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EATING RATE, FASTING GHRELIN CONCENTRATIONS AND WEIGHT STATUS IN FEMALE COLLEGE STUDENTS AT THE UNIVERSITY OF RHODE ISLAND

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EATING RATE, FASTING GHRELIN CONCENTRATIONS AND WEIGHT

STATUS IN FEMALE COLLEGE STUDENTS AT THE

UNIVERSITY OF RHODE ISLAND

BY

STEPHANIE E. PERRUZZA

A THESIS IN PARTIAL FULFILLMENT OF THE

REQUIREMENTS FOR THE DEGREE OF

MASTERS OF SCIENCE

IN

NUTRITION AND FOOD SCIENCES

UNIVERSITY OF RHODE ISLAND

MASTER OF SCIENCE THESIS

OF

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UNIVERSITY OF RHODE ISLAND 2011

ABSTRACT

Obesity is a preventable cause of morbidity and mortality and affects over one-third of the American adult population. Eating rate and ghrelin concentrations are two variables that may play a role in obesity. An ancillary study of female college students examined the differences in eating rate (grams/second) and fasting plasma ghrelin concentrations (pg/mL) between weight status categories (normal weight body mass index (BMI) = $18.5-24.9 \text{ kg/m}^2$, overweight BMI = $25-29.9 \text{ kg/m}^2$ and obese $BMI \ge 30 \text{ kg/m}^2$). Anthropometrics, clinical (blood pressure), and biochemical (glucose and ghrelin) data were assessed. Dietary information was obtained from three non-consecutive 24-hour recalls using the multiple pass method in conjunction with the Nutrition Data System for Research. Physical activity was assessed using the International Physical Activity Questionnaire Short Form. Primary outcomes were assessed using a one-way analysis of variance. Mean age, BMI and waist circumference for the 78 participants were 18.8 ± 0.9 years, 27.9 ± 5.5 kg/m², and 87.4 ± 13.9 inches, respectively. Mean eating rate and ghrelin concentrations were 0.26±0.13 grams (gms)/second and 216.2±269.9 pg/mL, respectively. Mean energy intake, food intake and physical activity were 1,731.9±507.1 kcals, 6,365.2±2,590.5 gms, and 2,542.6±2,107.5 MET-minutes/week, respectively. Mean energy density was 1.25±0.3 kcal/gm. There were no differences (p=0.49) in eating rate across weight status categories. Ghrelin concentrations were significantly different between overweight and obese groups; 217.1±106.6 vs. 150.1±77.4 pg/mL (p<0.05). Future studies in the college-aged population are warranted to determine the nature of these relationships in providing a basis for future studies in targets for weight maintenance and energy homeostasis.

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PREFACE

This thesis was written to comply with the University of Rhode Island graduate school Manuscript Thesis Format. This thesis contains one manuscript: *Eating rate, fasting ghrelin concentrations and weight status in female college students from the University of Rhode Island*. This manuscript has been written in a form suitable for publication in the *Obesity* journal.

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CHAPTER 1

Eating Rate, Fasting Ghrelin Concentrations and Weight Status in Female

College Students at the University of Rhode Island

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Abstract:

Obesity is a preventable cause of morbidity and mortality and affects over one-third of the American adult population. Eating rate and ghrelin concentrations are two variables that may play a role in obesity. An ancillary study of female college students examined the differences in eating rate (grams/second) and fasting plasma ghrelin concentrations (pg/mL) between weight status categories (normal weight body mass index (BMI) = $18.5-24.9 \text{ kg/m}^2$, overweight BMI = $25-29.9 \text{ kg/m}^2$ and obese $BMI \ge 30 \text{ kg/m}^2$). Anthropometrics, clinical (blood pressure), and biochemical (glucose and ghrelin) data were assessed. Dietary information was obtained from three non-consecutive 24-hour recalls using the multiple pass method in conjunction with the Nutrition Data System for Research. Physical activity was assessed using the International Physical Activity Questionnaire Short Form. Primary outcomes were assessed using a one-way analysis of variance. Mean age, BMI and waist circumference for the 78 participants were 18.8 ± 0.9 years, 27.9 ± 5.5 kg/m², and 87.4 ± 13.9 inches, respectively. Mean eating rate and ghrelin concentrations were 0.26±0.13 grams (gms)/second and 216.2±269.9 pg/mL, respectively. Mean energy intake, food intake and physical activity were 1,731.9±507.1 kcals, 6,365.2±2,590.5 gms, and 2,542.6±2,107.5 MET-minutes/week, respectively. Mean energy density was 1.25±0.3 kcal/gm. There were no differences (p=0.49) in eating rate across weight status categories. Ghrelin concentrations were significantly different between overweight and obese groups; 217.1±106.6 vs. 150.1±77.4 pg/mL (p<0.05). Future studies in the college-aged population are warranted to determine the nature of these relationships in providing a basis for future studies in targets for weight maintenance and energy homeostasis.

Introduction

Obesity and overweight are preventable causes of morbidity and mortality and affect over two-thirds of the American adult population (1). College students, traditionally between the ages of 18 and 24, are a population of interest as they experience the most rapid weight gain (2) with a mean weight gain of 0.73 lbs/month, a rate 36 times faster than same-aged non-college females (3). Female college students gain more weight in less time than their male counterparts (4), from 2.2 pounds in the first six months to 9.2 pounds in the first year (2, 5) due to lifestyle changes that affect diet and physical activity, when transitioning from high school to college (6). Dietary and physiological factors that may be associated with increased female weight status are still unclear and research is limited looking at free-living female populations.

One factor associated with weight status is the appetite-stimulating hormone ghrelin; as ghrelin shifts with changes in weight and controls appetite. To date, ghrelin is the first and only peripheral hormone to display increasing orexigenic, or appetite stimulating, effects through the hypothalamic appetite-regulating pathway (7), which has advanced research related to its impact on appetite enhancement and energy homeostasis. Ghrelin is highly associated with body weight regulation; concentrations increase before and decrease after food intake, proportional to calories and macronutrients consumed (8). Also, obese individuals are shown to have lower fasting ghrelin concentrations than leaner individuals, suggesting that weight status impacts ghrelin (9). Cummings *et al.* (9) observed mean area under the curve for ghrelin increased 24% (p = 0.006) with a mean weight loss of 17.4±1.5% from baseline following a 6-month weight loss program in obese participants. Similarly, Hansen *et*

al. (10) noted fasting ghrelin in obese individuals increased 12% (114±17 fmol/ml vs. 128±16 fmol/ml, p<0.01) following a 6-month weight loss intervention class. Obese individuals who lose even 5% of their body weight significantly increase circulating ghrelin (11). Exploring the effects of weight status on ghrelin concentrations more closely is essential to better understand its physiological control of feeding, which may lead to its therapeutic possibilities related to maintaining body weight. Likewise, exploring ghrelin's relationship with variables that may influence weight status, such as eating behaviors, may be important for weight management recommendations.

Eating rate can be defined as the amount of food consumed (gms) within a certain amount of time (seconds) (12). Eating rate could affect energy intake because adopting a slower eating rate may enhance satiety by allowing gastrointestinal peptides to register meal satiation (13). Research shows obese individuals take larger and faster bites than lean individuals, possibly contributing to overconsumption and increased body weight (14).

Though ghrelin and eating rate have been examined individually in relation to weight status, there is little evidence focusing on these two variables together (15, 16). Sobki *et al.* (15) reported participants with a slow eating rate had persistent elevations in ghrelin concentrations compared to those with a normal eating speed. However, Kokkinos *et al.* (16) reported no significant differences (p=0.3) in ghrelin concentrations after the same test meal was consumed at a two different meal durations. These two studies on eating rate and ghrelin have mostly been observed in controlled settings; however, there is no focus in free-living individuals. More exploratory research is needed to determine differences among weight status, ghrelin

and eating rate to provide novel insights in attenuating unhealthy weight gain in young adult college population.

The purpose of this study is to examine the differences in eating rate and ghrelin concentrations between weight status categories in free-living female college students (normal weight body mass index (BMI) = $18.5-24.9 \text{ kg/m}^2$, overweight BMI = $25-29.9 \text{ kg/m}^2$ and obese BMI $\geq 30 \text{ kg/m}^2$). Secondary aims were to examine the relationships between ghrelin and eating rate, as well a relationship between eating rate and energy density of meals. Exploratory research for effects of weights status on total daily eating time, energy and food intake as well the relationship between ghrelin and IPAQ-S scores will also be determined. Based on previous research, we hypothesize that obese college females will have a faster eating rate, as well as lower ghrelin concentrations, than overweight and normal weight college females.

Methods and Procedures

Study Design

This research is a cross-sectional ancillary study examining the relationships among weight status, ghrelin concentrations, and eating rate. Data for the present study were obtained from two other studies performed at the University of Rhode Island (URI): the Best Food Forward (BFF) and the Nutritional Assessment and Chronic Disease Risk Factor Identification (Hatch) studies. The BFF study was a randomized clinical trial and examined the effectiveness of a self-taught intervention versus an instructed intervention on coronary heart disease (CHD) risk in overweight/obese females at URI, however only baseline data were used in this study. The Hatch study was cross-sectional and determined the prevalence of metabolic

syndrome and CHD risk factors and their relationship to dietary intake and physical activity in first year URI students.

Participants

Seventy-eight female participants were analyzed for this study (26 normal weight, 26 overweight, and 26 obese). Prior to each of the studies, participants from each study read and signed the appropriate informed consent; both studies were approved by URI's Institutional Review Board. Participants were recruited via flyers posted around campus and in residence halls, student newspaper advertisements, presentations made in introductory science classes, and booths at the student union, job fairs and dining halls. Eligibility criteria included not being pregnant or lactating, having no diagnoses of CHD, diabetes, liver disease, kidney disease, bleeding disorders, not taking any lipid-lowering medications or having a history of an eating disorder. The BFF study needed to have a BMI between 25-39.9 kg/m² while the Hatch study had no BMI parameters.

Anthropometrics

All anthropometric measures were conducted in duplicate. Height was measured to the nearest 0.2 cm using a 220 stadiometer (Seca Corporation, Hamburg, Germany) and waist circumference was measured to the nearest 0.2 cm using a Gulick non-stretchable tape measure with an attached tensometer at the top of the iliac crest upon exhalation (Patterson Medical, Mouth Joy, PA). For the Hatch study, weight was measured using a calibrated digital Seca 769 scale (Seca Corporation, Hamburg, Germany) to the nearest 0.2 kg; for the BFF study, weight was measured to the nearest 0.1 kg using a calibrated digital read scale (Bod Pod, Version 2.14, Body Composition System; Life Measurement Instruments, Concord, CA). Height and weight were used to calculate participant BMI (weight in kilograms/height in meters²).

Biochemical

A trained phlebotomist conducted two 12-hour fasted venous blood draws on two nonconsecutive morning visits in the same week with each participant. Samples were processed according to the following protocol (17). Briefly, plasma was obtained from whole blood via centrifugation (Eppendorf Centrifuge 5810, Germany) for 20 minutes at 2200 RPM's at 4°C. The following preservation cocktail was added to the plasma samples: 0.01g/100g of phenylmethylsulfonyl fluoride (Roche, Indianapolis, IN), 0.01g/100g of sodium azide (Fisher, Fairlawn, NJ) and 0.05g/100g of aprotinin (Fisher, Fairlawn, NJ) samples were aliquoted into separate 500µL aliquots and were then stored at -80° until analyses.

Enzymatic assays were performed on glucose (GLC) concentrations were analyzed using a Wako Glucose Autokit (Wako Diagnostics, Richmond, VA) (18), and read in a Biotek ELX 808 plate reader (Biotek, Winooski, VT). Fasting ghrelin concentrations were analyzed using an enzyme-linked immunosorbent assay (ELISA) kit (manufactured by SPI-Bio, Montigny le Bretonneux, France and distributed by ALPCO Diagnostics, Salem, NH).

Clinical

Resting blood pressure (BP) was measured by a trained exercise physiologist after a five-minute seated rested period using a stethoscope (Littman, St. Paul, MN) an appropriately sized BP cuff (Fisher Scientific, Fairlawn, NJ and Welch-Allyn, Skaneateles Falls, NY). Blood pressure was measured in duplicate unless variance between measures exceeded the standard (two mmHg), in which case the measurement was repeated. The average of the two readings within the standard was used for data analysis.

Dietary and Physical Activity

Data collected from three 24 hour recalls (24HR) from each participant, using NDSR version 2007, 2008, and 2009 software (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN), were used to calculate a mean eating rate for each participant. The differences between the NDSR versions only pertains the food database that is updated with more food choices each year. All study participants completed three 24HR recalls: one in-person and two over the phone, collected by trained study personnel. Recalls were collected on three non-consecutive days (two weekdays and one weekend day); this method has been shown to provide a good estimate of an individual's usual intake (19). During the face-to-face interview, Nasco food models (eNASCO, Fort Atkinson, WI) were used to assist with portion size estimation. Participants also received food amount booklets designed by the Nutrition Coordinating Center at the University of Minnesota at the first 24HR, which they kept for the additional 24HR's. The food amounts booklets help participants estimate more accurate portion sizes.

Eating rate was expressed as grams of food consumed per second and included all foods, and only beverages that provide \geq 5 kilocalories per 100 grams (20). In addition to the NDSR prompted questions, there were few questions asked, one of which included "How long did you spend eating this meal?" That information was collected in minutes/meal, which then was computed to seconds/meal. The total number of grams of food and seconds of meal consumption time were expressed as means per day. The means of grams of food consumed and seconds of meal

consumption time were then averaged for a total mean of the three 24HRs. Those two means were used to calculate grams of food consumed per second.

Energy density included meals and snacks; including beverages containing \geq 5 kcals/100 grams (20). Energy density was calculated using average energy intake of the meal or snack (including beverages) in kcals divided by total food intake of the meal in grams, and expressed in kcals/gram. The total number of kcals and grams were expressed as means per day. The means of kcals of energy intake and grams of food intake were then averaged for a total mean of the three 24HRs. Those two means were used to calculate kcals of energy intake per gram of food, which is how energy density was defined.

Physical activity was measured using the International Physical Activity Questionnaire Short Form (IPAQ-S) and was scored based on the Guidelines for Data Processing and Analysis of the International Physical Activity Questionnaire (21). This tool has been validated in young adults to reflect habitual physical activity levels (21). Participants were asked to report their frequency (days per week) and duration (time in minutes) of low, moderate, and vigorous physical activity over the past seven days. Physical activity is expressed in metabolic equivalents (METs) in METs/minute/week (METs-min/wk); METs-min is the product of the MET score of an activity multiplied by the minutes performed.

Statistical Analysis:

One-way analyses of variance (ANOVA) for eating rate required 78 participants to achieve a power of 0.8 with a large effect size f=0.4; an effect similar to that found by Laessle *et al* (2007), with an expected difference of 1.2 grams/second between normal weight and overweight/obese groups. In order to have a total of 78 participants, 26 will be needed for each weight status classification: normal weight, overweight, and obese. Significance was set at α =0.025 by applying the Bonferroni correction to account for power calculations.

Data analyses were performed using the Statistical Package for the Social Sciences (SPSS) for Windows 19.0 (IBM Corp., Somers, NY). Normality was assessed by examining skewness and kurtosis. Outliers that were greater than three standard deviations from the mean were removed for TAG (n=1), IPAQ-S (n=2), percent kilocalories from fat (n=1), total grams (n=1), meal time (n=3), eating rate (n=3), energy density (n= 1), and ghrelin (n=1). Continuous variables are displayed as means and standard deviations and categorical variables are displayed as frequencies and percentages. A one-way analysis of variance (ANOVA) determined the differences in participant descriptive characteristics across weight status categories. One way ANOVA's were also used for both primary analyses; comparing eating rate as well as ghrelin across weight status categories. Pearson's correlations were used for the secondary aims to determine the relationships between eating rate and ghrelin and eating rate and energy density. Exploratory statistics using an ANOVA examined the differences in total daily eating time, total food and energy intake across weight status categories, as well as a Pearson's correlation to determine a relationship between ghrelin and IPAQ scores. Significance was set at p<0.05.

Results

The descriptives for all 78 participants are presented as mean ± standard deviation in Table 1, and descriptives across weight status categories in Table 2. Fifty-eight participants identified themselves as white, eight (10%) as Black/African American, nine (12%) as Hispanic, and four (5%) as Other (data not shown). Mean

reported physical activity was 11,088±2,107.5 MET-minutes/week. Mean total food intake, total energy intake, and total time spent eating/day were 1,420.1±476.8 grams/day, 1,729.4±509.5 kcals/day, and 6,225.8±3,445.9 seconds/day, respectively (Table 3).

Mean eating rate was 0.29±0.21 grams/second (Table 3). There were no significant differences (p=0.62) in eating rate across weight status categories. Ghrelin concentrations were significantly higher in the overweight than the obese group, 217.1±106.6 vs. 150.1±77.4 pg/mL, respectively, with normal weight females having 30.9% higher ghrelin concentrations (Figure 1).

There was a significant inverse correlation (r=-0.385, p<0.01) between eating rate and energy density (Figure 2). The average energy density of meals and snacks consumed did not differ among the different weight status categories, p=0.97 (data not shown). The correlation was not significant (r=-0.082, p=0.48) for eating rate and ghrelin concentrations (data not shown). Mean kcal/day as well as total food intake expressed as gms/day were significantly higher in the normal weight than the obese group; 1,965.5±528.0 kcal/day vs. 1,516.8±453.9 kcal/day (p<0.05) and 1,631.6±532.1 gms/day vs. 1,257.3±419.1 gms/day (p<0.05), respectively (Table 3). No significant correlation was found between ghrelin and IPAQ-S scores (r=0.061, p=0.60) (data not shown).

Discussion

To our knowledge, this is the first study that looks at the difference among weight status, with ghrelin and eating rate in the college-aged female population. Unexpectedly, eating rate was not different across weight status categories. The observed ghrelin differences support the finding that concentrations are significantly lower in obese individuals. The data observed between eating rate and ghrelin indicated no significant relationship. Eating rate and energy density indicated a small inverse relationship which was opposite of what was anticipated.

The expected difference of 1.2 grams/second we expected to find between normal weight and overweight/obese participants was not achieved, as we found 0.03 grams/second (12). This study measured differences in a laboratory setting and only used one test meal when determining the measurement of eating rate. In the current study we reviewed dietary data from three 24-hour recalls allowing for more a variety of dietary intake and meal consumption time. The dietary data also was collected using a self-reported method so this could have been a reason why we did not see a similar difference in eating rate between groups as the Laessle *et al.* study.

It has been suggested that there is a distinctive eating style of obese individuals characterized by shorter meal duration, larger bite size and therefore rapid eating pace (22). An earlier study found that body size positively correlates to eating responses, indicating that as body size increased there was an observed increased in the number of bites, bite size and bite speed (22). Eating rate studies of obese and nonobese women in natural settings have also found non-significant findings (23, 24); however, no studies have observed this finding in self-reported dietary intake and meal duration. In earlier well-controlled test meal studies, food intake rate was measured in number of bites or chews over a certain amount of time (22). These studies, however, reported few consistent eating differences between obese and nonobese participants in distinctive eating behaviors, and their sample sizes were relatively small (25). Additional research is needed to determine the generalization of the results to other populations, such as males, as eating rate is shown to differ by gender (26).

Several factors could have contributed to the contradictory findings seen in this study between obese and non-obese individuals. First eating behavior studies have used different techniques, such as direct observation, lab measures, food diaries, self-report questionnaires, which are known to have differing validity in different populations (25). Most studies have primarily been done in research laboratories, versus in a more natural realistic setting (12, 22). Lastly, many eating rate studies focus only on one meal, thus obscuring differences between normal and obese populations that may cause variability across different times and settings and individual meal patterns. For future studies, repeated observation in a more natural environment will be beneficial in determining accurate eating styles between obese and non-obese individuals.

Interestingly, ghrelin concentrations were significantly higher in overweight than obese females, there were no significant differences in normal weight females. Ghrelin concentrations being lower in obese individuals is consistent with other studies (27, 28), and seems to indicate that ghrelin is downregulated in obese individuals. Though still unclear in research, decreased plasma ghrelin concentrations represent a physiological adaption to the positive energy balance maintained in obese individuals (29). This downregulation may be due, in part, to increased insulin and leptin, an anorexigenic or appetite suppressant hormone, concentrations; as ghrelin is inversely associated with these hormones (28). Though insulin and leptin concentrations were not examined in this study, in the future they should be reviewed to clarify their relationships with outcomes related to the pathogenesis of obesity.

There was a moderate negative correlation between eating rate and energy density; the lower the energy density the eating occasion the faster it was eaten. This

finding is still unclear in research, however, Hogenkamp *et al.*(30) found when participants ate yogurt with a low energy density, their eating rate was significantly faster versus yogurt of a higher energy density. This finding is interesting in the college-aged female population, as it is an environment to be conducive to high energy dense foods and adopting new dietary patterns. There was a small but significant positive correlation between energy density and energy intake (r=0.295, p<0.01, data not shown), which is consistent with other findings (20, 31). Exploring energy density and energy intake in populations, such as those in college, who are exposed to a variety of high energy dense foods may be a crucial step to weight management.

There were no significant differences in energy density across weight status categories. This may be partially due to the controversy with energy density in regards to beverages; beverages tend to have a lower energy density than food and can disproportionately influence energy density values (20). When using methods that account for the exclusion or inclusion of water, energy density values can be underestimated as water provides no calories but a high volume. The energy density method used in this study is used in a free-living population and found a significant positive relationship between energy density and total reported energy intake. Interpretations are highly variable and hard to compare between different studies as different methodologies are used (20, 32). Future researchers must consistently define energy density methods to better understand their variability and relation to energy intake and weight status.

Obese females reported lower energy intake than normal weight females (p<0.05). This was unexpected, however, is noted in other studies (33, 34) that underreporting of dietary intake in obese participants is a frequent challenge in

nutrition research when using self-reported dietary assessment methods (35). Obese individuals underreport their dietary intake by 20-50% (36) but little is known about how it can be prevented. According to the National Health and Nutrition Examination Survey, adults with a BMI greater than 27.3 kg/m² underreport energy intake more than adults with a lower BMI (37). Kretsch et al. (37) concluded normal weight females underreported their energy intake by an average of 282 kcals/day and overweight females by 621 kcals/day, and when expressed in percentages of required energy intake, daily needs were underreported in normal weight and overweight females by 10% and 20%, respectively. Furthermore, some studies have reported that women underreport their dietary intake to a greater extent than males (34, 38). This could explain the present study's finding of obese women reporting that they consume less energy and food intake than normal weight participants. An explanation of why this occurs is still a question that remains unanswered in research; however, many psychological and physiological factors can occur such as weight conscientiousness, self-image, and social desirability. These are all factors that should be taken into account when interpreting dietary assessment data in obese women and can be the result of inconsistent data.

Though there was no significant relationship between ghrelin and IPAQ-S scores, it is still imperative to examine due to the fact that exercise has been shown to influence energy balance and therefore influences ghrelin levels (39). Previous research has indicated that ghrelin concentrations are increased in response to a negative energy balance (9); therefore it can be hypothesized that physically fit individuals will have higher concentrations. Inverse to under-reported dietary intake, obese individuals have a tendency to overestimate their physical activity, which can

result in misinterpretation of true habitual physical activity (40). Research necessitates further exploration on the effects of exercise and more direct measures of physical activity for understanding their interactions in body weight regulation.

There were some limitations to this study. Most eating rate studies are conducted in well-controlled laboratory settings, where food intake or eating condition is manipulated or standardized and the mealtime and weight of a meal is calculated by the researcher. This study focused on eating rate in free-living occasions with reported mealtime and energy intake reliant on memory, however, we used 24-hour recalls, a method that has been shown to be very accurate. This study may also lack generalizability since it only examined college-aged females; more research is needed in larger more diverse college populations.

In spite of these limitations, this study has several strengths. First, the nature of this ancillary study allowed for this study to be on the larger side (n=78) with equal weight status categories (n=26 per group) compared to sample sizes seen in other studies conducted in this area of research. Second, data was collected during both the fall-spring semesters (96% in the 2009-2010 academic year), allowing for a full academic year of dietary patterns to be reviewed.

In summary, we examine for the first time the college age female weight status, fasting ghrelin concentrations as well as self-reported eating rate. The findings of this study conclude that ghrelin concentrations are significantly lower in obese females than nonobese females and may be a target for body weight regulation. Though eating rate did not differ between weight status categories in this study, it is still imperative to review in the future as it influences energy homeostasis, which is positively associated to weight status. Future studies in the college-aged population

are warranted to determine the nature of these relationships in providing a basis for future studies on targets for weight maintenance interventions.

Disclosure Statement

There were no conflicts of interest in this study.

Literatures Cited

 Ogden CL, Carroll MD, McDowell MA, Flegal KM. Obesity among adults in the United States--no statistically significant chance since 2003-2004. NCHS Data Brief. 2007 Nov(1):1-8.

2. Racette SB, Deusinger SS, Strube MJ, Highstein GR, Deusinger RH. Changes in weight and health behaviors from freshman through senior year of college. J Nutr Educ Behav. 2008 Jan-Feb;40(1):39-42.

3. Hovell MF, Mewborn CR, Randle Y, Fowler-Johnson S. Risk of excess weight gain in university women: a three-year community controlled analysis. Addict Behav. 1985;10(1):15-28.

 World Health Organization: Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser. 2000;894:1-253.

5. Lloyd-Richardson EE, Bailey S, Fava JL, Wing R. A prospective study of weight gain during the college freshman and sophomore years. Prev Med. 2009 Mar;48(3):256-61.

6. Butler SM, Black DR, Blue CL, Gretebeck RJ. Change in diet, physical activity, and body weight in female college freshman. Am J Health Behav. 2004 Jan-Feb;28(1):24-32.

 Nakazato M, Murakami N, Date Y, Kojima M, Matsuo H, Kangawa K, et al. A role for ghrelin in the central regulation of feeding. Nature. 2001 Jan 11;409(6817):194-8.

