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## The association between the Framingham CVD risk profile, SCAT, VAT, and physical activity in older adults

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THE ASSOCIATION BETWEEN THE FRAMINGHAM CVD RISK PROFILE, SCAT,  
VAT, AND PHYSICAL ACTIVITY IN OLDER ADULTS

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A Thesis

Presented To

Eastern Washington University

Cheney, Washington

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In Partial Fulfillment of the Requirements

for the Degree

Master of Science

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By

Kristine B. Siler

Spring 2013

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## Abstract

**INTRODUCTION:** Cardiovascular Disease (CVD) is the leading cause of death in the world, even though it can be prevented. Research has identified many risk factors, including cholesterol, physical activity, and abdominal obesity. The use of ultrasound has made it possible to distinguish between visceral adipose tissue (VAT) and subcutaneous adipose tissue (SCAT). **PURPOSE:** To determine the relationship between the Framingham CVD risk profile, VAT, SCAT, and physical activity in older adults. **METHODS:** CVD risk factors were measured in 23 males and 39 females ranging in age from 39-82. To determine the Framingham CVD risk score, TC and HDL-C were measured and entered into a score calculator. An ultrasound scan of the abdomen was used to determine VAT and SCAT depths. Pearson's correlations were done to determine associations between all of the variables. **RESULTS:** SCAT was significantly correlated with the 30-year score, SBP, and TC ( $r = .342$ ,  $r = .350$ ,  $r = .402$ ,  $p \leq .01$ ). VAT was correlated with PA and SBP ( $r = -.254$ ,  $p \leq .05$ ;  $r = .395$ ,  $p \leq .01$ ). In individuals 54 years and under, VAT was correlated with PA and SBP ( $r = -.447$ ,  $p \leq .05$ ;  $r = .602$ ,  $p \leq .01$ ). SCAT was correlated with SBP and TC ( $r = .551$ ,  $r = .610$ ,  $p \leq .01$ ). In individuals over 54 years, SCAT was correlated with the 30-year score ( $r = .410$ ,  $p \leq .05$ ) and VAT was correlated with TC ( $r = -.420$ ,  $p \leq .05$ ). In obese individuals, VAT was correlated with PA ( $r = -.597$ ,  $p \leq .05$ ) and SCAT was correlated with the 30-year score ( $r = .770$ ,  $p \leq .01$ ). **DISCUSSION:** The results show that SCAT was correlated with the 30-year score, while VAT was correlated with PA. Age and BMI levels may play a role in the effect of abdominal fat on CVD risk.

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## Chapter 1



## Introduction

The Framingham Heart Study, established in 1948, is an ongoing research project that at its inception identified cardiovascular disease (CVD) risk factors throughout the years. The earliest milestone in the Framingham Heart Study established that cigarette smoking was directly related to CVD (Arruda, 2012). From there, cholesterol levels, blood pressure, and obesity were also found to play a role in the development of CVD. Today research focuses on the prevention of CV-related diseases. A 30-year CVD risk score has been developed which is used to predict an individual's risk for CVD (Pencina, D'Agostino, Larson, Massaro, & Vasan, 2009). The score is based on numerous risk factors, both modifiable and non-modifiable, including sex and age, body mass index (BMI), total cholesterol (TC), systolic blood pressure (SBP), type 2 diabetes, and smoking status.

Current research has focused on how the Framingham CVD risk score is related to other factors of CVD, such as abdominal obesity and physical activity levels. There are discrepancies in the literature that make it unclear as to how abdominal obesity and physical activity play a role in measuring CVD risk. While obesity is a known risk factor for CVD, it is unclear as to why some seemingly lean individuals are at greater risk of CVD than one who is clinically obese (Hamdy, Porramatikul, & Al-Ozairi, 2006; Lopez-Jimenez et al., 2013; Porter et al., 2009; Stefan et al., 2008). The distribution of body fat and the type of fat storage found in the abdomen may help to determine the degree of CVD risk. Furthermore, physical activity is associated with a more favorable CVD risk profile (Ford, 2002). However, the CVD response to physical activity tends to take longer in older individuals (King et al., 1995).

Unfavorable lipid profiles are associated with CVD. The earliest association was a positive correlation between TC and CVD (Irzanski, Sliwczynska-Rodziewicz, Pawlicki, & Kowalski, 2012; Ridker, Hennekens, Buring, & Nader, 2000; Wilson, Abbott, & Castelli, 1988). When the components of TC were identified and became a routine measurement, cholesterol was classified as “good”, high-density lipoprotein cholesterol (HDL-C) and “bad”, low-density lipoprotein cholesterol (LDL-C). The association for LDL-C was positive; as levels increased, so did the risk for CVD. High-density lipoprotein cholesterol and CVD were inversely related, showing that as levels increased, CVD risk decreased. At the same time, researchers were questioning the association of weight and body fat after positive correlations were found for CVD and obesity following the BMI data. Still, it was not clear whether general obesity was to blame or if it was the specific location of the fat in the abdomen, or possibly a combination of both. Perhaps it has to do with some of the non-modifiable risk factors, such as sex and age that seemed to change the risk ratio. For example, males are at greater risk than females until after menopause, even when both have equal amounts of fat (Spangenburg, Wohlers, & Valencia, 2012). This led researchers to investigate how each of the risk factors are associated to determine a way to predict the risk for CVD.

Low levels of HDL-C may be directly related to the depth of visceral adipose tissue (VAT) in the abdomen. However, this risk factor becomes less pertinent in older individuals due to the theory that as people age, increases in abdominal fat may not be correlated with lower HDL-C levels (Ford, 2002). Thus, the literature is unclear as to whether the CVD risk factors are related to increased abdominal fat in older individuals

(Nieves et al., 2003; DeNino et al., 2001). This leads to the question: can older individuals with abdominal obesity exhibit a metabolically benign form of abdominal fat?

The literature is unclear as to whether VAT or subcutaneous adipose tissue (SCAT) is responsible for the unfavorable CVD risk profile. Simple anthropometric measurements such as BMI, WC, and WHR are currently used to determine abdominal obesity. Although they are inexpensive and easy to use, these measurements do not effectively determine central adiposity and cannot distinguish between VAT and SCAT (Lopez-Jimenez et al., 2013; Sogabe, Okahisa, Hibino, & Yamanoi, 2012). Advances in technology have made it possible to measure abdominal fat more effectively. A variety of machines can measure abdominal fat and can identify SCAT from VAT, but until now the procedures were expensive. With a field ultrasound device, researchers are able to measure SCAT and VAT separately more efficiently and at a lower cost.

