THE EFFECTS OF AN ACUTE BOUT OF TRADITIONAL VERSUS CIRCUIT RESISTANCE EXERCISE ON ARTERIAL STIFFNESS AND BLOOD PRESSURE

BY

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THESIS

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ABSTRACT

Arterial stiffness increases cardiovascular disease risk for stroke, myocardial infarction, and mortality. The aim of this study was to examine the influence of resistance exercise intensity on arterial stiffness and blood pressure. Two resistance exercise conditions, low intensity (CREP; 40%1RM) and high intensity (TREP; 80%1RM), were compared. In a longitudinal, cross-over study design, thirty-two subjects (n=32, 17 male, and 15 female, mean age= 24.6 +/-4years) completed both conditions on separate days. Pulse wave velocity (PWV) was used as a measure of central and peripheral arterial stiffness at baseline, at 15 minutes post, and at 30 minutes post exercise. Central PWV increased in the CREP mode and returned toward baseline at 30 minutes post-exercise ($p<0.05$). Central PWV did not change in the TREP mode. Peripheral PWV increased in the TREP mode and remained elevated at 30 minutes post-exercise ($p<0.05$). Peripheral PWV did not change in the CREP mode. Mean blood pressure (MAP) ($p<.05$) decreased following CREP but did not change following TREP ($p<.05$). Neither CREP not TREP had any effect on indices of heart rate variability. These findings suggest that resistance exercise intensity has differential effects on blood pressure and arterial stiffness; and that acute resistance exercise influences central and peripheral arterial stiffness.

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CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

Arterial stiffness is known to be a risk factor for cardiovascular disease, morbidity, and mortality (Zieman et al 2005). There are a number of different factors that contribute to increases in arterial stiffness including hypertension, ageing, stress, smoking, and high blood cholesterol levels. Hemodynamic factors in addition to hormones, glucose, and sodium concentration all influence arterial alterations. Arterial wall stress may induce vascular damage due to turbulent flow and increased luminal pressure, in both the central aortic and conduit arteries. Increases in blood flow such as those seen during resistance exercise elevate stress against the vascular wall. Chronic hemodynamic stress like this can reduce elastin production and stimulate excessive arterial collagen production which leads to stiffening of vascular tissues (Johnson et al. 2001).

Aerobic vs. Resistance Exercise

Acute and chronic resistance exercise has been shown to increase central arterial stiffness whereas reductions occur with aerobic exercise (Heffernan et al. 2006; Sesso et al. 2000). However, past research suggests that changes in arterial function following acute resistance exercise are transient and dissipate 30 minutes after exercise (Devan et al. 2005). This would imply that the increase in arterial stiffness following resistance exercise is only temporary and not chronic. However, there is research to suggests that resistance trained individuals have a higher degree of arterial stiffness, both central and peripheral, than their sedentary counterparts

(Bertovic et al. 1999). This would suggests that resistance trained individuals are to some degree less healthy than sedentary individuals which is counterintuitive.

Studies examining resistance exercise with respect to arterial stiffness are limited. In a meta-analysis of 8 randomized, controlled trials in 193 subjects, resistance exercise intensity was associated with changes in arterial stiffness (Miyachi 2012). High intensity resistance exercise was shown to increase (11.6%) arterial stiffness significantly in 87 young adults. However, moderate intensity resistance exercise did not significantly alter arterial stiffness in 106 subjects. No changes in arterial stiffness were found in middle aged subjects. It is worth noting that these outcomes were not influenced by gender. Men and women exhibited similar arterial responses to acute resistance exercise (Cortez-Cooper et al. 2005).

Research has shown increases in arterial stiffness following acute resistance exercise versus decreases in arterial stiffness after aerobic exercise (Collier et al. 2010). Maximal aerobic exercise has been shown to reduce peripheral arterial stiffness (Naka et al. 2003). Acute aerobic exercise has been demonstrated to decrease central arterial stiffness (Kingwell et al. 1997). Heffernan et al. examined the effect of acute maximal aerobic exercise in 30 resistance trained and non-resistance trained men on vascular response and found a decrease in peripheral arterial stiffness (8.4m/s to 7.0m/s) in both groups (2007). Measurements of pulse wave velocity were taken at baseline, 10 minutes, 20 minutes, and 30 minutes post maximal aerobic exercise. PWV returned to normal at 20 and 30 minutes post exercise.

In contrast, it has been demonstrated that acute upper body resistance exercise increases central arterial stiffness (Heffernan et al. 2007). Collier et al. investigated vasodilatory and arterial distensibility responses to acute aerobic exercise versus resistance exercise and found a

decrease in central pulse wave velocity following aerobic exercise (-0.45m/s), but an increase in central pulse wave velocity (+0.29m/s) after resistance exercise (2010). This study examined 10 young, healthy males and measurements were taken at baseline, 40 minutes, and 60 minutes after exercise. The resistance exercise involved upper and lower body movements for 3 sets of 10 repetitions at 65%1RM.

Devan et al. examined the acute effects of resistance exercise on arterial compliance and found a decrease in central arterial compliance in men and women compared to a sham control group. However, arterial compliance returned to normal in less than 60 minutes. The study protocol included 9 resistance exercises at 75%1RM. The sample included sedentary and active adults.

Heffernan et al. examined arterial stiffness and baroreflex sensitivity following acute aerobic and resistance exercise in 13 males (2006). An increase in central PWV (4.8 to 5.8m/s) was found following resistance exercise. A decrease in central and peripheral PWV (5.3 to 4.8m/s; 8.9 to 8.3m/s) was found after aerobic exercise. The study protocol included 7 upper and lower body resistance exercises performed for 3 sets and 10 repetitons at 10RM.

