



The National Center on
Addiction and Substance Abuse

UNDERSTANDING AND ADDRESSING FOOD ADDICTION



**A SCIENCE-BASED APPROACH TO POLICY,
PRACTICE AND RESEARCH**

FEBRUARY 2016

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Accompanying Statement by Samuel A. Ball, PhD, President and Chief Executive Officer

For nearly 25 years, The National Center on Addiction and Substance Abuse has been studying the risks, characteristics and consequences of tobacco, alcohol, and other drug use and addiction and how best to prevent, reduce and treat these critical health problems. Along with other leaders in the field, we have learned a lot about why people engage in substance use, which groups are most at risk, which individual and environmental factors increase risk for addiction, and which interventions work best to help stem the tide of the most prominent public health problem facing the nation.

As part of our new partnership with the Yale University Schools of Medicine and Public Health, this white paper, *Understanding and Addressing Food Addiction: A Science-Based Approach to Policy, Practice and Research* by Dr. Linda Richter represents our Center's first foray into applying our knowledge and expertise in substance addiction to the behavioral addictions. It employs lessons learned from years of research on the addictive substances to improve our understanding and approach to an issue of utmost importance to our nation's public health: unhealthy or disordered eating.

The escalating obesity epidemic has been a critical focus of public health professionals for years and a catalyst for examining unhealthy eating behaviors in new ways. As a result, researchers and clinicians have begun to look to what is known about substance addiction to determine what types of eating behaviors can better be explained and addressed through the lens of addiction.

The term "food addiction" gets thrown around loosely in conversation and the media. Although the scientific study of food addiction is relatively new and not without controversy, much has been learned in recent years about this form of dysregulated eating and its relationship to other forms of addictive behavior, binge eating disorder and obesity. There are probably as many reasons why people overeat as there are causes of obesity. Not all overweight persons eat in ways that resemble an addictive or eating disorder, and not all people who score on a measure of food addiction are obese. These are complex human conditions that only partly overlap and require different interventions. This area of research is also important for calling attention to what appears to be the addictive nature of certain foods or food components and highlighting innovative approaches to more effective prevention and intervention.

This white paper reviews and summarizes hundreds of scientific research papers by leading experts in the fields of addiction, eating disorders, obesity and nutrition to lay out the shared characterizing symptoms, risk factors and underlying neurobiological characteristics between food and substance addiction. It describes the differences between food addiction and binge eating disorder (now an official psychiatric diagnosis). It offers evidence-based recommendations for policy, prevention, health care practice and research, and offers informative resources for further examination of this emergent area of work. Finally, it highlights how the knowledge and experience gained from years of research, prevention and intervention work in tobacco, alcohol and other drug control might be applied to controlling another leading threat to the public health: unhealthy eating, overweight and obesity. The National Center on Addiction and Substance Abuse looks forward to contributing to this body of research and policy work through its new program at Yale under the direction of Dr. Carlos Grilo.

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While many contributed to this effort, the opinions expressed herein are the sole responsibility of The National Center on Addiction and Substance Abuse.

Understanding and Addressing Food Addiction: A Science-Based Approach to Policy, Practice and Research

Public health concerns about the escalating obesity epidemic and its far-reaching health consequences,¹ coupled with a growing understanding of the shared features of addiction across its myriad forms,² have prompted some scientists to explore the possibility that certain eating behaviors might best be explained through the lens of addiction.

The interest in applying an addiction framework to understanding certain eating behaviors and food-related disorders has grown in recent years.³ This is a result of a large body of research highlighting the considerable overlap in the characterizing symptoms, risk factors and underlying neurobiological characteristics between substance addiction and what can be thought of as food addiction.⁴ It also arises from an attempt to explore how certain types of addictive-like eating might account for pathology that cannot be explained within the context of the currently recognized eating disorders of anorexia nervosa, bulimia nervosa and binge eating disorder.⁵ The growing interest in food addiction is also partially a result of an increasing awareness that lessons learned with regard to policy, prevention and clinical practice in relation to addictive substances might fruitfully be applied to the realm of food addiction.⁶

What is Food Addiction?

The construct of food addiction has been defined as a clinically significant physical and psychological dependence on high fat, high sugar, and highly palatable foods.⁷ It is not a recognized disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), the primary resource for diagnostic criteria standards for a range of psychiatric and behavioral disorders.⁸ However, many studies that have examined the construct have utilized the Yale Food Addiction Scale (YFAS), a validated and reliable instrument is based primarily on the diagnostic criteria for substance dependence in the DSM-IV-TR, which measures behavioral indicators of addictive eating.⁹

There currently are no data available to estimate the prevalence of food addiction on a national level. Smaller studies have found that the rate in non-clinical samples ranges from an estimated 5 to 26 percent.¹⁰ Estimates that include clinical samples (i.e., those with diagnosed eating disorders) frequently find higher rates of food addiction.¹¹

Characterizing Symptoms

The concept of food addiction has a long history.¹² The term itself has been used in the scientific literature for more than half a century,¹³ and scientific interest in investigating certain types of dysfunctional eating within an addiction framework has increased significantly in recent years.¹⁴ In drafting the latest version of the DSM, the inclusion of food addiction was considered but rejected. However, the manual's chapter on feeding and eating disorders indicates that, "Some individuals with disorders described in this chapter report eating-related symptoms resembling those typically endorsed by individuals with substance use disorders, such as strong cravings and patterns of compulsive use. The resemblance may reflect the involvement of the same neural systems, including those implicated in regulatory self-control and reward in both groups of disorders."¹⁵

In the absence of a DSM-5 diagnostic category, the YFAS currently is the most widely used measurement tool for examining symptoms of food addiction, even though some argue that it might not adequately capture the construct.¹⁶ The YFAS mirrors the DSM-IV's criteria for substance dependence, in keeping with the notion that food addiction and substance addiction are closely related. Responses to the 25-item scale (see Appendix A) are coded to determine the extent to which the respondent meets each of the

criteria that corresponds to those for substance dependence in the DSM-IV. According to the YFAS, individuals who endorse three or more symptoms on the scale and demonstrate clinically significant impairment or distress within the past 12 months meet the criteria for food addiction.¹⁷

Yale Food Addiction Scale: Key Symptoms¹⁸

1. Substance taken in larger amount and for longer period than intended;
2. Persistent desire or repeated unsuccessful attempts 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 obligations, use in physically hazardous situations);
6. Tolerance (marked increase in amount; marked decrease in effect);
7. Characteristic withdrawal symptoms; substance taken to relieve withdrawal;
8. Use causes clinically significant impairment or distress.

For more detailed information on the YFAS and its scoring, see: <http://fastlab.psych.lsa.umich.edu/yale-food-addiction-scale/>

Food Addiction and Obesity

The desire to better understand how and why people overeat or eat in an unhealthy manner is clear if one considers that more than one-third of the adult population¹⁹ and one-fifth of adolescents^{* 20} in the United States are obese[†] and that the prevalence of obesity and overweight has increased substantially since 1970.²¹ Only smoking surpasses overweight/obesity as a leading cause of preventable disease and death.²² Health care costs associated with overweight and obesity in the U.S. were estimated to be approximately \$114 billion in 2008, accounting for up to 10 percent of total health care spending.²³

Obesity is a complex condition driven by many factors (e.g., biological predispositions to gain weight, high caloric intake, availability of and accessibility to highly processed foods, large portion sizes, relatively higher cost and inconvenience of obtaining healthy versus unhealthy food, low physical activity).²⁴ While there is some overlap with regard to higher weight (i.e., body mass index), such that food addiction symptoms are more likely to be found in those who are overweight or obese,²⁵ obesity and food addiction are distinct concepts.²⁶ Most obese people in the general population do not have food addiction and individuals with food addiction are not necessarily overweight or obese. Still, food addiction is associated with higher body mass index (BMI) and with more binge eating episodes, and this relationship is at least partially mediated by food craving.²⁷

A recent large, population-wide study of adult women found that six percent met the YFAS criteria for food addiction, and that women with food addiction were more likely to be overweight.²⁸ Other research shows that the rate of food addiction in obese individuals in the general population ranges from approximately 7²⁹ to 15³⁰ percent. In clinical samples of obese patients (some with binge eating disorder), approximately 42 to 57 percent met YFAS criteria for food addiction.³¹ Some research suggests that the relationship between food addiction and BMI may not be linear, such that overweight and obese individuals are more likely to have food addiction than those who are of lower weight, but that the likelihood of food addiction levels off among the severely obese.³² Research also suggests that obese

* Aged 12 to 19.

