Overview of Binge Eating Disorder: Diagnosis, Etiology and Treatment

Grand Rounds, Department of Psychiatry, Duke University
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Characteristics of an Eating Disorder

1) The presence of disturbed eating behaviour

2) The presence of characteristic psychopathology

Obesity per se is not an eating disorder but is best conceptualized as a complex, heterogeneous multi-determined metabolic disturbance. However, a significant percentage, about 35%, of obese subjects do have disturbed eating behavior and characteristic psychopathology:

Binge Eating Disorder
ANOREXIA NERVOSA

BULIMIA NERVOSA

BINGE EATING DISORDER
Binge Eating Disorder – DSM 5 Criteria

A. Recurrent episodes of binge eating. An episode of binge eating is:

1. Eating, in a discrete period of time an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances
2. A sense of lack of control over eating during the episode

B. The binge-eating episodes are associated with the following:

1. Eating much more rapidly than normal
2. Eating until feeling uncomfortably full
3. Eating large amounts of food when not feeling physically hungry
4. Eating alone because of feeling embarrassed by how much one is eating
5. Feeling disgusted with oneself, depressed, or very guilty after overeating
Binge Eating Disorder – DSM 5 Criteria

C. Marked distress regarding binge eating is present.

D. The binge eating occurs at least once a week for 3 months.

E. The binge eating is not associated with the recurrent use of inappropriate compensatory behavior and does not occur exclusively during the course of Bulimia Nervosa or Anorexia Nervosa.
Binge Eating Disorder

Epidemiology:

• BED is more common than the other major eating disorders anorexia nervosa or bulimia nervosa

• Lifetime prevalence of ~3% among women; 2% among men in US (Hudson et al., 2007), and ~2.0% EU (pooled WMH survey; Kessler et al., 2013)

• VALIDATE Study: large Canadian epidemiologic study (over 10,000 respondents). Lifetime prevalence: 3.5%

• 65% female, 35% male
The epidemiology of eating disorders in six European countries: Results of the ESEMeD-WMH project

Cumulative lifetime prevalence of anorexia nervosa, bulimia nervosa, binge eating disorder, and any binge eating.

A 4-fold increase in morbid obesity over the same period as BED having emerged
Binge Eating Disorder: Comorbidity

Lifetime Prevalence

- Obesity: 88% (associated with lower quality of life than obesity without BED)
- Affective Disorder: 68%
- Anxiety Disorder: 42%
- Substance Use: 12%
- ADHD: 30%
Binge Eating Disorder: Comorbidity

Medical Comorbidity from VALIDATE STUDY:

- Stroke (OR 3.77)
- Liver Disease (OR 3.06)
- Sleep Apnea (2.68)
- Kidney Disease (OR 2.13)
- Cancer (OR 2.03)
- Elevated cholesterol (OR 1.6)
- Diabetes (OR 1.59)
- Hypertension (OR 1.2)
Binge Eating Disorder

Current clinical experience in North America:

- Most individuals with BED do not realize they have an eating disorder.
- As a result, individuals with BED do not seek treatment from eating disorder clinics/programs.
- When they do seek help, it is usually for obesity treatment for weight loss, including bariatric surgery, or for another comorbidity.
- We as professionals have an important responsibility to educate the public and primary care providers about BED and its treatment.
### Binge Eating Disorder Screener-7 (BEDS-7)
(Herman et al 2016)

1. **During the last 3 months**, did you have any episodes of excessive overeating (i.e., eating significantly more than what most people would eat in a similar period of time)?

2. Do you feel distressed about your episodes of excessive overeating?

<table>
<thead>
<tr>
<th>Within the past 3 months...</th>
<th>Never or Rarely</th>
<th>Sometimes</th>
<th>Often</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>3. <strong>During the episodes of excessive overeating</strong>, how often did you feel like you had no control over your eating, (e.g., not being able to stop eating, feel compelled to eat, or going back and forth for more food)?</td>
<td></td>
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<tr>
<td>4. <strong>During your episodes of excessive overeating</strong>, how often did you continue eating even though you were not hungry?</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>5. <strong>During your episodes of excessive overeating</strong>, how often were you embarrassed by how much you ate?</td>
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<tr>
<td>6. <strong>During your episodes of excessive overeating</strong>, how often did you feel disgusted with yourself or guilty afterwards?</td>
<td></td>
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<tr>
<td>7. <strong>During the last 3 months</strong>, how often did you make yourself vomit as a means to control your weight or shape?</td>
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</table>
Food Addiction
Yale Food Addiction Scale

