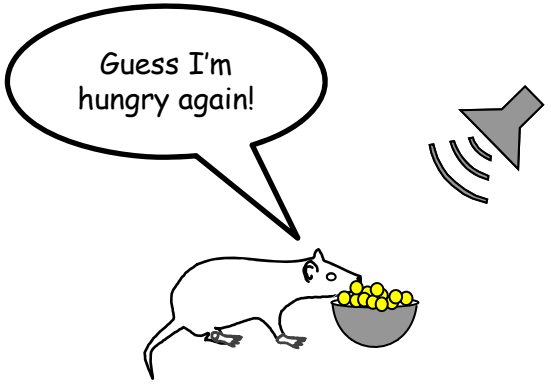
Homeostasis, Feeding (Ch. 13) II

- Multiple factors influence feeding behaviour
- Physiological mechanisms underlying hunger and satiety

Factors that influence when we eat (I)

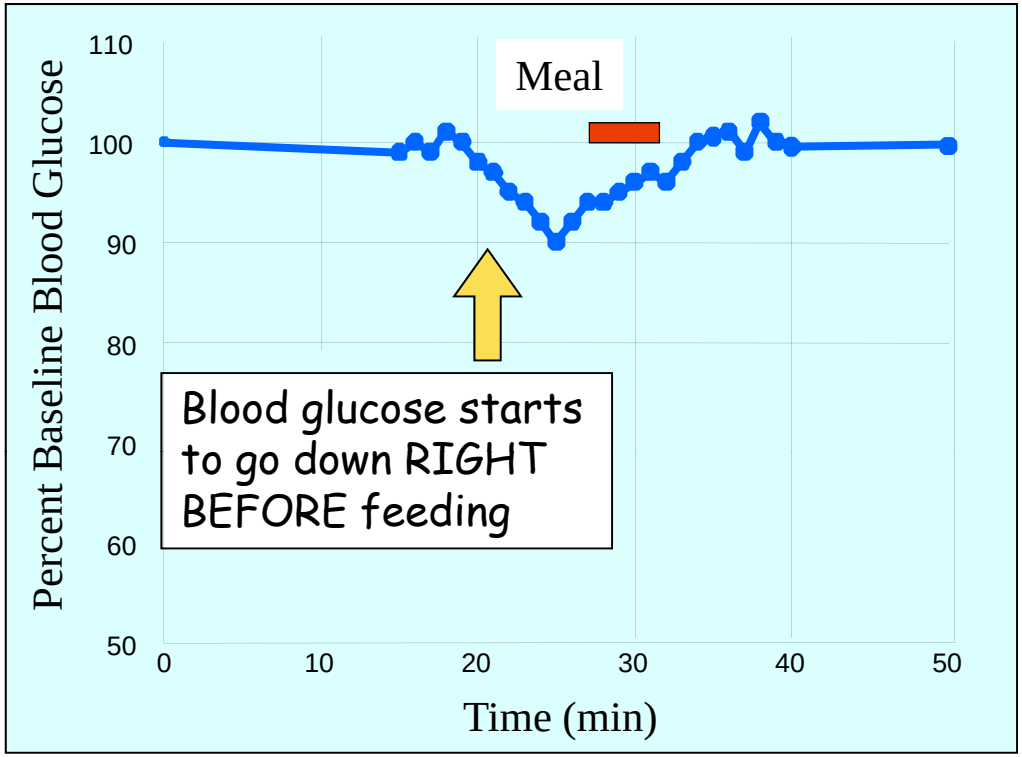
- **Pavlovian Conditioning:** Environment cues normally associated with eating can elicit hunger and feeding
  - Hunger can be caused by expectation of food, not energy deficit
  - Give rats 6 meals/day at irregular intervals; with each meal, rat gets a tone.
  - On test days, food is continuously available: rats will eat in response to tone, even if they had just eaten recently.



- **Premeal Hunger**: Time of day that one usually eats can trigger hunger
  - Can be viewed as a special type of Pavlovian conditioning
  - Eating is a stressful event on the body
  - Eating at regular times can condition the body to prepare itself for incoming food (**cephalic phase**)
  - "Hunger pangs" are the body getting ready for incoming food, not the body craving food

Factors that influence when we eat (II)

