Examination of Individual Differences in Susceptibility to Food Addiction using Alcohol and Addiction Research Domain Criteria (AARDoC): Recent Findings and Directions for the Future

Monika M. Stojek1,2 · Cara M. Murphy3

Accepted: 2 August 2022 / Published online: 30 August 2022 © The Author(s) 2022

Abstract

Purpose of Review Alcohol and Addiction Research Domain Criteria (AARDoC) is a transdiagnostic, circuits-based framework for studying addictive behaviors. We examined parallels in individual differences that might increase susceptibility to FA and other addictive disorders using the following units of analysis in AARDoC domains: craving, relative reinforcing value of food and attention bias in the incentive salience domain; decisional impulsivity (delay discounting) and inhibitory control (Go-No-Go, Conner’s Continuous Performance Test, and the flanker task) in the executive function domain; and emotion dysregulation and negative urgency in the negative emotionality domain.

Recent Findings There are a number of parallels between FA and other addictions in the incentive salience and negative emotionality domains, but somewhat divergent findings in the executive function domain. Trauma appears to be an important environmental stressor in maintenance of FA.

Summary AARDoC may be a useful organizing framework for studying addictions, including FA. Future studies should incorporate other units of analysis to better characterize FA.

Keywords Food addiction · Research domain criteria · Incentive salience · Impulsivity · Trauma · Negative affect

Introduction

Over the past decade, the addictive nature of ultra-processed foods has been studied using the Yale Food Addiction Scale [1, 2]—a validated measure that has become standard for assessing the presence and severity of food addiction (FA). Ultra-processed foods have been defined as foods with five or more ingredients that include substances not commonly used in culinary preparations [3]. These foods are proposed to have the most addictive potential due to their pharmacokinetic properties, similar to the properties of drugs of abuse (e.g., rapid rate of absorption) [4]. While FA has been a useful research construct in understanding maladaptive eating patterns contributing to obesity, it is not a diagnosis recognized in the DSM-5 [5] nor are there any empirically supported treatment protocols specifically to address FA. However, given the advent of transdiagnostic approaches to psychological interventions, it may be more important to focus on tailoring interventions to specific characteristics of FA (some of which overlap with symptoms of eating disorders) than to the diagnosis itself. Such approaches may facilitate the development of more personalized interventions that are, nevertheless, grounded in empirical findings. We propose using elements from the Research Domain Criteria (RDoC) framework [6], and, more specifically, from the Alcohol and Addiction Research Domain Criteria (AARDoC) model [7, 8], to briefly review the current state of knowledge on the neurobiologically based individual differences underlying FA and other addictive disorders, and to assess the similarities and differences in those characteristics between FA and other addictive disorders. Based on these findings, we also outline directions for future research.
The RDoC framework was proposed by the National Institutes of Mental Health [9] as an alternative to diagnosis-based approaches to characterizing psychopathology. RDoC proposes several empirically based domains subsuming various constructs that can be measured using “units of analysis” at different levels (e.g., behavioral, circuit-level, genetic). The RDoC approach is geared toward discerning the mechanisms of psychopathology based on the current neurobiological findings (https://www.nimh.nih.gov/research/research-funded-by-nimh/rdoc). The five RDoC domains include: negative valence, positive valence, cognitive systems, systems for social processes, and arousal/modulatory systems [9]. In the alcohol field, an addiction-specific RDoC model and assessment framework have been proposed as deeming further study [7, 10]. The AARDoC model [7] focuses on three domains of risk factors for addiction: incentive salience (the equivalent of positive valence), executive function (the equivalent of cognitive systems), and negative emotionality (the equivalent of negative valence). These domains are based on neurobiological models of addiction that emphasize the dysfunction of the reward and stress systems in addiction, and correspond to distinct phases of the “addiction cycle”: incentive salience to the binge/intoxication phase, negative emotionality to the withdrawal/negative affect phase, and executive function to the preoccupation/anticipation phase [11, 12]. The AARDoC model also acknowledges the influence of environmental factors, such as stress exposure. Kwako and others [10] have proposed a battery of assessments falling under the three AARDoC domains (see Table 2 in [10], as a starting point for circuit-based assessment of addictions as disorders of impulsivity and compulsivity.

In this article, we will “try on” the AARDoC framework to the construct of FA in order to examine the parallels and potential differences between processes present in FA and alcohol use disorder. We have identified several units of analysis to review in the context of FA within the AARDoC domains (see Table 1). Therefore, we will use the AARDoC framework to examine individual differences in neurobiologically based mechanisms underlying FA and other addictive disorders (using most recent findings on FA), while also acknowledging that we will not review all units of analysis (partially because not all of them have been used in the context of FA, and partially because we wanted to focus on the most salient ones). We will also discuss how these domains may interact with the environmental factor of trauma.

### Incentive Salience

#### Background

Incentive sensitization models have been adapted to the eating dysregulation field from addiction studies [13], and they posit that cues of the addictive substance capture attention and trigger food cravings more readily if they carry a heightened incentive value. Incentive salience refers to a learning process in which previously neutral cues become imbued with meaning (i.e., salience), making them “wanted”. There is extensive evidence indicating not just heightened incentive salience in individuals with alcohol use disorders but also associations between indicators of incentive salience (e.g., craving) and worse outcomes [13–15]. Incentive salience has been studied to a lesser degree in FA. In this review, we will concentrate on subjectively reported craving, relative reinforcing value of food, and attention bias to food as units of analysis for the incentive salience domain.

