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POST-EXERCISE HYPOTENSION IN BRIEF EXERCISE

A Thesis Presented to The Faculty of the Department of Kinesiology, Recreation, and Sport Western Kentucky University Bowling Green, Kentucky

> In Partial Fulfillment Of the Requirements for the Degree Master of Science

> > By Jeremiah G. Bush May 2011

Date Recommended 4.26.11 Dr. Mark Schafer, Director of Thesis Dr. James Javalta Dr. Scott Arnett

Dean, Graduate Studies and Research Date Date icnard &

ACKNOWLEDEGEMENTS

Thanks to Dr. Mark Schafer for chairing this Thesis committee and guiding the development and completion of this research, and for assisting with statistical analysis. Thanks also to Dr. James Navalta and Dr. Scott Arnett for serving on the thesis committee and providing additional insight and knowledge for the completion of the research.

Thanks to Western Kentucky University's Exercise Science Lab for allowing the use of all materials during the experiment.

Special thanks to the Western Kentucky University's Graduate Studies Research Department and the College of Health and Human Services for providing grant monies for the funding of the research.

TABLE OF CONTENTS

Title Page	
Signature Page	
Acknowledgements	i
Table of Contents	ii
Abstract	iv
 Chapter I: Introduction Statement of Problem Statement of Purpose Statement of Hypothesis Limitations Delimitations Definition of Terms Chapter II: Review of Literature Treadmill/Cycling Protocol Mechanisms of PEH Conclusions Chapter II Methods Isubjects Instruments Design A Data Analysis 	3 6 7 7 8 8 8 10 10 10 14 17 18 18 18 18 19 21
Chapter IV: Results	23
Chapter V: Discussion	26
Appendix A: Informed Consent	33
Appendix B: Physical Activity Readiness Questionnaire	36
Appendix C: Medical History	37
Appendix D: Bruce Protocol/Borg RPE Scale	38
Appendix E: Subject Demographic	39
Appendix F: 40% VO ₂ R ANOVA	40

Appendix G: 40% Systolic Post-Exercise BP Graph	41
Appendix H: 40% Diastolic Post-Exercise BP Graph	42
Appendix I: 70% VO ₂ R ANOVA	43
Appendix J: 70% Systolic Post-Exercise BP Graph	44
Appendix K: 70% Diastolic Post-Exercise BP Graph	45
Appendix L: 40% v 70% ANOVA	46
Appendix M: 40% v 70% Systolic Post-Exercise BP Graph	47
Bibliography	48

POST-EXERCISE HYPOTENSION IN BRIEF EXERCISE

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Directed by: Mark Schafer	, James Navalta, Scott Arnett	
Department of Kinesiology	v, Recreation, and Sport	Western Kentucky University

The purpose of this investigation was to examine whether a single 10 minute bout of exercise, performed at multiple intervals throughout the day to equal 30 minutes, can effectively elicit post-exercise hypotension (PEH). Secondly, it is important to explore whether a light (40% VO_2R) or moderate (70% VO_2R) intensity is required to elicit PEH within 10 minutes. Subjects (N=11) completed a VO₂max test utilizing the Bruce Treadmill protocol. Each subject returned within 3 - 5 days to complete two separate exercising trials. A counter balanced system was employed so that each subject did not perform the same intensity rotation (Counter Balance 1 = 40% VO₂R and 70% VO₂R for session 1 and session 2, respectively; Counter Balance 2 = 70% VO₂R and 40% VO₂R for session 1 and session 2). The first session consisted of 3 sessions (morning, noon, evening) separated by an average of 3.5 hours at one of two intensities (40% VO₂R or 70% VO₂R). The second group of sessions were performed identical to the first, however, the intensity was altered depending upon counter balance. Baseline BP was measured prior to exercising. After each session, BP was measured at 2 intervals for the morning and noon sessions (immediately following and 20 minutes post-exercise); and at 3 intervals for the evening sessions (60 minutes post-exercise added) for both intensities. At 40% VO₂R, BP decreased significantly at the morning (p = 0.007), noon (p = 0.018) and evening (p = 0.010) sessions at the 20 minute post-exercise interval. Although not significantly different, BP was observed to be lower at 60 minutes post-exercise interval. During the 70% VO₂R session, BP was significantly lower at the morning 20 minute (p = .029) and evening 60 minute post-exercise measurements (p = .006) when compared to baseline. There was no significant difference noted between 40% and 70% VO₂R intensities at eliciting a drop in BP at any interval at any time point. Although not statistically significant, 70% VO₂R appeared to produce a further decrease at the 60 minute post-exercise measurement (102 mmHg) than did the 40% session (106 mmHg). The results of this study indicate that PEH may be elicited after a single 10 minute exercise session. Furthermore, multiple bouts of 10 minutes produce an accumulated decrease in BP that can be observed at the completion of the day.

Chapter 1

Introduction

It has been estimated that approximately 50 million Americans, and 1 billion people worldwide, are affected by hypertension that requires some form of treatment $^{(1,2)}$. Characterized by elevated blood pressure with no definite cause, sustained hypertension will result in thickening of arterial walls, inelasticity, resistance to blood flow, distended and hypertrophied left ventricle⁽³⁾. These physiologic changes have deleterious effects on three main systems if left untreated; the heart, brain and kidneys. In a text by Guyton and Hall⁽⁴⁾, hypertensive effects on these three systems are outlined: 1) as a result of weight gain, adipose tissue proliferates increasing metabolic rate. In order for blood to be adequately supplied to all areas efficiently, cardiac output must increase to perfuse the extra tissue; 2) in the brain, sympathetic nerve activity is increased via release of hormones that directly stimulate the hypothalamus and excite the vasomotor centers; 3) angiotensin II and aldosterone levels are increased secondary to increased renin release in response to the sympathetic nervous system stimulation. This impairs the renal-pressure natriuresis mechanism, and the kidneys will not excrete adequate amounts of salt and water. Increased amounts of circulating salt and water raise blood volume, thereby resulting in higher blood pressure.

Studies have shown that prolonged exposure to high blood pressure (BP) will increase the amount of work for the heart, which can lead to increased risk for cerebrovascular disease, ischemic heart disease, and renal disease^(1,2,4,5,6). Due to

decreased blood supply through non-compliant vessels resulting from hypertension, one may experience angina, myocardial infarction, congestive heart failure, severe headaches, blurred vision, confusion, or uremia⁽³⁾. The Department of Health and Human Services has classified blood pressure in the following manner: normal = $\leq 120/80$ mmHg, prehypertension = 120-139/80-89 mmHg, hypertension = $\geq 140/90$ mmHg, Stage 1 hypertension = 140-159/90-99 mmHg, Stage 2 hypertension = $\geq 160/100$ mmHg⁽¹⁾. The Department of Health and Human Services has indicated that for every 20 mmHg increase in systolic (SBP), or 10 mmHg increase in diastolic (DBP) blood pressure, the risk for mortality doubles from both ischemic heart disease and stroke. The risk for cardiovascular disease is also doubled when BP values are between 130-139 mmHg systolic and 85-89 mmHg diastolic. As a result, hypertension has been stated as the number one attributable risk for death⁽¹⁾.

