CARDIOVASCULAR AUTONOMIC MODULATION FOLLOWING MAXIMAL EXERCISE: ITS RELATIONSHIP TO RACE, VO_{2MAX}, AND RESTING HEART RATE VARIABILITY

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CARDIOVASCULAR AUTONOMIC MODULATION FOLLOWING MAXIMAL EXERCISE: ITS RELATIONSHIP TO RACE, VO_{2MAX}, AND RESTING HEART RATE VARIABILITY

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A Dissertation

Submitted to

the Graduate Faculty of

Auburn University

in Partial Fulfillment of the

Requirements for the

Degree of

Doctor of Philosophy'

Auburn, Alabama
May 9, 2009

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DISSERTATION ABSTRACT

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Doctor of Philosophy, May 9, 2009
Masters of Education, May 2005
Bachelors of Science, December 2003

136 typed pages

Directed by Daniel Blessing

The purpose of this dissertation was to provide a better understanding of post maximal exercise cardiovascular autonomic function and its relationship to race, VO_{2max} and resting heart rate variability. Heart rate variability (HRV) and heart rate recovery (HRR) are two non-invasive methods to assess cardiovascular autonomic modulation. This dissertation covers three studies. The first study examined the difference in cardiac autonomic function at rest and following maximal exercise between 30 white and 30

black college-age men. The results showed that black men had significantly greater HRV measures at rest, significantly faster heart rate recovery 2-minutes after exercise and significantly greater HRV during a 30-minute post-exercise period compared to the white men. The second study sought to determine the association of race, VO_{2max}, and selected body composition measures to HRR. In this study, seventy-two college-aged men were divided into two groups above and below the mean VO_{2max} of the entire sample (46.32 ml/kg/min) as follows: group 1 (ModFit, n = 36) had a mean VO_{2max} of 39.99 ± 4.01 ml/kg/min; and Group 2 (HiFit, n = 36) had a mean VO_{2max} of 52.66 ± 5.30 ml/kg/min. After comparing the mean heart rate recovery between the two groups, no significant difference was found. However, after controlling for the influence of race, a significant difference between groups in heart rate recovery 2-minutes (HRR2) was revealed, i.e., the HiFit group had a greater HRR2 compared to the LoFit group. Also, backwards linear regression procedures revealed that race was the most significant variable at accounting for the variation in HRR2 and the heart rate at 2-minutes post-exercise. The third study examined the relationship between resting HRV and HRR. The results of this study revealed no association between HRR and any of the resting HRV. However, there were significant associations between resting HRV and the following variables: maximal heart rate, heart rate at 1-minute post-exercise, and the heart rate a 2-minutes post-exercise.

ACKNOWLEDGEMENTS

I would like to thank Dr. Daniel Blessing for agreeing to be my committee chair and advisor during the expansion of my academic career in Exercise Physiology. I would also like to thank the members of my committee: Dr. Michele Olson, Dr. Peter Grandjean, and Dr. David Shannon, for their guidance throughout this project. I am enormously grateful for Dr. Michele Olson. Her influence, encouragement, friendship and mentorship have been vital in the advancement of my academic and professional career. I would also like to thank Dr. Barbara Wilder for agreeing to be my outside reader. Moreover, I would like to express my appreciation to all of the excellent professors I have had at Auburn University.

In addition, I would like to thank Dr. Henry Williford for his friendship, assistance and advice, as well as Dr. Jennifer Brown for giving me the opportunity to begin my professional career at Auburn University Montgomery.

Most importantly, I am extremely grateful for my family and friends for the love and support they've given me throughout my life. My wife, Pam, has truly been a blessing and I am tremendously thankful for her patience, love, and support. I have been immensely blessed to have such wonderful people in my life, and I am forever grateful for the support that I have been given.

Journal format used: Medicine and Science in Sports and Exercise

Computer software used: Microsoft Word

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LIST OF ABBREVIATIONS

BMI	Body mass index
ECG	Electrocardiogram
HC	Hip circumference
HiFit	High fitness group
HR	Heart rate
HRV	Heart rate variability
HRR	Heart rate recovery
HR1	Heart rate at 1-minute post-maximal exercise
HR2	Heart rate at 2-minutes post-maximal 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
ModFit	Moderate fitness group
SNDD	Standard deviation of the normal to normal beats
HF	High frequency domain

LF:HF	High frequency to low frequency ratio
LF	Low frequency domain
pNN50	Proportion derived by dividing NN50 by the total number of R to R (Normal to Normal) intervals
RMSSD	Square root of the mean sum of the squares of differences between adjacent R to R (normal to normal) intervals
SDSD	Standard deviation of the differences between adjacent R to R (normal to normal) intervals
WC	Waist circumference
WHR	Waist to hip ratio

CHAPTER 1

INTRODUCTION

Compared to Caucasians, African Americans have a greater incidence of cardiovascular disorders and death at any age (12,39). Prevalence of cerebrovascular disorders is also significantly higher in African Americans (43). African Americans have a higher rate of hypertension, which can manifest in the teens and early twenties (65,102). The underlying mechanism behind these racial differences could partly be due to a dysfunctional autonomic nervous system (18).

The autonomic nervous system plays a critical role in the regulation of the cardiovascular system. The heart is myogenic, meaning that it spontaneously produces its own action potential for contraction. However, the two branches of the autonomic nervous system act in accord to maintain homeostasis of the heart's rate and contractility. Specifically, parasympathetic activity decrease heart rate and contractility, while sympathetic influence increase heart rate and contractility. The balance between sympathetic and parasympathetic activity at rest results in periodic fluctuations in heart rate. The oscillations that occur between adjacent QRS complexes, or more specifically the distance between successive R to R intervals as derived from an electrocardiogram (ECG) is known as heart rate variability (HRV).

Examining HRV has become a valuable, non-invasive procedure for analyzing cardiovascular autonomic influence (105). Typically, HRV is measured in both time and frequency domains. The time domain method involves the use of statistical procedures performed on ECG recordings, such as the standard deviation of R to R intervals, also known as N to N intervals (SDNN). The frequency domain method involves the transformation of the ECG recording into a power spectrum. The high frequencies (HF) of the power spectrum are purported to symbolize parasympathetic outflow (105). The low frequency (LF) bandwidths, while controversial (96), supposedly represent the combination of parasympathetic and sympathetic influences (105). The ratio between the two measures, LF:HF is believed to signify sympathovagal balance (105). Depressed HRV has been linked to abnormal cardiovascular autonomic modulation and is a valuable predictor of fatal and non-fatal cardiovascular events in clinical (61,90) and asymptomatic populations (110). Thus a high HRV is thought to be cardioprotective (105).

Whether differences exist in cardiovascular autonomic nervous control in African Americans compared to Caucasians is controversial. Lampert, et al., (69) concludes in their study that middle-age African American adults had lower HRV compared to their Caucasian counterparts. Similar results were also found in a younger group of African American men who were shown to have a higher sympathetic versus parasympathetic balance compared to non-African Americans (120). Choi, et al. (18) also reported that the young African American subjects had HRV indices comparable to older Caucasian subjects. In contrast, African American adolescents have been shown to exhibit a greater HRV at rest compared to age-matched Caucasians (45,113,115). Data from the ARIC

study (73) revealed greater parasympathetic influence, lower sympathetic activity, and more sufficient balance between parasympathetic and sympathetic influence in African American adults compared to their Caucasian counterparts.

Research suggests that fitness level, i.e., one's VO_{2max}, may also influence cardiovascular autonomic control (75). In addition, a higher body mass index (BMI) and waist to hip ratio (WHR) have been shown to exert a negative influence on sympathetic versus parasympathetic balance at rest (67). With respect to race, African Americans have been reported on average to have lower fitness levels and higher BMIs compared to Caucasians (54,70). Therefore, perhaps any existing racial differences in cardiovascular autonomic modulation could also be due to lower fitness levels and higher body mass indices. Interestingly, Gutin, et al., (45) reported that a more favorable HRV profile in African American youths was due to higher physical activity levels, better cardiovascular fitness, and lower body fat.

During exercise cardiac output (i.e., heart rate and stroke volume) rises with progressively graded work rates. This is due to a prompt decrease in parasympathetic activity followed by an increase of sympathetic influence (9,40). After exercise, the rapid return of heart rate towards resting is primarily due to parasympathetic reactivation followed by diminishing sympathetic outflow (42). The assessment of heart rate recovery (HRR) immediately after exercise has also become a useful non-invasive method for analyzing cardiovascular autonomic modulation (20,21,59). Lower/abnormal HRR is widely accepted as a predictor of premature cardiovascular disease and death (20,21,59,84,114,116). Healthy adults have been shown to have greater HRR compared to heart disease patients (2,56,57,91), those with the metabolic syndrome (25,62), and

type 2 diabetics (13,87).

Few studies have examined the relationship between HRR and resting HRV (6,31,58). Evrengul, et al. (31) suggested that HRR 3-minutes after submaximal treadmill exercise was significanlty associated resting HRV in a cohort of coronary artery disease patients. In contrast, two studies found no relationship between resting HRV and HRR 1-minute after submaximal (58) and maximal (6) exercise in healthy adults.

A number of studies have examined the acute effects of exercise on cardiovascular autonomic modulation. The results show that parasympathetic outflow is considerably reduced while sympathetic outflow is drastically heightened compared to resting (8,11,50,88,106). Brown and Brown (8) reported significant reductions in time domain parameters and normalized HF power after exercise in master trained athletes. Javorika, et al. (58) reported significant reductions in HRV up to 30 minutes after 8 minutes of stepping exercise at 70% of peak power output. Hautala, et al. (50) noted significant reductions in HF power up to twenty-two hours after prolonged exercise. Even greater reductions in parasympathetic outflow are found after high intense versus low intense exercise (88,98,106).

Significance of the Study

Although there have been a number of studies that have suggested differences in cardiovascular autonomic modulation in African Americans compared to Caucasians, the differences have yet to be fully clarified. African Americans have been reported to have lower (18,32,69), higher (45,113,115), and/or similar (34) HRV compared to Caucasians. This study adds to the current body of literature by analyzing resting HRV in the African

Americans and Caucasian subjects. In addition, as can be noted for the foregoing, the present cross-sectional data from the current body of literature on HRR and HRV after exercise has not addressed a possible effect for race. Therefore, a major purpose of this dissertation was to determine the effect of race (African Americans versus Caucasians) on HRR and HRV following a maximal graded exercise test.

Finally, there have been only a few studies that have examined the relationship between HRR and resting HRV. Evrengul, et al. (31) examined this relationship in a cohort of coronary artery disease patients after a submaximal treadmill test and found a significant negative relationship between HRR and resting sympathetic HRV markers, and a significant positive correlation between HRR and resting parasympathetic HRV variables. Javorka, et al. (58) also analyzed the relationship between resting HRV parameters and HRR. They found no correlation between the measures. However, the exercise protocol in the study was a submaximal 8-minute step test. This study was one of the first to analyze the relationship between HRV (at rest and following maximal exercise) and HRR following a maximal graded exercise test in African American and Caucasian men.

Assumptions

It was assumed that all subjects were apparently healthy, college-age men. In addition, it was assumed that each subject was not taking cardiovascular and/or anti-depressant medications. Last, it was assumed that each subject was able to perform a maximal graded exercise test on a treadmill.

Limitations

There are a number of limitations associated with the following studies. First, overall physical activity has been shown to exert a positive influence on autonomic influence of the heart (13). A difference in physical activity between the subjects was not assessed in this study. Second, it has been suggested that the racial differences in cardiac-autonomic modulation favor the black population at younger ages only to shift toward a lower HRV in older ages (45). The subjects in this study were limited to college-age men. Third, to further examine a physiological explanation to the racial differences in found HRV and HRR, it would've been helpful to examine arterial compliance and baroreflex sensitivity, which have been shown to relate to HRV (66,120). However, the author did not have access to the necessary equipment needed to analysis arterial compliance and the baroreflex loop. Fourth, this study addressed only the differences between African Americans and Caucasians. Other races, such as Hispanic and Asian was not examined in this study. Last, due to the cross-sectional nature of this project a causative factor to explain the differences, or variation, between the outcome variables could not be determined.

Operational Definitions

1) Heart rate variability (HRV) – a) Oscillations that occur between consecutive heartbeats, b) R to R interval variation, usually measured in milliseconds, on an electrocardiogram recording, c) non-invasive procedure for examining cardiovascular autonomic modulation.

2) Heart rate recovery (HRR) – The recovery in heart beat that occurs after a bout of exercise, which has been purported to be primarily due to the return of cardiac parasympathetic influence.
3) Autonomic nervous system – Branch of the peripheral nervous system that acts to maintain homeostasis within the physiological systems of the body. The ANS is composed of two branches, the sympathetic nervous system and the parasympathetic nervous system.
4) Sympathetic nervous system – Efferent arm of the autonomic nervous system that influences most adrenergic visceral receptors. Sympathetic influence to the heart increases its rate and force of contraction.
5) Parasympathetic nervous system – Efferent arm of the autonomic nervous system that influences most cholinergic visceral receptors. Parasympathetic activity to the heart decreases its rate and force of contraction.
6) Cardiovascular Autonomic Modulation – The influence of the autonomic nervous system on the cardiovascular system.
7) Time domain – used to describe signals with respect to time. Time domain analysis of HRV transforms the electrocardiogram recordings into a tachogram that plots the distance of each consecutive R-R interval against the number of total beats.
8) Frequency domain – used to describe signals with respect to frequency. When discussing HRV, the frequency domain method transforms the electrocardiogram into a power spectrum with the following frequency domains analyzed: high frequency (HF) and low frequency (LF).

9) Maximal graded exercise test (GXT) – a maximal exercise test that increases workload/intensity in a progressive manner, either in timed stages (i.e., Bruce protocol) or a slow, steady continuous manner (i.e., Ramping protocol).
10) Healthy, non-obese – A subject will be defined as healthy, non-obese if body mass index is between 20.0 and 29.9 kg/m^2 (American College of Sports Medicine, 2006).
11) Non-hispanic white – A person having origins in any of the original peoples of Europe, North Africa, or the Middle East .
12) Non-hispanic black – A person having origins in any of the black racial groups of Africa.

STATEMENT OF RESEARCH OBJECTIVES

The overall objective of this research was to provide a better understanding of cardiovascular autonomic modulation after exercise and its association with race, aerobic power, health-related anthropometric variables, and resting heart rate variability in a cohort of white and black college-aged men.

The specific objective of the first study was to determine the effect of race, independent of other known factors, on the following:

1. Resting heart rate variability.
2. Heart rate variability after maximal exercise.
3. The immediate drop in heart rate (i.e., heart rate recovery) after maximal exercise.

The specific objectives of the second study were the following:

1. To determine the effect of VO_{2max} on heart rate recovery, independent of race, body mass index, waist circumference, and body fat percentage.
2. To determine the degree of variation in heart rate recovery that could be explained with each of the following variables: race, VO_{2max}, body mass index, waist circumference, and body fat percentage.

The specific objective of the third study was the following:

1. To explore the relationship between heart rate variability and the recovery of heart rate after maximal exercise

CHAPTER 2

REVIEW OF THE LITERATURE

Overview

A study that involves analyzing cardiovascular autonomic control requires a detailed description of those portions of the nervous system that regulate cardiac activity. In the following paragraphs, an appropriate overview is provided.

Cardiac Activation

Cardiac muscle and skeletal muscle are similar in a number of ways. Both are striated, with myofibrils containing myosin and actin filaments, contract in a similar manner, by the sliding movement theory. However, there are many characteristics of cardiac muscle that are quite distinct from skeletal muscle. For instance, skeletal muscle relies strictly on extrinsic factors, action potentials from the central nervous system, for contraction. Cardiac muscle, on the other hand, is myogenic, meaning the fibers contract on their own, i.e., the fibers elicit action potentials without central nervous stimulation. Furthermore, cardiac muscle acts as a functional syncytium. That is, action potentials spread rapidly from cell to cell by way of special gap junctions known as intercalated discs. The right and left atria act as a separate syncytium (atria syncytium) from the right and left ventricles (ventricular syncytium). The atria and ventricals are separated by

fibrous tissue that surrounds the atrioventricular (A-V) valves, allowing both atria to contract before the ventricles.

Action potentials in cardiac muscle are 15 times longer compared to that of skeletal muscle. This is due to differences in ion channels within cardiac muscle. Cardiac muscle contains fast sodium channels that are similar to skeletal muscle. When activated, these channels allow for rapid influx of positive charged sodium ions, which depolarize the membrane. These channels quickly close to allow for repolarization. This is the only channel housed in skeletal muscle, i.e., is the only means for propagating action potentials. However, cardiac muscle also houses both "fast" sodium channels and slow calcium channels. Calcium channels also remain open for longer periods which cause a plateau phase in the action potential of cardiac muscle. During this plateau, potassium outflux is dramatically decreased. This allows for a prolonged refractory period in cardiac muscle fibers of approximately 0.15 seconds and 0.25 to 0.30 seconds in atria and ventricular cardiac cells, respectively. The prolonged refractory period also makes it difficult to re-excite a cardiac cell that's not fully recovered from stimulation.

Much like skeletal muscle, when an action potential is propagated across a cardiac muscle fiber's membrane and into its T-tubules, calcium is released from the lateral sacs of the sarcoplasmic reticulum and into the cytosol. Calcium can then bind to troponin, transforming the position of tropomyosin, and allowing for the strong connection of myosin head to the binding site of actin. There, myosin ATPase splits ATP, forming ADP and an inorganic phosphate, which energizes sliding of actin across the myosin chain, producing a muscle contraction. However, the sarcoplasmic reticulum of cardiac muscle fibers does not store high amounts of calcium compared to the sarcoplasmic

reticulum of skeletal muscle. In fact, if cardiac muscle fibers were to rely on this calcium source alone, the strength of contraction would be quite weak (46). Instead, large quantities of calcium ions diffuse from the T-tubules directly into the cytosol of a cardiac muscle cell. This is due to the presence of the aforementioned slow calcium channels. Thus, enough calcium is available for a sufficient contraction.

The heart's rate and force of contraction can be influenced by intrinsic or extrinsic factors. Intrinsic factors involve the amount of stretch of the atria and ventricles. An increase in venous return increases the stretch of the atria and ventricles. When the atria are stretched, the SA node increases its firing rate, which increase heart rate. Increasing the stretch of the ventricles causes a more optimal alignment of the myosin and actin filaments, which increases the heart's force of contraction. This is known as the Frank-Starling Law (46).

Extrinsic factors involve neural input. The autonomic nervous system is a key regulator of homeostasis of the heart (46). This system will be discussed in further detail in the following section.

The Autonomic Nervous System

The autonomic nervous system is the portion of the peripheral nervous system that controls most visceral functions within the body. For example, this system is an important modulator of cardiac activity, blood pressure, body temperature, gastrointestinal motility and secretions, urinary bladder emptying, sweating, etc. The autonomic nervous system is comprised of an afferent arm, which receives information

from visceral organs, and an efferent arm, which sends signals to visceral organs. The autonomic nervous system operates primarily by visceral reflexes (e.g., baroreflex, muscle metabo- and mechanoreflexes) and is divided into two sub-systems that are known as the sympathetic and parasympathetic nervous systems. Traditionally, the sympathetic nervous system has been described as having an excitatory and/or "fight or flight" effect. The parasympathetic nervous system has been described as having an inhibitory, "rest and digest" effect. However, a closer look at these two pathways will reveal that both branches can result in excitation or inhibition depending on the type of receptors that are innervated by each branch (refer to table 1).

