

## **The Binge and the Brain**

By Alice V. Ely, Ph.D., and Anne Cusack, Psy.D.

*Editor's Note: Who hasn't dipped into that pint of Häagen-Dazs with the best of intentions and ended up finishing the entire container? Knowing where the line is when it comes to out-of-control impulse consumption is at the heart of binge-eating disorder (BED), a newly recognized mental condition that affects millions of people and that we are just beginning to better understand—from both a neurobiological and clinical standpoint.*

The word “binge” is used casually to describe a meal that was larger than intended, or even a long evening of Netflix. But genuine binge eating looks very different from Thanksgiving dinner in which we go back for seconds on mashed potatoes, or a Super Bowl party where we devour an abundance of chips and dip. Genuine binge eating is recurrent and debilitating—physically and emotionally. Its medical consequences include high blood pressure, type 2 diabetes, high cholesterol, gallbladder disease, digestive problems, heart disease, and metabolic syndrome. It is frequently accompanied by anxiety and depression too, though these, like the binge-eating behavior itself, are relatively hidden.

Research suggests that more than 20 percent of college-aged women have engaged in binge-eating behavior, and that a similar percentage of the broader population will be affected by binge-eating disorder (BED) at some point in their lives.<sup>1</sup> Currently, about sixty percent of diagnosed cases are in women, and BED researchers believe that fewer than half of all cases receive adequate treatment. Indeed, research into the neural mechanisms of BED is still in its infancy, so there is no specific treatment for the disorder.

### **What Makes a Binge a Binge?**

BED was added to the Diagnostic and Statistical Manual of Mental Disorders only in the 2013 edition (DSM-5).<sup>2</sup> However, similar patterns of signs and symptoms have been reported in the medical literature since 1932, when German psychoanalyst Moshe Wulff described an eating disorder characterized by binge eating, depression, and disgust with one’s body.<sup>3</sup> DSM-5 criteria for a BED diagnosis include at least one binge episode per week without any compensatory purging behavior (which would then lead to a bulimia nervosa diagnosis). Binges must involve an objectively large amount of food, consumed within two hours, and accompanied by a feeling of loss of control.

Loss of control is what distinguishes a binge from simple overeating, and similar constructs exist in substance use disorders. Loss of control can take many forms, but there are commonalities in patients included in the DSM-5 that are necessary criteria for a BED diagnosis: being unable to stop consuming food despite a strong desire to stop, a feeling of fullness, eating alone because of being embarrassed about how much one is eating, or even a sense that the food’s taste is no longer appealing. Some also report eating much more rapidly than normal.

BED patients frequently report that their binge foods are foods that they view as forbidden or unhealthy. One of Wulff's patients, for example, described secretly eating large quantities of sweets, bread, and pastries because they were restricted from her daily diet due to medical obesity. "The worse [for me], the better," she explained.

In severe binges, the drive for overindulgence may cause patients to consume raw pancake batter, entire loaves of bread, frozen fish sticks, or other bulk foods, however lacking in taste they may be. The patient often plans secret eating in advance, and carries it out late in the day—and typically experiences disgust, depression, and guilt afterwards.

A patient in our clinic serves as an illustrative example. B, a 57-year-old African-American woman, reported significant struggles with food prior to entering treatment, and stated that she often thought about eating all day long. B said that she visited the grocery store several times a week, ate directly from bags or containers, and often consumed strange combinations of flavors, such as licorice, bread, peanuts, and beef jerky. Her remark that she "didn't even want those foods but couldn't stop eating them" pointed to a loss of control. B also reported frequently eating despite not being hungry, in order to fill a "void." She has tried to combat the resulting significant weight gain through a variety of fad diets, "wellness programs," and gastric bypass surgery. This has led to extreme weight fluctuations over 25 years, including a recent episode in which she lost 75 pounds on a crash diet. However, failing to address the psychopathology underlying her bingeing, she gained all that weight back.