The Department of Health and Human Services has recommended that every individual should engage in at least 30 minutes of physical activity per day, most days of the week⁽¹⁾. The Physical Activity Guidelines for Americans goes further, stating that physical activity should include 2 $\frac{1}{2}$ hours of moderate intensity (defined as a 5 or 6 on a 0 – 10 RPE Borg scale) or 1 $\frac{1}{4}$ hours of vigorous activity (defined as a 7 or 8 on a 0 – 10 RPE Borg scale) performed for at least 10 minutes and spread throughout the week⁽⁷⁾. Physical activity can provide an array of health benefits that can be observed in many people, from children to adults, and those with different types of illness and disease. Evidence suggests that regular physical activity can lower the risk of diseases such as heart disease, diabetes, and cancer, as well as increase cardiorespiratory fitness, increase muscular strength, reduce blood pressure, and decrease weight gain⁽⁷⁾. Weight loss of as little as 10 pounds can reduce BP in a large proportion of overweight persons by 5-20 mmHg⁽¹⁾. The ability to maintain a healthy weight range will reduce the incidence of developing health problems, leading to a longer life span.

Aerobic exercise can alter resting BP, which will lead to decreased risks for vascular damage $^{(11)}$. It has been well documented that a single bout of exercise is sufficient to produce a drop in BP, known as post-exercise hypotension $(PEH)^{(1,2,4-25)}$. PEH is a term used to describe the immediate reductions in BP that is noted during the first few hours following acute exercise. Exercise greatly reduces cardiovascular disease via maintaining moderately lower BP and reduced blood cholesterol⁽⁴⁾. As a result, multiple accumulated bouts of acute exercise can produce chronic BP adaptations that reduce the incidence of hypertension. Therefore, many clinicians have studied exercise and hypotension with the goal of regulating arterial pressure and chronic exercise as a non-pharmacologic method of decreasing resting $BP^{(10)}$. In their review, Kenney and Seals note this reduction has been demonstrated in both normotensive and hypertensive men and women in response to a number of large-muscle exercises such as walking, running, and cycling. They also state PEH has been observed in exercises utilizing submaximal intensities between 40 and 70% VO₂max, VO₂peak, age-predicted max HR, or resting HR reserve. As well, these decreased effects have been seen during a wide range of durations; with as little as 3 to 10 minutes up to 170 minutes. In hypertensive individuals, many clinicians have observed PEH to be sustained for a period of 1 to 22 hours following exercise^(2,10,12,13,14). The highest magnitude of BP reduction has been recorded to be as much as 10 mmHg^(5,10). However, a large majority of these effects have been recorded by use of either a running or cycling protocol. To date, none of the

previous investigations have examined if this phenomenon is present while simply walking or jogging at a brisk pace during short duration exercise bouts throughout the day.

Statement of Problem

It is well known that exercise can have positive effects on the body's overall health. A multitude of studies have displayed that the detrimental effects of hypertension can be altered with a single bout of light to high intensities $(40 - 100\% \text{ VO}_2\text{max})^{(2-6,8-25)}$. However, there appears to be an array of differing views when it comes to which intensity produces the greatest BP reductions. As was reviewed previously, PEH, be it great or small in magnitude, may be elicited with any intensity. Whereas the Department of Health and Human Services states that exercise should be performed at least 30 minutes per day, the present study aims at determining whether 30 minutes could be broken into multiple bouts equaling the total required time to produce a drop in blood pressure. To date, no articles reviewed prior to this study have examined the effects of exercise when divided into multiple bouts.

Statement of Purpose

The present study aimed at determining whether or not 30 minutes can be broken into multiple bouts equaling the total required time. Therefore, the purpose of this article was to examine whether a single 10 minute bout of exercise, performed at multiple intervals throughout the day to equal 30 minutes, can effectively elicit PEH. Secondly, it

is important to explore whether a light (40% VO₂R) or moderate (70% VO₂R) intensity will have an impact on the degree of PEH following each of the 10 minute exercise bouts at each of the post exercise time intervals.

Statement of Hypothesis

It is hypothesized that individuals will not elicit PEH after 10 minutes of walking (H_{01}) . Individuals will elicit some degree of PEH after 10 minutes of walking at a brisk pace (H_{A1}) . When comparing intensities, there will be no difference in blood pressure between the light and moderate intensities (H_{02}) . Subjects will show a decrease after the second and third 10 minute sessions at light intensity, but not after the first (H_{A2}) . Subjects will elicit blood pressure decreases after each of the 10 minute sessions at moderate intensity (H_{A3}) . In varying intensity, PEH will be observed after moderate exercise only (H_{A4}) .

In regards to blood pressure measurements, subjects will not exhibit a decrease in systolic or diastolic pressures after 10 minutes of exercising at either intensity (H_{03}). Subjects will show a decrease in systolic pressure after 10 minutes at light intensity (H_{A5}). Individuals will elicit decreases in systolic pressure only after 10 minutes of moderate intensity (H_{A6}). Subjects will show a decrease in diastolic pressure after 10 minutes after 10 minutes of minutes light intensity (H_{A7}). Subjects will show a decrease in diastolic pressure after 10 minutes of moderate intensity (H_{A7}).

Limitations

Limitations to this study include:

Compliance of the subject prior to testing

Performance on VO₂max test

Food and drink intake prior to walking trials

Amount of stress during study

Delimitations

Delimitations include:

Treadmill protocol

Activity levels prior to testing

Definition of Terms

- Post-Exercise Hypotension (PEH) immediate reductions in BP that is noted during the first few hours following acute exercise
- Systolic Blood Pressure (SBP) pressure exerted on the vessels during contraction of the heart
- Diastolic Blood Pressure (DBP) pressure exerted on the vessels during relaxation of the heart
- Hypertension elevated blood pressure with no definite cause

 Blood Pressure (BP) – force exerted by the blood against vessel walls, expressed as systolic/diastolic

Chapter 2

Literature Review

Aerobic exercise training can show reductions in resting BP that are considered to be the result of either reduced peripheral vascular resistance, or decreased cardiac output^(9,15). Typically, researchers have utilized testing methods involving either a treadmill or cycle protocol to elicit PEH. A vast majority of the articles reviewed incorporated a cycle protocol, while only one⁽²⁴⁾ employed a treadmill.

Treadmill/Cycling Protocol

An investigation conducted by Jones et al. examined the effects of PEH at different times of day using either a continuous or intermittent protocol. Eight normotensive participants completed one 30 minute cycling bout and three 10 minute cycling bouts at 70% VO₂peak. The investigators found that BP, mean arterial pressure (MAP), and heart rate (HR) were all lower following intermittent exercise compared to continuous⁽¹⁶⁾. In accordance to Jones, Smelker, et al. observed similar findings in their study. Ten volunteers with stage 1 hypertension (>130/80) were obtained to perform a total of 5 cycle ergometer sessions (1 baseline measurement, and 4 trials). Each session consisted of 120 minutes at 70, 80, 90, and 100% VO₂. Their findings showed a significant decrease in SBP following all four sessions, with PEH being greater following the 100% VO₂ exercise bout⁽¹⁷⁾. Pescatello, et al. also observed BP reduction in their study regarding the influence exercise intensity has on PEH. Forty-nine men with high-normal to stage 1 hypertension (>140/90) participated in 3 exercise bouts consisting of a

control session, light intensity of 40% VO₂max, and a moderate intensity of 60% VO₂max. The participants cycled for 30 minutes, followed by a 45 minute recovery period where SBP and DBP were monitored. An ambulatory cuff was utilized until the following morning. They observed reductions in both SBP and DBP during the 9 hours the cuff was worn. The moderate intensity produced the greatest amount of PEH during the first 5 hours; however, the light intensity was more effective after 9 hours⁽¹³⁾. These findings, while similar to Smelker in regards to eliciting BP reduction, differ in the intensities that produced the greatest effect. Smelker saw the greatest PEH with 100% VO₂max, while Pescatello saw the greatest overall effects with 40%VO₂max. Perhaps the reason for these different findings were that Smelker only observed BP readings for 120 minutes, whereas Pescatello utilized an ambulatory cuff until the following morning. Therefore, the sustained reductions observed could possibly be explained by the activities each subject engaged in throughout the day (i.e. work, exercise).

