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

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Running head: Sugar, 'addiction' and body weight

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.

Keywords: Food Addiction, Sugar, YFAS, BMI, Obesity, Eating behaviour

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 (WHO, 2015); already affecting more than 30 percent of the population in the US (1) and in Europe (2) 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 (3) resulting in a greater risk of developing type 2 diabetes (4), cardio vascular disease (5) as well as mood disorders, including depression (6).

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 (7). 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 (8-10).

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 (11-13). 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 (14).

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 (11, 15, 16). 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 hours 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 (12, 15, 17, 18). 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 (19), 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 (20-24). 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 (20, 24, 25). 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 and

colleagues (26) 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 (27), 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 (28) as well as elevated body weight and binge eating (29, 30). Furthermore, the prevalence of YFAS 'food addiction' diagnose (FA) is found to range between 0-11% in general population (31, 32) with a 4-5 times greater prevalence of 20-70% in binge-eating and/or overweight and obese participants (27, 33). Only few authors have yet attempted to assess YFAS 'food-addiction' signs for specific foods or food attributes/nutrients (31, 34-36).

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 (37, 38), '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.

METHODS

Participants

Fourteen hundred and ninety five university students, 1046 women and 449 men (age between 18-30; 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.

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; (26) and depressive symptomatology (39). 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).

Materials

Signs of food addiction.

To assess Food Addiction for food-specific categories; an elaborated version of the Yale Food Addiction Scale (YFAS) (26) was used. The YFAS is a 25-item scale that measures food addiction like symptomology based on the DSM-IV criteria for substance dependence. These items consists of both Likert scales and dichotomous scales that fall under criteria that resemble symptoms for substance-dependence as stated in the DSM and as operationalized in the Structured Clinical Interview for DSM disorders (American Psychiatric Association, 2000). These criteria scales include symptoms for tolerance, withdrawal, loss of control over consumption, continued use despite problems, repeated unsuccessful attempts to quit and/or clinically significant impairment/distress.

For eight YFAS clinical symptom scales, a total score of symptoms endorsed was calculated by first using an algorithm based on items under each symptom (score of ≥1 indicated that the substance dependent criteria were met, a score of "0" indicated that the criteria were not met) and then by summing up the total number of symptoms that met the criteria (ranging from 0-7). The classification (diagnose) of food addiction (FA) was made when respondents experienced three or more symptoms over the past year that met the substance dependent criteria with the presence of clinically significant impairment or distress from overeating. The YFAS is found to have good internal reliability (alpha = 0.86) along with good convergent and divergent reliability (26).

Because the current study aimed to explore the effects of sugar in particular as compared to other food categories; we included the following parts to the YFAS. First, to avoid priming, we modestly changed the starting instruction text to: "*when the following questions ask about "certain foods or snacks", please think of any food you had a problem with in the past year".* Then, three extra items were added (at the end of the original 25 items) for the purpose of

relating food addiction (FA) symptoms more directly to specific food categories. A first additional item (item 26) asked participants to assign the food problems they might have experienced in the past year (covered by the original first 25 items) to one of 4 explicitly given typical food categories: 1) Low-fat savoury foods (LFSA; light foods/snacks containing none/almost none fat or sugar; e.g. rice cake, crackers, vegetables, etc), 2) Sugar foods (foods mainly/totally containing sugar without fats or protein; e.g. sugar sweets, candy, wine gums, soda or lemonade, syrup, dried fruit), 3) High-fat sweet foods (HFSW; foods containing much fat either or not with sugar: e.g. cake, chocolate, patisserie, etc) or 4) High-fat savoury foods (HFSA; foods mainly containing much fat and protein like chips/fries, meat, cheese, etc). Two further items were added; asking whether there are any foods that have been experienced causing physical side effects (withdrawal-like effects: item 27) or whether there are foods that need to be consumed in greater amounts in order to experience the same pleasure/rewarding effect (tolerance: item 28).

Depression symptoms. Participants' ratings of depression symptoms were obtained using the Beck Depression Inventory (BDI), a validated self-report questionnaire containing 21 items measuring severity of depression symptoms. The BDI has been studied extensively and has been shown to be a reliable and valid measure of the severity of depression symptoms (39, 40).

