Ingestive Behavior

I. Physiological Regulatory Mechanisms
II. Drinking and Thirst
III. Eating and Metabolism
IV. Signals that Start a Meal
V. Signals that Stop a Meal
VI. Brain Areas Involved in Hunger & Satiety
I. Physiological Regulatory Mechanisms
Figure 11.1
An example of a regulatory system
I. Physiological Regulatory Mechanisms

• Negative feedback: the effect of an action serves to stop that action
• Satiety: the feeling of fullness
II. Drinking and Thirst
Intracellular fluid: 67%

Extracellular fluid:
- Interstitial fluid: 26%
- Intravascular fluid (blood plasma): 7%
- Cerebrospinal fluid (less than 1%)
Terminology

• Solutes: the substances that are dissolved in a solution
• Examples of solutes: ions such as sodium, chloride, & potassium dissolved in water
• Force of diffusion: molecules moving from areas of high concentration to areas of low concentration
• Molecules: ions and the water itself
Equal concentration of solute on both sides, so no net change.
Add water to one side.

Water molecules pass through semipermeable membrane, leading to equal concentration of solute on both sides. Concentration of solute is lower (on both sides) than it was before.
Add salt (NaCl) to one side.

Water molecules on left cross membrane to approach equal solute concentration on both sides, despite the influence of gravity.
Which way will the water move?

<table>
<thead>
<tr>
<th>Interstitial Fluid</th>
<th>Intracellular Fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Isotonic:</strong></td>
<td><strong>H₂O</strong></td>
</tr>
<tr>
<td>Solutes equally concentrated inside &amp; out</td>
<td>Solutes equally concentrated inside &amp; out</td>
</tr>
<tr>
<td><strong>Hypotonic:</strong></td>
<td><strong>H₂O →</strong></td>
</tr>
<tr>
<td>Solutes less concentrated relative to inside</td>
<td>Hypertonic: Solutes more concentrated relative to outside</td>
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<tr>
<td><strong>Hypertonic:</strong></td>
<td><strong>H₂O ←</strong></td>
</tr>
<tr>
<td>Solutes more concentrated relative to inside</td>
<td>Hypotonic: Solutes less concentrated relative to outside</td>
</tr>
</tbody>
</table>
Equilibrium

Equal concentrations of solute in both containers

Only water molecules pass through the semipermeable membrane.
Add salt

Higher concentration of solute

Equilibrium

Higher concentration of solute than before
Add water

Lower concentration of solute

Equilibrium

Lower concentration of solute than before
Two fluid areas that our homeostatic system pays very close attention to:

1. Intracellular fluid
2. Intravascular fluid

• It’s possible for these fluid levels to vary independently, so it turns out that there are two types of thirst...
Two Types of Thirst

• Osmometric/Osmotic thirst: loss of fluid from the cells

• Causes:
  – Evaporation
  – Eating a salty meal (pure osmometric thirst)
Two Types of Thirst

• Volumetric/Hypovolemic thirst: loss of fluid from the blood

• Causes
  – Evaporation
  – Bleeding (pure volumetric thirst)
(a) Hypovolemic thirst
Baroreceptors in major blood vessels detect any pressure drop from fluid loss.

(b) Osmotic thirst
Osmosensory neurons in the brain detect any increased osmolality of extracellular fluid.
Osmometric Thirst & Osmoreceptors near the AV3V

Increased solute concentration of interstitial fluid causes osmoreceptors to lose water and shrink in size.
Volumetric Thirst

• Baroreceptors in 1.) the blood vessels of the kidneys and 2.) the atria of the heart
• When they aren’t stretched out (due to lack of fluid in the blood), they send signals indicating that water levels are low
• One of these signals takes the form of a hormone...
Hypovolemia

Reduced flow of blood to kidneys

Kidney

Renin

Angiotensinogen

Angiotensin I

Angiotensin II

Retention of sodium

Retention of water

Increase in blood pressure

Salt appetite

Drinking
Volumetric Thirst

- ... and the other signal takes the form of a “hard wired” neural connection
- Atrial baroreceptors send signals about lack of fluid up into the brain via neural connections
III. Eating and Metabolism
The Fasting Phase

