Food and Addiction References

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Text blocks are quotes from abstracts or titles – read the full paper for complete information

Contents

Textbook

Food and Addiction by Brownell and Gold, 2012[1]

Intro / Definitions

Addiction potential of hyperpalatable foods[2]

Animal and human theoretical models of food reward and addiction have emerged, raising further interrogations on the validity of a bond between Substance Use Disorders, as clinically categorized in the DSM 5, and food reward. These models propose that highly palatable food items, rich in sugar and/or fat, are overly stimulating to the brain's reward pathways.[3]

Is Food Addiction a valid concept?[4,5]

The feeding process is required for basic life, influenced by environment cues and tightly regulated according to demands of the internal milieu by regulatory brain circuits. Although eating behaviour cannot be considered "addictive" under normal circumstances, people can become "addicted" to this behaviour, similarly to how some people are addicted to drugs. The symptoms, cravings and causes of "eating addiction" are remarkably similar to those experienced by drug addicts, and both drug-seeking behaviour as eating addiction share the same neural pathways. However, while the drug addiction process has been highly characterised, eating addiction is a nascent field. In fact, there is still a great controversy over the concept of "food addiction".[6]

Prospective evidence of both reward surfeit and reward deficit pathways to increased body weight are evaluated, and we argue that it is more complex than an either/or scenario when examining DA's role in reward sensitivity, eating, and obesity. The Taq1A genotype is a common thread that ties the contrasting models of DA reward and obesity; this genotype related to striatal DA is not associated with obesity class per se but may nevertheless confer an increased risk of weight gain. We also critically examine the concept of so-called food addiction, and despite growing evidence, we argue that there is currently insufficient human data to warrant this diagnostic label.[7]

Here, we review the neuroimaging literature to consider the validity of food addiction and the common neurobiological mechanisms that overlap in food and drug addiction. This review paper focuses on findings from Positron Emission Tomography (PET), functional Magnetic Resonance Imaging (fMRI) and structural imaging studies, as well as evidence from neuroimaging studies of bariatric surgery and pharmacological interventions on obese individuals. We examine not only functional and structural changes in the mesolimbic pathways, but also in other frontal areas shown to be involved in drug addiction, including the prefrontal cortex, orbitofrontal cortex and anterior cingulate cortex, as well as changes in neurotransmitter systems beyond dopaminergic systems.[8]

Introducing food addiction as a new disorder in diagnostic classification system seems redundant as most individuals with an addiction-like eating behavior are already covered by established eating disorder diagnoses. Food addiction may be a useful metaphor in the treatment of binge eating, but would be inappropriate for the majority of obese individuals. Implying an addiction to certain foods is not necessary when applying certain approaches inspired by the addiction field for prevention and treatment of obesity. The usefulness of abstinence models in the treatment of eating disorders and obesity needs to be rigorously tested in future studies. Some practical implications derived from the food addiction concept provide promising avenues for future research (e.g., using an addiction framework in the treatment of binge eating or applying abstinence models). For others, however, the necessity of implying an addiction to some foods needs to be scrutinized.[9]

Investigators at all levels have been looking for factors that have contributed to the development of this epidemic. Two major theories have been proposed: (1) sedentary lifestyle and (2) variety and ease of inexpensive palatable foods. In the present review, we analyze how nutrients like sugar that are often used to make foods more appealing could also lead to habituation and even in some cases addiction thereby uniquely contributing to the obesity epidemic. We review the evolutionary aspects of feeding and how they have shaped the human brain to function in "survival mode" signaling to "eat as much as you can while you can." This leads to our present understanding of how the dopaminergic system is involved in reward and its functions in hedonistic rewards, like eating of highly palatable foods, and drug addiction. We also review how other neurotransmitters, like acetylcholine, interact in the satiation

processes to counteract the dopamine system. Lastly, we analyze the important question of whether there is sufficient empirical evidence of sugar addiction, discussed within the broader context of food addiction.[10]

