Binge Eating Disorder: An overview and update

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Agenda

• Definition, history, and classification
• Prevalence, distribution, associated features
• Obesity and BED
• Weight stigma and its effects on eating, stigma toward BED
• Mechanisms: Our models based on BN are too simple!
• Treatment
• Take-home points
Went without caffeine once

It was terrible
9 SANHETTER OM SPISEFORSTYRRELSER

- **Sannhet #1**: Mange med spiseforstyrrelser ser friske ut selv om de kan være svært ekstremt syke.
- **Sannhet #2**: Familien skal ikke klandres. De kan være både pasientens og behandlerens viktigste støttespillere.
- **Sannhet #3**: En spiseforstyrrelse er en kritisk helsetilstand som sterkt forstyrer personens og familiens funksjon.
- **Sannhet #4**: Spiseforstyrrelser er ikke et valg, men en alvorlig lidelse som påvirkes av biologiske faktorer.
- **Sannhet #5**: Spiseforstyrrelser kan ramme alle uansett kjønn, alder, etnisk bakgrunn, kroppsform, vekt, seksuell legning og sosioøkonomisk status.
- **Sannhet #6**: Spiseforstyrrelser er forbundet med økt risiko for selvmord og medisinske komplikasjoner.
- **Sannhet #7**: Både arv og miljø spiller viktige roller i utviklingen av spiseforstyrrelser.
- **Sannhet #8**: Gener alene avgjør ikke hvem som utvikler spiseforstyrrelser.
- **Sannhet #9**: Det er mulig å bli helt frisk av spiseforstyrrelser. Tidlig oppdagelse og behandling er viktig.
• «It starts off with my thinking about the food that I deny myself when I am dieting. This soon changes into a strong desire to eat. First of all it is a relief and a comfort to eat, and I feel quite numb and zone out. But then I can’t stop, and I binge. I eat and eat fast and frantically until I am absolutely full. Afterward I feel so guilty and angry with myself.»

It starts off with my thinking about the food that I deny myself when I am dieting. This soon changes into a strong desire to eat. First of all it is a relief and a comfort to eat, and I feel quite numb and zone out. But then I can’t stop, and I binge. I eat and eat fast and frantically until I am absolutely full. Afterward I feel so guilty and angry with myself.

Clues to triggers/maintenance?
- Evidence of psychological and physical deprivation
- Craving
- Cognitive and behavioral restraint
- Dieting behavior may involve unhealthy eating pattern (e.g., fasting) and disruption of satiety and hunger signals
- Abstinence violation effect
- Operant conditioning (R+), escape/numbing

Restraint, escape and affect regulation models all possible here.
a. Recurrent episodes of binge eating: objectively large amount of food (larger than most people would eat under similar circumstances) within a discrete period of time (< 2 hrs) and **feeling a loss of control**.

b. Binge eating episodes are associated with three (or more) of the following:
   1. Eating much more rapidly than normal.
   2. Eating until feeling uncomfortably full.
   3. Eating large amounts when not physically hungry.
   4. Eating alone because of embarrassment.
   5. Feeling disgusted with oneself, depressed, guilty after.

c. Marked distress regarding binge eating is present.

d. At least once a week for 3 months.

e. The binge eating **is not associated** with compensatory bx.
If these symptoms are present and binge eating takes place on average at least once a week for three months, it may be Binge Eating Disorder.
A “new” phenomenon?

- Binge eating disorder is not “new” – described in obesity literature already in 1950’s (Stunkard, 1959)

- Binge eating disorder was diagnosable in DSM-IV only by applying the diagnosis of “EDNOS”

- BED was included in DSM-IV (APA, 1994) as a “Criteria Sets and Axes Provided for Further Study” in Appendix B.
Only 1 study in 1980, over 350 studies in 2015

Only 34 hits for studies conducted in Norway (or with authors affiliated with Norwegian institutions)
In recent years, clinicians and researchers have realized that a significant number of individuals with eating disorders did not fit into the DSM-IV categories of anorexia nervosa and bulimia nervosa. By default, many received a diagnosis of “eating disorder not otherwise specified.” Studies have suggested that a significant portion of individuals in that “not otherwise specified” category may actually have binge eating disorder.

**Binge Eating Disorder**

Binge eating disorder was approved for inclusion in DSM-5 as its own category of eating disorder. In DSM-IV, binge-eating disorder was not recognized as a disorder but rather described in Appendix B: Criteria Sets and Axes Provided for Further Study and was diagnosable using only the catch-all category of “eating disorder not otherwise specified.”

Binge eating disorder is defined as recurring episodes of eating significantly more food in a short period of time than most people would eat under similar circumstances, with episodes marked by feelings of lack of control. Someone with binge eating disorder may eat too quickly, even when he or she is not hungry. The person may have feelings of guilt, embarrassment, or disgust and may binge eat alone to hide the behavior. This disorder is associated with marked distress and occurs, on average, at least once a week over three months.

This change is intended to increase awareness of the substantial differences between binge eating disorder and the common phenomenon of overeating. While overeating is a challenge for many Americans, recurrent binge eating is much less common, far more severe, and is associated with significant physical and psychological problems.
Overeating vs. Binge Eating

No loss of control
• Occurs typically during normal routine (meals, social functions)
• Often others are eating, too
• Occurs in different places/situations
• Across moods, often relaxed or positive mood
• Regular speed of eating
• Awareness
• Can be associated with regret (‘‘wish I hadn’t’’)
• No specific triggers

Loss of control is key—feeling of not being able to stop, compelled to eat
• Done outside of normal meals, private
• Alone
• Often occurs same place
• Associated with negative moods
• Eating much more rapidly
• Lack of awareness/zoning out
• Strong feelings of shame and guilt (feeling of doing something ‘‘wrong’’)
• Triggers (...stress, being alone, bored, hunger, breaking a dietary rule, relationship px, unstructured time, etc.)

