CME

Overeating, Binge Eating, and Eating Disorders as Addictions

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Each year eating disorders affect approximately five million Americans.[1] In 2000, nearly 39 million adults in the United States met the classification of obesity, defined as having a Body Mass Index score of 30 or more.[2] Recent evidence suggests pathological obesity and drug addiction share common brain characteristics.[3] The process of addiction is mediated through brain mechanisms underlying reward or reinforcement. Reinforcement can be accomplished through both positive and negative mechanisms. The ultimate expres-

sion of reinforcement is in the brain's hierarchical organization and promotion of behaviors through neurobiological mechanisms that reward and thereby make survival and other desirable behaviors more likely to occur. The brain does not seem to differentiate whether the reward is provoked by natural rewards, licit or illicit drugs, gambling, or extreme environmental manipulations, or fasting. Feeding, sex, and other survival behaviors are reinforced or made more likely by events occurring within the ventral tegmental area (VTA) and nucleus accumbens (nAC).

Being stuffed with cake against one's will is a markedly different experience than sneaking around and raiding the refrigerator after midnight.

An increase in the extracellular level of dopamine in the nAC, a major target of the mesolimbic dopaminergic system,[4] during ingestion of food has been reported by numerous authors.[5-10] This is quite similar to the result produced by cocaine or after other drug self-administration.[11] The increase in the extracellular level of dopamine in the nAC suggests that there is a relationship between food intake and the activity of the mesolimbic dopaminergic system (MDS), just as it has been suggested that there is a relationship between reward, illicit drug reinforcement, self-stimulation and sexual behavior, and MDS dopaminergic activity. Recent positron emission tomographic imaging studies are further linking eating and addiction, showing that obese individuals and individuals who are substance dependent have significantly lower dopamine D2 receptor levels.[12] New functional magnetic resonance imaging techniques such as temporal clustering analysis may provide further evidence by identifying the onset and duration of activation, while functional mapping techniques further localize the brain regions activated in response to food.[13]

Food is reported to be comforting in times of stress, addicting or extremely rewarding by some patients who complain of compulsive candy, chips, or other food consumption. In animals it appears that food reward depends in part on its palatability or the hedonic component related to the sensory properties of foods.[14,15] Like recent immigrants to the United States, when animals are offered a more palatable diet, most animal species eat more and become obese.[16-18] Passive drug administration is usually not addicting, while active goal-directed behavior seeking and using drugs for euphoria production is addicting. Being stuffed with cake against one's will is a markedly different experience than sneaking around and raiding the refrigerator after midnight.

The numerous associations between eating disorders and drug addictions — craving, preoccupation, compulsive use/behavior despite adverse consequences, denial of problem, use of substance to relieve negative affect with guilt following the use, comorbidity, genetic links, high recidivism especially when exposed to cues or triggers, overeating and weight gain during early recovery from drug addiction, and common neurobiological pathways, modification of drug reward by eating or starvation — lead us to consider the possibility that binge-eating disorders represent a drug-free auto-addiction for a significant subset of the addiction prone. In some it is a trigger leading to a drug or alcohol relapse (eg, the cigarette smoker who gains weight after cessation). In others it is an apparent result of alcohol or drug abstinence when food becomes the new drug of choice (Figure).

DRUG REWARD

All drugs of abuse are self-administered by animals and man. Cocaine is self-administered to death in paradigms where use/access are unlimited.[19] However, the most convincing evidence that drugs access this primitive system is that they decrease the amount of brain stimulation required to motivate baseline responding.[20] Drug use motivates repetition of the behavior required for access and/or administration and creates a feeling of satisfaction like that produced by completion of a biological imperative or normal survival behavior. In many instances cocaine addicts stop eating, drinking, washing, and talking and interacting with others and look like patients with a primary eating disorder.[21]

Primary Reinforcers

Feeding behavior is typically a response to hunger. Hunger as a reason

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The authors have no industry relationships to disclose.

