EFFECT OF EXERCISE TRAINING ON TOTAL PERIPHERAL RESISTANCE, HEART RATE VARIABILITY, AND PREHYPERTENSION IN APPARENTLY HEALTHY AFRICAN AMERICAN WOMEN.

DISSERTATION

Presented in Partial Fulfillment of the Requirement for the Degree Doctor of Philosophy in the Graduate School of The Ohio State University

By

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Exercise training has consistently been shown, in the research literature, to be an effective non-pharmacological alternative to treat hypertension (HTN). However, the underlying mechanism that accounts for the reductive and possible preventive effects, induced by exercise have not been clearly established. To our knowledge, no study has examined the effect of an aerobic exercise-training program on prehypertension (resting blood pressure between 120/80 – 139/89 mm Hg) and the underlying variables that may exacerbate the disease’s development in apparently healthy African American women. Therefore, the aim of the present study was to investigate the effects of a 10-week aerobic exercise-training regimen on total peripheral resistance (TPR), heart rate variability (HRV), and prehypertensive blood pressure status in apparently healthy African American women. Twenty-three prehypertensive African American women served as study participants (12 exercised trained and 11 controls). Each participant was required to meet all inclusionary criteria which included: being an African American woman between the ages of 30-45 years, a sedentary lifestyle, non-smoking status, body mass index between 25-35 (kg m⁻²), and a prehypertensive blood pressure status. A VO₂ peak and submaximal exercise test were conducted on the cycle ergometer. HRV was assessed using power spectral analysis of beat-to-beat measurements in various frequency
domains. Results revealed a significant reduction in TPR in the trained group from pre to post intervention periods (pre $35.3 \pm 5.1$ mm Hg L min$^{-1}$ vs. post $26.9 \pm 4.3$ mm Hg L min$^{-1}$) ($p < .05$). The trained group also increased in VO$_2$ peak and absolute workload achieved ($19.0 \pm 1.1$ vs. $23.0 \pm 1.1$ ml kg$^{-1}$ min$^{-1}$; $116 \pm 4.7$ vs. $133 \pm 8.3$ watts, respectively) ($p < .05$). There were no observed significant changes in systolic, diastolic, or mean arterial blood pressure from pre to post-exercise periods in either group. However, the absolute change in resting SBP from pre to post when comparing the exercise and control group was statistically significant ($4.75$ and $-4.45$ mm Hg, respectively) (negative numbers indicate a increase from pre to post measurement periods) ($p < .05$). The findings of this investigation suggests that a 10-week aerobic exercise training protocol is sufficient enough to elicit changes in TPR, but was not a strong enough stimulus to reduce associated blood pressure levels in African American women with prehypertension.
DEDICATION

This dissertation is dedicated to all of the people that God placed in my life during the course of this journey who helped to encourage, motivate, and sustain me mentally, spiritually, and financially. I would also specifically like to dedicate this work to my family and friends who have believed in me every step of the way. Finally, a special dedication goes to my mother and best friend, Mary Goodwin who has shown me unconditional love and unwavering support through this entire process.
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CHAPTER 1

INTRODUCTION

1.1 Overview of Hypertension

Hypertension (HTN) is the most common clinical primary diagnosis in the United States, according to the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC) (Chobanian et al., 2003). Hypertension, also known as high blood pressure, is defined as a heightened pressure in the arteries, arterioles, and capillaries that is exerted by the heart as blood is pushed through the vasculature to the muscles of the body. To date, an estimated 50-60 million American adults have high blood pressure, which accounts for 60% of the total United States population. Unfortunately, of those that have high blood pressure 30% of these individuals don’t even know that they have the disease. However, among those who are aware that they have hypertension, an increased prevalence exists among African American women (44.7%) when compared to Caucasian and Mexican American women (25.5 and 29.9%, respectively) (Eisner, 1990; Frolich, 1990; & Dunn et al., 1983). Associated with having the highest hypertension rates in the world, more African Americans suffer from heart disease, kidney disease, and nonfatal stroke than any other ethnic group.
As a means to reduce the continuously increasing spread of HTN across all racial
groups and to address the growing disparity among minorities, a coalition of federal,
private, and professional entities was created to classify, identify and treat, or more
preferably, prevent high blood pressure development. Over the past decade the
committee has changed its blood pressure classification scale to recognize and
include individuals who don’t yet have high blood pressure, but have the propensity
to develop it. In a report published by the Joint National Committee (Chobanian et
al., 2003), persons with an increased risk of developing hypertension, based on
elevated blood pressure measurements, are identified as prehypertensive.
Prehypertension is a resting blood pressure measurement with a systolic reading
between 120-139mm Hg and/or a diastolic reading that falls within 80-89mm Hg.
Research has shown that individuals who are prehypertensive are 2-3 times more
likely to progress to stage 1 or 2 hypertension (Chobanian et al., 2003; Vasan et al.,
2001). Healthcare providers are encouraged to recommend lifestyle modifications
and/or employ preventive interventions in individuals who are classified as
prehypertensive (Chobanian et al., 2003). To date, epidemiological data reveal that
approximately 45 million or 22% of American adults have prehypertension. Of those
with prehypertension, some population groups, including African Americans, older
individuals, and obese persons are more susceptible and disproportionately affected
(Wang & Wang, 2004).
1.2 Pathogenesis of Hypertension: Possible Mechanistic Explanation

The etiology of HTN is an age-old question that still remains unanswered. The progress to better understanding high blood pressure has been limited to identifying different types of HTN, recognizing populations most prone to developing the disease, and strategizing effective anti-hypertensive treatment regimens. Little is known about the physiological mechanistic pathways that may help to explain why this disease is more common in certain populations. Although the specific cause(s) of high blood pressure remain unknown it is likely brought about by a combination of factors. In fact, there are two physiological mechanisms that are thought to greatly attribute to the disease’s maturation process. Morphological alterations in peripheral arteries that contribute to an increased total peripheral resistance (TPR) (Sihm et al., 1995; Lund-Johansen & Omvik, 1990) and a reduced heart rate variability, specifically heightened sympathetic activity (Singh et al., 1998) have been consistently seen in persons with HTN. The manifestation of either of these two physiological mechanisms is not dependent upon one another, but often occur dependently or simultaneously in hypertensive individuals.

1.2.1 Heart Rate Variability Hypothesis

Heart rate variability (HRV) refers to the balance of sympathetic and parasympathetic activity in the autonomic nervous system. Spectral analysis of HRV provides information about the modulation and interaction of cardiovascular autonomic control, through specific frequency bands that are associated with sympathetic and parasympathetic activity (Binkley & Cody, 1992). Variations in
instantaneous heart rate over time produces a waveform that is comprised of rapid fluctuations and slower sinusoidal variations. Computer-based processing isolates the variations within the waveform to reveal a low frequency and a high frequency band, which represents sympathetic and parasympathetic activity, respectively. Autonomic control governed by the sympathetic nervous system generates elevations in blood pressure and heart rate. The parasympathetically mediated high frequency band controls vagal tone and is linked to changes in the respiratory cycle. The interaction between HRV and blood pressure reflect a trend, which shows that a reduction in HRV is consistently associated with elevated blood pressure levels (Novak et al., 1994; Amano et al., 2001; Singh et al., 1998). This means that individuals that have lower total HRV typically have higher resting blood pressure levels. Therefore, the objective of most researchers concerned with HRV’s influence on blood pressure is to identify treatment interventions that will increase HRV and improve blood pressure. More specifically, studies have shown that sympathetic autonomic modulation is exaggerated in persons with high blood pressure (Piccirillo et al., 1996 & Singh et al., 1998) indicating low frequency hyper-reactivity. In turn, the parasympathetic activity tends to be diminished in persons with elevated blood pressure levels, which may play a significant role as a mechanism involved in the pathogenesis of hypertension (Julius, S., 1991).

1.2.2 Total Peripheral Resistance Hypothesis

The total peripheral resistance hypothesis suggests that a direct relationship exists between TPR and blood pressure. As TPR increases, an
elevation in blood pressure is also expected to occur. TPR is defined as the total amount of resistance to blood flow in all of the blood vessels in the peripheral extremities (Seely, Stephens, Tate; 1995). A structural change in the cardiovascular system of hypertensive individuals is a long-standing phenomenon that supports the elevation in TPR (Koerner et al., 1987). Common morphological changes in the peripheral resistance vessels of the person with high blood pressure includes an increased media thickness and a widened lumen diameter resulting in an increased media: lumen ratio. In a study conducted by Sihm et al., (1995) control subjects revealed a media: lumen ratio of 7.9% in the arteries while hypertensive patients showed a 10.2% ratio (1995). Other investigations confirmed the position taken by Sihm and associates (1995), by demonstrating that an increased media thickness and increased media: lumen ratio is consistently seen in persons with hypertension (Aalkjaer et al., 1987 & Izzard, A.S., et al., 1991). Furthermore, a significant correlation was found between the media: lumen ratio and ambulatory blood pressure levels. Structural remodeling in peripheral vessels may result from added pressure on the vessel walls as blood travels through a reduced space. The added thickness and diameter of the inner and outer structure of the vessel that is commonly seen in persons with HTN creates a constricted area in which blood must flow. Therefore, it is not a surprise that persons with HTN of any form have an increased total peripheral resistance.
1.3 Proposed Study

As was alluded to earlier, the pathogenesis of HTN development is likely a multifactorial approach, which includes behavioral, environmental, and physiological factors. However, two physiological explanations have recently emerged as constant links in providing mechanistic insight into the evolution of high blood pressure. These two mechanistic variables, which are often impaired in persons with HTN, include TPR and HRV. Therefore, developing interventions that can alter, attenuate the deleterious effects of HTN on these physiological factors is essential.

Structured physical activity has become a standard non-pharmacological intervention used to attenuate the development of hypertension and other related diseases. The existing literature reports that regular exercise training significantly reduces high blood pressure (Dickey & Janick, 2001; Pescatello et al., 2004; Silverberg et al., 1990, & Izdebska et al., 1998). In fact, the association between regular physical activity and improved health with a reduction in defective physiological traits, responses, and disease developments, such as hypertension, was so strong that in 1996 the Surgeon General published a report endorsing moderate intensity exercise. However, the mechanistic pathway by which aerobic exercise demonstrates its reductive effect is less conclusive. In large, the current stance regarding exercise’s protective effect is based on its ability to induce changes in various physiological markers. According to Iwasaki and associates (2003), exercise training regulates blood pressure by efficiently controlling dynamic cardiovascular processes, particularly vascular resistance and HRV. Despite landmark findings that suggest that exercise has been an influential intervention, research is often confined to exploring its effectiveness in a male Caucasian subject population (Hagberg
et al., 2000). To date, only a few studies have been published which have focused on the effects of aerobic exercise in a female African American cohort, none of which included women with a prehypertensive blood pressure status. Furthermore, we have not identified any studies that have examined the effects of exercise on the physiological responses of the cardiovascular system that are associated with the development of hypertension in African American women.

Since previous prospective investigations assessing the effectiveness of exercise on physiological parameters that affect hypertension did not include prehypertensive African American women, this study seeks to place this under-studied increased-risk population as the focus. Therefore, to address the paucity that exists in the scientific literature on this topic, we studied the effect of aerobic exercise training on TPR, HRV, and prehypertension in apparently healthy African American women.

1.3.1 Hypothesis

The following null hypotheses were tested during this research investigation: 1) Aerobic exercise training would have no effect on heart rate variability (HRV). 2) Aerobic exercise training would have no effect on total peripheral resistance (TPR). 3) Aerobic exercise training would have no effect on prehypertensive status. There were two secondary null hypotheses included: 1a) Social support would not change during the 10-week intervention period. 2a) Perceived mental stress would not change during the 10-week intervention period. Collectively, examining each specified hypothesis might help to identify the mechanisms by
which aerobic exercise training attenuates the development and progression of hypertension.
CHAPTER 2

THE EFFECTS OF AEROBIC EXERCISE TRAINING ON HEART RATE VARIABILITY IN APPARENTLY HEALTHY AFRICAN AMERICAN WOMEN WITH PREHYPERTENSION

2.1 Introduction

Heart Rate Variability (HRV) is a reliable and reproducible non-invasive technique used to assess the cardiac modulation of the autonomic nervous system. Previous studies have indicated that a sympathovagal imbalance exists in the HRV spectral analysis of persons with hypertension (HTN) (Takalo et al., 1994, Guzzetti et al., 1991, & Singh et al., 1998). Specifically, a greater reliance on heightened sympathetic activity and a depressed vagal (parasympathetic) tone are the most commonly seen alterations that contribute to a reduced HRV in hypertensive individuals (Julius, S., 1991). Diminished HRV is associated with an increased risk of developing coronary heart disease, ventricular arrhythmias, and cardiac sudden death (Davy et al., 1997). Although it has been established that an abnormal HRV is also associated with hypertension and may provide insight into the pathogenesis of the diseases’ development, little is known about persons with only a prehypertensive status (Singh et al., 1998).
Furthermore, age, ethnicity, and cardiorespiratory fitness level have demonstrated distinct associations with HRV, but none of these variables have been assessed in persons with an increased risk of progression to HTN (prehypertension). According to Gregoire et al., (1996) an increase in age is accompanied by a rapid decline in HRV, while Arthur and associates (2004) note that ethnicity, particularly the African American genealogy is also associated with a reduced HRV. Additionally, lower HRV has been consistently identified in sedentary and low fit individuals when compared to their physically active counterparts (De Meersman ME., 1993, Amano et al., 2001, & Goldsmith et al., 1997).

