Homeostasis, Feeding (Ch. 13) I

- Why we eat,
  - what we eat, how we make use of it
- Glucose Regulation
  - Insulin
- Older and contemporary theories on feeding
- Multiple factors that influence feeding behaviour
  - Factors that influence ***what*** *and* ***when*** we eat

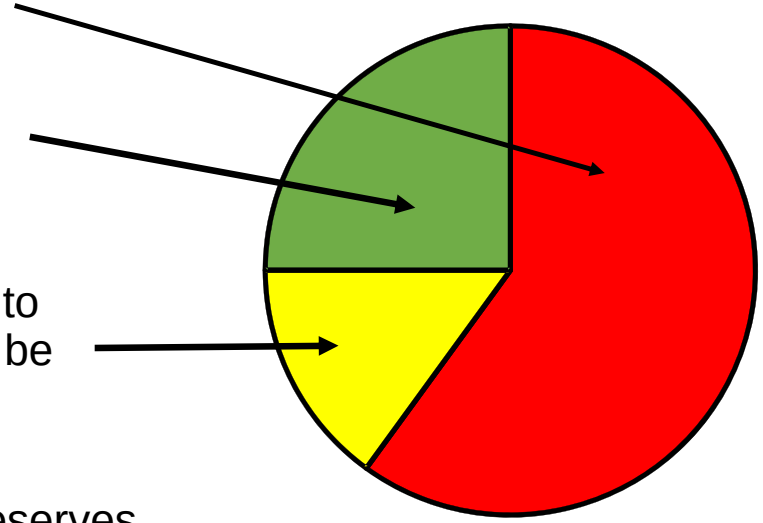
- NOTE: You will NOT be tested on pages dealing with Thermoregulation or Fluid Regulation
- ZOOM Q & As start this week- Wed, 2:30-3:30 pm!

Why do we have to eat?

There are many reasons why we eat, but the most fundamental answer is **we eat to maintain energy levels of the body**

- Body uses energy in 3 primary ways:

- 1) **Basal metabolism**: ~60% of energy usage maintains body heat and other resting functions
  - Can vary as a function of body weight
- 2) **Active behavioural processes:** ~25% is for behaviours other than rest
  - Can vary greatly depending on activity levels
- 3) **Digestion of food:** ~15% of energy usage is to processes food, break it down into molecules to be used by body
  - Can vary by type of food
- Any remainder typically gets stored as energy reserves



Note: textbook numbers are different, as it refers to lab animals

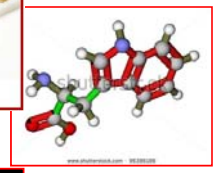
Basic Nutrients

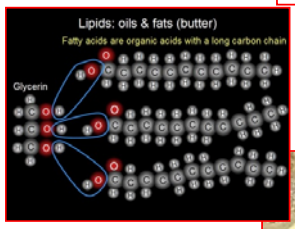
- **<u>Carbohydrates</u>**: ~4 kcal per gram, **glucose** is primary fuel of the body, all other carbs get converted to glucose
  - Storable form of carbohydrate is called **glycogen**: stored in liver and muscles

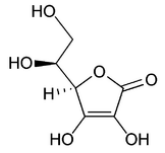
- **<u>Amino Acids:</u>** ~4 kcal per gram. Comes from proteins, basic building blocks for all cells
  - 20 amino acids, 9 cannot be produced by body = **essential amino acids**
  - Amino acids can be converted to glucose

- **<u>Lipids (Fats):</u>** ~9 kcal per gram. Long term energy source.
  - Fats can be converted to **free fatty acids** as alternate energy source for *most* cells of the body