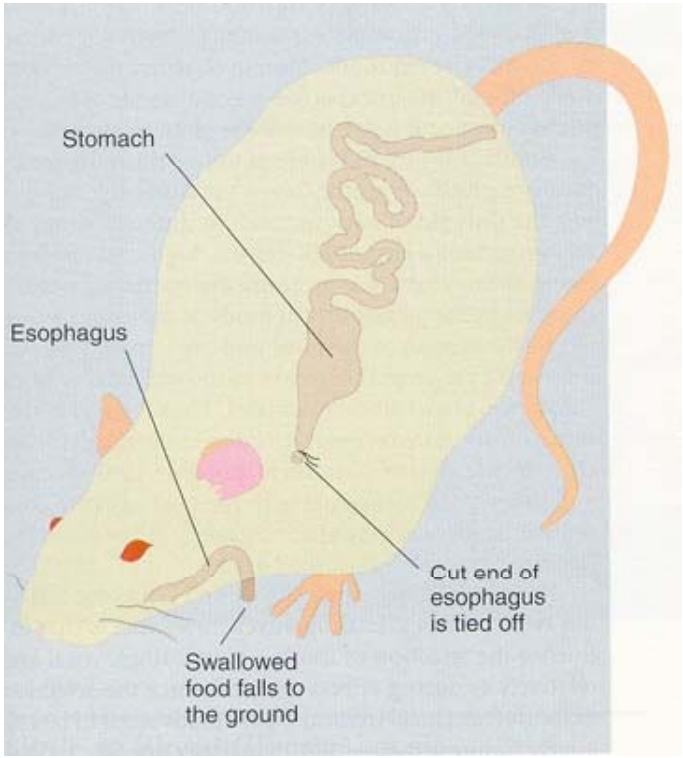
- Experiment: rats provided unlimited food and water
- Blood glucose levels monitored constantly
- Glucose levels remain constant through day
- A drop in blood glucose (~10%) occurs only before feeding is initiated by rat
- Unlikely that drop in glucose is responsible for feeding b/c:

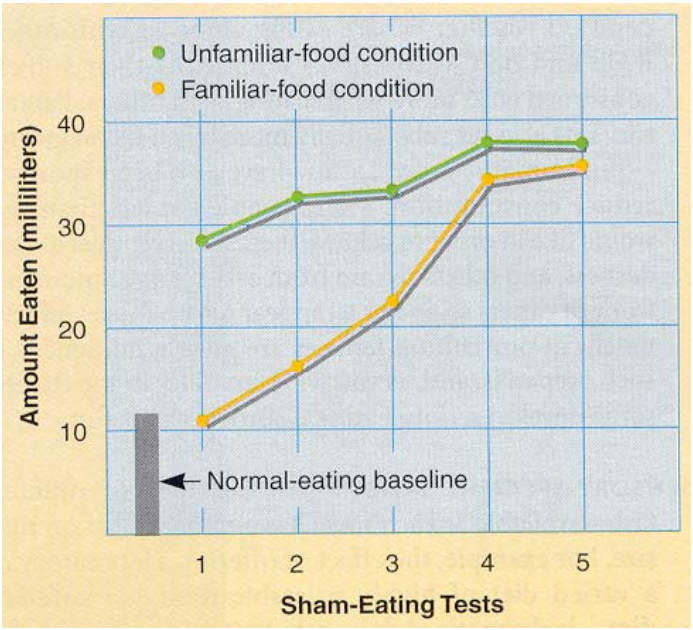


- No meal: glucose levels go back to their previous homeostatic levels.
- Decline may be related to animals' INTENTION to eat, not the other way around. Change was preceded by increase in blood insulin, so drop may have been actively produced rather than a decline in reserves.
- Changes in glucose levels may contribute to feelings of hunger, but does not seem to control eating behavior

Factors that influence satiety (I)

- **Previous experience about the nutritive value of a certain food can influence satiety**
- **Sham eating experiment**: Food is chewed and swallowed and then passes out of the body. Rats given either normal lab chow (which they are used to) or novel food
- With normal lab chow, rats start off eating same amount as before surgery. Novel food, they eat more.





Not until 4th meal that rats are eating 3 times as much as normal. Even then they stop feeding.

