HUNGER, EATING, & WEIGHT REGULATION (p1)

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"

HUNGER, EATING, & WEIGHT REGULATION (cont., p4)

3. **<u>Factors That Determine What, When & How Much to Eat</u>** (cont.)

d. <u>How Much</u> do we Eat

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. **<u>Weight Regulation</u>** (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 + correlated with body fat stores
- There are RSs on brain neurons for insulin
  - Stimulating these RSs --- reduced eating & weight loss in rats
- Rats unable to synthesize brain insulin --- are obese

Serotonin in brain seems to --- reduce eating, reduce + incentive value of food (esp. high calorie foods), reduces fat consumption, reduces subjective intensity of "hunger", reduces 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 + 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 + incentive value of food…does **not** do 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**