Physical activity level has both positive and negative effects on CVD risk. Research has shown that individuals who partake in leisure time physical activity present with a more favorable CVD risk profile (Held et al., 2011). Improved cholesterol levels, BMI, WHR, and inflammatory markers are among the benefits of physical activity (Abramson & Vaccarino, 2002; Ford, 2002; Glazer et al., 2012; Held et al., 2011; King, Haskell, Young, Oka, & Stefanick, 1995; Manini et al., 2006). In young adults this relationship is widely accepted; however, the literature on older adults is less consistent. HDL-C seems to improve with increased physical activity levels in older adults compared to TC, BMI, or WHR. However, the improvements may take longer to elicit a favorable CV response (King et al., 1995). It takes two years for older adults to see improvements

in CVD risk, whereas it takes only one year for younger individuals to see similar benefits.

### **Statement of the Problem**

Research on CVD prevention is an evolving field of study. Risk factors continue to be identified and are evaluated on their association with the known CVD risk profile. Technical advances are also making it possible to measure the risk markers more reliably and at lower cost to the researcher. Cholesterol levels, physical activity levels, and abdominal obesity are among the strongest CVD risk markers. Separating the abdominal fat, high levels of VAT are believed to contribute to the development of CVD, while the role of SCAT remains controversial. Finally, the association of advancing age and risk markers emphasizes the need to use a cross-section of ages in the research population. Therefore, the purpose of this study was to determine the association between the Framingham CVD risk profile (TC, HDL-C, SBP, smoking, BMI, sex, and age), SCAT, VAT, and physical activity levels in adults over the age of forty.

### **Hypothesis**

The null hypothesis was there would be no significant correlations between the Framingham CVD risk profile, SCAT, VAT, and physical activity levels in older adults.

### **Operational Definitions**

Abdominal fat was divided into two compartments: VAT and SCAT. Visceral adipose tissue is the fat that lies in and around the organs, beneath the muscle layer in the abdomen. Subcutaneous adipose tissue is the fat that is stored superficial to the muscle layer, just beneath the skin. The BodyMetrix Pro Systems ultrasonography machine (IntelaMetrix, Inc., Livermore, CA) was used to measure VAT and SCAT in mm. TC

and HDL-C were measured in mg/dL in a fasting state using the Cholestech LDX Analyzer. Resting blood pressure was measured in mmHg using an aneroid sphygmomanometer following a five-minute rest period. Height was measured in inches and weight was measured in pounds with a physician's scale. These measurements were converted to metric units to calculate BMI, using the formula weight in kilograms divided by height in meters squared.

### **Delimitations**

This study was delimited to a sample of convenience of 64 males and females over 39 years of age.

### **Assumptions**

It was assumed that all participants truthfully and accurately filled out the physical activity questionnaire. It was assumed that the ultrasound machine was a reliable and valid measurement of VAT and SCAT.

### **Significance**

It was hoped this study would provide a method of identifying CVD risk earlier, and possibly prevent or prolong the onset of CVD. If so, this may help to compress morbidity time, which could positively affect the current health care costs.

### **Summary**

This chapter discussed the evolution of the Framingham Heart Study in CVD risk factor analysis. Several risk factors have been identified and there is still a need for research to determine how each of them is associated. Technological advances have made it possible to efficiently and inexpensively evaluate CVD risk. Thus, the purpose

of this study was to determine the association between the Framingham CVD risk profile, SCAT, VAT, and physical activity levels in older adults.

## **Chapter 2**

### **Literature Review**

#### **Introduction**

CVD is the leading cause of death in the world, even though it can be prevented. Research has identified risk factors to aid in the diagnosis and prevention of CVD. Cholesterol levels, physical activity levels, and abdominal obesity are among the strongest CVD risk markers (Arruda, 2012). High levels of VAT are believed to contribute to the development of CVD, while the role of SCAT remains controversial with respect to CVD risk. The purpose of this study was to determine the association between the Framingham CVD risk profile, SCAT, VAT, and physical activity levels in older adults. This chapter discusses the associations between these four risk factors and CVD.

#### **Framingham CVD Risk Profile**

The Framingham Heart Study is an ongoing research project that began in 1948. When it began, CVD was just beginning to expand as Americans were living longer. No research had yet identified risk factors for the disease that were specifically associated with certain lifestyle behaviors. The original study consisted of adults aged 30-62, who have been evaluated annually with respect to CV-related diseases and mortality over their lifespan (Arruda, 2012). A secondary study evolved when the need for establishing guidelines for a younger population was identified. This study is known as the Offspring Study, and participants include the children of the original cohort. Once the researchers felt confident in the various risk factors identified, they wanted to use this cohort to determine the existence of genetic variables that may be related to CVD (Arruda, 2012).

Today, the research is focused on determining ways to predict an individual's risk of CVD by combining risk factors from previous data. Researchers developed a method to predict the 30-year risk score for CVD using identified risk factors from the Framingham Offspring Study (Pencina et al., 2009). The risk factors included in this score are sex, age, systolic blood pressure (SBP), BMI, TC, HDL-C, diabetes, smoking status, and treated hypertension (Pencina et al., 2009). Each of these risk factors has been identified as independently related to CVD, although CVD risk increases exponentially when these risk factors are combined. This grouping of risk factors has been named the metabolic syndrome when the variables are combined (Carnethon et al., 2004). The degree of CVD risk is simply evaluated by how many risk factors each individual possesses and is diagnosed when at least three are combined.

### **Framingham risk factors**

Risk factors for CVD can be classified as either fixed (non-modifiable) or modifiable. Fixed risk factors are those that cannot be altered to improve CVD risk, such as age, sex, and treated hypertension. Modifiable risk factors are those that can be changed to improve the CVD risk profile of an individual. These include cholesterol, smoking status, and BMI. Cholesterol is a modifiable risk factor that is frequently used in research as the focus for CVD prevention (Irmanski et al., 2012; Rein, Saley, Beer, Vonbank, & Drexel, 2010; Ridker et al., 2000; Wilson et al., 1988).

Cholesterol can be separated into two types: LDL-C and HDL-C. LDL-C is considered "bad" cholesterol because it consists mostly of lipids with a few protein components (McArdle et al., 2007). LDL-C molecules are attracted to the arterial wall, causing an unfavorable condition of narrowing the blood vessel (McArdle et al., 2007).



Therefore high levels found in the bloodstream are associated with increased cardiovascular risk. HDL-C is commonly referred to as “good” cholesterol because they remove cholesterol from the arterial walls and transport it to the liver where it is metabolized or excreted (McArdle et al., 2007; Silverthorn, 2004). As a result high levels of HDL-C decrease cardiovascular risk (Silverthorn, 2004). High levels of LDL-C, represented as TC, and low levels of HDL-C are included in the CVD risk profile due to their effects on the cardiovascular system.

