12-2019

The Correlation Among Metabolic Syndrome Risk Factors, Hemodynamics, and Arterial Elasticity in Hispanic College Students

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THE CORRELATION AMONG METABOLIC SYNDROME RISK FACTORS, HEMODYNAMICS, AND ARTERIAL ELASTICITY IN HISPANIC COLLEGE STUDENTS

A Thesis

by

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Submitted to the Graduate College of
The University of Texas Rio Grande Valley
In partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

December 2019

Major Subject: Exercise Science

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December 2019

ABSTRACT

Mendoza, Paloma J., The Correlations Between Metabolic Syndrome Risk Factors (MetS), and Arterial Elasticity in Hispanic College Students. Master of Science (MS), December, 2019, 91 pp., 10 tables, 21 figures, references, 103 titles.

PURPOSE: The purpose of this study was to determine the correlation among MetS risk factors, hemodynamics, and arterial elasticity in Hispanic students attending The University of Texas Rio Grande Valley.

RESULTS: Systolic blood pressure (SBP), diastolic blood pressure (DBP), and fasting glucose (GLU) were the only components of MetS that had a significant correlation with large arterial elasticity (SPB: males: -.477 and females: -.503; DBP: females: -.300; $p<0.01$) and small arterial elasticity (DBP: males: -.257; GLU: males: -.272 , $p<0.05$). Augmentation index (AIx) and corrected augmentation index at 75 beat per minute (AIx@ 75) were significantly correlated with two components of MetS: SPB/DBP, and triglyceride levels (TRG). Overall prevalence of MetS was 6.9%. Prevalence of 1 or 2 MetS criteria was 35.4% and 9.2%, respectively. Low high-density lipoprotein cholesterol was the most prevalent criteria.

CONCLUSION: MetS risk factors significantly correlated with both arterial elasticity and hemodynamic components. Uniquely in this population, waist circumference and systemic vascular resistance was reversely associated.

KEYWORDS: MetS risk factors, resting blood pressure, waist circumference, fasting blood glucose, triglycerides, body composition, arterial elasticity, hemodynamics

DEDICATION

For my mother, who always asked “¿Cómo va la tesis?”

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CHAPTER I

INTRODUCTION

Metabolic syndrome (MetS) is defined as a constellation of several risk factors. According to the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III), a positive diagnosis of MetS requires the presence of three or more of the following risk factors: abdominal obesity through measurement of waist circumference, elevated triglycerides, depressed high-density lipoprotein (HDL) levels, hypertension, and impaired fasting blood glucose. Available evidence indicates that having MetS increases the risk of developing CVD, type II diabetes, having a stroke, and all cause of mortality (Wannamethee, Shaper, Lennon, & Morris, 2005).

There has been a gradual increase of MetS diagnosis in the US for all ethnicities. It was estimated that nearly 35% of all adults and 50% of 60 years of age have MetS (Aguilar, Bhuket, Torres, Liu, & Wong, 2015). However, the prevalence of MetS varies between races and ethnicities (Moore, Chaudhary & Akinyemiju, 2017). The rate of prevalence of MetS in the US is higher among Hispanics when compared to non-Hispanic Whites and African Americans (National Institutes of Health, National Heart, Lung, and Blood Institute [NHLBI], 2017). Specifically, the prevalence rate for Hispanic males and for Hispanic females are reported as 27.8% and 30.4% respectively (Roger et al., 2010; Campbell et al., 2016; Moore, Chaudhary, & Akinyemiju, 2017). Furthermore, in the Hispanic age group of 18-29, the prevalence of MetS is

higher for both males (12.4%) and females (10.9%) when compared to non-Hispanic Whites and African Americans (Moore, Chaudhary & Akinyemiju, 2017).

The underlying cause of MetS is believed to be multidimensional. However, obesity, specifically abdominal obesity, is believed to be the prime factor for the drastic rise of MetS risk factors (Ferdinand et al., 2013). Being overweight or obese creates additional complications, such as increased risk of CVD (Bluher, 2014). As the leading cause of death around the word, CVD usually manifests itself in late adulthood. However, risk factors for CVD such as elevated blood pressure, excessive body weight, and increased levels of serum cholesterol, may be present from an early age (Bao et al., 1997; Winkleby, Robinson, Sundquist, & Kraemer, 1999).

Not all ethnicities hold the same risk of developing CVD (Centers for Disease Control and Prevention [CDC], 2017; Wang et al., 2016; Mozaffarian et al., 2015; WHO, 2014). Perplexingly, while Hispanics have higher rates of obesity, hypoglycemia, diabetes, and lower rates of physical activity, (all factors that contribute to the development of cardiovascular problems), they experience lower rates of cardiovascular specific diseases and all-cause mortality than non-Hispanic Whites; an epidemiological phenomenon and current scientific mystery that has been coined the Hispanic Paradox (Cortes-Bergoderi et al., 2013; Borrell & Lancet, 2012).

Two known methods to help in the early detection of increased CVD risk are metabolic Syndrome (MetS) and arterial elasticity (Cecelja & Chowienczyk, 2012; Cernes, Zimlichman, & Shargorodsky, 2008, Saely, 2006). Arterial elasticity is an early, non-invasive marker predicative of cardiovascular events and related to the artery's ability to expand and recoil with cardiac pulsation and relaxation (Mceniery et al., 2006). The capacity of the arterial system to receive blood pumped from the heart is related to its ability to expand for a given pressure as well as its

size. When this capacity of the arterial system is reduced, elasticity of the artery is decreased, resulting in stiffness of the artery (Simone, 1997).

Although prevalence of MetS is increasing, college students are still argued to be the least studied age group. Furthermore, no study has investigated the relationship among MetS risk factors, hemodynamics, and arterial elasticity, focusing on young Hispanic college students in the US. Therefore, the aim of this study was to investigate the correlation among MetS risk factors, hemodynamics, and arterial elasticity, in young Hispanic college students.

Problem and Purpose Statement

As a precursor of CVD and type II diabetes, MetS is now considered as one of the public health concerns and it is highly prevalent among Hispanic population. Surprisingly, it is common among young college age individuals as well. Therefore, this study aims to investigate the relationship among MetS risk factors, hemodynamics, and arterial elasticity among Hispanic college students, an area currently lacking in the literature. To do so, one-hundred seventy-five students from The University of Texas Rio Grande Valley (UTRGV) were recruited and completed the study.

Study Purposes

The purposes of this study were to 1) investigate the correlation of MetS risk factors on arterial elasticity in a college-aged Hispanic population; 2) investigate the correlation of MetS risk factors on hemodynamics (mean arterial blood pressure, pulse pressure, resting heart rate, cardiac ejection time, stroke volume, stoke volume index, cardiac output, cardiac index, large artery elasticity, small artery elasticity, systemic vascular resistance, and total vascular impedance); 3) assess the prevalence of MetS risk factors in the UTRGV Hispanic student

population using the NCEP-ATP III definition; and 4) compare sex differences among arterial elasticity, hemodynamics, and MetS risk factors.

Significance of the Study

Studies show that the prevalence of MetS in the US is higher among Hispanics when compared to non-Hispanic Whites and African Americans (National Institutes of Health, National Heart, Lung, and Blood Institute [NHLBI], 2017). This study will help provide insight on which MetS risk factors are predominant in the Hispanic student population, as well as which factors yield more significance in their influence on arterial elasticity. No previous studies have been done on the correlation among arterial elasticity, hemodynamics and MetS risk factors, in the college aged Hispanic population.

This study will further help with ongoing studies that are trying to understand the Hispanic Paradox. This paradox is still a scientific mystery (Borrell & Lancet, 2012). The Hispanic Paradox is that while Hispanics have higher rates of obesity, hypoglycemia, diabetes, and lower rates of physical activity, (all factors that contribute to the development of cardiovascular problems), they experience lower rates of cardiovascular specific diseases and all-cause mortality than non-Hispanic Whites (Cortes-Bergoderi et al., 2013).

Assumptions

1. All subjects provided accurate information on the Medical History Questionnaire.
2. All equipment used was reliable and provided accurate results.
3. All subjects would be 8-hours fasted and hydrated for blood work and arterial elasticity measurements.
4. All participants would complete the study.

Limitations

1. The study may not be representative of the population due to convenience sampling rather than randomly sampled.
2. The study was limited to only UTRGV student volunteers.
3. Medical History was gathered through self-report.

Delimitations

1. Individuals taking medication that possibly affects resting blood pressure, resting heart rate, fasting blood glucose or was taking any kind of medication that affects any of the factors being looked at were not eligible to participate in the study.
2. Individuals younger than 18 and older than 45 were excluded from this study.
3. Individuals were required to be 8-hours fasted and adequately hydrated before measurements.

Research Questions

In order to test the hypotheses, the following research questions were addressed:

1.) Is there a correlation between MetS risk factors and arterial elasticity in the Hispanic population?

2.) Is there a correlation between MetS risk factors and hemodynamics in the Hispanic population?

3.) What correlative differences are there for MetS risk factors, arterial elasticity and hemodynamics between males and females?

Hypotheses

1.) There would be a negative correlation between arterial elasticity and abdominal obesity, triglyceride level, systolic blood pressure, diastolic blood pressure, and fasting glucose. There will also be a positive correlation between high density lipoprotein and arterial elasticity.

2.) The presence of MetS risk factors would negatively impact hemodynamics.

3.) There would be correlative differences in MetS risk factors, arterial elasticity and hemodynamics between males and females.

Operational Definitions

To aid the reader, the following terms are defined as used in this study:

1) **Arterial elasticity:** The measurement of the elastic properties of the arteries, which has an inverse relationship with arterial stiffness.
2) **Hydration:** Hydration status was deemed adequate when urine specific gravity measured 1.010 and lower as determined by a clinical urine refractometer.
3) **MetS risk factors:** Waist circumference (> 102 cm for males or > 88 cm for females), blood pressure ≥ 130 mm Hg systolic or ≥ 85 mm Hg diastolic or taking blood pressure-lowering medications, triglyceride level ≥150 mg/dL or taking lipid-lowering medications, fasting high-density lipoprotein cholesterol level < 40 mg/dL for males and < 50 mg/dL for females or taking lipid management medications and fasting blood sugar ≥ 100 mg/dL or drug treatment for diabetes mellitus.
4) **Body Composition:** Body fat percentage.
5) **Tanita:** Dual-frequency body composition analyzer.
6) **Hypertension diagnostic:** A noninvasive equipment that conducts measurements of arterial stiffness via placing a sensor on the radial artery.
7) **Refractometer:** A device used for analyzing urine specific gravity to measure hydration by measuring the concentration of a urine sample.
8) **Hemodynamics:** Analysis of physical aspects of blood circulation and blood flow.

9) **Pulse Wave Analysis (PWA):** A technique that allows the accurate recording of peripheral pressure waveforms and generation of the corresponding central waveform, from which the augmentation index and central pressure can be derived.

Summary

Two known methods to help in the early detection of increased CVD risk are MetS and arterial elasticity (Cecelja & Chowienczyk, 2012; Cernes, Zimlichman, & Shargorodsky, 2008, Saely, 2006). The NCEP-ATP III's definition of MetS strongly predicts future cardiovascular events and has recommended that all individuals should screen for abnormal lipid values, as well as MetS criteria at the age of 20 (National Cholesterol Education Program. 2001). Pathologic disturbances in the arterial system such as arterial stiffening of the arteries has been found to occur early in life and can contribute to the development of CVD morbidity and mortality (Ahmadizar, & Voortman, 2018; Grey et al., 2003; Arnett, Evans, & Riley, 1994; McEniery, 2006; McEniery et al., 2006; Sugawara et al.,2005).) Although Hispanics tend to be susceptible to biological factors that contribute to the premature genesis of CVD, they experience lower rates of cardiovascular specific and all-cause mortality than other ethnicities (Cortes-Bergoderi et al., 2013).

The purposes of this study were to 1) investigate the correlation of MetS risk factors on arterial elasticity in a college-aged Hispanic population; 2) investigate the correlation of MetS risk factors on hemodynamics (mean arterial blood pressure, pulse pressure, resting heart rate, cardiac ejection time, stroke volume, stoke volume index, cardiac output, cardiac index, large artery elasticity, small artery elasticity, systemic vascular resistance, and total vascular impedance); 3) assess the prevalence of MetS risk factors in the UTRGV Hispanic student population using the NCEP-ATP III definition; and 4) compare sex differences among arterial elasticity, hemodynamics, and MetS risk factors.

Chapter 2 contains a literature review on the, metabolic syndrome, abdominal obesity, triglyceride level, blood pressure, high-density lipoprotein, glucose, role of ethnicity, age and gender related to metabolic syndrome, arterial elasticity, and Hispanic Paradox. Chapter 3 presents a discussion of the methodology used in the study. Chapter 4 provides the results and discussion. Chapter 5 then summarizes the study, give conclusions, and suggests future research possibilities.

CHAPTER II

REVIEW OF THE LITERATURE

The purposes of this study were to 1) investigate the correlation of MetS risk factors on arterial elasticity in a college-aged Hispanic population; 2) investigate the correlation of MetS risk factors on hemodynamics (mean arterial blood pressure, pulse pressure, resting heart rate, cardiac ejection time, stroke volume, stoke volume index, cardiac output, cardiac index, large artery elasticity, small artery elasticity, systemic vascular resistance, and total vascular impedance); 3) assess the prevalence of MetS risk factors in the UTRGV Hispanic student population using the NCEP-ATP III definition; and 4) compare sex differences among arterial elasticity, hemodynamics, and MetS risk factors. Included in this review are the following topics: (1) Hispanic paradox, (2) metabolic syndrome, (3) abdominal obesity, (4) triglyceride level, (5) blood pressure, (6) high-density lipoprotein, (7) glucose, (8) role of ethnicity, age and gender related to metabolic syndrome, and (9) arterial elasticity.

Metabolic Syndrome

Almost two decades ago the NCEP ATP-III recognized MetS as an important component in the screening for CVD risk (2001). The term Metabolic Syndrome or Syndrome X was credited to G.M. Reaven in 1988, however scientists have been looking into what we now call MetS for almost a century (Reaven, 1988; Sarafidis, & Nilsson, 2006). MetS is a clustering of physical, clinical and laboratory signs that have the capacity to anticipate the development of CVD or cardiometabolic complications. To be clinically diagnosed with MetS, according to the

NCEP ATP-III criterion, individuals must have three or more of the five following risk factors: waist circumference > 102 cm for males or > 88 cm for females, blood pressure ≥ 130 mm Hg systolic or ≥ 85 mm Hg diastolic or taking blood pressure-lowering medications, triglyceride level ≥150 mg/dL or taking lipid-lowering medications, fasting high-density lipoprotein cholesterol level < 40 mg/dL for males and < 50 mg/dL for females or taking lipid management medications and fasting blood sugar ≥ 100 mg/dL or drug treatment for diabetes mellitus.

The prevalence of MetS on university campuses varies from ethnicity and location. In the study conducted by Topè and Rogers (2013), the prevalence of MetS in a historically African American college in Kentucky was at 12%. A total of 376 individuals between the ages of 18 to 24 participated in the study. At a university in Rhode Island, the prevalence of MetS was at 3.7% with the same age group (Fernandes, & Lofgren, 2011). In two studies conducted in two different Indian universities (located in Secunderabad, Telangana, and Kalkata, West Bengal), with the same age range of the previous studies, it was concluded that the prevalence was between 3.6% and 4.5%, respectively (Manjunath, 2014; Das Paul et al., 2017). Ruano and colleagues (2015), investigated the prevalence of MetS in medical students in a university in Ecuador. This study consisted of 883 students in their first, second or third semester in university. It was found that the prevalence was at 8.2% (Rauno, Perez, Mogrovejo, De Paula Morales, & Espinoza, 2015). In a study conducted in Korea, Cha and colleagues (2010), looked at the prevalence of MetS in overweight and obese university students. A total of 73 students participated in this study, from two different universities in Seoul. In this group, prevalence of MetS was 12%.

Abdominal Obesity

One-third of adults in the US are affected by MetS (Ervin, 2009). However, adults are not the only age group at risk. Even though CVD usually manifests in late adulthood, risk factors for

CVD such as elevated blood pressure, excessive body weight, and increased levels of serum cholesterol, may be present from an early age (Bao et al., 1997; Winkleby, Robinson, Sundquist, & Kraemer, 1999). Additionally, in the presence of MetS, the risk of developing CVD is multiplied by two, while the probability of developing type II diabetes is multiplied by five (Grundy, Hansen, Smith, Cleeman, & Kahn, 2004). The annual cost of treating adults with these risk factor clusters is a financial strain of approximately $5477 (Sullivan, Ghushchyan, Wyatt, & Hill, 2007).

The growth of MetS prevalence mirrors the growth of obesity because abdominal obesity is the major component that influences the likelihood of MetS risk factors (Yau, Castro, Tagani, Tsu, & Convit, 2012; Dalleck, & Kjelland, 2012; Huang et al., 2004). The study conducted by Rexrode and colleagues (1998) sought to determine the risk of coronary heart disease (CHD) associated with abdominal adiposity. The study consisted of 44,702 US female registered nurses participating in the Nurses' Health Study. A total of 320 CHD events were documented, with 251 myocardial infarctions and 69 CHD deaths. It was found that "both [waist-hip ratio] and waist circumference were independently associated with risk of CHD, even after controlling for BMI" (Rexrode et al., 1998, p. 1848). In a similar study which examined the increased risk of abdominal obesity in participants of the Heart Outcomes Prevention Evaluation (HOPE) Study without congestive heart failure, it was concluded that abdominal obesity was detrimental to the prognosis of individuals with CVD (Dagenais et al., 2005). In another study conducted by Lakka and colleagues (2002), which aimed to investigate the association of abdominal obesity and the risk of CHD in males, it was also determined that waist circumference was an independent risk factor for CHD.

