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Eating Frequency and Obesity and Metabolic Disease Risk in Hispanic Youth

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Eating Frequency and Obesity and Metabolic Disease Risk in Hispanic Youth

by

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Dedication

To my wife who has supported me relentlessly and unquestionably throughout the graduate school process. To my mother and father who provided the fire and guidance to allow for this opportunity. To my brother who taught me that most of our struggles are trivial and to never give up, but also never forget to laugh when things get tough.

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Eating Frequency and Obesity and Metabolic Disease Risk in Hispanic Youth

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The objective of this study was to analyze the relationship between eating frequency and adiposity and associated metabolic disease risk. The main hypothesis was that increased eating frequency compared to infrequent eating frequency would be related to reduced adiposity and metabolic disease markers, as well as healthier dietary and physical activity behaviors in Hispanic youth.

For this investigation four cross-sectional analyses were performed in Hispanic youth age 8-19 y with a combined sample size of 1185. Three of the studies completed included gold standard measures of dietary intake, body composition, metabolic parameters, physical activity, and specific fat distribution. The forth included a questionnaire filled out by over 700 Hispanic college freshmen age 18-19 y at the University of Texas at Austin.

The first two analyses included two separate cohorts of overweight or obese minority youth ages 8-18 y (n=185 and n=191)[1,2]. Frequent Eaters (4.0 eating occasions (EOs) per day) compared to Infrequent Eaters (2.4 EOs per day) consumed more calories per day ($p \leq 0.01$), ate more often ($p \leq 0.01$), consumed less calories per EO ($p \leq 0.01$), yet

exhibited lower visceral adipose tissue (p=0.03), BMI ($p \leq 0.01$), waist circumferences ($p \leq 0.01$), fasting insulin (p=0.02), HOMA-IR values (p=0.02), and lower triglycerides ($p \leq 0.01$), and higher beta cell function (p=0.01) and acute insulin response (p=0.02).

To date, these were the first studies investigating eating frequency and adiposity and metabolic disease risk in Hispanic youth. We know minority youth tend to eat less often than Non-Hispanic Whites and that first year college students are particularly susceptible to weight gain and poor overall health. Given this combined with our previous findings and that in Hispanic high school graduates were more likely to be enrolled in college than NHW or blacks, we wanted to further explore this high risk population as they transitioned to college. We conducted a large cross-sectional study with 709 Hispanic college freshmen that completed a questionnaire to identify eating frequency, physical activity levels and reported height and weight. We found eating frequency to be positively related to moderate to vigorous physical activity (MVPA), but not overweight or obesity prevalence. Also, those who ate 4 or more times per day were 2.5 times more likely to spend at least 150 minutes in MVPA per week compared to those who ate 2 or less times per day.

We further brought in 100 of the 709 in for extensive in-person testing in which 92 subjects had complete data. We found frequent eaters ate 44% more often ($p \leq 0.01$) and consumed 27% less calories per eating occasion ($p \leq 0.01$), while consuming 21% more kcals per day (or 445 less kcals per day) ($p \leq 0.01$) compared to infrequent eaters. Frequent eaters had 8% lower BMIs (p=0.02), 60% lower BMI z-scores (p=0.03), 21% lower

visceral adipose tissue (p=0.03), 26% lower subcutaneous adipose tissue (p=0.03), and 8% higher total body fat (p=0.04) compared to infrequent eaters.

These findings suggest that increased eating frequency is related to decreased obesity and metabolic disease risk in Hispanic youth and Hispanic college freshmen, despite increases in energy intake. Additionally, in a very large and exclusively Hispanic college freshmen population eating frequency was related to self-reported increases in physical activity. Thus, increasing eating frequency may be a viable public health message among Hispanic youth and Hispanic college students, yet more research is needed to understand the potential mechanisms and investigate a causal relationship.

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Chapter 1: Introduction

Hispanics are the largest and fastest growing ethnic minority in the United States, representing 17% of the population[3]. Obesity and Type 2 Diabetes (T2D) also disproportionately affect Hispanics. National data collected in Hispanic adolescents (12-19 y) show that 42% are overweight and 23% are obese, compared to 30% and 16%, respectively, in Non-Hispanic Whites (NHW)[4]. Furthermore, over 30% of Hispanic children and adolescents (8-18 y) have pre-diabetes and the metabolic syndrome[5,6]. Thus, highlighting the need to understand what behaviors contribute to obesity and related disease risk, as well as the need to design and implement effective interventions to prevent and treat obesity and related metabolic diseases in this high-risk population

According to NHANES 2005-2008 American youth (2-19 y) consume an average of 5.1 eating occasions (EOs) per day and 80% of all Americans eat more than 4 times per day, which has increased by an average of 0.5 to 1 EO per day across the population over the last three decades[7,8]. Hispanic youth are likely to eat less often than NHW[9], and national data suggests that approximately 9% of Hispanic adults compared to 3% of NHW consume $\leq$2 EOs per day[10]. Yet, it remains unclear how eating frequency affects obesity and metabolic disease risk, especially within Hispanic youth. Previous research focused on eating frequency and its relationship to adiposity has yielded mixed results, especially during childhood and adolescence, both of which are crucial periods in obesity development and the shaping of lifelong habits[11,12]. Eating frequency research has more consistently found an inverse association between the number of EOs per day and

adiposity[13-15], as well as metabolic disease risk in both youth and adult populations[2,16]. Yet, eating frequency patterns among Hispanic youth remain largely unknown[17].

Furthermore, in 2012, for the first time in history, Hispanic high school graduates (69%) were more likely to be enrolled in college than Non-Hispanic Whites (NHW; 67%) and Blacks (63%)[18]. In 2013, Hispanic students represented 22.9% of freshman enrollment at the University of Texas at Austin (UT-Austin), having the largest increase among all minority groups[19]. College students are particularly susceptible to poor overall health and this is pivotal because the behavioral choices college students make likely affect their risk of chronic disease later in life [20]. The transition to college has been identified as a critical period contributing to the rise in obesity rates. Yet, very little research has been done on this expanding population and their eating frequency patterns are again largely unknown.

We hypothesize that increased eating frequency among Hispanic youth age 8-19 y will be inversely related to obesity status and metabolic disease risk, as well as positively related to healthy dietary and physical activity behaviors. These findings can potentially inform public health interventions during this critical time period in an extremely at risk population.

Chapter 2: Review of Literature

OBESITY AND METABOLIC DISEASE RISK IN HISPANIC YOUTH

According to the US Census bureau, by 2050, it is estimated that one third of the United States population will be of Hispanic descent. Hispanic youth have higher rates of obesity and insulin resistance than NHW and Hispanic youth (15-19 y) have the highest rates of pediatric type 2 diabetes[21-23]. The Hispanic Community Healthy Study/Study of Latinos, a large multi-center study including more than 16,000 Hispanics found that prevalence of diabetes in adults ranged from 10% to 18% depending on origin, but that the lifetime risk of diabetes was around 50% for both Hispanic men and women[24]. Furthermore, our group has shown that over 30% of Hispanic children and adolescents (8-18 y) have pre-diabetes and metabolic syndrome[5,6]. Childhood and adolescence are crucial periods in obesity development and the shaping of lifelong habits, thus it is pivotal to explore how different eating patterns may affect obesity and metabolic disease risk in Hispanic youth[11,12]

As mentioned above, Hispanic students are now more likely to attend college than NHW or African Americans, yet little is known about weight categories and metabolic disease risk among Hispanic college students. Yet, college students as a whole are particularly susceptible to poor overall health and this time period is pivotal because the behavioral choices college students make likely affect their risk of chronic disease later in

life [20]. More than 70% of colleges students gain weight in the first year of college[25] and the smallest weight gain found in prospective longitudinal trials in college freshmen was 1.6 lbs,[26], whereas the largest weight gain seen was close to 9 lbs[27]. A recent meta-analysis by Vella-Zarb et al.[28] of 24 studies using an exclusively freshmen college population, with a mean duration of 6.4 months and total sample size of 3,401, found that the average weight gain was 3.9 lbs and 22 out of 24 studies found a statistically significant weight gain during freshmen year. The potential impact of ethnicity on weight gain was not mentioned or examined, although the study did elucidate that females may be more at risk than males. To my knowledge, no study has examined the dietary habits or potential obesity and metabolic risk in Hispanic college students. Given that college freshmen are prone to weight gain and Hispanics are the fastest growing ethnic minority on the UT-Austin campus and [19,29] examining the relationship between dietary habits and obesity and related metabolic disorders in this population is warranted.

EATING FREQUENCY PATTERNS IN HISPANIC YOUTH

Multiple studies have found that African American youth eat less often and more inconsistently than NHWs, yet to our knowledge very little data is available on the eating frequency habits of Hispanic youth[9,14,15]. A recent prospective longitudinal study by Ritchie et al.[15], with 2,372 African American and NHW girls (9-19y) found that eating frequency among African American girls was 4.7 EOs per day compared to 5.2 in NHW. In this study, 54% of NHW ate 4 or more times per day compared to 40% of African American girls and both groups ate less often as they aged. Yet, in this study any food or

beverage consumed was counted as an EO and there were not designated time periods to denote different eating occasions. National data in 15,978 adults published by Kerver et al.[10] suggests that approximately 9% of Hispanic adults compared to 3% of NHW consume ≤2 EOs per day[10]. Popkin et al.[7] found that in a national sample of 28,404 youth (2-18 y) eating frequency increased over the last three decades and as of the 2006 National Health and Nutrition Examination Survey (NHANES) data the average American eating frequency was 5 EOs per day. Unfortunately, this analysis did not break down their sample into smaller age ranges or different ethnicity groups, they did however use a time-based determinant between eating occasions of 15 minutes. Our group has shown that the average eating frequency in overweight Hispanic youth is between 3.8 and 4.0 EOs per day, which is considerably less than the number of EOs found in the studies mentioned above. However, we defined an EO as ≥50 calories and ≥15 minutes from any previous EO, and this definition originally proposed by Gibney et al.[30] has been used often in the eating frequency research[1,2,13,30-38]. Recently the first eating frequency definition methodological study carried out by Leech et al.[39] in 5,242 adults investigating the efficacy of eight different EO definitions, found that the Gibney et al. definition predicted the highest proportion of variance in total energy intake among both sexes and across all age groups. Furthermore, inside of this definition Hispanic youth eat less often than all other samples. Yet, comparatively little is known about the specific eating patterns of Hispanic youth and it remains unclear how or if this decreased number of EOs per day affects obesity and metabolic disease risk within this population.

PHYSICAL ACTIVITY PATTERNS IN HISPANIC YOUTH

NHANES physical activity data found that 42.0% of children (6-11 y) attained the recommended public health recommendation of 30 or more minutes of moderate to vigorous physical activity (MVPA) at least five days per week, however this number plummeted to around 8.0% in adolescents (12-19 y)[40]. A study by Belcher et al.[41] in 3,106 subjects with accelerometry data found that minority youth were more active than NHW and that activity declined with age regardless of ethnicity. However, Hispanics age 12-15 y were the most inactive category among all ethnicities and age groups. Belcher et al. also found that subjects (6 to 11 y) recorded 88 minutes in MVPA, compared to older subjects (12-19 y) who recorded under 30 minutes of MVPA. Studies among exclusively Hispanic youth support this finding that physical activity falls with increasing age, also it was found that physical activity was reduced among those of lower social economic status[41-43]. Yet, to our knowledge physical activity patterns among Hispanic college students have yet to be investigated.

In a sub-sample of 48 children (9-11 y) with accelerometer data from a study by Zerva et al.[13] found that total physical activity was highest among those who ate most frequently. To our knowledge this is the only study to date that has examined the relationship of eating frequency with physical activity in a youth population. However, a study with 85 women (47-56 y) by Duval et al.[44] found that eating frequency was positively associated with physical activity via accelerometer and negatively associated with BMI, but that this association was no longer present when correcting for the increase in physical activity. Yet, the prospective longitudinal study by Ritchie et al.[15] with girls found an inverse relationship with eating frequency and the change in BMI and waist circumference across a ten year period even when controlling for physical activity, but physical activity

data in this study was collected via an annual survey and not by accelerometry. Another cross-sectional study by Moschonic et al.[45] with 2,043 children (9-13 y) found that higher eating frequency and MVPA resulted in lower metabolic disease risk. However, further research on the relationship between eating frequency and physical activity is needed, as well as further analysis investigating the possible mitigating effect of increased physical activity among more frequent eaters on metabolic and obesity risk in these populations, especially those at increased risk like Hispanic youth.

EATING FREQUENCY AND ADIPOSITY

Previous research focused on eating frequency and its relationship to measures of adiposity has yielded mixed results, especially during childhood and adolescence, both of which are crucial periods in obesity development and the shaping of lifelong habits[11,12]. In 1964, Fabry et al. was the first to demonstrate an inverse relationship between eating frequency and adiposity in adult populations[46,47]. In 1966, Fabry et al. conducted a yearlong randomized controlled trial with 226 children (6-16y) utilizing two intervention boarding schools (one consuming 3 EOs per day and another consuming 7 EOs per day) and one control boarding school which maintained the regular 5 EOs per day intake[48]. Fabry et al. found no difference in caloric intake between groups, however, did find the sum of skin fold measurements (arm, back, and abdomen) to increase more in the 3 EO school compared to the schools that consumed 5 and 7 EOs per day. Multiple cross-sectional and longitudinal studies have supported this finding in youth populations[13-15,31]. Zerva et al.[13] conducted a cross-sectional study with 151 children (9-11 y) and found an inverse association between eating frequency and percent body fat, as measured by sum of skinfolds. Similarly the prospective longitudinal study by Ritchie et al.[15], with over 2,000

African American and Caucasian girls found that lower eating frequency was related to greater increases in BMI and waist circumference over a ten year period, independent of socioeconomic status, total energy intake, and physical activity levels. Thompson et al. [32] conducted a longitudinal study with 101 predominantly NHW girls (8-19y) and found that consuming 4 or more EOs per day was inversely associated with the change in BMI z-scores over the ten year period. Yet, eating frequency patterns among Hispanic youth and the potential relationship with adiposity and obesity risk remains largely unknown[17].

A recent meta-analysis by Schoenfeld et al.[49] including 15 randomized controlled trials addressing the effects of eating frequency on changes in weight and body composition found that increases in eating frequency were associated with reductions in fat mass and body fat percentage, as well as increases in fat-free mass. However, these findings need to be interpreted cautiously as they seem to be driven by a single study in Japanese boxers[50]. Furthermore, the above meta-analysis was comprised of an exclusively adult population. To our knowledge, there has yet to be a randomized control trial investigating the potential association between eating frequency and obesity risk in any youth population, let alone high-risk Hispanic youth. Interestingly, a recent controlled feeding trial by Chapelot et al.[51] found that switching from a 4 EO pattern to a 3 EO pattern resulted in an increase in fat mass in a sample of 24 young adult males (19-25 y). Smeets et al.[52] found that consuming 3 EOs per day compared to 2 EOs per day resulted in an increase in 24-h fat oxidation in a sample of 14 females(19-29 y). Thus, further research on the mechanism of action concerning eating frequency and obesity risk is needed and the potential mechanisms will be discussed within subsequent sections.

EATING FREQUENCY AND CALORIC INTAKE

A national study by Kerver et al.[10] with 15,978 adults found that increased daily eating frequency to be associated with increased daily energy intake, where participants who consumed ≥5 EOs per day consumed approximately 800 kcals/d more than those who consumed ≤2 EOs per day. Among youth (2-19 y) each additional eating occasion predicted a 200 kcal increase in energy intake[8]. In free-living youth populations, research consistently shows a positive relationship between eating frequency and caloric intake[1,2,13], yet a more consistent inverse relationship with obesity and adiposity measures[13,15]. Surprisingly increased eating frequency has not been shown to have an effect on resting metabolic rate or total 24-h energy expenditure[52-56]. Whereas, data on the effect of eating frequency on the thermic effect of food has yielded mixed results[57-59]. The effect of eating frequency on metabolic rate and thermic effect of food will be explored in more detail below and understanding how eating frequency impacts adiposity and metabolic disease despite resulting in increased caloric intake warrants further investigation.

EATING FREQUENCY AND METABOLIC DISEASE RISK

As mentioned previously Hispanic youth are at an increased risk of metabolic diseases and it remains unknown how eating frequency may impact this risk. A cross-sectional study conducted by Smith et al.[60] with 2,775 multi-ethnic young adults (26-36 y) found increased eating frequency to be inversely associated with fasting glucose and insulin, triglycerides, total cholesterol, and low-density lipoprotein (LDL) cholesterol. However, these findings were only significant in men, and no explanation of exclusion factors was included. In a crossover controlled feeding trial by Leidy et al.[61] with 13 overweight or obese males found that frequent eating (6 EOs) compared to less frequent

eating (3 EOs) resulted in a 30% decrease in the glucose area under the curve (AUC) and a 20% decrease in the insulin AUC across the 11-h testing period. Carlson et al.[62] conducted an eight-week cross-over feeding trial with 15 healthy adults (40-50 y) and found that 1 EO per day compared 3 EOs per day condition, which were isocaloric, resulted in increased fasting glucose as well as more prolonged elevation of plasma glucose throughout an oral glucose tolerance test. However, no differences were seen in fasting insulin, Homeostatic model assessment of insulin resistance (HOMA-IR), leptin, ghrelin, adiponectin, or resistin, between the two feeding conditions. In a very similar cross-over feeding trial containing the same experimental groups and duration found that 1 EO per day compared to 3 EOs per day resulted in a significant increases in hunger, blood pressure, and total, LDL, and high-density lipoprotein (HDL) cholesterol, yet a slight reduction in fat mass[63]. It is arguable whether 1 EO per day is representational of habits that are commonly seen in a free living population and among our dietary data from Hispanic youth (8-18y) no subject averaged 1 EO per day[2]. Overall, there is a paucity of data linking eating frequency to specific metabolic disease markers and more research is warranted, specifically in high-risk Hispanic youth.

EATING FREQUENCY AND SATIETY

Increased eating frequency has consistently been related to increased satiety measures in adult populations[52,61,63,64]. In the cross-over controlled feeding study by Leidy et al.[61] with 13 overweight or obese males, less frequent eating (3 EOs) vs. frequent eating (6 EOs) led to higher satiety throughout the day, but no difference in ghrelin or peptide YY was observed between groups. Smeets et al.[52] also found that

consuming 3 EOs per day compared 2 EOs per day resulted in greater satiety in a sample of 14 females (19-29 y). These studies controlled for calories but did not address the impact eating frequency may have on ad libitum food intake. Thus, Speechly et al.[64] conducted a cross-over study with eight lean males (19-29 y) where an isocaloric breakfast was consumed in one EOs or five separate EOs consumed every hour over the testing period. Subjects who consumed breakfast in one EO ate 27% more at a subsequent ad libitum lunch, highlighting that infrequent eating may lead to poorer appetite control. Interestingly, utilizing an identical study design with seven obese men (20-55 y), Speechly et al.[65] replicated the findings and found the exact same increase (27%) in the ad libitum lunch intake after the single meal. It is reasonable to infer that increased satiety observed in regards to increased eating frequency may reduce the motivation to eat and therefore reduce energy intake overall, however the vast majority of the eating frequency research shows a positive relationship between eating frequency and energy intake. It is also plausible that reduced eating frequency may result in eating behaviors that resemble binge eating which has been related to increases on metabolic disease parameters and adiposity measures[66]. Thus, much more research is needed to examine the exact mechanisms of how eating patterns may impact satiety, hunger, and ad libitum dietary intake in free-living youth.

EATING FREQUENCY AND METABOLIC RATE AND THE THERMIC EFFECT OF FOOD

Popular media has consistently advocated more frequent eating or grazing as a healthy habit that may "stoke" or "rev" one's metabolism. A recent review of popular

media sources found the ideal eating frequency recommendation given was 6 EOs per day[67]. Yet, not a single study to date has found a statistically significant difference in metabolic rate between 1, 2, 3, 5, 6 or even 7 EOs in a 24 hour period[52-56]. A cross-over study with eight young adult males (18-23 y) examined the difference in metabolic rate between 2 EOs and 6 EOs per day which were isocaloric. Subjects stayed on each dietary regiment for two weeks and occupied a whole room calorimeter for two 31-h periods[53]. This study found no differences in metabolic rate or energy expenditure between the two eating frequency conditions, despite a small, albeit significant, observed weight gain in the 2 EOs per day group. However, this study kept the activity patterns constant and as previously mentioned increased eating frequency may subsequently increase physical activity levels. In a similar 2 day cross-over study with 13 males and females (18-23 y) no significant differences in 24-h energy expenditure as measured by a whole room calorimeter were found between 2 EOs and 7 EOs per day conditions[54]. Thus, to date it does not appear that eating more frequently impacts metabolic rate and given the pervasiveness of the current media recommendation further research into the potential impacts of eating frequency on obesity and metabolic disease risk is warranted to better inform the general public of the potential benefits of frequent eating.

