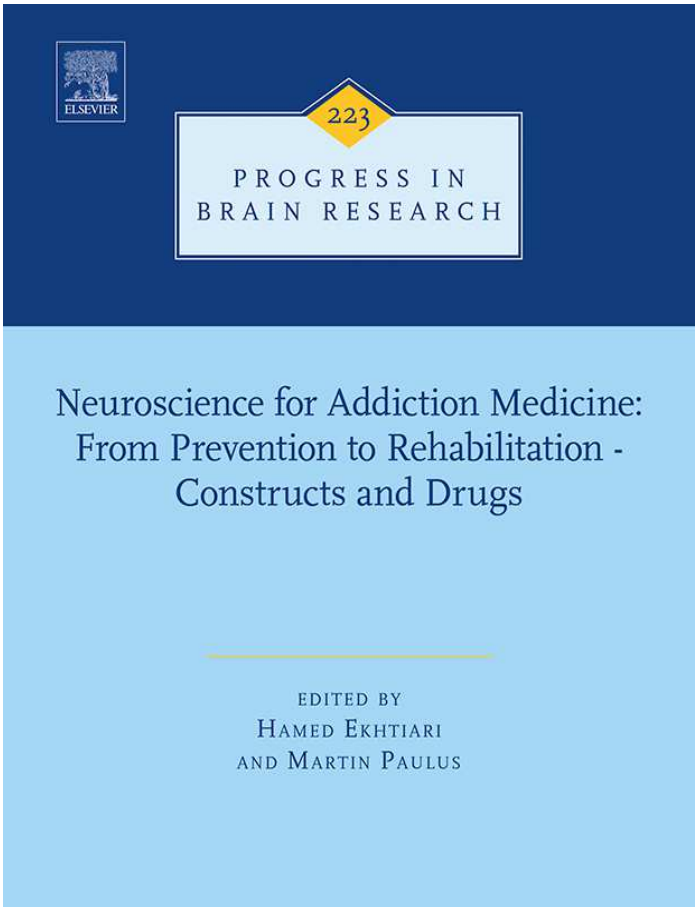

From Erica M. Schulte, Sonja Yokum, Marc N. Potenza and Ashley N. Gearhardt, Neural systems implicated in obesity as an addictive disorder: from biological to behavioral mechanisms. In: Hamed Ekhtiari and Martin Paulus, editors, *Progress in Brain Research, Vol. 223*, Amsterdam: Elsevier, 2016, pp. 329-346.
ISBN: 978-0-444-63545-7

CHAPTER

17 Neural systems implicated in obesity as an addictive disorder: from biological to behavioral mechanisms

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Abstract

Contributing factors to obesity have been identified, yet prevention and treatment efforts have had limited long-term success. It has recently been suggested that some individuals may experience an addictive-like response to certain foods, such as losing control over consumption and continued consumption despite negative consequences. In support, shared biological and behavioral features seem to exist between "food addiction" and traditional substance-use disorders. "Food addiction" may be another important contributor to obesity. The current chapter reviews existing literature regarding neural systems implicated similarly in obesity and addiction, discusses unique considerations for addictive-like eating, and proposes directions for future research regarding "food addiction" as an emerging construct for addiction medicine.

Keywords

Obesity, Addiction, Substance dependence, Food addiction, Reward

1 INTRODUCTION

As obesity rates continue to rise, increased attention has been given to mechanisms associated with overeating behaviors. It has been proposed that an addictive-like

Progress in Brain Research, Volume 223, ISSN 0079-6123, http://dx.doi.org/10.1016/bs.pbr.2015.07.011