 Castaneda TR, Tong J, Datta R, Culler M, Tschop MH. Ghrelin in the regulation of body weight and metabolism. Front Neuroendocrinol. 2010 Jan;31(1):44-60.

Cummings DE, Weigle DS, Frayo RS, Breen PA, Ma MK, Dellinger EP, et al.
 Plasma ghrelin levels after diet-induced weight loss or gastric bypass surgery. N Engl
 J Med. 2002 May 23;346(21):1623-30.

Hansen TK, Dall R, Hosoda H, Kojima M, Kangawa K, Christiansen JS, et al.
 Weight loss increases circulating levels of ghrelin in human obesity. Clin Endocrinol (Oxf). 2002 Feb;56(2):203-6.

11. Cummings DE, Shannon MH. Roles for ghrelin in the regulation of appetite and body weight. Arch Surg. 2003 Apr;138(4):389-96.

Laessle RG, Lehrke S, Duckers S. Laboratory eating behavior in obesity.
 Appetite. 2007 Sep;49(2):399-404.

13. Martin CK, Anton SD, Walden H, Arnett C, Greenway FL, Williamson DA. Slower eating rate reduces the food intake of men, but not women: implications for behavioral weight control. Behav Res Ther. 2007 Oct;45(10):2349-59.

14. Spiegel TA. Rate of intake, bites, and chews-the interpretation of lean-obese differences. Neurosci Biobehav Rev. 2000 Mar;24(2):229-37.

15. Sobki SH, Zaid AA, Khan HA, Alhomida AS, Hilal KA, Khan SA. Significant impact of pace of eating on serum ghrelin and glucose levels. Clin Biochem. 2010 Mar;43(4-5):522-4.

16. Kokkinos A, le Roux CW, Alexiadou K, Tentolouris N, Vincent RP, KyriakiD, et al. Eating slowly increases the postprandial response of the anorexigenic guthormones, peptide YY and glucagon-like peptide-1. J Clin Endocrinol Metab.Jan;95(1):333-7.

Lofgren IE HK, West KL, Zern TL, Patalay M, Koo SI, Fernandez ML.
 Carbohydrate intake is correlated with biomarkers for coronary heart disease in a population of overweight premenopausal women. Journal of Nutrition Biochemistry. 2005;16:245-50.

Reljic R RM, Anic N, Ries B. New chromogen for assay of glucose in serum.
 Clin Chem. 1992;38:522-5.

19. Nelson M, Black AE, Morris AD, Cole TJ. Between- and within-subject variation in nutrient intake from infancy to old age: estimating the number of days required to rank dietary intakes with desired precision. Am J Clin Nutr. 1989;50:155-67.

20. Ledikwe JH, Blanck HM, Khan LK, Serdula MK, Seymour JD, Tohill BC, et al. Dietary energy density determined by eight calculation methods in a nationally representative United States population. J Nutr. 2005 Feb;135(2):273-8.

 Craig CL, Marshall AL, Sjostrom M, Bauman AE, Booth ML, Ainsworth BE, et al. International physical activity questionnaire: 12-country reliability and validity. Med Sci Sports Exerc. 2003 Aug;35(8):1381-95.

22. Hill SW, McCutcheon NB. Contributions of obesity, gender, hunger, food preference, and body size to bite size, bite speed, and rate of eating. Appetite. 1984 Jun;5(2):73-83.

23. Dodd DK, Birky HJ, Stalling RB. Eating behavior of obese and normal-weight females in a natural setting. Addict Behav. 1976;1(4):321-5.

24. Adams N, Ferguson J, Stunkard AJ, Agras S. The eating behavior of obese and nonobese women. Behav Res Ther. 1978;16(4):225-32.

25. Wing RR, Carrol C, Jeffrey RW. Repeated observation of obese and normal subjects eating in the natural environment. Addict Behav. 1978;3(3-4):191-6.

26. Martin C AS, Walden H, Arnett C, Williamson D. Does slower eating rate reduce food intake? Obes Res. 2004;12(A6):22-OR.

27. Shiiya T, Nakazato M, Mizuta M, Date Y, Mondal MS, Tanaka M, et al. Plasma ghrelin levels in lean and obese humans and the effect of glucose on ghrelin secretion. J Clin Endocrinol Metab. 2002 Jan;87(1):240-4.

Tschop M, Weyer C, Tataranni PA, Devanarayan V, Ravussin E, Heiman ML.
 Circulating ghrelin levels are decreased in human obesity. Diabetes. 2001
 Apr;50(4):707-9.

29. Klok MD, Jakobsdottir S, Drent ML. The role of leptin and ghrelin in the regulation of food intake and body weight in humans: a review. Obes Rev. 2007 Jan;8(1):21-34.

30. Hogenkamp PS, Mars M, Stafleu A, de Graaf C. Intake during repeatedexposure to low- and high-energy-dense yogurts by different means of consumption.Am J Clin Nutr. Apr;91(4):841-7.

31. Rolls BJ, Bell EA, Castellanos VH, Chow M, Pelkman CL, Thorwart ML. Energy density but not fat content of foods affected energy intake in lean and obese women. Am J Clin Nutr. 1999 May;69(5):863-71.

32. Cox DN, Mela DJ. Determination of energy density of freely selected diets: methodological issues and implications. Int J Obes Relat Metab Disord. 2000 Jan;24(1):49-54.

33. Johansson L, Solvoll K, Bjorneboe GE, Drevon CA. Under- and overreporting of energy intake related to weight status and lifestyle in a nationwide sample. Am J Clin Nutr. 1998 Aug;68(2):266-74.

34. Braam LA, Ocke MC, Bueno-de-Mesquita HB, Seidell JC. Determinants of obesity-related underreporting of energy intake. Am J Epidemiol. 1998 Jun 1;147(11):1081-6.

35. Park HA, Lee JS, Kuller LH. Underreporting of dietary intake by body mass index in premenopausal women participating in the Healthy Women Study. Nutr Res Pract. 2007 Fall;1(3):231-6.

36. Vansant G, Hulens M. The assessment of dietary habits in obese women: influence of eating behavior patterns. Eat Disord. 2006 Mar-Apr;14(2):121-9.

37. Kretsch MJ, Fong AK, Green MW. Behavioral and body size correlates of
energy intake underreporting by obese and normal-weight women. J Am Diet Assoc.
1999 Mar;99(3):300-6; quiz 7-8.

38. Smith WT, Webb KL, Heywood PF. The implications of underreporting in dietary studies. Aust J Public Health. 1994 Sep;18(3):311-4.

39. King JA, Miyashita M, Wasse LK, Stensel DJ. Influence of prolonged treadmill running on appetite, energy intake and circulating concentrations of acylated ghrelin. Appetite. 2010 Jun;54(3):492-8.

40. Lichtman SW, Pisarska K, Berman ER, Pestone M, Dowling H, Offenbacher E, et al. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. N Engl J Med. 1992 Dec 31;327(27):1893-8.

Table 1. Descriptives Characteristics of					
Participants					
	Mean ± SD				
n	78 females				
Age, years	18.8±0.9				
Height, cm	163.8±6.4				
Weight, kg	74.9±15.3				
BMI, kg/m^2	27.9±5.5				
Waist circumference, cm	87.4±13.9				
Systolic BP, mmHg	106.5±9.7				
Diastolic BP, mmHg	66.6±7.8				
GLC, mg/dL	96.7±21.4				
Ghrelin, pg/dL	216.2±269.8				
Total energy, kcals	1731.9±507.1				
Total food weight, grams	2590.5±857.9				
fat, % kcals	30.8±6.7				
carbohydrates, % kcals	53.8±8.5				
protein, % kcals	15.1±3.6				
alcohol, % kcals	0.31±1.1				
IPAQ, MET-min/week	2,542.6±2107.5				
BMI = body mass index, BP = blood pressure, TC = total cholesterol, HDL-C high density lipoprotein cholesterol, LDL-C= low density lipoprotein cholesterol, TAG = triglycerides, kcals = kilocalories, IPAQ = international physical activity questionnaire					

Table 2. Descriptive Characteristics across Weight Status Groups								
	Normal Weight (n=26)		Overweight (n=26)		Obes (n=20	e 5)		
	Mean	SD	Mean	SD	Mean	SD		
Clinical								
Age, years	18.5	0.6	19.0	0.8	18.9	1.1		
Height, cm	164.4	1.5	164.1	5.2	162.8	6.9		
Weight, kg	59.0 ^a	5.9	74.7 ^b	7.4	90.8 ^c	10.1		
BMI, kg/m^2	21.7 ^a	1.5	27.7 ^b	1.6	34.2 ^c	2.9		
WC, cm	72.7^{a}	5.1	87.7 ^b	6.6	101.7 ^c	9.5		
Systolic BP, mmHg	103.7	7.0	106.8	10.8	109.0	10.5		
Diastolic BP, mmHg	65.6	5.7	67.1	8.8	67.2	8.8		
Biochemical								
GLC, mg/dL	85.4 ^a	8.4	103.7 ^b	26.6	101.1 ^b	20.8		
Ghrelin, pg/mL	196.2 ^a	78.8	$217.1^{+a,b}$	106.6	150.1 ^{a,c}	77.4		
Dietary								
Total energy, kcals	1968.0 ^a	526.3	1707.9 ^{a,b}	453.3	1519.8 ^b	451.8		
Total energy, grams	2740.6	737.4	2513.2^{\dagger}	635.0	2369.7	828.5		
kcals fat, %	28.8	6.3	32.0	6.5	31.5^{\dagger}	7.1		
kcals carbohydrates, %	55.8	8.0	53.1	7.7	52.5	9.4		
kcals protein, %	14.7	3.1	14.8	3.6	15.9	3.9		
kcals alcohol, %	0.7	1.6	0.1	0.6	0.1	0.6		
IPAQ, MET-min/week	2672.6	1612.1	1999.1^{\dagger}	1199.5	2288.2^{\dagger}	1997.4		
[†] Removed 1 outlier, normal weight (18.5-24.9 kg/m ²), overweight (25-29.9 kg/m ²) obese (>30 kg/m ²), data were determined using an ANOVA Note: different superscripts denote significant differences at p<0.05, BMI = body mass index, WC = waist circumference, BP = blood pressure, TC = total cholesterol, LDL-C = low density lipoprotein cholesterol, HDL-C = high density lipoprotein cholesterol, TAG = triglycerides, GLC = glucose, kcals = kilocalories, SD = standard deviation, IPAQ = International Physical Activity Questionnaire								

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Table 3. Total Daily Eating Time, Intake, Energy Density and Eating Rate									
	Normal Weight		Overweight		Obese		All		
	(n=	n=26)		(n=26)		(n=26)		(n=78)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Eating occasions (#/day)	4. 1 ^a	0.9	$4.2^{a,b}$	1.1	3.4 ^c	1.0	3.9	1.0	
Food intake (grams)	1631.6 ^a	532.1	1371.5 ^{a,b}	405.9	1257.3 ^b	419.1	1420.1	476.8	
Eating time/day (seconds)	5398.4*	1828.1	6179.7 [†]	2615.9	5727.2†	3171.9	6225.8	3445.9	
Energy intake (kcals)	1965.5 ^a	528.0	1705.9 ^{a,b}	457.5	1516.8 ^b	453.8	1729.4	509.5	
Energy density (kcals/gram)	1.27	0.32	1.25	0.35	1.25	0.28	1.28	0.37	
Eating rate (grams/second)	0.28*	0.11	0.24	0.12	0.26^{\dagger}	0.17	0.29	0.21	
[†] removed 1 outlier, [‡] removed 2 outliers, normal weight (18.5-24.9 kg/m ²), overweight (25-29.9 kg/m ²), obese (>30 kg/m ²), data was determined using ANOVA Note: different superscripts denote significant differences at p<0.05									



Figure 1. Ghrelin concentrations According to Weight Status normal weight (18.5-24.9 kg/m²), overweight (25-29.9 kg/m²), obese (>30 kg/m²). Data were determined using ANOVA with Tukey's follow-up Note: different superscripts denote significant differences at p<0.05 kg = kilograms, pg = picograms


Figure 2. Eating rate Correlated to Energy Density Pearson correlation r=-0.385, p<0.01, kcals = kilograms

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APPENDICES

Appendix A: Review of Literature

Introduction:

Overweight and obesity are preventable causes of morbidity and mortality and affect over two-thirds of the American adult population^{1, 2}. Compared to lean individuals, overweight and obese individuals tend to have lower concentrations of the hunger hormone ghrelin, suggesting that weight status has a negative relationship with ghrelin³. Eating rate and weight status are positively associated with energy intake⁴⁻⁶ and excess energy intake can lead to unhealthy weight gain. College females, ages 18 to 24, are a population of interest as they experience the greatest⁷⁻⁹ and most rapid^{10, 11} increase in overweight and obesity prevalence over the past 20 years. A better understanding of how weight status impacts ghrelin and eating rate will help researchers determine how to intervene with these variables in regards to body weight regulation^{5, 12}.

Weight Gain Trends over the Lifecycle

Unhealthy Weight Gain and Associated Health Risks

Unhealthy weight gain is a concern during early childhood development, starting around age seven, as adult obesity¹³ and chronic disease risk factors originate during this period and continue to develop throughout life¹⁴. Childhood obesity has nearly tripled in the past 30 years¹⁵ and the prevalence among adolescents aged 12-19 years has increased from 5% in 1980 to 18.1% in 2008^{1, 16-}

¹⁸. These increases in obesity give rise to additional health risks factors in young adults for coronary heart disease (CHD) such as hypertension, dyslipidemia, impaired glucose tolerance, and vascular abnormalities which are already present in overweight adolescents^{19, 20}. Anthropometric measures such as elevated body mass index (BMI) (\geq 25.0 kg/m²) and increased waist circumference (>40 inches for men and >35 inches for women) predict individuals at risk for adult obesity and onset of CHD^{13, 21-23}. Guo *et al.*²² reported that the older and more overweight a child or young adult is, the higher the risk of being overweight or obese at age 35.

The increased number of overweight and obese young women (18-27 years)^{11, 24, 25} is expected to increase female obesity in 2000 to 2020, from 34 to $44\%^{26}$. This prevalence of obesity is expected to be seen in the college female population in particular compared to non-university females²⁷. Since increased BMI is positively associated with CHD risk; and more young adults are now obese than ever seen in previous decades, incidence of CHD and CHD related deaths are projected to occur more frequently in adulthood^{20, 26}. Berenson *et al.*²⁸ conducted a longitudinal observational study of cardiovascular disease (CVD) risk factors from birth to 39 years in 204 males and females. There was an increased incidence of fatty streaks in coronary arteries from 50% at ages 2-15 to 85% (p=0.01) at ages 21-39 and the amount of fatty streaks was positively correlated with BMI (r=0.48, p<0.05). Preventing unhealthy weight gain during any life stage is critical in decreasing CHD risk but young adulthood is especially important as they are more likely to see increased unhealthy weight gain. If obesity and its associated health

risks are not properly managed during childhood or adolescence they will only worsen, as the additional weight gain seen in those aged 18-29 years of age will exacerbate disease progression²¹. Generally, young adults gain, on average, slightly less than one kilogram each year, the majority of this weight gain is seen during the early twenties²⁹. Preventing unhealthy weight gain and its associated health risks in this population will help decrease the prevalence of adverse health effects³⁰. Exploring the college aged population in particular is critical as unhealthy weight gain is seen more often during this period of life.

Transition to College and Unhealthy Weight Gain

College students, traditionally between the ages of 18 and 24, are of a population of interest as they experience the most rapid weight gain⁸. Three out of ten college students are overweight (BMI 25-29.9 kg/m²) or obese (BMI \geq 30 kg/m²) as indicated by the 2005 National College Health Assessment survey³¹. Freshman females entering college gain 2.2-9.2 pounds in the first 6 months to first year^{8, 29, 32} due to changes in dietary habits, physical activity or lifestyle alterations in making the transition from home to college³³. Unhealthy weight gain during college is of great concern and female college students gain more weight and gain it faster than their male counterparts³⁴. Therefore the college female population necessitates further exploration.

Transitioning from high school to college is when many students experience a decrease in physical activity³³ which can contribute to unhealthy weight gain. However, research has mainly focused on dietary modifications for

effective weight $loss^{35, 36}$. With the limited evidence available in college students investigators feel it is the increased energy intake in conjunction with decreased physical activity that is responsible for weight gain in college students³⁷. In a longitudinal, observational study, Butler et al.³³ reported weight gain (1.5±2.3 kg p=0.01), increase in BMI (23.6 \pm 3.9 kg/m² vs. 23.9 \pm 3.9 kg/m², p=0.01), increase in fat mass and percent body fat (32.0±14.4 pounds vs. 34.9±14.8 pounds, p=0.001 and 22.0 ± 5.7 vs. 23.8 ± 5.4 , p=0.001, respectively) and a decrease in fat-free mass $(108.8\pm13.2 \text{ pounds vs. } 107.5\pm12.6 \text{ pounds, } p=0.01)$ from August to December in female college freshmen. Wengreen et al.³⁸ found 23% of participants gained \geq 5% of their body weight (4.52±1.61 kg, p<0.001) and 60.7% (p=0.05) of those participants engaged in less physical activity while in college compared to high school³⁸. Another study, by Huang *et al.*²⁵, reported that most overweight and obese college students did not meet the minimum goal of ≥ 30 minutes of physical activity per day (aerobic and strength training) as recommended in the Surgeon General's report on Physical Activity and Health³⁹. Decreased physical activity contributes to unhealthy weight gain in college along with various dietary factors.

College and Unhealthy Weight Gain

Excess energy intake and decreased physical activity contributes to unhealthy weight gain during college, as the environment is conducive to overconsumption of energy dense foods⁴⁰⁻⁴². Levitsky *et al.*⁴¹ reported 58% (p<0.05) of the total variance of weight gain from high school through the first few months of college was from increased energy intake. Factors that contributed to unhealthy weight gain include consuming breakfast, lunch and dinner at 'all-youcan-eat' facilities (20% of total variance of weight change), high-fat food consumption (12% of total variance), evening snacking (12% of total variance) and increased "junk food" consumption (8% of total variance)^{41, 43}. Edmonds *et al.*¹⁰ reported a 2.4 kg increase with a concurrent BMI increase of 0.8 kg/m² (p<0.001) in young women making the transition from high school to freshman year of college. In Vella-Zarb *et al.*'s⁴⁴ meta-analysis of predictors of college freshman weight gain, there was a mean weight gain of 3.9 ± 1.6 pounds and contributors were from recent dieting, increased energy intake, decreased physical activity and high psychological stress.

Anderson *et al.*³² measured weight status during freshman year of college and reported a mean weight gain of 1.7 kg (p<0.05) from September to May. Racette *et al.*⁴⁵ examined weight changes from freshman to sophomore year of college and reported 70% of participants who were reassessed at the end of their sophomore year gained 4.1±3.6 kg (p<0.001). Another study performed by Racette *et al.*⁸ reported the prevalence of overweight and obesity increased 23% (p<0.004) from freshman to senior year.

In a quasi-experimental community-based comparison Hovell *et al.*²⁷ found that females attending university gained a mean of 0.73 pounds/month, a rate that is 36 times faster than college-aged females not attending university (p<0.001) and university females were 2.6 to 5.2 times more likely to gain \geq 15% above their initial ideal body weight. Pliner *et al.*⁴⁶ conducted a similar study and found women had a mean rate weight gain of 0.62 pound/month representing a total of 7 pounds in one year. The factors contributing to unhealthy college weight gain need to be further explored for more effective weight gain prevention and weight regulation.

Unhealthy Weight Status and CHD risk

The importance of understanding the interrelations of weight, health risk and mortality is underscored by the increasing prevalence of obesity in the United States²⁴, especially among women⁴⁷. Owen *et al.*⁴⁸ examined the relationship between BMI before the age of 30 to CHD risk later in life and found that between 18-30 years of age a one kgm⁻² increase in BMI was positively associated with a 8% increase in the risk of CHD (RR=1.08, 95% CI 1.05-1.11). Similarly, Bibbins-Domingo *et al.*⁴⁹ determined the prevalence of adolescent overweight was projected to increase the prevalence of obesity among 35 year-old women from 32.6 to 41.6%; and resulted in a 6% increase in overall CHD events leading to excess CHD deaths by age 35-65.

Kim *et al.*⁵⁰ reported an 86% higher risk of developing CHD for females with a BMI in the highest quartile ($\geq 27.61 \text{ kg/m}^2$) compared to the lowest quartile ($< 22.34 \text{ kg/m}^2$); and risk of death increased in the highest quartile (95% CI: 1.07, 3.24). Obesity is challenging to treat, especially in females⁵¹, therefore, prevention strategies are critical. Exploring increased weight status in the young adult population may lend insight to prevent its co-morbidities, such as CHD, later in life⁵². Another factor that may be associated with increased weight status is the appetite-stimulating hormone, known as ghrelin, as ghrelin shifts with changes in

weight and control appetite. Focusing on ghrelin and its associations with body weight in the young adult population is important in regards to body weight regulation.

Ghrelin

Ghrelin – an Orexigenic Hormone

Ghrelin, a 28 amino acid peptide constituted from a 117 amino acid human prepro-ghrelin chain⁵³, was identified in 1999 as a hormone that may be involved in weight regulation by maintaining energy balance^{4, 54}. To date, ghrelin is the only peripheral hormone to display orexigenic, or appetite stimulating, effects through the hypothalamic appetite-regulating pathway, and has thus, has led to much research on its effects to enhance appetite and energy homeostasis⁴.

Ghrelin was discovered more than a decade ago, after the identification of its receptor, growth-hormone secretagogue receptor (GHS-R)⁵⁵. Growth-hormone secretagogues (GHSs) are small synthetic molecules that stimulate the release of growth hormone (GH) from the pituitary gland⁵⁶. These secretagogues work through a GHS-R, a G-protein-coupled receptor, for a ligand which was unknown at the time. It was Kojima *et al.*⁵⁶ who purified and identified the endogenous ligand specific to GHS-R, in the stomach of rats and called it 'ghrelin'.

The name ghrelin is based on its role as a growth hormone-releasing peptide and comes from the proto-Indo-European word of '*ghre*' which means grow⁵⁷. Ghrelin is primarily synthesized and secreted from the stomach mucosa, but is also secreted in other tissues such as the hypothalamus, pancreas, kidney,

and placenta⁵⁸. Ghrelin neurons have been identified in the pituitary gland, hypothalamic accurate nucleus (ARC), and in a group of neurons in the hypothalamic nuclei⁵³. There are two main molecular forms of ghrelin in humans. octanoylated ghrelin at position Ser3 on the amino acid chain and des-acyl ghrelin⁵⁹. The acylation of ghrelin occurs when the peptide undergoes a posttranslational modification in which the serine-3 position is covalently bonded to a medium-chain fatty by ghrelin O-acyltransferase (GOAT) enzyme, which converts ghrelin to its acylated or "active" form^{4, 60, 61}. The acylated form, which accounts for 20% of total circulating ghrelin, is said to be essential in the binding and activation of GHS-R1a, its classical receptor, as well as triggering GH secretion and appetite⁴. Des-acyl (non-acylated, unacylated, des-acylated), ghrelin accounts for 80% of total circulating ghrelin and though it is unable to stimulate GH secretion, it may have other biological effects such as inhibiting cell proliferation on certain cancer cells and adipogenesis^{57, 62}. Total ghrelin is des-acyl and acylated ghrelin together⁶³ and studies have looked at both total and acylated ghrelin response to food consumption and weight regulation 68,69 .

Ghrelin and Short-Term Weight Regulation

The importance of ghrelin in body weight regulation was strengthened by the finding that circulating ghrelin (total, acylated, des-acyl) concentrations are elevated during fasting and decreased after food intake⁵⁷. Ghrelin was first administered to rodents which showed a rapid increase in food intake and body weight as well as stimulating their gastric motility and acid secretion⁶⁴. Ghrelin

administration in rodents also caused a decrease in fat oxidation and increased fat mass which resulted in increased weight gain⁶⁵. Tschöp *et al.*⁶⁶ reported that ghrelin administration promoted food intake by stimulating hunger and conserving fat, resulting in increased body weight in mice and rats⁴. Similarly, the effects of intravenous infusion of ghrelin administration on appetite and food intake have been reviewed in a human cross-over study⁶⁷. The orexigenic effects of ghrelin are short-lived (half-life is 8-11 minutes), as they require a signal to govern individual meals⁶⁶. This observation led to the belief that ghrelin may act as a physiologic meal initiator that takes part in short-term energy homeostasis by increasing hunger leading to significant increased food intake (average of 28% more calories)⁵⁴. Wren *et al.*⁶⁷ investigated the effects of ghrelin administration on food intake indicating by weighing the food before and after meals and appetite sensations as measured by the visual analog scale (VAS) in non-obese males and females. Participants reported an increase hunger rating of $16\pm10\%$ (p<0.05) and an increase in energy intake by 28±3.9% (p<0.001) after ghrelin administration.

Ghrelin and Long-Term Weight Regulation

Energy homeostasis is when energy intake equals energy expenditure⁶⁸. Circulating ghrelin concentrations show compensatory responses to body weight change^{54, 69}. Ghrelin increases respiratory quotient (RQ) in rodents, suggesting reduced fat oxidation and increased glycolysis^{67, 70, 71}, which favors fat deposition. Wren *et al.*⁶⁷ saw no change in RQ in humans, however a trend towards reduced post-prandial energy expenditure was seen with ghrelin administration. Weigle *et*

*al.*⁷² determined whether participants who lose weight on a low-fat diet experience the same increase in circulating ghrelin as those participants who lose weight on a low calorie diet. Weight loss achieved on a low-fat diet failed to obtain a compensatory increase of 24-hour ghrelin profiles, as lipids were replaced with carbohydrates, which suppresses ghrelin better than lipids. This suggests that macronutrient composition play a role in ghrelin regulation, and increased dietary lipids will offset the stimulatory effect of weight loss on ghrelin^{54, 73}. The effects of ghrelin on short- and long-term weight regulation may be important in the management and prevention of obesity.

