Are Eating Disorders Addictions?

13

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Abstract

Eating disorders overlap with substance use disorders and addictions in many important ways, including clinical phenomenology, comorbidity, pathophysiology, neurobiology, and treatment approaches. Evidence is reviewed for and against the contention that the eating disordered behaviors of dieting, binge eating, purging, and exercising are potentially addictive behaviors. In addition, abuse of and dependence upon substances meant to inhibit appetite (stimulants), reduce caloric absorption (laxatives, lipase inhibitors), decrease water weight (diuretics), or induce vomiting (ipecac) are characteristic features of eating disorders with bulimic features. The phenomenology of the eating disorders is viewed in light of the DSM-5 criteria for an addictive disorder and the new description of an addiction recently published by the American Society of Addiction Medicine. Both sets of criteria support the conclusion that eating disordered behaviors can be phenotypically conceptualized as addictive. On the other hand, eating disorders exhibit clinical characteristics not seen in classical substance use disorders or addictions, including distortions in body size and shape as well as intense fear of gaining weight. The therapeutic implications of treating eating disorders as addictive disorders are discussed. Taken together, eating disorders may be conceptualized as addictive disorders in at least a subset of individuals, but further research is required to determine if tolerance and withdrawal occur in humans.

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13.1 Introduction

Research evidence supports the contention that eating disorders (ED), substance use disorders (SUD), and addictions are complex disorders that are caused by multiple factors. There is simply no one cause; rather, ED and SUD result from an interaction of an array of genetic and environmental factors. These factors can be understood in light of a biopsychosocial, spiritual, and developmental continuum in which both nature and nurture interact over time. In the last century, most research studies focused on either some aspect of nurture or some aspect of nature and rarely were they truly integrated. It has not been until the dawn of this millennium that investigators have been studying them together and showing quite readily how much they interact and influence each other. A good way of thinking about it is that "genetics loads the gun, and environment pulls the trigger."

Latent vulnerability theory helps to explain why only four or five out of 100 girls who go on a serious diet will develop anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), or an other specified ED. An important point to emphasize for the purposes of this discussion is that all of the problematic behaviors associated with ED—dieting, binge eating, purging, and exercising—are known to alter brain neurochemistry, sometimes in very profound ways. This is particularly true for the prolonged starvation that results from chronic dieting and weight loss. Over time, these four basic behaviors proceed to expose the genetically mediated latent or hidden vulnerability in any given individual that in turn leads to the overt manifestations of an ED. Importantly, all of these four ED behaviors have been found to have addictive features, which are the focus of this chapter. Does the science support this perspective?

This chapter will first outline the new criteria for substance-related disorders and addictions set forth by the publication of the DSM-5 (American Psychiatric Association, 2013) and the Public Policy Statement on addictions published by the American Society of Addiction Medicine (ASAM, 2011) and discuss their relationship to ED (particularly bulimic-spectrum disorders). This will be followed by a summary of the similarities and differences between addictions and ED. Both the pros and cons of conceptualizing binge eating as a "food addiction" will be explored. Finally, the chapter will conclude with a discussion of treatment implications and directions for further research.

Table 13.1 DSM-5 criteria for unspecified substance use disorder as applied to food. There is a problematic pattern of use leading to clinically significant impairment or distress that is manifested by two of the following (American Psychiatric Association, 2013)

YES	1. The substance is often taken in larger amounts or over a longer period than was intended
YES	2. There is a persistent desire or unsuccessful efforts to cut down or control use of the substance
YES	3. A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects
YES	4. Craving, or a strong desire or urge to use the substance
YES	5. Recurrent use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use
YES	6. Continued use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use
YES	7. Important social, occupational, or recreational activities are given up or reduced because of use of the substance
YES	8. Recurrent use of the substance in situations in which it is physically hazardous
PROBABLY	9. Tolerance, as defined by either of the following: (a) A need for markedly increased amounts of the substance to achieve intoxication or desired effect (b) A markedly diminished effect with continued use of the same amount of the substance
NO[a]	10. Withdrawal, as manifested by either of the following: (a) The characteristic withdrawal syndrome for other substance (b) The substance (or a closely related substance) is taken to relieve or avoid withdrawal symptoms

[a]YES in animal studies for sugar

13.2 DSM-5 Criteria for Substance-Related Disorders and Addictions

When the DSM-5 (American Psychiatric Associations, 2013) criteria for substance dependence are examined in light of ED symptoms, such as binge eating, many behavioral similarities are apparent (see Table 13.1). For many years, substance dependence was defined by the presence of tolerance and withdrawal, but now they are not necessary for a diagnosis of SUD to be made. When it comes to the behaviors of dieting, binge eating, purging, and exercising, it is not yet clear whether there are true tolerance and withdrawal to these behaviors in humans.

The DSM-5 criteria for an "other (or unknown) substance-related disorder" will be reviewed here as it applies to food, which is the primary substance abused by patients with bulimic-spectrum disorders (see Table 13.1). The "A" criteria indicate "*a problematic pattern of use of an intoxicating substance not able to be classified with the alcohol*; *caffeine*; *cannabis*; *hallucinogen* (*phencyclidine and others*); *inhalant*; *opioid*; *sedative*, *hypnotic*, *or anxiolytic*; *stimulant*; *or tobacco categories*. . .)." The online Merriam-Webster dictionary definition of the word

"intoxicating" is as follows: "a) producing in a person a state ranging from euphoria to stupor, usually accompanied by loss of inhibitions and control; inebriating; b) stimulating, exciting, or producing great elation." It can be argued that certain foods are indeed "intoxicating" to some people, particularly those with bulimic-spectrum disorders. It has been shown that such individuals experience food in a different way than individuals without bulimic tendencies. In particular, highly palatable foods induce a high degree of "hedonic reward," and the saliency for food is enhanced. In addition, recent ecological momentary assessment (EMA) findings indicate that binge eating and purging induce positive affect and are therefore experienced as particularly pleasurable (see Chap. 16).

The DSM-5 criteria continue: "*and leading to clinically significant impairment or distress, as manifested by at least two of the following, occurring within a 12-month period.*"

1. "*The substance is often taken in larger amounts or over a longer period than was intended.*" This clearly occurs with binge eating and subsequent purging, which tends to be chronic once commenced. Fichter, Quadflieg, and Hedlund (2008) reported that 36 % of BED and 28 % of BN patients still received an ED diagnosis at 12-year follow-up.
2. "*There is a persistent desire or unsuccessful efforts to cut down or control use of the substance.*" By definition, a binge entails not only eating a large amount of food but also the subjective sense of loss of control over eating. Furthermore, binge eating is typically experienced as ego-dystonic, a behavior that, although pleasurable, is undesired and often shame based. Even with the best treatments for BN or BED, many patients do not obtain nor do they maintain full abstinence.
3. "*A great deal of time is spent in activities necessary to obtain the substance, use the substance, or recover from its effects.*" This is also present in bulimic-spectrum disorders, particularly when there are both binge eating and purging. Binge and/or purge episodes take up inordinate amounts of time and also take a toll mentally and physically, often leading to marked fatigue as well as other related medical effects (see Chap. 15). Individuals who binge and purge devote a great deal of time and money to acquiring food and getting rid of it, often in secret and with great distress, guilt, and shame.
4. "*Craving, or a strong desire or urge to use the substance.*" Craving has been described as a feature in bulimic ED (Van den Eynde et al., 2012), which is only aggravated by dietary restriction (Moreno-Dominguez, Rodriguez-Ruiz, Fernandez-Santaella, Ortega-Rolden, & Cepeda-Benito, 2012).
5. "*Recurrent use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use.*" By definition, bulimic disorders are recurrent and persistent, and as a result, a host of adverse consequences may ensue, including social, interpersonal, academic, economic, and/or medical. Negative reactions of family and friends to bingeing and other ED behaviors are common. Those who are most chronic are also the ones who are the most self-destructive (Fichter et al., 2008).