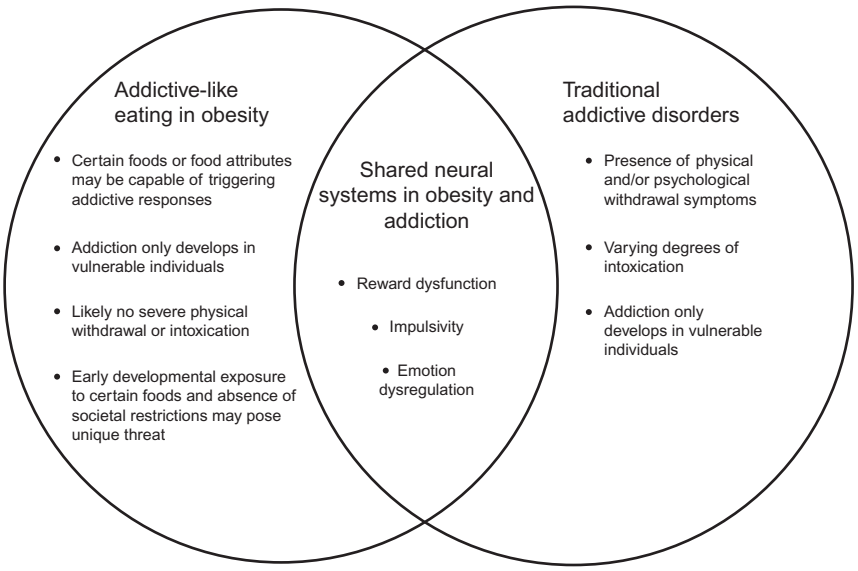

FIGURE 1

Shared neural systems in obesity and addiction and unique features for addictive-like eating and traditional addictive disorders.

process may underlie problematic eating for some individuals (Gearhardt et al., 2009b), although this point has been debated (Ziauddeen et al., 2012). Significant behavioral overlap exists between obesity and addictive disorders (particularly for some groups like those with Binge Eating Disorder; Gearhardt et al., 2011b), such as a loss of control over consumption and continued consumption despite negative consequences (Gearhardt et al., 2009a). The following chapter will review literature regarding shared neural systems in traditional addictive disorders and obesity (see Fig. 1), discuss differentiating factors of addictive-like eating, and offer essential next steps in neuroimaging research for "food addiction."

2 SHARED NEURAL SYSTEMS: REWARD DYSFUNCTION

Dopamine (DA) is a main catecholamine neurotransmitter implicated in reinforcement- and reward-related processes, such as motivation and craving. Food and drugs of abuse both increase DA signaling in the mesolimbic dopaminergic system (Heinz et al., 2004; Wang et al., 2002). Consumption of high-sugar or high-fat food results in DA release in the striatum in animals (Avena et al., 2009) and humans, with the amount released correlating with meal pleasantness ratings (Small et al., 2003) and energy density (Ferreira et al., 2012). In humans, consumption of palatable food is associated with increased activation in the reward-related circuitry, including the dorsal- and ventral striatum and orbitofrontal cortex (OFC; Stice et al., 2013a).

Likewise, all addictive drugs lead to DA release in the striatum and associated mesolimbic regions (Kalivas and O'Brien, 2008).

Utilizing functional magnetic resonance imaging (fMRI), previous research has observed parallels in neural responsivity to food/drug cues and intake between obesity and substance-use disorders (Tang et al., 2012). Obese versus lean humans show greater responsivity of brain regions associated with reward (e.g., striatum, amygdala, OFC) and attention (e.g., anterior cingulate cortex, ACC) to pictures of high-fat/sugar foods (versus control stimuli; Martin et al., 2010; Stice et al., 2010b) and to pictorial cues that signal impending palatable food receipt (Ng et al., 2011; Stice et al., 2008). Similarly, humans with, versus without, substance-use disorders show greater activation of reward regions (e.g., VTA, amygdala) and attention regions (e.g., ACC) to drug-related cues (Due et al., 2002; Myrick et al., 2004).

One distinct feature of addictive disorders is the transition from initially consuming drugs of abuse for their reinforcing properties to compulsive, habitual self-administration (Everitt and Robbins, 2005). Consuming drugs of abuse or highly palatable foods for hedonic effects (liking) activates the ventral striatum, whereas habitual, compulsive self-administration (wanting) appears to differentially implicate dorsal striatal regions (Everitt and Robbins, 2005; Volkow et al., 2006).

