CARDIOVASCULAR DISEASE RISK, FITNESS LEVEL AND DIETARY INTAKE IN OVERWEIGHT AND OBESE MALE COLLEGE ATHLETES VERSUS SEDENTARY TO MODERATELY ACTIVE COLLEGE STUDENTS WITH SIMILAR BODY MASS

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ABSTRACT

CARDIOVASCULAR DISEASE RISK, FITNESS LEVEL AND DIETARY INTAKE IN OVERWEIGHT AND OBESE MALE COLLEGE ATHLETES VERSUS SEDENTARY TO MODERATELY ACTIVE COLLEGE STUDENTS WITH SIMILAR BODY MASS

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Cardiovascular disease (CVD) is the leading cause of death in the United States. Cardiovascular disease risk factors in young adults predict future CVD morbidity and mortality. In college students, few studies have compared student athletes to less active non-athlete students matched based on age and body mass index (BMI) kg/m^2. The primary study objective was to compare CVD risk status between collegiate football student athletes (SA) and sedentary to moderately active student non-athletes (SMS) with similar BMI levels classified as overweight to stage 2 obesity ($\geq$25.0-<40 kg/m^2). Secondary objectives included evaluating differences in body composition, dietary intake, physical activity (PA) and fitness level. Forty college males (20 SA; 20 SMS) 20.3 $\pm$ 1.6 years of age with mean BMI 30.4±3.8 (25.6-39.5) kg/m^2; were analyzed. Risk factors were assessed individually and as a composite CVD risk score (cCVDs) including total blood cholesterol (TC), high-density lipoprotein (HDL), TC/HDL, low-density lipoprotein, triglycerides, fasting blood glucose, resting blood pressure and waist circumference. Covariates included body fat percentage, estimated maximal oxygen consumption, PA and dietary behavior. The cCVDs were not significantly different between SA and SMS (p=0.34). Both groups did not meet national heart health dietary targets except for total fat intake. In summary, >50% of participants had $\geq$1 risk factor though the prevalence of risk factors did not differ between SA and SMS despite SA having a higher daily average of minutes of moderate to vigorous PA.

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LIST OF ABBREVIATIONS

ATP III	Third Report of the NCEP Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III)
ACHA	American College Health Association
AHA	American Heart Association
BF%	Body fat percentage
BP	Blood pressure
BMI	Body mass index
cCVDs	Composite cardiovascular disease risk score
CHD	Coronary heart disease
CRP	C-reactive protein
DASH	Dietary Approaches to Stop Hypertension
FBG	Fasting blood glucose
FFQ	Food frequency questionnaire
HERL	Human Energy Research Lab
HR	Heart rate
HTN	Hypertension
Kcal	Kilocalories
KP	Kiloponds
LDL	Low-density lipoprotein
MAP	Mean arterial pressure
MbS	Metabolic Syndrome

MET	Metabolic equivalent
NFL	National Football League
NCAA	National Collegiate Athletic Association
NHANES	The National Health and Nutrition Examination Survey
PAR-Q	Physical activity readiness questionnaire
RHR	Resting heart rate
RMR	Resting metabolic rate
RPE	Rated perceived exertion
RPM	Repetitions per minute
SA	Student athletes
SCVN	Division of Sports and Cardiovascular Nutrition
SMS	Sedentary to moderately active non-athlete students
SNAPP	Spartan Nutrition and Performance Program
TC	Total Cholesterol
TC:HDL	Total cholesterol to high-density lipoprotein ratio
TDEE	Total daily energy expenditure
TG	Triglycerides
TLC	Therapeutic Lifestyle Changes
US	United States
$\dot{V}O2_{max}$	Maximal oxygen uptake
W	Watts
WC	Waist circumference

CHAPTER 1

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death in the United States (US) and had an estimated direct and indirect cost of $312.6 billion in 2009.[1,2] The large impact that CVD has on the US has led to the development of recommendations and strategies to promote CVD primary prevention.[3] Primary prevention of CVD is recognized as a lifelong process and should begin in early childhood since CVD risk factors in adolescence and young adults predict future CVD development.[4-7] For many, young adulthood is a transition period from adolescence to adulthood in which choices and behaviors are being made independently and for the first time, many of which affect disease risk and health status.[8] For example, physical in-activity is a modifiable CVD risk factor and when physical activity (PA) is incorporated into the lifestyle of an inactive person, multiple CVD risk factors can be positively affected.[9] Due to this, it is commonly believed that since athletes are physically active they are healthier than non-athletes. However, in selected sports and sports positions, athletes may have elevated CVD risks as compared to non-athletes.[10-13]

The majority of research evaluating the CVD risk factor status in college athletes in the US has been on male athletes and specifically football linemen.[10-12,14-17] These studies have reported the presence of CVD risk factors and undesirable health parameters like insulin resistance, increased waist circumference (WC), total cholesterol (TC), low-density lipoprotein cholesterol (LDL), triglycerides (TG), blood pressure (BP), and decreased high-density lipoprotein cholesterol (HDL). Other studies have also examined college athletes from a variety

of sports[13,18,19] and non-athlete college students and in general have shown SAs have more desirable health status than non-athletes.[20-23] Yet, few of these studies have compared SAs with non-athletes of similar size which can be done by matching the participants based on body mass index (BMI)(kg/m^2) of athletes with non-athletes to compare CVD disease risk. Also, many studies have not evaluated or controlled for dietary and PA behaviors or cardiorespiratory fitness measurement, which also influence their CVD risk.[24,25] The primary objective of this study was to compare CVD risk status between division I collegiate football SA and sedentary to moderately active student non-athletes (SMS) with similar BMI levels ranging from ≥25.0 kg/m^2 to <40 kg/m^2. Secondary objectives included evaluating differences in dietary intake and fitness level in these groups and determine how these factors influence CVD risk status. The CVD risk factors measured included TC, HDL, TC to HDL ratio, LDL, TG, fasting blood glucose (FBG), resting BP and WC and were assessed individually and as a composite CVD risk score (cCVDs). Covariates included body composition [body fat percentage (BF%)], estimated maximal oxygen consumption ($\dot{V}O2_{max}$), minutes of moderate and vigorous physical activity (MVPA), total daily energy expenditure (TDEE) and dietary behavior.

HYPOTHESES/AIMS

The overall hypothesis was that SA will have a lower level of CVD risk based on individual CVD risk factors and a composite cardiovascular disease risk score (cCVDs)* versus SMS. Aims of this study are:

Aim 1. To compare CVD risk between SA and SMS and national recommendations.

- Hypothesis 1a (H1a): SA will have lower cCVDs* versus SMS.

- H1b: A higher proportion of SA will meet national recommendations for blood lipids including TC, LDL, HDL and TG as compared to SMS.
- H1c: SA will have lower resting systolic and diastolic BP versus SMS.
- H1d: SA will have lower FBG versus SMS.
- H1e: SMS will have higher blood CRP versus SA. †

* Using a Z-score approach, a composite CVD risk score was calculated for each participant. This method was based on methodology used by Eisenmann et al. and has been validated for evaluating metabolic syndrome (MbS) risk in children.[26,27] The variables used in the cCVDs included TC:HDL ratio, TG, FBG, MAP, WC, and estimated $\dot{V}O2_{max}$ (an index of aerobic fitness). This is described in more detail in the Methods section (page 49).

† Prior to data collection, CRP was proposed to be included as a part of the cCVDs and to be analyzed as an individual risk factor. During the data collection process the Cholestech LDX organization informed the researchers that the CRP cassettes were faulty. For this reason, CRP was not included in the CVD risk analysis.

Aim 2. To compare CVD risk between BMI matched SA and SMS, based on body composition as measured by BF% and BMI level.

- H2a: SA will have a lower BF% versus SMS.#
- H2b: In SA and SMS groups separately, BF% will be directly correlated with cCVDs.
- H2c: In SA and SMS groups separately, BMI will be directly correlated with cCVDs.
- H2d: In SA and SMS groups separately, BF% will be a better predictor of cCVDs than BMI.

To help verify body composition (based on BF%), resting metabolic rate (RMR) was tested. It was hypothesized to be higher in SA compared to SMS due to low BF% and a higher lean body mass.

Aim 3. To compare estimated cardiorespiratory fitness and PA levels between SA and SMS and within each group and their relationship to CVD risk

- H3a: SA will have a higher cardiorespiratory fitness based on estimated $\dot{V}$ O2 $_{max}$ versus SMS.
- H3b: In SA and SMS groups separately, estimated $\dot{V}$ O2 $_{max}$ will be negatively correlated with cCVDs.
- H3c: In SA and SMS groups separately, PA level quantified by TDEE in kilocalories (Kcals)/day, will be inversely correlated with cCVDs.

Aim 4. To compare usual dietary intake between SA and SMS and within each group, relative to ATP III Therapeutic Lifestyle Changes (TLC)[28] guidelines and the American Heart Association (AHA) dietary guidelines and their relationship to CVD risk.

- H4a: In SA and SMS groups separately, total fat intake will be greater than 35% total Kcal from fat.
- H4b: SMS will have a higher intake of saturated and trans fat per 1000 Kcal versus SA and neither group will consume ≤7% Kcal intake from saturated fat and ≤1% Kcal intake from trans fat.
- H4c: SA will have a higher intake of simple sugars (total grams) versus SMS.
- H4d: SA will have higher intake of fiber per 1000 Kcal versus SMS and neither group will achieve guidelines of ≥12.5 grams fiber per 1000 Kcal of intake.

- H4e: SA will have a higher intake of fruits and vegetables (measured as total servings per day) versus SMS and neither group will achieve guidelines of ≥9 servings per day.
- H4f: SA will have a higher intake of potassium and sodium (total milligrams) versus SMS and neither group will achieve guidelines of ≥4700 mg of potassium and ≤1500 mg of sodium per day.
- H4g: In SA and SMS groups separately, intake of grams of fiber per 1000 Kcal of intake will be negatively correlated with cCVDs.

CHAPTER 2

LITERATURE REVIEW

Overview

The following literature review includes; 1) methods for assessing factors associated with CVD risk, with an emphasis on collegiate athletes; 2) prevalence of CVD risk factors, PA and dietary intake in young adults (particularly males), including collegiate athletes; 3) the strengths and weaknesses of dietary intake assessment methods, with emphasis on food frequency questionnaires (FFQ); 4) the strengths and weaknesses of cardiorespiratory fitness and PA behavior assessment methods, with emphasis on PA questionnaires; 5) the relationship and effect of dietary intake on CVD risk factor status in cross-sectional and prospective studies; 6) the relationship and effect of cardiorespiratory fitness and PA on CVD risk factor status in cross-sectional and prospective studies.

1. Assessment of Cardiovascular Disease Risk

There are several established CVD risk factors that are used to estimate overall CVD risk. **Table 1** list established CVD risk factors.[28-34] These risk factors are categorized into causal, predisposing and conditional/emerging risk factors. Causal risk factors have a direct causal relationship with atherosclerosis, predispose individuals to CVD and are often referred to as major risk factors. Predisposing risk factors contribute to and influence both causal and conditional/emerging risk factors and their role in the causal pathway is mediated by known and unknown causal risk factors. Conditional/emerging risk factors are associated with increased risk for CVD but their causal relationship with CVD has not been established.[34] These risk factors are the basis for evaluating CVD risk status and alteration of these risk factors are the objective of primary and secondary prevention of CVD.

Causal Risk Factors

Causal risk factors act independently from one another in CVD development and include cigarette smoking, high BP, high LDL cholesterol, low HDL cholesterol, diabetes, obesity, physical inactivity and older age.[3,34] In the 1990 report from the Surgeon General on the health benefits of smoking cessation, it was stated that smokers had a three to four times greater risk for heart attack then nonsmokers.[35] Once again, in 1997, the Surgeon General reported that one fifth of heart disease related deaths were due to cigarette smoking, making cigarette smoking the number one preventable cause of death in the US.[36] Decreasing cigarette smoking remains a major public health objective, with little progress being made in decreasing the percentage of adults who smoke.[37] In men, BP meeting criteria for hypertension (HTN) (≥140/90 mm Hg), has been associated with a relative risk of death from coronary heart disease (CHD) of 2.06, 95% confidence interval (CI) = 1.57-2.70[38], and BP in pre-HTN range (130-139/85-95 mm), has been associated with a risk-factor adjusted hazard ratio for developing CVD of 2.5, 95% CI = 1.6-4.1.[39] Type II diabetes is an independent risk factor for vascular disease and other CVD risk factors like HTN and dyslipidemia.[40] Controlling blood glucose levels in type 1 diabetics decreases the risk of CVD development[41] but controlling blood glucose in type 2 diabetics, may not decrease risk of CVD development.[42] Atherosclerotic plaque development is a risk factor for coronary events and, due to the severity of coronary atherosclerosis rising with age, age is used to estimate plaque burden.[34] Although, all the causal risk factors act independent of each other, elevated LDL levels are needed in order for other major risk factors to significantly contribute to

atherosclerosis.[43] For this reason, the Third Report of the NCEP Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III) distinguished LDL as the primary target of cholesterol lowering therapy.[28]

Overweight, obesity and related disease risks are often assessed through BMI classifications and measures of central adiposity, which are calculated through obtaining anthropometric measures like height, weight, WC and hip circumference. **Table 2** list classifications of overweight, obesity and disease risk per BMI and WC.[44] Body mass index is used to estimate the amount of body fat a person has by adjusting weight for height (kg/m^2). Body mass index was designed to assess populations but is often used at an individual level.[45] Body mass index is a practical measure for estimating body fat given that it is simple, inexpensive and safe to obtain. One drawback to BMI is that it assumes all individuals have the same relative amount of fat regardless of age, sex or ethnicity.[45] In other words, BMI can over or under-estimate body fatness in individuals who have varying amounts and densities of fat free mass.[45] Measures of central adiposity are often used in addition to BMI in order to identify the distribution of body fat and used as an independent predictor of disease risk and mortality.[44]

