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ACCEPTANCE

This thesis, THE RELATIONSHIP BETWEEN ALCOHOL INTAKE AND BODY FAT PERCENTAGE IN ADULT UNIVERSITY EMPLOYEES, by Jessica L. Beardsley was prepared under the direction of the Master's Thesis Advisory Committee. It is accepted by the committee members in partial fulfillment of the requirements for the degree Master of Science in the Byrdine F. Lewis School of Nursing and Health Professions, Georgia State University. The Master's Thesis Advisory Committee, as representatives of the faculty, certify that this thesis has met all standards of excellence and scholarship as determined by the faculty.

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ABSTRACT

THE RELATIONSHIP BETWEEN ALCOHOL INTAKE AND BODY FAT PERCENTAGE IN ADULT UNIVERSITY EMPLOYEES

by

Jessica L. Beardsley

Background: Factors that contribute to body fat and adiposity include energy consumption, macronutrient intake, and physical activity. Alcohol not only contributes to total energy consumed but also influences metabolic pathways that may alter fat oxidation and storage. Alcohol provides 7.1 kilocalories per gram (kcal/g) and makes up 6-10% of the daily caloric intake of adults in the United States. Cross-sectional studies have shown that increased alcohol intake is associated with higher body mass index (BMI), especially in men. Other studies suggest that there is a “U” shaped association whereby non-drinkers and heavy drinkers have a higher BMI and waist-to-hip ratio (WHR) than low to moderate drinkers. While many previous studies evaluate alcohol based on the average consumption (g/day), there is increasing evidence that it is the pattern of alcohol consumption (ie. frequency) that influences body composition. The purpose of this study is to evaluate the effect of the frequency of wine, beer, and liquor consumption on body fat percent (BF%) and WHR in a population of university faculty and staff.

Methods: The Center for Health Discovery and Well Being (CHDWB) cohort trial is being conducted at Emory University in Atlanta, GA. Recruitment of faculty and staff for the study began in 2007. Demographic, reported dietary intake including wine, beer, and liquor consumption, and anthropometric data including weight, height, BF%, and waist

circumference are collected at baseline and annually thereafter. We used linear regression models to determine the effect of frequency and quantity of wine, beer, and liquor consumption on BF% while controlling for age and the effects of the other types of alcohol. We applied the Kruskal-Wallis test to determine if the median BF% and waist-hip ratio (WHR) was significantly different for those that reported at different five different frequencies (several times a year to 5-7 days a week).

Results: Baseline visits have been conducted on 700 participants. Their median age was 51 years (66% female). Median weight was 76.9 kg (range, 65.3 - 90.5 kg) and mean BMI was $27.9 \pm 6.4 \text{ kg/m}^2$. A significant negative relationship was observed between frequency of beer consumption and BF% in women ($p < 0.05$) but not in men. As liquor intake increased in frequency, there was a positive relationship observed with WHR in males ($p < 0.05$). There was also a significant decline in median BMI and BF% as wine intake increased in frequency for women ($p < 0.05$). In regression analysis controlling for confounding factors including alcohol quantity, increasing frequency of wine intake was significantly predictive of lower BMI and BF%. No significance was found when introducing a square term to test quadratic relationship between intake frequency and BF%, BMI, and WHR.

Conclusions: The frequency of wine intake consumed by university employees and staff independently predicted BF% and BMI. Greater frequency of wine consumption was associated with lower BF%.

THE RELATIONSHIP BETWEEN ALCOHOL INTAKE AND BODY FAT
PERCENTAGE IN ADULT UNIVERSITY EMPLOYEES

By

Jessica L. Beardsley

A Thesis

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ABBREVIATIONS

ADH	Alcohol Dehydrogenase
BF%	Body Fat Percentage
BMI	Body Mass Index
CAPS	Cross-cultural Activity Participation Study
CHDWB	Center for Health Discovery and Well Being
CHO	Carbohydrate
CI	Confidence Interval
CVD	Cardiovascular Disease
DEXA	Dual Energy X-ray Absorptiometry
DNL	De Novo Lipogenesis
FFQ	Food Frequency Questionnaire
g	Grams
g/d	Grams per Day
h	Hours
kcal/g	Kilocalories per gram
kg/m ²	Kilograms per meters squared
lbs	pounds
MEOS	Microsomal Ethanol Oxidizing System
ml	Milliliter
NADH	Nicotinamide Adenine Dinucleotide

NHANES	National Health and Nutrition Examination Survey
NHIS	National Health Interview Surveys
NIAAA	National Institute on Alcohol Abuse and Alcoholism
NWO	Normal Weight Obesity
OR	Odds Ratio
oz	Ounces
SD	Standard Deviation
SREBP	Sterol Regulatory Element-Binding Protein
TOBALC	Tobacco and Alcohol Survey
UK	United Kingdom
US	United States
WC	Waist Circumference
WHO	World Health Organization
WHR	Waist-Hip Ratio
WHS	Womens Health Study
wk	Weeks
y	Years

CHAPTER I

THE RELATIONSHIP BETWEEN ALCOHOL INTAKE AND BODY FAT PERCENTAGE IN ADULT UNIVERSITY EMPLOYEES

Introduction

Many factors contribute to body fat including energy consumption, macronutrient composition, and physical activity.^{1,2} Alcohol, providing 7.1 kilocalories per gram (kcal/g), makes up 6-10% of the daily caloric intake of adults in the United States (US).³ By altering metabolic pathways alcohol consumption may also have an influence on the breakdown and storage of fat. Clinical studies indicate various relationships between alcohol intake and lipid oxidation. On one hand, alcohol ingestion favors positive lipid balance by its conversion to acetate, which inhibits lipid oxidation after a meal.^{4,5,6} On the other hand, alcohol has been found to increase energy expenditure,^{7,8} especially when it provides additional calories at a meal.^{6,7} In contrast, extreme alcohol use is associated with increased lipid oxidation rather than decreased fat oxidation as seen in moderate consumers.⁹ Even though fat oxidation is decreased with alcohol intake, frequent drinkers have the lowest odds ratio (OR) for being obese,¹⁰ and drinkers have significantly lower body fat percentage (BF%) than non-drinkers.¹¹ In fact it is the frequency and amount of alcohol intake that primarily links alcohol to body mass index (BMI) and body fat. Alcohol in frequent, moderate doses (one drink per day for women and two drinks per day for men)¹² is associated with low BMI¹³ and lower central adiposity¹⁴ while infrequent, high doses (over four drinks a day) is associated with the highest BMI.^{13,15}

Chronic alcohol users (frequent, high doses) have significantly less body fat than their non-alcoholic counterparts.⁹

While body weight and BMI are commonly used for quick health assessment, recent research suggests that BF% may be a better predictor of morbidity and mortality. A recent study found that people with normal BMI (18.5-24.9 kg/m²) and high BF% (>23.1% in men and >33.3% in women) were more likely to have metabolic syndrome.¹⁶ These high BF% subjects were referred to as normal weight obese (NWO) and were more likely to have a collection of disorders that raise the risk of cardiovascular disease (CVD) and type 2 diabetes: central adiposity, hypertension, dyslipidemia, and elevated blood glucose levels. In women, NWO was an independent predictor of CVD mortality.¹⁶ Due to the prevalence of alcohol consumption and the detrimental effects of elevated body fat, it is important to clarify the relationship between alcohol intake and body fat.

This proposed study is a secondary analysis of data previously collected. The Emory-Georgia Tech Predictive Health Institute recruited healthy Emory University employees to participate in an ongoing collection of health indicators including body composition, waist-hip ratio (WHR), and lifestyle data (e.g. tobacco and alcohol use and diet pattern). This sample of Atlanta university employees is an accessible population that is representative of the urban middle class. The goal of this observational retrospective study is to evaluate the relationship between reported alcohol usage and BF% in free-living individuals. We hypothesize that the participants with the most frequent alcohol consumption will have the lowest body fat percentage as measured by dual energy x-ray absorptiometry (DEXA). Also, those that consume greater quantity of alcohol drinks per day will have higher central adiposity and BF% as measured by WHR.

