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Research report

Asymmetric prefrontal cortex activation in relation to markers of overeating in obese humans

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ABSTRACT

Dietary restraint is heavily influenced by affect, which has been independently related to asymmetrical activation in the prefrontal cortex (prefrontal asymmetry) in electroencephalograph (EEG) studies. In normal weight individuals, dietary restraint has been related to prefrontal asymmetry; however, this relationship was not mediated by affect. This study was designed to test the hypotheses that, in an overweight and obese sample, dietary restraint as well as binge eating, disinhibition, hunger, and appetitive responsivity would be related to prefrontal asymmetry independent of affect at the time of assessment. Resting EEG recordings and self-report measures of overeating and affect were collected in 28 overweight and obese adults. Linear regression analyses were used to predict prefrontal asymmetry from appetitive measures while controlling for affect. Cognitive restraint and binge eating were not associated with prefrontal asymmetry. However, disinhibition, hunger, and appetitive responsivity predicted left-, greater than right-, sided prefrontal cortex activation independent of affect. Findings in this study add to a growing literature implicating the prefrontal cortex in the cognitive control of dietary intake. Further research to specify the precise role of prefrontal asymmetry in the motivation toward, and cessation of, feeding in obese individuals is encouraged.

Introduction

Chronic overeating has reached pandemic proportions (CDC, 2006). Such overeating ranges from chronic passive overconsumption (Blundell & MacDiarmid, 1997), to recurrent binge episodes reported in up to 40% of individuals seeking weight loss treatment (Spitzer, Devlin, Walsh, & Hasin, 1992). Theories explaining the propensity to overeat have been primarily based on behavioral studies and have not yielded effective long-term behavioral interventions. The need for improved methods of examining and conceptualizing the appetitive vulnerabilities that lead to overeating in obese individuals may be, in part, fulfilled by examining the neurobiological correlates of appetitive drive.

Although the investigation of the neural activity associated with appetitive drive remains in its infancy (Chowdhury & Lask, 2001), a relationship between ingestive behavior and activation in the prefrontal cortex (PFC) has emerged (Alonso-Alonso & Pascual-Leone, 2007; Le et al., 2006). Several authors suggest a prominent role of the PFC in the cognitive regulation of food intake (Le et al., 2006; Tataranni & DelParigi, 2003) and further evidence indicates that the (a)symmetry of PFC activation (activation in one, relative to the other, hemisphere of the PFC) may be integral in identifying the specific role of the PFC in appetitive behavior (Andreason et al., 1992; Karhunen et al., 2000; Silva, Pizzagalli, Larson, Jackson, & Davidson, 2002). At rest, individuals typically display relatively symmetrical activation in the PFC (Murphy, Nimmo-Smith, & Lawrence, 2003); however, recent research suggests that individuals reporting disordered eating patterns may experience asymmetry in activation of the PFC or "prefrontal asymmetry" (Andreason et al., 1992; Karhunen et al., 2000; Silva et al., 2002). Obese binge eaters, for example, display greater increases in left-, relative to right-, sided prefrontal asymmetry as compared to lean and obese non-binge eaters following exposure to palatable food (Karhunen et al., 2000). Strong linear correlations were also observed in obese binge eaters between increases in hunger and left-, greater than right-, sided (left-sided) prefrontal asymmetry (Karhunen et al., 2000).

The PFC is proposed to be responsible for instantiating the experience and execution of affect-related behavior (Davidson, Jackson, & Kalin, 2000; Miller & Cohen, 2001). According to the affective theory (Davidson, 2000, 2003), emotion results from neural signals in the PFC, separated into two systems: the approach-related positive affect, and withdrawal-related negative affect, systems. Accordingly, the positive affect system is activated

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as a person moves toward an appetitive goal, while the negative affect system facilitates withdrawal from sources of aversive stimulation (Davidson, 2003; Tomarken, Davidson, Wheeler, & Doss, 1992). Several neuroimaging studies have related positive affect to left-sided prefrontal asymmetry (Davidson, 2000; Sutton & Davidson, 2000; Tomarken et al., 1992) and negative affect to right-sided prefrontal asymmetry (Davidson et al., 2000; Davidson, 2003; Wheeler, Davidson, & Tomarken, 1993).