Table 1

*ACSM's criteria for CVD risk factors*

Risk Factors	Criteria
1. Family history	Myocardial infarction, coronary revascularization, or sudden death before 55 years of age in father or other male first degree relative, or before 65 years of age in mother or other female first degree relative
2. Cigarette smoking	Current or those who quit within the previous 6 months
3. Hypertension	SBP $\geq$ 140 mmHg or DBP $\geq$ 90mmHg, or on antihypertensive medication
4. Dyslipidemia	LDL-C $\geq$ 130 mg/dL, HDL-C $<$ 40 mg/dL, or TC $\geq$ 200 mg/dL Negative risk factor: HDL-C $\geq$ 60 mg/dL
5. Impaired fasting glucose	Fasting blood glucose $\geq$ 100 mg/dl
6. Obesity	BMI $\geq$ 30 kg/m <sup>2</sup> or WC $>$ 102 cm for men and $>$ 88 cm for women or WHR $\geq$ 0.95 for men and $\geq$ 0.86 for women
7. Sedentary lifestyle	Persons not participating in a regular exercise program or not meeting the minimal physical activity recommendations from the U.S. Surgeon General's Report

The American College of Sports Medicine risk stratification for individuals who are at risk for CVD has defined the criteria for each risk factor (Table 1) (Armstrong et al., 2006). This is supported in the literature as most researchers have found that HDL-C is inversely correlated with CVD risk, while TC and LDL-C are directly associated with CVD risk (Irzanski et al., 2012; Rein et al., 2010; Ridker et al., 2000; Wilson et al., 1988). In older adults, low levels of HDL-C are associated with increased coronary mortality. HDL-C is a strong predictor of CV related death, a finding that is less consistent with other types of death (Wilson et al., 1988). This association is the strongest at the lowest levels of HDL-C (Wilson et al., 1988). When participants were matched for similar baseline characteristics, TC and LDL-C levels were significantly higher among women with a history of CV events compared to those without CV events (Ridker et al., 2000). Individuals with a history of CV events also present with lower levels of HDL-C compared with those without CV events (Ridker et al., 2000). One theory suggests that BMI is inversely related to HDL-C, suggesting that leaner individuals have higher levels of HDL-C, thus are at less risk for CVD and coronary mortality (Wilson et al., 1988).

Furthermore, the metabolic syndrome is closely related to CVD. The metabolic syndrome is identified by visceral adiposity, unfavorable lipid profiles, and high levels of inflammation, similar to CVD (Carnethon et al., 2004). HDL-C is negatively correlated with vascular complications in individuals with the metabolic syndrome (Irzanski et al., 2012; Rein et al., 2010). Evidence suggests that HDL-C is strongly associated with the metabolic syndrome and is an independent predictor of CV events (Rein et al., 2010). Elevated levels of HDL-C have a protective influence on blood vessels, specifically by

reversing cholesterol transport in the blood (Irzanski et al., 2012; Rein et al., 2010). HDL-C particles are also negatively correlated with inflammatory markers, consequently lessening the pressure on arterial walls and protecting them from adverse CV events (Rein et al., 2010). In individuals with the metabolic syndrome, higher levels of HDL-C may help prevent CV incidents (Irzanski et al., 2012).

### **Abdominal Obesity**

Current research suggests that obesity is linked to CVD. Individuals who carry excess body weight are at a greater risk for CVD and death (Stevens et al., 1998). Researchers from the Framingham Heart Study suggested that obesity, measured as a percentage of desirable weight, is an independent predictor of CVD (Hubert, Feinleib, McNamara, & Castelli, 1983). This finding was greater among women, although it was significant for men as well. General obesity is not a specific enough measure to determine if an individual is at risk for CVD, however. Body fat distribution plays an important role in determining CVD risk. Individuals with greater abdominal fat are at a higher risk than those with greater lower body fat (Porter et al., 2006; Stefan et al., 2008). Still, this is not a definitive measurement to determine CVD risk.

Current research is focused on distinguishing between metabolically benign obesity and obesity that is detrimental to CV health (Angeras et al., 2012; Hamdy et al., 2006; Porter et al., 2009; Stefan et al., 2008; Stevens et al., 1998;). Using BMI as a measurement of obesity, there is evidence that the body may have a protective fat depot (Angeras et al., 2012). Underweight and normal-weight individuals with acute coronary syndromes have the highest risk of mortality compared to overweight or obese individuals (Angeras et al., 2012). There is evidence to suggest that an individual may be

clinically overweight and still have a lower risk for mortality than a normal or underweight individual (Flegal, Graubard, Williamson, & Gail, 2007). This supports the theory of a protective fat depot when BMI is used to calculate obesity. Similarly, a normal-weight individual with excess abdominal fat is at greater risk for death than an obese individual with less abdominal fat (Lopez-Jimenez et al., 2013). Thus, an apparently lean individual may be at a higher risk for CV related events than one who is clinically obese, depending on the distribution of body fat (Hamdy et al., 2006; Porter et al., 2009; Stefan et al., 2008).

### **VAT vs. SCAT**

There are discrepancies in the research with respect to the two types of fat found in the abdomen and their relationship to CVD risk. Most researchers agree that greater amounts of VAT increase CVD risk (Demerath et al., 2008; Fox, 2007; Liu et al., 2010). However, it remains controversial as to whether or not SCAT is associated with CVD risk, or if it is beneficial. The literature suggests that obesity may be a determinant of the lipoprotein profile (Nieves et al., 2003). There is evidence that VAT increases with age and that this relationship may be responsible for the unfavorable lipid profiles that increase the CVD risk in older individuals (DeNino et al., 2001). BMI is positively associated with CVD risk, demonstrated by increasing triglycerides and decreasing HDL-C, but is not associated with unfavorable LDL-C levels (Nieves et al., 2003). More specifically, increases in VAT are associated with a more atherogenic lipid profile, defined by an increase in triglycerides, TC, and LDL-C, and a decrease in HDL-C (Nieves et al., 2003). This relationship is also positively associated with age and VAT progressively increases with age (DeNino et al., 2001). One theory for the age-related

association in females is the hormonal changes that occur after menopause. A lack of estrogen seems to be strongly associated with increases in visceral fat cell size, thus rapidly increasing the risk for CVD (Spangenburg et al., 2012). This is associated with unfavorable changes in the lipid profile, including increasing LDL-C and TC levels (DeNino et al., 2001). Unfavorable changes in lipid profiles and increasing levels of VAT are also associated with a moderate decrease in insulin sensitivity (DeNino et al., 2001; Nieves et al., 2003). Thus, reducing VAT will help to improve lipid profiles and insulin sensitivity, especially in older adults (DeNino et al., 2001; Nieves et al., 2003).

The literature suggests that VAT is a reliable predictor of CVD risk, independent of BMI (Fox et al., 2007; Liu et al., 2010; Smith, J. et al., 2012; Taksali, S. E. et al., 2008). In a population of adolescents with similar BMI scores, those with greater VAT were at a higher risk for metabolic complications (Taksali et al., 2008). One study determined that while BMI is not related to LDL-C, VAT is positively associated with LDL-C and TC, suggesting that VAT is a better indicator of a more atherogenic lipid profile that increases CVD risk (Nieves et al., 2003). Increasing levels of VAT is associated with unfavorable lipid profiles, including higher levels of triglycerides and lower levels of HDL-C (Nieves et al., 2003; Smith et al., 2012). These findings are consistent in individuals with and without type 2 diabetes (Smith et al., 2012). Furthermore, the evidence suggests that VAT is a better independent predictor of CV related diseases than SCAT (Demerath et al., 2008).