CHAPTER II

Literature Review

Alcohol Usage and Prevalence of Alcohol Abuse

In the US, 37% of adults ages 18 years (y) and older drink at low-risk levels, while 28% drink at heavy or at-risk levels.¹⁷ Table 1 shows the National Institute on Alcohol Abuse and Alcoholism (NIAAA) definition of drinking levels. A single alcohol drink is defined as 14 g of alcohol or 0.6 ounces (oz) of pure alcohol.¹² This is equivalent to 12 oz of beer, 5 oz of wine, or 1.5 oz of 80 proof distilled spirits.¹² According to the Centers for Disease Control and Prevention (CDC), about 38 million adults in the US drink too much, resulting in health consequences such as CVD, breast cancer, sexually transmitted diseases, and liver disease.¹⁸ “Drinking too much” refers to heavy and at-risk drinking habits as defined in the table below as well as any alcohol used by pregnant women or consumed under the age of 21 years.¹⁸

Table 1. National Institute on Alcohol Abuse and Alcoholism Definition of Drinking Levels

Drinking Pattern Terms	Men	Women
“Low-Risk” Drinking	No more than 4 drinks on any day	No more than 3 drinks on any day
	AND	
	No more than 14 drinks per week	No more than 7 drinks per week
“Heavy” or “At-Risk” Drinking	5 or more drinks on any day	4 or more drinks on any day
	OR	
	15 or more drinks per week	8 or more drinks per week
“Moderate” Level	Up to 2 drinks per day	Up to 1 drink per day
*Sourced from NIH “Rethinking Drinking” Publication No. 13-3770. 2010. ¹⁷		

Alcohol Metabolism

Alcohol is a toxic substance that requires immediate breakdown upon ingestion. Alcohol is metabolized through two main pathways: alcohol dehydrogenase (ADH) and hepatic microsomal ethanol oxidizing system (MEOS).¹⁸ The ADH pathway produces acetaldehyde and NADH; acetaldehyde is further broken down into acetate, which can yield energy via the citric acid cycle.¹⁸ Excessive production of NADH and acetaldehyde are linked with metabolic disturbances such as lipid accumulation in the liver and decreased fat oxidation.^{18,19} While moderate alcohol intake requires ADH, chronic alcohol consumption relies primarily on MEOS for alcohol oxidation.²⁰ Recent data suggest that MEOS may be induced by a daily alcohol intake of 30 to 40 g or two to three standard alcohol drinks.²¹ In extreme alcohol users, MEOS is associated with increased energy wasting (energy lost through heat) compared to the ADH system in moderate alcohol users.²²

The body must expend energy in order to digest and distribute nutrients for energy or storage; this is known as the thermic effect of food or dietary thermogenesis.

While some macronutrients are efficiently processed and stored, others require a greater amount of energy. Fat increases energy expenditure up to 3% while carbohydrate (CHO) requires 5-10% and protein uses 20-30%.²³ Several clinical trials have found that alcohol is not efficiently metabolized and has a high thermic effect. For example, Suter et al. (1994) measured six non-alcoholic men with an average alcohol intake of 46 grams per week (g/wk) using indirect calorimetry and found the thermic effect of 95 g of alcohol in a 24-hour (h) period to be 22% compared with only 10% for a regular mixed meal of protein, CHO, and fat.²⁴ Sonko et al. (1994) conducted a similar study and found the thermic effect of alcohol to be only 15%, similar to their measurements for CHO.⁶ They also found that fat oxidation was suppressed immediately after alcohol ingestion (0-6 h).⁶ In another clinical trial, 19 normal-weight men and women were given four different meals that were high in either protein, fat, CHO, or alcohol.²³ There was no difference in the total calories consumed by participants, but the alcohol-rich meal had the largest thermogenesis (27%) and highest energy expenditure.²³ In a crossover trial by Murgatroyd et al. (1996) eleven men and women were given either a control meal, a meal with alcohol substituting for calories, or a meal with alcohol adding to total calories.⁵ Energy expenditure increased by 15% with the addition of alcohol compared to the control. These clinical trials indicate that alcohol has a higher thermic effect than CHO and fat, suggesting that alcohol's energy, 7.1 kcal/g, is not as readily available for use as other macronutrients.

Alcohol is used not only less efficiently for energy, but it alters metabolic pathways. In Murgatroyd's study, lipid oxidation was significantly decreased after both substitution and addition of alcohol in the meal.⁵ Fat oxidation was suppressed to the

degree that 74% of its energy was stored as fat.⁵ Similarly in a study by Raben et al. (2003), alcohol-rich meals were followed by significantly decreased fat oxidation compared with the other meals.²³ In 1992, Suter et al. found that alcohol given to eight healthy men (either in addition or isocalorically substituted for fat and CHO) decreased lipid oxidation rates up to 36%.⁷ As indicated in the introduction, heavy alcohol users exhibit the opposite effect by having enhanced lipid oxidation. In a study by Levine et al. (2000) comparing 36 alcoholics with matched healthy participants, the alcohol abusers had higher resting energy expenditures and lower respiratory quotients than the control group indicating that fat rather than CHO was used as a fuel source.⁸ However, after several days of abstinence the alcohol abusers' energy expenditure decreased.⁸ The effect of alcohol on lipid oxidation is dependent on the individual's level of consumption with a tendency for moderate alcohol use to decrease fat oxidation while chronic heavy use to increase it. Positive energy balance, excess fat intake, and decreased lipid oxidation favor weight gain and the development of obesity.² Increased lipid oxidation may explain why alcoholics are not obese despite high energy intakes.

Alcohol and Weight Gain

Siler et al. (1999) found that ethanol ingestion was followed by increased *de novo* lipogenesis (DNL) in eight healthy male participants.⁴ Using stable-isotopes, DNL and lipolysis were measured after ingestion of 24 g of alcohol. Lipogenesis was activated but only represented a small portion of ingested alcohol. The alcohol was primarily converted to acetate and circulated to be used as a source of fuel while decreasing fat oxidation. In a murine study, You et al. (2002) found that a low-fat diet with ethanol resulted in a

significant increase in sterol regulatory element-binding protein (SREBP), which was associated with increased expression of hepatic lipogenic genes in mice.²⁵ Activation of SREBP by alcohol can help explain alcoholic fatty liver caused by chronic alcohol consumption in humans as well. While alcohol ingestion decreases lipid oxidation (in nonalcoholics), a small amount of lipogenesis occurs, which can contribute to weight gain and increased adiposity over time.

Alcohol consumed before a meal tends to increase meal-calorie intake, which is likely due to inhibited satiety or intensified enjoyment of food. Participants in a crossover study who consumed beer with their meal increased their food intake by 16% compared to alcohol-free beer.²⁶ Another study using isocaloric aperitifs of wine, beer, or juice showed a significant increase in meal intake of approximately 28% after consuming the alcoholic drinks.²⁷ Increasing the dosage of alcohol also significantly increases the amount of food consumed at a meal. Twelve male participants consumed 17% more energy at a meal after consuming 32 g of alcohol compared to 8 g of alcohol. They consumed not only more calories, but they also chose more high fat and salty foods.²⁸ These studies show that alcohol can affect food choices and increase caloric intake that can contribute to overconsumption and positive energy imbalance leading to weight gain.

Longitudinal studies show how alcohol consumption habits can influence weight gain in men and women. In a ten-year longitudinal study, Liu et al. (1994) measured the body weight of 7,230 participants from the first National Health and Nutrition Examination Survey (NHANES I).²⁹ Females who consumed alcohol gained less weight than non-drinkers. However, male drinkers had a similar and higher OR of gaining weight compared to non-drinkers. The female drinkers had a lower OR of weight gain.²⁹

A five-year study in British men conducted by Wannamethee and Shaper (2003) found that stable and new heavy drinkers who consumed 30-60 grams per day (g/d) had the highest odds of weight gain (1.19-1.34) as well as the largest average weight gain (1.53-1.6 kg). This group also had the highest prevalence of BMI greater than 28 kg/m² (25.7-27.2%) after five years. Among the light-moderate alcohol consumers, those who consumed primarily liquor had the highest adjusted BMI compared to beer and wine.³⁰ A prospective analysis by Mozaffarian et al. (2011) of three cohorts at 4 year intervals for 20 years found that alcohol intake was independently associated with weight gain.³¹ Increasing alcohol by one drink per day was associated with a weight gain of 0.41 pounds (lbs). The largest weight gain (1-2 lbs) was associated with increases in liquor compared to beer and wine.³¹ In another prospective study, Wang et al. (2010) followed 19,563 normal weight women from the Women's Health Study (WHS).³² Over the 13 year follow-up, non-drinkers gained more weight than drinkers. The relative risk of becoming obese for those consuming 1-4 g alcohol per day, 5-14 g, 15-29 g, and over 30 g/d was 0.75, 0.43, 0.39, and 0.29 respectively.³² Both the Wannamethee and Shaper study in men and Mozaffarian et al. study in health employees demonstrated a propensity for weight gain with increasing alcohol intake. On the other hand, the Liu et al. and Wang et al. studies showed alcohol consumption to be associated with less weight gain than alcohol abstinence. Seeming inconsistencies in alcohol's association with weight gain could be due to the differences between gender, alcoholic beverage (ie. liquor, beer, or wine), or the method that alcohol intake was measured and quantified (average grams per day versus frequency and amount).

