Emerging Evidence of Addiction in Problematic Eating Behavior

ASHLEY GEARHARDT, SUSAN MURRAY, and NICOLE M. AVENA

Abstract

Obesity continues to be a pressing global health crisis with few nonsurgical means of long-term successful treatment. In addition, in the last year, binge eating disorder (BED), which shares several behavioral characteristics with traditional substance use disorders, has been recognized in the DSM-V as a distinct eating disorder diagnosis. In light of such trends, an emerging and controversial hypothesis is that an addictive response to certain types of foods may be contributing to eating-related problems. If certain individuals are experiencing an addiction to highly palatable foods, the treatment and prevention of problematic eating may need to be altered in such circumstances. Further, if certain food (or ingredients in food) are identified as having an addictive potential, policy approaches employed to reduce the public health impact of other types of addictive substances (e.g., reducing advertising to minors, taxation) may be of use in the obesity epidemic. In the following piece, we will review the research linking addiction and eating, but most importantly, we will identify directions for future research in this relatively new field of study.

FOUNDATIONAL RESEARCH

The food environment that we live in has changed dramatically over the past several decades. Ultraprocessed, highly palatable food stuffs are cheap, easily accessible and heavily marketed. The addition of higher levels of sugar has been one of the major drivers of this change, along with increased levels of fat, salt, refined flours, and food additives. Hyperpalatable food, such as ice cream, cookies, cakes and candy, exceed the level of reward associated with more natural, minimally processed food, such as vegetables, fruits, and nuts. The rising rates of obesity and recent recognition of BED (binge eating disorder) as a distinct diagnosis that have accompanied the influx of these types of food have led to the hypothesis that hyper-palatable food may be capable of triggering an addictive process.

Foods and drugs of abuse both activate the neurotransmitter dopamine, which has been implicated in reward processes. Further, increased food

Emerging Trends in the Social and Behavioral Sciences. Edited by Robert Scott and Stephen Kosslyn.
 ISBN 978-1-118-90077-2.

palatability appears to be associated with a more marked activation of the reward system (Volkow, Wang, Fowler, & Telang, 2008). Obesity and substance dependence share certain neural underpinnings, such as increased activation in motivation-related brain regions in response to cues and diminished activation in reward regions in response to consumption (Volkow *et al*., 2002, 2008). BED also shares significant overlap with addiction. The diagnostic criteria for BED and substance dependence include a number of similar characteristics, such as diminished control over consumption and continued use despite negative consequences (Volkow, Wang, Tomasi, & Baler, 2013). Study participants with BED also appear to exhibit potential dysfunction in neural regions implicated in executive control and reward processing relative to healthy controls (Balodis *et al*., 2013; Wang *et al*., 2011; Weygandt, Schaefer, Schienle, & Haynes, 2012), a pattern that has also been noted in addictive disorders. These findings raise the possibility that similar to addictive disorders, the diminished control over food consumption associated with BED may be related to neural differences in regions implicated in executive control and reward processing. Thus, it has been suggested that these parallels may reflect the role of an addictive-like process in obesity and binge eating.

RECENT RESEARCH

Animal Model Research

Intriguing animal model research suggests that certain food stuffs or ingredients may be capable of triggering an addictive process. Rats maintained on a limited access schedule to a sugar solution exhibit several behaviors that are commonly associated with substance dependence. For example, these animals tend to show larger and fewer eating episodes than controls, which is thought to reflect "bingeing" (Avena, Rada, & Hoebel, 2008). Further, after a period of abstinence, these animals show a marked increase in responding for sugar, which is considered an indication of craving (Avena, Long, & Hoebel, 2005). When administered an opioid antagonist, which blocks opioid receptors, or fasted from all food for 36 h, these animals also show evidence of opiate-like withdrawal, including teeth chattering, forepaw tremors, and head shakes (Avena, Bocarsly, Rada, Kim, & Hoebel, 2008; Colantuoni *et al*., 2002). In addition, Oswald, Murdaugh, King, and Boggiano (2011) found that rats prone to binge eating will endure greater magnitudes of electric shock to obtain palatable food than those resistant to binge eating. Further, unlike rats with no access or 1 h/day access to a cafeteria diet consisting of various meats, cakes, frosting, and chocolate, rats with extended access (18–23 h/day) to this diet do not decrease their intake of palatable food when shown