Pollart et al., (2013)
What counts as an “unusually large” amount of food?
EDE-interview classification of eating (Fairburn & Beglin, 2008)

Objectively large amount of food?

<table>
<thead>
<tr>
<th>Yes</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of control?</td>
<td>Loss of control eating</td>
</tr>
<tr>
<td>Yes</td>
<td>OBJECTIVE BINGE</td>
</tr>
<tr>
<td>No</td>
<td>OBJECTIVE OVEREATING</td>
</tr>
</tbody>
</table>
To investigate the association between overeating and binge eating (overeating with loss of control) and adverse outcomes. 16,882 males and females who were 9–15 years old at enrollment in 1996. Overeating and binge eating assessed via questionnaire every 12–24 months between 1996 and 2005. Weekly binge (LOC) episodes, but not overeating, predicted overweight and onset of depression among adolescent females.
• F 50.0 Anorexia nervosa
• F 50.1 Atypical AN
• F 50.2 Bulimia nervosa
• F 50.3 Atypical BN
• F 50.4 Overeating associated with other psychological disturbances
• F 50.5 Vomiting associated with other psychological disturbances
• F 50.8 Other eating disorders
• F 50.9 Eating disorder, unspecified

How is BED coded in Norway?
Dear AED members,

Beginning **October 1, 2016** the ICD codes for some Feeding and Eating disorders have changed.

They are as follows:

- BED was F50.8 and changed to F50.81
- OSFED was F50.8 and changed to F50.89
- ARFID was F50.8 and changed to F50.89
- PICA in adults was F50.8 and changed to F50.89

Please [follow this link](#) to download AED’s updated graphic listing the DSM-5 and ICD codes for all the Feeding and Eating Disorders.

Warm regards,

Eva Trujillo, MD, FAED
President
Expected changes in ICD-11

- Binge eating disorder will be included as a diagnosis in ICD-11

- The frequency and duration of binge eating will match DSM-5 criteria (1 x week frequency for 1 month)

- Debates ongoing:
  - Subjective vs objective binges? Does size matter? Similar level of distress/impairment between SBE and OBE (*Mond et al.*, 2010; *Uher & Rutter*, 2012)
  - Overvaluation of body weight and shape. Will this become a diagnostic specifier?

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Overvaluation of shape/weight appears to have diagnostic and clinical relevance and warrants consideration as a diagnostic specifier for BED in the DSM-5. The comparative literature reviewed here on the concurrent validity of overvaluation in BED) suggests that - unlike the diagnosis of BN in which the present (versus absence) of overvaluation is a required criterion - the presence of overvaluation could serve as a specifier or subtype reflecting the presence of a significant cognitive disturbance in body image that also signals greater severity. The addition of this cognitive feature to the diagnosis of BED regarding a disturbance in body image would parallel the nosologic structure of the other formal eating-disorder diagnoses in the DSM-5. The DSM-system has previously used such specifiers/subtypes for other eating disorders (e.g., purging versus non-purging subtypes of bulimia nervosa, binge-purge verses restricting subtypes of anorexia nervosa) as well as for other diagnostic categories (mood disorders with mood congruent psychotic features). For BED, the presence of overvaluation would convey important information about individual differences and severity and about a clinically relevant cognitive feature that has negative prognostic significance.

Grilo, C.M. (2013). Why no cognitive body image feature such as overvaluation of shape/weight in the binge eating disorder diagnosis? IJED, 46 (3), 208-211
That special moment ... THE COFFEE KICKS IN.
B.E.D. is the most common eating disorder in US adults, more common than anorexia and bulimia combined.**

**Data from a sample of 2,980 adults aged ≥18 years who were assessed for an eating disorder in a national survey.

Table 1. Lifetime and 12-month prevalence of DSM-IV/CIDI BN and BED in the WMH survey