• Defined: No nutrients in the digestive system (stomach and intestines are empty)
• Key hormones regulated by the pancreas: glucagon high and insulin low
The Fasting Phase

- Short Term Store: the liver
- Stored energy: glycogen (animal starch)
- Energy: glucose (for CNS)
- Glucagon converts glycogen to glucose
The Fasting Phase

- Long Term Store: adipose tissue (fat cells)
- Stored energy: triglycerides
- Energy: fatty acids (for PNS) & glycerol/glycerine (which can be converted into glucose for CNS)
- Glucagon converts triglycerides into fatty acids and glycerol/glycerine
The Absorptive Phase

- Defined: Nutrients are available from the digestive system
- Key hormones regulated by the pancreas: **glucagon** low and **insulin** high
The Absorptive Phase

- High levels of **insulin** allow all cells (CNS & PNS) to use **glucose**
- **Insulin** converts excess **glucose** into **glycogen** in the liver
- **Insulin** converts excess energy into **triglycerides** in the adipose tissue
IV. Signals that Start a Meal
Social/Environmental Factors that Effect Hunger

• Number of people present
• Time of day
The brain cannot metabolize fatty acids; receptors detect only glucose levels.

The liver can metabolize glucose and fatty acids; receptors detect levels of both nutrients.

Signal to brain via vagus nerve
Physiological Factors that Affect Hunger

• Peripheral Nervous System (PNS)
  – Lack of sugar (glucoprivation)
  – Lack of fat (lipoprivation)
  – Detected by the liver

• Central Nervous System (CNS)
  – Lack of sugar (glucoprivation)
  – Detected by neurons in area postrema and the nucleus of the solitary tract (NST)
V. Signals that Stop a Meal
Short-Term Satiety Mechanisms (act over the course of minutes)

- Head factors
  - how food looks, smells, tastes, & feels
- Stomach
  - distention and nutrient receptor activation
- Intestines
  - Duodeenum & CCK
- Liver
- Insulin
Long-Term Satiety Mechanisms (act over the course of days/weeks/months)

- Adipose tissue (fat cells) & the hormone leptin
ob/ob mouse lacks leptin
People sometimes lack leptin too (1 in 2,000)
V. Brain Areas Involved in Hunger & Satiety

• Question: where in the brain would you expect hunger to be regulated?
• Answer: the hypothalamus
Hunger in the Brain

- Decreased insulin secretion, decreased breakdown of fatty acids, decreased body temperature
- Brain stem nuclei that control ANS
- Excitatory effects on eating, reduction of metabolic rate
- Lateral hypothalamus

NPY/AGRP

- Arcuate nucleus
- Ventrolateral medulla

MCH

- Endocannabinoids facilitate release of MCH and orexin

Orexin

- Lateral hypothalamus

Stomach

- Ghrelin secretion increases when stomach empties
- Glucose-sensitive neurons in medulla (and liver?)
Ghrelin fluctuation during the day

![Graph showing ghrelin levels throughout the day with peaks at breakfast, lunch, and dinner.](image)
Satiety in the Brain: The Ventromedial Hypothalamus (VMH)
Satiety in the Brain
Figure 9.17 Hypothalamic Nuclei Participate in the Control of Hunger  
The initiation and cessation of feeding behavior may result from the activity of four important nuclei within the hypothalamus: the lateral hypothalamus, the ventromedial hypothalamus, the arcuate nucleus, and the paraventricular nucleus, shown here in a human brain. In (a), the structures are shown from a sagittal view, and in (b), from a coronal section.
Learning & Memory

I. The Nature of Learning
II. Learning and Synaptic Plasticity
III. Perceptual Learning
IV. Stimulus-Response Learning
V. Relational Learning
I. The Nature of Learning
Definitions of Learning

• Psych 100:
  – A relatively long lasting change in behavior or potential behavior that is due to experience.