Alcohol, Obesity, and Central Adiposity

Obesity in adults is defined as having a BMI greater than or equal to 30 kg/m². Obesity increases a person's risk of several maladies including heart disease, diabetes, and osteoarthritis. Evidence is mixed on the association between alcohol use and obesity. In a national alcohol survey, obese men had a significantly greater rate of alcohol dependence and abuse compared to normal weight men.³³ However, no significance association was found in women. Is alcohol consumption making men obese or is their obesity provoking them to consume alcohol?

Cross-sectional population studies show contradictory relationships between alcohol intake and body weight. In 2005 Breslow and Smothers analyzed data from the National Health Interview Surveys (NHIS), and they found that BMI was higher in those who consumed four or more alcoholic beverages per drinking day compared to those who had just one drink per day.¹³ However, increasing frequency of alcohol consumption (drinking days per year, month, or week), was correlated with significantly decreased BMI in men (from 27.4 to 26.3 kg/m²) and women (from 26.2 to 24.3 kg/m²). Their analysis controlled for age, race, education, marital status, physical activity, and region.¹³ Similarly, investigating non-smokers from the NHANES III data, Arif et al. (2005) found that heavy drinkers had the greatest odds of overweight and obesity (1.30-1.46) compared to non-drinkers (1.00). The low to moderate drinkers who consumed only one drink per day had the lowest odds of obesity (0.46, 95% confidence interval (CI) 0.34-0.62).³⁴ Rohrer et al. (2005) also found that low-income Americans who consumed alcohol three or more days a month had a lower odds of obesity compared to non-drinkers.³⁵ Liangpunsakul et al. (2010) analyzed NHANES III data to show that female moderate

and binge drinkers had a lower BMI (25.5 and 24.9 kg/m²) and body weight (66.9 and 64.2 kg) than non-drinkers (26.5 kg/m² and 67.2 kg).¹¹ The level of alcohol consumed by men was an independent predictor of BF%; in other words the more alcohol consumed, the lower the body fat.¹¹ In a study on older men in the United Kingdom (UK), Wannamethee et al. (2005) found that alcohol intake was positively associated with BMI, WHR, waist circumference (WC), and BF%. However, these strong associations were seen mainly in beer and spirits and not in wine.³⁶ A recent Spanish study found that men who consumed more than two alcohol drinks per day had significantly higher BMI compared to non-drinkers.³⁷ While some of these studies indicate that alcohol is negatively associated with BMI, others show positive relationships, particularly in men. Few studies differentiated alcohol consumption by frequency and rather than average alcohol intake which may considerably alter the results.

Several studies that investigated frequency of alcohol consumption compared to total alcohol intake suggest that increased frequency may be associated with less central adiposity and body weight. In a Flemish population Duvigneaud et al.'s (2007) study found a positive association between average alcohol intake (kcal/d) and WC in men but not women.³⁸ Dorn et al. (2003) analyzed the Western New York Health Study data: total alcohol intake was positively correlated with abdominal height.¹⁴ However, greater frequency of alcohol intake was associated with lower abdominal heights compared to less frequent but sizable doses of alcohol. A cross-sectional study by Tolstrup et al. (2005) found that total alcohol intake was positively associated with high BMI and large WC in men.¹⁰ Total alcohol consumption was also positively associated with obesity, however keeping volume constant, the highest frequency of drinking had the lowest odds

ratio for obesity.¹⁰ Tolstrup et al.'s study confirmed the findings by Dorn et al.: greater frequency of alcohol intake is associated with less weight and central adiposity. Other studies using total or average alcohol intake found that more alcohol consumed was associated with higher BMI and WC.

The type of alcoholic beverage (e.g. beer, liquor, wine) may also influence the relationship between alcohol intake and WC. Slattery et al. (1992) found that across race and gender, higher beer consumption was associated with elevated WHR compared to liquor and wine. While white women consumed the most wine per week, there was no significant association between wine intake and WHR in this group.³⁹ In a ten-year Danish study, Vadstrup et al. (2003) showed that high total alcohol intake was associated with larger WC.⁴⁰ Wine consumption, on the other hand, was associated with smaller WC when compared to beer and spirits.⁴⁰ Another Danish study by Halkjaer et al. (2004) found no significant association between alcohol type and WC in men. In women, however, there was a positive association between high intakes of beer and liquor and increases in WC.⁴¹ The same researchers studied 50-64 year old men and women (2006) and analyzed their macronutrient intake (including alcohol) and changes in WC after five years.⁴² Increases in WC had a "U" shaped association with wine intake indicating that non-drinkers and heavy wine drinkers increased their WC significantly more than those who partook in moderate amounts of wine (1-2 glasses per day).⁴² A 2005 study in France by Lukasiewicz et al. found that men and women who consumed less than one glass of wine per day had a lower WHR than non-drinkers and those who drank more than a glass per day.⁴³ As liquor intake increased, both WHR and BMI increased. No significant association was found with beer consumption, but this could be due to the low

number of people who consumed beer regularly.⁴³ Drinking regular, moderate amounts of alcohol have been shown to confer cardiovascular health benefits,⁴⁴ furthermore, these studies suggest that wine in particular may also protect against waistline and weight gain.

Longitudinal and cross-sectional studies have the benefit of analyzing lifestyle habits and determining correlations in a population. Although more challenging, controlled experimental trials are necessary to determine causal relationships between alcohol intake and weight gain and body composition. In 1984 Crouse and Grundy hospitalized 12 men for ten weeks to investigate the effects of alcohol in their diet. After four weeks of a controlled calorie diet, the men were given an additional 630 kcal of alcohol for the following six weeks.⁴⁵ Seven participants lost weight (average 0.85 kg) after alcohol was added to their diet while two men gained an average of 1.5 kg. The weight gain was only observed in men who were already obese or overweight at baseline.⁴⁵ A crossover trial of 14 free living men by Cordain et al. (1997) found that consuming wine did not adversely affect body weight or promote obesity. For six weeks participants consumed 270 ml of red wine with their evening meal and then abstained for six weeks or vice versa. Wine intake did not cause any significant changes in body fat, skinfold thickness, body weight, resting metabolic rate, or caloric intake.⁴⁶ While these results could be due to the short experimental time period, they serve as an indication that consuming wine does not seem to promote weight gain in the short term. Likewise, a randomized control trial in Germany conducted by Flechtner-Mors et al. (2004) placed 40 overweight and obese individuals on a calorie-restricted diet (1500 kcal/d) where 10% of their energy was consumed from grape juice or white wine.⁴⁷ Over 12 weeks both groups lost a significant amount of weight (~4%) in addition to reducing BF%, WC, blood

pressure, and blood glucose levels. In this study, wine consumption did not appear to impair or promote weight loss compared to isocaloric juice. A Spanish study by Romeo et al. (2007) found that 30 days of alcohol abstinence followed by 30 days of moderate beer consumption did not significantly influence weight or skinfold measures, save for one exception. There was a significant increase in bicep skinfold measurements in men after the beer consumption in men but not in women.⁴⁸ Randomized controlled trials investigating alcohol and body weight are scarce. However, these few trials imply that consuming alcohol (primarily wine) does not promote weight gain while keeping most other factors stable.