<table>
<thead>
<tr>
<th>Country</th>
<th>Lifetime BN</th>
<th>Lifetime BED</th>
<th>12-month BN</th>
<th>12-month BED</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>% (se)</td>
<td>% (se)</td>
<td>% (se)</td>
<td>% (se)</td>
</tr>
<tr>
<td>I. Lower-middle income</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Colombia</td>
<td>0.4 (0.1)</td>
<td>0.9 (0.2)</td>
<td>0.2 (0.1)</td>
<td>0.3 (0.1)</td>
</tr>
<tr>
<td>II. Upper-middle income</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brazil (São Paulo)</td>
<td>2.0 (0.2)</td>
<td>4.7 (0.3)</td>
<td>0.9 (0.2)</td>
<td>1.8 (0.3)</td>
</tr>
<tr>
<td>Mexico</td>
<td>0.8 (0.2)</td>
<td>1.6 (0.4)</td>
<td>0.3 (0.1)</td>
<td>0.5 (0.2)</td>
</tr>
<tr>
<td>Romania</td>
<td>0.0 (0.0)</td>
<td>0.2 (0.0)</td>
<td>0.0 (0.0)</td>
<td>0.1 (0.0)</td>
</tr>
<tr>
<td>III. High income</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Belgium</td>
<td>1.0 (0.5)</td>
<td>1.2 (0.4)</td>
<td>0.3 (0.3)</td>
<td>0.7 (0.4)</td>
</tr>
<tr>
<td>France</td>
<td>0.7 (0.4)</td>
<td>1.7 (0.8)</td>
<td>0.2 (0.2)</td>
<td>0.3 (0.2)</td>
</tr>
<tr>
<td>Germany</td>
<td>0.3 (0.1)</td>
<td>0.5 (0.2)</td>
<td>0.2 (0.1)</td>
<td>0.1 (0.1)</td>
</tr>
<tr>
<td>Italy</td>
<td>0.1 (0.1)</td>
<td>0.7 (0.3)</td>
<td>0.0 (0.0)</td>
<td>0.2 (0.1)</td>
</tr>
<tr>
<td>Netherlands</td>
<td>0.9 (0.5)</td>
<td>0.9 (0.5)</td>
<td>0.1 (0.1)</td>
<td>0.1 (0.1)</td>
</tr>
<tr>
<td>New Zealand</td>
<td>1.3 (0.1)</td>
<td>1.9 (0.2)</td>
<td>0.5 (0.1)</td>
<td>1.0 (0.1)</td>
</tr>
<tr>
<td>Northern Ireland</td>
<td>0.5 (0.1)</td>
<td>1.5 (0.3)</td>
<td>0.2 (0.1)</td>
<td>0.8 (0.3)</td>
</tr>
<tr>
<td>Portugal</td>
<td>0.8 (0.3)</td>
<td>2.4 (0.6)</td>
<td>0.5 (0.2)</td>
<td>1.1 (0.4)</td>
</tr>
<tr>
<td>Spain</td>
<td>0.7 (0.4)</td>
<td>0.8 (0.3)</td>
<td>0.1 (0.1)</td>
<td>0.5 (0.3)</td>
</tr>
<tr>
<td>US</td>
<td>1.0 (0.2)</td>
<td>2.6 (0.3)</td>
<td>0.3 (0.1)</td>
<td>1.2 (0.2)</td>
</tr>
<tr>
<td>IV. Total</td>
<td>1.0 (0.1)</td>
<td>1.9 (0.1)</td>
<td>0.4 (0.1)</td>
<td>0.8 (0.1)</td>
</tr>
</tbody>
</table>

Prevalence of BED in Norway

- Only a couple of studies from Norway (Lindvall Dahlgren & Wisting. 2016)

- Lifetime prevalence for BED was 1.2% for girls and 0.9% for boys based on N = 1960 adolescents, 14–15 years of age (Kjelsås et al., 2004) using self-report (SEDS).

- Point prevalence for BED was 1.0% for girls, 0.0% for boys (Rosenvinge et al., 1999) based on N = 678 adolescents screened with EDI, interviewed with DSED.

- We do not know how many have BED in Norway, or how many are in treatment in Norway.

Survey of N = 22,000 US adults

Estimate lifetime BED according to DSM-5 was 2.03%

Estimated lifetime BED according to DSM-IV was 1.52%

Among survey respondents who met DSM-5 criteria for BED, only 3.2% had ever received a formal diagnosis from a health care provider.

Those who had been diagnosed were younger, had a higher BMI and lower self-esteem (RSE)

*Unmet need in BED recognition and diagnosis!*


https://doi.org/10.4088/JCP.15m10059
Approximately 40-50% are men (Uher et al., 2012). Most common ED among men (Guerdjikova et al., 2017)

Age

- Age of onset is during late adolescence or young adulthood (median = 23.3 for BED vs 20.6 yrs for BN; Kessler et al., 2013)

- Mid-40s typical age at presentation (Lydecker and Grilo, 2016; APA, 2013)

- Although upon assessment, some recall binge eating during childhood “as long as I can remember”
In epidemiological studies, BED is as prevalent in Black and Hispanic as White individuals (Hudson et al., 2007; Marques et al., 2011). **But** mental health service utilization is lower among ethnic minorities.
Associated features

- 79% of individuals with BED had met lifetime criteria for at least one other DSM-IV disorder (Kessler et al., 2013), e.g., anxiety, depressive, impulse-control disorders (Grilo et al., 2013).

- Psychiatric comorbidity appear to be linked to severity of binge eating, not BMI (APA, 2013; Welch et al., 2016).

- Elevated risk of suicide (OR 1.8; 95% CI: 1.2-2.7) (Welch et al., 2016).

- Elevated risk of metabolic and obesity-related comorbidity.

- Lower QOL. Women with BED less likely to get married and men with BED are less likely to be employed (Kessler et al., 2013).

- Often a long history of dieting attempts (weight cycling).

• Avoidant PD was the most prevalent among the BED studies (0.18), followed by borderline (.12) and obsessive-compulsive PD (0.11) (N = 5 studies)

• The lowest proportions were found for schizotypal (0.01), schizoid (0.02), and histrionic (0.02) disorders.
Adults with B.E.D. may be of a normal weight, overweight, or obese.
BED can occur across the BMI spectrum, but is strongly associated with obesity (OR = 6.6; Kessler et al., 2013). Prevalence of obesity in BED estimated at approximately 36% to 42% (Villarego, Fernandez-Aranda et al., 2012) and is over-represented among those seeking weight loss treatment (Hilbert et al., 2014).
But most patients with obesity do not have BED.
BED and obesity

- The subset of obese patients with BED have a greater psychiatric illness burden than patients with just obesity:
  - Consume more kcal in laboratory studies of eating behavior
  - Greater functional impairment, lower QOL
  - More subjective distress
  - Higher body image dissatisfaction
  - Treatment needs (tackle both obesity and binge eating)
We hypothesized that binge eating was be a pathway to obesity and come first. However, 63% in our sample reported overweight came first.

