PREDICTORS OF COMPENSATORY CHANGES IN ENERGY BALANCE FOLLOWING EXERCISE AMONG OVERWEIGHT AND OBESE, SEDENTARY WOMEN

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Although exercise produces an acute energy deficit, there is substantial variability in behavioral and biological responses to exercise that influence propensity for weight change. Indeed, some individuals compensate for exercise by increasing energy intake or decreasing physical activity energy expenditure, leading to a positive energy balance. This maladaptive pattern of behavioral compensation ultimately undermines the efficacy of exercise as a weight loss strategy. The present study aimed to identify individual differences, such as disinhibited eating, that elevate risk for a positive energy balance following exercise. Participants were 48 overweight or obese (BMI $\geq$ 25), sedentary women. On average, women were 21.33 (*SD* = 2.09) years old and 62.5% were white. Women completed self-report measures of eating pathology and behavior and participated in two experimental conditions, exercise and nonexercise, one week apart in a counterbalanced order. Energy intake and physical activity energy expenditure were measured for 24-hours following each condition to compute an estimate of energy balance. Women were defined as compensators if they increased energy intake, decreased energy expenditure, or had a higher energy balance on the exercise day relative to the nonexercise day. Of the sample, 63% compensated for exercise, with 57% compensating by solely increasing energy intake, 27% compensating by solely reducing physical activity energy expenditure, and 17% compensating by both increasing energy intake and reducing physical activity energy expenditure. Separate linear mixed effects models were used to identify predictors of behavioral compensation following exercise. Contrary to expectations, disinhibited eating did not predict behavioral

compensation. However, objective binge eating was shown to predict compensatory increases in energy balance following exercise above and beyond relevant covariate effects. These findings provide preliminary evidence that women who report objective binge eating may be at greatest risk of compensating for exercise and further substantiate the need for a better understanding of psychosocial predictors and common mechanisms through which behavioral compensation is promoted to better inform intervention efforts.

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1.0 INTRODUCTION

Obesity is an increasingly prevalent and preventable cause of death and is associated with significant physical (Bray, 2004), psychosocial (Carr & Friedman, 2005; Puhl & Brownell, 2001), and financial consequences (Finkelstein, Fiebelkorn, & Wang, 2004; Finkelstein et al., 2008; Withrow & Alter, 2010). The prevalence of obesity has been attributed to many environmental factors, such as inflated portion sizes, sedentary lifestyles, and highly caloric food options that are both inexpensive and readily available (Fairburn & Brownell, 2002; French, et al, 2001). However, individual differences in weight status indicate that not everyone who is exposed to an obesogenic environment is at risk of developing obesity. Indeed, individual variability in psychological and biological factors, such as metabolic processes, trait and state variables, and reward responsivity, have been shown to promote differential responses to environmental triggers, leading some individuals to be susceptible to weight gain and others resistant (Blundell et al., 2005). Accordingly, identifying factors that contribute to individual differences in weight gain and weight regulation has important implications for obesity prevention and treatment efforts.

Obesity ultimately is a disorder of positive energy balance (EB; Epstein, Leddy, Temple, & Faith, 2007). A positive EB occurs when the amount of energy consumed is greater than the amount of energy expended, thereby creating a surplus of energy that is eventually converted to and stored as body fat (Blundell & Cooling, 2000). Over time, a sustained positive EB results in substantial fat deposition leading to excess weight gain (Epstein, Leddy, Temple, & Faith, 2007).

EB itself is determined by variability in energy intake (EI) and energy expenditure (EE), which are driven by a variety of psychological and biological mechanisms. EI is dictated by eating behaviors, which comprise a complex set of phenomena that influence food choice as well as the quantity of food consumed and the frequency of eating episodes (Blundell & Cooling, 2000) whereas EE is an aggregate of metabolic processes, diet induced thermogenesis, and physical activity (DeLany, 2012). Research has identified individual differences in these mechanisms that increase liability for a positive EB (Birch, Fisher, & Davison, 2003; Carnell & Wardle, 2008; Epstein, Leddy, Temple, & Faith, 2007; Hartmann, Czaja, Rief, & Hilbert, 2010; Martins, Morgan, & Truby, 2008; Martins, Robertson, & Morgan, 2010; Waxman, 2009). Thus, developing strategies to modify such individual differences is necessary to reduce risk for excess weight gain and to promote successful weight loss.

1.1 EXERCISE AND ENERGY BALANCE

In an effort to achieve weight loss, comprehensive lifestyle interventions target both sides of the EB equation by focusing on reducing EI and increasing EE (Greeno & Wing, 1994). Exercise is recognized as a modifiable component of EB that can potentially lead to an acute energy deficit and is often prescribed, in combination with dietary restriction, as a means of weight loss and control (Jakicic et al., 2001). However, despite the health benefits that accompany regular exercise (Wei, et al., 1999), exercise alone has been shown to be relatively ineffective as a weight loss strategy (Catenacci & Wyatt, 2007) and typically fails to produce expected amounts of weight loss (Lee, et al., 2005). This apparent lack of efficacy may be partly attributable to

maladaptive biological or behavioral responses following exercise, such as dietary compensation, that impede the ability of exercise to produce a sustained energy deficit necessary for weight loss (Blundell & King, 2000).

The majority of findings, however, have demonstrated that exercise has a beneficial impact on appetite regulation and eating behavior and should promote weight loss (King, et al., 2009; Martins, Robertson, & Morgan, 2010; Martins, Morgan, Bloom, & Robertson, 2007). Physiological evidence has shown that exercise elevates plasma levels of satiety hormones (Martins, Morgan, Bloom, & Robertson, 2007) and improves sensitivity to satiety signaling (King, et al., 2009; Martins, Robertson, & Morgan, 2010). Moreover, acute bouts of exercise, regardless of intensity, often are associated with reduced EI (King & Blundell, 1995; King, Lluch, Stubbs & Blundell, 1997; Thompson, Wolfe, Eikelboom, 1988; Westerterp-Plantenga, Verwegen, Ijedema, Wijckmans, & Saris, 1997), and the adoption of routine exercise motivates healthier food choices and positively impacts overall behavioral lifestyle (Muller-Riemenschneider, Reinhold, Nocon, & Willich, 2008). Thus, exercise appears to provoke both biological and behavioral adaptations that are advantageous for weight loss. However, these findings do not adequately explain why exercise interventions are unsuccessful in producing expected weight loss among overweight and obese individuals (Catenacci & Wyatt, 2007; Lee, et al., 2005).

Research has identified marked differences among individuals, such as gender and body weight, that affect exercise-induced weight change. With regard to gender, women are more likely than men to demonstrate elevated hedonic responses to food (King & Blundell, 1995; King, Snell, Smith, & Blundell, 1996) and report a stronger desire to consume palatable foods high in fat and sugar (Finlayson, Bryant, Blundell, & King, 2009) following exercise. This

gender difference is believed to result from cognitive rather than physiological factors, such as the belief that exercise increases hunger or the desire to use food as a reward for exercising (King, 1999). Evidence suggests that women are more likely than men to engage in compensatory behaviors following exercise that serve to either decrease or abolish the effects of exercise on overall EB. Specifically, women tend to increase EI in response to exercise whereas exercise has no effect on subsequent EI among men (Imbeault, Saint-Pierre, Alméras, & Tremblay, 1997; Pomerleau, Imbeault, Parker, & Doucet, 2004; Thompson, Wolfe, & Eikelboom, 1988). Taken together, this evidence suggests that exercise may be less effective as a weight loss strategy for women than for men.

Importantly, differences in EI following exercise vary as a function of weight status among women. That is, normal weight women often increase EI following exercise (Durrant, Royston, & Wloch, 1982; Kissileff, Pi-Sunyer, Segal, Meltzer, & Foelsch, 1990) whereas overweight and obese women tend to either suppress or maintain EI following exercise (Durrant, Royston, & Wloch, 1982; Tsofliou, Pitsiladis, Malkova, Wallace, & Lean, 2003; Unick, et al., 2010). Thus, exercise should promote a negative EB among overweight and obese women by promoting a reduction in EI. However, this evidence contradicts previous findings demonstrating that overweight and obese women often are unsuccessful at achieving predicted weight loss following exercise interventions (Catenacci & Wyatt, 2007; Lee, et al., 2005). Although poor treatment adherence partly explains these incompatible findings, it does not fully account for this effect, suggesting that additional factors must contribute to this relationship (Hopkins, King, Blundell, 2010). Indeed, there are individual differences among overweight and obese women that alter behavioral and biological responses to exercise and either promote or prevent weight

loss (Finlayson, Bryant, Blundell, & King, 2009; King, et al., 2009; Martins, Robertson, & Morgan, 2010).

Hill and colleagues (1995) proposed a model whereby EB is modulated by body weight and individual differences in eating behavior. This model purports that individuals with particular eating behaviors (i.e., unrestrained, restrained, and disinhibited eating) are either more or less responsive to physiological cues of hunger and fullness and subsequently present different patterns of energy compensation in response to exercise. Notably, this model suggests that overweight individuals characterized by high levels of disinhibited eating are particularly likely to increase EI in response to exercise. In fact, overweight women with high levels of disinhibited eating have been shown to increase EI in response to both acute (Visona & George, 2002) and recurrent bouts of exercise (Keim, Canty, Barbieri, & Wu, 1996) whereas normal weight women high on disinhibited eating report an increase in preference for low-fat foods (Bryant, Finlayson, King, & Blundell, 2006) and a reduction in motivation to eat with no significant differences in food intake following exercise (Bryant, King, & Blundell, 2005). Thus, overweight women who report high levels of disinhibited eating may be at particular risk of increasing EI following exercise.

1.2 DISINHIBITED EATING AND ENERGY BALANCE

Disinhibited eating itself is an individual difference factor characterized by a tendency towards opportunistic overeating that increases risk for a positive EB. Disinhibited eating is associated with susceptibility to weight gain over time (Hays et al., 2002), greater difficulties losing excess

weight (Butryn, Thomas, & Lowe, 2009), and weight regain following weight loss interventions (Bryant, King, & Blundell, 2008). Disinhibited eating also has been linked to other eating characteristics, such as dysregulated hunger and satiety mechanisms (Martins, Robertson, & Morgan, 2010; Burton-Freeman & Kiem, 2008), a heightened preference for palatable foods (Haynes, Lee, & Yeomans, 2003; Yeomans, Tovey, Tinley, & Haynes, 2004; Yeomans, Mobini, Bertenshaw, & Gould, 2009), a tendency to overeat in a variety of contexts (Bond, McDowell, & Wilkinson, 2001), greater levels of sedentary behavior (Bryant, 2006), higher risk for eating pathology (Brown, Bryant, Naslund, King, & Blundell, 2006; Crow, Kendall, Praus, & Thuras, 2001), and greater binge eating severity (d'Amore, et al., 2001; de Zwaan, et al., 2003).

The characteristic features of disinhibited eating are related to a heightened responsiveness to situational, habitual, and emotional cues that trigger eating behavior (Bond et al., 2001). Specifically, disinhibited eating is related to a failure to resist urges to eat despite satiation and an inability to maintain dietary restraint as well as a tendency to overeat in response to food palatability, negative affect, and social settings (Stunkard & Messick, 1985). The eating patterns characteristic of individuals who score high on measures of disinhibited eating lead to a sustained positive EB and are primary predictors of excess weight gain across the lifespan (Bond et al., 2001; Hays & Roberts, 2008). Moreover, disinhibited eating is predictive of increased EI following various challenges or interventions that threaten to disturb EB, including food preloads (Ouwens, Van Strien, & Van der Staak, 2003; van Strien, 2000; Westenhoefer, Broeckmann, Munch, & Pudel, 1994) and exercise interventions (Keim, Canty, Barbieri, & Wu, 1996; Bryant, King, & Blundell, 2005; Visona & George, 2002). As such, disinhibited eating may serve as an individual difference factor that increases risk for reestablishing a positive EB following exercise.

In general, the excess EI customary of disinhibited eating has been proposed to be partially driven by dysregulations in the homeostatic controls of EB. Evidence indicates that individuals high on disinhibited eating display physiological differences in hunger and satiety mechanisms. For example, Blundell and colleagues (2008) found disinhibited eating to be positively correlated with fasting levels of leptin and negatively correlated with ghrelin among a sample of adult women. Moreover, laboratory studies have shown that disinhibited eating is associated with blunted postprandial levels of hormones related to satiety regulation, including both cholecystokinin and peptide YY (Martins, Robertson, & Morgan, 2010; Burton-Freeman & Kiem, 2008). These differences contribute to a dampened satiety response (Blundell et al., 2005) and heightened sensations of hunger and appetite (Hays & Roberts, 2008), which act to foster increased EI. However, research indicates that excess EI is often driven by the rewarding properties of palatable foods rather than homeostatic controls over feeding (Appelhans, 2009). As such, sensory and motivational processes driving hedonic feeding may be better predictors of the increased EI associated with disinhibited eating.

Individuals high on disinhibited eating characteristically display a heightened sensitivity to palatable foods (Delparigi, et al., 2005) and report a strong preference for and intense desire to eat palatable foods (Lähteenmäki & Tuorila, 1995; Bryant, Finlayson, King, & Blundell, 2006). Disinhibited eating is positively related to the consumption of foods high in fats, salt, sugars, and carbohydrates as well as sweetened, caffeinated beverages and is negatively related to the intake of fruits, vegetables, and high-fiber bread (Borg, Fogelholm, Kukkonen-Harjulaas, 2004; Contento, Zybert, & Williams, 2005). Moreover, individuals high on disinhibited eating report little relation between hunger and eating (Barkeling, King, Näslund, & Blundell, 2007), and multiple laboratory demonstrations have shown that disinhibited eating is associated with a

hyperphagic response to palatable foods in the absence of hunger (Haynes, Lee, & Yeomans, 2003; van Strien, 2000; Westenhoefer, Broeckmann, Munch, & Pudel, 1994; Yeomans, Tovey, Tinley, & Haynes, 2004; Yeomans, Mobini, Bertenshaw, & Gould, 2009). Disinhibited eating thus appears to motivate eating through sensory reward processes independent of hunger sensations, thereby promoting increased consumption of energy-dense foods and poorer overall dietary composition.

Ample evidence has indicated that disinhibited eating distinguishes individuals who are motivated to eat by the hedonic qualities of food rather than physiological necessity and are likely to engage in an overeating response in a variety of contexts, such as the period following exercise. Indeed, past research has documented that overweight women high on disinhibited eating are at risk of increasing EI following exercise (Keim, Canty, Barbieri, & Wu, 1996; Bryant, King, & Blundell, 2005; Visona & George, 2002), which may ultimately serve to prevent weight loss (Hopkins, King, & Blundell, 2010). However, nothing is known about how disinhibited eating influences EB following exercise as past research has failed to measure compensatory changes in EE in addition to EI. Given that EI is only one half of the EB equation, the relationship between EE and disinhibited eating may be an important part of the link between disinhibited eating and weight gain. Because individuals high on disinhibited eating are particularly sensitive to reductions in their EB, they may respond to exercise by increasing EI and decreasing EE in order to maintain a positive EB.