Body weight. Height and weight values were obtained from each (N=1495) participant's general information questionnaire as part of the completed electronic questionnaire package. Body mass index (BMI) was calculated as weight (in kg) divided by the square of the height (in meters). To check for accuracy of self-reported height and weight values, half of the

population (N=786) was also actually assessed for these variable in the laboratory (1-3 weeks after completion of the electronic on-line survey).

Statistical analyses

All data were first examined for accuracy of data-entry and missing values. Testing for normal distribution was conducted using the Shapiro-Wilk test of normality. Cross-tab and/or chi-square tests were conducted to explore distribution and differences in the percentages of separate YFAS symptoms as well as for the prevalence of YFAS FA in the total sample of respondents; across the different problematic-food categories (Sugar, HFSW, HFSA or LFSA). Bivariate-comparisons were conducted to explore associations between total YFAS symptoms and BMI scores as well as between depression symptomatology (BDI scores) and YFAS symptoms. In addition, a hierarchical multiple regression analysis was conducted to examine whether 'problems for specific foods' may alter the association between YFAS and BMI (thus letting the model account for differences between problem-food categories with respect to YFAS effects on BMI). For this, *YFAS symptoms* (model 1), *Problem-food category* (Model 2) and *Problem-food category* by *YFAS symptoms* interaction terms (model 3) were sequentially added as a predictor of changes in BMI. *Problem-food category* (PFC) dummy variables were computed for PFC-HFSW, PFC-HFSA and PFC-Sugar by each time assigning value 1 for those belonging to the one category and 0 to the others. Interaction terms were computed by multiplying each of the dummy variable (PFC-HFSA, PFC-HFSW or PFC-Sugar) with the total YFAS scores (i.e.: YFAS*PFC-HFSA). During multiple regression analysis, PFC-HFSA and PFC-HFSW (as well as their interaction terms) were actually included in the model while keeping the PFC-Sugar constant (as reference).

To further interpret results from the hierarchical multiple regression analysis, additional analyses of variance (ANOVA's) were conducted including *Problem-food Category* (Sugar vs

HFSA vs HFSW) as between-subjects factor on YFAS symptoms and/or BMI as dependent variables. Only significant main or interaction effects revealed by these procedures were further examined by post-hoc tests. All statistics are evaluated at a two-tailed significance level of 5%. Data are reported as means ± SD.

RESULTS

YFAS addiction symptomatology

The total score of YFAS symptoms endorsed during the last year (given as a continuous measure) ranged from 0 to 8 (mean= 1.48, SD=0.32). Figure 1 shows the percentages of YFAS symptoms endorsed in the total sample of respondents that reported to have at least one symptom (n=1414; 95%). Results reveal that in particular the symptom '*persistent desire or repeated unsuccessful attempt to quit*" (YFAS symptom scale 2) was the most common and frequently experienced symptom (93,8%), whereas the percentages for remainder symptom scales all fell below or around the incidence of 10%. Additionally, the prevalence of the categorical diagnose FA as measured by the YFAS was 12.6%.

Further exploratory analysis in the group of respondents that reported having at least one symptom during the past year (95%, n=1414), revealed that the majority experienced such symptoms for the combined HFSA foods (29,5%) and HFSW foods (25%); whereas only a minority reported such symptoms for Sugar foods (5%) or for LFSA foods (1,8%). Further analysis revealed that the YFAS diagnose for FA was significantly more prevalent in respondents reporting to have problems for HFSW foods (4.7%) and HFSA foods (3.3%) as opposed to respondents reporting problems for sugar foods (0.8%) or LFSA foods (0.5%).

FIGURE 1 ABOUT HERE

In addition, analysis in the group of respondents that reported having at least one symptom during the past year (N=1414) also revealed that 9.5% of individuals experienced 'withdrawal-like' *physical* effects when stopping from eating the food (additional item nr 27), of which the majority experienced them for HFSA foods (3.8%) and HFSW foods (2.8%); whereas only a minority experienced this for sugar foods (1.6%) or LFSA foods (1.3%). Comparable results were found for 'adaptation-like' effects when stopping eating the food (additional item nr 28); of which the majority experienced this for HFSA foods (2.9%) and for HFSW foods (3.2%) as compared to sugar foods (1%) and LFSA foods (1.8%). ANOVA including *problem-food category* (Sugar rich vs HFSA vs HFSW) as between-subjects factor indeed revealed that the intensity of 'physical withdrawal' experiences were significantly higher for HFSW foods [6.5±1.5] than for sugar rich foods [6.0±2] (p=0.038) as well as for HFSA foods [6.6±1.2] compared to sugar rich foods (p=0.001). Also the intensity of 'adaptation problems' were higher for HFSA foods (6.6±1) than for sugar rich foods [6.0±2) (p=0.006) as well as for HFSW foods (6.6±1.3) compared to sugar rich foods (6.0±2) (p=0.04).