**WHY?** Increased preoccupation with weight and shape? Internalization of thin-ideal caused body dissatisfaction? Exposure to weight teasing? All of which may have kick started unhealthy dieting behavior, later leading to binge eating?
Weight teasing most common form of bullying at school

Rudd Center for Food Policy and Obesity  http://www.uconnruddcenter.org/
Fat-blaming and fat-shaming

Weight stigma means negative attitudes, bias, stereotypes, prejudice toward someone because of their weight. “Last form of socially acceptable discrimination” (Puhl and Suh, 2015)
“There tends to be this public perception that maybe fat shaming is O.K. because it will provide motivation to lose weight. Instead, it is very harmful to health.” Rebecca Puhl, PhD
Stigma occurs across multiple domains of life which means persons with overweight/obesity are vulnerable to stigmatizing situations everywhere and it starts early in childhood.

- Media
- Education
- Employment
- Interpersonal relationships
- Social media
- Healthcare settings
Impact of weight bias and stigma on quality of care and outcomes for patients with obesity

SM Phelan, DJ Burgess, MW Yeazel, WL Hellerstedt, JM Griffin, and M van Ryn

Abstract

The objective of this study was to critically review the empirical evidence from all relevant disciplines regarding obesity stigma in order to (i) determine the implications of obesity stigma for healthcare providers and their patients with obesity and (ii) identify strategies to improve care for patients with obesity. We conducted a search of Medline and PsychInfo for all peer-reviewed papers presenting original empirical data relevant to stigma, bias, discrimination, prejudice and medical care. We then performed a narrative review of the existing empirical evidence regarding the impact of obesity stigma and weight bias for healthcare quality and outcomes. Many healthcare providers hold strong negative attitudes and stereotypes about people with obesity. There is considerable evidence that such attitudes influence person-perceptions, judgment, interpersonal behaviour and decision-making. These attitudes may impact the care they provide. Experiences of or expectations for poor treatment may cause stress and avoidance of care, mistrust of doctors and poor adherence among patients with obesity. Stigma can reduce the quality of care for patients with obesity despite the best intentions of healthcare
PARADOXICAL EFFECTS OF WEIGHT STIGMA

• Weight stigma is a shared risk factor for the development of ED and obesity

• Being called “fat” at age 10 increases risk of obesity at age 19, even after controlling for actual weight (Tomiyama et al., 2014 JAMA). “We nearly fell off our chairs when we discovered this” said the UCLA researchers who conducted the study!

• Females who experienced weight teasing during childhood (ages 6 and 12 yrs) reported more binge eating and LOC eating at age 20 yrs (Quick et al., 2013, Am J Public Health)

• Experimental studies show overeating increases following exposure to weight-bullying (Major et al., 2014, J. Experimental Social Psychology)
Weight stigma can lead to weight gain via binge eating

HOW CAN WE TACKLE WEIGHT STIGMA?

Increasing acceptance of diversity in body shape and size—teach kids this message

Anti-bullying programs in school

Laws to protect against workplace discrimination

Sensitivity training and communication training in medical school and other healthcare settings

Weight stigma should be addressed in obesity treatment "as a target, not a tool"

Using person-first and neutral language

Examine YOUR OWN implicit bias against obesity: https://implicit.harvard.edu/implicit/

“There is powerful biology underlying both obesity and eating disorders, it's not just a matter of willpower." Walter Kaye, MD

Puhl and Suh (2015)
• Compared to AN and BN, BED is perceived as most reflective of poor self-control. Stigma toward BED **exceeds stigma** toward AN and BN and toward obese individuals without binge eating disorder (Murakami et al., 2016).

• Beliefs that BED is caused by a lack of self-discipline is positively correlated with stigma (Ebneter & Latner, 2013).

• Many patients internalize negative attitudes and biases about weight, eating, and shape. High levels of shame, unwillingness to self-disclose, and personal blame for one's eating disorder create a sense of guilt and powerlessness.

• “Stigma adds to the terrible burden of eating disorders and acts as a barrier to effective care” John Morgan, UK Royal College of Psychiatrists’ ED Section

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**Stigma toward BED**

Lack of willpower, lazy, weak, gluttonous, poor self-control, personal blame, own fault, a woman’s problem, shameful, disgusting
Hang in there!
Figure 1. Simple Restraint Model of Binge Eating in BN
Figure 2. Simple Dual Pathway of Binge Eating (*emphasizes role of affect regulation in addition to dietary restraint in maintaining binge eating*)
Social pressure to be thin
History of weight teasing

Thin ideal internalization
Weight bias internalization

Shape/Weight/Medical Concerns

Negative affect

Emotion Regulation

Binge eating

OTHER RISK FACTORS:
Personal vulnerability to weight gain
Reward dysfunction (dopamine system)
Impulsivity
“Hedonic hunger” = cognitive deprivation for highly palatable foods
Craving, cue reactivity
Propensity to addictive response to highly processed foods

History of Dieting
Cognitive restraint («I shouldn’t eat that»)
Weight Cycling
Weight Suppression

Leptin (controls hunger)
Ghrelin
Resting metabolism rate

Disrupted hunger/satiety signals
Biology of weight loss

• “After the Biggest Loser, Their Bodies Fought to Regain Weight” http://www.nytimes.com/2016/05/02/health/biggest-loser-weight-loss.html

• Before show, Danny weighed 430 pounds. At the finale, he weighed 191 pounds; 1 year later, up to 295 pounds

