

Obesity and Its Relationship to Addictions: Is Overeating a Form of Addictive Behavior?

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Obesity is a major public health problem and notoriously difficult to treat. There are many parallels between obesity/overeating and addictions to alcohol and drugs. This paper discusses similarities between obesity and addictive disorders, including common personality characteristics, disruptive behavior syndromes, and brain mechanisms. Although there are important differences between overeating and other addictive behaviors, an addiction model of overeating may effectively inform prevention and treatment of obesity. (Am J Addict 2009;18:439–451)

In recent years, the prevalence of obesity and concern about its impact on public health have grown dramatically. In the United States, 33% of men and 35% of women were classified as obese, with a body mass index (BMI) of 30 or greater, in 2005–2006.¹ In most cases, obesity results from a caloric imbalance—the number of calories consumed exceeds the number of calories expended.^{2,3} Sedentary lifestyles and the wide availability of low cost, calorie dense foods contribute to this energy imbalance,⁴ but what makes individuals consume more food than they need to survive? Despite the seemingly simple relationship between energy balance and body weight, obesity is a complex and refractory condition. Overeating has long been likened to drug and alcohol addictions in the popular imagination.⁵ As new technologies for studying brain activity have emerged, scientists have begun to seriously investigate the theory that overeating can be a form of addictive behavior.⁶

Traditionally, the term addiction was applied to excessive ingestion of substances leading to physical dependence, characterized by tolerance and withdrawal.⁵ Compulsive engagement in behaviors such as gambling, sex, or eating was not considered a true addiction, because the drive to engage in these behaviors was considered purely psychological. The

conceptual model of substance addictions has begun to change, however, with an increasing emphasis on the behavior of substance use rather than the chemical properties of the substances themselves.⁷ It is also becoming clear that repetitive engagement in many behaviors can lead to physiological changes in the brain similar to those observed in drug dependent individuals.⁶ According to recent models, addiction is a syndrome that can be expressed through a variety of specific behaviors.⁸ Overeating may be one of those behaviors.

This paper examines literature supporting a relationship between obesity and addictions and discusses evidence for and against an addiction model of overeating. First, we address whether obesity/overeating should be considered a psychiatric disorder with similar diagnostic criteria to substance use disorders. We then discuss the implications of epidemiological and clinical studies showing positive and negative associations between obesity and substance use disorders in the general population. Next we explore underlying characteristics and potential brain mechanisms associated with both overeating and addictions and point out important differences between overeating and addictions to drugs and alcohol. Finally, we discuss implications of an addictions model of overeating to prevention and treatment of obesity.

IS OBESITY A PSYCHIATRIC DISORDER?

Obesity is associated with a host of medical problems, and treating obesity can result in improved health.⁹ Theoretically, treatment of obesity is simple: reduce food intake and increase physical activity. Yet few obese people achieve significant weight reduction, and even fewer manage to maintain weight loss. This contradiction suggests that the drive to consume food beyond what is necessary to maintain physical functions can outweigh other considerations.

The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)¹⁰ criteria for substance dependence appear to have external validity when applied to the excessive overeating that can lead to obesity. Obese individuals often eat more than they intended and make frequent yet ultimately unsuccessful efforts to control overeating. Obesity can reduce an individual's ability to participate in a full range of social, occupational,

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and recreational activities. Many individuals continue to overeat despite knowledge that overeating causes obesity and may contribute to or complicate serious health problems. These parallels have led to the proposal that obesity, or the overeating that causes it, be included in the upcoming DSM-V, with diagnostic criteria modeled on those for substance dependence.^{11,12} DSM-IV currently includes a Binge Eating Disorder diagnosis, which requires loss of control over eating and consumption of large quantities of food over a short period of time.¹⁰ There is currently no diagnostic category for chronic overeating. Some researchers have expressed reservations

about the creation of an additional diagnosis along the lines of “Overeating Disorder” or “Food Dependence.” They point out that food, unlike drugs and alcohol, is necessary for life, that it is impossible to abstain from food, and that physiological markers of dependence like tolerance, withdrawal, and craving for food are not well characterized or understood at this time.¹³

Table 1 shows DSM-IV criteria for substance dependence and potential parallel symptoms for a hypothetical “Overeating Disorder,” illustrating some of the similarities and differences between overeating and substance dependence. Clearly, not

TABLE 1. DSM-IV criteria for substance dependence diagnosis and parallel criteria for a possible disorder of overeating

Substance Dependence Criterion	Parallel Criterion for “Overeating Disorder”
<p>(1) Tolerance, including need for more of a substance to achieve the same effect or a diminished effect when using the same amount of the substance over time. Example: Alcohol dependent individual does not feel intoxicated after consuming entire 6-pack in an evening.</p> <p>(2) Withdrawal, including characteristic syndrome of withdrawal symptoms for specific substance or use of the substance or a similar one to relieve or prevent those symptoms. Example: Heroin dependent individual experiences dysphoria, nausea, sweating, and insomnia when she can’t obtain heroin, takes oxycontin to compensate.</p> <p>(3) Individual frequently takes more of a substance than intended or takes it over a longer period of time than planned. Example: Alcoholic plans to stop at the local bar for one beer, ends up staying until closing and having several drinks.</p> <p>(4) Repeated unsuccessful efforts to reduce substance use or persistent desire to do so. Example: Cocaine dependent individual repeatedly vows to stop using at the start of the day, but ends up using by the end of the day</p> <p>(5) Substantial amount of time spent obtaining, using, or recovering from use of substance. Example: Cannabis dependent individual spends hours calling his various contacts to locate available marijuana, travels 2 hours to get it, then smokes for most of the weekend.</p> <p>(6) Individual abandons or cut back on social activities, work or family responsibilities, and recreational interests in order to use substances. Example: Drug user stops associating with non-drug using friends.</p> <p>(7) Substance use continues in spite of associated physical and psychological problems. Example: Alcohol dependent individual continues to drink after being diagnosed with hypertension and gastric ulcers.</p>	<p>(1) Physiological tolerance unlikely, but some individuals feel need for increased quantities of food in order to feel satisfied. Example: Overweight or obese individual feels hungry after a large meal.</p> <p>(2) Comparable withdrawal syndrome not yet identified, but dieters and other individuals deprived of food report psychological preoccupation with food, and some individuals use substances such as nicotine or stimulants to suppress appetite. Example: Dieter feels lethargic and depressed, smokes or drinks caffeinated beverages to compensate.</p> <p>(3) Food is often consumed in larger amounts or over a longer time than was intended. Example: Dieter plans to have one small serving of ice cream, but ends up having eating an entire pint.</p> <p>(4) Obese individuals who overeat often have a persistent wish to reduce or control how much they eat or try repeatedly to eat less. Example: Repeated, unsuccessful diets or regaining weight after successful diet are the norm for most obese individuals.</p> <p>(5) Overeaters can spend substantial time shopping for food, eating and snacking, and recovering from physical and psychological effects of overeating (eg, nausea, guilt about eating too much) Example: Obese individual snacks throughout the day in addition to or instead of eating regular meals.</p> <p>(6) A range of activities may be abandoned or reduced because of consequences of overeating (ie, obesity) and accompanying decreased mobility, increased social anxiety, etc. Example: Obese individual stops participating in sports or going to the beach because of embarrassment about weight.</p> <p>(7) Overeating continues in spite of associated physical and psychological problems. Example: Obese individual continues to eat candy after being diagnosed with type II diabetes mellitus.</p>

all overweight or obese persons would meet these criteria. Instead, this putative disorder may be reserved for a subset of overweight and obese individuals who exhibit chronic loss of control of overeating, similar to that observed with substance use disorders.

POPULATION ASSOCIATIONS BETWEEN OBESITY AND ADDICTIONS

If we assume that overeating is an addictive disorder and that overeating is more likely among individuals with elevated body weight, we might expect to find positive associations between obesity and substance use disorders in the general population and in clinical samples. On the other hand, overeating and use of substances may fulfill similar physical or psychological needs, making individuals who overeat less prone to other addictive behaviors.