#### Craving

Much of the support for FA highlights the fact that there are many similarities between cravings (i.e., a very strong or overwhelming desire) to consume food and cravings to use substances of abuse. Craving is thought to be a hallmark feature of addiction and was added as a diagnostic symptom of alcohol and substance use disorders in DSM-5 [5]. It is believed to play an important role in substance use and relapse after a period of abstinence, with individuals who experience greater cravings also being at higher risk of relapsing [16]. Many have theorized that addictive substances contribute to neuroadaptations in the reward system—hijack the brain’s natural reward system, so to speak—and that food cravings operate similarly to cravings that occur in addiction to psychoactive substances [17–20]. On the behavioral level, craving represents associative learning:

| Table 1 Domains of interest and associated units of analysis |
|---------------------------------------------------------------|
| **Incentive Salience** | **Executive Function** | **Negative Emotionality** | **Environmental factors** |
| Subjective rating of craving | Delay discounting | Emotion dysregulation | Trauma |
| Relative reinforcing value of food | Go/No-Go task | | |
| Attention bias to food | The Conners’ Continuous Performance | Negative urgency | |
| | The Eriksen flanker task | | |
When considering FA, individuals who self-identify as “chocolate addicts” experience more craving and negative affect when presented with chocolate cues relative to control participants [21]. Meeting the FA status on the YFAS is also associated with higher food craving [22, 23]. Interestingly, although individuals with FA experience more food cravings, they do not expect to achieve positive reinforcement from eating [24]. Craving is also a mediator between genetic vulnerability toward food reward sensitivity (expressed by the multilocus genetic profile score [MLGP]) and FA [25]. Reward sensitivity refers to an individual’s tendency to seek highly rewarding things (e.g., experiences, foods, substances) [26]. In the context of FA, individuals with higher reward sensitivity may be more sensitive to the rewarding properties of food, particularly highly palatable and highly caloric ones. Indeed, symptoms of FA correlate with anticipated food reward based on activation in the brain’s reward circuits [27]. Accordingly, in FA, just as in substance use disorders, craving for the reinforcing aspects of high-calorie foods and substances, respectively, are important factors in seeking out a substance and motivating consumption, contributing to an individual’s progression from an inherent biological susceptibility toward rewards to compulsive food seeking characteristic of addictive behaviors. Thus, craving is an individual difference that may increase susceptibility to negative outcomes related to FA as it does in other addictive behaviors. Therefore, it may be a useful unit of analysis, particularly when considering treatment outcomes in FA.

**Relative Reinforcing Value of Food**

People are willing to work for reinforcers that they want up to a certain point. Determining how far someone will go before they decide the reinforcer is not worth the effort demonstrates the reinforcing value of that item. Relative reinforcing value compares two available reinforcers (e.g., two types of food or food and an alternate reinforcer) [28]. Food reinforcement can also be measured using demand curves that demonstrate how much of a reinforcer they would purchase as a function of its price [29]. The demand curves are generated based on purchase tasks, and several indices of demand are produced to evaluate food reinforcement. Higher demand for addictive substances is associated with increased use of that substance and greater problem severity [30, 31]. Similarly, finding food highly reinforcing is associated with higher body mass index (BMI) [32, 33]. Relative to those who do not meet FA criteria, individuals with FA report greater reinforcement value based on nearly all demand indices for sweet and salty snacks [34]. Dopamine receptor gene (DRD2) and its relationship to addictive behavior such as opioid use disorder have also been studied in the context of addiction [35], and one recent study demonstrated that while FA was not associated with the DRD2 polymorphism, women with obesity who were carriers of a particular allele had higher snack food reinforcement relative to non-carriers [36]. Another study that assessed FA and reward-related eating found that while FA was not associated with diet quality during pregnancy or postpartum, greater reward-related eating was associated with reduced diet quality during pregnancy [37]. Therefore, relative reinforcing value of food should be examined more systematically as a promising unit for analysis of FA symptoms.

**Attention Bias to Food**

Attention bias is the tendency to attend selectively to stimuli that have acquired salience or meaning [38], and a process that has been shown to contribute to substance misuse [39, 40]. There is also evidence that attention bias to food is stronger in individuals with binge eating behaviors compared to those without [41]. Even though binge eating and FA often co-occur [42], there are only two studies to date that examined attention bias in FA [43, 44], and only one of these used the YFAS to assess the presence of FA [43]. In the first study, women with and without FA underwent both, a neutral and a sad mood induction prior to the attention bias task, and their reactions on the task were measured using eye-tracking. The study found that before the mood induction, participants with FA attended to food images significantly more than those without FA and that they had difficulty with disengaging from images of unhealthy foods, compared to those without FA (but that unhealthy food images initially captured the attention of both groups equally). This may suggest a general hypervigilance to food cues in the FA group, coupled with difficulties with redirecting attention away from the unhealthy food cues. Following the sad mood induction, participants with FA increased their sustained attention to unhealthy foods and decreased their sustained attention to healthy foods, suggesting an emotion regulation function of unhealthy foods in that group. For participants without FA, sad mood induction had no significant effect on sustained attention to healthy or unhealthy food cues. This suggests that individuals without FA may primarily regulate their emotions with ways other than food. In another study, no effect of FA diagnosis was found on attention bias to pictures of chocolate, such that participants in the control condition had similar reaction times to individuals with FA [44]. However, in that study, participants self-identified their FA and the YFAS was not used to verify FA status. Given all findings in the attention bias literature, it appears premature to draw conclusions regarding increased attention bias to food in individuals with FA. More studies
are warranted using attention bias paradigms due to their potential to measure incentive salience using non-self-report measures.