Triglyceride Level

Triglycerides are obtained through food sources or synthesized within the body. In the body, triglyceride formation involves the attachment of fatty acids to a glycerol molecule through dehydration synthesis. Excessive amounts of triglyceride production and consumption can result in negative physiological events that can be overwhelmingly damaging to the body. The effect of prolonged elevated TRG has long been established to be associated with an increased risk of CVD. In a meta-analysis conducted by Hokanson and Austin (1996), seventeen studies, with a total of 57,277 participants, were evaluated to understand triglyceride level as a risk factor for CVD. This analysis consisted of studies that reported the association between fasting triglyceride level and incident cardiovascular endpoints. At the conclusion of the study it was found that "increased plasma triglyceride level is associated with a 32% increase in risk of [CVD] in [males] and a 76% increase in risk among [females]." (Hokanson, & Austin, 1996, p. 218). In 2003, Ninomiya and colleagues examined the associations of medical history of non-fatal myocardial infarction, and stroke. The data source being from Third National Health and Nutrition Examination Survey (NHANES III). Using the NCEP-ATP III criteria for MetS, 10,357 NHANES III participants were evaluated in total. It was concluded that an elevated level of triglyceride was significantly associated with myocardial infarction, and stroke (Ninomiya et al., 2004). Another study, using data from the Helsinki Heart Study, Manninen and colleagues (1992) further investigated the effect of baseline triglyceride and lipoprotein cholesterol on levels on the incidence of cardiac end points. It was concluded that "triglyceride concentration is a marker of elevated CHD risk" (Manninen et al., 1992, p. 44).

Blood Pressure

Globally, elevated blood pressure is estimated to cause about 7.5 million deaths. This accounts for approximately 12.8% of the total of all deaths (WHO, 2015). In the US, it is

estimated that roughly 75 million individuals (estimated one in three adults) have high blood pressure (Merai, et al., 2016). In the report from the American Heart Association (AHA), Heart Disease and Stroke Statistics—2015 Update, an estimated seven out of 10 individuals experiencing their first myocardial infarction and eight out of 10 individuals experiencing their first stoke have high blood pressure (Mozaffarian et al., 2015). Additionally, it was also reported that seven out of 10 individuals with chronic heart failure also have high blood pressure (Mozaffarian et al., 2015).

In the study by Wu and colleagues (2015), data obtained from the Taipei City Geriatric Health Examination Database was used to investigate the association between blood pressure and all-cause, CVD and expanded-CVD mortalities among older adults to establish and appropriate rage of blood pressure with the lowest mortality risk. The cohort study consisted of 77,389 adults older than 65 years. The study showed that systolic blood pressure between 120 to 129 mm Hg was associated with the lowest CVD mortality risk and that systolic blood pressure over 160 mm Hg or diastolic blood pressure over 90 mm Hg significantly increased CVD (Wu et al., 2015).

Similarly, a study conducted by Franklin and colleagues (2001), that compromised of 2,033 participants from the original Framingham Heart Study cohort and 4,506 from the Framingham Offspring Study set to combine both cohort studies to broaden the age range of the study to better define the roles of systolic blood pressure, diastolic blood pressure and pulse pressure as predictors to CHD risk in various age groups. There were five age groups in total: < 40, 40 to 49, 50 to 59, 60 to 69 and 70 to 79 year of age. In the 17 years of follow-up time, 807 participants developed CHD. After the analysis it was found that “[blood pressure] measured in young and middle-aged adults is positively related to CHD risk in later life implies that the risk of cardiovascular disease starts early in adult life” (Franklin et al., 2001, p. 1248). Moreover, it

was also revealed that "[diastolic blood pressure] is stronger than [systolic blood pressure] as a predictor of CHD risk in young adults, whereas the opposite is true in older persons, emphasizes the importance of the roles of both DBP and SBP in the staging of hypertension (Franklin et al., 2001, p 1248). Parallel results for diastolic blood pressure were found in another study which further emphasized a lower risk of CVD in respect to lower blood pressure (Macmahon, 1990).

High-Density Lipoprotein

More commonly referred to as the "good cholesterol", HDL acts as the garbage man of the blood vessels. Broadly, HDL grabs cholesterol and takes it to the liver to either be recycled or disposed of. Having an HDL cholesterol count of over ≥ 60 mg/dL is a negative risk factor for CVD however, an HDL cholesterol count below 40 mg/dL is a positive risk factor (Roger et al., 2012). The Framingham study was the first large scale study that presented evidence that low levels of HDL were a major risk factor for coronary artery disease (CAD) (Gordon, Castelli, Hjortland, Kannel, & Dawber, 1977). In another study, Gordon and colleagues (1989) further noted that with each increase by 10 mg/dL of HDL cholesterol count, the risk for CAD decreased by 2-3%. In follow-up study for the Framingham study by Wilson, Abbot and Castelli, low HDL cholesterol count was associated with increased mortality and it was speculated that "[e]xcess risk for coronary death may cluster among individuals with the lowest HDL-C levels" (1988, p. 714).

Glucose

Impaired fasting glucose (IFG) of ≥ 100 mg/dL is a positive risk factor for CVD (Roger et al., 2012). Prediabetes is GLU levels between 100 to 125 mg/dL with diabetes being diagnosed at a fasting blood glucose of greater than or equal to 126 mg/dL. While type II diabetes is predominantly preventable, once developed it is irreversible. According to the CDC,

about 84 million US adults fall under the prediabetic category (one in three adults), with about 90% of that population being unaware of it (2018). About one in 10 adults in the US have diabetes (around 30 million), with 90-95% of that population having type II diabetes (CDC, 2018).

In investigations based on the data for the Framingham cohort studies, individuals with IFG and diabetes had a higher risk of CVD, morbidity, and mortality; more so for females (Kannel, 1979, Levitzky et al., 2008). In the Korean Heart Study, Kim and colleagues, wanted to determine whether IFG was associated with increased risk of CVD, ischemic heart disease (IHD), and/or stroke. This study consisted of 408,022 participants from six different provinces in South Korea from 1996 to 2004. The results concluded that "IFG is associated with CVD risk independent of other CVD risk factors" (Kim, et al., 2013). Furthermore, in a study by Haffner and colleagues (1998), which compared the risk of myocardial infarction for participants with (1,059) and without type II diabetes (1,373) determining that individuals with "type 2 diabetes who have not had a myocardial infarction have a risk of infarction similar to that among nondiabetic patients who have had a prior myocardial infarction" (Haffner, Lehto, Rönnemaa, Pyörälä, & Laakso, 1998, p. 233).

Role of Ethnicity, Age & Gender Related to Metabolic Syndrome

In the US, the prevalence of MetS and its risk factors varies in non-Hispanic whites, non-Hispanic blacks and Hispanics, and variation in prevalence also fluctuates between ages groups of each ethnicity and gender. According to the NHANES 1988-2012, MetS prevalence for non-Hispanic white males, non-Hispanic black males, and Hispanics males jumped from 26.8% to 35.1%, 17.3% to 26.8% and 24.7% to 27.8%, respectively (Moore, Chaudhary & Akinyemiju, 2017). For non-Hispanic white females, non-Hispanic black females, and Hispanics females,

prevalence increased from 24.7% to 35.5%, 24.6% to 34.7%, and 29.8% to 30.4%, respectively (Moore, Chaudhary & Akinyemiju, 2017). Hispanic males and females showed the least amount of prevalence increase overall.

There are variations in the prevalence of MetS risk factors between males and females (Das Paul, Sen, Saha, & Chaudhuri, 2017; Fernandes, & Lofgren, 2011; Ali, Karim, & Mohammad, 2012; Morrell, Lofgren, Burke, & Reilly, 2012; Topè, & Rogers, 2013). In the Amsterdam Growth and Health Longitudinal Study by Ferreira and colleagues (2005), one of the aims of the study was to investigate the prevalence of MetS in 364 apparently healthy adults. It was found that the most common factor for males was hypertension, while for females it was low levels of HDL. In addition, in the previously mentioned study by Nieto and colleagues, it was seen that for both males and females, the more risk factors that were present, the higher blood pressure tends to be (Nieto et al., 2015).

It is known that naturally, there is a plethora of physical and physiological changes that occur with the progression of age. Consequently, there is disparity between age groups in the prevalence of MetS and its risk factors. In a study by Ervin (2009), the profiles of 3,425 individuals from NHANES 2003-2006 ages 20 and over were analyzed to determine the prevalence of MetS by sex, age, race, and ethnicity. In this study the age groups were as follow: 20 to 39, 40 to 59 and 60 and older. It was found that the prevalence for MetS increased for each age group with "20% of males and 16% of females under 40 years of age [meeting] the criteria for [MetS], 41% of males and 37% of females 40–59 years of age and 52% of males and 54% of females 60 years of age and over [meeting] the criteria" (Ervin, 2009, p. 3). Meaning that individuals ages 40 to 59 were three times more likely than individuals ages 20 to 39 to have MetS.

Arterial Elasticity

Arterial elasticity or arterial stiffness (reduced arterial elasticity) is an early, non-invasive marker predicative of cardiovascular events (Arnett et al., 1994; McEniery, 2006; McEniery et al., 2006; Sugawara, Hayashi, Yokoi, Cortez-Cooper, DeVan, Anton, & Tanaka, 2005). Increase in arterial stiffness disrupts the arterial system, leading to hypertension, and left ventricular hypertrophy; well established factors that accelerate the decline of adequate supply of blood to tissues and organs (Safar, Levy, & Struijker-Boudier, 2003). Arterial stiffness is related to the artery's ability to expand and recoil with cardiac pulsation and relaxation. The capacity of the arterial system to receive blood pumped from the heart is related to its ability to expand for a given pressure as well as its size. When this capacity of the arterial system is reduced, elasticity of the artery is decreased, resulting in stiffness of the artery (Simone, 1997). Several methods exist to measure arterial stiffness; although, each method has its own limitations. However, there is yet to be a gold standard for the measurement (Arnett et al., 1994; Hickler, 1990; Toto-Moukouo, Achimastos, Asmar, Hugues, & Safar, 1986). Calculating arterial stiffness via carotid-to-femoral pulse wave velocity (PWV) and pulse contour analysis are reliable ways to predict future CV events and all-cause mortality (Vlachopoulos, Aznaouridis, & Stefanadis, 2010; Prisant, Pasi Jupin, & Prisant, 2002). While both methods are acceptable, PWV comparing carotid and femoral signals can be more accurate since the velocity of which the pulse wave propagates through the arterial system is a direction function of wall stiffness (Asmar et al., 1995).

Arterial stiffness is a normal physiological occurrence in response to the natural aging process. (Vaitkevicius et al., 1993; Hayward, Avolio, & O'Rourke, 1989). As aging occurs, the elastin in the arterial walls is replaced by collagen (Cernes, Zimlichman, & Shargorodsky, 2008). Conversely, in young adults, increased arterial stiffness may be a risk predictor for CVD

development in late adulthood (Bao et al., 1997). Past studies have shown that MetS and its components affect the elastic integrity of the arterial walls, which may cause arterial stiffness due to the release of pro-inflammatory adipocytokines from visceral adipose tissue (Esper et al.,2006; Grassi, & Giannattasi, 2005; Ridker, 2004; Paragano et al., 2010). Conjointly, there is an undisputable link between arterial stiffness and arteriosclerosis, type II diabetes, MetS, hypertension, smoking, elevated insulin and sodium intake (Palombo, & Kozakova, 2016; Della-Morte et al., 2010; Alecu, et al., 2006; Mahmud, & Feely, 2002; Salvi, Giannattasio, & Parati, 2018; AlGhatrif et al., 2013).

In a study conducted by Markert et al. (2011), it was noted that age-dependent arterial dilation of Hispanics varied from the other ethnicities tested. Furthermore, it was speculated that aging, oxidative stress and changes in inflammatory and other age-related factors may have vary by ethnicity. Oxidative stress is characterized by an imbalance between free radicals and antioxidants. Oxidative stress is magnified in MetS and type II diabetes, which appears to underlie the development of these complications (Tangvarasittichai, 2015).

In the relationship of arterial elasticity and MetS risk factors in adults, it was found that large-arterial elasticity was predominately affected by hypertension, overall body fat percentage, and abdominal obesity; while small-artery elasticity was predominately affected by hypertension (Fjeldstad et al, 2007). Additionally, hypertension and obesity were more predictive of impaired arterial elasticity than any other MetS risk factor. Impaired large-artery elasticity was most prominent in those who had three of more MetS risk factors (Fjeldstad et al, 2007). In the investigation conducted by Gardner, Parker, Krishnan, and Chalmers (2013), the only element of MetS that was found to have a positive association with both large and small artery elasticity in

the group with MetS was elevated glucose levels, however that is not to say that it was the sole component that negatively affected arterial elasticity.

In the Bogalusa Heart Study, it was documented that there was an increasing trend in PWV with every additional component of MetS that individuals had. Those findings suggest that young adults with MetS carry the burden of the risk of CVD (Li, Chen, Srinivasan, & Berenson, 2005). Previous studies have additionally shown that abdominal obesity rather than overall obesity may be more of a risk for increased arterial stiffening, however, the presence of MetS has previously been found to be more strongly associated with arterial stiffness than just abdominal obesity (Schillaci et al., 2005; Ferreira et al., 2005). In an experiment by Krzesiński et al. (2015), in which cardiovascular hemodynamics were compared between males and females with a history of hypertension; females tended to have lower large-artery elasticity when compared to males.

Hispanic paradox

The leading cause of death in the US is CVD regardless of gender or race; with the exception of Asian or Pacific Islander and Hispanic or Latino whose leading cause of death is cancer (Centers for Disease Control and Prevention [CDC], 2017; Wang et al., 2016; Mozaffarian et al., 2015; WHO, 2014). Paradoxically, Hispanics were found to have a 24% lower all-cause death rate and lower death rates for nine of the 15 leading causes of death in the US, even though Hispanics have less education, and lower health-care access with higher poverty rates than non-Hispanic whites; as well as higher rates of obesity, hypoglycemia, diabetes, and lower rates of physical activity (Cortes-Bergoderi et al., 2013; Borrell & Lancet, 2012; Dominguez et al., 2015). Hispanics develop CVD at a lower rate despite being 24% more likely to have poorly controlled high blood pressure, have a higher prevalence of diabetes by

133% and 23% more likely to be obese in comparison to non-Hispanic whites (CDC, 2015; Dominguez et al., 2015).

First known as Hispanic Epidemiological Paradox, this anomaly was first brought to light in 1986 (Markides & Coreil, 1986). This perplexity being that despite having a paradigm of a poor cardiovascular risk profile, Hispanics experience lower all-cause and cardiovascular-specific mortality and live longer when compared to non-Hispanic whites. This paradox has been criticized due to the questioning of the accuracy of the data used to conclude the longevity of Hispanics, however systematic reviews confirm the existence of a Hispanic paradox (Abraído-Lanza, Dohrenwend, Ng-Mak & Turner, 1999; Arias, Eschbach, Schauman, Backlund, & Sorlie, 2010; Cortes-Bergoderi et al., 2013; Ruiz, Steffen, & Smith, 2013; Smith, & Bradshaw, 2006).

The study conducted by Arias and colleagues (2010) found that the all-cause mortality rate for Hispanics is approximately 20% lower than that of non-Hispanics. This study hypothesized that the Hispanic paradox could be explained due to poor data quality and that there were discrepancies in reported data for cause of death for Hispanics in the US. Data used for this study was from the National Longitudinal Mortality Study. After correction of death certificate misclassification, it was concluded that Hispanic mortality estimated based on US vital statistics was reliable, and that "[c]orrection for death certificate misclassification did not have a large effect on death rates for the Hispanic population" (Arias et al., 2010, p. 174). Concluding that Hispanics still lived longer than non-Hispanic white. There have been several longitudinal studies that resulted in contradictory findings in regards to relative all cause and cardiovascular-specific mortality. To address these contradictions, Ruiz and colleagues (2013) conducted a systematic review and meta-analysis of 58 longitudinal studies that included Hispanics for a total of 4.6 million participants where mortality was a reported outcome. The

omnibus test, after accounting for standard covariates, revealed that when compared to other ethnicities, the Hispanics had a 17.5% lower risk of mortality (Ruiz et al., 2013). The existence of the Hispanic paradox presents an undeniable opportunity to detect predictive factors for CVD that may be applicable to other ethnicities.

Conclusion

In conclusion, the review was able to highlight the degenerative effects of MetS and its individual risk factors which are seldomly present independently, but rather interrelated in a complex matrix that compromises the integrity of the arterial system. The literature presented on arterial elasticity underscored the physiological impedance of natural processes with prolonged expose to the risk factors. MetS is an early predictor of CVD with each risk factor independently increasing the risk of CVD and/or CHD. And with prevalence of MetS varying between ethnicity, age and gender, more exploration is needed to determine the vulnerability of the Hispanic population to the aforementioned risk factors, and their decreased susceptibility to CVDs.

Chapter 3 contains a discussion of the methodology used to conduct the present study. In chapter 4, the results of the study are presented and discussed. Chapter 5 contains a summary of the study, conclusions that were drawn, and recommendations for future research related to metabolic syndrome and arterial elasticity in Hispanics.