6. "*Continued use of the substance despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of its use*." Impairment in the social life of individuals with BN, BED, subthreshold BED, or any recurrent binge eating was commonly reported in the National Comorbidity Survey Replication (Hudson, Hiripi, Pope, & Kessler, 2007).
7. "*Important social, occupational, or recreational activities are given up or reduced because of use of the substance*." In addition to impairment in social life, there is also impairment in home, work, and the personal life of individuals with bulimic-spectrum disorders (Hudson et al., 2007). Withdrawal from others and overt social phobia is commonly associated with bulimic disorders and can be exacerbated by the shame and secretiveness associated with this behavior (Brewerton, Lydiard, Ballenger, & Herzog, 1993; Hudson et al., 2007).
8. "*Recurrent use of the substance in situations in which it is physically hazardous*." Bulimic disorders and binge eating occur while driving a motor vehicle, and this is a recognized cause of accidents (Petridou & Moustaki, 2000). The author has personally known of many patients who engage in bulimic behaviors in their automobiles while driving, including one patient who was killed in a head-on collision while actively bingeing and purging.
9. "*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*." Such persistence in the face of negative outcomes, both medical and psychiatric, is also a common characteristic of ED (see Chap. 11). Examples include fatigue, fluid and electrolyte abnormalities leading to muscle weakness and spasms, cardiac arrhythmias, pharyngeal/esophageal irritation and bleeding, gastroesophageal reflux disease (GERD), anemia, dental erosion, as well as the effects of weight gain and resultant obesity, e.g., hypertension, diabetes, sleep apnea, etc. (see Chap. 15).
10. "*Tolerance, as defined by either of the following*:"
    (a) "*A need for markedly increased amounts of the substance to achieve intoxication or desired effect*."
    (b) "*A markedly diminished effect with continued use of the same amount of the substance*." Food tolerance has been demonstrated in animal studies (see Chap. 1). Over time rats with intermittent sugar and chow access have decreases in dopamine type 2 (D2) receptor messenger ribonucleic acid (RNA) levels in the nucleus accumbens compared with control rats on chow alone (Avena, Rada, & Hoebel, 2008). Evidence for tolerance in humans is indirect and implied and comes largely from clinical reports of bulimic patients eating larger and larger binge meals, eating more frequent binge meals, and/or gaining weight as the disorder progresses. The impaired satiety characteristic of full-blown bulimic-spectrum disorders can be seen as an indirect indicator of tolerance, as is the fact that individuals with BN and BED are typically overweight or obese (Brewerton, 1995; Dansky et al., 1998; Hudson, Hiripi, Pope, & Kessler, 2007). MRI studies indicate hypofunctioning of brain reward circuitry in response to a chocolate milkshake in patients with BN compared to

controls, which the authors speculated may be due to repeated bingeing on hyperpalatable foods (Bohon & Stice, 2011). In addition, the positive relationship between binge size, frequency, and BMI in individuals with BED also supports the notion of tolerance to food (Guss, Kissileff, Devlin, Zimmerli, & Walsh, 2002; Picot & Lilenfeld, 2003). As BMI increases, insulin resistance also increases, which can increase hunger and reduce satiety (Madden, Leong, Gray, & Horwath, 2012). In a prospective study over 8 years, Thomas, Butryn, Stice, and Lowe (2011) reported that substantial weight gain (or weight loss) resulted in a sevenfold increase in risk for subsequent onset of BN or subthreshold BN and that these individuals who developed a bulimic ED experienced greater increases in weight during the 2 years prior to ED onset when compared to healthy controls. In a prospective study of children over the course of 5 years, Tanofsky-Kraff and colleagues (2006, 2012) reported that binge eating predicted subsequent weight gain, increase in adipose tissue, and the development of signs of metabolic syndrome. In a prospective study of adolescents, Stice, Marti, Shaw, and Jaconis (2009) noted that subthreshold cases of BN and BED often progressed to threshold cases, thereby documenting a progressive increase in meal size and binge frequency.

11. "*Withdrawal*, *as manifested by either of the following*:"
    (a) "*The characteristic withdrawal syndrome for other* (*or unknown*) *substance* . . ."
    (b) "*The substance* (*or a closely related substance*) *is taken to relieve or avoid withdrawal symptoms*." A withdrawal syndrome from sugar has been described in animals (Avena, Bocarsly, Rada, Kim, & Hoebel, 2008; Avena, Rada, & Hoebel, 2008, 2009; Colantuoni et al., 2002; Ifland et al., 2009) (see Chap. 1) but remains to be clearly demonstrated in humans (Benton, 2010). However, there are clinical, anecdotal reports of "sugar withdrawal" in humans, which is characterized by irritability, headaches, and flu-like symptoms (Davis & Carter, 2009).

Taken together, the DSM-5 criteria for a substance-related disorder or addiction can be applied to ED behaviors, particularly binge eating, which commonly involves highly palatable, highly rewarding food. In a similar exercise, Cassin and von Ranson (2007) applied DSM-IV criteria for substance dependence to a group of women with BED and found that 92.4 % of them met criteria for food dependence. Likewise, Goodman (1990) applied DSM-III-R criteria for substance dependence to individuals with compulsive overeating and found significant phenotypic overlap. However, it is important to note that this phenotypic overlap between SUD and ED does not necessarily imply the same illness or mechanism.

13.3 The American Society of Addiction Medicine Definition of Addiction

Psychiatry and addiction medicine have had an interesting relationship over the years and have not always been in agreement (O'Brien, Volkow, & Li, 2006). The word "addiction" or "addictive" was thought to have a negative, stigmatizing connotation and therefore did not appear in any version of the Diagnostic and Statistical Manual of Mental Disorders until DSM-5 (American Psychiatric Association, 2013), where the classification of "substance-related disorders and addictive disorders" was listed for the first time.

The American Society of Addiction Medicine (ASAM, 2011) recently published an expanded definition of addiction. In summary, it reads as follows: "*Addiction is a primary, chronic disease of brain reward, motivation, memory and related circuitry. Dysfunction in these circuits leads to characteristic biological, psychological, social and spiritual manifestations. This is reflected in an individual pathologically pursuing reward and/or relief by substance use and other behaviors. Addiction is characterized by inability to consistently abstain, impairment in behavioral control, craving, diminished recognition of significant problems with one's behaviors and interpersonal relationships, and a dysfunctional emotional response. Like other chronic diseases, addiction often involves cycles of relapse and remission. Without treatment or engagement in recovery activities, addiction is progressive and can result in disability or premature death.*"

This definition is reminiscent of the DSM-5 definition as reviewed above. All of the clinical features and aspects described in the ASAM definition are seen in ED, although some of these may also be seen in other psychiatric disorders as well, such as impulse control disorders (see Chaps. 6 and 18). A common feature in both SUD and bulimic ED is the "inability to abstain" from harmful substances and/or behaviors. Like other chronic diseases, the ASAM definition notes that addiction often involves cycles of relapse and remission, which is true for many individuals with ED, as well as mood and anxiety disorders, which are highly comorbid to both SUD and ED. Without treatment or engagement in recovery activities, both ED and addictions can result in disability or premature death.

The ASAM expanded definition of addiction states that it is "*a primary chronic disease of brain reward, motivation, memory, and related circuitry, addiction substantially affects neurotransmission and interactions within reward structures of the brain, particularly the nucleus accumbens, anterior cingulate cortex, basal forebrain and amygdala, such that motivational hierarchies are altered and addictive behaviors, which may or may not include alcohol and other drug use, supplant healthy, self-care related behaviors.*" This has clearly been shown for ED, at least bulimic ED (see Chaps. 3 and 4). Addictions, SUD, and ED affect neurotransmissions and interactions between cortical and hippocampal circuits and brain reward structures, such that the memory of previous exposures to rewards (such as food) leads to a biological and behavioral response to external cues, thereby triggering craving and/or engagement in addictive and ED behaviors.

Problems in frontal cortex function in both SUD and ED contribute to altered judgment and the dysfunctional pursuit of rewards. Importantly, the frontal lobe morphology, connectivity, and functioning are still in the process of maturation during adolescence and young adulthood and do not fully mature until the mid- to late 20s (Lenroot & Giedd, 2008). Certainly, when children and adolescents are exposed to early substances or potentially addictive behaviors, there is a heightened risk in terms of developing an addiction and/or an ED, as well as their negative consequences. Most mental disorders, including SUD and ED, begin in childhood and adolescence (Kessler, Amminger, & Ustun, 2007).