The sympathetic nervous system originates in the spinal cord from spinal nerves T-1 to L-2. Each sympathetic pathway is composed of short preganglionic fibers that synapses with long postganglionic fibers that are described as either being para-verterbral or collateral ganglia. Para-vertebral ganglia are located in the cervical, thoracic, lumber, and sacral areas. The celiac and superior and inferior mesenteric neurons that innervate the gastrointestinal and urinary tracts are collateral ganglia.

The parasympathetic nervous system originates in cranial nerves III (Oculomotor nerve), VII (facial nerve), IX (glossopharyngeal nerve), and X (vagus nerve), and sacral spinal nerves S-2, 3, and 4. Most of the parasympathetic information travels via the vagus nerve. Thus, the parasympathetic nervous activity has also been described as vagal activity. The preganglionic fibers within parasympathetic pathways are long. They synapse with short postganglionic fibers that are usually located within the visceral organ being innervated.

Neurotransmitters are chemicals that are used to relay information between neurons and organs. Autonomic neurotransmitters are either adrenergic or cholinergic. Adrenergic fibers release mostly norepinephrine and a small percentage of epinephrine. Norepinephrine and epinephrine, along with dopamine, are the most abundant catecholamines within the body. Cholinergic fibers release the neurotransmitter acetylcholine. All preganglionic fibers within the autonomic nervous system are cholinergic. These fibers release acetylcholine onto receptors known as nicotinic-1 that are located on the postganglionic fiber.

Most sympathetic postganglionic fibers are adrenergic, with the exception of those that innervate sweat glands and piloerector muscles, which are cholinergic. These release norepinephrine and epinephrine onto visceral receptors known as alpha and beta receptors. Both of these receptors have different subtypes that are either excitatory or inhibitory in their actions. Also, some preganglionic sympathetic fibers innervate nicotinic-1 receptors located on the adrenal medulla. This produces an excitatory effect on the adrenal medulla and causes a release of norepinephrine and mostly epinephrine directly into the blood (i.e., circulating catecholamines).

All of the parasympathetic postganglionic fibers are cholinergic. These fibers release acetylcholine onto musarinic receptors of visceral organs. Muscarinic receptors can be are either excitatory (muscarinic-1 and 3) or inhibitory (muscarinic-2). For example, muscarinic-2 receptors are located on the sinoatrial node of the heart. When these are activated, heart rate decreases. In contrast, muscarinic receptors-1 and 3 are located on the gastrointestinal tract. When these are activated, gastrointestinal mobility and secretion increases.

Cardiovascular Autonomic Modulation

Even though cardiac muscle is myogenic, homeostatic control of the cardiovascular system is maintained via a balance between the two branches of the autonomic nervous system, which are the sympathetic and parasympathetic nervous systems (46). The autonomic nervous system acts to regulate the cardiovascular system in meeting certain demands placed on the body by influencing heart rate at rest, during and after exercise, and heart rate variability. The branch of the autonomic nervous system that is most active depends on the information relayed from the cardiovascular control center located in the pons and medulla of the brain stem. A discussion of each branch with regard to cardiovascular autonomic control will be undertaken in the following paragraphs.

Sympathetic fibers leave the spinal cord through the first four thoracic nerves (T-1 through T-4) and synapse with longer postganglionic fibers causing the release of epinephrine and norepinephrine on beta-1 receptors located on the SA and AV nodes, and the ventricular myocardium. Also, as stated before, certain sympathetic preganglionic fibers synapse directly with beta-receptors on the adrenal medulla, which causes the release of catecholamines (i.e., epinephrine and norepinephrine) into the blood allowing them to circulate throughout the body. The sympathetic effect causes excitatory postsynaptic potentials within the heart. This allows for the ion channels to open more rapidly, which increases the frequency of the action potentials across the heart's conduction pathway and the myocardium. Thus, the influence of the sympathetic innervation is both chronotropic and inotropic (i.e., increases heart rate and the force of contraction).

Sympathetic fibers also innervate certain blood vessels (e.g., small arteries, arterioles and veins). Sympathetic activity to small arteries and arterioles causes vasoconstriction, which decreases the amount of blood flow to certain organs. Sympathetic stimulation of veins also causes constriction, decreasing venous blood capacity, and increasing venous return to the heart. During rest, there is a continuous firing of the sympathetic nervous system causing constant, partial vasoconstriction. This is known as sympathetic vasomotor tone. During exercise, sympathetic impulse frequency increases, which acts to further increase the constriction of vessels that supply blood to inactive tissues, such as the kidneys and liver.

Parasympathetic fibers travel through cranial nerves III, VII, IX, and X and sacral nerves 1-4. Most of the parasympathetic fibers that innervate the heart do so via cranial nerve X (e.g., vagus nerve). Therefore, parasympathetic stimulation to the heart is also known as vagal influence. Long preganglionic vagal fibers synapse with short postganglionic fibers and secrete the neurotransmitter acetylcholine onto the muscarinic or cholinergic receptors of the sinoatrial (SA) and atrioventricular (AV) nodes, which are part of the heart's conduction system. In contrast to the sympathetic nervous system, the parasympathetic nervous system causes inhibitory postsynaptic potentials within the heart. The result is a decrease in the frequency of action potentials across the heart's conduction pathway and the myocardium. Thus, the influence of parasympathetic outflow to the heart causes a decrease in heart rate and force of contraction.

Under resting conditions the electrocardiogram (ECG) of a healthy individual (figure 1) will exhibit normal periodic variations in heart rate. These rhythmic oscillations occur with respiration (105) and have been described as the respiratory sinus

arrhythmia (119).

During expiration, cardiac parasympathetic preganglionic neurons slow heart rate (53,100). Conversely, during inspiration, inflation of the lungs inhibits cardiac vagal activity, which increases heart rate (53,100). Thus, resting heart rate is primarily under the influence of parasympathetic/vagal outflow and inhibition (105,119). Therefore, resting heart rate has been used as a crude indicator of autonomic control (107). Individuals with a higher heart rate have a reduced parasympathetic tone. However, it is not fully known whether training induced bradycardia that occurs after long-term endurance exercise is the result of increased parasympathetic influence at rest or other cardiac intrinsic factors (95).

Cardiovascular Adjustments During Exercise

During exercise, adjustments must be made within the cardiovascular system to meet the metabolic demands of active skeletal muscles. At rest, cardiac output is approximately 5 L/minute. Skeletal muscle receives about 15 to 20% of the total blood flow. During maximal exercise, cardiac output can exceed 20 L/minute. At this level of exertion, skeletal muscle blood flow increases to 80 to 85% of cardiac output. This is due to the increase in cardiac output and redistribution of blood flow from inactive tissues to active skeletal muscle that occurs during exercise. Therefore, the autonomic nervous system plays a critical role in making these adjustments.

For instance, heart rate, stroke volume, and myocardial contractility all increase due to a withdrawal of parasympathetic activity and an increase in sympathetic activity. During exercise, heart rate and stroke volume, i.e., cardiac output rises in proportion to

increasing exercise intensity. A heart rate increase of up to 100 beats/min is thought to be primarily due to parasympathetic withdrawal; with sympathetic predominance responsible for a further increase in heart rate (9,40). Sympathetic outflow to alpha-1 receptors on the arterioles that supply inactive tissue, such as the liver and kidney, is increased. This constricts the blood vessels which serve to shunt blood flow to these areas. Sympathetic influence to the arterioles that supply blood to the active skeletal muscles is overridden by local factors, such as nitric oxide, prostaglandins, adenosine diphosphate, inorganic phosphate, and lactate. Thus, vasodilation occurs within these arterioles. However, there is an upper limit to the extent of dilation that can occur within these blood vessels. If the arterioles that supply blood to active skeletal muscle were allowed to maximally vasodilate, blood pressure would plummet. But because of the feedback from mechano- and metaboreceptors, which are muscle afferents III and IV, respectively, this does not occur. When dilation reaches its upper limit, these receptors transmit information back to the cardiovascular control center. The cardiovascular control center then sends efferent sympathetic signals back to the arterioles to slightly constrict, i.e., override the local controlling vasodilation. None-the-less, the vasodilation of active skeletal muscle, the vasoconstriction of inactive tissue, and the increase in cardiac output allow for redistribution and increased blood flow to the active skeletal muscle during exercise.

Heart Rate Recovery

During the first minute of recovery from exercise, there is a prompt decay in heart rate towards resting level. This is primarily due to a rapid return of parasympathetic activity, with a decrease in sympathetic outflow occurring in the later stages of recovery,

i.e., more than 2 minutes following exercise. The examination of heart rate during the early post-exercise stage, i.e., heart rate recovery (HRR), has become a non-invasive marker of the parasympathetic nervous system (20,21,59,84).

To assess HRR, researchers, and/or clinicians use either ECG or high-quality heart rate monitors to examine heart rate immediately at the cessation of maximal or submaximal exercise and again at a certain time period during recovery, typically 60 seconds post-exercise. Heart rate recovery is the difference between heart rate obtained at maximal exercise and the heart rate obtained at 60 seconds post-exercise. Abnormal HRR has been defined as a drop in heart rate of $\leq$ 12 beats/min during the first minute of a post-exercise cool-down period (20). An abnormal HRR has been associated with chronic heart failure (2,56), atherosclerosis (57), hypertension (91), metabolic syndrome (25,62), type 2 diabetes (13), and glucose intolerance (87). Furthermore, a number of large-scale studies with thousands of subjects have found that a blunted HRR is an important predictor of all-cause mortality (20,21,59,84,114,116).

Heart Rate Variability

Historically, assessing cardiovascular autonomic control at rest, and during and after exercise was quite invasive, involving pharmacological interventions and/or surgical procedures (e.g., 33,35). More recently, a newer procedure has been developed that analyzes the oscillations that occur in heart rate (figure 1), known as heart rate variability (HRV). This measure has become a powerful non-invasive method for analyzing cardiovascular autonomic control and, as such, has significant applications in exercise physiology. Over a decade ago, the European Society of Cardiology and the North

American Society of Pacing and Electrophysiology issued a report entitled "Heart rate variability: standards of measurement, physiological interpretation and clinical use" (105). This report has been widely used as a guideline for HRV analysis. It's reported as being the third most cited text in *Circulation* (16) which illustrates the importance of HRV in being a reliable method for assessing cardiovascular autonomic modulation in research and clinical settings.

Heart rate variability is determined by examining the changes between adjacent QRS complexes (in milliseconds) as derived from an ECG. Specifically, the changes from R wave to R wave, known as R-R or N-N (normal-to-normal) intervals are examined. Heart rate variability from ECG can be measured for short intervals such as 5 minutes, or for longer periods such as 24 hours. The two most commonly used techniques for determining HRV are time domain and frequency domain methods (105).

The time domain method involves statistical calculations of the R-R intervals. Specifically, this technique converts the R-R intervals from the ECG recording into a tachogram (figure 2). The tachogram is a graph that represents the distance of each consecutive R-R interval in the y-axis and the number of total beats on the x-axis. There are a number of mathematical manipulations that are applied to the tachogram for assessing HRV when using the time domain method. Table 1 displays the time domain parameters that are most commonly used in the literature. Higher time domain values (i.e., SDNN, RMSSD, NN50, and/or pNN50) are desirable because higher values represent increased vagal influence, which is thought to be cardio-protective (105).

The frequency domain method is quite complex and involves the use of numerous mathematical algorithms (e.g., fast Fourier transform). The algorithms transform the

ECG data from time to frequency measures to form a power spectrum (see figure 3). Table 2 displays the major constituents of the power spectrum. The various power domains and the mechanisms they purportedly represent are often debated (1,27). The high frequency (HF) band is widely accepted as a representative marker of parasympathetic/vagal modulation. The low frequency (LF) band is purported to estimate both parasympathetic and sympathetic modulation (105). Both HF and LF bands are reported in values that are normalized to account for the changes in the total power of the entire power spectra (HFnu; LFnu). The LF:HF ratio is used as an indicator of overall sympathovagal balance (105). A Low LF:HF value represent sufficient HRV and has been found to be the most influential HRV determinant of mortality in patients with end stage renal disease (14). Most researchers agree that the HF band and LF:HF ratio accurately represent the mechanisms to which they are linked (105). The very low frequency (VLF) and the ultra low frequency (ULF) bands are not completely understood in terms of physiological mechanisms. However, it has been proposed that these very low frequencies could possibly represent thermoregulation (107) and/or the renin-angiotensin system (4). Total power is a measure of all frequencies under the waveform and is equal to the variance of the entire signal. Therefore, due to a possible lack of specificity, the use of total power remains controversial in both research and clinical applications (105). Thus, this study will utilize only the HF power and LF:HF ratio.

A high HRV is desirable and indicates sufficient parasympathetic control of heart rate (112). Reduced HRV indicates a decrease in parasympathetic tone and a hyperactive degree of sympathetic outflow (105) and has been associated with cardiovascular and cerebrovascular diseases/disorders (3,28,64,68,80). Evrengul, et al., (31) found

significantly lower SDNN, RMSSD, pNN50, HF power, and higher LF power and LF:HF ratio in coronary artery disease patients compared to healthy controls. HRV has been shown to be lower in patients with the metabolic syndrome (60) and obese subjects (29,77,92). Poor or reduced HRV is linked to a 5-fold increase in sudden cardiovascular related mortality (44,68). In addition, HRV is now thought to be a more powerful predictor of sudden death compared to any other cardiovascular disease marker, such as left ventricular dysfunction, particularly in myocardial infarction patients (64).

Time domain parameters, such as SDNN and RMSSD, have been validated as accurate measures of parasympathetic modulation after exercise. For instance, Goldberger, et al., (42) showed strong correlations between time domain parameters and parasympathic influence after exercise, with and without atropine. However, examining the frequency domain method during and after exercise with the use of traditional techniques, i.e., fast Fourier transformations, has been highly criticized (96). It seems that these techniques are most accurate at capturing autonomic control during steady state conditions. Thus, newer procedures have been developed that are purported to more accurately capture HRV during periods of non-steady state heart rate. For instance, poincare plot analysis is a method that has been developed to assess HRV during exercise. Tulppo, et al., (111) found significant reductions in parasympathetic markers captured by the Poincare plot during exercise combined with pharmacological blockade. In contrast, the more conventional fast Fourier method did not yield comparable results. These findings have been confirmed in other studies, as well (78,81), which indicates that analysis of HRV during exercise may necessitate the method of Poincare plotting.

It is not fully known whether HRV at rest can be associated with the recovery of heart rate (i.e., HRR) following exercise. This relationship has been examined in only a few studies which produced conflicting results. Javorka, et al., (58) compared SDNN, RMSSD, pNN50, LF power, and HF power to HRR after an 8-minute bout of stepping exercise in 17 healthy male subjects. The results showed no association between resting HRV and HRR. Bosquet et al. (6) also found no association between resting HRV and HRR. Evrengul, et al., (31) examined the association of similar HRV parameters to HRR after a submaximal treadmill exercise test to 85% of age predicted heart rate maximum in 38 healthy individuals and 38 coronary artery disease patients. In this study, HRR 3-minutes after the exercise test significantly correlated to SDNN ($r = 0.41$, $p < 0.01$), RMSSD ($r = 0.31$, $p < 0.01$), pNN50 ($r = 0.44$, $p < 0.01$), HF power ($r = 0.69$, $p< 0.01$), LF power ($r = -0.67$, $p < 0.01$), and LF:HF ratio ($r = -0.62$, $p < 0.01$). Correlation procedures were no used to compare resting HRV and HRR less than 3-minutes (31).

Heart Rate Variability and Acute Exercise

There is a large body of literature regarding assessment of HRV during and after exercise in the attempt to determine the effects of exercise on cardiac autonomic modulation. Significant reductions in HF power and increases in both the LF power and LF:HF ratio have been reported during exercise (55), indicating a shift from parasympathetic to sympathetic predominance. Furthermore, HRV is significantly depressed long after cessation of exercise. Studies have shown significant parasympathetic withdrawal following maximal exercise, as indicated by reductions in SDNN and HFnu (8,42). Javorika, et al., (58) reported significant reductions in HRV up

to 30 minutes after only 8 minutes of stepping exercise at 70% of peak power output. Hautala, et al., (50) reported depressed HRV parameters of up to 22 hours in subjects who completed a 75 km cross-country skiing race. HRV is even further blunted after high intensity versus low intensity exercise (88,106). Terzotti, et al., (106) showed a depressed HRV 15 minutes after exercise at 50% of the anaerobic threshold but up to 1 hour following exercise at 80% of the anaerobic threshold. When comparing two isocaloric bouts of exercise at differing intensities, Parekh & Lee (88) found significantly lower SDNN, HFnu, and higher LF:HF ratio after exercise at 80% compared to 50% of peak exercise. Seiler, et al., (98) showed a blunted parasympathetic recovery after exercise above versus below ventilatory threshold in highly trained endurance athletes. A clinical research investigation (17) compared HRV parameters following graded exercise between healthy versus myocardial infarction patients. The results showed significantly higher LF:HF ratio in the group of patients (17).

Fitness Status and Cardiovascular Autonomic Control

Research is available to suggest that a higher level of fitness (i.e., VO_{2max}) may lead to higher HRV and HRR. A number of cross-sectional studies suggest that both younger and older endurance trained and physically active subjects display greater resting HRV and HRR compared to non-athletic, sedentary, and clinical populations (11,23,24,26,41,56,75,85,99). However, research is also available that shows no difference in HRV between active and non-active individuals in response to a stressor, i.e., head up tilt (10,76). Also, Bosquet, et al., (6) compared subjects with similar VO_{2max} values but with differing levels of aerobic endurance, and found no differences in resting

HRV and HRR after a maximal graded exercise test. A large body of longitudinal data is also available suggesting that exercise training improves HRV in both healthy (48,49,72,79,95,118) and clinical populations (36,74,86,101,104,109). Melanson & Freedson (79) examined the effects of a 16-week moderate endurance training done 3 days per week, 30 minutes per session, at an intensity level of 80% heart rate reserve. The results showed a 13% increase in VO_{2max} along with increases in pNN50, RMSSD, and HF power after the training program (79). Six months of endurance training has also been shown to improve HRV in older and younger individuals (72). Raczak, et al. (93) examined HRV in competitive runners before and after a long-term preparatory training cycle. The results of the study revealed large increases in VO_{2max} and HRV time domain parameters, e.g., SDNN and pNN50. Iellamo, et al. (55) reported a decrease in LF and LF:HF ratio in world class rowers preparing for competition. Hedelin, et al. (55) investigated the effects of a 6-month competitive cross-country skiing season on HRV in young men and women. The results showed that all subjects had reduced LF power during a tilt test following the cross-country ski season (51). In clinical patients, Garet, et al. (36) reported that moderate to vigorous physical activity may counteract the decline in HRV that occurs with chronic heart disease.

Research examining the effects of exercise training on HRR has mostly focused on clinical, at risk, and older subjects. For instance, several studies have reported that patients who participated in cardiovascular rehabilitation programs that included aerobic exercise modalities, improved fitness and HRR while patients who did not participate exhibited either no improvement (71,82,103,108,117) or a further decline in fitness and HRR (37). Additionally, Christ et al. (19) reported that in subjects with the metabolic

syndrome, only those who incorporated exercise into a weight loss intervention improved HRR.

