THE EFFECTS OF A LOW VOLUME RECREATIONAL JOGGING PROGRAM ON CARDIOVASCULAR AUTONOMIC CONTROL

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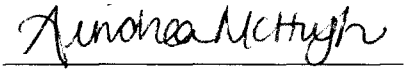

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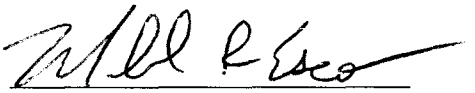

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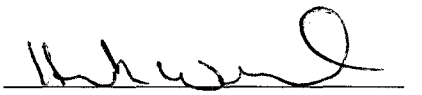

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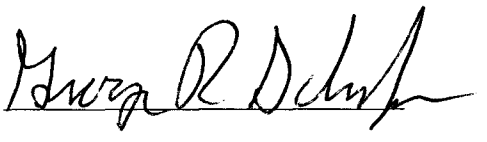

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THE EFFECTS OF A LOW VOLUME RECREATIONAL JOGGING PROGRAM ON CARDIOVASCULAR AUTONOMIC CONTROL

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LIST OF ABBREVIATIONS

BMI	Body Mass Index
ECG	Electrocardiogram
HR	Heart Rate
HRR	Heart Rate Recovery
HRV	Heart Rate Variability
HR1	Heart rate at 1-minute post-maximal exercise
HR2	Heart rate at 2-minutes post-maximal exercise
HRR1	The difference between maximal heart rate and heart rate at 1-min. post-exercise
HRR2	The difference between maximal heart rate and heart rate at 2 min. post-exercise
lnSDNN	Log transformed standard deviation of all the R to R (normal to normal) beats
lnHF	Log transformed high frequency domain
lnLF:HF	Log transformed high frequency to low frequency ratio
MHR	Maximal heart rate
SNDD	Standard deviation of the normal to normal beats
HF	High frequency domain
LF:HF	High frequency to low frequency ration
LF	Low frequency domain

INTRODUCTION

The autonomic nervous system is primarily responsible for cardiovascular activity. Two valid and important measures of cardiovascular autonomic control are heart rate variability (HRV) and heart rate recovery (HRR). The primary importance of HRV and HRR is due to its link to untoward, fatal events as low HRV and slow HRR are associated with morbidity and mortality from cardiac and non-cardiac causes.

There is substantial literature involving the influence of exercise on heart rate variability and heart rate recovery; however, most literature involves highly trained subjects who train on multiple days of the week. These subjects show heightened HRV values, lower resting heart rates, and accelerated recovery of heart rate post-exercise; therefore, they are characteristic of superior cardiovascular autonomic control when compared to sedentary or unfit subjects.

Purpose of Study

There is limited research investigating the effects of a low volume (i.e. <3 days per week) aerobic training program on cardiovascular autonomic control. The purpose of this study was to determine the effect of a two day per week recreational jogging program on indexes of heart rate variability and heart rate recovery.

Hypotheses

It is hypothesized that the jogging program will improve the HRV and HRR profile. There is considerable research associating improved HRV and accelerated HRR after engaging in a high volume aerobic training program. There will be a significant difference between pre- and post- measures of HRV and HRR. There will also be a significant difference in maximal aerobic power (VO_{2max}) and body composition.

CHAPTER 1

REVIEW OF THE LITERATURE

Overview

Cardiovascular activity is largely controlled by the complex interaction of the branches of the autonomic nervous system. In order to understand cardiovascular autonomic control, a brief overview of the autonomic nervous system is required. A discussion of important measures of cardiovascular autonomic control, heart rate variability and heart rate recovery, will also be discussed and literature involving the influence of exercise and body composition on these measures will be reviewed.

The Autonomic Nervous System

The autonomic nervous system, which stems from the peripheral nervous system, is responsible for controlling the functions of the organs. The autonomic nervous system involuntarily controls heart rate, blood pressure, body temperature, sweat output, gastric juice secretion and so on. The autonomic nervous system is comprised of two divisions: the sympathetic division and parasympathetic division. Most organs receive dual innervations, that is, from both divisions. In latent terms, the sympathetic division excites an organ whereas the parasympathetic division tends to inhibit the same organ. Each of these systems secretes neurotransmitters, which are chemicals that activate certain cell receptors of an organ. The sympathetic nerves secrete norepinephrine, which activates

adrenergic receptors, and the parasympathetic nerves secrete acetylcholine, which activates cholinergic receptors. The vagus nerve is primarily responsible for channeling parasympathetic feedback; therefore, parasympathetic modulation is often used interchangeably with the term vagal modulation.

Cardiovascular Autonomic Modulation

Heart rate is involuntarily controlled by the autonomic nervous system. The sympathetic branch is responsible for increasing heart rate whereas the parasympathetic branch is responsible for lowering heart rate. In response to a stimulus, the sympathetic system activates cardiac adrenergic receptors which are responsible for fluctuations in stroke volume, increases in the rate of sinoatrial (SA) node pacing, venous constriction and cardiac contractility. Conversely, the parasympathetic system inhibits those functions when the stimulus is removed. The parasympathetic division activates cholinergic receptors which are responsible for decreasing the rate of sinoatrial node pacing, decreasing the rate of conduction and decreasing the heart's contractility.

Heart rate variability (HRV) and heart rate recovery (HRR) are two important measures of cardiac autonomic control. HRV is the rhythmic time-related changes in successive heart beats, or R-R intervals, on an electrocardiogram recording (EKG) where as, HRR is the return of heart rate towards baseline immediately following exercise. Analysis of HRV and HRR can provide professionals with important information about autonomic control of the cardiovascular system.

Cardiovascular Adjustments during Exercise

During exercise, the autonomic nervous system is responsible for meeting the increased cardiac output demand placed on the heart by skeletal musculature. At the onset of exercise, heart rate is increased due to amplified sympathetic activity and reduced parasympathetic activity. Reduced parasympathetic activity is primarily responsible for increasing HR up to approximately 110 beats per min. After that point, the further increase in HR is due to amplified sympathetic activity. Subsequently, stroke volume is increased to meet the demands of working muscles. At the cessation of exercise, recovery of heart rate is a function of vagal reactivation first with decreasing sympathetic activity occurring at later stages of recovery (Cole, Blackstone, Pashkow, Snader, & Lauer, 1999).

Heart Rate Variability

Analysis of HRV has become a valid means to non-invasively and quantitatively evaluate cardiovascular-autonomic control. Specialized software has been developed over the past few decades to automatically measure HRV. A Task Force was developed in 1996 to develop appropriate standards for HRV analysis due to its increasing popularity and ease of use in research and clinical settings.

