# CARDIOVASCULAR DISEASE RISK FACTOR STATUS AND DIETARY INTAKE IN OVERWEIGHT AND OBESE FEMALE COLLEGE ATHLETES VERSUS SEDENTARY COLLEGE STUDENTS

By

Katilyn L. Murtha

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ABSTRACT

CARDIOVASCULAR DISEASE RISK FACTOR STATUS AND DIETARY INTAKE IN OVERWEIGHT AND OBESE FEMALE COLLEGE ATHLETES VERSUS SEDENTARY COLLEGE STUDENTS

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Although seen as a disease of middle age, cardiovascular disease (CVD) is the third leading cause of death in individuals 15-24 years of age. There is limited data on risk in female athletes overweight or obese classified by body mass index (BMI) ≥25 kg/m$^2$. The objective of this study was to compare CVD risk status and diet quality between female Division I student athletes (SA) and sedentary students (SS) with BMI ≥24.5 kg/m$^2$. Secondary objectives included evaluating the influence of total daily energy expenditure (TDEE), BMI, percent body fat (% BF), and dietary quality on CVD risk. Thirty-seven college women (20 SA; 17 SS) 19.7 $\pm$ 1.2 years old with a mean BMI of 28.6 kg/m$^2$ (range 24.7-39.6) participated. Primary analysis was a composite CVD risk score (cCVDs): resting blood pressure, waist circumference, fasting triglycerides, high density lipoprotein (HDL), blood glucose, and C-reactive protein. The remainder of the lipid profile and resting heart rate (RHR) were also assessed. The cCVDs was not significant between the two groups (p=0.373), however, total cholesterol, TC:HDL, low density lipoprotein, % BF, and RHR were significantly lower in SA. Significantly fewer SA (35%; 7/20) versus SS (71%; 12/17) had $\geq$ 2 risk factors. Neither group's diet met heart health guidelines. BMI and % BF were positively correlated with cCVDs in SA. Overall, the SA had a lower prevalence of CVD risk factors and a superior risk factor profile compared to the SS which was likely due to the significantly lower % BF and higher TDEE in the SA.

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## KEY TO ABBREVIATIONS

| | |
|---|---|
| % BF | Percent body fat |
| ACSM | American College of Sports Medicine |
| ADA | American Dietetics Association |
| AHA | American Heart Association |
| BIA | Bioelectrical impedance analysis |
| BMI | Body mass index |
| BMR | Basal metabolic rate |
| BP | Blood pressure |
| CARDIA | Coronary Artery Risk Development in Young Adults |
| cCVDs | Composite cardiovascular disease risk score |
| CDC | Center for Disease Control and Prevention |
| CHD | Coronary heart disease |
| CRP | C-reactive protein |
| CVD | Cardiovascular Disease |
| DM | Diabetes Mellitus |
| FBG | Fasting blood glucose |
| FFQ | Food frequency questionnaire |
| HDL | High-density lipoprotein |
| LDL | Low-density lipoprotein |
| Kcal | Kilocalories |
| MAP | Mean arterial pressure |
| MET | Metabolic energy equivalent |

| | |
|---|---|
| MUFA | Monounsaturated fatty acid |
| MVPA | Moderate-vigorous physical activity |
| NCAA | National Collegiate Athletic Association |
| NCEP | National Cholesterol Education Program |
| NHANES | National Health and Nutrition Examination Survey |
| PA | Physical activity |
| PUFA | Polyunsaturated fatty acid |
| RHR | Resting heart rate |
| RMR | Resting metabolic rate |
| SA | Student athletes |
| SFA | Saturated fatty acid |
| TFA | Trans fatty acid |
| SS | Sedentary students |
| TC/HDL | Ratio of total cholesterol to high-density lipoprotein |
| TC | Total cholesterol |
| TDEE | Total daily energy expenditure |
| TG | Triglyceride |
| TLC | Therapeutic Lifestyle Changes |
| WC | Waist circumference |

# CHAPTER 1

## INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death in women in the United States, with more women than men dying from CVD since 1984[1]. More than one in three females has some form of CVD. In 2006, all cardiovascular diseases combined claimed the lives of 432,709 females in the United States, topping the number of deaths from all forms of cancer combined (269,819 females)[1]. CVD is the third leading causes of death in those ages 15-24, after malignant neoplasms and accidents (unintentional injuries).[2] Major risk factors for heart disease include high blood pressure (BP), high cholesterol, diabetes, smoking, physical inactivity and obesity.[3] The prevalence of having no major risk factors is decreasing among men and women in nearly all states, racial/ethnic populations, age groups, and education levels.[3] Among women ages 20 and older, currently 31% of non-Hispanic whites, 45% of non-Hispanic blacks, and 32% of Mexican Americans have high BP. In 2006, females accounted for 57% of deaths from high BP.[1] In that same group, currently 49% of non-Hispanic whites, 42% of non-Hispanic blacks, and 49% of Mexican Americans have total blood cholesterol levels (TC) over 200 mg/dL.

Prevention of CVD has become a focus of population-based programs.[4,5] For example, the first goal of Healthy People 2010[4] is to help individuals of all ages to increase life expectancy and improve their quality of life. A specific objective of Healthy People 2010 focuses on CVD and stroke. The goal for this objective is to, "improve cardiovascular health and quality of life through the prevention, detection, and treatment of risk factors; early identification

and treatment of heart attacks and strokes; and prevention of recurrent cardiovascular events."[4]

In addition, the American Heart Association (AHA)[5] recently released its goals for cardiovascular promotion and disease reduction through 2020 and beyond. Their overall goal is "By 2020, to improve the cardiovascular health of all Americans by 20% while reducing deaths from cardiovascular disease and stroke by 20%". To achieve this goal, the AHA emphasizes *primordial prevention* which is (on an individual level) preventing the development of risk factors in the first place. Specifically, the AHA recommends screening potentially at-risk groups and encourages heart healthy nutrition and physical activity (PA) behaviors to prevent future morbidity.

It is possible that athletes may be one of these "at-risk groups". In 1956, Montoye et al.[6] found that when compared with 583 non-athletes, data on 629 athletes was not significantly difference in terms of life expectancy, cause of death, or the type of death. In 1998, Williams[7] investigated how intensity and volume of exercise affected CVD risk factors. Williams found that male and female recreational runners who ran at a greater intensity had lower BP, triglycerides (TG), ratio of TC to high-density lipoprotein (TC/HDL), body mass index (BMI), and waist circumference (WC). Running velocity had a 2.8 times greater calculated effect on diastolic BP, and a 13.3 and 5.7 times greater calculated effect on systolic BP in men and women, respectively. However, running distance had a more than 6-fold greater effect on high density lipoprotein (HDL) than running velocity in both sexes. There are definite dose-response relationships between exercise training volume and blood lipid changes.[8] Thresholds for observable changes occur at training volumes of 15-20 miles per week of brisk walking or jogging and expending between 1200-2200 Kcal/week. This exercise regimen is associated with

2-3mg/dl increases in HDL and 8-20mg/dl decreases in TG, and the effect is similar in both sexes.[8]

Recent studies that have evaluated the overall CVD risk status of collegiate and professional football player have revealed that some of these athletes are at increased risk for CVD.[9-11] Buell et al.[9] found that 34 of 70 collegiate football lineman exhibited metabolic syndrome based on having abnormal levels of three or more of the following CVD risk factors: BP, WC, fasting blood glucose (FBG), HDL, and TG. Two studies compared risk factors in National Football League (NFL) players versus the general population (obtained from the Coronary Artery Risk Development in Young Adults (CARDIA) study[10] and the National Health and Nutrition Examination Survey (NHANES)[11]). Tucker et al.[10] found that the NFL population had a CVD risk profile similar to the general population. Selden et al.[11] found a similar prevalence of metabolic syndrome in National Football League (NFL) players verses a similar age population from the National Health and Nutrition Examination Survey (NHANES) (6% in NFL versus 10% in NHANES; p=0.355). However, the prevalence of elevated resting BP and FBG were significantly higher in the NFL players verses the NHANES sample.

Compared to the amount of data in male athletes, there is limited data on the CVD risk factor status in female athletes. Orri et al.[12] conducted a cross-sectional investigation assessing CVD risk in a small sample of 30 male and female college students and intercollegiate athletes. Researchers found significant differences in lipid profiles and systolic BP between males and females. Women had a significantly lower systolic BP, lower TC/HDL, and higher HDL compared to men. Investigators concluded that this suggests a gender difference may exist in

CVD risk factors, which may have implications for gender specific interventions. Risk factors were compared between men and women versus between athletes and non-athletes.

It is common that crew (rowers), basketball, track and field throwers, and volleyball athletes are tall and have a large body build including more muscle that places them into the overweight or obese category based on BMI despite sometimes not being classified as over-fat. BMI is used in clinical and health promotion settings to categorize an individual as underweight, normal weight, overweight, obese, or morbidly obese, even though it was designed to assess groups in the public health setting.[13-15] While BMI is simple and reproducible, it does not differentiate between lean body mass and body fat.[16] Ode et al.[17] found that when measuring percent body fat (% BF) in non-athletes and athletes via BOD POD, which uses air displacement plethysmography, and comparing it to calculated BMI values, the current BMI cut points did not accurately correspond to % BF in athletes. BMI misclassified normal fat athletes a large percentage of the time (specificity 0.27-0.66 in all athletes). Optimal BMI cut points for overweight in male athletes, linemen, and female athletes were 27.9 $kg/m^2$, 34.1 $kg/m^2$, and 27.7 $kg/m^2$, respectively, suggesting a need for different BMI classifications in these athletic populations that tend to have more lean body mass and less fat mass than similar BMI non-athletes.

There is little known about the CVD risk factor status in larger female athletes (based on BMI), particularly when compared to less physically active females with similar BMIs but greater levels of adipose and less lean body mass than athletes. It is plausible that the larger athlete that is leaner and has a higher level of aerobic and overall fitness would have a superior CVD risk factor status verses a similar sized (based on BMI) and age sedentary woman. This

hypothesis stems from evidence that has demonstrated that BMI classifications do not correlate well with measures of body composition,[15] and as well as a body of research that indicates that moderate and vigorous PA that expends $\geq$ 1200Kcal/week is effective at elevating HDL levels 2 to 8 mg/dL and decreasing TG levels by 5 to 38 mg/dL.[8] This amount of training may also have an effect on TC and LDL levels, though this has been reported less frequently. This regimen of PA has also been show to improve measures of BP and WC.[8]

Body mass index has often been discounted as an inappropriate measure in selected athlete groups that have increased levels of lean body mass.[17, 18] While some studies have assessed CVD risk factor status of athletes verses sedentary non-athletes, the majority of these studies were conducted with male athletes.[19-21] There have been no studies that have exclusively compared female athletes and non-athletes with a similar BMI classification. For example, does a female athlete with a BMI of 31 kg/m$^2$ with a desirable level of fat, have the same amount of CVD risk as a sedentary college student with a BMI of 31 kg/m$^2$ who has more adipose? It is possible that the athlete has fewer CVD risk factors, but there is little known to date about this growing population of larger female athletes that tend to be leaner than similar size sedentary women.

**STATEMENT OF PURPOSE/AIMS**

The primary aim of this study was to compare the CVD risk factor status of female National Collegiate Athletic Association (NCAA) Division I student athletes (SA) classified by BMI as overweight or obese compared to a group of sedentary student (SS) non-athletes with similar BMI levels. The primary CVD risk factor status analysis was based on a composite CVD

risk score (cCVDs). Additionally, the levels and numbers of the individual risk factors will also be compared between the groups. The cCVDs included WC, mean arterial pressure (MAP), fasting levels of TG, HDL, FBG and C-reactive protein (CRP). Other individual risk factors assessed included systolic and diastolic BP, TC, LDL, TC/HDL, % BF, kilograms of fat-free and fat mass, total daily energy expenditure (TDEE), resting metabolic rate (RMR) and resting heart rate (RHR). Dietary behavior between the two groups and in reference to national heart health recommendations [National Cholesterol Education Program (NCEP) Therapeutic Lifestyle Changes (TLC)] was assessed. Secondary aims included evaluating the influence of TDEE, BMI, % BF, and dietary quality (defined as gm of fiber intake/1000 Kcal) on cCVDs.

**SIGNIFICANCE OF STUDY**

Despite the small sample size and that the subjects in the two groups were not matched based on BMI, this study provided information on CVD risk status in a group of larger female collegiate athletes (SA) compared to a group of sedentary college students (SS) - two populations for which there is little data. There is a gap in the literature for studies that assess risk in female athletes, but particularly those with a BMI at or above 25 $kg/m^2$. There are also few studies comparing risk between female athletes and non-athletes. The cCVDs used in this study has potential as a viable tool to assess CVD risk in a population for which no current risk scores exist (i.e. young adults). This study offered new insights that will be applicable to collegiate institutions. Thirty-five percent of SA and 70.5% of SS had at least two or more risk factors for CVD. This means that all college females, regardless of activity level, should be screened for CVD risk factors associated with heart disease. Student health centers should make an effort to target this age group with regard to heart disease specifically. Proper assessment of

CVD risk in college students may prevent further development of CVD risk factors, which will reduce the morbidity and CVD mortality associated with them.

## HYPOTHESIS/AIMS

The overall hypothesis was that SS individuals would have a less than desirable overall cCVDs. The specific aims of this study were:

Aim 1. To compare cardiovascular risk factor status between SA and SS and with national recommendations.

- Hypothesis 1a (H1a): SA will have a significantly more desirable CVD risk factor status (using a cCVDs*) as compared to SS.
- H1b: SA will have a more desirable fasting lipid profile [TC, LDL, HDL, TG, and the ratio of TC/HDL] compared to SS.
- H1c: CRP levels will be significantly higher (and therefore less desirable) in SA than SS.
- H1d: TDEE will be inversely related to CVD risk in the overall study population and within each study group (SA and SS).
- H1e: SA's RHR will be significantly lower than SS.

*A Z-score approach was used to calculate a cCVDs for each subject. Data included was WC, MAP, fasting TG and HDL, CRP, and FBG. This method is based on methodology for a metabolic syndrome score utilized by Eisenmann et al.[22] to evaluate metabolic syndrome risk. The score was recently validated in children[23] and adults.[24] The standardized residuals (Z-scores) for the individual risk factors were summed to create the cCVDs. Since the standardized HDL is inversely related to cardiovascular risk, it was multiplied by -1 before summing all of the risk factors. A higher score was indicative of a less favorable CVD risk status.

