Chapter 1

Eating Disorders as New Forms of Addiction

Francisco J. Vaz-Leal, María I. Ramos-Fuentes,
Laura Rodríguez-Santos and
M. Cristina Álvarez-Mateos

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/65248

Abstract

Eating disorders (ED) seem to share many characteristics with substance-related disorders (SRD). As very often the two conditions run together, it has been proposed that eating dysfunctions could be understood as behavioral forms of addiction. This has lead to the concept of "food addiction," a proposed new form of addiction. This chapter reviews recent research focusing on the relationship between ED and SRD. Three specific areas are addressed: (a) animal models that suggest the association between substance dependence and compulsive overeating; (b) epidemiological studies that confirm the comorbidity between ED and SRD; and (c) neuroimaging studies that reveal the existence of modifications in the reward circuits following binge eating and other eating dysfunctions. The data from the different studies can be integrated into a model based on the consideration of "food addiction" as a specific form of behavioral addiction that could be applied at least to a group of patients suffering from eating disorders.

Keywords: eating disorder, substance-related disorder, anorexia nervosa, bulimia nervosa, binge eating disorder

1. Introduction

In the past, the study of similarities between eating disorders (ED) and substance-related disorders (SRD) has been limited to the consideration of the similarities in their clinical presentation and the existence of some common behaviors. Nevertheless, in recent years there has been growing interest in the study of the association between ED and SRD, and a significant event in this context has been the inclusion of binge eating disorder (BED) as an ED with full

identity in the DSM-5. This new diagnosis has prompted the development of theories and studies that suggest the existence of a model of "food addiction," capable of explaining many observed similarities between ED and SRD, at least in some cases.

The term "food addiction" is a recent and in many ways controversial concept, proposed by Randolph in 1956 to refer to several eating patterns which were "descriptively similar to those of other addictive processes" [1]. According to the concept of "food addiction", certain foods will act as most psychoactive substances do, producing changes in brain function that would made necessary further use of the substance (or food) to restore balance in the affected areas. Although Randolph highlighted the addictive potential of foods such as wheat and corn derivatives, coffee, eggs or milk, nowadays the term is associated with the consumption of foods with high sugar and fat content.

A term related to "food addiction" is "food craving", a feature that goes beyond the simple desire to eat and the search for specific foods. It has to do in many cases with specific (negative) emotional states, in such a way that the seeking behavior can be explained in terms of emotional regulation and is not necessarily followed by increased eating [2, 3].

Several theories have been proposed to understand "food craving". Some of them link this feature with the evolution of the humans, and their tendency to overeat when food is clearly avalaible, specially after a scarcity period. Neurotransmiters (serotonin, dopamine, endogenous opiates, ghrelin, cholecystokinin, peptide YY and leptine, among others) appear to be involved in the complex control of (as well as in the loss of control over) eating [4]. Food intake is clearly influenced by metabolic activity in hypothalamic and extracerebral areas, including sensitivity of the hypothalamus to specific molecules, stomach mouvements or blood glucose levels [5], and organoleptic characteristics of some kinds of food (as for example, chocolate) seem to be element linked to the decisión to seek out for food and to start eating. Finally, from a cognitive framework, the so-called "elaborated intrusion theory" has postulated the existence of two main cognitive components in food craving: the emergence of intrusive thoughts about the craved substance via an associative process, and the emergence of supporting mental images via an elaborative process [6].

2. Food and substances

According to the American Society of Addiction Medicine, addiction is a chronic primary disease, which affects some brain circuits related to reward, memory, and motivation, producing significant changes at a biological and psychological level. Like other chronic diseases, cycles of remission and relapse are often observed. Without treatment or participation in recovery activities, addiction is progressive and can lead to disability or premature death [7].

Supporting the idea that food is "addictive" implies accepting that food contains ingredients capable of creating addiction in susceptible individuals. Except for caffeine, which has already been introduced in the DSM-5, so far there is no enough scientific evidence to support this assert. Some foods have rewarding properties, especially manipulated aliments which have

been designed to maximize flavor, and can therefore participate more easily in the processes of reinforcement and reward [8]. Nevertheless, despite the fact that food and substances can act on the same brain circuits, eating habits are not only regulated by central brain structures, but also by peripheral mechanisms [9]. Another important fact is that food is necessary for survival, while recreational drugs are not. However, it seems clear that both elements, food and substances, may have similar addictive capability, as they share several elements that seem to be able to initiate and maintain their use, and have in common their high resistance to treatment [10].

Animal research has revealed how the following factors (included in the DSM-5 definition of SRD) are not only associated with substance use but also with excessive food intake, especially of food with high sugar content [10]: (a) intake/use in larger amounts or over longer periods than was intended; (b) persistent desire or unsuccessful efforts to cut down or control the intake/use; (c) great deal of time spent in activities necessary to obtain, use, or recover from its effects; (d) craving or strong desire or urge to eat/use; (e) maintenance of the pattern of intake/use despite having social or interpersonal problems caused or exacerbated by the intake/use; (f) reduction in social, occupational, or recreational activities; and (g) recurrent intake/use in situations where it is physically hazardous.

