6 Clinical Evidence for the Validity of Food Addiction

6.1 Shared DSM Criteria

There are 11 criteria for a DSM-V diagnosis of a substance use disorder (SUD). These criteria can be categorized into four categories: impaired control, social impairment, risky use, and physiological criteria [3] (Box 6.1). Severity is classified as mild (2–3 symptoms), moderate (4–5 symptoms), or severe (> or = 6 symptoms), and symptom assessment should cover the previous 12 months [3]. Although not in the DSM-V criteria, SUD also, importantly, are vulnerable to relapse after long periods of sobriety [3].

Box 6.1 DSM-V Criteria for an SUD [3]

Impaired Control

1. Substance consumed in amounts or over longer periods of time than intended.
2. Persistent desire or unsuccessful attempts to decrease or limit substance use.
3. Significant amount of time spent acquiring, using, or recovering from a substance.
4. Strong craving to use the substance.

Social Impairment

5. Inability to fulfill obligations at work, school, or home due to use of a substance.
6. Continued use despite recurrent exacerbation of social or interpersonal problems.
7. Reduced engagement social, occupational, or recreational activities due to substance use.

Risky Use

8. Continued use of the substance in situations in which it is physically dangerous (e.g., driving under the influence).
9. Continued use despite physical or psychological problems that are caused or made worse by the substance.

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Physiological Criteria

10. Increased dose needed to achieve the desired effect and reduced effect when consumed (i.e., tolerance).
11. Negative physiological or psychological effects upon cessation (i.e., withdrawal).

Clinical distress or impairment must be evidenced by two or more of the above symptoms within a 12-month period.

People with obesity and eating disorders (EDs) experience impaired control over food, especially HP food [1, 7] (Chap. 11). Dieters usually fail to lose significant amounts of weight or maintain their weight loss, and, for some, dieting may ultimately lead to more weight gain, in the long run [1, 8]. Indeed, loss of control is a criteria in the DSM-V to diagnose binge eating disorder (BED) and bulimia nervosa (BN) (e.g., impaired control over food quantity of and length of time engaged in food consumption, consumption of large amounts of food in a short period of time, an inability to successfully stop or cut down on consumption despite an expressed desire to do so, and recurrent negative consequences) [1, 9, 10].

Food craving typically refers to an intense desire to consume a specific food [1, 11], is very commonly reported by people with obesity and eating disorders (ED)) [2], and occurs even in normal-weight individuals, with reports of 100% of women and 70% men experiencing a craving for at least one food in the past year [1]. Several standardized questionnaires to measure food cravings have been developed and show good internal consistency and construct validity [1]. More frequent and intense food cravings are associated with binge eating, increased food intake, self-reported FA symptoms, increased body mass index (BMI), BN, and BED [1, 12]. FA severity is also correlated with difficulty controlling eating and cravings and higher appetite ratings [3, 13, 14]. With more and more use, the reward system is primed, and craving increases. For example, in women who reported craving carbohydrates, a 100% carbohydrate sweetened beverage dispelled craving, but this quelling effect decreased over multiple exposures [3, 15]. That said, there may be a ceiling effect, as supported by several bodies of work. For example, in one study of morbidly obese individuals, as BMI increased, craving decreased [16, 17]. In the context of both food and alcohol or drug use, craving is often accompanied by rationalizations and justifications that lead the individual to engage in food or drug-seeking behavior that they had previously sworn against.

The SUD criterion of time spent obtaining, using, and recovering from use also translates to people who overeat, especially for those with BED and bulimia nervosa, in that bingeing is often a planned behavior which may require a great deal of effort to purchase and store foods ready for a binge episode [1]. Indeed there is a threshold for ED diagnosis based on this category, in that three episodes of binge eating per week are required to meet criteria for BED [1]. Moreover, people with BED often experience physical and emotional distress following a binge eating episode, and food addicts report feeling sleepy or "hung-over" [1, 7, 18] after overeating.

From the standpoint of social impairment, disorders of overeating and obesity are stigmatized (Chap. 5) resulting in social isolation. In one study, 60% of a sample of bariatric surgery candidates endorsed choosing to spend time eating over conducting other activities, and their addictive personality scores explained a significant amount of variance in social isolation [3, 19]. Those who meet criteria for FA score lower on physical, mental, and social aspects of health-related quality of life scales [1, 20] and report lower self-esteem, impaired sexual life, and poorer work performance. Interpersonal problems have also been associated with binge eating – a relationship which is likely to be bidirectional [1].

