Cognitive Neuroscience

Spring 2025

Type of Evaluation	Weightage (in %)	
Quiz 1	10 %	MCQs + short answers
Quiz 2	10 %	MCQs + short answers
Midterm	25 %	MCQs + short answers
In-class random quizzes	25 %	5 surprise class quizzes over the semester
End-term - Oral presentation	30 %	Viva/Oral – 15 minutes (student groups)
Experiment Participation	5% extra	Participate in experiments that are conducted in the Cognitive Science Lab

Grade	%
A	>= 88
A-	81-87.5
B	74-80.5
B-	66 -73.5
C	59-65.5
C-	52-58.5
D	40 – 51.5
F	Below 40

TAs

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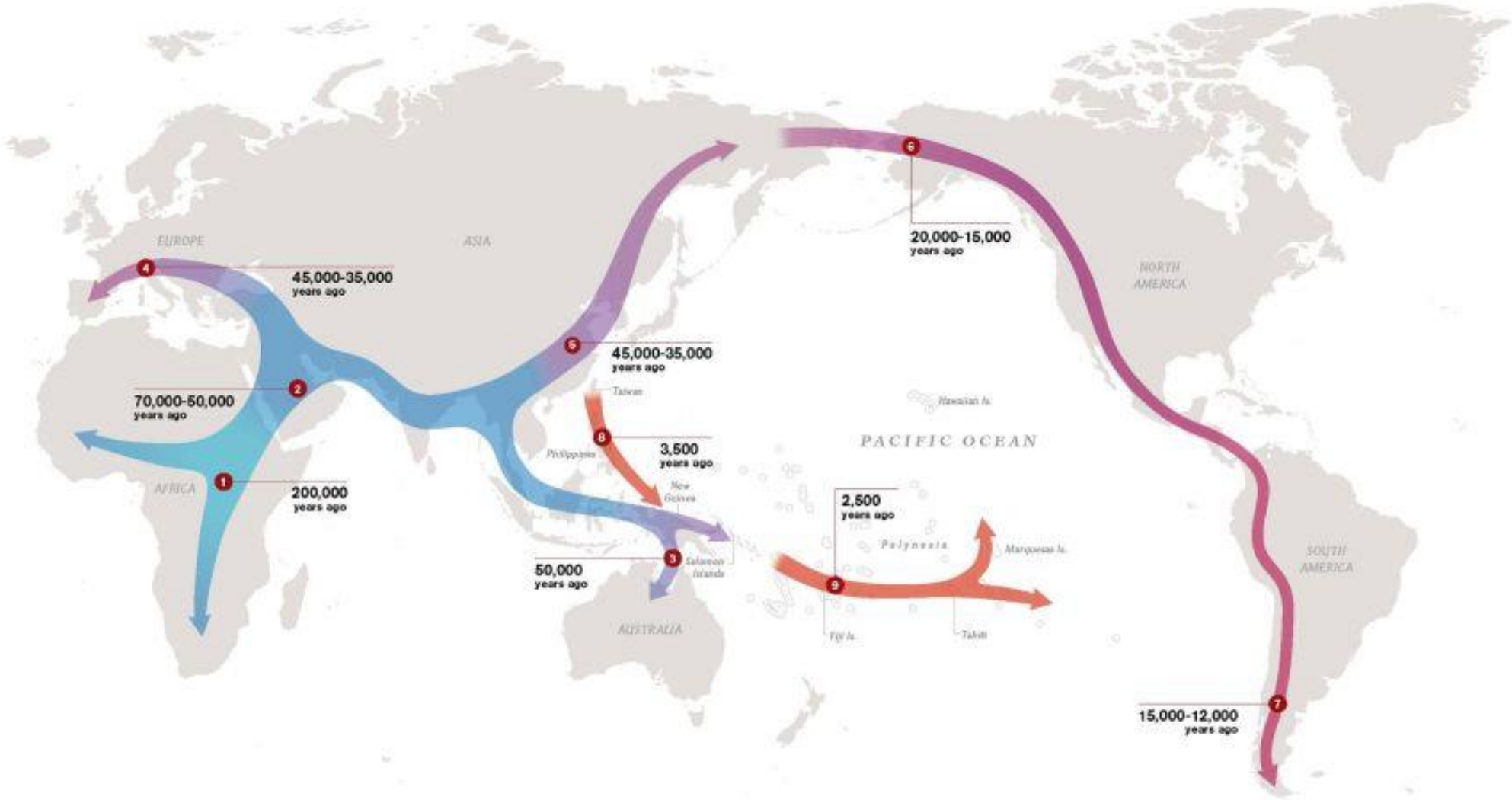


GLOBAL JOURNEY

Once modern humans began their migration out of Africa some 60,000 years ago, they kept going until they had spread to all corners of the Earth. How far and fast they went depended on climate, the pressures of population, and the invention of boats and other technologies. Less tangible qualities also sped their footsteps: imagination, adaptability, and an innate curiosity about what lay over the next hill.

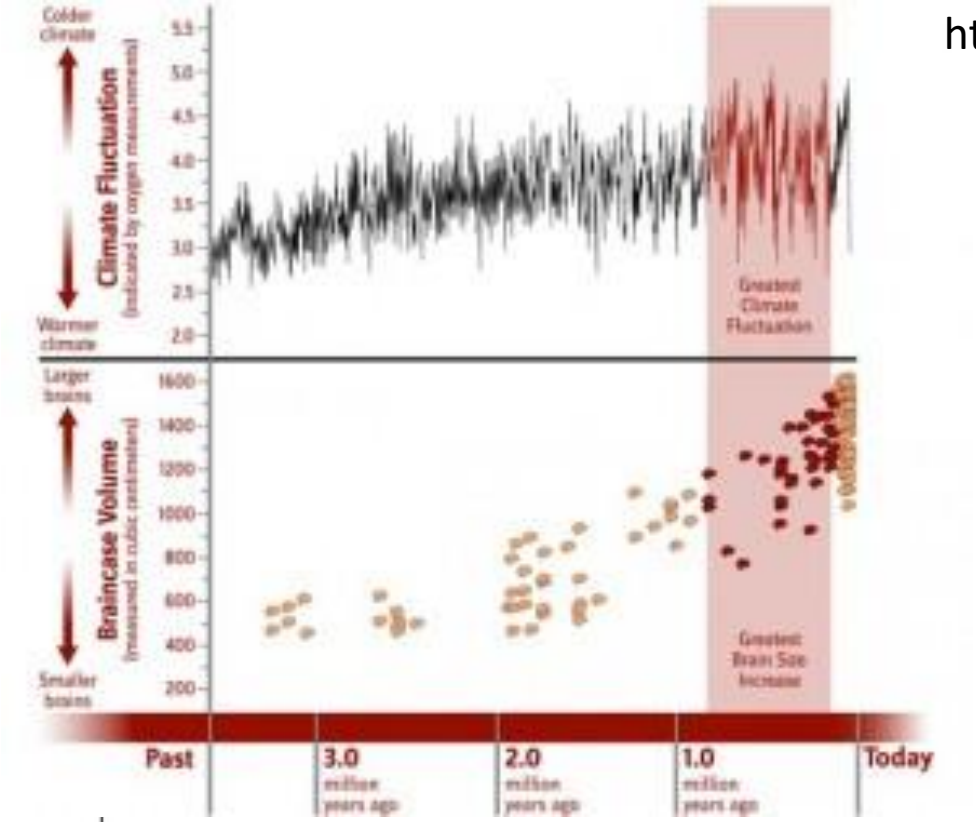
Generalized route with migration dates

200,000 50,000 20,000 2,500 years ago

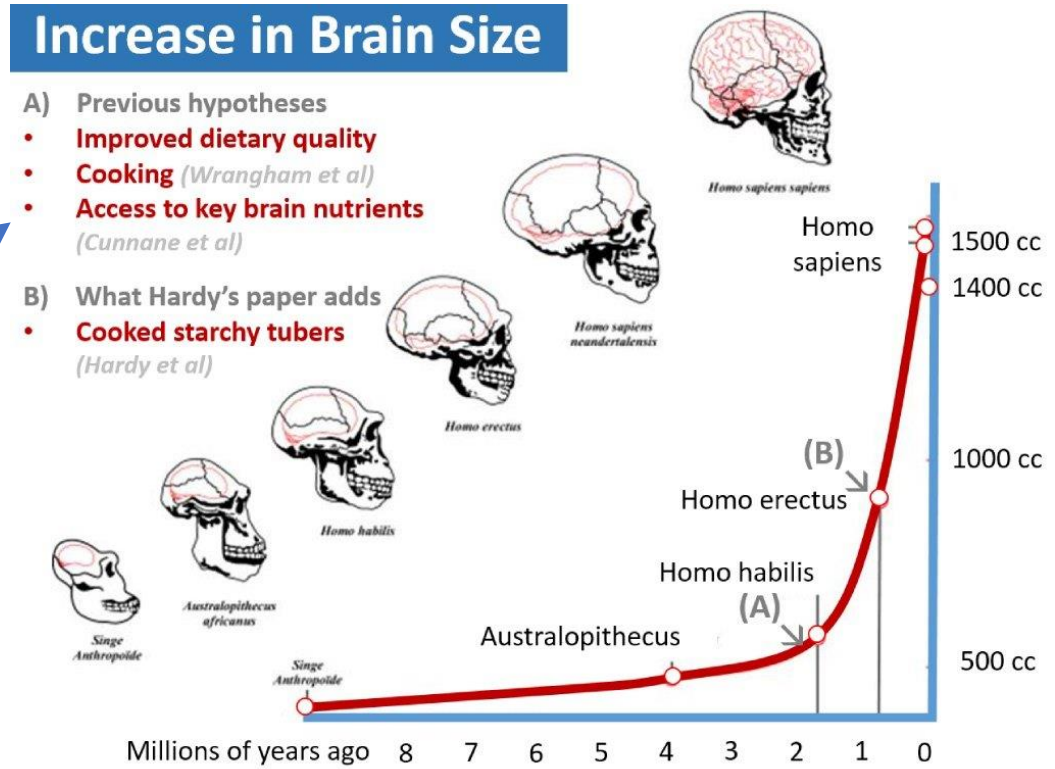
MAP: INTERNATIONAL MAPPING
SOURCES: CHRIS STRINGER, NATURAL HISTORY MUSEUM, LONDON;
SPENCER WELLS, NG STAFF

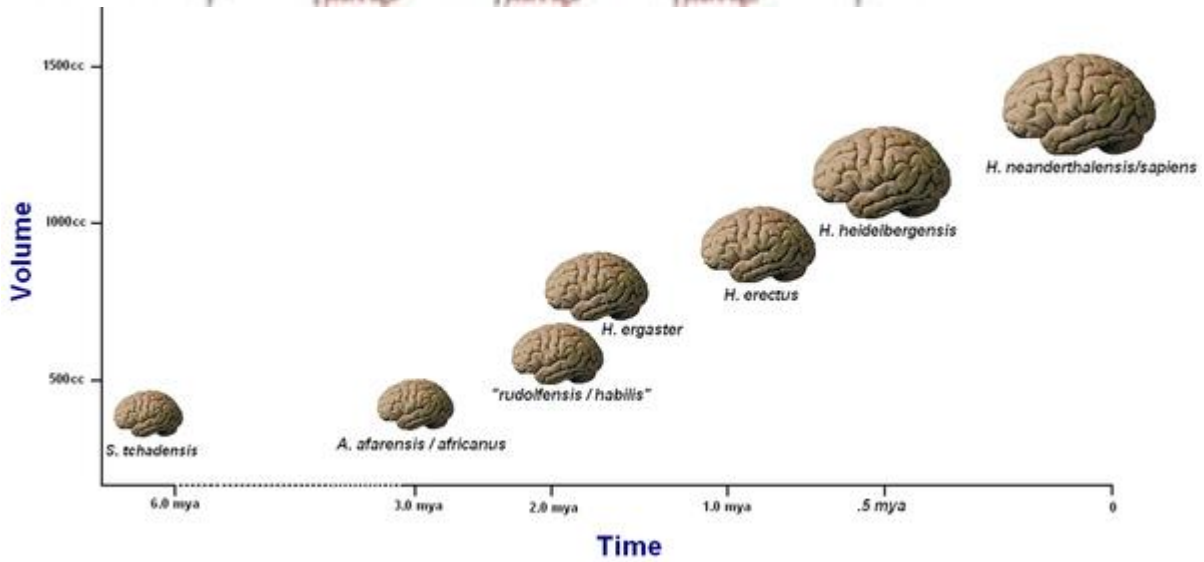
https://www.jstor.org/stable/full/10.1086/682587

[Kier Watson blog]

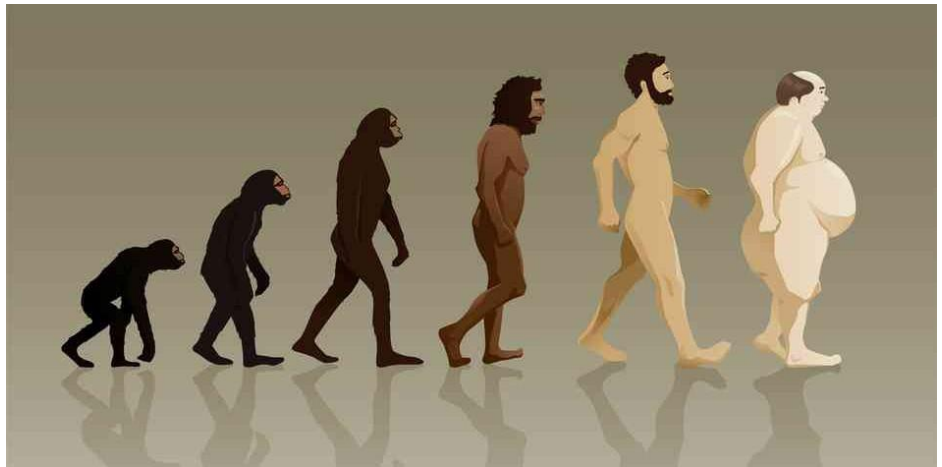


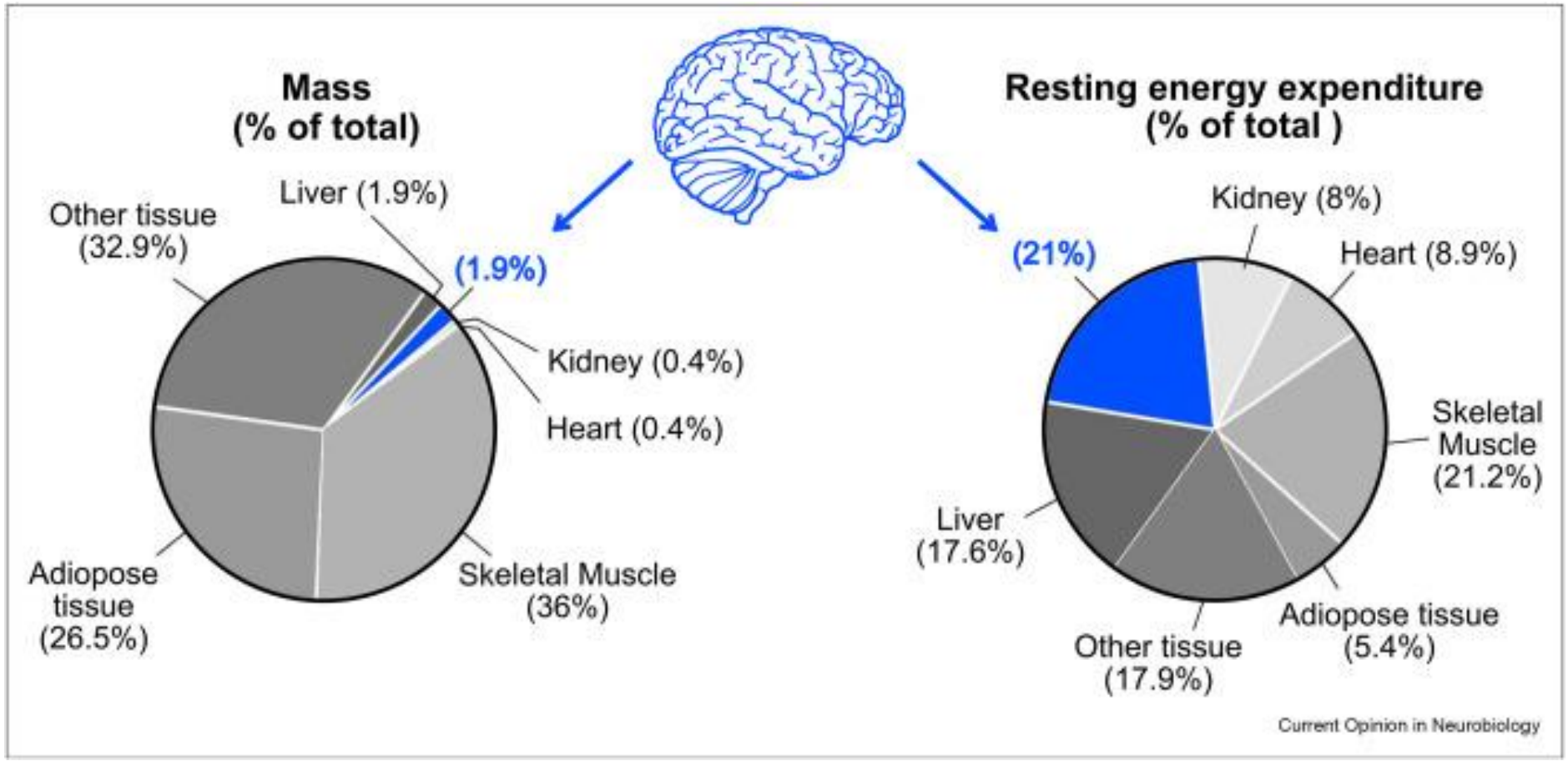
Why does this matter?





https://blogs.ntu.edu.sg/hg3040-2014-3/biological-evolution/increase-in-brain-size/





https://www.sciencedirect.com/science/article/pii/S0959438822001623

The brain accounts for about 20% of the body's energy consumption, despite only representing ~2% of its weight.

That's around 0.3 kilowatt hours (kWh) per day for an average adult, more than 100 times what the typical smartphone requires daily

How much energy do we expend thinking and using our brain?

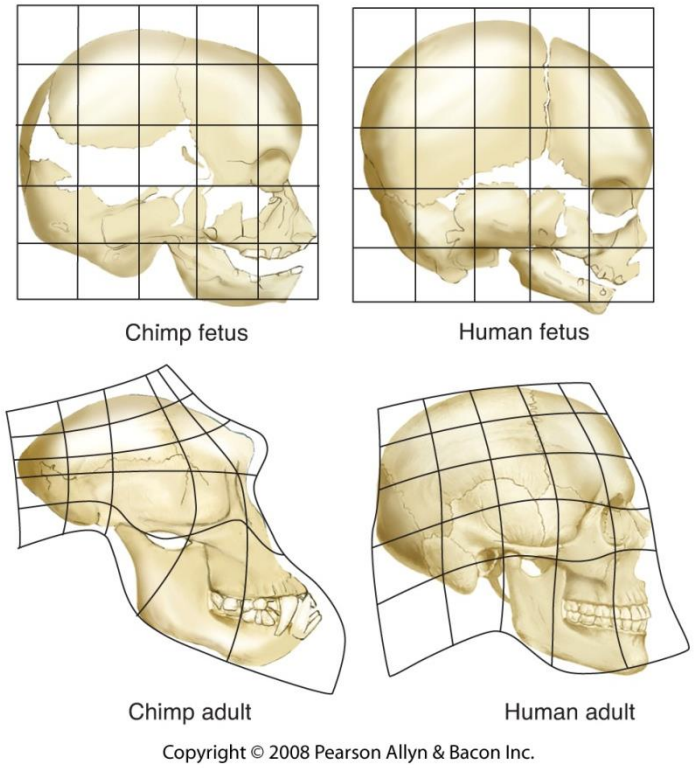
- What advantage does a larger brain, more neurons, offer?

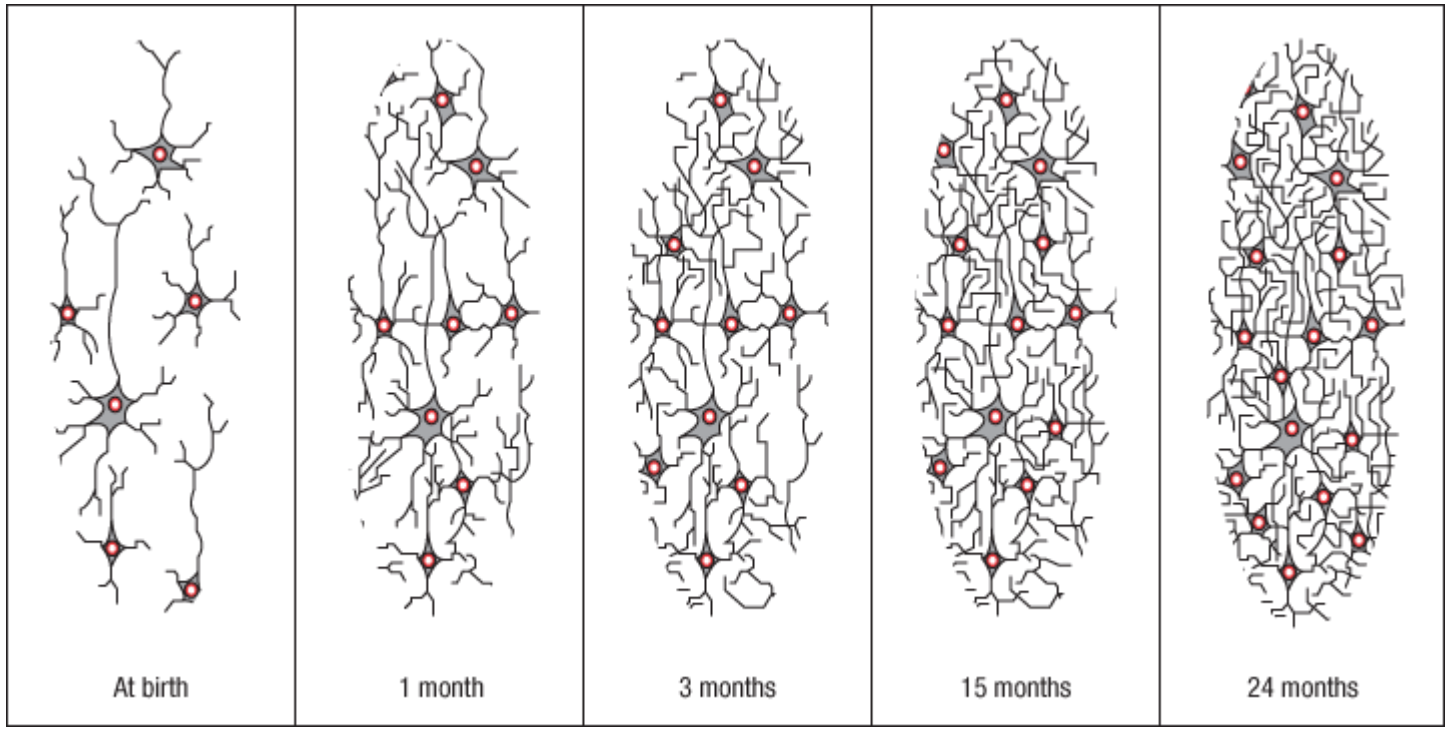
Evolution of human species

Large brains provide more memory capacity and the abilities to recognize patterns of events in the past and to plan for the future. Because an upright posture limits the size of a woman's birth canal and therefore the size of the head that can pass through it, much of the brain's growth must take place after birth, which means children require an extended period of parental care.

Our brains are 3 times larger than those of chimpanzees

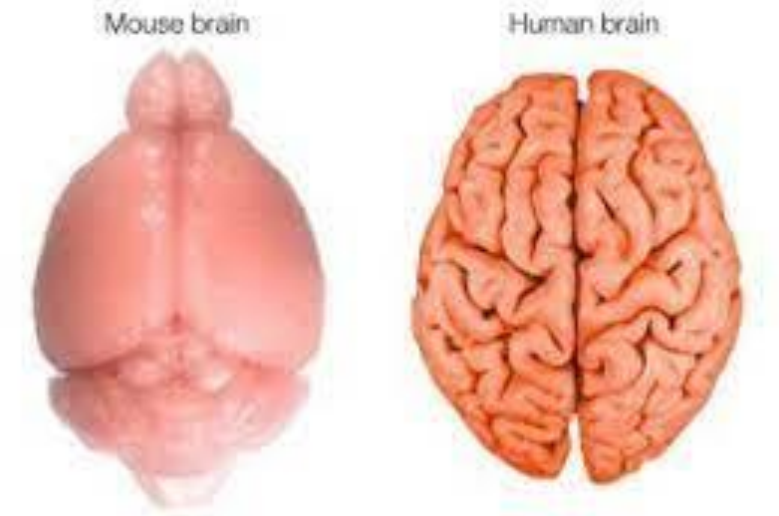
The grid lines indicate pattern of growth, showing much less change in the human skull from birth to adulthood

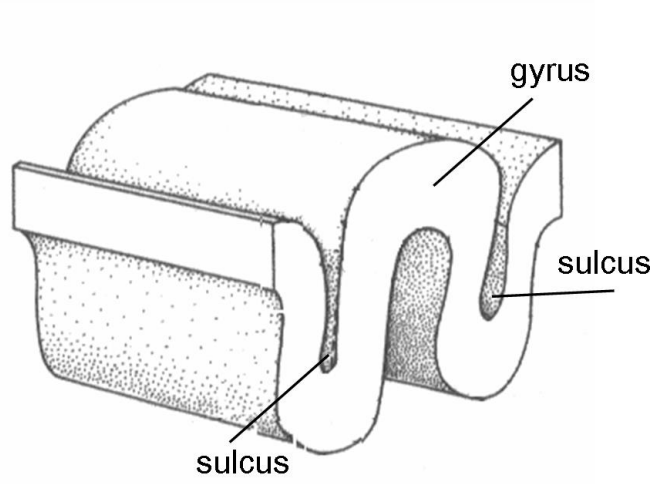




Synaptic pruning & synaptogenosis

- How does our brain accommodate high density of neurons in a limited space (skull)?
- ~86 billion neurons/interneurons
- ~86 billion non-neuron supporting cells (glial cells)

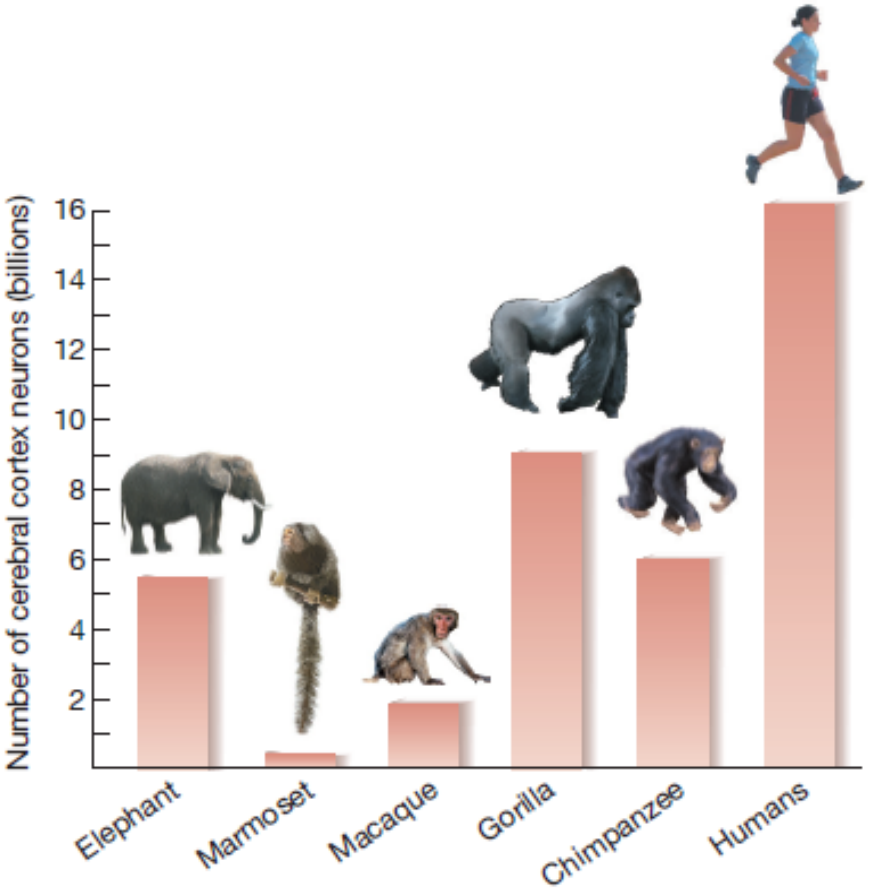




our brains contain many more neurons per gram

An increase in the number of **convolutions**—folds on the cerebral surface—has greatly increased the surface area of the *cerebral cortex*, the outermost layer of cerebral tissue

Species with more complex behaviors have brains with more neurons that are available for behavior, learning, remembering, reasoning, and making plans. Primate brains—especially large ones—contain many more neurons per gram than rodent brains and many more neurons in the cortex. Source: Herculano-Houzet, S., Marino, L. Brain Behav Evol 1998;51 230–238.

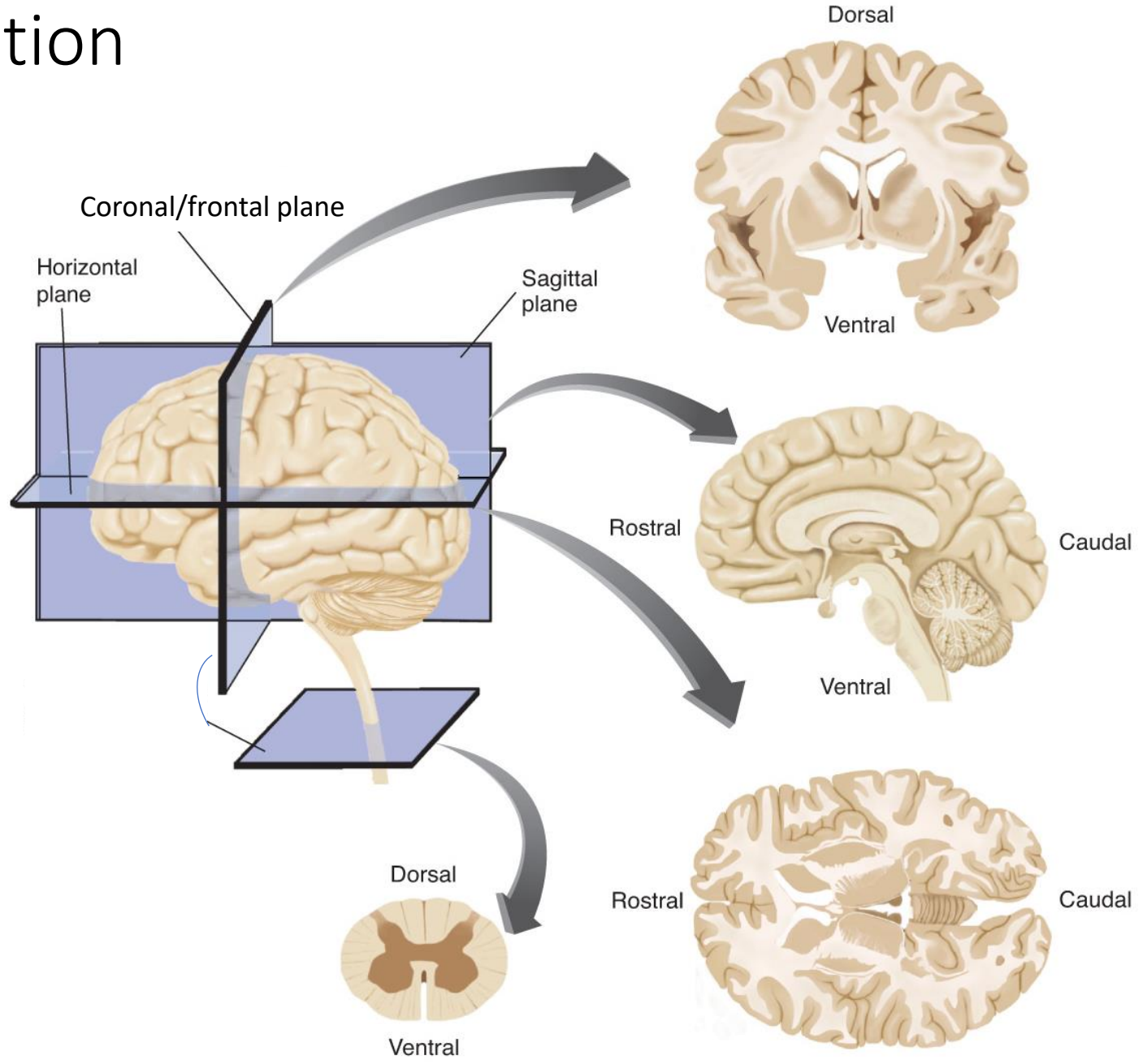


Humans have

- Larger surface area (especially cortical surface area)
- Higher neuronal density
- Higher ration of brain size to body size

Brain size → intelligence (non-linear relationship)
Humans → larger cerebral cortex (neocortex)
Elephants → larger cerebellum (trunk- sensory powerhouse)

Planes of section



Coronal section

Sagittal section

Horizontal section

Anatomical directions

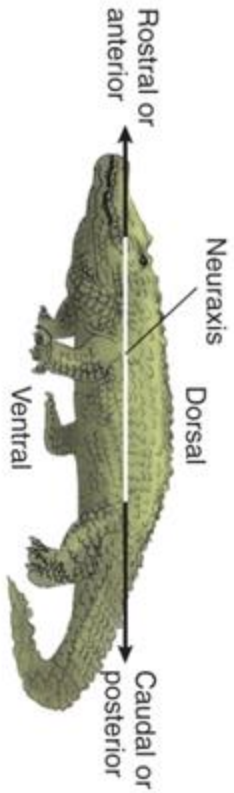
Neuraxis: An imaginary line drawn through the center of the length of the central nervous system, from the bottom of the spinal cord to the front of the forebrain

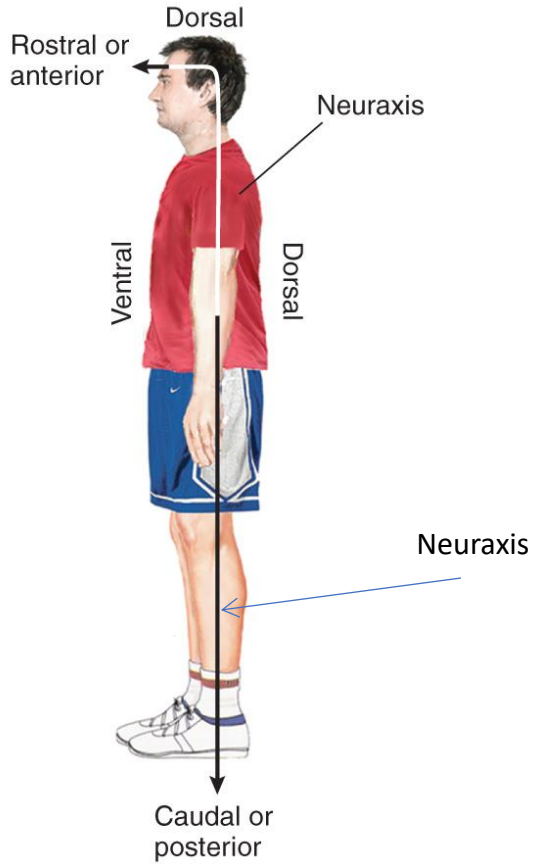
Dorsal: Towards the back

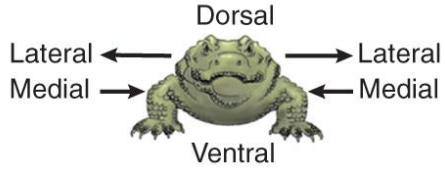
Ventral: Towards the belly

Ipsilateral: Located on the same side of the body.

