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Research report

Evidence that 'food addiction' is a valid phenotype of obesity

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ABSTRACT

There is growing evidence of 'food addiction' (FA) in sugar- and fat-bingeing animals. The purpose of this study was to investigate the legitimacy of this disorder in the human condition. It was also our intention to extend the validation of the *Yale Food Addiction Scale* (YFAS) – the first tool developed to identify individuals with addictive tendencies towards food. Using a sample of obese adults (aged 25–45 years), and a case–control methodology, we focused our assessments on three domains relevant to the characterization of conventional substance-dependence disorders: clinical co-morbidities, psychological risk factors, and abnormal motivation for the addictive substance. Results were strongly supportive of the FA construct and validation of the YFAS. Those who met the diagnostic criteria for FA had a significantly greater co-morbidity with Binge Eating Disorder, depression, and attention-deficit/hyperactivity disorder compared to their age- and weight-equivalent counterparts. Those with FA were also more impulsive and displayed greater emotional reactivity than obese controls. They also displayed greater food cravings and the tendency to 'self-soothe' with food. These findings advance the quest to identify clinically relevant subtypes of obesity that may possess different vulnerabilities to environmental risk factors, and thereby could inform more personalized treatment approaches for those who struggle with overeating and weight gain.

Introduction

Although the concept of *food addiction* (FA) is widely accepted among the general public – as seen by the many 12-Step treatment programs established for those who struggle with overeating – it has only recently gained some credibility in the scientific community. For example, there has been a steeply exponential increase in the number of academic publications relating to this putative condition over the past 5 years (Gearhardt, Davis, Kuschner, & Brownell, in press).[1] During the same period, there has also been a noticeable shift in perspective towards the view that addictions should be re-framed as unusually strong and maladaptive desires for pleasure (or reduction of distress), irrespective of the source of the reward (Hoebel, Avena, Bocarsly, & Rada, 2009). The current interest in FA as a clinically valid construct has undoubtedly been fostered by the worldwide shift in body mass index (BMI) towards the high end of the distribution, and by the concomitant *per capita* increase in daily calorie intake (Apovian, 2010).

The foods we most prefer to eat are those high in fat, sugar, and salt – substances which were generally found in small amounts, and intermittently, in the diets of our evolutionary ancestors (Armelagos, 2010). However, in the quantities we currently consume these hyper-palatable foods they appear to have an abuse potential similar to conventional addictive drugs (Gearhardt et al., in press; Kenny, 2011; Spring et al., 2008). Potent sweeteners such as high fructose corn syrup (HFCS) are particularly influential because they have been added voluminously to a large variety of processed foods including soft drinks, baked goods, and cereals (Bray, 2008). Indeed, our consumption of this monosaccharide has increased considerably in a few generations, from about 4% to 12% of our daily caloric intake (Vos, Kimmons, Gillespie, Welsh, & Blanck, 2008). Moreover, the special physiological properties of fructose enhance its similarity to other addictive drugs. The most direct parallels are seen between fructose and alcohol because the two are biochemically congruent – ethanol is simply the fermented byproduct of fructose (Lustig, 2010). Different from glucose, fructose blunts leptin signaling thereby promoting sensations of hunger and creating a desire for consumption independent of energy needs (Li, Li, Kong, & Hu, 2010). This seems to occur because fructose bypasses the insulin-driven satiety system. In other

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[1] Admittedly, research on the subject of compulsive overeating has been ongoing for many years, however, it is only recently that scientists have begun to openly use the term "food addiction" in their writing.

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words, while glucose stimulates the release of insulin, decreasing the desire to eat, fructose has this effect only to a very weak degree. Consequently, when eaten in abundance, fructose can result in biological changes that promote overconsumption and encourage further problematic use (Bocarsly, Powell, Avena, & Hoebel, 2010).

