1. Digestion & Energy Flow

Cephalic (“head”) Phase:
Thinking about/anticipation/expectation of eating
Often triggered by sight, smell, even sound of food
**Insulin** levels rise (secreted by pancreas)
Glucagon levels low

Absorptive Phase:
Actual eating (food enters the GI tract)
**Insulin** levels continue to be high, glucagons levels low
Thus, Blood glucose is the major source of energy
Used by body cells (w/ insulin), by neurons (w/o insulin)
Excess glucose is converted to glycogen and fat
Amino acids are converted into proteins
Glycogen (.5%) is stored in liver & muscles, fat (85%) in fat cells, and proteins (14.5%) in muscles

Fasting Phase:
Not eating, no food in GI tract
Insulin levels are low
**Glucagon** levels are high (secreted by pancreas)
Thus, Fats are converted to free fatty acids & used as source of energy
Body’s primary source of energy in fasting phase
Free fatty acids are converted into ketones & used by muscles
Glycogen and proteins converted to glucose & used
Neurons can use glucose without presence of insulin
Neurons can also use ketones if glucose low (in starvation)

So…in cephalic/absorptive phases, body is getting “rid” of all that extra glucose (by storing it) and in the fasting phase, body is using up the stored glucose/amino acids
All of this is designed to keep body’s circulating levels of glucose and stored calories in some sort of even/consistent state
So…why are 50-60% of USA citizens storing too much!
2. **Theories of Hunger & Eating**

   **Set Point Theory**
   Now rejected in face of newer research

   **Positive Incentive Theory**
   Animals (including humans) are drawn to eat by the anticipated pleasure of eating, by the **positive-incentive value** of food/eating.
   In that sense, eating is no different from other behaviors that our brain has been selected to perceive as “positive” (e.g. sex).
   Why so selected? For survival value, was evolutionarily useful under the conditions in which humans evolved…
   Are those conditions still present in the environment now…?
   Now that we are neurologically wired to find both food and sex pleasurable, are we now able to acquire too much of a good thing for our own good? Maybe…..
   How does this all play out in what, when, & how much we eat?

3. **Factors that Determine What, When & How Much to Eat**

   **a. What to Eat**
   That’s easy…anything that tastes **sweet, salty or fatty**!
   Sweet & fat signal lots of calories
   Salt signals sodium-rich foods
   Sour or bitter tastes often avoided (signaled spoiled or toxic substances)
   And **can learn taste (flavor) preferences/aversions**…
   Often from **conspecifics**, e.g. parents, siblings, members of tribe
   In mammals via taste of mother’s milk
   In social living Ss, from flavor of mouth, lips, etc., observation

   **Individual experience**
   Learned taste aversions to **novel stimuli**
   Usually to stimuli that preceded nausea/vomiting (up to 5-6 hours)
   Can last a very long time
   Of survival value…usually…
HUNGER, EATING & WEIGHT REGULATION (cont., p3)
3. Factors that Determine What, When & How Much to Eat (cont.)
   b. Learning to Eat Essential Vitamins/Minerals
      Innate preferences for sodium, expressed immediately & without prior
      experience with sodium deficiency
      S immediately seeks salty tasting substances
      e.g. S with adrenal cortex malfunction (no aldosterone)

      Learned preferences for essential vitamins/other minerals
      Based in S by chance eating a food that makes it feel better
      Evidently, there is not distinctive taste of substances other than Na+
      And thus no innate preference has evolved…
      This was easier to do in our evolutionary past, when we ate a small
      number of foods, ate seasonable available foods

   c. When do we Eat
      Depends on how readily available food is…evolutionary perspective
      If it is readily available and plentiful…

      If food is scare, difficult to obtain, not readily available…

Also, in humans depends on learning (cultural norms), work schedules,
family routines, wealth, etc.
When do you eat?
How do you feel if you do not eat?
Do people with more regular meal times become conditioned to feel
better if they eat “on time”? What might be the underlying
physiology?
Can you be conditioned to enter the cephalic phase at a certain
time? What would increase in insulin do?
Could other “eating” cues trigger the cephalic phase?
   “premeal hunger”
3. Factors That Determine What, When & How Much to Eat (cont.)

d. How Much

1) chemical & stretch receptor feedback from the gut
   nutritive density (calories per unit of volume)
   these cues can (easily) be overcome by other factors

2) past experience with a particular food/volume
   “sham eating” experiments
   if S is familiar with food X, it will eat same amount it usually eats
   if S is unfamiliar w/ food X, it will eat much more

3) “appetizer” effect
   small amounts of food eaten before the actual meal --- S gets more
   more hungry and usually ends up eating much more
   these small prefoods --- elicits the cephalic phase responses
   note: these small prefoods often are highly seasoned & variable in
   flavors in real life (“appetizers”)

4) sensory-specific satiety
   if S eats a food with a specific flavor, even a highly preferred one,---
   flavor loses its positive incentive value quickly and S stops
   eating…so is S no longer “hungry”? but if give S another food with another flavor, it starts of eat again…
   so “hunger” still exists
   were observing SSS
   Why did SSS evolve? Why does it have survival value?