Aim 2. To compare BMI and % BF and cardiovascular risk between SA and SS.

- H2a: SA and SS with a higher BMI will have a significantly less desirable cCVDs relative to lower BMI SA and SS, respectively.
- H2b: SA and SS with a significantly higher % BF will have a less desirable cCVDs relative to lower % BF SA and SS, respectively.
- H2c: SA will have a significantly lower % BF compared to BMI matched SS.
- H2d: SA will have a significantly higher RMR due to higher amount of lean body mass compared SS.

Aim 3. To compare current dietary intakes between SA and SS and relative to the NCEP TLC guidelines.[5]

- H3a: Total fat intake (measured as a percent of total calories (% Kcal)) of SA will be greater than the upper range of the guideline of $\leq$35% total Kcal from fat.
- H3b: SS will have a significantly higher intake of saturated and trans fat (measured as a % Kcal) compared to SA. However, overall neither group will meet the recommendation for $\leq$7% Kcal intake from saturated fat and $\leq$1% Kcal intake from trans fat.
- H3c: SA will have a significantly higher intake of simple sugars (defined and measured as total grams) when compared with SS.
- H3d: SA will have a significantly higher intake of fiber (measured as grams per 1000 Kcal of intake) when compared with SS. However, overall neither group will meet the recommendation of $\geq$12.5gm fiber per 1000 Kcal of intake.
- H3e: SA will have a significantly higher intake of fruits and vegetables (measured as total servings per day) when compared with SS. However, overall neither group will meet the recommendation for $\geq$5 servings per day.

- H3f: SA will have a significantly higher intake of sodium and potassium (measured as total milligrams of intake) when compared with SS. However, overall neither group will meet the recommendation for $\geq$4415 mg of potassium or $\leq$1600mg of sodium daily.
- H3g: The overall caloric intake of SA will be higher than SS.
- H3h: SA and SS with a more desirable dietary quality (measured as a higher intake of grams of fiber per 1000 Kcal of intake) will have a significantly more desirable cCVDs relative to those with a less desirable dietary quality in SA and SS, respectively.

# CHAPTER 2

## LITERATURE REVIEW

The following literature review includes; a) A discussion of cardiovascular disease (CVD) risk status in young adults (particularly females), including collegiate athletes. b) Methods for assessing CVD risk status in humans, with an emphasis on assessment in college athletes. c) Recommendations for macronutrient and micronutrient intake with respect to heart health and CVD risk factor and sports performance. d) Validity and reliability of dietary intake assessment methods, with special attention on food frequency questionnaires (FFQ). e) Methods for evaluating body composition in athletes and the relationship between body composition and CVD risk factors. **Table 1** lists known CVD risk factors and **Table 2** outlines heart health nutrient recommendations.

### 1. CARDIOVASCULAR DISEASE RISK IN YOUNG ADULTS

Contributors to the etiology of CVD in young adulthood include obesity and its associated etiologic factors (diet and exercise). Poor diet and low levels of physical activity (PA) (activity that causes muscle contraction) are increasing in the United States. Only 8.7% of college students consume the recommended five or more servings of fruits and vegetables daily.[25] The percentage of adults 18 years of age and over who engage in regular leisure-time PA has not increased in the past decade and remains at ~37.1% in those 18-24 years of age.[26] In the 2006 National Heart Interview Survey,[5] 62% of adults >18 years of age reported no vigorous activity lasting >10 minutes per session despite recommendations that some proportion of activity be vigorous. The development of CVD is progressive with origins of atherosclerosis in childhood and adolescence, but the majority of CVD events occur during mid-adulthood.[27] In

addition, CVD risk factors tend to track from childhood/adolescence into adulthood, predicting CVD morbidity.[28] This makes it important to study young adults to identify potential CVD risk in order to gain insight into the etiology and prevention of CVD.

Cardiovascular disease risk and metabolic syndrome has been evaluated in football linemen, in part because of their increasing size and also because many are classified as obese according to body mass index (BMI).[9-11] While BMI is often discounted in athletes as an inaccurate measure, researchers have hypothesized that perhaps these athletes in particular are not free of chronic disease risk, despite being active and having a large level of muscle mass. One cross-sectional study measured blood pressure (BP), percent body fat (% BF) (via skinfold measurement), waist circumference (WC), fasting insulin, fasting blood glucose (FBG), high-density lipoprotein (HDL), total cholesterol (TC), triglyceride (TG), C-reactive protein (CRP), and Hemoglobin A1c to assess the presence of metabolic syndrome.[9] Results indicated that 48.6% of Division I, II, and III linemen met criteria for metabolic syndrome, showing that although athletes are assumed to be protected from CVD risk factors, there is a high incidence of metabolic syndrome and biomarkers for heart disease in linemen. Two studies compared risk factors in NFL players to individuals in the general population (obtained from the Coronary Artery Risk Development in Young Adults (CARDIA) study[10] and the National Health and Nutrition Examination Survey (NHANES).[11] Tucker et al.[10] found that the NFL population had a CVD risk profile similar to the general population, while Selden et al.[11] found metabolic syndrome in current NFL players – particularly lineman. In addition, elevated FBG and a BMI ≥

30 kg/m$^2$ were significantly more prevalent in the NFL players verses similar aged males from NHANES.

A cross-sectional investigation in Nigeria compared plasma lipid profiles, and three CVD predictor ratios in fourteen male healthy runners (mean age 22 +/- 4 years-old) with fourteen male healthy non-athletes (mean age 25 +/- 5 years-old).[21] The two groups were matched for age, height, weight, and BMI. Dietary intake was not directly assessed. Mean levels of TC and low-density lipoprotein (LDL) were significantly lower in the athletes than in the controls ($p<0.01$). Overall, results indicated that exercise appeared to decrease the ratio of TC to HDL (TC/HDL) in athletes by lowering LDL, while the HDL was unaffected. In contrast to these results, Olchawa et al.[29] found that, when comparing 25 endurance-trained male athletes with 33 age-matched males who enjoyed an active lifestyle, plasma HDL was higher in the athlete group ($P<0.001$). Dietary intake was not assessed. Another study on males who had been sedentary for at least the previous two years examined the chemical composition of LDL (i.e. lipid and protein components, particle diameter, molecular weight and density) versus only LDL concentration.[20] The authors hypothesized that "alterations in the structure and/or subpopulation distribution of plasma lipoproteins could play a role in the development of coronary heart disease (CHD)." A three-day dietary intake was assessed before and during training, and no significant differences were identified including changes in kcal intake. Researchers found that a 14-week endurance-oriented exercise training program led to LDL chemical composition changes, including an increase in the lipid-to-protein ratio, primarily via an increase in LDL free cholesterol content, as well as changes in increases in LDL molecular weight and particle diameters. These changes were associated with a reduction in adiposity, basal plasma insulin and glucose concentration, fat

mass, and plasma TG concentration. Calculated plasma LDL concentrations remained unaltered (3.49 $\pm$ 0.24 versus 3.65 $\pm$ 0.23 mmol/L). Researchers also noted these changes were in contrast to the cholesterol-poor, protein-enriched LDL particles apparent in individuals with CHD and/or at risk for CHD. Change in LDL chemical alteration, however, were related to exercise-induced weight loss, including a reduction in fat mass and BMI. This provides additional evidence for the cardio-protective effect of long-term PA, particularly if it results in a reduction in BMI and fat mass.

Few studies have evaluated CVD risk status in female athletes. Orri et al.[12] conducted a cross-sectional investigation assessing CVD risk in a small sample of 30 male and female college students and intercollegiate athletes. They found that CRP was significantly correlated with BMI and percent body fat, and that WC and FBG were significant predictors of CRP. In addition, significant differences in lipid profiles and systolic BP between males and females were observed. Women had a significantly lower systolic BP, lower TC/HDL, and higher HDL compared to men. Investigators concluded that this suggests a gender difference may exist in CVD risk factors, and this may have implications for gender specific interventions. Risk factors were compared between men and women versus between athletes and non-athletes.

## 2. ASSESSMENT OF CARDIOVASCULAR DISEASE RISK

Many methods have been developed to measure and assess cardiovascular risk. **Table 1** lists known risk factors for CVD.[29,30,112,114-116] Associations have been found between a healthier diet (e.g. higher nutrient density, higher intake of fiber and lower intake saturated fat and simple sugars), greater PA, avoidance of smoking, and maintaining a lean body mass (proportionately higher amount of muscle and lower amount of body fat or adipose tissue) and reductions in CVD risk factors and events.[30] Using multivariate equations, composite scores

have been created from these risk factors in order to estimate the absolute likelihood of developing a major cardiovascular event (fatal and non-fatal stroke and myocardial infarction) within the next ten years.[30] The three major risk categories include: high-risk (20% or greater), intermediate-risk (10-20%), and low-risk (<10%) individuals. These scores can also be used to evaluate change in risk over time, as well as identify cross-sectional correlations across populations. Many 10-year risk score assessments are currently available. The 1998 Framingham Risk Score[30] has been most extensively used and has been validated on numerous populations over several decades of follow-up. Equations derived from the Framingham Heart Study use weighted risk factors: age, sex, systolic or diastolic BP, serum TC and HDL, and the presence and absence of left ventricular hypertrophy, diabetes mellitus (DM), and cigarette smoking.[31] Other examples of measures of CVD risk include the Dundee coronary risk-disk (developed in the United Kingdom),[32] as well as equations resulting from analysis of data from the British Regional Heart Study,[33] the Prospective Cardiovascular Munster Heart Study (PROCAM),[34] and the Systematic Coronary Risk Evaluation (SCORE) project (conducted in Europe).[35] However, as mentioned above, both the 1998[31] and the newer 2000[36] version of the Framingham Risk Score have been most widely validated across populations. The newer version of the Framingham 10-year risk scores can predict 10-year global CVD risk and specific CVD endpoints (e.g. CHD, stroke, heart failure, and peripheral arterial disease).[30]

***2a. Causal Risk Factors***

Completing a risk assessment includes identifying causal, predisposing, and conditional risk factors. Assessment of causal factors includes a smoking history, measuring BP and blood

lipids levels. To assess diabetes FBG level, fasting insulin level and Hemoglobin A1c may also be used. Age of the individual is a good initial indication of plaque burden as a risk factor.[36] Hypertension (BP > 140/90 mg/dL) is clinically the strongest predictor of coronary attack (though physical inactivity is strongest when assessing communities).[37] Pre-hypertension is often identified for prevention of hypertension; it is defined as a systolic BP from 120 to 139 mm Hg or a diastolic BP from 80 to 89 mmHg.[38]

***2b. Predisposing Risk Factors***

History and physical examination are typically used to assess predisposing factors such as overweight and obesity, physical inactivity, family history of premature CHD, and insulin resistance. Overweight and obesity are often assessed by body weight, BMI, and WC. A WC of > 40 inches in men and > 35 inches in women usually indicates significant insulin resistance, while the calculated number for BMI gives an approximation of total body fat.[36]

Many studies have examined the associations between anthropometric measures and risk of incident CVD. Gelber et al.[39] found that when comparing measures of BMI, WC, waist-to-hip ratio, and waist-to-height ratio to assess risk of incident CVD, the differences among the indexes were small and likely not clinically significant. Researchers noted that, among men, waist-to-height ratio demonstrated the strongest gradient in the association with CVD, followed by WC, BMI, and waist-to-hip ratio. Among women, waist-to-height ratio and WC demonstrated the strongest gradient in the association with CVD, with weaker associations for BMI and waist-to-hip ratio. Lafortuna et al.[40] identified that older age and excessive abdominal fat (via waist-to-hip ratio) appear to be significant factors in relation to increased cardiovascular

risk, independent of BMI. Freiberg et al.[41] investigated the relative contributions of BMI and WC for identifying risk of CVD events and found that in overweight women, larger WC was an independent predictor of CVD incidence longitudinally [multivariable-adjusted odds ratio (OR) of 1.86 per standard deviation increment in WC, 95% confidence interval (CI) = 1.03–3.36, $p<0.04$]. However, among normal weight or obese men and women, as well as in overweight men, WC did not substantially add to prediction of risk of vascular events. An analysis of data from the NHANES III demonstrated that WC was more predictive of cardiovascular risk than BMI, but the cutoff at which positive predictive values exceeded negative predictive values was 98 cm for men and 87 cm for women.[42] Other studies have found BMI to be a strong risk factor for total and ischemic stroke, hemorrhagic stroke, and ischemic heart disease.[43, 44] In contrast, Rexrode et al.[45] found that higher waist-to-hip ratio and greater WC were independently strongly associated with increased risk of CHD, with waist-to-hip ratio being a slightly better measure than WC. A recent review of evidence concluded that there is no clear agreement as to whether measures of central obesity (WC and waist-to-hip ratio) are more strongly associated with cardiovascular morbidity and mortality versus BMI.[46] Similar results were found when assessing which anthropometric measure was a better discriminator of CVD risk – the discriminatory capability of each measure at identifying those individuals with the highest CVD risk is comparable. Finally, differences in associations between the anthropometric measures and CVD risk were not identified across different ethnic groups. The authors did note, however, that the majority of the evidence for this paper was cross-sectional, and more prospective studies were needed.

***2c. Conditional/Emerging Risk Factors***

Conditional factors such as TG, homocysteine, and coagulation factors may provide additional information about the level of risk for the individual.[47-49] Inflammatory markers, specifically CRP, have been shown to predict future cardiovascular events. Inflammation plays a key role in the initiation and progression of atherosclerosis and development of atherosclerotic events – from lesion initiation to plaque rupture and associated thrombotic complications. In a review of prospective cohort and case-control studies, CRP was shown to be a reliable measure of underlying systemic inflammation.[47] Dhingra et al.[48] identified a high prevalence of common and uncommon inflammatory conditions in individuals with high CRP concentrations. A recent meta-analysis found that CRP concentration is as consistent within individuals during several years as are TC and systolic BP, and that CRP concentration has continuous associations with the risk of CVD.[49] Overall, data suggests that CRP may identify patients not identified by traditional risk factors such as LDL, and may play a role in global risk prediction of CVD because a larger number of individuals at risk will be identified.[47]

There is extensive literature on the synergistic effect of having multiple CVD risk factors.[50-52] In a study of 7,900 men and women, at age 50 those with an "optimal" risk factor burden (defined as BP below 120/80 mm Hg, TC below 180mg/dL, absence of diabetes, and nonsmoker) had a median life expectancy 10 or more years longer than those with two or more major risk factors.[50] In the NHANES II Mortality Follow-up Study,[51] the risk for fatal CHD was 51% lower for men and 71% lower for women with none of three major risk factors (hypertension, current smoker, and elevated TC $\geq$ 250 mg/dL) compared to those with one or more risk factors. Wilson et al.[52] found that the estimated 10-year CHD risk in adults older than

55 years of age increased with the number of risk factors, ranging from 5% in individuals with no risk factors to 37% and 27% in men and women, respectively, with all five risk factors (elevated BP and TC, low HDL, positive for diabetes, and current smoker). The aforementioned studies clearly demonstrate that having multiple CVD risk factors greatly increases risk of CVD and overall mortality.