Dietary Intakes of Processed Foods

According to 2009-2010 data from NHANES, Americans currently consume 60% of their calories from ultra-processed foods such as breads, cakes, cookies, pizza, French fries, salty and sweet snacks and desserts, with only 5% and <1% of calories attained from fruits and vegetables respectively.[11]The diet quality for most US adults is poor. The most recently scored national data for the HEI-2010 places the mean adult score at 58[12], in comparison to the MyPlate target of 100. Currently, approximately 14% of total energy comes from added sugars[13] while only 30% of calories comes from unprocessed or minimally processed foods such as meat or dairy, grains, legumes, and fruits and vegetables, while according to NHANES 2009-2010 data.[11] Only 42% of Americans meet the Dietary Guidelines recommendation to limit added sugars to <10% of calories.[14] With respect to sodium intake, the average intake among US adults is greater than the UL, at 3412 mg/day[14]), while average fiber intake is only 17 g/day (37), substantially lower than current recommendations (14 g/day per 1,000 kcal).[14]

Setting: All provinces and territories of Canada, 2001. Subjects: Households (n 5643). Results: Food purchases provided a mean per capita energy availability of 8908 (SE 81) kJ/d (2129 (SE 19) kcal/d). Over 61.7% of dietary energy came from ultra-processed products (Group 3), 25.6% from Group 1 and 12.7% from Group 2. The overall diet exceeded WHO upper limits for fat, saturated fat, free sugars and Na density, with less fibre than recommended. It also exceeded the average energy density target of the World Cancer Research Fund/American Institute for Cancer Research. Group 3 products taken together are more fatty, sugary, salty and energy-dense than a combination of Group 1 and Group 2 items. Only the 20% lowest consumers of ultra-processed products (who consumed 33.2% of energy from these products) were anywhere near reaching all nutrient goals for the prevention of obesity and chronic non-communicable diseases. Conclusions: The 2001 Canadian diet was dominated by ultra-processed products.[15]

Mechanisms – Drugs / Food

Neuroimaging studies in obese individuals have revealed alterations in reward/motivation, executive control/self-regulation, and limbic/affective circuits that are implicated in food and drug addiction. Psychophysical studies show that sensory properties of food ingredients may be associated with anthropometric and neurocognitive outcomes in obesity. However, few studies have examined the neural correlates of taste and processing of calories and nutrient content in obesity. The literature of neural correlated of bitter, sour, and salty tastes remains sparse in obesity. Most published studies have focused on sweet, followed by fat and umami taste. Studies on calorie processing and its conditioning by preceding taste sensations have started to delineate a dynamic pattern of brain activation associated with appetition. Our expanded understanding of taste processing in the brain from neuroimaging studies is poised to reveal novel prevention and treatment targets to help address overeating and obesity.[16]

Appetite for sugar is propelled by changes in the morphology and activity of the reward system reminiscent of addiction. Sugar intake also shifts the hunger-satiety continuum, facilitating initiation of consumption in the absence of energy needs and maintenance of feeding despite ingestion of large food loads that endanger homeostasis. Ingestion of excessive amounts of sugar relies on triggering mechanisms that promote addictive-like behaviors, and on overriding neuroendocrine signals that protect internal milieu.[17]

Three lines of evidence support the concept of food addiction: (*a*) behavioral responses to certain foods are similar to substances of abuse; (*b*) food intake regulation and addiction rely on similar neurobiological circuits; (*c*) individuals suffering from obesity or addiction show similar neurochemical- and brain activation patterns.High-glycemic-index carbohydrates elicit a rapid shift in blood glucose and insulin levels, akin to the pharmacokinetics of addictive substances. Similar to drugs of abuse, glucose and insulin signal to the mesolimbic system to modify dopamine concentration. Sugar elicits addiction-like craving, and self-reported problem foods are rich in high-glycemic-index carbohydrates. These properties make high-glycemic-index carbohydrates plausible triggers for food addiction.[18]

Opioid mediation of food intake was controlled by a distributed brain network intimately related to both the appetitive-consummatory sites implicated in food intake as well as sites intimately involved in reward and reinforcement. This emergent system appears to sustain the "positive addictive" properties providing motivational drives to maintain opioid-seeking behavior.