Multiple studies have suggested that moderate wine consumption is associated with less weight gain and normal-weight BMIs.^{31,36,39,42,43,46,47} Resveratrol, a phytochemical in wine, may offer protection against fat accumulation. In human adipocytes, resveratrol inhibits de novo lipogenesis as well as down-regulates lipogenic gene expression.⁴⁹ In mice, resveratrol supplementation protects against weight gain from a high-fat diet.⁵⁰ Additionally, resveratrol increases insulin-mediated glucose uptake thus effectively helping to reduce blood glucose levels.⁴⁹ The bioavailability of resveratrol from red wine is low and it is independent of its ingestion with a meal. The combination of antioxidants and alcohol in red wine is most likely responsible for its apparent benefits over beer and liquor.⁵¹

Lifestyle Confounders and Survey Validity

Suter et al. (2005)²² compiled a list of potential confounders on studies examining the effect of alcohol and body weight. These confounders are current body weight status,

food intake with alcohol, current body composition, absolute versus frequency of alcohol consumed, liver function, smoking status, physical activity, type of beverage (beer, wine, liquor), ethnicity, age, socioeconomic status, gender, and family history. Most longitudinal and cross-sectional population studies account for many of these confounders, however, many times the data are not available.

In addition to the effect of lifestyle confounders, there is also the issue of data quality and accuracy in survey completion. Normal weight adults underreport their energy intake by an average of 16% while obese individuals underreport 41% of their daily calories.⁵² One study found that lean women underreported their food intake because they found weighing and recording their food an arduous task.⁵³ Obese men also underreported their food intake; they used the food recording period as a time for dieting, underestimated their portion sizes, and simply omitted some food from their log.⁵⁴

In addition to diet habits, alcohol intake is susceptible to misreporting due to social pressures, individual characteristics and method of data collection.⁵⁵ While most clinicians assume that alcohol is underreported, self-reports of drinking can be reliable and valid when assessment situations are structured to minimize bias. Personal interviews compared with self-administered questionnaires have their strengths and limitations. An interviewer presents his or her own level of skill and may influence the interviewee by stimulating subconscious reactions to the interviewer's age, race, or gender.⁵⁶ A survey or questionnaire allows the subject to take the time needed to consider each question as well as answer questions anonymously. However, as most surveys are self-administered there is rarely the opportunity to clarify questions or responses. In addition to social stigma against heavy drinking, there is also the limitation in memory recall and serving size

estimation. According to Conrad and Cashman (1998),⁵⁷ subjects rely on three ways of estimating drinking behavior frequency; recalling and enumerating events, recalling episodes and counting the number of occurrences, and converting a general impression into a quantity. Each method of recall can contribute to inaccuracies of enumeration in alcohol intake both in amount and frequency.

CHAPTER III

Methods

Study Population

Beginning in 2007, the Center for Health Discovery and Well Being (CHDWB) Cohort trial recruited a random sampling of Emory University and Emory Healthcare faculty and staff. Five percent of the participants were Emory University board members and individuals who paid to take advantage of the services offered. The inclusion criteria were as follows:

1. Age 18 years and older.
2. No history of hospitalization due to an acute or chronic disease within the previous year (with exception of hospitalization for treatment of accidental trauma).
3. No history of severe Axis 1 psychosocial disorders within the previous year (e.g. delirium, dementia, schizophrenia, depression, bipolar disorder, hypochondriasis, dissociative disorder).
4. No history of addition of new prescription medications to treat a chronic disease condition (with exception of changes of anti-hypertensive or anti-diabetic agents) within the previous year.
5. No history of substance abuse within the previous year.
6. Minimum of two years employment at Emory University or Emory Healthcare.
7. Currently working at least 20 hours/week.
8. Covered by Emory health insurance plans.

Data Collection

The Emory CHDWB obtained informed consent from each volunteer involved in this study. Participants completed several questionnaires to obtain demographic information (e.g. date of birth and gender), lifestyle, and more specific information about tobacco and alcohol use and food consumption. Participants completed both the tobacco and alcohol survey (TOBALC) and the Block Food Frequency Questionnaire (FFQ); the FFQ is a semiquantitative, validated questionnaire including detailed questions about types of tobacco, alcohol, and food, and amount and frequency of consumption. Participants were also surveyed about their physical activity in a typical week using the Cross-cultural Activity Participation Study (CAPS) survey. The CAPS survey covered all daily living activities such as household, lawncare, childcare, walking, dancing, sport, and sleeping. Participants indicated which days (weekdays or weekends) they were active as well as the length of time (hours or minutes) and intensity: light, moderate or vigorous activity. Participants were scanned using a Lunar iDEXA to measure body composition (i.e. body fat, lean mass, and bone density). Weight, height, waist, and hip measurements were also taken to calculate BMI and WHR for each participant.

Data Analysis

Frequency analyses were used to describe the demographic, alcohol intake, physical activity and body composition characteristics of the population. Frequency of beer, liquor, and wine intake were categorized as follows: Never to a few times per year, 1-3 days per month, 1-3 days per week, 3-4 days per week, and 5-7 days per week.

Median values of BF%, WHR, and BMI by alcohol type and frequency of intake were compared for each gender using the Kruskal-Wallis test to determine significance given that the body composition data were not normally distributed. Multiple regression analysis was used to determine the relationship between BF%, WHR and BMI (dependent variables) and independent variables including age, energy intake, physical activity, and alcohol consumption by gender. Statistical analyses were conducted using SPSS (version 20.0 SPSS, Inc.; Chicago, IL).

CHAPTER IV

Results

Population

A total of 700 faculty and staff participated in the study. The demographic characteristics of the total population and after subdivision by gender are shown in Table 2. The majority of the participants were female (66%) and ranged in age from 17 to 82 years (Figure 1). A high percentage of the population was overweight or obese (65.4%), with a mean BMI of 27.9 kg/m². The median BF% for males and females fall within the high level of health risk based on the Tanita[®] health ranges (Table 3). The median WHR for males exceeded the cut-off point for central obesity defined by the World Health Organization (WHO) (≥ 0.90 for men; ≥ 0.85 for women).⁵⁸

Figure 1. Age Distribution of the CHDWB Population

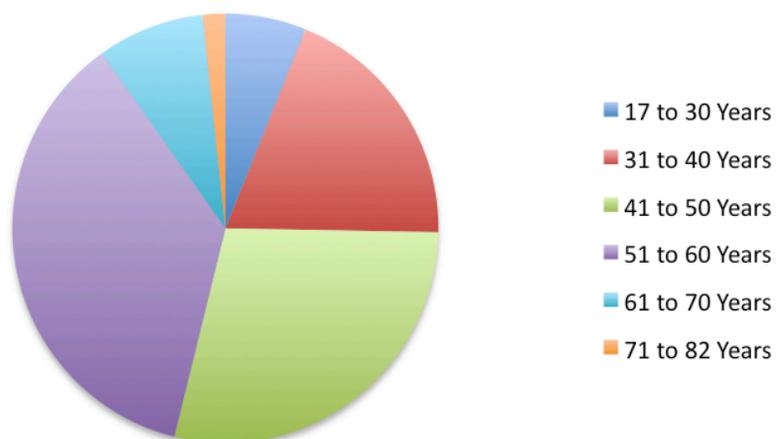


Table 2. Anthropometric Characteristics of the CHDWB Population

	Male	Female	Total
Number of Participants	239	461	700
Age (years)*	51 (41, 58)	48 (40, 55)	49 (40, 56)
BMI (kg/m ²)**	27.4 ± 4.0	28.1 ± 7.3	27.9 ± 6.4
BF%**	30.2 ± 6.5	40.7 ± 8.1	37.1 ± 9.1
WHR**	0.91 ± 0.075	0.79 ± 0.069	0.83 ± 0.092
CHDWB – Center for Health Discovery and Well Being; BMI – body mass index; BF% - body fat percentage; WHR – waist-hip ratio			
*Median (25%,75%), **Mean ±SD			