Based on the proposed relationship between negative affect and right-sided prefrontal asymmetry (Davidson, 2000), and the relationship between negative affect and dietary restraint in normal weight individuals (Sheppard-Sawyer, McNalley, & Fischer, 2000), Silva et al. (2002) hypothesized that restrained eating would be related to right-sided prefrontal asymmetry in a normal weight sample. Dietary restraint and prefrontal asymmetry were assessed using the Restraint Scale (Herman & Polivy, 1980) and EEG imaging (respectively), and results confirmed the proposed hypothesis. However, affect was not found to mediate the relationship between prefrontal asymmetry and Restraint Scale scores, suggesting a relationship between dietary restraint in lean individuals and prefrontal asymmetry independent of affect (Silva et al., 2002).

Silva et al. (2002) additionally suggest that right-sided prefrontal asymmetry may be related to other indicators of disordered eating, such as bulimia. However, bulimic individuals have been shown to display more left-sided PFC activation relative to normal individuals (Andreason et al., 1992) despite the strong association between bulimia and depression (Hinz & Williamson, 1987). Noting other findings inconsistent with the affective model, particularly the relationship between anger (a negative, but approach-related emotion) and left-sided prefrontal asymmetry (Harmon-Jones & Allen, 1997), Harmon-Jones (2003, 2004) proposed that affective valence (positive–negative) and approach–withdrawal tendencies were two related but distinct constructs. He suggests that left- and right-sided prefrontal asymmetry reflect motivational direction (approach vs. withdrawal, respectively) irrespective of associated affect (Harmon-Jones, 2003, 2004).

The present study was designed to test, in an overweight and obese sample, the primary hypotheses that prefrontal asymmetry would be related to dietary restraint as well as binge eating, disinhibition, hunger and appetitive responsivity and that these relationships would be found independent of affect at the time of assessment. The two competing models of prefrontal asymmetry predicted different outcomes in terms of the directionality of the asymmetry. The affective model would have predicted that dietary restraint, binge eating and disinhibition (appetitive behaviors associated with negative affect; Sheppard-Sawyer et al., 2000; Wardle, Waller, & Rapoport, 2001) would be related to right-sided prefrontal asymmetry. The motivation direction model would also have predicted that dietary restraint (reflecting a withdrawal-like tendency in the absence of disinhibiting stimuli; Herman & Polivy, 1980; Silva et al., 2002) would be associated with right-sided prefrontal asymmetry, but that binge eating, disinhibition, hunger and appetitive responsivity (reflecting approach-like tendencies) would be related to left-sided prefrontal asymmetry. Being the preeminent theory of prefrontal asymmetry, secondary hypotheses regarding the directionality of asymmetry were based on the affective model.

Methods

Participants

Forty participants were recruited through physician referral to a weight loss intervention study being conducted at Drexel University in Philadelphia, PA. Participants were told they were being recruited for an unrelated study of brain activity and all participants completed this study prior to any weight loss intervention. Nine female and three male participants either failed to arrive at their scheduled appointment, produced unusable EEG data due to equipment failure, or were eliminated from the study due to hair styles (e.g., weaves) that precluded the ability to establish a clean EEG connection, yielding 28 (26F; 2M) completers. All participants were right-handed, overweight or obese, were not participating in a weight control program and reported they were not currently dieting to lose weight. Baseline characteristics are shown in Table 1. Participants were not taking medications and had no physical or psychological conditions that may have affected body weight (e.g., pregnancy, depression) or brain activity (e.g., open head wound, learning disability). All applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during this research and approval for this study was granted from the Drexel University Medical Institutional Review Board.