2.0 SCOPE OF THE PRESENT STUDY

In summary, although exercise is often utilized as a strategy to encourage weight loss and improve health, some individuals respond unfavorably to exercise regimens and fail to achieve expected weight loss. Biological and behavioral responses to exercise show great interindividual variability, and these responses interact to determine propensity for weight change. It appears that some individuals can tolerate sustained periods of an exercise-induced energy deficit (Imbeault, Saint-Pierre, Alméras, & Tremblay, 1997; Pomerleau, Imbeault, Parker, & Doucet, 2004; Thompson, Wolfe, & Eikelboom, 1988; Tsofliou, Pitsiladis, Malkova, Wallace, & Lean, 2003; Unick, et al., 2010) whereas others compensate for this energy deficit by increasing EI (Durrant, Royston, & Wloch, 1982; Kissileff, Pi-Sunyer, Segal, Meltzer, & Foelsch, 1990). This maladaptive behavioral response to exercise prevents weight loss and subsequently undermines the efficacy of exercise as a weight loss and regulation strategy (Hopkins, King, & Blundell, 2010). Accordingly, identifying individual differences that elevate risk for a positive EB following exercise will help distinguish individuals who are likely to respond unfavorably to exercise regimens and may be at heightened risk for excess weight gain as a result.

Disinhibited eating is one such individual difference factor that has been linked to compensatory EI following exercise (Keim, Canty, Barbieri, & Wu, 1996; Visona & George, 2002). To date, three studies have examined the influence of disinhibited eating on EI following exercise. These studies demonstrated that disinhibited eating is associated with increased EI following acute and recurrent bouts of exercise among overweight (Keim, et al., 1996; Visona &

George, 2002) but not normal weight women (Bryant, Finlayson, King, & Blundell, 2006). However, past research has failed to investigate how disinhibited eating influences EE following exercise. Without collecting data on both EI and EE, it is impossible to know how overall EB is impacted by exercise among women high on disinhibited eating. Accordingly, the aim of the present study was to further elucidate the effect of disinhibited eating on EB following an acute bout of exercise by assessing EI *and* physical activity energy expenditure (PAEE) among both overweight and obese, sedentary women.

For the present study, overweight and obese, sedentary women were recruited to participate in an exercise and nonexercise condition, and EI and PAEE were measured for 24-hours. Relative to individuals low on disinhibited eating, it was hypothesized that individuals high on disinhibited eating would show a greater magnitude of compensatory changes in EI, PAEE, and overall EB in response to exercise. That is, relative to individuals low on disinhibited eating, individuals high on disinhibited eating would significantly increase EI, decrease PAEE, and have a higher EB following exercise.

3.0 METHODS

3.1 PARTICIPANT RECRUITMENT

Participants were overweight or obese, sedentary women recruited from introductory psychology courses at the University of Pittsburgh and the greater community. Recruitment materials were distributed through flyers and electronic announcements, and interested women were encouraged to complete an online screening survey to determine eligibility. To be eligible, participants had to be female, overweight or obese (body mass index [BMI] $\geq$ 25 kg/m^2), nonsmokers, not pregnant or lactating, not currently taking medications that affect weight or food intake, and report no chronic diseases. Participants were further required to be sedentary, as defined by having participated in less than 30 consecutive minutes of aerobic exercise no more than twice per week over the past six months (Visona & George, 2002). Women meeting these eligibility requirements were contacted by the experimenter to make scheduling arrangements. Ineligible women were electronically informed that they did not meet the minimum requirements for participation. In exchange for participation, women recruited from introductory psychology courses were compensated with course credit whereas women recruited from the greater community were compensated with a monetary reward.

3.2 PROCEDURE

Study procedures were approved by the Institutional Review Board of the University of Pittsburgh. The design of the present study was modeled after the methodological approach utilized in similar research investigating the influence of disinhibited eating on EI following an acute bout of exercise (Visona & George, 2002). The present study used a counterbalanced crossover design with two experimental conditions, exercise (E) and nonexercise (NE), which were completed one week apart. All women were randomly assigned to either the E or NE condition at their first assessment and completed the remaining condition one week later. Women completed study procedures independently of other participants. To reduce potential confounding effects associated with the timing of each assessment, all women were scheduled to arrive at the laboratory at 10:00 a.m. To further control for differences in breakfast habits and to ensure that all women began each assessment with the same baseline EI, women were instructed to abstain from eating or drinking anything aside from water on the morning of each assessment. Women were informed that they would receive a standard breakfast upon arrival at the laboratory. The standard breakfast consisted of a Nutri-Grain® bar (Kellogg's®, Battle Creek, MI) and water, which totaled 120 kilocalories (kcals).

3.2.1 Experimental conditions

For the E condition, women engaged in moderate-intensity exercise by walking on a treadmill in a private examination room for 30-minutes. The experimenter recorded heart rate at 1-minute intervals using a Polar heart rate monitor (Polar Pacer; Polar Electro Inc., Port Washington, NY),

and women were maintained at 60% to 70% of their age-predicted maximal heart rate. All women began the E condition by walking on a treadmill at three miles per hour with a 0% grade. If heart rate dropped below 60% of the age-predicted maximal heart rate, the grade of the treadmill was increased until heart rate was sufficiently elevated. Alternatively, if heart rate rose above 70% of the age-predicted maximal heart rate, either the speed or the grade of the treadmill was decreased until heart rate was sufficiently reduced. For the NE condition, women sat in a private examination room for 30-minutes and were given the option to listen to music, read quietly, or do homework and did not engage in any physical activity.

3.2.1.1 First assessment Upon arriving at the laboratory, study procedures were explained and consent was documented. Women were then provided with the standard breakfast and completed a questionnaire battery, which included measures of demographic information, eating pathology, eating behavior, and impulsivity. The examiner recorded the height and weight of each woman. Women were subsequently affixed with a Sensewear Pro Armband™ on their upper right arm above the triceps muscle and completed either the E or NE condition. Once finished with the experimental condition procedures, women were instructed to continue wearing the armband for 24-hours and to bring the equipment back to the laboratory for their second assessment. Women were contacted by the examiner via telephone the day immediately following their first assessment to complete a 24-hour food recall.

3.2.1.2 Second assessment The second assessment was scheduled to take place exactly one week after the first assessment. Upon arriving at the laboratory, women again were provided the standard breakfast and affixed with the Sensewear Pro Armband™. Women then completed the

remaining experimental condition (i.e., E or NE). After completing the experimental condition procedures, women were told to continue wearing the armband for 24-hours and were asked to return the equipment to the laboratory at their earliest convenience. Women were contacted by the examiner via telephone the day immediately following their second assessment to complete a 24-hour food recall.

3.3 MEASURES

3.3.1 Demographic information

Participants reported demographic information, including age, race and ethnicity, marital status, number of children, living situation, job status, income, and year in college.

3.3.2 Body mass index

Height was measured using a mounted stadiometer, and weight was measured using a portable digital scale while participants were dressed in street clothes without shoes. BMI was calculated by taking weight in kilograms divided by height in meters squared.

3.3.3 Impulsivity

Impulsivity was measured using the 59-item UPPS-P Impulsive Behavior Scale (Whiteside & Lynam, 2001), which assesses five distinct facets of impulsive behavior. The Negative Urgency

subscale consists of 12-items assessing the tendency to act impulsively in the face of intense negative affect. The Positive Urgency subscale consists of 14-items assessing the tendency to act impulsively in the face of intense positive affect. The Lack of Premeditation subscale consists of 11-items assessing the tendency to act rashly without regard to consequences. The Lack of Perseverance subscale consists of 10-items assessing the ability to maintain engagement with boring tasks. The Sensation Seeking subscale consists of 12-items assessing the tendency to pursue exciting and often dangerous activities. The items for each subscale are rated using Likert scale response options ranging from 1 to 4 and are averaged, with higher scores indicating greater levels of impulsivity. The internal consistency coefficients in this sample were 0.79 for the Negative Urgency subscale, 0.94 for the Positive Urgency subscale, 0.83 for the Lack of Premeditation subscale, 0.60 for the Lack of Perseverance subscale, and 0.86 for the Sensation Seeking subscale.

3.3.4 Eating pathology

The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994) was used to determine the presence of disordered eating cognitions and behaviors. The EDE-Q is a 38-item, self-report version of the Eating Disorder Examination interview, and assesses eating pathology over the previous 28 days (Fairburn & Cooper, 1993). The EDE-Q produces three types of data, including frequency counts of binge eating episodes, four subscale scores, including Restraint (5-items), Eating Concerns (5-items), Shape Concerns (8-items), and Weight Concerns (5-items), reflecting the severity of the disordered cognitions characteristic of eating disorders, and a Global Score denoting the overall magnitude of eating pathology. Specifically, women report whether they engaged in objective binge eating (OBE), characterized by the consumption of an

objectively large amount of food accompanied by a sense of loss of control over eating, and subjective binge eating (SBE), defined by a loss of control over eating without the necessary consumption of an objectively large amount of food, over the previous 28 days and record the number of OBE and SBE episodes they experienced. In addition, the items for each subscale consist of Likert scale response options ranging from 1 to 6 and are averaged, with higher scores indicating greater levels of disordered eating cognitions. The Global Score is subsequently computed by averaging each of the four subscales, with higher scores indicating greater levels of eating pathology. The internal consistency coefficients in this sample were 0.75 for the Restraint subscale, 0.78 for the Eating Concerns subscale, 0.86 for the Shape Concerns subscale, 0.78 for the Weight Concerns subscale, and 0.87 for the Global Score.

3.3.5 Eating behavior

The Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) is a 51-item measure assessing specific dimensions of eating behavior and yields three subscales, including Restraint, Disinhibited Eating, and Hunger. The Restraint subscale consists of 21-items measuring cognitive restriction of calorie intake. The Disinhibited Eating subscale consists of 16-items measuring responsiveness to emotional and situational stimuli that trigger eating behavior. The Hunger subscale consists of 14-items measuring internal and external sensitivity to hunger cues. The TFEQ consists of true or false and Likert scale response items. Each item is assigned a binary code and summed, with higher scores indicating greater levels of restraint, disinhibited eating, or hunger. The internal consistency coefficients in this sample were 0.74 for the Restraint subscale, 0.69 for the Disinhibited Eating subscale, and 0.61 for the Hunger subscale.

3.3.6 Energy intake

EI was assessed using the Nutrition Data System for Research (NDSR; University of Minnesota, Minneapolis, MN), which is a computer-assisted interview designed to collect and analyze 24-hour food recalls. EI data for each woman was gathered over the phone by the principal investigator the day following each assessment and entered directly into the NDSR program. The NDSR food recall interview is structured using a multiple-pass approach, which provided each woman several opportunities to recall EI for the prior 24-hour period. Women also were provided a booklet consisting of standard portion sizes and measurements, allowing for a more accurate estimation of the amount of food consumed. The NDSR program searches for foods and their variable ingredients and preparation methods and automatically calculates nutrient values. EI in kcals was determined from the foods and beverages consumed on each experimental day as were the total grams of macronutrient intake and the percent of total EI from macronutrients.

3.3.7 Physical activity energy expenditure

The SenseWear Pro Armband™ (Body Media, Pittsburgh, PA) is a commercially available device that has been found to provide accurate estimates of PAEE when compared to indirect calorimetry (Jakicic et al., 2004). The device records data from a variety of parameters including heat flux, accelerometry, galvanic skin response, skin temperature, near-body temperature, and demographic characteristics, including gender, age, height, and weight, which are collectively used to estimate PAEE as well as the amount of time spent being physically active or sedentary. Although women were instructed to wear the device for 24-hours, women also were provided with an activity log to record any times during which they removed the device and to note what

activities they performed while the device was removed. Metabolic equivalents (METs) were utilized to compute estimates of the energy expended from the reported activities performed during periods in which the monitor was removed using the following formula: *kcals = METs x weight (kg) x time (hours)*. Women who failed to report what activities they performed during periods in which the device was removed were assumed to have been sedentary, and a MET of 1.0 was utilized to compute estimates of PAEE (Esliger, Copeland, Barnes, & Tremblay, 2005). These computed estimates of PAEE were subsequently added to the PAEE data extracted directly from the device to obtain a total estimate of PAEE in kcals for each experimental day. The number of minutes women spent being physically active or sedentary on each experimental day also were computed.

4.0 STATISTICAL ANALYSIS

Sample size estimation was calculated *a priori* using R 3.0.1 (R Foundation for Statistical Computing, Vienna, Austria). Because there were no reported findings on the relationship between disinhibited eating and PAEE following exercise, sample size estimation was based on expected differences in EI across experimental days occurring as a function of disinhibited eating. Based on the results reported by Visona & George (2002), it was expected that overweight women higher on disinhibited eating would increase EI by approximately 40% following exercise whereas overweight women lower on disinhibited eating would decrease EI by approximately 15% following exercise. Using these reported outcomes to calculate parameter estimates for a regression model and accounting for attrition rates of 15%, a total sample size of 50 participants was estimated to provide a power of 0.82 to detect a moderate interaction effect ($\beta = 0.25$) between disinhibited eating and experimental day predicting EI. Given that EI following exercise is expected to be greater among obese relative to overweight women higher on disinhibited eating (Hill, Melby, Johnson, & Peters, 1995), these estimates were believed to be conservative. Accordingly, a total of 62 women were enrolled to provide sufficient power to detect statistical effects.

All statistical tests were conducted using SAS 9.4 (SAS Institute, Cary, NC) and were evaluated with a two-sided, Type I error rate of 0.05. Only women with complete data were retained for final analysis. Independent samples t-tests and chi-square analyses were utilized to

compare women with complete data against women with incomplete data. Any variable that significantly differed between women with complete and incomplete data was subsequently considered as a potential covariate in the final statistical models. Variable distributions were inspected graphically to identify influential points among the subset of women with complete data and statistical outliers were removed. Baseline characteristics were used to describe the final sample and statistical assumptions were tested and satisfied.

Only the EI and PAEE data recorded on each experimental day (i.e., from the initiation of the experimental condition through 11:59 p.m. of that same day) were used for analysis. EB was computed for each experimental day by subtracting PAEE from EI, such that positive scores denoted a positive EB. Average differences in EI, PAEE, and EB across experimental days were assessed using paired samples t-tests. Additional paired samples t-tests were conducted to compare differences in total and percent macronutrient intake and in the amount of time spent being physically active or sedentary across experimental days. To assess whether differences in PAEE and EB as well as the amount of time spent being physically active or sedentary across experimental days were driven by factors associated with the experimental conditions, these analyses were rerun accounting for the energy expended and the amount of time spent being physically active or sedentary during the experimental conditions. To identify individuals who engaged in behavioral compensation, difference scores were computed for EI, PAEE, and EB by subtracting the values for each variable obtained on the NE day from those obtained on the E day, such that positive scores reflected greater EI, PAEE, and EB on the E day relative to the NE day. Women were considered to have engaged in behavioral compensation if they increased EI, decreased PAEE, or had a higher EB on the E day relative to the NE day. Individual variability in the difference scores for EI, PAEE, and EB were illustrated graphically to depict variance in

behavioral compensation. Independent samples t-tests and chi-square analyses were performed to determine whether there were any differences in EI, PAEE, EB, total and percent macronutrient intake, the amount of time spent being physically active or sedentary, demographic characteristics, eating pathology, or eating behavior between women who engaged in behavioral compensation and those who did not.

