Section 7
Emotion & Learning

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COGS 17 A05

02/06/25

NOTICE

NEXT WEEK (MON June 9)

3:00-4:20 Midterm 3 (125 Pts) AND 4:30-5:50 Final Exam

Emotion

Theories of Emotion

James-Lange Theory (1880s)

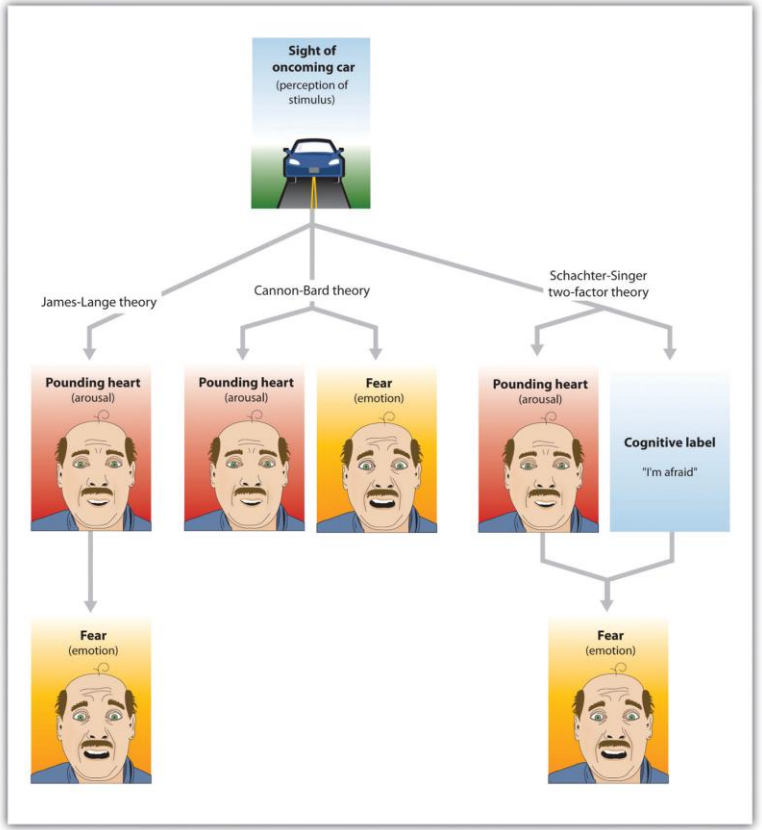
- Emotion = After-the-fact-label assigned to physiological arousal
- Subjective “feelings” are an interpretation we make of our body’s reaction to stimuli (🚙 → 🫀 → 😱)

Cannon-Bard Theory (1930s)

- Once threat is perceived, visceral & subjective experience of emotion is simultaneous (🚙 → 🫀 + 😱)
- via Perceptual input via Thalamus to Cortex and activation of the ANS (for somatic responses)

Schachter-Singer Theory (1980s)

- Contemporary model based on neuroscience
- Emotion = Interaction between cognitive appraisal and autonomic/limbic activity (🚙 → 🫀 + 🧠 → 😱)
- Physiology determines how strong emotion is, but identifying emotion depends on a cognitive appraisal of situation



Theories of Emotion

Q. The **Cannon-Bard** theory proposed that…

a. the brain arouses the body and triggers emotions simultaneously.

b. the physiological arousal occurs first, triggering emotions.

c. emotions trigger a physiological reaction in the body.

d. physiological reactions and thinking work together to trigger emotions.

Theories of Emotion

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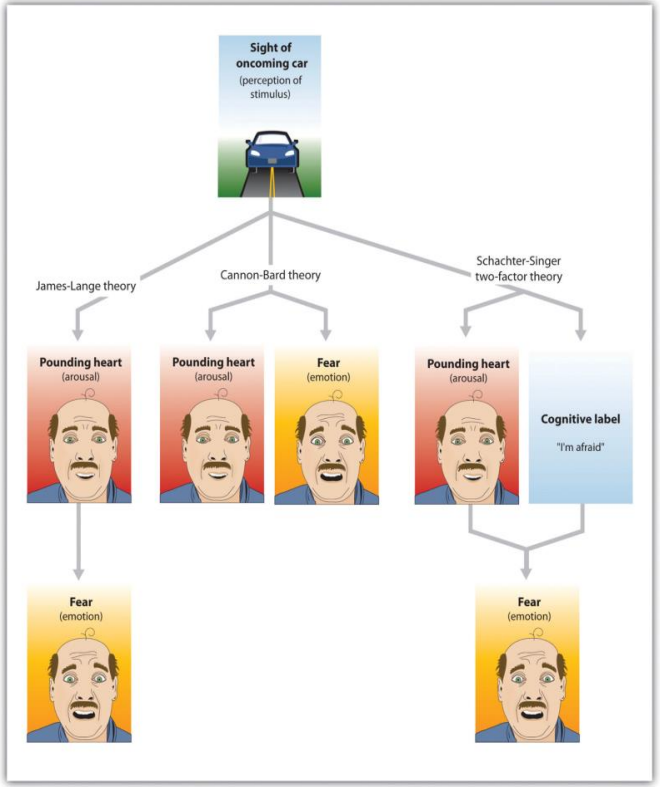
+ Q. Can you find descriptions for **James-Lange & Schacter-Singer theories**?