There are two domains, time and frequency, which are used to measure the variability between heart beats. In the time domain method, either the heart rates at any

point in time or the intervals between successive normal (NN) intervals between adjacent QRS complexes, are determined (Task Force of the European Society of Cardiology, 1996). Mean heart rate, the standard deviation of the NN intervals (SDNN), and the squared root of the mean squared differences of successive NN intervals (RMSSD) are time-domain measures that can be calculated. The frequency component of HRV emphasizes the controlled and balanced behavior of the two branches of the autonomic nervous system (Task Force of the European Society of Cardiology, 1996). The power spectrum is separated into low frequency (LF) and high frequency (HF) powers. The HF power (0.15-0.40 Hz) and LF power (0.04-0.15 Hz) components are measured in normalized units (HFnu, LFnu), which represent the relative value of each power component in proportion to the total power minus the Very Low Frequency (VLF) component (0.0033-0.04 Hz) (Task Force of the European Society of Cardiology, 1996). HFnu is used as an indicator of parasympathetic modulation, whereas LFnu has controversies that surround this parameter (Task Force of the European Society of Cardiology, 1996). The LF:HF ratio represents the balance between sympathetic and parasympathetic modulation.

Standard spectral analysis requires the cardiovascular system to be in steady state and results in a single value each for the LF and HF peaks (Davrath, et al., 2006). HRV recordings are typically recorded in a supine position before and after exercise and are normally 5 minutes long. Respiratory rates influence HRV so professionals should pace the subject's breathing with a metronome during the analysis.

A high HRV is desirable and indicates sufficient parasympathetic control of heart rate (Tulppo & Huikuri, 2004). On the other hand, depressed HRV is associated with a dysfunctional autonomic nervous system and has also been shown to be a strong risk for cardiovascular disease and mortality. Mortality risk after myocardial infarction has been reported to be lower in patients with greater parasympathetic nervous system activity (Goldsmith, Bigger, Steinman, & Joseph, 1992). Those who participate in regular exercise regimes demonstrate parasympathetic dominance over sympathetic nervous dominance The impact of exercise training on parasympathetic nervous system activity, as assessed by heart rate variability, will be discussed in great detail throughout this review of the literature.

Heart Rate Recovery

HRR following exercise has been used as a crude indicator of parasympathetic-cardiovascular control. It is measured as the return of heart rate towards baseline immediately following exercise. To measure HRR, HR is measured with an electrocardiogram or a digital heart rate monitor and recorded at the end of exercise and again at a certain time point after exercise during recovery or cool down. The selected time point during recovery usually occurs at either one or two minutes post-exercise. The HR at the selected time point of recovery is then subtracted from the HR at cessation of exercise.

The primary importance of HRR is due to its ability to predict untoward cardiovascular events. Delayed HRR is defined as having a decrease of $\leq$ 12 beats/min at 1 minute of active, cool-down recovery or as a decrease $\leq$ 22 beats/min at 2 minutes of supine recovery (American College of Sports Medicine, 2010). Cole et al. (1999) performed a maximal graded exercise test on 2428 adults (63% males with a mean age of 57 $\pm$ 12 years) and calculated their HRR. The investigators then followed the subjects for six years and found that of the 213 patients who died from all-cause mortality, 56% had abnormal heart rate recovery values (<12 beats per minute). Since this study, other researchers have found a strong independent relationship between HRR and risk of mortality (Jouven, Empana, Schwartz, Desnos, Courbon, & Ducimetiere, 2005; Nanas, et al., 2006; Nishime, Cole, & Blackstone, 2000). Additionally, HRR has been shown to be inversely associated with insulin resistance, body mass index, abdominal obesity, and dyslipidemia (Brinkworth, Noakes, Buckley, & Clifton, 2006). Therefore, HRR is becoming a merging risk factor for chronic disorders like cardiovascular disease.

Fitness Status and Cardiovascular Autonomic Control

Measures of HRV and HRR are often used to assess cardiovascular autonomic control in athletes versus non-athletes. Most research indicates that higher levels of cardiovascular fitness may lead to higher HRV and HRR. Cross-sectional investigations show that HRV is higher in fit individuals versus sedentary or unfit individuals (Sztajzel, Jung, Sievert, & Bayes De Luna, 2008; De Meersman, 1993; Goldsmith, Bigger,

Steinman, & Joseph, 1992). This increase is a result of regular physical training, which evokes increased parasympathetic modulation of heart rate. One investigation found that a group of female marathon runners had significantly higher levels of HRV and faster HRR after exercise than untrained controls (Du, et al., 2005). Du et al.'s investigation is in agreement with several other cross-sectional studies which also suggest that HRR is accelerated in fit individuals when compared to unfit individuals (Otsuki, et al., 2007; Darr, Bassestt, Morgan, & Thomas, 1988).

Cross-sectional studies also suggest that differing volumes of physical activity affect both HRV and HRR measures. A significant difference in HRV was evident between women who engaged in "high" compared with "moderate" volumes of physical activity as determined by the Allied Dunbar National Fitness Survey (Gilder & Ramsbottom, 2007). Similarly, Sandercock et. al. (2008) found that those who reported themselves as being "less active" had lower cardiac parasympathetic modulation and shorter mean RR-interval than their more active counterparts. Carnethon et al. (2005) found that participants who reported in the highest levels of physical activity had significantly faster HRR than participants in the lowest levels.

The physiological mechanisms underlying the improved HRV and accelerated HRR remain unclear. It is questioned whether aerobic fitness (ie. VO_{2max}) influences cardiovascular autonomic control. Previous literature has found that a higher aerobic capacity is associated with improved HRV (Du, et al., 2005; Sztajzel, Jung, Sievert, &

Bayes De Luna, 2008). Contrastingly, there is evidence associating enhanced HRV in athletic subjects compared to healthy subjects who were matched for aerobic fitness. Esco & Williford (2011) found that basketball players had superior cardiovascular autonomic profile, as measured by HRV and HRR, when compared to healthy subjects who were matched for age, aerobic fitness and body fat percentage. In comparison, HRV remained unchanged in subjects who had improved VO_{2max} values after they underwent an aerobic training program,. Due to contrasting findings, the question is raised as to whether or not aerobic fitness is a possible mechanism underlying changes in HRV.