Another potential mechanism to consider when investigating eating frequency is the potential impact on the thermic effect of food (TEF). The thermic effect of food is defined as the increase in metabolic rate after the ingestion of a meal. To date, studies examining the impact of eating frequency on TEF have yielded mixed results. One study found an increase in TEF in 1 EO compared to 6 EOs[57], while another study showed an increase in TEF in 4 EOs compared to 1 EO[58], whereas the majority of studies show no

significant effect of eating frequency on TEF[52,59,68]. Tai et al.[57] examined the effect of 1 large EO of 750 calories vs. 6 smaller EOs of 125 calories provided 30 minutes over the same time period on TEF in seven women (23-30 y) and found that the one large EO resulted in a slightly higher TEF of 3.4% and the authors hypothesized that this was due to a more rapid absorption of nutrients given that gastric emptying is slower when food is given continuously[69]. Similarly, another study by Leblanc et al. in six subjects (21-28 y) compared 1 vs. 4 EOs given over a four-hour period and found that the 4 EO pattern resulted in a slightly higher TEF. However, as mentioned previously eating one time per day is not reflective of normal eating patterns seen in free-living populations. A comprehensive review by Bellisle et al.[68] concluded that there is no strong evidence to support a biologically significant difference in TEF in response to different eating frequency and furthermore, the role of TEF on body weight regulation itself remains controversial[70]. To our knowledge, no research study has investigated any potential mediating effect that an increase in TEF from an increase or decrease in eating frequency may have on adiposity parameters. In addition, no study has examined how eating frequency impacts TEF in a youth population.

EATING FREQUENCY AND OTHER EATING PATTERNS

When investigating eating frequency it is pivotal to take into account the research on other eating patterns, specifically: breakfast consumption, snacking, and late night eating. These different eating patterns are likely inter-related and have been found to

impact energy balance, obesity, and metabolic disease risk, but research examining at the how these eating habits fit together in order to better inform public health messages is lacking[32,71]. Breakfast consumption has been identified as a eating behavior that may lower obesity risk[72], whereas more EOs in the evening/night has been related to greater obesity risk[32], and increased snacking has yielded mixed results[73]. The following section will address each of these eating patterns and any potential relationship with eating frequency.

Thus, when investigating different dietary patterns it is pivotal to understand that these patterns are likely inter-related. Research has shown that breakfast consumption and increased eating frequency are strongly associated[74,75]. Jennings et al.[76] reported that those with the most EOs per day were also the most likely to eat breakfast in a sample of children (9-10y). Conversely, Toschke et al.[74] found that eating frequency was inversely related to obesity risk in a sample of over 4,000 younger children (5-6y) and that this association was only marginally affected by breakfast consumption. In a population of 93 overweight/obese Hispanic youth (10-17y) our group found breakfast skipping to be related to decreased caloric intake, yet increased visceral adipose tissue (VAT)[77]. A recent review by Blondin et al.[78] highlighted the fact that even though eating frequency and breakfast consumption may be confounding variables few studies have actually analyzed the potential mediating effects between these two eating patterns.

Little research is available on the relationship between eating frequency and late night eating, and the potential confounding relationship these two eating patterns may have on obesity risk. However, a study by Thompson et al.[32] found that consuming 4 to

5.9 EOs per day with no more than 1.9 EOs between the hours 1700 and 0559 was negatively associated with the change in BMI z-scores over a ten year period in a sample of 101 healthy girls (8-12y). This study suggests that eating more frequently is healthier when these eating occasions do not come in the evening hours or late night when the body may be more apt to store fat[79].

Epidemiological research has found increased occurrence and percentage of energy consumed from snacking over the last three decades[80] and increased snacking has been found to be associated with increased caloric and carbohydrate intake in a sample of 4,357 adolescents (12-19 y)[81]. From the year 1977 to 2005 snacking frequency in adolescents (12-19 y) increased from 1.0 (14% of energy intake) to 1.7 (23% of energy intake) snacks per day[82]. However, higher snacking was not significantly associated with BMI in adolescents age (12-19 y)[82]. Furthermore, a recent review by Larson and Story[73] including more than 30 studies on snacking and the potential impact on obesity risk in children (2-19 y) found that the majority of studies found no relationship or an inverse relationship with obesity risk. Yet, one of the major limitations in eating frequency research is a lack of a standard definition of key terms such as meals and snacks which makes comparing these studies very difficult[83]. This is why we chose to use the Gibney et al.[30] eating occasion definition over breaking different EOs into snacks or meals depending on percentage of caloric intake or time of day given the highly irregular eating habits of Hispanic youth[2].

Furthermore, it remains unknown how all these eating patterns fit together, especially within high risk Hispanic youth, thus identifying how these eating patterns may uniquely or interdependently affect obesity and metabolic disease risk is pivotal to better inform public health messages. Thus, more research is warranted to examine the exact relationship between eating frequency, breakfast consumption, and late night eating, and how these different eating patterns may affect obesity and metabolic disease risk, especially in Hispanic youth.

SUMMARY

Hispanics are the largest growing ethnic minority in the US and Hispanic youth are at increased risk of obesity and metabolic diseases. This population has also been found to eat less often and more inconsistently than their NHW counterparts. Increased eating frequency has consistently been associated with decreased adiposity and metabolic disease parameters, as well as increases in satiety and physical activity, yet to our knowledge no study has been conducted evaluating the impact eating frequency on any of these markers in a population of exclusively Hispanic youth. Furthermore, to our knowledge there has not been a study published on the dietary habits of Hispanic college students, which is a growing and completely unstudied population.

AIMS

The aims of this research include: 1) determine the impact of eating frequency on adiposity measures, including anthropometrics, total body fat, and fat distribution and metabolic disease risk in Hispanic youth; 2) assess the relationship between eating frequency, breakfast consumption, obesity risk, and dietary and physical activity behaviors in a large sample of exclusively Hispanic college freshmen; and 3) investigate the relationship between eating frequency and adiposity measures, specifically visceral adipose tissue and habitual dietary intake in a population of exclusively Hispanic college freshmen.

Chapter 3: Meal Skipping Linked to Increased Visceral Adipose Tissue and Triglycerides in Overweight Minority Youth

House BT, Cook LT, Gyllenhammer LE, et al. Meal skipping linked to increased visceral adipose tissue and triglycerides in overweight minority youth. *Obesity (Silver Spring).* Apr 24 2013. DSM, MW, JD, and MG designed and supervised the various research studies used in this analyses; DSM, MW, JD, MG obtained the funding; BH, JS, JD, LG, and LC analyzed data; BH and JD wrote the paper; All authors contributed to editing the manuscript. BH wrote the manuscript and BH and JD take responsibility for final content presented.

ABSTRACT

Objective: To investigate the impact of eating frequency on dietary intake, physical activity (PA), metabolic, and adiposity measures in minority youth.

Design and Methods: This analysis included 185 overweight (≥85th BMI percentile) Hispanic and African American youth (8-18 years) with the following cross-sectional measures: height, weight, BMI, dietary intake, body composition, metabolic parameters, PA, visceral adipose tissue (VAT), and subcutaneous adipose tissue. Each eating occasion (EO) was defined as ≥50 calories and ≥15 minutes from any previous EO. Participants were dichotomized based on EOs per 24-h into meal skippers <3 EO (MS; n=27) or normal/frequent eaters ≥3 EO (NFE; n=158). ANCOVAs were used to assess dietary intakes, metabolic outcomes, adiposity, and PA between eating frequency groups.

Results: MS compared to NFE consumed 24% fewer calories per 24-h ($p \leq 0.01$), 21% more calories per EO ($p \leq 0.01$), ate 40% less often ($p \leq 0.01$), had 18% higher triglycerides ($p=0.03$), and 26% more VAT ($p=0.03$), with no differences in PA.

Conclusions: Although meal skipping was associated with decreased energy intake, it was linked to increased calories per EO and higher triglycerides and VAT, which are strong indicators of deleterious metabolic profiles. These findings elucidate that meal skipping may be associated with increased VAT and related metabolic diseases in high-risk minority youth.

INTRODUCTION

Previous research focused on eating frequency and its relationship to measures of adiposity has yielded mixed results, especially during childhood and adolescence, both of which are crucial periods in obesity development and the shaping of lifelong habits[11,12]. Currently, 16.9% of children in the United States are obese and 31.8% are either overweight or obese[84,85]. Hispanic and African American youth are both at increased risk of obesity, 21.2% and 24.3%, respectively compared to 14.0% in non-Hispanic whites , and it is not fully understood how eating frequency impacts obesity risk in overweight/obese minority youth[85]. Minority youth have also been shown to be more apt to eat less often, but eating frequency patterns in these populations remain largely unknown[17]. Thus, quantifying eating frequency in both Hispanic and African American

youth and assessing if it plays a role in the elevated propensity toward obesity and metabolic disorders is warranted[12,86].

Research on the association between eating frequency and adiposity in youth populations has more consistently shown an inverse relationship [13-15], although some studies have found this inverse relationship to be mitigated by or related to physical activity (PA) [13,44]. While other studies have shown eating frequency to have no relation to adiposity measures [17,87], or even a positive relationship[88]. A recent longitudinal study comparing African American and Caucasian adolescent girls from 9 to 19 years of age with a mean baseline BMI of 18.5 found that lower meal frequency was related to greater increases in BMI and waist circumference over the ten year period, independent of socioeconomic factors, total caloric intake, and PA[14]. Yet, currently no study has investigated how eating frequency may be related to specific fat distribution measured by magnetic resonance imaging (MRI), or the relationship between eating frequency and adiposity in a combined sample of Hispanic and African American youth. Thus, the overall goal of this study was to examine how eating frequency was associated with dietary, metabolic, adiposity, and PA measures. We hypothesized that meal skipping in relation to normal/frequent eating would be positively associated with energy intake, fasting glucose and insulin, lipid profiles, and adiposity measures and inversely associated with insulin action and PA in Hispanic and African American youth.

METHODS

Subjects:

For this analysis, we pooled subjects from a variety of studies using identical measures and measurement protocols from the University of Southern California Childhood Obesity Research Center [77,89-92]. In short, subjects were recruited from schools, community centers, health clinics, and health fairs by way of word of mouth, flyers/brochures, and in-person contact. There were a total of 413 subjects, 335 of these participants had specific fat distribution data and 221 had complete dietary data. The final sample included 185 Hispanic and African American children and adolescents (8–18 years) for whom both complete specific fat deposition and dietary data were available. Inclusion criteria for the studies were as follows (i) Hispanic or African American origin, (ii) 8 -18 years of age, (iii) BMI $\geq 85^{th}$ percentile for age and gender based on Center for Disease Control and Prevention guidelines [93], (iv) not participating in a PA, nutrition, or weight reduction program, and (v) an absence of diabetes via an oral glucose tolerance test. Participants were excluded for the following reasons: if they were taking any medication known to affect body composition, or if they had been diagnosed with a disease/s or syndrome known to affect body composition or fat distribution. All studies were approved by the Institutional Review Board of the University of Southern California. Informed written consent and assent were obtained from both parents and children before testing commenced.

Anthropometrics and Adiposity Measures:

A certified healthcare provider performed a detailed physical exam where Tanner staging was determined using established guidelines[94,95]. Height and weight were measured to the nearest 0.1 kg and 0.1 cm using a beam medical scale and a wall-mounted stadiometer, respectively, and the average of 2 measurements was used for the analysis. BMI and BMI z-scores were determined by using EPII 2000 software (version 1.1; Centers for Disease Control and Prevention, Atlanta, GA)[93]. Fat mass and total lean mass (n=155) were measured by dual-energy X-ray absorptiometry (DXA) using a Hologic QDR 4500W (Hologic, Bedford, MA). Abdominal fat distribution was measured by multiple slice MRI on a General Electric 1.5-Tesla magnet. Slices were acquired by using a 420-mm field of view and field of view phase of 75%. Three abdominal scans were performed consecutively, and the total acquisition time was 24 s per total abdominal scan. Each scan obtained 19 axial images of the abdomen with a thickness of 10 mm. After image acquisition, subcutaneous adipose tissue (SAT) and visceral adipose tissue (VAT) were segmented by using image analysis software (SliceOmatic Tomovision, Montreal, Canada) at the Image Reading Center (New York, NY). SAT and VAT volumes were calculated from these images as previously described[89]. Hepatic fat fraction (HFF; n=174) was assessed during the same MRI test by using a modification of the Dixon 3-point technique, method previously described[96].

Blood Assays:

After an overnight fast, a topical anesthetic (EMLA cream; AstroZeneca, Wilmington, DE) was applied to the antecubital area of both arms at 0730; 1 h later, a

flexible intravenous catheter was inserted into one of the arms. Two fasting blood samples, at 15 and 5 min, were obtained for measurement of basal glucose and insulin values (n=185). Homeostatic model assessment: insulin resistance (HOMA-IR) was calculated by multiplying fasting glucose by fasting insulin and dividing by an established constant[97]. At time zero, glucose (25% dextrose; 0.3 g/kg body wt) was administered intravenously. Blood samples were then collected at the following times: 2, 4, 8, 19, 22, 30, 40, 50, 70, 100, and 180 min. Insulin [0.02 units/kg body wt; Humulin R (regular insulin for human subjects), Eli Lilly and Company, Indianapolis, IN] was injected intravenously at 20 min. Plasma was analyzed for glucose and insulin concentrations, and values were entered into the MINMOD MILLENNIUM 2003 computer program (version 5.16; RN Bergman, USC, Los Angeles, CA) for determination of insulin sensitivity (SI; n=175), acute insulin response (AIR; n=175), and disposition index (DI; n=175). Blood samples from the frequently sampled intravenous glucose tolerance test were immediately centrifuged for 10 min at 2500 rpm and 8–10°C to obtain plasma, and aliquots were frozen at 70 °C until assayed. Glucose was assayed in duplicate by using the glucose oxidase method and a Yellow Springs Instrument 2700 Analyzer (Yellow Springs Instrument, Yellow Springs, OH). Insulin was assayed in duplicate by using a specific human insulin enzyme-linked immunosorbent assay kit (Linco, St Charles, MO). Fasting lipids (n=153) including triglycerides, low density lipoprotein (LDL)-cholesterol, and high density lipoprotein (HDL)-cholesterol were assessed using Vitros Chemistry DT Slides (Johnson and Johnson Clinical Diagnostics,

Inc., Rochester, NY).

Dietary Intakes:

Dietary intake was assessed from two or three 24-h diet recalls using the multiple-pass technique. A majority of the sample (72%) had three dietary recalls available. All recalls were administered or checked by a trained dietary technician in person or by telephone. Nutritional data was analyzed by using the Nutrition Data System for Research (NDS-R 2010 version 5.0_35). The NDS-R program calculated key dietary variables for this analysis, including mean energy, total fat, protein, carbohydrates, saturated fat, total sugar, added sugar, dietary fiber, soluble fiber, and insoluble fiber. We then calculated the percent caloric intake of total fat, protein, carbohydrates, saturated fat, sugar, and added sugar and the grams of fiber per 1000 kcals. The dietary data was carefully screened for plausibility. Data was first screened by evaluating the participants' comments, and one subject was excluded because they reported an illness. The dietary data was then examined for plausibility of caloric intake according to the Willett exclusion criteria and one subject was excluded[98]. We further screened for dietary plausibility by performing a regression of caloric intake on body weight, but no subject had a standardized residual greater than three SD above or below the mean.

Eating Frequency Analysis:

The following aspects of eating frequency were examined: average number of EOs per 24-h and dichotomized eating frequency groups. The dichotomized eating frequency groups were meal skippers (MS; n=27), classified as those subjects who ate

less than three times per 24-h, and normal/frequent eaters (NFE; n=158), classified as those subjects who ate three or more times per 24-h. We followed a previously established eating frequency methodology [30] and did not make a distinction between meals and snacks, and instead examined eating frequency based on the number of EOs; each EO had to be at least 50 calories and at least 15 minutes from any previous EO[30].

Physical Activity:

To assess PA in this population, subjects were instructed to wear Actigraph accelerometers (GT1M or 7164, Actigraph, LLC., Pensacola, FL) for seven days, except during water-based activities or when sleeping. Accelerometers were set to monitor activity in 15-second epochs, which were collapsed to 60-second epochs during analysis. Data was reduced using an adapted version of the SAS code used for the 2003–2004 National Health and Nutrition Examination Survey available at http://riskfactor.cancer.gov/tools/nhanes_pam. A correction factor was applied to allow for comparison between the two Actigraph monitor models[99]. The methodology for categorizing sedentary behavior (SED) and moderate and vigorous PA are previously described[100]. The amount of time the participant wore the device was determined by subtracting nonwear time from 24-h. Nonwear time was defined by an interval ≥60 consecutive minutes of 0 activity counts, with allowance for 1–2 mins of counts between 0 and 100. Days with less than 8h of wear data were not considered acceptable, and only participants with ≥3 days of acceptable accelerometry data were included in the PA analysis. Subjects with valid data (n=112) wore the accelerometers for a mean ± SD of

13.2 ± 1.6 hours/day for 5.7 ± 2.5 days, and there was no difference in the number of valid days or wear time between groups. Data from all acceptable days was averaged and included the following variables: number of wear days, average number of minutes worn, average counts per minute, percent of wear time, and minutes spent in moderate to vigorous PA (MVPA) and SED.

Statistics:

Data was examined for normality, and transformations were made if the data was found to be significantly different from normal. The following outcome variables were non-normally distributed and were either log or inversely transformed before the analysis: mean energy intake, total dietary fat, protein, carbohydrate, saturated fat, dietary fiber, soluble fiber, insoluble fiber, total sugar, added sugar, VAT, SAT, HFF, AIR, triglycerides, total cholesterol, and HDL cholesterol. However, we present nontransformed values in the tables and figures for ease of interpretation. Fasting Insulin, HOMA-IR, and percent time spent in MVPA were unable to be normalized within this population. Correlations and partial correlations were analyzed between the number of EOs per 24-h and age, height, weight, adiposity, metabolic, and PA measures, as well as energy/nutrient intakes. Multiple linear regressions were run to assess the percent of variance in eating frequency explained by gender, sex, tanner stage, age, height, and weight. A t-test or chi square analyses were used to assess differences in age, sex, ethnicity, and tanner stage between eating frequency groups. ANCOVA analyses were used to assess differences in dietary intake variables, adiposity and PA measures, and

metabolic parameters between the two eating frequency groups. In all models the following *a priori* covariates were included: tanner stage, sex, and ethnicity. Mean energy intake was used as a covariate in the analysis of metabolic parameters and adiposity and PA measures. BMI was included as covariate for all metabolic parameters and HFF. Total fat was included as a covariate for total lean mass and vice versa. Height was also included as a covariate for total fat and total lean mass. SAT was included as a covariate for VAT and vice versa, and SI was included as a covariate for AIR. These covariates have been found applicable in previous work by our group[89-91]. All analyses were performed by using SPSS version 20.0 (SPSS, Chicago, IL), and the significance was set at $p \leq 0.05$. Post-hoc power analyses revealed that power ranged from 0.6 (visceral adipose tissue), 0.8 (triglycerides), and 1.0 (energy intake) for the main outcome variables.

RESULTS

The basic demographic data and adiposity measures are presented in **TABLE 3.1** There were 185 overweight participants that had complete anthropometric, dietary, and fat distribution data, and body composition, metabolic, and PA data were available in smaller samples. The sample was 61% Hispanic and 71% female.

TABLE 3.1
Subject Characteristics [1,2]

Physical and Adiposity Measures (n=185)	
Sex M/F	54/131
Ethnicity Hispanic/African American[3]	113/72

TABLE 3.1 Continued

Age (y)	14.8 ± 2.7
Tanner stage	
1	16
2	24
3	6
4	24
5	112
Height (cm)	159.3 ± 12.7
Weight (kg)	84.0 ± 25.1
BMI (kg/m^2)	32.4 ± 7.1
BMI z score	2.0 ± 0.5
VAT **(cm^3)**	**1,536.3 ± 1,080.0**
SAT **(cm^3)**	**10,642.9 ± 6,175.3**
HFF (%)[3]	6.6 ± 6.7
Total fat (kg)[4]	31.9 ± 11.9
Total lean tissue mass (kg)[4]	49.4 ± 11.5
Total body fat (%)[4]	**37.8 ± 6.8**
Metabolic Parameters (n=185)	
Fasting glucose (mg/dL)	89.7 ± 5.9
Fasting insulin (μU/mL)	18.4 ± 15.9
HOMA-IR	4.2 ± 3.6
SI [x10-4 min-1/(μU/mL)][5]	1.6 ± 1.0
AIR (μU/mL X 10 min)[5]	1,565.3 ± 1,056.7
DI (x10-4/min-1)[5]	2,045.5 ± 1,111.1
Total cholesterol (mg/dL)[6]	144.4 ± 29.2
Triglyceride (mg/dL)[6]	85.3 ± 37.2
LDL-cholesterol (mg/dL)[6]	89.5 ± 28.3
HDL-cholesterol (mg/dL)[6]	37.8 ± 8.5

[1] Data presented as mean ± SD
[2] AIR - Acute Insulin Response, DI – Disposition Index, HOMA-IR - Homeostatic Model Assessment: Insulin Resistance, HDL - High Density Lipoprotein, HFF - Hepatic Fat Fraction, LDL - Low Density Lipoprotein, SAT - Subcutaneous Adipose Tissue, SI – Insulin Sensitivity, VAT - Visceral Adipose Tissue
[3] n = 174, [4] n = 155, [5] n = 175, [6] n = 153

TABLE 3.2 presents dietary and PA data. The average number of EO per 24-h was 3.8 and there was no difference in eating frequency between Hispanics and African Americans (p=0.73). In the subsample of subjects with accelerometer PA data, greater than 50% of wear time was spent in SED.

