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Emotional Eating and Obesity

Ignacio Jáuregui-Lobera and Marian Montes-Martínez

Abstract

The first time that terms such as food addiction and addictive eating were mentioned was in 1956, in an article by T.G. Randolph. Recently, from a psychosomatic point of view, some authors have linked obesity and food addiction. Along with the concept of food addiction (derived from the similarities between the consumption of certain foods and “substance addictions”), a couple of questions seem to arise: What if it’s not just the particular food (the substance) that we are addicted to? Could it be that we are addicted to something else that makes us eat it? Thus, the concept of eating addiction has its own set of particulars. It brings the attention back to the individual and not the external substance (the food or ingredient). The focus on confronting the obesity problem should be moved away from the food itself (the addictive substance) to the person’s act of eating (the addictive behavior). Undoubtedly, there are many links between emotions and overweight/obesity. This chapter aims to review the current state of this field of study which is the emotional basis of obesity (at least a particular case of obesity and weight-related disorders).

Keywords: food addiction, eating addiction, emotional eating, stress, negative emotions, posttraumatic stress disorder, overweight, obesity

1. Introduction

Some time ago, in a wonderful article by Adriaanse et al. [1], a question was suggested, “Emotional eating: Eating when emotional or emotional about eating?” Due the increasing number of people who are overweight and the increase in the worldwide prevalence of obesity over the past decades [2], some etiological factors have been proposed. Summarizing, both environmental and personal factors seem to be involved. With respect to the first, a “toxic food environment” was mentioned by Wadden et al. [3], and other authors have thought that tempting palatable foods available everywhere might be a relevant factor to explain the epidemic figures of overweight [4]. Along with food, personal factors must be considered. How does a person respond individually to food? Are there different possible responses depending on specific foods? Are there individuals prone to develop food addiction or addictive eating?

The first time that terms such as food addiction and addictive eating were mentioned was in 1956, in an article by T.G. Randolph [2]. Recently, from a psychosomatic point of view, some authors have linked obesity and food addiction [5, 6]. Along with the concept of “food addiction” (derived from the similarities between the consumption of certain foods and “substance addictions”), a couple of questions seem to arise: What if it is not just the particular food (the substance) that we are addicted to? Could it be that we are addicted to something else that makes us eat it? Thus, the concept of eating addiction has its own set of peculiarities. It brings the

attention back to the individual and not the external substance (the food or ingredient). The focus on confronting the obesity problem should be moved away from the food itself (the addictive substance) to the person's act of eating (the addictive behavior). Undoubtedly, there are many links between emotions and overweight/obesity.

It is well known that calorie-restricted diets are clearly ineffective for patients with overweight beyond the short term. In the long term, the most amount of weight lost is usually regained, with some patients even ending up weighing more than before the diet [7–9]. It is usual to think about emotional eating just linked to the abandon of restricted diets. Nevertheless, emotional eating may also occur, independently or regardless of dieting. Several authors have referred to emotional eating as an outcome of poor interoceptive awareness, a confusion of internal states of hunger and satiety and physiological symptoms associated with emotions, alexithymia, or poor emotion regulation strategies [9, 10]. Emotional eating has also been associated with a reversed stress response of the hypothalamic pituitary adrenal (HPA) axis (a blunted instead of the typical elevated cortisol response to stress) [11, 12].

2. When does emotional eating appear?

The prevalence of emotional eating in childhood is usually very low. In this regard, what tends to occur when food intake is linked to negative emotions/stress in children? The usual, natural response is that they tend to lose appetite. Then, in the transition from childhood to adulthood, emotional eating emerges in the form of overeating. Puberty (with its hormonal changes) would be the base for this phenomenon in adolescence [9]. As we referred, a common explanation for the increase in obesity over recent decades is the environment and, in particular, the availability of highly varied, palatable, and fattening foods—which have been considered to be addictive [13–16]. The point is that many individuals manage to resist these temptations and maintain a healthy weight, but others (e.g., overweight and obese individuals) have been shown to have preference for energy-dense foods compared to healthy-weight people [17–19]. In sum, not all children would become emotional eaters during adolescence.

Some research has shown that when food is eaten to satisfy one's feelings instead of satisfying hunger, it might result in emotional eating, which increases the risk of obesity. The study of emotional eating is complex because it is influenced by several risk factors, and some of these factors have been studied from a psychological point of view, such as self-regulation, effects of stress on eating behavior, parenting and emotional eating, and parental bonding and coping.

