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Shared and unique mechanisms underlying binge eating disorder and addictive disorders

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HIGHLIGHTS

- Shared mechanisms may explain phenotypic overlap between "food addiction" and BED.
- Mechanisms unique to addictive disorders may contribute to addictive-like eating.
- Future research needed to examine the utility of "food addiction" is recommended.
- Addiction perspectives may inform novel interventions for disordered eating.

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ABSTRACT

Scientific interest in "food addiction" is growing, but the topic remains controversial. One critique of "food addiction" is its high degree of phenotypic overlap with binge eating disorder (BED). In order to examine associations between problematic eating behaviors, such as binge eating and "food addiction," we propose the need to move past examining similarities and differences in symptomology. Instead, focusing on relevant mechanisms may more effectively determine whether "food addiction" contributes to disordered eating behavior for some individuals. This paper reviews the evidence for mechanisms that are shared (i.e., reward dysfunction, impulsivity) and unique for addiction (i.e., withdrawal, tolerance) and eating disorder (i.e., dietary restraint, shape/weight concern) frameworks. This review will provide a guiding framework to outline future areas of research needed to evaluate the validity of the "food addiction" model and to understand its potential contribution to disordered eating.

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1. Introduction

It has recently been proposed that "food addiction" may be a contributor to obesity and eating-related problems (Gearhardt, Corbin, & Brownell, 2009a,b). This hypothesis proposes that certain foods, such as those high in sugar and fat, may be capable of triggering an addictive response in individuals with vulnerable characteristics (e.g., reward dysfunction, impulsivity) (Gearhardt, Davis, Kuschner, & Brownell, 2011). Early evidence in animal and human studies suggests that high-fat, high-sugar foods may activate reward-related neural circuitry in a similar manner as drugs of abuse (Gearhardt et al., 2011a; Johnson & Kenny, 2010a). Additionally, behavioral indicators of substance-use disorders, such as loss of control and use despite negative consequences, have been observed in response to these foods (Gearhardt et al., 2009a; Ifland et al., 2009). Although many symptoms of addictive disorders are behavioral in nature (e.g., consuming more than intended, inability to cut down on consumption) (American Psychiatric Association, 2013) and behavioral circumstances may increase addictive potential (e.g., intermittent access, binge patterns of use) (Hwa et al., 2011; Koob & Le Moal, 2001), the "food addiction" perspective does not reflect a behavioral addiction or an "eating addiction." Rather, akin to substance-use disorders, this framework posits an interaction between the addictive potential of high-fat, high-sugar foods, behavioral factors that may increase addictive responses (e.g., intermittent, binge consumption), and an individual's propensity to develop an addiction (Ahmed, Guillem, & Vandaele, 2013; Davis & Carter, 2009; Gearhardt et al., 2009a; Gold, Frost-Pineda, & Jacobs, 2003; Ifland et al., 2015; Ifland et al., 2009).

The Yale Food Addiction Scale (YFAS) is currently the only validated measure to assess symptoms of "food addiction" (Gearhardt et al., 2009b). The YFAS is a 25-item self-report questionnaire that applies the diagnostic criteria for substance-use disorders to consumption of certain foods (see Table 1). The YFAS provides two scoring options: a symptom count (a sum of the seven diagnostic criteria) and a diagnostic threshold that reflects the criteria for a substance dependence diagnosis (the presence of three or more symptoms plus clinically significant impairment or distress). The YFAS has good internal consistency ranging from $\alpha = .76$–$.92$ (Meule & Gearhardt, 2014) and demonstrates convergent validity with measures of eating pathology (e.g., emotional eating, food craving) and incremental validity in predicting binge eating frequency above and beyond existing measures (for a review see Meule & Gearhardt, 2014) The YFAS has been used to assess "food addiction" in both community and treatment-seeking samples and has been translated to German, French, Spanish, and Italian (Granero et al., 2014; Meule & Gearhardt, 2014; Pursey, Stanwell, Gearhardt, Collins, & Burrows, 2014).

Though "food addiction" is receiving increased attention, the topic remains controversial (Avena, Gearhardt, Gold, Wang, & Potenza, 2012; Corsica & Pelchat, 2010; Corwin & Hayes, 2014; Ziauddeen & Fletcher, 2013). This model posits that certain foods are addictive akin to substance-use disorders; however, there have been few studies examining which foods or ingredients in foods may have addictive potential (Corwin & Hayes, 2014; Ziauddeen & Fletcher, 2013). While initial evidence in animals and humans suggest that high-fat, high-sugar foods are most associated with behavioral indicators of "food addiction" (Avena, Rada, & Hoebel, 2008; Johnson & Kenny, 2010a; Schulte, Avena, & Gearhardt, 2015), identifying the potentially addictive agent in these foods is a critical next step in this line of research. Additionally, it has been suggested that "food addiction" cannot account for obesity, as only a relatively small percentage of obese individuals meet for YFAS diagnosis (Corwin & Hayes, 2014; Ziauddeen, Farooqi, & Fletcher, 2012). Thus, there have been conflicting findings in neuroimaging studies examining whether neural circuits implicated in addiction are also relevant to obesity (Corwin & Hayes, 2014; Ziauddeen et al., 2012). However, obesity is a multi-faceted condition that can result from a complex combination of a number of potential genetic and environmental factors, including for example, physical inactivity, medication side effects, and sleep problems, in addition to excessive food intake (Grilo & Pogue-Geile, 1991; Keith et al., 2006; Marcus & Wildes, 2009; Wright & Aronne, 2012). Although "food addiction" is more prevalent in participants with obesity (Flint et al., 2014), it has been observed in a range of weight classes (Gearhardt et al., 2009b) and may explain a unique phenotype of problematic eating behavior. Thus, obesity should not be used as a proxy for "food addiction" in future behavioral and neuroimaging studies.

Another important critique is the substantial phenotypic overlap between binge eating disorder (BED) and definitions of "food addiction." Both BED and addiction are marked by loss of control over consumption, continued excess use despite negative consequences, and repeated, failed attempts to cut down on consumption (Gold et al., 2003). As a result of these similarities, measures of binge eating and "food addiction" (YFAS) are often highly correlated, both reflecting and resulting in the difficulty of evaluating and disentangling potential shared and unique aspects of these different constructs. For example, data from these types of measures cannot readily be placed in the same statistical model due to multi-collinearity concerns (Gearhardt, Rizk, & Treat, 2014).[1] YFAS "food addiction" and BED commonly co-occur, although these constructs do not completely overlap. In samples of individuals with BED, the frequency of "food addiction" ranges from 42% to 57% and "food addiction" symptoms predict the frequency of binge eating episodes above and beyond measures of eating pathology and depression (Gearhardt, White, Masheb, & Grilo, 2013; Gearhardt et al., 2012). Individuals who meet the criteria for both BED and "food addiction" exhibit more frequent binge eating episodes, intense cravings, and depressive symptoms than those with only BED (Davis & Carter, 2009; Gearhardt et al., 2012). Among individuals who meet the criteria for YFAS "food addiction," the frequency of BED ranges from 27% to 30% (Davis et al., 2011; Gearhardt, Boswell, & White, 2014). Notably, in community studies with diverse weight groups, individuals categorized with "food addiction," but not BED, report significant levels of impairment and distress, such as depressive symptoms, impulsivity, and negative affect (Gearhardt, Boswell, & White, 2014). However, by focusing primarily on the psychometric and phenotypic overlap of "food addiction" and BED, it is challenging to evaluate whether an addictive process

Table 1
YFAS symptoms based on DSM-IV criteria for substance dependence.

(1) Substance taken in larger amount and for longer period than intended
(2) Persistent desire or repeated unsuccessful attempt to quit
(3) Much time/activity to obtain, use, recover
(4) Important social, occupational, or recreational activities given up or reduced
(5) Use continues despite knowledge of adverse consequences (e.g. failure to fulfill role obligation, use when physically hazardous)
(6) Tolerance (marked increase in amount; marked decrease in effect)
(7) Characteristic withdrawal symptoms; substance taken to relieve withdrawal

[1] For example, a recent study by (Gearhardt, Rizk, et al., 2014) found that YFAS and Binge Eating Scale scores were correlated at .751.

uniquely contributes to some types of disordered eating or whether "food addiction" is BED slightly reframed.