In accordance with these facts, some authors have defended the idea that overeating can be considered as an addictive behavior, especially in the cases that the individual who overeats fulfills the diagnostic criteria for BED. It is evident that some individuals are physically and psychologically dependent on foods with high fat and sugar content in a way that resembles substance dependence. It is also usual in these subjects the emergence of feelings of loss of control over consumption, as well as the maintenance of such behaviors despite the serious consequences that can have on health and social life. Another relevant element is that both drug abuse and binge eating are often accompanied by strong feelings of guilt, remorse, and anguish [11]. In short, there are reasons to support the idea that at least some ED could be conceptualized as SRD. This not only applies to BED, as the addictive nature of other eating-related behaviors, such as diet, compulsive exercise, purging, or the use of substances that promote weight loss, can also be defended. In any case, further research is needed, because tolerance and withdrawal (two key elements in addictions) have not been clearly observed in humans in relation to food [10].

Numerous investigations have attempted to isolate the elements of the neurotransmission systems that may be involved in the pathophysiology of ED and SRD [10]. Serotonin seems to be a relevant one, as it plays an important role in behavioral inhibition and impulsivity, two clinical items related to the psychopathology of ED and SRD. Norepinephrine, by its part, seems to be more related to the cognitive and emotional factors able to trigger these behaviors, but undoubtedly the star of the reward system is dopamine, the main neurotransmitter in the mesolimbic system [10]. If we feel pleasure is because dopamine (released from the neurons of the ventral tegmental area in response to a reinforcing stimulus) increases in the nucleus accumbens. Once the system has been activated, endocannabinoids and endorphins are responsible for extending the pleasant sensation; finally, the interaction of these and other neurotransmitters, such as acetylcholine, glutamate, or gamma-amino-butyric acid, supports

the subjective feeling of reward [12]. In the case of restrictive eating disorders such as anorexia nervosa (AN), reward seeking seems to be associated to other elements, as drive for thinness, which is experienced as pleasurable; the maintenance of the feeling of satisfaction produced by weight lose and the immediate sense of control over hunger and eating associated with food restriction can also act as positive reinforcement elements in these cases [13].

ED and SRD overlap in many other important aspects, including clinical phenomenology, comorbidity, and response to certain types of treatment [10], but the final reasons for this association are unknown, and it is also difficult to determine which disorder occurs first, acting as a risk factor for the development of the other. What is not in doubt is the close relationship between them, as is revealed by many studies that report on the high comorbidity in people suffering from these disorders, when compared with general population.

3. Animal models: what are they teaching us?

Animal models are contributing to the understanding of the neurobiological basis of the behaviors associated with ED, bringing to light some of the mechanisms and the brain areas involved in food intake dysregulation [14].

A recent review of animal studies on compulsive overeating concludes that, under certain circumstances, overeating can produce behavioral and physiological changes similar to those observed in addictions, in such a way that addictive behaviors should not limit to substance use [15]. In fact, numerous animal studies have revealed that many of the behaviors observed in subjects with problems of drug addiction can be also associated with massive and persistent consumption of palatable food. Some of these common elements are as follows:

(a) Loss of control and compulsive consumption: In the model of sugar addiction, the animals are subjected to periods of food deprivation followed by equally long periods of free access to a sweet solution. When this routine is maintained for a time, an escalation in the daily intake of sweet solution is observed, especially in the first hour of access, with a change in eating patterns in the long term to make possible the intake of a larger amount of sugar during the period of free access [16].

(b) Use despite negative consequences: As has been already said, there are models of obesity and BED that measure the motivation of the animals to find and eat palatable foods despite potentially harmful consequences. It has been found that expanding access to palatable food of high energy density induces compulsive behavior in obese animals despite the application of a negative stimulus. It has also been found that reduction in dopamine D_2 receptors in the striatum of these animals exists, a phenomenon reported in humans addicted to drugs [17].

(c) Inflexible behavior: According to the results of some studies, animals often show an inflexible pattern of behavior, preferring limited access to appetizing food than continuous access to their usual food [18].

(d) Withdrawal symptoms and relapse: There are several animal studies on forced abstinence, but offer contradictory results, that in many cases depend on the type of food to be offered/restricted. According to the results of one of these studies, the excessive consumption of sugar will act similarly to opiate use both at brain and at behavioral levels, with the emergence of withdrawal symptoms following sugar restriction [19]. In animals fed with unlimited access to glucose, abstinence can be induced by the deprivation of sugar or the administration of naloxone, an opiate antagonist that increases the symptoms and somatic signs of withdrawal, such as tremors, shaking, teeth chattering, hyperphagia, and anxiety [20]. However, sugar and fat seem to have notable differences: while too much sugar can produce symptoms similar to those of opiates withdrawal and does not affect body weight, binges of fat cause weight gain but do not produce withdrawal symptoms [21]. The animals on a high-fat diet show no behavioral or somatic signs of withdrawal following the administration of naloxone [22]. According to these facts, combined diets (high sugar and fat content) could be able to produce both addictive symptoms and significant weight changes [23], with increased anxiety levels and psychomotor excitement following acute withdrawal [24].

