Intern. J. Neuroscience, 114:83–93, 2004

ISSN: 0020-7454 / 1543-5245 online
DOI: 10.1080/00207450490249356

Taylor & Francis
healthsciences

EXECUTIVE PERSONALITY TRAITS AND EATING BEHAVIOR

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Eating disorders, such as anorexia, bulimia, and binge eating disorder, commonly involve a dysregulation of behavior (e.g., a lack or excess of inhibition and impulsive eating patterns) that is suggestive of prefrontal dysfunction. Functional neuro-imaging studies show that prefrontal-subcortical systems play a role in eating behavior and appetite in healthy individuals, and that people with eating disorders have altered activity in these systems. Eating behavior is often disturbed by illnesses and injuries that impinge upon prefrontal-subcortical systems. This study examined relationships between executive functioning and eating behavior in healthy individuals using validated behavioral rating scales (Frontal Systems Behavior Scale and Eating Inventory). Correlations demonstrated that increased dysexecutive traits were associated with disinhibited eating and greater food cravings. There was also a positive association with cognitive restraint of eating, suggesting that increased compensatory behaviors follow disinhibited eating. These psychometric findings reinforce those of other methodologies, supporting a role for prefrontal systems in eating.

Keywords eating, eating disorder, executive, FrSBe, prefrontal

Received 31 May 2003.

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Executive functions play an important role in regulating a wide array of behaviors. Prefrontal-subcortical systems, involving the striatum and thalamic nuclei, are structures critical for mediating executive functions (Masterman & Cummings, 1997). Different subregions of prefrontal cortex are recognized to mediate different types of executive functioning: medial prefrontal cortex (mPFC) mediates initiation and persistence of behavior (Cohen, 1999; Devinsky, Morrell, & Vogt, 1995), dorsolateral prefrontal cortex (dlPFC) mediates such functions as conceptual reasoning, mental flexibility, and planning (Masterman & Cummings, 1997), and orbitofrontal cortex (OFC) mediates self-inhibition, social conduct, empathy, and decision making (Malloy, Bihrle, Duffy, & Cimino, 1993).

Eating disorders, including anorexia nervosa, bulimia nervosa, and binge eating disorder, are conditions that involve a dysregulation of eating behavior. Anorexia involves a failure to maintain minimum body weight, fear of gaining weight, and a disturbance of body image (American Psychiatric Association, 1994). Anorexic individuals tend to experience hunger, but control their weight by excessive restriction of eating or purging through means of vomiting or laxative abuse.

Bulimia nervosa involves repeated bingeing, followed by compensatory behaviors to avoid weight gain. After obtaining the hedonic effects of eating, such individuals often feel disgusted, guilty, and depressed. They may purge (e.g. vomiting, laxatives, enemas) or engage in non-purging, compensatory behaviors (e.g. exercise, fasting) to control their weight. In binge-eating disorder, individuals may eat in binges as in bulimia nervosa, but do not heavily engage in compensatory behaviors. Instead, obesity becomes a greater problem for this population. People with eating disorders are also more likely to engage in other maladaptive impulsive and compulsive behaviors, suggesting a more pervasive problem of behavioral control (Eddy, Keel, Dorer, Delinsy, Franko, & Herzog, 2002; McElroy, Keck, & Phillips, 1995).

Several lines of research indicate that the human prefrontal cortex plays a pivotal role in the control of eating behavior. While the hypothalamus is most frequently studied in relation to eating, prefrontal-subcortical networks regulate eating, via connections with the hypothalamus (Martin & Riskind, 1992; Ongur & Price, 2000). Neuro-imaging studies indicate that PFC, particularly OFC, play a

role in the reinforcing value of food. Taste and olfactory processing occur in OFC, with activity representing sensory-specific satiety and the control of eating (Rolls, 1997). Aversive tastes, such as a strong saline taste, activates the left anterior orbitofrontal cortex (Zald, Hagen, & Pardo, 1998). The pleasurable taste of chocolate activates the caudomedial region of the orbitofrontal cortex, where activity diminishes as the pleasantness decreases after repeated eating (Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001).