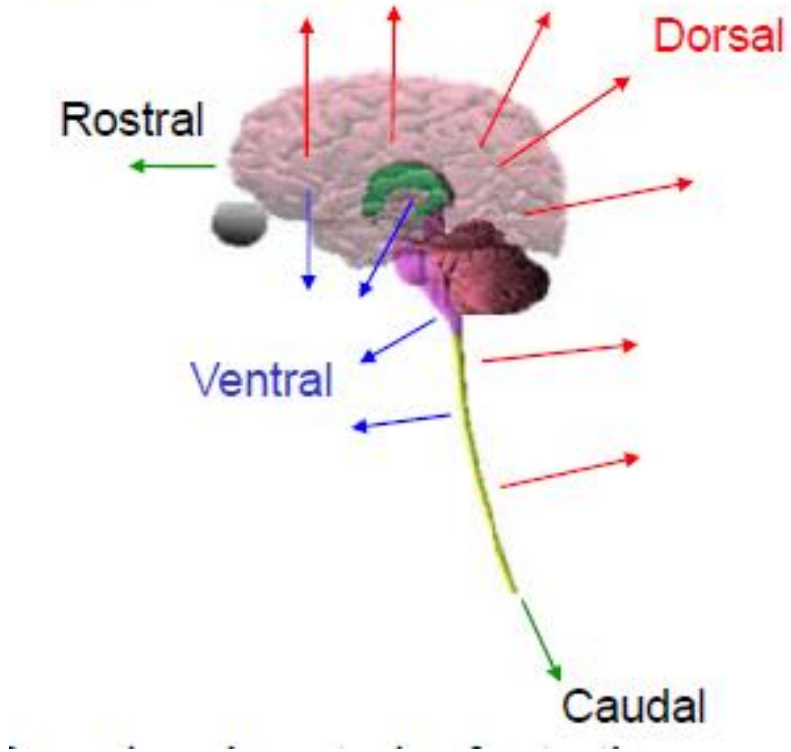
Contralateral: Located on opposite side of the body.

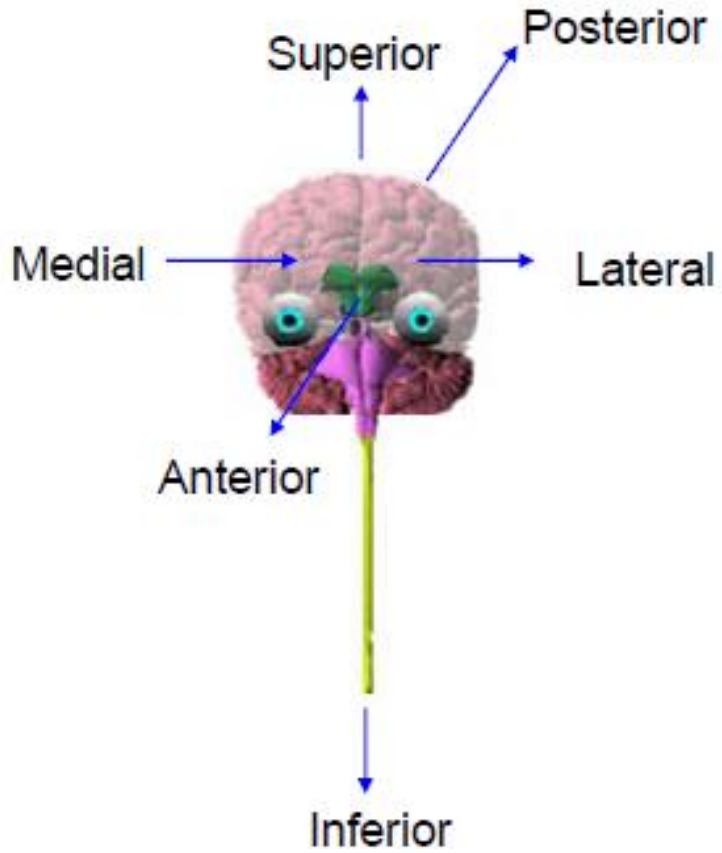


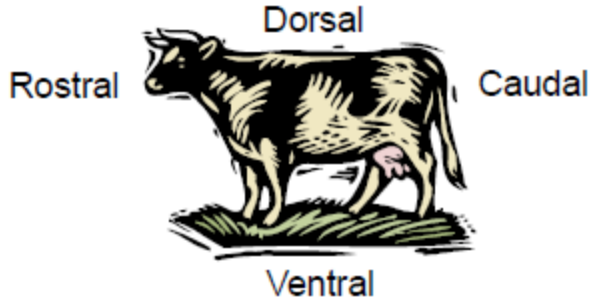


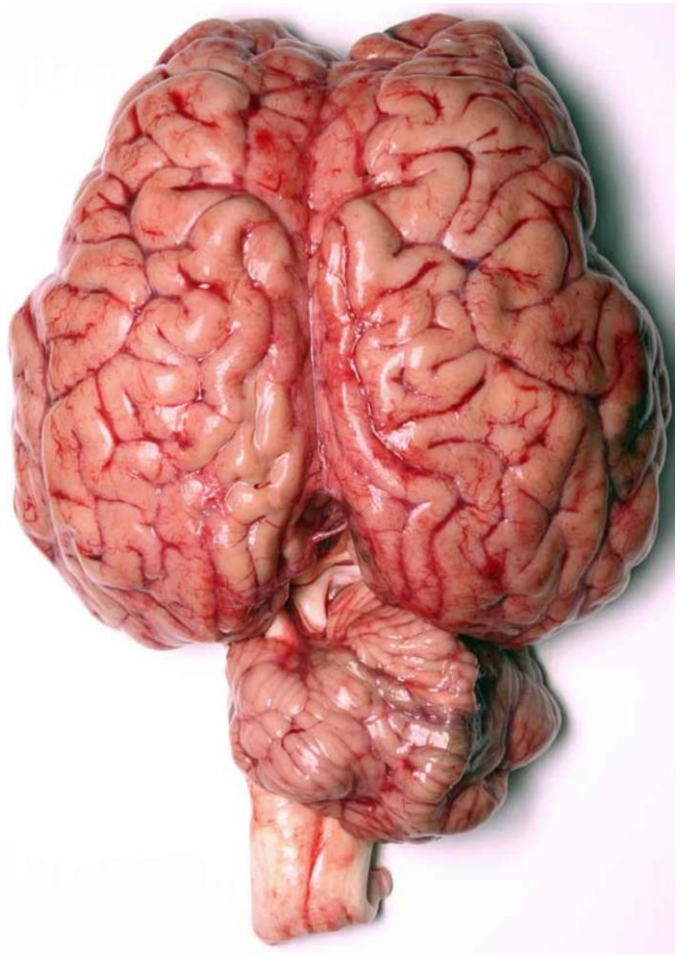


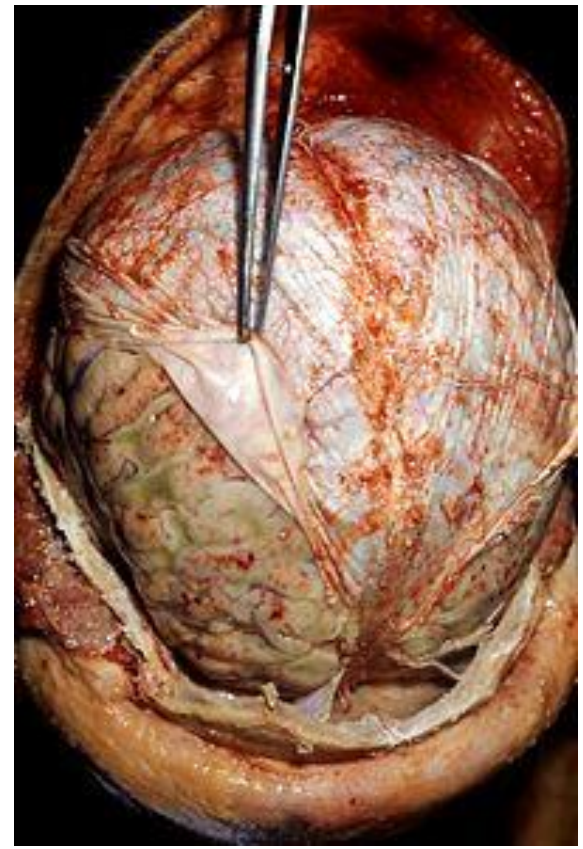
Orientation of CNS



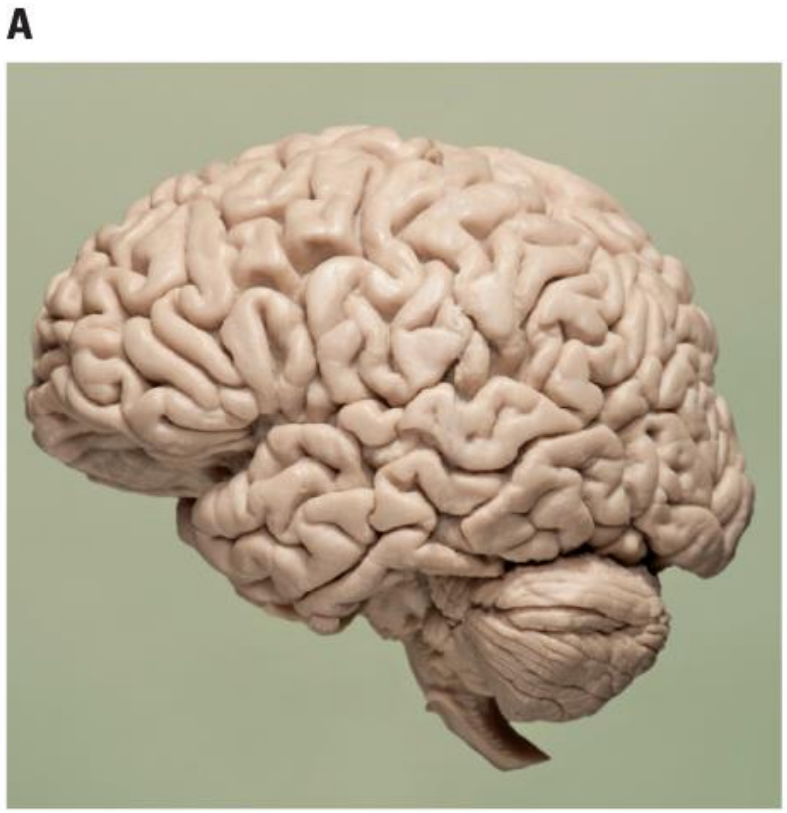


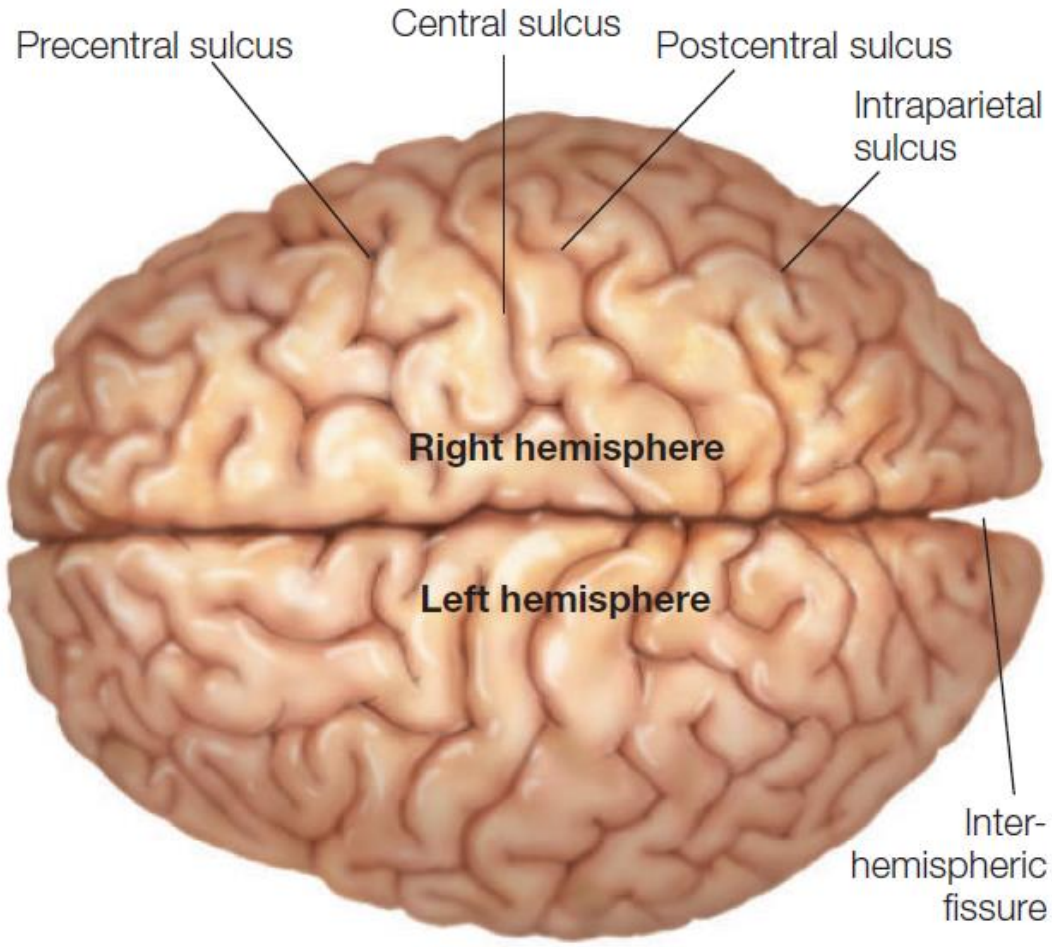


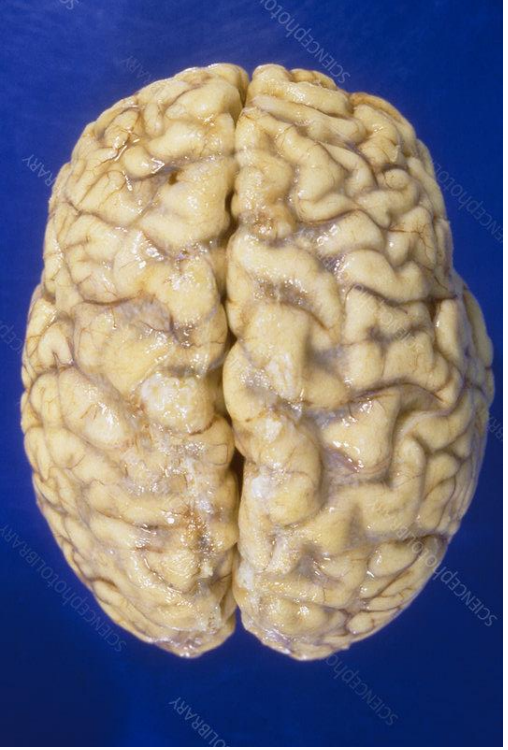


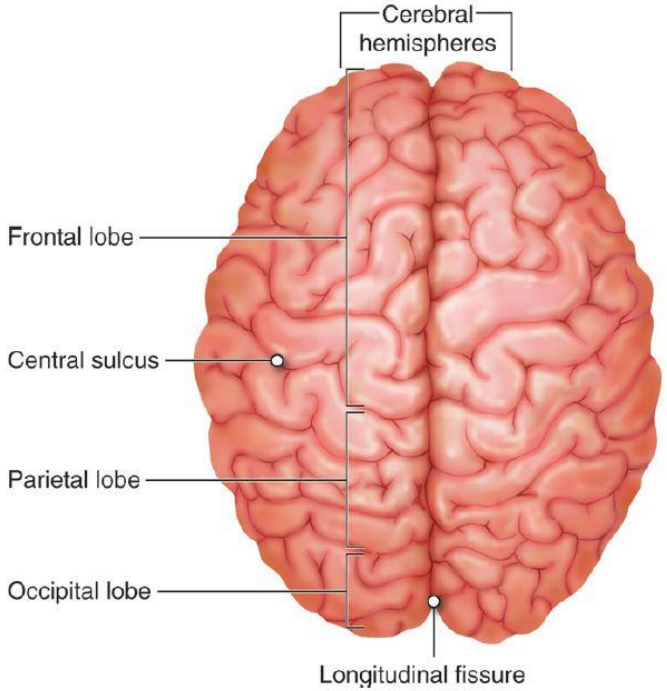


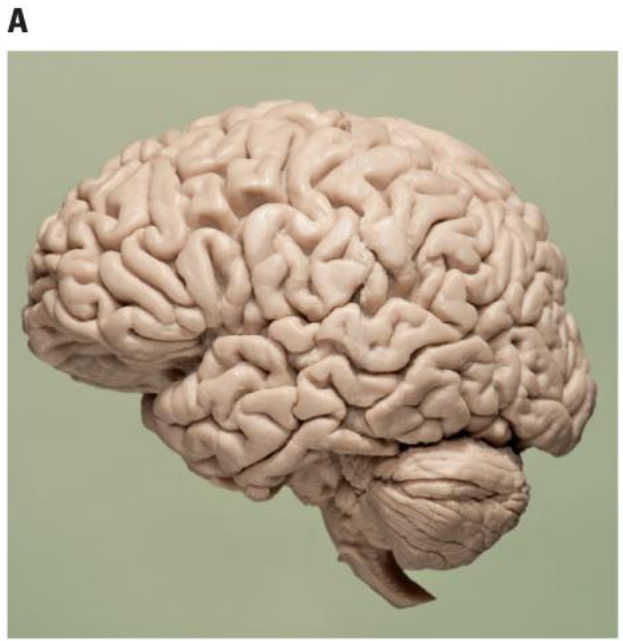
Reference points on the outer surface

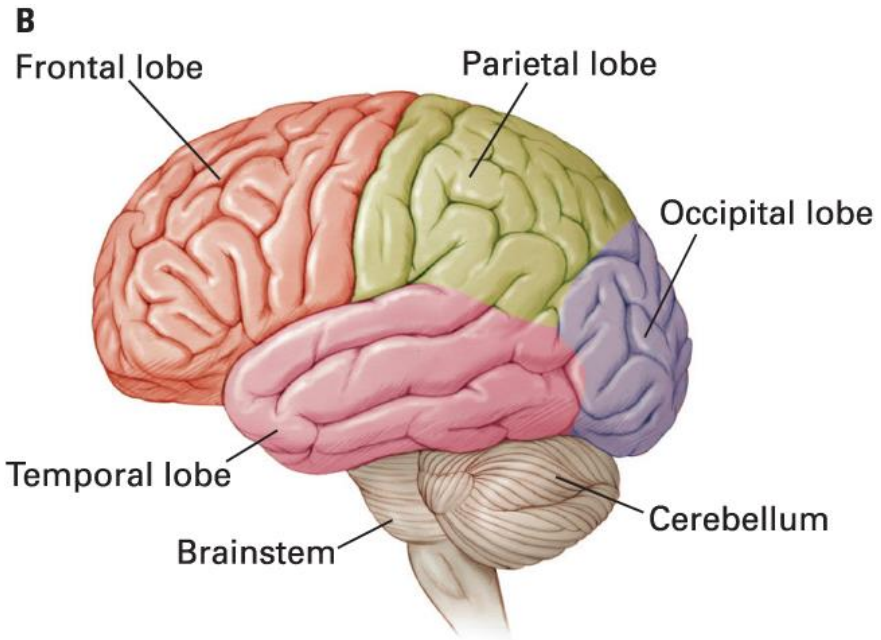






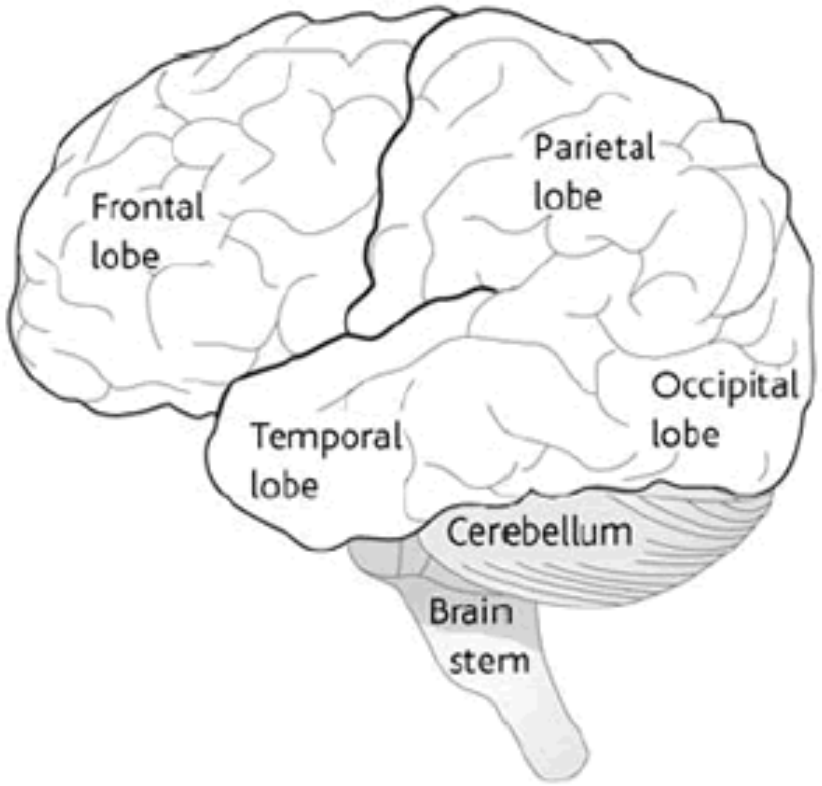


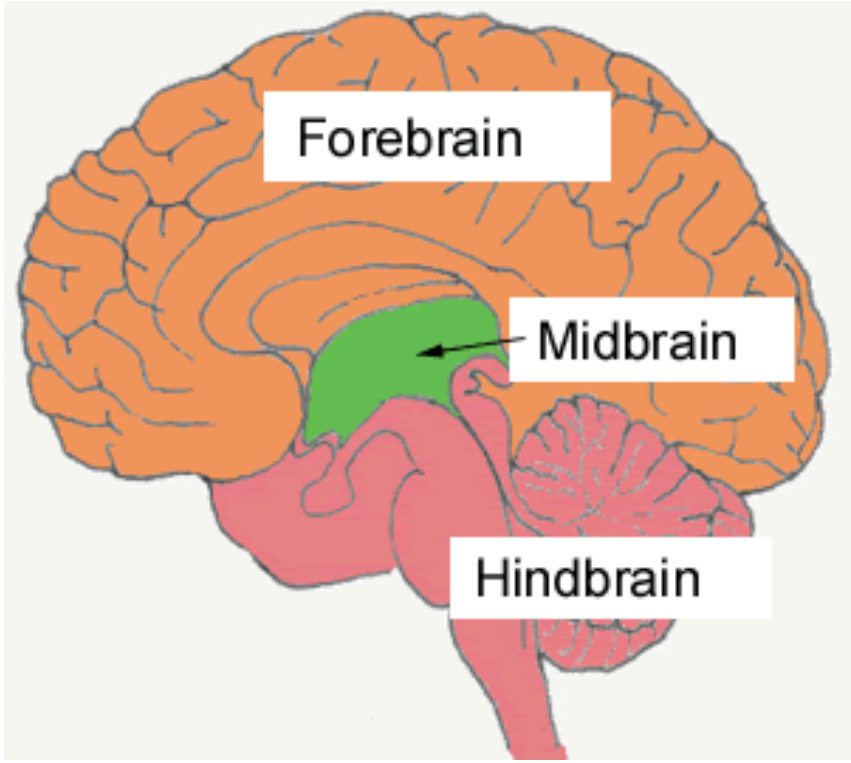




Gluck et al., *Learning and Memory*, 4e, © 2020 Worth Publishers

Major Divisions of the Brain





Forebrain – outer structures (frontal, temporal, parietal, occipital) – latest to evolve

Hindbrain – structures at the back (brain stem, cerebellum)

Midbrain – structures in the middle of the brain- cannot be seen for the outside – ancient in timescale (preserved across species)

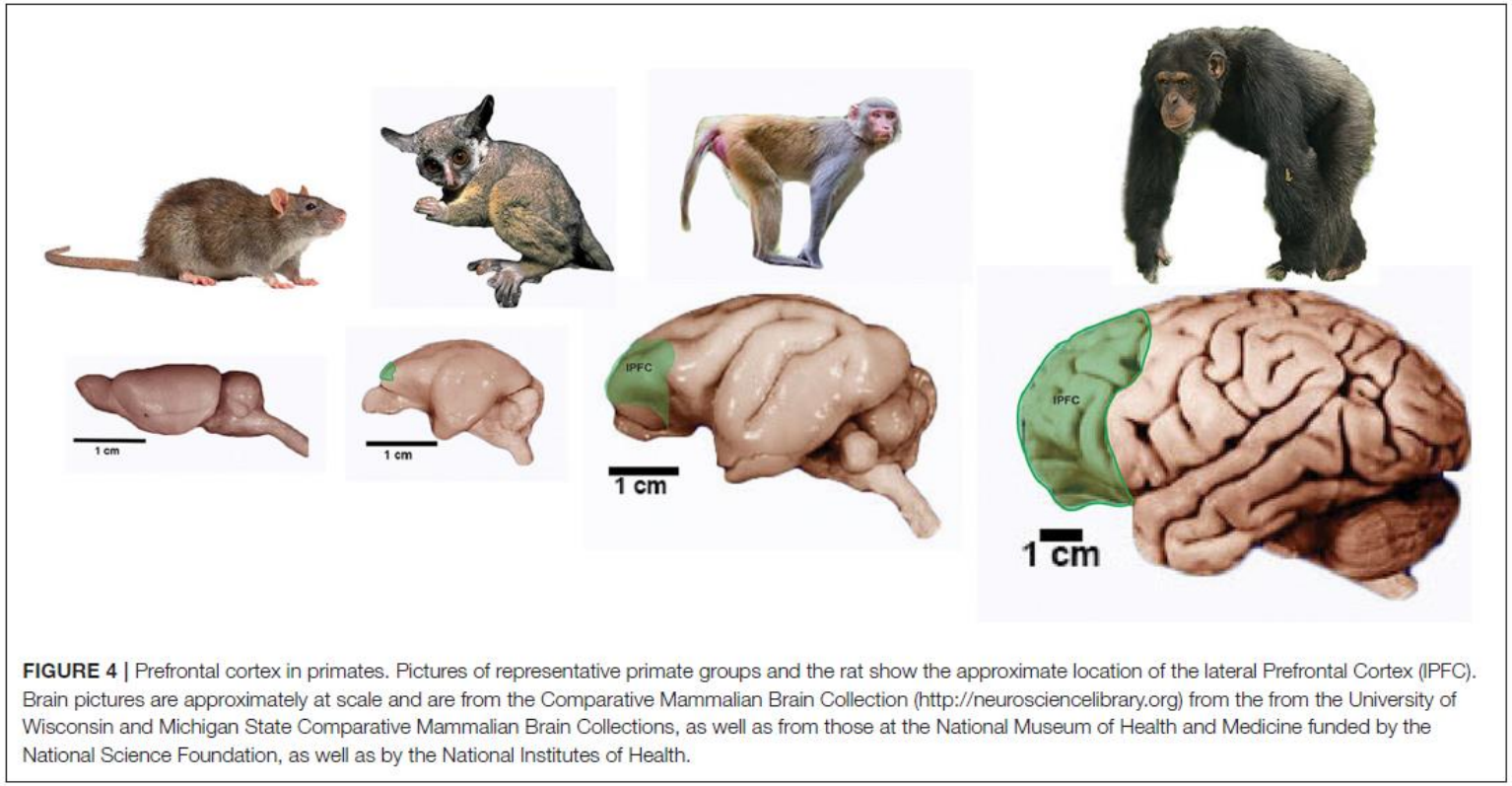
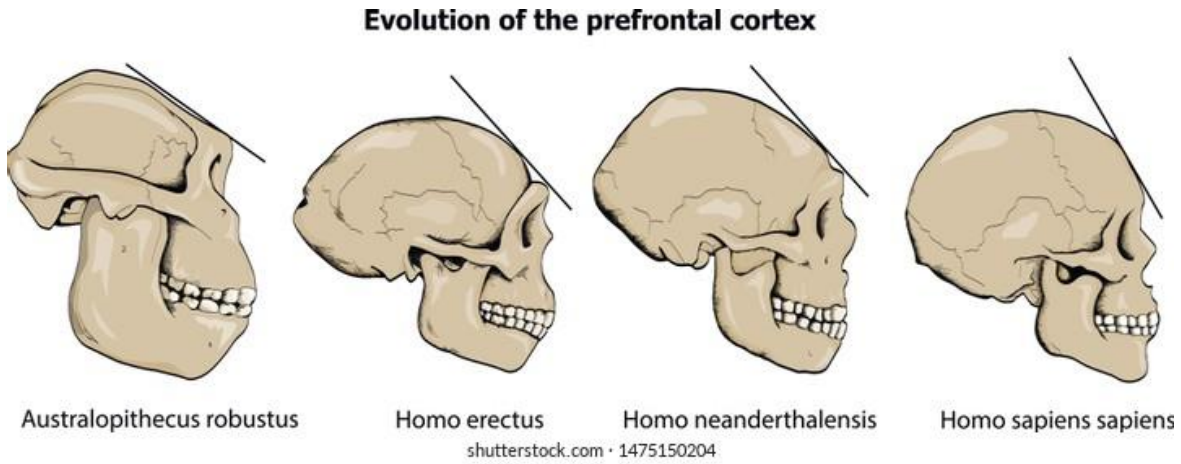
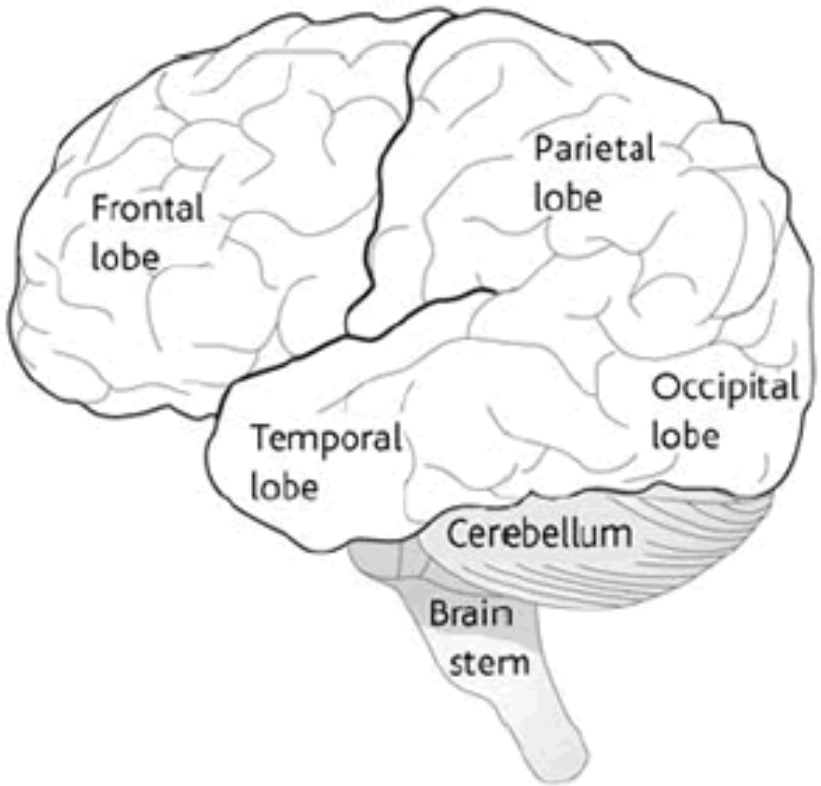


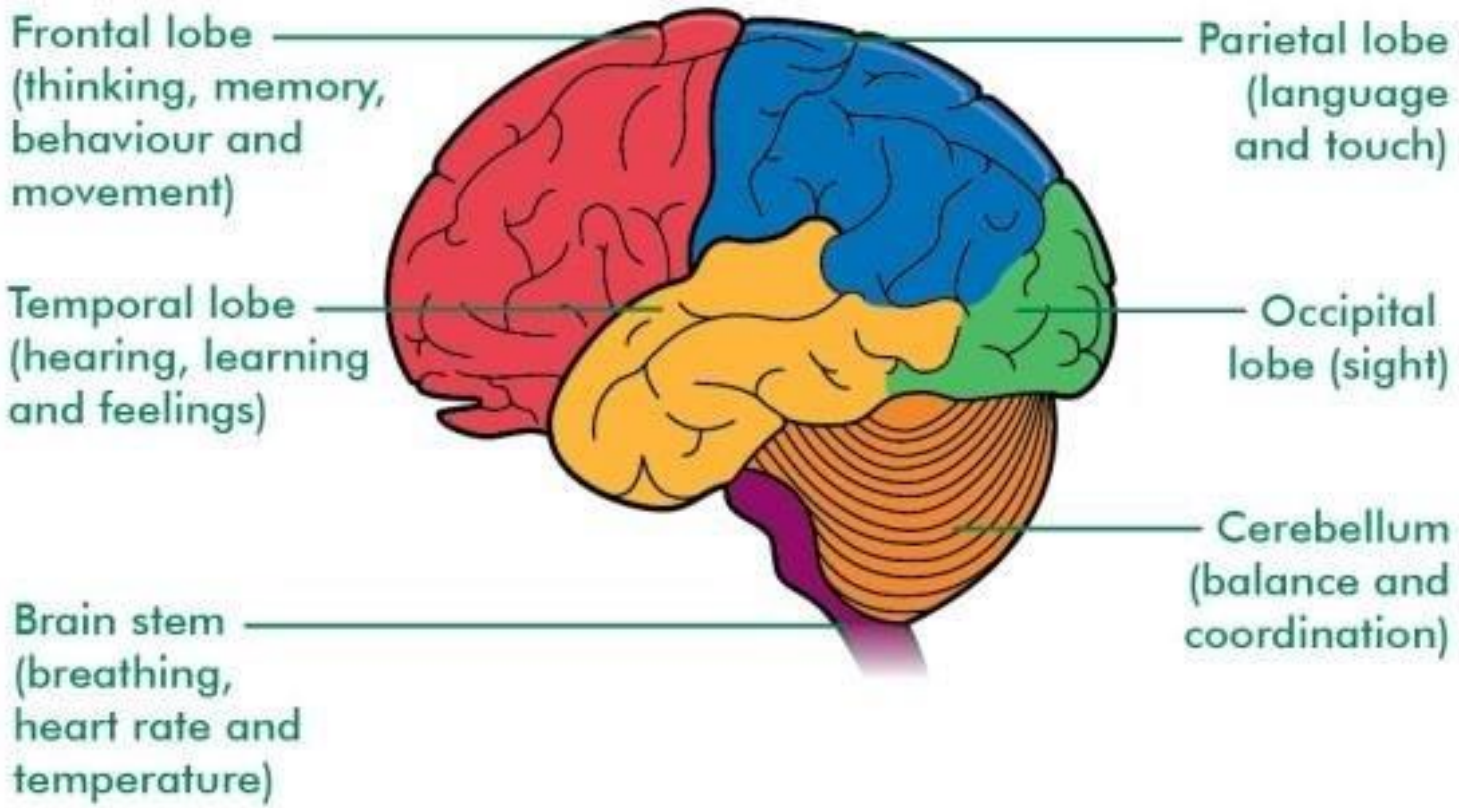
FIGURE 4 | Prefrontal cortex in primates. Pictures of representative primate groups and the rat show the approximate location of the lateral Prefrontal Cortex (lPFC). Brain pictures are approximately at scale and are from the Comparative Mammalian Brain Collection (http://neurosciencelibrary.org) from the from the University of Wisconsin and Michigan State Comparative Mammalian Brain Collections, as well as from those at the National Museum of Health and Medicine funded by the National Science Foundation, as well as by the National Institutes of Health.

