Asymmetric Brain Activation: Relation to Binge Eating in Overweight Subjects

A Thesis

Submitted to the Faculty

of

Drexel University

by

Christopher N. Ochner, M.A., M.S.

in partial fulfillment of the

requirements for the degree

of

Doctor of Philosophy

August 2006

Dedications

This dissertation is dedicated to my family whose unconditional love and support continues to remind me every day of how blessed I am. I owe my success in this and all future endeavors to my parents who taught me never to do the bare minimum. I blame the length of this paper on you. Finally, I dedicate this work to Dr. Robert Scott Ochner who never got to see me grow up. I hope you would be proud of me, Bobby.

Acknowledgements

Several individuals spent many hours and dollars in bringing this research project to fruition. I would first like to thank Dr. Michael Lowe, for not only making this research possible, but also facilitating my shift from behavioral inhibition to behavioral activation in research academics. Dr. John Kounios was instrumental in this research project from beginning to end. He generously provided time, EEG lab equipment, financial support, and staff; without any of which, this research would not have been possible. Deborah Green was responsible for the collection and reduction of all EEG data and was tireless in her efforts to overcome numerous difficulties through the process. Jason van Steenburgh played an integral role in participant recruitment and nearly doubled the number of successful participants in the final months of recruitment. Michelle Maier played several roles through the course of this research, and handled an unending stream of requests with extraordinary professionalism. Finally, this research project could not have been completed without support and understanding from family, friends, and God. With the sincerest of gratitude, I thank you all.

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Abstract

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Right-sided frontal asymmetry has been related to negative affect and an "avoidant" personality type. Research has demonstrated a relationship between right-sided frontal asymmetry and restrained eating in normal weight individuals. It has previously been shown that normal weight restrained eaters display similar frequencies of negative affect and patterns of eating as overweight binge eaters. This study tested the hypothesis that overweight individuals high in binge eating would exhibit greater right-sided frontal asymmetry than would overweight individuals low in binge eating. 30 overweight participants were recruited from a weight maintenance, or binge eating, study. Participants were assessed using the Binge Eating Scale (BES), Mood and Anxiety Symptom Questionnaire (MASQ), Positive and Negative Affect Schedule (PANAS), Power of Food Scale (PFS) and the Three Factor Eating Questionnaire (TFEQ). Asymmetrical brain activation in the frontal and parietal cortices was assessed using resting electroencephalogram (EEG) recordings.

Participants were grouped according to BES scores (low binge vs. high binge). ANOVAs were used to examine group differences in asymmetry by region (frontal vs. parietal) with and without controlling for state affect. Pearson correlations were used to examine the relationships between all self-report measures and asymmetrical activation. Results did not support the main hypothesis, indicating that binge eating was not related to asymmetry in this sample. High bingers scored significantly higher in MASQ Anxious

Arousal, however, state affect was not related to asymmetry. Post hoc analyses revealed that PFS scores were negatively correlated with positive affect, positively correlated with left-sided frontal asymmetry, and positively correlated with right-sided parietal asymmetry. An inverse correlation was also found between frontal and parietal asymmetry. Post hoc results encourage further investigation into the affect model of frontal asymmetry. It is suggested that the BIS-BAS model of asymmetry may better account for frontal asymmetry results, however data collected in this study did not allow for an evaluation of construct validity of differing theories. Future research is needed to unify asymmetrical activation models and further explore relationships between appetitive responsiveness, frontal asymmetry, and parietal asymmetry.

CHAPTER 1: INTRODUCTION

Excess body weight has been deemed a nationwide epidemic, as over 64% of Americans were overweight or obese according to the Center for Disease Control and Prevention's 1999-2000 National Health and Nutrition Examination Survey. These numbers have been growing at a disquieting rate (National Institutes of Health, 1998; Mokdad, Bowman, Ford, Vinicor, Marks, Koplan, 2001). Obesity is a risk factor for numerous serious health conditions such as cardiovascular disease, stroke, hypertension, gallbladder disease, diabetes, and several types of cancer (Pi-Sunyer, 1993; WHO Consultation on Obesity, 2000; Mokdad, Bowman, Ford, Vinicor, Marks & Koplan, 2001). Additionally, it is second only to tobacco use as the leading cause of preventable death in the United States (U.S. Department of Health and Human Services, 1990), and is soon expected to become the leading cause (Grundy, 1998; Center for Disease Control and Prevention, 2000; Sibbald, 2002; Mokdad, Marks, Stroup, & Gerberding, 2004). Worldwide, the total cost attributable to obesity and its negative health consequences was estimated to represent 2% to 7% of health expenditures in the year 2000. (WHO Consultation on Obesity, 2000). According to a study of national costs attributed to both overweight (BMI 25 kg/m^2 to 29.9 kg/m^2) and obesity (BMI $\geq$ 30 kg/m^2), medical expenses accounted for 9.1 percent of total U.S. medical expenditures in 1998 (Finkelstein, Fiebelkorn, and Wang, 2003). Current data indicates that the yearly cost of overweight and obesity in the United States has reached $117 billion (National Institutes of Diabetes & Digestive & Kidney Diseases, 2004).

The increasing prevalence of overweight and obesity in the United States requires a reevaluation of what 'normal weight' implies. If 'normal' is to be defined as 'statistically more likely', than it has now become normal to be overweight. In the year 2000, the average U.S. man (5 feet 9 inches tall) was approximately 31 pounds overweight and the average woman (5 feet 4 inches tall) was approximately 24 pounds overweight (Centers for Disease Control and Prevention, 2000). For the purposes of this study, however, the term 'normal' was used to designate persons with a body mass index (BMI) between 18.5 kg/m^2 and 24.9 kg/m^2. According to current US dietary guidelines, persons with a BMI between 25.0 kg/m^2 and 29.9 kg/m^2 are considered 'overweight', and those with BMIs of 30.0 kg/m^2 or more are considered 'severely overweight' or 'obese'.

Research indicates that modest weight losses of only 5 to 10% of body weight are enough to produce medically significant reductions in comorbidities associated with obesity (Blackburn, 1995). Most traditional CBT/lifestyle change diets have consistently been shown to produce this degree of weight loss (Foster, Wadden, Kendall, Stunkard & Vogt, 1996; Wadden & Sarwer, 1996; Jeffrey, Drewnowski, Epstein, Stunkard, Wilson, Wing & Hill, 2000). The problem, however, is that 1/3 to 1/2 of all lost weight is typically regained at 1-year follow up (FU), and approximately 90% is typically regained at 5-year FU (Wadden & Sarwer, 1996).

Such disappointing weight loss maintenance results are witnessed in virtually every clinical weight loss trial, even in trials specifically aimed at improving weight maintenance following diet (National Task Force on Prevention and Treatment of Obesity, 1994; Sarlio-Lahteenkorva, Rissanen, & Kaprio, 2000; Borg, Kukkonen-

Harjula, Fogelholm & Pasanen, 2002). The relative impotence of traditional lifestyle change techniques in reaching acceptable levels of weight maintenance has raised questions regarding conventional methods for studying and conceptualizing obesity, as well as some of the assumptions upon which traditional obesity treatments have been based. Namely, there has historically been a concentration on an obese vs. normal weight distinction. Though studying differences between these two populations holds a certain amount of intuitive appeal, traditional attempts to distinguish between obese and normal weight persons has, unfortunately, yielded as many questions as it has answers.

Obese vs. Normal Weight Distinction

In past decades, the majority of obesity research has focused on the differences between the overweight or obese population and the normal weight population (Rodin, 1975; Rosenthal & Marx, 1978; Fitzgibbon & Kirschenbaum, 1990; Wing, Blair, Epstein, & McDermott, 1990; Cilli, De Rosa, Pandolfi, Vacca, Cugini, Ceni & Bella, 2003). The assumption has been long held that the obese population displays relatively few individual differences and, on average, shows poorer psychological functioning as compared to nonobese individuals in the general population (Rodin, 1981; Kirschenbaum, 1988). Numerous studies have addressed this assumption. Moore, Stunkard, & Srole (1962) examined 1660 people and found that obese individuals scored higher than nonobese persons on only three of nine measures of psychological functioning (immaturity, suspiciousness, and rigidity), and that the differences between groups on these measures were so small that they were judged to be clinically insignificant. In 1983, Stewart & Brook found only small differences between nearly

6000 obese and normal weight subjects. Similar results indicating few or no differences in psychological status between obese and nonobese persons in the general population have been reported by several other authors in the United States (Friedman & Brownell, 1995; Stunkard & Wadden 1992; Wadden & Stunkard, 1985; Wadden, Foster, Stunkard, Linowitz, 1989) and Europe (Crisp & McGuiness, 1976; Hallstrom & Noppa, 1981; Larsson, 1978).

One study that did find a correlation between obesity and depression was that of Carpenter, Hasin, Allison & Faith (2000). In this study, the authors used a structured interview to establish a diagnosis of major depression in a nationally representative sample using criteria comparable to those in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV; American Psychiatric Association, 1994). This study was based on the 1992 National Longitudinal Alcohol Epidemiologic Survey (NLAES), which involved face-to-face interviews of 42,862 household residents 18 years or older in the contiguous United States, including the District of Columbia. 2776 respondents were excluded because they were either of other racial backgrounds (this study focused on whites and African Americans) or they were missing height or weight data. This resulted in a final sample size of 40,086. No significant differences were noted between the sample used for the analyses in this study and the complete sample on demographic (excluding race) or measures of psychopathology. Covariates included age, income and education, disease status, and drug and alcohol use. Within the obese population, striking gender differences were revealed. Obese women were found to be 37% more likely to have experienced a depressive episode in the past year, as well as reporting suicidal ideation and attempts, than were average weight women. (Note:

'obese' in this study was defined as having a BMI ≥ 30.0 kg/m^2, while 'normal' weight was defined as having a BMI between 20.8 and 29.9 kg/m^2). Obesity in men, however, was associated with a significantly *reduced* risk of major depression and suicide attempts. For men, being underweight (defined as having a BMI < 20.8 kg/m^2), not obese, was associated with an increased risk of depression, suicidal ideation, and suicide attempts.

Though the association between obesity and an increased risk of depressive symptoms in women is certainly cause for concern, Wadden et al. (2002) warn against inadvertently using such data to support prejudices against the obese population that may not necessarily be true. As these authors point out, Kessler, McGonagle, Zhao, Nelson, Hughes, Eshleman, Wittchen & Kendler (1994) reported that approximately 10% of normal weight women reported depression in the prior year. A 37% increase in the obese population would result in approximately 13.7% of obese women reporting depression in the prior year. Compared to an average of 10%, 13.7% indicates a significant increase in prevalence of depression to be sure, but not enough to base treatment on the assumption that the obese population as psychologically unhealthy relative to normal weight individuals. An additional point to note from the Carpenter et al. (2000) study is that no control for level of physical activity was able to be factored into the results, which may have explained some amount of the variance in levels of depression between individuals of varying body weights.

Internal Eating vs. External Eating

There is a separate area of research that has attempted to differentiate obese and normal weight individuals on dimensions of personality, specifically, responsiveness to internal and external cues. Rodin (1981) addresses an assumption that had been widely held in research on the development of obesity. Conventional theory has centered around a distinction between internal and external cues or motivations for eating behavior. Internal cues are considered to be physiological feelings of hunger or caloric deprivation, or feelings of 'satiety' or fullness. External cues are those that are present in the environment that prompt eating, even in the absence of caloric deprivation (e.g., commercials displaying highly palatable dishes, smells of foods, desire to sooth negative emotions, etc). This theory of the development of overeating habits and obesity was developed in the late 1960s when Nisbett (1968a; 1968b), Schachter & Gross (1968), and colleagues reported on an influential series of studies suggesting that the eating behavior of their overweight participants was mainly influenced by the taste and sight of food, and the number of highly palatable food cues present. These studies implied a dichotomy between internal and external control of feeding; suggesting that the eating behavior of normal weight people was responsive to internal stimuli and that, in contrast, the eating behavior of overweight people was unresponsive to internal stimuli and, instead, was primarily controlled by external cues (Schachter, Goldman, & Gordon, 1968). This internal-external distinction gained considerable attention and served to fuel further investigation into distinctions between the obese and normal weight populations that continues today.

Despite the popularity of the internal/external eating distinction in relation to body weight, many experiments have failed to demonstrate that overweight individuals are more responsive to external food and nonfood cues than are their normal weight counterparts (e.g., Goldman, 1969; Nisbett & Storms, 1975; Nisbett & Temoshok, 1976; Shaw, 1973). Additionally, several other studies failed to show reliable overweight vs. normal weight differences consistently from subject population to subject population, or even from study to study within the same population (Rodin, Moskowitz & Bray, 1976; Rodin, Slochower & Fleming, 1977). These, and further studies, found that in every weight category there were people who were externally responsive and people who were not. The same was true of internal responsiveness. Moreover, across all weight groups, degree of overweight was not strongly related to the degree of external or internal responsiveness demonstrated. In fact, even at extreme degrees of obesity, some individuals showed very little responsiveness to external cues. As Rodin (1981) described, the evidence is quite clear that all overweight individuals are not externally responsive nor are all normal weight individuals internally sensitive.

Several alternative explanations for the distinction between eating habits of obese and normal weight individuals have been posited. Nisbett (1972) suggested that the association between overweight and cue responsiveness was not the primary mechanism of action, but that obese people ingest calories, relative to their energy (caloric) expenditure, up to a certain "set point" of body weight. He argued that obese individuals may actually exist in a chronic state of energy deficit and are genuinely hungry because they attempt to hold their weight below its biologically dictated set point by dieting. This theory rapidly gained popularity amongst the research community, and was subsequently

tested in several empirical studies. In weight loss studies, however, none were able to demonstrate participants significantly increasing their responsiveness to environmental cues after weight loss, as the Nisbett hypothesis would require (Rodin et al., 1977; Rodin, 1981).

A different effort to explain the poor results of attempts to replicate the relationship between obesity and externality came from the studies of Herman and Polivy and colleagues (Herman & Mack, 1975; Herman, Polivy & Silver, 1979; Hibscher & Herman, 1977; Polivy & Herman, 1985). These authors suggested, like Nisbett, that the proposed associations between obesity and external eating were a result, not of a 'hyperresponsiveness' to the food environment, but of obese persons' chronic dieting. They propose that dieting promotes the adoption of a cognitively-regulated eating style to aid in the physiological defense of excess body weight, and that this cognitive regulation of eating leads to the behavioral reaction of overeating (Polivy & Herman, 1985). These cognitive attempts to control eating have been dubbed "dietary restraint". According to this theory, by replacing physiological regulatory controls of eating, obese individuals practice this dietary restraint that, in turn, leads to disinhibited eating and thus, further weight gain. This theory is based, in large part, on differences in the eating patterns between normal weight individuals who restrain their eating and those that do not restrain their eating. In such studies, participants tasted ice cream after drinking no, one, or two high-calorie milkshakes. This 'preload' has been shown to be anxiety-provoking in normal weight individuals who restrain their eating (Herman & Mack, 1975), and is referred to as a 'disinhibiting' stimulus. That is, normal weight restrained eaters (chronic dieters) have been shown to eat significantly more ice cream- disinhibiting their eating-

in response to preload whereas normal weight unrestrained eaters consume significantly less (regulate or inhibit their eating) following preload (Ruderman, 1986).

While this theory gained considerable attention in the literature, several criticisms of this theory began to arise in the 1980's. Numerous studies testing the assertions upon which restraint theory are based and have found counterregulatory eating in unrestrained individuals and failure to counterregulate eating in restrained individuals (Lowe & Maycock, 1988; Jansen, Oosterlaan, Merckelbach & van den Hout, 1988; Lowe et al., 1991). In addition to this evidence inconsistent with restraint theory, related studies of traditional restraint theory have found large amounts of variance in the eating behavior of restrained eaters, differential eating patterns of restrained eaters and dieters, and variation between the Restraint Scale (Herman & Polivy, 1980; traditionally used to measure dietary restraint in this theory) and other seemingly similar measure of the constructs presumed to be associated with dietary restraint (e.g., Jansen, Merckelback, Oosterlaan, Tuiten & van den Hout, 1988; Lowe, Whitlow & Bellwoar, 1991; Jeffery, Adlis & Forster, 1991; Kleinfield & Lowe, 1991; Eldredge, 1993). This and other evidence illustrated by Lowe (1993) demonstrates that the tendency towards weight gain described in restraint theory cannot be fully accounted for by frequency of dieting and overeating, current dieting, or weight suppression (significant diet-induced weight lost sustained for > 1 year) as restraint theory would suggest. Lowe (1993; 1994) asserts that eating behaviors typically described by restraint theory may not be a consequence of dietary restraint, but may reflect a pre-existing tendency towards overconsumption that may lead to excess body weight or obesity.

Taken together, this evidence suggests that dietary restraint itself may not explain the causal factor leading to weight gain, but describes a particular reaction to the desire to eat. That is, the feelings of deprivation trigger attempts to control intake; dietary restraint. Though dietary restraint may be related to overeating and weight gain, this theory does not adequately explain the mechanism responsible for the desire to eat, or overeat in the case of obese individuals. It may remain that persons particularly sensitive to external eating cues, in an environment saturated with eating triggers (Horgen & Brownell, 2002), practice dietary restraint in an attempt to control their intake when they feel compelled to eat by external cues. Rodin (1981) maintains that it is the external responsiveness that leads to restrained eating, disinhibited eating, or altering periods of the two which can be witnessed in both normal and overweight persons.

In keeping with the theory that dietary restraint is not itself a mechanism of action, Lowe (1993; 1994) and colleagues (Lowe et al., 1991; Lowe & Timko, 2004) have theorized that overweight individuals may be predisposed to weight gain; potentially due to a hypersensitivity to external cues to eat. These authors have theorized that obese individuals may feel chronically compelled to overeat, and may use dietary restraint as an (ineffective) method to ward off subsequent excess body weight. According to this theory, dietary restraint is viewed as more of a proxy between a predisposition toward weight gain (i.e., a hypersensitivity to external cues) and obesity. That is, restraint may not be the cause of weight gain, but an ineffective attempt to regulate a pre-established disposition towards overconsumption and, thus, weight gain. Support for this theory was obtained by Hill, Weaver & Blundell (1991). In a correlational analysis of 206 women, these authors showed food craving to be only

weakly related to dietary restraint, but highly and significantly correlated with external eating, emotional eating and susceptibility to hunger. Similarly, Lowe (in preparation) developed a scale to better assess "appetitive hyperresponsiveness" and has found evidence that this hypersensitivity to external cues may actually underlie both disinhibited eating and other phenomena (e.g., salivary output, distractibility) traditionally associated with restrained eating.

Current research suggests that restraint, therefore, is actually a *consequence* of passive overconsumption in an obesigenic or 'toxic' environment full of salient cues potentially triggering external eating (Lowe, 2002; 2003; Lowe & Timko, 2004; Blundell, 2002). This line of reasoning suggests that a hyperresponsiveness to external eating cues may be the primary mechanism of action causing individuals to either attempt to restrain their eating or to overeat. Thus, the long-term ineffectiveness of dieting may suggest that attempts to regulate eating on the part of overweight and obese individuals are not sufficient to control motivations to eat in the presence of the 'obesigenic' environment in which they live (Lowe, 2003). Additionally, it has been suggested that individuals who exercise dietary restraint and attempt weight loss diets may be exactly those individuals whose appetitive systems are most sensitive to the food-laden environment in which they live (Lowe & Levine, in preparation). That is, an explanation for the inconsistency of the internal/external distinction may be the fact that many normal weight individuals are just as restrained as overweight individuals and such dietary restraint may stem from their hyperresponsivity to the food environment (external responsiveness).

The idea that external responsiveness is not caused by obesity, deprivation, or restraint, but is a primary mechanism of action, and therefore could lead to overeating

and contribute to weight gain given a plentiful food environment is not new. In 1976, Rodin and Slochower conducted a study to test exactly this hypothesis. These authors observed children at an 8-week summer camp for normal weight girls where highly palatable (and calorically dense) food was abundantly available. The authors in this study gave all girls a pretest at the beginning of the summer to determine extent of individual responsiveness to external cues. Results indicated that the girls who were hyperresponsive to all kinds of external cues (according to pretest) were those who gained the most weight when exposed to a major change in their food-relevant environment. The significance of this result lies in the fact that these subjects were normal weight children with no prior history of overweight. Also of interest in this study was the fact that one-third of the participants reached their highest weights before the eighth week and then lost a significant amount of weight before the end of camp. This suggests that other factors became more important than external responsiveness in influencing the final levels of body weight attained. These variables could be physiological (e.g., metabolic or adipose tissue parameters) or psychological (e.g., mood or affect). Rodin (1981) sums up preceding points in saying; "What all this means is that it is no longer very useful, or valid, to conduct research that simply divides individuals on the basis of their degree of overweight and then looks for the external- or internal-sensitivity characteristics on which they differ" (p. 367).

Inherent within traditional attempts to distinguish between overweight and normal weight populations has resided the assumption that the obese population is suffering from the same disorder and represents a relatively homogeneous group. The differences between normal weight and overweight individuals are certainly of great import within

the field as they enable researchers to determine which differences are not only meaningful between overweight and normal weight persons, but which differences are malleable and, thus, potentially useful in the treatment and/or prevention of obesity. In concentrating on such differences, however, it becomes all too easy to study and treat each group with the assumption of homogeneity. There is substantial literature asserting that there is often as much variability demonstrated within the overweight population itself as is seen between overweight and normal weight groups. Due to the fact that most treatment programs and research protocols treat obesity as a uni-dimensional disorder, current treatment may reflect the assumption that one treatment or one protocol can be applied to the entire obese population. As the relative lack of success of traditional lifestyle change programs to curb the obesity epidemic might testify, valuable information may be gleaned through the examination of additional individual differences within the overweight and obese population itself.