## 3. NUTRITION AND CARDIOVASCULAR DISEASE RISK

Healthy diet changes have been implicated as preventive measures for CVD.[53,58,59,61-64] According to the bulletin of the World Health Organization[54] from 2005, "the total worldwide mortality currently attributable to inadequate consumption of fruits and vegetables is estimated to be 2.6 million deaths per year. Increasing individual fruit and vegetable consumption to up to 600 grams per day could reduce the total worldwide burden of disease by 1.8 percent, and reduce the burden of CHD and ischemic stroke by 31 and 19 percent respectively". Only 8.7% of college students consume the recommend five or more servings of fruits and vegetables daily.[25] In addition, many college students skip meals, eat on the run, consume energy-dense snacks and meals, and frequently diet.[55] Many modifiable risk factors of CVD are influenced by diet, including obesity and WC, elevated BP, dyslipidemia, elevated CRP, and FBG level.[53] Other dietary factors possibly linked with CVD risk include caffeine, alcohol, and meal frequency.

Typical dietary recommendations include increasing intake of dietary fiber and lowering total intake of dietary fat (with an emphasis on saturated fat (SFA) and cholesterol), sodium, and simple sugars.[53] This is often achieved by teaching individuals to read food labels, control

serving sizes, and select foods with high nutrient density and low energy density. In a review of 27 studies,[56] alteration in total dietary fat intake had small effects on total mortality (rate ratio 0.98), cardiovascular mortality was reduced by 9%, and cardiovascular events by 16%. The review noted that trials with two years follow up provided stronger evidence of protection from cardiovascular events. The type of fat ingested is also important. Saturated fat and trans fat (TFA) are the most atherogenic, while monounsaturated (MUFA) and polyunsaturated (PUFA) fat are associated with decreased CVD risk.[53] Saturated fat and TFA increase TC and LDL levels in a dose-dependent manner.[57] Although SFA raises HDL, TFA decreases HDL and increases the TC/HDL ratio in a dose-dependent manner. Polyunsaturated fats lower TC and LDL, while MUFAs have a neutral effect on these lipid values. Reduction of TC, LDL, and the TC/HDL ratio collectively reduces CHD/CVD risk.[57]

It is often difficult to relate sugar consumption directly to CVD due to many possible confounders, but short-term studies show adverse effects of sugar consumption on HDL and TG levels, which could accelerate atherosclerosis.[58] In addition, increased consumption of high-sugar foods, which are very calorically dense, could lead to weight gain and replace more nutrient-dense options. Research of sugar intake typically analyzes intake of disaccharides, naturally occurring sugars, or added sugars.[58, 59] Common disaccharides include sucrose (found in sugar beets, honey, and corn syrup), lactose (found in milk products), and maltose (from malt). Typically, naturally occurring sugars refer to sugar in whole fruit, vegetable, and milk products. The term added sugars refers to refined sugars (e.g., sucrose) in soft drinks and incorporated into food, fruit drinks, and other beverages.

Dietary fiber is not digested or absorbed in the human small intestines. There is an overall inverse relationship between dietary fiber intake and risk of cardiovascular disease and myocardial infarction.[60] In one study, dietary fiber better predicted CVD risk factor status than having a low intake of saturated fat (which included analysis of LDL, BP and insulin values).[61] In the Nurses' Health Study,[62] women in the highest quintile for fiber intake (median 22.9 gm/day) had an age-adjusted relative risk for major coronary events 47% lower than women in the lowest quintile (11.5 gm/day). Dietary fiber has recently been indicated as a possible link between diet and inflammation. A 2003 study relating fiber consumption to CRP levels was among the first to show a link between dietary intake and inflammation using a nationally representative sample (NHANES 99-00).[63]

## 4. EFFECT OF EXERCISE ON CARDIOVASCULAR DISEASE RISK FACTORS

The AHA classifies physical inactivity as a major risk factor for CVD.[64] It is well-noted that moderate-to-vigorous physical activity (MVPA) improves cardiorespiratory fitness and reduces cardiovascular-associated morbidity and mortality.[65] A curriculum on the prevention of CVD noted, "for each one metabolic equivalent (MET) increase in exercise capacity, there appears to be an 8-17% reduction in mortality. In addition, an approximate 1,000-Kcal/week increase in activity provide an equivalent survival benefit as increasing cardiorespiratory fitness by one MET."[65] Alternatively, data suggest that if people expend <1,000 Kcal/week with MVPA, their risk for developing atherosclerosis is multiplied.[19] In addition, regular aerobic exercise has been found to delay the usual loss of circulatory function with increasing age.[19]

Physical activity levels are challenging to measure, and so are often not included in risk assessments. In spite of this, increasing physical activity is still a target of prevention.[64] In 1997, The AHA[66] released a recommended exercise regimen intended to reduce the risk for CVD. The statement indicated that the activity should be done ideally for 30-60 minutes daily 4-6 times weekly, or 30 minutes most days of the week. Examples of exercise included aerobic activities such as bicycling (stationary or routine), walk-jog protocols, and swimming. It was also noted that the exercise regimen should include resistance training 2-3 times weekly. The statement emphasized the importance of individualized programs to establish exact time, type of exercise, and intensity requirements.

When assessing the effect of exercise quantity, type, and intensity on CVD risk, Williams[7] found that male and female recreational runners who ran at a greater intensity had lower BP, TG, TC/HDL ratio, BMI, and WC. Running velocity had a 2.8 times greater calculated effect on diastolic BP, and a 13.3 and 5.7 times greater calculated effect on systolic BP in men and women, respectively. However, running distance had a more than 6-fold greater effect on HDL than running velocity in both sexes. Thresholds for observable changes occur at training volumes of 15-20 miles per week of brisk walking or jogging and expending between 1200-2200 Kcal/week.[8] This exercise regimen is also associated with 2-3mg/dl increases in HDL and 8-20mg/dl decreases in TG, and the effect is similar in both sexes.[8]

However, those who are physically active, particularly professional and collegiate athletes, may actually be at higher CVD risk due to increased production of free radicals and elevated inflammation caused by frequent, vigorous exercise.[67] Although, this has not been

extensively researched at this point, and is often difficult to pinpoint what factors cause cardiovascular events.

## 5. NUTRITION AND PERFORMANCE AND CARDIOVASCULAR HEALTH: RECOMMENATIONS AND GUIDELINES

Athletes are a unique population with unique nutritional requirements. It is imperative that athletes meet these requirements to maintain body weight, replenish glycogen stores, and provide adequate nutrients for recovery, repair, and building of body tissue. **Table 2** outlines nutrient recommendations reflecting: American College of Sports Medicine/American Dietetic Association (ACSM-ADA) position statement, and the National Cholesterol Education Program (NCEP) Therapeutic Lifestyle Changes (TLC) (reflecting the 2006 update), which is endorsed by the American Heart Association (AHA) for the prevention of CVD.[5, 68, 69]

A comparison of the two recommendations shows similarities for most categories. The ACSM and ADA recommend macronutrients per kilogram verses by percent of calories.[70] This type of guideline allows recommendations to be better tailored to the individual athlete to insure needs are appropriately met with regard to body size, sport, and position. For example, 5-7 gm/kg/day is recommended for general training needs, and 7-10gm/kg/day is recommended for the increased needs of endurance athletes. Because caloric intake of athletes tends to be higher due to increased needs as compared with the general population, fiber intake is often better compared when expressed per 1000 kilocalories of intake (e.g. 12.5 gm per 1000Kcal intake for females) instead of as an absolute number (e.g. 25 gm daily).

## 6. MEASUREMENT OF DIETARY INTAKE: GENERAL POPULATION AND IN ATHLETES

There are a variety of methods for assessing dietary intake. This section will discuss diet records, 24-hour recalls, and FFQ and will include a discussion of strengths and limitations of each method.

There are a variety of diet records, including weighted records (where subjects weigh each item of food and drink at time of consumption), estimated records (items described in cups, spoons, etc.), and menu records (food items listed with portion sizes omitted).[71] When completing any type of diet record, respondents record food or drink as they are consuming it. In theory, this helps minimize errors related to memory recall. Therefore it can give a more complete picture of typical intake. However, it can be burdensome to the respondent due to the amount of time required to fill it out each day. Using this method requires individuals to be very committed and reasonably literate, especially if it is used for multiple days (typically 2-3 days). In addition, it is possible that the person's beverage and food choices will change when they know their intake will be reviewed and evaluated. Also, it is not always ideal to use with children, as a parent or other adult will need to record for them. A review by Hill et al.[72, 73] found that in studies where a person was responsible for filling out intake for another individual (such as parents of young children), energy intake generally corresponded well with energy expenditure determined by doubly-labeled water. However, when subjects reported their own intake, energy intake was generally under-reported when compared with energy expenditure. A review paper concluded that a small number of days of diet information does not represent an individual's longer term usual intake with any precision because an individual's diet is sufficiently variable from day to day.[74] It noted a minimum of seven to as many as ten days is

ideal in order to be within +/- 20 percent of the true usual macronutrient intake; for micronutrient intake to be truly reflective, it appears up to 15-20 days are needed.

During the 24-hour recall method, individuals recall all food and beverage intake over the past 24 hours. Portions are quantified, as in estimated diet records. Often a "multiple-pass method" is used in which the respondent is asked to recall the same day many times in a row, to assure all foods were reported (including condiments and beverages). The most ideal 24-hour recall includes use of three-dimensional models to help respondents estimate portion sizes and assure consistency when using this method to study groups verses individuals. Studies addressing validity of this method have shown some mixed results.[75, 76] In one investigation, researchers compared average daily protein intake estimated from a 24-hour dietary recall interview with protein intake estimated from urinary nitrogen excretion in 24-hour samples.[75] Results indicated that men over-reported protein intake by 12-19% while women reported a dietary intake almost exactly in agreement with urinary nitrogen levels. Another study assessing total energy intake found that overall energy intake tended to be underestimated in the vast majority of the sample population, although to varying degrees.[76] Underreporting was more prevalent in women than men. Interestingly, BMI and age were related to underreporting in this study, indicating that a person's BMI and age may affect how the level to which the person is underreporting. The 24-hour recall method has been used previously in NHANES surveys for children and infants as well as adults.[73] Based on studies of NHANES and other research, this method overall has been shown to provide the most valid data on mean intake of both groups and individuals over the prior 24 hour.[74, 77] Comparison of a 24-hour recall questionnaire with a 3-day record showed good agreement, with Spearman's correlation coefficients greater than 0.35

for all density measurements, with the highest correlation coefficients of about 0.60 for alcohol and dietary fiber intake.[77] A limitation of this method is that it does not necessarily reflect long-term or typical eating habits.

Food frequency questionnaires instruct individuals to indicate their frequency of consumption of each of a list of foods or food groups.[74] Most forms are designed for self-completion and may be sent by mail. By using this method, it is easier to get an idea of typical eating habits. The Gladys Block FFQ[71] is an example of a commonly used questionnaire. A limitation is that these questionnaires tend to be long, and may be burdensome for the respondent. However, these questionnaires are short compared to a 7-10 day food record. They also require a respondent to be literate, depend on memory and require respondents to have the ability to convert varying dietary patterns into frequency of consumption. In addition, foods specific to certain cultures or ethnicities may not be listed on standard food frequency questionnaires, so this should be addressed as appropriate. One study found correlations between a self-administered FFQ (that did not attempt to ascertain the respondent's portion sizes) and diet record nutrient estimates for the prior year were approximately 0.45 without adjustment for reported caloric intake and about 0.53 after adjustment for calories.[74] Researchers concluded that agreement in this range was "greater than chance and indicates a fair ability to categorize individuals at the extremes. For example, of individuals in the lower quintile by diet record, 40-50 percent were in the lowest quintile by FFQ". This is important to keep in mind when using FFQs.

Assessing dietary intake in athletes presents unique challenges. Athletes are a unique population with specialized needs.[70] Under-reporting of intake is widespread among this group,

so this should be assessed carefully.[71, 78] When working with athletes, the most feasible solution to this is to compare dietary reports with expected energy expenditure.[71] When energy requirements are expressed as a multiple of basal metabolic rate (BMR) (using PA level), some of the variation due to age, sex, and weight can be eliminated. Basal metabolic rate can be estimated from weight, or weight and height, using equations. Energy intake can then be expressed as a ratio of energy intake to BMR for comparison. Using this method provides a way to check if there is true underreporting occurring; this will help improve the overall quality of the data. Intake issues specific to athletes include adequacy of standard portion sizes, frequency of snacking, fluid intake, supplement use, weight-control practice, and seasonality of sport activities and food consumption.[78] One must also consider each separate sport, the training status, the level of competition, and unique nutrition-associated beliefs and dietary practices. Regardless of the method used, these areas should be addressed to meet the needs of the athlete. A major advantage of the 24-hour recall with athletes is that it involves minimal subject burden, can be scheduled around other daily activities, and can by conducted in 15-30 minutes face-to-face or by telephone.[71] However, intake may vary even more for an athlete whether the past 24 hours involved typical training, competition, or rest. Therefore, a single 24-hour diet recall may be inadequate. One major limitation of using a FFQ with athletes is that the questionnaires often contain foods not reflective of the athletic population, as their eating patterns tend to deviate from the mainstream. As with the general population, it is sometimes most useful to use more than one type of dietary assessment technique when working with athletes. Instead of evaluating actual food eaten, a common practice used with athletes to monitor caloric balance is to have the athlete weigh himself at the same time of day for two or three days per week. Monitoring the

weight trend throughout the week can help the athlete to understand when energy balance is less than desirable.

When deciding which method to use, variables such as cost and time also need to be considered. Although a 24-hour recall is the most reliable, it may not be feasible to perform this in a study that has a very large sample size. One must also keep in mind desired outcomes. A food frequency questionnaire might be much more appropriate when relating intake to health outcomes, as this gives a better picture of long-term or typical intake.[74] Any measure chosen will have a given amount of errors of precision (repeatability, reliability, reproducibility) and validity. Both underestimation and overestimation may occur.[78] Therefore, methods should be assessed and chosen carefully; proper training of all interviewers will help decrease some error.