Findings from Epidemiological Samples

Epidemiological studies examining relationships between obesity and substance use disorders yield ambiguous results, summarized in Table 2. Using a sample of over 40,000 individuals from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), Petry et al.¹⁴ found higher lifetime rates of alcohol use disorders with increasing BMI starting in the overweight range and increasing across BMI categories. Examining the same data separately by gender showed higher prevalence of lifetime alcohol abuse and dependence among overweight and obese men relative to normal weight men, with no association between BMI and lifetime alcohol use disorders in women.¹⁵ Overweight and obese women, but not men, were less likely to report past-year alcohol abuse than their normal weight counterparts.¹⁵ A study that surveyed respondents from 13 countries found an association between obesity and decreased likelihood of past-year alcohol use disorders in the United States but not the other 12 countries or the overall sample.¹⁶ The differing relationships observed for lifetime and past-year alcohol use disorders^{14–16} raise the possibility that recovery from an alcohol use disorder increases vulnerability to weight gain. Consistent with this hypothesis, John et al.¹⁷ found increased risk for overweight among former but not current male heavy alcohol drinkers. The gender differences may also suggest different patterns of drinking among women and men, with men adding alcohol calories to their diets and women substituting alcohol calories for other sources of energy.¹⁸

Relationships between BMI and illicit drug use disorders are more difficult to characterize, because epidemiological studies including significant numbers of individuals with drug use disorders are rare. Simon and colleagues¹⁹ found obesity to be associated with lower likelihood of a lifetime substance use disorder diagnosis, a category that included both alcohol and illicit drug use disorders. One study using NESARC data and controlling for life stressors and medical conditions found obesity was associated with lower odds of a past-year drug

dependence diagnosis but not a past-year diagnosis of drug abuse.²⁰ Analysis of the same data without controlling for life stress and medical conditions found no relationship between body weight and drug use disorders, over the lifetime or in the past year.¹⁴

Epidemiological studies of relationships between obesity and nicotine dependence also yield mixed results. Among men, John et al.²¹ found an association between overweight or obesity and a history of daily smoking but not current smoking. Other studies, however, find a negative association between body weight and the likelihood of both lifetime and past-year nicotine dependence among men.^{15,20} In contrast, another study found current smokers to have comparable obesity risk to nonsmokers, but risk for obesity increased with number of cigarettes per day among smokers.²² A study of young adults found higher rates of smoking among obese individuals relative to their overweight and normal weight counterparts, and obese smokers smoked more cigarettes per day than overweight or normal weight smokers.²³

Findings from Clinical Samples

Rates of substance use disorders are elevated among patients seeking treatment for obesity, but rates of overweight and obesity among patients receiving treatment for substance use disorders are similar to the general population. Findings from clinical samples are summarized in Table 3.

Kalarchian et al., found that 32.6% of bariatric surgery candidates reported a lifetime history of any substance use disorder,²⁴ more than twice the rate observed in the general population.²⁵ They found a striking difference between the lifetime and current prevalence of substance use disorders among bariatric surgery candidates—only 1.7% reported a current substance use disorder. Although the chronology of the development of obesity relative to substance use disorders was not explored, this striking difference in lifetime versus past-year prevalence raises the possibility of overeating substituting for substance use among some individuals.²⁴ Chart reviews of women seeking weight management treatments find lower rates of past-year alcohol and marijuana use with increasing body weight.^{26,27}

Examining the converse relationships, 54% of patients in a residential alcohol treatment program were overweight or obese.²⁸ A post-mortem study from Sweden found 45% of deceased individuals with illicit drug use disorders were overweight or obese, comparable to the Swedish general population rate.²⁹

Overall, the variation in findings across substances and across studies makes it difficult to draw any firm conclusions about potential relationships between obesity and addictions. It is important to note that relationships are complicated by the different potential physical effects of different substances on body weight. Alcohol, unlike illicit drugs and nicotine, has calories, which could contribute to higher body weight.¹⁸ Nicotine increases metabolism,³⁰ potentially contributing to lower body weight.

TABLE 2. associations between substance use disorders and body mass index (BMI) in epidemiologic studies

Substance Use Disorder	Overweight (BMI = 25.0–29.9)	Obesity (BMI ≥ 30.0)
<i>Epidemiologic Samples</i>		
Alcohol Use Disorders		
Barry & Petry ¹⁵		
Lifetime alcohol abuse	Positive in men	Positive in men
Lifetime alcohol dependence	Positive in men	Positive in men
Past year alcohol abuse	Negative in women	Negative in women
Past year alcohol dependence	No association	No association
John et al. ¹⁷		
Current heavy alcohol users	No association	No association
Former heavy alcohol users	Positive in men	No association
Petry et al. ¹⁴		
Lifetime alcohol abuse	Positive	Positive ^a
Lifetime alcohol dependence	No association	No association
Past year alcohol abuse	No association	No association
Past year alcohol dependence	No association	No association
Scott et al. ¹⁶		
Past year alcohol dependence	n.a.	Positive in U.S. only ^b
Illicit Drug Use Disorders		
Petry et al. ¹⁴		
Lifetime drug use disorder	No association	No association
Past year drug use disorder	No association	No association
Pickering et al. ²⁰		
Past year drug abuse	No association	No association ^a
Past year drug dependence	No association	Negative ^a
Simon et al. ¹⁹		
Lifetime substance use disorder ^c	n.a.	No association ^b
Nicotine Dependence		
Barry & Petry ¹⁵		
Lifetime nicotine dependence	Negative in men/Positive in women	Negative in men
Past year nicotine dependence	Negative in men	Negative
Chiolero et al. ²²		
Current smokers	Negative	No association
Former smokers	Positive in men	Positive
John et al. ²⁶		
Current smokers	No association	No association
Former smokers	Positive	Positive
Pickering et al. ²⁰		
Past year nicotine dependence	Negative in men	Negative in men ^a
Zimlichman et al. ²³		
Current smokers	No association	Positive

If not specified, reference group is normal weight (BMI = 18.5–24.9).

^aObese category includes BMI = 30.0–39.9.

^bBMI range of reference group is 18.5–29.9.

^cIncludes alcohol and drug use disorders.

SIMILARITIES BETWEEN OBESITY AND ADDICTIVE DISORDERS

Despite the ambiguity of epidemiologic findings, research aimed at understanding individual differences that increase vulnerabilities to obesity and addictive disorders reveal similar personality characteristics, likelihood of disruptive behavior

disorders, and functional brain abnormalities. These similarities are summarized in Table 3.

Personality Characteristics

Several studies have utilized the Temperament and Character Inventory (TCI)³¹ to measure personality characteristics in overweight and obese patients and patients with substance

TABLE 3. Associations between substance use disorders and body weight in clinical samples

Study	Sample	Finding
Studies Examining Rates of Substance Use Disorders in Patients Seeking Obesity Treatment		
Kalarchian et al. ²⁴		
Any lifetime substance use disorder	Candidates for weight loss surgery	Higher prevalence of lifetime substance use disorders than general population
Any past year substance use disorder	Candidates for weight loss surgery	Lower prevalence of lifetime substance use disorders than general population
Kleiner et al. ²⁶		
Past year alcohol use	Female weight management patients	Lower rates of past year alcohol use than in general population
Warren et al. ²⁷		
Past year marijuana use	Female weight management patients	Lower rates of past year marijuana use with increasing bmi
Studies Examining Rates of Overweight/Obesity in Substance Abusing Samples		
Jarvis et al. ²⁸		
Alcohol dependence	Residential alcohol treatment patients	Rates of overweight/obesity comparable to general population
Rajs et al. ²⁹		
Illicit drug use disorders	Deceased illicit drug users	Rates of overweight/obesity comparable to general population

dependence. Two TCI scales have shown associations with both obesity and substance use disorders. The novelty seeking scale reflects excitement in response to novel or rewarding stimuli. The self-directedness scale assesses self-acceptance, responsibility, goal directedness, and autonomy. On the TCI, obese persons are more likely than normal weight individuals to have high novelty seeking scores and lower self-directedness scores.³² Obese weight management participants who score high on novelty seeking are less successful at losing weight than those with lower scores.³²

Similar findings are noted among substance abusing populations. Substance dependent individuals have higher novelty seeking scores and lower self-directedness scores than individuals without substance use disorders.^{33,34} Patients with substance dependence who score high on the novelty seeking scale of the TCI are more likely to be dependent on two or more substances.³⁵ Among individuals with a family history of alcoholism, those who score higher on novelty seeking are more likely to be diagnosed with alcohol dependence, although novelty seeking is not a strong predictor of alcohol dependence in individuals without familial risk.³⁶

Normal weight and overweight women who experience food cravings are more likely to also report a history of alcohol abuse or dependence and to score high on the TCI novelty seeking scale.³⁷ These findings suggest that a stable tendency to respond strongly to novel stimuli may make the experience of eating flavorful foods and/or using drugs more

pleasurable, increasing likelihood of overconsumption. Self-directedness may allow individuals to restrain or moderate tendencies toward overeating and substance use, decreasing vulnerability to obesity or substance addictions.