Body mass index has been observed to have a dose response relationship with CVD risk, increasing the relative risk of stroke by 6% with each increase in BMI unit in males.[46] While some researchers have found that measures of central adiposity alone do not have significantly greater predictive power of CVD risk over BMI[47-49], others have found that measures of central adiposity are stronger predictors of CVD risk than BMI.[50-52] Freiberg et al. evaluated 4,195

participants from the Framingham Offspring Study and found that WC did not predict CVD risk beyond what BMI did.[47] In a recent review of literature, Huxley et al. concluded, while both BMI and measures of central adiposity consistently show an association with CVD mortality, there is limited evidence to which measures of obesity are better.[48] In contrast, a 2008 meta-analysis showed waist-to-height ratio to be a statistically better discriminator of the CVD risk factors HTN, diabetes and dyslipidemia compared to BMI.[52] However, the difference in these obesity measures were determined to be clinically insignificant. In two large cohort studies of men and women, waist-to-height ratio again showed the strongest relationship to incidence of CVD compared to BMI, WC and waist to hip ratio. However, differences were once again small and likely clinically insignificant.[51] While there may be inconsistent findings as to whether BMI or measures of central adiposity are best for determining CVD risk, observed differences in these measures have been small and non-significant in the clinical setting. Due to the relative ease of obtaining height and weight to determine BMI compared to central adiposity measures, BMI may be an adequate measure of obesity and CVD risk in the overall population.[49]

Over the past 50 years the weight, height and BMI of collegiate athletes has increased, and thus it is important to understand how BMI correlates to body fat and disease risk in collegiate athletes.[53] Athletes have higher amounts of fat free mass compared to non-athletes,[54-56] so while BMI has increased, the assumption that there has also been an equal corresponding increase in body fat may not be true. Nevill et al. compared fat mass measured by skinfolds in BMI matched athletes and non-athletes and found that male strength and speed trained athletes had significantly lower skinfolds at 32% and 23% ($P < 0.01$), respectively.[55] Witt et al. reported

similar findings in Division III collegiate athletes, with 71% of males who met criteria for overweight per BMI had the sum of tricep and subscapular skinfolds at the 85th percentile or above.[56] This combination of skinfolds indicates a high muscle mass and lower upper arm fat percentage in athletes classified as overweight. Ode et al. analyzed BMI and fat mass even further by measuring the BF% in collegiate athletes and non-athletes and determining the corresponding BMI cut points for those overweight as determined by BF% at 20% or greater.[54] The cut points in BMI for the corresponding amount of 20% body fat in male athletes, linemen and male non-athletes were 27.9, 34.1 and 26.5 kg/m^2, respectively. In this analysis, BMI not only overestimated fat mass in male athletes, but also in male non-athletes. These findings parallel findings from Gallagher et al. who observed that the relationship between BMI and BF% was age dependent and that younger adults tended to have higher amounts of fat free mass compared to older adults.[45] In contrast, those classified as normal weight per BMI could possibly have a higher fat mass than BMI predicts and thus would be at increased disease risk. Romeron-Corral et al. analyzed data on individuals classified as normal weight per BMI with high body fat content from The National Health and Nutrition Examination Survey (NHANES) III and found that men in this category had a higher prevalence of dyslipidemia and HTN.[57] In the overall population, BMI may be a practical and appropriate measure used to estimate BF%, but when evaluating athletes, more direct measures of BF% or appropriately adjusted BMI cut points may be needed to properly determine overweight and obesity.[54]

Predisposing Risk Factors

The relationship that predisposing risk factors have in the causal pathway for CVD development is complex. When predisposing risk are present they intensify at least one of the causal risk factors, which act as a mediator in CVD development.[34] Predisposing risk factors include male gender, insulin resistance, genetics and family history of premature CHD.[34] Male gender is a predisposing risk factor for CVD, partially due to males having lower HDL levels compared to females.[58] In addition males may be at increased CVD risk compared to females when hyperinsulinemia is present.[59,60] Hyperinulinemia is a consequence of insulin resistance, a state in which cellular action is impaired by metabolic alterations.[34] Insulin resistance is negatively impacted by obesity and physical inactivity and is associated with several causal and conditional CVD risk factors.[34,61] Family history of premature CHD also puts individuals at increased risk for CVD and was observed by Parikh et al. to have an increased odds ratio of developing coronary artery calcification of 2.22, 95% CI = 1.22-4.01 ($P<0.001$).[62] Nasir et al. confirmed the role that family history of CHD has in CVD development in an ethnically diverse sample[63] and also found that the odds ratio for coronary artery calcification in men was higher when siblings versus parents had CHD, 2.3 95% CI = 1.7-3.1 versus 1.3 95% CI = 1.1-1.6.[64] Predisposing risk factors have complex roles in CVD development and although do not directly cause CVD, they do interact with both causal and conditional/emerging risk factors. As a result, controlling predisposing risk factors is very important for primary and secondary prevention of CVD.[34]

Conditional/Emerging Risk Factors

Conditional risk factors may be associated with atherosclerosis but are not considered causal risk factors because either the level of atherosclerosis caused is smaller compared to causal risk factors or their presence in the population may be infrequent and undetected by prospective studies.[34] Clinical and epidemiological studies have observed elevated homocysteine levels to increase the risk of atherosclerosis and thromboembolism.[33] Yet, homocysteine is not a causal risk factor for CVD because several prospective studies have failed to show an association.[33] The role that elevated TG have in CVD development has long been debated, but the AHA affirms in the 2011 scientific statement that TG are not directly atherogenic but can be used as an important biomarker of CVD risk.[65]

According to Ballantyne and Nambi, CRP is a marker of inflammation and is a strong predictor of future cardiovascular events.[66] This statement was found true in a cohort of middle aged men in which Koenig et al. calculated a risk ratio for CHD when CRP was increased to be 1.5, 95% CI = 1.14-1.97, after controlling for age and cigarette smoking.[67] In a study by Dhingra et al. CRP was more likely to be elevated in individuals with inflammatory conditions and increased CRP levels had a risk ratio for CVD development of 1.15, 95% CI = 1.04-1.28.[68] When the inflammatory conditions were controlled for in this population, the risk for CVD development became insignificant with a risk ratio of 1.20, 95% CI .96-1.50. In a 2010 meta-analysis, after controlling for risk factors the association that CRP had with CHD was considerably weakened again.[69] Danesh et al. concluded in their 2004 study that CRP is only a moderate predictor of CHD.[70] C-reactive protein may not be a strong independent predictor of

CVD but when used in conjunction with other CVD risk factors may aid in CVD risk assessment.

Interaction of Multiple Risk Factors and Predicting Risk

CVD risk factors are often present simultaneously and have a synergistic effect on CVD development. In an examination of Framingham Heart Study participants, men age 50 and older with two or more risk factors had a 68.9% lifetime risk of developing CVD compared to those with no risk factors (BP below 120/80 mm Hg, TC below 180 mg/dL, absence of diabetes, and nonsmoker) who had a 5.2% chance.[71] When looking at NHANES II data, Mensah et al. noted that men with none of the three risk factors measured (HTN, current smoker, and elevated TC ≥ 250 mg/dL) had a 51% lower risk of CHD mortality compared to those with at least one risk factor.[72] Having multiple CVD risk factors present at one time has been shown to have an additive affect in future CVD morbidity and mortality.

In order to quantify the absolute risk for CHD, multivariable equations have been developed that include several of the established CVD risk factors and predict the risk of CHD in the next ten years.[29] These equations classify individuals as high risk (20% or greater risk), intermediate risk (10%-20% risk) and low risk (less than 10% risk). The Dundee coronary risk-disk[73], equations from the British Regional Heart Study[74], the Prospective Cardiovascular Munster Heart Study (PROCAM)[75] and the SCORE project are all examples of CHD predictive equations.[76] The 1998 Framingham Risk Score has been used and validated in a wide range of populations and versions of this risk score have been modified for use by ATP III.[29] The Framingham Risk Score equations weight the risk factors for age, sex, systolic or diastolic BP,

TC, HDL, presence and absence of left ventricular hypertrophy, diabetes, and cigarette smoking.[29,77] In 2000, a newer version of the Framingham Risk Score was released, which added triglyceride levels, alcohol use and menopausal status into prediction equations and also predicted specific CVD end points like CHD, stroke, heart failure and peripheral artery disease.[29,77] According to the American College of Cardiology Foundation and the AHA Writing Committee, the Framingham Risk Score is the preferred method for assessing risk but other risk equations can be used if appropriate for the specific population.[29]

2. Prevalence of Cardiovascular Disease Risk Factors in Young Adults

The following will review literature focused on studies reporting the prevalence of CVD factors in young adults. In selected studies in addition to reporting the CVD prevalence, reporting of dietary and PA behaviors and interactions and relationships between risk factors will also be reported.

In 2008, the overall leading cause of death in the US was CVD, causing 616,828 deaths.[2] Of this total, only 1065 deaths were 15-24 year olds, making CVD the fifth leading cause of death in that age range.[2] Although, there was a smaller quantity of deaths caused by CVD in individuals ages 15-24 compared to 65 and over, longitudinal studies have found that the presence of CVD risk factors in children and young adults is predictive of increases in atherosclerosis,[4,5] CVD morbidity and CVD mortality[6,7] in later adulthood. In addition, post mortem study of 15-19 year olds by McGill et al. found that the presence of CVD risk factors were associated with increased presence of aortic and coronary artery fatty streaks.[78] The influence that CVD risk factors have on later development of CVD is alarming considering the

prevalence of pediatric obesity and other CVD risk factors are on the rise.[28,79] Not surprisingly, there has also been an increase in CVD risk factors in young adults with the 2008 prevalence of diabetes at 3.7%, overweight 32.2%, obesity 25.4% and prevalence among all adults of high LDL at 33.5%.[80,81] So while CVD is not the number one cause of death during early adulthood, CVD development and progression is associated with risk factors in early life and primary prevention including healthy PA and diet behaviors should begin in childhood .[82]

Healthy lifestyle habits like participation in regular PA and consumption of a heart healthy diet contribute to primary prevention of CVD through preventing CVD risk factor development or improving the status of those with risk factors.[29] Although, healthy lifestyle habits are central to CVD prevention, many Americans do not meet PA and dietary recommendations. **Table 3 and 4** in the appendix list PA national guidelines and select dietary recommendations. According to the National Health Interview Survey from 2010, only 25.7% of adults 18-44 years old met the 2008 PA Guidelines for Americans[9] for both aerobic and muscle strengthening activity, with 43.1% meeting neither guideline.[83] Lack of PA contributes to decreased energy expenditure and when combined with increased energy consumption contributes to gain of fat mass. From 1971-1974 to 1999-2000, the NHANES indicated a significant increase of energy intake in men and women.[84] From 1999-2000 through 2007-2008 there was no significant change in energy consumption in the overall population. However, males 20-39 years old increased their daily energy intake from 2854 Kcal to 2946 Kcal.[37] The overall trend of decreasing levels of PA and increased energy consumption likely contribute to the increasing prevalence of obesity in men from 27.5% in 1999-2000 to 35.5% in 2009-2010.[85]

In 1999-2004, men ages 20-39 had an average BF% of 26.1% [86] and in 2003-2006, an average WC of 98.2 cm, which is approaching the CVD risk factor cut point for WC greater than 102 cm.[87] Aside from overall energy intake, males 19-30 years old consumed 0.9 and 1.7 cup equivalents per day of fruits[88] and vegetables[89] respectively, in 2001-2004. These intakes do not meet the recommendations of the TLC diet[28] or the Dietary Approaches to Stop Hypertension (DASH) diet,[90] which were originally used to lower BP and have also been shown to lower LDL cholesterol levels. Consuming diets similar to the TLC diet and DASH diet may be warranted in young adults, considering one in ten males ages 20 to 34 were on cholesterol lowering medications or had TC levels greater than 240 mg/dl and 9.1% had HTN or were on BP lowering medications in 2007.[37] Many young adult males do not meet PA and dietary national guidelines and CVD risk factors are present in many of this age group.

Non-athlete College Student Physical Activity and Cardiovascular Disease Risk

For many students, college is a period of transition from adolescence to adulthood. For most, it is the first time they are consistently independently making lifestyle choices (dietary, PA and healthcare), which can affect disease risk and health status.[91] In 2008, the American College Health Association (ACHA) surveyed 80,121 students on 106 campuses in North America and found 54.5% of students participated in less than 20 minutes of vigorous intensity PA or 30 minutes of moderate intensity PA at least 3 times in the previous 7 days.[20] Similar PA participation rates were reported in a 2000-2001 study of college students by Spencer, who found that 36% of students reported performing aerobic PA 2 or fewer times a week.[22] Spencer noted

that 7.7% of participants had TC greater than 240mg/dl, 18.8% had HDL less than 40mg/dl and 10.5% had a BP of greater than 140/90 mm. From a study by Racette et al., 62% of college freshman at a US university participated in 3-5 days per week of aerobic PA with 30% participating in no PA.[23] When following this sample into their sophomore year, the amount participating in aerobic PA significantly decreased to 55% and was accompanied by a significant increase in weight of 1.8 ± 5.2 kg, although change in weight was not statistically associated with PA and diet. PA recommendations are commonly not met by college students and may contribute to CVD risk development.

Non-athlete College Student Dietary Intake and Cardiovascular Disease Risk

The 2008 report by the ACHA found that only 8.5% of the 80,121 college students surveyed in the US ate 5 or more servings of fruits and vegetables daily. [20] Racette et al. reported higher fruit and vegetable intake rates with 30% of the freshmen consuming 5 or more servings of fruits and vegetables daily.[23] Upon follow-up with the participants in their sophomore year, the only significant change in diet was a decrease of individuals who ate 3 or more serving of fried foods a week from 54% to 43%, $p = .004$. In the study by Spencer, 52% of students ate 2 or more servings of foods high in saturated fat daily and 6.6% ate 5 or more.[22] In Spencer's analysis, high fat food consumption was positively correlated with TC:HDL ratio, TC and BP. Morrell et al. analyzed 2,103 university students' three day food records and discovered males' diets had 10% of their calories coming from saturated fat.[21] In the study by Morrell et al., the prevalence of MbS was assessed using criteria from the AHA and National Heart, Lung and Blood institute (WC ≥ 102 cm in men, triglyceride ≥ 150 mg/dl, HDL < 40 mg/dl in men, BP ≥ 130/85 mm Hg and FBG ≥100mg/dl).[92] Metabolic syndrome is a cluster of the risk factors that

are interrelated and directly promote atherosclerosis.[92] In this sample, 9.9% of males met criteria for MbS, 24.7% had TC greater than 200 mg/dl, 62.1% had BP greater than 130/85 mm Hg and the average WC was 82.8 cm. The percentage of males overweight and obese was 46.9% which is significantly higher compared to the Racette et al. study at 18% of the 764 students and the 2008 ACHA survey at 39.1%. Differences seen in the rates of overweight and obesity could possibly be explained by the use of convenience samples in these studies. Dietary intake of college students is often low in fruits and vegetables and high in high fat foods, which may increase CVD risk factor development.