The ASAM definition of addiction encompasses both the pursuit of pleasure and relief from negative affect. Typically, an addiction is a behavior that is performed to induce pleasure, at least initially, whereas a compulsion is something that is not, for the most part, pleasurable but instead results in relief from anxiety. Often these phenomena overlap, particularly once the behavior has become chronic. Frequently, individuals experience pleasure in getting relief from negative affect. Typically, after an addiction has progressed, the rewarding aspect or pleasure is diminished, and it becomes more of a sense of relief to mitigate the withdrawal symptoms. Similarly, binge eating may be initiated to induce pleasure but often serves as a method to relieve anxiety.

As noted in the expanded ASAM definition, both genetics and environment play a significant role in whether and how addictions develop. Genetic liability accounts for approximately 50 % of the likelihood that an individual will develop an addiction (ASAM, 2011) and 50–80 % of probability that someone will develop an ED (Bulik, Sullivan, Tozzi, Furberg, Lichtenstein, & Pedersen, 2006; Kendler, MacLean, Neale, Kessler, Heath, & Eaves, 1991; Klump, Miller, Keel, McGue, & Iacono, 2001). In other words, there is substantial evidence that SUD (like ED) aggregate in and are transmitted within families. Emerging research on genetic cross-transmission between disorders has been documented and is discussed in Chap. 5. We live in a toxic, fast-paced, highly stressful culture that is materialistic, image driven, food and pleasure oriented, and drug seeking. Exposure to trauma and other significant life stressors plays an important role in the development of both addictions and ED. There are clearly other factors that can contribute to the appearance of addiction and ED. Repeated engagement in drug use or other addictive behaviors causes a neuroadaptation at a neuronal level in motivational circuitry that leads to impaired control over further drug use or engagement in addictive behaviors. As a result, a reward deficit situation is created that leads to wanting more. Cognitive and affective distortions, which are often a target of treatment for both SUD and ED, impair perceptions and compromise the ability to deal with feelings, resulting in significant self-deception.

The ASAM definition notes the ABCs of addiction (see Table 13.2), which suggests that addiction is more than a behavioral disorder; it affects "*cognitions*, *emotions*, *interactions with others*, *ability to relate to family*, *community*, *themselves*, *and the Transcendent*." These criteria readily apply to bulimic-spectrum ED and to some extent AN-R (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004).

Table 13.2 American Society of Addiction Medicine (ASAM) ABCs of addiction as applied to substance use disorders (SUD), behavioral addictions (BA), bulimic eating disorders (bED), and anorexia nervosa, restricting type (AN-R)

	SUD/BA	Bulimic ED	AN-R
• Inability to consistently **A**bstain	+	+	–[a]
• Impairment in **B**ehavioral control	+	+	±
• **C**raving	+	+	–[a]
• **D**iminished recognition of problems	+	+	+
• A dysfunctional **E**motional response	+	+	+

[a]Applies to dieting, exercising, and/or pursuit of the thin ideal

In summary, both the DSM-5 criteria for "other substance-related disorders" and the ASAM criteria for addiction (with a few exceptions) appear to be consistent with current understanding of the ED, particularly bulimic-spectrum disorders.

13.4 Similarities and Differences Between Eating Disorders and Addictions

Many people have major misconceptions about the nature of addictions and ED. They are not conditions that individuals choose to have or develop. These are not desired conditions even though, to some extent, lay persons and many health professionals see them this way.

The notion of behavioral addictions or process addictions was described many years ago (Korolenko, 1991; Goodman, 1990), but it is only recently that this notion has begun to be accepted by mainstream psychiatry and psychology (Goodman, 2008; Smith, 2012) (see Chap. 18). Dieting, exercise, binge eating, and purging can be described as process addictions even though binge eating involves a substance, i.e., food, albeit a heterogeneous one chemically. Gambling, sex and love addiction, computer/Internet, and compulsive buying are other presumptive behavioral or process addictions that have been described in both SUD and ED populations, particularly those with bulimic symptoms (Korolenko, 1991; Goodman, 1990). One commonality between these conditions is that afflicted individuals tend to be elevated on measures of novelty seeking, sensation seeking, and impulsivity (see Chap. 6).

13.4.1 Dieting, Restricting, and Starvation Dependence

Severe dieting, food restriction, and/or fasting can progress to the point of inducing a starvation state. Several investigators described the concept of starvation dependence in AN (Luby, Marrazzi, & Sperti, 1987; Marrazzi & Luby, 1986; Marazzi et al., 1990; Szmukler & Tantam, 1984). Based on clinical observations and sharing of fundamental features between dieting and addictions, they argued for an

"auto-addiction" model of chronic AN in which severe dieting is potentially addicting and AN is viewed as a dependence disorder. This model proposes that endogenous opiates are released during the initial period of dieting or prolonged food deprivation that creates a psychological "high" and, in turn, initiates and reinforces a state of starvation dependence. This theory goes a long way toward explaining the intractable nature of the disorder as well as what is so reinforcing about dieting. Dr. Tom Insel (2013), the Director of the National Institute of Mental Health, recently stated in regard to AN, "I think about it as an addiction." There are a variety of studies that shed light on this theory.

For example, Kaye, Pickar, Naber, and Ebert (1982) reported increased endogenous opioid activity in the CSF of severely underweight patients with AN, although the same authors later reported decreased CSF levels of beta-endorphin as well as its three sister peptides, beta-lipotropin, adrenocorticotropic hormone (ACTH), and pro-opiomelanocortin (POMC), in underweight AN patients (Kaye et al., 1987). Brambilla and associates (1995) reported that patients with AN (both restricting type (AN-R) and binge-purge type (AN-BP)) had significantly higher lymphocyte concentrations of beta-endorphin than in controls. This was especially true for patients with AN-BP. Marrazzi, Luby, Kinzie, Munjal, and Spector (1997) reported elevated levels of endogenous plasma alkaloids in patients with AN and BN in comparison to controls, although there have been no new studies in this century of this idea. Taken together, these data indicate that the opioid system is dysregulated in AN during the low weight state and they are compatible with the auto-addiction model of AN. Results using the opiate antagonist naltrexone in the treatment of ED have been mixed, with the best studies showing no effect compared to placebo (Marrazzi, Bacon, Kinzie, & Luby, 1995; Marrazzi, Markham, Kinzie, & Luby, 1995; Mitchell et al., 1989). Therefore, the auto-addiction model of AN has not been supported by pharmacologic studies to date.

Results from a recent fMRI study were interpreted to be in support of the starvation dependence model (Fladung et al., 2010). Patients with AN and healthy controls underwent a functional MRI during evaluation of visual stimuli. Subjects were shown different images consisting of underweight, normal weight, and overweight whole body images and asked to process each image in a self-referring way. Healthy controls had a pleasurable reaction to the normal weight body image (compared to the other two images), while AN patients had a much more pleasurable reaction to the thin body image (compared to the other two images). Given that activation in the ventral striatal reward system was higher during processing of underweight stimuli in the AN patients, the authors concluded that this differential activation toward disease-related stimuli was consistent with theories of starvation dependence.

Despite these findings that are compatible with the auto-addiction hypothesis of AN and the similarities of AN with addictions, many ED investigators are not convinced (Barbarich-Marsteller, Foltin, & Walsh, 2011). They emphasize "fundamental differences" between AN and SUD and conclude that "AN is not an addiction in and of itself." They note differences in several important areas, which are briefly reviewed in Table 13.3. The goals of SUD v. AN are different

Table 13.3 Differences between substance use disorders (SUD) and anorexia nervosa (AN) (adapted from Barbarich-Marsteller, Foltin, & Walsh, 2011)

	SUD	AN
Goal	Pursue drug	Pursue dieting/weight loss
Time course	Immediate	Immediate and long term
Reward	Intoxication	Hunger, self-control, thinness
Cultural consequences	Negative reinforcement	Positive reinforcement
Societal acceptance	Less acceptable	More acceptable
Addicted to?	Psychoactive substance	Absence of food intake
Obsessing about?	Using	Not using

in that the substance abuser is pursuing an immediate intoxication, while the person with AN is pursuing both the immediate and long-term effects of dieting, i.e., thinness and the illusion of control. The authors also point out important differences in the cultural and social consequences of SUD v. AN. Dieting and thinness are much more socially acceptable than the relative negative attitude toward and consequences of substance dependence. Finally, the other obvious difference is in what individuals with SUD v. AN are addicted to, i.e., using a psychoactive substance (drug) v. not using a substance (food). The authors do mention that "the excessive exercise observed in some individuals with AN may more closely resemble the pattern of drug use among substance abusers" (see below and Chaps. 7 and 28).