† In adults, aged 20 years and older, obesity typically is defined as a body mass index (BMI) of 30.0 or higher; overweight is defined as a BMI of 25.0 to 29.9. In children and adolescents, aged 2 to 19 years, obesity typically is defined as a BMI at or above the 95th percentile of the CDC sex-specific BMI-for-age growth charts; overweight is defined as a BMI between the 85th and 95th percentiles.

individuals who meet criteria for food addiction, compared to those who do not but who are similar in age and weight, are more likely to engage in binge eating and emotional overeating and to demonstrate food cravings.³³ They also are more likely to have co-occurring psychiatric disorders including binge eating disorder (BED), depression, and attention-deficit/hyperactivity disorder and to demonstrate more impulsivity, craving and emotional reactivity.³⁴

Obesity and Substance Addiction. Although rates of food addiction are higher in obese individuals than in those who are not obese, substance addiction occurs at relatively lower rates among obese individuals. Researchers speculate that the inverse relationship between obesity and substance use disorders may be due to competition between food and addictive substances activating similar reward-related neurotransmitter receptors in the brain.³⁵ Still, there is little evidence that obesity itself is a form of addiction.³⁶

Food Addiction and Other Forms of Disordered Eating

Individuals with each of the DSM-5 diagnosable types of clinical eating disorders--anorexia nervosa, bulimia nervosa and binge eating disorder--may demonstrate addiction-like qualities, such as obsessions, compulsions, impulsivity or the need to engage in the disordered behavior (binge eating or purging) to feel normal.³⁷ As summarized in our 2003 report, *Food for Thought: Substance Abuse and Eating Disorders*, individuals with eating disorders are up to five times as likely as those without eating disorders to have an alcohol or other illicit drug use disorder and those with a substance use disorder are up to 11 times as likely as those without to have had an eating disorder.³⁸ Despite the significant co-occurrence, there are distinct differences between eating disorders and substance addiction.³⁹ Unlike the YFAS criteria for food addiction, which largely parallel those for substance addiction, the DSM-5 diagnostic criteria for the clinical eating disorders barely overlap with those for substance use disorders.⁴⁰ (See Appendix B for the DSM-5 diagnostic criteria for substance use disorders and each eating disorder.)

Overlap Between DSM-5 Diagnostic Criteria for Substance Use Disorders and the YFAS Food Addiction and DSM-5 Eating Disorders Diagnostic Criteria^a				
Substance Use Disorders^b	Food Addiction^b	Binge Eating Disorder	Bulimia Nervosa	Anorexia Nervosa
Substance often taken in larger amounts or over longer period than intended	XX	XX	XX	
Persistent desire or unsuccessful efforts to cut down or control use	XX	**		
Great deal of time spent in activities necessary to obtain the substance or recover from its effects	X			
Craving, or a strong desire to use the substance	**	**		
Recurrent use resulting in a failure to fulfill major role obligations at work, school or home	X			
Continued use despite persistent or recurrent social or interpersonal consequences	X			
Important activities given up or reduced because of use	X			
Recurrent use in situations in which it is physically hazardous	X			
Continued use despite knowledge of having a persistent or recurrent physical or psychological problem likely to have been caused or exacerbated by use	XX			
Tolerance	X			
Withdrawal	X			
^a See Appendix A for the YFAS food addiction criteria and Appendix B for the DSM-5 substance use disorder and eating disorder diagnostic criteria. ^b A problematic pattern of use/consumption leading to clinically significant impairment or distress. ‘XX’ indicates that the symptom corresponding to the diagnostic criteria has been observed in studies of humans. ‘X’ indicates that there is preliminary evidence for this symptom in animal studies or in less rigorous human studies. ‘**’ indicates that this is not an official diagnostic criteria but is clearly evident in those with the disorder.				

Despite the presence of certain addiction-like qualities in eating disorder behaviors,⁴¹ the lack of overlap in the diagnostic criteria between addictive disorders and eating disorders reflects the distinct conceptual traditions of the two conditions. Historically, when trying to understand eating disorders, the focus has not been primarily on food itself, but rather on the thoughts and ideas that individuals with eating disorders have about food and about their own bodies (e.g., body image, shape and weight concerns). In contrast, the addictive substances themselves are prominent in understanding the disease of addiction, particularly in terms of their ability to “hijack” the brain’s reward neurocircuitry.⁴² Clearly, any comprehensive theory regarding a disorder examines the interplay between the individual’s characteristics and vulnerabilities, the properties of the target of the disorder (food, nicotine, alcohol, other drugs), and the social or environmental factors that increase the risk of disorder. Still, the emphasis in eating disorder research and treatment has concentrated less on food than on the individual’s cognitions and feelings with regard to food and weight, whereas in addiction research and treatment, the power of the addictive substance to “hook” the person is paramount. The advent of the food addiction construct bridges these two traditions and, like substance addiction, puts significant emphasis on the target of the addiction: certain types of food (i.e., those that are highly palatable and usually highly processed or refined) and their ability to “hook” those with certain psychosocial vulnerabilities or risk factors.

What food addiction does share in common with eating disorders and with substance addiction are symptoms such as a loss of control (over one’s eating or substance use), continuing the behavior (unhealthy eating or substance use) despite negative physical or psychological consequences, repeated failed attempts to cut down or stop the behavior, cravings,⁴³ emotional dysregulation (difficulty with coping, negative moods and feelings), and impulsivity.⁴⁴

Binge Eating Disorder (BED). Of the eating disorders classified in the DSM, the one that most closely conforms to the addiction model is BED,⁴⁵ which is defined by a pattern of recurrent, periodic, uncontrolled consumption of large quantities of food (i.e., binge eating) without compensatory behaviors (e.g., purging) to control weight.⁴⁶ Individuals with BED tend to eat until the point of physical discomfort and then feel depressed and guilty after the binge episode.⁴⁷ Most demonstrate an overvaluation of body weight and shape.⁴⁸ They also are at increased risk of co-occurring psychiatric and medical disorders, including substance use disorders, bipolar disorder, major depressive disorder, anxiety disorders, body dysmorphic disorder, kleptomania, irritable bowel syndrome, and fibromyalgia.⁴⁹

An estimated 2.6 percent of the U.S. population has a lifetime diagnosis of BED.⁵⁰ Individuals with BED frequently are obese or at risk for obesity; approximately 36 to 42 percent of individuals with BED are obese.⁵¹ Obese individuals with BED are more likely to demonstrate food cravings, particularly for sweet foods,⁵² than obese individuals without BED.⁵³

Rates of food addiction among those with BED are higher than among those without BED and rates of BED among those with food addiction are higher than among those without food addiction.⁵⁴ About half of obese adults who meet criteria for food addiction also meet criteria for BED.⁵⁵ Individuals who meet criteria both for BED and food addiction tend to demonstrate more psychopathology than individuals with BED who do not also have food addiction.⁵⁶

BED and Substance Addiction. Approximately 20 to 27 percent of individuals with BED also meet diagnostic criteria for a substance use disorder* (about twice as many as in the general population).⁵⁷ Although there clearly are important differences between BED and substance use disorders, there is significant overlap in terms of the neurobiology of the conditions (e.g., in both, the neurotransmitter dopamine is implicated and neural dysfunction is evident in the medial orbitofrontal cortex of the brain)

* Alcohol or illicit drugs, but not nicotine.

and in the symptomatology (e.g., diminished control and ability to cut down/abstain, continued use/consumption despite negative consequences, tolerance, withdrawal).⁵⁸

Bulimia Nervosa. Bulimia nervosa is characterized by a recurrent pattern of binge eating (rapid, uncontrolled consumption of a large amount of food in a discrete period of time) followed by compensatory (e.g., excessive exercise) or purging behavior. Individuals with bulimia demonstrate an overvaluation of body weight and shape that drives the disordered eating patterns.⁵⁹ An estimated one percent of the U.S. population has a lifetime diagnosis of bulimia nervosa.⁶⁰ Approximately 30 percent of individuals with bulimia nervosa are obese;^{*}⁶¹ however, due to the compensatory behaviors characteristic of the disorder, most are of normal weight.⁶²

Food addiction is highly prevalent in individuals with bulimia nervosa, even more so than among individuals with BED.⁶³ One recent study found that 84 percent of participants with bulimia nervosa (vs. 47 percent with BED) also met YFAS criteria for food addiction. Individuals with bulimia who also met criteria for food addiction demonstrated higher levels of food-related pathology relative to other individuals with eating disorders (i.e., those with BED and food addiction, food addiction only, BED/bulimia only) and higher rates of more general pathology than individuals with bulimia who did not have food addiction.⁶⁴ This is consistent with the notion that individuals with eating disorders like BED or bulimia who also have food addiction may have a more severe manifestation of the eating pathology relative to individuals with those eating disorders who do not meet food addiction criteria.⁶⁵

Bulimia Nervosa and Substance Addiction. Approximately 37 percent[†] of individuals with bulimia nervosa also meet diagnostic criteria for a substance use disorder (approximately four times as many as in the general population).⁶⁶ Although there is some overlap in the clinical manifestation and in the neurobiological processes of bulimia and substance addiction, particularly with regard to binges that involve sugar consumption,⁶⁷ there are key differences in terms of the driving force of the condition. In the case of bulimia (and BED), the key driver is an overvaluation of one's body weight and shape rather than the particular ingredients or components of the food that is consumed, whereas in the case of substance addiction, the properties of the addictive substance themselves largely drive and maintain the disorder.⁶⁸

Anorexia Nervosa. Anorexia nervosa is characterized by severe food restriction and extreme weight loss or maintenance of low weight.⁶⁹ Despite some apparent overlap in symptoms,⁷⁰ research has not yet specifically examined the co-occurrence of anorexia and food addiction.