Functional neuro-imaging studies also show representations of hunger and satiety in prefrontal-subcortical systems (Tatranni et al., 1999; Del Parigi et al., 2002). Functional neuro-imaging studies also have shown differences in people with eating disorders. For example, obese men show greater activation associated with satiety in prefrontal-subcortical and limbic circuits when compared to controls (Gautier et al., 2000). Bulimic subjects show greater left prefrontal activation when exposed to food when compared to controls, which correlated with food cravings and hunger (Karhunen et al., 2000). The length of recovery in bulimia nervosa inversely correlated with activation in several prefrontal areas (Frank, Kaye, Greer, Meltzer, & Price, 2000). Within bulimic subjects, greater hemispheric asymmetry was noted in the binge eating state when compared to the anorexic, food-restricting state (Hirano, Tomura, Okane, Watarai, & Tashiro, 1999).

Cerebral glucose metabolism is elevated in inferior frontal cortex and basal ganglia in anorexic patients compared to those with bulimia (Delvenne, Goldman, De Maertelaer, & Lotstra, 1999). Caudate hyperactivity has also been noted in individuals during an anorexic state (Herholz, 1996; Krieg, Holthoff, Schreiber, Pirke, & Herholz, 1991). In contrast, weight-restored anorexic subjects show hypoperfusion in orbitofrontal cortex (Rastam et al., 2001). Anorexic patients showed an increase in rCBF in multiple brain regions, including interior frontal cortex, when comparing scans taken before and after eating (Nozoe et al., 1995). In contrast, patients with bulimia nervosa showed a decrease in multiple regions. These functional differences are not necessarily restricted to individuals with clinical severity. Even restrained eaters show greater right frontal electrophysiological activity when compared to unrestrained eaters (Silva, Pizzagalli, Larson, Jackson, & Davidson, 2002).

Eating behavior is also disturbed in several neurological conditions that involve prefrontal-subcortical systems. Traumatic brain injury (TBI) has a propensity to affect frontotemporal structures, and can induce a lack of self-restraint in eating that is resistant to behavior modification and appetite suppressants (Childs, 1987). Neoplastic lesions in the right frontal lobe have been associated with anorexia nervosa (Trummer, Eustacchio, Unger, Tillich, & Flaschka, 2002). Also, a case was reported regarding a putamen lesion associated with anorexia nervosa and obsessive-compulsive disorder (Hebebrand, Siemon, Lutcke, Mari, & Remschmidt, 1993). People with frontal variant frontotemporal dementia often manifest gluttonous eating and sweet food preference, suggesting greater responsiveness to the hedonic properties of food and lack of regulation of eating according to homeostatic signals (Bathgate, Snowden, Varma, Blackshaw, & Neary, 2001).

Thus, human prefontal systems and executive functions are logical areas to study in the context of eating and eating disorders. While people with eating disorders may show a greater degree of executive or prefrontal dysfunction, compared to the normal population, it is likely that these disorders represent the extremes of a normal continuum. This study examined the relationship between prefrontal-associated traits and eating behaviors using self-report inventories in a community sample.

METHODS

Participants

Participants were a convenience sample (n = 112; 80 female, 32 male) of community-dwelling individuals recruited by volunteer research assistants, who did not receive any financial compensation for participating. The study was approved by an institutional review board and all subjects agreed to an implied consent form, in accordance with the Declaration of Helsinki and the ethical principles of the American Psychological Association. Responses on the psychometric measures were sealed by the participants in an envelope

before returning them, so that responses were anonymous and confidential. Participants ranged in age from 15 to 55 years (mean 25.1 ± 10.1 years), and had completed between 11 and 19 years of education (mean 14.5 ± 1.7 years).