   To avoid SSS, one should eat foods with varied tastes…

5) social effects
   “social facilitation” in social living species
   Why does this have survival value?

   So, what is the effect of our ready access to a wide variety of abundant,
   variously seasoned, pleasant tasting foods? Coupled with our
   evolutionary past & brain mechanisms?…
4. **Brain Mechanisms**

Various hypothalamic areas receive a wide variety of peptide signals from the GI tract

- **e.g. CCK (cholecystokinin)**
  - seems to --- less eating, may do so by increasing nausea
  - can induce a learned taste aversion for a food eaten before CCK is administered

- **e.g. neuropeptide Y**
  - seems to increase eating when injected into paraventricular nucleus of hypothalamus

- **e.g. galanin**
  - seems to increase eating of fats especially
  - e.g. **leptin** (discussed below)

- **e.g. serotonin**
  - --- decreased appetite (decreases the positive incentive value of foods)
  - esp. decreases appetite for fats
  - e.g. Prozac
  - relationship to decrease in cravings for drugs of abuse?

5. **Weight Regulation**

Many people regulate their body weight within a fairly constant level, that

- **slowly increases** with time

  Why is it so **slow** to change? Why is it mostly **increasing**?

Note: many animal/human studies show that **calorie-restriction --- longevity** (along with good health and smaller stature)

- (less calories but a diet balanced nutritionally)
- (better I.S., less malignancies, greater lifespan)
- (some by-produce of energy consumption/storage accumulates in body’s cells and accelerates aging and health problems…?)
HUNGER, EATING, & WEIGHT REGULATION (cont.p.6)
5. Weight Regulation (cont.)
   a. Changes in energy utilization
      The body controls its fat levels by changing how efficiently it uses/stores energy, i.e. its basal metabolic rate
      as body’s fat levels decrease or as number of calories taken in decrease --- body becomes more efficient
      as body’s fat levels increase or as number of calories taken in increase --- body becomes less efficient

      termed “diet-induced thermogenesis”

      After a person loses a substantial amount of weight by dieting, exercise, or surgical removal of fat tissue, there is a about a 95%+ chance of the person regaining all of the lost weight and more once he/she returns to prior eating/exercise lifestyle
      Thus, to keep the weight off, the reduced caloric intake and/or increased exercise must be a permanent shift
      Why?

      Note: calories burned in exercise amount to a very small amount of of our daily caloric loss; 70% of our calories burned at “rest”
      Note: people who “figet” can increase calories burned, maybe significantly

   Role of genetics: ob/ob mice (homozygous recessive) & leptin
      Seems to have a negative feedback role for fat storage
      Ob/ob mice are obese, store more calories (as fat), more “efficient” metabolism (less use of fat calories)
      Their fat cells do not produce leptin but do have RSs in CNS
      If inject leptin in ob/ob mice --- lose weight
      But…obese humans have normal levels of leptin and if inject more leptin --- no reliable weight loss observed…why?
5. **Weight Regulation** (cont.)

**Insulin** may also be an important part of the brain’s feedback system for monitoring fat stores.
- Amount of insulin in brain remains relatively stable (despite what body levels are doing).
- Insulin does not easily cross the BBB.
- Brain levels of insulin are positively correlated with body fat stores.
- There are receptors (RSs) on brain neurons for insulin.
  - Stimulating these RSs reduces eating and weight loss in rats.
- Rats unable to synthesize brain insulin are obese.

**Serotonin** in brain seems to reduce eating, reduce the positive incentive value of food (esp. high calorie foods), reduce fat consumption, reduce subjective intensity of “hunger”, reduce the size of meals.
- 5HT does not act via leptin or insulin mechanism.
- Since there are many 5HT RS subtypes, hopeful that can synthesize a specific serotonin agonist to help reduce obesity.

In **anorexia nervosa** the positive incentive value of food is lowered.
- Note: almost all Ss with eating disorders have a *history of strict dieting* prior to the onset of the disorder…Beware!
- A special risk for Ss who are **highly controlled, rigid, ob-com**.
- Note: starvation usually triggers a radical increase in the positive incentive value of food…does not so in an anorexic…why?
- If anything, anorexics form learned taste aversions easily because they often feel nauseous when eating.
- Should be fed IV or with feeding tube small meals throughout the day…**do not let them form more LTAs**.