Ghrelin and Obesity

Ghrelin may regulate body weight via a negative-feedback loop suggesting that concentrations should increase with weight loss and decrease with weight gain⁵⁴. Decreased ghrelin concentrations were reported in participants as a result of being in a constant positive energy state; therefore, obese individuals have lower fasting ghrelin concentrations than leaner individuals^{4, 66, 74, 75}. It is also possible that decreased plasma ghrelin concentrations represent a physiological adaption to the positive energy balance maintained in obese individuals³. However, in mice, an over expression of GHS-R does not affect energy intake response to GHS-R expression ligands⁷⁶.

Although research is unclear about the effects of ghrelin in obesity it is still a fundamental target as ghrelin is reduced in a positive energy state. Wortley *et* $al.^{77}$ studied the effects of ghrelin deficiency on growth, development, and

metabolic effects by placing ghrelin-deficient 6-week old mice on a high-fat diet for three weeks. The ghrelin-deficient mice had less weight gain (30% lower body weight at 24 weeks, p<0.0001)⁷⁷. Zigman *et al.*⁷⁸ compared intracerebroventricular ghrelin administration in wild-type mice versus GHS-Rdeficient mice, fed a high fat diet for 19 weeks. Ghrelin did not stimulate food intake in GHS-R-deficient mice nor did it activate ARC neurons typically activated by ghrelin⁷⁸. After 8 weeks on the high-fat diet GHS-R deficient mice weighed significantly less than wild-type mice, and weight loss continued to progress such that after 19 weeks, GHS-R mice weighed 12.7% less than wildtype, $p < 0.0001^{78}$. Both of these studies suggest that a lack of ghrelin secretion results in energy conservation and a down regulation of appetite stimulation in rodents. Further research is warranted on the effects of appetite suppression as it relates to ghrelin in humans as obese individuals tend to have lower preprandial concentrations of ghrelin but postprandial ghrelin secretion is not sufficiently suppressed⁷⁹.

Ghrelin as a Potential Drug Target

Studies have started to investigate the potential therapeutic impacts of ghrelin on weight regulation and obesity^{4, 80}. Since ghrelin concentrations increase in obese individuals during weight loss, blocking ghrelin may prevent weight regain after weight loss⁵³. In addition, GHS-R antagonists may act as a potential drug target as they have high constitutive activity⁸¹. Individuals feel hungry between meals, even though ghrelin concentrations have returned to their pre-meal

concentrations at this point. If the constitutive activity of the receptor provides a signaling set-point, as suggested, in the stimulatory branch between meals, blocking the ghrelin receptor will result in lower food intake. The set point blockage will increase the sensitivity to inhibitory signals, such as leptin, insulin and PYY₃₋₃₆, and eliminate the craving for hunger⁸¹. Even with all the potential of using therapeutic drugs to treat obesity, researchers still feel medications need to be in combination with diet and physical activity modifications⁵³.

Ghrelin and Macronutrients

Gastric ghrelin expression is dependent on feeding state and macronutrient composition in the diet⁸². Ghrelin expression decreases more after a carbohydraterich compared with fat-rich meal⁸³. Some studies suggest a possible interaction between the gastric chemosensory system, which shares signaling mechanisms with gastric organs⁸⁴, and ghrelin regulation^{85, 86} but inconclusive data exists. Others have stated ghrelin is regulated post-absorption into the blood and not related to gastrointestinal sensing⁸⁷. Al Massadi *et al.*⁸⁵ investigated the direct effect of the three macronutrients in the forms of l-glutamine (protein), intralipid (fat), and glucose (carbohydrate) over gastric ghrelin secretion *in vitro* in a rat gastric explants model. Carbohydrates had a stronger inhibitory effect than fat on gastric and circulating ghrelin concentrations, and ghrelin secretion decreased with l-glutamine perfusion. They postulated, though a post-gastric mechanism

and lipids act directly on gastric ghrelin-producing cells in order to regulate ghrelin release⁸⁵.

Ghrelin suppression is diminished in obese, insulin-resistant participants, therefore, post-prandial ghrelin suppression may contribute to increased intake and obesity⁸⁸. Yang *et al.*⁸⁸ compared fasting and postprandial concentrations of ghrelin after meals with various macronutrient contents and how the macronutrient composition may contribute to obesity by affecting appetite and food intake. There was less ghrelin suppression in obese than lean individuals 30 minutes after each meal. There was also less ghrelin suppression after the high fat meal than the high carbohydrate meal. Decreased ghrelin secretion in obese participants is related to decreased satiation and overconsumption. The physiological mechanisms that modulate plasma ghrelin are not well understand, it has been shown that insulin can suppress ghrelin and glucose can regulate ghrelin independently of insulin⁸⁹. In the Yang *et al.*⁸⁸ study there was an inverse association between postprandial ghrelin and insulin concentrations in lean but not obese participants.

Ghrelin and Insulin

Ghrelin concentrations show a diurnal variation and may be influenced by age, gender, BMI, GH, leptin, glucose and insulin^{53, 64, 90}, however, the relationships between these variables are unclear⁹¹. McCowen *et al.*⁹² studied the effects of glucose and insulin infusion on ghrelin concentrations in mice and concluded ghrelin concentrations were reduced (p<0.01) with infusion of glucose

and insulin (85±2 and 103±0.6pM) versus the control (saline) infusion (163±9 pM). This suggests that ghrelin is strongly regulated by the rise insulin after a meal, which activates satiety signals of the hypothalamus. Some studies found ghrelin administration to be associated with a reduction in insulin secretion^{93, 94}, while other studies exhibited insulin infusion suppressing ghrelin concentrations⁹⁵. The mechanisms for this regulation in rodent's insulin receptors in the intestine retain signaling function, though regulation in humans remains unclear⁹⁶. It is suspected that in humans, insulin activates neural circuits that influence ghrelin release. Ghrelin stimulates glucagon secretion from isolated mouse pancreatic islets⁹⁷, so ghrelin likely blocks the inhibitory effect of insulin on gluconeogenesis demonstrated in hepatoma cells⁹⁸. These findings suggest that ghrelin may be involved in the regulation of glucose metabolism via down-regulating insulin secretion ⁹⁹.





Figure 1 shows how ghrelin signals insulin release from pancreatic β -cells. The ATP-sensitive K⁺ (K_{ATP}) channels are closed by increases in the ATP/ADP ratio following glucose metabolism; this induces membrane depolarization and an increase in cytosolic Ca^{2+} channels. This increase leads to insulin secretion by the β -cells. Ghrelin activates the β -cell GHS-R that is coupled with the G-protein $G_{\alpha i2}$ which activates K⁺ channels and consequently suppresses Ca^{2+} channels influx and suppresses insulin release.

Ghrelin concentrations are negatively associated with insulin resistance in humans¹⁰⁰, suggesting a pathophysiological role of ghrelin in the regulation of insulin release¹⁰¹. Ghrelin in obese individuals could be involved in energy and glucose metabolism in which insulin plays an important role. However, under conditions when the demand of insulin exceeds its production, such as individuals with insulin resistance, ghrelin can promote insulin secretion and prevent glucose intolerance. The function of ghrelin in regulating glucose metabolism, paired with GH release and feeding, supports that ghrelin may play an integrative role in regulation of energy homeostasis¹⁰¹.

Ghrelin and Leptin

The stimulatory effect of ghrelin on food intake has raised questions on its interaction with other appetite-related molecules, such as leptin. Leptin is an adipose tissue-derived hormone secreted in proportion to body fat which plays a crucial role in body weight regulation^{102, 103}. Receptors for leptin are expressed by hypothalamic neurons¹⁰³, such as those in the ARC, which are involved in energy balance. Leptin administration directly into the brain results in a decreased food intake and appetite^{103, 104}. Leptin reduces hypothalamic NPY mRNA expression which is reversed by action of ghrelin^{105, 106}. A competitive interaction between ghrelin and leptin is shown in *in vitro* calmodulin-dependent protein kinases (Ca²⁺)

signaling studies, where leptin inhibits a ghrelin-induced increase in Ca^{2+} in about 70% of the ARC neurons.

Leptin also significantly decreases ghrelin mRNA expression in both obese and lean mice, suggesting a downstream in gastric ghrelin by leptin¹⁰⁷. However, in one study chronic administration of leptin increased ghrelin mRNA expression in the stomach as well as decreased food intake and body weight^{103, 108}. Gastric ghrelin might reflect physiological changes with altered food intake rather in the presence of leptin. Though administration of ghrelin failed to modify concentrations of leptin, the interactions of these two hormones at the present time need to be further reviewed. It is also not clear whether abnormalities in these two hormone systems contribute to the development of obesity. Nevertheless, disturbances in their regulatory systems seems to play some sort of a role in the maintenance of obesity⁵³.



Figure 2 displays a simplified depiction of the appetite regulatory roles of leptin and ghrelin. Leptin acts as part of a feedback loop to maintain consistent fat

stores and reduces food intake by two hypothalamic pathways, both originating in the ARC and lateral hypothalamic area ¹⁰⁹. Ghrelin abruptly releases from the stomach, 20-30 minutes before a meal⁸¹, and has the opposite effect on the hypothalamus by stimulating an anabolic state and secretion of GH by acting on the anterior pituitary gland. Fasting decreases leptin and increases ghrelin, leading to an activation of the orexigenic ghrelin pathway¹⁰³.

Ghrelin and Energy Balance

The effects of ghrelin on energy balance are largely regulated through the hypothalamus, which is involved in feeding behavior regulation¹¹⁰. Korbonits *et al.*¹¹¹ hypothesized three pathways for ghrelin's satiating affects. First ghrelin may cross the blood brain barrier and bind to its receptors in the hypothalamus. Second, ghrelin will reach the brain through the vagal nerve^{112, 113}, and third, ghrelin is produced in the hypothalamus, where it may directly affect the local nuclei¹¹¹. Both central and peripheral administration of ghrelin in rats stimulates food intake through the activation of hypothalamic NPY/Y1 receptor pathway and the resulting antagonizing leptin action¹⁰⁶. Nakazato *et al.*¹⁰⁶ demonstrated decreased food intake after intracerebroventricular injection of ghrelin antiserum in rats, which further demonstrates ghrelin's biological role in energy balance.

Furthermore, obese individuals who lose even 5% of their body weight significantly increase circulating ghrelin concentrations⁵⁴. Cummings *et al.*¹¹⁴ found mean area under the ghrelin curve increased 24% (9,635±1127 pgs-day/ml to 11,585±1449 pgs-day/ml, p=0.006) with a mean weight loss of 17.4±1.5%

(p<0.001) of initial body weight. Similarly, Hansen *et al.*⁹¹ noted baseline fasting ghrelin in obese individuals increased 12% (114±17 fmol/ml vs. 128±16 fmol/ml, p<0.01) following a six month weight loss course. Fasting ghrelin was positively correlated with the extent of participant weight loss (r=0.68, p<0.05). The theory that ghrelin is involved in long-term weight regulation is supported by the fact that ghrelin concentrations adapt to changes in weight status⁵⁴.

Furthermore, the number of calories in a meal is related to the depth and duration of postprandial plasma ghrelin suppression in a dose-dependent manner when all other characteristics of a meal are held constant^{115, 116}. This suggests that consuming larger meals suppresses ghrelin and hunger more substantially than smaller meals. The magnitude of pre-prandial recovery of ghrelin concentrations correlates with the number of calories consumed in the following meal¹¹⁷. Research shows polymorphisms in the GSH-R gene are associated with alterations in eating patterns consistent with the belief that ghrelin may be a determinant for meal time¹¹⁸. A more complete understanding of ghrelin's role in energy homeostasis will help promote healthy weight loss by achieving a more negative energy balance.

Ghrelin and Energy Expenditure

Another area of research that is gaining momentum is the effect of exercise on ghrelin and energy balance. In rodent studies, ghrelin administration resulted in increased $RQ^{66, 67}$. The role of ghrelin in the regulation of energy homeostasis in humans, however, is less clear. Some studies noted the metabolic effects may

extend beyond the regulation of appetite and satiety and potentially serve as a biomarker of increased energy efficiency, meaning lower energy expenditure, in humans¹¹⁹.

St-Pierre *et al.*¹¹⁹ investigated the relationship between ghrelin and energy expenditure with resting metabolic rate (RMR) and thermic effect of food (TEF) in young women. They reported the negative correlation between ghrelin concentrations and RMR (r = -0.350, p=0.004) and TEF (r = -0.396, p=0.001) were independent of body composition. Marzullo et al.¹²⁰ explored the relationship between active and total ghrelin and resting energy expenditure and reported obese individuals with a positive energy expenditure ratio (measured/predicted energy expenditure) had higher concentrations of active ghrelin (214±22 vs. 159±30 pg/ml pmol/liter, p<0.05) than individuals with negative ratio values $(106\pm 2 \text{ vs. } 97\pm 1; \text{ p}<0.01)^{120}$. Huda *et al.*¹²¹ examined the effects of ghrelin on appetite and energy expenditure in three participant groups: lean, obese and post-gasterectomy. They reported that lean subjects postprandial RQ rose after ghrelin infusion but the rise was not significant $(0.92\pm0.03 \text{ vs.})$ 0.97 ± 0.03 , p=0.13). In contrast, obese individuals had no significant postprandial rise in RQ during the saline infusion (p=0.1) but did during the ghrelin infusion (0.86±0.01 vs. 0.93±0.03, p=0.03). However, there was a significant effect of ghrelin on displacement from baseline values compared with the saline infusion (f(8,168)=6.2, p<0.001) suggesting that peripheral ghrelin increased RQ variability in all three groups over time. Exploring the effects of weight status on ghrelin

concentrations more closely and paired with eating patterns and eating rate may benefit this unclear area of research.

CIII. Eating Rate

What is Eating Rate?

Though there is no widely accepted operational definition or validated objective measurement of eating rate, it can be defined as the amount of time it takes to consume a meal or snack. Eating rate has been measured in various ways including: grams/minute, kilocalories/minute, grams/second, bites/minute and number of bites/second $^{122-125}$. It is analyzed as a categorical and continuous variable. Eating rate may affect energy intake since a slower eating rate enhances satiety by allowing gastrointestinal peptides to register meal termination and prolong pleasurable aspects of eating⁶. This was first investigated by Spiegel *et* al.¹²⁶ who manipulated ingestion rates of a liquid diet during oral and gastric feedings and found eating rate was positively correlated with amount of food intake. Lean and obese individuals have different eating behavior microstructures, known as the basic reflexive units of general intake such as licks, chews, and swallows¹²⁷. These different behaviors have led researchers to focus on eating behavior and weight status in order to understand its role in body weight regulation^{128, 129}.

Eating Rate and Obesity

In 1962, Ferster *et al.*¹³⁰ first introduced the idea that obese individuals eat faster than lean; since then eating behaviors, particularly eating rate, gained importance as contributing factors in the progression of obesity¹³¹. Though eating behavior has been examined in naturalistic and laboratory settings, results are conflicting if obese individuals are faster eaters than lean individuals¹³². Teitelbaum and Campbell¹³³ examined eating behaviors during a liquid and solid diet in obese hyperphagic, nonobese hyperphagic and control female rats and the obese hyperphagic rats ate more than normal controls when consuming the liquid diet (45.3 mL vs. 42.7 mL) and ate faster when consuming the solid diet (5.1±0.85 vs. 4.0±1.1 pellets/minute). The increased food consumption of the hyperphagic rats is closely related to how obese they are meaning that these rats will overeat which indicates investigation on the relation of obesity and food intake.

It is unknown whether or not eating rate is an acquired eating behavior. An individual with a rapid eating rate during childhood may continue to eat rapidly as an adult¹³⁴. Otsuka *et al.*¹³¹ examined the association between self-reported eating rate and BMI as well as BMI-change from age 20 to current age (48.2±7.1 years). Female BMI was positively associated with self-reported eating rate (from very slow -1.06, relatively slow -0.35, relatively fast 0.81, and very fast 1.47 kg/m²).

Research also shows obese individuals take larger and faster bites than lean individuals¹³⁵, possibly contributing to overconsumption and increased body weight. Gaul *et al.*¹²² looked at eating rates in obese and non-obese participants during a 5-minute observational meal and reported obese participants took more

bites, chews/bite and spent less time chewing than non-obese (16.94 vs. 12.66, 9.24 vs. 18.6, respectively). Female obese participants took more bites than nonobese (16.8 ± 3.3 vs. 12.3 ± 2.5 bites, p<0.01) and spent less time chewing $(104.1\pm35.4 \text{ vs. } 153.5\pm33.9 \text{ seconds}, p<0.01)$. Similarly, Hill *et al.*¹²³ reported that obese male participants consumed more grams per second than non-obese during a dinner meal (0.95 vs. 0.73, p<0.05), partially due to slightly larger bites (7.2 vs. 6.2 grams/bite) and faster chewing or less chewing per bite (7.9 vs. 8.7 seconds/bite). Another study performed by Hill et al.¹³⁶ reported that as weight status increased bite size $(7.1\pm2.3 \text{ vs. } 6.3\pm2.0)$ and eating rate $(0.4\pm0.2 \text{ vs. } 0.4\pm0.1)$ increased, resulting in a faster eating time (286.2±108.8 vs. 329.2±133.8 seconds). Women took more bites (20.7 ± 6.6 vs. 15.8 ± 10.4 , p<0.05), more time to eat $(352.8 \pm 121.7 \text{ vs. } 290.7 \pm 128.3, \text{ p} < 0.05)$ and ate fewer grams per second (0.3 ± 0.1) vs. 0.4 ± 0.2 , p<0.05) than men. Eating rate and energy intake are mostly measured in a controlled lab setting, but research examining these variables in a more naturalistic setting is limited.

Obesity and Underreporting of Energy Intake

Underreporting of dietary intake is a frequent challenge in nutrition research when using self-reported dietary assessment methods¹³⁷. Goldberg *et al.*¹³⁸ suggested that reported energy intake (EI) can be used to evaluate reported EI against presumed energy requirement. Energy intake is expressed as multiples of estimated basal metabolic rate (BMR), and the ratio of EI/BMR; indicates energy reporting status. Obesity has been the common denominator with low

EI:BMR ratios, as a reported risk factor for underreporting¹³⁷. Obese individuals underreport their dietary intake by 20-50%¹³⁹ but little is known about how it can be prevented¹⁴⁰. According to the 1994 National Health and Nutrition Examination Survey, females^{141, 142}, as well as adults with a BMI greater than 27.3kg/m² underreport energy intake more than adults with a lower BMI¹⁴³.

Johansson *et al.*¹⁴⁴ found that underreporting of EI is more common in younger individuals (16-29 years, p<0.001) compared to older individuals. Percentages of overreporting were larger in males than females (15% compared to 29%)¹⁴⁴. Females that were underreported according to (EI:BMR of <1.14) accounted for 25% of the female sample; of these about 46% had a BMI of 25-35kg/m². Braam *et al.*¹⁴² also explored what predicts dietary underreporting and found each increase in BMI by 1 kg/m² was associated with a decrease in energy ratio in women (β = -0.0262). Age was also another independent determinant of underreporting, with energy ratios being the lowest among men and women older than 40 years. These studies suggest that the degree of obesity will influence dietary reporting quantitatively and qualitatively.

Eating Rate and Energy Intake

Duncan *et al.*¹⁴⁵ examined the effects of high energy density (HED) and low energy density (LED) diets on energy intake, satiety and eating time in obese and non-obese individuals. They speculated that longer meal duration and mastication time on a LED diet and eating to the point of comfortable versus an uncomfortable point on a HED diet, resulted in a lower energy intake (LED

1,570±290 kcal/day vs. HED 3,000±460 kcal/day, p<0.01)¹⁴⁵. Similarly, Andrade *et al.*¹²⁴ reported that eaters in the slow with-in meal eating condition consumed less energy compared to eaters in the quick condition (579.0±154.7 kcal vs. 645.7±155.9 kcal; p<0.05); but satiety for eaters in the quick condition remained lower at meal completion. Small bite sizes and pausing between bites resulting in a slower eating rate was shown to decrease the rate of ingestion, enhance satiety and energy intake. Eating slower may leave more time to consume water, creating distention, satiety and decreased energy intake; such as in this study as more water was consumed under the slow condition¹²⁴. Sasaki *et al.*⁵ reported a positive association between body weight, BMI and eating rate. Energy intake also increased with increased eating rate (8,304 kJ/day vs. 7,286 kJ/day, p<0.001).

CIV. Ghrelin and Eating Rate

Current Research on Ghrelin and Eating Rate

Though ghrelin and eating rate have received more attention recently, few studies have examined the effects these two variables together. Both, eating rate and ghrelin impact energy homeostasis which is positively related to weight status. Postprandial concentrations of ghrelin and other appetite-related hormones have minimally been examined in the context of different eating rates^{146, 147}. However, the mechanisms between these two variables are unclear and warrant further research.

Eating Rate and Ghrelin

Zijlstra et al.¹⁴⁸ reported desacyl ghrelin concentrations were higher after consumption of a semi-solid meal (p=0.004), and observed a greater and more prolonged reduction of total ghrelin after the solid meal. Sobki et al.¹⁴⁶ reported participants with a slow eating speed had persistent elevations in ghrelin concentrations (887.2±21.9 pg/ml vs. 827.5±16.5 pg/ml, p<0.01) compared to those with normal eating speed. Ghrelin concentrations were elevated at fasting, decreased after meal initiation and rose during the late postprandial period, suggesting that ghrelin is a signal for meal initiation and concentrations rise more steadily while eating at a normal pace. However, Kokkinos *et al.*¹⁴⁷ reported no difference in ghrelin area under the curve values after a 5 minute versus a 30 minute meal (29,133.1±11,392.1 pmol/liter • min vs. 136,304.2±12,619.6 pmol/liter \cdot min, p=0.3), though there was a trend for lower ghrelin at the 120 minute time point for the 30 minute meal duration. These results suggest that eating rate may not influence or exigenic ghrelin, as much as anorexigenic hormones. The inconsistent findings between eating rate and ghrelin concentrations are the reason for more research, especially when comparing to different weight status categories. Future studies are needed to explore the associations between ghrelin and eating rate to help reverse this universal trend towards increased weight gain in young adults and reduce the risk of health complications later on in life.

CV. Eating Rate and Energy Density

Energy Density

The roles that eating rate and energy density play in weight status have not been thoroughly investigated. A variable known to influence weight status and short-term energy intake is energy density (kilocalories/gram)¹⁴⁹. Therefore, it may play a role in the control of energy balance as it influences food and energy intake¹⁵⁰. It is recommended that following a LED diet, which provides fewer calories per gram, is preferred for weight management. Individuals consume more energy when presented with foods with a higher energy density compared with similar foods having a lower energy density^{149, 151-153}. Research examining the relationship between energy density and weight status in free-living individuals is difficult to interpret and hard to compare in part to methodological differences.

Methods of Calculating Energy Density

Little is known about energy density in free-living individuals, dietary analysis programs do not automatically calculate energy density nor is there a standard calculation¹⁴⁹. Many methodologies have been proposed; usually foods only^{150, 154-156}, but others vary with inclusion beverages^{150, 157, 158}. The primary controversy surrounds beverages, which tend to have a lower energy density than foods and can disproportionately influence energy density values^{149, 150}. Energy density values vary between 1.36 kcal/g and 1.82 kcal/g depending on the method used.

Ledikwe *et al.*¹⁴⁹ examined eight calculations of energy density (food only; food and liquid meal replacements; food and alcohol; food and juice; food and milk; food, juice, and milk; food and energy-containing beverages; and food and all beverages excluding water). The method of food only or food and liquid meal replacements had the highest value (1.85 kcal/g) and the all food and beverages method had the lowest value (0.94 kcal/g). There was a significant inverse linear trend for age with energy density values: the youngest group 20-29 year olds (2.02 kcal/g) and the oldest group \geq 70 years (1.61 kcal/g). A few conclusions could be drawn from this study regarding the variations in energy density calculations, the first is if beverages are included in the calculation they need to be clearly defined. Second, energy density values may not provide meaningful measures in food and all beverages excluding water methods, because individuals who drink a lot of water will have a higher energy density values.

Cox *et al.*¹⁵⁰ examined six different methods of energy density (all foods and all beverages; all foods and all energy-containing beverages; food and all milk; food only; all dry matter; and protein, carbohydrate, fat only) and their implications for differences in lean and obese participants as well as examining relationships between energy density and macronutrient intake. Obese participants reported a greater energy density intake compared to lean individuals under the food and all milk method, however the opposite result was found under the all dry matter method. These results have been seen in similar studies, the energy density values varied with the inclusion and exclusion of different beverages. Another implication with the concern of using different calculations was the lack of

significant correlations seen between five of the six methods related to dietary fat for the obese group. In contrast, the macronutrient only method had a high significant correlation was found with the percentage of dietary energy from fat $(r=0.56, p<0.0001)^{150}$. The conclusions drawn from energy density studies have shown there is no one method better for calculating than another. The results are widely different and interpretations are highly variable and hard to compare between different studies. Future researchers must carefully define energy density methods to better understand their variability and how they may relate to energy intake and weight status.

Energy Density, Energy Intake and Eating Rate

Consuming a HED diet results in increased energy intake^{153, 159, 160}, but whether the energy density of diet affects an individual's eating rate unclear. Duncan *et al.*¹⁴⁵ compared the effects of low and high energy dense diets on satiety and rate of energy intake in obese and nonobese participants. Eating time averaged 17 minutes or 33% longer per day on the low energy dense diet than the high energy dense diet (69 ± 14 versus 52 ± 11 min/day, p=0.0001) and there was no significant differences between obese and nonobese groups (60 ± 17 versus 61 ± 13 min/day, respectively). However, rate of energy intake on the LED diet was about one third (39%) that of the HED (23 ± 3 kcal/min versus 59 ± 9 kcal/min, p=0.0001). The prolonged eating time on the low energy dense diet might be in part to the sensation of satiety at a low energy intake¹⁴⁵. Hogenkamp *et al.*¹⁶¹ compared consumption time and eating rate with 3 groups (liquid/straw, liquid/spoon, and

semisolid/spoon) of yogurts offered at a LED and HED. Eating rate (intake in grams/min) was higher in the liquid/straw group (132±83g/min) than in the spoon groups (liquid/spoon: 106±53g/min, semisolid/spoon: 105±88g/min, p=0.01). Individuals ate more under the LED diet occasion for the semisolid meal (486±246g versus 458±204g p=0.002). Though energy intake was lower under the LED yogurt; it was eaten at a quicker rate. Therefore, energy intake of a meal may be dependent on the energy density of the foods available at a meal¹⁶¹. Exploring energy density and energy intake in populations exposed to a variety of high energy dense foods may be a crucial step in weight management.