People who meet the criteria for BED are more likely to be in the obese or overweight ranges, and to report struggling with their weight as children. Compared to healthy controls, those diagnosed with BED reported more frequent family histories of depression, greater vulnerability to obesity, more exposure to negative comments about shape or weight, greater perfectionism, and higher negative self-evaluation. Compared to obese individuals who do not binge, those diagnosed with BED reported greater weight and shape concerns, more personality disturbance, more mood/anxiety disorders, and a lower overall quality of life.

These factors highlight the importance of not treating obesity as a single clinical problem. If we treat BED patients only with weight loss interventions, we won't target the actual problem, as B's case suggests. While BED may occur in normal weight individuals, the patient in such cases is typically in the early stages of the disorder. For early BED patients, prompt intervention may stop the binge-eating behavior before medical and psychological consequences become irreversibly severe.

Research also suggests that patients binge-eat to distract themselves from uncomfortable feelings. Indeed, binge-eating is often viewed as emotion-driven eating, done in response to anxiety, depression, and/or boredom.<sup>4</sup> This has certainly been the case for B, who reports difficulty connecting to others since childhood as well as identifying and allowing herself to experience emotions.

### **Lessons from Neurobiology**

What might happen in the brain to drive this kind of extreme eating behavior? Even in healthy individuals, eating is a complex process, involving not just physiological but also psychological and emotional processes. We eat not just because we need sustenance but also because food tastes good, we're with friends who are also eating, and/or we're bored and food is available.

Three neural pathways interact to drive eating behavior: one codes for the perceived salience, or importance, of a food stimulus; another codes for the rewarding sensation of actual eating; and the third helps us control our consumption based on considerations of both short and long-term outcomes, such as weight gain.

Basic sensory information about food is processed by a brain region called the insula, along with the frontal operculum. The insula also plays a role in networks that determine salience. To identify and evaluate the rewarding properties of the food, closely connected brain regions collectively called the ventral striatum, including the nucleus accumbens and related regions, such as the putamen, and caudate, are called into action. Finally, the circuitry that helps us control our responses to food includes the dorsal caudate and dorsal ACC, ventrolateral PFC, and parietal cortex.<sup>5</sup> Together, these

pathways combine, in effect, to weigh the salience of a stimulus, its reward value, and the longer-term consequences of consuming it, and thereby determine eating behavior.

What's different about these pathways in BED? Generally, overeating is attributed to an elevated experience of reward, a reduced ability to inhibit the drive to eat, or some combination of the two. Typically, studies of these pathways in BED patients compare the latter to weight-matched controls as well as lean participants. While some of the research is conflicting, overall we see decreased inhibitory control and increased sensitivity to reward.

If we start to parse out different types of self-control, BED is associated with a range of deficits. Individuals with BED tend to do worse on tasks related to motor inhibition and attention, and these deficits seem to be related to binge eating severity rather than obesity.<sup>6</sup> While it's unclear whether increasingly severe binge eating leads to inhibitory control problems or vice versa, these impairments certainly help maintain the disordered eating. If individuals are more susceptible to impulsive decision-making, their vulnerability to binge-eating increases, and breaking the cycle of binge eating becomes more difficult.

However, we can't just tell BED patients that they need to make better choices and exert more willpower, given the differences in the neurobiology that underlie inhibition. Compared to both lean and weight-matched control participants, BED patients appear to have abnormally low activity in impulse-control related frontal brain regions. A recent neuroimaging study asked BED participants to complete a task in which they had to resist reading a word in favor of naming the color of the word's printed type—a classic test of inhibitory control.<sup>7</sup> The study showed reduced activation in the ventrolateral PFC, inferior frontal gyrus, and the insula. Further, the more impairment that BED participants had in recruiting those neural pathways involved in self-control, the worse they were at dietary restraint.

Studies of the relationship of reward sensitivity to BED are more numerous, and typically point to an elevation in the brain response to both food and non-food reinforcement. Functional magnetic resonance imaging (fMRI) research has shown links between aberrant brain activation and behavioral tasks of delaying gratification as well as self-report measures of sensitivity to reward.

