Motivation and the Regulation of Internal States
Chapter 6

Motivation and homeostasis
Hunger: a complex drive
Obesity
Anorexia, Bulimia, and Binge Eating Disorder
Motivation and Homeostasis

• **Motivation**
  • “To set in motion”
  • Factors that initiate, sustain, and direct behaviors.

• **Theoretical Approaches to Motivation**
  • **Instinct:**
    • Automatic and unlearned behavior
    • Occurs in all members of a species
  • **Drive theory:** The body maintains homeostasis—equilibrium—in its systems.
Motivation and Homeostasis

• Theoretical Approaches to Motivation
  • Drive theory: Is concerned primarily with explaining physical systems like hunger or thirst.
  • Incentive theory: People motivated by external stimuli
  • Arousal theory: People behave to satisfy a certain level of sensation

• Challenges to drive theory have driven emphasis on drives as states of the brain rather than conditions of tissues
  • Homeostatic Drive: A “control system” maintains conditions around a set point.
  • When conditions do not equal the set point, the organism will behave to return the drive to the set point.
Motivation and Homeostasis

Figure 6.2: Selected Nuclei of the Hypothalamus

- **Temperature Regulation**
  - **Ectotherms** cannot regulate body temperature internally using energy reserves...(Lizard / Snake)
  - **Endotherms** maintain a constant internal temp by changing metabolism, constricting/expanding blood vessels, or moving to warmer/cooler location [Mammal]
  - **Preoptic area** of the hypothalamus receives temperature signals from the blood/skin.

SOURCE: Adapted from Nieuwenhuys, Voogd, & Van Huijzen, 1988
Motivation and Homeostasis

Figure 6.3: Thirst Control Signals & Brain Centers

- Thirst
  - **Osmotic thirst**: when fluid in cells drop, cells take water from bloodstream
    - OVLT of hypothalamus signals **median preoptic nucleus** to trigger drinking
  - **Hypovolemic thirst**: when blood volume drops
    - Kidneys release renin, increasing **angiotensin II**.
    - Subfornical organ then signals **median preoptic nucleus**
Hunger: A Complex Drive
The Role of Taste.

- **Hunger**
  - Taste buds on tongue papillae detect five primary categories of chemicals.
    - **Sweet**: carbohydrates
    - **Salty**: ions for neural transmission
    - **Sour**: spoiled or rotten food
    - **Bitter**: toxic chemicals
    - **Umami**: protein content.
  - Some evidence that taste preference and sensitivity are developed early in life

- Signals travel to the **insula** (the primary gustatory cortex), and to the **nucleus of the solitary tract (NST)**.

- Rats with lesions in ventromedial hypothalamus eat uncontrollably and become very obese
Hunger: A Complex Drive
Sensory-Specific Satiety: Varying the Choices

• **Sensory-specific satiety**
  • Food is less appealing the more you eat, encouraging variation in choices
  • Area NST of the medulla.

• **Learned taste aversion**
  • Avoiding foods associated with illness or poor nutrition.

• **Learned taste preference**
  • Preference for the flavor of a food that contains a needed nutrient.
  • Wisdom of the body will have us naturally choosing a balanced diet, however...
  • Often counteracted by tasty, high-calorie foods
Hunger: A Complex Drive
Digestion & Two Phases of Metabolism. Figure 6.6: The Digestive System

- **Mouth**
  - Saliva starts breakdown of starches into **glucose**

- **Stomach**
  - **Hydrochloric acid** and **pepsin** mixes with food to digest proteins into **amino acids**.

- **Small Intestine**
  - **Duodenum** is where the rest of digestion takes place.
  - Fats transformed into **fatty acids** and **glycerol** by bile.

- **Hepatic portal vein** transports products to the liver
Hunger: A Complex Drive

Figure 6.7: Summary of the Absorptive and Fasting Phases

Requires capacity to store reserves, allocate them during fasting period, and monitor when reserves get low.