Several researchers have noted that demographic variables, such as ethnicity and gender, influence HRV function and cardiovascular reactivity (Gregoire et al., 1996; Calhoun et al., 1993; Gillin et al., 1996). In fact, cardio-reactivity, such as a reduced HRV, has been proposed as a potential mechanism partially responsible for the increased prevalence of HTN development in African American individuals when compared to white persons (Dysart et al., 1994). According to Arthur et al., (2004) Caribbean Americans and African Americans, two black ethnic groups, significantly differed from Caucasian Americans in cardiovascular reactivity. Findings of this study also revealed that differences even existed within two black populations (Caribbean and African Americans), which further underscore the need to consider ethnicity when evaluating potential causes of HTN and associated abnormalities (Arthur et al., 2004). Furthermore, in an investigation led by Dysart et al., (1994) reactivity was assessed in a group of black and white adolescent girls where African American girls exhibited higher blood pressures, increased total peripheral resistances, and elevated cardiac indexes, all indicators of an altered HRV pattern. Researchers have concluded that the hyper-
responsivity to various stressors may be a significant risk factor for the subsequent development of elevated blood pressure levels, which may be one pathway responsible for the greater prevalence of HTN seen in African Americans (Knox et al., 2002). Despite the fact that race and gender have been shown to have an affect on cardiovascular responses to various stressors few studies have highlighted African Americans or addressed the documented differences commonly seen in this population. Of the studies that have been conducted that do acknowledge race, few populations included women. This is very disturbing since the percentage of women that have HTN is greater than men after the age of 55 (Heart Disease and Stroke Statistics, 2004 Update). Therefore, it is greatly justified that more researchers should focus on African American women when studying HTN, because these women are more susceptible to developing the disease.

Based on the significance of a depressed HRV and the implications that are provided regarding the dynamic interaction of the sympathetic and parasympathetic branches, interventions that could possibly enhance autonomic nervous system activity are of great importance. Therefore, exercise has become a highly employed non-pharmacologic approach used to augment HRV status (Melanson & Freedson, 2001). The influence of exercise training on HRV has been seen in numerous studies (Jurca et al., 2004, Davy et al., 1996, & Leicht et al., 2003). In 1996, Davy and his colleagues measured HRV in nine physically active postmenopausal women and eleven of their age-matched sedentary counterparts. Results revealed that the physically active women, who ran an average 32.6 ± 3.6 miles per week, had a significantly higher total power of HRV than the control participants (Davy et al., 1996). In yet another study, HRV was analyzed at rest and exercise in 13 healthy untrained subjects (Leicht et al., 2003). Each
participant cycled for 5-days/ week for 25-60 minutes a day for 8 weeks. The results of
Leicht and associates (2003), revealed an increased HRV, particularly the high frequency
activity, which is associated with cardiac vagal modulation. These results (Leicht et al.,
2003) and the conclusions drawn by other researchers, support the notion that aerobic
exercise improves HRV. Despite the findings in a majority of studies and those
previously mentioned, the ideology regarding the effectiveness of exercise training on
HRV remains inconclusive. For instance, a year after completing previous supportive
exercise training research on HRV, Davy et al., (1997) found that 12 weeks of moderate
intensity aerobic exercise did not increase HRV or associated baroreflex sensitivity in
sedentary women with elevated blood pressure levels. Heart Rate Variability also
remained unchanged in a male cohort after 24 sessions of moderate intensity exercise
training (Boutcher & Stein, 1995). The contrasting findings about HRV suggests that the
intensity, duration, and frequency of training, as well as the subject population examined,
are major determinants regarding the effects of exercise. As such, little is known about
the implications that exercise training has on the autonomic nervous system in
prehypertensive African American women. To address the paucity in the literature, the
purpose of this study was to assess the effect of a 10-week aerobic exercise training
regimen on HRV in sedentary African American women with prehypertension. To draw
conclusions, we measured the frequency domains (total power, high, low & the ratio) of
HRV at rest, before and after, 10 weeks of aerobic exercise training in a prehypertensive
African American cohort.
2.2 Methods

2.2.1 Subject Population

Twenty-eight previously sedentary African American women between the ages of 30-45 years volunteered to participate as subjects in the present study. However, five women were not able to complete the study: two women were not able to comply with the training guidelines set for the exercise-training group and therefore were removed from the study; one participant became seriously ill during the course of the study and was not able to return for post testing; two other women withdrew from the study with no explanation or notice as to why. All potential participants were required to meet the following inclusionary criteria: (1) African American woman (2) nonsmoking status, (3) sedentary lifestyle, (4) between the ages of 30-45 years, (5) body mass index (BMI) between 25-35 kg/(m)$^2$, (6) absence of medication use, including birth control pills, that would affect blood pressure or cardiorespiratory fitness, (7) prehypertensive blood pressure status, as defined by the Joint National Committee on the Prevention, Detection, Evaluation, & Treatment of High Blood Pressure (JNC), and (7) freedom from all other known diseases that affect blood pressure status and/or response. Physical characteristics were obtained during the subject’s initial visit, and are presented in Table 2.1. Initial physical activity levels of each participant were assessed using the Beacke Physical Activity Questionnaire (Sternfeld et al., 1999). Each subject received a full explanation as to the purpose, procedures, and risks, associated with participating in the study prior to giving their written informed consent. Subjects were randomized to either the control or exercise training group prior to any physiological testing.
Table 2.1 Preliminary Physical Characteristics of Subjects (n = 23)

There were not any preliminary physical characteristics statistically significant at the beginning of the study between the control and exercise-trained group. Values presented as means ± SE.

### 2.2.2 Blood Pressure Measurements

Resting blood pressure was measured twice on two separate occasions, once during the initial orientation session and again at the start of the first testing day, abiding by the JNC 7 published guidelines to establish a prehypertensive blood pressure status. Subjects were seated in a chair for 5 minutes with their dominant arm resting on a table at heart level, prior to the blood pressure measurement being taken. A standard sphygmomanometer, stethoscope, blood pressure cuff, and mercury column pressure gauge was used to measure blood pressure by auscultation. An experienced and certified American College of Sports Medicine, Exercise Specialist manually took all of the resting blood pressure measurements. A systolic reading between 120-139 mm Hg or a
diastolic reading of 80-89 mm Hg was classified as prehypertensive (JNC 7 report - Chobanian et al., 2003).

Blood pressure was continuously monitored with the subject lying in the supine position during the HRV test using the finger photoplethysmography method (Finapres Blood Pressure Monitor, Ohmeda 2300 BOC Healthcare, Englewood, CO). Assessing blood pressure using this technique allowed arterial blood pressure to be measured in a beat-by-beat fashion, yielding similar results expected from intra-arterial monitoring (Parati et al., 1989). The blood pressure measurement taken during the HRV test was used to track changes in systolic and diastolic measurements that may have occurred simultaneously with HRV variations. This continuous method of tracking blood pressure was assessed before and after the exercise intervention in conjunction with the HRV evaluation.

Casual blood pressure was taken before and after the intervention period to examine if exercise had any effect on prehypertensive status. The pre-intervention blood pressure was taken during the subject’s third visit to the laboratory after resting for 5 minutes in the seated position. The post-intervention blood pressure was taken 3-5 days after completing the 10-week period. Again subjects were required to rest in the seated position for 5 minutes before the blood pressure reading was taken. A Colin STBP-780 automated blood pressure tracking system (Colin Medical Instruments, Plainfield, NJ) was used on all casual measurements. The Colin STBP-780 is a stand-alone noninvasive blood pressure stress test monitor that is capable of taking accurate and reliable blood pressures at rest and during exercise. The system is interfaced to work with a standard electrocardiogram using lead 2, complete with a TTL and analog input. These standard
built-in interface connections allow for easy cardiovascular assessment. Auscultatory sounds are monitored using two microphones synchronized to the R wave to help reduce inaccurate readings caused by “noise” (Colin STBP-780 Manual). A regular adult size cuff was placed on the subject’s upper arm with a stabilizing wrist strap to measure blood pressure at the brachial artery. Subjects were hooked up to four electrodes to create the standard lead two view. To reduce diurnal variations the researchers attempted to take casual pre and post intervention blood pressures at the same time of day.

2.2.3 Heart Rate Variability Analysis

Each subject’s autonomic nervous system activity was measured and analyzed in the resting condition using power spectral analysis. Analysis of the beat-to-beat RR interval spectral power has been detailed and fully explained by Binkley & Cody (1992) & Erhman, J. (1996). A standard analog output of a 5 lead ECG unit was used for all recordings. Beat-to-beat measurement of the frequency domain was recorded using a standard data tape recorder (Racal V-Store 8 channel Instrumentation recorder, Racal Recorders Limited, or TEAC R-61 4 channel data recorder, TEAC Corporation). For analysis, 4 minutes of collected data were played back into a Gateway 2000 computer using the DATAQ Corporation acquisition software. Heart Rate Variability was be reviewed by MVARAN custom written software (Enrico Nunziata copyrighted 1993). Complete autonomic nervous system activity measurement included low frequency (LF) (0.02-0.14 ms\(^2\) Hz\(^{-1}\)), high frequency (HF) (0.14-0.9 ms\(^2\) Hz\(^{-1}\)), and total power (TO) (.02-0.9 ms\(^2\) Hz\(^{-1}\)).
2.2.4 Peak Oxygen Uptake Test (VO₂ peak)

After completing the initial orientation session, eligible participants returned to the laboratory to perform a maximal graded exercise test on an electrically braked cycle ergometer (Cybex, Division of Lumex, Ronkonkoma, LI, NY). Subjects were prepped for a standard 12-lead ECG (Quinton 4500 Stress Test Machine, Seattle, WA) wearing 10 electrodes and a heart watch monitor (Polar Electro Inc., Polar A1, Woodbury, NY). Prior to the test, headgear was placed on the subject’s head to support the weight of the mouthpiece attached to the open circuit system used to assess VO₂ peak. Indirect calorimetry using a Parvo Medics True One Metabolic System package (OUSW Version 3.5, 2003) measured the amount of oxygen consumed and carbon dioxide produced. Gas and flow meter calibrations were done before each test to ensure accurate readings (Parvo MMS-2400, Computer/DAC & Adapter PCL 711B). The test was terminated when subjects could not maintain the prescribed pedal rate, reached volitional fatigue, demonstrated a leveling off or decrease in VO₂ peak with increasing workload, or attained a RER exceeding 1.0. The cycling protocol began with a work rate of 25 watts, which increased an additional 25 watts every two minutes. Each subject was instructed to cycle at or above the required pedal cadence of 60rpm.

2.2.5 Exercise Training Protocol

Subjects who were randomly assigned to the exercise-training group (N = 12) were required to undergo 10 weeks of supervised aerobic exercise. Their control counterparts (N = 11) were instructed to maintain their normal sedentary lifestyles during the 10-week intervention period. Over the training period, training subjects exercised 3
times per week for 30 minutes per session. Individual exercise prescriptions based on standard equations, from the 6th Edition of the American College of Sports Medicine Guidelines Resource book (Franklin et al., 2000), were written for each subject in the exercise-training group with an initial intensity corresponding to 70% of their peak oxygen consumption. The training dose was progressively increased by 5% every 2-½ weeks to match the expected physiological adaptation that would take place over the 10 weeks (see Table 2.2 for example). Heart rate was continuously monitored during training sessions using a Polar heart watch monitor and chest strap unit. Training intensity was maintained and monitored by keeping the subject within 5 beats of the specified target heart rate. At the end of each exercise session subjects completed a two minute cool down period of slow walking (1.7 mph/ 0% grade) or reduced cycling (25 watts). Exercise training was completed using the stationary cycle ergometer, air-dyne, and/or treadmill at the discretion of the participant.

In order to meet the study’s exercise training compliance requirements, subjects in the training group were required to attend 75% of the 30 original exercise sessions prescribed. This rate of adherence was consistent with the average range set by most researchers of 50% to 80% of prescribed sessions versus those completed (Buckworth & Sears, 2003). Three exercise sessions were set per week over the 10-week period for each training subject. Additional sessions were provided during the week and on the weekend to allow subjects to make-up any missed appointments.
2.2.6 Procedures Timeline

Each subject came into the laboratory following the same order of events (See Figure 2.1 for Graphical View). During the first visit to the exercise physiology laboratory each subject received a thorough orientation and facility tour. The orientation session included an explanation regarding the purposes, procedures, possible risks/benefits, and participant expectations of the study. If the potential subject met all inclusionary criteria, an informed consent was administered and resting blood pressure (BP) was taken. Medical clearance forms for physicians were given to each subject, to be returned signed upon their next visit. The subjects’ second visit to the laboratory included filling out two psychological questionnaires that included the Interpersonal Evaluation List (ISEL) to measure social support and the Perceived Stress Scale (PSS) to measure mental stress, a second resting blood pressure assessment, and a VO₂ peak test. During that same week, subjects were asked to return to the laboratory on a different day to complete the HRV analysis. A causal blood pressure reading was taken prior to the start of the HRV test. After successfully completing both pre-tests, subjects were randomized into the control or exercise group. Over the next ten weeks, subjects were instructed to follow the prescribed guidelines for the intervention group in which they were randomly assigned. After the 10-week intervention each subject returned to the exercise-testing laboratory, within one week, to repeat the VO₂ peak and HRV analysis. Post questionnaires were also administered after the 10-week intervention to re-assess social support and perceived stress. Blood pressure was again measured at rest following the 10-week intervention to assess any changes in prehypertensive status.
Subject ID # __000__ Wt __180 lbs__

<table>
<thead>
<tr>
<th>VO2 peak</th>
<th><strong>20.1 mL·kg⁻¹·min⁻¹</strong></th>
<th><strong>14.07 mL·kg⁻¹·min⁻¹</strong></th>
<th><strong>15.075 mL·kg⁻¹·min⁻¹</strong></th>
<th><strong>16.08 mL·kg⁻¹·min⁻¹</strong></th>
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<tr>
<td>80%</td>
<td>_</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Mode of Exercise</th>
<th>Intensity</th>
<th>Week of Training</th>
<th>Speed</th>
<th>Incline</th>
<th>Wattage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treadmill</td>
<td>70%</td>
<td>1-2 wks</td>
<td>2.5 mph</td>
<td>3.2%</td>
<td>53 watts</td>
</tr>
<tr>
<td>Bike</td>
<td>70%</td>
<td></td>
<td>53 watts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treadmill</td>
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<td>3-5 wks</td>
<td>3.0 mph</td>
<td>2.4%</td>
<td>61 watts</td>
</tr>
<tr>
<td>Bike</td>
<td>75%</td>
<td>3-5 wks</td>
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<td></td>
</tr>
<tr>
<td>Treadmill</td>
<td>80%</td>
<td>6-8 wks</td>
<td>3.4 mph</td>
<td>2.1%</td>
<td>68 watts</td>
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<tr>
<td>Bike</td>
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<td>6-8 wks</td>
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<tr>
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<td>9-10 wks</td>
<td>3.8 mph</td>
<td>1.8%</td>
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<tr>
<td>Bike</td>
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<td>9-10 wks</td>
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</tr>
</tbody>
</table>

**Table 2.2 Example Exercise Training Protocol**

Exercise prescriptions were derived using the American College of Sports Medicine (ACSM) regression equations outlined in the 6th Edition of the ACSM’s Guidelines for Exercise Testing and Prescription Handbook. Each subject received an individual exercise prescription based on her initially measured VO2 peak value. Calculated intensities served only as a guideline to implement training.