Separate linear mixed effects models with repeated measurement were used to determine whether disinhibited eating predicted differences in EI, PAEE, or EB across experimental days. Additional models were run to determine whether disinhibited eating predicted differences in total and percent macronutrient intake or the amount of time spent being physically active or sedentary across experimental days. To ensure that the effects of disinhibited eating on PAEE and EB as well as the amount of time spent being physically active or sedentary across experimental days were not driven by factors associated with the experimental conditions, the models were rerun accounting for the energy expended and the amount of time spent being physically active or sedentary during the experimental conditions. All models were fit using a restricted maximum likelihood (REML) method, and a compound symmetry covariance structure was specified. Subject was considered a random effect, and experimental day was designated as the repeated measurement. All other predictors were considered fixed effects. Continuous predictors were centered to facilitate the interpretation of interaction coefficients (Cohen, Cohen, West, & Aiken, 2003). The main effect of disinhibited eating and the interaction between disinhibited eating and experimental day were included in each model. Because BMI is predictive of both EI (Ledikwe, et al., 2006) and PAEE (Delany, 2012), BMI also was controlled for in each model. Although additional covariates, including age, race, income, and dietary restraint, were considered, none were found to be significant and therefore were not included in

the final models. In addition to disinhibited eating, impulsivity, eating pathology, and eating behavior were explored as potential predictors of EI, PAEE, and EB across experimental days.

5.0 RESULTS

5.1 PARTICIPANT CHARACTERISTICS

A total of 268 women were screened, and 62 women were enrolled (see Figure 1). However, due to issues of noncompliance and equipment failure, complete data were available for only 49 women. Women with incomplete data (n = 13) were more likely to be underclassmen (χ^2 = 11.61; p = 0.02) and unemployed (χ^2 = 13.80; p = 0.008) relative to women with complete data. Women with incomplete data also endorsed higher eating ($t(60) = 2.89$; p = 0.005) and shape ($t(60) = 3.20$; p = 0.002) concerns and reported consuming fewer kcals on the E day ($t(59) = -2.41$; p = 0.02) than did women with complete data. Among the subset of women with complete data, one woman was found to have a PAEE more than three standard deviations above the mean on both experimental days and reported an EI more than three standard deviations above the mean on the NE day. This woman was determined to be a statistical outlier and was subsequently excluded from further analysis.

The final analytic sample consisted of 48 women. Descriptive characteristics are displayed in Table 1. On average, women were 21.33 (SD = 2.09) years old. A majority were white (62.5%; n = 30), with 20.8% (n = 10) identifying as black, 10.4% (n = 5) Asian, and 6.3% (n = 3) “other.” The average BMI was 30.69 (SD = 5.07), with 56.3% (n = 27) of women being

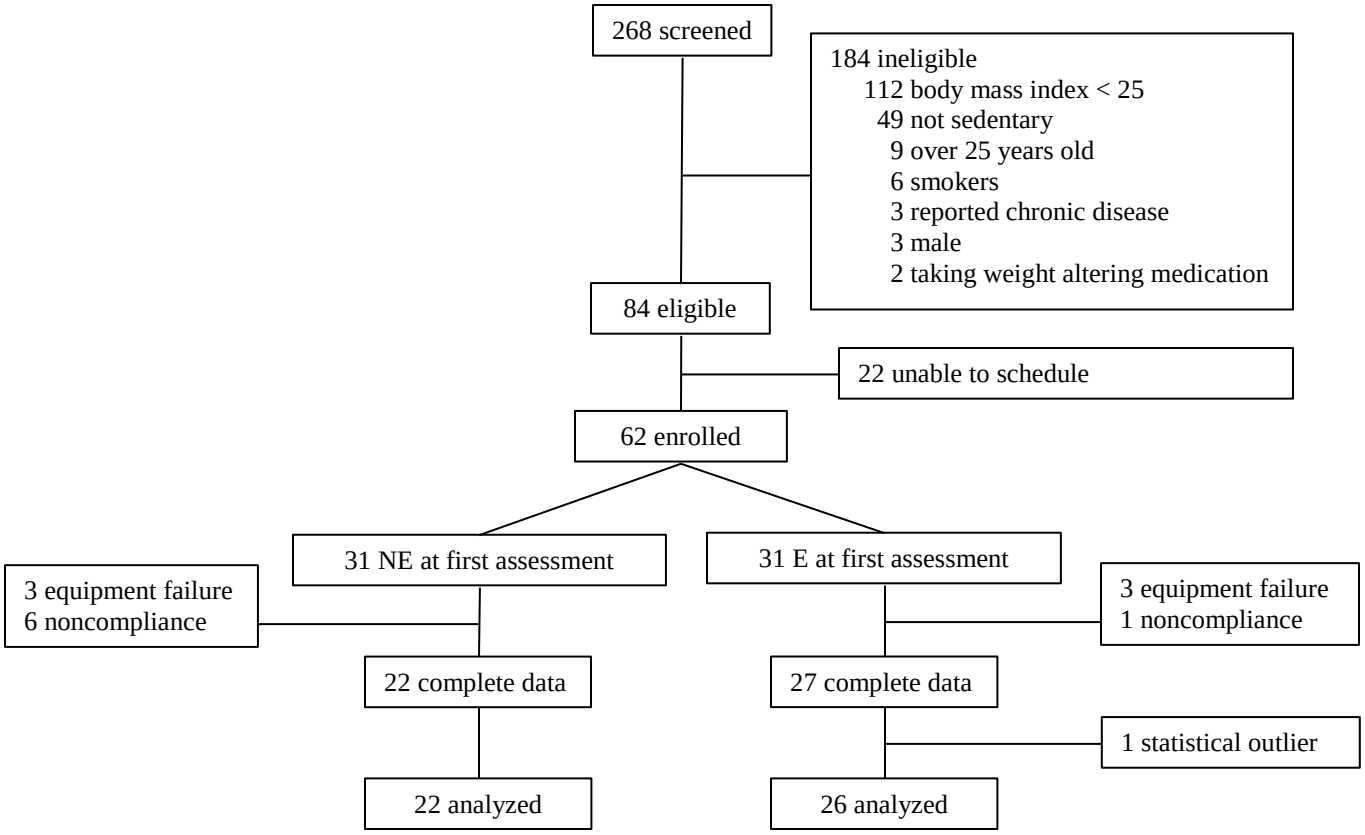

Figure 1. Diagram Detailing the Number of Women Screened, Enrolled, and Retained For Statistical Analysis
Note: NE, nonexercise; E, exercise.

overweight (BMI ≥ 25 and < 30) and the remainder being obese (BMI ≥ 30). As displayed in Table 2, women endorsed levels of disordered eating cognitions consistent with nonclinical undergraduate populations (Luce, Crowther, Pole, 2008). Moreover, 56.3% (*n* = 27) of women reported at least one episode of disordered eating behavior in the past 28 days. Specifically, 60.4% (*n* = 29) of women reported eating an objectively large amount of food (*Range* = 1 to 50 episodes), and 50% of women reported engaging in binge eating, with 43.8% (*n* = 21) reporting OBE (*Range* = 1 to 40 episodes) and 27.1% (*n* = 13) reporting SBE (*Range* = 1 to 15 episodes).

Table 1. Descriptive Characteristics

Characteristic (N = 48)	Mean	SD
Age	21.33	2.09
Weight (lbs.)	188.24	34.86
BMI (kg/m^2)	30.69	5.07
Restraint	3.77	1.78
Disinhibited Eating	6.56	2.2
Hunger	5.81	2.78
	%	n
% White	62.5	30
% College Freshman	25	12
% Employed	72.9	35
% Income < $30,000	29.2	14

Note: BMI; body mass index

Table 2. Eating Cognitions and Behaviors

Eating Cognitions (N = 48)	Mean	SD
Eating Concerns	1.04	0.93
Weight Concerns	2.88	1.21
Shape Concerns	3.00	1.26
Global Score	2.03	0.94
Eating Behaviors	%	n
Objective Overeating	60.4	29
Binge Eating	50.0	24
OBE	43.8	21
SBE	27.1	13

Note: OBE, objective binge eating; SBE, subjective binge eating

5.2 ENERGY INTAKE, PHYSICAL ACTIVITY ENERGY EXPENDITURE, AND ENERGY BALANCE ACROSS EXPERIMENTAL DAYS

Average EI, PAEE, and EB across experimental days are presented in Table 3. Paired samples t-tests indicated that the energy expended during the E condition (M = 215.47; SD = 60.60) was significantly higher than the energy expended during the NE condition (M = 40.81; SD = 5.52; $t(47) = -21.13$; $p < 0.0001$). In addition, PAEE was significantly higher on the E day relative to the NE day ($t(47) = -3.68$, $p = 0.001$), and EB was significantly lower on the E day relative to the NE day ($t(47) = 2.07$, $p = 0.04$). However, the differences in PAEE ($t(47) = -1.34$; $p = 0.19$) and EB ($t(47) = 0.97$; $p = 0.34$) across experimental days did not hold when the energy expended during each experimental condition was removed. There was no difference in EI across experimental days ($t(47) = 0.38$; $p = 0.71$).

Macronutrient intake and the time spent being physically active or sedentary across experimental days also are presented in Table 3. There were no differences in the grams of carbohydrate ($t(47) = 1.26$; $p = 0.21$), fat ($t(47) = -0.0005$; $p = 0.10$), or protein ($t(47) = -0.07$; $p = 0.94$) intake across experimental days. In addition, there were no differences in the percentage of EI from carbohydrates ($t(47) = 1.06$; $p = 0.30$), fat ($t(47) = -0.47$; $p = 0.64$), or protein ($t(47) = 0.45$; $p = 0.66$) across experimental days. The number of minutes women spent being physically active was significantly higher on the E day relative to the NE day ($t(47) = -4.008$, $p < 0.0001$), and the number of minutes women spent being sedentary was significantly lower on the E day relative to the NE day ($t(47) = 3.96$, $p < 0.0001$). However, after removing the number of minutes spent being physically active or sedentary during each experimental condition, the

Table 3. Changes in Energy Intake, Physical Activity Energy Expenditure, and Energy Balance across Experimental Days

	NE		E		Difference*		
	Mean	SD	Mean	SD	Mean	SD	p
EB (kcals)	110.50	949.04	-215.17	1010.11	-325.57	1089.66	**0.04**[a]
EI (kcals)	1850.98	901.97	1794.27	660.02	-56.70	1043.87	0.71
Total Carbohydrate (g)	231.55	128.38	205.15	70.03	-26.39	144.81	0.21
Total Fat (g)	68.54	35.75	68.54	34.68	0.003	44.06	1.00
Total Protein (g)	71.38	35.83	71.85	35.99	0.47	45.86	0.94
Percent Carbohydrate (% of total EI)	46.64	9.19	47.45	11.36	0.81	14.37	0.30
Percent Fat (% of total EI)	33.22	7.74	34.10	9.23	0.88	12.91	0.64
Percent Protein (% of total EI)	16.52	5.81	16.03	5.30	-0.49	7.54	0.66
PAEE (kcals)	1740.48	487.73	2009.44	739.81	268.96	506.84	**0.001**[b]
Physical Activity (min)	84.60	61.8	121.80	88.80	-37.20	64.80	**< 0.0001**[c]
Sedentary (min)	718.80	72.60	667.80	114.60	-51.60	90.00	**< 0.0001**[d]

Note: EB, energy balance; EI, energy intake; PAEE, physical activity energy expenditure; NE, nonexercise; E, exercise

*Difference scores were calculated by subtracting the values for each variable obtained on the exercise day from those obtained on the nonexercise day (E – NE), such that positive scores indicated a higher value on the exercise day relative to the nonexercise day.

[a]Difference no longer significant after accounting for the energy expended during nonexercise and exercise conditions ($t(47) = 0.97$; $p = 0.34$).

[b]Difference no longer significant after accounting for the energy expended during nonexercise and exercise conditions ($t(47) = -1.34$; $p = 0.19$).

[c]Difference no longer significant after accounting for the amount of time women were physically active during the exercise condition ($t(47) = -0.80$; $p = 0.43$).

[d]Difference no longer significant after accounting for the amount of time women were sedentary during the nonexercise condition ($t(47) = 1.64$; $p = 0.11$).

differences in the number of minutes women spent being physically active ($t(47) = -0.80$; $p = 0.43$) or sedentary ($t(47) = 1.64$; $p = 0.11$) across experimental days were no longer significant.

5.3 BEHAVIORAL COMPENSATION

Individual variability in difference scores for EI, PAEE, and EB are illustrated in Figures 2 through 4. As shown in Figure 2, 54% ($n = 26$) of women had a lower EI on the E day relative to the NE day ($M = -778.08$; $SD = 747.82$) whereas 46% ($n = 22$) of women had a greater EI on the E day relative to the NE day ($M = 795.83$; $SD = 606.06$). As shown in Figure 3, 73% ($n = 35$) of women had a higher PAEE on the E day relative to the NE day ($M = 469.49$; $SD = 431.10$) whereas 27% ($n = 13$) of women had a lower PAEE on the E day relative to the NE day ($M = -270.70$; $SD = 215.70$). Finally, as shown in Figure 4, 67% ($n = 32$) of women had a lower EB on the E day relative to the NE day ($M = -894.68$; $SD = 805.31$) whereas 33% ($n = 16$) of women had a higher EB on the E day relative to the NE day ($M = 812.36$; $SD = 554.96$).

In total, 63% ($n = 30$) of women were found to have engaged in behavioral compensation following exercise as defined by increased EI, decreased PAEE, or a higher EB on the E day relative to the NE day. Among compensators, 73% ($n = 22$) reported compensatory increases in EI whereas 43% ($n = 13$) demonstrated compensatory reductions in PAEE. Specifically, 57% ($n = 17$) compensated by solely increasing EI, 27% ($n = 8$) compensated by solely reducing PAEE, and 17% ($n = 5$) compensated by both increasing EI and reducing PAEE. These compensatory

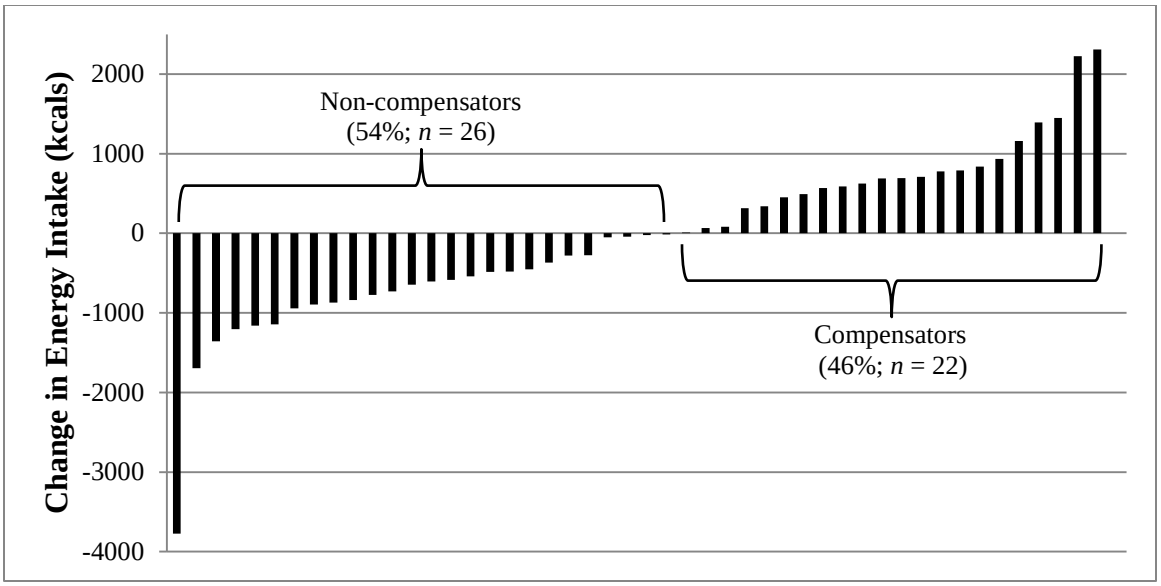

Figure 2. Individual Differences in Change in Energy Intake across Experimental Days
Note: The difference in energy intake across experimental days was calculated by subtracting the energy intake reported on the nonexercise day from that reported on the exercise day. Women who reported greater energy intake on the exercise day relative to the nonexercise day were considered to have engaged in behavioral compensation following exercise.