Compulsive overeating

Few would dispute that some individuals display an apparent 'loss of control' over food intake that is similar to the behaviour of those who abuse drugs and alcohol. We have previously argued that the compulsive overeating seen in Binge Eating Disorder (BED) has compelling parallels to conventional drug addictions based on their comparable clinical features, the biological mechanisms they have in common, and evidence of a shared diathesis (Davis & Carter, 2009). This perspective also aligns with some qualitative evidence that a high proportion of women diagnosed with BED endorse the criteria for drug dependence when the word 'substance' refers to binge eating instead of drugs (Cassin & von Ranson, 2007).

While early discussions described the mood enhancing effects and pronounced cravings that compulsive overeating has in common with drug abuse (Rogers & Smit, 2000), evidence of their biological parallels is more recent, and has relied largely on well-controlled animal research. Rodent models of FA have typically used behavioural paradigms based on analogues of the *Diagnostic and Statistical Manual* [DSM-IV-TR] (American Psychiatric Association, 2000) criteria for drug dependence. For example, escalation of intake is used as a marker of "taking the substance in larger amounts than was intended" (Corwin & Grigson, 2009). There is now reliable evidence that rats fed an intermittent diet of sugar, develop a pattern of copious consumption resembling human cases of binge eating (Avena & Hoebel, 2003).

These studies show that a sugar-enhanced diet increases daily food intake over time, and that following its removal, the animals show aggression, anxiety, teeth-chattering, and head-shaking – all symptoms associated with withdrawal from drugs like heroin. Similar results have been found when animals were given high-fat (Lutter & Nestler, 2009) or other highly palatable diets (Johnson & Kenny, 2010). Female rats prone to binge eating were also found to tolerate significantly higher levels of foot shock for access to Oreo cookies than their binge-resistant counterparts, confirming their abnormal motivation for sweet and fatty foods (Oswald, Murdaugh, King, & Boggiano, 2011). In addition, while numerous studies have found behavioural and consummatory cross-sensitization from one addictive drug to another (Avena, Rada, & Hoebel, 2008) – and between drugs and stress (Covington & Miczek, 2001) – there is now good evidence that sugar intake also cross-sensitizes to drugs of abuse, and *vice versa* (Avena et al., 2008).[2]

Despite the robust evidence of sugar/fat dependence in rodents, there are few parallel findings in human research. Some notable exceptions are the work of Pelchat (2009) who identified brain areas responsive to both food and drug cravings. Other important research has demonstrated the abuse potential of sugar, based on laboratory indicators similar to those used to test the abuse liability of drugs (Spring et al., 2008). Researchers at Yale University have recently moved the field forward by developing a measure to operationalize human cases of FA using the DSM-IV diagnostic criteria for substance dependence (Gearhardt, Corbin, & Brownell, 2009). Their preliminary evidence suggests that the *Yale Food Addiction Scale* (YFAS) has high convergent validity with other measures of eating pathology – especially binge eating – and may therefore be a useful tool to identify those with addictive tendencies towards food. In a recent neuroimaging study, they also found that YFAS scores correlated with neural activation in brain regions that play a role in the experience of cravings, and that high scorers exhibited activation patterns associated with reduced inhibitory control (Gearhardt et al., 2011). However, this study was somewhat limited by a small all-female sample, and because only two participants met the diagnostic criteria for FA.