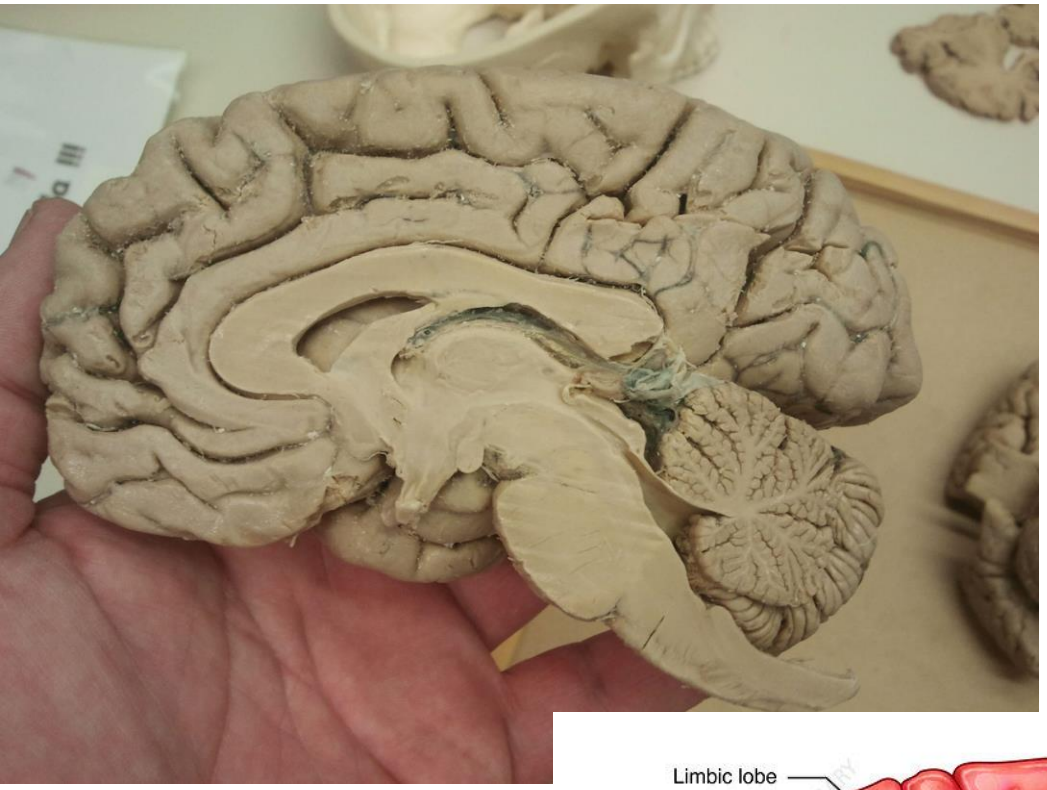


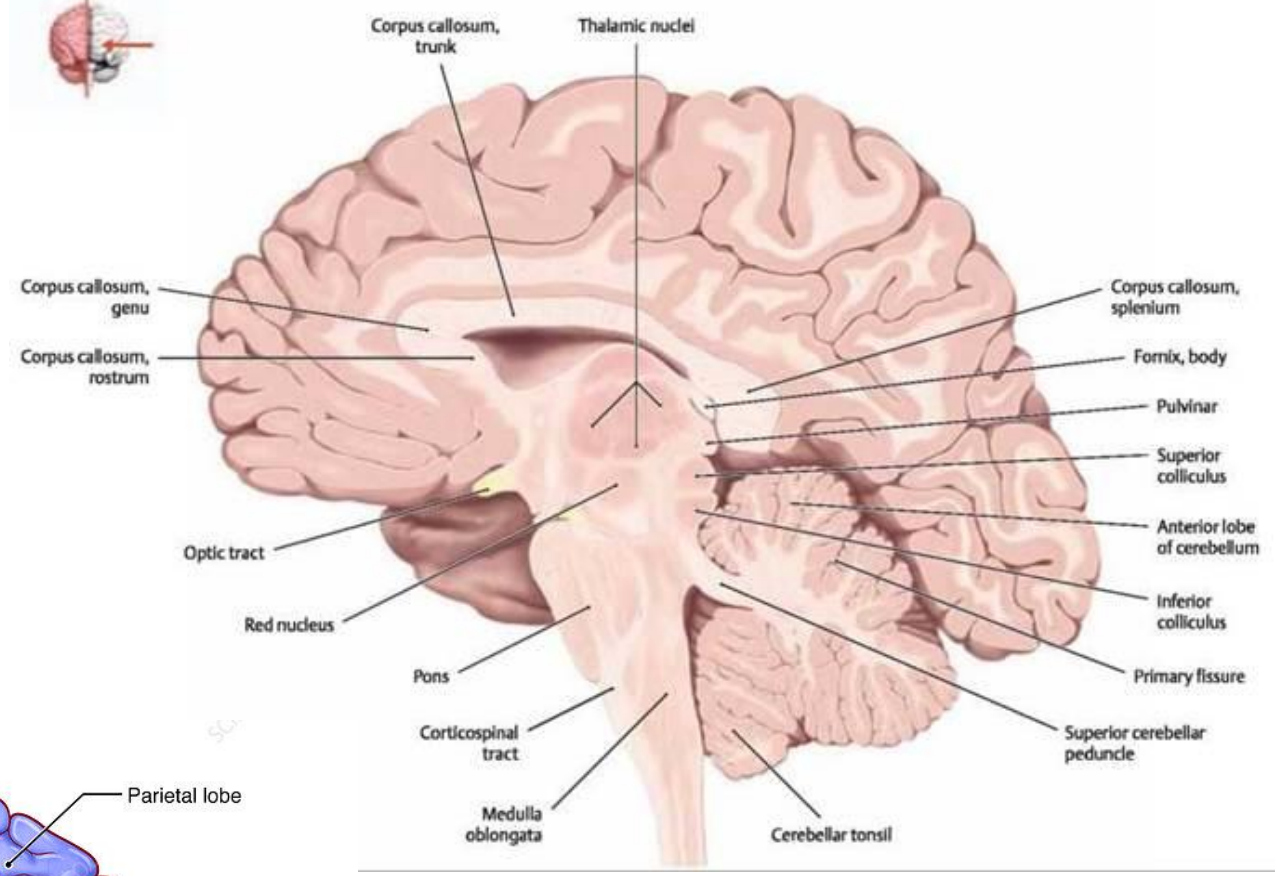
https://www.frontiersin.org/journals/cell-and-developmental-biology/articles/10.3389/fcell.2021.591017/full

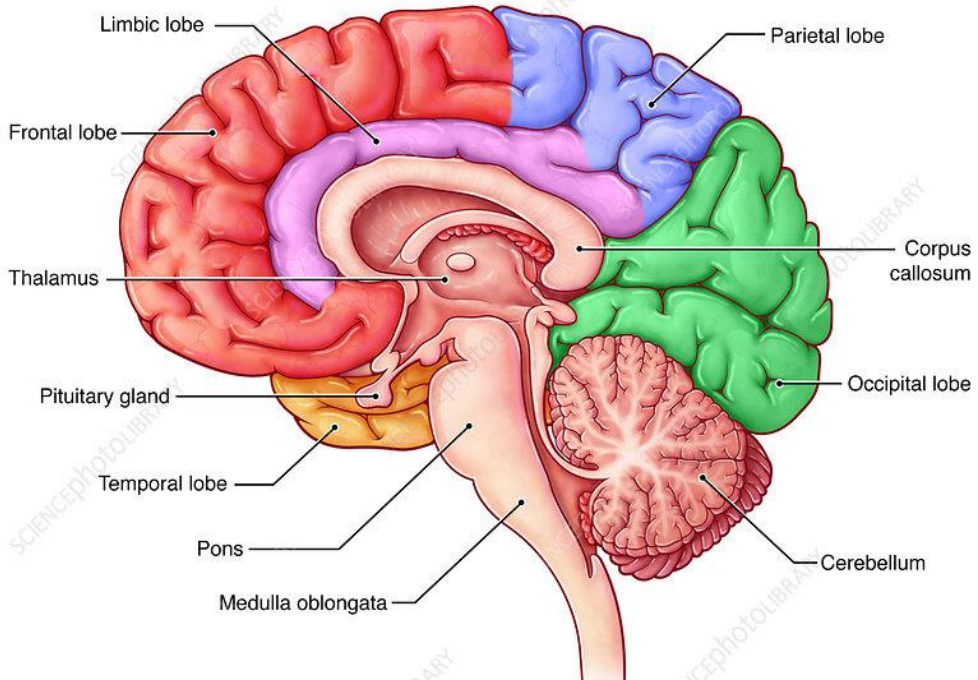
https://thebrain.mcgill.ca/flash/a/a_05/a_05_cr/a_05_cr_her/a_05_cr_her.html


Frontal lobe
Parietal lobe
Temporal lobe
Occipital lobe
Cerebellum
Brain stem

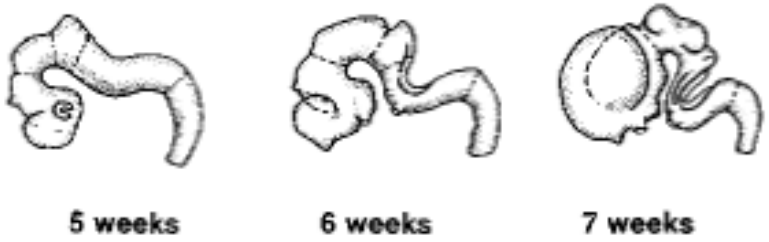

Frontal lobe (thinking, memory, behaviour and movement)
Parietal lobe (language and touch)
Temporal lobe (hearing, learning and feelings)
Occipital lobe (sight)
Cerebellum (balance and coordination)
Brain stem (breathing, heart rate and temperature)

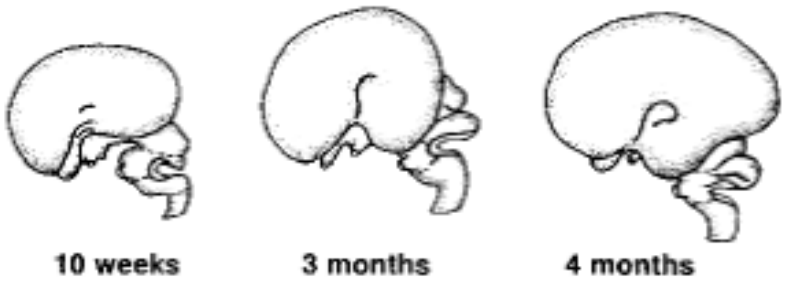


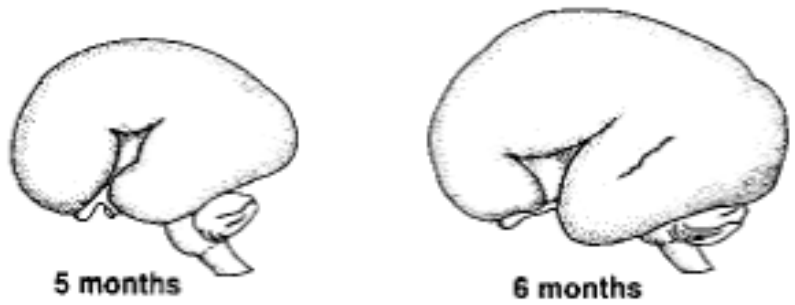

Corpus callosum, trunk
Thalamic nuclei
Corpus callosum, genu
Corpus callosum, rostrum
Corpus callosum, splenium
Fornix, body
Pulvinar
Superior colliculus
Anterior lobe of cerebellum
Inferior colliculus
Primary fissure
Superior cerebellar peduncle
Optic tract
Red nucleus
Pons
Corticospinal tract
Medulla oblongata
Cerebellar tonsil

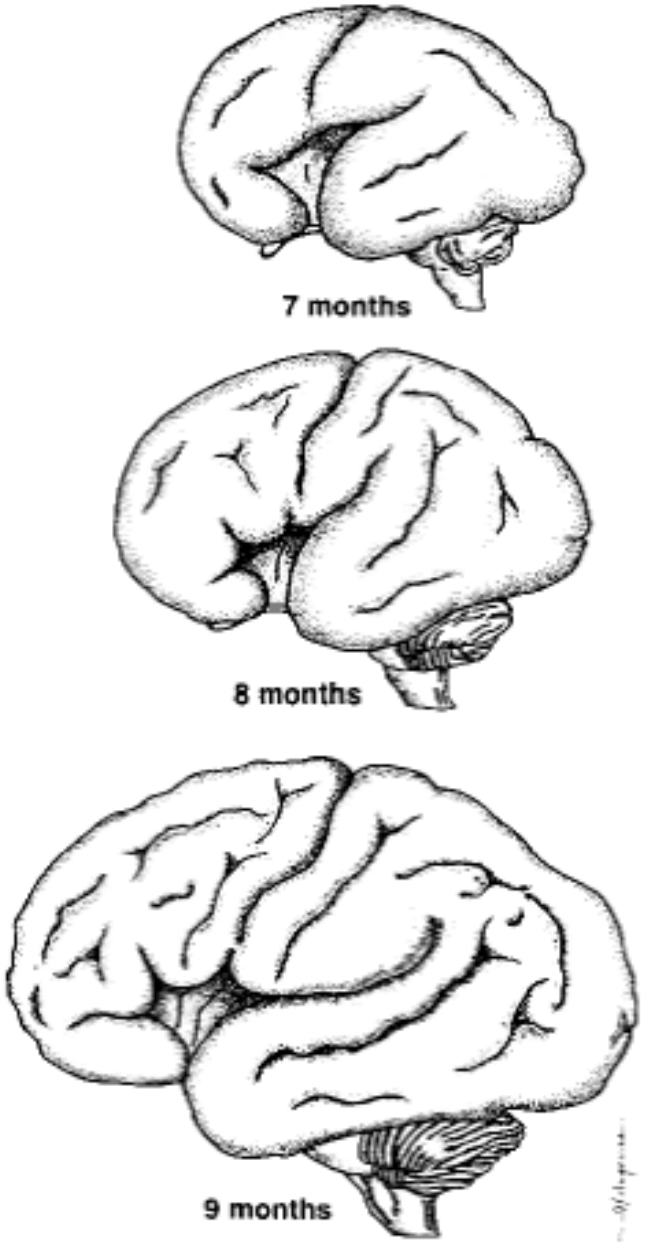

Limbic lobe
Parietal lobe
Frontal lobe
Thalamus
Corpus callosum
Occipital lobe
Pituitary gland
Temporal lobe
Pons
Cerebellum
Medulla oblongata

Development of the human nervous system begins around
the eighteenth day after conception









From conception to birth

Brain development continues after an animal is born. In fact, the human brain continues to develop for at least two decades. Every learning experience changes the brain.

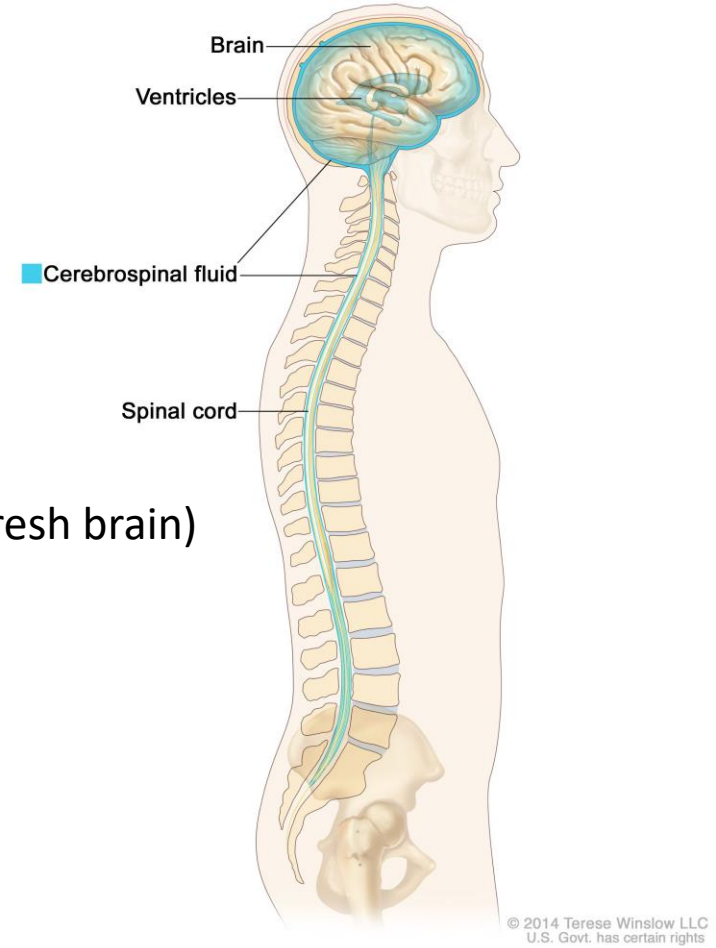
The last region to mature in the frontal cortex ~ 25 years

Legal age to drive or drink

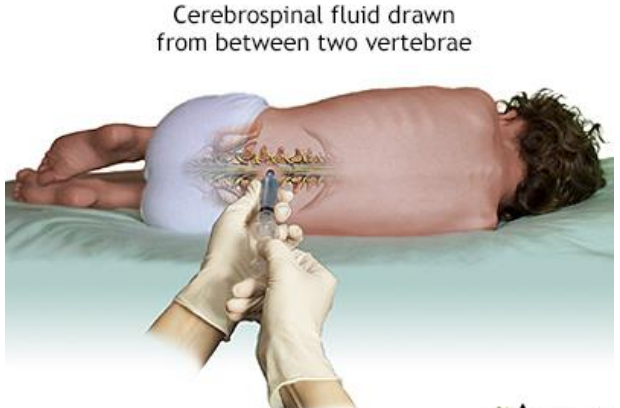
100 billion neurons

- How does your head and neck manage the weight of your brain?
- (1300-1400gms)

Cerebro-Spinal Fluid (CSF)

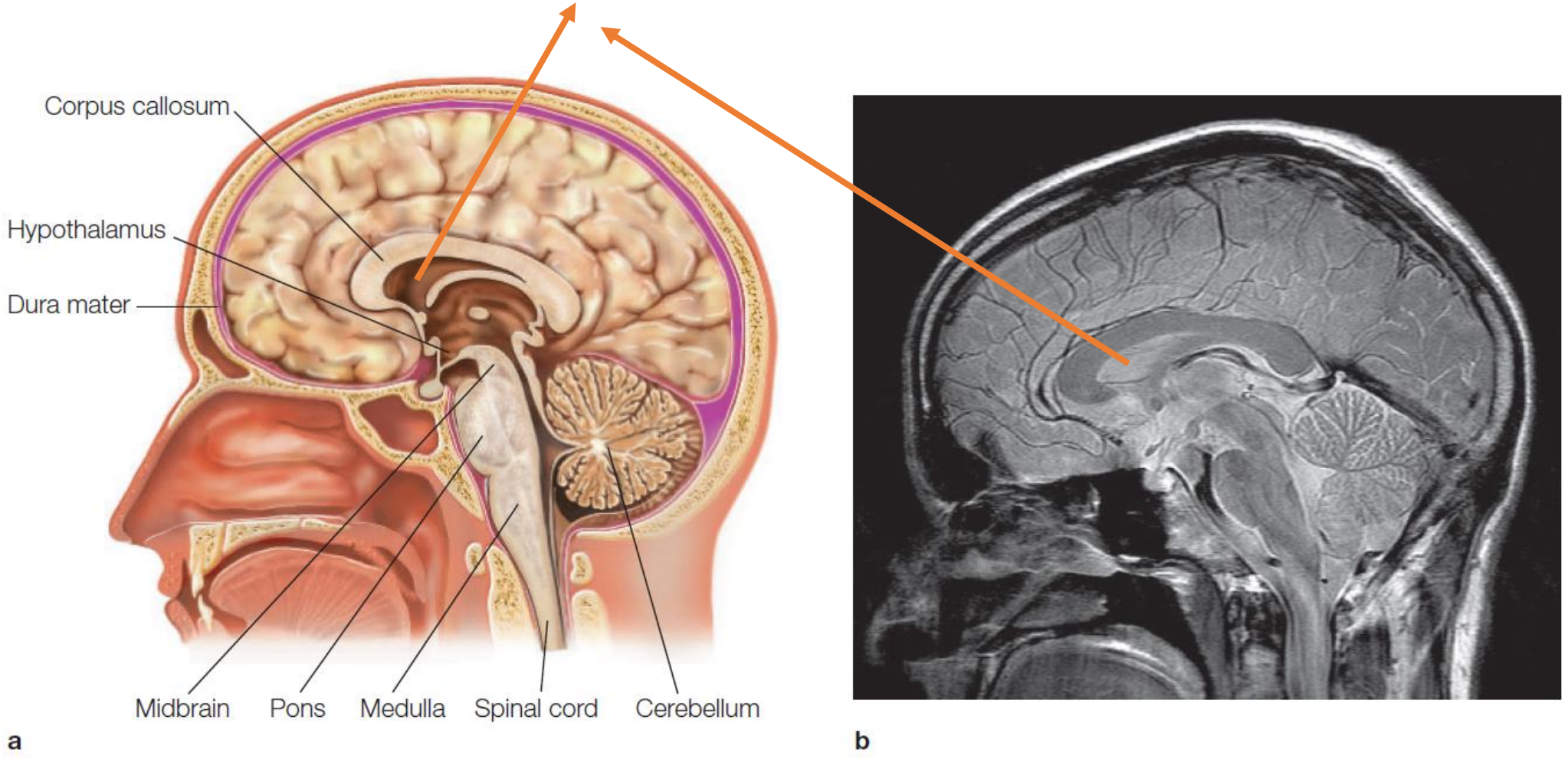


- ✓ A human brain cannot even support its own weight (extremely difficult to handle a fresh brain)
- ✓ An intact brain floats in CSF
- ✓ When floating, its weighs less, approx 80g!
- ✓ CSF also reduces the shock to the CNS due to sudden head movements and jolts.
- ✓ Total volume of CSF in brain – 125ml
- ✓ All of the CSF is replaced every six hours
- ✓ CSF is extracted to detect many infections in the brain



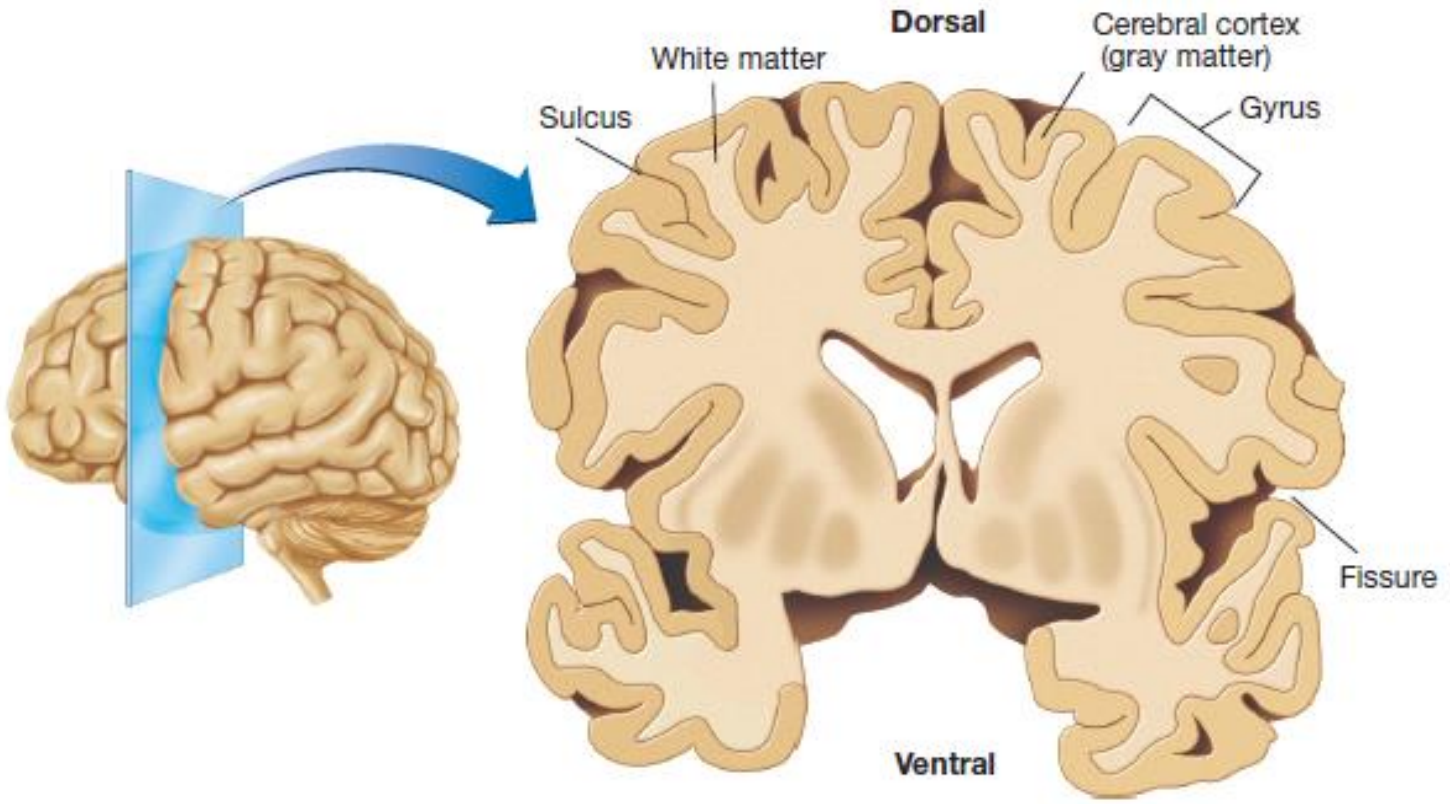
MRI Image

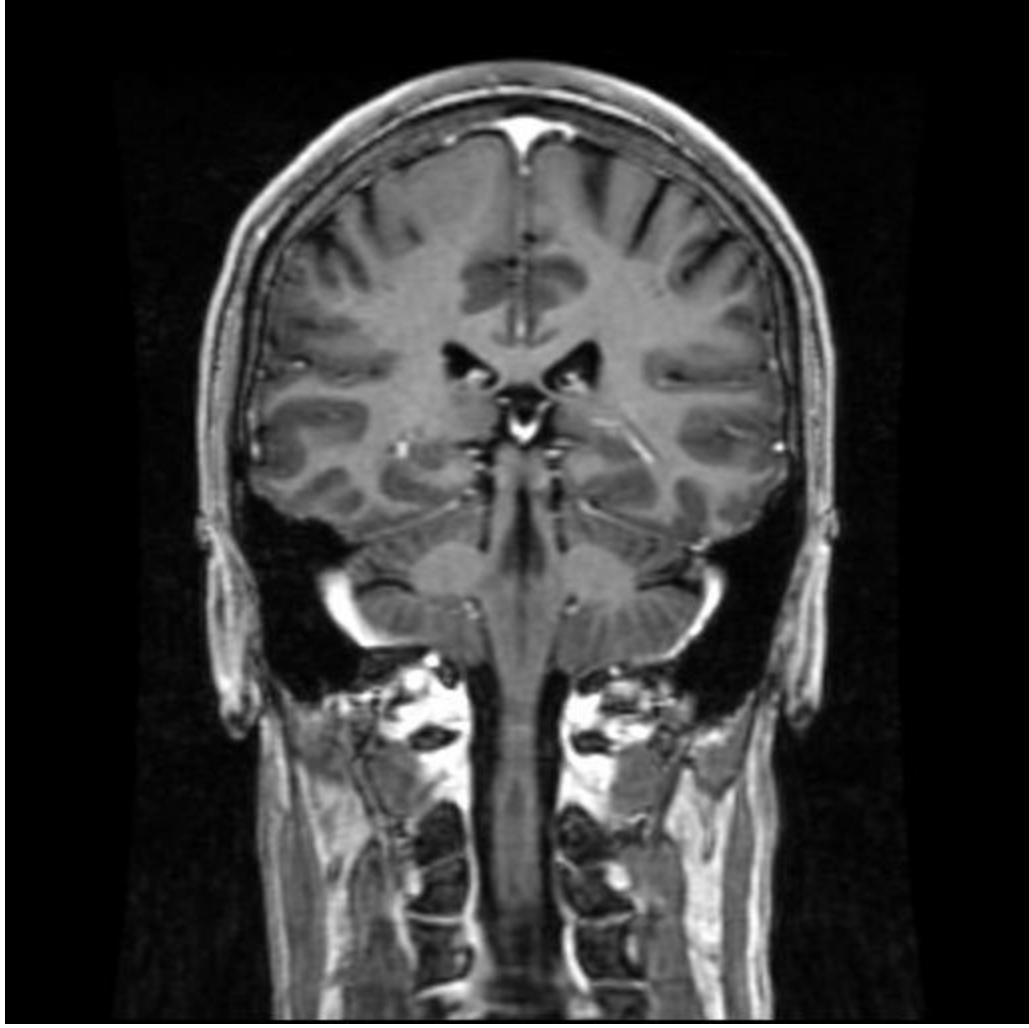
Ventricle (reference points)



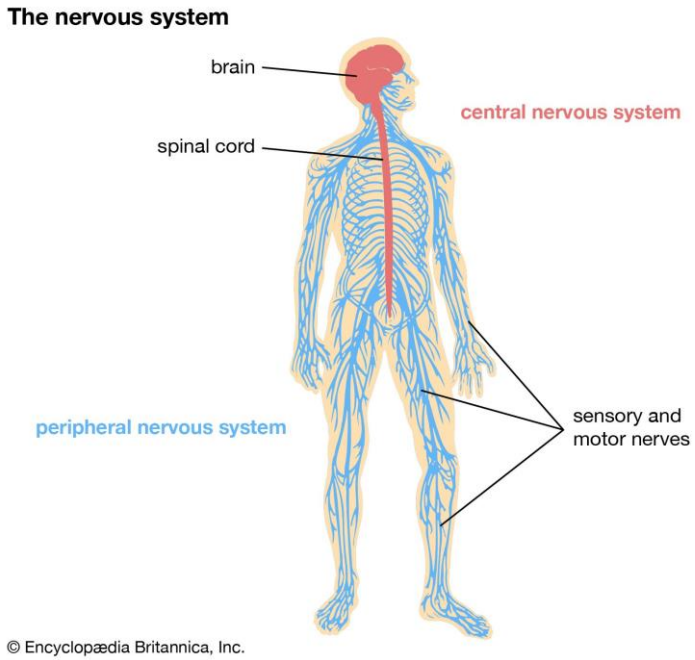
Gray matter **-** due to the grayish color of cell bodies (of neurons) that predominate the region

White matter: due to the whitish opaque color of the myelin (a protective covering on the neurons) that predominate this region

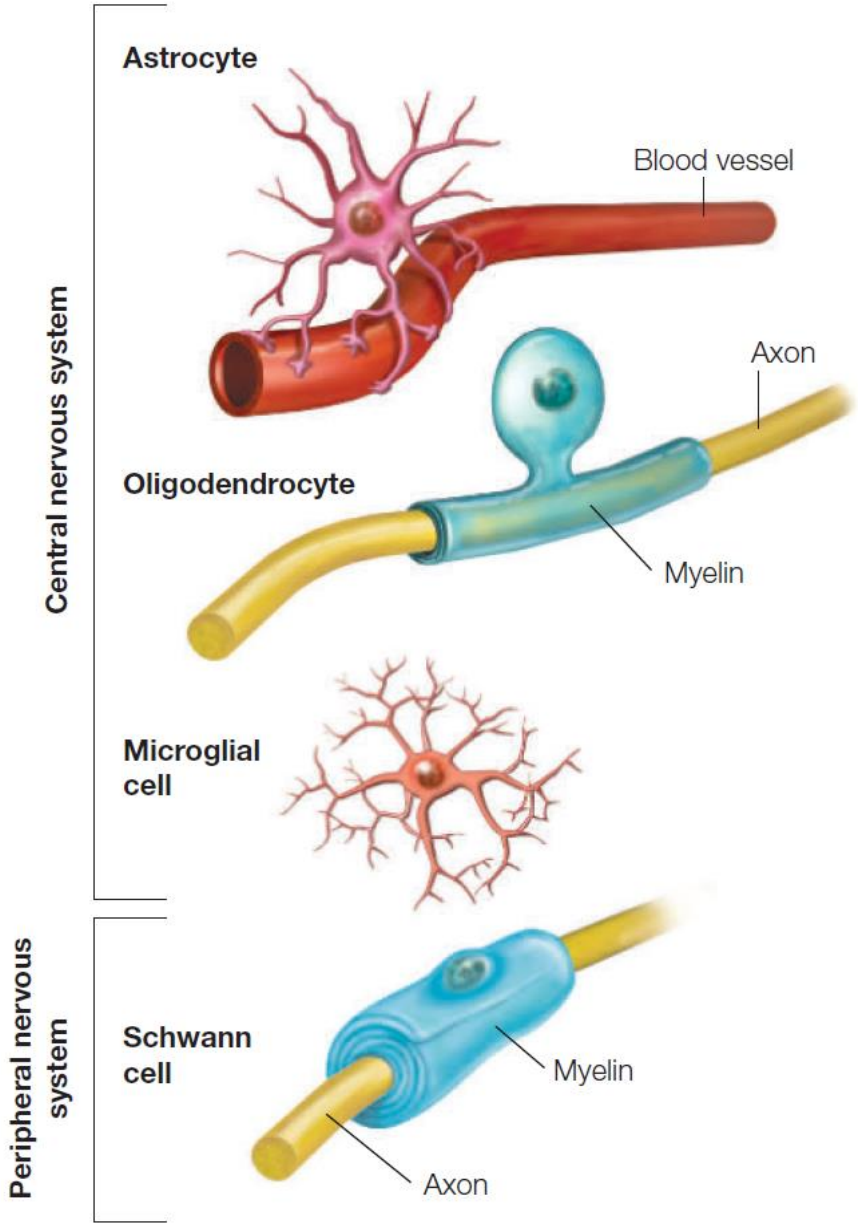




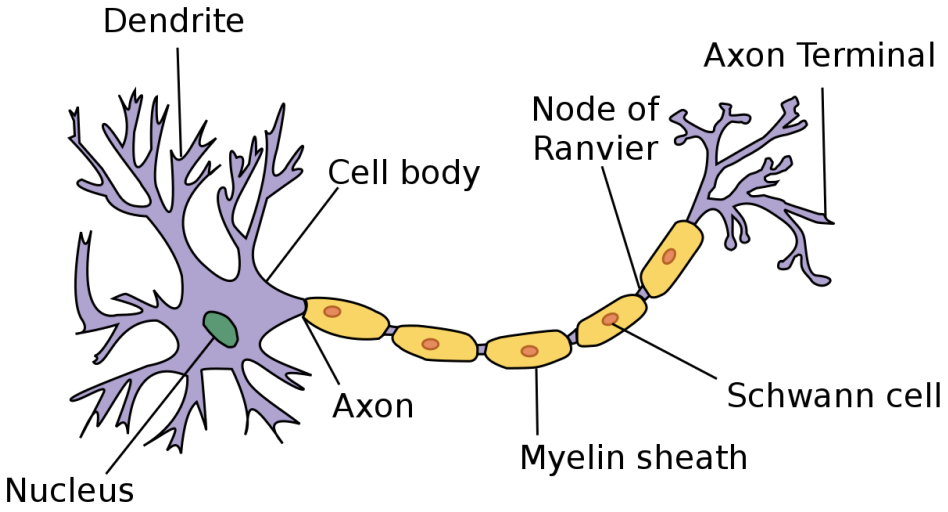
What gives the white color in the white matter seen in the MRI scans?



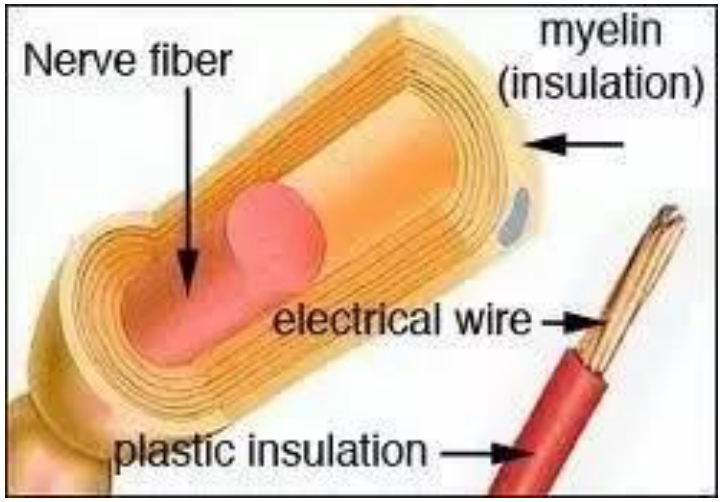
Glial cells (support neurons)



A
neuron



What is the function of the myelin sheath?