Overweight and obese individuals with binge eating symptoms had high scores on a personality measure of impulsivity and consumed more of a liquid meal supplement after an 8 hour fast.^{38,39} Impulsivity scores were correlated with the amount of meal supplement consumed.³⁸ Other studies utilize the Iowa Gambling Task (IGT),⁴⁰ a measure of impulsivity and decision making that requires inhibition of impulsive responses. Overweight and obese persons perform more poorly on the IGT than normal weight peers,⁴¹ and similarly to individuals with substance use disorders.⁴² Delay discounting is a measure of relative preference for small immediate rewards versus larger delayed rewards, an aspect of impulsivity. Obese women show greater delayed discounting than normal weight women, although body weight is not associated with delay discounting in men.⁴³

Substance use disorders are also associated with elevated scores on measures of impulsivity.^{44,45} Individuals with dependence on alcohol or drugs perform more poorly on the IGT than comparable individuals without substance use disorders.^{46–49} Long term abstinent alcoholics also respond impulsively on the IGT.⁵⁰ Individuals with cocaine, opiate and alcohol use disorders have higher rates of delay discounting than controls without substance use disorders.^{51–54} These

findings suggest that inability to suppress impulses plays a role in overeating and addictions.³⁸

Associations with Disruptive Behavior Disorders

Children with behavior disorders characterized by impulsivity and inattention, such as attention deficit hyperactivity disorder (ADHD) and conduct disorder, appear to be at increased risk for addictions as well as overweight and obesity in both clinical and community samples.^{55,56} Overweight children are more impulsive than their normal weight peers.⁵⁷ Overweight boys report more trouble focusing attention, and overweight boys and girls report more difficulty shifting attention compared to normal weight children.⁵⁷ Over half of children hospitalized for treatment of obesity meet criteria for ADHD.⁵⁸ Among adults treated for obesity, ADHD is highly prevalent, particularly among the extremely obese (BMI \geq 40).⁵⁹ In adult women, ADHD symptoms are associated with overeating, which in turn is associated with higher BMI.⁶⁰

Similarly, rates of ADHD and conduct disorder are also markedly elevated among patients receiving treatment for substance use disorders.⁶¹ Prospective studies suggest that ADHD in childhood increases risk for initiating substance use by age 14 and developing nicotine dependence and alcohol and cannabis use disorders by age 18.⁵⁶ ADHD, conduct disorder, and substance use disorders are often thought to represent varying manifestations of an underlying externalizing syndrome.⁶² The findings described above suggest that overeating and obesity could also be included in the externalizing syndrome. The concept of an underlying externalizing disorder can help explain comorbidity among more specific disorders and links between childhood behavior disorders and addictions or obesity in adulthood.⁶²

Externalizing disorders have been linked to executive functions deficits, including inhibition, self-monitoring and planning.^{63,64} Overeating fits the model of impaired executive ability quite well as it includes disinhibition of eating, a breakdown in self-monitoring of food intake, and failure to anticipate consequences (ie, weight gain). Recent studies find executive deficits in obese compared to normal weight persons.^{65,66} Similarly, executive deficits are commonly associated with a variety of substance use disorders.^{67–69}

Brain Mechanisms

Substance use disorders appear to arise from brain circuitry that promotes behaviors necessary for survival, including eating and sex. Neurotransmitters in these brain regions are sensitive to the reinforcing properties of food but also respond to chemicals in psychoactive substances.^{70,71} The last decade has seen the introduction and refinement of sophisticated brain imaging techniques that have revealed common neurological mechanisms underlying overeating and substance use.⁷²

Reinforcing effects of drugs and food arise from neuronal activity within the mesocorticolimbic dopamine system, including the ventral tegmental area where cell bodies of dopaminergic neurons originate, and the basal forebrain

(especially the nucleus accumbens, amygdala, and frontal and limbic cortices), where dopamine is released into synapses.^{73,74}

Food intake, particularly consumption of highly palatable and calorie dense foods, stimulates dopamine activity, either directly or indirectly through action on other neurotransmitters, creating a subjective feeling of pleasure and satisfaction.⁷⁵ Blocking dopamine receptors increases appetite and causes weight gain, suggesting that overeating may be an effort to compensate for blunting of the pleasurable response to eating. The dopamine receptor most associated with eating behaviors is the subtype 2 (D2) receptor.⁷⁰ Wang and colleagues⁷⁶ used positron emission tomography (PET) scans to compare metabolic activity in the brains of ten severely obese individuals to ten normal weight individuals. Obese individuals had significantly fewer dopamine D2 receptors than their normal weight counterparts, and the higher an individual's body mass, the fewer D2 receptors were observed.⁷⁶ These findings suggest that low dopamine activity could be the mechanism of vulnerability to obesity as individuals with fewer D2 receptors have to eat more in order to experience the rewarding properties of food intake. Alternatively, some researchers have speculated that tolerance to the pleasurable effects of food could arise from chronic overeating if elevated dopamine levels lead to downregulation of dopamine receptors.⁷²

Similar to food, drugs of abuse stimulate release of dopamine in the mesocorticolimbic dopamine system,⁷⁷ which causes a subjective experience of pleasure and euphoria that makes drug use highly reinforcing.⁷⁸ Neuroimaging research suggests that acute drug administration increases dopamine release from neurons, but D2 receptor availability is also significantly reduced in the brains of individuals with chronic drug and alcohol use disorders.⁷⁹ It therefore appears that chronic drug administration leads to a significant decrease in dopaminergic activity over time through downregulation in response to acute dopamine stimulation.

Some researchers have hypothesized a common "Reward Deficiency Syndrome" characterized by low numbers of D2 receptors and propensity for compulsive engagement in rewarding behaviors, such as drug use and eating.^{72,80} Other genetic and environmental variables contribute to the vulnerability to a specific compulsive behavior. For instance, obese people have greater increases in brain activity in response to mouth, lip, and tongue sensations, which could make eating particularly rewarding.⁸¹ Exposure to and availability of high calorie foods versus drugs or alcohol and association of positive experiences with a particular behavior can affect the specific choice of reinforcer as well.

Findings regarding common personality characteristics, behavior disorders, and brain mechanisms support an addiction model of obesity and shed light on difficulties obese people face when attempting to lose weight. Recognition of individual differences in vulnerability to substance use disorders has advanced understanding of addictions, and a similar model for overeating could prove useful in understanding the development of obesity.