Race and Heart Rate Variability

African Americans have been reported to be more sedentary, less fit, and more overweight compared to Caucasians (22,54,70). African Americans also have a significantly higher rate of hypertension (5), which places them at an elevated risk for developing heart disease (38), end stage kidney disease (63), and stroke (94). Furthermore, data from the ARIC study suggests that the risk of developing type-2 diabetes was about 2.4 times greater for African American women and about 1.5 times greater for African American men than their Caucasian age-matched counterparts (7). In view of the fact that fitness level, body mass, cardiovascular disorders, and type 2 diabetes influence the autonomic nervous system (13,29,44,85), it's been proposed that racial differences exist in cardiovascular autonomic control (18,69,120). However, the extent of a racial impact has yet to be fully elucidated.

Lampert, et al. (69) concluded that African American adults with a mean age of 48 had a lower HRV compared to their Caucasian counterparts. The effect of race in their study was independent of other known modifiable risk factors, such as smoking and inactivity. Similar results were found in a younger group of African American men who were shown to have lower HF power, and a higher sympathetic to parasympathetic balance (i.e., LF:HF ratio) compared to non-African Americans (120). Faulkner, et al. (2003) reported lower 24-hour HRV (e.g., SDNN, rMSSD, pNN50, HF power) in African American adolescents. In addition, a study by Choi, et al. (18) showed that African

American subjects across many age groups had lower HRV compared to their aged-matched Caucasian counterparts. Moreover, the finding that the younger African American subjects had HRV indices comparable to that of older Caucasian subjects suggested that these individuals might show signs of premature aging of the autonomic nervous system (18).

Other measures of the autonomic nervous system have also been shown to be lower in African Americans. For instance, African Americans have been shown to have reduced baroreflex sensitivity compared to Caucasians (120). The baroreceptors are stretch sensitive receptors located in the arch of the aorta, the bifurcation of the carotid arteries, within the atria of the heart, and the pulmonary arteries. Homestatic reflex of the baroreceptors (i.e., baroreflex) regulates beat to beat blood pressure by promoting necessary changes in cardiac output (46). An impaired baroreflex is associated with reduced HRV (66,120).

Furthermore, a lower elastic compliance of major arteries (i.e., arterial stiffness) can result in blunted baroreflex sensitivity and HRV (15,30,83). African Americans have been shown to have a higher prevalence of arterial stiffness compared to Caucasians (52,120). In addition, an acute bout of aerobic exercise can temporarily result in increased arterial compliance in Caucasian, but not African American men (52). Thus, it's possible that African Americans may have an altered autonomic nervous control of the cardiovascular system not only at rest, but after exercise.

Not all data is in agreement with the aforementioned studies. Adolescent African Americans have been shown to exhibit a greater HRV at rest compared to age-matched Caucasians (45,115). Urbina, et al. (113) compared HRV responses between African

American and Caucasian adolescents during several autonomic challenge tests, e.g., orthostatic challenge (supine to standing), isometric handgrip, Valsalva maneuver, and cold pressor tests. The results demonstrated that African American subjects had higher pNN50 and lower LF:HF ratio compared to the Caucasian subjects for autonomic challenge tests (113). Data from the Atherosclerosis Risk In Communities (ARIC) study (73) showed greater parasympathetic influence, lower sympathetic activity, and more sufficient balance between parasympathetic and sympathetic influence in African Americans adults compared to their Caucasian counterparts. Franke, et al. (34) reported no difference in HRV in young-adult African American and Caucasian men. From theses studies, it is evident that the data regarding the effect of race, though somewhat sparse, on cardiovascular autonomic modulation is mixed and more research in the area of cardiac autonomic control would be valuable.

Interestingly, African Americans with diagnosed hypertension have been shown to have either superior or similar autonomic control compared to Caucasians. For instance, Guzzetti, et al. (47) found that African-Caribbean hypertensive subjects' LF power and LF:HF ratio were lower compared to the Caucasian hypertensive subjects. Other research is available that shows no racial differences in cardiovascular autonomic modulation in hypertensive patients (89,97).

The limited data examining the difference in cardiovascular autonomic function between white/Caucasian Americans and black/African Americans have produced variable results. Table 4 gives a summary of the selected studies that have examined the influence of race on HRV. Further research in this area is needed. Moreover, there are no studies available that have investigated the difference in HRR and post exercise HRV

between African American and Caucasian subjects. The effects of race on cardiovascular autonomic nervous control after exercise may prove valuable in clarifying differences beyond those found at rest.

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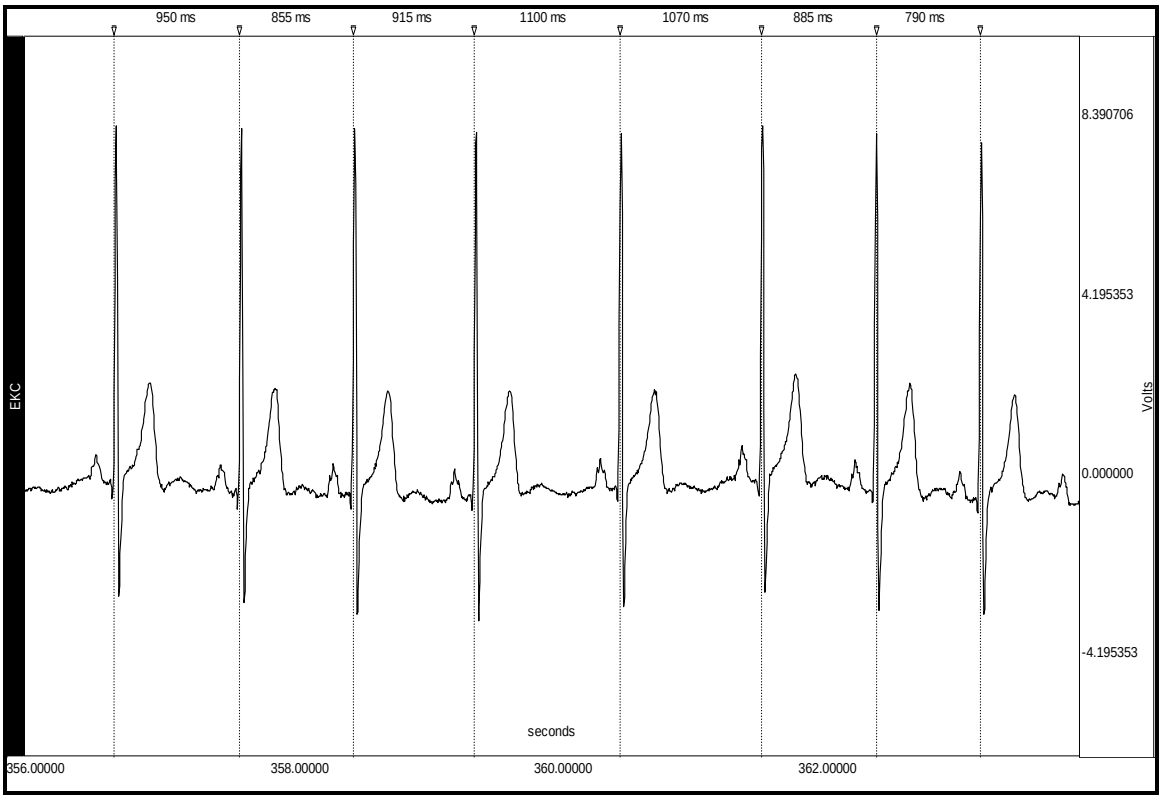

Figure 1. Electrocardiogram recording. Notice the variation in the R-R interval distances (ms).

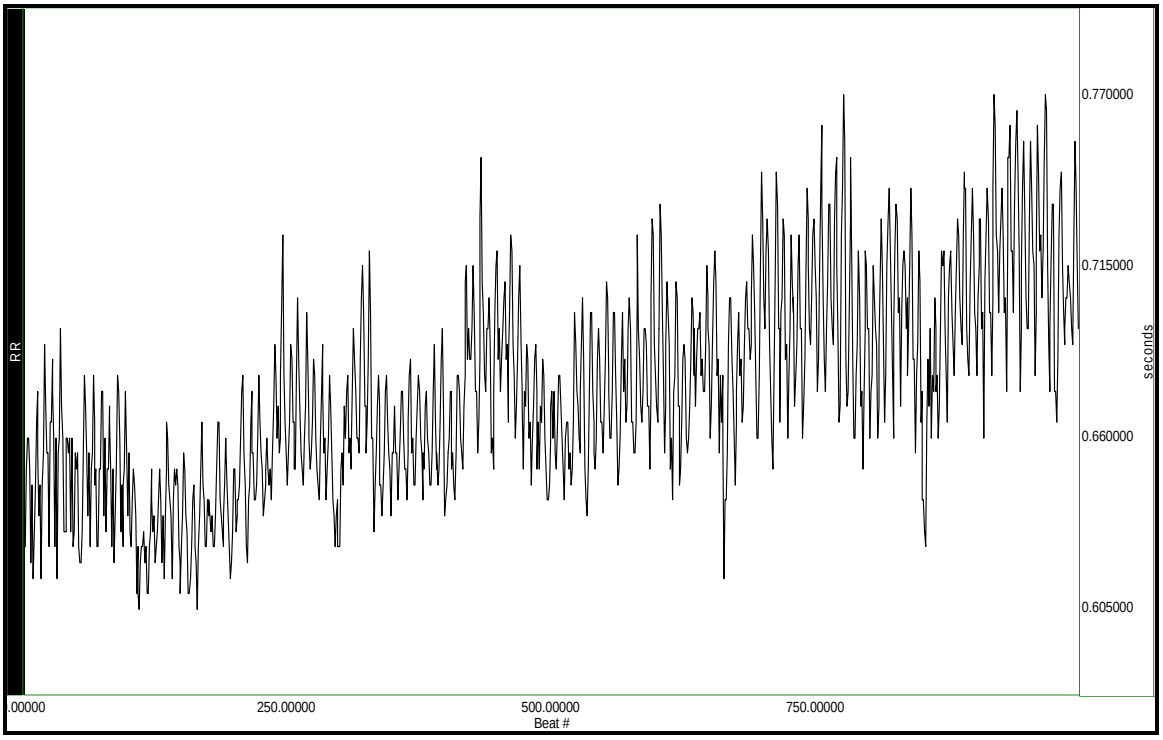

Figure 2. Tachogram derived from a 10-minute electrocardiogram. The R-R intervals (ms^2) are displayed on the y-axis. The x-axis plots the total number of beats recorded.

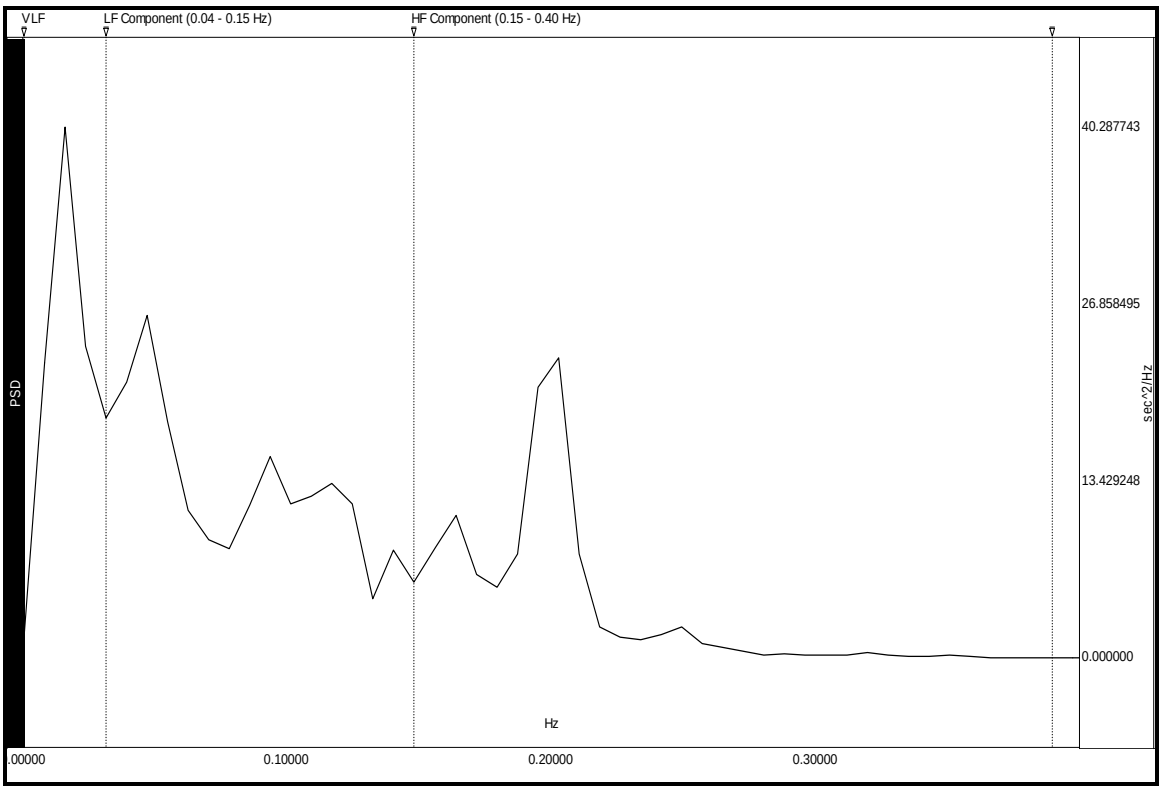

Figure 3. Power spectra density - The signal is transformed from time to frequency.

Table 1. Selected receptors located within the autonomic nervous system pathway.

Receptor	Location	Effect when stimulated
Nictonic-2	synaptic site of the pre- and post ganglionic fibers	cholinergic stimulation excites the post ganglion
Muscarinic-1	Exocrine sweat glands at the soles of the feet and palms of the hands	sympathetic cholinergic stimulation increase secretions
	Lacrimal (tear producing) and Nasopharyngeal (nasal mucus producing) glands	parasympathetic cholinergic stimulation increase secretions
Muscarinic-2	Sinoatrial node, atrioventricular node, and ventricular myocardium	parasympathetic cholinergic stimulation decrease heart rate and contraction

Receptor	Location	Effect when stimulated
Muscarinic-3	Smooth muscles of the gastrointestinal tract, alveolar sacs, and internal anal sphincter	parasympathetic cholinergic influence increases gastrointestinal motility and secretion, causes bronchioconstriction, dilates internal anal sphincter
	Ciliary muscle attached to the lens of the eye	parasympathetic cholinergic influence constricts the muscle which allows focus near objects
Alpha-1	apocrine sweat glands under arms and in the pubic region	sympathetic adrenergic innervation increase secretions
	Smooth muscles of arterioles	sympathetic adrenergic stimulation causes vasoconstriction

Receptor	Location	Effect when stimulated
Alpha-2	located at presynaptic sites	these receptors inhibit the release of norepinephrine from postganglionic
Beta-1	Sinoatrial node, atrioventricular node, and ventricular myocardium	sympathetic adrenergic stimulation increases heart rate and contraction
	Adipose tissue	sympathetic adrenergic outflow increase lipolysis
	Juxtaglomerular cells of the kidney	sympathetic adrenergic innervation increase renin production

Receptor	Location	Effect when stimulated
Beta-2	Ciliary muscle attached to the lens of the eye	sympathetic adrenergic influence relaxes muscle which allows focus on far objects
	Smooth muscles of the gastrointestinal tract, alveolar sacs, and internal anal sphincter	sympathetic cholinergic activity inhibits gastrointestinal motility and secretion, causes bronchiodilation, and constricts the internal anal sphincter

Table 2. Selected time domain parameters of HRV

Abbreviation	Parameter
SDNN	Standard deviation of all R-R (N-N) intervals
RMSSD	Square root of the mean of the sum of the squares of differences between adjacent R-R (N-N) intervals
SDSD	Standard deviation of differences between adjacent R-R (N-N) intervals
NN50	Number of interval differences of successive R-R (N-N) intervals greater than 50 ms
pNN50	Proportion derived by dividing NN50 by the total number of R-R (N-N) intervals

Table 3. Frequency domain parameters of HRV

Abbreviation	Parameter and Representationof
VLF (ms^2)	Very Low Frequency Power (0.0033 – 0.04Hz)
	Representation of: unknown
LF (ms^2)	Low Frequency Power (0.04 – 0.15 Hz)
	Representation of: sympathetic and parasympathetic modulation
HF (ms^2)	High Frequency Power (0.15 – 0.40 Hz)
	Representation of: parasympathetic modulation
Total Power (ms^2)	Total Variance of all N-N intervals (< 0.40 Hz)
	Representation of: total heart rate variability
LF:HF	Ratio of LF to HF
	Representation of: sympathetic-parasympathetic balance
LFnu	LF in normalized units (n.u.) = LF/(Total Power – VLF) x 100
	Representation of: sympathetic and parasympathetic modulation
HFnu	HF in normalized units (n.u.) = HF/(TotalPower – VLF) x 100
	Representation of: parasympathetic modulation

Table 4. Selected studies that have examined racial differences in heart rate variability.