Cornelissen and Fagard, similarly to Pescatello, performed a study examining the influence of a single bout of exercise using light (40% VO₂ max) and moderate (60% VO₂max) bouts of exercise. They obtained 49 participants with high normal to stage 1 hypertension that completed 3 experiments (control, 2 cycle sessions) lasting 40 minutes followed by a 45 minute recovery period. An ambulatory cuff was placed until the following day on each subject. Their findings showed that the light intensity was as effective in eliciting PEH as the moderate intensity. While the moderate intensity produced a greater amount of PEH after the first 5 hours, the light intensity elicited similar results for the remainder of the day⁽⁸⁾.

Another investigation conducted by Jones et al. utilized 7 normotensive individuals to determine whether variations in dose and intensity alter the effects of PEH. Each participant completed 4 cycle trials of 30 minutes at 70% VO₂peak, 30 minutes at 40% VO₂peak, and a second 40% VO₂peak corresponding to an equivalent time as the intense trial (70%). They observed similar SBP and MAP following the intense and long duration moderate intensity that was greater than the shorter duration moderate intensity. These findings indicated that PEH is different following bouts that differ in intensity and duration, however, not for total work done⁽¹⁸⁾. Conversely, Mach et al. observed that duration affects the amount of PEH. Nine subjects with mild hypertension (>130/85) completed 4 sessions of differing amounts (10, 20, 40, and 80 minutes). Each participant completed an incremental exercise test on a cycle ergometer to obtain VO₂peak, and each session performed at moderate intensity (80% ventilatory threshold). The investigation exhibited that while PEH was observed in some degree with all sessions utilizing moderate intensity, both the 40 and 80 minute bouts produced greater effects in PEH magnitude and duration following a 90 minute recovery $period^{(2)}$.

In a related study, Senitko et al. examined PEH as it relates to endurance trained verses sedentary men and women. A total of 32 participants were placed into 2 groups of 16 endurance trained and 16 sedentary individuals. Each subject completed a 60 minute cycle session at 60% VO₂peak with measurements of arterial pressure and HR, as well as, cardiac output estimation obtained pre and post exercise. Their findings observed larger stroke volume (SV) in the endurance trained individuals. Both groups showed similar amounts of PEH, with MAP, SBP and DBP being lower after exercise. The

experimenters' results suggest that the magnitude of PEH is similar when comparing endurance trained and sedentary men and women⁽¹⁹⁾.

Somers et al. did not come to similar conclusions regarding PEH while observing its ability to persist during the day. They obtained 12 normotensive and 12 mild and borderline hypertensive individuals. Each subject completed a graded maximum exercise cycle test to exhaustion. Blood pressure was measured every 5 minutes for 30 minutes following exercise, and a sphygmomanometer was sent home with each subject to measure BP and HR 3 times every 2 hours. The clinicians observed a fall in BP immediately following exercise that persisted for 1 hour. At 2 hours post exercise, BP was similar to that obtained on the control day. According to Somers et al. the PEH observed is not significant to reduce BP once normal daily activities are resumed, and therefore, cannot explain the sustained PEH exhibited by endurance trained athletes⁽²⁰⁾.

The previous investigations, with the exception of Somers et al., demonstrate that intensity is a major determining factor in PEH. However, it is not the only indicator that PEH will occur in an individual. Some clinicians have focused less on the magnitude of PEH and more on defining predicting factors. Tayler et al. examined the degree to which blood pressure status moderates PEH. The investigators utilized 32 participants that completed a 30 minute cycling bout of exercise at 70% VO₂peak. During the ensuing 20 minute rest period, BP was significantly lower than pre-exercise values. The authors also found that pre-exercise BP, VO₂peak values, and time of day were significant predictors of PEH, with pre-exercise blood pressure being the most significant. A combination of high BP and low VO₂peak led to overall greater reductions, as well as, exercise bouts performed in the afternoon⁽⁵⁾.

Mechanisms of PEH

While the mechanisms of PEH are not fully understood, many theories have attempted to explain the anti-hypertensive effects of exercise. Some believe that factors such as glucose homeostasis, blood lipids and psychobiological processes may play a role in hypotension, whereas others believe this phenomenon is a result of peripheral vascular resistance^(9,14). Hamer noted in his review that chemical mediators have been investigated to explain PEH. Endothelin-1, a vasoconstrictor, has been shown to decrease, while nitric oxide, a vasodilator, has been shown to increase during exercise training. As well, he also noted that studies have also observed larger arterial lumen diameter and greater arterial compliance in endurance-trained individuals when compared to untrained individuals.

In an attempt to gain a greater understanding of its effects on the body, Blanchard et al. examined the influence of PEH on the renin-angiotensin-aldosterone system (RAAS). They studied 47 men with high-normal to stage 1 hypertension (\geq 130/85) over the course of three 40 minute cycling bouts consisting of a control session, 40% VO₂max, and 60% VO₂max. The investigation demonstrated that those with multiple variant RAAS alleles showed greater blood pressure decreases following 40% VO₂max than 60%⁽²¹⁾. Similarly, Hayes et al. compared the changes in plasma volume and albumin following exercise recovery. They utilized 9 normotensive volunteers who performed a 60 minute cycling exercise bout at an intensity corresponding to 65% VO₂peak. The investigators found that hypovolemia occurred immediately after exercise, and then returned to normal levels during 90 minute recovery. Whereas hypovolemia decreases plasma volume, PEH is suggested to induce its re-establishment through increasing

intravascular protein. This process enables fluid to remain in the intravascular space and dilute the increased plasma solutes and sodium⁽²²⁾. Exercise can activate the RAAS, increasing the antidiuretic hormone which produces hypervolemia following an exercise bout. Those possessing more alleles may be more likely to resist these attempts at increasing plasma volume to raise blood pressure. However, this is merely one possible mechanism through which PEH may occur.

The sympathetic nervous system, baroreflex resetting, and histamine receptors have all been associated with vasodilation following an exercise bout. Due to the stimulation of the autonomic nervous system during physical exercise, parasympathetic nerve activity decreases while sympathetic activity increases⁽²³⁾. Therefore, the sympathetic nerve activity has also been examined as a potential mediator in PEH. In a study by Floras et al., 14 men ran on a treadmill at 70% HR reserve for 45 minutes to determine if PEH is associated with decreased sympathetic nerve activity. The clinicians observed significant decreases in SBP after a 60 minute recovery period that was accompanied by lower postganglionic sympathetic nerve activity in muscles⁽²⁴⁾. In a related study, Scott et al. examined the effects of an acute bout of exercise on cardiovascular and cerebral tissue oxygenation responses. The investigators utilized 10 healthy, normotensive males who completed a single session of interval or continuous cycling exercise. The interval session utilized 1 minute workloads of 120% VO₂max interspersed with 4 minute recovery periods of 40% VO₂max for a total of 12 reps and 60 minutes. The continuous session included 90% VO₂max for 60 minutes. They observed that, in addition to decreased MAP and total peripheral resistance (TPR), PEH does not alter cardiovascular and cerebral tissue oxygenation response⁽²³⁾.