Theories of Emotion

Emotional Behavior

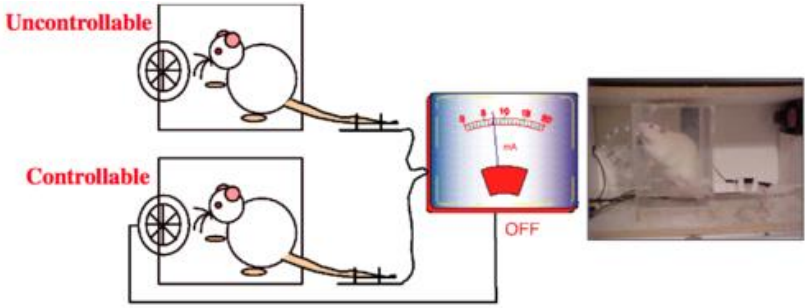
- Judgments of Emotional Stimuli
  - Subjects injected with arousing drug (amphetamine) have an exaggerated emotional response to stimuli

- **Facial Feedback**
  - Holding pen in teeth to put face muscles in smile-like configuration → judge comics as funnier
  - Subjects with specific muscle changes (e.g., "Raise brows") without using emotional terms → reported "feeling" emotions and physiological changes as well (e.g., fear: heart rate up, skin temperature down)

Emotional Behavior

Learned Helplessness

- Condition 1: A rat with a running wheel learns that it can stop the shock stimuli by running in wheel
- Condition 2: The rat given the same shock but without wheel option → becomes depressed ('There's nothing I can do…" or such appraisal of a situation) and developed ulcers from the stress of the shocks
- If the prefrontal cortex is removed, the rat in Condition 2 will not become depressed or develop ulcers → appraisal requires higher level cortical activity



Two groups of rats receive tailshock from the same source, The controllable-shock group can terminate the shock by turning a wheel that is mounted in the front of the chamber. The uncontrollable-shock group receives exactly the same amount of shock, but cannot terminate the shock.

Expressing Emotions

Universals

: 6 basic emotions with corresponding facial expressions

Neonatal imitation

- basic emotional expressions mimicked by newborn
- We are pre-wired to practice manipulating the muscles of emotional expression based on behavior of others

… with culture-specific “display rules” for <u>when</u> and <u>whom</u> you may show what kinds and the extent of emotions
→ may depend in part on learned associations

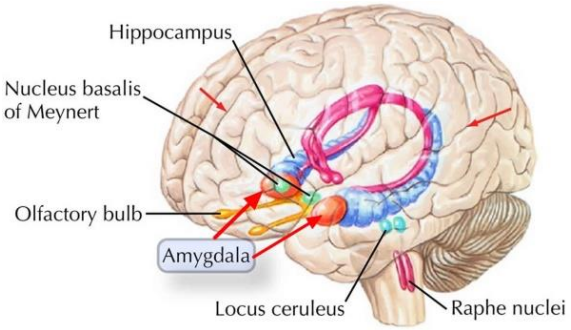
The Limbic System: Amygdala

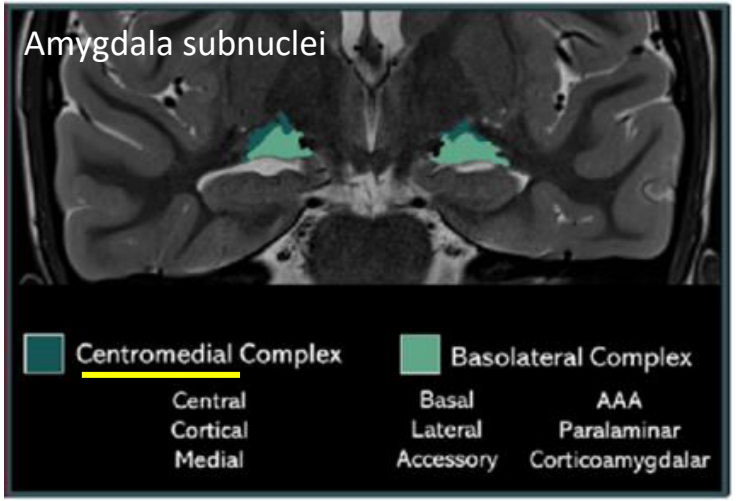
- 'Emotional Center' of the brain: critical in producing & interpreting emotions

- Next to the anterior horn of the hippocampus, directly connected to basal ganglia

- Multiple nuclei with various functions and patterns of connections

1. **Corticomedial** area
   - Direct stimulation leads to anger and rage - most primal emotion that is critical to survival
   - Rabies virus likely affects this area





The Limbic System: Amygdala

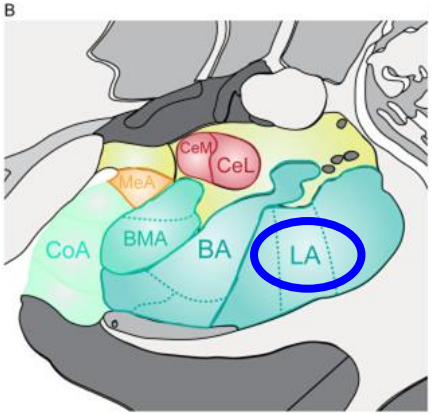
2. Lateral Nuclei

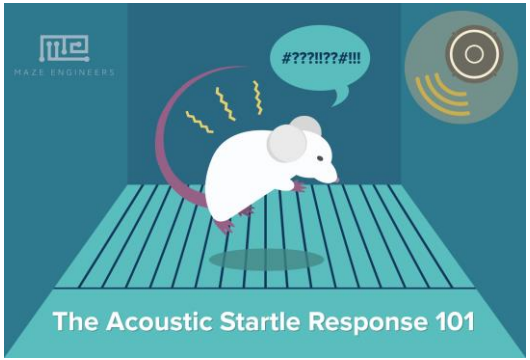
: Startle reflex (e.g., jumping and hunching shoulder, pushing arms from the floor) is influenced by amygdala connections