(Gearhardt, Corbin, & Brownell, 2009)

1. Taking in larger amounts of food and for longer periods.
2. Repeated unsuccessful attempts to quit overeating.
3. Increasing amount of time spent obtaining and eating food.
4. Social/recreational activities stopped or reduced.
5. Use despite knowledge of adverse consequences.
6. Tolerance.
7. Withdrawal symptoms.
8. Clinically significant impairment.
<table>
<thead>
<tr>
<th>Variable</th>
<th>FA Mean</th>
<th>SD</th>
<th>Non-FA Mean</th>
<th>SD</th>
<th>p =</th>
<th>Non-BED/FA Mean</th>
<th>SD</th>
<th>p =</th>
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</thead>
<tbody>
<tr>
<td>Age</td>
<td>34.2</td>
<td>5.6</td>
<td>31.8</td>
<td>6.1</td>
<td>0.254</td>
<td>32.6</td>
<td>0.649</td>
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<tr>
<td>BMI</td>
<td>37.1</td>
<td>5.6</td>
<td>39.2</td>
<td>5.1</td>
<td>0.258</td>
<td>37.5</td>
<td>0.291</td>
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<tr>
<td>Emotional Eating</td>
<td>4.1</td>
<td>0.5</td>
<td>3.2</td>
<td>0.9</td>
<td>0.002</td>
<td>3.0</td>
<td>0.343</td>
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<tr>
<td>External Eating</td>
<td>3.8</td>
<td>0.4</td>
<td>3.4</td>
<td>0.6</td>
<td>0.034</td>
<td>3.4</td>
<td>0.939</td>
<td></td>
</tr>
<tr>
<td>Binge Eating</td>
<td>4.5</td>
<td>0.7</td>
<td>3.2</td>
<td>1.2</td>
<td>0.001</td>
<td>1.8</td>
<td>&lt;0.0001</td>
<td></td>
</tr>
<tr>
<td>Hedonic Eating</td>
<td>82.9</td>
<td>15.4</td>
<td>64.1</td>
<td>18.0</td>
<td>0.003</td>
<td>53.0</td>
<td>0.023</td>
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<tr>
<td>Food Cravings</td>
<td>177.0</td>
<td>25.2</td>
<td>139.3</td>
<td>37.0</td>
<td>0.002</td>
<td>120.4</td>
<td>0.038</td>
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<tr>
<td>Addictive Personality Traits</td>
<td>17.9</td>
<td>5.3</td>
<td>12.5</td>
<td>4.2</td>
<td>0.003</td>
<td>12.3</td>
<td>0.868</td>
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<tr>
<td>Impulsivity</td>
<td>71.3</td>
<td>11.5</td>
<td>60.0</td>
<td>7.9</td>
<td>0.002</td>
<td>64.6</td>
<td>0.109</td>
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<tr>
<td>Depression Symptoms</td>
<td>21.2</td>
<td>13.8</td>
<td>10.4</td>
<td>7.4</td>
<td>0.008</td>
<td>10.6</td>
<td>0.925</td>
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<tr>
<td>ADHD Symptoms (adult)</td>
<td>35.4</td>
<td>17.2</td>
<td>25.6</td>
<td>13.5</td>
<td>0.076</td>
<td>25.2</td>
<td>0.908</td>
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<tr>
<td>ADHD Symptoms (child)</td>
<td>37.6</td>
<td>24.0</td>
<td>24.2</td>
<td>17.0</td>
<td>0.073</td>
<td>25.5</td>
<td>0.798</td>
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</tbody>
</table>
Binge Eating Disorder

\[ X^2 = 21.62 \]
\[ p < 0.0001 \]