TABLE 3.2
Behavioral Characteristics [1,2]

Dietary Variables (n=185)	
Eating occasions per day	3.8 ± 1.0
Energy per eating occasion (kcal)	508.4 ± 178.9
Energy (kcal)	1,836.9 ± 566.2
Total fat (g/day)	72.1 ± 29.1
(%kcal)	34.9 ± 6.9
Total protein (g/day)	69.9 ± 25.2
(%kcal)	15.4 ± 3.6
Total carbohydrate (g/day)	235.1 ± 80.1
(%kcal)	51.3 ± 8.5
Total saturated fat (g/day)	23.6 ± 9.9
(%kcal)	11.5 ± 3.0
Added sugars (g/day)	77.8 ± 46.2
(%kcal)	16.7 ± 7.8
Total sugars (g/day)	108.4 ± 50.6
(%kcal)	23.6 ± 7.8
Dietary fiber (g/day)	13.4 ± 5.6
(g/1000kcal)	7.5 ± 2.7
Insoluble fiber (g/day)	9.1 ± 4.2
(g/1000kcal)	5.1 ± 2.1
Soluble fiber (g/day)	4.2 ± 1.8
(g/1000kcal)	2.3 ± 0.9
Physical Activity (n=112)	
Average counts per min	366.6 ± 111.8
Percent wear time in MVPA (%)	7.1 ± 4.9
Percent wear time in SED (%)	58.3 ± 8.7

[1] Data presented as mean ± SD
[2] MVPA – Moderate to Vigorous Physical Activity, SED – Sedentary Behavior

Correlations, unadjusted and adjusted, between the number of EOs per 24-h and energy/nutrient intakes, metabolic parameters, and adiposity and PA measures are shown

in **TABLE 3.3** The number of EOs per 24-h was positively associated with energy intake ($p \leq 0.01$), percent calories from carbohydrate ($p \leq 0.01$) and sugar ($p=0.03$), all fiber intakes ($p \leq 0.01$), and negatively associated with percent of calories from total fat ($p \leq 0.01$), after adjusting for covariates. There were also significant negative correlations between the number of EOs per day and triglycerides ($p=0.02$) and **lean mass ($p=0.05$)**, and a positive association with HDL-cholesterol ($p=0.02$) and SAT ($p=0.02$), after adjusting for covariates. There were no significant associations between eating frequency and PA measures.

TABLE 3.3

Unadjusted and adjusted correlations between number of eating occasions per day and nutrient intake and adiposity measures.[1,2]

	unadjusted		**adjusted**	
Adiposity Measures (n=185)	r	p value	r	p value
BMI (kg/m^2)	-0.13	0.09	-0.02	0.75
BMI z-score	-0.08	0.27	-0.02	0.76
SAT **(cm^3)**	0.04	0.61	0.17	0.02
VAT **(cm^3)**	-0.10	0.19	-0.13	0.09
HFF (%)[3]	-0.04	0.60	-0.06	0.49
Total fat (kg)[4]	-0.05	0.50	**0.03**	**0.76**
Total lean tissue mass (kg)[4]	-0.22	≤0.01	**-0.16**	**0.05**
Total body fat (%)[4]	**0.11**	**0.17**	**0.10**	**0.21**
Metabolic Parameters (n=185)				
Fasting glucose (mg/dL)	-0.05	0.51	-0.13	0.08
Fasting insulin (μU/mL)	-0.06	0.40	-0.08	0.32
HOMA-IR	-0.06	0.40	-0.08	0.29
Insulin sensitivity [x10-4 min-1/(μU/mL)][5]	-0.06	0.49	-0.07	0.38
AIR (μU/mL X 10 min)[5]	0.03	0.68	-0.05	0.55
DI (x10-4/min-1)[5]	0.01	0.87	-0.04	0.61
Total cholesterol (mg/dL)[6]	-0.08	0.33	-0.07	0.40
Triglyceride (mg/dL)[6]	-0.18	0.03	-0.19	0.02
LDL-cholesterol (mg/dL)[6]	-0.08	0.32	-0.08	0.37
HDL-cholesterol (mg/dL)[6]	0.16	0.05	0.19	0.02
Nutrient Intakes (n=185)				

TABLE 3.3 Continued				
Energy (kcal)	0.28	≤0.01	0.40	≤0.01
Total fat (%kcal)	-0.21	≤0.01	-0.17	0.02
Total protein (%kcal)	-0.07	0.33	-0.13	0.07
Total carbohydrate (%kcal)	0.21	≤0.01	0.20	≤0.01
Total saturated fat (%kcal)	-0.11	0.13	-0.13	0.07
Added sugars (%kcal)	0.05	0.51	-0.04	0.60
Total sugars (%kcal)	0.21	≤0.01	0.18	≤0.01
Dietary fiber (g/1000kcal)	0.24	≤0.01	0.21	≤0.01
Insoluble fiber (g/1000kcal)	0.22	≤0.01	0.19	≤0.01
Soluble fiber (g/kcal)	0.24	≤0.01	0.21	≤0.01
Physical Activity (n=112)				
Average counts per min	-0.09	0.34	-0.06	0.55
Percent wear time in MVPA (%)	0.07	0.49	0.04	0.70
Percent wear time in SED (%)	-0.06	0.53	0.07	0.49

[1] AIR - Acute Insulin Response, DI - Disposition Index, HOMA-IR - Homeostatic Model Assessment: Insulin Resistance, HDL - High Density Lipoprotein, HFF - Hepatic Fat Fraction, LDL - Low Density Lipoprotein, MVPA - Moderate to Vigorous Physical Activity, SAT - Subcutaneous Adipose Tissue, SED - Sedentary Behavior , SI - Insulin Sensitivity, VAT - Visceral Adipose Tissue

[2] Pearson unadjusted and adjusted correlations between eating occasions per day and adiposity measures, nutrient intakes, and metabolic parameters. *A priori* covariates used in adjusted correlations: tanner stage, sex, ethnicity, mean energy (adiposity, metabolic parameters, and physical activity measures), BMI (for metabolic parameters and HFF), total fat **and height** (for total lean), total lean **and height** (for total fat), SAT (for VAT), VAT (for SAT), SI (for AIR)

[3] n = 174, [4] n = 155, [5] n = 175, [6] n = 153

TABLE 3.4 presents metabolic and adiposity measures by the two eating frequency groups. We found no significant difference in ethnicity, sex, age, tanner stage height, or weight between eating frequency groups and in a multiple linear regression only 5.8% of the variation in eating frequency was explained by ethnicity, sex, tanner stage, age, height, and weight. Independent of covariates, MS showed 26% higher VAT compared with NFE (p=0.03). Increased VAT significance among MS remained when age and/or height were included in the model. There were no other significant differences in adiposity measures between the two groups. There were significant differences in

metabolic parameters, MS had a 20% higher AIR (p=0.05) and 18% higher triglycerides (p=0.03).

TABLE 3.4

Adiposity measures and metabolic parameters between eating frequency groups[1,2,3]

	Meal Skippers (n=27)	Normal/Frequent Eaters (n=158)	p value
Physical and Adiposity Measures (n=185)			
Sex M/F	**12/15**	**42/116**	**0.07**
Ethnicity Hispanic/African American[3]	**16/11**	**97/61**	**0.83**
Age (y)	**15.5 ± 2.1**	**14.7 ± 2.8**	**0.16**
Tanner stage			**0.18**
1	**1**	**15**	
2	**3**	**21**	
3	**3**	**3**	
4	**3**	**21**	
5	**17**	**98**	
Height (cm)	**163.0 ± 9.3**	**158.7 ± 13.1**	**0.30**
Weight (kg)	**92.3 ± 21.9**	**82.5 ± 25.4**	**0.14**
BMI (kg/m^2)	34.5 ± 6.9	32.1 ± 7.1	0.12
BMI z score	2.2 ± 0.5	2.0 ± 0.5	0.12
SAT **(cm^3)**	**11,716.3 ± 6,263.7**	**10,459.5 ± 6,161.4**	0.44
VAT **(cm^3)**	**1,981.0 ± 1,607.0**	**1,460.4 ± 948.4**	0.03
HFF (%)[4]	8.6 ± 8.3	6.2 ± 6.4	0.41
Total fat (kg)[5]	34.2 ± 11.6	31.5 ± 12.0	**0.48**
Total lean tissue mass (kg)[5]	55.0 ± 10.8	48.4 ± 11.4	**0.06**
Total body fat (%)[5]	**37.1 ± 7.6**	**37.9 ± 6.7**	**0.95**
Metabolic Parameters (n=185)			
Fasting glucose (mg/dL)	90.4 ± 6.0	89.6 ± 5.9	0.35
Fasting insulin (μU/mL)	21.1 ± 16.2	18.0 ± 15.8	0.57
HOMA-IR	4.8 ± 3.9	4.0 ± 3.6	0.52
SI [x10-4 min-1/(μU/mL)][6]	1.4 ± 0.9	1.6 ± 0.9	0.56
AIR (μU/mL X 10 min)[6]	1,883.9 ± 1,121.9	1,509.9 ± 1,039.2	0.05
DI (x10-4/min-1)[6]	2,171.0 ± 1,231.1	2,023.7 ± 1,092.3	0.24
Total cholesterol (mg/dL)[7]	152.7 ± 35.0	142.8 ± 27.9	0.47
Triglyceride (mg/dL)[7]	100.4 ± 45.3	82.6 ± 35.0	0.03
LDL-cholesterol (mg/dL)[7]	96.9 ± 32.9	88.2 ± 27.3	0.49
HDL-cholesterol (mg/dL)[7]	35.8 ± 8.6	38.2 ± 8.5	0.10

[1] Data presented as mean ± SD

[2]AIR - Acute Insulin Response, DI - Disposition Index, HOMA-IR - Homeostatic Model Assessment: Insulin Resistance, HDL - High Density Lipoprotein, HFF - Hepatic Fat Fraction, LDL - Low Density Lipoprotein, SI - Insulin Sensitivity, SAT - Subcutaneous Adipose Tissue, VAT - Visceral Adipose Tissue

TABLE 3.4 Continued

[3] A t-test (for continuous variables) and chi-square analysis (for categorical variables) assessed differences in sex, ethnicity, and tanner stage between groups.

[4]ANCOVA analysis of adiposity measures, and metabolic parameters between meal skippers and normal/frequent eaters. *A priori* covariates included: tanner stage, sex, ethnicity, mean energy (for adiposity and metabolic parameters), BMI (for metabolic parameters and HFF), total fat **and height** (for total lean), total lean **and height** (for total fat), SAT (for VAT), VAT (for SAT), SI (for AIR)

[5] n = 174, [5] n = 155, [6] n = 175, [7] n = 153,

Dietary and PA variables between MS and NFE are depicted in **TABLE 3.5**. MS ate 40% less often ($p \leq 0.01$) and ate 21% more per EO ($p \leq 0.01$), while consuming 24% or on average 451 fewer calories ($p \leq 0.01$) than NFE. With this, MS ate less of all nutrients per day, but had a larger percent of calories from protein ($p=0.04$) and less from sugar ($p=0.04$) than NF. There were no significant difference in PA measures between MS and NFE.

TABLE 3.5

Dietary characteristics between eating frequency groups[1,2,3]

	Meal Skippers (n=27)	Normal/Frequent Eaters (n=158)	p value
Dietary Variables (n=185)			
Eating occasions per day	2.4 ± 0.3	4.0 ± 0.8	≤0.01
Energy per eating occasion (kcal)	621.7 ± 216.1	489.0 ± 164.9	≤0.01
Energy (kcal/day)	1,451.7 ± 469.0	1,902.7 ± 544.3	≤0.01
Total fat (%kcal)	36.8 ± 9.6	34.6 ± 6.4	0.25
Total protein (%kcal)	16.7 ± 5.1	15.2 ± 3.2	0.04
Total carbohydrates (%kcal)	49.4 ± 10.4	51.6 ± 8.1	0.28
Total saturated fat (%kcal)	12.46 ± 3.9	11.3 ± 2.9	0.11
Total sugars (%kcal)	20.4 ± 7.7	24.1 ± 7.7	0.04
Added sugars (%kcal)	15.5 ± 8.0	16.9 ± 7.7	0.46
Dietary fiber (g/1000kcal)	6.6 ±1.7	7.6 ± 2.8	0.16
Insoluble fiber (g/1000kcal)	4.4 ± 1.3	5.2 ± 2.1	0.13
Soluble fiber (g/1000kcal)	2.1 ± 0.7	2.3 ± 0.9	0.42
Physical Activity (n=112)			
Average counts per min	366.2 ± 116.1	366.6 ± 111.7	0.64
Percent wear time in MVPA (%)	5.7 ± 3.7	7.4 ± 5.1	0.41
Percent wear time in SED (%)	59.5 ± 10.1	58.0 ± 8.5	0.93

[1]Data presented as mean ± SD

[2] MVPA - Moderate to Vigorous Physical Activity and SED -Sedentary Behavior

[3]ANCOVA analysis of dietary variables between meal skippers and normal/frequent eaters. *A priori* covariates used: tanner stage, sex, ethnicity, and mean energy intake (for physical activity measures).

DISCUSSION

To our knowledge, this is the first analysis to examine the relationship between eating frequency and dietary, metabolic, adiposity, and PA measures in a combined sample of Hispanic and African American youth. Minority youth have higher rates of obesity [85]and are more likely to skip meals and eat less often[17], although it is not

understood how or if these findings are related. In the present analysis of overweight/obese minority youth, we show that increased eating frequency was positively associated with energy intake, and that MS ate significantly fewer calories, had 18% higher triglyceride values, and 26% more VAT in relation to NFE, while showing no significant differences in PA measures. These findings are consistent with other retrospective analyses[13,15] that have shown an inverse association between eating frequency and measures of adiposity while showing a positive association with caloric intake, specifically meal skippers in the present study consumed fewer calories, yet had higher amounts of visceral adipose tissue.

To date, the one controlled feeding study that examined how meal skipping impacted adiposity, metabolic, hunger, and PA measures in normal weight adults, found that those who ate a eucaloric diet in one vs. three EOs per day had reductions in fat mass, as measured via bioimpedance, and increases in hunger, total, LDL, and HDL cholesterol, and no changes in PA or fasting glucose[63]. However, this study utilized normal weight adults and no subject in our analysis averaged ≤1 EO per day. A small number of retrospective studies have examined eating frequency as it relates to body composition, but ours is the first with specific fat distribution attained via MRI. A study of children 9-10 years of age found that eating frequency was negatively associated with multiple measures of adiposity, but not percent body fat, and positively associated with vigorous PA in healthy-weight children[76]. Zerva et al.[13] found an inverse association between eating frequency and percent body fat, as measured by sum of skinfolds, in

children 9-11 years of age. We found no relationship between eating frequency and total body fat or percent body fat attained via DXA, but did see significant associations with VAT attained via MRI. Our analysis of EOs per 24-h as a continuous variable showed a positive correlation between eating frequency and SAT, but this did not remain significant in the ANCOVA analysis. Thus, further research assessing eating frequency and specific adiposity measures utilizing DXA and MRI is warranted, especially in minority youth.

Eating frequency has been found to be positively related to PA[13,44] and some studies have shown this to have a mitigating affect on the relationship between eating frequency and adiposity [44], while others have not[13,15]. All of these studies contained normal weight subjects and previous research has shown that overweight and minority youth are less active than their normal weight counterparts[101]. In this analysis, no relationship between eating frequency and PA measures was found and our sample included only overweight youth, with relatively low levels of PA (7% time spent in MVPA).

There are several potential mechanisms to discuss in relation to meal skipping and increased VAT. Research in adult populations consistently shows that increased eating frequency is not related to increases in resting metabolic rate; however, to our knowledge this has not been assessed in youth populations [53,68]. Eating frequency has been related to satiety measures, in a cross-over controlled feeding study by Leidy et al.[61] with 13 overweight or obese males, less frequent eating (3 EOs) vs. frequent eating (6 EOs) led to

higher satiety throughout the day, but also higher pre-meal hunger ratings. This study controlled for calories and it is unknown what the impact would have been on ad libitum caloric intake, however, increased daily satiety would presumably lead to decreased overall daily calories and increased pre-meal hunger would likely lead to increase calories per EO, thus supporting our energy intake findings. However, more research is warranted to examine the exact mechanism of how eating patterns impact satiety, hunger, and ad libitum dietary intake in free-living youth.

Higher VAT in MS may in part be due to higher insulin concentrations throughout the day, as elevated VAT stores have been associated with hyperinsulinemia[102]. The Leidy study showed that eating less often resulted in a 4% and 20% increase in plasma glucose and insulin, respectively, across the testing period[61]. However, we found no significant differences in fasting insulin or glucose. This may be due to the fact that MS ate significantly less sugar than NFE, which could be due to eating less often and thus snacking less throughout the day. Among adolescents 12-19 years of age snacking has been found to include items high in sugar, and increased snacking has been previously associated with increased sugar and energy consumption in adolescents[81] .Yet, despite this reduced sugar consumption, MS displayed higher triglycerides and AIR, but the AIR measurement is in response to a set glucose load and not reflective of the acute meal effects. Conducting an acute feeding trial to examine how eating frequency, specifically meal skipping, impacts metabolic rates, satiety and gut hormone measures, and insulin action in minority youth is warranted.

Another possible mechanism is that VAT content has consistently been linked to higher fasting triglycerides in adults[103], as well as in youth[104]. Most stored fat is found in the form of triglycerides, and in the present analysis meal skipping was related to increased triglycerides. These elevated triglyceride concentrations may be due to increased caloric intake per EO witnessed in MS, resulting in an increased production and storage of triglycerides and ultimately increased VAT deposition. A previous study by Tanofsky-Kraff et al.[66] with 180 children 5-12 years of age found that increased frequency of binge eating resulted in increased triglycerides and increased VAT over a two year period. In the present analyses MS consumed approximately 130 kcal more per EO than NFE, lending support to the hypothesis that a higher caloric intake per EO may lead to increased triglycerides and VAT. However, further research on the exact mechanism is warranted.

Several limitations of the current study need to be addressed. Previous research has highlighted the possible role of dietary underreporting[83], particularly in an overweight sample. However, we took multiple steps to assess energy intake plausibility and our entire sample was overweight or obese, thus we would expect a similar level of underreporting across this homogenous population. The disproportionately high number of females and Hispanics within this sample may also have been a limitation as differences have been found in fat distribution by sex and ethnicity[105,106] however we did control for sex and ethnicity in all analyses. Hispanics tend to show higher VAT and lower SAT depots than African Americans (39), and males higher VAT than females[106].

However, there were no significant differences in sex or ethnicity between eating frequency groups and in a multiple linear regression analysis sex and ethnicity together explained only 3.4% of the variance in eating frequency. We also stratified the sample by ethnicity. Within the Hispanic sample, MS had higher VAT ($p < 0.05$), and within the African American sample MS showed increased VAT, but significance was not reached. Of note, the sample size of MS within the African American population was quite low (n=11). When stratified by sex, there was a trend for MS and increased VAT in males, whereas this relationship was not significant in females, however the sample size for male and female MS was small (n=12 and n=15). Thus, further research with larger samples within each sex and ethnicity is warranted.

In conclusion, MS consumed 24% fewer calories per 24-h than NFE, yet showed 18% higher triglycerides and 26% higher VAT, while showing no difference in PA measures. Fat distribution is a critical determinant in metabolic disorders associated with obesity, and VAT is a strong indicator of deleterious metabolic profiles, such as dyslipidemia and glucose intolerance [106]. It is foreseeable that higher energy intake per EO in MS led to increased triglycerides which may have potentiated the elevated VAT storage, and further research to investigate this possible mechanism is warranted. Our findings, specifically the association between meal skipping and increased VAT together with recent research that has consistently found an inverse relationship between eating frequency and measures of adiposity in different study populations highlights the need for additional controlled feeding studies on meal skipping and its relationship with adiposity

and metabolic disease risk[13-15]. Given Hispanic and African American youth are at high risk for meal skipping, obesity, and associated metabolic disorders, it is important to identify how meal skipping may be linked to specific adiposity measures in these populations.

Chapter 4: Increased Eating Frequency Linked to Decreased Obesity and Improved Metabolic Outcomes

House BT, Shearrer GE, Miller SJ, Pasch KE, Goran MI, Davis JN. Increased eating frequency linked to decreased obesity and improved metabolic outcomes. *Int J Obes (Lond).* Jan 2015;39(1):136-141. JD and MG designed and supervised the research study used in this analyses; MG obtained the funding; BH, JD, SM, KP, and GS analyzed data. All authors contributed to editing the manuscript. BH wrote the manuscript and BH and JD take responsibility for final content presented.

ABSTRACT

Background: We previously reported that more frequent eating in overweight minority youth was linked to lower visceral adiposity and circulating triglycerides. The aim of this study was to examine this issue in more detail by assessing the relationship between eating frequency and adiposity and metabolic disease risk in a cohort of exclusively overweight Hispanic youth.

Methods: This analysis included 191 overweight ($\geq 85^{th}$ percentile BMI) Hispanic youth (8-18y) with the following cross-sectional measures: height, weight, BMI, dietary intake via multiple 24-h recalls, body composition via dual-energy X-ray absorptiometry, lipids, and insulin action (insulin sensitivity, acute insulin response (AIR) and disposition index (DI), a measure of beta cell function) via a frequently sampled intravenous glucose tolerance test. Each eating occasion (EO) was defined as ≥50 calories and ≥15 minutes

from any prior EO. Infrequent Eaters (IEs) were classified as any subject who ate <3 EOs on any dietary recall (n=32), whereas Frequent Eaters (FEs) always consumed ≥3 EOs (n=159).