1) from pain fibers and Visual/Auditory input: to trigger startle reflex, and to detect and learn negative associations

2) to Central gray (= periaqueductal gray) area of midbrain
- part of tegmentum for motor control, esp. neck muscles
- e.g., clench these muscles to help protect fragile cervical neurons near surface

3) to hypothalamus: influences Autonomic NS response (e.g., increase blood pressure, heart rate)



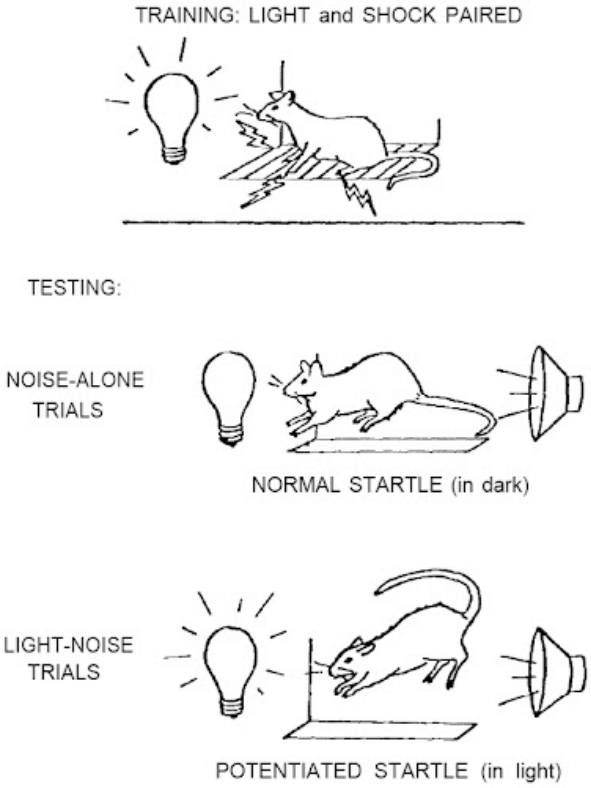


The Limbic System: Amygdala

3. Central and Basolateral Nuclei

- for “conditioned fear” via integrating sensory info (e.g., vision + pain)
- Unlearned startle reflex (e.g. loud noise → !!) can be modified via learning
  - Light 💡 + paired with a noxious stimulus (e.g., shock ⚡) light alone does not elicit Startle Reflex
  - but… once association is learned, shock enhances Startle Reflex (i.e. jump higher to noise if light on)
  - alternatively, when associated with pleasant stimulus, it will decrease Startle Reflex (e.g., 💡 + soothing warmth 🌻 → not jump as high to noise when the light is on)

>> Emotionally-laden stimuli (if it’s not too intense) is remembered better via connections to/from hippocampus



The Limbic System: Amygdala

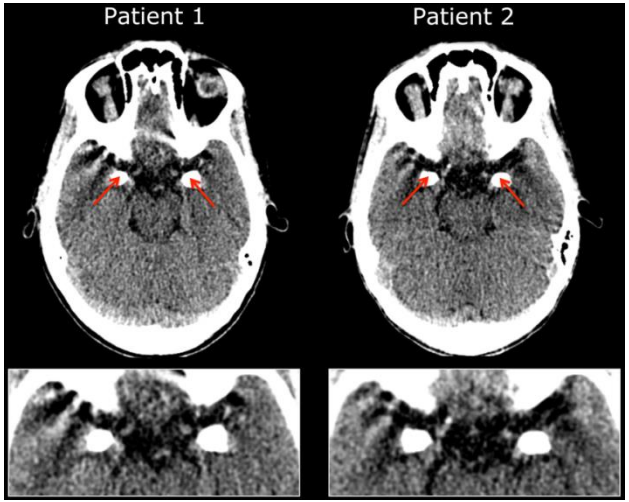
- Shared Emotion: Amygdala is not just active when feel/express but also when observe emotion in others

- **Urbach-Wiethe Disease** involves the calcification of the amygdala
- Symptoms

1) Patients exhibit a "flattening of affect" with less emotional expression, generally does not experience strong likes/dislikes

1) Impaired ability to recognize facial expressions, esp. fear and untrustworthiness

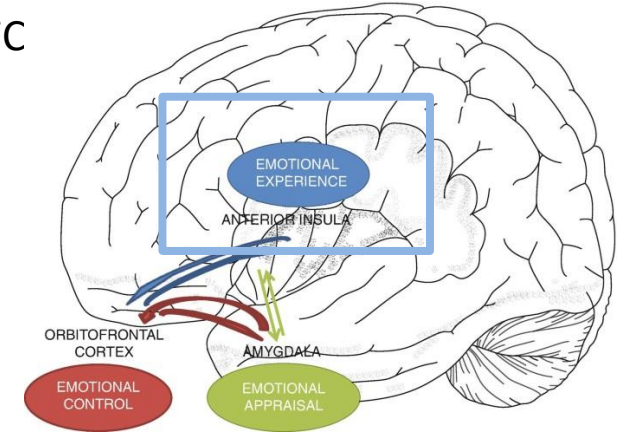
1) Recognizes individuals, gender, but have trouble naming emotions, judging if face is "approachable" or not



bilateral calcification damage to the amygdala in Urbach–Wiethe patients

Anterior Insula

- on the path from amygdala to the orbitofrontal cortex (OFC
- connected to hindbrain cranial nerves for control of facial muscles
- includes primary Gustatory Cortex
- vital for **emotional expression in social contexts**



“Emotional Facial Paresis”

- Damage to the left <u>anterior insula</u>
- Can produce a full smile on command but <u>doesn’t</u> spontaneously smile in a funny social situation