Aerobic Training and Cardiovascular Autonomic Control

There is extensive research regarding the effects of aerobic training in relation to HRV and HRR. Such research has highlighted the effects of exercise on cardiovascular autonomic modulation. Longitudinal studies suggest improved HRV in non-athletic groups after completing an endurance training program. One study investigated the implementation of 9 months of aerobic exercise at an intensity equivalent to approximately 70% of the subject's maximal aerobic power (VO_{2max}) in healthy older adults and found that HRV increased (Stein, Ehsani, Domitrovich, Kleiger, & Rottman, 1999) , which is in agreement with Melanson et al.'s (2001) conclusion that 12 weeks of aerobic exercise at 70-80% of Heart Rate Reserve improved parasympathetic tone in healthy older and young men. Aerobic training has also improved HRV in clinical

populations. Dougherty et al. (2008) reported improved HRV in survivors of sudden cardiac arrest with an implantable cardioverter defibrillator after the subject's engaged in 5 hours per week of aerobic exercise for 8 weeks.

Post-exercise heart rate recovery is also improved after endurance training. For example, after 8 weeks of moderate-intensity (70% of their VO_{2max}) cycle training, HRR was significantly accelerated in healthy untrained men (Sugawara, Mukrakami, Maeda, Kuno, & Matsuda, 2001). Most research regarding the effects of aerobic training on HRR focus on clinical, at risk, and older subjects. For instance, HRR accelerated in cardiovascular disease patients after exercise-based rehabilitation (Tiukinhoy, Beohar, & Hsie, 2003). This investigation is in agreement with Myer et al.'s (2007) findings who found that exercise training resulted in a faster HRR in patients with chronic heart failure. Research shows promising findings as regular aerobic training is associated with improved HRV and HRR which may decrease the risk of mortality in both healthy and clinical populations.

Anaerobic Training and Cardiovascular Autonomic Control

The importance of aerobic training and its influence on cardiovascular activity is well known; however, of recent interest are the effects of anaerobic training with regard to cardiovascular autonomic control. Research has shown that anaerobically trained athletes also exhibit accelerated HRR when compared to sedentary individuals. Otsuki et al. (2007) studied twelve strength-trained athletes (shot-put, discus, and hammer or

javelin throwers) and investigated their HRR for the first 30 seconds after exercise, and found that HRR values were accelerated in these strength-trained athletes compared to aged-matched sedentary controls. Similarly, Esco & Williford (2011) found that anaerobically trained basketball players had greater HRV and accelerated HRR when compared to physically fit, non-athletic control. Interestingly, Berkoff et al.'s (2007) investigation found that there was no significant difference between aerobically and power-based athletes, suggesting that anaerobic athletes reap the same cardiovascular autonomic benefits of training as endurance trained individuals. Furthermore, a longitudinal investigation reported improved HRR following a 6 week resistance training program (Hefferman, Fahs, Shinsako, Sae, & Fernhall, 2007).

In addition, HRR is being used to monitor recovery status between sets of resistance training exercises. A recent study compared hormonal and strength outcomes between two groups of strength training men whose between set recovery pattern was based on either HRR or a standard 2-minute period (Piirainen, et al., 2011). A Polar FT80 Heart Rate Monitor was used to notify the HRR group when recovery was sufficient, which was about 55 seconds between sets on average. At the end of 7-weeks, the HRR group had greater increases in muscular strength and testosterone levels compared to the standard group (Piirainen, et al., 2011). Athletic individuals, whether

they are aerobically trained, anaerobically trained or strength trained, have improved HRV and accelerated HRR after exercise, and thus improved cardiac vagal tone.

Body Composition and Cardiovascular Autonomic Control

There is considerable evidence describing the influence of body composition on autonomic control of heart rate. Literature suggests that poor HRV and HRR are associated with increased waist circumference (Lin, Kuo, Lai, Lin, Tseng, & Hwang, 2008), greater sum of skinfolds (Esco, Williford, & Olson, 2011), higher body mass index (BMI) (Molfino, Fiorentini, Tubani, Martuscelli, Fanelli, & Laviano, 2009; Piestrzeniewicz, Łuczak, Lelonek, Wranicz, & Goch, 2008), and higher body fat percentages (Millis R. M., et al., 2010). Not only are these findings evident in adults, but in children as well. Obese children showed a reduction in HRV as compared to lean children who have similar physical activity levels; therefore, such autonomic reduction was related to the amount of body fat in inactive state (Nagai & Moritani, 2004).

Contrastingly, weight loss has been shown to reverse and/or reduce these risk factors, which could subsequently improve parasympathetic control of heart rate as measured by HRV and HRR. One investigation found that weight loss via energy restriction improved HRR in overweight and obese men (Brinkworth, Noakes, Buckley, & Clifton, 2006). While these findings are significant, the subject's did not have a change in cardiorespiratory fitness levels. A combination of weight loss via caloric restriction in

combination with exercise has been found to have an even greater effect on cardiovascular autonomic control. Sympathetic and parasympathetic nervous system balance improved in overweight individuals when weight loss was combined with exercise (de Jonge, Moreira, Martin, & Ravussin, 2012). Weight loss combined with exercise also accelerated HRR in overweight and obese men with features of the metabolic syndrome (Brinkworth, Noakes, Buckley, & Clifton, 2006). Body composition can therefore be considered an important predictor of cardiovascular autonomic control. Those with excess body fat are at an increased risk for unfavorable events post exercise; however, weight loss in combination with exercise can help combat this risk.

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CHAPTER 2

THE EFFECTS OF A 7-WEEK LOW VOLUME RECREATIONAL JOGGING PROGRAM ON HEART RATE VARIABILITY

ABSTRACT

Heart rate variability (HRV) is the interval between heart beats. A depressed HRV is a predictor of early cardiovascular disease and mortality. **Purpose:** The purpose of this investigation was to determine if a 2 day per week jogging program would improve HRV after 7 weeks in young adults. **Methods**: Twenty-one male (n=11) and female (n=10) young adults (mean age: 23.38 ± 4.96) participated in this study. At baseline, body fat percentage (BF%) was analyzed with dual-energy x-ray absorptiometry (DXA). Maximal oxygen consumption (VO_{2max}) was determined via a maximal graded treadmill test with open circuit spirometry. Subjects remained in a supine position for 10 minutes while heart rate was analyzed by electrocardiography (ECG). The ECG was sampled at a frequency of 1,000 Hz. The last 5 minutes of the 10 minute recording was used for HRV analysis. Specialized software (Nevrokard) was used to analyze HRV in the frequency domains. After baseline testing, the subjects began a 2-day per week recreational jogging program for a 7-week period. At the end of the 7-week training program, the subjects returned to the laboratory for post-testing measures. **Results**: The 7-week training protocol did not induce any significant changes in VO_{2max} (Pre = 34.83 ± 9.61 $ml \cdot kg^{-1} \cdot min^{-1}$, Post = 35.99 ± 9.80 $ml \cdot kg^{-1} \cdot min^{-1}$, $p > 0.05$), BF% (Pre = 30.70 ± 12.47 %, Post =

30.56 ± 12.66%, $p > 0.05$), HFnu (Pre = 54.6 ± 7.6 ms^2, Post = 52.7 ± 7.8 ms^2, $p > 0.05$) or LF:HF (Pre = 0.56 ± 0.30 , Post = 0.60 ± 0.30 , $p > 0.05$). **Conclusions**: It can be concluded that there was no enhancement of HRV, as assessed by the frequency-domain, in response to a two day per week recreational jogging program.