Using supermarket foods high in fat and sugar, we showed that such a diet leads to changes in neurotransmitters involved in the hedonic appraisal of foods, indicative of an addiction-like capacity of foods high in fat and/or sugar. Importantly, withdrawal of the palatable diet led to a stress-like response. Furthermore, access to this palatable diet attenuated the physiological effects of acute stress (restraint), indicating that it could act as a comfort food. In more chronic studies, the diet also attenuated anxiety-like behavior in rats exposed to stress (maternal separation) early in life, but these rats may suffer greater metabolic harm than rats exposed to the early life stressor but not provided with the palatable diet. Impairments in cognitive function have been associated with obesity in both people and rodents. However, as little as 1 week of exposure to a high fat, high sugar diet selectively impaired place but not object recognition memory in the rat. Excess sugar alone had similar effects, and both diets were linked to increased inflammatory markers in the hippocampus, a critical region involved in memory. Obesity-related inflammatory changes have been found in the human brain.[3]

In this study, we examined three hypotheses to disentangle the potential overlap between addiction and overweight/obesity processing by examining (1) brain response to high vs. low calorie beverages, (2) the strength of correspondence between biometrics, including body mass index (BMI) and insulin resistance, and brain response and (3) the relationship between a measure of food addiction and brain response using an established fMRI gustatory cue exposure task with a sample of overweight/obese youth (M age = 16.46; M BMI = 33.1). Greater BOLD response was observed across the OFC, inferior frontal gyrus (IFG), nucleus accumbens, right amygdala, and additional frontoparietal and temporal regions in neural processing of high vs. low calorie beverages. Further, BMI scores positively correlated with BOLD activation in the high calorie > low calorie contrast in the right postcentral gyrus and central operculum. Insulin resistance positively correlated with BOLD activation across the bilateral middle/superior temporal gyrus, left OFC, and superior parietal lobe. No relationships were observed between measures of food addiction and brain response.[19]

Disordered eating was higher in MDD than controls, in females than males, and in depressed individuals with increased, compared to decreased, appetite/weight. Leptin levels were higher in females only. Leptin levels correlated positively, and ghrelin negatively, with disordered eating. The results provide further evidence for high levels of disordered eating in MDD, particularly in females. The correlations suggest that excessive eating in MDD is significantly linked to appetite hormones, indicating that it involves physiological, rather than purely psychological, factors.[20]

The hedonic pathway interacts with the obesogenic environment to override homeostatic mechanisms to cause increase in body weight. Weight gain sustained over time leads to "upward setting" of defended level of body-fat mass. There are neurobiological and phenotypic similarities and differences between hedonic pathways triggered by food compared with other addictive substances, and the entity of food addiction remains controversial.[21]

Repeated administration of the reinforcer (drugs, energy-rich foods) generates conditioned associations between the reinforcer and the predicting cues, which is accompanied by downregulated dopaminergic response to other incentives and downregulated capacity for top-down self-regulation, facilitating the emergence of impulsive and compulsive responses to food or drug cues. Thus, dopamine contributes to addiction and obesity through its differentiated roles in reinforcement, motivation and self-regulation, referred to here as the 'dopamine motive system', which, if compromised, can result in increased, habitual and inflexible responding. Thus, interventions to rebalance the dopamine motive system might have therapeutic potential for obesity and addiction.[22]

Ghrelin, which is mainly released from the stomach, is the most important orexigenic regulator of food intake, inducing appetite, enhancing adiposity and releasing growth hormone. Besides the hypothalamus, ghrelin receptors (GHS-R1A) are also expressed in the mesolimbic dopaminergic system, which increases the possibility that ghrelin plays an important role in reward regulation for substance use disorders such as alcohol addiction, especially through activating the cholinergic-dopaminergic reward link.[23]