Assumption of Homogeneity

It has been suggested that a significant portion of the explanation for such disappointing results of traditional weight loss methods in the long term may be due to the fact that treatment programs often reflect the assumption that the obese population represents a homogeneous group, containing little variability in personality, mental health, or sources of the obesity problem with which they struggle (Kirschenbaum, 1988; Fitzgibbon & Kirschenbaum, 1990). Traditional lifestyle change diets may not take into account the large amount of variance, not only in the factors contributing to obesity, but differences in eating habits, stressors, comorbidities, reactions to food (restraint,

disinhibition, etc.), and emotional connections to food within the obese population itself. Wadden & Stunkard (1985) and Wadden, Womble, Stunkard, and Anderson (2002) address the assumption of homogeneity, and warn against stereotyping the obese population; "Such beliefs are similar previous erroneous assertions that obese individuals have a specific personality style. Personality is as diverse in obese individuals as it is in those of average weight." (p. 148).

In the previously described Carpenter et al. (2000) study of incidence of depression, suicidal ideation, and suicide attempts in relation to body weight, differences were found between overweight and average weight individuals, however, more variability was found within the overweight population than within the average weight population on nearly all dimensions measured. Among overweight women, more variance was seen in percent of past-year depression, suicidal ideation, and suicide attempts as compared to their average weight counterparts. Among overweight males, more variance was seen in the percent of suicidal ideation and suicide attempts in the previous year than was witnessed in the average weight group. (Note: variance in the percent incidence of depression in the past year was equal for the overweight and normal weight males).

In one of the most thorough studies of psychopathology in relation to body weight, Fitzgibbon, Stolley, and Kirshenbaum (1993) examined the psychological status of obese persons who did (n = 59) or did not (n = 59) seek weight reduction, as well as that of nonobese individuals (n = 59) who were not seeking medical care. The authors in this study began with an initial subject pool of 547 persons. Groups were then matched

according to percentage overweight, age, race, and education, yielding a total N of 177. Results indicated that obese treatment-seeking patients reported significantly greater symptoms of psychological distress and binge eating than did either the obese or nonobese individuals who did not seek treatment. The two obese groups, however, did not differ in body weight or dieting history. Another major finding in this study was that obese and non-obese non treatment seekers did not differ from one another. That is, no differences in psychological distress or binge eating were found between obese and normal weight persons who were not seeking treatment. Though differences were observed between obese treatment seekers and normal weight individuals not seeking treatment, body weight was not the causal factor in the increased psychological distress observed in the obese treatment seeking group. Increased anxiety and depression are routinely observed in patients who seek medical care (Swenson, Pearson & Osborne, 1973). Additionally, emotional distress (i.e., symptoms of depression or anxiety) is likely to be one of the factors that prompts people to seek professional assistance, regardless of body weight (Wadden et al., 2002).

Further understanding of the multiple mechanisms involved in the regulation of eating and body weight is needed before the specific sets of characteristics and array of causal factors that relate to overweight and obesity can be identified for the purpose of treatment and prevention. The present state of the literature demands a shift of orientation to understanding the etiology of obesity or, indeed, the several 'obesities' (to be discussed further in following sections) that all have excess body weight as their common observable characteristic (Guy-Grand, 2003). It has not been possible to divide samples sensibly or productively without such information (Rodin, 1981). "It may never

be possible to find the 'magic bullet,' since obesity is not a single syndrome, has no single cause, and therefore probably does not have a single cure" (p370).

Individual Differences within the Obese Population

A substantial amount of research in this area has uncovered significant individual differences within this population in both physiological and psychological factors. Examples of individual differences in physiological variables within the obese population include gender differences in levels of and reaction to plasma and serum leptin concentrations (Nicklas, Katzel, Ryan, Dennis, & Goldberg 1997; Vettor, DePergola, Pagano, Enlardo, Laudadio, Giorgino, Blum, Giorgino, & Federspil, 1997), racial and ethnic differences in lipid profiles and metabolism (Brown, Dothern, Suskind, Udall, & Blecker, 2000; Punyadeera, van der Merwe, Crowther, Toman, Schlaphoff, & Gray, 2001), differences in glucose and insulin levels (Velazquez-Mieyer, Cowan, Umpierrez, Lustig, Cashion, & Burghen, 2003), and variation in areas of basal metabolism of subcutaneous fat (Arner, Engfeldt, & Lithell, 1981). Significant individual differences in psychological variables within the obese population include gender differences in general self-esteem (Israel & Ivanova, 2002), age differences in physical self-esteem and locus of control (Israel & Ivanova, 2002; Mills, 1990), as well as differences in psychological distress observed between obese people who seek medical intervention (e.g., gastric bypass surgery) vs. obese who seek support for dietary restriction (e.g., Weight Watchers, Jenny Craig, etc.; Higgs, Wade, Cescato, Atchison, Slavotinek & Higgens, 1997).

Discussion in the aforementioned Fitzgibbon et al. (1993) study directly addresses individual differences within the obese population itself in emphasizing that in their

findings, between matched obese groups (which did not differ in weight or degree of obesity), the reports of higher psychopathology and binge eating in those seeking treatment could not be attributed to differences in weight status. Similarly, Guisado, Vaz, Lopez-Ibor & Rubio (2001) found significant individual differences within the subgroup of obese individuals currently seeking treatment for obesity depending upon the presence or absence of a comorbid (non-eating related) psychiatric diagnosis. Using data collected from a battery of measures to assess eating behaviors, results indicated that obese treatment seekers with a psychiatric disorder had a more destructured eating pattern (with a predominance of binge eating and disinhibition) than obese treatment seekers without a psychiatric disorder.

Of potentially greater import, individual differences in response to obesity treatment within the obese population have also been found. Differences in response to weight loss involve a multitude of factors and are less easily explained by differences in variables such as age, gender, or race. These variables typically found only to account for a small amount of the variance in changes in most outcome variables measured, again reflecting the complex and challenging nature of obesity treatment (Tuck, Sowers, Dornfeld, Whitfield & Maxwell, 1983; Barbeau, Butin, Litaker, Owens, Riggs, & Okuyama, 1999). Within the obese population, researchers have uncovered physiological differences in body weight and composition changes in response to diet and exercise prescriptions (National Task Force on Prevention and Treatment of Obesity, 1994; Barbeau et al., 1999; Anderson, Konz, Frederich, & Wood, 2001), significant individual differences in changes in leptin, insulin, blood pressure (Masuo, Mikami, Ogihara, & Tuck, 2001; Kawamura, Adachi, Nakajima, Fujiwara & Hiramori, 1996), as well as

significant differences in changes in levels of certain neurotransmitters (Tuck et al., 1983; Masuo, Mikami, Ogihara, & Tuck, 1997a; 1997b; 2001) following weight loss. These data suggest that there are individual differences which suggest that different treatment may be appropriate for certain subgroups within the overweight population.

In a study relevant to this assertion, Berman, Raynes, Heymsfield, Ackerman & Fauci (1993) found that obese individuals diagnosed with a personality disorder lost more weight on a behavioral modification weight loss program than those without a personality disorder. However, when the personality disordered patients were put on a liquid protein diet, they lost less than half the weight of those without an Axis II diagnosis. These results, displaying further individual differences within the obese population, attest to the fact that additional factors, other than body weight, carry important implications for classification and treatment. Fitzgibbon and Kirschenbaum make reference to these results in their 1990 article, and continue on to reiterate the earlier point that current literature suggests that there are subgroups among obese individuals that may respond differently to treatment and therefore require further examination.

> Clearly, further research needs to clarify distinctions among obese patients. It may have important implications of for the assessment and treatment of obesity. Careful assessment may reveal subgroups of obese patients who evidence specific types of disturbance. Those findings would encourage far broader and more thorough assessments in obesity treatment programs than are currently conducted and more careful study of the impact of differential adjustment on treatment outcome (p. 291).

Classification of the "Obesities"

In keeping with evidence suggesting that 'obesity' represents a heterogeneous condition containing large individual differences potentially relevant to research and treatment, several authors have made attempts to classify different types of "obesities" as oppose to relying on an obese vs. nonobese distinction in gathering clinical information for use in the treatment of excess body weight (Sims, 1982; Harrison, 1984; Hansen, 1984; Leibel & Hirsch, 1985; Bray, 1989; Fitzgibbon and Kirschenbaum, 1990). The purposes of such classification schemes include functional, therapeutic, and prognostic factors, with the lack of such reliable schemes in turn limiting our ability to diagnose, prognose, treat appropriately, prevent, and evaluate interventions (Harrison, 1984). Several of these authors have suggested grading schemes to classify the (specific obese) conditions in which various therapeutic modalities can be used at different times under different circumstances. Sets of descriptors could potentially be decided upon, with the criteria that the set be patient-oriented, multi-dimensional (e.g., physical function, medical risk status, sociodemographic status, etc.), objective, and relevant to functional patient outcome (Harrison, 1984). Theoretically, these variables could be described in a multi-level format that would allow sufficient detail in order to enable the classification to change as new information became available.

Though such classification schemes of the obesities hold intuitive appeal, the application of such schemes has been more difficult and less practical than may have been anticipated. Harrison (1984) proposed a classification scheme that contains 71 major items in order to diagnose the specific obesity from which a patient may be suffering. Hansen (1984) stresses the importance of factoring in hormonal changes in

potential classification schemes and cites 24 separate dimensions of hormonal changes to be considered in the classification of the obesities, not mentioned in the Harrison (1984) schemata. Additionally, Bray (1989) suggests the use of a 28-cell flow chart for use once the presence of overweight has been established to determine the degree of risk for additional health-related complications associated with obesity in individual patients. Essential variables in this framework not addressed by other proposed schemata include such things as evaluation of clinical signs of Cushing's syndrome, Dexamethasone suppression, hypoventilation syndrome, central nervous system lesions, and polycystic ovarian syndrome.

The complex nature of individual differences between persons suffering with obesity is evident in efforts to classify individual obesities of overweight individuals. Though such recommendations for classifying subgroups within the obese population are logical, they must be supported by empirical data linking individual differences within the overweight or obese population to treatment outcome. Currently, relatively few such individual differences have been reliably identified. Thus, most current attempts to tailor treatment according to the most relevant individual characteristics associated with body weight, will necessarily be based on relatively arbitrary determinations of which factors to address in individual treatment as there remains no set criteria or protocol for doing so. This is evidenced by the fact that previous attempts to tailor treatment to the individual have met with mixed results (Ard, Rosati & Oddone, 2000; Keele-Smith & Leon, 2003), and several clinical trials have revealed little or no differences between traditional weight loss interventions and interventions tailored to individual participants (Straw, & Terre, 1983; DeLuci, & Kalodner, 1990). Henderson & Huon (2002) suggest that the failure of

past attempts to tailor treatment to the individual may have been due to the fact that most have mistakenly been focused around addressing dietary restraint in these patients. Additional practical concerns such as monetary costs of individualizing treatment, general applicability, utility, and reliability have been raised. The difficulty in coordinating efforts in this area in the past is not, however, reason to revert back to a 'one-size-fits-all' approach toward the classification and treatment of obesity. Indeed, it has become necessary to better identify which factors are most relevant in the treatment of obesity. Additional research is required in order to narrow down the vast field of factors associated with the etiology, presence, and treatment of obesity.

Harrison (1984) and others (Rodin, 1981; Fitzgibbon and Kirschenbaum, 1990; Guy-Grand, 2003) make a clear plea for the evaluation and treatment of obesity on a sounder basis, to promote and note improvement in function, than to measure severity and outcome by the criterion of body weight alone. Treatment of the obesity disorder has proven itself complex and challenging enough that adequate assessment must identify the most pertinent psychological, social, and physiological factors surrounding eating behaviors (Brownell, 1982). Therefore, in an attempt to predict and control behavior with an eye towards prevention and treatment, it becomes necessary to examine correlates of human behavior. A strong connection between affect and behavior has been extensively supported in numerous areas of psychology including, but not limited to, parenting behaviors (Adam, Gunnar, & Tanaka, 2004), drug abuse (Hien & Miele, 2003), cigarette smoking (Gilbert & Wesler, 1989), alcohol abuse (Kodituwakku, May, Clericuzio, & Weers, 2001), aggressive behavior (Verona, Patrick, & Lang, 2002), and

eating behavior (Lowe & Maycock, 1988; Meyer & Waller, 1999). A more in-depth discussion of the relationship between affect and behavior follows.

Affect in Relation to Cognition and Health-Related Behavior

Affect may be best understood as the outcome of an evaluation of the extent to which one's goals are being met in interaction with the environment (Ortony, Clore & Collins, 1988). There is a preponderance of evidence supporting the connection between affect, particularly negative affect, and cognition and behavior (see Dolan, 2002 for a review). Affect influences numerous aspects of cognition and behavior, including memory for past experiences and newly learned material (Blaney, 1986; Ellis, Thomas & Rodrigues, 1984), performance on problem-solving tasks (Masters, Barden, & Ford, 1979; Mitchel & Madigan, 1984), evaluation of performance outcomes (Wright & Mischel, 1982), and social behavior (Clark & Isen, 1982; Isen, 1984, 1987).

Several studies have proposed that the management of negative affect states is a major motivator for cigarette smoking (Britt, 1996; Hall, Munoz, Reus & Sees, 1993; Gilbert & Wesler, 1989) and a number of studies have demonstrated a strong positive relationship between cigarette smoking and depressive disorders (Breslau, Kilbey, & Andreski, 1991, 1993; Glassman et al., 1990; Pérez-Stable, Marín, Marín, & Katz, 1990; Leftwich & Collins, 1994). The correlation between negative affect and drug and alcohol abuse has also been well documented (Dorus & Senay, 1980; Caper, 1981; Cottler, Shillington, Compton & Mager, 1993; Satel, Kosten, Schuckit & Fischman, 1993; Pathiraja, Marazziti, Cassano & Diamond, 1995; Wilson & Hayes, 2000). Of particular

relevance to this research is the relationship between affect and eating behavior, discussed in the next section.

Negative Affect, Overeating, and Body Mass Index

In addition to associations with other health-related behaviors, affect and emotions have been extensively related to eating behaviors. The tendency to eat in reaction to emotional states, particularly negative emotional states, has been well documented in the obesity and disordered eating literature (Striegel-Moore, Morrison, Schreiber, Schumann, Crawford & Obarzanek, 1999; Stice, Akutagawa, Gaggar & Agras, 2000). Additionally, several review papers have been published in past years, each addressing the relationship between affect, eating, and obesity (Allison & Heshka, 1993; Christensen, 1993; Ganley, 1989; Greeno & Wing, 1994). Nearly a half-century ago, Kaplan and Kaplan (1957) proposed a theory attempting to explain the relationship between negative affect and overconsumption. According to the 'psychosomatic theory', obese individuals are unable to distinguish between hunger and negative emotional states. This inability leads to eating under stress and results in obesity due to excessive caloric intake. Whether or not this theory accurately identifies the specific mechanism of action, the relationship between negative affect and eating, commonly referred to as "emotional eating", has been continually demonstrated in the literature, with increasing attention in recent years.

In a study carried out by Macht (1999), 107 female and 103 male participants were asked to report how various characteristics of eating changed with positive and negative emotions induced experimentally. Participants were asked to imagine either

positive or negative emotions while two adjectives were given in parentheses after each associated emotion in order to facilitate imagination of emotional experiences. Participants then responded to items measuring behavioral characteristics of eating as well as food- and eating-related feelings, perceptions and cognitions. Results indicated that negative (Anger) emotions increased comfort and impulsive eating as well as the overall tendency to eat. In this study, individual characteristics such as dietary restraint, body mass index (BMI; weight relative to height) and gender were taken into account and were found to have less impact on food consumption than the emotional states themselves.

In 1997, Meyer and Waller performed a study where different affective states were induced by flashing words subliminally to participants and measuring subsequent amounts of eating. Each participant was exposed to only one of the five words: gallery (neutral); hungry (appetitive); happy (positive emotion); angry (hostile emotion); or lonely (abandonment). In examining mean amounts (of crackers left in the experimental room) eaten by participants under the five conditions, the participants who had been exposed to the two negative emotion cues (angry and lonely) ate significantly more than those exposed to the neutral, appetitive, or positive emotion cues. Participants in this study were also given the Eating Disorders Inventory (EDI; Garner, 1991) and divided up into high and low disordered eating groups, however there were no significant differences in the amount eaten by these two groups following exposure to any word type. Additionally, the findings that neither exposure to the positive emotion cue (happy) nor the appetitive cue (hungry) lead to an increase in eating relative to the neutral cue (gallery), indicate that eating may be facilitated specifically by negative emotional

stimulation rather than general emotional arousal or appetitive cues. Similarly, Lowe & Maycock (1988) produced neutral or depressed moods in normal weight college students using Velten's (1968) mood induction method. During the mood induction procedure, subjects were encouraged to sample candy (M & Ms) that were earlier placed on the table in front of them. Results indicated that depressed (negative mood induced), high-hunger subjects were more likely to eat than subjects in other conditions and, in one of two analyses of amount of candy consumed, were found to eat the most candy as well.

In an examination of more enduring affect Williams, Healy, Eade, Windle, Cowen, Green & Durlach (2002) examined the ways in which trait-like styles of eating behavior interact with changes in affect contributing to the success or failure of attempts to diet. In three studies carried out by the aforementioned authors, associations were consistently found between negative affect and emotional eating. Results from these studies indicated that high emotional eaters also showed more cognitive deficits following a negative mood induction procedure compared to low emotional eaters. The authors theorized that this was due to an increased vulnerability to negative affect in emotional eaters. Emotional eating has additionally been related to unhealthy eating characteristics in both eating-disordered populations (Arnow, Kenardy & Agras, 1992, 1995) and non-clinical groups (Waller & Osman, 1998). In addition, Pawlow, O'Neil & Malcolm (2003) recently showed improvements in eating habits in participants with night eating syndrome due to lowered stress, anxiety, anger and depression following a week-long progressive muscle relaxation therapy intervention.

In an examination of eating-disordered populations, Grilo (2004) recently showed that cluster-analytic studies of eating disorders in adult patients yield two subtypes; pure

dietary and mixed dietary-negative affect. Cluster analyses of 137 patients with eating-disordered features revealed a dietary-negative affect subtype (43%) and a pure dietary subtype (57%). The dietary-negative affect subtype was characterized by greater eating-related psychopathology and greater likelihood of binge eating. In several additional studies, negative attitudes and emotions were the *only* significant predictors of the future development of disordered eating, particularly binge eating (Leon, Fulkerson, Perry, Keel & Klump, 1999; Leon, Keel, Klump & Fulkerson, 1997; Kitsantas, Gilligan & Kamata, 2003).

Binge Eating

Current literature suggests that there may be a particularly noteworthy association between negative affect and binge eating. Though the majority of research in this area has focused on patients or others meeting the diagnostic criteria for Bulimia Nervosa (BN; Lynch, Everingham, Dubizky, Hartman & Kasser, 2000; Lynch et al., 2000), diagnosed bulimics make up only a small fraction of individuals who regularly binge eat (Krahn, Kurth, Demitrack & Drewnowski, 1992). The larger 'sub-clinical' group includes both men and women who meet some, but not all, of the diagnostic criteria for BN, as well as others who meet most or all the research criteria for binge eating disorder (BED; American Psychiatric Association, 1994; Lynch et al., 2000). Binge eating is characterized by eating, in a discrete period of time, an amount of food that is definitely larger than most individuals would eat under similar circumstances, accompanied by a sense of lack of control (American Psychiatric Association, 1994).

According to the DSM-IV, in 1994, the prevalence of BED in community samples was estimated to be as high as 4 percent (American Psychiatric Association, 1994). By contrast, however, a frequently cited study by Halmi, Falk & Schwartz, conducted in 1981, found that 13 percent of first-year college students reported current binge eating. Similar prevalence estimates of sub-clinical binge eating have been reported by several other studies. Kurth et al. (1995) interviewed nearly 1500 college women and found that 35 percent reported a history of binge eating at some point in their lives. Within the overweight population itself, binge eating appears to be especially prevalent (de Zwaan, Nutzinger, & Schoenbeck, 1992; Devling, Walsh, Spitzer, & Hasin, 1992; Spitzer, Yanovski, Wadden, Wing, Marcus, Stunkard, Devlin, Mitchell, Hasin, & Horne, 1993). Binge eaters are considered to represent a sizable and distinct subgroup of the obese population (Yanovski, Nelson, Dubbert & Sptitzer, 1993; Spitzer et al., 1993; Pinaquy, Charbrol, Simon, Louvet & Barbe, 2003). The exact prevalence of binge eating within the overweight population remains unknown (Marcus, 1993), however, estimates of 25 percent or higher are common (Spitzer et al., 1993). Binge eating has also been implicated in the development of obesity (Mussell, Mitchell, Weller, Raymond, Crow & Crosby, 1995; Yanovski, 2003), and has been shown to be a major risk factor for weight regain following a weight loss diet (McGuire, Wing, Klem, Lang & Hill, 1999). Additionally, it has been shown that amelioration of binge eating, even in the absence of obesity treatment, results in weight losses and decreased weight regain over time (Yanovski, 2003).

Two main theoretical formulations have been proposed to account for the way recurrent binge eating develops. The first views binge eating as a consequence of dietary

restraint, and the second as a response to negative affect. Restraint that dieters impose on their eating has been implicated as a potential contributor to the development of binge eating problems in normal weight individuals (Marcus, 1993). According to restraint theory mentioned earlier (Polivy & Herman, 1985), hunger that is induced by dieting establishes conditions that potentiate binge eating in the presence of specific disinhibitors, such as alcohol intake and abstinence violations. However, there is considerable evidence suggesting that the role of dieting in the binge eating problems of overweight individuals cannot be adequately explained by this theory (Howard & Porzelius, 1999). According to restraint theory, dieting *leads to* binge eating, however, in studies specifically examining the temporal sequence of dieting and binging, only a small minority of overweight binge eaters reported that their dieting preceded the onset of their binge eating. (Mussell et al., 1995; Henderson & Huon, 2002). Additionally, overweight binge eaters have been shown to be no more likely than overweight non-binging individuals to diet; contrary to what the restraint theory would suggest (Wilson, Nonas & Rosenblum, 1993).