Table 3. Tanita[®] Body Fat Ranges by Health Risk for Standard Adults

Health Risk by Body Fat Percentage						
Gender	Minimal	Low	Moderate	High	Very	Extremely
(Age in years)	%	%	%	%	High	High %
					%	
Female (18-39)	21-32.9	33-35.9	36-38.9	39-43	43-46	46-50+
Female (40-59)	23-33.9	34-36.9	37-39.9	40-43.9	44-46.9	47-50+
Female (60-99)	24-35.9	36-37.9	38-41.9	42-45.9	46-48.9	49-50+
Male (18-39)	8-19.9	20-21.9	22-24.9	25-28.9	29-31.9	32-50+
Male (40-59)	11-21.9	22-24.9	25-27.9	28-30.9	31-33.9	34-50+
Male (60-99)	13-24.9	25-26.9	27-29.9	30-33.9	34-36.9	37-50+

The median body composition (BMI, BF% and WHR) of the CHDWB population by frequency of beer, liquor, and wine consumption are shown in Tables 4-6. A higher frequency of liquor intake in males was associated with an increase in WHR ($p < 0.05$). No other significant relationships between alcohol intake and body composition were observed in males. In females, the frequency of beer consumption was negatively associated with median BF% ($p = 0.023$) but not BMI or WHR. However, the frequency of liquor consumption was positively associated with WHR ($p = 0.030$). The frequency of wine consumption was negatively associated with both BMI and BF% ($p = 0.000$ and $p = 0.004$, respectively) but not WHR.

The relationship between the outcome variables (BF%, BMI, WHR) and predictor variables (demographic characteristics and alcohol intake) in the CHDWB population is shown in Table 7. Using multiple regression analysis, we determined that 36% of the variance in BF% was predicted by wine intake category ($R = -0.170$), age ($R = 0.121$), gender ($R = 0.549$), and liquor quantity ($R = -0.078$). Although the model was statistically significant ($p < 0.001$) the correlations were weak. The regression model for BMI included wine intake category, total calorie intake, age, and quantity of liquor intake. However, these variables predicted only 6.7% of the variance in BMI. Close to half (44%) of the variability in WHR was predicted by age, gender, total calories, and activity level ($p < 0.01$) but not by alcohol intake. The correlations between age and total calories and WHR were positive but weak ($R = 0.247$ and $R = 0.180$, respectively).

Table 4: Median Body Composition of the CHDWB Population by Gender and Frequency of Beer Consumed

	Category 1: Never to a few days per year	Category 2: 1 to 3 days per month	Category 3: 1 to 2 days per week	Category 4: 3 to 4 days per week	Category 5: 5 to 7 days per week	P-value
Males						
BMI (kg/m ²)*	26.6 (24.4, 30.8)	26.6 (24.9, 29.3)	26.9 (24.9, 28.6)	28.0 (25.7, 30.6)	28.0 (23.6, 30.5)	0.600
BF%*	30.7 (25.5, 35.7)	29.8 (26.3, 33.0)	31.3 (26.8, 35.2)	29.0 (23.7, 34.5)	30.4 (25.3, 34.0)	0.780
WHR*	0.90 (0.86, 0.96)	0.90 (0.85, 0.94)	0.90 (0.86, 0.92)	0.93 (0.87, 0.97)	0.90 (0.86, 0.94)	0.334
Females						
BMI (kg/m ²)*	26.9 (23.1, 32.4)	26.0 (23.4, 31.1)	24.2 (21.7, 31.1)	24.7 (22.7, 29.5)	25.1 (20.9, 26.6)	0.067
BF%*	41.9 (37.3, 46.7)	40.7 (35.4, 46.0)	38.8 (31.6, 43.7)	36.0 (32.6, 42.2)	39.7 (32.0, 43.6)	0.023
WHR*	0.80 (0.73, 0.83)	0.79 (0.73, 0.82)	0.77 (0.73, 0.80)	0.77 (0.74, 0.86)	0.78 (0.75, 0.80)	0.600
CHDWB – Center for Health Discovery and Well Being; BMI – body mass index; BF% - body fat percentage; WHR – waist-hip ratio						
*Median (25%, 75%)						

Table 5: Median Body Composition of the CHDWB Population by Gender and Frequency of Liquor Consumed

	Category 1: Never to few days per year	Category 2: 1 to 3 days per month	Category 3: 1 to 2 days per week	Category 4: 3 to 4 days per week	Category 5: 5 to 7 days per week	P-value
Males						
BMI (kg/m ²)*	26.3 (24.3, 28.5)	28.0 (25.5, 30.1)	27.8 (26.2, 34.6)	27.4 (26.7, 33.6)	26.2 (25.5, 32.8)	0.080
BF%*	30.1 (25.4, 34.9)	30.6 (27.2, 35.0)	29.1 (26.2, 34.6)	31.0 (26.9, 41.3)	32.0 (30.0, 34.8)	0.554
WHR*	0.90 (0.86, 0.93)	0.90 (0.85, 0.94)	0.90 (0.87, 0.96)	0.91 (0.90, 1.00)	0.99 (0.90, 1.04)	0.030
Females						
BMI (kg/m ²)*	26.8 (23.1, 32.3)	25.4 (22.3, 31.5)	23.1 (20.7, 30.0)	29.9 (24.7, none)	25.9 (24.0, 28.7)	0.118
BF%*	41.5 (37.1, 46.6)	39.4 (32.3, 45.7)	39.4 (31.4, 44.0)	45.2 (37.6, none)	41.9 (40.0, 45.8)	0.134
WHR*	0.80 (0.73, 0.84)	0.77 (0.72, 0.81)	0.78 (0.74, 0.81)	0.82 (0.81, none)	0.79 (0.75, 0.82)	0.185
CHDWB – Center for Health Discovery and Well Being; BMI – body mass index; BF% - body fat percentage; WHR – waist-hip ratio						
*Median (25%, 75%)						

Table 6: Median Body Composition of the CHDWB Population by Gender and Frequency of Wine Consumed

	Category 1: Never to few days per year	Category 2: 1 to 3 days per month	Category 3: 1 to 2 days per week	Category 4: 3 to 4 days per week	Category 5: 5 to 7 days per week	P-value
Males						
BMI (kg/m ²)*	26.4 (24.2, 30.8)	28.0 (24.7, 29.5)	27.0 (25.3, 28.7)	27.0 (25.1, 29.0)	26.6 (24.9, 28.4)	0.981
BF%*	30.9 (25.3, 35.9)	30.4 (26.9, 33.4)	29.7 (25.4, 33.3)	30.0 (24.9, 32.7)	30.4 (27.5, 34.8)	0.693
WHR*	0.90 (0.85, 0.95)	0.90 (0.86, 0.94)	0.90 (0.86, 0.92)	0.90 (0.86, 0.95)	0.90 (0.87, 0.99)	0.570
Females						
BMI (kg/m ²)*	27.1 (23.5, 32.6)	27.5 (24.0, 33.2)	25.8 (22.2, 30.9)	23.8 (21.4, 22.8)	24.8 (22.1, 28.7)	0.001
BF%*	42.3 (37.4, 47.0)	41.0 (37.3, 48.5)	40.5 (34.3, 44.2)	37.6 (31.7, 44.8)	39.8 (31.5, 44.1)	0.004
WHR*	0.80 (0.73, 0.83)	0.78 (0.72, 0.83)	0.78 (0.73, 0.82)	0.78 (0.74, 0.82)	0.78 (0.74, 0.82)	0.858
CHDWB – Center for Health Discovery and Well Being; BMI – body mass index; BF% - body fat percentage; WHR – waist-hip ratio *Median (25%, 75%)						

Table 7: Relationship between Demographic Characteristics and Alcohol Intake and Body Composition in the CHDWB Population.

Independent Variables	BMI			BF%			WHR		
	R	Std Coef	P-value	R	Std Coef	P-value	R	Std Coef	P-value
Beer Category* (frequency)	-.087	-.036	.435	-.226	-.039	.304	.113	-.028	0.429
Liquor Category* (frequency)	-.023	-.018	.665	-.102	.000	1.000	.138	.051	0.110
Wine Category* (frequency)	-.152	-.195	.001	-.170	-.170	.001	.060	-.059	0.101
Beer Quantity	-.084	-.077	.138	-.222	-.039	.369	.142	-.005	0.906
Liquor Quantity	.031	.108	.020	.078	.076	.049	.038	-.021	0.555
Wine Quantity	-.042	.050	.321	-.096	.046	.276	.060	.017	0.668
Age	.032	.104	.009	.121	.223	.001	.247	.205	0.001
Gender	.048	.058	.149	.549	.557	.001	-.626	-.593	0.001
Calorie Intake	.113	.154	.001	-.107	.044	.168	.180	.073	0.015
Activity	-.048	-.057	.126	.024	-.038	.220	-.121	-.083	0.005

CHDWB – Center for Health Discovery and Well Being; BMI – body mass index; BF% - body fat percentage; WHR – waist-hip ratio; Std Coef – Standardized Coefficient
 *Category of alcohol intake: 1 to 5 in increasing frequency.

