



Research report

A history of the identification of the characteristic eating disturbances of Bulimia Nervosa, Binge Eating Disorder and Anorexia Nervosa [☆]



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ABSTRACT

During the last 25 years, the careful examination of the eating behavior of individuals with eating disorders has provided critical insights into the nature of these disorders. Crucially, studies investigating components of different eating behaviors have documented that Anorexia Nervosa (AN), Bulimia Nervosa (BN), and Binge Eating Disorder (BED) are characterized by objective disturbances in eating patterns that are significantly different than behaviors exhibited by individuals who do not have these eating disorders. The detailed description of the disturbances in eating behavior has helped to identify diagnostic criteria associated with each disorder, and has led to important hypotheses about the underlying pathophysiology. These advances in understanding have provided, and continue to provide, a foundation for translational research and for the development of novel treatment interventions. This review is based on a presentation given by B. Timothy Walsh, M.D. at the 40th anniversary symposium of the Columbia University Appetite talks outlining the evolution of the discovery of the characteristic eating disturbances seen with each disorder.

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Introduction

The eating disorders formally recognized by the American Psychiatric Association and included in the fourth edition of the Diagnostic and Statistical Manual (DSM-IV-TR, 2000) are Anorexia Nervosa and Bulimia Nervosa. A third eating disorder affecting adults, Binge Eating Disorder, is expected to be recognized in the next edition, DSM-5, which will be published in 2013.

Extensive research to identify and classify eating disorders helped define the characteristic eating patterns associated with each disorder. Early studies explored the excitatory and inhibitory controls of meals and how these were affected in those with normal and disordered eating behaviors. An investigation into the

components of different behaviors, as well as the perceived rewards and motivations for them, followed.¹

Bulimia Nervosa (BN)

Bulimia Nervosa (BN) is defined by eating behaviors that include episodes of binge eating which are followed by recurrent inappropriate behaviors (such as self-induced vomiting) performed in an attempt to avoid weight gain from the caloric overload. To satisfy the current DSM-IV-TR (2000) criteria, the behavior must be frequent (on average, at least two times per week for 3 months or longer).

Individuals with BN

How do individuals with Bulimia Nervosa eat?

When BN was first recognized in the early 1980s, self-reports by individuals who said they binged were not fully understood. Self-reports are often unreliable and interpretation can be difficult. Were these self-described binges truly episodes of over-eating? And if so, how much did they overeat? Or was the perception of having “binged” simply a reflection of anxiety over having eaten “too much”? When a person describes how much they eat—or overeat—their estimation is variably subjective: what constitutes a binge for one person (seven cookies, for example) might be viewed as a large snack by someone else. Early work in Bulimia Nervosa set out to define what a “binge” is.

In a pivotal series of studies, 25 individuals with Bulimia Nervosa and 18 non-bulimic controls were asked to binge eat in

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¹ The following review, based on a presentation given by B. Timothy Walsh, M.D. at the 40th anniversary symposium of the Columbia University Appetitive Seminar, summarizes knowledge that was accumulated by researchers affiliated with Columbia University, most in close collaboration with Harry R. Kissileff Ph.D., the long term chair of the Appetitive Seminar. For this reason, descriptions of other recent important avenues of related research, including neuroimaging and genetics, are not discussed.

a laboratory setting so that the behavior could be objectively described. The volunteers were instructed to eat whatever they wished from a multi-item buffet and to replicate, as much as possible, how they might eat during a binge at home. A bathroom was accessible if subjects wished to purge. Among those who were able to binge eat, it was found that individuals with BN consumed significantly more calories in a binge meal (almost 4000 kcal or 16.7 MJ) than did controls (just over 1000 kcal or 4.2 MJ) who were given the same instructions (Walsh et al., 1992).

This bingeing experiment showed that the salient disturbance described by individuals with BN was not simply their subjective perception of overeating, but a clear behavioral abnormality that could be quantified. The quantification of different meal sizes allowed the characterization of a specific behavior associated with BN and showed that it was significantly different from that of individuals without BN, thereby helping to document the validity of the diagnostic criteria.

Another observation from the same study that further elaborated the abnormal eating patterns was the behavior when participants were asked to consume a non-binge meal. Individuals with BN ate significantly less during a “normal” meal than during a binge and, surprisingly, consumed significantly fewer calories than controls (Walsh, Hadigan, Kissileff, & LaChaussée, 1992). The fact that bulimics ate less during non-binge meals revealed that, in BN, the overall eating pattern consisted of not only overeating, but also under-eating, suggesting that the central phenomenon underlying the disordered eating behavior was the dysregulation—in both directions—of the amount of food consumed.

Is there a disturbance in satiety in BN?