One proposed mechanism underlying the transition from "liking" to "wanting" is incentive sensitization (Robinson and Berridge, 1993). This explanation suggests that chronic consumption of addictive substances or highly palatable foods may result in sensitization of the DA system and increased salience of drug- and food-specific cues for some individuals (Berridge, 2009). In support, animal experiments indicate that firing of striatal and ventral pallidal DA neurons initially occurs in response to receipt of a novel palatable food, but that after repeated pairings of palatable food intake and cues that signal impending receipt of that food, DA neurons begin to fire in response to reward-predictive cues and no longer fire in response to food receipt (Tobler et al., 2005). In humans, midbrain and medial OFC activity in response to milkshake receipt positively correlated with subsequent *ad libitum* milkshake consumption, and BOLD response in the ventral striatum during exposure to food images positively correlated with later snack consumption (Lawrence et al., 2012; Nolan-Poupart et al., 2013). Healthy weight adolescents who were eating beyond basal metabolic needs (per objective measures) versus those who were not showed greater BOLD response during cues predicting impending palatable food receipt in regions that encode reward (striatum), salience (precuneus), and visual processing and attention (visual and anterior cingulate cortices; Burger and Stice, 2013). These latter data suggest that overeating, even if it has not yet resulted in excess weight gain, may be accompanied by elevated responsivity to food-predictive cues in reward and attentional regions. For persons susceptible to these neuroplastic changes, sensitization to relevant cues can trigger "wanting" and potentially lead to addictive-like consumption.

Reactivity in brain regions associated with reward appraisal like the striatum, amygdala, and OFC may be indicative of the incentive salience for drug and food

cues, where increased salience is associated with propensities for obesity and unsuccessful abstinence in addictive disorders (Tang et al., 2012). In support, elevated striatal response to monetary reward related prospectively to substance-use onset over a 1-year follow-up (Stice et al., 2013b) and elevated responsivity of reward regions (striatum, amygdala, OFC) to palatable food images (Demos et al., 2012), palatable food commercials (Yokum et al., 2014), cues that predict palatable food image presentation (Yokum et al., 2011), and palatable food receipt (Geha et al., 2013) related prospectively to future weight gain. Further, individual differences observed in animal models provide insight to certain characteristics that may predict who will sensitize to food cues. Individuals who exhibit greater motivation to engage with cues that predict a drug or food reward, an indication of increased incentive salience (so-called sign trackers), than to elements of reward receipt like the location of reward delivery (so-called goal trackers) appear to be at greater risk for sensitization (Flagel et al., 2009). Thus, while sensitization appears to contribute to continued overconsumption of palatable foods, greater engagement with food-predictive cues may predict which individuals will sensitize.

It has also been hypothesized that a reward deficiency may predispose some individuals to develop compulsive overeating or drug-taking behavior (Blum et al., 2014). In some forms of obesity and addictive disorders, individuals may be motivated to consume highly palatable food or drugs of abuse to compensate for diminished DA receptor availability (Koob and Le Moal, 2001). Consistent with the reward deficiency theory, adults with versus without alcohol, cocaine, heroin, and methamphetamine dependence show reduced striatal D2-like receptor availability and sensitivity (Volkow et al., 2001; Wang et al., 1997) and lower D2-like receptor density may increase risk for relapse after treatment (Heinz et al., 2004). Further, low striatal D2-like receptor availability in primates relates prospectively to increased future drug self-administration (Nader et al., 2006). Likewise, obese versus lean adults show lower striatal DA D2-like receptor availability (de Weijer et al., 2011; Volkow et al., 2008), although two other studies found no significant group differences (Eisenstein et al., 2013; Haltia et al., 2007), with differences across studies possibly attributable to small sample sizes, differences in degrees of obesity, different radioligands used, or other factors. Obese versus lean adults show lower capacity of nigrostriatal neurons to synthesize DA (Wilcox et al., 2009) and less striatal responsivity to tastes of high-fat/sugar beverages (Babbs et al., 2013; Stice et al., 2008). Obese versus lean rats likewise have lower basal DA levels and D2-like receptor availability and less *ex vivo* DA release in response to electrical stimulation in the nucleus accumbens and dorsal striatum (Geiger et al., 2009; Thanos et al., 2008).

