Food Addiction

A New Mental Health Disorder?

The World Health Organization (2018) reports that global obesity is an epidemic, a significant public health problem in industrialized and developing countries, is the second leading cause of preventable deaths, and has approximately tripled in prevalence since 1975. In the United States, the prevalence of obesity was 39.8% in adults and 18.5% in youth in 2015-2016 (Hales, Carroll, Fryar, & Ogden, 2017). These statistics prompt psychiatric–mental health nurses (PMHNs), who are knowledgeable about eating disorders, to ask what is the root cause of this global and national obesity epidemic and what can we do to treat the underlying causes as well as individuals diagnosed with binge eating disorder (BED)? This editorial proposes that we broaden our paradigm about eating disorders to include the concept of food addiction as a way to look at this global obesity epidemic. What is food addiction? How does it differ from BED? What are the causes of overeating? What is the role of PMHNs to treat these clients in a holistic manner?

BACKGROUND

Addiction is a chronic relapsing disorder characterized by impulsivity and compulsivity with a neurobiological basis (Koob, 2008). There are three stages to addiction: (a) the binge–intoxication stage, (b) the withdrawal–negative affect stage, and (c) the preoccupation–anticipation stage (Koob, 2008). A person with food addiction can been seen to go through these stages.

The concept of food addiction began in 2001 when Wang et al. (2001) reported research findings connecting dopamine and obesity. Dopamine modulates motivation and reward circuits. Dopamine deficiency perpetuates pathological and compulsive eating as a means to compensate for decreased activation of these circuits. Over the past 18 years, research publications have identified that the food industry has engineered palatable foods high in sugar, salt, and fat as addictive foods, compared to traditional foods (e.g., fruits, vegetables, nuts). Food cues and consumption can activate neurocircuitry in food addiction similar to that seen in drug addiction (Gearhardt, Grilo, DiLeone, Brownell, & Potenza, 2011; Schulte, Sonneville, & Gearhardt, 2019; Tomasi & Volkow, 2013; Volkow, Wang, Tomasi, & Baler, 2013).

A person who is addicted to food exhibits loss of control, tolerance, withdrawal, and continued use despite dangerous consequences (Shriner & Gold, 2013). Food addiction is one type of behavioral addiction; others include gambling disorder, internet addiction, exercise addiction, and sex addiction (Ascher & Levounis, 2015; Rosenberg & Feder, 2014; Wieland, 2015). Although food addiction is currently not an official diagnosis in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*; American Psychiatric Association [APA], 2013), Volkow and O'Brien (2007) had published a letter to the editor in the *American Journal of Psychiatry* that provided an argument for inclusion in the *DSM-5*. In the letter to the editor, Volkow and O'Brien (2007) proposed that the diagnosis and treatment of obesity purely as a metabolic disorder has not led to successful treatment outcomes, such as sustained weight loss for obese patients; therefore, a new paradigm is needed to focus on obesity as a brain disorder. Hypothalamic and limbic areas of the brain are involved with reward, motivation, learning, emotions, and stress responses, which can result in the loss of control over eating similar to the loss of control that occurs in drug use. Food activates brain reward circuitry via palatability and increases peptides such as leptin and insulin. Repeated exposure to certain foods by genetically vulnerable individuals results in poor food intake control and excessive weight gain (Volkow & O'Brien, 2007).

Similar to other behavioral addictions, food addiction has been documented to have a neurobiological basis (Gearhardt, Corbin, & Brownell, 2009; Volkow & O'Brien, 2007). Individuals with substance dependence and food-seeking behaviors share similar neurocircuitry (Gearhardt et al., 2009; Volkow & O'Brien, 2007), which has led to the concept of food as drugs (Davis & Carter, 2009). Dopamine is the neurotransmitter for pleasure and is a common denominator for food addiction and substance use disorders (Gold, Teitelbaum, & Gold, 2015; Peeke, 2012; Yau, Gottlieb, Krasna, & Potenza, 2014). In the reward center in the brain (i.e., the ventral tegmental area [VTA]), "almost 90% of the dopamine neurons get stimulated when we eat" (Peeke, 2012, p. 43). Neural projections from the VTA reach out to other brain areas, including the mesocortical and mesolimbic dopamine systems, nucleus accumbens (site for reward, pleasure,

and addiction), amygdala (where emotions are processed), and hippocampus (where short-term memory is converted to long-term memory) (Peeke, 2012). Dopamine binds to dopamine receptors. As demonstrated with Pavlov's dogs, sights, smells, sounds, and thoughts of favored foods are associated with pleasure. Over time, these associations become intense and the neural pathways become stronger. When favored foods are craved, the prefrontal cortex is motivated to retrieve the foods desired. The motivation-learning-memory-reward circuit interplay is key in addiction (Peeke, 2012). Hyperpalatable foods (i.e., those high in fat, salt, and sugar) have been suggested to increase food consumption (Gold et al., 2015; Peeke, 2012). Hyperpalatable foods quickly overwhelm the brain's dopamine receptors (downregulating dopamine receptors in the mesolimbic pathway), which in turn increases a person's appetite for the craved food (Gold et al., 2015; Peeke, 2012; Yau et al., 2014). This is the mechanism of the development of tolerance (Peeke, 2012), where more of the food is needed to cause satiety. For example, one or two scoops of chocolate ice cream may be satisfying initially, but for individuals with food addiction, tolerance builds. In the future, a pint of ice cream will need to be eaten to provide satiety. Given that BED and food addiction result in episodes of loss of control over eating, what is the difference between food addiction and BED?

