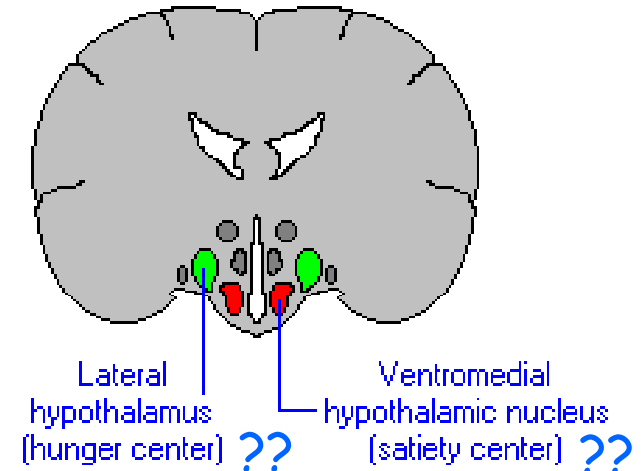


# Homeostasis, Feeding (Ch.13) III

- Neural mechanisms of feeding
  - Lateral Hypothalamus
  - Arcuate Nucleus of Hypothalamus
  - Interactions between body-based satiety signals and brain
  - Bypassing body signals
  - Serotonin and feeding
- When good feeding behaviour goes bad
  - Obesity
  - For next week: start reading Chapter 14 (sleep)

# Neural Basis of Hunger and Satiety (II)

- **Lateral Hypothalamus: Hunger center?**
  - Lesion this nucleus, animals stop eating (**aphagia**)