Authors, Date	Subjects	Duration of and activity during HRV assessment	Findings
Sloan et al., 2008	789 subjects (44% CA, 56% AA) between 33 – 47 years	10-min seated rest	CA had ↑ LF, and ↑ SDNN compared to AA subjects
Choi et al., 2006	135 subjects (n = 78 CA, n = 57 AA) Aged between 23 – 57 years	3-min HRV assessment in the seated position	CA had ↑ LF and ↑ HF compared to AA: ↔ LF:HF ratio between subjects Younger AA displayed HRV patterns similar to the older CA subjects
Gutin et al., 2005	304 subjects (n = 145 CA, n = 159 AA) Aged between 14 – 18 years	After 10-min supine rest, 256 consecutive R-R intervals were analyzed for HRV assessment	HRV profile more favorable in AA (i.e, AA had ↑ RMSSD, ↑ HF, ↓ LF:HF ratio) despite SBP being higher in AA,

Lampert at al., 2005	282 subjects of differing races (n = 214 CA, n = 41 AA) Mean age 48 ± 17 years	24-hr Holter monitor recordings	CA had greater HRV compared to AA, i.e, ↑ LF, ↑ HF, and ↑ SDNN in CA)
Wang et al., 2005	166 subjects (n = 103 CA, n = 63 AA) Mean age 16 ± 2 years	After 10-min supine rest, 256 consecutive R-R intervals were analyzed for HRV assessment	AA had ↑ RMSSD, ↑ HF, ↑ LF, ↓ LF:HF ↑ RMSSD: indicating greater HRV in AA compared to CA youth
Liao et al., 1995	1,984 subjects (n = 109 CA; n =107 AA) Aged 45 – 61 years	After 10-min supine rest, 256 consecutive R-R intervals were analyzed for HRV assessment	HRV profile more favorable in AA ↑ HF, ↑ HF:LF ratio (i.e., ↓ LF:HF ratio),
Guzzetti et al., 2000	52 hypertensive subjects n = 26 CA, mean age 45 ± 2 years n = 26 AA, mean age 46 ± 2 years	24-hr Holter monitor recordings	The AA patients had greater HRV compared to the CA patients (i.e., ↓ LF and ↓ LF:HF ratio in AA)

Zion et al., 2003	61 subjects (n = 32 AA, n = 29 NAA) Aged between 21 – 24 years	15-min seated rest of which 5-min was used for HRV	HRV profile more favorable in NAA NAA had ↑ HF and ↓ LF:HF ratio compared to AA.
Urbina et al., 1998	39 male subjects (50% AA) Aged 13 – 17 years	HRV assessed during: supine, standing, a 20% max isometric hand grip, Valsalva maneuver, and hand immersion in water at 4 degree Celsius	HRV more favorable during all tests in AA compared to CA youth subjects (i.e., AA had ↓ LF:HF and ↑ pNN50)

CA = Caucasian; AA = African American; NAA = Non-African American; HRV = heart rate variability; HF = high frequency power, a frequency domain marker of parasympathetic activity; LF = low frequency power, afrequency domain marker of both parasympathetic and sympathetic nervous activity; LF:HF = ratio of low frequency to high frequency power, a frequency domain marker of sympathetic-to-parasymathetic activity; SDNN = standard deviation of normal-to-normal heart beat intervals, a time domain marker of overall heart rate variability; RMSSD = square root of the mean of the sum of the squares of differences between adjacent normal-to-normal heart beat intervals, a time domain marker of parasympathetic activity; pNN50 = percentage of consecutive normal-to-normal heart beat intervals differing by more than 50%, a time domain marker of parasympathetic activity

CHAPTER 3

INFLUENCE OF RACE ON CARDIAC AUTONOMIC FUNCTION AT REST AND FOLLOWING MAXIMAL EXERCISE

Abstract

The purpose of this study was to determine if race has an independent effect on heart rate recovery (HRR) and heart rate variability (HRV) subsequent to a maximal graded exercise test (GXT). Sixty men (30 African Americans and 30 Caucasians) were volunteered as study subjects. Electrocardiogram (ECG) recordings were obtained in a supine position during a 10-minute baseline period pre exercise (PRE). All subjects performed a maximal graded exercise test on a treadmill. After exercise, ECG recordings were taken during a 30-minute post-exercise period (POST). The ECG recording taken during six distinct 5-minute epochs was used to analyze HRV as follows: PRE 5 – 10, POST 5 – 10, POST 10 – 15, POST 15 – 20, POST 20 – 25, and POST 25 – 30 minutes. The HRV was assessed in the frequency domain: i.e., log transformed normalized HF power (lnHF) and log transformed normalized LF:HF ratio (lnLF:HF). HRR was determined from the difference between HR recorded at maximal exercise and the HR recorded at 1-minute (HRR1) and 2-minutes (HRR2) post-exercise. A group effect was detected for HRR and HRV. Black men had greater HRR2 compared to the white men. Black men also had greater lnHF at rest and post exercise, and lower

lnLF:HF at rest and post exercise ($p < .05$). The results of this study suggest that young black men have a more favorable cardiovascular autonomic response after maximal exercise compared to young white men.
KEY WORDS: African American, Heart rate variability, Heart rate recovery

INTRODUCTION

Compared to Caucasians, African Americans suffer from greater cardiovascular disorders and death at any age (3,8). Furthermore, African Americans have a higher rate of hypertension, which can manifest itself in their teens and early twenties (18,32). On average, African Americans have also been reported to have lower fitness and higher Body Mass Index (BMI) levels compared to Caucasians (21). Generally, individuals who are at higher prevalence of cardiovascular disease (6,15,17), hypertension (19,28), sedentary lifestyle (4,24), and obesity (20) have been shown to have a dysfunctional autonomic nervous system. Therefore, it has been suggested that differences also exist in cardiovascular autonomic modulation between the Caucasian/White and African American/Black populations.

The autonomic nervous system plays a critical role in the regulation of the cardiovascular system. The heart is myogenic, meaning that it spontaneously produces its own action potential for contraction. However, the two branches of the autonomic nervous system act in accord to maintain homeostasis of the heart's rate and contractility. Specifically, parasympathetic activity decreases heart rate and sympathetic influence increases heart rate and strength of contraction. During exercise cardiac output rises with

progressively graded work rates because of a prompt decrease in parasympathetic activity followed by an increase sympathetic outflow (2,9). After exercise, heart rate quickly begins to return towards resting. This is primarily due to parasympathetic reactivation followed by diminishing sympathetic outflow (14). The assessment of heart rate recovery (HRR) immediately after exercise has become a useful method for analyzing cardiac autonomic modulation (6). Lower/abnormal HRR is widely accepted as a predictor of premature mortality independently of other markers of cardiovascular disease (6,16).

The oscillations that occur between adjacent QRS complexes, or more specifically the distance between successive R to R intervals, as derived from an electrocardiogram (ECG) is known as heart rate variability (HRV). Examining HRV is also a valuable procedure for analyzing autonomic influence (33). Typically, HRV is measured in both time and frequency domains, although other methods have been developed. The time domain method involves the use of statistical procedures performed on ECG recordings, such as the standard deviation of R to R intervals (SDNN). These values primarily represent vagal influence (33). The frequency domain method involves the transformation of the ECG recording into a power spectrum. The high frequencies (HF) of the power spectrum are purported to symbolize parasympathetic outflow (33). The low frequency (LF) bandwidths, while controversial, supposedly represent the combination of parasympathetic and sympathetic influences (33). The ratio between the two measures, LF:HF ratio is said to signify sympathovagal balance (33). Depressed HRV has also been linked to abnormal cardiovascular autonomic modulation, and is a valuable predictor of fatal and non-fatal cardiovascular events in clinical (17,29) and

asymptomatic populations (34).

Although evidence is available to suggest that African Americans differ from Caucasians in cardiac autonomic function, the extent of this difference has yet to be fully elucidated. For example, Lampert et al. (20) concludes that African American middle-age adults have lower HRV compared to their Caucasian counterparts. The effect of race noted in their study was independent of other known modifiable risk factors, such as smoking and inactivity. Similar results were found in a younger group of African American men who were shown to have a higher sympathetic to parasympathetic balance compared to non-African Americans (37). Choi et al. (5) also reported that the young African American subjects had HRV indices comparable to older Caucasian subjects. Data from the Coronary Artery Risk Development in Young Adults (CARDIA) have shown that black men have greater HRR compared to white men, and black and white women (4).

Not all data is in agreement with the aforementioned studies. Adolescent African Americans have been shown to exhibit a greater HRV at rest compared to age-matched Caucasians (10,35,36). Data from the ARIC study (22) demonstrated greater parasympathetic influence, lower sympathetic activity, and more sufficient balance between parasympathetic and sympathetic influence in African Americans adults compared to their Caucasian counterparts. Thus, the data regarding the effects of race on cardiac autonomic control are mixed and more research in this area is needed.

Currently, there are no studies available that has examined the influence of race on cardiovascular autonomic modulation, via both HRV and HRR, after maximal exercise. The aim of this investigation was to determine the effect of race, independent

of other known factors (such as VO_{2max} and BMI), on HRR and HRV at rest and following a maximal exercise test in a group of white and black college-age men.

METHODS

Participants

Sixty apparently healthy men (30 white and 30 black) volunteered to participate in this study. All data was collected in the Human Performance Laboratory at Auburn University Montgomery (AUM). This study was approved by the Institutional Review Board (IRB) for Human Participants. All participants had no history or clinical sign of cardiovascular or pulmonary diseases and were non-smokers. The subjects gave informed consent in writing and completed health history questionnaires to qualify them for the study. All participants were normotensive (i.e., blood pressure < 140/90 mmHg), not currently taking any prescribed blood pressure or anti-depressive medications, and displayed normal electrocardiogram (ECG) patterns. Subjects reported ethnicity/race as either Non-hispanic/White or Non-hispanic/Black over three generations.

Experimental Design

All data was collected for each subject on one visit to the lab during one of two 2-hour time slots: either between 7:00 and 9:00am, or between 9:00 and 11:00am, on any day of the week. The subjects were instructed to not consume alcoholic or caffeinated beverages 24 hours before the test, and to not eat at least 3 hours before the test. Upon entry into the lab, subjects were given verbal instruction to familiarize them of the testing procedures. After completing the necessary screening form and providing informed consent in writing, body weight and height were measured. Body fat percentage was then

estimated with a 7-site skinfold technique. Once anthropometric variables were assessed, resting HRV was examined for a 5-minute period while each subject assumed a supine position. After the 5-minute period, resting blood pressure was assessed with the use of standard auscultatory techniques while the subject remained in the quiet supine position. Then, each subject performed a graded maximal aerobic exercise test on a motor driven treadmill. HRR was analyzed during 2-minutes of the cool-down period. After the subjects completed the cool-down period, they once again assumed a supine position for 30-minutes. During this time, post-exercise HRV was analyzed.

Anthropometric variables

Height was measured with a wall mounted stadiometer (SECA) and rounded to the nearest 0.5 cm. Body weight was measured with a digital scale (TANITA BWB-800A) and rounded to the nearest 0.01 kg. BMI was calculated as height divided by weight squared (kg/m^2). Body fat percentage was estimated via the 7-site skinfold method as described by written standards (1).

Maximal Graded Exercise Test

All subjects performed a maximal graded exercise test on a Parker Treadmill (Parker Co., Opelika, AL). The Bruce protocol was employed during each exercise test. Specifically, the Bruce protocol incorporates a series of 3-minute stages with progressively increased workloads (i.e., speed and grade) until the subjects meets the criteria for VO_{2max}. During the test, an Applied Electrochemistry (AMETEK, Pittsburg, PA) metabolic analyzer was used to determine the concentration of expired gases (oxygen and carbon dioxide) via a continuous manner at the mouth with a pneumotach. All data was recorded on a personal computer every 30-seconds using Turbofit 5.06

software (VACUMED, Ventura, CA). Maximal oxygen consumption was considered to be achieved if two of the following criteria occur: a plateau in VO_2 with increasing work rate; RER $\geq$ 1.10; heart rate within 10 beats of age predicted maximum (220 – age); or volitional fatigue. Heart rate (HR) was monitored during the test with a Polar electronic heart monitor (Polar Electro Oy, Kemple, Finland). Blood pressure was measured during the last 45 seconds of each stage with a standard sphygmomanometer and stethoscope. Once the subject achieved VO_{2max}, a 3-minute cool-down period was allowed. During this time, the speed was decreased to 2.5 mph at a 1.5% grade.

Heart rate variability

In order to analyze HRV, electrocardiographic (ECG) recordings were examined before and after exercise. A modified Lead II configuration using three Ag/AgCl electrodes (BIOPAC ES509) was used for the ECG recordings. The electrodes were interfaced with a Biopac MP100 data acquisition system (Goletta, CA). All data was stored in a designated PC for analysis. Before exercise, each subject assumed a supine position. During this time, subjects maintained their normal breathing patterns. The subjects remained in this position for 10 minutes prior to the exercise test. Following exercise, subjects once again assumed a supine position for 30 minutes.

The ECG recordings were divided into six 5-min segments as follows: One PRE epoch, which was the last 5 minutes of the 10 minute baseline recording; and 5 POST epochs at 5 – 10, 10 – 15, 15 – 20, 20 – 25, and 25 – 30 minutes. All 5-minute ECG recordings were visually inspected. Any ectopic/non-sinus beats were removed and replaced by the adjacent normal R-R interval. If three or more ectopic beats were found

within any ECG segment, the reading was excluded from analysis. HRV was analyzed in the frequency domain. A power spectral analysis was completed on the ECG by applying a Hanning window and a fast Fourier transformation to the R-R intervals. In the frequency domain, HRV was separated into high frequency (HF) power (0.15-0.40 Hz), low frequency (LF) power (0.04 – 0.15 Hz). Both of these values were normalized (HFnu, LFnu) to account the influence total power of the entire wave and the very low frequency (VLF) band (0.0033 – 0.04 Hz) as follows: HFnu = HF/(Total power of the entire wave – VLF) x 100; LFnu = LF/(Total power of the entire wave – VLF) x 100. HFnu was recorded during the resting and post-exercise time intervals and utilized as a marker of parasympathetic modulation. The LF:HF ratio was also recorded during the selected time intervals and used as an index of sympatho-parasympathetic balance. For the purpose of this study, LFnu was not recorded for analysis.

Heart rate recovery

Heart rate was monitored continuously during the recovery period. To examine HRR, the heart rate that corresponded to VO_{2max} (i.e., the maximal heart rate), the heart rate at 1-minute cool-down (i.e., HR1) and the heart rate at 2-minutes cool-down (HR2). HRR was determined from the difference between MHR and the HR recorded at 1-minute (HRR1) and 2-minutes (HRR2) post-exercise.

Statistical Analysis

All statistical analysis was completed using SPSS version 16.0. Significant differences for descriptive variables (Table 1) between black and white men were assessed by one-way analysis of variance (ANOVA). A 2 (group) by 3 (time – MHR, HR1, HR2) repeated measures ANOVA was utilized to determine a group by time effect

for the change in HR after exercise. A one-way ANOVA was also utilized to determine significant differences between the mean values of HRR1 and HRR2 between the two groups. If a significant difference was noted between either HRR1 and/or HRR2, then an analysis of covariance (ANCOVA) was also performed with the following potential confounders: RHR, mean arterial pressure (MAP), VO_{2max}, and body mass (weight, BMI).

A natural logarithm transformation was used to normalize the distributions of all the HRV parameters (i.e., lnHF and lnLF:HF, PRE and POST). To determine the effect of race on HRV at rest, an ANCOVA was utilized for each variable (lnHF and lnLF:HF) with the aforementioned potential confounders as covariates in the analysis. To assess group differences after exercise in HRV, ANCOVA was performed at each POST epoch with resting HRV as the covariate. The ANCOVA procedure was then repeated with each of the potential confounders mentioned above.

A 2 X 6 (2 groups X 6 time points) mixed model repeated measures was also used to assess group differences for both lnHF and lnLF:HF over time (PRE, POST 5-10, POST 10-15, POST 15-20, POST 20-25, and POST 25-30).

Finally, to determine any significant differences from the first POST HRV recording to the last POST HRV recording within each race, one-way ANOVAs were utilized to compare each HRV parameter at POST 5-10 and POST 25-30. A priori statistical significance for all tests was set at $p < 0.05$.

RESULTS

All subjects that agreed to participate completed the testing procedures. Table 1 represents the subjects descriptive characteristics. The white subjects had significantly lower DBP, and MAP, and significantly higher VO_{2max}, and WHR values ($p < 0.05$, refer to Table 1). There was no significant difference in MHR and HR1 between the white (MHR = 190.17 ± 8.95, HR1 = 170.70 ± 11.91) and black (MHR = 187.67 ± 8.65, HR1 = 166.87 ± 11.59) subjects. However, the white subjects had significantly higher HR2 compared to the black subjects (149.73 ± 13.89 versus 141.73 ± 13.48, respectively, $p < 0.05$). The repeated measures procedure revealed a racial difference in the change in HR after exercise (Figure 1; $p < 0.05$). During the 2-minute recovery period, blacks had a faster trend for recovery compared to whites. Furthermore, there was not a difference in HRR1 (18.63 ± 6.27 beats/min for whites and 20.73 ± 6.17 beats/min for blacks), but the white subjects had lower HRR2 compared to the black subjects (39.93 ± 8.97 and 45.86 /- 9.05 beats/min, respectively, $p < .05$, Figure 2). According to the ANCOVA procedures, the racial difference in HRR2 remained when controlling for all of the potential confounders ($p < .05$).

Table 2 represents group means for resting HRV. Blacks had significantly higher HFnu and lnHF, and significantly lower LF:HF and lnLF:HF, at rest versus whites ($p < .05$). These differences also remained when controlling for the potential confounders ($p < .05$).

Blacks also had significantly higher lnHF and statistically lower lnLF:HF at POST 10-15, POST 15-20, POST 25-30 and POST 25-30. The racial differences in both HRV variables at POST 10-15, POST 15-20, and POST 25-30 remained after controlling

for PRE values (i.e., lnHF and lnLF:HF at rest), RHR, MAP, VO_{2max}, weight, and BMI (figures 2 and 3). Although blacks had a more favorable HRV profile during the 30-minute recovery period, according to the repeated measures procedure, the trend for recovery was not statistically significant. However, one-way ANOVAs suggested that lnHF and lnLF:HF at POST 25-30 was significantly different than POST 5-10 in the black subjects but there was no difference between the two time points in the white subjects (figure 2 and 3).

DISCUSSION

Both HRV and HRR are two widely used non-invasive procedures that analyze cardiac autonomic modulation. There are a number of studies available that have examined racial differences in cardiac autonomic function (i.e., HRV) at rest (5,10,11,20,22,31,35). This is the first study to report a racial difference in cardiovascular autonomic function after exercise, i.e., differences in post-exercise HRV and HRR. The major findings of this investigation were that young black men had faster HRR after maximal exercise and a more favorable HRV profile at rest and following maximal exercise compared to age-matched white men.

The research available that has examined the influence of race on resting HRV has yielded contradictory results. Liao et al. (22) were among the first to determine if a difference exists in HRV between races. They studied a cohort selected from the Atherosclerosis Risk in Communities (ARIC) study that consisted of 1,544 white and 440 black men and women between the ages of 45 and 64 years of age. Their results indicated that black subjects had lower LF power and higher HF power compared to the

white subjects. Sloan et al. (31) also examined the effects of race on cardiac autonomic function (e.g., HRV) in a large study that consisted of 757 subjects and found LF:HF ratio was lower in blacks compared to whites. Furthermore, black hypertensive patients have been shown to have lower normalized LF power and lower LF:HF ratio over a 24-hour period compared to white counterparts (11). In a group of adolescent boys (13 to 17 years of age), LF:HF ratio was lower and the time domain HRV parameter, pNN50, was higher in blacks versus whites (35). Moreover, the results of two studies (10,36) suggests that black youth subjects have a more favorable HRV profile during supine rest compared to age-matched whites. The current study's results concur with these findings. The black male subjects had greater HF power (HFnu and lnHF) and lower LF:HF ratio during supine rest compared to the white subjects.

Not all studies agree with these findings. For example, Choi et al. (5) found lower HF power in blacks compared to whites. No difference was noted for LF:HF ratio. In a group of outpatient subjects referred for Holter monitoring, Lampert et al. (20) suggested that whites have greater HRV over a 24-hour period than blacks. Moreover, in a group of subjects that were of similar ages to the present study, blacks had lower lnHF and higher LF:HF ratio compared to whites (37).

The fall in heart rate immediately after exercise is highly dependent on how quickly parasympathetic influence returns to lower cardiac activity (14). The measure of HRR has grown in popularity as a non-invasive tool to assess cardiac autonomic function (14). In the current study, compared to the white subjects, the black subjects also had a greater drop in heart rate two-minutes after reaching VO_{2max} (HR2 = 149.73 ± 13.89 and 141.73 ± 13.48; HRR2 = 39.93 ± 8.97 and 45.86 ± 9.05, in blacks and whites,

respectively). These results are in agreement with previous findings (4). In a large study, black men had greater HRR compared to white men, and white and black women (4). However, post-exercise HRV was not analyzed and, racial differences in aerobic fitness were not reported (4). Those with higher aerobic fitness generally have faster HRR (7). However, in the current study, the black men had lower aerobic power but greater 2-minute HRR.