Appetitive measures

Dietary restraint, disinhibition and hunger

The Three Factor Eating Questionnaire (TFEQ; Stunkard & Messick, 1985) has demonstrated good reliability and validity (Laessle, Tuschl, Kotthaus, & Pirke, 1989; Stunkard & Messick, 1985). The Cognitive Restraint, Disinhibition and Hunger subscales have also demonstrated adequate internal consistency (Laessle et al., 1989; Stunkard & Messick, 1985).

Binge eating

The Binge Eating Scale (BES; Gormally, Black, Datson, & Rardin, 1982) was designed specifically to assess binge eating severity within an obese population. The BES displays adequate psychometric properties (Timmerman, 1999) and successfully discriminates between individuals no, moderate, or severe binge eating problems (Gormally et al., 1982).

Appetitive responsivity

The Power of Food Scale (PFS; Cappelleri et al., in press) was designed to assess individual psychological reactions to the food environment. The PFS demonstrates good internal consistency, temporal stability, convergent validity, and discriminant validity (Annunziato, Lee, & Lowe, 2007; Cappelleri et al., in press). Validation studies suggest that the PFS reflects global levels of appetitive responsivity and latent potential for overeating (Forman et al., 2007; Lowe, 2006).

Affective measures

Anxiety and depression

The Mood and Anxiety Symptom Questionnaire (MASQ; Clark & Watson, 1991) Anhedonic Depression and Anxious Arousal

Table 1
Baseline sample characteristics.

			Male		Female
Gender (*n*)			2		26
		Range		Mean	SD
Age (years)		29–70		49.2	12.3
BMI (kg/m^2)		29.1–61.5		39.2	6.7
	AA[a]	Cauc[b]	Latino	≥1	Unknown
Ethnicity (%)	79	7	4	7	4

[a] African American.
[b] Caucasian.

subscales were used to measure state affect in the aforementioned (Silva et al., 2002) study of prefrontal asymmetry. The Anxious Arousal and Anhedonic Depression subscales display adequate levels of reliability and validity (Reidy & Keogh, 1997).

Positive and negative affect

The *state version* of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) consists of positive and negative mood scales designed to assess affect "at the present moment." These scales have shown to be highly internally consistent, uncorrelated, and stable over time (Watson et al., 1988), and both scales demonstrate good convergent and discriminant validity (Crawford & Henry, 2004).

General measures

Handedness

The Edinburgh Handedness Inventory (EHI; Oldfield, 1971) demonstrates good reliability and validity in the assessment of handedness, and has been used in previous prefrontal asymmetry research (e.g., Tomarken et al., 1992).

Height and weight

A standard physician stadiometer was used to measure height. Weight was measured in street clothes, without shoes, using a standardized Seca® 644 scale accurate to 0.1 kg.

Procedure

One week prior to EEG assessments, participants filled out appetitive measures, as a part of the parent study from which they were recruited. Dietary restraint was measured using the TFEQ Cognitive Restraint subscale as it has been shown to be more reliable than the Restraint Scale (Herman & Polivy, 1980) in assessing dietary restraint in overweight and obese individuals (Ruderman, 1986; van Strien, Herman, Engels, Larsen, & van Leeuwe, 2007). Binge eating, disinhibition, hunger, and appetitive responsivity were assessed using the BES, TFEQ Disinhibition subscale, TFEQ Hunger subscale and PFS, respectively. Immediately preceding EEG recordings, participants filled out the EHI and affective measures (MASQ, and PANAS state version). The specific research hypotheses of this study were withheld from participants so EEG recordings would not be affected. Resting-state EEG was then recorded.

All participants were instructed to consume a ~500 kcal breakfast (two eggs, two slice toast, and one glass orange juice suggested) and to not consume any caffeine in the morning before reporting for EEG assessments at 11 am. EEG was recorded using a stretchable lycra cap with 128 embedded electrodes (Electro-Cap International, Inc.). Electrodes were applied according to the extended International 10–20 System (digitally linked mastoid reference). Data were collected during 8 60-s trials, four with eyes open and four with eyes closed, presented in counterbalanced alternating order. Electrode impedances were kept below 20,000 Ω (per manufacturer recommendation). All EEG data were collected using a sample rate of 256 Hz and bandpass filtered at 0.02–100 Hz. EEG was amplified 20,000 times using the MICRO-AMPS™ data acquisition system (SAM Technology, Inc.). EEG signals were then digitized using the MANSCAN® RECORDER system (SAM Technology, Inc.).