The purpose of the present study was to provide further support for the FA construct, and to extend the validation of the YFAS by using a non-clinical sample of obese adults. We focused our assessments on three domains relevant to the characterization of conventional addiction disorders: clinical co-morbidities, psychological risk factors, and abnormal motivation for the addictive substance. In the first instance, we anticipated a higher prevalence of BED in those meeting criteria for FA, as well as more severe symptoms of depression and attention deficit/hyperactivity disorder since both these latter conditions have strong co-morbid links to drug abuse (Carpentier, van Gogh, Knapen, Buitelaar, & De Jong, 2011; de los Cobos et al., 2011; Fuemmeler, Kollins, & McClernon, 2007). In addition, almost a decade of research has established strong links between ADHD and obesity, both in adults, and in children and adolescents, and it appears that this relationship is not attributable to socio-demographic factors that influence people's dietary patterns and opportunity for physical activity (see Davis, 2010). We also expected food addicts – like drug addicts – to be more impulsive and to have higher scores on a measure of addictive personality traits (e.g. Gullo, Ward, Dawe, Powell, & Jackson, 2011). Finally, given the high incentive salience of 'drugs' to the drug addict (George & Koob, 2010), we hypothesized that food addicts would also report a greater hedonic motivation for food and be more likely to overeat in response to emotional and environmental triggers in the absence of hunger, when compared to their non-FA counterparts.

Methods

Participants

Seventy-two obese adult women ($n = 49$) and men ($n = 23$) between the ages of 25 and 46 years took part in the study. Participants were required to be fluent in English and to have lived in North America for at least 5 years prior to their enrolment. All female participants were also required to be pre-menopausal as identified by the self-reporting of regular menstrual cycles, and not to have had a pregnancy within the previous 6 months. Exclusion criteria included a current diagnosis of any psychotic disorder, substance abuse, alcoholism, or a serious medical/physical illness such as cancer, heart disease, or paralysis. Participants were recruited from posters placed at universities, local hospitals, and other public institutions soliciting volunteers who were "over-eaters and overweight". Advertisements were also placed in local newspapers and online sites like CraigsList. The procedures employed in this study were approved by the three Research Ethics Boards relevant to the institutional affiliations of the authors, and were carried out in accordance with the Declaration of Helsinki. As an initial step in the screening procedure, a short telephone interview was carried out to confirm basic eligibility criteria.

Clinical measures

Food addiction was assessed by the 25-item *Yale Food Addiction Scale* [YFAS] (Gearhardt et al., 2009), which was designed to

[2] Several recently published reviews provide more detailed and comprehensive information about the physiological underpinnings of the behaviours observed in these valuable animals models of food addiction (e.g. Avena, 2010; Avena et al., 2008; Blumenthal & Gold, 2010; Corwin & Grigson, 2009).

operationalize food addiction according to the 7 symptoms of substance dependence listed in the DSM-IV (e.g. withdrawal, tolerance, continued use despite problems), and modified for eating behaviours. The YFAS provides two scoring options – a dichotomous and a continuous version. Similar to the DSM substance-dependence criteria, a 'diagnosis' of FA is given if the respondent experienced 3 or more symptoms over the past year, and if the "clinically significant impairment" criterion was met. The dimensional score is the total number of symptoms endorsed and ranges from 0 to 7. For this sample, the Cronbach alpha coefficient was 0.92.

Binge Eating Disorder [BED] was diagnosed using ratings on the *Eating Disorder Examination* (Fairburn & Cooper, 1993). This definition was based on that provided in the main body of the DSM-IV where BED is defined as: "recurrent episodes of binge eating in the absence of the regular use of inappropriate compensatory behaviours characteristic of bulimia nervosa" (p. 550). This definition was operationalized in the following way. Participants had to report at least weekly objective binge episodes over the previous 6 months, but over this period they must not have vomited, fasted, or taken laxatives or diuretics as a means of controlling their shape or weight. Nor must they have met DSM-IV diagnostic criteria for bulimia nervosa or anorexia nervosa. BED diagnosis was established during a face-to-face interview carried out by trained clinical personnel.

Depression severity was assessed by the 21-item, *Beck Depression Inventory*, *2nd Edition* [BDI-II] (Beck, Steer, & Brown, 1996). Scores range from 0 to 63 with categorical depression ratings of *minimal* (0–13), *mild* (14–19), *moderate* (20–28), and *severe* (29–63). Although the questionnaire was designed to assess depression severity, it is often used for the purpose of diagnosis because of its consistency with the DSM-IV diagnostic criteria (Dozois & Covin, 2004). The Cronbach alpha coefficient for this sample was 0.93.