a stimulus associated with a foot shock (Johnson & Kenny, 2010). These two findings provide evidence that such animals will pursue the reward associated with palatable food consumption despite adverse consequences. Recent research has also shown that rats prefer a sucrose solution to both cocaine and heroin when given the option to press a lever associated with each (Madsen & Ahmed, 2014).

These behavioral indices of addiction are accompanied by neurochemical alterations. For example, sugar consumption has been shown to repeatedly increase dopamine within the nucleus accumbens, a brain region associated with reward, of rats given intermittent sugar and chow access (Rada, Avena, & Hoebel, 2005). In contrast, although rats given intermittent chow access show an initial spike in dopamine, over time, this response decreases. However, rats with intermittent sugar and chow access continue to release high levels of dopamine when given sugar, which reflects the pattern seen with morphine administration (Pothos, Rada, Mark, & Hoebel, 1991). In addition, Johnson and Kenny (2010) have found that rats with extended access to the cafeteria diet mentioned above show a decrease in a type of dopamine receptor (D2 receptors) within the striatum compared to control groups. This is noteworthy as a number of studies have shown reduced striatal D2 receptors among individuals with substance dependence disorders (Fehr *et al*., 2008). Further, rats that were genetically modified to have less striatal D2 receptors demonstrate increased reward thresholds, suggesting that they require greater stimulation to experience reward (Johnson & Kenny, 2010). Findings such as these indicate underlying neurochemical abnormalities that may result from and/or perpetuate addictive behaviors regarding food.

Human Research

Although there are strong parallels in brain regions that encode reward from drugs and palatable food and in neural abnormalities associated with substance dependence and obesity/BED, these findings may tell us little about true "food addiction." Obesity is often linked to excess food consumption, but other factors can contribute to unhealthy weight gain, including physical inactivity, medication side effects, and metabolic conditions. In addition, excess consumption of a substance is not necessarily indicative of substance dependence, or an "addiction." For example, 40% of college students are reported to binge drink (O'Malley & Johnston, 2002), but only 6% meet criteria for alcohol dependence (Knight *et al*., 2002). Further, addictive disorders are typically diagnosed in the presence of certain behavioral indicators (e.g., loss of control, continued use despite negative consequences) (Table 1). Thus,

Table 1
Diagnostic Criteria for Substance Dependence as Stated in the DSM-IV-TR (APA, 2000)

1. Tolerance, as defined by either of the following:
   a. The need for markedly increased amounts of the substance to achieve intoxication or desired effect
   b. Markedly diminished effect with continued use of the same amount of the substance
2. Withdrawal, as manifested by either of the following:
   a. The characteristic withdrawal syndrome for the substance
   b. The same (or closely related) substance is taken to relieve or avoid withdrawal symptoms
3. Taking the substance often in larger amounts or over a longer period than was intended
4. There is a persistent desire or unsuccessful effort to cut down or control substance use
5. Spending a great deal of time in activities necessary to obtain or use the substance or to recover from its effects
6. Giving up social, occupational, or recreational activities because of substance use
7. Continuing the substance use with the knowledge that it is causing or exacerbating a persistent or recurrent physical or psychological problem

when assessing food addiction, it would be useful to identify individuals who exhibit similar signs of addiction with respect to their eating behavior in addition to observing neural responses to food cues and receipt.