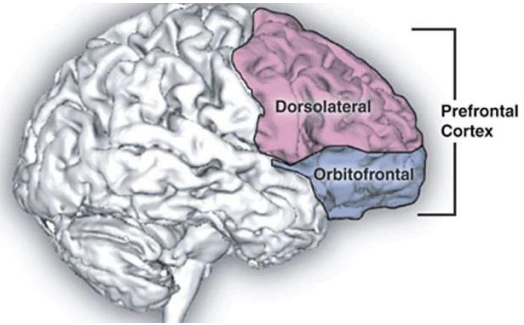
vs. “Volitional Facial Paresis”

- Damage to the right <u>motor cortex</u>
- <u>Cannot</u> produce a smile on command but can spontaneously smile

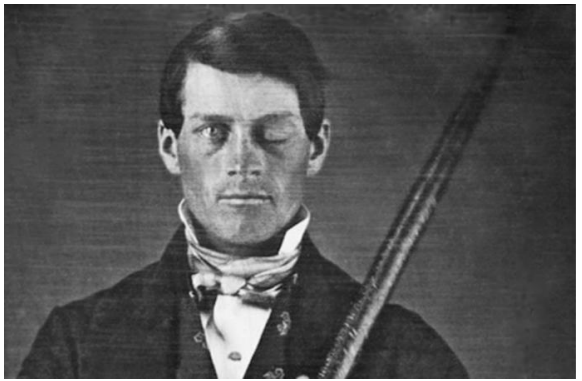
On command Spontaneous

Prefrontal Cortex

- Area anterior to the premotor cortex
- Particularly important for regulating social and emotional behavior (executive functions)
- Damage will lead to loss of inhibition, socially inappropriate behavior, sudden aggression, etc



- **Phineas Gage**
  - Sep 13th, 1848 (25 yo), was when accident happened where Steel rod damaged most of PFC
  - Cognitive abilities remain intact but radical change in personality



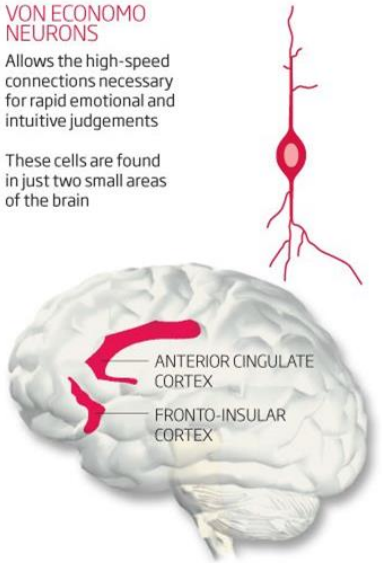
“Popular reports of Gage often depict him as a hardworking, pleasant man before the accident. Post-accident, these reports describe him as a changed man, suggesting that the injury had transformed him into a surly, aggressive heavy drinker who was unable to hold down a job, many of his acquaintances explained that after the injury, he was "no longer Gage."

Phineas Gage

Functions of Prefrontal Cortex

- **Theory of Mind**
  - Ability to attribute mental states to self and others
  - Inappropriate reciprocal connections between the OFC-amygdala have been implicated in Autism

- **Von Economo Cells, 'Spindle" Cells**
  - Specialized pyramidal neurons with long fibers, but branch little
  - e.g., connect Anterior Insula with Anterior Cingulate Cortex (social risk, cost/benefit analyses)
  - Found only in large brained animals (humans, elephant, whales 🧑‍🤝‍🧑 🐘 🐳)



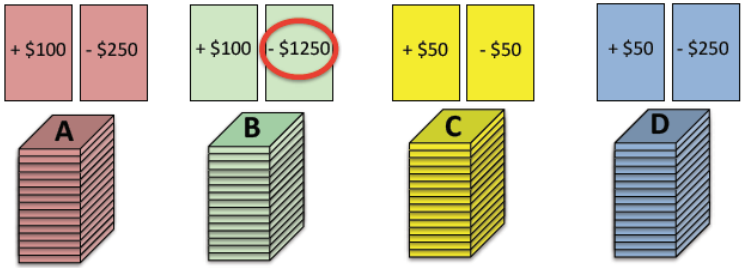
Gambling Task

- Compare groups: Normal Control, Amygdala-Lesioned, Prefrontal-Lesioned
- "Pick cards from one of 4 piles to learn about each pile's payoff/penalty"

- **Normal Control**
  - Shows anticipatory anxiety for picking from Pile B
  - Feels bad at penalty
  - shift to better, even before can explain decision

- **Prefrontal Lesioned**
  - X anticipatory anxiety or shift
  - Some negative emotion towards penalties
  - Does not learn to avoid Pile B

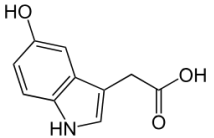
- **Amygdala Lesioned**
  - X anticipatory anxiety
  - X negative emotion towards penalties
  - Does not learn to avoid Pile B