Anorexia Nervosa and Substance Addiction. Approximately 27 percent of individuals with anorexia nervosa also meet diagnostic criteria for a substance use disorder.⁷¹ Although there are notable similarities between the two conditions--particularly with regard to the developmental trajectory, compulsivity, loss of control, interference with other activities, and engagement in the behavior to ameliorate negative mood or feelings⁷²--there are important differences. Like BED and bulimia, a key driver of anorexic behavior is an overvaluation of body shape and weight and a need to assert control over one's eating, which are not relevant features of substance addiction. Unlike using addictive substances, food restriction and low weight are socially and culturally acceptable and positively reinforced rather than condemned.⁷³

* Lifetime estimate.

† Lifetime estimate.

Food Addiction and Psychoactive Substance Addiction

Psychoactive substance addiction and food addiction are characterized by similar qualities. These include: the ingestion of a product in an unhealthy or excessive manner; loss of control over consumption; continuing the behavior despite negative consequences; an inability to cut down despite wanting to do so; obsessive thoughts; impulsivity; emotional reactivity; feelings of distress, regret, depression and disgust; and the tendency to relapse frequently if adequate treatment is not obtained.⁷⁴ Another important similarity is that both substance addiction and the compulsive eating that characterizes food addiction exist because of the extreme reinforcing properties of the targets of the addiction: nicotine, alcohol and drugs in the case of substance addiction and certain highly palatable or processed* food ingredients in the case of food addiction.⁷⁵

Although we do not generally think of food as an addictive drug, one way that food addiction has been conceptualized places the emphasis on the addictive qualities of certain ingredients found in most highly palatable and calorie-dense foods--sugar, fat and salt. These food ingredients, like psychoactive substances: (1) stimulate the reward and motivation-oriented regions of the brain during consumption or in the presence of related environmental cues, (2) promote craving, (3) reinforce continued consumption/use and tolerance and (4) elicit withdrawal symptoms when consumption/use is cut back or eliminated.^{† 76}

The ability of particular food ingredients to drive addictive behavior has become a key focus of public health concern due to their dominating and overabundant presence in our nation's diet.⁷⁷ When highly processed or consumed in large quantities, certain food ingredients are thought to affect the brain and body in ways similar to addictive substances like nicotine, alcohol and other drugs. In both cases, what makes a substance or ingredient addictive is that it is characterized by a concentrated dose or high potency and a rapid rate of absorption; in the case of food, these qualities often are manifested in fat or refined carbohydrates that have a high glycemic load.⁷⁸ For some people, certain types of food can set into motion physiological and psychological reactions akin to addictive licit and illicit drugs.⁷⁹ Highly processed and palatable food ingredients produce a powerful sense of pleasure via rapid activation of key regions in the brain that are designed to respond to rewarding stimuli and reinforce behaviors that ensure their repeated ingestion.⁸⁰

Particular food ingredients and cravings for those ingredients appear to relate to disordered eating in slightly different ways. One study found that craving for fat was associated with higher BMI, but not binge eating, and craving for sweets and non-sugar carbohydrates was associated with binge eating, but not higher BMI. This suggests that foods high in sugar and other carbohydrates may be more strongly associated with addiction-like eating than foods that are only high in fat.⁸¹ Other research suggests that processed food, more generally, is associated with food addiction, particularly if the processed food is high in glycemic load.^{‡ 82}

Food addiction differs to some extent from other recognized forms of addiction since eating--unlike smoking, drinking alcohol or using illicit drugs--is biologically necessary for an individual's survival. Unlike addictive substances, which usurp the circuits of reward and reinforcement that have evolved to respond to stimuli necessary for survival,⁸³ food (and the sugar, fat and salt found in many foods) is one

* Highly processed foods are those that have been designed to be particularly rewarding, usually through the addition of fat and/or refined carbohydrates.

† Much of the research on the physiological similarities between substance and food addiction is based on animal models.

‡ A food's glycemic load reflects the amount of refined carbohydrates it contains and the rate at which they are absorbed.

of the natural rewards for which that system was designed, making it uniquely challenging to curtail food addiction.⁸⁴ Therefore, abstinence, which may be a goal for those with substance addiction, is not an option when it comes to food; however, abstinence from high energy, dense, processed foods--the type most closely tied to symptoms of addiction--is possible.⁸⁵

A potential indicator of the validity of conceptualizing compulsive eating or overeating within the framework of addiction is the extent to which unhealthy eating behaviors serve as a replacement or substitution for unhealthy involvement with addictive substances or other compulsive behaviors like gambling. Research indicates that people who begin abstaining from nicotine, alcohol or other drugs following treatment may engage in unhealthy eating, particularly with regard to high sugar foods,⁸⁶ and may demonstrate weight gain.⁸⁷ Likewise, some research shows that individuals who have had certain types of bariatric surgery to facilitate weight loss may increase their use of addictive substances or develop addiction, particularly in relation to alcohol, following the surgery.⁸⁸ However, weight gain or loss is not necessarily an indicator of food addiction and the concepts of addiction transfer, substitution and replacement have not yet been studied specifically with regard to food addiction.

It is important to note that there are no established biological markers for addictive disorders that are relied upon to diagnose or classify those with the disorder. Diagnosis and assessment are based on subjective reports of behavioral symptoms rather than on physiological criteria. Likewise, the assessment of food addiction is based on behavioral symptoms rather than biological markers. Still, researchers have explored the similarities in physiological properties of those with substance and food addiction.

Finally, as is true of substance addiction, which does not develop in all individuals who use addictive substances, not all people who consume the types of food associated with problematic eating develop food addiction. Individuals' unique biological and personal qualities are critical in determining vulnerability to all types of addiction.⁸⁹

Risk Factors

Many factors that increase the risk of unhealthy eating or that are associated with this type of eating are similar to the factors associated with substance addiction. Although research has shown that certain individual characteristics are associated with food addiction--specifically being female, being older than 35 years and being overweight or obese⁹⁰--research on the specific factors that increase the risk that an individual will develop food addiction is limited. However, there is extensive research on the risk factors for obesity and the diagnosable eating disorders (especially BED) that may shed light on some of the individual characteristics and environmental conditions that increase the risk for developing food addiction.

Obesity is highly heritable⁹¹ and numerous genes have been implicated⁹² as well as in the development of BED.⁹³ Biological risk factors and correlates of excessive eating include reduced availability of dopamine receptors in certain brain regions⁹⁴ and deficiency of or insensitivity to certain hormones that help regulate food intake and fat storage in the body.⁹⁵ A number of psychological factors, including certain personality traits,⁹⁶ psychiatric disorders,⁹⁷ psychological pressures or stress,⁹⁸ diminished cognitive ability and impaired executive functioning⁹⁹ have been associated with an increased risk of obesity and BED. Risk factors external to the individual, such as the caloric-density,¹⁰⁰ price,¹⁰¹ availability¹⁰² and portion sizes¹⁰³ of foods may spur excess eating, as can a stressful home life,¹⁰⁴ the experience of trauma¹⁰⁵ and poverty.¹⁰⁶ As is true of maternal use of addictive substances like tobacco, alcohol and other drugs, which increases the risk of substance use in the offspring,¹⁰⁷ early (pre-

* Planning, problem solving, decision making.

or perinatal) exposure to maternal unhealthy eating is associated with the development of obesity in offspring.¹⁰⁸ Also consistent with findings related to early age of onset of substance use increasing the risk of substance use and addiction,¹⁰⁹ a young age of initiation of unhealthy eating (including being overweight and dieting) may contribute to the risk of developing food addiction or associated symptoms.¹¹⁰

Biological Characteristics

There is a considerable body of research examining the biological factors that contribute to obesity and certain other forms of disordered eating. Research on the specific biological risk factors for the development of food addiction itself is limited; however, there is some understanding of the biological mechanisms that facilitate or sustain food addiction symptoms.* However, in cases where excessive or disordered eating can be traced to particular genetic or medical conditions (e.g., Prader Willi Syndrome,¹¹¹ monosomy 1p36,¹¹² disorders associated with mutations of the leptin gene or its receptor¹¹³), a food addiction diagnosis typically would not be applicable or appropriate.