It is unknown whether hypoactivation in reward circuitry in response to acute administration may be a cause or consequence in either addictive disorders or obesity as prolonged overconsumption of drugs and rewarding food may decrease striatal DA availability. Animal experiments show that regular substance use reduces striatal D2-like receptors (Nader et al., 2006) and sensitivity of reward circuitry (Kenny et al., 2006), and humans with cocaine abuse show blunted DA release in response

to stimulant drugs relative to nonaddicted comparison subjects (Volkow et al., 2005) and tolerance to the euphoric effects of cocaine (O'Brien et al., 2006). Chronic cocaine use has been associated with downregulated dopaminergic responses to both cocaine and food cues (Tomasi et al., 2015). These data imply that substance use contributes to the downregulated reward circuitry observed in the cross-sectional studies. Similarly, prospective human (Stice et al., 2010a) and experimental animal studies (Geiger et al., 2009; Johnson and Kenny, 2010; Thanos et al., 2008) indicate that overeating may contribute to reward region hyporesponsivity during food consumption. Further, studies have suggested that individuals vulnerable to substance use (Stice et al., 2013b) and obesity (Stice et al., 2011; Verbeken et al., 2012) may initially exhibit hyperresponsiveness in reward-related brain regions to rewards in general. This hyperresponsiveness may increase motivation to seek out highly rewarding, palatable foods and these individuals may appear to be hyporesponsive to reward after neuroplastic changes associated with chronic overeating behavior have occurred.

3 SHARED NEURAL SYSTEMS: IMPULSIVITY

Another domain implicated in both obesity and addictive disorders is an executive-control deficiency, often evident by impulsive behavior. Obese individuals and persons with addictions appear to favor short-term rewards of food or drug instead of long-term health benefits (e.g., weight reduction in obesity; Mole et al., 2014). In decision-making tasks, obese women not only make more impulsive decisions than healthy women (Davis et al., 2010), but also exhibit decreased activation in executive-control brain regions (e.g., middle frontal gyri, medial prefrontal cortex [PFC]) during decision-making processes (Kishinevsky et al., 2012; Stoeckel et al., 2013). Similar patterns of impulsive decision-making coupled with diminished activation in executive-control regions have also been observed in addictive disorders (MacKillop et al., 2011), and decreased inhibitory-control activation may be predictive of relapse (Paulus et al., 2005). Similarly, Kishinevsky and colleagues (2012) observed that diminished activity in executive-control structures related to future weight gain.

One proposed explanation for this potentially maladaptive decision-making process is abnormal striato-cortical connectivity similarly observed in addictive disorders (Hanlon et al., 2011; Liu et al., 2009) and obesity (Garcia-Garcia et al., 2013; Tomasi and Volkow, 2013). In this dysfunctional connection, brain structures implicated in reward appraisal (e.g., OFC, ventromedial PFC) may be capable of overwhelming inhibitory-control regions (e.g., dorsolateral PFC) to result in impulsive decision-making (Weygandt et al., 2013; Zhang et al., 2015). Additionally, recent studies observed lower brain volumes in the OFC in obese women (Shott et al., 2014) and a lack of synchronicity between the OFC and PFC in fasted obese men (Zhang et al., 2015), providing further evidence that diminished inhibitory control may lead to overeating in obesity (Zhang et al., 2015) in a manner akin to compulsive

drug-taking in addictive disorders (Ma et al., 2010). For some obese individuals, food cues may activate regions associated with reward salience, and the executive-control neural system may be inefficient at suppressing the drive to seek certain foods. Though neuroimaging research should continue to examine deficits in striato-cortical connectivity in obesity, it appears likely that deficits in executive-control neural circuitry may similarly contribute to impulsive decision-making in obesity and addiction.