  - **Problems:** again, effects not permanent: force feed rats for a week, they eventually start eating again
  - **Reinterpretation:** LH lesions cause wide range of sensory and motor disturbances, including decreased appetite. Animals have problems with eating, but not lack of hunger.
- **Control of hunger and satiety is distributed across many brain regions**
  - Other hypothalamic subregions, as well as amygdala, frontal cortex are also involved

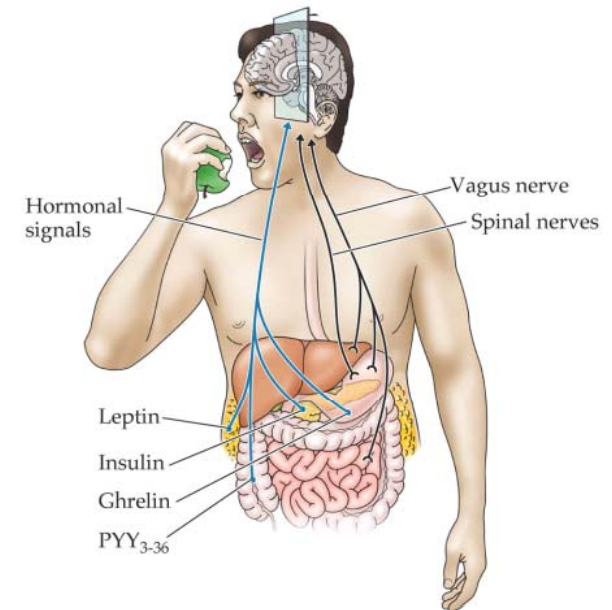
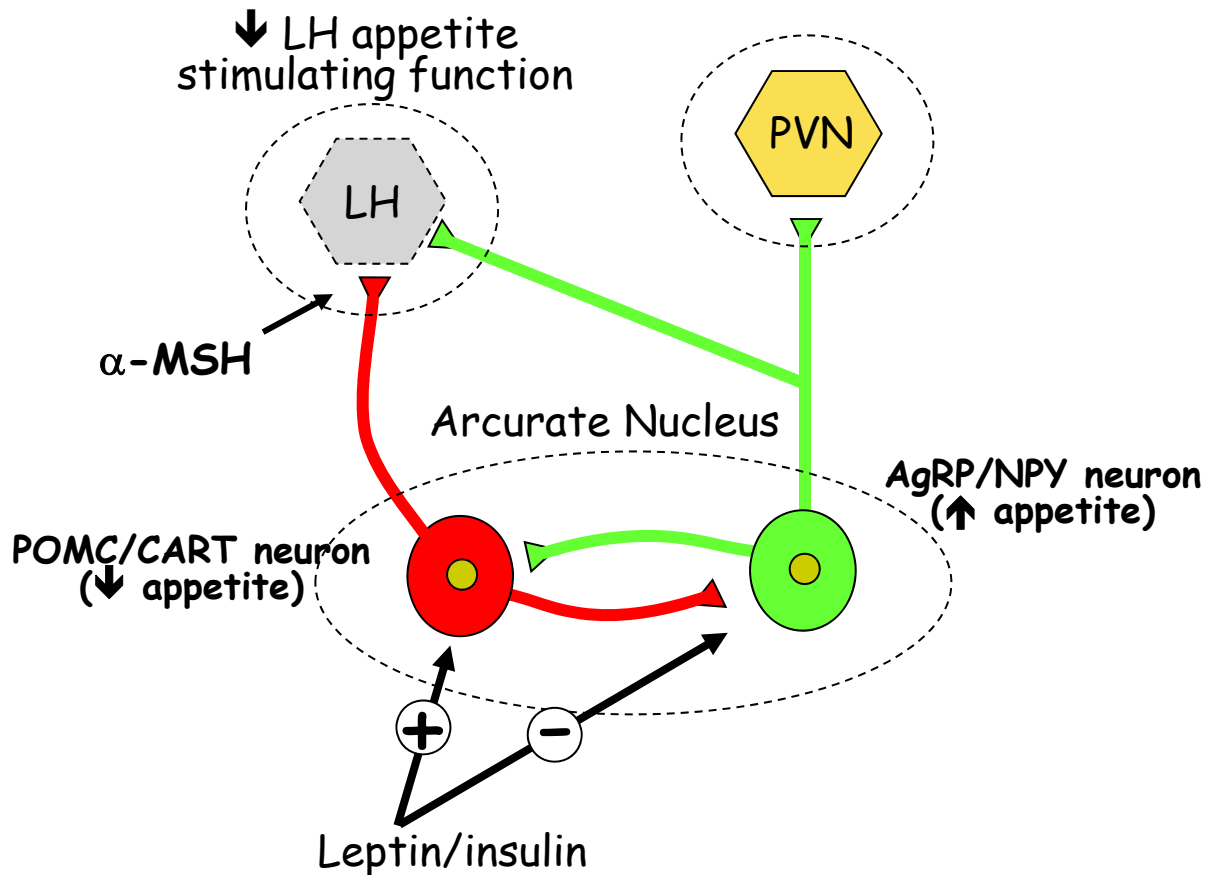


