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The Effects of Whole-Body Vibration on Cardiovascular and Autonomic Function in Overweight-Obese Premenopausal Women

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THE EFFECTS OF WHOLE-BODY VIBRATION ON CARDIOVASCULAR AND AUTONOMIC FUNCTION IN OVERWEIGHT-OBESE PREMENOPAUSAL WOMEN

By

RYAN GIL

A Thesis submitted to the
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Dolefully, human life succumbs to the nature of time as it is ultimately taken away. During my race against this outcome, the art of scientific research has enlightened me to an idea that all health issues are actually corrigible conundrums. Those who closely encompass my life have driven this insight into motion by encouraging my journey through education. Therefore, to those of my family that I may delay this chronological misfortune we face together, I dedicate this project to you.

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LIST OF ABBREVIATIONS

1-RM:	1-repetition maximum
AE:	Aerobic exercise
AIx:	Augmentation Index
AIx@75:	Augmentation index normalized for a heart rate of 75 beats•min^{-1}
baPWV:	Brachial-ankle pulse wave velocity
BMI:	Body mass index
BP:	Blood pressure
BRS:	Baroreflex sensitivity
cfPWV:	Carotid femoral pulse wave velocity
CVD:	Cardiovascular disease
DBP:	Diastolic blood pressure
DXA:	Dual-energy X-ray absorptiometry
eNOS:	Endothelial nitric oxide synthase
ECG:	Electrocardiogram
ET-1:	Endothelin-1
faPWV:	Femoral-ankle pulse wave velocity
FMD:	Flow-mediated dilation
HF:	High frequency
HR:	Heart rate
HRV:	Heart rate variability
RE:	Resistance exercise
LF:	Low frequency
LnLF:	Logarithmic low frequency
LnHF:	Logarithmic high frequency
MAP:	Mean arterial pressure
NO:	Nitric oxide
P1:	First systolic wave
P2:	Second systolic peak
POST-CON:	After the control period measurement
POST-WBVT:	Before the intervention period measurement
PP:	Pulse pressure
PRE-WBVT:	Before the intervention period measurement
PRE-CON:	Before the control period measurement
PWV:	Pulse wave velocity
SBP:	Systolic blood pressure
TR:	Time of reflected wave
TVR:	Tonic vibration reflex
WBV:	Whole-body vibration
WBVT:	Whole-body vibration training

ABSTRACT

Background: Being overweight or obese is associated with increased sympathetic activity and decreased vascular function which increases the cardiovascular risk. Current research has shown that conventional aerobic, resistance, and isometric exercise training has the capacity to elicit improvements in autonomic and cardiovascular function. Recently, exercise with whole-body vibration (WBV) has become of high interest to researchers in the field of exercise physiology due to its beneficial effects on bone mineral density, muscle mass, and muscle strength. **Purpose:** The purpose of this study was to evaluate the acute and chronic effects of WBV on arterial and cardiac autonomic function in overweight and obese (OV/OB; Body mass index 28.3 ± 0.9 kg/m^2) women. **Methods**: In a cross over design, eight young (21 ± 2 yr) OV/OB women were randomly assigned to either WBV training (WBVT) 3 days per week or 6 weeks of no-exercise (CON). After their assigned treatment period, the subjects underwent a 4 week washout period before beginning their next 6 week treatment period (either WBVT or CON). At the beginning and end of each period, brachial blood pressure (BP), heart rate variability (HRV), augmentation index (AIx), and pulse wave velocity (PWV) were obtained at rest and 3 min after (PE3) a 4 min bout of static squat with WBV at 30 Hz frequency and 1.0 mm amplitude. Maximal strength (1-RM) was assessed for the leg extension exercise. Measurements were repeated 6 week after WBVT and CON. **Results:** There were significant decreases ($p < 0.05$) in resting systolic BP (SBP, 8 ± 5 mmHg) and diastolic BP (4 ± 2 mmHg) after WBVT compared with CON ($p < 0.05$). There were significant decreases ($p < 0.05$) in heart rate (7 ± 4 bpm), SBP (5 ± 4 mmHg), and mean arterial pressure (7 ± 3 mmHg) at PE3 after WBVT compared with CON ($p < 0.05$). There was a significant increase ($p < 0.01$) in 1-RM (12 kg) after WBVT, but not after CON. There were no changes in HRV, AIx, and PWV after both WBVT and CON. **Conclusion:** These preliminary data indicate that 6 weeks of WBVT elicits reductions in resting and post-exercise BP in overweight-obese women. In addition, lower post-exercise HR suggests that cardiovagal regulation may improve after WBVT. Future research is needed to further evaluate this exercise mode as a potential adjunct treatment for cardiovascular diseases.

SPECIFIC AIMS

1. To determine the effects of 6 weeks of WBVT compared to control on cardiovascular function. This aim examined the working hypothesis that WBVT would result in a decrease in arterial stiffness, augmentation index, and blood pressure. This aim was tested by non-invasive measures of arterial stiffness using pulse wave velocity (PWV) of several arterial segments (carotid-femoral, femoral-ankle, and brachial-ankle PWV), pulse wave analysis (aortic BP and augmentation index) using applanation tonometry of the radial artery, and brachial BP using spygmomenometry.

2. To determine the effects of 6 weeks of WBVT compared to control on cardiac autonomic function, the main control mechanisms of heart rate. This aim examined the working hypothesis that WBVT would result in lower sympathetic activity and higher parasympathetic activity using power spectral analysis of heart rate variability (HRV).

3. To determine the effects of 6 weeks of WBVT compared to control on body composition and muscle strength. This aim examined the working hypothesis is that WBVT would result in improved lean body tissue and in turn improved muscle strength. This was tested by dual-energy x-ray absorptiometry (DXA) and one repetition maximum strength test (1-RM).

CHAPTER 1

INTRODUCTION

Despite advancements in medical research, cardiovascular disease (CVD) continues to be the primary cause of death in the United States, Europe, and much of Asia (Braunwald, 1997; Breslow, 1997; Ross, 1999; Casey et al., 2007). The autonomic nervous system is a primary regulator of the cardiovascular system and its impairment is associated with increased arterial stiffness, which is an independent risk factor for the development for CVD (Blacher et al., 1999; Miyachi et al., 2004; Casey et al., 2007). Being obese often leads to impaired HRV, a marker of autonomic function (Paolisso et al., 2000). In addition, being obese results in impaired vascular function that leads to increased BP, PWV and AIx, which are markers of vascular function (Ko et al., 2010; Gokce et al., 2002). If left untreated, these alterations result in an increased risk of morbidity and mortality (Laurent et al., 2001).

Current research has focused on traditional exercise modes such as resistance, aerobic, and isometric exercise training as potential therapies to improve autonomic (Collier et al., 2008) and vascular function (Zhu et al., 2007). Recently, WBVT has emerged as a potential treatment for factors that contribute to the development of chronic diseases. This new exercise mode has been shown to improve bone mineral density, muscle strength, and cardiorespiratory fitness, however, WBVT's potential to improve autonomic and vascular function has not been explored.

Statement of the Problem

The aim of this study was to determine if 6 weeks of a progressive, lower body (legs) WBVT program would improve PWV, AIx, and BP, which are markers of vascular function as well HRV, a marker of autonomic function, in young overweight-obese women. The study also

aims to find if WBVT training improves body composition and muscle strength in this population.

Significance of the Study

Obesity is now recognized as a global epidemic, affecting an increasing number of people worldwide. Being overweight or obese is often associated with both vascular and autonomic dysfunction. The vascular and autonomic nervous systems are the main mechanisms of cardiovascular regulation and their impairment is often associated with increased arterial stiffness, which is considered an independent risk factor for hypertension. It has been shown that aerobic exercise (AE), resistance exercise (RE), and isometric exercise training can improve both autonomic and vascular function. Although AE and RE training are recommended to reduce cardiovascular risk factors and improve skeletal muscle function, obese and sedentary individuals can hardly be motivated to exercise regularly for more than 30 min at least 3 days per week. The benefits from AE and RE require moderate to high intensities, which in the obese individuals may cause joint and skeletal muscle injuries. WBV is a new exercise modality that uses an oscillating platform, which evokes reflexive muscle contractions.

Recently, WBV exercise has been shown to be an effective method to improve muscle mass, muscle strength, and bone mineral density. These results are comparable to those observed after RE training. This mode of exercise is performed either dynamically (with controlled movements) or statically (without joint movement). WBV requires less physical skills and shorter training sessions than AE and RE. Thus, it is possible to expect a greater compliance in previously sedentary overweight and obese individuals. To date, there is scarce information regarding cardiovascular adaptations to WBVT. It has been reported that brachial-ankle pulse wave velocity (baPWV, a marker of systemic arterial stiffness) is decreased between 20 and 40 min after a single WBV session in healthy men. The purpose of this study was to determine the efficacy of WBVT as an intervention to improve arterial and cardiac autonomic control of HR in overweight and obese premenopausal women.

Research Hypotheses

The research hypothesis is that participation in a WBVT program would improve cardiovascular and autonomic measurements: More specifically:

- Ha_1: There would be a decrease in AIx, PWV, and BP after the WBVT treatment at rest and after an acute bout of WBV exercise compared to no-exercise control (CON).
- Ha_2: There would be decreased levels of cardiac sympathetic activity, increased levels of parasympathetic activity (improved HRV), and a decreased HR after the WBVT treatment at rest and after an acute bout of WBV exercise compared to CON.
- Ha_3: There would be improvements in body composition with decreased fat mass and increased lean mass after the WBVT treatment compared to CON.
- Ha_4: There would be increases in muscle strength after the WBVT treatment compared to CON.

Limitations

1. A limitation of comparing pre- and post-training values at rest was that variations in cardiovascular function can occur due to the influence of many factors (environmental and dietary). To overcome this limitation, the effects of training at rest and after an acute bout of exercise were evaluated.

CHAPTER 2

REVIEW OF LITERATURE

Vascular Function

The vascular system plays a major role in the homeostasis of the cardiovascular system through its regulation of arterial tone. The endothelial layer of arteries accomplishes this through the release of relaxing and constricting chemical factors. These factors modulate the contractile activity of the smooth muscle cells in the medial layer of the arterial wall (Panza et al., 1993). One of the major vasoactive factors that act on the smooth muscle cells of arteries is endothelial nitric oxide (NO), a potent vasodilator (Maeda et al., 2008). This factor plays a major role in the control of blood flow (Williams et al., 2002; Lisanti et al., 1994) and BP (Zaros et a., 2009). Another regulator of vascular tone is endothelin-1 (ET-1). Production of this endothelial molecule induces systemic vasoconstriction and results in increased BP and arterial stiffness (Zaros et al., 2009).

Obesity and Vascular Dysfunction

Being overweight or obese is associated with impaired vascular function (Hickner et al., 2006; Panza et al., 1993). Vascular dysfunction is believed to be due to reduced bioavailability of NO (Ross, 1999), which leads to increased arterial stiffness. The arterial stiffening associated with vascular dysfunction (Tanaka et al., 2000) is independent of the age-related increase in arterial stiffness associated with structural changes in the arterial wall. A recent study by Hickner et al. (2003) found that endothelial NO synthase (eNOS), which is a protein that catalyzes the formation of NO from L-arginine, is inversely related to body mass index (BMI). In addition, waist circumference was found to be an independent predictor of eNOS content (Hickner et al., 2003).

Increased arterial stiffness via increased vascular tone is an adverse functional change that occurs in obese individuals. As a result of an increase in central (aorta and carotid) arterial stiffness, there is a reduction in baroreflex function, which is associated with a sympathetic-mediated increase in vascular tone (Kaushal & Taylor, 2002). If left untreated, chronic arterial stiffness ultimately results in hypertension and resulting cardiovascular complications (Bray, 1992; Sjostrom, 1992; Gordon & Kannel, 1979; Gokce et al., 2002). In contrast, age-related arterial stiffness is due to structural changes of the arterial wall. Such structural changes are reduced elastin and increased collagen fiber composition in the arterial media layer (Tanaka et al., 1998; Tanaka et al., 2000).

Arterial stiffness increases the velocity of pulse pressure waves that move throughout the arterial system. Among the methodologies for the evaluation of arterial mechanical properties, PWV is considered the gold standard index of arterial stiffness (O'Rourke et al., 2002). PWV is calculated on the basis of a pulse transit time and the distance travelled by a pulse wave between two arteries in different segments of the body (Sugawara et al., 2005). Depending on the elastic properties of the arteries, the velocity of the pulse pressure waves may be altered. The pulse waves contain an initial incident wave, which originates from the stroke volume, which is the blood ejected during ventricular systole (myocardial contraction). The incident wave travels from the aorta to the peripheral arteries and meets bifurcations that are mainly at the small muscular arteries and arterioles (Tahvanainen et al., 2009) and cause a second wave that is reflected back to the aorta during ventricular diastole (myocardial relaxation). In healthy young individuals, the pressure of the reflected wave (P2) is smaller than the pressure of the incident wave (P1) at rest (Miyachi et al., 2009). However, in individuals with chronically increased arterial stiffess at rest or in healthy individuals during or immediately after acute exercise, P2 arrives earlier to the aorta during late systole. The difference between P2 and P1 (augmented pressure) expressed as a percentage of the pulse pressure is accepted as an index of wave reflection, the augmentation index (AIx). Since both the reflection time (Tr) and amplitude of wave reflections (P1 and P2) are dependant to the stiffness of the entire arterial system, AIx is used as a marker of systemic arterial stiffness (Edwards & Lang, 2005). Since the AIx is negatively influenced by HR, the values for AIx are normalized to a HR of 75 beats/min (Wilkinson et al., 2000). Recent evidence suggests that obese individuals have an elevated AIx which implicates vascular dysfunction in this population (Maher et al., 2009).

Autonomic Function

The autonomic nervous system is a main regulator of HR and BP. The autonomic nervous system consists primarily of two divisions, the sympathetic and the parasympathetic. In response to stress, such as exercise, the activation of the sympathetic division causes an increase in HR and BP. During resting conditions, the parasympathetic division is primarily active and is responsible for a lower HR. Increased sympathetic activity leads to systemic vasoconstriction, which results in an elevation of BP and a reduction of blood flow to body organs (Grassi et al., 2004). Recent evidence suggests that the parasympathetic branch has a greater influence over HR regulation than the sympathetic division (Iellamo et al., 1999). Since HR is a primary determinant of coronary blood flow, high levels of parasympathetic activity are important for the prevention of myocardial ischemia and detrimental cardiac events. Dysfunction of the autonomic nervous system has been found to persist in obese individuals with the metabolic syndrome (Alvarez et al., 2005).