Twin and adoption studies, which often are used to study genetic influences on behavior and disease, have shown that up to 70 percent of human variation in degree of overweight and obesity may be determined by heredity.¹¹⁴ Likewise, BED has been found to run in families and to be moderately heritable.¹¹⁵

Genetic Factors Associated with Overweight, Obesity and BED. Nearly 100 genes have been implicated in the determination of body weight,[†] with 22 of them consistently[‡] reported to be linked to obesity.¹¹⁶ Genes more subtly associated with eating behaviors and with a predisposition to obesity also have been identified.¹¹⁷ Variants of GHRL, a gene that codes for precursors to ghrelin (a hormone linked to hunger) and obestatin (a hormone linked to satiety) have been associated with obesity, metabolic syndrome[§] and BED.¹¹⁸ Certain variants of the gene for ghrelin and the neurotransmitter serotonin are found significantly more frequently in individuals with BED and are associated with a significant risk of developing BED.¹¹⁹ The “fat mass and obesity associated” (FTO) gene has been linked to overweight, obesity and increased caloric intake, particularly among people of East Asian and European descent.¹²⁰ The melanocortin 4 receptor, which regulates appetite and energy control, accounts for three to five percent of severe obesity in some groups of children and adults¹²¹ and mutations on this gene are strongly tied to binge eating.¹²² OPRM1, a gene associated in some studies with alcohol and opioid addiction, plays a role in preference for sweet and fatty foods and, in turn, overeating and higher BMI.¹²³ Variations in the number of copies of certain genes¹²⁴ and genetic influences on the sense of taste and food preferences¹²⁵ have been associated with obesity. There also is some evidence that meal quantity, frequency and timing may partly be determined by genes.¹²⁶

Hormonal Factors Associated with Weight Gain and Obesity. Certain hormonal disruptions have been associated with weight gain. Cushing’s syndrome, which occurs when a tumor of the pituitary or adrenal gland or elsewhere in the body produces excessively high levels of adrenocorticotrophic hormone, or cortisol, often is characterized by obesity. Low levels of testosterone or growth hormone, polycystic ovarian syndrome, and a tumor in the pancreas that causes elevated insulin levels are associated with weight gain. Disturbances of the thyroid** have been associated with weight gain as well; however,

* These will be discussed in more detail in a later section of the paper.

† Among these are the melanocortin 4 receptor gene, GHRL, and the dopamine receptor gene DRD2 TaqIA.

‡ In five or more studies.

§ Metabolic syndrome is a cluster of symptoms including central adiposity, hypertriglyceridemia, hypertension, low levels of high-density lipoprotein (HDL) cholesterol, and elevated fasting plasma glucose levels.

** A gland that releases hormones that, among other functions, influences the rate at which calories are burned.

thyroid hormone levels usually are normal in obese people.¹²⁷ Deficiency in leptin--a hormone secreted by fat cells that dampens hunger--has been associated with severe obesity in children.^{* 128}

Variations in Brain Structure and Function Associated with Weight Gain and Disordered Eating.[†]

Research suggests that differences in individuals' sensitivity and responses to food images and cues in the environment predict weight gain, such that elevated reward responsivity in certain regions of the brain to food cues predicts later increases in weight.¹²⁹ Reward sensitivity--a measure of a person's capacity to experience pleasure and reward--is related to overeating, a preference for sweet and fatty foods and food craving, which are characteristics associated with higher BMI, BED and obesity.¹³⁰ A recent study found individual differences in sensitivity to food advertising that related to future weight gain. Participants who demonstrated greater activation in certain areas of the brain in response to viewing food commercials showed greater increases in BMI over a one-year period.¹³¹

Aside from existing differences in brain function that might relate to eating behaviors, certain structural characteristics in the brain also can play a role in eating patterns and weight gain. When the ventromedial region of the hypothalamus in the brain--the "satiety" center--is damaged as a result of cancer, trauma or other causes, it can lead to overeating.¹³² Frontotemporal dementia, a disorder associated with the breakdown of the brain's gray matter in the frontal, temporal and insular cortices, is known to alter eating patterns and has been associated with binge eating.¹³³ Certain antipsychotic medications are associated with weight gain and uncontrolled eating.¹³⁴

Psychological Factors

A range of personality traits and psychological tendencies that frequently are found in those with substance addiction also are associated with obesity, BED and other forms of disordered eating. Most notably, impulsivity is associated with food addiction, higher BMI, binge eating and BED,¹³⁵ and the related characteristic of novelty seeking has been associated with BED and obesity.¹³⁶ A lack of perseverance or persistence has been associated with food addiction, higher BMI and obesity.¹³⁷ A tendency toward "delay discounting"¹³⁸--a preference for smaller immediate rewards over larger delayed rewards[‡]--is common among those with food addiction¹³⁹ and obesity,¹⁴⁰ as it is among those with substance¹⁴¹ and gambling¹⁴² addiction.

Individuals with food addiction are more prone than others of the same weight who do not meet criteria for food addiction to report eating to alleviate bad feelings,¹⁴³ a strong motivator for substance use found in many people with substance addiction.¹⁴⁴ They also are more likely to demonstrate higher levels of depression, negative mood and emotional dysregulation, and lower self-esteem.¹⁴⁵

A number of clinical psychiatric and personality disorders are associated with food addiction and disordered eating. Childhood attention-deficit/hyperactivity disorder (ADHD)¹⁴⁶ and symptoms of posttraumatic stress disorder (PTSD)¹⁴⁷ are higher among those who meet criteria for food addiction compared to those who do not meet the criteria. The majority of people who have ever had bulimia nervosa (85 percent) or BED (79 percent) has met criteria for another psychiatric disorder in their lifetime. Psychiatric disorders are somewhat more likely to precede the development of BED than to follow it.¹⁴⁸ An analysis of multiple studies found that 29 percent of people with BED had a personality

* Most obese individuals, however, have higher than normal levels of leptin. These high levels of leptin do not lead to thinness because people with obesity develop leptin insensitivity, a failure of the central nervous system to respond to leptin signals.

† Neurobiological factors that underlie weight gain, obesity, and certain food-related addictive processes will be discussed in greater detail later in this paper.

‡ Outcomes decrease in their perceived value, or their effectiveness to control behavior, when delayed.

disorder of any kind, with 12 percent having avoidant personality disorder, 10 percent having obsessive-compulsive personality disorder and 10 percent having borderline personality disorder.¹⁴⁹ Depression,¹⁵⁰ dysthymia (chronic low mood)¹⁵¹ and anxiety disorders¹⁵² also have been associated with BED. Importantly, the majority of these psychiatric and personality disorders also co-occur with substance addiction.¹⁵³

Stress. As is true in the case of substance use, a critical risk factor for obesity and disordered eating is acute and chronic stress, which is strongly associated with unhealthy eating patterns and weight gain.¹⁵⁴ The endocrine system, particularly the hypothalamic-pituitary-adrenal (HPA) axis, plays an important role in an individual's response to stress, regulates feeding behavior and energy storage and distribution, and is strongly implicated in addictive processes. Stress-induced stimulation of the HPA, via the corticotropin-releasing factor,¹⁵⁵ may produce neurobiological reactions that promote compulsive overeating as well as substance use.¹⁵⁶

Stress can derive from many sources but is a common feature of individuals with eating and addictive disorders. A study of adult women found that a history of physical or sexual abuse in childhood or adolescence was associated with higher weight and with food addiction in adulthood.¹⁵⁷ Research also has found higher rates of food addiction among adult women with symptoms of lifetime PTSD compared to women with no PTSD symptoms or trauma histories; the relationship between food addiction and PTSD increased with the number of PTSD symptoms and was strongest for those with a younger age of onset of PTSD symptoms.¹⁵⁸ One study found that, compared to women without psychiatric disorders, those with BED had higher rates of sexual abuse, physical abuse and bullying by peers--all sources of chronic stress.¹⁵⁹ Other research shows that children raised in homes with more conflict and neglect are at increased risk of overweight and obesity.¹⁶⁰ Finally, inadequate or erratic sleep patterns, a common stressor, may contribute to overeating and weight gain,¹⁶¹ and are associated with the use of addictive substances.¹⁶²

Early Exposure

There are well documented associations between early exposure to addictive substances and an increased risk of substance use, and between early initiation of substance use and an increased risk of addiction.¹⁶³ As is true of substance use,¹⁶⁴ individuals who are exposed to overeating and unhealthy eating in their family or social peer groups may be at increased risk of such behavior themselves, suggesting that overeating may in part be a learned behavior.¹⁶⁵ A widely-cited study from 2007 found a significantly increased risk of obesity in individuals with obese family members or peers in their social networks, sometimes referred to as "social clustering,"¹⁶⁶ which may be attributed to similar social norms around food, eating and acceptable body size.