13.4.2 Exercise as Addiction

Excessive exercise is another problematic behavior associated with both AN and BN that has been described as an addiction (Berczik et al., 2012; Freimuth, Moniz, & Kim, 2011). This topic is extensively discussed in Chap. 7 and therefore will not be reviewed here in detail. However, the authors conclude that the concept of exercise addiction is indeed supported by the literature, although compulsive exercising may be a preferred term when it is associated with ED.

There have been a number of psychophysiological hypotheses to explain exercise addiction, and these include (1) a thermogenic hypothesis, (2) a catecholamine hypothesis, (3) an endorphin hypothesis (which is most widely known and empirically studied), (4) a serotonin hypothesis, and (5) a brain wave asymmetry hypothesis (Krivoschekov & Lushnikov, 2011). Additionally, there are two types of exercise addiction. In primary exercise addiction, the physical activity per se is an object of dependence. Secondary exercise addiction (most often seen in association with ED) appears to be related to decreasing body weight or to change the body's shape or size.

13.4.3 Binge Eating and Food Addiction

This section will discuss the issues of binge eating and food addiction. Patients with bulimic-spectrum disorders may binge on any type of foods, and the proportion of macronutrients in binge meals is no different from that of non-binge meals (Brewerton, Murphy, & Jimerson, 1994; Walsh, Kissileff, & Hadigan, 1989). However, carbohydrates are the predominant macronutrient ingested during binge eating, and these are often simple, high glycemic carbohydrates (or sugars) in combination with fats, thereby making these foods highly palatable and rewarding (Hadigan, Kissileff, & Walsh, 1989; Yanovski, 2003).

An emerging body of evidence over the last few years has characterized highly palatable foods as potentially addictive and evidence to support this idea that they act much like licit and illicit substances of abuse in the brain has been extensively documented (Avena, Gold, Kroll, & Gold, 2012; Avena, Rada, & Hoebel, 2008, 2009; Avena, Wang, & Gold, 2011; Benton, 2010; Fortuna, 2010; Gearhardt, Corbin, & Brownell, 2009; Gearhardt, White, & Potenza, 2011; Gearhardt et al., 2012; Gearhardt et al., 2011; Gold, Graham, Cocores, & Nixon, 2009; Hoebel, Avena, Bocarsly, & Rada, 2009; Joranby, Pineda, & Gold, 2005; Liu, von Deneen, Kobeissy, & Gold, 2010; Lustig, 2010; Sheppard, 2009). Specifically, sugar, fat, salt, and caffeine, which are common components of fast-food menus, have all been posited to have addictive properties (Cocores & Gold, 2009; Garber & Lustig, 2011). Eating such foods in excess may therefore become another strategy, just like any other addictive substance or behavior, that traumatized individuals use to numb themselves from unpleasant feelings and memories and to decrease emotional arousal (Brewerton, 2011) (see Chap. 17).

In support of this concept, Hirth, Mahbubur, and Berenson (2011) surveyed over 3000 women attending five public health clinics in Texas in regard to their (1) ED behaviors, (2) fast-food and sugary soda consumption, and (3) PTSD symptoms. The researchers reported a statistically significant link between PTSD symptoms and the frequency of (1) fast-food and sugary soda consumption and (2) ED symptoms, including severe dieting, purging, and compulsive exercising, but not with BMI. This study was the first to demonstrate a relationship between PTSD (and hence, trauma history) and eating specific types of foods known to be relatively unhealthy and associated with the concept of food addiction, i.e., highly palatable foods containing high concentrations of simple sugars and saturated fats, as well as salt and often caffeine (see Chaps. 1 and 4). The relationships between bulimic-spectrum ED and SUD to prior traumas and subsequent PTSD are discussed in detail in Chap. 17. Although the results of the Hirth et al. (2011) study did not find that women with PTSD had higher BMIs than those women without such symptoms, the women in this study were young, averaging only 20.8 years, and the effects of chronic excessive intake may not have had time to manifest. They were also engaging in more strategies to lose weight, i.e., dieting, vomiting, and smoking behaviors, which probably counteracted the inevitable weight gain that a steady diet of fast foods would incur.

The notion of food addiction is not a new idea, despite the surge in recent interest. Many individuals who have struggled with binge eating or compulsive overeating have independently and spontaneously identified the problem as a "food addiction." Many patients present with the chief complaint, "I have a food addiction," or "I'm addicted to food," or some variation of this statement. Interestingly, in a groundbreaking paper published well over 55 years ago, Randolph (1956) simultaneously described addictive drinking (of alcohol) and addictive eating, which he termed "food addiction." Although alcohol addiction continued to be a major focus of clinical research, nothing was written on the topic of food addiction until many years later.

However, largely inspired by the success of Alcoholics Anonymous (AA), Overeaters Anonymous (OA) was founded in 1960 in Los Angeles, California, by individuals self-proclaimed to be addicted to food. OA defined compulsive overeating as a progressive, addictive illness and focused on processed sugar as the addicting substance, a supposition that is no longer so implausible (Avena, 2010; Avena, Rada, & Hoebel, 2008, 2009) (see Chap. 1). Nevertheless, clinicians and researchers in the ED community have largely discounted that model and still do. This stance has inadvertently alienated many professionals in the addiction community as well as many patients who perceive their ED as an addiction. It is thought by many that one cannot possibly be addicted to food, since it is necessary for life, and the brain requires glucose to function properly. Carbohydrates and fats are basic energy sources. So how could food be addictive when it is required for survival? On the other hand, the modern Western diet contains inordinately high concentrations of high glycemic sugars and saturated fats that are engineered to be more and more irresistible and hedonically pleasing. The dosage and form of a substance determine its addictive potential, e.g., coca leaf (unprocessed) v. cocaine (processed).

Nevertheless, those individuals with ED have often described using food to self-medicate as well as being addicted not only to binge eating but also purging, dieting, and/or exercising. Often these behaviors are done in concert with each other in cyclic fashion, i.e., dieting, bingeing, and purging. Not only do these behaviors become habitual methods of regulating negative affect or mood (see Chap. 16), but eating foods with high hedonic value can rapidly, legally, and cheaply offer a measure of comfort, stimulation, and alleviation of psychic pain, however fleetingly. Of course, only more negative consequences follow as this pattern of behavior becomes habitual (addictive) in susceptible individuals who are exposed to frequent and high doses. A convergence of knowledge has led modern clinical neuroscience to recognize that certain foods can act like addictive substances in the brain despite the fact that they do have other peripheral metabolic effects that substances of abuse do not have.

Both animal and human experiments show that food intake and drug use each cause dopamine release in parts of the brain that mediate pleasure and emotion and that the degree of dopamine release correlates with the subjective sense of reward or experience of pleasure from both food and drug use. Similar patterns of brain activation as seen on fMRI in response to food and drug use have also been

described (Gearhardt et al., 2011; Volkow, Wang, Fowler, & Telang, 2008; Volkow & Wise, 2005) (see Chap. 4). In the case of food, reward mechanisms override homeostatic mechanism involved in regulation of feeding and metabolism (Lutter & Nestler, 2009).

Dr. Nora Volkow, the Director of the National Institute on Drug Abuse (NIDA), is a major proponent of food addiction in humans. Volkow and Wise (2005) stated, "To the degree that drugs and food activate common reward circuitry in the brain, drugs offer powerful tools to understand the neural circuitry that motivates food-motivated habits and how the circuitry may be hijacked to cause appetitive behaviors to go awry" (p. 555).

Dr. Bart Hoebel, the eminent Princeton neuroscientist who dedicated his career to studying the mechanisms of feeding and appetite regulation, and his colleagues at Princeton made the following statement: "Rats with intermittent access to food and a sugar solution can show both a constellation of behaviors and parallel brain changes that are characteristic of rats that voluntarily self-administer addictive drugs. In the aggregate, this is evidence that sugar can be addictive" (Avena, Rada, & Hoebel, 2008, p. 15).

