Intro to Behavioral Neuroscience (B)
Lecture 2: Motivation

Richard Veale

Graduate School of Medicine
Kyoto University

https://youtu.be/hrcoDdEUzR8

Lecture video at above link.

Today

Motivation

- Homeostasis and motivation
- Hierarchy of needs
- Hunger and Satiety -> Feeding behavior
- Reward system, intracranial self-stimulation
- Runner's high

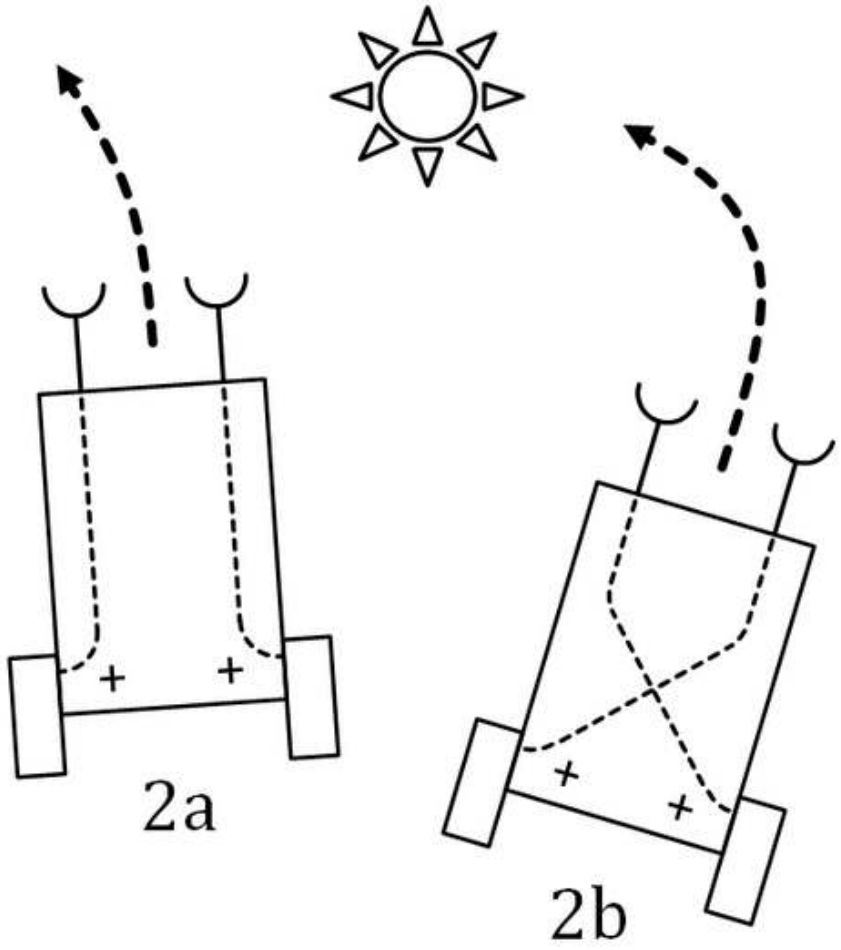
Motivation

Question: What makes people spend so much time on video games?

Obviously that is a very difficult problem
→ First, something “more basic”:

Pinning down motivation

Braitenberg vehicles 2a and 2b:



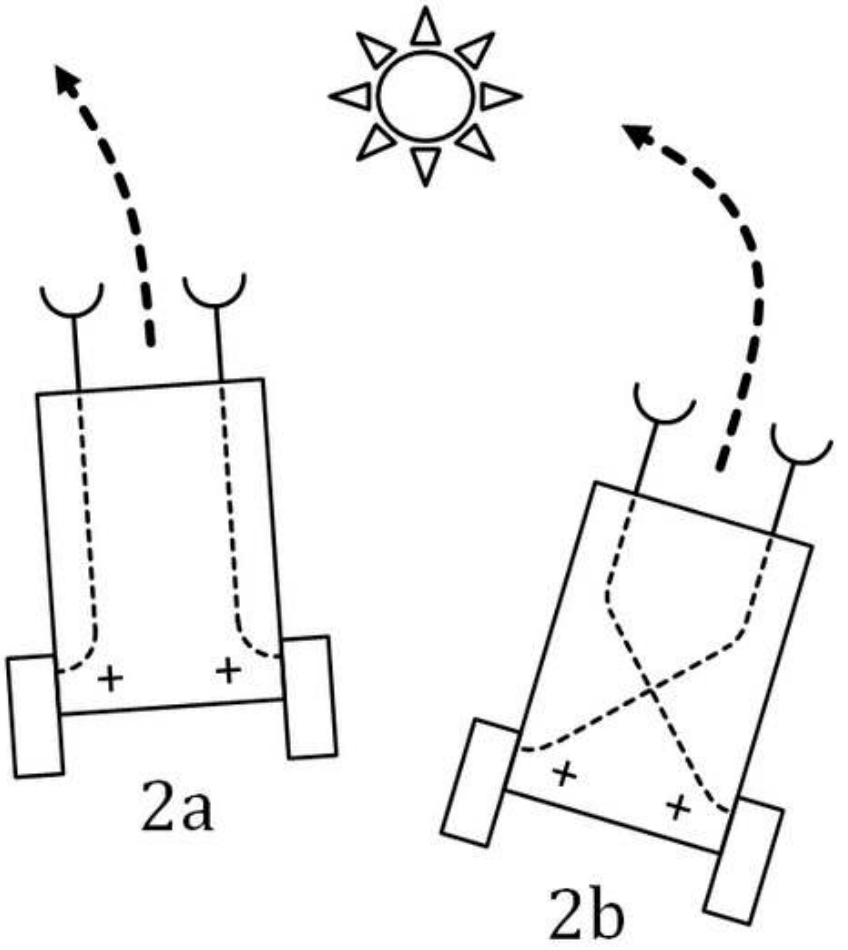
Vehicle 2a flees the sun
2b approaches it.

Braitenberg, Vehicles, 1984

Motivation

Vehicle 2a may flee the sun to avoid predators (think of cockroaches).

Vehicle 2b may approach the sun to charge its batteries.



The point of these vehicles is that when you want to understand the circuit (brain) of something, it is helpful to think of the problems/functions it has to solve.

“Purpose” of our behavior: Homeostasis

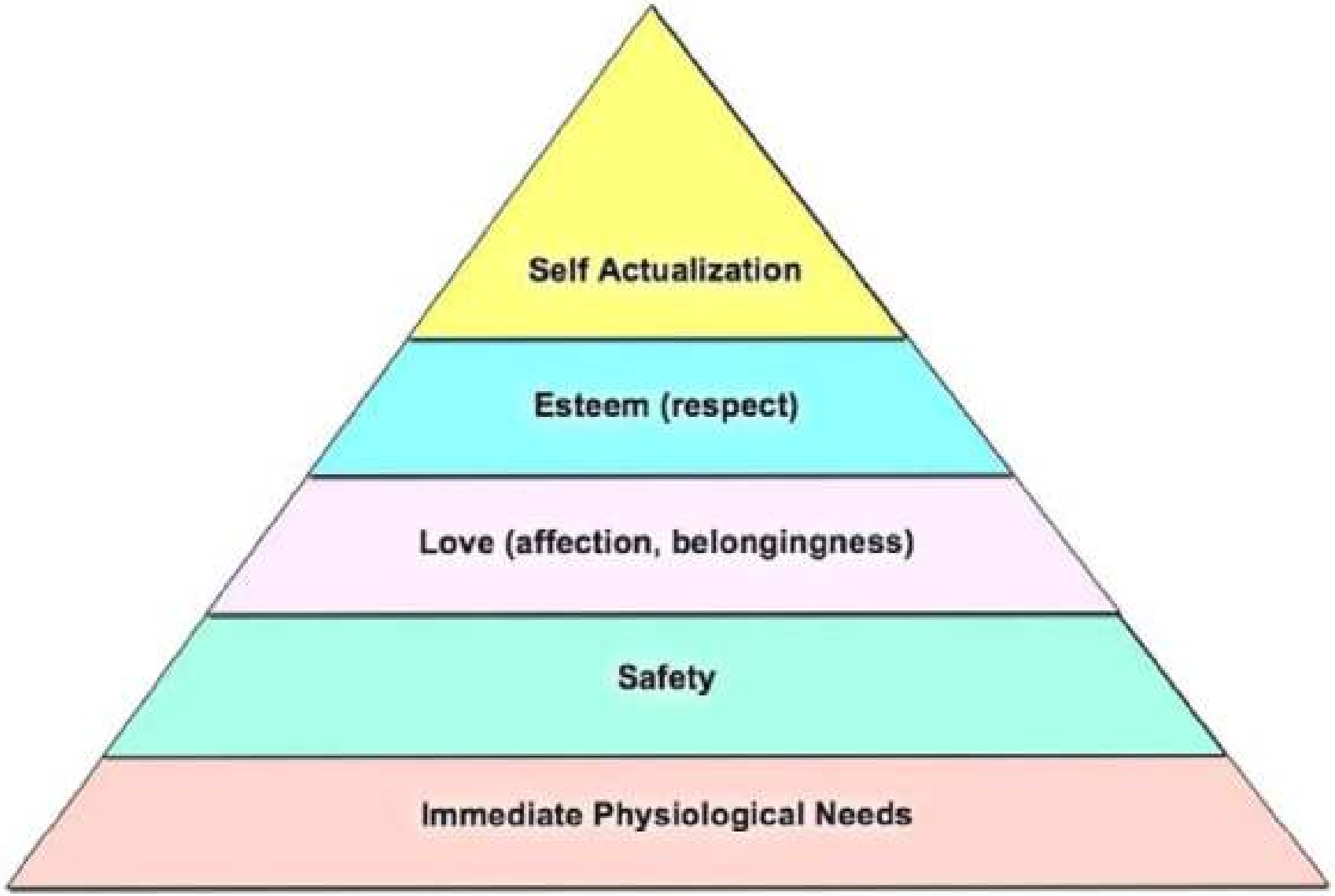
Our body functions only in a narrow physiological range:

Temperature (33 degrees → dead, 42 degrees → dead)
Oxygen (low blood oxygen → dead)
Nutrients (salt, sugar, lipids, amino acids)
(low blood sugar → dead, high blood sugar → dead)
Water, etc.

Our behavior changes in order to fulfill these needs
→ **homeostasis** (= to stay within a similar range)

Thermostat
(turn on heater if too cold)
(turn on cooler if too hot)