CHAPTER V

Discussion

The frequency of wine intake was an independent predictor of both BMI and BF% but not WHR. As the frequency of wine intake increased, BMI and BF% decreased. The quantity of liquor intake was positively associated with BMI. Age independently predicted BMI, BF%, and WHR while gender predicted BF% and WHR. Calories predicted BMI and WHR while activity level predicted WHR. The median BMI and BF% for women decreased as wine intake frequency progressed from category 1 to category 4. The lowest median BMI and BF% was found with category 4; however there was an increase in both BMI and BF% in the highest frequency, category 5. Similarly beer intake in females was significantly associated with decreasing BF% from category 1 to 4 with the lowest median BF% associated with category 4. In men, frequency of liquor intake was significantly associated with increasing median WHR where it was highest in category 5. We fail to reject the hypothesis that increasing frequency of alcohol, specifically wine, is related to lower BMI and BF%. We also fail to reject the hypothesis that the increase in quantity of liquor alcohol intake is associated with higher BMI and BF%. However, we reject the hypothesis that increased quantities of alcohol are related to increased WHR and central adiposity.

Alcohol-rich meals have been shown to increase thermogenesis 27% more than CHO and fat,²³ therefore a regular intake of wine can contribute to increased energy expenditure and lead to lower BMI and BF% compared to those who do not consume

alcohol regularly. While increasing energy expenditure, alcohol suppresses fat oxidation.^{5,9,23} This was not consistent with the lower BF% found with frequent wine intake. However, the resveratrol in red wine may have decreased adipogenesis in maturing preadipocytes, increased lipolysis, and reduced lipogenesis in mature adipocytes, contributing to a decrease in BF%.⁵⁰ Wine is often consumed with food thereby mitigating its effect on judgment and food choices compared to alcohol alone. When alcohol is consumed 30 minutes before a meal, food consumption, eating rate, and meal duration increase significantly.²⁷ Regular wine consumption may also be indicative of lifestyle or stress factors not included in this study that influence body composition leading to lower BMI and BF%.

While frequency of wine intake was predictive of lower BMI and BF%, it was not predictive of WHR. Instead, WHR was significantly predicted by gender, age, caloric intake, and activity level. Men have a greater central distribution of fat than women.⁵⁸ Women also gain central adiposity postpartum and generally have less lean mass and more fat mass than men. Additionally, post-menopausal women tend to gain fat mass as well as redistribute it to the abdominal area.⁵⁸ As people age, body fat may stay the same or increase, but there is a definite redistribution from subcutaneous fat to visceral depots indicating an increase in WC and WHR.⁵⁸

Age and gender also predicted BF%, but only age and calorie intake predicted BMI. This is consistent with previous research insofar as men have been found to have a higher lean body mass and lower fat mass compared to women after adjusting for height.⁵⁸ These differences can be attributed to sex steroid hormones – a decrease in free testosterone in men is associated with reduced muscle mass and increased fat mass.⁵⁸

Neither frequency nor quantity of beer intake was predictive for BF% or WHR in this study. What might be mistaken for a man's 'beer belly' is simply a result of an aging male with decreased testosterone and activity.

In the comparison of median body composition values, the association with wine intake was significant only in the female population. Women's smaller overall size may explain the differing effects on men and women. Women also have less water per pound of weight than men, and because alcohol disperses in water, women's blood alcohol will be greater than a man's after the same quantity of alcohol intake.¹⁷ Therefore the phytochemical and alcohol content of wine may have a greater effect in women than men. One study showed that adding two glasses of wine per day did not alter healthy males body composition (BMI and BF%).⁴⁶ While this trial was only 6-weeks long, the results could be indicative of wine's dissimilar interactions with each gender. Additionally, men tend to consume alcohol calories in addition to their dietary intake while women substitute alcohol calories for dietary calories without increasing total energy intake.⁵⁹

BMI was significantly predicted by the reported quantity of liquor consumed per drinking episode. Liquor consumed in great quantities can lead to inebriation and poor dietary choices such as high fat, high salt, calorie-dense foods.²⁷ Liquor is often combined with sugar-sweetened mixers like juice and soda which can significantly increase the caloric load for a liquor-based beverage. For example, a margarita or piña colada can provide almost 600 calories in one drink which can increase overall caloric intake, promote weight gain, and increase BMI.

In males, the highest frequency of liquor intake was associated with the highest WHR; this is consistent with the study conducted by Addolorato et al. (1997) that showed

male alcoholics (average alcohol intake of 194 g/d) had significantly higher WHR.⁶⁰

While present results did not show significant associations with wine or beer and WHR, it is possible the amount of alcohol consumed by frequent liquor consumers was similar to that of alcoholics.

Frequent wine intake predicted lower BMI while higher liquor quantity predicted higher BMI. Breslow et al. (2005) also found that increasing frequency of alcohol intake was associated with lower BMI.¹³ While within each frequency quartile, higher quantity was associated with higher BMI: 1 drink had lowest BMI, and 3 drinks highest BMI.¹³ The present study did not investigate overall (total or average) alcohol intake. However it is interesting that similar patterns were found with wine and liquor in this study. Frequent wine intake in this study predicted lower BF%, which is consistent with Liangpunsakul et al. (2010) who found levels of alcohol consumption (g/d) were independent predictor of lower BF% in males.¹¹

The median frequency of liquor intake was associated with WHR in men while liquor quantity independently predicted higher BMI. This is consistent with Lukasiewicz et al. (2005) who also found positive associations between liquor intake and BMI and WHR.⁴³ Also similarly Lukasiewicz et al. (2005) found no association between beer and BMI or WHR. Wannamethee et al. (2005) found positive relationships between alcohol intake and BMI, WHR, and BF% - most clearly observed in beer and liquor consumers.³⁶ Additionally, French et al. (2010) found that frequency and quantity of alcohol consumption were positively associated with BMI in men but not women.⁶¹ Tolstrup et al. (2005) found that the most frequent alcohol drinkers had the lowest OR of being obese. However, the highest total alcohol intake was associated with high BMI and WC.¹⁰

Many studies examined the average alcohol intake (g/d) rather than frequency and quantity specifically; therefore their results cannot be directly compared with this study. Wannamethee et al. (2004) found a significant inverse relationship between alcohol intake (g/d) and BMI in women (after adjusting for dietary and other confounders).⁶² Normal weight women gained less weight when consuming light to moderate alcohol in a 13 year follow up study compared to nondrinkers. Also, Wang et al. (2010) found that the relative risk for becoming overweight or obese decreased with increasing alcohol intake (average g/d).³²

Studies evaluating the effect of alcohol intake on body composition have been inconclusive. Some studies show that alcohol intake is associated with lower odds or risk for overweight and obesity,^{32,34,35} while others show total quantity of alcohol to be associated with higher BMI.^{13,36,37,43,61} These studies have looked at total and average alcohol intake and they rarely specify the type of alcohol imbibed as in this current study.

This project had several limitations. The study did not look at total or average alcohol intake but rather specified alcohol by type: wine, liquor, and beer. This limited the comparisons of results with other studies. Participants with alcoholic consumption habits may have been identified and analyzed separately if total alcohol had been calculated in this study. The interaction of frequency and quantity of alcohol was not investigated. Several confounding factors were not taken into account; ethnicity, alcohol consumed with/without meals, smoking status, alcohol history, and weight history. Previous studies show that different ethnicities have tendencies for specific body type and body composition.⁵⁸ Having incomplete data on ethnic backgrounds in the current study did not allow for complete analysis of variations due to ethnic differences. Concerning

whether alcohol was consumed with or without food may not make a difference.