• METABOLIC RATE: Now burns 800 fewer calories a day than would be expected for a man his size. Low levels of leptin (controls hunger) and high levels of ghrelin (stimulates hunger) (important to assess weight history)

Assessment and Treatment
Available self-reports

- EDE-Q (modified/short forms)
- Binge Eating Scale
- BULIT
- BITE
- EDI
- Patient Health Questionnaire
- QEWP-5***
- BED-7 (Shire) screening tool

*** Yanovski, Marcus, Wadden, & Walsh (2015). QEWP-5: An Updated Screening Instrument for BED. IJED, 48(3), 259–261. marcusmd@upmc.edu
Debate

Viewpoint #1: Treat the obesity

- "Public health experts who deal with obesity would say, 'If you're not treating the weight, it's like putting a band-aid on cancer,'" Kelly Brownell, PhD, Dean, Duke School of Public Health

Viewpoint #2: Treat the binge eating

- Fears in ED community that putting someone with an eating disorder on a "diet", that will make the eating problems worse (Restraint Model).
- Binge eating is distressing to patients and contributes to weight gain via a positive energy balance, so it’s important to treat outright

Viewpoint #3: Try to treat both (simultaneously or sequenced) in an interdisciplinary approach
Treatment goals

• Reduce binge eating
• Address body image concerns and associated ED psychopathology
• Tackle depressive and comorbid symptomology
• Improve metabolic health and weight

<table>
<thead>
<tr>
<th>Intervention Type</th>
<th>Treatment</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychological and behavioral</td>
<td>Cognitive behavioral therapy</td>
<td>Psychotherapy that focuses on identifying relations among thoughts, feelings, and behaviors, aiming to change negative thoughts about oneself and the world and, by doing so, reduce negative emotions and undesirable behavior patterns. Cognitive behavioral therapy is delivered in various ways—e.g., therapist-led individual and group sessions, self-help, and guided self-help.</td>
</tr>
<tr>
<td>Psychological and behavioral</td>
<td>Dialectical behavioral therapy</td>
<td>Behavioral therapy that focuses on increasing mindfulness and developing skills to improve emotion regulation, distress tolerance, and interpersonal relationships.</td>
</tr>
<tr>
<td>Psychological and behavioral</td>
<td>Interpersonal psychotherapy</td>
<td>Psychotherapy that focuses on the role of interpersonal functioning in negative mood, psychological distress, and unhealthy behaviors.</td>
</tr>
<tr>
<td>Psychological and behavioral</td>
<td>Behavioral weight loss</td>
<td>Treatment that incorporates various behavioral strategies to promote weight loss, such as caloric restriction and increased physical activity.</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>Second-generation and tricyclic antidepressants</td>
<td>Treatment with a class of medications that works by selectively inhibiting reuptake of neurotransmitters involved in the regulation of mood and appetite (i.e., dopamine, norepinephrine, and serotonin). Common examples include bupropion, citalopram, desipramine, duloxetine, fluoxetine, and sertraline, commonly indicated for patients with depression.</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>Anticonvulsants</td>
<td>Treatment with a class of medications used to treat epilepsy, bipolar disorder, major depression, and migraines; most commonly, topiramate.</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>Antiobesity</td>
<td>Treatment with medications used to treat obesity. One example is orlistat, which inhibits pancreatic lipase, thereby decreasing fat absorption in the gut.</td>
</tr>
<tr>
<td>Pharmacological</td>
<td>Central nervous system stimulants</td>
<td>Treatment with a class of medications generally used to enhance or accelerate mental and physical processes, and specifically for treating patients with attention-deficit hyperactivity disorder and certain sleep problems. The only medication approved by the U.S. Food and Drug Administration for binge-eating disorder (lisdexamfetamine) belongs to this class.</td>
</tr>
</tbody>
</table>
Table 1. Interventions Commonly Used in Treating Patients With Binge-Eating Disorder

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Psychological, behavioral, or both</strong></td>
<td></td>
</tr>
<tr>
<td>CBT</td>
<td>Focuses on identifying relationships among thoughts, feelings, and behaviors; aims to reduce negative emotions and undesirable behavior patterns by changing negative thoughts about oneself and the world. CBT may be delivered in various forms according to the level of therapist involvement—e.g., from therapist engaged in all aspects of treatment (therapist-led CBT) to no therapist engagement (self-help CBT). In self-help CBT, the patient follows a treatment manual or book, either with the help of a facilitator (e.g., guided or structured self-help) or alone. CBT may be tailored to the patient by focusing on problematic eating-related cognitions and behaviors.</td>
</tr>
<tr>
<td>Dialectical behavior therapy</td>
<td>Focuses on increasing mindfulness and developing skills to improve emotion regulation, distress tolerance, and interpersonal relationships to help patients respond to stress and negative affect more effectively.</td>
</tr>
<tr>
<td>Interpersonal psychotherapy</td>
<td>Focuses on identifying and changing the role of interpersonal functioning in causing and maintaining negative mood, psychological distress, and unhealthy behaviors.</td>
</tr>
<tr>
<td>Behavioral weight loss</td>
<td>Incorporates various behavioral strategies to promote weight loss, such as restricting caloric intake and increasing physical activity.</td>
</tr>
<tr>
<td><strong>Pharmacologic</strong></td>
<td></td>
</tr>
<tr>
<td>Antidepressants</td>
<td>Selectively inhibit reuptake of neurotransmitters involved in regulating mood and appetite (i.e., dopamine, norepinephrine, and serotonin). Common examples include buproprion, citalopram, desipramine, duloxetine, fluoxetine, fluvoxamine, and sertraline, which are indicated for treating patients with depression.</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>Indicated for treating patients with epilepsy, bipolar disorder, major depression, and migraines. Topiramate, a carbonic anhydrase inhibitor, is the most commonly used.</td>
</tr>
<tr>
<td>Antiobesity agents</td>
<td>Used to treat obesity. For example, orlistat inhibits pancreatic lipase and thus decreases fat absorption in the gut.</td>
</tr>
<tr>
<td>Central nervous system stimulants</td>
<td>Generally used to enhance or accelerate mental and physical processes; specifically used to treat attention deficit-hyperactivity disorder and certain sleep problems. Lisdexamfetamine, the only U.S. Food and Drug Administration-approved medication for binge-eating disorder, belongs to this class.</td>
</tr>
</tbody>
</table>