Some fMRI studies have lumped BED patients with non-bingeing obese subjects, and unsurprisingly have reported inconsistent findings. Overall, findings have tended to indicate increased reward sensitivity, particularly just after having eaten. Those studies that have looked at more pure samples of BED patients have found exaggerated responses in the orbitofrontal cortex to food reward compared to obese or lean controls.<sup>8</sup>

If we distinguish between viewing images of rewarding things, and anticipating or receiving actual rewarding things, the findings again tend to vary. During the anticipation of monetary rewards, for example, BED subjects show less activity in the ventral striatum compared to obese non-bingeing subjects—though they show no significant difference on this measure from lean control subjects.<sup>9</sup> Wang and colleagues<sup>10</sup> have demonstrated, using positron electron tomography (PET), that seeing, smelling, and tasting a food (but not eating it) significantly increases dopamine in the caudate in BED participants. Dopamine is a neurotransmitter that helps mediate the motivation to eat, and in the dorsal striatum is associated with habit learning, in which behavior becomes automatic and no longer necessarily linked to pleasurable outcomes. The Wang study linked higher caudate dopamine levels to binge eating severity rather than to weight, supporting the idea that separate brain circuits underlie eating pathology and the development of obesity.

Considering the two prior findings, in the context of the relevant literature, we could speculate that participants do not fully anticipate the effect that eating will have on their psyche, eating only out of habit. Thus, they become overwhelmed upon receiving the food, potentially driving the loss of control that seems central to binge eating. While the research in BED is still limited, similar results suggesting a blunted anticipatory response to reward has also been seen in substance abuse.

So how do these neurobiological differences manifest themselves in personality? Do characteristic temperament traits exist prior to the development of binge-eating behavior? Is there any way to predict the development of the disorder? A recent meta-analysis suggests that BED patients may demonstrate slightly elevated harm-avoidance (a measure of anxiety, inhibition and inflexibility) and potentially novelty-seeking (a facet of impulsivity).<sup>11</sup> Other studies have shown that impulsivity—specifically negative urgency, or the tendency to act impulsively when distressed—may be related to the emergence of binge eating.<sup>12</sup> This seems consistent with the emotional

dysregulation that is typically reported in this population—dysregulation that binge eating may be aimed at correcting. Overall, these two findings suggest that some level of impulsivity or harm avoidance may predispose some people to binge-eat. These temperament traits also have been linked to anorexia nervosa and bulimia nervosa.

### **Binge Eating Treatment**

There are a number of empirically based treatment approaches for BED that share the goal of reducing binge-eating episodes and the accompanying psychopathology. The gold standard is cognitive-behavioral therapy (CBT), which targets maladaptive eating behaviors through a combination of self-monitoring, normalizing eating patterns, and building behavioral and cognitive coping skills.

Cognitive strategies involve identifying and restructuring cognitive distortions that help maintain binge-eating behaviors. For instance, the thought that “I’ve already broken my diet with this slice of pizza, so I might as well have the whole pie” is an example of “all or nothing thinking,” and can be reframed as, “I may have had a slice of pizza, but I can still eat healthily the rest of the day.” CBT also targets maintenance of normal eating behavior and relapse prevention.

However, some research indicates that only 50 percent of BED patients who complete CBT treatment experience lasting improvement, and an estimated 33.3 percent of patients continue to meet criteria for eating disorders five years after beginning therapy.<sup>13</sup> These are dismal numbers, but there is evidence that a longer course of treatment may show better results.<sup>14</sup>

Given that binge eating is widely seen as a means of managing unwanted emotions, some treatments have targeted patients’ emotional regulation abilities. These treatments also have shown promise. For example, dialectic-behavior therapy (DBT) is a cognitive-behavioral treatment approach, originally developed for borderline personality disorder that focuses on mindful awareness and tolerance of emotion, building coping skills, and interpersonal effectiveness. In studies of DBT’s effectiveness, most participants significantly decreased their binge-eating behavior.<sup>15</sup> While differences compared to a control group that received nonspecific treatments

were not significant after follow-up, an additional study of DBT-based guided self-help suggests that persistence of binge-eating abstinence is influenced by changes in emotional regulation.<sup>16</sup>