Results indicated the presence of a BED-specific neuropsychological profile in the obesity spectrum. The additional trait FA was not related to altered executive functioning compared to the OB or BED groups.[24]

Together, our data demonstrate that DNA methylation regulating factors are differentially altered by cocaine and food. At the molecular level, they support the idea that neural circuits activated by both reinforcers do not completely overlap.[25]

Here, we describe robust associations between uncontrolled eating, body mass index (BMI), food intake, personality traits and brain systems. Reviewing cross-sectional and longitudinal data, we show that uncontrolled eating is phenotypically and genetically intertwined with BMI and food intake. We also review evidence on how three psychological constructs are linked with uncontrolled eating: lower cognitive control, higher negative affect and a curvilinear association with reward sensitivity. Uncontrolled eating mediates all three constructs' associations with BMI and food intake. Finally, we review and meta-analyse brain systems possibly subserving uncontrolled eating: namely, (i) the dopamine mesolimbic circuit associated with reward sensitivity, (ii) frontal cognitive networks sustaining dietary self-control and (iii) the hypothalamus-pituitary-adrenal axis, amygdala and hippocampus supporting stress reactivity. While there are limits to the explanatory and predictive power of the uncontrolled eating phenotype, we conclude that treating different eating-related constructs as a single

concept, uncontrolled eating, enables drawing robust conclusions on the relationship between food intake and BMI, psychological variables and brain structure and function.[26]

Cognition / Subjective Experiences and Compulsive Eating

The current mini-review synthesizes the available evidence for performance on compulsivity-related cognitive tasks for each cognitive domain among populations with excessive eating behavior. In three of the four cognitive domains, i.e., set-shifting, attentional bias and habit learning, findings were mixed. Evidence more strongly pointed towards impaired contingency-related cognitive flexibility only in obesity and attentional bias/disengagement deficits only in obesity and BED. Overall, the findings of the reviewed studies support the idea that compulsivity-related cognitive deficits are common across a spectrum of eating-related conditions, although evidence was inconsistent or lacking for some disorders.[27]

Highly processed, relative to minimally processed, foods were more associated with indicators of abuse liability, although individuals with food addiction reported decreased enjoyment for and intentions to consume highly processed foods. Subjective experiences were associated with greater consumption of highly processed foods for participants with food addiction. The present work provides further support for the rewarding nature of highly processed foods, evidenced by closer associations with subjective experiences reported for drugs of abuse compared to minimally processed foods. In addition, highly processed food intake was related to elevated subjective experience reports for these foods for those with food addiction, paralleling findings in individuals with a substance-use disorder for the relevant drug.[28]

Obesity is recognized as an important risk factor for many chronic diseases and is a major health issue. The current study examined attentional bias to food and the "cool" (inhibitory control and mental flexibility) and "hot" (affective decision making) executive functions (EFs) in obese patients preparing for bariatric surgery. In addition to body mass index (BMI), this study examined the impact of the binge-eating tendency and eating styles. METHODS: The study population comprised 21 morbidly obese patients preparing to undergo bariatric surgery (BMI ≥30 kg/m) and 21 normal-weight controls (24 kg/m > BMI ≥ 18.5 kg/m). The Visual Probe Task was adopted to examine attentional bias toward food-related cues. The Stop-Signal Task and the Color Trails Test were used to assess inhibitory control and mental flexibility, respectively. The Iowa Gambling Task was administered to assess the affective decision making. RESULTS: (1) The obese patients showed poorer performances on cool EFs (for Color Trails Test, $P = 0.016$, $\eta p = 0.136$; for Stop-Signal Task, $P = 0.049$, $\eta p = 0.093$) and hot EF (for Iowa Gambling Task, normal controls showed progressed performance, $P = 0.012$, $\eta p = 0.077$, but obese patients did not show this progress, $P = 0.111$, $\eta p = 0.089$) compared with the normal controls; (2) participants with low binge-eating tendency had larger attentional biases at 2000 milliseconds than at 200 milliseconds on food-related cues ($P = 0.003$, $\eta p = 0.363$); and (3) low-restrained participants exhibited attentional bias toward the low-calorie food cues, compared with the high-restrained group ($P = 0.009$, $\eta p = 0.158$). CONCLUSIONS: The current study contributes to the development of a different therapeutic focus on obese patients and binge eaters.[29]