Although the exact physiological aspects of PEH are debatable, what is known is arterial blood pressure is a function of cardiac output and TPR, and any observed reduction would be attributable to decreases in cardiac output, TPR, or both^(10,19). During a study attempting to investigate the occurrence of PEH mechanisms, Dujic et al. obtained 20 professional soccer athletes who performed a maximal exercise test. Each participant cycled at a speed of 8 km/h that increased by 1 km/h every minute until exhaustion while monitoring HR and spiroegometry. SBP and DBP were monitored at 30 and 60 minutes post exercise. Significant decreases in SBP, DBP and MAP at both 30 and 60 minutes were observed, with the greatest decrease being at 60 minutes. Their main findings showed that PEH is less in subjects with higher VO₂max values; PEH is seen after brief maximal exercise; PEH may occur more frequently in those with higher baseline SBP; and the mechanisms causing PEH are different for trained individuals than untrained, with PEH in the trained individuals being a result of reduced cardiac output caused by reduction in SV and cardiac filling⁽¹⁴⁾.

Senitko et al. claim that PEH is achieved through different methods in endurance trained men and women. As their findings were reviewed above, their explanation of PEH occurrence is warranted here. In endurance trained women, the clinicians attributed PEH to vasodilation. However, in endurance trained men, PEH was a result of reduced cardiac output due to no change in TPR. Their claims were based on the observation that endurance trained women did not exhibit the same fall in SV and cardiac output observed in endurance trained men⁽¹⁹⁾.

Conclusion

It is well known that exercise can have positive effects on the body's overall health. A multitude of studies have displayed that the detrimental effects of hypertension can be altered with a single bout of light to high intensity (40 - 100% VO₂max) exercise. However, there appears to be an array of differing views when it comes to which intensity produces the greatest BP reductions. As was reviewed previously, PEH, be it great or small in magnitude, may be elicited with any intensity.

Chapter 3

Methodology

Subjects

A power analysis was performed based on the study by Floras et al⁽²⁴⁾. It was determined that a total of 11 subjects were needed (effect size = 0.643, alpha = 0.05, beta = 0.80). A total of 13 subjects between the ages of 18 and 30 were recruited from the campus of Western Kentucky University, and the surrounding Warren County. One subject was unable to complete the required sessions due to time conflicts, and a second was dropped as a result of faulty data. Therefore, a total of 11 subjects completed the trials. Subjects were free of any current or prior cardiovascular disorders, were not currently on hypertensive medications, had no lower extremity orthopedic problems (sprains, breaks), diabetes, high cholesterol, or used tobacco in the past 6 months. Each subject completed a Medical History, Physical Activity Readiness Questionnaire (PARQ), and an informed consent form (See Appendix A, B, and C).

Instruments

Heart rate (HR) in beats/minute was obtained utilizing a wireless Polar Monitoring System (Woodbury, NJ). A heart rate monitor was placed around each subject's chest at just below the pectoralis major and secured in place with an elastic strap. The receiver was a wrist watch that the subjects placed on their non-dominant arm during the resting measurements only, while a second was used during the exercise sessions that had been secured to the treadmill. Blood pressure (BP) in mmHg was measured using a manual blood pressure cuff. A cuff was placed around the subjects left arm, over the brachial artery, just above the cubital fossa of the elbow. Pressure of the blood pressure cuff was increased to 200 mmHg, then decreased slowly to receive an accurate measurement. The same clinician measured blood pressure for each subject, as to reduce the incidence of inter-clinician error.

A Borg rating of perceived exertion scale (RPE) was utilized to determine a subjective level of exertion during the GXT (See Appendix D). Prior to the GXT, each subject received standard instructions on RPE scaling procedures. These procedures included: a definition of RPE, and scale instructions. The investigator first read the definition of RPE: *The perception of physical exertion is defined as the subjective intensity of effort, strain, discomfort, and/or fatigue that you feel during exercise.*

A Parvomedics Metabolic Systems VO₂ Analyzer (Utah) obtained oxygen consumption for all sessions. The analyzer was also attached to and controlled a Quinton Q-stress treadmill (Bothell, WA) that was utilized for both the GXT and walking trials.

Design

Prior to beginning, each subject was taken into the Exercise Physiology Laboratory to explain the nature of the study and what is to be expected. A total of three days were used for each subject. On day 1, each participant was given an informed consent form that was discussed aloud and signed. Each participant was equipped with a Polar Heart Rate Monitor that was worn during the familiarization session. At the end of this session, each participant rested for at least 5 minutes in an armed chair where resting BP and HR were recorded. Measurements in BP were collected manually using a blood pressure cuff. After each measurement was obtained, subjects completed a VO₂max treadmill test utilizing the Bruce protocol on a Quinton treadmill (See Appendix D). At the completion of the GXT, each subject was seated in order for BP measurements to be taken at 3 different intervals; immediately post exercise (\emptyset), 20, and 60 minutes post exercise. During this time, each participant was seated upright with back against the back rest, and arms placed on each arm rest. They sat quietly in the chair, and every attempt was made to refrain from talking to others for 60 minutes. If desired, a book or magazine could be read. Instructions were given to refrain from any tobacco or caffeine use 48 hours before reporting for the other exercise trials.

For Day 2 of the investigation, the participants reported back to the lab within the ensuing week (3 - 5 days after the GXT) for the exercise trial. A total of three sessions were completed: one in the morning (AM), one around noon (Noon), and one in the evening (PM). Each session was separated by 3-4 hours. Again, instructions were given to each participant regarding their involvement. A counter-balance design was utilized to eliminate the possibility of intensity order having an influence on the results. Even numbered subjects utilized 70 and 40% VO₂R on day 1 and day 2, respectively, whereas odd numbers utilized a 40 and 70% VO₂R day 1 and day 2, respectively. At the beginning of each session, participants were instructed to walk on a treadmill at a pace that elicited the prescribed percent of their VO₂R (based on the counter-balance) for a period of 10 minutes. A titration period of 3 minutes allowed the investigator to titrate

the speed of the treadmill to elicit the required intensity prior to starting the 10 minute bout. During exercise, treadmill grade was set to 1% to mimic normal terrain conditions and VO_2 was monitored and the speed of the treadmill was altered to maintain specific range. Once the participant had finished his/her 10 minutes, he/she was seated in order for BP measurements to be taken. After the morning and noon sessions (AM and Noon), BP was obtained at Ø and 20 minutes post exercise (AM 20 and Noon 20). At the completion of the evening session, BP was measured at Ø, 20, and 60 minutes post exercise (PM 20 and PM 60). BP was obtained in the same manner as described above for each time period.

The third session was conducted using the same methodology as the second data collection period. Intensity for each participant was prescribed according to counter-balance design for Day 3. Time of day for each testing session was kept similar for each participant as the second day, and BP was measured at the same intervals post-exercise.

Data Analysis

Descriptive data for anthropometric and physiological variables were calculated as mean \pm standard deviation (SD). All analyses were performed using the Statistical Package for the Social Sciences (SPSS, version 18.0, Chicago, Ill., USA). Statistical significance were set at an alpha \leq 0.05 level for all analyses (See Appendix E, Table 3.1).

