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COMPARING ANTHROPOMETRIC METHODS TO QUANTIFY RELATIONS BETWEEN
ADIPOSITY AND HEADACHE

Thesis Defense
presented in partial fulfillment of requirements
for the degree of Master of Arts
in the Department of Psychology
The University of Mississippi

By

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ABSTRACT

Obesity is associated with increased risk for chronic migraine and migraine progression, but associations with episodic migraine (EM) and episodic tension-type headache (TTH) are unclear. Most studies have relied on BMI as an indicator of adiposity. More accurate anthropometric measures that distinguish adipose tissue from other body tissue are critical to examine adiposity-headache associations, including validated measures of abdominal adiposity and established measurement formulas such as body adiposity index (BAI) and body composition equations developed by Peterson et al. (2003) and Garcia et al. (2005). The present study explored adiposity-headache associations by employing established anthropometric measures of adiposity and comparing individuals with migraine, with TTH, and without headache.

Participants were 109 young adults meeting ICHD-3 criteria for TTH or migraine, or without headache. Ninety-three percent of migraineurs had EM, and 92.5% of TTH sufferers had ETTH. Researchers measured each participant and calculated adiposity as a function of: BMI, waist circumference, BAI, waist-to-hip ratio, and the aforementioned body composition equations. Headache severity and frequency were obtained via diagnostic interview (SDIH-3), and headache-related disability was assessed by the Headache Impact Test (HIT-6).

MANOVA and a subsequent MANCOVA did not reveal significant differences in adiposity between migraine, TTH, and non-headache groups. Regression analyses indicated that among migraineurs, adiposity accounted for 11%, 13%, and 10% of the variance in headache severity, frequency, and disability, respectively, though these proportions were not statistically

significant. Among participants with TTH, adiposity accounted for 8% ($p = .82$), 21% ($p = .23$), and 39% ($p = .009$) of the variance in headache severity, frequency, and disability. The association with disability among those with TTH fell short of significance after Bonferroni correction for multiple comparisons.

Adiposity did not differ between headache groups, and no significant associations were found between adiposity and headache frequency, severity, and disability. Findings extend upon existing literature that has established a positive association between obesity and chronic headache, suggesting that adiposity may not be a distinguishing characteristic among individuals with EM and ETTH. Longitudinal studies that employ gold standard methods of adiposity measurement among diverse samples are needed to further clarify the role of adiposity in headache.

LIST OF ABBREVIATIONS

CM	Chronic Migraine
EM	Episodic Migraine
TTH	Tension-type Headache
ETTH	Episodic Tension-type Headache
CTTH	Chronic Tension-type Headache
ICHD	International Classification of Headache Disorders
BMI	Body Mass Index
BAI	Body Adiposity Index
PHQ-9	Patient Health Questionnaire-9
GAD-7	General Anxiety Disorder-7
PSS-10	Perceived Stress Scale-10
HIT-6	Headache Impact Test-6
SDIH-3	Structured Diagnostic Interview for Headache-3

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I. INTRODUCTION

Migraine and Tension-Type Headache: Definitions and Impact

The International Classification of Headache Disorders (ICHD-3; International Headache Society, 2013) differentiates the diagnostic criteria for all headache disorders, including migraine and tension-type headache (TTH). Migraine is characterized by a unilateral location, pulsating quality, moderate or severe pain intensity, and/or interference with routine physical activity. Headache attacks last 4-72 hours and may be accompanied by nausea, vomiting, photophobia, and/or phonophobia. While a diagnosis of episodic migraine (EM) requires at least five lifetime attacks, a diagnosis of chronic migraine (CM) requires at least 15 days with headache per month for a period of several months.

Migraine is a common neurological disorder that can be very disabling, with a 12% lifetime prevalence in the U.S (Lipton et al., 2007). Migraine is three times more common among women than men (18% vs. 6%, respectively; Lipton et al., 2007; Lipton, Bigal, Hamelsky, & Scher, 2008). Although migraine can affect people throughout their lives, peak prevalence is between the ages of 30-39 among both men and women, with lowest occurrence in adults aged 60 or over. Research has consistently confirmed that individuals with migraine experience many negative consequences as a result of the disorder, including reduced quality of life (Lipton, Hamelsky, Kolodner, Steiner, & Stewart, 2000; Terwindt et al., 2000), reduced work performance (Von Korff, Stewart, Simon, & Lipton, 1998), reduced school functioning (Smitherman, McDermott, & Buchanan, 2011), frequent medical visits (Edmeads et al., 1993), and negative impact on family relationships.

Tension-type headache (TTH) is characterized by symptoms typically opposite those of migraine, including bilateral location, a pressing or tightening quality, and mild to moderate intensity; TTH is not aggravated by routine physical activity. Headaches last from 30 minutes to 7 days and may be accompanied by photophobia or phonophobia (but not both). ICDH-3 specifies three different types of TTH based on headache frequency: infrequent episodic TTH occurring one day or less per month, frequent episodic TTH occurring 1-14 days per month, and chronic TTH with 15 or more headache days per month.

Tension-type headache is the most common primary headache disorder, affecting an estimated 38% of the U.S. population annually (Schwartz, Stewart, Simon, & Lipton, 1998); similar rates are found in other countries (Da Costa, Soares, & Heinisch, 2002; Pop, Gierveld, Karis, & Tiedink, 2002). Lifetime prevalence may be as high as 87% (Lyngberg, Rasmussen, Jørgensen, & Jensen, 2005). Although similar to migraine in being more common among females and having a peak prevalence between the ages of 30 and 39, gender differences in TTH prevalence are less pronounced, with the male:female ratio being 1:1.1-1.4 (Lyngberg et al., 2005).

Comorbidities of Migraine and TTH

Extensive literature supports an association of migraine with psychiatric disorders, including major depressive disorder, panic disorder, phobias, and bipolar disorder (Baskin, Lipchik, & Smitherman, 2006; Baskin & Smitherman, 2009; Jette, Patten, Williams, Becker, & Wiebe, 2008; Lipton, Hamelsky, Kolodner, Steiner, & Stewart, 2000; Ratcliffe, Enns, Jacobi, Belik, & Sareen, 2009). Other comorbid disorders include generalized anxiety disorder and obsessive-compulsive disorder, although prevalence of these comorbid disorders among migraineurs varies between studies (Breslau, Davis, & Andreski, 1991; Swartz, Pratt, Armenian, Lee, Eaton, 2000; Jette et al., 2008; Ratcliffe et al., 2009). Studies comparing EM and CM have

found that rates of psychiatric disorders are higher among individuals with CM (Antonaci et al., 2011). Migraineurs are also at increased risk for sleep disorders, especially insomnia (Rains & Poceta, 2006).

Depression, anxiety, and panic disorder are more prevalent among individuals with TTH compared to non-headache controls, particularly those with CTTH (Crystal & Robbins, 2010), as some studies have found no difference in psychiatric comorbidity rates between individuals with ETTH and those without headache (Merikangas, Stevens, & Angst, 1993; Merikangas, 1994). Fatigue and sleep disorders have also been found to be more prevalent among TTH sufferers than non-headache controls (Rasmussen, 1993).

In addition to these psychiatric comorbidities, researchers have most recently shown interest in the comorbidity between obesity and headache.

Obesity

Obese and overweight denote ranges of weight that the Centers for Disease Control and Prevention has established as greater than a healthy weight for a given height (“Defining,” 2012). Both weight ranges increase risk for numerous diseases and health problems. Although the body mass index (BMI) is not a direct measure of adipose tissue, the CDC defines overweight and obese categories according to BMI values. BMI is a function of the ratio of weight to height and calculated via the following equation: $BMI = \text{Weight}(\text{kg}) / \text{Height}(\text{m})^2$. BMI values between 18.5 and 24.9 indicate a healthy weight, 25 - 29.9 overweight, and 30 or greater obese. The American Heart Association categorizes BMI values between 35 and 39.9 as “moderate” obesity and BMI values of 40 or greater as “severe” obesity (Cornier et al., 2011). The World Health Organization defines abdominal obesity – an alternative measure of obesity – as a waist circumference greater than 88 centimeters (for women) and as a waist circumference greater than 102 centimeters (for men; Peterlin, Rosso, Rapoport, & Scher, 2010).

Using BMI as the metric, obesity prevalence among youth and adults in the U.S. has been increasing for the past several decades. Currently, an estimated 34.9% of U.S. adults are obese, along with 16.9% of U.S. children and adolescents (Ogden, Carroll, Kit, & Flegal, 2014). These rates represent nearly three-fold increases since the 1960s, when the adult obesity rate was 13% and the child rate was approximately 5%. In addition to the drastic rise in obesity rates, the number of children and adults who are overweight has also increased dramatically. An estimated 33.6% of adults in the U.S. are currently overweight but not obese, making the total number of adults who are above a healthy weight approximately 68.5%. A similar trend is seen among children, with 14.9% being overweight but not obese and 31.8% weighing above a healthy weight (Ogden et al., 2014).