We propose the need to move past the use of descriptive similarities and differences to understand possible relationships between disordered eating, such as binge eating, and "food addiction." Examining potential mechanisms underlying problematic eating behaviors may more appropriately determine whether an addictive process contributes to disordered eating for some individuals. Thus, there are three goals of this review. First, we review the shared and unique mechanisms implicated in addictive and eating-related problems to elucidate the relationship between addictive disorders and BED. Second, we examine how mechanisms unique to substance-use disorders may relate to problematic eating behavior in order to evaluate whether a "food addiction" framework may offer a novel understanding of eating-related issues. Third, we identify gaps in the literature and provide a theoretical roadmap to guide future research needed to determine the validity and utility of "food addiction" as a substance-based addictive disorder.

2. Shared mechanisms in addictive disorders and BED

Addiction and eating disorder perspectives share a number of proposed mechanisms, which may contribute partly to the similarities observed between BED and "food addiction" measures (Gearhardt et al., 2014c). This section will focus on how reward dysfunction, craving, emotion dysregulation, and impulsivity are relevant contributors to both addictive disorders and BED.

2.1. Reward dysfunction

Dysfunction in reward-related processes is one area similarly implicated in theories of addiction and disordered eating. Addictive substances and high fat/sugar foods appear to similarly implicate the mesolimbic dopamine system, which has been associated with wanting and motivation for a substance, and the mu-opioid systems, which are more closely related to the liking of the sensory experience (Berridge, 2007, 2009; Robinson & Berridge, 1993). The role of reward in addiction has long been discussed beginning in the 1950s and 1960s (Beach, 1957; Eveson, 1962). Specifically, addiction is thought to occur, in part, because addictive substances trigger hedonically pleasurable effects (Everitt & Robbins, 2005) and cause changes in reward-related neural systems (Dackis & O'Brien, 2001; Koob & Le Moal, 1997).

Although there is a consensus that reward dysfunction contributes to the risk of developing an addiction (Blum et al., 2000; Koob & Le Moal, 2001), there is a debate about whether hypo- or hyper- response to reward is more problematic. One hypothesis suggests that individuals with addiction use highly rewarding drugs or behaviors[2] as a way to compensate for a deficient reward system (Blum et al., 2000; Volkow, Fowler, & Wang, 1999). In other words, these individuals use drugs of abuse to feel a level of reward others experience with less potent rewards (e.g., social interactions). As a result, individuals with hypo-responsive reward systems may seek out drugs in order to experience pleasure not achievable through other means (Reuter et al., 2005). In support of the hypo-reward hypothesis, persons with addiction exhibit reduced dopamine D2/D3 receptor availability, have less mesolimbic dopaminergic response to non-drug rewarding stimuli (e.g., money), and are more likely to have the DRD2 receptor A1 allele (which is associated with less D2-type receptor availability) (Blum et al., 2000; Reuter et al., 2005; Volkow et al., 1999). However, excess consumption of drugs of abuse can alter the reward system in a manner that reduces reward responsivity and D2-type receptor availability (Di Chiara & Bassareo, 2007; Koob & Le Moal, 1997; Spanagel & Weiss, 1999). Thus, it is unclear whether hypo-responsivity in the reward system is a cause or a consequence of addictive behaviors.

In contrast, hyper-reward responses to substance-related cues (e.g., alcohol advertisements, cigarette packs) are consistently found in addiction (Di Chiara, 1998; Everitt & Wolf, 2002; Robinson & Berridge, 1993). In other words, the reward system may generally be hypo-responsive, but becomes hyper-responsive to addiction-relevant cues. Individuals with addictions exhibit elevated activation in reward-related neural circuitry, such as the dorsolateral anterior cingulate cortex (dACC) and the orbitofrontal cortex (OFC), in response to substance-related cues (Engelmann et al., 2012; Goudriaan, de Ruiter, van den Brink, Oosterlaan, & Veltman, 2010; Yang et al., 2009). These cues can also become highly salient, as reflected by elevated attention biases for drug-related cues among individuals with addiction (Field & Cox, 2008; Hester, Dixon, & Garavan, 2006; Lubman, Peters, Mogg, Bradley, & Deakin, 2000). Greater reactivity to these cues predicts intensity of drug craving and probability of relapse during quit attempts (Carter & Tiffany, 1999; Janes et al., 2010; Marissen et al., 2006). Thus, elevated reward sensitivity to addiction-relevant cues appears to be a major factor underlying addictive disorders.

Preliminary evidence suggests that reward dysfunction appears to be implicated in "food addiction." The only neuroimaging study that has directly examined "food addiction" by using the YFAS observed patterns in reward circuitry that are also seen in addictive disorders (Gearhardt et al., 2011a). Individuals endorsing three or more YFAS "food addiction" symptoms exhibited increased neural activity in reward-related regions when anticipating a highly processed food (i.e., chocolate milkshake), relative to those who only endorsed one YFAS symptom (Gearhardt et al., 2011a). This pattern is characteristic of individuals with addictive disorders with respect to the anticipation of drug rewards (Martinez et al., 2005; Volkow et al., 2006), which provides evidence that reward dysfunction may similarly contribute to traditional addictive disorders and "food addiction."

Reward dysfunction may also be a relevant mechanism in BED. Since the 1980s, reward sensitivity and dysfunctional reward responses to highly palatable, highly processed foods have been proposed as one potential explanation for binge eating (Dum, Gramsch, & Herz, 1983; Fullerton, Getto, Swift, & Carlson, 1985). Individuals prone to overeating may be driven to eat outside of homeostatic hunger to experience the hedonic, pleasurable effects of consuming certain foods (Lowe & Butryn, 2007). Though it has been argued that foods consumed during binge eating episodes vary in nutritional composition (Goldfein, Walsh, LaChaussee, Kissileff, & Devlin, 1993; Walsh, 2011), some studies have observed that highly processed foods (e.g., chocolate, French fries) are most frequently consumed when bingeing (Allison & Timmerman, 2007; Marcus, Wing, & Hopkins, 1988). Additionally, rats exhibit binge-eating behavior in response to highly processed food, but not to regular chow (Hagan, Chandler, Wauford, Rybak, & Oswald, 2003). As with drugs of abuse, consumption of these calorie-dense, nutrient-poor foods appears to activate the mesolimbic dopamine and endogenous opiate systems, which may be associated with the reinforcing properties of highly processed foods (Davis et al., 2009; Volkow, Wang, Fowler, & Telang, 2008).

Akin to addiction, the role of hypo- versus hyper-reward processes in binge eating is also unclear. Individuals who binge eat may be hypo-responsive to rewards compared to healthy controls, resulting in these individuals binging on highly processed food to mediate this reward deficiency (Blum, Cull, Braverman, & Comings, 1996). In support of this hypothesis, obese individuals with BED exhibit less activation in limbic regions implicated in reward compared to obese individuals without BED when exposed to monetary rewards (Balodis et al., 2013a). The presence of DRD2 receptor A1 allele is also linked to compulsive overeating, which may suggest that reward hyposensitivity is a genetic risk factor for the development of problematic eating behavior (Blum et al., 2000; Davis et al., 2008). However, as with addictive disorders, it is unclear whether reward hyposensitivity is a cause or effect of

[2] In this manuscript, we will use the terms "substance" or "consumption" for ease of writing, but in the case of gambling this can also be applied to behavior and engagement in the behavior.

BED because excess consumption of highly processed foods may result in changes to the dopamine system to decrease reward sensitivity (Bello, Lucas, & Hajnal, 2002; Berridge, 2009; Johnson & Kenny, 2010a).

As demonstrated in addiction, persons with BED appear hyper-responsive to food-related cues. Individuals with BED compared with overweight and healthy controls exhibit increased activation in neural regions associated with reward appraisal (such as the OFC) in response to food stimuli (Schienle, Schafer, Hermann, & Vaitl, 2009; Weygandt, Schaefer, Schienle, & Haynes, 2012). One study found that binge eating, but not body mass index (BMI), was associated with increased striatal dopamine release in response to food stimulation (Wang et al., 2011). Akin to addictive disorders, individuals with BED may exhibit attention biases to high-calorie foods (Svaldi, Tuschen-Caffier, Peyk, & Blechert, 2010). However, it may be challenging to measure reward-responsiveness to food, as the reinforcing properties may be greater when individuals who binge eat are in a binge setting compared to being instructed not to binge eat (Schebendach, Broft, Foltin, & Walsh, 2013). This suggests that hyper-responsiveness to food cues may vary based on state with increased responsiveness when bingeing is possible. Although there is limited literature on this topic, existing studies appear to support a hyper-reward response to food cues for individuals who binge eat, especially when in a binge setting. Thus, hyper-responsivity to cues appears to be a common mechanism in BED and addictive disorders.