4 SHARED NEURAL SYSTEMS: EMOTION DYSREGULATION

In both addiction and obesity, neural systems underlying emotion-regulation processes seem to be impaired. Strong emotional states frequently precipitate drug use and overeating behavior, which may suggest that some individuals utilize addictive substances and highly palatable foods to compensate for deficient emotion-regulation processes (Singh, 2014; Sinha and Jastreboff, 2013). Notably, existing research has observed that humans typically consume foods high in fat and/or refined carbohydrates in response to emotional states like stress or negative affect, which may be particularly relevant for the development of obesity (Morris et al., 2014).

Similar to addictive disorders, negative affect appears to implicate neural systems associated with increased craving and compulsive consumption in obesity (Sinha and Jastreboff, 2013). Jastreboff and colleagues (2013) found that obese, compared to lean, individuals exhibited greater activity in striatal regions when exposed to stress and highly palatable food cues, relative to neutral-relaxing cues, and increased activation of the dorsal striatum in response to stress and food cues was related to stronger food cravings. This suggests that emotional states may activate brain regions associated with habitual behavior, like the dorsal striatum, which motivates certain obese individuals to consume highly palatable foods (Jastreboff et al., 2013). Coupled with increased activation in reward motivation structures, Tryon et al. (2013) observed that chronic stress was associated with less prefrontal activation in response to high-calorie food cues, and this pattern of activation related to greater consumption of high-calorie foods. Collectively, it appears that emotional states may be related to neural activation subserving increased craving and diminished inhibitory control in obesity, which is also observed in addictive disorders (Sinha, 2008).

5 SUMMARY OF SHARED NEURAL SYSTEMS

Overlapping neural systems appear to be implicated in both obesity and addictive disorders, including reward dysfunction, executive-control deficiencies, and emotion regulation. The existing neuroimaging data suggests that addictive-like mechanisms may contribute to obesity for some individuals. Thus, exploration of the

"food addiction" construct may be clinically useful for understanding overeating behavior and informing intervention approaches for certain individuals with obesity.

6 DIFFERENCES BETWEEN OBESITY AND ADDICTIVE DISORDERS

While existing neuroimaging studies have observed similarities between obesity and substance-use disorders, important differences exist between "food addiction" and traditional addictive disorders. Notably, food is necessary for survival. However, many highly palatable foods are not in their natural state and have instead been processed with added amounts of potentially rewarding ingredients like fat and refined carbohydrates (Gearhardt et al., 2011a). Similar to the word "drug" which includes addictive (e.g., cocaine) and nonaddictive (e.g., aspirin) substances, future research is warranted to examine whether foods in a natural state (e.g., banana) are equally implicated in problematic, addictive-like eating behavior as highly processed foods (e.g., pizza).

Similarly, the current conceptualization of "food addiction" differs from traditional addictive disorders because an addictive agent in certain foods and the "dose" that may increase certain foods' addictive potential has not been investigated (Ziauddeen and Fletcher, 2013). For drugs of abuse, an addictive agent has been defined (e.g., ethanol in alcohol, nicotine in cigarettes) and high concentrations of those ingredients are linked to an increased addictive potential (Henningfield and Keenan, 1993). In contrast, although highly processed foods have been hypothesized to be most likely implicated in addictive-like eating due to high levels of fat and/or refined carbohydrates (Gearhardt et al., 2011a), it has not been examined whether specific food attributes (e.g., sugar content) may be capable of triggering an addictive-like response in certain individuals; however, there are preliminary data suggesting that sugar more effectively recruits reward and gustatory regions compared to fat (Stice et al., 2013a). Additionally, no previous research has evaluated whether a particular "dose" or quantity of the "addictive" food attribute would increase the abuse potential of an "addictive" food (Ziauddeen et al., 2012). For example, if future studies indicate that sugar may be implicated in "food addiction," a threshold may be set to describe the concentration of sugar that significantly elevates a food's addictive potential (e.g., 30% calories from sugar). Further, specific sugars may differentially elicit brain responses that may associate differentially with appetite and reward pathways and lead to addictive patterns of eating (Page et al., 2013). However, unlike drugs of abuse, certain foods may have multiple addictive agents, like fat and sugar, which may both increase the food's addictive potential, but perhaps through different mechanisms.