The Yale Food Addiction Scale (YFAS) was developed to operationalize the construct of palatable food dependence (Gearhardt, Corbin, & Brownell, 2009) based on the DSM-IV-TR substance dependence criteria (APA, 2000). A recent study found that the risk of YFAS "food addiction" increased with obesity status and the severity of addictive-like eating was positively related to measures of adiposity [e.g., body fat, BMI (body mass index)] (Pedram *et al*., 2013). Further, addictive-like eating, as measured by the YFAS, has been associated with a higher likelihood of a composite index of elevated dopamine signaling (Davis *et al*., 2013), as well as a greater severity of disordered eating (Gearhardt *et al*., 2012). In addition, when anticipating food receipt, higher YFAS scores have been related to increased activation of brain regions associated with craving and the motivation to eat, which may resemble elevated motivation elicited by cues in addicted populations (Gearhardt *et al*., 2011). Also akin to addictive disorders, this study found an association between higher YFAS scores and reduced activation in neural regions implicated in inhibitory control during palatable food receipt (Gearhardt *et al*., 2011). Notably, the high and low "food addiction" groups in this study did not differ by BMI and the results of the study remained

the same even after controlling for body weight. Therefore, behavioral indicators of addictive-like eating are related to neural activation implicated in addictive disorders even apart from the possible effects of elevated body weight. This finding highlights the importance of viewing BMI and "food addiction" as separate with overlap in certain cases.

Similarly, BED and YFAS "food addiction" are related to one another but there also appear to be distinctions between the two. Approximately fifty percent of obese patients diagnosed with BED meet the "food addiction" threshold (Gearhardt, White, Masheb, & Grilo, 2013; Gearhardt *et al*., 2012), which suggests that not all BED patients endorse addictive-like eating. In BED patients, elevated YFAS scores are related to more frequent binge eating episodes, elevated emotion dysregulation, and increased eating pathology (Davis, 2013; Gearhardt *et al*., 2012, 2013). Thus, "food addiction" may be associated with more severe pathology in the context of BED (Gearhardt *et al*., 2012; Gearhardt, Roberto, Seamans, Corbin, & Brownell, 2013), perhaps making it more difficult to treat. This is consistent with prior research suggesting that a BED diagnosis does not reflect a homogenous group, but rather that there are subtypes within the diagnosis. One BED subtype is indicated by high-levels of dietary restraint while another exhibits greater negative affect, impulsivity, and overall pathology (Grilo, Masheb, & Wilson, 2001; Stice *et al*., 2001). These two subtypes of BED could potentially be driven by different mechanisms, with an addictive process possibly associated with the latter subtype but not the former.

KEY ISSUES FOR FUTURE RESEARCH

Although there has been substantial progress in the study of addictive-like eating, this is a relatively new area of research and there are still more questions than answers. One of the most important areas for future research is to examine what types of food or components of food may be capable of triggering an addictive process. Especially in the human literature, the vast majority of research has focused on who may be experiencing an addictive-like response to food, but less attention has been paid to identifying the possible addictive ingredients of food. The identification of the addictive agent will provide important information about the mechanisms driving compulsive eating. It is clear that all food stuffs are not equal in their ability to trigger addictive-like eating. For example, it is very rare for someone to report binging on broccoli or eating chicken breasts until they are uncomfortably full. In contrast, food rich in added sugars, refined carbohydrates, and/or fats (e.g., candy, chocolate, French fries, cheese burgers, and pasta), are much more likely to be described as problematic. At this point, sugar has been identified as the most likely culprit in eliciting addictive behaviors based on findings

from animal models and some neuroimaging research (Avena, Rada, *et al*., 2008; Stice, Burger, & Yokum, 2013). However, many of the food stuffs that seem to induce cravings and loss of control contain ingredients other than sugar (e.g., fat, other refined carbohydrates, salt), indicating a need for further research in this area. In addition, it may be beneficial to assess possible interactions between ingredients.

A better understanding of which types of food or ingredients tend to result in addictive behaviors may also inform policy and treatment approaches. The greatest tobacco-related public health gains were made when policy approaches altered the price and access to cigarettes, as well as other nicotine products, and the companies creating and marketing these products became more culpable. If certain food stuffs are identified as addictive, this may be helpful in understanding the specific ways in which our environment encourages excessive consumption of these food stuffs and identifying effective targets for policy interventions. For example, food marketing is a major source of cues for potentially addictive food stuffs in our environment and the industry spends millions of dollars to ensure that these advertising campaigns are effective. Recent research has identified that food commercials, relative to other types of commercials, are more effective in triggering reward circuitry in the brains of adolescents (Gearhardt, Yokum, Stice, Harris & Brownell, 2014). If certain food stuffs are found to be capable of triggering an addictive response, this may inform policy debates about restricting the advertising of these products, especially to children.