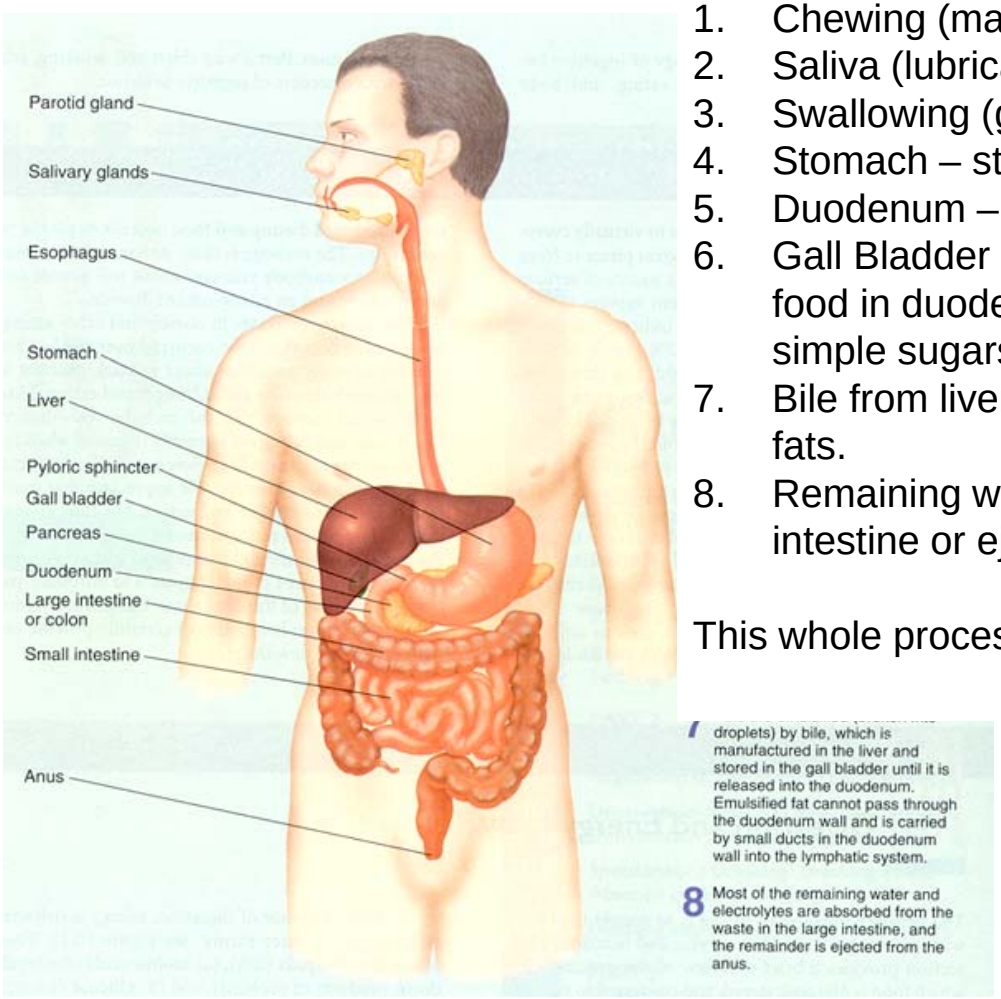
- **<u>Vitamins and Minerals:</u>** needed to assist in bodily functions
  - (digestion, cell building, regular homeostasis etc)







