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Associations between Measures of Obesity and Arterial Stiffness of Young Hispanic Men

Maria Perez

University of Texas at El Paso, mariap0825@yahoo.com

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ASSOCIATIONS BETWEEN MEASURES OF OBESITY AND ARTERIAL
STIFFNESS OF YOUNG HISPANIC MEN

MARIA PEREZ

Department of Kinesiology

APPROVED:

George King, Ph.D., Chair

Sudip Bajpeyi, Ph.D.

Christina Sobin, Ph.D.

Charles Ambler, Ph.D.

Dean of the Graduate School

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2015

ASSOCIATIONS BETWEEN MEASURES OF OBESITY AND ARTERIAL STIFFNESS OF
YOUNG HISPANIC MEN

by

MARIA PEREZ B.A.

THESIS

Presented to the Faculty of the Graduate School of
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Abstract

Arterial stiffness and obesity are independent predictors of coronary heart disease and cardiovascular disease mortality; however, there is conflicting evidence for the association between arterial stiffness and obesity. Arterial stiffness research of young Hispanic males is limited. **PURPOSE:** To examine the association between measures of obesity and peripheral and aortic stiffness of young Hispanic men. **METHODS:** All data are expressed as mean \pm SD. Participants were 23 non-obese (nOB; BMI 25.87 ± 3.09 kg/m²) and 21 obese (OB; BMI 35.42 ± 4.87 kg/m²) young Hispanic men (age range 18–25 years). Measures of obesity included body fat percentage, BMI, waist circumference, hip circumference, and waist-to-hip ratio. Arterial stiffness was determined at rest and post-exercise with pulse wave velocity (PWV) and augmentation index (AIx). Between-group comparisons (nOB vs. OB) were conducted with a mixed model analysis of variance and multivariate analysis was used to determine the strongest predictor of arterial stiffness. **RESULTS:** Peripheral PWV at rest was significantly greater ($p=0.013$) in the OB group (8.49 ± 1.25 m/s) compared to the nOB group (7.53 ± 1.17 m/s) and post-exercise differences between groups were uniformly in the hypothesized direction (OB: 8.16 ± 1.24 ; nOB: 7.42 ± 1.7 m/s, $p=0.055$). There was no significant between-group difference in AIx at rest (OB: $1.1 \pm 15.4\%$; nOB: $2.4 \pm 17.0\%$) or post-exercise (OB: -54.3 ± 20.8 ; nOB: -61.9 ± 12.6) ($p>0.05$). Of the predictors entered into the statistical model, waist-to-hip ratio was a stronger predictor of PWV than body fat percentage. **CONCLUSION:** The results of this study suggest that for young Hispanic men the distribution of adipose tissue may be a better predictor of arterial stiffness than total body fatness. These results for young Hispanic men reiterate the potential negative impact of central adiposity on the risk for cardiovascular disease. Additionally, acute exercise resulted in increased arterial compliance with no significant difference observed

between groups which may have been related to the exercise intensity or duration. Future studies are needed to determine if a higher intensity or longer duration of exercise may elicit significantly different changes between obese and non-obese young Hispanic men.

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Chapter 1 – Introduction

Although cardiovascular disease (CVD) related deaths have been on the decline in the U.S. since the 1960's, CVD continues to be the leading cause of death, accounting for 1 in 4 deaths each year (Centers for Disease Control and Prevention [CDC], 2010). Hispanics comprise 17% of the U.S. population, yet they account for 20.5% of CVD related deaths, compared to 63% of the U.S. population classified as non-Hispanic White but accounting for only 25.1% of CVD deaths (CDC, 2010). Alterations of the arterial system, such as increased stiffness, can contribute to CVD morbidity and mortality (Arnett, Evans, & Riley, 1994). Risk factors such as hypertension, hyperlipidemia, arterial stiffness, and obesity are present in young adults, and early detection of these risk factors may predict future CVD events (Sutton-Tyrrel et al., 2005).

Arterial stiffness and arterial compliance are both terms used to describe the structure of the arterial wall. Arterial stiffening refers to hardening of the arterial walls while arterial compliance refers to the distensibility of an artery. Compliance and distensibility are associated with an artery's ability to dilate and constrict in response to a change in pressure during a cardiac cycle and are directly related to blood flow velocity. The degree of arterial stiffening varies throughout the arterial tree. Muscular, peripheral arteries are not as compliant as central arteries, such as the aorta and carotid artery; up to 40% of the aorta is composed of elastic fibers and is the most compliant artery (Bader, 1982).

Two of the main determinants of arterial stiffness are collagen and elastin. Overproduction of collagen and/or diminished levels of elastin contribute to stiffening. Increased luminal pressure associated with hypertension leads to a disproportionate production of collagen which leads to an increase in thickness of the medial and intimal layers of the vascular wall (Zieman, Melenovsky & Kass, 2005) resulting in a resistance to blood flow caused by the same

volume of blood flowing through a smaller lumen. The greater blood flow velocity increases the chance of turbulent flow, which increases cardiac workload. In the heart's attempt to conserve energy, stroke volume decreases resulting in a decreased cardiac output (Smith & Fernhall, 2011).

Also contributing to arterial stiffness are advanced glycation end products (AGEs). AGEs are formed through the process of glycation, the non-enzymatic addition of a carbohydrate to a protein or fat. AGEs contribute to the formation of cross-links between collagen fibers, making cells stiffer. They also inhibit the vasodilator nitric oxide (NO) from forming in the endothelium, and promote the formation of reactive oxygen species (Goldin, Beckman, Schmidt, & Creager, 2006).

Obesity is an independent risk factor for CVD (Poirier et al., 2006) and obesity has been linked to increased arterial stiffness (Zebekakis et al., 2005). In a cross-sectional study, Danias et al. (2003) found that abdominal aortic elasticity of the obese group ($\text{BMI} \geq 30 \text{ kg/m}^2$) was significantly reduced compared to their lean counterparts ($\text{BMI} 19\text{--}25 \text{ kg/m}^2$). Endurance exercise can be used to combat obesity and to improve arterial compliance (Hayashi, Sugawara, Komine, Maeda, & Yokoi, 2005). The association between arterial stiffness and exercise is partly due to the vasodilator nitric oxide (NO). Exercise induces shear stress, which increases NO production and the production of extracellular superoxide dismutase, an inhibitor of NO breakdown (Gielen, Schuler, & Hambrecht, 2001).

Increased arterial stiffness is associated with increased blood pressure and increased systemic vascular resistance (SVR), the resistance to blood flow within the systemic circulation (Izzo & Shykoff, 2001). SVR is dependent on blood viscosity and blood vessel diameter (Smith & Fernhall, 2011), and decreases during exercise. A study of individuals aged 13–47 years

reported that an acute bout of exercise resulted in significantly decreased SVR, which peaked at 5 minutes and was maintained for 60 minutes post-exercise (Coats et al., 1989). The degree of SVR decrease varies among different populations. SVR during exercise has been reported to be greater for hypertensive compared to normotensive individuals (Frolich et al., 1967) and normotensive African-American boys compared to normotensive White boys (Arensman, Treiber, Gruber, & Strong, 1989) for the same exercise intensity.

In addition to disparities observed during exercise, between races, CVD risk factors disparities exist between race and ethnic groups. For adults aged 21 years and older, Mexican-Americans, non-Hispanic blacks, Native-Americans, and Alaska natives have a higher prevalence of diabetes compared to non-Hispanic Whites of the same age (Smith et al., 2005). African-Americans have a greater prevalence of hypertension compared to non-Hispanic Whites (Mozaffarian et al., 2015). The disparities in cardiovascular disease risk factors highlight the necessity of research for different races and ethnicities.

Most research for arterial stiffness has examined middle-aged and older adults although arterial stiffness is a chronic condition that begins to develop during childhood and appears to be exacerbated with obesity. To the authors' knowledge, the examination of SVR during exercise and comparison between obese and non-obese Hispanic men has not been studied. Research on arterial stiffness for Hispanics is limited. Of the limited existing research, Hlaing, & Prineas (2006) found no significant difference of arterial stiffness between Hispanics, non-Hispanic Whites, and non-Hispanic Blacks. However, their study did not examine obesity differences within groups. Therefore, we chose to examine obese Hispanics, an under-researched group for SVR and arterial stiffness.

Statement of the Problem

Hispanics have a greater prevalence of CVD risk factors, including obesity, than their non-Hispanic White counterparts (Hlaing, Koutoubi, & Huffman, 2006). Degree of vascular stiffening may predict future cardiac events (Sutton-Tyrrel et al., 2005). Research for Hispanics and arterial stiffening is limited and to the authors' knowledge, the association between obesity and extent of arterial stiffening for Hispanics has not been reported.

Purpose of the Study

The purpose of this study is to examine the extent of arterial stiffness as well as the degree of compliance in response to acute exercise of sedentary and low physically-active Hispanic young men.

Hypotheses

1. Obese Hispanic men will have significantly greater levels of arterial stiffness compared to non-obese Hispanic men at rest and immediately following an acute bout of submaximal exercise.
2. Obese Hispanic men will have greater SVR at rest and during an acute bout of submaximal exercise than non-obese Hispanic men.

Delimitations

The following were delimitations of this study.

1. Participants reported to the laboratory for testing on three separate sessions. Exercise testing was separated by approximately 48 hours in order to avoid excessive fatigue.
2. The sample size for this study was $n = 40$ ($n = 20$ per group) limiting the generalizability of the study results to the population at-large. An a-priori power analysis at a beta of 0.90 and an alpha of 0.05 indicated that a sample size of 40 should be sufficient to observe statistically significant differences between groups.
3. The population chosen was Hispanic men, age 18–25 years. This was chosen because most of the existing research has studied middle-aged and older populations, and few studies have examined Hispanic populations.
4. SVR was measured while participants exercised on a recumbent cycle ergometer. A recumbent cycle ergometer was chosen over an upright cycle to avoid artifact on the electrocardiogram.
5. Arterial stiffness was measured with pulse wave velocity (PWV) and augmentation index (AIx) because of the reproducibility of PWV and non-invasiveness of the techniques.
6. Because a cycle protocol was used when measuring SVR, a cycle protocol was used to estimate $\text{VO}_{2\text{max}}$.

Limitations

The following were limitations of this study.

1. Participants were asked to provide their physical activity habits, which may have resulted in self-reporting bias.
2. Because a recumbent cycle ergometer was used it may have caused the participants' legs to fatigue before the desired heart rate and exercise intensity was achieved.
3. Because the participants were not regularly active, and because of the high reproducibility of submaximal tests when compared to maximal tests, a submaximal test was used to estimate $\text{VO}_{2\text{max}}$. This was also chosen as an attempt to prevent participant drop-out.
4. All testing was performed at the University of Texas at El Paso laboratory.
5. Air-displacement plethysmography was used to calculate body fat percentage. Although all participants wore compression shorts and a swim cap to reduce error caused by air trapped in clothing, participant body and facial hair may have increased standard error.
6. Systemic vascular resistance was not measured directly, it was measured with an impedance cardiography device.

Definitions

Augmentation pressure – an absolute measure of arterial stiffness calculated as the difference between the first and second systolic peak (Weber et al., 2004)

Augmentation index – augmentation pressure divided by pulse pressure and expressed as a percentage (Weber et al., 2004)

Arterial Compliance – the ability of an artery to expand during systole (Pappano & Wier, 2013)

Arterial stiffness – vascular hardening and resistance to elasticity, developed by changes of the vessel wall (Belz, 1995)

Elastance – the ability of an artery to recoil during diastole; the inverse of compliance (Belz, 1995)

Endothelium – a layer of epithelial cells located between the lumen and the vascular smooth muscle that lines all blood vessels (Smith & Fernhall, 2011)

Pulse pressure – the difference between systolic and diastolic blood pressure (Domanski, Davis, Pfeffer, Kastantin, & Mitchell, 1999)

Pulse wave velocity – a measurement of arterial stiffness calculated as the time required for the pulse to travel between two sites (Blacher, Asmar, Djane, London, & Safar, 1999)

Shear Stress – the force exerted on the endothelium by the sliding movement of blood flow (Smith & Fernhall, 2011)

Systemic vascular resistance (SVR) – sum of resistance to blood flow in the vascular system (Powers & Howley, 2012)

Chapter 2 – Literature Review

Arterial stiffness affects the ability of the arterial walls to expand during systole and recoil during diastole and can be measured non-invasively with wave reflections. Arterial stiffness is associated with atherosclerosis, hypertension, and coronary heart disease (CHD) (Mattace-Raso et al., 2006). Several factors influence the degree of stiffening, including age (Laogun & Gosling, 1982), hypercholesterolemia (Wilkinson et al., 2002), impaired glucose tolerance (Henry et al., 2003), and physical activity level (Tanaka et al., 2000). Physical activity can reverse stiffening through the production of nitric oxide (NO), induced by shear stress. Arterial stiffness may affect the decrease of systemic vascular resistance (SVR) during exercise if stiffening leads to a diminished increase in cardiac output (Izzo & Shykoff, 2001). SVR can be measured by inserting a catheter into the pulmonary artery but non-invasive methods of measurement utilizing ultrasound and impedance cardiography are also available and are more suitable for exercise testing.