INTRODUCTION

Cardiovascular activity is largely controlled by the two branches of the autonomic nervous system. The sympathetic branch of the autonomic nervous system is responsible for increasing heart rate; whereas the parasympathetic branch is responsible for the lowering of heart rate. Sympatho-parasympathetic balance results in rhythmic time-related changes in successive heart beats, a phenomenon known as heart rate variability (HRV). Analysis of HRV has become a valid means to non-invasively evaluate cardiovascular-autonomic control. The primary importance of HRV is due to its link to untoward, fatal events as low HRV is associated with morbidity and mortality from cardiac and non-cardiac causes (Gilder & Ramsbottom, 2008).

From a practical perspective, HRV is becoming a useful, simplistic tool for monitoring the exercise-induced improvements with exercise training. The exact volume of exercise training required to elicit improvements in HRV is unknown, but most research indicates that changes occur after the implementation of a high frequency,

moderate-intense training program. For instance, longitudinal studies suggest improved HRV in non-athletic groups after an endurance training program. One study investigated the implementation of 9 months of aerobic exercise at an intensity equivalent to approximately 70% of the subject's maximal aerobic power (VO_{2max}) in healthy older adults and found that HRV increased (Stein, Ehsani, Domitrovich, Kleiger, & Rottman, 1999), which is in agreement with Melanson et al.'s (2001) conclusion that 12 weeks of aerobic exercise at 70-80% of Heart Rate Reserve improved parasympathetic tone in healthy older and young men.

HRV is also used to assess cardiovascular autonomic control in athletes versus non-athletes. Cross-sectional investigations show that HRV is higher in fit individuals versus sedentary or unfit individuals (Goldsmith, Bigger, Steinman, & Fleiss, 1992). This increase is a result of regular physical training, which evokes increased parasympathetic modulation of heart rate. One investigation found that a group of female marathon runners had significantly faster HRR and higher levels of HRV after exercise than untrained controls (Du, et al., 2004). Cross-sectional studies also suggest that differing volumes of physical activity affect HRV measures. A significant difference in HRV was evident between women who engaged in "high" compared with "moderate" volumes of physical activity as determined by the Allied Dunbar National Fitness Survey (Gilder & Ramsbottom, 2008). Similarly, Sandercock et al. (2008) found that those who reported themselves as being "less active" had lower cardiac parasympathetic modulation and shorter mean RR-interval than their more active counterparts.

Research suggests that greater amounts of physical activity, as reported by active populations, increases HRV; however, it is important to note that athletic individuals typically train on multiple days of the week. Most studies that report an improved HRV after training involve at least 3 days per week of exercise. There are no studies available determining the effects of a low-frequency (i.e., < 3 days/week) aerobic-training program on HRV in a non-athletic group. Therefore, the purpose of this investigation is to determine if a 2 day per week jogging program would improve heart rate variability (HRV) after 7 weeks in young adults with low baseline cardiovascular fitness levels. It is hypothesized that the jogging program will improve the HRV profile.

METHODS

Experimental Approach to the Problem

This study was designed to measure pre and post indexes of HRV in unfit individuals who underwent a 2 day per week recreational jogging program for a 7 week period. The dependent variables in this investigation are the cardiovascular-parasympathetic parameters, HRV, whereas the independent variable in this investigation is the recreational jogging program. At baseline, the following variables were analyzed: resting HRV; VO_{2max}, and selected body composition parameters. The subjects then engaged in a twice per week jogging program for 7 weeks. The intensity of the program

was not monitored; however, weekly increases in duration were made. At the end of the 7 week program, the post-measures of the dependent (i.e., HRV) and descriptive (i.e., VO_{2max} and the body composition parameters) variables were obtained.

Subjects

Twenty five apparently healthy young adults were recruited for this investigation. All data was collected in the Human Performance Laboratory at Auburn University Montgomery. The experiment was approved by the Institutional Review Board and the subjects provided written informed consent before starting the investigation. The participants completed a health-history questionnaire which was used as a screening tool to eliminate anyone with cardiovascular, pulmonary, or metabolic diseases. Subjects were allowed to participate in the data collection process if they were not hypertensive (blood pressure <140/90 mmHg), not participating in a competitive athletic sport, and not taking any prescribed or over-the-counter medications.

Baseline Measures

Body Composition

Subjects reported to the laboratory for preliminary testing between 7:00 am and 11:00 am on any day of the week. The subjects were instructed not to consume any caffeinated or alcoholic beverages 24 hours before the test, to not engage in any

strenuous exercise 12 hours before and to not eat at least 4 hours before the test. After completing the necessary consent forms and health-history questionnaires, the subjects were briefed on the design of the study and given verbal instructions in regard to the testing protocol. The following anthropometric variables were recorded: height, weight, and body mass index (BMI). Height was measured using a wall mounted stadiometer (SECA) and rounded to the nearest 0.1 cm and weight was recorded using a digital scale (TANITA BWB-800A) and rounded to the nearest 0.1 kg. BMI was kg in weight per meter of height squared. Additionally, body fat percentage (BF%) was measured via Dual Energy X-Ray Absorptiometry (DEXA).

