### COGS 17 \* Neurobiology of Cognition Lecture 10: Emotion

Theories of Emotion Emotion is hard to study and even to define...

James-Lange Theory (1880s) ... is the label we give after-the-fact to autonomic arousal and associated behavior - i.e. Our subjective "feelings" are an interpretation we make of our body's reaction to stimuli

Cannon-Bard Theory (1930s) Once threat perceived, visceral & subjective experience of emotion is simultaneous - Via perceptual input via Thalamus to Cortex & activation of the ANS (for somatic responses)

Schacter-Singer Theory (1980s) ... is the interaction between cognitive appraisal and autonomic/limbic activity

- Physiology determines how strong emotion is, but ID'ing emotion depends on a cognitive appraisal of situ

- Feedback between variety of brain regions involved (e.g. Prefrontal Cortex, Amygdala, Hypothalamus, etc!)

- That is, influence moves both bottom up (visceral to cognitive) AND top-down (cognitive to visceral) Behavioral and neurological data support aspects of all of these:

- e.g. Subjects given arousing drug (e.g. amphetamine) - or placebo - and shown scary, funny, or sad images - Aroused subjects all reported stronger emotion, but type reported varied with stimuli

- e.g. Subjects hold pen-in-teeth vs. pen-in-lips while judging comics; Rate them funnier if teeth exposed

- e.g. Direct subjects to make specific changes in facial muscles (e.g. Raise brows, pull them together, raise upper evelids, tighten lower evelids, stretch lips horizontally) w/o mentioning an emotion

- When asked, subjects reported "feeling" appropriate emotion (e.g. Above expression => fear)

- Expressions also produced changes in heart-rate, skin temp, etc. (e.g. Fear: heart up, temp down)

- e.g. Even in rats, individual's cognitive appraisal will influence it's autonomic response

- Learned Helplessness under duress can lead to overactive Parasympathetic Rebound.> ulcers

- 2 rats voked together, both receive same shocks to tail, until 1 runs in its wheel (other: no wheel)

- Rat who runs does NOT develop ulcers, but rat w/o active response DOES (even the = shock)

- But if non-active rat has Prefrontal lesion, does not appraise self as helpless, does not develop ulcers

COMPLEX! Emotion plays role in all evaluative (discrimination, motivation, learning) & communicative processes

- May depend in part on learned associations, but basic emotional expressions mimicked by newborn humans - So, from its inception, emotional expression is shaped by social interaction

- PLUS: Facial expressions of basic emotions are similar across cultures, although "display rules" vary

Many brain areas are implicated in the generation and control of emotional behavior...

- Including array of structures of Limbic System, and the Thalamus, the Hypothalamus, and the Frontal Cortex

- Here, we will focus on role of Amygdala (part of Limbic System) and Frontal Cortex

NOTE: Much more research has been done on responding to aversive stimuli than to attractive ones

- We will discuss circuits involved in "positive reinforcement" in lecture on Learning & Memory

**Amygdala** - Stimulation of particular regions (most of which interact with each other) leads to typical reactions

- **Corticomedial** Area => Primed to **Attack**, prolonged inclination toward aggression

- e.g. Rabies, from a virus that attacks, especially, temporal region of brain => extreme aggression

- Lateral Nuclei => Startle Reflex Sudden loud noise => Freeze, Tense neck to protect spine, Inc heart rate - Startle Reflex is influenced by Amygdala connections...

=> from Pain fibers, and Visual and Auditory input, to detect and learn negative associations

=> to Central Gray Area of Midbrain = Part of Tegmentum for motor control, esp of neck muscles

=> to Hypothalamus => Influences Autonomic NS response (e.g. inc blood pressure, heartrate, etc) - Recall that 2 of the Hypothalamic "4 Fs" = Fighting & Fleeing

- Central & Basolateral Nuclei => Conditioned fear, via integration of sensory info (e.g. vision + pain)

- <u>Unlearned</u> Startle Reflex (e.g. loud <u>noise</u> > !!) can be further <u>conditioned</u> (<u>modified via learning</u>)

- If visual stim (e.g. light) is paired w/noxious stimulus (e.g. shock) light alone comes to elicit fear

- Once association is learned, paired signal enhances the reflex (i.e. jump higher to noise if light on)

- In fact, any unpleasant stimulus (e.g. disturbing photo, sound or odor) presented simultaneously, whether or not previously paired with unconditioned (noxious) stimulus, enhances Startle Reflex

- Alternatively, a paired signal previously associated w/pleasant stimulus will decrease Startle Reflex - e.g. Light paired w/soothing warmth >> Not jump as high to noise if light is on
- All of above aid memory of emotionally-laden stimuli, via connections to/from Hippocampus

- i.e. Emotion-provoking (esp aversive) images or words, if not too intense, remembered better than neutral

- Contemporary research on Amygdala focused on shared emotion

- Amygdala active not just when feel/express but also when observe emotion in others
  - e.g. In many primates, infants learn what to fear by watching/imitating mother's reaction

- Urbach-Wiethe Disease = degenerative calcification in Amygdala, (or other damage) can lead to...