Other studies demonstrate that food can stimulate the opiate system, and there are striking similarities in use and withdrawal patterns of sugar and classical drugs of abuse in certain animal models (Avena, Rada, & Hoebel, 2008) (see Chap. 1). There often appear to be reciprocal relationships among food and other substances. Individuals often gain weight when they stop smoking or drinking (Saules, Pomerleau, Snedecor, Brouwer, & Rosenberg, 2004). Recent research has shown that BMI is inversely proportional to alcohol intake, so the bigger one is, the less likely one is to drink (Kleiner et al., 2004). The authors noted that obese women have lower rates of alcohol use than those found in the general population of women. As BMI increases, lower rates of alcohol consumption are seen. Overeating may compete with alcohol for the same dopaminergic receptor sites in the brain, making alcohol ingestion less reinforcing. In addition, research shows that obese subjects who have lost weight following bariatric surgery sometimes start drinking excessively (see Chap. 9). King and colleagues (2012) reported that approximately 10–15 % of individuals who lose the weight from bariatric surgery and keep it off, 2–3 years down the road, are starting to report higher levels of drinking behavior. Taken together, these observations support the conclusion that food and classic addictive substances compete for the same pathways in the brain and may serve the same purposes psychologically. However, there may be other physiological explanations for these findings in the post-bariatric surgery population (see Chap. 9).

This reorientation of scientific research toward entertaining and validating the concept of food addiction has been a result of the fact that at least one-third of people in the USA are obese and that this number seems to be only increasing. Mortality and morbidity associated with obesity have become a major focus throughout medicine and public health. Using an extremely large database of two samples of over 39,000 subjects each, Grucza et al. (2010) demonstrated a link between risk for familial alcoholism and obesity, particularly in women. The

authors speculated that this link has emerged in recent years due to an interaction between a changing food environment and predisposition to alcoholism and related disorders.

13.4.3.1 Risk Factors

The argument that highly palatable foods can be addicting to certain people, especially traumatized people, is very compelling. As with other addictions, when exposed to substances such as alcohol, nicotine, illicit drugs, or behaviors, such as gambling, a certain subset of people are going to be highly rewarded by these substances or behaviors and will continue to use or engage in these behaviors. Others will say, "not for me," whereas others will want more and more, particularly those with reward deficiency. What do we know about those risk factors that will help us identify those who are at greatest risk for developing addictions? Interestingly, individuals with reduced dopamine type 2 receptor availability have a predisposition to both obesity and SUD (Tomasi & Volkow, 2013; Volkow et al., 2008; Volkow, Wang, Telang, et al., 2008; Volkow & Wise, 2005; Wang et al., 2001). Additionally, evidence suggests that engaging in addictive behaviors further downregulates D2 receptors and a reward deficiency syndrome is created. Given these interrelationships between addiction and obesity, a "reward deficiency syndrome" has been proposed to describe individuals with low D2 receptor density and polymorphisms of the D2 gene. This condition increases the risk for substance abuse, including alcohol dependence, heroin craving, cocaine dependence, methamphetamine abuse, nicotine sensitization, and glucose craving (Blum et al., 2011; Blum, Gardner, Oscar-Berman, & Gold, 2012; Blum, Liu, Shriner, & Gold, 2011).

In addition, obese subjects with BED have been reported to have an increased prevalence of the G/G (Comings & Blum, 2000) allele (A118G) of the μ-opioid receptor (OPRM1) (Davis et al., 2009), which has been associated with greater sensitivity to reward and an increased preference for sweet and fatty foods (Davis et al., 2011) and higher rates of substance addiction (Miranda et al., 2010; Ramchandani et al., 2011). Such sensitivity to reward is a personality trait that has been linked to both obesity and drug addiction. This predilection toward lower reward sensitivity is illustrative of the reward deficiency hypothesis that results in compensatory overconsumption (Comings & Blum, 2000).

Other risk factors for both SUD and ED are environmental, such as a history of sexual, physical, or emotional abuse or experiencing/witnessing interpersonal violence (see Chap. 17). Any traumatic experiences that produce PTSD or pPTSD symptoms are risk factors (Brewerton, 2004, 2007; Dansky, Brewerton, O'Neil, & Kilpatrick, 1997; Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012). Given what is known about gene and environmental reactions, many genetic disorders will be manifested only when the environmental triggers are present. It is further validation of the self-medication hypothesis of PTSD or of addiction. Victims of violence not only resort to addictive and/or ED behaviors and smoking to alleviate psychic pain but also preferentially select highly palatable foods containing high concentrations of sugar, fat, salt, or caffeine or the combination, sometimes to the point of addiction, in an attempt to dampen arousal and facilitate

numbing and avoidance, which are specific symptoms of PTSD (Brewerton, 2011; Schoemaker, Smit, Bijl, & Vollebergh, 2002).

These environmental risk factors are particularly relevant to children. It is well known that traumatic experiences are extremely common in children, who are especially vulnerable to the complex neurodevelopmental changes of early stress and addiction. Children are also quite likely to be exposed to fast foods that can subsequently lead to the use of these foods to self-medicate negative mood states. It has already been reported that food addiction is very much a feasible concept in children (Merlo, Klingman, Malasanos, & Silverstein, 2009; Pretlow, 2011). It has become nearly impossible to find foods without added sugar, especially high fructose corn syrup (HFCS), in grocery stores. Not only are these foods highly palatable and potentially addicting, evidence has suggested that those that contain high concentrations of HFCS may be literally toxic (Lustig, 2010).

Interestingly, animal studies inform us further about the intimate interrelationships between eating, stress, and relief (Maniam & Morris, 2012). A highly palatable cafeteria diet consisting of high fat and high sugar content ameliorates anxiety and depression-like symptoms in rats exposed to very early life adversity, i.e., with early separation (Maniam & Morris, 2010). This effect is mediated by the effects of feeding on the glucocorticoid receptor gene, a very fundamental early effect that involves food as a direct comfort to the nervous system. The anatomical circuits underlying feeding and stress responses are interconnected and operate in dual directions. What this means is that palatable food can actually reduce central stress responses at a genetic level, so comfort food works at a deep biological level to soothe the early effects of stress.

13.4.3.2 The Yale Food Addiction Scale

Gearhardt, Corbin, and Brownell (2009) developed and validated the Yale Food Addiction Scale (YFAS), which was based on the DSM-IV criteria of substance dependence among other measures. The goal of this study was to determine if there was a subset of BED patients that met the criteria for "food addiction." The authors administered the YFAS to 81 obese patients with BED, along with a number of other measures of psychopathology. The investigators were able to classify 57 % of this group of BED patients as *also* having food addiction. Those who were classified as such had significantly higher levels of depression, more negative affect, more emotional dysregulation, more ED psychopathology, and lower self-esteem (Gearhardt et al., 2012). These YFAS scores were significant predictors of binge eating frequency above and beyond other measures. This food addiction group identifies, even within BED, a particularly disturbed variant characterized by greater ED psychopathology and associated pathology. In another study of 96 obese patients with BED, a classification of food addiction was met by 39 (41.5 %) of BED patients (Gearhardt, White, Masheb, & Grilo, 2013). Similarly, those meeting YFAS food addiction criteria had significantly higher levels of emotion dysregulation, negative affect, and eating disorder psychopathology and lower self-esteem than those not meeting these criteria. In addition, higher scores on the YFAS were related to an earlier age of first being overweight and dieting onset.