## 7. DIET CHARACTERIZATION OF THE ATHLETE AND NON-ATHLETE

While it is important to have guidelines for intake, along with valid and reliable methods for measurement of that intake, this does not mean that individuals will meet those guidelines. Because nutrient requirements tend to be higher and purposeful restriction to meet weight goals is common, athletes' intake is often suboptimal.[79] Determination of which nutrients are deficient, however, is gender and sport specific. Limited time and access to grocery stores or facilities for food preparation are also obstacles to athletes meeting guidelines.[79] Research indicates that while male athletes typically achieve carbohydrate intake within the recommended range, female athletes (particularly endurance athletes) are less likely to meet these guidelines.[80] This is often due to an effort to restrict in order to maintain or achieve low levels of body fat. Inadequate intake is most often associated with females participating in aesthetic sports that are scored based on physical appearance or in male athletes who are wrestlers trying to make

weight.[79] Marginal intake of carbohydrate and fiber has been reported in many female collegiate teams.[81] One study reported that mean energy intake of both female field hockey players and cross country runners was less than recommended for their mean body weight and age in-season and post-season.[82] This study noted that the cross country runners were consuming high carbohydrate/low fat diets; protein consumption was higher than recommended. Alternatively, Steen et al.[83] found that 16 female heavyweight rowers consumed low carbohydrate/high fat diets, with satisfactory protein consumption. Another study showed that only 15% to 26% of male and female athletes assessed had adequate intake of carbohydrate and protein, respectively based on recommendations for athletes.[79] Males derived a significantly greater proportion of energy from fat compared to females, actually exceeding the Dietary Guidelines for fat, SFA, cholesterol, and sodium. The desire in female athletes to lose weight was associated with decreased energy and macronutrient consumption. Interestingly, the study showed no significant differences in dietary behaviors among sports assessed. Clark et al.[81] found that female soccer players failed to meet carbohydrate recommendations, while protein and fat intakes were above minimum recommendations. Researchers noted that foods higher in fat and protein displaced more carbohydrate-rich and nutrient-dense foods in the athletes' diets.

Non-athletic college students clearly have different nutritional needs compared to their athletic counterparts. Their food choices are often also based on convenience, due to limited time and resources. Based on data from the 1995 National College Health Risk Behavior survey,[84] 26.3% of 4600 undergraduate students surveyed were eating ≥ 5 servings of fruits & vegetables daily and 78% were eating ≤ 2 high-fat foods daily (defined as eating "≤ 2 servings of

hamburger, hot dogs, sausage, French fries, potato chips, cookies, doughnuts, pie, or cake yesterday"), as recommended by the American Cancer Society. Another study involving 760 freshman and sophomore students found that 30% of respondents surveyed were eating ≥ 5 servings, or 2 ½ cups, of fruits & vegetables daily, 41% ate ≥ 3 fried foods during the previous week, and 46% ate ≥ 3 high-fat fast foods during the previous week.[85] From these, one can conclude that non-athletic college-students' diets may be too high in fat and the majority are not achieving recommend intakes for fruits and vegetables.

## 8. MEASUREMENT/USE OF BODY MASS INDEX

Body mass index is used as an indicator of obesity and fat distribution that is often used as an alternative to actual measurements of adipose tissue mass in individuals or populations. It was developed as an attempt by the 19$^{th}$ century mathematician Lambert Adolphe Jacques Quetelet to derive a measure of adiposity by adjusting body weight for individual differences in stature.[15] It assumes, however, that after adjusting body weight for stature, all subjects have the same relative fatness regardless of age, sex, or ethnicity. Limitations with usage of this indicator are recognized, but it is these effects (sex, ethnicity, and age) on the relationship between BMI and body composition that is not as well recognized. The Expert Panel on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults[86] classify a BMI of ≤ 18.5 kg/m$^2$ as underweight, 18.5-24.9 kg/m$^2$ as normal weight, 25-29.9 kg/m$^2$ as overweight and a BMI ≥ 30 kg/m$^2$ as obese. Obesity is classified into Stage 1 (BMI 30-35 kg/m$^2$) and Stage 2 (BMI >35 kg/m$^2$). Therefore, the cutpoint for where adults are considered to have excess percent fat is a BMI >25 kg/m$^2$.

There is, however, much variability in the individual use of BMI, as some individuals with low BMIs have as much fat as those with high BMIs.[13] Therefore, it is often a poor indicator for body fatness. A review of studies indicated that BMI is moderately strongly correlated (30-50%) with fat-free mass, but is more strongly correlated (60-90%) with fat-mass.[13] Body mass index is also strongly correlated (80-85%) with measured waist circumference. In the ten-country European Prospective Investigation into Cancer and Nutrition (EPIC) prospective study,[14] using WC improved the ability of BMI to predict vascular and all-cause mortality. A recent study by Romero-Corral et al.[16] assessed metabolic dysregulation and risk of cardiovascular mortality in subjects with a normal BMI, but high body fat content (which researchers categorized as normal weight obesity - NWO). Compared with those with low body fat, the prevalence of metabolic syndrome in subjects with NWO was four-fold higher (16.6% versus 4.8%, $p<0.0001$). Subjects with NWO also had higher prevalence of dyslipidemia, hypertension (men) and CVD (women). Notably, women with NWO showed a 2.2-fold increased risk for cardiovascular mortality compared to the low body fat group.

A separate issue is that at different ages, the same levels of BMI correspond to different amounts of fat and fat-free mass.[11,13] Studies show that older adults have a lower amount of fat-free mass (on average) compared with younger adults, due to the loss of muscle mass with age. Therefore, an elderly individual matched for BMI with a young adult is likely at much higher chronic disease risk due to a proportionately smaller amount of fat-free mass compared with the young adult.[15] As a result of these and other issues not mentioned here, risk for chronic diseases may be misestimated using BMI.[13]

Regardless of these limitations, a review of numerous studies indicated that increased weight, relative weight, BMI, and skinfold (SF) thicknesses are positively associated with hyperinsulinemia, hyperglycemia, hyperlipidemia, and hypertension, increasing risk of CVD and type 2 DM.[13] Another review established high BMIs as a risk factor for ischemic heart disease, stroke, and cancers of the large intestine, kidney, endometrium, and breast (post-menopausal).[14] An asymmetric "U"-shaped relationship is assumed between BMI and chronic disease morbidity and mortality risk. This chronic disease risk increases with larger BMI values due to associated excess body fatness. However, it is unclear if increased risk may also be caused by increased fat-free mass (muscle). Increased chronic disease risk at lower BMI levels appears to be due primarily to decreases in fat-free mass; it is unclear how much of the risk in this group might also be due to loss of fat mass.[13] In a collaborate analysis of data from almost 900,000 adults,[14] overall mortality was lowest at a BMI of 22.5-25 $kg/m^2$ in both sexes and at all ages. Above this, each 5 $kg/m^2$ increase in BMI was associated with ~30% higher all-cause mortality (40% for vascular). Below 22.5-25 $kg/m^2$, the only inverse association with BMI was for smoking-related respiratory disease (including cancer). For stroke, being in the upper range for BMI is associated positively with ischemic, hemorrhagic, and total stroke; this is related to the effect of BMI on blood pressure. Being in the lower BMI range, however, is actually inversely related to risk of having a stroke.

***8a. Use of Body Mass Index in Athletes***

The classification categories of BMI are derived from cutpoints obtained from the general population. Therefore, applying these same cutpoints to athletes and young adult non-athlete

might be misleading. Because athletes tend to have more fat-free mass, this added weight might misclassify them as overweight or obese. As a result, it might be more appropriate to use a measured percent body fat verses a calculated BMI.[17]

One study by Ode et al.[17] assessed the relationship between the BMI and percent fat of collegiate athletes and college-aged nonathletes. For this study, body fat was measured by air displacement plethysmography using the BOD POD version 1.69. Researchers found that a BMI $\geq 25$ kg/m$^2$ was not an accurate predictor of overfatness in college athletes and nonathletes. A low specificity indicated that BMI misclassifies normal fat individuals as overweight a large percentage of the time. The exception to this was with female college nonathletes in which a high specificity and low sensitivity suggested that a large percentage of overfat individuals were classified as normal weight by BMI. The researchers suggested that the current BMI cut point for overweight ($\geq$25 kg/m$^2$) should be increased to limit misclassifications within the athletic population. However, in female nonathletes, a BMI cut point of 25 kg/m$^2$ may be too high.

## 9. BODY COMPOSITION: METHODS OF ASSESSMENT

Body composition is affected by sex, race, age, genes, and environment (altitude, climate), as well as behavioral (diet, exercise, tobacco, and alcohol) factors.[13] Therefore associations between health and longevity are likely to depend on these influencing factors. Over time, improvements in body composition models and in laboratory and field measurement techniques have lead to a better understanding of the associations between body composition, health, and disease. With this, there has been an increasing understanding that the location of body fat may be just as important as the total amount of fat. Centralized adiposity distribution, characterized by more fat on and within the trunk than extremities is associated with risk of

chronic diseases independent of level of fatness. On the other extreme, lack of body fat has typically not been viewed as a risk factor for chronic disease, unless it is accompanied by a reduction in fat-free mass – though this is often considered a consequence rather than a cause of disease.

Many methods have been developed to either directly or indirectly measure body composition. Laboratory techniques are often used as reference methods in clinical and research settings. Common laboratory methods include hydrodensitometry, air discplacement plethysmography (e.g. BOD POD), isotope dilution, and dual-energy x-ray absorptiometry (DXA).[87] Multicomponent models also exist, which combine several laboratory methods. More costly laboratory methods include neutron activation, computed tomography, and magnetic resonance imagining (MRI); these are infrequently used due to cost and lack of availability.

While the more valid methods are laboratory-based, they also require more training, and are more time-consuming, inconvenient and costly than field methods.[87] The most common methods used include bioelectrical impedance analysis (BIA), near-infrared interactance (NIR), SF, and athropometric circumference measurements.

Each field and laboratory method mentioned above has both strengths and limitations. As a general rule, each method is only as good as the measurement technique and prediction or conversion formula applied.[87] Following standards and protocols is essential to limit measurement error. This review will focus on use of BIA. Bioelectric impedance analysis involves measuring the impedance (Z) to the flow of a low-level electrical current introduced into the body of the client at a fixed frequency. It is based on the idea that lean tissue, which contains large amounts of water and electrolytes, is a good electrical conductor, while fat, which

is anhydrous, is a poor conductor. Therefore, the higher amount of fat-free mass and total body water an individual has, the less resistance to the flow of the electric current and a lower Z value. Bioelectric impedance analysis measurements of the body are converted by the instrument to estimate percent body fat and provided as a direct digital instrument readout.[88]

Bioelectric impedance analysis is a quick method of body composition assessment that is low cost and does not require a high degree of technician skill. In 1996, Houtkooper et al.[89] concluded that, following extensive review of the literature, "with proper standardization of methods, instrumentation, and subject preparation, this noninvasive body composition assessment approach can quickly, easily, and relative inexpensively provide accurate and reliable estimates of fat-free mass and total body water in healthy population". This method is especially ideal in obese and/or older populations where it can be difficult to obtain accurate SF measurements.[87] A 2005 study by Daniel et al.[88] found that, when compared to four SF protocols, three girth measurement protocols, and one near-infrared instrument, two "whole-body" BIA instruments provided the most accurate and reliable estimations of percent body fat in 121 subjects aged 21 to 51 years. Researchers noted, however, that this was "whole-body" BIA verses instruments intended to measure between the wrists (upper body) or between the feet (lower body). Therefore, if the latter instrument is used, they concluded that it would eliminate the ability of BIA to account for differences in body fat distribution, as is observed with the "whole-body" BIA. A limitation of BIA is that it is influenced by variability in the distribution of fluid between intra- and extra-cellular spaces and in the ionic concentrations of these compartments.[13] In other words, the individual's state of hydration can influence the results. This is especially important to be aware of when working with athletes, whose hydration levels

fluctuate often throughout the day. To control for this, a standard should be in place for hydration levels prior to BIA testing.

## IMPLICATIONS OF LITERATURE REVIEW

The literature review included a summary of various methods, variables and statistical procedures used to assess CVD risk. The majority of the methods used to assess an overall CVD risk factor score that have been developed to date were developed for older populations.[29,30,32,34] These scores are not applicable to the college population we will be assessing. Therefore, a continuous CVD composite score was used that was originally developed for assessing CVD risk factors and metabolic syndrome in children.[23] In addition, this review revealed there is a small body of literature on the CVD risk factor status in athletes, most of which has been conducted with overweight and obese male athletes. Few studies have investigated CVD risk with overweight and obese female athletes (based on BMI), and in particular there is limited research comparing sedentary females that are matched based on BMI. Therefore, this study offers new insights on the CVD risk status in sample of larger (overweight to obese based on BMI) female college students and the influence of physical activity level and nutrition behavior.

# CHAPTER 3

## MANUSCRIPT

## INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of death in women, and more women than men have died from CVD since 1984.[1] Among young adults 15-24 years of age, CVD is the third leading cause of death, after malignant neoplasms and accidents.[2] The major risk factors for CVD include high blood pressure (BP), dyslipidemia, diabetes, smoking, and obesity. The prevalence of these and several other CVD risk factors, with the exception of smoking, has increased over the past two decades among women of all ages, racial groups and education levels.[3] The increases in most CVD risk factors is attributed to reductions in physical activity (PA) level[18] and non-heart healthy dietary behaviors including dietary patterns that are high in caloric density, simple sugars, saturated and trans-fat, and low in nutrient density and dietary fiber.[19, 36] In response to these trends, prevention of CVD has become a primary focus of population-based programs.[4, 5] The American Heart Association (AHA)[5] emphasizes *primordial prevention* to prevent the development of risk factors. Specifically, the AHA recommends screening potentially at-risk groups and encourage heart healthy nutrition and PA behaviors to prevent future morbidity.[5]

Multiple studies[6,9-11] have investigated whether college students, specifically athletes, may be one of these at-risk groups. In 1956, Montoye et al.[6] found no differences in life expectancy, cause of death, or the type of death between athletes (defined as a letter winner in a

varsity sport) and non-athletes. The study did not specify if the sample included only men, or women, or both. Recent studies indicate that selected athletes, particularly males with a relatively larger body size, are at increased risk for CVD.[9-11] Buell et al.[9] found that 48.6% of collegiate football lineman exhibited metabolic syndrome based on players that had $\geq$ 3 of the following CVD risk factors: elevated resting BP, waist circumference (WC), fasting blood glucose (FBG), high-density lipoprotein (HDL), or serum triglycerides (TG). Selden et al.[11] found a similar prevalence of metabolic syndrome in National Football League (NFL) players verses a similar age population from the National Health and Nutrition Examination Survey (NHANES) (6% in NFL versus 10% in NHANES; p=0.355). Although, the prevalence of elevated resting BP and FBG were significantly higher in the NFL players verses the NHANES sample.