Measures

Frontal Systems Behavior Scale

The Frontal Systems Behavior Scale (FrSBe) is an instrument that measures neurobehavioral traits associated with regions of the prefrontal cortex (Grace, Stout, & Malloy, 1999). The FrSBe is a self-rating scale of 46 items that yields scores for three scales of dysfunction: apathy (FrSBeA; e.g., "I sit around doing nothing."), disinhibition (FrSBeD, e.g., "I do risky things just for the heck of it."), and executive dysfunction (FrSBeE, e.g., "I mix up a sequence, and get confused when doing several things in a row."), in addition to the total score (FrSBeT). Based on the neurobehavioral syndromes produced by prefrontal lesions, these scales were designed to measure neurobehavioral syndromes associated with medial prefrontal, orbitofrontal, and dorsolateral prefrontal cortex, respectively. An adaptation of the FrSBe was used to measure prefrontal-associated traits in this study. Whereas the original version was designed for clinical populations and asks for pre- and post-injury ratings for each item, participants in this study were only asked for one global self-rating per item. The normative data for the instrument indicate that all scales of the FrSBe have been noted to vary with age, sex, and level of education.

Reliability studies of the FrSBe have shown high intrascale reliability in normal and clinical samples, and factor analytic data support the validity of the three scales (Grace et al., 1999; Grace & Malloy, 2001). Validity of the instrument is further supported by three lines of evidence: (1) people with frontal lobe injuries have higher scores compared to their pre-injury status, (2) people with frontal lobe injuries score higher than healthy controls, and (3) people with frontal lobe injuries score higher than those with nonfrontal injuries. The instrument has also shown validity in psychiatric and dementia populations.

Eating Inventory

The Eating Inventory (EI) is a measure of eating behaviors developed by factor analysis (Stunkard & Messick, 1985). Three factors have emerged: hunger (EIh), disinhibition (EId), and cognitive restraint (EIcr). EIh measures the level of desire for food experienced by an individual, while EId measures the person's sense of loss of control over eating. EIcr is the element of thought or cognition involved with control of eating. Binge eating is strongly related to the EId scale (Keel, Mitchell, Miller, David, & Crow, 2000; De Zwaan, Mitchell, & Seim, 1994), while restricting behaviors are more closely associated with EIcr. However, binge eating, in particular, may have different characteristics among different subgroups within one diagnosis. For example, binge behavior may be related to dietary control in some bulimics, while these variables may be unrelated in impulsive bulimics (Steiger, Lehoux, & Gauvin, 1999).

RESULTS

Bivariate correlations using the two-tailed significance levels were performed to examine the influences of age and education on the FrSBe and EI, and point-biserial correlations were performed to determine the influence of sex. EIcr correlated inversely with sex ($r = -.25$, $p = .009$), suggesting greater cognitive restraint in females (M = 1, F = –1). EIcr correlated inversely with education ($r = .22$, $p = .019$). EId did not correlate with age, sex, or education in this sample, but EIh correlated inversely with age ($r = -.19$, $p = .047$). Consistent with the normative data for the instrument, FrSBe scores correlated inversely with age: FrSBeT ($r = -.37$, $p < .001$). Scores also correlated positively with sex, indicating higher scores in males: FrSBeT ($r = .23$, $p = .016$). Education correlated negatively with FrSBEA ($r = -.21$, $p = .029$).

Thus, partial correlations were performed between EI and FrSBe subscales to remove the influence of age, sex, and education. On subscales of the EI, intercorrelations were noted between the EIcr and EId subscales ($r = .28$, $p = .003$), and between the EIh and EId subscales ($r = .55$, $p < .001$). Consistent with past research on the

FrSBe, all subscales intercorrelated, even after partialling out the effects of age, sex, and education (df = 107): FrSBeA and FrSBeD (r = .42, p < .00l), FrSBeA and FrSBeD (r = .60, p < .001), FrSBeD and FrSBeE (r = .61, p < .001).

Several significant correlations were observed between subscales of the EI and FrSBe, even after removing the influences of age, sex, and education (see Table 1). EId and EIh showed significant positive correlations with all subscales and the total score of the FrSBe. EIcr also showed all positive correlations, but only the correlation with FrSBeD reached significance. Adjusting the alpha level for multiple comparisons (e.g., to .005) would cause a few of these results to fall below significance. However, the pattern of overall results would essentially remain the same, suggesting the difference is more likely a matter of sample size.