FOOD ADDICTION AND BINGE EATING DISORDER

Food addiction and BED appear to be characterized by excessive food consumption, which results in weight gain and elevated body mass index (Gearhardt, White, & Potenza, 2011). Food addiction and BED may represent unique but overlapping conditions. Food addiction does not explain all cases of obesity, but for some individuals, overeating may be experienced as a passive event, which includes liberal snacking, large portions, and physical inactivity (Avena, Bocarsly, Hoebel, & Gold, 2011; Yau et al., 2014). Food addiction has been controversial because eating, compared to drug use, is a normative behavior necessary for survival. This controversy stems from lack of a formal definition for food addiction and scientific studies to determine the addictive properties of some foods.

The construct of food addiction is relevant to a certain subtype of obese people, such as those with BED (Avena, Gearhardt, Gold, Wang, & Potenza, 2012). BED is a recent diagnosis in the *DSM-5* (APA, 2013). Criteria, etiology, treatment, and nursing care for BED have been presented in the psychiatric nursing literature (Ambrogne, 2017). Although not labeled as such, several criteria for BED approximate criteria for substance use disorders (SUDs) including reduced control over eating (APA, 2013). BED has diagnostic criteria of (a) recurrent binge eating episodes of large amounts of food within a discrete 2-hour period and a sense of lack of control over what or how much one is eating during the episode; (b) marked distress over the binge eating episode; (c) binging occurring, on average, once per week for 3 months; and (d) not being associated with inappropriate compensatory mechanisms of weight loss (e.g., purging, using laxatives/diuretics, exercising) as in bulimia nervosa and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa (APA, 2013).

ROLE OF PSYCHIATRIC–MENTAL HEALTH NURSES IN FOOD ADDICTION

Nursing assessment for food addiction begins with a family history for obesity and alcohol dependency. Heredity has been shown to account for as much as 60% of overall risk for obesity (Volkow & Wise, 2005). In addition, a parental history of alcohol dependence increases the preference for sweet foods, suggesting a similar genetic mechanism could contribute to both conditions (Volkow & Wise, 2005).

Nurses can further assess for food addiction using the Yale Food Addiction Scale (YFAS), a self-administered tool that has been demonstrated to be reliable and valid (Gearhardt et al., 2009). Nurses can use the data obtained from the YFAS to identify common features of food addiction that are similar to SUDs such as: repeated unsuccessful attempts to cut down on food consumption; continued use despite problems (e.g., excessive weight gain); and large amounts of time spent obtaining food, eating, or recovering from eating. These three features are also those most endorsed by obese individuals with BED (Gearhardt et al., 2009). This assessment provides nurses with data to formulate a nursing diagnosis and nursing interventions to provide nutritional counseling, meal planning, and psychotherapy to address negative emotions and environmental cues underlying compulsive eating.

Treatment of food addiction includes use of selective serotonin reuptake inhibitors to target binge eating and depressive symptoms, cognitive-behavioral therapy, interpersonal therapy, family therapy, nutritional counseling, and 12-step self-help groups, such as Overeaters Anonymous (OA) (Gold et al., 2015; Yau et al., 2014). Nurses can be the mental health providers keeping clients accountable to follow through with the treatment plan, including *The Tools of Recovery* endorsed through OA (2005), such as daily journaling, daily reading of OA materials, attending 12-step meetings, providing service to OA, getting an OA sponsor, working through the 12 steps of OA, and executing weekly meal planning.

CONCLUSION

The abundance of hyperpalatable foods available to consumers in the United States and around the world and the rising epidemic of obesity result in a growing public health problem that contributes to many chronic diseases.

Research evidence supports that obesity be recognized not only as a metabolic disorder but also a brain disorder (Volkow & O'Brien, 2007). Could food addiction be a new and evolving mental disorder in the future? If so, PMHNs will need to be ready to provide holistic care to clients with this disorder.

REFERENCES

Ambrogne, J. A. (2017). Assessment, diagnosis, and treatment of binge eating disorder. *Journal of Psychosocial Nursing and Mental Health Services, 55*(8), 32–38. https://doi.org/10.3928/02793695-20170627-01 PMID:28771285

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Ascher, M., & Levounis, P. (2015). *The behavioral addictions*. Washington, DC: American Psychiatric Publishing.