CHAPTER III

METHODS

The purposes of this study were to 1) investigate the correlation of MetS risk factors on arterial elasticity in a college-aged Hispanic population; 2) investigate the correlation of MetS risk factors on hemodynamics (mean arterial blood pressure, pulse pressure, resting heart rate, cardiac ejection time, stroke volume, stoke volume index, cardiac output, cardiac index, large artery elasticity, small artery elasticity, systemic vascular resistance, and total vascular impedance); 3) assess the prevalence of MetS risk factors in the UTRGV Hispanic student population using the NCEP-ATP III definition; and 4) compare sex differences among arterial elasticity, hemodynamics, and MetS risk factors

Subjects

A total of 175 Hispanic students between the ages of 18 to 43 participated in the current study. Subjects were recruited from the University of Texas Rio Grande Valley campus in Brownsville. The University of Texas Rio Grande Valley Institutional Review Board approved the study procedure for Human Subjects. All subjects read and signed an informed consent before any measurement took place. Subjects' participation was completely voluntary and were allowed to withdraw at any time without consequence. Total time commitment was approximately 1 hour and a half to 3 hours and forty-five minutes, for a total of 2 sessions.

Inclusion Criteria

1. Participants who were within the 18 to 45-year age range.
2. Hispanic ethnicity.
3. Were enrolled as a UTRGV student.

Exclusion Criteria

A participant was excluded from the study if s/he

1. was taking medication for hypertension, cardiovascular disease, chronic pain, or cardiometabolic disorder.
2. was taking medication that may interfere with vascular function.
3. was taking nonsteroidal anti-inflammatory drugs (NSAID).
4. was outside the 18 to 45-year age range.
5. Identified self as anything other than Hispanic.

Recruitment

Participants were recruited from The University of Texas Rio Grande Valley through classroom recruitment in which the professor permission script and in-person script was used. Participants were also recruited by means of fliers (see appendix for flier). Participation in this study was voluntary and participants were allowed to withdraw at any time.

Experimental Protocol

All study procedures were conducted in the Exercise Science Laboratory (M-1 building, room 216 in UTRGV Brownsville Campus). Appointments were agreed on by the subject and researcher. The study consisted of two sessions.

On the first day, participants were required to read and sign the informed consent form, of which they were provided a hard copy. Participants then completed a Medical History form.

Participants who were taking any medication that affected hemodynamics were excluded from the study. Contact information was then attained along with demographic data: age, ethnicity, gender, major, year in school. Next, two anthropometric measures were taken: height, and waist and hip circumference.

A Charder HM200P Portstad Portable Stadiometer (Charder Electronics Co., Ltd., Taichung, Taiwan), was used to measure height. Participants were asked to remove shoes; thin socks were permitted. Participants were asked to put their hair down and low to the head. Next, they stepped onto the platform scale and turned away from the stadiometer. Participants stood as tall as possible with heels together, feet evenly balanced and hands on their hips. Then, participants were instructed to inhaled deeply and maintained that position. The hinged lever was then lowered until it made contact with the crown of the head. Height was recorded to the closest .1 cm.

To measure hip and waist circumference a Gulick tape was used. American College of Sports Medicine (ACSM) guidelines were followed. For hip circumference participants were asked to stand erect with their feet together, a horizontal measure was taken at the maximal circumference of the buttocks. For waist circumference, participants were asked to stand with their arms at their sides, feet together, and abdomen relaxed, then a horizontal measure was taken at the narrowest part of the torso (above the umbilicus and below the xiphoid process. Alternating duplicate measurements were taken at each site; if the measurements were not within 2 mm at each site, additional measurements were taken. This session would last for 45 minutes.

The second day consisted of collecting measurements using SphygmoCor® CPV Pulse Wave Analyzer (AtCor Medical, Itasca, IL, USA), HDI/Pulse Wave CR-2000 TM Research

Cardio Vascular Profiling System (Hypertension Diagnostic, Inc., Eagan, Minnesota, USA), DC-430U Dual Frequency Total Body Composition Analyzer (Tanita, Tokyo, Japan), and Cholestech LDX® Analyzer (Alere, Hayward, California). Before arriving for session two, participants were sent a reminder to be hydrated, fasted for a minimum of 8 hours, wear a thin loose-fitting shirt and shorts and obtain from exercising before their session. Participants were also instructed to have a full night's sleep and refrain from any caffeine or alcohol consumption for 24 prior to their appointment. Participants who could not fulfill any of these requirements were rescheduled. After verification, hydration was measured. This session would last for 45 minutes to 3 hours.

Hydration was monitored with the use of a urine S.G. refractometer, Pocket Refractometer 4410 PAL-10S (Atago Co., Ltd., Tokyo, Japan), which required participants to provide a urine sample to determine the level of current hydration (hydration was at or below 1.010). Second, body composition was measured via bioelectrical impedance (Tanita DC-430U). Participants were instructed to remove their footwear and socks and stepped on the platform with their forefeet and heels on the electrodes.

When performing measurements using SphygmoCor™ and HDI/Pulsewave™, participants were asked to lie down in the supine position for 5 to 10 minutes and baseline arterial elasticity and hemodynamics were measured using HDI/Pulsewave™ (noninvasive equipment conducts measurements of arterial stiffness via placing a sensor on the radial artery at the right wrist and a cuff to the left arm to measure blood pressure) and pulse wave analysis (PWA) using SphygmoCor™. During these measurements, participants were asked to be as relaxed as possible, but to stay in the same position with minimal to no movement for accurate results. If participants significantly moved during the testing, the results were voided and the test was re-done.

Following these measurements, a blood sample was collected. Participants were instructed to wash hands with warm water and soap and rinsed thoroughly. Then the ring finger was cleaned with an alcohol swab and allowed to air dry. The skin was then punctured using lancets, and fingerstick samples was collected into heparin-coated 40-mL capillary tubes. Blood was allowed to flow freely from the fingerstick into the capillary tubes without milking of the finger. Samples were then placed immediately into the Cholestech LDX analyzer. The Cholestech LDX analyzer measured total cholesterol, HDL cholesterol, LDL cholesterol, triglycerides, and blood glucose. A daily optics check was performed on the LDX Cholestech analyzer used for the study. The method is certified by the Centers for Disease Control and Prevention's (CDC) Cholesterol Reference Method Laboratory Network (CRMLN) (Shepherd MD, Mazzachi BC, Shephard AK, 2007; Carey M, Markham C, Gaffney P, Boran C, Maher V, 2006).

Each equipment was calibrated according to instructions provided by the manufacture before each use.

Instruments

Clinical Urine Refractometer

Participants were required to provide a urine sample at the beginning of the second session. Hydration was measured by using 5-7 drops of the urine sample on to the lens of the urine refractometer. The device was then shielded from any light source for 30 seconds and the level of refraction, within the sample was recorded. The device was then cleaned and the rest of the urine was discarded into the biohazard waste.

HDI/PulseWave CR-2000TM Research Cardiovascular Profiling System

Participants were instructed to lie in the supine position with their arms abducted from their bodies by roughly 15 degrees and legs separated comfortably while an appropriately sized blood pressure cuff was placed around the participants left upper-arm, and a rigid plastic wrist stabilizer was placed on their right wrist to minimize wrist movement and stabilize the radial artery during the measurement. An Arterial Pulsewave™ Sensor was placed on the skin directly over the radial artery at the point of the strongest pulse, while the arm rested in a supine position. The sensor was adjusted to signal strength of 18-22 before each recording. When recording, the participants would be required to stay as still as possible for a duration of about a minute, in which blood pressure would be taken followed by a 30 second recording. The device measured blood pressure, heart rate, stroke volume, left ventricular ejection time, systemic vascular resistance, total vascular impedance, and small and large arterial elasticity.

Pulse Wave Analysis

PWA was conducted noninvasively using a pulse wave analyzer; SphygmoCor® Pulse Wave Analyzer (AtCor Medical Pty. Ltd., Sydney Australia). Measurements were taken to determine peripheral pressure waveforms and for the generation of the corresponding central waveform.

Body Composition Analyzer

A body composition analyzer. Participants removed all personal items from their person and stood bare foot at the base of the unit which has four stainless steel circular foot-pad electrodes fasted to a metal platform set on force transducers. Measurements recorded included weight (kg), body mass index (BMI), body fat percentage, body fat mass (kg), fat free mass (FFM) (kg), muscle mass(kg), physique rating, body water percentage, total body water mass

(TBW), basal metabolic rate (BMR), metabolic age, bone mass(kg), visceral fat rating, bioelectric data.

Alere Cholestech LDX® System

The Cholestech LDX System uses reflectance photometry (the amount of light reflected from a solid surface) to measure the amount of substances in blood. For this study Lipid Profile•GLU cassettes were used. Data obtained: total cholesterol, HDL levels, triglyceride levels, LDL levels, non-HDL levels, LDL/HDL ratio, and fasting glucose levels.

Statistical Analysis

Data were entered into Microsoft Excel spreadsheet (Microsoft, Redwoods, WA, USA) and accuracy was checked. Analyses were performed using Windows based software, version 23 (Statistical Package for the Social Sciences Inc, IBM Corporation, New York, USA). Demographics were calculated using median and frequencies. Unless otherwise indicated, data are presented as mean ± SD, or percentages. A one-way analysis of variance (ANOVA) was used to determine if there were any statistically significant differences between the means. Using Pearson Correlation Coefficient, the relationships between individual MetS criteria, hemodynamics, and arterial elasticity variables were assessed. An alpha of ≤ 0.05 was used to determine statistical significance.

CHAPTER IV

RESULTS

The purposes of this study were to 1) investigate the correlation of MetS risk factors on arterial elasticity in a college-aged Hispanic population; 2) investigate the correlation of MetS risk factors on hemodynamics (mean arterial blood pressure, pulse pressure, resting heart rate, cardiac ejection time, stroke volume, stoke volume index, cardiac output, cardiac index, large artery elasticity, small artery elasticity, systemic vascular resistance, and total vascular impedance); 3) assess the prevalence of MetS risk factors in the UTRGV Hispanic student population using the NCEP-ATP III definition; and 4) compare sex differences among arterial elasticity, hemodynamics, and MetS risk factors

Subject Characteristics

Two hundred eight participants were recruited for the study and one hundred seventy-two completed the study; sixty-four males and one hundred eight females. Participants were recruited from the University of Texas Rio Grande Valley at the Brownsville campus.

Table 1. Anthropometric Measures

Variables	Male (n = 69 - 76)	Female (n = 128 - 132)
Age (years)	23.0 (± 4.0)	22.2 (± 3.7)
Height (cm)	175.6 (± 7.6)	159.9 (± 5.7)**
Waist Circumference (cm)	86.4 (± 11.0)	78.5 (± 13.4)**
Hip Circumference (cm)	102.1 (± 8.6)	102.9 (± 12.1)

Note. Sample sizes varied due to missing data for some assessments.
Values are reported as means (±SD).
*p < 0.05; **p < 0.01 indicates differences between males and females.

Table 2. Body Composition

Variables	Male (n = 52 - 65)	Female (n = 107 - 113)
Weight (kg)	83.4 (± 15.2)	68.1 (± 17.8)**
Body Fat Percentage (%)	19.2 (± 6.2)	30.4 (± 7.3)*
Fat Mass (kg)	16.7 (± 8.2)	21.8 (± 11.4)*
Fat Free Mass (kg)	66.6 (± 8.4)	46.2 (± 7.0)**
Muscle Mass (kg)	63.3 (± 8.0)	43.8 (± 6.6)**
Visceral Fat Rating	4.9 (± 3.5)	3.3 (± 2.8)*
Degree of Obesity (%)	24.3 (± 19.6)	21.0 (± 28.1)
Body Mass Index (kg/m^2)	27.1 (± 4.2)	26.5 (± 6.2)

Note. Sample sizes varied due to missing data for some assessments.
Values are reported as means (±SD).
*p < 0.05; **p < 0.01 indicates differences between males and females.

Metabolic Syndrome Risk Factors and Biochemical Measures

For males, there was a positive, but moderately significant correlation between WC and total cholesterol (TC) ($r = .457$, $p<.01$), and low-density lipoprotein (LDL) levels ($r = .419$, $p<.01$); a positive and strong significant correlation between WC and TRG levels ($r = .630$, $p<.01$); a negative, but moderate significant correlation between WC and HDL levels ($r = -.429$, $p<.01$); and a positive, but weak significant correlation between WC and age ($r = .308$, $p<.01$). For females, there was a positive, but weak significant correlation between WC and TC ($r = .204$, $p<.01$), LDL ($r = .244$, $p<.01$) and GLU levels ($r = .345$, $p<.01$); a positive, but moderate significant correlation between WC and TRG levels ($r = .491$, $p<.01$); and a negative, but weak significant correlation between WC and HDL levels ($r = -.368$, $p<.01$).

For males, there was a positive, but moderate significant correlation between TRG levels and LDL levels ($r = .457$, $p<.01$); a positive and stronger significant correlation between TRG levels and WC ($r = .630$, $p<.01$), and TC ($r = .631$, $p<.01$); and a negative, but weak significant correlation between TRG levels and HDL levels ($r = -.386$, $p<.01$). For females, there was a positive, but weak significant correlation between TRG levels and LDL levels ($r = .293$, $p<.01$) and GLU levels ($r = .271$, $p<.01$); a positive, but moderate significant correlation between TRG levels and TC ($r = .437$, $p<.01$); and a negative, but weak significant correlation between TRG levels and HDL levels ($r = -.214$, $p<.05$).

For males, there was a positive, but weak significant correlation between SBP and TC ($r = .324$, $p<.01$) and LDL levels ($r = .260$, $p<.05$). For females there is a positive, but weak significant correlation between SBP and GLU levels ($r = .229$, $p<.05$)

For males, there was a positive, but weak significant correlation between DBP and TC ($r = .347$, $p<.01$), and LDL levels ($r = .300$, $p<.05$). For females there was a positive, but weak significant correlation between DBP and TC ($r = .214$, $p<.05$).

For males there was a negative, but weak significant correlation between HDL levels and LDL levels ($r = -.346$, $p<.01$). For females there was a positive, but weak significant correlation between HDL and TC ($r = .196$, $p<.05$); and between age and GLU levels ($r = .202$, $p<.01$); and a negative but weak significant correlation between HDL levels and GLU levels ($r = -.195$, $p<.05$).

Table 3. Biochemical Measure

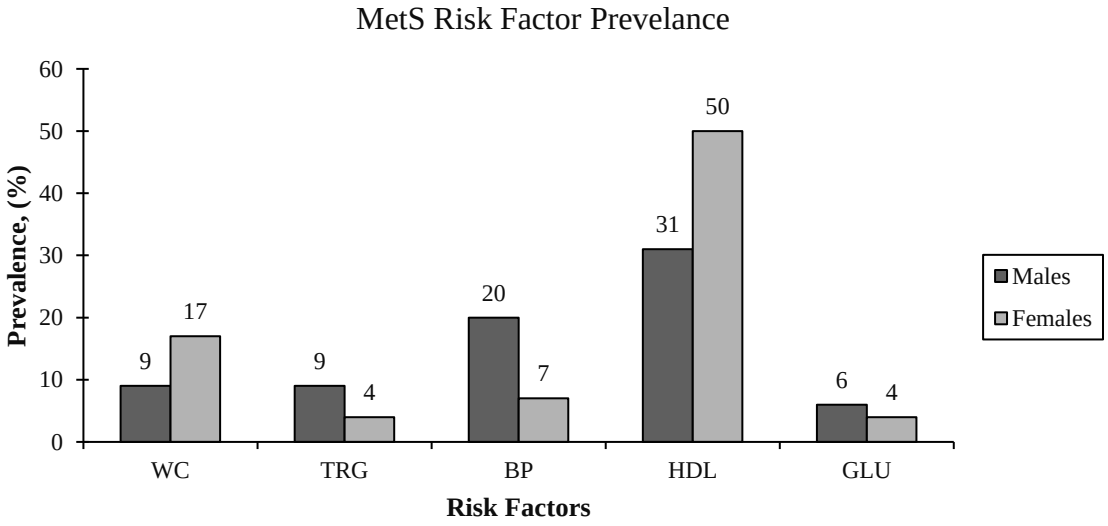
Variables	Male (n = 65)	Female (n = 114)
Total Cholesterol (mg/dL)	159.8 (± 32.0)	160.1 (± 29.6)
HDL (mg/dL)	44.6 (± 9.9)	50.5 (± 11.2)*
Triglyceride (mg/dL)	80.8 (± 56.0)	76.7 (± 38.3)
LDL (mg/dL)	99 (± 28.2)	94.4 (± 26.4)
Non-HDL (mg/dL)	115.2 (± 34.7)	109.5 (± 29.5)
LDL/HDL Ratio	2.4 (± 1.3)	2.0 (± 0.8)*
Glucose (mg/dL)	89.2 (± 6.8)	85.9 (± 7.4)*

Note. Sample sizes varied due to missing data for some assessments.
Values are reported as means (±SD).
HDL = high-density lipoprotein cholesterol; LDL = low-density lipoprotein.
*p < 0.05; **p < 0.01 indicates differences between males and females.

Metabolic Syndrome

Figure 1 shows the prevalence of each MetS risk factor using the NCEP-ATP III criteria for males and females.

Figure 1. Prevalence of Metabolic Syndrome (MetS) and its components



WC, waist circumference greater than 102 cm (males) and greater than 88 cm (females); BP indicates blood pressure of 130/85 mm Hg or higher; TRG, triglyceride level of 150 mg/dL or higher; HDL, fasting high-density lipoprotein cholesterol level less than 40 mg/dL (males) and less than 50 mg/dL (female); GLU, fasting blood sugar less than or equal to 100 mg/dL.

Figure 2 shows the prevalence of MetS criteria for males and females.

