

● ● ● | Psy 3061: Biological Psychology

Hunger & Eating

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● ● ● | Reward

A stimulus that the brain perceives as intrinsically positive



● ● ● | Reinforcer – what is it?

A stimulus that increases the probability that behaviors paired with it will be repeated.

Can be “positive” or “negative”

● ● ● | Reward vs. Reinforcement



Strength of reinforcement= how good is the reward?

● ● ● | What use is a reward system?

Direct behavior towards “advantageous” stimuli

Food



Sex



Social network



● ● ● | Reward gone awry

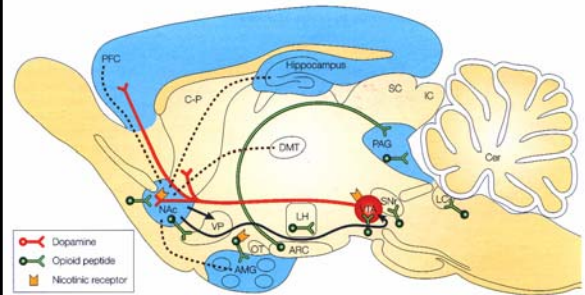
- Eating disorders (anorexia/bulimia)
- Obsessive/compulsive disorder
- Depression
- Drug addiction

Psychological components

- **Learning** about relationships among stimuli and consequences of actions
- **Emotional** response to reward consumption
- **Motivation** to learn and act

Each has explicit and implicit elements

Reward and Reinforcement Circuitry



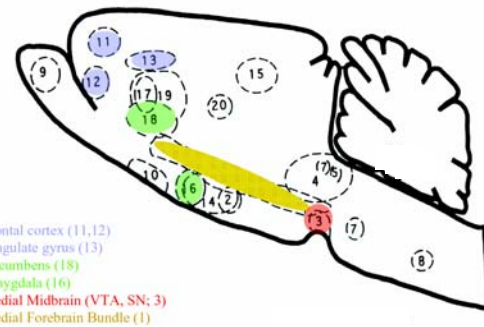
(from EJ Nestler, *Nature Reviews Neuroscience*, 2001)

Brain Stimulation Reward (BSR)

- Early 1950's— Olds and Milner
 - Implant electrodes in specific brain regions
 - Will animals return to location where they received stimulation?
- Later experiments— Intracranial self-stimulation

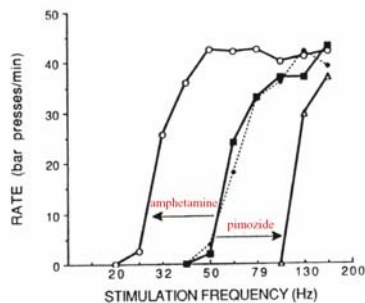


Circuitry associated with BSR



(Wise, 1996)

Dopamine involved in BSR



Gallistel and Karras (1984)

Hunger & Eating

- What do our bodies use for energy?
- When are different stores used?
- What factors control when and how much we'll eat?
- What are brain mechanisms involved in eating control?
- How do they go awry in eating disorders?

Energy Metabolism

- Energy-containing components of diet:
 - Lipids (fats) → Free fatty acids → ketones
 - Carbohydrates → glucose
 - Amino acids
- Utilizable fuels:
 - Glucose
 - Free fatty acids
 - ketones

Energy Metabolism Phases

- **Cephalic:** Preparatory; initiated by cues and expectation of food
- **Absorptive:** Energy utilized directly during food consumption. Excess is stored.
- **Fasting:** Energy drawn from stores

Theories of Hunger & Eating

Why do we eat?

- “Set-point” theories
- “Positive-incentive” theories
- Conditioned and unconditioned factors
- “Settling-point” theory

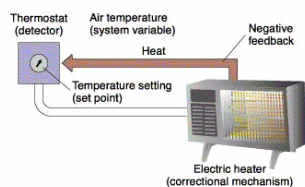
Theories of Hunger & Eating

“Set-point” theories

We’re running out of energy!

