



Eating dependence and weight gain; no human evidence for a ‘sugar-addiction’ model of overweight

C. Rob Markus ^{a,*}, Peter J. Rogers ^b, Fred Brouns ^c, Robbie Schepers ^a

^a Faculty of Psychology and Neuroscience; Dept of Neuropsychology & Psychopharmacology; Maastricht University, Maastricht, The Netherlands

^b Nutrition and Behaviour Unit, School of Experimental Psychology, University of Bristol, Bristol, UK

^c Human Biology, Faculty of Health Medicine and Life Sciences, University Maastricht; Maastricht, The Netherlands

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ABSTRACT

Background and aims: There is an increasing societal concern that consumption of specific foods such as sugar might become ‘addictive’ and, hence, promote weight gain. Claims about the addictiveness of sugar however are based largely on findings from few animal studies, whereas there is a lack of direct human evidence for symptoms of sugar-related substance dependence. The current study examined in a large sample of human participants whether foods mainly containing sugar in particular might cause ‘addiction-like’ problems that meet clinical DSM criteria for substance dependence, and, also whether in turn this relates to body weight and negative affectivity (depressed mood).

Methods: In a cross-sectional study, $n = 1495$ university students from a variety of faculties were assessed for DSM-related signs of food addiction for particular food categories (YFAS), and, also BMI and negative affectivity.

Results: Results revealed that from the total sample, 95% experienced at least one symptom of food dependence and 12.6% met the YFAS classification for ‘food addiction’ as related to DSM-IV criteria. The majority of respondents experienced these problems for combined high-fat savoury (30%) and high-fat sweet (25%) foods, whereas only a minority experienced such problems for low-fat/savoury (2%) and mainly sugar-containing foods (5%). Overweight correlated only with addictive-like problems for high-fat savoury and high-fat sweet foods ($P < 0.0001$), while this was not found for foods mainly containing sugar.

Conclusion: The current findings indicate that sugary foods contribute minimally to ‘food dependence’ and increased risk of weight gain. Instead, they are consistent with the current scientific notion that food energy density, and the unique individual experience of eating, plays an important role in determining the reward value of food and promoting excessive energy intake.

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1. Introduction

The global epidemic of overweight is considered one of the greatest threats to human health. Since 1980, the prevalence of clinical overweight or obesity has nearly doubled; already affecting more than 30 percent of the population in the US (Flegal, Carroll, Ogden, & Curtin, 2010) and in Europe (Berghofer et al., 2008) and it is expected to further increase to more than 80% in the upcoming years. People with obesity are known to suffer from low grade

inflammation (Calder et al., 2011) resulting in a greater risk of developing type 2 diabetes (Mokdad et al., 2001), cardio vascular disease (Butchart et al., 2006) as well as mood disorders, including depression (Dong, Sanchez, & Price, 2004).

With the increase in worldwide prevalence of overweight and its relation to severe health consequences and psychopathologies, the concept of ‘food addiction’ has regained popularity; both among the lay public and professionals, including food researchers and nutrition practitioners (Brownell & Gold, 2013, pp. 439–46). This concept in particular has regained popularity since increasingly greater importance has been given to environmental obesogenic factors, such as the over-whelming availability of inexpensive palatable calorie-dense sweet and/or fatty foods (Mattes & Foster, 2014; Rogers & Brunstrom, 2016; Wing et al., 2001).

* Corresponding author. Faculty of Psychology and Neuroscience, Dept of Neuropsychology & Psychopharmacology, Universiteitssingel 40, 6229 ER, Maastricht, The Netherlands.

E-mail address: r.markus@maastrichtuniversity.nl (C.R. Markus).

In most current discussions of guilty snacks for weight gain and obesity, in particular the hedonic rewarding characteristic of sugar has been recently suggested by some to have abuse potential that is similar to classical drugs by stimulating shared brain reward pathways involved in drug addiction (Avena, 2007; Avena, Rada, & Hoebel, 2008; Hoebel, Avena, Bocarsly, & Rada, 2009). Consequently, in lay terms this ‘sugar-addiction’ hypothesis now seems to offer an attractive explanation, and possibly an excuse, for excessive consumption of sugary foods (foods mainly containing sugar), including binge eating and hence overweight (Rogers & Smit, 2000).

This sugar-addiction hypothesis is mainly based on findings from rodent experiments that are interpreted in favor of the addiction-like properties of sweetness from sucrose, glucose and/or saccharine liquid (Avena, 2007; Hone-Blanchet & Fecteau, 2014; Rada, Avena, & Hoebel, 2005). In most of these sugar-bingeing experiments, rats given intermittent access to sucrose –with a 12-h ad libitum access to sucrose solution followed by 12 h without sucrose–revealed signs of what the authors describe as sugar ‘bingeing’ behavior. Although in these studies sugar bingeing was not accompanied by animal weight gain, it often increased dopamine (DA) within the nucleus accumbens (NAcc) and caused ‘withdrawal’ signs after cessation of sucrose or glucose and administration of an opioid antagonist (Avena et al., 2008; Cottone et al., 2009; Hajnal, Smith, & Norgren, 2004; Rada et al., 2005). Since drugs of abuse (e.g. amphetamine, cocaine), among other ‘rewards’, act on the same mechanisms and are also found to increase extracellular concentrations of DA in the striatum and related mesolimbic regions (Di Chiara, 2013), these apparent similarities between sugar and drugs on DA have given rise to the sugar addiction hypothesis of binge eating. However, acting on the same mechanism does not necessarily say anything about (shared involvement in) addiction.