There are also a number of treatment implications of "food addiction" that are yet to be explored. One commonly held approach in the treatment of eating-related problems is that there are no "good" or "bad" types of food. For individuals struggling with overeating highly palatable food, the implicit message is that their struggles are a result of deficiencies in their willpower. Although there are certainly individual differences that may increase the risk of overeating, there are also likely properties of certain food stuffs that differentially activate the reward system and may perpetuate compulsive eating. One of the most controversial implications of the addiction construct is that some individuals may benefit from the elimination of certain types offood from their diet, similar to the elimination of alcohol for individuals with alcohol use disorders. This raises concerns from other perspectives, including the fear that this increase in dietary restraint may backfire and trigger more disordered eating (Polivy, 1996). Although support for the restraint hypothesis of disordered eating is mixed, this is a valid concern and a research topic that needs to be examined empirically, particularly among those who met the criteria for a "food addiction." For example, it may be insightful to examine the outcomes of existing programs that employ an abstinence approach to kinds of certain food, such as Overeaters

Anonymous and Food Addicts Anonymous. Other aspects of addiction treatment are consistent with traditional approaches to treating problematic eating behaviors and many addiction-related strategies are already being implemented in eating-focused interventions, such as identifying triggers, coping with cravings, developing alternative ways to cope with negative emotions, motivational interviewing and relapse prevention (Gearhardt, White, & Potenza, 2011; von Ranson & Robinson, 2006).

To increase the effectiveness of these intervention approaches and to more appropriately evaluate the "food addiction" hypothesis, it will be important to identify phenotypes that reflect addictive-like eating rather than using rough proxies. As mentioned previously, obesity has been frequently used as a proxy for addictive eating and critics of the "food addiction" hypothesis point out that the neurobiological parallels between obesity and addiction are somewhat inconsistent (Ziauddeen, Farooqi, & Fletcher, 2012). Yet, obesity is a heterogeneous condition and it is highly unlikely that all obesity results from "food addiction." Further, not all individuals with a healthy BMI have a normal relationship with food. Thus, the use of weight status to indicate addictive-like eating likely results in both the over- and under-identification of "food addicts," which may contribute to discrepancies in the literature (Avena, Gearhardt, Gold, Wang, & Potenza, 2012). Developing a better understanding of the subtypes of individuals most prone to addictive-eating behavior will be essential to more precisely examine whether addictive mechanisms are truly contributing to problematic eating and to match patients to the most appropriate treatment. The YFAS is one tool that attempts to classify this subtype but further evaluation is needed and additional tools, such as clinical interviews, may be warranted.

Further, certain mechanisms implicated in addiction have received little empirical evaluation regarding their role in problematic eating. Tolerance and withdrawal are key indicators of physiological dependence to an addictive substance. Although neither of these criteria is required for a diagnosis of substance dependence, tolerance and withdrawal are often considered key characteristics of addiction. Reviews critical of the validity of the food addiction construct will state that there is no evidence of withdrawal or tolerance with such eating behaviors (Drewnowski & Bellisle, 2007; Wilson, 2010; Ziauddeen *et al*., 2012), but this is premature given the marked lack of studies in the area and the evidence for withdrawal and tolerance to sugar demonstrated in animal models. In one of the only studies on this topic in humans, Spring *et al*. (2008) found that after repeated administrations, carbohydrate cravers exhibited tolerance to the pleasant affective consequences of consuming a carbohydrate-rich shake (compared to a placebo shake). Regarding withdrawal, there have been anecdotal reports of headaches, irritability, sleep

disturbance, chills and low mood among dieters cutting down on refined carbohydrates (Atkins, 2002). Empirical research is needed to explore whether behavioral, biological, and psychological indicators of withdrawal and tolerance are present among humans who report problematic eating behavior, and if certain types of food are more likely to trigger these responses than others.