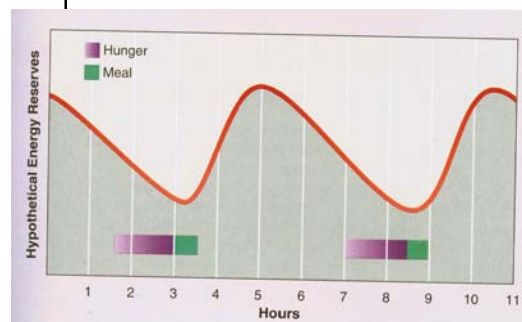
- Thermostat-like mechanism
 - Involves negative feedback
- Homeostasis
 - Maintenance of a constant internal environment
- Types of set-points
 - Glucostatic
 - Lipostatic

► An Example of a Regulatory System



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Energy Set-Point View



Theories of Hunger & Eating

Positive-Incentive theory

Just like grandma used to make...

- Eat because of positive-incentive value of food (i.e., eating is pleasurable)
- Hunger related to positive-incentive value of eating. Influenced by factors such as:
 - Time of day
 - Behavior of other people
 - Learned taste preferences

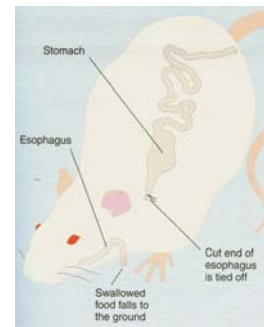
Theories of Hunger & Eating

- Are set-point and positive-incentive theories mutually exclusive?
- Maybe more like two sides of the same coin:
 - Set-point regulation: Unconditioned
 - Positive-incentive regulation: Conditioned

Unconditioned factors

- Hunger associated with
 - Low glucose
 - Low lipids
 - Low salt

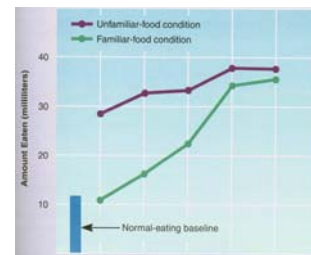
Sham-Eating Preparation



Conditioned Factors

- Flavors associated with high calorie foods
- Flavors associated with nutritive foods
- Meal times
- Conditioned taste aversion
- Social Influences
 - Rats eat more when in groups
 - Transmission of flavor preferences

Sham-Eating Preparation



Amount eaten determined by prior experience of eating the same flavor

Social Transmission of Food Preference

Demonstrator



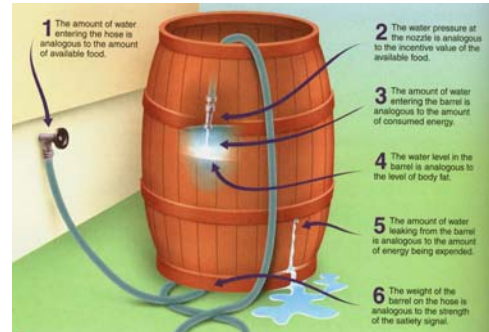
Exchange of information



Preference test



Settling Point



Settling Point

“... As body-fat levels increase, changes occur that tend to limit further increases until a balance is achieved between all factors that encourage weight gain and all those that discourage it.”

--Pinel

Settling Point

- Set point can change!
 - The dieting “yo-yo”



- 1 Weight loss occurs rapidly at beginning of diet
- 2 As weight declines, the amount of energy “leakage” or substrate oxidized, and this reduces the rate of weight loss
- 3 Gradually the reduced rate of intake is matched by the reduced energy output, and a new stable settling point is achieved
- 4 When the diet is terminated, weight gain is rapid because of the high incentive value of food and the low level of energy leakage
- 5 As weight accumulates, the incentive value of food gradually decreases and the energy leakage increases until the original settling point is regained

Putting strain on the settling point

- What affect does this have on body weight?
 - Persistent overeating and lack of exercise causes disturbances of mechanisms involved in regulating body weight
 - Causes shift in set point to new weight
 - Over time, set point drifts increasingly from optimal weight