Automatic artifact detection, followed by visual inspection was used to remove artifacts due to eye blinks, gross muscle activity, and movement. Artifact-free epochs of data were extracted through a Hanning window. Fast Fourier Transform was applied to all extracted epochs that were four seconds in duration (ranging from 129 to 228 epochs per condition), with epochs overlapping 50%. 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.

Log-transformed EEG power values in the alpha band (8–13 Hz) were computed for all electrodes. Frontal asymmetry scores were computed by subtracting the value obtained at the left-frontal electrode F3 from the corresponding value at the homologous right-frontal electrode F4 (log F4–log F3). Because alpha-band EEG power is inversely proportional to magnitude of neural activity, positive asymmetry scores reflect greater left-sided neural activity (i.e., greater alpha band power density on the right than on the left). Conversely, negative asymmetry scores reflect greater right-sided activity.

Mean (M) and standard deviation (SD) values were calculated for scores on all measures, as well as asymmetry scores in the PFC (Table 2). Scores on the MASQ and the state version of the PANAS were used to remove the variance in asymmetry accounted for by depressive or anxious symptomatology and affective valence at the time of measurement. The relationships between all self-report measures were calculated using Pearson correlations. Individual linear regression analyses were then used to test the relationships between affective measures, appetitive measures and prefrontal asymmetry, both with and without controlling for BMI. Finally, a stepwise regression analysis was performed to determine the best model for predicting prefrontal asymmetry in this sample. All analyses were additionally repeated controlling for age, and gender.

Results

Relationships between appetitive and affective measures

Pearson correlations between all self-report measures are shown in Table 3. Unsurprisingly, anhedonic depression [MASQ subscale] was inversely related to positive affect [PANAS subscale], and positively related to negative affect ($p < 0.001$ and $p = 0.043$, respectively). Binge eating [BES] was positively related to disinhibition [TFEQ subscale] ($p = 0.002$) and appetitive responsivity [PFS] ($p = 0.005$). Disinhibition was also positively related to hunger ($p = 0.01$) and both disinhibition and hunger were positively related to appetitive responsivity ($p < 0.0005$ and

Table 2
Descriptive statistics across measures.

	N	Range	Mean	SD
Appetitive measures				
TFEQ Cog Restraint	24	7–30	18.9	5.9
BES	26	16–40	25.4	6.4
TFEQ Disinhibition	26	1–12	5.1	2.8
TFEQ Hunger	21	1–12	3.7	2.6
PFS	21	21–89	36.8	15.8
Affective measures				
MASQ Anhed Depression	28	32–81	53.0	11.3
MASQ Anxious Arousal	28	18–35	21.9	3.8
PANAS Positive Affect	28	15–49	34.2	7.8
PANAS Negative Affect	28	10–16	11.5	1.6
Asymmetrical Activation				
Prefrontal asymmetry[a]	28	−0.07–0.34	0.039	0.082

Note: differences in *N*s between measures reflect both incomplete questionnaire data provided by participants, as well as the addition of certain measures (TFEQ Hunger, PFS) after several participants had completed the study.

[a] Positive asymmetry scores reflect left-, greater than right-, sided PFC activation.

Table 3
Pearson correlations (r) between appetitive and affective measures.