Research also demonstrates an increased risk of obesity in childhood and adulthood among those who are exposed to diets rich in carbohydrates or fat in utero and during childhood.¹⁶⁷ Preliminary research suggests that food addiction symptoms in children are associated with higher BMIs.¹⁶⁸ However, research on the risk of early exposure with regard to the consumption of highly palatable, "addictive" foods and the later development of food addiction is not yet available. Still, it is possible that early exposure to or consumption of food ingredients with high addictive potential may alter the brain in ways similar to addictive substances like nicotine, alcohol and other drugs, increasing the risk of food addiction later in life.¹⁶⁹

Availability and Accessibility

Many factors* have contributed to the increase in overweight and obesity in the United States and other developed countries in recent years.¹⁷⁰ However, the most frequently identified driver of this public health epidemic is the increase in the availability and accessibility of a wide variety of low priced, heavily promoted, hyper-palatable and energy-dense foods that are offered in large portion sizes.¹⁷¹ Greater availability and accessibility of addictive substances and of venues to engage in addictive behaviors (tobacco and alcohol outlets, gambling casinos) are well-documented risk factors for risky substance use, gambling and addiction.¹⁷² The tremendous increase in the availability and accessibility of unhealthy food in recent years likewise might help to explain much of the increase in overweight and obesity.

Energy (caloric) intake has increased significantly over the past several decades, from an average of 1,955 per day in the early 1970's to 2,195 per day in the late 2000's.¹⁷³ Eating more energy-dense food (food with more calories for a given weight) is associated with the consumption of more calories and with weight gain.¹⁷⁴ Portion sizes have risen in concert with rates of obesity, and research has shown that people respond to larger servings of food by eating more.¹⁷⁵ Consumption of sugar-sweetened beverages (such as sodas, sports drinks and sweetened coffees and teas) make up an increasingly larger percentage of total calories consumed in the U.S.,¹⁷⁶ and the consumption of such sugar-sweetened beverages is associated with weight gain and obesity.¹⁷⁷ While there has been an overall decline in such consumption in recent years, primarily due to reduced soda consumption, the consumption of other sugar-sweetened beverages has been increasing.¹⁷⁸

Easy access to a wide variety of foods also increases food consumption, as visible, varied and readily accessible food such as that served in fast food venues is consumed more quickly.¹⁷⁹ In the United States, the cost of sweets, fats and high calorie beverages fell substantially in relation to fresh vegetables and fruit between 1985 and 2000,¹⁸⁰ which may make unhealthy foods especially attractive to people living in poverty or on very tight budgets.

The abundance of enticing but unhealthy food stimuli within the context of societal pressure to diet, be thin and overvalue appearance contributes to body dissatisfaction and low self-esteem--important predictors of binge eating and other food-related disorders.¹⁸¹ That, coupled with our evolutionary-based tendency to store calories and fat,¹⁸² may help to explain in part the recent rise in obesity and the prevalence of other food-related disorders.

Shared Neurobiological Characteristics Underlying Certain Forms of Disordered Eating and Substance Addiction

Efforts in recent years to better understand substance addiction increasingly has focused on the neurobiological basis of addiction. Examining the structural and functional variations in the brains of those who are vulnerable to addiction, who engage in substance use, and who have become addicted has been helpful in understanding the seeming intractability of the disease and in reducing some of the stigma associated with those who have it. Despite the growing understanding of the physiological mechanisms underlying the addictive process, diagnosis and assessment of the disease remains tied to behavioral and subjective criteria rather than biomarkers. Still, the appeal of identifying and potentially manipulating the physiological underpinnings of addiction has led some researchers to explore the ways in which certain unhealthy eating behaviors and their neurobiological effects might resemble or overlap with these processes.

* Including increases in sleep deficits, endocrine disruptors (substances that disturb hormone regulation), psychotropic medication use, and higher maternal age at childbirth.

The brain has evolved to reward and reinforce behaviors that are necessary for survival such as those that serve the fundamental drives of hunger, thirst and sex.¹⁸³ The rewards--either an increase in positive feelings or a reduction in negative feelings--inherently motivate the individual to repeat these behaviors in the future.¹⁸⁴ Addictive substances and engagement in certain other compulsive behaviors can affect this reward process, altering the structure and function of the brain, and driving continued substance use or continued engagement in the compulsive behavior despite adverse consequences.¹⁸⁵ The addictive process essentially kicks in when the positive reinforcement that accompanies early stages of substance use (pleasurable and comforting effects) largely shifts to negative reinforcement (the need to consume the substance in order to prevent or mitigate negative feelings and symptoms).¹⁸⁶

Despite some critical differences between the processes involved in the development and expression of addiction involving substances versus food,^{*}¹⁸⁷ the similarities in the mechanisms of action within the brain's circuitry that are involved in reward, motivation and decision-making are striking, lending support to the conceptualization of certain forms of overeating or compulsive eating as a potential manifestation of addiction.¹⁸⁸ Research on food addiction has highlighted the addicting nature of certain types of food or food components, specifically those that are hyper-palatable, calorie-dense, and nutrient-poor (most processed foods). But the ingestion of these foods alone is not sufficient to account for food addiction, since most people consume them without demonstrating addiction symptoms. Animal research suggests that it is not only individual vulnerabilities toward addiction, but also access to these foods and the pattern in which they are consumed that facilitate the addiction process. The research also shows that repeated, intermittent access to hyper-palatable foods leads to classic addiction symptoms in animals, including withdrawal when the food is no longer available, tolerance or the dampening of reward with continued or repeated consumption, and behavioral indicators of craving.¹⁸⁹

More than 100 chemicals in the brain and body (including the neurotransmitters dopamine, GABA, norepinephrine, serotonin)¹⁹⁰ and certain peptide hormones and amino acids¹⁹¹ (including orexin,¹⁹² leptin,¹⁹³ insulin,¹⁹⁴ ghrelin,¹⁹⁵ neuropeptide Y¹⁹⁶ and melanocortin) play critical roles in regulating energy balance, appetite, food intake and weight. Neurons in the ventral tegmental area and the nucleus accumbens, brain regions where the rewarding nature of drugs and other reinforcing agents is processed, have been found to have receptors for these appetite-related chemicals,¹⁹⁷ and research suggests that these chemicals may play a role in alcohol and other drug addiction.¹⁹⁸

The brain chemical most closely associated with rewarding stimuli and strongly implicated in many addictive disorders is dopamine.¹⁹⁹ Dopamine is particularly significant in driving feeding behavior and in exemplifying the potential link between certain unhealthy eating behaviors and addiction.²⁰⁰

One way to think about this link is to explore the anecdotal evidence regarding the seeming overlap and competition between the ingestion of food and addictive substances, both of which involve the release of dopamine and the eventual downregulation (reduced sensitivity) of dopamine pathways upon continued or repeated ingestion.²⁰¹ Individuals who misuse or are addicted to cocaine, amphetamines, methamphetamines, Ecstasy, and to some extent nicotine often eat less, are thin and have nutritional deficiencies.²⁰² Likewise, obese individuals tend to be at lower risk of substance addiction; this may be due to competition between food and addictive substances activating similar reward-related circuits in the brain,²⁰³ but more research is needed to better understand this complex phenomenon. Research on animals supports the cross-sensitization of certain food- and drug-related rewarding stimuli with studies of rats showing that sugar may act on the same neural mechanisms as amphetamines²⁰⁴ and ethanol (alcohol)²⁰⁵ and may lead to physiological dependence and withdrawal symptoms.²⁰⁶

* Such as the fact that multiple physiological systems throughout the body are implicated in food-related processes whereas what we know of substance-related addiction (with the exception of alcohol) primarily involves neural circuits in the brain.