With respect to SCAT, the evidence is controversial. Some researchers have found that increasing levels of SCAT are accompanied with a higher CVD risk score (Fox et al., 2007; Liu et al., 2010). This theory is justified with the evidence that the amount of

SCAT is directly related to the amount of VAT. Conversely, other researchers believe that SCAT is beneficial to CV health, specifically in individuals with unfavorable lipid profiles (Porter et al., 2009; Smith et al., 2012; Taksali et al., 2008). Elevated levels of SCAT tend to be more protective against CV related diseases as levels of VAT increase (Demerath et al., 2008; Porter et al., 2009). Triglyceride levels tend to decrease with greater amounts of SCAT, although this is only evident in individuals with the highest levels of VAT (Porter et al., 2009). Conversely, Taksali et al. (2008) found that those with higher levels of SCAT tend to have lower levels of VAT. These individuals exhibit a more favorable metabolic profile (Taksali et al., 2008). The evidence is unclear as to whether the higher levels of SCAT or the lower levels of VAT are responsible for the improved lipid profile.

### **Measurement tools**

Current guidelines for measuring abdominal obesity include BMI with waist circumference (WC) and waist-to-hip ratio (WHR). These measurements are easily attainable and do not require expensive equipment; however, they are not reliable methods to measure abdominal fat, they are estimates at best. BMI is a ratio of height and weight, but it does not take into account body fat distribution or muscle mass (Lopez-Jimenez et al., 2013). A seemingly thin individual with normal BMI and excess abdominal fat is likely to have less muscle mass as well, an indication of a lower fitness level (Lopez-Jimenez et al., 2013). WC is often used in addition to BMI to determine if there is a greater amount of fat around the abdomen. The WHR operates on a similar concept, comparing the girth around the hips to abdominal girth, assuming that these measurements reflect the amount of fat mass in the respective areas. Still, none of these

methods can accurately assess the amount of fat in the abdomen, nor can they differentiate between VAT and SCAT. While an MRI or CT scan is the gold standard for measuring VAT and SCAT separately, these methods are expensive and not readily available to clinicians as a preventative screening tool (Hamdy et al., 2006). Valsamakis et al. (2004) sought to determine the best anthropometric measure that most closely relates to the results of an MRI or CT scan. They determined that WC is the best measure compared with BMI and/or WHR (Valsamakis et al., 2004). However, WC cannot effectively distinguish between SCAT and VAT, thus it is not an accurate measurement of obesity as a CVD risk factor (Sogabe et al., 2012).

Ultrasonography is a reliable method for measuring VAT and SCAT (Gong et al., 2007; Hirooka et al., 2005; Johnson, Naccarato, Corder, & Repovich, 2012; Sogabe et al., 2012). The BodyMetrix is an ultrasound machine that can determine body fat percentage as accurately as bioelectrical impedance (BIA) and air displacement plethysmography (Johnson et al., 2012; Shojaei et al., 2010). The BodyMetrix also has the ability to distinguish between VAT and SCAT, which makes it a more desirable measure to determine CVD risk. In a study that measured VAT by determining the depth of the perirenal fat deposits, the ultrasonic measurement was equivalent to that of the MRI scan (Gong et al., 2007). Compared with computed tomography, ultrasonography is equally effective in measuring VAT and SCAT (Hirooka et al., 2005). However, the accuracy of ultrasonography is dependent on the ability of the operator to be precise and consistent with the measurements (Shojaei et al., 2010). The ultrasound operators need to be trained for proper scanning technique and should have adequate knowledge of the anatomical landmarks around the area being measured (Shojaei et al., 2010).

### **Physical Activity Level**

The American College of Sports Medicine guideline for exercise prescription states that adults aged 18-65 years need a minimum of 30 minutes of moderate-intensity aerobic exercise on five days each week, or a minimum of 20 minutes of vigorous-intensity aerobic exercise on three days each week (Haskell et al., 2007). Muscular strengthening exercises should be performed at least two days per week (Haskell et al., 2007). This protocol is recommended to promote and maintain a healthy lifestyle.

Researchers have determined that individuals who partake in regular exercise have lower CVD risk (Abramson & Vaccarino, 2002; Ford, 2002; Glazer et al., 2012; Held et al., 2011; King et al., 1995; Manini et al., 2006). Current literature suggests that physical activity level is inversely related to TC, WHR, BMI, and C-reactive protein levels (Ford, 2002). This association was prevalent in a younger population; however, there is discrepancy in the literature with respect to older adults (King et al., 1995). The HDL-C may be a more accurate measure of physical activity on CVD risk for older adults. The evidence suggests that HDL-C is directly associated with physical activity level in both younger and older adults (Ford, 2002; Glazer et al., 2012; King et al., 1995). However, it may take longer for older adults to see a significant change in the lipid profile. In a study of men and women ages 50-65 years, HDL-C improved significantly over a two-year period of regular exercise (King et al., 1995). These results are similar to those of a younger population over a one-year period of regular exercise, indicating that the benefits of exercise take more time to evolve in older individuals. Older adults who demonstrate a higher energy expenditure and resting metabolic rate have a lower risk of mortality (Manini et al., 2006).



Evidence suggests that more frequent, shorter bouts of exercise have a greater CV benefit than less frequent, prolonged exercise (Abramson & Vaccarino, 2002; Glazer, et al., 2012; King et al., 1995). Shorter bouts of physical activity are significantly related to a lower Framingham CVD risk score (Glazer et al., 2012). Researchers found a stronger association between the frequency of exercise and HDL-C levels compared to the intensity of exercise and HDL-C levels (King et al., 1995). Similarly, Abramson and Vaccarino (2002) determined that individuals who participate in regular exercise more frequently have lower levels of C-reactive protein and other inflammatory markers. Increased levels of HDL-C and decreased levels of C-reactive protein are beneficial to CV health. Physical activity levels are significantly correlated with a lower prevalence of obesity (Glazer et al., 2012). Lower waist circumference, BMI, and triglycerides are associated with physical activity, independent of duration (Glazer et al., 2012). However, leisure time physical activity is more beneficial to CV health than occupational physical activity (Held et al., 2011). The INTERHEART study determined that individuals who partake in occupational physical activity exhibit more CVD risk markers than those who participate in leisure time physical activity (Held et al., 2011). The theory behind this finding is that occupational physical activity is more anaerobic than aerobic, such as heavy lifting. Anaerobic activity is not as beneficial to CV health. Overall, physical activity is associated with favorable metabolic profiles that improve CV health.