# Neural Basis of Hunger and Satiety (III)

- **Arcuate Nucleus of Hypothalamus:** First pass appetite control center
  - Satiety and hunger signals from body interact with this nucleus to regulate feeding behaviour
  - 4 main ones:
    - **Pancreas:** Insulin (↓ feeding)      **Intestines:** PYY<sub>3-36</sub> (↓ feeding)
    - **Fat Cells:** Leptin (↓ feeding)      **Stomach:** Ghrelin (↑ feeding)
  - Each of these activate different types of neurons in Arcuate
- **Neuropeptide Y (NPY) & agouti-related peptide (AgRP)**
  - These cells ↑ appetite and ↓ metabolism
- **Pro-opiomelanocortin (POMC) & Cocaine/amphetamine regulated transcript (CART)**
  - These cells ↓ appetite and ↑ metabolism

# Arcuate Neural Circuitry and Appetite (I)

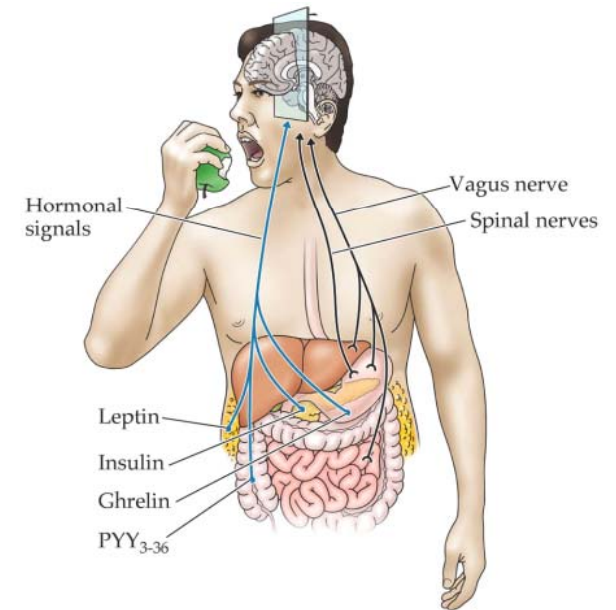
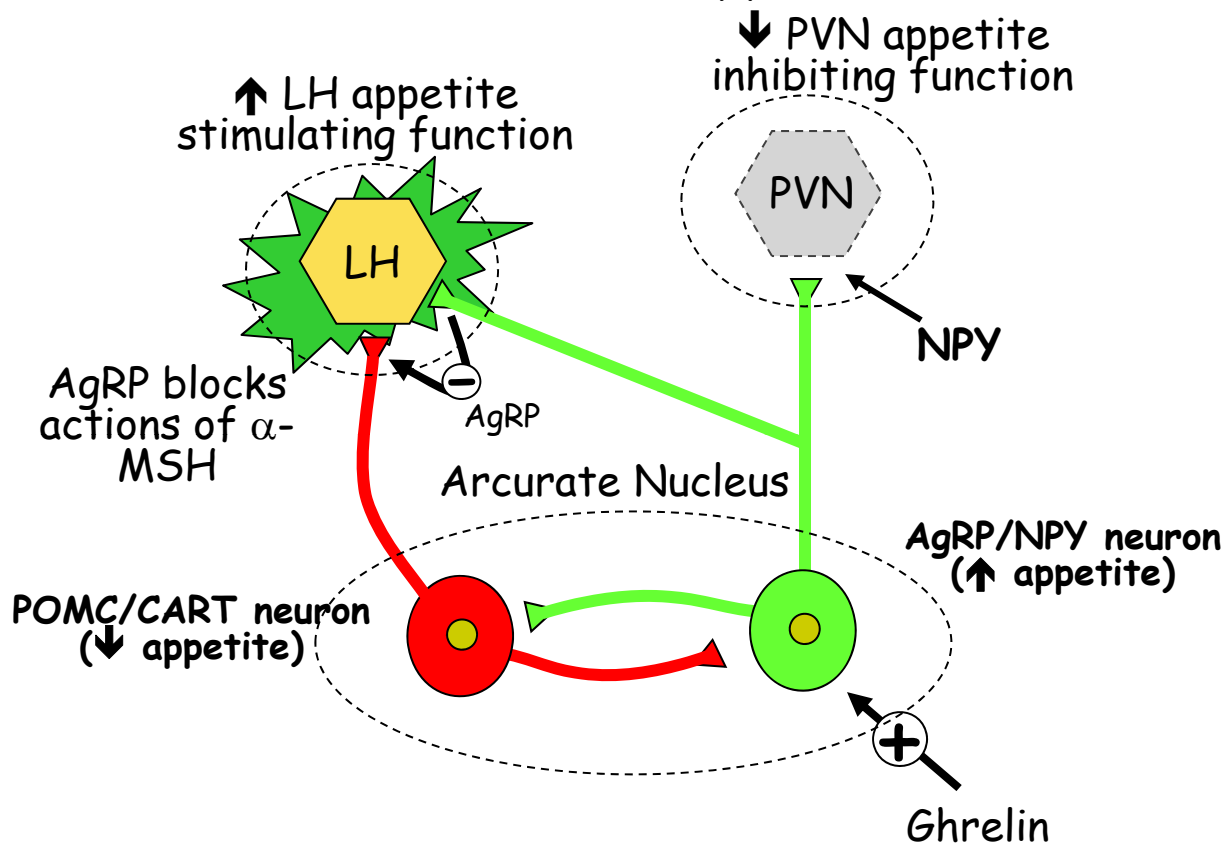
(Long term appetite control)



- Leptin & Insulin work as long term modulators of appetite
- Activate POMC/CART neurons
- Inhibit AgRP/NPY neurons
- POMC/CART neurons inhibit LH via  $\alpha$ -melanocyte stimulating hormone