Energy Density and College Students

Many research studies have identified college students as a population with poor and inconsistent compliance to healthy diet practices and nutrient dense foods^{40, 162, 163}. Anding *et al.*¹⁶³ reported that two-thirds (66%) of the female participants exceeded recommended levels of saturated fat and 20% exceeded cholesterol recommendations. Approximately 43% of the women complied to at least one dietary guideline recommendation, 32% adhered to at least two guidelines, but none followed all seven guidelines studied¹⁶³. This suggests that college females are not aware of the dietary guidelines or they are exposed, in the college environment, to higher fat and energy dense foods. Melby *et al.*¹⁶⁴ observed a random sample of students from a large university to assess dietary-related behaviors, knowledge and beliefs. The majority of students had an understanding of basic nutrition concepts, but 69% failed to consume fruit once a

day and 43% indicated they ate one vegetable less than once per day. There is a significant percentage of college students have unfavorable dietary habits, more research is needed to determine exactly what factors contribute to these habits. Strong et *al.*⁴⁰ sought to identify health behavior changes related to weight management in college students and reported students generally skipped breakfast, snacked on chips or sweets, consumed water, juice, sweetened beverages and when on short time (\geq 1-3 times per week) consumed fast foods. College students are introduced to a variety of energy dense foods and adapt new dietary patterns, the decline of healthful meals and snacks as part of a routine decreases from high school to college. It is important for health care professionals and researchers to identify the causes of these changes when working with the young adult college population and consider energy dense foods as a contributing factor.

CVI. Conclusion

Overweight and obesity are preventable causes of morbidity and mortality and affect over two-thirds of the American adult population ^{1, 2}. Obesity is positively correlated with increased risk of chronic diseases, the most prevalent being CHD-related deaths^{20, 48, 49}. The increased number of overweight and obese young women (18-27 years)^{11, 24, 25} is expected to increase from 34 to 44% between 2000 and 2020, ²⁶. The prevalence of obesity is seen more in the college female population compared to non-university females²⁷. The factors contributing to unhealthy college weight gain need further exploration for more effective body weight regulation.

Both eating rate and ghrelin influence energy homeostasis^{125, 165-167}, which is positively associated with weight status. Research shows obese individuals take larger and faster bites than lean individuals¹³⁵, which may possibly contribute to overconsumption and increased body weight. Circulating ghrelin concentrations show compensatory responses to body weight change^{54, 69}, suggesting it plays a role in long-term weight regulation. Postprandial concentrations of ghrelin and other appetite-related hormones are minimally examined in the context of different eating rates^{146, 147}, and the mechanisms between eating rate and ghrelin are unclear and warrant further research.

Energy density and eating rate play a role in weight status and the control of energy balance by influencing food and energy intake¹⁵⁰. Research examining the relationship between energy density and weight status is crucial because individuals consume more energy when presented with high energy dense foods compared to a low^{149, 151-153}. Excess energy intake contributes to unhealthy weight gain during college, as the environment is conducive to overconsumption of energy dense foods⁴⁰⁻⁴².

It is important to determine the relationship between weight status, eating rate and ghrelin concentrations in order to maintain a healthy body weight and energy homeostasis. Though some factors are clear on the effects of weight status, ghrelin, eating rate, and energy intake individually, there is limited research focusing on the relationships among these variables. Once a better understanding is established, researchers and healthcare professionals can provide insight on the beneficial effects to improve individuals' overall quality of life.

Literatures Cited:

 Ogden CL, Carroll MD, McDowell MA, Flegal KM. Obesity among adults in the United States--no statistically significant chance since 2003-2004. NCHS Data Brief 2007:1-8.

2. What is Obesity? Obesity Society 2010.

3. Shiiya T, Nakazato M, Mizuta M, et al. Plasma ghrelin levels in lean and obese humans and the effect of glucose on ghrelin secretion. J Clin Endocrinol Metab 2002;87:240-4.

4. Castaneda TR, Tong J, Datta R, Culler M, Tschop MH. Ghrelin in the regulation of body weight and metabolism. Front Neuroendocrinol 2010;31:44-60.

5. Sasaki S, Katagiri A, Tsuji T, Shimoda T, Amano K. Self-reported rate of eating correlates with body mass index in 18-y-old Japanese women. Int J Obes Relat Metab Disord 2003;27:1405-10.

Martin CK, Anton SD, Walden H, Arnett C, Greenway FL, Williamson
DA. Slower eating rate reduces the food intake of men, but not women:
implications for behavioral weight control. Behav Res Ther 2007;45:2349-59.

7. Malinauskas BM, Raedeke TD, Aeby VG, Smith JL, Dallas MB. Dieting practices, weight perceptions, and body composition: a comparison of normal weight, overweight, and obese college females. Nutr J 2006;5:11.

Racette SB, Deusinger SS, Strube MJ, Highstein GR, Deusinger RH.
Changes in weight and health behaviors from freshman through senior year of college. J Nutr Educ Behav 2008;40:39-42.

Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, Koplan JP.
The spread of the obesity epidemic in the United States, 1991-1998. JAMA
1999;282:1519-22.

10. Edmonds MJ, Ferreira KJ, Nikiforuk EA, et al. Body weight and percent body fat increase during the transition from high school to university in females. J Am Diet Assoc 2008;108:1033-7.

 Al-Rethaiaa AS, Fahmy AEA, Al-Shwaiyat NM. Obesity and eating habits among college students in Saudi Arabia: a cross sectional study. Nutrition Journal 2010;9.

Yunsheng. Eating Patterns in Free-Living Healthy U.S. Adult Population.
Ecology of Food and Nutrition 2005;44:37-56.

13. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 1997;337:869-73.

14. Sinaiko AR, Donahue RP, Jacobs DR, Jr., Prineas RJ. Relation of weight and rate of increase in weight during childhood and adolescence to body size, blood pressure, fasting insulin, and lipids in young adults. The Minneapolis Children's Blood Pressure Study. Circulation 1999;99:1471-6.

16. Health Topics: Childhood Obesity. 2010. (Accessed 11/15/10, 2010, at http://www.cdc.gov/HealthyYouth/obesity/.)

Adams MH, Carter TM, Lammon CA, Judd AH, Leeper J, Wheat JR.Obesity and blood pressure trends in rural adolescents over a decade. Pediatr Nurs 2008;34:381-6.

18. Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007-2008.

JAMA;303:242-9.

19. National Center for Health Statistics. Health, United States, 2004 with Chartbook on Trends in Healthy of Americans. 2004.

20. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. N Engl J Med 1992;327:1350-5.

21. Flegal KM, Carroll MD, Ogden CL, Curtin LR. Prevalence and trends in obesity among US adults, 1999-2008. JAMA;303:235-41.

22. Baker JL, Olsen LW, Sorensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. N Engl J Med 2007;357:2329-37.

23. Burke V, Beilin LJ, Simmer K, et al. Predictors of body mass index and associations with cardiovascular risk factors in Australian children: a prospective cohort study. Int J Obes (Lond) 2005;29:15-23.

24. Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35 y. Am J Clin Nutr 1994;59:810-9.

25. Flint AJ, Rexrode KM, Hu FB, et al. Body mass index, waist circumference, and risk of coronary heart disease: a prospective study among men and women. Obes Res Clin Pract;4:e171-e81.

26. Manson JE, Willett WC, Stampfer MJ, et al. Body weight and mortality among women. N Engl J Med 1995;333:677-85.
27. Huang TT, Harris KJ, Lee RE, Nazir N, Born W, Kaur H. Assessing overweight, obesity, diet, and physical activity in college students. J Am Coll Health 2003;52:83-6.

Bibbins-Domingo K, Coxson P, Pletcher MJ, Lightwood J, Goldman L.
 Adolescent overweight and future adult coronary heart disease. N Engl J Med
 2007;357:2371-9.

29. Hovell MF, Mewborn CR, Randle Y, Fowler-Johnson S. Risk of excess weight gain in university women: a three-year community controlled analysis. Addict Behav 1985;10:15-28.

30. Berenson G.S. ea. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The New England Journal of Medicine 1998;338:7.

31. Lloyd-Richardson EE, Bailey S, Fava JL, Wing R. A prospective study of weight gain during the college freshman and sophomore years. Prev Med 2009;48:256-61.

32. Willett W. Nutritional epidemiology: issues and challenges. Int J Epidemiol 1987;16:312-7.

33. The American College Health Association National College Health Assessment (ACHA-NCHA), Spring 2003 Reference Group report. J Am Coll Health 2005;53:199-210.

34. Anderson DA, Shapiro JR, Lundgren JD. The freshman year of college as a critical period for weight gain: an initial evaluation. Eat Behav 2003;4:363-7.

35. Butler SM, Black DR, Blue CL, Gretebeck RJ. Change in diet, physical activity, and body weight in female college freshman. Am J Health Behav 2004;28:24-32.

36. World Health Organization: Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser 2000;894:1-253.

37. Foster GD, Wyatt HR, Hill JO, et al. A randomized trial of a lowcarbohydrate diet for obesity. N Engl J Med 2003;348:2082-90.

Freedman MR, King J, Kennedy E. Popular diets: a scientific review. Obes
 Res 2001;9 Suppl 1:1S-40S.

39. Heini AF, Weinsier RL. Divergent trends in obesity and fat intake patterns:The American paradox. American Journal of Medicine 1997;102:259-64.

40. Wengreen HJ, Moncur C. Change in diet, physical activity, and bodyweight among young-adults during the transition from high school to college. NutrJ 2009;8:32.

41. Prevention CfDCa. Physical Activity and Health: A Report of the Surgeon General Department of Health and Human Services 1996.

42. Strong KA, Parks SL, Anderson E, Winett R, Davy BM. Weight gain prevention: identifying theory-based targets for health behavior change in young adults. J Am Diet Assoc 2008;108:1708-15.

43. Levitsky DA, Halbmaier CA, Mrdjenovic G. The freshman weight gain: a model for the study of the epidemic of obesity. Int J Obes Relat Metab Disord 2004;28:1435-42.

44. Levitsky DA, Garay J, Nausbaum M, Neighbors L, DellaValle DM.

Monitoring weight daily blocks the freshman weight gain: a model for combating the epidemic of obesity. International Journal of Obesity 2006;30:1003-10.

45. Levitsky DA, Garay J, Nausbaum M, Neighbors L, Dellavalle DM.Monitoring weight daily blocks the freshman weight gain: a model for combating the epidemic of obesity. Int J Obes (Lond) 2006;30:1003-10.

46. Vella-Zarb RA, Elgar FJ. The 'Freshman 5': A Meta-Analysis of Weight Gain in the Freshman Year of College. Journal of American College Health 2009;58:161-6.

47. Racette SB, Deusinger SS, Strube MJ, Highstein GR, Deusinger RH.Weight changes, exercise, and dietary patterns during freshman and sophomore years of college. J Am Coll Health 2005;53:245-51.

48. Pliner P, Saunders T. Vulnerability to freshman weight gain as a function of dietary restraint and residence. Physiology & Behavior 2008;93:76-82.

49. Harlan WR, Landis JR, Flegal KM, Davis CS, Miller ME. Secular trends in body mass in the United States, 1960-1980. Am J Epidemiol 1988;128:1065-74.

50. Owen. Is body mass index before middle age related to coronary heart disease risk in later life? Evidence in obersvational studies. Int J Obes (Lond) 2009;33:12.

51. Bibbins-Domingo K. Projected Increase in Rates of Future Adult Coronary Heart Disease Associated with Current Adolescent Obesity. Circulation 2006;114.

52. Kim K. A Comparison between BMI and Conicity Index on Predicting Coronary Heart Disease: The Framingham Heart Study. Annals of Epidemiology 2000;10.

53. Lobstein T, Baur L, Uauy R. Obesity in children and young people: a crisis in public health. Obes Rev 2004;5 Suppl 1:4-104.

54. Klok MD, Jakobsdottir S, Drent ML. The role of leptin and ghrelin in the regulation of food intake and body weight in humans: a review. Obes Rev 2007;8:21-34.

55. Cummings DE, Shannon MH. Roles for ghrelin in the regulation of appetite and body weight. Arch Surg 2003;138:389-96.

56. Howard AD, Feighner SD, Cully DF, et al. A receptor in pituitary and hypothalamus that functions in growth hormone release. Science 1996;273:974-7.

57. Kojima M, Hosoda H, Date Y, Nakazato M, Matsuo H, Kangawa K. Ghrelin is a growth-hormone-releasing acylated peptide from stomach. Nature 1999;402:656-60.

 Marta Korbonits APG, Maria Gueorguiev, and Ashley B. Grossman.
 Ghrelin - a hormone with multiple functions. Frontiers in Neuroendocrinology 2004;25:27-68.

59. van der Lely AJ, Tschop M, Heiman ML, Ghigo E. Biological, physiological, pathophysiological, and pharmacological aspects of ghrelin. Endocr Rev 2004;25:426-57. 60. Schwandt S, SC P, Riley L. Differential Roles for Octanoylated and Decanoylated Ghrelins in Regulating Appetite and Metabolism. International Journal of Peptides 2009:1-6.

61. Staes E, Absil PA, Lins L, et al. Acylated and unacylated ghrelin binding to membranes and to ghrelin receptor: towards a better understanding of the underlying mechanisms. Biochim Biophys Acta;1798:2102-13.

62. Granata R, Baragli A, Settanni F, Scarlatti F, Ghigo E. Unraveling the role of the ghrelin gene peptides in the endocrine pancreas. J Mol Endocrinol;45:107-18.

63. Broom DR, Stensel DJ, Bishop NC, Burns SF, Miyashita M. Exerciseinduced suppression of acylated ghrelin in humans. J Appl Physiol 2007;102:2165-71.

64. Garcia JM, Garcia-Touza M, Hijazi RA, et al. Active ghrelin levels and active to total ghrelin ratio in cancer-induced cachexia. J Clin Endocrinol Metab 2005;90:2920-6.

65. Cummings DE, Purnell JQ, Frayo RS, Schmidova K, Wisse BE, Weigle DS. A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. Diabetes 2001;50:1714-9.

66. St-Pierre DH, Wang L, Tache Y. Ghrelin: a novel player in the gut-brain regulation of growth hormone and energy balance. News Physiol Sci 2003;18:2426.

67. Tschop M, Smiley DL, Heiman ML. Ghrelin induces adiposity in rodents. Nature 2000;407:908-13.

68. Wren AM, Seal LJ, Cohen MA, et al. Ghrelin enhances appetite and increases food intake in humans. J Clin Endocrinol Metab 2001;86:5992.

69. De Vriese C, Perret J, Delporte C. Focus on the short- and long-term effects of ghrelin on energy homeostasis. Nutrition 2010;26:579-84.

70. Leidy HJ, Gardner JK, Frye BR, et al. Circulating ghrelin is sensitive to changes in body weight during a diet and exercise program in normal-weight young women. J Clin Endocrinol Metab 2004;89:2659-64.

71. Wortley KE, Anderson KD, Garcia K, et al. Genetic deletion of ghrelin does not decrease food intake but influences metabolic fuel preference.
Proceedings of the National Academy of Sciences of the United States of America 2004;101:8227-32.

72. Thompson NM, Gill DAS, Davies R, et al. Ghrelin and des-octanoyl ghrelin promote adipogenesis directly in vivo by a mechanism independent of the type 1a growth hormone secretagogue receptor. Endocrinology 2004;145:234-42.

73. Weigle DS, Cummings DE, Newby PD, et al. Roles of leptin and ghrelin in the loss of body weight caused by a low fat, high carbohydrate diet. J Clin Endocrinol Metab 2003;88:1577-86.

74. Beck B, Richy S. Differential long-term dietary regulation of adipokines, ghrelin, or corticosterone: impact on adiposity. Journal of Endocrinology 2008;196:171-9.

75. Takaya K, Ariyasu H, Kanamoto N, et al. Ghrelin strongly stimulates growth hormone release in humans. J Clin Endocrinol Metab 2000;85:4908-11.

Tschop M, Weyer C, Tataranni PA, Devanarayan V, Ravussin E, Heiman ML. Circulating ghrelin levels are decreased in human obesity. Diabetes 2001;50:707-9.

77. Lall S, Balthasar N, Carmignac D, et al. Physiological studies of transgenic mice overexpressing growth hormone (GH) secretagogue receptor 1A in GH-releasing hormone neurons. Endocrinology 2004;145:1602-11.

78. Wortley KE, del Rincon JP, Murray JD, et al. Absence of ghrelin protects against early-onset obesity. J Clin Invest 2005;115:3573-8.

79. Zigman JM, Nakano Y, Coppari R, et al. Mice lacking ghrelin receptors resist the development of diet-induced obesity. J Clin Invest 2005;115:3564-72.

80. Koliaki C, Kokkinos A, Tentolouris N, Katsilambros N. The effect of ingested macronutrients on postprandial ghrelin response: a critical review of existing literature data. Int J Pept;2010.

81. Leite-Moreira AF, Soares JB. Physiological, pathological and potential therapeutic roles of ghrelin. Drug Discov Today 2007;12:276-88.

82. Holst B, Schwartz TW. Constitutive ghrelin receptor activity as a signaling set-point in appetite regulation. Trends Pharmacol Sci 2004;25:113-7.

83. Sanchez J, Cladera MM, Llopis M, Palou A, Pico C. The different satiating capacity of CHO and fats can be mediated by different effects on leptin and ghrelin systems. Behav Brain Res;213:183-8.

84. Sanchez J, Oliver P, Palou A, Pico C. The inhibition of gastric ghrelin production by food intake in rats is dependent on the type of macronutrient.
Endocrinology 2004;145:5049-55.

85. Sbarbati A, Osculati F. The taste cell-related diffuse chemosensory system.Prog Neurobiol 2005;75:295-307.

86. Al Massadi O, Pardo M, Roca-Rivada A, Castelao C, Casanueva FF, Seoane LM. Macronutrients act directly on the stomach to regulate gastric ghrelin release. J Endocrinol Invest;33:599-602.

87. Hass N, Schwarzenbacher K, Breer H. T1R3 is expressed in brush cells and ghrelin-producing cells of murine stomach. Cell Tissue Res;339:493-504.

88. Williams DL, Cummings DE, Grill HJ, Kaplan JM. Meal-related ghrelin suppression requires postgastric feedback. Endocrinology 2003;144:2765-7.

89. Yang N, Liu X, Ding EL, et al. Impaired ghrelin response after high-fat meals is associated with decreased satiety in obese and lean Chinese young adults.J Nutr 2009;139:1286-91.

90. Briatore L, Andraghetti G, Cordera R. Acute plasma glucose increase, but not early insulin response, regulates plasma ghrelin. Eur J Endocrinol 2003;149:403-6.

91. Ariyasu H, Takaya K, Tagami T, et al. Stomach is a major source of circulating ghrelin, and feeding state determines plasma ghrelin-like immunoreactivity levels in humans. J Clin Endocrinol Metab 2001;86:4753-8.

92. Hansen TK, Dall R, Hosoda H, et al. Weight loss increases circulating levels of ghrelin in human obesity. Clin Endocrinol (Oxf) 2002;56:203-6.

93. McCowen KC, Maykel JA, Bistrian BR, Ling PR. Circulating ghrelin concentrations are lowered by intravenous glucose or hyperinsulinemic euglycemic conditions in rodents. J Endocrinol 2002;175:R7-11.

94. Broglio F, Arvat E, Benso A, et al. Ghrelin, a natural GH secretagogue produced by the stomach, induces hyperglycemia and reduces insulin secretion in humans. J Clin Endocrinol Metab 2001;86:5083-6.

95. Broglio F, Gottero C, Prodam F, et al. Non-acylated ghrelin counteracts the metabolic but not the neuroendocrine response to acylated ghrelin in humans. J Clin Endocrinol Metab 2004;89:3062-5.

96. Saad MF, Bernaba B, Hwu CM, et al. Insulin regulates plasma ghrelin concentration. J Clin Endocrinol Metab 2002;87:3997-4000.

97. Marandi S, De Keyser N, Saliez A, et al. Insulin signal transduction in rat small intestine: role of MAP kinases in expression of mucosal hydrolases. Am J Physiol Gastrointest Liver Physiol 2001;280:G229-40.

98. Salehi A, Dornonville de la Cour C, Hakanson R, Lundquist I. Effects of ghrelin on insulin and glucagon secretion: a study of isolated pancreatic islets and intact mice. Regul Pept 2004;118:143-50.

99. Murata M, Okimura Y, Iida K, et al. Ghrelin modulates the downstream molecules of insulin signaling in hepatoma cells. J Biol Chem 2002;277:5667-74.
100. Damjanovic SS, Lalic NM, Pesko PM, et al. Acute effects of ghrelin on insulin secretion and glucose disposal rate in gastrectomized patients. J Clin Endocrinol Metab 2006;91:2574-81.

101. Poykko SM, Kellokoski E, Horkko S, Kauma H, Kesaniemi YA, UkkolaO. Low plasma ghrelin is associated with insulin resistance, hypertension, and the prevalence of type 2 diabetes. Diabetes 2003;52:2546-53.

102. Dezaki K, Sone H, Yada T. Ghrelin is a physiological regulator of insulin release in pancreatic islets and glucose homeostasis. Pharmacol Ther 2008;118:239-49.

103. Schwartz MW, Woods SC, Porte D, Jr., Seeley RJ, Baskin DG. Central nervous system control of food intake. Nature 2000;404:661-71.

104. Inui A. Ghrelin: an orexigenic and somatotrophic signal from the stomach.Nat Rev Neurosci 2001;2:551-60.

105. Woods SC, Seeley RJ, Porte D, Jr., Schwartz MW. Signals that regulate food intake and energy homeostasis. Science 1998;280:1378-83.

106. Shintani M, Ogawa Y, Ebihara K, et al. Ghrelin, an endogenous growth hormone secretagogue, is a novel orexigenic peptide that antagonizes leptin action through the activation of hypothalamic neuropeptide Y/Y1 receptor pathway. Diabetes 2001;50:227-32.

107. Nakazato M, Murakami N, Date Y, et al. A role for ghrelin in the central regulation of feeding. Nature 2001;409:194-8.

 Asakawa A, Inui A, Kaga T, et al. Ghrelin is an appetite-stimulatory signal from stomach with structural resemblance to motilin. Gastroenterology 2001;120:337-45.

109. Toshinai K, Mondal MS, Nakazato M, et al. Upregulation of Ghrelin expression in the stomach upon fasting, insulin-induced hypoglycemia, and leptin administration. Biochem Biophys Res Commun 2001;281:1220-5. 110. Ravussin E, Tschop M, Morales S, Bouchard C, Heiman ML. Plasma ghrelin concentration and energy balance: overfeeding and negative energy balance studies in twins. J Clin Endocrinol Metab 2001;86:4547-51.

111. Korbonits M, Goldstone AP, Gueorguiev M, Grossman AB. Ghrelin--a hormone with multiple functions. Front Neuroendocrinol 2004;25:27-68.

112. Banks WA, Tschop M, Robinson SM, Heiman ML. Extent and direction of ghrelin transport across the blood-brain barrier is determined by its unique primary structure. J Pharmacol Exp Ther 2002;302:822-7.

113. Tortorella C, Macchi C, Spinazzi R, Malendowicz LK, Trejter M, Nussdorfer GG. Ghrelin, an endogenous ligand for the growth hormonesecretagogue receptor, is expressed in the human adrenal cortex. Int J Mol Med 2003;12:213-7.

114. Cummings DE, Weigle DS, Frayo RS, et al. Plasma ghrelin levels after
diet-induced weight loss or gastric bypass surgery. N Engl J Med 2002;346:162330.

115. Blom WA, Stafleu A, de Graaf C, Kok FJ, Schaafsma G, Hendriks HF. Ghrelin response to carbohydrate-enriched breakfast is related to insulin. Am J Clin Nutr 2005;81:367-75.

116. Callahan HS, Cummings DE, Pepe MS, Breen PA, Matthys CC, Weigle DS. Postprandial suppression of plasma ghrelin level is proportional to ingested caloric load but does not predict intermeal interval in humans. J Clin Endocrinol Metab 2004;89:1319-24.

117. Erdmann J, Topsch R, Lippl F, Gussmann P, Schusdziarra V. Postprandial response of plasma ghrelin levels to various test meals in relation to food intake, plasma insulin, and glucose. J Clin Endocrinol Metab 2004;89:3048-54.

118. Korbonits M, Lecoeur C, Froguel P, Grossman AB, Gueorguiev M.Characteristics of the ghrelin receptor in relation to eating behaviour 5th.International Symposium on GH Secretagogues, Portofino-Vetta, Italy. 2004.

119. St-Pierre DH, Karelis AD, Cianflone K, et al. Relationship between ghrelin and energy expenditure in healthy young women. Journal of Clinical Endocrinology & Metabolism 2004;89:5993-7.

Marzullo P, Verti B, Savia G, et al. The relationship between active ghrelin levels and human obesity involves alterations in resting energy expenditure.Journal of Clinical Endocrinology & Metabolism 2004;89:936-9.

121. Huda MSB, Dovey T, Wong SP, et al. Ghrelin restores 'lean-type' hunger and energy expenditure profiles in morbidly obese subjects but has no effect on postgastrectomy subjects. International Journal of Obesity 2009;33:317-25.

122. Gaul DC, W. Mahoneym M. Relationships between Eating Rate and Obesity. Journal of Consulting and Clinical Psychology 1975;43:123-5.

123. Hill SW, McCutcheon NB. Eating responses of obese and nonobese humans during dinner meals. Psychosom Med 1975;37:395-401.