YFAS addiction symptomology & BMI

To check for accuracy of the self-reported height and weight values during the on-line survey (N=1495; via the digital research platform 'EMIUM'), half of the sample (N=786) was also actually measured at the University laboratory. This half of participants was only invited/informed for these extra measures after full completion of the on-line survey (only after receiving all n=1495 completed packages). Bivariate correlation analysis revealed a high association between BMI scores as calculated on basis of the objective laboratory measures and BMI scores based on self-report questionnaire data (r= 0.95: P<0.0001); indicating that

current self-reported questionnaire data are highly adequate predictive measure for the actual BMI of the participants.

Bivariate comparison between YFAS symptoms and BMI scores (n=1495) revealed a modest but significant positive correlation [r=0.18; P<0.0001]. Hierarchical regression analyses in respondents reporting at least one symptom during the past year (n=1414) revealed that when including YFAS symptoms as the only predictor (Model 1) it explained a small but significant proportion (3%) of variance in BMI scores (R^2 =0.032, F(1,901)=30.523, p<0.001). When adding *Problem-Food-Category* (PFC) dummy variables (PFC-Sugar = constant) as an extra predictor (Model 2) the model explained 5% of variance in BMI scores (R^2 =0.046, F(1,901)=14.358, p<0.001). After finally adding *YFAS*PFC* interaction terms into the model (Model 3, YFAS*PFC-Sugar interaction term = constant), the explanatory capacity significantly increased (R^2_{change} = 0.012, p=0.001) and produced a regression model that explained 6% of variation in BMI (R^2= 0.057, F(5,901)=10,907; p<0.0001) (see Table 1). As the Model-3 part of Table 1 shows, the PFC-HFSA association between YFAS and BMI (expressed by: the YFAS*PFC-HFSA interaction term) is significantly different from the PFC-Sugar one at the P<0.001 level; whereas the PFC-HFSW association between YFAS and BMI (expressed by the YFAS*PFC-HFSW interaction term) only moderately differs from the PFC-Sugar one (P=0.06). Further post-hoc regression analysis for all *problem-food categories* separately indeed revealed that YFAS symptoms predicted greater BMI in participants reporting to experience symptoms for HFSA (R^2 = 0.070, F(1,302) = 33.850; p<0.0001) and, to a lesser degree, for HFSW (R^2 = 0.030, F(1,359) = 12.141; p = 0.001); but not in participants reporting YFAS symptoms for sugar (R^2 = 0.007, F(1,72) = 1.489; p = 0.23).

TABLE 1 ABOUT HERE

In addition, univariate analysis of variance including *problem-food category* (Sugar rich vs HFSA vs HFSW vs LFSA) as between-subjects factor on BMI scores revealed a significant effect of *problem-food category* [F(3,898)=3.189; P=0.023], indicating significant differences in BMI across type of problem foods. As shown in Figure 2, BMI was highest in subjects reporting problems with HFSA foods (BMI= 23±3) and gradually declined across problems with HFSW foods (BMI= 22.4±3), sugar foods (BMI= 22.2±3) and LFSA foods (BMI= 21.3±4). Table 2 represents percentage of YFAS FA diagnose across weight and problem-food categories.