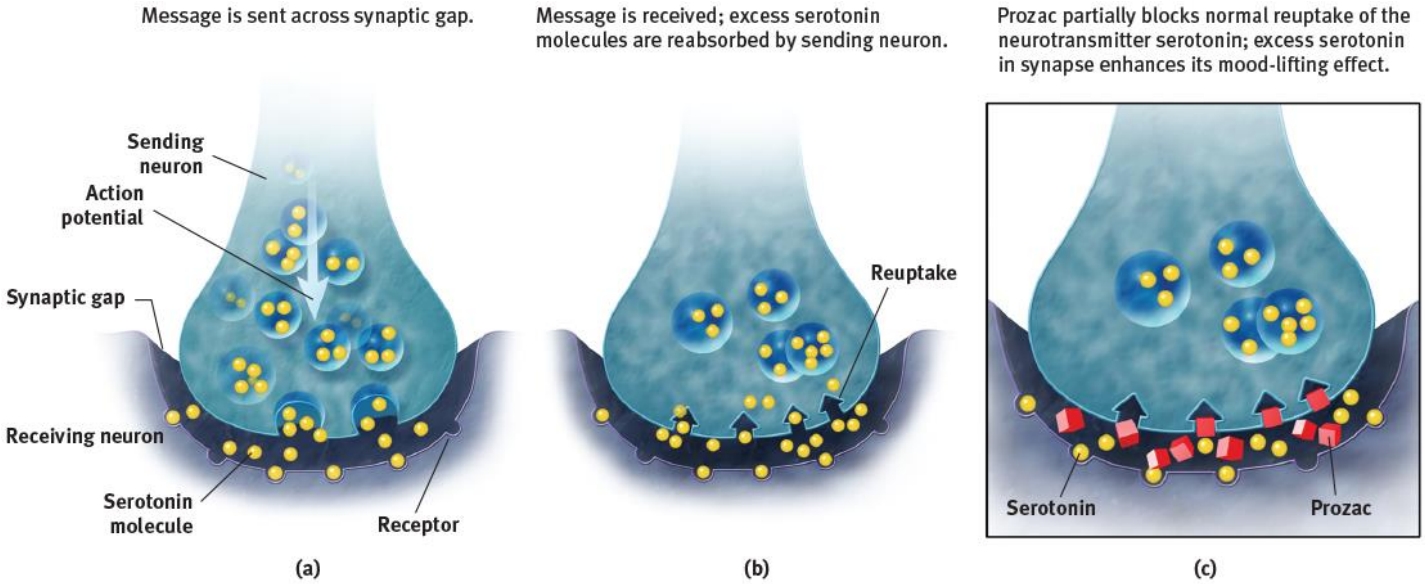


	Feel Bad at Penalty	Show Anticipatory Anxiety	Shift from Worst (B)	
Normal	+	+	+	
Prefrontal Lesion	+	−	−	Don't Learn!
Amygdala Lesion	−	−	−	

Neurotransmitters and Emotion

1. **Serotonin (5HT) in Amygdala**
   - Reuptake of Serotonin into presynaptic cell, creates a metabolic byproduct 5-HIAA
   - Low levels of 5-HIAA in bloodstream: linked w/ impulsivity and depression
   - **Antidepressants** like Prozac **blocks reuptake of 5HT**, keeping **serotonin active in synapses longer**
   - But, long-term use will ultimately make you produce less serotonin

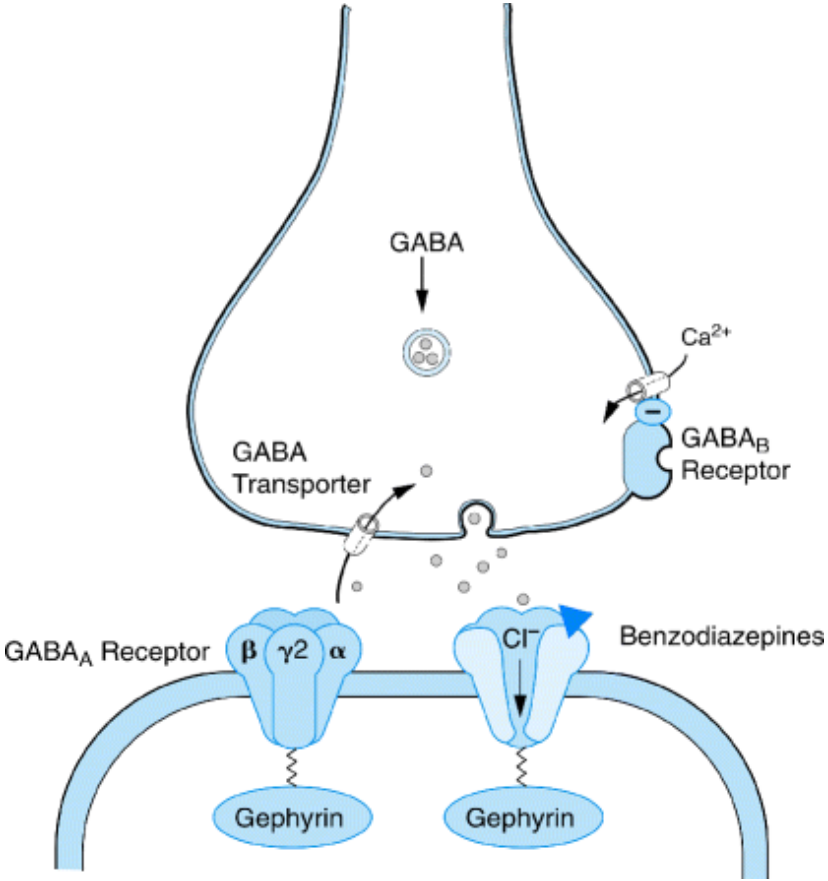




Neurotransmitters and Emotion

2. GABA

- Inhibitory effect on post-synaptic cell by admitting Cl- ions (hyperpolarized cell, more negative)
- in amygdala, this helps suppresses startle reflex and reduce anxiety
- Anti-anxiety drugs like Benzodiazepines (e.g., Valium) act as GABA agonists
    - BZD binds to GABA receptors, increasing inhibitory effect of GABA (more Cl- coming into the cell)
    - enables GABA to bind more easily and longer