With respect to BED, several models that highlight the etiological role of factors such as stress or food deprivation have been proposed. A recent review of the literature on animal models has evaluated the contribution of different studies to the understanding of BED [25]. The animal studies analyzed suggest that binge eating is often associated with changes in behavior similar to those observed in individuals who use/abuse substances. These behaviors are not observed in animals that, taking the same food, do not binge. This has led to the conclusion that food has no addictive qualities in itself and that the really important factor is the conditions under which food intake takes place, suggesting the replacement of the term "food addiction" by "addiction to eat" [8].

The role of stress in the genesis of binge eating has been one of the items analyzed in these studies. When animals are subjected to cycles of caloric restriction and exposed to tasty food associated (or not) with a stressor, the food intake of animals exposed to the stressor seems to be significantly higher than those who do not receive negative stimuli [25].

Another element analyzed is the propensity to binge eating. Once the animals have been classified into susceptible or resistant to binge, on the basis of their previous intake of palatable food, important behavioral differences that suggest the existence of an addictive pattern in animals prone to binge seem to exist. In this sense, whereas binge-resistant animals tend to reduce food intake following the exposure to negative stimuli, the animals prone to binge do not reduce their intake and continue eating the same amount of tasty food, enduring negative contingencies to obtain the appetizing food, not only when they are hungry, but also when they are satiated [26].

The results from another group of animal studies defend the idea that a limited and intermittent access to palatable food can be a sufficient cause for excessive intake. The intermittent access to palatable food attempts to reproduce one of the main items of the diagnostic criteria for BED, that is, the massive food intake occurring over a limited period of time. The model is based on the hypothesis that "forbidden" foods that people use to restrict when dieting are

the trigger element of binge eating. This hypothesis has been supported by one study in which a group of animals with continuous access to their usual food and water were divided into two groups: animals with limited access to palatable food in the short time, but with daily access, and animals with intermittent access to tasty food on alternate days. According to the results of the study, the group of animals with intermittent access tended to present a significantly higher intake of palatable food. In addition, as the animals have no periods of caloric restriction during the experience, the statement of "overeating in the absence of hunger" is fulfilled, which corresponds to other main criteria of BED [27, 28].

Some studies have analyzed the changes in food intake after a mild and daily food restriction period which is followed by access to palatable food. In this kind of studies, and in contrast to the previously reported ones, the animals cannot have access to other types of food. The justification for this difference in the design of the study is that often people with BED are involved in periods of energy deprivation. In these studies, the animals are deprived for a period of time of several hours, and then, they are exposed to palatable food for a short time. Studies have shown that consumption in the early stages is higher in this group of animals than in the control groups, consisting of subjects with continuous access to palatable food or limited access to their usual food [29].

Combining different approaches, such as acute intermittent access or deprivation of food, another group of studies have attempted to reproduce BED in laboratory animals. The animals used in these studies had access to tasty food for short periods of time in two noncontiguous days per week. Over a long period of time prior to access, the animals were deprived of all food, while the rest of the day had continuous access to their usual food. At the end of the study period, an escalation in food intake was observed in these animals, compared with control subjects [30].

In summary, animal models seem to be able to provide valuable information for the understanding of the pathophysiology and course of eating disorders, especially for the recently identified as BED. They also reveal the existence of some addictive components of binging, being the results of vital importance for the rising of the concept of "food addiction." However, due to the undeniable influence of psychosocial factors, more research is needed before we can translate the results of basic research to the field of clinical management of the human patients.

4. Drugs and addictions: a question of comorbidity

A number of clinical studies have revealed the existence of high comorbidity between ED and SRD. These studies are usually based on population surveys or comparisons between groups and address comorbidity from different perspectives, whether looking for SRD in samples of patients with ED or investigating the presence of TCA in people with SRD. There are also some meta-analysis studies and others related to specific populations, such as adolescents, or specific subtypes of eating disorders.

A meta-analysis of studies published between 1994 and 2007 has concluded that substance use is higher in patients with ED than in healthy controls. In addition, it seems that differences

between the subtypes of ED exist, being the risk of substance abuse higher in bulimia nervosa (BN), followed by BED and nonsignificant in AN [31].

One of the studies on the prevalence of SRD in AN has been developed after classifying patients according to their subtype. The findings are in accordance with the above-mentioned results. A quarter of the total sample referred history of substance use, being more common in purgative AN (about 30%). Thirteen percent of the participants in the study met criteria for substance abuse or dependence, especially the AN+BN group (32%). Restrictive AN patients had the lowest percentages for substance use (23%) and dependence (6%). Alcohol intake was detected in 20% of the sample, being more frequent in AN+BN patients (35.5%) and again less frequent in the restrictive forms of AN (14%). In addition, a statistically significant relationship between substance use and purges, but not between substance use and binge eating, was found in this study [32].

Although obesity is not considered an ED, but a chronic multifactorial disease, there is growing evidence on the coexistence of psychopathological disturbances. On the issue that is concerning us, there are a pair of publications that found a strong association between obesity and alcohol abuse [33, 34].