Measuring Arterial Stiffness and Systemic Vascular Resistance

The gold standard of arterial stiffness measurement is pulse wave velocity (PWV) (DeLoach, & Townsend, 2008) and can be measured invasively with pressure catheters or non-invasively with applanation tonography and Doppler ultrasound. Normal ventricular contraction during a cardiac cycle generates a pulse pressure wave that travels through the arteries. PWV is determined from the amount of time required for the pressure wave to travel between two measurement sites. The distance between the measurement sites divided by time required for detection of the pulse at each site determines PWV. An electrocardiogram recording is used to

determine pulse time, with the R wave used as a time marker; the faster the PWV, the stiffer the artery.

When measured at the carotid artery and femoral artery, PWV represents arterial stiffness of the aorta, known as aortic, or central stiffness. PWV represents peripheral stiffness when radial-carotid artery sites are measured (arm artery stiffness). Both are illustrated in Figure 2.1. Additionally, peripheral stiffness can be measured at the femoral-tibial sites (leg artery stiffness, not pictured). PWV has been used by a number of studies including (Blacher et al., 1999) who found that aortic PWV was related to arterial stiffness, independent of blood pressure and age.

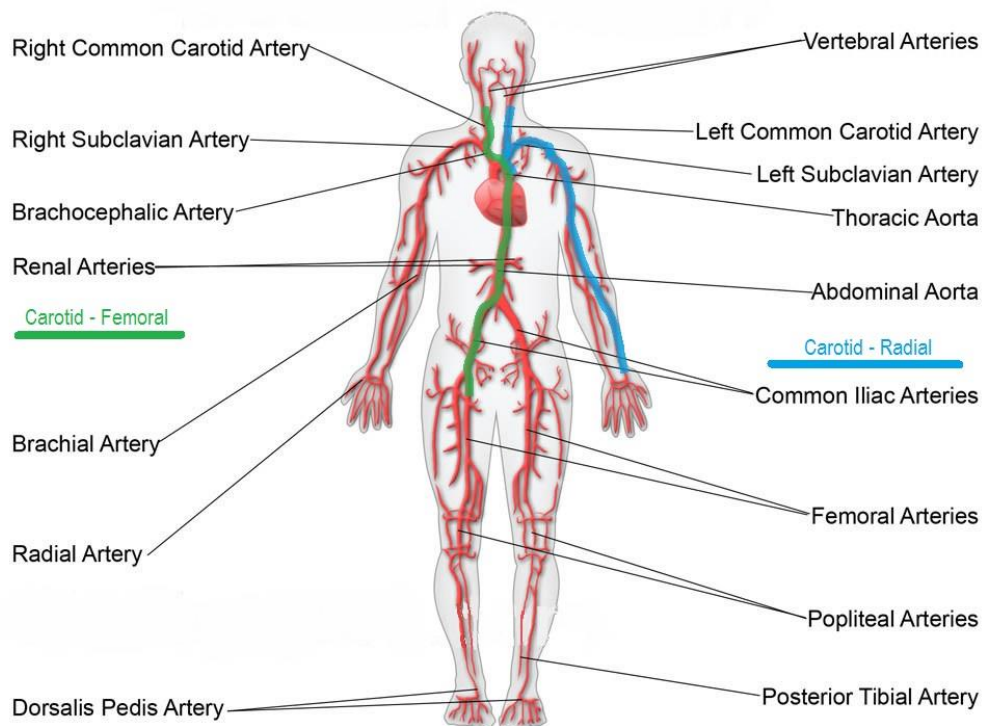


Figure 2.1 Travel pathway measured between the carotid-femoral sites (central stiffness) and carotid-radial sites (peripheral stiffness).

PWV can be measured invasively with two pressure catheters inserted on each measurement site. Alternatively, pulse wave velocity can be evaluated non-invasively with the use of applanation tonometry and sphygmomanometry equipment which uses a tonometer that is sensitive to pressure wave forms such as the SphygmoCor system (AtCor Medical). The use of the SphygmoCor system has been previously validated as a measurement of arterial stiffness (Ding et al., 2011). Placed over each site, the tonometer can detect the pulse without the use of a catheter. Another non-invasive method to assess PWV is with the use of Doppler ultrasound and involves the placement of sensors at each site.

Arterial stiffness can also be measured by analyzing wave reflections. The ejection of blood from the left ventricle during systole creates a forward pulse-wave in the arteries. The wave then reflects off the arterial wall and creates a reflective wave back toward the heart while still in systole. The measurement of augmentation pressure (AP) is one method for determining arterial stiffness from wave reflections and is the difference between the pressure of the reflected wave (P2) and the initial systolic wave (P1), measured in millimeters of mercury (mm Hg). This can be further expressed as an augmentation index (AIx) by dividing AP by pulse pressure (PP)—the difference between systolic and diastolic blood pressure—and expressed as a percentage. Earlier reflections indicate stiffer arteries (Weber et al., 2004) and a greater AIx. A reflected wave greater than the systolic wave results in a positive AIx, whereas a reflected wave lower than the systolic wave results in a negative AIx. This is demonstrated in Figure 2.2. Augmentation pressure and AIx have been correlated with coronary artery disease (CAD) when using the SphygmoCor system (Weber et al., 2004).

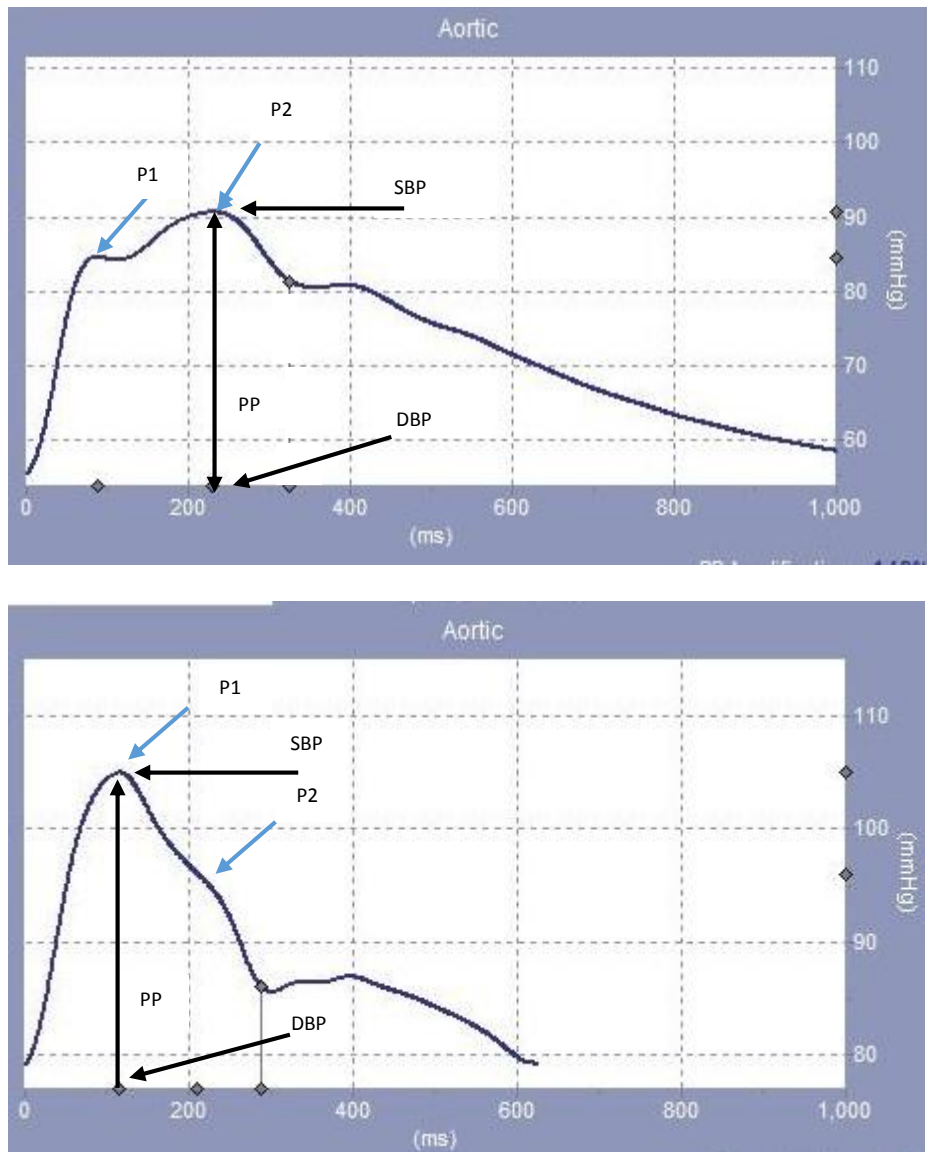


Figure 2.2. Augmentation Pressure (AP) is the difference in height between the systolic wave (P1) and the reflected wave (P2). AP is then divided by pulse pressure to obtain augmentation index (AIx). A reflected wave higher than the systolic wave results in a positive AIx (top figure). The reflected wave (P2) is lower than the systolic wave (P1) resulting in a negative AIx. SBP (systolic blood pressure), DBP (diastolic blood pressure), PP (pulse pressure)

Central stiffness may be greater for women than for men (Waddell, Dart, Gatzka, Cameron, & Kingwell 2001) and there is some variation between race and ethnic groups. Reported data for healthy, normotensive young men and women for PWV is presented in Table 2.1 and data for AIx is presented in Table 2.2.

Table 2.1 Pulse Wave Velocity Reference Values for Healthy, Normotensive Men and Women

Population	Age (years)	n	PWV (m/s)	Reference
Argentinian Men and Women	20-29	110	5.86±0.92	Díaz et al., 2014
European Men and Women	<30	896	6.1±4.6	Reference Values for Arterial Stiffness Collaboration, 2010
Finnish Men	25–41	283	7.7±1.2	Koivisto et al., 2007
South African Men and Women	<30	97	5.0±1.2	Shiburi et al., 2006

Data are presented as mean ± SD

Table 2.2 Augmentation Index Reference Values for Healthy, Normotensive Men and Women

Population	Age (years)	n	Central AIx (%)	Reference
Korean Men and Women	≤39	134	23.4±8.4	Chung et al., 2010
South African Men and Women	<30	97	15.3±13.2	Shiburi et al., 2006
European Men	<30	106	-1.5±12.1	Wojciechowska, 2006

Data are presented as mean ± SD

Measurement of arterial compliance and elastance, and indicators of arterial stiffness (Belz, 1995) can be performed with Doppler ultrasound by measuring the change in systolic and diastolic blood pressure and change in volume, calculated with the formula:

$$C = \Delta P / \Delta V$$

where C represents arterial compliance, ΔP is the change in pressure, and ΔV is the change in volume. Elastance (E) is the inverse of compliance and is therefore calculated as:

$$E = \Delta V / \Delta P$$

Although brachial pulse pressure is not a direct measure of central pulse pressure, brachial pulse pressure may indicate aortic stiffness and has been used as a surrogate measure

(Benetos et al., 1997; Hlaing et al., 2006; Hlaing, & Prineas, 2006). Increased pulse pressure is due to diminished aorta elasticity. During systole, a fraction of stroke volume is preserved in the proximal arteries for an elastic aorta. The remainder of the stroke volume is delivered to the peripheral arteries during diastole, resulting in a smooth flow and narrow pulse pressure. For stiff aortas, the lack of elasticity results in stroke volume's full delivery to peripheral arteries during systole, with minimal diastolic flow, resulting in increased pulse pressure (Izzo & Shykoff, 2001). Pulse pressure has been reported to be a predictor of cardiac risk in French men 40–69 years old (Benetos et al., 1997) and men and women 50–79 years old (Franklin, Khan, Wong, Larson, & Levy, 1999). One of the limitations of pulse pressure is that while it may be an indicator of aortic stiffness, it cannot be utilized to assess peripheral stiffness.

Systemic vascular resistance (SVR) is calculated as:

$$SVR = (MAP - CVP)/\dot{Q}$$

where MAP represents mean arterial pressure, CVP is central venous pressure, and \dot{Q} is cardiac output. Because CVP is close to zero, the formula is often simplified to $SVR = MAP/\dot{Q}$ (Kelman, 1977). SVR is measured in dynes \cdot s/cm⁵. One dyne is the force necessary to accelerate one gram of mass at a rate of one centimeter per second squared. One mm Hg of pressure is equal to 1,330 dynes/cm² and cardiac output is expressed as cm³/s. Alternatively, SVR can be measured as pressure divided by cardiac output and expressed as mmHg/min/mL. One mmHg/min/mL is equal to 80 dynes \cdot s/cm⁵; therefore, in order to convert from mmHg/min/mL, one must multiply by 80 to obtain SVR in dynes \cdot s/cm⁵.