The black subjects also had higher HF power and lower LF:HF ratio at rest and during the 30-minute, post-exercise period. Since those with higher resting HRV also have higher HRV post-exercise (15), an ANCOVA procedure was utilized to control for the influence of HRV at rest. This result showed that the difference in HRV at 10-15, 15-20, and 25-30 remained after controlling for the influence of HRV at rest (figure 2 and 3). In addition, both lnHF and lnLF:HF at POST 25-30 was significantly higher and lower, respectively, than POST 5-10 in the black subjects. However, no difference between the two time points was found in the white subjects (figure 2 and 3). Since parasympathetic influence is greatly reduced after exercise (14), these findings are indicative of a faster parasympathetic rebound after exercise in the black compared to white men.

As mentioned previously, the black subjects in the current study had a more favorable cardiac autonomic profile despite them having lower aerobic power (i.e., VO_{2max}) values compared to whites (42.03 ± 6.89 and 47.23 ± 6.99 ml/kg/min, blacks and whites, respectively). This is very intriguing because evidence is available to suggest that fitter subjects have a more favorable HRV profile (24,30) and a faster HRR (7,14,28).

Blacks have been shown to have a higher prevalence of hypertension compared to whites (18,32). The black men in the current study had significantly higher MAP

compared to the white subjects (88.3 ± 7.2 vs 92.9 ± 6.5 mmHg, respectively, $p < .05$). This, too, is interesting because blood pressure has been shown to have a negative impact on cardiovascular autonomic function (27). Since most research, along with the current study, suggests that blacks have more favorable autonomic control of heart activity, it is doubtful that differences in cardiovascular autonomic modulation contribute to the discrepancy in blood pressure.

Low arterial compliance has been suggested as a possible contributor to development of hypertension in the black population (37). By using a technique known as pulse wave velocity (PWV), Zion et al. (37) showed that blacks had significantly less arterial compliance compared to whites. Maximal aerobic exercise purportedly leads to an immediate and transient reduction in arterial stiffness (26). This is a possible mechanism explaining the acute hypotensive response to aerobic training (26). However, blacks differ from whites in the post-exercise adjustment in arterial compliance. By comparing a group of black to a group of white college-aged men, Heffernan et al. (12) revealed a decrease in arterial stiffness in the white group, but no difference in arterial stiffness in black subjects after maximal exercise. The current study did not assess arterial compliance. However, if the black subjects in this study had a reduced arterial compliance after exercise, then perhaps the increased cardiac-vagal activity (i.e., higher HF power and lower LF:HF ration) seen in the black subjects after exercise could be a method to attempt to regulate homeostasis of blood pressure during the post-exercise period. Further research is necessary to determine if such a relationship exists.

To help determine a physiological explanation to the racial differences in found HRV and HRR, it would've been helpful to examine arterial compliance and baroreflex

sensitivity, which have been shown to relate to HRV (25,37). However, the author did not have access to the necessary equipment needed to analysis PWV and the baroreflex loop. This should be considered in future research.

In conclusion, this cross-sectional study is the one of the first to suggest that cardiovascular autonomic modulation differs following maximal exercise between college-aged white and black men. The black men in this study had a more favorable heart rate recovery and a superior HRV both at rest and after exercise compared to the white men. These findings were significant despite the black subjects having significantly higher resting mean arterial blood pressure and significantly lower VO_{2max} values. Clearly, further research in this area is warranted.

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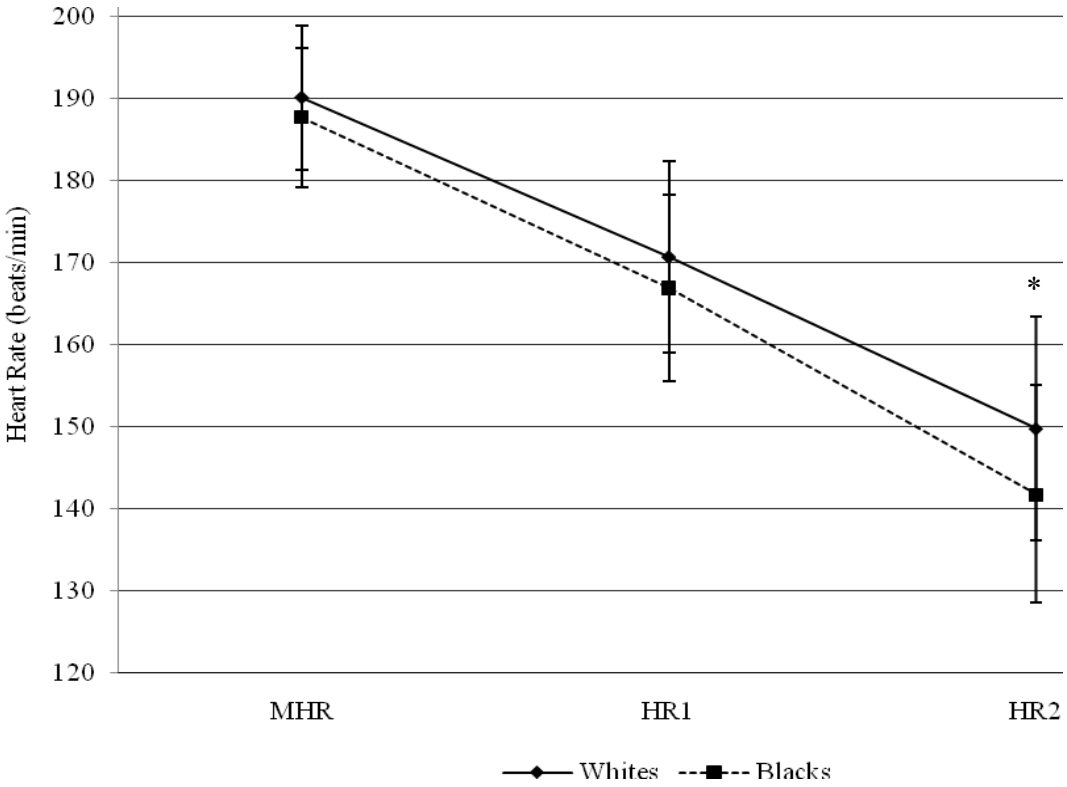

Figure 1 - Heart rate responses post-maximal exercise between groups

There was no difference between whites and blacks in maximal heart rate (MHR) or the heart rate at 1-minute post-exercise (HR1). The black men had significantly lower heart rates at 2-minutes post-exercise (HR2) compared to the white men (* $p < 0.05$).

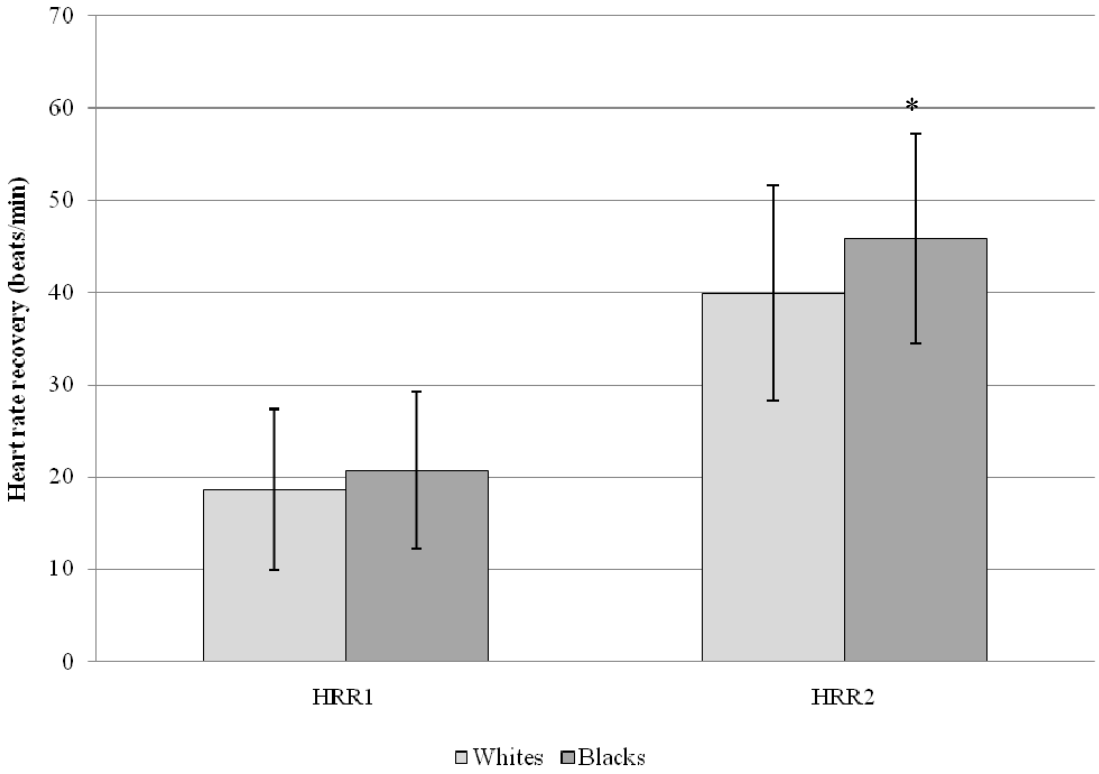

Figure 2 - Heart rate recovery values (HRR1 and HRR2) between white and black subjects.

HRR1 represents the heart rate recovery 1-minute post-exercise (i.e., difference between maximal heart rate and the heart rate at 1-minute post exercise). HRR2 represents the heart rate recovery 2-minutes post-exercise (i.e., difference between maximal heart rate and the heart rate at 2-minutes post exercise). *Blacks had significantly greater HRR2 compared to whites, $p < 0.05$.

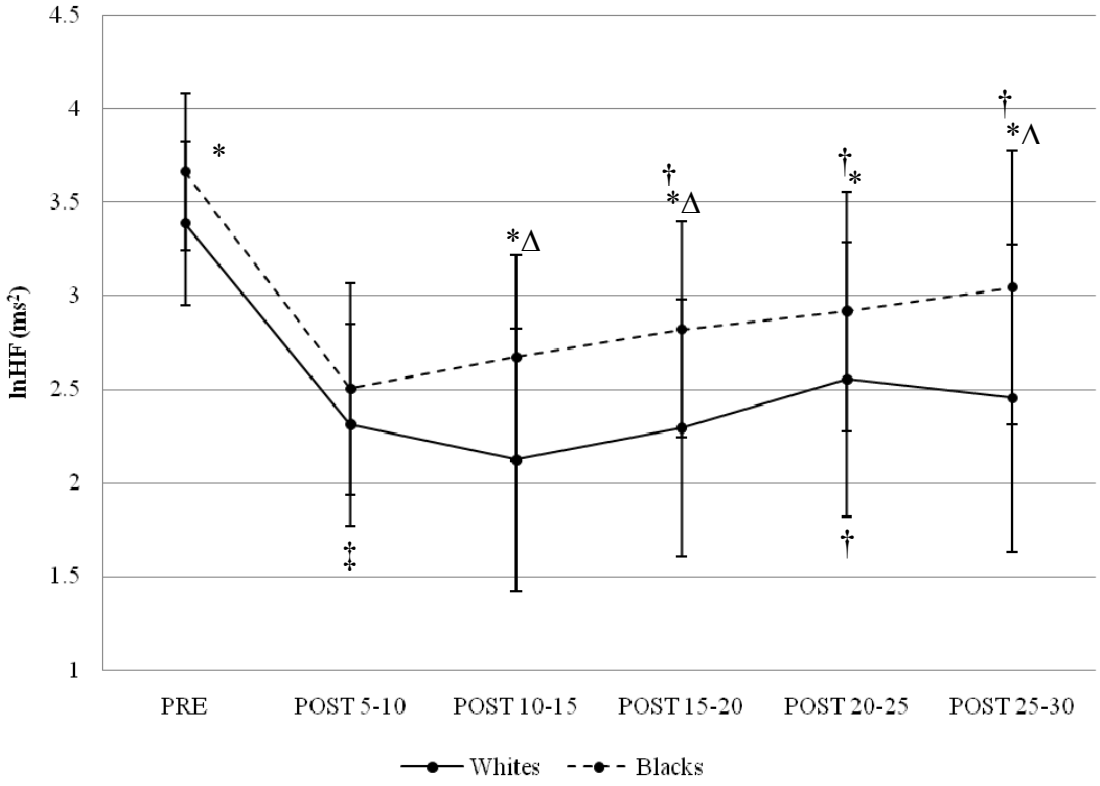

Figure 3 - PRE and POST lnHF values for white and black subjects.

* Blacks had higher lnHF values vs whites, $p < 0.05$. Δ Difference between blacks vs whites remains when controlling for PRE, $p < 0.05$. † significantly greater than POST 5-10, $p < 0.05$. ‡ significantly lower vs POST 5-10, $p < 0.05$

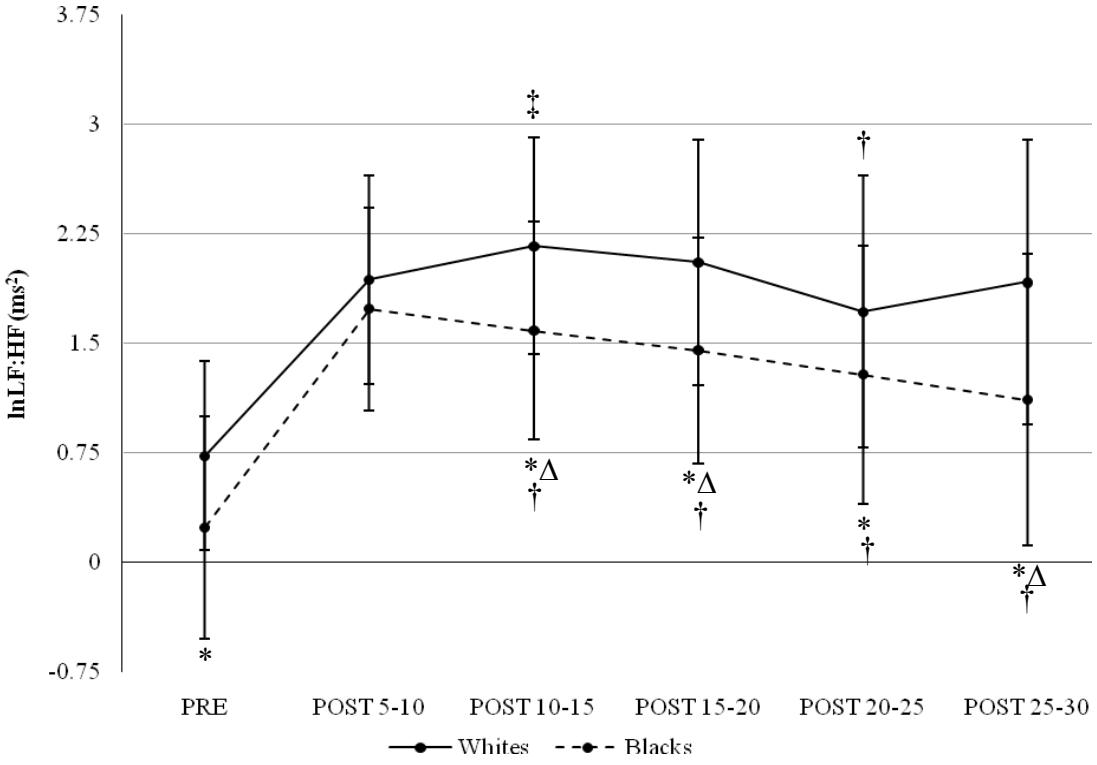

Figure 4 - PRE and POST lnLF:HF values for white and black subjects

* Blacks had significantly lower lnLF/HF ratio vs whites, $p < 0.05$. Δ Difference between blacks vs whites remains when controlling for PRE, $p < 0.05$. † significantly lower than POST 5-10, $p < 0.05$.
‡ lnLF/HF significantly greater than POST 5-10, $p < 0.05$

Table 1: Descriptive Statistics

	Whites (n = 30)	Blacks (n = 30)
Age (yrs)	22.00 ± 3.14	23.2 ± 3.23
Height (cm)	181.28 ± 6.74	182.39 ± 8.42
Weight (kg)*	80.35 ± 9.87	85.67 ± 11.02
BMI (kg/m^2)	24.46 ± 2.38	25.80 ± 3.41
WC (cm)	82.81 ± 11.79	82.22 ± 8.33
HC (cm)	97.64 ± 12.91	101.22 ± 7.77
WHR*	0.84 ± 0.04	0.81 ± 0.05
Sum of Skinfolds (mm)	75.42 ± 24.84	73.99 ± 35.19
Predicted Body Fat (%)	9.96 ± 3.79	9.74 ± 5.13
VO_{2max} (ml/kg/min)*	47.23 ± 6.99	42.03 ± 6.89
SBP (mmHg)	121.73 ± 10.35	123.30 ± 8.90
DBP (mmHg)*	71.60 ± 7.82	77.73 ± 8.06
MAP (mmHg)*	88.31 ± 7.24	92.90 ± 6.53
RHR (beats/min)	59.77 ± 7.22	60.70 ± 9.26

All values are reported as means ± standard deviations. BMI = body mass index, WC = waist circumference, HC = hip circumference, WHR = waist-to-hip ratio, SBP = systolic blood pressure, DBP = diastolic blood pressure, MAP = mean arterial pressure, RHR = resting heart rate, * $p < 0.05$.

Table 2. Resting HRV parameters between groups.

	Whites (n = 30)	Blacks (n = 30)
HFnu (ms^2)*	32.24 ± 12.40	42.20 ± 16.47
LF:HF (ms^2)*	2.60 ± 2.07	1.68 ± 1.37
lnHF (ms^2)*	3.39 ± 0.45	3.66 ± 0.43
lnLF:HF (ms^2)*	0.73 ± 0.66	0.24 ± 0.77

All values are reported as means ± standard deviations. HFnu = normalized high frequency power, LF:HF = low frequency to high frequency power ratio, lnHF = natural log transformed high frequency power, lnLF:HF = natural log transformed low frequency to high frequency ratio. The white subjects had lower HFnu and lnHF, and higher LF:HF and lnLF:HF compared to the black subjects (*$p < 0.05$).

CHAPTER 4

HEART RATE RECOVERY AFTER MAXIMAL EXERCISE: EFFECTS OF RACE, AEROBIC POWER, AND SELETED HEALTH-RELATED ANTHROPOMETRIC VARIABLES

Abstract

The purpose of this investigation was determine the effect of VO_{2max} on HRR, independent of race, body mass index (BMI), waist circumference (WC), and predicted body fat percentage (BF), in a cohort of white and black college-age men. Seventy-two college-aged men participated in this study and were divided into two groups derived by VO_{2max} values as follows: ModFit (n = 36) had a mean VO_{2max} of 39.99 ± 4.01 ml/kg/min; and HiFit (n = 36) had a mean VO_{2peak} of 52.66 ± 5.30 ml/kg/min. Each subject performed a maximal graded exercise test on a treadmill to determine VO_{2max}. When VO_{2max} was reached, a three minute cool-down period was performed with the subjects walking 2 mph at 1.5% grade. Heart rate (HR) was monitored with electrocardiography during the test and cool-down periods. HRR was determined from the difference between HR recorded at maximal exercise and the HR recorded at 1-minute (HRR1) and 2-minutes (HRR2) post-exercise. The results showed that there were no significant differences in HRR1 or HRR2 between the two groups. However, when controlling for the influence of race, the HiFit group had significantly greater HRR2 and lower HR2 compared to the ModFit group. Furthermore, a regression analysis showed

that race was a stronger contributor the variation in HRR2. These results suggest that aerobic fitness is related to the recovery in heart rate after exercise. However, race is a more powerful determinate of heart rate recovery after exercise.