Another difference that may exist between "food addiction" and substance-use disorders is the presentation of withdrawal symptoms. Although limited, preclinical literature suggests that dieters may experience headaches and psychological preoccupation with food (Gearhardt et al., 2009a), but it is unlikely that individuals with "food addiction" would experience severe, life-threatening physiological withdrawal

symptoms if highly processed foods were removed from their diet (Ziauddeen et al., 2012). Consequently, "food addiction" may vary from traditional addictive disorders like opioid-dependence that produce acute physiological withdrawal symptoms, such as vomiting and sweating. On the other hand, removing certain foods from the diet may be more likely to trigger psychological withdrawal symptoms like anxiety. Such psychological features are experienced by a subset of individuals with pathological gambling (or gambling disorder) and are included in a withdrawal-related inclusionary criterion for the condition (American Psychiatric Association, 2013). Although withdrawal appears to manifest differently across addictive disorders, no previous research has examined whether withdrawal symptoms involving psychological states may contribute to addictive-like eating behavior.

Although "food addiction" is less likely to be associated with life-threatening withdrawal symptoms, there may be severe risks associated with "food addiction" that are not relevant in substance-use disorders. Unlike substance-use disorders, many individuals are exposed to highly processed foods within the first year of life (Fox et al., 2004). If certain foods have addictive potential, exposure to these foods during critical developmental periods may contribute to the onset of persistent, lifelong obesity for some people (Epstein et al., 1985). Unlike drugs of abuse that are illegal, expensive, or age-restricted, highly processed foods are easily accessible and affordable in our modern food environment. In this respect, "food addiction" may be considered a greater threat than substance-use disorders because there are fewer societal restrictions on the consumption of highly processed foods. Thus, while "food addiction" may not be associated with severe physiological withdrawal, it may pose a unique risk in infancy and childhood due to early first exposure that may increase its severity.

7 DIFFERENCES BETWEEN ADDICTIVE DISORDERS

Though premature acceptance of "food addiction" has been cautioned against due to inconsistencies with other addictive disorders (Ziauddeen and Fletcher, 2013; Ziauddeen et al., 2012), it is important to note differences that exist among addictive disorders included in the DSM-5 (American Psychiatric Association, 2013). Notably, the characteristics required for a substance to be considered addictive have changed over time. As previously mentioned, alcohol and opioid dependence may trigger intense physiological withdrawal symptoms (Leshner, 1997; Skinner and Allen, 1982), whereas cocaine and nicotine dependence appear more likely to produce psychological symptoms like involving anxiety and irritability (Brower and Paredes, 1987; Weddington et al., 1990). Additionally, the inclusion of the behavioral addiction gambling disorder in the DSM-5 reflects a shift away from physiological withdrawal as a necessary component of addiction (American Psychiatric Association, 2013). Importantly, regardless of whether the symptoms are physical or psychological, the experience of withdrawal appears to increase the probability of relapse across addictive disorders (Kenford et al., 2002; Ray, 1961). Thus, a key, shared component of withdrawal within addiction may involve the increased chance of relapse associated with experiencing withdrawal symptoms. In this respect, it will be important for

future research to examine whether individuals with "food addiction" experience withdrawal symptoms that may trigger addictive-like eating behavior.