**Executive Function**

**Background**

Executive function can be measured in several ways and includes cognitive control and decision making, key facets of self-regulation (i.e., self-control) [45]. Impulsivity and self-regulation, which typically refer to one’s capacity to regulate their impulses and desires, have shown strong associations with addictive disorders and behavior [46–48]. Executive function has been investigated in the context of FA as well. For instance, among recommended treatment strategies for FA is targeting four core features: craving, impulsivity, compulsivity, and motivation, this includes the recommendation to target impulsivity as a personality trait [49]. As impulsivity has several facets, including the trait-based personality facet (covered in Sect. 4.3.), we will concentrate on decisional (delay discounting) and behavioral (difficulties in inhibitory control) impulsivity [50] as manifestations of executive dysfunction.

**Delay Discounting**

One area of executive dysfunction is impulsive choice. This reflects a tendency to make impulsive choices that do not support one’s long-term interests. This type of difficulty with self-control is known as discounting of delayed rewards (i.e., one’s tendency to choose less valuable rewards that are available now over more valuable rewards that could be received after a delay [51]. If an individual decides to select a smaller reward on the basis of its immediate receipt, foregoing a larger reward that would have been available later, this is thought to demonstrate difficulties with decision making and behavioral control. A small number of studies have examined the relationship between FA and executive dysfunction using measures of impulsive choice. These studies have generally suggested a small relationship between delay discounting and FA [22, 52–55]. In addition, a recent meta-analysis suggested aggregate correlations between FA and steeper discounting were significant but small in magnitude ($r = 0.12$) [56]. This is in contrast to a meta-analysis that concluded a robust association between delayed reward discounting across many addictive substances [46]. As relatively few studies have been conducted regarding impulsive choice and FA, more research is needed to determine whether delay discounting may be a transdiagnostic unit of analysis for FA symptomatology.

**Inhibitory Control**

Another area of executive dysfunction is inhibitory control. This refers to the ability to inhibit impulse choices when needed. The inability to inhibit specific behaviors, including the use of a substance itself, is considered a key component in both addictions to alcohol/substances as well as in behavioral addictions (e.g., video games, the internet) with abnormalities in the prefrontal cortex thought to play a role in the loss of behavioral control that often occurs [57]. This form of impulsivity, often thought of in the context of “inhibition” and “disinhibition,” relates to active and willful processes of cognitive control during which the prefrontal cortex must enact control over a particular response [58]. Several studies have measured cognitive control and impulsive action using tasks such as a Go/No-Go task [59]. While considerable variation between task stimuli exists, the general premise in this type of task is to press a button for certain stimulus but inhibit pressing for others. Failing to react (i.e., press the button) at the target stimulus (i.e., an omission error) is thought to reflect inattention, whereas failing to inhibit pressing the button when the indicated stimulus is shown (i.e., a commission error) is thought to reflect difficulty with inhibitory control. These studies have generally failed to find a relationship between FA and inhibitory control/failed inhibition [53, 55, 60–63]. Another similar task, The Conners’ Continuous Performance Task [64], similarly requires pressing a key when a stimulus is present and inhibiting pressing when absent. One study comparing individuals with obesity with and without FA failed to find differences between the groups on commission errors when using this task [65]. Another inhibitory control task, the Eriksen flanker task [66], requires individuals to press a key with their left index finger and if the central letter in a series of letters presented is one letter (e.g., “S”) and with their right index finger if it is a different letter (e.g., “H”). On this task, individuals with FA were found to make more errors overall in one research study [67]. Past research of individuals with addictive disorders has shown the individuals who smoke cigarettes made more errors on incongruent tasks than individuals who did not smoke cigarettes [66]. Thus, the preponderance of available evidence to date does not suggest that individuals with FA show similar deficits on tasks of inhibitory control as is seen in other addictions.

**Negative Emotionality**

**Background**

The AARDoC domain of negative emotionality has been proposed to represent the withdrawal/negative affect phase of the “addiction cycle” [7]. This constitutes the presence
of physiological or psychological symptoms in response to substance deprivation or in order to relieve these symptoms [5]. Negative affect regulation is one of the main processes proposed to contribute to the transition from casual to compulsive substance use. The opponent-process theories hypothesize that substances of abuse at first activate the neurocircuitry associated with reward, thus producing the feelings of “high”, contentment, and well-being [12]. To downregulate the reward neurocircuitry, the opponent process involving the stress neurocircuitry follows, contributing to increases in negative affect, vigilance, and tension [68]. The negative reinforcement theory of addiction proposes that in addition to the withdrawal state eliciting negative affect coupled with craving, negative affective states (e.g., disappointment, anxiety, frustration) also become conditioned cues eliciting urges to use the substance [69]. In fact, this theory was reformulated into the affective processing theory of negative reinforcement, proposing that negative affect is the main motivational factor contributing to drug-seeking behavior [70]. With repeated use of a substance, individuals detect a negative affective state (conditioned stimulus) automatically (i.e., outside of awareness) and identify craving (conditioned response) that has been coupled with it but not necessarily the affective state [70]. Craving seems uncontrollable because it seemingly “comes out of nowhere.” Therefore, in this model, negative affect, and not physical withdrawal, is seen as the motivational core of substance misuse [71].