Steps of Digestion

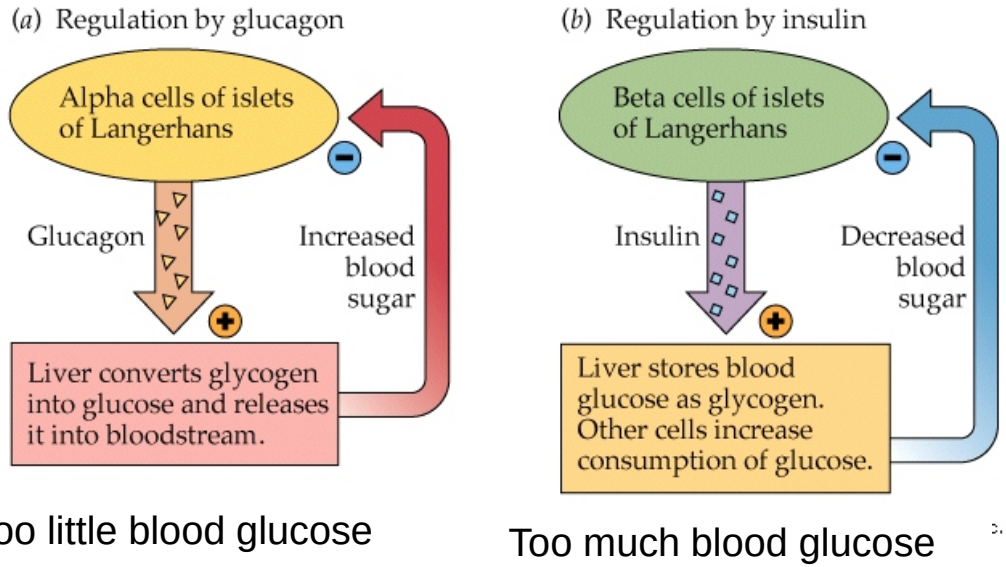


1. Chewing (mastication)
2. Saliva (lubrication)
3. Swallowing (getting there)
4. Stomach – storage and breakdown (HCl, Pepsin)
5. Duodenum – absorption
6. Gall Bladder and Pancreas fluids further break down food in duodenum (proteins → amino acids; starch → simple sugars).
7. Bile from liver (stored in gall bladder) breaks down fats.
8. Remaining water and electrolytes absorbed by large intestine or ejected via the anus.

This whole process takes 18-24 hrs!

Glucose regulation (I)

- **PANCREAS**: regulates blood glucose levels in bloodstream
- Two main hormones: **glucagon** and **insulin**
  - **Glucagon** converts glycogen (stored carbs) into glucose
  - Insulin has multiple functions



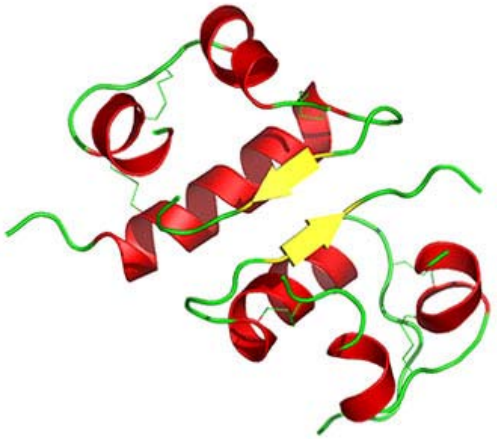
Too little blood glucose

Too much blood glucose

- Release of both hormones controlled by negative feedback system

Glucose regulation (II)

➢ Two primary actions of INSULIN



- **1)** promotes use of glucose as primary energy source for body
  - Most cells of body need insulin to get glucose in cells
  - **Brain** is one exception: it uses glucose without need for insulin. Only energy source that brain can use is glucose.

- **2)** Converts bloodborne fuels to storable forms
  - Glucose→ glycogen (muscles/liver), glucose & fatty acids→ adipose tissue (body fat), amino acids→ protein (muscles)

•Aside from negative feedback, other mechanisms control insulin release

- ✓ Brain (via vagus nerve): sight/smell/taste (THOUGHT) of food can trigger insulin release before food hits gut ***(cephalic phase)***
- ✓ Other hormones in bloodstream released by gut during ***digestive phase***
- ✓ *Nutrients entering bloodstream* ***(absorptive phase)***

Diabetes Mellitus

(Aka: Type 1 (juvenile-onset) diabetes)

- Pancreas stops producing insulin, excess glucose in blood stream
- Brain cannot use it all, and cells of the body CANNOT use glucose w/out insulin, start using fatty acids
  - Fatty acids not the best energy source for cells, and cells cannot make use of glucose

- Thus, when left untreated, diabetes can lead to more eating that doesn't satisfy hunger and paradoxical weight loss

Glucose and Insulin as Satiety Signals?

- Lower animal's insulin level, it becomes hungry and eats large meal: give *moderate* level of insulin, eats much less. **Satiety signal?**
- **Not exactly:** give **LARGE** amount of insulin, that converts most glucose to fat, less glucose in bloodstream.
- Brain detects glucose deficit, initiates hunger: animals will now eat a large meal just the same.