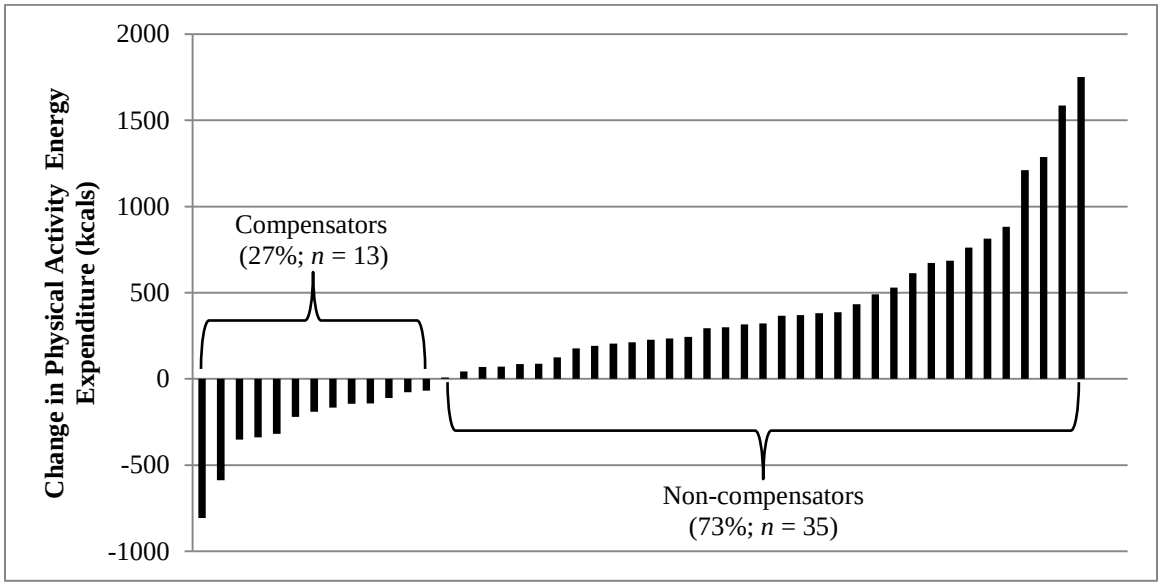

Figure 3. Individual Differences in Change in Physical Activity Energy Expenditure across Experimental Days
Note: The difference in physical activity energy expenditure across experimental days was calculated by subtracting the physical activity energy expenditure recorded on the nonexercise day from that recorded on the exercise day. Women with less physical activity energy expenditure on the exercise day relative to the nonexercise day were considered to have engaged in behavioral compensation following exercise.

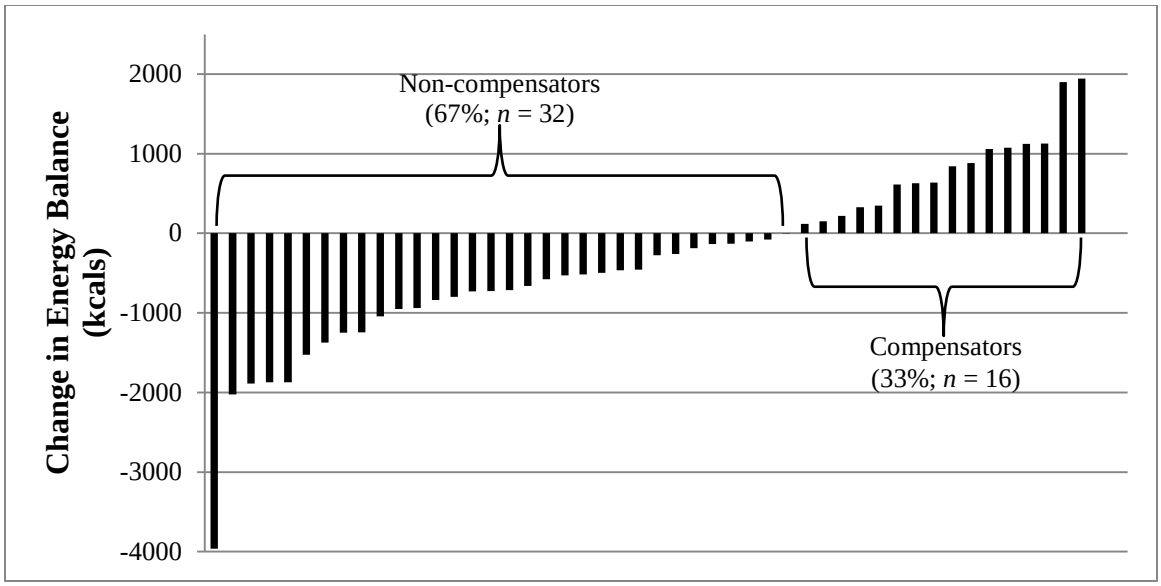

Figure 4. Individual Differences in Change in Energy Balance across Experimental Days
Note: The difference in energy balance across experimental days was calculated by subtracting the energy balance determined on the nonexercise day from that determined on the exercise day. Women who reported a higher energy balance on the exercise day relative to the nonexercise day were considered to have engaged in behavioral compensation following exercise.

changes in EI and PAEE resulted in a higher EB on the E day relative to the NE day among 53% (n = 16) of compensators.

As shown in Table 4, compensators had a significantly higher EB ($t(46) = -5.65$; $p < 0.001$) and EI ($t(46) = -4.76$; $p < 0.001$) as well as greater carbohydrate ($t(46) = -4.19$; $p < 0.001$), fat ($t(46) = -2.93$; $p = 0.005$), and protein ($t(46) = -3.13$; $p = 0.003$) intake on the E day relative to the NE day compared to non-compensators. More specifically, compensators had both a lower EB ($t(46) = 2.81$; $p = 0.007$) and EI ($t(46) = 2.73$; $p = 0.009$) on the NE day and both a higher EB ($t(46) = -2.38$; $p = 0.02$) and EI ($t(46) = -2.94$; $p = 0.005$) on the E day compared to non-compensators. Of note, the effect of EB persisted after accounting for the energy expended during the experimental conditions for both the E ($t(46) = -2.45$; $p = 0.02$) and NE ($t(46) = 2.82$; $p = 0.01$) day. With regard to macronutrient intake, compensators reported lower carbohydrate

Table 4. Differences in Energy Intake, Physical Activity Energy Expenditure, and Energy Balance between Non-compensators and Compensators

	Non-compensators (n = 18)		Compensators (n = 30)		
	Mean	SD	Mean	SD	p
$EB_{(NE)}$	574.88	958.91	-168.13	840.55	**0.007**
$EI_{(NE)}$	2281.51	946.37	1592.66	780.65	**0.009**
Total $Carbohydrate_{(NE)}$	303.67	146.08	188.27	95.04	**0.002**
Total $Fat_{(NE)}$	81.03	30.79	61.04	36.89	0.06
Total $Protein_{(NE)}$	80.94	29.35	65.65	38.54	0.15
Percent $Carbohydrate_{(NE)}$	52.84	7.16	47.72	9.83	0.06
Percent $Fat_{(NE)}$	32.48	7.21	33.66	8.14	0.61
Percent $Protein_{(NE)}$	14.93	4.47	17.48	6.35	0.14
$PAEE_{(NE)}$	1706.63	477.84	1760.79	500.55	0.71
Physical $Activity_{(NE)}$	82.08	66.48	85.74	60	0.84
$Sedentary_{(NE)}$	728.34	78.78	713.28	69.3	0.49
$EB_{(E)}$	-641.65	921.17	40.73	987.7	**0.02**
$EI_{(E)}$	1458.69	514.89	1995.62	664.47	**0.005**
Total $Carbohydrate_{(E)}$	180.09	63.17	220.19	70.61	**0.05**
Total $Fat_{(E)}$	58.73	27.46	74.43	37.57	0.13
Total $Protein_{(E)}$	56.83	28.41	80.86	37.44	**0.02**
Percent $Carbohydrate_{(E)}$	50.53	12.24	45.60	10.57	0.15
Percent $Fat_{(E)}$	35.85	9.02	33.05	9.34	0.31
Percent $Protein_{(E)}$	15.24	4.58	16.50	5.71	0.43
$PAEE_{(E)}$	2100.34	863.7	1954.89	664.47	0.52
Physical $Activity_{(E)}$	136.02	66.48	113.34	82.62	0.40
$Sedentary_{(E)}$	658.02	110.04	673.28	118.32	0.66
$EB_{(Diff)}$	-1216.54	900.06	208.86	813.20	< **0.0001**
$EI_{(Diff)}$	-822.82	866.79	402.97	861.80	< **0.0001**
Total $Carbohydrate_{(Diff)}$	-123.58	151.13	31.92	105.90	< **0.0001**
Total $Fat_{(Diff)}$	-22.31	27.39	13.39	47.04	**0.005**
Total $Protein_{(Diff)}$	-24.11	34.61	15.21	45.90	**0.003**
Percent $Carbohydrate_{(Diff)}$	-2.31	14.64	-2.12	14.46	0.97
Percent $Fat_{(Diff)}$	3.37	12.08	-0.62	13.35	0.31
Percent $Protein_{(Diff)}$	0.31	6.75	-0.97	8.04	0.57
$PAEE_{(Diff)}$	393.72	468.32	194.11	521.84	0.19
Physical $Activity_{(Diff)}$	53.94	56.40	27.54	68.39	0.18
$Sedentary_{(Diff)}$	-70.32	51.18	-39.90	105.78	0.26

Note: $EB_{(NE)}$, energy balance in kcals on nonexercise day; $EI_{(NE)}$, energy intake in kcals on nonexercise day; Total $Carbohydrate_{(NE)}$, carbohydrate intake in grams on nonexercise day; Total $Fat_{(NE)}$, fat intake in grams on nonexercise day; Total $Protein_{(NE)}$, protein intake in grams on nonexercise day; Percent $Carbohydrate_{(NE)}$, percent of total energy intake from carbohydrates on nonexercise day; Percent $Fat_{(NE)}$, percent of total energy intake from fat on nonexercise day; Percent $Protein_{(NE)}$, percent of total energy intake from protein on nonexercise day; $PAEE_{(NE)}$, energy expenditure in kcals from physical activity on nonexercise day; Physical $Activity_{(NE)}$, physical activity in minutes on nonexercise day; $Sedentary_{(NE)}$, sedentary behavior in minutes on nonexercise day; $EB_{(E)}$, energy balance in kcals on exercise day; $EI_{(E)}$, energy intake in kcals on exercise day; Total $Carbohydrate_{(E)}$, carbohydrate intake in grams on exercise day; Total $Fat_{(E)}$, fat intake in grams on exercise day; Total $Protein_{(E)}$, protein intake in grams on exercise day; Percent $Carbohydrate_{(E)}$, percent of total energy intake from carbohydrates on exercise day; Percent $Fat_{(E)}$, percent of total energy intake from fat on exercise day; Percent $Protein_{(E)}$, percent of total energy intake from protein on exercise day; $PAEE_{(E)}$, energy expenditure in kcals from physical activity on exercise day; Physical $Activity_{(E)}$, physical activity in minutes on exercise day; $Sedentary_{(E)}$, sedentary behavior in minutes on exercise day; Diff, all differences scores computed by subtracting the variable total on the nonexercise day from that on the exercise day (E – NE), such that positive scores indicated a higher value on the exercise day relative to the nonexercise day

intake ($t(46) = 3.32$; $p = 0.002$) on the NE day and higher carbohydrate ($t(46) = -1.98$; $p = 0.05$) and protein ($t(46) = -2.34$; $p = 0.02$) intake on the E day relative to non-compensators. Compensators did not differ from non-compensators in protein ($t(46) = 1.45$; $p = 0.15$) or fat ($t(46) = 1.93$; $p = 0.06$) intake on the NE day or fat intake ($t(46) = -1.54$; $p = 0.13$) on the E day. In addition, there were no significant differences between compensators and non-compensators in the percentage of EI from carbohydrates ($t(46) = -0.04$; $p = 0.97$), fat ($t(46) = 1.04$; $p = 0.31$), or protein ($t(46) = 0.57$; $p = 0.57$) across experimental days. There also were no significant differences in PAEE ($t(46) = 1.32$; $p = 0.19$) or the amount of time spent being physically active ($t(46) = 1.38$; $p = 0.18$) or sedentary ($t(46) = -1.38$; $p = 0.19$) across experimental days between compensators and non-compensators.

As shown in Table 5, compensators were more likely to report OBE ($\chi^2 = 7.80$; $p = 0.05$) than were non-compensators. No additional differences in demographic characteristics, eating pathology, or eating behavior between compensators and non-compensators emerged. Women who reported OBE were subsequently found to be higher on disinhibited eating ($t(46) = -2.67$, $p = 0.006$), more likely to report SBE ($\chi^2 = 7.97$; $p = 0.005$), and had greater eating ($t(46) = -4.10$, $p < 0.0001$), weight ($t(46) = -3.35$, $p = 0.002$), and shape concerns ($t(46) = -2.93$, $p = 0.005$) than were women who did not report OBE.

Table 5. Differences in Demographic Characteristics, Eating Cognitions, and Eating Behaviors between Non-compensators and Compensators

	Non-compensators (*n* = 18)		Compensators (*n* = 30)		
	Mean	SD	Mean	SD	p
Age	21.34	2.09	21.3	2.15	0.89
Weight (lbs.)	182.14	36.19	191.91	34.13	0.35
BMI (kg/m2)	29.91	5.7	31.16	4.7	0.42
Restraint	3.78	1.73	3.77	1.83	0.98
Disinhibited Eating	6.67	2.52	6.5	2.03	0.80
Hunger	6.39	2.85	5.47	2.73	0.27
Eating Concerns	1.02	1.09	1.05	0.84	0.91
Weight Concerns	2.82	1.33	2.91	1.15	0.81
Shape Concerns	3.06	1.37	2.96	1.22	0.79
Global Score	2.00	1.11	2.05	0.83	0.86
	%	n	%	n	
% White	39.29	11	67.86	19	0.94
% College Freshman	17.86	5	25.00	7	0.77
% Employed	39.29	11	89.29	25	0.72
% Income < $30,000	17.86	5	32.14	9	0.61
% Objective Overeating	32.14	9	103.57	29	0.25
% OBE	25.00	7	46.57	14	**0.05**
% SBE	17.86	5	28.57	8	0.93

Note: BMI, body mass index; OBE, objective binge eating; SBE, subjective binge eating

5.4 DISINHIBITED EATING AS A PREDICTOR OF BEHAVIORAL COMPENSATION

5.4.1 Energy intake, physical activity energy expenditure, and energy balance

There was no significant main effect of disinhibited eating or interaction between disinhibited eating and experimental day predicting EI, PAEE, or EB (see Table 6).