FIG 2 & Table 2 ABOUT HERE

Food addiction symptomology & depression

To test whether YFAS symptomatology might change as a function of depression, a first regression analysis was conducted with BDI scores as predictor in the group of respondents having at least one YFAS symptom during the past year (n=1414). Analysis revealed a strong positive correlation [r=0.41; P<0.0001], indicating that an increase in BDI scores explained a significant proportion (17%) of variance in YFAS symptomatology (Stage 1: R^2 = 0.167, F(1,867)=174,146; P<0.0001). When letting the regression model account for possible differences between the main problem-food categories Sugar, HFSA and HFSW (Stage 2: adding dummy interaction terms 'HFSA x BDI' and 'HFSW x BDI'), BDI capacity to predict YFAS symptoms only increased marginally (R^2_{change} = 0.009, p=0.013), now predicting about 18% of the variation in YFAS (F(3,865)= 61,393; p<0.0001, R^2 = 0.176). Further regression analysis for the *problem-food categories* separately revealed that BDI scores only significantly predict increases in YFAS in participants reporting problems for HFSA (R^2 =

0.155, $F(1,436) = 81.354$; $p<0.0001$) and HFSW ($R^2 = 0.196$, $F(1,359) = 88.895$; $p<0.0001$) but not in participants reporting problems for sugar rich foods ($R^2 = 0.004$, $F(1,72) = 1.306$; $p = 0.26$).

DISCUSSION

The current study explored in a large (n=1495) sample of healthy participants the incidence of 'food addiction', defined via the YFAS, for mainly sugar containing foods compared with other food sources, and also whether this revealed relationships with BMI and depression. While a majority (95%) of participants experienced at least one YFAS symptom, of which 12% actually met the YFAS diagnosis for food addiction, these problems were mostly reported for the combined high-fat savoury and/or high-fat sweet foods (30%) as opposed to sugary specifically (5%). Moreover, there was a positive relationship between YFAS symptoms and increases in body weight that was only significant in participants reporting to have YFAS problems for HFSA and/or HFSW.

A 12% prevalence for YFAS food addiction diagnose (FA) in the current large sample of n=1495 healthy college student participants is broadly consistent with previous reports (26, 27, 31, 33, 41). On average, the prevalence of YFAS food addiction ranges between only 0-8% in general population (31, 32) and between 20-70% in binge-eating and/or obese participants [see: (27, 33) for systematic reviews]. Furthermore, also consistent with previous studies, the most common 'food addiction' symptom in the current study was '*persistent desire or repeated unsuccessful attempt to quit*" (YFAS scale 2: >93%) whereas all the other symptoms were reported relatively rarely.

The major aim of the current study was to explore whether YFAS 'food addiction' symptoms are more often expressed for sugary foods and whether this relates to a greater risk for weight

gain. Analyses however revealed that just a minority reported food-addiction symptoms for sugar-sweetened specific foods (5%) whereas most participants reported to have experienced YFAS symptoms more frequently in relation to combined high-fat sweet and/or high-fat savoury foods (25-30%). In addition, FA as diagnosed by the YFAS was most prevalent in participants having YFAS problems related to HFSW foods and HFSA foods as compared with participants experiencing problems for sugary foods. Furthermore, 'withdrawal' and/or 'adaptation' signs (included as extra items in YFAS) were more frequently reported for HFSA and HFSW foods than for sugar-specific foods. These results all are in line with past studies showing that YFAS FA is most prevalent in participants reporting high proportion of energy intake from fat and/or protein, or from 'fast' processed foods (34, 36).

The next important question was the extent to which YFAS symptoms for sugar rich foods may be related to weight gain compared to YFAS symptoms expressed for other foods. Results first revealed that BMI significantly varied with the number of YFAS symptoms with BMI increasing directly as the number of 'food-addiction' experiences. Ample previous studies have already revealed that BMI scores are positively related to rates of YFAS FA (29, 42) and/or the total number of YFAS symptoms (43); although contradictory findings have also been reported (44). Consequently, consistently greater prevalence of YFAS FA has been reported in overweight and obese samples relative to normal-weight samples [see for a review: (27, 33). Hence, whereas the prevalence of YFAS FA is found to range between 0-8% in the general population (31, 32), it has a four or more times greater prevalence of between 20-70% (with a mean symptom count score of 3.5) in binge-eating and/or overweight and obese participants (27, 33). The current study extends these findings by indicating that an increase in BMI as a function of YFAS symptoms is most profound in individuals that attribute such YFAS problems to combined high-fat savoury and/or high-fat sweet foods rather than sugary and LF foods. This also fits the average findings from prospective cohort

studies showing that HFSA foods play an equal or even greater role in weight gain and/or risk for obesity than sugar itself or HFSW food sources enriched with sugars (25). In addition, there nowadays exists consensus that palatability and/or energy density of food is a more determining factor for food intake and/or the risk to binge rather than that a particular type of macro-nutrient directly causes obesity.