Avena, N. M., Bocarsly, M. E., Hoebel, B. G., & Gold, M. S. (2011). Overlaps in the nosology of substance abuse and overeating: The translational implications of "food addiction." *Current Drug Abuse Reviews, 4*(3), 133–139. https://doi.org/10.2174/1874473711104030133 PMID:21999687

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. https://doi.org/10.1038/nrn3212-c1 PMID:22714023

Davis, C., & Carter, J. C. (2009). Compulsive overeating as an addiction disorder: A review of theory and evidence. *Appetite, 53*(1), 1–8. https://doi.org/10.1016/j.appet.2009.05.018 PMID:19500625

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Food addiction: An examination of the diagnostic criteria for dependence. *Journal of Addiction Medicine, 3*(1), 1–7. https://doi.org/10.1097/ADM.0b013e318193c993 PMID:21768996

Gearhardt, A. N., Grilo, C. M., DiLeone, R. J., Brownell, K. D., & Potenza, M. N. (2011). Can food be addictive? Public health and policy implications. *Addiction (Abingdon, England), 106*(7), 1208–1212. https://doi.org/10.1111/j.1360-0443.2010.03301.x PMID:21635588

Gearhardt, A. N., White, M. A., & Potenza, M. N. (2011). Binge eating disorder and food addiction. *Current Drug Reviews, 4*(3), 201–207. https://doi.org/10.2174/1874473711104030201 PMID:21999695

Gold, J., Teitelbaum, K., & Gold, M. (2015). Food addiction. In M. Ascher & P. Levounis (Eds.), *The behavioral addictions* (pp. 43–52). Washington, DC: American Psychiatric Publishing.

Hales, C., Carroll, M., Fryar, C., & Ogden, C. (2017). *Prevalence of obesity among adults and youth: United States, 2015-2016*. Retrieved from https://www.cdc.gov/nchs/data/databriefs/db288.pdf

Koob, G. (2008). Neurobiology of addiction. In M. Galanter & H. Kleber (Eds.), *Textbook of substance abuse treatment* (pp. 3–16). Washington, DC: American Psychiatric Publishing.

Overeaters Anonymous. (2005). *The tools of recovery*. Rio Rancho, NM: Author.

Peeke, P. (2012). *The hunger fix. The three-stage detox and recovery plan for overeating and food addiction*. New York, NY: Rodale.

Rosenberg, K., & Feder, L. (2014). *Behavioral addictions: Criteria, evidence, and treatment*. New York, NY: Elsevier.

Schulte, E. M., Sonneville, K. R., & Gearhardt, A. N. (2019). Subjective experiences of highly processed food consumption in individuals with food addiction. *Psychology of Addictive Behaviors, 33*(2), 144–153. http://dx.doi.org/10.1037/adb0000441

Shriner, R., & Gold, M. (2011). Is your patient addicted to food? *Obesity Consults, 1*(1), 8–10.

Tomasi, D., & Volkow, N. D. (2013). Striatocortical pathway dysfunction in addiction and obesity: Differences and similarities. *Clinical Reviews in Biochemistry and Molecular Biology, 48*(1), 1–19. https://doi.org/10.3109/10409238.2012.735642 PMID:23173916

Volkow, N., Wang, G., Tomasi, D., & Baler, R. D. (2013). The addictive dimensionality of obesity. *Biological Psychiatry, 73*(9), 811–818. https://doi.org/10.1016/j.biopsych.2012.12.020 PMID:23374642

Volkow, N. D., & O'Brien, C. P. (2007). Issues for DSM-V: Should obesity be included as a brain disorder? *The American Journal of Psychiatry, 164*(5), 708–710. https://doi.org/10.1176/ajp.2007.164.5.708 PMID:17475727

Volkow, N. D., & Wise, R. A. (2005). How can drug addiction help us understand obesity? *Nature Neuroscience, 8*(5), 555–560. https://doi.org/10.1038/nn1452 PMID:15856062

Wang, G. J., Volkow, N. D., Logan, J., Pappas, N. R., Wong, C. T., Zhu, W.,...Fowler, J. S. (2001). Brain dopamine and obesity. *Lancet, 357*(9253), 354–357. https://doi.org/10.1016/S0140-6736(00)03643-6 PMID:11210998

Wieland, D. (2015). Behavioral addictions. *Journal of Psychosocial Nursing and Mental Health Services, 53*(10), 13-15. doi:1.03928/02793695-20150923-22 PMID:26489097

World Health Organization. (2018). *Obesity and overweight*. Retrieved from https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight

Yau, Y., Gottlieb, C., Krasna, L. & Potenza, M. (2014). Food addiction: Evidence, evaluation, and treatment. In K. Rosenberg & L. Feder (Eds.), *Behavioral addiction: Criteria, evidence, and treatment* (pp. 184–243). New York, NY: Elsevier.

Diane M. Wieland, PhD, RN, PMHCNS-BC
Widener University, School of Nursing
Chester, Pennsylvania

The author has disclosed no potential conflicts of interest, financial or otherwise.

doi:10.3928/02793695-20191112-01