Table 6. Linear Mixed Effects Models Assessing Disinhibited Eating as a Predictor of Behavioral Compensation

Energy Balance	B	SE	p
Intercept	110.50	143.18	0.14
Experimental Day	-325.66	158.92	**0.05**[a]
BMI	-8.79	23.87	0.71
Disinhibited Eating	--	--	--
NE Day	40.41	66.00	0.54
E Day	53.54	66.00	0.54
Disinhibited Eating*Experimental Day	13.13	72.95	0.86
Energy Intake	**B**	**SE**	**p**
Intercept	1850.98	114.21	**< 0.0001**
Experimental Day	-56.70	152.25	0.71
BMI	5.59	17.06	0.75
Disinhibited Eating	--	--	--
NE Day	54.09	52.60	0.31
E Day	65.85	52.60	0.22
Disinhibited Eating*Experimental Day	11.77	69.89	0.87
Physical Activity Energy Expenditure	**B**	**SE**	**p**
Intercept	1740.48	91.48	**< 0.0001**
Experimental Day	268.96	73.95	**0.0007**[b]
BMI	14.38	16.77	0.40
Disinhibited Eating	--	--	--
NE Day	12.31	42.21	0.74
E Day	13.68	42.21	0.74
Disinhibited Eating*Experimental Day	1.36	33.94	0.97

Note: BMI, body mass index; NE, nonexercise; E, exercise

[a]Difference no longer significant after accounting for the energy expended during the exercise and nonexercise conditions (B = -151.01; SE = 140.56; p = 0.34).

[b]Difference no longer significant after accounting for the energy expended during the exercise and nonexercise conditions (B = 94.31; SE = 70.90; p = 0.19).

5.4.2 Macronutrient intake

There was no significant main effect of disinhibited eating or interaction between disinhibited eating and experimental day predicting fat or protein intake (see Table 7). Although there was no significant main effect of disinhibited eating predicting carbohydrate intake on the NE day, disinhibited eating was a significant predictor of carbohydrate intake on the E day above and beyond covariate effects. Specifically, for every one unit increase in disinhibited eating there was a 13.84 g increase in carbohydrate intake on the E day. The interaction effect between disinhibited eating and experimental day predicting carbohydrate intake was not significant. In addition, there was no significant main effect of disinhibited eating or interaction between disinhibited eating and experimental day predicting the percentage of EI from carbohydrates, fat, or protein.

5.4.3 Physical activity and sedentary behavior

There was no significant main effect of disinhibited eating or interaction between disinhibited eating and experimental day predicting the amount of time spent being physically active or sedentary (see Table 7).

5.5 EXPLORATORY PREDICTORS OF BEHAVIORAL COMPENSATION

Given that compensators were more likely to report OBE than were non-compensators, OBE was explored as a potential predictor of EI, PAEE, and EB across experimental days. Additional

Table 7. Linear Mixed Effects Models Assessing Disinhibited Eating as a Predictor of Specific Components of Behavioral Compensation

Total Carbohydrate	B	SE	p
Intercept	231.55	14.77	< **0.0001**
Experimental Day	-26.39	20.89	0.21
BMI	1.02	2.09	0.63
Disinhibited Eating	--	--	--
NE Day	4.00	6.80	0.54
E Day	13.84	6.80	**0.05**
Disinhibited Eating*Experimental Day	9.84	9.59	0.31
Total Fat	**B**	**SE**	**p**
Intercept	68.54	5.16	< **0.0001**
Experimental Day	0.003	6.43	0.99
BMI	0.35	0.81	0.67
Disinhibited Eating	--	--	--
NE Day	0.32	2.38	0.89
E Day	0.43	2.38	0.86
Disinhibited Eating*Experimental Day	0.10	2.95	0.97
Total Protein	**B**	**SE**	**p**
Intercept	71.85	5.24	< **0.0001**
Experimental Day	0.47	6.67	0.94
BMI	0.67	0.81	0.41
Disinhibited Eating	--	--	--
NE Day	-0.37	2.41	0.88
E Day	1.33	2.41	0.58
Disinhibited Eating*Experimental Day	1.70	3.06	0.58
Percent Carbohydrate	**B**	**SE**	**p**
Intercept	47.45	1.50	< **0.0001**
Experimental Day	-2.19	2.09	0.30
BMI	0.13	0.22	0.54
Disinhibited Eating	--	--	--
NE Day	0.24	0.69	0.72
E Day	0.60	0.69	0.39
Disinhibited Eating*Experimental Day	0.36	0.96	0.71
Percent Fat	**B**	**SE**	**p**
Intercept	34.10	1.22	< **0.0001**
Experimental Day	-0.88	1.88	0.64
BMI	0.05	0.16	0.73
Disinhibited Eating	--	--	--
NE Day	0.51	0.56	0.37
E Day	1.02	0.56	0.08
Disinhibited Eating*Experimental Day	0.51	0.86	0.56
Percent Protein	**B**	**SE**	**p**
Intercept	16.03	0.81	< **0.0001**
Experimental Day	-0.49	1.10	0.66
BMI	-0.02	0.12	0.84
Disinhibited Eating	--	--	--
NE Day	-0.33	0.37	0.39
E Day	0.26	0.37	0.49
Disinhibited Eating*Experimental Day	0.59	0.50	0.89

Table 7 (cont'd). Linear Mixed Effects Models Assessing Disinhibited Eating as a Predictor of Specific Components of Behavioral Compensation

Physical Activity	B	SE	p
Intercept	84.38	11.09	< **0.0001**
Experimental Day	37.46	9.44	**0.0003**[a]
BMI	-2.15	2.01	0.29
Disinhibited Eating	--	--	--
NE Day	3.51	5.11	0.51
E Day	5.11	5.11	0.32
Disinhibited Eating*Experimental Day	1.70	4.33	0.70
Sedentary Behavior	**B**	**SE**	**p**
Intercept	718.92	13.86	< **0.0001**
Experimental Day	-51.31	13.11	**0.0003**[b]
BMI	2.84	2.44	0.25
Disinhibited Eating	--	--	--
NE Day	-5.15	6.39	0.42
E Day	-5.23	6.39	0.42
Disinhibited Eating*Experimental Day	-0.06	6.02	0.99

Note: BMI, body mass index; NE, nonexercise; E, exercise

[a]Difference no longer significant after accounting for time spent being physically active during exercise condition (B = 7.46; SE = 9.44; p = 0.43).

[b]Difference no longer significant after accounting for time spent being sedentary during nonexercise condition (B = -21.31; SE = 13.11; p = 0.11).

models were run to determine whether OBE predicted differences in total and percent macronutrient intake or the amount of time spent being physically active or sedentary across experimental days. Furthermore, because women who reported OBE were higher on disinhibited eating than were women who did not report OBE, the synergistic effect of disinhibited eating and OBE also was explored in each model. Although additional predictors related to impulsivity, eating pathology, and eating behavior also were explored, none were significant predictors of EI, PAEE, EB, total or percent macronutrient intake, or the amount of time spent being physically active or sedentary across experimental days and are thus not discussed further.

5.5.1 Energy intake, physical activity energy expenditure, and energy balance

There were no significant main effects of disinhibited eating or OBE predicting EI, PAEE, or EB (see Table 8). There also were no significant interactions between disinhibited eating, OBE, and experimental day predicting EI or PAEE. Although there was no significant interaction between disinhibited eating and experimental day or three-way interaction between disinhibited eating, OBE, and experimental day predicting EB, the interaction between OBE and experimental day was a significant predictor of EB above and beyond covariate effects. Specifically, women who reported OBE had an average EB 742.37 kcals higher than did women who did not report OBE on the E day relative to the NE day, and this effect held after accounting for the energy expended during each experimental condition ($p = 0.04$).

5.5.2 Macronutrient intake

There were no significant main effects of disinhibited eating or OBE or interaction effects between disinhibited eating, OBE, and experimental day predicting fat or protein intake (see Table 9). Similarly, there was no significant main effect of OBE or significant interaction between OBE and experimental day predicting carbohydrate intake. Although there was no significant main effect of disinhibited eating predicting carbohydrate intake on the NE day, there was a significant main effect of disinhibited eating on carbohydrate intake on the E day above and beyond covariate effects, such that, for every one unit increase in disinhibited eating, there was a 57.33 g increase in carbohydrate intake. Moreover, the interaction between disinhibited eating and experimental day was a significant predictor of carbohydrate intake above and beyond covariate effects. Specifically, the effect of disinhibited eating on carbohydrate intake was

Table 8. Linear Mixed Effects Models Assessing Exploratory Predictors of Behavioral Compensation

Energy Balance	B	SE	p
Intercept	192.84	202.21	0.35
Experimental Day	-541.30	214.80	**0.02**[a]
BMI	-9.39	24.97	0.71
OBE	--	--	--
NE Day	-320.55	330.95	0.33
E Day	421.82	330.95	0.21
Disinhibited Eating	--	--	--
NE Day	39.90	83.41	0.57
E Day	40.61	83.41	0.63
OBE*Experimental Day	742.37	347.85	**0.04**
Disinhibited Eating*Experimental Day	0.71	88.47	0.99
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	-137.68	165.78	0.41
E Day	121.86	165.78	0.47
Energy Intake	**B**	**SE**	**p**
Intercept	1966.25	160.97	< **0.0001**
Experimental Day	-200.63	210.01	0.35
BMI	5.66	17.78	0.75
OBE	--	--	--
NE Day	-330.96	262.89	0.21
E Day	217.65	262.89	0.41
Disinhibited Eating	--	--	--
NE Day	-75.81	113.50	0.51
E Day	144.20	113.50	0.21
OBE*Experimental Day	548.61	340.09	0.11
Disinhibited Eating*Experimental Day	220.01	147.96	0.14
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	-70.18	131.85	0.60
E Day	158.29	131.85	0.24
Physical Activity Energy Expenditure	**B**	**SE**	**p**
Intercept	1773.42	130.23	< **0.0001**
Experimental Day	340.67	104.41	**0.002**[b]
BMI	15.05	17.39	0.39
OBE	--	--	--
NE Day	-10.40	213.51	0.96
E Day	-204.17	213.51	0.34
Disinhibited Eating	--	--	--
NE Day	33.40	53.72	0.54
E Day	42.57	53.72	0.95
OBE*Experimental Day	-193.76	169.08	0.26
Disinhibited Eating*Experimental Day	9.17	43.00	0.83
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	67.50	106.85	0.53
E Day	36.44	106.85	0.73

Note: BMI, body mass index; OBE, objective binge eating; NE, nonexercise; E, exercise

[a]Difference no longer significant after accounting energy expended during exercise and nonexercise conditions (B = -354.45; SE = 211.98; p = 0.11).

[b]Difference no longer significant after accounting energy expended during exercise and nonexercise conditions (B = 153.82; SE = 100.20; p = 0.13).

enhanced by a magnitude of 48.53 g on the E day relative to the NE day, and this effect further was found to be marginally moderated by OBE status. However, there were no significant main effects of disinhibited eating or OBE or interaction effects between disinhibited eating, OBE, and experimental day predicting the percentage of EI from carbohydrates, fat, or protein.

5.5.3 Physical activity and sedentary behavior

There were no significant main effects of disinhibited eating or OBE predicting the amount of time spent being physically active or sedentary (see Table 9). In addition, there was no significant interaction between disinhibited eating and experimental day or three-way interaction between disinhibited eating, OBE, and experimental day predicting the amount of time spent being physically active or sedentary. However, the interaction between OBE and experimental day was a significant predictor of the amount of time spent being physically active above and beyond covariate effects. Specifically, women who reported OBE spent 42.48 less minutes being physically active on the E day relative to the NE day than did women who did not report OBE, and this interaction held after accounting for the 30-minute period women spent exercising during the E condition ($p = 0.05$). In addition, the interaction between OBE and experimental day was a significant predictor of the amount of time women spent being sedentary above and beyond covariate effects. Specifically, women who reported OBE spent 59.22 more minutes being sedentary than did women who did not report OBE on the E day relative to the NE day, and this interaction held after accounting for the 30-minute period women were sedentary during the NE condition ($p = 0.05$).

Table 9. Linear Mixed Effects Models Assessing Exploratory Predictors of Specific Components of Behavioral Compensation

Total Carbohydrate	B	SE	p
Intercept	233.61	20.68	< **0.0001**
Experimental Day	-27.26	28.48	0.34
BMI	0.89	2.18	0.69
OBE	--	--	--
NE Day	-22.26	33.75	0.51
E Day	26.27	33.75	0.26
Disinhibited Eating	--	--	--
NE Day	10.35	8.53	0.23
E Day	58.33	8.53	**0.05**
OBE*Experimental Day	48.53	46.18	0.30
Disinhibited Eating*Experimental Day	47.98	20.09	**0.02**
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	-18.26	16.93	0.29
E Day	30.17	16.93	0.08
Total Fat	B	SE	p
Intercept	76.68	7.22	< **0.0001**
Experimental Day	-8.06	8.92	0.37
BMI	0.31	0.83	0.71
OBE	--	--	--
NE Day	-16.09	11.80	0.18
E Day	8.09	11.80	0.50
Disinhibited Eating	--	--	--
NE Day	2.55	2.98	0.40
E Day	2.79	2.98	0.10
OBE *Experimental Day	24.18	14.44	0.10
Disinhibited Eating*Experimental Day	0.24	3.67	0.95
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	2.63	5.92	0.67
E Day	8.61	5.92	0.15
Total Protein	B	SE	p
Intercept	77.65	7.41	< **0.0001**
Experimental Day	-7.46	9.23	0.42
BMI	0.74	0.84	0.38
OBE	--	--	--
NE Day	-17.59	12.10	0.15
E Day	7.59	12.10	0.53
Disinhibited Eating	--	--	--
NE Day	0.15	3.05	0.96
E Day	1.84	3.05	0.69
OBE *Experimental Day	25.18	14.95	0.10
Disinhibited Eating*Experimental Day	1.69	3.80	0.66
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	-3.39	6.07	0.58
E Day	3.96	6.07	0.52

Table 9 (cont'd). Linear Mixed Effects Models Assessing Exploratory Predictors of Specific Components of Behavioral Compensation