Two methods to measure cardiac output are the Fick method and the thermodilution method. Both methods are based on the principle of the Conservation of Matter which states that over any period of time the amount of a substance that enters any closed system is equal to the

amount that leaves during the same period (Kelman, 1977). Measurement of cardiac output using the Fick principle consists of measuring oxygen consumption and insertion of a catheter into the pulmonary artery in order to obtain samples of mixed-venous blood. The Fick principle states:

$$\dot{Q} = \text{VO}_2 / a\text{-vO}_2 \text{ diff}$$

where VO_2 represents oxygen consumption, and $a\text{-vO}_2 \text{ diff}$ is the arteriovenous oxygen difference or, the difference between the oxygen content in arterial blood and the oxygen content in venous blood (McMichael, & Sharpey-Schafer, 1944). The amount of oxygen entering the system in the venous blood each minute is:

$$\dot{Q} \times \text{CvO}_2$$

where CvO_2 is the concentration of oxygen in the venous blood.

The amount of oxygen leaving the system each minute in the arterial blood is:

$$\dot{Q} \times \text{CaO}_2$$

where CaO_2 is the concentration of oxygen in the arterial blood. Therefore, the rate of oxygen removal from the lungs by the blood is:

$$\dot{Q}(\text{CaO}_2 - \text{CvO}_2)$$

which is equal to oxygen consumption at steady state. Therefore, $\dot{Q} = (\text{VO}_2) / (\text{CaO}_2 - \text{CvO}_2)$ or also stated as $\dot{Q} = \text{VO}_2 / a\text{-vO}_2 \text{ diff}$ (Kelman, 1977).

Cardiac output can also be measured with thermodilution. Cold saline of a known volume and temperature is injected into the right atrium. Another catheter with a thermistor tip is inserted into the pulmonary artery through a peripheral vein. The cold saline mixes with and cools the blood and the change in blood temperature is measured. Cardiac output is inversely proportional to the change in blood temperature over time (Pappano & Wier, 2013).

A non-invasive measurement of cardiac output can be performed with Doppler ultrasound. Ultrasound is used to measure the cross-sectional area of the aortic arch and the amount of blood ejected into the aorta during systole, measuring stroke volume. Cardiac output is then calculated as the product of stroke volume and heart rate (Huntsman et al., 1983).

Alternatively, SVR and other parameters can be measured non-invasively by an impedance cardiography device, such as the PhysioFlow (Manatec Biomedical). Electrodes are placed on the thorax and thoracic fluids are measured with a high-frequency, low-magnitude current that measures the level of change in impedance of the thoracic fluid. A study by Charloux et al. (2000) compared the measurement of cardiac output using the PhysioFlow and the Fick method. Patients were measured with both methods at rest and during exercise at a workload of 10 to 50 watts. Researchers found that the PhysioFlow provided an acceptable, non-invasive assessment of cardiac output at rest and during exercise with a difference between the two methods of 0.04 L/min at rest and 0.29 L/min during exercise (Charloux et al., 2000).

Several methods for the measurement of arterial stiffness are available, including the gold standard method of PWV whose non-invasive methods have been validated and reproducible (Ding et al., 2011). While SVR can be directly measured with catheters in order to obtain cardiac output, assessment can be performed non-invasively with the use of impedance cardiography and may be more suitable for exercise than invasive techniques.

Effect of Age and Physical Activity

In general, arterial stiffness increases with age (Laogun & Gosling, 1982). Aerobic exercise can attenuate the age-related increase of arterial stiffness of men and women, whereas resistance training can increase stiffness (Bertovic et al., 1999). The improvements in

compliance may be due to the increase in NO and the decrease in the vasodilator, endothelin-1 (ET-1).

Arterial stiffness appears to be a contributing factor to the age-associated increase of cardiovascular disease. In a study of 600 healthy Caucasian infants, children, and adults, Laogun and Gosling (1982) reported that aortic compliance peaked between ages 8 and 10 years, and steadily declined thereafter. Other researchers have also reported on the increase of arterial stiffness with increased age (Tanaka, DeSouza, and Seals, 1998; Tanaka et al., 2000).

Conflicting results regarding arterial stiffness and exercise have been reported. Seals et al. (2001) reported no change for aortic stiffness of sedentary post-menopausal women (age 69 ± 9 years) following a 3-month walking program. The women began with 30 minutes of walking, 3–4 days per week at 40%–50% maximal heart rate (HR_{max}). After the initial weeks, this was increased to 45 minutes of walking at 65%–80% of HR_{max} for as many days per week as possible. There was no difference in carotid AIx or aortic PWV following the intervention (Seals et al., 2001).

Contrary to this, Tanaka et al. (1998) examined the effect of age and physical activity on healthy sedentary and active pre-menopausal and post-menopausal women. As measured by PWV, there was no difference in peripheral arterial stiffness between pre- and post-menopausal sedentary women; however, central arterial stiffness was significantly greater for sedentary post-menopausal (1065 ± 110 cm/s) than for sedentary pre-menopausal (690 ± 80 cm/s) women. Conversely, there were no significant differences in peripheral or central arterial stiffness between the physically active pre- and post-menopausal women. Additionally, aortic PWV was approximately 30% to 50% lower for the physically active post-menopausal women than for the sedentary post-menopausal women. These results indicate an age-related increase in central

arterial stiffness among women and suggest that exercise can delay aortic arterial stiffening due to increased age (Tanaka, et al., 1998).

An exercise effect on arterial stiffness has also been observed for men. In a study by Tanaka et al. (2000), men were divided into two protocols: a cross-sectional study and an intervention study. Participants of the cross-sectional study were either sedentary, recreationally active (light to moderate exercise ≥ 3 days/week), or endurance trained (vigorous endurance exercise ≥ 5 days/week) and divided into a young (18–37 years), middle-aged (37–57 years), or older (58–77 years) group. The authors reported that central arterial compliance was lower for the middle-aged and older groups compared to the younger group. Central arterial compliance was 20–35% greater for the endurance-trained group than for the sedentary and recreationally active groups of the same age. Additionally, there were no differences in central arterial compliance between the sedentary and recreationally active men for any of the age groups (Tanaka et al., 2000).

The intervention arm of the study (Tanaka et al., 2000) examined the effect of a 3-month aerobic exercise training program on healthy middle-aged, and older men. Participants walked 25–30 minutes a day at approximately 60% of HR_{max} . As exercise tolerance improved, they walked or jogged for 40–45 minutes a day, 4–6 days per week at 70%–75% HR_{max} . Following 3 months of training, arterial compliance increased by 25% and was similar to the arterial compliance of the endurance-trained middle-aged and older groups of the cross-sectional arm of the study. No changes were observed for weight, body composition, or blood pressure after the 3 months of exercise training. The results of this study suggest that arterial compliance decreases with age and that vigorous endurance training can improve arterial compliance. Additionally,

improvement in arterial stiffness occurs independent of changes in weight, body composition, or blood pressure (Tanaka et al., 2000).

Exercise capacity may be associated with improvements of arterial stiffness due to exercise. A study by Cameron, Rajkumar, Kingwell, Jennings, and Dart (1999) with older (age 67 ± 7 years) men and women reported a positive correlation between central arterial compliance and exercise capacity during a Bruce treadmill protocol. Those with higher arterial compliance had a greater time to exhaustion than those with lower arterial compliance leading the authors to conclude that more compliant arteries result in greater exercise capacity (Cameron et al., 1999).

A bout of exercise results in acute physiological changes to the arterial wall. Kingwell et al. (1997) reported an increase in whole-body arterial compliance immediately following 30 minutes of cycle ergometry exercise at 65% of VO_{2max} , independent of mean arterial pressure. These immediate modifications were temporary and arterial compliance returned to baseline 60 minutes post-exercise (Kingwell et al., 1997). When examining arterial stiffness of an exercised vs. a non-exercised leg for healthy men (age 24 ± 1 years), Sugawara et al. (2003) reported a decreased arterial stiffness for the exercised leg only. Changes may be due to relaxation of vascular smooth muscle, increasing arterial wall compliance (Belz, 1995).

Resistance training has the opposite effect on arterial stiffness as aerobic exercise and increases arterial stiffness. For young men (age 26 ± 4 years) with no significant difference for age, MAP, lipids or VO_{2max} between groups, Bertovic et al. (1999) reported decreased aortic compliance for men who weight trained a minimum of 3 times a week for at least 12 months compared to a control group who had not performed any exercise for at least 12 months. Similar results were reported for women by Cortez-Cooper (2005) and Miyachi et al. (2004).

Concomitant strength and endurance training may prevent the increase in arterial stiffness associated with strength training (Kawano, Tanaka, & Miyachi, 2006). A study of previously sedentary young men had a 20% decrease in aortic compliance following 4 months of moderate-intensity strength training (3 sets, 14–16 repetitions, 50% of 1 repetition maximum), three days a week. However, after 4 months of vigorous-intensity strength training (3 sets, 8–10 repetitions, 80% of 1 repetition maximum) 3 days a week followed by 30 minutes of cycling at 60% HR_{max} , previously sedentary young men experienced no significant change in arterial compliance compared to pre-intervention. The reduced compliance observed for the resistance training group was limited to the aorta. No differences were seen pre- vs. post-intervention for femoral compliance for either group (Kawano, et al., 2006).

Mechanisms that may explain the improved arterial compliance following endurance training are the effect of aerobic training on NO and ET-1 levels. In a study by Maeda et al. (2001), healthy untrained men (age 20.3 ± 0.5 years) underwent 8 weeks of exercise training on a cycle ergometer for 1 hour a day, 3–4 days a week. Exercise intensity was 60% of VO_{2max} for the first week and 70% thereafter. Resting venous plasma concentration of NO and ET-1 were measured at baseline, immediately following the training intervention, and at 4 and 8 weeks after training ended. The authors found that the vasodilator NO had significantly increased (30.69 ± 3.20 vs. 48.64 ± 8.16 mmol/L, $p < 0.05$) and the vasoconstrictor ET-1 had significantly decreased (1.65 ± 0.14 vs. 1.23 ± 0.12 pg/mL, $p < 0.05$) immediately following the training intervention. Compared to baseline, NO remained elevated and ET-1 remained lower at 4 weeks post-intervention, but both returned to pre-training levels by 8 weeks after exercise had ceased (Maeda et al., 2001).

NO is important for many reasons. An insufficient production of NO can lead to endothelial dysfunction. Endothelial dysfunction refers to pathological changes in the structure and functioning of the endothelium and occurs when NO is lacking in vascular smooth muscle cells (Smith & Fernhall, 2011). Modifications in endothelial function can result in imbalances in the release of factors that control vascular tone and hemostasis (Smith & Fernhall, 2011). Flow-mediated dilation (FMD), the dilation of vessels due to an increase in blood flow, is dependent on NO (Smith & Fernhall, 2011). Impaired FMD can be improved with exercise training. Watts et al. (2004) found that an 8-week program of 1-hour circuit training, 3 days a week significantly improved previously impaired FMD for obese adolescents (age 14.3 ± 1.5 years). Additionally, following the intervention, FMD was similar to that of 20 lean controls with no FMD impairment (Watts et al., 2004).

Endothelial dysfunction is associated with increased prevalence of cardiac events (Neunteufl et al., 2000). Endothelial dysfunction and impaired FMD are precursors to atherosclerosis (Kelm, 2002), the build-up of plaque in arteries, causing them to narrow over time (McArdle, Katch, & Katch, 2010). Both atherosclerosis and peripheral artery disease result in arterial stiffening, due to the plaque accumulation in the arteries (van Popele et al., 2001).

Measurement of arterial stiffness may be a way to predict CHD. Cameron et al. (1996) found that patients with CHD had higher levels of aortic stiffness than a control group matched for age and blood pressure. Similarly, Mattace-Raso et al. (2006) found that aortic PWV was a strong predictor of CHD among men and women age 55 years and older.

Advanced age is associated with increased arterial stiffness but endurance exercise can be utilized to attenuate the increase. The improvements in arterial compliance related to exercise may be related to physiological changes of the vascular wall which may be attributed to

improvements in endothelial function and increases in NO. The intensity necessary to elicit improvements in arterial compliance is contradictory but age and gender may be confounders. Resistance training has the opposite effect and increases stiffness but concomitant strength and endurance training may prevent the increases in arterial stiffness as a result of resistance training.

Obesity

Conflicting results have been observed between arterial stiffness and obesity. Wildman, Mackey, Bostom, Thompson, and Sutton-Tyrrell (2003) reported that aortic stiffness was significantly correlated with body mass index (BMI), waist circumference, and waist-to-hip ratio (WHR) of White and African-American men and women, aged 20–70 years. For obese and lean Belgian men and women, Zebekakis et al. (2005) reported an increase in peripheral stiffness of the brachial and femoral arteries with increased BMI for men and women, aged 10–86 years. However, no association was observed between BMI and aortic stiffness for men. Increased BMI was associated with increased aortic stiffness for middle-aged and older, but not younger women.

Associations between measures of obesity and arterial stiffness are also conflicting for morbidly obese populations. Nordstrand et al. (2011) reported a significant positive correlation for morbidly obese women (mean BMI 43.5 ± 4.8 kg/m²) between PWV and BMI, waist circumference, and WHR. For men (mean BMI 46.1 ± 6.3 kg/m²) there was no significant association between PWV and WHR or waist circumference, and there was a negative correlation between PWV and BMI (Nordstrand et al., 2011).

Body fat distribution may be a better indicator of arterial stiffness than BMI. A longitudinal study reported that waist circumference for adolescent boys and girls, but not total body fat, predicted future increased aortic stiffness at age 36 years (Ferreira et al., 2004).