Short-term Resting Heart Rate Variability

For resting HRV assessment, the subjects assumed a supine position on an athletic training table for a 10 minute period before the maximal exercise test. During this time, heart rate was analyzed by electrocardiography (ECG), which involved a customized lead II configuration using three Ag/AgCl electrodes (BIOPAC ES509). The electrodes were interfaced with a Biopac MP100 data acquisition system (Goletta, CA). During the 10 minute supine period, the subjects were instructed to pace their breathing at 12 breaths per minute as set by a Metronome. External stimuli, and noise was removed and one of the two panels of lights within the laboratory was turned off. The ECG was sampled at a frequency of 1,000 Hz. The last 5 minutes of the 10 minute recording was used for HRV

analysis. Specialized software (Nevrokard) was used to analyze HRV in the frequency domains. Each 5-minute time period was visually inspected and all abnormal beats and artifacts were removed. Any segment with three or more ectopic beats was not included in the final analysis. The 5-minute ECG recordings were converted to a power spectrum by applying a Hanning window with Fast fourier transformation. The power spectrum was separated into low (LF) and high (HF) frequency powers. The HF power (0.15-0.40 Hz) and LF power (0.04-0.15 Hz) components were measured in normalized units (HFnu, LFnu), which represents the relative value of each power component in proportion to the total power minus the Very Low Frequency (VLF) component (0.0033-0.04 Hz) (Task Force). HFnu was used in data analysis as an indicator of parasympathetic modulation. However, LFnu was recorded but not included in data analysis due to the controversies that surround this parameter (Task Force). The LF:HF ratio was recorded and analyzed to represent the balance between sympathetic and parasympathetic modulation.

Maximal Aerobic Power

A maximal graded exercise test was completed on a Trackmaster treadmill (Full Vision, Inc., Carrollton, TX) to determine VO_{2max}. The Bruce protocol was used which involved a series of 3-minute stages with consecutive increases in speed and grade until VO_{2max} was reached. Expired gas fractions were evaluated with a ParvoMedics TrueOne® 2400 metabolic cart (Sandy, UT). VO_{2max} was achieved if at least two of the

following criteria occurred: a plateau in VO_2 ($\pm$ 2 $ml \cdot kg^{-1} \cdot min^{-1}$) with increasing work rate; respiratory exchange ratio $\geq$ 1.15; heart rate within 10 beats of age predicted maximum (220 – age); or volitional fatigue. Heart rate was monitored during the test by a Polar electronic heart monitor (Polar Electro Oy, Kemple, Finland). A standard sphygmomanometer and stethoscope were used to measure blood pressure during the last 45 seconds of each stage.

Training Protocol

Once the baseline testing was completed, the subjects began the 2-day per week recreational jogging program. The protocol duration was 7 weeks. Each training session began at 6:45 am CST on Tuesdays and Thursdays. During the first week, the subjects were instructed to jog 1 mile. There after weekly increases in distance of 0.25 mile were made until week 3, at which the distance increased 0.50 mile. If a subject could not complete the required distance during any portion of the protocol, they were encouraged to include brief bouts of walking until they were eventually able to jog the entire distance. By the end of the 7-week training period, each subject was able to complete 3 total miles. It should be noted that each subject was told to maintain and control their own pace during each jogging session. Heart rate, oxygen consumption, and ratings of perceived exertion were not accounted for.

Post Measures

After the seven week training program, all of the subjects returned to the lab for post-testing of the following variables: BMI, BF%, $VO_{2max,}$ and the frequency HRV parameters. The exact testing procedures that were performed during the pre-testing were followed for post-testing.

Statistical Analyses

All statistics were completed using SPSS/PASW version 19.0 (Cary, NC). Means and standard deviations were determined for the following descriptive statistics: age (years), height (cm), weight (kg), BMI (kg/m^2), predicted body fat (%), VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$), systolic blood pressure (mmHg), and diastolic blood pressure (mmHg). A repeated measures paired t-test was used to determine significance between the following variables: VO_{2max}, BF%, HFnu and LF:HF. Statistical significance for all tests was set at $P < 0.05$.

RESULTS

After the 7-week training protocol, twenty one male (n = 11) and female (n = 10) subjects met the criteria to be included in data analysis. There were 3 subjects whose data were excluded due to participation in a competitive sport. There were also 2 subjects

whose data were excluded due to the interference of artifacts on their transformed EKG recording.

The descriptive variables (mean ± standard deviation) of all the participants at baseline can be found in Table 1. The 7-week training protocol did not induce any significant changes in VO_{2max} (Pre = 34.83 ± 9.61 $ml \cdot kg^{-1} \cdot min^{-1}$, Post = 35.99 ± 9.80 $ml \cdot kg^{-1} \cdot min^{-1}$, $p > 0.05$), BF% (Pre = 30.70 ± 12.47 %, Post = 30.56 ± 12.66%, $p > 0.05$), HFnu (Pre = 54.6 ± 7.6 ms^2, Post = 52.7 ± 7.8 ms^2, $p > 0.05$) or LF:HF (Pre = 0.56 ± 0.30 , Post = 0.60 ± 0.30 , $p > 0.05$).

DISCUSSION

The present study sought to determine if a 2 day per week jogging program would improve resting HRV after 7 weeks in young adults with low baseline cardiovascular fitness levels. The major finding of the present study was that there was no enhancement of HRV as assessed by the frequency-domain in response to a two day per week recreational jogging program. This is most likely associated to the training program failing to improve cardiovascular fitness or BF%. Previous literature has found that HRV is related to cardiovascular fitness and BF%. A higher aerobic capacity is associated with improved HRV (Du, et al., 2004; Levy, et al., 1998). Additionally, an increasing BMI is associated with increased sympathetic activity and lowered parasympathetic activity

(Molfino, Fiorentini, Tubani, Matruscelli, Fanelli, & Laviano, 2009; Piestrzeniewicz, Łuczak, Wranicz, & Goch, 2008). Furthermore, excess BF% is associated with decreased HRV (Millis, et al., 2010). In our investigation, there were no significant improvements in cardiovascular fitness or BF%, subsequently there was no improvement in HRV.

HFnu was used in data analysis as an indicator of parasympathetic modulation (Task Force of the European Society of Cardiology in the North American Society of Pacing Electrophysiology, 1996) . The results indicate that there was no significant change between pre and post measures of HFnu; although, the values did decrease slightly. The LF:HF ratio was analyzed to represent the balance between sympathetic and parasympathetic modulation (Task Force of the European Society of Cardiology in the North American Society of Pacing Electrophysiology, 1996) and it remained relatively unchanged in this investigation. The findings are not in agreement with the majority of previous reports. It is generally implied that exercise training improves HRV, as Sandercock et al.'s (2005) meta-analysis concludes that exercise training causes significant increases in vagal modulation. However, the presumed association between aerobic exercise training and increased HRV is mostly based on longitudinal and cross-sectional studies involving high frequency training regimes.