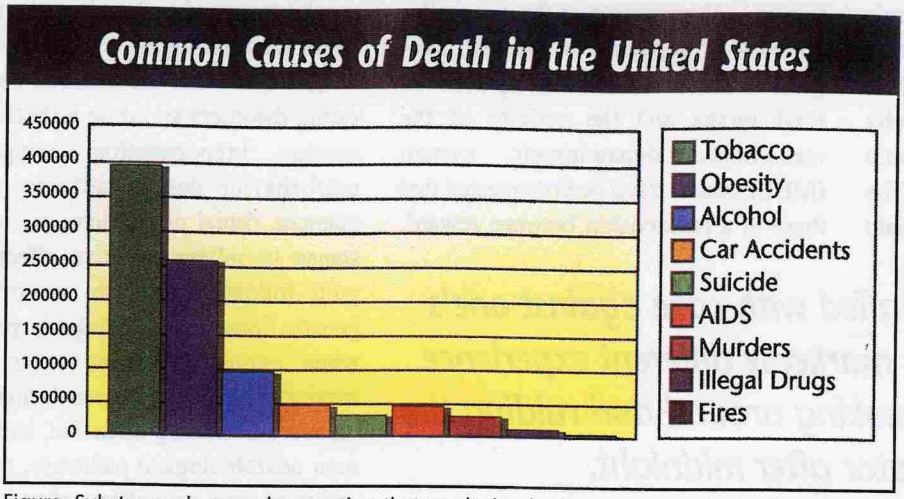


Figure. Substance abuse and overeating that results in obesity are among the leading causes of death and disability in the United States .

to eat may have been more important in our past than it is today in the United States. Hunger is likely generated by depletion of nutrient and energy stores. The reward generated by feeding, paired with the state of hunger and its resolution into satiety, thus positively reinforces feeding behavior in a state of hunger. Studies show that a state of hunger via food deprivation in rats actually enhances the reward effect of feeding. State of hunger also potentiates reward from drugs of abuse. In this sense hunger and craving could be seen as secondary reinforcers paired with the primary reward generated by feeding. Eating or the use of cocaine produces satiation.

Neuropeptides

Some of the major neuropeptides involved in regulating appetite and feeding are listed in Table 1.

Neuropeptide Y

An understanding of neuropeptide Y (NPY) provides an excellent foundation to explain uncontrolled hyperphagia. Neuropeptide Y is one of the most abundant and widely distributed neuropeptides known. Studies indicate that serotonin may play an antagonistic role with neuropeptide Y in the regulation of feeding.[22] Serotonin neurons innervate NPY-containing neurons in the arcuate nucleus. Central injections of serotonin inhibit food intake through NPY as we might expect selective serotonin reuptake inhibitors to act. Neuropeptide Y infusions in the hypothalamus decrease serotonin release. Neuropeptide Y is the most potent activator of feeding behavior yet discovered; its levels seem to vary inversely with those of serotonin. Administration of NPY into the hypothalamic paraventricular nucleus induces feeding in satiated animals and may selectively induce extraordinary carbohydrate intake. Administration of anti-NPY decreases spontaneous carbohydrate intake in animals. Chronic administration of NPY produces an obesity syndrome indistinguishable from naturally occurring obesity. Neuropeptide Y is clearly the single most potent orexigenic compound known and a viable model for driven over-eating and obesity.

BEHAVIORAL SIMILARITIES

Addictions and eating disorders share many common clinical features. Drugs of abuse and food go hand-in-hand—a cup of coffee after a good meal and the use of marijuana or alcohol before sex and a cigarette afterwards. Patients in early alcoholism recovery begin eating ice cream, potato chips, and bingeing at night. They note that this behavior calms them down. Alcoholics Anonymous has long suggested that its members avoid HALT (hungry, angry, lonely, tired). It seems likely that one of the stimulatory peptides is involved in the release of driven eating. Still, eating is much more complex, involving numerous stimulatory and inhibitory peptides and messengers.

Addiction is defined as a disease characterized by the repetitive and

TABLE 1

Neuropeptides That Regulate Food Intake[22]

*Stimulate Feeding**	*Inhibit Feeding†*
Neuropeptide Y	Serotonin
Galanin	Insulin
Dynorphin	Neurotensin
β-endorphin	CRF
GHRH	Dopamine
Norepinephrine	Cholecystokinin
Anandamide	Leptin
GABA	TRH, MSH,
Ghrelin	Glucagon, Enterostatin, Calcitonin, Amylin, Bombesin, Somatostatin, Cytokines

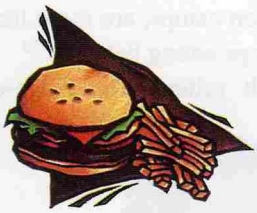
**Usually decrease energy expenditure*
†Usually increase energy expenditure
CRF = corticotropin-releasing factor; GHRH = growth hormone-releasing hormone; GABA = Ɣ-aminobutyric acid; TRH = thyrotropin-releasing hormone; MSH = melanocyte-stimulating hormone

TABLE 2

Comparison of DSM-IV Binge Eating and Substance Dependence Criteria

DSM-IV Binge Eating Disorder

A. Recurrent episodes of binge eating characterized by both:
   1. Eating in a discrete period of time (eg, 2 hours) an amount of food that is much larger than most people would eat in a similar time period under similar circumstances.
   2. A sense of lack of control over eating during the episode (a feeling that one cannot stop eating or control what or how much one is eating).