Results: Using analyses of covariance, FEs compared to IEs consumed 23% more calories per day ($p \leq 0.01$), ate 40% more often, and consumed 19% less calories per EO ($p \leq 0.01$). FEs also exhibited 9% lower BMI z-scores ($p \leq 0.01$), 9% lower waist circumferences ($p \leq 0.01$), 29% lower fasting insulin ($p=0.02$), 31% lower HOMA-IR values ($p=0.02$), and 19% lower triglycerides ($p \leq 0.01$), as well as an 11% higher AIR ($p=0.02$) and 31% higher DI ($p=0.01$). The following *a priori* covariates were included: Tanner, sex, body fat, and reported energy intake.

Conclusion: These findings suggest that increased eating frequency is related to decreased obesity and metabolic disease risk in overweight Hispanic youth, despite increases in energy intake.

INTRODUCTION

Hispanics are the largest and fastest growing ethnic minority in the United States, representing 17% of the population[3]. Obesity and Type 2 Diabetes (T2D) also disproportionately affect Hispanics. National data collected in Hispanic adolescents (12-19 y) show that 42% are overweight and 23% are obese, compared to 30% and 16%, respectively, in Non-Hispanic Whites (NHW)[4]. Furthermore, our group has shown that

over 30% of Hispanic children and adolescents (8-18 y) have pre-diabetes and the metabolic syndrome[5,6].

Hispanic youth are also more likely to eat less often than NHW[9], and national data suggests that approximately 9% of Hispanic adults compared to 3% of NHW consume ≤2 eating occasions (EOs) per day[10]. Yet, it remains unclear how or if this decreased number of EOs per day affects obesity and metabolic diseases within this high-risk population. Our group has recently shown in a combined sample of overweight African American and Hispanic youth (8-18y) that frequent eating (≥3 EOs per day) compared with infrequent eating (<3 EOs per day) was associated with decreased visceral adiposity and triglycerides, despite being linked to increased reported daily energy intake[1]. The goal of the current study is to expand on our previous research utilizing the same EO definition[30] and examine these relationships in a younger and more diverse tanner staged sample of exclusively overweight Hispanic Youth, while also broadening the scope of our dietary analysis by examining the percentage of calories eaten in the morning, afternoon, and evening based on previous work by Thompson et al.[32].

METHODS

Subjects:

The design, data collection procedures, and findings of the University of Southern California longitudinal SOLAR (Study of Latino Adolescents at Risk for Diabetes) cohort, which began in the year 2000, have been described in detail previously[107,108].

Although cross-sectional dietary analyses have previously been conducted in this cohort[109,110], the present analysis is the first to examine eating frequency and adiposity, as well as metabolic disease risk using this cohort. One hundred and ninety one participants for whom both complete body composition and dietary data were available were included in this analysis. One subject did not have a 2-h oral glucose tolerance test (OGTT) and smaller subsamples had specific metabolic measures from a frequently sampled intravenous glucose tolerance test (FSIVGTT). Inclusion criteria for SOLAR were as follows (i) 8 -18 years of age, (ii) BMI $\geq 85^{th}$ percentile for age and gender based on Center for Disease Control and Prevention guidelines[93], (iii) Latino ancestry (all 4 grandparents of Latino origin as determined by parental self-report), and (iv) family history of T2D in one parent, sibling, or grandparent determined by parental self-report. Participants were excluded for the following reasons: if they were taking any medication known to affect body composition, or if they had been diagnosed with a disease/s or syndrome known to affect body composition or fat distribution. SOLAR was approved by the Institutional Review Board of the University of Southern California. Informed written consent and assent were obtained from both parents and children before testing commenced.

Anthropometrics and Adiposity Measures:

A licensed pediatric healthcare provider performed a detailed physical exam where Tanner staging was determined using established guidelines[94,95]. Height, weight, and waist circumference (at the umbilicus) were measured to the nearest 0.1 cm, 0.1 kg,

and 0.1 cm, respectively. BMI and BMI z-scores were determined by using EPII 2000 software (version 1.1; Centers for Disease Control and Prevention, Atlanta, GA[93]). Whole-body fat and soft lean tissue were measured by dual-energy X-ray absorptiometry (DXA) with the use of a Hologic QDR 4500W (Hologic, Bedford, MA).

Insulin and Glucose Indexes:

After an overnight fast, a 2-h oral-glucose-tolerance test was conducted with a dose of 1.75 g glucose/kg body weight (to a maximum of 75 g). Blood was sampled and assayed for glucose and insulin at –5 min (fasting state) and 120 min (2 h) relative to glucose ingestion. Within 1 month after the OGTT visit, non-diabetic children were asked to come back to the General Clinical Research Center for an overnight visit when a FSIVGTT was performed. At time 0, glucose (25% dextrose, 0.3 g/kg body weight) was administered intravenously, and insulin [0.02 U/kg body wt; Humulin R (regular insulin for human injection); Eli Lilly, Indianapolis, IN] was injected intravenously at 20 min. A total of 13 blood samples were collected. Plasma was collected during the FSIVGTT was measured for glucose and insulin, and values were entered into the MINMOD MILLENIUM 2003 computer program (version 5.16; Richard N Bergman, University of Southern California, Los Angeles, CA) for assessment of insulin sensitivity (SI), acute insulin response (AIR), and disposition index (DI - an index of β cell function).

Assays:

Blood samples from all time points taken during the OGTT and FSIVGTT were centrifuged (10 min, 2500 rpm, 8–10 °C) immediately to obtain plasma, and aliquots

were frozen at –70 °C until assayed. Glucose from the OGTT was analyzed on a Dimension Clinical Chemistry system with the use of an in vitro Hexokinase method (Dade Behring, Deerfield, IL). Glucose from the FSIVGTT was assayed in duplicate on an analyzer (model 2700; Yellow Springs Instrument, Yellow Springs, OH) using the glucose oxidase method. Insulin was assayed in duplicate by using a specific human insulin enzyme-linked immunoabsorbent assay kit (Linco, St Charles, MO). Fasting lipids (n=135) including triglycerides, low-density lipoprotein (LDL)-cholesterol, and high-density lipoprotein (HDL)-cholesterol were assessed using Vitros Chemistry DT Slides (Johnson and Johnson Clinical Diagnostics, Inc., Rochester, NY).

Dietary Intakes:

Dietary intake was assessed from two or three 24-h diet recalls using the multiple-pass technique. A bilingual dietary technician administered one recall in person during the outpatient visit with the use of 3-dimensional food models. The second recall was administered by telephone by the same technician in the week after the visit. All recalls were administered or checked by a trained dietary technician in person or by telephone. Nutritional data was analyzed by using the Nutrition Data System for Research (NDS-R 2010 version 5.0_35). The NDS-R program calculated key dietary variables for this analysis, including mean energy, total fat, protein, carbohydrates, saturated fat, total sugar, added sugar, dietary fiber, soluble fiber, and insoluble fiber. We then calculated the percent caloric intake of total fat, protein, carbohydrates, saturated fat, sugar, and added sugar and the grams of fiber per 1000 kcals. The dietary data was carefully screened for

plausibility. Data was first screened by evaluating the participants' comments and no subjects were excluded; the dietary data was then examined for plausibility by performing a regression of caloric intake on BMI, and one subject was excluded because they had a standardized residual greater than three SD above the mean.

Eating Frequency Analysis:

The following aspects of eating frequency were examined: average number of EOs per 24-h and infrequent eating versus frequent eating. Infrequent Eaters (IEs; n=32) were classified as those subjects who ate less than three times per 24-h on any dietary recall, and Frequent Eaters (FEs; n=159) were classified as those subjects who ate three or more times per 24-h on all dietary recalls. We followed a previously established eating frequency methodology[30] and did not make a distinction between meals and snacks, and instead examined eating frequency based on the number of EOs; each EO had to be at least 50 calories and at least 15 minutes from any previous EO[30]. Also, using methodology first published by Thompson et al.[32] we calculated the eating frequency and the amount of energy consumed in three different time blocks - morning (6:00AM–10:59AM), afternoon (11:00AM–4:59PM), or evening/night (5:00PM–5:59AM).

Statistics:

Data was examined for normality, and transformations were made if the data was found to be significantly different from normal. The following outcome variables were non-normally distributed and were log transformed before the analysis: protein (% kcal), 2-h glucose, fasting insulin, HOMA-IR, AIR, SI, DI, triglycerides, and HDL cholesterol,

but back-transformations are displayed in the text and tables. BMI z-score, carbohydrates (%kcals), and all fiber intakes were unable to be normalized within this population. A t-test or chi square analyses were used to assess differences in age, sex, ethnicity, and tanner stage between eating frequency groups. ANCOVA analyses were used to assess differences in eating pattern and dietary intake variables, adiposity and metabolic parameters between the two eating frequency groups. In all models, the following *a priori* covariates were included: Tanner stage and sex. Mean energy intake was used as a covariate in the analysis of metabolic parameters and adiposity measures. Total fat was included as covariate for all metabolic parameters. Total fat was also included as a covariate for total lean mass and vice versa. Height was included as a covariate for both total fat and total lean mass. SI was included as a covariate for AIR. All analyses were performed by using SPSS version 20.0 (SPSS, Chicago, IL), and the significance was set at $p \leq 0.05$.

RESULTS

The basic demographic data and adiposity measures are presented in **TABLE 4.1.** There were 191 overweight participants that had complete anthropometric, dietary, and body composition data. Metabolic outcomes were available in smaller samples. The sample was 57% male and averaged 13.5 years of age.

TABLE 4.1

Subject Characteristics [1,2]

Physical and Adiposity Measures (n=191)	
Sex M/F	108/83
Age (y)	13.5± 1.9
Tanner stage	3.3± 1.3
Height (cm)	159.9± 10.9
Weight (kg)	78.6± 20.5
BMI (kg/m^2)	30.3± 5.5
BMI z score	2.0± 0.5
Waist circumference (cm)	92.1± 12.3
Total fat (kg)	29.0± 10.4
Total lean tissue mass (kg)	46.2± 11.7
Total body fat (%)	37.1± 6.7
Metabolic Parameters from OGTT (n=190)	
Fasting glucose (mg/dL)	91.0± 7.3
2-h glucose (mg/dL)	124.1± 19.7
Metabolic Parameters from FSIVGTT (n=169)	
Fasting glucose (mg/dL)	90.7± 7.5
Fasting insulin (μU/mL)	13.7± 7.7
HOMA-IR	3.1± 1.8
SI [x10-4 min-1/(μU/mL)][3]	1.8± 1.0
AIR (μU/mL X 10 min)[3]	1,466.5± 834.2
DI (x10-4/min-1)[3]	2,113.8± 1,013.6
Total cholesterol (mg/dL)[4]	148.2± 25.7
Triglyceride (mg/dL)[4]	112.0± 55.3
LDL-cholesterol (mg/dL)[4]	88.1± 22.0
HDL-cholesterol (mg/dL)[4]	37.6± 9.6

[1] Data presented as mean ± SD

[2] AIR - Acute Insulin Response, DI – Disposition Index, FSIVGTT – Frequently Sampled Intravenous Glucose Tolerance Test, HOMA-IR - Homeostatic Model Assessment: Insulin Resistance, HDL - High Density Lipoprotein, LDL - Low Density Lipoprotein, OGTT – Oral Glucose Tolerance Test, SI – Insulin Sensitivity.

[3] n = 128, [4]n = 135

TABLE 4.2 presents dietary data. The average number of EO per 24-h was 4.0, and 45% of the daily calories were consumed between the hours of 11:00AM and 4:59PM.

TABLE 4.2
Behavioral Characteristics [1]

Dietary Variables (n=191)	
Eating occasions per day	4.0± 1.0
Energy per eating occasion (kcal)	465.3± 153.8
Energy (kcal)	1,792.6± 582.0
Eating occasions in the morning	0.9± 0.5
% energy consumed in the morning	19.8± 14.0
Eating occasions in the afternoon	1.7± 0.7
% energy consumed in the afternoon	45.2± 18.8
Eating occasions in the evening/at night	1.4± 0.8
% energy consumed in the evening/at night	35.2± 18.5
Total fat (g/day)	65.5± 26.5
Total fat (%kcal)	31.6± 6.1
Total protein (g/day)	68.4± 24.2
Total protein (%kcal)	15.9± 4.6
Total carbohydrate (g/day)	238.1± 81.8
Total carbohydrate (%kcal)	52.5± 7.7
Total saturated fat (g/day)	23.4± 9.7
Total saturated fat (%kcal)	10.9± 2.5
Added sugars (g/day)	66.7± 42.7
Added sugars (%kcal)	14.5± 6.9
Total sugars (g/day)	108.6± 49.8
Total sugars (%kcal)	24.1± 7.2
Dietary fiber (g/day)	14.6± 6.2
Dietary fiber (g/1000kcal)	8.4± 3.2
Insoluble fiber (g/day)	9.7± 4.5
Insoluble fiber (g/1000kcal)	5.6± 2.4
Soluble fiber (g/day)	4.2± 2.3
Soluble fiber (g/1000kcal)	2.7± 1.2

[1] Data presented as mean ± SD

TABLE 4.3 presents adiposity and metabolic measures by the two eating frequency groups. We found no significant difference in sex or weight between eating frequency groups, however, we did find significant differences in age and Tanner staging. Infrequent eaters tended to be older and higher in pubertal status. Independent of covariates, FEs showed a 9% lower BMI z-scores (p=0.01), a 9% lower waist circumference (p≤0.01), and a 17% lower amount of lean tissue (p=0.05) compared with IEs, as well as a significantly lower BMI z-score (p≤0.01) There were no other significant differences in adiposity measures between the two groups. There were also significant differences in metabolic outcomes, FEs compared to IEs had a 29% lower fasting insulin value (p=0.02), a 31% lower HOMA-IR value (p=0.01), an 11% higher acute insulin response (p=0.02), a 31% higher disposition index (p≤0.01), and 19% lower triglycerides (p≤0.01), all of which indicate that FEs had a healthier metabolic profile than IEs.

TABLE 4.3

Adiposity measures and metabolic parameters between eating frequency groups[1,2,3]

	Infrequent Eaters (n=32)	Frequent Eaters (n=159)	p value
Physical and Adiposity Measures (n=191)			
Sex M/F	23/9	85/74	0.06
Age (y)	14.4± 1.7	13.3± 1.9	≤0.01
Tanner stage	4.0± 1.2	3.2± 1.3	0.02
Weight (kg)	90.8± 8.0	90.0± 7.3	0.09
BMI z score	2.2± 0.3	2.0± 0.5	≤0.01
Waist circumference (cm)	99.6± 10.7	90.6± 12.1	≤0.01
Total fat (kg)	33.1± 10.5	28.2± 10.3	0.61
Total lean tissue mass (kg)	54.0± 9.9	44.6± 11.4	0.05
Total body fat (%)	36.6± 7.9	37.2± 6.4	0.25
Metabolic Parameters from OGTT (n=190)			
Fasting glucose (mg/dL)	93.3± 9.0	90.5± 6.9	0.44
2-h Glucose (mg/dL)	126.3± 18.5	123.7± 20.0	0.50
Metabolic Parameters from FSIVGTT (n=169)			
Fasting glucose (mg/dL)	94.0± 7.9	90.0± 7.3	0.20
Fasting insulin (μU/mL)	18.0± 10.2	12.7± 6.8	0.02
HOMA-IR	4.2± 2.5	2.9± 1.6	0.01
SI [x10-4 min-1/(μU/mL)][4]	1.5± 1.2	1.8± 1.0	0.78
AIR (μU/mL X 10 min)[4]	1,304.1± 902.6	1,466.5± 819.2	0.02
DI (x10-4/min-1)[5]	1,463.7± 830.4	2,127.0± 938.9	0.01
Total cholesterol (mg/dL)[6]	144.7± 20.3	149.0± 26.8	0.72
Triglycerides (mg/dL)[6]	132.3± 51.7	107.2± 55.3	≤0.01
LDL-cholesterol (mg/dL)[6]	84.1± 14.9	89.1± 23.3	0.75
HDL-cholesterol (mg/dL)[6]	34.1± 6.7	38.5± 10.0	0.28

[1] Data presented as mean ± SD

[2]AIR - Acute Insulin Response, DI - Disposition Index, FSIVGTT – Frequently Sampled Intravenous Glucose Tolerance Test, HOMA-IR - Homeostatic Model Assessment: Insulin Resistance, HDL - High Density Lipoprotein, LDL - Low Density Lipoprotein, OGTT – Oral Glucose Tolerance Test, SI - Insulin Sensitivity.

[3] A t-test (for continuous variables) and chi-square analysis (for categorical variables) assessed differences in age, sex, and Tanner stage between groups.

[4]ANCOVA analysis of adiposity measures and metabolic parameters between infrequent eaters and normal/frequent eaters. *A priori* covariates included: tanner stage, sex, mean energy (for adiposity and metabolic parameters), total fat (for metabolic parameters), total fat and height (for total lean), total lean and height (for total fat), and SI (for AIR).

[4] n = 128, [5] n = 124 (four outliers were omitted), [6] n = 135

Dietary variables between IEs and FEs are depicted in **TABLE 4.4** FEs ate 40% more often ($p \leq 0.01$) and ate 19% less per EO ($p \leq 0.01$), while consuming 23% or on

average 431 more calories per day ($p \leq 0.01$) than IEs. There were no significant differences in macronutrients, fiber, or sugar intake between the two groups. Additionally, FEs had significantly more EOs than IEs in the morning, afternoon, and evening; however, the percentage of calories consumed during these time periods was not significantly different between the two groups.

TABLE 4.4

Dietary characteristics between eating frequency groups[1,2]

	Infrequent Eaters (n=32)	Frequent Eaters (n=159)	p value
Dietary Variables (n=191)			
Eating occasions per day	2.6± 0.6	4.3± 0.8	≤0.01
Energy per eating occasion (kcal)	553.9± 177.6	447.4± 142.7	≤0.01
Energy (kcal/day)	1,434.0± 523.8	1,864.8± 567.6	≤0.01
Eating occasions in the morning	0.6± 0.5	0.9± 0.5	≤0.01
% energy consumed in the morning	19.5± 20.9	19.9± 12.3	0.88
Eating occasions in the afternoon	1.1± 0.5	1.8± 0.7	≤0.01
% energy consumed in the afternoon	45.3± 20.6	45.2± 18.4	0.97
Eating occasions in the evening/at night	0.9± 0.4	1.4± 0.6	≤0.01
% energy consumed in the evening/at night	37.5± 20.6	34.7± 18.1	0.43
Total fat (%kcal)	32.4± 6.9	31.4± 5.9	0.44
Total protein (%kcal)	17.1± 5.4	15.6± 4.4	0.16
Total carbohydrates (%kcal)	50.4± 9.3	52.9± 7.4	0.14
Total saturated fat (%kcal)	11.5± 2.9	10.7± 2.4	0.15
Total sugars (%kcal)	21.9± 8.1	24.6± 7.0	0.07
Added sugars (%kcal)	13.2± 7.1	14.7± 6.9	0.15
Dietary fiber (g/1000kcal)	8.5± 3.9	8.4± 3.1	0.76
Insoluble fiber (g/1000kcal)	5.7± 2.9	5.6± 2.3	0.65
Soluble fiber (g/1000kcal)	2.7± 1.5	2.7± 1.1	0.97

[1]Data presented as mean ± SD

[2]ANCOVA analysis of dietary variables between infrequent eaters and normal eaters. *A priori* covariates used: tanner stage and sex.

DISCUSSION

To our knowledge, this is the first analysis to examine the relationship between eating frequency and dietary, metabolic, and adiposity measures in a sample of exclusively overweight Hispanic youth, and the first within this population to calculate the eating occasions and percentage of calories consumed in the morning, afternoon, and evening. Hispanic youth have higher rates of obesity than NHW[4] and are also at increased risk of T2D and cardiovascular disease[108,111,112]. Our previous work has shown that frequent eating (≥3 EOs per day) in minority youth is related to decreased visceral fat and triglycerides, despite an increased caloric intake[1]. In the present analysis, we replicated and expanded on these results in a larger and younger cohort of exclusively Hispanic youth and found that FEs compared to IEs have lower BMIs, waist circumferences, fasting insulin values, insulin resistance, triglycerides, and higher insulin responses and beta cell functioning, despite reporting consuming more calories per day.

Numerous epidemiology studies have shown that increased eating frequency is linked to decreased obesity rates[14,15,74,76,113], while some studies have shown no or opposite relationships[114-116]. A recent longitudinal study by Ritchie et al.[15], with 2,372 African American and Caucasian girls (9-19 y) found that lower meal frequency was related to greater increases in BMI and waist circumference over a ten year period, independent of socioeconomic status, total energy intake, and physical activity levels. Other studies have found increased eating frequency to be inversely related to waist circumferences

[15,60,76], body fat percent as measured by skinfolds[13], and fasting glucose, insulin and lipids [60]. The current findings are similar to this and demonstrate that eating frequency is associated with lower BMI parameters and waist circumference, however we did not see an effect on body composition as measured via DXA.