The influence of an aerobic training program involving a protocol of three or more days per week has been extensively studied with relation to HRV. Such investigations suggest that a training program involving three or more days per week of

aerobic exercise increases HRV (Jurca, Church, Morss, Jordan, & Earnest, 2004; Stein, Ehsani, Domitrovich, Kleiger, & Rottman, 1999; Melanson & Freedson, 2001; Hautala, et al., 2003; Myslivecek, Brown, & Wolfe, 2002). Despite the aforementioned findings, there is limited research analyzing the affects of a low-frequency aerobic training program. To our knowledge, this was the first study that analyzed the effects of a low-frequency program, involving less than three days per week of exercise, on HRV in a non-athletic population. The results from this investigation suggest that a recreational jogging program of low prevalence is not sufficient enough to induce significant changes in HRV. Establishing the minimal guidelines necessary to elicit enhancements in cardiovascular autonomic control warrants further research.

The results of the current investigation are in agreement with Boutcher & Stein (1995), who also found no changes in HRV after participation in an aerobic training program. It should be noted however that the subjects in Boutcher's investigation were middle-aged men who had improved aerobic fitness values at the end of the training program. Due to contrasting findings in relation to aerobic fitness between the current study and Boutcher's investigation, the question is raised as to whether or not aerobic fitness is a possible mechanism underlying changes in HRV.

One of the limitations of the study was that exercise intensity was self-monitored by the subjects and not laboratory controlled. Exercising at a self-monitored intensity may be one the reasons explaining why there was no improvement in cardiovascular

fitness. The American College of Sports Medicine suggests that exercising at a moderate intensity of 40% to 59% of VO_2 reserve will result in an increase in cardiorespiratory capacity and endurance (American College of Sports Medicine, 2010). Whether or not individuals were meeting this guideline is indefinite; thus, this could be a possible reason why aerobic fitness failed to improve. From a practical perspective, however, most individuals do not have the means nor equipment that is used in a laboratory setting to monitor their exercise intensity. Another limitation of the study was that only young adults were recruited; therefore, the results cannot be extrapolated to subjects of older ages or to clinical populations. Furthermore, there was no dietary monitoring and subjects did not have to provide dietary recalls, which could explain why the results did not show any improvements in BF%.

The findings of this study suggest that a two day per week, low-intensity jogging program is not sufficient enough to elicit improvements cardiac autonomic function. An aerobic training protocol of a higher frequency than the current study is possibly needed to enhance these parameters, as prior literature has suggested (Sandercock, Hardy-Shepherd, Nunan, & Brodie, 2008; Stein, Ehsani, Domitrovich, Kleiger, & Rottman, 1999). Further research is needed to determine the most appropriate ranges of exercise frequency for improving cardiovascular autonomic function.

Table 1. Descriptive characteristics of the subjects (n=21)

	Pre	**Post**
Age (yrs)	23.38 ± 4.96	23.49 ± 4.90
Height (cm)	169.51 ± 8.24	169.27 ± 8.37
Weight (kg)	77.73 ± 19.87	77.57 ± 20.21
BMI (kg/m^2)	27.00 ± 6.47	27.02 ± 6.59
Body Fat (%)	30.70 ± 12.47	30.56 ± 12.66
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	34.83 ± 9.61	35.99 ± 9.80
SBP (mmHg)	112.48 ± 16.92	113.52 ± 15.62
DBP (mmHg)	70.14 ± 14.29	75.52 ± 12.21

Table 2. HRV Parameters

Variable	**Mean**	**SD**
HFnu (ms^2) Pre	54.6404	7.55089
HFnu (ms^2) Post	52.6617	7.84506
LFnu (ms^2) Pre	28.9035	10.43144
LFnu (ms^2) Post	29.8878	9.08481
LF:HF Pre	0.5643	0.29609
LF:HF Post	0.6013	0.26793

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CHAPTER 3

TWO DAYS PER WEEK OF RECREATIONAL JOGGING DOES NOT IMPROVE HEART RATE RECOVERY AFTER 7 WEEKS

ABSTRACT

Heart rate recovery (HRR) is the return of heart rate towards baseline after a bout of exercise. A delayed HRR is a predictor of early cardiovascular disease and mortality. **Purpose:** The purpose of this investigation was to determine if a 2 day per week jogging program would improve HRR after 7 weeks in young adults. **Methods**: Twenty-one male (n=11) and female (n=10) young adults (mean age: 23.38 ± 4.96) participated in this study. At baseline, body fat percentage (BF%) was analyzed with dual-energy x-ray absorptiometry (DXA). Maximal oxygen consumption (VO_{2max}) was determined via a maximal graded treadmill test with open circuit spirometry. HRR was determined as the difference between maximum heart rate (HR_{max}) and 1 minute (HRR1) and 2 minute (HRR2) post-exercise heart rate. After baseline testing, the subjects began a 2-day per week recreational jogging program for a 7-week period. At the end of the 7-week training program, the subjects returned to the laboratory for post-testing measures. **Results**: The 7-week training protocol did not induce any significant changes in HRR1 (Pre = 21.48 ± 9.95 bpm, Post = 21.10 ± 9.16 bpm, $p > 0.05$) and HRR2 (Pre = 37.71 ± 13.80 bpm, Post = 39.76 ± 12.54 bpm, $p > 0.05$). **Conclusions**: It can be concluded that a twice per week recreational jogging program does not enhance HRR after 7 weeks.

INTRODUCTION

Heart rate recovery (HRR) is the return of heart rate towards baseline immediately following exercise. Recovery is thought to result primarily from parasympathetic reactivation, thus making HRR a useful estimate of autonomic nervous function (Carnethon, et al., 2004). HRR is a valuable way to assess cardiovascular parasympathetic influence as a delayed decrease in heart rate during the first minute after exercise has been suggested to be a powerful and independent predictor of all-cause mortality (Javorka, Zila, Balharek, & Javorka, 2002). A slow HRR is associated with poor prognosis, and is defined as a decrease of ≤ 12 beats/min at 1 minute of walking recovery or as a decrease ≤ 22 beats/min at 2 minutes of recovery in a supine position (American College of Sports Medicine, 2010).

Regular aerobic training is associated with improved HRR which may contribute to decreased risk of mortality. The exact volume of exercise training required to elicit improvements in HRR is unknown, but most research indicates that changes occur after the implementation of a high frequency training program. For instance, Du et al. (2005) found that female marathon runners had faster HRR after exercise than untrained, age-matched controls. Furthermore, Otsuki et al. (2005) found that both strength trained individuals and endurance trained individuals who exercised 5 days per week had faster post exercise HRR than sedentary controls. In addition, longitudinal studies also suggest that regular physical activity is associated with accelerated HRR. Carnethon et al. (2004)

found that participants who reported in the highest tertile of frequent physical activity had significantly faster HRR than participants in the lowest tertile.