B. Episodes include 3 or more of the following:
   1. Eating much more rapidly than normal.
   2. Eating until feeling uncomfortably full.
   3. Eating large amounts of food when not feeling physically hungry.
   4. Eating alone because of being embarrassed by how much one is eating.
   5. Feeling disgusted with oneself, depressed or very guilty after overeating.

C. Marked distress regarding binge eating is present.

D. The binge eating occurs on average at least 2 days a week for 6 months.

E. The binge eating is not associated with the regular use of inappropriate compensatory behavior.

DSM-IV Substance Dependence

A. A maladaptive pattern of substance use leading to clinically significant impairment or distress, manifested by three or more of the following, occurring within a 12-month period:
   1. *Tolerance.*
      a. Need for *increased amount* to achieve the desired effect.
      b. Diminished effect with continued use of the same amount.
   2. Withdrawal.
      a. Characteristic withdrawal symptom for the substance.
      b. The same or similar substance is taken to avoid withdrawal symptoms.
   3. Substance is taken *in larger amounts* or over a longer period than intended.
   4. Persistent *desire or unsuccessful attempts to cut down or control use.*
   5. *Great deal of time is spent obtaining, using or recovering* from the substance.
   6. *Important activities are reduced or given up* because of the substance.
   7. *Continued use despite adverse consequences.*

destructive use of one or more mood-altering drugs that stems from a biological vulnerability exposed or induced by environmental factors.[23] The *DSM-IV* defines substance dependence as a maladaptive pattern of substance use leading to clinically significant impairment or distress with three or more criteria occurring in the same 12 months. If the *DSM-IV* did not define addiction in terms of a psychoactive substance, would eating disorders qualify? With the change away from considering tolerance and withdrawal as essential pieces of substance dependence disorders, eating disorders share many of the most salient features of addictions.

Eating Disorders

Restrictions on food intake in anorexia, exercising, or purging produces alterations of brain feeding mechanisms that are gradually recognized as a normal state, much like drug addicts of all types consume their drugs of choice in order to feel normal. But, as Halmi asks, "What is unique about the individual who goes on to develop anorexia nervosa?."[24] It is the same question that has been contemplated by addiction specialists for decades: why are millions exposed to drugs such as alcohol and tobacco, but only a smaller percentage become addicts? Exposure is the common risk factor, but a common neurobiological process must be operant in all those who use a substance (food in case of anorexics) in a manner that is dangerous to their survival with little insight or regard for negative consequences.

Bulimia Nervosa

Like adolescent alcohol and drug use, many young men and women experiment with severe diets, starvation, fasting, and self-induced vomiting, yet few apparently loose control and become anorexic or bulimic. Despite the significant negative consequences, these behaviors are engaged in by approximately 0.5% of the women between the ages of 15 and 40 years in the case of anorexia nervosa, and 1% to 1.5% of women in the case of bulimia.[25-26]

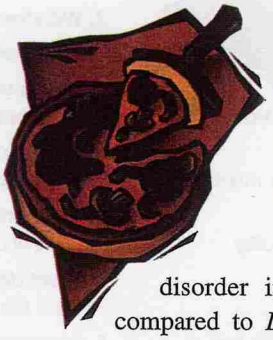
As with classical addictions, eating disorders interfere with normal life patterns. Preoccupation with the substance in question, such as supermarket gazing, watching food shows on television, and cooking for others is common. Just as with other addictive substances, the overconsumption of food or bingeing is frequently conducted at night or in secret.[27] The chronic compromised nutritional state induced by both of these disorders produces cognitive changes, mood labili-

ty, apathy, irritability, decreased libido, and sleep disturbances commonly seen in other addictions.[28] Eating disorders, like classical addictions, can have quite tragic outcomes, with a high degree of mortality. It is quite common to hear of a bulimic trigger in the same way an addict describes a drug cue that precipitates intense cravings and use. Patients with bulimia describe external and internal cues such as seeing a pizza delivery truck or feeling depressed as triggering thoughts that they are fat or in need or a binge-purge episode. They describe rituals similar to drug using rituals with similar foods and environments and report feeling the high beginning with the ritual. The reported incidence of drug abuse among patients with bulimia is as high as 46%.[29] Bulimia has long been known to have a high comorbidity with substance abuse, particularly alcoholism.[30-31]