**Emotion Dysregulation**

In the alcohol use disorders field, the affect regulation model has been extensively supported with data [72–74], using various units of analysis. While there is evidence of affective processing theory in binge eating, both in experimental and ecological momentary assessment studies [75, 76], the evidence of this process strictly in FA has been studied to a lesser extent. However, there are numerous studies indicating an association between negative emotionality and food addiction. Therefore, we will review studies that utilized self-report measures of negative emotionality, including symptoms of anxiety, depression, and stress.

DASS-21 is a transdiagnostic self-report measure of negative emotionality [77], measuring levels of depression, anxiety, and stress. In a large Australian sample, high levels of depression measured by DASS-21 were associated with higher odds of having severe FA [78]. In a sample of individuals with type 2 diabetes mellitus, the level of depression, anxiety, and stress reported on DASS-21 increased with the severity of food addiction [79]. More broadly, a meta-analysis of comorbidity between FA and mental health disorders found significant correlations between FA and depression as well as FA and anxiety [80]. Among individuals seeking addictive eating treatment, those with higher DASS-21 scores were less likely to engage in treatment [81]. Overall, there is considerable evidence for the association between FA symptom severity and the severity of negative emotionality, although it is impossible to discern the temporal occurrence of these constructs as they relate to each other.

**Negative Urgency**

Several studies have examined self-reported impulsivity on the trait level. One measure commonly used for this is the UPPS Impulsive Behavior Scale (UPPS; [82]) that generates subscales including urgency (positive and negative), sensation seeking, lack of premeditation, and lack of perseverance [83, 84]. In the AUD literature, there is extensive evidence that increased level of urgency, and negative urgency in particular (i.e., a tendency to act rashly when experiencing negative emotions), are associated with problematic drinking and AUD symptoms [47, 85]. Research has suggested that negative urgency can impact problematic substance use for a variety of substances [86]. Similarly, across numerous studies, negative urgency has been shown to have a relationship with FA [54, 55, 63, 87–91]. Related, in one study of FA, negative urgency, emotional eating, and FA associations contributed to reduced quality of life [90]. Taken together, negative urgency appears strongly associated with FA, both directly and indirectly, consistent with findings in substance use disorders. Therefore, it may be a useful unit of analysis of the negative emotionality (negative valence) domain.

**Interactions of Negative Emotionality Domain with Other Domains**

It is important to recognize ways in which domains may interact with one another. Some examples include findings that negative affect increases incentive salience of high-calorie foods in individuals with FA [43] or that craving is not associated with anticipation of reward in individuals with FA [24], potentially reflecting the transition to compulsive food seeking marked by motivation to decrease negative affect [70]. In fact, some [71] have suggested that reactivity to food cues associated with increased activation in the amygdala is an important element of compulsive food intake and evidence for the opponent-process theory, whereby consumption becomes a strategy to regulate negative emotions. Such negative emotional states in turn may contribute to impulsive food-seeking behavior.

**Stress and Trauma as Environmental Factors**

In addition to aversive emotional states, stressful events in the environment (e.g., adverse childhood events, traumatic experiences) may promote behaviors consistent with
food addiction and contribute to neural adaptations in the stress-reward neurocircuitry that might increase susceptibility to FA in some individuals. The link between trauma and alcohol/substance use disorders has been extensively documented [92, 93]. Emerging body of literature is finding similar links between trauma and FA. A cross-sectional retrospective study found a positive relationship between childhood abuse (physical and sexual) and FA symptoms in women [94], and exposure to trauma earlier in life is associated with more FA symptoms [95]. Among Black women with type 2 diabetes, women with FA reported higher severity of childhood trauma and had higher insulin resistance [96]. FA also mediated the relationship between severity of childhood trauma and insulin resistance in that sample [96]. Exposure to at least one traumatic event and lifetime presence of at least one posttraumatic stress disorder (PTSD) symptom have been associated with higher prevalence of FA symptoms [95, 97]. In primarily male veteran samples, current and lifetime diagnoses of PTSD were associated with FA symptoms [98, 99]. In a clinical sample of men and women veterans, those with FA (18% of the overall sample) reported higher severity of PTSD and depression [100]. Overall, recent findings consistently indicate an overlap between experience of trauma and food addiction symptoms and suggest that men are as susceptible to FA in trauma-exposed samples as women (e.g., [98–100]).

It has been proposed that changes in the neuroendocrine system that develop as a result of traumatic experiences in some individuals are a vulnerability factor to experiencing negative metabolic outcomes [101]. As with alcohol or other psychoactive substances, palatable food’s soothing properties [102, 103] may serve as an emotion regulation (via negative reinforcement) strategy among trauma survivors trying to avoid trauma-related emotions, thoughts, and memories [104]. Over time, using food in such a manner becomes a habit consistent with the opponent-process theory [71] whereby negative affective states and PTSD symptoms become cues to seek highly palatable food. Overall, more research is needed on the mechanism by which trauma and PTSD symptoms operate in FA, as it may be a promising direction in treatment of certain subgroup of individuals with FA.