To date, our group is the first to examine the relationship between eating frequency and specific metabolic outcomes attained from an FSIVGTT. In the current analysis we found sizable differences in insulin action, with FEs having lower fasting insulin values, insulin resistance, and more robust beta cell function, elucidating that FEs have a reduced risk of T2D and other metabolic disorders. A cross-sectional analysis performed by Smith et al.[60] in a mixed population of 2,775 young adults (26-36 y) found increased eating frequency to be negatively associated with fasting glucose and insulin, triglycerides, total cholesterol, and LDL cholesterol. However, these findings were only significant in men, and no explanation of exclusion factors was included. In a crossover controlled feeding trial by Leidy et al.[61] with 13 overweight or obese males, frequent eating (6 EOs) vs. less frequent eating (3 EOs) resulted in a 4% decrease in fasting glucose and a 20% decrease in fasting insulin, across the 11-h testing period. However, overall there is a paucity of data linking eating frequency to specific metabolic disease markers and more research is warranted, specifically in high-risk populations, such as Hispanic youth.

There are numerous potential mechanisms to explain our findings. Several experimental studies in which the eating frequency groups were designed to be eucaloric (i.e., calories to support weight maintenance) or isocaloric (i.e., matched on caloric

intake), found that frequent eating was linked to decreases in hunger[63,117] and increased satiety responses[52]. However, the previous study by Leidy et al.[61] found increased EOs led to higher satiety throughout the day, but also higher pre-meal hunger ratings[61]. Thus, in a free-living environment it is foreseeable that more EOs may lead to more total calories throughout the day, but possibly less calories consumed per eating occasion. National adult studies support this finding and have shown that increased EOs results in increased daily quantity of foods/beverages consumed, as well as daily energy density (kcal/g) of all foods and beverages reported[118]. One national adult study[10] showed increased daily eating frequency to be associated with increased daily energy intake, where participants who consumed ≥5 EOs per day consumed approximately 800 kcals/d more than those who consumed ≤2 EOs per day. In free-living populations, research consistently shows a positive relationship between eating frequency and caloric intake, yet also is more regularly inversely related to obesity and adiposity measures, thus challenging the energy balance theory. In the present analysis, FEs ate more often than IEs throughout the day including, morning, afternoon and evening, revealing that the current finding is not reflective of IEs skipping any one meal. This data provides evidence that the particular meal skipped may be less relevant to adiposity than eating less than three EOs per day. Therefore, analyses that only look at skipping any one particular meal, such as breakfast, may not be gleaning the entire story of how eating patterns relate to health outcomes. Given the irregular eating patterns of Hispanic youth, more research is warranted examining how the number of eating occasions per day

affects energy intake, satiety/hunger measures, and subsequent adiposity and metabolic parameters.

It is likely that eating frequency impacts obesity and related metabolic diseases through a combination of different mechanisms. For decades, the public and media outlets have advertised and promoted more frequent EOs as a means to increase one's metabolism and optimize weight loss. Yet, research conducted in whole-room calorimetry chambers has consistently shown that eating frequency is not associated with changes in the thermic effect of food, basal metabolic rates, or 24-hour energy expenditure[53,68]. However, in the present study, we did find numerous relationships between eating frequency and metabolic outcomes. The current analysis showed that eating more often is linked to lower fasting insulin values, as well as to decreased insulin resistance, higher insulin secretion rates, and improved β-cell functioning. Elevated HOMA-IR values, fasting insulin, and insulin resistance have been shown to elevate obesity risk in youth populations[119], but further research in this area is needed.

Another possible mechanism involves lipid metabolism. Our study found that FEs had lower circulating triglycerides than IEs. Infrequent Eaters showed an increased caloric intake per EO, and binge eating behaviors have been previously linked to increased triglycerides[66]. It is also possible that FEs have less visceral fat. The accumulation of visceral fat has been positively associated with fasting insulin and triglycerides[1,120]. It is also hypothesized that visceral fat increases hepatic portal free fatty acid concentrations, which in turn are stored as triglycerides, stimulate hepatic

gluconeogenesis, and hinder hepatic clearance of insulin, thus promoting a vicious cycle of hyperinsulinemia, elevated plasma glucose concentrations, and dyslipidemia[121]. We did not have enough subjects with magnetic resonance imaging data to include visceral fat as a measure within this analysis, but did see both reduced visceral fat and triglycerides among FEs in our previous analysis of minority youth[1]. More research, especially randomized controlled feeding trials analyzing the possible relationship between visceral adiposity, triglycerides, eating frequency, and metabolic disease risk is merited.

There are several limitations to consider in the present study. Eating frequency has been found to be positively related to physical activity[13,44], and some studies have shown this to have a mitigating effect on the relationship between eating frequency and adiposity[44], while others have not[13,15]. The SOLAR study did not collect physical activity data; however, our previous work did not find an association between eating frequency and physical activity (measured by accelerometry) in overweight minority youth. Another limitation is possible underreporting, especially by overweight/obese participants[122,123]. However, this population is very homogenous, with 79% being obese, and thus underreporting would be expected to be consistent throughout [83]. Furthermore, we used multiple exclusion criteria to assess implausible reporters, including subject comments, as well as running a regression with caloric intake and BMI z-scores and excluding subjects who were greater or less than three SD from the mean. SOLAR is a longitudinal trial, yet the number of participants with complete dietary and body composition data at multiple

time points was too low to conduct mixed modeling, thus driving us to the current cross-sectional analysis. This sample also had a relatively small number of IEs, yet the percentage of the sample is consistent with our previous work[1] and others[124]. Due to the cross-sectional nature of this data set inferring a causal pathway is difficult. However these results replicate our previous findings and because this is a youth population, we expect that the eating frequency patterns are in fact playing a role in promoting obesity and metabolic diseases. These results highlight the need for more intervention work in this area to truly assess causality.

In summary, eating three or more times per day is associated with healthier outcomes for obesity and metabolic risk, despite being linked to increased reported daily energy intake. Given that Hispanic youth are at such a high risk of obesity and associated metabolic disorders, it is important to identify nutrition behaviors that may potentially reduce this risk. These results as well as our group's and other's previous findings support that intervention work is needed to investigate the potential causal mechanism of how eating frequency affects obesity and metabolic disease risk, particularly in Hispanic youth.

Chapter 5: Increased Eating Frequency and Breakfast Consumption linked to Increased Physical Activity and Reduced Sugar Sweetened Beverage Consumption in Hispanic College Freshmen

ABSTRACT

Background: There is little research on the association between eating frequency, breakfast consumption and obesity parameters available in college freshmen. This is an important gap, as behavioral choices formed during the transitional period are likely to continue throughout adulthood and may increase chronic disease risk later in life. Within this population, Hispanics represent an understudied population particularly susceptible to obesity and metabolic diseases. **Methods:** A cross-sectional sample of Hispanic college freshmen (n=709, 18-19 y, 56% female, 31% overweight or obese) from the University of Texas at Austin with self-reported height and weight and a variety of dietary and physical activity behaviors including: eating frequency, breakfast consumption, sugar sweetened beverage, fast food intake, moderate to vigorous physical activity, and screen time via a questionnaire. T-tests, chi square analysis, and logistic regressions were used to examine how eating frequency and breakfast consumption were associated with dietary behaviors, physical activity, and BMI parameters.

Results: No difference in obesity risk was observed among breakfast or eating frequency groups. However, participants who consumed ≥4 eating occasions (EOs) per day compared to those who consumed ≤2 EOs had significantly greater odds of accumulating ≥2.5 hours of moderate to vigorous physical activity (MVPA) per wk (AOR: 2.6, CI: 1.6-

4.1 $p \leq 0.01$) and of never drinking soda (AOR: 1.9, CI: 1.2-3.1, $p \leq 0.01$). Similarly, those who consumed breakfast on 6 or 7 days compared to those who consumed breakfast on 0 or 1 day had substantially greater odds of accumulating ≥2.5 hours of MVPA per wk (AOR: 2.3, CI: 1.3-4.2, $p \leq 0.01$) and of never drinking soda (AOR: 3.3, CI: 1.7-6.7, $p \leq 0.01$).

Conclusions: These findings suggest that increased breakfast consumption and eating frequency are related to healthy dietary and physical activity behaviors but not obesity risk in Hispanic college

INTRODUCTION

In 2012, for the first time in history, Hispanic high school graduates (69%) were more likely to be enrolled in college than Non-Hispanic Whites (NHW; 67%) and Non-Hispanic Blacks (NHB; 63%)[18]. In 2013, Hispanic students represented 23% of freshman enrollment at the University of Texas (UT) at Austin, showing the largest increase among all minority groups[19]. Currently, 67% of Hispanics, 12-19 years of age, are either overweight or obese[125], yet little is known about the behavioral correlates of weight status in Hispanic college students. Several studies have shown that the initial transition to college is associated with rapid weight gain, with the average weight gain in the first year of college ranging from 3.5 to 8.8 pounds [27]. A growing body of research has identified correlates of increased obesity among primarily NHW students, including decreased dietary fiber, fruits, vegetables[26,126], and increased junk food consumption[127]. Further, this

period has been identified as a critical period contributing to the rise in obesity rates as the behavioral choices college students make likely affect their risk of chronic disease later in life [20]. However, there is no comparable research investigating the associations between eating patterns and obesity risk in a population of exclusively Hispanic college freshmen.

Eating frequency research has consistently found an inverse association between the number of eating occasions (EOs) per day and adiposity [13-15], as well as metabolic disease risk in both youth and adult populations [2,16]. Additionally, minority youth have been found to eat less often [9] and infrequent eating has been linked to increased obesity measures in multiple populations of overweight Hispanic youth [1,2]. Breakfast research has also consistently found an inverse association between regular breakfast consumption and obesity [128-131] and regular breakfast consumption has been recognized as a possible means to avert overweight or obesity [132]. Furthermore, breakfast skipping has been found to be prevalent in Hispanic youth populations [133].

Thus, the overall goal of this study is to examine the relationship between eating frequency, breakfast consumption, overweight/obese prevalence, dietary behaviors, and physical activity in a population of Hispanic college freshmen. We hypothesize that increased eating frequency and regular breakfast consumption will be inversely related to obesity status and unhealthy dietary behaviors, and positively related to increased physical activity. These findings can potentially inform public health interventions during this critical time period in an understudied and potentially at risk population.

METHODS

Data Collection:

Data was collected from the 2014-2015 freshmen class at UT-Austin. Hispanic college freshmen were recruited via announcements in classes, word of mouth, electronic posted notices, and tabling at dorms around the UT-Austin campus. Each participant filled out a 21-item screener. The screener included basic demographic, dietary and physical activity questions which were derived from the 2009-2010 National Health and Nutrition Examination Survey (NHANES) dietary screener [134], and from Project EAT 2010 [135-137]. **Table 5.1** displays the relevant behavior questions and response options on the screener. Inclusion criteria for the study were as follows: (i) self-reported Hispanic origin (ii) 18-19 years of age, and (iii) in their first year of college. Students were only excluded if they did not complete the entire screener (n=62). This study was approved by the Institutional Review Board of the UT-Austin. Informed verbal consent was obtained before screeners were distributed.

Table 5.1: List of relevant dietary and physical activity questions on screener	
Questions	Response Options
During the past mo, how often did you drink 100% pure fruit juice, such as orange, mango, apple, grape and pineapple juices? Do not include fruit-flavored drinks with added sugar or fruit juice you made at home and added sugar to.	• Never • 1 time per mos • 2-3 times per mos • 1 time per wk • 2-3 times per wk • 4-6 times per wk • 1 time per day • 2 times per day • ≥ 3 times per day
During the past mo, how often did you drink regular soda or pop that contains sugar? Do not include diet soda.	
During the past mo, how often did you drink **sweetened** fruit, sports or energy drinks such as Kool-aid, lemonade, Hi-C, cranberry drink, Gatorade, Red Bull, or Vitamin Water? Include fruit juices you made at home and added sugar to. Do not include diet drinks or artificially flavored drinks.	
During the past mo, how often did you drink coffee or tea that had **sugar or honey added** to it? Include coffee and tea you sweetened yourself and presweetened tea and coffee drinks such as Arizona Iced Tea and Frappuccino. Do not include **artificially** flavors coffee or diet tea.	
In the past wk, how many days did you eat breakfast?	• Never • 1 day • 2 days • 3 days • 4 days • 5 days • 6 days • Every day
In the past wk, how often did you eat something from a fast food restaurant? (like McDonalds, Pizza Hut, etc.)	• Never • 1-2 times • 3-4 times • 4-5 times • 6 times • ≥ 7 times
On average, how many eating occasions do you consume per day (an eating occasion being defined as any time you eat or drink something greater than 50 calories and more than 15 minutes from any prior eating occasion)?	• Never • 1 • 2 • 3 • 4 • 5 • 6 or more
On average, how many eating occasions did you consume when you were in high school (an eating occasion being defined as any time you eat or drink something greater than 50 calories and more than 15 minutes from any prior eating occasion)?	
In a usual wk, how many hours did you spend doing the following activities? Strenuous physical activity (heart beats rapidly). Examples: biking, aerobics, dancing, running, basketball, tennis, swimming laps, soccer.	• None • < ½ hr per wk • ½ -2 hrs per wk • 2 ½ -4 hrs per wk • 4 ½ -6 hrs per wk • > 6 hrs per wk
In a usual wk, how many hours did you spend doing the following activities? Moderate physical activity (not exhausting). Examples: walking quickly, baseball, easy biking, volleyball, skateboarding.	
In a usual wk, how many hours did you spend doing the following activities? Mild physical activity (little effort). Examples: walking slowly (to school, to friend's house, etc.), bowling, golfing, fishing, yoga.	
On an average day, how many hours do you spend watching television, playing video games, talking/texting on the phone, or using social media Facebook/Twitter etc?	• None • < 1 hr/day • 1-2 hrs/day • 2-3 hrs/day • 3-4 hrs/day • 4-5 hrs/day • > 5 hrs per day

Anthropometrics:

Height and weight was self-reported in all subjects n=709 and measured in a subsample of participants (n=100) to the nearest 0.1 kg and 0.1 cm via a beam medical scale and a wall-mounted stadiometer, respectively, and the average of two measurements was used for the analysis. BMI percentile and BMI z-scores were determined by using EPII 2000 software (version 1.1; Centers for Disease Control and Prevention, Atlanta, GA) [93]. Subjects were categorized as overweight if they had a BMI of 25.0 to < 30.0 and obese if they had a BMI ≥30.0. Pearson correlations were run to assess the relationship of self-reported BMI to measured data. Coefficients can be interpreted as follows: 0.1-0.3 weak, 0.3 -0.5 moderate, > 0.5 strong [138]. The Pearson correlation between self-reported BMI and measured BMI was 0.9 indicating a very strong relationship. Informed written consent was obtained before measured height and weight were obtained.

Dietary Behaviors:

Dietary behaviors were self-reported for all subjects. Eating frequency was divided into three ordinal categories, those with 2 or less EOs per days (n=193), 3 EOs per day (n=258), or 4 more EOs per day (n=258). Breakfast consumption was also categorized into three groups including, Breakfast Skippers who consumed breakfast on zero or one day per week (n=99), Intermittent Breakfast Consumers who consumed

breakfast on 2 to 5 days per week (n=375), and Regular Breakfast Consumers who consumed breakfast on 6 or more days per week (n=235).

In a subsample of subjects (n=148), three or more 24-h diet recalls using the multiple-pass technique were collected to assess dietary intake and were compared to the dietary intake obtained via the screener. All subjects had at least three recalls (one weekend and two weekdays). All recalls were collected by a trained staff member, supervised and trained by a Registered Dietitian. Trained staff checked all recalls for completeness, consistency and accuracy. Nutritional data was analyzed by using the Nutrition Data System for Research (NDS-R 2014). The Siega-Riz breakfast definition [77,139] of any food or beverage consumed between 0500 and 1000 hours with a combined total energy ≥100 calories was used to define breakfast. Seventy-four percent of breakfast skippers on the screener reported either never eating breakfast or eating breakfast on one day of the dietary recalls. Conversely, 84% of those who were regular breakfast consumers on the screener, reported eating breakfast on at least two of their diet recalls. To classify eating frequency, the Gibney et al. definition was used to characterize an eating occasion (EO) as ≥50 calories and ≥15 minutes from any previous EO. Fifty nine percent of participants who reported < 3 EO per day on the screener, reported < 3 EO per day on their dietary recalls [30]. Agreement between the two measures of breakfast frequency was modest, at 41% concordance. Likewise, there was a modest 39% concordance between the screener and the 24-hour recall data on frequency of eating occasions. We further stratified by time between recall administration and screener

collection and there was no significant difference in concordance for both breakfast and eating frequency if dietary recalls were obtained within one week of screener collection or after. In both cases, we decided to utilize the screener data as a more valid measure of usual breakfast frequency and usual eating frequency. Verbal consent was obtained before dietary recalls were administered.

Dietary intake:

Dietary intake measures of interest derived from the screener included fast food intake, frequency of consumption of soda, fruit juice, and sweetened juice respectively. Four category ordinal measures of each of these variables were constructed for preliminary examination. Further to preliminary analysis, binary dietary intake measures were constructed for use in logistic regression: never eating fast food, never drinking soda, never drinking fruit juice and never drinking sweetened juice.

Physical Activity:

Physical activity behaviors were self-reported for all subjects (n=709) and moderate to vigorous physical activity (MVPA) was calculated by collapsing both moderate and vigorous physical activity together. This collapsed MVPA category was then used to assess if subjects met the American Heart Association's (AHA) physical activity recommendation of at least 30 min of either MPA or VPA 5 days per week or 2.5 hours per week [140].

A subsample of participants (n=87) wore accelerometers (wGT3X-BT, Actigraph, LLC., Pensacola, FL) to assess physical activity levels, which were compared to physical activity levels reported on the screener. Subjects were instructed to wear the accelerometers for 7 days and upon receipt data was immediately downloaded and wear time was assessed. Subjects with at least 3 days of 8 h or more of wear time were included in the analyses and one subject needed to be reissued the accelerometer to acquire the required wear time. Eighty-seven subjects had valid data, with accelerometer wear time averaging 12.9 ± 1.6 hours/day for 6.2 ± 1.5 days. Freedson adult cut offs were used to determine sedentary, light and MVPA [141]. Time spent in MVPA was then used to assess whether subjects met AHA physical activity recommendation. Interestingly, 88% of those who said they did not meet the recommendations on the screener actually met the recommendations when wearing an accelerometer highlighting that these subjects may actually be underestimating MVPA. Light physical activity was also extremely under-reported given that subjects reported averaging less than 30 minutes of light physical activity per day, but those with accelerometer data averaged over 100 minutes per day. Activity measures were specified both as ordinal measures of and binary measures, depending on the purpose of analysis. For preliminary data examination, four-category ordinal measures were used to describe light, moderate and vigorous physical activity, as well as screen time. Summary binary measures of physical activity constructed for the purpose of regression analysis included: meeting AHA physical

activity recommendation, and accumulating less than 2-h per day of screen time. Informed written consent was obtained before accelerometers were distributed.

Statistics:

For preliminary analyses, ANOVAs and chi-square tests were used to assess differences in distribution of dietary intake, physical and sedentary activity, overweight/obesity prevalence, and BMI measures across eating frequency and breakfast consumption groups. For these analyses, ordinal specifications of diet and activity behaviors were utilized (**Tables 3 and 4**). Chi-square statistics from the preliminary analysis that were found to be significant were further extended as adjusted logistic regressions with binary specifications of dependent variables (**Tables 5 and 6**). For the logistic regressions, eating frequency and breakfast consumption categories (separately) were the independent variables with the "unhealthy" group being the referent category (2 or less EOs per day and breakfast skippers). For ease of interpretation only three significant binary logistic regressions for eating frequency and breakfast consumption groups were then included in Tables 5 and 6. Bivariate dependent variables included the percent of those meeting AHA physical activity recommendation, never drinking soda, never eating fast food, and accumulating less than 2-h per day of screen time. Covariates included age, sex, and semester of screener collection. All analyses were performed using SPSS version 22 (SPSS, Chicago, IL), and the significance was set at $p \leq 0.05$.

RESULTS

Demographic data, adiposity measures, and dietary and physical activity behaviors of the entire sample (n=709) that completed the screener are presented in **Table 5.2** The sample was 56% female, and 30.7% were overweight or obese.