Another difference that exists within addictive disorders is the experience of intoxication. Some drugs of abuse, such as heroin, may result in intoxication where the individual enters a mind-altered state and may behave recklessly or break the law (Inciardi, 1979). Other examples of heroin intoxication symptoms are unpredictable mood swings, risky behavior, and breathing problems. However, other substances like nicotine may not trigger similar intoxication symptoms and allow individuals to function while using the drug. For example, it is often legal to smoke cigarettes while at work or operating a vehicle. Although nicotine does not produce acute negative consequences due to intoxication in the same way heroin does, prolonged nicotine use often leads to long-term negative health consequences like coronary heart disease, stroke, lung cancer, and emphysema (US Surgeon General, 1982). In behavioral addictions like pathological gambling or gambling disorder, the negative consequences may not manifest in acute intoxication, but rather in long-term outcomes like loss of money and familial stress. Similarly, while certain foods are likely incapable of triggering intoxication, long-term negative consequences may be associated with chronic addictive-like eating behavior, such as obesity, heart disease, and diabetes (Bray, 2004). In summary, while addictions vary in their degree of intoxication, individuals with addictive disorders share a characteristic of continued behavioral engagement in the addictive process despite negative consequences.

If certain foods are identified as "addictive," it is unlikely that all individuals who consume these foods will develop addictive-like eating behavior. In traditional addictive disorders, a small fraction of individuals who use addictive substances or engage in addictive behaviors develop dependence (Anthony et al., 1994). Applying this logic to addictive-like eating behavior, it would follow that only a subset of individuals who are exposed to potentially "addictive" foods would later develop "food addiction." Additionally, overconsumption of "addictive" foods would likely not lead to addictive-like eating behavior for all individuals. This is similarly observed in addictive disorders, where a larger percentage of individuals overconsume drugs of abuse than develop dependence (Dawson et al., 2004; Hasin et al., 2007). Further, it is unlikely that clinically significant symptoms of addictive-like eating would occur exclusively in obesity, and symptoms have been observed across a range of body mass indexes (Gearhardt et al., 2011c). Thus, subtyping may be one fruitful avenue that emerges from examining whether an addiction perspective can be applied to problematic eating behavior for some individuals. This may increase the efficacy of treatment and prevention efforts for obesity and for disordered eating more generally.

8 FUTURE DIRECTIONS IN FOOD ADDICTION RESEARCH

One important gap in the existing literature on "food addiction" is the examination of which foods are most likely associated with addictive-like eating. An addiction framework suggests that an addictive agent in some foods would interact with individual vulnerabilities to result in "food addiction." It follows that identifying whether

certain foods or food attributes (e.g., specific sugars) may be capable of triggering an addictive-like process is essential to evaluating this perspective. Further, a drug of abuse has a greater addictive potential when a high "dose" of an addictive agent is rapidly absorbed by the system (Verebey and Gold, 1988). In our modern food environment, many highly processed foods contain added fat and/or refined carbohydrates (like white flour and sugar) in quantities exceeding what is found in naturally occurring foods. Akin to addictive disorders, the way in which these ingredients are absorbed in the body may also contribute to a food's addictive potential. Foods that have been processed by adding fat and/or refined carbohydrates while simultaneously stripping nutrients that slow digestion, like fiber and water, may have an elevated addictive potential. For example, a highly processed food, like chocolate cake, with added amounts of fat and rapidly absorbed refined carbohydrates may be expected to have a greater addictive potential than an apple, which contains natural sugars, but fiber and water to slow digestion. In highly processed foods, the increased concentration of rewarding ingredients coupled with the rapid rate that refined carbohydrates are absorbed may contribute to an increased abuse potential. In support, Schulte et al. (2015) found that highly processed foods were most likely to be associated with addictive-like eating behaviors, particularly for individuals who endorsed symptoms of "food addiction." Future studies should examine whether these highly processed foods are capable of producing neuroplastic changes in the brain, akin to those associated with consumption of drugs of abuse. Demonstrating that certain foods are capable of changing neural systems in a similar manner as addictive substances would provide further support for the validity and unique explanatory power of "food addiction" for some individuals with obesity.