The National Institutes of Health (NIH) has declared obesity a major public health challenge in the U.S., citing obesity as a major contributor to preventable death. Obesity substantially increases risk for hypertension, type 2 diabetes, coronary heart disease, dyslipidemia, stroke, osteoarthritis, gallbladder disease, sleep apnea, respiratory problems, and certain cancers (Expert Panel, 1998). Most recently, headache researchers have attempted to ascertain the role of obesity in headache disorders.

Obesity and Headache

A relationship between obesity and headache was first suggested in 2000. In a population based cross-sectional study, Brown, Mishra, Kenardy, and Dobson (2000) examined a sample of 14,779 women between the ages of 18 and 23 who self-reported their height and weight along with various medical conditions and symptoms. Obese women, as defined by a BMI of 30 or greater, were 47% more likely to report headache than women with a BMI less than 30. In this study, Brown and colleagues did not differentiate among headache diagnoses. In another

population-based study with over 55,000 participants and self-reported height, weight, and headache frequency, Scher, Stewart, Ricci, and Lipton (2003) found the prevalence of chronic daily headache to be higher (OR = 1.34) among individuals with BMIs greater than 30 compared to those with BMIs within the normal range. An additional finding in Scher et al.'s study was that among participants who had reported episodic headache at baseline, those with BMIs greater than 30 were five times as likely as those with normal BMIs to have progressed to chronic daily headache at 11-month follow-up, suggesting that obesity may be a risk factor for headache chronification.

Obesity and Migraine

Most of the research on obesity and headache has specifically focused on associations between obesity and migraine prevalence, and few studies have differentiated episodic from chronic migraine in their samples. However, these studies have consistently found a positive association between BMI and migraine prevalence. Ford, Li, Pearson, Zhao, Strine, and Mokdad (2008) examined data from the National Health and Nutrition Examination Survey 1999–2002, which included over 7000 U.S. men and women ages 20 and older. Although Ford et al. (2008) directly measured height and weight, they did not differentiate headache diagnoses or adhere to ICHD criteria. Rather, they asked participants, “During the past 3 months, did you have severe headaches or migraines?” Logistic regression analyses confirmed that participants with BMIs greater than 30 were 37% more likely (OR = 1.37) than those of normal weight to endorse the occurrence of severe headaches or migraines. In a study of 3,733 women who were in the early stages of pregnancy, Vo, Ainalem, Qiu, Peterlin, Aurora, and Williams (2011) also found that obese women were more likely than women of normal weight to have had a diagnosis of migraine (OR = 1.48). Height and weight were self-reported in this study, and migraine was

defined as a positive response to the question, “Has a doctor ever told you that you have migraine headache?” Additionally, analyses revealed that migraine prevalence increased as BMI increased, such that morbidly obese (OR = 2.07; BMI = 35-39.9) and extremely obese (OR = 2.75; BMI \geq 40) women were at increased risk for migraine compared to those of normal weight. Further, women with migraine as children had 1.67-fold increased odds of gaining at least 10 kilograms during adulthood, suggesting that migraine may also predispose one to obesity.

Age, gender, and adipose tissue distribution affect the association between obesity and migraine. In a U.S. sample of 10,623 men and 11,160 women whose height and weight were directly measured and who responded to the question, “During the past 3 months, did you have severe headaches or migraines?”, Peterlin, Rosso, Rapoport, and Scher (2010) found that obesity (BMI > 30) was associated with increased migraine prevalence among both men (OR = 1.38) and women (OR = 1.2) under the age of 55. They found no association between BMI and migraine prevalence among men and women over the age of 55. In this same study, Peterlin et al. (2010) included waist circumference (WC) as an additional measure of obesity and observed that migraine prevalence in obese participants varied as a function of sex, age, and adipose tissue distribution pattern. Independent of total body obesity (as estimated by BMI), women with abdominal obesity (> 88cm) under the age of 55 were at *increased* risk for migraine (OR = 1.26), but women with abdominal obesity over the age of 55 were at *decreased* risk for migraine (OR = .73). Among men of all ages, abdominal obesity (> 102cm) was not associated with migraine prevalence when controlling for total body obesity. Peterlin et al. concluded that gender differences may be a result of the sexual dimorphism of adipose tissue distribution: Men deposit adipose tissue in the abdominal region throughout adulthood, while women deposit adipose

tissue primarily in the gluteofemoral region until after menopause, at which point they also deposit adipose tissue abdominally (Peterlin et al., 2010).

Although abdominal and total body obesity are associated with an increased risk of migraine among women under age 55, studies have demonstrated consistently that obesity is not associated with increased risk for migraine or migraine-related variables in women over the age of 45 or in men over the age of 55 (Keith, Wang, Fontaine, Cowan, & Allison, 2008; Mattsson, 2007; Peterlin et al., 2013; Peterlin, Rosso, Rapoport, & Scher, 2010; Winter, Berger, Buring, & Kurth, 2009).

Although migraine is not positively associated with obesity among adults of post-reproductive age, both migraine frequency and migraine-related disability are positively associated with BMI among adults over age 18. Tietjen, et al. (2007) collected data from eight U.S. outpatient headache centers on 721 patients who met ICHD-II criteria for episodic or chronic migraine. Participants self-reported their height and weight. Tietjen et al. (2007) found positive associations between BMI and migraine frequency/disability, although these associations were no longer significant when controlling for depression and anxiety. However, compared to migraineurs within a normal BMI range and without depression or anxiety, likelihood of higher migraine frequency increased as a function of BMI for those with depression [normal (OR = 2.63), overweight (OR = 3.26), obese (OR = 4.16)] and for those with anxiety [overweight (OR = 2.17), obese (OR = 1.96)]. Similarly, normal (OR = 4.19), overweight (OR = 6.68), and obese (OR = 7.1) migraineurs with depression were at increased risk for migraine-related disability, as were normal (OR = 2.24), overweight (OR = 6.05), and obese (OR = 3.59) migraineurs with anxiety. Migraineurs with concomitant depression and anxiety were the most likely to have higher migraine frequency and migraine-related disability. Results suggested that

the positive association between BMI and migraine frequency/disability may be largely explained by comorbid depression and anxiety symptoms that commonly occur with both migraine and obesity (Tietjen et al., 2007).

In addition to depression and anxiety being moderators in the positive association between BMI and migraine frequency/disability, race is another potential moderator, although most studies have included primarily White participants in America and European countries. However, Yu et al. (2012) conducted a study of 5,041 adults throughout China, directly measuring participant height and weight and assessing migraine based on ICHD-II criteria. Although no association between BMI and prevalence of migraine was observed among participants with a BMI less than 30, those with a BMI greater than 30 were twice as likely as those with BMIs between 18.5 and 23 to have a diagnosis of migraine (OR = 2.1). However, contrary to studies with primarily White samples, Yu and colleagues found no relationship between BMI and other migraine variables (i.e., severity, frequency, or disability), suggesting that different BMI-migraine associations may exist for different ethnicities. Both obesity rates (Ogden et al., 2014) and body fat distribution patterns (Rahman, Temple, Breitkopf, & Berenson, 2009) vary across races, however, which may influence BMI-migraine associations.

Obesity in Relation to Chronic Migraine and Chronic Tension-Type Headache

Studies to date indicate that CM prevalence is higher among people who are obese compared to those of normal weight, although differences in prevalence of CTTH between those who are obese vs. normal weight are unclear. Bigal and Lipton (2006) surveyed individuals between 18 and 89 who self-reported their height and weight. Unlike prior studies, Bigal and Lipton differentiated CM from CTTH. Of 30,849 participants, 1243 (4%) met criteria for chronic daily headache (CDH), 1.3% with CM and 2.8% with CTTH. BMI had an influence on CM

prevalence, such that while CM prevalence among participants of normal weight was 0.9%, it increased to 1.2% in overweight (OR = 1.4), 1.6% in obese (OR = 1.7), and 2.5% in morbidly obese (OR = 2.2) participants. However, Bigal and Lipton did not find this positive association between BMI and headache among participants with CTTH, as prevalence was not different between normal weight, overweight, and obese, although participants categorized as morbidly obese (BMI > 35) did exhibit a significantly higher rate of CTTH (OR = 1.4).

Schramm, Obermann, Katsarava, Diener, Moebus, and Yoon (2013) also compared the prevalence of CTTH and CM in a population-based study in Germany that included over 7,000 participants with headache. 108 met ICHD-II criteria for CM and 50 met criteria for CTTH. Participants who met criteria for CM were more likely to be obese (using self-reported height and weight) than those with CTTH (OR = 1.86) and without any headache diagnosis (OR = 1.39). However, Schramm et al. also found the positive association between CM and obesity was no longer evident after controlling for acute pain medication use (OR = .85), though it remained among CTTH participants (OR = 1.85).