124. Andrade AM, Greene GW, Melanson KJ. Eating slowly led to decreases in energy intake within meals in healthy women. J Am Diet Assoc 2008;108:1186-91.

125. Kral JG, Buckley MC, Kissileff HR, Schaffner F. Metabolic correlates of eating behavior in severe obesity. Int J Obes Relat Metab Disord 2001;25:258-64.
126. Spiegel TA, Jordan HA. Effects of Simultaneous Oral-Intra-Gastric Ingestion on Meal Patterns and Satiety in Humans. Journal of Comparative and Physiological Psychology 1978;92:133-41.

127. Kissileff HR, Guss JL. Microstructure of eating behavior in humans.Appetite 2001;36:70-8.

128. Val-Laillet D, Guerin S, Malbert CH. Slower eating rate is independent to gastric emptying in obese minipigs. Physiology & Behavior 2010:1-7.

129. Spitzer L, Rodin J. Human Eating Behavior - a Critical-Review of Studies in Normal Weight and Overweight Individuals. Appetite 1981;2:293-329.

130. Ferster CB, Nurnberger JI, Levitt EB. The control of eating (Reprinted from J Mathetics, vol 1, pg 87-109, 1962). Obesity Research 1996;4:401-10.

131. Otsuka R, Tamakoshi K, Yatsuya H, et al. Eating fast leads to obesity: findings based on self-administered questionnaires among middle-aged Japanese men and women. J Epidemiol 2006;16:117-24.

132. Spiegel TA, Shrager EE, Stellar E. Responses of lean and obese subjects to preloads, deprivation, and palatability. Appetite 1989;13:45-69.

133. Teitelbaum P, Campbell BA. Ingestion patterns in hyperphagic and normal rats. J Comp Physiol Psychol 1958;51:135-41.

134. Spiegel T, Wadden T, Foster G. Objective measurement of eating rate during behavioral treatment of obesity. Behav Ther 1991;22:61-7.

135. Spiegel TA. Rate of intake, bites, and chews-the interpretation of leanobese differences. Neurosci Biobehav Rev 2000;24:229-37.

136. Hill SW, McCutcheon NB. Contributions of obesity, gender, hunger, food preference, and body size to bite size, bite speed, and rate of eating. Appetite 1984;5:73-83.

137. Park HA, Lee JS, Kuller LH. Underreporting of dietary intake by bodymass index in premenopausal women participating in the Healthy Women Study.Nutr Res Pract 2007;1:231-6.

138. Goldberg GR, Black AE, Jebb SA, et al. Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. Eur J Clin Nutr 1991;45:569-81.

139. Vansant G, Hulens M. The assessment of dietary habits in obese women: influence of eating behavior patterns. Eat Disord 2006;14:121-9.

140. Scagliusi FB, Polacow VO, Artioli GG, Benatti FB, Lancha AH. Selective underreporting of energy intake in women: Magnitude, determinants, and effect of training. Journal of the American Dietetic Association 2003;103:1306-13.

141. Smith WT, Webb KL, Heywood PF. The implications of underreporting in dietary studies. Aust J Public Health 1994;18:311-4.

Braam LA, Ocke MC, Bueno-de-Mesquita HB, Seidell JC. Determinants of obesity-related underreporting of energy intake. Am J Epidemiol 1998;147:10816.

143. Kretsch MJ, Fong AK, Green MW. Behavioral and body size correlates of energy intake underreporting by obese and normal-weight women. J Am Diet Assoc 1999;99:300-6; quiz 7-8.

144. Johansson L, Solvoll K, Bjorneboe GE, Drevon CA. Under- and overreporting of energy intake related to weight status and lifestyle in a nationwide sample. Am J Clin Nutr 1998;68:266-74.

145. Duncan KH, Bacon JA, Weinsier RL. The effects of high and low energy density diets on satiety, energy intake, and eating time of obese and nonobese subjects. Am J Clin Nutr 1983;37:763-7.

146. Sobki SH, Zaid AA, Khan HA, Alhomida AS, Hilal KA, Khan SA.Significant impact of pace of eating on serum ghrelin and glucose levels. ClinBiochem 2010;43:522-4.

147. Kokkinos A, le Roux CW, Alexiadou K, et al. Eating slowly increases the postprandial response of the anorexigenic gut hormones, peptide YY and glucagon-like peptide-1. J Clin Endocrinol Metab;95:333-7.

148. Zijlstra N, Mars M, de Wijk RA, Westerterp-Plantenga MS, Holst JJ, de Graaf C. Effect of viscosity on appetite and gastro-intestinal hormones. Physiol Behav 2009;97:68-75.

149. Ledikwe JH, Blanck HM, Khan LK, et al. Dietary energy density determined by eight calculation methods in a nationally representative United States population. J Nutr 2005;135:273-8.

 Cox DN, Mela DJ. Determination of energy density of freely selected diets: methodological issues and implications. Int J Obes Relat Metab Disord 2000;24:49-54.

151. Stubbs RJ, Harbron CG, Murgatroyd PR, Prentice AM. Covert manipulation of dietary fat and energy density: effect on substrate flux and food intake in men eating ad libitum. Am J Clin Nutr 1995;62:316-29.

152. Rolls BJ, Castellanos VH, Halford JC, et al. Volume of food consumed affects satiety in men. Am J Clin Nutr 1998;67:1170-7.

153. Bell EA, Castellanos VH, Pelkman CL, Thorwart ML, Rolls BJ. Energy density of foods affects energy intake in normal-weight women. Am J Clin Nutr 1998;67:412-20.

154. Seagle HM, davy BM, Grunwald G, Hill JO. Energy density of selfreported food intake: variation and relationship to other food components. Obes Res 1997;5:78S.

155. Grunwald GK, Seagle HM, Peters JC, Hill JO. Quantifying and separating the effects of macronutrient composition and non-macronutrients on energy density. Br J Nutr 2001;86:265-76.

156. Bowman SA, Gortmaker SL, Ebbeling CB, Pereira MA, Ludwig DS.Effects of fast-food consumption on energy intake and diet quality among children in a national household survey. Pediatrics 2004;113:112-8.

157. Stookey JD. Energy density, energy intake and weight status in a large free-living sample of Chinese adults: exploring the underlying roles of fat, protein, carbohydrate, fiber and water intakes. Eur J Clin Nutr 2001;55:349-59.

158. Marti-Henneberg C, Capdevila F, Arija V, et al. Energy density of the diet, food volume and energy intake by age and sex in a healthy population. Eur J Clin Nutr 1999;53:421-8.

159. Ledikwe JH, Blanck HM, Kettel Khan L, et al. Dietary energy density is associated with energy intake and weight status in US adults. Am J Clin Nutr 2006;83:1362-8.

160. Rolls BJ, Bell EA, Castellanos VH, Chow M, Pelkman CL, Thorwart ML. Energy density but not fat content of foods affected energy intake in lean and obese women. Am J Clin Nutr 1999;69:863-71.

161. Hogenkamp PS, Mars M, Stafleu A, de Graaf C. Intake during repeated exposure to low- and high-energy-dense yogurts by different means of consumption. Am J Clin Nutr;91:841-7.

162. Hertzler AA, Frary RB. Family factors and fat consumption of college students. J Am Diet Assoc 1996;96:711-4.

163. Anding JD, Suminski RR, Boss L. Dietary intake, body mass index, exercise, and alcohol: are college women following the dietary guidelines for Americans? J Am Coll Health 2001;49:167-71.

164. Melby C, Femra P, Sciacca J. Reported dietary and exercise behaviors,beliefs and knowledge among university undergraduates. Nutrition Research1986;6:799-808.

165. Cummings DE. Ghrelin and the short- and long-term regulation of appetite and body weight. Physiol Behav 2006;89:71-84.

166. Gualillo O, Lago F, Gomez-Reino J, Casanueva FF, Dieguez C. Ghrelin, a widespread hormone: insights into molecular and cellular regulation of its expression and mechanism of action. FEBS Lett 2003;552:105-9.

167. Karl JP, Young AJ, Montain SJ. Eating rate during a fixed-portion meal does not affect postprandial appetite and gut peptides or energy intake during a subsequent meal. Physiol Behav;102:524-31.

Appendix B: Extended Materials and Methods

METHODOLOGY:

Research Design

This research is an ancillary study examining the effect of weight status on ghrelin concentrations and eating rate. It will also look for a relationship between eating rate and energy density of a meal consumed. Cross-sectional data from the Best Foot Forward Study (BFF) and the Nutrition Assessment and Chronic Disease Risk Factor Identification (Hatch) study will be used. The BFF study was a randomized clinical trial and examined the effectiveness of a self-taught intervention versus an instructed intervention on coronary heart disease (CHD) risk factors in overweight and obese female students at the University of Rhode Island (URI) over eight weeks. However, in the present study only baseline data will used for analysis. The Hatch study was cross-sectional and determined the prevalence of metabolic syndrome and CHD risk factors and their relationship to dietary intake and physical activity in first year URI students.

Subjects

All BFF and Hatch participants read and signed informed consents for their respective studies, which were approved by URI's Institutional Review Board. Participants for both studies were recruited via flyers posted around campus and in dormitories, through "The Good 5 Cent Cigar" advertisements, presentations made in classes, and informational booths at Hope dining hall and the Memorial Union

Building on URI's Kingston campus. Eligibility criteria for each study is outline in

Table 1.

Table 1. Eligibility Criteria for the Hatch and BFF Studies				
Hatch Study	BFF Study			
First year URI student	Female URI student			
No body mass index (BMI) parameters	BMI 25-39.9 kg/m ²			
Between the ages of 18 and 24 years	Between the ages of 18 and 24 years			
Not pregnant or lactating	Not pregnant or lactating			
No diagnosis of: CHD, diabetes mellitus	No diagnosis of: CHD, DM, liver			
(DM), liver disease, cancer, bleeding	disease, kidney disease, cancer,			
disorder	bleeding disorder			
Not on any lipid-lowering medications	Not on any lipid-lowering medications			
No history of an eating disorder	No history of an eating disorder			

Seventy-three participants signed informed consents for the BFF study, 9 were unwilling or unable to complete the study, leaving a final sample size of 64. Participants were recruited in two cohorts, spring and fall of 2009. The BFF participants had a mean BMI of 29.7 ± 3.63 kg/m² and consisted of ~64% participants who were overweight (n=41) and ~36% who were obese (n=23). Sixty-seven percent of the participants identified themselves as Caucasian (n=43), 16% Black/African American (n=10), 8% Hispanic (n=5), and 9% as other (n=6). Two-hundred-and-ninety-four participants signed informed consents for the Hatch study, 33 were unwilling or unable to complete the study, leaving a final sample size of 261, 67% females (n=176) and 33% males (n=85). Reasons for attrition included lack of time, illness, and lack of response to study contacts. The Hatch participants were recruited in four cohorts, spring and fall of 2008 and spring and fall of 2009. The Hatch female participants had a mean BMI of 22.9±3.84 and consisted of 4.5% underweight participants (n=8), 77.3% normal (n=136), 13.1% overweight (n=23) and 5.1% obese (n=9). Of the female sample 82% identified them as Caucasian (n=144), 3.4% as Asian (n=6), 3.9% as African American (n=7), 0.5% as American Indian (n=1) and 10.2% as other (n=18).

Baseline Measurements

Anthropometric

For both studies, all anthropometric measurements were performed in duplicate to assure that measurements were within 0.2 cm for height and 0.1 kg for weight (1). Height was measured using a 220 stadiometer (Seca Corporation, Hamburg, Germany). Waist circumference was measured using Gulick nonstretchable tape with an attached tensometer at the top of the iliac crest upon exhalation (Patterson Medical, Mouth Joy, PA) while participants were in a standing position (1). In the Hatch study, weight was measured using a calibrated digital Seca 769 scale (Seca Corporation, Hamburg, Germany) to the nearest 0.2 kg; for the BFF study, weight was measured to the nearest 0.1 kg by using a calibrated digital read scale (Bod Pod, Version 2.14, Body Composition System; Life Measurement Instruments, Concord, CA). Both studies used height and

weight to calculate participant BMI (weight in kilograms/height in meters²). The participants will be broken up into three categories based on their weight status: normal weight (18.5-24.9 kg/m²), overweight (25-29.9 kg/m²) and obese (\geq 30 kg/m²) (2, 3).

Biochemical

For both studies, a trained phlebotomist conducted two 12-hour fasted venous blood draws on two nonconsecutive morning visits in the same week. Samples were processed according to a previously described protocol (4). Briefly, plasma was obtained from whole blood via centrifugation (Eppendorf Centrifuge 5810, Germany) for 20 minutes at 2200 RPM's at 4°C. The following preservation cocktail was added to the plasma samples: 0.01g/100g of phenylmethyl sulfonyl fluoride (Roche, Indianapolis, IN), 0.01g/100g of sodium azide (Fisher, Fairlawn, NJ) and 0.05g/100g of aprotinin (Fisher, Fairlawn, NJ) and were stored at -80° until analyses.

Enzymatic assays for analyses were performed for glucose (GLC) concentrations using a Wako Glucose Autokit (Wako Diagnostics, Richmond, VA) (5).

Clinical

Resting blood pressure (BP) was measured by a trained exercise physiologist after a five-minute seated rested period using a stethoscope (Littman, St. Paul, MN) an appropriately sized BP cuff (Fisher Scientific, Fairlawn, NJ and Welch-Allyn, Skaneateles Falls, NY). Blood pressure was measured in duplicate unless variance between measures exceeded the standard (two mmHg), in which

case the measurement was repeated. The average of the two readings within the standard was used for data analysis.

Physical Activity

Both studies assessed physical activity using the International Physical Activity Questionnaire Short Form (IPAQ-S) (6) which was scored based on the Guidelines for Data Processing and Analysis of the International Physical Activity Questionnaire (7). This tool has been validated in young adult research reflecting habitual physical activity levels of low, moderate and vigorous activity (6, 8). Participants were asked to report their frequency (days per week) and duration (time in minutes) of low, moderate, and vigorous physical activity over the past seven days. Physical activity is expressed in metabolic equivalents (METs) in METs/minute/week (METs-min/wk); METs-min is the product of the MET score of an activity multiplied by the minutes performed (9). Metabolic equivalents were calculated using the scoring protocol: 1 MET= resting energy expenditure, 3.3 METs=walking, 4 METs=moderate intensity physical activity, and 8 METs=vigorous intensity physical activity (10). These values were each multiplied by the amount of minute each activity was performed and the amount of days (i.e. walking MET-min/week = 3.3 x walking minutes x walking days).

Required Training

All study staff that worked with blood and hazardous waste completed the URI Blood Borne Pathogens and Hazardous Waste Management Training courses and were certified by the Collaborative Institutional Training Initiative Program

for Humans Subjects Protection. All study staff were properly trained to perform anthropometric, clinical, biochemical and dietary assessment measurements.

Table 2. Study Timeline.							
	9/10	10/10	11/10	12/10	1/11	2/11	
Biochemical		X	X				
Assays							
Data Entry	X	X	Х				
Data Analysis			Х	X	X		
Manuscript						X	

Methodology for Primary Aim Hypothesis #1:

Participants classified in the three different weight categories will have different eating rates.

Dietary data was collected using 24 hour recalls (24HR), and analyzed by the Nutrition Data System for Research (NDSR) software versions 2007, 2008, and 2009 (Nutrition Coordinating Center, University of Minnesota, Minneapolis, MN). The NDSR program is a validated dietary assessment tool developed to minimize the underreporting of dietary intake and to increase accuracy (11, 12). All study participants completed three 24HR recalls with trained study personnel: one in-person and two over the phone. Recalls were collected on three nonconsecutive days (two weekdays and one weekend day); as this method provides a good estimate of an individual's usual intake (13, 14). The 24HR was used to gather the participant's food and dietary supplement intake within the past 24

hours, using a multiple-pass system. The interviewer asked a series of guided computer-generated questions regarding food and beverage intake in the past 24 hours. This interactive program prompts the interviewer to probe for detailed information using neutral and non-leading questions about specific quantities, brand names and cooking methods for each food. The participant is first asked for a quick list of foods consumed, then the interviewer probes for details of the foods consumed including types, amounts, additions/toppings, and preparation method. Then finally the final pass of the interview includes a review of the foods consumed, details of the food eaten, and amounts consumed; thus the interviewee can correct any data that is inaccurate (13). In addition to the NDSR prompted questions, both studies asked two other questions: 1) How many individuals did you eat this meal with? and 2) How long did you spend eating this meal?

Participants also received food amounts booklets designed by the Nutrition Coordinating Center at the University of Minnesota at the first 24HR, which they kept for the additional 24HR's, The food amounts booklet to help participants better estimate amount and sizes of foods eaten. During this first, in-person 24HR, Nasco food models (eNASCO, Fort Atkinson, WI) were available to help participants visually estimate portion sizes and accurately estimate the amount of the food consumed.

Based on previous research, eating rate was expressed as grams of food consumed per second and included all foods and only beverages that provide ≥ 5 kilocalories per 100 grams (15). The total number of grams of food and seconds of meal consumption time were determined and expressed as the mean per day.

The means of grams of food consumed and seconds of meal consumption time were then averaged for a total mean of the three 24HRs. Those two means were used to calculate grams of food consumed per second, which is how eating rate was defined.

Methodology for Primary Aim Hypothesis #2:

Participants classified in the three different weight categories will have different total fasting ghrelin concentrations.

All samples were labeled with the participant ID number and processed according to protocol stated in the above Biochemical section. Plasma samples will be removed from the freezer and thawed to room temperature to complete the ghrelin assays. Plasma unacylated ghrelin concentrations were measured in duplicate using an enzyme-linked immunosorbent assay (ELISA) kit 96 well plate assay (manufactured by SPI-Bio, Montigny le Bretonneux, France and distributed by ALPCO Diagnostics, Salem, NH). Ghrelin will be processed according to the protocol provided below. Different tips to pipette the buffer, standard, quality control, samples, tracer, and other reagents were used throughout the assay procedure. Briefly, once all samples are at room temperature, and reagents are reconstituted, the wash buffer was prepared by diluting 1 mL into 400 mL of distilled or de-ionized water and 200 µl of the tween 20 reagent. The well plate was removed from the packet and sufficient strips with be used, each well was rinsed with 300 μ l of wash buffer five times. Just before the reagents and samples were distributed, the buffer was removed from the wells by inverting the plate and

drying by inversion on absorbent paper. The provided suggested plate layout was used. The contents of each well were recorded, using sample ID numbers, on the sheet provided by the kit. The first column was left empty for blanking Ellman's reagent. One hundred microliters of the EIA buffer was distributed to each Non-Specific Binding well. After, 100 μ l of each of the eight standards was dispensed in duplicate to the appropriate wells. The lowest concentration standard was used first and the pipette tip was equilibrated in the next higher standard before pippetting. Then, 100 µl of the quality control and samples were dispensed in duplicates into the appropriate wells. If there were any highly concentrated samples they were diluted in the EIA buffer. Then $100 \,\mu$ l of the anti-unacylated ghrelin-AChE tracer was dispensed in each well except for the blank wells. The well plate was then covered with adhesive film and incubated for three hours at room temperature. After incubation, Ellman's reagent was reconstituted. The well plate was washed five times with the wash buffer, with 300 μ l being dispensed into each well. The well plate then was shaken on an orbital shaker for five minutes and then re-washed five times with 300 μ l of the wash buffer dispensed into each well. The liquid was removed by inverting the plate and drying by inversion on absorbent paper. After, 200 µl of Ellman's reagent was dispensed into all 96 wells. The well plate was then incubated in darkness and placed on an orbital shaker for optimal development. The well plate was read at 405 nm and 414 nm after 30 to 60 minutes from adding Ellman's reagent. The ghrelin protocol was properly entered and set up on a Gen 5.1.08 Data Analysis software (BioTek Instruments Inc., Winooski, VT) in order to obtain and analyze ghrelin

concentrations. All readings were used for analysis if the coefficient of variation (CV) values were $\leq 15\%$; samples were re-run if above 15%.

Methodology for Secondary Aim Hypothesis:

Participants classified in the three different weight status categories will have different energy densities

Energy density was calculated using the average energy intake in kilocalories and food intake in grams of the three 24HRs. The total number of kcals and grams were expressed as means per day. The means of kcals of energy intake and grams of food intake were then averaged for a total mean of the three 24HRs. Those two means were used to calculate kcals of energy intake per gram of food, which is how energy density was defined. There is no agreed upon method for calculating energy density as many different methodologies for calculation are proposed and recognized in nutrition research (16). In this study, energy density was calculated using total energy intake of meal in kilocalories divided by total weight of the meal in grams, and is expressed as kilocalories/gram. Energy density included all meals and snacks except for beverages containing at \leq 5 kilocalories/100 grams (16). Ledikwe *et al* (16) used this method in a free-living population and found a significant positive relationship between energy density and total reported energy intake.

DATA ANALYSIS AND STATISTICS

To maintain confidentiality, participants' identification number contained no identifying information. The list of names and ID numbers is kept locked in a filing cabinet located in Dr. Lofgren's office, Ranger Hall Room 301 Ranger Hall on the Kingston campus at URI.

Sample Size

One-way analyses of variance (ANOVA) for ghrelin concentrations and eating rate were used to calculate sample size needed as they are the primary outcomes. The ANOVA for ghrelin only required six participants to achieve a power of 0.8. The ANOVA for eating rate required 78 participants to achieve a power of 0.8 with a large effect size f=0.4; an effect similar to that found by Laessle *et al* (2007) as interpreted by Cohen (17), with an expected difference of 1.2 grams/second between normal weight and overweight/obese groups. In order to have a total of 78 participants, 26 will be needed for each weight status classification: normal weight, overweight, and obese. Participants with full data sets were randomly selected and 44 BFF and 34 Hatch participants were used. Significance was set at α =0.025 by applying the Bonferroni correction to account for power calculations for the two main analyses.

Analysis

Data analyses were performed using the Statistical Package for the Social Sciences (SPSS) for Windows 19.0 (IBM Corp., Somers, NY). Normality was assessed by examining skewness and kurtosis, and non-normally distributed data.

Outliers that were greater than three standard deviations from the mean were removed for TAG (n=1), IPAQ (n=2), percent kilocalories from fat (n=1) and energy density (n= 1). Descriptive statistics were reported in mean and standard deviations for age, weight, height, BMI, WC, blood pressure, TC, HDL-C, LDL-C, TAG, GLC, total energy in kilocalories, % kilocalories from fat, protein, and carbohydrates, IPAQ, ghrelin and eating rate; frequencies and percentages were reported for weight status categories. Significance was set at p<0.05.

Primary Analysis

Two ANOVA's were run for the primary analyses, one examined the eating rate differences across the three weight statuses and the other examined the ghrelin differences across the same weight statuses. If any significant differences were found between groups, a Tukey follow-up test was used to find where those differences occurred.

Secondary Analyses

For secondary analyses, independent samples *t*-tests were used to compare the effect of eating rate on ghrelin concentrations and energy density.

RESOURCES REQUIRED

No university resources were required other than departmental computers, laboratory equipment and data analysis software. Laboratory supplies such as pipettes, pipette tips, plastic microtubes, glass beakers and gloves were used to perform biochemical analysis. Data and samples from the Nutritional Assessment and Chronic Disease Risk Factor Identification in Young Adults (IRB# HU0708-

029) and the Best Foot Forward Study (IRB# HU0809-043) were used. Dr. Ingrid Lofgren provided funding necessary to buy the ELISA kits and laboratory supplies. Dr. Katherine Peterson provided laboratory machines and equipment.

Literatures Cited:

1 Janssen I, Katzmarzyk PT, Ross R. Body mass index, waist circumference, and health risk: evidence in support of current National Institutes of Health guidelines. *Arch Intern Med* 2002;162:2074-9.

2 NCCDPHP. Overweight and Obesity. 2010.

3 Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National Institutes of Health. *Obes Res* 1998;6 Suppl 2:51S-209S.

4 Lofgren IE HK, West KL, Zern TL, Patalay M, Koo SI, Fernandez ML. Carbohydrate intake is correlated with biomarkers for coronary heart disease in a population of overweight premenopausal women. *Journal of Nutrition Biochemistry* 2005;16:245-50.

5 Reljic R RM, Anic N, Ries B. New chromogen for assay of glucose in serum. *Clin Chem* 1992;38:522-5.

6 Craig CL, Marshall AL, Sjostrom M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc* 2003;35:1381-95.

7 Guidelines for Data Processing and Analysis of the International Physical Activity Questionnaire (IPAQ) - Short and Long Forms. 2005:1-15.

8 Zanovec M, Johnson LG, Marx BD, Keenan MJ, Tuuri G. Self-reported physical activity improves prediction of body fatness in young adults. *Med Sci Sports Exerc* 2009;41:328-35.

9 International Physical Activity Questionnaire. Short last 7 days selfadministered format. 2002.

10 King S, Gibney M. Dietary advice to reduce fat intake is more successful when it does not restrict habitual eating patterns. *J Am Diet Assoc* 1999;99:685-9.

11 Schakel S. Maintaining a nutrient database in a changing marketplace: Keeping pace with changing food products - A research perspective. *J of Food Composition and Analysis* 2001;14.

12 Jonnalagadda SS, Mitchell DC, Smiciklas-Wright H, et al. Accuracy of energy intake data estimated by a multiple-pass, 24-hour dietary recall technique. *J Am Diet Assoc* 2000;100:303-8; quiz 9-11.

13 Willett W. Nutritional epidemiology: issues and challenges. *Int J Epidemiol* 1987;16:312-7.

14 Nelson M, Black AE, Morris AD, Cole TJ. Between- and within-subject variation in nutrient intake from infancy to old age: estimating the number of days required to rank dietary intakes with desired precision. *Am J Clin Nutr* 1989;50:155-67.

Ledikwe JH, Blanck HM, Kettel Khan L, et al. Dietary energy density is associated with energy intake and weight status in US adults. *Am J Clin Nutr* 2006;83:1362-8.