The second explanation of binge eating focuses on the aforementioned influence of affect on eating. There is considerable evidence that negative affect is a salient predictor of bulimic behaviors in overweight persons (Agras & Telch, 1998), and binge eating has been repeatedly shown to occur in response to negative affect (Henderson & Huon, 2002; Abraham & Beumont, 1982; Arnow, Kenardy, & Agras, 1992; 1995; Heatherton & Baumeister, 1991; Herman & Polivy, 1975; Lingswiler, Crowther, & Stephens, 1989; Ruderman, 1985; Davis, Freeman & Garner, 1988). One widely cited theory consistent with extant literature proposes that binge eaters learn to regulate

negative emotions by binge eating (Heatherton & Bauumeister, 1991; Stice, 1994; McManus & Waller, 1995). In support of this explanation, Arnow, Kenardy, and Agras (1992) examined the relationship between fluctuations in mood and binge episodes among obese, non-purging binge eaters and found that typical binge episodes were precipitated by negative emotional states. More recently, Arnow, Kenardy, and Agras (1995) showed that scores on all three subscales of the Emotional Eating Scale (anger/frustration, anxiety, and depression) correlated significantly with the occurrence of binge eating episodes across a one-week recall by overweight women. Additionally, Eldredge and Agras (1996) found that, compared to obese individuals who did not binge eat, obese binge eaters were significantly more likely to report eating in response to negative affect.

There is also evidence to suggest that, not only negative affective states, but enduring negative affect is also associated with increases in binge eating in overweight individuals. Henderson & Huon (2002) recently examined the relationship between trait-like negative affect and binge eating. 105 overweight women were asked to fill out the general version of the Positive and Negative Affect Scales (PANAS; Watson, Clark & Tellegen, 1988); a trait measure of positive and negative affect where participants indicate next to each item to what extent they "generally felt that way". Ratings of how participants generally felt were given on a 5-point scale from 1 (very slightly or not at all) to 5 (extremely), and total scores were derived from the sum of all responses. Results from this study indicated that overweight women with higher levels of trait-like negative affect had more severe binge eating problems than those who generally experienced a low level of affective distress. Similarly, Wolff, Crosby, Roberts & Wittrock (2000)

found that binge eaters in their experiment, measured daily over a three-week period and averaged together, reported a significantly more pervasive negative mood than did non-binge eaters. Also, in 1993, Yanovski et al. found that there was a significant relationship between lifetime prevalence of major depression, panic disorder, borderline personality disorder, and avoidant personality disorder in overweight or obese binge eaters. The relationship between trait depression, anxiety, perceived stress and binge eating has been replicated in several other studies as well (Cargill, Clarck, Pera, Niarua & Abrams, 1999; Pinaquy et al., 2003).

It has also been suggested that individuals who binge eat have a trait-like hypersensitivity to perceived threatening or negative affective cues. Several studies have found that binge eaters show attentional biases towards self-directed ego threats (McManus, Waller, & Chadwick, 1996; Waller, Watkins, Shuck, & McManus, 1996) as well as a bias towards body weight- and shape-related cues (Cooper, 1997; Schotte, McNally, & Turner, 1990). It has been demonstrated that binge eaters may have an attentional bias towards negative emotion words (Rieger, Schotte, Touyz, Beumont, Griffiths, & Russell, 1998). Additionally, Wolff et al. (2000) found that binge eaters reported that they experienced twice as many negative affect-evoking events daily as compared to controls, and rated the emotional impact of such events as significantly greater than did controls experiencing identical events. In a particularly interesting study, Pinaquy et al. (2003) found that overweight binge eaters were more likely to suffer from alexithymia (the inability to identify and express emotions and affects). In this study, perceived stress and depression were significant predictors of emotional eating in both binge eating and non binge eating obese individuals, however, presence of alexithymia

only significantly predicted emotional eating in obese binge eaters. Overall, these findings suggest that binge eating is associated with overelaborated cognitive representations of negative emotional and threat-related information (Meyer & Waller, 1998), and that binge eaters may be less able to deal with their negative affect in a healthy manner than non binge eaters. Evidence from these studies support the model of emotionally-driven binge eating and are consistent with research showing that overweight binge eaters report more negative affect prior to binges than they or controls do prior to normal eating (Davis, Freeman & Garner, 1988; Lingswiler et al., 1989). That is, several above authors suggest that binge eaters are likely to experience more enduring negative affect, experience more negative affect in response to external stimuli, and are more likely to binge eat in response to negative affect.

In light of the considerable evidence supporting connections between affect, particularly negative affect, and eating behaviors, particularly binge eating, coupled with the implications of binge eating in the development of obesity, further research in this area is warranted. Henderson & Huon (2002) state that further investigation of this relationship may help effectively tailor treatments to individuals within the overweight of obese population and that efforts should be directed towards helping patients recognize the way that eating is employed as a means of coping with affective distress. Similarly, Stice et al. (2000) suggest that new techniques for clarifying the effect of negative affect, particularly "naturally occurring negative affect", on binge eating should be sought. These authors state; "…it would be useful for future research to explore alternative analytic techniques that are more sensitive to this type of [naturalistic] affect." (p. 227). This research attempted to do exactly that. As such, a brief review of conventional and

current analytic techniques for studying affect, and their associated theoretical models, is provided.

Methods for Studying Affect

In past years, methods of studying affect have predominantly been through the use of various self-report measures (Vastfjall, Friman, Garling, & Kleiner, 2002; Roger & Najarian, 1989; Lorr, 1989; Lubin, Hanson & Colquitt, 1992). Conventional thought surrounding the measurement of affect lead to the assumption that emotional experience of this sort could only be studied via the introspective report of the experiencing subject. In previous decades, many believed that even if we were able to obtain physiological measurements and objective measures of expressive behavior, we would not be measuring the emotional experience, since researchers had no way of analyzing the manner in which the subject experiences the physiological and expressive changes other than by self-report (Wallbott & Scherer, 1989). Self-report measures, however, are subject to numerous biases and introspective limitations on the part of the respondent (Aiken & West, 1990). Additionally, specific categories of emotion are not considered to be mentally represented as independent or mutually exclusive, but related in groups of emotion (Russel, 1989), and research has also demonstrated that people often display emotional behavior in the absence of concomitant conscious emotional experience (Ohman, Flykt & Lundqvist, 2000).

More recent research into the study of emotion has lead to the development of computer-based techniques such as measuring reactions to digitized sounds, words,

musical pieces, or faces (e.g., Shubert, 1999; Bradley & Lang, 1999). Using these techniques, emotional reactions are assessed in response to differing stimuli by having participants squeeze a hand-grip, slide a lever, turn a dial, or move a cursor along a computer-generated scale. Such techniques, however, are still dependent on self-reported responses by participants and thus subject to many of the same limitations as traditional oral and paper-and-pencil self-report measures (Schmidt, 1996). Lang, Cuthbert, & Bradley (1998) articulate the need for further methods of studying affect; "Emotions are multisystem response arrays. We will need to measure these arrays more broadly than we routinely do now if we are to enhance understanding of emotional pathology and significantly improve success in reducing patient distress" (p. 671).

The apparent limitations of relying on the subjective report of individuals have inspired new frameworks for viewing, and assessment techniques for quantifying, emotion. Investigative methodology from the field of cognitive neuroscience has recently been incorporated into, and has subsequently expanded, the study of human affect. It has been established that evaluation of internal affect involves a cognitive process of some type (Lane et al., 2000). Since fundamental discoveries have been made regarding the neurobiological basis of emotion, the neuroscience of both conscious and unconscious processes in emotion have allowed this branch of research to advance rapidly in recent years (Lane & Nadel, 2000). Many associations between affect and brain functioning are now able to be accurately measured and recent literature in this area is beginning to burgeon (Dolan & Morris, 2000; Heilman, 2000; Jackson, Mueller, Dolski, Dalton, Nitschke, Urry, Rosenkranz, Ryff, Singer & Davidson, 2003). For example, Jeong, Joung & Kim (1998) developed a new system that determines and

quantifies the way in which the emotional response to music is reflected in the electrical activities of the brain. Authors in this study found that the brains of individuals who feel more pleased show decreased chaotic electrophysiological behavior in response to music. In 1997, Lane, Fink, Chau, & Dolan measured brain activation, via Positron Emission Tomography (PET), of participants as they viewed picture sets designed to elicit either positive, negative, or no (neutral) emotions. While being scanned, participants were also asked to categorize the presented stimuli into one of those three categories. Results from this study indicated participants experienced consistent increases in activity in the medial prefrontal cortex during selective attention to their subjective emotional responses, implicating this region in the processing of human emotion.

Researchers have, indeed, begun to answer the call for more psychometrically sound affective measures, and are recruiting new assessment strategies and tools, enabling research in this field to look beyond traditionally employed methods in studying affect and emotion. Modern technology may allow more of a window into some of the biological underpinnings of human affect and its relation to behavior. As in the examples cited above, advances in Functional Magnetic Resonance Imaging (fMRI), PET, Evoked Response Potential (ERP), Single Photon Emission Computed Tomography (SPECT), and sophisticated Electroencephalograph (EEG) measurement technology have all been incorporated into the study of emotion in recent years. With these new and powerful techniques for measuring brain functioning and well-established connections between brain functioning, affect, and behavior (Dolan & Morris, 2000; Bradley & Lang, 2000; Dolan, 2002), researchers are now able to examine affect, and subsequent human behavior, through potentially more reliable and valid methods. With the ability to

examine affect in such ways, ‘affect’ itself must be operationalized, as it has commonly been associated with a wide array of feelings and cognitions (Lorr, 1989). With a focus on human behavior, researchers have made an attempt to identify which specific dimensions of affect are most closely associated with behavior and, thus, potentially most worthy of more intensive examination.

Models of Human Affect

Cannon (1927) described emotions as primarily adaptive; they serve to assist in preparing the organism to deal with important events. In a similar vein, Bradley and Lang (2000) describe emotions as having evolved from simple reflexive actions, many of which are still a part of the human response repertoire. Among the most primitive and essential of these responses are movements toward positive, appetitive things and movements away from negative, unpleasant things. In humans, elaborate neural systems support these responses, better facilitating adaptation to the environment. Within this theory, affect is considered a dimensional construct consisting of both valence and motivational characteristics. Affective 'valence' is described as whether a particular emotion is generally perceived of as more positive or more negative. The associated 'motivational' characteristics are either appetitive, causing us to move towards (approach) a positive stimulus; or aversive, causing us to move away from (withdraw) from a negative stimulus. Heilman (2000) also describes emotion as a major motivating factor in approach and avoidance behaviors. This author expands on this theory in stating that it is not only the direct experience of emotion that motivates behavior, but also that the perception that certain stimuli, situations, and actions could in the future produce emotional states is a strong and important motivating factor. Many researchers support this contention and now believe that neurophysiological data can elucidate the valence and motivational characteristics of emotion and can be of great value in diagnoses and treatment of various psychological and behavioral problems (Lang et al., 1998; Jeong, Joung & Kim, 1998; Bradley & Lang, 2000; Lane, Nadel, Allen & Kaszniak, 2000).

Several authors argue that affect may best be quantified in terms of physiological reactions, including changes in the brain and in the somatic and visceral systems that are conceived to be the logistic support of intended action (Lang et al., 1998; Bradley & Lang, 2000) and that these physiological reactions commonly referred to as 'emotions' are driven by deep and subcortical motivation systems (Fanselow, DeCola, De Oca & Landeira-Fernandez, 1995; Davis, 1997; Bradley & Lang, 2000). From the evolutionary perspective cited above, it is held that affect evolved from these reflexive, overt reactions to appetitive or aversive stimulation that served immediate survival functions (e.g., nurturance, sexual approach; fight, flight). Within this framework, negative emotion or affect is seen as a state in which the defensive components of the motivation centers in the human brain are active, and related primitive autonomic and somatic reflexes are readied for action. The evolution of the human brain, particularly that of the cerebral corticies, made possible more complex responses to appetite- and threat- inhibition, delay, evaluation of context, future planning- which were even more effective in ensuring survival. Thus, in human emotion, complex information networks are activated that react to perceived cues, including memorial representations, and result in varied cognitive and behavioral outputs (Lang et al., 1998). Damasio (1994) coined the term "somatic marker" and describes emotion as a "gut feeling" that influences decision making in practice (p. 184). In sum, the prevailing current theory views emotion as measurable activation in a motive system that is often indexed by the consequent actions (Bradley & Lang, 2000).

Positive and Negative Affect

Subjective experience often leads to the assumption that there exists an array of emotions or different affective states and traits that one can experience over the course of a lifetime (Lorr, 1989). Another concept of affect, however, arose from studies by Zevon and Tellegen (1982), Tellegen (1985), Watson (1988) and Watson & Tellegen (1985). These authors' contentions were, in part, derived from research conducted by Osgood, Suci & Tannebaum (1957) who determined that the largest amount of variance in semantic evaluation was accounted for by affective valence, which mapped onto a continuous dimension from unpleasantness to pleasantness. The more modern theory proposed by Zevon, Tellegen and Watson, in fact, goes a step beyond that of Osgood and colleagues in asserting that *all* emotional experiences can be reduced to a two-dimensional framework of Positive Affect and Negative Affect. This conceptual framework has attracted considerable attention and is generally seen as complimentary to, rather than competitive with, multi-factorial structures (Lorr, 1989). In initial studies, the authors assembled a basic checklist of adjectives from available lists in extant literature in order to assure a broad representation of state and affect variables. The list was constructed by selecting three adjectives from each of 20 affective and emotion categories, yielding a total of 60 adjectives. Illustrative content categories are as follows: Excited, Strong, Joyful, Tired, Angry, Fearful, Jittery, and Content.

In initial analyses, (Zevon & Tellegen, 1982), a principal component analyses (R-analysis) was applied to the correlations among the 60 adjectives rated by 284 participants. Two broad orthogonal dimensions of affect emerged. The Positive Affect factor was loaded positively by all the positive adjectives and negatively by *sleepy*,

sluggish, and *tired*. The Negative Affect factor was defined by all the negative adjectives, with *content*, *at ease*, and *calm* loading negatively on this factor. In a second study, the authors investigated the structure of intraindividual mood by P-factor analysis. Here 23 participants rated their moods on the 60 adjectives during mornings, afternoons, and evenings for 90 days. The main analysis consisted of a series of P-factor analyses of each individual's response protocol, checking for the presence of two (positive and negative) affect dimensions. Applications of coefficients on congruence indicated large similarities between continuous mood ratings and the individual two-factor positive and negative affect structures. The multifactorial solutions derived for each participant yielded three positive mood factors (Joy, Physical Well Being, and Interest) and five negative mood factors (Guilt, Fear, Fatigue, Distress, Loneliness, and Surprise).

Seeking a consensus regarding the dimensional structure of affect, Watson and Tellegen (1985) reviewed published studies that would lend themselves to reanalyses and that were adequate in sample size. Included were studies by Thayer (1967), Hendrick and Lilly (1970), Borgatta (1961), McNair, Lorr, and Droppleman (1971), Lebo and Nesselroade (1978), and Russell and Ridgeway (1983). These were compared with Zevon and Tellegen (1982) and with Japanese data collected by Watson, Clark, and Tellegen (1984) for analyses. An approximation of the original correlation matrices of the eight studies was reanalyzed by principal factor analysis. Two large orthogonal factors emerged in every case, each accounting for approximately one-half to three-quarters of the common variance. These factors represented positive and negative affect. In addition, correlation coefficients were calculated between loadings on the factors

obtained in each of the solutions, and the pattern of results indicated considerable agreement.

Based on the research and empirical support for partitioning affect into a dimensional construct consisting of positive and negative affect, this research focused on these two affective valences, particularly negative affect, and their respective motivational correlates. One additional dimension of relevance to the study of human affect that has been alluded to but not yet explicitly addressed is the chronicity of emotional experience. There is a relative dearth in the literature focused on the delineation between affective states versus affective traits (Zuckerman, 1983). Questions that have long permeated throughout the study of human affect involve the distinction between an ephemeral affective state, recurring affect, an affect that endures for a relative length of time, commonly referred to as 'mood', and an affect that is enduring enough to be considered a 'personality trait'. It is possible that future research into human emotion will provide definitive answers to such questions, however there currently exist no such clear-cut distinctions.

Differentiation Between Trait Affect and State Affect

For a long time, there has been confusion and conflict over the nature of states and traits (Allen & Potkay, 1981; Zuckerman, 1983). Typically, individual attributes are viewed as more or less state-like or more or less trait-like. The two concepts are not sharply differentiated because they overlap in meaning. Terms denoting states and feelings typically refer to brief and temporary experiences that are manifested

sporadically and irregularly. In general, states are feelings evoked by situational pressures, social-environmental conditions, cognitions, temporary physiological changes or some combination thereof. Traits, in contrast, are viewed as stable, long-lasting, behaviors manifested in a variety of situations. Consequently, traits must be observed more frequently than states and across more situations before they are attributed to a person (Lorr, 1989).

It is possible that states and traits can interact with one another. There is evidence that trait-like affect can affect state-like affect and subsequent behaviors. Heilman (2000) points out that moods, commonly described as long-lasting emotions, modulate, influence, or bias perception, cognition, memory, and emotion. This author refers to an example where persons with a depressed mood are able to remember unpleasant events better than pleasant events. Verona, Patrick, & Lang (2002) found that participants high in trait negative emotionality were more likely to react faster and more aggressively in response to state negative affect induced by aversive air blasts. It has also been suggested that certain traits exist at a more basic level of cognition and that other trait moods exist as a mediator between those primary traits and mood states (Nemanick & Munz, 1997). Similarly, Williams and colleagues suggest that the effect of long-term traits on current behavior depends on whether they are 'activated' by current mood (Williams et al., 2002). There is also evidence that trait-like negative affect may increase the likelihood that persons will eat in response to state-like negative affect. Results from two studies performed by Williams et al. (2002) indicated that dieters who reported poorer overall psychological well-being showed a greater tendency to eat in response to emotional cues, and more disturbed mood.

The trait vs. state distinction becomes relevant in measuring emotion as short-term feelings of negative affect surely hold differential implications for treatment than longer-term negative affect. Though state-like negative affect certainly should not be ignored and necessarily assumed benign, in the absence of stressors, negative mood states are generally low in healthy personas and may not be considered problematic in psychological or behavioral treatment (Zevon & Tellegen, 1982; Scherer, Wallbott & Summerfield, 1986). When negative affect states become chronic or enduring, it is unclear where exactly the threshold of a trait-like attribute is crossed, however it is evident that this condition could potentially be far more problematic than an occasional feeling of malaise (Clark, Watson & Mineka, 1994; Macht, 1999; Davidson, 1998a). Though distinguishing between states and traits may hold utility in the prediction and control of human behavior, no explicit set of criteria has been established for distinguishing states from traits as of yet. For example, the terms *anxious* or *depressed* are classified according to the views of the investigator. Generally, these concepts are seen as fuzzy, overlapping categories, organized around a core set of prototypic exemplars (Rosch & Lloyd, 1978; Lorr, 1989). Class membership is a matter of degree and there are no clear-cut boundaries separating categories (Lorr, 1989).

It has been noted that community-based studies measuring longitudinal negative affect (Stice, Killen, Hayward & Taylor, 1997; Stice et al., 2000) show more consistent and stronger main effects on binge eating than do negative affect inductions in laboratory settings (Stice et al., 2000). These authors indicate that the temporal duration of negative affect may be an important factor in this relationship. Similar results indicating a greater impact of enduring naturalistic negative affect vs. induced, short-term negative affect

have been obtained by several authors (Kwiatkowski & Parkinson, 1994; Grimmett, 1998; Tagami, 2002). As such, and given the apparent complexity and still yet-to-be-identified nature of the exact relationship between states and traits, this research did not attempt to make clear-cut distinctions between state affect and trait affect, but focused on the side of the spectrum involving relatively more enduring affect.

Affect in the Human Brain: The Anatomy of Approach and Withdrawal

The overwhelming majority of research in this area has been conducted by Dr. Richard Davidson and colleagues at The University of Wisconsin, Madison. Davidson and colleagues emphasize that, to date, relatively few studies of emotion have been performed using modern neuroimaging procedures that afford a high degree of spatial resolution (Tomarken et al., 1992; Davidson & Irwin, 1999; Davidson, 2003), and that the circuitry involved in emotion in the human brain is extremely complex, involving numerous interrelated structures and substrates (Davidson, 2000). As such, hypotheses about the set of structures that participate in the production of emotion are still considered somewhat speculative and are based, in large extent, on animal literature and theoretical accounts of the processes involved in human emotion (Davidson, 2000; Miller & Cohen, 2001). Based on theory and available strands of evidence in extant literature, scientists have proposed two basic circuits mentioned earlier, each mediating different forms of emotion (Lang et al, 1990; Davidson, 1995; Bradley & Lang, 2000). The approach system facilitates appetitive behavior and generates certain types of positive affect that are approach related (e.g., enthusiasm, pride, agency; Depue & Collins, 1999). This form of positive affect is typically generated in the context of moving toward a desired goal (Lazarus, 1991; Stein & Trabasso, 1992).

It should be noted that the activation of this approach system is hypothesized to be associated with a particular form of positive affect and not all forms of such emotion (Davidson, 2000; 2003). It is specifically predicted to be associated with pre-goal attainment positive affect; the form of positive affect that is elicited as a person moves closer toward a specific appetitive goal. Within this circuit, it has been demonstrated that large individual differences exist in the tonic level of activation of the approach system, which alters an individual's propensity to experience approach-related positive affect (Tomarken et al., 1992; Davidson, 2003). Similarly, evidence suggests that there is a second system concerned with the neural implementation of withdrawal. This system facilitates the withdrawal of an individual from sources of aversive stimulation and generates certain forms of negative affect that are withdrawal related (Davidson, 2003). Both fear and disgust are associated with increasing the distance between the organism and the source of this aversive stimulation (Tomarken et al., 1992; Davidson, 1995). This research examined aspects and correlates of both the positive and negative affect systems, however, specific hypotheses mainly involved the negative affect system.