Table 5.2: Demographics, Adiposity, Dietary and Physical Activity Behaviors of Subjects (n=709)	
Sex	
Male	312(44.0)
Female	397 (56.0)
Age (y)	18.7 ±0.4
Semester of data collection(% - Fall/Spring/Summer)	
Fall	396 (55.8)
Spring	204 (28.8)
Summer	109 (15.4)
Height (cm)	167.7 ±10.0
Weight (kg)	66.8 ±14.4
BMI (kg/m^2)	23.7 ±4.3
BMI z score	0.3 ±1.0
BMI Percentile (%)	58.0 ±4.3
Overweight (%)	161 (22.7)
Obese (%)	57 (8.0)
Overweight/Obese Prevalence (%)	218 (30.7)
Eating Occasions	
≤2 EOs	193 (27.2)
3 Eos	258 (36.4)
≥4 Eos	258 (36.4)
Eating Occasions - High School	
≤2 EOs per day	126 (17.5)
3 EOs per day	254 (35.8)
≥4 EOs per day	329 (46.4)
Breakfast Consumption	
Breakfast Skippers	99 (14.0)
Intermittent Breakfast Consumers	375 (52.9)
Regular Breakfast Consumers	235 (33.1)
Fast Food Consumption (visits per wk)	
Never	122 (17.2)
1-2 visits	353 (49.2)
3-4 visits	147 (20.5)
More than 4 visits	86 (12.0)
Soda (servings)	
Never	152 (21.2)
1-3 times per mo	250 (34.8)
1-3 times per wk	211 (29.4)
4 or more times per wk	96 (13.4)
Fruit Juice (servings)	
Never	111 (15.5)
1-3 times per mo	283 (39.4)
1-3 times per wk	225 (31.3)
4 or more times per wk	90 (12.5)

Table 5.2 Continued	
Sweetened Juice (servings)	
Never	90 (12.5)
1-3 per mo	210 (29.2)
1-3 per wk	274 (38.2)
4 or more per wk	135 (18.8)
Vigorous Physical Activity	
Less than ½ hr per wk	23.8 (171)
½ to 2 hrs per wk	24.7 (177)
2½ to 4 hrs per wk	18.9 (136)
More than 4 hrs per wk	31.3 (225)
Moderate Physical Activity	
Less than ½ hr per wk	108 (15.3)
½ to 2 hrs per wk	224 (31.2)
2½ to 4 hrs per wk	169 (23.5)
More than 4 hrs per wk	207 (28.8)
Light Physical Activity	
Less than ½ hr per wk	65 (9.1)
½ to 2 hrs per wk	512 (29.5)
2½ to 4 hrs per wk	155 (21.6)
More than 4 hrs per wk	277 (38.6)
Meeting AHA Recommendation	565 (78.7)
Screen-time	
Less than 2 hrs per day	282 (39.3)
2-3 hrs per day	172 (24.0)
3-4 hrs per day	133 (18.5)
More than 4 hrs per day	122 (17.0)
Data are mean ±SD or n (%)	

Table 5.3 presents the differences in demographics, obesity parameters, and dietary and physical activity behaviors between the three eating frequency groups. Chi-square analysis found significant differences in semester, soda intake, vigorous and light physical activity, and fast food intake between eating frequency groups. Participants consumed more EOs in the fall compared to the spring and summer semesters. Participants who consumed 4 or more EO per day compared to those who consumed less than 2 EO per day were more likely to accumulate 4 or more hours per week of vigorous physical activity and never drink soda. No significant differences in sex, age, BMI, or weight status groups were observed between eating frequency groups.

Table 5.3: Demographics, obesity parameters, dietary intake and physical activity behaviors by eating frequency groups

	≤ 2 EOs Per Day (n=193)	**3 EOs Per Day (n=258)**	**≥4 EOs Per Day (n=258)**	**P-value**
Age (y)	18.7 ±0.4	18.7 ±0.4	18.7 ±0.4	0.86
Semester				**0.03**
Fall	94 (23.7)	143 (36.1)	159 (40.2)	
Spring	60 (29.4)	72 (35.3)	72 (35.3)	
Summer	39 (35.8)	43 (39.4)	27 (24.8)	
Sex				0.93
Male	87 (27.9)	113 (36.2)	112 (35.9)	
Female	106 (26.7)	145 (36.5)	146 (36.8)	
BMI	23.6 ±4.5	23.9 ±4.2	23.5 ±4.3	0.61
BMI percentile	57.0 ±29.5	59.8 ±28.2	56.9 ±27.5	0.42
Weight Status				0.75
Underweight	12 (35.3)	12 (35.3)	10 (29.4)	
Normal weight	123 (26.9)	160 (35.0)	174 (38.1)	
Overweight	44 (27.3)	61 (37.9)	56 (34.8)	
Obese	14 (24.6)	25 (43.9)	18 (31.6)	
Fast food				**0.02**
Never	46 (37.7)	33 (27.0)	43 (35.2)	
1-2 visits per wk	81 (22.9)	156 (41.4)	126 (35.7)	
3-4 visits per wk	40 (27.2)	54 (36.7)	53 (36.1)	
>4 visits per wk	26 (30.2)	24 (27.9)	36 (41.9)	
Regular soda				0.06
Never	30 (19.7)	55 (36.2)	67 (44.1)	
1-3 times per mo	73 (29.2)	146 (41.4)	126 (35.7)	
1-3 times per wk	40 (25.6)	54 (36.7)	53 (36.1)	
≥4 times per wk	26 (30.2)	24 (27.9)	36 (41.9)	
Fruit juice				0.70
Never	34 (30.6)	44 (29.7)	44 (39.6)	
1-3 times per mo	78 (27.6)	108 (38.2)	126 (34.3)	
1-3 times per wk	55 (24.4)	86 (38.2)	84 (37.3)	
≥4 times per wk	26 (28.9)	31 (34.4)	33 (36.7)	
Sweetened juice				0.98
Never	24 (26.7)	31 (34.4)	35 (38.9)	
1-3 times per mo	55 (26.2)	81 (38.6)	74 (35.2)	
1-3 times per wk	77 (28.1)	95 (34.7)	102 (37.2)	
≥4 times per wk	37 (27.4)	51 (37.8)	47 (34.8)	
VPA				**≤0.01**
< ½ hr per wk	63 (36.8)	65 (38.0)	43 (25.1)	
½ to 2 hrs per wk	60 (33.9)	60 (33.9)	57 (32.2)	
2½ to 4 hrs per wk	29 (21.3)	61 (44.9)	46 (33.8)	
>4 hrs per wk	41 (18.2)	72 (32.0)	112 (49.8)	
MPA				0.07
< ½ hr per wk	42 (38.9)	30 (27.8)	36 (33.3)	
½ to 2 hrs per wk	61 (27.2)	82 (36.6)	81 (36.2)	
2½ to 4 hrs per wk	45 (26.6)	63 (37.3)	61 (36.1)	
>4 hrs per wk	44 (21.3)	83 (40.1)	80 (38.6)	
LPA				**≤0.01**
< ½ hr per wk	20 (30.8)	27 (41.5)	18 (27.7)	
½ to 2 hrs per wk	76 (35.8)	65 (30.7)	71 (33.5)	
2½ to 4 hrs per wk	39 (25.2)	60 (38.7)	56 (36.1)	
>4 hrs per wk	58 (20.9)	106 (38.3)	113 (40.8)	

Table 5.3 Continued				
AHA Recommendations				**≤0.01**
< 2.5 hrs per wk	58 (40.3)	49 (34.0)	37 (25.7)	
> 2.5 hrs per wk	135 (23.9)	209 (37.0)	221 (39.1)	
Screen Time				0.14
< 2 hrs per day	76 (27.0)	103 (36.5)	103 (36.5)	
2-3 hrs per day	44 (25.6)	73 (42.4)	55 (32.0)	
3-4 hrs per day	36 (27.1)	51 (38.3)	46 (34.6)	
> 4 hrs per day	37 (30.3)	31 (25.4)	54 (44.3)	

Data are mean ±SD or n (%);
AHA = American Heart Association, EOs = Eating Occasions, LPA – Light Physical Activity, MPA = Moderate Physical Activity, VPA = Vigorous Physical Activity;
Chi-square analyses were run for categorical outcomes;
ANOVAs were run to compare BMI and BMI percentile, the only continuous outcome measures, across levels of eating frequency.

Table 5.4 presents differences in demographics, obesity parameters, dietary intake and physical activity behaviors between breakfast consumption groups. Chi-square analysis found significant differences in soda intake, sweetened juice intake, AHA MVPA guidelines, VPA, MPA, and screen time between breakfast consumption groups. Regular breakfast consumers compared to breakfast skippers were more likely to accumulate more time in VPA, MPA, meet AHA activity guidelines and never drink soda. No significant differences in age, sex, BMI, or weight status groups were observed between breakfast groups.

Table 5.4: Demographics, obesity parameters, dietary intake and physical activity behaviors by Breakfast groups

	Breakfast Skippers (n=99)	**Intermittent Breakfast Consumers (n=375)**	**Regular Breakfast Consumers (n=235)**	**p-value**
Age (y)	18.7 ±0.4	18.7 ±0.4	18.7 ±0.4	0.92
Semester				0.28
Fall	56 (14.1)	199 (50.3)	141 (35.6)	
Spring	28 (13.7)	109 (53.4)	67 (32.8)	
Summer	15 (13.8)	67 (61.5)	27 (24.8)	
Sex				0.12
Male	53 (17.0)	161 (51.6)	98 (31.4)	
Female	46 (11.6)	214 (53.9)	137 (34.5)	
BMI	23.4 ±4.1	23.8 ±4.4	23.6 ±43	0.68
BMI percentile	55.9 ±28.7	58.7 ±29.0	57.7 ±26.9	0.67
				0.33

Table 5.4 Continued

Weight Status				
Underweight	5 (14.7)	23 (67.6)	6 (17.6)	
Normal weight	66 (14.4)	229 (50.1)	162 (35.4)	
Overweight	22 (13.7)	89 (55.3)	50 (31.1)	
Obese	6 (10.5)	34 (59.6)	17 (29.8)	
Fast food				0.17
Never	15 (12.3)	59 (48.4)	48 (39.3)	
1-2 visits per wk	47 (13.3)	183 (51.8)	123 (34.8)	
3-4 visits per wk	23 (15.6)	79 (53.7)	45 (30.6)	
>4 visits per wk	14 (16.3)	54 (62.8)	18 (20.9)	
Regular soda				**≤0.01**
Never	11 (7.2)	70 (46.1)	71 (46.7)	
1-3 times per mo	31 (12.4)	143 (57.2)	76 (30.4)	
1-3 times per wk	26 (12.3)	115 (54.5)	70 (33.2)	
≥4 times per wk	31 (32.3)	47 (49.0)	18 (18.8)	
Fruit juice				0.71
Never	14 (12.6)	57 (51.4)	40 (36.0)	
1-3 times per mo	42 (14.8)	147 (51.9)	94 (33.2)	
1-3 times per wk	35 (15.6)	122 (54.2)	68 (30.2)	
≥4 times per wk	8 (8.9)	49 (54.4)	33 (36.7)	
Sweetened juice				**0.05**
Never	10 (11.1)	40 (44.4)	40 (44.4)	
1-3 times per mo	23 (11.0)	114 (54.3)	73 (34.8)	
1-3 times per wk	40 (14.6)	146 (53.3)	88 (32.1)	
≥4 times per wk	26 (19.3)	75 (55.6)	34 (25.2)	
VPA				**≤0.01**
< ½ hr per wk	38 (22.2)	90 (52.6)	43 (25.1)	
½ to 2 hrs per wk	23 (13.0)	100 (56.5)	54 (30.5)	
2½ to 4 hrs per wk	19 (14.0)	73 (53.7)	44 (32.4)	
>4 hrs per wk	19 (8.4)	113 (49.8)	94 (41.8)	
MPA				**0.02**
< ½ hr per wk	21 (19.4)	62 (57.4)	25 (23.1)	
½ to 2 hrs per wk	30 (13.4)	122 (54.5)	72 (32.1)	
2½ to 4 hrs per wk	25 (14.8)	94 (55.6)	50 (29.6)	
>4 hrs per wk	23 (11.1)	96 (46.4)	88 (42.5)	
LPA				0.73
< ½ hr per wk	11 (16.9)	37 (56.9)	17 (26.2)	
½ to 2 hrs per wk	30 (14.2)	117 (55.2)	65 (30.7)	
2½ to 4 hrs per wk	23 (14.8)	78 (50.3)	54 (34.8)	
>4 hrs per wk	35 (12.6)	143 (51.6)	99 (35.7)	
AHA Recommendation				**≤0.01**
< 2.5 hrs per wk	27 (18.8)	83 (57.6)	34 (23.6)	
> 2.5 hrs per wk	72 (12.7)	292 (51.7)	201 (35.6)	
Screen Time				**≤0.01**
< 2 hrs per day	33 (11.7)	144 (51.1)	105 (37.2)	
2-3 hrs per day	22 (12.8)	88 (51.2)	62 (36.0)	
3-4 hrs per day	17 (12.8)	84 (63.2)	32 (24.1)	
> 4 hrs per day	27 (22.1)	59 (48.4)	36 (29.5)	

Data are mean ±SD or n (%);
AHA = American Heart Association, EOs = Eating Occasions, LPA = Light Physical Activity, MPA = Moderate Physical Activity, MVPA = Moderate to Vigorous Physical Activity, VPA = Vigorous Physical Activity;
Chi-square analyses were run for categorical data;
ANOVAs were run to compare BMI and BMI percentile, the only continuous outcome measures, across levels of breakfast consumption.

Adjusted odds ratios of subjects meeting AHA physical activity recommendation, never drinking soda, and never eating fast food between eating frequency groups are depicted in **Table 5.5.** Those who consumed 4 or more EOs were more likely to meet the AHA physical activity recommendation compared to those who consumed 2 or less EOs (AOR: 2.6, CI: 1.6-4.1 $p \leq 0.01$). Those who consumed 4 or more EOs more likely to never drink soda compared to those who consumed 2 or less EOs (AOR: 1.9, CI: 1.2-3.1, $p \leq 0.01$). Finally, compared to participants who consumed 2 or less EOs per day, those who consumed 3 EOs per day and those who consumed 4 or more EOs per day were more likely (AOR: 2.2, CI: 1.3-3.6, $p \leq 0.01$) and 60% more likely (AOR: 1.7, CI: 1.0-2.7, $p = 0.05$) to never consume fast food.

Table 5.5: Adjusted Odds Ratios of Dietary and Physical Activity Variables by Eating Frequency Groups

	Meeting AHA Recommendation n (%)	AOR (95% CI)	p-value
≤ 2 EOs Per Day (n=193)	135 (69.9)	reference	reference
3 EOs Per Day (n=258)	209 (81.0)	1.8 (1.2-2.9)	**≤0.01**
≥4 EOs Per Day (n=258)	221 (85.7)	2.6 (1.6-4.1)	**≤0.01**
	Never Drink Soda n (%)	AOR (95% CI)	p-value
≤ 2 EOs Per Day (n=193)	30 (15.5)	reference	reference
3 EOs Per Day (n=258)	55 (21.3)	1.5 (0.9-2.5)	0.13
≥4 EOs Per Day (n=258)	67 (26.0)	1.9 (1.2-3.1)	**≤0.01**
	Never Eat Fast Food n (%)	AOR (95% CI)	p-value
≤ 2 EOs Per Day (n=193)	46 (23.8)	reference	reference
3 EOs Per Day (n=258)	33 (12.8)	2.2 (1.3-3.6)	**≤0.01**
≥4 EOs Per Day (n=258)	43 (16.7)	1.7 (1.0-2.7)	**0.04**

AOR = Adjusted Odds Ratio, CI = Confidence Interval, EOs = Eating Occasions, MVPA – Moderate to Vigorous Physical Activity;
Binary logistic regressions were run, covariates included age, sex, and semester of screener collection.

Adjusted odds ratios of subjects meeting physical activity guidelines, never drinking soda, and accumulating less screen time between breakfast consumption groups are depicted in **Table 5.6.** Regular breakfast consumers compared to breakfast skippers were more likely to meet AHA's physical activity guideline (AOR: 2.3, CI: 1.3-4.2, $p \leq 0.01$) and more likely to never drink soda (AOR: 3.3, CI: 1.7-6.7, $p \leq 0.01$). Regular breakfast consumers compared to breakfast skippers were also more likely to accumulate less than 2 hours per day of screen time (AOR: 1.7, CI: 1.0-2.7, $p=0.05$).

Table 5.6: Adjusted Odds Ratios of Dietary and Physical Activity Variables by Breakfast Groups

	Met AHA Recommendation n (%)	AOR (95% CI)	p-value
Breakfast Skippers (n=99)	72 (72.7)	reference	reference
Intermittent Breakfast consumers (n=375)	292 (77.9)	1.4 (0.8-2.3)	0.20
Regular Breakfast consumers (n=235)	201 (85.5)	2.3 (1.3-4.2)	**≤0.01**
	Never Drink Soda n (%)	AOR (95% CI)	p-value
Breakfast Skippers (n=99)	11 (11.1)	reference	reference
Intermittent Breakfast consumers (n=375)	70 (18.7)	1.8 (0.9-3.5)	0.11
Regular Breakfast consumers (n=235)	71 (30.2)	3.3 (1.7-6.7)	**≤0.01**
	Less Than 2 hr/day of Screen Time n (%)	AOR (95% CI)	p-value
Breakfast Skippers (n=99)	33 (33.3)	reference	reference
Intermittent Breakfast consumers (n=375)	144 (38.4)	1.3 (0.8-2.0)	0.35
Regular Breakfast consumers (n=235)	105 (44.7)	1.7 (1.0-2.7)	**0.05**

AOR – Adjusted Odds Ratio, CI – Confidence Interval, MVPA – Moderate to Vigorous Physical Activity
Binary logistic regression was run, covariates included age, sex, and semester of screener collection.

DISCUSSION

To our knowledge, this is the first study to examine the relationship of eating frequency and breakfast consumption with obesity parameters, dietary intake, and physical activity behaviors in a sample of exclusively Hispanic college freshmen. Contrary to our hypothesis, there were no significant relationships between eating frequency and breakfast consumption groups with any BMI parameters or overweight/obese prevalence utilizing the adult or child cut offs. However, increased eating frequency and increased breakfast consumption were linked to healthier dietary and physical activity behaviors. Participants who ate more frequently compared to those who ate infrequently were more likely to meet the AHA's physical activity recommendation, never drink soda, and never eat fast food. Regular breakfast consumers compared to breakfast skippers were more likely to meet the AHA's physical activity recommendation, never drink soda, and accumulate less screen time.

Possible explanations for the null findings with obesity parameters should be explored. Potential reasons for this null finding could be that this study is cross-sectional and the effects of eating frequency and breakfast intake on obesity levels may not have occurred yet. The freshmen year is characterized as a transition year and it is very possible that the shift in breakfast and eating occasion habits is relatively new and it has not yet adversely affected obesity levels. Interestingly, 67% those who had 2 or less EOs as freshmen in college indicated consuming 3 or more EOs in high school. In addition, a higher percentage of students consumed 4 or more EOs per day from screeners collected

during the fall semester compared to the spring and summer semesters. These findings suggest that subjects decrease their eating occasions when entering college and it is possible that the adverse effects of reduced EOs on weight status have not had time to occur yet.

Another explanation for the null obesity findings is that this sample of Hispanic college freshmen from UT-Austin had significantly lower prevalence levels of overweight/obesity than national samples. National data collected in Hispanic adolescents (12-19 y) show that 38.1% are either overweight or obese [125], compared to the only 30.7% in the current sample of Hispanic college freshmen. The 2015 American College Health Association – National College Health Assessment II (ACHA-NCHA II) found that out of 74,438 undergraduate students from around the country, 33% of college students reported being overweight or obese [142]. Of note, this study included a much larger age range starting at age 18 and going above age 30 and only 13% were of Hispanic decent and the overweight/obese prevalence rates were not broken down age range or by race/ethnicity. To date, no other study has reported the overweight/obese prevalence of Hispanic college students and the current data suggests that this population is less overweight/obese than other national Hispanic adolescent populations.

Even less is known about physical activity patterns among Hispanic college students and exploring the physical activity patterns of Hispanic youth as they transition to young adulthood is crucial given their higher risk of obesity. The National Health and Nutrition Examination Survey (NHANES) physical activity data found that 42.0% of

children (6-11 y) attained the recommended public health recommendation of 30 or more minutes of MVPA at least five days per week, however this number plummeted to around 8.0% in adolescents (12-19 y) [40]. A study by Belcher et al. [41] in 3,106 subjects with accelerometry data found that minority youth were more active than NHW and that activity declined with age regardless of ethnicity. Comparatively, the ACHA-NCHA II found that 47% of college students reported meeting the AHA's physical activity recommendation and this percentage is much higher than NHANES data from the general population [142]. However, a recent study by Downs et al. [143] in 77 college students (18-22 y) with accelerometer data found that this may be an overestimation as just 35% of their sample met the physical activity recommendation. Our sample appears to be more active than these studies, with 80% of our sample reporting at least 30 or more minutes of MVPA at least five days per week on the screener and a subsample of 87 subjects with accelerometery data found that 94% met the physical activity recommendation. This may be due to the fact that the UT-Austin campus is very large and students walk to and from classes. Our sample may not be representational of other college campuses and Hispanic college freshmen populations as University of Texas at Austin has consistently been ranked among national polls as one of the healthiest colleges in the nation, yet it does highlight the need for further research in this area [144,145].

Furthermore, to our knowledge few studies have examined the relationship between eating frequency and physical activity in youth and college populations. The current study found that those who ate most frequently were more likely to meet the

AHA physical activity recommendation compared to those who ate least often. Similarly, Zerva et al.[13] found that total physical activity was highest among those who ate most frequently in a sub-sample of 48 children (9-11 y) with accelerometer data. Duval et al.[44] found the amount of physical activity to be the confounding factor in the relationship between increased eating frequency and healthier adiposity measures in a population of 85 women (47-56 y). In contrast, our previous findings found no statistically significant difference in MVPA collected via accelerometer between infrequent and frequent eaters, however, this sample was low income and exclusively overweight and obese of 185 minority youth (8-18 y), which is very different from the current sample [1]. Thus, further research exploring the relationship that eating frequency may have on physical activity and unraveling the potential effects each of these may have on obesity risk is warranted across all populations.

Comparatively, missing breakfast has also been related to decreased physical activity in multiple youth samples [146-148]. A two year longitudinal study by Laska et al. with 693 subjects (12-17 y) found those who consumed breakfast more frequently at baseline were more likely to have lower BMI and percent body fat at two years and this finding remained after controlling for physical activity collected via accelerometer [149]. To our knowledge the current study is the first to examine the relationship between eating frequency and breakfast consumption with physical activity patterns in a Hispanic college freshmen population. Our findings and the available literature on this topic are largely cross-sectional and more longitudinal and intervention studies are warranted to

understand causation and the mechanisms driving how eating patterns influence physical activity levels and obesity risk.