![Figure 2.1 Timeline Graphical View](image-url)
2.2.7 Statistical Analyses

Physiological data (total, high and low frequency values) of HRV and psychosocial indices were analyzed as an absolute change between pre and post measures. To ensure the accuracy of the randomization process comparative analysis on pre-intervention descriptive (age, BMI, physical activity status) and dependant variables were conducted.

To assess the effectiveness of the 10-week aerobic exercise training protocol on total, high, and low frequencies of HRV (pre vs. post intervention) within the control and exercise groups a Wilcoxon signed-rank test was used. Another nonparametric test, Mann-Whitney U test (Wilcoxon rank-sum), was applied to identify any differences in absolute change between the control and trained groups in regard to the specified frequency bands of HRV. Statistical significance was set a priori with a p value ≤ .05. Data was expressed as mean ± SE. All statistical procedures were performed using SPSS (Window version 13.0).

2.2.8 Results

Initial physical characteristics for the control and exercise trained group are listed in Table 2.1. There were no significant differences (p > .05) in age, height, weight, BMI, physical activity status, or blood pressure between the control and exercise trained group. In addition, we examined three frequency bands of heart rate variability (total, LF, and HF) before and after the 10-week intervention to assess if the control and exercise trained group were statistically different in absolute change from pre to post and we found no significant difference between the two groups (see Table 2.3). However, the apparent
difference in absolute changes seen in total HRV suggest that moderate intensity exercise may maintain HRV in prehypertensive African American women, protecting the trained group from the obvious drop demonstrated in the control group over the 10-week period. Negative numbers in absolute change indicate that there was an increase in post-exercise values when compared to pre-exercise values. Absolute change is calculated by taking the pre value minus post value. Furthermore, independent analyses were run for the control and exercise training group. As expected, the control group remained unchanged in total, LF, and HF HRV activity before and after the 10-week intervention period (total pre 366.7 ± 49.7 ms² Hz⁻¹, total post 310.2 ± 23.4 ms² Hz⁻¹; LF pre 207 ± 39.1 ms² Hz⁻¹, LF post 188.9 ± 27.8 ms² Hz⁻¹; HF pre 158.8 ± 27.2 ms² Hz⁻¹, HF post 119.7 ± 21.9 ms² Hz⁻¹)(see Figures 2.2 & 2.3). In the present study our primary focus was to evaluate the effects of a 10-week aerobic exercise training protocol on the frequency domain of heart rate variability. Our findings demonstrated that pre vs. post total, LF, and HF HRV also remained unchanged in the exercise-trained group (total pre 321.3 ± 29.2 ms² Hz⁻¹, total post 329.8 ± 19.4 ms² Hz⁻¹; LF pre 218.1 ± 23.0 ms² Hz⁻¹, LF post 196.2 ± 21.6 ms² Hz⁻¹; HF pre 100.7 ± 17.0 ms² Hz⁻¹, HF post 131.9 ± 22.0 ms² Hz⁻¹) (see Figures 2.2 & 2.3).

Despite the lack of change in any of the HRV frequency bands, within and between the control and exercise trained groups other physiological changes were noted. Exercise training statistically increased VO₂ peak in the exercise group (pre 19.0 ± 1.1 vs. post 23.0 ± 1.1 ml·kg·min⁻¹) (see Figure 2.4). No difference was found among the control group before and after the 10-week intervention period (pre 18.0 ± .98 vs. post 17.5 ± .88 ml·kg·min⁻¹) (see Figure 2.4). The effectiveness of the exercise training protocol was also shown in the maximal workload achieved in the exercise group. Results revealed a
significant increase in workload expressed in wattage among the exercise group (pre 116 ± 4.7 vs. post 133 ± 8.3 watts) (see Figure 2.5). There were not any significant differences observed in workload in the control group (pre 106.8 ± 4.0 vs. post 95.4 ± 7.6) (see Figure 2.5). Although submaximal heart rate was not statically significant in the exercise group a trend towards significance was revealed (exact significance 2-tailed, p = .07), which may have clinical relevance, was also noted. It is important to note that the effect size was small for submaximal heart rate.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Trained</th>
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<tbody>
<tr>
<td>HF (ms² Hz⁻¹)</td>
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</tr>
<tr>
<td>LF (ms² Hz⁻¹)</td>
<td>18</td>
<td>21</td>
</tr>
<tr>
<td>Total (ms² Hz⁻¹)</td>
<td>56</td>
<td>-8</td>
</tr>
</tbody>
</table>

Table 2.3 Absolute Changes in HRV Frequency from Pre and Post Intervention

There were no significant differences in any of the frequency bands of HRV in the control or exercise-trained group when looking at absolute values from pre and post periods. Values are expressed as the mean.

2.2.9 Discussion

The present study evaluated the effectiveness of aerobic exercise training in apparently healthy African American women. The findings of this study do not support the premise that moderate intensity exercise improves HRV and therefore agrees with
researchers such as Boucher et al., (1995), Davy et al., (1997), and Aronne et al., (1995) who also did not demonstrate the effectiveness of this type of training to influence HRV. On the contrary, popular belief among the majority of researchers is that aerobic exercise training is a beneficial modulator in increasing HRV (Jurca et al., 2004; Pigozzi et al., 2001; Melanson & Freedson, 2001; De Meersman, 1993). This notion was particularly promising because reduced HRV has been associated with a greater incidence of ventricular arrhythmias, sudden cardiac death, and all cause mortality (Davy et al., 1997). Moreover, associations between reduced HRV and physical/behavioral characteristics such as older age, high blood pressure, sedentary lifestyle, and smoking status have also been observed. Specifically important to this investigation is the finding of Singh et al., (1998), which states that HRV is reduced among men and women who are hypertensive. Additionally, in their follow-up analysis men who were previously normotensive with a reduced LF of HRV had a greater risk of developing high blood pressure than did their normotensive counterparts who had higher LF activity (Singh et al., 1998). Therefore, introducing exercise as an intervention to augment HRV became a well-accepted alternative. The supportive stance that many people take regarding exercise on HRV is based on several comparative studies that documented that persons who are physically active have higher HRV than those persons who have led inactive lifestyles. For instance, Gregoire et al., (1996) demonstrated that significant elevations in HRV, particularly a greater reliance on the high frequency band of HRV, which is associated with parasympathetic activity, was seen in those individuals who were trained. It is important to note that the individuals in the Gregoire et al., (1996) study were already either trained or untrained when coming into the study and were placed into groups based
on their previous physical activity choices. The present study and many other studies of this type usually employ an exercise program in a designated population (sedentary, athletes, etc), which allows the researcher to actually assess the subject’s fitness level at the beginning of the study and monitor changes after the intervention. In Gregoire et al’s (1996) study the effects of a specific training regimen were not examined. Furthermore, the persons who were considered trained were those individuals who participated in moderate to high intensity exercise 5 days per week for at least 45 minutes a day prior to participating in the study (Gregoire, 1996). This may be problematic when assessing the potential benefits of exercise in improving HRV among the general public because epidemiologic data reveal that 70% of United States adults are sedentary or are active below the recommended 30 minutes of exercise per day (Trafton & Gordon, 2004). This point suggests that since most Americans are not at or above the physical fitness level of the persons described as trained individuals in this study, they may not achieve the training threshold needed to have a significant impact on HRV. Although not statistically significant, the higher HRV seen in the trained versus untrained group may also be due to the fact that the trained group’s HRV prior to them adopting an active lifestyle was already higher than those individuals classified as untrained. Although the groups were age and gender matched other variables that were not accounted for, could be possible explanations for the differences seen between untrained and trained subjects. In yet another investigation, it was concluded that moderate to vigorous intensity exercise programs were successful in eliciting increases in both the time and frequency domains of HRV (Melanson & Freedson, 2001). In a investigation carried out by Melanson & Freedson (2001), eleven sedentary males, trained 3 times per week for 30 minutes per
session at an intensity equivalent to 80% of their heart rate reserve. Five sedentary males did not engage in physical activity during the 16-week intervention and served as controls. The findings of this study revealed an increase in the HF component of HRV, adjacent intervals differing more than 50ms, and the root mean square of successive differences between RR intervals in the exercise trained group. Although, improvements were seen in HRV these changes did not manifest until week twelve of the study. Since HRV was measured every four weeks in this study, it is logical to assume that exercise training was not effective with only 4 or 8 weeks of training. Amano et al., (2001) further supported the need of at least 12 weeks of aerobic exercise training in their study. Eighteen obese individuals (BMI < 25) served as subjects by participating in an exercise training protocol that required each subject to exercise for 12-weeks at his/her anaerobic threshold for 20 minutes, three times per week. There was not a control group in this study. The 12-week exercise training resulted in improvements in both HF and LF bands of HRV, which corresponds to greater parasympathetic and sympathetic activity, respectively (Amano et al., 2001). In addition to the 12-week long exercise intervention introduced, the significant reduction in body weight and percent fat, as well as the gender of participants which were not identified may have accounted for the significant changes seen in HRV.

In contrast to the findings of Gregoire et al., (1996), Melanson & Freedson, (2001), and Amano et al., (2001), Boutcher & Stein, (1995) did not demonstrate any change in HRV after a 24-session moderate intensity exercise training protocol. Forty sedentary middle-aged men were randomly assigned to either the control or exercise training group. Subjects exercised three days per week at an intensity of 60% of their
heart rate range. The lack of change in heart rate variability may be due to a variety of things. First, the exercise training was only implemented for eight weeks. This is four weeks shorter than the length required by Amano et al.’s to see a training effect in HRV. Secondly, Boutcher & Stein (1995) noted that the pre-exercise HRV levels of their subjects were substantially lower than other researchers who revealed an enhancing effect of exercise. Finally, the method used by Boutcher & Stein (1995) to measure HRV may not have been sensitive enough to detect changes in the various frequency bands analyzed in their study. Further supporting the stance that exercise does not increase HRV was a study carried out by Davy et al., (1997). The target population in this study was eight sedentary older women between 50-60 years of age with high blood pressure. Subjects in this study engaged in an aerobic exercise training regimen 3 times per for 12-weeks at an average intensity of 70% ± 2% of their heart rate maximal, which is equivalent to 59% ± 3% of their VO₂peak. Each session lasted for 43 ± 3 minutes per day. The results of this study showed that while the exercise program employed was sufficient to elicit reductions in blood pressure the training stimulus was not strong enough to evoke an increase in HRV. Therefore, the expected concomitant improvement in both blood pressure and HRV was not observed.

As previously demonstrated there are a number of factors that can account for the inconclusiveness regarding the effectiveness of exercise to improve HRV. The main differences in many of the studies that disagree about the effect of exercise on HRV include the intensity of exercise, the session duration, frequency of sessions, program length, method used to measure HRV, and the population of interest. In comparing the previous studies discussed, several inherent differences can be seen. One of the obvious
differences seen among these studies is the length of the training protocol. In the studies that support exercise as an effective treatment intervention to improve HRV, a 12-week protocol was the shortest duration used. Where as, Boutcher and Stein (1995) and the present study implemented an 8-week and 10 week intervention, respectively. Although Davy et al., (1997) had a training regimen that was equivocal in length to the minimum time used in the supportive studies, their subject’s training intensities were significantly lower. Based on the previous discussion, despite the findings of Davy et al., (1997), it appears that at minimum a 12-week protocol is needed to induce changes in HRV and since the present study only employed a 10-week program changes in HRV were unlikely to occur. However, implementing a 10-week exercise program was not a design flaw, but a strategic plan to induce improvements in HRV, while preserving a high compliance rate. In an investigation done by Jurca et al., (2004) eight weeks of moderate intensity aerobic exercise elicited increases in HRV. The training protocol designed by Jurca et al., (2004) was set at an intensity that was equivalent to only 50% of the VO2 max, with an average duration of 44 minutes per session for 3-4 times per week. Results of this study demonstrated an increase in all absolute time and frequency domains of HRV in the exercise-trained group. In yet another study, findings revealed that a five-week exercise program decreased the day-night difference in the time domain of HRV (Pigozzi et al., 2001). The decrease in the time domain indexes of HRV from day to night indicate that a five-week exercise training program was effective in inducing an increase in sympathetic modulation of the primary pacemaker which the researchers suggest coexists with an relatively reduced vagal tone (Pigozzi et al., 2001). In 1992, an investigation was carried out by La Rovere and associates which assessed the effectiveness of a 4-week physical
training program on autonomic balance in post-myocardial infarction patients. Low frequency and HF autonomic activity was measured at rest and during a tilt table test. Their data suggests that the LF and HF spectral profile was slightly improved after the 4-week regimen and if continued could lead to further restoration towards normal autonomic balance (La Rovere et al., 1992). Epidemiological data indicate that compliance rates of exercise programs is significantly reduced the longer the training period (Trafton & Gordon, 2004). Since sedentary individuals, which were used in the present study, do not exercise on a regular basis it was important to set realistic goals as to what length of a program they would successfully adhere. We surmise that a longer length program would have significantly increased the mortality rate of the subject population in the present study. In regards to the training intensity, a vast number of measurements have been used. Despite the fact that the majority of studies report that they used a moderate intensity training protocol to determine the effects of exercise on HRV there are still variations that exist. For example, Davy et al., (1996) used 70% of the subject’s heart rate, Boutcher & Stein (1995) used 60% of the subject’s heart rate range obtained from baseline and peak exercise testing, Amano et al., (2001) determined the anaerobic threshold to calculate intensity, and Melanson & Freedson (2001) used 80% of heart rate reserve. The present study used 70% of the subject’s measured VO₂ peak value as the training intensity. Commonly, three days per week for 30 minutes per session is the frequency and duration used, which is consistent with the protocol used in this study.