In the human brain, it is the prefrontal cortex (PFC) that is hypothesized to contain the representation of a goal state or motivation relative to learned behavioral-reinforcement contingencies; which may be experientially described as an emotion (Fanselow et al., 1995; Davis, 1997; Bradley & Lang, 2000; Davidson, 2000; Miller & Cohen, 2001). Support for this proposed relationship has stemmed from several branches of cognitive neuroscience. In 2002, Zald, Mattson & Pardo performed two studies examining brain activity in the absence of stimulation through resting regional cerebral blood flow (rCBF; index of activation in PET studies; similar to a reduction of alpha

amplitude in EEG studies). 51 participants in the first study and 38 participants in the second study retrospectively rated the extent to which they had experienced negative affect related mood states over the prior month using the PANAS. In both studies, a correlation emerged between self-ratings of negative affect for the month before scanning and resting rCBF in the prefrontal cortex. In a previously described study, Lane et al. (1997) found increased activation in the PFC during subjective emotional responses to stimuli using PET. In another PET-derived study, Drevets, Price, Simpson, Todd, Reich, Vannier & Raichle (1997) found that, across three samples of depressed subjects, the PFC consistently showed reduced activity compared to controls. Similar results have been obtained using both SPECT (Galynker, Cai, Ongseng, Finestone, Dutta, Serseni, 1998) and fMRI measures (Ochsner, Bunge, Gross & Gabrieli, 2002).

The Prefrontal Cortex

Miller and Cohen (2001) have outlined a comprehensive theory of the role of the PFC based on an impressive quantity of the extant literature from current computational modeling, nonhuman primate neurophysiological and anatomical studies, and human neuroimaging and neurophysiological studies. They describe the PFC as a collection of interconnected neocortical areas that send and receive projections from virtually all cortical sensory systems, motor systems, and many subcortical structures. The core feature of their model is that the PFC maintains the internal representation of goals and the means to achieve them. That is, consistent with theory stated above, the PFC is asserted to contain and maintain desired end state(s) and, based on past experience, evaluate competing alternatives and ultimately direct action potentials in a manner that

will either move the organism closer to reaching a desired end state (approach) or farther away from an aversive stimulus (withdraw). Davidson (2003) asserts that these decisions in which the PFC is activated are not experienced as cold calculus in weighing pros and cons and recalling similar situations and experiences in the past, but that such decisions are commonly made on the basis of, and experienced as, 'feelings'.

According to this theory, the PFC becomes most important when "top-down" processing is needed; when behavior must be guided by internal states or intentions, particularly when there are competing alternatives. A common example of this is in the case of delayed gratification, where an immediately available reward may impede the acquisition of a more long-term goal (e.g., choosing not to eat a desired desert in the hopes of fitting into a smaller size dress for a particular event). In this case, the PFC would be required to produce a bias signal to other brain regions that guide behavior to the more desired goal. The effect of these bias signals is to guide the flow of neural activity along pathways that establish the proper connections between inputs, internal states, and outputs needed to perform a given task. This is especially important whenever stimuli are ambiguous or when a task-appropriate response must compete with stronger alternatives. Miller & Cohen (2001) describe this flow of neural activity for goal-directed decision making "activity flow" and relate the function of the PFC to that of a switch operator in a system of railroad tracks. Within this model, the brain is seen as a set of tracks (pathways) connecting various origins (e.g., stimuli) to destinations (responses).

Davidson (2003) describes this same process as "affect-guided planning and anticipation that involves the experience of emotion associated with an anticipated outcome" (p. 656). Though not quite as parsimonious as "activity flow", both Davidson

and Miller & Cohen ascribe to the same theory of central the role of the PFC. Davison describes this affect-guided anticipation as the hallmark of adaptive, emotion-based decision making and, in agreement with the above theorists, contends that it is most often accomplished in situations where there is strong competition from alternatives. According to this model, it is in these cases that the PFC is particularly essential and, therefore, would expect to find more activation in this area during periods where evaluation of alternatives is not easily executed. In further support of this contention, Damasio (1994) found that patients with lesions in certain areas of the PFC were shown to exhibit profoundly impaired decision making abilities. Davidson (2003) and colleagues (Davidson et at., 2000; Davidson & Irwin, 1999) provide evidence that the failure to anticipate positive incentives and direct behavior toward the acquisition of appetitive goals are symptoms of depression that may arise from irregularities in the circuitry that implements this positive affect-guided anticipation. Additionally, it has been demonstrated that patients with brain lesions in the frontal cortex area fail to show normal autonomic responses to socially and emotionally meaningful stimuli (Damasio, Tranel & Damasio, 1990) and demonstrate a marked impairment in the ability to appreciate future risks and fail to produce anticipatory autonomic responses to potential rewards or punishments (Bechara, Damasio, Damasio & Anderson, 1994; Bechara, Tranel, Damasio & Damasio, 1996).

Lateralization of Affect and Motivation and the Role of the PFC

It is commonly acknowledged that cognitive functions such as language acquisition, memory, and face recognition are hemispherically localized in the human brain (Ross, 1984; Damasio & Damasio, 1992; Gaillard, Bookheimer, Hertz-Pannier, & Blaxton, 1997; Burgess, & Gruzelier, 1997). The idea that the two hemispheres of the human brain may also have differential roles in the control of emotion and affect dates back more than 60 years (Alford, 1933, Goldstein, 1939). Goldstein (1939) reported that left hemisphere lesions produced severe reactions characterized by fearfulness and depression, whereas right hemisphere lesions produced a state of "indifference". One theory regarding the role of the two hemispheres suggests that the right hemisphere is dominant for emotional expression in a manner parallel to that of the left hemisphere dominance of language (Ross, 1984). Current theory and research now suggests the two hemispheres have a complimentary specialization for the control of different aspects of mood and affect. In particular, the left hemisphere is considered to be dominant for "positive" affect and the right hemisphere for "negative" affect (Sackheim, Greenberg, Weiman, Gur, Hungerbuhler, & Geschwind, 1982; Canli, Desmond, Zhao, Glover, Gabrieli, 1998; Davidson, 2000; 2003)

In electrophysiological studies of humans in the late 1970's, Davidson and colleagues (Davidson, Schwartz, Saron, Bennett & Goleman, 1979) noted that a pattern of asymmetrical activation in the prefrontal cortex that related to positive and negative affect. This 'asymmetrical activation' denoted activation in either the right or left side of the PFC, relative to activation in the opposite side, and has come to be referred to as 'prefrontal asymmetry'. Davidson reported that in studies of both adults and infants, left-

sided prefrontal asymmetry was associated with elicitors of positive affect, while right-sided prefrontal asymmetry was associated with elicitors of negative emotions (Davidson et al., 1979; Davidson, 1982). In 1989, Davidson & Fox reported that 10-month old infants who cried in response to maternal separation were more likely to have shown more right-sided prefrontal asymmetry during a preceding resting baseline assessment compared to infants who did not cry in response to this challenge. Soon after, Davidson, Ekman, Saron, Senulis & Friesen (1990) demonstrated that greater right-sided prefrontal asymmetry was associated with what he would later refer to as "withdrawal-related negative affect" (Davidson, 2000). In this study, both positive and negative emotion-eliciting film clips were shown and, in all participants, more right-sided prefrontal asymmetry was found while viewing the negative film clip compared to the positive film clip. Seeking to expand on these findings, and utilizing existent theory regarding the role of the PFC at the time, Davidson and colleagues produced an extremely influential series of studies using EEG measurements of activation in the PFC to examine its relationship with these approach and withdrawal dimensions of positive and negative affect in humans (Davidson, Kalin, & Shelton, 1993; Davidson, 1994; 1998; Davidson & Irwin, 1999; Davidson et al., 2000).

In 1992, Tomarken, Davidson, Wheeler & Doss administered the trait version of the PANAS to 90 undergraduate students and found that participants with more left-sided asymmetrical activation in the PFC reported more trait-like positive and less trait-like negative affect than participants with more right-sided prefrontal asymmetry. In 1997, Sutton and Davidson administered the behavioral activation system and behavioral inhibition system (BAS/BIS) scales (Carver & White, 1994; measure designed to assess

approach and withdrawal tendencies) to 23 participants. The authors in this study found that scores on these scales were even more strongly predicted by electrophysiological measures of prefrontal asymmetry than were scores on the PANAS scales. Participants with greater left-sided prefrontal asymmetry reported more relative behavioral activation (approach tendencies) than behavioral inhibition (withdrawal tendencies) compared with participants exhibiting more right-sided prefrontal asymmetry; consistent with the theorized role of the PFC described in the preceding section. Davidson and his colleagues have also replicated their earlier findings (Davidson et al., 1990), demonstrating that measures of prefrontal asymmetry predict reactivity to experimental elicitors of positive and negative affective states in both human and nonhuman primate studies (Davison et al., 1993; Davidson et al., 2000).

Davidson (1993) developed a model that can still be considered consistent with extant literature today, though this area of research is yet to reach full consensus on the exact role of the PFS in relation to approach- and withdrawal- related affect (see Harmon-Jones, 2003a for a review). Davidson's framework features individual differences in prefrontal activation as a reflection of a diathesis which modulates reactivity to emotionally significant events. That is, individuals with more right-sided prefrontal asymmetry (previously shown to be associated with negative affect and behavioral inhibition or withdrawal reactions) are theorized to react to stimuli (both internal and external) in a more negative manner than individuals with more left-sided prefrontal asymmetry and vice versa. Additionally, according to this theory, this predisposition to react more negatively to stimuli should remain regardless of the particular emotional state the individual is experiencing at the time the PFC becomes activated in response to the

need to choose between competing goal states (i.e., either approach an appetitive stimulus or withdraw from an aversive stimulus).

According to the above model, individuals who differ in prefrontal asymmetry should respond differently to an elicitor of positive or negative emotion regardless of baseline mood. Wheeler, Davidson, & Tomarken (1993) performed a study to examine this hypothesis. Authors in this study presented short film clips designed to elicit positive or negative affect. Brain electrical activity was recorded before the presentation of the film clips. Just after the clips were presented, participants were asked to rate their emotional experience during the preceding film clip. In addition, participants completed scales designed to reflect their mood at baseline. Results indicated that individual differences in prefrontal asymmetry predicted the emotional response to the film clips even after measures of baseline mood were statistically removed. Those individuals with more left-sided prefrontal asymmetry at baseline reported more positive affect to the positive film clips, and those with more right-sided prefrontal asymmetry reported more negative affect in response to the negative film clips.

In a more recent study testing this hypothesis (Zald, Mattson & Pardo, 2002), 89 participants were studied using PET. All subjects retrospectively rated the extent to which they had experienced negative affect-related mood states over the previous month by using the PANAS. Results indicated that resting rCBF (activation) in the PFC correlates with self-reported ratings of negative affect during the month preceding the scan. It is important to note in this study that the negative affect ratings that correlated with PFC activity involved a retrospective rating of mood during the month before scanning. These authors contend that when measured over time period such as a month,

negative affect scores reflect an implicit aggregation of situations in which negative affect was elevated. Given the stability of such long-term ratings over time, these data suggest that they reflect trait-wise differences in the disposition to experience negative affect states. Findings in this, and previously mentioned studies, support the contention that individual differences in prefrontal asymmetry may, indeed, mark some aspect of a trait-like vulnerability to emotional stimuli, particularly negative affect (Davidson, 2000; 2003; Zald, Mattson & Pardo, 2002).

In Sum

Due to the complex nature and somewhat disparate strands of evidence gathered in this research thus far, a brief synopsis is provided. Evidence from the aforementioned research of emotion, indicate that prefrontal asymmetry is related to both state- and trait-like affect and approach- and withdrawal-related decision making, such that right-sided prefrontal asymmetry is associated with negative affect, a predisposition towards experiencing more negative (or less positive) affect, and a tendency to act in an 'avoidant' manner in response to both internal and external stimuli. Concurrently, left-sided prefrontal asymmetry is associated with positive affect, a predisposition towards experiencing more positive (less negative) affect, and a tendency to act in an 'approaching' manner in response to internal and external stimuli.

Some authors argue that there is an entanglement between the relationship between prefrontal asymmetry, affective valence, and motivational direction (Harmon-Jones, 2003a). These authors contend that prefrontal asymmetry is more so related to approach vs. withdrawal than positive and negative affective valence, and that these two

dimensions are distinct from one another. The preponderance of evidence, however, suggests that prefrontal asymmetry is significantly associated with both positive and negative affect and approach- and withdrawal - related decision making (see Davidson, 2003 for a review). Further, evidence suggests that these constructs are not only intimately connected, but represent two dimensions of the same system (see Miller & Cohen, 2001 for a review).

Directionality in the Relationship between Prefrontal Asymmetry and Affect

Although previous studies of prefrontal asymmetry are correlational in nature, infant research in this area has provided some insight into the potential directionality between asymmetrical activation and the aforementioned behavioral and affective correlates. In 1986, Fox & Davidson conducted a study to determine at what age differential lateralization for approach- and withdrawal-related emotions emerges. In a study with newborn infants (tested within the first 72 hours of life) who were presented with tastes differing in hedonic quality, right-sided prefrontal asymmetry was associated with the production of facial signs of disgust in response to tastes. Conversely, left-sided asymmetry was associated with the production of facial signs of interest in response to tastes (Fox & Davidson, 1986). From these data, it appears that differential anterior lateralization for emotion is present at birth.

Additionally, in a previously mentioned study that remains well-cited in this body of literature, Davidson & Fox (1989) found that 10-month old infant who cried in response to maternal separation were more likely to show right-sided prefrontal asymmetry, measured *before* the maternal separation challenge. Similarly, Calkins, Fox & Marshall (1996) showed that greater right-sided prefrontal asymmetry measured at 9-

months of age was related to negative affect and inhibited behavior measured at 14-months of age. It is important to note that although the temporal layout of these studies may imply that prefrontal asymmetry *causes* individual differences in subsequent behavior, these studies remain correlational and do not allow definitive conclusions to be drawn.

In an attempt to provide further insight into the potential causal role of frontal asymmetry, Allen, Harmon-Jones & Cavender (2001) conducted a study using biofeedback training designed to directly alter frontal brain asymmetry. Participants were randomly assigned to one of two conditions designed to increase either left- or right-sided frontal asymmetry. Subsequent self-reported affect, as well as facial expressions, in response to emotionally evocative film clips was measured. Results revealed that self-reported affect, as well as facial muscle activity, was altered in the direction of the biofeedback training. Participants trained to increase right-sided frontal asymmetry reported significantly less interest, amusement, and happiness when viewing a happy film than did participants trained to increase left-sided frontal asymmetry. In addition, participants trained to increase right-sided frontal asymmetry displayed significantly more "frown" facial expressions while viewing a sad film and significantly less "smile" facial expressions while viewing a happy film, as compared to participants trained to increase left-sided frontal asymmetry. Again, these results do not provide definitive evidence of a causal effect of frontal asymmetry on affect, however may provide some indirect support for such a contention. Also consistent with these implications are results from Robinson, Kubos, Starr, Rao & Price (1984) who found that lesions in the left prefrontal cortex are associated with depressive symptomatology in stroke patients.

Although the implications drawn from these findings are clear, additional research remains necessary to elucidate the causal mechanism in the relationship between prefrontal asymmetry and approach- and withdrawal- related affect.

Prefrontal Asymmetry and Binge Eating

To date, no study has examined the potential relationship between binge eating and brain asymmetry, however, relevant research may warrant such investigation. Binge eating in overweight persons has been extensively linked to negative affective states (e.g., Meyer and Waller, 1997) as well as trait-like negative affect (e.g., Henderson & Huon, 2002). There is also evidence implicating a possible relationship between binge eating in overweight individuals and a trait-like predisposition towards negative affective states (e.g., Wolff et al., 2000). Similarly, right-sided prefrontal asymmetry has been extensively linked to negative affective states (e.g., Davidson et al., 1990), trait-like negative affect (e.g., Tomarken et al., 1992), and there is evidence to suggest that individuals displaying right prefrontal asymmetry may also have a trait-like predisposition towards negative affective states (e.g., Wheeler et al., 1993).

Although this relationship has not been explicitly explored, one existing study has established a relationship between prefrontal asymmetry and eating behavior. In 2002, Silva, Pizzagalli, Larson, Jackson, & Davidson conducted a study testing the relationship between restrained normal weight participants (n = 23) and prefrontal asymmetry. Based on prior research suggesting that restrained normal weight individuals may be especially prone to experience negative emotions, particularly depression and anxiety (Sheppard-

Sawyer, McNalley & Fischer, 2000), these authors hypothesized that normal-weight restrained eaters would exhibit greater right-sided prefrontal asymmetry as compared to normal-weight unrestrained eaters (n = 32). All participants had taken part of a larger study of prefrontal asymmetry 1.5 to 2 years before being assessed for restrained eating in the present study. Additionally, all participants were assessed for depressive or anxious symptomatology using the Mood and Anxiety Symptom Questionnaire (MASQ; Watson, Clark, Weber, Assenheimer, Strauss & McCormick, 1995; Watson, Weber, Assenheimer, Clark, Strauss & McCormick, 1995). Scores on this measure were used to remove the variance of depressive or anxious symptomatology from the EEG data in order to test the relation between restraint eating and the levels of asymmetry independently of affective symptomatology at the time of the EEG evaluation. Results confirmed the proposed hypothesis and indicated that lower levels of restraint in normal weight individuals were significantly correlated with left-sided prefrontal asymmetry, while higher restraint scores were significantly correlated with right-sided prefrontal asymmetry.

The theory upon which the above Silva et al. (2002) study was based asserts that individuals 'disinhibit' their eating or binge in response to stress and/or negative affect because they are restraining their eating (Polivy & Herman, 1985). Based on evidence previously discussed evidence suggesting that restrained eaters disinhibit their eating following a preload, these authors suggest that dietary restraint is a precursor to binge eating. In previous research, however, it has been shown that *obese non-binge eaters* on diets tend to eat less, not more, following a preload (Lowe et al., 1991).

In 1992, McCann, Perri, Nezu & Lowe conducted an experiment testing the assertion that it is the dietary restraint that leads to binge eating. These authors investigated obese individuals seeking weight-loss treatment in order to capture a high degree of both dietary restraint and history of binge eating. As such, the authors theorized that this obese sample would show disinhibited eating following preload and measured their levels of dietary restraint, binge eating history, and body image disparagement in order to identify which variable(s) moderated the effect of preloads on eating. As predicted, those participants that drank the milkshake(s) did disinhibit their eating; an eating behavior widely viewed as an analogue of binge eating. The main finding from this study, however, was that level of dietary restraint was not significantly correlated with amount eaten. In fact, the *only* variable associated with disinhibited eating was history of binge eating. In this way, obese binge eaters showed the very same eating patterns in response to a preload as normal weight restrained eaters. These similarities suggest that obese binge eaters may show similar right-sided prefrontal asymmetry as did the normal weight restrained eaters in the Silva et al. (2002) study.

An additional interesting feature to note in the Silva et al. (2002) study was that the authors explicitly point out the possibility that the significant brain asymmetry found in restrained normal weight individuals in their sample may have actually been *caused* by their disordered eating (i.e., dietary restraint; p. 679). That is, the authors allude to the prospect that prefrontal asymmetry is potentially malleable and, thus, may have been seen as a result of the effects of eating patterns on brain circuitry in their sample.

Specific Aims

This research attempts to add to extant literature in the areas of human affect, electrophysiological brain activity, disordered eating, and obesity. This study was designed to examine the relationship between asymmetrical activation in the human brain and binge eating in overweight individuals, independent of affect at the time of assessment.

Hypotheses Tested

There is a significant body of literature relating frontal asymmetry and negative affect, as well as binge eating and negative affect. It has also been demonstrated that overweight individuals who binge eat show similar eating patterns as normal weight restrained eaters. On the basis of their documented sensitivity to negative affect, and the established relationship between frontal asymmetry in normal weight restrained eaters, it was hypothesized that participants high in binge eating would exhibit greater relative right-sided asymmetrical activity in the frontal cortex as compared to participants low in binge eating. It was also hypothesized that this relationship would remain significant after removing the variance accounted for by depressive or anxious symptomatology, as well as affective valence, at the time of assessment. Binge eating, examined as a dimensional variable (using scores on the BES), was also related to frontal asymmetry. It was hypothesized that higher scores on the BES would be associated with more right-sided frontal asymmetry and that this relationship would remain significant when controlling for affect at the time of assessment. In this way, this research tested the prediction that binge eating in overweight individuals is associated with right-sided prefrontal asymmetry independent of state affect. Parietal asymmetry was also examined

in order to test the specificity of frontal results. It was hypothesized that parietal asymmetry would not be significantly related to binge eating or affective measures.

CHAPTER 2: METHODS

Participants

26 female and 2 male participants were recruited from an NIH-supported grant study on weight-loss maintenance. In addition, 1 female and 1 male diagnosed with binge eating disorder (BED) were recruited directly from a binge eating study being conducted at the University of Pennsylvania Weight and Eating Disorder Center[1]. All participants were right-handed, overweight (but otherwise healthy) individuals ranging in age from 29 to 70 (M = 49, SD = 12) years. Participants ranged in BMI from 29.1 to 61.5 (M = 39.22, SD = 6.68) kg/m^2. The ethnic breakdown of the sample was as follows: 73% African American, 7% Caucasian, 7% more than one ethnicity, 3% Latino, and 3% unknown. Sample characteristics are illustrated in Table 1. Participants were compensated $50, $75, or $125 for completion of the study (due to increases in remuneration paid to participants in an attempt to increase recruitment). Approval for this study was granted from the Drexel University Medical Institutional Review Board.

Exclusion criteria for this study were as follows:

1. Current or history in the past ten years of bulimia nervosa or anorexia nervosa (Note: binge-eating disorder (BED) was an exclusionary criteria in the parent study limiting participants with diagnosed BED to those directly recruited for The University of Pennsylvania)
2. Current bi-polar disorder, major depressive episode, substance abuse or dependence disorder
3. A psychiatric disorder that affects body weight or energy expenditure
4. Current use of medications that affect body weight or energy expenditure (unless medication is long-term and dosage is unchanging – e.g., Synthroid)
5. Plans to leave the Philadelphia area within the next three years
6. Lactose-intolerance
7. Lactating, pregnant, or planning to become pregnant in next two years
8. A myocardial infarction within the past three months
9. Malignant arrhythmias

10. Unstable angina
11. Current or recent history of cancer, cerebrovascular, renal, or hepatic disease
12. Protein wasting disease (i.e., lupus, Cushing's syndrome)
13. Gouty attack within the past year
14. End-stage renal disease, indicated by creatinine greater than 1.8
15. Left handed dominance[2]
16. Open head wound
17. Skull defect
18. Learning disability or neurological disorder
19. Use of psychiatric medications
20. Use of recreational drugs or alcohol within 24 hours

[1]Note: BMI and Frontal asymmetry scores for participants recruited from the parent study (M = 39.214, SD = 6.919 and M = 0.039, SD = 0.083 respectively) were very similar to BMI and frontal asymmetry scores for participants recruited from The University of Pennsylvania (M = 39.350, SD = 0.070 and M = 0.030, SD = 0.089 respectively). As such, the two samples were combined in this study for a total N of 30 participants.