### **Summary**

The literature presents evidence that CVD risk is an area that requires continuous research to develop preventative measures to decrease such risk. Abdominal obesity is associated with lipid profiles and physical activity; however, the evidence linking VAT

and SCAT to CVD risk is unclear. With improvements in technology, the use of ultrasound has recently been introduced as a way of distinguishing between VAT and SCAT. Thus, with the use of ultrasound, the purpose of this study was to determine the association between the Framingham CVD risk profile, SCAT, VAT, and physical activity levels in older adults.

## **Chapter 3**

### **Methods**

#### **Introduction**

The purpose of this study was to determine the relationship between the Framingham CVD risk profile, SCAT, VAT, and physical activity levels in adults older than 40 years of age. Specifically, we used ultrasonography to determine whether SCAT or VAT is a better indicator of abdominal obesity as a risk factor for CVD compared to the Framingham CV risk profile. This chapter describes the methodology that was employed to test the hypotheses of the current study. The methodology includes participant recruitment, information on the instrumentation used, an overview of the procedures, and a description of the statistical analysis that was performed.

#### **Participants**

A convenience sample of 64 volunteers was recruited for the present study through flyers, word of mouth, and referrals. This study was delimited to males and females over the age of 39 with no restriction for current disease diagnosis. The Institutional Review Board for Human Subjects at Eastern Washington University approved this study prior to recruitment. The detailed descriptive statistics of the study population are included in Table 2

Table 2

*Descriptive Statistics*

Variable	Mean $\pm$ Standard Deviation
Age (yrs)	56.16 $\pm$ 9.64
Height (in)	67.05 $\pm$ 3.63
Weight (lbs)	173.91 $\pm$ 40.11
BMI (kg/m <sup>2</sup> )	27.13 $\pm$ 5.80
SBP (mmHg)	118.77 $\pm$ 9.82
Physical Activity (min/day)	35.16 $\pm$ 22.83
TC (mg/dl)	203.03 $\pm$ 40.43
HDL-C (mg/dl)	61.52 $\pm$ 19.31
VAT (cm)	19.40 $\pm$ 8.31
SCAT (cm)	13.63 $\pm$ 7.40
Total US (cm)	33.03 $\pm$ 12.95
30-yr. Risk Score (%)	30.70 $\pm$ 15.14

**Instrumentation**

The following variables were collected for each participant: height, weight, age, sex, current medications, smoking status, blood pressure, HDL-C, TC, VAT, SCAT, and physical activity level. Age, sex, current medications, smoking status, and physical activity levels were collected via a questionnaire, given orally by the researchers.

Blood pressure was measured manually with a standard aneroid sphygmomanometer and stethoscope. Height and weight were measured with a standard physician's scale (Cardinal Manufacturing Company, Webb City, MO) and stadiometer. A Cholestec LDX blood analyzer (Cholestec Corporation, Hayward, CA) was used to

measure TC and HDL-C. VAT and SCAT were measured with a BodyMetrix Pro System ultrasonography machine (IntelaMetrix, Inc., Livermore, CA).

### **Procedure**

The purpose of the study was explained and all questions were answered at the time of recruitment. Participants were asked to fast for 12 hours prior to testing. All testing took place in the Human Performance Lab at Eastern Washington University. Upon entering the lab, participants were briefed again on the procedures of the study. They were asked to sit down and rest for five minutes, during which time they signed the informed consent and completed the physical activity questionnaire. Once the participant had been seated for at least five minutes the participant's blood pressure was measured. Height and weight were then measured and recorded. A small blood sample (35 microliters) was taken from a finger stick to measure TC and HDL-C. The finger stick was performed using a sterile lancet, collecting enough blood to fill the capillary tube. The blood was then transferred to an analyzer cassette and placed in the Cholestech LDX Analyzer. The final procedure was the ultrasound scan of the abdomen to assess VAT and SCAT. A 4 cm horizontal scan across the abdomen at a level of the umbilicus was performed, starting 1 cm to the right of the umbilicus to measure the VAT and SCAT levels. All data was collected on a separate data collection sheet, void of any identifiers to prevent bias. The data was then entered into a Microsoft Excel spreadsheet before being imported into SPSS. Participants received a handout with their results as well as educational material for their reference.

### **Analysis**

Statistical analysis was performed using SPSS version 20 software. Data was screened for outliers and checked for normality. Descriptive statistics were provided for both males and females on all variables and frequencies were calculated to determine the distribution of each of the variables. Pearson's correlations were used to examine the relationships between the Framingham CVD risk profile, all independent variables included in the risk profile, SCAT, VAT, and physical activity. Partial Pearson's correlations were calculated to adjust for age and BMI.

### **Summary**

This chapter presented the methods and procedures that were conducted for this study. Participants and instrumentation were identified, as well as a description of the type of statistics that was used to analyze the data.

## Chapter 4

### Results

#### Introduction

The purpose of this study was to determine the relationship between the Framingham CVD risk profile, SCAT, VAT, and physical activity levels in older adults. Specifically, we used ultrasonography to determine whether SCAT or VAT is a better indicator of abdominal obesity as a risk factor for CVD compared to the Framingham CV risk profile. This chapter discusses the results of the current study.

The detailed descriptive statistics of the study population are included in Table 2. A total of 64 participants were recruited for this study. Two participants were unable to complete all the data collection, thus were excluded from the study. This study consisted of 23 males and 39 females aged 39-82. The data was normally distributed, thus Pearson's correlations were calculated to determine relationships among the variables.

Initially, the entire population was analyzed to determine correlations between VAT, SCAT, Framingham CVD risk score, and physical activity levels (Table 3). There was a significant moderate positive correlation between VAT and SCAT ( $r = .355$ ,  $p = .005$ ) as well as between SCAT and the Framingham CVD risk score ( $r = .342$ ,  $p = .007$ ). Physical activity was negatively correlated with VAT ( $r = -.254$ ,  $p = .047$ ). There were no significant correlations between VAT and the Framingham CVD risk score, nor were there significant correlations between physical activity and SCAT or the Framingham CVD risk score.

Table 3

*Correlations between VAT, SCAT, 30-year risk score, and physical activity (PA)*

	VAT	SCAT	Total US	30-yr Risk	PA
VAT	1				
SCAT	.355**	1			
Total US	.845**	.800**	1		
30-yr Risk	.059	.342**	.238	1	
PA	-.254*	-.209	-.282*	.037	1

\*Significant at an alpha level  $p < .05$

\*\*Significant at an alpha level  $p < .01$

The data was then split into two age groups: individuals aged 54 and under and individuals aged 55 and over. This was determined by the median age of 54. In individuals aged 54 and under, there were no significant correlations with respect to the Framingham 30-year risk score (Table 4). VAT was significantly correlated with SCAT ( $r = .469$ ,  $p = .005$ ) and physical activity ( $r = -.447$ ,  $p = .012$ ). Physical activity was negatively correlated with the total US measurement ( $r = -.392$ ,  $p = .029$ ). In individuals ages 55 and over, the only significant correlation was found between SCAT and the Framingham CVD risk score ( $r = .410$ ,  $p = .022$ ). There were no significant correlations with respect to VAT or physical activity in individuals aged 55 and over.