	Dietary restraint (TFEQ)	Binge eating	Disinhibit	Hunger	Appetitive Respons	Depress	Anxiety	Pos Affect
Binge eating (BES)	0.11							
Disinhibition (TFEQ)	−0.20	0.60**						
Hunger (TFEQ)	−0.40	0.40	0.58**					
Appetitive Respons (PFS)	−0.33	0.60**	0.78**	0.55*				
Depression (MASQ)	0.07	0.21	−0.02	−0.05	0.27			
Anxiety (MASQ)	0.02	0.21	0.48*	−0.05	0.20	−0.13		
Pos Affect (PANAS)	0.38	−0.31	−0.25	−0.19	−0.49*	−0.66**	−0.10	
Neg Affect (PANAS)	−0.06	0.00	0.08	0.04	0.23	0.39*	−0.02	−0.42*

BMI was not related to any measure (all $ps > 0.2$).
* Significant at $p < 0.05$.
** Significant at $p < 0.01$.

$p = 0.014$, respectively). The only significant relationships between affective, and appetitive, measures were the inverse relationship between positive affect and appetitive responsivity ($p = 0.025$) and positive relationship between anxiety and disinhibition ($p = 0.014$). All analyses were repeated controlling for BMI with no significant change in results (not shown).

Relationships between appetitive and affective measures and prefrontal asymmetry

Dietary restraint

Dietary restraint did not predict prefrontal asymmetry in this sample with or without controlling for BMI and/or affective measures.

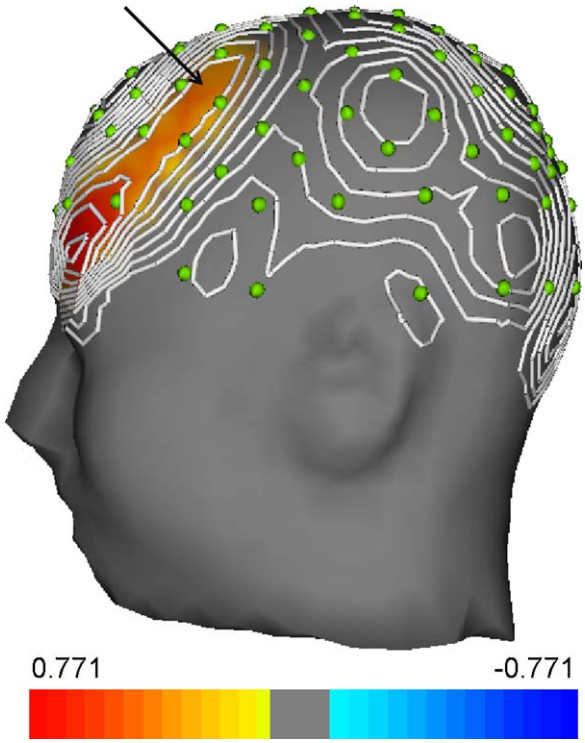

Fig. 1. Topographic map of the relationship (standardized coefficient [*Beta*]) between log-transformed scalp alpha-power hemispheric asymmetry scores across all electrode sites, and scores on the Power of Food Scale (PFS). The map was created by computing Beta for homologous pairs of electrodes. These coefficients were then used to generate a spline-interpolated map on a lateral view of the head. Each green dot represents the location of an electrode, and the arrow denotes the region of the prefrontal cortex. Orange and red regions on the map (*Beta* coefficients) reflect the positive relationship between appetitive responsivity and alpha asymmetry. That is, more neural activity (lower alpha power) measured at left-hemisphere electrodes for each corresponding (hemispheric) pair of electrodes in relation to PFS scores. Only Beta coefficients significant at $p < 0.05$ are shown.

Binge eating

Binge eating did not predict prefrontal asymmetry in this sample with, or without, controlling for BMI and/or affective measures.

Disinhibition

Disinhibition predicted prefrontal asymmetry ($t(25) = 2.5$, $p = 0.018$), such that higher Disinhibition scores were associated with greater left-sided PFC activation. This relationship remained unchanged when controlling for BMI ($t(25) = 2.6$, $p = 0.017$) or affective measures ($t(25) = 2.7$, $p = 0.016$) but showed a modest but nonsignificant increase in strength when controlling BMI and affective measures simultaneously ($t(25) = 3.1$, $p = 0.007$).