Considering self-regulation, emotional eating, as a learned response, is suggested to be associated with depressive feelings and inadequate parenting in adolescence [20], and it seems that self-regulation plays an important role in this respect. Galloway et al. [21] investigated the association between feeding practices used in childhood, eating behaviors, and weight status in early adulthood, and they found a significant positive correlation between practices based on controlling child feeding, emotional eating, and body mass index among children. Moreover, controlling child feeding practices are linked with poorer self-regulation of food intake [22]. On the one hand, the probability of eating in the absence of hunger is increased by restrictive feeding practices [23]. On the other hand, children's natural ability to self-regulate would be interfered by eating under pressure [24].

Although research shows that children tend to have poorer self-regulation because of immaturity of the brain [25], proper parenting such as being a positive role model [26] is a crucial factor to consider a successful self-regulation.

The effects of stress on eating behavior might be summarized, highlighting that the response to stressful circumstances is usually loss of appetite. Therefore, emotional eating would be an atypical response to this factor [27]. Besides, it can influence preference for sweet and fatty foods, among other unhealthy foods [11]. Studies have found that stressful circumstances such as examinations or times of high workloads are associated with greater energy and fat intake [28], so that emotion regulation through eating is experienced in a student population during stress under real life conditions with distraction as a possible mediating mechanism [29].

Other fields of study are the parenting styles and their influence on eating. In this regard, studies have found that authoritative parenting style is associated with higher levels of emotional eating in children and adolescents [30] as well as less maternal support, more maternal psychological control, and less maternal behavioral control [31], which is, on the other hand, associated with alexithymia [32]. Emotional awareness among obese children has been associated with other parenting styles such as over-protection and, in turn, emotional awareness with emotional eating [33]. In addition, the ability to cope with challenging situations, which determines children's well-being and success in college, seems to be positively associated with parental bonding [34]. Besides, the use of problem solving, active distraction, social-support seeking, and less passive resignation of failure has been linked with maternal bonding [35].

3. Food addiction, eating addiction, and emotional eating

If we focus on specific foods, the person's act of eating, and emotions linked to eating, it seems that two aspects of self-regulatory failure that are particularly pertinent in both substance use and overeating are impulsivity and reward sensitivity [36–38]. With respect to impulsivity (a multifaceted construct, it can be defined broadly as “the tendency to think and act without sufficient forethought, which often results in behavior that is discordant with one's long-term goals”) [39], it has been implicated in overeating and obesity [40–43]. Regarding reward sensitivity, a heightened general sensitivity to reward has also been linked to overeating [44–46]. Nevertheless, the causal direction between reward sensitivity and overeating remains uncertain. On the one hand, increasing reward sensitivity may lead to overeating by increasing motivation toward pleasurable activities, such as consuming energy-dense foods that elicit dopamine and opioid activation. On the other hand, decreased reward sensitivity may cause individuals to seek out rewarding activities as a form of “self-medication” in order to boost dopamine functioning (i.e., addictive behavior would be the result of a “reward deficiency syndrome”) [47, 48].

It seems that we face two possibilities: (a) an increased reward sensitivity might lead to overeating (which will produce an activation of dopamine and opioids) via increased motivation to obtain gratification; and (b) a decreased reward sensitivity might push individuals to seek “something” capable to stimulate dopamine functioning. Burger and Stice have proposed several theories about the way these two causal directions would combine to explain obesity [49]. Thus, high sensitivity to reward might initially cause individuals to over-consume palatable foods. Nevertheless, this sensitivity would be modified over time as the brain's reward system adapts and shows divergent changes in food motivation (“wanting”) versus hedonic pleasure (“liking”). The point is that with repeated exposures to palatable foods, the hedonic pleasure derived from the ingestion would decrease due to neural habituation, while the anticipation of reward would increase. As a result, the individual is experiencing less pleasure from the food (“liking”), but simultaneously he/she experiences an increased desire (“wanting”) for the food, driving further food seeking and consumption [50–55].