ADHD symptoms were assessed by two self-report inventories. The 5-point *Wender Utah Rating Scale* [WURS-25] (Ward, Wender, & Reimherr, 1993) comprises the best 25 items (from a larger selection of items) discriminating between ADHD and controls, and assesses the frequency of childhood ADHD symptoms and behaviours retrospectively. According to the developers of this scale, a cut-off score of 36 is 96% sensitive and specific for identifying childhood ADHD in the general population. The Cronbach alpha coefficient for this sample was 0.93. Adult ADHD symptoms were assessed by the *Conners' Adult ADHD Rating Scale – Self-report Screening Version* (CAARS-SSV: Conners, Erhardt, & Sparrow, 1999). The questionnaire consists of two scales. The *Inattentive* and the *Hyperactive/Impulsive* subscales (9-items each) reflect the symptoms listed for the two ADHD subtypes of the same name in the DSM-IV-TR. Cronbach alpha coefficients for the two subscales were 0.86 and 0.81 respectively.

Personality measures

Impulsivity was assessed by the 30-item *Barratt Impulsivity Scale* [BIS] (Patton, Stanford, & Barratt, 1995), which identifies three factors of impulsivity: the non-planning aspects of this construct, as well as the tendency for one to act rashly, and to make quick decisions. Currently, this is the most widely used self-report measure of trait impulsivity. The highly significant correlations among the factors ($r = 0.52–0.54$), and the high alpha coefficient in this study (0.86) for all 30 items, provide a good justification for our use of the total score in the statistical analyses.

Impulsivity was also assessed by two computerized behavioural measures. The *Delay Discounting Task* (Richards, Mitchell, de Wit, & Seiden, 1997; Richards, Zhang, Mitchell, & de Wit, 1999) assesses the value of immediate rewards relative to delayed rewards. It is based on the evidence that more impulsive individuals prefer more immediate rewards (Crean, de Wit, & Richards, 2000; Monterosso & Ainslie, 1999), and they will discount the value of reinforcers more steeply in time. Several questions are posed to participants asking whether they would prefer to receive a given amount of money immediately (e.g. $65.00), or $100 in a certain number of days. The amount of money available immediately is adjusted with each trial to calculate an indifference point for five delay periods (2, 30, 180, 365, and 730 days). The indifference point indicates the amount of immediate money deemed equal to the $100 delayed reward. Those who are less able to delay gratification are expected to have lower indifference points as this means they are willing to accept less money immediately rather than wait for a larger amount at a later date. In this study, the total of the 5 indifference scores was used as an index of impulsivity. The alpha coefficient for these 5 variables was 0.90.

The *Delay of Gratification Task* (Newman, Kosson, & Patterson, 1992) provides a measure of participant's willingness to wait for a monetary reward. The participant responds by pressing either of two buttons: one associated with immediate reward with a relatively low probability; the other associated with a delay but also a higher probability of reward. Participants get immediate feedback (either "you win" or "you lose") and their accumulated earnings appear on the screen. The primary dependent variable is the proportion of trials on which the participant choses the delayed option. Lower scores reflect greater impulsivity.

Addictive personality traits were assessed by the 32-item *Addiction Scale* of the *Eysenck Personality Questionnaire-Revised* [EPQ-R] (Eysenck & Eysenck, 1975). This scale was derived empirically to measure addiction proneness by identifying those items of the EPQ-R, at or beyond the 0.001 level of significance, which differentiated drug addicts from normal controls (Gossop & Eysenck, 1980). This scale has also been validated with groups of problem drinkers (Ogden, Dundas, & Bhat, 1988) and pathological gamblers (Clarke, 2003). The alpha coefficient in the present study was 0.74.