- Impaired ability to recognize Facial Expression, especially fear and untrustworthiness
- Patients recognize individuals, gender, but trouble naming emotion, judging if face is "approachable"
- May be related to disinterest in eyes; forced to consider eyes only, judgments are a bit better
- Patients generally placid, do not experience strong likes/dislikes, act less timid in strange environment

- Overall, <u>Amygdala</u> seems particularly involved in <u>mediating critical soc (e.g. aggression, mating) interactions</u> and learning/responding to these and other potentially threatening or exciting stimuli

Anterior Insular Cortex (medial to anterior Temporal Lobes; also includes primary Gustatory Cortex)

- Damage => impairs recognition & production of <u>Disgust</u>; Co-opted in humans as a <u>social</u> reaction
- This area also connected to hindbrain cranial nerves for control of facial muscles
  - Damage=>Patients can show teeth voluntarily, but <u>cannot spontaneously smile</u>

# = Emotional Facial Paraesis

- Contrast to Primary Motor Cortex for facial area and/or its connections to facial nerves

## - Damage=> Patients can spontaneously smile, but <u>cannot show teeth voluntarily</u>

### = Volitional Facial Paraesis

**Frontal Cortex** - Has <u>many reciprocal connections with **Amygdala**</u>, moreso than any other cortical area - Important in expressing, inhibiting and reading emotion...

- <u>Prefrontal Cortex</u> Damage > loss of inhibition, socially-inappropriate behavior, sudden aggression
  - Famous example: **Phineas Gage**, survived steel rod through cheek, in/out of Prefrontal Cortex
  - Cognition ok but "personality" radically changed, irresponsible, volatile, indifferent to consequences - Contemporary work implicates Prefrontal in **Theory of Mind** = attribute knowledge, emotion to others
    - Inappropriate <u>Prefrontal-Amygdala</u> interaction may be critical in Autism (involves ToM deficits)
      ? May result from premature devel of Prefrontal, *before* sufficient connections with Amygdala?
- Gambling Task: Consider diff performance of Normals, Amygdala-Lesioned &Prefrontal-Lesioned subjects - Task: Pick cards from 4 piles to learn about each pile's payoff/penalty profile
  - 2 have good payoffs but very bad occasional penalties. Other 2 have smaller payoffs & smaller penalties -After some experience with this task...
    - Normals feel anxiety before taking from bad piles & shift to better, even before can explain decision
    - Prefrontal-Lesioned Do not develop anticipatory anxiety or shift, tho show neg emotion at penalty
    - <u>Amygdala-Lesioned</u> No anticipatory anxiety, no shift, no feeling negative emotion at penalty
- Von Economo ("Spindle") Cells
  - Long fibers, but <u>branch little</u> for communication between distant brain areas w/out intervening influence
  - Found only in large-brained animals (humans, elephants, whales)
  - e.g. Connect Anterior Insula with Anterior Cingulate (social risk, cost/benefit analyses)

<u>Neurotransmitters</u> implicated in the generation and control of emotional behavior:

<u>Serotonin (5HT) Turnover</u> = reuptake & resynthesis, as determined by levels of metabolite **5-HIAA** in blood - Low Serotonin Turnover associated with increased levels of impulsive behavior including...

- Sudden aggression, rapid cocaine addiction, no pause at choice pts, poor delayed gratification, etc.
- Also associated with (esp seasonal disorders that can lead to) depression & suicide
- Recall that drop in Serotonin associated w/crankiness that arises when overdue to begin sleep cycle - Anti-depression drug **Prozac** blocks 5HT re-uptake, prolonging synapse
- Though problematic long-term, since, when use less serotonin, produce less, exacerbating problem
- <u>GABA</u> = Inhibitory NT, hyper-polarizes cell by admitting **Cl- ions**, affects emotion in Amygdala & Hypothalamus - Most anxiety-reducing (axiolytic) drugs are Benzodiazepines (e.g. Valium, Xanax), act as **GABA-agonists** 
  - Most GABA receptor sites also bind Benzo's, enables GABA to bind more easily and longer
  - Note: Decreases both own experience of anxiety and how perceive anxiety in others!
  - In contrast, blocking of GABA sites in Amygdala can lead to Panic Attacks

## <u>CCK</u> - Stimulation of (excitatory) CCK sites in <u>Amygdala</u> => <u>enhanced Startle Reflex</u>

- CCK an NT/Hormone also involved in appetite suppression, released after eating when blood sugar rises
  - Some diet pills are CCK-agonists w/side effect of anxiety; Antagonists calming, but promote overeating