Finally, the method used to assess HRV is also a critical component to note when discussing the lack of clarity in the research literature as it pertains to the effects of
exercise on HRV. Examining the time and frequency domains of HRV have been the most commonly used methods in measuring autonomic nervous tone. While, other methods such as the peak-to-trough method, explained by Boutcher & Stein (1995), have been used they have not proven to be as reliable and sensitive as the latter methods previously mentioned. The spectral analysis of HRV that allows specific frequency bands to be analyzed seems to be the method of choice in the research community. Using the frequency domain technique allows sympathetic and parasympathetic activity to be defined independently and in relation to one another. The LF variations typically occur at less than 0.1 Hz and correspond to sympathetic activity. The HF oscillations are demonstrated at a Hz level exceeding 0.1 and are exclusively under parasympathetic control (Binkley & Cody, 1992). It is noteworthy to highlight that specific frequency bands can slightly vary among studies. In a study conducted by Amano et al., (2001) the LF band was considered .03 to .15 Hz and the HF band was measured as .15 to .40 Hz. In this study the frequency bands were identified as .02 to .14 Hz and .14 to .90 Hz, which is consistent with the standards identified and used by Erhman (1996). Studies that use the spectral analysis of HRV generally have a better understanding regarding autonomic tone, can more readily identify if exercise was effective, and determine the modulating branch (sympathetic vs. parasympathetic) that is predominant in an individual’s nervous system.

The basis for the findings in the present study have been clearly supported in the research literature. First, as previously stated the length of the training intervention used in this study was 10-weeks long. It is possible that longer duration protocols are necessary to evoke changes in sedentary individuals. This possibility is likely plausible
because exercise training effects on cardiorespiratory fitness have a specified amount of time that an individual must train in order to elicit physiological benefits. The only difference maybe that the threshold that must be reached to see beneficial effects of exercise in HRV is much longer. Another explanation that may account for the inability of this study to demonstrate improvements in HRV is the study population used. In most studies, Caucasian middle-aged men were used as the target population. Some studies have shown that gender is an important determinant that should be considered when examining HRV responses (Tuluppo et al., 1998). The obvious hormonal and physiological differences between men and women often predispose or protect women from disease and dysfunctions (Safar & Smulyan, 2004). For example, after menopause, women go through several changes that increase their risk of developing cardiovascular disease. This is of great importance because low HRV has been reported to be an individual risk factor for the development of coronary artery disease (Davy et al., 1997).

Even more sparse than the use of women as the population of interest when examining the effects of exercise on HRV is the use of African American women. According to Arthur and associates (2004), individuals of color experience larger decreases in HRV than their control counterparts during psychological stress. African Americans have also been known to have an overactive sympathetic drive with little vagal modulation, which suggests a reduced HRV, which predisposes them to HTN. Since very few studies have examined HRV in African American women it is not known if their initial autonomic status plays a role in the effect of exercise. Similar to Boutcher & Stein’s study (1995) it is possible that the pre-exercise HRV levels of the subjects used in the present study were so diminished that significant changes as a result of training
could not be statistically seen. However, a trend that may be clinically important was observed in the present study. Prior to exercise the total HRV was $321.3 \pm 101 \text{ ms}^2 \text{ Hz}^{-1}$ and after the intervention the training group exhibited a total HRV of $329.8 \pm 67 \text{ ms}^2 \text{ Hz}^{-1}$. Moreover, HF activity slightly increased from $100.7 \pm 59 \text{ ms}^2 \text{ Hz}^{-1}$ (pre) to $131.9 \pm 76 \text{ ms}^2 \text{ Hz}^{-1}$ (post). This may be relevant because a HF activity is suggestive of improvements in parasympathetic (vagal) tone, which is associated with less resistance in the blood vessels, reduced heart rate, and lower blood pressure. This observation is important because it may be possible to see additional increases in total and HF HRV with a significantly longer duration exercise protocol.

Finally, it cannot be ignored that the subjects in this study were not only African American women, but they were also prehypertensive. Currently, to our knowledge no studies have examined the effects of exercise on HRV in prehypertensive women. Although there have been studies on the effects of exercise on HRV in women with elevated blood pressure levels (Davy et al., 1996) we are not certain if prehypertensive women follow the same physiological response pathways. Although a reduced HRV has been seen in elevated blood pressure states (Novak et al., 1994) it is not safe to assume that women who are predisposed to developing HTN automatically have a diminished HRV. A much larger and more heterogeneous prehypertensive population must be examined to determine if all prehypertensive women have reduced HRV. The women in this study may have displayed a reduced HRV simply because they were of a low physical fitness level. Goldsmith et al., (1997) found that $\text{VO}_2\text{max}$ was highly correlated with HF power indicating that physical activity is strongly associated with vagal modulation. It is also not possible to extrapolate from the findings of a normotensive
Clearly, the pathological pathways of normotensive and prehypertensive women are not the same in that the risk of developing HTN and associated cardiovascular abnormalities are twice as likely to occur in those with a prehypertensive status. Therefore, the complete demographic makeup of the target population must be addressed when examining the effects of exercise on HRV. Other factors that could possibly explain the improvement in HRV as a result of exercise in other studies are the lower levels of body weight and fat in the subjects in comparison with this study. Even in Amano et al.’s study, which purported that the subjects were obese their BMI only had to be 25 to be included in the study. Twenty-five was the minimal BMI criteria required to participate in this study with the maximum end approaching 35. Additionally, measures such as total blood volume that required more invasive techniques to assess were not accounted for in this study, but were noted in other studies in which exercise improved HRV (Davy et al., 1996).

Even though there were not any significant differences in HRV among the pre vs. post intervention comparisons in the exercise trained group there were beneficial effects of the exercise-training program observed. Improvements in both VO₂ peak and absolute workload were achieved. Cardiorespiratory fitness levels statistically increased (p < .05) from pre to post, 19.0 ± 3.9 and 23.0 ± 3.9 ml/kg/min⁻¹, respectively. The absolute amount of work that could be done by the exercise-trained group was significantly different with an initial workload of 116 ± 16 watts when compared to the post level of 133 ± 28 watts. The obvious benefits of an improved VO₂ peak are greatly needed in this research population. Yet the importance and implications of improved fitness on HRV is the finding that would be valuable, because several researchers believe that decreases in
HRV modulation may largely be a function of declining fitness rather than some of the other factors that have been cited (Goldsmith et al., 1997; Boutcher & Stein, 1995; Kenny, 1998). If this is true, increasing aerobic capacity may be the first step in improving HRV in African American women.

2.2.10 Remarks

To fully explore the effectiveness of aerobic exercise training on autonomic nervous system function it is essential to understand the underlying mechanisms of the population’s system that you are interested in exploring. It seems that effective approaches to improving HRV would address both the physiological and psychological states of their subjects, as well as exercise design factors. Often the implications of psychological variables are overlooked and discarded in studies that are more physiological in nature. In the present study to control for the possible extraneous variables of social support and perceived stress, two questionnaires were given to each subject before and after the 10-week intervention period. The Interpersonal Support List Evaluation (ISEL) developed by Cohen et al., (1985) was used to assess social support. The Perceived Stress Scale (PSS), which was also developed by Cohen and associates (1983), was administered to measure perceived stress. Prior to the treatment, control and training subjects were not significantly different on the ISEL (20.5 ± .49 vs. 21.1 ± .43, respectively) nor the PSS questionnaire (24.1 ± 2.2 vs. 21.5 ± 2.9). There were also observed differences within the exercise group from pre to post, which indicates that changes in stress and social support are not responsible for the improvements seen in workload and fitness. In comparison with other populations the participants in the
present study exhibited lower scores on the ISEL, suggesting that they felt less socially supported. In a study conducted by Mermelstein et al., (1983) a general smoking population and college students were administered the ISEL, results demonstrated that after a smoking cessation program social support scores were 34.4 and 38.8, respectively. These scores are significantly higher than the post score of 21.1 that is seen in the present study. On the other hand, PSS scores obtained from subjects in the present study were consistent with those found by other investigations. Farabaugh et al., (2004) found that older women had a score of 23.6 while female college students had a score of 23.8 on the PSS. In a community sample the average PSS score was 24 (Cohen, 1983). After the 10-week intervention, participants in the present study exhibited a PSS score of 21.5. The score range for the PSS scale is from 0 to 56. Higher scores are indicative of more appraised stress (Farabaugh et al., 2004). If unaccounted for, reductions in stress or enhancements in social support could alter the subject’s attitude toward exercise testing or training causing them to try harder or perform better during post-test, thereby inducing transient improvements in the variables of interest. Stress and social support are important indices to note because research has shown that higher amounts of stress and lower social support is associated with a greater incidence of hypertension (Bosworth et al., 2003; Vogele & Steptoe, 1992).

In spite of the findings of the present study and other studies that did not demonstrate an enhancing effect of exercise on HRV (Davy et al., 1996; Boutcher & Stein, 1995) several key concepts were uncovered. One, a dose response relationship may exist between exercise and improvements in HRV. In the present study it is possible that the training regimen was not long enough to initiate statistically significant changes in HRV.
More studies with differing training lengths must closely be monitored to determine what level of training is sufficient enough to cause promising alterations in HRV activity. Two, understudied populations such as African Americans and women must become the focus of HRV research in order to establish expectations about HRV responses to training. Three, a wide array of factors, that include but are not limited to weight, fitness level, and disease status, must be considered when developing an exercise training program. Finally, acknowledging that the alteration of one or more of the previously mentioned concepts may produce major differences in study outcomes is of the utmost importance. Learning what does not work in improving HRV among different populations is just as useful as learning about what has been productive. Both identification and elimination of possible sources, such as exercise that may improve HRV is one step closer to reducing cardiovascular complications and all-cause mortality associated with reduced autonomic tone.
Figure 2.2 Pre-exercise comparisons in Heart Rate Variability (HRV) between control and trained exercise groups. There were no significant differences in total, high frequency (HF), or low frequency (LF) HRV in the control or trained group prior to exercise. Values are expressed as means ± SE. (p < .05).
Figure 2.3 Post-exercise comparisons in Heart Rate Variability (HRV) between control and trained exercise groups. There were no significant differences in total, high frequency (HF), or low frequency (LF) HRV in the control or trained group after exercise training. Values are expressed as means ± SE. (p < .05).
Figure 2.4 Cardiorespiratory (VO₂ peak) fitness levels in control and trained group before and after the ten-week aerobic exercise training intervention. Before training there was no significant difference in (VO₂ peak) between the exercise and trained group. However, the exercise-trained group significantly improved their cardiorespiratory fitness after the ten-week protocol. There are no significant differences in pre vs. post values in the control group. All values are expressed as means ± SE (p < .05).*
Figure 2.5 Workload before and after the ten-week exercise training intervention in the control and exercise trained group. There were no significant differences before exercise between the control and exercise-trained group. A significant increase in absolute workload was seen in the exercise-trained group when comparing pre and post periods. There were no significant differences observed within the control group. All values are expressed as means ± SE. (p < .05)*.
CHAPTER 3

EXERCISE TRAINING EFFECTS ON PREHYPERTENSIVE BLOOD PRESSURE STATUS AND ASSOCIATED MECHANISTIC VARIABLES IN APPARENTLY HEALTHY AFRICAN AMERICAN WOMEN.

3.1 Introduction

The warning signs for the development of hypertension are scarce, but the implications of the disease are widespread. This phenomenon contributes to the “silent killer” cliché that is commonly associated with high blood pressure (HTN). Until recently, there weren’t any objective, scientifically tested, quantitative indicators used to identify individuals who were prone to develop HTN. Fortunately, in 2003 the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC) issued a report, which included a revised blood pressure classification scale with a new category, termed prehypertension, designed to identify persons with an increased risk for progression to HTN (Chobanian et al., 2003). Persons who are diagnosed with prehypertension must have systolic blood pressure between 120-139 mm Hg or a diastolic reading between 80-89 mm Hg on two or more occasions. Historically,
a systolic blood pressure measurement in the 120’s accompanied by a diastolic pressure in the 80’s was considered healthy and often overlooked. However, since the formulation of the JNC 7 report, some persons previously considered “normal” are now targeted as individuals with an elevated risk for progression to hypertension and require intervention to prevent the disease’s development (Chobanian et al., 2003).