Maslow's Hierarchy of Needs

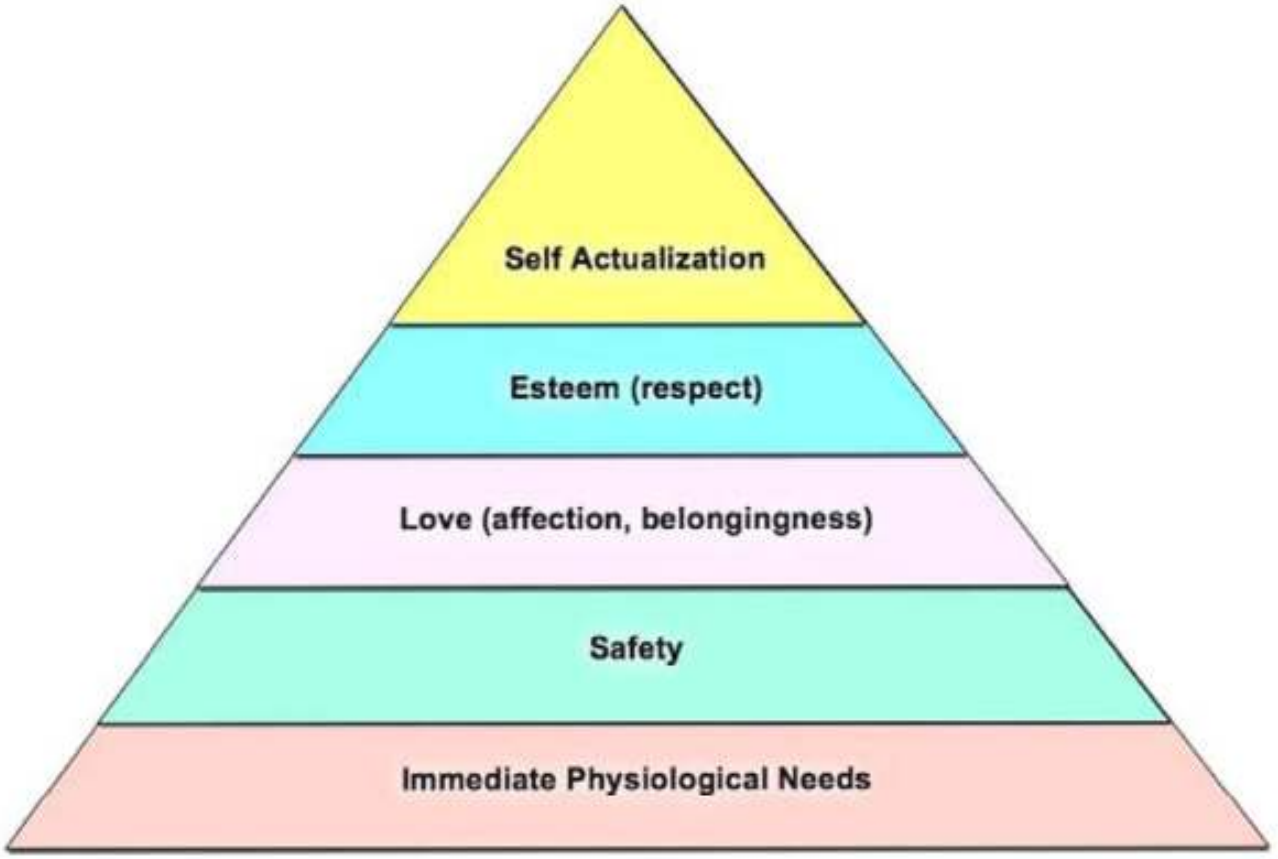


Kenrick et al., 2010; after Maslow, 1943

Maslow's Hierarchy

Two important ideas

1) Independent motivational systems
2) These are hierarchically organized (lower needs must be fulfilled before we want to achieve higher needs)



Kenrick et al., 2010; after Maslow, 1943

Maslow's Hierarchy

Two important ideas

1) Independent motivational systems
2) These are hierarchically organized (lower needs must be fulfilled before we want to achieve higher needs)

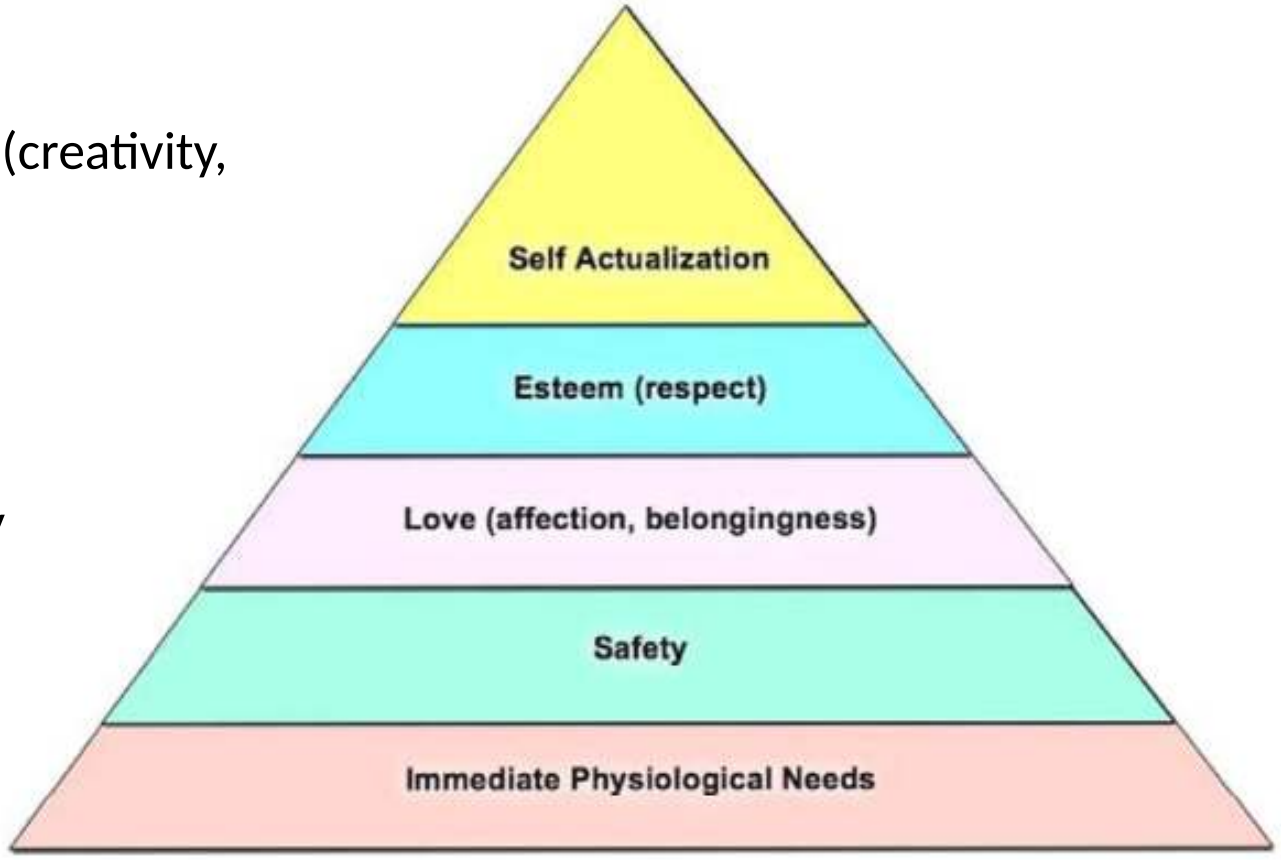
Fulfilling one's potential (creativity, etc.)

Social status

Friends, Intimacy, Family

Not being harmed

Hunger, Thirst, Sex

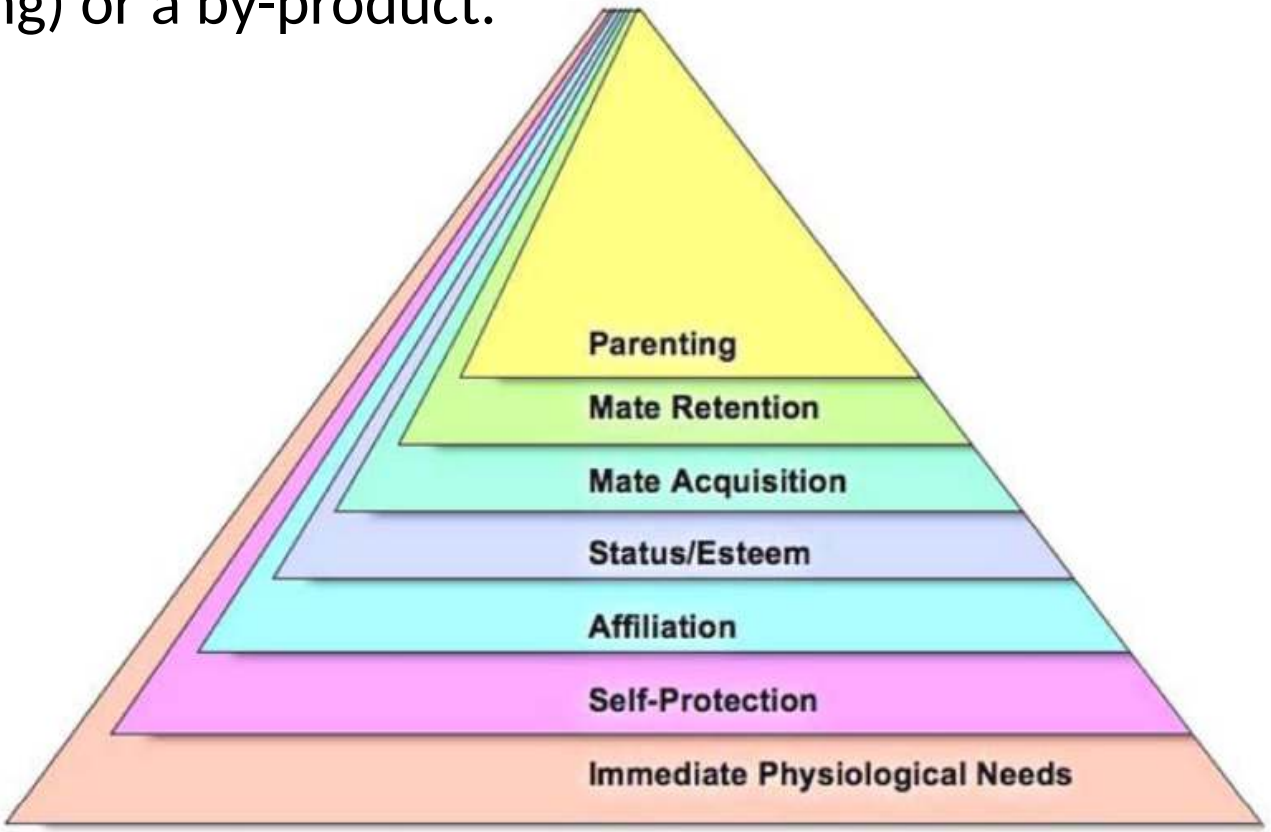


Kenrick et al., 2010; after Maslow, 1943

“Update” to Maslow’s Hierarchy

Motivational systems can coexist,
no strict hierarchy

Self-actualization may be part of other motivational systems (status, mating) or a by-product.

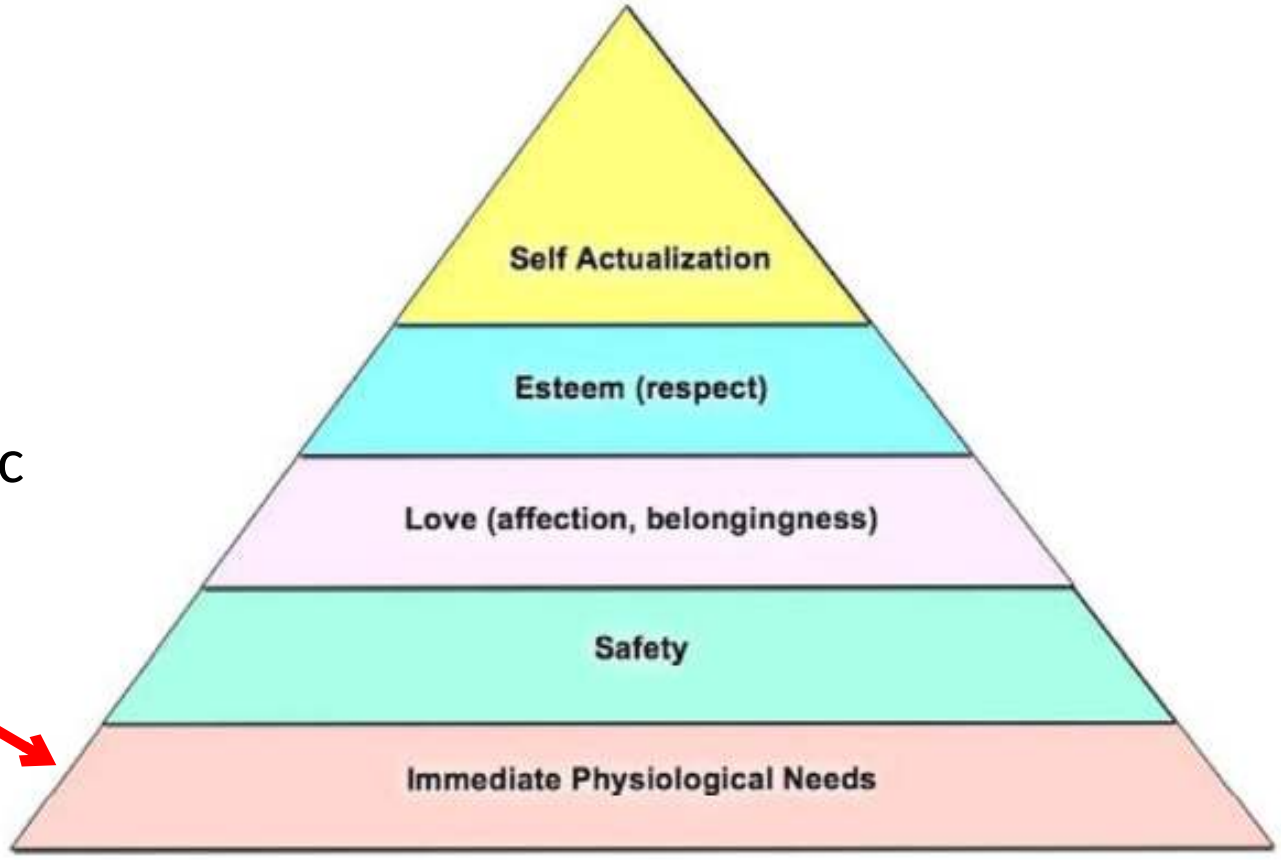


Kenrick et al., 2010

Maslow's Hierarchy: Let's start at the bottom…

Where is the need for video gaming?