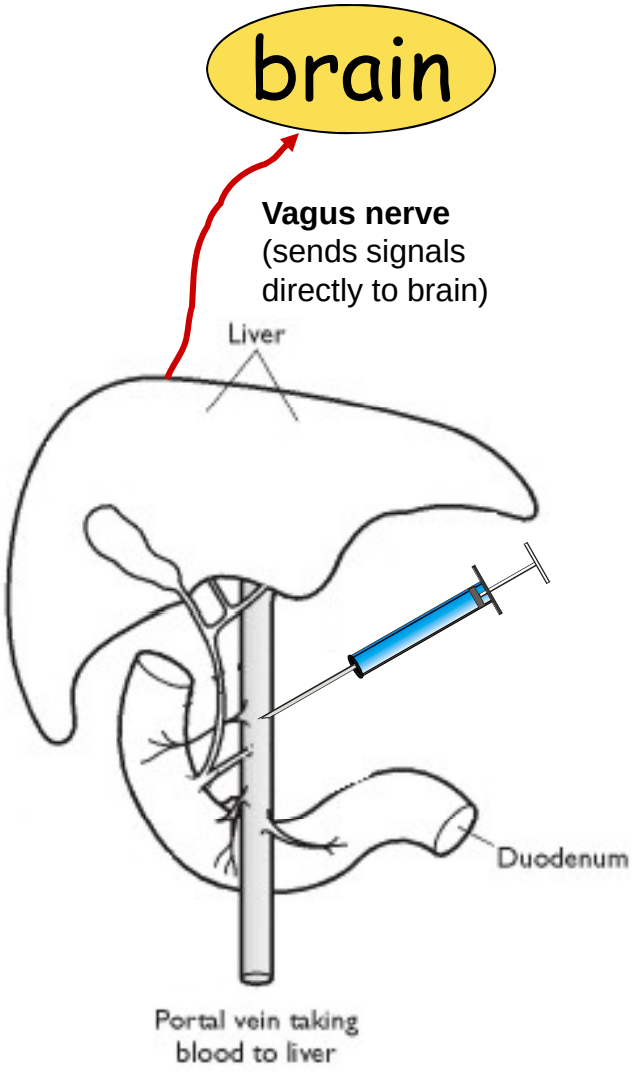
Factors that influence satiety (II)

- **Social Influences:** Humans and rats will eat more if they are in groups rather than alone
  - Other social pressures may decrease eating (desire to be slim)

- **Sensory Specific Satiety**: Humans and animals take in more calories if they are given varied (cafeteria, buffet) diet
  - Satiety is taste specific: new taste = more consumption
  - Encourages animals to consume varied diets and to take advantage when different foods are abundant
  - Study: humans asked to rate palatability of 8 foods, then given one of foods for meal
  - When asked again to rate same 8 foods, one they just ate got lower rating: when given new meal right after, they ate more.

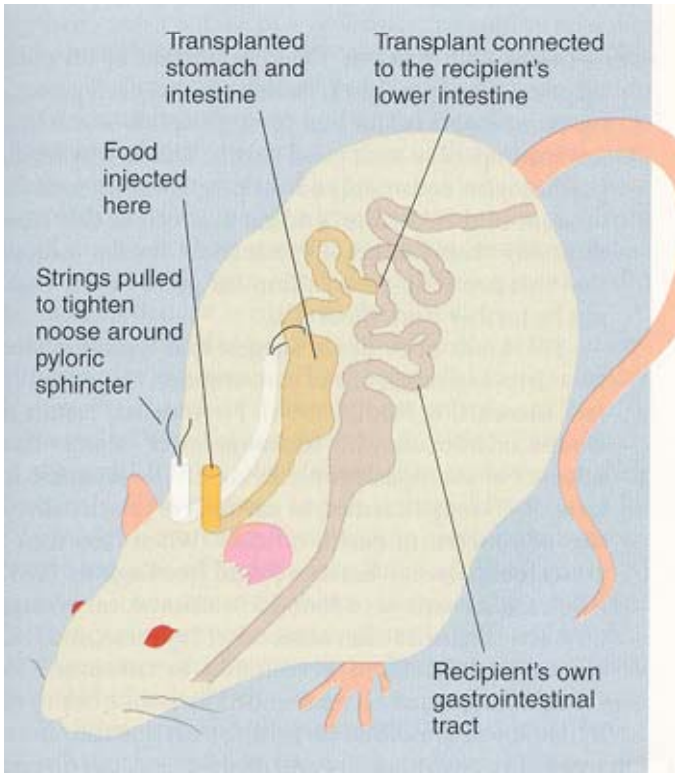
Physiology of Hunger and Satiety (I)

- How does the brain get signals about nutrients in the body?
- Liver can signal brain about what's going on in the bloodstream via vagus nerve
  - Liver receives blood from small intestine and has detectors for glucose and fatty acids
- Experiment: inject chemicals that "trick" system to act as if glucose/fat levels are low
  - 2-deoxyglucose (2-DG) = compete with normal glucose for absorption, but doesn't activate glucose detectors
  - Methyl palmoxirate = disrupt metabolism of fatty acids
- Inject directly into vein that goes from small intestine to liver = immediate increase in feeding
- Cut vagus nerve = abolish this effect
- **Note**: brain also has receptors for glucose (but not fats) in a number of regions. Infusing 2-DG in certain regions also stimulates feeding