Although intensive research has been done, little is known regarding the mechanistic cause(s) that perpetuate(s) the development of HTN and its precursor. The absence of known pathogenic explanations makes it difficult to prevent or reduce the increased prevalence of HTN and prehypertension that exists in African Americans, overweight, elderly, and low socio-economic status individuals, all who are disproportionately affected by the disease. Despite the fact that no single variable or collective mechanism has been definitively accepted as the primary model responsible for triggering the development of high blood pressure, there is a proposed hemodynamic dysfunction commonly identified among the likely culprits. For years an elevated total peripheral resistance (TPR), at rest and during exercise, has been thought to be a dominant disturbance in persons with HTN (Folkow et al., 1958; Lund-Johansen & Omvik, 1990). In a study conducted by Sihm and associates, (1995) 103 age and sex matched volunteers (83 hypertensives and 20 controls) underwent arterial biopsies to determine the amount of peripheral resistance in the arteries. Results revealed that indices of TPR, which included media thickness, cross-sectional area of the artery, and the media: lumen ratio, were substantially greater in the hypertensive subjects when compared to their control counterparts (Sihm et al., 1995). A significant correlation between TPR and blood pressure was also exposed during Sihm’s study (1995). Such
findings suggest that an impaired TPR may be linked to the initial cascade of neurohumoral, vascular, and structural events that take place, which leads to HTN.

Exercise has been employed to reduce high blood pressure and optimize associated cardiovascular responses. The exercise-generated reduction in blood pressure is often paralleled and accompanied by a decrease in TPR and an increase in cardiac output (Izdebska et al., 1998; Cleroux et al., 1992). One postulated antihypertensive effect of exercise is vasodilation (Pescatello et al., 2004). Stimulation of dilator nerves and the inhibition of constrictor substances and nerves occur when exercise relaxes the blood vessels by widening the diameter of the lumen space. This in turn reduces the amount of pressure or force that is generated against the vasculature structure as blood travels through the body. The effect of exercise is thought to be so strong that many researchers believe that as little as a single bout of exercise can cause considerable changes in physiological responses to reduce blood pressure. According to Izdebska, (1998) sixty minutes after one training session systolic blood pressure, diastolic blood pressure, and TPR were still significantly lower than pre-exercise values in the hypertensive subjects. Whereas, in the normotensive group SBP, DBP, and TPR only remained reduced for 20 minutes post exercise (Izdebska et al., 1998). Furthermore, a significant correlation was observed between DBP and TPR in hypertensive persons. The mechanisms of exercise that account for the noticeable reductions in blood pressure and vascular resistance are suggestive of a complex interaction between cardiovascular, structural, and hemodynamic variables.

Although the effects of exercise on blood pressure and associated cardiovascular variables have been consistently demonstrated, little is known regarding its effectiveness
in sedentary African American women with prehypertension. Therefore, the primary purpose of this investigation was to explore the effects of exercise training on blood pressure and TPR. To assess the outcome of exercise training on prehypertension and TPR a ten-week supervised aerobic exercise-training program was employed. Pathogenic possibilities were scrutinized to identify the origin or links in the etiologic cycle responsible in the development of high blood pressure.

3.2 Methods

3.2.1 Subject Population

Initial physiologic testing was performed on 28 sedentary African American women volunteers between the ages of 30-45 years. However, five women were not able to complete the study. Two women were not able to comply with the training guidelines set for the exercise-training group and therefore were removed from the study. One participant became seriously ill during the course of the study and was not able to return for post testing. Finally, two other women withdrew from the study with no explanation or notice as to why. All subjects were required to be an African American woman, 30-45 years of age, have a body mass index (BMI) between 25-35 kg/(m)^2^, non-smokers, sedentary, free from medication use, including birth control pills that would affect the blood pressure or cardiorespiratory fitness, absence of any known disease, and prehypertensive blood pressure status. Initial physical activity levels of each participant were assessed using the Baecke Physical Activity Questionnaire. To participant as a volunteer in the present study, subjects had to score less than eight on the Baecke Physical Activity Questionnaire which is consistent with the average score found by
(Sternfeld et al., 1999) who also investigated a relatively sedentary population. In addition, the physical activity score used for inclusionary criteria in this study was less than half of the maximal score that could be earned. The Baecke questionnaire score scale range from 4-20 (Sternfeld et al., 1999). The institutional review board at The Ohio State University approved this study. Written consent was obtained from each subjects prior to them participating in the study. Physical characteristics are presented in Table 3.1.

<table>
<thead>
<tr>
<th></th>
<th>Control (n=11)</th>
<th>Trained (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>36.9 ± 1.5</td>
<td>38.2 ± 1.5</td>
</tr>
<tr>
<td>Weight (kgs)</td>
<td>87.5 ± 10.4</td>
<td>86.9 ± 10.7</td>
</tr>
<tr>
<td>Height (in)</td>
<td>65.3 ± 1.2</td>
<td>65.6 ± .82</td>
</tr>
<tr>
<td>Body Mass Index (kg * m⁻²)</td>
<td>31.5 ± 1.1</td>
<td>30.83 ± 1.1</td>
</tr>
<tr>
<td>Systolic Blood Pressure (mm Hg)</td>
<td>120.3 ± 1.5</td>
<td>126.5 ± 2.6</td>
</tr>
<tr>
<td>Diastolic Blood Pressure (mm Hg)</td>
<td>84.2 ± .94</td>
<td>83.4 ± 1.3</td>
</tr>
<tr>
<td>Physical Activity Status</td>
<td>5.74 ± .30</td>
<td>5.88 ± .27</td>
</tr>
</tbody>
</table>

Table 3.1 Demographic characteristics of subjects (n = 23)

There were no significant differences between the control and exercise-trained group at the pre-exercise period. Values presented as mean ± SE.
3.2.2 Prehypertension Assessment

Resting blood pressure was measured twice in the exercise-testing laboratory and confirmed by the subject’s medical doctor, during the required physical for physician clearance, to establish prehypertension. A calibrated mercury filled sphygmomanometer (Gen-Med, American Diagnostic Group, 1993) blood pressure cuff appropriate in size, which has a cuff bladder encircling at least 80% of the arm, was used for each subject in the exercise-testing laboratory (Chobanian et al., 2003). Each potential subject was asked to sit in a chair with their dominant arm resting on a table at heart level for 5 minutes prior to the blood pressure measurement being taken. An experienced and certified American College of Sports Medicine, Exercise Specialist manually took all of the resting blood pressure measurements in the exercise-testing laboratory. If the subject’s blood pressure reading was between 120/80 and 139/89 mm Hg for either the SBP or DBP readings in at least 2 of the 3 measurements, then the subject was considered to have prehypertension (www.nhlbi.nih.gov).

3.2.3 Determination of Total Peripheral Resistance

A noninvasive indirect method of determining mixed venous gas tensions, known as CO₂ rebreathing, was used to assess cardiac output and associated variables including TPR following the method described by Jones & Campbell (1982). During the CO₂ procedure subjects rebreathed from a bag, attached to a two-way valve, containing gas with a PCO₂ content in excess of the mixed venous partial pressure of CO₂. This technique of assessing cardiac output is commonly known as the equilibration method. This assessment was done twice during one testing period, first during the resting phase
and again when the subject was exercising at 70% of the subject’s previously measured VO₂ peak. Subjects wore head gear with an attached mouth piece and nose clip to obtain cardiorespiratory values. An open circuit system employing the indirect calorimetry method (Parvo Medics True One Metabolic System package, OUSW Version 3.5, 2003) was used to measure the oxygen consumed and the carbon dioxide produced. During rest and exercise CO₂ rebreathing, breath by breath fluctuations in CO₂ percentage were analyzed using an Ametek CO₂ analyzer (Pittsburgh, PA) and were recorded using the Microscribe 4500 Strip Chart Recorder (Houston Instrument, Division of Bausch & Lomb, Austin, TX). The procedures followed for each subject were based on the methodology suggested by Collier et al., (1956) and modified by Jones & Campbell (1982). If the CO₂ procedure was unsuccessful by yielding an increasing plateau or a prolonged time to achieve equilibrium the CO₂ bag concentration or volume was adjusted and the procedure was repeated. After properly carrying out this procedure the data was analyzed and cardiac output was determined, also allowing stroke volume (SV), TPR, and mean arterial pressure (MAP) to be calculated using the following equations:

\[
\text{Stroke Volume} = \frac{\text{Cardiac Output}}{\text{Heart Rate}}
\]

\[
\text{Total Peripheral Resistance} = \frac{\text{Mean Arterial Pressure}}{\text{Cardiac Output}}
\]

\[
\text{Mean Arterial Pressure} = \left( \frac{\text{systolic pressure} - \frac{\text{diastolic pressure}}{3}}{} \right) + \text{diastolic pressure}.
\]

**3.2.4 Peak Oxygen Uptake Test (VO₂ peak)**

After completing the initial orientation session, eligible participants returned to the laboratory to perform a maximal graded exercise test on an electrically braked cycle
ergometer (Cybex, Division of Lumex, Ronkonkoma, LI, NY). Subjects were prepped for a standard 12-lead ECG (Quinton 4500 Stress Test Machine, Seattle, WA) wearing 10 electrodes and a heart watch monitor (Polar Electro Inc., Polar A1, Woodbury, NY). Prior to the test, headgear was placed on the subject’s head to support the weight of the mouthpiece attached to the open circuit system used to assess VO$_2$ peak. Indirect calorimetry using a Parvo Medics True One Metabolic System package (OUSW Version 3.5, 2003) measured the amount of oxygen consumed and carbon dioxide produced. Gas and flow meter calibrations were done before each test to ensure accurate readings (Parvo MMS-2400, Computer/DAC & Adapter PCL 711B). The test was terminated when subjects could not maintain the prescribed pedal rate, reached volitional fatigue, demonstrated a leveling off or decrease in VO$_2$ peak with increasing workload, or attained a RER exceeding 1.0. The cycling protocol began with a work rate of 25 watts, which increased an additional 25 watts every two minutes. Each subject was instructed to cycle at or above the required pedal cadence of 60rpm.

3.2.5 Submaximal Graded Exercise Test

To determine the 70% submaximal exercise workload each subject first performed the previously described maximal graded exercise test on a cycle ergometer. Following the maximal exercise test, the submaximal exercise intensity and the associated heart rate and VO$_2$ peak values expected, were calculated. Subjects were prepped with four chest electrodes to obtain an electrocardiogram (ECG) tracing throughout the test. The submaximal test began with a 3 minute seated rest period. At the end of rest blood pressure, heart rate, an ECG, and VO$_2$ measurements were obtained.
The carbon dioxide (CO₂) rebreathing procedure was conducted immediately following the resting phase. The exercise portion of the test started with the subject cycling at an initial intensity of 25 watts (W) for 2 minutes and then increased to a workload that corresponded to 70% of the subject’s VO₂ peak. Subjects cycled at this predetermined submaximal intensity for 3 minutes at the end of which HR, VO₂, and blood pressure were again recorded. Again, immediately following this period, the CO₂ procedure was repeated while the subject continued to cycle. The test concluded once an equilibrium plateau was achieved during the CO₂ rebreathing procedure following the resting and exercise phases of the test. Immediately following the submaximal exercise test each subject completed a two-minute active recovery period. This cool down period consisted of light cycling (<25 watts, rpm < 30) to prevent venous pooling in the subject’s lower extremities.

3.2.6 Exercise Blood Pressure and Heart Rate Measurement

Blood pressure was assessed during the peak and submaximal exercise tests using a Colin STBP-780 automated tracking system (Colin Medical Instruments, Plainfield, NJ). The Colin STBP-780 is a stand-alone noninvasive blood pressure stress test monitor that is capable of taking accurate and reliable blood pressures during exercise. The system is interfaced to work with a standard electrocardiogram using lead 2, complete with a TTL and analog input to simultaneously and continuously track exercise heart rate. These standard built-in interface connections allow for easy cardiovascular assessment. Auscultatory sounds are monitored using two microphones synchronized to the R wave to help reduce inaccurate readings caused by “noise” during constant movement (Colin
STBP-780 Manual). A regular adult size cuff was placed on the subject’s upper arm with a stabilizing wrist strap to measure blood pressure at the brachial artery. Subjects were hooked up to ten electrodes during the VO$_2$ peak test to obtain a 12-lead ECG and four electrodes were used during the submaximal exercise test to create the standard lead two view. Both set ups allowed exercise heart rates to be generated on the electrocardiograph and Colin STBP system, allowing a check and balance system to ensure accuracy.

### 3.2.7 Exercise Training Protocol

After completing the initial orientation session, subjects were randomly assigned to the exercise (N=13) or control (N=10) group for a 10 week period. During the intervention, control subjects were asked to maintain their normal dietary and physical activity lifestyles. Persons in the exercise group were required to come into the laboratory 3 times per week to exercise 30 minutes per session during the 10-week period. Exercise training was completed using the stationary cycle ergometer, air-dyne, and/or treadmill at the discretion of the participant. Exercise prescriptions were written for each participant using each mode of exercise available for training. The training intensity was initially set for 70% of the VO$_2$ peak and progressively increased by 5% every 2½ weeks (see Table 3.2). During each training session subjects wore a Polar heart watch and chest strap unit (Polar Electro Inc., Polar A1, Woodbury, NY) to ensure that the training intensity was being maintained within 5 beats of the specified target heart rate. Each exercise session concluded with a 2 minute cool down period, which consisted of a low level intensity of the exercise previously completed.
In order to meet the study’s exercise training compliance requirements, subjects in the training group were required to attend 75% of the 30 original exercise sessions prescribed. This rate of adherence was consistent with the average range set by most researchers of 50% to 80% of prescribed sessions attended versus those completed (Trafton & Gordon, 2004). Three exercise sessions were set per week over the 10-week period for each subject. Additional sessions were provided during the week and on the weekend to allow subjects to make-up any missed appointments.