The arterial baroreflex mechanism is the main regulator of the autonomic nervous system for the control of BP; therefore, its impairment is associated with cardiovascular morbidity and mortality (La Rovere et al., 2002). Under normal conditions, increases in BP activate this mechanism through the resultant distension of central arteries. This distension produces mechanical stretch of the baroreceptors embedded in the arterial wall, which signal the cardiac centers in the medulla oblongata to increase the activity of the parasympathetic nervous system and reduce sympathetic activity. Increased sympathetic activity leads to systemic vasoconstriction, which reduces blood flow to the body organs and causes elevations in BP (Grassi et al., 2004). A decreased level of parasympathetic activity also negatively affects the cardiovascular system through an increased HR (Iellamo et al., 1999).

Obesity and Autonomic Dysfunction

Being overweight or obese is associated with dysfunction of the autonomic nervous system (Vaz et al., 1997; Poehlman et al., 1995). More specifically, being overweight or obese results in decreased levels of parasympathetic activity and increased levels of sympathetic

activity. Excess adipose tissue induces increased levels of sympathetic activity through its secretion of the adipocytokine leptin. Leptin has been shown to increase sympathetic activity (Crouse et al., 1995). Obesity-related hypertension has therefore been attributed to elevated sympathetic activity (Straznicky et al., 2005).

Research comparing obese individuals with their lean counterparts has shown other physiological differences that explain the increased incidence in autonomic dysfunction in this population. It has been reported that obese individuals have increased plasma norepinephrine concentrations (Troisi et al., 1991), which are attributed to an increased spillover rate of norepinephrine from the sympathetic nerve terminals (Poehlman et al., 1995). Elevated plasma norepinephrine levels have been shown to be predictor for future higher BP (Alvarez et al., 2002). In addition, a study conducted by Grassi et al. (1995) found that young obese individuals had twice the sympathetic nerve traffic to skeletal muscle compared to their lean counterparts even in the presence of normal BP. The observed increased sympathetic activity in the obese individuals was attributed to the impairment of the arterial baroreflex, since it is a primary regulator of the sympathic-parasympathetic balance (Grassi et al., 1995).

There is is a general recognition of the relationship that exists between autonomic function and cardiovascular mortality (Levy & Schwartz, 1994). An increase in the ratio between sympathetic and parasympathetic activity (increase in sympathetic and decrease in parasympathetic) is an independent risk factor for the development of CVD (Dekker et al., 2005). Measuring beat-by-beat variations in HR using a continous electrocardiogram can assess the activity of the sympathetic and parasympathetic nervous system. Measuring HRV detects the ratio of activity between the sympathic and parasympathic components of the autonomic nervous system. Through a transformation function, a power spectral domain can be generated (Kingsley et al., 2010). The power spectral domain displays low frequency bands (LF) and high frequency bands (HF), which are thought to reflect sympathetic activity and parasympathetic (vagal) activity, respectively. The ratio of LF to HF is therefore a reflection of sympathovagal balance (Breuer et al., 1993; Pagani et al., 1986).

In research, HRV is accepted as a non-invasive tool (Jonge et al., 2010) for assessing cardiac autonomic responses to exercise (Taylor et al., 2003; Kamath et al., 1991). A reduced HRV is an early indictor of cardiac complications and is frequently observed in obese individuals (Emdin et al., 2001) when compared to their non-obese counterparts (Rechlin et al., 1998).

Previous studies have shown that AE training can improve HRV in obese individuals (Kanaley et al., 2009; Amano et al., 2001). Improvements in HRV are attributable to alterations in central command and BRS, which are primary mediators that exert influence on autonomic nervous system activity (Yamamoto et al., 2001).

Resistance Exercise Training

In recent years, RE training has gained widespread acceptance as a therapeutic modality in cardiopulmonary rehabilitation programs (ACSM, 1998). However, RE training is primarily used for muscular strengthening to attenuate the risk of falling in the elderly and individuals with functional disabilities (Feigenbaum and Pollock, 1999). Currently, there is little information on the influence of RE training on non-musculoskeletal components, in particular, cardiovascular function (Miyachi et al., 2004). For this reason, RE is not regularly considered for improving cardiovascular function in clinical settings.

RE training is recommended as an integral component of overall cardiorespiratory and muscular fitness programs (Kraemer et al., 2002). Immediately after a bout of RE, blood flow to the working muscles is elevated posing an alternative stimulus to the endothelial cells. Olson et al. (2006) demonstrated that 1 year of RE training can improve vascular function in overweight women. In this study, there were improvements in vascular function, strength, and lean body mass (Olson et al., 2006).

Research on RE training also suggests that it may increase central arterial stiffness. An increase in central arterial stiffness impairs the buffering capacity of the arteries to accommodate the blood ejected by the heart (stroke volume) thereby contributing to elevations in SBP, left ventricular hypertrophy, coronary ischemic disease, and reduction in arterial BRS (Monahan et al., 2001). A study by Miyachi et al. (2004) showed that 4 months of conventional RE training resulted in an increase in carotid arterial stiffness. Although these results suggest a deleterious effect of RE training, the increase in arterial stiffness observed in this study was reverted after a 4 month detraining period (Miyachi et al., 2009). In contrast, Rakobowchuk et al. (2005) found that 12 weeks of RE training resulted in a reduction in brachial and carotid PP without any structural changes in the carotid intima-media thickness. These findings suggest that the

observed increase in central arterial stiffness in the study by Miyachi et al. were functional changes and not structural modifications in the large arteries. Differences between these studies exist in regards to the intensity and periodization of the training protocols. Miyachi et al. incorporated 4 months of training at a frequency of 3 sessions of repeated exercises per week; in contrast, Rakobowchuk et al. incorporated 3 months of training at a frequency of 5 sessions of different exercises (3 day cycles) per week. This difference allowed a greater recovery time for specific muscle groups in the latter study (Rakobowchuk et al., 2005). A study by Collier et al. (2008) which compared RE and AE training found that both exercise modes improve brachial SBP and DBP in individuals with pre- to essential-hypertension after 4 weeks. Similar to Miyachi's observation, this study found that RE training resulted in increased arterial stiffness, whereas AE training decreased arterial stiffness, despite similar reductions in BP (Collier et al., 2008).

A few studies on isometric exercise training suggest that it is a plausible treatment to improve cardiovascular health. This exercise mode is performed with isometric (no joint movement) contractions that are potentially dangerous because of their acute effect of dramatically increasing BP. However, McGowan et al. (2007) found improvements in vascular function through improvements in flow mediated vasodilation after 8 weeks of isometric handgrip training. Another group found reductions in SBP, DBP, and MAP after isometric exercise training. These changes may be associated with alterations in the function of the autonomic nervous system (Taylor et al., 2003). An improved baroreflex function may result in decreased levels of muscle sympathetic nerve activity and decrease brachial BPs (Sinoway et al., 1996).

Aerobic Exercise Training

In healthy subjects it is well established that AE training improves vascular function (Cameron & Dart, 1994; Tanaka et al., 2000). In diseased and at risk populations, the efficacy of AE training is well documented for reducing the risk of CVD. Numerous studies have shown that AE training has a positive effect on risk factors for CVD through improvements in vascular and autonomic function. The benefits of AE training on BP and arterial stiffness appear to be

derived from modifications in arterial structural integrity whereas there is an improvement in the artery's elasticity (Bertovic et al., 2009). It has been shown that the elasticity of the central arteries is greater in AE-trained men in comparison to their age-matched sedentary counterparts. This difference was suggested to be due to AE training's effect on the structural integrity of the arteries, whereas it increases the density of their elastic fibers (Tanaka et al., 2000).

Improvements in vascular function have also been shown through a decrease AIx in patients with coronary artery disease (Edwards et al., 2004). More recently, Collier et al. (2008) showed that 4 weeks a treadmill AE program decreases central arterial stiffness in hypertensive subjects resulting in decreased carotid-femoral PWV (cfPWV; Collier et al., 2008). A decrease in central arterial stiffness is important since the central arteries play a role in determining both SBP and DBP and left ventricular function (Rajkumar et al., 1997). Animal studies suggest that central arterial stiffness increases myocardial oxygen demand and reduces blood flow to the myocardium (Watanabe et al., 1993; Saeki et al., 1995).

AE training has been found to enhance brachial artery flow-mediated vasodilation (FMD) in obese individuals (Kelly et al., 2004). Since FMD is a marker of vascular function (Anderson et al., 1995), AE has been proposed as a suitable treatment for improving BP. This change explains the results of several studies that found that AE training decreased resting BP (Frick et al., 1963; Hagberg et al., 1983). Improvements in FMD have also been shown with a single bout of AE (Zhu et al., 2007). This response was attributed to the shear stress induced by exercise on the endothelial layer, which promotes the production of NO (Olson et al., 2006; Niebauer et al., 1996). In addition, Xiang et al. (2009) observed improvements FMD to be associated with decreases in low density lipoproteins and triglycerides after 6 months of AE training in middle-aged women. Therefore, this group suggested that another possible mechanism for an improvement in FMD is mediated by improvements in lipid metabolism (Xiang et al., 2009).

AE training augments NO release by introducing shear stress to the endothelial layer (Higashi et al., 2004). The stress induced by AE training improves the endothelial layer's function by enhancing its abiity express the eNOS gene. An increased number of the eNOS enzyme content in the arteries leads to increased acetylcholine-stimulated release of the vasodilator NO (Sessa et al., 1994). Subsequent to these changes, BRS improves since stimulation to baroreceptors is dependent on the stiffness of the arterial wall (Zoppini et al., 2007).

In addition to improvements in vascular function, improvements in autonomic function have been shown to result from this training mode at low (Zoppini et al., 2007), moderate (Higashi et al., 1999; Higashi et al., 2004; Pagkalos et al., 2008), and high intensities. Improvements in resting BP resulting from AE training in obese women were demonstrated by Figueroa et al. (2007). This group demonstrated that improvements in BRS and HRV could be attained with 16 weeks of moderate-intensity AE (Figueroa et al., 2007). Although adaptations in cardiac autonomic function were not observed at rest, the improvements were apparent during the recovery from the acute exercise.

Whole-body Vibration Exercise

WBVT is a new exercise mode that is currently being investigated in the field of space travel, sports, and rehabilitation (Otsuki et al., 2008). This exercise mode uses an oscillating platform that delivers sinusoidal vibrations (Cochrane et al., 2008) that evoke reflexive muscle contractions (Bazrgari et al., 2008) while the person performs steadily controlled dynamic and static exercises. The vibration mechanically stimulates sensory receptors, leading to the activation of alpha-motoneurons and consequently muscle contractions through the tonic vibration reflex (TVR) (Delecluse et al., 2003). These contractions explain the increased levels of electromyography in working muscle during WBV exercise (Cardinal & Lim, 2003; Hazell et al., 2008), and the increased energy output that is shown to result from the addition of vibration to squat exercises (Silva et al., 2007). These findings have brought about research based on the usefulness of this method as a suitable exercise (Delecluse et al., 2003) for special populations such as those that cannot perform conventional exercise, which include the obese.

Previous research has shown that WBVT is an effective exercise mode to improve individuals' functional parameters. Neuromuscular adaptations have been the primary area of focus for this new training mode. Recent evidence suggests that WBV exercise could be an alternative exercise modality for eliciting strength in clinical (Furness et al., 2010) and frail populations (Runge et al., 2000; Kawanabe et al., 2007; Bogaerts et al., 2007). Findings of improved strength have also been shown to result from WBVT in younger individuals. The addition of vibration to exercise programs has been shown to increase strength and power more

than exercise programs without vibration (Ronnestad, 2004; Kawanabe et al., 2007). Additionally, increases in strength of the knee extensor muscles and bone mineral density have been shown after 6 months (Verschueren et al., 2004) and 1 year of WBVT in post-menopausal women (Rubin et al., 2004). Improvements in bone mineral density hsvr been suggested to be due to the observed increases in muscle mass that result from WBVT (Bogaerts et al., 2007).

Research on the effect of WBV on the cardiovascular system is scarce. Most research in this area has focused only on the acute hemodynamic changes that occur following a bout of WBV exercise. Kerschan-Schindl et al. (2001) demonstrated increased blood flow in the popliteal artery with 9 min of continuous standing in a static semi-squat position using WBV. Lohman et al. (2007) found increases in skin blood flow while their subjects rested their calves on the vibration platform, which suggests that direct vibration affects inactive musculature (Lohman et al., 2007). Increases in blood flow were also demonstrated by Yamada et al. (2005) who found that blood volume in the vastus lateralis acutely increases after WBV with a dynamic squat exercise (Yamada et al., 2005). These results are supported by Otsuki et al. (2008) who found a decrease in baPWV after a session of WBV static squat exercises (Otsuki et al., 2008).

In addition to the increased popliteal blood flow, it has been shown that static squat with WBV does not significantly increase HR and BP (Kerschan-Schindl et al., 2001). These findings were similar to the previously mentioned study by Otsuki et al. (2008) who found that HR and BP were unchanged 20 and 40 min after a WBV exercise session. A later study found that HR and BP could be increased using WBV dynamic squat exercises. However, the investigators added a weighted-load of 35-40% of the body weight to their subjects, which was later determined to have an effect on metabolic rate. The addition of a load to WBV exercise was later evaluated in young and older individual on metabolic activity. It was observed that with an external load on the subject performing WBV exercises, metabolic activity was increased as shown by increased oxygen consumption (Cochrane et al., 2008).

The cardiovascular adaptations to WBVT are not well understood. In turn, this presents a gap in knowledge for the use of this exercise modality in improving cardiovascular function in individuals with cardiovascular risk factors. Therefore, the purpose of this study is to determine the changes in cardiovascular and autonomic function following 6 weeks of WBVT in overweight and obese premenopausal women.

CHAPTER 3

RESEARCH METHODOLOGY

The purpose of the study was to determine if a progressive WBVT program would improve physiological indicators of cardiovascular and autonomic function in overweight-obese women. Thus far, the effects of WBVT on these parameters have not been investigated. The data from this investigation may provide clinicians in cardiac rehabilitation settings with a new adjunct treatment for CVD.