Collegiate Athlete Cardiovascular Disease Risk

In general, due to increase levels of overall PA in athletes, collegiate athletes are commonly thought to be healthier compared to non-athletes. However, in selected sports and sports positions, athletes may have elevated CVD risks as compared to non-athletes. For example, several studies have identified the presence of MbS in football players.[10-12,14,17] Buell et al. examined 70 football linemen sampled from National Collegiate Athletic Association (NCAA) division I, II and III teams, and found that 49% met criteria for MbS.[10] In a cross-sectional study, 90 players from a NCAA division I football team were examined and MbS was found in 9% of the total sample with the mean BF% of the sample equaling 17±7 %. However, the players who had a BF% of 25% or greater had a 42.1% prevalence of Mbs.[14] All the players who met criteria for MbS in this study were linemen. In 2010, Wilkerson et al. identified that 19% of 62 players on another division I team met the criteria for MbS and 49 of 62 had at least one component of MbS.[11] In another study, division I linemen, skilled position players and sedentary college students were compared by Dobrosielski et al., and 6 of 13 linemen, 4 of 13

sedentary students and 0 of 13 skilled position players met criteria for MbS.[12] Cardiovascular disease risk factors were still observed in skilled players including 3 players, who had low HDL levels. Based on these studies on collegiate football players, the prevalence of MbS is greatest in linemen at 42-66%, which appears to be related to body size, BF% and the distribution of fat; however, 54-79% of all players had one or more CVD risk factors increasing their risk for CVD.

As seen in football linemen, weight status and body composition play an important role on the CVD risk status in athletes. Mathews et al. reported that BMI, WC and BF% were all positively correlated but indicated that BMI often underestimates the amount of fat-free mass in athletes.[15] Based on an assessment of players on a NCAA division I football team BMI overestimated obesity in 50.6% of individuals compared to measurement of BF% from bioelectrical impedance. When incorporating BMI, WC and BF% only 16% met all three criteria for obesity.[15] In research by Haskins et al. comparing football linemen to non-athletes students of similar age and BMI, football players had a significantly lower BF% and BF% was a better predictor of CVD risk factors than BMI.[16] The football players in this study had lower BP and significantly more individuals meeting recommended levels for LDL.

Numerous studies have evaluated CVD risk factors in college athletes participating in a variety of sports. Munoz et al. evaluated 135 male and female athletes (sports included golf, tennis, baseball, softball, volleyball, soccer, cross country, track, synchronized swimming and basketball) from a NCAA division II school for CVD risk, and found 29% were overweight or obese, 22% had prehypertension or HTN and 24% of males had HDL less than 40mg/dl.[18] In this study, cardiorespiratory fitness was measured and males were determined to have an average $\dot{V}O2_{max}$ of 67 ml/kg/min. In 2010, Orri et al. conducted a cross-sectional study of 30 male and

female students and athletes (sports included baseball, basketball, track and field, volleyball, football, soccer and rugby).[13] As shown in the literature involving non-athletes, CRP was positively correlated with BF% and BMI, and CRP was predicted by FBG and WC. When comparing Nigerian male college students to medium distance Nigerian runners, Oyelola and Rufai reported the runners had significantly lower TC, LDL and TC:HDL.[19] The students' and runners' BMI and weight were not statistically different in this study.

Collegiate Athlete Dietary Intake and Cardiovascular Disease Risk

Dietary intake plays an important role in sports related performance[93] and health promotion,[94] yet many collegiate athletes do not meet dietary recommendations for performance or health.[95-98] Jonnalagadda et al. looked at 31 NCAA division I football freshman and found that 55% ate out 4.8 times a week with 55% of those times being at fast food establishments.[96] In this sample, 24% had TC levels greater than 200 mg/dl. Jonnalagadda et al. also reported that 26% of the sample did not know or disagreed that carbohydrates and fat are the main source of energy for muscles and 61% reported protein is the primary source of energy for muscles. Burke et al. reported in a review of literature that male athletes typically achieve carbohydrate recommendations.[98] In contrast, Cole et al. had 28 NCAA division 1 football players complete two separate three day diet records and reported that on average the athletes did not consume the recommended amount of carbohydrates.[95] This sample also had an average consumption of 2.9 fruits and vegetables daily, 10% of daily calories from saturated fat and did not consume the recommended amount of calories, which was not accompanied by weight loss. Hinton et al. analyzed the diet of NCAA division I male and female athletes (male sports including track,

basketball, golf swimming, diving, football, baseball and wrestling) using a FFQ and reported that male athletes consumed approximately 400 Kcal below recommended levels and only 10% and 19% consumed recommended amounts of carbohydrates and protein, respectively.[97] Hinton et al. also reported that 32% of total calories came from fat and 11% from saturated fat, and that total fiber intake was 18 grams per day. The literature highlights trends of less than desirable fruit and vegetable intake, saturated fat intake and total calorie intake, which could possibly be due to under reporting of dietary intake.[99] While there have been many studies examining CVD risk factors in collegiate athletes, few have incorporated into one study, athletes from several sports that include a wide range of BMIs and BF%, as well as assessment of dietary intake and cardiorespiratory fitness level compared to BMI matched non-athlete students. Additional research will shed light on how higher levels of PA from sport and interactions with diet influence CVD risk status.

3. Measuring Dietary Intake

There are several methods used for assessing dietary intake and all methods have strengths, limitations and some form of systematic error. The best method for assessment depends on specific factors, such as the individual being assessed, the aspect of the diet being assessed and the ease of obtaining the dietary information. In order to obtain practical and valid measures of dietary intake, the proper assessment method should be selected for the given situation. The assessment method also needs to be validated for the population in which it is being used. Validation consists of comparing the chosen assessment method to an appropriate reference which is considered to be the "gold standard".[100] Often, alternate dietary assessment methods like diet records, multiple 24 hour recalls or biological measures like nitrogen balance

are used as references for validation. However, comparing alternate dietary assessment methods may only portray agreement and not necessarily validity of measures and biomarkers can also be affected by factors other than diet.[100] Three methods that are often used for dietary assessment are diet recalls, diet records and FFQ.

Diet recalls are retrospective and usually consist of individuals describing the food and beverages consumed in the past 24 hours. Diet recalls are an inexpensive method that can quickly and easily be administrated, with minimal burden on the respondent.[101] Limitations of diet recalls include the reliance on the respondent's memory and often require a trained interviewer .[101] Dietary recalls provide valid data for the mean intake of groups within the previous 24 hours and has been used by NFCS and NHANES surveys. However, dietary recalls may be inadequate for estimating usual intake.[102] Using 24 hour recalls, men have been observed to over report protein intake by 12-19% when compared to urinary nitrogen levels.[103] In contrast, other studies have observed underreporting of dietary intake with 24 hour recalls,[104,105] with underreporting being more likely in those who were overweight compared to those who were at lower BMI levels.[99,105] Dietary recalls may be an assessment method best suited for clinical settings.[101] Dietary recall's ability to estimate usual dietary intake increases when several consecutive days or several dispersed days throughout a year are obtained, but this may not be a practical method for assessing usual dietary intake.[102]

Diet records are prospective and consists of individuals recording food and beverages as they are consumed, usually over a period of 1-7 days.[101] Diet records have an increased

accuracy due to minimizing errors in memory, and can also incorporate weighing foods to determine precise quantities.[102] The increase in accuracy is accompanied by increased respondent burden and intake may be affected by the change in routine associated with food consumption.[101] Food records also provide valid data on groups, and the validity associated with individuals is dependent on the length of the food record.[102] In theory, as the length of the diet record increases, the ability of estimating usual dietary intake increases. However, some studies show that after 2-3 days, the accuracy and completeness of multi-day records decreases due to respondent burden.[102]

Food frequency questionnaires require individuals to describe the average frequency that they consume specific food items from a pre-established list over a given time span and are retrospective.[101] In the past, FFQs were used to establish the usual intake of single nutrients in the diet. In addition, FFQs can also be used to determine usual total nutrient intake.[102] In order to do this, a food list representing all major sources of nutrients needs to be established for a given population.[102] While this method can be self-administered and relatively inexpensive, the respondent burden can increase as the length of the questionnaire increases to fit the given population.[101] Food frequency questionnaires can be valid measures of usual intake and different FFQs, for specific populations, have been validated by several studies.[106-108] In a review of literature, Cade et al. reported that 54% of FFQs reviewed were modified from previous versions of established FFQs, with 25% of those adapted from the questionnaire developed by Block et al.[100,109] Development of new FFQs can be costly and time consuming, so while modifying pre-

existing questionnaires is a viable option for assessing usual intake, care needs to be taken in establishing the purpose and the population in which a FFQ is valid.[100]

In the past, dietary assessment performed on athletes provided data that athletes were consuming relatively small amounts of energy, while expending high amounts of energy and yet were still able to maintain body mass.[99] These data led to the development of a theory that athletes were more efficient at using energy than non-athletes. This concept was later dismissed when it was discovered that athletes tend to underreport energy consumption.[99] It has also been found that training has an effect on underreporting of energy intake. As training levels increase, athletes report the same amount of energy intake without an expected drop in body mass.[99] Underreporting can be due to intentional and non-intentional underreporting of energy intake or intentional and non-intentional decrease in actual energy intake.[101] Over-estimation of energy consumption can also be seen in athletes who have low intakes of energy.[101] Once again, the type of dietary assessment method chosen for assessing the dietary intakes of athletes, is dependent on the purpose, practicality and the specific population being assessed.[101]

4. Measuring Cardiorespiratory Fitness and Physical Activity Behavior

Measuring Cardiorespiratory Fitness

Physical fitness has been defined as the ability to carry out daily tasks with adequate energy and without undue fatigue in order to enjoy leisure activities.[110] Fitness can be measured as health or skilled related attributes, including cardiorespiratory fitness, muscular strength and endurance, body composition and flexibility.[110] Measurement of cardiorespiratory fitness is

indicated for use when exercise tolerance, undiagnosed exercise intolerance, patients with CVD and respiratory disease and other indications require evaluation.[111] Evaluating cardiorespiratory fitness is an objective method of determining functional capacity, factors limiting exercise, underlying pathophysiological mechanisms and early detection of disease.[111] Two modes of cardiorespiratory exercise testing that are commonly used are treadmill and cycle ergometer. Cycle ergometers are commonly used in clinical settings and the main advantage for use is the rate of external work being performed is easily quantified.[111] With treadmill testing, external work rate is more difficult to quantify due to the variation in speed and grade of the treadmill, along with the weight of the subject.[111] Gas exchange measurement of oxygen uptake and carbon dioxide output are often measured, and when maximal exercise testing protocols are used, $\dot{V}O2_{max}$ can be obtained. Measuring an individual's $\dot{V}O2_{max}$ is the gold standard for assessing cardiorespiratory fitness.[111]

There are also several submaximal tests that use the linear relationship between heart rate and oxygen consumption to predict $\dot{V}O2_{max}$.[112] The benefits of submaximal fitness testing versus directly measuring an individual's $\dot{V}O2_{max}$ include decreased time of testing, less labor intensive protocols, less equipment needed, and a lower participant burden.[112] The Astrand Ryhming cycle ergometer protocol is a widely used submaximal cycle test that uses a nomogram to predict $\dot{V}O2_{max}$.[113] This submaximal cycle protocol takes six minutes to administer, requires limited equipment and in studies on different populations has shown to have a correlation with measured $\dot{V}O2_{max}$ values between r=.71 and r=.98.[112] There are several tests that can be used

to predict $\dot{V}O2_{max}$ and test selection should be determined by the population being tested, the equipment, time and number of participants and also the experience of the tester.

Measuring Physical Activity Behavior

Regular PA contributes to increased physical fitness.[114] PA has been defined as bodily movement produced by skeletal muscle contraction that increases energy expenditure above a resting level.[9] There is a wide range of activities that contribute to activity related energy expenditure, including occupational, leisure time, sports, transportation and household activities.[114] When deciding what type of PA measurement method should be utilized, factors like research goals, sample size, budget, participant burden, environment, accuracy and precision need to be considered.[115] There are five general classifications of PA assessment methods: calorimetry, behavioral observation, physiological markers, motion sensors and questionnaires. Each assessment method has individual strengths and weaknesses.[116]

Calorimetry and direct observation are often used as validation criteria for other PA assessment methods.[114] Direct calorimetry directly measures the heat produced from energy expenditure, but due to complexity and mobility of equipment, it is not a usable measure of free living energy expenditure. Doubly labeled water is a method in which a standardized amount of stable isotopes ^{2}H and ^{18}O are ingested, and the difference in excretion of the isotopes is calculated to estimate CO^{2} production and then energy expenditure. This method is the "gold standard" for measuring free living energy expenditure.[116] However, doubly labeled water is unable to distinguish between intensities of PA, and the isotopes are very expensive.[114] Indirect

calorimetry measures oxygen consumption and/or carbon dioxide production in order to calculate energy expenditure and is a valid measure, but it can be difficult and expensive to use to measure free living energy expenditure outside of a laboratory setting. Direct observation of PA behaviors by a trained observers has long been used to record PA, although this method is very time consuming, incorporates subjectivity of the observer and the observer's presence in the environment could alter PA behavior.[114]

Physiological markers and motion sensors are objective measures of PA and can be applied to individuals over an extended period of time while recording large amounts of data.[114] Heart rate (HR) is a physiological marker which has a linear relationship with oxygen consumption and can be used to predict energy expenditure.[114] HR can be used to record duration, frequency and intensity of activity, but can also be skewed by other variables like caffeine intake, stress, smoking and body position, which all can increase HR.[114] Pedometers and accelerometer are examples of motion sensors which register body movement and are light weight and relatively inexpensive. Pedometers measure movements on a vertical plane and are quantified as steps over a given period of time. Pedometers thus can be used to estimate walking or running distance when the individuals stride length is known but is unable to measure intensity, cycling, swimming, upper body movements, load carrying or grade of terrain.[114] Accelerometers can register movement on one plane like pedometers or on several planes increasing the sensitivity of PA measurement. Accelerometers have similar limitations as pedometers but are able to indicate intensity level.[114] Accelerometers have also been combined

with HR monitors to measure total energy expenditure and show potential for future improvement in PA assessment.[117]

Subjective measures of PA assessment include interviews, activity diaries and activity questionaires.[116] Activity questionnaires are widely used due to their low cost and ability to survey large groups quickly.[116] Questionnaires are valid when used to make gross classifications for assessing PA level in populations, but are less suited for PA assessment at an individual level due to reliance on respondent's memory and interpretation of questions.[114] There have been many PA questionnaires that have been developed and tested for validity and reliability in given populations.[118-120] Some studies reutilize or modify questionnaires for alternate populations, which requires revalidation.[121-123] Van Poppel et al. preformed a systematic review of 85 versions of self-administered PA questionnaires and concluded the quality of assessment measures for the questionnaires was poor with overall content validity lacking and reliability being tested in only half the questionnaires.[124] Although questionnaires have limitations to their ability to obtain PA levels with questionable validity, questionnaires used in studies that classify gross levels of PA may be a quick and cost effective measure.