Further highlighting the role of obesity in frequent headache, Bigal and Lipton (2006) assessed the proportion of CDH sufferers with daily headache as a function of BMI. While 36% of CDH sufferers of normal weight reported daily headaches, 48.7% [OR = 1.5] of obese participants reported daily headaches, as did 51% [OR = 1.7] of the morbidly obese. This positive association between BMI and headache frequency was not observed among participants with CTTH. Similarly, the proportion of chronic migraineurs who reported missing at least three days of work during the previous three months and/or who reported severe pain in more than 50% of attacks was significantly higher among the overweight, obese, and morbidly obese when compared to those of normal weight. Bigal and Lipton (2006) concluded the association between

headache and obesity is stronger for CM than for CTTH, although Schramm et al.'s (2013) findings suggest that this conclusion may be a function of patterns of acute pain medication use.

Obesity and Episodic Migraine

Although research has consistently demonstrated that migraine prevalence is higher among individuals meeting criteria for obesity, most studies did not differentiate between CM and EM. The two existing studies on EM and obesity have yielded conflicting results regarding the association between obesity and migraine prevalence, even though both adhered to ICHD criteria for EM diagnosis and relied on self-reported height and weight for BMI calculations. In a U.S. sample including 3,791 episodic migraineurs, Bigal, Liberman, and Lipton (2006) found the prevalence of EM did not significantly differ as a function of BMI for any age or gender.

However, another U.S. study using data from a nationally representative survey (National Comorbidity Survey Replication; Kessler et al., 2004) that included 188 episodic migraineurs found BMI was positively associated with EM prevalence among females under age 50 (Peterlin et al., 2013). They did not find any association between obesity and migraine prevalence among males. Stratified analyses indicated increased EM prevalence in obese participants compared with normal-weight participants among those who were younger than age 50 (OR = 1.86), white (OR = 2.06), and female (OR = 1.95), suggesting the association between EM prevalence and obesity is strongest among these subgroups.

Both studies also reached different conclusions regarding the association between obesity and EM frequency. Peterlin et al. (2013) found no association between BMI and migraine frequency, while Bigal et al. (2006) found that number of headache days per month increased as a function of BMI. While only 4.4% of the normal weight group had 10-15 headache days per

month, 5.8% of overweight (OR = 1.3), 13.6% of obese (OR = 2.9), and 20.7% of morbidly obese (OR = 5.7) had headache of this high frequency.

Bigal et al. (2006) also found that the proportion of migraineurs reporting severe attacks increased with BMI. 53% of normal-weight participants endorsed severe migraine attacks, while 57% of the overweight (OR = 1.25), 59% of the obese (OR = 1.31), and 65% of the morbidly obese (OR = 1.9) participants had severe attacks. Obese (OR = 1.5) and morbidly obese (OR = 2) participants were also more likely to report missing at least one day of school or work each month due to migraine, indicating greater migraine-related disability. This is the only study to date that has examined migraine severity and disability specifically among episodic migraineurs.

Adiposity Measurement

In research on obesity, including research on headache-obesity associations, BMI is widely used as a measure of adiposity due to its accessibility and simplicity (Cornier et al., 2011). However, BMI is a measure of body mass specifically and thus does not quantify body composition. BMI does not distinguish between lean and fat body mass, making it possible for an individual with a “normal” BMI value to actually have a higher-than-normal percent body fat, or for one with a “high” BMI to have a body composition of mostly lean (non-adipose) tissue. BMI thus inaccurately quantifies the adiposity of many individuals, particularly those with excess adipose tissue. Okorodudu et al. (2010) conducted a meta-analysis of 32 studies examining the accuracy of BMI in terms of adiposity measurement and concluded that BMI had an overall sensitivity of 50%, indicating that half of individuals with excess adipose tissue were misclassified as being of normal weight. However, BMI had a specificity of 90% to identify excess body adiposity, meaning that BMI usually correctly identifies those who are not obese. Among male athletes, Pivarnik, Reeves, and Knous (2007) found that specificity of BMI to

diagnose excess adiposity was only 27%, such that BMI inaccurately categorized 73% of their healthy-weight sample as obese.

In light of the poor sensitivity of BMI, debate exists regarding the most accurate method of measuring adiposity. Cadaver analysis is the only measure of body composition with 100% sensitivity and specificity; thus, no perfect measure exists for use in clinical settings (Hu, 2008). Currently, the most thorough assessment of body composition is a “four-component model” (i.e., four-compartment model), which requires precise measurement and quantification of water, mineral, fat, and protein levels in the body (Fuller, Jebb, Laskey, Coward, & Elia, 1992; Williams et al., 2006). Given the resources and time required, the four-component model has limited clinical utility. In its stead several single-procedure methods have been promoted after being validated against the four-component model, and each is considered to be reliable in estimating percent body fat and/or fat distribution (Hu, 2008). These include dual-energy X-ray absorptiometry (DEXA), densitometry or underwater weighing, air-displacement plethysmography, hydrometry or dilution, and imaging in the form of computerized tomography (CT) and magnetic resonance (MRI) (Hu, 2008). When compared to the four-component model, CT and MRI combined consistently exemplify the highest level of accuracy (Hu, 2008; Ross & Janssen, 2005; Ross, 2003).

Studies examining associations between adiposity and headache would ideally rely on at least one of these methods to determine percent body fat and fat distribution. However, utility lies in more practical and affordable methods of quantifying obesity that are appropriate to clinical settings. Anthropometric measurements are noninvasive, quantitative techniques for determining body fat composition by measuring, recording, and analyzing specific dimensions of the body (Hu, 2008) such as waist circumference, hip circumference, neck circumference, thigh

circumference, ratios (waist-to-hip, waist-to-height, waist-to-thigh), skinfold thickness, and body adiposity index (BAI). Of these, waist circumference, waist-to-hip ratio, BAI, and skinfold thickness equations have the most literature supporting their validity in quantifying overall adiposity and fat distribution.

Waist circumference and waist-to-hip ratio are both acceptable measures of central (abdominal) adiposity, and some evidence suggests that waist circumference may be a superior measure to waist-to-hip ratio (Clasey et al., 1999; Kamel et al., 1999). The World Health Organization (2011) defines abdominal obesity for women as a waist circumference greater than 88 centimeters or a waist-to-hip ratio greater than .85; and for men as a waist circumference greater than 102 centimeters or a waist-to-hip ratio greater than .9. Clasey et al. (1999) compared various anthropometric measures to results of CT scans and found waist circumference to have high correlations with total abdominal fat ($r = .87$ to $.93$) and abdominal visceral fat ($r = .84$ to $.93$). Kamel et al. (1999) found waist-to-hip ratio, waist circumference, and DEXA scans to predict intra-abdominal fat in men equally well as MRI, although among women, DEXA was a more accurate predictor.

The Body Adiposity Index (BAI) is a mathematical formula that uses height and hip circumference to estimate percent body fat (Bergman et al., 2012). The formula is: $BAI = ((\text{hip circumference in cm}) / ((\text{height in m})^{1.5} - 18))$. Bergman and colleagues used DEXA scanners to determine percent body fat in over 1700 Mexican-Americans and derived a formula in comparison to the DEXA data. They then validated their equation using data from a separate study of African-Americans and found a high correlation between BAI and DEXA-determined percent body fat ($R = .85$) and a Lin's concordance correlation coefficient of $.95$. Johnson, Chumlea, Czerwinski, and Demerath (2012) further validated the BAI in a sample of over 600

Whites age 20-50 and concluded that the BAI was a “robust” equation applicable to their sample. In spite of the applicability of the equation to multiple ethnicities and the ease of measurement and calculation, BAI is not as accurate in estimating percent body fat as DEXA scans and CT or MRI and is thus not a true surrogate measure for percent body fat. BAI may, however, be a more accurate predictor than BMI given its greater concordance with percent fat ($\rho_c = 0.752$ vs $\rho_c = 0.445$ for BMI) and given its significantly stronger correlation with DEXA-measured percent fat (Johnson et al., 2012).

Another method of assessing adiposity that has been compared to the four-component model is measurement of skinfold thickness. No universally accepted method for estimating body fat based on skinfolds exists, and many equations have been promoted. All involve pinching the skin with the thumb and index finger and placing a pair of calipers on the fold to measure the thickness of two layers of skin and the underlying fat. In a review of anthropometric methods, Cornier et al. (2011) recommend measuring each skinfold site three times, and taking the average of those three measurements as the final value for that site. Cornier et al. further recommend that variance between readings should not be more than one millimeter, otherwise measurements should continue until three readings agree within one millimeter. The two most widely used methods were published by Durnin and Womersley (1974) and Jackson and Pollock (1978; 1979). However, both of these methods were based on the two-component model, which separates the composition of the body into fat mass and fat-free mass. Since then, researchers have developed different skinfold equations based on the four-component model (Fuller, Jebb, Laskey, Coward, & Elia, 1992), and study results indicate these equations are more accurate than the original methods (Peterson, Czerwinski, & Siervogel, 2003; Garcia et al., 2005).