Let's look at the basic needs first!



Kenrick et al., 2010; after Maslow, 1943

What motivates you?

What motivates you to *eat*?

→ You feel hungry?

What motivates you to *study*?

→ Curiosity?

→ Social expectations (from parents?)

→ Fear (of failure?)

What motivates you to play video games instead of studying?

What motivates you?

What motivates you to *eat*?

→ You feel hungry?

What motivates you to *study*?

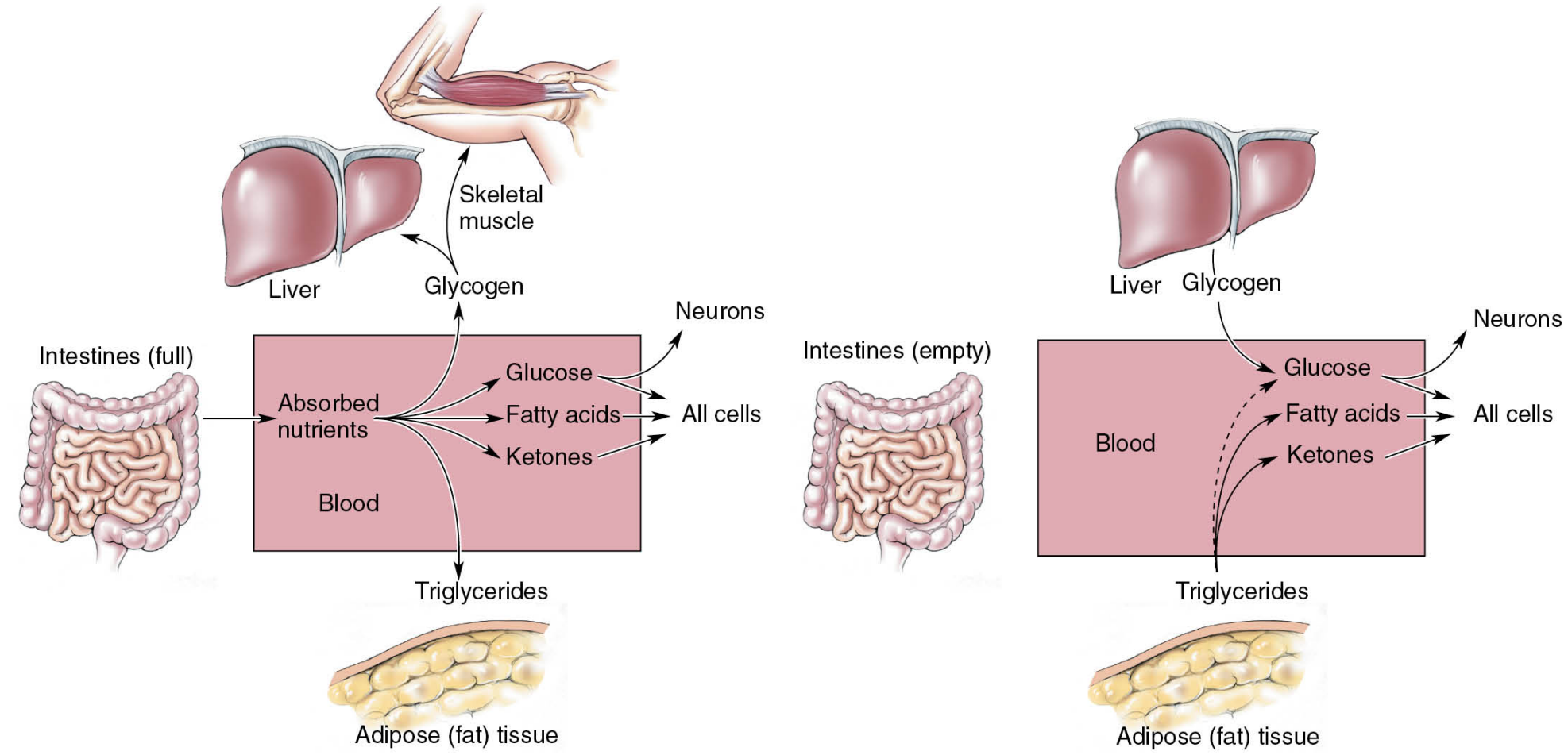
→ Curiosity?

→ Social expectations (from parents?)

→ Fear (of failure?)

What motivates you to play video games instead of studying?

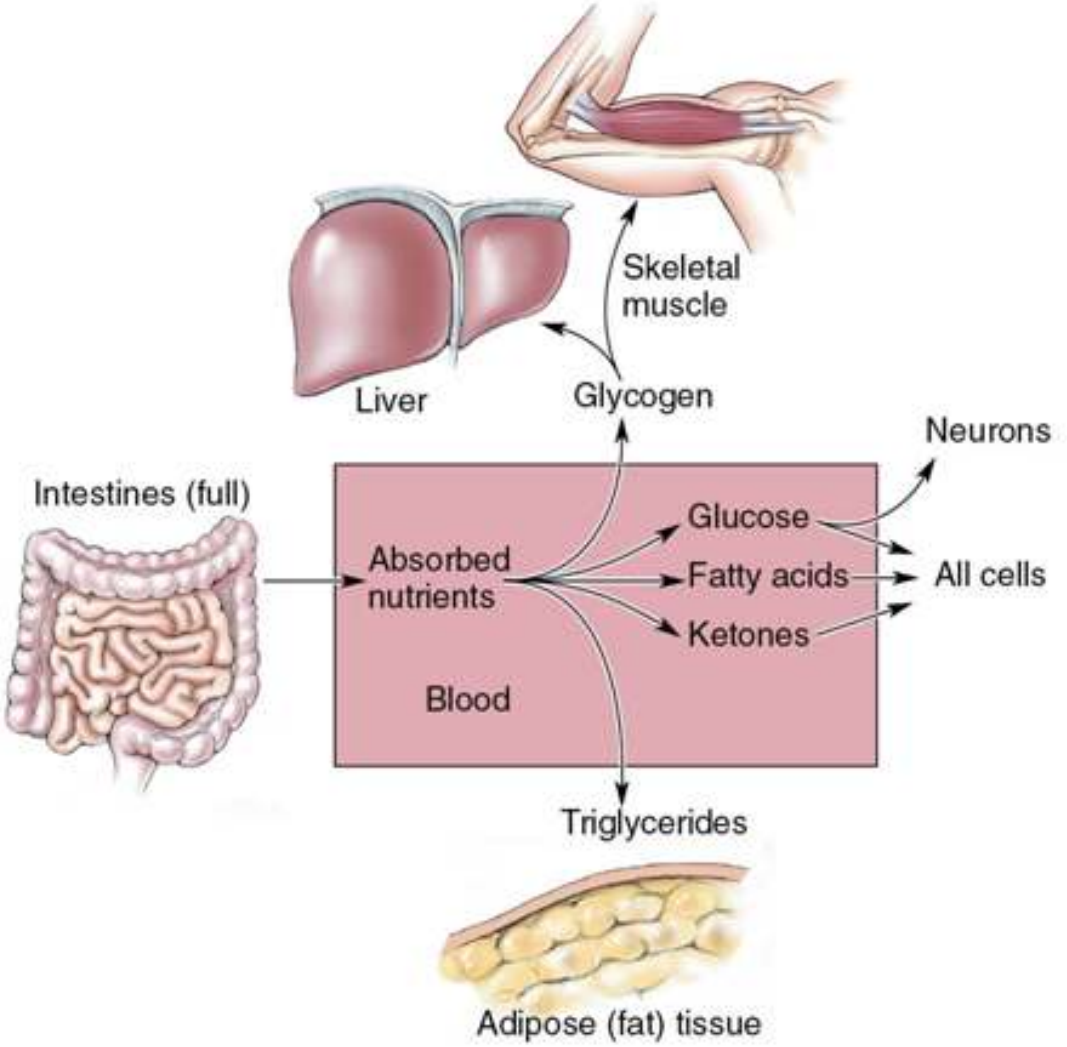
Metabolism (motivation to eat?)



(a) Anabolism during the prandial state

(b) Catabolism during the postabsorptive state

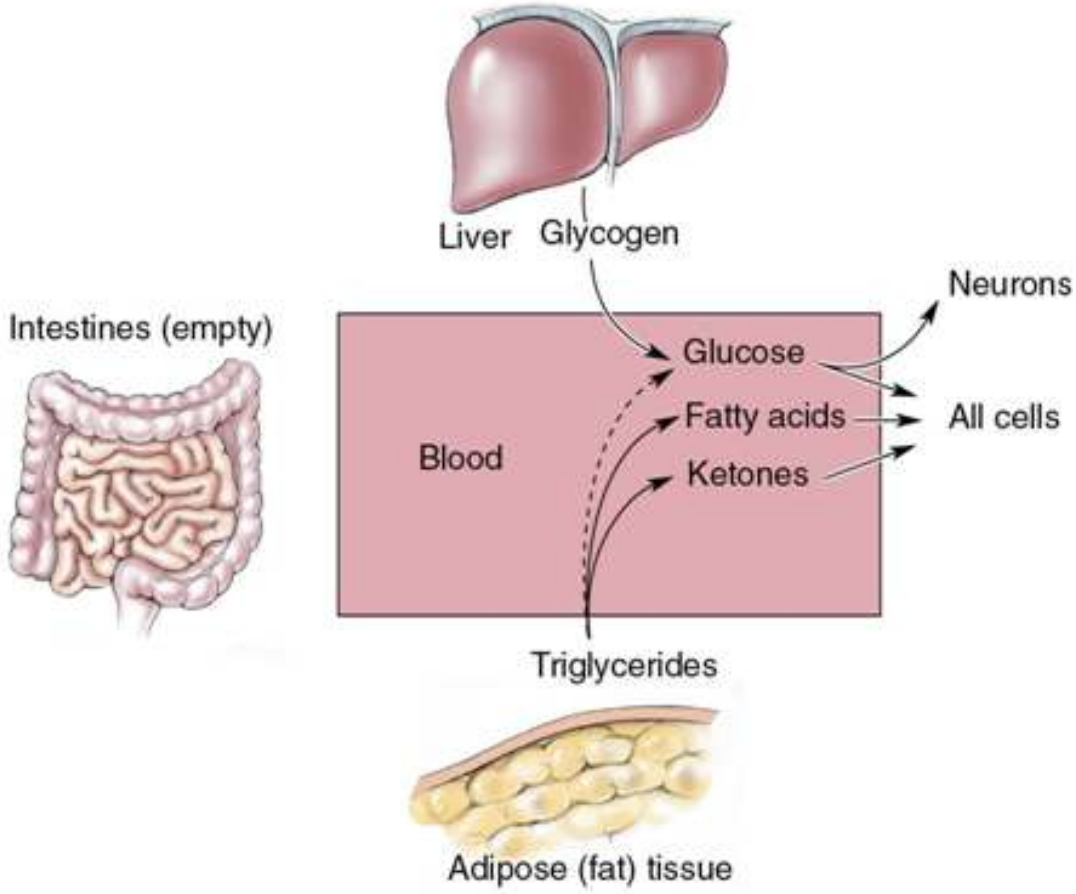
Metabolism 1:
Anabolism (net positive nutrients)



(a) Anabolism during the prandial state

Anabolism:
Nutrients come in and are stored as glycogen and triglycerides.