5. <u>Dietary Intake and Cardiovascular Disease Risk</u>

The following will review literature focused on the relationship and the effect of dietary intake on the development of CVD and CVD risk factors in cross-sectional and prospective studies. Dietary intake affects several of the modifiable CVD risk factors including BP, lipid levels, obesity, WC, FBG, CRP and homocysteine.[24,28,90,125] Healthy diet behaviors are also associated with decreased CVD mortality, and when combined with other healthy lifestyle

behaviors have a greater overall protective influence on CVD mortality.[126] Common dietary recommendations for prevention and treatment of CVD include decreasing total fat intake (specifically saturated fat, trans-fat and cholesterol), increasing fiber intake, decreasing sodium intake and decreasing simple sugar intake.

Saturated fat, trans-fat and dietary cholesterol intake have a dose-response relationship with TC and LDL, with saturated fat and trans-fat having a greater effect than dietary cholesterol.[24] HDL is increased by saturated fat intake but is decreased by trans-fat intake.[24] In a meta-analysis of 27 studies, Hooper et al. found that decreases in dietary intake of fat showed a 16% decrease (95% CI = 0.72-0.99) in cardiovascular events and an even stronger protective influence of 24% (95% CI = 0.65-0.90) after 2 years of follow up.[127] Not all dietary fats have a negative influence on CVD risk factors, with several studies showing monounsaturated fats and polyunsaturated fats having positive effects on TC and LDL.[24,28,94]

Total fiber intake of greater than 25g/day is associated with a decreased risk of CVD.[24] Soluble fiber has a been shown to have a greater effect on lowering LDL levels than insoluble fiber but still total high fiber intake is inversely related to CHD.[24] In a study by Ludwig et al., fiber intake predicted CVD risk factors stronger than total or saturated fat intake, showing the protective role fiber has in CVD risk factor development.[128] When examining 1990-2000 NHANES data, King et al., found that those in the highest quartile of fiber intake had a 42% (95% CI = 0.38-0.88) reduction in CRP levels compared to those in the lowest quartile.[125]

Reducing dietary sodium is a well-established dietary modification used to decrease BP.[90] The DASH diet is often associated as a low sodium diet that lowers BP. However, when

following the guidelines of the DASH diet, increased consumption of potassium and weight loss also contribute to a decrease in BP.[90] Yang et al. found that a higher sodium to potassium ratio put individuals at an increase mortality risk for CVD and ischemic heart disease.[129] Sacks et al. looked at varying levels of sodium intake in the DASH diet and found that as the intake of sodium decreased a greater degree of decrease was seen in BP.[130] Along with decreasing sodium intake the DASH diet incorporates several dietary recommendations that effectively lower BP.[90]

Excessive dietary sugar contributes to increased calorie consumption and possibly weight gain but is also thought to possibly affect multiple CVD risk factors.[131] Studies have noted that added sugar may increase BP,[131,132] lower HDL,[133] increase TG[134] and possibly promote inflammation.[131] While many emerging studies have highlighted the effects of sugar on CVD risk the evidence has not been conclusive and more research needs to be done.[131] Still with the evidence that is present, the AHA "Diet and Lifestyle Recommendations Revision 2006", recommended to decrease intake of beverages with added sugar and Johnson et al. recommends the upper limit of added sugar in men's diet to be nine teaspoons.[131]

6. <u>Physical Activity, Cardiorespiratory Fitness and Cardiovascular Disease Risk</u>

The following will review literature focused on the relationship and the effect of PA behavior and cardiorespiratory fitness on the development of CVD and CVD risk factors in cross-sectional and prospective studies. PA has an inverse relationship with all-cause mortality[135,136] and CVD mortality.[136,137] Popovic et al. reported that in a cross-sectional study of 150, 20-40 year old endurance trained, recreational sport and sedentary individuals, lipid

profile, inflammation markers and body fat levels were significantly better in the endurance trained and recreational sport groups.[138] McGavock et al. reported an increased risk of insulin resistance in sedentary individuals compared to endurance trained individuals,[139] while other studies have documented the inverse relationship of PA with BP.[140,141] Kokkinos et al. reported a dose-response relationship between miles ran per week and HDL levels with HDL levels rising .308 mg/dl per mile ran and most changes associated with running 7-14 miles per week.[142] Marrugat et al. surveyed 537 men on their PA behaviors and found that with every 100 Kcal increase in the average daily energy expenditure at an intensity of 7 Kcal per minute over the previous year, an associated 2.09 mg/dl increase in HDL was observed.[143] When the intensity of activity was increased to 9 Kcal per minute TC, LDL and TG were observed to decrease.

Physical fitness also has an inverse relationship with all-cause mortality[144,145] and CVD mortality.[25,145,146] In a study of 25,714 men, Wei et al. reported that the relative risk for overweight men to develop CVD was similar for those who had low cardiorespiratory fitness and those with other major CVD risk factors like diabetes.[146] In a review of literature by Fogelholm, high cardiorespiratory fitness was reported to attenuate the increased risk of CVD development in individuals who were obese, and indicating that fit obese individuals had lower risk of CVD development compared to normal weight individuals who had low cardiorespiratory fitness.[147] Increased cardiorespiratory fitness has also been reported to protect against the development of CVD risk factors including HTN, hypercholesterolemia and MbS.[141,148,149]

Implications of the Literature Review

This review of literature provided a summary of the prevalence of CVD risk factors in young adults and the relationship that dietary intake, cardiorespiratory fitness and PA behavior have with CVD risk factors. Additionally, this review summarized techniques and statistical procedures for assessment of individual CVD risk factors, and methods for estimating overall CVD risk. The majority of these methods were developed for estimating overall CVD risk in older populations, leaving a gap in the literature regarding estimating overall CVD risk in young adult populations.[29,73] For this reason, a continuous composite CVD risk score was used that was based on methods used for developing a continuous MbS score for pediatric research.[26] In addition, few studies have measured CVD risk in athletes from sports that include a wide range of BMIs and BF% and compared BMI matched non-athlete students while controlling for dietary intake and estimated cardiorespiratory fitness. The primary objective of this study was to compare CVD risk status between division I collegiate football SA and SMS with similar BMI levels ranging from ≥25.0 kg/m^2 to <40 kg/m^2. Secondary objectives included evaluating differences in dietary intake and fitness level in these groups and determine how these factors influence CVD risk status. The CVD risk factors measured included TC, HDL, TC to HDL ratio, LDL, TG, FBG, resting BP and WC and were assessed individually and as a cCVDs. Covariates included body composition (BF%), estimated $\dot{V}O2_{max}$, minutes of moderate and MVPA, TDEE and dietary behavior.

CHAPTER 3

MANUSCRIPT

1. INTRODUCTION

Cardiovascular disease is the leading cause of death in the US and had an estimated direct and indirect cost of $312.6 billion in 2009.[1,2] The large impact that CVD has on the US has led to the development of recommendations and strategies to promote CVD primary prevention.[3] Primary prevention of CVD is recognized as a lifelong process and should begin in early childhood since CVD risk factors in adolescence and young adults predict future CVD development.[4-7] For many, young adulthood is a transition period from adolescence to adulthood in which choices and behaviors are being made independently and for the first time, many of which affect disease risk and health status.[8] Physical inactivity is a modifiable dependent CVD risk factor that has a multi-factorial influence on various other risk factors including hypertension, dyslipidemia, thrombotic factors and body composition. When PA is incorporated into the lifestyle of an inactive person, multiple CVD risk factors and overall CVD risk status tends to improve.[9]

It is commonly believed that since athletes are physically active they are healthier than non-athletes. However, in selected sports and sports positions, athletes may have elevated CVD risks as compared to non-athletes.[10-13] Most research evaluating college athletes' CVD risk status in the US has been on male athletes and football linemen specifically.[10-12,14-17] These studies have reported the presence of CVD risk factors and undesirable health parameters like insulin resistance, increased WC, TC, LDL,TG, BP, and HDL. Other studies have also examined

other college athletes from a variety of sports[13,18,19] as well as non-athlete college students and in general have shown SAs have more desirable health status than non-athletes.[20-23] Yet, few studies have compared SAs with non-athletes of similar size. Also, many studies have not evaluated or controlled for dietary and PA behaviors or cardiorespiratory fitness, which also influences CVD risk.[24,25] The primary objective of this study was to compare CVD risk status between division I collegiate football SA and SMS with similar BMI levels ranging from 25.0-40 kg/m^2. Secondary objectives included evaluating differences in dietary intake and fitness level in these groups and determining how these factors influence CVD risk status. The measured CVD risk factors included TC, HDL, TC to HDL ratio, LDL, TG, FBG, resting BP and WC and were assessed individually and as a cCVDs. Covariates included body composition (BF%), $\dot{V}O2_{max}$, minutes of MVPA, TDEE and dietary behavior.

The overall hypothesis was that SA, compared to SMS, will have lower CVD risk based on individual CVD risk factors and a composite cCVDs. Aims of this study are:

Aim 1. To compare CVD risk between SA and SMS and national recommendations.

- Hypothesis 1a (H1a): SA will have lower cCVDs versus SMS.
- H1b: A higher proportion of SA will meet national recommendations for blood lipids including TC, LDL, HDL and TG as compared to SMS.
- H1c: SA will have lower resting systolic and diastolic BP versus SMS.
- H1d: SA will have lower FBG versus SMS.
- H1e: SMS will have higher blood CRP versus SA.

Aim 2. To compare CVD risk between BMI matched SA and SMS, based on body composition as measured by BF% and BMI level.

- H2a: SA will have a lower BF% versus SMS.
- H2b: In SA and SMS groups separately, BF% will be directly correlated with cCVDs.
- H2c: In SA and SMS groups separately, BMI will be directly correlated with cCVDs.
- H2d: In SA and SMS groups separately, BF% will be a better predictor of cCVDs than BMI.

Aim 3. To compare estimated cardiorespiratory fitness and PA levels between SA and SMS and within each group and their relationship to CVD risk

- H3a: SA will have a higher cardiorespiratory fitness based on estimated $\dot{V}\ \mathrm{O2}_{\ \mathrm{max}}$ versus SMS.
- H3b: In SA and SMS groups separately, estimated $\dot{V}\ \mathrm{O2}_{\ \mathrm{max}}$ will be negatively correlated with cCVDs.
- H3c: In SA and SMS groups separately, PA level quantified by TDEE in kilocalories (Kcals)/day, will be inversely correlated with cCVDs.

Aim 4. To compare usual dietary intake between SA and SMS and within each group, relative to ATP III Therapeutic Lifestyle Changes (TLC)[28] guidelines and the American Heart Association (AHA) dietary guidelines and their relationship to CVD risk.

- H4a: In SA and SMS groups separately, total fat intake will be greater than 35% total Kcal from fat.
- H4b: SMS will have a higher intake of saturated and trans fat per 1000 Kcal versus SA and neither group will consume ≤7% Kcal intake from saturated fat and ≤1% Kcal intake from trans fat.
- H4c: SA will have a higher intake of simple sugars (total grams) versus SMS.

- H4d: SA will have higher intake of fiber per 1000 Kcal versus SMS and neither group will achieve guidelines of ≥12.5 grams fiber per 1000 Kcal of intake.
- H4e: SA will have a higher intake of fruits and vegetables (measured as total servings per day) versus SMS and neither group will achieve guidelines of ≥9 servings per day.

2. METHODS

Study Design and Sample

This study was a cross-sectional two group comparison of a convenience sample of Michigan State University (MSU) college males ages 18-24 classified as overweight to stage 2 obesity based on BMI 25-<40 kg/m^2. The groups included MSU varsity male SA recruited from the football team and a group of SMS matched for BMI. The SA were recruited first followed by SMS. Student athletes were recruited and measured by the research staff during their July 2012 preseason training through their 2012 football season ending in December. Ninety-one SA were eligible to participate in the study (based on BMI). After screening and explanation of the study, 28 consented to participate. The SMS were recruited through flyers posted on the MSU campus, sent through email and passed out after announcements made in undergraduate classes. Recruitment and measurement for SMS began in September 2012 and ended March 2013. Sixty-one SMS contacted research staff about participation in the study but after screening and explanation of the study, 20 consented to participation. Known reasons for not participating included time commitment, disinterest, not meeting all inclusion criteria and BMI mismatch.

Inclusion criteria for the SMS participants included, if in the previous two months the individual had participated in <300 minutes of moderate-intensity PA [3.0-5.9 metabolic equivalents (MET)] a week, or <150 minutes of vigorous-intensity PA (≥6 MET) a week, or when moderate- and vigorous intensity PA was combined <1000 MET minutes per week.[9]

Anything higher than these activity levels would classify an individual as having a high PA level as defined by the 2008 PA Guidelines for Americans. Minimum levels of PA recommended by the guidelines are >150 minutes a week of moderate intensity PA or >75 minutes of vigorous intensity PA. [9] Eligibility included PA history, height, weight, and BMI and was ascertained through telephone screening and reviewing the estimated MET minutes per week calculated form the Block Adult Energy Expenditure Survey.[109,150] Exclusion criteria for both study groups included those who were taking medications or supplements that significantly influenced the dependent measures (cholesterol, BP, etc.). Individuals who had type 1diabetes or any disease process that affected study outcomes were excluded. Also, those who reported smoking ≥1 cigarette daily were excluded from participation. All potential participants who did not pass pre-exercise screening criteria using a PA readiness questionnaire (PAR-Q)[151], were excluded. Participants who were injured and unable to participate in the sub-maximal fitness test were excluded.

Those who met all inclusion criteria and participated, received a $15 Subway gift card and a summary of their assessment results (CVD risk factor status, nutrition assessment, submaximal exercise testing with basic recommendations to sustain or improve their health status). This study was approved by the MSU Institutional Review Board and all participants completed written informed consent before study participation. The methodology paralleled a similar study conducted on female SA and SMS in 2010-11.[152]

Measurement Protocol

All measurements were conducted in the Nutrition and Exercise Lab in the MSU Department of Radiology or in a private room within the MSU intercollegiate athletic facilities.