Wannamethee et al.'s study in 2005 showed positive association between alcohol intake (mainly beer and liquor) and BMI, WHR, and BF% regardless of whether the alcohol was drunk with or without a meal.³⁶ However this population was elderly men ages 60-79 y, unlike this present study.

This study was a cross-sectional analysis of free living adults. It did not gather data on previous weight or weight gain or history of alcohol intake. Some studies have found that weight history significantly predicted future weight gain. Concerning alcohol and body weight, non-drinkers tend to gain more weight than alcohol consumers.²⁹ Also, the FFQ collected data on typical food and beverage intakes but did not include questions concerning whether beverages were consumed with or without food, or over a long or short period of time. The method of consumption may affect the daily caloric intake, which can lead to changes in body weight. This study also did not account for smoking status of participants. Limited data were available on the participants and the sample size would have been greatly reduced if the smokers were to be excluded or analyzed separately. Moreover, it was difficult to determine if each participant was a current smoker or previous smoker, which would have significant influence on their weight and BMI. Smokers tend to have lower body weight and BMI while those who have quit smoking tend to gain weight.⁶³ Smokers also have more visceral fat leading to increased WC and subsequent WHR. The paradoxical high WHR with low BMI is frequent in smokers compared to nonsmokers.⁶³

Further limitations are the inaccuracies due to limited memory or biased recall when answering surveys and questionnaires. During the initial assessment, participants

fill out many surveys including the lengthy semi-quantitative Block FFQ. The process of completing numerous questionnaires is tiring and can affect the mood, memory, and alter participants' accuracy in answering questions. The surveys concerning alcohol intake were also subject to bias from the effect of social desirability. The drive for social approval and acceptance may cause some individuals to alter their report of alcohol intake frequency or quantity, thereby masking true relationships or showing spurious relationships.⁶⁴ While these surveys were self-administered on the computer, the effects from social biases may have been reduced but still present.

Furthermore, the FFQ included non-alcoholic beer with its beer intake question. As discussed previously, alcoholic beer and non-alcoholic have differing effects on appetite and metabolism. Therefore, if a substantial number of participants primarily consumed non-alcoholic beer, these analyses would not accurately capture the effects of alcohol intake in the form of beer and their relationship with body composition.

Emory University and Emory Healthcare employees were recruited by random selection each year to take part in the health study. Once recruited, individuals could choose to participate or not. In this way there was self-selection for those individuals who were willing take the time and energy to engage in the tests and procedures involved in the study. This sample population may not be representative of the larger university employee population who may not have the time or willingness to be involved.

The sample of Emory employees was relatively small, only 700 participants with baseline data. Therefore there were not equal portions of participants in each category of alcohol intake. The data were positively skewed therefore they were not parametric and medians were used for analysis. The median WHR for males who consumed category 1

through 4 was between 0.90 and 0.91, but only at category 5 did it increase to 0.99. This could be due to small number of people (n=8) reporting alcohol intake in the highest frequency. There were also not enough participants to analyze each age group separately. Age showed significant association with body composition, it is possible that the analyses used could not tease out alcohol effects.

Conclusion

These data show that Emory employees are over the WHO cut-off for WHR and are at increased risk for chronic diseases. This study found that high quantity of liquor independently predicts higher BMI. In males the higher frequency of liquor intake was significantly associated with higher WHR. High BMI and central adiposity are components of metabolic syndrome which can significantly raise the risk for cardiovascular disease and diabetes. The average male and female participants were in the high health risk range for BF%. Normal weight obesity increases risk for central adiposity, hypertension, dyslipidemia, and elevated blood glucose levels. A review of the results with Emory employees along with educational sessions and health programming would help improve wellness and reduce risk for chronic diseases. Regular wine intake (5-7 d/wk) may have benefits including lower BMI and BF%. There is evidence that regular wine intake is associated with lower rates of CHF incidents.⁴⁴ Longitudinal studies in this population would be beneficial to determine if regular wine intake is associated with cardioprotective effects.

To investigate interactions between alcohol and body composition over time, future research should include revisiting this population each year to analyze trends in

BF%, BMI, and WHR along with any changes in alcohol drinking pattern. Furthermore, analysis could be continued in each age subgroup to find the odds ratio of overweight and obesity within each alcohol intake category. Considering the limitations of cross-sectional studies and lack of control over variations, randomized control trials would be useful to determine any causative effects of alcohol intake. By controlling alcohol intake, caloric intake, and physical activity, outcomes could be measured concerning cardiovascular and metabolic health as well as body composition.

REFERENCES

1. Evans EM, Mojtahedi MC, Thorpe MP, et al. Effects of protein intake and gender on body composition changes: a randomized clinical weight loss trial. *Nutr Metab.* 2012;9(55):1-9.
2. Jéquier E, Tappy L. Regulation of body weight in humans. *Physiol Rev.* 1999;79:451-480.
3. Block G, Dresser CM, Hartman AM, Carroll MD. Nutrient sources in the American diet: quantitative data from the NHANES II survey. *Am J Epidemiol.* 1985;122:27-40.
4. Siler S, Neese R, Hellerstein M. De novo lipogenesis, lipid kinetics, and whole-body lipid balances in humans after acute alcohol consumption. *Am J Clin Nutr.* 1999;70:928-936.
5. Murgatroyd P, Van-de -Ven M, Goldberg G, Prentice A. Alcohol and the regulation of energy balance: overnight effects on diet-induced thermogenesis and fuel storage. *Br J Nutr.* 1996;75:33-45.
6. Sonko B, Prentice A, Murgatroyd P. Effect of alcohol on postmeal fat storage. *Am J Clin Nutr.* 1994;59:619-625.
7. Suter P, Schutz Y, Jéquier E. The effect of ethanol on fat storage in healthy subjects. *New Engl J Med.* 1992;326:983-987.
8. Levine J, Harris M, Morgan MY. Energy expenditure in chronic alcohol abuse. *Eur J Clin Invest.* 2000;30:779-786.
9. Addolorato G, Capristo E, Greco AV, et al. Energy expenditure, substrate oxidation, and body composition in subjects with chronic alcoholism: new findings from metabolic assessment. *Alcohol Clin Exp Res.* 1997;21:962-967.
10. Tolstrup J, Heitmann B, Tjønneland A, Overvad O, Sørensen T, Grønbaek M. The relation between drinking pattern and body mass index and waist and hip circumference. *Int J Obesity.* 2005;29:490-497.
11. Liangpunsakul S, Crabb D, Qi R. Relationship between alcohol intake, body fat, and physical activity – a population-based study. *Ann Epidemiol.* 2010;20:670-675.

12. US Department of Agriculture, US Department of Health and Human Services. Dietary Guidelines for Americans 2010. 2010. Available at: <http://www.health.gov/dietaryguidelines/dga2010/DietaryGuidelines2010.pdf>. Accessed November 19, 2013.
13. Breslow R, Smothers B. Drinking patterns and body mass index in never smokers: National health interview survey, 1997-2001. *Am J Epidemiol.* 2005;161:368-376.
14. Dorn J, Hovey K, Muti P, et al. Alcohol drinking patterns differentially affect central adiposity as measured by abdominal height in women and men. *J Nutr.* 2003;133:2655-2662.
15. Istvan J, Murray R, Voelker H. The relationship between patterns of alcohol consumption and body weight. *Int J Epidemiol.* 1995;24:543-546.
16. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Normal weight obesity: a risk factor for cardiometabolic dysregulation and cardiovascular mortality. *European Heart Journal.* 2010;31:737-746.
17. National Institutes of Health. Rethinking drinking: Alcohol and your health. 2010.
18. Gropper SS, Smith JL, Groff JL. Advanced nutrition and human metabolism. 5th ed. Wadsworth Cengage Learning; 2009.
19. Lieber C. Medical and Nutritional Complications of Alcoholism: Mechanisms and Management. New York: Plenum; 1992.
20. Lieber C. Microsomal ethanol-oxidizing system (MEOS), the first 30 years (1968-1998)—a review. *Alcohol Clin Exp Res.* 1999;23:991-1007.
21. Oneta C, Lieber C, Li J, et al. Dynamics of cytochrome P4502E1 activity in man: induction by ethanol and disappearance during withdrawal phase. *J Hepatol.* 2002;36:47-52.
22. Suter P. Is alcohol consumption a risk factor for weight gain and obesity? *Rev Clin Lab Sci.* 2005;42:197-227.
23. Raben A, Agerholm-Larsen L, Flint A, Holst J, Astrup A. Meals with similar energy densities but rich in protein, fat, carbohydrate, or alcohol have different effects on energy expenditure and substrate metabolism but no appetite and energy intake. *Am J Clin Nutr.* 2003;77:91-100.
24. Suter P, Jéquier E, Schutz Y. Effect of ethanol on energy expenditure. *Am J Physiol.* 1994;266:R1204-R1212.
25. You M, Fischer M, Deeg M, Crabb D. Ethanol induces fatty acid synthesis pathways by activation of sterol regulatory element-binding protein (SREBP). *J Bio Chem.* 2002;277:29342-29347.