An analysis of variance (ANOVA) with Repeated Measures was used to determine differences between the initial resting BP and the BP measurements at each of the post exercise periods following the 10 minute exercise bouts at 40% VO₂R. The

repeated measures analysis performed compared resting baseline BP with AM 20, Noon 20, PM 20 and PM 60 minute post-exercise measurements. Post hoc analysis for the multiple comparisons were analyzed using the LSD post hoc procedure.

An analysis of variance (ANOVA) with Repeated Measures was used to determine differences between the initial resting BP and the BP measurements at each of the post exercise periods following the 10 minute exercise bouts at 70% of VO₂R. The repeated measures analysis performed compared the resting baseline BP with AM 20, Noon 20, PM 20 and PM 60 minute post-exercise measurements. Post hoc analysis for the multiple comparisons were analyzed using the LSD post hoc procedure

A 2 x 2 (time x intensity) mixed analysis of variance (ANOVA) with repeated measures on the first factor was used to examine the 40% VO₂R sessions compared to the 70% VO₂R sessions at each post exercise time point (AM 20, Noon 20, PM 20, and PM 60). Post hoc analysis for the multiple comparisons were analyzed using the LSD post hoc procedure.

Chapter 4

Results

*40% VO*₂*R*

Baseline AM Systolic BP was compared to each 20 minute post-exercise (AM 20, Noon 20, PM 20) BP, and the PM 60 minute post exercise BP measurements at 40% VO₂R. A significant main effect was found F(4,40 = 4.855, p \leq 0.05) (See Appendix F, Table 4.1). Post hoc analysis indicated that SBP significantly decreased from baseline BP (mean \pm SD = 109.8 \pm 7.2) at AM 20 minutes post-exercise (104.7 \pm 7.4), Noon 20 minute post-exercise (105.6 \pm 5.9) and PM 20 minute post exercise (102.1 \pm 10.2) (See Appendix G, Figure 4.1). There was no difference between baseline (109.8 \pm 7.2) and 60 minute post exercise (106.1 \pm 6.4).

Baseline AM Diastolic BP was compared to each 20 minute post-exercise BP and the PM 60 minute post exercise BP measurements at 40% VO₂R. There was no significant main effect noted F(9,90 = 4.555, p > 0.05). Baseline BP (75.4 ± 5.2) remained at similar levels at the AM 20 minute (74.0 ± 7.4), Noon 20 minute (72.3 ± 6.8), PM 20 minute (75.6 ± 7.8), and PM 60 minute post-exercise (76.3 ± 7.2) BP measurements (p = 0.316), (p = 0.053), (p = 0.926), (p = 0.612) respectively. (See Appendix H, Figure 4.2)

70% VO₂R

Baseline AM Systolic BP was compared to each 20 minute post exercise (AM 20, Noon 20, PM 20) BP and the PM 60 minute post exercise BP measurement at 70% VO₂R. A significant main effect was observed F(4,4.416, $p \le 0.05$). Post hoc analysis

indicated that SBP significantly decreased from baseline BP (108.5 ± 8.5) at AM 20 minute post (104.9 ± 8.5), Noon 20 minute (104.3 ± 8.4) and PM 60 minute post-exercise measurements (102.0 ± 7.2). There was no significance at PM 20 minute post-exercise (105.2 ± 8.4) compared to baseline (Appendix I, Table 4.2). Additionally, a statistically significant decrease was noted between PM 20 and PM 60 minute post-exercise (p = 0.022) showing a decrease from 105 mmHg to 102 mmHg (See Appendix J, Figure 4.3).

Baseline AM Diastolic BP was compared to each 20 minute post-exercise BP and the PM 60 minute post exercise BP measurements at 70% VO₂R. There was no significant main effect noted F(9,90 = 4.555, p > 0.05). DBP was not significantly different at AM 20 minute (74.9 ± 7.0), Noon 20 minute (74.5 ± 7.0), PM 20 minute (74.9 ± 5.6), or PM 60 minute (74.9 ± 6.6) post-exercise BP measurements when compared to baseline (74.9 ± 7.0) (See Appendix K, Figure 4.4).

40% VO₂R vs. 70% VO₂R

A comparison between exercise intensities was made to determine whether or not a there was an intensity effect associated with the decreased blood pressure. A significant main effect was found for time F(4,80 = 6.846, p < 0.05). In addition, there was no significant interaction (time x intensity) found F(4,80 = 2.476, p > 0.05). SBP at each time point was analyzed between intensities (See Appendix L, Table 4.3). At morning baseline, resting blood pressure was no different at 40% VO₂R (109.8 ± 7.2) than at 70% (108.5 ± 8.2). This comparison indicates that each subject began both sessions with similar measurements. In regards to each sessions effect, the analysis showed no significant difference between intensities to produce a greater amount of PEH at any time point. At AM 20 minute post-exercise, no difference was noted between 40% and 70%. The Noon 20 minute and both PM post-exercise sessions (20 minute and 60 minute) showed similar results. No significant difference was observed between Noon 40% and Noon 70%. For both evening sessions, 40% PM 20 minute and PM 60 minute were not significantly different compared to 70% PM 20, or PM 60 minute post-exercise measurements.

Chapter 5

Discussion

This study examined the effects of single and multiple 10 minute bouts of exercise and varying intensities, on BP. The main findings of the investigation were that (1) PEH can be elicited at 40% and 70% intensity after 10 minutes of exercise on a treadmill, (2) though not all were statistically significant, SBP decreased from resting baseline after each session, (3) there was a cumulative effect with each intensity for SBP to decrease throughout the day, and (4) though there was no statistical difference, 70% VO_2R appeared to reduce SBP to a greater extent after the 60 minute measurement compared to 40% VO_2R .

After a single bout of 10 minutes of exercise at 40% VO₂R, SBP was significantly decreased at AM 20 minutes, Noon 20 minutes, and PM 20 minutes post-exercise measurements. At 60 minutes post-exercise measurement, SBP appeared to be returning towards the baseline resting measurement. Although not significant, SBP continued to be lower than baseline at this particular time point. These findings of decreased SBP after a 40% bout of exercise are in accordance with Pescatello et al.⁽¹³⁾, Cornelissen and Fagard⁽⁸⁾, Jones et al.⁽¹⁶⁾, and Blanchard et al.⁽²¹⁾. All reported a decrease in SBP after a 40% VO₂ exercise bout. These findings indicate that exercising at 40% VO₂ (which equated to a brisk walk for most individuals) is sufficient at eliciting a drop in SBP, even after a single bout of 10 minutes.

After the first session of 10 minutes at 40%VO₂R, SBP decreased from 109 mmHg to 104 mmHg (a difference of 5 mmHg). The noon session showed a slight increase from the morning session at 105 mmHg, yet still a decrease from morning

baseline. At the evening 20 minute mark, SBP had again decreased to 102 mmHg, and then showed a return towards baseline at 60 minutes after the evening session to 106 mmHg. In similar fashion, Somers et al. observed that a reduction in BP after exercise was only sustained for merely 1 hour post-exercise. After 2 hours BP returned to resting values. Although not significant after the evening ten minute bout, SBP was still lower at 60 minutes post-exercise than the morning baseline (Appendix G). This indicates that a continual decrease was seen from morning baseline to the evening showing an acute effect of SBP after each session. Therefore, it can be reasonably assumed that at the end of the day and after the last 10 minute exercise session has been performed, one may see a lower BP measurement from morning.