Central arterial stiffness has been shown to increase significantly following acute resistance exercise (Lydakis et al. 2008). 15 young adults performed a leg extension and hand grip exercise and experienced an increase in central arterial stiffness. Subjects performed a static handgrip contraction at 40%MVC to fatigue, short bouts at 20% and 70%MVC with 1 minute rest periods, and a leg ergometer exercise with added load increments.

Lydakis et al. examined central arterial stiffness following an acute isometric handgrip and found a reduction in transit time, an index of arterial stiffness (2008). These results were

from a pool of 13 healthy adult men and women. An isometric handgrip contraction was performed at 40%MVC until fatigue. When compared to a lower body negative pressure condition, sympathetic nervous system activation was similar. An increase in pulse pressure was also observed in the isometric condition and this was related to the increase in arterial stiffness. Yoon et al. demonstrated that acute resistance exercise significantly increases central arterial stiffness (2010). 13 young men completed a series of 8 exercises at 60%1RM and a resting control period on separate days. There were no significant changes in blood pressure. PWV measurements were taken at 20 minutes and 40 minutes post exercise.

In contrast, acute resistance exercise has been shown to decrease arterial stiffness locally in exercised limbs (Heffernan et al. 2006). This phenomenon is currently unexplained. Heffernan et al. examined the effects of lower-limb unilateral resistance exercise on central and peripheral arterial stiffness in 13 young, healthy adult men and women (2006). No changes were found in central pulse wave velocity; however, there was a significant decrease in peripheral pulse wave velocity in the exercised leg from baseline to 5 minutes and 25 minutes post exercise (8.7m/s to 7.8m/s). As for blood pressure, systolic blood pressure increased significantly from baseline to 5 minutes post exercise (126.9 to 133.7mmHg), but returned to normal 25 minutes post exercise. No change was found in diastolic blood pressure and heart significantly increased from baseline to 5 minutes post, and 25 minutes post exercise. The exercise chosen in this study was a single leg press performed for six sets at 85% of 1RM to volitional fatigue with two minute rest periods between sets.

Regional arterial stiffness has been shown to decrease following external mechanical compression (Heffernan et al. 2007). Mechanical compression to the limb simulates the concentric/eccentric cycle during resistance exercise. Heffernan et al. found a decrease in

peripheral PWV (8.6 to 7.6m/s) following external mechanical compression in 16 young men. The compression bouts were administered in 6 sets of 12 compression cycles which is similar to 6 sets of 12 repetitions of leg resistance exercise.

Exercise Mode and Intensity

It is unknown how traditional resistance exercise compares to circuit exercise with respect to arterial stiffness and blood pressure. Studies examining different modes and intensities of resistance exercise with respect to arterial stiffness are scarce. Tanimoto et al. examined low-intensity slow resistance exercise versus high intensity normal speed resistance exercise and found increased basal femoral blood flow for both exercise modes (2008). This study protocol involved 36 untrained men assigned to either condition or a resting control. The low intensity condition consisted of a 55%1RM load and the high intensity condition was set at 85%1RM. Upper and lower body exercises were performed for 3 sets of 8 repetitions with 60 second rest periods between sets and 3 minute rest periods between exercise types. Individuals who are relatively stronger than others may experience more cardiovascular benefit with circuit resistance exercise since heavy resistance circuit exercise has been shown to elicit greater cardiovascular load than traditional strength training sets (Alcaraz et al. 2008).

Performance of the Valsalva maneuver during resistance exercise has been shown to be a factor in altering arterial stiffness. Heffernan et al. examined arterial stiffness and blood pressure following repeated Valsalva maneuvers versus resistance exercise in 14 young men (2007). Blood pressure was similar between conditions, however central PWV increased only after repeated Valsalva maneuvers (7.1 to 7.8 m/s) with no change after resistance exercise alone. Also, no change was found in peripheral PWV after either conditions. The resistance exercise

condition consisted of a leg press and leg extension performed for 15 total sets for 10 repetitions at 75%1RM with 70 second rest periods.

Blood Pressure

Researchers have closely examined the influence of resistance exercise on blood pressure. In a meta-analysis of 28 randomized, controlled trials in 1012 young adults, resistance exercise significantly reduced systolic blood pressure (-3.9mmHg) in normotensive individuals (Cornelissen et al. 2011). Resistance was categorized as dynamic forms of exercise or isometric handgrip contractions. Moving against resistance for a set number of repetitions characterizes dynamic resistance exercise. Dynamic resistance exercise showed greater increases in arterial stiffness compared to isometric handgrip exercise. This may be due to the relatively greater work demand necessary for dynamic resistance exercises than that of isometric hand grip exercise.

An important note is that blood pressure does vary throughout the day. Blood pressure has been shown to fluctuate after exercise in different ways depending on the time of day (Jones et al. 2008). Following acute aerobic exercise, blood pressure decreased (7mmHg) in the 4pm condition but increased (3mmHg) in the 8am condition. The mechanism for this daily blood pressure fluctuation may be circadian rhythm. For this reason, it is advisable to set testing visits for the same time of day. There is strong evidence that acute aerobic exercise significantly reduces clinical and ambulatory blood pressure post exercise (Cardoso et al. 2010).

Lamotte et al. found that systolic blood pressure and heart rate returns to resting levels with recovery periods of 90 seconds or more following a set of resistance exercise (2006). This study involved both men and women and the resistance exercise protocol consisted of 4

conditions each with leg extension for 3 sets of 10 repetitions at 75%1RM. Only rest periods were manipulated for each condition at 30, 60, 90, and 120 seconds between sets.