Key Words: African American, Aerobic Power, Anthropometry, Parasympathetic

INTRODUCTION

The heart rate recovery (HRR) after exercise is highly dependent on the autonomic nervous system. Parasympathetic reactivation that occurs immediately after completing a bout of exercise is the primary mechanism responsible for the return of heart rate toward baseline (15). Reduced parasympathetic influence after exercise can lead to an abnormal HRR and is strongly linked to cardiovascular disease risk. For example, a blunted HRR after exercise is independently associated with increased coronary artery calcium (19), dysfunctional glucose control (24), and a higher prevalence of atherosclerosis (16). Moreover, HRR has been shown to be delayed in heart failure patients (2,32), in those with the metabolic syndrome (11), and in those who have diabetes mellitus (22). An abnormal HRR has been viewed as a valid predictor of morbidity and mortality in both clinical and non-clinical populations (7,8,18,29). Thus, HRR is becoming viewed as an emerging independent risk factor for cardiovascular disease.

A number of cross-sectional studies show that both younger and older endurance trained and physically active subjects display a more favorable cardiovascular autonomic profile and a faster HRR compared to non-athletic, sedentary, and clinical populations (4,6,9,10,11,12,13,15,20,23,26). This body of research suggests that a higher level of

fitness (i.e., VO_{2max}) may lead to greater HRR. However, in most studies, the relationship between cardiovascular autonomic influence and maximum oxygen uptake (i.e., VO_{2max}) has been investigated without regard for a possible confounding influence of race or health-related anthropometric variables, such as body mass index (BMI), waist circumference (WC), and body fat percentage. Both race and body size has been shown to exert an influence of cardiovascular autonomic modulation (6,14,28,30,31).

The purpose of this investigation was two-fold: 1) to determine the effect of VO_{2max} on HRR, independent of race, BMI, WC, and predicted body fat percentage (BF), in a cohort of white and black college-age men; and 2) to determine the degree of variation in HRR that could be explained with each of the following variables: race, VO_{2max}, BMI, WC, and BF.

METHODS

Participants

Seventy-two college-aged white (n = 41) and black (n = 31) men participated in this study. All data was collected in the Human Performance Laboratory at Auburn University Montgomery. This study was approved by the Institutional Review Board for Human Participants. All participants were non-smokers and had no history or clinical sign of cardiovascular or pulmonary diseases. The subjects gave informed consent in writing and completed health history forms to qualify them for the study. All participants were normotensive (i.e., blood pressure < 140/90 mmHg), not currently taking any blood pressure or anti-depressive medications. Subjects reported ethnicity/race as either Non-hispanic/White or Non-hispanic/Black over three generations.

Experimental Design

All data was collected for each subject on one visit to the lab during one of two 2-hour time slots: either between 7:00 and 9:00am, or between 9:00 and 11:00am, on any day of the week. The subjects were instructed to not consume alcoholic or caffeinated beverages 24 hours before the test, and to not eat at least 3 hours before the test. Upon entry into the lab, subjects were given verbal instruction to familiarize them of the testing procedures. After completing the necessary screening form and providing informed consent in writing, the following anthropometric variables were measured: height, weight, BMI, WC, and the skinfold thickenss of 7-sites were used to estimate body fat percentage. Once anthropometric variables were assessed, the subjects performed a graded maximal aerobic test on a treadmill. HRR was analyzed during 2-minutes of the cool-down period.

Anthropometric variables

Height was measured with a SECA stadiometer and rounded to the nearest 0.05 cm. Weight was measured with a TANITA (BWB-800A) digital scale and rounded to the nearest 0.05 kg. Body mass index (BMI) was calculated as weight in kg divided by height in meters squared (kg/m^2). Waist circumference was measured with a retractable tape measure and rounded to the nearest 0.05 cm. Body fat percentage was estimated via the 7-site skinfold method (1). Skinfolds were measured from the following seven sites on the right side of the body: a diagonal fold on the chest/pectoralis major half way between the nipple and the anterior axiallary line; a vertical fold on the triceps half way between the olecranon and acromion processes, with the arm held freely to this side of the body; a diagonal fold on the subscapularis 1 to 2 cm below the inferior angle of the

scapula; a vertical fold in the mid-axillary line at the level of the xiphoid process; a vertical fold on the abdomen 2 cm to the right of the umbilicus; a diagonal fold on the suprailliac crest in the anterior axillary line; and a vertical fold on the anterior mid-line of the thigh half way between the proximal border of the patella and the inguinal crease. Each skinfold measurement was measured in was rounded to the nearest 0.1 millimeter.

Maximal Graded Exercise Test

All subjects performed a symptom-limited graded exercise test on a Parker Treadmill (Parker Co., Opelika, AL) to assess maximal oxygen consumption (VO_{2max}). This protocol, i.e., Bruce Protocol, involved a series of 3-minute stages, with each stage progressed from the previous by increasing workload (speed and grade), until maximal oxygen consumption (VO_{2max}) was reached. Expired gases (oxygen and carbon dioxide) were collected continuously at the mouth with a pneumotach and analyzed with a metabolic analyzer (Applied Electrochemistry, AMETEK, Pittsburg, PA). All data was recorded on a personal computer every 30-seconds using Turbofit 5.06 software (VACUMED, Ventura, CA). Maximal oxygen uptake was reached if two of the following occur: a plateau in VO_2 with increasing work rate; respiratory exchange ratio (RER) $\geq$ 1.15; heart rate within 10 beats of age-predicted maximum (220 – age); or volitional fatigue. Heart rate was monitored during the test with a Polar electronic heart monitor (Polar Electro Oy, Kemple, Finland). A standard sphygmomanometer and stethoscope was used to assess blood pressure during the last 45 seconds of each stage. After a maximal value was obtained, the subject performed a 3-minute cool-down period with the speed at 2.5 mph and a 1.5% grade.

Heart rate recovery

Heart rate was monitored continuously during the recovery period. To examine HRR, the heart rate that corresponded to VO_{2max} (i.e., maximal heart rate [MHR]) and the heart rate at 1-min (HR1) and 2-min (HR2) post exercise (i.e., during the cool-down period) were recorded. The difference between HR1 and MHR was defined as HRR1. The difference between HR2 and MHR was defined as HRR2.

Statistical Analysis

All statistical analysis was completed using SPSS version 16.0. The participants were divided into two groups either below or above the mean for VO_{2max} of the entire sample. Significant differences for descriptive variables between the two groups were assessed by one-way analysis of variance (ANOVA) and are displayed in Table 1. Univarate analysis of variance (ANOVA) procedures were utilized to examine group differences in MHR, HR1, HR2, HRR1 and HRR2. Follow-up analysis covariance (ANCOVA) procedures were utilized on each variable to determine a group effect independent of race, BMI, WC, and predicted body fat % (BF). An impact of VO_{2max} for MHR, HR1, HR2, HRR1, and HRR2 was also determined separately for the white group and black group of men. Similar for the entire sample, each race was divided into two groups either below or above the mean for VO_{2max} within each race.

Last, a backwards linear regression procedure was then performed to determine the extent of variation in MHR, HR1, HR2, HRR1, and HRR2 that could be explained by race, VO_{2max} (as a continuous variable), BMI, WC, and BF. Statistical significance for all tests was set at $p < .05$.

RESULTS

All subjects that agreed to participate completed the testing procedures. The mean VO_{2max} for the entire sample was 46.32 ml/kg/min. Therefore, the subjects were divided into two groups derived by VO_{2max} values as follows: group 1 (ModFit, n = 36) had a mean VO_{2max} of 39.99 ± 4.01 ml/kg/min; and Group 2 (HiFit, n = 36) had a mean VO_{2max} of 52.66 ± 5.30 ml/kg/min. The ModFit group had significantly higher weight, BMI, WC, BF, and RHR compared to the HiFit group (Table 1). Group means in MHR, HR1, HR2, HRR1, and HRR2 are displayed in Table 2. There were no significant differences revealed between the two groups in any of the post-exercise heart rate parameters. Theses findings were unchanged after controlling for the independent influences of BMI, WC, or BF. However, after controlling for the influence of race with an ANCOVA procedure, a significant difference in two of the post-exercise heart rate variables were revealed between the two groups. After controlling for race, the HiFit group had a lower HR2 ($p < .05$) and a higher HRR2 ($p < .05$) compared to the ModFit group.

For each race separately, the mean VO_{2max} was 49.04 ± 7.90 ml/kg/min for whites and 42.73 ± 6.40 ml/kg/min for blacks, which was signficantly different ($p < .05$). Therefore, the whites were divided into two groups as follows: ModFitW (n = 20) had a mean VO_{2max} of 42.54 ± 4.23 ml/kg/min; and HiFitW (n = 21) had a mean VO_{2max} of 55.23 ± 5.06 ml/kg/min. The blacks were divided into the following two groups: ModFitB (n = 14) had a mean VO_{2max} of 37.15 ± 3.19 ml/kg/min; and HiFitB (n = 17) had a mean VO_{2max} of 47.33 ± 4.32 ml/kg/min. There were significant differences revealed for HR2 (156.05 ± 12.28 beats/min for ModFit vs 144.10 ± 11.10 beats/min for

HiFit, $p < .05$) and HRR2 (34.25 ± 9.17 beats/min for ModFit vs 43.67 /- 8.66 beats/min for HiFit, $p < 0.05$) between ModFitW and HiFitW (Figure 1). There were also significant differences revealed for MHR (191.71 ± 7.39 beats/min for ModFit vs 184.88 ± 8.61 beats/min for ModFitB, $p < .05$), HR1 (172.57 ± 9.57 beats/min for ModFitB vs 162.88 ± 11.53 beats/min, $p < .05$), HR2 (149.21 ± 11.69 vs 136.65 ± 12.75 beats/min, $p < .05$), and HRR2 (42.50 ± 7.85 vs 48.11 ± 9.38 beats/min, $p < .05$) between ModFitB and HiFitB (Figure 2).

Univariate ANOVAs were also utilized to compare the difference in MHR, HR1, HR2, HRR1, and HRR2 between the white and black men. There were no significant differences in MHR, HR1, or HRR1. The whites had significantly higher HR2 (149.93 ± 13.03 beats/min for whites and 138.35 ± 23.01 beats/min for blacks, $p < .05$) and lower HRR2 (39.07 ± 45.86 beats/min for whites and 45.86 ± 9.05 beats/min for blacks, $p < .05$) compared to the black subjects.

The results of the regression procedures are shown in Table 3 for HR2 and Table 4 for HRR2. Race and VO_{2max} were the only significant contributors to the regression model for HR2. Together, the two variables explained 16% of the variation in HR2 ($p < 0.05$). Race, and VO_{2max} were also the only significant contributors to the regression model for HRR2. Together, the two variables explained 20% of the variation in HRR2 ($p < .05$). Race was the strongest contributor to both regression models. Last, none of the variables were associated with MHR, HR1, or HRR1.

DISCUSSION

The increase in cardiac output (i.e., heart rate and stroke volume) with exercise is due to a decrease in parasympathetic nervous input followed by an increase in sympathetic nervous activity. The immediate drop in heart rate following exercise is primarily due to a quick return of parasympathetic influence, with a decrease in sympathetic activity occurring in the later stages of recovery. Thus, examining the initial recovery in heart rate after maximal exercise (i.e., HRR) has grown in popularity as a non-invasive marker of cardiovascular parasympathetic influence (7,8,15,18).

There were three main findings of this investigation. First, there were no differences between the ModFit and HiFit groups in the post-exercise HRR variables when race was not accounted for. However, after controlling for race, the HiFit group had a better HRR profile (i.e., lower HR2 and faster HRR2) compared to the ModFit group. Therefore, the entire sample was divided by race; comparing high fit whites (HiFitW) to moderate fit whites (ModFitW), and high fit blacks (HiFitB) to moderate fit blacks (ModFitB). When comparing the intra-racial groups, the higher fit subjects had a significantly lower HR2 and higher HRR2 ($p < .05$). In addition, the HiFitB had lower MHR and HR1 compared to the ModFitB ($p < .05$).

The second major finding was that race had the greatest influence on HRR compared to VO_{2max}, BMI, WC, and predicted body fat percentage. Backward linear regression procedures were utilized to examine the association of race, VO_{2max}, and the selected anthropometric measures (BMI, WC, and BF) on MHR, HR1, HR2, HRR1, and HRR2. None of the variables were associated with MHR, HR1, or HRR1. Race and VO_{2max} were significantly associated with HR2 ($R = .400$, $R^2 = .160$, $p < .05$)and HRR2

($R = 0.449$, $R^2 = 0.201$, $p < .05$). Furthermore, according to both models, race held the highest association to both HR2 and HRR2.

The third major finding was that the black men had a significantly better heart rate recovery profile compared to white men (i.e., lower HR2 and higher HRR2, $p < .05$). Thus, the results of this study suggest that when comparing white versus black college-age men, race holds a stronger relationship to the recovery in heart rate after maximal exercise compared to VO_{2max} and selected anthropometric variables.

It has been known for sometime that the recovery of heart rate after exercise is highly dependent on individual aerobic fitness. However, the association between aerobic fitness and HRR has not fully been clarified. Some studies suggest that HRR is faster in those individuals who are fitter (9,21,27) while others suggest no association between individual fitness and HRR. Darr et al. (9) found that younger and older trained subjects had a larger reduction in heart rate after a maximal exercise bout on a cycle ergometer compared to their untrained counterparts. Short & Sedlock (27) found that HRR was faster in trained vesus untrained subjects after an acute bout of cycle exercise at an absolute workrate of 1.5 L/min oxygen uptake (i.e., VO_2). In contrast, a more recent investigation (3) found no relationship between VO_{2max} and HRR. The racial characteristic of these studies participants were not accounted for. Furthermore, Carnethon et al. (6) examined the association of HRR and physical activity and aerobic fitness in a large sample (n = 3,446) of black and white men and women. The results of the study revealed two important findings relative to the current study. First, a weak correlation (r = .15) was found between 2-minute HRR and aerobic fitness, i.e., time on treadmill during a symptom-limited Balke treadmill protocol. Second, in agreement with

the current study, the black men were shown to have the fastest HRR 2-minutes after maximal exercise (6). However, it was not stated if the black and white subjects had differing levels of aerobic fitness. In the current investigation, race was more strongly associated to the variation in HRR2 compared to VO_{2max} and any of the health-related anthropometric variables. Moreover, the black subjects had faster HRR2 compared to the whites, despite having lower VO_{2max} values.

The reason blacks had a lower HR2 and HRR2 after exercise compared to the white subjects cannot be fully explained. It is possible that blacks have a higher cardiovascular parasympathetic influence compared to whites. While this is the one of the only studies that has accounted for an influence of race on autonomic influence after exercise, there are several studies that have examined the difference in autonomic influence at rest between white and black subjects (14,28,30,31). By analyzing the variation in heart rate (i.e., heart rate variability [HRV]), most studies suggest that young black men have a more favorable sympathetic-parasympathetic balance at rest compared to whites (14,28,30,31). However, more research is needed to determine if there is a relationship between autonomic control of heart rate at rest and post-exercise. Two studies have found no relationship to resting HRV and post-exercise HRR (5,17).

There are a number of limitations with the present investigation that concern the sample chosen. This study was limited to only black and white college-aged men. Differences across other races, sex, and with aging were not studied. Also, overall physical activity has been shown to exert a positive influence on autonomic influence of the heart (6,25). A difference in daily physical activity between the subjects was not assessed in this study.

This study adds to the current body of research on the association of heart rate recovery and race, aerobic fitness, and health-related anthropometrics. The findings of this investigation suggest that race significantly alters the influence of VO_{2max} on HRR. If race was not controlled for, the results would have been skewed, i.e., an effect of VO_{2max} on HRR would not have been revealed. After controlling for race, the higher fit subjects had a significantly faster drop in heart rate 2-minutes post-exercise. With respect to all of the variables, race yielded the greatest association to the heart rate recovery parameters analyzed in this study. Most available research examining heart rate recovery fails to mention the racial characteristics of the participants. The current study shows the importance of the confounding effect of race when studying the heart rate recovery pattern post-maximal exercise.

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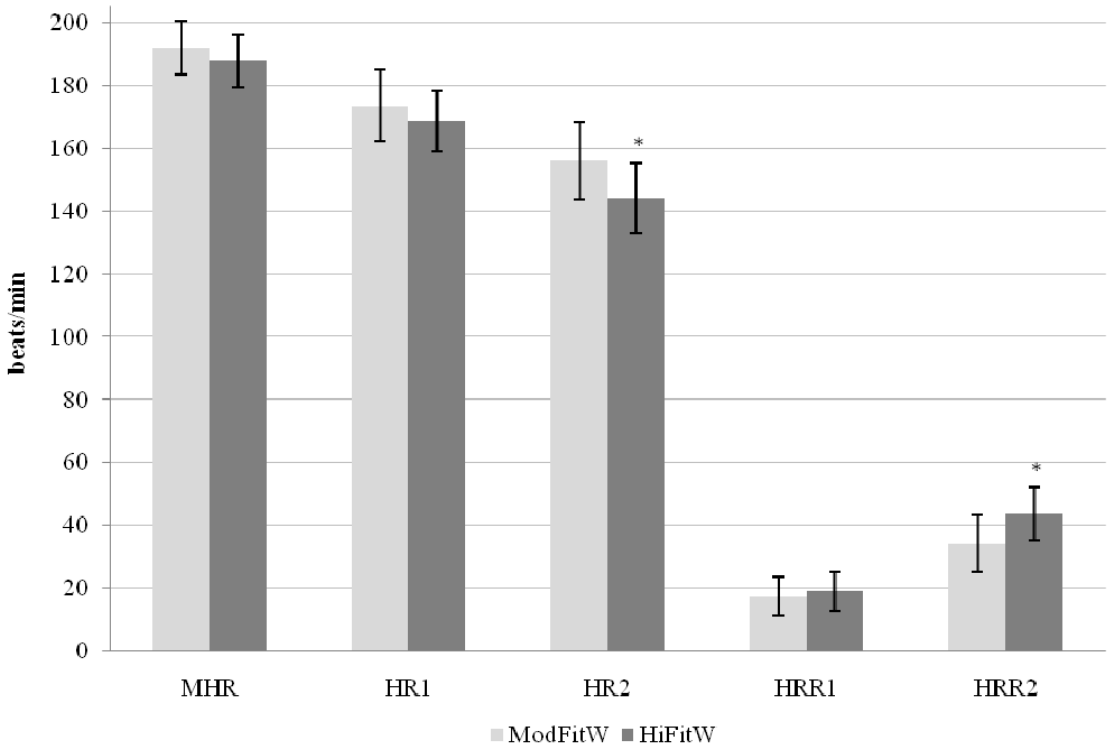

Figure 1. Differences in the post-exercise heart rate recovery variables between the moderate fit white group (ModFitW) and the high fit white group (HiFitW).
MHR represents the heart rate recorded at VO_{2max}. HR1 represents the heart rate recorded at 1-minute post-exercise. HR2 represents the heart rate recorded at 2-minutes post-exercise. HRR1 represents the 1-minute heart rate recovery (i.e., the difference between MHR and HR1). HRR2 represents the 2-minute heart rate recovery (i.e., the difference between MHR and HR2). *The HiFitW had lower HR2 and higher HRR2 compared to the ModFitW ($p < 0.05$).