Peterson, Czerwinski, and Siervogel (2003) determined percent body fat in a sample of over 600 Whites ages 18-55 in the U.S. by using DEXA scans, total body water assessment, and hydrodensitometry – measurements allowing for conformity to the four-component model. To aid in determining skinfold equations, they measured seven skinfold sites (triceps, subscapular, biceps, midaxillary, suprailiac, midthigh, and lateral calf) and five circumferences (abdomen, hip, thigh, calf, and upper arm). Using a series of regression analyses, they developed skinfold equations for women and men to predict percent body fat in adults. The final equations were as follows:

$$\begin{aligned} \text{For men: } \%BF_{\text{new}} &= 20.94878 + (\text{age} \times 0.1166) \\ &- (\text{height} \times 0.11666) + (\text{sum4} \times 0.42696) \\ &- (\text{sum4}^2 \times 0.00159) \end{aligned}$$

$$\begin{aligned} \text{For women: } \%BF_{\text{new}} &= 22.18945 + (\text{age} \times 0.06368) \\ &+ (\text{BMI} \times 0.60404) - (\text{height} \times 0.14520) \\ &+ (\text{sum4} \times 0.30919) - (\text{sum4}^2 \times 0.00099562) \end{aligned}$$

Height is quantified in cm and “sum4” is the sum of the triceps, subscapular, suprailiac, and midthigh skinfold thicknesses. Within a cross-validation group, percent body fat as quantified by the skinfold equations did not differ significantly from percent body fat quantified by the four-component model ($\%BF_{4c}$; Peterson et al., 2003). The authors also validated the widely used Durnin and Womersley (1974; $\%BF_{\text{DW}}$) and Jackson and Pollock (1978; 1979; $\%BF_{\text{JP}}$) equations against the four-component model and found $\%BF_{\text{DW}}$ and $\%BF_{\text{JP}}$ to underestimate percent body fat, with mean underestimations of 2.4-3.1% and 6.6% respectively. Precision, as determined by root mean square error (RMSE) values of predicted $\%BF$ in the cross-validation groups, was similar for all skinfold equations, ranging between 4.6% and 5%. Peterson and colleague’s equation was thus a more accurate and equally precise estimate of percent body fat compared to Durnin and Womersley’s and Jackson and Pollock’s equations.

Garcia et al. (2005) developed a skinfold equation that also took bone-breadths (chest, elbow, knee, wrist, ankle), additional skinfolds (chin, biceps, triceps, subscapular, chest, abdominal, hip, thigh, knee, calf), and circumferences (waist, hip, thigh) into account. Using DEXA as their reference method, researchers measured body fat in 117 German males and females between the ages of 26 and 66 with a BMI range of 19.0 to 39.4 kg/m². The equations for body fat mass (BFM_{New}) most predictive of body fat mass as determined by DEXA (BFM_{Dexa}) and cross-validated in a separate sample were:

$$\text{BFM}_{\text{New}} \text{ (kg) for men} = -40.750 + \{(0.397 \times \text{waist circumference}) + [6.568 \times (\log \text{triceps SF} + \log \text{subscapular SF} + \log \text{abdominal SF})]\}$$

$$\text{BFM}_{\text{New}} \text{ (kg) for women} = -75.231 + \{(0.512 \times \text{hip circumference}) + [8.889 \times (\log \text{chin SF} + \log \text{triceps SF} + \log \text{subscapular SF})] + (1.905 \times \text{knee breadth})\}$$

BFM_{New} correlated highly with BFM_{Dexa} in both men ($r = 0.938$, $p < 0.001$) and women ($r = 0.949$, $p < 0.001$). Mean differences between percent body fat by DEXA and percent body fat by equation were lowest for Garcia's equation ($0.1 \pm 3.1\%$ in men and $0.1 \pm 4.4\%$ in women), followed by Peterson's ($2.3 \pm 4.1\%$ in men and $-2.4 \pm 3.8\%$ in women), then Durnin and Womersley's ($6.7 \pm 4.5\%$ in men and $-9.8 \pm 4.4\%$ in women). Results suggested that Garcia's equation was the most accurate predictor of percent body fat among equations tested and that Peterson's equation was a more accurate predictor than Durnin and Womersley's equation.

Methodological Concerns of Prior Obesity/Headache Studies

Existing studies on the obesity-headache relationship share two common methodological problems: measurement of adiposity and assessment of headache. Regarding adiposity measurement, all but one published study have relied solely on BMI as a measure of adiposity, and participants often self-reported their height and weight to derive BMI, which is less reliable method than directly measuring height and weight (Gorber, Tremblay, Moher, & Gorber, 2007).

Additionally, even when height and weight are directly measured, BMI has limited utility in measuring adiposity as compared to other anthropometric methods, as discussed above.

Therefore, relying solely on BMI may not be accurately estimating potential associations between obesity and headache.

A second methodological concern pertinent to existing studies is lack of adherence to ICHD criteria to determine headache diagnoses. While some researchers used ICHD criteria in their studies, many relied on other methods such as simply asking participants if they “had severe headaches or migraines” or if a doctor had diagnosed them with migraine at some point (Brown et al, 2000; Scher et al., 2003; Ford et al., 2008; Peterlin et al., 2010; Robberstad et al., 2010; Vo et al., 2011). Given Bigal et al.’s (2006) and Schramm et al.’s (2013) findings that headache-adiposity associations are more salient among those with particular headache subforms (i.e., CM than CTTH), and that the diagnostic criteria for some primary headache disorders have recently changed (ICHD-3; International Headache Society, 2013), differentiating headache diagnoses using validated criteria is of particular importance.

Goals of Present Study

In light of the limitations of prior studies, the present study sought to use established anthropometric measures of adiposity and strict adherence to ICHD-3 criteria to examine associations between adiposity and episodic primary headache disorders among young adults. A secondary goal was to compare the anthropometric methods in terms of their differential utility in predicting headache variables (i.e., frequency, severity, and disability).

Hypotheses

Study goal 1: To assess whether body fat (as calculated by BMI, waist circumference, waist-to-hip ratio, body adiposity index (BAI), and Peterson and Garcia's skinfold equations) varies as a function of primary headache.

Hypothesis 1: Participants with EM would have the highest average body fat, followed by those with ETTH, followed by non-headache controls, who would have the lowest average body fat.

Study goal 2: To assess differences in strength of association between adiposity (as calculated by BMI, waist circumference, waist-to-hip ratio, body adiposity index (BAI), and Peterson and Garcia's skinfold equations) and headache-related variables (frequency, severity, and disability) among both headache conditions (ETTH and EM).

Hypothesis 2a: Headache frequency, severity, and disability would be positively associated with all measures of adiposity.

Hypothesis 2b: Compared to BMI, other anthropometric measures (waist circumference, waist-to-hip ratio, BAI, and Peterson and Garcia's skinfold equations) would yield stronger associations between adiposity and headache-related variables.

Hypothesis 2c: Associations between adiposity and headache-related variables would be more salient among participants with EM than participants with ETTH.

Ancillary goal: To determine if associations between adiposity and headache prevalence, severity, frequency, and disability still exist after controlling for scores on anxiety, depression, and stress measures.

Hypothesis 3: After controlling for scores on anxiety, depression, and stress measures, positive associations between adiposity and headache prevalence, severity, frequency, and disability would remain for participants with EM only.

II. METHODS

Participants

The initial sample consisted of 133 undergraduate students age 18 years and older enrolled in psychology courses at the University of Mississippi who received modest course credit for participation. Students meeting ICHD-3 criteria for ETTH or EM and those not meeting criteria for any primary headache disorder were identified following their completion of a series of online questionnaires via Qualtrics, and they were invited by email to participate in the laboratory session. Assuming a small to moderate effect size ($f^2=.085$), a power level of 0.80, and an alpha level of 0.05, a total sample size of 108 participants was required to determine whether body fat varies as a function of primary headache.

Measures

Demographic Questionnaire. The Demographic Questionnaire included questions regarding basic demographic information such as age, gender, and race.

Structured Diagnostic Interview for Headache – 3 (Brief Version). The Structured Diagnostic Interview for Headache (SDIH-3; Smitherman, Penzien, Bartley, Rhudy, & Rains, 2014) is a modified version of the original computer-administered and well-validated SDIH (Andrew, Penzien, Rains, Knowlton, & McAnulty, 1992), revised to comport with ICHD-3 diagnostic criteria. The SDIH-3 is a 17-item instrument that assesses for primary headache disorders by querying headache symptoms, frequency, severity, and other diagnostic characteristics. Additionally, the SDIH-3 includes appendix questions for assessing aura symptoms, cluster headache, medication overuse, and post-traumatic headache.