Metabolism 2:
Catabolism (net negative nutrients)



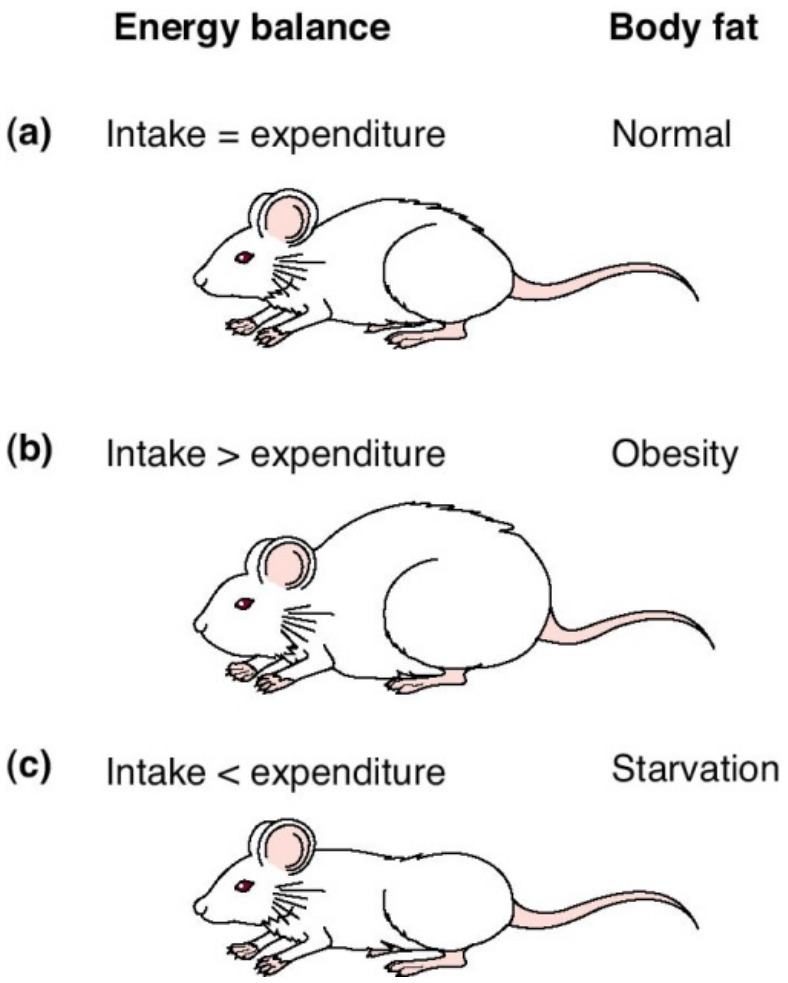
(b) Catabolism during the postabsorptive state

Catabolism:
The body needs energy and takes it from stored glycogen and triglycerides. They are broken down and enter the blood.

If intake does not meet demands, the energy stores are used up -> starvation

Long-term regulation
Feeding behavior

Weight gain/loss depends on the energy balance of intake of food to expenditure (e.g., by physical activity).

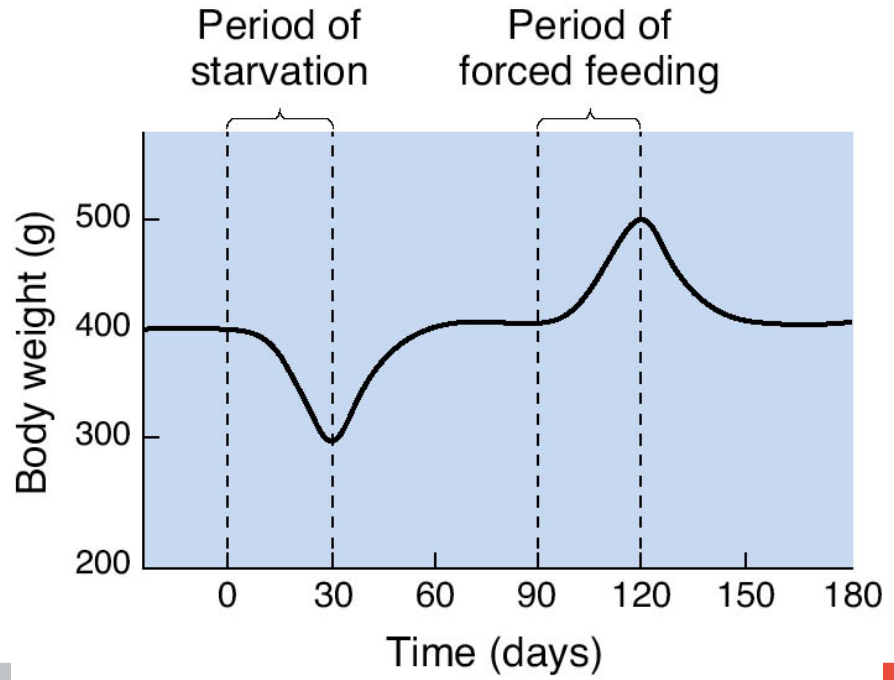


Long-term feeding regulation

In the long run, we and also mice (graph) tend to compensate weight loss/gain. So, our weight tends to return to a set-point.

For this to happen, the brain should have a record of current body weight and accordingly inhibit/stimulate feeding behavior.

In reality, the set-point theory does not explain the obesity epidemic: when people have sufficient nutrition, many become obese.
Evolution might have biased us towards overfeeding, because most of our history the problem was starving.

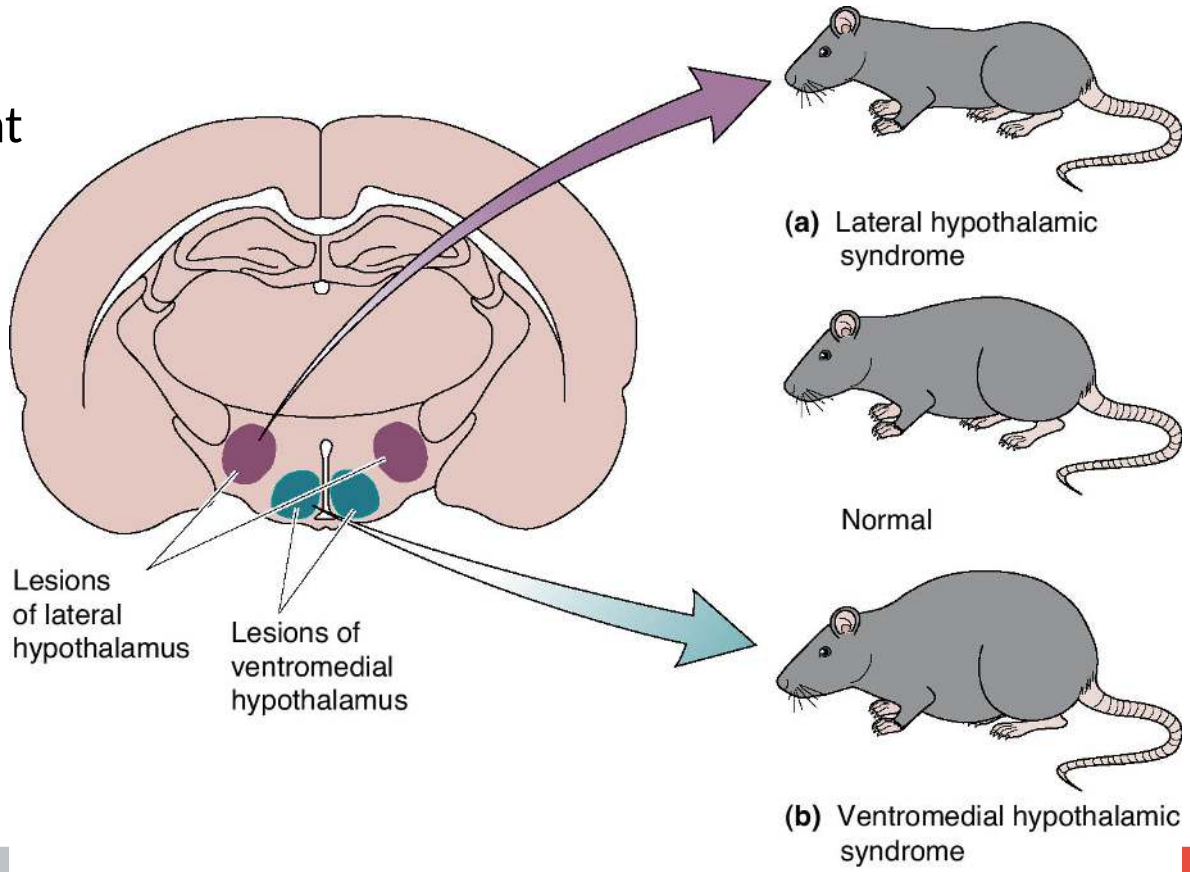


Long-term feeding regulation

Lesions in the hypothalamus can lead to anorexia/obesity depending on the lesion (rat experiments, Hetherington and Ranson, 1940).

a) Lesions in the bilateral lateral hypothalamus lead to a failure to induce sufficient feeding behavior.

b) lesions of the bilateral ventromedial hypothalamus lead to overeating, the animals are insatiable.

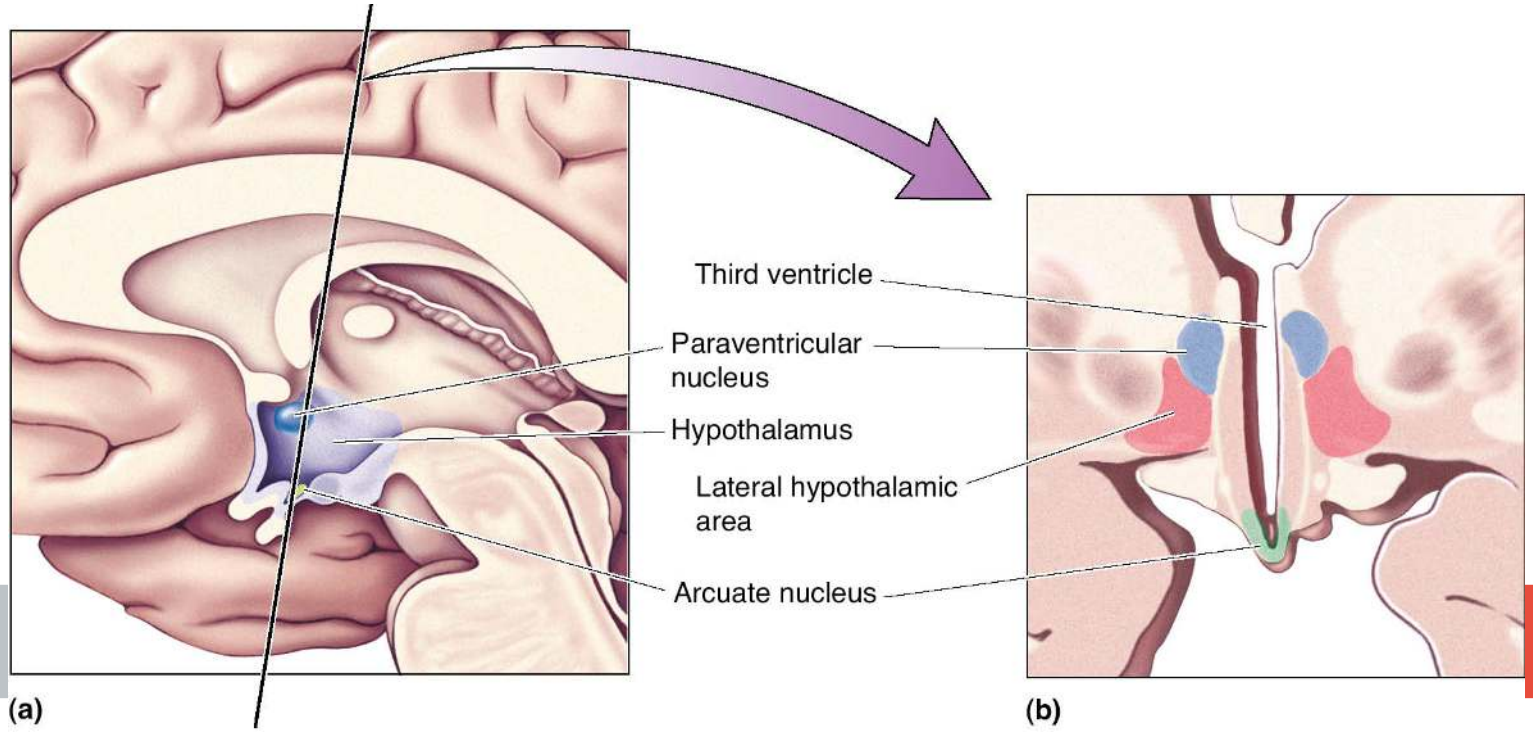


Long-term feeding regulation: Leptin → Hypothalamus

Fat cells release the hormone **leptin** to signal when they have “full stores”.