Baroreceptor sensitivity, which affects changes in blood pressure, is reduced up to 60 minutes following heavy resistance exercise (Niemela et al. 2008). This study examined baroreceptor sensitivity recovery patterns after aerobic, light and heavy resistance exercise, and a resting control in 12 healthy men. The resistance exercise protocol consisted of a leg press, chest curl, leg extension, and biceps curl performed for 3 sets of 12 repetitions. The light resistance intensity was set at 30%1RM and the heavy resistance intensity was set at 80%1RM. BRS was measured from 30 to 180 minutes after each condition. Low frequency and high frequency power were measured as an index of autonomic activity with low frequency indicating sympathetic activity and high frequency indicating parasympathetic activity. High frequency power was reduced and low frequency power increased following heavy resistance exercise which will be explained later.

Autonomic Function

Autonomic modulation contributes to variability in heart rate and vasomotor tone which can influence arterial blood flow. Measurements of heart rate variability (HRV) can be analyzed via power spectral analysis to determine autonomic modulation. High frequency (HF) and low frequency (LF) components of HRV reveal cardiac vagal and both sympathetic and vagal activity, respectively. High frequency bandwidth ranges from 0.15-0.40Hz while low frequency bandwidth ranges from 0.04-0.15Hz. High frequency peak is responsible for HRV at maximal intensity exercise (Perini et al. 2003). Low frequency power increases with high intensity resistance exercise, indicating sympathetic activation.

Heffernan et al. examined the effects of acute resistance exercise on autonomic function in 12 healthy, active men and found a reduction in cardiac vagal control (2007). Normalized high frequency power decreased following resistance exercise, indicating reduced parasympathetic activity (0.57 to 0.44). In addition, LF/HF ratio increased, indicating sympathetic activation. This study protocol incorporated a leg press and leg extension performed for 15 total sets for 10 repetitions at 75%1RM with 1.5 minute rest periods between sets.

Teixeira examined cardiac autonomic modulation in 20 healthy adults following both aerobic and resistance exercise and found an increase in sympathovagal balance in all modes (2011). The greatest increase in cardiac sympathetic activation was observed following concurrent aerobic and resistance exercise. Blood pressure decreased in all exercise conditions. The resistance exercise protocol consisted of 6 upper and lower body exercises performed for 3 sets of 20 repetitions at 50%1RM with 45 second rest periods between sets and 90 seconds between exercises.

Millar et al. investigated autonomic modulation following acute isometric handgrip exercise and found improvements in acute cardiac vagal activity (2009). These results were found in 18 healthy older men and women. The hand grip protocol consisted of 4 repetitions at 30%MVC with 1 minute rest periods. This is contrary to alternate findings for acute resistance exercise which may be explained by the age difference in groups.

Lima et al. examined the acute effect of resistance exercise intensity in cardiac autonomic modulation (2011). A group of 15 young men performed 5 upper body resistance exercises for 3 sets of 12, 9, and 6 repetitions at 50%1RM and at 70%1RM, in addition to a control rest condition. High frequency power decreased and low frequency power and LF/HF ratio increased

significantly following the high intensity condition. It is evident that resistance exercise alters cardiac sympathovagal balance by reducing parasympathetic activity and increasing sympathetic activity in young adults.

The current literature suggests that acute resistance exercise negatively affects central arterial stiffness; however, positively influences peripheral arterial stiffness. In addition, high intensity resistance exercise has the most profound effect on arterial stiffness in comparison to low and moderate intensity resistance exercise. Dynamic resistance exercise increases central arterial stiffness compared to isometric resistance exercise. Although remarkable increases in blood pressure occur during resistance exercise, blood pressure appears to decrease below baseline levels post exercise. As for autonomic modulation, acute resistance exercise increases sympathetic activity and reduces parasympathetic activity in young adults. The major discrepancy in study design methods for several experiments is the type of resistance exercise protocol included. Few studies utilize the same protocol for resistance exercise which may confound findings. More research will be necessary on different intensities and modes of acute resistance exercise with respect to arterial stiffness and autonomic modulation.

CHAPTER 2

HYPOTHESES

The purpose of this study was to examine the effects of an acute bout of high intensity resistance exercise versus low intensity resistance exercise on arterial stiffness and blood pressure. We hypothesized that (**1a**) Central arterial stiffness would decrease and peripheral arterial stiffness would decrease from baseline following the low intensity circuit resistance exercise protocol at 15 minutes post exercise and remain the same at 30 minutes post exercise. (**1b**) Central arterial stiffness would increase and peripheral arterial stiffness would decrease from baseline following the high intensity traditional resistance exercise protocol at 15 minutes post and remain the same at 30 minutes post exercise. (**2a**) Blood pressure would decrease from baseline following both the low intensity circuit resistance exercise protocol and the high intensity traditional resistance exercise protocol at 15 minutes post exercise and remain the same at 30 minutes post exercise. (**3a**) High frequency power would decrease and LF/HF ratio would increase following the high intensity traditional resistance exercise protocol at 15 minutes post exercise and remain the same at 30 minutes post exercise. (**3b**) High frequency power and LF/ratio would not change following the light intensity resistance exercise protocol.

CHAPTER 3

METHODOLOGY

Subjects

There were 36 total subjects signed up to participate in this study. 32 subjects participated in this study to completion. See table I for baseline values and characteristics of the participants. The subject pool was comprised of resistance exercise trained and untrained males and females who were non-smokers, and had no cardiovascular limitations, especially with respect to exercise. The requirement for resistance trained was performing resistance exercise more than 3 days per week. The majority of the subjects were young adults living in the vicinity of the University of Illinois at Urbana-Champaign. The research was approved by the University of Illinois at Urbana-Champaign Institutional Review Board. Study participants voluntarily signed a document of informed consent which disclosed the purpose, protocol, and subsequent risk associated with participation in the study. The identities of the subjects were kept confidential and each subject was assigned a number for data collection purposes. Study participants also voluntarily provided their gender information, age and whether or not they exercise regularly. This information may be used later to analyze the influence of gender on study results. Subjects completed a Physical Activity Readiness Questionnaire (PAR-Q) to assess any issues they might have with performing exercise. Subjects indicating any issue that would prevent them from safely engaging in physical activity such as musculoskeletal injury, loss of consciousness, and chest pain, were excluded from the study. Baseline measures were taken for each subject including height in meters, weight in kilograms, body mass index (BMI), and blood pressure. Subjects were excluded from participating in the study if their resting

systolic blood pressure was beyond 140 mmHg, indicating hypertension. Subjects were instructed to refrain from eating 3 hours prior, consuming alcohol 12 hours prior, and exercising 24 hours prior to the exercise protocol.