Impulsivity, reward sensitivity, and the experience of intense craving (the intense desire to consume a specific food) [56, 57] would be the three facets of food addiction in the field of overeating, overweight, and obesity. Chocolate, carbohydrates, and salty snack are the most commonly craved foods [58–62]. Studies on cue-reactivity research have repeatedly shown similarities between drug and food craving. In both cases craving is more likely to occur in the presence of substance-related stimuli. Thus, substance cues or food cues tend to increase the craving [63].

4. Between personal risk factors and overweight/obesity: emotional eating as mediator?

One might emphasize dispositional factors (biological or psychological), which would lead to overweight or obesity, or focus the attention on food properties (to some extent “addictive”) or the mere fact of eating (eating behavior, eating addiction). Alternatively, the mediation of emotions might be considered (emotional eating).

The tendency to eat in response to negative emotions or stress is an atypical stress response, as the typical stress response consists of not eating because the physiological stress reactions mimic the internal sensations associated with feeding-induced satiety [27] (see for empirical support [64]). Emotional eating, as “disinhibitor,” requires prior inhibition (i.e., restraint) by definition. However, it has not yet been resolved whether restraint eating is a cause of the consequence of emotional eating [65, 66], and this may also differ in various subgroups [67]. Nevertheless, as it was mentioned above, emotional eating may also occur, independently of food restrictions. We noted that emotional eating tends to co-occur with external eating (i.e., overeating in response to food-related cues such as the sight and smell of attractive food) [68]. In addition, Slochower [69] reported that negative emotions and food cues were shown to operate conjointly to elicit overeating in female students with obesity—the participants only overate in the high anxiety-high food salience condition, but not when the anxiety and/or the food salience was low.

With respect to “negative emotions,” feeling depressed is normally associated with loss of appetite and subsequent weight loss. There exists, however, a subtype of depression that is characterized by the atypical features of increased appetite and subsequent weight gain [70]. Emotional eating has been considered a marker of this depression subtype [71] because it shares with this subtype the atypical feature of increased appetite in response to distress such as feelings of depression (for support, see [72]). In various cross-sectional studies, emotional eating was indeed found to act as a mediator between depression and obesity [73–76].

Generally speaking, life adverse experiences are defined as all kinds of traumatic experiences occurring in childhood, adolescence, and adulthood, which include emotional abuse, physical abuse, sexual abuse, sexual harassment, rape, bullying by peers, witnessing domestic violence, and serious accidents that threatened the lives of subjects. As an example of traumatic experiences, abuse-related PTSD symptoms are associated with hyperactivation of HPA axis and with subsequent increases in peripheral cortisol, which in turn have been linked to accumulation of fat in adipose tissues and, consequently, an increase in abdominal obesity [77, 78]. In line with these findings, the hyperactivation of HPA axis with an exaggerated cortisol response to stress has been observed in obese patients [79] and was also put in relation with stress-induced eating [80], with night eating syndrome (NES) [81] and with waist adiposity in binge eating disorder (BED) patients [82].

Stress, depression, life adverse experiences, abuse-related PTSD, etc. might be potential risk factors for obesity via emotional eating. Some studies have focused their interest on the relationship between trauma, dissociation, and binge eating

disorder. Generally, it is concluded that dissociation may play an important mediating role between the presence of early trauma and the development of eating disorders (e.g., [83]). In this regard, it has been hypothesized that when negative emotional states are activated, a shift toward lower levels of cognition and self-awareness is initiated, which involves cognitive processes similar to dissociation. This mechanism tends to remove the inhibitions, thereby facilitating the start of binge eating or overeating, both in clinical (e.g., [84]) and in nonclinical subjects [85]. Several studies seem to support the hypothesis that dissociation may have a mediating role in the abuse and binge eating link [86, 87].

Along with dissociation, other authors have proposed some specific psychological variables that function as mediators in the relationship between childhood abuse, obesity, and bingeing, such as depression [88], trait anger [89], and perceived stress [90]. With respect to depression, Moyer et al. [88] even suggested that depression may be the only significant variable in the link between childhood abuse and adult obesity. Depression has been consistently associated with obesity and central obesity [91]. Once again, a possible way to interpret the link between childhood abuse, depression, and obesity is emotional eating. Regarding trait anger, (a) it seems to be related to the increase of visceral adipose tissue [92]; and (b) it is associated with emotional eating [93].