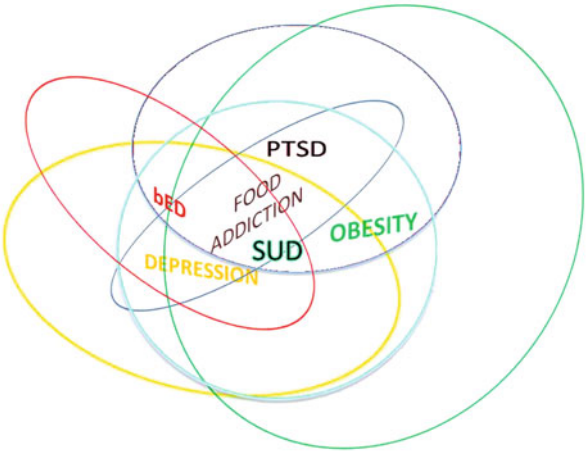

Fig. 13.1 Venn diagram depicting relationships between bulimic eating disorders (bED), substance use disorders (SUD), food addiction, posttraumatic stress disorder (PTSD), depression, and obesity

YFAS scores were also significant predictors of binge eating frequency above and beyond other measures. Unfortunately, the authors did not measure trauma history or PTSD in either of these studies. However, Mason, Flint, Field, Austin, and Rich-Edwards (2013) reported that 8 % of 57,321 women in the National Nurses' Study met criteria for food addiction using the YFAS, and the diagnosis of food addiction was significantly associated with a history of severe childhood physical and sexual abuse as well as obesity. Based on these findings, there appears to be a group of individuals (with and without a diagnosis of BED) who meet the criteria for "food addiction" based on the YFAS. See Fig. 13.1 that depicts the interrelationships between bulimic ED, food addiction, obesity, depression, SUD, and PTSD (Brady, Killeen, Brewerton, & Sylverini, 2000).

13.4.3.3 The Case Against Food Addiction

Several authors have been ardent critics of the food addiction hypothesis and have made a number of useful objections and arguments (Benton, 2010; Wilson, 2010; Ziauddeen, Farooqi, & Fletcher, 2012; Ziauddeen & Fletcher, 2013). The salient features of the case against food addiction will be summarized here.

1. One question involves the specific identity of the so-called addicting substance, and why it does not manifest as the preferred substance in laboratory studies of eating behavior? The macronutrient content of meals consumed by individuals with BN and BED has not been shown to substantially differ from that of controls, and carbohydrates (CHO) do not necessarily improve mood or relieve negative affect (Wilson, 2010). These are no doubt fair points. Nevertheless, the most commonly consumed types of food by binge eaters and non-binge eaters alike are CHO and FAT. Thus, the combination of CHO and FAT in much larger quantities, or doses, is in fact what binge eaters are most likely to consume (Brewerton, Murphy, & Jimerson, 1994; Hadigan et al., 1989), and the dose of a substance is an important factor in whether it causes addiction. What has not

been well documented in laboratory studies of binge eating is to what extent high glycemic versus low glycemic CHO are preferred by binge eaters, much less binge eaters who are also "food addicts." However, Hadigan et al. (1989) noted that patients with BN spent more of their mealtime eating dessert and snacks than did control subjects, and they also began their dessert and snack consumption earlier than control subjects. Tanofsky-Kraff and coworkers (2009) reported that children with loss of control (LOC) binge eating (a core feature of binge eating and food addiction) consumed more high-calorie snack and dessert-type foods than did those without LOC when presented with an array of different types of foods. In another study of patients with BED, Yanovski and colleagues (1993) reported that adult patients with BED consumed a greater percentage of energy as fat during a binge meal as well as a lesser percentage as protein than did subjects without BED. In other words, available data would suggest that it is highly palatable foods, which are both sugary and fatty that may be the potentially addicting combo. Nevertheless, the specific addicting agent or agents do need to be further clarified in controlled, scientific studies.

2. Wilson (2010) claims that "epidemiological data are inconsistent with an addiction model" of bulimic ED. Although higher rates of SUD are seen in bulimic ED, and vice versa, he rightly points out that substance abuse is not specific to ED and that depression is a more common comorbid disorder for both SUD and ED.
3. Wilson (2010) also notes some evidence that SUD and ED do not show evidence of a shared genetic or familial etiology. However, the results are mixed. This issue is discussed at length in Chap. 5.
4. One of the best arguments against the food addiction model is the simple fact that the core psychopathology of bulimic-spectrum ED is more complex than simply binge eating. This includes the "undue influence of body weight or shape on self-evaluation" as well as the "overvaluation of body weight or shape" (American Psychiatry Association, 2013) that is characteristic of all ED types and subtypes (Wilson, 2010). Individuals with BN, and to some extent, also those with BED, engage in significant dietary restraint and are distressed by their inability or difficulty controlling their eating. Wilson (2010) quotes Fairburn (1995) that "there is no equivalent phenomenon in SUD." However, this is highly disputable and at best doubtful. Certainly there are individuals with SUD who want to stop using but because of impaired control and/or distress intolerance repeatedly relapse and are subsequently distressed, demoralized, and/or depressed by their failed efforts to maintain abstinence.
5. Another important argument against the food addiction concept is the lack of evidence for withdrawal effects in humans, which has been discussed earlier. However, the food addiction field is new and good studies have not yet been designed or implemented to test this hypothesis in humans. In addition, it is notable that without exception every substance that has ever been found to be addicting in animals has subsequently been shown to be addicting in humans.
6. Wilson (2010) also correctly makes the point that outcome research clearly supports the efficacy of CBT in the treatment of BN and BED (see Chap. 24),

while the "diametrically opposed" 12-step approach has never been studied in bulimic ED (see Chap. 27). However, he does not mention that CBT is also effective for a variety of SUD and related comorbid disorders (see Chap. 24). In fact, in Project MATCH (Project MATCH Research Group, 1997), response to CBT was found to be no different from 12-step facilitation therapy or from motivational enhancement therapy (MET) in a large group of patients with alcohol abuse followed long term. Furthermore, the efficacy of CBT for BN and BED in no way disproves the food addiction hypothesis. It is highly questionable that CBT is "so effective" that it "represents a refutation of the addiction model." In many of the major trials of CBT in BED and BN, major comorbid disorders such as SUD were excluded, while other trials have shown that comorbidity is a negative prognostic factor (Castellini et al., 2012). Finally, the abstinence model does not necessarily "encourage dietary restriction" in the classical sense of dieting, generalized food restriction, or restrained eating. Instead certain foods or types of foods are eliminated, while a healthy meal plan consisting of all major food groups, vitamins, and minerals is encouraged or prescribed (see Chap. 23).

7. Politically and socially speaking, many people object to the term "addiction" in reference to food or ED given its negative connotations. As one prominent ED researcher said to me, the term has "baggage," and this is partly why the term was avoided for so long by the American Psychiatric Association (O'Brien et al., 2006). In addition, if addiction is a chronic disease, then what hope is there for those with ED to fully recover? Presumably a substantial number of ED patients do fully recover from their overt symptomatology (Fichter, Quadflieg, & Hedlund, 2006; Fichter et al., 2008; Strober, Freeman, & Morrell, 1997). However, there are important personality traits, such as perfectionism, obsessionality, harm avoidance, novelty seeking, and impulsivity that remain and may predispose to relapse. Is it accurate that patients with SUD can never fully recover? Probably not. But there has been intense philosophical debate as to whether anyone with a SUD or an ED is ever fully "recovered" versus always "recovering," which is more traditionally the position of 12-step programs. However, there is very little good, comprehensive data to illuminate this issue. The fact of the matter is that both SUD and ED patients have highly variable courses, with some doing extremely well and never relapsing on one end of the spectrum and some never improving and dying on the other.

13.4.4 Purging as an Addiction

What about purging as an addiction? Purging specifically refers to vomiting, laxative abuse, and/or diuretic abuse, each of which will be discussed in terms of its addictive potential. Vomiting is the most common compensatory behavior associated with BN, AN-BP, and subclinical ED (see Chap. 11).

13.4.4.1 Vomiting

Consider the following statement by a young woman in a blog on the Internet: "*I am only 17 years old, and I'm scared that I am becoming bulimic. One day I was crying very hard, so hard that I vomited, and after vomiting I felt relieved. What happened is that after that day, each time I'm either crying, sad, or angry, I either force myself, (which is so very easy to me), or naturally vomit. It's only after vomiting that I feel relieved and all right. I can't stop doing it because it makes me feel so much better. Am I addicted to it? Is it unhealthy? I haven't had any weight loss or anything like that.*"

This perspective provides a different twist on vomiting behavior, which in this case is not only being used as a modulator of mood but is also clearly becoming habitual to the point that she is concerned about whether she is "addicted." This case illustrates an under recognized aspect that vomiting is not just about "undoing" the effects of binge eating and getting rid of calories. It is often just as much about the emotional regulation effect that it has. Like binge eating, vomiting may have a distinct calming effect, both simultaneously decreasing negative affect and increasing positive affect (see Chap. 21). After binge eating, individuals are typically extremely anxious and even panicked about the increased intake and inevitable resultant weight gain, and vomiting is a way of relieving the physical and mental distress, calming down, getting rid of, and feeling better. Sometimes it happens that people get so upset that they actually vomit as a result of the emotional activation. This can happen naturally, as in "psychogenic vomiting," which appeared in the literature well before BN was described (Hill, 1968). In fact, Breuer and Freud (1893) noted that chronic vomiting was associated with emotional disturbances well over 100 years ago.