CBT = cognitive behavioral therapy.
UK, US, ANZP Guidelines

• CBT self-help is recommended as first-line treatment by NICE guidelines with a methodological grade “A” (2004)

• “Strong recommendation” for individual and group CBT, as well as guided self-help (gshCBT). APA Practice Guideline for the Treatment of Patients with Eating Disorders, 3rd edition (2012).

• CBT-therapist led rated as “high benefit” by 2015 US Agency for Healthcare Research and Quality Report (Berkman et al., 2015)

• RANZCP Clinical Practice Guidelines (2014) recommends an individual psychological therapy for which the “best evidence” is for therapist-led cognitive behavioural therapy (CBT). There is also a role for CBT adapted for internet delivery, or CBT in a non-specialist guided self-help form.

Also see: Peterson et al. (2016). The three-legged stool of evidence-based practice in ED treatment: research, clinical and patient perspectives. BMC Medicine, 14:69.
• CBT generally achieves 50% binge abstinence within 16 wks, maintained up to 2-4 years.

• Placebo rates in BED trials are app. 30% (about the same as pain trials)
Binge-Eating Disorder in Adults
A Systematic Review and Meta-analysis

Kimberly A. Brownley, PhD; Nancy D. Berkman, PhD; Christine M. Peat, PhD; Kathleen N. Lohr, PhD; Katherine E. Cullen, BA; Carla M. Bann, PhD; and Cynthia M. Bulik, PhD

Background: The best treatment options for binge-eating disorder are unclear.

Purpose: To summarize evidence about the benefits and harms of psychological and pharmacologic therapies for adults with binge-eating disorder.

Data Sources: English-language publications in EMBASE, the Cochrane Library, Academic OneFile, CINAHL, and ClinicalTrials.gov through 18 November 2015, and in MEDLINE through 12 May 2016.

Study Selection: 9 waitlist-controlled psychological trials and 25 placebo-controlled trials that evaluated pharmacologic (n = 19) or combination (n = 6) treatment. All were randomized trials with low or medium risk of bias.

Data Extraction: 2 reviewers independently extracted trial data, assessed risk of bias, and graded strength of evidence.

Data Synthesis: Therapist-led cognitive behavioral therapy, lisdexamfetamine, and second-generation antidepressants (SGAs) decreased binge-eating frequency and increased binge-eating abstinence (relative risk, 4.95 [95% CI, 3.06 to 8.00], 2.61 [CI, 2.04 to 3.33], and 1.67 [CI, 1.24 to 2.26], respectively). Lisdexamfetamine (mean difference [MD], −6.50 [CI, −8.82 to −4.18]) and SGAs (MD, −3.84 [CI, −6.55 to −1.13]) reduced binge-eating-related obsessions and compulsions, and SGAs reduced symptoms of depression (MD, −1.97 [CI, −3.67 to −0.28]). Headache, gastrointestinal upset, sleep disturbance, and sympathetic nervous system arousal occurred more frequently with lisdexamfetamine than placebo (relative risk range, 1.63 to 4.28). Other forms of cognitive behavioral therapy and topiramate also increased abstinence and reduced binge-eating frequency and related psychopathology. Topiramate reduced weight and increased sympathetic nervous system arousal, and lisdexamfetamine reduced weight and appetite.

Limitations: Most study participants were overweight or obese white women aged 20 to 40 years. Many treatments were examined only in single studies. Outcomes were measured inconsistently across trials and rarely assessed beyond end of treatment.

Conclusion: Cognitive behavioral therapy, lisdexamfetamine, SGAs, and topiramate reduced binge eating and related psychopathology, and lisdexamfetamine and topiramate reduced weight in adults with binge-eating disorder.

Primary Funding Source: Agency for Healthcare Research and Quality.

For author affiliations, see end of text.
This article was published at www.annals.org on 28 June 2016.
For binge eating, combining pharmacotherapy with psychotherapy was better than pharmacotherapy alone, but not better than psychotherapy alone. For weight loss, topiramate (anti-epileptic) produced better weight loss than with CBT alone (e.g., 6.8 kg vs 0.9 kg). Orlistat (prevents absorption of fats) has improved weight losses with CBT alone, albeit minimally.
• Rapid response seems to be a treatment-specific positive prognostic indicator of sustained remission from binge-eating in CBTgsh and CBT therapist-led. About 50% of patients have a rapid response from CBT by week 4, maintained up to 1 year.

• Rapid response typically defined as 70% reduction in binge eating by week 4

• What is going on early in treatment in CBT?