Interrelationships between YFAS-FA, depression symptomatology and overweight

In support of previous findings (33), the current results revealed a high positive correlation between depression symptomatology (BDI scores) and YFAS symptom scores. Moreover, depression symptoms predicted increases in YFAS scores markedly in participants who reported addiction symptoms for the HFSA and/or HFSW foods. This might even suggest that the often reported relationship between food addiction and weight gain is mediated by negative affectivity and/or depression symptomatology. In support of this, studies have shown that around 40% of individuals increase energy intake for energy-dense high-fat sweet and/or savoury foods under negative mood (45-50); most likely to fulfill emotional needs (51-54), indicating that this 'emotional-eating' behavior might be major risk factor for overweight (50).

Evidence for a sugar addiction model of binge eating and/or obesity?

The current societal focus on foods responsible for weight gain has nowadays been shifted to sugar, including its suggested addictive properties. This assumption relies to a great extent on findings from rodent studies in which sucrose was found to cause brief moments of bingeing behavior (yet without affecting body weight) and concomitant stimulation of activity in brain dopaminergic pathways also involved in drug addiction (12, 16, 55, 56). This then has boosted the proposition that the animal model of sugar-bingeing provides valuable insight into

the mechanisms involved in the onset and course of human obesity. Yet, ample evidence from human (and animal) studies fails to support the assumption that sugar differs from fat or other carbohydrates in equi-caloric amounts in increasing risk of overweight and obesity (25, 57-59). Furthermore, trends for dietary sugar intake are in decline (60) whereas the prevalence of overweight and obesity is clearly rapidly increasing. The present results are consistent with the notion that food palatability and/or density, more often represented by combined HFSW and HFSA foods, play a more critical role in determining the reward value of food and promoting excessive energy intake than a particular macronutrient (10). Moreover, food selection and intake in human is also strongly mediated by cognitive, emotional and behavioural intervening factors (but this is beyond the aim of the current study).

Regarding addiction, parallel the effects of drugs on dopaminergic changes by a food does not say anything necessarily about addiction and/or does not mean that the food is also addictive. First, alterations in the dopaminergic and/or opioid reward system more generally indicate a change in the neural circuitry involved in positive experiences and pleasure; which in human is also found after watching nice events (61), winning a prize (62), listening to music (63) or feeling love (64) and humor (65). In line, rodent dopaminergic and/or opioid changes to food appear to be related to palatability (reward value) rather than sweetness or nutrient per se and, hence, there are significant differences in neurochemical and/or electrophysiological reward changes caused by drugs than caused by foods or other natural rewards (20, 24). For instance, animal studies clearly show that dopaminergic changes caused by foods, unlike drug of abuse, undergo rapid adaptation and, hence, that compulsive seeking and/or continuation of substance (ab)use, -in spite of negative consequences,- is only found for drugs and not for foods like sugar (66-69). Consequently, the suggestion that sugar is addictive like a drug of abuse receives little support from the human literature (20, 21, 70). A more suitable

conclusion is that sugar has much more in common with normal experiences of pleasure that cause the release of DA rather than with the effects of drugs of addiction.

Critical remarks

A first remark is that a real 'food addiction' is hardly to be assessed (if it exists at all) by only using the YFAS questionnaire. Since YFAS scores are totally based on self-report responses, it has also been fairly criticized for its inherent limitations concerning the diagnose for addiction and/or substance-related disorders (27). Like most other diagnoses of scientifically validated addictive disorders, 'food addiction' should also be based on evaluations from trained/experienced clinicians (and even supported by pharmacological assessment methods). Yet, this limitation is not restricted to the present study; in the field of human research for food-dependence the YFAS is currently the most frequently used method available.

