Modern Understanding of Eating Disorders
Objectives

- Understanding the concept of research domain criteria and how it will shape the understanding of psychiatric diagnoses, such as eating disorders, in the future.

- Understanding the cycle of eating disorders and the importance of state and trait criteria.

- Understand some of the current neurobiology of eating disorders and how it can help in understanding those that suffer with eating disorders.
Anorexia Nervosa

- Biogenetically mediated illness which usually strikes in adolescence.
- Restriction of energy intake relative to requirements, leading to a significant low body weight in the context of age, sex, developmental trajectory and physical health.
- Intense fear of gaining weight or becoming fat even though underweight.
- Body image disturbance or denial of seriousness of low body weight.
- Binge/purge or restricting subtypes.
- Severity criteria (Mild = BMI above 17, Moderate 16-16.99, Severe 15-15.99, Extreme <15)
**Bulimia Nervosa**

- Biogenetically mediated illness which usually strikes in adolescence.
- Recurrent episodes of binge eating.
- An episode of binge eating is characterized by both of the following:
  - (1) Eating, in a discrete period of time an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
  - (2) A sense of lack of control over eating during the episode.
- Recurrent inappropriate compensatory behavior in order to prevent weight gain.
- The binge eating and inappropriate compensatory behaviors both occur, on average, at least once a week for 3 months.
- Self-evaluation is unduly influenced by body shape and weight.
- Purging and non-purging types.
- Severity Criteria – episodes per week (Mild = 1-3; Moderate = 4-7; Severe = 8-13; Extreme = 14 or more)
Binge Eating Disorder

• Recurrent episodes of binge eating
• An episode of binge eating is characterized by both of the following:
  1. Eating, in a discrete period of time an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances.
  2. A sense of lack of control over eating during the episode.
• 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 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 afterward.
• Marked distress during the binge eating episode is present.
• Occurs, on average, at least once a week for 3 months.
Feeding or Eating Disorder Not Elsewhere Classified

Examples:
- Atypical anorexia, all criteria met except being within or above normal range.
- Low frequency Bulimia Nervosa or Binge Eating Disorder
- Purging Disorder
- Night Eating Syndrome
- Misconception that this is a less serious version of the illness.
Research Domain Criteria
The Future of Diagnosis and Treatment.

- Conceptualizes mental illnesses as brain disorders with pathology affecting brain circuits.
- These disordered brain circuits can be identified through electrophysiology, genetics, functional neuroimaging such as Positron Emission Tomography (PET) and Functional Magnetic Resonance Imaging (fMRI). Also there are new methods for quantifying connections in vivo (Protein analysis).
- Developed “Biosignatures” will augment current methods of obtaining clinical signs and symptoms to provide more accurate diagnosis and management.
- Examples of Diabetes and Syphilis in general medicine.
McGrath et al. JAMA psychiatry 2013 used Neuroimaging as a treatment selection biomarker for Major Depressive Disorder.

- Randomization to escitalopram or CBT for 12 weeks, with additional 12 weeks of combination therapy.
- Used remission, HAM-D score less than 7 as endpoint.
- Findings:
  - Anterior Insula hypometabolism relative to whole brain mean was associated with remission to CBT.
  - Anterior Insula hypermetabolism was associated with remission to escitalopram.

*Greater anticipatory anterior insula activation correlated positively in studies to alexithymia in recovered AN subjects.*
Eating Disorders Are A Stress/Anxiety Disorder

- Define mental illness currently by DSM V– definitions formulated from a collection of symptoms that generally meet criteria due to influence on normal function.
- Mental illness defined by lack of perspective available without the illness.
- Underlying traits are acted upon by hormonal, developmental and environmental pressures to create illness.
- Illness is sustained by states, both intrinsic and environmental which maintain the illness.
- Initial recovery must first correct state dependent factors and then work on alternative mechanisms of coping.
- Long term recovery focuses on minimizing the risk of a specific individual’s journey into illness.
The Cycle of Eating Disorders

Brain with Risks (Trait) + Environmental Factors = Cycle of Illness (State)
What traits place one at risk?