While studies have been conducted on the CVD risk factor status between male athletes and non-athletes,[9-11, 19-21] few studies have evaluated the CVD risk factor status in female athletes, and in particular larger-sized female athletes' compared to less physically active females with a similar body mass index (BMI; height in meters squared/weight in kgs). While BMI is relatively simple to obtain and reproducible, a limitation is it does not differentiate between lean body mass and body fat.[16] Ode et al.[17] found that BMI cut points for overweight (25 kg/m$^2$) and obesity (30 kg/m$^2$) did not accurately correspond to levels of body fat associated with overweight or obesity in male and female athletes. Athletes classified with normal levels of body fat were misclassified by BMI a large percentage of the time (specificity 0.27-0.66 in all athletes). Optimal BMI cut points for overweight in male athletes, linemen, and female athletes

were 27.9 kg/m$^2$, 34.1 kg/m$^2$, and 27.7 kg/m$^2$, respectively, indicating a need for different BMI classifications of overweight for these populations.

Given that level of body fat (adiposity) is related with CVD risk factors,[14] it is plausible that a larger athlete that is leaner and/or has a higher level of aerobic and overall fitness would have a superior CVD risk factor status verses a similar sized (based on BMI) and aged sedentary woman. For example, does an athlete who has a BMI of 30 kg/m$^2$ but a desirable percent body fat have a lower CVD risk than a sedentary non athlete college student with the same BMI but more adipose? The primary aim of this study was to address this question by comparing the CVD risk factor status of female National Collegiate Athletic Association (NCAA) Division I student athletes (SA) classified by BMI as overweight or obese to a group of sedentary student (SS) non-athletes with similar BMI levels. The primary CVD risk factor status analysis was based on a composite CVD risk score (cCVDs). Other individual risk factors assessed included systolic and diastolic BP, TC, LDL, ratio of TC to HDL (TC/HDL), total daily energy expenditure (TDEE), resting metabolic rate (RMR), percent body fat (% BF), kilograms of fat-free mass and fat mass and resting heart rate (RHR) as a marker of fitness. Secondary aims were to assess dietary intake compared to national recommendations,[5] as well as evaluate the influence of TDEE, BMI, % BF, and dietary quality on CVD risk factor status for the total sample as well as separately for each group. Dietary quality was defined by total grams of fiber/1000 Kcal intake because dietary fiber intake is a better predictor of a desirable CVD risk factor status than saturated fat (gm/1000 Kcal) or dietary cholesterol (mg/1000 Kcal) intake.[61, 90, 91]

We hypothesized that SA would have a more desirable cCVDs and a lower number of individual CVD risk factors with the exception of CRP levels. Additional hypotheses included a higher TDEE and a lower RHR and % BF in SA as well as a lower RMR in SS. When comparing dietary intake between the two groups, we hypothesized a higher intake of percent of Calories (% Kcal) from saturated and trans fat in SS and a higher intake of grams of sugar, grams of fiber per 1000 Kcal, total cups of fruits and vegetables, sodium, potassium, and total calories in SA. We hypothesized that neither SA nor SS would meet National Cholesterol Education Program (NCEP) Therapeutic Lifestyle Changes (TLC)[5] recommendations for saturated or trans fat, fiber intake, total cups of fruits and vegetables, sodium or potassium, and that SA would not meet the guideline for % Kcal from total fat. Finally, when assessing the influence of energy expenditure, BMI, %BF, and diet quality we hypothesized that the cCVDs would improve as energy expenditure and dietary quality increased, but worsen as either BMI or % BF increased.

## METHODS

### *Study Design and Sample*

This study was a cross-sectional two group comparison of a convenient sample of 18-23 year-old female SA and SS with BMIs $\geq$25 kg/m$^2$. The protocol was approved by the Michigan State University's Institutional Review Board (IRB) and written informed consent was obtained from each participant. Inclusion criteria for the SS included expending $\leq$ 1000 Kcals per week of moderate and vigorous physical activity (MVPA). This includes planned activity that causes muscle contraction and is > 3 metabolic energy equivalents (METs) and equates to MVPA $\leq$ 3x per week for $\leq$ 30 min each day.[92] This was assessed by verbal questioning during telephone screening and by reviewing TDEE estimated from the Block Adult Energy Expenditure Survey.[93, 94] Exclusion criteria for SA and SS included the use of dietary supplements or

medications that influence the outcome variables. Additionally, individuals with type 1 diabetes or current smokers (≥1 cigarette per day) were excluded. Caffeine consumption was allowed up to the equivalent to ~300 mg of caffeine per day, which is equivalent to approximately three cups of coffee (but not on the day of testing). The SAs who had an injury that did not permit them to train and participate in their sport or SS who had an injury that did not allow them to walk were excluded. Incentives, including a meal gift card and a copy of their CVD risk factor and dietary behavior analysis, were provided to participants who met the inclusion criteria and completed the study.

The SA participants were recruited by researchers and team athletic staff at fall physicals and via emails that included a summary of the study. Thirty-one SA were eligible for the study. Nine SA declined participation due to disinterest and two were ineligible due to injuries, leaving 20 SA who consented and participated. Their mean BMI was 27.5 $\pm$ 3.3 kg/m$^2$ (range 24.7-37.4). After recruiting SA, SS were recruited via email and with flyers posted on campus. Efforts were made to enroll SS participants that had BMI levels that were equal or similar to SA. One hundred sixty-eight SS expressed interest in the study, but after phone and email screening and further explanation of the study, only 19 SS consented to the study. Known reasons for not participating include time commitment, disinterest, and not meeting all inclusion criteria. Two of the 19 SS subjects were excluded due to BMI levels (both significantly higher than the SA mean BMI), leaving a final SS group of 17 subjects with a mean BMI of 30.0 $\pm$ 4.4 kg/m$^2$ (range 26.0-39.6). The total sample (N= 37) had a mean BMI of 28.6 $\pm$ 4.0 kg/m$^2$ (range 24.7-39.6).

***Measurement Protocol***

All participants were measured between September 2010 and January 2011. The SA were measured September 2010-October 2010; SS were measured October 2010-January 2011. The measurement battery included CVD risk factor assessment, anthropometric measures, and dietary intake and energy expenditure assessment using standardized procedures. Participants were assessed once in the order outlined in **Table 3**. The order was adjusted if participants biked or walked to the measurement location to ensure the participant was in a resting state before measuring RMR and RHR. In this case, the participant completed questionnaires first with physical measures following in the normal order. Data for most subjects was collected in the morning after an overnight fast of $\geq 8$ hours and no formal exercise for $\underline{\geq}$ 12 hours. Participants were instructed to drink 16 fl. oz. of water in the morning prior to measurement; compliance was assessed by verbal questioning when participants arrived. Two SS participants requested to be measured in the afternoon, but followed the protocol with respect to fasting and exercise.

***Resting Metabolic Rate and Resting Heart Rate***

After resting for ten minutes, RMR was measured using the Korr ReeVue (Korr Medical Technologies, Salt Lake City, UT), a device that estimates resting energy expenditure by measuring $O_2$ uptake and inferring a typical resting $CO_2$ level. The Korr ReeVue has been assessed for validity and reliability based on a comparison with the Deltatrac II Metabolic Monitor.[95] The RMR within-subject coefficient of variation was 12.2% while the estimated RMR coefficient of variance was 11.9%. During the RMR test, average RHR was used to determine if the participant was truly at rest using a Nonin Onyx II 9550 digital fingertip pulse oximeter (Nonin Medical, Inc., Plymouth, MN). For example, if the RHR was highly variable or rose during the test, this would indicate an invalid test, as the participant was likely not in a

resting state.[96] Additionally, RHR was measured since it is a marker of cardiorespiratory fitness.[97]

***Resting Blood Pressure***

A manual resting systolic and diastolic BP was taken following standardized procedures using a stethoscope and a standard blood pressure aneroid with an appropriately sized inflatable cuff on the subject's non-dominant upper arm with a Professional Aneroid Sphygmomanometer (AllHeart, Louisiana, MO).[98, 99] Three measures were taken with one minute between each measure; all measures had to be within 5mm Hg. The last two measures were averaged and used for analysis. Mean arterial pressure (MAP) was calculated using the following equation: MAP= (Systolic BP-Diastolic BP)/3+Diastolic BP.

***Blood Samples for Lipid Panel, Fasting Blood Glucose, and C-Reactive, Protein***

Fasting blood samples were obtained by fingerprick and collected in heparinized capillary tubes (two per person) following standardized procedures.[99] One sample was taken to be analyzed for TC, LDL, HDL, TG, TC/HDL and FBG. A second sample was collected to measure CRP. The samples were analyzed using the Cholestech LDX System following manufacturer guidelines. The Cholestech LDX has been validated and assessed for reliability when compared with the CardioCheck PA and commercial laboratory methods.[100, 101] When compared with the Cholesterol Reference Method Laboratory Network (CRMLN), the average percent bias for TC, HDL, TG, and LDL was -3%, -4%, +2.5%, and -4%, respectively.[97] The correlation coefficient for the fingerstick method of testing CRP was 0.97 when compared with values measured by a commercial clinical diagnostic laboratory.[100] If the fingerprick blood

draw was inadequate or the cassette assay did not run properly, participants were asked for permission to take a second sample or set of samples.

***Anthropometric Measures***

Standing height, body weight, body composition, and WC were measured in duplicate using standardized procedures[98,100] with the average of the two measures used for analysis. Standing height was measured to the nearest 0.1cm using a wall mounted, calibrated stadiometer (210 Holtain Limited, Dyfed, UK). Weight was measured to the nearest 0.1 kilogram using a calibrated electronic scale (BC-534 Tanita co., Tokyo, Japan). Height and body weight were used to calculate BMI (weight (kg) divided by height ($m^2$)). Body composition (% BF) was assessed using a foot-to-foot bioeletric impedance analysis (BIA) device (BC-534 Tanita co., Tokyo, Japan) which estimates % BF and derives % lean body mass. This device has been found to have test-retest intra-class correlation values exceeding 0.97 with a margin of error of ~ 3-4% when contrasted with skinfolds in females.[102] Waist circumference was measured to the nearest 0.1 centimeter using a Gulick anthropometric tape (Gulick co., Tokyo, Japan). The measurement tape was placed in a horizontal plane around the abdomen at the level 1cm above the superior border of the iliac crest.[103, 104]

***Composite Cardiovascular Disease Risk Score***

The primary CVD risk factor status analysis was based on a composite CVD risk score (cCVDs). The cCVDs included WC, MAP, fasting levels of TG, HDL, FBG and CRP. The standardized residuals (Z-scores) for the individual risk factors were summed to create the cCVDs. Since the standardized HDL is inversely related to cardiovascular risk, it was multiplied by -1 before summing all of the risk factors. A higher score was indicative of a less favorable

CVD risk status. This method is based on methodology for a metabolic syndrome score utilized by Eisenmann et al.[22] to evaluate metabolic syndrome risk. The score was recently validated in children[23] and adults.[24]

***Food Frequency Questionnaire***

The 2005 Gladys Block 110-item electronic-format food frequency questionnaire (FFQ)[105] was used to evaluate the dietary behavior of participants. The FFQ evaluates food and beverage intake frequency over the past year and provides an estimate of daily macro- and micro-nutrient intake as well servings of key food groups, and has shown to be both valid ($r>0.7$) and reliable ($r=0.7$ in women).[106, 107] Dietary intake was evaluated between the two groups and in reference to national NCEP TLC recommendations.[5]

After the FFQ were reviewed, those with < 1000 kilocalories per day or > 4000 kilocalories per day were further scrutinized by two registered dietitians to determine validity. This intake range of 1000-4000 Kcal /day cut point was established based on the typical range found in results of previous studies of collegiate female athlete intake.[79, 82] The questionnaire also asked participants if they were actively trying to lose weight, which was also considered when determining if the FFQs were valid.

***Physical Activity Questionnaire***

The electronic-format Block Adult Energy Expenditure Survey was used the estimate the TDEE of study participants.[93, 94] The survey takes into account occupational activity as well as activities of daily life and leisure time for the previous seven days. With intentions of increasing accuracy, instructions were modified slightly for SA participants, who were instructed to exclude

collegiate sport-related training in their PA survey. The expenditure from collegiate sport-related training was calculated for each SA using MET levels from the Compendium of PA[93] using a standardized procedure for all calculations. See **Table 4** for examples of MET levels used in this study.

***Statistical Analysis***

Two members from the study team independently entered all data into SPSS (Version 18, IBM SPSS Statistics, Chicago, IL). Outliers and potentially invalid entries received a third review and were discussed by the research staff prior to conducting the analyses. Descriptive statistics (mean $\pm$ standard deviation) were used to describe the anthropometrics, CVD risk factor variables, and dietary intake variables. Analysis of variance (ANOVA) was used to assess differences between SA and SS for every variable assessed. To determine the presence of CVD risk factors, criteria variables were evaluated for each participant, the number of positive risk factors were summed and percentages were calculated. Chi-squared tests were used to calculate odd ratios and confidence intervals when comparing subjects with $<2$ or $\geq 2$ CVD risk factors in each group. Mean intake of selected dietary variables (those mentioned in the FFQ section) was used for comparison to national recommendation.[5] The influence of TDEE, BMI, % BF, and dietary quality on cCVDs was assessed using linear regression. Statistical significance was set at $p \leq 0.05$ level.

## RESULTS

The final sample included 37 individuals (20 SA and 17 SS) who participated in the study; however complete data was not obtained for all participants for all variables for various reasons. One SA and two SS RMR measures were determined to be invalid due to not being completely at rest. Data were not obtained due to mechanical malfunction for the lipid panel and

FBG analysis in one SS participant, and the samples for CRP did not run in 6 subjects (2 SA, 4 SS). In addition, those subjects with a measured TG level <45 or $\geq$ 300 mg/dL did not have a calculated LDL (N= 9 subjects; 5 SA and 4 SS). Two participants FFQ were determined to be invalid and excluded from the analysis. One individual in the SA group did not complete the FFQ or energy expenditure survey, despite repeated reminders. Therefore, the mandatory practice energy expenditure for this athlete was also not included in analysis.