Second, since the current study focused on macro-nutrient related categorical differences (sugary foods vs HFS, HFSA and LFSA) the present findings cannot tell us anything about how palatability and/or energy-density of the different foods may relate to YFAS symptoms. Third, the current data does also not allow drawing conclusion about the possible rewarding influences of even smaller portions of (added) sugar to combined HFSW and/or HFSA foods. However, it seems justified to assume that if it's the sugar it than should become most visible for foods mainly containing sugar. In this respect, the 'synergy' among different food ingredients rather than one typical macronutrient seems to be most important. Finally, by using self-report strategies for food preferences and or dependencies; it is still possible that the foods to which participants reported to develop a food addiction are incorrectly classified into the pre-defined food categories and/or product descriptions. Firm conclusions from these databases should therefore be (ideally) further supported xx

General conclusions

Consuming palatable energy-dense foods and drinks, including those that mainly/only contain sugar, can become an emotionally rewarding experience that promotes energy intake beyond immediate nutritional need. In particular, under adverse circumstances, this rewarding value of sugar food can become increasingly emotionally comforting and/or stress-releasing (37, 38). This emotional rewarding value of sugary food will naturally be reflected by altered brain reward mechanisms including the endogenous opioid and/or DA systems, and will certainly have the potential to cause overeating. But even though this may involve comparable neuronal pathways that are also involved in drug use disorders; this overlap itself does not validate the model of 'binge-eating and/or weight-gain by sugar-addiction'. The brain does not appear to respond to food and/or sugar in the same way as to drugs; and 'addictive-like' overeating seems to be distinct from drug-addiction disorders. Sugar does not seem to contribute to weight-gain more so than other sources of energy in the diet. Instead, the current scientific community nowadays seems to reach consensus that 'food addiction' (and its role in weight gain) might be better explained by 'eating dependence' as a result of the unique individual experience with food and eating (instead of being caused by a specific food).

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Table 1: Hierarchical regression analysis testing differences between problem-food category (PFC) for predicting the YFAS effects on BMI

Model	b	SE	β	*t*	*p*	Δ R^2	R^2
1] Main effect							
YFAS	0.435	0.079	0.181	5.525	*<0.001*		
						0.033	**0.032***
2] Adding PFC							
YFAS	0.461	0.079	0.192	5.858	*<0.001*		
PFC-HFSA	0.473	0.183	0.169	2.588	*0.010*		
PFC-HFSW	0.122	0.180	0.049	0.677	*0.499*		
[PFC-Sugar = Constant]							
						0.013	***0.046****
3] Adding Interaction terms							
YFAS	-0.049	0.203	-0.021	-0.244	*0.808*		
YFAS **x** PFC-HFSA	0.771	0.238	0.270	3.239	*<0.001*		
YFAS **x** PFC-HFSW	0.447	0.234	0.175	1.909	*>0.06*		
[YFAS x PFC-Sugar= Constant]							
						0.012	***0.057****
						ΔF: 5.513	**F: 10.907**

b, unstandardized regression coefficience; **SE**, standard error; **β**, standardized regression coefficient; ***t***, obtained t-value; ***p***, probability; R^2, proportion variance explained; ΔR^2, change in proportion variance. *, $p \leq 0.001$

Model 3

Testing whether PFC-HFSA and PFC-HFSW associations between YFAS and BMI (*YFAS * PFC-HFSA* and *YFAS * PFC-HFSW*) are different from the PFC-Sugar one (*YFAS * PFC-Sugar* = constant). The interaction term's coefficient for PFC-HFSA is greater than for YFAS alone (-0.049 + 0.771= 0.722 as against -0.049: p<0.001). The interaction coefficient for PFC-HFSW is also greater (-0.049 + 0.447=0.398) but not significant (P=0.06). Accounting for these interactions increased predictability of the model (from R^2 0.032 to R^2 0.057; P<0.001); that YFAS predicts greater BMI when having problems with HFSA (and to a less degree with HFSW) than with Sugar.

Table 2: Percentages of YFAS FA diagnoses specified for weight- and problem-food categories

Weight category	BMI [kg/m2]	Sample size	Diagnoses (%)	Problem food
Normal weight	20-24	*n*=1125	11	☐ 5% ▨ 25% ■ 29%
Overweight	25-29	*n*=175	18	☐ 4,6% ▨ 26% ■ 39%
Obese	≥ 30	*n*=33	52	☐ 3% ▨ 30% ■ 33%