2.2. Craving

As with reward dysfunction, craving is implicated in both addiction and BED. Craving is a central component of addiction (Potenza & Grilo, 2014) that has been added to the diagnostic criteria for addictive disorders in the DSM-5 and is defined as a "strong desire or urge to use" (American Psychiatric Association, 2013). In addictive disorders, cravings can be triggered by environmental cues of the drug and its hedonic effects ("reward craving") or by the internal experience of withdrawal symptoms ("relief craving") (Kilts et al., 2001; Verheul, van den Brink, & Geerlings, 1999). Triggers of craving (e.g., drug cues) may produce a dopaminergic release akin to consuming an addictive substance, which can increase drug-seeking behavior (Volkow et al., 2006; Volkow et al., 2008a). Activation of reward-related brain regions such as the insula, amygdala, OFC, and hippocampus have been observed in response to cue-induced craving (Bonson et al., 2002; Childress et al., 1999; Schneider et al., 2001; Wang et al., 1999). Thus, cravings appear to contribute to problematic use and compulsive drug seeking and may be related to elevated relapse rates in addictive disorders (Bottlender & Soyka, 2004; Killen & Fortmann, 1997).

Craving has also been examined as a potential mechanism in "food addiction." Food craving is frequently described as "an intense desire to consume a specific food that is difficult to resist" (White, Whisenhunt, Williamson, Greenway, & Netemeyer, 2002), and food and drug cravings appear to activate similar brain regions (Kelley & Berridge, 2002; Pelchat, 2002; Pelchat, Johnson, Chan, Valdez, & Ragland, 2004; Tang, Fellows, Small, & Dagher, 2012). Individuals who endorse greater YFAS symptoms of "food addiction" report elevated food cravings (Davis et al., 2011; Gearhardt et al., 2014c; Meule & Kubler, 2012), particularly for high-fat, high-sugar foods (Gearhardt et al., 2014c), but do not have an expectation to be positively reinforced by consuming the craved foods (Meule & Kubler, 2012). This may reflect incentive sensitization, a common process in addictive disorders, where individuals experience a strong wanting or craving for a drug/food reward, but do not necessarily experience increased liking or pleasure (Berridge, 2009; Robinson & Berridge, 1993). While research in this area is in its nascent stages, existing evidence suggests that craving may contribute to "food addiction" in a manner akin to substance-use disorders.

Similar to addictive disorders, craving may be a relevant mechanism in BED. Craving may differ across weight categories, and associations between cravings and other clinical measures may vary across specific groups (White & Grilo, 2005). Notably, obese persons with BED generally have greater cravings for food and exhibit increased craving prior to consumption, relative to obese individuals without BED or normal-weight controls (Greeno, Wing, & Shiffman, 2000; Innamorati et al., 2014; Ng & Davis, 2013; White & Grilo, 2005). Akin to substance-use disorders, craving in BED may occur in response to food cues (Sobik, Hutchison, & Craighead, 2005), which may be associated with overeating (Jansen et al., 2003) or binge eating episodes. For example, Ng and Davis (2013) observed that the level of craving before food exposure predicted overeating in individuals with BED. Another study found that craving, even in the absence of hunger, was the best predictor for the onset of bingeing (Greeno et al., 2000). However, literature in this topic remains limited and further research is needed to determine whether cue-elicited craving may produce a dopaminergic response and activate reward-related regions (e.g., insula, OFC), akin to addictive disorders. Though it has been suggested that sweets (White & Grilo, 2005) and/or fats (Yanovski et al., 1992) may be implicated in binge eating episodes, future studies should examine which foods and food attributes (e.g., high-fat, highly processed) are frequently craved. In summary, craving may be an underlying mechanism in both binge eating behavior and compulsive drug use.

2.3. Emotion dysregulation

Emotion dysregulation, or the ineffective modulation of negative affect, is another important mechanism in both addictive disorders and BED. Negative affect has been associated with elevated cravings for an addictive substance since the 1960s (Ikard, Green, & Horn, 1969; Ikard & Tomkins, 1973; Tomkins, 1966). Negative affect can occur in response to a stressor or physical or psychological withdrawal symptoms, and is linked to increased consumption of drugs of abuse (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Kenford et al., 2002; Kenny, Chen, Kitamura, Markou, & Koob, 2006; Sinha, 2001; Sinha, Catapano, & O'Malley, 1999; Sinha, Fuse, Aubin, & O'Malley, 2000). For some individuals, the substance may be used to cope with negative affect, which may reinforce continued self-administration (Baker et al., 2004; Brandon, 1994; Li & Sinha, 2008). Neuroimaging studies have shown that individuals addicted to psycho-stimulants demonstrate diminished activation in emotion processing regions (e.g., anterior cingulate cortex) and increased activation in regions associated with craving (e.g., striatum) in response to stress (Sinha et al., 2005). This supports the idea that individuals with addictive disorders may crave drugs of abuse in order to regulate negative emotional states. Further, negative affect appears to be a predictor of relapse, especially for individuals using drugs to cope (Miller, Westerberg, Harris, & Tonigan, 1996; Myers & Brown, 1990; Sinha, Garcia, Paliwal, Kreek, & Rounsaville, 2006) and when the onset of negative affect is rapid (Shiffman & Waters, 2004).

Akin to substance-use disorders, emotion dysregulation may contribute to "food addiction." As individuals with addiction may utilize drugs in response to strong emotional states (Fox, Hong, & Sinha, 2008; Li & Sinha, 2008), persons with "food addiction" may consume certain foods in an effort to regulate emotions. In support of this theory, YFAS symptoms have been associated with greater endorsement of emotion dysregulation on self-report questionnaires (Gearhardt et al., 2013b; Gearhardt et al., 2012; Pivarunas & Conner, 2015). Further, individuals with elevated YFAS symptoms report that they are often motivated to consume highly processed foods in an effort to cope with negative emotions (Joyner, Schulte, Wilt, & Gearhardt, 2015). This preliminary work suggests that emotion dysregulation may be a mechanism implicated in "food addiction." Future research could utilize advanced methodologies as ecological momentary assessment to dissect the process of how certain emotional states may trigger consumption of highly processed foods among individuals reporting YFAS "food addiction" symptoms and whether these patterns parallel precipitants of drug use in persons with substance-use disorders.

Emotion dysregulation has been proposed as a relevant mechanism in BED since the 1990s (Greeno et al., 2000; Grilo, Shiffman, & Carter-Campbell, 1994; Heatherton & Baumeister, 1991; Mussell et al., 1996; Stice, Akutagawa, Gaggar, & Agras, 2000; Telch & Agras, 1996). The experience of negative affect may induce cravings for highly processed foods and often precedes overeating (Grilo, Shiffman, & Wing, 1989) and specifically binge-eating episodes (Berg et al., 2014; Goldschmidt et al., 2014; Greeno et al., 2000; Telch & Agras, 1996). Binge eating, as substance-use, has long been suggested as a coping mechanism for intense emotional states (McManus & Waller, 1995; Nasser, Gluck, & Geliebter, 2004). In support of this hypothesis, Ranzenhofer et al. (2014) observed that interpersonal problems predict loss-of-control eating episodes, a potential precursor to BED, in adolescent girls. Ansell, Grilo, and White (2012) reported that interpersonal problems are associated with binge eating in women and that these effects were statistically mediated by negative affect. Additionally, Whiteside et al. (2007) found that emotion regulation deficits were the strongest predictor of binge eating above gender, restriction, and shape and weight concerns. Research is needed to examine whether the neural correlates of negative affect are similar among individuals with BED and substance-use disorders. As seen in addictive disorders, it appears that binge eating may be an attempt to cope with negative affect.