• This Class:
  – The process by which experience changes our nervous system and ultimately our behavior.
Types of Learning

• Perceptual Learning
• Stimulus-Response Learning
  – Classical Conditioning
  – Operant Conditioning
• Motor Learning (special case of Stimulus-Response Learning?)
• Relational Learning
  – Spatial Learning
  – Episodic Memory
  – Observational Learning
The Hebb Rule

- A synapse will be strengthened (more easily activated and/or produce larger depolarizations) if...
  1. a synapse is repeatedly active when...
  2. the post-synaptic neuron is firing.
II. Learning & Synaptic Plasticity
Long-Term Potentiation (LTP)

• Defined: A long-term increase in the excitability of a neuron to a particular synaptic input caused by repeated high-frequency activation of that input.

• In other words: if you use a synapse a lot in a short period of time, it will strengthen.

• Though found to occur in numerous brain areas, LTP was initially demonstrated in the hippocampal formation
Figure 12.16 The Hippocampus and Its Associated Structures
LTP Version 1.0

To septum, mammillary bodies

Schaffer collateral axon

Field CA3

Mossy fiber

Dentate gyrus

Record from dentate gyrus

Stimulate axons in perforant path

Entorhinal cortex

Fimbria

Schaffer commissural axon

Field CA1

Axon in perforant path

Subicular complex
LTP Version 2.0
LTP Version 3.0: Associative LTP

Field CA3
Weak stimulus
Record EPSP
Strong stimulus
Field CA1
Dentate gyrus
Entorhinal cortex
Action potential reaches terminal button of strong synapse; produces strong EPSP (depolarization) in pyramidal cell.

Dendritic spine

Dendritic spike washes back along dendrite; primes NMDA receptors in dendritic spines.

Action potential reaches terminal button; glutamate is released.

Long-term potentiation: synapse is strengthened.

Depolarization is sufficient to trigger action potential in axon of pyramidal cell.

Axon

Action potential in axon

Dendrite of pyramidal cell

Strong synapse
If a molecule of glutamate binds with the NMDA receptor, the calcium channel cannot open because the magnesium ion blocks the channel.
Depolarization of the membrane evicts the magnesium ion and unblocks the channel. Now glutamate can open the ion channel and permit the entry of calcium ions.
Figure 12.13  
**Chemistry of Long-term Potentiation.** These chemical reactions appear to be triggered by the entry of an adequate amount of calcium into the dendritic spine.
Long Term Depression

• A long-term decrease in the excitability of a neuron to a particular synaptic input caused by stimulation of the terminal button while the postsynaptic membrane is hyperpolarized or weakly stimulated

• In other words, if you don’t use a synapse, with will weaken (“use it or lose it”)
III. Perceptual Learning
Object-Memory Task (inferior temporal lobe more active)

Spatial-Memory Task (parietal lobe more active)
IV. Stimulus-Response Learning
Tone (CS) → Aversive stimulus (US) → Strong synapse → Synapse strengthened by pairing of CS and US

Central nucleus

Conditioned emotional responses: hypothalamus, midbrain, pons, and medulla
Debiec et al. (2002)

Train → 45 days → Inject drug → Test: Good memory

Train → 45 days → CS → 90 sec → 1 day → Inject drug → Test: No memory
Reinforcement Pathways

- Mesolimbic System: projects from the midbrain to parts of the limbic system, including the nucleus accumbens (NA)
- Mesocortical System: projects from the midbrain to the frontal lobes
- Both rich in dopamine
- Originally thought dopamine release in the NA was responsible for the experience of pleasure
- Now many consider dopamine release to more about desire, craving or wanting
V. Relational Learning

- Amnesias
- Retrograde Amnesia: loss of ability to recall old memories
- Anterograde Amnesia: loss of ability to form new memories
Retrograde Amnesia
Cannot remember events prior to brain damage

Brain damage occurs

Anterograde Amnesia
Cannot later remember events that occur after brain damage

Time
Anterograde Amnesia & the Case of H.M.