Figure 2. Prevalence of Metabolic Syndrome Criteria

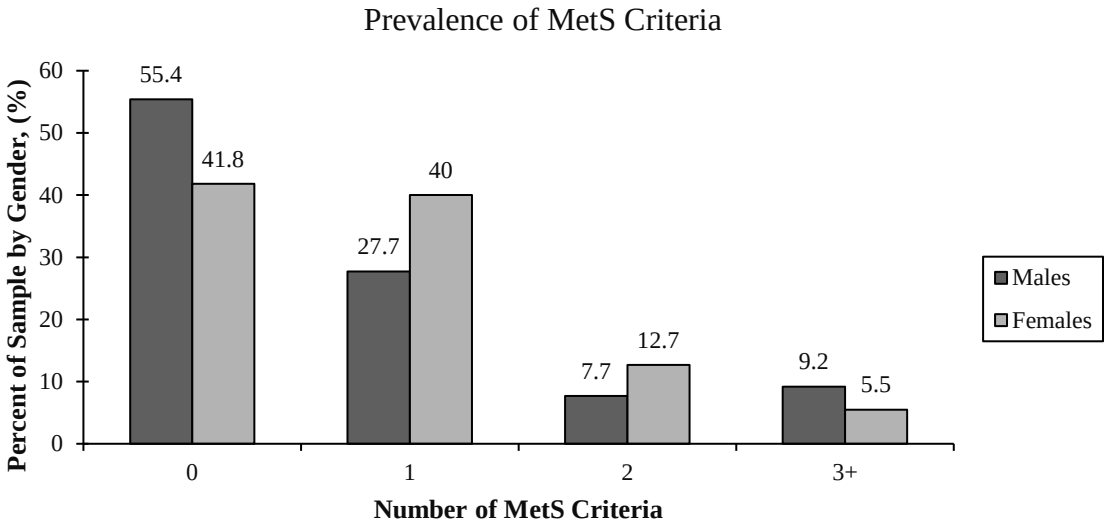


Table 4. Correlation Matrix for Females and Males

Variables	Waist	SBP	DBP	Triglycerides	HDL	Glucose
Waist	1	.583**	.304**	.491**	-.368**	.345**
SBP	.406**	1	.726**	.395**	-.163	.229*
DBP	.291*	.782**	1	.380**	.046	.191*
Triglycerides	.630**	.332**	.320**	1	-.214*	.271**
HDL	-.429**	-.060	-.085	-.386**	1	-.195*
Glucose	.088	.128	.225	.152	-.054	1

Note: Correlations for females are above and for males are below the diagonal. SPB = systolic blood pressure; DBP = diastolic blood pressure; HDL = high-density lipoprotein cholesterol.
*p < 0.05; **p < 0.01

Hemodynamics

Table 5. Hemodynamics

Variables	Male (n = 65)	Female (n = 109)
SBP (mm Hg)	122.5 (± 9.8)	115 (± 10.1)**
DBP (mm Hg)	65.5 (± 6.7)	65.7 (± 6.8)
MAP (mm Hg)	85.5 (± 7.8)	82.2 (± 7.1)*
Pules Pressure (mm Hg)	57.0 (± 6.1)	49.4 (± 6.9)**
Pulse Rate (beats/min)	57.9 (± 9.4)	64.1 (± 9.5)**
Cardiac Ejection Time (msec)	310.2 (± 22.8)	321.1 (± 22.6)*
Stroke Volume (ml/beat)	102.5 (± 11.7)	90.5 (± 13.4)**
Stroke Volume Index (ml/beat/m^2)	51.8 (± 5.7)	53.5 (± 6.5)
Cardiac Output (L/min)	6.0 (± 0.8)	5.8 (± 0.7)
Cardiac Index (L/min/m^2)	3.0 (± 0.3)	3.4 (± 0.2)**
Systemic Vascular Resistance (dyne•sec•cm-^{5})	1145.7 (± 148.7)	1163.2 (± 139.5)
Total Vascular Impedance (dyne•sec•cm^{-5})	121.5 (± 20.6)	116.6 (± 17.9)

Note. Sample sizes varied due to missing data for some assessments.
Values are reported as means (±SD).
SPB = systolic blood pressure; DBP = diastolic blood pressure; MAP = mean arterial blood pressure
*$p < 0.05$; **$p < 0.01$ indicates differences between males and females.

Table 6. Correlation Between MetS risk Factors and Hemodynamics for Males

Variables	SBP	DBP	WC	TRG	HDL	GLU
SBP (mm Hg)	1	.782**	.406**	.332**	-0.060	0.128
DBP (mm Hg)	.782**	1	.291*	.320**	-0.085	0.225
MAP (mm Hg)	.873**	.846**	.418**	.396**	-0.061	0.222
Pules Pressure (mm Hg)	.732**	0.148	.327**	0.177	-0.003	-0.044
Pulse Rate (beats/min)	.353**	.467**	.271*	.248*	-0.079	0.155
Cardiac Ejection Time (msec)	-0.146	-.334**	-.251*	-.269*	0.167	-.297*
Stroke Volume (ml/beat)	-0.010	-.266*	0.210	-0.036	-0.059	-0.094
Stroke Volume Index (ml/beat/m^2)	-.341**	-.525**	-.460**	-.386**	0.191	-.296*
Cardiac Output (L/min)	.436**	.419**	.573**	.400**	-0.176	0.136
Cardiac Index (L/min/m^2)	0.176	.252*	-0.044	0.110	0.068	-0.033
Systemic Vascular Resistance (dyne•sec•cm^{-5})	0.111	0.162	-.398**	-0.200	0.144	-0.056
Total Vascular Impedance (dyne•sec•cm^{-5})	0.094	-.275*	-.253*	-0.095	0.210	-0.139

Note: Correlations for females are above and for males are below the diagonal. SPB = systolic blood pressure; DBP = diastolic blood pressure; HDL = high-density lipoprotein cholesterol.
*p < 0.05; **p < 0.01

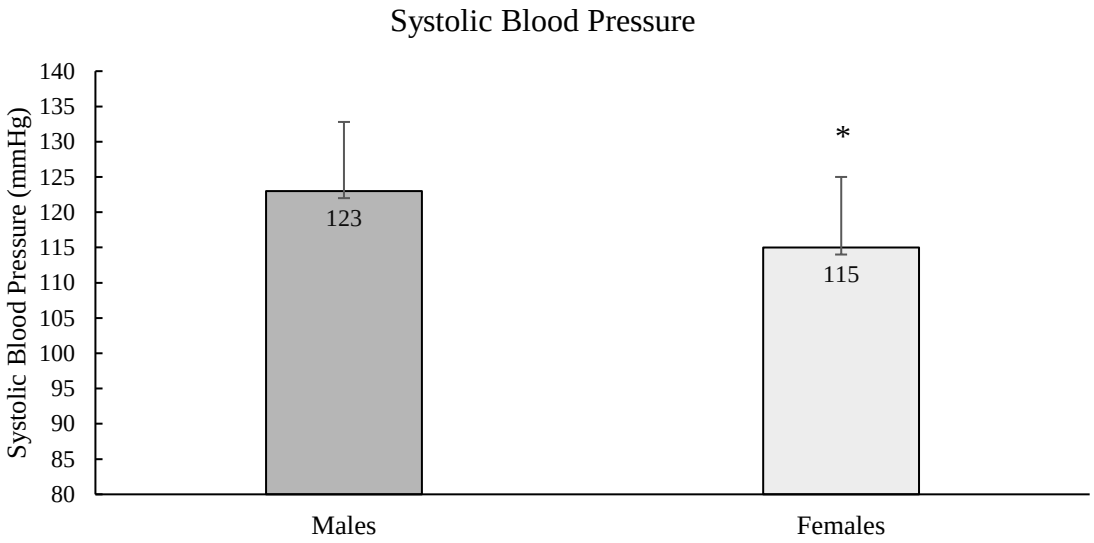
Table 7. Correlation Between MetS risk Factors and Hemodynamics for Females

Variables	SBP	DBP	WC	TRG	HDL	GLU
SBP (mm Hg)	1	.726**	.583**	.395**	-0.163	.229*
DBP (mm Hg)	.726**	1	.304**	.380**	0.046	.191*
MAP (mm Hg)	.905**	.834**	.481**	.368**	-0.062	0.160
Pules Pressure (mm Hg)	.756**	0.104	.612**	.291**	-.291**	0.181
Pulse Rate (beats/min)	.327**	.375**	0.149	.197*	-0.011	0.100
Cardiac Ejection Time (msec)	-.244*	-.265**	0.035	-0.097	0.003	-0.016
Stroke Volume (ml/beat)	0.052	-.227*	.479**	0.084	-.204*	0.055
Stroke Volume Index (ml/beat/m^2)	-.414**	-.434**	-.257**	-.236*	0.078	-0.160
Cardiac Output (L/min)	.443**	.189*	.800**	.393**	-.279**	.229*
Cardiac Index (L/min/m^2)	-0.158	0.018	-0.185	0.019	0.133	-0.034
Systemic Vascular Resistance (dyne•sec•cm-^{5})	.253**	.560**	-.438**	-0.066	.245*	0.134
Total Vascular Impedance (dyne•sec•cm^{-5})	.334**	-0.009	-0.040	0.030	-0.062	-0.065

Note: Correlations for females are above and for males are below the diagonal. SPB = systolic blood pressure; DBP = diastolic blood pressure; HDL = high-density lipoprotein cholesterol.
*p < 0.05; **p < 0.01

Figure 3 shows mean SBP for males and females. One-way ANOVA detected a difference between males and females ($p < 0.01$).

Figure 3. Systolic Blood Pressure

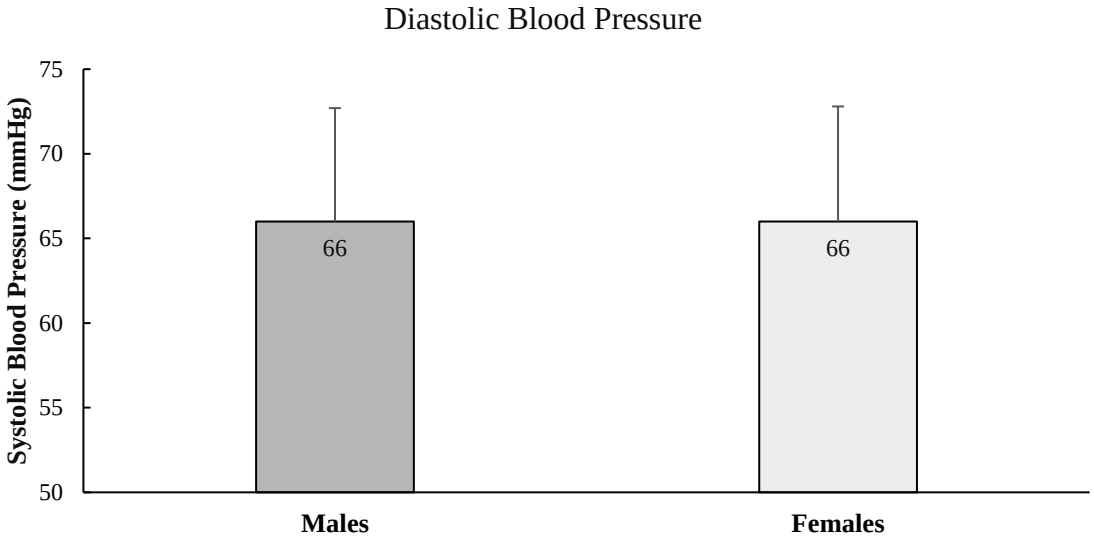


**p < 0.01* indicates differences between males and females.

For both males and females there was a positive, but weak significant correlation between TRG levels and SBP (r = .332, and r = .395, p<.01); a positive, but moderate significant correlation between WC and SBP (r = .406 and r =.583, p<.01, respectively); and a positive and strong significant correlation between SBP and DBP (r = .782 and r = .726, p<.01; respectively).

Figure 4 shows mean DBP for males and females. There was no difference between males and females.

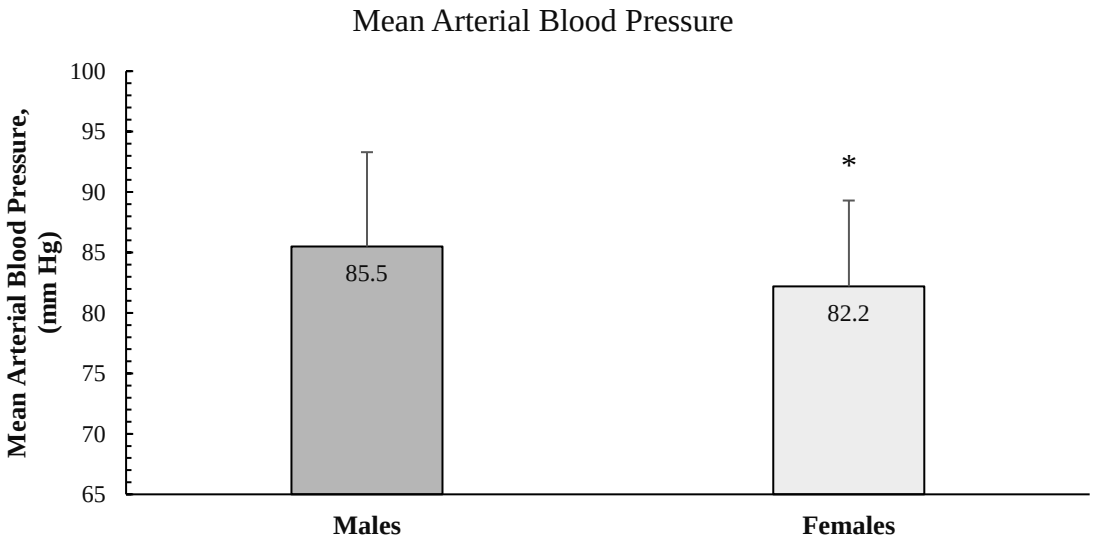
Figure 4. Diastolic Blood Pressure



For both males and females there was a positive, but weak significant correlation between WC and DBP ($r = .291$, $p<.01$, and $r =.304$, $p<.05$, respectively); and a positive, but weak significant correlation between TRG and DBP ($r =.320$ and $r =.380$, $p<.01$). For males, there was a positive, but weak significant correlation between age and DBP ($r = .319$, $p<.01$). For females there was a positive, but weak significant correlation between DBP and GLU levels ($r = .191$, $p<.05$).

Figure 5 shows the mean arterial blood pressure (MAP) for males and females. One-way ANOVA detected a difference between males and females ($p < 0.05$).

Figure 5. Mean Arterial Blood Pressure

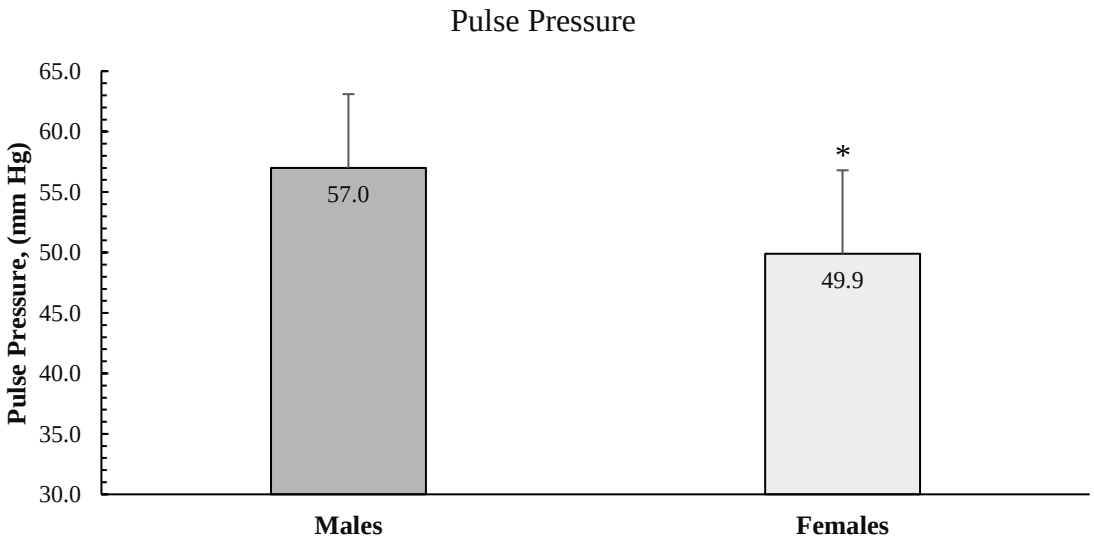


**p < 0.05* indicates differences between males and females.

For both males and females, there was a positive, but moderate significant correlation between WC and MAP (r =.418, and r =.481, p<.01, respectively); and a positive, but weak significant correlation between TRG levels and MAP (r = .396 and r =.368, p<.01, respectively). For males, there was a positive, but weak significant correlation between age and MAP (r = .257, p<.01).

Figure 6 shows the mean pulse pressure (PP) for males and females. One-way ANOVA detected a difference between males and females ($p < 0.01$).