2.4. Impulsivity

Impulsivity is another mechanism underlying both addictive disorders and BED. Impulsivity refers to responding to internal or external stimuli in an unplanned manner, with little regard for potentially negative consequences that may result (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). The role of impulsivity in addictive disorders started receiving attention in the 1950s (Lolli, 1951; Zimmering, Toolan, Safrin, & Wortis, 1951). While impulsivity may be a risk-factor for whether an individual develops an addiction, chronic substance use may also lead to worsening executive control deficits throughout the course of the addiction (de Wit, 2009). Addicted individuals typically show dysfunction in brain regions associated with impulsivity during decision-making tasks (Hester & Garavan, 2004; Hester, Lubman, & Yucel, 2010) and are impaired at delaying gratification of rewards (Hoffman et al., 2008; Monterosso et al., 2007). Thus, impulsivity may explain maladaptive decision-making in addiction where individuals engage in behavior that is rewarding in the short-term (e.g., drug use), despite being detrimental to them in the long-term (e.g., health problems, interpersonal difficulties). Importantly, impulsivity has been linked to higher rates of relapse in addictive disorders (Doran, Spring, McChargue, Pergadia, & Richmond, 2004; Miller, 1991).

Impulsivity may also be implicated in "food addiction." Previous studies have found that YFAS "food addiction" symptoms are associated with attention-based impulsivity, marked by a faster response time to food cues (Meule, Lutz, Vogele, & Kubler, 2012), negative urgency, reflecting the tendency to act impulsively while experiencing negative mood states (Murphy, Stojek, & MacKillop, 2014; Pivarunas & Conner, 2015), and a lack of perseverance in tasks (Murphy et al., 2014). In the only neuroimaging study of YFAS "food addiction," individuals reporting three or more YFAS symptoms exhibited decreased activation in inhibitory neural systems when consuming a highly processed food reward (i.e., chocolate milkshake), relative to persons endorsing one or fewer symptoms (Gearhardt et al., 2011a). Thus, akin to substance-use disorders, impulsivity may be a relevant mechanism in "food addiction" and warrants further empirical examination.

Impulsivity has also been discussed as an important mechanism in BED beginning in the 1990s (McManus & Waller, 1995; Mitchell & Mussell, 1995). Previous studies have found that individuals who binge eat demonstrate deficiencies in impulsivity, have trouble focusing attention, make risky decisions, and are poor at utilizing feedback to guide future behavior (Mobbs, Iglesias, Golay, & Van der Linden, 2011; Svaldi, Brand, & Tuschen-Caffier, 2010). It has also been suggested that impulsivity plays a role on the initiation of binge cravings (Dawe & Loxton, 2004; McManus & Waller, 1995). Similar to addictive disorders, binge eaters are also less successful at delaying gratification of rewards (Davis, Patte, Curtis, & Reid, 2010; Manwaring, Green, Myerson, Strube, & Wilfley, 2011). Thus, as seen in addiction, a maladaptive decision-making process may occur in individuals with BED, where the short-term reward of highly processed food is chosen instead of long-term health benefits. Schag et al. (2013) observed that individuals with BED exhibited impulsive eye movements in response to food stimuli. Neuroimaging studies suggest that individuals with BED have greater activation of executive control regions during a cognitive-control task, suggesting a maladaptive decision-making process (Balodis et al., 2013b; Gearhardt, Boswell, & Potenza, 2014). Akin to addictive disorders, high levels of impulsivity may also have implications in treatment of BED and rates of relapse. Several studies have found that impulsive children are more likely to binge eat and are less successful in treatment programs (Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006; Nederkoorn, Jansen, Mulkens, & Jansen, 2007). Therefore, impulsivity appears to be a mechanism implicated similarly in substance-use disorders and BED.

3. Summary of shared mechanisms

In summary, addiction and binge eating share a number of proposed mechanisms. For both of these disorders, individuals may exhibit elevated reward responsivity to relevant cues and may act compulsively in the presence of these cues to satisfy intense cravings. Cravings may also be triggered by negative mood states, in which the drug or food may be used to regulate emotions. Finally, impulsivity has been identified as a shared mechanism underlying both substance-use disorders and BED and may have implications for continued administration and relapse.

Collectively, these shared mechanisms likely contribute to the behavioral and biological similarities between binge eating and traditional addictive disorders (Cassin & von Ranson, 2007; Gearhardt, White, & Potenza, 2011; Gold et al., 2003). Notably, smokers with BED are more likely to have a substance-use disorder than non-smokers with BED (White & Grilo, 2006), which provides further evidence for similar processes underlying BED and addiction at least in some persons or subgroups. If addictive disorders and BED are both explained in part by shared mechanisms, we propose that it may be plausible that an addictive process may be contributing to binge eating for some individuals. In support, preliminary evidence suggests that reward dysfunction, craving, emotion dysregulation, and impulsivity may all also contribute to "food addiction," as measured by the YFAS. However, it is challenging to disentangle BED and "food addiction" given the high degree of phenotypic overlap and these common mechanisms. There is evidence that individuals who experience "food addiction," but who do not meet criteria for BED or other eating disorders, exhibit clinically significant levels of pathology and eating-related concerns (Gearhardt, Boswell, & White, 2014). Further, individuals with an eating disorder diagnosis who also meet for YFAS "food addiction" have particularly severe levels of pathology (Gearhardt, Boswell, & White, 2014). Thus, the assessment of "food addiction" appears to provide clinically relevant information to the study of disordered eating. In order to further evaluate whether an addictive process may explain problematic eating behavior in some individuals, it is essential to examine mechanisms unique to an addiction perspective (e.g., withdrawal) and traditional eating disorder approaches (e.g., shape and weight concerns).

4. Unique aspects of addiction perspectives

We now review several core mechanisms of substance-use disorders: the importance of the substance, withdrawal, and tolerance. We evaluate whether these processes may contribute to problematic eating

behavior in a manner similar to addictions. If certain foods share features with drugs of abuse and may be capable of triggering addictive responses in some individuals, this would provide support for the validity and utility of a "food addiction" framework.

4.1. Importance of the substance

An addiction framework posits that an addictive substance (e.g., nicotine) or behavior (e.g., gambling) interacts with individual risk factors (e.g., genetic predisposition, personality characteristics) to trigger an addictive process. Without exposure to the addictive substance, an individual at risk will not develop an addiction. Thus, the addictive substance or behavior is an essential aspect of the problematic behavior. Drugs of abuse are altered from their natural state to contain an increased concentration (or dose) of the addictive agent and to increase the rate in which the addictive agent is absorbed into the system (Barnett, Hawks, & Resnick, 1981; Henningfield & Keenan, 1993). For example, coca leaves have little addictive potential when chewed (Hanna & Hornick, 1977), but when they are refined into cocaine and consumed by nasal insufflation, they become highly addictive (Verebey & Gold, 1988). Further, addictive substances may be capable of producing neuroplastic changes in brain regions responsible for processing rewards and making decisions (Koob, 1992; Leshner, 1997). These changes in the mesolimbic dopamine system and prefrontal cortex may perpetuate self-administration (Chang, Alicata, Ernst, & Volkow, 2007; Volkow & Fowler, 2000). In other words, an individual's brain may become "hijacked" by the addictive substance, resulting in compulsive consumption despite negative consequences (Volkow & Wise, 2005).

Highly processed foods (e.g., pizza, chocolate, chips) may share characteristics with drugs of abuse, as they are altered from their natural state to include higher concentrations of fat and/or refined carbohydrates like sugar or white flour, and the rate in which the refined carbohydrates are absorbed into the system is rapid (Gearhardt, Davis et al., 2011). For example, a minimally processed food, such as a banana, contains natural sugars but would be expected to have little or no addictive potential compared to a candy bar, which is processed to contain increased levels of fat and rapidly absorbed sugars.