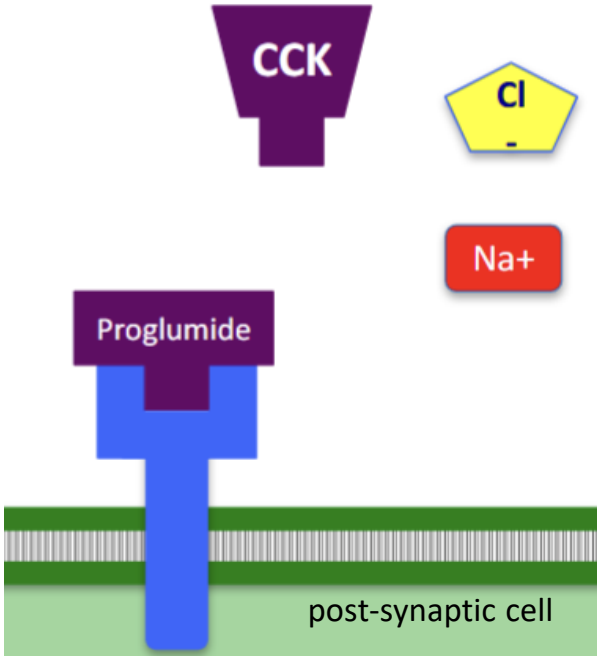
3. Cholecystokinin (CCK)

- Stimulates post-synaptic cells by opening Na+ gates

- CCK in amygdala: involved in learned enhancement of startle reflex
- CCK in hypothalamus: suppresses hunger when blood sugar rises

- Some diet pills are CCK agonists that mimic CCK effects
  - Side effects involve increases levels of anxiety

- CCK-antagonists (e.g., Proglumide for treating Ulcers) can block receptor sites w/o opening Na+ gates in the hypothalamus. This has a calming effect in the amygdala but promotes overeating



Learning

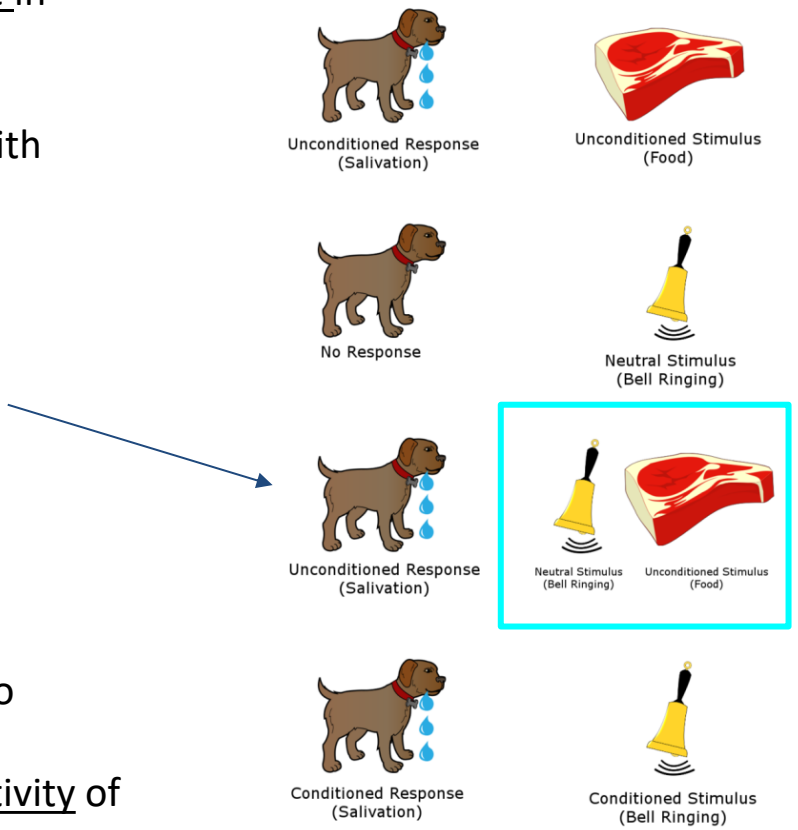
Learning

- Generic Definition: Development of a permanent change in behavior based on experience

- **Law of Effect:** Any stimulus/action/context associated with positive reinforcement will tend to repeat

- **Classical** Conditioning (1897)
  - Learning happens when the two stimuli are linked together
  - Pavlov's Dog
  - Bell & Food must **co-occur** for the conditioning to succeed

- **Temporal Contiguity**
  - Both types of conditioning require co-occurrence to become associated in the learner's mind
  - Proposed that co-occurrence leads to neural co-activity of the stimulated circuits, thus leading to learning