In order to examine comorbidities related to the new BED diagnosis, a group of patients were examined; the results demonstrated that the psychopathological associations were evident. Seventy-four percent of the patients suffer from at least one lifetime additional psychiatric disorder, being SRD present in one in four subjects, especially in male subjects. Individuals suffering from BED plus other psychiatric comorbid disorder had significantly higher body mass index (BMI) and higher levels of psychopathology [35].

Addressing comorbidity from the other side, some studies have been carried out on SRD women, revealing that at least 15% of them were at high risk of developing ED, reaching the lifetime prevalence 20%. On the other hand, the relationship of these disorders with the prognosis seems to be statistically significant, so that ED would represent an important predictor of relapse or unsuccessful treatment for patients with SRD [36]. In a similar way, other study of women in treatment for SRD has examined how "weight-related concerns" affected substance use. More than 70% of the participants admitted to be worried about gaining weight during recovery and 45% were concerned about whether weight gain could be in the future a reason for relapse. Approximately one-third of the sample admitted that weight loss had been a reason to initiate or maintain substance use and most of them admitted having used a variety of methods to change their figure, including substance use. Twenty-six percent of the subjects ate too much, and 13% skipped meals. Up to 40% of the participants admitted that their addictive behavior affected their eating patterns. Women who were concerned about weight gain had higher levels of body dissatisfaction, more eating symptoms (especially bulimic symptoms) and tended to use substances capable of modifying eating and weight [37].

Adolescents have been a target population for several epidemiological studies on the potential risk to develop psychopathological disorders. Studies in this field have stressed the influence of family and environmental factors in the process. When BMI (stratified by gender), dietary behaviors, and substance use have been examined, controlling for the influence of social and

family factors, both obesity and underweight have appeared initially associated with increased substance use (20–40%), being overweight women more prone to ED and substance use more frequent in male participants. When a subset of them (people meeting criteria for overweight and substance abuse) was compared with teens who had just overweight, the first ones were characterized by an unconventional family structure, previous history of negative sexual experiences, and serious family and school problems. When, finally, the underlying psychosocial factors were controlled, the relationship between substance use and overweight disappeared [38]. In this same group of age, some studies have revealed the influence of the spectrum of compulsiveness on the emergence of ED and excessive alcohol intake. Compulsive symptoms seem to be especially associated with ED and obsessive-compulsive disorder and to a lesser extent with SRD and excessive alcohol intake. Nevertheless, in this last case the external factors seemed to be more relevant [39]. In a cohort study, aimed at determining the prevalence of ED in a university sample, the risk of developing an ED was 19.5%, being higher in women than in men. Among the isolated risk factors, excessive intake of alcohol ("binge drinking"), smoking, and/or problematic Internet use appeared as specially significant [40].

5. Does food addiction exist?

Despite the similarities between substance addiction and excessive food intake, only recently specific tools to measure "food addiction" have been developed. In 2008, the Yale Food Addiction Scale (YFAS) was proposed as an instrument for identifying signs of addiction to certain foods. The scale was based on a selection of criteria for substance dependence included in the fourth revised edition of the DSM (DSM-IV-TR), and other scales used to assess behavioral addictions, such as pathological gambling [41]. The items of the scale were adapted to evaluate excessive intake of high-calorie foods and were reviewed by experts in addictions, obesity, and ED. In 2013, a version of the YFAS for children (YFAS-C) was proposed [42]. According to the results, both YFAS for adults and YFAS for children seem to be solid psychometric tools, capable of identifying dysfunctional addictive eating patterns.

To study the association of "food addiction" with different ED, a wide group of subjects with their BMI ranging from underweight to severely obese were studied. According to YFAS, "food addiction" was found in more than a quarter of participants, being more prevalent in participants with BN than in those with BED. People with "food addiction" not only had higher BMI, greater fluctuations in body weight, and more dietary behaviors; all the variables related to ED (binge eating, dietary restrictions and concerns about weight and food) were also more prevalent in them, in such a way that the study revealed significant relationship between "food addiction" and eating psychopathology [43]. Other study analyzed the prevalence of "food addiction" in a sample of women diagnosed with different subtypes of ED, using healthy women as controls. The lowest scores on the YFAS were associated with the restrictive subtype of AN and the highest ones with the subtypes of ED associated with binge eating: purging AN (85.7%), BN (81.5%), and BED (76.9%). Higher YFAS scores were also associated with higher levels of negative affect, depressive symptoms, higher BMI, more severe eating pathology, and general psychopathology [44].

The association of BED and "food addiction" could be considered a severe form of the disorder, as shown by a study on a sample of obese patients with BED. [45]. In that sample, 41.5% of the subjects met the criteria for "food addiction" according to the YFAS. These patients had higher levels of negative affect, emotional dysregulation and eating psychopathology, and lower self-esteem. Higher scores in the YFAS were significant predictors of a higher frequency of binging and were also associated with earlier age of onset of overweight and dietary behaviors. In the same way, another study analyzed a clinical set of variables in a sample of overweight men and women who met diagnostic criteria for BED [46]. The subjects with higher scores in "food addiction" were more prone to impulsively overeat for emotional reasons, had more frequent and severe episodes of binge eating, and were more sensitive to the rewarding properties of food. In addition, they had more addictive personality traits, higher levels of impulsivity and reported more depressive symptoms. A third study tried to test the hypothesis that "food addiction" and BED were inseparable conditions [47], suggesting that overeating should be understood as a dimension that reflects different levels of severity. According to the results, the lower edge of the continuum will be represented by people who overeat passively, with little (or no) associated psychopathology; the upper edge, by its part, will be characterized by significant clinical deterioration and strong addictive behaviors and will correspond with BED. In this context, "food addiction" will represent a more severe and more psychopathologically serious form of BED.