Additionally, highly processed foods may also be capable of causing neuroplastic changes in reward-related brain regions that contribute to behavioral indicators of "food addiction" (Bello et al., 2002; Berridge, 2009; Johnson & Kenny, 2010b), although research on this topic is in its nascent stages. Rats who consume a diet of highly processed foods, such as cheesecake, experience downregulation in the dopamine system, akin to using drugs of abuse (Johnson & Kenny, 2010b). Notably, binge-prone rats only exhibit addictive-like responses to foods high in fat and sugar (e.g., Oreo Double-Stuf cookies) and not chow (Boggiano et al., 2007; Klump, Racine, Hildebrandt, & Sisk, 2013). Animal models suggest that sugar may be most closely associated with "food addiction," as rats with intermittent access to sugar exhibit binge eating, enhanced motivation to obtain the substance (which may model craving), cross-sensitization, and withdrawal when the sugar is removed (Avena et al., 2008b) (for a review see Avena, 2010). In humans, Stice, Burger, and Yokum (2013a, 2013b) observed that a high-sugar milkshake strongly activated reward-related regions. Further, pharmacology used to treat alcohol-related problems by blocking opioid response (i.e., naloxone) has also been associated with reduced consumption of ultra-processed foods in binge eaters (Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1995). Additionally, in the only neuroimaging study of "food addiction," individuals with elevated YFAS symptoms exhibit neural responses akin to other addictive disorders in response to cues and consumption of a highly processed food (Gearhardt et al., 2011a).

One study has examined which foods are most likely to be implicated in "food addiction" in humans (Schulte et al., 2015). Schulte et al. (2015) asked participants to complete the YFAS then rate how likely they were to experience YFAS symptoms with 35 nutritionally diverse foods. Highly processed foods, with added fat and refined carbohydrates were identified as the most problematic. Foods with a high glycemic load (which reflects a faster speed of absorption of carbohydrates and a greater blood sugar spike) were especially problematic for individuals endorsing "food addiction" symptoms on the YFAS. Thus, like with drugs of abuse, the rapid speed of absorption of rewarding ingredients may make highly processed foods particularly triggering for susceptible individuals. Laboratory studies with direct observations and measurements of eating behavior and reward response are needed to confirm and extend these preliminary findings. Though more research is needed, if highly processed foods cause similar biological and behavioral changes as drugs of abuse, this would support applying an addiction perspective to problematic eating behavior in some individuals.

If certain foods are capable of triggering addiction-like changes in the brain, it would challenge one core tenet of traditional eating disorder treatment models such as cognitive-behavioral therapy (CBT) (Fairburn, 2008; Grilo & Mitchell, 2011) which states that there are no "bad" or "forbidden" foods. CBT perspectives on eating disorders emphasize individual differences (e.g., shape and weight overconcern, excessive or rigid dietary restraint, mood regulation difficulties, poor self-esteem) as factors that serve to either lead to and/or maintain problematic eating behaviors (Fairburn, 1995, 2008; Grilo & Mitchell, 2011); in such models, how individuals under- or over-eat (in the case of BED) is a key aspect but that the types of food consumed less importance (Fairburn, 1995, 2008; Grilo & Mitchell, 2011) In contrast, an addiction perspective suggests that some foods (or how some foods are processed) may trigger the reward system in a manner that makes it more challenging for at-risk individuals to moderate their consumption (Gearhardt, Davis et al., 2011). Thus, it is possible that certain foods may not merely be "bad" foods per a common cognitive bias in CBT models but may actually possess chemical properties that make them more difficult to eat in a controlled manner for both cognitive and neurobiological reasons.

Importantly, exposure to addictive substances does not universally trigger compulsive consumption, and individual differences (e.g., genetics, patterns of consumption, personality factors) play an important role in who becomes addicted. For example, 90% of people consume alcohol (an addictive substance) during their lifetime, but only 5–10% of individuals develop alcohol dependence (American Psychiatric Association, 2000; Grant, 1997). However, there are many individuals with subclinical addictive-like responses to alcohol, which emphasizes the broad public health implications of classifying alcohol as an addictive substance. Similarly, if some foods have an addictive potential, it is unlikely that all or most people would develop a clinical-level of "addiction." It is also probable that other individuals would experience subclinical addictive-like responses to certain foods, which increases the scope of potential public health initiatives (Gearhardt, Grilo, DiLeone, Brownell, & Potenza, 2011). It is likely that individual differences associated with addiction risk (e.g., impulsivity, family history of addiction) will interact with the potentially addictive nature of certain foods to trigger "compulsive" eating in some, but not all, individuals. In summary, for some cases of BED, mechanisms associated with dietary restraint or shape and weight concerns may precipitate binge-eating behavior. For others, highly processed foods may result in reward-related, neuroplastic changes in the brain and trigger an addictive process for at-risk individuals. This highlights the necessity of a mechanism-focused approach to understanding problematic eating behavior and for whom each mechanism may be most applicable.

4.2. Withdrawal and tolerance

Withdrawal and tolerance are unique markers of an addictive process that may provide insight into craving and relapse. Withdrawal is defined in the DSM-5 as the development of physiological or psychological symptoms in response to abstinence or decreased consumption of a substance (American Psychiatric Association, 2013). Though physical

withdrawal symptoms were previously considered a core component of addiction, they are no longer a mandatory characteristic of an addictive disorder, as withdrawal from drugs such as cocaine and behavioral addictions such as gambling result in minimal physical symptoms (Brower & Paredes, 1987; Rosenthal & Lesieur, 1992; Weddington et al., 1990). Psychological withdrawal symptoms, such as preoccupation and anxiety, are considered more universal across addictive disorders and are predictive of relapse rates (Kenford et al., 2002; West, Hajek, & Belcher, 1989).

Animal-model researchers have observed opioid-like withdrawal symptoms following addictive-like consumption of sucrose (Avena, Rada, & Hoebel, 2009). When sucrose is removed from the diet, rats experience anxiety, teeth chattering, and more aggressive behavior (Avena, Bocarsly, Rada, Kim, & Hoebel, 2008; Galic & Persinger, 2002). No previous studies have examined withdrawal symptoms in humans for highly processed foods. However, individuals who cut back on high-sugar foods (e.g., dieters) may exhibit metabolic changes in response to food cues, such as a decrease in blood sugar, and this may trigger cravings (Herman & Mack, 1975). Similar physiological changes can occur in response to cues for individuals with addictive disorders, which also results in strong cravings (Gearhardt et al., 2009a). Further, individuals experiencing problematic eating behavior anecdotally report withdrawal-like symptoms to carbohydrates (Gearhardt et al., 2009a; Pelchat, 2002). Thus, it is plausible that some individuals may experience withdrawal symptoms to highly processed foods. We are unaware of any prior studies that have investigated whether individuals endorsing YFAS "food addiction" symptoms develop a withdrawal in response to cutting down on high-fat/high-sugar foods, and this is an important area that requires further examination.

Similar to withdrawal symptoms, tolerance is another unique component of an addiction perspective. Tolerance is characterized by 1) the need to consume increased amounts of a substance to experience the desired, hedonic effects or 2) the diminished effect of a constant dose over time (American Psychiatric Association, 2000). Reductions in reward-related neural pathways over time in response to a constant dose of a substance may model the development of tolerance. This has been demonstrated in traditional addictive disorders (Nestler & Malenka, 2004; Volkow, Fowler, Wang, Baler, & Telang, 2009). Importantly, a recent study observed that frequent ice-cream consumption was associated with reduced striatal responsivity during the receipt of an ice-cream based milkshake, which is consistent with the development of tolerance (Burger & Stice, 2012). Behavioral indicators of tolerance have also been explored. Spring et al. (2008) observed that a beverage high in carbohydrates became less effective at reducing self-reported dysphoric mood in obese individuals after repeated administration. No studies to date have investigated whether individuals with YFAS "food addiction" symptomology exhibit greater tolerance to the consumption of high-fat/high-sugar foods. The lack of empirical examination of withdrawal and tolerance is a significant gap in understanding how addiction-specific mechanisms may contribute to eating-related problems and an essential next step for evaluating the validity of the "food addiction" concept.

In summary, exploring whether withdrawal and tolerance contribute to binge eating behavior is important for evaluating the "food addiction" hypothesis because these components are unique to an addiction framework. There is no existing research examining the association of "food addiction" or BED with these mechanisms. If symptoms of withdrawal and tolerance contribute to eating pathology this would provide evidence that an addiction framework has unique explanatory power for BED. It is likely that while addiction mechanisms may be relevant for certain individuals with BED, the unique aspects of the eating disorder framework may be important for others.

