests that maximal anaerobic exercise elicits similar systemic arterial response as aerobic exercise in all participants. When controlled for HR, aortic wave reflection, measured by AIx@75, was increased immediately following both bouts of exercise. These findings are in agreement with previous findings of increased wave reflection 5 minutes following three consecutive bouts of supramaximal anaerobic exercise (Kingsley et al., 2017). Heffernan et al. found that
HR corrected aortic wave reflection was increased 10 minutes following maximal aerobic exercise and stayed elevated at 30 minutes of recovery (2007b). Our findings show AIx@75 has a similar immediate increase following maximal anaerobic exercise as was observed following maximal aerobic exercise and returned to baseline at 30 minutes following exercise. These findings are consistent across both bouts of exercise, suggesting a second bout of exercise elicits a similar response as the first.

The current study found no racial differences in regional arterial stiffness, measured by cPWV, at rest or following repeated maximal anaerobic exercise in AA and CA women. This is in contrast with previous findings suggesting AA men have higher cPWV at rest compared to CA men (Heffernan et al., 2007) and cPWV responds differently between AA and CA men and women following maximal aerobic exercise (Yan et al., 2014). The discrepancies between previous reports and our study may be due to the mode and volume of exercise. Repeated bouts of maximal anaerobic exercise may not induce transient stiffening of the central arteries, regardless of race. This is supported by previous studies reporting no changes in central arterial stiffness immediately following consecutive bouts of supramaximal exercise (Kingsly et al., 2017).

Taken together, the racial differences in arterial distensibility between AA and CA women in response to maximal anaerobic exercise were not consistently exhibited throughout different parts of arteries. Our observations suggest maximal anaerobic exercise only elicit differential localized changes in the predominantly elastic carotid artery between AA and CA women, and we didn’t observe any racial differences in systemic or segmental
arterial stiffness following repeated bouts of SRT. This is consistent with a previous study reporting racial differences between CA and AA in local carotid arterial stiffness, but not in cPWV following an acute bout of submaximal exercise (Yan et al., 2017). Localized carotid artery distensibility demonstrates a dose-response association with calcification of the thoracic aorta and is one of the earliest detectable manifestations of adverse change within the vessel wall (Blaha et al., 2009). Due to the limited research available, it’s not feasible to establish clinically significant standards for arterial stiffness responses following exercise. We didn’t observe changes in local carotid arterial stiffness following maximal anaerobic exercise in AA, therefore it may be suggested that this type of exercise is favorable for AA women. It is unclear, however, if this acute response results in favorable chronic adaptations. Heffernan et al. observed favorable changes in central hemodynamics in both AA and CA men following a 6-week resistance training program, however, peripheral arterial stiffness was increased in AA but not CA (2009). More research is needed to investigate the transient changes in local carotid distensibility following exercise and potential translation for long-term health outcomes. Further research is also warranted to examine possible mechanisms responsible for the racial differences and differential response to exercise between local, systematic and central arterial stiffness.
Strengths and Limitations

The strength of our study is due to the rigorous measures used to obtain local, systemic and regional arterial stiffness. Ultrasound and applanation tonometry were used to obtain a comprehensive review of racial differences during recovery from maximal anaerobic exercise. All research assistants were trained under the graduate research assistant, and the graduate research assistant obtained all measurements to ensure consistency and reliability in data collection. Additionally, all participants had similar baseline hemodynamics, and body weight.

Our study was not without limitations. Physical activity levels were determined based on self-reported questionnaires, and the type of exercise (aerobic or resistance) was not reported. While cardiorespiratory fitness differed between AA and CA for estimated relative VO$_2$peak, the equation utilized to determine VO$_2$peak from the SRT was validated in clinical populations, including adults with cancer and with diabetes (Rozenberg et al., 2015; De Backer et al., 2007), as well as in children and adolescents (Bongers et al., 2013), but not in young and healthy individuals (Werkman, Bongers, & Wittink, 2019). Therefore, it may not be generalizable to young, healthy populations. We did not control for socioeconomic status or education level, however, the majority of the participants were recruited from UMB student population, therefore they may share similar education status. UMB is primarily a commuter school in an urban area with a diverse population and a reported 54% of the student body being persons of color.
Our small sample size may have limited our ability to detect potential racial differences. While it was our intention to recruit more participants, with an equal number of participants in each group, data collection was interrupted by COVID-19 pandemic which led to the closure of the UMB campus. However, similar studies examining the effect of exercise on arterial stiffness have include similar sample sizes between 14-24 participants (Rossow et al., 2010; Heffernan et al, 2007). Lastly, all participants were young, healthy women, therefore our results can’t be generalized to men, older or younger adults or clinical populations.