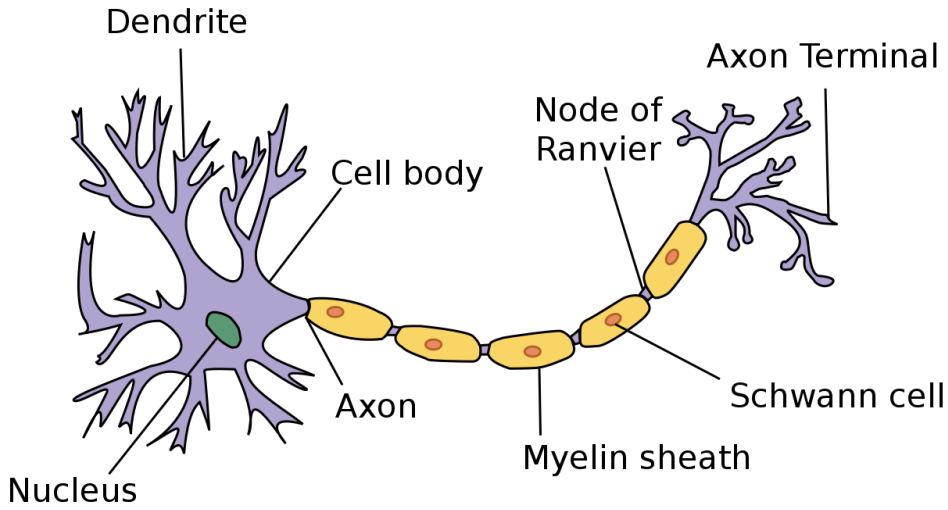


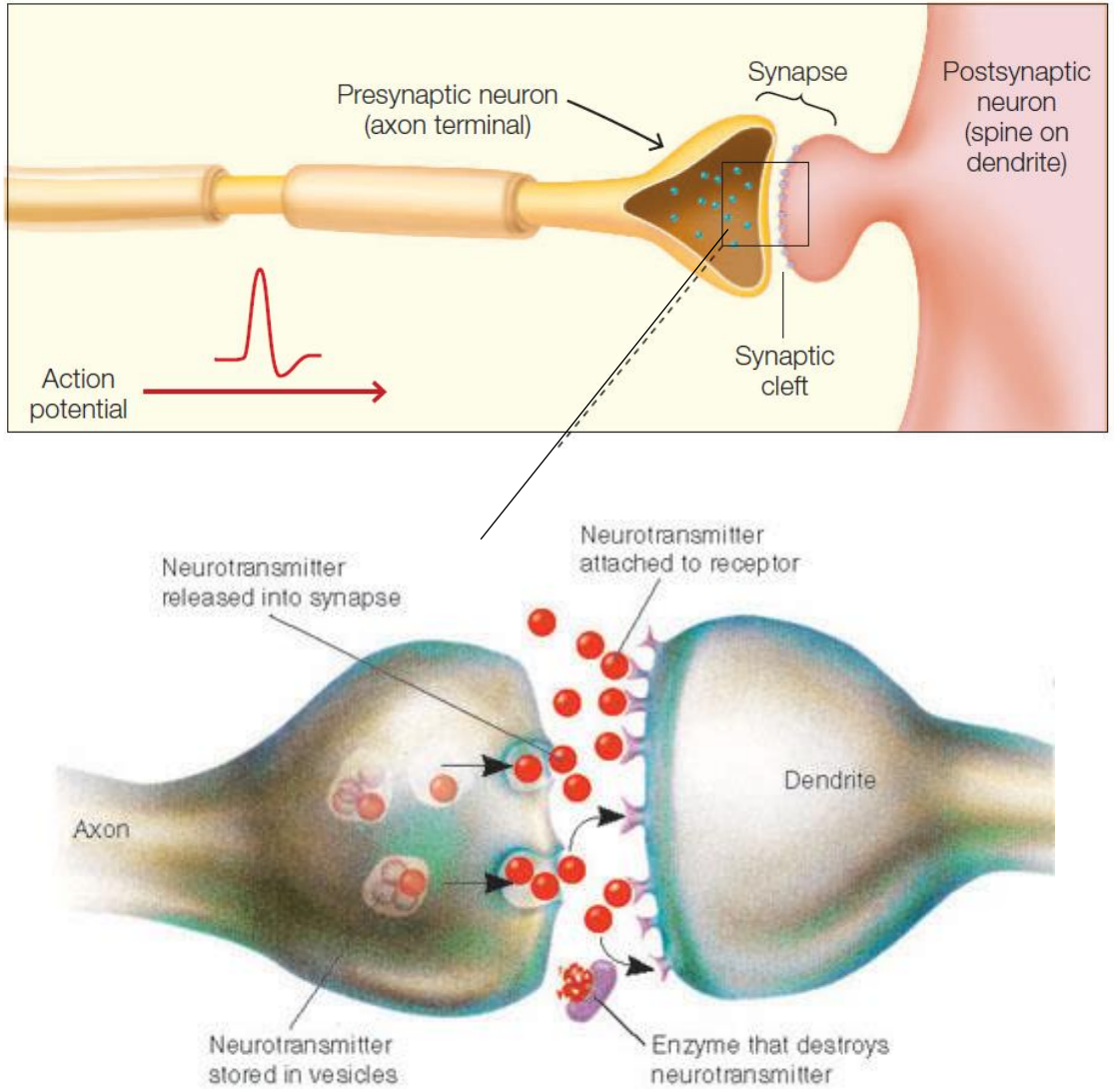
How does a neuron transmit a signal?

How does a neuron switch on?

Class 2
Neuronal Function

How does a neuron transmit a signal?





Synaptic cleft

a narrow gap of about 20 nanometers

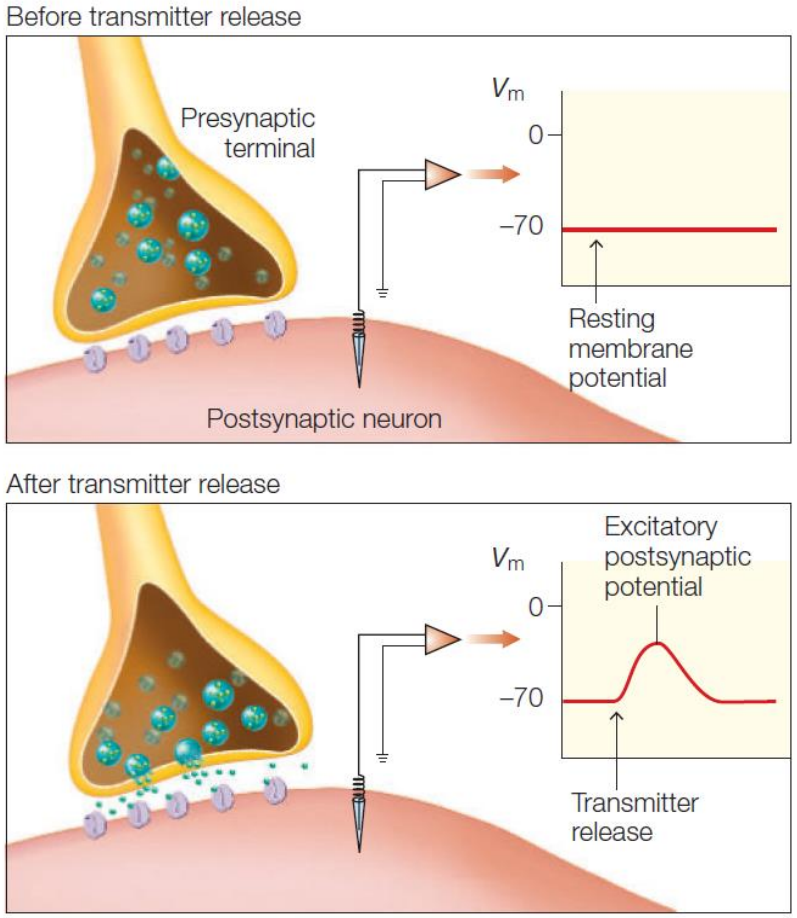
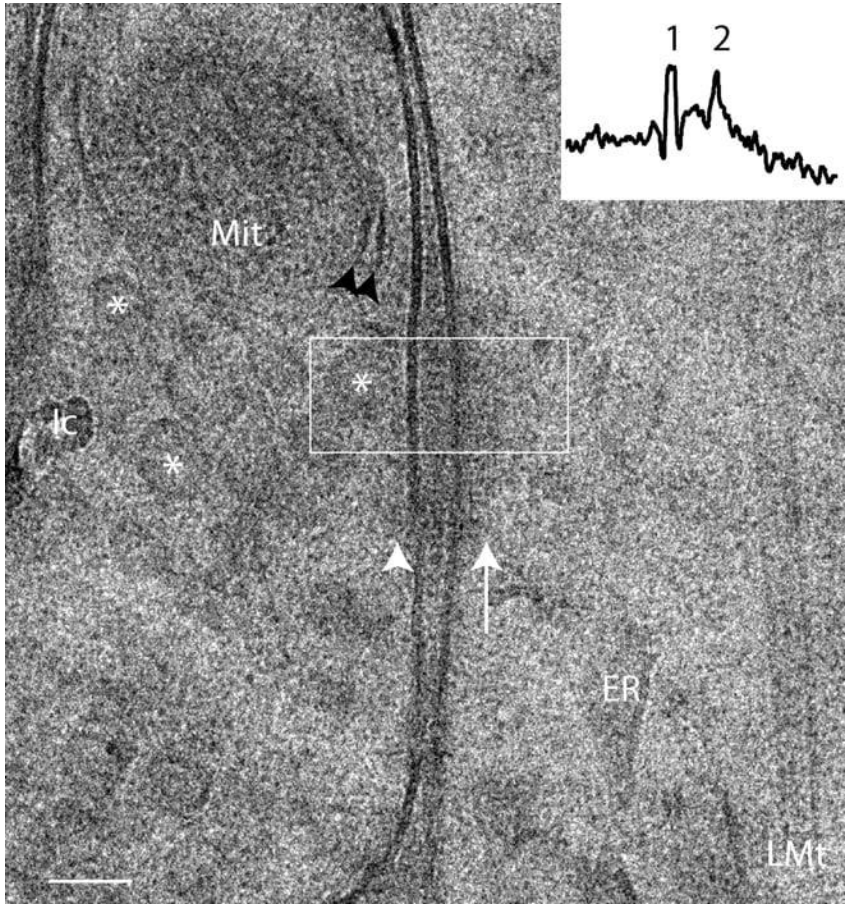
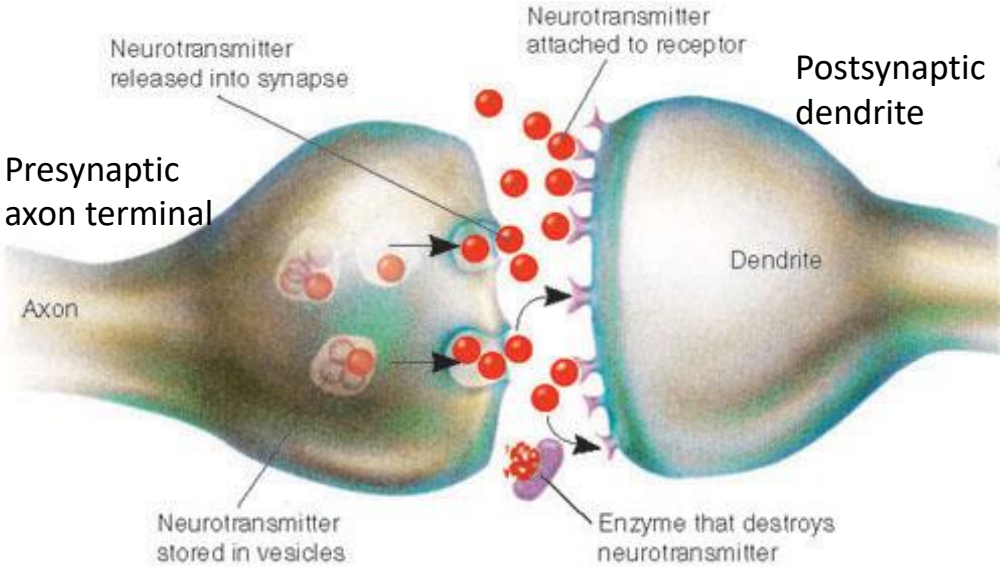


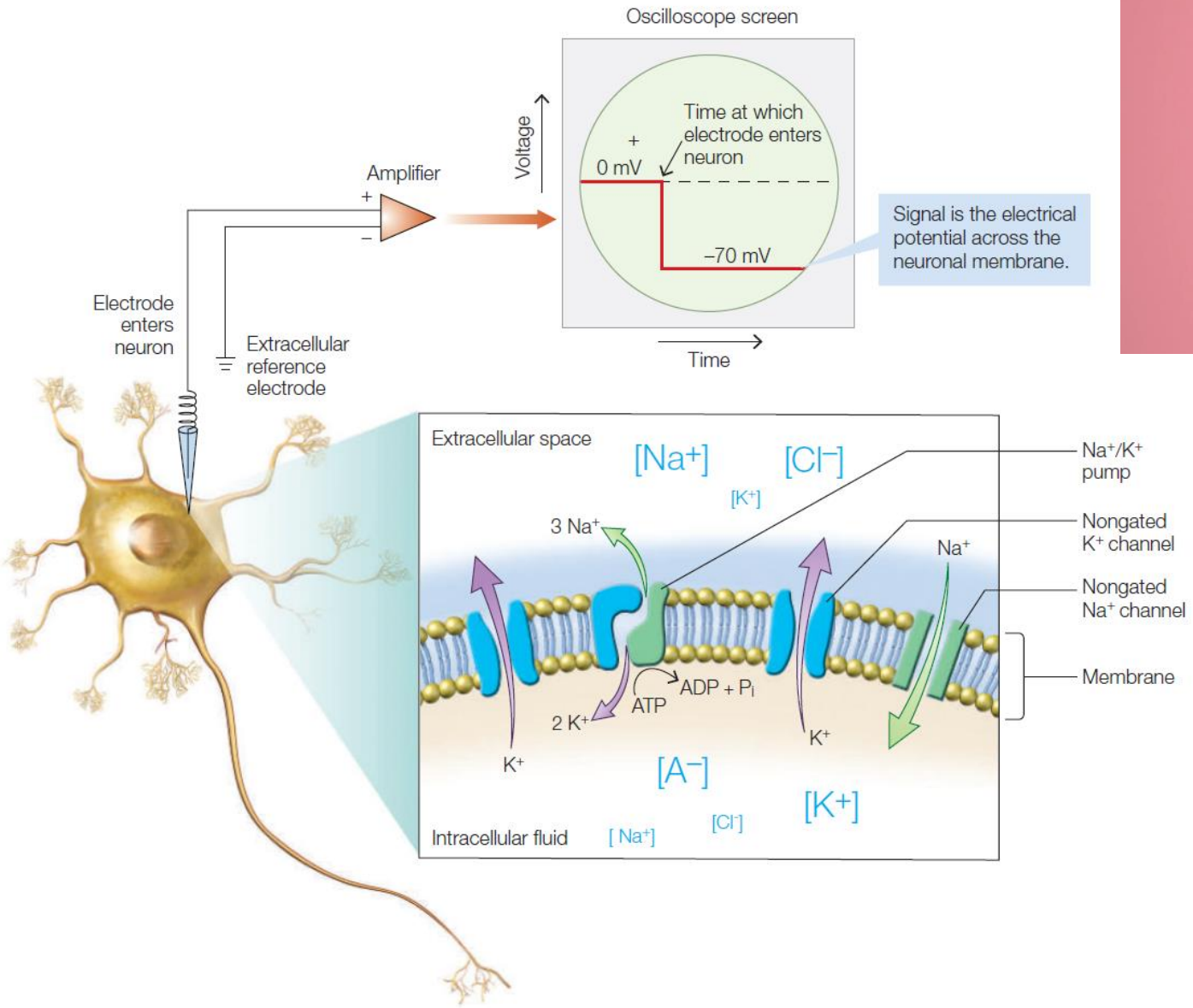
FIGURE 2.14 Neurotransmitter leading to a postsynaptic potential. The binding of neurotransmitter to the postsynaptic membrane receptors changes the membrane potential (V_m). These postsynaptic potentials can be either excitatory (depolarizing the membrane), as shown here, or inhibitory (hyperpolarizing the membrane).

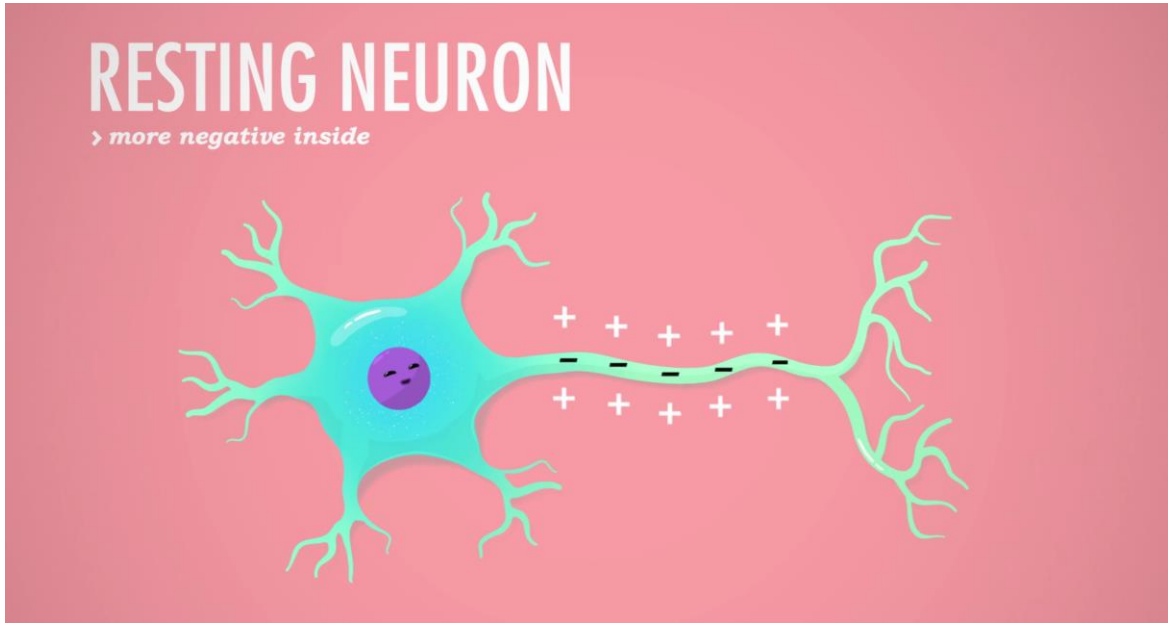
Synaptic cleft/gap



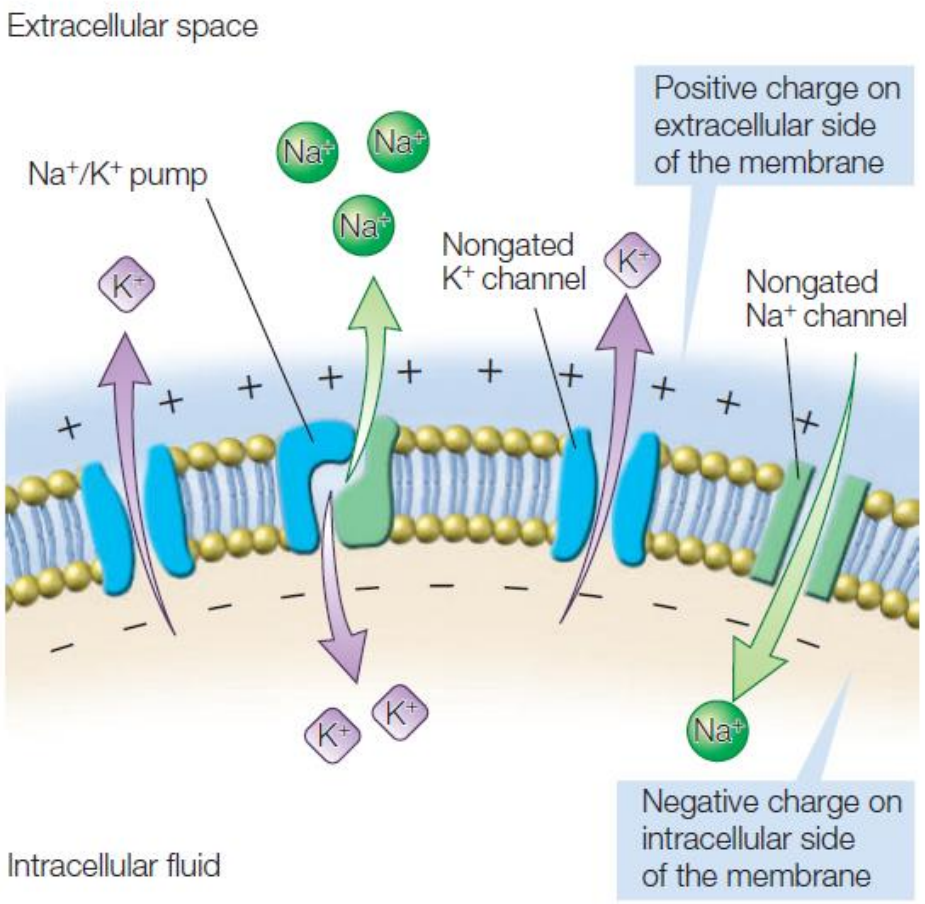


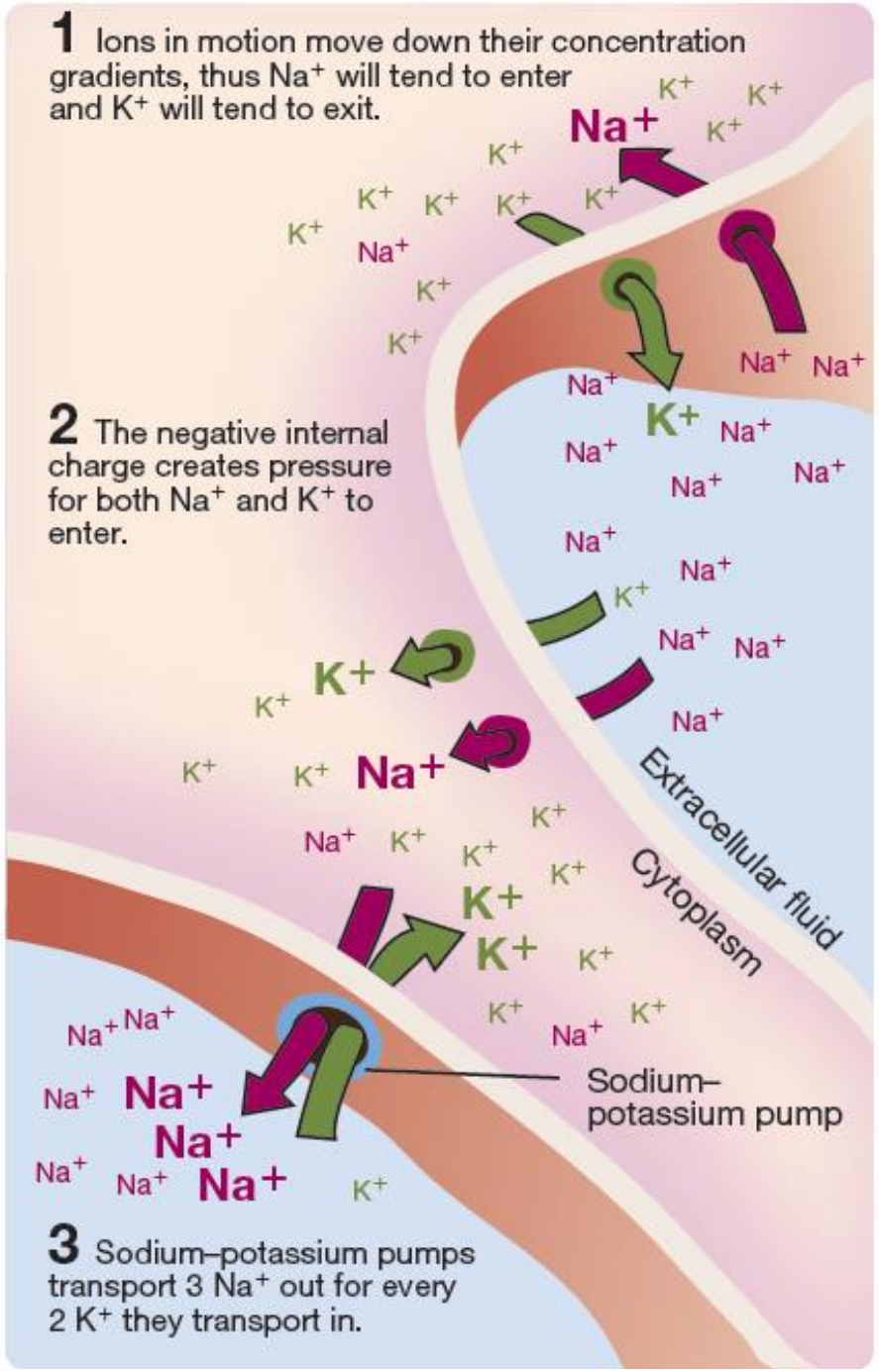
A neuron at rest



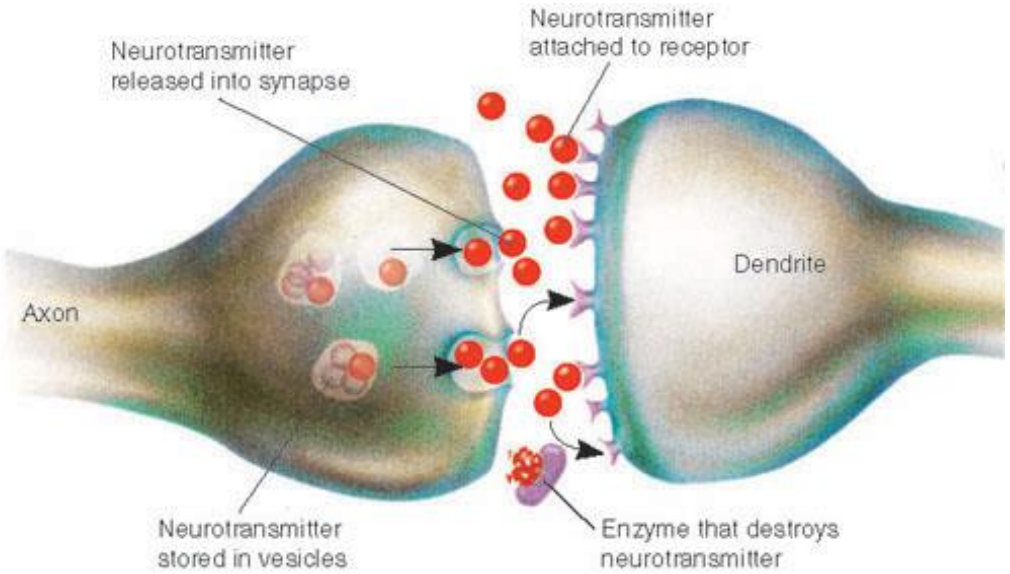


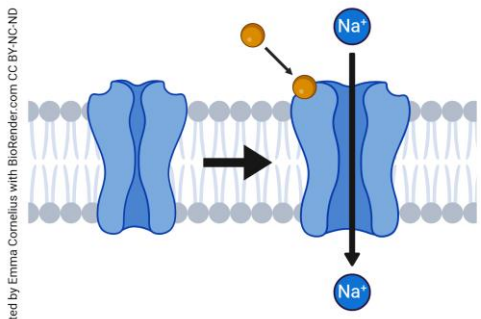
Na-K pump


Extracellular space
Positive charge on extracellular side of the membrane
Na+/K+ pump
Nongated K+ channel
Nongated Na+ channel
Negative charge on intracellular side of the membrane
Intracellular fluid


1 Ions in motion move down their concentration gradients, thus Na+ will tend to enter and K+ will tend to exit.
2 The negative internal charge creates pressure for both Na+ and K+ to enter.
Extracellular fluid
Cytoplasm
Sodium–potassium pump
3 Sodium–potassium pumps transport 3 Na+ out for every 2 K+ they transport in.

https://uen.pressbooks.pub/anatomyphysiology2/chapter/ion-channels/





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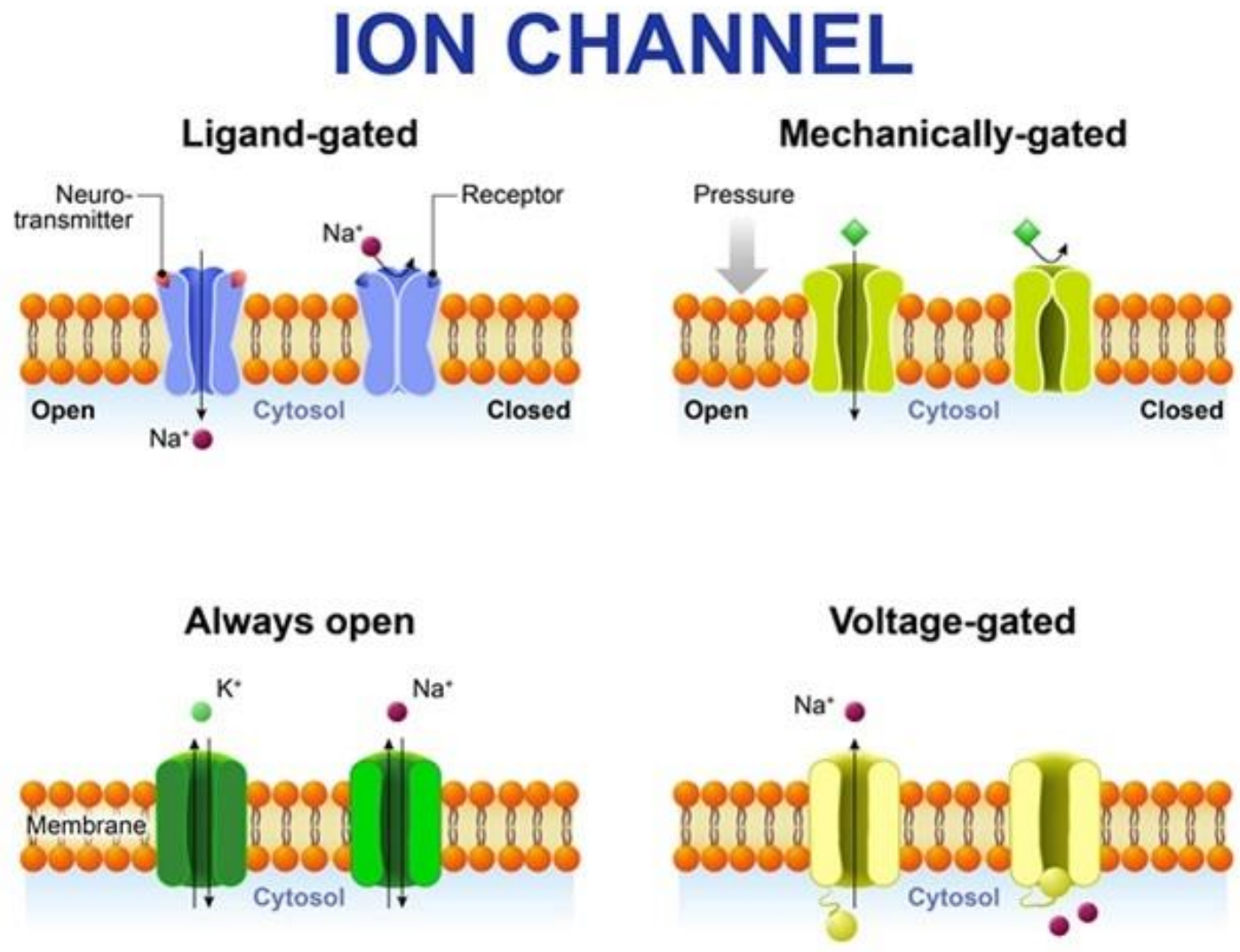
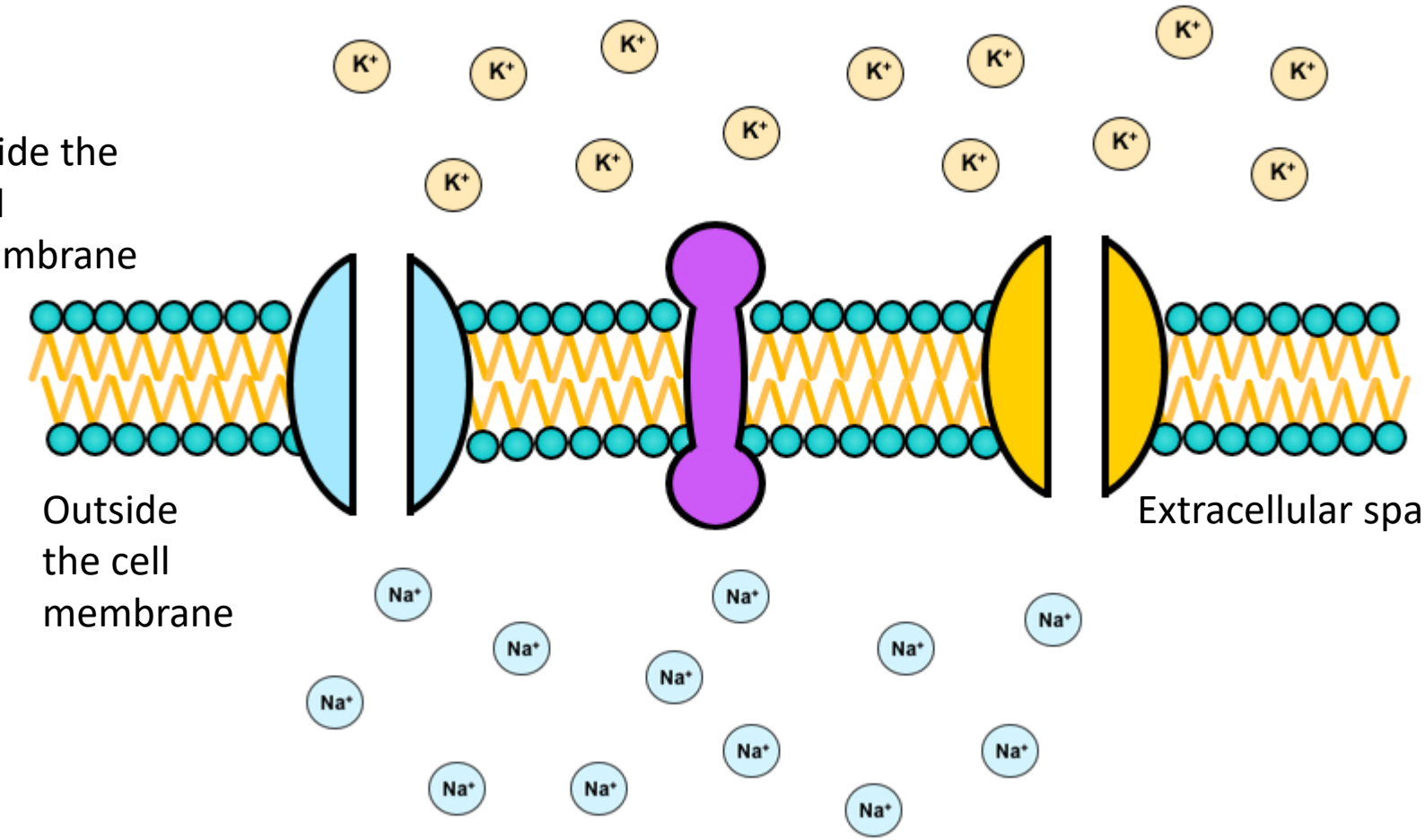
Ligand-gated channel