DIFFERENCES BETWEEN OBESITY AND ADDICTIONS

Although there are many similarities between obesity and addictions, there are also important differences. An addiction model of obesity assumes that overeating is the primary cause of obesity. Although obesity is usually associated with food intake greater than is needed to maintain normal body weight, human beings vary greatly in their caloric needs, and the human metabolism resists significant changes in body weight by adjusting to changes in food intake.⁸²

General Differences

Addictive drugs do not generally serve a beneficial homeostatic or reproductive purpose.⁷⁷ In contrast, food is necessary for survival.¹³ There is evidence that the quantity of food consumed by the average person has not increased substantially as obesity rates have risen, and that changes in the nutritional content of diets and declines in physical activity may be more significant contributors to elevated body weight.⁸³ From an evolutionary perspective, overeating is an adaptive behavior that promotes survival and reproduction by replenishing energy stores depleted through strenuous physical activity.⁸⁴ It may only be the rapid decline in human energy requirements, coupled with greater availability of food, that make overeating a maladaptive behavior in modern society. Although effects of drugs and alcohol, including pain relief, relaxation, mental stimulation, and even mild loss of inhibition, may promote survival and reproduction when used in moderation, it is difficult to identify a survival benefit conferred by overindulgence in drugs or alcohol, similar to that once offered by overeating. In fact, excessive alcohol and drug use reduces fitness by dampening unpleasant but adaptive emotions like fear.⁸⁵

Role of Leptin in Appetite and Body Weight Regulation

Vulnerabilities to obesity and substance use disorders are at least partially hereditary. The hormone leptin is secreted by fat tissue, and as fat is added to the body, organisms respond by eating less.⁸⁶ Leptin therefore appears to be a key regulator of body weight.^{87,88} Some obese individuals have a genetic mutation that reduces leptin production, preventing them from regulating food intake in response to increased body fat. Individuals with leptin deficiency have stronger than normal appetites and feel hungry much of the time. For them, overeating is not primarily related to pleasure and reward, but is a response to inaccurate hunger cues. Reducing body fat leads to a decline in leptin production and a corresponding increase in appetite, possibly explaining why permanent weight loss is so difficult.⁸⁹ However, similar to the downregulation in D2 receptors thought to occur when dopamine activity increases, sensitivity to leptin appears to decline with chronic elevations in activity. Chronic overeating may therefore continue after weight gain even in individuals without pre-existing leptin

deficiencies as their brains become less sensitive to leptin's signal to reduce intake.^{90,91}

Unlike dopamine, which is involved in a variety of rewarding activities, leptin appears to be specifically related to regulation of food intake and body weight. However, elevated leptin levels have been associated with cravings for alcohol during alcohol withdrawal, leading to speculation that leptin interacts with the brain reward system in producing its effects on food and alcohol intake.⁹²

Ghrelin

Ghrelin is a peptide hormone secreted by the stomach that stimulates appetite.⁹³ Ghrelin levels are high when the stomach is empty and decline following meals.^{94,95} Ghrelin levels are positively associated with feelings of hunger, and intravenous administration of ghrelin induces hunger and food intake in humans.⁹³ Circulating ghrelin levels in the bloodstream are negatively associated with body mass in humans, and weight loss through dieting results in increasing ghrelin levels, suggesting that ghrelin is involved in regulation and maintenance of body weight.⁹⁵ Obese individuals show abnormalities in diurnal variation of ghrelin, and ghrelin concentration in the blood is abnormally high among individuals with Prader-Willi syndrome, a condition marked by extreme appetite and obesity.⁹⁶ These findings suggest that abnormalities in secretion of ghrelin can lead to overeating and weight gain. Ghrelin, like leptin, may also play a role in alcohol use disorders. Alcohol dependent individuals have higher ghrelin levels than persons without alcohol dependence, and ghrelin levels increase during alcohol withdrawal.⁹⁷ Unlike leptin, however, ghrelin levels do not appear to be associated with alcohol cravings.⁹⁷

Just as abnormalities in leptin and ghrelin secretion appear to be related more strongly to dysregulation of eating than to substance use disorders, there are other genetic predisposition specific to dysfunctional substance use. For example, the best characterized genetic factors affecting alcoholism are the alcohol and aldehyde dehydrogenase genes that determine an individual's ability to metabolize alcohol.⁹⁸ Each gene has an allele that results in accumulation of acetaldehyde, a toxic metabolite that causes an unpleasant flushing reaction and leads most people who have the allele to avoid alcohol.⁹⁹ This genetically determined variation in response to the specific chemical properties of alcohol does not have a parallel in overeating.

The differences discussed above indicate that the addiction model of overeating does not adequately account for some aspects of obesity. Furthermore, there are characteristics of addictions to alcohol and drugs that vary by specific substance⁹⁸ and do not appear relevant to overeating.

PREVENTION AND TREATMENT IMPLICATIONS OF AN ADDICTIONS MODEL OF OBESITY

Although overeating differs in some respects from other addictive behaviors, the many similarities can inform prevention

and treatment recommendations. For some individuals, substance addiction can be a chronic, relapsing condition, requiring lifelong management to prevent relapse.¹⁰⁰ If obesity arises from an addictive pattern of eating, we might anticipate that at least a subset of persons who lose weight will require lifelong management of eating behaviors in order to maintain their losses.

Prevention

Given the challenges of treating addictions, prevention efforts may be the best way to reduce the impact of addictive behaviors on individuals and society. For instance, smoking cessation is extremely difficult, yet smoking rates have declined dramatically in the last quarter century due to prevention efforts and interventions to make smoking more difficult.⁶ Education about the dangers of smoking begins in elementary school, and physicians are expected to inquire about smoking, advise patients of its dangers, and provide information about smoking cessation. It is illegal to sell cigarettes to minors, and cigarettes are regulated and taxed to make them less accessible, particularly to young people. Smoking has been banned in most public settings in most states. Concurrent with these changes, smoking rates have declined from 42% in 1965 to 21% in 2004.¹⁰¹

Similar efforts have been suggested to prevent obesity. Education on healthy eating and the calorie and fat content of foods could be provided to children and to their parents to help them plan healthy meals.¹⁰² Researchers and public policy experts have recommended restricting sales of snack foods and soft drinks to children, particularly in schools, taxing unhealthy, high calorie foods, and subsidizing healthy foods such as fruits and vegetables.^{103,104} There may also be some benefit to restricting or banning eating in public settings not specifically designed for eating, such as offices, classrooms, theaters, and public transportation.

Pharmacological Treatments

Medications that are effective in reducing substance use are also effective for reducing food intake. Topiramate is thought to inhibit dopamine release in the mesocorticolimbic system, thus dampening the rewarding effects of alcohol.¹⁰⁵ Topiramate similarly appears to be effective in producing weight loss in obese individuals.¹⁰⁶

Rimonabant, a drug that blocks the cannabinoid receptors, has been tested as a treatment for both substance use disorders and obesity.¹⁰⁷ Preliminary findings suggested it was effective as a treatment for nicotine and alcohol dependence, as well as reducing food intake and improving lipid and blood sugar levels in obese patients.¹⁰⁸ However, rimonabant was associated with a high incidence of serious psychiatric side effects, leading the U.S. Food and Drug Administration to deny its approval.¹⁰⁹

Behavioral Treatments

Some behavioral treatments for addictions can also help obese individuals control food intake. Examples of treatments

that may be effective for both obesity and substance use disorders include cognitive behavioral therapy, 12-step programs, and contingency management.

Cognitive Behavioral Therapy

Cognitive-behavioral therapies (CBT) for drug and alcohol addictions have been widely studied. CBT is based on social learning theory and the premise that addictive behaviors are learned.^{110,111} The first stage of CBT for addictions is a detailed evaluation of thoughts, feelings and beliefs that contribute to substance use. CBT treatment focuses on training clients to modify thoughts and feelings and develop skills for recognizing and coping with cravings, triggers, and pressures to use, and for planning ahead for situations that increase risk for substance use.¹¹² Relapse prevention is an important component of CBT as well.¹¹³ CBT interventions have been effectively applied to alcohol, cocaine, and marijuana use disorders.^{114–120}

CBT treatments for obesity generally include three components, dietary change, increased physical activity, and behavior therapy techniques such as goal-setting, self-monitoring, stimulus control, and behavioral contracting.^{121–125} In addition to weight loss itself, a goal of cognitive behavioral interventions is lifestyle changes that increase the likelihood losses will be maintained. Similar to CBT for substance use disorders, clients are taught to identify thoughts and feelings that contribute to overeating, and they are taught skills for preventing and dealing with relapse. Cognitive behavioral interventions have demonstrated efficacy in promoting weight loss.^{126–129}

Twelve Step Groups

Self-help groups based on the Alcoholics Anonymous (AA) model are among the most widely used interventions for individuals trying to overcome addictions to alcohol and drugs. These groups, which focus on completing twelve steps to recovery, are based on a model of addiction as a physical, mental, and spiritual disease.¹³⁰ Key tenets of AA and 12-step groups for drug use disorders (Narcotics Anonymous, Cocaine Anonymous) are acceptance and surrender. Participants are encouraged to accept the premise that they suffer from a chronic, progressive disease of addiction for which there is no cure, and that complete abstinence from alcohol or drugs is the only alternative to addiction. Participants are asked to surrender their will to that of a “higher power.” Fellowship with other alcoholics or addicts is a crucial component of 12-step groups as well. Participants are assigned a sponsor, usually a more experienced member with a history of recovery, who can help them through the challenges of defeating addiction.