- Anorexia
  - Temperament and personality traits
    - Negative emotionality and low self-esteem
    - Perfectionism
    - Anxiety
    - Inhibition
    - Picky eating
    - Altered introceptive awareness
    - Obsessive-compulsive personality traits (especially order/symmetry)
    - High levels of reward dependence, low reward reactivity
    - Harm avoidance
• Anxious-Fearful-Harm Avoidant
• Neophobic - prefer the familiar
• Shy - social anxiety
• Orderly - inflexible
• Cognitively concrete - black and white
• Prefer small versus big
• Prefer ritualized simplicity to abstract complexity
• Slow to transition
• Cautious - low risk
• Serotonin Driven
What traits place one at risk?

- Bulimia
  - Temperament and personality traits 5,6
    - Negative emotionality and low self-esteem
    - Anxious/fearful
    - Obsessive-compulsive personality traits
    - Mood lability/Affect dysregulation
    - Impulsivity and “Hare” temperament
• Dramatic and Novelty Seeking
• Dramatic - Big
• Impulsive
• Chaotic
• Volatile moods/ reactive
• Easily bored
• Extroverted
• Argumentative/ oppositional
• Drawn to complexity, but overwhelmed by it
• Dopamine Driven
Concordance in twin studies (MZ) of Anorexia and Bulimia suggest there is a 50-80% genetic contribution to these disorders. Estimates similar to that found in Schizophrenia and Bipolar Disorder. Noted that AN and BN are cross transmitted in families. Similar to OCD there is subthreshold eating related behaviors in 1st degree family members. Search for endophenotypes such as set-shifting, low self-esteem, ego-syntonic vomiting, binging and obsessive compulsive traits. Future will point to genetic influences on specific neural circuits. Genetics load the gun, environment pulls the trigger.
Gene-Environment Interaction

Probability of depression at age 26; stressful life events between 21-26
Genetic variation of short allele in Promoter region for Serotonin Transport

Caspi et al 8
What Exposures Affect ED Patients?

- Puberty, Puberty, Puberty
  - Estrogen changes (serotonin, cortisol and developmental changes in Frontal and limbic circuits).
  - Increased complexity.
  - Body weight increase and distribution.
  - Prime time of onset of other mental illnesses such as anxiety and depression.
- Psychological and environmental influences
  - Increased propensity for sexual abuse with sexual maturity.
  - Family trauma/stressors.
  - Separation and individuation in high stress environment.
  - A fat phobic culture that holds thinness as ideal of success.
Why Don’t Anorexics Stop?

• The brain, once malnourished is now state-dependent to continue the illness.
  ▪ Minnesota Starvation Experiment (1945) – Male conscientious objectors 9
    • Increased depression, somatization, preoccupation with food, social withdrawal and isolation.
    • Cognitive changes included decline in concentration and judgment.
  • Regression to pre-pubertal gonadal function.
  • Simplification of neurochemistry – excess limbic serotonin.
  • Starvation causes increased Corticotrophin Releasing Hormone (CRH) known to cause dysphoria, hyperactivity and decreased feeding behavior.
Cycle of Serotonin, Estrogen and Anorexia

Excess Serotonin in Limbic system leads to mood and error detection.

Dieting → Decreased Serotonin/Estrogen (Improved Mood)

Worsening Mood

Increased CRH with weight loss

INTERVENTION = WEIGHT RESTORATION (Increased Serotonin and Estrogen)
Why Don’t Bulimics Stop?

- Disconnect between ideal body image and urges.

- Positive reinforcement from undoing behaviors.
  - Undoing behaviors reduce dysphoria.

- Neurobiological model of intermittent excessive behavior.
  - Common to binge eating, abhorrent sexual conduct, drug abuse, alcoholism and excessive gambling.
  - Dopamine reward systems drive excessive eating.
  - Addictions model of illness and treatment. Primary goal is to stop binge/purge cycle.
The Cycle of Eating Disorders

Brain with Risks (Trait) + Environmental Factors = Cycle of Illness (State)
So… we have a group of patients who have:

- Elevated rates of somatic preoccupation and gut sensations
- Overcome powerful visceral homeostatic signaling systems
- Heightened level of fear and anxious rumination
- Enduring anxiety-related personality traits and co-morbid anxiety disorders

**Visceral Interoception**
Floatation as novel therapy for eating disorders

- Directly tied to Interoception and anxiety relief.
- Lose all exteroceptive sense of the body.
- Different than sensory depravation.
- No previous studies in Eating Disorders.
Visual body image change – pre float

Actual body

Current body

Ideal body
Visual body image change – post float

Actual body  Current body  Ideal body