Headache Impact Test-6 (HIT-6). The HIT-6 (Kosinski et al., 2003) is a 6-item self-report measure that assesses disability resulting from headache. Specifically, the HIT-6 assesses for headache impact on psychological, cognitive, occupational, and social functioning. The 6 items provide response options on a 5-point Likert-type scale from “Never” to “Always” to assess for frequency and severity of impairment. Scores range from 36 to 78, with 36 indicating little impact and 78 indicating very severe impact of headache on functioning. Kosinski et al. (2003) found the HIT-6 to have internal consistency, alternate forms, and test–retest reliability estimates of 0.89, 0.90, and 0.80, respectively. The measure also has high discriminate validity across headache diagnostic groups (Kosinski et al., 2003).

Perceived Stress Scale (PSS-10). The PSS-10 (Cohen, Kamarck, & Mermelstein, 1983; Cohen & Williamson, 1988) is a 10-item self-report instrument that assesses perceived stress in the past month. Items were designed to assess how unpredictable, uncontrollable, and overloaded respondents find their lives. The measure is widely used and has demonstrated strong reliability among college samples (i.e., Cronbach’s alpha of .89; Roberti, Harrington, & Storch, 2006).

Generalized Anxiety Disorder-7. The Generalized Anxiety Disorder-7 (GAD-7; Spitzer, Kroenke, Williams, & Lowe, 2006) is a seven-item self-report measure of anxiety symptoms consistent with the Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev.; *DSM-IV-TR*; American Psychiatric Association, 2000) diagnostic criteria for generalized anxiety disorder. The respondent answers each item on a Likert-type scale of 0 to 3, with 0 indicating “not at all” and 3 “nearly every day.” Total scores range from 0-21; scores of 5-9 are indicative of mild anxiety, 10-14 of moderate anxiety, and 15 or greater of severe anxiety. Spitzer and colleagues (2006) demonstrated the GAD-7 to have good internal consistency ($\alpha =$

.92) and test-retest reliability ($r = .82$). The GAD-7 also effectively screens other anxiety disorders (Kroenke, Spitzer, Williams, Monahan, & Lowe, 2007).

Patient Health Questionnaire-9. The Patient Health Questionnaire-9 (PHQ-9; Kroenke, Spitzer, & Williams, 2001) is a nine-item self-report measure of depressive symptoms consistent with DSM-IV-TR (2000) diagnostic criteria for major depressive disorder. The respondent answers each item on a Likert-type scale of 0 to 3, with 0 indicating “not at all” and 3 “nearly every day.” Total scores range from 0-27; scores of 5-9 are indicative of mild depression, 10-14 of moderate depression, 15-19 of moderately severe depression, and 20 or greater of severe depression. The PHQ-9 has good internal consistency ($\alpha = .89$) and test-retest reliability ($r = .84$) (Kroenke et al., 2001).

Anthropometric Measures of Obesity.

Body Mass Index (BMI). The BMI is a measure of body mass calculated by body weight in kilograms divided by height in meters squared.

Waist Circumference. Waist circumference is measured midway between lower rib margin and superior anterior iliac spine (Garcia et al., 2005; World Health Organization, 2011).

Waist-to-Hip Ratio. Waist-to-hip ratio is calculated by waist circumference divided by hip circumference. Hip circumference is the maximal circumference at the level of the trochanters (Garcia et al., 2005; World Health Organization, 2011).

Body Adiposity Index (BAI). The BAI is a measure of adiposity calculated by the following equation: $BAI = ((\text{hip circumference in cm}) / ((\text{height in m})^{1.5} - 18))$.

Skinfold Equations of Peterson et al. (2003). Peterson et al.’s (2003) skinfold equations are measures of percent body fat.

$$\begin{aligned} \%BF_{\text{new}} \text{ for men} &= 20.94878 + (\text{age} \times 0.1166) \\ &- (\text{height} \times 0.11666) + (\text{sum4} \times 0.42696) \\ &- (\text{sum4}^2 \times 0.00159) \end{aligned}$$

$$\begin{aligned} \%BF_{\text{new}} \text{ for women} &= 22.18945 + (\text{age} \times 0.06368) \\ &+ (\text{BMI} \times 0.60404) - (\text{height} \times 0.14520) \\ &+ (\text{sum4} \times 0.30919) - (\text{sum4}^2 \times 0.00099562) \end{aligned}$$

Height is quantified in cm and “sum4” is the sum of the triceps, suprailiac, subscapular, and midthigh skinfold thicknesses. The triceps skinfold site is the midpoint of the back of the upper arm between the lateral projection of the acromion process of the scapula and the inferior border of the olecranon process of the ulna, and the measurement is taken parallel to the longitudinal axis of the upper arm (Lohman, Roche, & Martorell, 1988; Peterson et al., 2003). The suprailiac skinfold site is an oblique fold, 45° to the horizontal, along the midaxillary line immediately above the iliac crest. The subscapular skinfold site is below the inferior angle of the scapula, at 45° to the vertical, along the natural cleavage lines of the skin. The midthigh skinfold site is the front of the thigh, halfway between the inguinal crease and the proximal border of the patella, and the measurement is taken along the long axis of the femur while the participant is standing and shifting his/her weight to the other leg (Lohman et al., 1988; Peterson et al., 2003).

Skinfold Equations of Garcia et al. (2005). Garcia et al.’s (2005) skinfold equations are measures of body fat mass (BFM).

$$\begin{aligned} \text{BFM}_{\text{New}} \text{ (kg) for men} &= -40.750 + \{(0.397 \times \text{waist circumference}) + \\ &[6.568 \times (\log \text{triceps SF} + \log \text{subscapular SF} + \log \text{abdominal SF})]\} \end{aligned}$$

$$\begin{aligned} \text{BFM}_{\text{New}} \text{ (kg) for women} &= -75.231 + \{(0.512 \times \text{hip circumference}) + [8.889 \\ &\times (\log \text{chin SF} + \log \text{triceps SF} + \log \text{subscapular SF})] + (1.905 \times \text{knee breadth})\} \end{aligned}$$

Height and circumferences are quantified in centimeters, and skinfold thicknesses and knee breadth are quantified in millimeters. Triceps and subscapular sites are identical to those in Peterson et al.’s (2003) method. The abdominal skinfold site is a horizontal fold, 5 cm lateral to

and at the level of the midpoint of the umbilicus (Garcia et al., 2005). The chin skinfold site is a submental fold concentric to the chin, parallel to the longitudinal axis of the body. Knee breadth is the distance between the medial and lateral epicondyles of the femur, and the measurement is taken with sliding calipers while the participant is seated with the leg forming a right angle (Garcia et al., 2005).

Procedures

The study commenced once adequate inter-rater reliability ($\geq 90\%$ agreement) was obtained for each anthropometric measurement. Pre-screened participants presented individually for the laboratory session, at which time their headache diagnosis was confirmed with in-person administration of the SDIH-3. Subsequently, participants completed the aforementioned surveys, after which the anthropometric measurements were taken: height, weight, waist circumference, hip circumference, knee breadth, and skinfold thicknesses (triceps, subscapular, suprailiac, abdominal, midthigh, and chin). Surveys were administered before anthropometric measures to ensure that any stress or discomfort from anthropometric measures did not influence survey responses. Each participant was measured in an otherwise unoccupied room to ensure privacy.

Statistical Analyses

Differences in adiposity (as measured by BMI, waist circumference, waist-to-hip ratio, BAI, and skinfolds) between headache groups (ETTH, EM, and control) were assessed using a MANOVA. Post-hoc univariate ANOVAs were used to assess potential specific differences. To test if adiposity was associated with increased headache frequency, severity, and disability, multiple linear regressions (among migraine and TTH conditions separately) were run with adiposity measures as “predictor” variables and frequency, severity, and disability as criterion variables. Significance was assessed before and after Bonferroni corrections for multiple

comparisons. Results of the MANOVA and regressions were predicted to indicate which adiposity measure would be most strongly associated with headache variables and thus of most potential use clinically. Regressions were repeated in hierarchical form after first entering sex, anxiety, depression, and stress scores in a block prior to the headache variables, in light of data that sex and negative affectivity influence adiposity-headache associations (Peterlin, Rosso, Rapoport, & Scher, 2010; Peterlin et al., 2013; Tietjen et al., 2007).

III. RESULTS

Participant Demographics and Primary Headache Diagnosis Prevalence

One hundred thirty-three students ages 18-54 participated in the study. Of these, 12 provided responses to the SDIH-3 indicative of a headache diagnosis other than migraine or tension-type headache ($n = 5$) or that precluded establishing a clear diagnosis ($n = 7$). Four participants with migraine without aura, 3 participants with migraine with aura, and 3 participants with tension-type headache reported headache frequencies of less than one day with headache per month. One individual was a univariate outlier on age (i.e., over 23; age = 54, ETTH participant). These participants were excluded from analyses to ensure distinct headache subgroups. The remaining 110 participants were checked for multivariate outliers by group using Mahalanobis distance, and 1 outlier (0.9%; migraine without aura participant) was found using a conservative $p < .001$ chi-square cut-off (Tabachnick & Fidell, 2007). After deleting this participant, the final sample included 109 participants: 26 (23.9% of retained sample) without headache, 3 (2.8%) with chronic migraine, 30 (27.5%) with episodic migraine without aura, 10 (9.2%) with episodic migraine with aura, 3 (2.8%) with chronic tension-type headache, and 37 (33.9%) with episodic tension-type headache.