• Had hippocampi removed in an attempt to reduce seizures

• Surgery did reduce seizures but afterward, it appeared H.M. could form no new memories
Initial Conclusions Reached

- Is the hippocampus the location of long term memory (LTM)?
  - NO
- Is the hippocampus the location of short term memory (STM)?
  - NO
- Is the hippocampus involved in moving new memories from STM to LTM?
  - YES
Two items from the incomplete-pictures test. H.M.'s memory for the 20 items on the test was indicated by his ability to recognize the more fragmented versions of them when he was retested. Nevertheless, he had no conscious awareness of having previously seen the items.
Revised Conclusions Reached

• Is the hippocampus the location of long term memory (LTM)?
  • NO

• Is the hippocampus the location of short term memory (STM)?
  • NO

• Is the hippocampus involved in moving new **CONSCIOUS** memories from STM to LTM?
  • YES
Anterograde Amnesia Redefined

• A failure of declarative/conscious/relational memory
Spatial Memory & LTP

 Constant start position
(stimulus–response task)

Hidden platform
Language & Communication

I. Speech Production
II. Speech Comprehension
III. Aphasias in the Deaf
IV. Reading Disorders
I. Speech Production
Broca & Patient Tan
I. Speech Production

• Broca’s Area

• Broca’s Aphasia
  – Anomia
  – Articulation difficulties
  – Agrammatism
Broca’s Aphasia & Agrammatism

Though they primarily have production problems, people with Broca’s aphasia can also show minor comprehension problems.
II. Speech Comprehension

• Wernicke’s Area
Wernicke’s Aphasia

• Core symptoms:
  - Fluent but meaningless speech
  - Poor speech comprehension
Two Aspects of Speech Comprehension

• Recognition: knowing that a sound you’ve heard is part of language you know

• Comprehension: understanding what a particular sound means
A Recognition Deficit: Pure Word Deafness

- Poor auditory speech comprehension (reading is fine)
- Speech production is normal
- Produced by damage to Wernicke’s area

Dear Dr. Wellington,

Thank you for your letter of September 20. I shall be pleased to be at your office between 10 to 12 am on Friday 1st October.

I still find it very odd to be able to write this letter but not to be able to read it back a few minutes later. I much appreciate the opportunity to see you.

Yours very truly,

Harry A.
A Comprehension Problem: Transcortical Sensory Aphasia

• Poor speech comprehension
• Fluent but meaningless speech
• **BUT** can copy speech they hear without understanding it (what differentiates it from Wernicke’s aphasia)
• Produced by damage to the posterior language area
Putting It All Together

• Damage to what area would produce speech recognition problems?
  • Wernicke’s area
• Damage to what area would produce speech comprehension problems?
  • Posterior language area
• If you damage both of these areas, you get...
  • Wernicke’s aphasia
The Dictionary Analogy

• The way the brain accomplishes language can be likened to a dictionary

• Word entries (by sound):
  • Wernicke’s Area
  • Definitions of the Words:
  • All over the cortex
  • Evidence that this is so...
The Dictionary Analogy

• Autotopagnosia/Autopagnosia
• The inability to name one’s one body parts
• Most common: finger agnosia
• Produced with damage to the left parietal lobe
The Dictionary Analogy

• Anomic Aphasia
• Difficulty finding and using particular words
• Often use circumlocutions (they talk around the word they can’t remember)
• Lose nouns with damage to the temporal or parietal lobes
• Lose verbs with damage to... (anyone?)
• Frontal lobes
Lesion results summarized

<table>
<thead>
<tr>
<th></th>
<th>Persons</th>
<th>Animals</th>
<th>Tools</th>
</tr>
</thead>
<tbody>
<tr>
<td>TP</td>
<td>59.8</td>
<td>93.3</td>
<td>96.0</td>
</tr>
<tr>
<td>IT</td>
<td>75.5</td>
<td>80.1</td>
<td>84.5</td>
</tr>
<tr>
<td>IT+</td>
<td>91.7</td>
<td>88.3</td>
<td>78.5</td>
</tr>
</tbody>
</table>
Conduction Aphasia

• What differentiates TSA from Wernicke’s aphasia
• People with TSA can copy or parrot speech
• Suggests there is a copy circuit in the brain: the arcuate fasiculus
• Direct connection from Wernicke’s area to Broca’s area.
Conduction Aphasia

- Own speech is fluent and meaningful (no problems)
- Show good comprehension much of the time
- BUT, they can’t copy what they don’t understand
- Struggle with nonsense speech sounds and “repeat after me” scenarios
- Produced by damage to the arcuate fasiculus
Wernicke’s aphasia is caused by damage to both regions; patients can neither understand the meanings of words nor repeat them.