Obesity

- In US:
 - 2/3 of population is overweight
 - 1/3 is obese
 - Obesity unknown 100 years ago
 - 1/12 suffers from diabetes
 - Incidence of diabetes has doubled over last 10 years

Source: NATURE|VOL 428 |18 MARCH 2004

● ● ● Obesity: Treatment

- Current treatments ineffective, or drastic, or both
 - Diet
 - Exercise
 - Drugs
 - Xenical
 - Phentermine
 - Gastric stapling

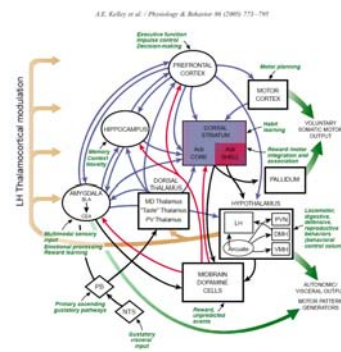
● ● ● Neural Mechanisms

- To more effectively treat obesity and other eating disorders, first we need to understand neural mechanisms:
 - Role of nuclei in the hypothalamus
 - Role of the hormone, leptin
 - Role of neurotransmitters (especially peptides)

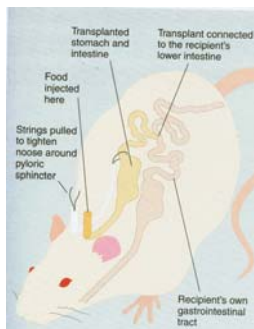
● ● ● Neural Structures

- What brain structures important in regulating feeding behavior?
 - Hypothalamus
 - Nucleus accumbens
- Current thinking is that the following nuclei in the hypothalamus are important:
 - Lateral nucleus
 - Paraventricular nucleus
 - Arcuate Nucleus

● ● ● Neural circuits for eating control



● ● ● Two stomachs experiment



● ● ● Brain Mechanisms: Leptin

- Ob/ob mutant mice: “grossly” obese
- Ob gene codes for leptin protein (Friedman, 1994)



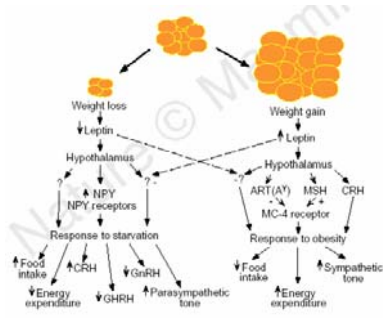
Brain Mechanisms: Leptin



Brain Mechanisms: Leptin

- Injections of leptin in rats suppress feeding
- Leptin secreted by adipose tissue
- Transported across blood-brain barrier
- Acts in hypothalamus via neuropeptides (see ahead)

Brain mechanisms: Peptides



Leptin in Obesity?

- Leptin treatment?
 - Obese patients already have high levels of leptin in blood stream.
 - Obese patients' brains are leptin-insensitive
- Thus, although leptin dysfunction seems to be involved in obesity, leptin cannot generally be used to treat obesity.

Neurotransmitters

Table 1 Hypothalamic modulators of food intake

Increase food intake	Decrease food intake
NPY	CART
MCH	CKK
Galanin	CRH
Orexin a and b	α -MSH
Peptide YY	Insulin
Noradrenaline	GLP-1
(α 2 receptor)	Bombesin
	Urocortin
	Serotonin

Many factors regulate feeding behaviour. The neurotransmitters and neuropeptides known to regulate food intake are shown. Leptin probably modulates the activity some or all of these factors (and vice versa). A detailed understanding of the functional relationships among leptin and these (and other) neuropeptides and neurotransmitters will be necessary to determine the mechanisms regulating food intake and body weight. GLP-1, glucagon-like peptide-1.

Neurotransmitters and neuropeptides in hypothalamus that modulate food intake

Anorexia nervosa

- Can be fatal
- Common in cultures that put premium on thinness
- Cause unknown
- No effective drug treatment
- Treatment tends to be in-patient, counseling, diet management
- Pinel's hypothesis:
 - loss of motivation to eat due to conditioned taste aversion?