Exercise Order

The majority of studies observing the influence of acute resistance exercise on arterial function involve total body exercise (Heffernan et al. 2006). Therefore, total body resistance exercise is incorporated into this study protocol. Both exercise conditions were randomized for all subjects to prevent any order effect. The exercise protocol consisted of eight traditional resistance exercises, recognized by the American College of Sports Medicine, that targeted different muscle groups over the whole body (Amercican College of Sports Medicine Position Stand 2002). The exercise order is as follows: Barbell Bench Press (Chest), Standing Dumbbell Curl (Biceps), Seated Vertical Pulldown (Back), Standing Rope Pushdown (Triceps), Overhead Barbell Press (Shoulders), Seated Leg Press (Gluteal muscles), Seated Leg Extensions (Quadriceps), and Bench Leg Curl (Hamstrings). The exercises order was balanced in such a way to prevent subjects from overexerting themselves at any particular moment during the protocol. Major muscle group exercises were separated with minor muscle group exercises. Furthermore, upper body exercises were grouped together as well as lower body exercises to avoid extensive peripheral heart action. Each exercise protocol included a warm-up segment prior to exercise and a cool-down segment following exercise. Both segments included range of motion activities such as arm swings, knee hugs, ankle pulls, and crouching.

Resistance Exercise Capacity

Whereas aerobic exercise capacity is based on maximum oxygen uptake (VO2max), resistance exercise capacity is based on repetition maximum (RM). In order to determine one repetition maximum, subjects performed up to 10 repetitions of the highest manageable load for each exercise. Multiple sets of exercise were required for some subjects who could perform more than 10 repetitions at one load and needed a heavier load to assess their maximum. In this case, weight was added accordingly until the subjects reached their repetition maximum. The 1RM was measured based on a repetition maximum scale authorized by the National Strength and Conditioning Association. For instance, if a subject performed 8 repetitions at one load their estimated 1RM could be determined using the RM calibration scale. This was performed on a separate day before the exercise protocol visits.

Circuit Resistance Exercise Protocol (Low Resistance Condition)

The low intensity condition was labeled as the Circuit Resistance Exercise Protocol (CREP) to reflect the continuous nature of the exercise. Circuit training is a series of exercises with no rest periods. It is evident that circuit training improves peripheral muscular endurance and aerobic capacity (Allen et al. 1976; Gettman et al. 1978). The low intensity condition consisted of all resistance loads being set at 40 percent of the subject's one repetition maximum (40% of 1RM). In the low resistance condition, subjects engage in continuous resistance exercise. Rest periods are absent from the beginning to the end of the exercise circuit. The rest between sets significantly affects cardiovascular responses to resistance exercise (Fleck 1988). To prevent subjects from performing two consecutive sets of the same exercise, the order of circuit was staggered in which two different exercises would be performed back to back.

Traditional Resistance Exercise Protocol (High Resistance Condition)

The high intensity condition was labeled as the Traditional Resistance Exercise Protocol (TREP) to reflect the general definition for resistance exercise involving sets of anaerobic muscle contractions followed by rest periods. This condition consisted of all resistance loads being set at 80 percent of the subject's one repetition maximum (80% of 1RM). This exercise protocol included a 1 minute rest period after each set of resistance exercise. It has been shown that high intensity resistance training (85%1RM) and low intensity resistance training (55%1RM) both increase basal limb blood flow in untrained, young men (Tanimoto et al. 2008). We chose to examine this phenomenon in both men and women.

Cardiovascular Measurements

All measurements for both conditions were taken at baseline, 15 minutes post-exercise, and 30 minutes post-exercise. Research suggests significant changes to arterial wall characteristics following acute exercise at 20 minutes post and 30 minutes post exercise (Kingwell et al. 1997). Therefore, the aforementioned time periods were selected for measurements.

Central and peripheral arterial stiffness- PWV

All measurements were conducted in adherence to the guidelines of the Clinical Application of Arterial Stiffness, Task Force III (Van Bortel et al. 2002). Arterial pulse wave velocity (PWV) was used to measure arterial stiffness which has been demonstrated to be highly reproducible (Wilkinson et al. 1998). PWV and pulse wave quality were recorded using a single high fidelity straingauge transducer and accompanying software (SphygmoCor, AtCor Medical, Sydney, Australia). Pressure waveforms were captured between (1) the right common carotid artery and the right femoral artery, (2) the right femoral artery and the ipsilateral superior

dorsalis pedis artery, and (3) the right common carotid artery and the ipsilateral radial artery. ECG recordings for heart rate were conducted via a 3-lead CM5 configuration. Distances from the carotid artery sampling site to the radial artery, carotid artery to the suprasternal notch, carotid artery to the femoral artery, and femoral artery to the superior dorsalis pedis artery were measured with a tape measure. The carotid-femoral segment length was used to measure central arterial stiffness. The femoral-dorsalis pedis and the carotid-radial segment lengths were used to measure peripheral arterial stiffness. The difference was taken from the distances of the (1) carotid artery to the sternal notch, and (2) the sternal notch to the femoral artery, to account for fluctuations in propagation direction along the arterial path. Blood pressure was measured using an automated oscillometric cuff (HEM-712C, Omron Corporation, Japan). The average of two subsequent blood pressure measurements was recorded.