Finally, little is known about the role that addictive-like eating may play in childhood obesity. In the substance dependence literature, an earlier age of exposure to addictive substances (e.g., alcohol, nicotine) is implicated in risk for the development of future problematic substance use (DeWit, Adlaf, Offord, & Ogborne, 2000). This risk may result from adaptations in a vulnerable neural system (Tapert, Caldwell, & Burke, 2005), as well as an increased likelihood to use substances to cope psychologically (Clark, Thatcher, & Tapert, 2008). If certain types of food also have addictive potential, children may be especially susceptible to this effect as a result of neural and psychological vulnerabilities.

There is some evidence to suggest that addictive processes may be at play in problematic eating behavior in children. In a qualitative study of overweight/obese 8–21 year olds, behaviors consistent with addiction (e.g., tolerance, cravings) were frequently described and 66% identified addiction as a contributor to their eating problems (Pretlow, 2011). In addition, 15.2% of children receiving treatment at a pediatric lipid clinic reported that they often, usually, or always felt addicted to food and reports of addictive eating were related to more significant eating problems (Merlo, Klingman, Malasanos, & Silverstein, 2009). Recently, the YFAS was adapted to be administered to children. Elevated scores on this measure were associated with greater BMI. Further, children with higher YFAS scores were more likely to emotionally eat and to be less responsive to satiety signals (Gearhardt *et al*., 2013). An important area of future research is to examine whether neurobiological parallels to addiction are seen in children who report addictive-like eating behaviors. In addition, animal models suggest that addictive-like consumption of sugar increases the propensity to be sensitized to other drugs of abuse (Avena, Carrillo, Needham, Leibowitz, & Hoebel, 2004; Avena & Hoebel, 2003). Thus, it will be important to examine whether addictive-like eating patterns early in development increase the likelihood for future problems with drugs of abuse.

Accumulating evidence suggests that addictive processes marked by behavioral changes and brain adaptations may develop in response to palatable food and beverage consumption. Further research is needed, however, to more fully understand the specific components of such food and beverages that may contribute to addiction-like symptoms. In addition,

further study is needed to explore and characterize certain features of addiction, such as tolerance and withdrawal, among human samples. Finally, it is critical to begin assessing the effects of palatable food consumption during vulnerable periods of development, such as childhood and adolescence. With progress in these areas, this emerging field of research may lend practical insight into the public health and psychological issues of both binge eating behavior and obesity.

REFERENCES

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. text revision). Washington, DC: Author.

Atkins, R. C. (2002). *Dr. Atkins' new diet revolution* (2002 ed.). North Yorkshire, England: Guill.

Avena, N. M., Bocarsly, M. E., Rada, P., Kim, A., & Hoebel, B. G. (2008). After daily bingeing on a sucrose solution, food deprivation induces anxiety and accumbens dopamine/acetylcholine imbalance. *Physiolpgy & Behavior*, *94*(3), 309–315.

Avena, N. M., Carrillo, C. A., Needham, L., Leibowitz, S. F., & Hoebel, B. G. (2004). Sugar-dependent rats show enhanced intake of unsweetened ethanol. *Alcohol*, *34*(2–3), 203–209.

Avena, N. M., Gearhardt, A. N., Gold, M. S., Wang, G. J., & Potenza, M. N. (2012). Tossing the baby out with the bathwater after a brief rinse? The potential downside of dismissing food addiction based on limited data. *Nature Reviews Neuroscience*, *13*(7), 514.

Avena, N. M., & Hoebel, B. G. (2003). A diet promoting sugar dependency causes behavioral cross-sensitization to a low dose of amphetamine. *Neuroscience*, *122*(1), 17–20.

Avena, N. M., Long, K. A., & Hoebel, B. G. (2005). Sugar-dependent rats show enhanced responding for sugar after abstinence: Evidence of a sugar deprivation effect. *Physiology & Behavior*, *84*(3), 359–362.