<table>
<thead>
<tr>
<th>Subject ID #</th>
<th>Wt</th>
<th>Pre VO₂ peak</th>
<th>70% VO₂ peak</th>
<th>75% VO₂ peak</th>
<th>80% VO₂ peak</th>
<th>85% VO₂ peak</th>
</tr>
</thead>
<tbody>
<tr>
<td>000</td>
<td>180 lbs</td>
<td>20.1 mL.kg⁻¹.min⁻¹</td>
<td>14.07 mL.kg⁻¹.min⁻¹</td>
<td>15.075 mL.kg⁻¹.min⁻¹</td>
<td>16.08 mL.kg⁻¹.min⁻¹</td>
<td>17.08 mL.kg⁻¹.min⁻¹</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mode of Exercise</th>
<th>Intensity</th>
<th>Week of Training</th>
<th>Speed</th>
<th>Incline</th>
<th>Wattage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treadmill</td>
<td>70%</td>
<td>1-2 wks</td>
<td>2.5 mph</td>
<td>3.2%</td>
<td>53 watts</td>
</tr>
<tr>
<td>Bike</td>
<td>70%</td>
<td>1-2 wks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treadmill</td>
<td>75%</td>
<td>3-5 wks</td>
<td>3.0 mph</td>
<td>2.4%</td>
<td>61 watts</td>
</tr>
<tr>
<td>Bike</td>
<td>75%</td>
<td>3-5 wks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treadmill</td>
<td>80%</td>
<td>6-8 wks</td>
<td>3.4 mph</td>
<td>2.1%</td>
<td>68 watts</td>
</tr>
<tr>
<td>Bike</td>
<td>80%</td>
<td>6-8 wks</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treadmill</td>
<td>85%</td>
<td>9-10 wks</td>
<td>3.8 mph</td>
<td>1.8%</td>
<td>76 watts</td>
</tr>
<tr>
<td>Bike</td>
<td>85%</td>
<td>9-10 wks</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 3.2 Example Exercise Training Regimen**

Exercise prescriptions were derived using the American College of Sports Medicine (ACSM) regression equations outlined in the 6th Edition of the ACSM’s Guidelines for Exercise Testing and Prescription Handbook. Each subject received an individual exercise prescription based on her initially measured VO₂ peak value. Calculated intensities served only as a guideline to implement training.
3.2.8 Procedures Timeline

Each subject came into the laboratory following the same order of events (See Figure 3.1 for graphical view). During the first visit to the exercise physiology laboratory each subject received a thorough orientation and facility tour. The orientation session included an explanation regarding the purposes, procedures, possible risks/benefits, and participant expectations of the study. If the potential subject met all inclusionary criteria, an informed consent was administered and resting blood pressure (BP) was taken. Medical clearance forms for physicians were given to each subject to be returned, signed upon their next visit. The subjects’ second visit to the laboratory included filling out two psychological questionnaires that included the Interpersonal Evaluation List (ISEL) to measure social support and the Perceived Stress Scale (PSS) to measure mental stress, a second resting blood pressure assessment, and a VO₂ peak test. During that same week, subjects were asked to return to the laboratory on a different day to complete a submaximal exercise test. After successfully completing both pre-exercise tests, subjects were randomized into the control or exercise group. Over the next ten weeks, subjects were instructed to follow the prescribed guidelines for the intervention group in which they were randomly assigned. After the 10-week intervention each subject returned to the exercise-testing laboratory within one week to repeat the VO₂ peak and submaximal exercise test. Post questionnaires were also administered after the 10-week intervention to re-assess social support and perceived stress.
3.2.9 Statistical Analyses

Physiological data (TPR, SBP, DBP, & MAP) and psychosocial indices were analyzed as an absolute change between pre and post measures. To ensure the accuracy of the randomization process comparative analysis on pre-intervention descriptive (age, BMI, physical activity status) and dependant variables were conducted.

To assess the effectiveness of the 10-week aerobic exercise training protocol on various measures of blood pressure and TPR (pre vs. post intervention) within the control and exercise groups, a Wilcoxon signed-rank test was used. Another nonparametric test, Mann-Whitney U test (Wilcoxon rank-sum), was applied to identify any differences in absolute change between the control and trained groups in regard to the previous physiological dependant variables mentioned. Statistical significance was set a priori with a p value ≤ .05. Data was expressed as mean ± SE. All statistical procedures were performed using SPSS (Window version 13.0).
3.3.1 Results

Initial physical characteristics for the control and exercise trained group are listed in Table 3.1. There were no significant differences ($p > .05$) in age, height, weight, BMI, physical activity status, or resting blood pressure between the control and exercise trained group. In addition, we examined resting TPR, SBP, DBP, and MAP before and after the 10-week intervention to assess if the control and exercise trained group were statistically different in absolute change from pre to post. We found no significant difference between the two groups in TPR, DBP, and MAP. However, a significant difference was demonstrated in the absolute change in SBP when comparing the control and exercise trained group (control, 4.45 mmHg increase in SBP; trained, 4.75 mmHg decrease in SBP) ($p < .05$)(see Figure 3.2). In fact, eight individuals moved from the prehypertensive status to a normotensive classification after the 10-week aerobic exercise training intervention. Specifically, four subjects decreased their systolic reading from a prehypertensive status to a normotensive status and four participants exhibited reductions in their DBP from a prehypertensive to normotensive level. Furthermore, independent analyses were run for the control and exercise training group. As expected, the control group remained unchanged in SBP, DBP, and MAP before and after the 10-week intervention period (SBP pre $120.3 \pm 1.5$ mm Hg, SBP post $124.8 \pm 2.4$ mm Hg; DBP pre $84.2 \pm .95$ mm Hg, DBP post $82.7 \pm .905$ mm Hg; and MAP pre $97.2 \pm 2.7$ mm Hg, MAP post $95.2 \pm 2.71$ mm Hg) ($p < .05$). Surprisingly, there was a significant decrease in TPR in the control group ($p < .05$) (TPR pre $27.1 \pm 2.9$ mm Hg$L^{-1}min^{-1}$; TPR post $18.1 \pm 1.9$ mm Hg$L^{-1}min^{-1}$) (see Figure 3.3). However, in the present study our primary focus was to evaluate the effects of a 10-week aerobic exercise training protocol on TPR, SBP, DBP,
and MAP in the exercise trained group of individuals. Our findings demonstrated that pre vs. post SBP, DBP, & MAP remained unchanged in the exercise-trained group (SBP pre 126.5 ± 2.6 mm Hg, SBP post 121.8 ± 2.6 mm Hg; DBP pre 83.1 ± 1.3 mm Hg, DBP post 81.7 ± 2.1 mm Hg; MAP pre 97.1 ± 1.8 mm Hg, MAP post 92.8 ± 2.7 mm Hg) (p < .05). Similar to the control group there was also a significant reduction found in the TPR of the exercise-trained group (TPR pre 35.3 ± 5.1 mm Hg L min⁻¹, TPR post 26.9 ± 4.3 mm Hg L min⁻¹) (p < .05) (see Figure 3.3). In addition to the changes seen in TPR other physiological changes were noted in the exercise trained group. Exercise training statistically increased VO₂ peak in the exercise group (pre 19.0 ± 1.1 vs. post 23.0 ± 1.1 ml kg⁻¹ min⁻¹) (see Figure 3.4). However, no difference was found among the control group before and after the 10-week intervention period (pre 18.0 ± .98 vs. post 17.5 ± .88) (see Figure 3.4). The effectiveness of the exercise training protocol was also shown in the maximal workload achieved in the exercise group. Results revealed a significant increase in workload expressed in wattage among the exercise group (pre 116 ± 4.7 vs. post 133 ± 8.3 watts) (see Figure 3.5).

3.3.2 Discussion

The present study evaluated the effectiveness of aerobic exercise training on TPR, SBP, DBP, and MAP in apparently healthy African American women. The findings of the study only partially support the popular notion that exercise training can reduce blood pressure. Several researchers have demonstrated that a reduction in blood pressure occurs in individuals who engage in moderate intensity exercise (Pescatello et al., 2004; Hagberg et al., 2000; Fish et al., 1997). In the majority of studies that examine the effect
of exercise on blood pressure, both measurement levels (SBP and DBP) were found to be significantly different when examining pre vs. post intervention values. For instance, in a study conducted by Akinpelu (1990), twenty hypertensive African individuals (10 trained and 10 control) were examined before and after an exercise-training regimen using an intensity of 60-70 % of the heart rate reserve. The findings of this study demonstrated small (2-6 mm Hg change) but statistically significant reductions in both resting systolic and diastolic blood pressure after training (Akinpelu, 1990). The concern with this study and others whose findings are similar in nature, showing reductions in both SBP and DBP, may not be clinically relevant because the reductions often seen in one or both of the variables is so minuet. In yet another study, thirty-four hypertensive persons (30 men and 4 women) engaged in an exercise training protocol that required them to attend 3 supervised exercise sessions per week, training at an intensity that corresponds to 70% VO₂₅ max (Westheim et al., 1986). Findings reveal that resting SBP and DBP were significantly reduced, 152 ± -2 mm Hg to 149 ± -2 and 106 ± 102mm Hg, respectively (Westheim et al., 1986). Westheim and associates (1986) believe that the significant differences seen in systolic blood pressure from pre to post values are primarily due to reduced sympathetic activity. While, the reduction seen specifically in DBP is suggestive of a vasodilating effect of the exercise training. These mechanisms are very likely to occur as a result of exercise, however they lend no explanation as to why some studies only reveal a decrease in one component of blood pressure. It is noteworthy to point out that the subject’s ethnicity was not revealed in the previously discussed investigation (Westheim et al., 1986). Fish et al., (1997) further supported the sentiment that exercise is effective in reducing resting systolic as well as diastolic blood pressure. Using a step
treadmill intervention, subjects exercised 20-30 minutes, three times per week, for a 16-week program duration. Statistical analysis revealed a 10 mm Hg decrease in SBP and a 6 mm Hg decrease in DBP (142.2 ± 9.1 to 132.7 ± 8.2 mm Hg and 93 ± 4.9 to 87.4 ± 5.4 mm Hg; both statistically significant at p < .01). It is not clearly understood why some studies demonstrate a reduction in both components of blood pressure while other studies only demonstrate a decrease in one. Although the present study did not display a significant reduction in blood pressure in the exercise-trained group from pre to post-intervention, a reductive trend in the systolic component of blood pressure was observed (pre 126 ± 2.6 vs. post 121.8 ± 2.6 mm Hg). Moreover, a significant difference was demonstrated in the absolute change of SBP from pre to post-intervention periods when comparing the exercise and control groups (4.75 and –4.45 mm Hg, respectively) (p < .05). Also finding a statistically significant basis Tsai et al., (2002) & Vriz et al., (2002), revealed that exercise training elicited only a significant reduction in SBP. Tsai and associates (2002) tested twenty-three mildly hypertensive Taiwanese patients. After 12 weeks of aerobic exercise training, 3 times per week at a moderate intensity, it was found that SBP showed a dramatic decrease from 139.1 ± 11.4 to 121.1 ± 10.4 mm Hg (p < .05) with no equivocal drop in DBP. Tsai and associates (2002) suggested that increased sodium losses when sweating and a decreased tonic sympathetic nerve activity might be enhanced by exercise. The reductive relationship of aerobic exercise on SBP was also seen in an investigation conducted by Vriz et al., (2002). Although a specific exercise-training regimen was not implemented, the effect of blood pressure on physical activity status was examined. Participants were divided into 3 fitness levels that included non-exercisers, mild exercisers, and heavy exercises. One drawback to consider in this study
is that no women were classified as heavy exercisers (Vriz et al., 2002). The data showed that SBP was reduced in the mild and heavy exercising groups. No changes in ambulatory DBP were seen across the 3 groups of participants. The lower sympathetic activity seen in exercisers was thought to be the counter-balancing mechanism responsible for the reduction in SBP (Vriz et al., 2002). The partial lowering effect of exercise in many studies can be caused by a variety of factors. First, in several of the studies that demonstrated a reduction in both SBP and DBP the subject population was considered to have moderate to high levels of hypertension (Fish et al., 1997; Westheim et al., 1986; Akinpelu, 1990). Similar to the present study, which only approached significance, Tsai et al., (2002) and Vriz et al., (2002) used prehypertensive and mildly hypertensive persons as subjects, respectively. This is of great importance because exercise induced drops in blood pressure last longer (up to 22 h after training) and are the greatest in subjects who have the highest baseline or pre-exercise values (Pescatello et al., 2004). The use of persons with prehypertension may be one primary reason that the present study did not reduce any significant changes in SBP, DBP, or MAP within the exercise-trained group. In a prehypertensive state an individual’s blood pressure could still be as low as 120/80 mm Hg. To date, there is no evidence to determine if persons with lower levels of prehypertension are more like normotensive persons. Where as, high levels of prehypertension (139/89) are more associated with full scale HTN. Secondly, the intensity, duration, and frequency of exercise must be considered. The present study only used a 10-week protocol. Subjects in the present study exercised 3 days per week at an intensity equal to 70% of their measured VO2 peak. This may be problematic because other researchers have shown that it takes a minimum of 12 to 16 weeks to induce
changes in blood pressure (Gregoire et al., 1996; Amano et al., 2001). Since few studies have focused on African American women it is unknown if there is a different exercise dose response required for systolic and diastolic blood pressures changes to be seen in this group. Lastly, physical characteristics of the population of interest should be carefully examined, as they could potentially alter exercise responses. Age, ethnicity, and body weight, particularly have been shown to affect blood pressure levels before and after training (Hagberg et al., 2000). The apparent differences seen between studies that exhibit a decrease in both systolic and diastolic blood pressure and those that demonstrated only a reduction in SBP should be noted, because several scientists have indicated that reductions in SBP may be more relevant and clinically useful (Vriz et al., 2002). This is primarily due to the strong associations that SBP has to left ventricular mass, which is an independent risk factor for mortality and morbidity (Prisant et al., 1990). Higher systolic blood pressure measurements are thought to be a significant indicator for heart complications primarily in middle aged and older adults (Prisant et al., 1990). This premise that highlights the importance of SBP is promising, because despite the fact that blood pressure levels did not meet significance in the training group, SBP revealed a p value of.08 in the present study. This suggests that changes in SBP was approaching significance and may have been statistically relevant under different conditions. Additionally, relevant blood pressure changes were seen as evidenced by the significant difference between the trained and untrained individuals (absolute change form pre to post). It is possible that with a longer training protocol, an increased intensity, and/or a larger sample size, SBP results may have been statistically significant revealing a similar pattern to the one exhibited by Tsai et al., (2002) and Vriz et al.,
Furthermore, it has been suggested that the effect of an exercise training intervention shows greater significance in SBP (Vriz et al., 2002). This does not suggest that reductions in diastolic blood pressure are not important, instead it exemplifies that a decrease in DBP further reduces cardiovascular risk and improves the abnormalities associated with elevated diastolic pressures. In fact, DBP appears to be a strong predictor of heart attack and stroke in persons with essential or idiopathic HTN and younger populations. Disparities in determining which blood pressure measurement, systolic or diastolic, is more relevant in identifying and classifying individuals as hypertensive has been a long-standing concern. The diastolic number indicates the pressure in the major arteries between beats or when the heart is in the resting phase. The systolic measurement determines the force the blood places on the arterial walls during a contraction. Initially, the importance of knowing the diastolic pressure was at the forefront of treatment strategies. However, the Framingham Heart Study highlighted the significance of systolic blood pressure as possibly a more useful measure in diagnosing and managing HTN (Franklin et al., 2005). One source even ventured to say that the knowledge of SBP alone correctly classified blood pressure in 96% of the cases examined when compared to DBP when used alone only correctly classified 68% (www.Fitrex.com). An update of the JNC VI report was even made after research confirmed the importance of “identifying, treating, and controlling systolic HTN” (Kaplan, 1998). Therefore, it seems more important to identify which physiological mechanisms and variables exercise influences to reduce blood pressure. In examining the effects of exercise training on blood pressure it appears that maybe there are different mechanisms that come into play when assessing which blood pressure measurement will
be reduced. There may be exercise specific effects of a particular training protocol. Perhaps the population of interest, elderly, younger, prehypertensive, essential or secondary hypertensives, etc., require different program durations and intensities to see changes in both systolic and diastolic measures. Similar to pharmaceutical interventions, exercise treatments must be more specific and take into account the patient’s fitness level, age, medical status, gender, and ethnicity. The phrase “one-size fits all” is not true when designing exercise regimens to attenuate or treat HTN.