Heart Rate Variability

Participants lay in the supine position on a cushioned table in a quiet room with the lights dim to eliminate sound and light noise interference. An Electrocardiogram (ECG) with a single lead CM5 configuration was used to record heart rate (Biopac Systems, Santa Barbara, USA). Subjects performed a 5 minute epoch of unpaced breathing followed by another 5 minute epoch of paced breathing. Signal processing was performed using software at a later date (WinCPRS, Turku, Finland). Normalized low frequency and high frequency bandwidths, in addition to LF/HF balance ratio were recorded to determine sympathovagal balance and autonomic activity. Electrodes remained on the participants pre and post exercise to reduce alterations in the heart rate measurements.

Statistical Analysis

The baseline data are reported as means ±SEM while the pre, post-15, and post-30 minute data for both modes are reported as means ±SD. A priori significance was set at an alpha <0.05. Analysis of variance (ANOVA) with repeated measures was conducted to assess differences over time in each of these dependent variables: HR; nLF, nHF, LF/HF; aortic and brachial SBP, DBP, MAP, PP; and peripheral and central PWV. A 2 x 3 ANOVA with repeated measures was used to assess differences in BP, HR, HRV, and central and peripheral PWV between the TREP condition and the CREP condition over time (pre, 15-min post, and 30-min post). A paired samples t-test was used to assess within subject differences in resting BP, HR, HRV, and central and peripheral PWV. All data analysis was conducted using Statistical Package for the Social Sciences (SPSS, v 19, SPSS, Inc. Chicago, Il).

CHAPTER 4

RESULTS

See table II for baseline values of PWV, BP, and HRV. See Table III and Table IV for values at baseline, 15 minutes post-exercise, and 30 minutes post-exercise for CREP and TREP modes, respectively.

Central and Peripheral Arterial Stiffness

For central PWV, see Figure I, there was a significant time and mode effect, in addition to a time x mode interaction ($F=6.61, 6.64, 16.34$; $p<0.05$). Central PWV increased from baseline to 15 minutes post-exercise in the low intensity CREP mode (7.2 to 7.4m/s), then returned toward baseline values at 30 minutes post-exercise. No change in central PWV was witnessed in the high intensity TREP mode. For brachial PWV, see Figure II, there was a significant time effect and time x mode interaction ($F=15.34, 16.16$; $p<0.01, 0.05$). Brachial PWV increased from baseline to 15 minutes post-exercise in the high intensity TREP mode (7.5 to 7.7m/s), and remained elevated at 30 minutes post-exercise. No change in PWV occurred in the low intensity CREP mode. For femoral PWV, see Figure III, there was a significant time effect and time x mode interaction ($F=5.77, 18.82$; $p<0.05$). Femoral PWV increased from baseline to 15 minutes post-exercise in the high intensity TREP mode (6.4 to 6.7m/s), and remained elevated at 30 minutes post-exercise. No changes in PWV were observed in the low intensity CREP mode.

Heart Rate Variability

See Table II, Table III, and Table IV for values. For heart rate, there was a significant time effect ($F=8.71$; $p<0.01$). Heart rate increased for both modes. For normalized high frequency power (nHF), there was a significant mode effect ($F=5.31$; $p<0.05$). High frequency power was higher in the CREP mode compared to the TREP mode. For normalized low frequency power (nLF), there was a significant mode effect ($F=4.82$; $p<0.05$). Low frequency power was lower in the CREP mode compared to the TREP mode. For LF/HF ratio, there was a significant mode effect ($F=5.17$; $p<0.05$). LF/HF ratio increased for both modes.

Central Blood Pressure

See Table II, Table III, and Table IV for values. For aortic systolic blood pressure, there was a significant time effect ($F=7.84$; $p<0.01$). Central systolic blood pressure increased in both modes from baseline to 15 minutes post, and 30 minutes post-exercise. For aortic diastolic blood pressure, there was a significant time and mode effect, in addition to a time x mode interaction ($F=6.95, 11.75, 4.71$; $p<0.05, 0.01, 0.05$). Central diastolic blood pressure increased in both modes from baseline to post-exercise, and remained elevated at 30 minutes post-exercise. For aortic pulse pressure, there was a significant time and mode effect, in addition to a time x mode interaction ($F=5.45, 7.42, 4.31$; $p<0.01, 0.05, 0.05$). Central pulse pressure increased in the CREP mode and decreased in the TREP mode. For aortic mean arterial pressure, there was a significant time x mode interaction ($F=7.27$; $p<0.05$). Central Mean arterial pressure decreased in the CREP mode while it increased in the TREP mode.

Peripheral Blood Pressure

See Table II, Table III, and Table IV for values. For brachial systolic blood pressure, there was a significant time effect ($F=6.89$; $p<0.05$). Peripheral systolic blood pressure increased

for both modes. For brachial diastolic blood pressure, there was a significant time and mode effect, in addition to a time x effect interaction ($F=10.91, 14.07, 5.48$; $p<0.01, 0.01, 0.05$). Peripheral diastolic blood pressure decreased for both modes. For brachial pulse pressure, there was a significant time and mode effect, in addition to a time x mode interaction ($F=14.68, 10.27, 5.55$; $p<0.01, 0.01, 0.05$). Peripheral pulse pressure increased for both modes. For brachial mean arterial pressure, there was a significant mode effect ($F=7.14$; $p<0.05$). Peripheral mean arterial pressure decreased for the CREP mode and did not change for the TREP mode.