5. Unique aspects of eating disorder perspectives

We now describe mechanisms implicated in eating disorders like BED and discuss why these processes would not be considered causal factors in the "food addiction" model. If mechanisms unique to a traditional eating disorder perspective explain BED but not "food addiction," this would suggest that these two phenotypes do not overlap completely.

5.1. Restraint

Eating disorder models have long held that inappropriate (e.g., extreme or rigid) dietary restraint is a causal antecedent to binge eating (Fairburn & Harrison, 2003; Herman & Polivy, 1990; Howard & Porzelius, 1999; Polivy & Herman, 1985; Telch & Agras, 1993). Restraint was initially thought to be an intentional attempt to reduce calories below a biological "set point" of consumption (Herman & Mack, 1975; Nisbett, 1972). However, individuals reporting high levels of restraint often do not appear to be biologically deprived of calories (Lowe, 1993), suggesting that restraint may be more cognitive in nature. The perspective of cognitive restraint posits that a high level of restraint is indicative of an individual forming rigid dietary rules due to concerns about shape or weight (Herman & Polivy, 1980). In support of cognitive restraint, measures of restraint (e.g., Restraint Scale) appear to be associated with the utilization of rigid dietary rules (Herman & Polivy, 1980) but have not been related to biological factors such as acute or long-term caloric intake (Stice, Cooper, Schoeller, Tappe, & Lowe, 2007; Stice, Fisher, & Lowe, 2004; Stice, Sysko, Roberto, & Allison, 2010). Further, restrained eaters may exhibit increased food consumption following challenges to their cognition, such as a high-calorie preload (Polivy, 1976; Spencer & Fremouw, 1979) or tasks that engage cognitive resources (Bellisle & Dalix, 2001; Lowe & Kral, 2006; Ward & Mann, 2000).

More recently, restraint has been re-conceptualized by some as a reflection of hedonic hunger, or a perceived, cognitive deprivation for highly palatable foods despite meeting biological caloric needs (Lowe & Butryn, 2007; Lowe & Levine, 2005; Markowitz, Butryn, & Lowe, 2008). Hedonic hunger, as measured by the Power of Food Scale (PFS), is related to increased food craving, and greater consumption of highly processed food (e.g., chocolate) (Lowe & Butryn, 2007). Scores on the PFS are higher in obese individuals with BED (relative to those without the diagnosis) and higher PFS scores are associated with binge eating severity in BN (Lowe & Butryn, 2007; Witt & Lowe, 2014). Although scores on various restraint measures do not appear to be related to objectively measured caloric deprivation (Stice et al., 2007; Stice et al., 2004; Stice et al., 2010), restraint is associated with increased hedonic hunger (Lowe et al., 2009). Thus, dietary restraint may reflect, in part, perceived deprivation for highly palatable foods associated with hedonic hunger.

Dieting may also influence eating behavior and be associated with disordered eating. While restraint appears to reflect a cognitive, perceived deprivation of highly palatable foods, dieting – in contrast - is associated with decreased caloric intake for weight loss (Allen, Byrne, & McLean, 2012). Dieting prospectively predicts future weight gain (Lowe et al., 2006; Lowe, Doshi, Katterman, & Feig, 2013; Stice, Cameron, Killen, Hayward, & Taylor, 1999) and may be a proxy for an individual's vulnerability for problematic eating behavior and past problems with eating regulation (Lowe, 2015). Individuals who diet for weight loss tend to endorse high levels of restrained eating, but the behavior of restrained eaters may vary based on whether the individual is currently dieting (Lowe, 1993, 1995). For example, restrained dieters reduce their food intake following a high-calorie preload (Lowe, 1995), which may be a response to a direct threat to weight-loss efforts. This contrasts restrained non-dieters who overeat after a high-calorie preload, perhaps due to a violation of rigid dietary rules (Polivy, 1976; Spencer & Fremouw, 1979).

Dieting for weight loss appears to be a relevant factor in BED, and data from retrospective self-report suggest that individuals with BED have histories of frequent dieting attempts and weight fluctuations although importantly dieting appears to precede binge eating in only about 50% of those who develop BED (Grilo & Masheb, 2000; Hilbert

et al., 2014; Mussell et al., 1995; Reas & Grilo, 2007; Spurrell, Wilfley, Tanofsky, & Brownell, 1997). Fasting seems to predict binge eating across diverse weight and disordered eating groups (Stice, Davis, Miller, & Marti, 2008) and caloric restriction is associated with increased brain activity in regions associated with attention and reward processing in response to food cues (Stice et al., 2013a, 2013b).

While dieting may be a risk factor for binge eating in some individuals, binge eating precedes dieting in others (Grilo & Masheb, 2000; Hilbert et al., 2014; Reas & Grilo, 2007; Spurrell et al., 1997). In BED, those who report bingeing before dieting may have an earlier onset of binge behaviors and endorse lower levels of restraint than those who diet first (Spurrell et al., 1997). Although retrospective/prospective studies and CBT models suggest that dieting and restraint increase the risk of binge eating and eating disorders for some individuals (Fairburn, Cooper, & Shafran, 2003; Grilo & Masheb, 2000; Herman & Polivy, 1990; Spurrell et al., 1997), controlled intervention studies have challenged this idea in some respects for certain patient groups. Specifically, behavioral weight loss treatments, which decrease caloric intake, have been shown to effectively reduce binge eating behavior and eating disorder pathology in obese patients with BED (Grilo, Masheb, Wilson, Gueorguieva, & White, 2011; Wilson, Wilfley, Agras, & Bryson, 2010). Further, a carefully designed study demonstrated that a low-calorie diet (e.g., 1000 kcal/day) coupled with behavioral therapy does not appear to induce increased binge eating behavior (Wadden et al., 2004). Thus, in at least some circumstances, behavioral weight loss and low-calorie diets do not appear to increase the frequency of binge eating episodes or worsen BED symptoms.

An alternative understanding of restraint and dieting behaviors is that they are strategies utilized to overcome one's personal vulnerability for weight gain and obesity (Agras, 2010; Lowe, 2015; Lowe et al., 2013). Thus, some individuals may be vulnerable to shape and weight ideals and engage in dietary restraint to maintain their weight (Lowe & Levine, 2005). Consistent with this idea, weight concern/importance may prospectively predict the onset of dieting and disordered eating behavior (Loth, MacLehose, Bucchianeri, Crow, & Neumark-Sztainer, 2014). However, other individuals may be vulnerable to a food's hedonic properties and employ dietary restraint to counteract the rewarding nature of the food (Lowe & Butryn, 2007). Notably, individuals with BED exhibit lower levels of restraint than persons with BN, but binge-eating frequency is similar (Grilo et al., 2009; Wilfley, Schwartz, Spurrell, & Fairburn, 2000), which suggests that are mechanisms other than restraint contributing to BED.

Multiple pathways likely interact to result in the development of binge eating. Consistent with this idea, one prospective study demonstrated that body dissatisfaction interacts differently with depressive symptoms and dieting to increase the risk of disordered eating (Stice, Marti, & Durant, 2011). Further, models of binge eating that include both dieting and affect-related difficulties as contributors to bingeing behavior seem to most appropriately represent potential pathways to the development of BED (Allen et al., 2012). Thus, dietary restraint may be one contributing component to binge eating in combination with multiple other risk factors. Addiction-related mechanisms may be another relevant factor that interacts with dietary restraint to increase risk for BED.

Though dietary restraint as a putative underlying, causal mechanism for BED is unique to eating disorder models, restraint is not absent in addiction. Notably, the restraint behaviors in BED are not successful (Howard & Porzelius, 1999), which parallels the substance dependence criteria of being unable to cut down or abstain from use, despite the desire to do so (American Psychiatric Association, 2000). Repeated, unsuccessful attempts to cut back on consumption or quit occur as part of an addiction (Best, Ghufran, Day, Ray, & Loaring, 2008; Fagan et al., 2007), and substance-related restraint, or attempts to cut down, has been measured in addictive disorders (Collins & Lapp, 1992; Ruderman & McKirnan, 1984). Further, the abstinence violation effect, where an individual excessively uses a substance after experiencing a lapse in restraint, is often applied to BED (Grilo & Shiffman, 1994), but originated from the addiction literature (Marlatt, 1979). Thus, for some persons with BED, dietary restraint may be a consequence, rather than a cause, of binge eating behavior (Grilo & Masheb, 2000; Reas & Grilo, 2007). For some individuals, restraint may be the failed attempt to control addictive-like consumption of highly rewarding, calorie-dense foods.