Subjects

The Florida State University Human Subjects Committee approved this investigation. The subjects consisted of eight women (18-35 years) with a BMI between 25 kg/m^2 and 40 kg/m^2. Women with diverse ethnic backgrounds were recruited from the Tallahassee metropolitan area by advertisement (fliers) and direct communication. The exclusion criteria were based on contraindications to exercise. Subjects were nonsmokers and did not exercise regularly, defined as more than 120 min per week. Individuals were excluded if they fell outside of the specified BMI range and/or had chronic diseases including epilepsy, gallstones, kidney stones, acute inflammations, arthritis, joint implants, recent thrombosis, recent operative wounds, intense migraines, tumors, hernias, and uncontrolled diabetes, cardiovascular, and kidney diseases. Participants were excluded if they were pregnant or taking dietary supplementations (e.g., L-arginine, L-citrulline, and antioxidants) or medications (e.g., beta blockers, antidepressants, and stimulants) that may influence our outcome variables. The Florida State University Human Subject committee approved the experimental procedures, and written informed consent was obtained from all subjects. Subjects were asked to maintain their diet habits during the study period.

Research Design

In a crossover design, participants were randomized to either the WBVT or CON treatment for the first 6 weeks of the study followed by a 4 week washout period and then crossed over to their next treatment. The participants came to the Cardiovascular Physiology Laboratory at The Florida State University on 9 different visits. On the first visit they were oriented to the study and were measured for their height and weight to calculate BMI. If participants met the criteria, they were asked to fill out a medical history questionare and consent form. Thereafter, subjects were randomly assigned to either CON or the WBVT period for 6 weeks. During visit two, measurements were conducted in the morning after at least 8 hours of an overnight fast and during the early follicular phase of their ovarian cycle to avoid the vasoactive effects of estrogen. All measurements were performed in the morning in a quiet temperature-controlled room (22-24°C). Participants rested in the supine position for at least 10 min before data collection. Subsequent to the resting period, baseline measurements of electrocardiogram (ECG), BP, PWV, and pulse wave analysis (wave reflection and aortic BP) were obtained. Measurements were repeated 3 min after the cessation of a 4 min bout of WBV static squat. During the third visit, participants were measured with a noninvasive DXA to determine whole body and regional body composition. Subsequent to the DXA measurement, subjects performed a muscle strength test. After the body composition and strength testing, participants began their assigned 6 week period of CON or WBVT along with a 4 week washout period (detraining). In the control and washout period, subjects did not participate in any scheduled exercise. During all periods, the subjects were told not to divert from their regular diet or take any dietary supplements. Measurements were taken at the beginning and the end of each period to assess changes in the variables being assessed. After each of these periods the same measurements as in visits 2 and 3 were repeated.

Cardiovascular Health/History Questionnaire

The Cardiovascular Health/History Questionnaire was used to assess the subjects' current health status for either inclusion or exclusion to the study. It consisted of 21 questions that

indicated whether the subject had any current or past underlying diseases, including medical problems that may disrupt the training regimen or measurements.

Anthropometrics

Height was measured using a stadiometer to the nearest 0.5 cm, and body weight was measured using a Seca Scale (Sunbeam Products, Boca Raton, FL) to the nearest 0.1 kg. BMI was calculated as kg/m^2 to determine if they fell into the inclusion criteria.

Body Composition

Total mass, fat mass, body fat percentage, and lean mass were evaluated by a whole-body DXA scan (Lunar ™ DPX-IQ, Madison, WI). DXA scans were conducted in the Exercise Physiology Laboratory on the first floor of the Sandels Building on the campus of The Florida State University.

Cardiovascular Parameters

After 10 minutes of rest in the supine position, brachial and ankle BPs, aortic and baPWV were measured using an automatic device (VP-2000; Omron Healthcare, Vernon Hills, IL). BP cuffs were wrapped around both arms (brachial artery) and ankles (posterior tibial artery), and two tonometers were placed over the right carotid and femoral arteries to obtain BP and PWV measurements from three arterial segments: baPWV, cfPWV, and femoral-ankle PWV (faPWV). The cfPWV and faPWV are considered measurements of aortic and leg arterial stiffness respectively. The carotid and femoral arterial waveforms were recorded simultaneously by tonometers, and the transient time was calculated automatically by relating the foot of each waveform to the R-wave of the electrocardiogram. The distance from the carotid and femoral artery was measured with a nonelastic tape measure as a straight line, while the distance between

sampling points of baPWV and faPWV was calculated automatically according to the height of the subjects. PWV was calculated as the distance between carotid and femoral sites divided by the transit time. HR was obtained from the ECG. Two measurements were collected at each time point and averaged.

To measure wave reflection and aortic BP, the arterial pulse waveforms were obtained non-invasively using a pencil-like high fidelity transducer (SPT-301B; Millar Instruments, Houston, Texas, USA) to obtain BP waveforms from the radial artery of the left arm by applanation tonometry. Aortic BP waveforms were derived using a vailidated generalized transfer function (SphygmoCor; ATCor Medical, Sydney Australia).

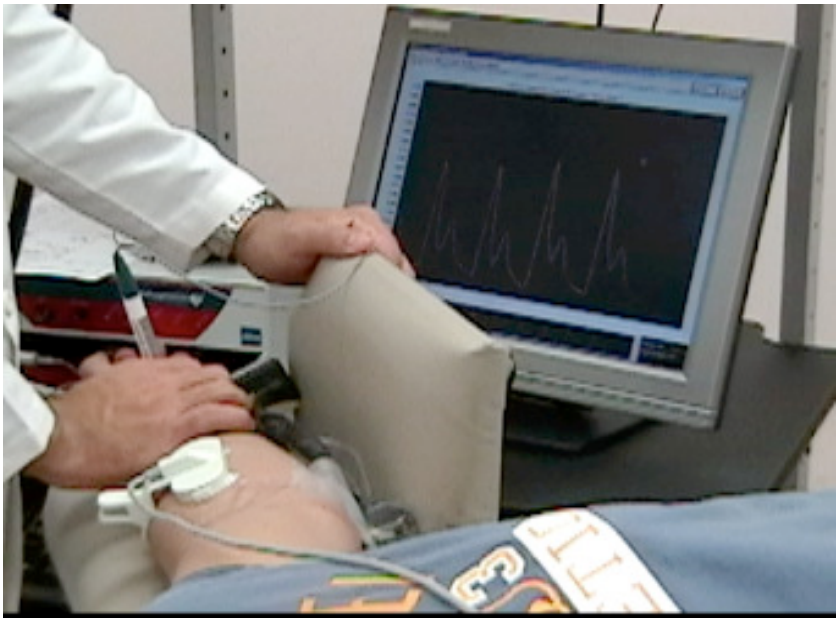

Figure 1. Applanation Tonometry

Brachial SBP and brachial DBP were used to calibrate radial waveforms, which were obtained from the transducer. PP was the difference between SBP and DBP. The aortic wave is composed of a forward wave, caused by stroke volume ejection, and a reflected wave that returns to the aorta from peripheral sites. Aortic pressure waveforms were synthesized from the radial pressure waveform using a transfer function (SphygmoCor, AtCor Medical, Sydney, Australia). Augmentation pressure was defined as the difference between P2 and P1 of the pressure wave. In addition, AIx was defined as the augmentation pressure expressed as a percentage of the aortic

pulse pressure. AIx was normalized for a HR of 75 beats/min (AIx@75). The transit time for the reflected wave (Tr) indicates the travel time of the forward wave to the peripheral reflecting sites and back to the aorta. AIx and Tr have previously been used as markers of wave reflection and aortic stiffness, respectively (Figueroa. et al., 2010).

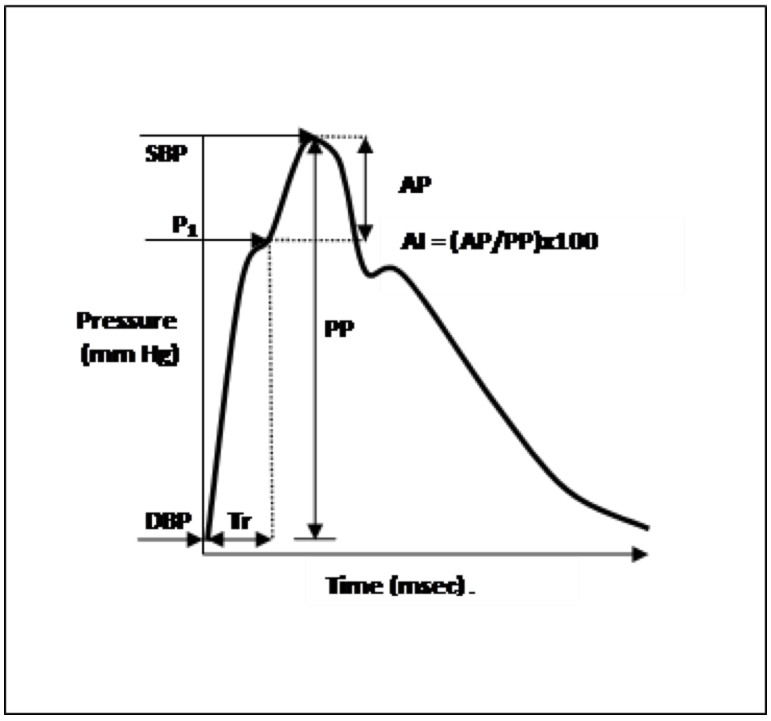

Figure 2. Aortic pulse waveform, AIx, augmentation index; AP, augmentation pressure, DBP, diastolic blood pressure; P1, first systolic peak; PP, pulse pressure; SBP, systolic blood pressure; Tr, transit time of reflected wave

Heart Rate

HR was obtained from a continuous electrocardiogram by the use of a bipolar lead sampled by a data acquisition system (Biopac, Santa Barbara, CA).

Cardiac Autonomic Regulation of Heart Rate

For autonomic function, a continuous ECG recording was obtained from a modified CM5 lead sampled by a data acquisition system (Biopac, Santa Barbara, CA, USA) and stored on a computer. Breathing was controlled by using a metronome, which was set to 12 breaths per min. The R-to-R (RR) intervals were inspected and edited for sporadic ectopic beats and technical artifacts were removed. Fast Fourier transformation was used to attain the total power of HRV and its main components: HF (0.15-0.4 Hz) and LF (0.04-0.15 Hz) using the WINCPRS software (Absolute Aliens, Turku, Finland). The HF power is considered an indicator for cardiac parasympathetic activity while the LF component of HRV is mediated by the sympathetic and parasympathetic nervous systems.

Strength Testing

Maximal strength was measured after a light warm-up set of 10-12 repetition on a MedX^{TM} leg extension. After the warm-up and a 2 min resting period, participants performed a maximal leg-extension through a full range of motion with controlled movement. If the participant was able to achieve the estimated weight (intensity), it was increased until the subject was unable to achieve a complete leg-extension through the full range of motion. All of the measurements were recorded within three to five attempts. If the subject exceeded five attempts, they were asked to return to the Exercise Physiology Laboratory to perform the strength test again. The highest measurement for the leg extension was considered the 1-repetition maximum (1-RM). All of the strength measurements were taken by the same researcher who was a certified strength and conditioning specialist.

Acute Bout of WBV Exercise

The subjects performed a 4 min static squat exercise at a knee angle of 120˚ on the WBV platform. This method has previously been shown to sufficiently increase in the activity of the

cardiovascular system (Figueroa et al., 2010). The frequency and the amplitude of the vibrations were set to 30 Hz and 1.0 mm, respectively. Continuous ECG and beat-by-beat measurements were taken continuously during and after the acute bout for later analysis. PWV, pulse wave analysis, and BP measurements were taken at minute 3 following the exercise bout.

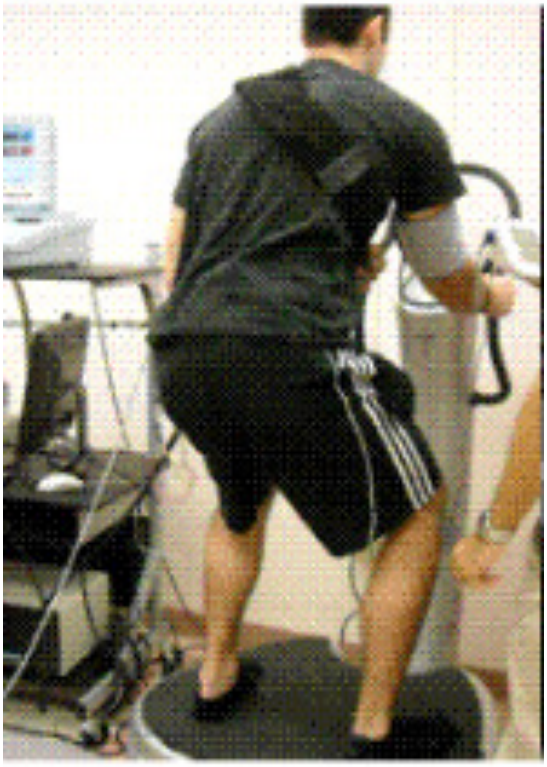

Figure 3. Acute bout of WBV

Whole-Body Vibration Training

All subjects underwent 3 supervised training sessions three times weekly for 6 weeks. The subjects performed static and dynamic exercises for the legs (squats and calf raises) on a vibration platform. The squat and toe-stand exercises were performed with slow controlled movements starting from an upright position into a 60 degree knee flexion (squat) and maximal heel elevation (toe-stand). These movements were performed at a rate of 3 seconds down and 3 seconds up. Static exercises were performed without movement in the joint angles described previously. The training volume increased progressively over the 6 week training period by

increasing the intensity of vibration (25 to 30 Hz of frequency and 1 mm to 2 mm of amplitude), duration of the exercise set (30-60 sec), and total duration of the training session (8-30 min), and decreasing the duration of rest periods (60 sec to 30 sec). During the last 2 weeks, resistance was added progressively by the use of a weighted vest that did not exceed 20% of the subject's body weight.