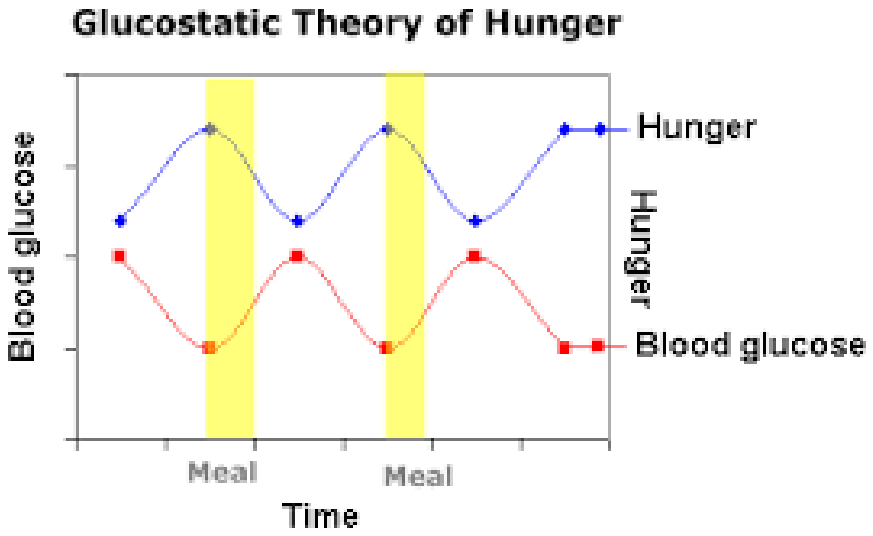
- **Glucose as a satiety signal?**
- **Not exactly:** untreated diabetes leaves a lot of glucose in bloodstream, but also increases hunger
- Under normal conditions, blood glucose levels can stay relatively stable for hours-days, but we still get hungry
- **MULTIPLE SIGNALS in addition to glucose and insulin regulate hunger and satiety.**

Why do we get hungry? (I)

- Set Point theory:
  - It is tempting to attribute hunger as an energy deficit, with a negative feedback system maintaining homeostasis. Low energy = hunger.
  - Genetics, environment play a role in establishing set point

Two basic types of theories

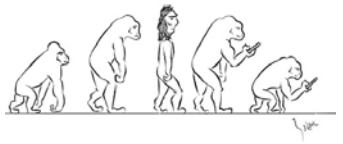
Glucostatic or **Lipostatic** Theories: Eating is controlled by deviations from a hypothetical **blood glucose** or **body fat** set-points, respectively.



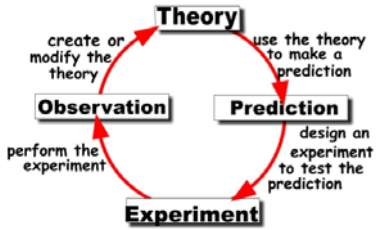
Why do we get hungry? (II)

➢ Problems with set-point theories:

- **<u>Evolution argues against it</u>**:
  - Food availability was inconsistent and unpredictable until recently.
  - Best strategy was to eat large quantities when food was available to store calories for periods when its less available.



- **<u>Major predictions of set-point theories fail when tested.</u>**
  - Drinking high glucose/caloric drinks before meals doesn't reduce eating significantly.
  - Reductions in blood glucose levels (insulin injection) or body fat levels (starvation) that increase eating rarely occur normally



- **<u>Set-point theories ignore factors that stimulate eating</u>**
  - Taste of food (eg; dessert)
  - Social factors (going out with friends even though you're not hungry).

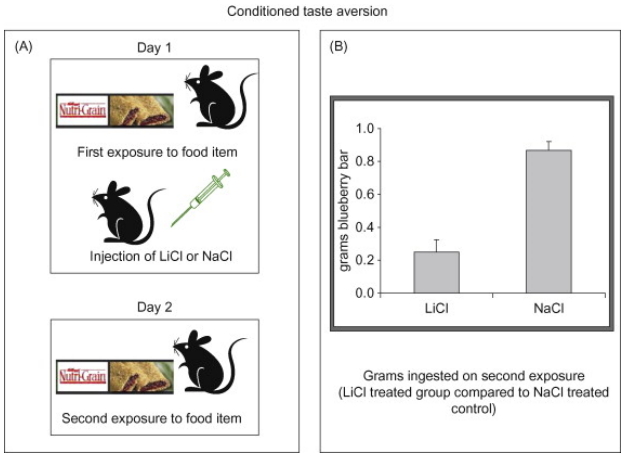
Why do we get hungry? (III)

- **Positive-Incentive Theory:**

- The anticipated pleasure of eating is the main factor controlling feeding.
- We have evolved to crave food not because we are in a deficit, but b/c we like it (like sexual behaviour)
- No one factor is singled out as “the” motivation to eat.