From the standpoint of risky use, many obese individuals continue to eat unhealthy foods even in the face of severe negative consequences, such as diabetes, heart disease, and stigmatization [9, 10]. In EDs, individuals continue to binge eat despite physical and emotional distress [1] and despite increased risk for severe neurological,

cardiac, dental, and gastrointestinal medical consequences (Chap. 3). Furthermore, those who have undergone weight loss treatment often fail to lose weight or gain weight following intervention [1, 21], even after bariatric surgery [1, 22].

Numerous studies have shown that tolerance to HP food occurs [3, 23]. Neurobiologically, tolerance is manifested by reduced dopamine receptor density (in particular, the type-2 dopamine receptor, due to downregulation), and blunted dopamine and opioid release to natural rewards, both of which have been observed in animal models and humans with obesity and EDs [3, 23] (Chap. 8). Clinically, tolerance may manifest as anhedonia or hyposensitivity to reward in obese participants compared to controls [1, 24, 25]. For example, one study showed that although overweight women were more sensitive to reward than healthy-weight women, those who were obese were significantly less reward-sensitive than overweight women [1, 25]. Another study showed that those who regularly consumed ice cream exhibited a blunted reward-related neural response to a small portion of ice cream, relative to those who did not frequently eat ice cream [2, 26]. Finally, another interesting study supporting the existence increased tolerance over time reported that although sucrose has an analgesic effect in young infants, but this diminishes as they get past 18 months, which is the time when sugar consumption increases [1, 27]. Like with drugs of abuse, tolerance may lead animals and humans to consume progressively increased amounts of food, in an attempt to achieve its beneficial mood-enhancing effect [1, 3, 23–25]. For example, normal weight participants provided with chocolate for 3 weeks increased their intake over time while at the same time reporting a reduction in food liking [1, 28]. In a study of bariatric surgery candidates [3, 19], 69% reported increasing quantities of food to reach satiation over time, and those who endorsed this symptom also had higher scores on an addictive personality measure. In BED, as the illness duration grows longer, the frequency of binges, the amount of food consumed, the length of the episode, and the feeling of being out of control all increase [9, 10].

Withdrawal is defined by the presence of physical or psychological symptoms in response to substance deprivation and/or the use of the substance in order to relieve these symptoms [1]. Like with tolerance, numerous animal models have demonstrated withdrawal behaviors upon cessation of high sugar or high fat foods that mirror behaviors that occur during withdrawal from drugs, including teeth chattering, forepaw tremor, head shaking, and reduced body temperature as well as increased aggression and anxiety [1] (Chap. 8). Human studies also support the existence of food withdrawal [5, 6, 29] which is marked by affective, cognitive, and physical symptoms [30]. Anecdotal reports indicate that when individuals reduce their consumption of highly processed foods, they experience cravings and negative affect as well as fatigue, anxiety, depression, and agitation [1, 2, 7, 9, 31]. For example, a case study documented the reemergence of anxiety and panic symptoms when a patient went on the Atkins diet (a low-carbohydrate diet), and Atkins himself warned dieters that they may experience "fatigue, faintness, palpitations, headaches, and cold sweats" [9, 10]. Although the frequency of withdrawal symptoms in various clinical populations varies, withdrawal has been reported in up to 50% of individuals with obesity and BED [1, 31]. However, rates were lower in a study of 1414 participants who had at least one YFAS symptom in the last year, where 10% endorsed "withdrawal-like" effects in response to cessation of either high-fat savory foods (4%), high-fat sweet foods (3%), low-fat sugary foods (2%), or low-fat savory foods (1%) [3, 32]. Importantly, in humans, withdrawal may hinder dietary change [30]. People with withdrawal symptoms report the tendency to eat to avoid the emotional symptoms such as fatigue, anxiety, and depression [1, 7]. Another study of bariatric surgery candidates showed that higher scores on an addictive personality measure was related to higher levels of anxiety when they were not near food [3, 19].

A food withdrawal scale called the Highly Processed Food Withdrawal Scale (ProWS) has been developed and validated with the YFAS for use in adults [30, 33] and children [30], supporting the validity of an HP food withdrawal construct, although it's important to note that the original vali-

dation study was done on individuals who had been trying to diet, and the questions were retrospective, and so the possibility that some of the symptoms were due to a caloric deficit cannot be ruled out [2, 33]. The ProWS validity studies utilized items from the Wisconsin Smoking Withdrawal Scale and Cannabis Withdrawal Scale [2, 33] including psychological symptoms (e.g., craving, anxiety, irritability) and physical symptoms (e.g., headaches, sleep disruption). The time course of withdrawal from food has been measured to peak at 2–5 days, and then symptoms improved over time, similar to what is seen with many drugs of abuse [2, 33]. In adults, higher scores are related to higher addictive tendencies less self-reported success with dieting attempts and impaired dietary restraint in adults [2, 33]. In children, the scale shows discriminant validity with child food neophobia, and higher scores are associated with less success in reducing child highly processed food intake independent of BMI [30].