- **Absorptive Phase**
  - Glucose increases → Parasympathetic activation → Pancreas secretes insulin
  - Glucose enters body cells.
  - Glucose stored in liver and muscles as glycogen.
  - Fat stored in adipose cells as triglycerides.

- **Fasting Phase**
  - Glucose decreases → Sympathetic activation → Pancreas secretes glucagon
  - Glycogen transformed to glucose (for brain).
  - Stored fat released as:
    - fatty acids (for body), and
    - glycerol (for brain, after conversion to glucose).
Hunger: A Complex Drive

Figure 6.8: Hunger Control Signals and Brain Centers

- **Hunger from low**
  - Glucose *(Glucoprivic)*
  - Fatty Acids *(Lipoprivic)*
  - **Ghrelin** is released (excess may lead to stubborn obesity)

- **Nucleus of solitary tract (NST) of medulla**

- **Arcuate Nucleus**
  - Monitors nutrient levels
  - Releases NPY and AgRP which excite...

- **Paraventricular nucleus (PVN) and Lateral hypothalamus (LH)** trigger eating

Green pathways
Hunger: A Complex Drive

Figure 6.8: Hunger Control Signals and Brain Centers

- Stomach stretch receptors stimulated
- When food enters duodenum, intestines release
  - Cholecystokinin (CCK) inhibits NST and Lateral hypothalamus (LH)
  - Peptide YY$_{3-36}$ (PYY) inhibits arcuate nucleus & helps us conserve energy
- Eating slows
- Leptin (released by fat cells) inhibits hunger on a long-term basis
### Hunger: A Complex Drive

#### Table 6.1: Summary of Feeding Signals

<table>
<thead>
<tr>
<th>Stimuli</th>
<th>Signal Source</th>
<th>Pathway</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Start meals</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Glucose, fatty acids</td>
<td>Liver, as nutrients in blood are depleted</td>
<td>Vagus nerve &gt; NST &gt; Arcuate nucleus</td>
</tr>
<tr>
<td>2. Glucose (brain)</td>
<td>Receptors near 4(^{th}) ventricle</td>
<td>Medulla to Arcuate nucleus</td>
</tr>
<tr>
<td>3. Ghrelin</td>
<td>Stomach, during fasting</td>
<td>Blood stream &gt; Arcuate nucleus</td>
</tr>
<tr>
<td><strong>End meals</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Stomach volume</td>
<td>Stomach: stretch receptors</td>
<td>Vagus nerve &gt; NST &gt; Arcuate nucleus</td>
</tr>
<tr>
<td>5. CCK</td>
<td>Stomach, Intestines</td>
<td>Vagus &gt; NST; Blood &gt; brain</td>
</tr>
<tr>
<td><strong>Long term</strong></td>
<td></td>
<td></td>
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<tr>
<td>6. PYY</td>
<td>Intestines</td>
<td>Blood &gt; Arcuate nucleus; Inhibits NPY Neurons</td>
</tr>
<tr>
<td>7. Leptin</td>
<td>Fat cells</td>
<td></td>
</tr>
<tr>
<td>8. Insulin</td>
<td>Pancreas</td>
<td></td>
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</table>
## Obesity

**Figure 6.14: Body Mass Calculation Chart (for adults)**

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<th>Height in Feet and Inches</th>
<th>Weight in Pounds</th>
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<td>6'4&quot;</td>
<td>12 13 13 14 15 15 16 16 17 18 18 19 19 20 21 21 22 23 23 24 24 25</td>
</tr>
</tbody>
</table>

Obesity

Health Effects. Figure 6.13: Underweight and Obesity According to Country

- Correlated with higher risk of
  - Diabetes (type 2), Heart disease, High Blood Pressure, Stroke, Colon Cancer, Reduced lifespan
- Brain changes: reduced temporal lobe, cognitive decline, Alzheimer’s risk
- Doubled since 1980 in U.S., global epidemic
- Myths about Obesity
  - Lack of impulse control, poor eating styles, temptation to eat.