# Arcuate Neural Circuitry and Appetite (II)

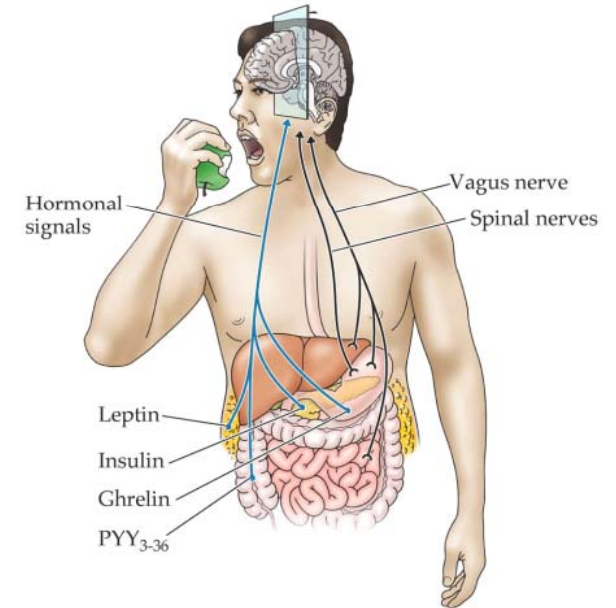
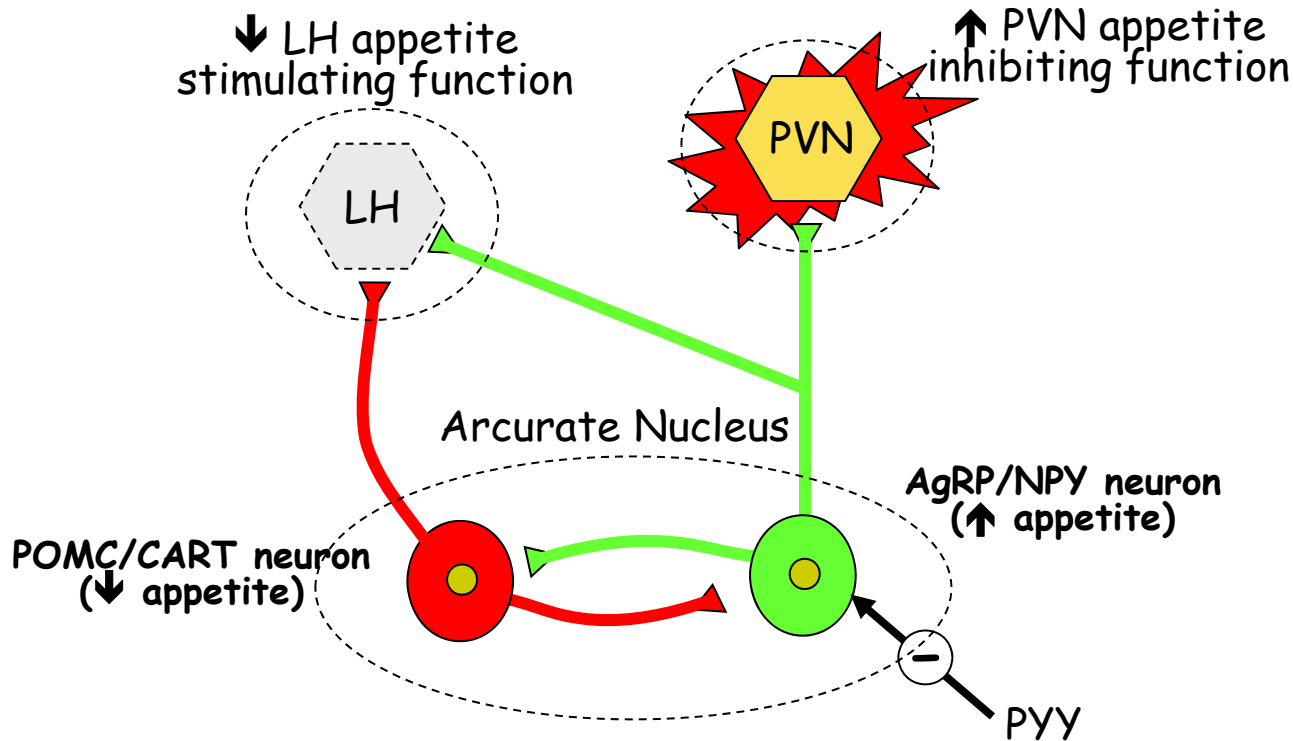
(Short term appetite **increase** during fasting)



- Ghrelin released by stomach when empty, stimulates AgRP/NPY neurons
- Activation of these neurons
  - Inhibits PVN cells via NPY
  - Removes inhibition of LH cells via  $\alpha$ -MSH, leads to activation of LH

# Arcuate Neural Circuitry and Appetite (III)

(Short term appetite **decrease** after meal)