Although relapse has not been officially investigated in human studies of obesity and binge eating disorder, clearly the fact that people end up usually gaining back weight they lost (Chap. 4) indicates that a relapse-like phenomena exists in relation to food. Indeed, animal and neuroimaging studies show that the neurobiological underpinnings of relapse to SUD and overeating have significant overlap [3] (Chap. 8).

6.2 Yale Food Addiction Scale Development

The YFAS [9, 34] was originally published in 2009, and revised in 2016 (YFAS 2.0), and it is currently the only validated measure to operationalize FA and addictive-like eating behavior [2]. The original YFAS and the updated version have been translated and validated in many languages and for many different cultures [2, 34].

The original YFAS is a self-report questionnaire that screens for the seven DSM-IV [35] symptoms of substance dependence as applied to the addictive-like consumption of certain foods, prompting for peoples' experiences in relation to HP foods in particular (e.g., foods with high amounts of sugar, processed carbohydrates, fat or salt) [2, 9]. Like for SUD, a diagnosis is obtained if three symptoms are endorsed above a certain severity threshold plus clinically significant impairment or distress [35]. It asks about whether the symptoms have been experienced over the last year.

The advent of the DSM-V saw major changes in how SUD were evaluated, merging criteria for substance abuse and dependence into one diagnosis, adding craving as a criteria, removing legal consequences, listing eleven total criteria, and applying a severity spectrum (mild, moderate, severe) [36]. In response, the YFAS 2.0 was developed in 2016 to parallel these changes [2, 34]. Like the original YFAS, it produces both a continuous score based on the number criteria met, and a cutoff score, and like in the DSM-V, severity can be determined from the number of criteria met [34].

The validity of a test is established if it is shown to measure what it claims to measure. Construct validity has to do with whether a test captures a constellation of symptoms that truly exist in the real world [37]. In our case, a test of FA should measure a true biologically based set of addictive behavior patterns around food and eating and symptoms that are problematic for some individuals. A subtype of construct validity, named convergent validity, depends on the internal consistency of a measure and requires that the symptoms of a proposed syndrome cluster together in the sample [9, 34]. For example, in the case of FA, internal consistency of a measure of FA would be established if withdrawal symptoms also seem to occur in a population of people who report continued use despite negative consequences [9, 34]. A test of FA would also have convergent validity if it related something else to which it should theoretically relate, like weight gain or more severe binge eating [37, 38]. Finally, a good test needs to capture something that isn't already captured by our existing constructs or diagnoses, and not be redundant [39]. This is determined through tests of discriminant validity, another subtype of construct validity, or incremental validity [34, 38]. For example, a tool to determine FA needs to measure something that is not captured already by BED, bulimia nervosa, or

obesity. There is no single test for construct validity, and the more evidence to support it, the better [37].

The original YFAS exhibited excellent internal consistency [2]. It also demonstrated adequate internal reliability, and good convergent validity, being associated with BMI and binge eating [9]. The YFAS 2.0 demonstrated even better internal consistency [2], loading onto a single factor, and also had good reliability, similar to the original YFAS [34].

The YFAS 2.0 is apparently more inclusive than the original YFAS, and, in a sample of more than 200 participants, individuals were more likely to meet criteria for FA on the YFAS 2.0 than the YFAS (16 versus 10%). This makes sense, since the DSM-V is more likely to diagnose SUD than the DSM-IV, as a consequence of the fact that it adds four new diagnostic criteria to the list of dependence and abuse criteria from DSM–IV and lowers the threshold for diagnosis from three to two symptoms. Indeed, some have argued that the YFAS is over-inclusive because of these high rates of positivity, but rates of ED are almost 20%, too. To narrow the range of positives, one could always use the severity thresholding (e.g., only clinically refer people with moderate or severe FA for treatment) in the case of limited resources, for example [9, 34]. Finally, FA as assessed by the YFAS 2.0 differed by weight class, in that obese participants had a higher prevalence of food addiction (24.6%) than overweight (16.7%) or normal weight (7.8%) participants, an improvement on the convergent validity from the original YFAS. Otherwise both scales were associated with BMI, binge eating frequency, and weight cycling [34].

Further support for convergent validity of the YFAS 2.0, in particular, can be inferred from the fact that scores on the YFAS 2.0 and other measures relevant to problematic eating behaviors were associated with several other important clinical items (disinhibition and impulsive eating, hunger, current BMI, highest lifetime BMI, and frequency of binge eating episodes) and a history of more frequent weight cycling (a weight loss and regain of 20 pounds or more, excluding pregnancy) [34]. Studies supporting the convergent validity of the FA construct continues to build, with each month seeing more publications in the area [3].