Statistical Analysis

An unpaired *t*-test was used to detect differences in parameters between both treatments (WBVT vs CON) at baseline. Normal distribution of the data was examined with the Shapiro-Wilk test. Because of non-normal distribution of LF and HF powers were not normally distributed, they were analyzed after a natural log transformation (Ln). Comparison between and within treatments was performed by two-way (treatment x time) ANOVA with repeated measures at rest and 3 min post-exercise [treatment (CON vs. WBVT) X rest or PE3 (before vs. after treatment)] was used to detect possible differences between groups. When significant treatment-by-time interactions were found, *t*-tests were used for post hoc comparisons. Significance was set at a prior of α at 0.05. Data are shown as means ± standard error. Statistical analyses were performed using SPSS, version 16.0 (SPSS, Chicago, IL).

CHAPTER 4

RESULTS

All of the eight subjects (n=8), who started the study, completed it in its entirety. Following the crossover design, these women completed a 6 week training period along with a 6 week control period of no exercise. Those subjects that began with the WBVT period completed a sequential 4 week detraining period before they began the 6 weeks of no training. All subjects completed the designated training protocol, which consisted of 18 training sessions. The same trainer conducted all of the training sessions. Participants were asked not to deter from their normal diet or start another exercise program.

Participant Characteristics and Strength

Participant characteristics before (PRE-WBVT) and after (POST-WBVT) the intervention as well as before (PRE-CON) and after (POST-CON) the control period are presented in Table 1. There were no significant differences in total mass, fat mass, lean mass, percent fat (% Fat), and waist circumference between all of the measurements throughout the study. Maximal leg extension strength significantly (p <0.01) increased from PRE-WBVT to POST-WBVT while it did not change from PRE- to POST-CON.

Table 1 Participant body composition and strength (n=8)

Variables	**PRE-WBVT**	**POST-WBVT**	**PRE-CON**	**POST-CON**
Total Mass (kg)	78 ± 3	77 ± 3	82 ± 4	80 ± 4
Fat Mass (kg)	32 ± 2	31 ± 2	35 ± 3	33 ± 3
Lean Mass (kg)	43 ± 1	43 ± 1	43 ± 2	44 ± 1
% Fat	42 ± 1	41 ± 2	42 ± 2	41 ± 2
Waist Circumference (cm)	97 ± 3	97 ± 4	97 ± 4	97 ± 3
Muscle Strength (kg)	126 ± 6	138 ± 8*	123 ± 6	127 ± 3

Data are mean ± SE. WBVT, whole-body vibration training; CON, no-exercise control; PRE, before treatment; POST, after treatment; %Fat, whole-body fat percentage. *Significantly different from PRE (p < 0.01).

Peripheral and Central Blood Pressures

Peripheral and central BPs for PRE-WBVT and POST-WBVT as well as PRE-CON and POST-CON for rest and PE3 are presented in Table 2 and Table 3, respectively. Resting brachial SBP and DBP significantly ($p < 0.05$) decreased after WBVT. There was a significant ($p < 0.05$) time-by-treatment interaction for resting brachial SBP (Figure 2 A) and DBP (Figure 2 B). There was a significant ($p < 0.05$) decrease in post-exercise brachial SBP and MAP (time effect) after WBVT, which was significantly different than CON (time-by-treatment interaction, $p < 0.05$) (Figure 2 A and B).

Table 2 Resting peripheral and central blood pressures before and after the WBVT and CON treatment.

	WBVT		CON	
	PRE	**POST**	**PRE**	**POST**
Brachial SBP (mmHg)	119 ± 4	111 ± 2*†	114 ± 3	121 ± 5
Brachial DBP (mmHg)	65 ± 2	61 ± 1*†	63 ± 1	65 ± 2
Brachial MAP (mmHg)	85 ± 3	83 ± 2	84 ± 2	89 ± 3
Brachial PP (mmHg)	52 ± 1	51 ± 2	51 ± 3	55 ± 4
Aortic SBP (mmHg)	97 ± 1	96 ± 2	96 ± 2	102 ± 4
Aortic DBP (mmHg)	64 ± 1	62 ± 2	64 ± 1	66 ± 2
Aortic MAP (mmHg)	80 ± 1	78 ± 2	80 ± 1	83 ± 2
Aortic PP (mmHg)	33 ± 1	34 ± 1	33 ± 1	36 ± 3

Data are mean ± SE. WBVT, whole-body vibration training; CON, no-exercise control; PRE, before treatment; POST, after treatment; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure; *Significant ($p < 0.05$) time-effect. †Significant ($p < 0.05$) time-by-treatment interaction.

Table 3 Post-exercise peripheral and central blood pressures before and after the WBVT and CON treatment.

	WBVT		CON	
	PRE	**POST**	**PRE**	**POST**
Brachial SBP (mmHg)	126 ± 3	121 ± 3*†	123 ± 4	125 ± 2
Brachial DBP (mmHg)	68 ± 2	66 ± 2	69 ± 2	70 ± 2
Brachial MAP (mmHg)	94 ± 2	87 ± 2*†	89 ± 2	93 ± 2
Brachial PP (mmHg)	55 ± 3	55 ± 2	54 ± 3	56 ± 3
Aortic SBP (mmHg)	107 ± 4	106 ± 3	105 ± 3	106 ± 3
Aortic DBP (mmHg)	70 ± 3	69 ± 2	69 ± 2	68 ± 2
Aortic MAP (mmHg)	88 ± 3	86 ± 2	87 ± 2	86 ± 3
Aortic PP (mmHg)	38 ± 3	37 ± 2	36 ± 2	37 ± 2

Data are mean ± SE. WBVT, whole-body vibration training; CON, no-exercise control; PRE, before treatment; POST, after treatment; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; PP, pulse pressure. *Significant ($p < 0.05$) time-effect. †Significant ($p < 0.05$) time-by-treatment interaction.

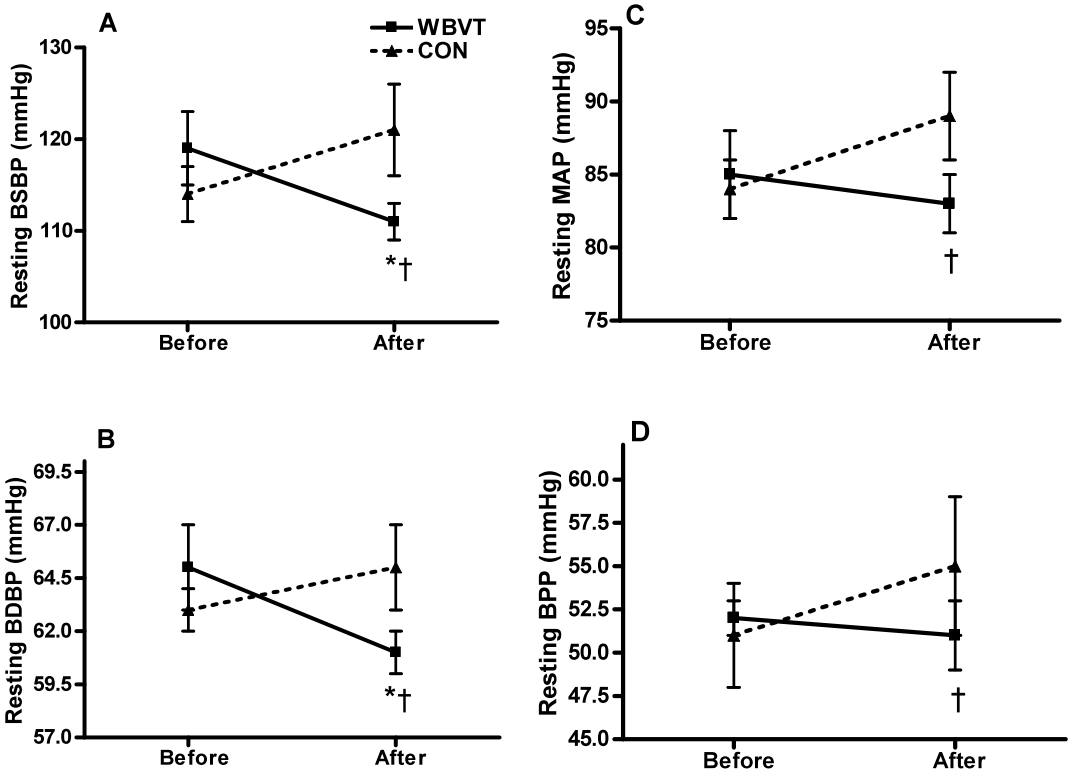

Figure 4. Cardiovascular variables at rest before and after each treatment. Changes in resting brachial systolic blood pressure (A), brachial diastolic blood pressure (B), brachial mean arterial pressure (C), and brachial pulse pressure (D) from before until after each treatment. *Significant ($p < 0.05$) time-effect. *Significant ($p < 0.05$) time-effect. †Significant ($p < 0.05$) time-by-treatment interaction.

Arterial Stiffness and Wave Reflection

Arterial stiffness and wave reflection for PRE-WBVT and POST-WBVT as well as PRE-CON and POST-CON are presented in Table 3. No changes were detected for arterial stiffness and wave reflection at rest and PE3 after WBVT and CON. However, it is important to note that there was a non-significant change in resting faPWV after WBVT.

Table 4 Resting arterial stiffness and wave reflection before and after the WBVT and CON treatment.

	WBVT		CON	
	PRE	**POST**	**PRE**	**POST**
FaPWV (cm/sec)	926 ± 90	818 ± 23¶	816 ± 35	851 ± 37
CfPWV (cm/sec)	688 ± 68	693 ± 22	773 ± 52	719 ± 64
BaPWV (cm/sec)	1012 ± 43	1007 ± 30	999 ± 25	1021 ± 72
AIx @75(%)	1.1 ± 0.8	0.6 ± 0.9	1.9 ± 0.9	0.3 ± 0.5
Tr (m•sec)	170 ± 9.6	179 ± 14.5	151 ± 4.1	156 ± 11.5

Data are mean ± SE. WBVT, whole-body vibration training; CON, no-exercise control; PRE, before treatment; POST, after treatment; PWV, pulse wave velocity; FaPWV, femoral-ankle PWV; CfPWV, carotid-femoral PWV; BaPWV, brachial-ankle PWV; AIx @75, AIx adjusted for 75 beats/min; Tr, reflection time. ¶, tendency for significance ($p < 0.1$; time effect).

Table 5 Post-exercise arterial stiffness and wave reflection before and after the WBVT and CON treatment.

	WBVT		CON	
	PRE	**POST**	**PRE**	**POST**
FaPWV (cm/sec)	833 ± 26	839 ± 55	850 ± 45	834 ± 20
CfPWV (cm/sec)	1067 ± 229	1004 ± 228	667 ± 62	882 ± 90
BaPWV (cm/sec)	1017 ± 54	1041 ± 41	1090 ± 60	1041 ± 52
AIx @75(%)	3.1 ± 1.2	3.1 ± 1.2	3.7 ± 0.8	3.7 ± 0.8
Tr (m•sec)	142 ± 3.3	149 ± 7.4	146 ± 7.1	154 ± 13.4

Data are mean ± SE. WBVT, whole-body vibration training; CON, no-exercise control; PRE, before treatment; POST, after treatment; PWV, pulse wave velocity; FaPWV, femoral-ankle PWV; CfPWV, carotid-femoral PWV; BaPWV, brachial-ankle PWV; AIx @75, AIx adjusted for 75 beats/min; Tr, reflection time.

Autonomic Function

Spectral analysis of HRV and HR for PRE-WBVT and POST-WBVT as well as PRE-CON and POST-CON are presented in Table 6. There was no significant change in resting HR after both WBVT and CON. Post-exercise HR was significantly ($p < 0.05$) decreased after WBVT (Table 7 and Figure 2C). A significant ($p < 0.05$) time-by-treatment interaction was also detected for post-exercise HR for POST-WBVT.

Table 6 Spectral analysis of heart rate variability during rest before and after the WBVT and CON treatment.

	WBVT		CON	
	PRE	**POST**	**PRE**	**POST**
HR (beats • min^{-1})	64 ± 2	64 ± 2	67 ± 3	63 ± 4
LnLF (ms^2)	6.4 ± 0.2	6.5 ± 0.2	6.2 ± 0.2	6.2 ± 0.1
LnHF (ms^2)	7.2 ± 0.3	7.4 ± 0.3	7.0 ± 0.3	6.8 ± 0.3

Data are mean ± SE. WBVT, whole-body vibration training; CON, no-exercise control; PRE, before treatment; POST, after treatment; HR, heart rate; LnLF, natural log of low frequency values; LnHF, natural log of high frequency values. *Significant ($p < 0.05$) time-effect. †Significant ($p < 0.05$) time-by-treatment interaction

Table 7 Spectral analysis of heart rate variability during post-exercise before and after the WBVT and CON treatment.

	WBVT		CON	
	PRE	**POST**	**PRE**	**POST**
HR (beats • min^{-1})	69 ± 2	62 ± 3*†	69 ± 2	72 ± 6
LnLF (ms^2)	6.8 ± 0.2	6.7 ± 0.3	6.7 ± 0.1	6.5 ± 0.3
LnHF (ms^2)	6.7 ± 0.5	7.0 ± 0.4	7.0 ± 0.4	6.4 ± 0.5

Data are mean ± SE. WBVT, whole-body vibration training; CON, no-exercise control; PRE, before treatment; POST, after treatment; HR, heart rate; LnLF, natural log of low frequency values; LnHF, natural log of high frequency values. *Significant ($p < 0.05$) time-effect. †Significant ($p < 0.05$) time-by-treatment interaction.