Figure 6. Pulse Pressure

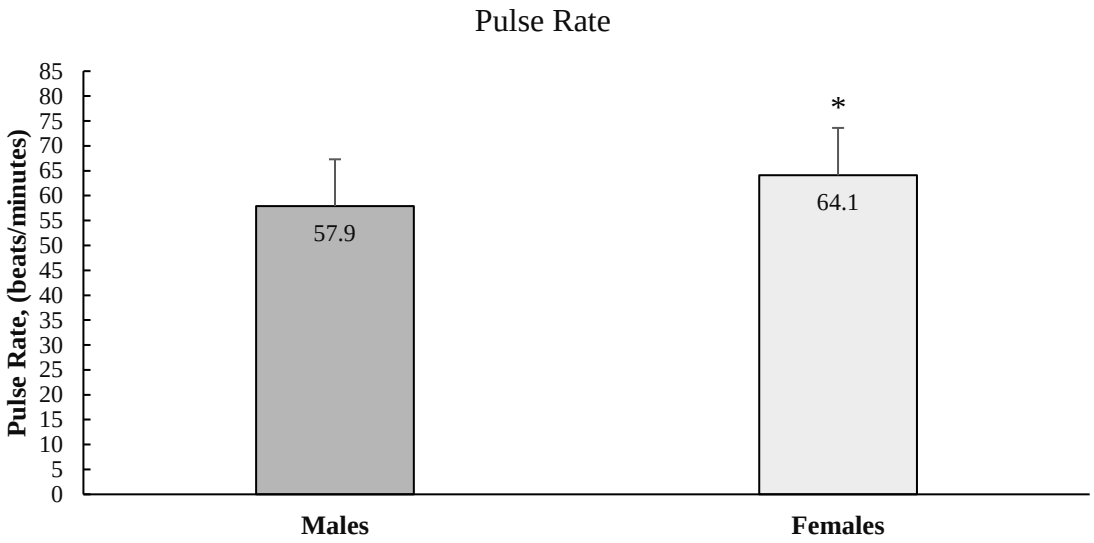


**p < 0.01* indicates differences between males and females.

For males, there was a positive, but weak significant correlation between WC and PP (r = .327, p<.01). For females there was a positive and strong significant correlation between WC and PP (r = .612, p<.01); a positive, but weak significant correlation between TRG levels and PP (r = .291, p<.01); and a negative, but weak significant correlation between HDL levels and PP (r = -.291, p<.01).

Figure 7 shows the mean pulse rate (PR) for males and females. One-way ANOVA detected a difference between males and females ($p < 0.01$).

Figure 7. Pulse Rate

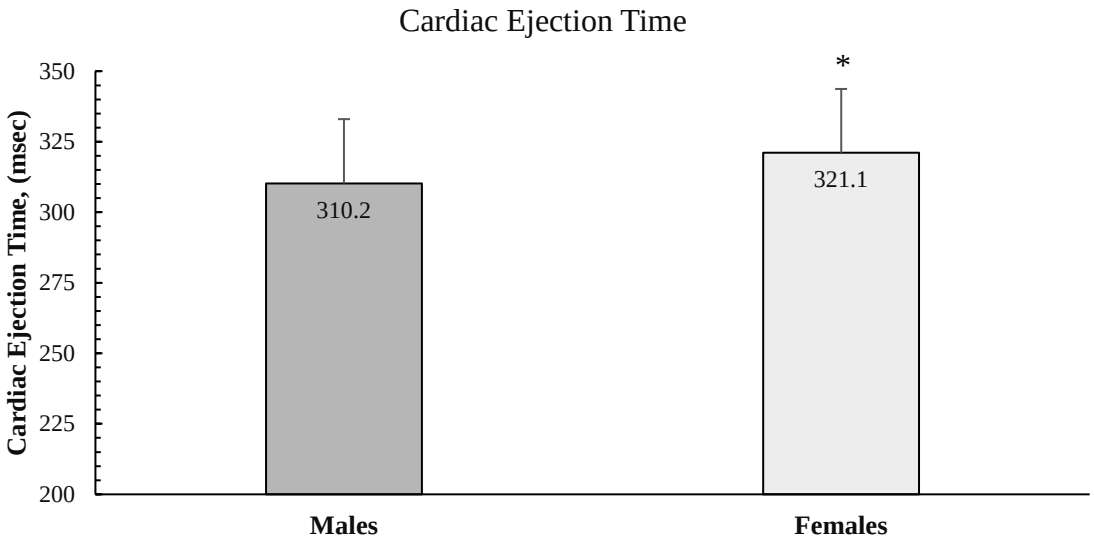


**p < 0.01* indicates differences between males and females.

For both males and females there was a positive, but weak significant correlation between TRG levels and PR (r = .248, and r = .197, p<.05, respectively); and a positive, but weak significant correlation between SBP and PR (r = .353 and r = .327, p<.01; respectively). For males, there was a positive, but weak significant correlation between WC and PR (r = .271, p<.05); and a positive, but moderate significant correlation between DBP and PR (r = .467, p<.01). For females there was a positive, but weak significant correlation between DBP and PR (r = .375, p<.01).

Figure 8 shows the mean cardiac ejection time (CET) for males and females. One-way ANOVA detected a difference between males and females ($p < 0.05$).

Figure 8. Cardiac Ejection Time

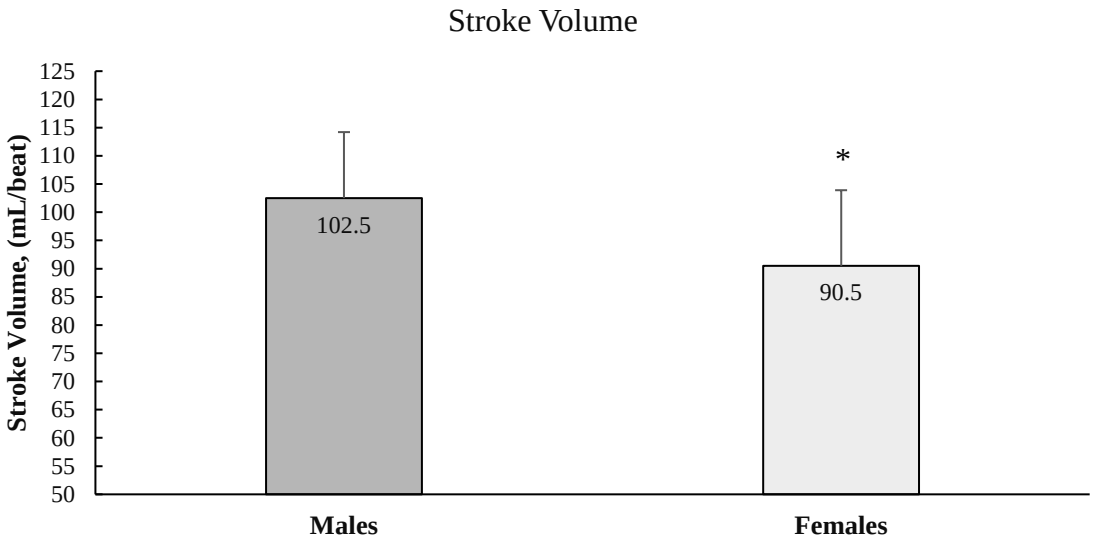


**p < 0.05* indicates differences between males and females.

For males, there was negative, but weak significant correlation between WC and CET (r = -.251, p<.05); a negative, but weak significant correlation between TRG levels and CET (r = -.269, p<.05); and a negative, but weak significant correlation between GLU levels and CET (r = -.297, p<.05). For females there was a negative, but weak significant correlation between SBP and CET (r = -.244, p<.05); and between age and CET (r = .272, p<.01). For both males and females there was a negative, but weak significant correlation between DBP and CET (r = -.334 and r = -.265, p<.01; respectively).

Figure 9 shows the mean stroke volume (SV) for males and females. One-way ANOVA detected a difference between males and females ($p < 0.01$).

Figure 9. Stoke Volume

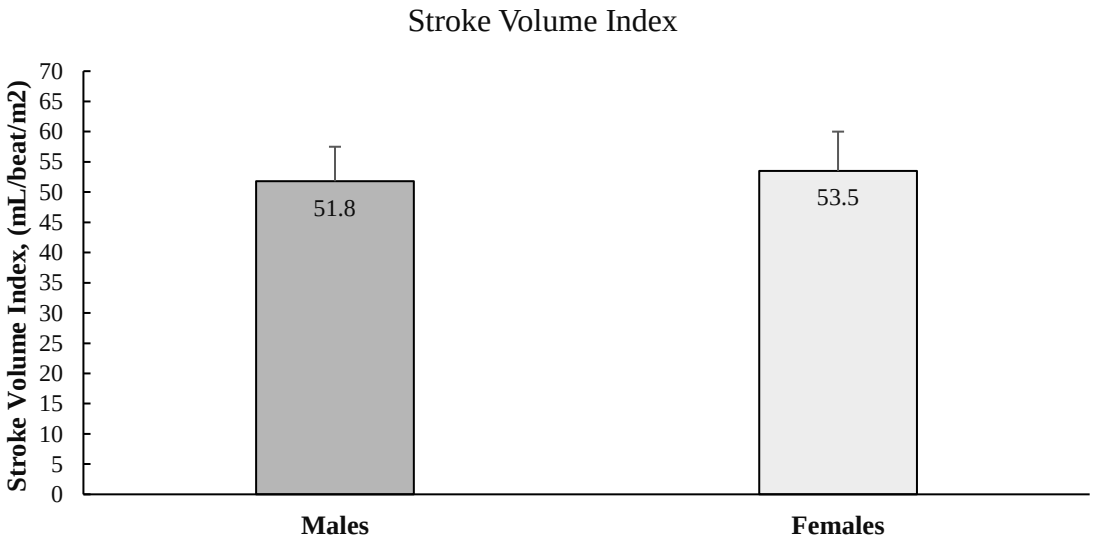


**p < 0.01* indicates differences between males and females.

For females there was, a positive, but moderate significant correlation between WC and SV (r =.479, p<.01); and a negative, but weak significant correlation between HDL levels and SV (r = -.204, p<.05). For both males and females there was a negative, but weak significant correlation between DBP and SV (r = -.266, and r = -.227, p<.01, respectively).

Figure 10 shows the mean stroke volume index (SVI) for males and females. There was no difference between males and females.

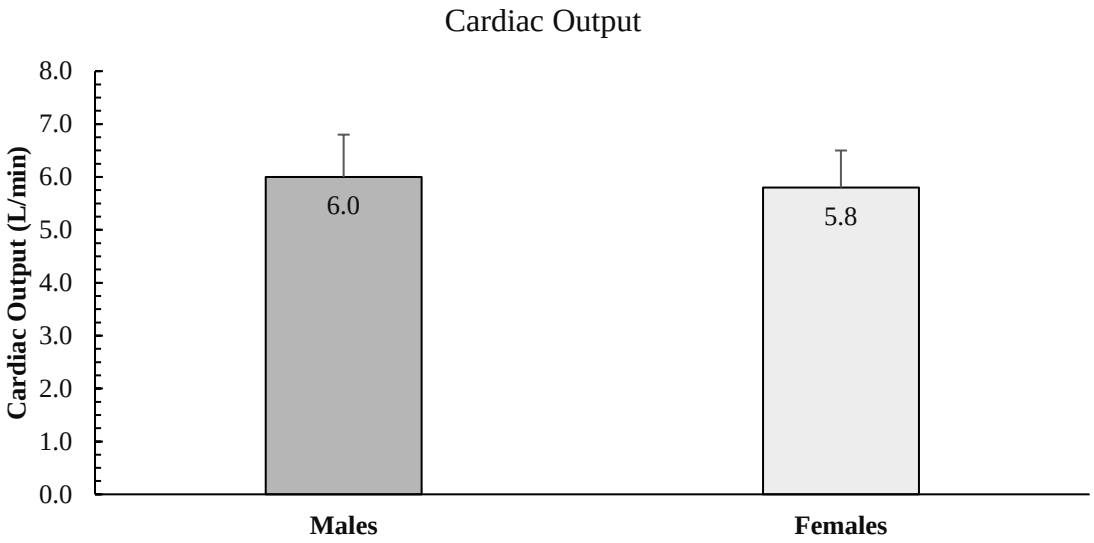
Figure 10. Stroke Volume Index



For males there was a negative, but weak significant correlation between TRG levels and SVI ($r = -.386$, $p<.01$); a negative, but weak significant correlation between SBP and SVI ($r = -.341$, $p<.01$); a negative, but weak significant correlation between GLU levels and SVI ($r = -.296$, $p<.05$). For females there was a negative, but weak significant correlation between WC and SVI ($r = -.257$, $p<.01$); a negative, but weak significant correlation between TRG levels and SVI ($r = -.236$, $p<.05$); and a negative, but moderate significant correlation between SBP and SVI ($r = -.414$, $p<.01$). For both males and females there was a negative, but moderate significant correlation between DBP and SVI ($r = -.525$ and $r = -.434$, $p<.01$; respectively).

Figure 11 shows the mean cardiac output (output) for males and females. There was no difference between males and females.

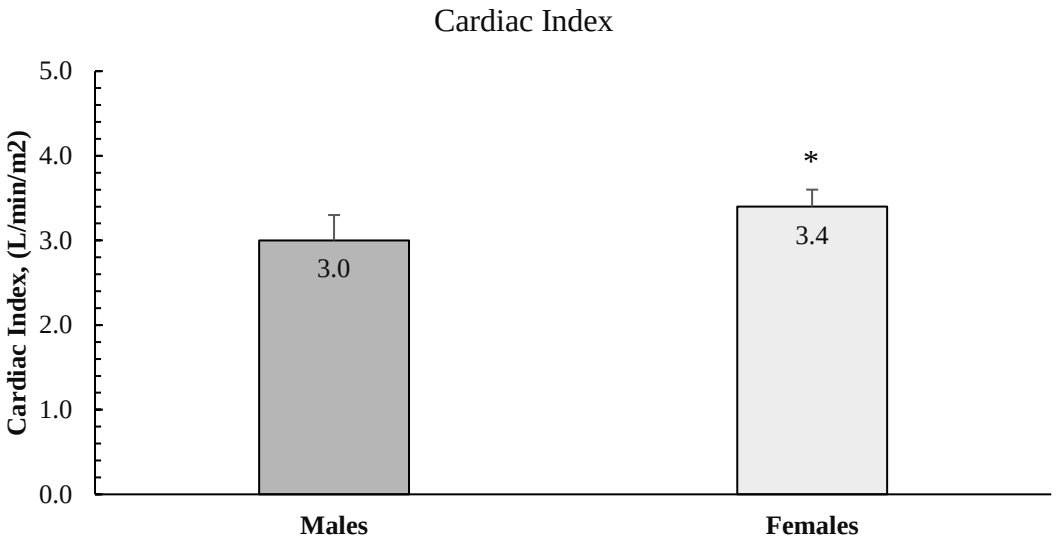
Figure 11. Cardiac Output



For males, there was a positive, but moderate significant correlation between WC and CO ($r = .573$, $p<.01$); a positive, but weak significant correlation between TRG levels and CO ($r = .400$, $p<.01$); a positive, but moderate significant correlation between SBP and CO ($r = .436$, $p<.01$); and positive, but moderate significant correlation between DBP and CO ($r = .419$, $p<.01$). For females there was, a positive and strong significant correlation between WC and CO ($r = .800$, $p<.01$); a positive, but weak significant correlation between TRG and CO ($r =.393$, $p<.01$); positive, but moderate significant correlation between SBP and CO ($r =.443$, $p<.01$); a positive, but weak significant correlation between DBP and CO ($r =.189$, $p<.05$); a negative, but weak significant correlation between HDL levels and CO ($r = -.279$, $p<.01$); and positive, but weak significant correlation between GLU levels and CO ($r = .229$, $p<.05$).

Figure 12 shows the mean cardiac index (CI) for males and females. One-way ANOVA detected a difference between males and females ($p < 0.01$).

Figure 12. Cardiac Index

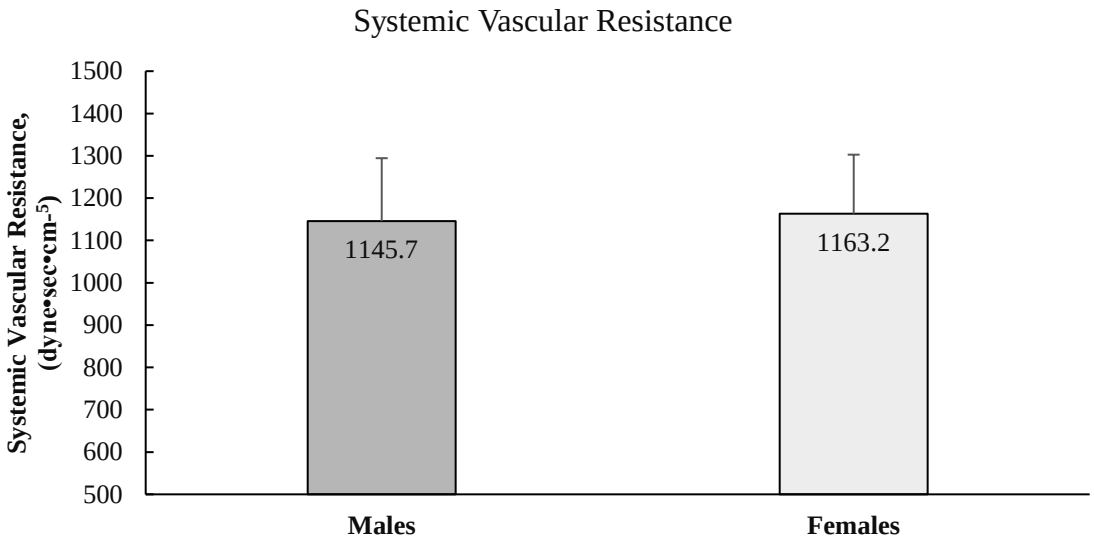


**p < 0.01* indicates differences between males and females.

For males there was a positive but weak significant correlation between DBP and CI (r = .252, p<.05); and a negative, but weak significant correlation between age and CI (r = -.287, p<.01).

Figure 13 shows the mean systemic vascular resistance (SVR) for males and females. There was no difference between males and females.