Although not a specific variable of interest in the present study, pulse pressure (PP) should also be assessed when discussing blood pressure and its implications on cardiovascular disease and complications (Kwagyan et al., 2005). Currently HTN classification guidelines only take into account systolic and diastolic levels independently without considering the relationship and combined importance of the two variables (Vaccarino et al., 2000). Pulse pressure is the numeric difference between systolic and diastolic measurements and is simply calculated by subtracting the diastolic value from the systolic value. Several studies have shown that PP represents arterial stiffness and total arterial compliance (de Simone et al., 1999 & Domanski et al., 2001). In fact, PP is thought to be an independent predictor of the incidence of heart disease, congestive heart failure, cardiovascular end points, and general mortality in certain populations (Vaccarino et al., 2000). It is thought that the greater the difference between the systolic and diastolic pressures, the stiffer and more damaged is the conduit arteries. Domanski and associates (2001) assessed the correlation between increased risk of death and elevated pulse pressures in a cohort of subjects from the first National Health and Nutrition Examination Survey population. Results revealed that a 10 mm Hg increase in PP was
associated with a 26% increase in cardiovascular death in persons between the ages of 25-45. A smaller increased risk of 10% in cardiac related deaths were associated with a 10 mm Hg elevation in PP among persons 46-77 years of age (Domanski et al, 2001). In yet another study carried out by Vaccarino et al., (2000), associations between coronary heart disease (CHD), congestive heart failure (CHF), mortality and PP were examined. Findings showed that a 10 mm Hg elevation in PP was associated with a 12% increased incidence of CHD, 14% increase in CHF, and a 6% increase in overall mortality (Vaccarino). In the present study there were no significant differences seen in PP between the control and exercise-trained groups. Exercise training also showed no statistically significant effect on PP from pre to post intervention periods in the trained group (pre PP 43.1 ± 4.7 mm Hg vs. post PP 40.1 ± 3.2 mm Hg). This finding is consistent with the premise outlined in the literature that suggests that changes in PP are only a result of changes in systolic and/or diastolic pressures (de Simone et al., 1999 & Kwagyan et al., 2005). Therefore, since no changes in SBP nor DBP were evident in the present study alterations in PP should not have been expected.

Exercise has conclusively been thought to be an effective non-pharmacological therapy in attenuating HTN. However, the mechanism(s) by which this occurs is not as established. Several variables have been identified as likely pathological explanations responsible for exercise’s reductive effect. These proposed mechanisms include vascular, structural and neurohormonal adaptations that are induced as a result of engaging in planned physical activity (Pescatello et al., 2004). However, prior to such activity many populations, such as African Americans, have underlying abnormalities in the vascular, structural, and neurohormonal systems that predispose them to disease developments.
Consistent emerging data suggests that improved TPR plays a major role in the protective effect of exercise. Total peripheral resistance is linked to several other physiological factors. Thus, abnormalities in TPR can directly lead to dysfunctions in a wide array of variables. For instance, Sihm and associates (1995) examined left ventricular mass, and resistance in the peripheral arteries in 83 hypertensive and 20 control (normotensive) individuals. The media: lumen ratio, media thickness, and cross-sectional area of peripheral arteries were assessed in each group. Results showed that the media thickness of hypertensive participants was significantly higher than their control counterparts, 21.0 ± 4.2 and 16.2 ± 2.6 microns, respectively. Additionally, the media: lumen ratio was also statistically greater in persons with hypertension than in normotensive persons (10.2 ± 2.6% vs. 7.9 ± 2.0%, respectively). This difference between hypertensive and normotensive subjects was even seen in arteries with similar internal diameters and cross-sectional areas (Sihm et al., 1995). Media cross-sectional area was not greater in hypertensives subjects in comparisons to normotensive subjects in Sihm et al’s., (1995) study. However, other studies have identified a smaller lumen size in persons with HTN (Aalkjaer et al. 1987 & 1989; Izzard et al., 1991). The structural changes in the peripheral vasculature of hypertensives undoubtedly increases the pressure in these vessels as blood travels through the system, thereby accounting for the elevations in blood pressure. Sihm et al., (1995) also did correlational analyses showing a significant relationship between media: lumen ratio and blood pressure. Aalkjaer et al., (1987) and Izzard et al., (1991) also displayed significant increases in the same measures of TPR explored in Sihm et al’s., (1995) study. Although a cause and effect relationship between TPR and blood pressure have been consistently seen in the research literature, it is
unclear weather morphological changes in blood vessels cause elevated blood pressure levels or if HTN induces the morphological changes in the vessels. In addition, changes in TPR can also occur independent of changes in local blood pressure. This is the phenomenon that was observed in the present study, which revealed a significant decrease in TPR from pre to post-intervention values (27.1 ± 2.85 mm Hg L min⁻¹ vs. 18.1 ± 1.94 mm Hg L min⁻¹, respectively) with no corresponding significance found in SBP (pre 123.6 ± 1.7 mm Hg vs. post 123.3 ± 1.7 mm Hg) (Sihm et al., 1995).

The commonly seen beneficial effects of exercise training in reducing TPR, MAP, SBP, and DBP is likely a multi-factorial approach. Regular physical activity has been commonly used as a nonpharmacological intervention because of its ability to reduce TPR, decrease catecholamine levels, improve insulin sensitivity, enhance lipid profiles, and increase the release and circulation of powerful vasodilators, while inhibiting the activity of vasoconstrictors (Pescatello et al., 2004). The exercise dose response was displayed in an investigation done by Iwasaki et al., (2003) on several of the previously mentioned variables. Eleven young men and women engaged in a progressive aerobic exercise program, for 12 months, that required them to exercise 3 to 4 times per week for 30 –45 minutes per session (Iwasaki et al., 2003). Steady-state hemodynamics were measured at the beginning and again at the end of the 1-year period. According to Iwasaki et al., (2003) exercise can induce significant effects on several dynamic cardiovascular variables. Findings discovered that three months of exercise evoked significant reductions in SBP (baseline, 126 ± 4 vs. 3 mm Hg month, 116 ± 3mm Hg) and DBP (baseline, 74 ± 3 mm Hg vs. 3 month, 62 ± 2 mm Hg). Significant changes were also seen in cardiac output and TPR. The associated reduction displayed in TPR was
thought to be the mechanism responsible for the exercise-induced decrease in blood pressure (Iwasaki et al., 2003). Another postulated explanation thought to account for the decrease in blood pressure is an increase in the production and blood circulating levels of vasoactive factors. The later postulated justification for the reduction in blood pressure seems more feasible because alterations in vessel structure, which may change TPR usually occur after several sustained months of exercise. This would mean that reductions in blood pressure would not only have been seen at 3 and 6 months, but also at 9 and 12 month assessments. Typically, transient changes in TPR do not have a lasting impact on blood pressure and would not still be visible at 6 months. However, the observations of Iwasaki et al., (2003) indicate that the combined effect of a reduced TPR and vasodilation in the blood vessels account for the marked reduction in blood pressure. This finding seems logical in a longitudinal study since a morphological increase in vessel size would clearly reduce the amount of pressure exerted on peripheral vessels. With that stated, caution must be taken when identifying closely linked variables as distinct mechanisms that contribute to blood pressure reductions. It appears that the prolonged training regimen administered in Iwasaki et al’s., (2003) study, did not warrant further reductions in cardiovascular variables. Therefore, training regimens lasting longer than 3 months did not reveal any additional significant improvements in the variables studied. Decreases in blood pressure in the post-exercise period were also seen in a study conducted by Izdebska et al., (1998). A single bout of exercise was assessed to determine if short duration physical activity would have any effect on physiological variables. Seventeen young adults (9 normotensive & 8 hypertensive) volunteered to participate as subjects. Each participant performed a progressive cycle ergometer test up
to a level equivalent to 55% of the subject’s heart rate reserve. Outcomes reported a sustained lower SBP, DBP, and TPR from baseline values one whole hour after only a single bout of exercise (Izdebska et al., 1998). It seems as if different cardiovascular variables may require varying lengths of exposure to exercise training to exhibit effects. For instance, the changes in TPR that were seen in Izdebska et al.’s., (1998) were unlikely caused by morphological changes in vessel structure. Instead, a temporary vasodilatory response probably accounted for this observation. Finally, as mentioned in several studies it is highly likely that a reduced or diminished sympathetic activity is responsible for exercises’ reductive effect on blood pressure (Tsai et al., 2002; Westheim et al., 1986). Exercise is particularly promising in the present study because the JNC, World Health Organization scientific report have stated that non-drug therapy, such as exercise, can prevent several cases of pre-hypertension from progressing to stage 1 and 2 HTN (Sainai, 2003).

In the present study there are a number of constituents that may have attributed to the lack of significance in SBP, DBP and MAP. In addition, to those previously mentioned it must be recognized that African Americans exhibit greater levels of resting vascular resistance when compared to their Caucasian counterparts (Sherwood et al., 2003). Based on the premise explained by Pescatello et al., (2004), it is possible that because the TPR was initially greater in the participants examined in this study compared to those in other studies an observed effect was seen. Pescatello and associates (2004) reason that the greatest improvements in cardiovascular variables are seen in subjects who have the highest baseline or pre-exercise values. Dunn et al., (1983) have stated that, “epidemiologic data point to racial differences in cardiac adaptations to
hypertension”. Not only are racial differences of concern in this population, but gender also plays a role. Because few studies have specifically examined a female cohort it is difficult to surmise what exercise interventions will be most effective in reducing blood pressure (Anastos et al., 1991).

Several other factors such as body weight, dietary habits and psychological well-being are also important to note in the participants of the present study. Obesity has been associated with elevated levels of blood pressure and is considered a risk factor for associated cardiovascular disease (Kaufman et al., 1996). The women in this study had a BMI between 25-35 (kg m$^{-2}$), which is considered overweight and obese class I. Subjects were weighed before and after the 10-week intervention and no significant differences were found. Salt intake is another variable that can also affect human hypertension, especially in blacks (Wright et al., 2003). Some researchers believe that and increased salt sensitivity is a major component in hypertension development among African Americans (Eisner, 1990). Knowing this, the participants in the present study completed a four-day food recall before and after the training intervention to control the possible effects that dietary salt intake might have on the study’s outcome. Analysis of food records did not demonstrate any major differences in eating patterns among the subjects. Finally, psychological stress and associated feelings of support or the lack thereof have been cited as major variables in the pathogenesis of HTN (Fauvel et al., 2003). Blood pressure response to mental stress is even thought to have a predictive effect on future blood pressure status (Carroll et al., 2003). Due to the length of the present study it was possible that the subject’s mental stress and social support could change causing an alteration in blood pressure. Two psychological questionnaires were administered at pre
and post training periods. The Interpersonal Support List Evaluation (ISEL) developed by Cohen (1985) was used to assess social support. The Perceived Stress Scale (PSS), which was also developed by Cohen and associates (1983) was administered to measure perceived stress. Prior to the treatment, control and training subjects were not significantly different on the ISEL (20.5 ± .49 vs. 21.1 ± .43, respectively) nor the PSS questionnaire (24.1 ± 2.2 vs. 21.5 ± 2.9). There were also no observed differences within the exercise group from pre to post, which indicates that changes in stress and social support are not responsible for the improvements seen in total peripheral resistance.