Whether FA is simply a reflection of BED, and therefore not providing anything new, is an area of continuing controversy [4, 39, 40] (Chap. 5): if FA is not distinct from BED, it could be argued that it is not a necessary construct [3, 41, 42] because it simply captures a more severe presentation of BED or does not add meaningfully to BED [2]. Indeed these arguments are supported by the fact that there is phenomenological overlap between the behaviors seen in BED and FA (e.g., excessive consumption of food, loss of control, continued use despite negative consequences post-binge distress, binges involved consumption of high fat, high sugar, highly processed foods, etc.) [34], that FA scores correlate highly with measures of binge eating and diagnoses of BN and BED [1, 2, 39, 41, 43, 44], and that there are high rates of FA in ED populations, as high as 100% in some studies of BN patients and 77% in BED [1, 45, 46] (Chap. 12).

However, despite these concerns, in the aforementioned validation papers, the YFAS scales show good discriminant and incremental validity, supporting arguments on the other side: that FA is distinguishable enough from already existing constructs like BED to provide something conceptually and even possibly clinically useful [9, 34]. Of note, the participants in the YFAS validation studies were drawn from a community, non-treatment-seeking population, instead of from a treatment-seeking sample, and for them, less than half of participants with an ED diagnosis met criteria for FA, and about half of participants with a FA diagnosis did not meet criteria for an ED diagnosis [34]. Only 42% of participants with BN and 47% of participants with BED also met criteria for a FA diagnosis, and 44% of participants with a FA diagnosis did not meet criteria for AN, BN, or BED [34]. In another study, 19% of overweight and obese participants, some of whom did engage in regular binge eating, were classified as food addicted according to the YFAS, but most of these did not meet criteria for BED [4, 47]. Therefore, it is likely that although EDs associated with binge eating and FA con-

structs overlap quite a bit in some populations, they are distinct entities, and degree of their overlap depends on the population in which it is measured [3, 48, 49].

Other evidence to support the discriminant validity of the FA measuring scales and which indicate that FA is a separate entity than BED/BN relate to the fact that patients differ slightly in their phenotypic presentations [45, 49, 50]. For example, grazing is reported by individuals with FA [45, 51], and bingeing is not required to meet diagnostic criteria for FA, whereas it is a criteria for BN and BED. Indeed, post bariatric surgery, one can meet criteria for FA, but it is physically difficult or impossible to binge (at least initially) [45, 51].

Finally, in discussions of YFAS discriminant validity, and the validity of the FA construct in general, it's important to mention some expert's concerns that the FA construct might simply be a byproduct of over-restricting [43]. Recall that in ED treatment communities, restrictive behavior is widely considered an important causal factor and contributor to ED behavior, in that it correlates with restriction in ED treatment populations [52]. However, that scores on the YFAS 2.0 and dietary restraint (i.e., the intention to restrict food for weight loss purposes) were *not* correlated and that dietary restraint was also not associated with FA scores on the original YFAS [53, 54] indicate that, at least in community populations, restrictive behavior is unlikely to be the sole contributor to the FA syndrome. Still, screening for a history of caloric restriction behaviors is essential in clinical settings, because a history of restriction could create a risk for false-positives in FA diagnoses and/or a negative outcome with abstinence-based approaches in food-plan development such as rebound bingeing [43] (Chaps. 12, 13, and 14).

6.3 SUD and Disordered Eating Co-occur

That SUD and some overeating problems tend to co-occur in the same people further supports the validity of the FA construct [3]. That said, SUD and obesity travel together less than SUD and BED/BN do, however [55]. Therefore, addictive models of overeating may apply more frequently to those with EDs associated with binge eating than obesity.

High rates of SUD in ED populations have been documented by numerous studies in teens and adults [39, 42, 56–58]; in one study, 50% of women affected with an ED were found to have a comorbid substance abuse or dependence diagnosis [58, 59]. Moreover, higher rates SUD are seen in people with binge and binge-purge ED, with one meta-analysis showing rates of alcohol use disorder (AUD) at 20%, more than 1.5 times higher than controls [60], and another one showing rates of SUD at 22% [43, 61]. Comparing the different ED diagnoses, BN has the strongest correlation with SUD, followed by BED, but rates of SUD in anorexia nervosa are almost the same as that of normal controls [58]. People with SUD also have higher ED rates. For example, one study showed that women with comorbid AUD and nicotine use disorder (NUD) report a higher prevalence of ED symptoms and ED than women with AUD or NUD only, who in turn had a higher prevalence than those without SUD [43, 62].

