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

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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.

Keywords

Eating Behaviors; Life Styles; Minorities; Visceral Fat; Triglyceride

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Conflict of Interest Statement:

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 and JD had primary responsibility for the final content presented. All authors read and approved the final manuscript. There are no identified financial conflicts of interest for any author in regards to this manuscript or its content.

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 (1, 2). Currently, 16.9% of children in the United States are obese and 31.8% are either overweight or obese (3, 4). 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 (4). Minority youth have also been shown to be more apt to eat less often, but eating frequency patterns in these populations remain largely unknown (5). 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 (2, 6).

Research on the association between eating frequency and adiposity in youth populations has more consistently shown an inverse relationship (7–9), although some studies have found this inverse relationship to be mitigated by or related to physical activity (PA) (9, 10). While other studies have shown eating frequency to have no relation to adiposity measures (5, 11), or even a positive relationship (12). 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 (7). 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 and Design

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 (13–17). 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 ≥ 85th percentile for age and gender based on Center for Disease Control and Prevention guidelines

(18), (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 (19, 20). 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) (18). Fat mass and total lean mass (n=155) were measured by dualenergy 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 (14). 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 (21).

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 (22). 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 (23). 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 (24) 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 (24).

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 (25). The methodology for categorizing sedentary behavior (SED) and moderate and vigorous PA are previously described (26). 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 (14–16). 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 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 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.

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. 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 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).

Dietary and PA variables between MS and NFE are depicted in TABLE 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.

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 (4) and are more likely to skip meals and eat less often (5), 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 (8, 9) 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 (27). 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 (28). Zerva et al. (9) 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 (9, 10) and some studies have shown this to have a mitigating effect on the relationship between eating frequency and adiposity (10), while others have not (8, 9). 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 (29). 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 (30, 31). Eating frequency has been related to satiety measures, in a cross-over controlled feeding study by Leidy et al. (32) 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 (33). 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 (32). 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 (34).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 (35), as well as in youth (36). 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. (37) 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 (38), 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 (39, 40), 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 (40). 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 (40). 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 (7–9). 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.

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Abbreviations

AIR	Acute Insulin Response
BMI	Body Mass Index
DI	Disposition Index
DXA	Dual-Energy X-ray Absorptiometry
EO	Eating Occasion
HOMA-IR	Homeostatic Model Assessment: Insulin Resistance
HDL	High Density Lipoprotein
HFF	Hepatic Fat Fraction
LDL	Low Density Lipoprotein
MET	Metabolic Equivalents of Task
MS	Meal Skippers
MRI	Magnetic Resonance Imaging
MVPA	Moderate to Vigorous Physical Activity
NDS-R	Nutrition Data System for Research
NFE	Normal/Frequent Eaters
PA	Physical Activity
SI	Insulin Sensitivity
SAT	Subcutaneous Adipose Tissue
SED	Sedentary Behavior
VAT	Visceral Adipose Tissue

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What is known about this subject?

(**1**) Minority youth are at increased risk of obesity compared to non-Hispanic whites. (**2**) Minority youth have been shown to be more apt to eat less often. (**3**) Eating frequency and adiposity in youth populations has more consistently shown an inverse relationship.

What this study adds?

(**1**) This is first eating frequency analysis in a combined sample of Hispanic and African American youth. (**2**) This is the first analysis in any population to examine the relationship between eating frequency and specific fat distribution attained via magnetic resonance imaging. (**3**) We show meal skipping to be related to higher visceral adipose tissue and elevated triglycerides in a sample of overweight minority youth.

TABLE 1

Subject Characteristics [1,2]

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

TABLE 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)				
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 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

[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,

TABLE 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).