[2]Note: In keeping with previous research, only right-handed participants were included in this study due to evidence that patterns of hemispheric activation for cognitive functions may vary according to handedness (Byrden, 1982). Inter-individual differences in handedness may have been picked up, and subsequently skewed, EEG data.

Measures

Binge Eating Scale (BES):

The 16-item BES (Gormally, Black, Daston & Rardin, 1982) was designed specifically to identify binge eaters within and obese population (Celio, Wilfley, Crow, Mitchell & Walsh, 2004). The BES describes both behavioral manifestations (e.g., eating large amounts of food) and feelings and/or cognitions surrounding a binge episode (e.g., guilt, fear of being unable to stop eating). Empirical data shows that the Binge Eating Scale successfully discriminated among persons judged by trained interviewers to have no, moderate, or severe binge eating problems (Gormally et al., 1982). Scores > 27 on the BES are considered indicative of severe binge eating (Greeno, Marcus & Wing, 1995; Wheeler, Greiner & Boulton, 2005).

Edinburgh Handedness Inventory (EHI):

The EHI (Oldfield, 1971) has been shown to have good reliability and validity in the assessment of handedness. Additionally, this measure has been used in previous relevant research on brain asymmetry (e.g., Tomarken et al., 1992). An inventory was necessary for the assessment of handedness (in addition to asking participants) as some people may not be 'completely' right-handed. That is, individuals may write with his/her right hand and call themselves 'right-handed' while they typically throw balls and cut food with their lift hand. Due to the high potential for variability associated with handedness in spectral EEG analyses, such individuals may have confounded the data and would not have been included in analyses.

EEG Recording:

Shaw (2003), in his book *The Brain's Alpha Rhythms and the Mind* provides a current and comprehensible description of spectral EEG analyses conducted in this study. A summation of the information relevant to this work is provided. The source of the EEG is the complex network of nerve cells making up the outer layers of the brain- the cerebral cortex- and is the part of the brain involved in those aspects of behavior typically referred to as "higher mental activity". Electrical activity in this area is considered the neurological correlate of cognition: memory and learning, perception, emotion, reasoning, and decision making. As mentioned, hypotheses in this study surround electrical activity specific to the prefrontal region of the cerebral cortex. EEG recording measures alpha rhythms through electrodes fixed to the scalp with good electrical contact. In nearly all clinical investigations, electrodes are placed on the scalp in accordance with an internationally agreed upon standard called the '10-20 system' in which electrode positions are designated by specific letter/number combinations (Jasper, 1958). These electrodes connect to a visual display unit (VDU) through a particular type of amplifier displaying an electrical signal that fluctuates with time. An amplifier is necessary to measure the relatively small bioelectric brain signals on the surface of the scalp in the presence of larger, interfering electrical fields present in the surrounding environment. Alpha rhythms are then displayed on a VDU, enabling statistical analyses. Alpha rhythms are a particular type of electrical oscillation or "brain wave" generated through several potential sources; of relevance to this research are those generated by neuro-electrical activity in the PFC.

Alpha waves fluctuate in complete cycles of change lasting for approximately one tenth of a second and are most prominently detected in the healthy awake adult at rest. Electrical activity in EEG recording is measured as a difference of voltage, called as a "potential difference", between two electrodes though it is commonly referred to as "voltage" or "potential". An analogy can be drawn to describing the height of a mountain, which technically describes its difference in height with respect to sea level. As such, voltage in this study was measured in the PFC relative to an average of measurements taken form two electrodes placed behind each ear of the participant. These areas are used as reference points, because they generate a very low level of electrical activity, enabling more accurate detection of activity localized within the specific areas of interest. An additional note is that, when alpha waves are graphed, an upward deflection represents a negative change- opposite of typical standards seen in physics and engineering.

Although alpha waves are describes as rhythmical, they are not stationary (stable and predictable) like the regular function of a sine wave. An individual's alpha rhythm holds some unpredictable variation however, given constant conditions, an individual's EEG holds a fairly consistent pattern in terms of mean amplitude and the variance of the amplitude and these factors are considered stationary. Alpha rhythm mean amplitude is measured from peak to trough of the waves and is usually within 10-50 μV (microvolts). Alpha waves are defined in terms of average frequency; the number of complete cycles in one second; referred to as Hertz (Hz). By definition, alpha rhythms range between 8 and 13 Hz, inclusively. There do exist waves of other frequencies within the EEG, however alpha waves are the most dominant EEG activity and are those examined in nearly all

prior research in this area. Alpha waves are traditionally thought to reflect synchronous neuronal activity associated with areas of the cortex that are not processing information (at rest). Within this line of reason, when an area of the cortex is involved in processing information (activated), this synchronicity is lost and the distribution on the scalp changes. Because alpha amplitude in the area of investigation reduces or blocks information processing, a reduction of alpha levels (amplitude) in a particular region is taken to indicate increased activation in that area relative to other areas. Although some theorists have suggested that this model may be overly simplistic, it forms the basis for all prior research in this area and so was adhered to in this protocol as well.

In this study, asymmetrical activation was quantified by measuring alpha amplitude in the right hemisphere (of the PFC), relative to alpha amplitude in the left hemisphere. Greater alpha amplitude in the right hemisphere, relative to alpha amplitude in the left hemisphere, represents greater left-sided asymmetrical activation. Concurrently, greater relative alpha amplitude in the left hemisphere represents greater relative activation in the right hemisphere. Therefore, right-sided asymmetry reflects relatively greater activation (decreased alpha amplitude) in the right hemisphere while left-sided asymmetry reflects relatively greater activation (decreased alpha amplitude) in the left hemisphere.

Height:

A standard physician stadiometer was used to measure height. Height measurement was used in order to calculate BMI.

Mood and Anxiety Symptom Questionnaire (MASQ) Anhedonic Depression and Anxious Arousal subscales:

Clark and Watson (1991) proposed a tripartite model of anxiety and depression defined in terms of common symptoms relating to general distress, anxiety-specific symptoms of hyperarousal, and depression-specific symptoms of low positive affect/loss of interest. In order to aid the measurement of and discrimination between anxiety and depression, they developed the MASQ. Participants are asked to rate 60 items[3] on a 5-point Likert scale from "not at all" to "extremely" in accordance to how much they have experienced each item "during the past week, including today". In an examination of its psychometric properties, the MASQ Anxious Arousal and Anhedonic sub-scales were shown to reliably discriminate between anxiety and depression and showed high levels of convergent validity (Reidy & Keogh, 1997). This measure has also been used in the study of prefrontal asymmetry and restrained eating (Silva et al., 2002) with which this research attempted to align itself. Data from the anxious arousal and anhedonic depression subscales from this measure were used in statistical analyses in order to control for the variance accounted for by depressive or anxious symptomatology.

[3]Note: due to IRB constraints, one question from each subscale asking about suicide or death was removed. Each participant's average of scores on that subscale was entered in for that item during analyses.

Positive and Negative Affect Schedule (PANAS):

The PANAS (Watson, Clark & Tellegen, 1988) consist of 20 words describing emotions: 10 positive and 10 negative. Participants were asked to rate each word to indicate "to what extent you feel this way at the present moment". This state version of the PANAS was used to enable a control for affective state at the time of testing in examining the relationship between binge eating and asymmetry Participants rated their affect on a five-point rating scale ranging from 'very slightly' or 'not at all' to 'extremely'. Each list of 10 emotions constitutes a positive and negative mood scale. The scales are shown to be highly internally consistent, largely uncorrelated, and stable at appropriate levels over a 2-month time period. Both scales of the PANAS also demonstrate good convergent and discriminant validity (Watson, Clark & Tellegen, 1988).

Weight:

Weight was measured in street clothes using a standardized Secca® scale accurate to 0.1 kg. Weight measurement was used in order to calculate BMI.

Post Hoc Measures

The Power of Food Scale (PFS):

The 21-item version of the PFS (Lowe, Butryn, Didie, Annunziato, Crerand, Ochner, Coletta & Halford, 2006) is a self-report measure designed to assess psychological reactions to the food environment. Items on the scale are statements such as "I find myself thinking about food even when I'm not physically hungry" to which the

person responds on a 5-point Likert scale from "don't agree at all" to "strongly agree". The PFS has been found to be internally consistent and temporally stable, as well as demonstrating good convergent and discriminant validity (Lowe et al., 2006). Validations studies of the PFS suggest it may reflect global level of appetitive responsiveness to the food environment (Lowe et al., 2006).

Three Factor Eating Questionnaire (TFEQ) Disinhibition and Hunger subscales:

The TFEQ (Stunkard & Messick, 1985) is a self-report questionnaire designed to assess three aspects of eating behavior: cognitive restraint, disinhibition, and hunger. The TFEQ Disinhibition subscale is designed to assess overeating that occurs after exposure to various cognitive, social, and emotional triggers. Higher scores on the TFEQ Disinhibition subscale are associated with increased eating and degree of overweight (Westenhoefer, 1991; Williamson et al., 1995). The TFEQ Cognitive Restraint subscale is designed to measure the tendency to consciously restrict food intake either to prevent weight gain or to promote weight loss by control over energy intake or types of food eaten. The TFEQ has demonstrated good psychometric properties, and the Disinhibition and Cognitive Restraint subscales have demonstrated adequate internal consistency (Stunkard & Messick, 1985; Laessle, Tuschl, Kotthaus, & Pirke, 1989).

Power Analysis and Sample Size

Although there are suggestions on methodology for conducting power analyses with fMRI data, these techniques have not yet been made applicable to EEG data (Desmond & Glover, 2002; Murphy & Garavan, 2004). There currently exist no set

criteria for conducting power analyses in neurophysiological studies utilizing EEG recordings. This is mainly due to the fact that this field of study is young enough that researchers are yet to determine what effect sizes to expect from such investigations. Within this arena, effect size itself is not as concrete a dimension as it is in most traditional behavioral studies. It could very well be that a diminutive effect in neurological activity is associated with a large cognitive or behavioral correlate. In turn, substantial neurological effects may only correlate with small changes in cognition or behavior. As such, formal power analyses are traditionally not performed in neurophysiological studies (Kounios, personal communication).

Hagemann et al. (2002) attempted to compile an exhaustive list of studies of resting frontal EEG asymmetry and affective and/or motivational correlates, available in the past two decades (1980 - 2000). These authors present a list of 33 studies and their respective sample sizes. With one outlier removed (N = 197), the mean sample for studies in the past two decades was 39 participants (SD = 26.71). The total N of 30 in this study was less than one standard deviation below the mean sample size in past related studies.

Note: Through the summer and fall of 2005, approximately 20 participants were recruited, however less than half of those participants yielded usable data for this study. Numerous participants were compensated and dismissed from the study after being unable to secure acceptable sensor connections to the scalp. Initial hypotheses for the difficulty ranged from too much perspiration in summer months to too much natural oil on the scalp of a predominantly obese African American sample. Through several

months of crippled recruitment, equipment failure proved to be the culprit. Unfortunately, it was not immediately apparent which piece of equipment was malfunctioning. Further data loss ensued as different pieces of equipment were replaced in a forced process-of-elimination method of resolution. Full recruitment resumed in February 2006 with usable data from only 10 participants. As such, planned follow up analyses became impossible. Aggressive recruitment and increases in remuneration through June 2006 resulted in a total N of 30. This included 2 participants diagnosed with binge eating disorder who were gained through several months of direct recruitment from the University of Pennsylvania Weight and Eating Disorder Center in an attempt to increase the number of participants in the binge eating group.

Procedure

Participants were recruited from either a larger NIH-supported study of weight-loss maintenance or a study of binge eating conducted at The University of Pennsylvania Weight and Eating Disorder Center. The specific research hypothesis of the study was withheld from participants due to the sensitive nature of the EEG recordings, as results may have been skewed. Participants were scheduled for an assessment in the EEG laboratory at Drexel University where each individual was consented and handedness was confirmed by administering the Edinburgh Handedness Inventory. No participants were determined to be left-hand dominant at the assessment. Before collecting EEG data, each participant filled out the MASQ and PANAS state version. Resting EEG recordings were then be collected by trained lab technicians. Detailed EEG methodology follows in the next section. Following completion of EEG recording, participants were

compensated and dismissed. Research hypotheses were then tested as specified in the proceeding design section.

EEG Recording

For means of consistency and comparison, EEG methodology closely aligned with that used by Silva et al. (2002) in their study of frontal brain asymmetry in restrained eaters. EEG measures were recorded using a lycra stretchable cap with 128 imbedded electrodes (Electro-Cap International, Inc.). EEG sensors were applied using the standard 10-20 system and referenced to an average of two electrodes, placed behind each ear. Data was collected during eight sixty-second trials, four with eyes open and four with eyes closed, presented in counterbalanced order. Sensor impedances were kept below 20,000 Ohms. All EEG data was collected using a sample rate of 256 Hz and bandpass filtered at 0.02 - 100 Hz. EEG was amplified 20,000 times using the MICROAMPS™ data acquisition system (SAM Technology, Inc.). EEG signals were then digitized using the MANSCAN® data analysis system (SAM Technology, Inc.). Automatic artifact detection, followed by visual inspection was used to remove artifact due to eye blinks, gross muscle activity, and movement. Artifact-free epochs of data were extracted through a Hanning window. A Fast Fourier Transform was applied to all extracted data that are four seconds in duration, with epochs overlapping 50 percent. Power density was then computed for the alpha band by summing power values across each 1-Hz bin within a band and dividing by the number of bins. Mean alpha power was computed separately for eyes-open and eyes-closed trials, weighted by the number of available artifact-free epochs. A mean of alpha power for eyes open and closed was then

computed. Finally, all power density values were log transformed to normalize the distribution of the data.

Prior research indicates that the frontal sites F3 and F4 are reliably related to dimensions of approach- and withdrawal-related emotion (Wheeler et at., 1993; Davidson, 2003). Parietal sites P3 and P4 have been shown not to covary with these affect-related dimensions and were thus used as control sites used to test the specificity of frontal results (Sutton & Davidson, 1997; Silva et al., 2002). On the basis of prior research (Silva et al., 2002), these four sites were selected a priori to test hypotheses regarding relations between EEG asymmetry and binge eating. All asymmetry scores were calculated by subtracting the log-transformed power density value in the alpha band for the left side from that of the right side (i.e., log F4 – log F3 and log P4 – log P3). Positive asymmetry scores thus reflect greater left-sided activity (associated with greater alpha band power density on the right, relative to the left, side). Conversely, negative asymmetry scores reflect greater right-sided activity.

Note: Because an asymmetry ratio does not reveal the actual amount of activation present in particular brain regions (i.e., the left PFC independent of the right PFC), the absolute magnitude of cortical activation was also calculated in the frontal and parietal cortices. A priori predictions, however, only involved testing the research hypotheses in this study (asymmetry scores) at the aforementioned frontal sites.

Design and Analyses

Binge eating was assessed in the parent study using the Binge Eating Scale (BES). In order to examine binge eating as a categorical variable, participants were grouped in either “low binge” or “high binge” categories. Participants in the low binge category scored in the lowest tertile (0 - 21) of the BES. Participants in the high binge category either scored in the highest tertile ($\geq$ 28) of the BES or were diagnosed with Binge Eating Disorder (BED; directly recruited from the University of Pennsylvania Weight and Eating Disorder Center). There were two such BED participants. Wheeler et al. (2005) note that "Scores [on the BES] higher than 27 strongly suggest the presence of a BED" (p.117). Binge group categorization yielded 9 participants in the low binge group, 12 participants in the high binge group, and 9 participants excluded from group analyses.

Asymmetry scores were analyzed in the prefrontal and parietal cortices. Asymmetry scores were calculated by subtracting the log-transformed power density value in the alpha band for the left hemisphere site (frontal; F3 or parietal; P3) from that of the corresponding right hemisphere site (frontal; F4 or parietal; P4). Asymmetry scores in the PFC were calculated (log F4 – log F3), while asymmetry scores in the parietal cortex were calculated (log P4 – log P3). Therefore, positive asymmetry scores reflect greater left-sided activity (i.e., greater alpha band power density on the right than on the left). Asymmetry scores in the parietal cortex were entered into analyses as a control to test the specificity of frontal results.

In order to assess affect at the time of measurement, all participants were administered the MASQ and the state version of the PANAS. Scores on these measures were to be used in order to remove the variance in asymmetry accounted for by affect at

the time of measurement in evaluating the relationship between binge eating and frontal asymmetry. Independent samples t-tests were used to identify significant differences between groups (low binge vs. high binge) on both measures. No a priori hypotheses were formulated about group differences in state affect.

Note: The state (vs. trait) version of the PANAS was administered in concordance with the methodology used by Silva et al. (2002) in order to better evaluate the main hypothesis in this study. The goal of this research was not to confirm the relationship between trait affect and asymmetry but to evaluate the relationship between binge eating and asymmetry independent of state affect. Administration of both versions would likely have yielded invalid responding; the only difference between the two measures being that the instructions specify for the participant to respond according to how they feel "right now" vs. "generally" for the state and trait versions (respectively). Thus, responses on the first version administered would likely have influenced responses on the second version. Counterbalancing for order would not have alleviated this issue. As such, the state version of the PANAS was determined to be the more appropriate option.

In order to examine group differences in asymmetry, A 2 X 2 mixed-model analysis of variance (ANOVA) was performed on prefrontal asymmetry scores. Binge eating group (low binge vs. high binge) was the between groups variable and brain region (frontal vs. parietal) the within groups variable. This analysis tested for main effects of binge eating and brain region on asymmetry scores, as well as a binge eating Group X Region interaction. The main hypothesis in this study predicted a main effect of Group,

qualified by a Group X Region interaction reflecting binge group differences in frontal asymmetry only. The above ANOVA was then repeated controlling for MASQ and PANAS subscale scores, both individually and simultaneously (2 X 2 mixed-model ANCOVAs). It was predicted that (if found) the main effect of group and interaction would remain significant indicating a relationship between binge eating and asymmetry controlling for affect at the time of measurement.

In order to test for differential activity in the left vs. right frontal hemisphere, a 2 X 2 mixed-model ANOVA was conducted with binge eating group and frontal hemisphere (log F3 vs. log F4) as the between and within groups variables (respectively). These analyses were also repeated for parietal sites (log P3 vs. log P4). A priori hypotheses predicted a Group X Hemisphere interaction in frontal analyses only such that the high binge group would show a significant pattern of right, greater than left, frontal activity.

Pearson correlations were then used to test relationships between BES (as a dimensional variable), MASQ, PANAS, and asymmetry scores. A priori hypotheses predicted a significant negative correlation between binge eating severity (measured by BES scores) and frontal asymmetry scores indicating that higher scores on the BES would be related to right-sided frontal asymmetry. It was also predicted that this relationship would remain significant when controlling for affect at the time of assessment. No a priori predictions were made regarding the relationships between binge eating severity and state affect, or state affect and asymmetry.

Post hoc analyses

21 participants (mean BMI=38.53, SD=5.93) completed the Power of Food Scale (PFS). Additionally, 25 participants (mean BMI=39.43, SD=7.21) completed the Three-Factor Eating Questionnaire (TFEQ). By definition, no a priori hypotheses were formulated for post hoc analyses. Independent samples t-tests were used to examine binge eating group differences on the PFS and TFEQ subscales. Pearson correlations were calculated to examine the relationships between scores on the BES, PFS, and TFEQ subscales. Correlational analyses were then used to test the relationships between PFS, TFEQ, affective measures, and asymmetry scores. A partial correlation analysis was used to test the relationship between scores on the PFS and asymmetry scores while controlling for state affect. Finally, stepwise regression analyses, with scores on all measures entered, were performed in order to determine the best model for predicting asymmetry scores.

Note key points when reviewing results: 1. alpha power level is considered to be inhibitory of activation, therefore, lower (or more negative) alpha power levels indicate greater activation in a particular area of the brain; 2. Asymmetry scores were calculated so that positive scores indicate greater activation in the left-hemisphere relative to activation in the right hemisphere (greater alpha power levels in the right hemisphere relative to the left); 3. Conversely, negative asymmetry scores are indicative of greater activation in the right (relative to the left) hemisphere; 4. A negative correlation between a measure and left frontal (or parietal) asymmetry is equivalent to a positive correlation between that measure and right frontal (or parietal) asymmetry; 5. On all tables, “Left”

frontal or parietal asymmetry indicates greater relative activation in the left hemisphere (also referred to as “left-sided asymmetrical activation” or “left-to-right asymmetrical activation”); 6. According to the approach- and withdrawal- related affect model, left-sided frontal asymmetry is theorized to be associated with positive or approach-related affect; and 7. Conversely, right-sided frontal asymmetry is theorized to be associated with negative or withdrawal-related affect.

CHAPTER 3: RESULTS

Mean (M) and standard deviation (SD) values were calculated for scores on the MASQ Anhedonic Depression and Anxious Arousal subscales, the PANAS Positive and Negative Affect subscales, the BES, and asymmetry scores in the frontal and parietal regions. Descriptive statistics are reported in Table 2.

Comparisons by Binge Eating:

As described, participants were divided into low binge vs. high binge eating groups according to scores on the BES (0 -21 vs. > 28) with two BED participants included in the high binge group. Independent samples t-tests were used to examine binge eating group differences across all measures. No significant differences were found between groups on the MASQ Anhedonic Depression subscale scores ($p = 0.349$). On the MASQ Anxious Arousal subscale, however, participants in the high binge group scored significantly higher than did participants in the low binge group ($t(19) = -2.194$, $p = 0.041$). Group differences on both the PANAS Positive and Negative Affect subscale scores were nonsignificant ($p = 0.240$ and $p = 0.560$ respectively). Mean age and BMI were very similar across groups. As such, age and BMI were not entered into analyses as controls in group comparisons. Data on group differences is reported in Table 3. These results show that participants in the high binge group reported more anxious arousal at the time of assessment than did participants in the low binge group.