Individuals with elevated substance use and SUD also have higher rates of FA and vice versa [63]. These relationships have been documented in a large Dutch adolescent sample [43, 56, 58, 64], in men with heroin use disorder who had triple the odds of meeting criteria for BED or FA compared to controls [43, 65], in a large Italian sample of substance-using patients where the overall prevalence of FA was 20% (the rates were highest among cannabis users (31%) and the lowest among tobacco users (11%), and FA risk increased with the number of substances used) [43, 58], and among alcohol abusers (35% had FA, as opposed to 3% in the general population) [58, 59].

Rates of FA are also higher in people with behavioral addictions and general addictive personality scores. Food cravings were found to be higher in people with addictive personalities in a study of bariatric surgery candidates with BED, explaining a significant amount of the variance in cravings [3, 19]. Another study

showed that addictive phone use in adolescents was associated with overeating behavior and higher BMI [66]. Finally, a higher prevalence of FA has been demonstrated among gamblers [58, 67] and people with exercise dependence [58, 68].

6.4 Sweet Preference, Addiction Transfer, and Cross-Sensitization

People with AUD and other SUD also have higher rates of sweet preference compared to normal controls, arguing for shared neurobiological underpinnings [69–73]. For example, heroin users reportedly seek convenient, sweet foods, and eat more sporadically with a more binge-like pattern [73, 74]. Furthermore, subjects on methadone often report high cravings for sugar [69, 73, 75], with reports that a third of their calorie intake is from sugar [73, 76]. Research in cocaine use disorder (CUD) is mixed with some data showing a preference for high fat and carbohydrate-rich foods, but not sugary foods [73, 77], and others reporting preferences for sweet taste [72, 73].

Sweet preference may also be linked to both FA and SUD via propensities for depression or impulsivity [73, 78, 79]. For example, young children who had both a family history of AUD and self-reported depressive symptoms showed the strongest preference for sweetness [73, 80]. Furthermore, a preference for sweetness combined with a novelty-seeking (impulsive) personality markedly increased the risk of suffering from AUD [73, 81].

Genetic factors may contribute to this overlap as well, and genetic factors may explain as much as 50% of the variation in both sugar consumption and substance consumption suggesting similar biological underpinnings [73]. In AUD in particular, men with a genetic link to AUD had a greater sweet preference than men without that link [73, 82].

Reasons for higher rates of sweet preference in SUD may also result from the effects of substances on the brain, as indicated by the fact that craving and sweet preference changes depending on where patients are in their recovery. In AUD, cravings for chocolate increased significantly in the month following alcohol cessation [73]. However, the sweet preference among subjects with AUD may decline following longer periods of abstinence [70, 73].

Some have posited that increased sweet cravings in SUD during recovery may be due to primed reward pathways from chronic substance use and the increased craving and motivational value of food during states of withdrawal [73, 83]. Relatedly, many animal studies also show that consuming sugar [73, 84, 85] and "bingeing" on fat-rich foods can alleviate opiate withdrawal in rats [73, 83]. The nutritional depletion seen in SUD is another important contributor [73, 86–88], since deficits in certain micronutrients and hunger or food deprivation can further prime reward pathways [73, 89]. Indeed, previous work has shown that AUD given nutrition counselling, who may have chosen to eat more food or at least more regularly, had less alcohol craving and more periods of abstinence [73].

Addiction transfer refers to the phenomena that when some people become abstinent from one substance of abuse, they switch to a new substance and can rapidly develop compulsive use of that new substance. Addiction transfer from drugs of abuse to HP food occurs in humans. For example, in a large longitudinal study from Australia, illicit substance users had significant risk of developing recurrent binge eating in addition to, or in place of, their substance use [43, 90]. Anecdotally, many individuals early in recovery from SUD report increased cravings for food, both sweet and fat [73], which can then segue into disordered eating behavior and binge eating, studies show [3, 58].

There is less evidence that obese individuals or individuals with BED in recovery switch to substances of abuse than there is in humans that people in recovery from substances switch to overuse of food [58]. For example, recurrent binge eating was not found to predict later substance use in a large longitudinal study in Australia [43, 90]. However, certain subgroups of bariatric surgery patients might be at risk of

addiction transfer to drugs or alcohol [43]. For example, patients who have lost greater weight after the Roux-en-Y gastric bypass procedure have been found to be at enhanced risk of SUD [73, 91]. Furthermore, increases in alcohol intake have been reported in a group of patients who also had reductions in FA scores after weight loss surgery [58, 92]. Finally, those who report more problems with high glycemic index and high-sugar/low-fat foods before surgery were more likely to develop a new SUD post-surgery, indicating a possible subgroup of BA surgery candidates that had higher FA tendencies [3, 93]. Not all studies have shown this to happen post-surgery, however [94], indicating that addiction transfer may be a phenomena that occurs only in particular subgroups of people who struggle with overeating.