Similarly, Recio-Rodriguez et al. (2012) reported that for men and women aged 20–75 years, WHR and waist circumference were better predictors of aortic PWV than BMI and body fat percentage.

The increase in arterial stiffness can be reversed with weight loss (Wildman et al., 2005). For healthy White and African-American men and women aged 20–40 years, BMI was associated with aortic stiffness and at a 2-year follow-up, weight gain was associated with increased stiffness whereas the weight loss was associated with decreased stiffness. Additionally, for the same amount of weight gain, African-Americans had significantly larger increases in arterial stiffness compared to Whites.

Obesity is associated with increased arterial stiffness; however, certain measures of obesity—such as BMI—provide conflicting results. The contradictory data may be confounded by factors such as age and gender. Degree of stiffness incurred through weight gain is not uniform and may be dependent on race. The current literature suggests a need for additional research to better understand the effects of obesity on arterial stiffness.

Glucose and Blood Lipids

Fasting blood glucose levels and cholesterol levels may be associated with greater arterial stiffness in healthy and diseased populations. Regarding blood lipids, conflicting research in regards to the strongest predictor of arterial stiffness exists.

High levels of fasting plasma glucose are associated with arterial stiffness. In a study of older men and women (age 68.5 years), Henry et al. (2003) found that participants with type 2 diabetes had increased central and peripheral arterial stiffness compared to those with normal glucose metabolism. Non-diabetics with impaired glucose metabolism had an increase in peripheral stiffness but not central stiffness, compared to participants with normal glucose

metabolism (Henry et al., 2003). Similarly, Lukich, Matas, Boaz, and Shargorodsky, (2010) reported a significantly greater aortic PWV in middle-aged and older type 2 diabetic men and women than non-diabetics with impaired fasting glucose (7.5 ± 3.3 vs. 6.5 ± 1.1 m/s, $p < 0.05$) and significantly higher aortic PWV compared to men and women with normal fasting glucose (5.7 ± 1.1 m/s, $p < 0.05$). Similar results have been reported for healthy middle-aged men and women (Shin, Lee, and Lee, 2011). Greater arterial stiffness may be caused by advanced glycation end-products (AGEs), which may be caused by impaired fasting glucose (Schram et al., 2004).

Inconsistent results have been reported for the association between arterial stiffness and blood lipids. Wilkinson et al. (2002) found that central stiffness measured with AIX was significantly greater ($24.8 \pm 11.3\%$ vs. $15.6 \pm 12.1\%$, $p < 0.001$) in a hypercholesterolemic group compared to normocholesterolemic counterparts. Total cholesterol was not an independent predictor of central stiffness, but low-density lipoprotein (LDL) cholesterol was. Contrary to these results, Lehmann, Watts, Fatemi-Langroudi, and Gosling (1992) reported that participants < 24 years of age whose only cardiovascular risk factor was familial hypercholesterolemia, aortic compliance was greater than in matched normocholesterolaemic controls. Similarly, Dart et al. (1991) found that aortic compliance was greater for participants with isolated hypercholesterolemia than a normocholesterolemic control group. These studies (Wilkinson et al., 2002; Lehmann et al., 1992; Dart et al., 1991) did not control for physical activity level and it is possible that physical activity level may have contributed to the lower arterial compliance of the control group. As shown from these conflicting results, more research is needed to understand the association between cholesterol and arterial stiffness.

Systemic Vascular Resistance

SVR decreases during exercise due to the increase in cardiac output and MAP. SVR has been reported to increase with age, even among children (Schieken, Clarke, and Lauer, 1983). Disparities in the extent to which SVR decreases during exercise have been previously reported (Arensman et al., 1989)

Coats et al. (1989) measured SVR of men and women (age 17–47 years) after a maximal bicycle exercise protocol. SVR was significantly lower post-exercise when compared to the control of the same participants sitting quietly on a separate testing day. Sixty minutes post-exercise, SVR remained lowered. One of the regulators of SVR during exercise is NO. Stamler, Loh, Currie, & Creager (1994) reported that when healthy subjects were given N^G-monomethyl-L-arginine, a NO synthase inhibitor, blood pressure increased by 15.5% and SVR increased by 63.4%.

Higher SVR at rest is associated with increased age. Julius, Amery, Whitlock, and Conway (1967) reported that during maximal exercise SVR was higher for older participants. Participants were divided into younger (18–34 years), middle-aged (35–49 years), and older (50–69 years) age groups. No significant difference was observed for SVR between the younger and middle-aged groups, but a significant difference was seen in the older group when compared to both the younger and middle-aged groups.

An increase in SVR with age has been reported for children and adolescents as well. Schieken et al. (1983) reported a positive correlation between SVR and age in boys and girls 9 to 18 years old at rest and during cycling exercise. Differences in SVR have been observed between White and Black children. Arensman et al. (1989) found that among normotensive healthy 10-

year-old Black and White boys with similar blood pressure, Black boys had significantly ($p < 0.001$) greater SVR than White boys at rest (31.2 ± 8.67 vs. 24.1 ± 7.05 mmHg/min/mL) and at maximum exercise on a recumbent cycle (20.05 ± 6.68 vs. 15.28 ± 3.2 mmHg/min/mL). These results demonstrate the inconsistencies observed among different populations.

Disparities among Race and Ethnic Groups

Differences for cardiovascular risk factors have been observed among race and ethnic groups. Studies have reported higher rates of obesity, hypertension, diabetes, and dyslipidemia for Hispanics compared to non-Hispanic Whites (Selvin, & Erlinger, 2004; Sundquist, Winkleby, & Pudaric, 2001). Some of these differences appear to be gender dependent. Sundquist et al. (2001) reported a greater prevalence of type 2 diabetes and hypertension among older Mexican-American women compared to older non-Hispanic White women. However, the prevalence of type 2 diabetes and hypertension were similar between older Mexican-American men and older non-Hispanic White men. Winkleby, Robinson, Sundquist, and Kraemer (1999) reported a greater BMI for Mexican-American and Black children and young adults (age 6–24 years) compared to their White counterparts. The increased CVD risk factors in Mexican-American men and women may be related to the amount (or lack) of physical activity. Magoc, Tomaka, and Thompson (2010) reported that Mexican-American men and women engage in less physical activity than non-Hispanic Whites. Regular aerobic exercise has been shown to reduce CVD risk factors (Thompson et al., 2003).

Arterial stiffness is an independent predictor of CHD (Mattace-Raso et al., 2006) and is associated with atherosclerosis and hypertension. Arterial stiffness varies throughout the vascular tree and stiffness is influenced by many factors including age, physical activity level, obesity,

and blood lipids. The increased stiffness is the result of an increase in collagen and decrease in elastic of vascular walls which may be altered due to endothelial dysfunction, which in turn may inhibit NO release. Inconsistent data exists regarding measures of obesity and arterial stiffness. One of the factors that is affected by arterial stiffness is SVR. SVR decreases during exercise and those with stiffer arteries may experience a limited decrease in SVR during exercise due to a limited cardiac output. Race and ethnic group differences exist for cardiovascular disease risk factors, emphasizing the necessity for research for different race and ethnic groups.

Chapter 3 – Methods

This study was approved by the University of Texas at El Paso Institutional Review Board. All investigators associated with this study completed the CITI training modules in compliance with professional standards and university research policies. Flyers were posted around campus and word of mouth recruiting methods were used to obtain participants. The volunteer participants were asked to attend three separate data collection sessions separated by a minimum of 48 hours. Participants were between the ages of 18–25 years and had a low physical activity level. All participants were Mexican-American as defined by having one or both parents of Mexican descent. Participants on medication to control blood pressure or that could impact heart rate or vasodilation, or were taking any diuretics were excluded.

Protocol

Session 1

Session 1 involved obtaining consent, screening, explanation of the study procedures, and answering participant questions. An approved informed consent form that explained the procedures and protocols for the three testing sessions was provided to all participants. The form was explained and all questions were answered before participants gave their oral and written consent to participate.

Participants completed a Physical Activity Readiness Questionnaire (PAR-Q), an International Physical Activity Questionnaire (IPAQ), a health information survey, and a family medical questionnaire (Appendix E). Participants' physical activity level was considered low if self-reported physical activity was less than 600 MET minutes per week.

Participants were instructed to refrain from consuming energy drinks, coffee, or any other caffeinated drinks 12 hours prior to their assigned testing time. The participants were also asked to refrain from exercise 12 hours prior to testing sessions and to fast overnight prior to the third session.

Session 2

The purpose of the second session was to collect anthropometric data and perform a submaximal exercise test to estimate maximal oxygen uptake ($\text{VO}_{2\text{max}}$) to study a possible association between cardiorespiratory fitness and arterial stiffness of Hispanic men. The anthropometric measurement consisted of height (cm), body mass (kg), waist circumference (cm), and hip circumference (cm). Body mass index (BMI) was calculated as body mass in kilograms divided by height in meters squared (kg/m^2). Participants whose BMI was $\geq 30 \text{ kg}/\text{m}^2$ were categorized as obese (ACSM, 2014). Waist circumference was measured (Rosscraft measuring tape) immediately superior to the iliac crest and hip circumference was measured at the widest part of the hip (ACSM, 2014). Waist circumference was divided by hip circumference to obtain waist-to-hip ratio (WHR).

$\text{VO}_{2\text{max}}$ was estimated from heart rate during an Åstrand-Ryhming cycle ergometer submaximal exercise protocol (Åstrand & Ryhming, 1954) on an upright cycle ergometer (Corival; Lode Medical Technology, Groningen, the Netherlands). Heart rate was measured with a Polar heart rate monitor and recorded every 10 seconds. Participants cycled at 75 watts and maintained 50 revolutions per minute (RPM) for 6 minutes with the goal of achieving a steady-state heart rate between 125 and 170 beats per minute (bpm). If measured heart rate at minutes 5 and 6 differed by more than 5 bpm, participant continued to exercise at one minute increments until the steady-state heart rate criterion was met. If at the end of the 6 minutes heart rate was

less than 125 bpm, workload was increased by 25 watts and the participant exercised for an additional 6 minutes. The workload and average steady-state heart rate of the final two minutes of exercise were used to estimate absolute $\text{VO}_{2\text{max}}$ (L/min) with the Åstrand-Ryhming nomogram (Appendix C). Estimated $\text{VO}_{2\text{max}}$ was adjusted for age using the age correction factors for participants aged 18–24 years old. Age-corrected estimated $\text{VO}_{2\text{max}}$ (L/min) value was then multiplied by 1000 and divided by body mass (kg) to determine relative estimated $\text{VO}_{2\text{max}}$ (mL/kg/min). Cross-validation studies of the Åstrand-Ryhming cycle ergometer test have yielded a validity coefficient of 0.82 (Swain et al. 2004) and 0.84 (Cink & Thomas, 1981). Following the completion of the exercise test, participants were provided instruction for Session 3 and released from the laboratory.

Session 3

Participants returned for the 3rd session approximately 48 hours after the 2nd session. Session 3 involved blood pressure, blood glucose, blood lipids, body fat percentage, arterial stiffness, and systemic vascular resistance (SVR) assessment. Upon reporting to the laboratory following an overnight fast, each participant sat quietly for at least 5 minutes before blood pressure was measured with a digital blood pressure monitor (model HEM-907 XL, Omron) three times on each arm and the mean measurement for the left arm was recorded as the resting blood pressure. Bi-lateral measurements of blood pressure were used to determine the inter-arm difference as recommended by the American Heart Association (AHA, 2014) because an inter-arm difference >10 mmHg has been linked to peripheral vascular disease (Clark, Campbell, Evans, & Millward, 2006). However, results of inter-arm difference were not used for data analyses.

Fasting blood glucose, triglycerides, and cholesterol were measured once with an Alere Cholestech LDX system. An optics check was run on the system each day the Cholestech system was used to ensure the analyzer was functioning properly and a standard control solution (Alere Cholestech LDX Multianalyte control) was analyzed with each box of cassettes to confirm the accuracy of the system. Each cassette was removed from the refrigerator at least 10 minutes prior to use and allowed to warm to room temperature. Following a minimum of an 8 hour fast, participants sat quietly while the middle finger was cleaned with an alcohol pad then dried with sterile gauze. A lancet was used to puncture the finger for a capillary blood sample. The first drop of blood was discarded before a 35 μ L blood sample was collected with a capillary tube within 10 seconds. The whole blood was then transferred to the cassette and inserted into the Cholestech system for analysis of blood glucose, triglycerides, total cholesterol, non-high density lipoprotein (non-HDL) cholesterol, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein cholesterol (LDL) cholesterol, and total cholesterol/HDL ratio.