The top of **Table 5** includes height, weight, and BMI of the two groups. The SA group was 80% Caucasian, 15% African American or black, and 5% Hispanic, while the SS group was 47% Caucasian, 18% African American or black, 18% Hispanic and 5% Asian (12% of the SS group did not report ethnicity). The SA included athletes from the following teams: rowing (n=4), field hockey (n=2), basketball (n=1), softball (n=2), swimming (n=2), golf (n=2), soccer (n=2), volleyball (n=1), throwing (n=3), and tennis (n=1). There was a statistically significant difference ($p \leq 0.05$) between the two groups for height and BMI.

***Cardiovascular Disease Risk Assessment***

The main hypothesis of the study was that SA would have a significantly more desirable cCVDs when compared to SS. **Table 5** also includes means and standard deviations by group for all CVD risk factors assessed, including the 6 variables used for the cCVDs: WC, MAP, HDL, TG, FBG, and CRP. There was not a statistically significant difference in the cCVSs between the two groups (p=0.373). However, when evaluating the six individual risk factors in the cCVDs, as well as the additional CVD risk factors measured there was a statistically significant difference ($p \leq 0.05$) between the two groups in favor of the SA for % BF and fat-free mass, RHR, TC, non-HDL, LDL, and TC/HDL. Although not statistically significant, there was also a trend favoring the SA group for fat mass (p=0.07). There was not a significant difference

for TG, FBG, systolic or diastolic BP, or CRP. Finally, **Table 5** includes the percent of individuals in each group that met the NCEP[68] cut-off for "at risk". The highest percent at risk for both groups was recorded for WC (60% of SA and 70.6% of SS). Only one participant (SS) was classified as hypertensive based on a diastolic BP > 90 mmHg. When compared to SA, the SS group had a higher percent of participants that were at risk for every risk factor assessed with the exception of having a diastolic BP > 80 mmHg, for which the prevalence was higher in the SA group.

**Figure 1** shows the percent of individuals with < 2 or ≥ 2 CVD risk factors present when assessing only the seven risk factors included in the cCVDs (using systolic and diastolic BP versus MAP). The blood pressure cut-off used was systolic BP >120mmHg and diastolic BP >80mmHg. Overall, 69% of SA and 29.5% of SS had <2 CVD risk factors, and 35% of SA and 70.5% of SS had ≥ 2 CVD risk factors. The differences in prevalence were significant between the two groups (p=0.049). Odds ratios revealed that SA were 4.46 times more likely than SS to have <2 CVD risk factors (OR=4.46; CI 1.11-17.9) and that SA were 80% less likely than SS to have ≥ 2 CVD risk factors (OR=0.22; CI 0.06-0.90).

***Energy Expenditure and Dietary Intake***

**Table 6** shows the energy expenditure and dietary intake variables for both groups. The TDEE was significantly higher and RHR was significantly lower in SA. However, RMR was not significantly higher in SA.

In **Table 6**, the cut-off point for "at risk" is listed in column four for each variable. Contrary to what was hypothesized, the mean intake of total fat (in % Kcal) in SA was <35% and both groups consumed >2.5 cups of fruits and vegetables, which met current NCEP TLC guidelines.[5] However, neither group met the recommendations for saturated or trans fat, grams

of fiber per 1000 Kcal, sodium, or potassium. The following values were significantly higher for the SA: total Kcal, grams of carbohydrate and protein, mg of potassium, grams dietary fiber, and cups of vegetables. Percent of Kcal from trans fat was significantly higher in the SS. Though not statistically significant, the intake of total and added grams of sugar was higher in SA. The greatest percent not meeting recommendations was for % Kcal from saturated fat and milligrams of potassium for both groups (95% for SA and 100% for SS for both variables).

***Relationship of Energy Expenditure, Body Mass Index, and Body Composition with the Composite Cardiovascular Disease Risk Score***

**Table 7** summarizes the relationship of TDEE, BMI, % BF, and dietary quality with cCVDs. Overall there was no relationship between TDEE and cCVDs ($r=0.12$, $p=0.53$). In SA there was a trend for significance that energy expenditure is directly related to the cCVDs ($p=0.07$). **Figure 2** shows a visual display of these results for SA and SS. The relationship in SA was likely weakened by high cCVDs found in two of the SA individuals. No significance was found between TDEE and cCVDs in SS (**Table 7**).

There was a significant positive correlation between BMI and cCVDs as well as % BF and cCVDs for the total sample (**Table 7**). So, as either BMI or % BF increased, cCVDs (CVD risk) also increased. As individual groups, there was a significant positive correlation for BMI and cCVDs in SA, but not in SS. This data was analyzed further using a scatter plot (see **Figure 3**). This reveals that the unexpected significant trend for BMI was strongly affected by two high BMI and cCVDs data points. There was a significant positive correlation between % BF and cCVDs for both SA and SS (**Table 7**). There was no significant correlation between dietary quality (gm fiber intake/ 1000 Kcal intake) and cCVDs for SA, SS, or the entire sample.

## DISCUSSION

This is the first study to compare not just CVD risk status, but also energy expenditure, body composition, and dietary intake in a group of female collegiate SA versus collegiate SS. For the primary aim we used a sample-specific, continuous cCVDs. This approach provides a better assessment of overall risk versus previously developed scores because since each variable is continuous versus dichotomous, even small changes above or below normal were identified (versus. only absolute risk or no risk). This study revealed that being an active athlete with a lower BF % (despite having a BMI classification as overweight or obese) versus a similar-BMI sedentary student is associated with a more desirable lipid profile. In addition, the SS had a higher percentage of at risk individuals for nearly every risk factor that comprised the cCVDs. In contrast, a greater percentage of SA had <2 risk factors present when compared to SS. Both groups had a similar dietary intake with respect to heart health recommendations, but overall neither group was meeting NCEP TLC guidelines. Key limitations of this study were the small sample size and a difference in BMI which was significantly higher in the SS.

***Cardiovascular Disease Risk Assessment***

There was not a significant difference between the two groups for cCVDs. The effect size for this variable was calculated at 0.32, indicating a small effect.[108] The mean cCVDs for SA was -0.95 $\pm$ 2.93 and the mean cCVDs for SS was 0.11 $\pm$ 3.50. After re-running the power analysis with this cCVDs data, it would have taken a sample size of over 3000 individuals per group to give a statistical power >0.50.[109] When displayed as a scatter plot (**Figure 1)**, there is a cluster of cCVDs below zero for SA and at or above zero for SS, which could indicate a better CVD risk profile in the majority of SA versus the majority of SS. A similar SA and SS ranges for WC and CRP, as well as large variance for TG likely decreased the chance of a statistical difference.

The overall prevalence of risk factors was higher in the SS versus the SA (**Table 5**). Specifically, as hypothesized the lipid profile values were all significantly more favorable in SA, with the exception of TG. It was hypothesized in the current study that TG would be significantly lower in SA, but this was not found (though mean levels favored SA). This might be attributed to the fact that TG level can vary substantially from day-to-day and with phase of menstrual cycle. There was also no significant difference between the two groups for HDL. These results are similar to those found by Martinez-Gomez et al.[110] in a comparison of 210 adolescents (ages 13-16) with a sedentary, low or high level of activity. Those with a sedentary level of activity had less than favorable TG, FBG, and systolic BP levels, and continuous cardiovascular risk factor indexes (which included MAP, TG, HDL, and FBG). When evaluating the lipid results of SS, measured mean levels were all less desirable (with the exception of TG) than in a study by Sparling et al.[111] of female college students. That study, however, grouped all participants into one sample, regardless of physical activity level; mean BMI was also lower at 22.4 kg/m$^2$. However, although higher than what was previously reported, the mean values found in this study were not considered "at risk". When considering the percent of individuals with a given risk factor the results again favored SA (**Table 5**). With respect to HDL levels, 47.1% of SS and 10% of SA did not meet recommended levels. This finding in SS is significantly higher than in a previous assessment of traditional-aged college students, in which 18% had lower than desirable HDL levels.[112] However, the study had a much larger sample size (N=226) and included both athletes and non-athletes in one group. It was hypothesized that CRP levels would be significantly higher in SA due to the physiological stress of training[113] but this was not found. Results showed that levels were similar between the two

groups and on average the levels were within normal limits (<3 mg/L) (see **Table 5**). In SS, 23.1% of the group had levels ≥ 3mg/L, likely due to a higher % BF in SS.

***Energy Expenditure***

The SS had an unexpectedly high value for reported TDEE, although as anticipated it was significantly lower than SA. It is possible that some students had a poor understanding or realization of the amount of daily activity they perform, which then resulted in a high reported TDEE. Regardless the SS did have a significantly lower TDEE and a higher RHR, which indicates SS as a whole was less active than SA. A higher than expected TDEE in SS might have affected CVD risk factor status including the variables used in the cCVDs. It is well documented that there is a dose-response relationship between exercise and HDL and TG levels.[7, 8] Training programs that elicit 1200-2200Kcal/wk have been shown to elevate HDL levels from 2-8mg /dl and lower TG levels by 5-38 mg/dl. This amount of weekly expenditure is also associated with reductions in TC and LDL.[8] Both HDL and TG were used when creating the cCVDs. Neither value was statistically different between the two groups, which could also be partially due to the higher than expected TDEE in SS.

The SA did not have a significantly higher RMR despite having a significantly lower percent body fat, which was unexpected. This is in contrast to Ratcliff et al.[114] who found that RMR was significantly higher in endurance and resistance-trained groups of males versus sedentary (p=0.45). The lack of significance in RMR could have been related to the significantly higher RHR in SS.

***Dietary Intake***

Overall, the dietary intake with respect to targets for heart health based on NCEP TLC guidelines[5] between the two groups was similar. SA had a greater overall total caloric intake when compared to SS, which was expected. Similarly, Garcin et al.[115] found that endurance athletes had a significantly higher intake of Kcal when compared to sedentary individuals ($p<0.006$).

While it was expected that SA would have a mean fat intake above the current guideline of 35% total Kcal from fat, this was not observed. Previous studies of women in college have shown a fat intake of 37% ($\pm$ 4%) of total calories with 11% ($\pm$ 4%) of calories from saturated fat[116] and 36.7% ($\pm$ 6%) of total calories with 13.7% ($\pm$ 3%) of calories from saturated fat.[117] These two studies, however, were conducted with college students not specifically categorized as athletes. A prior study that was conducted in 1991 with collegiate female athletes reported a range for percent of kilocalories from fat identical to what was observed here (24-38%).[82] More recently, in 2004 Hinton et al.[79] assessed the dietary intake of NCAA Division I female athletes. The authors reported a fat intake of 28.2% ($\pm$ 5%) of total Kcals from fat with 9.3% ($\pm$ 3.1%) of calories from saturated fat. This low intake was correlated with desire to lose weight ($p=0.002$). The results of the prior two studies match best the results shown in the current study. Similar to those studies, the low intake in SA reported here could also be due to the fact that 75% of SA reported they were currently trying to lose weight. As expected, neither group met the recommendations for trans or saturated fat, although only trans fat intake was higher in SS when compared to SA. The SA and SS had similar intake of grams of sugar, which could also be attributed to the fact that the majority of both groups were trying to lose weight (75% of SA; 82% of SS).

Low intake of dietary fiber has been reported in many female collegiate athletes.[81] Similarly, 100% of our sample of athletes was not meeting the recommended level of fiber intake overall and when assessed using grams of fiber/1000 Kcal (see **Table 6**). Also, while it was expected that SA would have a significantly higher intake of fiber compared to SS, this was not observed. This was likely due to the unexpected high intake of fruits and vegetables reported by SS. A 2008 assessment of college students showed that only 8.5% of the sample reported eating 5 or more servings of fruits and vegetables daily.[25] In contrast, the total mean intake of fruits and vegetables in the current sample was ~8 one-half cup servings (4 cups) for SA and ~6 one-half cup servings (3 cups) for SS daily. When compared to SS, the SA intake was significantly higher only for vegetables, but not fruit. Our results coincided more with study conducted in 2000 that reported 25% of female college students eating ≥ 5 servings of fruits and vegetables daily.[84] The authors associated trying to maintain weight and using exercise to lose weight with this high intake of fruits and vegetables.

As expected, neither group met the recommendations for either sodium or potassium intakes. In fact, 100% of SA did not meet sodium or potassium recommendations. The significantly higher intake of potassium, by the SA versus the SS was likely due to the higher vegetable intake in the SA. A previous assessment of collegiate women showed an average intake of approximately 3200 mg of sodium per day, with more than half of respondents reporting sodium intakes ≥ 2400mg/day.[116] When specifically assessed in collegiate female athletes, sodium intake was 2689 mg (± 1021). Our reported SA intake was higher at 3283 (± 999). A previous study reported a potassium intake of 1410 mg (± 401) daily in a similar demographic (though not categorized specifically as athletes).[117] Potassium intake levels

reported here were significantly higher (2949mg ± 854 in SA; 2593mg ± 900 for the entire sample).

***Relationship of Energy Expenditure, Body Mass Index, and Body Composition with the Composite Cardiovascular Disease Risk Score***

Previous studies have shown that as total daily energy expenditure increases, cardiovascular risk decreases.[110] Thus it was hypothesized that in SA, as TDEE increased, cCVDs would decrease however the opposite result was found. When evaluating **Figure 2** it should be noted that this unexpected direct relationship with TDEE and CVD risk was driven by two participants with a high cCVDs while ~ 75% of SA risk factor status did not increase with increasing TDEE. When the data was re-run excluding these two subjects, there was no significant relationship between TDEE and cCVDs for SA ($r=0.38$; $p=0.18$). Another reason for this unexpected finding may have been due to the timing of measurement. While some athletes were in-season at time of measurement, others were out of season, pre-season, or just about to start pre-season. Thus, this likely makes the TDEE variable difficult to use with SA because little can be concluded from it. Ideally, all athletes should have been measured in the same period of training. No significant trends were seen between TDEE and cCVDs in SS, which was unexpected.