16 Ledikwe JH, Blanck HM, Khan LK, et al. Dietary energy density determined by eight calculation methods in a nationally representative United States population. *J Nutr* 2005;135:273-8.

17 Laessle RG, Lehrke S, Duckers S. Laboratory eating behavior in obesity. *Appetite* 2007;49:399-404.

Appendix C: BFF Phase 1 Participant Survey

BFF Online Survey Questions Welcome to the BFF Study! Please answer all of the following questions. **Personal Information**

- 1. Please enter your URI student ID (All information will remain confidential)
- 2. What is your e-mail?

3. What is a phone number where we can reach you?

Please answer all of the following questions......

1. What is your gender?

Male or Female

2. What is your current age?

3. What is your race or ethnic group?

White (not of Hispanic origin)
Black (not of Hispanic origin)
Hispanic/Latino
American Indian/Alaska Native
Asian/Pacific Islander
Other
4. Where do you live?

On-campus or Off-campus

5. What is your major?

Agricultural Sciences

Biological Sciences Business Administration Education English Exercise Science Fine Arts Humanities Kinesiology/Physical Education Nutrition and Food Sciences Social Science Undeclared Other

Consent Form

Can I participate?

You must be

- 18-24 years of age
- A URI student

• Willing to complete an on-line questionnaire.

You must not Be:

• Pregnant or lactating

What will I be asked to do?

If you choose to participate, you will be asked to:

• Complete an online survey (approximately 20 minutes).

What do I get out of it?

You may be eligible to participate in a weight-loss study being done by the Nutrition and Food Science Department.

Risks

There is no risk in participating in this survey.

Voluntary Participation

Your participation is voluntary. You may stop participating in this survey at any point.

Confidentiality

All information that you provide will be kept confidential and your privacy will be protected to the maximum extent allowable by law. The website is password protected for both the researcher and subjects. The data will be stored on a disk in the Lipid Lab at the University of Rhode Island. Printouts of the data will be stored in locked offices at The University of Rhode Island for up to 5 years (as required by law) and then destroyed. Data will be reported in summary format, and no names will be used.

Questions

If you have any questions or concerns, please contact the researchers listed below. If you have concerns regarding your rights as a research participant, please contact the human subjects representative listed below. Researchers: Emily Cook email: lipidlab@etal.uri.edu (Subject: BFF) or Ingrid Lofgren email: ingridlofgren@uri.edu

Human Subjects Representative: Vice President for Research and Economic Development 70 Lower College Road University of Rhode Island Kingston, RI 02881 Phone (401) 874-4328. email: robind@uri.edu

This project has been reviewed and approved by the human subject review board of the University of Rhode Island.

Thank you for your time and interest in this study.

Continuing in this study indicates that you have read and understand the above information. If you would like a copy of this form, please print it now.

1. Continuing in this study indicates that you have read and understand the above information.

I am ready to begin the survey.

I am not interested in this study.

Dietary History

1. How many meals (i.e., breakfast, lunch, dinner) do you eat per day?

2. How many snacks (i.e.; candy/energy bars, chips, pieces of fruit) do you eat per day?

3. How many days per week do you eat breakfast?

4. How fast is your rate of eating?

very slow relatively slow medium relatively fast very fast

5. Choose the statement that best describes your typical eating behavior.
I always eat whatever I want, whenever I want.

I often eat whatever I want, whenever I want.

I only sometimes eat whatever I want, whenever I want.

I often refrain from eating what I want but often "give in" and eat it anyway.

I often refrain from eating what I want and rarely "give in" and eat it anyway.

I often refrain from eating what I want and never "give in" and eat it anyway.

Body Weight History

1. What is the length of time you have maintained your present weight?

Years? Months? Days?

- 2. How tall are you?
- 3. How much do you weigh?
- 4. How much would you like to weigh?

5. How many times has your weight fluctuated by at least 6. Please describe any long-term weight changes you have experienced (e.g., lost 50 lb. in 1995):

7. How would you describe the typical weight of your parents over the last few years?

Your Mother: Underweight? Just right? Overweight? Obese? Unknown?

Your Father: Underweight? Just right? Overweight? Obese? Unknown?

8. What is the amount of moderate or vigorous activity (such as brisk walking, jogging, biking, aerobics, or yard work) you do in addition to your normal daily routine, most days?

Less than 30 minutes 30 - 60 minutes More than 60 minutes

9. How many hours do you work (a paying job) per week?

0-3 hours 4-10 hours 10-20 hours 20-30 hours 30-40 hours More than 40 hours

10. Are you currently participating in a sports team or sports club?

Yes or No

Fruit Intake

Please use the following information to answer the questions on this page: 1 CUP OF FRUIT EQUALS small apple
 large strawberries
 large plums
 seedless grapes
 cup 100% juice
 cup cooked fruit
 About how many cups of FRUIT (including 100% pure fruit juice) do you eat or drink each day?
 None
 1/2 to 1 cup
 to 2 cups
 to 3 cups

3 to 4 cups

1 cup fruit

4 cups or more

2. How many cups of FRUITS do you think you should eat each day for good health?

None

1/2 to 1 cup

1 to 2 cups

2 to 3 cups

3 to 4 cups

4 cups or more

Vegetable Intake

Please use the following information to answer the questions on this page:

1 CUP OF VEGETABLES EQUALS

1 cup of vegetables

5 broccoli spears 5 inches long

1 cup cooked leafy greens

2 cups lettuce or raw greens

12 baby carrots

1 large potato or sweet potato

2 large celery sticks

1 cup cooked beans

1. About how many cups of VEGETABLES (including 100% vegetable juice) do you eat or drink each day?

None 1/2 to 1 cup to 2 cups
 to 3 cups
 to 4 cups
 cups or more
 About how p

2. About how many cups of VEGETABLES (including 100% vegetable juice) do you think you should eat each day for good health?

None

1/2 to 1 cup

1 to 2 cups

2 to 3 cups

3 to 4 cups

4 cups or more

Eating Competence

These questions are about your eating and food-providing skills, attitudes, and behaviors. As you answer the questions, think about how often you feel or behave a particular way with your eating. Please select your response.

1. I am relaxed about eating.

Always Often Sometimes Rarely Never

2. I am comfortable about eating enough.

Always Often Sometimes Rarely Never

3. I enjoy food and eating.

Always Often Sometimes Rarely Never

4. I am comfortable with my enjoyment of food and eating.

Always Often Sometimes Rarely

Never

5. I experiment with new food and learn to like it.

Always Often Sometimes Rarely Never

6. I feel it is okay to eat food that I like.

Always Often Sometimes Rarely Never

7. If the situation demands, I can "make do" by eating food I don't much care for.

Always Often Sometimes Rarely Never

8. I eat a wide variety of foods.

Always Often Sometimes Rarely Never

9. I assume I will get enough to eat.

Always Often Sometimes Rarely Never

10. I eat as much as I am hungry for.

Always Often Sometimes Rarely Never

11. I eat until I feel satisfied.

Always Often Sometimes Rarely Never

12. I tune into food and pay attention to myself when I eat.

Always Often Sometimes Rarely Never

13. I make time to eat.

Always Often Sometimes Rarely Never

14. I have regular meals.

Always Often Sometimes Rarely Never

15. I think about nutrition when I choose what I eat.

Always Often Sometimes Rarely Never

16. I generally plan for feeding myself. I don't just grab food when I get hungry.

Always Often Sometimes Rarely Never

WEIGHT RELATED BEHAVIORS QUESTIONNAIRE: Physical Activity Behaviors

Please choose the response that best expresses how well each statement describes you.

1. I find being physically active gives me a lot of energy. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

2. I feel good physically after I've exercised. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

3. I schedule all events in my life around my exercise routine (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

4. I schedule exercise at specific times of the week in order to maintain a routine. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

5. I set goals for myself in order to keep physically active. (Describes me) Not at all; Slightly; More or Less; Pretty Well; Completely

6. I make commitments to exercise and stick to them. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

7. I'm just too lazy to exercise regularly. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

8. I make back up plans to be sure I get enough exercise. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

9. Being physically active gives me a strong sense of accomplishment. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

10. I have too many things to do during the day and can never find time to exercise. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

11. My lack of motivation stops me from being physically active. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

12. When I am exercising, I often feel as though I would rather be doing something else. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

13. Being physically active improves my mood. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

14. I consider being physically active an effective way of relieving stress. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

15. I don't exercise as regularly when I get depressed or upset about something. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

WEIGHT RELATED BEHAVIORS QUESTIONNAIRE: Eating Behaviors

Please choose the response that best expresses how well each statement describes you.

1. I purposefully hold back at meals in order not to gain weight. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

2. I tend to eat more when I am anxious, worried, or tense. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

3. I count calories as a conscious means of controlling my weight. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

4. When I feel lonely I console myself by eating. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

5. I tend to eat more food than usual when I have more available places that serve or sell food. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

6. I tend to eat when I am disappointed or feel let down. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

7. I often refuse foods or drinks offered because I am concerned about my weight. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

8. If I see others eating, I have a strong desire to eat too. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

9. Some foods taste so good I eat more even when I am no longer hungry. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

10. When I have eaten too much during the day, I will often eat less than usual the following day. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

11. I often eat so quickly I don't notice I'm full until I've eaten too much. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

12. If I eat more than usual during a meal, I try to make up for it at another meal. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

13. When I'm offered delicious food, it's hard to resist eating it even if I've just eaten. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

14. I eat more when I'm having relationship problems. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

15. When I'm under a lot of stress, I eat more than I usually do. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

16. When I know I'll be eating a big meal during the day, I try to make up for it by eating less before or after that meal. (Describes me)

Not at all; Slightly; More or Less; Pretty Well; Completely

Mindful Attention Awareness Scale (MAAS)

Below is a collection of statements about your everyday experience. Using the 1-6 scale below, please indicate how frequently or infrequently you currently have each experience. Please answer according to what really reflects your experience rather than what you think your experience should be.

1. Please answer according to what really reflects your experience rather than what you think your experience should be.

-almost always-very frequently-somewhat frequently-somewhat infrequently-very infrequently-almost never

I could be experiencing some emotion and not be conscious of it until some time later.

I break or spill things because of carelessness, not paying attention, or thinking of something else.

I find it difficult to stay focused on what's happening in the present.

I tend to walk quickly to get where I'm going without paying attention to what I experience along the way.

I tend not to notice feelings of physical tension or discomfort until they really grab my attention.

I forget a person's name almost as soon as I've been told it for the first time.

It seems I am "running on automatic" without much awareness of what I'm doing.

I rush through activities without being really attentive to them.

2. Please answer according to what really reflects your experience rather than what you think your experience should be.

-almost always -very frequently -somewhat frequently

-somewhat infrequently -very infrequently -almost never

I get so focused on the goal I want to achieve that I lose touch with what I am doing right now to get there.

I do jobs or tasks automatically, without being aware of what I'm doing.

I find myself listening to someone with one ear, doing something else at the same time.

I drive places on "automatic pilot" and then wonder why I went there.

I find myself preoccupied with the future or the past.

I find myself doing things without paying attention.

I snack without being aware that I'm eating.

Physical Activity Readiness Questionnaire (PAR-Q)

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people, physical activity should not pose any problems or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advise concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read the carefully and check **YES** or **NO** opposite the question if it applies to you. If yes, please explain.

<u>YES</u><u>NO</u>

	1.	Has your doctor ever said you have heart trouble?
	2	Do you frequently have pains in your heart and chest?
	2.	Ves
	3	Do you often feel fain or have spells of severe dizziness?
	5.	Yes
	4.	Has a doctor ever said your blood pressure was too high?
		Yes.
	5.	Has your doctor ever told you that you have a bone or joint
problem(s	s), such as a	thritis that has been aggravated by exercise, or might be
made		
worse wit	th exercise?	
		Yes,
	6.	Is there a good physical reason, not mentioned here, why
you shoul	ld notfollow	an activity program even if you wanted to?
		Yes,
	7.	Are you over age 60 and not accustomed to vigorous
exercise?		
		Yes,
	8.	Do you suffer from any problems of the lower back, i.e.,
chronic p	ain, or numb	oness?
		Yes,
	9.	Are you currently taking any medications? If YES, please
specify.		
		Yes,
	10.	Do you currently have a disability or a communicable
disease?	If YES,	
		Please specify,
		Yes

If you answered NO to all questions above, it gives a general indication that you may participate in physical and aerobic fitness activities and/or fitness evaluation testing. The fact that you answered NO to the above questions, is no guarantee that you will have a normal response to exercise. If you answered Yes to any of

the above questions, then you may need written permission from a physician before participating in physical and aerobic fitness activities and/or fitness evaluation testing.

International Physical Activity Questionnaire

INSTRUCTIONS: We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the last 7 days. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the vigorous activities that you did in the last 7 days. Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

1. During the last 7 days, on how many days did you do vigorous physical activities like heavy lifting, digging, aerobics, or fast bicycling?

0 days per week
1 day per week
2 days per week
3 days per week
4 days per week
5 days per week
6 days per week
7 days per week

2. How much time did you usually spend doing vigorous physical activities on one of those days?

Think about all the moderate activities that you did in the last 7 days. Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

1. During the last 7 days, on how many days did you do moderate physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

0 days per week 1 day per week 2 days per week 3 days per week 4 days per week5 days per week6 days per week7 days per week

2. How much time did you usually spend doing moderate physical activities on one of those days?

Think about the time you spent walking in the last 7 days. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

1. During the last 7 days, on how many days did you walk for at least 10 minutes at a time?

0 days per week

- 1 day per week
- 2 days per week
- 3 days per week
- 4 days per week
- 5 days per week
- 6 days per week
- 7 days per week

2. How much time did you usually spend walking on one of those days?

This question is about the time you spent sitting on weekdays during the last 7 days. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

1. During the last 7 days, how much time did you spend sitting on a week day?

2. During the last 7 days, on how many days did you do any strength training activities designed to strengthen muscles such as lifting weights, push-ups, or sit-ups?

1 day 2 days 3 days 4 days 5 days 6 days 7 days

No strengthening exercises

3. How much time did you usually spend doing strength training activities on

one of those days?

Physical Activity Behaviors

Directions: For each of the statements below, please choose the response which describes you best.

-Not at all -Slightly -More or Less -Pretty Well -Completely

I find being physically active gives me a lot of energy.

I feel good physically after I've exercised.

I schedule all events in my life around my exercise routine.

I schedule exercise at specific times of the week in order to maintain a routine.

I set goals for myself in order to keep physically active.

I'm often so tired that I don't have enough energy to exercise.

I'm just too lazy to exercise regularly.

I make back up plans to be sure I get enough exercise.

Being physically active gives me a strong sense of accomplishment.

My lack of motivation stops me from being physically active.

When I am exercising I often feel as though I would rather be doing something else.

I consider being physically active an effective way of relieving stress.

Being physically active improves my state of mind.

I don't exercise as regularly when I get depressed or upset about something.

I try to exercise at the same time on the same days each week to help stay physically active.

University of Rhode Island Pittsburgh Sleep Quality Index

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

1. During the past month, when have you usually gone to bed?

2. How long (in minutes) has it taken you to fall asleep each night?

3. When have you usually gotten up in the morning?

4. How many hours of actual sleep do you usually get (This may be different than the number of hours you spend in bed)?

University of Rhode Island Pittsburgh Sleep Quality Index (continued)

1. During the past month, how often have you had trouble sleeping because you...

-not during the past month -less than once a week week -three or more times a week

-once or twice a

Cannot get to sleep within 30 minutes

Wake up in the middle of the night or early morning

Have to get up to use the bathroom

Cannot breathe comfortably

Cough or snore loudly

Feel too cold

Feel too hot

Have bad dreams

Have pain

During the past month, how often have you taken medicine (prescribed or overthe-counter) to help you sleep?

During the past month, how often have you had trouble staying awake while in class, studying, eating meals, or engaging in social activity?

During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?

Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s):

Please describe:

2. During the past month, how would you rate your sleep quality overall?

Very good Fairly good Fairly bad Very bad

Motivational Change

1. Rate the degree to which the following MOTIVATED you to make a change to improve your health

-Not at all motivating -Slightly motivating -Moderately motivating -Highly motivating -Most motivating

Completing the on-line surveys Feedback on my BMI Feedback on my waist circumference Feedback on my blood pressure Attending the classes Completing the homework for class

Ineligible

Based on your previous response, you are ineligible to participate in this study. Thank you for your interest.

If you have any questions regarding your ineligibility please email Emily Cook or Chelsea Smith at lipidlab@etal.uri.edu. You may close out this screen at this time.

Thank You!

Thank you for participating in the BFF Survey! We will be in contact with you soon and schedule your next appointment.

You may close out this screen at this time.

Appendix D: BFF Phase 2 Baseline Participant Survey

BFF Baseline Survey
Welcome to the BFF Study!
Please answer all of the following questions.
Personal Information
Please answer all of the following questions
1. What is your gender?
2. What is your current age?
3. What is your race or ethnic group?
O White (not of Hispanic origin)
O Black (not of Hispanic origin)
O Hispanic/Latino
O American Indian/Alaska Native
O Asian/Pacific Islander
Mixed Race or Other (please specify)
4. Where do you live? (During the URI 09-10 school year)
On-Campus
Off-Campus

5. What is your major?

- O Agricultural Sciences
- Biological Sciences
- O Business Administration
- C Education
- C English
- O Fine Arts
- O Humanities
- C Kinesiology/Physical Education
- O Nutrition and Food Sciences
- O Nursing
- Social Science
- O Undeclared
- Other

Consent Form

Can I participate? You must be • 18-24 years of age • A URI student • Willing to complete an on-line questionnaire.

You must not Be: • Pregnant or lactating

What will I be asked to do?If you choose to participate, you will be asked to:Complete an online survey (approximately 20 minutes).

What do I get out of It? You may be eligible to participate in a weight-loss study being done by the Nutrition and Food Science Department.

Risks There is no risk in participating in this survey.

Voluntary Participation Your participation is voluntary. You may stop participating in this survey at any point.

Confidentiality All information that you provide will be kept confidential and your privacy will be protected to the

maximum extent allowable by law. The website is password protected for both the researcher and subjects. The data will be stored on a disk in the Lipid Lab at the University of Rhode Island. Printouts of the data will be stored in locked offices at The University of Rhode Island for up to 5 years (as required by law) and then destroyed. Data will be reported in summary format, and no names will be used.

Questions

If you have any questions or concerns, please contact the researchers listed below. If you have concerns regarding your rights as a research participant, please contact the human subjects representative listed below.

Researchers:

Emlly Cook email: lipidlab@etal.uri.edu (Subject: BFF) or Ingrid Lofgren email: ingridlofgren@uri.edu

Human Subjects Representative: Vice President for Research and Economic Development 70 Lower College Road University of Rhode Island Kingston, RI 02881 Phone (401) 874-4328. email: robind@uri.edu

This project has been reviewed and approved by the human subject review board of the University of Rhode Island.

Thank you for your time and interest in this study.

Continuing in this study indicates that you have read and understand the above information. If you would like a copy of this form, please print it now.

1. Continuing in this study indicates that you have read and understand the above information.

- \bigcirc I am ready to begin the survey
- I am not interested in this study

Personal Information

1. Please enter your URI student ID (All information will remain

confidential)

2. What is your e-mail?

3. What is a phone number where we can reach you?

dv Weight	History				
1. What is th	ecessary:	e you have i	maintained yo	ur present	weight?
	· · · ·				
2. How tall a	are you? (Please	e round to t	he nearest ind	ch)	
Example: If the box next Feet	you are 5 feet 8 t to inches	inches tall,	, put 5 in the t	oox next to	feet and 8 i
3. How muc	h do you weighi	? (Please ro	ound to the ne	arest poun	d)
			<i>i</i> – 1		areat pound
		s to succieda?			
4. How much Pounds 5. How many	h would you like	e to weigh? r weight fluc	(Please round	east 5 lbs i	in the last
4. How much Pounds 5. How many year? Please 6. Please des occurred in t	h would you like y times has you e explain if nece scribe any signif the past (e.g., lo	e to weigh? r weight flue ssary. ficant chang ost 50 lb. in	(Please round ctuated by at l ges in your we 2006):	east 5 lbs i	in the last nay have
4. How much Pounds 5. How many year? Please 6. Please dea occurred in the C I have not explanate the second	h would you like y times has you e explain if nece scribe any significant the past (e.g., lo	e to weigh? r weight flue essary. ficant chang ost 50 lb. in	(Please round ctuated by at l ges in your we 2006):	east 5 lbs i	in the last nay have
4. How much Pounds 5. How many year? Please 6. Please des occurred in the C I have not explored I have had signified () I have had signifi	h would you like y times has you e explain if nece scribe any significant the past (e.g., lo perienced any significant gnificant changes in my v	e to weigh? r weight flue essary. ficant chang ost 50 lb. in r weight changes t weight. Please exp	(Please round ctuated by at l ges in your we 2006): to date. plain:	east 5 lbs i	in the last nay have
4. How much Pounds 5. How many year? Please 6. Please des occurred in the O I have not explored I have had sign 7. How would few years?	h would you like y times has you e explain if nece scribe any significant the past (e.g., lo perienced any significant gnificant changes in my v Id you describe f	e to weigh? r weight flue essary. ficant change ost 50 lb. in tweight. Please exp the typical weight	(Please round ctuated by at l ges in your we 2006): to date. blain:	east 5 lbs i east 5 lbs i ight that m parents or	in the last nay have ver the last
4. How much Pounds 5. How many year? Please 6. Please des occurred in the C I have not ext C I have not ext C I have had sign 7. How would few years? Your Mother	h would you like y times has you e explain if nece scribe any significant the past (e.g., lo perienced any significant gnificant changes in my v ld you describe f Under-weight	e to weigh? r weight flue essary. ficant change ost 50 lb. in t weight changes t weight. Please exp the typical t Just right	(Please round ctuated by at l ges in your we 2006): to date. blain:	east 5 lbs i east 5 lbs i ight that m obese	in the last nay have ver the last unknown

	Bulvey
8. What is the walking, jogg normal daily i	amount of moderate or vigorous activity (such as brisk ing, biking, aerobics, or yard work) you do in addition to your routine, most days?
○ Less than 30 m	inutes
🔿 30 - 60 minute	5
O More than 60 n	linutes
9. How many	hours do you work (a paying/non-paying job)per week?
O 0-3 hours	
4-10 hours	
10-20 hours	
O 20-30 hours	
O 30-40 hours	
O More than 40 h	ours
o No	
1. On average per day?	e, how many meals (i.e., breakfast, lunch, dinner) do you eat
2. On average fruit) do you Number of snacks	e, how many snacks (i.e.; candy/energy bars, chips, pieces of eat per day?

4. How fast is your rate of eating?

- very slow
- relatively slow
- 🔿 medium
- O relatively fast
- 🔿 very fast

5. Choose the statement that best describes your typical eating behavior.

- $\bigcirc\$ I always eat whatever I want, whenever I want.
- $\bigcirc\$ I often eat whatever I want, whenever I want.
- $\bigcirc\$ I only sometimes eat whatever I want, whenever I want.
- \bigcirc I often refrain from eating what I want but often "give in" and eat it anyway.
- \bigcirc I often refrain from eating what I want and sometimes "give in" and eat it anyway.
- $\odot~$ I often refrain from eating what I want and never "give in" and eat it anyway.

pharm questions

1. Do you drink bottled water?

- O Yes
- 🔿 No

2. If you answered yes to the previous question, how much? (in ounces/day)?

3. Do you drink water/fluids from hard-plastic containers or cups - specifically ones that have a **#7** on the bottom?

- O Yes
- 🔿 No

🔿 Don't know

4. If you answered yes to the previous question, how much (in ounces/day)?

6 d	. How many servings of canned fruit/vegetables/foods do you eat per lav?
n	npliance questions
1 n	. How many resources, out of the following list, have you used to get autrition, diet, or physical activity information in the last three months?
D	octor
N	lurse
N	lutritionist/Registered Dietitian
P F	ersonal Trainer
F	amily Member of Friend
N	
v	Vehsites
в	iloas
Γ	
2 fi A	. In the last three months have you used and diet/nutrition information rom the American Dietetic Association (ADA), My Pyramid Guidelines, or American Heart Association (AHA)?
Γ	
3	. Within the last three months, on average how many hours PER WEEK
h	ave vou spent reading, being taught, learning about, or doing
d	liet/nutrition and physical activity?
Γ	

8 large strawberries

2 large plums 32 seedless grapes 1 cup 100% juice 1/2 cup cooked fruit

1. About how many cups of FRUIT (including 100% pure fruit juice) do you eat or drink each day?

- O None
- 🔿 1/2 to 1 cup
- ① 1 to 2 cups
- O 2 to 3 cups
- 3 to 4 cups
- 4 cups or more

2. How many cups of FRUIT do you think you SHOULD eat each day for good health?

- O None
- 1/2 to 1 cup
- 1 to 2 cups
- O 2 to 3 cups
- 🔿 3 to 4 cups
- 4 cups or more

Vegetable Intake

Please use the following information to answer the questions on this page:

1 CUP OF VEGETABLES EQUALS

- 1 cup of sliced/chopped vegetable(amounting to approximately the size of your fist)
- 5 broccoli spears 5 inches long
- 1 cup cooked leafy greens
- 2 cups lettuce or raw greens
- 12 baby carrots
- 1 large potato or sweet potato
- 2 large celery sticks
- 1 cup cooked beans

1. About how many cups of VEGETABLES (including 100% vegetable juice) do you eat or drink each day?

- None
- 1/2 to 1 cup
- 🔿 1 to 2 cups
- O 2 to 3 cups
- 🖸 3 to 4 cups
- ⊙ 4 cups or more

2. About how many cups of VEGETABLES (including 100% vegetable juice) do you think you SHOULD eat each day for good health?

- None
- 1/2 to 1 cup
- 1 to 2 cups
- O 2 to 3 cups
- ⊙ 3 to 4 cups
- 4 cups or more

pyramid questions

How much of the following food items do you think you need to consume PER DAY to maintain a HEALTHY diet?