The inverse relationship between soda intake and both increased eating frequency and breakfast consumption is most interesting given that national adult studies have shown that increased EOs result in increased daily quantity of foods/beverages consumed, as well as daily energy density (kcal/g) of all foods and beverages reported [118]. Furthermore, among youth (2-19 y) each additional eating occasion predicted a 200 kcal increase in energy intake [8] and research consistently shows a positive relationship between both eating frequency and breakfast consumption with caloric intake [1,2,13,77]. However, breakfast consumption has been linked to healthier eating behaviors in youth samples and the current findings suggest that regular breakfast consumers and those who eat more often are more likely to never consume soda. No significant difference in fruit juice consumption was observed between breakfast or eating frequency groups and this could be due to subjects perceiving fruit juice as a healthier option. Another explanation is the clustering of healthy dietary behaviors, i.e., participants who regularly consume breakfast and eat more frequently are also more likely to not drink soda [150]. However, more research to further elucidate how eating frequency and breakfast consumption influences sugar sweetened beverage consumption is needed.

There are a several potential limitations in the current analysis. The first limitation is that this is a cross-sectional study and thus causation cannot be assessed. Another limitation is the screener is quite short and more questions regarding snack and meal

intake may have helped understand these subject's dietary intake and patterns further. Another limitation is that the screener data for eating frequency and breakfast consumption was around 40% concordant with the dietary recall data from three or more days. However, it is important to note that three dietary recalls may not be enough to truly ascertain habitual intakes [151,152]. It is also likely more difficult for participants to remember and properly count EOs throughout the day on the screener and the number of times they consumed breakfast over the previous week compared to dietary recalls of the previous day. Yet, given the literature and media consistently advocate increased eating frequency and breakfast consumption as healthy behaviors developing a better definition and more accurate methodology for measuring eating frequency and breakfast consumption in large populations is warranted [67].

Conclusions:

Breakfast consumption has been identified as an eating behavior that may lower obesity risk [72] and eating frequency research has more consistently found an inverse association between the number of EOs per day and adiposity [13-15]. However, the current data did not find any statistically significant difference between eating frequency, breakfast consumption, and overweight or obesity status among Hispanic college freshmen. However, both increased breakfast consumption and increased eating frequency were linked to healthier dietary (i.e., less soda and sweetened juice) and physical activity behaviors (i.e., increased MVPA) and it is possible that not enough time

has accrued to see significant differences in adiposity measures among these groups. Thus, further research especially longitudinal and intervention studies analyzing the relationships between eating frequency and breakfast consumption and the potential impact these habits have on other dietary behaviors, physical activity and obesity risk is warranted in order to better inform public health messages, particularly within Hispanic youth populations.

Chapter 6: Decreased Eating Frequency Linked to Increased Visceral Adipose Tissue, Body Fat, and Obesity Risk in Hispanic College Freshmen.

ABSTRACT

Background: Previous research on eating frequency and adiposity has yielded mixed results. Our group found a negative relationship between eating frequency and adiposity in Hispanic youth (8-18 y). However, this relationship has never been investigated in Hispanic college freshmen, an understudied population, which may be particularly susceptible to obesity and metabolic diseases. Furthermore, behavioral choices formed during this transitional period are likely to continue throughout adulthood and may increase chronic disease risk later in life.

Objective: To investigate the impact of eating frequency on dietary intake and comprehensive adiposity measures in Hispanic college freshmen.

Design: This study included 92 Hispanic college freshmen (18-19 y) with the following cross-sectional measures: height, weight, waist circumference, body mass index (BMI), dietary intake, body composition, physical activity, hepatic fat, visceral adipose tissue (VAT), and subcutaneous adipose tissue (SAT). Each eating occasion (EO) was defined as ≥50 calories and ≥15 minutes from any previous EO. Only infrequent eaters who averaged less than three EOs per 24 hours (n=45) or frequent eaters who averaged 4 or more EOs per 24 hours on the majority of their dietary recalls (n=47) were included.

Results: Infrequent eaters ate 44% less often ($p \leq 0.01$) and consumed 27% more calories per EO ($p \leq 0.01$), while consuming 21% less kcals per day (or 445 less kcals per day) ($p \leq 0.01$) compared to frequent eaters. Infrequent eaters had 8% higher BMIs ($p=0.02$), 60% higher BMI z-scores ($p=0.03$), 21% higher VAT ($p=0.03$), 26% higher SAT ($p=0.03$), and 8% higher total body fat ($p=0.04$) compared to frequent eaters.

Conclusions: These findings suggest that decreased eating frequency is related to increased adiposity in Hispanic college freshmen, despite decreases in daily energy intake. Yet, more research is needed to understand the underlying mechanisms of these findings, as well as investigate any potential causal relationship between eating frequency and adiposity in Hispanic youth.

INTRODUCTION

College students are particularly susceptible to poor overall health and the transition to college has been identified as a critical period contributing to the rise in obesity rates as the behavioral choices college students make likely affect their risk of chronic disease later in life[20]. In 2012, for the first time in US history, Hispanic high school graduates (69%) were more likely to be enrolled in college than Non-Hispanic Whites (NHW; 67%) and Blacks (63%)[18]. In 2013, Hispanic students represented 23% of freshman enrollment at the University of Texas at Austin (UT-Austin), having the largest increase among all minority groups[19]. Currently, 67% of Hispanics (12-19 y) are either overweight or obese[125], yet little is known about overweight or obese prevalence rates of

Hispanic college students. Several studies have shown that the initial transition to college is associated with rapid weight gain and the average weight gain in the first year of college ranges from 3.5 to 8.8 pounds[27]. Decreased dietary fiber, fruits and vegetables[26,126] and increased junk food consumption[127] are among the dietary factors linked to increased obesity rates in primarily NHW college students. However, no study has investigated the relationship eating frequency patterns and obesity risk in a population of exclusively Hispanic college freshmen.

Eating frequency research has consistently found a negative association between the number of eating occasions (EOs) per day and adiposity, as well as metabolic disease risk in both youth and adult populations[13,15,31]. Our group has shown that infrequent eating is linked to increased obesity measures, blunted insulin action, and deleterious lipid parameters in multiple populations of overweight Hispanic youth (8-18 y)[1,2]. However, to date, no group has looked at the effect of eating frequency on adiposity and metabolic disease risk in a sample of Hispanic college freshmen. Thus, the goal of this study is to examine the relationship between eating frequency and specific adiposity markers in this potentially high-risk population of Hispanic college freshmen to better inform interventions that may reduce this risk within such a crucial period of life. We hypothesized that infrequent eating in relation to frequent eating would be inversely associated with energy intake and physical activity, but positively associated with adiposity measures in Hispanic college freshmen.

METHODS

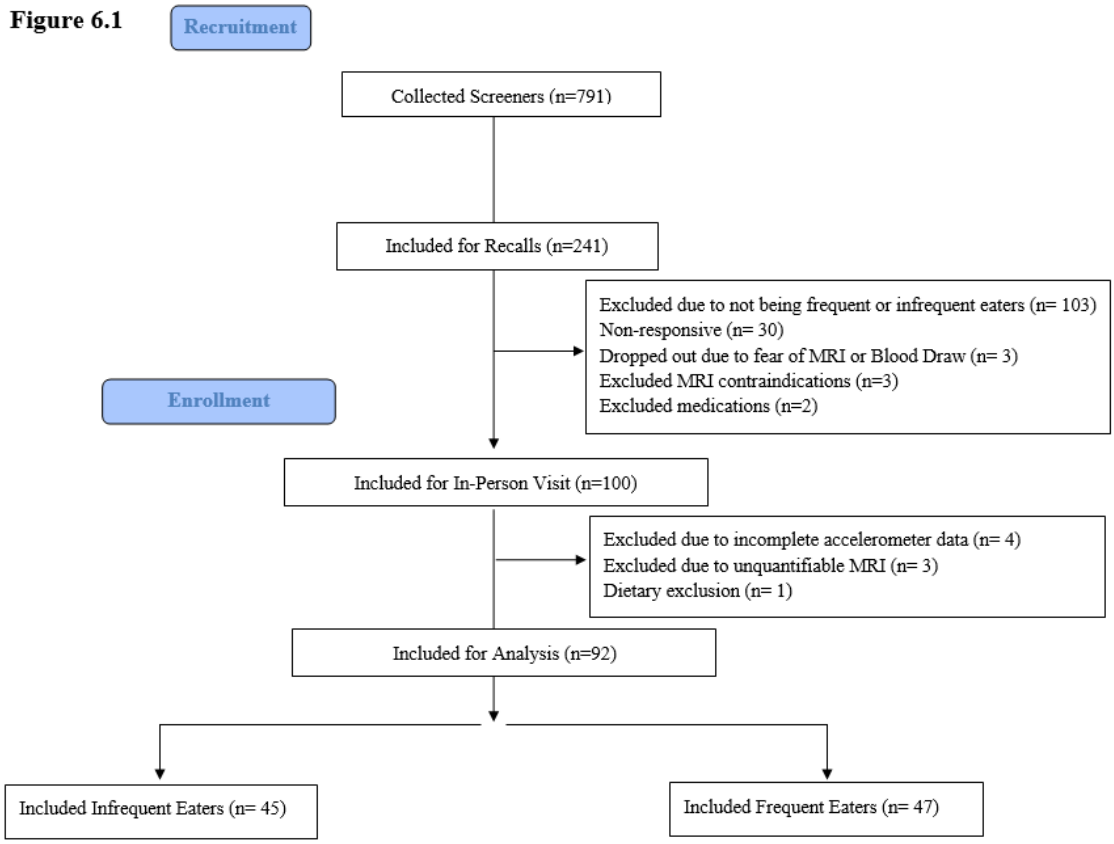
Subjects:

Inclusion criteria for the study were as follows: (i) self-reported that all four of their grandparents were of Hispanic origin (ii) 18-19 years of age, and (iii) in their first year of college. Exclusion criteria for the study were as follows: (i) if they were pregnant, (ii) if they were taking any medication known to affect body composition or any psychoactive medication, (iii) if they had been diagnosed with a disease/s or syndrome known to affect body composition or fat distribution, (iv) if they had a learning impairment that would complicate survey administration, (v) if they had braces, a pacemaker, or any of other contraindications to magnetic resonance imaging (MRI) scanning, or (vi) if they had taken part in a weight loss, dietary, or physical intervention in the previous 6 months. This study was approved by the Institutional Review Board of the University of Texas at Austin. Informed written consent was obtained before testing commenced.

Figure 6.1 provides a detailed flow of study participants. Participants were recruited via announcement in classes, word of mouth, and electronic posted notices on the UT-Austin campus (n=791). Subjects initially completed a 21 item dietary, which asked about eating frequency habits, adapted from Project Eat[135-137]. Only infrequent eaters who averaged less than 3 eating occasions (EOs) per 24 hours (n=45) or frequent eaters who averaged 4 or more EOs per 24 hours on the majority of their dietary recalls (n=47) were brought in for the in-person visit. Three or more 24 hour multiple-pass

dietary recalls were collected in Twenty-seven percent (n=241) of the total subject pool to verify eating frequency. Of those subjects, 43% (n=103) were not frequent or infrequent eaters as determined via dietary recalls, and an additional 38 participants were either non-responsive or did not qualify due to other exclusionary criteria. One-hundred subjects were then brought in for the in-person visit. There were a total of 100 subjects who completed the in-person visit, one subject did not have adequate dietary data, three participants did not have specific fat distribution data, and four subjects did not attain three days of eight hours or more of physical activity data via accelerometer, leaving the final sample size at 92 subjects.

Figure 6.1



Anthropometrics and Adiposity Measures:

Height and weight were measured to the nearest 0.1 kg and 0.1 cm using a beam medical scale and a wall-mounted stadiometer, respectively, and the average of two measurements was used for the analysis. BMI was calculated utilizing adult cut offs and BMI percentiles and z-scores were determined by using EPII 2000 software (version 1.1; Centers for Disease Control and Prevention, Atlanta, GA)[93]. Subjects were categorized as overweight if they had a BMI of 25.0 to < 30.0 and obese if they had a BMI >30.0 utilizing adult cut offs. Waist circumference was measured and recorded to the nearest 0.1 cm. Body fat and soft lean tissue were measured using air displacement plethysmography, which has been validated against hydrodensitometry in overweight adults (BOD POD, Cosmed 2007B, Concord, CA)[153].

Image Acquisition

Abdominal fat distribution: visceral adipose tissue (VAT), subcutaneous adipose tissue (SAT), and hepatic fat were assessed via magnetic resonance imaging (MRI) at the UT- Austin Imaging Research Center on a research-dedicated Siemens Skyra 3 Tesla scanner. Non-Alcoholic Fatty Liver Disease (NAFLD) was categorized utilizing the cut point derived from the Dallas Heart Study[154]. Visceral adipose tissue (VAT), subcutaneous adipose tissue (SAT), and liver fat volume were measured using a vibe 3D

DIXON technique. This scanning protocol contained one slab with 88 slices, each 3mm thick to allow for imaging of the entire abdominal area in the coronal direction. The field of view (FoV) was 380mm, and the phase FoV was 80.6%, with a repetition time (TR) of 3.90 seconds, an echo time (TE) of 2.46ms, and a flip angle of 9.0 degrees. One set each of in-phase images and one out-of-phase images were acquired [6]. The Siemens Skyra 3T used a portion of a 32-channel coil array integrated into the patient bed/table. A 4-channel large array coil or a 4-channel small array coil was placed anteriorly and used in combination with the 32-channel coil to provide full abdominal coverage. Using a high number of coil elements in this way made an ideal experimental configuration to take advantage of a partially parallel image acquisition acceleration method. The GeneRalized Auto-calibrating Partially Parallel Acquisitions (GRAPPA) technique used for this study to accelerate image acquisition. Fat volume fraction and fat mass fraction were computed on a voxel-by-voxel basis, and averaged over each segmented organ. VAT and SAT were measured as a region of interest (ROI) from the top of the ileac crest to the bottom of the ribcage. The benefits of using ROI for liver fat and body fat quantification as well as challenges associated with fat quantification from MRI can be found here [11,153]

Fat mass quantification

Percent water and fat were calculated using a novel quantification program developed by Dr. Jeff Luci and the Imaging Research Center staff based on the Otsu

method [12] and run in MATLAB (version R2013a, MathWorks Inc, Natick, MA). Initially, the total body fat was calculated and the subcutaneous fat was then calculated. The removal of the SAT from the total body fat yielded VAT. An average of 26 slices were taken from the abdominal area and there was not significant difference in the number of slices between groups. At least two researchers quantified the fat values for each subject. No significant differences in any of the outcome variables or MRI slices were seen between coders utilizing t-tests.

Dietary Intakes:

Dietary intakes were assessed from three to four 24-h diet recalls using the multiple-pass technique, research staff were trained and supervised by a Registered Dietitian. All subjects had at least three recalls (one weekend and two weekdays). On average recalls were administered within 5 days from the in-person testing visit. Quality assurance was performed on all dietary recall data by additional trained research staff. Nutritional data was analyzed by using the Nutrition Data System for Research (NDS-R, 2014). The NDS-R program calculated key dietary variables for this analysis, including mean energy, total fat, protein, carbohydrates, saturated fat, total sugar, added sugar, dietary fiber, soluble fiber, and insoluble fiber. Prospectively, no recall was performed if the subject indicated being ill. One subject was excluded from all the analyses due to having a very low carbohydrate intake (< 10 grams per day) and being an extreme outlier in percentage of caloric intake from carbohydrate. The Gibney et al. eating occasion

definition was used to quantify eating frequency and each EO was defined as ≥50 calories and ≥15 minutes from any previous EO[155].

Physical Activity:

Physical activity was measured by accelerometers (wGT3X-BT, Actigraph, LLC., Pensacola, FL). Physical activity was measured for seven days and on the same week as the in-person visit and dietary recalls. All accelerometer data was immediately downloaded and wear time was assessed. Days with less than 8h of wear data were not considered acceptable, and only participants with ≥3 days of acceptable accelerometry data were included in the physical activity analysis. Subjects with valid data (n=96) wore the accelerometers for a mean of 12.9 ±1.6 hours/day for 6.2 ±1.5 days. Data from all acceptable days was averaged and included the following variables: minutes and percent time spent in, light physical activity (LPA), moderate to vigorous PA (MVPA), and sedentary behavior (SED). Freedson adult cut offs were used to quantify and classify the accelorometry data[141].

Statistics:

Data was examined for normality, and transformations were made if the data was found to be significantly different from normal. The following outcome variables were non-normally distributed and were either log or inversely transformed before the analysis: BMI, VAT, SAT, hepatic fat, mean energy intake, percent dietary protein,

saturated fat, dietary fiber, total fiber, soluble fiber, and insoluble fiber. However, non-transformed values are presented in the tables and figures for ease of interpretation. Chi-square, t-test, and MANCOVA analyses were used to assess differences in demographics, dietary intake variables, and adiposity and physical activity measures between the two eating frequency groups. In all models the following *a priori* covariates were included: sex, age and MVPA (when adiposity measures were the dependent variables). All analyses were performed by using SPSS version 20.0 (SPSS, Chicago, IL), and the significance was set at $p \leq 0.05$.

RESULTS

The basic demographic data and adiposity measures are presented in **TABLE 6.1**. Ninety-two subjects had complete anthropometric, dietary, and body composition data. The sample was 51% female and averaged 18.8 years of age.

TABLE 6.1
Subject Characteristics [1,2]

Physical and Adiposity Measures (n=92)	
Sex M/F	45/47
Age (y)	18.8 ±0.4
Height (cm)	167.4 ±9.8
Weight (kg)	67.1 ±13.8
Waist circumference (cm)	84.6 ±9.7
BMI (kg/m^2)	23.8 ±3.9
BMI percentile	59.9 ±27.3
BMI z score	0.3 ±1.0
Overweight Prevalence	21 (22.8)
Obese Prevalence	8 (8.7)
Overweight/Obese Prevalence	29 (31.5)
Total lean tissue (%)	73.6 ±9.9
Total body fat (%)	26.4 ±9.9
VAT (ml)	267.0 ±124.4
SAT (ml)	999.7 ±654.9
Hepatic fat (ml)	29.2 ±36.0
NAFLD	20 (21.7)

[1] Data presented as mean ±SD or n (%)
[2] NAFLD = Non Alcoholic Fatty Liver Disease, SAT = Subcutaneous Adipose Tissue, VAT = Visceral Adipose Tissue

TABLE 6.2 presents dietary and physical activity data. The average number of EOs per 24 hours was 3.6, the average energy intake was close to 2,000 kcals per day, and subjects averaged more than 60 min per day of MVPA.

TABLE 6.2

Behavioral Characteristics [1,2]

Dietary and Physical Activity Variables (n=92)	
Eating occasions per day	3.6 ±1.1
Energy per eating occasion (kcal)	580.9 ±241.6
Energy (kcal/d)	1,941.9 ±729.4
Total fat (g/day)	75.8 ±31.5
Total fat (% kcal)	33.9 ±5.6
Total protein (g/day)	82.7 ±42.1
Total protein (% kcal)	17.1 ±4.5
Total carbohydrate (g/day)	237.4 ±94.1
Total carbohydrate (% kcal)	48.8 ±7.4
Total saturated fat (g/day)	23.7 ±11.4
Total saturated fat (% kcal)	10.5 ±2.5
Total sugars (g/day)	95.3 ±52.6
Total sugars (% kcal)	20.0 ±7.7
Added sugars (g/day)	62.3 ±46.0
Added sugars (% kcal)	12.5 ±6.7
Dietary fiber (g/day)	16.9 ±7.5
Dietary fiber (g/1000 kcal)	9.0 ±3.4
Insoluble fiber (g/day)	11.3 ±5.5
Insoluble fiber (g/1000 kcal)	6.0 ±2.5
Soluble fiber (g/day)	5.4 ±2.3
Soluble fiber fiber (g/1000 kcal)	2.9 ±1.1
Min per day in MVPA	67.2 ±27.0
Percent wear time in MVPA (%)	8.7 ±3.4
Min per day in LPA	100.5 ±32.4
Percent wear time in LPA (%)	12.9 ±3.5
Min per day in SED	606.8 ±81.4
Percent wear time in SED (%)	78.4 ±4.7

[1] Data presented as mean ±SD

[2] LPA = Light Physical Activity, MVPA = Moderate to Vigorous Physical Activity, SED = Sedentary Behavior

TABLE 6.3 presents adiposity measures by the two eating frequency groups. Infrequent eaters compared to frequent eaters were slightly older (p=0.05) and no significant difference in sex was found between eating frequency groups. Using MANCOVA analyses, infrequent eaters had 8% higher BMIs (p=0.02), 60% higher BMI z-scores (p=0.03), 21% higher VAT (p=0.03), 26% higher SAT (p=0.03), and 8% higher total body fat (p=0.04). However, no significance difference was found in liver fat or risk of NAFLD.