Addiction transfer could be secondary to shared reward and motivational pathways involving the opioidergic system, in particular [73] (Chap. 8). This is supported by work showing that sweet preference increases with exposure to opioids, that opiate antagonists decrease sweet preference, but that opiate agonist use acutely reduces cravings in heroin users [73, 76]. Further support comes from studies showing that individuals with AUD who initially have a greater liking for sweetness respond more robustly from the standpoint of drinking reduction when they take opioid antagonists such as naltrexone (commonly used as a relapse prevention treatment for AUD) [73, 95].

The dopamine system also plays an important role in addiction transfer, as evidenced by studies showing that cross-sensitization (cross-sensitization is a dopaminergic process and measures the ability of drug or food use to prime excitatory pathways for facilitating rapid development of addictive behavior around another substance) can occur between drugs and food, such that exposure to a drug will make an animal more sensitive to food and vice versa. Indeed, numerous studies have shown that sugar-binging rats or rats sensitized to palatable food such as high fat food or western diet reliably increase behavioral and locomotor responses to cocaine, amphetamine, and opioids and drug-associated cues or contexts [3, 9, 73, 96–99]. These studies have lead experts to propose a Gateway Theory of food, wherein overuse of palatable food should be examined as a vulnerability factor that might increase the later risk of SUD development [96].

6.5 Overlapping Neuropsychological, Emotional, and Personality Traits, Psychiatric Diagnoses, and Predisposing Conditions (Trauma and Stress)

SUDs are associated with numerous personality and neuropsychological traits or tendencies and psychiatric symptoms that are likely both made worse by excessive substance use and contribute to SUD development and maintenance and the vicious cycle and downward spiral of addiction [1, 73, 100–103]. Neuropsychological traits of note include heightened reward sensitivity, cognitive bias, impulsivity and executive dysfunction, negative urgency, proclivity towards negative affect (including emotion regulation difficulties), and alexithymia [1, 43, 60, 102–108] (Box 6.2).

Box 6.2 Neuropsychological and Emotional Traits and Tendencies Associated with SUD and Disordered Eating [1, 43, 100–102, 106–110]

- Reward sensitivity: the degree of subjective response to positive stimuli (e.g., food or drugs), also associated with sensation- and novelty-seeking
- Cognitive bias (e.g., attentional, approach, and affective bias): the tendency to unconsciously attend to and approach cues such as smell, sights, sounds, or environments associated with past and potential future experiences. Underlies drug, food, or aversive cue-elicited craving.

- Impulse or cognitive/executive control (impulsivity often refers to self-report scale scores and cognitive/executive control often refers to task performance, but terms are often used interchangeably): the ability to stay on task in the face of distractors, inhibit habitual responses, and make adaptive decisions. There are several subcategories/contributors:
  - Response inhibition (the ability to inhibiting a prepotent response like on a Go/No-go task)
  - Delay discounting (the ability to delay gratification)
  - Self-reported impulsivity (as assessed with the Barratt Impulsiveness Scale or through tests of risky decision-making)
  - Interference control (the ability to stay on task in the context of a distractor and suppress influence of irrelevant information like on a Stroop-like task depends on attention)
  - Working memory (the ability to hold information in memory in order to perform a task)
  - Negative urgency (the tendency to act rashly and impulsively when experiencing strong negative emotions, dependent on both impulse control and negative mood proclivity)
- Negative mood proclivity: Higher proclivity towards irritability, depression, anxiety, etc. There are several subcategories/contributors:
  - Emotion dysregulation [related to alexithymia (difficulty identifying one's emotions) and struggles with acceptance of what is and reframing)]
  - Heightened stress reactivity
  - Withdrawal intensity
  - Underlying psychiatric diagnosis

In terms of personality traits, high neuroticism, low conscientiousness, low agreeableness, high extraversion, high harm avoidance, and low self-directedness are associated with SUD [12]. Relatedly, posttraumatic stress disorder (PTSD) and depressive and anxiety disorders both result from and are known to increase risk of development of SUD and cause overuse of substances of abuse and impede recovery [43, 102, 111, 112]. Furthermore, attention deficit hyperactivity disorder (ADHD) can result from and fuel and worsen SUD, not surprising given the role of impulsivity in loss of control [43, 113, 114]. Finally, a history maltreatment or trauma in childhood, trauma in general, chronic stress states, and chronic uncontrollable feelings of stress increases SUD risk and severity [43, 102].

There is a growing and large body of literature to indicate that these same traits and diagnoses are seen at higher rates in some groups of obese individuals and in people with EDs and FA, such that they both result from or are made worse by and contribute to disordered eating. Likewise, a history of trauma and stress increase ED and obesity risk and severity [43, 63, 115].