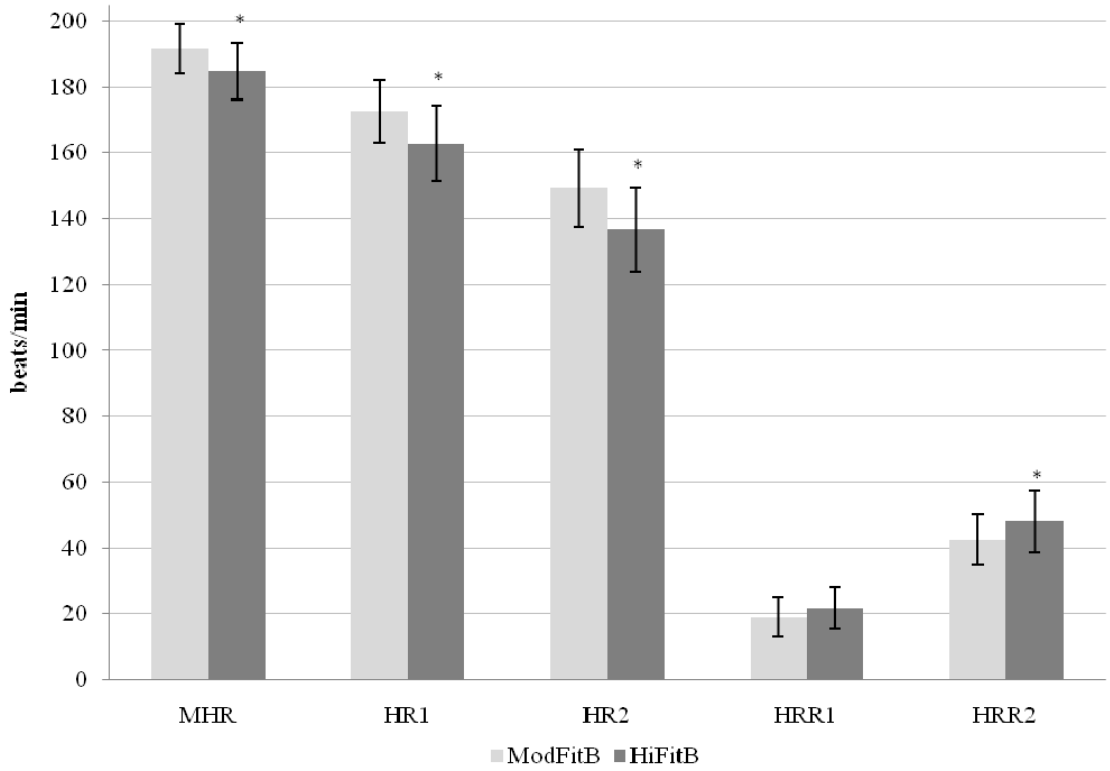

Figure 2. Differences in the post-exercise heart rate recovery variables between the moderate fit black group (ModFitB) and the high fit black group (HiFitB).
MHR represents the heart rate recorded at VO_{2max}. HR1 represents the heart rate recorded at 1-minute post-exercise. HR2 represents the heart rate recorded at 2-minutes post-exercise. HRR1 represents the 1-minute heart rate recovery (i.e., the difference between MHR and HR1). HRR2 represents the 2-minute heart rate recovery (i.e., the difference between MHR and HR2). *The HiFitB had lower MHR, HR1, HR2 and higher HRR2 compared to the ModFitB ($p < 0.05$).

Table 1. Descriptive Statistics of ModFit and HiFit subjects

	ModFit (n = 36)	HiFit (n = 36)
Age (yrs)	22.86 ± 3.32	22.61 ± 3.93
Height (cm)	183.02 ± 6.54	179.84 ± 7.61
Weight (kg)*	87.09 ± 10.69	76.21 ± 9.08
BMI (kg/m^2)*	26.10 ± 3.54	23.53 ± 1.97
WC (cm)*	86.04 ± 9.46	78.32 ± 9.74
Sum of Skinfolds (mm)*	87.11 ± 34.02	57.93 ± 18.58
Predicted Body Fat (%)*	11.66 ± 4.99	7.39 ± 2.89
RHR (beats/min)*	62.33 ± 8.27	58.15 ± 8.56
VO_{2max} (ml/kg/min)*	39.99 ± 4.01	52.66 ± 5.29

All values are reported as means ± standard deviations. BMI = body mass index, WC = waist circumference, RHR = resting heart rate. The HiFit subjects had significantly lower weight, BMI, WC, sum of skinfolds and predicted body fat precentage and higher VO_{2max} values compared to the ModFit subjects (*$p < 0.05$).

Table 2. Mean heart rate recovery (beats/min) varaibles at- and post- maximal exercise and before controlling for the influence of race

	ModFit (n = 36)	HiFit (n = 36)
MHR	190.14 ± 9.12	187.89 ± 7.98
HR1	170.14 ± 13.15	168.69 ± 8.74
HR2*	148.92 ± 15.98	144.39 ± 10.83
HRR1	19.58 ± 6.66	19.14 ± 5.76
HRR2*	40.39 ± 10.87	43.39 ± 9.12

All values are reported as means ± standard deviations. MHR = maximal heart rate, HR1 = heart rate at 1-minute post exercise, HR2 = heart rate at 2-minutes post exercise, HRR1 = 1-minute heart rate recovery (i.e., difference between MHR and HR1), HRR2 = 2-minutes heart rate recovery (i.e., difference between MHR and HR2). There was no difference between the groups in any of the post-exercise heart rate recovery variables before controlling for the influence of race. *However, after controlling for the influence of race with the ANCOVA procedure, a significant difference was revealed between HR2 and HRR2 ($p < 0.05$).

Table 3. Regression coefficients for HR2 as the dependent variable.

	Full Model			Restricted Model		
Variable	R^2	Beta	Sig.	R^2	Beta	Sig
	.180*			.160*		
Race		-.366	.009		-.402	.001
VO_{2max}		-.279	.068		-.316	.011
BMI		-.126	.531			
WC		-.125	.423			
BF		.232	.279			

BMI = Body Mass Index, WC = waist circumference, BF = Predicted Body Fat Percentage, *p < .05.

Table 4. Regression coefficients for HRR2 as the dependent variable.

	Full Model			Restricted Model		
Variable	R^2	Beta	Sig.	R^2	Beta	Sig
	.229*			.201*		
Race		.422	.002		.458	.000
VO_{2max}		.401	.008		.341	.005
BMI		.241	.220			
BF		.071	.733			
WC		.011	.943			

BMI = Body Mass Index, WC = waist circumference, BF = Predicted Body Fat Percentage, *p < .05.

CHAPTER 5

HEART RATE RECOVERY: IT'S RELATIONSHIP TO RESTING AND POST-EXERCISE HEART RATE VARIABILITY

Abstract

There is limited research available regarding a possible relationship between resting HRV and post-exercise HRR. The aim of this study was to examine the relationship between resting HRV and HRR after maximal exercise. Sixty-six college-age men participated in this study. HRV was assessed in a supine position before and for 30-minutes after a maximal exercise test on a treadmill. HRV was assessed in the frequency (i.e., log transformed normalized HF power [lnHF] and log transformed normalized LF:HF ratio [lnLF:HF]) domains. Heart rate was recorded at maximal exercise (MHR), and at 1- (HR1) and 2- (HR2) minutes of the cool-down recovery period. HRR was determined from the difference between MHR and HR1 (HRR1) and the difference between MHR and HR2 (HRR2). No significant relationship was found between the frequency domain parameters of resting HRV and HRR1 or HRR2. Therefore, resting HRV may not be related to the recovery of HR expressed as a slope (i.e., HRR) within 2-minutes following a maximal exercise test. This is possibly due to a higher HRV at rest being associated with lower MHR and lower HR1 and HR2 during recovery.

Key Words: Cardiovascular, Autonomic Modulation, Maximal Exercise

INTRODUCTION

During exercise, adjustments must be made within the cardiovascular system to meet the metabolic demands of active skeletal muscle. The autonomic nervous system plays a critical role in making these adjustments. For instance, heart rate, stroke volume, and myocardial contractility all increase with exercise due to a withdrawal of parasympathetic activity and an increase in sympathetic activity (4,7,9,16). During recovery from exercise, the initial return of heart rate towards baseline is primarily due to parasympathetic reactivation (10). Assessing heart rate recovery (HRR) after exercise has become a valuable, non-invasive procedure to assess cardiovacular-parasympathetic influence (5,10). Blunted HRR has valuable clinical applications. For instance, the results of a number of large studies suggest that a delayed HRR is an independent predictor of mortality (5,12,14,19,20).

Another non-invasive approach for examining cardiovascular autonomic control is the assessment of heart rate variability (HRV). This procedure involves examining the oscillations that occur in adjacent QRS complexes (specifically, R wave to R wave) on an electrocardiogram (ECG) recording. Depressed HRV is purported to be due to a dysfunctional autonomic nervous system and also has been viewed as an important predictor of premature and sudden mortality, especially in clinical populations (2,6,8,13). Increases in HRV are thought to be cardioprotective (2).

HRV can be measured in both time and frequency domains. For time domain analysis, an ECG recording is plotted as a tachogram, which plots the distance of each consecutive R-R interval (in milliseconds) against the number of total beats. The time domain method involves manual calculations of the R-R intervals, such as the standard

deviation of the R-R intervals (SDNN). The frequency domain method, which is more powerful at capturing short-term HRV (17), involves the transformation of an ECG signal into a power spectrum. The different frequency ranges from the power spectrum that are typically used to represent HRV are high frequency (HF) power (0.015 Hz to 0.40 Hz), and low frequency (LF) power (0.004 to 0.015 Hz). HF power is said to represent parasympathetic influence and LF power is purported to represent both parasympathetic and sympathetic activities. LF-to-HF ratio (i.e., LF:HF) represents sympathetic-to-parasympathetic balance (17).

Because of the parasympathetic influence of both HRV and HRR, it has been hypothesized that those with higher HRV at rest would also have greater HRR (11). However, the limited data available suggests no association between resting HRV and HRR. Javorka et. al. (11) found no relationship between supine resting HRV and the percent decrease in heart rate (i.e., HRR) from the end of an 8-minute stepping exercise to the first minute of recovery. Bosquet et al. (3) also found no association between resting HRV and 1-minute HRR after maximal exercise in a group of trained distance runners.

HRV is most accurately assessed during steady state conditions, such as during supine rest (17). HRR, on the other hand, is a value that is derived from the difference between exercise heart rate (e.g., maximal heart rate) and the heart rate (HR) of a selected time point recorded during recovery from exercise, typically either one or two minutes post-exercise. Therefore, HRR represents a non-steady state negative slope between two points. Because only a few studies have examined the association between resting HRV and HRR, the aim of this investigation was to further explore the relationship between

HRV and the recovery of heart rate after maximal exercise.

METHODS

Participants

Sixty-six apparently healthy college-aged men were recruited to participate in this study. All data was collected in the Human Performance Laboratory at Auburn University Montgomery (AUM). This study was approved by the Institutional Review Board (IRB) for Human Participants. All participants had no history or clinical sign of cardiovascular or pulmonary diseases and were non-smokers. The subjects gave informed consent in writing and completed health history questionnaires to qualify them for the study. All participants were normotensive (i.e., blood pressure < 140/90 mmHg), not currently taking any prescribed blood pressure or anti-depressive medications, and displayed normal electrocardiogram (ECG) patterns. Subjects reported ethnicity/race as either Non-hispanic/White or Non-hispanic/Black over three generations.

Experimental Design

All data was collected for each subject on one visit to the lab during one of two 2-hour time slots: either between 7:00 and 9:00am, or between 9:00 and 11:00am, on any day of the week. The subjects were instructed to not consume alcoholic or caffeinated beverages 24 hours before the test, and to not eat at least 3 hours before the test. Upon entry into the lab, subjects were given verbal instruction to familiarize them of the testing procedures. After completing the necessary screening form and providing informed consent in writing, weight, height, and body mass index (BMI) were determined and body fat percentage was estimated for descriptive purposes. Height was assessed with

the use of a wall mounted stadiometer (SECA) and rounded to the nearest 0.1 cm and weight was measured with a digital scale (TANITA BWB-800A) and rounded to the nearest 0.1 kg. Body mass index (BMI) was calculated as weight in kg divided by height in meters squared (kg/m^2). Body fat percentage was estimated via 7-site skinfold technique (1). Resting HRV was examined during a 5-minute period while each subject assumed a supine position. After the 5-minute period, resting blood pressure was assessed with the use of a sphygmomanometer and stethoscope while the subjects remained in the supine position. After completing these preliminary assessments, each subject performed a maximal graded exercise test on a treadmill. HRR was analyzed during 2-minutes of a cool-down period. After the subjects completed the cool-down period, they once again assumed a supine position for 30-minutes. During this time, post-exercise HRV was analyzed.

Heart Rate Variability

In order to analyze HRV, electrocardiographic (ECG) recordings were examined before and after exercise. A modified lead II configuration using three Ag/AgCl electrodes (BIOPAC ES509) was used for the ECG recordings. The electrodes were interfaced with a Biopac MP100 data acquisition system (Goletta, CA). All data was stored in a designated PC for analysis. Before exercise, each of the subjects assumed a supine position. During this time, subjects maintained their normal breathing patterns. The subjects remained in this position for 10 minutes prior to the exercise test. Following exercise, subjects once again assumed a supine position for 30 minutes.

The ECG recordings were divided into six 5-min segments as follows: One PRE

epoch, which was the last 5 minutes of the 10 minute baseline recording; and 5 POST epochs at 5 – 10, 10 – 15, 15 – 20, 20 – 25, and 25 – 30 minutes. All 5-minute ECG recordings were visually inspected. Any ectopic/non-sinus beats were removed and replaced by the adjacent normal R-R interval. If three or more ectopic beats were found within any ECG segment, the reading was excluded from analysis.

The frequency domain method was used to assess HRV. This method involved a a power spectral analysis on the ECG by applying a Hanning window and a fast Fourier transformation to the R-R intervals. In the frequency domain, HRV was separated into high frequency (HF) power (0.15-0.40 Hz) and low frequency (LF) power (0.04 – 0.15 Hz). Both of these values were normalized (HFnu, LFnu) to account for the influence of total power of the entire wave and the very low frequency (VLF) band (0.0033 – 0.04 Hz) as follows: HFnu = HF/(Total power of the entire wave – VLF) x 100; LFnu = LF/(Total power of the entire wave – VLF) x 100. HFnu was recorded and analyzed during the resting and post-exercise time intervals and utilized as a marker of parasympathetic modulation. The LF:HF ratio was also recorded during the selected time intervals and used as an index of sympatho-parasympathetic balance. For the purpose of this study, LFnu was not recorded for analysis.

Maximal Graded Exercise Test

Each subject performed a maximal graded exercise test, i.e., Bruce Treadmill Protocol, on a Parker Treadmill (Parker Co., Opelika, AL). Each stage was progressed from the previous stage, every 3-minutes, by increasing work rate (speed and grade), until maximal oxygen consumption (VO_{2max}) was reached. Expired gas fractions (oxygen and carbon dioxide) were collected at the mouth in a continuous manner, utilizing a mixing

chamber and gas analyzers from Applied Electrochemistry (AMETEK, Pittsburg, PA). All data was recorded on a personal computer every 30-seconds using Turbofit 5.06 software (VACUMED, Ventura, CA). Maximal oxygen uptake was reached if two of the following occured: a plateau in oxygen consumption despite on increased work rate; respiratory exchange ratio (RER) $\geq$ 1.10; heart rate within 10 beats of age-predicted maximum (220 – age); or volitional fatigue. Heart rate was monitored continuously during the exercise test using a Polar Heart Rate Monitor (Polar Electro Oy, Kemple, Finland). Blood pressure was also measured during the last 45 seconds of each stage. After the termination of the exercise test, a 3-minute period was utilized as a cool-down, with the treadmill workload decreased to 2.5 mph and 1.5% grade.

Heart rate recovery

Heart rate was recorded at three time points as follows: at maximal exercise (MHR), and at 1- (HR1) and 2-minutes (HR2) of the cool-down period. HRR was derived as the difference between MHR and the HR1 (HRR1) and HR2 (HRR2). HRR1, HRR2, MHR, HR1 and HR2 were recorded and analyzed.

Statistical Analysis

Subject data was entered into SPSS 16.0 for statistical analysis. Means and standard deviations were determined for the following descriptive statistics: age (yrs), height (cm), weight (kg), BMI (kg/m^2), predicted body fat (%), VO_{2max} (ml/kg/min). Normal distribution of the HRV data was verified by the Shapiro-Wilk test. A natural logrithmic transformation was performed on the HRV variables before the analysis when the data were skewed. Changes in the HRV variables from rest to post-exercise and the change in post-exercise heart rate (i.e., MHR to HR1 to HR2) were assessed with

repeated measures analysis of variance (ANOVA). Pearson product correlations were used to examine the relationship between the heart rate recovery parameters (i.e., MHR, HR1, HR2, HRR1, and HRR2), and HRV at rest (i.e., the PRE values of lnHF and lnLF:HF). Statistical significance for all tests was set at $p < .05$.

RESULTS

All sixty-six subjects completed resting/pre-exercise supine HRV analysis, the maximal exercise protocol, and the 30-minute post-exercise supine HRV analysis. The descriptive statistics (means ± standard deviation) for the all of the subjects were as follows: age = 22.74 ± 3.64 years; height = 181.36 ± 7.51 cm; weight = 82.11 ± 11.62 kg; BMI = 24.97 ± 3.20 kg/m^2; predicted body fat = 9.64 ± 4.73 %; and VO_{2max} = 46.39 ± 8.23 ml/kg/min.

Changes in HRV from PRE to POST

The natural log transformation of each HRV parameter was analyzed (i.e., lnHF, lnLF:HF). According to the repeated measures ANOVA procedures, each HRV parameter (lnHF, and lnLF:HF) changed during the experiment ($p < .05$). For instance, lnHF significantly dropped from PRE values and gradually increased during the 30-minute recovery period, but did not return to PRE (i.e., supine rest) values (Figure 1 for lnHF). In addition, lnLF:HF significantly increased from PRE values and gradually decreased during the 30-minute recovery period, but like the other HRV parameters, did not return to PRE values (Figure 2).

Heart rate recovery

Heart rate significantly dropped during the cool-down period. The heart rate recorded at maximal effort (i.e., MHR) was 188.91 ± 8.83 beats/min. The heart rate at 1-min recovery (i.e., HR1) was 169.03 ± 11.40 beats/min. The heart rate at 2-min recovery (i.e., HR2) was 146.08 ± 13.89 beats/min. Both HR1 and HR2 were significantly lower that MHR ($p < .05$) and HR2 was significantly lower than HR1 ($p < .05$). The mean HRR1 and HRR2 was 19.62 ± 6.20 beats/min and 42.33 ± 10.03 beats/min, respectively, which was significantly different ($p < .05$).