Overeaters Anonymous (OA) is a 12-step program that views obesity as one symptom of compulsive overeating, and compulsive overeating, like alcoholism, is viewed as an addictive disease.¹³¹ Like AA and other 12-step groups, OA emphasizes the mental and spiritual aspects of compulsive overeating and focuses on fellowship, self-acceptance, recognizing the limits of willpower, surrendering to a higher power, and taking a “moral inventory” in the interest of

identifying interpersonal issues that contribute to loss of control over eating. Whereas in AA abstinence is easily defined as complete avoidance of alcohol consumption, the definition is more flexible in OA, as abstinence from food is impossible. Some members abstain from certain foods thought to trigger overeating, such as refined sugar, while others commit to refrain from overeating or binge eating. Despite the popularity of 12-step groups, there is little published research examining the efficacy or effectiveness of OA as a treatment for overeating and obesity.

Contingency Management

Contingency management (CM) is an intervention based on operant conditioning principles that provides tangible reinforcements for target behaviors such as abstinence from drugs, alcohol or nicotine. The key components of CM are identifying a target behavior (eg, drug abstinence), obtaining an objective measure of the behavior (eg, negative urine specimen), and providing reinforcement each time the target behavior is detected. CM using vouchers exchangeable for goods and services has been highly efficacious in promoting addictions treatment retention and extending duration of abstinence from a range of substances.^{132–134} Prize-based CM¹³⁵ reduces the cost of providing material goods by using prize-drawings as the reinforcement. In prize-based CM, individuals are allowed to draw cards from a bowl each time they demonstrate the target behavior. In a typical intervention, about 50% of the cards result in prizes, most of which are worth about \$1, with smaller chances of winning prizes worth \$20 or \$100. Prize-based CM has demonstrated efficacy for improving outcomes in treatment for cocaine, amphetamine/methamphetamine, opiate, alcohol, and nicotine use disorders.^{136–143}

Given its efficacy when applied to a range of substance use disorders, CM may also be an effective treatment for reducing overeating and promoting weight loss. Reinforcement can be provided for achieving weight loss, as well as for activities associated with weight loss such as keeping food and physical activity diaries, purchasing and preparing healthy meals, counting calories and limiting calorie intake, and exercising. CM approaches to weight loss have been effective among children.^{144,145} We currently have studies underway to evaluate the efficacy of CM in promoting weight loss in adults.

CONCLUSION

Amid growing concern about obesity rates and the limited success of weight loss treatments, greater understanding of behaviors that contribute to unhealthy weight gain is necessary. There is a growing body of evidence to support similarities between overeating and substance use disorders, including potential commonalities in symptom presentations, comorbidities, behavioral and personality characteristics, and biological mechanisms. While differences also exist, an addiction based model of overeating provides a compelling

TABLE 4. Characteristics common to individuals with over-weight/obesity and substance use disorders

Personality Characteristics

- Elevated scores on novelty-seeking scale of the Temperament and Character Inventory (TCI)
- Low scores on the self-directedness scale of the TCI
- Higher scores on self-report measures of impulsivity.
- Poorer scores on the Iowa Gambling Task.
- Preference for smaller immediate vs. larger delayed rewards on Delayed Discounting Task.

Disruptive Behavior Disorders

- Higher rates of Attention Deficit Hyperactivity Disorder
- Higher rates of Conduct Disorder
- Deficits on tests of executive functions.

Brain Mechanisms

- Overeating and substance use stimulate mesocorticolimbic dopamine system acutely
- Number of D2 dopamine receptors decreased from normal levels in brains of obese individuals and chronic substance users, suggesting downregulation of receptors with chronic stimulation of dopamine system.

theory for understanding obesity and the difficulties involved in controlling food intake (see Table 4).

The disease model of addictions has reduced some of the stigma attached to drug and alcohol addictions and challenged the view that they represent moral failings.¹⁴⁶ Viewing substance use disorders as psychiatric disorders facilitates greater understanding of the disordered behaviors involved in addictions, particularly compulsive use and loss of control. Similarly, obese individuals are highly stigmatized and their excess weight is often viewed as a sign of irresponsibility and moral weakness.¹⁴⁷ Obesity is sometimes treated as a medical disease, and the treatment for obesity usually includes reducing the amount of food consumed. However, there has been little discussion of the possibility that at least a subset of obese individuals may suffer from a psychiatric disorder that makes it particularly difficult for them to limit food consumption, just as it is difficult for individuals with alcohol or drug dependence to limit consumption of those substances. Some of the differences between overeating and substance dependence may have implications for future definitions of substance use disorders. Physiological tolerance and withdrawal are currently prominent among the symptoms for substance dependence, but are not as salient for overeating. While it could be argued that this weakens the argument for an addiction model of overeating, it may rather be that the current model of substance dependence places too much emphasis on these symptoms. Advances in understanding brain mechanisms of reward may shift the focus to other symptoms, such as loss of control and inability to curtail use.

An addictions model of overeating can inform prevention and treatment efforts to abate the spread of obesity, along with

the medical, psychological, and social consequences of this growing public health problem. As with nicotine, alcohol, and drugs, limiting access to foods that are high in calories and low in nutritive value may be one of the most effective ways to reduce their overconsumption. Pharmacological treatments that make overeating less rewarding, and behavioral treatments that offer alternative rewards, may also prove effective. Further collaborations between experts in the fields of obesity and addictions may prove fruitful in developing accurate models of overeating behavior and using them to design effective interventions to reduce obesity.

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Declaration of interest

The authors report no conflict of interest. The authors alone are responsible for the content and writing of the paper.