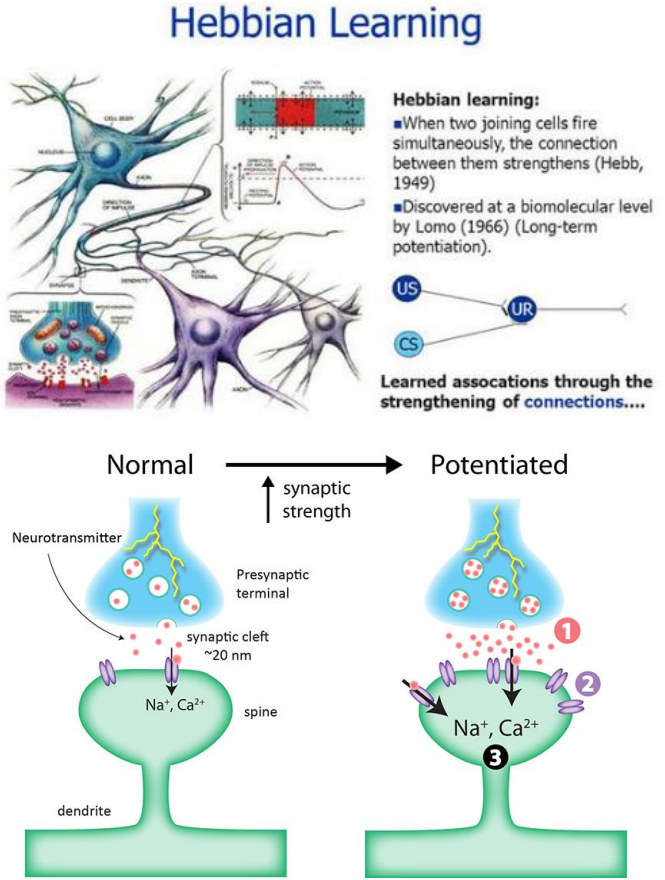


Hebbian Learning

- Proposed by Hebb as the fundamental neural process involved in learning

- *Neurons that fire together, wire together*
  - Repeated co-activated neural circuits, involved in learning and retrieval of associations, are reinforced and increases the likelihood of circuits co-firing. **"Hebbian Synapse"**
  - by structural/metabolic changes in NT availability, release, and reception

- **Long-Term Potentiation (LTP)**
  - Key mechanism underlying learning
  - Over time, LTP can lead to (semi-) permanent structural and connectivity changes among neurons
  - Ultimately, increases the likelihood of activity along repeated circuits



Long-Term Potentiation (LTP)

- Typically changes occur on the post-synaptic neuron
  - Changes to # of receptor sites
  - Dendritization (building new dendritic branches), etc.

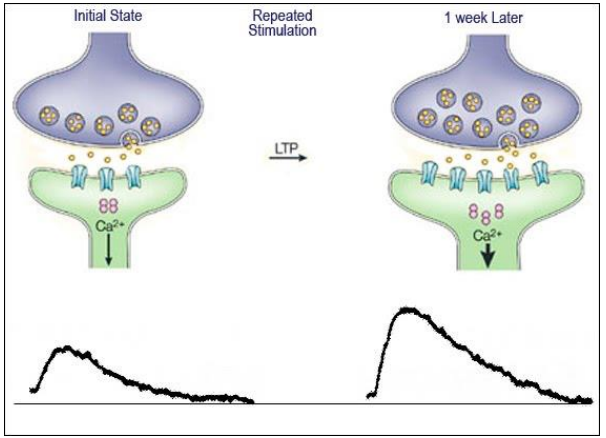
- The best-studied mechanisms of LTP use Glutamate as the primary NT

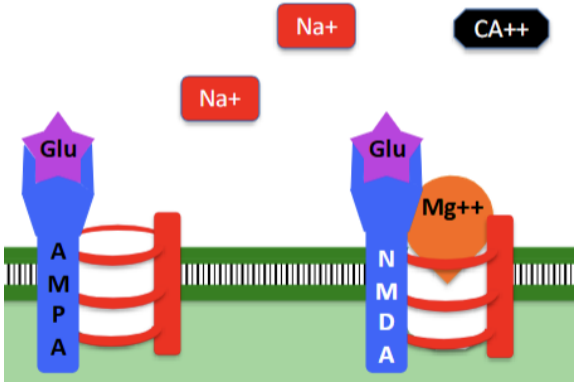
- 2 types of Receptor Sites for Glu

1. **AMPA** Receptors
   - Glu attaches → ion gates open to admit Na+ and CA ++
   - Pumps Mg++ out of the cell/receptor sites

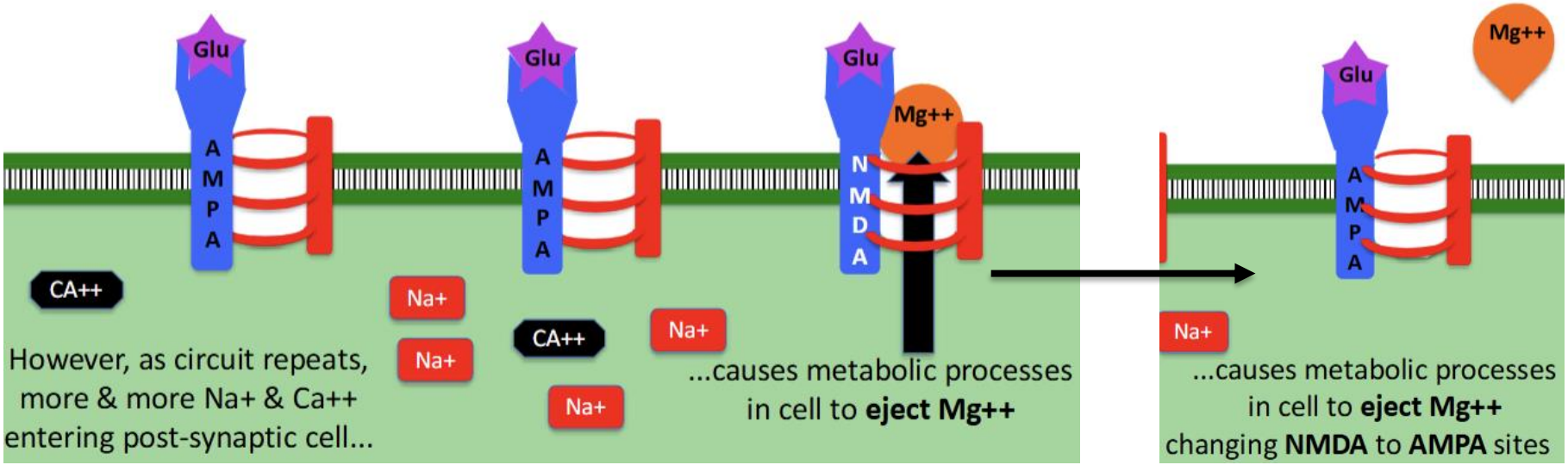
2. **NMDA** Receptors
   - In contrast to AMPA receptors, when Glu attaches to NMDA sites Mg++ blocks the NMDA channel