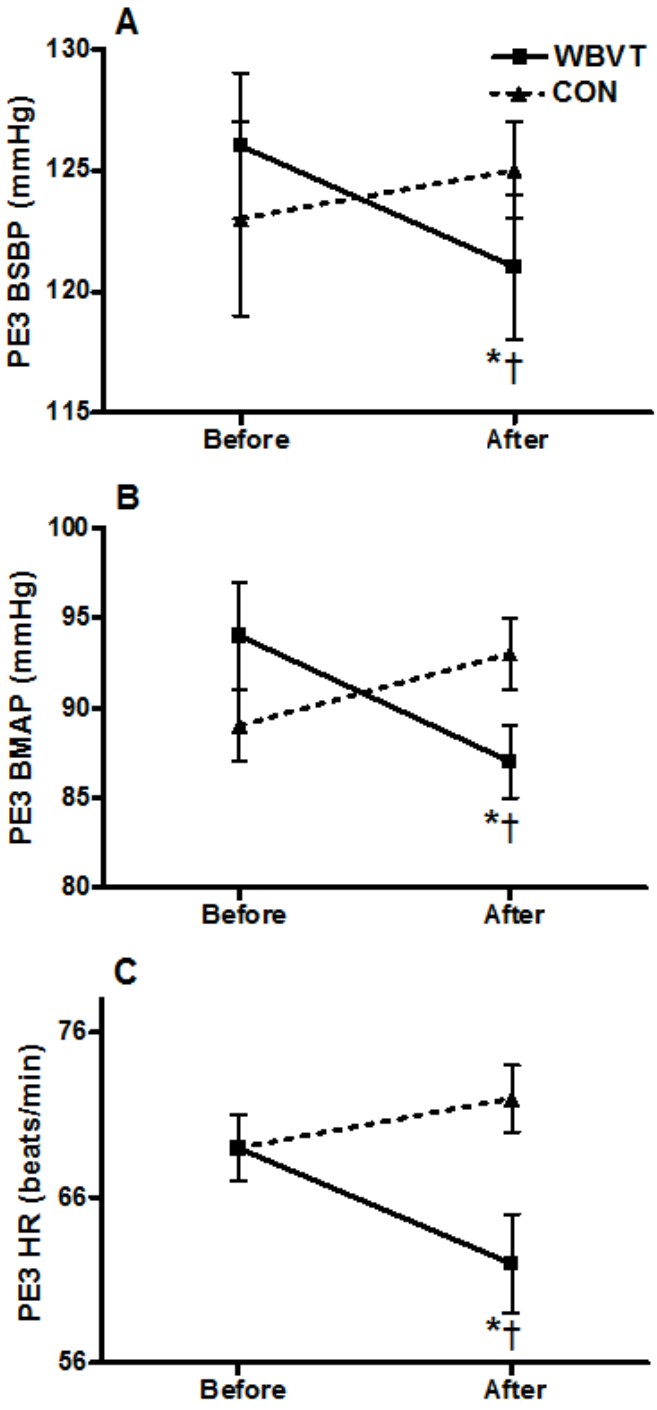

Figure 5. Cardiovascular variables during 3 min post exercise before and after each treatment. Changes in 3 min post-exercise brachial systolic blood pressure (A), brachial mean arterial pressure (B), and heart rate (C) from before until after each treatment. *Significant ($p < 0.05$) time-effect. †Significant ($p < 0.05$) time-by-treatment interaction.

CHAPTER 5

DISCUSSION

Previous studies have shown that WBVT elicits improvements in lower body strength (Ronnestad, 2004; Kawanabe et al., 2007; Ness & Field-Fote, 2009), muscle mass, balance (Torvinen et al., 2002), bone mineral density (Bogaerts et al., 2007), and peak oxygen uptake (Bogaerts et al., 2009), which are similar to the results of conventional resistance and aerobic exercise training. Before this study, was a gap in research regarding the effects on the cardiovascular and autonomic system after acute an acute bout of WBV after WBVT. This investigation therefore sought to determine the effects of WBV exercise and WBVT on BP, PWV, wave reflection, and HRV in overweight-obese women since it has been shown that these parameters are impaired in this population.

Resting Blood Pressure

In this study, WBVT significantly decreased resting SBP (8 ± 5 mmHg) and DBP (4 ± 2 mmHg) similar to the outcomes that occur after conventional AE, RE (Collier et al., 2008), and isometric exercise training (Ray & Carrasco, 2000). The reduction in SBP ater 6 weeks of WBVT was similar to the change reported previously in middle-aged obese women with and without type 2 diabetes after 16 weeks of AE training (Figueroa et al., 2007). These changes in BP have been found to be independent of body weight, body fat percentage, and changes in arterial stiffness after AE training (Collier et al., 2008; Figueroa et al., 2007). Furthermore, the decreases in resting peripheral BP were found to be independent of changes in cardiac autonomic function, in agreement with previous findings after isometric and AE training (Figueroa et al., 2007; Ray & Carrasco, 2000). Although the reductions in peripheral DBP were modest, recent evidence suggests that small reductions in this variable can provide significant health benefits. A drop in DBP of only 2-mmHg leads to a 6% reduction in coronary heart disease as well as a 15%

reduction in stroke related events (Cook et al., 1995). Therefore, the 4-mmHg decrease reported in this study would have an important clinical impact in populations with CVD.

Although there was no apparent reduction in sympathetic nerve outflow to the heart after training, it is possible that sympathetic outflow to other vascular beds (e.g., visceral regions) was reduced. It has been suggested that decreases in sympathetic activity to visceral regions may contribute to reductions in peripheral arterial pressure at rest (Ray & Carrasco, 2000). This suggested mechanism of decreased regional sympathetic nerve activity would be beneficial for overweight-obese women since it has been established that obesity is associated with elevated sympathetic nerve traffic to viscera, such as the kidneys (Vaz et al., 1997). Regional sympathetic activity to the kidney has been proposed as a physiological mechanism that leads to hypertension and chronic kidney disease (Schlaich et al., 2009). In addition, a reduction of sympathetic activity to the blood vessels in the skeletal muscle may also explain the decrease in peripheral BPs. It has been reported that young obese individuals have twice the sympathetic nerve traffic to skeletal muscle when compared to their lean counterparts even in the presence of normal BP (Grassi et al., 1995).

The repetitive exposure of shear stress on the blood vessels that occurs during exercise upregulates the production of eNOS enzymes in the endothelium (Delp & Laughlin et al., 1995). Results from past studies have shown that the expression of eNOS is increased in rats following AE training (Tatchum-Talom et al., 2000; Vassilakopoulos et al., 2003). A change in the number of eNOS enzymes can be proposed as a possible mechanism for the decreases in resting peripheral BP. A higher concentration or activity of these enzymes may be responsible, at least in part, for the reductions in BP due increased NO-mediated relaxation of the vascular smooth muscle (Negrão et al., 2003).

Previous studies on RE training have shown an increase in arterial stiffness in young adults. This was shown to be evident through increased leg PWV in male strength-trained athletes (Bertovic et al., 1999) and decreased compliance (increased stiffness) of the central elastic arteries in young men (Miyachi et al., 2004). It has been shown that RE training increases AIx and aortic PWV, which are markers of wave reflection and central arterial stiffness, respectively, in young healthy women (Cortez-Cooper et al., 2005). In contrast, recent evidence indicates that RE training does not increase arterial stiffness in post-menopausal women (Casey et al., 2007) and more importantly RE training can reduce PWV when RE is prescribed at low to

moderate intensities (Okamoto et al., 2009). In contrast to the previous studies who found increased arterial stiffness following RE training, the present investigation found no significant changes in arterial stiffness and wave reflection after WBVT. Our results are similar to those found by Casey et al. (2007) with increases in strength without increases in arterial stiffness. It is important to note that systemic arterial stiffness (baPWV) and markers of wave reflection tended to improve in the present study. However, due to the small number of participants, the study was underpowered to observe a significant effect of WBVT on vascular function. In addition, it may take longer than 6 weeks to observe changes in PWV.

Post-exercise Blood Pressure and Heart Rate

Through this study, there was a decrease in post-exercise brachial SBP (5 ± 4 mmHg), MAP (7 ± 3 mmHg), and HR (7 ± 4 bpm) after 6 weeks of WBVT. Although post-exercise pulse wave reflection and cardiac autonomic regulation did not reveal significant alterations after WBVT, the lower post-exercise BP and HR may be associated with an improved cardiovascular autonomic regulation after WBVT. Alterations in cardiac autonomic control, however, were not found to be statistical significant, possibly due to the small sample size. A previous report found an increase in post-exercise BRS after 16 weeks of AE training in obese women (Figueroa et al., 2007). An improved post-exercise BRS implies a reduction in post-exercise sympathetic activity (O'Sullivan & Bell, 2000) occurring during PE3 and would therefore explain the reduction in brachial BP (Irigoyen et al., 2005). Improvements in BRS through exercise training may be related to repeated activation of the baroreflex responses. Exercise training will therefore "train" the baroreflex to be more responsive (sensitive) to changes in blood pressure (Liomaala et al., 2003). In contrast, there are also investigations that report exercise training having no effect on BRS (Smith et al., 2000; Mack et al., 1987; Shi et al., 1993). Another possible mechanism for the decrease in post-exercise BP after WBVT is that vascular sensitivity to norepinephrine may have been reduced (Kingwell et al., 1996). Furthermore, a study using a rat model reported a decrease in alpha-adrenergic receptor sensitivity following 6 weeks of AE training (Wiegman et al., 1981).

In the present study, 6 weeks of WBVT decreased post-exercise HR. We also found that cardiac parasympathetic (vagal) activity (LnHF power) tended to increase after WBVT. This alteration may explain the decrease in post-exercise HR since the parasympathetic nervous system has the greatest influence on HR (Iellamo et al., 1999). At present, the underlying mechanisms that explain how exercise training improves vagal tone are speculative (Castello et al., 2010). However, it has been proposed that an improved BRS is the primary regulatory mechanism that explains a decrease in HR (Yamamoto et al., 2001). A decrease in post-exercise HR was attributable to improvements in BRS after AE training in a previous study on obese women (Figueroa et al. 2007). It is likely that the short duration of the study and the small sample size may explain the unapparent effect of WBVT on HF power of HRV (index of cardiac parasympathetic activity).

Strength

Consistent with a previous study (Delecluse et al., 2003), the current study found a significant increase in knee-extensor strength (11.8 ± 7.0 kg) following WBVT. The current study therefore emphasizes that WBVT is an efficient alternative to standard exercise programs to improve the strength of the knee-extensors in untrained females (Roelants et al., 2004). Although the present study did not find changes in body composition, the results of improved muscle strength are similar to other RE training studies (Bosco et al., 1998). Van Der Heijden et al. (2010) found that a 12 week progressive low-moderate intensity RE training program on sedentary adolescent obese individuals improved muscle strength, and consistent with the current study, found this change without any change in total body fat. Another study on RE training with overweight postmenopausal women found significant improvements in muscle strength after 8 weeks at low intensity RE training without changes in body mass, percentage of body fat, or BMI (Elliott et al., 2002). The increase in muscle strength may be explained by the well known neural adaptations that typically occur in the first two months of RE training. Following a high-intensity RE training program, an enhancement in the performance of leg extensors is associated with improvements in neuromuscular adaptations induced by the increased activity of higher motor centers. Also, gains in muscle strength are credited to increased synchronization

between the motor cortex and the spinal motor neurons after training (Milner-Brown et al., 1975). In addition, increased activity of the motor cortex induces the secretion of anabolic hormones that have been associated with improvements neuromuscular function, muscle strength, and mass (Cardinale & Bosco, 2003). These mechanisms may explain the increases in strength that occurred through this study.

A past study on endocrine control after vibration exposure has shown that vibration increases activity of central motor command, which is associated with an exaggerated activation of the pituitary-adrenocortical axis. Increased activation of the pituitary-adrenocortical complex is associated with an increased production of growth hormone (Kajaer et al., 1987), which has been demonstrated to be associated with increases in muscle mass and strength (Godfrey et al., 2003). This study however did not find changes in strength that can be attributed to increases in lean muscle mass. It can be suggested that a longer training duration is necessary to increase lean mass since the shortest training study to show increases in lean mass is 8 weeks (Coburn et al., 2006).

CHAPTER 6

CONCLUSION

The present study was carried out in young normotensive overweight-obese but otherwise healthy women. Participants underwent a 6 week period of WBVT and a 6 week CON period. In this study, the hypotheses that were tested were that: 1) WBVT decreases resting AIx, PWV, and BP after the treatment at rest and after an acute bout of WBV exercise compared to CON; 2) WBVT decreases levels of cardiac sympathetic activity, increases levels of parasympathetic activity, and decreases HR after the treatment at rest and after an acute bout of WBV exercise compared to CON; 3) WBVT improves body composition with decreases in fat mass and increases in lean mass after the treatment compared to CON; and 4) WBVT improves muscle strength after the treatment compared to CON. Following 6 weeks of WBVT, there are decreases in brachial SBP and DBP at rest and also decreases in brachial SBP, MAP, and HR after an acute bout of WBV. Also, it was found that muscle strength increased following WBVT training without changes in muscle mass. These improvements show that 6 weeks of WBVT is a useful method for improving fitness parameters similar to RE and AE training for improving cardiovascular and strength parameters. Although these changes were small, they were found to be statistically significant ($p < 0.05$). Further research aimed to identify the training effects of this exercise mode are needed before implications can be suggested in regards to its usefulness in clinical settings.

Limitations

A major limitation of this study was the small sample size. Only 8 women successfully completed the study. The small sample size disallowed more significant data to emerge from the analysis that may have better explained the physiological changes that occurred. Another limitation may have been that the exercise training volume was not high enough to elicit

adaptations. Research involving traditional exercise modes incorporates considerably longer training session durations for the training protocols. The short duration for the training sessions may have limited the potential benefits that can occur through this type of exercise.

Future Research

This study is the first to show that 6 weeks of combined dynamic and static WBVT elicits reductions peripheral BPs in overweight-obese premenopausal women. It may be beneficial for future research to target populations at a greater risk for cardiovascular complications. The current study, targeted apparently healthy young women who were free of chronic diseases and fell into a BMI category of >25 kg/m^2. Populations with physiological complications, such as elevated BP, may elicit greater responses to this type of exercise training. In addition, there is scarce knowledge on the acute and chronic cardiovascular effects of WBV on populations with chronic diseases. Therefore, additional studies on this exercise mode are needed to recommend its use for clinical therapy.

In addition, this project opened future research to investigate the cardiovascular physiological mechanisms that follow WBVT. Measurable substances in the blood such as leptin, C-reactive protein, NO, and other endothelial factors should be considered valuable to explain the cardiovascular benefits of this exercise mode.