Body composition was measured using whole-body air displacement plethysmography (Bod Pod, Life Measurement Instruments, Concord, CA). Immediately prior to each test, the system was calibrated according to the manufacturer's specifications. The volume of the chamber was measured while empty followed by the measurement of a calibration cylinder of known volume (\approx 50 L). The system's associated scale used to determine body mass was also calibrated using weights of known mass (20 kg). Following the calibration procedure, each participant entered the chamber and sat as still as possible with hands on lap while body volume was measured. During testing, each participant wore only a minimal, tight-fitting swimsuit and a swim cap to reduce the potential error due to the expansion and contraction of air trapped within clothing and hair (King et al., 2006; Mattsson & Thomas, 2006). When comparing air

displacement plethysmography to hydrostatic weighing, cross validation studies have yielded a coefficient determination of 0.94 (Fields, Hunter, & Goran, 2000) and a correlation coefficient of 0.94 (Ginde et al., 2005).

Arterial stiffness was determined from peripheral PWV (m/s) and AIx (%) utilizing ECG and applanation tonometry (SphygmoCor, AtCor Medical). Participants lay in a supine position and rested quietly for 10 minutes. Blood pressure was measured in the supine position. The skin was cleaned using an alcohol prep pad and dried with gauze prior to the placement of electrodes on the suprasternal notch, the xiphoid process, and the left iliac crest. Once electrodes were placed, the radial pulse was located and marked on the right side of the body. With the participant's arm extended laterally, the distance between the suprasternal notch and radial pulse was measured in millimeters. The right carotid pulse was located and marked and the distance between the carotid pulse and the suprasternal notch was measured. Values were then entered into the SphygmoCor software program. To obtain PWV, the tonometer (17 mm) was placed on the mark for the radial pulse and a reading of at least 10 seconds was obtained. This was repeated for the carotid site.

Peripheral arterial stiffness was measured with PWV. Pulse wave velocity is calculated as the distance traveled divided by pulse travel time. Distance traveled is defined as the measured distance from the carotid site to the radial site. Simultaneous readings of ECG and pulse tonometry were overlaid. Time was determined from the ECG recording and measured from an R wave to the pulse recorded at the tonometer for the associated cardiac cycle. Based upon the time and distance from the R wave to the tonometer recorded pulse site, the velocity of the pulse wave was obtained. The main factors that influence PWV are thickness of the arterial wall and

lumen diameter. PWV measurement has been shown to be reliable and highly reproducible and a valid indicator of arterial stiffness (Asmar et al., 1995; DeLoach, & Townsend, 2008).

Augmentation index (AIx) was also obtained during this process (SphygmoCor, AtCor Medical) which examines aortic stiffness. When observed graphically, AP is the difference between the first and second systolic peak. AIx is AP divided by pulse pressure (PP) and expressed as a percentage (Figure 2.2). Because AIx is influenced by heart rate (HR), AIx was normalized to a HR of 75 bpm. PWV and AIx were also assessed post-exercise.

Another measure of interest was systemic vascular resistance (SVR). Prior research (Wittenburg, Montoya & Martinez, 2012) has reported a decreased response in SVR for obese and unfit young adults. However, these studies did not actively examine the SVR response during exercise. In this study, SVR was measured at rest and throughout exercise while the participant exercised on a lower limb recumbent cycle ergometer (Lode, Gronigen, The Netherlands) (Appendix D). SVR was obtained using a thoracic bioelectric impedance plethysmography technique (PhysioFlow system, Manatec Biomedical), a non-invasive method that analyzes the differences in blood volume within the arteries using a low amperage current. SVR_i was then created by normalizing SVR for body surface area and expressed as dynes · s/cm⁵/m². All measures were monitored continuously and were recorded at 15 second intervals. Measurement of cardiac output using the PhysioFlow has been shown to be highly accurate (Charloux et al., 2000; Kemps et al., 2008; Collette, Humeau, & Abraham, 2008). Physioflow electrode placement is shown in Figure 3.1; electrodes were placed on the following landmarks: a) the supraclavicular fossa (Z2), b) to the right and slight superior to the previously placed supraclavicular fossa electrode (Z1), c) the fourth intercostal space to the right of the sternum, d) the xiphoid process (Z3), e) the left anterior axillary line on the fifth intercostal space (EKG2),

and f) to the right of and slightly inferior to the previously placed xiphoid process electrode (Z4 & EKG3).

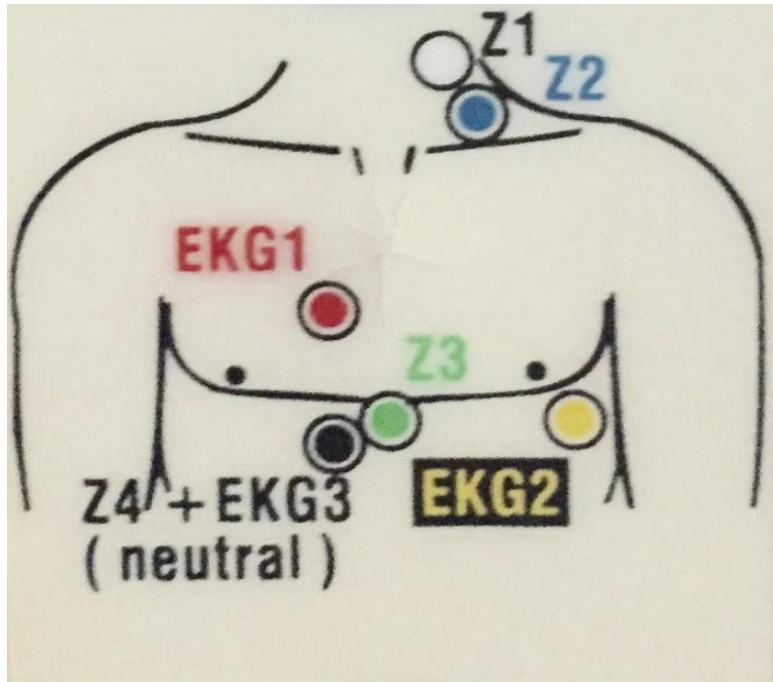


Figure 3.1. Physioflow electrode placement

In order to ensure a clear signal participants remained silent and still through 30 cardiac cycles. A one-minute pre-exercise baseline recording was obtained. At the end of the baseline data collections the participants began exercise at 50 RPM with a resistance of 60 watts. Exercise intensity was increased in 20 watt increments every 3 minutes. Participants were encouraged to continue to exercise until achieving 70% of heart rate reserve (% HRR). Once 70% HRR was achieved each participant continued to exercise until the end of the stage. Participants then performed a 3-minute cool-down before they ceased exercise. Heart rate, stroke volume, cardiac output, and SVR were recorded continuously during rest, exercise, and recovery. Cardiac output and SVR were normalized for body surfaced area and cardiac index and SVR_i were also obtained. Blood pressure was monitored and recorded before, during, and after exercise using an

automated system (Tango M2, Sun Tech Medical, Morrisville, North Carolina) at 1–2 minute intervals.

Design and Analysis

This cross-sectional study was designed to examine arterial stiffness and systemic vascular resistance for Hispanic young men. The dependent variables for this study were: PWV (m/s), AIx (%) normalized at 75 heart beats per minute, and SVRi, ($\text{dynes} \cdot \text{s}/\text{cm}^5/\text{m}^2$). SVRi was examined at rest, and during exercise at 70% HRR. Because some participants fatigued before achieving 70% HRR was reached, SVRi was also examined at 50% HRR.

A power analysis was performed to determine sample size. The design of this study is a matched-subjects design utilizing analysis of variance on three variables (PWV, AIx, SVRi). The calculated sample size with a power of 0.90 and an effect size of 0.95 was 40 (obese: $n=20$, non-obese: $n=20$). Based on BMI, each participant was placed in either the obese or non-obese group, which served as the independent variable. Each participant served as their own control when examining the differences in PWV and AIx (rest vs. post-exercise), and SVRi (rest vs. 50% HRR vs. 70% HRR). Mixed model ANOVAs were created to detect for significance between SVRi at rest, 50% HRR, and 70% HRR. Hierarchical multiple regression models were created with total cholesterol, body fat percentage, and WHR as the independent variables for each measure of stiffness. Between-group comparisons (obese vs. non-obese) were conducted with a mixed model analysis of variance. Pearson correlations were used to determine association between variables. The a-priori alpha level was set at ≤ 0.05 . The data were analyzed with SPSS (v. 23) and graphs were created with GraphPad Prism (v.6). Results are presented as mean \pm SD.

Chapter 4 – Results

Twenty-one obese Hispanic men aged 22.1 ± 2.5 years and 23 non-obese Hispanic men aged 21.4 ± 1.41 years participated in the study. To initially compare the obese group to the non-obese group, a MANOVA was conducted. There was no significant difference in age, height, physical activity level, HDL cholesterol, or fasting blood glucose between groups but estimated $\text{VO}_{2\text{max}}$ was significantly greater for the non-obese group ($p=0.006$). Total cholesterol was significantly greater for the obese group ($p=0.002$) as well as non-HDL cholesterol ($p=0.002$), LDL cholesterol, ($p=0.019$) and triglycerides ($p=0.002$). Body fat percentage, BMI, waist-to-hip ratio, waist circumference, and hip circumference were significantly greater for the obese group ($p<0.001$). A summary of participant characteristics is presented in Table 4.1.

Mean (\pm SD) resting hemodynamics values are presented in Table 4.2. Fifteen second intervals were averaged during 1 minute of rest for heart rate, SVR, SVRi, and cardiac index. The results of the MANOVA revealed no significant difference in systolic blood pressure, heart rate, cardiac index, or SVRi between groups. Diastolic blood pressure and mean arterial pressure (MAP) were significantly greater for the obese group ($p=0.02$ and $p=0.026$, respectively). Mixed model ANOVAs did not detect a significant difference in SVRi at rest (Table 4.2), at 50% HRR, or 70% HRR (Table 4.3) or SVRi percent change from rest to 50% HRR, rest to 70% HRR, or 50% to 70% HRR (Table 4.4). Mixed model ANOVA revealed significantly greater ($p=0.013$) peripheral PWV at rest for the obese group (8.49 ± 1.25 m/s) compared to the non-obese group (7.53 ± 1.17 m/s) and post-exercise, differences between groups were uniformly in the hypothesized direction (non-obese: 7.42 ± 1.7 m/s, obese: 8.16 ± 1.24 m/s, $p = 0.055$). There was a significant decrease ($p<0.001$) in aortic AIx post-exercise (non-obese: $-61.9 \pm 12.6\%$, obese: -

54.3±20.8%) compared to rest (non-obese: 2.4±17.0%; obese: 1.1±15.4%) for both groups with no significant between-group difference observed ($p>0.05$).

Table 4.1 Descriptive Characteristics of Non-Obese and Obese Hispanic Men

	Non-Obese (n=23)	Obese (n=21)
Age (years)	21.4±1.4	22.1±2.5
Height (m)	1.8±0.1	1.8±0.1
Body Mass (kg)	79.3±8.8	110.6±15.3*
Body Mass Index (kg/m ²)	25.9±3.1	35.4±4.9*
Body Fat (%)	21.3±8.1	35.3±7.2*
Waist Circumference (cm)	89.0 ±7.8	112.0±11.6*
Hip Circumference (cm)	103.3±7.2	118.1±7.8*
Waist-Hip-Ratio	0.9±0.1	1.0±0.1*
Physical Activity Level (MET minutes/Week)	409.7±221.8	340.4±244.5
Estimated VO _{2max} (ml/kg/min)	39.5±5.3	31.8±9.8*
Total Cholesterol (mg/dL)	156.5±29.3	185.9±24.7*
High Density Lipoprotein (mg/dL)	42.5±12.2	39.2±11.9
Triglycerides (mg/dL)	89.7±44.8	160.5±94.1*
Non-HDL Cholesterol (mg/dL)	114±33.5	146.2±22.8*
Low Density Lipoprotein Cholesterol (mg/dL)	99.1±28.4	117.7±17.5*
Fasting Blood Glucose (md/dL)	88.0±6.4	91.6±7.9

Data are mean ± SD. *Significantly different from non-obese group ($p<0.05$)

Table 4.2. Resting Hemodynamics and Arterial Stiffness of Non-Obese and Obese Hispanic Men

	Non-Obese (n=23)	Obese (n=21)
Heart Rate (bpm)	65.7±8.9	69.3±12.7
Systolic Blood Pressure (mm Hg)	114.2±13.0	119.5±8.6
Diastolic Blood Pressure (mm Hg)	69.7±7.3	75.8±9.6*
Mean Arterial Pressure (mm Hg)	84.5±8.7	90.4±8.3*
Cardiac Index	3.0±0.4	3.3±0.7
Systemic Vascular Resistance	1198.2±194.6	993.3±232.6*
Systemic Vascular Resistance Index	2362.1±364.5	2323.2±424.6
Pulse Wave Velocity Rest (m/s)	7.5±1.2	8.5±1.3*
Pulse Wave Velocity Post-Exercise (m/s)	7.4±1.7	8.2±1.2
Augmentation Index Rest (%)	2.4±17.0	1.1±15.4
Augmentation Index Post-Exercise (%)	-61.9±12.6**	-54.3±20.8**

Data are mean ± SD. *Significantly different from non-obese group (p<0.05). **Significantly different from rest (p<0.05).