1. Whole Grains

2. Saturated Fat

3. Monounsaturated and Polyunsaturated Fats

4. Fiber

L

L

5. Sodium

BFF Baseline Survey
6. Low Fat Dairy Products
Eating Competence
These questions are about your eating and food-providing skills, attitudes, and behaviors. As you answer the questions, think about how often you feel or behave a particular way with your eating. Please select your response.
1. I am relaxed about eating.
2. I am comfortable about eating enough.
3. I enjoy food and eating.
4. I am comfortable with my enjoyment of food and eating.
5. I experiment with new food and learn to like it.
6. I feel it is okay to eat food that I like.
7. If the situation demands, I can "make do" by eating food I don't much care for.
8. I eat a wide variety of foods.
9. I assume I will get enough to eat.
10. I eat as much as I am hungry for.
11. I eat until I feel satisfied.

BFF Baseline Survey
12. I tune into food and pay attention to myself when I eat.
13. I make time to eat.
14. I have regular meals.
15. I think about nutrition when I choose what I eat
16. I generally plan for feeding myself. I don't just grab food when I get hungry.
Behaviors
Please choose the response that best expresses how well each statement describes you.
1. I find being physically active gives me a lot of energy. (Describes me)
2. I feel good physically after I've exercised. (Describes me)
3. I schedule all events in my life around my exercise routine (Describes me)
4. I schedule exercise at specific times of the week in order to maintain a routine. (Describes me)
5. I set goals for myself in order to keep physically active. (Describes me)
6. I make commitments to exercise and stick to them. (Describes me)

BFF Baseline Survey
7. I'm just too lazy to exercise regularly. (Describes me)
8. I make back up plans to be sure I get enough exercise. (Describes me)
9. Being physically active gives me a strong sense of accomplishment. (Describes me)
10. I have too many things to do during the day and can never find time to exercise. (Describes me)
11. My lack of motivation stops me from being physically active. (Describes me)
12. When I am exercising, I often feel as though I would rather be doing something else. (Describes me)
13. Being physically active improves my mood. (Describes me)
14. I consider being physically active an effective way of relieving stress. (Describes me)
15. I don't exercise as regularly when I get depressed or upset about something. (Describes me)
WEIGHT RELATED BEHAVIORS QUESTIONNAIRE: Eating Behaviors
Please choose the response that best expresses how well each statement describes you.
1. I purposefully hold back at meals in order not to gain weight. (Describes me)

Γ

F Baselir	ne Survey
2. I tend t	o eat more when I am anxious, worried, or tense. (Describes me
3. I count	calories as a conscious means of controlling my weight.
(Describe:	s mej
4. When I	feel lonely I console myself by eating. (Describes me)
5. I tend t	o eat more food than usual when I have more available places
that serve	or sell food. (Describes me)
6. I tend t	o eat when I am disappointed or feel let down. (Describes me)
7. I often	refuse foods or drinks offered because I am concerned about my
weight. (D	Describes me)
8. If I see	others eating, I have a strong desire to eat too. (Describes me)
9. Some fo	oods taste so good I eat more even when I am no longer hungry.
(Describe	s me)
10. When	I have eaten too much during the day, I will often eat less than
usual the	following day. (Describes me)
11. I ofter	eat so quickly I don't notice I'm full until I've eaten too much.
(Describe	s me)
12. If I ea	t more than usual during a meal, I try to make up for it at anothe
meal. (De	scribes me)

13. When I'm offered delicious food, it's hard to resist eating it even if I've just eaten. (Describes me)

14. I eat more when I'm having relationship problems. (Describes me)

15. When I'm under a lot of stress, I eat more than I usually do. (Describes me)

16. When I know I'll be eating a big meal during the day, I try to make up for it by eating less before or after that meal. (Describes me)

17. What was the date of your last menstrual cycle? (MM-DD-YYYY)

Mindful Attention Awareness Scale (MAAS)

Below is a collection of statements about your everyday experience. Using the 1-6 scale below, please indicate how frequently or infrequently you currently have each experience. Please answer according to what really reflects your experience rather than what you think your experience should be.

1. Please answer according to what really reflects your experience rather than what you think your experience should be.

	almost always	very frequently	somewhat frequently	somewnat infrequently	very infrequently	almost never
I could be experiencing some emotion and not be conscious of it until some time later.	С	C	0	C	0	С
I break or spill things because of carelessness, not paying attention, or thinking of something else.	O	C	O	O	0	C
I find it difficult to stay focused on what's happening in the present.	С	O	0	O	0	0
I tend to walk quickly to get where I'm going without paying attention to what I experience along the way.	O	C	O	0	0	O
I tend not to notice feelings of physical tension or discomfort until they really grab my attention.	C	С	O	C	0	С
I forget a person's name almost as soon as I've been told it for the first time.	C	C	O	Ó	O	Ō
It seems I am "running on automatic" without much awareness of what I'm doing.	С	С	0	C	0	C
I rush through activities without being really attentive to them.	C	o	O	O	O	C

2. Please answer according to what really reflects your experience rather than what you think your experience should be.

	almost always	very frequently	somewhat frequently	somewhat infrequently	very infrequently	almost never
I get so focused on the goal I want to achieve that I lose touch with what I am doing right now to get there.	С	C	0	C	0	C
I do jobs or tasks automatically, without being aware of what I'm doing.	O	O	C	Ō	O	O
I find myself listening to someone with one ear, doing something else at the same time.	C	C	C	C	C	¢
I drive places on "automatic pilot" and then wonder why I went there.	O	C	O	O	O	C
I find myself preoccupied with the future or the past.	C	O	О	C	C	C
I find myself doing things without paying attention.	0	0	0	0	O	0
I snack without being aware that I'm eating.	C	O	O	O	O	O

International Physical Activity Questionnaire

INSTRUCTIONS: We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the last 7 days. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

International Physical Activity Questionnaire (continued)

Think about all the vigorous activities that you did in the last 7 days. Vigorous physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

1. During the last **7** days, on how many days did you do vigorous physical activities like heavy lifting, digging, aerobics, or fast bicycling?

- O days per week
- 1 day per week
- O 2 days per week
- 3 days per week
- 4 days per week
- 5 days per week
- 6 days per week
- 🔿 7 days per week

ipaq2

1. During the days you did do vigorous physical activities how much time did you usually spend doing those activities?

(MINUTES)

International Physical Activity Questionnaire (continued)

Think about all the moderate activities that you did in the last 7 days. Moderate activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

1. 3. During the last **7** days, on how many days did you do moderate physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

- O days per week
- 1 day per week
- O 2 days per week
- 3 days per week
- O 4 days per week
- 5 days per week
- 6 days per week
- 7 days per week

ipaq2.2

1. During the days you did

do moderate physical

activities how much time did

you usually spend doing

those activities?

Amount of time

(MINUTES)

International Physical Activity Questionnaire (continued)

Think about the time you spent walking in the last 7 days. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

BFF Baseline Survey
1. During the last 7 days, on how many days did you walk for at least 10
minutes at a time?
C 0 days per week
C 1 day per week
○ 2 days per week
O 3 days per week
O 4 days per week
O 5 days per week
O 6 days per week
O 7 days per week
Ipaq2.3
1. How much time did you
usually spend walking on
one of those days?
Amount of time
(MINUTES)
International Physical Activity Questionnaire (continued)
This question is about the time you spent sitting on weekdays during the last 7 days. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.
1. During the last 7 days,
how much time did you
spend sitting on a week day?
Amount of time
(MINUTES)

BFF Baseline Survey					
2. During the last 7 days, on how many days did you do any strength					
training activities designed to strengthen muscles such as lifting weights,					
push-ups, or sit-ups?					
O no strengthening exercises					
🔿 1 day					
○ 2 days					
O 3 days					
○ 4 days					
○ 5 days					
O 6 days					
O 7 days					
3 How much time did you					
usually spend doing strength					
training activities on one of					
those days?					
Amount of					
University of Rhode Island Pittsburgh Sleep Quality Index					
Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.					
1. During the past month when have you usually gone to bed?					
USUAL BED TIME:					
2. How long (in minutes) does it take you (on average) to fall asleep each					
night?					
# OF MINUTES:					
3. When do you usually get up in the morning?					
11ME:					

4. How many hours of actual sleep do you usually get (This may be different than the number of hours you spend in bed)?

HOURS OF SLEEP PER

NIGHT:

University of Rhode Island Pittsburgh Sleep Quality Index (continued)

1. During the past month, how often have you had trouble sleeping because you...

	not during the past month	less than once a week	once or twice a week	three or more times a week
Cannot get to sleep within 30 minutes	O	O	C	0
Wake up in the middle of the night or early morning	O	O	C	0
Have to get up to use the bathroom	O	O	С	0
Cannot breathe comfortably	O	C	C	0
Cough or snore loudly	0	0	C	0
Feel too cold	Ō	\odot	C	O
Feel too hot	0	O	C	0
Have bad dreams	O	O	O	0
Have pain	O	O	C	0
During the past month, how often have you taken medicine (prescribed or over- the-counter) to help you sleep?	0	C	С	O
During the past month, how often have you had trouble staying awake while in class, studying, eating meals, or engaging in social activity?	C	C	С	O
During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?	Ō	O	C	O
Other reason(s), please describe, including how often you have had trouble	O	O	С	0
BFF Baseline Survey				

sleeping because of this reason(s):				
Please describe:				
2. During the past month, how would you rate your sleep quality overall?				
○ Very good				
○ Fairly good				
C Fairly bad				
🔘 Very bad				
Motivational Change				
become more active and eat a healthy diet. PLEASE DESCRIBE IN THE BOX BELOW WHY YOU SELECTED YOUR WHEREABOUTS. 1 = Not ready to change 10 = Ready to change 1 0 2 0 3 0 4 0 5 0 6 0 7 0 8 0 9 0 10 Please Explain:				
Ineligible				
Based on your previous response, you are ineligible to participate in this study or have decided not to participate. Thank you for your interest.				
If you have any questions regarding your ineligibility please email Emily Cook or Chelsea Smith at lipidlab@etal.uri.edu. You may close out this screen at this time.				
Thank You!				
Thank you for participating in the BFF Survey! We will be in contact with you soon and schedule your next appointment.				
close out screen				

BFF Baseline Survey

You may close out of this screen at this time. Thank you!

Appendix E: BFF Consent Form for Research

The University of Rhode Island Department of Nutrition & Food Sciences 106 Ranger Hall, Kingston, RI 02881

CONSENT FORM FOR RESEARCH

Title of Project: The University of Rhode Island Best Foot Forward (BFF) Study.

You have been asked and have elected to take part in the research project described below. The researchers will explain the project to you in detail. Please feel free to ask questions. If you have more questions later, Dr. Ingrid Lofgren (401-874-5706), or Emily Cook (401-874-2785) from the Department of Nutrition and Food Sciences at the University of Rhode Island (URI), will discuss them with you.

Description of the project:

The primary purpose of this study is to assess the impact of two combined dietary and exercise interventions on chronic disease health risks in young, female adults. This study will compare various outcomes after eight weeks of intentional weight loss in the two groups. One group, the Manual Intervention Group, will receive diet and physical activity education via manual instruction only at baseline and will receive no additional contact with study staff until post-intervention. The other group, the Step Forward Intervention Group, will receive diet and physical activity education via group and one-on-one meetings. Participants in both groups will be assessed for changes in weight, body composition, blood pressure, blood sugar and lipids, nutrient metabolism, urine properties, and how certain genes may influence the changes. To participate in this study, you must be female, between 18 and 24 years old, and you may not be pregnant or lactating. You are not eligible for this study if you have a BMI of less than 25 kg/m². You are not eligible for this study if you have diabetes (Type I or Type II), cancer, coronary heart disease, liver disease, a bleeding disorder, or if you are on lipid lowering medications. All phases of this study will be completed on the URI campus. You will receive \$150 once you complete all parts of this study.

What will be done:

If you choose to participate, the study requires your involvement in 4 phases. During phase 1, you will complete 4 preliminary assessment visits over 2 weeks. Phase 2 will last 8 weeks and all participants will receive dietary education and pedometers to use. During phase 2 some participants will be involved in additional activities that include weekly meetings. For phase 3 you will complete 4 postassessment visits similar to the phase 1 assessment visits. Phases 1 through 3 will be completed before the end of this semester. During phase 4 you will be contacted 6 months after the completion of phase 3. Phase 4 includes a total of 4 visits with similar activities to the assessment visits in phase 1.

Phase 1: Assessment:

Assessment Visit 1 (20-30 minutes):

• You will be asked questions about what you ate the day before. You do not need to write anything down prior to coming.

Day Prior to Assessment Visit 2:

• For the 12 hours prior to the second assessment visit, you will be asked not to eat or drink anything unless it's water. We encourage you to drink as much water as you would like. For example, if your screening visit is scheduled for 8 am on Tuesday do not eat or drink anything except water after 8 pm on Monday evening.

Assessment Visit 2 (20-30 minutes):

• Your height, weight, and waist circumference will be measured.

Assessment Visit 2 (20-30 minutes - continued):

- A 7-ml blood draw (less than 1 tablespoon) will be taken. The blood draw will be used to analyze blood sugar and lipids, molecules that impact metabolism-like hormones, blood proteins, and your DNA (genetic material).
- A urine sample will be collected in a glass or plastic urine specimen cup labeled with a non-identifying ID number. You will be sent to private, gender specific bathroom to collect the sample.

Day Prior to Assessment Visit 3:

• Again, for the 12 hours prior to the second assessment visit, you will be asked not to eat or drink anything unless it's water.

Assessment Visit 3 (20-30 minutes):

- Blood pressure will be measured.
- A 42-ml blood draw (approximately 4 tablespoons) will be taken.
- A urine sample will be collected in a private gender specific bathroom.

Assessment Visit 4 (90 minutes):

- Your percent body fat will be performed using an air-displacement machine ("BodPod") located in the Independence Square II building. This will require you to sit still in a chamber a wearing a bathing suit for about 10 minutes at a time totaling less than 30 total minutes for the entire procedure.
- VO_{2max} will be measured on a treadmill using a reviewed protocol. The test will begin at a 2.5% grade and will remain at a constant speed of 3 mph throughout the length of the test. The percent grade will slowly increase throughout the duration of the test at a 2.5% increase every 2 minutes. The test will continue until you signal that you are exhausted. You will wear a mouthpiece (like that worn by a scuba diver, connected to a small tube) during the entire test. You will be provided with your own mouthpiece for the duration of the study.
- A urine sample will be collected in a private, gender specific bathroom.

Phone Calls:

• You will receive two phone calls from research staff prior to the start of phase 2. You will be asked questions about what you ate the day before. You do not need to write anything down prior to these phone calls.

Phase 2: Intervention:

• At the end of the phase 1 (assessment), you will be <u>randomly</u> assigned to either the <u>Manual Intervention Group</u> or the <u>Step Forward Intervention</u> <u>Group</u> for the second phase. You are not allowed to choose which group you will be assigned to. Participants in each group, the Manual Intervention Group and the Step Forward Intervention Group, will be given dietary and physical activity information and instructed on how to use a study-provided pedometer. The goal of both intervention Group will also participate in regularly scheduled group and one-on-one meetings with research staff at to review nutrition and physical activity information.

Phase 3: Post Assessment Visits:

• The four post assessment procedures and two phone calls are similar to the phase 1 assessment visits.

Phase 4: Six month follow-up:

• In fall 2009 you will be contacted via telephone and/or e-mail to complete the follow-up assessment visits. The four post assessment visits and two phone calls are very similar to the phase 1 and 3 assessment visits.

Benefits of this study:

Participating in this study may result in weight-loss and may reduce your risk for heart disease. Participation will result in increased knowledge of weight-loss techniques, healthy eating, and physical activity information. You may keep the study-provided pedometers once the study is over. You will gain knowledge about your health status (examples: blood pressure, total cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol, and triglycerides), your body composition (examples: BMI, % lean body mass), and your cardiovascular fitness. These values are not diagnostic, however, if you are concerned, you can share the results with your health practitioner and tests can be repeated if deemed necessary. The genetic information we obtain from your blood sample will help the researchers in this study better understand the role genetics plays in chronic disease risks for young adults. The information we obtain from your urine sample will be analyzed for the presence of various compounds that provide information about nutrient metabolism such as bis-phenol A. There will be no direct benefit to you from genetic or urine analysis as you will not be provided with these results or information regarding your DNA or urine tests.

Risks or discomfort:

There are no known risks for the following procedures: completing the questionnaires, having height, weight, and waist circumference measured, body composition via the BodPod and urine collection. Even though experienced personnel will perform the blood draw using a sterile technique, there are a few risks associated with the procedure: there is a small chance of discomfort from the needle, bruising, formation of blood clot, and infection. A trained professional will perform the blood draw and we will take all steps possible to minimize the risks to you.

It is possible that you could slip or fall while running on the treadmill for the VO_{2max} test. For your safety you will be reminded to focus your attention on the treadmill belt and to stay in the center of the treadmill. Also there is a support railing at the front of the treadmill, and a technician will be stationed at the treadmill during all tests. Tiredness may occur after finishing the VO_{2max} test.

Confidentiality:

All information collected in this study is confidential, and your name will not be identified and linked to any study data at any time to anyone other than the principle investigators of the study. All study data, including this consent form, will be locked and secured in our study lab (Ranger Hall, room 301). All

computer-based data will be kept on a computer that is password protected and all study paperwork will be stored in a locked file cabinet (locked file cabinet and password protected computer are in Ranger 301). An individual code will be assigned to you and will not be directly associated with your name. The researchers and URI will protect your privacy, unless they are required by law to report information to city, state or federal authorities, or give information to a court of law.

Genetic testing:

Previous research indicates that certain gene variations might influence body composition and lipid metabolism and exercise responses to exercise training and weight-loss. Only gene variations that are related to body composition, nutrient metabolism, and exercise responses will be examined using your DNA sample. Your DNA sample will be coded only with a study number without any personal identifiers (including initials or birth dates). A sample of your DNA will be collected during blood draw one. Your DNA sample will be stored for up to 30 years in order to allow for pooling of data and more thorough analyses with future study data. For the 30 year storage period, URI will maintain a record of your personal identification (signed informed consent documentation) that is linkable to your DNA sample by your subject ID. This information will be locked in a file cabinet for sample location purposes if necessary. The information obtained from your DNA will be held at URI, at the lipid lab, for 30 years and will be shared only with researchers from the URI in the form of a computerized database free of personal identifiers. Should the principle investigator (PI) relocate to a different institution after the completion of the study, your sample may also be moved to that institution with the PI. However, you still retain the right to have your sample destroyed by contacting the PI or URI's Vice President for Graduate Studies, Research and Outreach. NO ADDITIONAL GENETIC TESTING beyond what you are consenting to by signing this document will be performed without your written permission. If you agree, you may be contacted during the 30 year storage period by these or other researchers associated with this project for permission to use your sample in research beyond the scope of this study. After the 30 year time period, all remaining samples will be destroyed. If you wish to have your DNA destroyed for these future analyses, you may do so by contacting Dr. Ingrid Lofgren (401-874-5706).

Urine Samples:

The urine samples will be sent to Angela Slitt, Professor of Biomedical and Pharmaceutical Sciences, here at URI for analysis. Urine samples will be analyzed for the presence of various compounds that provide information about nutrient metabolism such as bis-phenol A. Bis-phenol A is a metabolite found in plastic food and drink containers.

In case there is any injury to the subject:

If you have any injury or discomfort as a result of the study, you should notify Dr. Ingrid Lofgren at (401) 874-5706, or Emily Cook (401-874-2785). Additionally, if this study causes you any injury, you should write or call the office of the Vice Provost for Graduate Studies, Research and Outreach, 70 Lower College Road, University of Rhode Island, Kingston, Rhode Island, telephone: (401) 874-4328.

Decision to quit at any time:

It is your decision and your decision alone whether or not you consent to participate in this study. You are free to ask questions about this study before you decide whether or not to consent to participate in it. If you consent to participate in the study you are free to withdraw from participation at any time without penalty or coercion, or without any requirement that you provide an explanation to anyone of your decision to withdraw.

Incentives:

You will receive partial compensation (\$100.00) after completing phase 1, 2, and 3 of the study. You will receive an additional compensation (\$50.00) after completion of phase 4.

You have read and understand the above information in the consent form and have been given adequate opportunity to ask the investigators any questions you have about the study. Your questions, if any, have been answered by the investigators to your satisfaction. Your signature on this form means that you understand the information and you agree to voluntarily participate in this study.

Signature of Participant

Signature of Researcher

Typed/printed Name

Typed/printed name

Date

Date

By signing again below you understand that a portion of your blood and/or DNA sample may also be used for potential future studies by the principle investigators

only. Your sample will be used only to examine body composition, nutrient metabolism, and bodily responses to exercise training and weight loss.

Signature of Participant

Date

Printed name of Participant

Date

Signature of Researcher

Date

By signing a third time below you are stating that you are willing to be contacted for involvement in future studies done at URI.

Signature of Participant mail

Contact phone number and E-

Appendix F: BFF Assessment Materials

NAME:	BIRTH	IDATE:
HOW DID YOU HEAR	ABOUT THIS STUI	DY?
MAJOR:	STUDI	ENT ID:
HOME ADDRESS (OR C	CONACT INFORM	ATION INCASE YOU MOVE):
DAY PHONE #:	C	ELL PHONE #:
E-MAIL ADDRESS:		
BEST WAY TO CONTA	CT YOU (Check an	y applicable choice):
e-mailday	phone cell pho	one other, specify:
		••••••
NOTE: THE FOLLOW	ING IS FOR COM	PENSATION PURPOSES ONLY.
PAYMENT # 2 DATE	PPT ID #:	
6 MONTH FOLLO	W UP ASSESSME	NT
3 DIETARY ASSESSME	′ RECALLS ENT SHEET COMP	LETE
I THE (PARTICIPANT	HAVE RE(CEIVED \$50.00 FOR PARTICIPATION IN
BFF STUDY.		
PPT SIGNATURE:		DATE:
	•••••	••••••
PAYMENT #1 DATE	PPT ID #:	
BASELINE ASSES	SMENT	POST INTERVENTION
3 DIETARY	RECALLS	3 DIETARY RECALLS

COMP	ASSESSMENT SHEET COMPLETE ASSESSMENT SHEET LETE
8	WEEKS
T	HAVE RECEIVED \$100 00 FOR PARTICIPATION
IN THI	E (PARTICIPANT NAME) STUDY.
PPT SI	GNATURE: DATE:
	Assessment Checklist
Date:	Time:a.m. Visit: Assessment#1 In-person recall
visit 1	or 2
Partic	ipant: Subject ID No.
Assess <u>Questi</u>	ment evaluator:
1.	At what time did you last have something to eat? a.m. or p.m.
2.	At what time did you last have something to drink? a.m. or p.m.
3.	Have you had any caffeine, tobacco, or tobacco products today? Yes No If yes, please have participant explain: What: When: a.m. or p.m.
4.	Are you currently ill? Yes No If yes have participant explain:

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Have you ever fainted or passed out while having blood drawn? Yes
 No

If yes	, have participant
explain:	

6. Is there any reason you feel you are unable to participate in testing today? Yes No

If yes, have participant explain:

7. Please ask participant if there is any additional information they would like to provide to the assessment staff member:

Reasons to Reschedule

_

- 1. Reschedule if participant has eaten in the last 12 hours.
- 2. Reschedule if participant drank something in the last 12 hours (exception water).
- 3. Reschedule if participant had any caffeine, tobacco, or tobacco products in the last 4 hours
- 4. Reschedule if participant has participated in structured exercise the day of or day before assessment.
- 5. Reschedule if the participant is ill.
- 6. Reschedule if participant states they are unable to participate today.

Results

- □ Participant is cleared for their assessment today.
- □ Participant will need to be rescheduled because

Notes:

Name:		Date:		
Date of Birth Gender	M F	Height	Weight	
Are you Hispanic or Latino?				
Which one or more of the following w	ould you	say is your race?		
White		Black or African	American	

Asian	Native Hawaiian or other Pacific		
Islander			
American Indian or Alaska Native	Other (please specify		
)			

HEALTH HISTORY

Have you been told that you have any of the following? Check all that apply.

Diabetes	Heart disease	Heart attack
Cancer		
GI disorders	Kidney disease	Liver disease
Ulcers		
Thyroid disease	High blood pressure	High/borderline blood
sugar		
Lung disease	Stroke	Anemia
Other:		

Have you been told that anyone in your family have any of the following? Check all that apply.

M for mother, F for father, GM for grandmother, GF for grandfather, A for aunt, U for uncle, S for sibling, C for cousin.

Diabetes	Heart disease	Heart attack
Cancer		
GI disorders	Kidney disease	Liver disease
Ulcers		
Thyroid disease	High blood pressure	High/borderline blood
sugar		
Lung disease	Stroke	Anemia
Other:		
Do you have allergies?	Yes*	No
*If yes please list any n	medication environmental or	food allergies you have:
in yes, piease list any li	inconcation, environmental, or	iood anergies you nave.

Have you ever had any surgeries? _____ Yes* _____ No

*If so, what surgeries have you had?

MEDICATION/SUPPLEMENT HISTORY					
What over-the-counter medication do you take at least one time a month?					
Name of medication	Reason for taking	Dose	Frequency	Duration of	
intake					

MEDICATION/SUPPLEMENT HISTORY

What **over-the-counter vitamin or mineral supplements** do you take at least one time a month?

Name of supplement	Reason for taking	Dose	Frequency	Duration of
intake				

What **prescription medications** do you take at least one time a month? Please include birth control.

Name of medication Reason for taking Dose Frequency Duration of intake

	<u></u>		
T. 1 1 1 1	1 1 1 1 1 1	1 1 1 1 10	
To your knowledge, hav	'e you had your cholesterol	levels checked?	
Yes	No	Don't knov	V
Would you say that in g	GENERAL HIST eneral, your health is	ORY	
Excellent	Very good	Good Fair	
Poor			
D (111		×7	
Do you use/did you use	tobacco products?	Yes	
No			
Current	smoker: packs/day	Nur	nber of years
Previou	s smoker: quit date		
N	s smoker. quit dute		
Never			
Do you use other tobacc	to products? Yes	s, please specify below	
No			
Pine	Cigar	Snuff	
Chew			

Do you drink? No Yes	Number of drinks/week		
My current diet is	Satisfactory		
Unsatisfactory			
My current exercise/activity level is	Satisfactory		
Unsatisfactory			
My current weight is	Satisfactory		
Unsatisfactory			
Caffeine Intake None	Coffee/tea/soda	_ Cups/day	

Do you have any kind of health care coverage, including health insurance, prepaid plans such as HMOs, or government plans such as Medicare?