Demographics of the retained sample are presented in Table 1. Nearly three-quarters (72.5%) of participants were female, and the mean age was 18.95 years ($SD = 1.08$). The majority of the sample was Caucasian (67.9%), followed by 19.3% African American, 5.5% Asian, 4.6% multiracial or other, and 2.8% Hispanic/Latino. Based on BMI ranges established by the Centers for Disease Control, 1.8% of participants were underweight, 62.4% were of a healthy

weight, 23.9% were overweight but not obese, and 11.9% were obese. Using waist circumference as an indicator of obesity and cutoffs established by the World Health Organization, 8.3% of the sample was obese. Participants with a primary headache disorder reported, on average, experiencing 5.0 headache days per month ($SD = 5.1$), a pain severity of 5.7 out of 10 ($SD = 1.7$), mean disability on the HIT-6 of 56.9 ($SD = 7.5$), GAD-7 score of 6.08 ($SD = 4.0$), PHQ-9 score of 6.3 ($SD = 4.5$), and PSS-10 score of 17.3 ($SD = 6.2$). More than three quarters (79.5%) of those with headache reported moderate or higher disability on the HIT-6.

Differences in Adiposity

Participants with a primary headache disorder had an average waist circumference of 76.3 cm ($SD = 13.0$), BMI of 24.5 ($SD = 5.7$), waist to hip ratio of .78 ($SD = .07$), BAI of 27.5 ($SD = 5.6$), percent body fat via Peterson's equations of 32.8% ($SD = 7.3$), and body fat mass (kg) via Garcia's equations of 21.2 ($SD = 9.6$). Contrary to hypotheses, a MANOVA did not reveal significant differences between the three headache groups (control, migraine, TTH) for adiposity. The Wilks' Lambda multivariate criterion for overall group differences was not significant; $F(12, 202) = .979, p = .47$. Exploratory post-hoc analyses revealed no significant effect for any single adiposity variable, including waist circumference ($p = .54$), BMI ($p = .98$), waist to hip ratio ($p = .21$), BAI ($p = .49$), Peterson's percent fat equations ($p = .35$), and Garcia's body fat mass equations ($p = .90$). A subsequent MANCOVA assessed whether differences in adiposity between groups would exist when controlling for sex, depression, anxiety, and perceived stress. As with the MANOVA results, the MANCOVA after controlling for these covariates was also not significant, Wilks' Lambda $F(12, 194) = .914, p = .70$.

Adiposity as a Predictor of Headache Disability, Frequency, and Pain Severity

A series of multivariate linear regressions assessed whether any of the six adiposity measures predicted HIT-6 scores, headache frequency, or self-reported headache pain severity. Separate regressions were run for participants with TTH and migraine. Results were not significant for migraineurs, although R-squared values indicated that adiposity accounted for 11% ($p = .62$), 13% ($p = .51$), and 10% ($p = .67$) of the variance in headache severity, frequency, and disability, respectively. Among participants with TTH, adiposity was not a significant predictor of headache severity (R-squared = 8%; $p = .82$) or frequency (R-squared = 21%; $p = .23$), but adiposity was a significant predictor of headache disability (R-squared = 39%; $p = .009$). After controlling for sex, anxiety, depression, and stress, results remained the same with all adiposity measures insignificant predictors of headache variables, with the exception of disability among those with TTH (R-squared = 48%; $p = .020$). To control for multiple comparisons using Bonferroni correction, a p-value of less than .008 was required for significance ($.05 \div 6$ comparisons). Thus, after employing Bonferroni correction, adiposity was no longer a significant predictor of headache disability among participants with TTH. Regression results are presented in Tables 2-4.

IV. DISCUSSION

Existing literature has established a positive association between BMI and risk for migraine, particularly chronic migraine, among adults under age 45 years (Bigal and Lipton, 2006; Ford et al., 2008; Peterlin et al., 2010; Vo et al., 2011). Adiposity's association with prevalence of TTH and episodic migraine is less clear, with previous studies yielding contradicting results (Bigal et al. 2006; Peterlin et al., 2013). However, previous studies have not consistently adhered to ICHD diagnostic criteria to establish primary headache diagnoses nor used measures of adiposity other than BMI. The present study sought to use established anthropometric measures of adiposity and a diagnostic headache interview to determine associations between adiposity and primary headache disorders of migraine and TTH. Based on findings of previous studies, we hypothesized that adiposity would differ between control (non-headache), migraine, and TTH groups.

Adiposity across Diagnostic Groups

Contrary to hypotheses, no differences in adiposity were found between those without headache, those with migraine, and those with TTH. Based on a priori power analysis, the present sample size was sufficient to detect statistically significant differences of small to moderate size between groups. Anthropometric measures of adiposity employed in this study have empirical support, and headache conditions were determined via strict adherence to ICHD criteria. Given a sufficient sample size and sound methods, results suggest a lack of association between adiposity and primary headache disorders. Existing literature has provided support both for and against a relationship between adiposity and episodic migraine, and the present findings

are consistent with those of Bigal et al. (2006), who found that the prevalence of EM did not significantly differ as a function of BMI. Consistent findings may be due to similarities in the sample composition. Specifically, 3% of participants in Bigal et al.'s (2006) study were underweight, 51% were of healthy weight, 31% were overweight, and 15% were obese. This BMI distribution is very similar to that of the present study, while Peterlin et al.'s (2013) sample consisted of 3% underweight, 37% healthy weight, 34% overweight, and 26% obese. Possibly Peterlin et al. (2013) found EM prevalence and BMI to be positively associated due to their sample's higher proportion of overweight and obese participants. Additionally, Peterlin et al.'s (2013) sample had an average participant age of 46.6 years, while Bigal et al.'s (2006) average participant age was 38.7 years – a mean closer to that of the present study.

The finding that adiposity is not associated with EM is in contrast to the consistent positive associations observed between obesity and chronic headache subforms (Bigal and Lipton, 2006; Scher et al., 2003; Schramm et al., 2013), suggesting that headache frequency is more strongly associated with obesity than is migraine per se. Only 7% of migraineurs in the present study had chronic migraine, and only 7.5% of TTH sufferers had CTTH. Thus, although the present study did not find any associations between adiposity and primary headache disorder prevalence, these conclusions are applicable to young adults with episodic migraine or TTH, as associations with chronic headache conditions cannot be established.

Adiposity as a Predictor of Headache Variables

Based on findings of previous studies that headache frequency, severity, and disability may increase as a function of BMI (Bigal et al., 2006; Bigal and Lipton, 2006; Tietjen, et al., 2007), the adiposity measures in this study were expected to be positively associated with headache frequency, severity, and disability. However, contrary to hypotheses, only small and

largely non-significant associations were found between the adiposity measures and headache variables among migraine and TTH groups. After controlling for sex, depression, anxiety, and perceived stress levels, results remained similar. These contradicting findings are likely due to the fact that the present sample consisted of primarily episodic headache subforms, whereas studies that found associations between adiposity and headache variables had higher proportions of chronic headache patients. However, Bigal et al., (2006) found that headache severity and disability were positively associated with BMI among episodic migraineurs, although they relied on self-reported BMI as the only measure of adiposity. Because the literature on adiposity measurement indicates the anthropometric methods in the current study are more accurate than BMI, a positive association between obesity and headache variables may be a spurious result that is not present when accurate measures of adiposity are employed.

Because the adiposity measures used in this study are highly correlated with “gold standard” methods, BAI, waist circumference, waist-to-hip ratio, and skinfold equations by Peterson and Garcia were expected to account for more variance in headache variables than BMI alone. Findings did not support this hypothesis; no consistent pattern was revealed when comparing the standardized betas for each adiposity measure, and none were statistically significant.

Adiposity in Episodic vs. Chronic Headache

As the present study found no relationships between adiposity and episodic migraine and TTH, multiple possible interpretations exist. One interpretation is that these findings, in conjunction with prior studies on adiposity and chronic headache, suggest that obesity characterizes chronic but not episodic primary headache disorders (Bigal and Lipton, 2006; Schramm et al., 2013). However, if adiposity is a risk factor for headache chronification, then

headache frequency would be expected to have a stronger relationship with adiposity than was observed in the present study. Previous studies have found a positive association between BMI and migraine frequency (Bigal and Lipton, 2006; Bigal et al., 2006; Tietjen et al., 2007), although the association was no longer significant when controlling for anxiety and depression, conditions common among individuals who are obese or who have migraine (Tietjen et al., 2007). Given the existing literature and current findings, a second interpretation is that the temporal direction of any relationship between adiposity and headache may be opposite than hypothesized; that is, rather than obesity being a risk factor for headache chronicity, people with chronic headache may subsequently gain weight and/or become obese (e.g., as a result of medication side effects, reductions in physical activity, and hypothalamic alterations). This hypothesis is consistent with the findings of Vo et al. (2011), who found women with migraine as children had 1.67-fold increased odds of gaining at least 22 pounds during adulthood. In contrast, Scher et al. (2003) found obese participants with episodic headache at baseline to be at increased risk of progressing to chronic daily headache at 11-month follow-up. Clearly, more longitudinal research is needed to determine the direction of the adiposity-headache relationship.