Although rodent research may generate meaningful hypotheses for understanding human behavior, their translational value for human eating behavior, food addiction and binge eating or obesity is highly controversial in the current scientific community (Benton, 2010; Benton & Young, 2016; Hebebrand et al., 2014; Ziauddeen & Murray, 2010; Ziauddeen et al., 2013). Ample evidence from systematic reviews and meta-analyses of controlled human dietary trials do not support the assumption that a particular macronutrient like sugar causes binge eating and weight-gain more so than other food sources (Benton, 2010; Benton & Young, 2016; Choo, Ha, & Sievenpiper, 2015). Yet, in contrast to the available human evidence regarding the association between food and weight gain, the validity of the sugar-specific addiction model of binge eating and/or weight gain has only rarely been explored in human studies.

A most frequently used method to explore ‘addiction-like’ properties of foods in human participants is to assess whether they may experience food harms that meet approximate DSM criteria for ‘substance use disorder’ (e.g. tolerance, withdrawal, considerable time spent on finding, using and recovering from the abuse, inability to cut down using despite the desire to do so, continued usage despite negative consequences). For this purpose, Gearhardt, Corbin, & Brownell (2009) developed the Yale Food Addiction Scale (YFAS) that uses some (not all) DSM criteria for clinical-related substance disorders to quantify symptoms of ‘addiction-like eating’ for highly palatable, energy-dense foods. Although the use of the self-report YFAS questionnaire has also been criticized for its little value concerning the clinical diagnose for addiction and/or substance-related disorders (Long, Blundell, & Finlayson, 2015), it is today’s most common and frequently used method to explore ‘food-related dependency like’ symptoms in human eating research. For this, it mainly relies on its validation in normal weight as well as obese and/or binge eating participants; revealing strong

correlations between YFAS symptoms scores to elevated craving (Gearhardt, Rizk, & Treat, 2014) as well as elevated body weight and binge eating (Davis et al., 2011; Gearhardt, Boswell, White, 2014). Furthermore, the prevalence of YFAS ‘food addiction’ diagnose (FA) is found to range between 0 and 11% in general population (Brunault, Ballon, Gaillard, Reveillere, & Courtois, 2014; Pedram et al., 2013) with a 4–5 times greater prevalence of 20–70% in binge-eating and/or overweight and obese participants (Long et al., 2015; Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014). Only few authors have yet attempted to assess YFAS ‘food-addiction’ signs for specific foods or food attributes/nutrients (Kromann & Nielsen, 2012; Pedram et al., 2013; Pepino, Stein, Eagon, & Klein, 2014; Schulte, Avena, & Gearhardt, 2015).

To our knowledge, the present study is the first to investigate the prevalence of YFAS ‘food addiction’ symptomatology –as well as its association with body weight–, while considering (controlling for) the possible mediating role of specific types of problem-macronutrients (including sugar). Moreover, since preferences for sugary foods are often found to rise under negative affective and/or depressive circumstances (Markus, 2008; Rogers, 1995), ‘addiction-like’ problems for sugar in particular might coexist with depression symptoms. Specifically, the study explored in a large population sample ($n = 1495$) of healthy young male and female participant whether YFAS ‘food-addiction’ symptoms more often occur for foods that largely/only contain sugar (as compared to combined foods) and, in turn, whether this relates to BMI and depression.

2. Methods

2.1. Participants

Fourteen hundred and ninety five university students, 1046 women and 449 men (age between 18 and 30; 21.6 ± 3 years), participated in this study by completing an on-line survey. Exclusively after full completion of the online survey (thus after receiving $n = 1495$ completed packages), more than half were randomly invitation to the laboratory for extra actual weight-related measurements. Students from different faculties of the Maastricht University were recruited via e-mail, advertisements, and fliers placed and posted in the local paper of the University. The study was approved by the Ethics committee of the Faculty of Psychology and Neurosciences (ECP, Maastricht, The Netherlands) and participants were paid for participation.

2.2. Procedure

Besides advertisements and fliers, University students across different faculties and study-years at the University Maastricht received a personal invitation mail to participate ($N = 6000$). Responding students ($N = 2500$) received a confirmation mail including further information about the study as well as a personal link to complete an on-line survey set on the digital research platform ‘EMIUM’. The on-line survey included standardized questionnaires assessing general characteristics, signs of food addiction (Yale Food Addiction Scale or YFAS; (Gearhardt et al., 2009a) and depressive symptomatology (Beck, Steer, & Garbin, 1988). From all responding students, finally 1495 students fully completed and returned the electronic questionnaire packages within 3–4 weeks. Half of these participant ($N = 786$) were also actually tested for (to check) weight-related variables in the laboratory. Invitations for these additional laboratory tests were exclusively and randomly done 1–3 weeks after receiving $n = 1495$ completed electronic packages and these tests were conducted within 1 month after completion of the electronic on-line survey).

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