Opens (closes) in response to a specific extracellular neurotransmitter



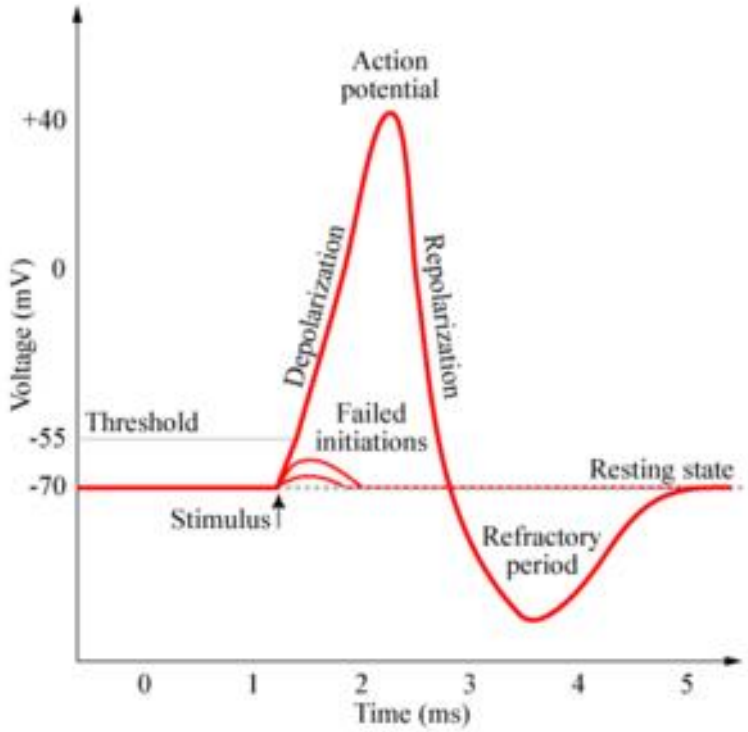
Depolarizace

Inside the cell membrane



Outside the cell membrane

Extracellular space

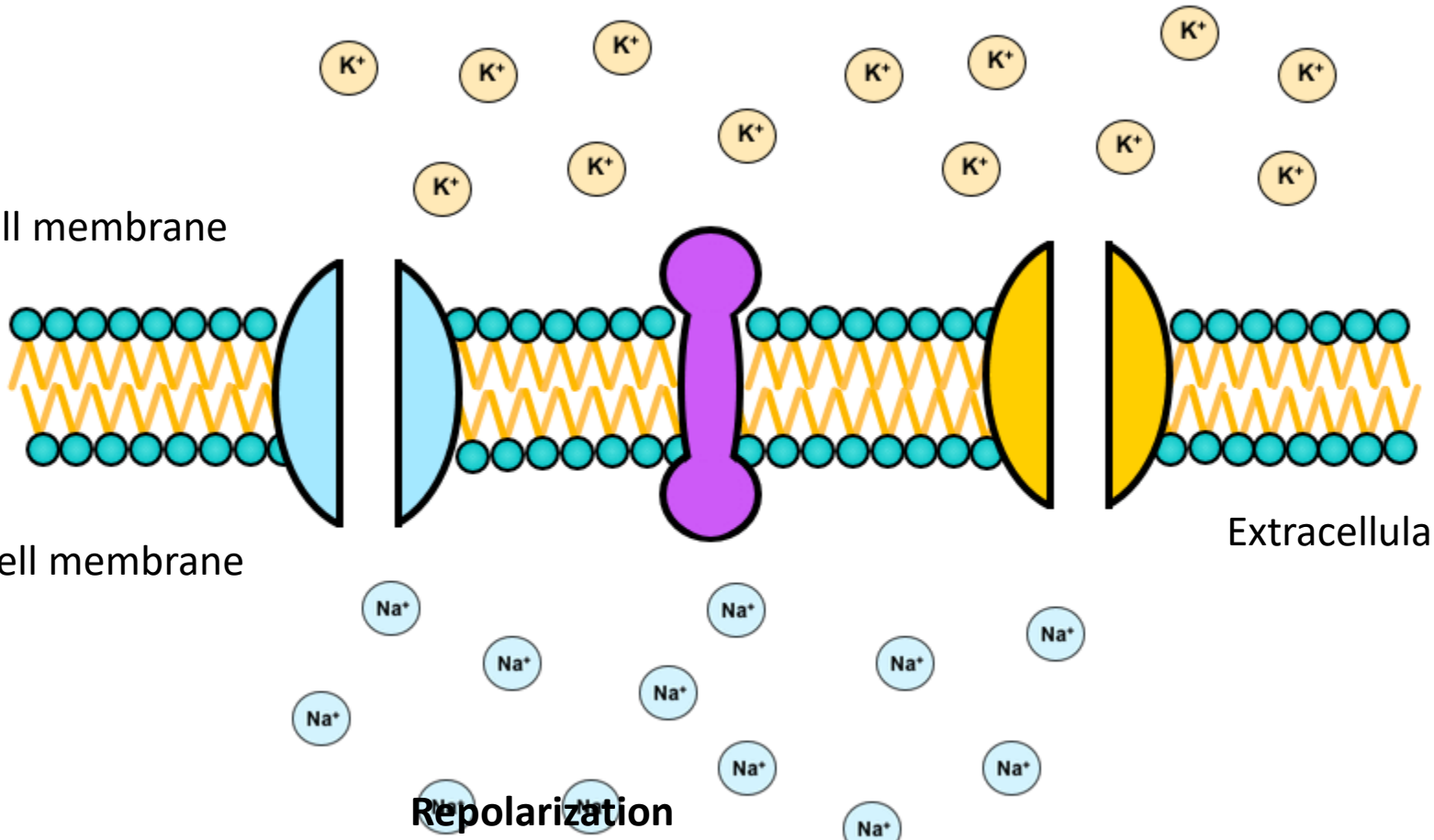


Because of the tension created by the resting membrane potential, the Na+ ions rush inside once the Na+ channels are opened

→ 1 millisecond

With too many + ions inside, the K+ gates open and K+ ions are pushed out to balance the electrostatic & concentration gradient

Depolarizace



Depolarization
Because of the tension created by the resting membrane potential, the Na+ ions rush inside once the Na+ channels are opened

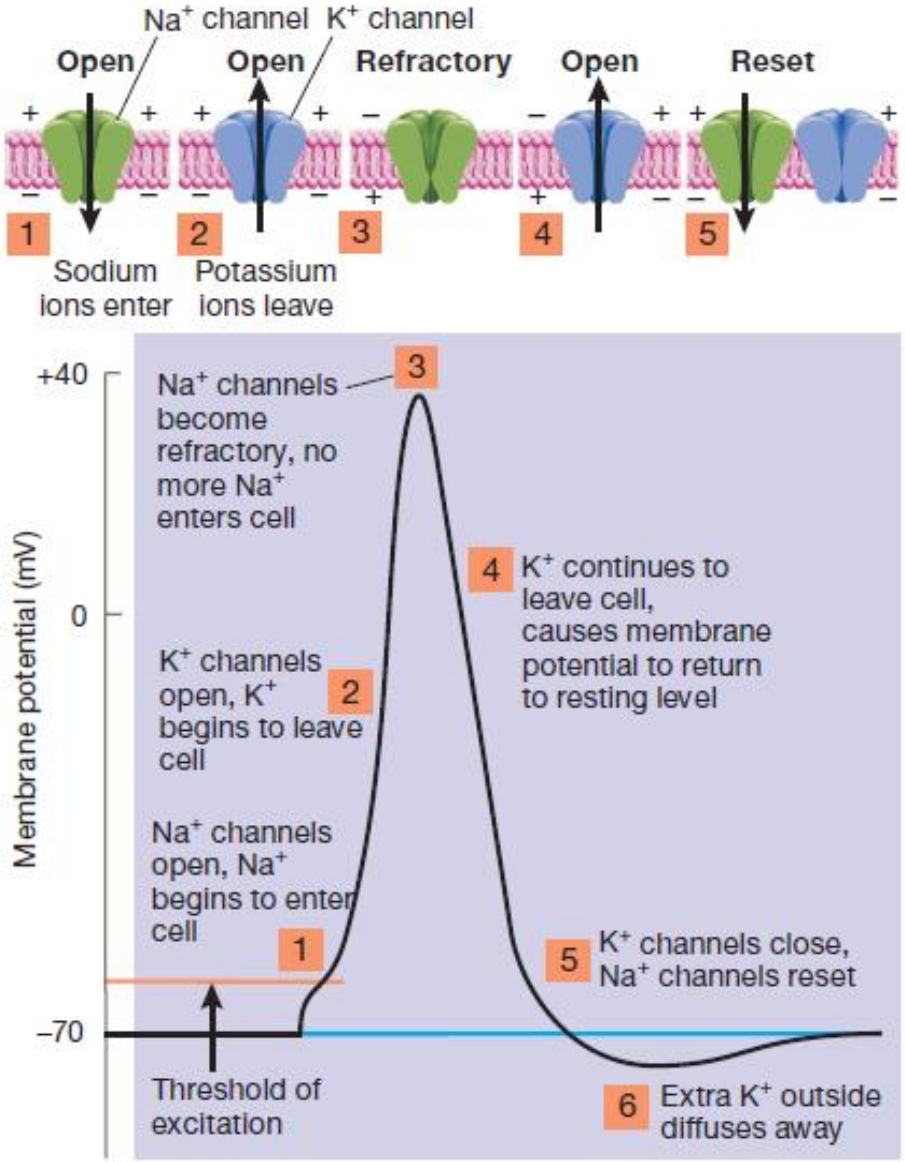
1 millisecond →

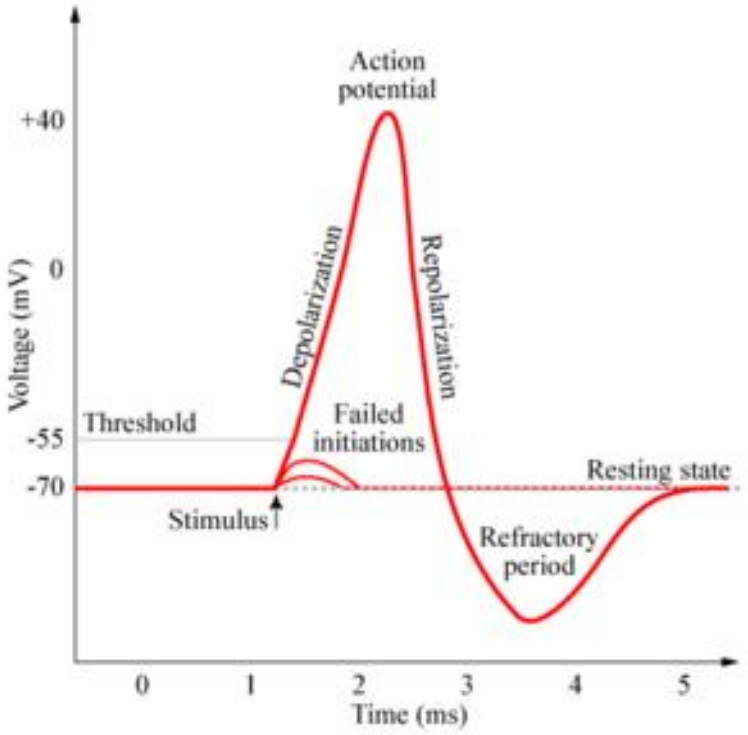
Repolarization
With too many + ions inside, the K+ gates open and K+ ions are pushed out to balance the electrostatic & concentration gradient

1 millisecond →
Na/K pump

Resting membrane potential is restored

Dynamics of an Action Potential (AP)





The whole process takes about 2-3 msec

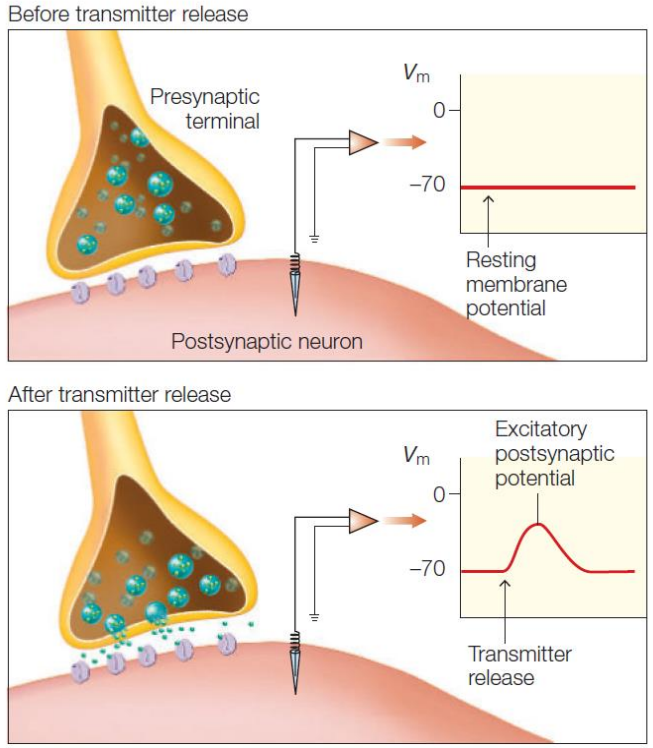
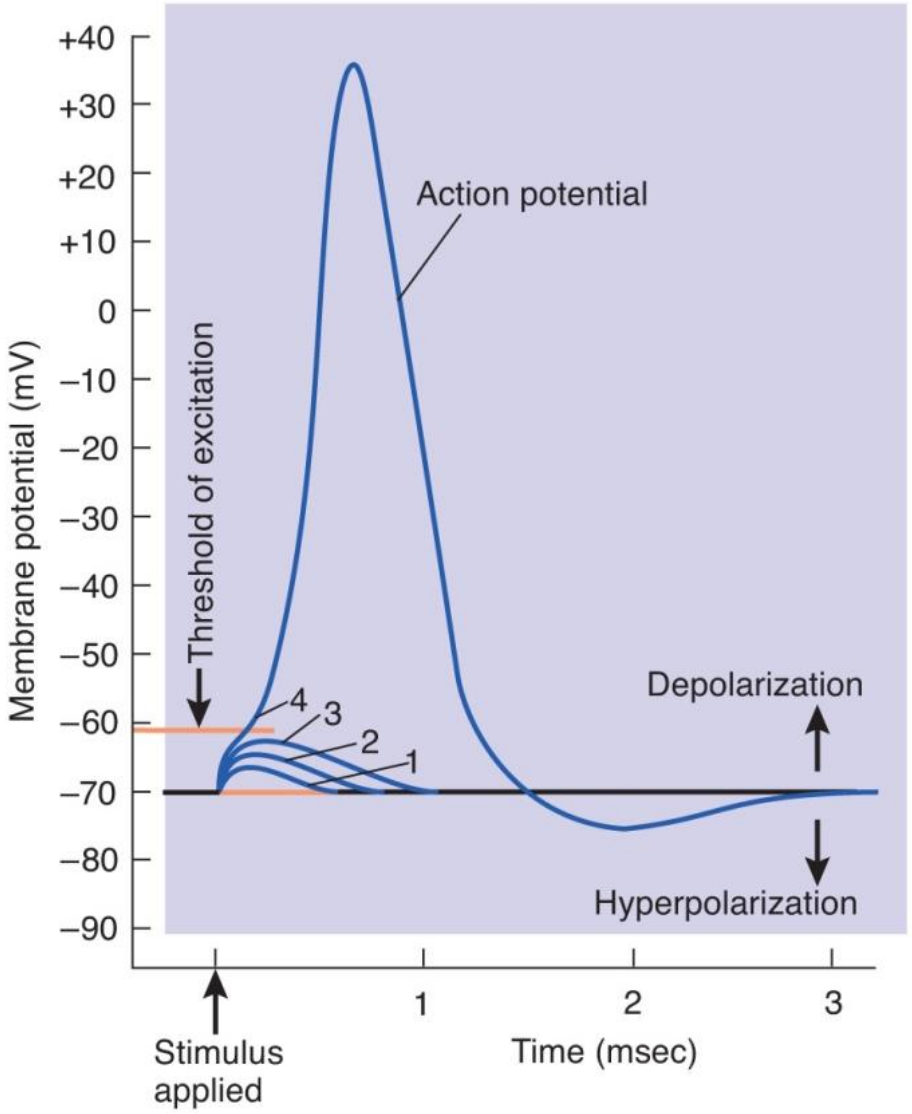
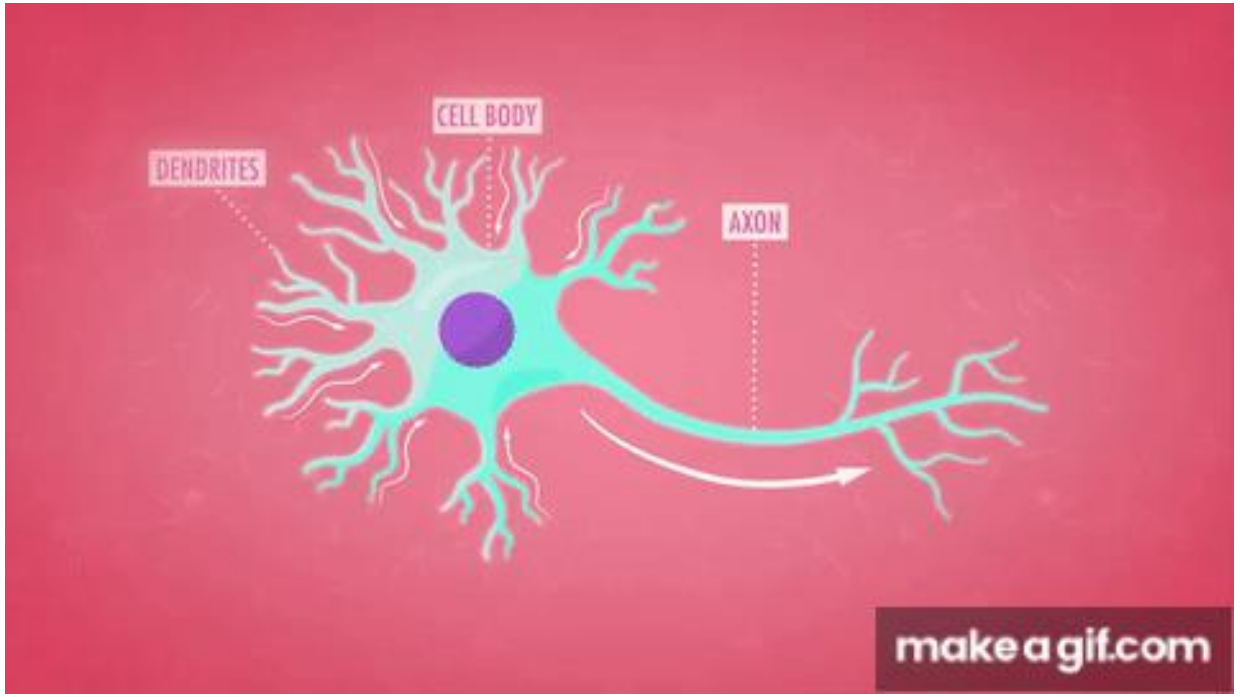


FIGURE 2.14 Neurotransmitter leading to a postsynaptic potential. The binding of neurotransmitter to the postsynaptic membrane receptors changes the membrane potential (V_m). These postsynaptic potentials can be either excitatory (depolarizing the membrane), as shown here, or inhibitory (hyperpolarizing the membrane).

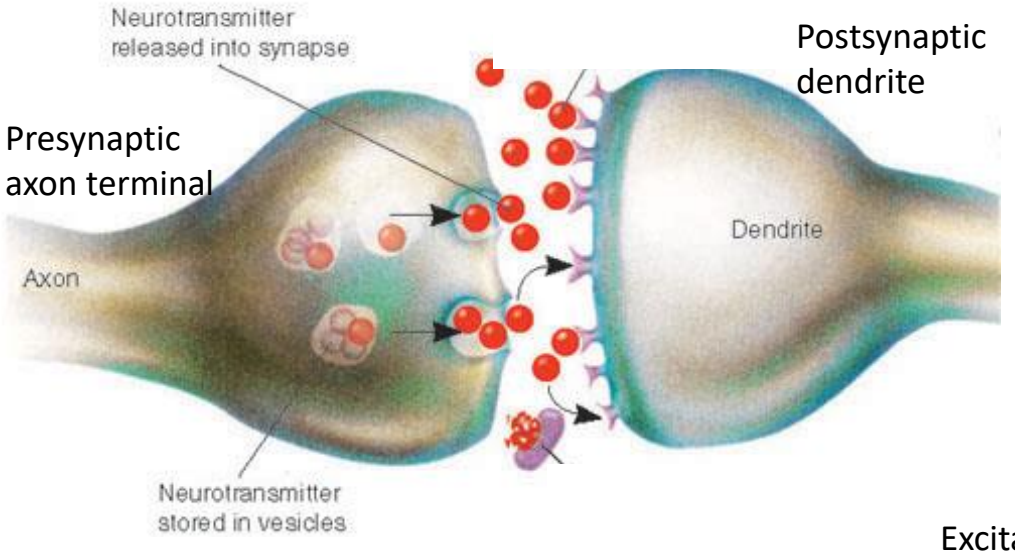
Action Potential (AP) in an axon

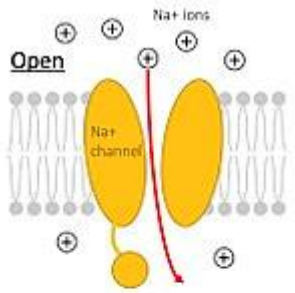
An action potential is an **all or none phenomenon**, i.e. only when depolarization crosses the threshold, an action potential occurs.



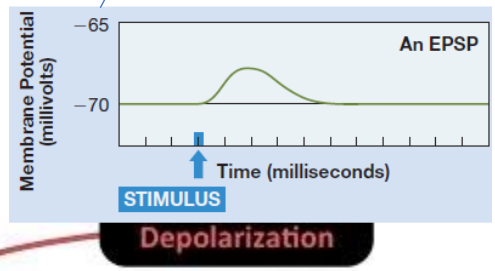


Threshold varies from -65mV to -55mV, across different brain regions

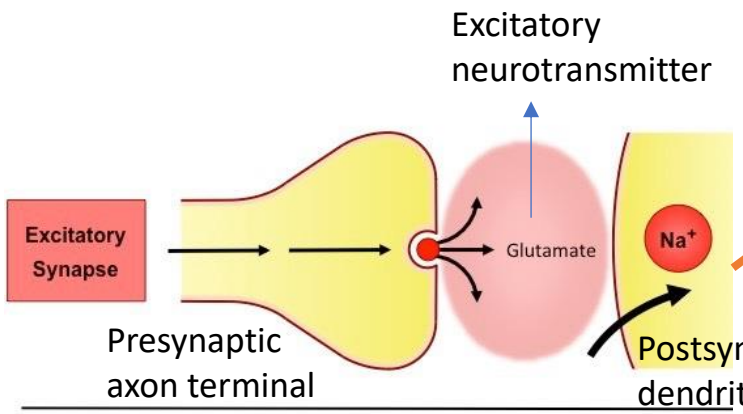


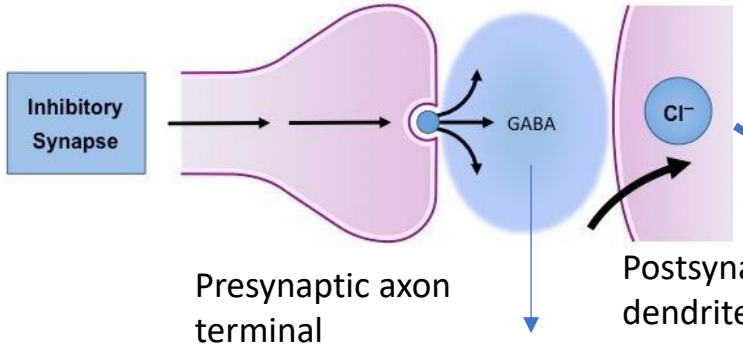


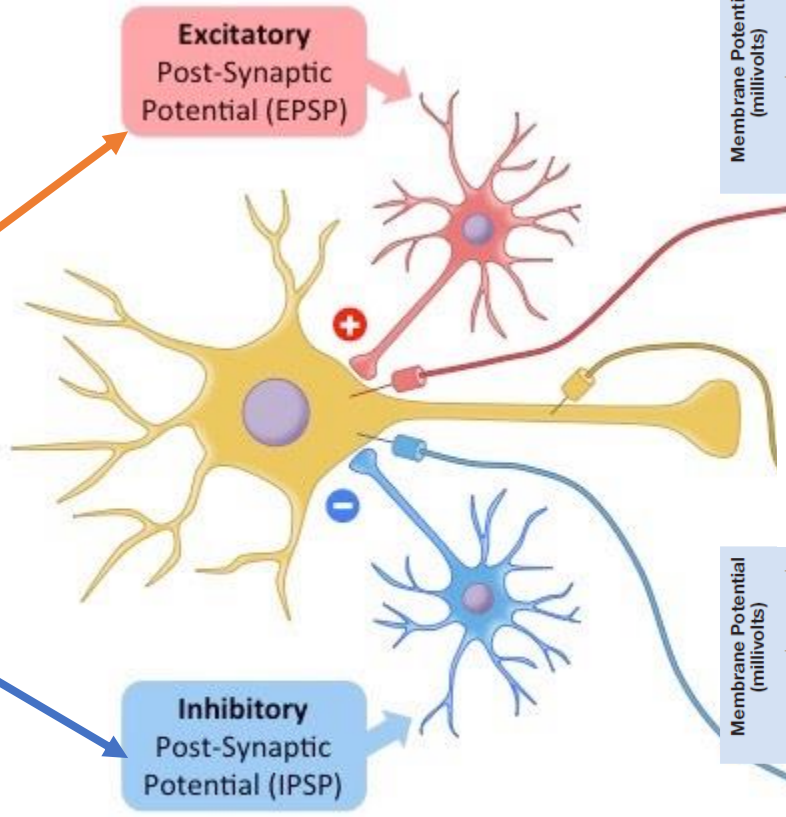
threshold

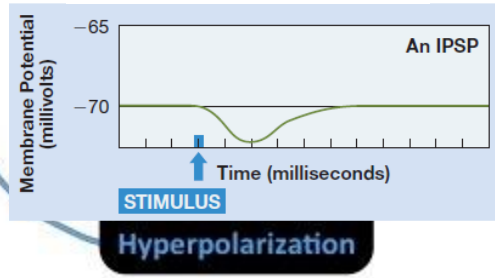


Ion channels/gates are specific to neurotransmitter molecules (shape/size). Depending on which type of ion channels are opened, the signal can be excitatory or inhibitory.



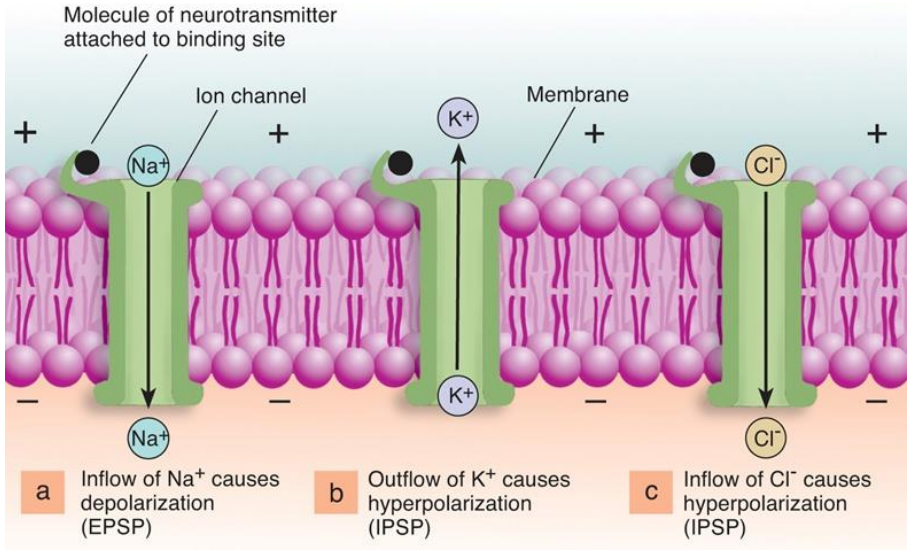






An action potential occurs if the combination of graded potentials exceeds a threshold

Postsynaptic potentials

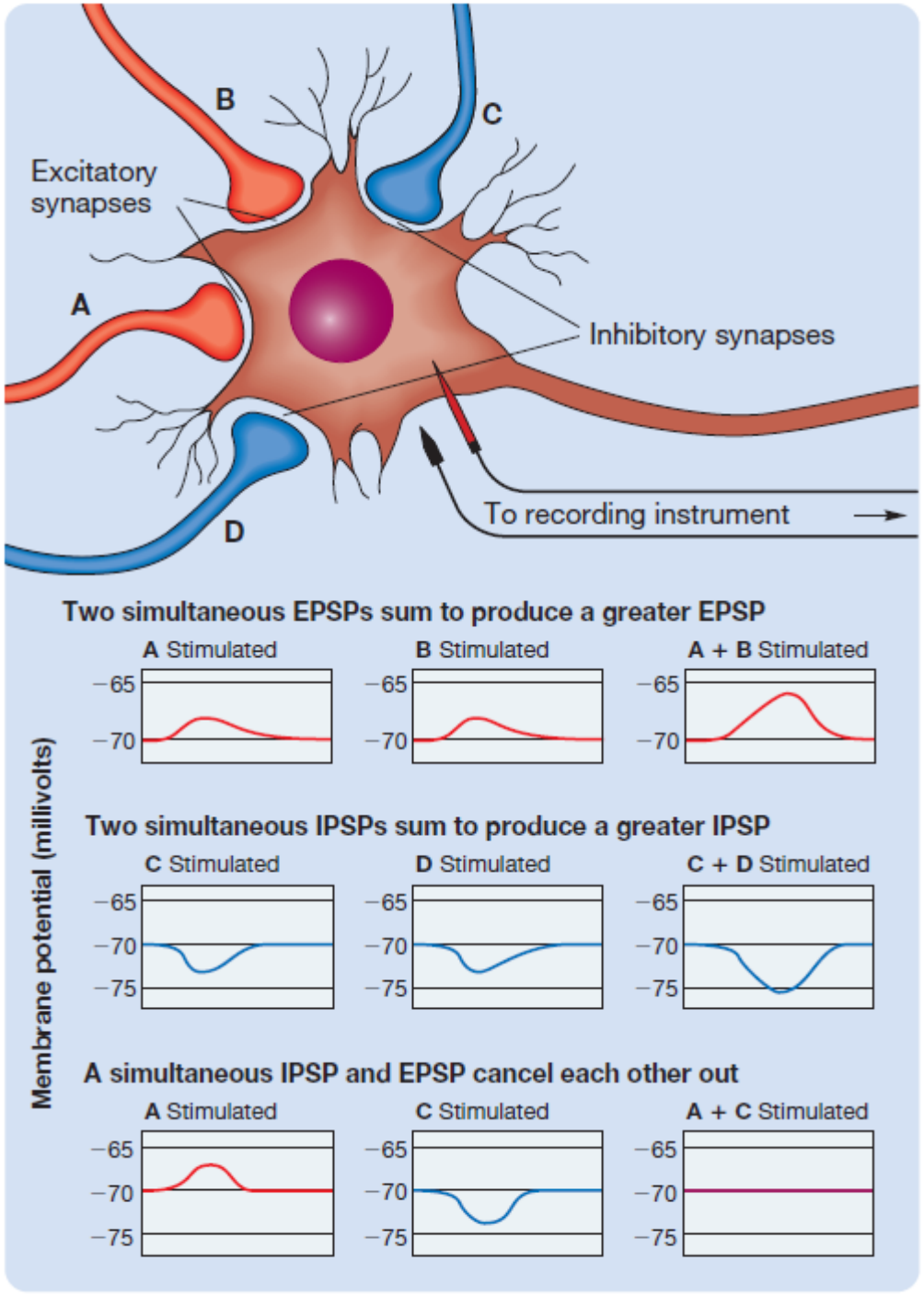


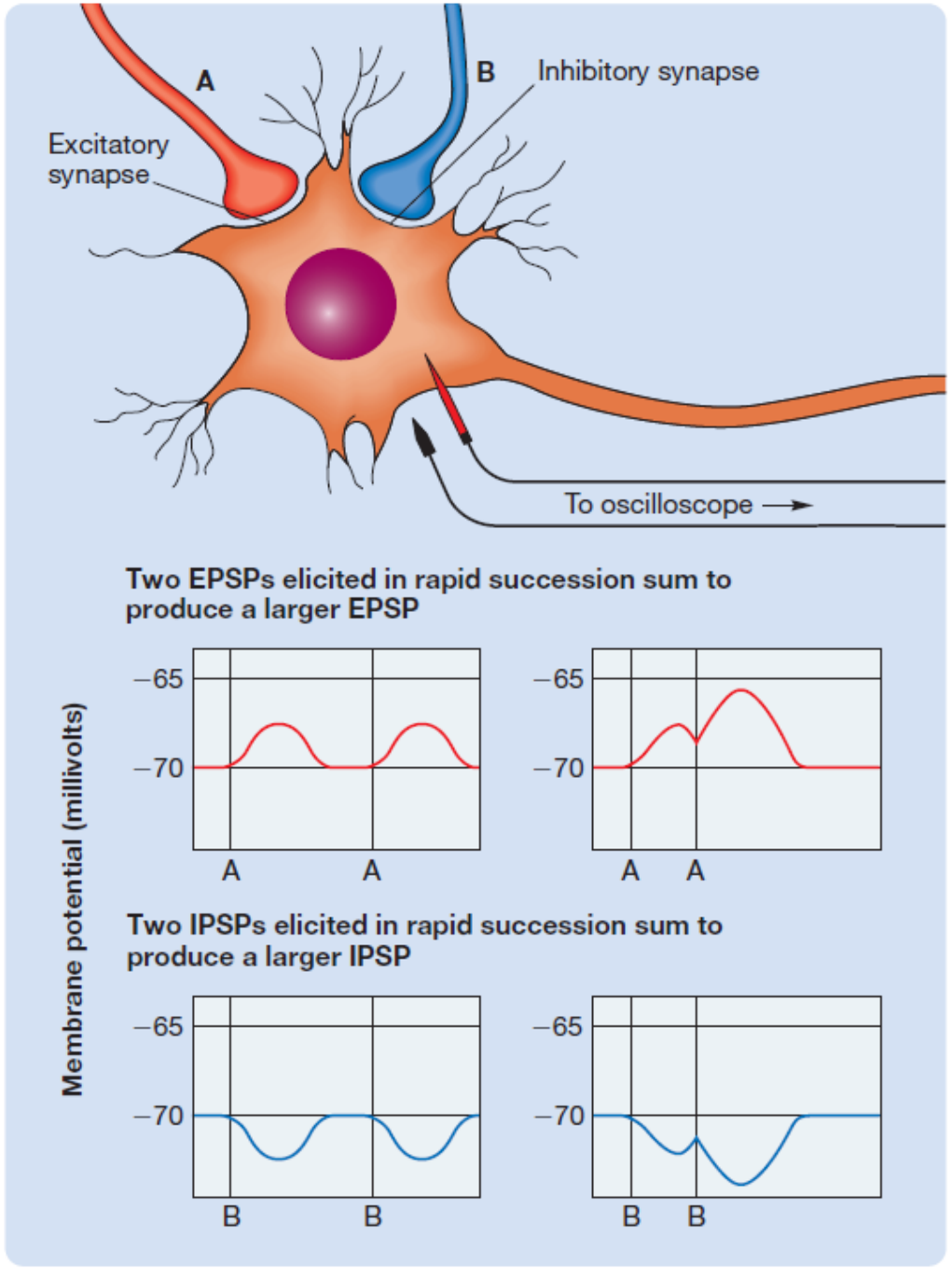
- ***Excitatory postsynaptic potential (EPSP):***
  - An excitatory depolarization of the postsynaptic membrane of a synapse caused by the liberation of a neurotransmitter by the terminal button.
  - Mainly due to the opening of Na^+ channels
  - Positive ions can enter or negative ions can leave the cell to cause a depolarization (Na^+ enter or A^- leave)
  -

- ***Inhibitory postsynaptic potential (IPSP):***
  - An inhibitory hyperpolarization of the postsynaptic membrane of a synapse caused by the liberation of a neurotransmitter by the terminal button
  - Due to the opening of K^+ or Cl^- channels
  - Positive ions can leave or negative ions can enter the cell to cause hyperpolarization (K^+ leave or Cl^- enter)

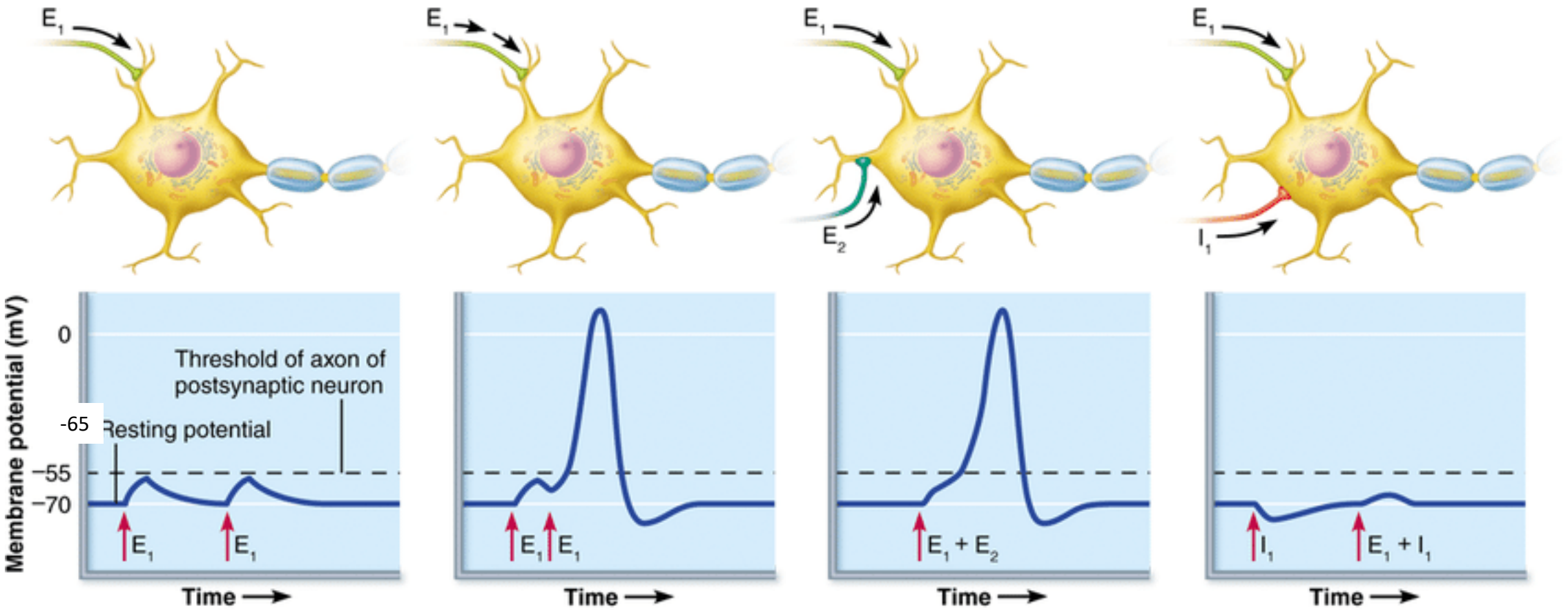
- How does a neuron know when to fire?