Pharmacological treatments that target brain chemistry directly have shown mixed success. A meta-analysis of pharmacotherapy for BED suggests a medium effect of antidepressant use for the reduction of binge-eating behavior.<sup>17</sup> Studies suggest that combining pharmacotherapy with CBT or interpersonal therapy (IPT) does result in a significantly improved response rate, but combination treatments may not be any better than psychotherapy alone. In general, for prospective BED drug therapies; there has been a relative lack of controlled clinical trials in large samples of patients. Lisdexamfetamine (Vyvanse), an appetite suppressing amphetamine, is currently the only medication that is FDA-approved specifically for treating BED. Like other stimulants, lisdexamfetamine works by increasing extracellular dopamine and norepinephrine, and while the mechanism of action is not definitively known, it is possible that increasing synaptic dopamine in the striatum may weaken or reduce the characteristic under-response to anticipation of palatable food seen in BED. Clinical trial results indicate that it leads to the cessation of binge-eating behavior in up to 50 percent of treated subjects.<sup>18</sup> However, there are significant side effects, experienced by over 80 percent of participants on the medication, and there is a significant risk of abuse and dependence. Furthermore, it's unclear that lisdexamfetamine works for people who have depression or other medical issues related to obesity—as most BED patients do. Psychological and behavioral treatments are necessary to fully address the complex psychological and medical syndrome associated with BED.

If pharmacotherapy is not a magic bullet, how can we improve cognitive behavioral treatment to target the neurobiology? Reducing the rewarding properties of food is one possible approach. It may be difficult, but preliminary research in obesity suggests the possibility of habituating patients to food through repeated exposure without actual consumption, thereby reducing cravings.<sup>19</sup> The same research suggests that control over eating behavior may be strengthened by a combination of stimulus control—such as minimizing exposure to binge foods, changing environmental cues for binge eating, and reducing vulnerability factors for binge eating, such as extended periods of time alone—and inhibitory training. Both approaches may help individuals develop the needed skills to resist urges to initiate binges or stop them once they've started.



We might also consider working with the temperament traits that promote binge eating rather than against them. If individuals with BED are more reward-sensitive, then frequent and salient rewards for abstinence during therapy may be more effective than punishments for lapses. Rewarding social experiences that don't involve food may also reduce reliance on food, as well as potentially reducing any underlying depression.

We propose that further advances in BED treatment can come from behavioral interventions that target BED's apparent neurobiological mechanisms. Anecdotally, this strategy appears to be working for B: she has spent four months in a partial hospitalization program using DBT and CBT treatment to advance her learning skills and become more mindful of her emotions, as well as better tolerate distress and improve interpersonal relationships. While she hasn't lost any weight, she has binged only a handful of times. Meanwhile, she's learning to cook for herself and eat regularly, rather than depriving herself or following fad diets. She's also worked significantly on regulating her emotions: "Learning other ways of dealing with emotions improved my life one-hundred percent. If I lose weight because of it, that is a bonus. If not, at least I have my life back."

### **Bios**

**Alice V. Ely, Ph.D.**, is a postdoctoral research fellow in biological psychiatry and neuroscience at the University of California, San Diego's Eating Disorders Center for Treatment and Research. Ely's work has focused on behavioral and neurobiological risk factors for weight and eating disorders, primarily concerning frontostriatal reward processing in response to anticipation and receipt of food reward. Her specific focus is on impulsive decision-making in individuals with bulimia nervosa and the relationship between neurobiological response to food reward and anxiety. She received her undergraduate degree in psychology from the University of Pennsylvania and her Ph.D. in clinical psychology from Drexel University in Philadelphia, following her clinical internship at UCSD and the San Diego VA Health System.

**Anne Cusack, Psy.D.**, is a postdoctoral fellow at the UCSD Eating Disorders Center for Treatment and Research and co-manages the adult treatment program. Cusack's research focus has been on

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