Table 4



*Correlations between VAT, SCAT, 30-year risk score, and physical activity in individuals age 54 and under*

	VAT	SCAT	Total US	30-yr Risk	PA
VAT	1				
SCAT	.496**	1			
Total US	.884**	.844**	1		
30-yr Risk	.135	.256	.225	1	
PA	-.447*	-.215	-.392*	-.016	1

\*Significant at an alpha level  $p < .05$

\*\*Significant at an alpha level  $p < .01$

The data was then stratified into four levels by BMI measurements: underweight (BMI < 18.5), normal-weight (BMI = 18.5-24.9), overweight (BMI = 25-29.9), and obese (BMI  $\geq$  30). There was only one participant in the underweight group, thus that group was excluded. The remaining three groups had sufficient numbers in the n list (n = 23, n = 23, n = 14 respectively) for analysis. The normal and overweight groups did not yield any significant correlations between VAT, SCAT, total US, Framingham CVD risk score, or physical activity (Table 5). In the obese group, there was a significant negative correlation between VAT and physical activity ( $r = -.597$ ,  $p = .028$ ). The Framingham CVD risk score was positively correlated with SCAT ( $r = .770$ ,  $p = .001$ ) and total US ( $r = .719$ ,  $p = .006$ ). There were no significant correlations between VAT and physical activity with respect to the Framingham CVD risk score (Table 6).

Table 5

*Correlations between VAT, SCAT, 30-year risk score, and physical activity in normal weight individuals (BMI = 18.5-24.9)*

	VAT	SCAT	Total US	30-yr Risk	PA
VAT	1				
SCAT	.441*	1			
Total US	.887**	.805**	1		
30-yr Risk	.174	.092	.162	1	
PA	.132	.024	.100	.255	1

*\*Significant at an alpha level  $p < .05$*

*\*\*Significant at an alpha level  $p < .01$*

Table 6

*Correlations between VAT, SCAT, 30-year risk score, and physical activity in obese individuals (BMI  $> 30$ )*

	VAT	SCAT	Total US	30-yr Risk	PA
VAT	1				
SCAT	.347	1			
Total US	.830**	.811**	1		
30-yr Risk	.402	.770**	.719**	1	
PA	-.597*	-.215	-.500	-.183	1

*\*Significant at an alpha level  $p < .05$*

*\*\*Significant at an alpha level  $p < .01$*

Further analysis sought to determine the relationship between VAT, SCAT, total US, and physical activity on the Framingham CVD risk score and its individual components, including sex, age, SBP, TC, and HDL-C. Analysis of the entire sample

yielded significant correlations with respect to sex, SBP, and TC (Table 7). Sex was negatively correlated with SCAT ( $r = -.413$ ,  $p = .001$ ) and total US ( $r = -.324$ ,  $p = .010$ ). SBP was positively correlated with VAT ( $r = .395$ ,  $p = .001$ ), SCAT ( $r = .350$ ,  $p = .005$ ), and total US ( $r = .454$ ,  $p = .000$ ). TC was positively correlated with SCAT ( $r = .402$ ,  $p = .001$ ).

Table 7

*Correlations between the 30-year Risk Score variables (sex, age, SBP, TC, and HDL-C) and VAT, SCAT, Total US, and PA*

	Sex	Age	SBP	TC	HDL-C
VAT	-.138	-.141	.395**	-.036	-.036
SCAT	-.413**	.051	.350**	.402**	.140
Total US	-.324*	-.061	.454**	.207	.057
PA	-.101	.081	-.178	-.186	.017

\*Significant at an alpha level  $p < .05$

\*\*Significant at an alpha level  $p < .01$

In individuals aged 54 and under, there were no significant correlations with respect to age and HDL-C as they relate to VAT, SCAT, and physical activity (Table 8). There were significant negative correlations between total US and sex ( $r = -.359$ ,  $p = .048$ ), as well as between physical activity and SBP ( $r = -.386$ ,  $p = .032$ ). Significant correlations were found between SCAT and sex ( $r = -.503$ ,  $p = .004$ ), SBP ( $r = .551$ ,  $p = .001$ ), and TC ( $r = .610$ ,  $p = .000$ ). VAT and SBP were positively correlated as well ( $r = .602$ ,  $p = .000$ ). Total US was positively correlated with SBP ( $r = .668$ ,  $p = .000$ ) and TC ( $r = .487$ ,  $p = .006$ ). For individuals over the age of 54, there were no significant correlations with respect to sex, age, SBP, or HDL-C. The only significant correlation

that was found for this age group was a negative correlation between VAT and TC ( $r = -.420, p = .019$ ).

Table 8

*Correlations between the 30-year Risk Score variables (sex, age, SBP, TC and HDL-C) and VAT, SCAT, Total US, and PA in individuals age 54 and under*

	Sex	Age	SBP	TC	HDL-C
VAT	-.143	-.149	.602**	.256	-.102
SCAT	-.503**	.074	.551**	.610**	.190
Total US	-.359*	-.052	.668**	.487**	.039
PA	.116	-.131	-.386*	-.258	.140

\*Significant at an alpha level  $p < .05$

\*\*Significant at an alpha level  $p < .01$

After stratification of the sample into three groups based on BMI, significant correlations were found for the normal and obese groups, but not the overweight group. In obese individuals, SCAT was negatively correlated with sex ( $r = -.697, p = .009$ ) and positively correlated with SBP ( $r = .780, p = .007$ ) (Table 9). VAT was positively correlated with SBP ( $r = .625, p = .017$ ), and total US was positively correlated with SBP ( $r = .854, p = .000$ ). Physical activity was positively correlated with age ( $r = .713, p = .004$ ) and negatively correlated with SBP ( $r = -.574, p = .032$ ). Normal weight individuals did not yield significant correlations with respect to VAT and physical activity (Table 10). SCAT was positively correlated with HDL-C ( $r = .476, p = .022$ ) and negatively correlated with sex, and these correlations were significant ( $r = -.440, p = .036$ ). Total US was negatively correlated with sex as well ( $r = -.438, p = .037$ ).

Table 9

*Correlations between the 30-year Risk Score variables (sex, age, SBP, TC, and HDL-C) and VAT, SCAT, Total US, and PA in obese individuals (BMI • 30)*

	Sex	Age	SBP	TC	HDL-C
VAT	-.256	-.493	.625*	-.145	-.093
SCAT	-.697**	.053	.780**	.404	.217
Total US	-.574*	-.276	.854**	.150	.071
PA	.053	.713**	-.574*	-.191	.144

\*Significant at an alpha level  $p \cdot .05$

\*\*Significant at an alpha level  $p \cdot .01$

Table 10

*Correlations between the 30-year Risk Score variables (sex, age, SBP, TC, and HDL-C) and VAT, SCAT, Total US, and PA in normal weight individuals (BMI = 18.5-24.9)*

	Sex	Age	SBP	TC	HDL-C
VAT	-.321	-.121	.163	-.053	.149
SCAT	-.440*	.040	-.082	.257	.476*
Total US	-.438*	-.059	.065	.097	.343
PA	.040	.106	.065	.016	-.228

\*Significant at an alpha level  $p \cdot .05$

### Summary

Overall, the results of this study refuted the null hypothesis that there would be no significant associations between the Framingham CVD risk score, SCAT, VAT, and physical activity in older adults. There were significant correlations between SCAT and the Framingham CVD risk score in individuals over 54 years old as well as in obese individuals. VAT remained insignificant with respect to the Framingham CVD risk score

in all the groups. Physical activity was negatively correlated with VAT, and this correlation grew stronger in obese individuals.