Graded Potential at dendrites





Factors that give rise to an action potential



(a) No summation: 2 stimuli separated in time cause EPSPs that do not add together.

(b) Temporal summation: 2 excitatory stimuli close in time cause EPSPs that add together.

(c) Spatial summation: 2 simultaneous stimuli at different locations cause EPSPs that add together.

(d) Spatial summation of EPSPs and IPSPs: Changes in membrane potential can cancel each other out.

Excitatory synapse 1 (E_1)

Excitatory synapse 2 (E_2)

Inhibitory synapse (I_1)

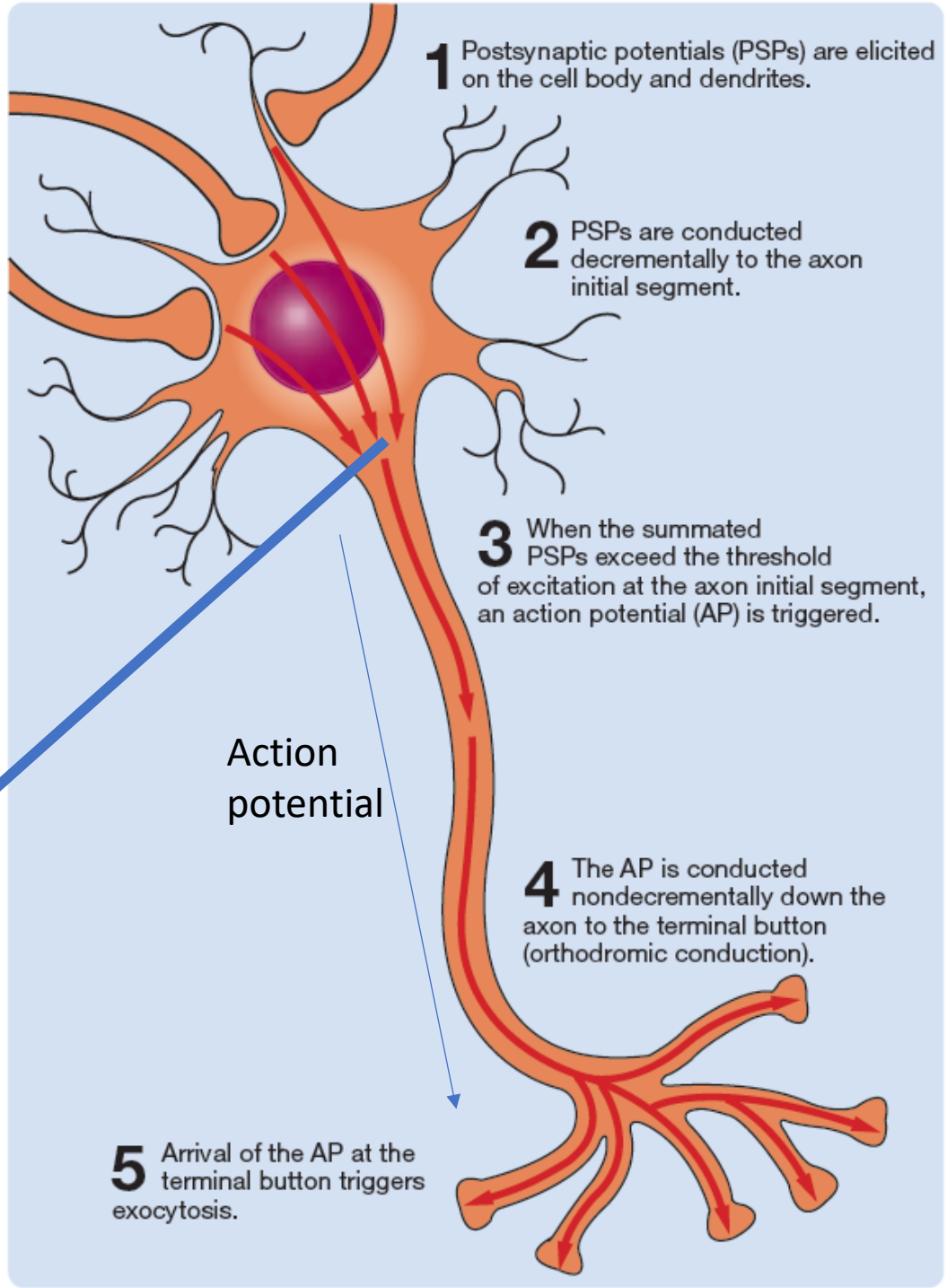
However,

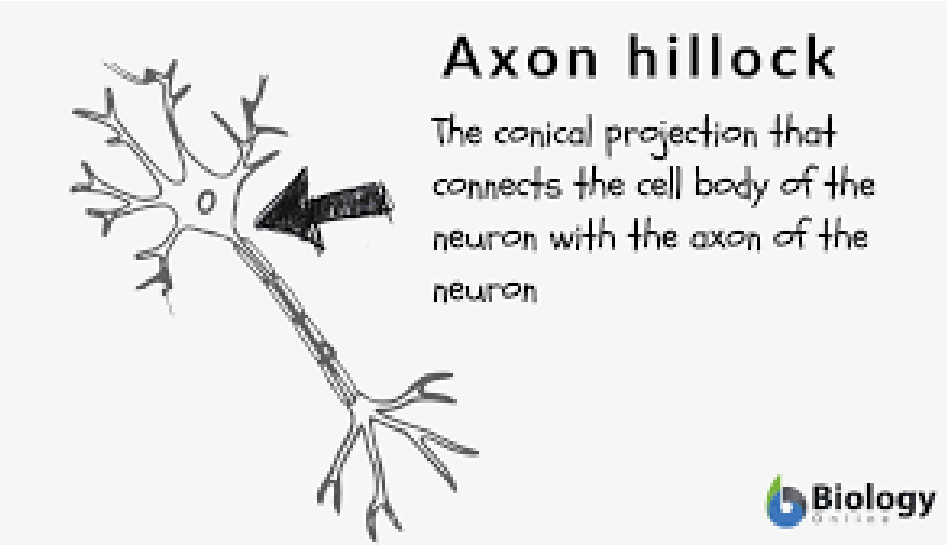
If EPSP > IPSP → AP is produced

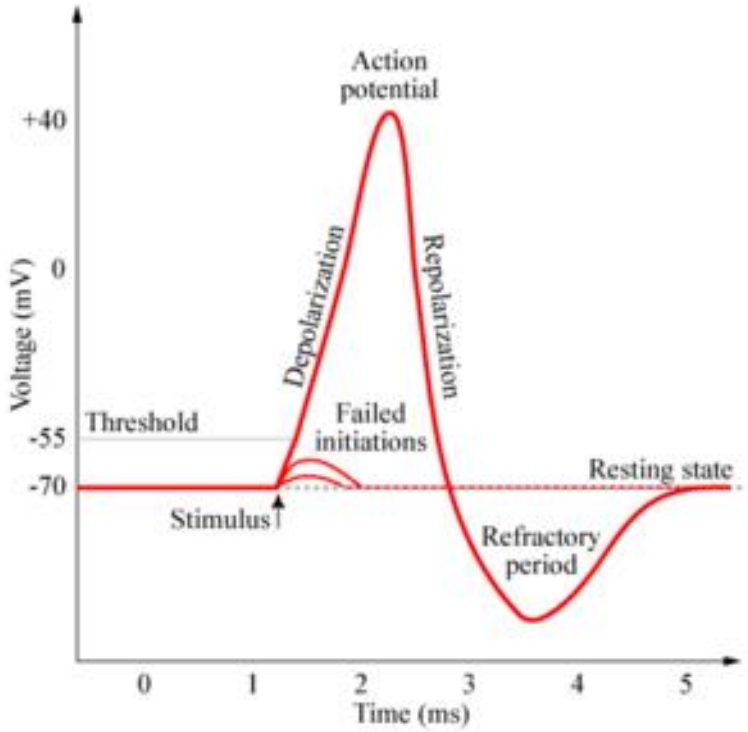
If EPSP < IPSP → no AP is produced

Graded potential

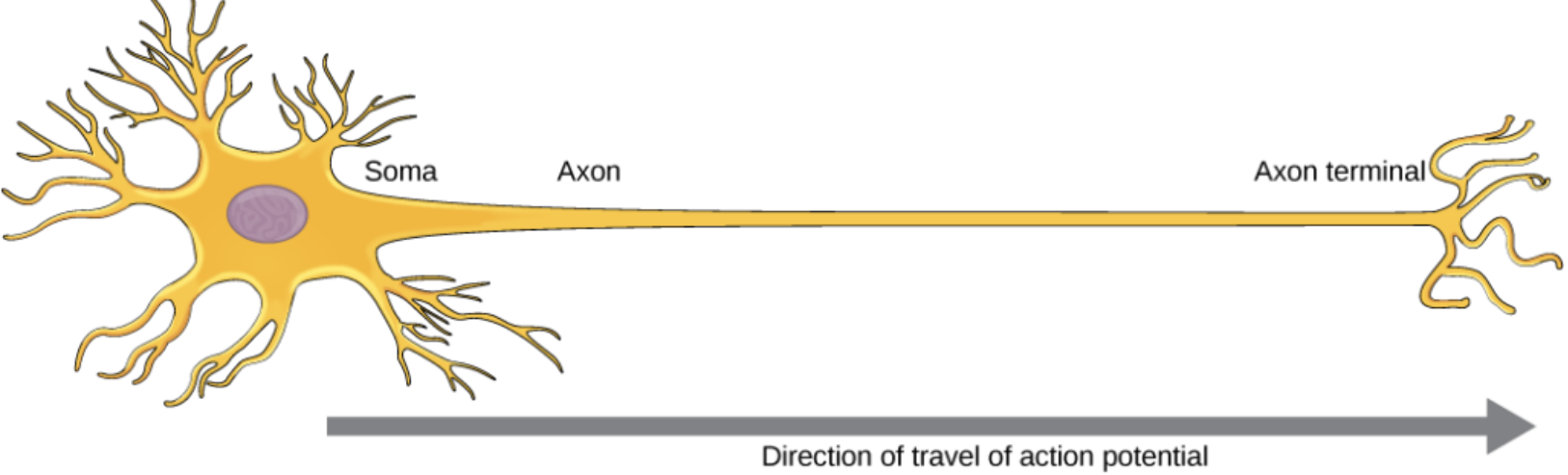
Voltage gated ion channels







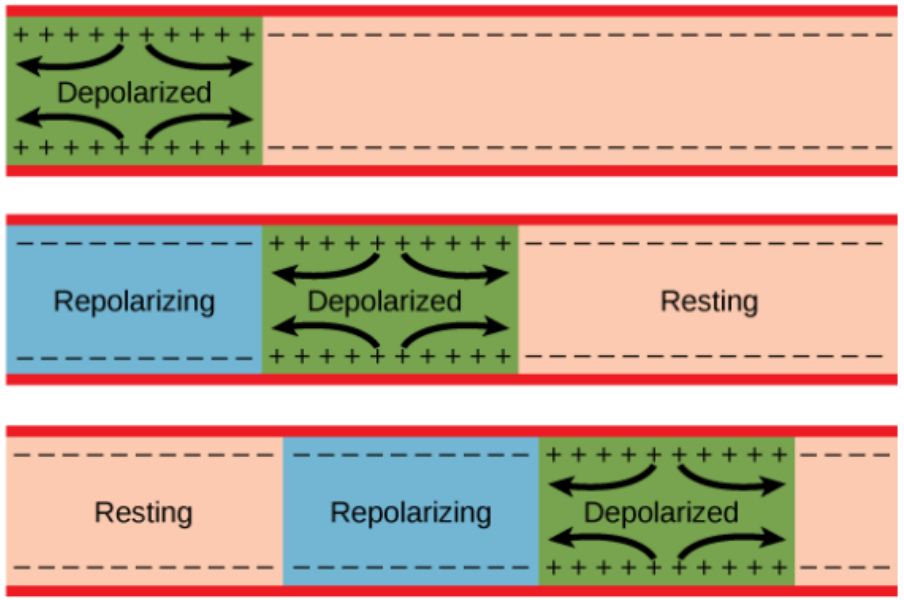
Propagation of an action potential

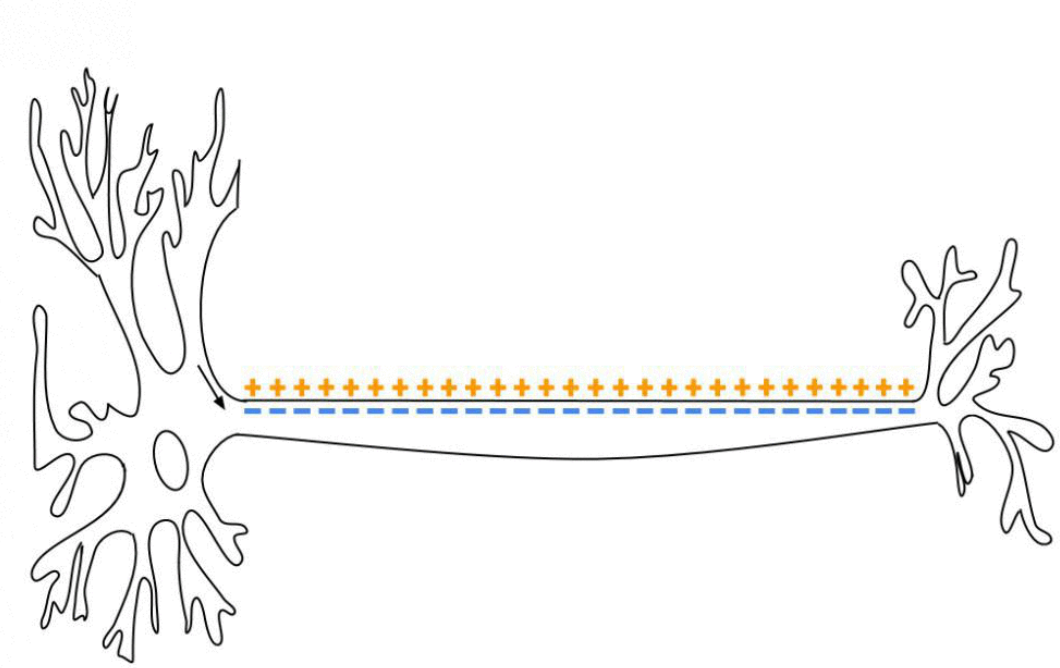


a. In response to a signal, the soma end of the axon becomes depolarized.

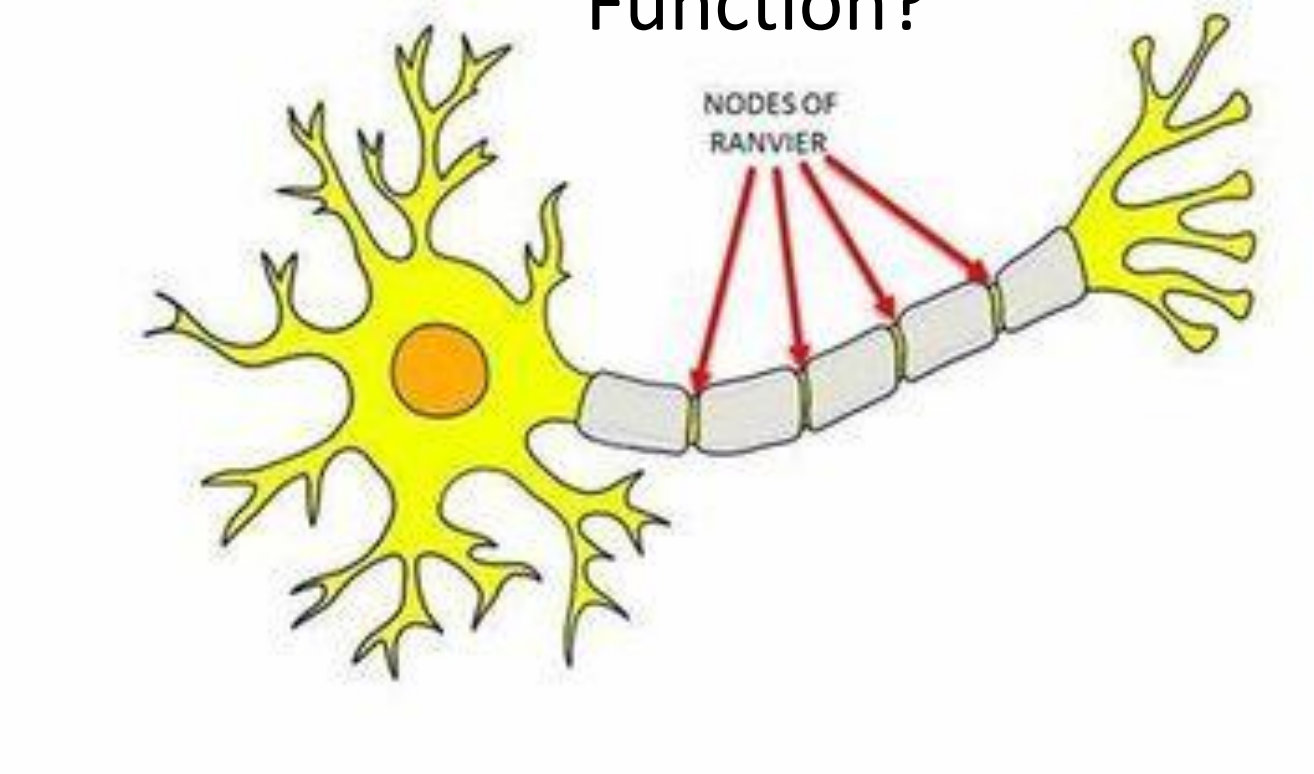
b. The depolarization spreads down the axon. Meanwhile, the first part of the membrane repolarizes. Because Na^+ channels are inactivated and additional K^+ channels have opened, the membrane cannot depolarize again.

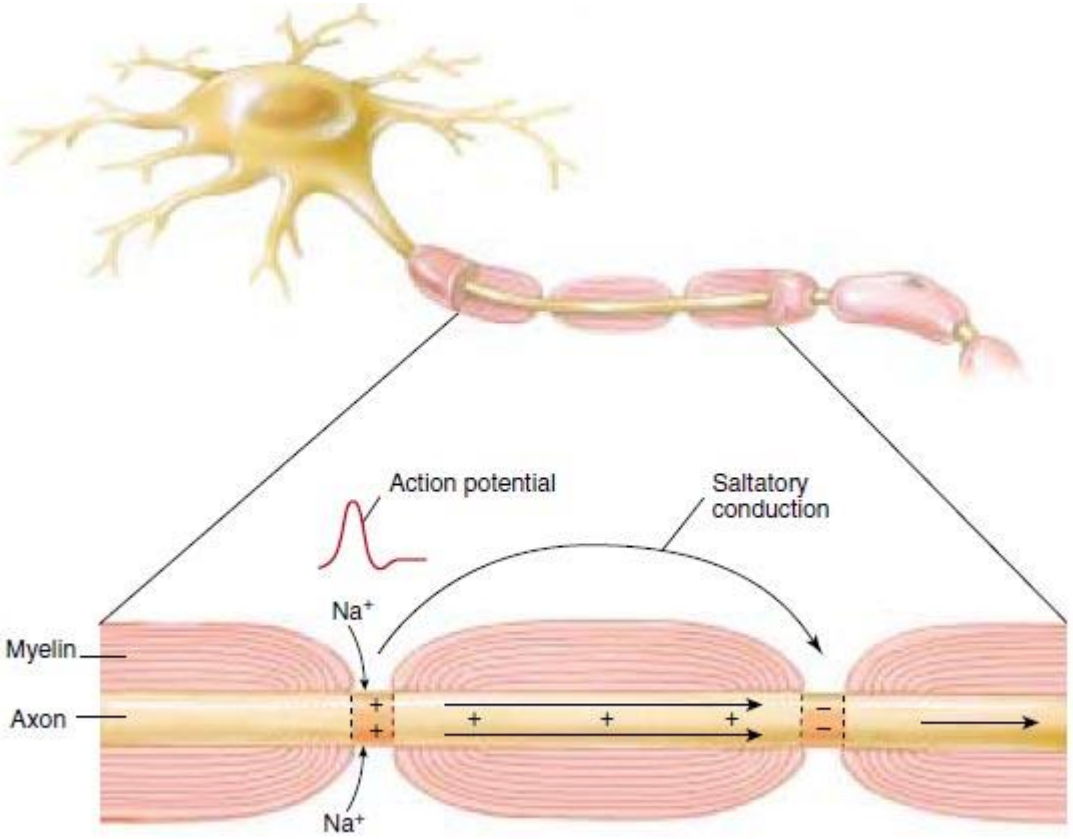
c. The action potential continues to travel down the axon.

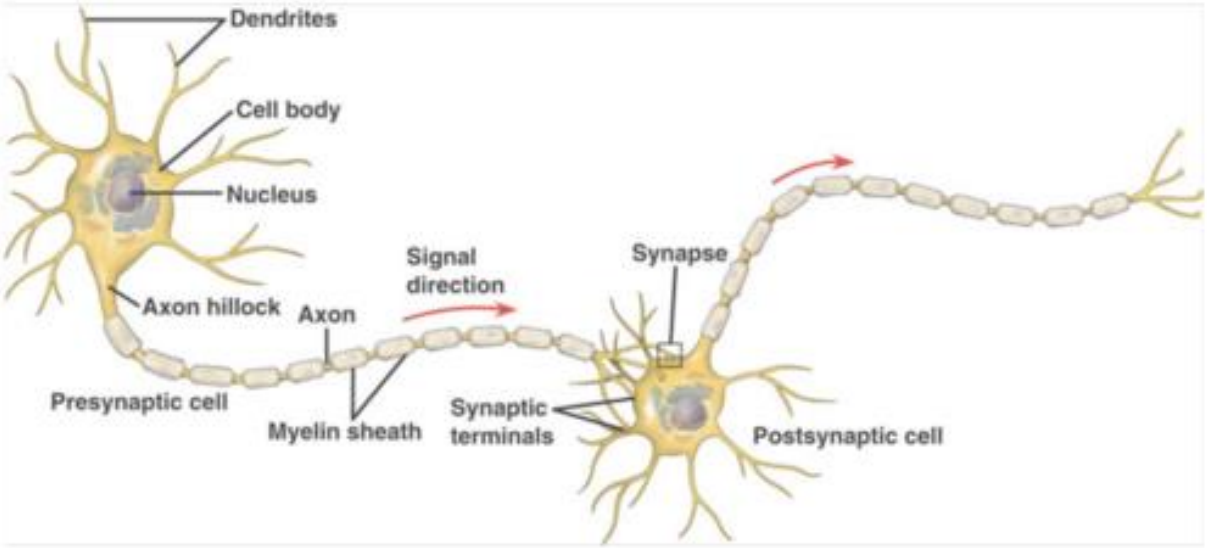


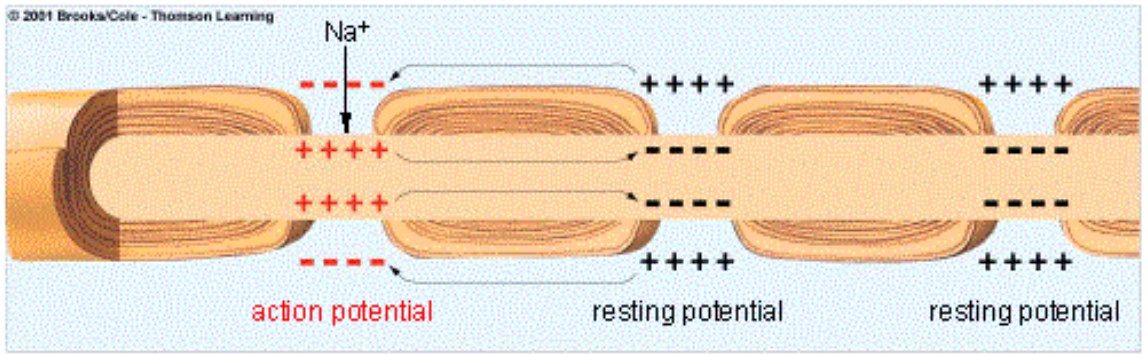


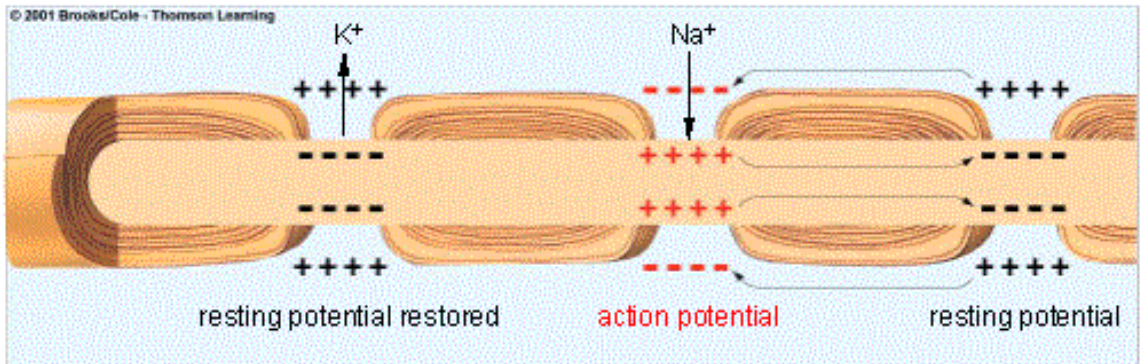
Function?





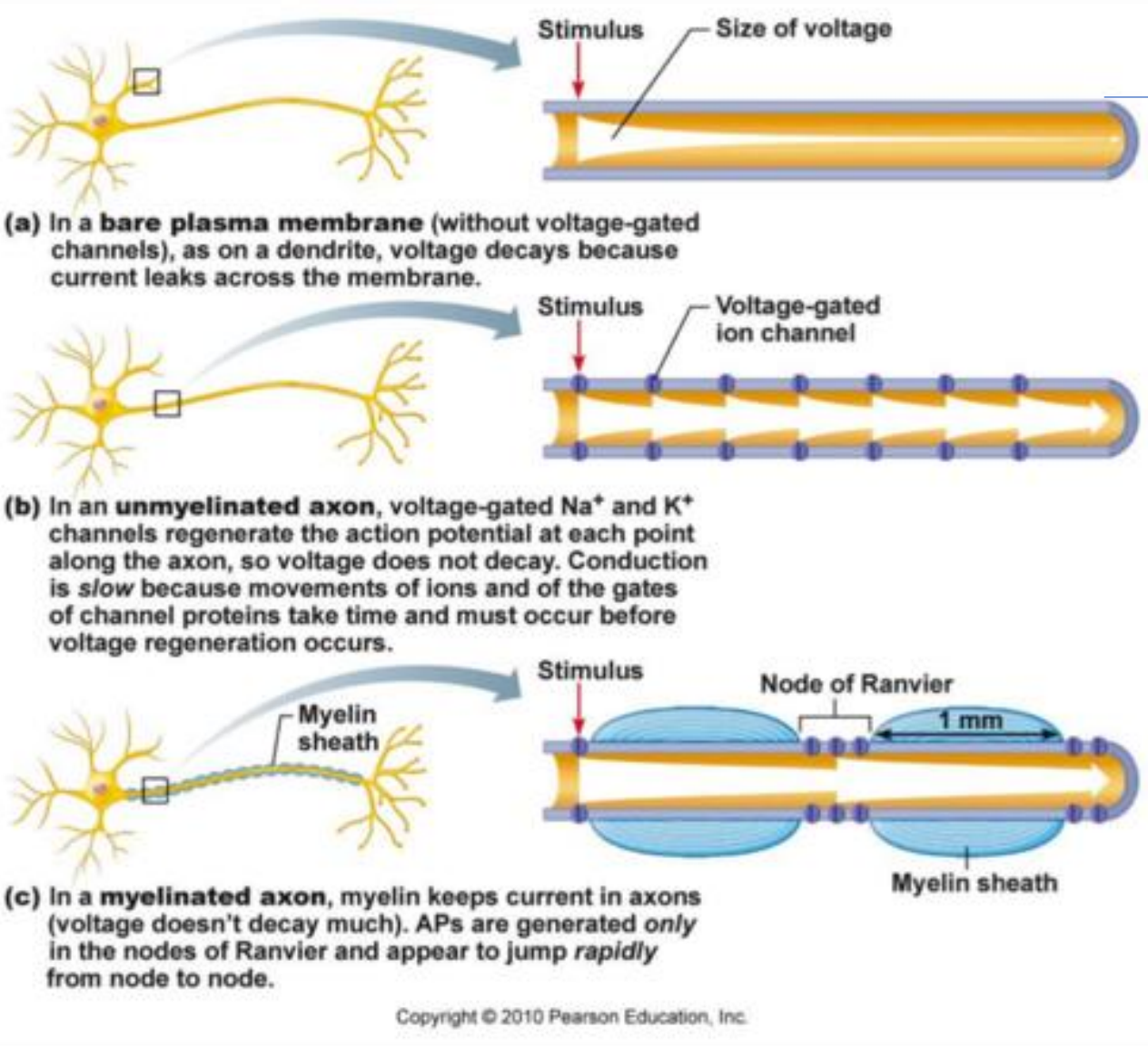




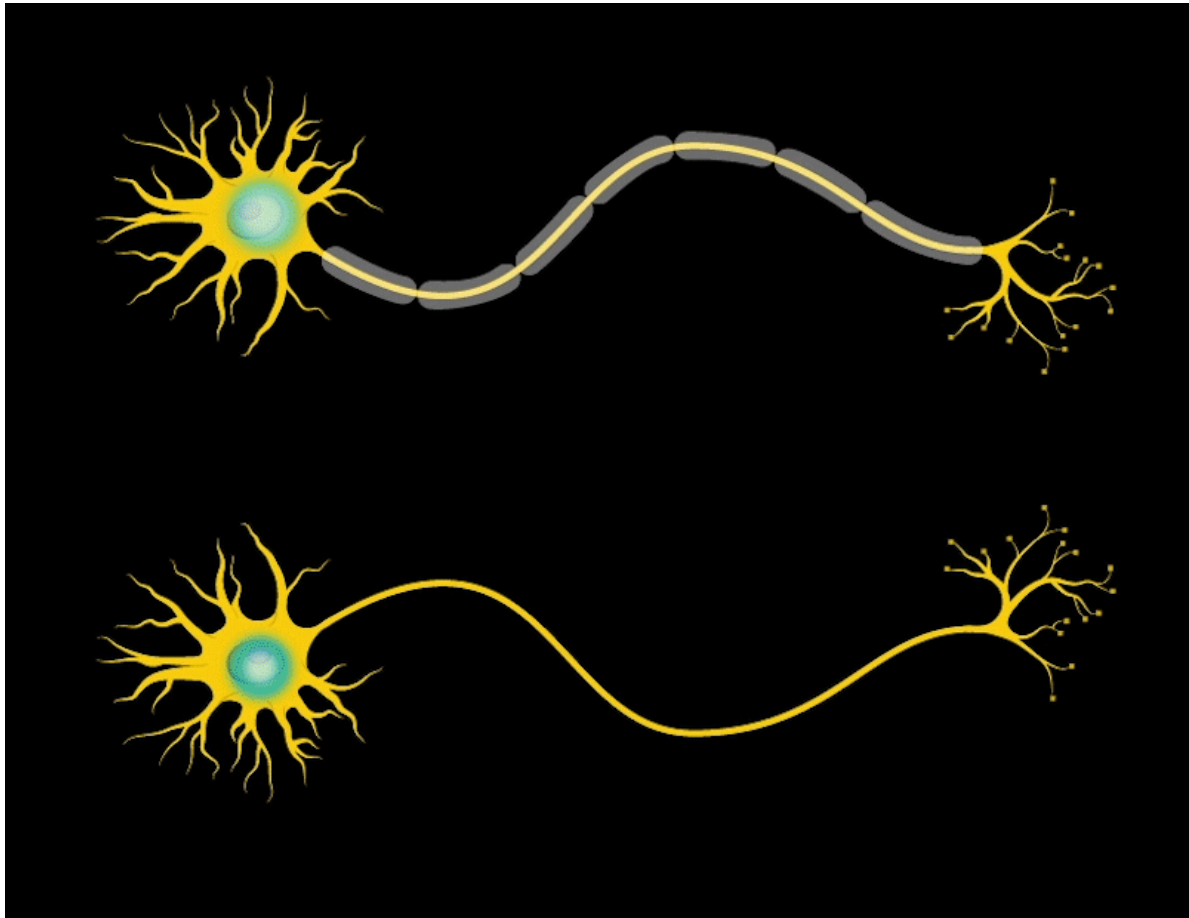


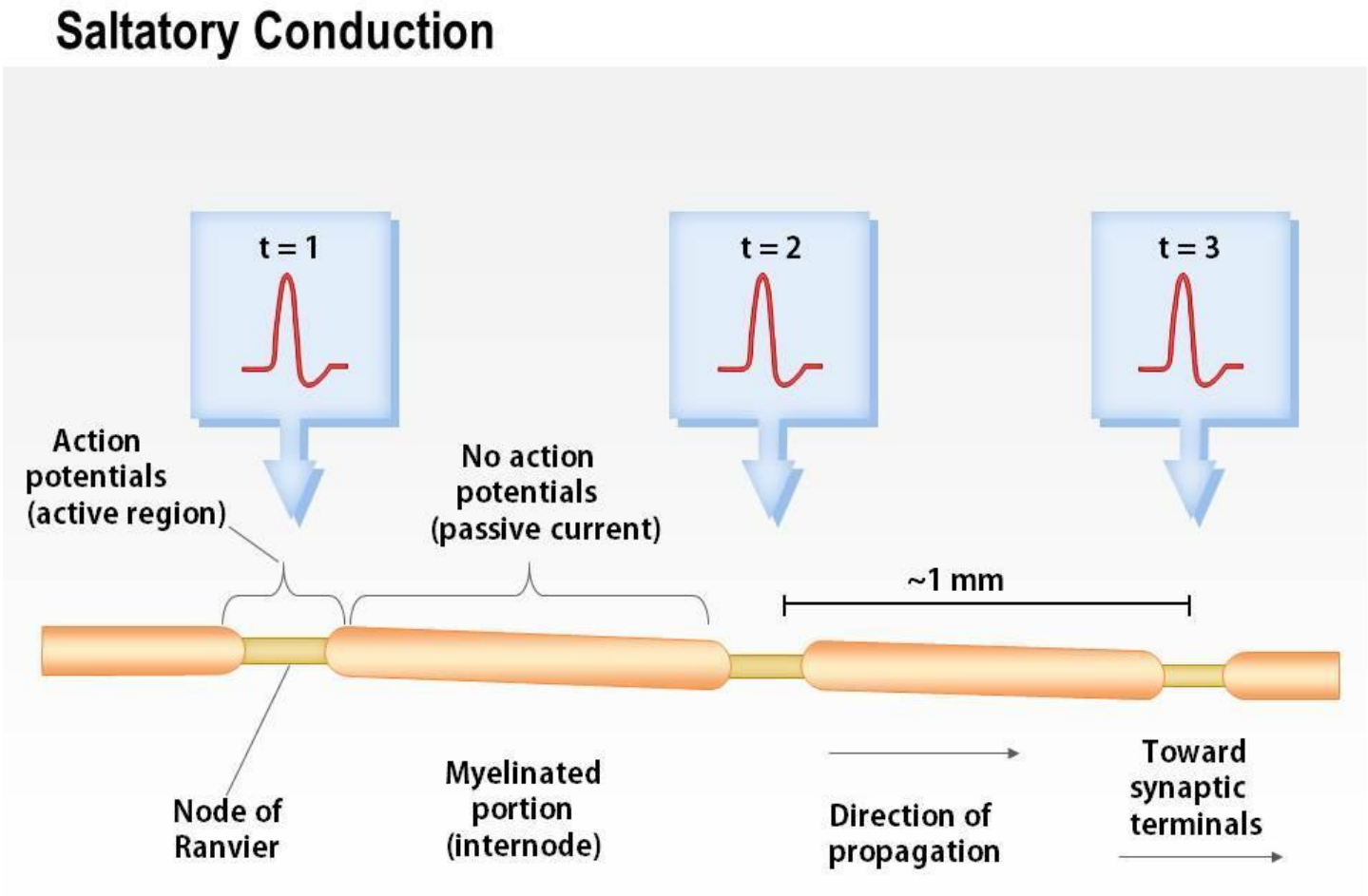
Saltatory conduction:

Conduction of action potentials by myelinated axons. The action potential appears to jump from one node of Ranvier to the next where it strengthens before it propagates further.