Participants received instructions to fast for 8-12 hours, abstain from exercise for >12 hours and complete study measurement within an hour of waking, with the exact measurement time dependent on the time the participant usually awoke. After reviewing and signing consent and completing the PAR-Q, participants relaxed in a supine position for ten minutes. They then proceeded with RMR measurement, resting heart rate (RHR), resting BP, finger prick blood samples to assess their lipid panel and FBG. Additionally, anthropometric measurement included, standing height, body weight, BF% and WC. After physiological measures were obtained, participants were asked to complete FFQ and PA questionnaires. A majority of participants completed all assessments in one day however many SA were measured over multiple days due to the complexity of their schedules. Additionally, two SA reported drinking alcohol the night before their study measurement and were rescheduled.

Resting Metabolic Rate and Resting Heart Rate

Participants' RMR was measured with indirect calorimetry using the Korr ReeVue (Korr Medical Technologies, Salt Lake City, UT), using procedures described by the manufacturer. The Korr ReeVue measures oxygen uptake to estimate energy expenditure based on ml of O^2/min/kg body weight. The Korr ReeVue has been tested for the validity and reliability compared to the Deltatrac Metabolic Monitor, an established valid and reliable measure of RMR.[153] The Korr ReeVue's within-subject coefficient of variation was 12.2% compared to the Deltatrac Metabolic Monitor. The Korr ReeVue test time takes 10 minutes, the first 5 minutes of data are not used, with the estimated energy expenditure based on the average of data collected the second 5 minutes. During the RMR test, RHR was recorded to determine if the participant is in a steady resting state using a Nonin Onyx II 9550 digital fingertip pulse oximeter (Nonin

Medical, Inc., Plymouth, MN). If RHR varied by >10% during the final 5 minutes that the RMR values are generated from, the participant was asked to be retested.[154]

Resting Blood Pressure

A manual resting systolic and diastolic BP were taken following procedures described by The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure.[155] A stethoscope and standard BP aneroid with an appropriately sized inflatable cuff were used on the participants' non-dominant arm (Professional Aneroid Sphygmomanometer, AllHeart, Louisiana, MO). Two measures were taken with one minute between each measure. In order for measures to be accepted, measures had to be within 5 mm HG of each other. The average of the measures were used for analysis. Mean arterial pressure was calculated for analysis by using the equation:

MAP=(Resting Systolic BP-Diastolic BP)/3+Diastolic BP

Lipid Panel, Fasting Blood Glucose and C-reactive Protein

Two fasting blood samples were collected by finger prick in heparinized capillary tubes. One blood sample was used to analyze TC, LDL, HDL, TG, TC/HDL ratio and FBG by the portable Cholestech LDX System according to manufacturer guidelines (Cholestech LDX, Hayward, CA).[156] The blood samples were dispensed into a disposable cassette, placed in Cholestech LDX, and reflectance photometry was used to measure concentration of blood substances, which took approximately five minutes. Results were shown on a Cholestech LDX System display and also printed on paper. The other blood sample was analyzed for CRP using the same protocol, however during data collection the Cholestech LDX organization informed the researchers that the CRP cassettes used were faulty. For this reason, CRP was not included in

the CVD risk analysis. The Cholestech LDX has been assessed for validity and falls within National Cholesterol Education Program analytical goals and reliability has been tested in comparison to the CardioCheck PA.[157,158]

Standing Height, Body Weight, Body Fat Percentage and Waist Circumference

The protocol used in the current study has been used in another study in which only small measurement errors were reported.[159] Participant's standing height were measured using a portable stadiometer (Shorr Productions, Olney, Maryland, USA). Participants were asked to stand with shoulders, back and heels touching the wall with head in the Frankfort plane. Standing height was measured until two measured are ≤0.3 cm from each other and then the average of the two measures were used in the statistical analysis. Body weight was measured using a calibrated electronic scale, measured to the nearest 0.1 kilogram (BC-534 Tanita co., Tokyo, Japan). Body fat percentage was assessed using a foot-to-foot BIA measurement device and then lean body mass % was derived (BC-534 Tanita co., Tokyo, Japan). This device was found to have correlations ranging from 0.74-0.81 with 2-site skinfold and Omron BIA device in males.[160] A Gulick anthropometric tape was used to assess WC (Gulick co., Tokyo, Japan). Waist circumference was measured to the nearest 0.1cm where the uppermost lateral border of the iliac crest and the midaxillary line intersect, according to National Heart, Lung and Blood Institute protocol.[44] The WC measurement was repeated until two measurements were within 1.0 cm and the average of the two measurements were used for analysis.

Composite Cardiovascular Disease Risk Score

The primary CVD risk factor status analysis was based on a cCVDs. The following risk factors were included in the CVD risk score: TC:HDL ratio, TG, FBG, MAP, WC, estimated

$\dot{V}O2_{max}$ and BMI. Using a Z-score approach, the cCVDs was calculated for each participant. Risk factors were regressed onto age to account for age related differences. The cCVDs was created from the summation of the standardized residual Z-scores for the individual risk factors. HDL and $\dot{V}O2_{max}$ are inversely related to CVD risk and were multiplied by -1 before summing all of the risk factors. A higher score indicated a less favorable CVD risk status. This method was based on methodology used by Eisenmann et al. and has been validated for evaluating MbS risk in children.[26,27]

Food Frequency Questionnaires and Physical Activity Questionnaires

Dietary intake was assessed by Nutritionquest's 2005 Gladys Block 110 item electronic format FFQ.[109] The 2005 version of the Gladys Block FFQ has been updated from previous versions that found correlations of 0.5-0.6 with 4-day food records and a correlation of 0.8 between two tests administered 3 months apart.[161,162] The electronic 2005 version of the Gladys Block FFQ is self-administered on a computer and is used to estimate the usual intake of a wide range of nutrients and food groups. Pictures of portion sizes were given to participants to increase accuracy of reporting. The electronic version prevented participants from skipping questions and when completed was electronically submitted for nutrient analysis. Football players have higher amounts of lean body mass compared to the general population and in theory would need larger portions of food to maintain body weight. Accordingly, all participants were asked if they would eat a larger portion size than was given by the FFQ.[12] If the participant chose a larger serving size than the FFQ provided, then the researcher altered the serving size and frequency of consumption to reflect the actual intake of the given food. This technique was

developed from the guidance by the FFQ supplier, Nutritionquest senior staff member Jean C. Norris MS, RD, DrPH. Data were stored and accessed online in a user account set up for this study, which was password protected. Participant's analyzed nutrition data were flagged if their estimated average calorie intake was greater than or less than 30 percent of their daily calorie expenditure estimated from the Nutritionquest's Block Adult Energy Expenditure survey (electronic format) and calculated sports related activities.[150,163] Two registered dietitians reviewed the nutrition data and determined whether the flagged submissions were valid, taking into account if participants reported trying to lose or gain weight. After analysis, dietary components were assessed for achievement of national guidelines as listed in **Table 4** the appendix.

The evaluation of daily PA was assessed by Nutritionquest's Block Adult Energy Expenditure electronic-format survey which was developed by determining the 26 most relevant daily-life and leisure-time activities and calculating energy expenditure from MET levels.[150,163] Based on the reported activities, the survey estimates average energy expenditure, amount of PA in minutes per day, and average MET minutes by activity type. This tool has been assessed for validity by its ability to predict body fat in men ($r = 0.73$).[164] In order to increase accuracy, SA were told not to report required sports-related training, which was calculated for each participating athlete based on their practices and workouts using the MET levels from the Compendium of PA and previous research on energy expenditure in collegiate football players.[150,165] The estimated activity from the energy expenditure survey and calculated activity were added together. The questionnaire also used to confirm whether SMS fell within the

sedentary to moderately active category. Similar to the Gladys Block FFQ, a user account was set-up for which data are submitted and accessed.

Cardiorespiratory Fitness Test

Measurement of estimated aerobic fitness consisted of a submaximal cycle ergometer test using the Astrand's cycle ergometer protocol.[113] This protocol uses the relationship between HR and VO2 to predict $\dot{V}O2_{max}$ at submaximal workloads using the Astrand Ryhming Nomogram. The participant had a 2-3 minute warm-up pedaling at 50 revolutions per minute (RPM) and a workload of 0 kiloponds (KP) and during this time testing protocol was explained. The Astrand Protocol is a 6 minute single stage test and required the participant to pedal at 50 RPM and began with an initial workload based on estimating his physical conditioning status. Sedentary to moderately active student participants began at a power output of 100 watts (W) and SA participants at 150 W. Heart rate was recorded every minute after the participant was 2 minutes into the test (minutes 3, 4, 5 etc.). At the 2 minute mark if the participant's HR was not within125-170 beats per minute (BPM) then the mean power output was adjusted accordingly to meet the desired HR range. The HR was recorded using a Polar HR telemetry system (Gays Mills, WI). Blood pressure and rating of perceived exertion (RPE) based on the Borg Scale were recorded at the 2-3 minute mark and the 5-6 minute mark and beyond in participants going longer than 6 minutes.[166] If the HRs at the 5 and 6 minute mark were within 5 beats/min of each other, this was considered as metabolic steady state. Thus, the average of the 2 HRs along with the power output could be used to estimate $\dot{V}O2_{max}$ and rank fitness level according to the Astrand Ryhming Protocol. If the last two HRs differed by more than five beats/min, then another minute was added to the test to allow for a steady state to be achieved. Participants then

participated in an active cool down and had BP and HR monitored for 5 minutes or until HR stabilized and dropped below 120 beats/min. Data collected were used to estimate $\dot{V}O2_{max}$ based on the Astrand Ryhming Nomogram using the formula developed by Shephard.[167]

Statistical Analysis

All study variables were entered into SPSS (Version 21, IBM SPSS Statistics, Chicago, IL) by a research team staff and trained upper level undergraduate student volunteers. Once all variables were entered into SPSS, the data entry was checked by an independent reviewer. Potential outliers or implausible data were discussed by the research team staff to determine if the potential outliers were impossible numbers due to measurement error or equipment malfunctions. Descriptive statistics (mean ± standard deviation) were used to describe the characteristics of each group including age, anthropometrics, CVD risk factors, and dietary intake variables.

Analysis for Aim 1

Hypothesis a, c, d and e was be tested using analysis of variance. Hypothesis b was tested using Pearson's chi square test.

Analysis for Aim 2

Hypothesis a was tested using analysis of variance. Hypothesis b and c were tested using Pearson's correlation. Hypothesis d was tested using William's modification to the Hotelling test to determine the differences in the correlations established in hypothesis b and c.

Analysis for Aim 3

Hypothesis a was tested using analysis of variance. Hypothesis b and c were tested using Pearson's correlation.

Analysis for Aim 4

Hypothesis a was tested using a one sample t-test. Hypothesis b, d, e and f were tested by using analysis of variance to test the differences between the groups and then Pearson's chi square test to determine differences in the rate meeting national recommendations. Hypothesis c was tested using analysis of variance. Hypothesis g was tested using Pearson's correlation.

Z-score Calculation

Using a Z-score approach, the cCVD s was calculated for each participant. Risk factors were regressed onto age to account for age related differences. The cCVDs was created from the summation of the standardized residual Z-scores for the individual risk factors. HDL and $\dot{V}O2_{max}$ are inversely related to CVD risk and were multiplied by -1 before summing all of the risk factors. A higher score indicated a less favorable CVD risk status. Both groups were combined to calculate Z-scores for each cCVDs risk factor. The following risk factors were included in the CVD risk score: TC:HDL ratio, TG, FBG, MAP, WC, and estimated $\dot{V}O2_{max}$ (an index of aerobic fitness). The alpha level of $\alpha \leq 0.05$ was adjusted to $\alpha \leq 0.001$ based on the Bonferroni adjustment (α/number of tested hypothesis=0.005/26=0.0019), in order to protect against a type one error.

3. RESULTS

Descriptive Statistics

The final sample included 40 participants (20 SA and 20 SMS) who participated in the study and were matched based on BMI. The matching of BMI was planned be within 1 kg/m^2 unit of BMI however 2 SMS participants had BMI matching outside of criteria but were still included in the analysis (1.4 kg/m^2 and 1.5 kg/m^2 BMI unit difference). There were no

statistically significant differences in age, weight and BMI between SA and SMS with the exception of a significantly greater height in the SA ($p<0.01$). In total, 28 SA were measured however 8 were not included in the final sample due to not having a participant in the SMS group with a matching BMI. Descriptive statistics for the SA and SMS groups are listed in **Table 5**. The SA group was 70% Caucasian, 25% African American and 5% Hispanic. The SMS group was 60% Caucasian, 15% African American, 10% Hispanic, 5% Asian, and 10% unspecified.

Not all of the participants completed the total measurement protocol and some participant's data were determined invalid. Thirty-six participants had their finger stick blood sample taken and analyzed for CRP, however during the measurement period the Cholestech LDX manufacturer reported the CRP cassettes were faulty. Due to this, CRP was not measured in the remaining participants and no CRP results are reported. Low-density lipoprotein cholesterol could not be calculated for 4 SA and 5 SMS due to TG being <45 mg/dl. Resting metabolic rate was not measured for 2 SA and 9 SMS due to the Korr ReeVue device requiring maintenance. Dietary variables were not analyzed for 3 SA and 4 SMS due to reported dietary intakes determined to be invalid as described in the methods. Inclusion criteria required SMS to meet criteria for classification as sedentary to moderately active, however upon analysis of the energy expenditure survey, 15 of 20 SMS were estimated to participate in greater than 1000 MET minutes per week. Participation in >1000 MET minutes per week would result in a classification of PA greater than moderately active and thus would require those participants to be excluded from data analysis. Due to the large portion of SMS not meeting inclusion criteria, these participants were not excluded due to the still significant difference between the SA and SMS in minutes of MVPA ($p<0.001$). The variables that were invalid or missing data, analysis were performed for the participants who had valid data.

Aim 1. Comparison of Cardiovascular Disease Risk

All the CVD risk factors assessed are reported in **Table 5** including the cCVDs and the individual risk factors used for the cCVDs. The overall hypothesis of the study was that SA would have a significantly more desirable cCVDs compared to SMS. There was not a statistically difference in the cCVDs between the two groups (p=0.34). **Figure 1** shows the percentage of participants not meeting national recommendations for the given CVD risk factor cut points.[28-34] There were no statistical differences between SA and SMS in the level of those not meeting national recommendations for TC, LDL, HDL and TG. There was no statistical difference between groups for systolic BP, diastolic BP, and FBG.