For heart rate, cardiac index, and SVR_i during exercise, 15-second intervals were averaged during the last minute of the exercise stage in which 50% HRR and 70% HRR were achieved. During exercise there was no significant difference between groups for heart rate, blood pressure, or SVR_i. Cardiac index was significantly greater for the obese group compared to the non-obese group at 50% HRR (p=0.024) and 70% HRR (p=0.01). Percentage of HRR and heart rate did not differ between groups for either exercise intensity as summarized in Table 4.3

Table 4.3 Hemodynamics during Exercise of Non-Obese and Obese Hispanic Men

	50% Heart Rate Reserve		70% Heart Rate Reserve	
	Non-Obese (n=23)	Obese (n=21)	Non-Obese (n=20)	Obese (n=18)
Heart Rate (bpm)	132.1±5.2	132.3±6.3	158.6±6.3	155.4±10.5
Intensity (%)	49.7±2.7	49.1±3.3	69.5±4.7	68.2±4.9
Systolic Blood Pressure (mm Hg)	167.7±17.7	175.3±19.4	181.8±22.3	190.1±26.1
Diastolic Blood Pressure (mm Hg)	70.5±14.0	72.9±13.3	70.4±15.3	62.7±12.7
Mean Arterial Pressure (mm Hg)	102.9±11.1	107±12.6	107.6±10.9	105.2±12.8
Cardiac Index	6.8±0.6	7.7±1.7*	8.6±0.9	9.4±0.1*
Systemic Vascular Resistance	521.1±87.3	400.2±88.1*	423.1±80.3	329.9±80.3*
Systemic Vascular Resistance Index	1026.9±186.0	939.5±172.2	825.2±162.6	775.6±119.8

Data are mean ± SD. *Significantly different from non-obese (p<0.05).

Table 4.4 Systemic Vascular Resistance Index percent change

	Non-Obese (n=20)	Obese (n=18)
SVRi % Δ Rest to 50% HRR	-59.43±.43	-58.36±8.56
SVRi % Δ Rest to 70% HRR	-67.70±9.63	-66.23±6.87
SVRi % Δ 50% to 70% HRR	-7.81±4.21	-7.87±3.52

SVRi (Systemic Vascular Resistance Index) HRR (Heart Rate Reserve)

An initial evaluation of the relationships between variables (Table 4.5) indicated that SVRi was significantly correlated with age during rest (p=0.009) but not during exercise. Fasting blood glucose was significantly correlated with SVRi at 50% HRR (p=0.044) and 70% HRR (p=0.027) but was not significantly correlated with SVRi at rest. Correlations between measures of obesity and PWV are illustrated in Figure 4.1.

A significant negative correlation was observed between estimated $\text{VO}_{2\text{max}}$ and peripheral PWV ($p=0.007$). Peripheral PWV was significantly correlated with total cholesterol ($p=0.04$), triglycerides ($p=0.04$), and non-HDL cholesterol ($p=0.04$). AIX was significantly correlated with HDL cholesterol ($p=0.004$), and total cholesterol/HDL ratio ($p=0.05$).

Table 4.5 Pearson's Correlation Coefficients for Arterial Stiffness and Systemic Vascular Resistance of Non-Obese and Obese Hispanic Men

	PWV (m/s)	AIx (%)	SVRi Rest	SVRi 50%	SVRi 70%
Body Mass Index (kg/m ²)	0.50*	0.01	-0.19	-0.21	-0.13
Body Fat (%)	0.39*	0.01	-0.16	-0.16	-0.13
Age (yrs)	0.18	-0.01	0.34*	-0.21	0.17
Physical Activity Level (MET minutes/week)	-0.33*	-0.35*	0.15	0.02	-0.05
Resting Heart Rate (bpm)	0.35*	0.32*	-0.12	0.3*	0.44*
Systolic Blood Pressure (mmHg)	0.37*	0.20	0.21	0.37*	0.49*
Diastolic Blood Pressure (mmHg)	0.45*	0.16	0.16	0.37*	0.53*
Mean Arterial Pressure	0.46*	0.23	0.20	0.4	0.54*
Waist (cm)	0.52*	0.15	-0.12	-0.17	-0.10
Hip (cm)	0.38*	0.13	-0.17	-0.13	-0.11
Waist-to-Hip Ratio	0.53*	0.14	-0.05	-0.16	-0.06
Height (cm)	0.18	0.06	0.02	0.08	0.08
Estimated VO _{2max} (ml/kg/min)	-0.41*	-0.27	0.16	0.04	0.06
Total Cholesterol (mg/dL)	0.31*	-0.01	0.05	-0.01	0.03
High Density Lipoprotein (mg/dL)	-0.05	-0.42*	0.01	-0.01	-0.06
Triglycerides (mg/dL)	0.31*	0.19	0.08	0.20	0.30
Non-High Density Lipoprotein (mg/dL)	0.32*	0.15	-0.02	-0.03	-0.09
Low Density Lipoprotein (mg/dL)	0.29	0.21	-0.07	-0.11	-0.05
Total Cholesterol/High Density Lipoprotein Ratio	0.17	0.30*	0.04	-0.04	0.16
Glucose (mg/dL)	0.28	0.01	0.21	0.31*	0.36*

PWV (Pulse Wave Velocity), SVRi (Systemic Vascular Resistance Index), AIx (Augmentation Index) *Significantly correlated (p<0.05).

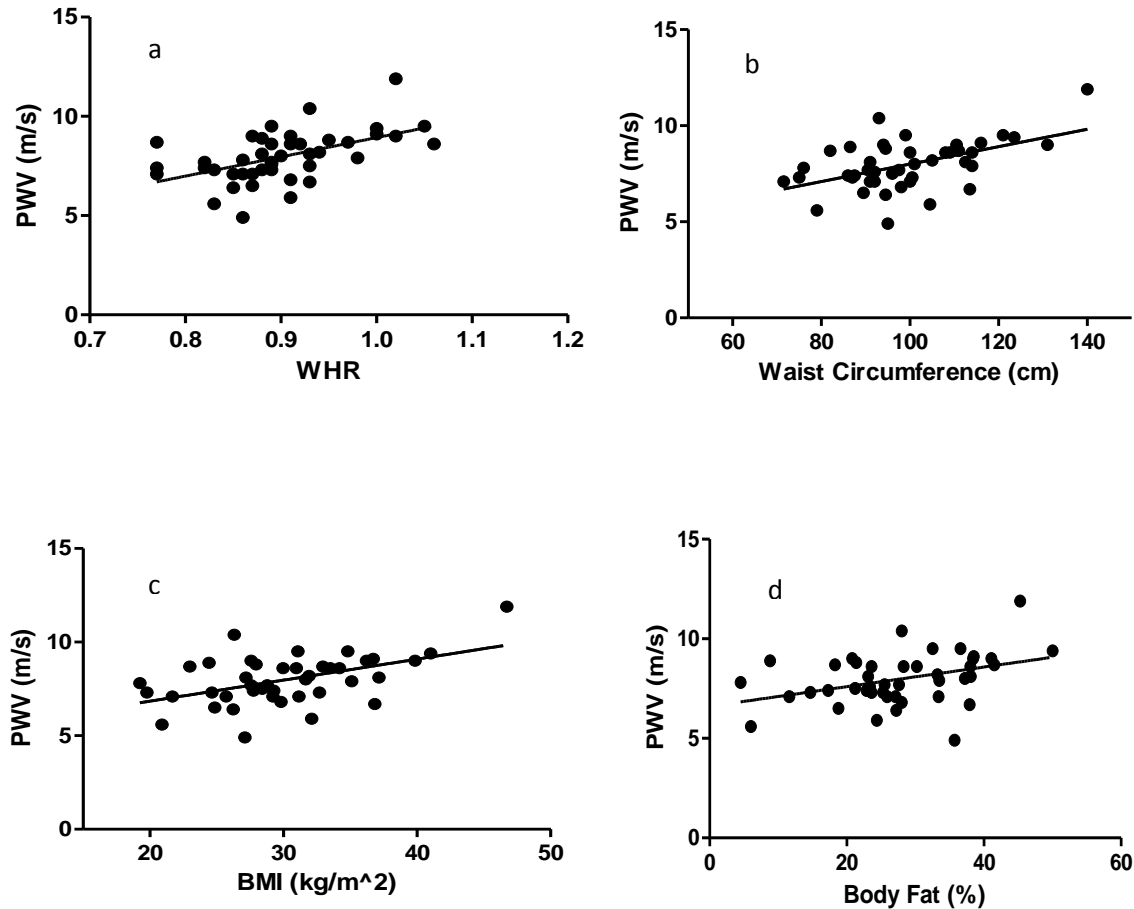


Figure 4.1 Correlations between PWV and a) WHR ($r=0.53$, $p<0.001$); b) waist circumference ($r=0.52$, $p<0.001$); c) BMI ($r=0.5$, $p=0.001$), and d) body fat percentage ($r=0.39$, $p=0.009$).

Stepwise regression with measures of obesity as the independent variables, and peripheral PWV as the dependent variable revealed WHR was the strongest predictor of peripheral PWV accounting for 28.5% ($p<0.001$) of the variability. A hierarchical multiple regression model was created with peripheral PWV as the dependent variable and WHR, body fat percentage, and total cholesterol as independent variables and accounted for 29.0% of the variability for PWV ($p=0.003$). Waist circumference was not entered into the regression model because of the high collinearity between waist circumference and every other measure of obesity. Summaries of the hierarchical multiple regression model is presented for PWV in Table 4.6.

Table 4.6 Multiple Hierarchical Regression Summary for Pulse Wave Velocity

	Model 1**			Model 2**			Model 3**		
Variable	B	SEB	β	B	SEB	β	B	SEB	β
Waist-to-Hip Ratio	9.99	2.57	0.53*	8.81	3.16	0.47*	8.83	3.27	0.47*
Body Fat (%)				0.01	0.02	0.10	0.01	0.02	0.11
Total Cholesterol (mg/dL)							0.00	0.01	-0.01
R²		0.29			0.29			0.29	

*p<0.05

**p<0.05 for model

Chapter 5 – Discussion

The two main purposes of this study were 1) to examine arterial stiffness for Hispanic, college-aged men and 2) to examine systemic vascular resistance (SVR) during exercise for Hispanic, college-aged men. Because obesity has been associated with increased arterial stiffness (Wildman et al., 2003; Czernichow, et al., 2005) and the reduced arterial compliance caused by arterial stiffness is associated with reduced cardiac output (Smith & Fernhall, 2011) it was hypothesized that 1) the degree of arterial stiffening would be greater for obese Hispanic young men compared to their non-obese counterparts and 2) obese Hispanic young men would have greater SVR after normalized for body surface area (SVR_i) at rest and during exercise. The main finding of this study was that peripheral PWV is linked to obesity for these sedentary and low physically active Hispanic men, aged 18–25 years. Body fat percentage, BMI, waist circumference, and WHR were positively correlated with peripheral PWV. The results of this study suggest that obesity is associated with peripheral vascular stiffening in young men. Stepwise regression indicated that WHR was the only predictor of peripheral PWV for these data. When WHR was removed from the model, waist circumference was the only predictor of peripheral PWV. Hierarchical multiple regression determined the strongest predictor of peripheral stiffness was WHR. Previous research for obesity and peripheral stiffness has been examined by Toto-Moukouro, Achimastos, Asmar, Hugues, and Safar (1986) who reported that peripheral PWV was significantly greater for hypertensive obese men and women aged 20–69 years compared to their non-obese counterparts, and that peripheral PWV was significantly correlated with BMI, which was the only measure of obesity recorded in their study.

Contradicting data exists for obesity and arterial stiffness depending on the measure of obesity utilized as well as the age and gender of the population examined. Czernichow et al.

(2005) reported that waist circumference, but not BMI, was a significant predictor of aortic PWV for French men and women aged 49–69 years. Wildman et al. (2003) reported that BMI was a stronger predictor of aortic PWV than waist circumference or WHR for 20–77 year old men and women. When examining younger and older groups separately, BMI continued to be the strongest predictor of aortic PWV for men and women aged 41–77 years. However, waist circumference was the strongest predictor of aortic PWV for 20–40 year old men and women (Wildman et al., 2003). The inconsistencies between predictors of arterial stiffness utilizing different measures of obesity may be due to one of BMI's limitations: the inability to discriminate between fat mass and fat-free mass. However, it consistently appears that central adiposity is a stronger predictor of arterial stiffness than total body fat when the distribution of adipose tissue is considered (Ferreira et al., 2004; Recio-Rodriguez et al., 2012). In a longitudinal study Ferreira et al. (2004) reported that waist circumference measured at adolescence and again at age 36 years was an independent predictor of carotid, femoral, and brachial artery stiffness in adulthood but that BMI and body fat percentage were not.

The conflicting results regarding the best predictor of arterial stiffness (Czernichow et al., 2005; Ferreira et al., 2004; Wildman et al., 2003) may be partially explained by gender differences in body fat distribution. Body fat distribution for men is associated with more abdominal, or central, adiposity (android obesity) which is more associated with the hypertrophy—enlargement—of fat cells. On the other hand, women tend to have an accumulation of gluteal adiposity (gynoid obesity) that is more associated with the hyperplasia—increased number—of fat cells (Krotkiewski, Björntorp, Sjöström, & Smith, 1983; Vague, 1956). Therefore, this may indicate that the predictors of arterial stiffness are different for men than for women, or that the distribution of adipose tissue is the more critical consideration; however, the

current study is unable to address a potential gender issue because women were not included in the participant pool.