These studies indicate that a greater volume of physical activity results in significantly faster HRR. It is important to note that most studies reporting an improved HRR after training involve at least 3 days per week of exercise. However, there are no studies available determining the effects of a low-frequency (i.e., < 3 days/week) aerobic-training program on HRR in a non-athletic group. Therefore, the purpose of this investigation was to determine if a 2 day per week jogging program would improve HRR after 7 weeks in young non-athletic adults.

METHODS

Subjects

Twenty five apparently healthy young adults were recruited for this investigation. All data was collected in the Human Performance Laboratory at Auburn University at Montgomery. The experiment was approved by the Institutional Review Board and the subjects provided written informed consent. The participants completed a health-history questionnaire which was used as a screening tool to eliminate anyone with cardiovascular, pulmonary, or metabolic diseases. Subjects were allowed to participate in the data collection process if they were not hypertensive (blood pressure <140/90

mmHg), not participating in a competitive athletic sport, and not taking any prescribed or over-the-counter medications.

Subjects reported to the laboratory for preliminary testing between 7:00 am and 11:00 am on any day of the week. The subjects were instructed not to consume any caffeinated or alcoholic beverages 24 hours before the test, to not engage in any strenuous exercise 12 hours before and to not eat at least 4 hours before the test. After completing the necessary consent forms and health-history questionnaires, the subjects were briefed on the design of the study and given verbal instructions in regard to the testing protocol.

Baseline Measures

Body Composition

The following anthropometric variables were recorded: height, weight, and body mass index (BMI). Height was measured using a wall mounted stadiometer (SECA) and rounded to the nearest 0.1 cm and weight was recorded using a digital scale (TANITA BWB-800A) and rounded to the nearest 0.1 kg. BMI was calculated as kilograms in weight per meter of height squared. Body fat percentage (BF%) was measured via Dual Energy X-Ray Absorptiometry.

Maximal Graded Exercise Test and Heart Rate Recovery

A maximal graded exercise test was completed on a Trackmaster treadmill (Full Vision, Inc., Carrollton, TX) to determine maximal oxygen consumption (VO_{2max}). The Bruce protocol was used which involved a series of 3-minute stages with consecutive increases in speed and grade until VO_{2max} was reached. Expired gas fractions were evaluated with a ParvoMedics TrueOne® 2400 metabolic cart (Sandy, UT). VO_{2max} was achieved if at least two of the following criteria occurred: a plateau in VO_2 (± 2 $ml \cdot kg^{-1} \cdot min^{-1}$) with increasing work rate; respiratory exchange ratio ≥ 1.15; heart rate within 10 beats of age predicted maximum (220 – age); or volitional fatigue. Heart rate was monitored during the test by a Polar electronic heart monitor (Polar Electro Oy, Kemple, Finland). A standard sphygmomanometer and stethoscope were used to measure blood pressure during the last 45 seconds of each stage. Heart rate at VO_{2max} was recorded as maximal heart rate (MHR). Recovery HR was recorded during the first (HR1) and second minute (HR2) of the cool down period, during which the subjects walked at 2.5 mph at 1.5 % grade. Heart rate recovery 1- and 2-minutes was calculated as the difference between MHR and HR1 (HRR1) and HR2 (HRR2), respectively.

Training Protocol

Once the baseline testing was completed, the subjects began the 2-day per week recreational jogging program. The protocol duration was 7 weeks. Each training session began at 6:45 am CST on Tuesdays and Thursdays. During the first week, the subjects

were instructed to jog 1 mile. There after, weekly increases in distance of 0.25 mile were made until week 3, at which the distance increased 0.50 mile per week. If a subject could not complete the required distance during any portion of the protocol, they were encouraged to include brief bouts of walking until they were eventually able to jog the entire distance. By the end of the 7-week training period, each subject was able to complete 3 total miles. It should be noted that each subject was told to maintain and control their own pace during each jogging session. Heart rate, oxygen consumption, and ratings of perceived exertion were not accounted for.

Post Measures

One week after the seven week training program, all of the subjects returned to the lab for post-testing of the following variables: BMI, BF%, $VO_{2max,}$ and HRR. The exact testing procedures that were performed during the pre-testing were followed for post-testing.

Statistical Analyses

All statistics were completed using SPSS/PASW version 19.0 (Cary, NC). Means and standard deviations were determined for the following descriptive statistics: age (years), height (cm), weight (kg), BMI (kg/m^2), body fat (%), and VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$). A paired t-test was used to determine significance between the pre- and post-variables of

the following: VO_{2max}, BF%, HRR1 and HRR2. Statistical significance for all tests was set at $p < 0.05$.

RESULTS

After pre-testing, twenty-one male (n = 11) and female (n = 10) subjects met the criteria to be included in data analysis. There were 4 subjects whose data were excluded due to participation in a competitive sport.

The descriptive variables (mean ± standard deviation) of all the participants at baseline can be found in Table 1. The 7-week training protocol did not induce any significant changes in VO_{2max} (Pre = 34.83 ± 9.61ml·kg^{-1}·min^{-1}, Post = 35.99 ± 9.80 ml·kg^{-1}·min^{-1}, $p > 0.05$), BF% (Pre = 30.70 ± 12.47%, Post = 30.56 ± 12.66 %, $p > 0.05$), HRR1 (Pre =21.48 ± 9.95 bpm, Post = 21.10 ± 9.16 bpm, $p > 0.05$), or HRR2 (Pre = 37.71 ± 13.80 bpm, Post = 39.76 ± 12.54 , $p > 0.05$).

DISCUSSION

The present investigation sought to determine whether a 2 day per week jogging program would improve HRR after 7 weeks in young adults with low baseline cardiovascular fitness levels. The major finding of the present study was that there was no change in HRR after the 7 week training protocol.

It is important to highlight that the 7 week training protocol also did not induce any significant changes in cardiovascular fitness, or body composition. Previous literature reports that faster HRR after exercise is dependent on higher aerobic capacity (Darr, Bassett, Morgan, & Thomas, 1988; Du, et al., 2005). Aerobic capacity did not improve after the training program in this investigation which is most likely associated to the failure of HRR to improve. Literature also suggests that increased waist circumference (Lin, Kuo, Lai, Lin, Tseng, & Hwang, 2008) and sum of skinfolds (Esco, Williford, & Olson, 2011) influence autonomic control of heart rate. Further to this, one study involving dietary weight loss found improved HRR values (Brinkworth, Noakes, Buckley, & Clifton, 2006). In our investigation, there was no significant decrease in neither weight nor body fat percentage which may be one of the mechanisms explaining the lack of improvement in HRR values. Because the training program failed to improve body composition, there was subsequently no significant improvement in HRR.