Obese Controls

- Food Addiction
- No Food Addiction

\[ X^2 = 21.62 \]
\[ p < 0.0001 \]
A Spectrum of Severity

- Passive Overeating
- Compulsive Eating
- Binge Eating Disorder
- BED with Food Addiction

Davis (2013) *Current Obesity Reports*
Neurobiology of BED
Responsiveness of the Ventral Striatum (VS)

• VS regulates reward-related and appetitive behaviours.
• Increased dopamine signaling would imply increased VS reactivity.
• Increased VS reactivity would reflect greater ‘reward sensitivity’.
Neuroimaging and Binge Eating Disorder

1. Obese adults with BED had increased dopamine activity in the striatum to food versus neutral cues.

2. Greater fronto-cortical activation to palatable food cues in women.

3. Men had enhanced ability to suppress fronto-cortical activation to food cues when requested to do so.

Wang et al. (2009) *PNAS*, 106
Biopsychosocial Model of Etiology for Eating Disorders

Predisposing Factors
- Genetics

Precipitating Factors
- Premorbid Obesity + Mood Instability
- Puberty/Psychological Event
- Body Dissatisfaction

Perpetuating Factors
- Caloric Restriction

Psychologic
- Identity, Self-esteem, Autonomy, Sexuality

Socio-cultural
- Family, Vocation, Media

Binge Eating
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Perpetuating Factors

Binge Eating
A Range of Genetic Influence

COMpletely GENETIC
Cystic fibrosis
Huntington’s Disease

COMpletely ENVIRONMENTAL
Religious affiliation

Most human characteristics are partially inherited
Genes or Jeans?

Sources of family resemblance and differences

- Resemblance:
  - Genes
  - Shared environment

- Differences:
  - Psychiatric disorder
  - Non-shared environment
Characteristics of Partially Inherited Disorders

• Aggregates (clusters) in families

• Does binge eating disorder run in families?

• Yes, but family studies CANNOT tell us if due to genes or environment

• Only twin studies can:

  Twin studies report higher concordance rates in MZ vs DZ twins
Quantification of Effects

(A) Additive effects of genes
   complex trait
   influenced by many genes of small/moderate effect

(C) “Shared” Environmental Effects
   religion
   parental rearing style
   socioeconomic status

(E) “Unique” Environmental Effects
   events experienced by one twin only
Heritability of Binge Eating Disorder

- **A**: 50%-65% (Additive Genetic)
- **E**: 35%-50% (Unique Env't)
- **C**: 0% (Shared Env't)
What Do Twin Study Results Mean?

• 50-65% of variance in liability to BED is due to additive genetic factors

• Impact of shared environment not substantial
• Heightened DA signaling
• Higher Reward Sensitivity
• More appetitive and hedonic response to food

**Reward Surfeit Syndrome:** (Hyper-sensitivity to rewarding stimuli)

\[ p = 0.008 \]
Biopsychosocial Model of Etiology for Eating Disorders

Predisposing Factors
- Biologic
  - Genetics
- Psychologic
  - Identity, Self-esteem, Autonomy, Sexuality
- Socio-cultural
  - Family, Vocation, Media

Precipitating Factors
- Premorbid Obesity + Mood Instability
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Perpetuating Factors

Binge Eating
"You can't find love in a bag of chips. But chips and salsa come close."
Biopsychosocial Model of Etiology for Eating Disorders

Predisposing Factors
- Genetics

Psychologic Factors
- Identity, Self-esteem, Autonomy, Sexuality

Socio-cultural Factors
- Family, Vocation, Media

Precipitating Factors
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Perpetuating Factors

Binge Eating
Sociocultural Factors

1) Family

Can be protective by providing a forgiving environment or can magnify the cultural pressure for thinness and stigma against overweight/obesity

Know your child’s vulnerabilities

Be mindful of experiences that focus unduly on weight and shape and can potentially further damage child’s self esteem:

- Bullying (weight related)
- Dance (Ballet)
- Modeling
Sociocultural Factors cont.