Arterial stiffness might be one of the factors that links obesity with cardiovascular disease. Larsson et al. (1984) found that WHR was a stronger predictor of cardiovascular disease and all-cause mortality than BMI and body fat percentage. The hormone leptin may be a contributing factor for the greater PWV observed for the obese individuals of this and previous studies. Leptin was not measured in the current study but leptin is produced by adipocytes and research indicates that obese individuals have greater levels of leptin than non-obese (Smith, Al-Amri, Sniderman, & Cianflone, 2006). Additionally, hypertrophy of fat cells is more associated to leptin than hyperplasia and as mentioned, hypertrophy is more associated with abdominal adipose tissue than hyperplasia (Couillard, 2000). Windham et al. (2010) reported an association between leptin and PWV. Leptin receptors are located on endothelial walls (Sierra-Honigmann et al., 1998) and leptin has been shown to increase vascular calcification (Demer, & Tintut, 2008). Leptin has proinflammatory effects (Iikuni, Lam, Lu, Matarese, & La Cava, 2008) and increases oxidative stress (Bouloumie, Marumo, Lafontan, & Busse, 1999). Inflammation and oxidative stress have been associated with increased vascular stiffening of middle-aged men (Yamada et al., 2006).

Additionally, leptin increases sympathetic nervous activity (Haynes, Morgan, Mark, & Sivitz, 1997) which may increase sympathetic tone, possibly resulting in the acceleration and disintegration of elastin in the arterial wall and the accumulation of mechanical stress on the vascular system (Mitchell et al., 2004). In the current study, peripheral PWV and aortic AIx were positively correlated with heart rate. The association between heart rate and AIx has been previously reported (Cameron, McGrath, & Dart, A. M., 1998; Wilkinson et al., 2000; Gatzka et

al., 2001) due to the effect of heart rate on left ventricle ejection time. However, in the current study, aortic AIx was normalized for a heart rate of 75 bpm for all participants, therefore the positive correlation between resting heart rate and aortic AIx may suggest an association between arterial stiffness and increased sympathetic activity.

The significance of fat mass distribution could be related to the amounts of visceral adipose tissue (VAT). Sutton-Tyrrel et al. (2001) reported that VAT was a better predictor of arterial stiffness than total fat in older men (age 70–79 years). The association between VAT and arterial stiffness may be explained by insulin resistance. VAT is the principal fat store responsible for insulin resistance (Brunzell, & Hokanson, 1999). Like leptin, insulin increases sympathetic nervous activity. In addition to the increased sympathetic nervous activity that may accelerate the deterioration of elastin within the arterial wall as previously mentioned, insulin may stimulate hypertrophy of the vascular wall leading to the formation of collagen, and result in a propagation of arterial stiffness (DeFronzo, & Ferrannini, 1991). Although VAT was not specifically measured in the current study, waist circumference and WHR have been reported to be independent predictors of VAT for non-Hispanic White men and women (Janssen, Heymsfield, Allison, Kotler, & Ross, 2002; Pouliot et al., 1994), and Hispanic and non-Hispanic White men and women have similar levels of VAT when matched for waist circumference and BMI (Carroll et al., 2008). Future research is needed to determine if VAT is linked to arterial stiffness of young adult populations.

Peripheral PWV was negatively correlated with estimated VO_{2max} suggesting that cardiovascular fitness level is inversely associated with peripheral stiffness. Although all participants in the current study were sedentary or had a low physical activity level, there was a large range in estimated VO_{2max} which may be attributed to heredity (Klissouras, Pirnay, & Petit,

1973). Previous research has demonstrated a similar inverse relationship between aortic stiffness and $\text{VO}_{2\text{max}}$ for endurance trained men and women (aged 18–77 years) (Tanaka et al., 2000). Previous research by Vaitkevicius et al. (1993) also reported an inverse association between arterial stiffness (aortic PWV and aortic AIx) and $\text{VO}_{2\text{max}}$ of sedentary men and women (aged 21–96 years). Future studies are needed to determine if associations between arterial stiffness and $\text{VO}_{2\text{max}}$ occur independent of physical activity level.

A negative correlation was observed between physical activity level and peripheral PWV (Figure 5.1) which may indicate that for this group of Hispanic men, a small volume of physical activity may be enough to influence arterial stiffness. However, this correlation may not be meaningful given the cluster of data points at the end of each range. Additionally, previous research (Tanaka et al., 2010) reported no significant difference for arterial stiffness between sedentary and recreationally active men; only men who were highly trained showed significantly lower arterial stiffness when compared to sedentary and recreationally active counterparts. Therefore, it is not likely that the physical activity level for these subjects affected arterial stiffness.

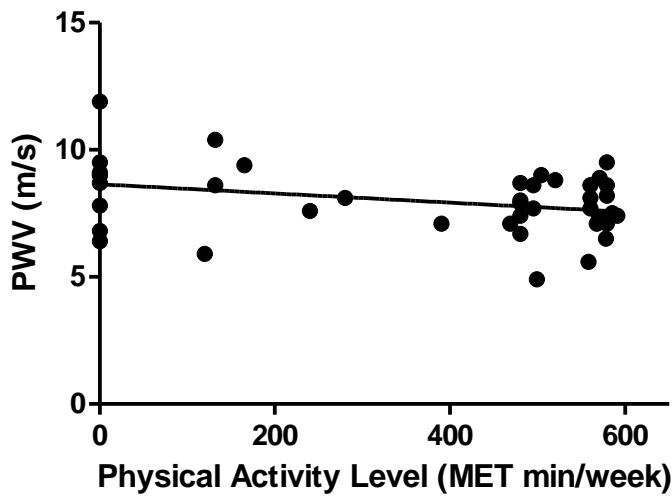


Figure 5.1. Correlation between PWV and physical activity level ($r=-0.33$, $p<0.031$)

Total cholesterol, non-HDL cholesterol and triglycerides were significantly positively correlated with PWV. HDL cholesterol was significantly negatively correlated and total cholesterol/HDL ratio was significantly positively correlated with aortic AIx. Similar results between lipids and arterial stiffness have been reported. Relf, Lo, Myers, and Wahlqvist (1986) reported a significant negative correlation between aortic compliance and total cholesterol as well as triglycerides, and a significant positive correlation between aortic compliance and HDL cholesterol by PWV Doppler ultrasound for healthy men aged 20–78 years. Wilkinson et al. (2002) reported that LDL cholesterol was a significant predictor of aortic AIx but not total cholesterol or HDL cholesterol. Unlike the current study where all participants were normocholesterolemic men, Wilkinson et al. (2002) examined normocholesterolemic as well as hypercholesterolemic men and women. The current and previous research suggests that the best blood lipid predictor of arterial stiffness may differ depending on the population being studied.

For these data, post-exercise AIx decreased similarly for the obese and non-obese men. Peripheral PWV was not significantly different at rest vs. post-exercise. Previous research

reported a post-exercise improvement for aortic PWV and whole-body arterial compliance of sedentary young men (aged 24 ± 6 years) following 30 minutes of exercise at 65% of $\text{VO}_{2\text{max}}$ independent of changes in MAP (Kingwell et al., 1997). The increased arterial compliance may have been due to smooth muscle relaxation which is regulated by nitric oxide (NO). The lack of significant decrease for peripheral PWV post-exercise for the current study may have been due to the duration and intensity of exercise. Due to the incremental workload of the current study, participants did not spend a large amount of time on high intensity exercise: total exercise time (including a 3 minute warm up) was 18.0 ± 3.4 minutes for the non-obese group and 19.8 ± 5.07 minutes for the obese group. Future research is needed to determine if a longer exercise duration or greater exercise intensity may result in decreased peripheral PWV for young Hispanic men.

The other main finding of this study was that although SVR ($\text{dynes} \cdot \text{s}/\text{cm}^5$) was significantly lower for the obese men at rest and during exercise, there was no significant difference when SVR was normalized for body surface area (SVR_i , $\text{dynes} \cdot \text{s}/\text{cm}^5/\text{m}^2$) between these obese vs. non-obese young Hispanic men at rest or during exercise. Because obese individuals have a greater cardiac output than non-obese individuals due to greater metabolic demands (Backman, Freyschuss, Hallberg, & Melcher, 1973), SVR_i was used to compare groups as has previous research that compared differences for SVR between obese and non-obese participants (Messerli et al., 1982). Frohlich et al. (1967) examined SVR between normotensive and hypertensive men and women at rest and reported a significantly greater SVR for the hypertensive group due to a decreased cardiac index as well as an increased MAP. Messerli et al. (1982) reported no significant difference for SVR_i between obese (aged 31.8 ± 3.1 years) and non-obese (aged 31.2 ± 2.3 years) men and women at rest, however SVR_i was not examined during exercise.

Cardiac index at rest was not significantly different between groups but MAP was significantly greater for the obese group. During exercise at 50% and 70% HRR cardiac index was significantly greater for the obese group but no significant difference in MAP between groups was observed. Similarly, Stelfox et al. (2006) reported no association between BMI and cardiac index at rest but cardiac index was not examined during exercise for their study. The differences in cardiac index between groups may indicate greater stress and exertion for the heart for obese men. Previous research (Vella, Paul, & Bader, 2012) reported no significant difference for cardiac index at rest between obese and lean Hispanic men and a significantly greater cardiac index during exercise despite a significantly reduced ejection fraction for obese men, suggesting greater cardiac stress for the obese men, which is a possible explanation for the results of the current study.

Previous research has reported that resting SVR increases with age but the age-related increase is not observed during exercise. Julius et al. (1967) found that at rest SVR was significantly greater for older men and women (aged 50–69 years) compared to middle-aged men and women (35–49 years), and that SVR for both older and middle-aged men and women was significantly greater compared to younger men and women (18–34 years) as a result of an age-related decrease of cardiac output. However, no significant difference was observed between groups during incremental exercise on a cycle ergometer (Julius et al., 1967). Similarly, the current study found a significant positive correlation between age and SVR_i at rest but no significant correlation was observed during exercise at 50% or 70% HRR. The association between SVR_i and age at rest for the current study is due to MAP, which was significantly positively correlated with age. No significant correlation was observed between cardiac index and age. Although exercise capacity decreases with age (Fleg et al., 2005) data reported by Julius

et al. (1967) suggests that although SVR_i at rest may increase with age, it is likely not a factor that contributes to the age-related decrease in exercise capacity. The results of the current study support this, even though participants from a narrow age range were examined.

SVR at rest and during exercise was not associated with aortic AIx or peripheral PWV. Although peripheral PWV was significantly greater for the obese group, mean peripheral PWV was within a normal range for both groups (Table 2.1). It may be possible that older or hypertensive populations with greater degrees of arterial stiffening may exhibit a more similar pattern for SVR, PWV, and AIx. The current study suggests that for healthy young men whose only cardiovascular risk factor is obesity, the degree of arterial stiffness does not influence SVR at rest or during exercise.

The majority of studies that have researched PWV have examined aortic stiffness but not peripheral stiffness, as did the current study. Additionally, previous reports of the association between aortic PWV and obesity were of children (Tounian, 2001), middle-aged adults (Czernichow et al., 2005; Feirrera et al., 2001) and the elderly (Sutton-Tyrrel et al., 2001; Acree et al., 2007) and few were focused on Hispanics (Krantz et al., 2011). The current study was novel due to the examination of young Hispanics and comparing obese to non-obese.

Limitations of this study were that participant physical activity level was self-reported and participants may have overestimated or underestimated the amount of exercise they engage in which has the potential of skewing the results. Another limitation is that VO_{2max} was estimated with a submaximal protocol, therefore VO_{2max} may have been overestimated or underestimated. Additionally, the current study grouped all men with a BMI ≥ 30 as obese and all other men as non-obese, resulting in some participants on the border of the cutoff point to be classified as

obese, which may have contributed to the lack of significant differences between groups for SVR_i and aortic AIX. Future studies are needed that examine these parameters between lean, overweight, and obese groups. Additionally, different results may be observed by examining morbidly obese Hispanic young men. Because peripheral stiffness was associated with central adiposity in the current study, future research is needed to determine if VAT is associated with arterial stiffness in young populations similar to results reported for older men (Sutton-Tyrrel et al., 2001). Furthermore, acute exercise resulted in increased compliance of the aorta (aortic AIX) but not peripheral compliance (peripheral PWV) for these Hispanic young men which may have been related to the exercise intensity or duration. Future studies are needed to determine if greater intensity exercise may more clearly delineate compliance differences between obese and non-obese Hispanic men.

Arterial stiffness and SVR are important factors for cardiovascular health. The main findings of this study were that for young, Hispanic men, SVR_i was similar for obese and non-obese men and that peripheral stiffness is greater for obese, young, Hispanic men with WHR being a stronger predictor of arterial stiffness than waist circumference, BMI, body fat percentage. The results of this study suggest that for young Hispanic men the distribution of adipose tissue may be a better predictor of arterial stiffness than total body fatness. These results for young Hispanic men reiterate the potential negative impact of central adiposity on the risk for cardiovascular disease.