REFERENCES

- Ogden CL, Yanovski SZ, Carroll MD, Flegal KM. The epidemiology of obesity. *Gastroenterology*. 2007;132:2087–2102.
- Brownell KD. *The LEARN program for weight management*. 10th ed. Dallas: American Health Publishing Company; 2004.
- Wing RR. Behavioral treatment of obesity. In: Wadden TA, Stunkard AJ, eds. *Obesity Handbook*. New York: Guilford Press; 2000:455–462.
- French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Annu Rev Public Health*. 2001;22:309–335.
- Holden C. 'Behavioral' addictions: do they exist? *Science*. 2001;294:980–982.
- Volkow ND, Wise RA. How can drug addiction help us understand obesity? *Nat Neurosci*. 2005;8:555–560.
- Gawin FH. Cocaine addiction: psychology and neurophysiology. *Science*. 1991;251:1580–1586.
- Shaffer HJ, LaPlante DA, LaBrie RA, Kidman RC, Donato AN, Stanton MV. Toward a syndrome model of addiction: multiple expressions, common etiology. *Harv Rev Psychiatry*. 2004;12:367–374.
- Must A, Spadano J, Coakley EH, Field AE, Colditz G, Dietz WH. The disease burden associated with overweight and obesity. *JAMA*. 1999;282:1523–1529.
- American Psychiatric Association. *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. 4th ed. Washington, DC: American Psychiatric Association; 2000.
- James GA, Gold MS, Liu Y. Interaction of satiety and reward response to food stimulation. *J Addict Dis*. 2004;23:23–37.
- Volkow ND, O'Brien CP. Issues for DSM-V: should obesity be included as a brain disorder? *Am J Psychiatry*. 2007;164:708–710.
- Devlin MJ. Is there a place for obesity in DSM-V? *Int J Eat Disord*. 2007;40 S83–88.
- Petry NM, Barry D, Pietrzak RH, Wagner JA. Overweight and obesity are associated with psychiatric disorders: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Psychosom Med*. 2008;70:288–297.
- Barry D, Petry NM. Associations between body mass index and substance use disorders differ by gender: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Addict Behav*. 2009;34:51–60.
- Scott KM, Bruffaerts R, Simon GE, et al. Obesity and mental disorders in the general population: results from the world mental health surveys. *Int J Obes*. 2008;32:192–200.
- John U, Meyer C, Rumpf HJ, Hapke U. Relationships of psychiatric disorders with overweight and obesity in an adult general population. *Obes Res*. 2005;13:101–109.
- Colditz GA, Giovannucci E, Rimm EB, et al. Alcohol intake in relation to diet and obesity in women and men. *Am J Clin Nutr*. 1991;54:49–55.
- Simon GE, Von Korff M, Saunders K, et al. Association between obesity and psychiatric disorders in the US adult population. *Arch Gen Psychiatry*. 2006;63:824–830.
- Pickering RP, Grant BF, Chou SP, Compton WM. Are overweight, obesity, and extreme obesity associated with psychopathology? Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *J Clin Psychiatry*. 2007;68:998–1009.
- John U, Meyer C, Rumpf HJ, Hapke U, Schumann A. Predictors of increased body mass index following cessation of smoking. *Am J Addict*. 2006;15:192–197.
- Chiolero A, Jacot-Sadowski I, Faeh D, Paccaud F, Cornuz J. Association of cigarettes smoked daily with obesity in a general adult population. *Obesity*. 2007;15:1311–1318.
- Zimlichman E, Kochba I, Mimouni FB, et al. Smoking habits and obesity in young adults. *Addiction*. 2005;100:1021–1025.
- Kalarchian MA, Marcus MD, Levine MD, et al. Psychiatric disorders among bariatric surgery candidates: relationship to obesity and functional health status. *Am J Psychiatry*. 2007;164:328–334; quiz 374.
- Kessler RC, Demler O, Frank RG, et al. Prevalence and treatment of mental disorders, 1990 to 2003. *N Engl J Med*. 2005;352:2515–2523.
- Kleiner KD, Gold MS, Frost-Pineda K, Lenz-Brunsmann B, Perri MG, Jacobs WS. Body mass index and alcohol use. *J Addict Dis*. 2004;23:105–118.
- Warren M, Frost-Pineda K, Gold M. Body mass index and marijuana use. *J Addict Dis*. 2005;24:95–100.
- Jarvis CM, Hayman LL, Braun LT, Schwartz DW, Ferrans CE, Piano MR. Cardiovascular risk factors and metabolic syndrome in alcohol- and nicotine-dependent men and women. *J Cardiovasc Nurs*. 2007;22:429–435.
- Rajs J, Petersson A, Thiblin I, Olsson-Mortlock C, Fredriksson A, Eksborg S. Nutritional status of deceased illicit drug addicts in Stockholm, Sweden—a longitudinal medicolegal study. *J Forensic Sci*. 2004;49:320–329.
- Schechter MD, Cook PG. Nicotine-induced weight loss in rats without an effect on appetite. *Eur J Pharmacol*. 1976;38:63–69.
- Cloninger CR. A systematic method for clinical description and classification of personality variants. A proposal. *Arch Gen Psychiatry*. 1987;44:573–588.
- Sullivan S, Cloninger CR, Przybeck TR, Klein S. Personality characteristics in obesity and relationship with successful weight loss. *Int J Obes*. 2007;31:669–674.
- Hosak L, Preiss M, Halir M, Cermakova E, Csemy L. Temperament and character inventory (TCI) personality profile in amphetamine abusers: a controlled study. *Eur Psychiatry*. 2004;19:193–195.
- Le Bon O, Basiaux P, Streel E, et al. Personality profile and drug of choice: a multivariate analysis using Cloninger's TCI on heroin addicts, alcoholics, and a random population group. *Drug Alcohol Depend*. 2004;73:175–182.
- Conway KP, Kane RJ, Ball SA, Poling JC, Rounsaville BJ. Personality, substance of choice, and polysubstance involvement among substance dependent patients. *Drug Alcohol Depend*. 2003;71:65–75.
- Gruzca RA, Robert Cloninger C, Bucholz KK, et al. Novelty seeking as a moderator of familial risk for alcohol dependence. *Alcohol Clin Exp Res*. 2006;30:1176–1183.