Eating behaviour measures

Binge eating was assessed by 5-items of the *Binge Eating Questionnaire* (Halmi, Falk, & Schwartz, 1981), which obtains information about frequency and severity of symptoms such as loss of control over eating, and negative affect following a binge (e.g. "are there times when you cannot voluntarily stop eating?"). The alpha coefficient for this study was 0.76.

Hedonic eating was assessed by the *Power of Food Scale* (Lowe et al., 2009), a 21-item self-report questionnaire that reflects individual differences in the appetitive responsiveness to food in environments replete with highly palatable food – independent of the amount that is eaten. In other words, it differentiates the motivation and appetitive drive to obtain food from the tendency to (over)eat food. As such, there are no items in the questionnaire that describe food consumption. Based on a factor analysis of the items, Lowe et al. concluded that a one-factor solution was most appropriate. Accordingly, the Cronbach's alpha coefficient in this study was very high (0.97).

Emotional eating and externally driven eating were assessed by the *Dutch Eating Behaviour Questionniare* [DEBQ] (Van Strien, Frijters, Bergers, & Defares, 1986). The *Emotional Eating* subscale reflects the degree to which eating is prompted by emotional states like tension and worry rather than by hunger; and the *External* subscale, the degree to which one tends to overeat prompted by the sight and smell of food. The alpha coefficients in the present study for Emotional Eating and External Eating were 0.96 and 0.80, respectively.

Food cravings were assessed by the *Food Craving Questionnaire – Trait* (Cepeda-Benito, Gleaves, Williams & Erath, 2000). This 39-item questionnaire derives from the evidence that food cravings can be expressed both physiologically and psychologically. The nine factor-analytically derived scales measure cravings experienced as, or associated with (i) positive reinforcement, (ii) negative reinforcement, (iii) cue-dependent eating, (iv) feelings of hunger, (v) preoccupation with food, (vi) intentions to eat, (vii) lack of control, (viii) negative affect, and (ix) guilty feelings. The total-item score was used in this study and provided an alpha coefficient of 0.97.

Snacking on sweets was assessed by the 6-item subscale of the *Eating Behaviours Patterns Questionnaire* (Schlundt, Hargreaves, & Buchowski, 2003), which was designed to measure the frequency and quantity of sugary snacks consumed between normal meals. The alpha coefficient in the present study was 0.77.

Procedures

On the day of testing, informed consent was obtained, and all relevant demographic information obtained in a face-to-face interview. A structured clinical interview was also carried out to confirm eligibility, and height and weight were measured with the participant standing in stocking feet and wearing light indoor clothing. The questionnaire package was completed at home and returned at a later date. All subjects were paid a stipend for their participation.

Results

Food addiction diagnosis

According to the YFAS diagnostic scoring procedure, which is based on the DSM-IV criteria for substance dependence, 18 adults (female = 13; male = 5) were classified as food addicts.

Demographic characteristics: Compared to their non-food addict counterparts (female = 36; male = 18), the group of food addicts did not differ significantly in age or BMI. Nor was there a different gender ratio, ethnic composition, or education level between the two groups (see Table 1).

Clinical features: The groups were also compared on relevant clinical co-morbidities. Among the food addicts, a significantly greater proportion was diagnosed with BED and had a higher prevalence of severe depression as indicated by the Beck Depression Inventory categorical scoring. A greater proportion of this group also met the criteria for a probable diagnosis of childhood ADHD according to the WURS cut-off score (see Table 2). Finally, the food addict group had significantly higher mean scores (12.8 ± 5.5; 11.4 ± 5.7) than the obese controls (7.1 ± 4.6; 7.6 ± 4.1) on the CAARS Inattentive ($p < 0.0001$) and Hyperactive/Impulsive ($p = 0.005$) scales respectively.