☐ Problems with sugar; ▨ Problems with HFSW; ■ Problems with HFSA

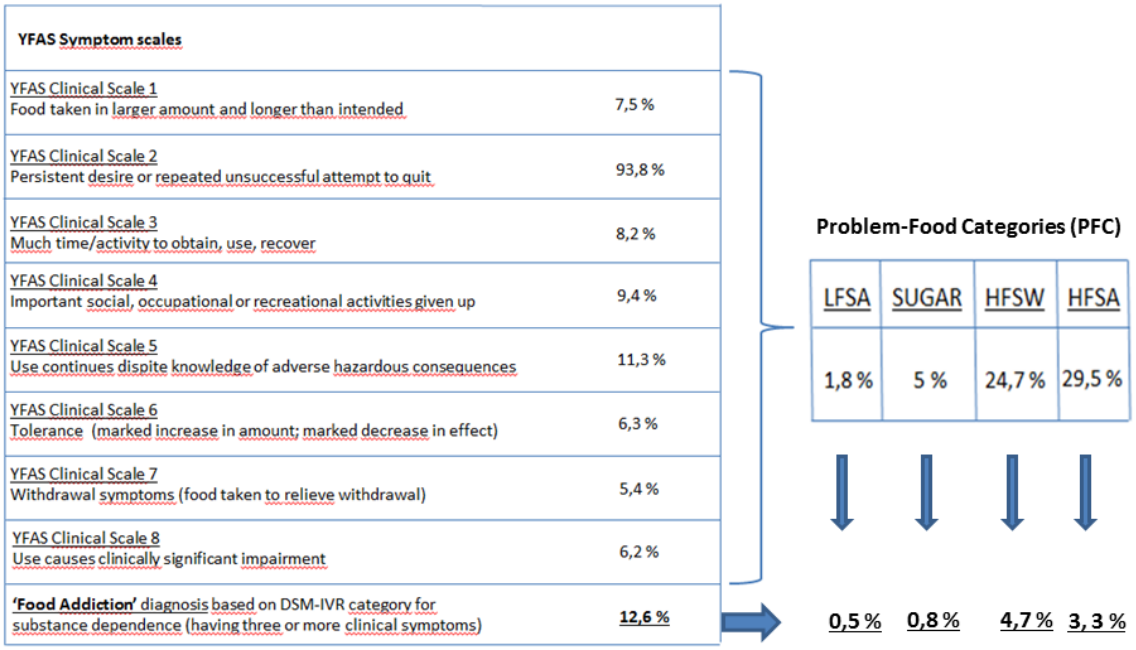

Figure 1: Endorsement of YFAS symptoms in subjects having experienced at least one symptom in the last year (N=1414). Symptoms were attributed to one of the 'guilty' problem-food categories (**PFC**) : Low-Fat Savory (**LFSA**), **Sugar**, High-Fat Sweet (**HFSW**) and High-Fat Savory (**HFSA**) foods.
[i.e. 1.8% of subjects attributed their experienced YFAS symptoms to LFSA foods; of which 0.5% fulfilled YFAS criteria for **Food Addiction**].

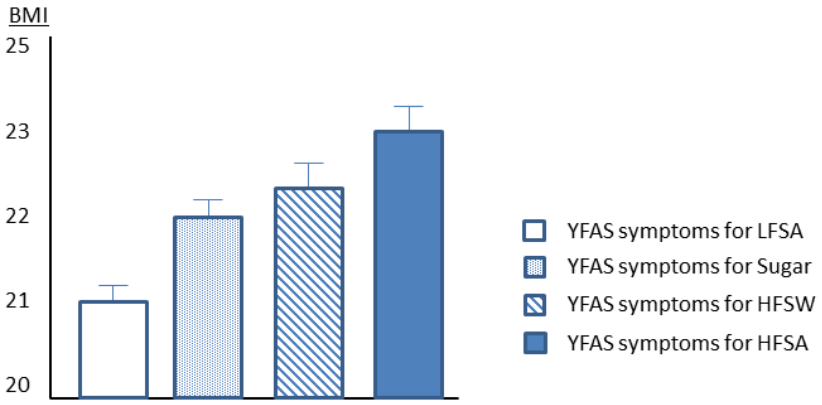

Fig 2. BMI scores differ as a function of YFAS food-problems. The highest BMI scores were reported in participants having problems with high-fat savory foods (HFSA: 23±3) and decline respectively when having problems with high-fat sweet (HFSW: 22.4±3), Sugar (22.2±3) and low-fat Savory (LFSA: 21.3±4) foods (P=0.023).