Receptors in the arcuate nucleus of the hypothalamus receive and process this long-term satiety signal. -> Less feeding (via the lateral hypothalamic area), increased metabolism (burning of energy, via hormone release from the paraventricular nucleus).

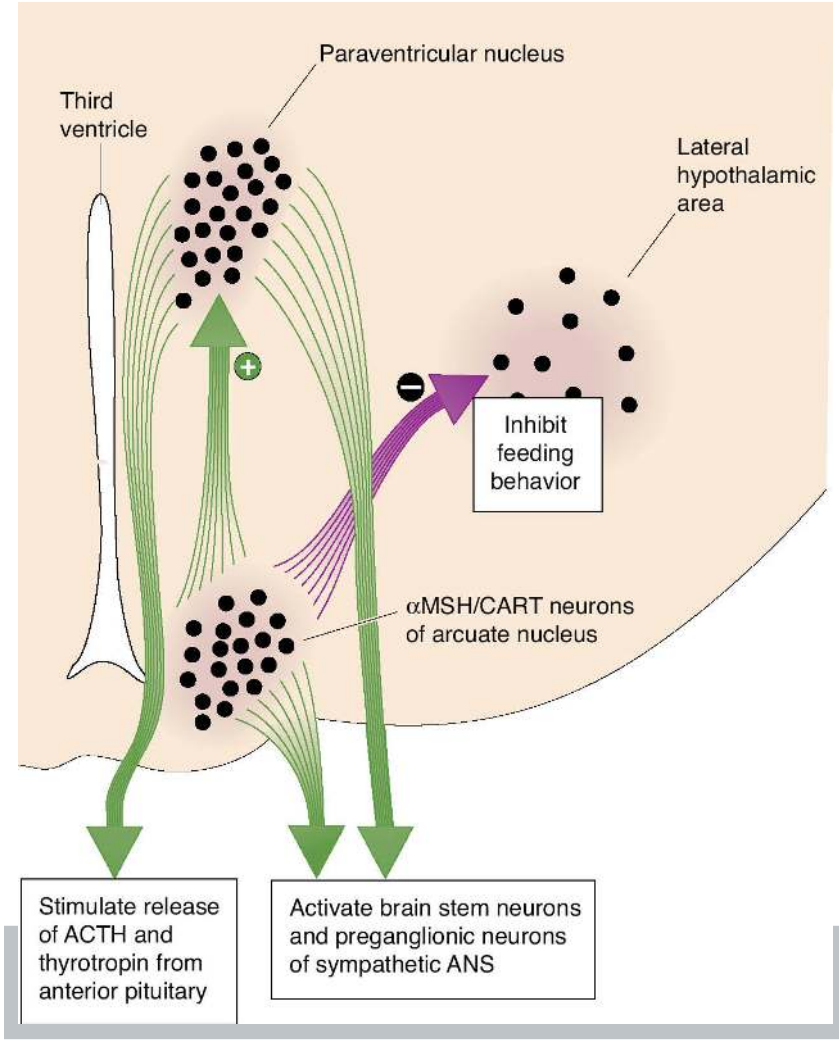
Lack of leptin activates other neurons in the arcuate nucleus that stimulate feeding behavior via the lateral hypothalamic area.



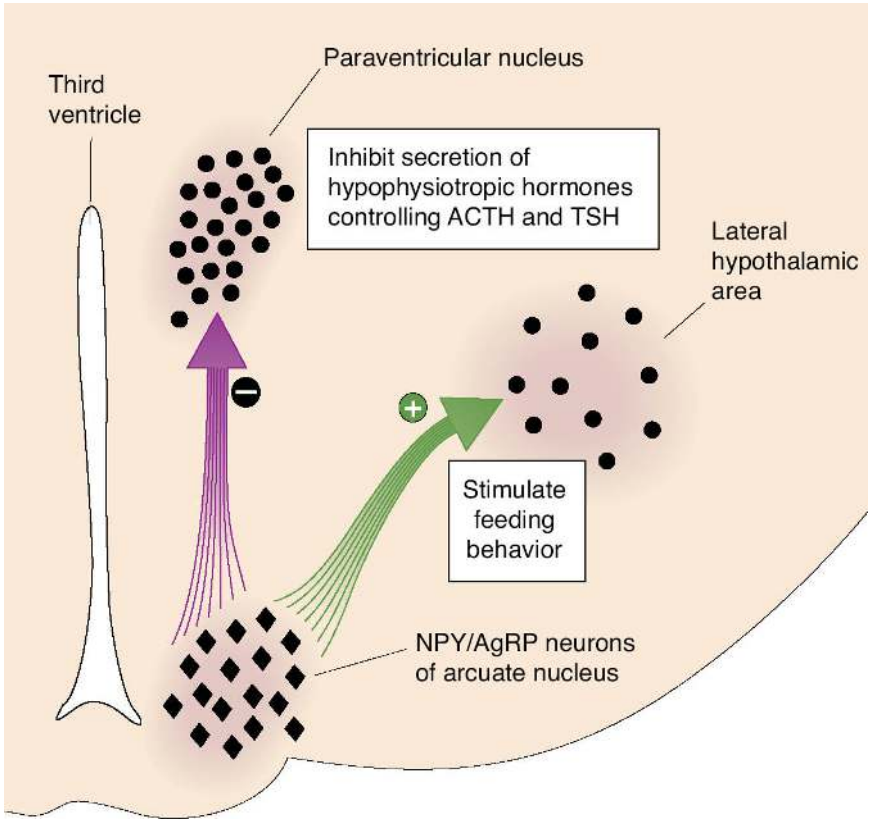
Long-term feeding regulation: Leptin → Hypothalamus

ACTH: adrenocorticotropic releasing hormone
TSH: thyrotropin, thyroid-stimulating hormone

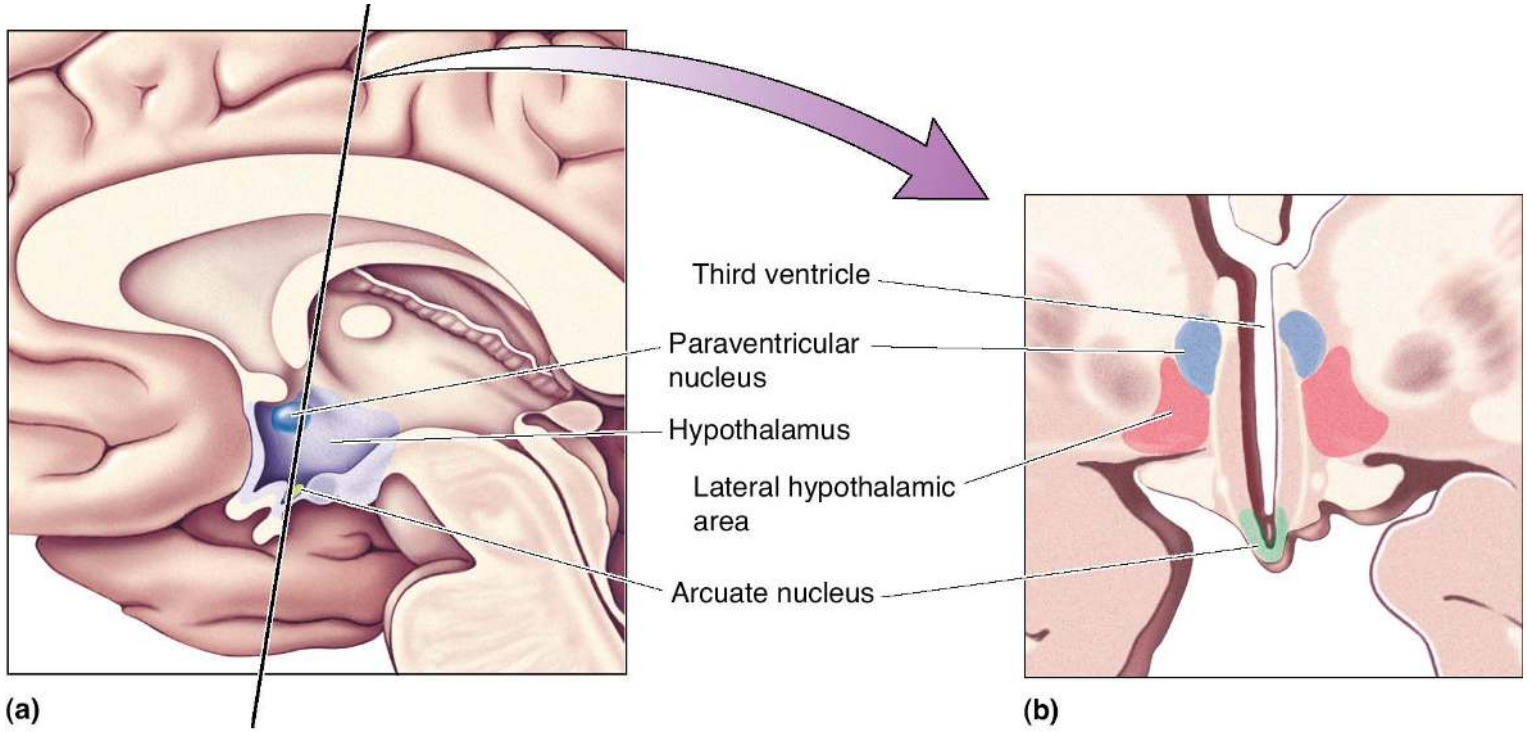
leptin level high (body fat high)



leptin level low (body fat low)

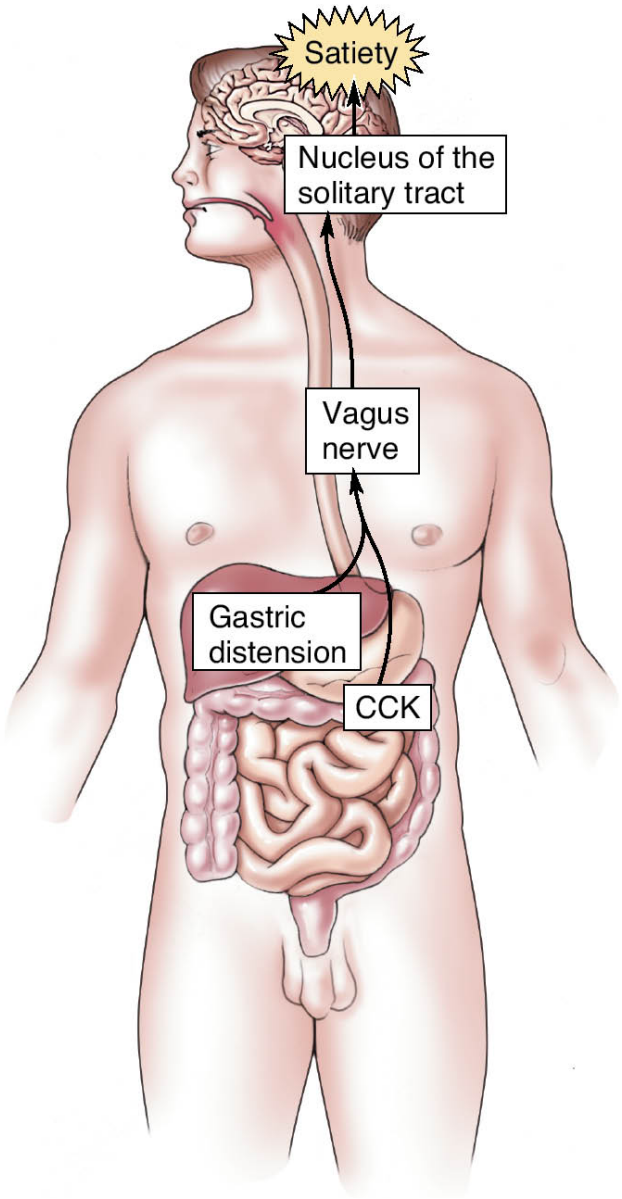


Long-term feeding regulation: Is leptin the whole story?