There was a significant positive correlation between cCVDs and BMI in SA, but not in SS (see **Table 7**). As mentioned already, this unexpected significant trend for BMI was strongly affected by two high BMI and cCVDs data points (a golfer and thrower). However, when analysis was re-run without these subjects, the significant relationship remained ($r=0.77$; $p=0.001$), indicating that as BMI increased in SA, CVD risk also increased. As was expected, there was also a significant positive association between % BF and cCVDs for the entire sample,

as well as each group individually. A positive correlation between adiposity and CVD risk has also been demonstrated in a previous study.[110] One surprising result was that BMI and cCVDs were more strongly correlated than % BF and cCVDs (r=0.81 versus r=0.67). Due to the poor correlations reported between BMI and percent body fat in athlete populations,[17] one would expect that there would be a stronger correlation when comparing cCVDs to % BF than when comparing to BMI. It is possible that if a more accurate measurement of % BF was used (e.g. Bod Pod or underwater weighing), the correlation between % BF and cCVDs may have been stronger.

***Strengths and Limitations***

Since this was a cross-sectional study, no inference of cause and effect can be made. Additionally, this study was powered for the cCVDs and individual CVD risk factors only. This may explain why few statistically significant differences were found for variables related to dietary intake. Two major limitations were that the two groups were not BMI-matched, with the SS having a significantly higher BMI; also the sample size was small. The mean BMI was significantly different between the two groups (see **Table 5**). Having BMI-matched groups was the original protocol desired for the study, but due to recruiting and time issues, matching by BMI was not achieved. The significantly lower BMI in SA could explain some of the more desirable results seen in SA versus SS, such as lipids, % BF, and WC. Due to problems recruiting, the sample size was smaller than originally planned (actual N=37 versus planned N=60). A third limitation was that the individuals recruited in SA were from a variety of teams that were in different periods of training. Several studies have found that energy intake, energy expenditure, body composition and blood values can vary dramatically before, during, and post-season.[79, 81, 82] An investigation of swimmers showed that changes in body composition over a

season were concentrated mainly in the beginning part of the season.[118] Swimmers in this study were measured immediately prior to fall training. In 1991, Nutter[82] also compared seasonal changes in dietary intake between athletes and non-athletes. The study found no significant differences in total energy intake between in-season to post-season in 24 athletes from four different sports (when assessed as total Kcal or Kcal/kg). In non-athletes, there was a significant difference in total Kcals and percent of kilocalories from carbohydrate. No other significant differences were seen in carbohydrate, protein, or fat intake among the athletes and non-athletes in the study. A fourth limitation of this study was that all individuals in the study were volunteers, which may indicate an already present interest in health resulting in more desirable CVD risk values and behaviors. This might have been the reason for the unexpectedly high intake of fruits and vegetables reported by the sample. A fifth limitation is the differences in ethnicity between the two groups. While both groups were primarily Caucasian and included a similar percentage of African American or black individuals, the SS group had a higher percentage of Hispanic individuals versus SA (18% versus 5%, respectively). However, prevalence of CVD risk factors in Hispanic populations is similar to Caucasian, so this likely did not affect the outcome variables.[1] Among women ages 20 and older, currently 31% of non-Hispanic whites, 45% of non-Hispanic blacks, and 32% of Mexican Americans have high blood pressure (BP).[1] In that same group, currently 49% of non-Hispanic whites, 42% of non-Hispanic blacks, and 49% of Mexican Americans have total blood cholesterol levels (TC) over 200 mg/dL.[1] Twelve percent of the SS group did not report ethnicity, so it is unknown if ethnicity contributed to measured values in these individuals.

There are some limitations with using the cCVDs. The score is sample specific, and so may not be applicable to the population as a whole, unless it is similar to this study's sample and distribution. In addition, the weighting of each individual variable to the final score is considered equal in the Z-score approach. This may not actually be true. Using factor analysis and principal components analysis instead would calculate the loadings of each variable independently. A limitation when using BIA for body composition analysis is the error caused by differences in hydration status.[102] To control for this, measurements were taken after an overnight fast and participants were asked to drink a standardized amount of fluid prior to arrival. Another limitation is that foot-to-foot BIA is not sensitive to fat distribution above the waist. However, BIA was chosen because it requires little training and the measure is safe, rapid, and reliable. Even though the FFQ and the PA questionnaire are validated tools, there are limitations to each of these. These limitations were controlled for as much as possible by standardized procedures for collection and analysis. However, some variables, such as recall bias or poor memory, are inevitable when using these dietary intake tools. It was also assumed that participants would be truthful when answering all questionnaires.

One strength of this study is the focus on the female athletic population, for which there is little data on cardiovascular disease risk factor status. Most of the current literature on CVD risk factor status in athletes has been conducted with overweight and obese male athletes; few studies have investigated CVD risk with overweight and obese female athletes (based on BMI) and compared them to women of a similar age and BMI. A related strength is that when assessing CVD risk, validated procedures were used for each measure. Using a continuous cCVDs is beneficial in this population because the population is young, and this methodology has a greater sensitivity for detecting differences than the tools that have been used for older

populations such as the Framingham Risk Factor Score[119] which includes dichotomous measures (i.e. hypertensive or normal BP). Another strength of this study was that the analysis also evaluated the influence of % BF, BMI, and amount of TDEE on CVD risk status while concurrently assessing heart health related dietary intake.

## CONCLUSION

In conclusion, this study found that, when compared to a collegiate female SS, being an active collegiate female athlete with a lower % BF (despite having a BMI classification as overweight or obese) is associated with a significantly more desirable lipid profile and an overall lower prevalence of CVD risk factors. This was likely due to the lower % BF, higher TDEE and fitness level in SA compared to SS. The SA had a similar dietary intake when compared to SS, and neither group was meeting the majority of the NCEP TLC guidelines for heart health. Finally, there is a positive relationship between CVD risk and either BMI or % BF when assessed as individual variables. Two major limitations of this study were the small sample size and the fact that while the two groups had similar BMIs they were not-matched. Future studies should address these limitations.

This study offers new insights that will be applicable to collegiate institutions. Thirty-five percent of SA and 70.5% of SS had at least two or more risk factors for CVD. This means that all college females, regardless of activity level, should be screened for risk factors associated with heart disease. Student health centers should make an effort to target this age group with regard to heart disease specifically. However, this study also shows that one should not assume an athlete is at risk for CVD when based only on BMI classification. Despite having a BMI over 25 $kg/m^2$, the majority of the SA in the sample had relative low CVD risk.

These findings indicate that continued research in this area is warranted. Future studies should include a multi-university assessment of CVD risk in BMI-matched women of varying activity levels (assessed via TDEE). It would also be advantageous to study the change in risk from freshman to senior year and assess how that change affects overall health and performance acutely and long-term. Additionally, continued CVD risk and morbidity monitoring, post college for both SS and SA (or once they stop playing their sport) would provide insight into if there are significant differences in long-term heart health between former SA and SS.

**APPENDICES**

## APPENDIX A: TABLES

| ***Cardiovascular Disease Risk Factors**** |
|---|
| ***Causal Risk Factors*** |
| Smoking |
| Resting Blood Pressure ≥140 Systolic >140 Diastolic >90 mmHg |
| Overweight (BMI>25) or Obesity (BMI>30) |
| Low density lipoprotein ≥160 mg/dl |
| High density lipoprotein < 40mg/dl (males), <45mg/dl (females) |
| Total Cholesterol > 240mg/dl |
| Triglyceride >150mg/dl |
| Elevated fasting blood glucose |
| Males >55 years, Females >65 years |
| ***Predisposing Risk Factors*** |
| Overweight or Obesity (BMI ≥25) |
| Waist circumference >40 in. (men) or >35 in. (women) |
| Physical inactivity |
| Genetics |
| Family history of premature death (< 65 y/o) from heart disease |
| Insulin resistance |
| ***Emerging/Conditional Risk Factors*** |
| Homocysteine >10 micromoles/L |
| C-reactive protein ≥3mg/L |
| BMI=body mass index<br>*Summarized from American Heart Association and American College of Cardiology recommendations[29,30,112,114-116] |

**Table 1: Cardiovascular disease risk factors**

| *Dietary Component* | *AHA Recommendation for CVD Prevention (NCEP TLC Diet)** | *ACSM-ADA Position Statement for Athletes*** |
|---|---|---|
| Carbohydrates | 50-65% total Kcal | 6-10gm/kg |
| Fiber grams/day | ≥ 12.5gm/1000 Kcal | Suggest 30-35 gm/day |
| Fat | ≤ 35% total Kcal | 20-35% Kcal |
| Saturated Fat | ≤ 7% total Kcal | < 10% Kcal |
| Trans Fat | ≤ 1% total Kcal | None given |
| Protein | 0. 8 g/kg or 10-35% *** | 1.2-1.7gm/kg body weight |
| Potassium | ≥ 4415 mg | None given |
| Sodium | ≤ 1600 mg | None given |

*Summarized from American Heart Association (AHA) recommendations[5, 68, 69]

**Summarized from American College of Sports Medicine (ACSM) and American Dietetic Association (ADA) guidelines[70]

***There is no AHA recommendation; value is dietary reference intake (DRI)

**Table 2: Daily nutrient recommendations from various governing bodies**

| ***Measurement and Order*** | ***Time Required*** |
|---|---|
| ≥ 8 hour overnight fast<br>≥ 12 hours since last exercise bout | ----- |
| Participant enters and sits quietly | 10 min |
| Resting metabolic rate and resting heart rate | 15 min |
| Resting blood pressure (x3) | 10 min |
| Finger stick blood sample taken for lipid panel and fasting blood glucose and one sample for c-reactive protein | 15 min |
| Anthropometrics: Standing height, body weight, body composition, and waist circumference | 10 min |
| Food Frequency Questionnaire and Energy Expenditure Survey | 55 min |
| ***Total time required per subject*** | **115 min** |

**Table 3. Measurement protocol summary**

| *Activity* | *Intensity/Activity Type* | *MET Level* |
|---|---|---|
| Abdominal exercises | Moderate | 3.5 |
| Basketball | Game | 8 |
| Basketball | Shooting | 4.5 |
| Calisthenics | Easy | 4 |
| Calisthenics | Moderate | 6 |
| Calisthenics | Hard | 8 |
| Cycling | Moderate | 7 |
| Field Hockey | Moderate/Scrimmage | 6.5 |
| Field Hockey | Hard/Scrimmage | 8 |
| Golf | Chip/Putt | 3 |
| Golf | Game | 4.5 |
| Rowing | Moderate | 7 |
| Rowing | Hard | 8.5 |
| Running | Easy (11.5min/mi) | 9 |
| Running | Moderate (10 min/mi) | 10 |
| Running | Hard (7.5 min/mi) | 13.5 |
| Running | Sprints (7 min/mi) | 14 |
| Soccer | Moderate/Scrimmage | 7 |
| Soccer | Hard/Scrimmage | 10 |
| Softball | Moderate/Scrimmage | 4 |
| Swimming | Moderate | 8 |
| Swimming | Hard | 11 |
| Tennis | Doubles | 6 |
| Tennis | Singles | 8 |
| Throwing | Easy | 3 |
| Throwing | Moderate | 4 |
| Volleyball | Moderate | 5 |
| Volleyball | Hard | 8 |
| Weight Lifting | Moderate | 5 |
| Weight Lifting | Hard | 6 |
| MET=metabolic energy equivalent | | |

**Table 4: MET levels used for athlete mandatory practice expenditure calculations**

| *Variable & Risk Cut-off†* | *SA (n=20#)‡* | *SS (n=17#)‡* |
|---|---|---|
| Age (years) | 19.7 ± 1.2 (18-22) | 19.5 ± 1.6 (18-23) |
| Height (cm) | 170.9 ± 8.8 (156.1-183.6)* | 164.3 ± 6.0 (151.6-174.8) |
| Weight (kg) | 80.8 ± 15.9 (61.4-125.7) | 80.9 ± 12.0 (63.6-104) |
| Body Mass Index (kg/m$^2$) | 27.5 ± 3.3 (24.7-37.4)* | 30.0 ± 4.4 (26-39.6) |
| Waist Circumference (cm)** >88 cm | 89.8 ± 9.9 (78.1-114.8) 60% | 96.4 ±10.8 (78-113.4) 70.6% |
| Percent Body Fat | 31.5 ± 5.8 (21.2-42.9)* | 38.7 ± 4.7 (30.8-47.1) |
| Fat-free Mass (kg) | 54.8 ± 7.7 (41.6-73.7)* | 49.1 ±4.3 (41.1-56.1) |
| Fat Mass (kg) | 26.1 ± 9.5 (13.6-52.0) | 31.7 ± 8.5 (22.5-47.9) |
| Systolic Blood Pressure (mmHg) >120 mmHg | 116.5 ± 6.0 (103-124) 10% | 114.1 ± 6.7 (100-126) 17.6% |
| Diastolic Blood Pressure (mmHg) > 80 mmHg | 78.9 ± 5.9 (66-88) 40% | 77.8 ± 5.2 (68-93) 17.6% |
| Mean Arterial Pressure** | 91.4 ± 5.5 (78-99) | 90 ±5.2 (81-103) |
| Total Cholesterol (mg/dL) >240 mg/dL | 153.9 ± 22.3 (116-191)* 0%; | 177.1 ± 31.7 (126-224) 0% |
| High Density Lipoprotein (mg/dL)** <45 mg/dL | 56.2 ± 8.5 (41-73) 10% | 52.7 ± 19.0 (33-100) 47.1% |
| Triglyceride (mg/dL)** >150mg/dL | 76.6 ± 32.8 (45-155) 5% | 93.3 ± 50.7 (45-195) 23.5% |
| Non-High Density Lipoprotein (mg/dL) | 97.9 ± 21.1 (62-133)* | 125.5 ± 37.3 (70-183) |
| Low Density Lipoprotein (mg/dL) ≥160 mg/dL | 86.8 ± 17.1 (53-110)* 0% | 107 ± 30.2 (59-151) 0% |
| Total Cholesterol to High Density Lipoprotein Ratio ≥3.5 | 2.8 ± 0.49 (2.2-3.7)* 20% | 3.8 ± 1.2 (1.9-5.5) 56.3% |
| Fasting Blood Glucose (mg/dL)** ≥100 mg/dL | 85.4 ± 7.1 (72-98) 0% | 87.3 ± 7.8 (67-100) 5.9% |
| C-Reactive Protein (mg/L)** ≥3 mg/L | 1.13 ±1.0 (0.30-3.61) 11.1% | 1.51 ± 1.4 (0.30-4.2) 23.1% |

SA=student athletes; SS=sedentary students

†NCEP[68] at risk level noted below variable title, where applicable

‡Values expressed as mean ± SD with range in parentheses; percent at risk below

*$p \leq 0.05$ between two groups

#Sample size varies by measure due to missing variables n=15 to 20 for SA; n= 13 to 17 for SS