These observations are in accordance with the results of a systematic review of the studies published from 2009 to 2014. The meta-analysis revealed that, according to the YFAS, the mean prevalence of the diagnosis of "food addiction" was almost 20%, being higher in patients suffering from BN than in cases of BED. In accordance with most of the aforementioned studies, "food addiction" was associated with higher tendency to binge eating, depressive symptoms, impulsivity, and emotional eating [48].

So far we have only considered those studies that measured "food addiction" using the YFAS, because it is the most standardized instrument. However, there are other scales and questionnaires that have been created with the same purpose. One example is a study performed on a sample of obese women in which the DSM-5 criteria for SRD were adapted, replacing "substance" by "food" [49]. The subjects fulfilled the new criteria for "food addiction," but those with BED described their binges as more "addictive," especially with regard to craving and the "use despite having persistent or recurrent physical or psychological problems." The group with BED also reported greater number and frequency of their binge episodes. Similar findings are provided by a study that applied the Goodman's criteria for SRD to the study of different subtypes of ED [50], comparing patients with restrictive AN, purgative AN, BN, SRD, and healthy controls. According to the results of the study, BN patients met the Goodman's criteria in the same proportion as the subjects with SRD (60 and 65%, respectively, compared with only 35% of patients with restrictive AN), in such a way that the concept of "food addiction" seems to be closely tied to binge and uncontrolled eating.

6. Looking at the brain: neuroimaging studies

One of the emerging fields of research in the study of the relationships between ED and SRD is neuroimaging. The literature provides us with a number of studies that shed some light on the biological basis of addictive behaviors and disordered eating. In general, neuroimaging techniques have revealed the existence of common neurotransmitter disturbances in specific anatomical regions and circuits in SRD and ED. These abnormalities mainly affect the prefrontal cortex (involved in inhibitory control), hippocampus (strongly linked to memory), and striatum (involved in reward, motivation and habit formation) [51].

Functional magnetic resonance imaging (fMRI) studies have shown specific changes in brain activity in patients with ED following the exposure to food images. Patients with AN, for example, tend to react with an increase in visual responses and prefrontal cortical activity that seem to be in accordance with their cognitive biases and excessive control over intake [52]. When patients with AN, BN, and controls are compared, each group seems to have a more or less specific pattern of response. The women with BN tend to present greater neural activity in the visual cortex, right dorsolateral prefrontal cortex, right insular cortex, and precentral gyrus. Compared to controls, the patients with BN had lower activity in the superior temporal gyrus, bilateral insula, and visual cortex. In contrast to women with AN, the patients with BN had lower activity in the posterior dorsal and parietal lobe of the cingulate cortex, but increased activity in caudate, superior temporal gyrus, right insula, and supplementary motor area [53].

When the response to the stimulation preceding the administration of food has been studied in patients with "food addiction," the results have also shown specific changes, as increased activation of anterior cingulate cortex, medial orbitofrontal cortex, and amygdala. A positive correlation between YFAS score and brain activity, with a pattern similar to that produced by drugs or alcohol, has also been found. These results reveal that specific patterns of neural activation are involved in "addictive" eating behavior and that in subjects with "food addiction," the activity of reward circuits increases in response to signals related to food [54].

There are several studies aimed at examining whether the neuronal hyperstimulation of reward pathways, typically associated with SRD, also occurs in individuals with binge episodes. The fMRI images obtained from overweight individuals who binge when they are exposed to food-related stimulus reveals that the increase in reward system activity correlates with BMI and with the number of reported binges. Caloric stimulation seems to activate the cingulate gyrus, the supplementary motor area, the thalamus and the temporal lobe, as well as the occipital areas related to visual processing and the regions involved in emotional processes, such as amygdala and hippocampus. According to these data, "food addiction" would be determined by a failure of self-regulation mechanisms [55].

Some studies of positron emission tomography (PET) have suggested that the striatal dopamine signaling process is impaired in obese people, as a consequence of the decrease in D_2 receptors, in a way similar to that observed in patients with SRD. A review of PET studies confirmed this point of view, revealing that the binding to the dopamine D_2/D_3 receptors is decreased not only in cocaine, methamphetamine, alcohol, heroin, and nicotine addicts, but

also in subjects with morbid obesity, compared with healthy controls [56]. PET studies have also been performed to assess the role of dopamine in the neurobiology of BED and obesity [57], through the quantification of the changes in extracellular striatal dopamine after food stimulation. The food stimuli, when administered with methylphenidate (a drug that blocks dopamine reuptake), caused changes in binge eaters, but not in the obese subjects who did not binge. In addition, increased levels of dopamine in the caudate were significantly correlated with binge eating, but not with BMI. In the same way, another study of PET imaging has detected alterations in dopamine pathways similar to those described in patients with SRD in a group of women with BN. The analysis of the data found a significant association between striatal dopamine release and the frequency of binge eating and vomiting in these patients [58].