Personality traits: Multivariate Analysis of Variance (MANOVA) was used to compare food addicts and non-food addicts on relevant personality characteristics. Impulsivity was assessed both by self-report and by two computerized behavioural tasks. Participants were also assessed on a questionnaire measure of addictive personality traits. The multivariate F ratio generated from Pillai's statistic was statistically significant ($F_{4,65} = 5.18$, $p = 0.001$), as were the four univariate F values (see Table 3). Results indicated that food addicts reported more impulsive traits on the BIS, and showed poorer performance on the delay of gratification and delay discounting tasks. They also had significantly higher scores on the measure of addictive personality traits.

Eating behaviours: A second MANOVA compared the two groups on six behaviours related to overeating. Again, the multivariate F ratio ($F_{6,64} = 8.70$, $p \leq 0.0001$) and all the univariate F values – except for External Eating – were highly statistically significant (see Table 4). Food addicts reported more binge eating, hedonic eating, and emotionally driven eating. They also reported greater food cravings and snacking on sweets.

Food addiction symptoms

As a further examination of the FA construct we also used the symptom-count version of the YFAS scoring, which provides a continuous measure of symptom severity. Stepwise regression analysis was used to find the set of independent variables that best explained the variance in symptom scores. The personality variables were entered in the first block, and the five, previously significant, eating-behaviour variables were entered in the second block. Table 5 summarizes the findings of this analysis, and indicates that in the final model four variables reached statistical significance, collectively accounting for 56% of the variance in FA symptoms. Greater symptom severity was associated with more pronounced addictive personality traits, and with greater hedonic eating, snacking on sweets, and binge eating.

Table 1
Participant characteristics listed separately for food addicts ($n = 18$) and non-food addicts ($n = 54$).

Characteristic	Food addict	Non-food addict	p
Age in years	35.3	33.0	0.191
BMI	37.5	38.8	0.460
% University Degree	61.1	42.6	0.173
% Female	72.2	66.7	0.662
% Smoker	33.3	25.9	0.543
% Caucasian	83.3	80.8	0.809

Table 2
Differences between food addicts and non-food addicts, and associated p-values, for relevant clinical symptoms.

Disorder	Food addict Percentage	Non-food addict Percentage	χ^2 (p-value)
Binge Eating Disorder	72.2	24.1	13.56 (<0.0001)
Severe depression	27.8	3.8	10.12 (0.018)
Childhood ADHD	33.3	11.1	4.80 (0.028)

Table 3
Means, standard deviations, and p-values for the differences between food addicts and non-food addicts on the personality variables.

Variable	Food addict Mean (SD)	Non-food addict Mean (SD)	p
Impulsivity	73.2 (12.0)	62.2 (10.5)	<0.0001
Addictive traits	16.2 (6.2)	12.5 (3.7)	0.003
Delay of gratification	35.9 (22.1)	47.8 (19.7)	0.036
Delay discounting (total)	231.7 (138.2)	306.5 (123.2)	0.035

Table 4
Means, standard deviations, and p-values for the differences between food addicts and non-food addicts on the eating-behaviour variables.

Variable	Food addict Mean (SD)	Non-food addict Mean (SD)	p
Binge eating	4.2 (1.2)	2.1 (1.5)	<0.0001
Hedonic eating	84.5 (15.4)	56.7 (18.8)	<0.0001
Emotional eating	4.2 (0.6)	3.1 (1.0)	<0.0001
External eating	3.6 (0.5)	3.4 (0.6)	0.193
Food cravings	177.8 (28.2)	127.2 (32.3)	<0.0001
Snacking on sweets	22.8 (5.0)	18.0 (4.2)	<0.0001

Table 5
Hierarchical regression analysis summary for personality and eating-behaviour variables predicting symptoms of food addiction.