Vomiting results in release of the major stress hormone corticotropin-releasing hormone (CRH), which stimulates ACTH to release cortisol from the adrenal glands (Abraham & Joseph, 1986–1987; Kaye, Gwirtsman, & George, 1989). Plasma cortisol is known to increase after bingeing and purging, and with repeated episodes, it remains elevated for some time (Fullerton, Swift, Getto, & Carlson, 1986). So vomiting both relieves and creates stress at the same time. Whenever the stress hormone cortisol is stimulated, beta-endorphin is automatically released as well because it is cleaved off of the larger pro-opiomelanocortin molecule. When stressed, the body provides an endorphin pain relief. Evolutionarily, stress meant threat, which in turn meant possible impending pain, so a self-protective beta-endorphin release is produced, and this happens with vomiting. Over time, repeated vomiting causes a depletion of this peptide and opiate-receptor downregulation because of these chronic behaviors, and intermittent binge eating and semi-starvation only add to that. As discussed previously, the opiate effect of dieting results in decreased central beta-endorphin over a time, and more vomiting may be required to produce the same opioid effect.

This hypothesis is compatible with other findings. There are several studies that show that ED patients have a higher pain threshold, probably because of this opiate effect (Abraham & Joseph, 1986–1987; Stein et al., 2003). Brewerton and colleagues (1992) reported significantly decreased cerebrospinal fluid (CSF) levels

of beta-endorphin in women with BN in comparison to healthy controls. CSF concentrations of beta-endorphin were inversely correlated to measures of depression, suggesting that bingeing and vomiting aggravate mood disturbance, much like the secondary depression seen in SUD.

In desperation, some people have resorted to using emetine (syrup of ipecac) to induce vomiting. Emetine is a medicine previously used in emergency rooms to induce vomiting after poisonings. It is extremely toxic to muscle tissue, especially the heart, and its toxicity is cumulative over time. Syrup of ipecac used to be available over the counter, but it was taken off the shelves because of its toxic side effects. In addition, since 2010, its production has been discontinued. However, as of this writing, it can still be obtained online. The use of emetine to facilitate vomiting illustrates the compulsory or addictive nature of vomiting, which can become a goal that must be accomplished at all costs.

Recurrent, chronic vomiting may also result in dopamine depletion and therefore may predispose toward other addictive behavior. In a prospective study, Field et al. (2012) reported that female participants with purging disorder (PD) had a significantly increased risk of starting to use drugs (OR: 1.7) and starting to binge drink frequently (OR: 1.8).

13.4.4.2 Laxatives

The abuse of laxatives is a well-known compensatory behavior seen in patients with bulimic-spectrum disorders (see Chap. 12). The stimulant type of laxatives is the most dangerous since they typically produce excessive fluid and electrolyte losses leading to a variety of complications (see Chap. 15). Laxatives have very little effect on caloric absorption but rather cause evacuation of the colon and significant fluid and mineral losses. Laxatives, which are often taken in overdose, do produce weight loss resulting from diarrhea, loss of water weight, and resultant dehydration, as well as major discomfort due to abdominal and muscle cramping. The decrease in the number on the scale is highly rewarding, but then there is rebound water retention and edema that leads to anxiety and more abuse, and a vicious addictive cycle is established. Laxatives are not addictive per se in the classical sense (producing intoxication), but it is the process of using them repeatedly to excess with the mind-set that weight loss will be achieved no matter what that is problematic. Tolerance builds with stimulant laxatives, and abusers take more and more to achieve the desired effects. Therefore, the use of laxatives can be thought of as a behavioral or process addiction.

13.4.4.3 Diuretics

The use of diuretics is less common than vomiting or laxative abuse but nevertheless occurs in a substantial minority of patients. Diuretics have no effect on caloric absorption whatsoever, but individuals may resort to using them in an attempt to decrease bloating and swelling, which may be in part rebound phenomena from bingeing, vomiting, and/or laxative abuse. As with laxative abuse, weight loss is achieved via loss of water weight. The decreased weight on the scale has an extremely powerful reinforcing effect; however, chronic diuretic use leads to

rebound edema and in turn leads to further use. Diuretic abuse can cause major medical problems, including chronic kidney disease from hypokalemic nephropathy.

13.5 Treatment Implications

Why does it matter whether ED are called addictive disorders or not? How might one treat dieting, exercising, binge eating, and purging using the insights gleaned from an addictive perspective? From one standpoint, it may not matter at all. The treatment goals are similar if not identical for most behaviors. Normalization of weight and the cessation of dieting, excessive exercising, binge eating, purging, and the use of harmful substances to facilitate weight loss remain primary goals, followed by reduction of anxiety, normalization of mood, and resolution of traumatic or conflictual issues. Ultimately, acceptance of self, including body self, is a goal common to ED. However, how these goals are approached and achieved may be modified as discussed below using an addiction perspective.

13.5.1 Dieting

Diets don't work in the long term for the treatment of obesity, as most people regain all of their lost weight and more regardless of the type of diet or pharmacologic agent employed. Extreme dieting is a well-recognized risk factor for the initiation of bulimic-spectrum disorders (Brewerton, Dansky, Kilpatrick, & O'Neil, 2000) and a trigger for relapse. A core goal of treatment for all types of ED, including food addiction, is *abstinence from dieting*. Diet is a "4-letter word" in the ED world. Instead of using the word "diet," which implies restriction of intake or restrained eating, it is preferable to use the term "meal plan." Following a reasonable, well-balanced meal plan is an essential part of nutritional rehabilitation, weight restoration and/or stabilization, healthy eating, and a healthy life. This is an essential ingredient to the nutrition therapy and cognitive behavioral therapy for ED (see Chaps. 23 and 24).

13.5.2 Exercising

Since weight restoration is a primary goal in the treatment of AN, exercising and resultant energy expenditure are generally antagonistic to this process and often add danger to the medically compromised. ED patients who have been abusing exercise often need a rest. It is anti-therapeutic to be doing much of any kind of aerobic exercise, certainly, in the weight gain phase and in the early phases of treatment for BN—until medical stabilization. Initially, very gentle stretching or yoga or meditation is allowed and even encouraged as an anxiety-reducing strategy, but any intense exercise is not recommended. In a later phase of treatment, exercising to

moderation is encouraged in weight-restored patients with AN as well as in individuals with BN and BED. Exercising in moderation is obviously important to overall health and weight maintenance. The degree and frequency of exercise allowed and prescribed depend on the illness being treated and the clinical status at the time. Moderation is the rule, which is part of good CBT. Please see Chaps. 7 and 28 for a more detailed discussion of the treatment of exercise addiction and use of exercise as treatment.

13.5.3 Purging

Complete cessation of all purging behavior is a clear goal of treatment regardless of one's perspective on the addiction model of ED. Vomiting behavior and laxative abuse should be stopped as soon as possible, regardless of any possible withdrawal effects. Patients who have been taking high doses of diuretics chronically usually need to be tapered gradually off of these substances to avoid extreme rebound edema and associated triggering of any and all ED behaviors to compensate and respond to worsening negative affect. If constipation is a problem, the use of stool softeners, liberal fluids, and enhancement of dietary fiber intake are usually sufficient to treat this effectively (see Chaps. 12 and 15).