Dietary restraint may be a strategy for overcoming one's personal vulnerability for weight gain in an obesogenic environment (Lowe, 2015; Lowe et al., 2013). While some individuals may be vulnerable to shape and weight ideals, others may have a propensity to develop an addictive-like response to hedonically pleasurable, highly processed foods and engage in dietary restraint after bingeing in an effort to avoid weight gain. Since restraint may be associated with perceived deprivation of these foods (Lowe & Levine, 2005), individuals who find these foods especially rewarding or "addictive" may experience hedonic deprivation more acutely, which may increase their risk for engaging in extreme dietary restraint to combat binge eating and weight cycling. This pattern of alternating between periods of binge eating and dietary restraint may lead to addiction-like changes in the brain, such as sensitization of the dopamine system (Avena et al., 2008b; Hyman, Malenka, & Nestler, 2006; Robinson & Berridge, 1993). Thus, for some individuals, dietary restraint may be used as an attempt to overcome weight gain related to a propensity for developing addictive-like eating in response to highly processed foods.

Relatedly, YFAS symptoms of "food addiction" have been associated with greater weight cycling (e.g., repeated loss and regain of twenty pounds or more), earlier age of first diet, and time spent dieting (Gearhardt, Boswell, & White, 2014). While some studies have found an association between YFAS symptomology and a measure of dietary restraint, others have not (Gearhardt, Boswell, & White, 2014; Gearhardt et al., 2013b; Gearhardt et al., 2012). In some cases, "food addiction" appears to be related to dieting and restraint, though the nature of this relationship is poorly understood, as the existing studies are cross-sectional or retrospective. Thus, it will be necessary to examine whether dieting and restraint may play some causal role in the development of "food addiction" or whether dietary restraint/dieting might develop in response to an individual's propensity to consume highly processed foods in an addictive manner. Further, future work should evaluate whether alternating between periods of restriction and excess consumption of highly processed foods may create an intermittent pattern that, akin to substance use disorders (Berridge & Robinson, 1995; Robinson & Berridge, 2001), exacerbates addictive-like responses.

In summary, restraint and dieting may be causal mechanisms for some individuals with BED, as posited by traditional eating disorder models. In others, restraint may be linked with binge eating, but it may reflect attempts to resist biologic drives towards obesity (Agras, 2010; Lowe, 2015; Lowe et al., 2013) and/or attempts to manage an addictive response to highly processed foods. Additional longitudinal research is needed to understand how an individual's personal vulnerabilities (e.g., shape/weight concern, propensity for addiction) may interact with binge eating to result in disordered eating behavior. Future studies might also examine whether periods of intermittent restraint or fasting (followed by episodes of binging) may contribute to addiction-related neural sensitization in humans.

5.2. Shape and weight concerns

In addition to dietary restraint, shape and weight concerns may be antecedents or risk factors for BED (Fairburn et al., 1998; Killen et al., 1996). This refers to the overvaluation of an individual's own shape and/or weight (Hrabosky, Masheb, White, & Grilo, 2007). Though not a DSM-5 diagnostic criterion for BED, it has been suggested that shape and weight concerns represent a core psychopathology of eating disorders and may play a key role in the maintenance of binge eating and associated eating pathology (Fairburn et al., 2003). Overvaluation of shape and weight may prospectively predict the onset of binge eating for some

individuals (Allen, Byrne, McLean, & Davis, 2008; Killen et al., 1994; Loth et al., 2014; Sonneville et al., 2015; Stice, Presnell, & Spangler, 2002). Additionally, high reports of body dissatisfaction appear to greatly increase the risk for disordered eating behavior (Stice et al., 2011), especially in early adolescence (Rohde, Stice, & Marti, 2015). While shape and weight concerns may precede eating problems for some, others may experience elevated shape and weight concerns due to negative outcomes such as weight gain associated with bingeing. Binge eaters who report shape and weight overvaluation present with more severe eating psychopathology and depression than binge eaters without the characteristic (Grilo, 2013; Grilo et al., 2008; Grilo, Masheb, & White, 2010; Sonneville et al., 2015). This underscores the idea that shape and weight concern is an important contributor to the trajectory of BED, though, akin to restraint, it may be a causal mechanism for some individuals but a consequence of bingeing for others.

The idea that shape and weight concerns may precipitate and/or maintain problematic eating is not characteristic of an addiction perspective. If one were to attempt to conceptualize overvaluation of shape/weight from an addiction perspective, it might perhaps be speculated that such shape and weight concerns develop as problematic eating continues. For example, it is logical that smokers would have higher rates of concern about lung cancer because they are at higher risk due to their addiction. Similarly, "food addiction" has been associated with elevated shape and weight concerns in cross-sectional research (Gearhardt, Boswell, & White, 2014; Gearhardt et al., 2013b; Gearhardt et al., 2012; Schebendach et al., 2013), which may be related to the increased risk of weight gain linked to greater levels of highly processed food consumption. In a parallel manner, from such a perspective, binge eating might be associated with greater shape and weight concerns because binge eating increases risk of future weight gain or obesity. However, studies with BED (Grilo et al., 2008) and other eating disorder groups with binge-eating behaviors (Grilo et al., 2009), have consistently demonstrated that overvaluation is not associated with either BMI or obesity (i.e., it appears to reflect a subjective cognitive feature unrelated to body weight). Thus, shape and weight concerns appear to be related to both binge eating and YFAS "food addiction" symptomology, however, longitudinal and experimental studies are needed to develop an improved understanding of these associations.

6. Practical implications

If addiction mechanisms may contribute to problematic eating behavior for some individuals, this framework may inform new treatments or improve existing interventions. While research has identified specific psychological (Wilson, Grilo, & Vitousek, 2007) and pharmacological (Reas & Grilo, 2014, 2015) treatment interventions for BED, even with the best available treatments (Grilo et al., 2011; Wilson et al., 2010), a sizeable minority of patients do not achieve remission and most fail to lose clinically meaningful weight. (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007; Grilo et al., 2011; Vocks et al., 2010; Wilson et al., 2010), Treatment can be improved further by an improved understanding of predictors/moderators (Grilo, Masheb, & Crosby, 2012) and of mediators (Kraemer, Wilson, Fairburn, & Agras, 2002; Wilson et al., 2007). Research on mediators of BED treatments is lacking and represents a priority for research. Novel treatments that target underlying mechanisms may increase efficacy. For example, if an intervention designed to reduce smoking focused on changing the individual's thoughts and beliefs about cigarettes, but did not address the cigarettes' addictive nature, this therapeutic technique would likely have limited success.

Future research is warranted to determine whether interventions that target addictive-like mechanisms may be beneficial for some individuals with problematic eating behavior. Notably, CBT for BED and addictive disorders already share common features, such as emotion regulation, trigger identification, and craving management (Gearhardt et al., 2011d). The greatest point of contention between existing treatments tailored to "food addiction" (e.g., Overeaters Anonymous) relative to treatment addressing traditional eating disorder tenets (e.g., restraint) is the role of the food (Gearhardt et al., 2011d). In eating disorder models, there are no "bad" or "forbidden" foods (Fairburn et al., 2003). However, an addiction perspective would suggest that some foods have a greater "addictive potential" and for some individuals, these foods may be capable of "hijacking" neural circuitry (Gearhardt, Davis et al., 2011) and making it more difficult to regulate eating behaviors and patterns. If an addictive process contributes to problematic eating behavior for some individuals (or if highly processed foods create greater challenges to controlled intake for both cognitive and neurobiological reasons), then perhaps specific interventions utilized in the treatment of addictive disorders should be evaluated for their utility in addressing "food addiction." The assessment of novel treatment approaches may be clinically useful for improving outcomes, though research is also needed to identify the potential harm of incorporating addiction intervention perspectives into the treatment of eating problems.