From the standpoint of traits, reward sensitivity has been found to be positively associated with disordered eating in several studies and is associated with higher BMI and levels of food craving as well as preferences for foods high in fat and sugar [1, 116, 117]. If reward sensitivity is combined with poor impulse control (labeled the "hot-cold empathy gap" by some experts), then this can create an especially challenging situation for people who overeat [116, 118]. In terms of associations with FA scores in particular, reward eating and sensitivity to food cues are also associated with FA scores and diagnoses [12, 119, 120].

Attentional and approach bias to reward-related food cues also likely drive disordered eating [1, 4, 121], with higher attentional bias to food cues seen in unsuccessful restrained eaters, those with higher trait food craving, people with disordered eating patterns, and in those who are overweight or obese [1, 6, 110]. In terms of FA in particular, higher attentional bias for food cues has been found to be associated with higher FA

scores or FA diagnoses in several studies [1, 4, 34, 46, 57, 116, 118, 121], and sad mood induction can increase this bias [16].

Impulsivity and impaired cognitive control have been identified as key shared mechanisms underlying both binge eating and addictive disorders [43, 109, 122]. Impulsivity is believed to be one of the most important and consistently identified traits contributing to disordered eating, obesity, and overweight as well as is the tendency to make poorer food choices, lack of physical activity, and greater likelihood of snacking on high fat foods [1, 3, 12, 16, 34, 43, 46, 57, 109, 117, 122–132]. Impulsivity levels often distinguish eating disordered populations, especially BED and BN, from non-eating disordered patients [43, 129, 130]. Higher scores on the Barratt Impulsiveness Scale (BIS), greater delay discounting, and impaired response inhibition are associated with higher rates of compulsive overeating, eating disorder psychopathology, and likelihood to engage in poor health behaviors [1, 11, 116, 118, 124, 133]. One study showed that reward sensitivity was associated with greater food intake but that impaired response inhibition was associated with being overweight [1], indicating that reward sensitivity may cause overeating, but inhibitory control impairment may maintain it. Higher FA scores and FA diagnoses are associated with higher levels of impulsivity, as well [1, 2, 12, 16, 34, 43, 57, 109, 124, 126–129, 131, 134], as measured with the BIS [109], tests of delay discounting [1, 11, 116, 118, 124, 126, 127], response inhibition tasks [1, 16, 124, 126, 127, 135], and other tasks of cognitive control [3, 103]. Studies show that FA may be a mediator between impulsivity and obesity, such that impulsivity leads to FA which then leads to obesity [43, 124]. On the other hand, not all studies have found associations between FA and all forms of impulsivity and executive or cognitive control [16, 109], with some reporting a stronger association between FA and self-report measures (like the BIS) than with task performance (like on a response inhibition task) [109].

Negative urgency, which depends on impulsivity and proclivity towards negative affect, is also commonly measured at higher levels in BED [60, 136], obesity [137], and other forms of disordered eating [12, 129, 130]. Negative urgency and lack of perseverance were shown to be strongly associated with FA, and tests of mediation indicated that, like with impulsivity in general, FA was a mediator of the effect of negative urgency on BMI [12]. Deficiencies in related areas such as the aforementioned hot-cold empathy gap [118] and intolerance of uncertainty [138] are increasingly recognized as important contributors to binge eating and loss of control around food. Finally, an important longitudinal study, important because it demonstrates causality more than a cross-sectional associative study, showed that negative urgency predicted increases in expectancies for reinforcement from eating which in turn predicted increases in binge eating behavior over several years [139]. In terms of FA in particular, individuals with high levels of negative urgency have been found to be at greater risk of later developing addictive eating patterns [60, 129, 130] and have higher levels of FA [12, 109].

A tendency towards irritability, anxiety, and depressed mood, made worse by heightened stress reactivity, and difficulty regulating emotions, is also related to and believed to cause and make worse binge eating-based eating disorders and obesity [2, 12, 53, 54, 125, 140, 141]. High psychological distress has been found to directly contribute to higher BMI by its effect on eating disorder attitudes, FA, and insomnia [142, 143]. Several studies using ecological momentary assessment, which is a method that allows for moment-to-moment tracking of behaviors and emotional states to get more at causality, identify negative affect as important predictors of binge eating behavior [141, 144]. Another longer longitudinal study confirmed the importance of negative affect as a contributor to eating disordered behavior, showing that negative affect predicted increases in thinness expectancies, which in turn predicted increases in purging several years later [139]. Socio-evaluative stress may play a particularly important role in BED [145]. Alexithymia, or difficulty identifying one's emotions, is also seen at higher rates in people with EDs [2, 12, 53, 54, 141, 143, 146]. Higher scores on the YFAS

and FA-positive diagnoses are also associated with heightened negative affect due to low self-esteem (feeling ashamed or critical of oneself, upset, or worried due to one's eating habits) [103], higher levels of emotional eating [147], emotion dysregulation and alexithymia [2, 12, 53, 54, 141, 143, 146], and more frequent irrational beliefs, the latter of which were in turn related to higher depression and anxiety and emotional eating [148].