Similarly to the 40% VO₂R sessions, a reduction in SBP was noted after 10 minutes of exercising at 70% VO₂R. Likewise, this reduction continued late in the evening with even greater effects than the morning session. These findings are in accordance with Jones et al.^(16, 18), Smelker et al.⁽¹⁷⁾, Taylor et al.⁽⁵⁾, and Floras et al.⁽²⁴⁾. These articles reported a decrease in SBP for the first few hours after exercising at 70% VO₂. The findings of this study, along with others, indicate that 70%VO₂R is a sufficient intensity to elicit a drop in SBP after a single 10 minute session of exercise on a treadmill.

Although not statistically different in this study, SBP decreased from baseline at each time point after 10 minutes of exercise at 70% VO₂R. During the morning session, a decrease in SBP of 4 mmHg (from 108 to 104 mmHg) was observed from resting baseline to 20 minutes after exercising. For the noon session, there was a similar SBP measurement at 20 minutes post-exercise as the morning at 104 mmHg. There was a

slight increase from 104 mmHg to 105 mmHg at the evening 20 minute post-exercise that can be attributed to activities of daily living (ADL). However, at 60 minutes postexercise in the evening, SBP decreased further by a degree of 6 mmHg (from 105 mmHg to 102 mmHg) (Appendix J). This session, similarly to the 40% VO₂R session, showed a gradual decrease in SBP throughout the day. The last time point (60 minutes postexercise) should be noted, as it is an extremely important measurement that will be discussed below.

The increases that were observed during the day for both intensities (noon session for 40%, and evening session for 70%) can be attributed to activities of daily living (ADL). All subjects were college aged students that generally had class between each session. Each participant's time of exercising were kept the same for both sessions, which limited the likelihood for BP variation at each session from the previous testing day. The activities that were engaged in by each participant were not able to be controlled for, and therefore, are more likely a determining factor for the observed increase.

When comparing 40% to 70% VO₂R, SBP decreases were similar with both intensities at each time point. Both sessions showed a generalized trend for blood pressure to decrease after a single 10 minute bout, as well as multiple exercise sessions, regardless of intensity. Statistically, neither intensity was more effective at eliciting PEH over the other. One would expect to find that a higher intensity (70% for this study) would show a larger decrease in BP simply as a result of the body having to work harder. After exercise, a period of oxygen debt (exercise post oxygen consumption or EPOC) is observed where the vascular system dilates to restore proper levels of oxygen. The more

strenuous the exercise, the greater amount of EPOC produced. Exercising at 70% VO₂R should exhibit a greater decrease in BP when compared to 40% VO₂R. Interestingly enough, that appeared not to be the case from a statistical standpoint. This analysis indicated that SBP continued a downward trend at 60 minutes post-exercise. Secondly, SBP did not begin its return towards baseline signifying a cumulative effect. The finding at 60 minutes post-exercise during the 70% evening session of SBP to decrease instead of increase, as was seen in the 40% evening session, shows that intensity may be a determining factor in the amount of overall decrease throughout the day (Appendix M). This finding is in similar accordance to Jones⁽¹⁸⁾, Smelker⁽¹⁷⁾, Taylor⁽⁵⁾, and Dujic⁽¹⁴⁾ in that 70% VO₂R produces a greater reduction in SBP than a lower intensity (in the case of this study, 40% VO₂R).

Opposed to these findings were the results noted by Pescatello et al.⁽¹³⁾, Cornelissen and Fagard⁽⁸⁾, and Blanchard et al.⁽²¹⁾. These investigators noted that exercising at 40% VO₂ resulted in a longer lasting PEH effect when compared to a higher intensity. At 40% VO₂R, SBP decreased from a morning resting baseline of 109 mmHg to 106 mmHg 60 minutes after the evening exercise session. During the 70% VO₂R session an observed decrease from 108 mmHg morning resting SBP measurement to an evening 60 minute post-exercise SBP of 102 mmHg was observed. It is clear that the results of this study indicated that, while 3 sessions of 10 minute treadmill exercise at 40% VO₂R is sufficient at producing PEH, the effects appear to be mediated to a greater extent in both magnitude and duration after 3 sessions of 70%VO₂R at the end of the day.

While this study showed a decrease in SBP, there were some differences that should be noted. First, other studies have utilized a cycle ergometer as their mode of

exercise while this study used a treadmill. The differences noted in SBP may be different when comparing modes of exercise. Secondly, the length of exercise was different between the studies (30 and 40 minutes vs. 10 minutes in the present study). As to date, no studies have evaluated the effectiveness of 30 minutes versus 10 minutes of treadmill exercise. It may be likely that 30 minutes of continuous exercise on a treadmill at any intensity would elicit either greater or longer duration PEH than a single 10 minute or multiple 10 minute bout(s) of exercise. Although Floras et al. (1989) utilized a treadmill in their study of PEH, the intensity employed was mediated by a different factor (HRR vs. VO_2R in the present study). Therefore, the results of this study could not be compared to identify whether length of treadmill exercise is a determining factor of PEH magnitude and duration. Lastly, the length of time BP was measured post-exercise in the current study (20 minutes for the morning and noon sessions, and 1 hour for the evening session) differed from time points in other studies (≥ 2 hours). While other studies measured BP until the following day, the investigators of this study were more concerned with whether or not 10 minutes is enough to elicit PEH and what intensity is required to do so. As this study obtained college aged normotensive individuals, it was impractical to expect participants to sit quietly for multiple hours, nor were ambulatory cuffs readily available. Therefore, BP results may have been different if measurements were observed over a longer period of time (2 hours or more vs. 20 and 60 minutes). As well, those with higher BP may exhibit greater reductions in SBP or some decrease in DBP. The study performed by Taylor et al. exemplifies this as a result of the observed finding that preexercise BP was a significant indicator in the amount of PEH mediated by exercise.

For DBP during each of the sessions, no significance was noted between any of the sessions. These findings are in opposition with other studies that showed DBP was also decreased after exercise. The exact reasoning for this finding could not be elicited with the data collected. It is possible that the 40% intensity was too light to produce a drop in the DBP. However, other studies have observed these decreases at 40% utilizing a different mode of exercise. As well, 70% did not decrease DBP either, making it less likely that the intensity was the determining factor. Another possibility for the absence of a decrease is that the duration chosen for this study may have not been long enough. Contrary to this, other studies have employed a 10 minute bout resulting in a decrease of DBP. Lastly, it is possible that the mode was not sufficient enough. A large majority of studies that have observed DBP decreases reviewed in this study used a cycle ergometer to perform exercise.

Future studies should evaluate whether the mode of exercise (treadmill vs. cycle), duration of treadmill exercise (30 minutes continuous vs. multiple 10 minute bouts), and if the effects of PEH after exercising on a treadmill are evident past 60 minutes postexercise in both duration and magnitude.

Conclusion

The purpose of this investigation was to examine the effects of single 10 minute bout of exercise, performed at multiple intervals throughout the day to equal 30 minutes, can effectively elicit PEH and at what intensity is required to do so. The main findings of the investigation were that (1) PEH can be elicited at 40% and 70% intensity after 10 minutes of exercise on a treadmill, (2) though not all were statistically significant, SBP decreased from resting baseline after each session, (3) there was a cumulative effect with

each intensity for SBP to decrease throughout the day, and (4) though there was no statistical difference, 70% VO₂R appeared to reduce SBP to a greater extent after the 60 minute measurement compared to 40% VO₂R. Although these results can only be generalized to the college aged population, it can be reasonably assumed that those with BP measurements at the pre-hypertension level and higher may observe some benefit from exercising at a comfortable level of low intensity. Furthermore, those individuals who have a decreased exercise capacity can reduce overall resting BP while working towards longer duration exercise. Still, further examination is warranted to determine whether treadmill exercise is as effective as other modes in longer durations.