One of the limitations of the study was that exercise intensity was self-monitored by the subjects and not laboratory controlled. Exercising at a self-monitored intensity may be one reason explaining why there was no improvement in cardiovascular fitness. The American College of Sports Medicine suggests that exercising at a moderate intensity of 40% to 59% of VO_2 reserve will result in an increase in cardiorespiratory capacity and endurance (American College of Sports Medicine, 2010). Whether or not individuals were meeting this guideline is indefinite; thus, this could be a possible reason why our protocol failed to improve the variables. Another limitation of the study was that

only young adults were recruited; therefore, the results cannot be extrapolated to subjects of older ages or to clinical populations.

It can be concluded that a twice per week recreational jogging program does not enhance HRR after 7 weeks. Further research is warranted to determine the minimal guidelines necessary for improving HRR after exercise.

Table 1. Descriptive characteristics of the subjects

	Pre	**Post**
Age (yrs)	23.38 ± 4.96	23.49 ± 4.90
Height (cm)	169.51 ± 8.24	169.27 ± 8.37
Weight (kg)	77.73 ± 19.87	77.57 ± 20.21
BMI (kg/m^2)	27.00 ± 6.47	27.02 ± 6.59
Body Fat (%)	30.70 ± 12.47	30.56 ± 12.66
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	34.83 ± 9.61	35.99 ± 9.80
SBP (mmHg)	112.48 ± 16.92	113.52 ± 15.62
DBP (mmHg)	70.14 ± 14.29	75.52 ± 12.21

Table 2. Mean heart rate recovery (beats/min) variables at- and post-maximal exercise

	Pre	**Post**
Max HR	182.48 ± 15.76	184.05 ± 14.90
HR1	161.00 ± 18.51	165.62 ± 15.50
HR2	144.76 ± 19.72	145.52 ± 17.06
HRR1	21.48 ± 9.95	21.10 ± 9.16
HRR2	37.71 ± 13.80	39.76 ± 12.54

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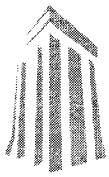
APPENDIX A



AUBURN

MONTGOMERY

RESEARCH STUDY

The Influence of Stretching in Recreational Runners

We are seeking recreational runners in the physical education department to participate in a study to determine the influence of stretching on cardiovascular markers in runners.

To participate, the following criteria must be met:

1. **Physical education student at AUM**
2. **Free from cardiovascular, pulmonary, or metabolic disease**
3. **Not currently taking any prescription medications**

If you meet the criteria for the study, you will receive a fitness test and body fat assessments performed by trained exercise professionals.

If you are interested in participating in this study please contact:

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Auburn University Montgomery
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APPENDIX B

INFORMED CONSENT

The Influence of Stretching on Cardiovascular Markers in Recreational Runners

Auburn University Montgomery

Department of Physical Education and Exercise Science

You are invited to participate in a study to assess the effects of a stretching program on heart rate recovery and heart rate variability. You were selected as a possible participant because you are a Physical Education student at AUM.

If you decide to participate, you will be asked to report to the Human Performance Laboratory at Auburn University Montgomery between 8:00am and 4:00pm on two occasions. There will be a pre-testing session at the start of the semester, and a post-testing session at the end of the half semester period, with multiple stretching sessions between. The testing sessions should not last longer than 2 hours. The following variables will be also collected for each subject during the first and final visit: Height; body weight; body mass index (BMI); waist and hip circumferences; waist to hip ratio (WHR); resting blood pressure; and body composition. Body composition will be assessed with the use of Dual Energy X-Ray Absorptiometry (DEXA). Human performance laboratories that undertake bone mineral density and body composition studies use DEXA as the standard technology due to its specificity, accuracy, and safety. There are usually no complications from this procedure. There is a small amount of radiation exposure, less that 1/10 the dose of a standard chest x-ray. Measures of flexibility will also be tested using a sit and reach box as well as a goniometer. Heart rate variability (HRV) will also be examined. The measurement of HRV is a painless procedure that involves the use of electrocardiogram with three surface electrodes attached to the thoracic and lumbar regions. During the assessment of HRV you will be asked to assume a supine position (lying on your back) for a 20-minute period.

Additionally, you will complete a maximal graded exercise test (GXT) on a treadmill. Blood pressure and heart rate will be assessed before, during and after exercise.

The death rate associated with exercise is less than 1 in 20,000 exercise tests. Every effort will be made to minimize risks through preliminary screenings and observations during the test. Some discomforts and inconveniences are possible. Musculoskeletal injury (strain or sprains) could occur. There is a possibility of nausea, dizziness, fainting, and/or fatigue as a result of exercise. Should injury occur as a result of the experimental protocol it would be your responsibility to seek medical attention. Muscle soreness in the lower body could also occur 24 to 48 hours after the test.

Personnel in charge will attempt to minimize all risks. For instance, the test will be terminated if you experience any of the following: chest pain, fatigue, shortness of breath, wheezing, leg cramps, claudication, dizziness, syncope, cyanosis or pallor. You will be screened to determine if you have any health problems that would prevent you from performing exercise. All personnel have been trained in CPR. Emergency procedures are posted in the Human Performance Laboratory. You are free to withdraw from the study at any time.

The subjects participating in this study will gain a better understanding of the effects of exercise on cardiovascular autonomic modulation. The subjects will obtain a maximal aerobic test and body composition measures. Additionally, they will have improved flexibility levels at the end of the 2 month period. This information will be useful to aid the subject in future fitness testing and establishing an exercise program.

Any information obtained in connection with this study that can be identified with you will remain confidential and will be disclosed only with your permission. If you give me your permission by signing this document, I plan to disclose your information to Dr. Mike Esco only for the purpose of assisting with statistical analysis. Your information will be coded numerically and stored in private filling cabinets in the Human Performance Laboratory.

Your decision whether to participate will not prejudice your future relations with Auburn University Montgomery. If you decide to participate, you are free to withdraw your consent and to discontinue participation at any time without penalty. If you decide later to withdraw from the study, you may also withdraw any information that has been collected about you.

If you have questions concerning this study please feel free to ask Aindrea McHugh directly or via phone at (334) 244-3472. If you have questions concerning your rights as a human subject please call Debra Tomblin at (334) 244-3250.

YOU ARE MAKING A DECISION WHETHER TO PARTICIPATE. YOUR SIGNATURE INDICATES THAT YOU HAVE DECIDED TOPARTICIPATE, HAVING READ THE INFORMATION PROVIDED ABOVE.

_____ _____

Subject's Signature Date

_____ _____

Investigator's Signature Date