Physiology of Hunger and Satiety (II)

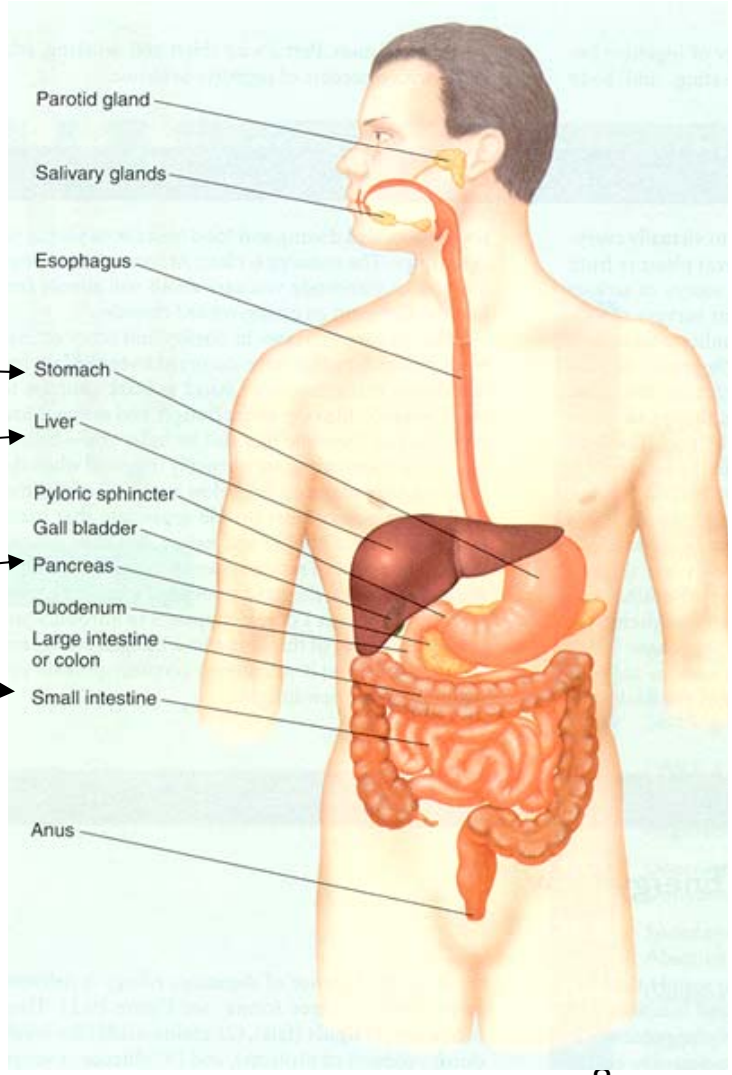
- **Satiety peptides:** transplant a 2nd stomach in rats, tie it off from rest of intestinal tract
- Inject food directly into 2nd stomach, but don't allow it to fully digest
- Rats decrease their feeding in proportion to caloric content and volume of food in 2nd stomach



- **Conclusion**: Gut releases hormones in bloodstream that stimulate receptors in multiple brain areas to decrease feeding.

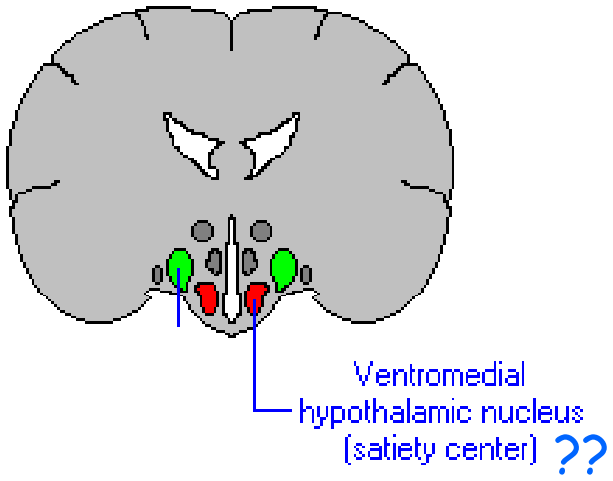
Satiety/Hunger Signals: Body to Brain

- We stop eating long before nutrients get into bloodstream
- Body uses multiple hormones to signal brain to start/stop eating
- Food in gut, glucose in bloodstream can initiate signals to stop eating before food is fully digested
  - **From Stomach**: CCK, bombesin, somatostatin
  - **From Liver**: detects changes in blood glucose, direct input to brain via vagus nerve (non-hormonal)
  - **From Pancreas**: Insulin
  - **From Intestines**: PYY_{3-36}
  - **From Fat Cells:** Leptin, gives brain continuous feedback on body's energy stores
- Other peptides can stimulate feeding
  - **From Stomach:** Ghrelin, levels remain high during fasting, drop during meal (short term)