Percent Carbohydrate	B	SE	p
Intercept	49.60	2.11	< **0.0001**
Experimental Day	1.39	2.88	0.63
BMI	0.13	0.23	0.57
OBE	--	--	--
NE Day	-3.18	3.46	0.36
E Day	2.55	3.46	0.46
Disinhibited Eating	--	--	--
NE Day	0.15	0.87	0.86
E Day	1.07	0.87	0.23
OBE*Experimental Day	5.73	4.67	0.23
Disinhibited Eating*Experimental Day	0.92	1.19	0.44
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	0.74	1.73	0.67
E Day	1.82	1.73	0.30
Percent Fat	B	SE	p
Intercept	33.56	1.72	< **0.0001**
Experimental Day	-0.85	2.66	0.75
BMI	0.02	0.16	0.92
OBE	--	--	--
NE Day	-0.78	2.80	0.78
E Day	2.23	2.80	0.43
Disinhibited Eating	--	--	--
NE Day	0.18	0.71	0.80
E Day	0.89	0.71	0.21
OBE *Experimental Day	3.01	4.30	0.49
Disinhibited Eating*Experimental Day	0.71	1.10	0.33
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	1.05	1.41	0.46
E Day	2.03	1.41	0.16
Percent Protein	B	SE	p
Intercept	15.23	1.14	< **0.0001**
Experimental Day	-0.83	1.57	0.60
BMI	-0.01	0.12	0.91
OBE	--	--	--
NE Day	0.06	1.86	0.97
E Day	0.89	1.86	0.64
Disinhibited Eating	--	--	--
NE Day	0.13	0.47	0.84
E Day	0.58	0.47	0.23
OBE *Experimental Day	0.83	2.54	0.74
Disinhibited Eating*Experimental Day	0.45	0.65	0.84
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	0.98	0.94	0.30
E Day	1.03	0.94	0.28

Table 9 (cont'd). Linear Mixed Effects Models Assessing Exploratory Predictors of Specific Components of Behavioral Compensation

Physical Activity	B	SE	p
Intercept	83.80	15.70	< **0.0001**
Experimental Day	53.02	12.94	**0.0002**[a]
BMI	-2.16	2.09	0.31
OBE	--	--	--
NE Day	12.44	25.74	0.63
E Day	-30.04	25.74	0.25
Disinhibited Eating	--	--	--
NE Day	3.30	5.33	0.38
E Day	8.62	5.33	0.67
OBE*Experimental Day	-42.48	20.95	**0.05**
Disinhibited Eating*Experimental Day	11.56	9.11	0.26
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	11.56	12.88	0.37
E Day	4.37	12.88	0.74
Sedentary Behavior	B	SE	p
Intercept	711.97	19.34	< **0.0001**
Experimental Day	-68.47	17.79	**0.0004**
BMI	2.69	2.50	0.29
OBE	--	--	--
NE Day	-5.41	31.69	0.87
E Day	53.81	31.69	0.09
Disinhibited Eating	--	--	--
NE Day	11.31	7.98	0.16
E Day	10.35	7.98	0.51
OBE*Experimental Day	59.22	28.80	**0.05**
Disinhibited Eating*Experimental Day	-0.96	7.33	0.90
OBE*Disinhibited Eating*Experimental Day	--	--	--
NE Day	-22.16	15.84	0.17
E Day	-1.33	15.84	0.93

Note: OBE, objective binge eating; BMI, body mass index; NE, nonexercise; E, exercise
[a]Difference no longer significant after accounting for time spent being physically active during exercise condition (B =23.02; SE = 12.94; p = 0.08).

6.0 DISCUSSION

Previous research has documented an almost equal response of either weight loss or weight gain among overweight and obese women following supervised exercise (Donnelly & Smith, 2005), suggesting that there is a subset of women who compensate for exercise by increasing dietary intake or decreasing physical activity. Indeed, behavioral compensation can attenuate or even reverse the energy deficit generated by exercise and is estimated to result in 55% to 64% less weight loss than predicted for exercise interventions (Dhurandhar, et al., 2014). Investigations designed to identify individual differences that distinguish women who are at risk for behavioral compensation in response to exercise have shown that overweight but not normal weight women high on disinhibited eating are more likely to engage in dietary compensation following exercise (Keim, Canty, Barbieri, & Wu, 1996; Visona & George, 2002). However, prior work has failed to assess how disinhibited eating relates to energy expended from physical activity in response to exercise, making it impossible to determine whether the magnitude of behavioral compensation observed was sufficient to impact EB. The present study was the first to evaluate the effect of disinhibited eating on EB following an acute bout of exercise by assessing both EI *and* PAEE among a sample of overweight and obese, sedentary women.

Results from the present study demonstrate that the average response to exercise was advantageous for weight loss as evidenced by a decrease in EI and an increase in PAEE, resulting in a lower EB. Importantly, this average response to exercise obscured considerable individual variability, and 63% of the sample was found to behaviorally compensate for exercise.

Of the women who engaged in behavioral compensation, 73% reported compensatory increases in EI following exercise whereas 43% demonstrated compensatory reductions in PAEE. Although the majority of compensators showed compensatory changes in either EI or PAEE following exercise, 17% were found to have both increased EI and decreased PAEE. The magnitude of behavioral compensation was sufficient to produce notable increases in EB among 53% of compensators, which largely was promoted through compensatory increases in EI.

Consistent with prior theories (Thomas, et al., 2012), increased EI was the largest source of behavioral compensation. Indeed, nearly half of women reported increasing EI in response to exercise by an average of 796 kcals. However, this marked compensatory response to exercise was masked by an overall reduction in EI, which highlights the importance of scrutinizing individual variability in behavioral compensation. To date, the majority of studies in this area have based their conclusions on average effects and have reported either no relationship between exercise and subsequent EI (Donnelly, et al., 2003; Donnelly, et al., 2000; Bryner, Toffle, Ullrich, & Yeater, 1997; Lluch, King, & Blundell, 2000; Martins, Kulseng, King, Holst, & Blundell, 2010; Pritchard, Nowson, & Wark, 2004; Snyder, Donnelly, Jacobsen, Hertner, & Jakicic, 1997) or a reduction in EI following exercise (King & Blundell, 1995; King, Lluch, Stubbs & Blundell, 1997; Martins, Morgan, Bloom, & Robertson, 2007; Thompson, Wolfe, Eikelboom, 1988; Westerterp-Plantenga, Verwegen, Ijedema, Wijckmans, & Saris, 1997). Given the present findings and those of additional work documenting large individual variability in behavioral compensation (Donnelly, et al., 2013; Imbeault, Saint-Pierre, Alméras, & Tremblay, 1997; Pomerleau, Imbeault, Parker, & Doucet, 2004; Thompson, Wolfe, & Eikelboom, 1988; Westerterp, Meijer, Janssen, Saris, & Hoor, 1992), previous research may have underestimated compensatory changes in EI following exercise by focusing on the group rather than the

individual level. Accordingly, compensatory increases in EI may partly explain why predictions for weight change are frequently overestimated in exercise interventions, and the accuracy of weight loss targets might be improved by accounting for compensatory responses to exercise (Dhurandhar, et al., 2014).

Although reports of compensatory changes in PAEE also are inconsistent (Alahmadi, Hills, King, & Byrne, 2011; Church, et al., 2009; Hollowell, et al., 2009; Janssen, Fortier, Hudson, & Ross, 2002; Manthou, Gill, Wright, & Malkova, 2010; Meijer, Westerterp, & Verstappen, 1999; McLaughlin, Malkova, & Nimmo, 2006; Rosenkilde, et al., 2012; Ross, et al., 2004; Westerterp, Meijer, Janssen, Saris, & Ten Hoor, 1992), the present study found that nearly one-third of women reduced PAEE following exercise by an average of 271 kcals. This average reduction in PAEE was sufficient to offset the 215 kcals expended during exercise but did not result in notable increases in EB, suggesting that compensatory reductions in PAEE may not substantially impact weight change. These findings are comparable to those of a recent trial in which half of women participating in an exercise training program compensated for exercise by decreasing PAEE at a rate of 233 kcals per day (Di Blasio, et al., 2012) but contrast with evidence showing that compensatory reductions in PAEE can inhibit weight loss (Manthou, Gill, Wright, & Mankova, 2010). However, even modest compensatory reductions in PAEE can attenuate the physiological benefits of exercise (Di Blasio, et al., 2012), indicating that compensatory changes in PAEE remain an important consideration in the context of exercise interventions regardless of weight outcome.

The external validity of prior research on compensatory changes in EB following exercise is limited by the exclusive use of laboratory designs (Finlayson, Bryant, Blundell, & King, 2009; Unick, et al., 2010), which have poor external validity and only consider behaviors that occur

during the time spent in the laboratory. The present design thus extends prior research through the prolonged naturalistic assessment of EI and PAEE and is the first to document that compensatory increases in EB could have profound consequences for weight loss if sustained over time. Over one-third of women had an average increase in EB of 812 kcals following exercise, indicating that compensatory increases in EB could attenuate weight loss or even promote weight gain during exercise intervention. However, despite the high prevalence of behavioral compensation observed, compensatory responses to exercise did not invariably produce a higher EB. Accordingly, identification of individual characteristics that enhance susceptibility for behavioral compensation could be key to differentiating women likely to need additional help losing or maintaining weight.

Contrary to expectations, disinhibited eating was not a significant predictor of behavioral compensation. Interestingly, having experienced binge eating in the past month was the only psychosocial predictor of behavioral compensation. Women who reported binge eating had an average compensatory increase in EB 742 kcals higher following exercise than did women who did not report such behavior. Although binge eating was not predictive of compensatory changes in overall EI or PAEE, women who reported binge eating spent less time being physically active and more time being sedentary following exercise than did women who did not report binge eating. Thus, women who endorse a tendency to lose control over eating may be more likely to compensate for exercise by reducing physical activity rather than increasing food consumption.

Women who reported binge eating also were higher on disinhibited eating, a finding consistent with extant literature (Brown, Bryant, Naslund, King, & Blundell, 2006; Boerner, Spillane, Anderson, & Smith, 2004; d'Amore, et al., 2001; de Zwaan, et al., 2003). Like disinhibited eating, binge eating is associated with a susceptibility to emotional eating (Ricca, et

al., 2009) and a preference for palatable foods (Mathes, Brownley, Mo, & Bulik, 2009) but is further characterized by intense psychological distress and a loss of control over eating (American Psychiatric Association, 2013). Given the high association between disinhibited eating and binge eating, the previous link between disinhibited eating and behavioral compensation (Keim, Canty, Barbieri, & Wu, 1996; Visona & George, 2002) may have been representative of more pathological eating disturbances. Women who endorse binge eating often fail to achieve predicted weight loss in response to comprehensive lifestyle interventions and show significant weight regain (Grilo, Masheb, Wilson, Gueorquieva, & White, 2011; Pagoto, et al., 2007; Wilson, Wilfley, Agras, & Bryson, 2010), causing such individuals frequently to be excluded from weight loss trials. Given the present findings, the poor treatment outcomes observed among women with binge eating may be partly explained by a greater tendency to engage in behavioral compensation. Although future work is needed to further investigate the relationship between binge eating and behavioral compensation, these findings may have important clinical implications. For instance, women who engage in binge eating may require more extensive self-regulatory skills training prior to initiating an exercise regimen to reduce the likelihood of behaviorally compensating for exercise.

Despite the non-significant findings linking disinhibited eating to behavioral compensation, it is worth noting that women higher on disinhibited eating reported greater carbohydrate intake following exercise. There is some evidence to suggest that exercise prompts increased consumption of carbohydrates (Burton, Malkova, Caslake, & Gill, 2010; Galgani, de Jonge, Most, Bray, & Smith, 2010; Pannacciulli, et al., 2007) although this effect has not consistently been demonstrated (Deighton, Zahra, & Stensel, 2012; Penesova, et al., 2011). Moreover, a recent study by Hopkins and colleagues (2013) found that women who relied more

heavily on carbohydrate oxidation during exercise were more susceptible to compensatory increases in EI following exercise, which the authors attributed to an elevated drive to restore carbohydrate balance. Accordingly, it may be that women high on disinhibited eating are more sensitive to perturbations in carbohydrate balance and thus are more likely to compensate for such disturbances by increasing carbohydrate intake. However, because the present study was not specifically designed to highlight compensatory changes in macronutrient intake, it is possible that this relationship occurred as a function of the total energy consumed rather than a preferential selection of carbohydrates. Indeed, disinhibited eating was not predictive of a higher proportion of energy consumed from carbohydrates. Nevertheless, the relationship between disinhibited eating and carbohydrate intake may represent a potential mechanism driving compensatory responses to exercise and warrants further study.

The findings of the present study should be considered in the context of certain limitations. First, 21% of the sample was excluded from the final analysis for incomplete data, resulting from equipment failure and noncompliance with the study protocol. Although the final sample was sufficiently powered according to *a priori* sample size estimation, the exclusion of this subset of women limits power to detect significant effects and may introduce concern regarding sample bias. Indeed, women with incomplete data were more likely to be underclassmen and unemployed than were women with complete data and also endorsed higher eating and shape concerns. However, these variables did not relate to the primary study outcomes. Second, EI was measured through self-report, which is subject to demand characteristics that may have caused women to underreport EI (Scagliusi, et al., 2009). In an effort to limit demand effects, the present study utilized a multiple-pass food recall approach using standardized food amounts and neutral probing questions, which has previously been

shown to reduce the error of self-reported EI to within 3% of that obtained from the doubly-labeled water method (Donnelly, et al., 2013). Third, because the primary aims of the study were to assess modifiable components of EB, only the EE associated with physical activity and not metabolic processes was assessed. As a result, the measure of EB used in this study is not comprehensive. Finally, the present study assessed behavioral compensation following an acute bout of exercise among overweight and obese, sedentary women. Thus, these findings may not be generalizable to other populations.

Despite these limitations, the present study is the first to assess the relative importance of compensatory changes in dietary intake and physical activity in blunting the expected benefits of exercise on EB among overweight and obese, sedentary women. Findings indicate that there is wide individual variability in compensatory responses to exercise that is not captured at the group level and that behavioral compensation is common. Although women tended to compensate for exercise by increasing dietary intake, compensatory reductions in the amount of energy expended from physical activity also were notable. Not all women who engaged in behavioral compensation demonstrated concomitant increases in EB, however, suggesting that assessing compensatory changes in EB may be the most important factor in predicting individual responses to weight loss in the context of exercise. Although disinhibited eating did not predict behavioral compensation, women who reported a recent history of binge eating were at elevated risk of compensatory increases in EB following exercise. Given that over half of the present sample compensated for exercise, these findings substantiate the need for a better understanding of psychosocial predictors and common mechanisms through which behavioral compensation is promoted to better inform intervention efforts.

7.0 BIBLIOGRAPHY

Alahmadi, M. A., Hills, A. P., King, N. A., & Byrne, N. M. (2011). Exercise intensity influences nonexercise activity thermogenesis in overweight and obese adults. *Medicine & Science in Sports & Exercise, 43*(4), 624-631. doi:10.1249/MSS.0b013e3181f7a0cb

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.