Overeating and obesity are characterized by a heightened dopamine response and sensitivity to rewards, both with regard to the cues for food and food itself.²⁰⁷ A number of studies have linked certain variants of dopamine's DRD2 receptor gene, which is associated with substance addiction,²⁰⁸ to obesity* and to BED.²⁰⁹ With regard to food addiction specifically, one study found that adults who met criteria for food addiction had a genetic predisposition related to stronger dopamine signaling strength and, by implication, a greater sensitivity to reward and motivation for reinforcing stimuli, compared to adults of similar age and BMI who did not meet food addiction criteria.²¹⁰ Another study, which administered a stimulant medication† (known to have appetite suppressing effects) to individuals with and without food addiction found that individuals with food addiction reported stronger food craving and greater appetite after tasting their favorite snack than those without food addiction, even in the presence of the appetite suppressing stimulant.²¹¹

Despite these and related findings highlighting the important role of reward sensitivity in those with food addiction, the exact role that dopamine plays in overeating, obesity and food addiction is complex.²¹² Individuals with low levels of reward response or sensitivity may have a genetic tendency toward lower baseline levels of dopamine or fewer receptors for the dopamine neurotransmitter, reduced activation of the dopamine pathway in the brain as a result of drugs that block dopamine receptors, or dopamine deficiency resulting from the brain's adaptation to or compensation for the dopamine increases induced by excessive eating (or substance use).²¹³ Some research suggests that low levels of dopamine in the brain may impede one's ability to inhibit impulses and may increase the risk that an individual will engage in excessive eating of highly palatable foods to boost reward-related dopamine levels.²¹⁴ This "dopamine deficiency" theory has been examined extensively with regard to addictive drugs, wherein naturally low levels of dopamine, reduced activation of the dopamine pathway, or dopamine deficiency resulting from the brain's adaptation to prolonged exposure to addictive substances can result in attempts to boost dopamine levels via the ingestion of reinforcing or rewarding addictive substances.²¹⁵

In addition to dopamine, other chemicals produced by the body that are similar to the active ingredients in marijuana and opioids (i.e., endogenous cannabinoids, or endocannabinoids, and endogenous opioids)--and critically involved in substance-related addiction--also are involved in regulating food consumption.²¹⁶ The brain's endocannabinoid system, which plays a complex but significant role in driving addiction involving marijuana, nicotine, alcohol and opioids,²¹⁷ also is important in appetite and feeding behavior. The system works by modulating the effects of other neurotransmitters that play an important role in appetite regulation and food craving.²¹⁸ Supporting the overlap between substance and food addiction, several clinical trials have found that the pharmaceutical rimonabant, which blocks certain cannabinoid receptors, leads to significant weight loss²¹⁹ and to reduced drug-seeking behavior in those with addiction.‡²²⁰

The endogenous opioid system is also strongly implicated in substance-related addiction and certain symptoms consistent with food addiction.²²¹ Research suggests that administration of opioid-blocking drugs may reduce eating in the short term. While opioids do not appear to affect hunger or taste directly, they seem to affect how much pleasure is derived from foods, which in turn can influence how much is eaten.²²² Rats who had been allowed repeated and excessive consumption of sugar have demonstrated withdrawal symptoms during abstinence that mimic the behavioral and neurochemical symptoms of opioid withdrawal, suggesting that food dependence can develop much like opioid dependence, and highlighting the role of the natural opioid system in food-related addiction.²²³ There also is some

* Notably, studies of overweight rather than obese individuals have not found the same association with these genetic variants.

† Methylphenidate.

‡ This medication has not been approved for use in the U.S. and it has been removed from the market in Europe due to potentially serious side effects.

evidence of sugar binges and cravings in individuals in recovery from opioid dependence, providing further support for the role of the opioid system in food-related addiction.²²⁴ Despite the importance of endogenous cannabinoids and opioids in feeding behavior, and their obvious relation to drug addiction, research is not yet available on the link between these chemicals and disordered eating or food addiction in particular.

Another type of brain chemical involved in motivated behaviors, including substance use and feeding, is the neuropeptide orexin²²⁵ (or hypocretin). Orexin regulates arousal and appetite and stimulates food intake. It is strongly involved in drug seeking that is stimulated by external cues and in food seeking that is reward based (hedonic, rather than homeostatic, or based on energy needs) and that occurs beyond natural caloric or nutrition needs.²²⁶

Two key regions of the brain, the lateral hypothalamus and the ventromedial hypothalamus, colloquially known as the “hunger” and “satiety” centers, respectively, are instrumental in modulating appetite.²²⁷ Other brain regions, such as other parts of the hypothalamus, parts of the midbrain, forebrain and brain stem--many of which are involved in substance addiction--are important in regulating food intake as well.²²⁸ For example, exposure to food-related cues stimulates a desire to eat via activation of specific regions of the brain--including the amygdala, insula, orbital frontal cortex and the striatum--that are associated with reward and that also are activated by exposure to drug-related cues.²²⁹ These regions of the brain, particularly the insula and the orbital frontal cortex, also are implicated in food cravings and preference for palatable foods;²³⁰ when the normal functioning of these brain regions is disrupted, overeating and weight gain may occur.²³¹

Implications and Recommendations for Policy, Practice and Research

Food addiction, BED and obesity are distinct but related health problems that typically share the common behavior of excessive consumption of certain types of foods. As such, recommendations for addressing these disorders through policy, practice and research overlap to some extent. However, given how much is still unknown about these disorders, research should explore not only their shared characteristics (both among the food-related disorders and between the food-related disorders and substance addiction), but also their unique features, risk factors, and consequences. Findings from this research can help in the design and implementation of effective and evidence-based policy, prevention and treatment interventions.

Regardless of the extent to which food addiction can be framed in the same context as addiction involving tobacco, alcohol and other drugs, much can be learned from the many strategies that have been used to prevent and reduce substance-related addiction and its costly health and social consequences.²³²

As is true of substance addiction, the best approach to addressing the compulsive eating of certain foods that are associated with an increased risk of food addiction and, in some cases, obesity, is a comprehensive one that targets all the domains of influence on an individual’s addictive behavior, including biological and personal influences, as well as social and environmental influences.

On an individual level, biological and personal risk factors for disordered eating (including food addiction) are difficult to prevent. But to help stave off more severe expressions of food addiction, obesity and related disorders such as BED, risk factors and symptoms can be identified and addressed early through routine screening conducted throughout the lifespan and can be minimized by lifestyle changes and adaptive coping. Screenings should be performed by health professionals trained in evidence-based approaches to screening and with the knowledge of how to intervene effectively if a patient presents with signs and symptoms of risk.

Individual-level interventions, however, are not enough to stem the tide of rising rates of obesity or to prevent and reduce instances of food addiction. Restricting the response to these problems to early detection and intervention would miss key opportunities to prevent the development of obesity or food addiction before clinical interventions are even warranted. As is the case for preventing tobacco-, alcohol- and other drug-related addiction and their adverse health, social and economic consequences, broader public health measures must be employed to really make a difference.²³³

The modeling of unhealthy eating by peers, family members, celebrities and other role models; the overabundance of highly accessible unhealthy foods; and the attributes and properties of certain foods themselves increase the risk for obesity and may contribute to the risk for food addiction. As such, each is a potential target for prevention. Using tobacco control and, to some extent, alcohol prevention as a model, much can be done to change the landscape of eating in this country. As has occurred with varying levels of success in relation to tobacco and alcohol, public health initiatives should be implemented to reduce the appeal and accessibility of unhealthy and potentially addictive foods and to shift the focus from blaming people for lacking willpower to acknowledging the strong pull of the object of the addiction itself.²³⁴ To the extent that certain highly palatable and calorie-dense foods or food components increase the risk of food addiction and obesity, population-wide interventions that influence their composition, marketing and accessibility can help to prevent and reduce the incidence of food- and weight-related disorders.²³⁵ In contrast, a focus on individual vulnerabilities independent of environmental influences may be more useful from a clinical or treatment perspective and more applicable to those already demonstrating symptoms of disordered eating.²³⁶

As is true of substance addiction, the particular explanatory focus for food addiction (e.g., on the individual's vulnerabilities versus the food environment) has important implications for public attitudes toward individuals with addiction and support of policies and interventions aimed at addressing it. In the case of tobacco, the tobacco industry typically is assigned relatively more blame for the adverse effects of smoking and nicotine addiction than are smokers, whereas in the case of alcohol, those who misuse it are more likely than alcohol manufacturers to be held accountable for its adverse effects.²³⁷ With regard to food addiction, a national survey of adults found that respondents who were presented with a food addiction explanatory model of obesity (which likened food addiction to the physiological processes of drug addiction and described the condition in accordance with its diagnostic criteria^{*}) versus an explanatory model that was contrary to an addiction perspective, demonstrated less stigma and blame toward obese people and attributed less psychopathology to them.²³⁸

A significant dietary shift toward the consumption of less highly processed and calorie-dense food is needed in the U.S. (and globally) to stem the tide of obesity and symptoms associated with food addiction. Part of the onus for that shift is on the individual but, from a public policy perspective, much can be done to modify the production and marketing of food and the incentives that the food industry currently has to produce and market foods that, for many people, are high in addictive potential. The aim of such policies would be to make unhealthy and potentially addictive food constituents less easily accessible and less alluring to the public, and especially to children.²³⁹