APPENDIX A

HUMAN SUBJECTS COMMITTEE APPROVAL EMAIL

Use of Human Subjects in Research - Approval Memorandum

Office of the Vice President For Research Human Subjects Committee
Tallahassee, Florida 32306-2742
(850) 644-8673. FAX (850) 644-4392

APPROVAL MEMORANDUM

Date: 9/25/2009
To: Arturo Figueroa-Galvez
Address: 1493
Dept.: NUTRITION FOOD AND MOVEMENT SCIENCES
From: Thomas L. Jacobson, Chair
Re: Use of Human Subjects in Research
The Effect of Whole Body Vibration Training on Cardiovascular and Autonomic Function in Obese Individuals

The application that you submitted to this office in regard to the use of human subjects in the research proposal referenced above has been reviewed by the Human Subjects Committee at its meeting on 09/09/2009. Your project was approved by the Committee.

The Human Subjects Committee has not evaluated your proposal for scientific merit, except to weigh the risk to the human participants and the aspects of the proposal related to potential risk and benefit. This approval does not replace any departmental or other approvals, which may be required.

If you submitted a proposed consent form with your application, the approved stamped consent form is attached to this approval notice. Only the stamped version of the consent form may be used in recruiting research subjects.

If the project has not been completed by 9/8/2010 you must request a renewal of approval for continuation of the project. As a courtesy, a renewal notice will be sent to you prior to your expiration date; however, it is your responsibility as the Principal Investigator to timely request renewal of your approval from the Committee.

You are advised that any change in protocol for this project must be reviewed and approved by the Committee prior to implementation of the proposed change in the protocol. A protocol change/amendment form is required to be submitted for approval by the Committee. In addition, federal regulations require that the Principal Investigator promptly report, in writing any unanticipated problems or adverse events involving risks to research subjects or others.

By copy of this memorandum, the Chair of your department and/or your major professor is reminded that he/she is responsible for being informed concerning research projects involving human subjects in the department, and should review protocols as often as needed to insure that the project is being conducted in compliance with our institution and with DHHS regulations.

This institution has an Assurance on file with the Office for Human Research Protection. The Assurance Number is IRB00000446.

Cc: Bahram Arjmandi, Chair
HSC No. 2009.3195

APPENDIX B

IRB APPROVAL: CONSENT FORM

Effects of whole-body vibration training on cardiovascular and autonomic function in overweight/obesity

INFORMED CONSENT FORM

1. I voluntarily and without element of force or coercion, consent to be a participant in the research study entitled "the effect of whole body vibration training on cardiovascular and autonomic function in obese individuals." This study is being conducted by Arturo Figueroa, M.D., Ph.D. and Ryan Gil, B.S. who are associated with Florida State University in the Department of Nutrition, Food & Exercise Sciences.

2. The purpose of the study is to evaluate the chronic effects of whole body vibration (WBV) exercise on cardiovascular and autonomic function at baseline, following 6 weeks of exercise training with or without WBV, and following 4 weeks of detraining in individuals with overweight and obesity defined as body mass index (BMI) between 25 and 39.9 kg/m^2. Ten men and ten women 18-35 years of age with resting blood pressure lower than 160/100 mmHg (stage1-hypertension) will be recruited in this study.

3. My participation in this study will require coming to the Cardiovascular Physiology Laboratory at the Florida State University on six different days to complete the experiments described below.

On the first visit, I will be oriented to the study, answer questions on my medical history, and to sign an informed consent if I qualify. I will have my blood pressure, blood flow, maximal muscle strength (maximal weight that I can lift through a full range of motion for the leg extension exercise), height, weight, waist circumference, body composition (DEXA scan) and three skinfolds measured (chest, abdomen and thigh). The first visit should take approximately one hour.

On visit 2 to the laboratory, a blood sample (about 4 tablespoons) will be drawn from my vein in my forearm to measure several markers of vascular function (leptin, homocysteine, C-reactive protein, endothelin-1, and adiponectin) by an experienced phlebotomist. On the second visit, I will have my cardiovascular function evaluated after 20 min of lying down on a tilt table in the horizontal position. A total of 4 cuffs, one in each extremity (around arm and ankles) and 2 tonometers (sensors applied to the skin to obtain pulse waves), one on the neck and the second on the inner thigh, will be used to measure pulse wave velocity (arterial stiffness). My blood pressure will be also monitored by placing a small cuff around the middle finger and a tonometer on the wrist. Eleven electrodes will be positioned on the skin of my chest, neck, lower back, and forearms to measure heart rate (electrocardiogram) and stroke volume (volume pumped out by the heart in each beat). Following the resting measurements, the table will be tilted up to an almost standing position. Following the tilt test, I will undergo an exercise cycling test until volitional fatigue to determine my cardiorespiratory capacity. After the test, I will return to the supine position for additional 35 min to have my cardiovascular function measured at 5, 15, and 30 min of recovery. The second visit will take approximately 1.5 hour.

On the third and fourth visits (6-week training period), and the fifth and sixth visits (end of 4-week detraining period), I will undergo all measurements conducted on visits 1 and 2.

I will be attending WBV exercise training sessions of 15-30 min duration, 3 times a week for a period of 6 weeks. I will perform static and dynamic leg exercises including bilateral and one-leg squat, lunges, and calf raises on the vibration platform. The dynamic exercises will be performed with slow controlled movements (3 seconds down and 3 seconds up) starting from an upright position into a 60 degree knee flexion (squat and lunges) and maximal heel elevation (toe-stand). The static exercises will be performed without movement with the joint angles described previously (60˚ knee flexion and maximal heel elevation). The training volume will be increased progressively over the 6-week training period by increasing duration of the exercise set (30-60 sec), duration of rest periods (30-60 sec), and total duration of the training session (15-30 min). In the last 2 weeks, external resistance will be added progressively by the use of a weighted vest that will not exceed 20% of my body weight. In the whole body vibration group, the intensity of vibration and amplitude will also be increased progressively (25-30 Hz of frequency and 2.5-5.0 mm of amplitude).

FSU Human Subjects Committee approved on 9/25/09 VOID after 9/8/2010 HSC# 2009.3195

I will not be involved in any other exercise program or make changes in my lifestyle during the 10-week study.

4. I understand there is a possibility of a minimal level of risk involved if I agree to participate in this study. I may experience some muscle soreness from the vibration training sessions. The risks associated with vibration training are minimal and the selected amplitudes have been previously used in other studies using human subjects. There is the possibility of muscle fatigue or soreness related with exercise or testing. The risk will be minimized by using trained technicians and by strict supervision of the training and testing sessions. I will complete a medical history before I can participate in the study. I am aware that I cannot participate in this study if I answered yes to any of the exclusion criteria. I understand that to reduce muscle fatigue and soreness my trainer will make adjustments to my training program.

5. The possible benefits of my participation in this study include learning about blood pressure and heart rate control and arterial function during and after WBV exercise. I will also be given a number of training sessions and tests at no charge.

6. The result of this study may be published but my name or identity will not be revealed. Information obtained during the course of the study will remain confidential, to the extent allowed by law. My name will not appear on any of the results. No individual responses will be reported. Only group responses will be reported in the publications. Confidentiality will be maintained by assigning each subject a code number and recording all data by code number. The only record with the subject's name and code number will be kept by the investigator, Dr. Arturo Figueroa, in a locked drawer in his office. Data will be kept for 10 years and then destroyed.

7. I will not be paid for my participation in this study. In case of an injury, first aid (free of charge) will be provided to me by the laboratory personnel working on the research project. However, any other treatment or care will be provided at my expense.

8. Any questions I have concerning the research study or my participation in it, before or after my consent, will be answered by the investigators or they will refer me to a knowledgeable source. I understand that I may contact Ryan Gil at and Dr. Figueroa (afiguero@fsu.edu) at 850-644-8089 for answers to questions about this research study or my rights. Group results will be sent to me upon my request.

9. In case of an injury, or if I have questions about my rights as a subject/participant in this research, or I feel I have been placed at risk, I can contact the chair of the human subject committee (humansubjects@magnet.fsu.edu), Institutional Review Board, through the office of the Vice President of Research at (850) 644-8633.

10. The nature, demands, benefits and risks of the study have been explained to me. I knowingly assume any risk involved.

11. I have read the above informed consent form. I understand that I may withdraw my consent and discontinue participation at any time without penalty or loss of the benefits to which I may otherwise entitled. In signing this consent form, I am not waiving my legal claims, rights or remedies.

_____

Subject Date

Approved

APPENDIX C

IRB APPROVAL: HEALTH HISTORY QUESTIONNAIRE

CARDIOVASCULAR HISTORY

ID# _____
DATE _____

Answer the following questions, indicating the month and year of the event or diagnosis where appropriate.

		Yes	No	Month/Year
1.	Has a doctor ever told you that you have heart disease?	___	___	____/____
2.	Have you ever had a heart attack?	___	___	____/____
3.	Have you ever had chest pain?	___	___	____/____
4.	Have you ever had cardiac catheterization?	___	___	____/____
5.	Have you ever had balloon angioplasty?	___	___	____/____
6.	Have you had coronary artery bypass graft surgery?	___	___	

If yes, list date and number of grafts:

____/____ # grafts: ___ 1 ___ 2 ___ 3 ___ 4^{+}
Mo. Yr.

7.	Have you ever had a stroke?	___	___	____/____
8.	Do you have hypertension (high blood pressure)?	___	___	____/____

If yes, how long have you had hypertension?

_____ less than 1 year
_____ 1-5 years
_____ 6-10 years
_____ more than 10 years

9. Do you have diabetes mellitus? ___ ___ ____/____

Yes No Month/Year

10. Do you take insulin for diabetes? ___ ___
If yes, how long have you taken insulin?

_____ less than 1 year
_____ 1-5 years
_____ 6-10 years
_____ more than 10 years

11. Do you take oral hypoglycemics for diabetes? ___ ___
12. Do you have a cardiac pacemaker? ___ ___

If yes, how long have you had a cardiac pacemaker?
_____ less than 1 year
_____ 1-5 years
_____ 6-10 years
_____ more than 10 years

13. Have you had a carotid endarterectomy? ___ ___ ____/____

14. Has your doctor ever told you that you have a heart valve problem? ___ ___ ____/____

15. Have you had heart valve replacement surgery? ___ ___ ____/____

If yes, what heart valves were replaced? ___ mitral ___ aortic

16. Have you had cardiomyopathy? ___ ___ ____/____
17 Have you had a heart aneurysm? ___ ___ ____/____
18. Have you had heart failure? ___ ___ ____/____
19. Have you ever suffered cardiac arrest? ___ ___ ____/____
20. Do you smoke? Yes ___ No ___
If yes, how long have you smoked _____ years
How many cigarettes per day _____ per day
If you stopped smoking, when did you do it? _____ years ago

21. OTHER MEDICAL PROBLEMS: Indicate if you have had any of the following medical problems:

Past	Now	
___	___	Alcoholism
___	___	Allergies
___	___	Anemia
___	___	Arthritis
___	___	**Asthma**
___	___	Back injury or problem
___	___	Blood clots

___ ___ Bronchitis
___ ___ Cirrhosis
___ ___ Claudication
___ ___ Elbow or shoulder problems
___ ___ Emotional disorder
___ ___ Eye problems
___ ___ Gall bladder disease
___ ___ Glaucoma
___ ___ Gout
___ ___ Headaches/Migraines
___ ___ Hemorrhoids
___ ___ Hernia
___ ___ Herpes simplex
___ ___ Hip, knee, or ankle problems
___ ___ Intestinal disorders
___ ___ Kidney disease
___ ___ Liver disease
___ ___ Lung disease
___ ___ Mental illness
___ ___ Neck injury or problem
___ ___ Neuralgic disorder
___ ___ OB/GYN problems
___ ___ Obesity/overweight
___ ___ Osteoporosis
___ ___ Parkinson's disease
___ ___ Phlebitis
___ ___ Prostate trouble
___ ___ Rheumatic fever
___ ___ Seizure disorder
___ ___ Stomach disease
___ ___ Thyroid disease
___ ___ Tumors or cancer - List type: _____
___ ___ Ulcers
___ ___ Other - specify: _____

List medications you are taking below:

Name of Drug	Dosage	Times/day	Duration of drug use
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

APPENDIX D

ADVERTISEMENT

A New Type of Exercise

The Cardiovascular Laboratory of the College of Human Sciences is beginning a research project with a new exercise mode, the *whole-body vibration*.

Availability:	18 Volunteer Spots Open
Criteria:	Female, Nonsmoking
	Overweight/obese
	18-35 yrs of age
Measurements:	Aortic pressure, Arterial Function, and Blood Pressure Control Anthropometry and Densitometry (DEXA Scan)
Benefits:	Free supervised training 3 days/wk for 6 weeks Improved cardiovascular health and muscle strength

Contact: Ryan Gil

REFERENCES

1. **Alvarez GE, Beske SD, Ballard TP, Davy KP**. Sympathetic neural activation in visceral obesity. Circulation 106:2533–2536, 2002.