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

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

| *Variable and Risk Cut-off†* | *SA (n=20#)‡* | *SS (n=17#)‡* |
|---|---|---|
| Total Daily Energy Expenditure (Kcal) | 3878 ± 1273 (2068-8192)* | 2908 ±587 (2158-3933) |
| Moderate-Vigorous Physical Activity (min/day)*** | -18.56 ± 243 (-339-675) | -79.18 ± 154 (-479-263) |
| Resting Heart Rate | 60.1 ± 11.0 (43-79)* | 71.2 ± 7.6 (58.7-86) |
| Resting Metabolic Rate (Kcal) | 1733 ± 358 (1181-2390)* | 1685 ± 221 (1195-2102) |
| Total Kcal | 2061 ± 566 (1239-3072)* | 1598 ± 631 (838-3148) |
| Carbohydrate (gm) | 275 ± 77.5 (149-385)* | 205 ± 84.8 (103-391) |
| Protein (gm) | 79.7 ± 29.2 (44-157)* | 57.9 ± 20.4 (28-97) |
| Fat (gm) | 72.9 ± 22.5 (47-115) | 60.7 ± 25.0 (31-127) |
| Saturated Fat (gm) | 23.1 ± 7.5 (13-41) | 18.8 ± 6.7 (10-34) |
| Trans Fat (gm) | 2.5 ± 1.02 (1-5) | 2.3 ±0.98 (1-4) |
| Monounsaturated Fatty Acids (gm) | 28.32 ± 8.8 (17-44) | 23.5 ± 9.8 (11-48) |
| Polyunsatured Fatty Acids (gm) | 16 ± 5 (9-26) | 13.8 ± 7.0 (7-35) |
| % Kcal Carbohydrate | 53.4 ± 5.8 (48-69) | 51.2 ± 6.2 (42-59) |
| % Kcal Protein | 15.4 ± 2.6 (10-21) | 14.7 ± 2.1 (11-17) |
| % Kcal Fat<br>>35% Kcal | 31.9 ± 4.1 (24-38)<br>16% | 34.5 ± 5.3 (26-43)<br>33.3% |
| % Kcal Saturated Fat<br>>7% Kcal | 10.1 ± 1.4 (7-12)<br>95% | 10.7 ± 2.0 (8-17)<br>100% |
| % Kcal Trans Fat<br>>1% Kcal | 1.1 ± 0.2 (1-2)*<br>5.3% | 1.4 ± 0.6 (1-3)<br>20% |
| Sodium (mg)<br>>1600 mg | 3283 ± 999 (1918-5943)<br>100% | 2593 ± 1035 (1165-5094)<br>87% |
| Potassium (mg)<br><4415 mg | 2949 ± 854 (1332-4609)*<br>95% | 2141 ± 763 (1206-3518)<br>100% |
| Dietary fiber (gm) | 20.6 ± 6.3 (7-33)* | 14.0 ± 4.3 (6-20) |
| Fiber intake (gm fiber/1000Kcal)<br><12.5gm/1000 Kcal | 10.1 ± 2.2 (6-13)<br>89.5% | 9.3 ± 2.8 (6-16)<br>87% |
| Fruit (cups)<br><1 cup | 1.8 ± 0.9 (0-3.0)<br>5.3% | 1.7 ± 1.2 (0.3-4.0)<br>20% |
| Vegetables (cups)<br><1.5 cups | 2.1 ±0.8 (1-3)*<br>26.3% | 1.4 ± 0.6 (0.8-2.4)<br>60% |
| Sugars (gm) | 139.4 ± 48.5 (64.3-266.5) | 104.3 ± 57.9 (34.3-224.8) |
| Added sugars (gm) | 66.7 ± 26.9 (22.2-144) | 46.5 ± 32.7 (13.2-134) |
| SA=student athletes; SS=sedentary students; Kcal=Total calories; gm=grams; mg=milligrams<br>*p≤0.05 between two groups | | |

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

**Table 6 (cont'd)**

†NCEP TLC[5] risk level noted below variable title, where applicable

‡Values expressed as mean ± SD with range in parentheses; percent at risk below

#Sample size varies by measure due to missing variables n=18 to 20 for SA; n= 15 to 17 for SS

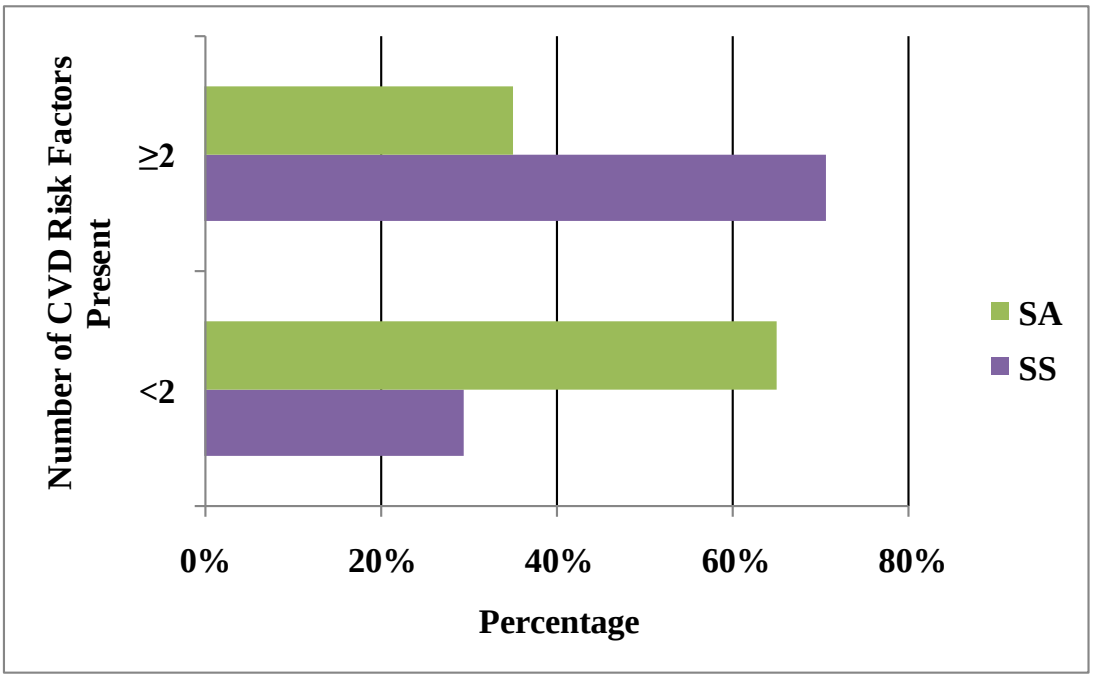
***Calculated variable from energy expenditure survey:

Minutes walk/run/swim/treadmill/other exercise /sports/other fitness
*minus* minutes TV/internet/sit

| *Sample Analyzed* | *Total Daily Energy Expenditure* | *Body Mass Index* | *Percent Body Fat* | *Dietary Quality* |
|---|---|---|---|---|
| Total Sample (N=29#) | r=0.12 (p=0.53) | r=0.59 (p=0.001) | r=0.60 (p=0.00) | r=0.28 (p=0.14) |
| SA (n=17#) | r=0.47 (p=0.07) | r=0.81 (p=0.00) | r=0.67 (p=0.003) | r=0.07 (p=0.80) |
| SS (n=13) | r=0.14 (p=0.65) | r=0.30 (p=0.32) | r=0.58 (p=0.004) | r=0.47 (p=0.13) |
| SA=student athletes; SS=sedentary students<br>#Sample size varies by measure due to missing variable N=28 to 29; n=16 to 17 for SA | | | | |

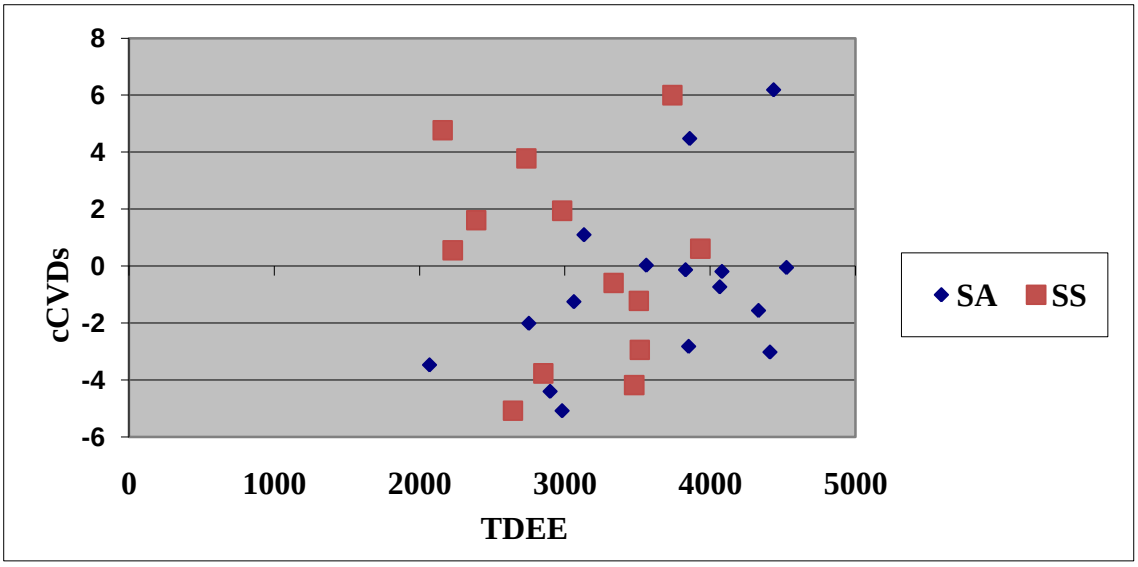
**Table 7: Influence of energy expenditure, body mass index, percent body fat, and dietary quality on cardiovascular risk**

## APPENDIX B: FIGURES



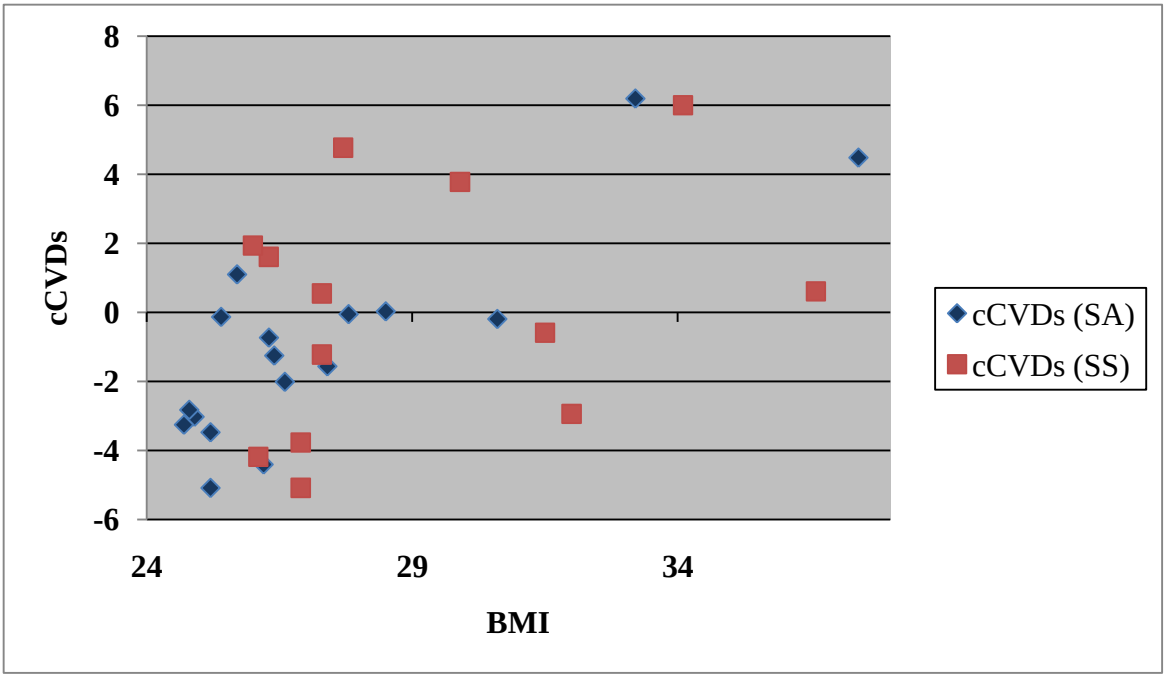

SA=student athletes; SS=sedentary students; CVD=cardiovascular disease
For interpretation of the references to color in this and all other figures, the reader is referred to the electronic version of this thesis.

**Figure 1: Prevalence of cardiovascular disease risk factors by group**



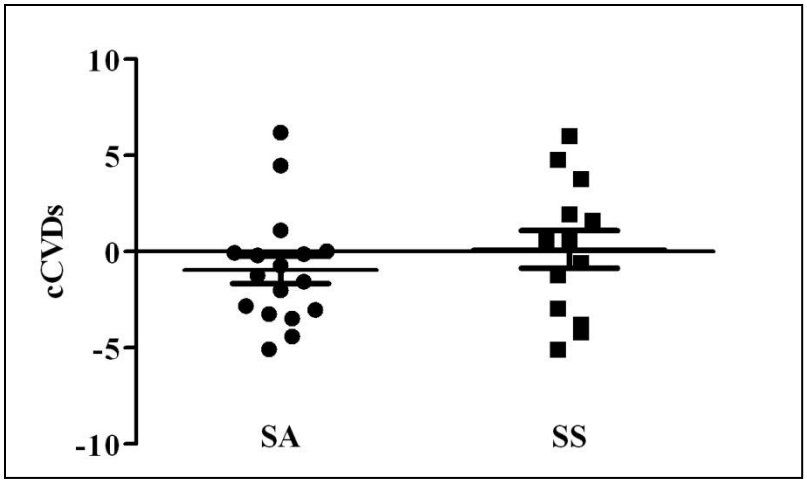

cCVDs=composite cardiovascular disease risk score; TDEE=total daily energy expenditure; SA=student athletes; SS=sedentary students

**Figure 2: Influence of energy expenditure on cardiovascular risk**




cCVDs=composite cardiovascular disease risk score; BMI=body mass index; SA=student athletes; SS=sedentary students

**Figure 3: Influence of body mass index on cardiovascular disease risk**




cCVDS=composite cardiovascular disease risk score; SA=student athletes; SS=sedentary students

**Figure 4: Composite cardiovascular disease risk score plot for each group**

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## REFERENCES


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