26. Yeomans M, Phillips M. Failure to reduce short-term appetite following alcohol is independent of beliefs about the presence of alcohol. *Nutr Neurosci.* 2002;5:131-139.
27. Westerterp-Plantenga M, Verwegen C. The appetizing effect of an aperitif in overweight and normal-weight humans. *Am J Clin Nutr.* 1999;69:205-212.
28. Caton S, Ball M, Ahern A, Hetherington M. Dose-dependent effects of alcohol on appetite and food intake. *Physiol Behav.* 2004;81:51-58.
29. Liu S, Serdule M, Williamson D, Mokdad A, Byers T. A prospective study of alcohol intake and change in body weight among US adults. *Am J Epidemiol.* 1994;140:912-920.
30. Wannamethee S, Shaper A. Alcohol, body weight, and weight gain in middle-aged men. *Am J Clin Nutr.* 2003;77:1312-1317.
31. Mozaffarian D, Hao T, Rimm E, Willett W, Hu F. Changes in diet and lifestyle and long-term weight gain in women and men. *New Engl J Med.* 2011;364:2392-2404.
32. Wang L, Lee IM, Manson JE, Buring JE, Sesso HD. Alcohol consumption, weight gain, and risk of becoming overweight in middle-aged and older women. *Arch Intern Med.* 2010;170:453-461.
33. Barry D, Petry NM. Associations between body mass index and substance use disorders differ by gender: results from the National Epidemiologic Survey on alcohol and related conditions. *Addict Behav.* 2009;34:51-60.
34. Arif A, Rohrer J. Patterns of alcohol drinking and its association with obesity: data from the third national health and nutrition examination survey, 1988-1994. *BMC Public Health.* 2005;5(126):1-6.
35. Rohrer JE, Rohland BM, Denison A, et al. Frequency of alcohol use and obesity in community medicine patients. *BMC Fam Pract.* 2005;6(17):1-8.
36. Wannamethee SG, Sharper AG, Whincup PH. Alcohol and adiposity: effects of quantity and type of drink and time relation with meals. *Int J Obesity.* 2005;29:1436-1444.
37. Alcacera MA, Marques-Lopes I, Fajo-Pascual M, et al. Lifestyle factors associated with BMI in a Spanish graduate population: the SUN study. *Obes Facts.* 2008;1:80-87.
38. Duvigneaud N, Wijndaele K, Matton L, et al. Dietary factors associated with obesity indicators and level of sports participation in Flemish adults: a cross-sectional study. *Nutr J.* 2007;6:26-38.

39. Slattery M, McDonald A, Bild D, et al. Associations of body fat and its distribution with dietary intake, physical activity, alcohol and smoking in blacks and whites. *Am J Clin Nutr.* 1992;55:943-949.
40. Vadstrup ES, Petersen L, Sørensen TIA, Grønbaek M. Waist circumference in relation to history of amount and type of alcohol: results from the Copenhagen City Heart Study. *Int J Obesity.* 2003;27:238-246.
41. Halkjær J, Sørensen TIA, Tjønneland A, et al. Food and drinking patterns as predictors of 6-year BMI-adjusted changes in waist circumference. *Br J Nutr.* 2004;92:735-48.
42. Halkjær J, Tjønneland A, Thomsen B, et al. Intake of macronutrients as predictors of 5-y changes in waist circumference. *Am J Clin Nutr.* 2006;84:789-797.
43. Lukasiewicz E, Mennen LI, Bertrais S, et al. Alcohol intake in relation to body mass index and waist-to-hip ratio: the importance of type of alcoholic beverage. *Public Health Nutr.* 2005;8:315-20.
44. Kloner RA, Rezkalla SH. To drink or not to drink? That is the question. *Circulation.* 2007;116:1306-1317.
45. Crouse JR, Grundy SM. Effects of alcohol on plasma lipoproteins and cholesterol and triglyceride metabolism in man. *J Lipid Res.* 1984;25:486-496.
46. Cordain L, Bryan ED, Melby CL, Smith MJ. Influence of moderate daily wine consumption on body weight regulation and metabolism in healthy free-living males. *J Am Coll Nutr.* 1997;16:134-139.
47. Flechtner-Mors M, Biesalski HK, Jenkinson CP, et al. Effects of moderate consumption of white wine on weight loss in overweight and obese subjects. *Int J Obesity.* 2004;28:1420-1426.
48. Romeo J, González-Gross M, Wärnberg J, et al. [Does beer have an impact on weight gain? Effects of moderate beer consumption on body composition]. *Nutr Hosp.* 2007;22:223-228.
49. Fischer-Posovszky P, Kukulius V, Tews D, et al. Resveratrol regulates human adipocyte number and function in Sirt1-dependent manner. *Am J Clin Nutr.* 2010;92:5-15.
50. Baile CA, Yang J-Y, Rayalam S, et al. Effect of resveratrol on fat mobilization. *Ann NY Acad Sci.* 2011;1215:40-47.
51. Vitaglione P, Sforza S, Galaverna G, et al. Bioavailability of trans-resveratrol from red wine in humans. *Mol Nutr Food Res.* 2005;49:495-504.

52. Westerterp KR, Goris AH. Validity of the assessment of dietary intake: problems of misreporting. *Curr Opin Clin Nutr Metab Care*. 2002;5:489-93.
53. Goris AH, Westerterp KR. Underreporting of habitual food intake is explained by undereating in highly motivated lean women. *J Nutr*. 1999;129:878-882.
54. Goris AH, Westerterp-Plantenga MS, Westerterp KR. Undereating and underrecording of habitual food intake in obese men: selective underreporting of fat intake. *Am J Clin Nutr*. 2000;71:130-134.
55. Del Boca FK, Darkes J. The validity of self-reports of alcohol consumption: State of the science and challenges for research. *Addiction*. 2003;98(Suppl. 2):1-12.
56. Del Boca FK, Noll JA. Truth or consequences: the validity of self-report data in health services research on addictions. *Addiction*. 2000;95(Suppl. 3):S347-S360.
57. Conrad FG, Cashman NR. Strategies for estimating behavioral assessment. *Memory*. 1998;6:339-366.
58. World Health Organization. Waist circumference and waist-hip ratio: Report of a WHO expert consultation. 2008.
59. Colditz GA, Giovannucci E, Rimm EB, et al. Alcohol intake in relation to diet and obesity in women and men. *Am J Clin Nutr*. 1991;54:49-55.
60. Addolorato G, Capristo E, Marini M, et al. Body composition changes induced by chronic ethanol abuse: evaluation by dual energy x-ray absorptiometry. *Am J Gastroenterol*. 2000;95:2323-2327.
61. French MT, Norton EC, Fang H, Maclean J. Alcohol consumption and body weight. *Health Econ*. 2010;19:814-832.
62. Wannamethee SG, Field AE, Colditz GA, Rimm EB. Alcohol intake and 8-year weight gain in women: A prospective study. *Obes Res*. 2004;12:1386-1396.
63. Chioloro A, Faeh D, Cornuz J. Consequences of smoking for body weight, body fat distribution, and insulin resistance. *Am J Clin Nutr*. 2008;87:801-809.
64. Podsakoff PM, MacKenzie SB, Lee J-Y, Podsakoff NP. Common method biases in behavioral research: A critical review of the literature and recommended remedies. *J Appl Psych*. 2003;88:879-903.