However, taking leptin as a drug against obesity works only in a small group of people with congenital deficiency of this hormone. Normal obese people often have high leptin levels. Possibly, they are less sensitive to leptin.

Something else is causing feeding… Short-term regulation?



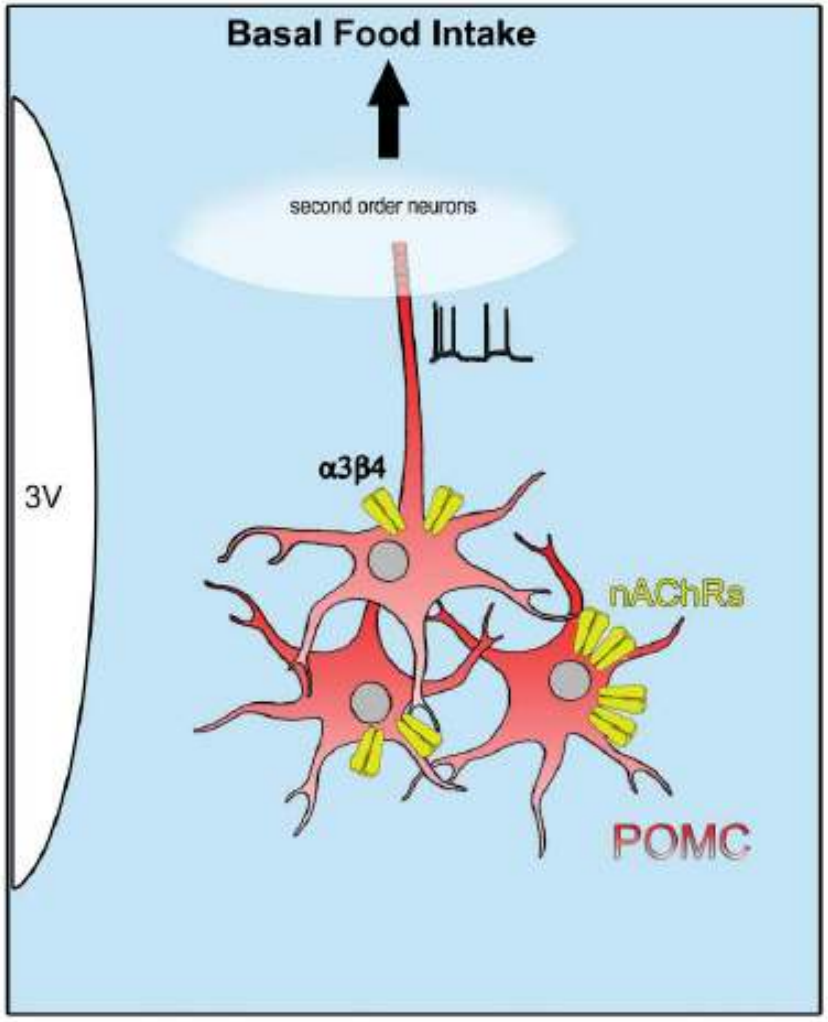
Hunger signal:
Ghrelin is secreted from the stomach when empty. It is detected by the hypothalamus (arcuate nucleus).

Satiety signals:
→ Gastric distension ("full stomach").
→ CCK (Cholecystokinin) is released from intestines in response to stimulation.

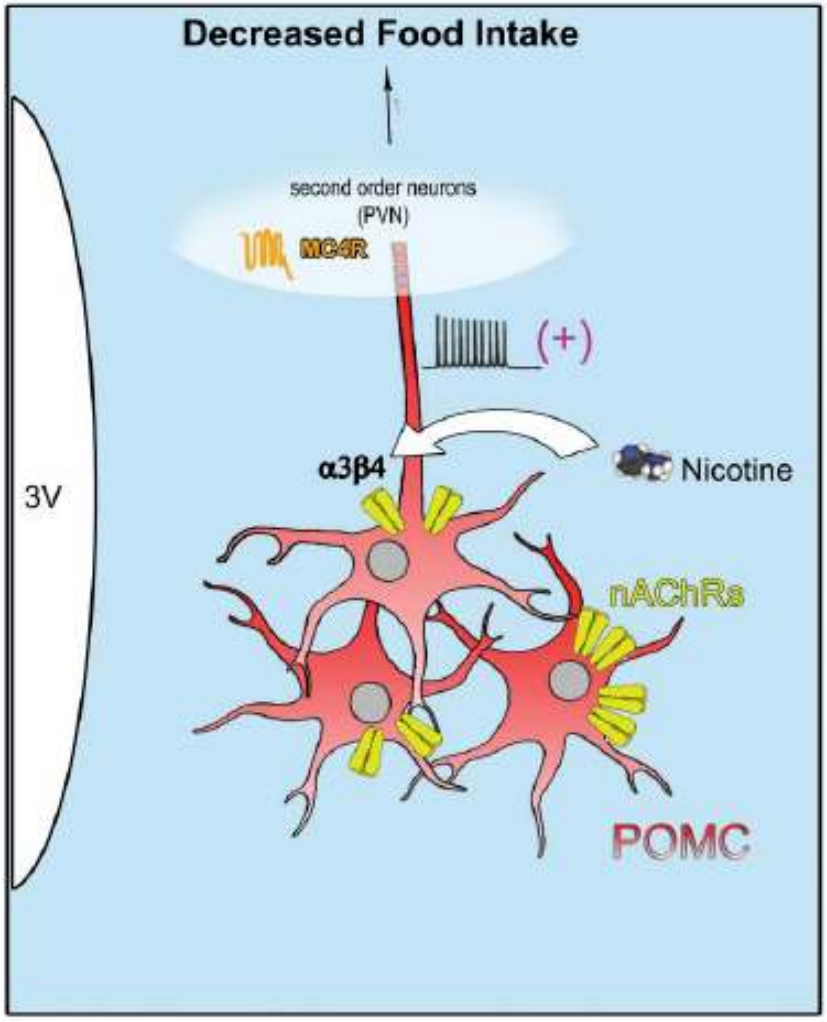
→ Insulin regulates the blood glucose level (storage). Insulin also acts on hypothalamic nuclei to inhibit feeding. Insulin secretion in turn is mainly triggered by high glucose levels.

Nicotine:
Effect on feeding behavior (appetite)

In mice, nicotine binds to acetylcholine receptors (nAChRs) of certain hypothalamus neurons (POMC) in the arcuate nucleus → **less food intake.**



Basal state



Nicotine-activated state

So…nicotine is a good diet drug (But, smoking gives you cancer)

But don't forget:
Tobacco smoking is the leading cause of preventable deaths!

Doll and Hill, British Medical Journal, 1964

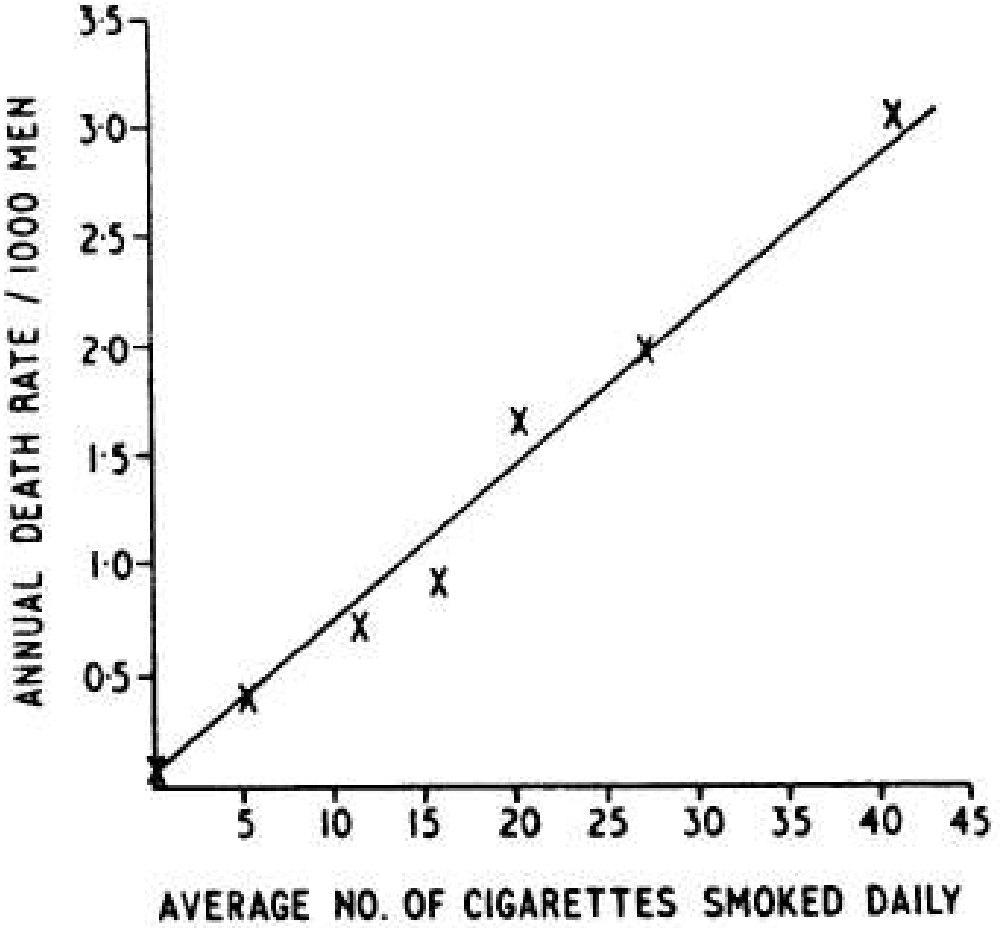


FIG. 1.—Death rate from lung cancer, standardized for age, among men smoking different daily numbers of cigarettes at the start of the inquiry (men smoking pipes or cigars as well as cigarettes excluded).

Endogenous vs Exogenous

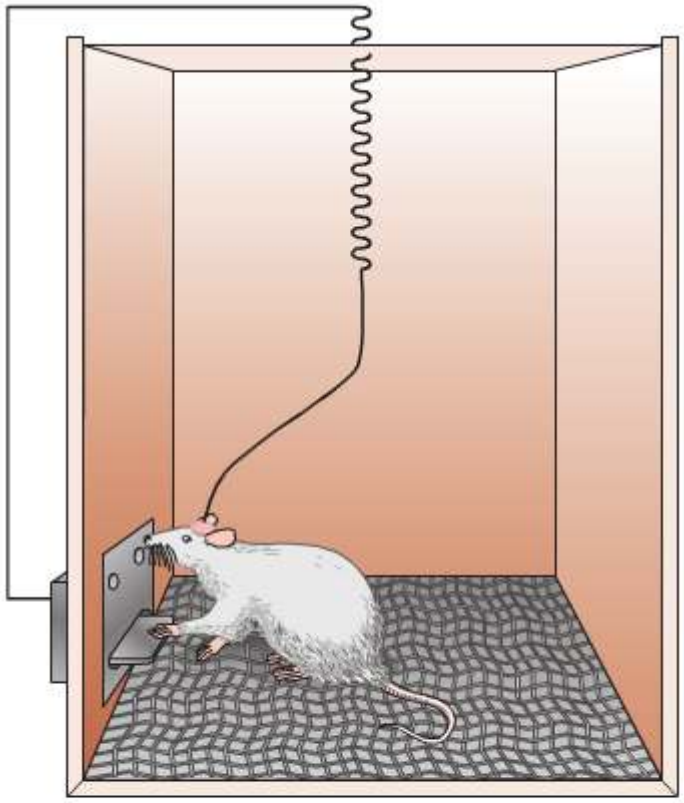
Feeding Behavior is *Endogenous* motivation

→ Based on signals from within the body

But, what about *Exogenous* motivation?

→ Signals from *outside* the body

Reward System



▲ FIGURE 16.15
Electrical self-stimulation by a rat. When the rat pr esses the lever, it receives a brief electrical curr ent to an electrode in its brain.

Olds & Milner, 1954

Rats were implanted with stimulation electrodes.

They could stimulate themselves by pressing a lever → intracranial self stimulation.