Finally, in terms of personality traits, mirroring much of what is seen in SUD populations, high extraversion, high neuroticism, high harm avoidance, low self-directedness, high novelty seeking, and low agreeableness have been found to be associated with FA and ED diagnoses [12, 129, 130, 146, 149].

As with SUD, obesity and EDs are associated with higher rates of psychiatric disorders and symptoms, and evidence suggests these associated psychiatric diagnoses cause heightened obesity and eating disorder severity and persistence. These associations have been seen for depression [137, 150, 151], anxiety [16, 43, 47, 103, 104, 134, 149, 150, 152–154], and social anxiety [43]. Rates of PTSD are also higher in ED populations, and higher rates of ED are seen in patients with PTSD [43]. Depression in particular may contribute to greater levels of shame and lower self-esteem people with obesity and disordered eating [103], as well as poorer quality of life [16], all of which could contribute to further disordered eating behavior and weight gain. Depression, anxiety, and PTSD can fuel overeating via self-soothing which is consistent with reports of "comfort food" consumption when under stress [43, 155]. Higher FA scores are also correlated with higher depression scores and a higher likelihood of an MDD diagnosis [2, 12, 16, 43, 137, 147, 150, 152, 156] as well as higher levels of anxiety and anxiety sensitivity [12, 43, 48, 149, 152–154, 157] and higher levels of PTSD symptomatology [43, 158]. In a study of surgery patients 6 month post-op complaining of loss of control eating, individuals with high FA scores also had high depression levels [147, 154, 159]. Irrational beliefs may be one source of the anxiety associated with FA [43, 148].

ADHD is also highly related to disordered eating, as has been discussed in several excellent systematic reviews [160–162]. Notably, these and other studies show strong associations between bingeing diagnoses (BED BN) and overeating behavior with ADHD in both childhood and adult populations [43, 160, 161, 163]. Restrictive behavior and anorexia nervosa, by contrast, are not highly correlated with ADHD [160, 161]. ADHD is posited to contribute both to higher eating disorder severity and treatment dropout [43, 164] via increased impulsivity, increased negative affect, or both [162]. Obesity is also associated with ADHD diagnoses and ADHD-related executive dysfunctions [151, 160], although, unlike in eating disorders, a large genome-wide association study suggests that higher BMI increases risk of developing ADHD but not the other way around [43, 165]. It is worth noting that stimulant medications (i.e., amphetamines) often used in the treatment of ADHD can also suppress appetite and are used in the treatment of both BED and obesity [43] (Chaps. 2 and 3). ADHD and FA, proper, have been found to be associated with one another as well [43, 163].

Like in SUD, there is a clear and positive association between adiposity, elevated BMI, and weight gain and a history of uncontrollable stressful events and chronic stress states such as job strain, unemployment, family caregiving, marital conflicts, and poverty [102]. The relationship between stress and weight is strongest among individuals who binge eat. Stress and adversity increase binge consumption of fast food snacks and calorie-dense and highly palatable foods, and stress-driven eating is more severe in obese women compared to lean women [102]. Perceived stress, per a self-report scale, is also correlated with BMI [166].

Like what is seen in SUD, childhood trauma and childhood maltreatment increase the risk of development of obesity [43, 158, 167–170] even after controlling for confounders [171], although in some cases studies show the link occurs via increased impulsivity [43, 170, 172] especially in the binge forms (BN, BED, binge eating without ED criteria) [43]. In a related area of research, higher attachment insecurity – as defined by

failure to form trusting and reliable relationships with others and which often results from childhood trauma and poor parenting or at least poor child-parent emotional match – systematically characterized individuals with EDs and unhealthy eating behaviors from those without, in the general population [173].

Both chronic stress and childhood trauma also relate to FA scores and diagnoses. For example, self-reported perceived stress has been found to be associated with FA, and FA significantly mediated the relationship between perceived stress and BMI [166]. Childhood trauma and maltreatment, early life adversity, and psychological and sexual abuse have also been highlighted by several authors as a likely precursor to FA as well [12, 43, 48, 153, 154, 157, 158, 167–169, 171, 174] with childhood physical abuse and childhood sexual abuse increasing risk for FA by as much as 90% [43, 167]. Highlighting the importance of childhood, earlier onset of PTSD predicts a stronger association between PTSD and FA [43, 158].

6.6 Conclusion

In summary, there is a rapidly ballooning body of clinical evidence indicating that people can develop an "addictive" relationship to certain foods and that the FA construct is likely a valid one.

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