Appetite 57 (2011) 711–717

Contents lists available at SciVerse ScienceDirect

Appetite

journal homepage: www.elsevier.com/locate/appet

Research report

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

Caroline Davis [a,b,*], Claire Curtis [a], Robert D. Levitan [b], Jacqueline C. Carter [c], Allan S. Kaplan [b], James L. Kennedy [b]

[a] Faculty of Health, York University, 343 Bethune College, 4700 Keele Street, Toronto, Ontario M3J 1P3, Canada
[b] Centre for Addiction and Mental Health, Toronto, Canada
[c] Department of Psychiatry, University Health Network, University of Toronto, Canada

ARTICLE INFO

Article history:
Received 2 June 2011
Received in revised form 7 August 2011
Accepted 25 August 2011
Available online 3 September 2011

Keywords:
Food addiction
Obesity
Yale Food Addiction Scale
Validation

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

* Corresponding author.
E-mail address: cdavis@yorku.ca (C. Davis).

[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.

0195-6663/$ – see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.appet.2011.08.017

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).