Grilo et al. (2015). Predicting Meaningful Outcomes to Medication and Self-Help Treatments for Binge-Eating Disorder in Primary Care: The significance of early rapid response; JCCP dx.doi.org/10.1037/a0038635)
• **Behavioral Phase:** The patient and therapist work together to formulate a plan to establish a regular pattern of eating (3 meals, 2 snacks per day), educate them about BED, and start daily self-monitoring. Because emotions often intensify during this phase of treatment, tools (coping strategies) for managing these feelings are developed and become an important part of the work. CBT includes in-session activities as well as “homework” so that new behaviors can be practiced.

• **Cognitive Phase:** As treatment progresses, cognitive restructuring techniques (e.g., techniques aimed at recognizing and changing problem thinking patterns) are introduced. Phase 2 teaches patients to identify and challenge negative thoughts regarding their weight, body image, interpersonal interactions, or stressful situations that may trigger binge-eating episodes. Thoughts and beliefs that perpetuate the problems are identified and work aimed at developing new perspectives and ideas ("my self-worth doesn’t depend on my size or shape") begins. Additionally, during this stage of treatment, broader concerns such as relationship problems, body image, self-esteem problems, and emotion regulation are addressed.

• **Maintenance & Relapse Prevention Phase:** The final stage of CBT concentrates on problem-solving, reducing triggers, preventing relapse and maintaining the progress that’s been made.

Regarding an evidence-based stepped care model, IPT, equally efficacious for rapid and non-rapid responders, could be investigated as a second-line treatment in case of non-rapid response to first-line CBTgsh.

• **But...** CBT and other specialist psychological interventions (IPT and DBT) do not address nor produce significant weight loss
Review and Meta-analysis of Pharmacotherapy for Binge-eating Disorder

Deborah L. Reas¹,² and Carlos M. Grilo³,⁴

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²Regional Eating Disorders Service (RASP), Psychiatric Division, Ullevål University Hospital, Oslo, Norway
³Department of Psychiatry, Yale University School of Medicine, New Haven, Connecticut, USA
⁴Department of Psychology, Yale University, New Haven, Connecticut, USA.

Abstract

This study evaluated available controlled treatment studies to determine utility of pharmacotherapy for binge-eating disorder (BED). The authors identified randomized placebo-controlled trials testing pharmacotherapy-only treatments and controlled trials testing pharmacotherapy with psychotherapy treatments. Meta-analysis was performed on placebo-controlled trials with data for attrition, remission, and weight loss. Qualitative review was performed on remaining controlled treatment literature. A total of 33 studies were considered of which 14 studies with a total of 1,279 patients were included in the meta-analysis of pharmacotherapy-only treatment and 8 studies with a total of 683 patients were included in the qualitative review of pharmacotherapy combined with psychotherapy interventions. No evidence suggested significant differences between medication and placebo for attrition. Evidence suggested that pharmacological treatments have a clinically significant advantage over placebo for achieving short-term remission from binge eating (48.7% vs. 28.5%) and for weight loss, although weight losses are not substantial. No data exist to allow evaluation of longer-term effects of pharmacotherapy-only treatment for BED. Combining medications with psychotherapy interventions failed to significantly enhance binge outcomes, although specific medications (orlistat, topiramate) enhanced weight losses achieved with cognitive behavioral therapy and behavioral weight loss. In summary, BED patients can be advised that certain pharmacotherapies may enhance likelihood of stopping binge eating short term, but that longer-term effects are unknown. Although some weight loss may occur, it is unlikely to be substantial with available medications. Combining medications with cognitive or behavioral treatments is unlikely to enhance binge outcomes, but specific medications (orlistat, topiramate) may enhance weight losses, albeit modestly.
Current and emerging drug treatments for binge eating disorder

Deborah L Reas & Carlos M Grilo†
†Yale University School of Medicine, Department of Psychiatry, Program for Obesity Weight Eating Research, New Haven, CT, USA

**Introduction:** This study evaluated controlled treatment studies of pharmacotherapy for binge eating disorder (BED).

**Areas covered:** The primary focus of the review was on Phase II and III controlled trials testing medications for BED. A total of 46 studies were considered and 26 were reviewed in detail. BED outcomes included binge eating remission, binge eating frequency, associated eating disorder psychopathology, associated depression and weight loss.

**Expert opinion:** Data from controlled trials suggest that certain medications are superior to placebo for stopping binge eating and for producing faster reductions in binge eating, and – to varying degrees – for reducing associated eating disorder psychopathology, depression and weight loss over the short term. Almost no data exist regarding longer-term effects of medication for BED. Except for topiramate, which reduces both binge eating and weight, weight loss is minimal with medications tested for BED. Psychological interventions and the combination of medication with psychological interventions produce binge eating outcomes that are superior to medication-only approaches. Combining medications with psychological interventions does not significantly enhance binge eating outcomes, although the addition of certain medications enhances weight losses achieved with cognitive-behavioral therapy and behavioral weight loss, albeit modestly.
Summary

- Between 1985-2016, over 30 phase II and III RCTS for BED. Anti-obesity, anti-epileptic, and anti-depressive, anti-craving, anti-addiction, ADHD, narcolepsy. Two anti-obesity drugs have been pulled from market (sibutramine and rimonabant).

- In 2015, first FDA-approved medication (lisdexamfetamine) was approved in US for moderate-to-severe BED in doses 50 mg/day and 70 mg/day. Based on 85% reduction in binge eating days versus 50% for placebo at 11-weeks.