A potential avenue to investigate how certain foods may change reward-related circuitry may involve examining neural mechanisms across the lifespan. Interestingly, childhood-onset obesity typically persists into adulthood (Epstein et al., 1985). Similarly substance-use disorders that emerge during early adolescence also often last throughout one's life (Chen et al., 2009). One potential explanation is that similar changes in reward mechanisms may be occurring, resulting in chronic addictive responses. While hypoactive reward-responsiveness may motivate obese individuals to seek highly rewarding food in adulthood (Blum et al., 2006), recent research has suggested that obese children actually exhibit greater functional connectivity between reward-related brain regions, including the left lateral OFC, and executive-control structures, such as the left ventromedial prefrontal cortex (Black et al., 2014). Increased input from reward neural systems to regions of cognitive control may make obese children particularly responsive to food cues (Bruce et al., 2010). However, no previous studies have examined reward-related neural mechanisms in infancy that may predispose certain individuals to become obese as children. Given the existing literature, it is uncertain whether increased functional connectivity between reward and executive-control regions may represent a cause or consequence of obesity in children. This is an essential area to explore, since children are exposed to highly rewarding food at young ages. If certain foods may be associated with addictive-like eating behaviors, it is possible that marketing

restrictions may help decrease potential risks that some children have to develop "food addiction" and persistent obesity.

Finally, applying an addiction perspective to problematic eating behavior for some obese individuals may increase treatment options and efficacy. Behavioral treatments for addictive disorders that focus on craving management and relapse prevention may be adapted for the treatment of addictive-like eating behaviors. Pharmacological approaches may also be relevant. For example, naltrexone and buproprion, which are used for the treatment of addictive disorders, also appear to be a successful intervention technique for obesity (Greenway et al., 2010). Additionally, neurobiological treatments, such as neurofeedback, have recently been explored as a treatment method for substance-use disorders. This technique uses real-time fMRI (rtfMRI) feedback to help individuals reduce cue-induced craving (Sokunbi et al., 2014). For example, individuals are first shown a substance-relevant cue (e.g., cigarette) and receive feedback about increased brain activity in regions associated with craving, like the ACC. Next, the patient is asked to reduce activity in this region and self-report craving for the substance. The existing literature suggests that neurofeedback may be an effective technique for reducing craving for addictive substances, including nicotine (Li et al., 2013) and opiates (Dehghani-Arani et al., 2013). Since craving appears to be implicated in both substance-use disorders and "food addiction," neurofeedback may be a useful treatment tool for obese individuals reporting addictive-like eating behavior.

9 CONCLUDING REMARKS

Obesity appears to share common neural systems with traditional addictive disorders, suggesting that an addictive-like process may contribute to problematic eating behavior for some obese individuals. If certain foods are identified as "addictive," treatment and prevention efforts that adopt an addiction framework would likely be efficacious for the subtype of obese individuals endorsing "food addiction." However, future research is needed to determine whether an addiction perspective is clinically useful to explain some forms of obesity. Important next directions may involve investigating if certain foods can produce neuroplastic changes in the brain, characterizing the potentially addictive agents in these foods, looking longitudinally at changes in reward mechanisms, and examining whether treatments developed for drug addiction translate to effective interventions for food addiction.

ACKNOWLEDGMENTS

This was supported by P20 DA027844, CASAColumbia and the National Center for Responsible Gaming.

Disclosures: The authors report that they have no financial conflicts of interest with respect to the content of this manuscript. Dr. Potenza has received financial support or compensation

for the following: Dr. Potenza has consulted for and advised Somaxon, Boehringer Ingelheim, Lundbeck, Ironwood, Shire, INSYS and RiverMend Health; has received research support from the National Institutes of Health, Veteran's Administration, Mohegan Sun Casino, the National Center for Responsible Gaming, and Forest Laboratories, Ortho-McNeil, Oy-Control/Biotie, Glaxo-SmithKline, Pfizer, and Psyadon pharmaceuticals; has participated in surveys, mailings or telephone consultations related to drug addiction, impulse control disorders or other health topics; has consulted for gambling entities, law offices and the federal public defender's office in issues related to impulse control disorders; provides clinical care in the Connecticut Department of Mental Health and Addiction Services Problem Gambling Services Program; has performed grant reviews for the National Institutes of Health and other agencies; has edited or guest-edited journals or journal sections; has given academic lectures in grand rounds, CME events and other clinical or scientific venues; and has generated books or book chapters for publishers of mental health texts.

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