The expected three-way interaction between TV content, eating restraint, and PSRS on hedonic eating goal accessibility was not found in Experiments 1 and 2. In Experiment 3, a three-way interaction was found although effects were short-lived. As expected, watching food-related versus non-food related TV content resulted in more hedonic eating goal accessibility among people relatively high in eating restraint but low in PSRS (i.e., unsuccessful restrained eaters), but in less accessibility among participants relatively high in both eating restraint and PSRS (i.e., successful restrained eaters). DISCUSSION: As effects were found after watching a cooking show (Experiment 3) but not after watching TV commercials (Experiments 1 and 2), future research should explore whether the type of TV content might play a role in the effects of food-related TV content on hedonic eating goal accessibility[30]

We used functional magnetic resonance imaging (fMRI) to assess brain regions associated with food choices between appetizing (i.e., high sugar, high fat) and plain food in adolescents with excess weight and those with normal weight. The associations between choice-evoked brain activation and subjective food craving and behavioral food choices were also evaluated. METHODS: Seventy-three adolescents (aged 14-19 years), classified into excess weight (n = 38) or normal weight (n = 39) groups, participated in the study. We used a food-choice fMRI task, between appetizing and plain food, to analyse brain activation differences between groups. Afterwards, participants assessed their "craving" for each food presented in the scanner. RESULTS: Adolescents with excess weight showed higher brain activation in frontal, striatal, insular and mid-temporal regions during choices between appetizing and standard food cues. This pattern of activations correlated with behavioral food choices and subjective measures of craving. CONCLUSIONS: Our findings suggest that adolescents with excess weight have greater food choice-related brain reactivity in reward-related regions involved in motivational and emotional responses to food. Increased activation in these regions is generally associated with craving, and increased dorsolateral prefrontal cortex is specifically associated with appetizing food choices among adolescents with excess weight, which may suggest greater conflict in these decisions. These overweight- and craving-associated patterns of brain activation may be relevant to decision-making about food consumption.[31]

Associations with Other Risk Factors

Exciting new avenues of study including genomics, sex as a moderator of the stress response, and behavioral addictions (gambling, hypersexuality, dysfunctional internet use, and food as an addictive substance) are also briefly presented within the context of stress as a moderator of the addictive process.[32]

FA prevalence was 10.1% with a 95% confidence interval of 7.8-12.4%. 56.5% of all the participants had a poor quality of sleep, whereas 81.2% of the participants presenting FA experienced a poor sleep quality (versus 57.2% when no FA is present). 70.2% of the students presented an intermediate chronotype, 20.5% an evening chronotype and 8.7% a morning chronotype. Age, smoking status, BMI, PSS and PSQI remained significantly correlated to the continuous YFAS score in multivariate analysis. CONCLUSIONS: Our findings denote the importance of identifying and offering help to individuals presenting a FA because it is frequent among youth, associated to higher BMI and to smoking, seems to

be a very intertwined and complex phenomenon coexisting with other neuropsychiatric problems, such as stress and poor sleep quality and therefore can have serious health implications.[33]

Contrary to our hypotheses, SPFAs did not show increased AB to food-cues, and this was not moderated by hunger condition or the expectancy information. Exploratory analyses revealed that higher desire-to-eat (DtE) chocolate was associated with increased AB to chocolate pictures. These findings partially support contemporary theoretical models of AB by indicating a key role for state factors (reward expectancy, DtE) in determining AB to food-cues, while a trait factor (SPFA) was not a significant determinant of food AB.[34]