Hunger

Hunger predicted prefrontal asymmetry ($t(20) = 3.4$, $p = 0.003$), such that higher Hunger scores were associated with greater left-sided PFC activation. This relationship remained significant when controlling for BMI ($t(20) = 3.0$, $p = 0.008$), affective measures ($t(20) = 3.4$, $p = 0.004$) or BMI and affective measures simultaneously ($t(20) = 3.0$, $p = 0.01$).

Appetitive responsivity

Appetitive responsivity predicted prefrontal asymmetry scores ($t(20) = 2.3$, $p = 0.011$), such that higher Appetitive Responsivity scores were associated with greater left-sided PFC activation. The strength of this relationship showed a nonsignificant increase when controlling for BMI ($t(20) = 3.2$, $p = 0.005$), affective measures ($t(20) = 3.3$, $p = 0.005$), and BMI and affective measures simultaneously ($t(20) = 3.4$, $p = 0.004$).

Affect

No scores on any affective measure were related to prefrontal asymmetry in this sample.

A stepwise regression analysis with all self-report measures and BMI entered revealed that the single best predictor of prefrontal asymmetry was appetitive responsivity ($t(17) = 2.7$, $p = 0.013$, beta = 0.6), accounting for 32% of the variance (Fig. 1). The tolerance and variance inflation factor (VIF) values for this model were both 1.00, indicating little colinearity. All regression analyses were repeated with age and gender entered as covariates with no change in results. Analyses were additionally repeated with only female ($n = 26$) participants with no change in results.

Discussion

In this overweight and obese sample, measures of dietary restraint, binge eating, disinhibition, hunger, and appetitive responsivity were examined in relation to prefrontal asymmetry and affect at the time of assessment. Dietary restraint was not related to other appetitive measures; however, consistent with previous literature (Cappelleri et al., in press; Marcus, Wing, &

Lamparski, 1985), individuals reporting more binge eating also reported greater levels of disinhibition and appetitive responsivity. Affect at the time of assessment was generally unrelated to appetitive measures in this study. The only exceptions were an inverse relationship between scores on the PFS and PANAS Positive Affect subscale and positive relationship between scores on the TFEQ Disinhibition and MASQ Anxious Arousal subscales, indicating that individuals higher in appetitive responsivity reported less positive affect and individuals with a greater tendency to become disinhibited reported higher levels of anxiety at the time of assessment.

Dietary restraint, as measured by the TFEQ, was not related to prefrontal asymmetry in this overweight and obese sample. Evidence of restraint theory (Herman & Mack, 1975) has not been consistent with measures of dietary restraint other than the Restraint Scale (Lowe & Kleifield, 1988; Westenhoefer, Broeckmann, Munch, & Pudel, 1994), previously shown to correlate with prefrontal asymmetry in lean individuals (Silva et al., 2002). It has also been suggested that the Restraint Scale, used in the Silva et al. (2002) study, actually measures disinhibition more so than dietary restraint (Stunkard & Messick, 1985; Westenhoefer et al., 1994). "The restraint subscales of the DEBQ and TFEQ measure the tendency of actually restricted caloric intake in everyday eating behavior (Laessle et al., 1989), whereas the Restraint Scale identifies dieters who have a tendency to get disinhibited" (Westenhoefer et al., 1994; p. 28). This assertion would suggest a relationship between disinhibition and right-sided PFC activation in lean individuals (Silva et al., 2002), and a relationship between disinhibition and left-sided PFC activation in obese individuals found in the present study. The finding that binge eating was not related to prefrontal asymmetry in this sample was somewhat surprising, given correlations between BES scores and scores on the PFS and TFEQ Disinhibition subscales (see Table 3). Although past literature has demonstrated increased left-sided prefrontal asymmetry in obese binge eaters (Karhunen et al., 2000), this relationship was only found during exposure to highly palatable food images, which may have elicited positive affect and/or approach motivation.