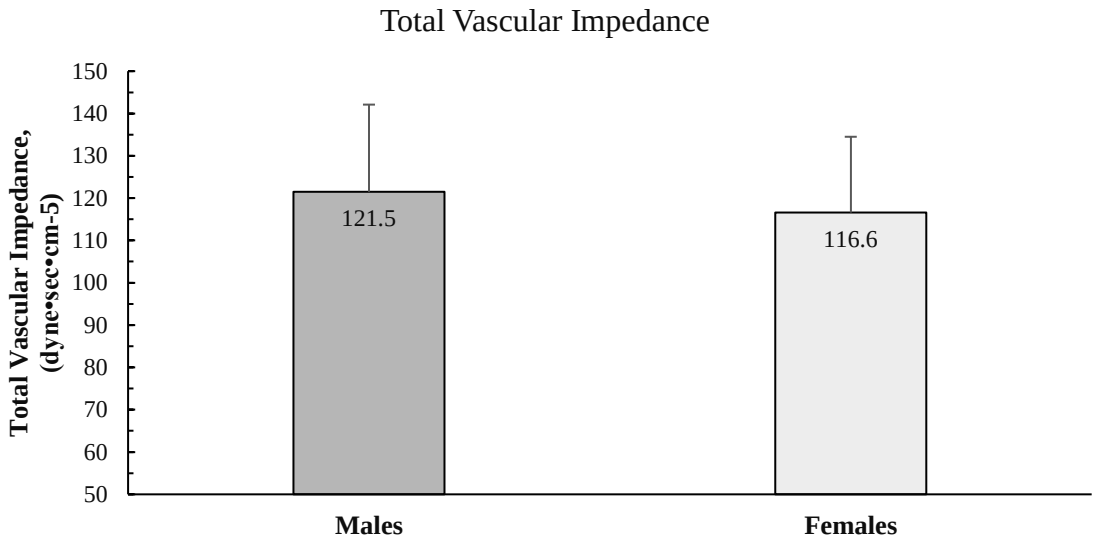
Figure 13. Systemic Vascular Resistance



For males there was a negative, but weak significant correlation between WC (SVR) ($r = -.398$, $p<.01$); and a positive, but weak significant correlation between age and SVR ($r = .344$, $p<.01$). For females there was a negative, but moderate significant correlation between WC and SVR ($r = -.438$, $p<.01$); a positive, but weak significant correlation between SBP and SVR ($r = .253$, $p<.01$); a positive, but moderate significant correlation between DBP and SVR ($r = .560$, $p<.01$); and a positive, but weak significant correlation between HDL levels and SVR ($r = .245$, $p<.05$).

Figure 15 shows the mean total vascular impedance (TVI) for males and females. There was no difference between males and females.

Figure 14. Total Vascular Impedance



For males, there was a negative, but weak significant correlation between WC and TVI ($r = -.253$, $p<.05$); and a negative, but weak significant correlation between DBP and TVI ($r = -.275$, $p<.05$). For females there was a positive, but weak significant correlation between SBP and TVI ($r = .334$, $p<.01$).

Arterial Elasticity

Table 8. Arterial Elasticity

Variables	Male (n = 64 - 65)	Female (n = 107 - 109)
LAE (ml/mm Hg x 10)	17.9 ± 3.1	16.8 ± 2.7*
SAE (ml/mm Hg x 100)	10.5 ± 1.9	9.4 ± 2.1*
AP (mm Hg)	-1.3 ± 3.8	0.8 ± 3.8**
AP@75 (mm Hg)	-4.2 ± 3.8	-1.0 ± 3.9**
AIx (%)	-4.5 ± 11.0	1.8 ± 11.7*
AIx@75 (%)	-13.0 ± 11.3	-3.8 ± 11.6**
AI (%)	96.0 ± 11.9	103.1 ± 13.8*

Note. Sample sizes varied due to missing data for some assessments.
Values are reported as means (±SD).
LAE = large artery elasticity; SAE = small artery elasticity; AP = central augmentation pressure; AP@75 = corrected central augmented pressure at heart rate 75 beats per minute; AIx = augmentation index (AP/PP); AIx@75 = corrected augmentation index at heart rate 75 beats per minute; AI = central augmentation index (P2/P1).
*p < 0.05; **p < 0.01 indicates differences between males and females.

Table 9. Correlation Between MetS Risk Factors and Arterial Elasticity for Males

Variables	SBP	DBP	WC	TRG	HDL	GLU
LAE (ml/mm Hg x 10)	-.477**	-0.238	-0.022	-0.225	-0.123	-0.02
SAE (ml/mm Hg x 100)	-0.177	-.257*	0.082	-0.152	-0.04	-.272*
AP (mm Hg)	0.234	.368**	0.014	0.071	0.021	-0.095
AP@75 (mm Hg)	.274*	.501**	0.044	0.127	-0.009	-0.066
AIx (%)	0.236	.354**	0.022	0.058	0.033	-0.101
AIx@75 (%)	.344**	.496**	0.073	0.129	0.017	-0.084
AI (%)	0.239	.368**	0.017	0.054	0.027	-0.115

LAE = large artery elasticity; SAE = small artery elasticity; AP = central augmentation pressure; AP@75 = corrected central augmented pressure at heart rate 75 beats per minute; AIx = augmentation index (AP/PP); AIx@75 = corrected augmentation index at heart rate 75 beats per minute; AI = central augmentation index (P2/P1).

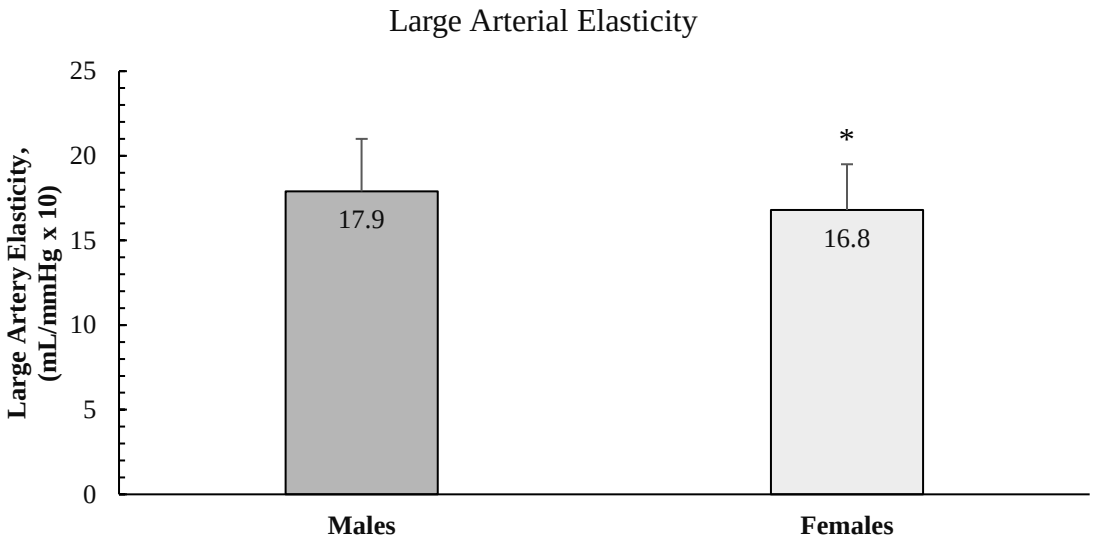
Table 10. Correlation Between MetS Risk Factors and Arterial Elasticity for Females

Variables	SBP	DBP	WC	TRG	HDL	GLU
LAE (ml/mm Hg x 10)	-.503**	-.300**	0.025	-0.154	0.020	-0.154
SAE (ml/mm Hg x 100)	-0.116	-0.183	0.077	-0.040	-0.01	0.134
AP (mm Hg)	0.142	.274**	0.115	.194*	-0.042	0.137
AP@75 (mm Hg)	.236*	.379**	0.122	.268**	-0.039	0.135
AIx (%)	0.106	.255**	0.091	0.172	-0.035	0.102
AIx@75 (%)	.216*	.382**	0.134	.275**	-0.051	0.149
AI (%)	0.111	.263**	0.094	0.183	-0.030	0.129

LAE = large artery elasticity; SAE = small artery elasticity; AP = central augmentation pressure; AP@75 = corrected central augmented pressure at heart rate 75 beats per minute; AIx = augmentation index (AP/PP); AIx@75 = corrected augmentation index at heart rate 75 beats per minute; AI = central augmentation index (P2/P1).

Figure 15 shows the mean large artery elasticity (LAE) One-way ANOVA detected a difference between males and females ($p < 0.05$).

Figure 15. Large Arterial Elasticity

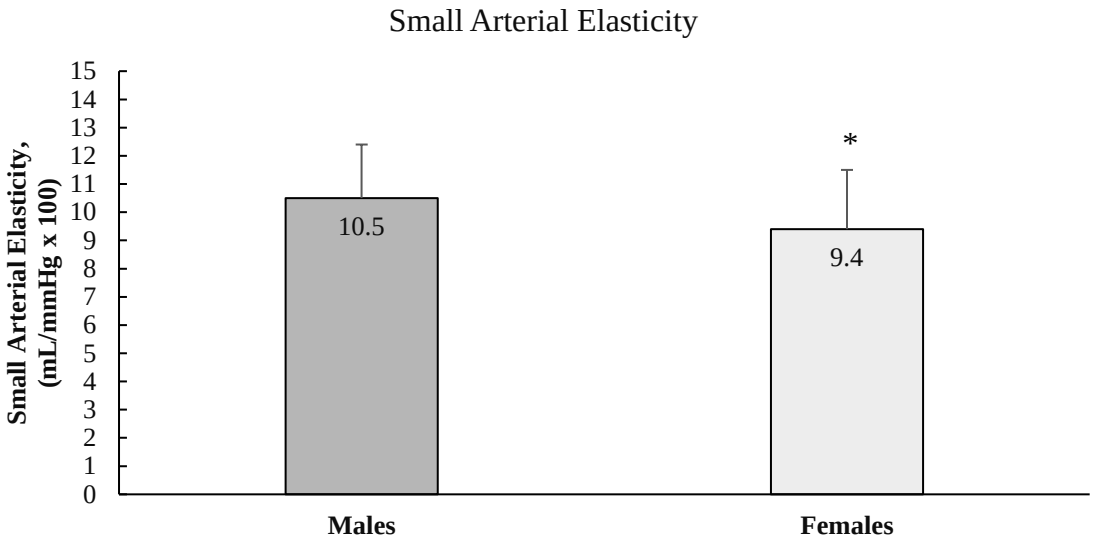


**p < 0.05* indicates differences between males and females.

For females there was a negative, but weak significant correlation between DBP and LAE (r = -.300, p<.01). For both males and females there was a negative, but moderate significant correlation between SPB and LAE (r = -.477 and r = -.503, p<.01; respectively).

Figure 16 shows the mean small artery elasticity (SAE). One-way ANOVA detected a difference between males and females (p < 0.05).

Figure 16. Small Arterial Elasticity

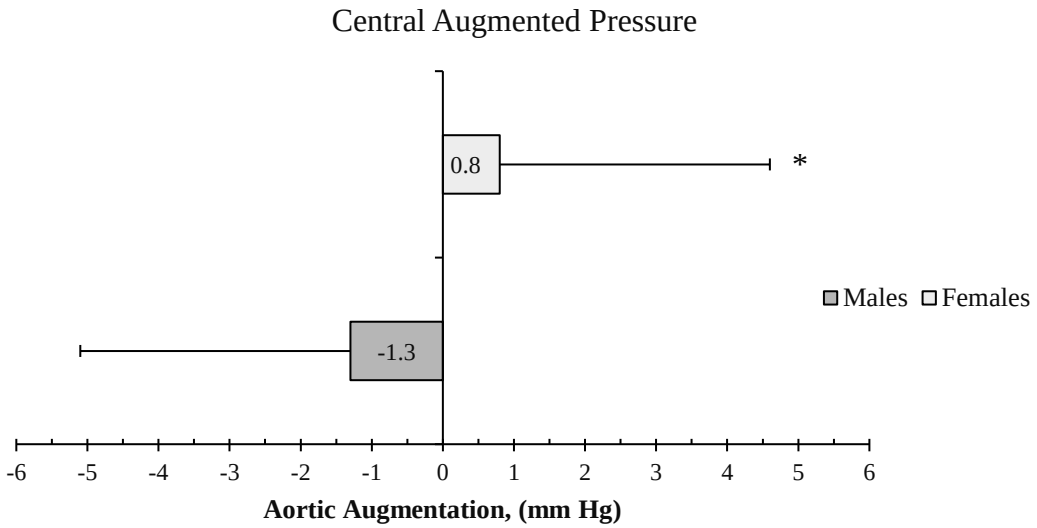


**p < 0.05* indicates differences between males and females.

For males there was a negative, but weak significant correlation between GLU levels and SAE ($r = -.272$, $p<.05$); and a negative, but weak significant correlation between DBP and SAE ($r = -.257$, $p<.05$).

Figure 17 shows the mean for central augmented pressure (AP) for males and females. One-way ANOVA detected a difference between males and females ($p < 0.01$).

Figure 17. Central Augmented Pressure

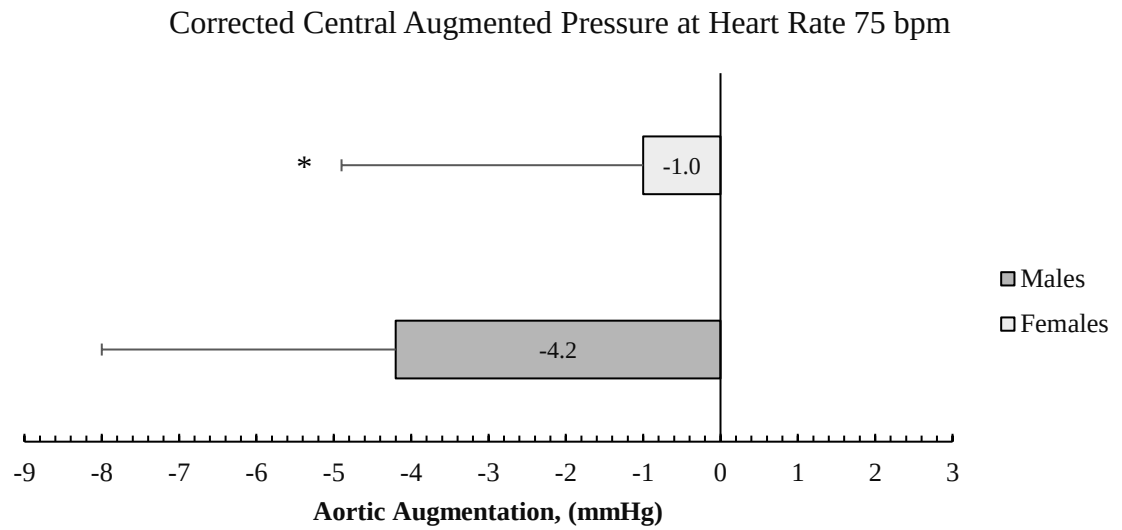


**p < 0.01* indicates differences between males and females.

For males, there was a positive, but moderate significant correlation between age and AP ($r = .434$, $p<.01$). For females, there was a positive, but weak significant correlation between TRG levels and AP ($r = .194$, $p<.05$); and between age and AP ($r = .368$, $p<.01$). For both males and females there is a positive, but weak significant correlation between DBP and AP ($r = .368$ and $r = .274$, $p<.01$; respectively).

Figure 18 shows mean corrected central augmented pressure at heart rate 75 beats per minute (bpm) (AP@75). One-way ANOVA detected a difference between males and females ($p < 0.01$).

Figure 18. Corrected Central Augmented Pressure at Heart Rate 75 bpm

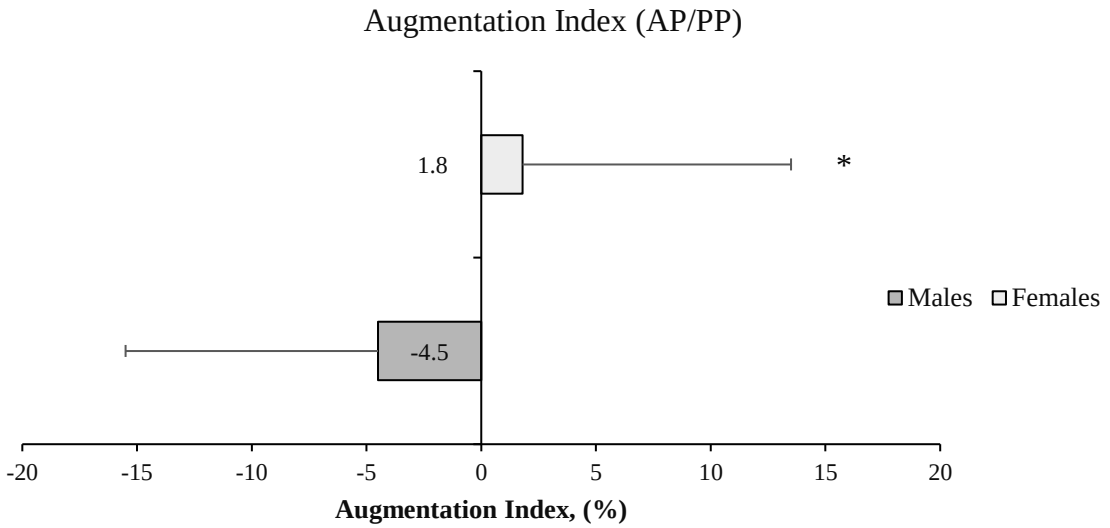


**p < 0.01* indicates differences between males and females.

For males there was a positive, but moderate significant correlation between DBP and AP@75 (r = .501, p<.01). For females there was a positive, but weak significant correlation between DBP and AP@75 (r = .379, p<.01); and a positive, but weak significant correlation between TRG levels and AP@75 (r = .268, p<.01). For both males and females there was a positive, but weak significant correlation between SPB and AP@75(r = .274 and r = .236, p<.05; respectively); and between age and AP@75 (r = .373 and r = .286, p<.01; respectively);

Figure 19 shows mean augmentation index (AIx) for males and females. One-way ANOVA detected a difference between males and females (p < 0.05).