In dendrites

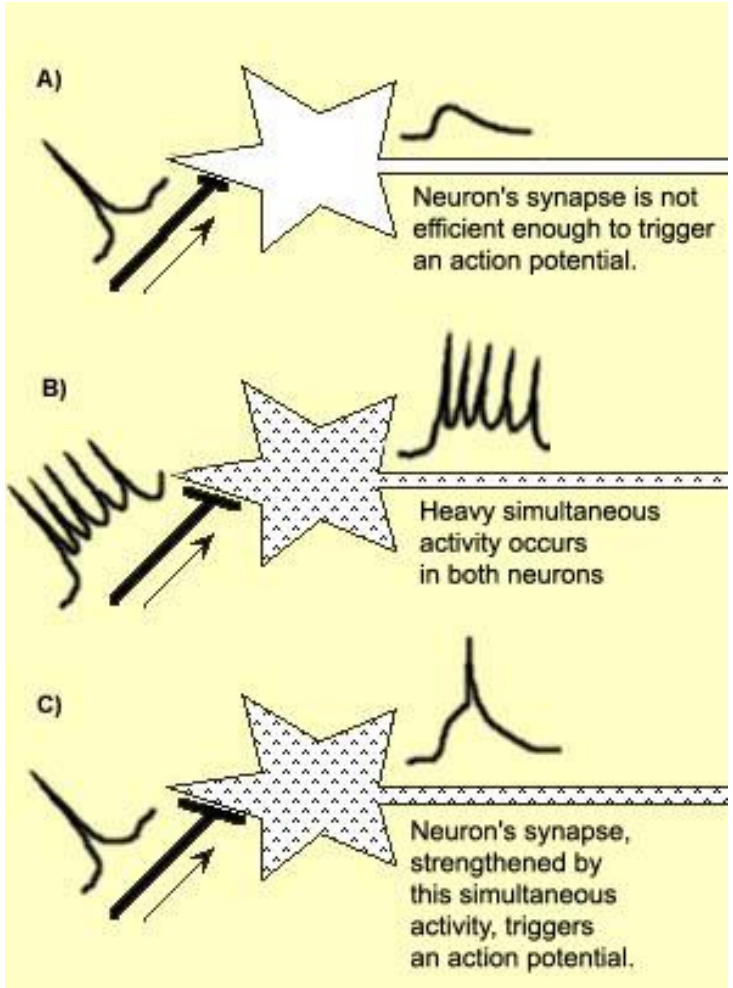




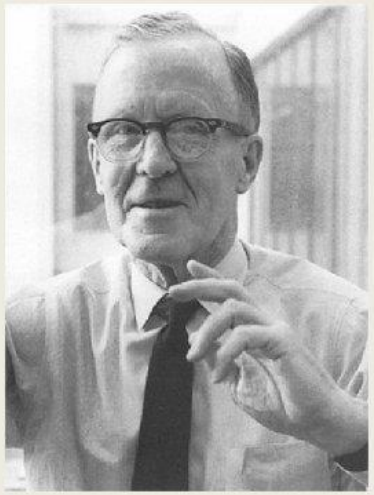
increases the conduction velocity of action potentials from 10 m/s in unmyelinated nerves to 150 m/s in myelinated nerves

- How do we understand that a neuron has undergone change or learning or had fired ?

Hebb's Rule



Donald Hebb



Neurons that fire together, wire together.

1. *Cooperativity*. More than one input must be active at the same time.
2. *Associativity*. Weak inputs are potentiated when co-occurring with stronger inputs.
3. *Specificity*. Only the stimulated synapse shows potentiation.

Long term potentiation (LTP) – strengthening of synapses

upto 3:30min

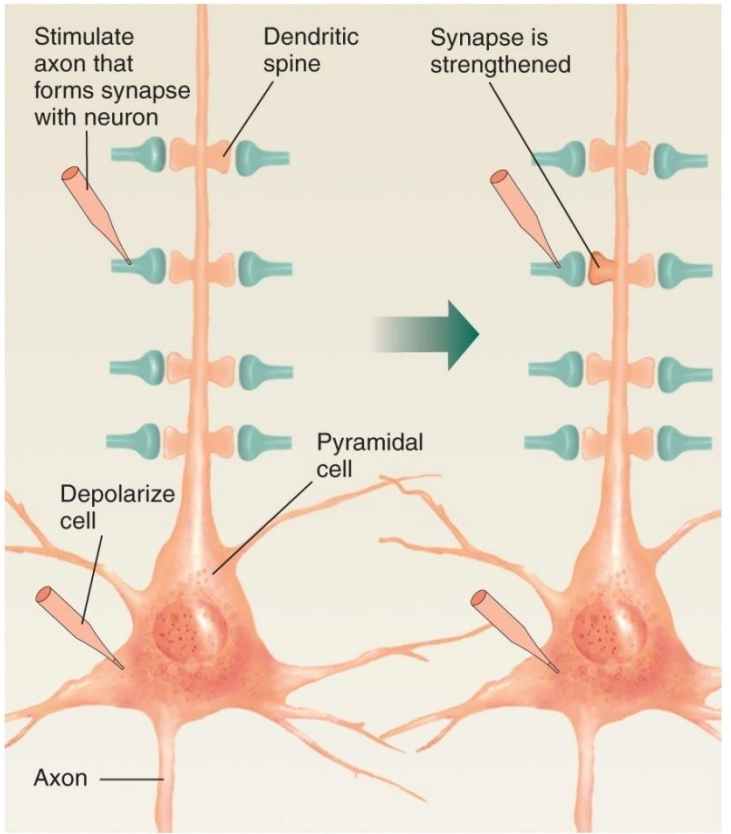
- The postsynaptic membrane needs to stay depolarized when the next incoming burst of neurotransmitters is released and continue to depolarize the postsynaptic cell.

- The pre-synaptic and postsynaptic terminals potentiate and their synapse is strengthened, so even a weak impulse in the pre-synaptic membrane can now activate the post synaptic cell.

Synaptic strengthening occurs when a post synaptic membrane becomes depolarized and before the depolarized fades, the next burst of neurotransmitter released fires another EPSPS in the post synaptic membrane

(while the membrane of the postsynaptic cell is still depolarized from a previous excitation)

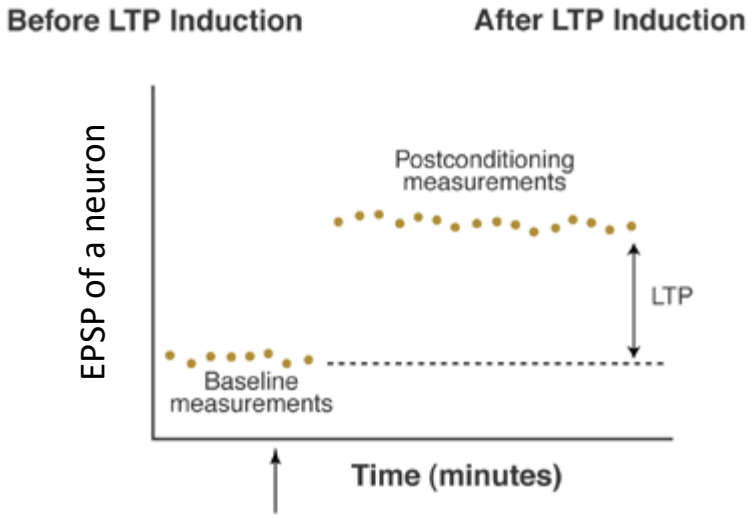
C7B12F07.eps



Synaptic Plasticity

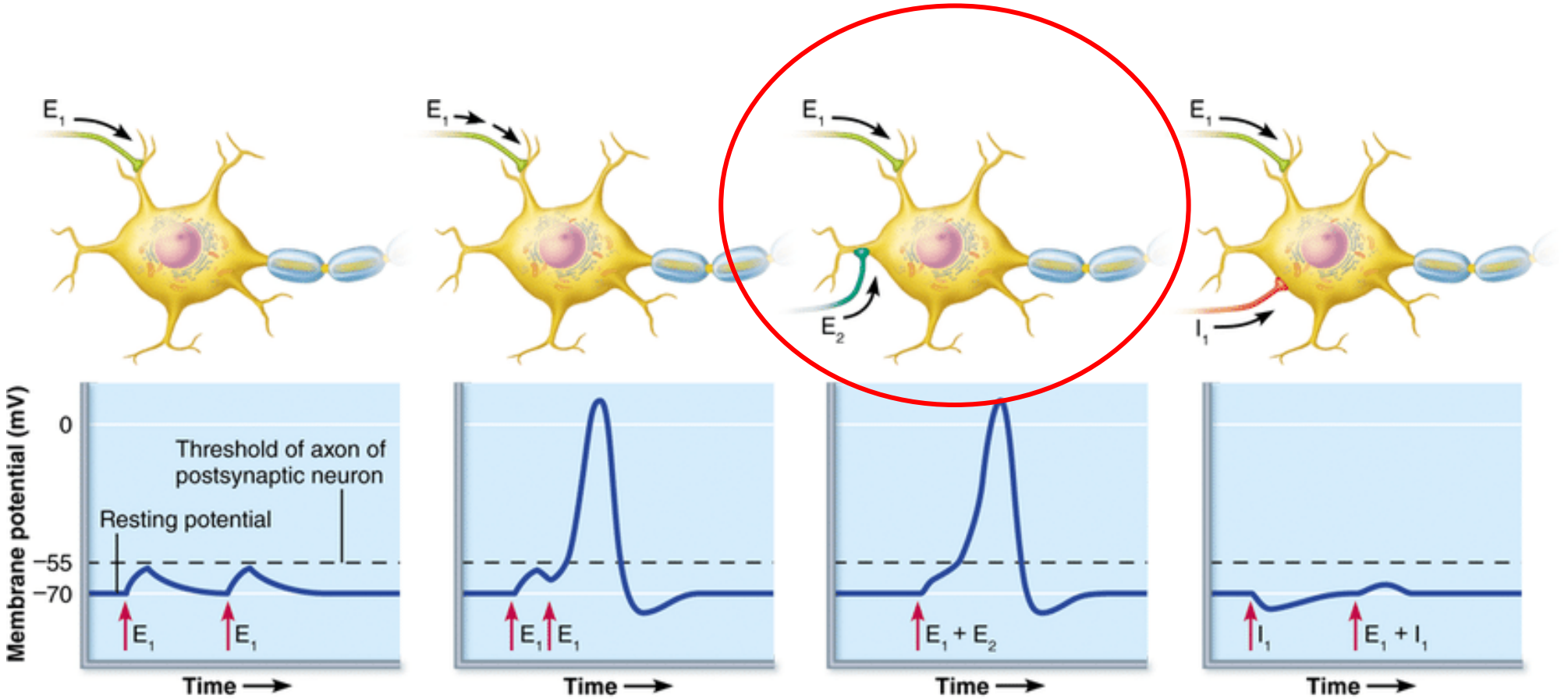
Long-term potentiation (LTP): (potentiate "strengthen")

- EPSPs (excitatory post synaptic potential):
  - High frequency stimulation increases the magnitude of post synaptic excitatory potentials in the postsynaptic neurons. This increase is called as LTP.
  - LTP can be induced by stimulating axons of presynaptic neurons (burst of approx. 100 pulses of electrical stimulation, delivered within a few seconds)
  - EPSPs in the postsynaptic neurons are recorded and if the activity is greater than it was before delivering the pulses, then LTP has occurred.



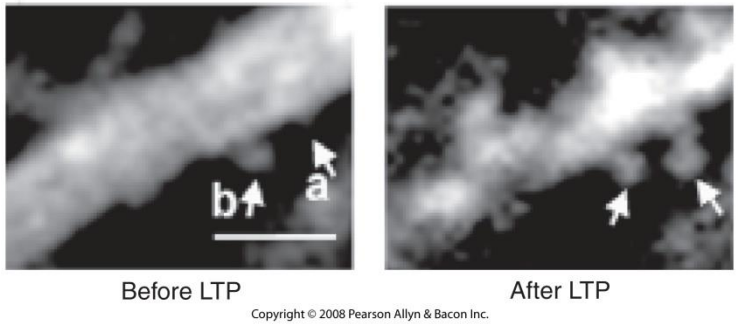
Applying strong electrical stimulation to entorhinal cortex neuron to release more neurotransmitter

Can neurons predict?

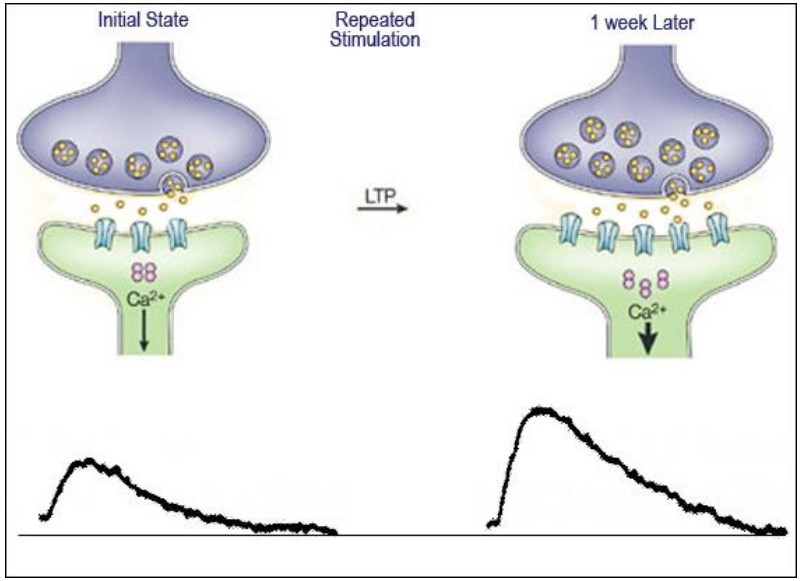


Measuring Synaptic Plasticity

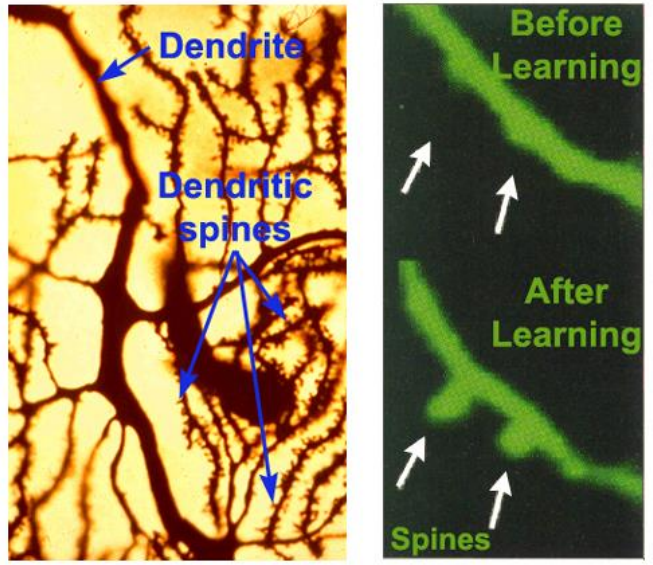
Insertion of receptors

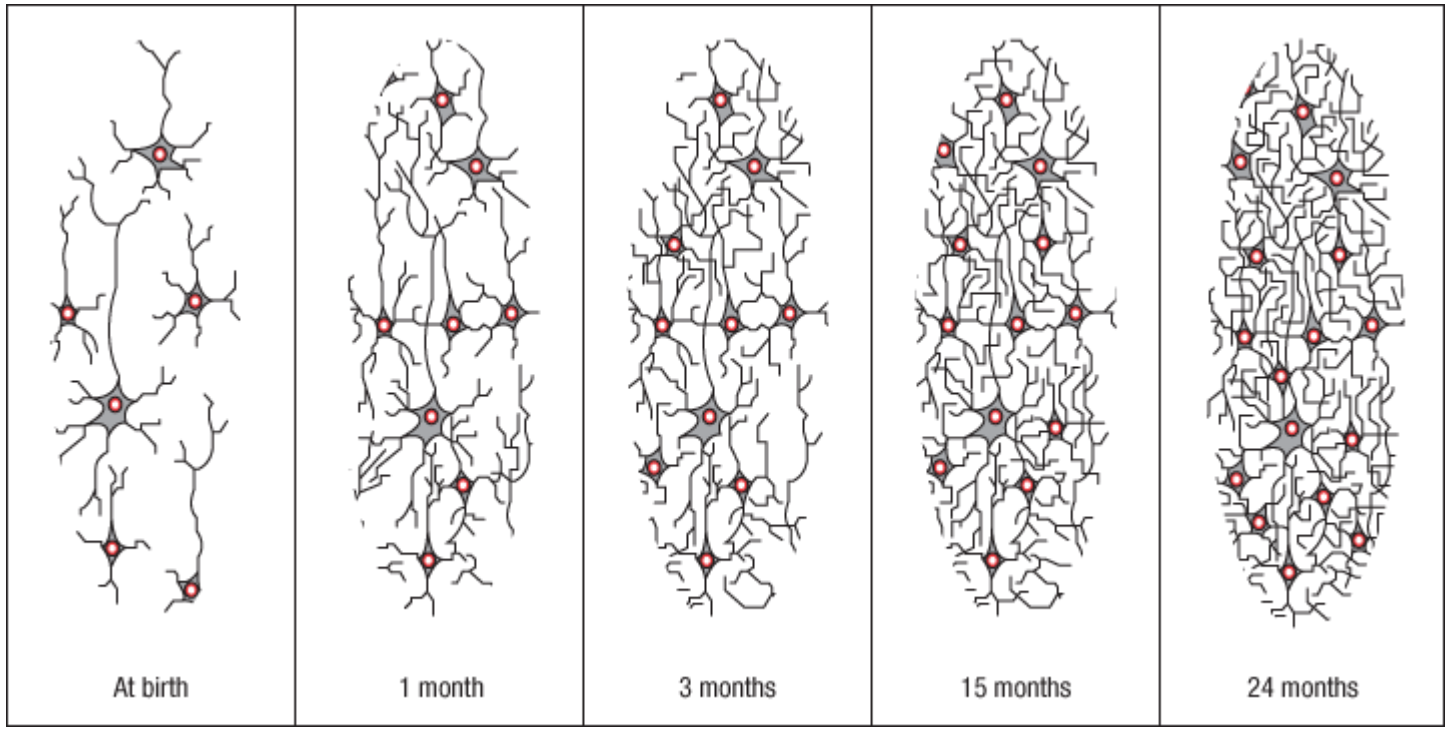


- When synapses get strengthened, what changes occur that increase their strength?
  - More receptors on the postsynaptic membrane
  - More dendrites



Dendritic Spines Increase with Learning





Synaptic pruning & synaptogenosis

The unbearable slowness of being: Why do we live at 10 bits/s?

The unbearable slowness of being: Why do we live at 10 bits/s?: Neuron

https://arxiv.org/pdf/2408.10234

Neurotransmitters and Drugs

TABLE 3A.1

SOME NEUROTRANSMTTERS AND THEIR FUNCTIONS

Neurotransmitter	Function	Examples of Malfunctions
Acetylcholine (ACh)	Enables muscle action, learning, and memory.	With Alzheimer's disease, ACh-producing neurons deteriorate.
Dopamine	Influences movement, learning, attention, and emotion.	Excess dopamine receptor activity is linked to schizophrenia. Starved of dopamine, the brain produces the tremors and decreased mobility of Parkinson's disease.
Serotonin	Affects mood, hunger, sleep, and arousal.	Undersupply linked to depression. Prozac and some other antidepressant drugs raise serotonin levels.
Norepinephrine Or Noradrenaline	Helps control alertness and arousal.	Undersupply can depress mood.
GABA (gamma-aminobutyric acid)	A major inhibitory neurotransmitter.	Undersupply linked to seizures, tremors, and insomnia.
Glutamate	A major excitatory neurotransmitter; involved in memory.	Oversupply can overstimulate brain, producing migraines or seizures (which is why some people avoid MSG, monosodium glutamate, in food). (damage to the brain in stroke)

Since glutamate and GABA are found in very simple animals, it is speculated that these neurotransmitters may have been the first to evolve

Controls muscular movements, arousal, sleep, memory

coordinating movement, attention, learning and memory, prediction, reinforcement

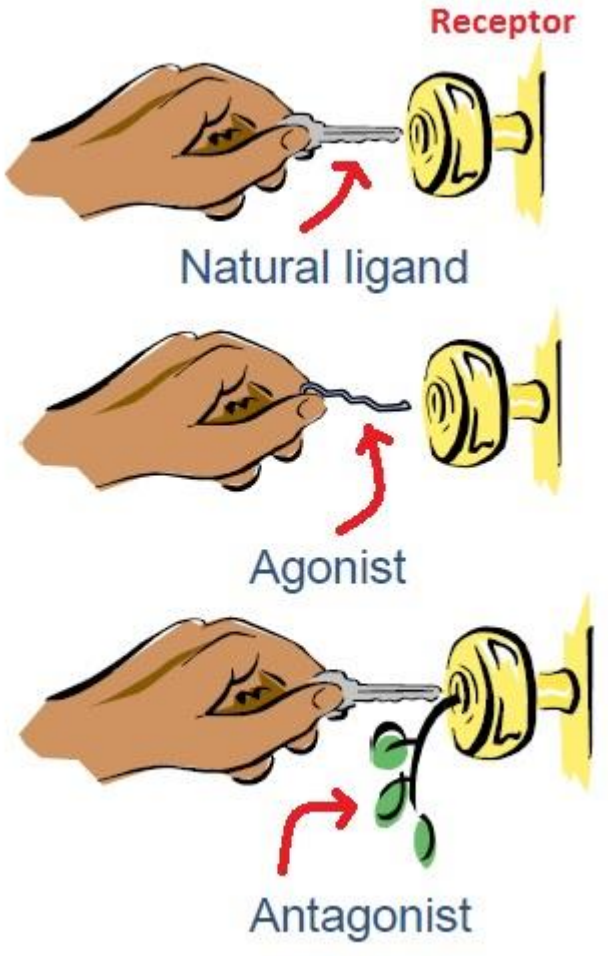
LSD and MDMA (ecstasy) release more serotonin or prevent reuptake of serotonin giving hallucinogenic effects

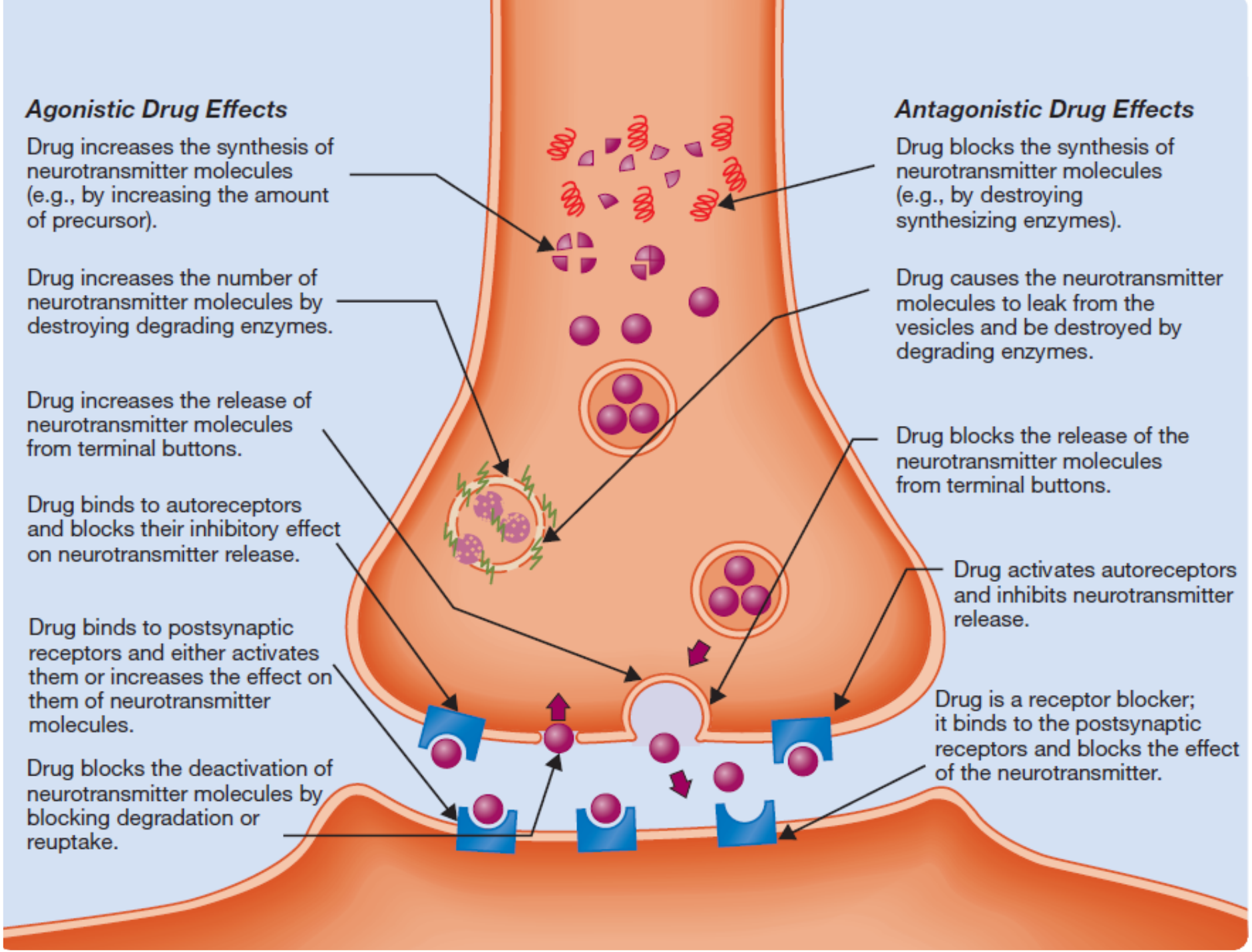
vigilance, attentiveness, high levels lead to euphoria

Found in Sleep meds, alcohol, anxiety meds, sedative

- Agonist:
  - A drug/chemical that facilitates that activates receptors on the postsynaptic cell and causes depolarization
  - Mimics the action is an excitatory neurotransmitter

- Antagonists:
  - A drug/chemical that blocks receptors on the postsynaptic cell and prevent depolarization
  - Prevents excitation of postsynaptic cell





Re-uptake of neurotransmitters

Neuro-peptides

- Released along with neurotransmitters
- Neuromodulators – regulate the sensitivity of presynaptic or postsynaptic receptors to the neurotransmitter
- E.g. endorphins (act like analgesics, diminish the perception of pain)
- E.g. Oxytocin (childbirth, maternal bonding, social interaction)
- Neuropeptides produce a slow but prolonged response at the postsynaptic cell
- Neurotransmitters produce a fast but short-term response in the post-synaptic cell

https://www.researchgate.net/publication/318305704_Difference_Between_Neuropeptides_and_Neurotransmitters

Lipid-based transmission

- Endocannabinoids – lipid (fat) based neurotransmitters - are naturally occurring substance in our body that produces a response similar to Marijuana or cannabis
- they are produced and released as needed and are not stored in synaptic vesicles.
- E.g. Anandamide – binds to endocannabinoid receptors
  - Reduces pain,
  - improves mood – bliss like feeling
- Paracetamol (fever drug) – produces similar effects in body but does not cross the blood-brain-barrier

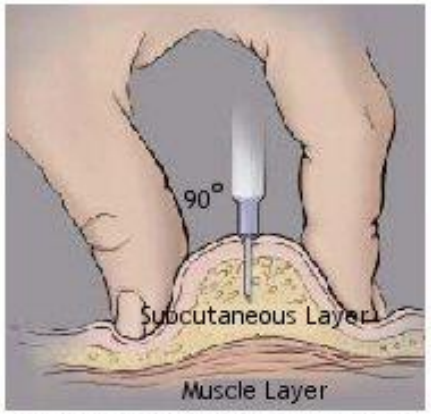
Routes of drug administration

Intravenous injection (IV)

- Injection into vein
- Enters bloodstream within few seconds
- Skill required to administer right dosage

Subcutaneous injection (SC)

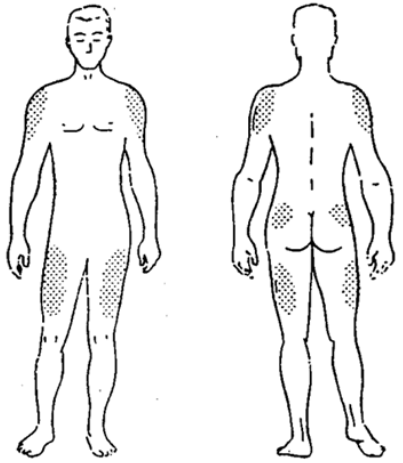
- Injected into the space beneath the skin.
- Useful for small amounts of drugs only (large amounts are painful).
- e.g. COVID vaccine



A subcutaneous injection into the fatty layer of tissue under the skin.

Intramuscular injection (IM) *(most older vaccinations)*

Injected into large muscles (of upper arm, thigh or buttocks)
Absorbed very slowly via capillary action into the bloodstream



Topical administration

Absorbed directly through skin
E.g. ointments, creams or patches

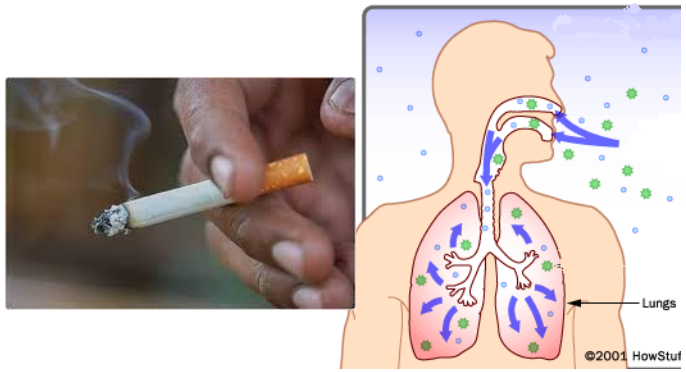
Routes of drug administration

Intranasal administration

- Inhaled by nasal cavity
- Shortest route to reach the brain
- Bypasses Blood-brain-barrier
- E.g. cocaine, anti-allergy

Inhalation

- Absorbed through lungs
- E.g. nicotine and marijuana smoking
- Very rapid entry into the brain since lungs to brain – mixes with blood
- E.g. tobacco, marijuana

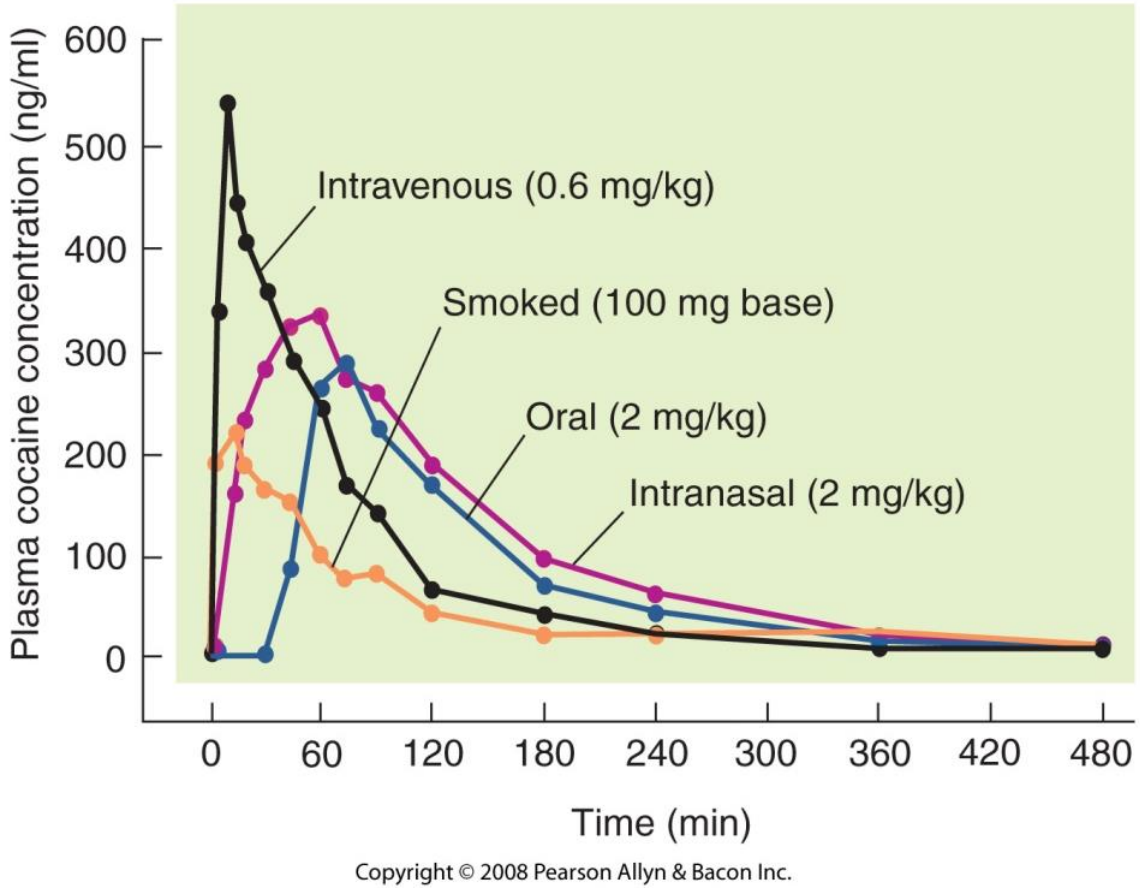


Sublingual administration

- Drugs placed beneath the tongue
- Absorbed by capillaries that supply the mucous membrane in the mouth
- E.g. marijuana

Oral administration

- Not all drugs can be injected or inhaled. A few of them are consumed orally. The effect is slightly slower.
- E.g. medical drugs, alcohol



Cocaine absorption in the blood via different routes of administration

Effects of repeated drug administration

- **Dependance –** a regular usage of the drug is required to function normally

- **Tolerance** – over repeated use, larger amounts of the drug are required for it to be effective
    - It is body's mechanism to compensate for the effects of the drug in order to maintain body homeostasis
    - Reduced no. of receptors
    - Receptors become less sensitive to the drug molecules
    - Decrease receptor binding efficiency

- **Sensitization** – exact opposite of tolerance: same doses of drug produce larger and larger effects.
    - Reduced reuptake of the neurotransmitter by the pre-synaptic cell – the neurotransmitter is available for a longer time – increase in post-synaptic stimulation

- **Withdrawal symptoms** – a person who has developed tolerance for a drug will experience opposite effects of the drug if intake is suddenly stopped.
    - If drugs were creating an excitatory effect , withdrawal leads to less excitation – depressive like symptoms, fatigue, irritability, etc. E.g. dopamine withdrawal
    - If drugs were creating an inhibitory effect (GABAergic), withdrawal leads to more excitation – anxiety or restlessness. E.g. alcohol withdrawal

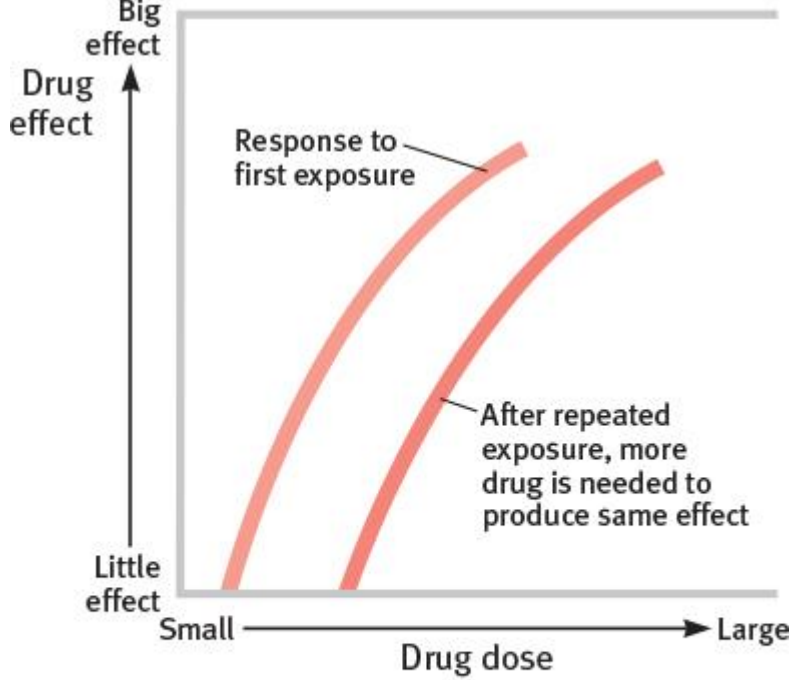
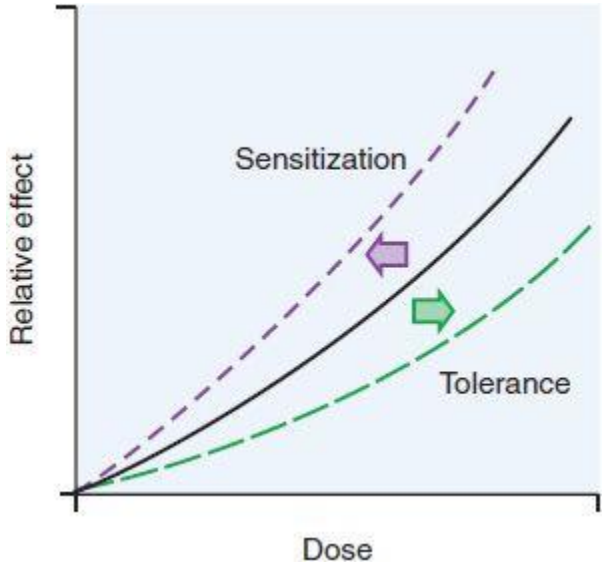


Figure 13.2
Myers/DeWall, *Psychology in Everyday Life*, 4e,
© 2017 Worth Publishers



Your roommate began the semester drinking one cup of coffee in the morning. Now you see that your roommate needs three cups to achieve the same level of alertness. Which effect of repeated administration has occurred?