Stimulation of dopaminergic neurons (neurons that use dopamine as a neurotransmitter) projecting from midbrain to forebrain (mesocorticolimbic system) leads to more and more lever presses.

Reward system: Rat

Rat brain:
dopaminergic neurons project from the ventral tegmental area (midbrain: meso) to the nucleus accumbens (part of the ventral striatum) and cortex.

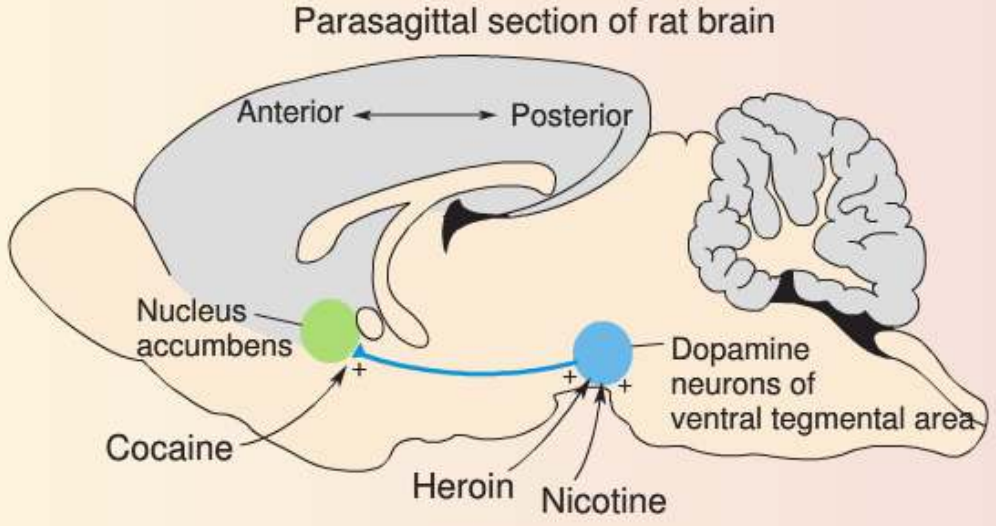


Figure A
Addictive drugs act on the dopaminergic pathway from the ventral tegmental area to the nucleus accumbens. (Source: Adapted from Wise, 1996, p. 248, Fig. 1.)

Many drugs (such as heroin, cocaine, nicotine) also affect this dopaminergic system.

Opioid Epidemic (e.g. heroin...)

Economist

In 2016, 64,000 people have died from drug overdose in the USA, making it the leading cause of death for those under 50 (New York Times).

Deaths in traffic accidents are only around one half of that.

The main reason for this current epidemic is a new opioid crisis that probably resulted by lax prescription of synthetic opioids (such as fentanyl or oxycodone) since the 1990s for (chronic) pain.

Opiods in the brain

Opioids also affect the reward system via the ventral tegmental area (VTA below).

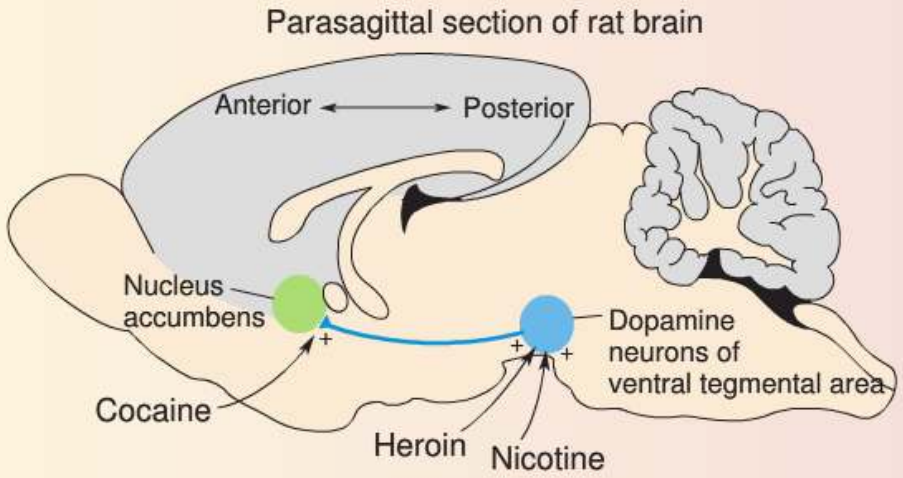
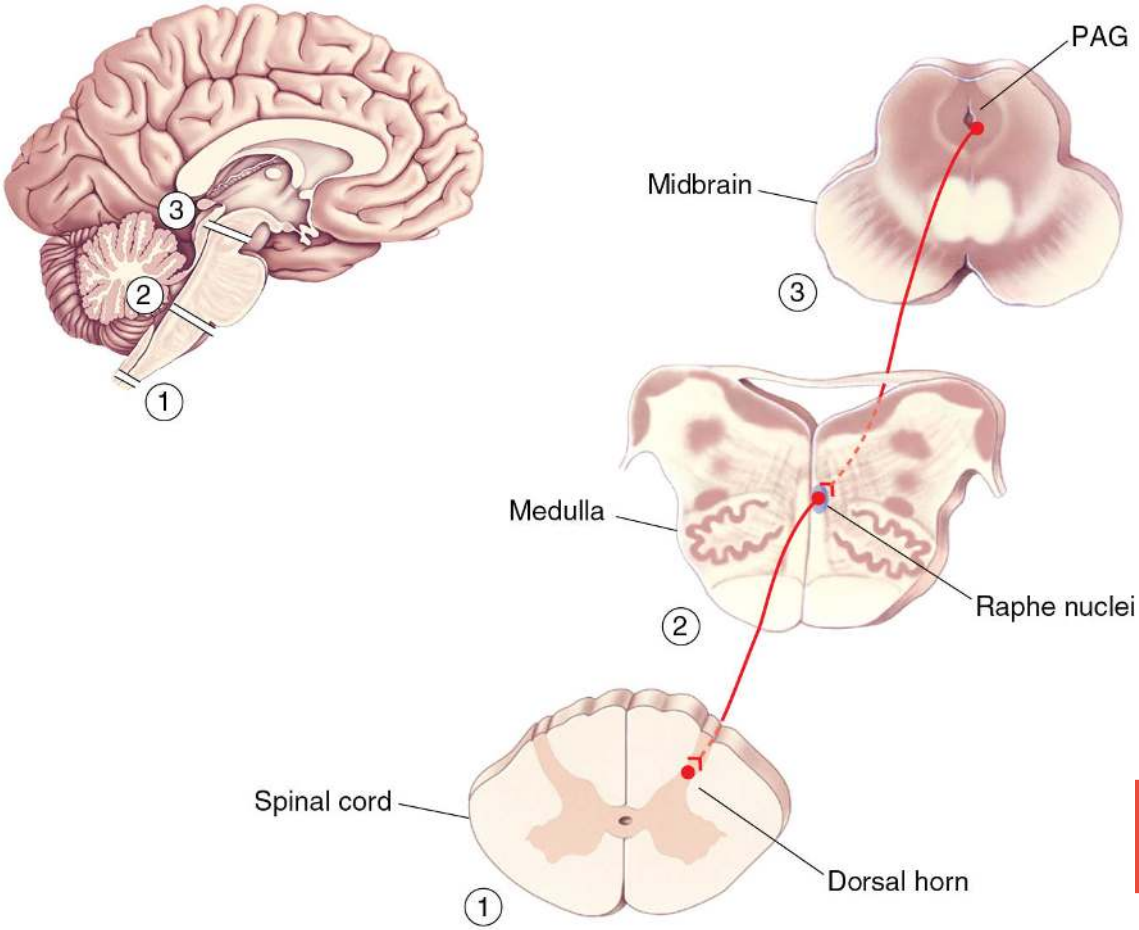
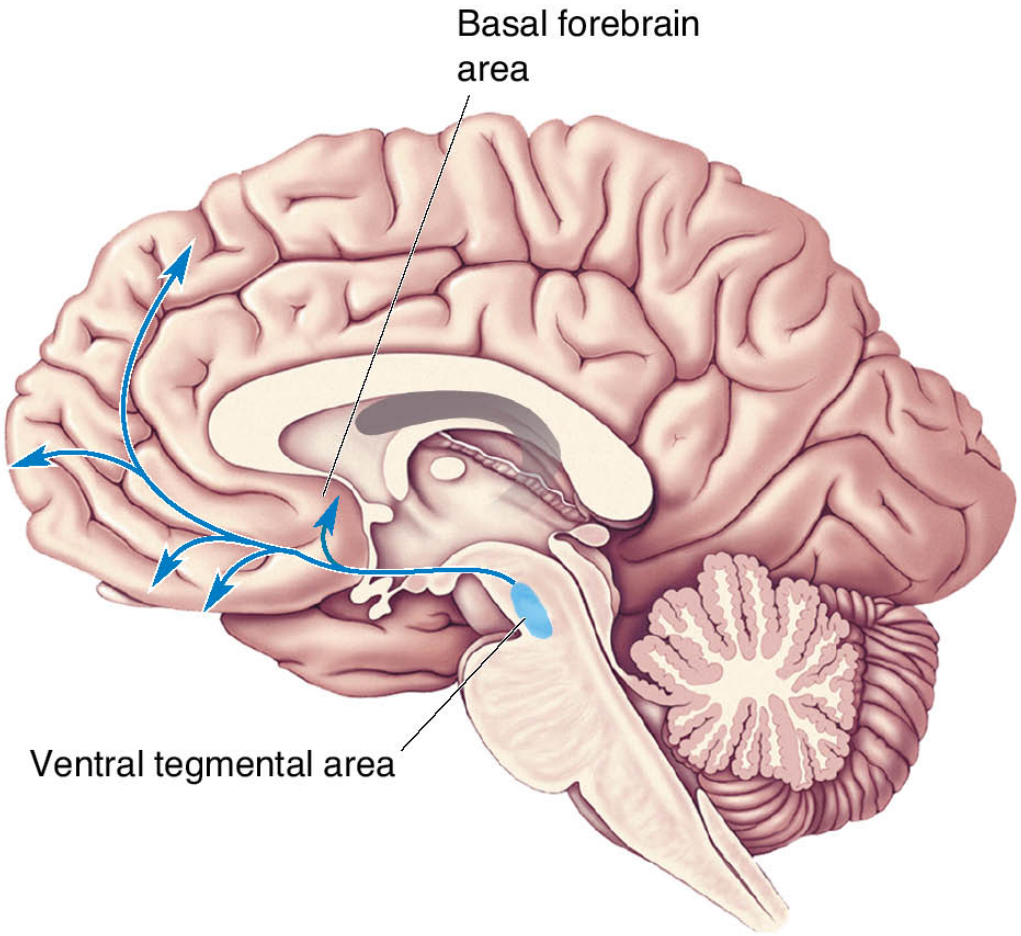


Figure A
Addictive drugs act on the dopaminergic pathway from the ventral tegmental area to the nucleus accumbens. (Source: Adapted from Wise, 1996, p. 248, Fig. 1.)

Receptors for opioids, in particular for the endogenous opioids (endorphins) are found throughout the nervous system. They can inhibit pain transmission at various sites: e.g., spinal cord and midbrain (PAG = periaqueductal gray).



Reward System: Human



Human mesocorticolimbic dopaminergic system (the nucleus accumbens is not shown).