The impact of high levels of stress and trauma and altered metabolic environment (e.g. higher weight, altered insulin sensitivity) on prefrontal cortical self-control processes that regulate emotional, motivational and visceral homeostatic mechanisms of food intake and obesity risk are also discussed.[35]

Circadian Rhythms and Sleep

Participants were 1323 university students, filled out a package of psychological tools, including the Morningness-Eveningness Questionnaire, Insomnia Severity Index, Barratt Impulsiveness Scale Short Form, and Yale Food Addiction Scale. Logistic regression analysis was used to investigate direct relation of food addiction with insomnia, impulsivity and obesity, and mediation regression analysis was used to investigate the indirect effect of circadian rhythm differences on food addiction. RESULTS: Our findings indicated that evening types were more prone to insomnia and impulsivity, and also insomnia and impulsivity significantly contributed to the variance of food addiction. Although there was no significant linear relationship between circadian rhythm differences and food addiction, evening-type circadian preferences were indirectly associated with higher food addiction scores mediated by insomnia and impulsivity. CONCLUSION: The most remarkable result of our work was that circadian rhythm differences seem to indirectly effect on food addiction through elevated insomnia and impulsivity.[36]

When the brain circadian system is compromised in eating disorders, such perturbations may be in part the causes of compulsive feeding, night eating and addictive-like eating behavior. Therefore, food intake is regulated by the central circadian-metabolic-hedonic network, which is functionally interconnected to avoid perturbing the eating behavior physiology.[37]

Interventions

Eighteen participants were recruited to app intervention. App participants had higher retention (100% vs. 37%) and lower total cost per patient ($855.15 vs. $1428.00) than the EMPOWER clinic participants. App participants exhibited a significant decrease in zBMI and $\%BMI_{p95}$ over the 6 months ($p < 0.001$ and $p = 0.001$), which was comparable to the age-matched EMPOWER program completers ($p = 0.31$ and $p = 0.06$). Conclusions: An addiction medicine-based mHealth intervention targeted for adolescents was feasible to implement, resulted in high retention and adherence rates, and reduced zBMI and $\%BMI_{p95}$ in a more cost-effective manner than an in-clinic intervention.[38]

A key strategy in reducing the public health burden of cigarette smoking is preventing youth from ever becoming addicted to cigarettes in the first place. This study shows that by framing addiction as a loss of

control and tying that loss of control to short-term health and social consequences, addiction becomes more concrete and understandable, and the consequences feel more relatable and relevant to youth.[39]

Yale Food Addiction

The prevalence of food addiction was investigated using the Yale Food Addiction Scale for Children in overweight 9-11 year-old children (BMI/age ≥1 Z score) of both sexes from two schools (n = 139). Food intake was estimated by a food frequency questionnaire and the food items were classified into 4 categories: minimally processed, culinary ingredients, processed foods and ultra-processed foods (UPF), based on their degree of processing. Among the children, 95% showed at least one of the seven symptoms of food addiction and 24% presented with a diagnosis of food addiction. In analysis of covariance adjusted for age and sex, a tendency of higher consumption of added sugar (refined sugar, honey, corn syrup) and UPF was found among those diagnosed with food addiction. Multiple logistic regression adjusted for sugar, sodium and fat ingestion showed that consumption of cookies/biscuits (OR = 4.19, p = 0.015) and sausages (OR = 11.77, p = 0.029) were independently associated with food addiction.