Environmental and policy-level interventions that reduce accessibility and restrict marketing long have been used to reduce demand for addictive substances such as tobacco and alcohol. Likewise, policymakers increasingly are being urged by researchers and health care professionals to use similar

^{*} For example, food-addicted individuals were described as having neurobiological vulnerabilities similar to those with substance addiction, leading to uncontrollable, compulsive food cravings that may overshadow the motivation to engage in other activities. Participants read about a person with food addiction who was described as being compelled to seek and consume foods, especially high-fat, high-calorie foods, even in the face of negative consequences and despite repeated attempts to stop.

techniques to help curtail unhealthy or addictive eating and its adverse and costly health and social consequences.²⁴⁰ Following a public health approach to prevention, and based largely on approaches that have proven effective in preventing and reducing risky substance use and addiction, the following measures can help to reduce the risk and the adverse impact of food addiction, obesity and certain eating disorders:

Improve Public Awareness

Educating the public about the individual and environmental factors that increase the risk of food addiction, binge eating and obesity is one component of a comprehensive strategy for helping people make healthy food choices and avoid food options that compromise health. Such educational efforts must be implemented throughout the lifespan via population-wide public information campaigns that support the development of attitudes and social norms that foster healthy beliefs about acceptable body size, portion size and food consumption.

Public information campaigns should be supplemented by policies that allow the public to be fully informed about the contents of the food they buy and eat so that those who are at higher risk for obesity or addictive eating and/or motivated to eat in a healthy manner are supported in their efforts. This can be accomplished by requiring the food industry to improve packaged food ingredient labeling that minimizes the use of confusing and misleading terms and labels (e.g., describing sugar with multiple terms that are unfamiliar to most consumers rather than the simple term, sugar; calculating ingredient quantities and their nutritional values on the basis of unrealistic portion sizes) and requiring the posting of calorie counts and salt content on all food establishment menus, vending machines, alcoholic beverage products, and certain prepared food options in supermarkets and groceries.*²⁴¹

In addition to improving public awareness and assuring informed food choices by requiring greater transparency from the food industry, specific preventive messages and programming should be implemented in communities, schools and health care settings that have the most leverage to influence at-risk populations, which include:

- New or expectant parents who must make critical food choices during pregnancy and, as parents, for their children;
- Children, for whom early education about nutrition can have lasting effects on food choices and eating behaviors;
- Educators, who make important choices about food availability and quality in schools, and who should resist food industry incentives to promote unhealthy foods to students;
- Health professionals, who often have significant sway with patients of all ages, including parents and children, and who should help educate and motivate patients to make healthy food choices.

* In November, 2014, the Food and Drug Administration (FDA) announced a ruling consistent with this recommendation, which was required under the 2010 Affordable Care Act. The rule, which is expected to face legal and political challenges from the food industry, requires calorie information to be listed on menus and menu boards in chain restaurants and similar retail food establishments and vending machines by December 1, 2016. On September 11, 2015, the FDA issued a draft guidance document to help companies comply with the menu labeling rule.

Reduce Accessibility of Addictive Food Components through Incentives, Regulations and Policies

Much like the tobacco and alcohol industries, which rely in part on users of their products to become addicted in order to ensure continued profits,²⁴² the food industry has a vested interest in maintaining and enlarging its customer base by recruiting new consumers of its products and ensuring the continued loyalty of existing consumers. The former typically is accomplished by marketing highly palatable foods directly to young people while the latter is accomplished by ensuring that the food has appealing (and likely addicting) qualities and is highly accessible so that existing consumers will keep coming back for more. To counteract this powerful financial incentive on the part of the food industry to produce and market unhealthy foods, and the powerful pull on the public to consume highly palatable but potentially addictive food products, policymakers should:

- Increase financial incentives to food manufacturers through subsidies and other means to produce more healthy foods, and to groceries, supermarkets and other food venues to offer these foods at lower cost to consumers, thereby ensuring their affordability and accessibility.
- Limit existing financial incentives to farms and food manufacturers for producing large quantities of unhealthy foods that are high in corn starch, fructose, cheese, animal products and other relatively inexpensive and mass-produced ingredients. These highly processed, calorie-dense and nutrient-poor ingredients currently make their way into the majority of food products sold at low cost to consumers.
- Require sustainable and humane farming practices that will necessarily increase the cost of cheese, meat, eggs and other farm products, thereby limiting their disproportionate inclusion in processed food products, despite the health (and environmental) risks.
- Institute zoning restrictions to limit the number of fast food restaurants and other venues for unhealthy foods, particularly in low-income neighborhoods and near schools.
- Levy taxes on high sugar beverages and other unhealthy, highly processed, calorie-dense and nutrient-poor foods to increase their cost and reduce their desirability and accessibility.
- Prohibit direct-to-consumer marketing to children of highly processed foods in schools and in all media venues.
- Hold food industries financially accountable for health care costs associated with the consumption of their products.
- Limit the influence of the food industry on public policy, including the development of federal nutrition guidelines, via campaign finance reform and greater restrictions on lobbying.

Incorporate Screening and Evidence-Based Interventions into Routine Health Care Practice

Even when effective prevention initiatives are in place, too often people at risk for or experiencing addiction are not identified in time to receive needed interventions. Routine health care screening can identify individuals at risk and help to ensure that they are provided evidence-based interventions to reduce risk and adverse health outcomes. With regard to food addiction specifically, several versions of

the YFAS can be employed to screen for symptoms and make a “diagnosis,”* including one designed specifically for children (YFAS-C).²⁴³

With regard to effective interventions, there is limited research focused specifically on food addiction. However, several approaches that have been found to be effective in addressing substance addiction have shown promise for helping some individuals control their food intake. Certain medications (e.g., naltrexone, bupropion, baclofen) known to be effective in treating nicotine, alcohol and opioid dependence appear to help some obese and overweight individuals control their food intake and lose weight.²⁴⁴ Likewise, certain psychotherapeutic interventions, such as cognitive-behavioral therapy (CBT), typically employed with individuals who have substance-related addiction, also are effective in the treatment of BED and may be useful in treating individuals with food addiction.²⁴⁵ Notably, unlike substance addiction treatment which employs CBT to foster avoidance or abstinence of addictive substance use, CBT for the treatment of unhealthy eating focuses on enhancing the individual’s sense of control over food intake.²⁴⁶

Fund and Conduct Quality Research on Food Addiction, as well as Binge Eating Disorder and Obesity

The research base for establishing the existence of food addiction is considerable, but many questions remain regarding the utility of the construct, its prevalence, factors that increase its risk and effective interventions and treatments for it. Given the high rates and adverse effects of obesity and other eating disorders, and the largely unchecked influence of the processed food industry on children and adults alike, it is important for researchers to pursue critical areas of inquiry, including, but not limited to, identifying:

- The validity of the food addiction construct; whether it is a useful concept that is distinct from existing measures of other food- and eating-related disorders, and its utility as a diagnosable disorder.
- Valid and reliable diagnostic criteria for food addiction.
- The prevalence of food addiction, as currently conceived, nationally and in at-risk populations.
- The extent to which food addiction co-occurs with obesity, binge eating and other eating disorders, other psychiatric disorders, and other manifestations of addiction, or increases the risk for them.
- The specific food ingredients and modification and processing approaches that may contribute to addictive eating in humans.
- Risk and protective factors for food addiction, BED and obesity, including biologically based sensitivity to highly processed, refined foods.
- Effective prevention and intervention approaches, including the applicability to food addiction of interventions used for obesity, other food-related disorders and for substance addiction.
- Evaluations of the effectiveness of existing and emerging prevention approaches, such as the inclusion of calorie counts on restaurant menus and in other food venues.

* Accessible at: <http://fastlab.psych.lsa.umich.edu/yale-food-addiction-scale/>

Conclusions

The field of addiction has come a long way in recent years, with a growing understanding of the factors that increase the risk that substance use will progress to addiction, the psychological and physiological mechanisms that ensure its maintenance in the face of adverse consequences, and best practices for prevention, intervention and treatment. The scientific evidence pointing to addiction as a health condition rather than a moral failing has helped to chip away at the stigma surrounding the disease and has allowed for innovative public health and clinical approaches to prevention and treatment.