## **Chapter 5**

### **Discussion**

#### **Introduction**

The purpose of this study was to determine the relationship between the Framingham CVD risk profile, SCAT, VAT, and physical activity levels in older adults. Specifically, we used ultrasonography to determine whether SCAT or VAT is a better indicator of abdominal obesity as a risk factor for CVD compared to the Framingham CVD risk profile. This chapter discusses the outcomes of the study and provides recommendations for future research on this topic.

#### **Principal Findings**

The Framingham CVD risk score, developed by researchers from the Framingham Heart Study, is a way to predict an individual's 30-year risk for being diagnosed with CVD (Pencina et al., 2009). Being able to assess CVD risk early may help to prevent or delay the onset of the disease. Since many of the risk factors are lifestyle related, an individual will have more time to change behavior and reduce risk. The original Framingham Study and more recently the Offspring Study identified numerous risk factors that are linked to the chance of developing CVD (Pencina et al., 2009). Through a statistical analysis to identify the strengths of the associations of the various risk factors, the Framingham CVD risk score is comprised of the strongest of these variables, some of which can be modified to improve the CVD risk score. The variables include sex, age, SBP, TC, HDL-C, BMI, smoking status, and treated hypertension (Pencina et al., 2009). These variables are entered into a calculator available on the Framingham website ([www.framinghamheartstudy.org](http://www.framinghamheartstudy.org)) that generates a

score representing CVD risk. The score represents CVD risk as a percentage, estimating the probability of developing CVD within 30 years.

In the current study, the results show that VAT is positively correlated with SCAT, although this relationship is weak ( $r = .355$ ,  $p = .005$ ). Surprisingly, SCAT, but not VAT, was positively correlated (albeit weakly) with the Framingham CVD risk score ( $r = .342$ ,  $p = .007$ ). After breaking the Framingham CVD risk score down into its individual components, SCAT remained significantly correlated with sex ( $r = -.413$ ,  $p = .001$ ), SBP ( $r = .350$ ,  $p = .005$ ), and TC ( $r = .402$ ,  $p = .001$ ), while VAT was only correlated with SBP ( $r = .395$ ,  $p = .001$ ). Past research has yielded similar results as the present study, determining that the amount of SCAT is directly related to the amount of VAT, and that both VAT and SCAT are associated with CVD risk (Fox et al., 2007; Liu et al., 2010). The CVD risk factors that were included in these studies are similar to those that were measured in the present study. Fox et al. (2007) used the Framingham Heart Study guidelines to determine the risk factors. These researchers used larger sample sizes of a similar age group as the current study. Other than the sample size, the population of the current study was similar to that of Fox et al. (2007) with respect to hypertension and diabetes. The participants in the Jackson Heart Study did not have CVD, however over half of them were obese and had been diagnosed with the metabolic syndrome (Liu et al., 2010). Unlike the sample in the current study, most of the participants also had hypertension (Liu et al., 2010).

The findings of the current study failed to support the theory that VAT is more strongly associated than SCAT with increased CVD risk, which many researchers have determined (Demerath et al., 2008; Fox et al., 2007; Liu et al., 2010; Smith et al., 2012;



Taksali et al., 2008). Most of these studies used a CT scan rather than ultrasound to measure VAT and SCAT, which may account for some of the inconsistencies between the current study and previous research. Also, most previous studies evaluated populations that were either overweight or obese, on average, while the current study was a mix of all weights and fitness levels. It is possible that fitness may be a stronger correlate than weight for risk, which has led to the “fit and fat” theory (Flegal et al., 2007).

Contrary to the current findings, Demerath et al. (2008) suggests that VAT is a better predictor of CVD than SCAT. This association was found in a larger sample with an average age that was 13 years younger than the average age in the current study. In adolescents, high levels of VAT with low levels of SCAT provided evidence of a phenotype for partial lipodystrophy (Taksali et al., 2008). These individuals were at greater risk for CVD and the metabolic syndrome at an age not generally associated with CVD (Taksali et al., 2008). While the population of the present study was older, greater than 40 years, there was no restriction based on current health status. Thus, while age is one of the non-modifiable risk factors for CVD and VAT is strongly associated as well, there may be another variable with a stronger influence that was not identified in the present study. One study found that SCAT is strongly associated with CVD risk only in those with the highest levels of VAT (Porter et al., 2009). The current study may not have had enough participants with great amounts of VAT to see this effect, as the sample may have been healthier than the general population.

## Effect of BMI

Further analysis indicated that these correlations grew stronger after stratifying the sample into groups based on BMI. Three groups were identified: normal-weight (BMI = 18.5-24.9), overweight (BMI = 25-29.9), and obese (BMI  $\geq$  30). The obese group yielded a strong positive correlation between SCAT and the Framingham CVD risk score ( $r = .770$ ,  $p = .003$ ), as well as with SBP ( $r = .780$ ,  $p = .003$ ). The total ultrasound measurement yielded a strong positive correlation with the Framingham CVD risk score ( $r = .719$ ,  $p = .006$ ) as well, suggesting that total abdominal fat may be more atherogenic than VAT or SCAT alone. Interestingly, SCAT was positively correlated with HDL-C in the normal weight group ( $r = .476$ ,  $p = .022$ ), adding to the literature that SCAT may have beneficial effects on CVD risk (Hamdy et al., 2006). Still, VAT was not significantly correlated with the Framingham CVD risk score in any of the BMI groups. The overweight group did not yield any significant correlations with the Framingham CVD risk score or any of the variables measured as a part of the score, providing additional evidence to the “fit and fat” theory (Flegal et al., 2007).

The “fit and fat” theory supports the idea that overweight is the new normal. The growing trends in America today suggest that on average, people are weighing more than they used to. Research suggests that overweight, but not obese, individuals are less likely to develop CVD and may live longer than one who is clinically normal-weight (Flegal et al., 2007). In a more recent review, it was also determined that those in the highest levels of obesity are at greater risk for mortality, whereas overweight is associated with lower mortality, and even the lowest level of obesity was not associated with mortality (Flegal et al., 2013). The current study suggests that overweight individuals can still be fit

according to CVD risk. Those in the overweight group did not display unfavorable measures of VAT or CVD risk factors. In fact, the average physical activity level in the current study was above the health recommendation of 30 minutes of physical activity on most days of the week (Armstrong et al., 2006). Physical activity was recorded in minutes per day, on average, and was not exercise specific. The current study found a negative correlation between VAT and physical activity ( $r = -.254$ ,  $p = .042$ ), a finding that grew stronger in obese individuals ( $r = -.574$ ,  $p = .032$ ). This suggests that physical activity may help to lower VAT levels, thus lowering CVD risk. Future research should focus on the roles of VAT and SCAT in a more diverse population, specifically those who may not be physically active, to determine how they are associated with BMI and CVD risk.

