Ingestive Behaviors

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class 18

Ingestive behavior

Why worry about ingestive behaviors?
- Can be the *symptoms* of many psychological disorders
    o Mind and body need to be in balance
    o Psychological dysfunctions may cause biological dysfunctions: drinking (alcohol), eating (anorexia, bulimia), sexual…
        ▪ Video: WGBH NOVA: Dying to be thin
    o Eg; depression
- Cen be the *source* of psychological disorders
    o Metabolic encephalopathy: dementia, seizures
    o Alcohol→ liver degeneration→ lack of estrogen breakdown→ feminization
    o Pickwickian syndrome: type of obesity accompanied by irritability, aggressiveness, jealously, suspiciousness
        ▪ Charles dickens' The pickwick papers

Homeostasis and Regulation 11.1
- Body: needs proper nutrition (stored in fat, muscle...)
- Mind/brain: need proper neurochemical environment (hormones, ions...)
    o Homeostasis: process used to regulate the mind/brain-body balance (to correct and compensate for variations)
        ▪ Process of regulations
            ● System variable (actual)
            ● Detector
            ● Set point (desired)
            ● Mechanism to change the variable
                o Negative feedback loop

Homeostasis and regulation: drinking 11.2
1. Body loses water
2. Detectors signal loss of water
3. Drinking occurs
4. Stomach fills with water sends signal to brain
5. Satiety mechanism inhibits further drinking (fast loop ~ seconds)
6. Water is absorbed: body fluids back to normal (slow loop ~20 min)
- Drinking trigger= low fluid detectors
- Drinking termination=satiety mechanism (fast signals in anticipation of the slow signals). Partly established through experience.

Ingestive behaviors: drinking 11.3
- 4 fluid compartments
    o 1. Intracellular: 2/3 of body water
    o Extracellular
        ▪ interstitial: between cells

- ▪ intravascular: in blood vessels
- ▪ Cerebro-Spinal fluid (CSF)
- Vascular balance
  - o Blood/plasma volume is regulated independently. Important for a good functioning of heart and kidneys
  - o Loss of blood volume: <u>hypovolemia</u>. Dehydration, blood loss, severe burns
  - o Too much blood volume: urination. Edema (Feet + ankles)
  - Exceptions: pregnant women, about 50% more blood stored by vasodilation. Increased cardiac output and rate.
- Intracellular Vs. Interstitial balance 11.4
  - o Tonicity= concentration of solutes (e.g. ions)
  - o Cell membranes are permeable to water not solutes
  - o Hypertonic Vs. Isotonic Vs. hypotonic
  - o Solution a: in hypertonic to solution B; water is drawn out of solution B
  - o Solution C is hypotonic to solution B; water is drawn into solution B
  - o Solution A ← solution B←Solution C
- 2 types of thirsts 11.5
  - o Osmometric thirst: when interstitial tonicity increases (e.g. after a salty meal)
  - o mechanical detection of changes in tonicity: Osmorereceptor
    Osmoreceptors are located in the hypothalamus, near the third ventricle (AV3V)

Osmorereceptor in AV3V 11.6
- cell volume increase→ membrane potential decreases→firing rate decreases
- cell volume decrease→membrane potential increases→firing rate increases

Osmometric thirst: AV3V 11.7
- experiment: injection of hypertonic saline in humans
- just after drinking: Anterior Cingulate cortex and AV3V are active. Few minutes after drinking, only AV3V remains active
  - o Anterior Cingulate is part of the satiety system (~seconds) AV3V contains the osmoreceptors (~minutes)
- AV3V→ <u>Median Preoptic Nucleus</u> → drinking

Ingestive Behaviors: drinking 11.8
- Volumetric thirst: when blood plasma volume decreases
  - o Loss of blood→loss of water + loss of sodium = Hypovolemia
- Renin/angiotensin system: 'blood flow' detector in the kidneys
- Angiotensin (blood)→subfornical organ (SFO)→<u>Median Preoptic nucleus</u> → drinking

Neural Substrate of Drinking: the big picture
- <u>Median preoptic nucleus</u>: is a site of integration of thirst information