Symptoms of food addiction were positively associated with smoking, alcohol use, cannabis use and sugar intake. (adolescents)[40]

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The aim of this paper was to review the clinical significance of food addiction diagnoses made with the YFAS and to discuss the results in light of the current debate on behavioral addictions. METHODS: We performed a systematic review of the studies that assessed food addiction with the YFAS published between January 2014 and July 2017 by searching the electronic databases PsycINFO, MEDLINE, and PsycARTICLES. RESULTS: Sixty publications were included in the analysis. Thirty-three studies examined nonclinical samples and 27 examined clinical samples. All studies used YFAS scoring results to define food addiction. The prevalence of food addiction according to the YFAS varied largely by the studied samples. In general, a higher body mass index and the presence of eating disorders (EDs), especially binge eating disorder (BED), were associated with higher YFAS scores. CONCLUSION: The concept of food addiction has not been established to this day although it can be grouped with other EDs such as BED.[41]

Articles were identified through PubMed and SCOPUS databases, resulting in a total of 19 studies which assessed food addiction among pre-bariatric and/or post-bariatric surgery patients using the Yale Food Addiction Scale. Most studies were cross-sectional, and only two studies prospectively measured food addiction both pre-operatively and post-operatively. The presence of pre-surgical food addiction was not associated with pre-surgical weight or post-surgical weight outcomes, yet pre-surgical food addiction was related to broad levels of psychopathology. The relationship between food addiction and substance misuse among individuals undergoing bariatric surgery is mixed. In addition, very few studies have attempted to validate the construct of food addiction among bariatric surgery patients. Results should be interpreted with caution due to the methodological limitations and small sample sizes reported in most studies.[42]

Tool was validated in Korean[43]

YFAS validated in Italy[44], and then Yale Food Addiction Scale Version 2.0 used in an Italian non-clinical sample.[45]
also used in Italy among undergraduates (2.0)[46]

2.0 used in Germany[47]

Validated for use among children in Hungary

Symptoms of 'food addiction' in binge eating disorder using the Yale Food Addiction Scale version 2.0. BED participants who met criteria for Moderate/Severe 'food addiction' reported significantly higher eating disorder psychopathology (except dietary restraint) as well as higher levels of anxiety and depression than BED participants with No/Mild 'food addiction'. Scores on the YFAS 2.0 positively predicted binge frequency, but not global eating disorder psychopathology, in the BED group after controlling for body mass index (BMI), depression and anxiety. The high rate of 'food addiction' symptoms in the BED group may reflect overlap between the symptoms assessed by the YFAS 2.0 and the clinical features of BED. A focus on identifying overlapping and distinctive underlying mechanisms rather than similarities and differences in clinical features might be a more fruitful avenue for future research on BED and 'food addiction'.[48]

One hundred thirty-one adults sought treatment for weight/eating concerns approximately 6 months post-sleeve gastrectomy surgery. The Eating Disorder Examination-Bariatric Surgery Version assessed LOC eating, picking/nibbling, and eating disorder psychopathology. Participants completed the Yale Food Addiction Scale (YFAS), the Beck Depression Inventory-Second Edition (BDI-II), and the Short-Form Health Survey-36 (SF-36). RESULTS: 17.6% met food addiction criteria on the YFAS. Compared to those without food addiction, the LOC group with food addiction reported significantly greater eating disorder and depression scores, more frequent nibbling/picking and LOC eating, and lower SF-36 functioning.[49]

A survey including the Yale Food Addiction Scale and several demographic questions demonstrated four distinct Yale Food Addiction Scale symptom severity groups (in line with Diagnostic and Statistical Manual of Mental Disorders (5th ed.) severity indicators): non-food addiction, mild food addiction, moderate food addiction and severe food addiction. Analysis of variance with post hoc tests demonstrated each severity classification group was significantly different in body mass index, with each grouping being associated with increased World Health Organization obesity classifications.[50]

The YFAS 2.0 exhibited a unidimensional structure, adequate internal consistency, and convergent and incremental validity. YFAS 2.0 scores contributed the largest percentage of unique variance in psychological distress and impairment over other BED features (overvaluation of weight and shape, binge eating, BMI), highlighting the clinical significance of the FA construct in BED. Support for the validity and reliability of the YFAS 2.0 in individuals with BED-like symptoms was found.[51]