Conclusion

Local carotid arterial compliance and brachial blood pressure responds differently in AA women compared to CA women following repeated bouts of maximal anaerobic exercise. These differences exist despite similar resting values, suggesting maximal anaerobic exercise may be used to elicit cardiovascular differences that are not apparent at rest. Stiffening of the large arteries is independently associated with the development of CVD. Therefore, understanding racial differences in arterial stiffness in response to acute maximal anaerobic exercise may add to the current knowledge regarding the disparity in CVD and hypertension prevalence between AA and CA women. No racial differences were observed in systemic or central arterial stiffness following repeated bouts of maximal anaerobic exercise. While central arterial stiffness is considered the gold standard for evaluating arterial stiffness, it may not directly reflect the degree of arteriosclerosis.
The distensibility of the carotid artery reflects the level of arteriosclerosis, and future research is needed to investigate the relationship between transient changes following acute exercise and long-term health implications. Understanding racial differences and the overall cardiovascular response to maximal anaerobic exercise may help guide exercise prescription and training programs.
APPENDIX

A. DEMOGRAPHIC QUESTIONNAIRE

Demographic Questionnaire

What is your race/ethnicity based on BOTH of your parents? Please mark the one box that describes the race/ethnicity category with which you primarily identify:

☐ (A) Asian or Pacific Islander: Persons having origins in any of the peoples of the Far East, Southeast Asia, the Indian subcontinent, or the Pacific Islands. This area includes, for example, China, Japan, Korea, the Philippine Islands and Samoa.

☐ (B) African American (not of Hispanic origin): Person having origins in any of the black ethnic groups.

☐ (H) Hispanic: Persons having origins in any of the Mexican, Puerto Rican, Cuban, Central or South American or other Spanish Cultures, regardless of ethnicity.

☐ (I) Native American or Alaskan Native: Persons having origins in any of the original peoples of North America, and who maintain cultural identification through tribal affiliation or community recognition.

☐ (W) Caucasian (not of Hispanic origin): Persons having origins in any of the original peoples of Europe, North Africa or the Middle East.
B. HEALTH HISTORY QUESTIONNAIRE

SELF-ADMINISTERED PRE-EXERCISE MEDICAL HISTORY FORM

All information given is confidential. It will enable us to better understand you and your health and fitness habits. To ensure that you do not meet any of the exclusion criteria, you must answer all the questions included here.

SUBJECT ID._______________________________ Date:______________
Birth date:_______________ Male/Female _____ Age: ______
The first day of your last menstrual period _______________________

Medical History

1. Do you have any allergic reaction to any medication?    ____    ____
2. If you are a female, are you currently breast-feeding?  ____    ____
3. If you are a female, are you currently pregnant?        ____    ____
4. Do you know your blood pressure?                       ____    ____
   If yes, what is your blood pressure? ________/__________

Medicines/Drugs you are now taking (please list dosages):________________________

Family History

Please indicate the number of blood relatives (mother, father, grandparents, siblings who have or have had the following):

Heart attack or stroke before age 50         ______
Heart attack or stroke after age 50          ______
Congenital heart disease ______
Heart operations ______
High blood pressure ______
Diabetes ______
Substantially overweight ______
High cholesterol levels ______

**Health Inventory**

**Smoking Habits**
Do you smoke cigarettes at present? Yes ______ No _____
If yes, how many per day? <1/2 pack ______ 1/2 to 1 pack
1 - 2 packs ______ >2 packs _____
Did you smoke cigarettes in the past and quit permanently? Yes _____ No _____
How many years has it been since you quit? ______
How many packs per day were you smoking before you quit? ______
How many years did you smoke before you quit? ______

**Exercise History**
1. Are you physically active? ______ If yes, for how many years? ______
   If yes, what type(s) of activity do you perform regularly?
   ______________________________________________________________
2. How often and for how long do you perform physical activity? ______ times per week
   _____ minutes
3. How intense is your exercise? (Please circle one)
   Low    Moderate    High
C. SRT DATA SHEET

SRT Data Collection:

<table>
<thead>
<tr>
<th>Subject ID:</th>
<th>Date:</th>
<th>Time:</th>
</tr>
</thead>
</table>

Age: ______ years  Weight: ______ lbs ______ kg  Height: ______ in ______ cm

Resting IMT Measurement:  YES  NO

Initial blood pressure:

Blood Pressure Measurements:

<table>
<thead>
<tr>
<th>Time Point</th>
<th>Systolic(mmHg)</th>
<th>Diastolic (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 min post</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 min post</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 min post</td>
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<td></td>
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<tr>
<td>SECOND SRT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 min post</td>
<td></td>
<td></td>
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<tr>
<td>15 min post</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 min post</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Time on bike for first SRT: ________ mins ______ secs

Amount of kg during the last completed 10 seconds _____ kg + weight of basket (.5kg) =
Time on bike for second SRT: _______ mins _____ secs

Amount of kg during the last completed 10 seconds _____ kg + weight of basket (.5kg)=

Distances for SphygmoCor:
   Carotid to Sternal Notch ____ mm
   Sternal Notch to Cuff ____ mm
   Femoral to Cuff ____ mm
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