Aim 2. Comparison of Body Composition and its Relationship to Cardiovascular Disease Risk

All anthropometric measurements are reported in **Table 5**. Contrary to what was hypothesized, SA did not have a significantly lower BF% than SMS (p=0.011). **Table 7** shows the relationship primary variables including the cCVDs and anthropometric variables of both SA and SMS separately. Body fat percentage was directly correlated to the cCVDs in SMS (r=0.72, p<0.001) but not in SA (r=0.16, p=0.49). Body mass index was directly correlated to the cCVDS in SMS (r=0.67, p=0.001) and but not SA (r=0.58, p=0.008). It was hypothesized that BF% would be a better predictor of the cCVDs than BMI, however BMI and BF% did not have a statistically significant relationship with the cCVDs in SA and the correlation of BF% was not statistically different from BMI in the SMS (0.1>p>0.05).

Aim 3. Comparison of Estimated Cardiorespiratory Fitness and Physical Activity and its Relationship to Cardiovascular Disease Risk

Estimated $\dot{V}$ O2 $_{max}$ is reported in **Table 5** and TDEE is reported in **Table 6**. Contrary to what was hypothesized, SA did not have a significantly higher estimated $\dot{V}$ O2 $_{max}$ versus SMS

(p=0.002). Estimated $\dot{V}$ O2 $_{max}$ was not negatively correlated with the cCVDs in the SMS (r=-0.64, p=0.003) or SA (r=-0.37, p=0.11). It was hypothesized that PA level quantified by TDEE would be inversely correlated to the cCVDs, however there was no statistically significant relationship in both SA (r=0.54, p=0.013) and SMS (r=0.51, p=0.022).

Aim 4. Comparison on Dietary Intake and its Relationship to Cardiovascular Disease Risk

Dietary intake variables for SA and SMS are reported in **Table 5**. Contrary to what was hypothesized, mean total fat intake was <35% for both SA and SMS. However, in SA and SMS mean intake of saturated fat and trans fat were above 7% and 1% respectively. There was no statistical difference in saturated fat (p=0.58) and trans fat (p=0.10) intakes between groups. As was hypothesized, SA had a higher intake of simple sugars versus SMS (p=0.001). Mean fiber intake per 1000 Kcal was not significantly different between groups (p=0.43), and neither group achieved the national recommendation of 12.5 g/1000 Kcal. Fruit and vegetable intake was not significantly different between groups (p=0.37), and neither group achieved the national recommendation of ≥9 servings per day. The hypothesis that SA would have a higher intake of potassium and sodium was not upheld (p=0.009 and p=0.002 respectively), and neither group achieved the national recommendations. Grams of fiber per 1000 Kcal did not have a statistically significant relationship with the cCVDs in either SA (p=0.23) or SMS (p=0.20).

4. DISCUSSION

This is the first study to BMI match a group of SMS to SAs with BMI ranging from 25-40 kg/m^2 and compare CVD risk factor status, energy expenditure, body composition, cardiorespiratory fitness, PA and dietary behaviors. The primary aim of the study was to use sample specific, cCVDs to compare CVD risk using a continuous score for each variable as opposed to dichotomous variables with a single cut point “at risk” or “not at risk”. Also, it was

anticipated that the cCVDs would be more sensitive than other risk factor assessment scores like the Framingham Risk Score when comparing CVD risk status in a younger population.[29]

Cardiovascular Disease Risk Assessment

There was not a statistical difference between SA and SMS for the cCVDs. The mean cCVDs for SA was -0.54 ± 3.45 and the mean cCVDs for SMS was 0.54 ± 3.64. The effect size for this variable was calculated to be 0.30.[168] The power for this variable was calculated to be 0.15.[169] In order to have a power of >0.5 with the given effect size, 1052 participants would have been needed per group.[170] Figure 3 shows the distribution of the cCVDs for SA and SMS. Both groups have a similar distribution of scores although the SA group had 9 participants with a cCVDs score over 0 and the SMS group had 11 over 0. The SMS group also, had the highest recorded cCVDs at 8.81. Of the variables used to calculate the cCVDs, SA and SMS did not significantly differ between any variables, which may account for the lack of difference between groups.

It was hypothesized that SA would have a lower prevalence of dyslipidemia based on the cut points shown in **Table 1**. No participants in either the SA or SMS met the high risk cut point for TC (>240 mg/dl), which is below the national average of 7.6% males 20-34 years of age being at risk. This may be in part due to the level of PA in SA and the fact that the majority of the SMS were moderately active.[2] Total cholesterol in SMS was also lower than reported by Spencer in a sample of non-athlete college students who had 7.7% of participants at risk.[22] Yet, compared to that same sample that had 10.5% of participants at risk for low HDL, SMS had a substantially greater amount of participants at risk with 40%. This trend could be due to the

relationship that TC has with HDL. As TC increases so does HDL and as TC decreases HDL does as well. Morrell et al. reported that in a sample of college males mean TC was 177 mg/dl, mean LDL was 110 mg/dl and 30.6% of men met at risk criteria for low HDL. When comparing SA with previous research of collegiate football players, SA's lipid values were similar for HDL and LDL and were more desirable for TC and TG. Previous athlete samples had TC ranging from 162 to 170 mg/dl, HDL from 39 to 49 mg/dl, LDL from 91 to 106 mg/dl, and TG from 83 to 151 mg/dl.[11,12,14,16,17] The SA group had higher percentage of participants at risk for HTN compared to SMS, however this was not statistically different. Both SA and SMS had a higher percentage at risk for HTN compared to the national average of 6.8% in males 20-34 years of age.[2] The SMS percent at risk was similar to a previous study with a college student sample that reported 10.5% of participants were at risk for elevated systolic BP and 11.5% were at risk for elevated diastolic BP.[22] In a study by Haskins et al., sedentary students who were compared to a sample of collegiate football players, average systolic and diastolic BP was 148 and 84 mmHg for systolic and diastolic BP respectively. These levels were significantly higher as compared to the collegiate football players in the study.[16] When comparing SA to other collegiate football player samples, SA's BP was similar, with mean systolic BP ranging from 122 to 136 mmHg and diastolic BP from 70 to 82 mmHg.[11,14,16,17] The SA did not have a lower FBG level than SMS and neither group had participants classified as at high risk. Previous research has reported mean FBG levels for male college students ranging from 86 to 89 mg/dl in convenience samples and a mean FBG of 136 mg/dl in a sedentary student population.[12,16,21] Reported FBG values for collegiate football players have ranged between 86 and 102 mg/dl.[11,12,14,16,17]

Body Composition

It was hypothesized that the SA group would have a significantly lower BF% (21.4 ± 6.6 %) compared to the SMS group (27.1 ± 6.8%; p=0.01), however this was not found. The SMS group had a similar BF% to a sample of US males 20-39 years of age, who averaged 26.1 BF% in 2010.[86] Similarly, a 2012 study reported non-athlete college students had a body fat percentage of 26.8% and which was significantly higher percentage than skilled position football players in the study, but not the lineman.[12] In a study by Haskins et al. comparing sedentary students to collegiate football players which included a comparison of BMI, BF%, and CVD risk status, the sedentary students had a BF% of 27.1% and athletes had a BF% of 21.8% and had a stronger correlation with CVD risk factors than BMI. This contrasts the current study results, as BMI and BF% was positively correlated with the cCVDs in SMS and not SA and there was no statistical difference between BMI and BF% correlations with the cCVDs in the SMS group. The failure of BF% to better predict the cCVDs may be due to the small sample size of the current study.

Cardiorespiratory Fitness and Energy Expenditure

The hypothesis that SA would have a significantly higher estimated $\dot{V}O2_{max}$ than SMS was not upheld (SA=47.6 ± 7.2 ml/kg/min; SMS=40.0 ± 7.2 ml/kg/min; p=0.002). In neither group was estimated $\dot{V}O2_{max}$ negatively correlated with the cCVDs (SA r=-0.37, p=0.114; SMS r=-0.64, p=0.003). In a study by Munoz et al., estimated $\dot{V}O2_{max}$ was also negatively correlated with WC but not with any other CVD risk factors in a sample of collegiate athletes.[18] The lack

of correlation of estimated $\dot{V}O2_{max}$ to any variable in SA and SMS was likely due to a small sample size.

Opposite to what was hypothesized, TDEE was not significantly higher in SA versus SMS however TDEE was approaching a significantly positive correlation with the cCVDs in both SA and SMS (SA r=0.54, p=0.013; SMS r=0.51, p=0.022) and was opposite of what was hypothesized. This finding conflicts with data that has shown that higher PA levels result in a decrease of CVD risk. Related to this, TDEE was also positively correlated with weight, BMI, and WC. Because the cCVDs was calculated with WC, a variable strongly correlated to weight, and TDEE was calculated using participant's weights, it is possible that the relationship between weight and TDEE could overpower the negatively correlated relationships that TDEE may or may not have with other CVD risk factors. An additional potential reason why TDEE was directly correlated with the cCVDs, is the fact that the dietary patterns of both groups was far from dietary and heart health guidelines. As summarized in the results and **Table 6**, the intake of both groups was high in their total intake of both saturated fat and trans fat and had an overall low nutrient density as reflected by low intakes of dietary fiber per 1000 Kcals.

During the screening of SMS for inclusion criteria and exclusion criteria, daily PA measured as MET minutes per week was estimated for each individual. Upon completion and analysis of the energy expenditure survey, 15 of 20 SMS were estimated to participate in >1000 MET minutes per week. At this level of PA, 75% of individuals in the SMS group would be categorized as highly active. This compares to the only 57% of individuals 18-24 years of age that met aerobic and muscle strengthening guidelines in 2010.[2] The highly active participants were still included in the analysis because minutes of moderate to vigorous PA was still significantly less than SA. If the energy expenditure questionnaire correctly reflected SMS actual

activity levels then the SA group was compared to a sample that was participating in sufficient amounts of PA. If this was the case this could possibly explain the lack of difference between groups in the cCVDs and individual risk factors.

Dietary Intake

Overall, SA had a higher intake of Kcal, saturated fat, simple sugars, and added sugars than SMS. Yet, when many of these variables were compared as a percentage of total Kcals or standardized per 1000 Kcal, the differences between groups disappeared. We hypothesized that both groups would have total fat intakes of >35% of total Kcal, however both groups achieved national recommendation targets of <35% of Kcal from fat. However, as summarized in **Table 6**, both groups were slightly above recommendations for percentage of calories from saturated fat (~11%) and trans fat (~1%). The saturated fat intake as a % of total Kcals (~11%) reported for both groups was similar to the 10% value that Morrell et al. reported in a sample of 2,103 university students.[21] Cole et al. reported a similar intake of saturated fat in collegiate football players at 10%.[95] In another collegiate football sample, 11% of Kcal came from saturated fat and in that same sample, average fiber intake was 7.4 grams per 1000 kcal which was under the national recommendation of 12.5 grams per 1000kcal.[97] Neither, SA or SMS met guidelines for intake of fiber per 1000 Kcal or ≥9 servings of fruit and vegetables per day. Fruit and vegetable intake in SMS was higher than what Cole et al. reported at 2.9 servings per day in collegiate football players. In the SMS group, 100% did not meet the goal for fruits and vegetables (≥9 servings). In a 2008 report, 91.5% of the 80,121 college students surveyed in the US ate <5 servings of fruits and vegetables.[20] Both SA and SMS had higher intakes of fruits and vegetables than other researched samples which in part could be due to the large body size of our study

population. Regardless, the higher fruit and vegetable intake as compared to other studies may contribute to why several CVD risk factors in SA and SMS were more desirable than previous research on college athletes. Yet, fruit and vegetable intake did not have a significant measured relationship with any of the CVD risk factors measured in this study.

The SA group had a total Kcal intake of 3445 ± 1233 and a TDEE of 3467 ± 552 Kcal. These values were obtained after 3 SA were excluded from analysis for having impossibly low Kcal intakes and reporting they were not trying to lose weight. Yet, compared to general recommendations for Kcal intake for athletes training on a near daily basis for several hours (41 Kcal/day), SA Kcal intake was less than recommended (4,399 Kcal/day).[171] Cole et al. reported this same Kcal intake discrepancy in a sample of football players and noted that there was no accompanied weight loss that would be expected with a calorie deficit.[95] Hinton et al. also noted that a sample of collegiate athletes that consumed 400 Kcal below recommended levels.[97] These findings may be explained by athlete's having a tendency to underreport energy consumption.[99]

While TDEE and Kcal intake for SA were very similar, SMS had a 859 Kcal energy deficit when comparing TDEE to total Kcal intake. This deficit seems plausible in the SMS group because 14 of the 16 participants analyzed were trying to lose weight. In a national sample, the total Kcal intake in 2008 for males 20-39 years of age was 2946 kcal which is very similar to the SMS group's TDEE.[37] This similarity suggests that the SMS group may have been trying to lose weight through diet but there is also the possibility that dietary intake was under reported or was reduced as a result of enrolling in the study.

Study Strengths and Limitations

The first limitation is this study was cross-sectional, thus causal relationships cannot be determined. A second limitation is the lack of statistical power. When this study was being planned there was no previous research that used a continuous CVD risk score, which was what was used to power the study. Thus, when the pre-study power analysis was performed it was based on the effect size of several values in the score and not on the score itself. This may be why there were fewer statistically different results than hypothesized. The lack of statistical significance was also affected by having a smaller sample size than was planned, 40 total participants versus 60. While the original SA group had 28 participants it became increasingly difficult to find SMS that BMI matched. Another limitation is that previous research focused on CVD risk in collegiate populations and the cut points chosen for those at risk, or meeting recommended levels are not consistent. For this reason, the most rigorous cut points for determining "at risk" versus using cut points for meeting recommended or desirable levels were used in this study. For example, for total cholesterol the cut point was 240mg/dl versus the 200mg/dl or less for recommended level; for systolic BP cut point of 140mmHg (cut point for hypertension) versus the recommended level of 120mmHg or less; diastolic BP cut point of 90mmHg (cut point for hypertension) versus the recommended level of 80mmHg or less; for FBG the cut point was 126mg/dl (cut point for diabetes) versus 100mg/dl or less which is the recommended level. If the cut points were based on levels for those that met the recommended level it would have resulted in a higher prevalence of this young population being classified at risk. A forth limitation was that the 20 SA participants were recruited and measured over several months spanning from preseason to the end of their season. There have been several studies showing that Kcal intake, TDEE, body composition and lipid values can change throughout an athletic season due to variation in training and playing time.[97,172,173] A fifth limitation to this

study was a convenience sample of college students was used that may have resulted in a selection bias for individuals that were more interested in CVD risk factor status and other health measures as compared to the general college population. This could possibly be the reason why fruit and vegetable intake was higher than expected in the SMS group. Another limitation is that while the SA and SMS had a similar percentage of Caucasian, African American and Hispanic participants, the SMS group also had 2 Asian participants and there were 4 participants that had an unspecified ethnicity. It is unknown whether the participants who did not specify their race were more or less predisposed to CVD risk or it was related to their lifestyle. There are also limitations to using a Z-score approach cCVDs. The cCVDs is specific to the population that it was computed in and may not be comparable to the population as a whole. The Z-score approach also does not weigh individual risk factors that may play a greater or lesser role in CVD risk. Using BIA for analysis of body composition is beneficial due to the ease of administration, safety and speed of assessment, though the result could be altered by hydration status, which influences estimations of BF%.[160] To help control for this limitation, participants were asked to come into measurement after an overnight fast and were instructed to drink fluid prior to arrival. Food frequency questionnaires and PA questionnaires are useful due to their small participant burden, cost, time requirement and ease of administration. However, they rely heavily on participants' memory which makes recall bias and poor memory a possible limitation. Standardized procedures were used during survey administration to try to control for this limitation as much as possible.