2. **Alverez GE, Davy BM, Ballard Tp, Beske SD, and Davy KP.** Weight loss increases cardiovagal baroreflex function in obese young and older men. *Am J Physiol Endocrinol Metab. 289(4) E665-9, 2005.*

3. **Amano M, Kanda T, Ue H, Moritani T**. Exercise training and autonomic nervous system activity in obese individuals. *Med Sci sports and Exerc 2001; 33: 1287–1291.*

4. **American College of Sports Medicine Position stand.** Exercise and physical activity for older adults. *Med Sci Sports Exerc. 30: 992-1008, 1998.*

5. **Anderson TJ, Uehata A, Gerhard MD, Mededith IT, Knab S, Delagrange D, Lieberman EH, Ganz P, Creager MA, Yeung AC et al.** Close relation of endothelial function in the human coronary and peripheral circulations. *J Am Coll Cardiol 26:1235-1241, 1995.*

6. **Bazrgari BA, Shirazi-Adl, and Kasra M.** Seated whole body vibrations with high-magnitude accelerations-relative roles of inertia and muscle forces. *J Biomech 41(12)L 2639-2646, 2008.*

7. **Bertovic DA, Waddel TK, Gatzka CD, Cameron JD, Dart AM, and Kingwell BA.** Muscular strength training is associated with low arterial compliance and high pulse pressure. *Hyperten 33: 1385-1391, 1999.*

8. **Blacher J, Asmar R, Djane S, London GM, and Safar ME.** Aortic Pulse Wave Velocity as a Marker of Cardiovascular Risk in Hypertensive Patients. *Hyperten 33: 1111-1117, 1999.*

9. **Bogaerts A, Delecluse C, Classen AL, Coudyzer W, Boonen S, and Verschueren SM.** Impact of whole-body vibration training versus fitness training on muscle strength and muscle mass in older men: a 1-year randomized controlled trial. *J Gerontol A: Biol Sci Med Sci 62: 630-63, 2007.*

10. **Bosco C, Cardinale M, Tsarpela O, Colli R, Tihanyi J, Von-Duvillard SP, and Viru A.** The influence of whole body vibration on jumping performance. *Biol. Sport 15:157-164, 1998.*

11. **Bray GA.** Pathophysiology of obesity. *Am J Clin Nutr 55: 488S-494S, 1992.*

12. **Breslow JL.** Cardiovascular disease burden increases, NIH funding decreases. *N Engl J Med 337: 1360-1369, 1997.*

13. **Breuer H-W M, Skyschally A, Schulz R, Martin C, Wehr M, and Heusch.** Heart rate variability and circulation catecholamine concentrations during steady state exercise in healthy volunteers. *Br Heart f 70: 144-149, 1993.*

14. **Cameron J and Dart A.** Exercise training increases total systemic arterial compliance in humans. *Am J Physio 266, H693-H701, 1994.*

15. **Cardinale M and Bosco C.** The use of vibration as an exercise intervention. *Exerc Sport Sci Rev 31: 3-7, 2003.*

16. **Casey DP, Beck DT, and Braith RW.** Progressive resistance training without volume increases does not alter arterial stiffness and aortic wave reflection. *Exp Biol Med 232(9): 1228-1235, 2007.*

17. **Castello V, Simoes RP, Bassi D, Catai AM, Arena R, and Borghi-Silva A.** Impact of aeorobic exercise training on heart rate variability and functional capacity in obese women after gastric bypass surgery. *Obes Surgery: In Press, 2010.*

18. **Coburn JW, Housh TJ, Malek MH, Beck TW, Cramer JT et al.** Effects of leucine and whey protein supplementation during eight weeks of unilateral resistance training. *J Strength Cond Res 20(2): 2840291, 2006.*

19. **Cochrane DJ, Sartor F, Winwood K, Stannard SR, Narici MV, and Rittweger J.** A comparison of physiologic effects of acute whole body-vibration exercise on young and older people. *Arch Phys Med Rehabil 89:815-821, 2008.*

20. **Collier SR, Kanaley JA, Carhart R Jr, Frechette V, Tobin MM, Hall AK, Luckenbaugh AN, and Fernhall B.** Effect of 4 weeks of aerobic or resistance exercise training on arterial stiffness, blood flow, and blood pressure in pre- and stage-1 hypertensives. *J of Human Hyperten 22: 678-685, 2008.*

21. **Cole CR, Blackstone EH, Pashkow FJ, Snader CE, and Lauer MS.** Heart-rate recovery immediately after exercise as a predictor of mortality. *N Engl J Med 341: 1351-7, 1999.*

22. **Cook NR, Cohen J, Herbert PR, Taylor JO, and Hennekens CH.** Implications of small reductions in diastolic blood pressure for primary prevention. *Arch Inter Med 155: 701-709, 1995.*

23. **Cortez-Cooper MY, Devan AE, Anton MM, Farrar RP, Beckwith KA, Todd JS, and Tanaka H.** Effects of high intensity resistance training on arterial stiffness and wave reflection in women. *Amer J of Hyperten 18: 930-934, 2005.*

24. **Crouse JR III, Craven TE, Hagaman AP, and Bond MG.** Associations of coronary disease with segment specific intimal-medial thickenin of the extracranial carotid artery. *Circ 92: 1141-1147, 1995.*

25. **Dekker JM, Girman C, Rhodes T, et al. Metabolic syndrome and 10-yr cardiovascular disease in the Hoorn Study.** *Circulation 112: 666-673, 2005.*

26. **Delecluse C, Roelants M, and Verschueren S.** Strength increase after whole-body vibration compared with resistance training. *Med Sci Sports Exerc 35(6): 1033-41, 2003.*

27. **Delp MD and Laughlin MH**. Time course of enhanced endothelium-mediated dilation in aorta of trained rats. *Med Sci Sports Exerc* 29: 1454–1461, 1997.

28. **Edwards DG, Schofield RS, Magyari PM, Nichols WW, and Braith RW.** Effect of exercise training on central aortic pressure wave reflection in coronary artery disease. *AJH 17: 540-543, 2004.*

29. **Edwards DG and Lang JT.** Augmentation index and systolic load are lower in competitive endurance athletes. *Am J Hypertens 18: 679-683, 2005.*

30. **Elliott KJ, Sale C, Cable NT.** Effects of resistance training and detraining on muscle strength and blood lipid profiles in postmenopausal women. *Br J Sports Med 36(5):340-344, 2002.*

31. **Emdin M, Gastaldelli A, Muscelli E, Macerata A, Natali A, Camastra S et al.** Hyperinsulinemia and autonomic nervous system dysfunction in obesity: effects of weight loss. *Circ 103L 513-519, 2001.*

32. **Feigenbaum MS and Pollock ML.** Prescription of resistance training for health and disease. *Med Sci Sports Exerc 31: 38-45, 1999.*

33. **Figueroa A, Baynard T, Fernhall Bo, Carhart R, and Kanaley JA.** Endurance training improves post-exercise cardiac autonomic modulation in obese women with and without type 2 diabetes. *Euro J Appl Physiol 100: 437-444, 2007.*

34. **Figueroa A, Gil R, Sanchez-Gonzalez MA.** Whole-body vibration attenuates the increase in leg arterial stiffness and aortic blood pressure during post-exercise muscle ischemia. *Euro J Appl Physiol: In Press, 2010.*

35. **Figueroa A, Trivino JA, Sanchez-Gonzalez MA, and Vicil F.** Oral L-Citrulline Suppplementation attenuates blood pressure response to cold pressor test in young men. *Amer J of Hypertension 23: 12-16, 2010.*

36. **Frick, M. H., A. Konttinen, and S. Sarajas.** Effects of physical training on circulation at rest and during exercise. Am. J. Cardiol. 12: 142–150, 1963.

37. **Furness TP, Maschette WE, Lorenzen C, Naughton GA, and Willianms MD.** Efficacy of a Whole-Body Vibration Intervention on Functional Performance of Community-Dwelling Older Adults. *J Altern Complement Med. 16(7): 795-7, 2010.*

38. **Godfrey RJ, Madgwick Z and Whyte GP.** The exercise-induced growth hormone response in athletes. *Sports Med. 33: 599-613, 2003.*

39. **Gokce N, Vita JA, Bader DS, Sherman DL, Hunter LM, Holbrook M, O'Malley C, Keaney JF Jr, and Balady GJ.** Effect of exercise on upper and lower extremity endothelial function in patients with coronary artery disease. *Am J Cardiol 90: 124-127, 2002.*

40. **Gordon T and Kannel WB.** Obesity and cardiovascular disease: the Framingham study. *Clin Endocrinol Metab. 5: 367-375, 1976.*

41. **Grassi G, Dell'Oro R, Facchini A, Trevano FQ, Bolla GB, and Mancia G.** Effect of central and peripheral body fat distribution on sympathetic and baroreflex function in obese normotensives. *J of Hyperten 22: 2363-2369, 2004.*

42. **Grassi G, Seravalle G, Cattaneo BM, Bolla GB, Lamfranchi A, Colombo M, et al.** Sympathetic activation in obese normotensive subjects. *Hyperten 25: 560-563, 1995.*

43. **Hagberg, J., D. Goldring, A. Ehsani, et al.** Effect of exercise training on the blood pressure and hemodynamic features of hypertensive adolescents. *Am. J. Cardiol. 52: 763–768, 1983.*

44. **Hazell TJ, Thomas GWR, Deguire JR, and Lemon PWR.** Vertical whole-body vibration does not increase cardiovascular stress to static semi-squat exercise. *Euro J Appl Physiol 104: 903-908, 2008.*

45. **Hickner RC, Kemeny G, Stallings HW III, Manning SM, and KcIver KL.** Relationship between body composition and skeletal muscle eNOS. *Intern J of Obes 30: 308-312, 2006.*

46. **Higashi Y, Sasaki S, Sasaki N, Nakagawa K, Ueda T, Yoshimizu A, Kurisu S, Matsurura H, Kajiyama G, and Oshima T.** Daily aerobic exercise improves reactive hyperemia in patients with essential-hypertension. *Hyperten 33:591-597, 1999.*

47. **Higashi Y and Yoshizumi M.** Exercise and endothelial function: role of endothelium-derived nitric oxide and oxidative stress in healthy subjects and hypertensive patients. *Pharmacol Ther 102: 87-96, 2004.*

48. **Iellamo F, Pizzinelli P, Massaro M, Raimondi G, Peruzzi G, and Legramante JM.** Muscle metaboreflex contribution to sinus node. *Circ 100: 27-32, 1999.*

49. **Irigoyen M-C, Paulini J, Flores LJF, Flues K, Bertagnolli M, Moreira ED, Consolim-Colombo F, Bello-Klein A, and De Angelis K.** Exercise training improves baroreflex sensitivity associated with oxidative stress reduction in ovariectomized rats. *Hypertension 46(4): 998-1003, 2005.*

50. **Jonge L, Moreira EA, Martin CK, Ravussin E, and the Pennington CALERIE Team.** Impact of six-month caloric restriction on autonomci nervous system activity in healthy, overweight, individuals. *Obesity 18 (2) : 414-416, 2010.*

51. **Kamath MV, Fallen EL, and Mckelvie R.** Effects of steady state exercise on the power spectrum of heart rate variability. *Med. Sci. Sports Exerc. 23: 428-434, 1991.*

52. **Kanaley JA, Goulopoulou S, Franklin RM, Baynard T, Holmstrup ME, Carhart Jr R, Weinstock RS, and Fernhall.** Plasticity of heart rate signaling and complexitiy with exercise training in obese individuals with and without type 2 diabetes. *Int Journ of Obes 33: 33, 1198-1206, 2009.*

53. **Kaushal P and Taylor JA.** Inter-relations among decline in arterial distensibility, baroreflex function and respiratory sinus arrhythmia. *J Am Coll Cardiol. 39(9):1524-1530, 2002.*

54. **Kawanabe K, Kawashima A, Sashimoto I, Takeda T, Sato Y, and Iwamoto J.** Effect of whole-body vibration exercise and muscle strengthening, balance, and walking exercises on walking ability in the elderly. *Keio J Med. 56(1): 28-33, 2007.*

55. **Kelly AS, Wetzsteon RJ, Kaiser DR, Steinberger J, Bank AJ, and Dengel DR.** Inflammation, insulin, and endothelial function overweight children and adolescents: the role of exercise. *J Pediatr 145: 731-736, 2004.*

56. **Kerschan-Schindl K, Grampp S, Henk C et al.** Whole-body vibration exercise leads to alterations in muscle blood volume. *Clin Physiol 21:377-382, 2001.*

57. **Kingsley JD, McMillan V, and Figueroa A.** The effects of 12 weeks of resistance exercise training on diseases severity and autonomic modulation at rest and after acute leg resistance exercise in women with fibromyalgia. *Arch Phys Med Rehabil 91(10): 1551-7, 2010.*

58. **Ko MJ, Kim MK, Shin J, and Choi.** Relations of pulse wave velocity to waist circumference independent of hip circumference. *Epidemiol Health 32: 1-7, 2010.*

59. **Kjaer M, Secher NH, Bach FW, Sheikh S, and Galbo H.** Hormonal and metabolic responses to exercise in humans: effect of sensory nervous blockade. *Am J Physiol 257: E95-E101, 1987.*

60. **Kraemer WJK, Adams E, Cafarelli E et al.** Amercian College of Sports Medicine position stand. *Ed Sci. Sports Exerc. 34: 364-380, 2002.*

61. **La Rovere MT, Bersano C, Gnemmi M, Specchia G, and Schwartz PJ.** Exercise-induced increase in baroreflex sensitivity predicts improved prognosis after myocardial infarction. *Circ 106: 945-949, 2002.*

62. **Laurent S, Boutouyrie P, Asmar R, Gautier I, Laloux B, Guize L, Ducimetiere P, and Benetos A.** Aortic stiffness is an independent predictor of all-cause and cardiovascular mortality in hypertensive patients. *Hypertension 37: 1236-1241, 2001.*

63. **Levy MN, Schwartz PJ, eds.** *Vagal Control of the Heart: Experimental Basis and Clinical Basis and Clinical Implications. Armonk, NY: Futura, 1994.*

64. **Leyva F, Goldland IF, Ghatei M, Proudler AJ, Aldis S, Walton C, Bloom S, and Stevenson JC.** Hyperleptinemia as a component of a metabolic syndrome of cardiovascular risk. *Arterioscler Thromb Vasc Biol 18: 928-933, 1998.*

65. **Li M, Wu CY, Zhan ZW, Yang J, Zhang K, Li XG, Gan LH, and Ji BH.** Role of leptin in the pathogensis of obesity-related hypertension. *Zhonghua Yi Zue Za Zhi 83(12) 1058-1061, 2003.*

66. **Liomaala A, Huikuri HV, Koobi T, Rinne M, Nenonen A, and Vuori I.** Exercise training improves baroreflex sensitivity in type 2 diabetes. *Diabetes 52(7): 1837-1842, 2003.*

67. **Lohman EB, Petrofsky JS, Maloney-Hinds C, Betts-Schwab H, and Thorpe D.** The effect of whole body vibration on lower extremity skin blood flow in normal subjects. *Med Sci Monit 13: CR71-CR76, 2007.*

68. **Mack GW, Shi XG, Nose H, Tripathi A, Nadel ER.** Diminished baroreflex control of forearm vascular resistance in physically fit humans. *J Appl Physiol. 63: 105–110, 1987.*

69. **Maher V, O'Dowd M, Carey M, Markham C, Byrne A, Hand E, and Mc Inerney D.** Association of central obesity with early Carotid intima-media thickness is independent of that from other risk factors. *Int Journal of Obes 33: 136-143, 2009.*

70. **Maeda S, Tanabe T, Otsuki T, Sugawara J, Ajisaka R, and Matsuda M.** Acute exercise increases systemic arterial compliance after 6-month exercise training in older women. *Hypertens Res 31: 377-381, 2008.*