Analyses were then performed to test the main hypothesis that participants in the high binge group would demonstrate more right-sided frontal asymmetrical activation as

compared to participants in the low binge group. Group differences in asymmetrical activation were examined by region using a 2 X 2 mixed-model analysis of variance (ANOVA), with binge group (low vs. high) as the between-groups variable and brain region (anterior vs. parietal) as the repeated measures variable, performed on the asymmetry scores (log F4 - log F3; log P4 - log P3). This analysis revealed no main effects of group ($p = 0.699$) or region ($p = 0.250$), and no interaction ($p = 0.615$). Analyses were repeated controlling for MASQ and PANAS scores (individually and simultaneously) with no significant effect on results.

To test for differential activity over left or right frontal regions, a 2 X 2 mixed-model ANOVA on log alpha power values with group and hemisphere (log F3 vs. log F4) as the between and within groups factors was performed. The only significant effect emerging was that of hemisphere ($F(1, 19) = 4.890$, $p = 0.039$) with lower alpha power levels (more activation) in the left frontal hemisphere. There was no main effect of group ($p = 0.104$) and no interaction ($p = 0.303$). This analysis was repeated comparing Group X Hemisphere differences in parietal sites (log P3 vs. log P4) revealing no main effects and no interaction. Similarly, a 2 X 2 repeated measures ANOVA comparing region (frontal vs. parietal) and hemisphere (right vs. left) yielded no main effects and no interaction. Participants in the high binge group did show lower alpha (more activation) across all sites, however, this relationship failed to reach significance (no main effect of group in frontal or parietal analyses). These results reveal a significant difference in hemispheric activation in the frontal cortex (only), however, this difference was not dependant upon binge eating group.

Binge eating was also examined dimensionally, using scores on the BES (excluding BED participants). BES scores were not related to MASQ Anhedonic Depression or Anxious Arousal subscale scores ($p = 0.305$ and $p = 0.300$ respectively). BES scores were not related to PANAS Positive or Negative affect scores ($p = 0.129$ and $p = 0.996$ respectively). Finally, correlational analyses were used to test the relationship between binge eating severity and asymmetrical activation. BES scores were not related to frontal or parietal asymmetry scores ($p = 0.410$ and $p = 0.861$ respectively). Pearson correlations are reported in Table 4. Correlational analyses were repeated with BMI, age, and affective measures entered as controls (individually and simultaneously) with no significant change in results (not shown).

Comparisons by Asymmetrical Activation:

Correlations between frontal asymmetry scores and both MASQ subscale scores were nonsignificant (both $ps > 0.38$), as were correlations between parietal asymmetry scores and MASQ subscale scores (both $ps > 0.72$). Correlations between asymmetry scores at both regions and both PANAS scale scores were all nonsignificant (all $ps \geq 0.440$). Detailed results are reported in Table 5. Analyses were repeated controlling for BMI with no significant change in results (not shown). Therefore, no relationship was found between self-reported affect at the time of assessment and asymmetrical activation.

Note: in order to address the possibility that the log transformation of the EEG data may have suppressed asymmetry values, all analyses were repeated using the raw alpha power values (F3 – F4; P3 – P4) with no significant change in results (not shown). Analyses

were also repeated using asymmetry ratio (F3/F4; P3/P4) as opposed to differential asymmetry (F3 – F4; P3 – P4) with no significant change in results (not shown).

Post hoc analyses:

Mean and SD values were calculated for scores on the Power of Food Scale (PFS) and Three Factor Eating Questionnaire (TFEQ) Disinhibition and Cognitive Restraint subscales. Descriptive statistics are reported in Table 6.

Comparisons by Binge Eating:

Independent samples t-tests were used to examine between groups (low binge vs. high binge) comparisons across post hoc measures. Participants in the high binge group scored significantly higher on the PFS ($t(15) = -2.786$, $p = 0.014$) and the TFEQ Disinhibition Subscale ($t(16) = -3.217$, $p = 0.005$) than did participants in the low binge group. No group differences were found on the TFEQ cognitive restraint subscale ($p = 0.339$). Detailed results are reported in Table 7.

Examined dimensionally, scores on the BES (excluding BED participants) were significantly correlated with scores on the PFS ($r = 0.601$, $p = 0.005$) and TFEQ Disinhibition subscale scores ($r = 0.546$, $p = 0.005$). BES scores were not correlated with TFEQ Cognitive Restraint subscale scores ($p = 0.488$). Detailed results are reported in Table 8. Analyses were repeated controlling for BMI with no significant difference in results (not shown).

Comparisons by Asymmetrical Activation:

Left-sided frontal asymmetry was significantly correlated with PFS scores ($r = 0.543$, $p = 0.011$). Frontal asymmetry was not related to TFEQ Disinhibition or Cognitive Restraint subscale scores ($p = 0.231$ and $p = 0.356$ respectively). Detailed results are reported in Table 9. Analyses were repeated controlling for BMI with the only change in results being an increase in the strength of the relationship between PFS scores and left-sided frontal asymmetry ($p = 0.005$). Detailed results are reported in Table 10. The only affective measure significantly correlated with the PFS was the PANAS Positive Affect subscale ($r = -0.487$, $p = 0.025$). This inverse correlation demonstrates that higher PFS scores were associated with lower PANAS Positive Affect subscale scores. The PFS remained significantly correlated with left-sided frontal asymmetry when controlling for PANAS Positive Affect subscale scores ($r = 0.635$, $p = 0.003$), as well as when controlling for all affective measures simultaneously ($r = 0.644$, $p = 0.005$). A stepwise regression analysis with all self-report measures (and BMI) entered revealed that the best model for predicting frontal asymmetry was the inclusion of only the PFS ($p = 0.008$) and BMI ($p = 0.049$), accounting for 46% of the variance. Detailed results are reported in Table 11. These results suggest a moderate to strong relationship between PFS scores and left-sided frontal asymmetrical activation. Higher scores on the PFS were also associated with less positive affect at the time of measurement. However, affect at the time of assessment did not mediate the relationship between PFS scores and frontal asymmetry.

Post hoc analyses were repeated with parietal asymmetry scores in order to test the specificity of frontal results. Surprisingly, right-sided parietal asymmetry was

correlated with left-sided frontal asymmetry ($r = 0.754$, $p < 0.001$). Right-sided parietal asymmetry was also correlated with PFS scores ($r = 0.560$, $p = 0.008$). Parietal asymmetry was not related to TFEQ Disinhibition or Cognitive Restraint subscale scores ($p = 0.183$ and $p = 0.487$ respectively). Detailed results are reported in Table 9. Controlling for BMI only increased the strength of the relationship between PFS scores and right-sided parietal asymmetry ($p = 0.004$). Detailed results are reported in Table 10. The PFS remained significantly correlated with right-sided parietal asymmetry when controlling for PANAS Positive Affect subscale scores ($r = 0.520$, $p = 0.019$), as well as when controlling for all affective measures simultaneously ($r = 0.542$, $p = 0.025$). With all affective measures entered as controls, the strength of the relationship between the PFS and parietal asymmetry increased ($r = 0.644$, $p = 0.005$). A second stepwise regression analysis revealed that the best model for predicting parietal asymmetry was the PFS alone ($p = 0.017$), accounting for 31% of the variance. Detailed results are reported in Table 12. Results from parietal analyses revealed a moderate to strong relationship between PFS scores and right-sided parietal asymmetrical activation. This relationship was also not mediated by affect at the time of measurement. Due to the fact that parietal asymmetry was analyzed only to test the specificity of frontal results, no a priori hypotheses were formulated for analyses of parietal asymmetry. Potential interpretations of parietal results will be discussed in the proceeding section.

CHAPTER 4: DISCUSSION

This study was devised to test the relationship between binge eating in overweight individuals and asymmetrical activation in the frontal cortex, independent of affect at the time of assessment. The design of the study attempted to mirror methodology used by Silva et al. (2002), who found a relationship between restrained eating in normal weight individuals and right-sided frontal asymmetry. These authors stated; "Our data imply that extreme right-sided prefrontal asymmetry may represent a diathesis that increases an individual's vulnerability to a pattern of restrained eating and possibly to other eating disorders such as bulimia, for example." (p. 678). The main hypothesis in this study drew on past literature relating restrained eating in normal weight individuals to binge eating in overweight individuals (e.g., McCann et al., 1992); binge eating to negative affect (e.g., Wolff et al., 2000); and negative affect to right-sided frontal asymmetry (e.g., Davidson, 2003). It was hypothesized that overweight participants high in binge eating would show significantly more right-sided asymmetrical activation in the frontal cortex as compared to participants low in binge eating. As in the Silva et al. (2002) study, the main hypothesis in this study was predicated upon the theoretical relationship between disturbed eating and affect, as well as the theoretical relationship between affect and frontal asymmetry. In both studies, it was also hypothesized that the relationship between the disturbed eating and frontal asymmetry would remain after removing the variance accounted for by affective state at the time of assessment. Results in this study did not support the main hypotheses; potential causes for the lack of support for this hypothesis are described below.

In examining affective data in this study, it should first be noted that scores indicated a restriction in range in anxious and negative affective variance. Exclusion criteria of the parent study included current major depressive disorder or any other psychiatric disorder that might affect body weight. As such, it was anticipated that our sample would contain few individuals with *extreme* affective disturbance, however, a fair range of general negative affect and anxiety was anticipated. Data from this sample did not meet this expectation. Total possible scores on the MASQ Anxious Arousal subscale range from 17 to 85. However, frequency calculations indicated that over 90% of our sample scored below 26. Similarly, total possible scores on the PANAS Negative Affect subscale range from 10 to 50. The mean score from our sample was 11.48 with a SD of only 1.55. Scores on the Anhedonic Depression and Positive Affect subscales of the MASQ and PANAS (respectively) did reflect sufficient variance to covary with strong or moderate affective correlates. There is evidence to suggest that African American women (constituting over 70% of the sample) who binge eat report significantly less psychological distress than Caucasian women who binge eat (see Pike, Dohm, Striegel-Moore, Wilfley & Fairburn, 2001). Although the racial composition of the sample may have influenced the results in this study, an examination of cultural differences on affective measures was not possible due to the limited number of non-African American participants.

Binge Eating and Affect:

The first association discussed is that of the relationship between binge eating and negative affect. The majority of the literature connecting negative affect to binge eating is expressed either in terms of trait affect (Henderson & Huon, 2002; Wolff et al., 2000; Yanovski, 1993; Cargill et al., 1999; Pinaquy et al., 2003) or state affect directly preceding a binge (Agras & Telch, 1998; Henderson & Huon, 2002; Arnow et al., 1992; 1995; Heatherton & Baumeister, 1991; Lingswiler et al., 1989; Ruderman, 1985; Davis et al., 1988). Although the proposed relationship between binge eating and asymmetry may have been mediated by affect, the main goal of this research was to test the prediction that there would be a relationship between binge eating and asymmetry while removing the variance accounted for by affect at the time of assessment. Therefore, affect in this study was measured mainly in state (vs. trait) form, in order to mirror the methodology used by Silva et al. (2002) described previously. There is evidence that binge eaters report significantly more pervasive stress, depression, anger, and self-blame than obese non-bingers (Wolff et al., 2000). However, it would have been a theoretical leap to hypothesize a relationship between state affect and binge eating when a binge could not follow affective assessment (i.e., participants knew they would be observed for the next two to three hours and not given access to food). Thus, the results in this study allow for inferences to be made about the relationship between binge eating and state affect, not preceding a binge, only.

Despite the potential restriction in range, participants in the high binge group did score significantly higher on the MASQ Anxious Arousal subscale (see Table 3). Examined dimensionally, however, no significant relationships were found between

binge eating and affective measures (see Table 4). Although no a priori hypotheses were formed relating scores on the BES and affective measures, the directionality of nonsignificance was examined in terms of the theory relating binge eating to negative affect. Results revealed a nonsignificant trend in the direction of more negative affect in relation to higher scores on the BES. Thus, the results in this study lend no direct support for the theoretical framework relating binge eating to negative affect, however the direction of nonsignificance appears generally consistent with this theory.

Affect and Asymmetry:

The second, more theoretical, association discussed is that of the relationship between affect and frontal asymmetry. As mentioned, the foundational framework for the proposed relationship between binge eating and asymmetry was, in large part, based on the theoretical association between affect and asymmetry. As the goal was to use state affect as a control variable, this research did not specifically aim to test the relationship between affect and asymmetry. Although there is literature linking state affect and frontal asymmetry (e.g., Davidson et al., 1990; Wheeler et al., 1993; Davidson & Irwin, 1999; Davidson, Marshall, Tomarken, & Henriques, 2000; Davidson, 2003), these studies all use mood induction techniques to invoke positive or negative affect. Several of these studies, as well as the Silva et al. (2002) study, used baseline state affect (i.e., in the absence of a mood induction) as a control and found no relationship between state affect and asymmetry. These results suggest that the proposed relationship between state affect and frontal asymmetry may depend upon whether or not it was induced. Thus, results in this study provide the ability to make inferences about the relationship between non-

induced state affect and asymmetry only. Associations between affective measures and asymmetry did not approach significance for any subscale (see Table 5). Despite there being no a priori hypothesis, the directionality of nonsignificance was examined for consistency with the theoretical model linking trait, or experimentally-induced, negative affect and right-sided frontal asymmetry. Results revealed a nonsignificant trend in the direction of more negative affect at the time of assessment in relation to right-sided frontal asymmetry. Although non-induced state affect has been shown not to be related to asymmetry (Harmon-Jones & Allen, 1997; Davidson et al., 2000; Silva et al., 2002), the direction of the nonsignificance appeared consistent with the theory relating trait, and induced state, affect to frontal asymmetry.

Binge Eating and Asymmetry:

Finally, the central proposed association of this study was that of a relationship between binge eating and right-sided frontal asymmetry. Results in this study revealed no binge eating group differences in asymmetrical activation. As described, participants were grouped according to scores on the BES. Analyses, however, revealed a potential restriction in range across scores on the BES in addition to the affective measures mentioned. Prior research examining scores on the BES as a categorical variable has used cutoffs scores of < 17 to indicate no binge eating and scores of > 27 to indicate severe binge eating or BED (Wheeler et al., 2005; Greeno et al., 1995; Brody, Walsh & Devlin, 1994; Wilson, Rossiter, E., Kleifield & Lindholm, 1986; Celio, Wilfley, Crow, Mitchell & Walsh, 2004). Gormally et al. (1982) published normative data for binge eaters and non binge eaters on the BES (see Table 13) upon which these cutoffs are

based. According to this data, scores ranging from 12.4 to 14.9 were associated with no binge eating. Scores ranging from 17.5 to 22 were associated with moderate binge eating, and scores ranging from 26 to 34 were associated with severe binge eating. It was the original intent of this study to compare overweight individuals with no binge eating to individuals with severe binge eating. In our sample, twelve participants scored over 27 on the BES, however, only one participant scored below 17. That is, only one participant would have been grouped in a "no binge eating" category according to BES normative data. As opposed to group comparisons between participants who do not binge eat and participants who do, group comparisons in this study may have been between limited to moderate vs. severe binge eaters. Although this restriction of range in binge eating may have been a contributory factor, it is unlikely to have accounted for the failure to reject the null hypothesis in this study. Examined both categorically and dimensionally, binge eating was not significantly associated with left-sided asymmetry ($p = 0.30$ and $p = 0.41$ respectively). Furthermore, the direction of the nonsignificant findings between binge eating and frontal asymmetry is opposite that predicted by the main hypothesis in this study.

Post Hoc Discussion:

Post hoc analyses were conducted in an attempt to gain more insight into potential correlates of binge eating and asymmetrical activation. Data from the Power of Food Scale (PFS) and Three-Factor Eating Questionnaire (TFEQ) Cognitive Restraint and Disinhibition subscales was examined. Evidence suggests that PFS scores reflect an individual's global level of appetitive responsiveness to the food environment, and has

been shown to be a better predictor of various self-report measures of overeating than the Restraint Scale (Herman & Polivy, 1980) used in the Silva et al. (2002) study (Lowe et al., 2006). Furthermore, PFS scores are much more strongly related to eating than to BMI (Lowe et al., 2006). Disinhibition measures types of overeating that are typically less extreme than binge eating (Lowe, personal communication). Additionally, there is evidence to suggest that the Restraint Scale may have actually been measuring disinhibition more so than cognitive restraint (see Stunkard & Messick, 1984). Cognitive restraint measured by the TFEQ is a measure of an individual's conscious control of intake, independent of disinhibition (Stunkard & Messick, 1984).

Post hoc results revealed that binge eating, measured both categorically and dimensionally, was significantly related to scores on the PFS and TFEQ Disinhibition subscale. Participants in the high binge group scored significantly higher on the PFS and TFEQ Disinhibition subscale. Similarly, scores on the BES were positively correlated with scores on the PFS and Disinhibition subscale (see Tables 7 & 8). No relationship was found between binge eating and cognitive restraint. Therefore, as compared to individuals low in binge eating, individuals high in binge eating reported greater levels of appetitive responsiveness to the food environment and disinhibited eating, but not conscious restriction of their intake. These results are consistent with those found in previous literature (Marcus & Wing, 1983; Lowe et al., 2006).

In relation to asymmetry, a significant correlation was found between PFS scores and left-sided frontal asymmetry. Asymmetrical activation was not related to scores on either TFEQ subscale. Therefore, post hoc analyses reveal that left-sided frontal asymmetry was related to appetitive responsiveness but not overeating or conscious

control of eating. Scores on the PFS were also negatively correlated with PANAS Positive Affect subscale scores, indicating that higher scores on the PFS were associated with lower positive state affect. Taken together, scores on the PFS were associated with lower positive affect and left-sided frontal asymmetry. The relationship between PFC scores and left-frontal asymmetry remained after removing the variance in asymmetry accounted for by Positive Affect subscale scores. In addition, a regression analysis including all variables revealed that the most variance in frontal asymmetry was accounted for by PFS scores and BMI.

The relationship between PFS scores and asymmetry was not found to be specific to the frontal cortex; a significant relationship was found between PFS scores and right-sided parietal asymmetry. Heller (1990) proposed a theory of parietal asymmetry asserting that greater right-sided parietal asymmetry is associated with states of elevated emotional arousal, irrespective of the specific emotional valence. It should be noted, however, that parietal data in this study was analyzed as a control only and lend no support for any particular theory of asymmetry. Although parietal asymmetry has often been used as a control to test the specificity of the relationship between frontal asymmetry and affect, several studies have reported significant findings in the parietal cortex (Heller, Etienne & Miller, 1995; Heller, Nitschke & Lindsay, 1997; Hagemann et al., 1998; Wacker et al., 2003). The relationship between PFS scores and both frontal and parietal asymmetry warrants further exploratory analyses in order to identify activation in additional brain regions that may be associated with PFS scores. Although theories of asymmetry in other areas of the brain may not yet be developed, such data might assist

future research in identifying additional areas of the brain that vary in activation in relation to appetitive responsiveness.

Finally, a significant inverse relationship was also found in this study between frontal and parietal asymmetry. Although unanticipated, other studies have also noted the same findings (Davidson, Schaffer & Saron, 1985; Henriques & Davidson, 1990; Schmidt & Fox, 1994). Henriques and Davidson (1990) have argued that individuals who display a pattern of right frontal activation with left parietal activation may have deficits in social skills compared with individuals who display the opposite pattern. However, more research is needed to test the replicability of parietal results, and how such findings may be related to affect or behavior.

A Closer Examination of Models of Frontal Asymmetry:

Despite the fact that inferences could not be made based upon the affective data in this study, post hoc results inspired a reexamination of the approach- and withdrawal-related affect model of asymmetrical activation proposed by Davidson (1995; 2000; 2003). As this model predicts more right-sided activation in relation to lower positive affect (see Tomarken et al., 1992), the findings that PFS scores were associated with lower positive affect and left-sided asymmetry appeared discrepant. Although the most prominent model of frontal asymmetry in current literature, it has been noted that the approach- and withdrawal- related affect model of frontal asymmetry (affect model) is still relatively young, and host to discordant results (Harmon-Jones, 2003a; Wacker, Heldmann & Stemmler, 2003; Nimmo-Smith & Lawrence, 2003; Harmon-Jones, Lueck, Fearn & Harmon-Jones, 2006). In light this information, coupled with a nonsignificant

trend between binge eating and frontal asymmetry opposite to that hypothesized, a re-review of the literature on frontal asymmetry and the affect model was conducted. A number of discrepant findings are reviewed below.

In 1983, Schaffer, Davidson & Saron found a significant differences in frontal activation between depressed and nondepressed individuals (measured by scores on the BDI), such that depressed individuals showed significantly less left-sided frontal activation. In 1991, Henriques & Davidson replicated these findings comparing clinically depressed individuals with healthy control subjects. However, Reid, Duke and Allan (1998) designed two studies specifically to replicate these results, and found no significant differences between depressed and nondepressed individuals in frontal activation. Tomarken et al. (1992) found a significant relationship between trait positive and negative affect (measured by the trait version of the PANAS) and left- and right-sided frontal asymmetry (respectively). In 1997, Sutton and Davidson failed to replicate this finding, and reported no relationship between trait PANAS scores and asymmetry. Several studies described in the introduction have noted a relationship between experimentally-induced mood and frontal asymmetry (e.g., Davidson et al., 1993; Davidson et al., 2000). In 1998, however, Hagemann, Naumann, Becker, Maier & Bartussek demonstrated that, depending on the particular analysis procedure, there were associations between frontal asymmetry and affectivity in line with the published findings, opponent to those findings, or no relation between frontal asymmetry and affective reactivity. Perhaps the most troubling are results from a meta-analysis of 106 PET and fMRI studies conducted by Murphy, Nimmo-Smith and Lawrence in 2003. These authors reported that, across all studies included in the meta-analysis, the spatial

distributions associated with positive and negative emotions did not differ significantly, and greater left-sided asymmetry was not found in the analysis of positive emotions. These authors did report a greater left-sided asymmetry for approach-related emotions, although the pattern was not restricted to frontal brain regions. In addition, asymmetry was not found to vary as a function of withdrawal-related emotions.