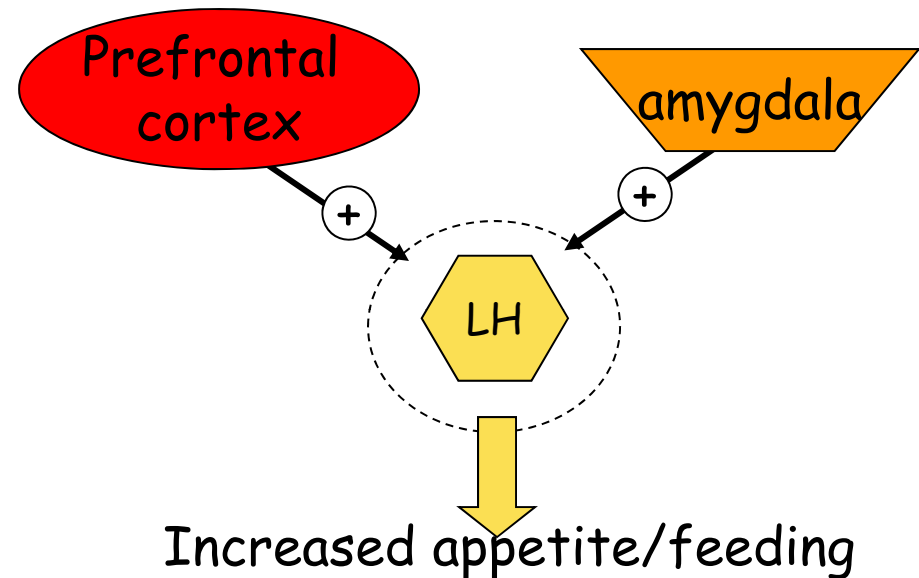


- PYY (from intestines in response to meal), inhibits AgRP/NPY neurons
- Inhibition of these neurons
  - Increases PVN activity
  - Allows  $\alpha$ -MSH to inhibit LH
- Other signals from body, and inputs from other parts of brain can regulated activity of these cells

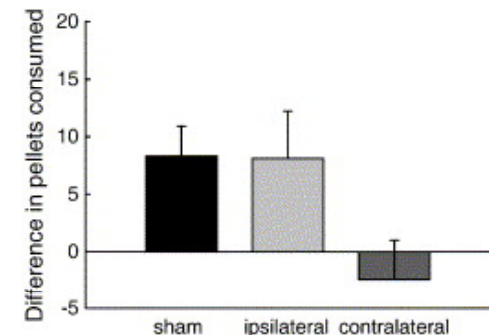
# Bypassing the Hypothalamic Feeding Circuit

- The entire satiety part of the circuit can be bypassed by cues associated with feeding
- Rats are fed repeatedly with an auditory cue that predicts food
- Let rats free feed until sated
- Play cue in sated rats, they eat more (Pavlovian conditioned feeding), but...
- **Lesions to the prefrontal cortex or the amygdala, or disconnection of the amygdala-LH pathway abolishes this effect.**
- **NORMAL** feeding patterns unaffected, only cue-induced feeding disrupted
- Other cortical and subcortical brain regions feed into the LH, can bypass the effect of satiety signals from the body

These regions are activated by cues associated with feeding



Feeding induced by cue



Amygdala-LH disconnection

# Neurochemistry of Hunger and Satiety

- **Serotonin (5-HT):** a main satiety signal in brain

- From an evolutionary perspective, one of the oldest neurotransmitters, involved in many basic functions

- 5-HT neurons in raphe nucleus sends diffuse projections to multiple cortical and subcortical areas

- 5-HT agonists or releasers (Prozac) in humans and animals:

- ↓ feeding, even with cafeteria diets

- ↓ amount of food consumed/meal, not number of meals

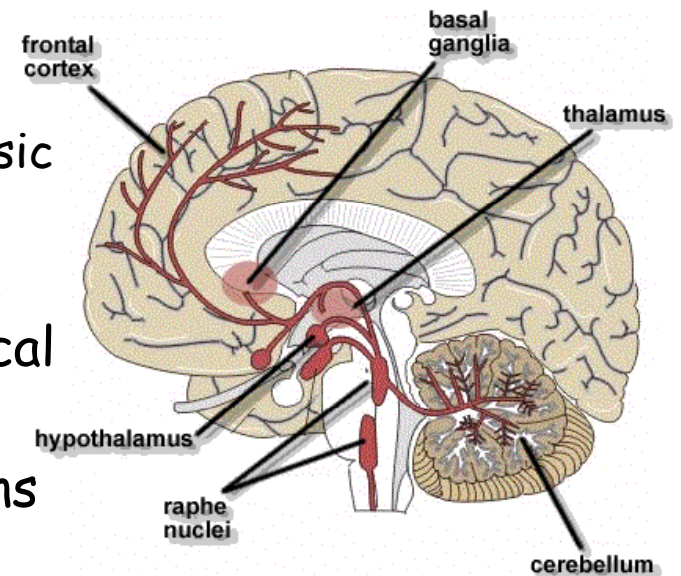
- Shift food preference away from fatty foods