CHAPTER 5

DISCUSSION AND CONCLUSION

Significant findings

The noteworthy findings in this study are the arterial stiffness outcomes. A significant time x mode interaction was observed for aortic, brachial, and femoral PWV measures indicating that the two exercise modes influenced arterial stiffness differently from baseline to post-exercise. Central PWV increased in the low intensity CREP mode from baseline to 15 minutes post-exercise (+0.20m/s), and returned toward baseline values at 30 minutes post-exercise (7.3m/s). Central PWV did not change in the high intensity TREP mode from baseline to post-exercise which does not support the literature. Acute resistance exercise has been demonstrated to increase central arterial stiffness (Lydakis et al. 2008; Heffernan et al. 2006; Sesso et al. 2000). Collier et al. observed an increase in central arterial stiffness following acute resistance (+0.29m/s) exercise compared to a decrease following acute aerobic exercise (2010). Our findings support the literature on the effects of acute resistance exercise on central arterial stiffness. Our findings do not support the hypothesis that low intensity circuit resistance exercise (CREP) would reduce central arterial stiffness compared to traditional resistance exercise (TREP). During the familiarization demonstration, subjects were instructed to avoid using a Valsalva maneuver in the exercise modes. However, it is possible that subjects accidently, or intentionally, performed Valsalva breathing during the exercise modes which has been shown to increase central arterial stiffness (Heffernan et al. 2007).

In addition, our findings do not support our hypothesis that both exercise modes would decrease peripheral PWV. The current literature suggests that acute resistance exercise

decreases peripheral PWV (Heffernan et al. 2006). We found no change in peripheral PWV after low intensity exercise; however, peripheral PWV increased (brachial +0.2m/s; femoral +0.3m/s) after high intensity exercise and remained elevated. Bertovic et al. found that resistance trained individuals have a higher level of central and peripheral stiffness compared to non-resistance trained individuals (1999). Several of the subjects were resistance trained and this, in addition to the relatively small sample size, may have confounded the findings.

Furthermore, peripheral brachial and femoral PWV increased in the TREP mode from baseline to 15 minutes post-exercised (+0.2m/s and +0.3m/s). Peripheral brachial and femoral PWV did not change for the CREP mode. Heffernan et al. demonstrated that acute resistance exercise reduces peripheral PWV (2007). These findings do not support the literature and the hypothesis that high intensity traditional resistance exercise (TREP) would reduce peripheral arterial stiffness.

Although time x mode interactions were observed in both aortic and brachial blood pressure measures, there was no significant time x mode interaction in aortic and brachial systolic blood pressure. Acute resistance exercise has been shown to decrease systolic blood pressure (Cornelissen et al. 2011). We found an increase in aortic and brachial systolic blood pressure; however, mode had no influence on systolic blood pressure over time. This does not support the literature which suggests a post-exercise hypotensive effect.

There was no significant time x mode interaction seen in heart rate variability measures. Time effects were of little importance since they did not explain the influence of the mode. In addition, mode effects were of little importance since they only indicated that values were different between modes without respect to time. Normalized high frequency power was higher

at baseline in the CREP mode compared to the TREP mode. This suggests that subjects had a higher degree of parasympathetic activation in the CREP mode but it did not change significantly over time. It may be that the subjects were more relaxed at baseline in the CREP mode; and the low intensity resistance stimulus was not a sufficient influence for altered heart rate variability. Lima et al. found no changes in high frequency power or LF/HF ratio following light resistance (50%1RM) exercise (2011). In contrast, normalized high frequency power was lower at baseline in the TREP mode compared to the CREP mode. Subjects may have been less relaxed or more anxious at baseline in the TREP mode. Although no significant change in normalized high frequency power occurred over time, at 80%1RM, it is unlikely that the TREP mode provided insufficient intensity to alter heart rate variability. Based on the literature, high intensity resistance exercise decreases high frequency power and increases LF/HF ratio (Heffernan et al. 2007). LF/HF ratio increased for both modes but there was no significant time x mode interaction. These findings do not support the hypothesis that high intensity resistance exercise would decrease high frequency power and increase LF/HF ratio from baseline to post-exercise. This could be contributed to the differences at baseline between modes apparent within subjects.

Heart rate increased from baseline to 15 minutes post-exercise for both modes. However, the exercise mode did not significantly influence heart rate over time. There is evidence to suggest that increases in heart rate increase both central and peripheral PWV (Liang et al. 1999). Our findings for heart rate and PWV support the literature.

Mechanisms

Evidence supporting reductions in peripheral arterial stiffness has been explained by particular mechanisms. Heffernan et al. proposed that resistance exercise induces sheer stress on

the arterial wall which in turn releases nitric oxide (NO) from the endothelium and causes vascular smooth muscle cell relaxation, and increased vasodilatation of the arterial wall (2006). Exercise induced NO release has been identified as a significant contributor to changes in arterial wall characteristics (Joyner 2000). It is possible that the low intensity resistance exercise mode did not provide sufficient stimulus, or sheer-stress, to induce NO release from the endothelium, and subsequent vasodilatation in the peripheral arteries of the exercising region. Another mechanism that may be responsible for arterial wall adaptations is the flow-mediated dilation of arteries stemming from the metabolite-induced dilation of capillaries in the exercising region (Kingwell 2000). The vasodilation occurring in the capillary beds of exercising muscle influences arterial wall function (Kingwell et al. 1997). It is possible that this mechanism prevented an increase in central arterial stiffness in the high intensity resistance exercise condition.

Gender Differences

There did not appear to be any gender differences since each subject completed both exercise modes. Men and women have similar arterial responses to acute resistance exercise (Cortez-Cooper et al. 2005). This study included women to further examine gender differences as they relate to acute resistance exercise. Individuals who are relatively stronger than others may experience more cardiovascular benefit with circuit resistance exercise since heavy resistance circuit exercise has been shown to elicit greater cardiovascular load than traditional strength training sets (Alcaraz et al. 2008).