37. Gendall KA, Sullivan PF, Joyce PR, Fear JL, Bulik CM. Psychopathology and personality of young women who experience food cravings. *Addict Behav.* 1997;22:545–555.
38. Galanti K, Gluck ME, Geliebter A. Test meal intake in obese binge eaters in relation to impulsivity and compulsivity. *Int J Eat Disord.* 2007;40:727–732.
39. Nasser JA, Gluck ME, Geliebter A. Impulsivity and test meal intake in obese binge eating women. *Appetite.* 2004;43:303–307.
40. Bechara A, Damasio H, Tranel D, Damasio AR. Deciding advantageously before knowing the advantageous strategy. *Science.* 1997;275:1293–1295.
41. Davis C, Levitan RD, Muglia P, Bewell C, Kennedy JL. Decision-making deficits and overeating: a risk model for obesity. *Obes Res.* 2004;12:929–935.
42. Bechara A, Damasio H. Decision-making and addiction (part I): impaired activation of somatic states in substance dependent individuals when pondering decisions with negative future consequences. *Neuropsychologia.* 2002;40:1675–1689.
43. Weller RE, Cook EW, 3rd, Avsar KB, Cox JE. Obese women show greater delay discounting than healthy-weight women. *Appetite.* 2008;51:563–569.
44. Dom G, D'Haene P, Hulstijn W, Sabbe B. Impulsivity in abstinent early- and late-onset alcoholics: differences in self-report measures and a discounting task. *Addiction.* 2006;101:50–59.
45. Hanson KL, Luciana M, Sullwold K. Reward-related decision-making deficits and elevated impulsivity among MDMA and other drug users. *Drug Alcohol Depend.* 2008;96:99–110.
46. Bechara A, Dolan S, Denburg N, Hindes A, Anderson SW, Nathan PE. Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. *Neuropsychologia.* 2001;39:376–389.
47. Grant S, Contoreggi C, London ED. Drug abusers show impaired performance in a laboratory test of decision making. *Neuropsychologia.* 2000;38:1180–1187.
48. Petry NM, Bickel WK, Arnett M. Shortened time horizons and insensitivity to future consequences in heroin addicts. *Addiction.* 1998;93:729–738.
49. Whitlow CT, Liguori A, Livengood LB, et al. Long-term heavy marijuana users make costly decisions on a gambling task. *Drug Alcohol Depend.* 2004;76:107–111.
50. Fein G, Klein L, Finn P. Impairment on a simulated gambling task in long-term abstinent alcoholics. *Alcohol Clin Exp Res.* 2004;28:1487–1491.
51. Kirby KN, Petry NM, Bickel WK. Heroin addicts have higher discount rates for delayed rewards than non-drug-using controls. *J Exp Psychol Gen.* 1999;128:78–87.
52. Kirby KN, Petry NM. Heroin and cocaine abusers have higher discount rates for delayed rewards than alcoholics or non-drug-using controls. *Addiction.* 2004;99:461–471.
53. Petry NM. Discounting of money, health, and freedom in substance abusers and controls. *Drug Alcohol Depend.* 2003;71:133–141.
54. Vuchinich RE, Simpson CA. Hyperbolic temporal discounting in social drinkers and problem drinkers. *Exp Clin Psychopharmacol.* 1998;6:292–305.
55. Anderson SE, Cohen P, Naumova EN, Must A. Relationship of childhood behavior disorders to weight gain from childhood into adulthood. *Ambul Pediatr.* 2006;6:297–301.
56. Elkins JJ, McGue M, Iacono WG. Prospective effects of attention-deficit/hyperactivity disorder, conduct disorder, and sex on adolescent substance use and abuse. *Arch Gen Psychiatry.* 2007;64:1145–1152.
57. Braet C, Claus L, Verbeken S, Van Vlierberghe L. Impulsivity in overweight children. *Eur Child Adolesc Psychiatry.* 2007;16:473–483.
58. Agranat-Meged AN, Deitcher C, Goldzweig G, Leibenson L, Stein M, Galili-Weisstub E. Childhood obesity and attention deficit/hyperactivity disorder: a newly described comorbidity in obese hospitalized children. *Int J Eat Disord.* 2005;37:357–359.
59. Altfas JR. Prevalence of attention deficit/hyperactivity disorder among adults in obesity treatment. *BMC Psychiatry.* 2002;2:9.
60. Davis C, Levitan RD, Smith M, Tweed S, Curtis C. Associations among overeating, overweight, and attention deficit/hyperactivity disorder: a structural equation modelling approach. *Eat Behav.* 2006;7:266–274.
61. Schubiner H, Tzelepis A, Milberger S, et al. Prevalence of attention-deficit/hyperactivity disorder and conduct disorder among substance abusers. *J Clin Psychiatry.* 2000;61:244–251.
62. Krueger RF, Hicks BM, Patrick CJ, Carlson SR, Iacono WG, McGue M. Etiologic connections among substance dependence, antisocial behavior, and personality: modeling the externalizing spectrum. *J Abnorm Psychol.* 2002;111:411–424.
63. Young SE, Friedman NP, Miyake A, et al. Behavioral disinhibition: Liability for externalizing spectrum disorders and its genetic and environmental relation to response inhibition across adolescence. *J Abnorm Psychol.* 2009;118:117–130.
64. Finn PR, Rickert ME, Miller MA, et al. Reduced cognitive ability in alcohol dependence: Examining the role of covarying externalizing psychopathology. *J Abnorm Psychol.* 2009;118:100–116.
65. Boeka AG, Lokken KL. Neuropsychological performance of a clinical sample of extremely obese individuals. *Arch Clin Neuropsychol.* 2008;23:467–474.
66. Gunstad J, Paul RH, Cohen RA, Tate DF, Spitznagel MB, Gordon E. Elevated body mass index is associated with executive dysfunction in otherwise healthy adults. *Compr Psychiatry.* 2007;48:57–61.
67. Bates ME, Bowden SC, Barry D. Neurocognitive impairment associated with alcohol use disorders: implications for treatment. *Exp Clin Psychopharmacol.* 2002;10:193–212.
68. Fals-Stewart W, Bates ME. The neuropsychological test performance of drug-abusing patients: an examination of latent cognitive abilities and associated risk factors. *Exp Clin Psychopharmacol.* 2003;11:34–45.
69. Verdejo-Garcia A, Perez-Garcia M. Profile of executive deficits in cocaine and heroin polysubstance users: common and differential effects on separate executive components. *Psychopharmacology.* 2007;190:517–530.
70. Del Parigi A, Chen K, Salbe AD, Reiman EM, Tataranni PA. Are we addicted to food? *Obes Res.* 2003;11:493–495.
71. Wise RA. Drug self-administration viewed as ingestive behaviour. *Appetite.* 1997;28:1–5.
72. Wang GJ, Volkow ND, Thanos PK, Fowler JS. Similarity between obesity and drug addiction as assessed by neurofunctional imaging: a concept review. *J Addict Dis.* 2004;23:39–53.
73. Kelley AE, Berridge KC. The neuroscience of natural rewards: relevance to addictive drugs. *J Neurosci.* 2002;22:3306–3311.
74. Koob GF, Le Moal M. Plasticity of reward neurocircuitry and the “dark side” of drug addiction. *Nat Neurosci.* 2005;8:1442–1444.
75. Abizaid A, Gao Q, Horvath TL. Thoughts for food: brain mechanisms and peripheral energy balance. *Neuron.* 2006;51:691–702.
76. Wang GJ, Volkow ND, Logan J, et al. Brain dopamine and obesity. *Lancet.* 2001;357:354–357.
77. Hyman SE, Malenka RC, Nestler EJ. Neural mechanisms of addiction: the role of reward-related learning and memory. *Annu Rev Neurosci.* 2006;29:565–598.
78. Wise RA, Bozarth MA. Brain mechanisms of drug reward and euphoria. *Psychiatr Med.* 1985;3:445–460.
79. Volkow ND, Fowler JS. Addiction, a disease of compulsion and drive: involvement of the orbitofrontal cortex. *Cereb Cortex.* 2000;10:318–325.
80. Blum K, Cull JG, Braverman ER, Comings DE. Reward Deficiency Syndrome. *The American Scientist.* 1996;84:132–145.
81. Wang GJ, Volkow ND, Felder C, et al. Enhanced resting activity of the oral somatosensory cortex in obese subjects. *Neuroreport.* 2002;13:1151–1155.
82. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. *N Engl J Med.* 1995;332:621–628.