Obesity
Contributions of Heredity and Environment.
Figure 6.15: Correlations of Body Mass Index Among Twins
Figure 6.16: \textit{ob/ob} mouse

- Heritability
  - Obesity: 50-90%
  - BMR: 40%

- Genes
  - Obesity (\textit{ob})
  - Diabetes (\textit{db})
  - FTO gene (A allele)

- Environment
  - Epigenetic characteristics: gene expression changes due to environment (AgRP).

Obesity

Obesity and Reduced Metabolism

• **Basal metabolic rate (BMR):**
  • Energy required to fuel the brain and body
  • 75% of energy expenditure at rest.
  • Average: 1800 calories/day

• **Set Point** defended by shifting metabolism (increasing or decreasing energy expenditures)
  • 33% of women on restricted diet who didn’t lose weight had lower BMRs.
  • Prolonged weight change can shift the set point.
Obesity
Treating Obesity. Dietary Restriction versus Medication

• Dietary restriction is effective, especially when coupled with exercise (which increases BMR).
  • Formerly obese women- did they exercise afterwards?
    • YES: 90% kept the weight off
    • NO: 33% kept the weight off
  • Half of all calories consumed in U.S. were from carbs, and another 1/3 were from fats (as of year 2000)
• Medication is not as effective.
  • Drugs that work through increasing serotonin, *leptin and insulin, or through decreased fat absorption have been promising but do not work for all patients
  • All have significant side effects
Obesity

Treat as an Addiction
- Obese people share several characteristics with addicts.
- Reduced D2 receptors and prefrontal metabolism.
- Peptides that induce eating target dopamine neurons.

Anti-addiction drugs are showing effectiveness in weight loss.

Obesity

Treating Obesity. Figure 6.21: Gastric Bypass Surgery

- An option for the morbidly obese.
  - Weight loss averages 25% after 10 years, compared to 5%-10% with dieting and, most often, relapse within a year.
  - Reduces ghrelin and increases PYY and GLP-1, reducing hunger.
- Benefits include reduced mortality and many health improvements.
Anorexia, Bulimia, & Binge Eating Disorder

Figure 6.24: French Model Isabelle Caro in Late Stages of Anorexia

- **Anorexia nervosa**: The “starving disease.”
  - Restrictors - reduce food intake and exercise excessively to maintain weight
  - Hunger battle: NPY, ghrelin high; leptin low.

- **Bulimia nervosa**:
  - Binge and purge cycles, but usually normal weight
  - **Eat large meals, then vomiting food back up**
  - High relapse rates

- **Binge-Eating Disorder**
  - Eat large meals
  - Usually high weight
Anorexia, Bulimia, & Binge Eating Disorder
Environmental and Genetic Contributions

- Environmental contributions
  - Cultural emphasis on thinness, as seen in the Fiji study.
  - The incidence is higher in females, who experience more pressure.
- Genetic influence is suggested by:
  - Identical twins 3x more concordance than in fraternal twins
  - Heritability: 56% for anorexia, 54-83% for Bulimia, 45% for binge eating disorder
  - Adolescent stress, hormones, and dieting may produce epigenetic changes in genes
- Co-morbidity with obsessive-compulsive disorder (anorexia) and depression (bulimia).
Anorexia, Bulimia, & Binge Eating Disorder
Roles of Serotonin, Dopamine, and Cannabinoids

- Reduced serotonin activity
  - Bulimics
  - SSRI antidepressants increase serotonin, reducing bingeing and purging and lowers relapse rates

- Imbalanced ratio of serotonin receptors
  - Anorexics and bulimics have impaired executive control over emotional responses

- Low activity in cannabinoid, dopamine reward systems
  - Lack of enjoyment of food as well as other life pleasures.
  - Eating increases dopamine levels, and viewing pictures of food stimulates cannabinoid receptors in insular cortex of anorexics and bulimics
  - Eating also increases food-related anxiety. Therefore, food restriction, while not pleasurable, reduces anxiety.