Although the application of neuroimaging in the field of Psychiatry is still at an early stage of development, it represents a solid promise for the understanding of ED, SRD, and their relationships, in such a way that it is very possible that future research can help us to treat and prevent relapse in these patients more effectively.

Acknowledgements

Supported by grant no. GR15131 (Support to the Action Plans of Registered Research Groups). Consejería de Economía e Infraestructuras. Junta de Extremadura (Spain), and European Social Fund.

Author details

Francisco J. Vaz-Leal[1,2*], María I. Ramos-Fuentes[1], Laura Rodríguez-Santos[1] and M. Cristina Álvarez-Mateos[1]

*Address all correspondence to: fjvazleal@gmail.com

1 University of Extremadura School of Medicine, Badajoz, Spain

2 University Hospital Network, Badajoz, Spain

References

[1] Randolph TG. The descriptive features of food addiction; addictive eating and drinking. Q J Stud Alcohol 1956;17:198-224.

[2] Giuliani NR, Berkman ET. Craving is an affective state and its regulation can be understood in terms of the extended process model of emotion regulation. Psychol Inq 2015;26:48–53. DOI: 10.1080/1047840X.2015.955072

[3] Meule A, Gerhardt. Food addiction on the light of DSM-5. Nutrients 2014;6:3653–71. DOI: 10.3390/nu6093653

[4] Schwartz MW, Woods WC, Porte D, et al. Central nervous system control of food intake. Nature. 2000;404:661–71.

[5] Woods SC, Ramsay DS. Food intake, metabolism and homeostasis. Physiol Behav. 2011; 104:4–7. DOI: 10.1016/j.physbeh.2011.04.026

[6] Andrade J, Pears S, May J, Kavanagh DJ. Use of a clay modeling task to reduce chocolate craving. Appetite. 2012;58:955-63. DOI: 10.1016/j.appet.2012.02.044.

[7] American Society of Addiction Medicine (ASAM). Public policy statement: definition of addiction [Internet]. 2011. Available from: http://www.asam.org/quality-practice/definition-of-addiction [Accessed: 2016-06-27].

[8] Hebebrand J, Albayrak Ö, Adan R, Antel J, Dieguez C, de Jong J, et al. "Eating addiction", rather than "food addiction", better captures addictive-like eating behavior. Neurosci Biobehav Rev. 2014;47:295-306. doi:10.1016/j.neubiorev.2014.08.016.

[9] DiLeone RJ, Taylor JR, Picciotto MR. The drive to eat: comparisons and distinctions between mechanisms of food reward and drug addiction. Nat Neurosci. 2012;15:1330-5. DOI:10.1038/nn.3202.

[10] Brewerton T, Baker A, editors. Eating Disorders, Addictions and Substance Use Disorders: Clinical and Treatment Perspectives. New York: Springer; 2014. 664 p. DOI: 10.1007/978-3-642-45378-6.

[11] Smith DG, Robbins TW. The neurobiological underpinnings of obesity and binge eating: a rationale for adopting the food addiction model. Biol Psychiatry. 2013;73:804-10. DOI:10.1016/j.biopsych.2012.08.026.

[12] Méndez M, Ruiz AE, Prieto B, Romano A, Caynas S, Prospero O. Brain and substances: their neurobiological mechanisms. Salud Mental. 2010;33:451-56. Available from: http://www.redalyc.org/articulo.oa?id=5821079309. [Accessed: 2016-06-27].

[13] Godier LR, Park RJ. Compulsivity in anorexia nervosa: a transdiagnostic concept. Front Psychol. 2014;5:778. doi:10.3389/fpsyg.2014.00778.

[14] Avena NM, Bocarsly ME. Dysregulation of brain reward systems in eating disorders: neurochemical information from animal models of binge eating, bulimia nervosa, and anorexia nervosa. Neuropharmacology. 2012;63:87-96. doi:10.1016/j.neuropharm.2011.11.010.

[15] Di Segni M, Patrono E, Patella L, Puglisi-Allegra S, Ventura R. Animal models of compulsive eating behavior. Nutrients 2014;6:4591-609. doi:10.3390/nu6104591.

[16] Hoebel BG, Avena NM, Bocarsly ME, Rada P. Natural addiction: a behavioral and circuit model based on sugar addiction in rats. J Addict Med. 2009;3:33–41. doi:10.1097/ADM.0b013e31819aa621.

[17] Johnson PM, Kenny PJ. Addiction-like reward dysfunction and compulsive eating in obese rats: role for dopamine D_2 receptors. Nat Neurosci. 2010;13:635–41. Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2947358/pdf/nihms181674.pdf [Accessed: 2016-06-27]. doi:10.1038/nn.2519.

[18] Heyne A, Kiesselbach C, Sahún I, McDonald J, Gaiffi M, Dierssen M, Wolffgramm J. An animal model of compulsive food-taking behaviour. Addict Biol. 2009;14:373-83. doi:10.1111/j.1369-1600.2009.00175.x.