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Appendix A

Cardiovascular Risk Factors Consent Form

You are being asked to participate in a research study that will assess college student cardiovascular risk factors. Please read this form carefully and ask any questions you may have before agreeing to participate.

Purpose of this study: The purpose of this study is to assess cardiovascular risk factors in college-aged men

What we will ask you to do: You will need to attend three sessions.

- First session:
 - You will be asked to fill out a medical questionnaire and a survey regarding your physical activity level.
- Second session (48 hours within first session):
 - You will be asked to walk and possibly jog on a treadmill on an incline. Do not exercise 12 hours before your session.
 - Height, weight, blood pressure, and waist and hip measurements will be taken.
- Third session (48 hours within second session):
 - You will be asked to come in fasted; no food 8 hours before your session; water is allowed. Additionally, no coffee, energy drinks, or any other caffeinated drinks 12 hours before your session, and no exercise 12 hours before.
 - During this session we will collect 2 vials of blood through a venous blood draw.
 - You will have your body composition assessed. You will be asked to sit quietly in a chamber with your hands on your lap for about a minute. You will be asked to wear a swim cap and spandex shirt and shorts during this procedure..
 - You will also have 9 ECG electrodes placed on the chest and neck area of your body.
 - We will be measuring the stiffness of your arteries and during this procedure you will be asked to lie still while blood pressure is taken at your wrist and neck using a stylus-device that measures the pulse.
 - You will be asked to exercise on a recumbent bicycle at a moderate to vigorous intensity.

Risks and Benefits:

There is a risk that you might feel uncomfortable while performing exercise if you are not accustomed to exercising. You may also experience slight muscle soreness a day or two after the exercise testing. Slight bruising at the sight of the blood draw may also occur, but it is not expected.

You will have the benefit of having your health risks for cardiovascular disease assessed which will include free bloodwork and physical activity plan.

Your information and answers will be confidential: The records of this study will be kept private. In any sort of report we make public we will not include any information that will make it possible to identify you. Only the researchers will have access to the records. The records will be kept secured on a computer and filing cabinet in the principle investigator's office for a period of five years and at the end of five years all records will be destroyed.

Taking part is voluntary: Taking part in this study is completely voluntary. If you decide to participate, you are free to withdraw at any time.

If you have questions: The researchers conducting this study are Maria Perez and Dr. David Wittenburg. Please ask any questions you have now. If you have questions later, you may contact Maria Perez at mariap0825@yahoo.com or at 312-350-286. You can reach Dr. Wittenburg at wittenbu@utep.edu or 915-747-7208.

You will be given a copy of this form to keep for your records.

Statement of Consent: I have read the above information, and have received answers to any questions I asked. I consent to take part in the study.

Your Signature _____ Date _____

Your Name (printed) _____

Signature of person obtaining consent _____ Date _____

Printed name of person obtaining consent _____ Date _____

This consent form will be kept by the researcher for at least three years beyond the end of the study.

Appendix B

Men's Health and Fitness Study

Looking to jump-start a healthier lifestyle? This study is for you

Open to Hispanic UTEP male students, ages 18 – 25

Study will examine

- cardiovascular disease factors
- body composition
- cardiovascular fitness
- blood pressure
- cholesterol and glucose

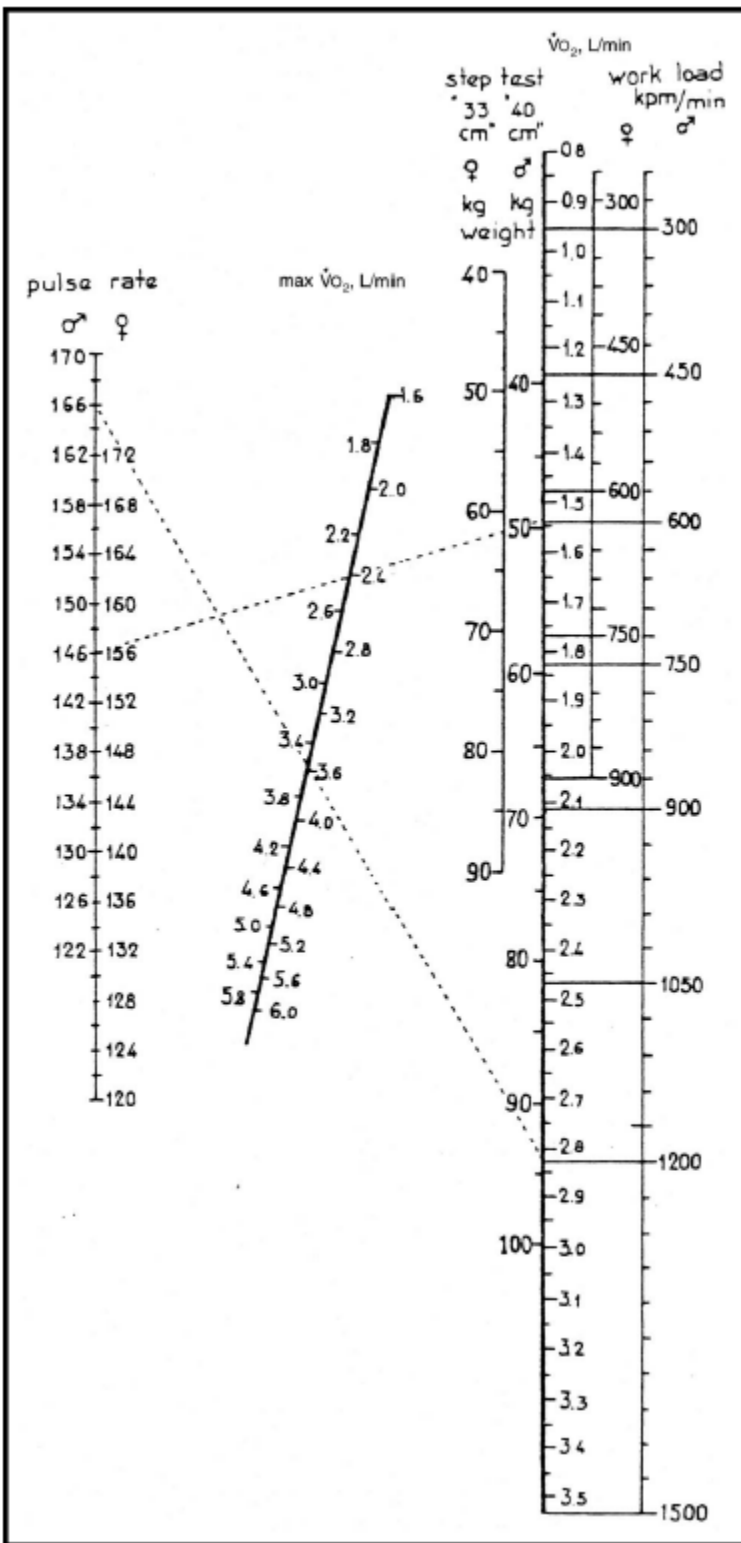
Participants will be compensated with FREE

- cardiovascular disease profile
- health-risk assessment
- activity recommendations

If interested, contact Maria Perez at mariap0825@yahoo.com or 312-350-2861 for more information



Appendix C



Appendix D



Appendix E

Health Information Survey

Thank you for taking the time to complete this survey. Please complete the following pages by providing the information that best represents your characteristics, attitudes, beliefs, and behaviors.

What is your age? _____

What is your gender? _____

What is your ethnicity? _____

Instructions: Please read and respond honestly to the following questions. The information you provide is personal and confidential for the purpose of this study. Thank you.

Are you currently taking any medication for high blood pressure as prescribed by a doctor? **YES or NO**

Are you currently taking any medication for diabetes as prescribed by a doctor? **YES or NO**

Are you currently taking any diet pills or weight loss supplements? **YES or NO**

Official Use Only

Investigator: Compute BMI and WHR and provide the following information.

Height: _____ cm

Weight: _____ Kg

BMI _____

Waist Circumference _____ cm

Hip circumference _____ cm

WHR _____

SYS _____ DIA _____ Pulse _____

Please answer each question even if you do not consider yourself to be an active person.

In answering the following questions,

- ❖ **vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. You are typically unable to carry on a conversation because the activity is so hard.
- ❖ **moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. A good way to measure this is if you can carry on a conversation but with some difficulty.

1a. During the last 7 days, on how many **days** did you do **vigorous** physical activities like heavy lifting, digging, aerobics, or fast bicycling?

Think about **only** those physical activities that you did for at least 10 minutes at a time.

_____ **days per week** **or** **none**

1b. How much time in total did you usually spend on **one** of those days doing vigorous physical activities?

_____ **hours** _____ **minutes**

2a. Again, think **only** about those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

_____ **days per week** **or** **none**

2b. How much time in total did you usually spend on one of those days doing moderate physical activities?

_____ **hours** _____ **minutes**

3a. During the last 7 days, on how many days did you **walk** for at least **10 minutes at a**

time without stopping? This includes walking at work and at home, walking to travel from place to place, and any other walking that you did solely for recreation, sport, exercise or leisure.

_____ **days per week** **or** **none**

3b. How much time in total did you usually spend walking on one of those days?

_____ **hours** _____ **minutes**

The last question is about the time you spent sitting on weekdays while at work, at home, while doing course work and during leisure time. This includes time spent sitting at a desk, visiting friends, reading, traveling on a bus or sitting or lying down to watch television.

4. During the last 7 days, how much time in total did you usually spend *sitting* on a weekday?

_____ hours _____ minutes

The Psychological Need Satisfaction in Exercise Scale

The following statements represent different experiences people have when they exercise. Please answer the following questions by considering how YOU TYPICALLY feel while you are exercising.

1: Strongly Agree 2: Agree 3: Neutral 4: Disagree 5: Strongly Disagree

1. I feel that I am able to complete exercises that are personally challenging. 1 2 3 4 5

2. I feel attached to my exercise companions because they accept me for who I am. 1 2 3 4 5

3. I feel that I share a common bond with people who are important to me when we exercise together. 1 2 3 4 5

4. I feel confident I can do even the most challenging exercises. 1 2 3 4 5

5. I feel a sense of camaraderie with my exercise companions because we exercise for the same reasons. 1 2 3 4 5

6. I feel confident in my ability to perform exercises that personally challenge me. 1 2 3 4 5

7. I feel close to my exercise companions who appreciate how difficult exercise can be. 1 2 3 4 5

8. I feel free to exercise in my own way. 1 2 3 4 5

9. I feel free to make my own exercise program decisions.	1	2	3	4	5
10. I feel capable of completing exercises that are challenging to me.	1	2	3	4	5
11. I feel like I am in charge of my exercise program decisions.	1	2	3	4	5
12. I feel like I am capable of doing even the most challenging exercises.	1	2	3	4	5
13. I feel like I have a say in choosing the exercises that I do.	1	2	3	4	5
14. I feel connected to the people who I interact with while we exercise together.	1	2	3	4	5
15. I feel good about the way I am able to complete challenging exercises.	1	2	3	4	5
16. I feel like I get along well with other people who I interact with while we exercise together.	1	2	3	4	5
17. I feel free to choose which exercises I participate in.	1	2	3	4	5
18. I feel like I am the one who decides what exercises I do.	1	2	3	4	5

Family Medical History Questionnaire

The next set of questions refers to your medical history and that of your parents and siblings. Please circle your response for the following questions. This is important information that assists us in analyzing your exercise data.

1. Have you ever been diagnosed with diabetes?

Yes

No

2. Has your father ever been diagnosed with Type 2 diabetes?

Yes *No* *Don't Know*

3. Has your mother ever been diagnosed with Type 2 diabetes?

Yes *No* *Don't Know*

4. Have any of your siblings ever been diagnosed with Type 2 diabetes?

Yes *No* *Don't Know*

5. Have you ever been diagnosed with coronary heart disease?

Yes *No*

6. Has your father ever been diagnosed with coronary heart disease?

Yes *No* *Don't Know*

7. Has your mother ever been diagnosed with coronary heart disease?

Yes *No* *Don't Know*

8. Have any of your siblings ever been diagnosed with coronary heart disease?

Yes *No* *Don't Know*

9. Have you ever been diagnosed with high blood pressure?

Yes *No*

10. Has your father ever been diagnosed with high blood pressure?

Yes *No* *Don't Know*

11. Has your mother ever been diagnosed with high blood pressure?

Yes *No* *Don't Know*

12. Have any of your siblings ever been diagnosed with high blood pressure?

Yes *No* *Don't Know*

Would you be interested in participating in a follow-up research study of this nature?

Yes or No

If *Yes*, please provide the following information.

Name: _____ (please print)

Email address: _____

Phone # _____

Thank you

Curriculum Vita

Maria Perez was born in El Paso, Texas. The daughter of Gregorio and Maria Perez, she graduated from Del Valle high school, El Paso, Texas. She received her bachelor's degree from the University of Texas at El Paso in May of 2006. She entered the kinesiology master's program in the spring 2013 semester and worked as a teaching assistant for the kinesiology program teaching Fitness Programs and Appraisals and Biomechanics labs.

Permanent Address:

11828 Jim Ryan

El Paso, Texas 79936

maperez@miners.utep.edu

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