83. Blair SN, Nichaman MZ. The public health problem of increasing prevalence rates of obesity and what should be done about it. *Mayo Clin Proc.* 2002;77:109–113.
84. Lieberman LS. Evolutionary and anthropological perspectives on optimal foraging in obesogenic environments. *Appetite.* 2006;47:3–9.
85. Nesse RM, Berridge KC. Psychoactive drug use in evolutionary perspective. *Science.* 1997;278:63–66.
86. Zhang Y, Proenca R, Maffei M, Barone M, Leopold L, Friedman JM. Positional cloning of the mouse obese gene and its human homologue. *Nature.* 1994;372:425–432.
87. Friedman JM, Halaas JL. Leptin and the regulation of body weight in mammals. *Nature.* 1998;395:763–770.
88. Friedman JM. Leptin, leptin receptors, and the control of body weight. *Nutr Rev.* 1998;56:S38–46; discussion S54–75.
89. Friedman JM. The function of leptin in nutrition, weight, and physiology. *Nutr Rev.* 2002;60:S1–14; discussion S68–84, 85–117.
90. Considine RV, Caro JF. Leptin and the regulation of body weight. *Int J Biochem Cell Biol.* 1997;29:1255–1272.
91. Considine RV. Leptin and obesity in humans. *Eat Weight Disord.* 1997;2:61–66.
92. Kiefer F, Jahn H, Jaschinski M, et al. Leptin: a modulator of alcohol craving? *Biol Psychiatry.* 2001;49:782–787.
93. Wren AM, Seal LJ, Cohen MA, et al. Ghrelin enhances appetite and increases food intake in humans. *J Clin Endocrinol Metab.* 2001;86:5992.
94. Cummings DE, Purnell JQ, Frayo RS, Schmidova K, Wisse BE, Weigle DS. A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. *Diabetes.* 2001;50:1714–1719.
95. Klok MD, Jakobsdottir S, Drent ML. The role of leptin and ghrelin in the regulation of food intake and body weight in humans: a review. *Obes Rev.* 2007;8:21–34.
96. Paik KH, Jin DK, Song SY, et al. Correlation between fasting plasma ghrelin levels and age, body mass index (BMI), BMI percentiles, and 24-hour plasma ghrelin profiles in Prader-Willi syndrome. *J Clin Endocrinol Metab.* 2004;89:3885–3889.
97. Kraus T, Schanze A, Groschl M, et al. Ghrelin levels are increased in alcoholism. *Alcohol Clin Exp Res.* 2005;29:2154–2157.
98. Buckland PR. Will we ever find the genes for addiction? *Addiction.* 2008;103:1768–1776.
99. Goldman D, Oroszi G, Ducci F. The genetics of addictions: uncovering the genes. *Nat Rev Genet.* 2005;6:521–532.
100. Leshner AI. Addiction is a brain disease, and it matters. *Science.* 1997;278:45–47.
101. Centers for Disease Control and Prevention. Cigarette smoking among adults—United States, 2004. *Morbidity and Mortality Weekly Report.* 2005;54:1121–1124.
102. Skidmore PM, Yarnell JW. The obesity epidemic: prospects for prevention. *QJM.* Dec 2004;97:817–825.
103. Battle EK, Brownell KD. Confronting a rising tide of eating disorders and obesity: treatment vs. prevention and policy. *Addict Behav.* 1996;21:755–765.
104. Schwartz MB, Brownell KD. Actions necessary to prevent childhood obesity: creating the climate for change. *J Law Med Ethics.* 2007;35:78–89.
105. Chiu YH, Lee TH, Shen WW. Use of low-dose topiramate in substance use disorder and bodyweight control. *Psychiatry Clin Neurosci.* 2007;61:630–633.
106. Bray GA, Hollander P, Klein S, et al. A 6-month randomized, placebo-controlled, dose-ranging trial of topiramate for weight loss in obesity. *Obes Res.* 2003;11:722–733.
107. Muccioli GG. Blocking the cannabinoid receptors: drug candidates and therapeutic promises. *Chem Biodivers.* 2007;4:1805–1827.
108. Janero DR, Makriyannis A. Targeted modulators of the endogenous cannabinoid system: future medications to treat addiction disorders and obesity. *Curr Psychiatry Rep.* 2007;9:365–373.
109. Stapleton JA. Trial comes too late as psychiatric side effects end hope for rimonabant. *Addiction.* 2009;104:277–278.
110. Carroll KM. *A cognitive-behavioral approach: Treating cocaine addiction.* Vol 1. Rockville, MD: National Institute on Drug Abuse; 1998.
111. Kadden R, Carroll KM, Donovan D, et al. *Cognitive-behavioral coping skills therapy manual.* Rockville, MD: National Institutes of Health; 1994.
112. Monti PM, Kadden RM, Rohsenow DJ, Cooney NL, Abrams DB. *Treating alcohol dependence: A coping skills training guide.* 2nd ed. New York: The Guilford Press; 2002.
113. Marlatt GA. Part I. Relapse Prevention: General Overview. In: Marlatt GA, Gordon JR, eds. *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors.* New York: The Guilford Press; 1985:1–348.
114. Copeland J, Swift W, Roffman R, Stephens R. A randomized controlled trial of brief cognitive-behavioral interventions for cannabis use disorder. *J Subst Abuse Treat.* 2001;21:55–64; discussion 65–66.
115. Carroll KM, Rounsaville BJ, Keller DS. Relapse prevention strategies for the treatment of cocaine abuse. *Am J Drug Alcohol Abuse.* 1991;17:249–265.
116. Carroll KM, Rounsaville BJ, Nich C, Gordon LT, Wirtz PW, Gawin F. One-year follow-up of psychotherapy and pharmacotherapy for cocaine dependence. Delayed emergence of psychotherapy effects. *Arch Gen Psychiatry.* 1994;51:989–997.
117. Carroll KM, Rounsaville BJ, Gordon LT, et al. Psychotherapy and pharmacotherapy for ambulatory cocaine abusers. *Arch Gen Psychiatry.* 1994;51:177–187.
118. Chaney EF, O’Leary MR, Marlatt GA. Skill training with alcoholics. *J Consult Clin Psychol.* 1978;46:1092–1104.
119. Larimer ME, Palmer RS, Marlatt GA. Relapse prevention. An overview of Marlatt’s cognitive-behavioral model. *Alcohol Res Health.* 1999;23:151–160.
120. Maude-Griffin PM, Hohenstein JM, Humfleet GL, Reilly PM, Tusel DJ, Hall SM. Superior efficacy of cognitive-behavioral therapy for urban crack cocaine abusers: main and matching effects. *J Consult Clin Psychol.* 1998;66:832–837.
121. Fabricatore AN. Behavior therapy and cognitive-behavioral therapy of obesity: is there a difference? *J Am Diet Assoc.* 2007;107:92–99.
122. Brownell KD, Heckerman CL, Westlake RJ. The behavioral control of obesity: a descriptive analysis of a large-scale program. *J Clin Psychol.* 1979;35:864–869.
123. Brownell KD, Cohen LR. Adherence to dietary regimens. 2: Components of effective interventions. *Behav Med.* 1995;20:155–164.
124. Brownell KD, Cohen LR. Adherence to dietary regimens. 1: An overview of research. *Behav Med.* 1995;20:149–154.
125. Brownell KD. Diet, exercise and behavioural intervention: the nonpharmacological approach. *Eur J Clin Invest.* Sep 1998;28 Suppl 2:19–21; discussion 22.
126. Ashley JM, St Jeor ST, Schrage JP, et al. Weight control in the physician’s office. *Arch Intern Med.* 2001;161:1599–1604.
127. Brownell KD, Stunkard AJ, McKeon PE. Weight reduction at the work site: a promise partially fulfilled. *Am J Psychiatry.* 1985;142:47–52.
128. Gardner CD, Kiazand A, Alhassan S, et al. Comparison of the Atkins, Zone, Ornish, and LEARN diets for change in weight and related risk factors among overweight premenopausal women: the A TO Z Weight Loss Study: a randomized trial. *JAMA.* 2007;297:969–977.
129. Marchesini G, Natale S, Chierici S, et al. Effects of cognitive-behavioural therapy on health-related quality of life in obese subjects with and without binge eating disorder. *Int J Obes Relat Metab Disord.* 2002;26:1261–1267.
130. Alcoholics Anonymous Big Book. 4th ed. New York: Alcoholics Anonymous World Services, Inc.; 2002.
131. Weiner S. The addiction of overeating: self-help groups as treatment models. *J Clin Psychol.* 1998;54:163–167.

132. Higgins ST, Budney AJ, Bickel WK, Foerg FE, Donham R, Badger GJ. Incentives improve outcome in outpatient behavioral treatment of cocaine dependence. *Arch Gen Psychiatry*. 1994;51:568–576.
133. Higgins ST, Wong CJ, Badger GJ, Ogden DE, Dantona RL. Contingent reinforcement increases cocaine abstinence during outpatient treatment and 1 year of follow-up. *J Consult Clin Psychol*. 2000;68:64–72.
134. Lussier JP, Heil SH, Mongeon JA, Badger GJ, Higgins ST. A meta-analysis of voucher-based reinforcement therapy for substance use disorders. *Addiction*. 2006;101:192–203.
135. Petry NM, Simcic F, Jr. Recent advances in the dissemination of contingency management techniques: clinical and research perspectives. *J Subst Abuse Treat*. 2002;23:81–86.
136. Peirce JM, Petry NM, Stitzer ML, et al. Effects of lower-cost incentives on stimulant abstinence in methadone maintenance treatment: a National Drug Abuse Treatment Clinical Trials Network study. *Arch Gen Psychiatry*. 2006;63:201–208.
137. Petry NM, Martin B, Cooney JL, Kranzler HR. Give them prizes, and they will come: contingency management for treatment of alcohol dependence. *J Consult Clin Psychol*. 2000;68:250–257.
138. Petry NM, Martin B, Finocche C. Contingency management in group treatment: a demonstration project in an HIV drop-in center. *J Subst Abuse Treat*. 2001;21:89–96.
139. Petry NM, Martin B. Low-cost contingency management for treating cocaine- and opioid-abusing methadone patients. *J Consult Clin Psychol*. 2002;70:398–405.
140. Petry NM, Alessi SM, Marx J, Austin M, Tardif M. Vouchers versus prizes: contingency management treatment of substance abusers in community settings. *J Consult Clin Psychol*. 2005;73:1005–1014.
141. Petry NM, Peirce JM, Stitzer ML, et al. Effect of prize-based incentives on outcomes in stimulant abusers in outpatient psychosocial treatment programs: a national drug abuse treatment clinical trials network study. *Arch Gen Psychiatry*. 2005;62:1148–1156.
142. Petry NM, Alessi SM, Hanson T. Contingency management improves abstinence and quality of life in cocaine abusers. *J Consult Clin Psychol*. 2007;75:307–315.
143. Petry NM, Alessi SM, Hanson T, Sierra S. Randomized trial of contingent prizes versus vouchers in cocaine-using methadone patients. *J Consult Clin Psychol*. 2007;75:983–991.
144. Epstein LH, Masek BJ, Marshall WR. A nutritionally based school program for control of eating in obese children. *Behavior Therapy*. 1978;9:766–778.
145. Jason LA, Brackshaw E. Access to TV contingent on physical activity: effects on reducing TV-viewing and body-weight. *J Behav Ther Exp Psychiatry*. 1999;30:145–151.
146. Hyman SE. The neurobiology of addiction: implications for voluntary control of behavior. *Am J Bioeth*. 2007;7:8–11.
147. Oliver JE. *Fat Politics: The real story behind America's obesity epidemic*. New York: Oxford University Press; 2005.