Conclusions

We used selected elements of the Alcohol and Addiction Research Domain Criteria to examine the similarities and differences in the individual characteristics of FA and other addictive disorders. Given that FA is not a DSM-5 diagnosis and that it remains a controversial construct [105, 106], the RDoC framework seems particularly useful and suited for further investigation of FA. While clinical studies have historically studied participants who meet certain diagnostic criteria (often with exclusion of comorbid conditions), the AARDoC approach allows for greater heterogeneity in recruited samples, while the units of analysis allow for multidimensional characterization of the studied construct.

Based on the literature in this narrative review, several transdiagnostic units of analysis appear to be particularly useful in either distinguishing those with FA from those without FA, or appear to be important moderators of processes present in food-seeking behavior. Within the incentive salience domain, self-reported craving may be an expression of sensitivity to reward. As such, it may be particularly useful to include in treatment outcome studies as a unit of analysis predicting outcomes in treatment and by extension, be a treatment target for those who report elevated levels of food craving. Relative reinforcing value of food also appears to be a promising unit of analysis, with the few studies that used it in the context of FA, indicating that on average it is associated with FA symptoms. Attention bias to food should be investigated further as the number of studies is too small to draw conclusions. It appears that investigating the interaction between attentional processes and emotional states may be useful in identifying the mechanisms by which exposure to highly palatable foods leads to consumption. Within the executive dysfunction domain, FA appears to be divergent from many other addictive disorders—while there are statistically significant associations between delay discounting and FA, they are generally small. Individuals with FA also do not appear to differ from controls on measures of inhibitory control. It is possible that the relationship between executive dysfunction and FA is more nuanced and moderated by third variables. It is also possible that the anticipation/preoccupation phase of the addictive cycle in FA is not best represented by measures of decisional and behavioral impulsivity. In the negative emotionality domain, there are strong parallels between FA and other addictive disorders, generally indicating an association between severity of FA and severity of different units of analysis of negative emotionality.

Limitations and strengths

This was not a systematic review and the AARDoC approach was grounded in the most used paradigms in the FA field (thus it was not exhaustive). No quantitative meta-analysis was performed; therefore, empirically based conclusions are more difficult to put forth. Moreover, the AARDoC framework is a recent model and it has not been validated in the addiction field; therefore, it is possible that with more research, that conceptualization of this organizing framework will evolve. However, one of the strengths of the RDoC approach is the transdiagnostic nature of the units of analysis (in most instances) allowing for a
characterization of the spectrum of a population. Another strength of this review is incorporating the role of trauma (as an environmental factor) into the framework (as environmental factors were a peripheral part of the AARDoC framework; see Witkiewitz, 2019 for a graphic). It appears that there are several parallels between substance use and FA in the relationship between their severity and trauma exposure.

Future Directions

Considering that the RDoC and AARDoC frameworks may be a helpful organizing principle for future considerations of FA as a clinical construct, future studies should consider incorporating other units of analysis within the RDoC framework. Few studies have considered physiological correlates of FA compared to individuals without FA (such as heart rate variability or skin conductance) and these may offer additional information based on objective measurement in the Arousal domain (subsumed under RDoC, but not AARDoC; [9]). It may also be informative to study the interaction between different domains. For instance, findings in other addictions as well as in binge eating indicate that individuals with high incentive salience combined with high delay discounting, a combination termed reinforcement pathology [107, 108], tend to have the worst outcomes—therefore, it would be important to test that interaction in individuals with FA as it may be a predictor of treatment success. As the RDoC framework’s premise is to identify subtypes of different presentations and pursue precision medicine, it may be useful to generate profiles of individuals based on units of analysis in different domain using person-centered approaches. For example, given the strong link between trauma exposure and FA, it would be helpful to understand whether there is a trauma-exposed subtype of FA and whether such subtype is associated with specific units of analysis in the RDoC framework. Longitudinal studies would also be of use, to ascertain the causality of the reported associations. Overall, the AARDoC framework should be further investigated and validated in the addiction field, including as it applies to FA.

Funding Prof. Monika Stojek receives funding from the National Science Center (Narodowe Centrum Nauki) for project 2021/41/B/HS6/04029 (Relationship between PTSD symptoms, eating behaviors and physical health: Inflammatory, cardiometabolic, and psychophysiological correlates).

Declarations

Conflicts of Interest The authors do not have existing conflict of interest.
continuous associations. Addiction. 2017;112(1):51–62. https://doi.org/10.1111/ADD.13535.

47. Coskunpinar A, Cyders MA. Impulsivity and substance-related attentional bias: A meta-analytic review. Drug Alcohol Depend. 2013;133(1):1–14. https://doi.org/10.1016/j.DRUGALCDEP.2013.05.008.

48. Stanford MS, Mathias CW, Dougherty DM, Lake SL, Anderson NE, Patton JH. Fifty years of the Barratt Impulsiveness Scale: An update and review. Personality Individ Differ. 2009;47(5):385–95. https://doi.org/10.1016/j.PAID.2009.04.008.

49. Vella SLC, Pati NB. A narrative review of potential treatment strategies for food addiction. Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity 2017. 2017;22(3):222(3), 387–393. https://doi.org/10.1007/S40519-017-0400-2.

50. de Wit H. Impulsivity as a determinant and consequence of drug use: a review of underlying processes. Addict Biol. 2009;14(1):22–31. https://doi.org/10.1111/j.1369-1600.2008.00129.x.