Reward System: Human

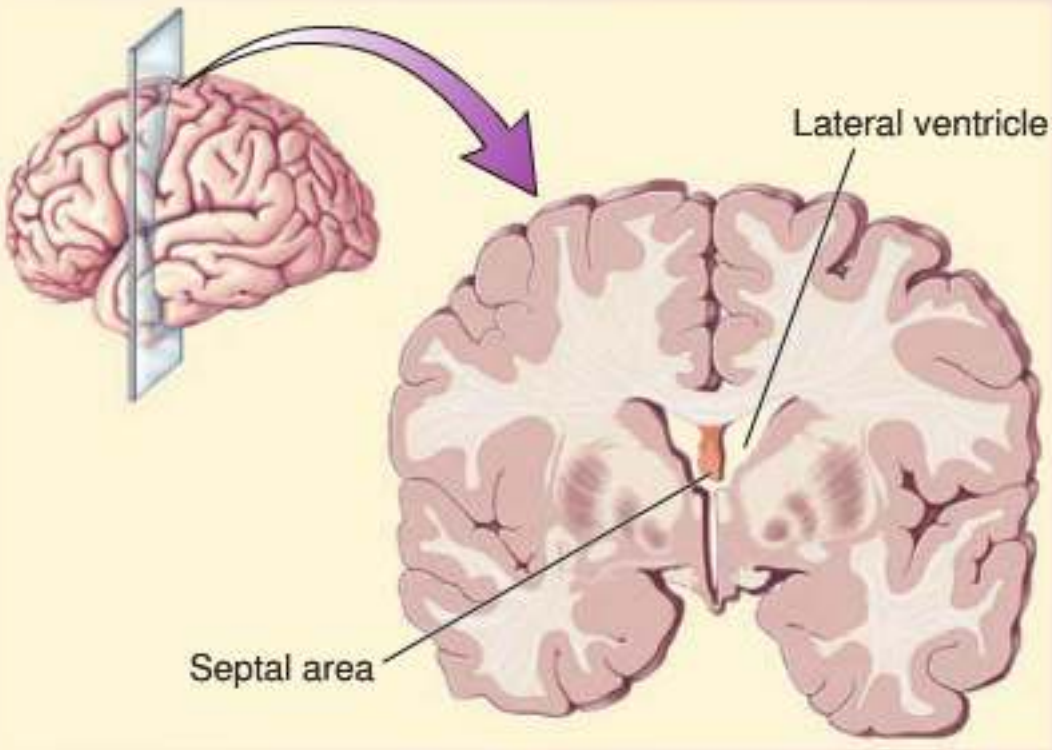


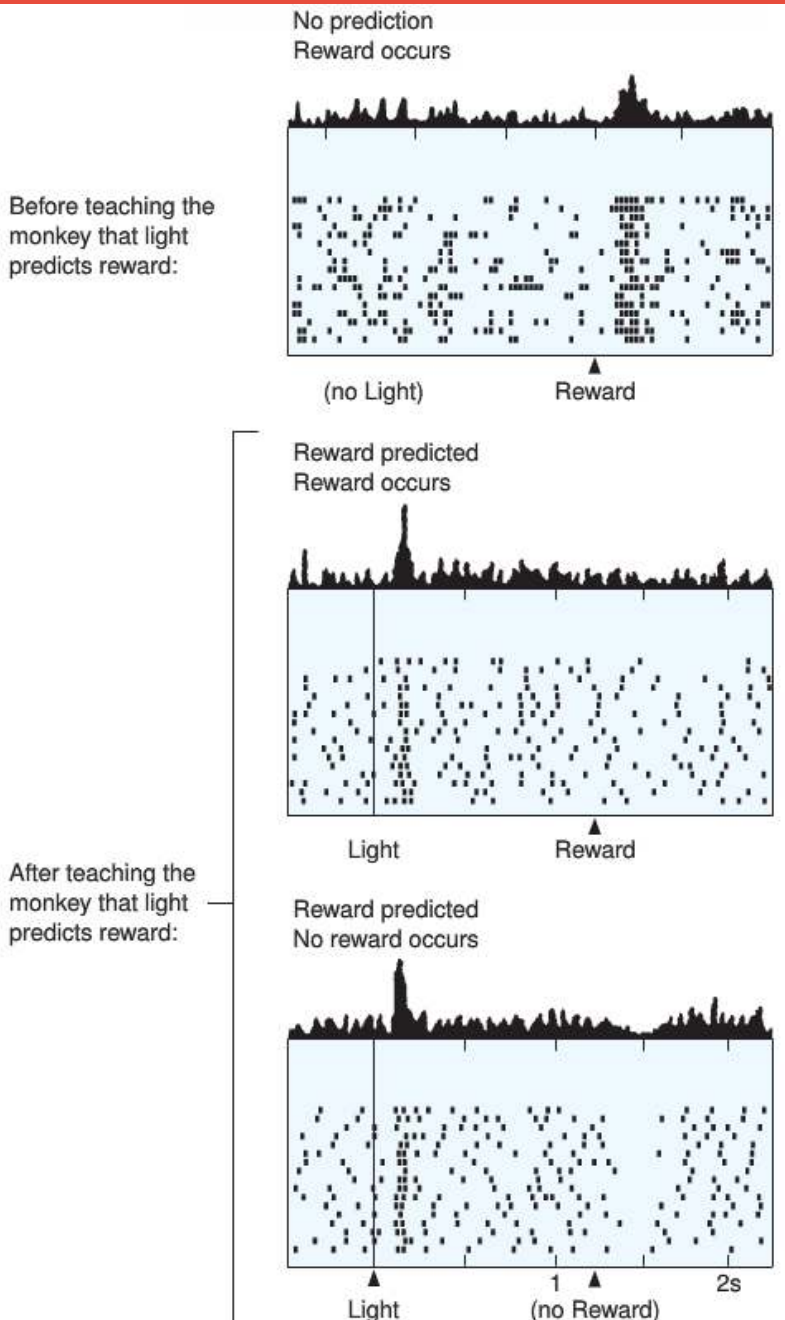
Figure A
The septal area, a site of electrical self-stimulation in humans, is in the rostral forebrain below the lateral ventricle.

Human patient studies were not so clear:

One patient felt unpleasant when midbrain tegmentum was stimulated, another had pleasant feelings.

Stimulation of the septal area led to sexual feelings ("approaching orgasm").

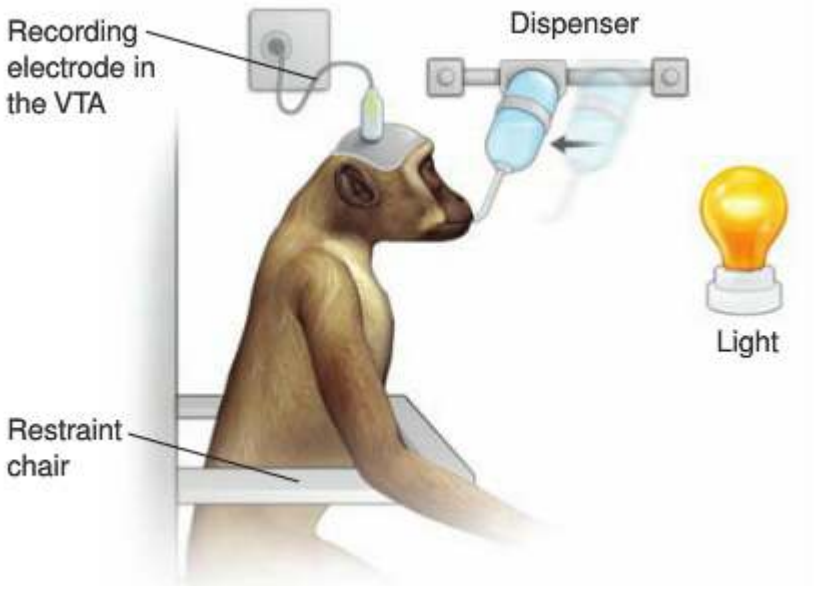
Reward Prediction



Neurons in the ventral tegmental area fire when an unexpected reward (juice) occurs (left top).

When a light predicts the reward, they will fire when the light stimulus occurs, but not for the reward (left center).

If after a light no reward occurs, the neurons will show inhibition, firing below the spontaneous firing rate (left bottom).



Motivation

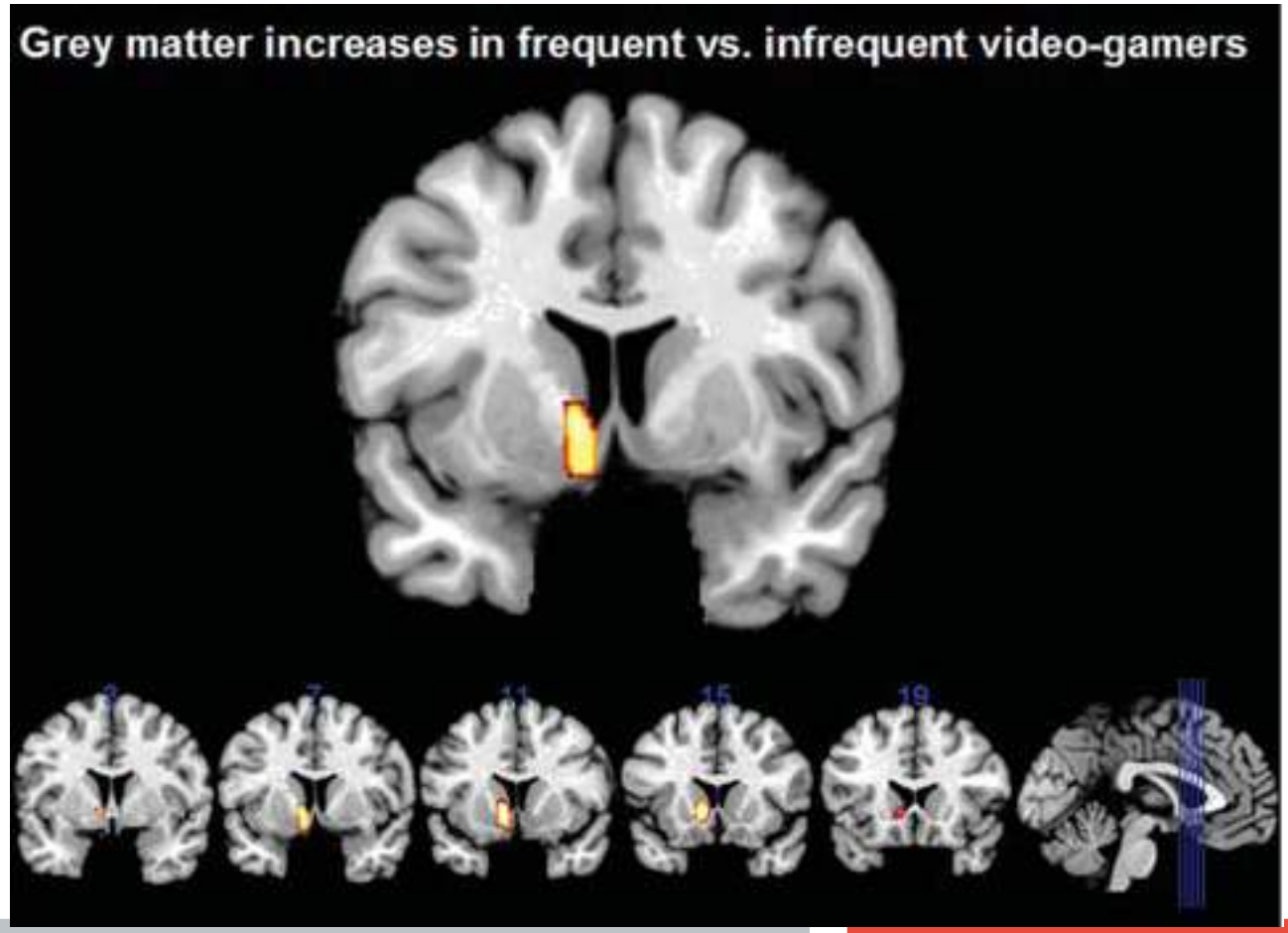
So, what makes people spend so much time on computer games?

Different brains in gamers?

So, what makes people spend so much time on computer games?

Gamers have increased grey matter (neurons) in gamers' ventral striatum (nucleus accumbens): dopamine reward system.

The enlargement could be because of the gaming, but it could also be that enlarged ventral striatum leads to more gaming (it might feel more rewarding).



Kühn, Translational Psychiatry, 2011

“Runner’s High”

Some people describe euphoric feelings, reduction of anxiety, and reduction of pain sensitivity when running for a longer period. Why is that?

Recent studies suggest that endocannabinoids – “endogenous cannabis” might play a role (Fuss et al., PNAS, 2015): In mice, anxiety (tested by their willingness to stay in an illuminated place when a dark hideout is available, dark-light box) is reduced by previous running. Mutated mice that lack the receptor for endocannabinoids do not show reduced anxiety after running.

Summary: Motivation

Motivation

- Homeostasis: Organism needs to keep itself alive
  Importance of the hypothalamus
  Example: Feeding behavior

- Reward system: Mesocorticolimbic dopamine system
  involved in “wanting” and reward prediction