Limitations

One limitation that may have affected study results was the use of pulse wave velocity to determine aortic stiffness. There is evidence indicating differences in measurement between the use of pulse wave velocity and ultrasonography to determine aortic stiffness (Paini et al. 2006). Another potential limitation is that there was not an aerobic exercise condition built into the study design to compare to the resistance exercise conditions. Furthermore, there was not a control rest day in which arterial stiffness and blood pressure measures were taken at baseline, 15 minutes post, and 30 minutes post. Fitness status was assessed via self-report measures and may have failed to correctly represent the sample. VO2 is a strong predictor of fitness; however, it was not employed in this study protocol. For select exercises, there were subjects whose 1RM (up to 10RM) was far greater than the maximum load available in the laboratory. As a result, these particular subjects would perform their high intensity exercises at the maximum available load even if it was below their respective 80% 1RM values. This may have influenced subject real outcomes. None of the subjects were tested for steroid use, which has been associated with increased arterial stiffness and decreased vasodilation (Kasikcioglu et al. 2007). Subject serum lipid profiles or plasma glucose levels were not measured. Extreme differences in these measurements between subjects and within subjects could confound findings regarding arterial changes. It was expected that subjects would fast at least 3 hours prior to each testing visit.

Conclusion

In conclusion, this study revealed that exercise intensity has differential effects on central and peripheral arterial stiffness. In addition, acute resistance exercise does influence central and peripheral arterial stiffness. High intensity resistance exercise increased peripheral arterial stiffness while low intensity resistance exercise increased central arterial stiffness. These

findings are contrasting to the current literature. More research will be needed to determine if this result holds for other populations including older adults, and clinical groups.

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TABLES AND FIGURES

Table I. Subject Characteristics	
Variable	(n=32)
Age (years)	24.6 +/- 4.0
Height (cm)	172.2 +/- 10.3
Weight (kg)	70.3 +/- 10.7
BMI	23.8 +/- 4.0
1-RM (kg)	62.9 +/- 25.5
Values are means +/- SEM	
1-RM 1 repetition max for chest press	

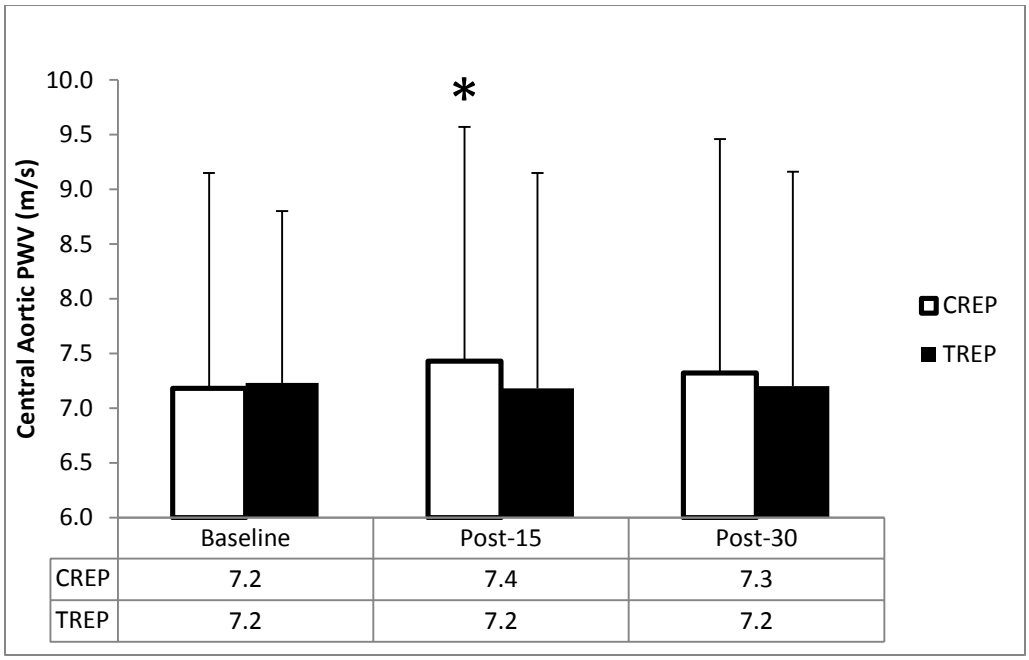
Table II. Baseline values for both conditions				
Variable	CREP	(+/-)SEM	TREP	(+/-)SEM
SBP	113.1	1.4	112.6	1.4
DBP	71.0	1.0	71.2	1.1
MAP	84.9	0.9	85.0	0.9
PP	41.5	1.5	41.0	1.5
aorSBP	96.8	1.6	96.5	1.6
aorDBP	70.8	1.5	71.0	1.0
aorMAP	82.1	1.5	79.9	0.9
aorPP	29.1	2.1	29.3	2.2
bPWV	7.6	0.2	7.4	0.2
cPWV	7.1	0.2	7.2	0.2
lPWV	6.7	0.3	6.3	0.2
HR	61.0	1.7	60	1.6
LF/HF	57.52	6.51	144.64	36.32
nLF	0.33	0.02	0.43	0.04
nHF	0.66	0.02	0.56	0.04

Table III. Values at baseline, 15 minutes post, and 30 minutes post exercise for the low intensity CREP mode.						
Variable	Baseline	(+/-)SD	Post-15min	(+/-)SD	Post-30min	(+/-)SD
SBP	113.1	8.1	119.0	12.4	119.0	12.4
DBP	71.0	5.9	61.7*	15.0	63.3	9.2
MAP	84.9	5.3	80.8	11.0	81.9	7.3
PP	41.5	8.7	57.2*	18.9	55.6	15.6
aorSBP	96.8	9.2	102	10.3	104	12.7
aorDBP	70.8	8.8	62*	15.4	63	9.4
aorMAP	82.1	8.9	77*	11.5	78.4	8.2
aorPP	29.1	11.9	40*	18.0	39	14.8
HR	61	10.1	68	11.3	61	9.6
bPWV	7.6	1.2	7.7*	1.31	7.6	1.2
cPWV	7.1	1.4	7.4*	1.6	7.3	1.6
lPWV	6.7	1.9	6.7*	2.1	6.6	2.1
LF/HF	57.52	36.83	73.19	93.46		
nLF	0.33	0.13	0.34	0.19		
nHF	0.66	0.14	0.65	0.18		

*Significant time x mode interaction.