51. Ainslie G. Specious reward: A behavioral theory of impulsiveness and impulse control. Psychol Bull. 1975;82(4):463–96.

52. Kekic M, McCllelland J, Bartholdy S, Chamali R, Bartholdy S, Campbell IC, Kekic M, McClelland J, MacKillop J. Multidimensional elements of impulsivity as shared and unique risk factors for food addiction and alcohol misuse. Appetite. 2021;159: 105052. https://doi.org/10.1016/j.APPET.2021.105052.

53. Minhas M, Murphy CM, Balodis IM, Acuff SF, Buscemi J, Murphy JG, Mackillop J. Multidimensional elements of impulsivity as shared and unique risk factors for food addiction and alcohol misuse. Appetite. 2021;159: 105052. https://doi.org/10.1016/j.APPET.2020.105052.

54. Peng-Li D, Sørensen TA, Li Y, He Q. Systematically lower structural brain connectivity in individuals with elevated food addiction symptoms. Appetite. 2020;155: 104850. https://doi.org/10.1016/j.APPET.2020.104850.

55. VanderBroek-Stice L, Stojeck MK, Beach SRH, vanDellen MR, Mackillop J. Multidimensional assessment of impulsivity in relation to obesity and food addiction. Appetite. 2017;112. https://doi.org/10.1016/j.APPET.2017.01.009.

56. Weinszotk S, Brassard S, Balodis I, Martin LE, Amlung M. Delay Discounting in Established and Proposed Behavioral Addictions: A Systematic Review and Meta-Analysis. Front Behav Neurosci. 2021;15. https://doi.org/10.3389/FBNEH.2021.786358/FULL.

57. Luijten M, Machielsen MJW, Veltman DI, Hester R, de Haan L, Franken IHA. Systematic review of ERP and fMRI studies investigating inhibitory control and error processing in people with substance dependence and behavioural addictions. J Psychiatry Neurosci. 2014;39(3):149–69. https://doi.org/10.1503/JPN.130052.

58. Aron AR. The neural basis of inhibition in cognitive control. Neuroscientist. 2007;13(3):214–28. https://doi.org/10.1177/107358407299288.

59. Kiehl KA, Liddle PF, Hopfinger JB. Error processing and the rostral anterior cingulate: An event-related fMRI study. Psychophysiology. 2000;37(2):216–23. https://doi.org/10.1111/1469-8986.3720216.

60. Blume M, Schmidt R, Hilbert A. Executive Functioning in Obesity, Food Addiction, and Binge-Eating Disorder. Nutrients 2019. 2018:11(1):54. https://doi.org/10.3390/NU111010054.

61. Hsu JS, Wang PW, Ko CH, Hsieh TJ, Chen CY, Yen JY. Altered brain correlates of response inhibition and error processing in females with obesity and sweet food addiction: A functional magnetic imaging study. Obes Res Clin Pract. 2017;11(6):677–86. https://doi.org/10.1016/j.ORCP.2017.04.011.

62. Meule A, Lutz A, Vogele C, Kübler A. Women with elevated food addiction symptoms show accelerated reactions, but no impaired inhibitory control, in response to pictures of high-calorie food cues. Eat Behav. 2012;13(4):423–8. https://doi.org/10.1016/J.EATBEH.2012.08.001.

63. Minhas M, Murphy CM, Balodis IM, Samokhvalov AV, Mackillop J. Food addiction in a large community sample of Canadian adults: prevalence and relationship with obesity, body composition, quality of life and impulsivity. Addiction. 2021;116(10):2870–9. https://doi.org/10.1111/ADD.15446.

64. Conners CK. Conners’ Continuous Performance Test II User’s Manual. Multi Health Systems. 2000.

65. Steward T, Mestre-Bach G, Vintró-Alcaraz C, Lozano-Madrid M, Agüera Z., Fernández-Formoso JA, Granero R, Jiménez-Murcia S, Vílarrasa N, García-Ruiz-de-Gordejuela A, Veciana de las Heras M, Custal N, Virgili N, López-Urdiales R, Gearhardt AN, Menchón JM, Soriano-Mas C, Fernández-Aranda F. Food addiction and impaired executive functions in women with obesity. Eur Eat Disord Rev. 2018;26(6):574–84. https://doi.org/10.1002/ERV.2636.

66. Franken IHA, van Strien JW, Kuijpers I. Evidence for a deficit in the salience attribution to errors in smokers. Drug Alcohol Depend. 2010;106(2–3):181–5. https://doi.org/10.1016/j.DRUGALCDEP.2009.08.014.

67. Franken IHA, Njs IMT, Toes A, van der Veen FM. Food addiction is associated with impaired performance monitoring. Biol Psychol. 2018;131:49–53. https://doi.org/10.1016/j.BIOPS.YCHO.2016.07.005.

68. Koob GF, Bloom FE. Cellular and Molecular Mechanisms of Drug Dependence. Science. 1988;242(4879):715–23. https://doi.org/10.1126/SCIEN.CE.2903550.

69. Sherman JE, Morse E, Baker TB. Urges/craving to smoke: Preliminary results from withdrawing and continuing smokers. Adv Behav Res Ther. 1986;8(4):253–69. https://doi.org/10.1016/0146-6402(86)90008-1.