Even though there were not any significant differences in SBP, DBP, & MAP among the pre vs. post intervention comparisons in the exercise group, there were beneficial effects of the exercise training program observed. Possibly the most important finding of this study is the transition of eight exercise-trained subjects from a prehypertensive to a normotensive blood pressure status in one or both of the blood pressure measurements. Clinically a shift from a prehypertensive status to a normotensive category in systolic and/or diastolic blood pressure is significant and relevant because it suggests that exercise training is an effective first line of defense in fighting high blood pressure progression and severity. Improvements in both VO$_2$ peak and absolute workload were achieved. Cardiorespiratory fitness levels statistically increased (p < .05) from pre to post, 19.0 ± 3.9 and 23.0 ± 3.9 ml·kg·min$^{-1}$, respectively. The absolute amount of work that could be done by the exercise-trained group was significantly different with an initial workload of 116 ± 16 watts when compared to the post level of 133 ± 28 watts. The obvious benefits of an improved VO$_2$ peak are greatly needed in this research population.
To the surprise of the researchers in the present investigation TPR was also significantly reduced in the control group from pre to post-intervention (pre $27.1 \pm 2.9$ mm Hg L min$^{-1}$ vs. post $18.1 \pm 1.9$ mm Hg L min$^{-1}$). Our concern could be that some, if not all, of the women in the control group engaged in physical activity during the course of the study. However, we called each participant every 2-½ weeks during the 10-week training period and asked a list of standard questions to ensure that they had not started exercising. Results in VO$_2$ peak and absolute workload in watts also revealed that the control subjects did not exercise because there were no significant improvements in either one of these variables, eliminating the possibility that they were physically active during the study. In fact, the examination of exact numbers revealed that VO$_2$ peak and workload actually decreased in the control group (pre VO$_2$ peak $18.4 \pm .98$ mL kg$^{-1}$ min$^{-1}$ vs. post VO$_2$ peak $17.5 \pm .88$ mL kg$^{-1}$ min$^{-1}$; pre workload $106.8 \pm 4.0$ watts vs. post workload $95.4 \pm 7.6$ watts). Although we cannot be sure what actually accounted for the decrease in TPR among control subjects we can hypothesize to the likely causes. One, the control subjects may have been more anxious on the pre-test than they were on the post-test. In turn subjects could have experienced familiarization to the testing conditions and environment, allowing them to be more comfortable and confident in performing the post-test, decreasing anxiety and resulting in a decrease in TPR. This is feasible because all participants were sedentary and many had never used a cycle ergometer prior to participating in the study. A reduction in anxiety could have relaxed the entire vascular system by increasing the lumen space of the vessels via increased vasodilator activity. Also there may be some other unknown variable(s) that is not related to training status that is inherently present in all prehypertensive African Americans. This unknown
variable may have an effect causing a significant reduction in TPR without a concomitant
decrease in blood pressure. For example, some studies suggest that opiate and other
chemical substances may be up-regulated in obese and hypertensive individuals (Pugsley,
2002). Opiate is a chemical substance (hormone) that acts by dulling the senses, which
causes relaxation and deadens physiological structures in the body, such as vessels. An
increase in opiate activity could relax the blood vessels, allowing blood flow to occur
more smoothly. Since all the participants in this study are classified as obese and
prehypertensive this may be a feasible explanation as to why both the control and
exercise-trained subjects revealed a decrease in TPR. In addition, a decrease in alpha-
adrenergic activity may be present in prehypertensive individuals to act as a protective
antihypertensive mechanism that elicits reductions in TPR (Velliguette & Ernsberger,
2003). Alpha-adrenergic receptors are responsible for inducing brief inotropic activity,
which could affect the force of blood flow through the vessels. It is possible that
although the subjects in the present study have not exhibited high blood pressure at rest,
other pathologic changes associated with HTN have already occurred, like heightened
alpha-adrenergic activity. However, additional research in this population must be
done to speak to these possible explanations and to identify other variables that may be
responsible regarding the decrease seen in TPR among non-exercising individuals.

3.3.3 Remarks

In the present study exercise improved TPR, cardiorespiratory fitness, and
workload in the exercise trained group. Although no changes in SBP, DBP, and MAP
were observed in either the exercise trained or control group (pre vs. post) the results of this investigation still prove to be useful. It may be that a moderate intensity 10-week training protocol is not sufficient enough to act as a stimulus to induce changes in blood pressure in prehypertensive African American women. Knowing this can guide future research in developing exercise program designs and may be insightful in determining what type of exercise protocol will likely induce changes in blood pressure and associated variables. Since the research literature is sparse on prehypertensive individuals, especially those who are African American women, any addition to this body of knowledge is important. Although the present study did not statistically alter the prehypertensive status in the exercise-trained group this does not necessarily indicate that women who engaged in the exercise-training group are still twice as likely to develop high blood pressure. The clinical reductive trend in SBP that was observed in the training may reduce their risk of developing HTN. It is also feasible that the exercise-induced reductions in blood pressure converted those individuals with lower prehypertensive blood pressure levels to mimic normotensive patterns in physiological responses. The 5 mm Hg reduction in SBP seen in the exercise-training group may be greater if the exercise-training regimen was longer. However, by simply becoming physically active, it is expected that the exercise group would have decreased their risk of other chronic diseases like cardiovascular heart disease. As more studies are done on the population used in the current study and on other minority groups, a clearer picture will be developed regarding the implementation of exercise therapy to continually reduce the atrocious effects of HTN on African Americans and the United States population as a whole.
Figure 3.2 Absolute changes in systolic blood pressure (SBP) between the control and exercise group before and after the ten-week intervention. There was a statistically significant decrease in SBP in the exercise group in pre vs. post intervention absolute values. There was not a statistically significant change in SBP in the control group. Pre and post SBP values were not significantly different between the control and exercise-trained groups. All values are expressed as means (p < .05)*.
Figure 3.3 Changes in total peripheral resistance (TPR) in the control and exercise trained group before and after the ten-week intervention. There were no significant differences between the control and exercise group before or after the training period. However, there were significant reductions in TPR within both the control and exercise groups when comparing pre to post values. All values are expressed as means ± SE (p < .05)*.
Figure 3.4 Cardiorespiratory (VO$_2$ peak) fitness levels in control and trained group before and after the ten-week aerobic exercise training intervention. Before training there was no significant difference in (VO$_2$ peak) between the control and exercise-trained group. However, the exercise-trained group significantly improved their cardiorespiratory fitness after the ten-week protocol. There are no significant differences in pre vs. post values in the control group. All values are expressed as means ± SE (p < .05)*.
Figure 3.5 Workload before and after the ten-week exercise training intervention in the control and exercise trained group. There were no significant differences before exercise between the control and exercise-trained group. A significant increase in absolute workload was seen in the exercise-trained group when comparing pre and post periods. There were no significant differences observed within the control group. All values are expressed as means ± SE. (p < .05)*.
CHAPTER 4

CONCLUSION

4.1 Review

Exercise training has consistently been shown, in the research literature, to be an effective non-pharmacological alternative to prevent and treat hypertension (HTN). Conversely, the underlying mechanism that accounts for the reductive and possibly preventive effects induced by exercise has not been clearly established. Moreover, few studies have examined the effect of exercise training on HTN in African American women, and to our knowledge no study exists that has observed prehypertensive individuals. Therefore, the present study investigated the effects of a 10-week aerobic exercise-training regimen on total peripheral resistance (TPR), heart rate variability (HRV), and prehypertensive blood pressure status in apparently healthy African American women. Listed below is a synoptic review of the present study’s findings:

- Moderate intensity exercise training did not improve HRV, systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), or pulse pressure (PP) in apparently healthy prehypertensive African American
women when comparing pre to post periods within both the control and exercise-trained group.

- Exercise training did induce statistical changes in TPR, cardiorespiratory fitness and absolute workload among individuals in the exercise-trained group (TPR pre 35.3 ± 5.1, TPR post 26.9 ± 4.3 mm Hg L min⁻¹; pre VO₂peak 19.0 ± 1.1 vs. post VO₂peak 23.0 ± 1.1 ml kg⁻¹ min⁻¹; pre 116 ± 4.7 vs. post 133 ± 8.3 watts) (p < .05).

- A significant difference was also seen in the absolute change of SBP from pre to post-intervention when comparing the exercise and control groups (4.75 and – 4.45 mm Hg, respectively) (p < .05).

- Unexpectantly, the control group also revealed a significant decrease in TPR before and after exercise training (TPR pre 27.1 ± 2.9; TPR post 18.1 ± 1.9) (p < .05).

- A clinical reductive trend in SBP was seen in the exercise-trained group. This trend was demonstrated in the observation that SBP was reduced from 126.5 ± 2.6 to 121.8 ± 2.6 mm Hg, while DBP only showed a slight change in absolute values from 83.1 ± 1.3 to 81.7 ± 2.1 mm Hg.
• Eight participants were converted from a prehypertensive status to a normotensive classification. Specifically, four persons demonstrated a decrease in systolic blood pressure and four individuals exhibited reductions in diastolic blood pressure.

• There was no significant difference in PP between the control and exercise trained group after the 10-week intervention (control PP 42.1 ± 4.1 vs. trained PP 40.1 ± 3.2).

• As expected, the control group remained unchanged in total, LF, and HF HRV activity before and after the 10-week intervention period (total pre 366.7 ± 49.7 ms² Hz⁻¹, total post 310.2 ± 23.4 ms² Hz⁻¹; LF pre 207 ± 39.1 ms² Hz⁻¹, LF post 188.9 ± 27.8 ms² Hz⁻¹; HF pre 158.8 ± 27.2 ms² Hz⁻¹, HF post 119.7 ± 21.9 ms² Hz⁻¹).

• Our findings demonstrated that pre vs. post total, LF, and HF HRV also remained unchanged in the exercise-trained group (total pre 321.3 ± 29.2 ms² Hz⁻¹, total post 329.8 ± 19.4 ms² Hz⁻¹; LF pre 218.1 ± 23.0 ms² Hz⁻¹, LF post 196.2 ± 21.6 ms² Hz⁻¹; HF pre 100.7 ± 17.0 ms² Hz⁻¹, HF post 131.9 ± 22.0 ms² Hz⁻¹).

• Although no statistical significance was found in HRV in the present study a reductive trend that may be important clinically was observed in the exercise group. Prior to exercise the total HRV was 321.3 ± 101 ms² Hz⁻¹ and after the
intervention the training group exhibited a total HRV of $329.8 \pm 67$ ms$^2$ Hz$^{-1}$. Moreover, HF activity slightly increased from $100.7 \pm 59$ ms$^2$ Hz$^{-1}$ (pre) to $131.9 \pm 76$ ms$^2$ Hz$^{-1}$ (post).

- A reduction in TPR and an increase in HRV appear to be independent events that do not necessarily occur simultaneously in prehypertensive African American women.

- Social support measured by the Interpersonal Support List Evaluation (ISEL) questionnaire and perceived stress assessed by the Perceived Stress Scale (PSS) were not statistically different in the control or trained group during pre and post-intervention periods ($20.5 \pm .49$ vs. $21.1 \pm .43$ and $24.1 \pm 2.2$ vs. $21.5 \pm 2.9$, respectively) ($p < .05$).

- Dietary intake was also the same in both groups before and after the 10-week training period. Changes in dietary patterns were measured using a 4-day food recall.

The present investigation tested several null hypotheses. First, aerobic exercise training did not have a significant effect on HRV. Therefore, we support the hypothesis that aerobic exercise training would have no effect on HRV. In contrast we do not support our second hypothesis, which postulated that aerobic exercise training would have no effect on total peripheral resistance. Thirdly, exercise training was not effective
in reducing prehypertensive status in the present study, thus we accept the hypothesis, which stated that aerobic exercise training would have no effect on prehypertensive status. Both secondary hypothesis regarding psychological factors (social support & perceived stress) were confirmed.

The present investigation suggests that a 10-week aerobic exercise training protocol is sufficient to elicit changes in TPR, but was not effective in reducing associated blood pressure levels. However, significant improvements in cardiorespiratory fitness support the well-documented benefits of exercise training. Since little to no information has been gathered regarding the effect of exercise on cardiac related physiological variables in prehypertensive African American women, the present study is a valuable investigation. As more studies are done on this population a clearer picture will be developed regarding the implementation of exercise therapy to continually reduce the number of not only African Americans, but also Americans as a whole who are affected by HTN.

4.2 Study Limitations

Generalizations from the present investigation to normotensive, hypertensive, and other prehypertensive populations should be done with caution, primarily because of the unique dynamics of the sample used in this study. The women who volunteered in this study had a significantly reduced cardiorespiratory fitness level (mean = 18.7 ml·kg·min⁻¹). Generally, a VO₂ peak value this low is associated with cardiovascular complications and predisposes individuals to other chronic diseases. It is also important to note that during the submaximal exercise test only the relative workload was used to
assess TPR. To better understand the changes seen in TPR, absolute workload should have also been measured; this would have allowed the researchers to determine if vascular resistance would have improved in the exercise-training group at the same given workload. This measure may have also provided more insight about the significant change seen in TPR among the control group. Although an absolute workload was not used during the submaximal exercise test, using a relative workload provided meaningful findings in the exercise-trained. Despite the fact that the exercise intensity (workload in watts) increased from the pre to post-test period in the exercise-trained group there was still a significant reduction in TPR. This suggests that even at higher workloads the blood vascular maintained its relaxed state, which suggests that if absolute workloads were used a greater decrease in TPR may have been seen. The small sample size in this study may have also limited the ability of statistical relevance to be demonstrated in variables that approached significance (SBP). Additionally, although the present study accounted for changes in body weight no measure of percent body fat was taken throughout the course of the study. Therefore, the results of this study cannot be extrapolated to other populations, but instead are only relevant to sedentary prehypertensive African American women between 30-45 years of age with an increased BMI, non-smoking status, and who are free from medication use.