APPENDIX A

INFORMED CONSENT DOCUMENT

Project Title: Post-Exercise Hypotension in Brief Exercise

Investigator: Jeremiah Bush, Kinesiology, Recreation, & Sport, 276-219-2827 (include name, department and phone of contact person)

You are being asked to participate in a project conducted through Western Kentucky University. The University requires that you give your signed agreement to participate in this project.

The investigator will explain to you in detail the purpose of the project, the procedures to be used, and the potential benefits and possible risks of participation. You may ask him/her any questions you have to help you understand the project. A basic explanation of the project is written below. Please read this explanation and discuss with the researcher any questions you may have.

If you then decide to participate in the project, please sign on the last page of this form in the presence of the person who explained the project to you. You should be given a copy of this form to keep.

1. Nature and Purpose of the Project:

The purpose of this investigation is to examine the effectiveness of 10 minute bouts of exercise at varying intensities in eliciting post-exercise hypotension.

2. **Explanation of Procedures:**

If you decide to take part in this research study, you will complete 3 separate study sessions separated by a 1 week period. For each session, you will report to the Exercise Physiology Laboratory at Western Kentucky University. To minimize risks, you will be asked to complete a Physical Activity Readiness Questionnaire (PAR-Q) and a medical history form which asks questions about your current health status. If you have an orthopedic (muscle or bone), cardiovascular (Heart), and/or metabolic disease (i.e. coronary artery disease (Heart Disease), prior myocardial infarction (Heart Attack), peripheral vascular disease (Blockages in legs), chronic obstructive pulmonary disease (Lung disease), Stage 1 hypertension (blood pressure of 140-159/90-99 mmHg) or higher, and diabetes mellitus (High/low Blood sugar) you will be excluded from participation in this research study.

Procedures:

1). Blood pressure will be taken with a standard manual cuff.

2). A heart rate monitor will be placed around your chest and secured in place with an elastic strap to determine your heart rate when exercising.

3). You will be asked to rate your level of exertion during exercise testing.

4). On day one of the study, you will be asked to walk and run on a treadmill while wearing a mask to measure your oxygen consumption. The speed will begin at 3 MPH and increases every three minutes. At the completion of the exercise, blood pressure will be measured at 5, 20, and 60 minutes post exercise.

5). On the second day of the study, you will be asked to report to the laboratory for three separate sessions: morning, noon, evening. For each session, you will be asked exercise on a treadmill for 10 minutes at a pace that elicits 40% of your exercise capacity. After the morning and noon sessions, blood pressure will be measured at 5 and 20 minutes post exercise. At completion of the evening session, blood pressure will be measured at 5, 20, and 60 minutes post exercise.

6). On the third day the study, you will be asked to report to the laboratory for three separate sessions: morning, noon, evening. For each session, you will be asked exercise on a treadmill for 10 minutes at a pace that elicits 70% of your exercise capacity. After the morning and noon sessions, blood pressure will be measured at 5 and 20 minutes post exercise. At completion of the evening session, blood pressure will be measured at 5, 20, and 60 minutes post exercise.

3. **Discomfort and Risks:**

Risk associated with the use of a heart rate monitor and blood pressure cuff include redness, irritation, and chafing. The risk of an incident during the graded exercise test and the 10 minute bout is low however; there are potential risks such as: cardiovascular injury (heart attack or stroke), severe acute fatigue, lightheadedness, dizziness and death.

4. **Benefits:**

You will learn about your resting blood pressure, heart rate and aerobic capacity. As well, you will gain the knowledge of how to maintain a healthy blood pressure to reduce the future risk of medical problems associated with high blood pressure. Pending research grant approvals, subject may receive some monetary benefit for time and effort.

5. **Confidentiality:**

Any information about you obtained from this research will be kept confidential. All records related to your involvement in this research study will be stored in a locked file cabinet. Your identity on these records will be indicated by a case number rather than by your name, and the information linking these case numbers with your identity will be kept separate from the research records. You will not be identified by name in any publication of the research results.

6. **Refusal/Withdrawal:**

Your refusal to participate in this study will have no effect on any future services you may be entitled to from the University. Anyone who agrees to participate in this study is free to withdraw from the study at any time with no penalty.

You understand also that it is not possible to identify all potential risks in an experimental procedure, and you believe that reasonable safeguards have been taken to minimize both the known and potential but unknown risks.

Signature of Participant	Date
Witness	Date
THE DATED APPROVAL ON THIS CON THIS PROJECT HAS BEEN REVIE	NSENT FORM INDICATES THAT

THE WESTERN KENTUCKY UNIVERSITY INSTITUTIONAL REVIEW BOARD Paul Mooney, Human Protections Administrator

TELEPHONE: (270) 745-4652

APPENDIX B

Exercise Physiology Laboratory

Physical Activity Readiness Questionnaire (PAR-Q)

Now I am going to ask you a few questions to determine if you are eligible to participate in the study.

1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

No Yes If yes, specify:

2. Do you feel pain in your chest when you do physical activity?

No ____ Yes ____ If yes, specify: _____

3. In the past month, have you had chest pain when you were not doing physical activity?

No ____ Yes ____ If yes, specify: _____

4. Do you lose your balance because of dizziness or do you ever lose consciousness?

No ____ Yes ____ If yes, specify: _____

5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?

No Yes If yes, specify:

6. Is your doctor currently prescribing drugs (for example, water pills) for a blood pressure or heart condition?

No Yes If yes, specify:

7. Do you know of any other reason why you should not do physical activity?

No ____ Yes ____ If yes, specify:_____

APPENDIX C

Exercise Physiology Laboratory

MEDICAL HISTORY

Now I am going to ask you a few questions to determine if your health status ...

		YES	NO
1.	History of heart problems, chest pain, or stroke?		
2.	Increased blood pressure?		
3.	Any chronic illness or condition?		
4.	Difficulty with physical exercise?		
5.	Advice from a physician not to exercise?		
6.	Recent surgery? (Last 12 months)		
7.	Pregnancy? (Now or within the last 3 months)		
8.	History of breathing or lung problems?		
9.	Muscle, joint, back disorder, or any previous injury still affecting		
	you?		
10.	Diabetes or thyroid conditions?		
11.	Do vou use tobacco (any form)?		
12.	Increased blood cholesterol?		
13.	History of heart problems in your immediate family?		
14	Are you currently on any blood pressure medication?		
15	Do you have any condition limiting your movement?		
16	Are you aware of being allergic to any drugs or insect hites?		
17	Do you have asthma?		
18	Do you have enilensy convulsions or seizures of any kind?		
10.	Do you follow any specific diet?		
19.	bo you follow any specific diet:		

Please explain in detail any "YES" answers:

Family History

Has any member of you family had any of those listed above?