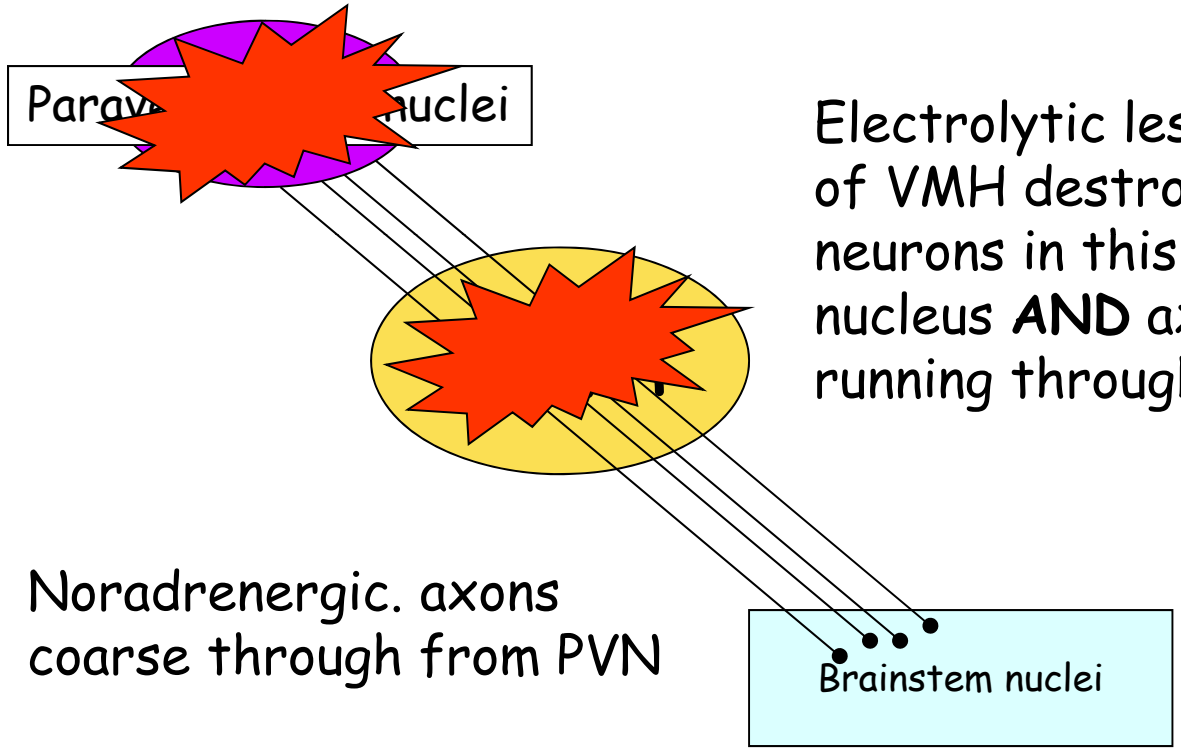
Neural Basis of Hunger and Satiety (I)

- **Ventromedial Hypothalamus:**
  Satiety center?
  - Lesion this nucleus, animals become obese: starts with massive consumption to get to new weight **(dynamic phase)** then they maintain that weight **(static phase)**
  - **Problems:** give regular chow instead of high fat diet, minimal weight gain; become finicky eaters
  - Effects are not permanent
  - **Reinterpretation**: VMH regulates energy metabolism not eating.
  - VMH lesions increase insulin levels (↑ lipogenesis) while decreasing lipolysis (breakdown of fat into usable forms)



VMH lesions

-Neurons in VMH may not be what causes effects. These lesions also destroy noradrenergic axons projecting from the paraventricular nuclei of the hypothalamus. Lesions of these fibers alone also produce hyperphagia and obesity.



Electrolytic lesion of VMH destroys neurons in this nucleus **AND** axons running through

Noradrenergic. axons coarse through from PVN

- Destroy PVN or axons = same obesity and hyperphagia as VMH lesions
- **VMH not the only satiety center**