Participants with YFAS-defined food addiction (6.7%) reported more frequent overall food cravings relative to those without food addiction. More frequent food cravings at baseline were associated with less weight loss over the 14 weeks. Analyzed categorically, participants in the highest tertile of baseline food cravings lost 7.6 ± 0.5% of initial weight, which was significantly less compared to those in the lowest tertile who lost 9.1 ± 0.5%. Percent weight loss did not differ significantly between participants with YFAS-defined food addiction (6.5 ± 1.2%) and those who did not meet criteria (8.6 ± 0.3%). Addictive-like eating behaviors significantly declined from pre- to post-treatment. Participants with frequent food cravings lost less weight than their peers. Targeted interventions for food cravings could improve weight loss in these individuals. Few participants met YFAS-defined criteria for food addiction. Addictive-like eating behaviors tended to decline during behavioral weight loss, but neither baseline nor change in YFAS scores predicted weight loss.[52]

Other Scales / Tools

Food addiction posits that highly processed foods may be capable of triggering addictive-like symptoms in some individuals, including withdrawal. The current study developed and assessed the psychometric properties of the first self-report measure of highly processed food withdrawal. Individuals (n = 231) aged 19-68 (51.9% female) were recruited online through Amazon Mechanical Turk and reported cutting down on highly processed foods in the past year. The Highly Processed Food Withdrawal Scale (ProWS) was adapted from self-report measures of drug withdrawal and internal consistency and validity were evaluated. Paralleling the course of drug withdrawal, symptoms assessed by the ProWS were reported as most intense between days 2-5 during an attempt to cut down. The ProWS demonstrated convergent validity with addictive-like eating ($r = 0.48$, $p < .001$), body mass index (BMI) ($r = 0.16$, $p = .02$), and weight cycling ($r = 0.29$, $p < .001$) and discriminant validity with dietary restraint: ($r = -0.13$, $p = .04$). The ProWS explained 11.2% of variance in self-reported success in last diet attempt beyond addictive-like eating and BMI. The ProWS seems to be a psychometrically sound tool for future research investigating highly processed food withdrawal in humans, and the present data may provide preliminary insight into the plausibility of withdrawal symptoms occurring in response to cutting down on highly processed food[53]

The Binge Eating Scale (BES) is a widely used self-report questionnaire to identify compulsive eaters. However, research on the dimensions and psychometric properties of the BES is limited. Objective: The aim of this study was to examine the properties of the Spanish version of the BES.[54]

This article proposes a heuristic framework for the Addictions Neuroclinical Assessment that incorporates key functional domains derived from the neurocircuitry of addiction. We review how addictive disorders (ADs) are presently diagnosed and the need for new neuroclinical measures to differentiate patients who meet clinical criteria for addiction to the same agent while differing in etiology, prognosis, and treatment response. The need for a better understanding of the mechanisms

provoking and maintaining addiction, as evidenced by the limitations of current treatments and within-diagnosis clinical heterogeneity, is articulated[55]

Policy and Solutions

The concept of food addiction has generated much controversy. In comparison to research examining the construct of food addiction and its validity, relatively little research has examined the broader implications of food addiction. The purpose of the current scoping review was to examine the potential ethical, stigma, and health policy implications of food addiction. Major themes were identified in the literature, and extensive overlap was identified between several of the themes. Ethics sub-themes related primarily to individual responsibility and included: (i) personal control, will power, and choice; and (ii) blame and weight bias. Stigma sub-themes included: (i) the impact on self-stigma and stigma from others, (ii) the differential impact of substance use disorder versus behavioral addiction on stigma, and (iii) the additive stigma of addiction plus obesity and/or eating disorder. Policy implications were broadly derived from comparisons to the tobacco industry and focused on addictive foods as opposed to food addiction. This scoping review underscored the need for increased awareness of food addiction and the role of the food industry, empirical research to identify specific hyperpalatable food substances, and policy interventions that are not simply extrapolated from tobacco.[56]

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