The Framingham Heart Study currently uses BMI to measure obesity (Hubert et al., 1983). However, more recent studies have argued that body fat distribution is more important than BMI calculations to determine CVD risk (Angeras et al., 2012; Fox et al., 2007). There is evidence that an apparently lean individual may be at greater risk for CVD than an obese individual, based on where the fat is located on the body (Angeras et al., 2012; Porter et al., 2006; Stefan et al., 2008). Typically, SCAT is associated with hip circumference, whereas VAT is associated with WC (Hamdy et al., 2006). This is one reason why some researchers believe that BMI is not sufficient to determine CVD risk. This theory is confirmed by the current study as shown by significant and strong positive correlations between SCAT and hip circumference ( $r = .780$ ,  $p = .000$ ). VAT was moderately correlated with both hip circumference ( $r = .566$ ,  $p = .000$ ) and WC ( $r = .505$ ,

$p = .000$ ) just as SCAT was correlated with WC ( $r = .535$ ,  $p = .000$ ), although these correlations are weaker.

### **Effect of Age**

Age may be an important factor when evaluating CVD risk. In the present study, individuals who were younger than 55 years old with increased VAT and SCAT exhibited a more unfavorable CVD risk profile compared with those who were 55 years and older. This is not consistent with the literature, which suggests that CVD risk increases with age as a direct result of increased abdominal fat (DeNino et al., 2001; Nieves et al., 2003; Spangenburg et al., 2012). Previous studies have concluded that VAT increases with age, and that this increase is associated with a more atherogenic lipid profile (DeNino et al., 2001; Nieves et al., 2003). For women, the effect of age on VAT and CVD risk may be a result of the hormonal changes that take place after menopause. Without estrogen, VAT cells increase in size, thus potentially increasing the risk for CVD (Spangenburg et al., 2012). This effect was found in animals, so it is only theoretically attributable to humans. Spangenburg et al. (2012) stated that human studies are often affected by confounding variables, which could be one reason why the results of the current study did not support this finding. One study suggested that obesity, measured by BMI, is associated with CVD risk (Stevens et al., 1998). In conjunction with the current study, Stevens et al. (1998) determined that the association of obesity and relative risk of death and CVD is stronger for younger individuals than older individuals.

In the current study, the average age of participants was 56.16 years. This is similar to the samples studied by DeNino et al. (2001) and Nieves et al. (2003). DeNino et al. studied a population of women ages 20-78, dividing the participants into four age

groups: 20-35 years, 40-50 years, 51-60 years, and 61-78 years. The youngest age group included nearly half of the participants, while the oldest age group consisted of less than one-quarter of the total sample. The most significant differences between these groups with respect to VAT, SCAT, and TC were found between the first two groups and the last group, suggesting that VAT, SCAT, and TC increase significantly after the age of 50 years (DeNino et al., 2001). After controlling for VAT, the researchers determined that the increases in TC were a direct result of the increases in VAT.

The results of the current do not support this theory and suggest that SCAT, but not VAT, is correlated with increased CVD risk as age increases. SCAT was strongly associated with TC in the younger age group ( $r = .610$ ,  $p = .000$ ), and was still significantly correlated with the Framingham CVD risk score in the older group ( $r = .410$ ,  $p = .022$ ). While VAT was not associated with any of the variables in the younger population, it did reach significance with TC in the older age group ( $r = -.420$ ,  $p = .019$ ) but not with the Framingham CVD risk score. The relationship between SCAT and the Framingham CVD risk score seemed to be more affected by age than the relationship between VAT and the Framingham CVD risk score. The results of the current study did not support previous research that VAT increases with age, warranting further research to determine if VAT is related to age or if another confounding factor could be associated with age that may affect CVD risk. The disagreement between the current study and past research may be due to the limitations in the sample that was chosen for the current study. Other study populations are larger in number and may have other variables that increase CVD risk other than abdominal fat. The current sample is similar to most other

research based on age, but the small size and inability to control for other variables may have weakened the results of the study.

### **Strengths and Limitations**

The results of this study show that SCAT and VAT are strongly and positively correlated with the total US measurements. Also, the individual components of the Framingham CVD risk score are strongly correlated with each other as well as with the score itself. This adds strength to the current study, indicating that the measurements were consistent among all participants. One limitation to this study is the measurement of VAT. It is possible that the measurement of VAT was not an accurate measure as the VAT that sits in and around each organ may not have been included. This may be one reason why there were lower amounts of VAT than expected in this study. Validation studies should be performed on the BodyMetrix ultrasonography machine to determine its ability to measure VAT.

This study was limited to a small, convenience sample from a homogenous population. Age was the only delimitation, as this study was targeting older individuals. It was initially thought that the older population would be at greater risk for CVD due to the effects aging has on the body. According to previous research, VAT levels are thought to increase as people age, thus increasing the risk for CVD (DeNino et al., 2001; Nieves et al., 2003). The results of the current study did not support this theory, suggesting that another factor, such as fitness, may be linked to CVD risk more so than age. The study population seems to exhibit traits that coincide with the fit and fat theory (Flegal et al., 2007). Because there were no significant associations between VAT and the Framingham CVD risk score, it can be concluded that the sample may have been

healthy, even if they were considered overweight. The current study agrees with Flegal et al. (2013) that evidence of CVD risk increases with levels of obesity. Future research should divide the obese group into more specific groups based on BMI to determine any relationships between the grades of obesity.

This study did not control for current diseases, medications, or family history. These variables may play a larger role in the development of CVD than was initially thought, although the sample size of the current study was too small to develop any theories with respect to these variables. Future research should focus on obtaining a larger, more diverse population to analyze other variables that may be confounding the results of the current study. It may be interesting to perform a follow-up study to determine how many people in the present study develop CVD 10 years from now, and how their levels of VAT and SCAT have changed over the years.

### **Summary**

The current study investigated the relationship between SCAT, VAT, the Framingham CVD risk score, and physical activity in older adults. The results indicate that SCAT may be associated with CVD risk, and that this correlation strengthens with age. Obese individuals as defined by BMI exhibit a more atherogenic CVD risk profile, independent of SCAT and VAT levels. As expected, physical activity levels are negatively associated with abdominal obesity and CVD risk. Although this study failed to determine an association of VAT and the Framingham CVD risk score, it will expand the literature regarding the role of SCAT and VAT in CVD risk identification and prevention.

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