In light of inconsistent results noted in the asymmetry literature, Harmon-Jones and colleagues proposed that affective valence and approach-withdrawal tendencies were two correlated but distinct constructs (Harmon-Jones & Allen, 1997; Harmon-Jones, 2003a; 2004). These authors attempted to rectify discrepant results by suggesting that frontal brain asymmetry was due to motivational direction (approach vs. withdrawal) irrespective of associated affect. Evidence for this theory was obtained by demonstrating that anger, a negative but approach-related emotion, was related to left-sided frontal asymmetry (Harmon-Jones & Sigelman, 2001; Harmon-Jones, 2003a; 2003b; 2004). The motivational direction model, however, may not be adequate to unify the literature on asymmetry. Due to the correlation between positive (negative) affect and approach (withdrawal) motivational direction (Davidson, 2003), relatively few studies exist that allow for direct comparisons of the two models. In 1992, Sobotka, Davidson & Senulis performed a study independently manipulating motivational direction (approach vs. withdrawal) and valence (reward vs. punishment), while measuring both affective response to trials (self-report) and EEG asymmetry. Participants either pressed a button (approach) or released a button (withdraw) in order to gain (monetary) reward or avoid punishment (losing money). All participants gave either approach or withdrawal responses for equal halves of the experiment (counterbalanced for order). A

reward/punishment signal before each trial denoted whether they had the opportunity to gain or lose money. Trials were either; reward, where the participant could gain money or have no change; or punishment, where participants could either lose money or have no change. Following the reward/punishment signal, participants were required to respond to a cue (either press or depress a button) within a limited amount of time to gain, or avoid losing, money. The authors found significantly greater left-sided frontal activation across both rewarding and punishing trials, and that asymmetry did not depend on motivational direction (approach vs. withdrawal) of response. The authors also found that the greater the left-sided frontal activation during the task, the more intense was the positive affect reported in response to the feedback stimulus which denoted winning. Contrary to their initial hypothesis, however, no significant relation was found between frontal asymmetry and negative affect for the trials where participants lost money. Findings from this study are moderately consistent with the affect model and inconsistent with the motivational direction (approach vs. withdrawal) theory of frontal asymmetry.

In 2003, Wacker et al. designed a study of asymmetry using vignettes of personal relevance to participants (i.e., soccer-related scenarios with all participants being active soccer players) to induce either anger or fear. In each vignette, the protagonist could either approach or withdraw (e.g., confront the coach for pulling them from the championship game, or leave the field) and each participant was asked how they would respond to the given scenario. Following this assessment, the final piece of the vignette was revealed with the protagonist either approaching or withdrawing from the feared or anger-inducing stimulus. The compatibility of the participant's individual goals (approach vs. withdraw) with the course of action taken by the protagonist in the vignette

was used as an index of goal-conflict. Thus, 'action agreement' between the protagonist in the vignette and subject was taken to indicate low goal-conflict. Independent affect ratings confirmed that the vignettes were successful in eliciting the desired (negative) emotions (i.e. anger in the vignette where the protagonist was pulled from the championship game or fear in the vignette where a known better player approaches the protagonist in an attempt to steal the ball). Results in this study revealed that induced anger was significantly correlated with left-sided frontal asymmetry irrespective of activated motivational direction (whether the subject reported they would approach or withdraw). Results also revealed a significant relationship between goal-conflict and frontal asymmetry such that action agreement was significantly correlated with left-sided frontal asymmetry. More importantly, the relationship between goal-conflict and frontal asymmetry did not depend on affect (as both conditions were negative emotion inducing) or motivational direction (approach vs. withdrawal). The authors in this study also note that induced fear elicited greater approach ratings than did induced anger, while anger was associated with greater left-sided frontal asymmetry. These findings are at odds with both the motivational direction and affect models.

Several papers have been written in an attempt to address the conundrums found across the frontal asymmetry literature (e.g., Davidson, 1998b; Hagemann et al., 1998; Harmon-Jones 2003a). Researchers have been striving to improve methodologies and adjust the main theories of asymmetry in order to rectify discrepant results (Canli & Amin, 2002; Hagemann et al., 1998; Wacker et al., 2003). Two particular constructs that have appeared to be consistently related to asymmetry are those of Gray's (1994; Gray & McNaughton, 2000) behavioral inhibition system (BIS) and behavioral activation system

(BAS). Both Harmon-Jones (2003a) and Davidson (2003) have used these constructs in support of their respective theories of asymmetrical brain activation. A brief description of the BIS-BAS theory is provided below.

The BIS-BAS Theory of Frontal Asymmetry:

Gray (1994) and Gray and McNaughton (2000) proposed a theoretical framework for the control of motivated behavior involving three core systems; the behavioral activation system (BAS), fight-flight freezing system (FFFS), and the behavioral inhibition system (BIS). The BAS serves to activate goal-directed behavior. It mediates approach behavior and is engaged by stimuli signaling reward or safety (omission of punishment). The FFFS also serves to activate goal-directed behavior and mediates avoidance behavior. The FFFS is activated by stimuli signaling punishment or frustrating nonreward. The third system, the BIS, is engaged whenever there is a conflict between competing alternatives. That is, when it is unclear which behavior will results in the best, or least bad, outcome. The BIS serves to inhibit on-going behavior in an attempt to further assess behavioral options before acting. The BIS does this by increasing arousal and vigilance in order to allow the individual to choose the best, or least bad, option. This conflict can arise when there are competing good alternatives (approach – approach conflict), a reason for approaching a threatening stimuli (approach – avoid conflict), or competing bad or threatening alternatives (avoid – avoid conflict). Any *confliction* in how to act in order to achieve a particular goal (either gain reward or escape punishment) is proposed to engage the *BIS*, resulting in an *inhibition* of on-going action and a more careful consideration of competing options. Conversely, stimuli presenting clear

opportunity for reward or safety, *without conflict*, are proposed to engage the *BAS*. This BAS activation results in non-conflicted/on-going or *motivated action* either towards an appetitive goal, or towards safety (away from aversive stimuli).

Within this theory, different systems can be engaged independently or concurrently. For example, the BIS can be engaged without concurrent FFFS activation during a conflict between two equal appetitive goals (approach-approach). However, it is assumed that the BIS is typically activated with concurrent FFFS activation as the majority of goal conflicts experienced are assumed to be approach-avoid (when we are required to approach danger or threat in order to obtain reward). In the BIS-BAS model, the FFFS system has been removed due to the rarity of pure FFFS activation in modern times. No longer do individuals have to run from saber-tooth tigers, but have to go to work and deal with a boss in order to earn a paycheck. The elimination of the FFFS system, however, has led to a misinterpretation of Gray's theory in assuming the BIS and BAS are orthogonal dimensions (Carver & White, 1994; Wacker et al., 2003; McNaughton & Gray, 2000; McNaughton & Corr, 2004). That is, due to the fact that the BAS is proposed to govern approach to reward or safety, it is presumed to be associated only with appetitive or rewarding goals. Conversely, because the BIS is typically activated with the FFFS in mediating goal-conflict most often arising from the need to approach a threat, it is presumed to be associated only with aversive stimuli. Proponents of both the approach- and withdrawal- related affect (i.e., Davidson, 2003) and motivational direction (approach-withdrawal; i.e., Harmon-Jones, 2003a) models of asymmetry have incorporated the BIS-BAS framework into their respective work. Davidson uses evidence relating the BIS-BAS model to asymmetry as evidence in

support of his affect model (Davidson, 2003; Sutton & Davidson, 1997). Similarly, Harmon-Jones uses evidence relating the BIS-BAS model to asymmetry as evidence in support of his motivational-direction model (Harmon-Jones, 2003b; Harmon-Jones & Allan, 1997). Both Sutton and Davidson (1997) and Harmon-Jones and Allan (1997) used the BIS/BAS scales, designed by Carver & White (1994), and demonstrate that the BAS is significantly related to left-sided frontal asymmetry, while the BIS is significantly related to right-sided frontal asymmetry, while the BAS is significantly related to left-sided frontal asymmetry. The presupposition of both interpretations of the BIS-BAS model is that the BIS regulates only negative affect or withdraw tendencies, while the BAS regulates only positive affect or approach tendencies. These interpretations both misconstrue the essential delineation between BIS and BAS as negative/withdraw vs. positive/approach (respectively), when it is actually goal-conflict vs. no goal-conflict (respectively).

The BIS-BAS model referred to in the proceeding discussion is a framework for interpreting asymmetrical activation that this author feels better represents Gray's (1994; Gray & McNaughton, 2000) theory of motivated behavior. A review of the basic tenets of this theory is provided: The BIS is responsible for inhibiting on-going goal-directed action when there is goal-conflict (i.e., competing alternatives), *irrespective of motivational direction (approach or withdraw) or affective valence (appetitive or aversive),* in order to better evaluate options. The BAS is responsible for on-going goal-directed behavior in the absence of goal-conflict, irrespective of direction or valence. The BIS-BAS model contends that individuals not only experience more behavioral inhibition or activation in response to stimuli, but also have trait-like tendencies towards

behavioral inhibition or activation in general (i.e., more apt to behavioral activation as compared to behavioral inhibition). Thus, individuals higher in BIS tendencies would be more likely to be conflicted and think longer about how to act (irrespective of direction), and individuals with higher relative BAS tendencies would be more likely to act decisively (irrespective of direction). For illustrative purposes, a crude example of an individual high in BIS tendencies might be someone who can never make up their mind and struggles with every little decision they have to make. The BIS-BAS model would predict that this individual would show right-sided frontal asymmetry. Conversely, an individual high in BAS tendencies might be someone who is always very decisive in their decisions and is very quick to act. This individual would be predicted to show left-sided frontal asymmetry.

Reevaluation of Theoretical Models of Asymmetry:

Unfortunately, neither the dimensions of approach/withdrawal independent of affect, nor BIS-BAS were included in the aforementioned meta-analysis by Murphy et al. (2003). Wacker et al. (2003) suggest that the BIS-BAS model may better account for some of the discrepancies seen in the asymmetry literature. In this study, described above, these authors demonstrate that goal-conflict (as measured by agreement with vignette protagonist) was related to frontal asymmetry independent of both affect and motivational direction. Similarly, Sobotka et al. (1992) demonstrated that participants, required to act quickly with no competing stimuli (i.e., no conflict), showed more left-sided frontal activation independent of motivational direction or valence (reward or punishment). More explicit measures of BIS-BAS and trait affect would, however, be

necessary for direct inferences to be made. If a relationship was found between asymmetry and BIS-BAS, and that relationship was mediated by trait positive and negative affect, it would provide support for the affect model. Alternatively, if trait affect was found not to mediate the relationship between asymmetry and BIS-BAS, it would indicate that asymmetry may be associated with the BIS-BAS construct more so than approach- and withdrawal- related affect.

In the aforementioned article, Sutton and Davidson (1997) collect data using the BIS/BAS scales and the trait version of the PANAS, in an attempt to demonstrate consistency between the BIS-BAS and affect models of asymmetry. Results revealed significant relationships between BIS-BAS and right- and left- sided frontal asymmetry (respectively). The authors state that frontal asymmetry in their study "better predicted the BIS-BAS construct than the PA-NA construct" (p. 209). Similarly, when referencing the findings in later work, Davidson (2003) states "Behavioral Inhibition and Behavioral Activation were even more strongly predicted by electrophysiological measures of prefrontal asymmetry than were scores on the [trait] PANAS scales." (p. 658). The Sutton & Davidson (1997) publication, however, reveals that *no* relationship was found between frontal asymmetry and either positive or negative trait affect, or the relative strength of positive vs. negative trait affect (all ps > 0.18). In their conclusions, Sutton and Davidson write; "The findings support the hypothesized role of lateralalized prefrontal systems in approach and withdrawal (or inhibitory) motivational tendencies…" (p. 209). Thus, the authors insert "motivational tendencies" in place of "affect"; however, they fail to address the implication that the BIS-BAS and affect models of asymmetry may not be synonymous. According to the Baron and Kenny (1983)

mediational model, the failure to find a relationship between state affect and asymmetry in this study indicates that state affect did not mediate the relationship between BIS-BAS and asymmetry. Thus, these findings suggest that the BIS-BAS and affect models may be distinct, and that asymmetry may be more related to BIS-BAS than approach- and withdrawal- related affect.

Reinterpretation of Results:

Taking into account results found in this study, as well as evidence suggesting that the BIS-BAS model may better account for frontal asymmetry results (Sutton & Davidson, 1997; Wacker et al., 2003), a reinterpretation of relevant previous research (i.e., Silva et al., 2002) is presented in conjunction with results obtained in this study and other current work in this area (i.e., Coletta, Platek, Mohammed & Lowe, 2006). Silva et al. (2002) found that normal weight restrained eaters (measured by the Restraint Scale; Herman & Polivy, 1980) showed greater right-sided frontal asymmetry. These authors also note that unrestrained eaters showed the opposite pattern, however, this effect failed to reach significance. Utilizing the BIS-BAS model of interpreting asymmetry results, these results would suggest that restrained eaters at rest display more goal-conflict tendencies. That is, when not presented with a disinhibiting stimulus (e.g., ingesting a "forbidden food" or receiving a preload), restrained normal weight individuals have a tendency to inhibit goal-directed behavior. This interpretation is consistent with the basic tenet of restraint theory that suggests that restrained eaters at rest are over-inhibited (Herman & Mack, 1975; Herman & Polivy, 1975; 1980). According to results found by Coletta et al. (2006), frontal asymmetry in restrained eaters does not change upon visual

cues of palatable foods, which would be predicted according to the BIS-BAS model as restrained eaters are postulated to inhibit their eating until they actually ingest palatable food or are "preloaded". Following a preload and presentation of appetitive stimuli, restraint theory dictates that these individuals become overly behaviorally activated, or disinhibited, in their eating (Herman & Mack, 1975, Herman & Polivy, 1980; Ruderman & Christensen, 1983; Ruderman, 1985). The BIS-BAS model would then predict greater left-sided asymmetry once restrained eaters received a preload and are presented with appetitive stimuli. The recent study by Coletta et al. (2006) obtained data commensurate with this prediction using fMRI. That is, following preload, restrained eaters displayed a shift towards left-sided asymmetry when exposed to highly palatable food cues. According to the BIS-BAS model, these results indicated that restrained eaters at rest show more inhibition of goal-directed behavior before preload, and greater motivation to obtain a (food) reward following preload. These results would not have been predicted by the affect model of asymmetry, as negative affect following preload has not been shown to decrease in restrained eaters (Herman & Mack, 1975; Herman & Polivy, 1975; 1980). In addition, there is evidence to suggest that a preload, or eating a forbidden food, may be an anxiety-provoking event for restrained eaters (Rogers & Hill, 1989; Heatherington & Macdiarmid, 1993). Thus, the affective model would have predicted continued, or even stronger, right-sided activation following preload.

No clear speculations can be made on whether the BIS-BAS model would predict a relationship between binge eating and frontal asymmetry at rest. Following preload, however, McCann et al. (1992) demonstrated that obese binge eaters become disinhibited or over-activated in their eating. Thus, upon exposure to preload and food cues, the BIS-

BAS theory would likely predict that binge eaters should display an increase in left-sided frontal activation similar to that found in normal weight restrained eaters (Coletta, Platek, Mohammed & Lowe, 2006). In a study of overweight binge eaters, Karhunen, Vanninen, Kuikka, Lappalainen, Tihonen & Uusitupa (2000) examined changes in regional cerebral blood flow (rCBF; taken as an indicator of activation) during exposure to food using PET. Results from this study did indicated that, as compared to non binge eaters, obese binge eaters displayed marked increase in rCBF in the left frontal regions upon exposure to food. This speculation, however, does not differentiate between models as there is evidence suggesting short term mood-enhancing effects of food in binge eaters (Arnow et al., 1992).

The main analyses in this study provide no evidence in support of either model of asymmetry as no significant relationships were found between affect and asymmetry. In addition, the directionality of nonsignificant trends between state affect and asymmetry was consistent with both models. This is not surprising given that BAS and BIS have been related to approach- and withdrawal- related state affect, respectively (Harmon-Jones & Allen, 1997). Post hoc results, however, may be better accounted for by the BIS-BAS model of asymmetry. As mentioned, PFS scores are thought to indicate global level of appetitive responsiveness to the food environment (Lowe et al., 2006). Although speculative, there may be an association between a tendency towards "incentive-motivated goal-directed action" (BAS; Wacker et al., 2003; p. 168) and appetitive responsiveness which can be defined as how readily a person responds to a strong urge or desire, especially one for food or drink. This hypothesis is supported by Dawe and colleagues (Dawe, Gullo & Loxton, 2004; Dawe and Loxton, 2004) who conducted two

reviews of eating disorder and substance abuse literature. These reviews illustrated a consistent relationship between the BAS scale and both binge eating and substance abuse. These authors also contend that the BAS Drive subscale[4] is a clear measure of appetitive motivation. In addition, a recent study conducted by Beaver, Lawrence, van Ditzhuijzen, Davis, Woods & Calder (2006) demonstrated that individual differences in BAS Drive subscale scores were very highly correlated ($r = 0.77$) with left-sided activation in the frontal cortex following exposure to images of appetizing food. These authors concluded that; "Individuals high in this [reward sensitivity] trait experience more frequent and intense food cravings and are more likely to be overweight or develop eating disorders associated with excessive food intake" (p. 5160). It should, however, be noted that these results were not limited to the left hemisphere or frontal cortex only; following exposure to appetizing food images, BAS Drive scores were highly correlated with activation in other areas of the brain implicated in reward (i.e. right ventral striatum, left amygdala, substantia nigra, and left ventral pallidum).

[4]Note: the BAS scale is comprised of three subscales; Reward Responsiveness, designed to measure positive responses to the occurrence or anticipation of reward; Fun Seeking, designed to measure desire for new rewards and a willingness to approach a potentially rewarding stimulus; and Drive, designed to measure the persistent pursuance of desired goals (Carver & White, 1994). It is also important to note that the BIS/BAS scales were developed on the basis of an older conception of BIS and BAS, where BIS was originally conceived of as activated only in response to aversive stimuli and the BAS activated only in response to rewarding stimuli (Gray, 1981; 1982). Thus, the BAS Drive subscale most

closely approximates the BAS of the BIS-BAS theory of frontal asymmetry described in the preceding section, as it is not confounded with items assessing affect or motivational direction. Interpretations of all three subscales, however, remain speculative (Carver & White, 1994; Carver, 2004).

Finally, although the BIS-BAS theory of asymmetrical activation may appear more consistent with post hoc results, no direct inferences can be made on the basis of the unanticipated post hoc relationship between PFS scores and left-sided asymmetry. Theoretical connections between appetitive responsiveness and behavioral activation can be drawn, however, results in this study provide no evidence in support of this supposition. Future research (specifying a priori hypotheses) is necessary before making inferences about what construct frontal asymmetry is actually tapping, and whether this construct is related to appetitive responsiveness as measured by the PFS.

Limitations:

Several weakness of this study should be noted. Difficulties in recruitment resulted in a low sample size. This limited the statistical power of analyses conducted, particularly for analyses examining individual differences. In addition, 27 out of 28 participants recruited from the parent study reported moderate to severe binge eating (as measured by BES scores); therefore, this sample may have constituted an unusually high percentage of individuals with a history of at least moderate binge eating. Participants also reported what may be an unusually low amount of anxiety and negative affect, which may or may not have been affected by the large percentage of African American

participants in this sample. The unanticipated restriction in range across these variables reduced the ability to detect an effect in this study if one existed. Moreover, these issues severely restrict the generalizability of results from this study.

Significant results obtained from this study were mainly post hoc, centering on the PFS and asymmetry. Due to the correlational nature of these results, there is no way to determine the nature of the apparent relationship between asymmetrical activation and appetitive responsiveness. Although these results may inspire future research, they do not provide the ability to make inferences about overweight individuals who binge eat as compared to overweight individuals who do not binge eat, as was the original intent. Finally, post hoc results considered in conjunction with other related literature, suggest that there may have been a fundamental flaw in the theoretical foundation upon which this research was predicated. Data in this study, however, does not provide the opportunity to make direct inferences about models of asymmetrical activation. Future research should include measures of both state and trait affect, as well as the BIS-BAS scale in order to enable comparisons between models. As these measures were not included in this study, all suggestions in favor of the BIS-BAS model in this study are purely speculative in nature.

Conclusion:

Results in this study did not support the main hypothesis that, as compared to overweight individuals low in binge eating, individuals high in binge eating would display more right-sided asymmetrical activation in the frontal cortex. Post hoc analyses revealed a strong relationship between left-sided frontal asymmetry and right-sided

parietal asymmetry. Significant relationships were also found between appetitive responsiveness (measured by the PFS), left-sided frontal asymmetry, and right-sided parietal asymmetry. PFS scores were inversely correlated with positive affect, yet positively correlated with left-sided frontal asymmetry, calling into question the approach- and withdrawal- related affect theory of frontal asymmetry. Data from this, and other related research, suggest that the BIS-BAS model of frontal asymmetry may be considered independent of other models, and may better account for frontal asymmetry findings. Results from related research were reinterpreted using the BIS-BAS model with no inconsistencies. This reinterpretation, however, does not account for the failure to find a relationship between binge eating and frontal asymmetry in this study. It remains possible that there is no relationship between asymmetrical brain activity and binge eating, regardless of theoretical model used. Findings in this study do, however, encourage greater exploration into the potential role of asymmetrical activation in eating behavior. In particular, there may be a strong relationship between left-sided frontal asymmetry, the behavioral activation system, and appetitive responsiveness as measured by the PFS. Future research may be able to test these predictions, as well as providing support for a much-needed unifying model of asymmetrical brain activation.