Binge-Eating Disorder

Obesity has many possible causes and may be divided into groups including hypothyroidism, diabetes mellitus, or binge-eating disorders. There is some evidence that genetic defects in cocaine- and amphetamine-related transcript (CART), an endogenous satiety factor, may contribute to obesity.[32] Binge-eating disorder is perhaps the most common, yet least studied, of the eating disorders.[33] Binge-eating disorders, like drug addiction, are characterized by pathological attachment. Both include obsessive thoughts and compulsions, with binge eaters thinking about food and compulsively eating an amount of food larger than most people would eat in similar amounts of time under similar circumstances. Like cocaine addiction or alcoholism, the binge eater cannot take it or leave it; they "can't eat just one." They take it and take it and take it. The binge episodes generally cause the binge eater much distress, leaving him or her with feelings of guilt, disgust, and depression. Some studies show that the amount of food consumed is directly proportional to the amount of dopamine release in the lateral hypothalamic area.[34] Researchers have shown that persons who have survived extreme food deprivation, such as survivors of Nazi concentration camps, are more likely to develop binge-eating behavior.[35]

Research criteria for binge-eating disorder in the *DSM-IV* compared to *DSM-IV* criteria for substance dependence are presented in the Table 2 (page 120). Italicized criteria under substance dependence are commonly found among binge eaters.



Like cocaine addiction or alcoholism, the binge eater cannot take it or leave it; they "can't eat just one."

In essence, binge-eating disorder is bulimia nervosa without compensatory behavior and it has many characteristics similar to substance dependence.

CAGE QUESTIONNAIRE

The CAGE questionnaire is often used in screening for alcohol dependence. Here the questions have been modified to address binge eating

- Have you ever felt the need to cut down on eating?
- Have you ever felt annoyed by criticism of your eating?
- Have you ever had guilty feelings about your eating?
- Have you ever gotten up in the middle of the night or early in the morning to eat?

Multiple positive responses may indicate a problem.

CONCLUSION

On the basis of the well-known adage: if it looks like a duck, acts like a duck and quacks like a duck, it must be a duck, patients with binge eating, obesity, anorexia, and bulimia have a chronic disorder characterized by loss of control, relapse, compulsivity, reprioritization, and continuation despite severe and adverse consequences. The *DSM-IV* eating disorder category naturally encompasses a diverse patient group. Some restrict, some purge, and some binge. Body mass index may be high, low, or even normal. Arguments have been made to consider eating disorders as personality disorders, obsessive-compulsive disease, depression, and as an addictive disorder. Addiction treatment programs have alerted us to the high comorbidity of eating disorders in addiction patients. There is a high degree of eating disorders that appear to run in families. It is common for eating binges or starvation to be relapse triggers in newly abstinent patients. Patients also report enhancing their drug's euphorogenic properties through starvation or purging. These observations support the importance of continued questioning and consideration to the hypothesis that eating disorders are related to tobacco, alcohol, and other addictive disorders.

Should eating disorders be classified as addictions? Both disorders involve similar brain systems and result in similar behaviors and feeling states. Both are important diseases where loss of control and compulsive use are preeminent. Both are diverse patient groups of people with illnesses of unknown etiology, characterized by a chronic relapsing

course without specific pathophysiology or treatment. Both involve the acquired pathological attachment with the agent(s) of their ultimate compromise and possible destruction. Both may involve host or risk factors that predispose a person to extreme reward after consumption or use, thereby making repetition more likely to occur. In both there is considerable experimental evidence of biologic vulnerability. Both involve denial and reluctance to accept that the patient is in fact ill and in need of treatment. Both can result in early death. Both generally require early experimentation, one with drugs, the other with dieting and/or binging. Both can be relapse triggers for each other. Drugs are used to decrease eating and eating is used to decrease drug-taking. Both are used to accentuate the other. The similarities are numerous and striking. Food is a powerful mood altering substance that is repetitively and destructively used (or restricted) in eating disorders just as drugs are in substance use disorders.

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