Figure 19. Augmentation Index (AP/PP)

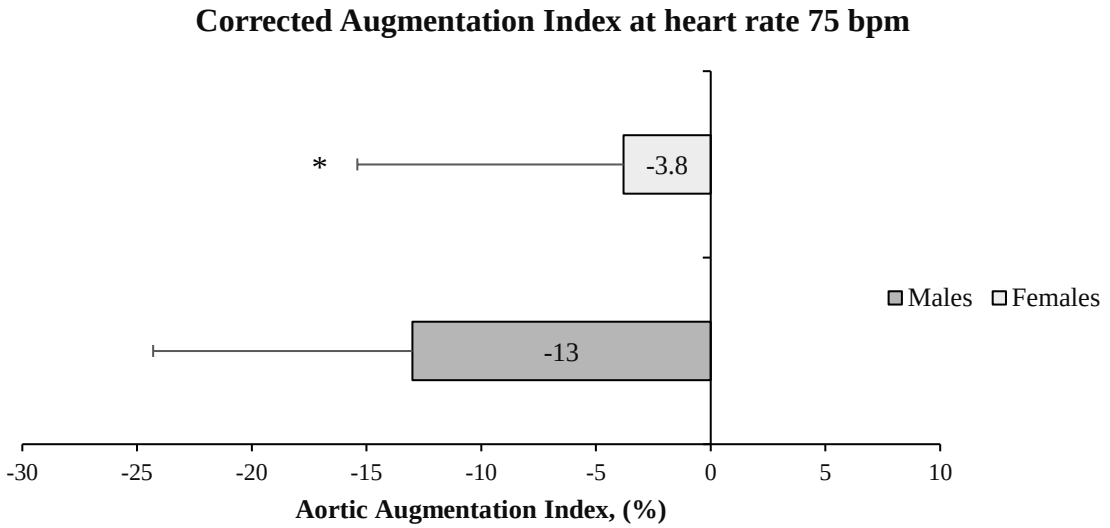


**p < 0.05* indicates differences between males and females.

For both males and females there is a positive, but weak significant correlation between DBP AIx (r =.354 and r = .255, p<.01; respectively). For males, there was a positive, but moderate significant correlation between age and AIx (r = .415, p<.01) For females there as a positive, but weak significant correlation between age and AIx (r = .378, p<.01).

Figure 20 shows mean corrected augmentation index at heart rate 75 beats per minute (AIx@75). One-way ANOVA detected a difference between males and females ($p < 0.01$).

Figure 20. Corrected Augmentation Index at Heart Rate 75 bpm

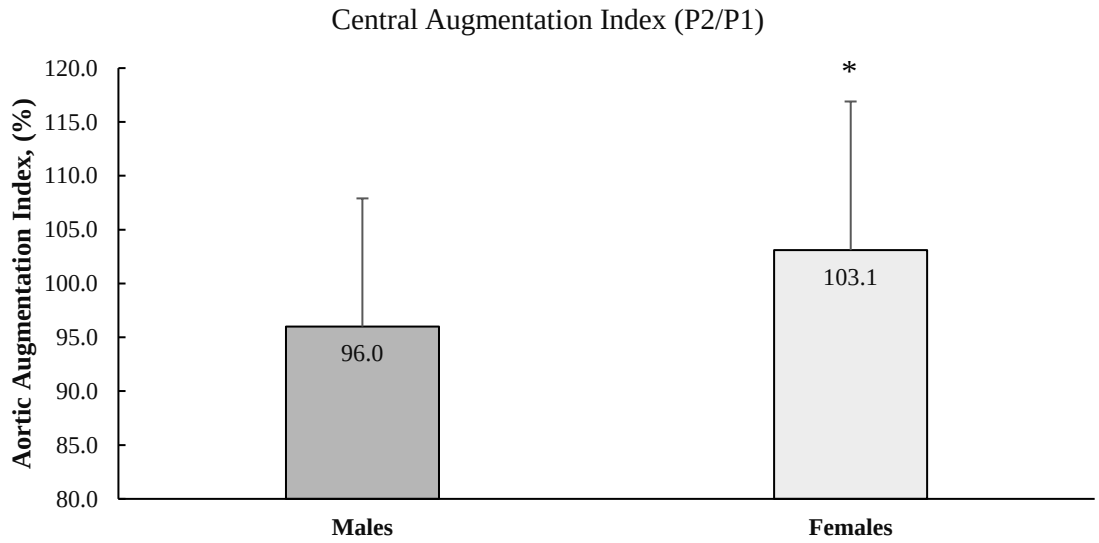


**p < 0.01* indicates differences between males and females.

For males there was a positive, but moderate significant correlation between DBP and AIx@75 (r = .496, p<.01). For females, there was a positive, but weak significant correlation between TRG levels and AIx@75 (r = .275, p<.01); and a positive, but weak significant correlation between DBP and AIx@75 (r = .382, p<.01). For both males and females there was a positive, but weak significant correlation between SPB and AIx@75 (r = .344, p<.01, and r = .216, p<.05; respectively); and between age and AIx@75 (r = .364, and r = .358, p<.01, respectively).

Figure 21 shows mean central augmentation index (AI) for males and females. One-way ANOVA detected a difference between males and females ($p < 0.05$).

Figure 21. Central Augmentation Index (P2/P1)



**p < 0.05* indicates differences between males and females.

For males there was a positive, but weak significant correlation between DBP and AI (r = .368, p<.01). For both males and females there was a positive, but moderate significant correlation between age and AI (r = .432, and r = .424, p<.01, respectively).

CHAPTER V

DISCUSSION

The purposes of this study were to 1) investigate the correlation of MetS risk factors on arterial elasticity in a college-aged Hispanic population; 2) investigate the correlation of MetS risk factors on hemodynamics (mean arterial blood pressure, pulse pressure, resting heart rate, cardiac ejection time, stroke volume, stoke volume index, cardiac output, cardiac index, large artery elasticity, small artery elasticity, systemic vascular resistance, and total vascular impedance); 3) assess the prevalence of MetS risk factors in the UTRGV Hispanic student population using the NCEP-ATP III definition; and 4) compare sex differences among arterial elasticity, hemodynamics, and MetS risk factors.

Arterial Elasticity

All the blood vessels of the circulatory system have a conduit function with the purpose to supply organs with blood and have a cushioning function that decreases oscillation, however large arteries have a more prominent function of dampening oscillation, whereas the small arteries are better suited for blood distribution (Blacher, Protogerou, & Safar, 2005).
There are no studies known to the authors that observe the correlation between LAE and SAE and MetS and its risk factors in college aged students. In the current study, SBP/DBP and GLU were found to be significantly correlated with LAE and SAE, while no significant correlations were found for WC, TRG, and HDL.

The findings of this study partially support the findings in a Chinese study by Ge and associates (2008) that investigated the relationship between components of MetS and LAEs and SAEs in individuals ages 33 to 65. LAE and SAE was associated with SBP and DBP, and LAE was significantly associated with GLU levels and SAE was associated with HDL levels. However, in the present study, LAE was negatively associated with SBP for both sexes, while DBP was negatively associated with LAE for females only. Additionally, DBP and GLU was found to be negatively associated with SAE for males only. Finding differences could be potentially explained due to variances in the age range, ethnicities and population sample size.

Unlike in the study by Gardner et at. (2013) which looked at MetS and arterial elasticity in two-hundred six youth, ages 10 to 20, that found elevated TRG levels to reduce arterial elasticity, the current study found no correlation between TRG levels and LAE or SAE. For the current study, LAE and SAE was higher (at 17.4 and 9.9, respectively) than that of the younger population tested by Gardner and colleagues (at 15.8 and 8.8, respectively). Differences in results may be explained due to averages no being separated by gender, demographic variables and subject population size.

To the knowledge of the author, the current study is the first to specifically investigate the correlation between MetS risk factors and central augmented pressure (AP), heart rate corrected at 75 bpm central augmented pressure (AP@75), augmentation index (AP/PP) (AIx), corrected augmentation index at 75 bmp (AIx@75) and augmentation index (P2/P1) (AI). Furthermore, SBP/DBP and TRG were the MetS components that were found to be significantly correlated with tested arterial elasticity components. For both males and females, SBP was positively correlated with AIx@75, and DBP was positively associated with all arterial elasticity

components: AP, AP@75, Alx (AP/PP), Alx@75, and AI. For females, TRG were positively correlated with AP, AP@75, and AIx@75. No associations were found for WC, HDL, and GLU.

In a previous European study by Wojciechowska et al. (2006), which included 534 Europeans without cardiovascular disease (57.3% females; mean age 34.9 years), proposed that for the European Caucasian population upper 95th diagnostic thresholds of middle-aged males be 90% for AI and 30% for AIx and that thresholds need adjustment for sex and age. In the study by Shiburi and colleagues (2006) proposed the thresholds of AI at 100% and AIx at 40% for individuals of African descent. These same values were also proposed in the study by Chung and colleagues (2010), which included 522 participants, for individuals of Korean descent. Different values were given in the study by Li and colleagues (2008), which included 924 individuals from an ongoing Chinese study on genes in hypertension from 2003 through 2005, which proposed that the thresholds be 105% and 45% for AI and AIx, respectively. There are no studies that suggest preliminary thresholds to diagnose decreased arterial elasticity in Hispanics. In the current study the average AI and AIx for males was 96% and -4.5%, respectively; and for females it was 103% and 1.8%, respectively; with AIx falling well below all previously mentioned thresholds. This enormous discrepancy in AIx emphasizes the need for validated reference values for the Hispanic population.

Hemodynamics

To the knowledge of the author, the current study is the first to specifically investigate the correlation between MetS risk factors and the hemodynamics: systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP), pulse pressure (PP), pulse rate (PR), cardiac ejection time (CET), stroke volume (SV), stroke volume index (SVI), cardiac output (CO), cardiac index (CI), systemic vascular resistance (SVR), and total vascular

resistance (TVI) in college students. A significant association was found with SBP and two other MetS risk factors: TRG, WC. DBP was associated with TRG levels, GLU levels (only for females), and WC. MAP was significantly associated with two risk factors: WC and TRG levels. PP was significantly associated with three risk factors: WC, HDL levels (only for females) and TRG levels (only for females). PR was significantly associated with three risk factors: TRG levels, blood pressure (SBP and DBP), and WC (only for males). CET was significantly associated with four risk factors: WC (only for males), TRG levels (only for males), GLU levels (only for males), and BP (only for females). SV was significantly associated with three risk factors: HDL levels (only for females), WC (only for females) and DBP. SVI was associated with four risk factors: TRG levels, BP, GLU levels (only for males) and WC (only for females). CO was associated with all five risk factors in females; with males having three significant associations: WC, TRG levels, and BP. CI only had one significant association with DPB, but only for males. SVR has significant associations with three risk factors: WC, BP (for females only), and HDL (for females only). Lastly, TVI had two risk factor associations: WC and DBP (for males only) and SBP (for females only).

The findings of this study present unique results for WC and SVR. These finding may be explained due to increased adiponectin levels present in Hispanics. In a cross-sectional study conducted by Hanley and colleagues (2007), which looked at the associations of adiponectin with body fat distribution and insulin sensitivity in 1,636, non-diabetic Hispanics and African Americans, it was found that Hispanics had greater visceral adipose tissue and insulin sensitivity, with higher levels of adiponectin and triglyceride. However, when compared to African-Americans the inverse association with visceral adipose tissue and adiponectin levels were not as strongly present in Hispanics. Furthermore, Mirza and colleagues (2012) aimed to study the

presence of pro- and anti-inflammatory cytokines and adipokines in 367 Mexican-American in cross-sectional study conducted. It was unexpectedly found that for individuals with type II diabetes, there were higher levels of adiponectin with lower levels of leptin. Moreover, it may be suggested that for the Hispanic population tested, there is an unknown mechanism that may cause higher levels of adiponectin secretion while suppressing leptin synthesis, which may explain the inverse relationship found with WC and SVR.

For both males and females SBP was found to be associated with PR, SVI, and CO. DBP was found to be associated with MAP, PR, CET, SV, SVI, and CO. WC was found to be associated with SBP/DBP, MAP, PP, SVI, CO, and SVR. TRG was found to be associated with SBP/DBP, MAP, PR, SVI, and CO. For females HDL was associated with PP, SV, CO, and SVR; however, for males no hemodynamic associations were present. For females, GLU was associated with SBP/DBP, and CO, while for males, GLU was associated with CET and SV. These varying finding for sexes maybe explained due to the small sample size of males. Further studies are needed to compare hemodynamic correlations and MetS risk factors.

Metabolic Syndrome

In this study, 6.9% of the sample could currently be diagnosed with MetS according to the NCEP ATP III guidelines. These findings are nearly identical to the 6.8% incidence of MetS reported by Dalleck and Kjalland (2012), in a study which looked at the prevalence of MetS in 207 college students, ages 18 to 24 years in Wisconsin. Moreover, when looking at the prevalence of each individual risk factor, low-HDL was the most common at 44%. These findings are consistent with those of earlier studies. Low HDL levels were the most prevalent risk factor for the studies Dalleck and Kjallend (2012) at 47%, Manjunath et al. (2014) at 38.9%, Topè and Rogers (2013) at 37.3%, Fernandes et al. (2011) (20.1%), Paul Das et al. (2017)

(28.7%) and Morrel et al. (2013) (27.8%). Low-HDL levels are common in college students, however another explanation for such a high prevalence of low HDL in this study is that 64% of the tested population were females and 50.4 % had low HDL levels. In the study by Ervin (2009), Hispanic females were found to have lower HDL levels when compared to non-Hispanics whites and non-Hispanics blacks.

Furthermore, in the current study 35.4% of the sample had at least one MetS risk factor and 10.9% had at least two risk factors. These findings were higher than those reported by Huang and colleagues (2007) (25.2% and 1.2%, respectively), Fernandes and Lofgren (2011) (28% and 7.4%, respectively), and Topè and Rogers (2013) (31.4% and 20.7%, respectively), but lower than those reported by Nieto et al. (2015) (50% and 16.1%, respectively), and Dalleck and Kjallend (2012) (42.5% and 13%, respectively). This suggests that there are variances on prevalence that could be explained due to ethnic and geographic differences.

The findings of this study also mirror the findings in previous studies that males predominantly had a higher prevalence for elevated triglyceride levels, hypertension, and impaired fasting blood glucose, while for females, increased abdominal obesity, and low-HDL levels were predominant (Morrell, et al, 2013; Nieto et al., 2015), but are different than those in the study by Morrell et al. (2013), in which males had a higher prevalence of increased triglyceride levels, hypertension, impaired fasting blood glucose and lower levels of HDL and also differ from what was reported Paul Das et al. (2017), in which females had a higher prevalence of low HDL levels, increased abdominal obesity and impaired fasting glucose. These differences in prevalence could be explained due to ethnic, geographic and sample size differences.

Conclusions

The purposes of this study were to 1) investigate the correlation of MetS risk factors on arterial elasticity in a college-aged Hispanic population; 2) investigate the correlation of MetS risk factors on hemodynamics (mean arterial blood pressure, pulse pressure, resting heart rate, cardiac ejection time, stroke volume, stoke volume index, cardiac output, cardiac index, large artery elasticity, small artery elasticity, systemic vascular resistance, and total vascular impedance); 3) assess the prevalence of MetS risk factors in the UTRGV Hispanic student population using the NCEP-ATP III definition; and 4) compare sex differences among arterial elasticity, hemodynamics, and MetS risk factors.

The research questions asked were:

1.) Is there a correlation between MetS risk factors and arterial elasticity in the Hispanic population?
2.) Is there a correlation between MetS risk factors and hemodynamics in the Hispanic population?
3.) What correlative differences are there for MetS risk factors, arterial elasticity and hemodynamics between males and females?

Research Hypothesis 1. There would be a negative correlation between arterial elasticity and abdominal obesity, triglyceride level, systolic blood pressure, diastolic blood pressure, and fasting glucose. There will also be a positive correlation between high density lipoprotein and arterial elasticity.

The results of the present study only partially supported this hypothesis. It was hypothesized that all MetS risk factors would negatively impact arterial elasticity, however not all factors had a significant correlation with arterial elasticity.

Research Hypothesis 2. The presence of MetS risk factors would negatively impact hemodynamics.

The results of the present study only partially supported this hypothesis. It was hypothesized that all MetS risk factors would be detrimental to hemodynamics, however not all factors had a significant correlation with tested hemodynamic components.

Research Hypothesis 3. There would be correlative differences in MetS risk factors, arterial elasticity and hemodynamics between males and females.

The results of the present study support this hypothesis. Correlations for arterial elasticity and hemodynamic and MetS risk factors were sometimes present for males, but not females and vice versa.

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APPENDIX A

APPENDIX A

DEFINITIONS

1) **Arterial elasticity:** The measurement of the elastic properties of the arteries, which has an inverse relationship with arterial stiffness.
2) **Hydration:** Hydration status was deemed adequate when urine specific gravity measured 1.010 and lower as determined by a clinical urine refractometer.
3) **MetS risk factors:** Waist circumference (> 102 cm for males or > 88 cm for females), blood pressure ≥ 130 mm Hg systolic or ≥ 85 mm Hg diastolic or taking blood pressure-lowering medications, triglyceride level ≥150 mg/dL or taking lipid-lowering medications, fasting high-density lipoprotein cholesterol level < 40 mg/dL for males and < 50 mg/dL for females or taking lipid management medications and fasting blood sugar ≥ 100 mg/dL or drug treatment for diabetes mellitus.
4) **Body Composition:** Body fat percentage.
5) **Tanita:** Dual-frequency body composition analyzer.
6) **Hypertension diagnostic:** A noninvasive equipment that conducts measurements of arterial stiffness via placing a sensor on the radial artery.
7) **Refractometer:** A device used for analyzing urine specific gravity to measure hydration by measuring the concentration of a urine sample.
8) **Hemodynamics:** Analysis of physical aspects of blood circulation and blood flow.