Avena, N. M., Rada, P., & Hoebel, B. G. (2008). Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neuroscience & Biobehavioral Reviews*, *32*(1), 20–39.

Balodis, I. M., Kober, H., Worhunsky, P. D., White, M. A., Stevens, M. C., Pearlson, G. D. … Potenza, M. N. (2013). Monetary reward processing in obese individuals with and without binge eating disorder. *Biological Psychiatry*, *73*(9), 877–886.

Clark, D. B., Thatcher, D. L., & Tapert, S. F. (2008). Alcohol, psychological dysregulation, and adolescent brain development. *Alcoholism: Clinical and Experimental Research*, *32*(3), 375–385.

Colantuoni, C., Rada, P., McCarthy, J., Patten, C., Avena, N. M., Chadeayne, A., & Hoebel, B. G. (2002). Evidence that intermittent, excessive sugar intake causes endogenous opioid dependence. *Obesity Research*, *10*(6), 478–488.

Davis, C. (2013). Compulsive overeating as an addictive behavior: Overlap between food addiction and binge eating disorder. *Current Obesity Reports*, *2*, 171–178.

Davis, C., Loxton, N. J., Levitan, R. D., Kaplan, A. S., Carter, J. C., & Kennedy, J. L. (2013). 'Food addiction' and its association with a dopaminergic multilocus genetic profile. *Physiology & Behavior*, *118*, 63–69.

DeWit, D. J., Adlaf, E. M., Offord, D. R., & Ogborne, A. C. (2000). Age at first alcohol use: A risk factor for the development of alcohol disorders. *American Journal of Psychiatry*, *157*(5), 745–750.

Drewnowski, A., & Bellisle, F. (2007). Is sweetness addictive? *Nutrition Bulletin*, *32*, 52–60.

Fehr, C., Yakushev, I., Hohmann, N., Buchholz, H.G., Landvogt, C., Deckers, H., … Schreckenberger, M. (2008). Association of low striatal dopamine d2 receptor availability with nicotine dependence similar to that seen with other drugs of abuse. *American Journal of Psychiatry*, *165*(4), 507–514.

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Preliminary validation of the Yale Food Addiction Scale. *Appetite*, *52*(2), 430–436.

Gearhardt, A. N., Roberto, C. A., Seamans, M. J., Corbin, W. R., & Brownell, K. D. (2013). Preliminary validation of the Yale Food Addiction Scale for children. *Eating Behaviors*, *14*(4), 508–512.

Gearhardt, A. N., White, M. A., Masheb, R. M., & Grilo, C. M. (2013). An examination of food addiction in a racially diverse sample of obese patients with binge eating disorder in primary care settings. *Comprehensive Psychiatry*, *54*(5), 500–505.

Gearhardt, A. N., White, M. A., Masheb, R. M., Morgan, P. T., Crosby, R. D., & Grilo, C. M. (2012). An examination of the food addiction construct in obese patients with binge eating disorder. *International Journal of Eating Disorders*, *45*(5), 657–663.

Gearhardt, A. N., White, M. A., & Potenza, M. N. (2011). Binge eating disorder and food addiction. *Current Drug Abuse Reviews*, *4*(3), 201–207.

Gearhardt, A. N., Yokum, S., Orr, P. T., Stice, E., Corbin, W. R., & Brownell, K. D. (2011). Neural correlates of food addiction. *Archives of General Psychiatry*, *68*(8), 808–816.

Gearhardt, A. N., Yokum, S., Stice, E., Harris, J. L., & Brownell, K. D. (2014). Relation of obesity to neural activation in response to food commercials. *Social Cognitive Affective Neuroscience*, *9*(7), 932–938.

Grilo, C. M., Masheb, R. M., & Wilson, G. T. (2001). Subtyping binge eating disorder. *Journal of Consulting & Clinical Psychology*, *69*, 1066–1072.

Johnson, P. M., & Kenny, P. J. (2010). Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. *Nature Neuroscience*, *13*(5), 635–641.