Another goal of this study was to determine which measure of adiposity had the strongest association with headache variables and thus would have the most clinical utility. Given null findings and insignificant associations of all adiposity measures with headache variables, the present study does not suggest any of the included adiposity measures to have much clinical utility in predicting headache variables for young adults with episodic migraine or episodic tension-type headache. However, this study does not speak to their utility in predicting headache variables for chronic headache patients, who comprised a small majority of the present sample, or treatment-seeking patients. Given findings in previous studies and the strong methodology of

the present study, it seems most likely that obesity-headache associations are unique to chronic migraine and chronic tension-type headache; thus future research with chronic headache patients may find clinical utility in the adiposity measures used in this study.

Limitations and Future Directions

Strengths of this study include the use of well-established anthropometric measures of adiposity other than BMI, examiner-measured adiposity instead of reliance on participant self-report, and strict adherence to ICHD diagnostic criteria via in-person structured interviews (SDIH-3). However, this study includes several limitations, and caution should be used when generalizing these findings to the population. First, the final sample consisted of 109 participants, which although sufficient assuming a small-to-moderate effect size, would be insufficient for a smaller effect size. Thus, it is possible that a larger sample would have yielded significant results and positive associations between adiposity and headache variables. However, p-values for the majority of statistical analyses were far short of and did not even “trend” toward significance. Even if larger samples yielded statistically significant differences, group differences in adiposity would likely be quite small and not of clinical significance. Second, all participants were non-treatment-seeking undergraduate students between the ages of 18 and 23, and thus results may not generalize to older or clinical populations. However, young adults are a desirable population in headache research, given their high prevalence of primary headache disorders and low frequency of variables that complicate conclusions from headache studies, such as long histories of medication overuse and chronification (Smitherman et al., 2011). Additionally, participants with extremely low headache frequencies (<1 day per month) were omitted from analyses, and retained EM and ETTH participants had an average of approximately four headache days per month. Nearly 80% of participants with headache reported moderate to severe headache-related

disability, suggesting the majority of this young non-clinical sample had headache that significantly affected their lives. Third, although this study included a variety of adiposity measures with empirical support, for financial and practical reasons it did not include a “gold standard” method of assessing body composition such as DEXA or MRI and CT. Thus, it is possible that different associations would have been found between adiposity and headache variables had a more accurate measure of adiposity been employed. However, given that previous studies found the anthropometric methods used in this study to have high correlations with “gold standard” methods and the four-component model, and given this study’s sufficient sample size based on power analysis, the anthropometric methods used likely would have detected headache-adiposity associations if present (Bergman et al., 2012; Clasey et al., 1999; Garcia et al., 2005; Johson et al., 2012; Kamel et al., 1999; Peterson et al., 2003). Finally, the proportion of participants who were overweight or obese (35.8%) was lower than that of the national average (68.5%), and thus findings may not be generalizable to the larger population.

Given the established relationship between BMI and migraine in the existing literature, future studies are needed that assess the utility of these anthropometric measures of adiposity among individuals with chronic migraine and TTH, as well as those using DEXA or MRI and CT to further examine the relationships between adiposity and primary headache disorders. Studies including older adults or treatment-seeking samples would yield results applicable to a wider range of people and those with larger samples would also be able to detect small effects of adiposity on headache variables. Another area of needed research are longitudinal studies examining adiposity as a risk factor for headache chronification. Such findings would have preventative and treatment implications for clients presenting with headache, such as determining the value of integrating weight monitoring or weight loss interventions within

existing headache treatments (see Bond, Roth, Nash, & Wing, 2011). Currently, Bond et al. (2013) are conducting a RCT examining the effects of a behavioral weight loss program on migraine frequency among women, and other studies of this type will further clarify the role of obesity weight loss in migraine.

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APPENDIX

Table 1

Demographics and Average Adiposity

		No Headache N = 26	Chronic migraine N = 3	Episodic migraine N = 30	Episodic migraine w/ aura N = 10	Chronic TTH N = 3	Episodic TTH N = 37
Demographics							
Age	M (SD)	19.0 (1.2)	18.7 (0.6)	19.0 (1.0)	18.5 (0.5)	18.7 (0.6)	19.1 (1.2)
Female	N %	15 (58%)	2 (67%)	26 (87%)	10 (100%)	3 (100%)	23 (62%)
Male	N %	11 (42%)	1 (33%)	4 (13%)	0 (0%)	0 (0%)	14 (38%)
Caucasian	N %	17 (65%)	2 (67%)	14 (47%)	8 (80%)	3 (100%)	30 (81%)
Headache Days/Month	M (SD)	N/A	20.7 (8.1)	4.2 (3.4)	3.8 (3.0)	17.3 (2.3)	3.8 (2.9)
HIT-6 Score	M (SD)	N/A	65.0 (8.1)	60.5 (6.7)	60.8 (6.1)	61.3 (1.5)	52.0 (6.0)
Pain Severity	M (SD)	N/A	5.3 (1.5)	6.6 (1.4)	7.5 (1.6)	5.5 (1.3)	4.6 (1.2)
PHQ-9 Score	M (SD)	3.8 (3.3)	10.3 (8.4)	7.1 (4.8)	6.5 (6.9)	7.3 (2.5)	5.2 (2.7)
GAD-7 Score	M (SD)	3.5 (3.6)	10.3 (6.8)	6.6 (4.0)	4.8 (4.4)	4.7 (3.1)	5.8 (3.7)
PSS-10 Score	M (SD)	12.4 (4.8)	21.3 (6.0)	18.9 (6.8)	14.7 (6.9)	21.0 (5.3)	16.1 (5.1)
Adiposity Measures							
Waist Circumference	M (SD)	77.8 (9.6)	66.9 (5.6)	77.6 (16.0)	69.7 (7.4)	66.7 (6.5)	78.5 (11.4)
BMI	M (SD)	24.8 (4.6)	21.1 (2.7)	25.5 (7.6)	22.6 (3.8)	21.6 (2.5)	24.8 (4.5)
Waist to Hip Ratio	M (SD)	.81 (.06)	.75 (.06)	.79 (.08)	.75 (.04)	.72 (.05)	.80 (.06)
BAI	M (SD)	26.2 (4.4)	24.0 (2.5)	28.2 (7.1)	27.5 (3.9)	28.8 (2.8)	27.0 (4.9)
Peterson Percent Body Fat	M (SD)	31.1 (7.1)	26.8 (6.3)	34.4 (8.7)	32.9 (5.4)	33.2 (4.5)	31.9 (6.6)
Garcia Body Fat Mass (kg)	M (SD)	20.3 (8.5)	12.9 (4.4)	22.6 (11.3)	19.1 (8.6)	18.8 (6.2)	21.6 (8.6)

Note. TTH = tension-type headache, M = mean, SD = standard deviation, N = number of participants, % = percentage of participants. Waist circumference is in centimeters; Garcia body fat mass is in kilograms.

Table 2

Adiposity as a Predictor of Headache Severity

Migraine (n = 43)										
	Coefficients					Model Summary				
	<i>B</i>	<i>SE</i>	<i>Beta</i>	<i>t</i>	<i>P-value</i>	<i>R²</i>	<i>Adjusted R²</i>	<i>SE</i>	<i>F</i>	<i>P-value</i>
Waist Circumference	.10	.13	1.00	.81	.42					
BMI	-.14	.16	-.62	-.88	.38					
Waist to Hip Ratio	-8.45	12.10	-.43	-.70	.49					
BAI	-.07	.13	-.30	-.55	.59					
Peterson equations	.15	.11	.81	1.40	.17					
Garcia equations	-.10	.11	-.69	-.89	.38					
						.11	-.04	1.51	.74	.62
Tension-Type Headache (n = 40)										
	Coefficients					Model Summary				
	<i>B</i>	<i>SE</i>	<i>Beta</i>	<i>t</i>	<i>P-value</i>	<i>R²</i>	<i>Adjusted R²</i>	<i>SE</i>	<i>F</i>	<i>P-value</i>
Waist Circumference	.03	.08	.27	.37	.72					
BMI	-.19	.13	-.69	-1.42	.17					
Waist to Hip Ratio	1.35	7.12	.07	.19	.85					
BAI	.04	.10	.15	.35	.73					
Peterson equations	-.02	.08	-.08	-.21	.84					
Garcia equations	.04	.09	.28	.45	.66					
						.08	-.09	1.25	.48	.82

Note: n = number of participants, SE = standard error, BMI = body mass index, BAI = body adiposity index.