[19] Avena NM, Gold MS. Food and addiction-sugars, fats and hedonic overeating. Addiction. 2011;106:1214–5. doi:10.1111/j.1360-0443.2011.03373.x.

[20] Avena NM, Rada P, Hoebel BG. Evidence for sugar addiction: behavioral and neurochemical effects of intermittent, excessive sugar intake. Neurosci Biobehav Rev. 2008;32:20–39. Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2235907/[Accessed: 2016-06-27] doi:10.1016/j.neubiorev.2007.04.019.

[21] Avena NM, Rada P, Hoebel BG. Sugar and fat bingeing have notable differences in addictive-like behavior. J Nutr. 2009;139:623–8. Available from: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2714381/[Accessed: 2016-06-27]. doi: 10.3945/jn.108.097584.

[22] Bocarsly ME, Berner LA, Hoebel BG, Avena NM. Rats that binge eat fat-rich food do not show somatic signs or anxiety associated with opiate-like withdrawal: implications for nutrient-specific food addiction behaviors. Physiol Behav. 2011;104:865–72. doi: 10.1016/j.physbeh.2011.05.018.

[23] Berner LA, Avena NM, Hoebel BG. Bingeing, self-restriction, and increased body weight in rats with limited access to a sweet-fat diet. Obesity. 2008;16:1998–2002. doi: 10.1038/oby.2008.328.

[24] Teegarden SL, Bale TL. Decreases in dietary preference produce increased emotionality and risk for dietary relapse. Biol Psychiatry. 2007;61:1021–29. doi:10.1016/j.biopsych.2006.09.032.

[25] Corwin RL, Babbs RK. Rondent models of binge eating: are they models of addiction?. ILAR J. 2012;53:23-34. doi:10.1093/ilar.53.1.23.

[26] Boggiano MM, Artiga AI, Pritchett CE, Chandler-Laney PC, Smith ML, Eldridge AJ. High intake of palatable food predicts binge-eating independent of susceptibility to obesity: an animal model of lean vs obese binge-eating and obesity with and without binge-eating. Int J Obes. 2007;31:1357-67. doi:10.1038/sj.ijo.0803614.

[27] Corwin RL, Avena NM, Boggiano MM. Feeding and reward: perspectives from three rat models of binge eating. Physiol Behav. 2011;104:87–97. doi:10.1016/j.physbeh.2011.04.041.

[28] Corwin RL, Wojnicki FH. Binge eating in rats with limited access to vegetable shortening. Curr Protoc Neurosci. 2006;36:9.23B:9.23B.1–9.23B.11. doi:10.1002/0471142301.ns0923bs36.

[29] Colantuoni C, Rada P, McCarthy J, Patten C, Avena NM, Chadeayne A, Hoebel BG. Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. Obes Res. 2002;10:478–88. doi:10.1038/oby.2002.66.

[30] Bello NT, Guarda AS, Terrillion CE, Redgrave GW, Coughlin JW, Moran TH. Repeated binge access to a palatable food alters feeding behavior, hormone profile, and hindbrain c-Fos responses to a test meal in adult male rats. Am J Physiol Regul Integr Comp Physiol. 2009;297:622-63. doi:10.1152/ajpregu.00087.2009.

[31] Calero-Elvira A, Krug I, Davis K, Lopez C, Fernández-Aranda F, Treasure J. Meta-analysis on drugs in people with eating disorders. Eur Eat Disord Rev. 2009;17:243–59. doi:10.1002/erv.936.

[32] Root T, Pinheiro AP, Thornton L, Strober M, Fernández-Aranda F, Brandt H, et al. Substance use disorders in woman with anorexia nervosa. Int J Eat Disord. 2010;43:14–21. doi:10.1002/eat.20670.

[33] Petry NM, Barry D, Pietrzak RH, Wagner JA. Overweight and obesity are associated with psychiatric disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. Psychosom Med. 2008;70:288-97. doi:10.1097/psy.0b013e3181651651.

[34] Grucza RA, Krueger RF, Racette SB, Norberg KE, Hipp PR, Bierut LJ. The emerging link between alcoholism risk and obesity in the United States. Arch Gen Psychiatry. 2010;67:1301-08. doi:10.1001/archgenpsychiatry.2010.155.

[35] Grilo CM, White MA, Masheb RM. DSM-IV psychiatric disorder comorbidity and its correlates with binge eating behavior. Int J Eat Disord. 2009;42:228–34. doi:10.1002/eat.20599.

[36] Bonfá F, Cabrini S, Avanzi M, Bettinardi O, Spotti R, Uber E. Treatment dropout in drug-addicted women: are eating disorders implicated? Eat Weight Disord. 2008;13:81-6. doi:10.1007/BF03327607.

[37] Warren CS, Lindsay AR, White EK, Claudat K, Velasquez SC. Weight-related concerns related to drug use for women in substance abuse treatment: prevalence and relationships with eating pathology. J Subst Abuse Treat. 2013;44:494-501. doi:10.1016/j.jsat.2012.08.222.