Variable	*B*	SEB	*t*	*p*	R^2
Step 1					
Addictive traits	0.22	0.05	4.80	<0.0001	0.26
Step 2					
Addictive traits	0.15	0.04	3.60	0.001	0.48
Hedonic eating	0.05	0.01	5.33	<0.0001	
Step 3					
Addictive traits	0.13	0.04	3.05	0.003	
Hedonic eating	0.04	0.01	4.10	<0.0001	0.52
Snacking on sweets	0.10	0.04	2.35	0.022	
Step 4					
Addictive traits	0.11	0.04	2.26	0.027	0.56
Hedonic eating	0.02	0.01	2.40	0.050	
Snacking on sweets	0.12	0.04	2.00	0.002	
Binge eating	0.34	0.14	2.46	0.017	
Excluded variables					
Impulsivity			−1.22	0.228	
Delay discounting			−0.27	0.787	
Delay of gratification			1.06	0.133	
Food cravings			0.83	0.411	
Emotional eating			1.03	0.309	

Discussion

Our findings provide very good validation for the YFAS and its ability to identify individuals with addictive tendencies towards food. In so doing, they strongly reinforce the view that FA is a classifiable condition with clinical symptomatology and a psycho-behavioural profile similar to conventional drug abuse disorders. These results also deliver much needed *human* support for the growing evidence of addiction in sugar- and fat-bingeing rats (Avena, 2010). Importantly, our findings are not confounded by any demographic differences – especially those of age and BMI – between the FA and non-FA groups in our study.

Clinical evidence

Although we found considerable overlap between FA and BED, as anticipated, it was also clear that the two conditions are not indistinguishable. For example, half of those with BED did not meet the diagnostic criteria for FA, and close to 30% of the FA group were not clinically significant binge eaters. This outcome suggests – similar to other drug and alcohol addictions – that bingeing is just one form of excessive intake and not the only consumption pattern leading to dependence and impairment. Chronic and copious use over time may also perturb neural circuitry, and foster feelings of dependence and loss-of-control (Koob & Volkow, 2010).

In parallel with other addiction disorders, there was also more severe depression and greater childhood symptoms of ADHD in the FA group compared to their non-FA counterparts. In community samples, the prevalence of ADHD is about 25% in those with drug dependence (Kessler et al., 2006) – a value roughly equivalent to the percentage of food addicts who met the diagnostic cut-off score for childhood ADHD. These associations mesh well with the view that a common underlying process contributes to the increased risk for disorders like depression, ADHD, and the addicted brain – all of which are known to be mesolimbic dopamine-dyregulated conditions (Rodrigues, Leao, Carvalho, Almeida, & Sousa, 2011). While low brain dopamine levels may be an inherited trait in some individuals, there is also very good evidence that prenatal and early postnatal life present "windows of susceptibility" to the deleterious effects of stress (in the form of abuse and neglect) on the brain development crucially necessary for optimal dopaminergic functioning (Meaney, Brake, Gratton, 2002; Rodrigues et al., 2011).

Personality evidence

Strong links have been established between *impulsivity* and a host of drug and behavioural addictions (Brewer & Potenza, 2008; Crews & Boettiger, 2009; Davis, 2010; van Holst, van den Brink, Veltman, & Goudriaan, 2010). While deficits in self-regulation are at the heart of 'impulsivity', most agree it is a trait with many facets, including the tendency to act without thinking, to display difficulties withholding a pre-potent response, and to prefer immediate pleasures over delayed outcomes (Odum, 2011). Unfortunately, the absence of a simple operational, and uniformly-agreed upon, definition for this complex construct has hampered the study of impulsivity and resulted in the development of several self-report and behavioural measures – most of which are only weakly correlated because they assess different aspects of the trait (Potenza & de Wit, 2010). Taking account of these issues, the current study employed three commonly used measures of impulsivity. Food addicts demonstrated greater impulsive responding on all of them compared to the control participants, underscoring the strength and consistency of our findings.