71. **McGowan CL, Visocchi A, Faulkner M, Verduyn R, Rakobowchuk M, Levy AS, McCartney N, and MacDonald MJ.** Isometric handgrip training improves local flow-mediated dilation in medicated hypertensives. *Euro J Apply Physiol 99: 227-234, 2007.*

72. **Milner-Brown HS, Stein RB, and Lee RG.** Synchronization of human motor units: possible roles of exercise and supraspinal reflexes. *Electroencephalogr Clin Neurophys 38: 245-254, 1975.*

73. **Miyachi M, Kawano H, Sugawara J, Takahashi K, Hayashi K, Yamazaki K, Tabata I, and Tanaka H.** Unfavorable effects of resistance training on central arterial compliance. *Circ 110: 2858-2863, 2004.*

74. **Miyachi M, Tanaka H, Yamamoto K, et al.** Greater age-related reductions in central arterial compliance in resistance-trained men. *Hyperten 41: 130-135, 2009.*

75. **Monahan KD, Tanaka H, Dinenno FA, et al.** Central arterial compliance is associated with age- and habitual exercise-related differences in cardiovagal baroreflex sensitivity. *Circ 104L 1627-1632, 2001.*

76. **Ness LL and Field-Fote EC.** Whole-Body vibration improves walking function in individuals with spinal cord injury: A pilot study: *Gait & Posture: Article In Press, 2009.*

77. **Niebauer JJ and P Cooke.** Cardiovascular effects of exercise: role of endothelial shear stress. *J Am Cardiol. 28: 1652-1660, 1996.*

78. **Olson TP, Dengel DR, Leon AS, and Schmitz KH.** Moderate resistance training and vascular health in overweight women. *Med Sci Sports Exerc 38(9): 1558-1564, 2006.*

79. **O'Rourke MF et al.** Clinical applications of arterial stiffness; definitions and reference value. *Am J Hyperten 15: 426-444, 2002.*

80. **Otsuki T, Maeda S, Iemitsu M, Saito Y, Tanimura Y, Ajisaka R, and Miyauchi T.** Systemic arterial compliance, systemic vascular resistance, and effective arterial elastance during exercise in endurance-trained men. *Am J Physiol Regul Comp Physiol 1: R228-35, 2008.*

81. **Otsuki T, Takanami Y, Aoi W, Kawai Y, Ichikawa H, and Yoshikawa T.** Arterial stiffness acutely decreases after whole-body vibration in humans. *Acta Physiol 194: 189-194, 2008.*

82. **Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, Pizzinelli P, et al.** Power spectral analysis of heart rate and arterial pressure variabilities as marker of sympathovagal interaction in man and conscious dog. *Circ Res 59: 178-92, 1986.*

83. **Pagkalos M, Koutlianos N, Kouidi E, Pagkalos E, ManDroukas K, and Deligiannis A.** Heart rate variability modifications following exercise training in type 2 diabetic patients with definite cardiac autonomic neuropathy. *Br J Sports Med 42: 47-54, 2008.*

84. **Panza JA, Casino PR, Kilcoyne CM, and Quyyumi AA.** Role of endothelium-derived nitric oxide in the abnormal endothelium-dependent vascular relaxation of patients with essential-hypertension. *Circ 87(5): 1468-1474, 1993.*

85. **Paolisso G, Manzella D, Montano N, Gambardella A, Varricchio M.** Plasma leptin concentrations and cardiac autonomic nervous system in healthy subjects with different body weights. *J Clin Endocrinol Metab 85:1810–1814, 2000.*

86. **Poehlman ET, Gardner AW, Goran MI, Arciero PJ, Toth MJ, Ades PA, and Calles-Escandon J.** Sympathetic nervous system activity, body fatness, and body fat distribution in younger and older males. *J of App Phys 78 I3: 802-806, 1995.*

87. **Rakobowchuk M, McGowan CL, de Groot PC, Bruinsma D, Hartman JW, Phillips SM, and MacDonald MJ.** Effect of whole body resistance training on arterial compliance in young men. *Exp Physiol 90: 645-651, 2005.*

88. **Rajkumar C, Cameron JD, Christophidis N, Jennings GL, and Dart AM.** Reduced systemic arterial compliance is associated with left ventricular hypertrophy and diastolic dysfunction in older people. *J Am Geriatr Soc 45: 803-808, 1997.*

89. **Ray CA and Carrasco DI.** Isometric handgrip training reduces arterial pressure at rest without changes in sympathetic nerve activity. *Am J Physiol Heart Circ Physiol 279: H245-H249, 2000.*

90. **Rechlin T, Weis M, Ott C, Blechner F, Joraschky P.** Alterations of autonomic cardiac control in anorexia nervosa. *Biological Pyschiatry 43: 358-363, 1998.*

91. **Roelants M, Delecluse C, Goris M, and Verschueren.** Effects of 24 weeks of whole body vibration training on body composition and muscle strength in untrained females. *Int J Sports Med 25(1): 1-5, 2004.*

92. **Ronnestad BR.** Comparing the performance-enhancing effects of squats on a vibration platform with conventional squats in recreationally resistance-trained men. *J Strength Cond. Res 18: 839-845, 2004.*

93. **Ross R.** Atherosclerosis- an inflammatory disease. *New Engl J Med 340: 115- 126, 1999.*

94. **Rubin C, Recker R, Cullen D, Ryaby J, McCabe J, and McLeod K.** Prevention of postmenopausal bone loss by a low-magnitude, high-frequency mechanical stimuli: a clinical trial assessing compliance, efficacy, and safety. *J Bone Miner Res 19: 343-351, 2004.*

95. **Runge M, Rehfeld G, and Resnicek E.** Balance training and exercise in geriatric patients. *J Musculoskelet Neuronal Interact 1: 61-65, 2000.*

96. **Saeki A, Recchia F, Kass DA.** Systolic flow augmentation in hearts ejecting into a model of stiff aging vasculature. Influence on myocardial perfusion-demand balance. *Circ Res 76:132-141, 1995.*

97. **Schlaich MP, Socratous F, Hennebry S, Eikelis N, Lambert EA, Straznicky N, Esler MD, and Lambert GW.** Sympathetic activation in chronic renal failure. *J Am Soc Nephrol 20: 933-939, 2009.*

98. **Sessa WC, Pritchard K, Seyedi N, Wang J, and Hintze T.** Chronic exercise in dogs increases coronary vascular nitric oxide production and endothelial cell nitric oxide synthase gene expression. *Circ Res 74: 349-353, 1994.*

99. **Shi X, Andresen JM, Potts JT, Foresman BH, Stern SA, Raven PB.** Aortic baroreflex control of heart rate during hypertensive stimuli: effect of fitness. *J Appl Physiol. 74: 1555–1562, 1993.*

100. **Silva ME, Fernandez JM, Castillo E, Nunez VM, Vaamonde DM, Poblador MS, and Lancho JL.** Influence of vibration training on energy expenditure in active men. *J Strength Cond Res 21(2): 470-5, 2007.*

101. **Sinoway, L., J. Shenberger, L. Gretchen, et al.** Forearm training attenuates sympathetic responses to prolonged rhythmic forearm exercise*. J. Appl. Physiol. 81: 1778–1784, 1996.*

102. **Sjostrom L.** Morbidity and mortality of severely obese subjects. *Am J Clin Nutri 55: 508S-515S, 1992.*

103. **Smith SA, Querry RG, Fadel PJ, Welch-O'Connor RM, Olivencia-Yurvati A, Shi X, Raven PB.** Differential baroreflex control of heart rate in sedentary and aerobically fit individuals. *Med Sci Sports Exerc.* 32: 1419–1430, 2000.

104. **Straznicky NE, Lambert EA, Lambert GW, Masuo K, Esler MD, and Nestel PJ.** Effects of dietary weight loss on sympathetic activity and cardiac risk facrots associated with the metabolic syndrome. *Clin Endocrinol Metab 90: 5998-6005, 2005.*

105. **Sugawara J, Hayashi K, Yokoi T, Cortez-Cooper MY, Devan AE, Anton MA, and Tanaka H.** Brachial-ankle pulse wave velocity: an index of central arterial stiffness? *J Hum Hypertens 19: 401-406, 2005.*

106. **Tahvanainen A, Koskela J, Tikkakoski A, et al***.* Analysis of cardiovascular responses to passive head-up tilt using continous pulse wave analysis and impedance cardiography. *Scandinavian Journal of Clinical and Laboratory Investigation 69: 128-37, 2009.*

107. **Tanaka H, DeSouza CA, and Seals DR.** Absence of age-related increase in central arterial stiffness in physically active women. *Arteriosclerosis, Thrombosis, and Vascular Biology 18: 127-132, 1998.*

108. **Tanaka H, Dinenno FA, Monahan KD, Clevenger CM, DeSouza CA, and Seals DR.** Aging, habitual exercise, and dynamic arterial compliance. *Circ 102: 1270-1275, 2000.*

109. **Taylor AC, McCartney N, Kamath MV, and Wiley RL.** Isometric training lowers resting blood pressure and modulates autonomic control. *Med Sci Sports Exerc 35: 251-256, 2003.*

110. **Tatchum-Talom R, Schulz R, McNeill JR, and Khadour FH.** Upregulation of neuronal nitric oxide synthase in skeletal muscle by swim training 3. *Am J Physiol Heart Circ Physiol 279,(4): H1757-H1766, 2000.*

111. **Troisi RJ, Weiss ST, Parker DR, Sparrow D, Young JB, and Landsberg L.** Relation of obesity and diet to sympathetic nervous system activity. *Hyperten 17: 669-677, 1991.*

112. **Van Der Heijden GJ, Wang ZJ, Chu Z, Toffolo G, Manesso E, Sauer PJ, Sunehag AL.** Strength exercise improves muscle mass and hepatic insulin sensitivity in obese youth. *Med Sci Sports Exerc. 42(11):1973-80, 2010.*

113. **Vassilakopoulos T, Deckman G, Kebbewar M, Rallis G, Harfouche R, and Hussain SN.** Regulation of nitric oxide productin in limb and ventilatory muscles during chronic exercise training 1. *Am J Physiol Lung Cell Mol Physiol 284(3): L452-L457, 2003.*

114. **Vaz M, Jennings G, Turner A, Cox H, Lambert G, and Esler M.** Regional sympathetic nervous activity and oxygen consumption in obese normotensive human subjects. *Circ 96: 3423-3429, 1997.*

115. **Verschueren SM, Roelants M, Delecluse C, Swinnen S, Vanderschueren D, and Boonen S.** Effect of 6-month whole body vibration training on hip density, muscle strength, and postural control in postmenopausal women: a randomized controlled pilot study. *J Bone Miner Res 19: 352-359, 2004.*

116. **Watanabe H, Ohtsuka S, Kakihana M, Sugishita Y.** Coronary circulation in dogs with an experimental decrease in aortic compliance. *J Am Coll Cardiol 21: 1497-1506, 1993.*

117. **Wiegman DL, Harris PD, Joshua IG, and Miller FN.** Decreased vascular sensitivity to norepinephrine following exercise training. *J Appl Physiol 2: 282-7, 1981.*

118. **Wilkinson B, MacCallum H, Flint L, Cockcroft JR, Newby DE, and Webb DJ.** The influence of heart on augmentation index and central arterial pressure in humans. *J Physiol 525 263-270, 2000.*

119. **Williams IL, Wheatcroft SB, Shah AM, and Kearney MT.** Obesity, atherosclerosis and the vascular endothelium: mechanisms of reduced nitric oxide bioavailability in obese humans. *Int J Obes Relat Metab Disord 26; 754-764, 2002.*

120. **Xiang G-d, Pu J, Sun H, Zhao L, Yue L, and Hou J.** Regular aerobic exercise training improves endothelium-dependant arterial dilation in patients with subclinical hypothyroidism. *Euro J of Endocrin 161: 755-761, 2009.*

121. **Yamada E, Kusaka T, Miyamoto K, Tanaka S, Morita S, Tanaka S, Tsuji S, Mori S, Norimatsu H, and Itoh S.** Vastus lateralis oxygenation and blood volume measured by near-infrared spectroscopy during whole body vibration. *Clin Physiol Funct Imaging 25: 203-208, 2005.*

122. **Yamamoto K, Miyachi M, Saitoh T, Yoshioka A, Onodera S.** Effects of endurance training on resting and post-exercise cardiac autonomic control. *Med Sci Sports Exerc 33: 1496-1502, 2001.*

123. **Zaros PR, Romero Pires CEM, Bacci M, Moraes C, and Zanesco A.** Effect of 6-month of physical exercise on the nitrate/nitrite levels in hypertensive postmenopausal women. *BMC Womens Health 9:17, 2009.*

124. **Zhu W, Zhong C, Yu Y, and Li K.** Acute effects of hyperglycaemia with and without exercise on endothelial function in healthy young men. *Euro J Appl Physiol 99:585-591, 2007.*

125. **Zoppini G, Cacciatori V, Gemma ML, Moghetti P, Targher G, Zamboni C, Thomaseth K, Bellavere F, and Muggeo M.** Effect of moderate aerobic exercise on sympatho-vagal balance in Type 2 diabetic patients. *Diabetic Medicine 24: 370-376, 2007.*

BIOGRAPHICAL SKETCH

Ryan Gil

During Ryan Gil's undergraduate studies at Florida State University (FSU), he first became inspired to conduct research while sitting amongst his colleagues in the class Anatomy & Physiology, taught by Arturo Figueroa M.D., Ph.D. After receiving his bachelor's degree he matriculated into a graduate program in exercise physiology, driven by the idea of directing a research project. While in the program, Ryan instructed laboratory classes in Functional Anatomy & Physiology, attended to his coursework, and worked on his thesis while assisting the production of additional research projects with his colleagues. His thesis aims to progress research being conducted on whole-body vibration exercise, a developing area in clinical rehabilitation. This thesis project is the first to determine specific cardiovascular and autonomic nervous system training effects that result from whole-body vibration exercise. It is his hope that this project will make a contribution to the expanding knowledge in healthcare that focuses on improving the quality and duration of life in individuals. During his final semester of his master's degree at FSU, he was accepted to a Doctor of Physical Therapy program at Columbia University. Ryan's highest personal goal is to make global impacts in healthcare by continuing the expansion of knowledge for the rehabilitation community.