A direct connection between Wernicke’s area and Broca’s area enables patients with transcortical sensory aphasia to repeat words that they cannot understand.

Transcortical sensory aphasia is caused by damage to the posterior language area; patients cannot understand the meanings of words but can repeat them.
Damage to the arcuate fasciculus disrupts repetition of speech sounds; causes conduction aphasia.

Broca’s area (speech production)

This connection enables patients with conduction aphasia to express their thoughts in words.

Meanings of words

Perceptions and memories

Perceptions and memories
To Broca’s area

Anterior segment

To inferior parietal lobe

Posterior segment

Long segment

To Wernicke’s area
III. Aphasias in the Deaf
IV. Reading Disorders
Alexia

- Defined: the complete inability to read
- Produced by brain damage
Damage to left primary visual cortex causes blindness in right visual field.

Broca's area

Wernicke's area

Extrastriate cortex receives information from left visual field through corpus callosum.

Left primary visual cortex is destroyed.

Information from left visual field

Damage to posterior corpus callosum prevents information from right extrastriate cortex from reaching left hemisphere.

(a)  (b)
Dyslexia

• Defined: difficulty in reading
Types of Reading

• Phonological reading (phonetic coding): recognizing and sounding out letter combinations

• Whole word reading: recognizing the entire word and pronouncing it from memory
Aoccdrnig to a rscheearch at Cmabrigde Uinervtisy, it deosn't mttaer in waht oredr the ltteers in a wrod are, the olny iprmoetnt tihng is taht the frist and lsat ltteer be at the rghit pclae. The rset can be a toatl mses and you can sitll raed it wouthit porblem. Tihs is bcuseae the huamn mnid deos not raed ervey lteter by istlef, but the wrod as a wlohe.
Sight of word

Whole-word recognition

Whole-word reading

Control of speech

Phonetic coding (sounds of letters)

Phonetic reading

Saying word aloud

Letter recognition
Acquired Dyslexias

- Surface Dyslexia
  - Defined: difficulty reading via the whole word method
- Consider these words: may, disk, moon
- Now consider these words: cough, through, rough, bough
- Damage to visual word-form area (VWFA) in the left temporal lobe
Damage to VWFA (left temporal lobe)

Whole-word recognition

Sight of word

Letter recognition

Phonetic coding (sounds of letters)

Control of speech

Saying word aloud

Whole-word reading is damaged

Phonetic reading
Acquired Dyslexias

• Phonological Dyslexia
• Defined: difficulty reading phonetically
• Difficulty sounding out unfamiliar words
• If vocabulary is developed prior to damage, they can often read OK relying on whole word reading
• Damage to left temporoparietal complex
Damage to left temporoparietal complex
登
の
ぼ
る
漢字

かんじ

Kanji
Acquired Dyslexias

• Direct Dyslexia
• Can read but can no longer understand what they are reading
• Damage to left frontal and temporal lobes
• Some people reading via whole word method, others reading via phonetic reading
Developmental Dyslexia

• Difficulty reading that appears to have a biological/genetic basis
• People with developmental dyslexia struggle with reading from the very beginning
• 5%-10% of the population of this country
• Concordance rates for MZ twins between 84% and 100%
Developmental Dyslexia

• Poor phonological awareness
• Example: have a hard time hearing the three “duh”-”aw”- “n” sounds in the word Don
• Biological difference: magnocellular layer of the LGN doesn’t have uniformly large cells
• Magnocellular layer known to be involved in the processing of motion
Developmental Dyslexia

• The handy man versus the plumber
Developmental Dyslexia

• Other related behaviors
• Often have balance/coordination issues
• Hit major developmental milestones later (walking, riding a bike, etc...)
• Often don’t develop strong handedness
Three regions of the brain activated in the brains of children as they read words and pronounceable nonwords. Proficient readers showed activation of all three areas: the left occipitotemporal and parietotemporal regions, and the left and right inferior frontal cortex. Reading ability was positively correlated the level of activation of the occipitotemporal region; activation of this area was lower in dyslexic children.

V5/Occipitotemporal Complex