Ingestive behaviors: eating
- <u>Some facts about weight</u>
  - o 60% of Americans 20 years older or older are overweight. 30% are obese
  - o Mexican and African Americans have 10% more incidence for overweight/obesity
  - o American Indians with most obesity are in AZ
  - o Cities with most obesity: Huston, New Orleans

- o Cities with least obesity: San Diego, Boston, Tucson
- o Reason???: environmental factors (e.g. exercise), attitude factors (e.g. brain/psychology related)

Energy set point theory
- Hypothesis: hunger is a low energy detection signal. Eating starts when the signal decreases bellow a given set point
- Energy, fuels
  - o Glucose: carbohydrates(calories).
  - o Fatty acids: Fat.
  - o Amino acids: proteins

Ingestive behaviors: eating 11.10
- 2 fuel storage systems
  - o 1%- short term: carbohydrates (e.g glycogen). IN liver and muscles
  - o 85%- long term: triglycerides, in fate cells
  - o 14%- long term amino acids
- short term: the energy rush
  - o glucose is the only source of energy of the CNS/PNS (neurons and glia). Detected in PET scans
  - o extra glucose is used to form triglycerides
  - o glucagon secreted early morning when glucose is low
  - o glucagon stimulates the release of energy from long-term storage
- long term storage: dieting/prolonged fasting
  - o triglycerides (fat tissue)→ glycerol + fatty acids
    - ▪ glucose   body
    - ▪ brain
  - o triglyceride breakdown under neural control (sympathetic system) and hormonal control (glucagon and adrenaline).
  - o The brain has priority!
    - ▪ PNS-CNS cells; passive absorption of glucose
    - ▪ Other cells: need insulin receptors and active glucose transport
- Long term storage: the benefits of exercising 11.11
    - ▪ Muscles proteins → amino acids→body
  - o Absorptive and fasting phases: summary

Ingestive Behaviors: Control 11.12
- Starting a meal
  - o Cultural signals: eating can be a social behavior
  - o Eating can be triggered by learned external signals (Pavlovian conditioning, sight of food)
  - o Eating can be triggered by hormonal (Ghrelin) signals from the stomach and duodenum (part of small intestine). Injections of Ghrelin with trigger thoughts of food/eating.
  - o Ghrelin secretion is inhibited by digestive activity
- Starting a meal: metabolic signals 11.13

- o Stomach not necessary to feel 'hungry'
- o Glucoprivation. Hypoglycemia: glucose does not enter the cells
- o Lipoprivation: fatty acids are not converted to fuel
- o Evidence:
    - ▪ Glucoprivic hunger
        - ● Injection of 2-DG (blocks glucose entry into cells)→hunger
        - ● Lesion of vague nerve→ ~~Hunger~~
    - ▪ Lipoprivic hunger:
        - ● Injection of drugs that blocks fatty acid metabolism →hunger
        - ● Lesion of vagus nerve → ~~hunger~~

<mark>Conclusion: liver and Vagus nerve monitor glucose and fat levels and send signals to the brain to start a meal</mark>

- Ending a meal: satiety 11.15
    - o Gastric factors: nutrient receptors in the stomach. Stretch receptors in the stomach
    - o Intestinal factors:
        - ▪ Detection of fat in duodenum –(CCK)-> - stomach (Decrease outflow)
            - ▪ Bile (increase fat break-down)
            - ▪ Brain (stop signal via Vagus nerve)
        - ▪ PPY released by the duodenum after a meal proportional to calories ingested.
            - Injections of PPY→ less caloric intake
            - PPY provides a satiety signal
    - o Liver factors: glucose receptors→ satiety signal in brain Via Vagus nerve
    - o Pancreatic facto: absorptive phase. Insulin→ insulin receptors in brain → satiety signals (hypothalamus
    - o Cephalic (cognitive) factors: learning to identify rich foods
- Long term satiety: fatty storage factors 11.16, 1..17
    - o Leptin secreted by fat cells, increase fat metabolism
    - o OB mouse: leptin deficient
    - o Injecting leptin→smaller meals
        - ▪ Leptin Is a (fat related) satiety signal