Strengths identified in this study include the following. This study examined CVD risk factors in SA compared to SMS which were matched based on BMI (on average <1kg/m^2 BMI unit difference). Previous research in this area has primarily focused on football players with few

studies utilizing BMI matching for athletes with non-athlete college students. The use of a continuous cCVDs is more sensitive for the college populations than previous cCVDs assessment methods including the, Framingham Risk Factor Score which was developed for assessment of CVD risk in older populations.[71] This study also controlled for several variables that have roles in CVD risk status that many studies do not account for including estimated fitness level, dietary intake and total energy expenditure. A final strength was that the alpha level was adjusted to a conservative level in order to protect against type one errors.

5. CONCLUSION

This study's results showed that when comparing collegiate male SA to collegiate male SMS who were matched based on BMI, groups did not have significantly different composite or individual CVD risk factor levels. Their overall level of risk was similar or worse than national averages with 35% having 2 or more CVD risk factors and 58% having one or more. Despite SA having a significantly higher amount of daily MVPA, there was a lack of difference between the SA and SMS in most other study variables. The lack of difference may be due in part to the fact that majority of the SMS participants were not sedentary. Also, both groups had a similar dietary composition, which did not meet heart health recommendations. The SA had a higher caloric level that corresponded with their high lean body mass. These findings may reflect the importance of combining both moderate levels of PA with healthy dietary behaviors to prevent CVD risk development. A major limitation to the study was the lack of statistical power due to a smaller sample size. Also, the criteria for selected individual CVD risk factors variables used the most rigorous cut points for determining "at risk" versus using cut points based on desirable or recommended levels. If the cut points were based on meeting the recommended levels, a higher prevalence of this young population would be classified at risk.

This study added to the present literature on CVD risk in young adults by BMI matching overweight and obese SA and SMS and evaluating body composition, aerobic fitness, and dietary and PA behaviors. Previous research with similar study groups did not evaluate dietary intake or cardiorespiratory fitness in relation to CVD risk. The cCVDs used to assess CVD risk may be a useful tool for assessment of CVD risk in groups of young adults, however further research needs to be performed with larger sample sizes and consistent cut points. Overall, this study shed light into the relationship that body composition, PA, cardiorespiratory fitness and diet have with CVD risk.

Based on this data, an intervention target would be to increase the proportion of those achieving dietary targets for cardiovascular health, and to reduce the BMI and BF% particularly in those with ≥1 CVD risk factor. Given that young adults in the U.S. commonly have ≥1 CVD risk factor, proper CVD screening is important for identifying those at risk so lifestyle modifications and or pharmacological treatment can be used to reduce the risk of CVD morbidity and mortality. While it is commonly assumed that athletes are at a decreased risk for CVD due to high levels of PA, we found that overweight and obese BMI matched SA and SMS had similar CVD risk status despite higher amounts of MVPA in the SA.

APPENDIX

Table 1: Cardiovascular disease risk factors

CVD Risk Factors*
Causal Risk Factors
Cigarette Smoking
BP ≥140/90 mg/dl
LDL ≥160 mg/dl
HDL <40 mg/dl (males), <50 mg/dl (females)
TC ≥240 mg/dl
Diabetes (FBG >126 mg/dl)
Age ≥45 years (males) ≥55 years (females)
Obesity (BMI ≥ 25; WC ≥ 40 in (≥102 cm) males or ≥35 in (≥88 cm) females)
Physical Inactivity
Predisposing Risk Factors
Male Gender
Genetics
Family history of premature CHD (first degree relative; <55 years in males and <65 years in females)
Insulin resistance
Emerging/Conditional Risk Factors
Triglycerides ≥150 mg/dl
Homocysteine ≥10 micromoles/L
CRP ≥3 mg/L
*Risk factors adapted from American Heart Association, American College of Cardiology and ATP III. [28-34]

Table 2: Classifications of overweight, obesity and disease risk for men

Classifications of Overweight, Obesity and Disease Risk Per BMI and WC for Men[44]				
			Disease Risk Relative to Normal Weight & WC*	
Weight Class	BMI (kg/m^2)	Obesity Class	WC ≤102 cm (≤40 in)	WC >102 cm (>40 in)
Underweight	<18.5		-	-
Normal	18.5-24.9		-	-
Overweight	25.0-29.9		Increased	High
Obesity	30.0-34.9	I	High	Very High
	35.0-39.9	II	Very High	Very High
Extreme Obesity	≥40	III	Extremely High	Extremely High
*Diseases risk includes HTN, diabetes and CVD				

Table 3: Physical activity recommendations for adults

Physical Activity Recommendations for Adults		
Physical Activity Component	2008 Physical Activity Guidelines for Americans *	ACSM Position Stand**
Cardiorespiratory Exercise	Moderate-intensity for ≥150 min/wk or vigorous-intensity for ≥75 min/wk For additional health benefit moderate-intensity for ≥300 min/wk or vigorous-intensity for 150 min/wk	Moderate-intensity for ≥30 min/day ≥5 d/wk for a total of ≥150 min/wk, vigorous-intensity for ≥20 min/day ≥3 d/wk for a total of ≥75 min/wk, Or a combination of moderate- and vigorous-intensity to achieve energy expenditure of ≥500-1000 MET/min/wk
Resistance Exercise	≥2 d/wk high to moderate-intensity in major muscle groups	2-3 d/wk for each of the major muscle groups
Neuromotor Exercise	Encouraged only to older adults but no quantified recommendation	2-3 d/wk exercise involving balance, agility and coordination
Flexibility Exercise	Encourages to adults but no quantified recommendation	≥2 d/wk flexibility exercise for major muscle tendon groups for 60 sec. per exercise
*Summarized from Department of Health and Human Services[9] **Summarized from American College of Sports Medicine (ACSM) Position Stand: Quality and Quantity of Exercise for Developing and Maintaining Cardiorespiratory, Musculoskeletal, and Neuromotor fitness in Apparently Healthy Adults[110]		

Table 4: Dietary recommendations

Dietary Recommendations		
Dietary Component	AHA Recommendations for CVD Prevention (ATP III TLC Diet)*	ACSM-ADA Position Statement for Athletes**
Carbohydrates	50-60% total calories	6-10 gm/kg body weight
Fiber grams/day	≥ 12.5 gm/1000Kcal	Suggests 30-35 gm/day
Added sugars	≤9 teaspoons/day for men	None given
Fat	25-35% total Kcal	20-35% total Kcal
Saturated Fat	<7% total Kcal	<10% total Kcal
Trans Fat	<1% total Kcal	None given
Protein	~15% of total Kcal	1.2-1.7 gm/kg body weight
Potassium	4700 mg/day	None given
Sodium	<1500 mg/day	None given
Fruits and Vegetables	≥4.5 servings of each/day	≥5 servings of each/day

*Summarized from American Heart Association (AHA) recommendations[3,28,90,94,131,174]

**Summarized from American College of Sports Medicine (ACSM) and Academy of Nutrition and Dietetics (AND) formerly the American Dietetic Association (ADA) guidelines[93]

Table 5: Differences in cardiovascular disease risk factor variables by group

Variable & Risk Cut-off†	SA (n=20) ‡	SMS (n=20) ‡
Age (years)	20.1 ± 1.5 (18-22)	20.5 ± 1.6 (18-24)
Height (cm)	187.6 ± 6.2 (174.0-199.8)*	179.0 ± 6.8 (167.2-191.0)
Weight (kg)	107.3 ±17.9 (80.5-141.1)	97.4 ± 14.6 (74.2-133.1)
Body Mass Index (kg/m^2)**	30.3 ± 3.7 (25.8-38.0)	30.4 ± 4.0 (25.6-39.5)
Waist Circumference (cm)** ≤102 cm	94.9 ± 10.9 (79.3-114.9)	97.2 ± 10.9 (76.0-121.5)
Body Fat Percentage	21.4 ± 6.6 (12.0-37.4)	27.1 ± 6.8 (17.5-40.8)
Fat-free Mass (kg)	83.6 ± 10.3 (65.4-104.8)*	70.3 ± 6.2 (61.1-80.1)
Fat Mass (kg)	23.7 ± 10.9 (11.5-51.2)	27.2 ± 10.6 (13.0-54.3)
Systolic Blood Pressure (mmHg) ≥140 mmHg	124.0 ± 14.9 (112-182)	123.1 ± 9.8 (108-141)
Diastolic Blood Pressure (mmHg) ≥90 mmHg	81.3 ± 7.5 (62-99)	80.1 ± 7.3 (63-96)
Mean Arterial Pressure**	95.6 ± 7.5 (81.3-112.7)	94.4 ± 7.5 (78.7-108.7)
Total Cholesterol (mg/dl)** ≥240 mg/dl	151.9 ± 28.6 (111-216)	155.1 ± 30.8 (103-211)
High-density Lipoprotein(mg/dl) ** <40 mg/dl	43.6 ± 12.8 (16-71)	45.5 ± 15.7 (24-81)
Triglyceride (mg/dl)** ≥150	79.5 ± 27.3 (45-120)	88.8 ± 45.8 (45-203)
Low Density Lipoprotein (mg/dl)#** ≥160 mg/dl	96.7 ± 32.5 (48-166)	95.7 ± 29.2 (41-143)
Total Cholesterol to High Density Lipoprotein Ratio**	3.9 ± 1.7 (2.1-8.5)	3.7 ± 1.3 (1.7-6.4)
Fasting Blood Glucose (mg/dl)** >126 mg/dl	92.8 ± 8.0 (76-108)	93.5 ± 5.7 (82-107)
Estimated Maximum Oxygen Consumption (ml/kg/min)**	47.6 ± 7.2 (38.5-59.0)	40.0 ± 7.2 (29.8-53.0)
cCVDs	-0.54 ± 3.4 (-7.25-6.07)	0.54 ± 3.6 (-5.42-8.81)

SA = student athlete; SMS = sedentary to moderately active students

†NCEP at risk level noted below variable title, where applicable

‡Values expressed as mean ± SD with range in parentheses

*p≤0.001 between two groups

#Sample size varies by measure due to missing variables

**Indicates variable used for calculation of composite cardiovascular disease risk score

Table 5 (cont'd)

mg=milligrams; min=minute; ml=milliliter; dl=deciliter; kg=kilogram; cCVDs=cardiovascular disease risk score

Table 6: Differences in daily energy expenditure and dietary intake by group

Variable & Risk Cut-off†	SA (n=20) ǂ	SMS (n=20) ǂ
Total Daily Energy Expenditure (kcal)	3467 ± 552 (2573-4843)	2923 ± 771 (2054-5321)
Moderate-Vigorous Physical Activity (min/day)	46.7 ± 6.4 (38.4-61.4)*	9.1 ± 8.8 (0.1-29.6)
Resting HR (beats/min)#	61.4 ± 7.7 (49.0-75.3)	66.4 ± 7.7 (60.3-86.7)
Resting Metabolic Rate (Kcal)#	2352 ± 382 (1627-2851)	2213 ± 352 (1570-2810)
Total Kcal#	3445 ± 1233 (2054-6252)*	2064 ± 588 (1279-3488)
Carbohydrate (gm)#	425.4 ± 175.1 (238.1-741.8)*	243.5 ± 60.0 (112.1-366.0)
Protein (gm)#	139.2 ± 56.8 (75-285.7)*	83.1 ± 30.7 (50.3-157.7)
Saturated Fat (gm)#	41.3 ± 14.5 (20.4-71.7)*	26.0 ± 8.9 (15.5-47.0)
Saturated Fat (gm/1000Kcal)#	12.2 ± 2.2 (7.8-17.3)	12.6 ± 1.6 (9.5-15.7)
Trans Fat (gm)#	4.2 ± 1.5 (2.4-7.0)	3.5 ± 2.5 (1.4-10.6)
Trans Fat (gm/1000Kcal)#	1.3 ± 0.3 (0.7-1.9)	1.6 ± 0.6 (0.8-3.2)
% Kcal Carbohydrate#	48.7 ± 5.7 (38.0-58.2)	47.9 ± 8.3 (35.1-65.9)
% Kcal Protein#	16.1 ± 1.8 (12.3-18.6)	16.0 ± 3.0 (10.8-22.6)
% Kcal Fat# <35% Kcal	34.6 ± 4.3 (26.8-41.1)	34.6 ± 5.4 (24.5-44.9)
% Kcal Saturated Fat# <7% Kcal	10.9 ± 2.0 (7.0-15.6)	11.3 ± 2.0 (8.5-14.1)
% Kcal Trans Fat# <1% Kcal	1.1 ± 0.3 (0.6-1.7)	1.4 ± 0.6 (0.8-2.9)
Sodium (mg)# <1500 mg	5386 ± 2196 (2564-10763)	3297.3 ± 1059.4 (1974-5643)
Potassium (mg)# ≥4700 mg	4274 ± 2147 (2420-9992)	2651 ± 939 (1444-4900)
Dietary Fiber (gm)#	27.1 ± 14.9 (12.9-63.5)	17.2 ± 7.1 (8.8-34.8)
Fiber Intake (gm fiber/1000 Kcal)# ≥12.5 gm/1000Kcal	7.7 ± 2.0 (4.4-12.3)	8.3 ± 2.3 (5.0-13.5)
Fruit and Vegetable Servings# ≥9 servings/day	4.9 ± 4.0 (1.4-16.7)	3.9 ± 2.2 (1.1-8.5)
Sugars (gm)#	215.3 ± 98.4 (99.1-403.7)*	114.2 ± 37.0 (58.4-168.3)
Added Sugars (teaspoons)# ≤9 teaspoons/day	28.1 ± 14.4 (11.1-56.7)*	14.0 ± 5.8 (3.1-25.2)
SA = student athlete; SMS = sedentary to moderately active students		