The relationship of HRR and resting HRV

No significant relationship was found between any resting HRV parameter and HRR1 or HRR2 (Table 1). However, there were significant associations between PRE lnHF and PRE lnLF:lnHF and the two following variables: MHR ($r = -.331$, $p < .01$ and $r = .369$, $p < .01$, respectively), and HR1 ($r = -.322$, $p < .01$ and $r = .360$, $p < .01$, respectively). In addition, PRE lnLF:HF significantly correlated to HR2 ($r = .264$, $p < .05$).

DISCUSSION

The major findings of the study were: 1) lnHF were drastically reduced and lnLF:HF was significantly increased after exercise and did not return to resting values 30-minutes after exercise; 2) Resting HRV was not associated with the recovery of HR expressed as a slope (i.e., HRR1 and HRR2); 3) Resting HRV was significantly associated with the heart rate values at selected time points of recovery (i.e., MHR, HR1 and HR2).

It has been shown that HRV is significantly reduced following exercise (11,15,18). Javorka et al. (11) revealed a reduction in HRV 30-minutes after an 8-min stepping exercise protocol at 70% of maximal power output. Similar to these findings, HRV did not return to resting values 30-minutes after exercise (11). The post-exercise decline in HRV is greater with higher intensity exercise (15,18). Terziotti et al. (15), found that HRV returned to baseline 1-hour after and 3-hours after 20-minutes of light (50% anaerobic threshold) and moderate (80% anaerobic threshold) intense steady state exercise, respectively. The results of the current investigation also showed a considerable reduction in parasympathetic influence (i.e., lnHF), and an increase in sympatho-vagal balance (i.e., lnLF:HF) 30-minutes after maximal graded exercise compared to resting values. Although there was a significant increase in HRV from the beginning to the end of the 30-minute recovery period, HRV after exercise did not reach resting (i.e., PRE) values.

Since both HRV at rest and HRR after exercise are tools used to examine autonomic influence, a few studies have examined the relationship between the two variables (3,11). Javorka et al. (11) examined the association of selected HRV parameters (at rest and after exercise) and the recovery in heart rate immediately after exercise. In their study, heart rate recovery was expressed as a percent decrease in the heart rate from cessesation of an 8-min stepping exercise at 70% maximal power output to the heart rate at 1-minute post-exercise. They found no significant correlation between resting HRV and post-exercise heart rate recovery (11). Bosquet et al. (3) also showed no correlation between resting HRV and the recovery of heart rate 1-min after exercise. The results of the current investigation concur with these two studies. There was no

significant relationship between resting HRV and either HRR1 or HRR2.

HRR is indicative of a non-steady state slope of heart rate at cessation of exercise to a selected time-point during recovery, usually either 1 or 2-minutes post exercise. HRV parameters, on the other hand, are values that represent the variation in heart rate and are most accurately assessed during resting conditions. In the present study, no relationship was found between resting HRV and the recovery of heart rate expressed as a slope (i.e., HRR1 or HRR2). However, significant correlations were noted between the two resting frequency domain HRV parameters (i.e., lnHF and lnLF:HF) and the heart rate at maximal exhaustion (MHR) and the heart rate at 1-minute post exercise (HR1). These findings suggest that those with greater HRV at rest display lower heart rates recorded at maximal exercise and at 1- and 2-minutes post exercise. It seems that the change in heart rate immediately after exercise is irrelevant in terms of a relationship to resting cardiovascular autonomic modulation, as assessed by resting HRV.

The results of this study suggest that there is no association of resting HRV and HRR. This is perhaps due to those with higher HRV at rest having lower heart rates at maximal exercise and at 1- and 2-minutes post exercise. Further research is warranted to further explore the relationship of autonomic tone at resting and autonomic influence following exercise.

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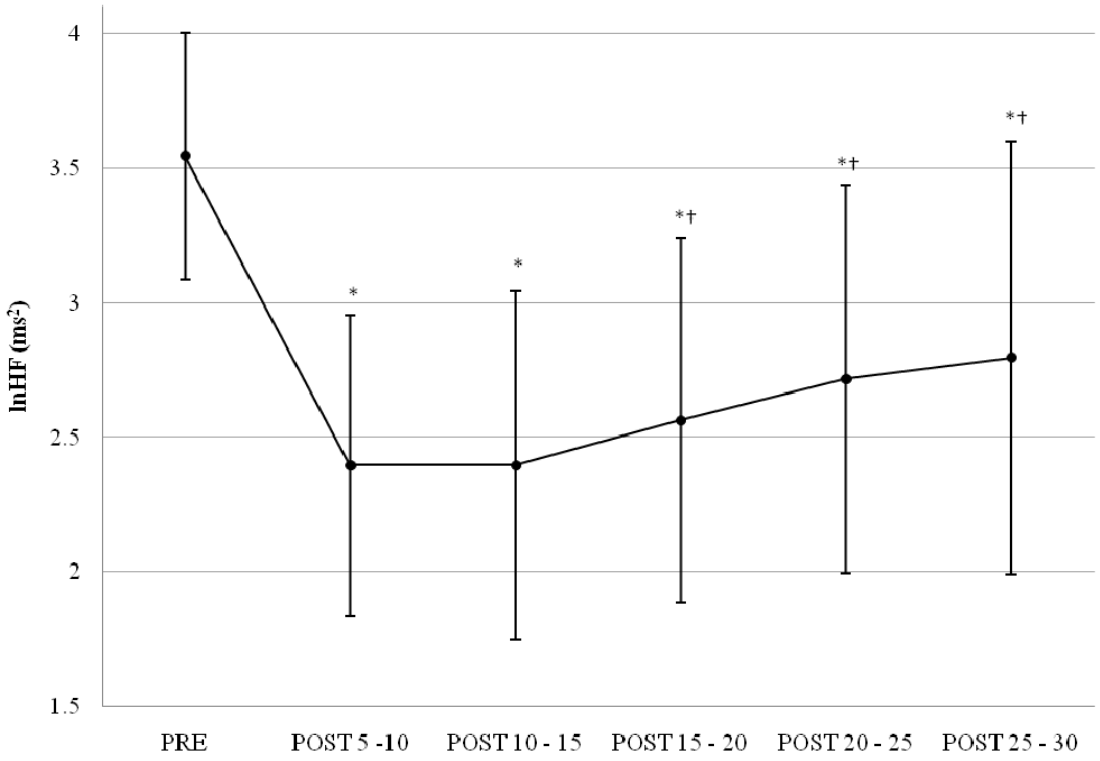

Figure 1. lnHF before (PRE) and after (POST) maximal exercise
lnHF = natural log tranformation of the high frequency power of heart rate variability. * Compared to PRE values, lnHF was significantly reduced at each of the POST 5-minute epoch ($p < 0.05$). † lnHF was significantly higher compared to the previous epoch ($p < 0.05$), i.e., lnHF gradually increased after POST 10 – 15 until POST 25 - 30, but did not reach resting values.

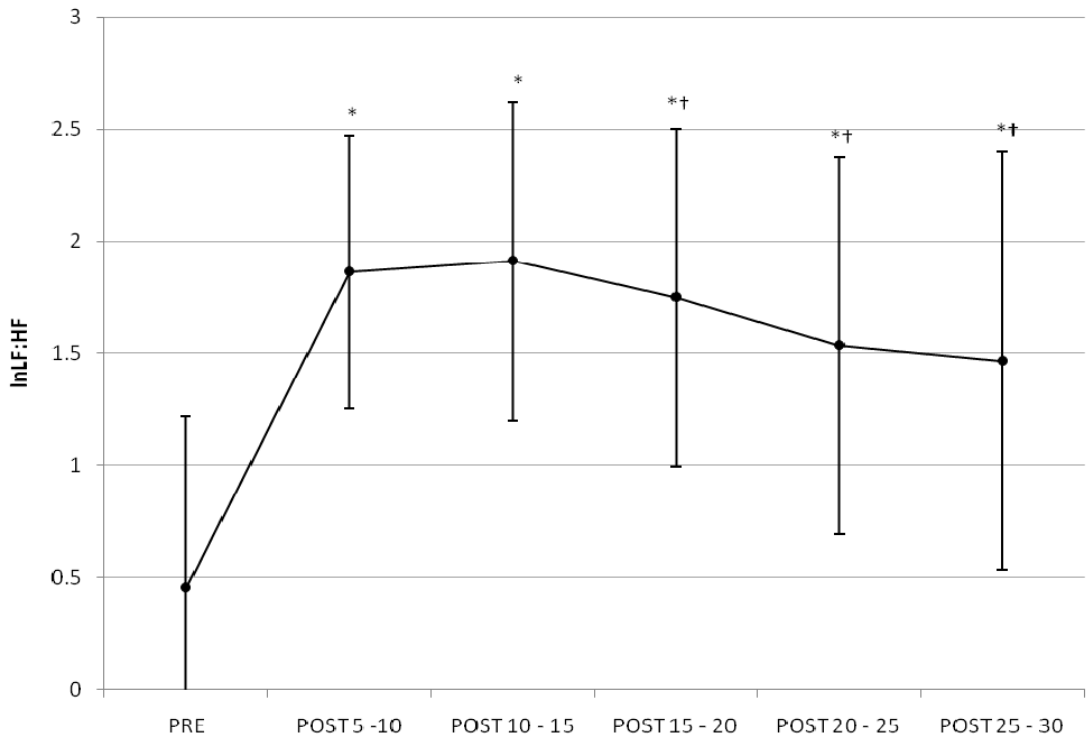

Figure 2. lnLF:HF before (PRE) and after (POST) maximal exercise
lnLF:HF = natural log tranformation of the low frequency to high frequency ratio. * Compared to PRE values, lnLF:HF was significantly increased at each POST 5-minute epoch ($p < .05$). † lnLF:HF was significantly lower compared to the previous epoch ($p < .05$), i.e., lnLF:HF gradually declined after POST 10 – 15 until POST 25 - 30, but did not reach resting values.

Table 1: Pearson Product Correlation values of HR recovery values and resting HRV.

	lnHF	lnLF:HF
MHR	-.331**	.396**
HR1	-.322**	.360**
HR2	-.241	.264*
HRR1	.181	-.192
HRR2	.048	-.037

MHR represents the heart rate recorded at VO_{2max}. HR1 represents the heart rate recorded at 1-minute post-exercise. HR2 represents the heart rate recorded at 2-minutes post-exercise. HRR1 represents the 1-minute heart rate recovery (i.e., the difference between MHR and HR1). HRR2 represents the 2-minute heart rate recovery (i.e., the difference between MHR and HR2). lnHF represents the log transformed high frequency parameter of resting (PRE) heart rate variability. lnLF:HF represents the log transformed ratio of low frequency power to high frequency power of resting (PRE) heart rate variability. * $p < .05$, ** $p < .05$

APPENDICES

APPENDIX A

RESEARCH STUDY

Racial Differences in Heart Rate Response to Maximal Exercise

We are seeking male volunteers between the ages of 19 and 35 to participate in a study examining the effects of race on heart rate recovery following a maximal graded exercise test.

To participate, the following criteria must be met:

1. African American/Black or Caucasian/White (over 3 generations)
2. Male
3. Between the ages of 19 to 35 years
4. Free from cardiovascular, pulmonary, or metabolic disease
5. Currently not taking prescribed cardiovascular or psychological medications

If you meet the criteria for the study you will receive a fitness assessment performed by trained exercise professionals that include the following:

1. Maximal aerobic treadmill test
2. Body fat assessment
3. Heart rate and blood pressure measurements at rest and during exercise

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

Michael Esco
Department of Physical Education and Exercise Science
Auburn University Montgomery
(334) 244-3161; mesco@aum.edu

APPENDIX B

(NOTE: DO NOT SIGN THIS DOCUMENT UNLESS AN IRB APPROVAL STAMP WITH CURRENT DATES HAS BEEN APPLIED TO THIS DOCUMENT.)

"The effect of race on cardiovascular autonomic modulation at rest and following maximal exercise"

INFORMED CONSENT

You are invited to participate in a research study to determine the effects of race on cardiovascular autonomic modulation at rest and following maximal exercise. The study is being conducted by Michael R. Esco, M.Ed., Instructor in the Auburn University Montgomery Department of Physical Education and Exercise Science and doctoral student in the Auburn University Department of Kinesiology, under the direction of Dr. Daniel Blessing, Ph.D. in the Auburn University Department of Kinesiology. You were selected as a possible participant because you have met the following inclusion criteria:

1. White/Caucasian or Black/African American of three generations
2. Male
3. Between the ages of 19 and 35 years
4. Free from cardiovascular, pulmonary, or metabolic disease
5. Currently not taking prescribed cardiovascular or psychological medications

If you decide to participate in this research study, you will be asked to report to the Human Performance Laboratory at Auburn University Montgomery on one day of the week during one of two time periods: from 7:00am to 9:00am; or 9:00am to 11:00am. During the visit, you will be screened with a health history questionnaire, height and weight will be measured, resting blood pressure will be examined, and your body fat percentage will be estimated with the use of a skinfold caliper before exercise testing.

You will also be asked to complete a maximal graded exercise test on a treadmill. Blood pressure and heart rate will be examined throughout the exercise test. Before and after the test you will be asked to assume a supine position (i.e., lying on your back, face up) while your heart's rhythm is analyzed with the use of electrocardiogram (ECG). Your total time commitment will be approximately 2 hours.

Participant's initials _____ Page 1 of 3

Your data could be excluded from analysis if any of the following are present:

1. You do not meet the above inclusion criteria
2. You are a smoker
3. Your blood pressure is above 140/90 mmHg

There are minimal risks associated with participation in this study. 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. There is also a possibility of nausea, dizziness, fainting, and/or fatigue as a result of exercise. The exercise test will be terminated if you experience any discomfort. Musculoskeletal injury (strain or sprains) and/or muscle soreness in the lower body could also occur 24 to 48 hours after the test. Should injury occur as a result of the experimental protocol you are responsible for any cost associated with medical treatment.

If you participate in this study, you can expect to obtain the results of your maximal graded exercise test and body fat percentage estimation. However, we/I cannot promise you that you will receive any or all of the benefits described. This study may benefit society in general in that the results will contribute to the body of knowledge regarding cardiovascular autonomic modulation.

If you change your mind about participating, you can withdraw at any time during the study. Your participation is completely voluntary. If you choose to withdraw, your data can be withdrawn as long as it is identifiable.

Your decision about whether or not to participate or to stop participating will not jeopardize your future relations with the Department of Physical Education and Exercise Science at Auburn University Montgomery, or the Department of Kinesiology at Auburn University, Auburn.

Your privacy will be protected. Any information obtained in connection with this study will remain confidential. Information obtained through your participation will be used to fulfill a doctoral dissertation and possibly published in a professional journal as well as presented at a professional meeting. None of your information will be identifiable.

If you have questions about this study, please inform Michael R. Esco (telephone: 244-3161, e-mail: mesco@aum.edu). A copy of this document will be given to you to keep.

If you have questions about your rights as a research participant, you may contact the Auburn University Office of Human Subjects Research or the Institutional Review Board by phone (334)-844-5966 or e-mail at hsubjec@auburn.edu or IRBChair@auburn.edu.

HAVING READ THE INFORMATION PROVIDED, YOU MUST DECIDE WHETHER OR NOT YOU WISH TO PARTICIPATE IN THIS RESEARCH STUDY. YOUR SIGNATURE INDICATES YOUR WILLINGNESS TO PARTICIPATE.

_____ _____

Participant's Name (Printed) Date

_____

Participant's Signature

_____ _____

Investigator's Signature Date

APPENDIX C

Emergency Procedures
AUM Human Performance Laboratory
7031 Senators Drive
Montgomery, AL 36117
Second Floor of the Physical Education Complex/Gym (Room 201G)
North Parking Entrance

1. Laboratory personnel will be currently certified in CPR
2. Telephone numbers and written copies of the following emergency procedures will be posted by the telephone in the laboratory
3. Laboratory personnel will be familiar with the practice emergency procedures
4. When performing testing procedures that could possibly cause a life-threatening situation, at least two persons should be present while the test is being performed.

Procedures for Life-Threatening Emergency:

1. Evaluate the extent of the accident or emergency.
2. If the situation is life threatening, one person will start emergency procedures while the second person notifies he emergency personnel.
3. Activate EMS (dial 911 on campus); after activating EMS, notify campus police (ext. 3424) of the situation.
4. Meet the ambulance and give EMS the following information
    a. Identify yourself
    b. The nature of the emergency – bleeding, unconscious, etc.
    c. Give the telephone number and physical location with directions on how to get there
    d. Speak slowly and clearly
    e. Advise them if extra help is needed due to stairs, number of people, etc.
    f. Allow the person on the other end to give instructions and ask questions until help arrives
    g. Always be the last to hang up
    h. Administer proper first aid until advanced medical personnel arrive
    i. Assist medical personnel in transporting the victim
    j. Fill out accidental report after emergency situation is resolved

APPENDIX D

Pre-participation Screening Questionnaire

Name: _____ Date: _____
Date of Birth: _____ Age: _____
Address: _____
City, State: _____ Zip: _____
Contact Phone Number: _____ E-mail address: _____
Personal Physician: _____ Physician's Phone: _____
Emergency Contact: _____ Relationship: _____

Mark all true statements in the following sections

You have or have had: ____ A heart attack ____ Heart surgery ____ Cardiac Catheterization ____ Heart valve disease ____ Heart failure ____ Heart transplantation ____ Coronary angioplasty (PCTA) ____ Congenital heart disease ____ Pacemaker / Implantable cardiac defibrillator / Rhythm disturbance ____ Diabetes. ____ A burning or cramping sensation in your legs when walking short distances. ____ You have asthma or other lung disease. ____ You have musculoskeletal problems. ____ Experienced chest discomfort with exertion ____ Experienced unreasonable breathlessness ____ Experienced dizziness, fainting, blackouts ____ You have concerns about the safety of exercise	*If you marked **any** of the statements consult your healthcare provider before engaging in exercise. You will not be allowed to participate in this study.*
____ You smoke, or quit smoking within the previous 6 months. ____ Your blood pressure is > 140/90 mm Hg (blood pressure will be analyzed before testing). ____ You take blood pressure medication. ____ You take prescription medication(s). If so, please list: _____ _____	*If you marked **any** of the statements in this section you will not be allowed to participate in this study.*
____ None of the above is true	*If you marked none of the above is true you should be able to safely participate in this study.*

Race/Ethnicity Background

Participant's ethnicity/race

Circle one:
Non-hispanic/black
Non-hispanic/white
Hispanic
Asian/Pacific Islander
Native/American Indian

Parent's ethnicity/race

Mother (circle one):	*Father (circle one):*
Non-hispanic/black	Non-hispanic/black
Non-hispanic/white	Non-hispanic/white
Hispanic	Hispanic
Asian/Pacific Islander	Asian/Pacific Islander
Native/American Indian	Native/American Indian

Maternal grandparent's ethnicity/race

Grandmother (circle one):	*Grandfather (circle one):*
Non-hispanic/black	Non-hispanic/black
Non-hispanic/white	Non-hispanic/white
Hispanic	Hispanic
Asian/Pacific Islander	Asian/Pacific Islander
Native/American Indian	Native/American Indian

Paternal grandparent's ethnicity/race

Grandmother (circle one):	*Grandfather (circleone):*
Non-hispanic/black	Non-hispanic/black
Non-hispanic/white	Non-hispanic/white
Hispanic	Hispanic
Asian/Pacific Islander	Asian/Pacific Islander
Native/American Indian	Native/American Indian

Other information/Comments: _____