- suggest that 5-HT acts as short term satiety signals associated with meal consumption

- Brain regions:** 5-HT inhibits release of NPY in the PVN of hypothalamus, which then disinhibits PVN neurons to promote satiety

- \*Drugs such as fenfluramine have been used to treat obesity with some success: yet heart disease side effect caused it to be taken off market

The serotonergic system consists of ascending axons from cell bodies in the raphe nuclei



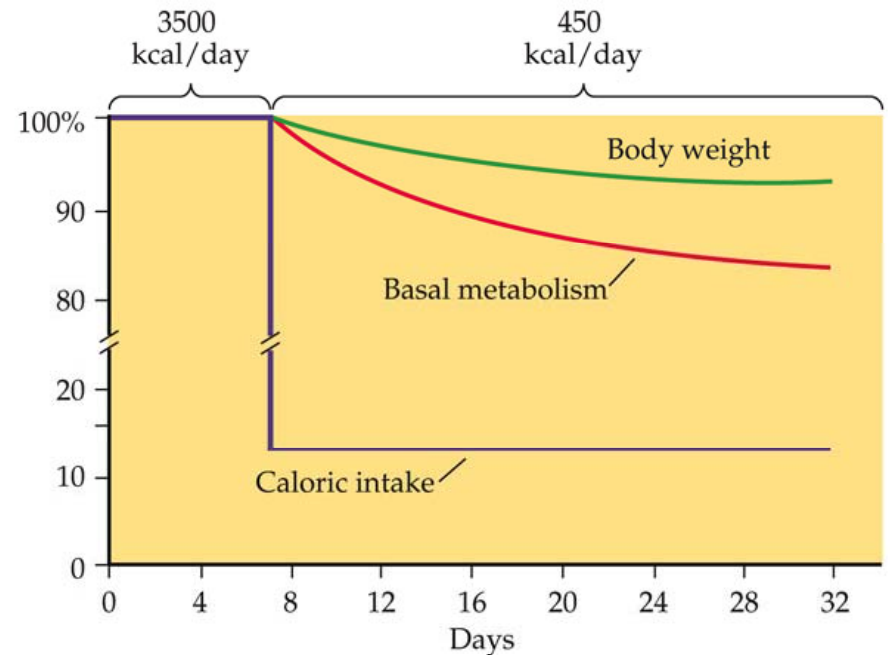
# Obesity

- **What is it?** Characterized by excessive adipose tissue.
- Adult males = >25%, Females = >30% fat content.
- 10-12% of Canadian adults are obese
- Body Mass Index: (mass/height<sup>2</sup>): Obesity = BMI >30
  - (note, this method assumes minimal muscle, so weightlifters (or your professor) shouldn't use this as an estimate.)
- **Causes:** U.S. twin study: environmental and genetic factors equally responsible for obesity.
- Obese subjects have larger insulin response to sight, sound and smell of food (cephalic phase).
- **Treatments:**
  - Exercise and proper diet.
  - Low calorie diets (often give immediate results, then but then weight comes back on)
  - Appetite suppressants, surgical procedures (drastic, last resort)



# Why is it so freakin' hard to lose weight?

- Our metabolisms are geared to prepare for times of starvation
  - Reduce food intake, metabolism slows down
- Many diets work, but for most, you put the weight back on after you go off of it
- Exercise helps, but you have to do a lot of it to make a dent in the calories in/calories out equation
  - 1 pound of fat = 4100 calories!!
- We tend to eat more when we're more active
- NOT ALL CARBS ARE EQUAL (glycemic index)
- Reducing caloric intake (50-75% of ad lib levels) prolongs life in humans and animals
- Our current environment is "pathological", not our weight problems
  - Many times, radical lifestyle change is needed to lose weight



Biological Psychology 5e, Figure 13.17

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