Knight, J. R., Wechsler, H., Kuo, M., Seibring, M., Weitzman, E. R., & Schuckit, M. A. (2002). Alcohol abuse and dependence among U.S. college students. *Journal of Studies on Alcohol*, *63*(3), 263–270.

Madsen, H. B., & Ahmed, S. H. (2014). Drug versus sweet reward: Greater attraction to and preference for sweet versus drug cues. *Addiction Biology* [Epub ahead of print].

Merlo, L. J., Klingman, C., Malasanos, T. H., & Silverstein, J. H. (2009). Exploration of food addiction in pediatric patients: A preliminary investigation. *Journal of Addiction Medicine*, *3*(1), 26–32.

O'Malley, P. M., & Johnston, L. D. (2002). Epidemiology of alcohol and other drug use among American college students. *Journal of Studies on Alcohol. Supplement*, *14*, 23–39.

Oswald, K. D., Murdaugh, D. L., King, V. L., & Boggiano, M. M. (2011). Motivation for palatable food despite consequences in an animal model of binge eating. *International Journal of Eating Disorders*, *44*(3), 203–211.

Pedram, P, Wadden, D, Amini, P, Gulliver, W, Randell, E, Cahill, Fa, … Zhai, G. (2013). Food addiction: Its prevalence and significant association with obesity in the general population. *PLoS One*, *8*(9), e74832.

Polivy, J. (1996). Psychological consequences of food restriction. *Journal of the American Dietetic Association*, *96*(6), 589–592.

Pothos, E., Rada, P., Mark, G. P., & Hoebel, B. G. (1991). Dopamine microdialysis in the nucleus accumbens during acute and chronic morphine, naloxone-precipitated withdrawal and clonidine treatment. *Brain Research*, *566*(1–2), 348–350.

Pretlow, R. A. (2011). Addiction to highly pleasurable food as a cause of the childhood obesity epidemic: A qualitative internet study. *Eating Disorders*, *19*(4), 295–307.

Rada, P., Avena, N. M., & Hoebel, B. G. (2005). Daily bingeing on sugar repeatedly releases dopamine in the accumbens shell. *Neuroscience*, *134*(3), 737–744.

Spring, B., Schneider, K., Smith, M., Kendzor, D., Appelhans, B., Hedeker, D., & Pagoto, S. (2008). Abuse potential of carbohydrates for overweight carbohydrate cravers. *Psychopharmacology*, *197*(4), 637–647.

Stice, E., Agras, W. S., Telch, C. F., Halmi, K. A., Mitchell, J. E., & Wilson, T. (2001). Subtyping binge eating-disordered women along dieting and negative affect dimensions. *International Journal of Eating Disorders*, *30*(1), 11–27.

Stice, E., Burger, K. S., & Yokum, S. (2013). Relative ability of fat and sugar tastes to activate reward, gustatory, and somatosensory regions. *American Journal of Clinical Nutrition*, *98*(6), 1377–1384.

Tapert, S. F., Caldwell, L., & Burke, C. (2005). Alcohol and the adolescent brain: Human studies. *Alcohol Research & Health*, *28*, 205–212.

Volkow, N. D., Wang, G. J., Fowler, J. S., Logan, J., Jayne, M., Franceschi, D., … Pappas, N. (2002). "Nonhedonic" food motivation in humans involves dopamine in the dorsal striatum and methylphenidate amplifies this effect. *Synapse*, *44*(3), 175–180.

Volkow, N. D., Wang, G. J., Fowler, J. S., & Telang, F. (2008). Overlapping neuronal circuits in addiction and obesity: evidence of systems pathology. *Philosophical Transactions of the Royal Society, B: Biological Sciences*, *363*(1507), 3191–3200.

Volkow, N. D., Wang, G. J., Tomasi, D., & Baler, R. D. (2013). Obesity and addiction: Neurobiological overlaps. *Obesity Reviews*, *14*, 2–18.

von Ranson, K. M., & Robinson, K. E. (2006). Who is providing what type of psychotherapy to eating disorder clients? A survey. *International Journal of Eating Disorders*, *39*(1), 27–34.