Factors that determine *what* we eat

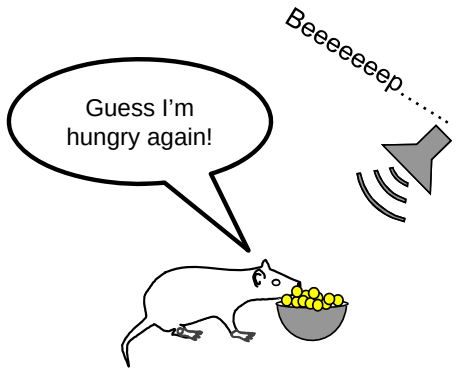
- **Taste preferences/aversions**: Some tastes have innate high incentive values (sweet, salty, fatty foods)
- Others are learned from experience or social situations
  - give animal novel food, then give toxin to make it sick, animal learns to avoid food (**conditioned taste aversion)**



- **Learning to eat vitamins/minerals**: animals can learn to choose foods that contain the vitamins/ minerals they are lacking.
  - Give rats on a vitamin B1-deficient diet a choice between "low B1" and "high B1" foods, rat eat more B1 high food
  - Give rat 10 choices, only one is "high B1", they cannot learn: too many choices makes it difficult to learn

Factors that influence *when* we eat (I)

- **Pavlovian Conditioning:** Environment cues associated with eating can elicit hunger/feeding
  - Hunger can be caused by expectation of food
  - ***Study****:* 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.

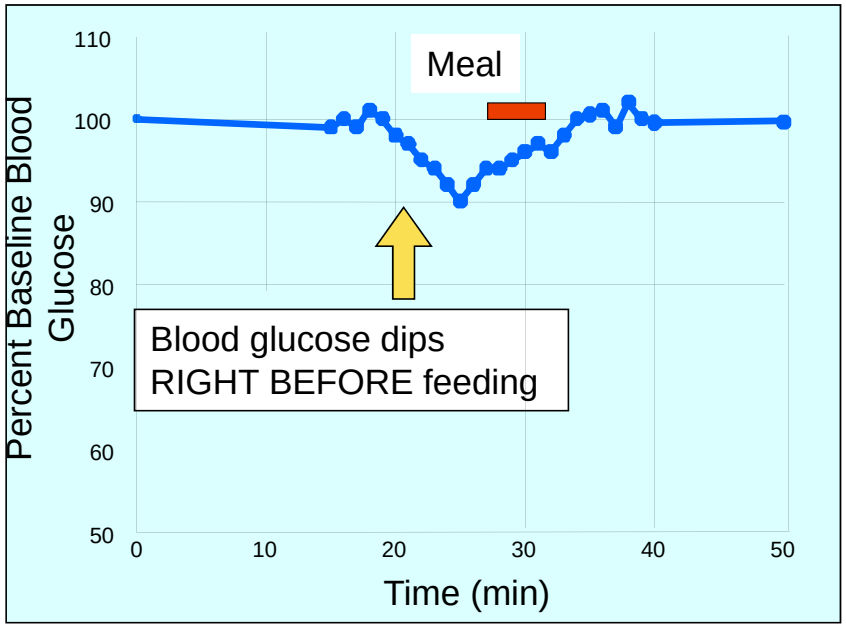


Factors that influence *when* we eat (II)

- **Pre-meal Hunger**: Time of day that one usually eats can trigger hunger
  - A special type of Pavlovian conditioning
  - Eating at regular times can condition brain/body to prepare itself for incoming food
  - "Hunger pangs" are the body getting ready for incoming food, not the body craving food

Changes in blood glucose around feeding time

- ***Study***: provide rats w/unlimited food- monitor glucose levels
- Blood glucose levels remain constant through day *except…*
- Glucose levels drop ~10% before feeding is initiated
- **However,** it is unlikely that drop in glucose is directly responsible for feeding because:
- **If food taken away (no meal)** = glucose levels return to previous homeostatic levels in about 10-15 min.



- Decline may be related to INTENTION to eat (not other way around). Drop in blood glucose is preceded by increased insulin, so drop may have been actively produced (not a decline in "energy reserves"**.**

- Changes in glucose levels may contribute to feelings of hunger, but does not seem to be the main controller of eating behavior