Individuals suffering from another leading public health problem--overweight and obesity--have faced problems similar to those who suffer from substance addiction, including stigma, shame and tremendous difficulty overcoming a significant threat to their health and well-being. For years, eating disorders and obesity were examined primarily through the lens of individual vulnerability, with prevention and interventions focused on changing how the individual interacts with his or her family and food environment. Lessons learned from substance addiction, specifically the power of certain addictive substances to directly affect an individual's brain and behavior, have helped launch a new and potentially fruitful paradigm for understanding and addressing certain cases of obesity and eating disorders. The food addiction model, like that of substance addiction, describes the ways in which certain food properties or ingredients can produce addiction in individuals who are susceptible to their effects and who consume them in a manner that induces the addictive process (i.e., eating certain types of highly palatable, calorie-dense, and nutrient-poor food on an intermittent but repeated basis). It allows for an explanation and an intervention strategy for those cases of disordered eating that are not adequately accounted for by existing psychological or medical causes.

Clearly the term addiction can be over-applied and misused, and caution is especially needed when using it in the context of behaviors like eating and sex that must be engaged in repeatedly for survival. However, when certain types of food are overconsumed despite detrimental effects on health and survival, the concept of addiction becomes relevant. As researchers and clinicians in both the addiction and the eating disorders/obesity fields continue to explore the validity of the food addiction construct, its physiological mechanisms, and its potential applications to prevention, intervention and treatment, the desire for a simple and powerful explanation and solution to our nation's weight problem must be balanced with the need to explore these issues via a reasoned, deliberate and scientifically sound approach.

SELECTED RESOURCES FOR ADDITIONAL INFORMATION

A number of research-based institutions and advocacy organizations have undertaken initiatives to help educate the public, conduct research and inform health professionals and policymakers about overweight, obesity, eating disorders and food addiction. Some examples include:

The Rudd Center for Food Policy and Obesity at the University of Connecticut:

<http://www.uconnruddcenter.org/>

Cornell University Food and Brand Lab:

<http://foodpsychology.cornell.edu/#>

The Food and Addiction Science and Treatment (FAST) Lab at the University of Michigan:

<http://fastlab.psych.lsa.umich.edu/>

The Psychology of Eating And Consumer Health (PEACH) lab at the Perelman School of Medicine at the University of Pennsylvania:

<http://www.peachlab.org/peach/>

The Center for Eating and Weight Disorders at the Perelman School of Medicine at the University of Pennsylvania:

<http://www.med.upenn.edu/weight/index.shtml>

Program for Obesity, Weight and Eating Research (POWER) at the Yale School of Medicine:

http://psychiatry.yale.edu/research/programs/clinical_people/power.aspx

Weight Management and Eating Disorders Research Program at Washington University:

<http://www.psychiatry.wustl.edu/wilfleylab>

The Child Study Center at New York University Langone Medical Center:

<http://nyulangone.org/locations/child-study-center>

The Weight-control Information Network (WIN) of the National Institutes of Health:

<http://www.niddk.nih.gov/health-information/health-communication-programs/win/Pages/default.aspx>

The National Eating Disorders Association (NEDA):

<https://www.nationaleatingdisorders.org/>

The Binge Eating Disorder Association (BEDA):

<http://bedaonline.com/>

Obesity Action Coalition (OAC):

<http://www.obesityaction.org/>

The Obesity Society:

<http://www.obesity.org/home>

Appendix A

Yale Food Addiction Scale*

Individuals who endorse three or more symptoms on the scale and demonstrate clinically significant impairment or distress within the past 12 months:

1. I find that when I start eating certain foods, I end up eating much more than planned.
2. I find myself continuing to consume certain foods even though I am no longer hungry.
3. I eat to the point where I feel physically ill.
4. Not eating certain types of food or cutting down on certain types of food is something I worry about.
5. I spend a lot of time feeling sluggish or fatigued from overeating.
6. I find myself constantly eating certain foods throughout the day.
7. I find that when certain foods are not available, I will go out of my way to obtain them. For example, I will drive to the store to purchase certain foods even though I have other options available to me at home.
8. There have been times when I consumed certain foods so often or in such large quantities that I started to eat food instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy.
9. There have been times when I consumed certain foods so often or in such large quantities that I spent time dealing with negative feelings from overeating instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy.
10. There have been times when I avoided professional or social situations where certain foods were available, because I was afraid I would overeat.
11. There have been times when I avoided professional or social situations because I was not able to consume certain foods.
12. I have had withdrawal symptoms such as agitation, anxiety, or other physical symptoms when I cut down or stopped eating certain foods. (Please do NOT include withdrawal symptoms caused by cutting down on caffeinated beverages such as soda pop, coffee, tea, energy drinks, etc.)
13. I have consumed certain foods to prevent feelings of anxiety, agitation, or other physical symptoms that were developing. (Please do NOT include consumption of caffeinated beverages such as soda pop, coffee, tea, energy drinks, etc.)
14. I have found that I have elevated desire for or urges to consume certain foods when I cut down or stop eating them.
15. My behavior with respect to food and eating causes significant distress.
16. I experience significant problems in my ability to function effectively (daily routine, job/school, social activities, family activities, health difficulties) because of food and eating.
17. My food consumption has caused significant psychological problems such as depression, anxiety, self-loathing, or guilt.
18. My food consumption has caused significant physical problems or made a physical problem worse.
19. I kept consuming the same types of food or the same amount of food even though I was having emotional and/or physical problems.
20. Over time, I have found that I need to eat more and more to get the feeling I want, such as reduced negative emotions or increased pleasure.
21. I have found that eating the same amount of food does not reduce my negative emotions or increase pleasurable feelings the way it used to.

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

22. I want to cut down or stop eating certain kinds of food.
23. I have tried to cut down or stop eating certain kinds of food.
24. I have been successful at cutting down or not eating these kinds of food.
25. How many times in the past year did you try to cut down or stop eating certain foods altogether?

Appendix B

DSM-5 Diagnostic Criteria for Substance Use Disorders and for Feeding and Eating Disorders^{*}

Substance Use Disorders

A problematic pattern of substance use leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period (severity is specified as mild (2-3 symptoms), moderate (4-5 symptoms), or severe (6 or more symptoms)):

- The substance is often taken in larger amounts or over a longer period than was intended.
- There is a persistent desire or unsuccessful effort to cut down or control use of the substance.
- A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects.
- Craving, or a strong desire or urge to use the substance.
- Recurrent use of the substance resulting in a failure to fulfill major role obligations at work, school, or home.
- Continued use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use.
- Important social, occupational, or recreational activities are given up or reduced because of use of the substance.
- Recurrent use of the substance in situations in which it is physically hazardous.
- Use of the substance is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance.
- Tolerance, as defined by either of the following:
 - A need for markedly increased amounts of the substance to achieve intoxication or desired effect.
 - A markedly diminished effect with continued use of the same amount of the substance.
- Withdrawal, as manifested by either of the following:
 - The characteristic withdrawal syndrome for that substance (as specified in the DSM- 5 for each substance).
 - The substance (or a closely related substance) is taken to relieve or avoid withdrawal symptoms.

Binge Eating Disorder

- Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
 - Eating, in a discrete period of time (for example, within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances.
 - A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

^{*} American Psychiatric Association. (2013). *The Diagnostic and Statistical Manual of Mental Disorders: DSM 5*. Arlington, VA: Author.

- The binge-eating episodes are associated with three (or more) of the following:
 - Eating much more rapidly than normal.
 - Eating until feeling uncomfortably full.
 - Eating large amounts of food when not feeling physically hungry.
 - Eating alone because of feeling embarrassed by how much one is eating.
 - Feeling disgusted with oneself, depressed, or very guilty afterwards.
- Marked distress regarding binge eating is present.
- The binge eating occurs, on average, at least once a week for three months.
- The binge eating is not associated with the recurrent use of inappropriate compensatory behavior as in bulimia nervosa and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.

Bulimia Nervosa

- Recurrent episodes of binge eating, characterized by both of the following:
 - Eating, in a discrete amount of time (e.g., within any two-hour period), an amount of food that is definitely larger than what most individuals would eat in a similar period of time under similar circumstances.
 - A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).
- Recurrent inappropriate compensatory behavior in order to prevent weight gain (e.g., self-induced vomiting; misuse of laxatives, diuretics, or other medications; fasting; or excessive exercise).
- The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for three months.
- Self-evaluation is unduly influenced by body shape and weight.
- The disturbance does not occur exclusively during episodes of anorexia nervosa.

Anorexia Nervosa

- Restriction of energy intake relative to requirements, leading to a significantly low body weight in the context of age, sex, developmental trajectory, and physical health.
- Intense fear of gaining weight or becoming fat, or persistent behavior that interferes with weight gain, even though at a significantly low weight.
- Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or persistent lack of recognition of the seriousness of the current low body weight.
- Specify whether restricting type or binge-eating/purging type.

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