Our measure of addiction proneness was also significantly higher in the FA participants. Indeed, this variable accounted for the greatest variance in YFAS symptom scores, indicating its centrality as a risk factor for FA. While the Addiction Scale comprises items from all three factors of *Eysenck Personality Questionnaire*, the largest proportion comes from the Neuroticism scale. Therefore, high scores tend to reflect elevated levels of emotional reactivity, proneness to stress, and negative affect. Of relevance to our findings is the high co-morbidity between anxiety disorders and addiction disorders (Kessler et al., 1994; Maremmani et al., 2011). There is also evidence that individuals' mental state influences their response to addictive drugs. In particular, negative affect and anxiety have been shown to correlate positively with increases in brain dopamine levels and greater subjective feelings of happiness ('high') in response to a psychomotor stimulant drug (Volkow et al., 1994).

Eating behaviour evidence

The powerful urges and cravings that compel drug seeking behaviours – often against the individual's better judgement – are cardinal features of all addiction disorders (Garavan, 2010). As expected, the food addicts reported stronger food cravings than the non-FA group. Since differences in the hedonic value of drugs of abuse directly establish their level of compulsive use (Volkow & Wise, 2005), we also expected, and found, that food addicts were more sensitive and responsive to the pleasurable properties of palatable foods as indicated by higher scores on a measure of the hedonic impact of food, and by more frequent snacking on sweets. Similar to the preliminary validation research by Gearhardt et al. (2009), we found that food addicts reported more overeating in response to emotional triggers like depression and anxiety, and were more likely to self-soothe with food compared to control participants. The two groups did not differ, however, on the measure of overeating driven by external cues from the environment.

Limitations and future directions

In conclusion, it is important to acknowledge that the number of YFAS food addicts in this study was relatively small. It is therefore particularly impressive that we were able consistently to observe statistically significant differences where the power to detect even substantial differences is low. The size of our sample also precluded the opportunity to examine gender differences and

possible gender by FA interactions in this study. There are many compelling reasons why such investigations are an important next-step in this area of research. For example, while the prevalence of the highest BMI's (those >50) is increasing exponentially in all segments of society, the most pronounced increase has been in women and children (Popkin, 2010). Moreover, BED occurs more frequently in women (Javaras et al., 2008), and women are more likely than men to experience food cravings (Pelchat, 1997). In the field of addiction research, we have also learned that women tend to progress more rapidly from drug use to abuse (Giffin, Wise, Mirin, & Lange, 1989), are more sensitive to the subjective effects of cocaine, and report stronger drug cravings (Elman, Karlsgodt, & Gastfriend, 2001). These findings converge with recent evidence of gender and drug use interactions in neurodevelopmental brain processes (Medina et al., 2008).

A parallel body of pre-clinical research has shown greater reinforcement response to cocaine and amphetamine in female animals (Becker, Molenda, & Hummer, 2001), and found that ovariectomy attenuates their cocaine-stimulated locomotor behaviour (Johnson et al., 2010), suggesting that oestrogen mediates these behavioural sex differences. Female animals also demonstrate stronger reinstatement of cannabinoid-seeking after a drug or cue priming (Fattore, Melis, Fadda, Pistis, & Fratta, 2010). This sexual dimorphism in brain reward activation and response to conventional drugs of abuse, highlights the importance of examining gender effects in the context of FA and responsiveness to highly palatable diets. Such an approach is especially timely since many previous studies of gender differences in overeating behaviours have relied largely on sociocultural explanations (e.g. McCabe, Ricciardelli, & Holt, 2010; Neighbors, Sobal, Liff, & Amiraian, 2008).

To summarize, our findings have demonstrated strong parallels between food and substance abuse in a group of obese adults recruited from the community. This is also the first study to use a case–control design to investigate the clinical, psychological, and behavioural characteristics of YFAS food addicts in comparison to those of equivalent age and BMI. In addition, these findings advance the quest to identify clinically relevant subtypes of obesity that may possess different vulnerabilities to environmental risk factors. This information may also inform more personalized treatment approaches for those who struggle with overeating and weight gain.

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