Considering adverse experiences, the following ideas seem to be relevant:

- Subjects with adverse childhood experiences have a higher risk of developing maladaptive coping strategies, including stress-induced emotional eating [94].
- Perceived stress may explain the link between child abuse and the development of obesity in adulthood [90].
- Activation of the stress response can lead to emotional dysregulation that has been associated with increased appetite, a preference for foods high in sugar and fat [11, 95–97], fat visceral accumulation, and obesity in adults [97–99] and adolescents [98, 99].
- Some authors have reported that overweight subjects tend to gain weight when stressed [11] and that obese individuals increase their food intake after having experienced negative emotions and perceived stress [100, 101].
- Laboratory studies have demonstrated that acute physical or emotional distress was followed by high cortisol reactivity, which induces increased intake of “comfort” foods [102–104].
- Stress-related adaptation involves the concept of allostasis, which is the ability to achieve the physiological balance through the change of the internal environment [96, 105, 106].
- Conditions of repeated or uncontrollable chronic stress are followed by higher cortisol response and tend to activate a state of allostatic load, resulting in neural and emotional dysregulation, which contribute to maladaptive behaviors such as repeated consumption of high caloric food [96], lack of control over eating, and binge eating [82, 107, 108].

Overall these abovementioned results suggest that psychophysiological responses to stress may influence subsequently eating behavior and hence may also mediate between the trauma and eating disorder link.

5. Posttraumatic stress disorder (PTSD): a psychosomatic paradigm of emotional eating?

It is well known that PTSD is usually associated with significantly higher rates of substance use disorders, other comorbid psychiatric disorders, and a variety of self-destructive and impulsive behaviors, including suicide [109–111]. It has been suggested that the ingestion, and especially over-ingestion, of fatty or sugary energy sources may be just another strategy that traumatized individuals use to numb themselves from their unpleasant feeling states and memories [112]. Thus, certain foods might act just like other substances that alter brain chemistry and, hence, consciousness. As we mentioned above, Randolph first described the phenomenon of food addiction and linked it with addictive drinking in 1956 [2]. Since then, the notion that certain foods can act like other addicting substances in the brain (despite having other peripheral metabolic effects that substances of abuse do not necessarily have) has been accepted. In fact, food intake and drug use both cause dopamine release in parts of the brain that mediate pleasure and emotion. The degree of subjective reward or experience of pleasure is clearly linked with the amount of dopamine release. Comparing similarities between action of certain foods and other substances of abuse, it must be noted that (a) food can stimulate the opiate system and there are similarities in use and withdrawal patterns of sugar and of classic drugs of abuse; (b) similar patterns of brain activation occur in response to food and drug cues; and (c) people may gain weight when they stop smoking or drinking.

It has been proposed that certain foods can be addicting to certain people, especially traumatized people. Part of the people exposed to alcohol, nicotine, drugs of abuse, etc. are prone to be attracted for these substances/behaviors, and finally they are at high risk to develop addictions. The point is how can we determine if someone will go on to develop an addiction to food or to any substance or behavior? From a genetic perspective, it has been proven that people with reduced dopamine type 2 receptor availability have a predisposition toward obesity and substance dependence. Other risk factors are environmental. In this regard, a history of psycho-trauma would be an example and leads to the self-medication hypothesis of PTSD. This way, victims of interpersonal violence may select highly palatable foods containing high concentrations of sugar, fat, salt, or caffeine, sometimes to the point of addiction, in an attempt to dampen arousal and facilitate numbing and avoidance specific symptoms to PTSD [112].

Hirth et al. [113] observed an association between PTSD symptoms and drinking more than one serving of soda per day as well as consumption of fast food more often. The participants of this study may have eaten more fast food to reduce