[38] Denoth F, Siciliano V, Iozzo P, Fortunato L, Molinaro S. The association between overweight and illegal drug consumption in adolescents: is there an underlying influence of the sociocultural environment? PLoS One. 2011;6(11):e27358. doi:10.1371/journal.pone.0027358.

[39] Montigny C, Castellanos-Ryan N, Whelan R, Banaschewski T, Barker GJ, Buechel C, et al. A phenotypic structure and neural correlates of compulsive behaviors in adolescents. PLoS One. 2013;8(11):e80151. doi:10.1371/journal.pone.0080151.

[40] Martínez-González L, Fernández T, Molina AJ, Ayán C, Bueno A, Capelo R, et al. Prevalence of eating disorders in college students and associated factors: Unhicos project. Nutr Hospital. 2014;30:927-34. doi:10.3305/nh.2014.30.4.7689.

[41] Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale Food Addiction Scale. Appetite. 2009;52:430-36. doi:10.1016/j.appet.2008.12.003.

[42] Gearhardt AN, Roberto CA, Seamans MJ, Corbin WR, Brownell KD. Preliminary validation of the Yale Food Addiction Scale for children. Eat Behav. 2013;14(4):508-12. doi:10.1016/j.eatbeh.2013.07.002.

[43] Gearhardt AN, Boswell RG, White MA. The association of "food addiction" with disordered eating and body mass index. Eat Behav. 2014;15:427-433. doi:10.1016/j.eatbeh.2014.05.001.

[44] Granero R, Hilker I, Agüera Z, Jiménez-Murcia S, Sauchelli S, Islam MA et al. Food addiction in a Spanish sample of eating disorders: DSM-5 diagnostic subtype differentiation and validation data. Eur Eat Disord Rev. 2014;22:389-96. doi:10.1002/erv.2311.

[45] Gearhardt AN, White MA, Masheb RM, Grilo CM. An examination of food addiction in a racially diverse sample of obese patients with binge eating disorder in primary care settings. Comp Psychiatry. 2013;54:500-5. doi:10.1016/j.comppsych.2012.12.009.

[46] Davis C. Compulsive overeating as an addictive behavior: overlap between food addiction and binge eating disorder. Curr Obes Rep. 2013;2:171-8. doi:10.1007/s13679-013-0049-8.

[47] Davis C. A narrative review of binge eating and addictive behaviors: shared associations with seasonality and personality factors. Front Psychiatry. 2013;4:183. doi:10.3389/fpsyt.2013.00183.

[48] Pursey KM, Stanwell P, Gearhardt AN, Collins CE, Burrows TL. The prevalence of food addiction as assessed by the Yale Food Addiction Scale: a systematic review. Nutrients. 2014;6:4552-4590. doi:10.3390/nu6104552.

[49] Curtis C, Davis C. A qualitative study of binge eating and obesity from an addiction perspective. Eat Disord. 2014;22:19-32. doi:10.1080/10640266.2014.857515.

[50] Speranza M, Revah-Levy A, Giquel L, Loas G, Venisse J, Jeammet P, et al. An investigation of Goodman's addictive disorder criteria in eating disorders. Eur Eat Disord Rev. 2012;20:182-9. doi:10.1002/erv.1140.

[51] Michaelides M, Thanos PK, Volkow ND, Wang GJ. Translational neuroimaging in drug addiction and obesity. ILAR J. 2012;53:59-68. doi:10.1093/ilar.53.1.59.

[52] Brooks SJ, O'Daly O, Uher R, Friederich HC, Giampietro V, Brammer M, et al. Thinking about eating food activates visual cortex with reduced bilateral cerebellar activation in

females with anorexia nervosa: an fMRI study. PLoS One. 2012;7(3):e34000. doi:10.1371/journal.pone.0034000.

[53] Brooks SJ, O'Daly OG, Uher R, et al. Differential neural responses to food images in women with bulimia versus anorexia nervosa. Tsakiris M, ed. PLoS One. 2011;6:e22259. doi:10.1371/journal.pone.0022259.

[54] Gearhardt AN, Yokum S, Orr PT, Stice E, Corbin WR, Brownell KD. Neural correlates of food addiction. Arch Gen Psychiatry. 2011;68:808-16. doi:10.1001/archgenpsychiatry.2011.32.

[55] Filbey FM, Myers US, DeWitt S. Reward circuit function in high BMI individuals with compulsive overeating: similarities with addiction. Neuroimage. 2012;63:1800-6. doi:10.1016/j.neuroimage.2012.08.073.

[56] Tomasi D, Volkow ND. Striatocortical pathway dysfunction in addiction and obesity: differences and similarities. Crit Rev Biochem Mol Biol. 2013;48:1-19. doi:10.3109/10409238.2012.735642

[57] Wang GJ, Geliebter A, Volkow ND, Telang FW, Logan J, Jayne MC, et al. Enhanced striatal dopamine release during food stimulation in binge eating disorder. Obesity. 2011;19:1601-8. doi:10.1038/oby.2011.27.

[58] Broft A, Shingleton R, Kaufman J, Liu F, Kumar D, Slifstein M, et al. Striatal dopamine in bulimia nervosa: a PET imaging study. Int J Eat Disord. 2012;4:648-56. doi:10.1002/eat.20984.