9) **Pulse Wave Analysis (PWA):** A technique that allows the accurate recording of peripheral pressure waveforms and generation of the corresponding central waveform, from which the augmentation index and central pressure can be derived.

LIST OF ABBREVIATIONS

AI -Central Augmentation Index (P2/P1)

AIx -Augmentation Index (AP/PP)

AIx@75 -Corrected Augmentation Index at Heart Rate 75 Beats Per Minute

ANOVA -Analysis of Variance

AP -Central Augmentation Pressure

AP@75 -Corrected Central Augmented Pressure at Heart Rate 75 Beats Per Minute

CET -Cardiac Ejection Time

CI -Cardiac Index

CO -Cardiac Output

DBP -Diastolic Blood Pressure

GLU - Glucose

HDL -High-Density Lipoprotein

LAE -Large Arterial Elasticity

LDL -Low-Density Lipoprotein

MAP -Mean Arterial Pressure

MetS -Metabolic Syndrome

PP -Pulse Pressure

PR -Pulse Rate

PWA -Pulse Wave Analysis

SAE -Small Arterial Elasticity

SBP -Systolic Blood Pressure

SV -Stroke Volume

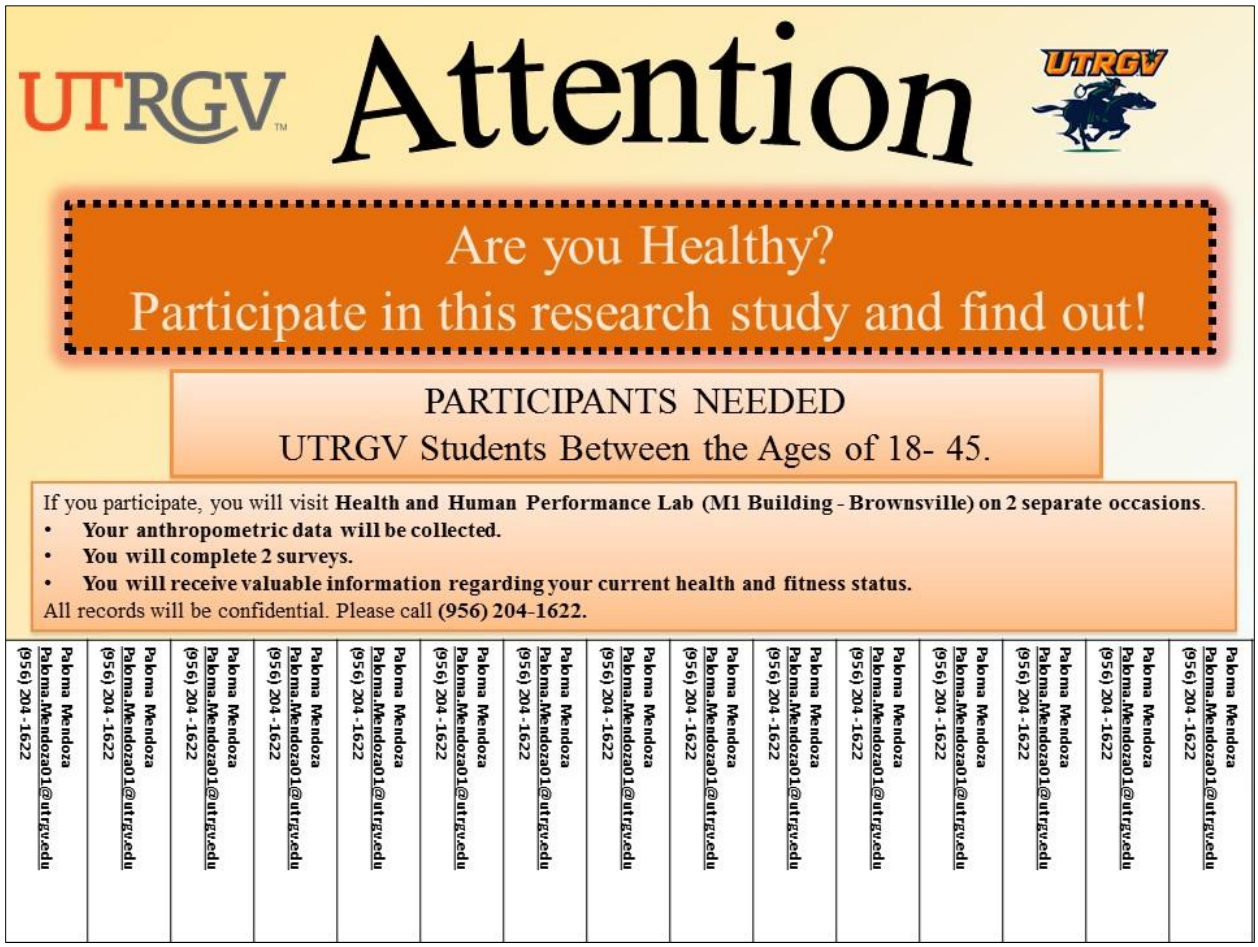
SVI -Stroke Volume Index

SVR -Systemic Vascular Resistance

TRG -Triglyceride

TVI -Total Vascular Impedance

1. RECRUITMENT FLYER



University of Texas Rio Grande Valley

Informed Consent to Participate in a Research Study

Project Title: The Relationship among Physical Activity, Diet, Blood Glucose, and Arterial Elasticity in College Students.

Principal Investigator: Dr. Ulku Karabulut, Ph.D.

Department: Health and Human Performance

You are being asked to volunteer for this research study. We are conducting a research study to investigate the association among Physical Activity, Diet, Blood Glucose, and Arterial Elasticity. You are selected as a possible participant because of your inquiry into the study. *If you are not 18 or older, please inform the researcher and do not participate the study.*

Please read this form and ask any questions that you may have before agreeing to take part in this study.

Procedures

If you agree to participate in this study, you will be asked to do the following:

a. You will be required visit Exercise Science Laboratory in the Department of Health and Human Performance located at M1 Building Room 216 in Brownsville Campus on 2 separate days for a total time commitment of, approximately, 75 - 90 minutes.
b. On the first visit, you will be required to read and sign an informed consent form before any kind of data collection takes place. You will be provided a hard copy of the consent form for your records.
c. After all paperwork is completed, you will be asked to complete 2 surveys - Metabolic Syndrome Questionnaire (MetSQ), Dietary screener questionnaire (DSQ) and 1 form – and Medical History Form.
d. Next, your contact information – 1 phone number, e-mail, and demographic and anthropometric data will be collected. Anthropometric measures that include resting heart rate (RHR), resting blood pressure (RBP), height, weight, hip and waist circumference will be recorded.
e. You will be assigned an accelerometer to measure your physical activity and will be instructed on how to properly wear it. An accelerometer simply provides objective measurements of physical activity level. It is a very small instrument. You will wear the accelerometer for **3 consecutive days (2 weekdays and 1 weekend**). You will take it off when off swimming or taking a shower or bath. This will conclude your first visit.
f. After your 1st visit, you will be contacted via phone or e-mail (depending on your preference) to schedule your second visit. Your second visit, if possible, will be scheduled one week later than your first visit.
g. For your second visit, you will **come to the lab early in the morning, hydrated** and be fasting.

The University of Texas Rio Grande Valley
IRB APPROVED
IRB# 2017-058-03
Expires: 04/10/2018

a. On your second visit, we will collect two drops of blood via finger stick to measure your fasting-blood glucose, HDL/LDL, and Total Cholesterol (TC) levels.
b. Next, we will collect urine sample to check your hydration level. Hydration level is essential accurately measure your body composition and how fast your blood travels. If you are not hydrated, you will be asked to drink some more water. If you are hydrated, your body composition will be measured.
c. Then, you will lie down on your back for a minimum of 10 minutes and the velocity of the blood flow from your neck to your wrist will be measured using two analyzers. This will conclude the study.

This study has the following risks:
There is no risk associated with your participation to this study. However, collecting blood sample via finger stick might cause a temporary discomfort on your finger. Blood sample will be collected only once. In the unlikely event you will seek medical care, you will be responsible for providing transportation to your family doctor, and are financially responsible for medical expenses.

Benefits of being in the study are:
The benefits of participating in this study is that you will receive very valuable information regarding your current health including blood glucose, body composition, arterial elasticity, resting blood pressure, heart rate, and physical activity level for **FREE**.

Compensation
A professor can offer extra a credit, but there will be alternatives to students who do not wish to participate. The individual can acquire extra credit by means of a written report that is relevant to the class material.

Voluntary Participation:
Participation in this study is voluntary. If you decline not to participate, you will not be penalized or lose benefits or services unrelated to the study. If you decide to participate, you may decline to answer any question and may choose to withdraw at any time.

Confidentiality:
In published reports, there will be no information included that will make it possible to identify you without your permission. Research records will be stored securely for 3 years after completion of the study and only approved researchers will have access to the records.
Results of this study may be used in publications and presentations.

Contacts and Questions:
For questions about the research itself, or to report any adverse effects during or following participation, contact the researcher, Dr. Ulku Karabulut at 956 – 882 - 5990,
Ulku.Karabulut@utrgv.edu, One West University Blvd.
LHSB 2.728, Brownsville, TX 78520.

The University of Texas Rio Grande Valley
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IRB# 2017-058-03
Expires: 04/10/2018

have any questions about your rights as a participant, or if you feel that your rights as a participant were not adequately met by the researcher, please contact the IRB at (956) 665-2889 or irb@utrgv.edu.

Signatures: By signing below, you indicate that you are voluntarily agreeing to participate in this study and that the procedures involved have been described to your satisfaction. The researcher will provide you with a copy of this form for your own reference. In order to participate, you must be at least 18 years of age. If you are under 18, please inform the researcher.

_____ ____/_____/_____

Participant's Signature Date

The University of Texas Rio Grande Valley
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IRB# 2017-058-03
Expires: 04/10/2018

3. MEDICAL HISTORY FORM

MEDICAL HISTORY FORM

Full Name _____ **Date** _____

Please list ALL medications that you are presently taking:

_____

_____

_____

Please answer the following questions:

Please answer the following questions: **(Note: Please base your answers on episodes over the past THREE years, EXCEPT those questions marked with *)**	**Yes**	**No**	**Don't Know**
1. Do you have any chronic or recurrent illness?			
2. Do you have a history of heart problems? 3. Episodes of chest pain at rest or during exertion? 4. Shortness of breath?			
5. Have you experienced uneven, irregular or skipped heartbeats?			
6. Do you have high blood pressure?			
7. Do you have any episodes of dizziness, seizures or convulsions? 8. Have you ever fainted?			
*9. Is heart disease present in your family?			
*10. Have you ever been told that a member of your family died suddenly or had a heart attack at an early age?			
11. Do you have high blood cholesterol?			
12. Do you have a history of lung problems?			
13. Do you have a cigarette-smoking habit? How much? 14. Are you a diabetic?			
15. Are you asthmatic?			
16. Are you obese? (More than 30% overweight?) 17. Do you have anorexia or bulimia? 18. Have you had recent surgery?			
19. Do you have any muscle, joint, or back disorder that could be aggravated by physical activity? 20. Have you been diagnosed with mononucleosis?			
21. Do you have difficulty with physical exercise?			
22. Have you received advice from a physician not to exercise?			

Adapted from the State University of New York - Binghamton

4. DATA COLLECTION SHEET 1

College Students' Knowledge on Health, Physical Activity, and Nutrition

Day 1 Date: ___ /____ /_____ **ID number:**

Name: _____ Major:_____

Gender _M / F_ DOB:___/___/___ Age: _____

Year in School: First year Second year Third year Fourth Year No response

Ethnicity: _____

Contact Information	
Cell Phone #:	
e-mail :	

☐ Please circle your preferred method to be contacted (**E-mail / Phone call / Both**).

Height: _____ (cm) Weight: _____ (kg) Blood Pressure: _____/_____

Heart Rate: _____ Waist / hip circumference: _____

Tanita:_____

DON'T FORGET to attach Leisure Time Exercise Questionnaire

DON'T FORGET to provide Study Code, participant ID, and a password

Day 2 Date: ___ /____ /_____

Height: _____ (cm) Weight: _____ (kg) Blood Pressure: _____/_____

Heart Rate: _____ Waist / hip circumference: _____

Tanita:_____

DON'T FORGET to attach Leisure Time Exercise Questionnaire

DON'T FORGET to provide Study Code, participant ID, and a password

5. DATA COLLECTION SHEET 2

HDI/*PulseWave*™ CR-2000

Research CardioVascular Profile Report

Research Subject ID:

Research Subject Name:

Date:
Time:
Age:
Gender:
Height:
Weight:
BSArea:
Body Mass Index:

Average Blood Pressure Waveform

PARAMETER	RESEARCH SUBJECT VALUE
SYSTOLIC BLOOD PRESSURE	
DIASTOLIC BLOOD PRESSURE	
MEAN ARTERIAL BLOOD PRESSURE	
PULSE PRESSURE	
PULSE RATE (beats/min)	
ESTIMATED CARDIAC EJECTION TIME (msec)	
ESTIMATED STROKE VOLUME (ml/beat)	
ESTIMATED STROKE VOLUME INDEX ($ml/beat/m^2$)	
ESTIMATED CARDIAC OUTPUT (L/min)	
ESTIMATED CARDIAC INDEX ($L/min/m^2$)	
LARGE ARTERY ELASTICITY INDEX (ml/mmHg x 10) (Capacitive Arterial Compliance)	
SMALL ARTERY ELASTICITY INDEX (ml/mmHg x 100) (Oscillatory or Reflective Arterial Compliance)	
SYSTEMIC VASCULAR RESISTANCE ($dyne \cdot sec \cdot cm^{-5}$)	
TOTAL VASCULAR IMPEDANCE ($dyne \cdot sec \cdot cm^{-5}$)	

Eagan, MN 55121 +1-651-687-9999 Toll-Free: 1-888-*PulseWave* (785-7392)

Form: 00017-001 (Rev. A / 08.Oct. 99)

"For Research Purposes Only"

6. PROFESSOR PERMISSION SCRIPT

The University of Texas Rio Grande Valley
Professor Permission Script

My name is Paloma Mendoza. I am a graduate student from the Department of Health and Human Performance at the University of Texas Rio Grande Valley (UTRGV). I would like to ask permission to enter your classroom to invite your students to participate in my research study. My study is about The Relationship among Physical Activity, Diet, Blood Glucose, and Arterial Elasticity in College Students.

As part of participation, students will be asked to visit the Exercise Physiology Lab twice and complete 2 surveys. As part of the study, descriptive and anthropometric measurements (height, weight, resting heart rate (HR), blood pressure (BP), hip and waist circumference, body mass index (BMI)) will also be collected. Physical activity level (PA) will be estimated. Second session will include the same measurements (height, weight, resting heart rate (HR), blood pressure (BP), hip and waist circumference, body mass index (BMI) and surveys. The total commitment will be about 75 – 90 minutes. Participation in this research is completely voluntary; students may choose not to participate without penalty. All data will be confidential and stored in a locked file cabinet for 3 years and deleted after.

If allowed, I would like to come in at the beginning of the class time. I will ask you to please exit the classroom prior to and during students' involvement in my study to reduce any possible feeling of coercion to participate in the study.

This research study has been reviewed and approved by the UTRGV Institutional Review Board for the Protection of Human Subjects (IRB).

If you have questions about the researcher, please feel free to contact Dr. Ulku Karabulut at (956) 882 – 5990 or Ulku.Karabulut@utrgv.edu. Or, if you have any questions regarding your students' rights as participants in the study, please call the IRB at (956) 665-2889 or email at irb@utrgv.edu.

Do I have your permission to recruit students from your classroom(s)?

7. IN-PERSON RECRUITMENT SCRIPT

The University of Texas Rio Grande Valley
Recruitment Script

My name is Paloma Mendoza. I am a graduate student from the Department of Health and Human Performance at the University of Texas Rio Grande Valley (UTRGV). I would like to invite you to participate in my research study to **College Students' Knowledge on Health, Physical Activity, Nutrition and Healthy Eating**.

This research study has been reviewed and approved by the UTRGV Institutional Review Board for the Protection of Human Subjects (IRB).

In order to participate you must be between 18 and 45 years old. Participation in this research is completely voluntary; you may choose not to participate without penalty.

As a participant, you will be asked to visit the Exercise physiology lab in Brownsville campus 2 times for your physical activity (PA) and basic anthropometric measurements (height, weight, resting HR, blood pressure, body composition, hip & waist circumference). You will also be asked to complete 2 surveys about "diet and nutrition". The total commitment for the study is 75 – 90 minutes. All data will be confidential.

If you would like to participate in this research study, please contact one of the graduate students via e-mail or phone (956) 882 - 5991 to schedule an appointment.

Do you have any questions now? If you have questions later, please contact PI by telephone at (956) 882 - 5990 or by email at Ulku.Karabulut@utrgv.edu.

BIOGRAPHICAL SKETCH

Paloma J. Mendoza, Bachelor's degree in Exercise Science May 2015 acquired from The University of Texas at Brownsville, Master's degree in Exercise Science December 2019 acquired from the University of Texas Rio Grande Valley; 1200 Guadalupe Circle, Brownsville, Texas 78526. Paloma-mendoza@att.net