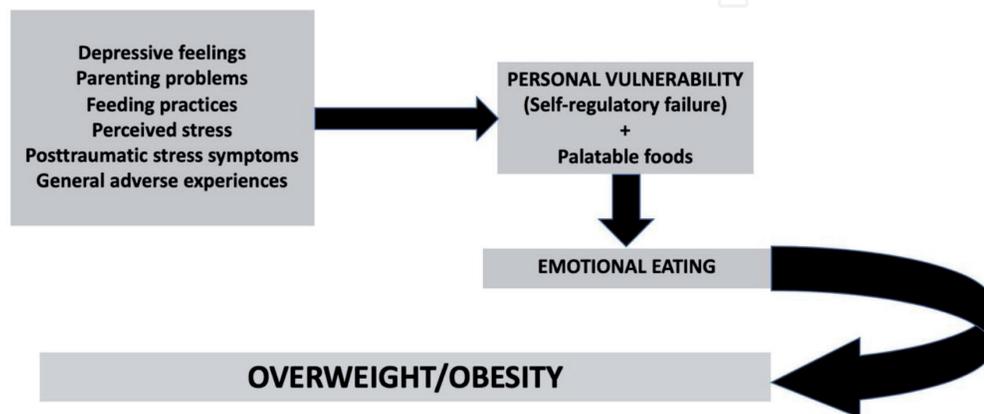


Figure 1.
Negative emotions, emotional eating, and weight gain.

trauma-induced PTSD symptoms, using that food as self-medication. Both fast food consumption and sugary soda consumption are usually associated with weight gain. PTSD symptoms may initiate a process of overindulging in unhealthy food and beverages in an attempt to compensate for the way trauma-induced memories make trauma victims feel. Through this process overeating behavior would lead to overweight/obesity. The problem would get worse when patients with PTSD symptoms try to reduce the effects of bingeing with unhealthy dieting behaviors and possibly develop eating pathology, consistent with the model of Stice and Shaw [114]. The results reported by Hirth et al. are consistent with theories that unhealthy dieting behaviors, such as vomiting and laxative abuse, are linked to PTSD [115, 116]. In sum, PTSD symptoms are associated with specific food and drink choices, and PTSD symptoms are also associated with unhealthy eating behaviors, which would be the gate to develop overweight/obesity and eating disorders. **Figure 1** tries to summarize the model which links negative emotions with weight gain.

6. Conclusions

In accordance with our review, we found that in spite of the fact that there are many risk factors involved in the increase in prevalence of obesity all around the world over the past few years, from those related to environmental and personal factors, particularly emotional eating plays a crucial and complex role in it. In the meantime, emotional eating is as well influenced by several risk factors: from social and physical environment to genetics, psychology, and food preferences. In addition, concepts such as food addiction and addictive eating were profoundly analyzed in order to explain the person's behavior toward food, and we concluded that there are many links between emotions and overweight/obesity. From our exploration we concluded that the epidemic of overweight and obesity is not only a matter of palatable and addictive foods available everywhere but also the individual responses to food.

Undoubtedly, we explored that emotional eating emerges in response to negative emotions, but it was also important to examine how self-regulation, effects of stress, parenting, and parental bonding and coping would have an effect on the act of eating and subsequently on emotional eating and its correlation with the body mass index. Although it is well known that the typical response of stress on eating behavior is usually loss of appetite, we found that stressful circumstances are associated with greater energy and fat intake.

Considering the field of study focused on overeating, it was seen that impulsivity, reward sensitivity, and the experience of intense craving result from self-regulation failures regarding both substance use and overeating. Nevertheless, some more research is needed in order to prove the causal direction between reward sensitivity and overeating.

Another important factor to consider in order to self-regulate successfully among children is the proper parenting styles, which would influence positively on eating behavior. Authoritative parenting style, less maternal support, the lack of parental bond, and overprotection would disrupt the well-being of children and the ability to cope with challenging situations. Subsequently, these factors would interfere with their attitudes toward the act of eating.

On the other hand, regarding negative emotions and feeling depressed, it is known that they are associated with loss of appetite and, as a result, weight loss. However, research showed that emotional eating would act as a mediator for a specific subtype of depression and it would have just the opposite effect on eating behavior, increasing weight as a result. In this regard, life adverse experiences,

childhood trauma, and abuse-related PTSD, among other traumatic and stressful situations, were associated with dissociation, which plays an important role in the development of eating disorders, emotional eating, and obesity. In the field of over-ingestion of specific kinds of foods, such as sugary and fatty sources, these foods were proposed to be addicting to traumatized people. The genetic hypothesis proved the link between reduced dopamine type 2 receptor availability and the predisposition toward obesity and substance dependence. On the other hand, the PTSD hypothesis showed the over-ingestion of palatable foods in traumatized people derives from an attempt to lessen arousal and to avoid specific symptoms derived from PTSD, that is, emotional eating as self-medication.

In conclusion, in order to face the obesity problem, the addictive behavior should be the focus of research and treatment and not the addictive substance as traditionally has been considered.

Conflict of interest

The authors declare no conflict of interest.

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