- Hunger and satiety signals arise from periphery and each reach the brain.

- Eating and drinking are evolutionary.

Control mechanisms do not require the cortex.
  * Cannot seek food
  * Can eat, can respond to hunger and thirst
  * Can differentiate different kinds of food
  * Can vomit/reject bad food: postrema is intact.

**Lateral hypothalamus**
  * Control hunger
  * Lesion - decreases eating and drinking and body weight
  * Stimulation - increase eating and drinking
  * Block glutamate transmission -> decreases food intake.

2 types of neurons producing:
  * Melanin concentrating hormone (MCH)
  * Food deprivation increase MCH
  * Satiety decrease MCH

Simulation of MCH neuron:
  * Appetite inducing, decrease metabolic rate, increase motivation of movement.

***Stomach (Ghrelin)/ Brain stem (liver) → Arcuate (NPY) → lateral (MCH/ Orexin) → increase eating decrease metabolism

**How do we stop eating? Two parallel inhibitory pathways**

- Leptin (from fat cells) inhibits the NPY neuron on the arcuate nucleus.

- Cocaine and amphetamine Regulated Transcript (CART) and (a-MSH) neurons inhibit and MCH Orexin neurons via the MC-4R receptor.

**Leptin**

- Hereditary leptin deficient (OB-like) in humans. Genetic deficit in the production of Leptin

- Leptin no longer used in weight loss diets: leptin resistance.

**Ingestive Behaviors: Obesity**
Increasing problem → diabetes

- Type 1 - deficiency in insulin production (requires injections)
- Type 2 – deficiency in insulin receptors (treated with pills)

Average energy consumption
  * Muscles -20%
  * Brain -20%
  * Heat/ digestion - 60%

***Body weight ← → energy stores - energy

Definition of obesity – more than 20% of normal weight
Body mass index (BMI) - body fat based on height and weight
  * 25-30% (overweight)
  * 30-40% (obese)
  * 40 and above (morbidly obese).

Why are people overweight??
  * Kind of foods eating
  * On average 2500 Kcals, but only 300Kcal out…

Obesity

Biological causes of Obesity:

- Metabolic disorder (more calories in then out). Due to Fast metabolism. In general, not due to deficiency in Leptin production.

Genetic factors: Different metabolic rates
  * Twins studies (tested with high/low calorie diets).
  * Epidemiological studies (study of populations).

- High metabolic rates → increases availability of calories → spent if needed, stored if not (hence obesity).
- Low metabolic rates → no opportunity for fat storage (no obesity).

Mouse: obesity is due to leptin deficit.
Human: no evidence for leptin production deficits, but…
*Deficit in leptin transport through the BBB
*Deficit in sensitivity of leptin receptors (MC4 receptors, age related)

-In humans, high fat diets inherently decrease satiety signals.

_Treatments_
*Exercises
*Wire in jaw
*Gastric bypass
*Intestinal bypass
*Gastric bubble

**Conclusion**
*Eat slowly
*Eat regularly
*Exercise
*Don’t eat at night

**Ingestive behaviors: Anorexia Nervosa**

*Definition:*
*Refusal to maintain weight over the lowest weight considered normal for age/ height.
*Intense fear of gaining weight or becoming fat (even when overweight)
*In women: three consecutive missed menstrual periods without pregnancy.

-80% of cases are young women (age 15-24%) 15% death rate.

-Can be due to too much exercise (too much exercise decrease hunger signals). Restricting food results in increase physical activity (and weight loss)

-Respond physiologically correctly to food → not a loss of interest in foods.
-Normally…6 months starvation has psychological consequences… OCD?
-Genetic factors (evidence by twin studies)
-Brain imbalance of NE, S-HT and NPY. No effective drug treatment.

_Treatment:_
*Psychotherapy
(In the video showed in class) …
*Psychologist tried to get patients back to normal weight before they left the treatment center.
*Addressed psychological issues
  * Many patients have been sexually abused.