Appelhans, B. M. (2009). Neurobehavioral inhibition of reward-driven feeding: Implications for dieting and obesity. *Obesity, 17*(4), 640-647. doi:10.1038/oby.2008.638

Barkeling, B., King, N. A, Näslund, E., & Blundell, J. E. (2007). Characterization of obese individuals who claim to detect no relationship between their eating pattern and sensations of hunger or fullness. *International Journal of Obesity (2005), 31*(3), 435-439. doi:10.1038/sj.ijo.0803449

Birch, L. L., Fisher, J. O., & Davison, K. K. (2003). Learning to overeat: Maternal use of restrictive feeding practices promotes girls' eating in the absence of hunger. *American Journal of Clinical Nutrition, 78*, 215-220. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12885700

Blundell, J. E., & Cooling, J. (2000). Routes to obesity: Phenotypes, food choices and activity. *The British Journal of Nutrition, 83*(Suppl 1), S33–S38. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10889790

Blundell, J. E., & King, N. A. (2000). Exercise, appetite control, and energy balance. *Nutrition, 16*(7-8), 519-522. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10906542

Blundell, J. E., Levin, F., King, N. A., Barkeling, B., Gustafson, T., Hellstrom, P. M.,…Naslund, E. (2008). Overconsumption and obesity: Peptides and susceptibility to weigth gain. *Regulatory Peptides, 149*(1-3), 32–38. doi:10.1016/j.regpep.2007.10.009

Blundell, J. E., Stubbs, R. J., Golding, C., Croden, F., Alam, R., Whybrow, S.,…Lawton, C. L. (2005). Resistance and susceptibility to weight gain: Individual variability in response to a high-fat diet. *Physiology & Behavior*, *86*(5), 614-622. doi:10.1016/j.physbeh.2005.08.052

Boerner, L. M., Spillane, N. S., Anderson, K. G., & Smith, G. T. (2004). Similarities and differences between women and men on eating disorder risk factors and symptom measures. *Eating Behaviors, 5*(3), 209-222. doi:10.1016/j.eatbeh.2004.01.011

Bond, M. J., McDowell, A. J., & Wilkinson, J. Y. (2001). The measurement of dietary restraint, disinhibition and hunger: An examination of the factor structure of the Three Factor Eating Questionnaire (TFEQ). *International Journal of Obesity and Related Metabolic Disorders, 25*(6), 900-906. doi:10.1038/sj.ijo.0801611

Borg, P., Fogelholm, M., & Kukkonen-Harjula, K. (2004). Food selection and eating behaviour during weight maintenance intervention and 2-year follow-up in obese men. *International Journal of Obesity and Related Metabolic Disorders, 28*, 1548-1554. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15543160

Bray, G. A. (2004). Medical consequences of obesity. *The Journal of Clinical Endocrinology and Metabolism, 89*(6), 2583-2589. doi:10.1210/jc.2004-0535

Brown, K., Bryant, E., Naslund, E., King, N., & Blundell, J. (2006). Traits that promote weight gain in obesity, bulimia nervosa and EDNOS. *Obesity Reviews, 7,* 330.

Bryant E. (2006). Understanding disinhibition and its influences on eating behaviour and appetite. PhD Thesis. University of Leeds: Leeds.

Bryant, E. J., Finlayson, G., King, N., Blundell, J. (2006). The influence of acute exercise on liking and preferences for food on high trait disinhibition women. *Obesity Reviews, 7*(2), 343.

Bryant, E. J., King, N. A, & Blundell, J. E. (2005). Can exercise regulate appetite in women with high trait Disinhibition? *BPS proceedings.*

Bryant, E. J., King, N. A, & Blundell, J. E. (2008). Disinhibition: Its effects on appetite and weight regulation. *Obesity Reviews*, *9*(5), 409-419. doi:10.1111/j.1467-789X.2007.00426.x

Bryner, R. W., Toffle, R. C., Ullrich, I. H., & Yeater, R. A. (1997). The effects of exercise intensity on body composition, weight loss, and dietary composition in women. *Journal of the American College of Nutrition, 16*(1), 68-73. doi:10.1080/07315724.1997.10718651

Burton, F., Malkova, D., Caslake, M., & Gill, J. M. (2010). Substrate metabolism, appetite and feeding behaviour under low and high energy turnover conditions in overweight women. *British Journal of Nutrition, 104*(8), 1-11. doi:10.1017/S0007114510002023

Burton-Freeman, B., & Keim, N. L. (2008). Glycemic index, cholecystokinin, satiety and disinhibition. Is there an unappreciated paradox for overweight women? *International Journal of Obesity, 32,* 1647-1654. doi:10.1038/ijo.2008.159

Butryn, M. L., Thomas, J. G., & Lowe, M. R. (2009). Reductions in internal disinhibition during weight loss predict better weight loss maintenance. *Obesity*, *17*(5), 1101-1103. doi:10.1038/oby.2008.646

Carnell, S., & Wardle, J. (2008). Appetite and adiposity in children: Evidence for a behavioral susceptibility theory of obesity. *American Journal of Clinical Nutrition*, *88*, 22-29. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/18614720

Carr, D., & Friedman, M. A. (2005). Is obesity stigmatizing? Body weight, perceived discrimination, and psychological well-being in the United States. *Journal of Health and Social Behavior*, *46*(3), 244-259. doi:10.1177/002214650504600303

Catenacci, V., & Wyatt, H. (2007). The role of physical activity in producing and maintaining weight loss. *Nature Clinical Practice Endocrinology & Metabolism, 3*, 518-529. doi:10.1038/ncpendmet0554

Church, T. S., Martin, C. K., Thompson, A. M., Earnest, C. P., Mikus, C. R., & Blair, S. N. (2009). Changes in weight, waist circumference and compensatory responses with different doses of exercise among sedentary, overweight postmenopausal women. *PLoS One, 4*(2), e4515. doi:10.1371/journal.pone.0004515

Contento, I. R., Zybert, P., & Williams, S. S. (2005). Relationship of cognitive restraint of eating and disinhibition to the quality of food choices of Latina women and their young children. *Preventive Medicine*, *40*(3), 326-336. doi:10.1016/j.ypmed.2004.06.008

Crow, S., Kendall, D., Praus, B., & Thuras, P. (2001). Binge eating and other psychopathology in patients with Type II diabetes mellitus. *International Journal of Eating Disorders, 30*(2), 222-226. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11449458

d'Amore, A., Massignan, C., Montera, P., Moles, A., De Lorenzo, A., & Scucchi, S. (2001). Relationship between dietary restraint, binge eating, and leptin in obese women. *International Journal of Obesity and Related Metabolic Disorders, 25*, 373-377. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11319635

de Zwaan, M., Mitchell, J. E., Howell, L., Monson, N., Swan-Kremeier, L., Crosby, R. D.,…Seim, H. C. (2003). Characteristics of morbidly obese patients before gastric bypass surgery. *Comprehensive Psychiatry, 44*(5), 428-434. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14505305

Deighton, K., Zahra, J. C., & Stensel, D. J. (2012). Appetite, energy intake and resting metabolic responses to 60 min treadmill running performed in a fasted versus a postprandial state. *Appetite, 58*(3), 946-954. doi:10.1016/j.appet.2012.02.041

DeLany, J. P. (2012). Measurement of energy expenditure. *Pediatric Blood & Cancer, 58*(1), 129-134. doi:10.1002/pbc.23369

Delparigi, A., Chen, K., Salbe, A. D., Reiman, E. M., & Tataranni, P. A. (2005). Sensory experience of food and obesity: A positron emission tomography study of the brain regions affected by tasting a liquid meal after a prolonged fast. *NeuroImage, 24*, 436-443. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15627585

Dhurandhar, E. J., Kaiser, K. A, Dawson, J. A, Alcorn, A. S., Keating, K. D., & Allison, D. B. (in press). Predicting adult weight change in the real world: A systematic review and meta-analysis accounting for compensatory changes in energy intake or expenditure. *International Journal of Obesity*. doi:10.1038/ijo.2014.184

Di Blasio, A., Ripari, P., Bucci, I., Di Donato, F., Izzicupo, P., D'Angelo, E.,…Napolitano, G. (2012). Walking training in post-menopause: Effects on both spontaneous physical

activity and training-induced body adaptations. *Menopause, 19*(1), 23-32. doi:10.1097/gme.0b013e318223e6b3

Donnelly, J. E., & Smith, B. K. (2005). Is exercise effective for weight loss with ad libitum diet? Energy balance, compensation, and gender differences. *Exercise and Sport Sciences Reviews, 33*(4), 169-174. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16239833

Donnelly, J. E., Hill, J. O., Jacobsen, D. J., Potteiger, J., Sullivan, D. K., Johnson, S. L.,…Washburn, R. A. (2003). Effects of a 16-month randomized controlled exercise trial on body weight and composition in young, overweight men and women: The Midwest Exercise Trial. *Archives of Internal Medicine, 163*(11), 1343-1350. doi:10.1001/archinte.163.11.1343

Donnelly, J. E., Honas, J. J., Smith, B. K., Mayo, M. S., Gibson, C. A., Sullivan, D. K.,…Washburn, R. A. (2013). Aerobic exercise alone results in clinically significant weight loss for men and women: Midwest Exercise Trial 2. *Obesity, 21*(3), E219-E228. doi:10.1002/oby.20145

Donnelly, J. E., Jacobsen, D. J., Heelan, K. S., Seip, R., & Smith, S. (2000). The effects of 18 months of intermittent vs. continuous exercise on aerobic capacity, body weight and composition, and metabolic fitness in previously sedentary, moderately obese females. *International Journal of Obesity Related Metabolic Disorders, 24*(5), 566-572. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10849577

Durrant, M. L., Royston, P. J., & Wloch, R. T. (1982). Effect of exercise on energy intake and eating patterns in lean and obese humans. *Physiology & Behavior, 29*(3), 449-454. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7178249

Epstein, L. H., Leddy, J. J., Temple, J. L., & Faith, M. S. (2007). Food reinforcement and eating: A multilevel analysis. *Psychological Bulletin, 133*(5), 884-906. doi:10.1037/0033-2909.133.5.884

Esliger, D. W., Copeland, J. L., Barnes, J. D., & Tremblay, M. S. (2005). Standardizing and optimizing the use of accelerometer data for free-living physical activity monitoring. *Journal of Physical Activity & Health, 2*(3), 366-383. Retrieved from http://journals.humankinetics.com/jpah-back-issues/jpahvolume2issue3july/standardizingandoptimizingtheuseofaccelerometerdataforfreelivingphysicalactivitymonitoring

Fairburn, C. G. & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders, 16*(4), 363-370. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7866415

Fairburn, C. G., & Brownell K. D. (2002). *Eating Disorders and Obesity: A Comprehensive Handbook.* New York, NY: Guilford.

Fairburn, C. G., & Cooper, Z. (1993). The eating disorder examination. In C. Fairburn & G. T. Wilson (Eds.), *Binge eating: Nature, assessment, and treatment* (12th ed.). New York, NY: Guilford Press.

Faul, F., Erdfelder, E., Lang, A.G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods, 39*, 175-191. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17695343

Finkelstein, E. A, Fiebelkorn, I. C., & Wang, G. (2004). State-level estimates of annual medical expenditures attributable to obesity. *Obesity Research*, *12*(1), 18-24. doi:10.1038/oby.2004.4

Finkelstein, E. A, Trogdon, J. G., Brown, D. S., Allaire, B. T., Dellea, P. S., & Kamal-Bahl, S. J. (2008). The lifetime medical cost burden of overweight and obesity: Implications for obesity prevention. *Obesity*, *16*(8), 1843-1848. doi:10.1038/oby.2008.290

Finlayson, G., Bryant, E., Blundell, J. E., & King, N. A. (2009). Acute compensatory eating following exercise is associated with implicit hedonic wanting for food. *Physiology & Behavior*, *97*, 62-67. doi:10.1016/j.physbeh.2009.02.002

Flint, A., Raben, A., Blundell, J. E., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *International Journal of Obesity and Related Metabolic Disorders*, *24*(1), 38-48. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10702749

French, S. A., Jeffery, R. W., Story, M., Breitlow, K. K., Baxter, J. S., Hannan, P., & Snyder, M. P. (2001). Pricing and promotion effects on low-fat vending snack purchases: The CHIPS Study. *American Journal of Public Health*, *91*(1), 112-117. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1446491/

Galgani, J. E., de Jonge, L., Most, M. M., Bray, G. A., & Smith, S. R. (2010). Effect of a 3-day high-fat feeding period on carbohydrate balance and ad libitum energy intake in humans. *International Journal of Obesity*, *34*(5), 886-891. doi:10.1038/ijo.2010.16

Greeno, C. G., & Wing, R. R. (1994). Stress-induced eating. *Psychological Bulletin*, *115*(3), 444-464. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8016287

Grilo, C. M., Masheb, R. M., Wilson, G. T., Gueorquieva, R., & White, M. A. (2011). Cognitive-behavioral therapy, behavioral weight loss, and sequential treatment for obese patients with binge-eating disorder: a randomized controlled trial. *Journal of Consulting and Clinical Psychology, 79*(5), 675-685. doi:10.1037/a0025049

Hartmann, A., Czaja, J., Rief, W., & Hilbert, A. (2010). Personality and psychopathology in children with and without loss of control over eating. *Comprehensive Psychiatry, 51*(6), 572-578. doi:10.1016/j.comppsych.2010.03.001

Haynes, C., Lee, M. D., & Yeomans, M. R. (2003). Interactive effects of stress, dietary restraint, and disinhibition on appetite. *Eating Behaviors, 4*(4), 369-383. doi:10.1016/j.eatbeh.2003.07.005

Hays, N. P., & Roberts, S. B. (2008). Aspects of eating behaviors "disinhibition" and "restraint" are related to weight gain and BMI in women. *Obesity, 16*(1), 52-58. doi:10.1038/oby.2007.12

Hays, N. P., Bathalon, G. P., McCrory, M. A, Roubenoff, R., Lipman, R., & Roberts, S. B. (2002). Eating behavior correlates of adult weight gain and obesity in healthy women aged 55-65 y. *The American Journal of Clinical Nutrition, 75*(3), 476-483. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11864852

Hill, J. O., Melby, C., Johnson, S. L., & Peters, J. C. (1995). Physical activity and energy requirements. *American Journal of Clinicla Nutrition, 62*(5), 1059S-1066S. Retrieved from http://ajcn.nutrition.org/content/62/5/1059S.abstract

Hollowell, R. P., Willis, L. H., Slentz, C. A., Topping, J. D., Bhakpar, M., & Kraus, W. E. (2009). Effects of exercise training amount on physical activity energy expenditure.