70. Baker TB, Piper ME, McCarthy DE, Majeskie MR, Fiore MC. Addiction Motivation Reformulated: An Affective Processing Model of Negative Reinforcement. Psychol Rev. 2004;111(1):33–51. https://doi.org/10.1037/0033-295X.111.1.33.

71. Zorrilla EP, Koob GF. The dark side of compulsive eating and food addiction. In: P. Cotton, V. Sabino, C. F. Moore, & G. F. Koob (Eds.), Compulsive Eating Behavior and Food Addiction: Emerging Pathological Constructs. 2019:115–192. Academic Press. https://doi.org/10.1016/B978-0-12-816207-1.00006-8.

72. George O, Koob GF, Vendruscolo LF. Negative reinforcement via motivational withdrawal is the driving force behind the transition to addiction. Psychopharmacology 2014. 2014;231:19, 231(19), 3911–3917. https://doi.org/10.1007/S00213-014-3623-1.

73. Koob GF, Buck CL, Cohen A, Edwards S, Park PE, Schlosburg JE, Schmeichel B, Vendruscolo LF, Wade CL, Whitleff TW, George O. Addiction as a stress surfeit disorder. Neuropharmacology. 2014;76(PART B), 370–382. https://doi.org/10.1016/J.NEUROPHARM.2013.05.024.

74. Sinha R. The role of stress in addiction relapse. Curr Psychiatry Rep. 2007;9:388–95.

75. Naish KL, Laliberte M, Mackillop J, Balodis IM, Joseph’, S., Hamilton, H., & Hamilton, C. Systematic review of the effects of acute stress in binge eating disorder. Eur J Neurosci. 2019;50(3):2415–29. https://doi.org/10.1111/EJIN.14110.

76. Schaefer LM, Smith KE, Anderson LM, Cao L, Crosby RD, Engel SG, Crow SJ, Peterson CB, Wonderlich SA. The Role of Affect in the Maintenance of Binge-Eating Disorder: Evidence From an Ecological Momentary Assessment Study. J Abnorm Psychol. 2020. https://doi.org/10.1037/ABN0000517.

77. Henry JD, Crawford JR. The short-form version of the Depression Anxiety Stress Scales (DASS-21): Construct validity and
normative data in a large non-clinical sample. Br J Clin Psychol. 2005;44(2):227–39. https://doi.org/10.1348/014466605X29657.

78. Burrows T, Hides L, Brown R, Dayas CV, Kay-Lambkin F. Differences in Dietary Preferences, Personality and Mental Health in Australian Adults with and without Food Addiction. Nutrients 2017. 2017;9(3):285. https://doi.org/10.3390/NU9030285

79. Raymond KL, Kannis-Dymand L, Lovell GP. A graduated food addiction classifications approach significantly differentiates depression, anxiety and stress among people with type 2 diabetes. Diabetes Res Clin Pract. 2017;132:95–101. https://doi.org/10.1016/J.DIABRES.2017.07.028.

80. Burrows, T, Kay-Lambkin, F., Pursey, K., Skinner, J., & Dayas, C. (2018). Food addiction and associations with mental health symptoms: a systematic review with meta-analysis. In Journal of Human Nutrition and Dietetics (Vol. 31, Issue 4, pp. 544–572). Blackwell Publishing Ltd. https://doi.org/10.1111/jhn.12532

81. Pursey KM, Collins R, Skinner J, Burrows TL. Characteristics of individuals seeking addictive eating treatment. Eat Weight Disord. 2021;26(8):779–86. https://doi.org/10.1007/S40519-021-01147-Y/TABLES/2.

82. Whiteside SP, Lynam DR, Miller JD, Reynolds SK. Validation of the UPPS impulsive behavior scale: A four-factor model of impulsivity. Eur J Pers. 2005;19:559–74.

83. Lynam D, Smith GT, Whiteside SA, Cyders MA. The UPPS-P: Assessing five personality pathways to impulsive behavior. 2006.

84. Cyders MA, Smith GT. Mood-based rash action and its components: Positive and negative urgency. Personality Individ Differ. 2007;43:839–50.

85. McCarty KN, Morris DH, Hatz LE, McCarthy DM. Differential Associations of UPPS-P Impulsivity Traits With Alcohol Problems. J Studies Alcoh Drugs. 2017;78(4):617–622. https://doi.org/10.15288/jصاد.2017.78.617

86. Kaiser AJ, Milich R, Lynam DR, Charnigo RJ. Negative Urgency, Distress Tolerance, and substance abuse among college students. Addict Behav. 2012;37(10):1075–83. https://doi.org/10.1016/J.appet.2017.09.026.

87. Murphy CM, Stojek MK, MacKillop J. Interrelationships among impulsive personality traits, food addiction, and Body Mass Index. Appetite. 2014;73:45–50. https://doi.org/10.1016/j.appet.2013.10.008.

88. Pivarnaras B, Conner BT. Impulsivity and emotion dysregulation as predictors of food addiction. Eat Behav. 2015;19:9–14. https://doi.org/10.1016/j.eatbeh.2015.06.007.

89. Rodrigue C, Gearhardt AN, Bégin C. Food Addiction in Adolescents: Exploration of psychological symptoms and executive functioning difficulties in a non-clinical sample. Appetite. 2019;141: 104303. https://doi.org/10.1016/J.appet.2019.05.034.