Table IV. Values at baseline, 15 minutes post, and 30 minutes post exercise for the high intensity TREP mode.						
Variable	Baseline	(+/-)SD	Post-15min	(+/-)SD	Post-30min	(+/-)SD
SBP	112.6	8.1	114.4	9.1	120.2	14.5
DBP	71.2	6.3	69.8*	7.3	70.3	5.9
MAP	85.0	5.1	84.7	6.0	86	5.2
PP	41.0	8.6	44*	11.0	49	17.2
aorSBP	96.5	9.0	99	9.2	102	11.7
aorDBP	71.0	5.9	70*	7.2	70	6.2
aorMAP	79.9	5.5	82*	6.7	82	6.3
aorPP	29.3	12.8	29*	12.1	31	13
HR	60	9.1	65	9.5	61	9.6
bPWV	7.4	1.1	7.7*	1.3	7.7	1.3
cPWV	7.2	1.4	7.1*	1.4	7.2	1.4
lPWV	6.3	1.5	6.7*	1.9	6.7	1.9
LF/HF	144.64	205.46	181.25	323.8		
nLF	0.43	0.23	0.43	0.24		
nHF	0.56	0.23	0.55	0.24		

*Significant time x mode interaction.



	Baseline	Post-15	Post-30
CREP	7.2	7.4	7.3
TREP	7.2	7.2	7.2

Figure I. Central aortic pulse wave velocity for both conditions at baseline, 15 minutes post-exercise, 30 minutes post-exercise.

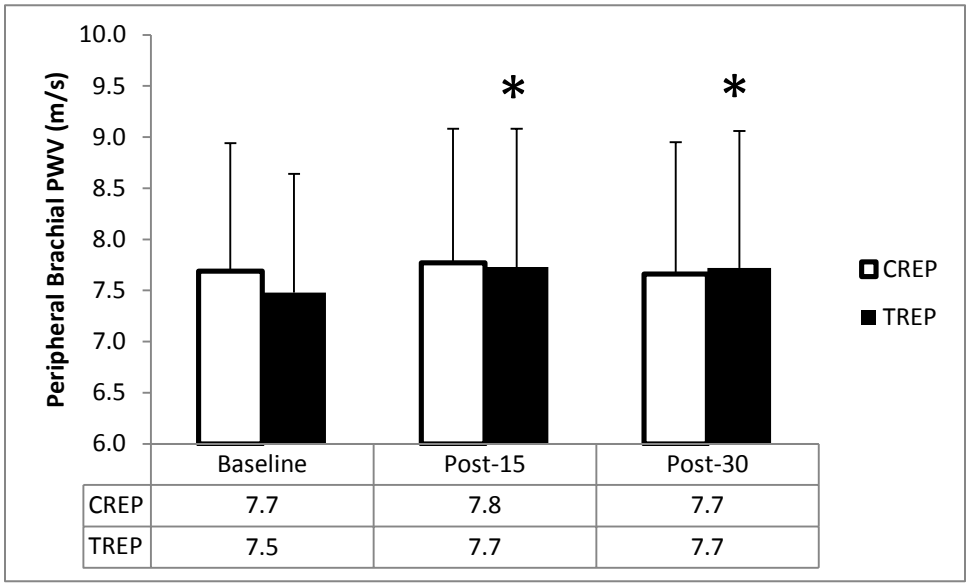


Figure II. Peripheral brachial pulse wave velocity for both conditions at baseline, 15 minutes post-exercise, 30 minutes post-exercise.

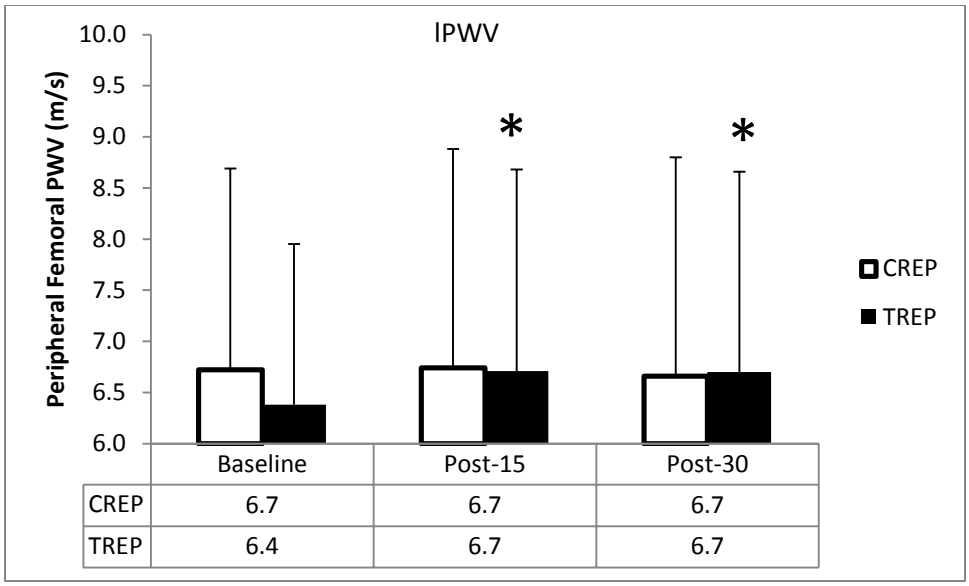


Figure III. Peripheral femoral pulse wave velocity for both conditions at baseline, 15 minutes post-exercise, 30 minutes post-exercise.