Table 1. Sample Characteristics

Gender	Male	Female		
	3	27		
Variable	**Minimum**	**Maximum**	**Mean**	**SD**
Age	29	70	49.23	12.29
BMI	29.1	61.5	39.22	6.68
Ethnicity	**Percentage**			
African American	73%			
Caucasian	7%			
Latino	3%			
> 1 Ethnicity	7%			
unknown	3%			

Table 2. Descriptive Statistics across Primary Measures

Measure	N	Min	Max	M	SD
MASQ Anhed Dep	30	32.43	80.62	53	11.34
MASQ Anx Arous	30	18	35	21.86	3.81
PANAS Pos Affect	29	15	49	34.17	7.77
PANAS Neg Affect	29	10	16	11.48	1.55
BES	26	16	40	25.38	6.41
Left Frontal Asym	30	-0.074	0.342	0.387	0.082
Left Parietal Asym	30	-0.529	0.274	0.011	0.168

Table 3. Binge Eating Group Differences across Primary Measures

Measure	Group	N	M	SD	t-value	df	Sig.
BMI	Low	9	38.589	4.624	0.181	19	0.858
	High	12	38.092	7.169			
MASQ Anx Arous	Low	9	20.007	2.783	-2.194*	19	0.041*
	High	12	22.516	2.445			
MASQ Anhed Dep	Low	9	50.471	9.908	-0.960	19	0.349
	High	12	54.516	9.290			
PANAS Pos	Low	9	36.440	6.635	1.214	19	0.240
	High	12	32.080	9.090			
PANAS Neg	Low	9	11.670	1.936	0.593	19	0.560
	High	12	11.250	1.288			
Left Frontal Asym	Low	9	0.022	0.015	-1.059	19	0.303
	High	12	0.062	0.112			
Left Parietal Asym	Low	9	0.017	0.081	0.230	19	0.820
	High	12	0.036	0.233			

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 4. Pearson Correlations between Primary Measures and BES scores

Measure	N	r-value	p-value
MASQ Anhed Dep	30	0.209	0.305
MASQ Anx Arous	30	0.211	0.300
PANAS Pos Affect	29	-0.312	0.129
PANAS Neg Affect	29	-0.001	0.996
Left Frontal Asym	30	0.169	0.410
Left Parietal Asym	30	-0.036	0.861
BMI	27	-0.149	0.468

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 5. Pearson Correlations between Primary Measures and Asymmetrical Activation by Region

		Left Frontal		Left Parietal	
Measure	N	r-value	p-value	r-value	p-value
MASQ Anhed Dep	30	-0.166	0.381	-0.046	0.807
MASQ Anx Arous	30	-.051	0.791	0.066	0.728
PANAS Pos Affect	29	0.087	0.655	0.097	0.618
PANAS Neg Affect	29	-0.031	0.874	-0.149	0.440
BES	26	0.169	0.410	-0.036	0.861
BMI	27	0.206	0.302	-0.249	0.210

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 6. Descriptive Statistics across Post Hoc Measures

Measure	N	Min	Max	M	SD
PFS	21	21	89	36.76	15.81
TFEQ Disinhibit	25	0	18	3.88	3.81
TFEQ Cog Restraint	24	16	40	26.21	5.7

Table 7. Binge Eating Group Differences across Post Hoc Measures

Measure	Group	N	M	SD	t-value	df	Sig.
PFS	Low	7	26.000	2.449	-2.786	15	0.014*
	High	10	45.80	18.510			
TFEQ Disinhibit	Low	9	1.890	1.269	-3.217	16	0.005**
	High	9	7.110	4.702			
TFEQ Cog Restr	Low	9	25.556	5.918	0.987	15	0.339
	High	8	23.375	2.066			

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 8. Pearson Correlations between Post Hoc Measures and BES scores

Measure	N	r-value	p-value
PFS	20	0.601**	0.005**
TFEQ Disinhibit	25	0.546**	0.005**
TFEQ Cog Restraint	24	-0.204	0.339

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 9. Pearson Correlations between Post Hoc Measures and Asymmetrical Activation by Region

		Left Frontal		Left Parietal	
Measure	N	r-value	p-value	r-value	p-value
PFS	20	0.543*	0.011*	-0.560**	0.008**
TFEQ Disinhibit	25	0.248	0.231	-0.276	0.183
TFEQ Cog Restraint	24	-0.197	0.356	0.149	0.487
Left Frontal Asym	30	1	-	-0.754**	0.000**
Left Parietal Asym	30	-0.754**	0.000**	1	-

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 10. Pearson Correlations between Post Hoc Measures and Asymmetrical Activation by Region while Controlling for BMI

		Left Frontal		Left Parietal	
Measure	df	r-value	p-value	r-value	p-value
PFS	17	0.602**	0.005**	-0.618**	0.004**
TFEQ Disinhibit	22	0.273	0.197	-0.308	0.143
TFEQ Cog Restraint	21	-0.200	0.360	0.153	0.487
Left Frontal Asym	24	1	-	-0.774**	0.000**
Left Parietal Asym	24	-0.744**	0.000**	1	-

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 11. Stepwise Regression Coefficients Predicting Left Frontal Asymmetry

Measure	B	Std. Error	Beta	t-value	p-value
PFS	0.003	0.001	0.576	3.032**	0.008**
BMI	0.006	0.003	0.407	2.140*	0.049*

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 12. Stepwise Regression Coefficients Predicting Left Parietal Asymmetry

Measure	B	Std. Error	Beta	t-value	p-value
PFS	-0.007	0.003	-0.555	-2.672*	0.017*

* significant at $p < 0.05$
** significant at $p < 0.01$
Note: all tests for significance were 2-tailed

Table 13. Gormally et al. (1982) Normative Data for BES Scores

Table 1. Binge eating scale scores for the three severity levels

	Level of Severity					
	None		Moderate		Severe	
Sample	*M*	*S.D.*	*M*	*S.D.*	*M*	*S.D.*
1	14.9	8.2	19.6	6.7	28.9	7.5
(n = 65)	(n = 11)		(n = 40)		(n = 14)	
2 Total	13.4	5.2	21.1	7.0	31.3	6.6
(n = 47)	(n = 14)		(n = 21)		(n = 12)	
Males	14.4	4.9	17.5	4.1	26.0	8.0
(n = 15)	(n = 7)		(n = 4)		(n = 4)	
Females	12.4	5.6	22.0	7.4	34.0	3.9
(n = 32)	(n = 7)		(n = 17)		(n = 8)	

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APPENDIX A: BINGE EATING SCALE

Instruction: Below are groups of numbered statements. Read all of the statements in each group, and mark on this sheet the one that best describes the way you feel about the problems you have controlling your eating behavior. (Choose one statement per group)

#1

(1) I don't feel self-conscious about my weight or body size when I'm with others.
(2) I feel concerned about how I look to others, but it normally does not make me feel disappointed with myself.
(3) I do get self-conscious about my appearance and weight which makes me feel disappointed in myself.
(4) I feel very self-conscious about my weight and frequently, I feel intense shame and disgust for myself. I try to avoid social contacts because of my self-consciousness.

#2

(1) I don't have any difficulty eating slowly in the proper manner.
(2) Although I seem to "gobble down" foods, I don't end up feeling stuffed because of eating too much.
(3) At times, I tend to eat quickly, and then I feel uncomfortably full afterwards.
(4) I have the habit of bolting down my food, without really chewing it. When this happens I usually feel uncomfortably stuffed because I've eaten too much.

#3

(1) I feel capable to control my eating urges when I want to.
(2) I feel like I have failed to control my eating more than the average person.
(3) I feel utterly helpless when it comes to feeling in control of my eating urges.
(4) Because I feel so helpless about controlling my eating I have become very desperate about trying to get in control.

#4

(1) I don't have the habit of eating when I'm bored
(2) I sometimes eat when I'm bored, but often I'm able to "get busy" and get my mind off food.
(3) I have a regular habit of eating when I'm bored, but occasionally, I can use some other activity to get my mind off eating.
(4) I have a strong habit of eating when I'm bored. Nothing seems to help me break the habit.

#5

(1) I'm usually physically hungry when I eat something.
(2) Occasionally, I eat something on impulse even though I am not hungry.
(3) I have the regular habit of eating foods, which I might not really enjoy, to satisfy a hunger feeling, even though physically I don't really need the food.
(4) Even though I'm not physically hungry, I get a hungry feeling in my mouth that only seems to be satisfied when I eat a food, like a sandwich, that fills my mouth. Sometimes, when I eat the food to satisfy my mouth hunger, then I spit the food out so I won't gain weight.

#6

(1) I don't feel any guilt or self-hate after I overeat.
(2) After I overeat, occasionally I feel guilt or self-hate.
(3) Almost all the time I experience strong guilt or self-hate after I overeat.

#7

(1) I don't lose control of my eating when dieting even after periods when I overeat.
(2) Sometimes when I eat a "forbidden food" on a diet, I feel like I "blew it" and eat even more.
(3) Frequently, I have the habit of saying to myself, "I've blown it now, why not go all the way" when I overeat on a diet. When that happens I eat even more.
(4) I have a regular habit of starting strict diets for myself, but I break the diets by going on an eating binge. My life seems to be either a "feast" or "famine".

#8

(1) I rarely eat so much food that I feel uncomfortably stuffed afterwards.
(2) Usually about once a month, I eat such a quantity of food that I end up feeling very stuffed.
(3) I have regular periods during the month when I eat large amounts of food, either at mealtime or at snacks.
(4) I eat so much food that I regularly feel quite uncomfortable after eating and sometimes a bit nauseous.

#9

(1) My level of calorie intake does not go up very high or go down very low on a regular basis.
(2) Sometimes after I overeat, I will try to reduce my caloric intake to almost nothing to compensate for the excess calories I've eaten.
(3) I have a regular habit of overeating during night. It seems that my routine is not be hungry in the morning but overeat in the evening.
(4) In my adult years, I have had week-long periods where I practically starve myself. This follows periods when I overeat. It seems like I live a life of either "feast or famine".

#10

(1) I usually am able to stop eating when I want to. I know when "enough is enough".
(2) Every so often, I experience a compulsion to eat which I can't seem to control.
(3) Frequently, I experience strong urges to eat which I seem unable to control, but at other times I can control my eating urges.
(4) I feel incapable of controlling urges to eat. I have a fear of not being able to stop eating voluntarily.

#11

(1) I don't have any problem stopping eating when I feel full.
(2) I can usually stop eating when I feel full but occasionally overeat, leaving me feeling uncomfortably stuffed.
(3) I have a problem stopping eating once I start and usually I feel uncomfortably stuffed after I eat a meal.
(4) Because I have a problem not being able to stop eating when I want, I sometimes have to induce vomiting to relieve my stuffed feeling.

#12

(1) I seem to eat just as much when I'm with others (family, social gathering) as when I'm by myself.
(2) Sometimes, when I'm with other persons, I don't eat as much as I want to eat because I'm self-conscious about my eating.
(3) Frequently, I eat only a small amount of food when others are present, because I'm very embarrassed about my eating.
(4) I feel so ashamed about overeating that I pick times to overeat when I know no one will see me. I feel like a "closet eater".

#13

(1) I eat three meals a day with only an occasional between meal snack.
(2) I eat three meals a day, but I also normally snack between meals.
(3) When I am snacking heavily, I get in the habit of skipping regular meals.
(4) There are regular periods when I seem to be continually eating, with no planned meals.

#14

(1) I don't think much about trying to control unwanted eating urges.
(2) At least some of time, I feel my thoughts are preoccupied with trying to control my eating urges.
(3) I feel that frequently I spend much time thinking about how much O ate or about trying not to eat anymore.
(4) It seems to me that most of my waking hours are preoccupied by thoughts about eating or not eating. I feel like I'm constantly struggling not to eat.

#15

(1) I don't think about food a great deal.
(2) I have strong cravings for food but they only last for brief periods of time.
(3) I have days when I can't seem to think about anything else but food.
(4) Most of my days seem to be preoccupied with thoughts about food. I feel like I live to eat.

#16

(1) I usually know whether or not I'm physically hungry. I take the right portion of food to satisfy me.
(2) Occasionally, I feel uncertain about knowing whether or not I'm physically hungry. At these times it's hard to know how much food I should take to satisfy me.
(3) Even though I might know how many calories I should eat, I don't have any idea what is a "normal" amount of food for me.

APPENDIX B: Edinburgh Handedness Inventory

Please indicate your preferences in the use of hands in the following activities *by putting a check in the appropriate column*. If in any case you are really indifferent, *put a check in both columns*.

Some of the activities listed below require the use of both hands. In these cases, the part of the task, or object, for which hand preference is wanted is indicated in parentheses.

Please try and answer all of the questions, and only leave a blank if you have no experience at all with the object or task.

	Left Hand	Right Hand
1. Writing		
2. Drawing		
3. Throwing		
4. Scissors		
5. Toothbrush		
6. knife (without fork)		
7. Spoon		
8. Broom (upper hand)		
9. Striking match (match)		
10. Opening box (lid)		

	Left	Right
11. Which foot do you prefer to kick with?		
12. Which eye do you use when using only one?		

APPENDIX C: MOOD AND ANXIETY SYMPTOMS QUESTIONNAIRE

Below is a list of feelings, sensations, problems, and experiences that people sometimes have. Read each item and then mark the appropriate choice in the space next to that item. Use the choice that best describes how much you have felt or experienced things this way during the past week, including today. Use this scale when answering:

1	2	3	4	5
not at all	a little bit	moderately	quite a bit	extremely

_____	1. Felt sad	_____	22. Felt like I was having a lot of fun
_____	2. Startled easily	_____	23. Blamed myself for a lot of things
_____	3. Felt cheerful	_____	24. Hands were cold or sweaty
_____	4. Felt afraid	_____	25. Felt withdrawn from other people
_____	5. Felt discouraged	_____	26. Felt keyed up, "on edge"
_____	6. Hands were shaky	_____	27. Felt like I had a lot of energy
_____	7. Felt optimistic	_____	28. Was trembling or shaking
_____	8. Had diarrhea	_____	29. Felt inferior to others
_____	9. Felt worthless	_____	30. Had trouble swallowing
_____	10. Felt really happy	_____	31. Felt like crying
_____	11. Felt nervous	_____	32. Was unable to relax
_____	12. Felt depressed	_____	33. Felt really slowed down
_____	13. Was short of breath	_____	34. Was disappointed in myself
_____	14. Felt uneasy	_____	35. Felt nauseous
_____	15. Was proud of myself	_____	36. Felt hopeless
_____	16. Had a lump in my throat	_____	37. Felt dizzy or lightheaded
_____	17. Felt faint	_____	38. Felt sluggish or tired
_____	18. Felt unattractive	_____	39. Felt really "up" or lively
_____	19. Had hot or cold spells	_____	40. Had pain in my chest
_____	20. Had an upset stomach	_____	41. Felt really bored
_____	21. Felt like a failure	_____	42. Felt like I was choking

MASQ (cont.)

1	2	3	4	5
not at all	a little bit	moderately	quite a bit	extremely

_____ 43. Looked forward to things with enjoyment

_____ 44. Muscles twitched or trembled

_____ 45. Felt pessimistic about the future

_____ 46. Had a very dry mouth

_____ 47. Felt like I had a lot of interesting things to do

_____ 48. Felt like I had accomplished a lot

_____ 49. Felt like it took extra effort to get started

_____ 50. Felt like nothing was very enjoyable

_____ 51. Heart was racing or pounding

_____ 52. Felt like I had a lot to look forward to

_____ 53. Felt numbness or tingling in my body

_____ 54. Felt tense or "high-strung"

_____ 55. Felt hopeful about the future

_____ 56. Felt like there wasn't anything interesting or fun to do

_____ 57. Seemed to move quickly and easily

_____ 58. Muscles were tense or sore

_____ 59. Felt really good about myself

_____ 60. Had to urinate frequently

APPENDIX D: POSITIVE AND NEGATIVE AFFECT SCHEDULE

This scale consists of a number of words that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you feel this way right now, that is, at the present moment. Use the following scale to record your answers:

1	2	3	4	5
very slightly or not at all	a little	moderately	quite a bit	extremely

_____ interested	_____ irritable
_____ distressed	_____ alert
_____ excited	_____ ashamed
_____ upset	_____ inspired
_____ strong	_____ nervous
_____ guilty	_____ determined
_____ scared	_____ attentive
_____ hostile	_____ jittery
_____ enthusiastic	_____ active
_____ proud	_____ afraid

APPENDIX E: POWER OF FOOD SCALE

Please indicate the extent to which you agree that the following items describe you. **Base your answers on your experience during the past month only**. Use the following scale for your responses.

A don't agree at all
B agree a little
C agree somewhat
D agree
E strongly agree

---

1. I find myself thinking about food even when I'm not physically hungry.
2. When I'm in a situation where delicious foods are present but I have to wait to eat them, it is very difficult for me to wait.
3. I get more pleasure from eating than I do from almost anything else.
4. I feel that food is to me like liquor is to an alcoholic.
5. If I see or smell a food I like, I get a powerful urge to have some.
6. When I'm around a fattening food I love, it's hard to stop myself from at least tasting it.
7. I often think about what foods I might eat later in the day.
8. It's scary to think of the power that food has over me.
9. When I taste a favorite food, I feel intense pleasure.
10. When I know a delicious food is available, I can't help myself from thinking about having some.
11. I love the taste of certain foods so much that I can't avoid eating them even if they're bad for me.
12. When I see delicious foods in advertisements or commercials, it makes me want to eat.
13. I feel like food controls me rather than the other way around.

14. Just before I taste a favorite food, I feel intense anticipation.

15. When I eat delicious food I focus a lot on how good it tastes.

16. Sometimes, when I'm doing everyday activities, I get an urge to eat "out of the blue" (for no apparent reason).

17. I think I enjoy eating a lot more than most other people.

18. Hearing someone describe a great meal makes me really want to have something to eat.

19. It seems like I have food on my mind a lot.

20. It's very important to me that the foods I eat are as delicious as possible.

21. Before I eat a favorite food my mouth tends to flood with saliva.

APPENDIX F: THREE-FACTOR EATING QUESTIONNAIRE

Read each of the following statements carefully. If you agree with the statement, or feel that it is true as applied to you, fill in the "A" **on the scantron form** next to the corresponding number. If you disagree with the statement, or feel that it is false as applied to you, fill in the "B" **on the scantron form** next to the corresponding number. Be certain to answer each question.

Remember **A = True**
B = False

1. When I smell a sizzling steak or see a juicy piece of meat, I find it very difficult to keep from eating, even if I have just finished a meal.

2. I usually eat too much at social occasions, like parties and picnics.

3. When I have eaten my quota of calories, I am usually good about not eating any more.

4. I deliberately take small helpings as a means of controlling my weight.

5. Sometimes things just taste so good that I keep on eating even when I am no longer hungry.

6. When I feel anxious, I find myself eating.

7. Life is too short to worry about dieting.

8. Since my weight goes up and down, I have gone on reducing diets more than once.

9. When I am with someone who is overeating, I usually overeat too.

10. I have a pretty good idea of the number of calories in common foods.

11. Sometimes when I start eating, I just can't seem to stop.

12. It is not difficult for me to leave something on my plate.

13. While on a diet, if I eat a food that is not allowed, I consciously eat less for a period of time to make up for it.

14. When I feel blue, I often overeat.

15. I enjoy eating too much to spoil it by counting calories or watching my weight.

16. I often stop eating when I am not really full as a conscious means of limiting the amount that I eat.

17. My weight has hardly changed at all in the last ten years.

18. When I feel lonely, I console myself by eating.

19. I consciously hold back at meals in order not to gain weight.

20. I eat anything I want, any time I want.

21. Without even thinking about it, I take a long time to eat.

22. I count calories as a conscious means of controlling my weight.

23. I do not eat some foods because they make me fat.

24. I pay a great deal of attention to changes in my figure.

25. While on a diet, if I eat a food that is not allowed, I often then splurge and eat other high calorie foods.

26. If I eat a little bit more on one day, I make up for it the next day.

27. I pay attention to my figure, but I still enjoy a variety of foods.

28. I prefer light foods that are not fattening.

29. If I eat a little bit more during one meal, I make up for it at the next meal.

30. I eat diet foods, even if they do not taste very good.

31. A diet would be too boring a way for me to lose weight.

32. I would rather skip a meal than stop in the middle of one.

33. I alternate between times when I diet strictly and times when I don't pay much attention to what and how much I eat.

34. Sometimes I skip meals to avoid gaining weight.

35. I avoid some foods on principle even though I like them.

36. I try to stick to a plan when I lose weight.

37. Without a diet plan I wouldn't know how to control my weight.

38. Quick success is most important for me during a diet.

TFEQ PART II

Each question in this section is followed by a number of answer options. After reading each question carefully, fill in the letter on the scantron form that corresponds to the option which most applies to you. Be certain to answer all questions.

39. How often are you dieting in a conscious effort to control your weight?
    a rarely
    b sometimes
    c usually
    d always

40. Would a weight fluctuation of 5 lbs. affect the way you live your life?

    a not at all
    b slightly
    c moderately
    d very much

41. Do your feelings of guilt about overeating help you to control your food intake?

    a never
    b rarely
    c often
    d always

42. How conscious are you of what you are eating?

    a not at all
    b slightly
    c moderately
    d extremely

43. How frequently do you *avoid* "stocking up" on tempting foods?

    a almost never
    b seldom
    c usually
    d almost always

44. How likely are you to shop for low calorie foods?

a unlikely
b slightly likely
c moderately likely
d very likely

45. Do you eat sensibly in front of others and splurge alone?

a never
b rarely
c often
d always

46. How likely are you to consciously eat slowly in order to cut down on how much you eat?

a unlikely
b slightly likely
c moderately likely
d very likely

47. How likely are you to consciously eat less than you want?

a unlikely
b slightly likely
c moderately likely
d very likely

48. Do you go on eating binges even though you are not hungry?

a never
b rarely
c sometimes
d at least once a week

49. Do you deliberately restrict your intake during meals even though you would like to eat more?

a never
b rarely
c often
d always

50. To what extent does this statement describe your eating behavior?

"I start dieting in the morning, but because of any number of things that happen during the day, by evening I have given up and eat what I want, promising myself to start dieting again tomorrow."

a not like me
b little like me
c pretty good description of me
d describes me perfectly

51. On a scale of 1 to 5, where 1 means no restraint in eating (eat whatever you want, whenever you want it) and 5 means total restraint (usually or constantly limiting food intake and rarely or never "giving in"), what number would you give yourself?

a eat whatever you want, whenever you want it
b usually eat whatever you want, whenever you want it
c often eat whatever you want, whenever you want it
d often limit food intake, but often "give in"
e usually or constantly limit food intake, rarely or never "give in"

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Designed and taught undergraduate Personality Theory course

Publications

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