2) Media

- Tends to glorify eating disorders among actors and others in the public eye
- Disseminates misinformation about eating disorders
- Fashion industry misrepresents body shapes and sizes “photoshopping”
The famous faces of eating disorders

Nicole Richie
Paula Abdul
Alanis Morissette
Victoria Beckham
Elton John

Oprah Winfrey
Sandra Dee
Princess Diana
Jane Fonda
Michael Jackson

Mary-Kate Olsen
Lady Gaga
Richard Simmons
Joan Rivers
Portia de Rossi
Teasing Out Genetic and Environmental Effects
Gene X Environment Interaction

Genotype

aabb

AABB

Environment

Phenotype

aabb

AABB
“GENES LOAD THE GUN, THE ENVIRONMENT PULLS THE TRIGGER”
Drug Treatment of Binge Eating Disorder

Goals of Pharmacotherapy in BED

• Efficacy in reducing binge eating
• Efficacy in maintaining abstinence from binge eating
• Efficacy in treating comorbid psychopathology, including weight loss/obesity
• Efficacy in treating the core disturbances in BED: affect regulation, self esteem, impulsivity
• Tolerability and safety
Randomized Placebo Controlled Trials of Medication in Binge Eating Disorder (N=27 placebo controlled RCTs)

- **Trycyclic antidepressants (3 RCTs):** Remission rates: 40% versus 22% for placebo; no significant weight loss. Total N = 95

- **SSRI (9 RCTs):** 4 of 7 showed drug > placebo for binge frequency; no significant weight loss

- **SNRI- 5 studies:** 3 of 4 showed drug > placebo for binge frequency. Total N = 453
Randomized Placebo Controlled Trials of Medication in Binge Eating Disorder

- Antiepileptics (5 RCTs): 3 of 5 showed drug > placebo for binge frequency. Total N= 639

- Weight Loss/Obesity Drugs (4 RCTs): only one study showed drug > placebo for binge frequency. Total N= 728
Current Status: Pharmacologic Treatments for Binge Eating Disorder

1. There is currently only one approved drug for BED in North America (lisdexamfetamine)

2. Current most RCTs other than for LDX are characterized by small samples, brief treatment and no long term F/U

3. Overall, pooling all studies, approximately 45% of subjects receiving medication achieved 100 remission from binge eating compared to 28% on placebo

4. Across all studies, mean weight loss was 3.4 kg greater on drug vs placebo.

6. No apparent advantage of drug added to CBT

8. No currently published trials of maintenance therapy
New Pharmacologic Treatment for Binge Eating Disorder- Why ADHD Drugs?

High rates of comorbidity of BED and ADHD:

- Obesity, BED, and ADHD commonly co-occur (30%), and symptoms of ADHD have been proposed to contribute to the disinhibited eating characterizing binge eating and weight gain.

- BED and ADHD are both characterized by dopamine deficiency and heightened reward sensitivity (“reward deficiency syndrome” and deficient tonic DA signalling) as well as impulsivity, both of which are associated with overeating.

- Psychostimulant medications, utilized to manage ADHD, target the dopamine system, and have been associated with increased behavioural regulation and decreased appetite and weight.
Lisdexamfetamine LDX (Vyvanse) in the Treatment of BED (McElroy et al 2015)*

Methodology:

- Multicenter, randomized, double blind, parallel group, forced dose (30mg, 50mg, 70mg/day) titration, placebo controlled clinical trial

- 30 sites, 255 subjects with BED treated for 11 weeks; 3 weeks titration and 8 weeks maintenance

- Exclusion criteria: any comorbid psychiatric condition

- Efficacy - change from baseline to endpoint in number of binge days/week

- Psychometric measures: CGI; TFEQ; YBOCS-BE; Impulsivity Scale (BIS); MADRAS; HAM –A; QOL (SF-12)

* JAMA Psychiatry 2015 :72 : 235-246
Lisdexamfetamine LDX (Vyvanse) in the Treatment of BED (McElroy et al 2015)*

Results:

- 50 mg and 70 mg groups significantly greater reduction in binge eating compared to 30mg and placebo treated groups.
- Cessation rates at 4 weeks: 70mg (50%) > 50mg (47%) > 30mg (35%) > Placebo (21%).
- Global improvement greater in 50mg and 70 mg groups compared to 30mg and placebo treated groups.
- 1.5% participants had serious treatment emergent adverse effects (consistent with findings in adults with ADHD treated with this drug).