DISCUSSION

Consistent with past research on these instruments, demographic variables showed an influence on both executive functions and eating behavior. Nonetheless, significant relationships in the anticipated

TABLE 1. Partial correlations between Frontal Systems Behavior Scale (FrSBe) and Eating Inventory (EI) subscales (controlling for age, sex, and education; n = 112, df = 107); significant correlations are boldfaced

	EIcr	*EId*	*EIh*
FrSBeA			
r	0.11	**0.30**	**0.25**
p	0.262	**0.001**	**0.010**
FrSBeD			
r	**0.20**	**0.23**	**0.38**
p	**0.033**	**0.016**	**<0.001**
FrSBeE			
r	0.13	**0.30**	**0.26**
p	0.173	**0.001**	**0.006**
FrSBeT			
r	0.18	**0.33**	**0.36**
p	0.062	**<0.001**	**<0.001**

cr = cognitive restraint, d = disinhibition, h = hunger, A = apathy, D = disinhibition, E = executive dysfunction, and T = total score.

direction were observed even afterward for controlling for demographic influences. Perhaps the most expected finding is the positive correlations between the EId scale and all FrSBe scales. Executive dysfunction is associated with a loss of self-inhibition, which would extend to multiple areas of behavior, including eating. However, this relationship is not limited to overall behavioral disinhibition (FrSBeD), but rather related to apathy (FrSBeA) and executive dysfunction (FrSBeE) as well. These suggest that prefrontal system dysfunction relationships regarding eating are not specifically limited to disinhibition, but may accompany more widespread symptoms.

The relationship of hunger (EIh) to FrSBe scales also agrees with a role for prefrontal systems in desire for food or cravings. While homeostatic aspects of hunger are regulated by the hypothalamus, prefrontal interactions with the hypothalamus can mediate the reward aspects of eating, such as food cravings (Martin & Riskind, 1992; Ongur & Price, 2000). There are ample neuro-imaging studies to support this (Rolls, 1997; Small et al., 2001, Tatranni et al., 1999; Del Parigi et al., 2002). Indeed, prefrontal involvement in cravings is not limited to food, and may apply to all rewarding stimuli, including drugs (Volkow et al., 1999).

Cognitive restraint of eating (EIcr) also related positively to behavioral disinhibition (FrSBeD). On the surface, these two constructs may seem antithetical; cognitive restraint refers to increased self-control, while disinhibition refers to a lack of self-control. It is important to recognize, however, that the cognitive restraint measured here is limited to eating behavior. Further, a higher degree of cognitive restraint does not necessarily address the issue of how consistent these behaviors may be. A person could need to exert more cognitive restraint in order to compensate for greater disinhibition of eating, i.e., yo-yo dieting. Agreeing with this interpretation is the fact that the EIcr and EId scales correlate with each other positively, regardless of relationships with the FrSBE, and even after controlling for demographic variables. People with greater eating disinhibition and hunger (EId and EIh) tend to exercise greater cognitive restraint of eating (EIcr). It is worthwhile to note that FrSBeD was the only FrSBe scale to correlate significantly with cognitive restraint (EIcr), although the other scales also had lower, positive, but nonsignificant correlations. It is appropriate that disinhibition is

the aspect of prefrontal function most relevant to exerting cognitive restraint in eating.

These findings support those of other methodologies supporting a role for prefrontal systems in regulating eating behavior. However, the interpretations of this study are limited by the self-rating methodology. While self-report inventories cannot be used conclusively to make anatomical distinctions in behavior, they provide a sound psychometric complement to existing clinical and neuroimaging research. In particular, the FrSBe has shown good validity towards measuring prefrontal system function in clinical populations. Further, while many studies addressing eating and prefrontal function have been done in clinical populations (e.g., neurological disorders and eating disorders), these findings help extend the relationship into the normal population. Thus, it is possible that executive strategies for behavioral control will be useful in the treatment of eating disorders and regulating eating in many who do not reach the severity of eating disorders, but who still have maladaptive eating habits. Further research along these lines is warranted.

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