Table 3

Adiposity as a Predictor of Headache Frequency

Migraine (n = 43)										
	Coefficients					Model Summary				
	<i>B</i>	<i>SE</i>	<i>Beta</i>	<i>t</i>	<i>P-value</i>	<i>R²</i>	<i>Adjusted R²</i>	<i>SE</i>	<i>F</i>	<i>P-value</i>
Waist Circumference	-.47	.48	-1.20	-.99	.33					
BMI	-.26	.58	-.31	-.45	.66					
Waist to Hip Ratio	63.60	45.18	.86	1.41	.17					
BAI	.60	.48	.68	1.27	.21					
Peterson equations	-.20	.40	-.29	-.51	.61					
Garcia equations	.16	.40	.30	.40	.70					
						.13	-.015	5.65	.90	.51
Tension-Type Headache (n = 40)										
	Coefficients					Model Summary				
	<i>B</i>	<i>SE</i>	<i>Beta</i>	<i>t</i>	<i>P-value</i>	<i>R²</i>	<i>Adjusted R²</i>	<i>SE</i>	<i>F</i>	<i>P-value</i>
Waist Circumference	-.23	.27	-.57	-.85	.40					
BMI	-.25	.47	-.24	-.53	.60					
Waist to Hip Ratio	1.83	25.15	.03	.07	.94					
BAI	.43	.36	.45	1.19	.24					
Peterson equations	-.35	.26	-.48	-1.31	.20					
Garcia equations	.25	.31	.46	.80	.43					
						.21	.06	4.43	1.45	.23

Note. n = number of participants, SE = standard error, BMI = body mass index, BAI = body adiposity index.

Table 4

Adiposity as a Predictor of Headache Disability

Migraine (n = 43)										
	Coefficients					Model Summary				
	<i>B</i>	<i>SE</i>	<i>Beta</i>	<i>t</i>	<i>P-value</i>	<i>R²</i>	<i>Adjusted R²</i>	<i>SE</i>	<i>F</i>	<i>P-value</i>
Waist Circumference	.18	.56	.39	.32	.75					
BMI	-.76	.68	-.79	-1.12	.27					
Waist to Hip Ratio	27.32	52.78	.32	.52	.61					
BAI	.28	.56	.27	.50	.62					
Peterson equations	-.16	.47	-.19	-.34	.74					
Garcia equations	.10	.47	.16	.21	.84					
						.10	-.05	6.60	.68	.67
Tension-Type Headache (n = 40)										
	Coefficients					Model Summary				
	<i>B</i>	<i>SE</i>	<i>Beta</i>	<i>t</i>	<i>P-value</i>	<i>R²</i>	<i>Adjusted R²</i>	<i>SE</i>	<i>F</i>	<i>P-value</i>
Waist Circumference	-.18	.33	-.33	-.56	.58					
BMI	-.76	.57	-.53	-1.34	.19					
Waist to Hip Ratio	.55	30.43	.01	.02	.99					
BAI	.61	.44	.46	1.39	.18					
Peterson equations	-.06	.32	-.06	-.18	.86					
Garcia equations	.27	.38	.36	.72	.48					
						.39	.28	5.36	3.51	.009*

Note. n = number of participants, SE = standard error, BMI = body mass index, BAI = body adiposity index. * $p < .01$.

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2014-present	<u>Psychological Assessment Clinic</u> , University of Mississippi Supervisor: Scott Gustafson, Ph.D. Position: Psychological Examiner Duties: Conduct comprehensive psychological evaluations to assess for psychological disorders. Administer and score tests, write integrated reports, and present feedback.
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- 2014-2015 Autism Center of North Mississippi, Tupelo, MS
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 Supervisor: Shannon L. Hill, Ph.D.
 Position: Education and Research Intern
 Duties: Provided individual therapy to adults with intellectual disabilities, conducted assessments (intellectual, adaptive behavior, medication side-effects, dementia, functional behavior), developed and implemented behavior plans, led a grief group, and taught staff trainings on resident empowerment.
- 2010-2011 Crisis Clinic of Thurston and Mason Counties, Olympia, WA
 Supervisor: Jill Joanis, LMHC
 Position: Crisis Intervention Specialist
 Duties: Provided crisis intervention and/or community resource referrals to a wide range of callers. Trained new volunteers in crisis intervention skills and clinic procedures.
- 2010-2011 Safe Place Domestic Violence Shelter, Olympia, WA
 Supervisor: Casi Totten
 Position: Support Group Facilitator
 Duties: Facilitated weekly in-house support group for domestic violence survivors. Provided residents with community resources.

ADMINISTRATIVE AND RESEARCH EXPERIENCE

- 2015 Office of Student Disability Services, University of Mississippi
 Supervisor: Stacey Reycraft, Director
 Position: Disability Specialist
 Duties: Review and verify applications for student accommodations and disability services.
- 2013-2014 Psychological Services Center, University of Mississippi
 Supervisor: Scott Gustafson, Ph.D.
 Position: Clinic Assistant
 Duties: Assistant to clinic director. Managed Titanium software, record keeping, and client notes throughout department. Advertised clinic services to the community, designed clinic publication and outreach materials.

- 2011-2012 Trauma Lab, University of Mississippi
 Supervisor: Tom Lombardo, Ph.D.
 Position: Graduate Research Assistant
 Duties: Assisted with data collection on study of Pennebaker writing task and reduction of PTSD symptomology among undergraduates reporting trauma. Administered CAPS structured interview to undergraduate participants.
- 2010-2011 Saint Martin's University, Lacey, WA
 Supervisor: Katia Shkurkin, Ph.D.
 Position: Psychology Research Assistant
 Duties: Assisted with study on women survivors' self-descriptions of severity and consequences of childhood sexual abuse. Entered and analyzed data using PASW, coded and cleaned data, assisted with IRB application process, and designed and distributed online survey for study.
- 2007-2008 Reconnecting Youth Program, School of Nursing,
 University of Washington, Seattle, WA
 Supervisor: Deborah Thomas-Jones, Ph.D.
 Position: Research Assistant
 Duties: Assisted with studies on school-based models designed to prevent maladaptive behaviors and depression among high-risk youth. Interviewed middle school students and parents for multiple studies, recruited participants, entered data using SPSS and Achenbach software, managed database, designed visual aids, and completed office administration tasks.
- 2006-2008 Project MARS (Motivating Adolescents to Reduce Sexual risk),
 University of Washington, Seattle, WA
 Supervisor: Joshua A. Ginzler, Ph.D.
 Position: Research Assistant
 Duties: Entered data using SPSS, assisted with grant proposal preparation, conducted literature searches, wrote article summaries, and helped maintain and update EndNote database.
- 2005-2006 Department of Psychology, University of Washington, Seattle, WA
 Supervisor: Ursula Whiteside, Ph.D. candidate
 Position: Research Assistant
 Assisted with study on eating disorders among college undergraduates. Administered surveys to participants, designed questionnaire using DatStat, and completed organizational tasks.

PRESENTATIONS

Moynahan, V. L., Rogers, D. G., & Smitherman, T. A. (November 2015). *Comparing Anthropometric Methods to Quantify Relations Between Adiposity and Headache*. To be

presented at the 49th annual meeting of the Association for Behavioral and Cognitive Therapies, Chicago, IL.

Black, A. K., **Moynahan, V. L.**, Fulwiler, J. C., & Smitherman, T. A. (June 2015). *The Role of Fear of Pain in Headache*. Poster presented at the annual scientific meeting of the American Headache Society, Washington D.C.

Hamer, J., **Moynahan, V. L.**, & Smitherman, T. A. (April 2015). *Traumatic Event Exposure and Headache-Related Variables*. Oral presentation at the 2nd annual Conference on Psychological Science, University of Mississippi, Oxford, MS.

Foote, H. L., Crudup, B. M., **Moynahan, V. L.**, Landy, S. H., Roland, M., & Smitherman, T. A. (June 2014). *Psychological Flexibility in Migraine: Acceptance and Values-Based Action*. Poster presented at the annual scientific meeting of the American Headache Society, Los Angeles, CA.

Peck, K. R., **Moynahan, V. L.**, & Smitherman, T. A. (November 2013). *Relationships among Headache, Self-Efficacy, and Impairment*. Poster presented at the 47th annual meeting of the Association for Behavioral and Cognitive Therapies, Nashville, TN.

Moynahan, V. L., McDermott, M., Tull, M. T., Gratz, K. L., Houle, T. T., & Smitherman, T. A. (June 2013). *Comorbidity of Migraine and Affective Disorders among Substance Dependent Inpatients*. Poster presented at the 2013 International Headache Congress, Boston, MA.