Results of this and one other study lead to FDA approval of Vyvanse for BED (January 30, 2015)

*JAMA Psychiatry 2015:72:235-246*
Aim: To evaluate the therapeutic effect of long acting methylphenidate compared to CBT in patients with BED.

Hypotheses:

• Subjects who are randomized to receive long acting methylphenidate will demonstrate significant decrease in binge eating episode frequency and BED severity

• Pre-treatment ADHD symptom severity will be associated with a preferential treatment response to medication as compared to CBT

• Pre-treatment depression symptom severity will be associated with a preferential treatment response to CBT as compared to medication
RCT of Long Acting Methylphenidate Compared to CBT in the Treatment of BED

Protocol:

CBT treatment:
Participants randomly assigned to receive individual CBT will attend 16 50-minute appointments over the course of 12 weeks.

Medication:
Participants randomly assigned to receive long acting methylphenidate will attend weekly appointments with study psychiatrists for the first four weeks, and then biweekly appointments for the last eight weeks.

Dosage will be 18 mg/day, to be increased to 36 mg/day at week 2, 54 mg/day at week 3, and 72 mg/day at week 4. Dosage levels may be maintained or decreased to manage medication side effects.
RCT of Long Acting Methylphenidate Compared to CBT in the Treatment of BED

Measurements:

- ADHD Symptoms:
  - Wender-Utah Rating Scale
  - Conner’s Adult ADHD Scale

- Eating Symptoms: Eating Disorder Examination

- BMI

- Mood: Hamilton rating Scale for Depression;
  - Beck Depression Inventory

- Anxiety: Beck Anxiety Inventory

- Quality of Life: Quality of Life Inventory
Participants:

- \( N = 51 \) females randomized to drug, age 18-55 years (\( M = 28.44; SD = 7.64 \))
- DSM-5 diagnosis of BED
- 50% met criteria for a comorbid psychiatric condition
  - Mood Disorders
  - Anxiety Disorders
Long Acting Methylphenidate vs CBT in BED: Results

1. Patients receiving psychostimulants and CBT exhibited comparable clinical outcomes:
   • Decreases in binge frequency
   • Decreases in eating, shape, and weight concerns
   • Increases in quality of life
2. Compared to CBT, patients receiving psychostimulants exhibited greater decrease in cravings and BMI
3. Results provide support for efficacy of psychostimulant medication in BED
4. Dopamine dysregulation may represent a vulnerability for the disinhibition that characterizes BED, and which can be successfully treated by psychostimulant medication
Results: Binge Episodes

- Time: $F = 28.65$, $p < .01$, $\eta = .541$
- Time $\times$ Group: $F < .01$, $p = .93$

Estimated Marginal Means

Week 0

Week 6
Results: BMI

- Time: $F = 27.69, p < .01, \eta = .47$
- Time × Group: $F = 7.01, p = .01, \eta = .18$
Psychological Treatment for BED
The CB Model Of Eating Disorders

Behavioural Symptoms
(Dieting, Bingeing, etc.)

Extreme Concerns about Weight and Shape
Lack of Dietary Restraint

Deficits in Self-Concept; Self Image
Cognitive Behavior Therapy for Binge Eating Disorder ~ 3 Phases

Phase I focuses on normalizing eating behavior

• Self-monitoring for food intake, including binge episodes.

• Self-monitoring of thoughts and feelings associated with dysregulated eating.

• Specific interventions designed to normalize eating behavior, including learning greater dietary restraint.
Phase II focuses on dysfunctional thinking

- Cognitive restructuring directed at dysfunctional thoughts that are related to the development and maintenance of the eating disorder.
Phase III focuses on relapse prevention

• Strategies to consolidate and facilitate maintenance of changes after treatment ends.
Psychological Treatments for Binge Eating Disorder

- CBT remission rates 56-79%; 1 year follow up 40-50%
- IPT remission rates 50-70%; One year follow up 55%
  More acceptable to patients
- DBT remission rates up to 80%; One year follow up 55%
- Self Help – remission rates 65%; high-drop out rates
- Behavioral Weight Loss – not recommended

No significant loss of weight from psychological treatment
Thank you for your attention!
Questions/ Discussion