Wang, G. J., Geliebter, A., Volkow, N. D., Telang, F. W., Logan, J., Jayne, M. C., … Fowler, J. S. (2011). Enhanced striatal dopamine release during food stimulation in binge eating disorder. *Obesity (Silver Spring)*, *19*(8), 1601–1608.

Weygandt, M., Schaefer, A., Schienle, A., & Haynes, J. D. (2012). Diagnosing different binge-eating disorders based on reward-related brain activation patterns. *Human Brain Mapping*, *33*(9), 2135–2146.

Wilson, G. T. (2010). Eating disorders, obesity and addiction. *European Eating Disorders Review*, *18*(5), 341–351.

Ziauddeen, H., Farooqi, I. S., & Fletcher, P. C. (2012). Obesity and the brain: How convincing is the addiction model? *Nature Reviews Neuroscience*, *13*(4), 279–286.

ASHLEY GEARHARDT SHORT BIOGRAPHY

Ashley Gearhardt, PhD, is an Assistant Professor of Clinical Psychology at the University of Michigan. While working on her doctorate in clinical psychology at Yale University, Dr. Gearhardt became interested in the possibility that certain food stuffs may be capable of triggering an addictive process. To explore this further, she developed the Yale Food Addiction Scale (YFAS) to operationalize addictive-like eating behaviors. Scores on this scale have recently been linked with more frequent binge eating episodes in clinical populations, increased prevalence of obesity and patterns of neural activation implicated in other addictive behaviors. Dr. Gearhardt also investigates the impact of certain components of the food environment, such as food advertising, on obesity risk through the use of multi-method approaches (e.g., neuroimaging, eye tracking). She is currently the director of the Food and Addiction Science and Treatment (FAST) lab to further evaluate whether addictive-like mechanisms contribute to certain types of problematic eating behavior. Web site: http://fastlab.psych.lsa.umich.edu/

SUSAN MURRAY SHORT BIOGRAPHY

Susan Murray is currently a research assistant in Dr. Nicole Avena's laboratory at the Icahn School of Medicine, Mount Sinai. Susan received a BS in psychology from Fordham University in 2012. Her academic interests include the study of eating disorders, obesity, and coping mechanisms. She has published five articles in scholarly journals and two book chapters on topics related to the neural correlates of under- and overeating, obesity, and food addiction.

NICOLE M. AVENA SHORT BIOGRAPHY

Dr. Nicole M. Avena is a research psychologist and neuroscientist with interests in the fields of nutrition, diet and addiction. She received a PhD in Psychology and Neuroscience from Princeton University in 2006, followed by a postdoctoral fellowship at Rockefeller University. Dr. Avena presently holds

a faculty position in the Department of Pharmacology and Systems Therapeutics at the Icahn School of Medicine, Mount Sinai. She has published over 60 scholarly journal articles on topics related to diet, nutrition and overeating. Her research achievements have been honored by awards from groups including the New York Academy of Sciences, the American Psychological Association, and the National Institute on Drug Abuse.

Dr. Avena has a website www.DrNicoleAvena.com, a blog on Psychology Today, and you can also follow her on Twitter or Facebook.

RELATED ESSAYS

Cognitive Remediation in Schizophrenia *(Psychology)*, Clare Reeder and Til Wykes
Cognitive Bias Modification in Mental *(Psychology)*, Meg M. Reuland *et al*.
Born This Way: Thinking Sociologically about Essentialism *(Sociology)*, Kristen Schilt
Clarifying the Nature and Structure of Personality Disorder *(Psychology)*, Takakuni Suzuki and Douglas B. Samuel
Taking Personality to the Next Level: What Does It Mean to Know a Person? *(Psychology)*, Simine Vazire and Robert Wilson
A Gene-Environment Approach to Understanding Youth Antisocial Behavior *(Psychology)*, Rebecca Waller *et al*.
Crime and the Life Course *(Sociology)*, Mark Warr and Carmen Gutierrez
Rumination *(Psychology)*, Edward R. Watkins
Emotion Regulation *(Psychology)*, Paree Zarolia *et al*.