Medicine & Science in Sports & Exercise, 41(8), 1640-1644. doi:10.1249/MSS.0b013e31819c71a4

Hopkins, M., Blundell, J. E., & King, N. A. (2013). Individual variability in compensatory eating following acute exercise in overweight and obese women. *British Journal of Sports Medicine*, *48*(20), 1472-1476. doi:10.1136/bjsports-2012-091721

Hopkins, M., King, N. A, & Blundell, J. E. (2010). Acute and long-term effects of exercise on appetite control: Is there any benefit for weight control? *Current Opinion in Clinical Nutrition and Metabolic Care*, *13*(6), 635-640. doi:10.1097/MCO.0b013e32833e343b

Imbeault, P., Saint-Pierre, S., Alméras, N., & Tremblay, A. (1997). Acute effects of exercise on energy intake and feeding behaviour. *The British Journal of Nutrition, 77*(4), 511-521. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9155502

Jakicic, J. M., & Gallagher, K. I. (2003). Exercise considerations for the sedentary, overweight adult. *Exercise and Sport Sciences Reviews*, *31*(2), 91-95. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/12715973

Jakicic, J. M., Clark, K., Coleman, E., Donnelly, J. E., Foreyt, J., Melanson, E.,…Volpe, S. L. (2001). Appropriate intervention strategies for weight loss and prevention of weight regain for adults. *Medicine & Science in Sports & Exercise*, *33*(12), 2145-2156. doi:10.1097/00005768-200112000-00026

Jakicic, J. M., Marcus, M., Gallagher, K. I., Randall, C., Thomas, E., Goss, F. L., & Robertson, R. J. (2004). Evaluation of the SenseWear Pro Armband to assess energy expenditure during exercise. *Medicine & Science in Sports & Exercise*, (14), 897-904. doi:10.1249/01.MSS.0000126805.32659.43

Janssen, I., Fortier, A., Hudson, R., & Ross, R. (2002). Effects of an energy-restrictive diet with or without exercise on abdominal fat, inter-muscular fat, and metabolic risk factors in obese women. *Diabetes Care, 25*(3), 431-438. doi:http://www.ncbi.nlm.nih.gov/pubmed/11874926

Karlsson, J., Persson, L. O., Sjöström, L., & Sullivan, M. (2000). Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *International Journal of Obesity and Related Metabolic Disorders, 24*(12), 1715-1725. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11126230

Keim, N. L., Canty, D. J., Barbieri, T. F., & Wu, M. M. (1996). Effect of exercise and dietary restraint on energy intake of reduced-obese women. *Appetite, 26*(1), 55-70. doi:10.1006/appe.1996.0005

King, N. A. (1999). What processes are involved in the appetite response to moderate increases in exercise-induced energy expenditure? *Proceedings of the Nutrition Society, 58*(1). 107-113. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10343347

King, N. A., & Blundell, J. E. (1995). High-fat foods overcome the energy expenditure induced by high-intensity cycling or running. *European Journal of Clinical Nutrition, 49*(2), 114-123. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7743984

King, N. A., Lluch, A., Stubbs, R. J., & Blundell, J. E. (1997). High dose exercise does not increase hunger of energy intake in free living males. *European Journal of Clinical Nutrition, 51*(7), 478-483. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9234032

King, N. A., Snell, L., Smith, R. D., & Blundell, J. E. (1996). Effects of short-term exercise on appetite responses in unrestrained females. *European Journal of Clinical Nutrition, 50*(10), 663-667. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/8909932

King, N., Caudwell, P., Hopkins, M., Stubbs, J., Naslund, E., & Blundell, J. E. (2009). Dual-process action of exercise on appetite control: Increase in orexigenic drive but improvement in meal-induced satiety. *American Journal of Clinical Nutrition, 90,* 921-927. doi:10.3945/ajcn.2009.27706.1

Kissileff, H. R., Pi-Sunyer, F. X., Segal, K., Meltzer, S., & Foelsch, P. A. (1990). Acute effects of exercise on food intake in obese and nonobese women. *The American Journal of Clinical Nutrition, 52*(2), 240–245. Retrieved from http://ajcn.nutrition.org/content/52/2/240.abstract

Lähteenmäki, L., & Tuorila, H. (1995). Three-Factor Eating Questionnaire and the use and liking of sweet and fat among dieters. *Physiology & Behavior, 57*(1), 81-88. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7878129

Ledikwe, J. H., Blanck, H. M., Khan, L. K., Serdula, M. K., Seymour, J. D., Tohill, B. C., & Rolls, B. J. (2006). Dietary energy density is associated with energy intake and weight status in US adults. *American Journal of Clinical Nutrition, 83*(6), 1362-1368. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/16762948

Lee, S., Kuk, J. L., Davidson, L. E., Hudson, R., Kilpatrick, K., Graham, T. E., & Ross, R. (2005). Exercise without weight loss is an effective strategy for obesity reduction in obese individuals with and without Type 2 diabetes. *Journal of Applied Physiology, 99*(3), 1220-1225. doi:10.1152/japplphysiol.00053

Lluch, A., King, N. A., & Blundell, J. E. (2000). No energy compensation at the meal following exercise in dietary restrained and unrestrained women. *British Journal of Nutrition, 84*(2), 219-225. doi:http://dx.doi.org/10.1017/S0007114500001458

Luce, K. H., Crowther, J. H., & Pole, M. (2008). Eating Disorder Examination Questionnaire (EDE-Q): Norms for undergraduate women. *International Journal of Eating Disorders, 41*, 273-276. doi:10.1002/eat.20504

Manthou E., Gill, J. M., Wright, A., & Malkova, D. (2010). Behavioral compensatory adjustments to exercise training in overweight women. *Medicine & Science in Sports & Exercise, 42*(6), 1121-1128. doi:10.1249/MSS.0b013e3181c524b7

Martins, C., Kulseng, B., King, N. A, Holst, J. J., & Blundell, J. E. (2010). The effects of exercise-induced weight loss on appetite-related peptides and motivation to eat. *The Journal of Clinical Endocrinology and Metabolism, 95*(4), 1609-1616. doi:10.1210/jc.2009-2082

Martins, C., Morgan, L. M., Bloom, S. R., & Robertson, M. D. (2007). Effects of exercise on gut peptides, energy intake and appetite. *The Journal of Endocrinology, 193*(2), 251-258. doi:10.1677/JOE-06-0030

Martins, C., Morgan, L., & Truby, H. (2008). A review of the effects of exercise on appetite regulation: An obesity perspective. *International Journal of Obesity, 32*(9), 1337-1347. doi:10.1038/ijo.2008.98

Martins, C., Robertson, M. D., & Morgan, L. M. (2010). Impact of restraint and disinhibition on PYY plasma levels and subjective feelings of appetite. *Appetite, 55*(2), 208-213. doi:10.1016/j.appet.2010.05.091

Mathes, W., Brownley, K., Mo, X., & Bulik, C. (2009). The biology of binge eating. *Appetite, 52*(3), 545-553. doi:10.1016/j.appet.2009.03.005

McLaughlin, R., Malkova, D., & Nimmo, M. A. (2006). Spontaneous activity responses to exercise in males and females. *European Journal of Clinical Nutrition, 60*(9), 1055-1061. doi:10.1038/sj.ejcn.1602417

Meijer, E. P., Westerterp, K. R., & Verstappen, F. T. (1999). Effect of exercise training on total daily physical activity in elderly humans. *European Journal of Applied Physiology and Occupational Physiology, 80*(1), 16-21. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10367718

Muller-Riemenschneider, F., Reinhold, T., Nocon, M., & Willich, S. N. (2008). Long-term effectiveness of interventions promoting physical activity: A systematic review. *Preventive Medicine, 47*, 354-368. doi:10.1016/j.ypmed.2008.07.006

Ouwens, M. A., Van Strien, T., & Van der Staak, C. P. F. (2003). Absence of a disinhibition effect of alcohol on food consumption. *Eating Behaviors, 4*(4), 323-332. doi:10.1016/S1471-0153(03)00033-3

Pagoto, S., Bodenlos, J. S., Kantor, L., Gitkind, M., Curtin, C., & Ma, Y. (2007). Association of major depression and binge eating disorder with weight loss in a clinical setting. *Obesity, 15*(11), 2557-2559. doi:10.1038/oby.2007.307

Pannacciulli, N., Salbe, A. D., Ortega, E., Venti, C. A., Bogardus, C., & Krakoff, J. (2007). The 24-h carbohydrate oxidation rate in a human respiratory chamber predicts ad libitum food intake. *American Journal of Clinical Nutrition, 86*(3), 625-632. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/17823426

Penesova, A., Venti, C. A., Bunt, J. C., Bonfiglio, S. M., Votruba, S. B., & Krakoff, J. (2011) Short-term isocaloric manipulation of carbohydrate intake: Effect on subsequent ad libitum energy intake. *European Journal of Nutrition, 50*(6), 1-9. doi:10.1007/s00394-010-0152-5

Pomerleau, M., Imbeault, P., Parker, T., & Doucet, E. (2004). Effects of exercise intensity on food intake and appetite in women. *The American Journal of Clinical Nutrition, 80*(5), 1230-1236. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15531670

Pritchard, J. E., Nowson, C. A., & Wark, J. D. (1997). A worksite program for overweight middle-aged men achieves lesser weight loss with exercise than with dietary change. *Journal of the American Dietic Assocation, 97*(1), 37-42. doi:http://dx.doi.org/10.1016/S0002-8223(97)00015-1

Puhl, R., & Brownell, K. D. (2001). Bias, discrimination, and obesity. *Obesity Research*, *9*(12), 788-805. doi:10.1038/oby.2001.108

Ricca, V., Castellini, G., Lo Sauro, C., Ravaldi, C., Lapi, F., Mannucci, E.,…Faravelli, C. (2009). Correlations between binge eating and emotional eating in a sample of overweight adults. *Appetite, 53*(3), 418-421. doi:10.1016/j.appet.2009.07.008

Rosenkilde, M., Auerbach, P. L., Reichkendler, M. H., Ploug, T., Stallknecht, B. M., & Sjodin, A. (2012). Body fat loss and compensatory mechanisms in response to different doses of aerobic exercise—A randomized controlled trial in overweight sedentary males. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology*, 303(6), R571-R579. doi:10.1152/ajpregu.00141.2012

Ross, R., Janssen, I., Dawson, J., Kungl, A. M., Kuk, J. L., Wong, S. L.,…Hudson, R. (2004). Exercise-induced reduction in obesity and insulin resistance in women: A randomized controlled trial. *Obesity Research, 12*(5), 789-798. doi:10.1038/oby.2004.95

Savage, J. S., Hoffman, L., & Birch, L. L. (2009). Dieting, restraint, and disinhibition predict women's weight change over 6 y. *American Journal of Clinical Nutrition*, *90*, 33-40. doi:10.3945/ajcn.2008.26558.Am

Scagliusi, F. B., Ferriolli, E., Pfrimer, K., Laureano, C., Cunha, C. S. F., Gualano, B.,…Lancha Jr, A. H. (2009). Characteristics of women who frequently under report their energy intake: A doubly labelled water study. *European Journal of Clinical Nutrition*, *63*, 1192-1199. doi:10.1038/ejcn.2009.54

Schubert, M. M., Desbrow, B., Sabapathy, S., & Leveritt, M. (2013). Acute exercise and subsequent energy intake. A meta-analysis. *Appetite*, *63*, 92-104. doi:10.1016/j.appet.2012.12.010

Snyder, K. A., Donnelly, J. E., Jabobsen, D. J., Hertner, G., & Jakicic, J. M. (1997). The effects of long-term, moderate intensity, intermittent exercise on aerobic capacity, body composition, blood lipids, insulin and glucose in overweight females. *International Journal of Obesity and Related Metabolic Disorders*, *21*(12), 1180-1189. doi:10.1038/sj.ijo.0800533

Stunkard, A., & Messick, S. (1985). The Three-Factor Eating Questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research, 29*(1), 71-83. Retrieved from http://www.sciencedirect.com/science/article/pii/0022399985900108

Thomas, D. M., Bouchard, C., Church, T., Slentz, C., Kraus, W. E., Redman, L. M.,…Heymsfield, S. B. (2012). Why do individuals not lose more weight from an

exercise intervention at a defined dose? An energy balance analysis. *Obesity Reviews, 13*(10), 835-847. doi: 10.1111/j.1467-789X.2012.01012.x

Thompson, D. A., Wolfe, L. A., & Eikelboom, R. (1988). Acute effects of exercise intensity on appetite in young men. *Medicine & Science in Sports & Exercise, 20*(3), 222–227. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/3386499

Tsofliou, F., Pitsiladis, Y. P., Malkova, D., Wallace, A. M., & Lean, M. (2003). Moderate physical activity permits acute coupling between serum leptin and appetite-satiety measures in obese women. *International Journal of Obesity, 27,* 1332-1339. doi:10.1038/sj.ijo.0802406

Unick, J. L., Otto, A. D., Goodpaster, B. H., Helsel, D. L., Pellegrini, C. A., & Jakicic, J. M. (2010). Acute effect of walking on energy intake in overweight/obese women. *Appetite, 55*(3), 413-419. doi:10.1016/j.appet.2010.07.012

Van Strien, T. (2000). Ice-cream consumption, tendency toward overeating, and personality. *International Journal of Eating Disorders*, *28*(4), 460-464. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/11054795

Visona, C., & George, V. A. (2002). Impact of dieting status and dietary restraint on postexercise energy intake in overweight women. *Obesity Research*, *10*(12), 1251-1258. doi:10.1038/oby.2002.170

Waxman, S. E. (2009). A systematic review of impulsivity in eating disorders. *European Eating Disorders Review*, *17*(6), 408-425. doi:10.1002/erv.952

Wei, M., Kampert, J. B., Barlow, C. E., Nichaman, M. Z., Gibbons, L. W., Paffenbarger, R. S. Jr, & Blair, S. N. (1999) Relationship between low cardiorespiratory fitness and mortality in

normal-weight, overweight, and obese men. *JAMA, 282,* 1547-1553. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/10546694

Welborn, T. A., & Dhaliwal, S. S. (2007). Preferred clinical measures of central obesity for predicting mortality. *European Journal of Clinical Nutrition, 61*(12), 1373-1379. doi:10.1038/sj.ejcn.1602656

Westenhoefer, J., Broeckmann, P., Munch, A. K., & Pudel, V. (1994). Cognitive control of eating behavior and the disinhibition effect. *Appetite, 23*(1), 27-41. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/7826055

Westerterp, K. R., Meijer, G. A.,Janssen, E. M., Saris,W. H., & TenHoor, F. (1992). Long-term effect of physical activity on energy balance and body composition. *British. Journal of Nutrition, 68*(1), 21-30. doi:http://dx.doi.org/10.1079/BJN19920063

Westerterp-Plantenga, M. S., Verwegen, C. R., Ijedema, M. J., Wijckmans, N. E., & Saris, W. H. (1997). Acute effects of exercise or sauna on appetite in obese and nonobese men. *Physiology & Behavior, 62*(6), 1345-1354. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9383124

Whiteside, S., & Lynam, D. (2001). The Five Factor Model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences, 30*(4), 669-689. doi:10.1016/S0191-8869(00)00064-7

Wilson, G. T., Wilfley, D. E., Agras, W. S., & Bryson, S. W. (2010). Psychological treatments of binge eating disorder. *Archives of General Psychiatry, 67*(1), 94-101. doi:10.1001/archgenpsychiatry.2009.170

Withrow, D., & Alter, D. A. (2011). The economic burden of obesity worldwide: A systematic review of the direct costs of obesity. *Obesity Reviews, 12*(2), 131-141. doi:10.1111/j.1467-789X.2009.00712.x

Yeomans, M. R., Mobini, S., Bertenshaw, E. J., & Gould, N. J. (2009). Acquired liking for sweet-paired odours is related to the disinhibition but not restraint factor from the Three Factor Eating Questionnaire. *Physiology & Behavior, 96*(2), 244-252. doi:10.1016/j.physbeh.2008.10.001

Yeomans, M. R., Tovey, H. M., Tinley, E. M., & Haynes, C. J. (2004). Effects of manipulated palatability on appetite depend on restraint and disinhibition scores from the Three-Factor Eating Questionnaire. *International Journal of Obesity and Related Metabolic Disorders, 28*(1), 144-151. doi:10.1038/sj.ijo.0802483