No relationship was found between affect at the time of assessment and prefrontal asymmetry, indicating that affect did not mediate the relationship between appetitive measures and prefrontal asymmetry (Baron & Kenny, 1986). Paired with the findings that disinhibition, hunger, and appetitive responsivity were related to prefrontal asymmetry after controlling for affect at the time of assessment, outcomes from this study suggest that there may be a relationship between prefrontal asymmetry and the propensity to overeat independent of affect. These results are also consistent with previous studies of behavioral and psychological measures in relation to prefrontal asymmetry, found to be independent of affect (Davidson et al., 2000; Harmon-Jones & Allen, 1997; Karhunen et al., 2000; Silva et al., 2002; Sutton & Davidson, 2000; Wheeler et al., 1993). The use of state affect measurements in this study leaves open the possibility that *trait* affect may mediate such relationships; however, Karhunen et al. (2000) found that depressive symptomatology, assessed by the BDI (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), were not associated with prefrontal asymmetry; "The observed differences in the asymmetry of the hemispheric blood flow between the binge and non-binge eating subjects could thus be suggested to be associated with the core features of eating behavior, rather than with depression." (p. 40). In addition, the directionality of results (i.e., measures of overeating related to left-sided PFC activation), reduce the likelihood that trait affect could have mediated the relationships between prefrontal asymmetry and appetitive measures found in the present study.

According to the affective hypothesis of prefrontal asymmetry (Davidson, 2000, 2003), individuals higher in appetitive responsivity and disinhibition should be prone to experience more positive affect, due to the relationships between both PFS and TFEQ Disinhibition subscale scores and left-sided PFC activation. The inverse relationship between PFS and PANAS Positive Affect Scale scores, as well as the relationship between TFEQ Disinhibition subscale and MASQ Anxious Arousal subscale scores, seem to contradict this theory. The relationship between left-sided PFC activation and disinhibition may be particularly disconcerting for proponents of the affective theory, as disinhibition has frequently been associated with negative affect (Sheppard-Sawyer et al., 2000; Stunkard et al., 1991). Although not allowing for direct comparison across models, results in this study appear more consistent with the approach-withdrawal (Harmon-Jones, 2003, 2004) model. That is, increased disinhibition, hunger, and appetitive responsivity may reflect more "approach" tendencies, rather than reflecting positive affect. This contention is also consistent with a meta-analysis of 106 studies (Murphy et al., 2003) revealing that left-sided frontal asymmetry was associated with approach, but not necessarily positive, emotions.

Limitations of this study include the questionnaire-based assessment of appetitive behavior, limited generalizability to other populations, and the heterogeneity of the sample (large BMI and age ranges). Equipment failure resulted in unusable data for seven additional patients, and missing self-report data was not interpolated, resulting in unusable outcome measure scores for several patients; however, outcome measures with the lowest sample size (TFEQ Hunger and PFS) yielded significant results in relation to prefrontal asymmetry. It is important to note that functional and anatomical divisions exist within the PFC and that EEG imaging, suggested to reflect mainly dorsolateral regions of the PFC (Davidson, 2004), does not provide the spatial resolution necessary to isolate and examine specific regions within the PFC. Finally, the authors would like to point out that asymmetrical activation was not found exclusively in the PFS (as reflected in Fig. 1), however, *a priori* predictions involved only the PFC as reliable interpretation of asymmetry in other brain areas is not yet supported by the literature.

Conclusion

Left-sided PFC activation in obese individuals was related to measures of disinhibition, hunger, and appetitive responsivity, but not dietary restraint or binge eating in this overweight and obese sample. In addition, disinhibition was correlated with negative affect (anxiety) and appetitive responsivity was inversely correlated with positive affect; however, affect at the time of assessment was not related to prefrontal asymmetry. These results partially support the proposed relationship between the tendency to overeat and asymmetrical activation in the prefrontal cortex, but do not support the affective model of prefrontal asymmetry. Findings in this study encourage further exploration into the motivational model of prefrontal asymmetry and its relation to overeating in overweight and obese humans.

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