APPENDIX D

Bruce Treadmill Test

Borg RPE Scale

	0⁄0			Perceived
			Scale	Exertion
Minute	MPH	Grade	6 7 8	Very, very light
		1.0	9	Very light
0	1.7	10	10	
3	2.5	12	11 12	Fairly light
			13	Somewhat hard
6	3.4	14	14	
			15	Hard
9	4.2	16	16	
			17	Very hard
12	5.0	18	18	
			19	Very, very hard
15	5.5	20	20	Maximum exertion

APPENDIX E

Table 3.1

Subjects	Male = 4		
	Female = 7		
Height (in)	66.08 <u>+</u> 2.8		
Weight (lbs)	150.18 <u>+</u> 34.2		
Age (yrs)	21.18 <u>+</u> 0.8		
VO2max ml/kg/min	48.83 <u>+</u> 7.0		
GXT Resting Systolic (mmHg)	110.3 <u>+</u> 11.0		
GXT Resting Diastolic (mmHg)	73.63 <u>+</u> 7.7		

APPENDIX F

Table 4.1

ANOVA for 40% VO₂R

Interval	df Ol	bserved	FS	Significance
		Power		
AM Resting	4	0.933	4.855	
AM 20 min Post				0.007*
Noon 20 min				0.018*
Post				0.010*
PM 20 min Post				0.101
PM 60 min Post				

* Denotes a significance level of $p = \le 0.05$

APPENDIX G

Figure 4.1

Systolic Blood Pressure pre and post 10 minutes of 40% VO₂R exercise



* Denotes significance of $p = \le 0.05$

APPENDIX H

Figure 4.2

Diastolic Blood Pressure pre and post 10 minutes of 40% VO₂R exercise



APPENDIX I

Table 4.2

ANOVA for 70 % VO₂R

Interval	df Observed		F Si	gnificance
		Power		
AM Resting	4	0.906	4.416	
AM 20 min Post				0.029*
Noon 20 min post				0.050*
PM 20 min Post				0.121
PM 60 min Post				0.006*

* Denotes a significance level of $p = \le 0.05$

APPENDIX J

Figure 4.3

Systolic Blood Pressure pre and post 10 minutes of 70% VO₂R exercise



* Denotes significance of $p = \le 0.05$

APPENDIX K

Figure 4.4

Diastolic Blood Pressure pre and post 10 minutes of 70% VO₂R exercise



APPENDIX L

Table 4.3

ANOVA for 40% and 70% VO_2R

df Ob	served	F	Significance
	Power		
4	0.682	2.476	
			0.494
			0.913
			0.439
			0.243
			0.122
	df Ob	df Observed Power 4 0.682	df ObservedFPower440.6822.476

APPENDIX M

Figure 4.5

40% versus 70% Systolic Blood Pressure pre and post 10 minutes



BIBLIOGRAPHY

- 1. Chobanian, Aram V. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. NIH Publication; 2004.
- 2. Mach, C. et. al. *Effect of Exercise Duration on Postexercise Hypotension*. Journal of Cardio Pulmonary Rehabilitation. 2005; 25: 366-369.
- 3. Anderson, D; Novak, P; Elliot, M. *Mosby's Medical, Nursing, & Allied Health Dictionary: Sixth Edition.* St. Louis, Missouri: Mosby, Inc; 2002: 849-850.
- 4. Hall, J; Guyton, A. *Guyton and Hall Textbook of Medical Physiology: Twelfth Edition.* Philadelphia, Penn: Saunders; 2011: 218-229, 1041.
- 5. Tayler, C. E. et. al. *Blood Pressure Status and Post-Exercise Hypotension: An Example of Spurious Correlation in Hypertension Research?* Journal of Human Hypertension. 2010; 1-8.
- 6. Simao, R. et. al. *Effects of Resistance Training Intensity, Volume, and Session Format on the Postexercise Hypotensive Response.* Journal of Strength and Conditioning Research. 2005. 19(4): 853–858
- 7. Leavitt, M. 2008 Physical Activity Guidelines for Americans. Dept. of Health and Human Services. 2008. Available at: http://www.health.gov/paguidlines.
- 8. Cornelissen, V; Fagard, R. *Exercise Intensity and Postexercise Hypotension*. Journal of Hypertension. 2004; 22: 1859-1861.
- 9. Hamer, M. The Anti-Hypertensive Effects of Exercise: Integrating Acute and Chronic Mechanisms. Sports Med 2006; 36 (2): 109-116.
- 10. Kenney, M; Seals, D. Postexercise Hypotension: Key Features, Mechanisms, and Clinical Significance. Hypertension. 1993. 22; 653-664.
- Boroujerdi, S; Rahimi, R.; Noori, S. Effect of High Versus Low Intensity Resistance Training on Post-Exercise Hypotension in Male Athletes. International Sports Medicine Journal. 2009. 10 (2): 95-100.
- Fagard, R; Vanhees, L. Twenty-four Hour Blood Pressure After Exercise in Patients with Coronary Artery Disease. Journal of Human Hypertension. 2000. 14: 231-234.

- 13. Pescatello, L. et. al. *Exercise Intensity Alters Postexercise Hypotension*. Journal of Hypertension. 2002. 22: 1881-1888.
- 14. Dujic, Z. et. al. *Postexercise Hypotension in Moderately Trained Athletes After Maximal Exercise*. Medicine & Science in Sports & Exercise. 2006: 318-322.
- 15. Rezk, C. et. al. *Post-Resistance Exercise Hypotension, Hemodynamics, and Heart Rate Variability: Influence of Exercise Intensity.* European Journal of Applied Physiology. 2006. 98:105-112.
- 16. Jones, H. et. al. *Post-Exercise Blood Pressure Reduction is Greater Following Intermittent than Continuous Exercise and is Influenced Less by Diurnal Variation.* Chronobiology International. 2009. 26(2): 293–306.
- 17. Smelker, C. et. al. *Effect of Exercise Intensity on Postexercise Hypotension*. Journal of Cardiopulmonary Rehabilitation. 2004. 24: 269-273.
- Jones, H. et. al. Is the Magnitude of Acute Post-Exercise Hypotension Mediated by Exercise Intensity or Total Work Done? European Journal of Applied Physiology. 2007. 102: 33–40.
- 19. Senitko, A; Charkoudian, N; Halliwill, J. *Influence of Endurance Exercise Training Status and Gender on Postexercise Hypotension*. Journal of Applied Physiology. 2002. 92: 2368–2374.
- 20. Somers, V. et al. Postexercise Hypotension is Not Sustained in Normal and Hypertensive Humans. Hypertension. 1991. 18: 211-215.
- Blanchard, B. et. al. RAAS Polymorphisms Alter the Acute Blood Pressure Response to Aerobic Exercise Among Men with Hypertension. European Journal Applied Physiology. 2006. 97: 26–33.
- 22. Hayes, P.M. et. al. Importance of Post-Exercise Hypotension in Plasma Volume Restoration. Acta Physiol Scand. 2000. 169: 115-124.
- 23. Scott, J. et. al. *Post-Exercise Hypotension and Cardiovascular Responses to Moderate Orthostatic Stress in Endurance-Trained Males.* Applied Physiology, Nutrition, & Metabolism. 2008. 33: 246–253.
- 24. Floras, J. et. al. Postexercise Hypotension and Sympathoinhibition in Borderline Hypertensive Men. Hypertension. 1989. 14: 28-35.
- 25. Boone, J. et. al. *Postexercise Hypotension Reduces Cardiovascular Response to Stress.* Journal of Hypertension. 1993. 11: 449-453.