Rather than continuing to eat after they feel full, individuals with BN may overeat because they do not feel sufficiently full after consuming a normal amount of food. An influential study showed that when individuals with BN and controls were given a fixed-sized meal and asked to rate subjective satiety afterwards, those with BN reported feeling less full than the controls (Geraciotti & Liddle, 1988). Furthermore, a potential biological mechanism was identified: the release of cholecystokinin (CCK) was diminished after a meal in the BN, but not in the control group. CCK plays an important role in controlling food intake because the post-prandial increase in CCK is an important factor in meal termination. This study demonstrated a potential translational link between a biological control mechanism from the gut and clinical symptoms, suggesting that post-ingestive satiety signals are disturbed in BN. Subsequent studies confirmed the presence of a physiological abnormality where CCK was diminished in BN (Devlin et al., 1997; Keel, Wolfe, Liddle, De Young, & Jimerson, 2007; Pirke et al., 1994).

Do individuals with BN experience gastric dysfunction?

Subsequent studies identified other physiological impairments potentially related to the control of eating behavior. A reduced rate of emptying of the gastric load into the duodenum was shown by several groups (Devlin et al., 1997; Faris et al., 2000). It was also shown that individuals with BN displayed excess capacity, allowing them to ingest greater amounts of food. A pathophysiological model was proposed, depicting a vicious cycle in which people engaged in binge eating and vomiting, leading to biological changes in gastric functioning that then affected satiety, gastric load and aspects of eating. These alterations might then contribute to disturbances in sensing how much had been consumed, thereby setting the stage for continued binge eating and vomiting (Geliebter et al., 1992).

To investigate the link between gastric load and satiety, individuals with BN and controls were asked to binge on a single-item meal and rate how full they were while they were eating. The change in fullness over the course of the meal was similar in the

BN and control groups, but individuals with BN consumed 50% more calories than the controls. Therefore, the change in fullness per unit of food consumed was much lower in the individuals with BN, consistent with a disturbance in the development of satiety (Kissileff et al., 1996).

However, in a subsequent experiment, individuals with BN and controls were asked to consume a liquid meal from an opaque container that automatically re-filled. Participants were instructed to continue to eat until research staff signaled them to stop, which occurred after they had consumed 975 g. Therefore, the quantity of food consumed and the total amount to be provided were unknown to the participants while they ate. Under these conditions, the average ratings of fullness of the individuals with BN during the course of the meal were identical to those of controls (Zimmerli, Devlin, Kissileff, & Walsh, 2010). In contrast to the previous study where study participants determined how much they ate and were aware of the quantity of what they had eaten during the course of the meal, data from this experiment indicated that the change in fullness per unit of food consumed was quite normal, and therefore did not suggest an impairment in the development of satiety. Notably, in the earlier study, subjects were asked specifically to binge eat, suggesting the possibility that a person's decision to overeat then alters their how they interpret subjective sensations and biological signals resulting from food ingestion.

Another study focused on the potential role of delayed gastric emptying. If individuals with BN had slowed emptying and this, in turn, contributed to a decrease in the release of CCK, perhaps binge eating could be reduced by accelerating gastric emptying. A placebo-controlled study using erythromycin, a motilin antagonist, was recently completed. Treatment with erythromycin did, indeed, accelerate gastric emptying, but resulted in neither an increase in CCK release nor a decrease in binge eating (Devlin et al., 2012). These more recent studies, while they have significant limitations, raise doubts about how critical gastric functioning disturbances and peripheral GI signaling are in the persistence of the symptoms of BN.

Binge Eating Disorder (BED)

Binge Eating Disorder (BED) is currently characterized by recurrent episodes of binge eating occurring at least twice a week for 6 months or longer. In contrast to *Bulimia Nervosa*, there are no inappropriate compensatory behaviors performed to counteract the potential weight-gaining effects of bingeing. BED is included in the Appendix of the *DSM-IV-TR (2000)* with identifying criteria noted for further study. The characteristic eating behaviors were first described in 1959 (Stunkard, 1959), but research was greatly stimulated by the identification of tentative diagnostic criteria in *DSM-IV-TR*, and BED is likely to be formally recognized in *DSM-5* in 2013.

Individuals with BED

How do individuals with BED eat?

Several studies have investigated the eating behavior of individuals with BED in laboratory studies and come to remarkably consistent conclusions. When individuals with BED were asked to binge eat in a laboratory setting, they consumed 500–1000 kcal (2.1–4.2 MJ) more than controls (Goldfein, Walsh, LaChaussée, Kissileff, & Devlin, 1993; Guss, Kissileff, Devlin, Zimmerli, & Walsh, 2002; Yanovski et al., 1992). This observation that people with BED consumed more than those people without BED during a binge provided objective evidence that a disturbance in eating behavior exists in BED, supplying a critical piece of the foundation for the official recognition of BED in *DSM-5*.

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