One major concern regarding treatments for "food addiction" is that the goal of abstinence from certain foods may be harmful and increase disordered eating behaviors, such as dietary restraint (Wilson, 1993). Although abstinence-based interventions for problematic eating behavior, such as Overeaters Anonymous and Food Addicts Anonymous, have been around since the 1960's, there has been limited empirical examination of their efficacy (Schwartz & Brownell, 1995). Due to the potential harm of abstinence-based programs, future research is needed to examine the utility of this treatment approach as applied to "food addiction." Until there is significant empirical evidence of positive treatment outcomes, the identification of potential contraindications, and the assessment of possible adverse consequences, abstinence-based treatments for eating should be approached with caution.

However, there are empirically supported, addiction-focused interventions that do not require abstinence, such as harm-reduction. Harm-reduction aims to minimize potential harm associated with addictive substances by focusing on using in moderation and reducing consumption in high-risk situations (Cheung, 2000; Marlatt, 1996; Marlatt & Tapert, 1993). For example, a harm-reduction intervention for individuals with alcohol-use disorders would aim to reduce binge drinking episodes through strategies such as alternating between alcoholic beverages and glasses of water and monitoring the quantity of alcoholic drinks consumed (Marlatt, Somers, & Tapert, 1993; Marlatt & Witkiewitz, 2002). Harm-reduction approaches also focus on tolerating craving and developing alternative coping strategies to regulate intense mood states (Marlatt, Larimer, & Witkiewitz, 2011). While harm-reduction strategies are empirically supported for reducing negative outcomes associated with substance-use disorders (Langendam, van Brussel, Coutinho, & van Ameijden, 2001; McBride, Farringdon, Midford, Meuleners, & Phillips, 2004; Monti et al., 1999), it is uncertain whether this intervention would be well-suited for addictive-like consumption of highly processed foods. Thus, future examination of a harm-reduction approach for treating "food addiction" may be warranted.

Another consideration for the utility of "food addiction" in a clinical context is whether the construct predicts treatment outcomes. We are unaware of previous studies that have examined if addiction-specific treatments like harm reduction or abstinence-based interventions are effective in reducing symptoms of "food addiction" or eating-related problems like binge eating and obesity. However, there have been mixed findings regarding whether an individual's endorsement of "food addiction" symptoms predicts outcomes in treatments for obesity, with some studies finding that "food addiction" was related to less weight loss (Burmeister, Hinman, Koball, Hoffmann, & Carels, 2013) and others observing no association with weight loss or attrition (Lent, Eichen, Goldbacher, Wadden, & Foster, 2014).

The literature regarding the predictive significance of BED for obesity treatment outcomes is mixed and suffers from methodological limitations, consisting of mostly retrospective and non-randomized studies,

limited further by self-report measures and varying ways of categorizing "binge eaters" (Wilson et al., 2007). Blaine and Rodman (2007), in a matched-study meta-analysis of obesity trials, reported that obese binge eaters, on average, lost significantly less weight than obese non-binge-eaters (1.3 kg vs 10.5 kg). Such aggregate findings across studies suggest that BED is a negative prognostic indicator for obesity treatment, although the comparison strategy and the methodological limitations of many of the studies suggests caution and need for further research. More recently, Grilo and White (2013), in the first controlled, prospective study of BED status on obesity outcome treatment, reported that BED significantly predicted worse eating and depression outcomes and significantly moderated the effects of anti-obesity medication (orlistat) on weight loss (i.e., medication added to behavioral therapy enhanced weight losses among non-binge-eaters but not BED).

Given the mixed findings in the literature, further examination is warranted for how constructs as "food addiction" and binge eating may predict intervention outcomes for obesity. However, obesity is not synonymous with "food addiction" or binge eating, and future research should also focus on identifying which treatments may be most effective for particular eating-related problems (e.g., harm reduction for the treatment of "food addiction") and whether individual characteristics (e.g., impulsivity) may moderate treatment outcome.

7. Future directions

Given the unique mechanisms relevant to applying an addiction framework to food (e.g., withdrawal), there are six key research directions that are important to further evaluate the "food addiction" construct (Table 2). First, it is essential to identify the addictive agent in food to determine which foods or ingredient(s) have an addictive potential. Though animal models and the only existing study in humans support the role of highly processed foods in "food addiction" (Avena et al., 2008b; Johnson & Kenny, 2010b; Murray, Tulloch, Chen, & Avena, 2015; Schulte et al., 2015), the addictive agent(s) is unknown, as are the specific individual characteristics that may enhance one's risk of developing "food addiction." The absence of a defined addictive agent remains one of the most significant criticisms of the food addiction hypothesis (Hebebrand et al., 2014; Ziauddeen & Fletcher, 2013). The identification of the potentially addictive agent in food is fundamental to the proposal of an addictive process and should be prioritized in future studies. Second, it is important to determine whether mechanisms unique to addictive disorders, such as withdrawal and tolerance, may contribute to addictive-like consumption of certain foods. Third, longitudinal work is needed in order to understand the temporal relations between "food addiction," obesity, binge eating, and BED. The YFAS-C, a measure of "food addiction" symptoms in children, is associated with elevated BMI (Gearhardt, Roberto, Seamans, Corbin, & Brownell, 2013), emphasizing the necessity of examining how "food addiction" develops and relates to other eating-related problems across the lifespan. Further, longitudinal methodologies used in other fields (Cain et al., 2012; Shea et al., 2004) may help to clarify further the nature of the associations among the constructs and especially whether the possible mechanisms described in this review are causal factors or contributors to the maintenance or course of eating-related problems. Fourth, treatment approaches for addictive disorders, such as abstinence-based interventions or harm-reduction, should be evaluated for the treatment of "food addiction." This is especially important given the potential for certain addiction-focused treatment approaches to increase restrictive eating practices. Fifth, there is emerging evidence that YFAS scores may be associated with other forms of binge eating, such as bulimia nervosa (Meule, von Rezori, & Blechert, 2014) and anorexia nervosa- binge/purge subtype (Granero et al., 2014). As such, the role of addiction-specific mechanisms in problematic eating behavior should be explored across various forms of binge eating and disordered eating in future research. Sixth, BED and "food addiction" are both associated with obesity but do not occur exclusively in obese individuals (Davis et al., 2011; de Zwaan, 2001; Gearhardt et al., 2009b). However, many of the mechanisms implicated in addictive disorders and BED (e.g., impulsivity, reward dysfunction) may also contribute to obesity (Davis & Carter, 2009; Volkow et al., 2008b). Thus, it will be important to examine whether certain mechanisms are particularly relevant to individuals with both obesity and compulsive patterns of food consumption.

Table 2
Future directions in food addiction research.

1. Identify the addictive agent(s) in food to determine which foods or ingredient(s) have an addictive potential
2. Determine whether mechanisms unique to addictive disorders, such as withdrawal and tolerance, may contribute to addictive-like consumption of certain foods
3. Utilize longitudinal research approaches to a) examine temporal relations between "food addiction," obesity, binge eating, and BED, and b determine whether the mechanisms described in this review may be causal factors or contributors to the maintenance of eating-related problems
4. Evaluate whether treatment approaches for addictive disorders, such as abstinence-based interventions or harm-reduction, may be appropriate and efficacious for the treatment of "food addiction"
5. Explore whether addiction-specific mechanisms may contribute to eating-related problems more broadly (e.g., binge eating, bulimia nervosa)
6. Examine whether certain mechanisms implicated in BED and "food addiction" are particularly relevant to individuals with both obesity and compulsive patterns of food consumption

8. Concluding remarks

"Food addiction" and BED have significant phenotypic overlap, such as loss of control over consumption and continued use despite negative consequences. To evaluate whether an addictive process contributes to problematic eating, increased attention to underlying mechanisms is warranted. While many mechanisms appear to be shared among BED and addictive disorders, such as reward dysfunction, craving, emotion dysregulation, and impulsivity, there are also important differences. For some individuals, dietary restraint and shape or weight concerns may be causal mechanisms in the development and/or maintenance of binge eating behavior. However, for others, highly processed foods may be capable of triggering neuroplastic changes in the brain to result in an addictive-like process. In conclusion, though "food addiction" and BED share several underlying mechanisms, the utility of "food addiction" may be most appropriately understood by investigating whether mechanisms unique to an addiction perspective offer novel explanatory power for problematic eating in some individuals. Further, a mechanistic approach may contribute to the development of new intervention approaches to improve treatment outcomes.

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