TABLE 6.3

Adiposity measures and metabolic parameters between eating frequency groups[1,2,3,4]

	Infrequent Eaters (n=45)	Frequent Eaters (n=47)	p value
Sex M/F	24/21	21/26	0.42
Age (y)	18.9 ±0.4	18.7 ±0.4	**0.05**
Height (cm)	167.0 ±10.4	167.8 ±9.4	0.16
Weight (kg)	69.4 ±16.2	64.7 ±11.2	0.29
Waist circumference (cm)	86.2 ±11.6	83.1 ±7.2	0.19
BMI (kg/m^2)	24.8 ±4.4	22.9 ±3.2	**0.02**
BMI percentile	64.0 ±27.8	55.9 ±26.6	0.08
BMI z score	0.5 ±1.0	0.2 ±1.0	**0.03**
VAT (ml)	298.3 ±153.8	236.8 ±78.2	**0.03**
SAT (ml)	1150.1 ±765.4	855.6 ±494.6	**0.03**
Hepatic Fat (ml)	31.4 ±39.9	27.1 ±32.1	0.35
NAFLD n (%)	9 (20)	11 (23.4)	0.80
Total lean tissue (%)	72.4 ±10.8	74.7 ±8.8	**0.04**
Total body fat (%)	27.6 ±10.8	25.3 ±8.8	**0.04**

[1] Data presented as mean ± SD or n (%)
[2] NAFLD = Non Alcoholic Fatty Liver Disease, SAT = Subcutaneous Adipose Tissue, VAT = Visceral Adipose Tissue
[3] A t-test (for continuous variables) and chi-square analysis (for categorical variables) assessed differences in sex, age, height, and weight between groups
[4] MANCOVA analysis of adiposity measures between Infrequent Eaters and Frequent Eaters (n=92). *A priori* covariates included: age, sex, and percent time spent in moderate to vigorous physical activity.

Dietary and physical activity variables between frequent eaters and infrequent eaters are depicted in **TABLE 6.4**. T-tests found that infrequent eaters compared to frequent eaters ate 44% less often ($p \leq 0.01$) and ate 27% more calories per EO ($p \leq 0.01$). Infrequent eaters compared to frequent eaters consumed 21% less daily energy intake, or on average 445 less calories per day ($p \leq 0.01$). Infrequent eaters also consumed 8% more dietary fat ($p=0.02$), 17% less total dietary fiber ($p=0.02$) and 18% less insoluble fiber ($p=0.02$) than frequent eaters.

TABLE 6.4

Dietary intake and physical activity between eating frequency groups[1,2,3,4]

	Infrequent Eaters (n=46)	Frequent Eaters (n=53)	p value
Eating occasions per day	2.5 ±0.2	4.5 ±0.8	**≤0.01**
Energy per eating occasion (kcal)	670.9 ±254.2	494.6 ±195.1	**≤0.01**
Energy (kcal/day)	1,714.8 ±542.7	2159.5 ±819.7	**≤0.01**
Total fat (%kcal)	35.5 ±5.1	32.5 ±5.6	**0.01**
Total fat (g/day)	70.0 ±26.4	81.4 ±35.0	**0.04**
Total protein (%kcal)	17.2 ±4.4	17.0 ±4.6	0.70
Total protein (g/day)	71.8 ±25.9	93.2 ±51.4	**≤0.01**
Total carbohydrates (%kcal)	47.3 ±7.8	50.3 ±6.7	0.11
Total carbohydrates (g/day)	202.2 ±69.4	271.1 ±102.6	**≤0.01**
Total saturated fat (%kcal)	10.7 ±2.2	10.3 ±2.7	0.38
Total saturated fat (g/day)	21.1 ±8.7	26.1 ±13.1	**0.02**
Total sugars (%kcal)	18.7 ±7.2	21.3 ±8.0	0.20
Total sugars (g/day)	77.8 ±35.0	112.1 ±60.9	**≤0.01**
Added sugars (%kcal)	12.6 ±6.9	12.4 ±6.5	0.61
Added sugars (g/day)	60.7 ±36.5	75.6 ±52.9	0.14
Dietary fiber (g/1000kcal)	8.1 ±2.8	9.9 ±3.7	**≤0.01**
Dietary fiber (g/day)	13.5 ±5.7	20.0 ±7.8	**≤0.01**

TABLE 6.4 Continued

Insoluble fiber (g/1000kcal)	5.3 ±2.3	6.6 ±2.5	**≤0.01**
Insoluble fiber (g/day)	9.1 ±4.3	8.1 ±2.8	**≤0.01**
Soluble fiber (g/1000kcal)	2.6 ±0.7	13.5 ±5.7	0.06
Soluble fiber (g/day)	4.4 ±1.6	6.4 ±2.5	**≤0.01**
Min per day in MVPA	63.5 ±27.0	70.8 ±26.8	0.29
Percent wear time in MVPA (%)	8.3 ±3.7	9.0 ±3.1	0.50
Min per day in LPA	96.8 ±32.0	104.2 ±32.7	0.16
Percent wear time in LPA (%)	12.5 ±3.5	13.3 ±3.5	0.20
Min per day in SED	607.1 ±95.8	606.5 ±65.7	0.93
Percent wear time in SED (%)	79.1 ±4.8	77.7 ±4.7	0.15

[1] Data presented as mean ± SD
[2] LPA = Light Physical Activity, MVPA = Moderate to Vigorous Physical Activity, SED = Sedentary Behavior
[3] T-test assessed differences in eating occasions per day, energy intake, and energy intake per eating occasion between groups
[4] MANCOVA analysis of dietary and physical activity variables between Infrequent Eaters and Frequent Eaters. *A priori* covariates used: age and sex.

DISCUSSION

To our knowledge, this is the first analysis to examine the relationship between eating frequency and dietary and adiposity measures in a sample of exclusively Hispanic college freshmen. To date, the dietary habits and obesity risk of this population remains almost completely unstudied. In the present analysis, infrequent eaters consumed significantly fewer calories per day, yet had significantly higher BMI, BMI z-scores, body fat percentage, and visceral and subcutaneous adipose tissue, while showing no significant differences in physical activity measures. These findings are consistent with other retrospective analyses[1,2,13,15] that have shown a positive association between eating

frequency and caloric intake while showing an inverse association with adiposity measures.

To date, the majority of epidemiology studies have shown an inverse association between eating frequency and adiposity[1,2,14,15,74,76,113], while a few studies have found no relationship or an inverse association[114-116]. A longitudinal study by Ritchie et al.[15], with 2,372 African American and Caucasian girls (9-19 y) found that lower meal frequency was related to greater increases in BMI and waist circumference over a ten year period, independent of socioeconomic status, total energy intake, and physical activity levels. Other studies have found increased eating frequency to be inversely related to waist circumferences[15,60,76] and body fat percent as measured by skinfolds[13]. A recent meta-analysis by Schoenfeld et al.[49] including 15 randomized controlled trials addressing the effects of eating frequency on changes in weight and body composition found that increases in eating frequency were associated with reductions in fat mass and body fat percentage, as well as increases in fat-free mass. However, these findings need to be interpreted cautiously as they seem to be driven by a single study[50]. The above meta-analysis was comprised of an exclusively adult population and to our knowledge, there has yet to be a randomized controlled trial investigating the potential association between eating frequency and obesity risk in any youth population, let alone one of Hispanic descent.

There are numerous potential mechanisms to explain our findings. The first being that increased eating frequency has consistently been related to increased satiety

measures in adult populations[52,61,63,64]. In the cross-over controlled feeding study by Leidy et al.[61] with 13 overweight or obese males, less frequent eating (3 EOs) vs. frequent eating (6 EOs) led to higher satiety throughout the day, but no difference in ghrelin or peptide YY was observed between groups. Smeets et al.[52] found that consuming 3 EOs per day compared 2 EOs per day resulted in greater satiety in a sample of 14 females (19-29 y). These studies controlled for calories but did not address the impact eating frequency may have on ad libitum food intake. Thus, Speechly et al.[64] conducted a cross-over study with eight lean males (19-29 y) where an isocaloric breakfast was consumed in one EOs or five separate EOs consumed every hour over the testing period. Subjects who consumed breakfast in one EO ate 27% more at a subsequent ad libitum lunch, highlighting that infrequent eating may lead to poorer appetite control. Interestingly, utilizing an identical study design with seven obese men (20-55 y), Speechly et al.[65] replicated the findings and found the exact same increase (27%) in the ad libitum lunch intake after the single meal. It is reasonable to infer that increased satiety observed in regards to increased eating frequency may reduce the motivation to eat and therefore reduce energy intake overall, however the vast majority of the eating frequency research shows a positive relationship between eating frequency and energy intake. It is also plausible that reduced eating frequency may result in eating behaviors that resemble binge eating which has been related to increases in metabolic disease parameters and adiposity measures[66]. Thus, much more research is needed to

examine the exact mechanisms of how eating patterns impact satiety, hunger, and ad libitum dietary intake in free-living populations.

Another potential mechanism to consider is how eating frequency impacts metabolic rates. Popular media has consistently advocated more frequent eating or grazing as a healthy habit that may "stoke" or "rev" one's metabolism. A recent review of popular media sources found the ideal eating frequency recommendation given was 6 EOs per day[67]. Yet, not a single study to date has found a statistically significant difference in total energy expenditure between 1, 2, 3, 5, 6 or even 7 EOs in a 24-hour period[52-56]. A cross-over study with eight young adult males (18-23 y) examined the difference in metabolic rate between 2 EOs and 6 EOs per day, which were isocaloric, subjects stayed on each dietary regiment for two weeks and occupied a whole room calorimeter for two 31-h periods[53]. This study found no differences in metabolic rate or energy expenditure between the two EO conditions, despite a small, albeit significant, observed weight gain in the 2 EOs per day condition. However, this study kept the activity patterns constant and eating frequency may subsequently increase physical activity levels, however this and previous analysis in Hispanic youth have not found a difference in physical activity measures between eating frequency groups. In a similar two day cross-over study with 13 male and female young adults (18-23 y) no significant differences in 24-h energy expenditure as measured by a whole room calorimeter were found between 2 EOs and 7 EOs per day conditions[54]. Thus, to date it does not appear that eating more frequently increases metabolic rates. Given the pervasiveness of the current media recommendation further

research into the potential impacts of eating frequency on obesity and metabolic disease risk is warranted to better inform the general public of the potential benefits of frequent eating or deleterious outcomes of infrequent eating.

Another potential mechanism to consider when investigating eating frequency is the potential impact on the thermic effect of food (TEF). TEF is defined as the increase in metabolic rate after the ingestion of a meal. To date, studies examining the impact of eating frequency on TEF have yielded mixed results. One study found an increase in TEF in 1 EO compared to 6 EOs[57], while another study showed an increase in TEF in 4 EOs compared to 1 EO[58], whereas the majority of studies show no significant effect of eating frequency on TEF[52,59,68]. Tai et al.[57] examined the effect of 1 large EO of 750 calories vs. 6 smaller EOs of 125 calories provided every 30 minutes over the same period on TEF in seven women (23-30 y) and found that the one large EO resulted in a slightly higher TEF of 3.4%. The authors hypothesized that this was due to a more rapid absorption of nutrients given that gastric emptying is slower when food is given continuously[69]. Similarly, another study by Leblanc et al. in six subjects (21-28 y) compared 1 vs. 4 EOs given over a four-hour period and found that the 4 EO pattern resulted in a slightly higher TEF. However, eating one time per day is not reflective of normal eating patterns seen in free-living populations and in the current analysis there was not a single subject who averaged 1 EO per day. Furthermore, a comprehensive review by Bellisle et al.[68] concluded that there is no strong evidence to support a biologically significant difference in TEF in response to different eating frequency and furthermore, the role of TEF on body weight regulation itself remains

controversial[70]. To our knowledge, no research study has investigated any potential mediating effect that an increase in TEF from an increase or decrease in eating frequency may have on adiposity parameters. In addition, no study has examined how eating frequency impacts TEF in a youth population.

Another possible mechanism involves lipid metabolism. Our previous studies have found infrequent eating to be associated with higher circulating triglycerides[1,2]. Infrequent eaters also consistently show an increased caloric intake per EO, and binge eating behaviors have been previously linked to increased triglycerides[1,66]. We have also found that infrequent eaters have increased visceral adipose tissue[1] and the accumulation of visceral adipose tissue has been positively associated with fasting insulin and triglycerides[1,120]. It is also hypothesized that visceral fat increases hepatic portal free fatty acid concentrations, which in turn are stored as triglycerides, stimulate hepatic gluconeogenesis, and hinder hepatic clearance of insulin, thus promoting a vicious cycle of hyperinsulinemia, elevated plasma glucose concentrations, and dyslipidemia[121]. The current analysis replicates our previous finding that infrequent eaters have higher visceral adipose tissue than frequent eaters and thus more research especially randomized controlled feeding trials analyzing the possible causal relationship between visceral adiposity, triglycerides, eating frequency, and metabolic disease risk is merited.

Another potential explanation for the current findings is that healthy behaviors tend to cluster together[150]. In the current study, frequent eaters are just consuming more in general, more calories, total carbohydrates, total protein, total fat, and total sugars, but

they are also consuming significantly more fiber per 1000 kcals and less percent intake from fat. Conversely, infrequent eaters may be more apt to partake in other unhealthy behaviors. Thus, further investigation is needed into understand these subpopulations more thoroughly. Specifically analyzing sleep patterns and any dyssnchrony in diurnal rhythms, which have previously been related to increased obesity and metabolic disease risk[156]. Furthermore, the gut microbiota has been found to be critical in maintaining normal circadian rhythms, in turn disruption of the circadian rhythm results in changes in the composition of the gut microbiota and changes in the feeding and fasting patterns have been found to influence circadian rhythm[157,158]. A dysfunctional gut microbiota has also been found to be detrimental to the host's metabolism and potentially may increase obesity risk[159] and increased fiber intake has been found to be related to increased microbiota diversity and an increase in beneficial bacteria[160]. Increased fiber has also been associated with lower overweight and obesity risk in adults and children[161]. To our knowledge, no study has investigated the potential impact of eating frequency on the gut microbiota or the mediating effect that eating frequency may have on fiber intake and circadian rhythm and future studies further examining this relationship are warranted.

There are a several potential limitations in the current analysis. The first is that this is a cross-sectional study and thus causation cannot be assessed. Additionally, this analysis included normal, overweight, and obese subjects and our prior eating frequency research has explored this relationship in only overweight and obese Hispanic youth. However, we chose to include all weight categories given that no other group has

investigated the dietary habits and obesity risk of Hispanic college freshmen. Another limitation is that this sample may not be representative of other Hispanic college populations, as only 28% of our sample were overweight or obese, which is lower than national prevalence rates for this age range and ethnicity/race. In addition, UT-Austin has consistently been ranked in national polls as one of the healthiest colleges in the nation[144,145] and the current population was extremely active and participants on average spent more than 60 minutes in MVPA per day. But regardless, increased eating frequency was linked to reduced adiposity, even within this fairly healthy population. Thus, the current findings may not be universal among all Hispanic youth populations, although our previous work has found similar relationships in younger overweight and obese minority youth.

In summary, eating less than three eating occasions per day is associated with increased BMI, BMI z-scores, body fat percentage, visceral and subcutaneous adipose tissue, despite being linked to decreased daily energy intake in a population of Hispanic college freshmen. Given that the first year of college is such a pivotal time in the development of lifelong habits it is important to identify nutrition behaviors that may potentially reduce the risk of obesity later in life. These results as well as previous findings from our group and others support that further longitudinal trials are needed to investigate the potential causal relationship between eating frequency and obesity risk in Hispanic youth, as well as establish the need for intervention work in this area.

Chapter 7: Conclusions and Implications

To our knowledge, these are the first and only analyses to examine the relationship between eating frequency and dietary and adiposity measures in Hispanic youth and young adults. The first two analyses included two separate cohorts of overweight or obese minority youth and found very similar results. These studies found that frequent eaters compared to infrequent iaters consumed more calories per day, consumed less calories per EO, yet exhibited lower adiposity measures and reduced metabolic disease risk, while showing no significant difference in physical activity measures. Furthermore, in a large cross-sectional study with 709 Hispanic college freshmen that completed a questionnaire to identify eating frequency, physical activity levels, and reported height and weight, we found eating frequency to be positively related to moderate to vigorous physical activity (MVPA), but not overweight or obesity prevalence. Also, those who ate 4 or more times per day were 2.5 times more likely to spend at least 150 minutes in MVPA per week compared to those who ate 2 or less times per day. Finally, in sample of 92 Hispanic college freshmen we found that frequent eaters compared to infrequent eaters consumed more kcals per day, less calories per EO, and yet again had lower adiposity measures, while showing no significant difference in physical activity measures.

There are numerous potential mechanisms to explain our findings. The first being that increased eating frequency has consistently been related to increased satiety measures in adult populations[52,61,63,64]. It is reasonable to infer that increased satiety

observed in regards to increased eating frequency may reduce the motivation to eat and therefore reduce energy intake overall, however our findings consistently show a positive relationship between eating frequency and caloric intake which is in agreement with the vast majority of eating frequency research. It is also plausible that reduced eating frequency may result in eating behaviors that resemble binge eating which has been related to increases in metabolic disease parameters and adiposity measures[66]. Thus, much more research is needed to examine the exact mechanisms of how eating patterns impact satiety, hunger, and ad libitum dietary intake in free-living populations.

Another potential mechanism to consider is how eating frequency impacts metabolic rates. This important as popular media has consistently advocated more frequent eating or grazing as a healthy habit that may "stoke" or "rev" one's metabolism. A recent review of popular media sources found the ideal eating frequency recommendation given was 6 EOs per day[67]. Yet, not a single study to date has found a statistically significant difference in total energy expenditure between 1, 2, 3, 5, 6 or even 7 EOs in a 24-hour period[52-56]. Given the pervasiveness of the current media recommendation further research into the potential impacts of eating frequency on obesity and metabolic disease risk is warranted to better inform the general public of the potential benefits of frequent eating or deleterious outcomes of infrequent eating and the mechanisms behind these findings.

Another possible mechanism involves lipid metabolism. Our previous studies have found infrequent eating to be associated with higher circulating triglycerides[1,2]. Infrequent eaters also consistently show an increased caloric intake per EO, and binge

eating behaviors have been previously linked to increased triglycerides[1,66]. We have also found that infrequent eaters have increased visceral adipose tissue[1] and the accumulation of visceral adipose tissue has been positively associated with fasting insulin and triglycerides[1,120]. It is also hypothesized that visceral fat increases hepatic portal free fatty acid concentrations, which in turn are stored as triglycerides, stimulate hepatic gluconeogenesis, and hinder hepatic clearance of insulin, thus promoting a vicious cycle of hyperinsulinemia, elevated plasma glucose concentrations, and dyslipidemia[121]. Kelley and Mandrino termed this vicious cycle metabolic inflexibility[162-167]. Metabolic inflexibility has been defined as the impaired ability to transition between fatty acid and glucose metabolism and this inability leads to the accumulation of visceral adipose tissue [168,169]. Interestingly, African Americans have been found to be more metabolically flexible than their Caucasian counterparts, but to our knowledge no research has investigated metabolic flexibility in any Hispanic population[170]. Metabolic inflexibility is higher in obese adolescents and those with a family history of type II diabetes[169], therefore it is critical to investigate if Hispanic youth in general are more metabolically inflexible and thus at further obesity risk if they eat less often given this propensity to store excess fuel as visceral adipose tissue. The current analyses have consistently found that infrequent eaters have higher visceral adipose tissue, increased fasting triglycerides, and reduced insulin sensitivity when compared to frequent eaters. Thus, more research especially longitudinal studies analyzing the possible causal relationship between visceral adiposity, triglycerides, eating frequency, and metabolic disease risk is merited.

Another potential explanation for the current findings is that healthy behaviors tend to cluster together[150]. In the study of 92 Hispanic college freshmen, frequent eaters not only ate more often, but they are consumed more fiber and less fat. Conversely, infrequent eaters may be more apt to partake in other unhealthy behaviors. Thus, further investigation is needed to understand these subpopulations more thoroughly. Specifically analyzing sleep patterns and any dyssnchrony in diurnal rhythms, which have previously been related to increased obesity and metabolic disease risk. Furthermore, the gut microbiota has been found to be critical in maintaining normal circadian rhythms, in turn disruption of the circadian rhythm results in changes in the composition of the gut microbiota and changes in the feeding and fasting patterns have been found to influence circadian rhythm[157,158]. A dysfunctional gut microbiota has also been found to be detrimental to the host's metabolism and potentially may increase obesity risk[159]. To our knowledge, no study has investigated the potential impact of eating frequency on the gut microbiota or the mediating effect eating frequency may have on circadian rhythm and future studies to further understand this relationship, especially in college students is warranted.

The combined findings suggest that increased eating frequency is related to decreased obesity and metabolic disease risk in Hispanic youth and Hispanic college freshmen, despite increases in energy intake. Additionally, in a large and exclusively Hispanic college freshmen population eating frequency was related to self-reported increases in physical activity. In each of these studies we saw that increased eating

frequency was related to healthier behaviors and outcomes even though no distinction was made on what made up these eating occasions or when they occurred. Thus, increasing eating frequency in at-risk Hispanic youth and Hispanic college students may be a viable public health message. Yet, more research is needed to understand the potential mechanisms and investigate any causal relationships, as well as identify if increased eating frequency is a driver of other healthy behaviors. Encouraging Hispanic youth to eat more often is a relatively modest lifestyle change with potentially large implications on obesity and metabolic disease risk in a population where more than three quarters of adults are overweight or obese and half will become type two diabetics [125,171].

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