Table 6 (cont'd)

†AHA recommendation noted below variable title, where applicable

‡Values expressed as mean ± SD with range in parentheses

*$p \leq 0.001$ between two groups

#Sample size varies by measure due to missing variables

Kcal=Total calories; gm=grams; mg=milligrams; min=minute; ml=milliliter

Table 7: Correlation (r value) among study variables for SA and SMS individually

Variables	cCVDs	TC	HDL	LDL	TG	WT	BMI	BF%	WC	MAP	RMR	VO2 Max	TDEE	Daily Min	Sat. Fat	Trans Fat	Na	K	Fiber	Fr/Veg
cCVDs		.13	**-.86**	.24	.37	**.67**	.58	.16	**.70**	.55	.64	-.37	.54	-.02	.06	-.11	.44	.40	.31	.46
TC	.43		-.14	**.92**	.29	.08	.14	.02	.10	-.27	.10	-.31	-.06	-.06	.23	.10	-.24	-.20	-.13	-.28
HDL	-.21	.26		-.49	-.02	**-.67**	-.50	-.10	-.65	-.45	**-.70**	.38	-.43	.27	-.05	.09	-.31	-.27	-.34	-.36
LDL	.30	**.85**	-.34		-.13	.15	.10	-.15	.14	-.40	.29	-.49	-.16	-.42	.29	.18	-.33	-.31	-.10	-.34
TG	**.69**	.37	-.23	.11		.33	.44	.35	.36	-.08	.02	-.03	.43	.34	-.24	-.39	.25	.35	.27	.38
WT	**.70**	.14	-.03	.10	.06		**.94**	**.67**	**.97**	.23	**.73**	-.34	**.83**	-.15	-.02	-.07	.43	.47	.27	.43
BMI	**.67**	.08	-.10	.05	.29	**.87**		**.82**	**.95**	.13	.69	-.33	**.80**	-.15	-.19	-.20	.50	.57	.29	.51
BF%	**.72**	.24	-.04	.14	.29	**.80**	**.90**		**.70**	-.07	.51	-.28	.60	-.11	-53	-.20	.28	.42	.34	.42
WC	**.76**	.26	-.18	.29	.39	**.91**	**.89**	**.87**		.20	**.76**	-.38	**.79**	-.20	-.09	-.15	.43	.48	.31	.46
MAP	.38	-.04	.20	-.19	.04	.50	.38	.37	.26		.41	.18	.23	.15	.18	.02	.27	.21	-.04	.30
RMR	-.10	.09	.25	-.21	-.05	.34	.04	-.08	-.03	.14		-.32	.48	-.38	.01	.00	.26	.27	.29	.41
VO2 max	-.64	-.44	.15	-.46	-.21	-.27	-.43	-.55	-.46	.10	.37		.01	.38	.20	-.08	.23	.16	-.21	.16
TDEE	.51	.29	.30	.06	.14	**.86**	**.71**	.63	**.70**	.50	.60	-.15		.29	-.07	-.17	**.80**	**.71**	.10	.59
Daily Min	.27	.26	.25	-.04	.37	.35	.31	.14	.25	.22	.60	-.08	.52		.24	-.32	.41	.33	-.03	.24
Sat Fat	.59	.16	-.03	.08	.51	.39	.38	.19	.40	-.14	.09	-.38	.26	.42		.61	-.21	-.32	-.37	-.41
Trans Fat	.41	.29	.07	.19	.20	.11	.20	.23	.30	-.26	-.67	-.43	.05	-.22	.39		-.42	-.55	-.52	-.62
Na	.07	-.00	.23	-.13	-.10	.17	.37	.34	.25	-.25	-.23	-.24	.22	-.11	.26	.69		**.94**	.34	**.84**
K	.20	.24	.11	.17	.07	.25	.34	.23	.35	-.36	-.12	-.23	.29	-.04	.23	.71	**.74**		.50	**.92**
Fiber	-.34	-.37	-.30	-.03	-.20	-.23	-.14	-.22	-.18	-.56	-.22	.10	-.31	-.44	-.24	.14	.14	.47		.69
Fr/Veg	-.09	-.04	-.01	.02	-.03	.09	.10	-.03	.11	-.38	.02	.15	.15	-.17	-.06	.49	.51	**.86**	.72	

#Correlations for SA are above the black line intersecting the table and correlations for SA are below the black line.

* Bolded underlined Darker grey boxes indicate a p-value≤0.001; Darker grey boxes p-value<0.01; lighter grey boxes p-value<0.05

cCVDs=composite cardiovascular disease score, TC=total cholesterol, HDL=high-density lipoprotein, LDL=Low-density lipoprotein, TG=triglyceride, WT=weight, BMI=body mass index, BF%=body fat percentage, WC=waist circumference,

Table 7 (cont'd)

MAP=mean arterial pressure, RMR=resting metabolic rate, VO2 max=maximum oxygen consumption, TDEE=total daily energy expenditure, Daily Min=daily minutes of moderate to vigorous physical activity, Sat Fat=% kcal saturated fat, Trans Fat=% kcal trans fat, Na=daily sodium intake, K=daily potassium intake, Fiber=fiber per 1000 kcal, FR/Veg=fruit and vegetable servings

Figure 1: Prevalence of cardiovascular disease risk factors by group

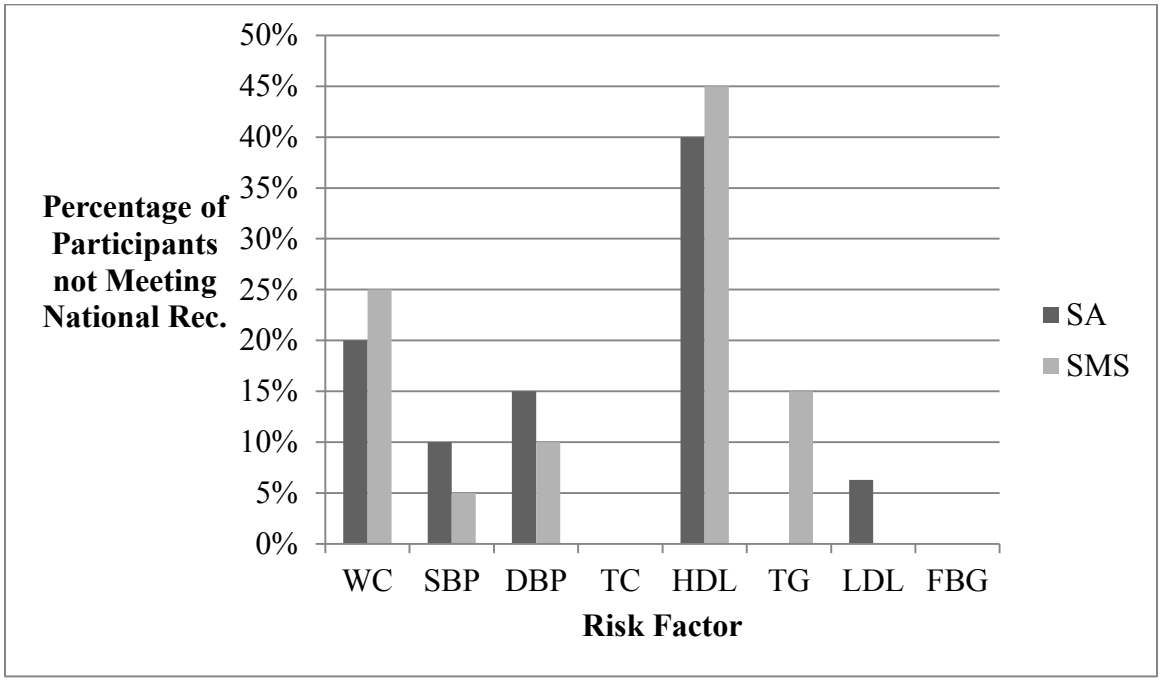


Figure 2: Prevalence of participants not meeting dietary guidelines by group

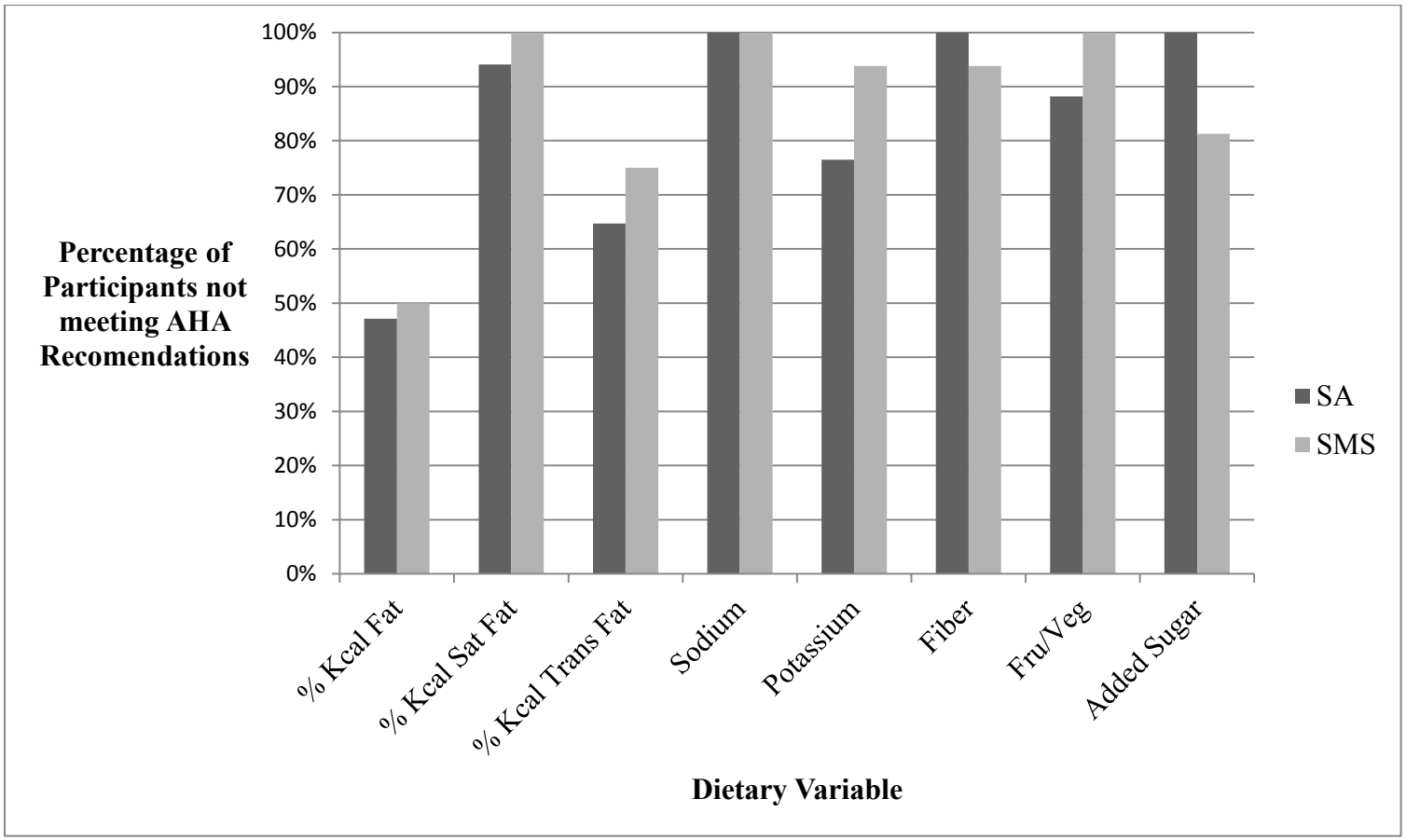
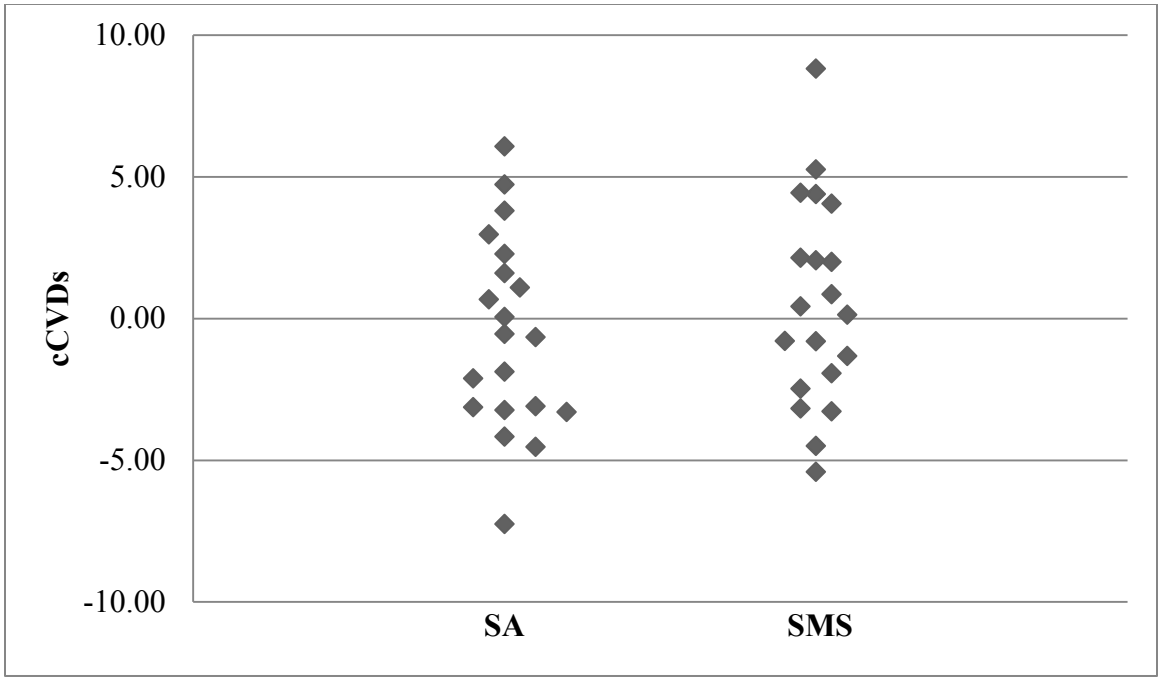


Figure 3: Distribution of cCVDs by group



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