90. Rose MH, Nadler EP, Mackey ER. Impulse Control in Negative Mood States, Emotional Eating, and Food Addiction Are Associated with Lower Quality of Life in Adolescents with Severe Obesity. J Pediatr Psychol. 2018;43(4):443–51. https://doi.org/10.1093/JPPES/JXS127.

91. Wolz I, Hilker I, Granero R, Jiménez-Murcia S, Gearhardt AN, Dieguez C, Casanueva FF, Crujeiras AB, Menchón JM, Fernández-Aranda F. “Food Addiction” in Patients with Eating Disorders is Associated with Negative Urgency and Difficulties to Focus on Long-Term Goals. Frontiers in Psychology. 2016;7:61. https://doi.org/10.3389/FPSYG.2016.00061/BIBTEX

92. Brady KT, Back SE. Childhood Trauma, Posttraumatic Stress Disorder, and Alcohol Dependence. Alcohol Res Curr Rev. 2012;34(4):408. /pmc/articles/Pmc3860395/

93. Jacobsen LK, Southwick SM, Kosten TR. Substance use disorders in patients with posttraumatic stress disorder: A review of the literature. Am J Psychiatry. 2001;158(8):1184–90. https://doi.org/10.1176/APPALIP.158.8.1184/ASSET/IMAGES/LARGE/J52F1.JPEG.

94. Mason SM, Flint AJ, Field AE, Austin SB, Rich-Edwards JW. Abuse victimization in childhood or adolescence and risk of food addiction in adult women. Obesity. 2013;21(12):E775–81. https://doi.org/10.1002/oby.20500

95. Mason SM, Flint AJ, Roberts AL, Agnew-Blais J, Koenen KC, Rich-Edwards JW. Posttraumatic Stress Disorder Symptoms and Food Addiction in Women by Timing and Type of Trauma Exposure. JAMA Psychiat. 2014. https://doi.org/10.1001/jamapsychiatry.2014.1208.

96. Stojek MM, Maples-Keller JL, Dixon HD, Umierrez GE, Gillespie CF, Michopoulos V. Associations of childhood trauma with food addiction and insulin resistance in African-American women with diabetes mellitus. Appetite. 2019;141: 104317. https://doi.org/10.1016/J.appet.2019.104317.

97. Hardy R, Nani N, Jovanovic T, Michopoulos V. Food addiction and substance addiction in women: Common clinical characteristics. Appetite. 2018;120:367–73. https://doi.org/10.1016/J.appet.2017.09.026.

98. Masheb RM, Ruser CB, Min KM, Bullock AJ, Dorrflinger LM. Does food addiction contribute to excess weight among clinic patients seeking weight reduction? Examination of the Modified Yale Food Addiction Survey. Comp Psychiatry. 2018;84:1–6. https://doi.org/10.1016/j.comppsych.2018.03.006.

99. Mitchell KS, Wolf EJ. PTSD, food addiction, and disordered eating in a sample of primarily older veterans: The mediating role of emotion regulation. Psychiatry Res. 2016;243:23–9. https://doi.org/10.1016/j.psychres.2016.06.013.

100. Stojek MM, Lipka J, Maples-Keller JM, Rauch SAM, Black K, Michopoulos V, Rothbaum BO. Investigating Sex Differences in Rates and Correlates of Food Addiction Status in Women and Men with PTSD. Nutrients. 2021;13(6):1840. https://doi.org/10.3390/nu13061840.

101. Michopoulos V, Vester A, Neigh G. Posttraumatic stress disorder: A metabolic disorder in disguise? Exp Neurol. 2016;284(Pt B):220–9. https://doi.org/10.1016/j.expneurol.2015.05.038.

102. Avena NM, Rada P, Hoebel BG. Evidence for sugar addiction: behavioral and neurochemical effects of intermittent, excessive sugar intake. Neurosci Biobehav Rev. 2008;32(1):20–39.

103. Cottone P, Sabino V, Stuedo L, Zorrilla EP. Consummatory, anxiety-related and metabolic adaptations in female rats with alternating access to preferred food. Psychoneuroendocrinology. 2008;33(4):68–82. https://doi.org/10.1016/j.psyneuen.2008.01.010.

104. Brewerton TD. Posttraumatic Stress Disorder and Disordered Eating: Food Addiction as Self-Medication. Journal of Women’s Health. 2011;20(8):1133–4. https://doi.org/10.1089/jwh.2011.3050.

105. Fletcher PC, Kenny PJ. Food addiction: a valid concept? Neuropsychopharmacology. 2018;43(13):2506–13. https://doi.org/10.1038/s41386-018-0203-9.

106. Hebebrand J, Gearhardt AN. The concept of “food addiction” helps inform the understanding of overeating and obesity: NO. Am J Clin Nutr. 2021;113(2):268–73. https://doi.org/10.1093/ajcn/qnq344.

107. Bickel WK, Johnson MW, Koffarnus MN, MacKillop J, Murphy JG. The behavioral economics of substance use disorders: reinforcement pathologies and their repair. Annu Rev Clin Psychol. 2014;10:641–77. https://doi.org/10.1146/annurev-clinpsych-sy-032813-153724.

108. Carr KA, Daniel TO, Lin H, Epstein LH. Reinforcement pathology and obesity. Curr Drug Abuse Rev. 2011;4(3):190–6.

Publisher’s Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.