13.5.4 Binge Eating

The primary goal of any treatment approach is to move toward *abstinence from binge eating* while developing a healthy relationship with food. However, the comorbid presence of food addiction complicates this situation. One can become abstinent from binge eating, but not from food itself. However, the elimination of specific "binge foods," which patients may perceive they are addicted to, may be eliminated at least at the beginning of treatment in order to facilitate abstinence from binge eating. Again, it's a matter of moderation, and sometimes it may be a matter of identifying a hierarchal list of which foods are most triggering. The OA idea of avoiding highly refined, white processed carbohydrates is still an open, unresolved question, but as this chapter has attempted to illustrate, this approach may be highly relevant and appropriate for a subset of individuals with bulimic-spectrum disorders, perhaps those who score high on the YFAS. The high fluctuations in glucose levels that come from ingestion of foods containing high concentrations of simple sugars or high glycemic carbohydrates result in high levels of insulin that in turn drives essential amino acids, such as L-tryptophan and L-tyrosine into cells and across the blood–brain barrier to causes sudden surges in monoamine neurotransmitter levels (Brewerton, 1995). Glucose and insulin very much influence L-tryptophan and L-tyrosine uptake into the brain and subsequent neurotransmitter function, including serotonin, norepinephrine, and dopamine, all of which are prominently involved in ED, particularly serotonin in bulimic disorders (Brewerton, 1995) (see Chap. 3). The intermittent bingeing on "high

doses" of high glycemic carbohydrates can cause an intermittent surge of serotonin and dopamine, which in turn causes downregulation of serotonin and dopamine receptors and a reward deficit. Recent research has revealed that DA and insulin systems do not operate in isolation from each other but rather work together to regulate the motivation to engage in consummatory behavior and to adjust the associated level of reward. Insulin signaling regulates DA neurotransmission and affects the ability of drugs that target the DA system to exert their neurochemical and behavioral effects (Daws et al., 2011).

Carbohydrates are a necessary component of a healthy diet, but the kind of carbohydrates eaten may significantly influence the course of illness. There is sound reason to believe that working toward eating primarily low glycemic carbohydrates may be very advantageous in avoiding binge eating. As previously noted, the idea that some people with binge eating are addicted to high sugar, high fat foods has been empirically validated (Gearhardt et al., 2012, 2013; Gearhardt, White, & Potenza, 2011). No matter how stable one may become, eating certain foods can be triggering and destabilizing. This is not simply a psychological matter but a physiological one as well. Blouin et al. (1993) reported that patients with BN who received double-blind, placebo-controlled injections of glucose experienced heightened urges to binge at 10 and 60 min (compared to placebo), whereas healthy controls experienced reduced food cravings for sweets. Page and colleagues (2011) tested the hypothesis that circulating levels of glucose influence brain regions that regulate the motivation to consume high-calorie foods. The authors demonstrated that mild hypoglycemia preferentially activated limbic-striatal brain regions in response to food cues to produce a greater desire for high-calorie foods. However, euglycemia preferentially activated the medial prefrontal cortex and resulted in less interest in food stimuli. Higher circulating glucose levels predicted greater medial prefrontal cortex activation, but this response was absent in obese subjects. These findings demonstrate that circulating glucose modulates neural stimulatory and inhibitory control over food motivation and suggest that this glucose-linked restraining influence is lost in obesity.

Traditionally, the ED community has not been receptive to the idea of eliminating specific "binge foods." The dietary dogma has been that there are "no bad foods" and that all foods should be eaten in moderation. No foods should be eliminated according to this perspective unless there is a clearly and carefully diagnosed food allergy or intolerance, e.g., gluten intolerance with celiac disease. This viewpoint maintains that one can always eat a little bit of any "forbidden" food and that prolonged exposure will result in a desensitization and extinction of the fear of specific foods. However, as successful as CBT has been for BN and BED, it is not effective for everyone, with on average only 25–50 % of patients in RCT becoming completely abstinent, and relapse rates are high. Perhaps some people with bulimic ED get so triggered by highly refined sugar and fat that they are better off just not being exposed to it, at least for some time. Some patients don't want to be exposed to such foods, and the foundation upon which therapists and dieticians insist that they do is now on shaky ground. It is important to listen to patients, who often tell their treatment providers exactly what they need.

The usefulness of 12-step programs has been discussed in Chaps. 12 and 27, both as a self-help tool and as a therapist-facilitated approach. There is evidence of its utility in the treatment of SUD, but not ED; it hasn't been systematically studied in this population.

What is especially questionable to many professionals in the ED field is OA's prohibition on white flour and sugar. Anecdotally, many individuals report that avoidance of white flour and sugar is extremely useful. Furthermore, the success of low carbohydrate diets, such as the Atkins diet, for the treatment of obesity, attests to the feasibility of this approach, at least in some individuals (Casazza et al., 2012; Iqbal et al., 2010; Maeir et al., 2011). In the treatment of individuals with food addiction, OA can be offered in an unbiased manner as an option to be explored that may or may not work.

Another issue for ED professionals is that OA (like AA in the treatment of alcoholism) is a viable substitute for professional intervention. Conceptualizing an ED as an "addiction" does not imply that professional intervention in the treatment of an ED is not essential. Programs such as OA are considered as adjuncts to treatment, not the foundation of recovery. Twelve-step programs provide needed fellowship, have the advantage of being free and readily available, and are considered the most successful self-help programs worldwide. Patients can be encouraged to attend one or more meetings and decide for themselves if OA philosophy and practice is for them. They can also follow the 12 steps without adhering to the dietary guidelines. This kind of stance with individuals with bulimic ED can be useful (Giannini et al., 1998; Johnson & Sansone, 1993; McAleavey, 2008).

There is a lot of overlap between the types of therapies that are used in ED and addictions, which a large portion of this book reviews. When ED and SUD occur together, the rates of PTSD and prior trauma are higher than with each disorder alone (see Chap. 17). Often the PTSD symptoms are obscured or hidden until the ED symptoms and the addiction symptoms are well under control. In a major way, the treatment of ED and SUD and related comorbidity requires a phasic yet integrated approach to treatment. One starts with weight restoration, nutritional rehab, and/or detox first, and then one moves into the intensive psychotherapy phase, which is followed by the maintenance phase upon successful resolution of symptoms.

The decision of what to focus on first after a full medical and psychiatric evaluation is still debatable, but there is general consensus that it is a matter of which disorder or problem is most life threatening. What symptoms have the greatest lethality? Alcoholic delirium tremens is obviously a life-threatening condition that must take precedence. Severe hypokalemia and associated cardiac arrhythmia or seizures also have high priority. So sometimes it is the SUD and sometimes it is the ED that takes precedence. The medical and psychiatric sequelae of both ED and SUD need to be simultaneously addressed. This is a matter of good clinical judgment in collaboration with patients and their families.

Often when patients are not ready to change, one needs to start with motivational interviewing (MI) or motivational enhancement therapy (MET) to help them get ready to do the work that is required for recovery. Patients are often in different

stages of change depending on the symptom that is being discussed. Some patients are ready to work on ED symptoms but are not ready to address their SUD. Likewise, some patients are actively ready to confront their SUD but not the ED. Programs that offer comprehensive, integrated services for this comorbid condition provide the best opportunity for recovery from both disorders (see Chap. 21).

Conclusions

In summary, there continues to be a significant chasm in both the ED and SUD field in the conceptualization and treatment of patients that present with both an ED and SUD. In the past, similarities between these two disorders have centered on clinical presentation and behavioral similarities. However, emerging evidence in animal research, genetics, and neuroimaging has provided further evidence that supports the addictive nature of ED behaviors, including dieting (starvation dependence), compulsive exercising (exercise addiction), binge eating (food addiction), purging (purging addiction), as well as the addictive use of a variety of substances to promote weight loss (appetite suppressants, lipase inhibitors, ipecac, laxatives, and diuretics). All of these behaviors meet the contemporary definitions of addiction, including those delineated in the DSM-5 and ASAM definitions of addiction. Despite the fact that food and drugs of abuse act on the same or similar central reward networks, food consumption is also regulated by peripheral signaling systems which adds to the complexity of how the body regulates eating and manages pathological eating habits. Traditional pharmacologic and behavioral interventions for other SUD, however, may prove useful in treating obesity and ED, and the field is wide open in terms of the research that needs to be done in this area of overlap. The addiction field and the ED field need to work much more closely together in order to make further progress in research and treatment in order to further delineate the similarities and differences between these two classes of disorder.

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