____ Yes ____ No ____ I don't know

About how long has it been since you last visited a doctor for a routine check-up? A routine checkup is a general physical exam, not an exam for a specific injury, illness, or condition.

- _____ Within past year (anytime less than 12 months ago)
- _____ 5 or more years ago
- _____ Within past 2 years (1 year but less than 2 years ago)
- _____ Don't know/not sure
- _____ Within past 5 years (2 years but less than 5 years ago)
- _____ Never

About Genetic Research

What is genetics?

Genetics is the study of inheritance. Genes are the instruction manuals for making building blocks called proteins which, combine in many ways to create all the parts of our bodies. Genes also create unique body features called "traits". Some traits are visible, like eye color. Other traits are not like your body's ability to fight certain diseases.

What is genetic testing?

Genetic testing identifies changes in <u>chromosomes</u> or genes. Most of the time, testing is used to find changes that are associated with inherited disorders. The results of a genetic test can confirm or rule out a suspected genetic condition or help determine a person's chance of developing or passing on a <u>genetic disorder</u>.

Another type of testing called "genome scanning" looks at DNA variations on a more minute level known as single nucleotide polymorphism (SNP, pronounced "snip"). Most SNPs are insignificant. However, some have effects on disease risk and vulnerability to disease-causing agents. In many cases, SNPs are not related to specific risks but to a general increase in risk which may be important in research.

Is having your DNA tested safe?

The physical risks associated with most genetic tests are very small, particularly for those tests that require only a blood sample or cheek swab.

Many people are also concerned about issues of privacy. In the <u>United States</u>, federal law requires that this kind of medical information be kept confidential.

Researchers take great care with DNA samples obtained from research volunteers. Researchers must obtain your permission to collect, analyze, and store your samples. Researchers must also get your permission to use your DNA sample in future studies.

Your stored DNA sample is not identifiable by donor. Confidentiality is of utmost importance. You have the right to request that your sample be discarded at anytime through the institution which collected the sample.

Genetic testing can only tell you if you have inherited the risk of developing a certain disease or condition. Having a genetic risk does not mean you will develop a particular condition, but rather that you have a higher change of developing it than if you did not have the risk. But genes are not the only thing. What you eat, whether and how much you exercise and where you live and work are all factors that can profoundly affect your overall health.

Energy Metabolism Lab Appetite Rating Scale

Subject #	Subject Initials	Visit #	Condition ID	Visit Date

Clock Time: _____ (0)

1. How hungry are you right now?





2. How satisfied (satiated) are you right now?



Not at all

Extremely



Energy Metabolism Lab Palatability Scale – Drink

Subject #	Subject Initials	Visit #	Condition ID	Visit Date

Clock Time: _____ (1 min)

1. How pleasant do you find this drink to be?



Extremely

Extremely

2. How sweet do you find this drink to be?



Not at all

3. How sour do you find this drink to be?



Extremely



Body Composition Measurements BodPod

Participant Involvement:

- You will be asked to wear a bathing suit or anything tight and fitting such as spandex and a sports bra.
- All jewelry and watches must be removed before the test can be performed so you can leave them at home or take them off there.
- The test is done in a comfortable, private room.
- The test takes 5-10 minutes total.



Maximal Oxygen Uptake Measurements VO₂max

Participant Involvement:

- You will be asked to wear comfortable clothes to work out in, preferably shorts, a t-shirt and sneakers.
- You will be asked to walk on a treadmill at a constant speed and progressively increasing grade until you feel you are maximally fatigued and can't proceed any further.
- This test will not require you to run at anytime.
- Each test takes about 30 minutes total, including set-up and equipment orientation.





BodPod Data Sheet – Brozek Equation

Reading 1:	
------------	--

Reading 2:_____

Reading 3:

Mean:

Percent Body Fat:

Body Weight:

Fat Weight:

Lean Weight:

Lung Volume (predicted):

Comments:



Yamax Digi-Walker SW-701

- Step counter Accurately counts your steps while walking, jogging, or hiking
- Displays number of steps taken, distance walked and calories burned
- Cover helps protect from unexpected resets and keeps counts confidentially
- Model is about 2" X 1 ¹/₂" X ³/₄" and weighs less than ¹/₄ ounce
- Your personal stride will be set on the pedometer along with your weight 20 feet / # of steps walked in 20 feet = stride
- Please wear it on your hip and have it clipped onto your pants. Please do not wear it anywhere else such as your shoes
- We will provide a log so you can write down how many steps you take each day
- You will need to reset the step count on your pedometer every day before putting it on to get an accurate number for that day (Be sure to write down the number of steps you took before you press yellow 'Reset' button)

6	No exertion at all		
7	Extremely light		
8			
9	Very light		
10			
11	Light		
12			
13	Somewhat hard		
14			
15	Hard (heavy)		
16			
17	Very hard		
18			
19	Extremely hard		
20	Maximal exertion		

Borg-RPE-Scale³ © Gunnar Borg 1970, 1985, 1998

BORG

Blood Pressure Record Sheet - VO₂max Test

 Resting BP 1 _____
 Resting BP 2 _____

Stage	Time (min)	BP (mmHg)
1	2:00	
3	6:00	
5	10:00	
7	14:00	
8	16:00	
9	18:00	
10	20:00	
Recovery	2:00	

Post-Exercise BP

Name:

Student ID#:

Age:

Ht. (in):

Wt. (kg):

Resting HR (bpm):

Max HR: 220-age=

		Speed ¹⁸²	Grade (%)	HR (bpm)	RPE
	Warm-Up				
	1:00	3.0	0		
Stage	Exercise				
]	0:00	3.0	2.5		
	1:00	3.0			
	2:00	3.0	5		
	3:00	3.0			
3	4:00	3.0	7.5		
	5:00	3.0			
4	6:00	3.0	10.0		
	7:00	3.0			
4	8:00	3.0	12.5		
	9:00	3.0			
(10:00	3.0	15.0		
	11:00	3.0			
-	12:00	3.0	17.5		
	13:00	3.0			
8	3 14:00	3.0	20.0		
	15:00	3.0			
	16:00	3.0	22.5		
	17:00	3.0			
	Recovery				
	1:00		0		
	2:00		0		
	3:00		0		

Appendix G: BFF Nutrition and Physical Activity Education Outline

BFF Intervention: Physical Activity Lesson Plan Outline Session 1:

- A. Welcome Staff introduction and background. BFF Study overview and requirements
- B. Introduction to the ACSM guidelines.
- C. Review and Goal Setting what do we want to accomplish in our time?
 - 1. Each participant set one goal.
 - 2. Brainstorm on how to accomplish this goal.
 - 3. Review of today's topics.
 - 4. Have participants keep a food and exercise log.

Handouts Needed:

"Following the DASH Eating Plan" (from the DASH diet website) Copies of food and exercise logs

Session 2:

- A. Welcome Back! Weigh In
 - 1. Review of last week Calculate step goals
 - 2. Discussion of goal achievement.
 - 3. Overview of Session 2.
- B. ACSM Guidelines-Meetings those guidelines
- C. Types of exercise.

Session 3:

- A. Welcome Weigh In/Charts
 - 1. Review of last week.
 - 2. Discussion of goal achievement.
 - 3. Overview of session 3.
- B. Common myths of exercise.
- C. Pros/Cons
- D. Barriers to exercise/overcoming barriers
- E. Wrap-up

Session 4:

- A. Welcome-Weigh In/Charts
 - 1. Review of last week.
 - 2. Discussion of goal achievement.
 - 3. Overview of session 4.
- B. Fun Fact Gaining muscle and burning fat
- C. FITT Principle
- D. Exercise Sheets-Abdominals, Arms/Shoulders, Lower-body

Session 5:

- A. Welcome-Weight In/Charts
 - 1. Review of last week try any new exercises
- B. Build up those muscles Anaerobic Activity
- C. Machine Weights vs. Free Weights

Session 6:

- A. Welcome-Weigh In/Charts
 - 1. Review of last week.
 - 2. Discussion of goal achievement.
 - 3. Overview of session 5.
- B. Warm up /Cool Down
 - 1. Anaerobic activity
 - 2. Warm-up routines (samples)
- C. Stretching-Importance
- D. Stretching Sheet

Session 7:

- A. Welcome-Weight In/Charts
 - 1. Review of last week.
 - 2. Discussion of goal achievement.
 - 3. Overview of session 6.
- B. Putting it all together
 - 1. Programs
 - 2. Beginner vs Intermediate/Advanced
 - 3. Examples

Session 8:

- A. Welcome-Weigh In/Charts
 - 1. Review of last week.
 - 2. Discussion of goal achievement.
 - 3. Overview of session 7.
- B. Exercise Program
 - 1. Maintaining Weight
 - 2. Activity vs Food
- C. Review
 - 1. Review of previous sessions (any questions)
 - 2. Were goals achieved?
 - 3. Maintaining the weight loss
 - 4. Congratulations!

BFF Intervention: Dietary Education Lesson Plan Outline

Session 1: Introduction to BFF Study and DASH Diet

A. Welcome – Staff Introduction and Background

a. BFF study review and background

- B. Introduction to dietary guidelines for CHD DASH Diet
- C. Review Goal Setting what do we want to accomplish in our time?
 - 1. Each participant set a goal
 - 2. Review today's topics
 - 3. Have participants keep food and exercise log

Handouts need:

Following the DASH Eating Plan" (from the DASH diet website) "Making the DASH Difference" "Healthy Eating the DASH Way" "Get Smart About Salt" Copies of food and exercise logs

Session 2: Cutting Calories, Healthy Food Substitutions, Eating in Dining Halls

- A. Welcome back! Collect pedometer sheets
 - 1. Review of last week DASH diet
 - 2. Overview of today's session
- B. Review Food logs
 - a. Compare their log to DASH sample day
- C. Review Goal setting
 - a. Each participant set a goal
 - b. Review today's topics

Handouts needed:

"How to Lower Calories on the DASH Eating Plan" "Healthy Food Substitutions From Dining Hall" Sample Day from DASH Website

Session 3: Know Your Fats

- A. Welcome back!
 - 1. Review of last week Nutrient Density, Cutting Calories
 - 2. Overview of today's session
- B. Review Goal Setting
 - a. Each participant set a goal for next week
 - b. Have goal be related to topics discussed in today's session
 - c. Encourage participants to engage in 30 minutes physical activity/day
- C. Review Take Home Message
 - a. Fun Food Fact

Handouts Needed:

"Heart Friendly Fats" (from SNAP office)

"The Truth About Trans Fats" (from SNAP office) "Cutting Down Your Cholesterol" (from SNAP office)

Session 4: Grains

- A. Welcome back!
 - 1. Review of last week's topic
 - 2. Overview of today's session
- B. Review Goal Setting
 - a. Each participant set a goal for next week
 - b. Replace one common food they eat that is not whole grain with a whole grain option
- C. Review Take Home Message
 - a. Fun Food Fact

Handouts Needed:

"Go for Whole Grains" – Nutrition to GO

Session 5: Fruits and Vegetables

- A. Welcome back!
 - 1. Review of last week's topic
 - 2. Overview of today's session
- B. Review Goal Setting
 - a. Each participant set a goal for next week
 - b. Encourage participants to engaged in 30 min physicial activity/day
 - c. Fun Food Fact
- C. Review Take Home Message

Handouts Needed:

"The Power of Produce" (SNAP office) "Fantastic Fruits" (SNAP office)

"Very Veggies" (SNAP office)

Session 6: Protein

A. Welcome back!

- 1. Review of last week's session
- 2. Overview of this week's session
- 3. DASH diet recommendations for protein
- B. Review Goal Setting
 - a. Each participant set a goal for next week
 - b. Encourage physical activity
 - c. Fun Food Fact
- C. Review Take Home Message

Handouts Needed:

"An Egg-Cellent Choice" (from SNAP office) "Fast Seasonal Facts" (from SNAP office)

Session 7: Dairy

A. Welcome back!

- 1. Review of last week's session
- 2. Overview of this week's session
- 3. DASH diet recommendations/servings
- B. Review of All Nutrition Sessions up to this point
 - a. Discuss key points
 - b. Review principles of DASH Diet
- C. Review Goal Setting
 - a. Each participant set a goal for next week
 - b. Encourage physical activity
 - c. Fun Food Fact
- D. Review Take Home Message
- E. Feedback from group on sessions

Handouts needed:

"Got Calcium?" (from SNAP office) "Lactose Intolerance" (from SNAP office) "BFF Nutrition Session Guide"

Session 8: Maintenance of Lifestyle Changes and Weight Loss

- A. Welcome back!
 - 1. This is the last diet session; thank everyone for sticking through the diet sessions
 - 2. Review of last week's session
- B. Overview of this week's session
 - a. Tips to Maintain the Diet and Exercise Changes

Appendix H: Hatch Consent Form for Research

The University of Rhode Island Department of Nutrition and Food Sciences 302 Ranger Hall Nutritional Assessment and Chronic Disease Risk Factor Identification in Young Adults

CONSENT FORM FOR RESEARCH

Thank you for considering being part of our study, *Nutritional Assessment and Chronic Disease Risk Factor Identification in Young Adults*. By participating in this research, you will help us determine what risk factors of chronic disease are present in young adults. To participate in this research, you must be a first-year University of Rhode Island student between the ages of 18 and 24 years, and you may not be pregnant or lactating. You are not eligible for this study if you have diabetes (Type I or Type II), cancer, coronary heart disease, liver disease, a bleeding disorder, or if you are on lipid-lowering medications.

Description of the project:

You have been asked to take part in a research study aimed at determining what chronic disease risk factors are present in a college population 18 to 24 years of age. The presence of chronic disease risk factors will be determined by taking measurements and dietary information during three visits and two phone calls over a six to eight week period.

What will be done:

If you decide to take part in this study, this is what will happen over the course of three visits and two phone calls:

First assessment visit (approximately 20-30 minutes):

• We will ask you questions about what you ate the day before. You do not need to write anything down prior to coming.

Day prior to second screening visit (overnight):

• For the twelve hours prior to the second assessment visit in Ranger 301, you will be asked to refrain from eating or drinking anything unless it's water. We encourage you to drink as much water as you would like. For example, if your screening visit is scheduled for 8 am on a Tuesday, you will not eat or drink anything except for water after 8 pm on Monday evening.

The second assessment visit (approximately 20-30 minutes):

• Your height, weight, and waist circumference will be measured.

- After you have been fasting for 12-hours, 60-ml blood draw (approximately 4 tablespoons) will be taken while you are in a seated position. We will use your blood to measure different things in your blood such as lipids (example: total cholesterol), hormones (example: insulin), and protein (example: apolipoproteins).
- You will complete questionnaires on your personal and family medical history, your daily activity and exercise routine, and how hungry you are. You may skip any question(s) in the surveys.

Day prior to third assessment visit (overnight):

• As with the day prior to the second assessment visit, you will be asked to refrain from eating or drinking anything unless it's water for the twelve hours prior to the second screening visit in Ranger 301. Again, we encourage you to drink as much water as you would like.

The third assessment visit (approximately 15 minutes):

- Blood pressure will be measured two times on the right arm while you are in a seated position.
- A 12 hour fasting 10-ml blood draw (approximately 1 tablespoon) will be taken while you are in a seated position.

After the third assessment visit, you will receive two phone calls from study staff. During these two phone calls you will answer questions about the foods you eat. Each of these phone calls will take approximately 20-30 minutes.

From the time of the first assessment visit until the final phone call will be approximately six to eight weeks and a total of 2 hours of time on your part.

Risks or discomfort:

There are no known risks for the following procedures: completing the questionnaires and having height, weight, and waist circumference measured. Even though experienced personnel will perform the blood draw using a sterile technique there are a few risks associated with the procedure: discomfort from the needle, bruising, formation of a blood clot, and infection. A trained professional will perform the blood draw, and we will take all steps possible to minimize the risks to you.

Benefits of this study:

This study will help in understanding what chronic disease risk factors are most prevalent in a young adult population. The direct benefits to you include learning about your health status. You will receive the results with your blood pressure and the fats in your blood (total cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol, and triglycerides). You will also be provided with your weight and body mass index status. Although these values are not diagnostic, the results can be shared with a health practitioner and repeated if deemed necessary. If your blood pressure or total cholesterol, low density lipoprotein cholesterol, high density lipoprotein cholesterol or triglycerides do not fall within recommended values, you will be encouraged to follow-up with your primary care physician or at Student Health Services.

Confidentiality:

Your participation in this study is confidential. When you enroll in the study, you will be assigned a code. This code will be used for data management only and your results will not be associated directly with your name. The researchers and the University of Rhode Island will protect your privacy, unless they are required by law to report information to city, state or federal authorities, or to give information to a court of law. All records, including data and consent forms, will be stored in a locked office that is not accessible except for personnel involved in the study.

In case there is any injury to the subject:

If you have any injury or discomfort as a result of the experiment, you should notify Dr. Ingrid Lofgren at (401) 874-5706. Additionally, if this study causes you any injury, you should write or call the office of the Vice Provost for Graduate Studies, Research and Outreach, 70 Lower College Road, University of Rhode Island, Kingston, Rhode Island, telephone: (401) 874-4328.

Decision to quit at any time:

The decision to take part in this study is up to you. You do not have to participate. If you decide to take part in the study, you may quit at any time. Whatever you decide will in no way penalize you. If you wish to quit, you simply inform Dr. Ingrid Lofgren at (401) 874-5706 of your decision.

Incentives

You will receive full compensation (\$30.00) if you complete the study.

Rights and Complaints:

This study is part of research being conducted by the University of Rhode Island. If you have any questions or if you are not satisfied with the way this study is performed, you may discuss your complaints with Dr. Ingrid Lofgren (401) 874-5706, anonymously, if you choose. In addition, you may contact the office of the Vice President for Graduate Studies, Research and Outreach, 70 Lower College Road, University of Rhode Island, Kingston, Rhode Island, telephone: (401) 874-4328.

You have read the Consent Form. Your questions have been answered. Your signature on this form means that you understand the information and you agree to

participate in this study.

I,	
residing at	(zip)
telephone	age (date of birth)
agree to participate in this research j	project.
Signature of Participant	Signature of Researcher
Typed/printed Name	Typed/printed name
Date	Date

Please sign and date two consent forms, keeping one for yourself.
Appendix I: Hatch Assessment Materials

CONTACT INFORMATION:

NAME: Last, First	Gender (circle)	Gender (circle) Male Female		
BIRTHDATE (mo/day/yr):	How did you hea	r of this study?		
MAJOR:	Are you willing to evaluate the prog	Are you willing to be contacted after the study is over to evaluate the program? (circle) Yes No		
CURRENT ADDRESS:	<u> </u>			
HOME ADDRESS OR CONTACT INFO	DRMATION IN CASE YOU MOVE:			
DAY PHONE # (with area code):	CELL PHONE # (with area code):	EMAIL ADDRESS:		
BEST WAY TO CONTACT (check any a	applicable box): email day phone	cell phone Other, specify:		
METHOD OF RECRUITMENT (explain	how subject was recruited):			

NOTE: The following is for compensation purposes.

NAME: Last, First			
		ID #	
			Phone recall 1
Assessment #1	Assessment #2	In-person recall	Phone recall 2
Compensation given upon completion of final recall			
PPT Signature		Researcher Initials	Date
Notes:			

Data Collection Form

Subject ID #	A	ssessment v	isit #1	Assessment Visit#2			
DATE							
TIME							
RESEARCHER who completed assessment							
	Measure 1	Measure 2	Average of 1 & 2	Measure 1	Measure 2	Average of 1 & 2	
WEIGHT (kgs)			*	N/A	N/A		
HEIGHT (cm)			*	N/A	N/A		
BP (systolic/diastolic)			*			*	
WAIST CIRCUMFERENCE (cm)			*	N/A	N/A		
BMI				N/A	N/A		
In-person recall? (circle)		yes/no			yes/no		
CONCERNS FOR ERROR (for weight, height, bp, waist circum., other)							
COMMENTS							
* Calculate average of the two measurements							
Blood Pressure Readings:							

If applicable: Date Subject becomes inactive ______. Date Subject changes from inactive to active ______.

If applicable: Date Subject becomes a drop-out ______.

Date Subject becomes inactive _____. Date Subject changes from inactive to active _____.

INTERNATIONAL PHYSICAL ACTIVITY QUESTIONNAIRE (August 2002)

SHORT LAST 7 DAYS SELF-ADMINISTERED FORMAT

FOR USE WITH YOUNG AND MIDDLE-AGED ADULTS (15-69 years)

The International Physical Activity Questionnaires (IPAQ) comprises a set of 4 questionnaires. Long (5 activity domains asked independently) and short (4 generic items) versions for use by either telephone or self-administered methods are available. The purpose of the questionnaires is to provide common instruments that can be used to obtain internationally comparable data on health–related physical activity.

Background on IPAQ

The development of an international measure for physical activity commenced in Geneva in 1998 and was followed by extensive reliability and validity testing undertaken across 12 countries (14 sites) during 2000. The final results suggest that these measures have acceptable measurement properties for use in many settings and in different languages, and are suitable for national population-based prevalence studies of participation in physical activity.

Using IPAQ

Use of the IPAQ instruments for monitoring and research purposes is encouraged. It is recommended that no changes be made to the order or wording of the questions as this will affect the psychometric properties of the instruments.

Translation from English and Cultural Adaptation

Translation from English is supported to facilitate worldwide use of IPAQ. Information on the availability of IPAQ in different languages can be obtained at <u>www.ipaq.ki.se</u>. If a new translation is undertaken we highly recommend using the prescribed back translation methods available on the IPAQ website. If possible please consider making your translated version of IPAQ available to others by contributing it to the IPAQ website. Further details on translation and cultural adaptation can be downloaded from the website.

Further Developments of IPAQ

International collaboration on IPAQ is on-going and an *International Physical* Activity Prevalence Study is in progress. For further information see the IPAQ website.

More Information

More detailed information on the IPAQ process and the research methods used in the development of IPAQ instruments is available at www.ipag.ki.se and Booth, M.L. (2000). Assessment of Physical Activity: An International Perspective. Research Quarterly for Exercise and Sport, 71 (2): s114-20. Other scientific publications and presentations on the use of IPAQ are summarized on the website.

INTERNATIONAL PHYSICAL ACTIVITY OUESTIONNAIRE

We are interested in finding out about the kinds of physical activities that people do as part of their everyday lives. The questions will ask you about the time you spent being physically active in the **last 7 days**. Please answer each question even if you do not consider yourself to be an active person. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Think about all the **vigorous** activities that you did in the **last 7 days**. **Vigorous** physical activities refer to activities that take hard physical effort and make you breathe much harder than normal. Think *only* about those physical activities that you did for at least 10 minutes at a time.

1. During the **last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, digging, aerobics, or fast bicycling?

days per week

No vigorous physical activities

Skip to question 3

2. How much time did you usually spend doing **vigorous** physical activities on one of those days?

> hours per day minutes per day

Don't know/Not sure

Think about all the **moderate** activities that you did in the **last 7 days**. **Moderate** activities refer to activities that take moderate physical effort and make you breathe somewhat harder than normal. Think only about those physical activities that you did for at least 10 minutes at a time.

3. During the **last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads, bicycling at a regular pace, or doubles tennis? Do not include walking.

	days per week
How activ	No moderate physical activities
	hours per day minutes per day
	Don't know/Not sure

Think about the time you spent **walking** in the **last 7 days**. This includes at work and at home, walking to travel from place to place, and any other walking that you might do solely for recreation, sport, exercise, or leisure.

5. During the **last 7 days**, on how many days did you **walk** for at least 10 minutes at a time?

 _days per we	ek	
No walking	\rightarrow	Skip to question 7

6. How much time did you usually spend **walking** on one of those days?

_____hours per day

4.

_____ minutes per day

Don't	know/Not	sure
DONI		Suic

The last question is about the time you spent **sitting** on weekdays during the **last 7 days**. Include time spent at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading, or sitting or lying down to watch television.

7. During the **last 7 days**, how much time did you spend **sitting** on a **week day**?

hours per day			
	_minutes per day		
	Don't know/Not sure		

This is the end of the questionnaire, thank you for participating.

articipant Feedback Name:
The following is a summary of your results from the Nutrition Assessment Study. We
re sorry for the long delay in getting these results to you. We had some equipment
roblems that were just resolved. We would like to restate that these values are used
or screening purposes only and not for diagnosis. We encourage you to follow up
ith your primary care physician if you have concerns about your values. If you have
uestions about the data or the study, please call us at 401-874-2785.

Height: cm		Weight:	kg
Waist Circumferen	ice: cm	BMI:	kg/m ²

BMI Classifications:

Underweight: <18.5 kg/m²

Overweight: 25-29.9 kg/m²

Normal: 18.6-24.9 kg/m² Obese: >30 kg/m²

Blood Pressure: mmHg

Blood Pressure Classifications: Optimal = <120/<80 mmHg Pre-hypertensive = 120-139/80-89 mmHg Hypertensive = $\geq 140/\geq 90$ mmHg

Average Total Calories Consumed:

% Carbohydrate: % % Protein: % % Fat: %

Blood Lipid Values

Total Cholesterol:	mg/dL	LDL-Cholester	ol: mg/dL
Triglycerides:	mg/dL	HDL-Cholester	ol: mg/dL
Blood Lipid	Recommenda	ions:	
Total Cholesterol: <	200mg/dL	LDL-Cholesterol: <130	mg/dL
Triglycerides: <150	mg/dL	HDL-Cholesterol: Fem	ales >50 mg/dL

Males >40 mg/dL