- **But… as circuit repeats,** more Na+ and Ca++ enter the post-synaptic cell → causes metabolic processes in cell to eject Mg
- **Result: 1)** NMDA receptors are converted into AMPA, 2) build new dendritic branches



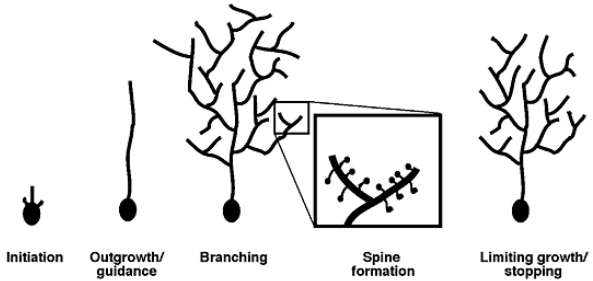


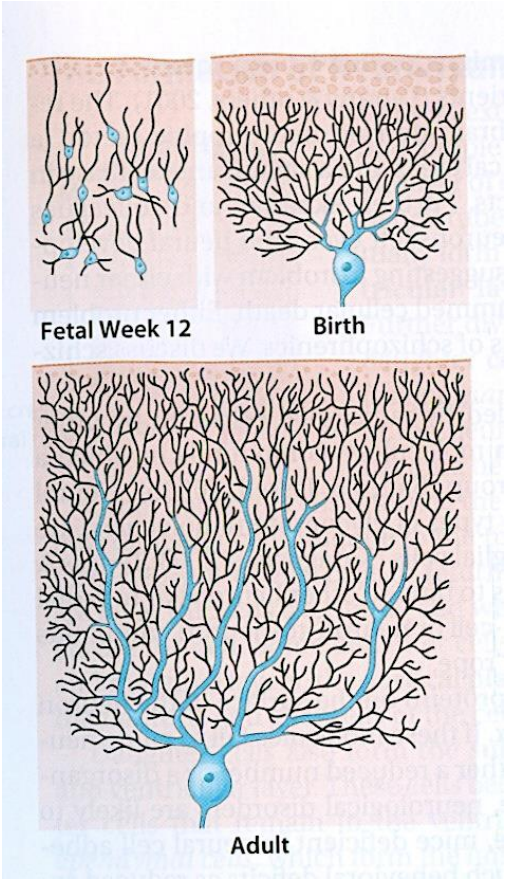
Long-Term Potentiation (LTP)



Dendritization

- Significant experiences (e.g., learning new languages, how to play guitar etc) over time changes how neuron looks like
- How? It introduces physical changes in cells: increasing **# of branches** which increase the **# of receptor sites**
- Repetition is the key: Continued activity along circuit → growth of new dendritic branches
- In some memory circuits, **retrograde messengers** (e.g., Nitrous oxide) are released by **post-synaptic cells**, throughout life, to create a positive feedback loop and prolong release of NTs by pre-synaptic cells
    - RECALL: exceptional case which does not usually happen after birth





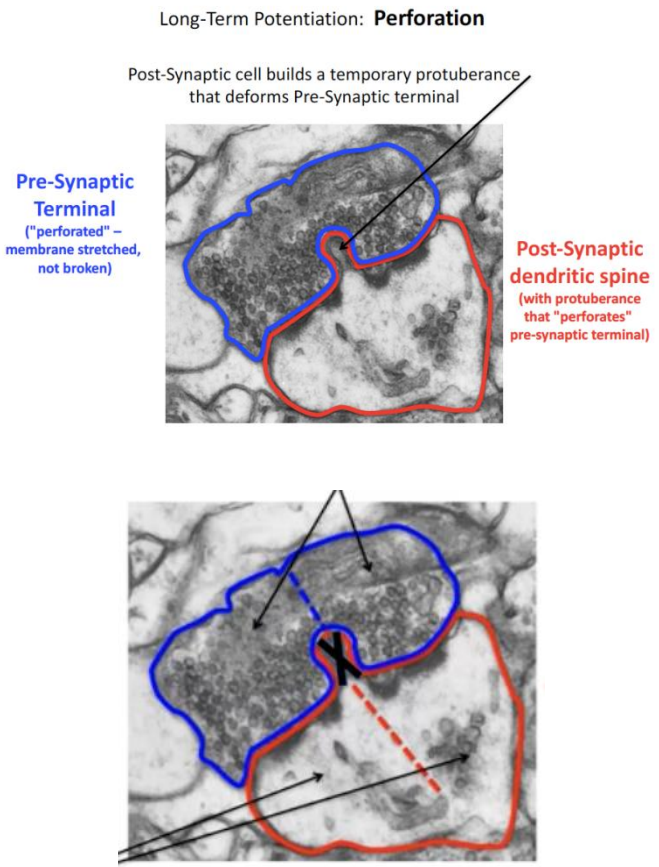
Perforation (Splitting of Synapses)

- Occurs where the terminal button meets the axons of the next cell

- **Post-synaptic** (red) **branch will grow protuberance** pokes into the pre-synaptic (blue) terminal (membrane of the pre-synaptic terminal is stretched but not broken)

- Results of Perforation
  1) duplicates the metabolic mechanisms in both sides
  2) promotes the **<u>division</u> of the pre-synaptic terminal into two terminal buttons**

- The dendrites then dismantles the protuberance and divides into two dendritic spines that each receive NT from one of the new terminal buttons



So close to the end of the quarter now!