- LDX vs placebo (50 and 70 mg/day) produced -4.9 kg losses (about 5-6% loss), but LDX is not indicated or recommended for weight loss due to cardiovascular risk (“Limitation of Use”) and there are other restrictions (Schedule 2)

- As with CBT, DBT, and IPT, pharmacological-only treatment have generally failed to produce clinically meaningful weight loss. Exception is topiramate (-4.5 kg loss vs + 0.2 for placebo, but topiramate is associated with cognitive adverse events.
## Outcome: Weight change (kg)

<table>
<thead>
<tr>
<th>Study or subcategory</th>
<th>N</th>
<th>Medication Mean (SD)</th>
<th>Placebo Mean (SD)</th>
<th>WMD (random) 95% CI</th>
<th>Weight %</th>
<th>WMD (random) 95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>01 SSRIs</strong></td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Arnold et al. [26]*</td>
<td>23</td>
<td>−3.30 (0.00)</td>
<td>0.70 (0.00)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grijal etc [14]</td>
<td>27</td>
<td>−2.19 (7.40)</td>
<td>0.00 (4.20)</td>
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<td></td>
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<tr>
<td>Guardjikova et al. [19]</td>
<td>20</td>
<td>−1.00 (2.60)</td>
<td>0.60 (2.40)</td>
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<td></td>
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<tr>
<td>Hudson et al. [16]</td>
<td>42</td>
<td>−1.22 (0.00)</td>
<td>−0.14 (0.00)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>McElroy et al. [17]</td>
<td>18</td>
<td>−5.60 (0.00)</td>
<td>−2.40 (0.00)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>McElroy et al. [16]*</td>
<td>16</td>
<td>−2.10 (0.00)</td>
<td>0.20 (0.00)</td>
<td></td>
<td></td>
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<tr>
<td>Pearlstein et al. [15]*</td>
<td>9</td>
<td>−0.45 (10.79)</td>
<td>1.80 (8.90)</td>
<td>0.88</td>
<td>21.05</td>
<td>−1.72 [−3.06, −0.37]</td>
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<tr>
<td><strong>02 SNRI</strong></td>
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<tr>
<td>McElroy et al. [20]</td>
<td>20</td>
<td>−2.70 (3.70)</td>
<td>0.00 (3.20)</td>
<td></td>
<td>9.87</td>
<td>−2.70 [−4.84, −0.56]</td>
</tr>
<tr>
<td><strong>03 Antiepileptics</strong></td>
<td></td>
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<tr>
<td>McElroy et al. [21]</td>
<td>28</td>
<td>−5.90 (0.00)</td>
<td>−1.20 (0.00)</td>
<td></td>
<td>10.03</td>
<td>−3.80 [−5.92, −1.68]</td>
</tr>
<tr>
<td>McElroy et al. [22]</td>
<td>30</td>
<td>−4.80 (5.10)</td>
<td>−1.00 (3.00)</td>
<td></td>
<td>21.77</td>
<td>−4.70 [−5.54, −3.86]</td>
</tr>
<tr>
<td>McElroy et al. [23]</td>
<td>195</td>
<td>−4.50 (5.10)</td>
<td>0.20 (3.20)</td>
<td></td>
<td>31.80</td>
<td>−4.58 [−5.36, −3.79]</td>
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<tr>
<td><strong>04 Obesity medication</strong></td>
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<tr>
<td>Appolinaro et al. [24]*</td>
<td>23</td>
<td>−7.40 (0.00)</td>
<td>1.40 (0.00)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Milano et al. [13]</td>
<td>10</td>
<td>−4.48 (2.10)</td>
<td>−0.59 (0.05)</td>
<td></td>
<td>16.67</td>
<td>−3.89 [−5.19, −2.59]</td>
</tr>
<tr>
<td>Willey et al. [25]</td>
<td>152</td>
<td>−4.30 (4.80)</td>
<td>−0.80 (3.50)</td>
<td></td>
<td>20.61</td>
<td>−3.50 [−4.44, −2.56]</td>
</tr>
<tr>
<td><strong>Subtotal (95% CI)</strong></td>
<td>185</td>
<td></td>
<td></td>
<td></td>
<td>37.28</td>
<td>−3.63 [−4.40, −2.87]</td>
</tr>
<tr>
<td><strong>Total (95% CI)</strong></td>
<td>613</td>
<td></td>
<td></td>
<td></td>
<td>100.00</td>
<td>−3.42 [−4.25, −2.58]</td>
</tr>
</tbody>
</table>

Test for heterogeneity: $\chi^2 = 0.12$, df = 2 ($P = 0.94$), $I^2 = 0$

Test for overall effect: $Z = 2.50$ ($P = 0.01$)

Test for heterogeneity: $\chi^2 = 0.60$, df = 1 ($P = 0.44$), $I^2 = 0$

Test for overall effect: $Z = 11.46$ ($P < 0.000001$)
Methodological limitations of treatment trials

- RCTs short (generally 6-24 weeks)
- Lack of long-term FUP (“acute-care” design for BED compared to obesity trials generally “life-long”).
- Most subjects are female, white
- Many trials exclude patients with depression or comorbidity, reducing generalizability and representativeness. INCLUDING THE LDX TRIALS
• BED is the most prevalent eating disorder
• Binge eating is different than overeating
• About 40% are men, older age at presentation than AN and BN
• Highly stigmatized group
• BED is a real condition and it can be treated.

• More research on what works for whom. How can we better tailor and sequence treatments? How can we tackle complex treatment needs?
• The Binge Eating Disorder Association (BEDA) is the largest national organization focused on providing leadership, recognition, prevention, and treatment of BED and associated weight stigma. Founded in 2008. Through outreach, education and advocacy, BEDA facilitates increased awareness, proper diagnosis, and treatment of BED.  http://bedaonline.com/

THANK YOU!