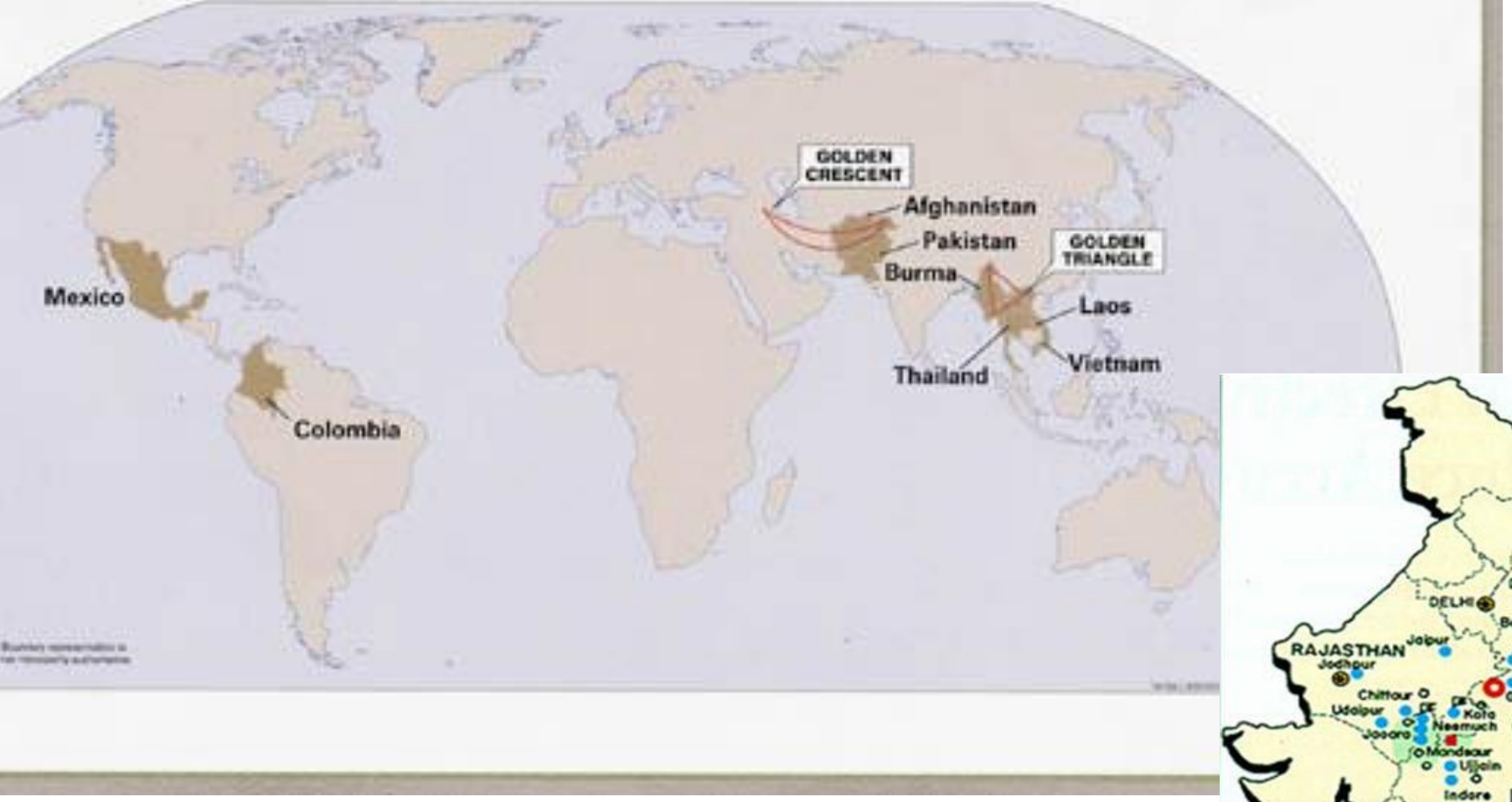
Tolerance

Withdrawal

Sensitization

Opiates

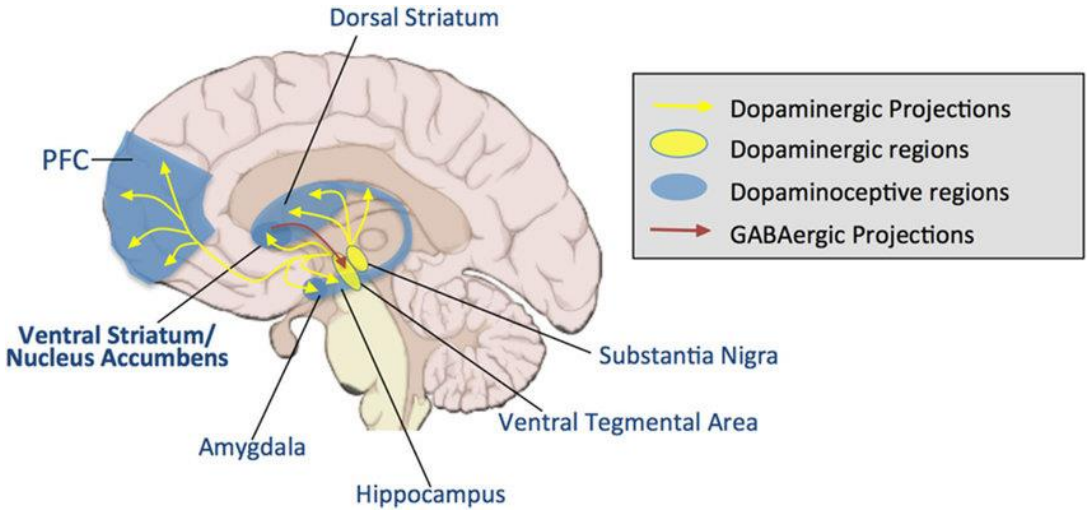
- Opium (dried sap) → morphine → heroin (higher ability to cross the blood brain barrier → more addictive)
- Poppy seeds (khas khas) contain negligible opium (consuming too many poppy seeds makes you drowsy)
- Heroin is snorted or taken intravenously, morphine - intravenous
- Opium – typically smoked



Opium has been consumed for thousands of years

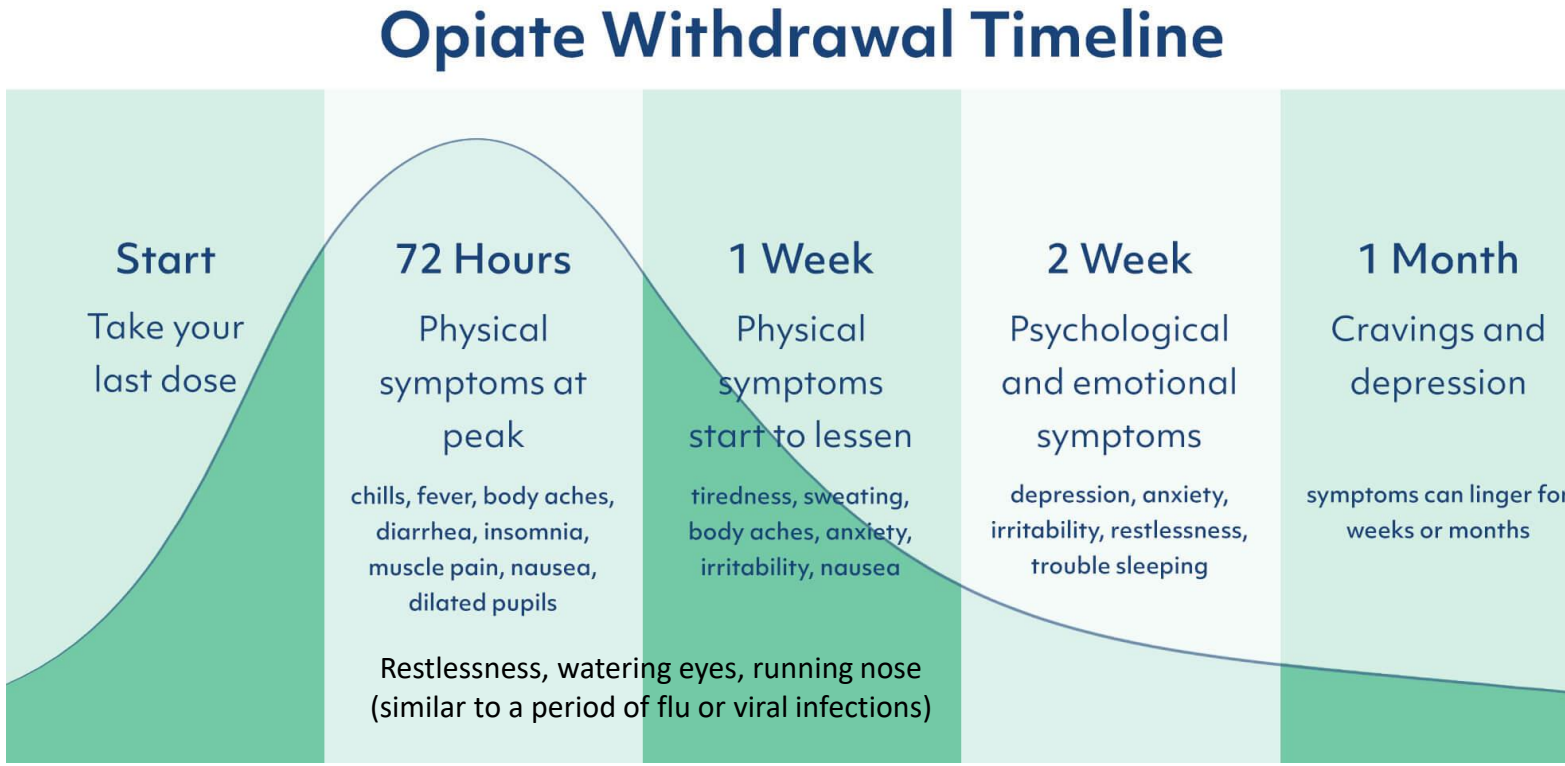
In India legal cultivation is permitted to farmers only in **Madhya Pradesh, Rajasthan and Uttar Pradesh**.

Opiates



- "heroin rush" is a wave intense pleasure that evolves into a state of serene, drowsy euphoria.
  - Morphine/heroin – increased dopamine release
  - At higher doses – serotonin is released (Wei et al., 2018)
  - Temporary decrease in norepinephrine levels
- Positives –
  - pain killer/analgesic – blocks endorphin/opioid receptors that carry pain signals from the PNS to the brain.
- Negative – high risk of addiction (tolerance → higher doses)
  - reinforce reward circuitry (any behaviour that causes pleasure is reinforced and repeated for more pleasure – drug seeking behaviour)
  - The addiction occurs due to activation of receptors in the NA and VTA
- Other effects – hypothermia, sedation

Opiates – withdrawal symptoms



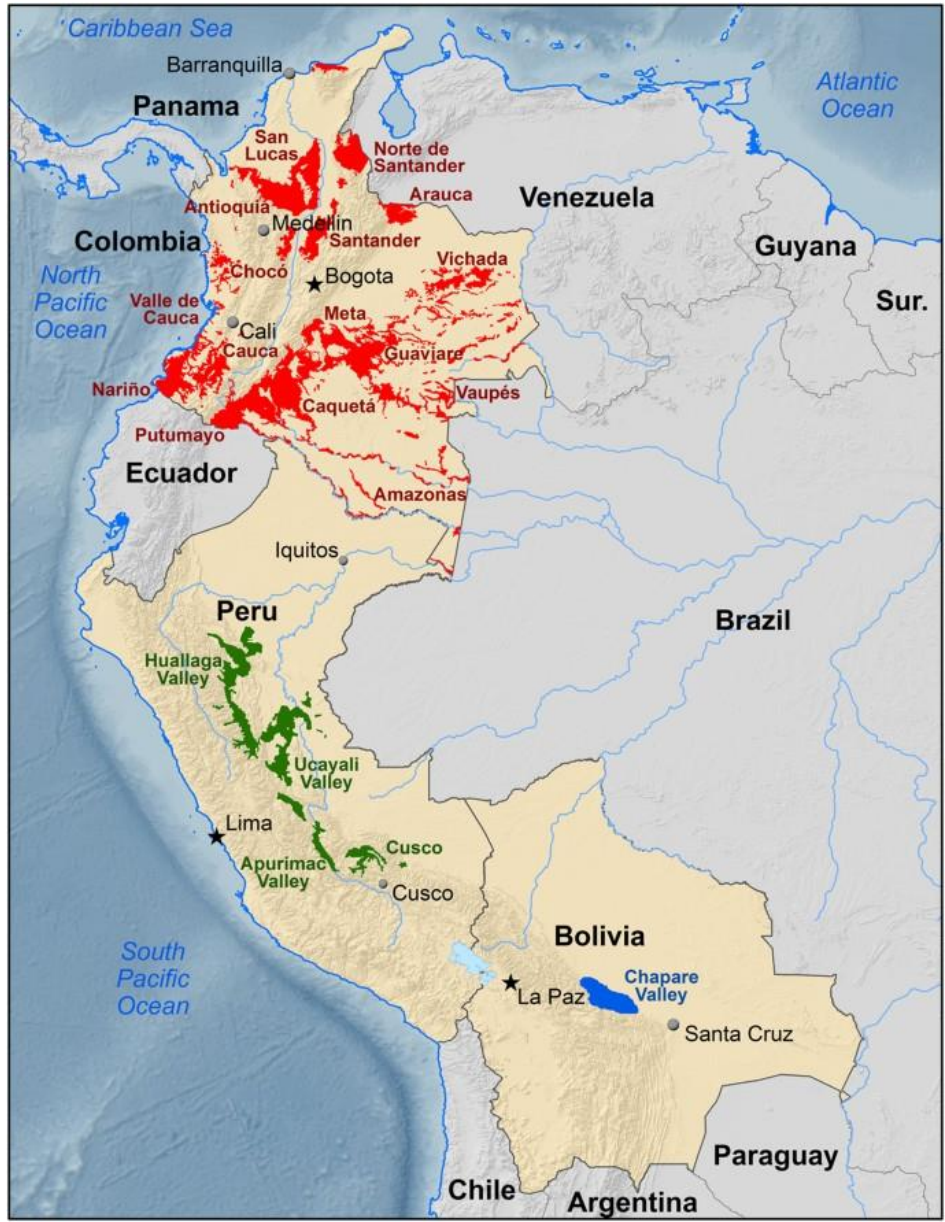
- For gradual withdrawal – drugs that bind to same receptors as opium – produce less pleasure – dose is gradually lowered

- Withdrawal is not as severe or difficult as alcohol (opium dependence can be over come gradually)

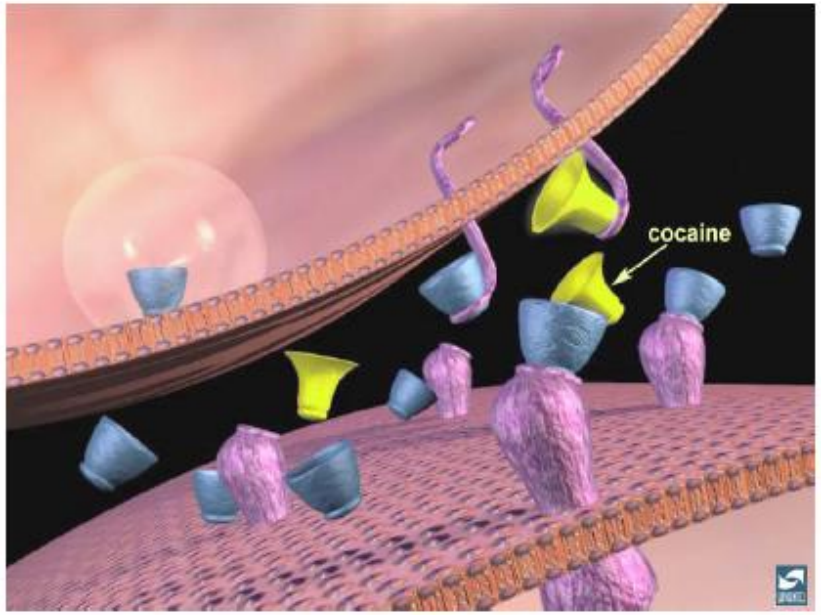
Cocaine

- Cocaine is prepared from the leaves of the coca shrub
- Grows primarily in western South America
- In olden times, coca paste was made directly from the leaves and eaten.
- Current form –highly processed - white powder – cocaine hydrochloride
- Impure residue after processing – Crack – cheap but potent
- Cocaine is typically snorted, smoked or taken intravenously

- First coca-cola recipe had cocaine in small amounts
- Now it has caffeine



Dopamine binding to receptors and uptake pumps in the nucleus accumbens: the action of cocaine



Cocaine binds to the uptake pumps and prevents them from transporting dopamine back into the neuron terminal. So more dopamine builds up in the synaptic space and causes a net increase in dopamine neurotransmission. This is the same general outcome as morphine, but morphine increased dopamine release from the terminal to produce more dopamine in the synaptic space.
Cocaine results in as much as a 10-fold increase in dopamine.

Cocaine blocks dopamine reuptake

Cocaine

- Cocaine blocks the reuptake of dopamine after it is released by the terminal buttons.
- Effects of cocaine –
  - euphoric, active and alert, talkative, friendly, feeling self-confident and powerful
  - have less than their usual desire for food and sleep

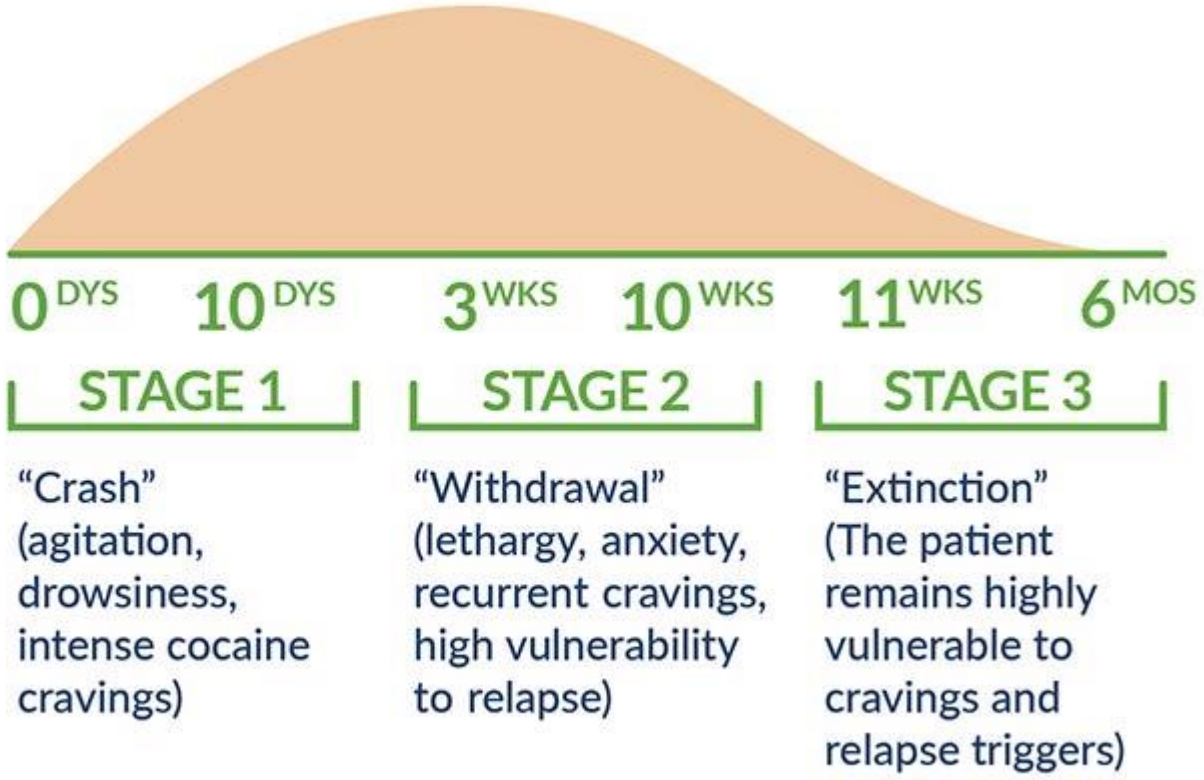
- Addictive – behaviour resulting in pleasure in reinforced (drug seeking)
  - cocaine sprees - binges in which extremely high levels of intake are maintained for periods of a day or two.
  - binging makes users increasingly tolerant to cocaine
  - psychotic behavior: hallucinations, delusions of persecution, mood disturbances, and repetitive behaviors.
  - These symptoms so closely resemble positive symptoms of schizophrenia that even a trained mental health professional cannot distinguish them unless he or she knows about the person's history of substance abuse.
  - This confirms that excessive dopaminergic activity causes these symptoms in schizophrenia as well

- Cocaine causes long-lasting suppression of both dopamine and serotonin neurons (Wei et al 2018)

  - Transgenerational epigenetic effects of cocaine consumption.
    - increased propensity for cocaine self-administration, the offspring of male rats given cocaine are more likely to display increases in anxiety- and depression-like behaviors

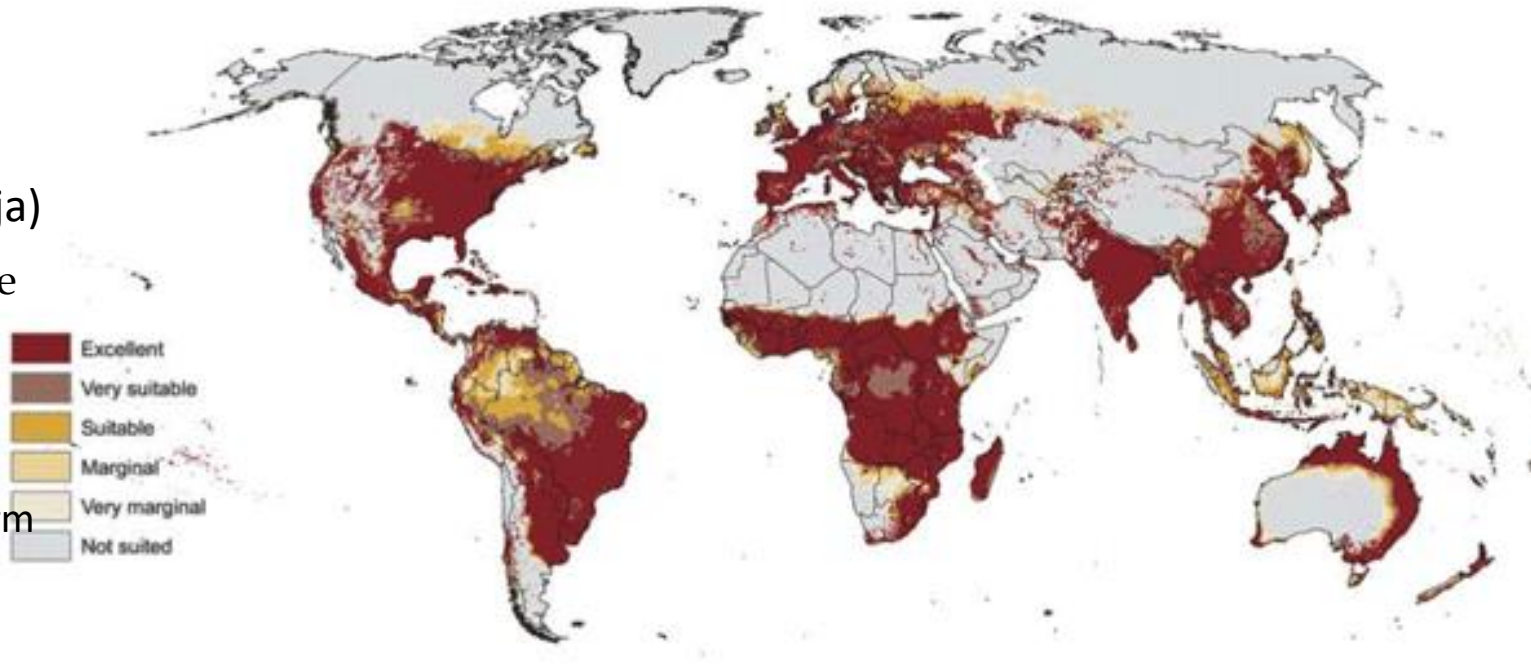
Cocaine withdrawal

- The withdrawal effects triggered by abrupt termination of a cocaine spree are relatively mild.
- Common cocaine withdrawal symptoms include a negative mood swing and insomnia.



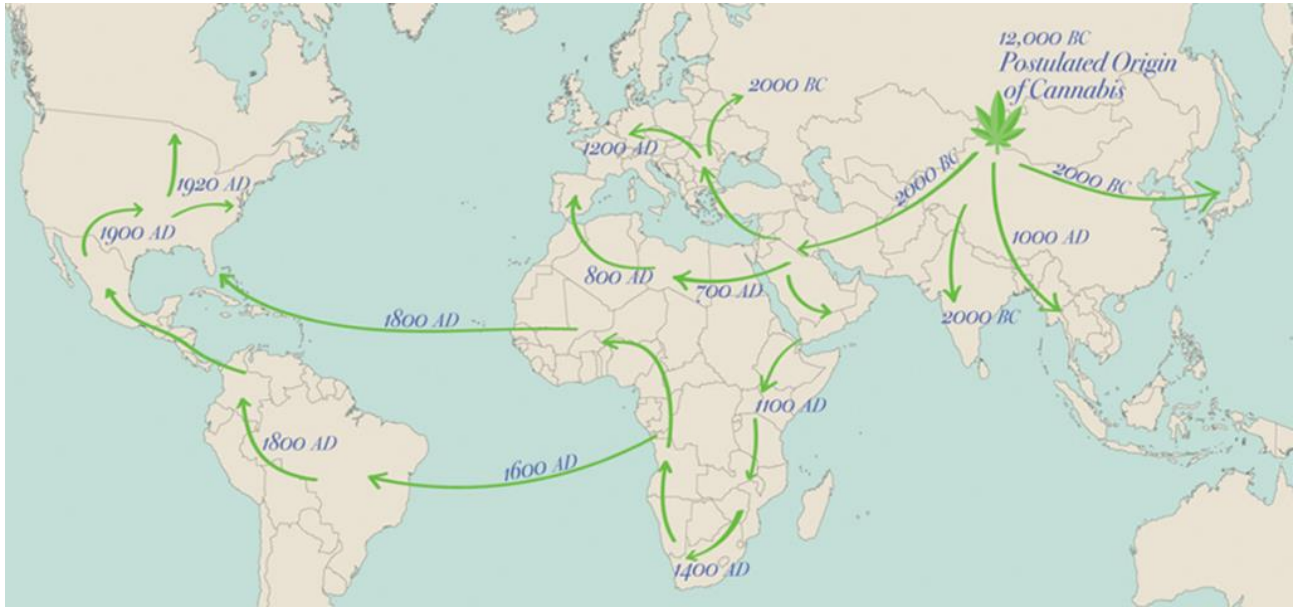
Cannabis/Marijuana

- Dried flower buds of cannabis → marijuana (ganja)
- a sticky resin covering the leaves and flowers of the plant → extracted and dried → a dark corklike material '**hashish' (charas)**
- Weed, grass, pot
- Bhang- wet grinding of the leaves of the plant – this form has been consumed in India for more than 2000 years
- Mostly smoked



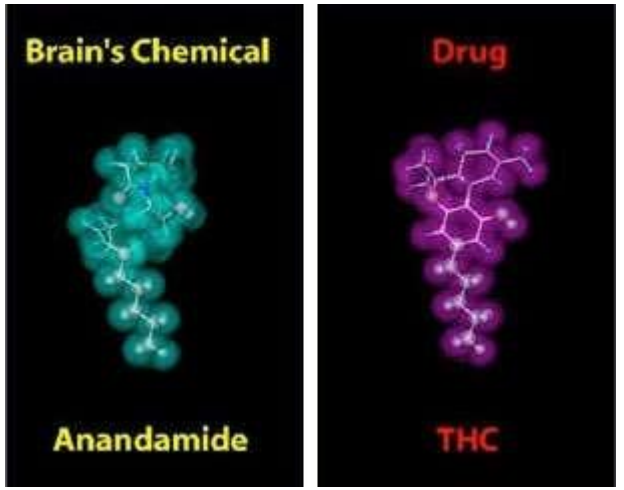
Written records of cannabis use go back 6,000 years in China, where its stems were used to make rope, its seeds were used as a grain, and its leaves and flowers were used for their psychoactive and medicinal effects

Cannabis Sativa



Marijuana effects

THC's chemical structure is similar to the brain chemical anandamide. Similarity in structure allows drugs to be recognized by the body and to alter normal brain communication.

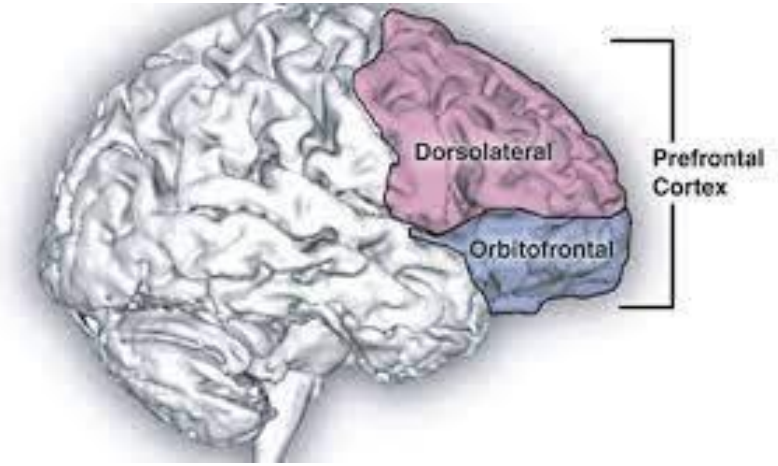


- THC (delta-9-tetrahydrocannabinol) is the main ingredient
- THC binds to the same receptors as anandaminde (endocannabinoid – naturally produce)
- Stimulates neurons in the reward system - high levels of dopamine released – addictive - down-regulation of cannabinoid receptors
- transgenerational epigenetic effects - increased vulnerability to stress-induced anxiety.

small doses of marijuana produce subtle effects -

- pleasant euphoria
- sense of relaxation.
- Other effects, which may vary dramatically among different people
  - include heightened sensory perception (e.g., brighter colours),
  - laughter
  - altered perception of time
  - increased appetite.

At high doses of marijuana



- Learning and memory is impaired (hippocampus - Chronic THC exposure may permanently damage hippocampal neurons.

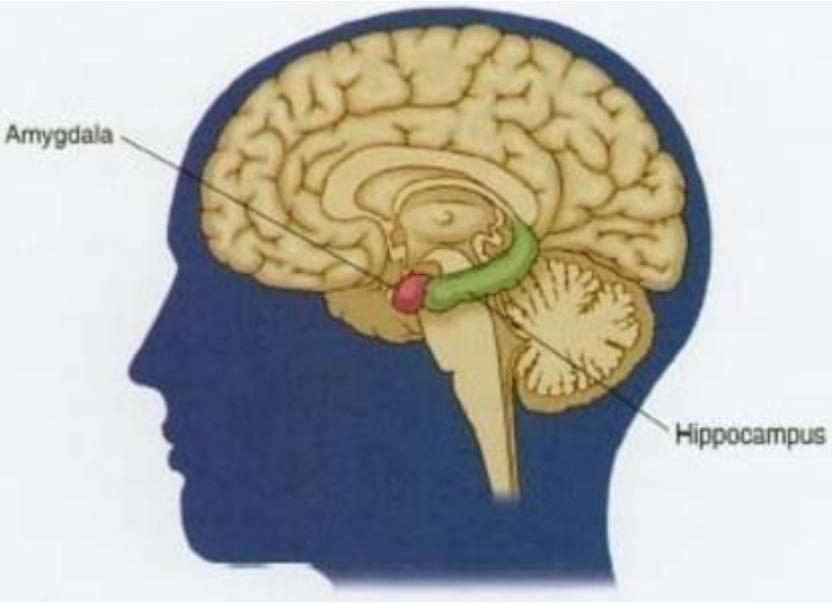
- impaired thinking - reduced ability to perform complicated tasks (orbitofrontal cortex)

- Speech becomes slurred
- Meaningful conversation becomes difficult.

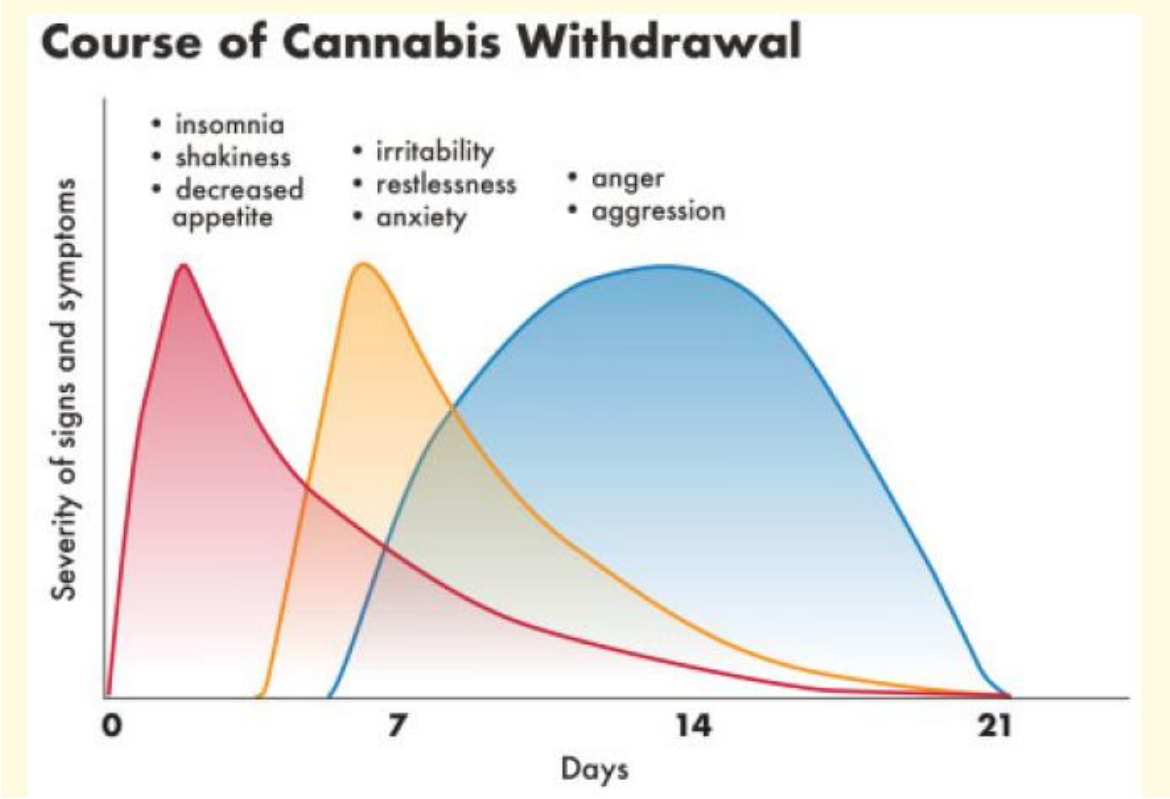
- Disruption of balance, posture, coordination, and reaction time, motor impairment (basal ganglia & cerebellum)

- A sense of unreality, emotional intensification, sensory distortion, feelings of paranoia,
- Acute psychosis, which includes hallucinations, delusions, and a loss of the sense of personal identity.

- Higher risk for multiple sclerosis (loss of myelin sheath)



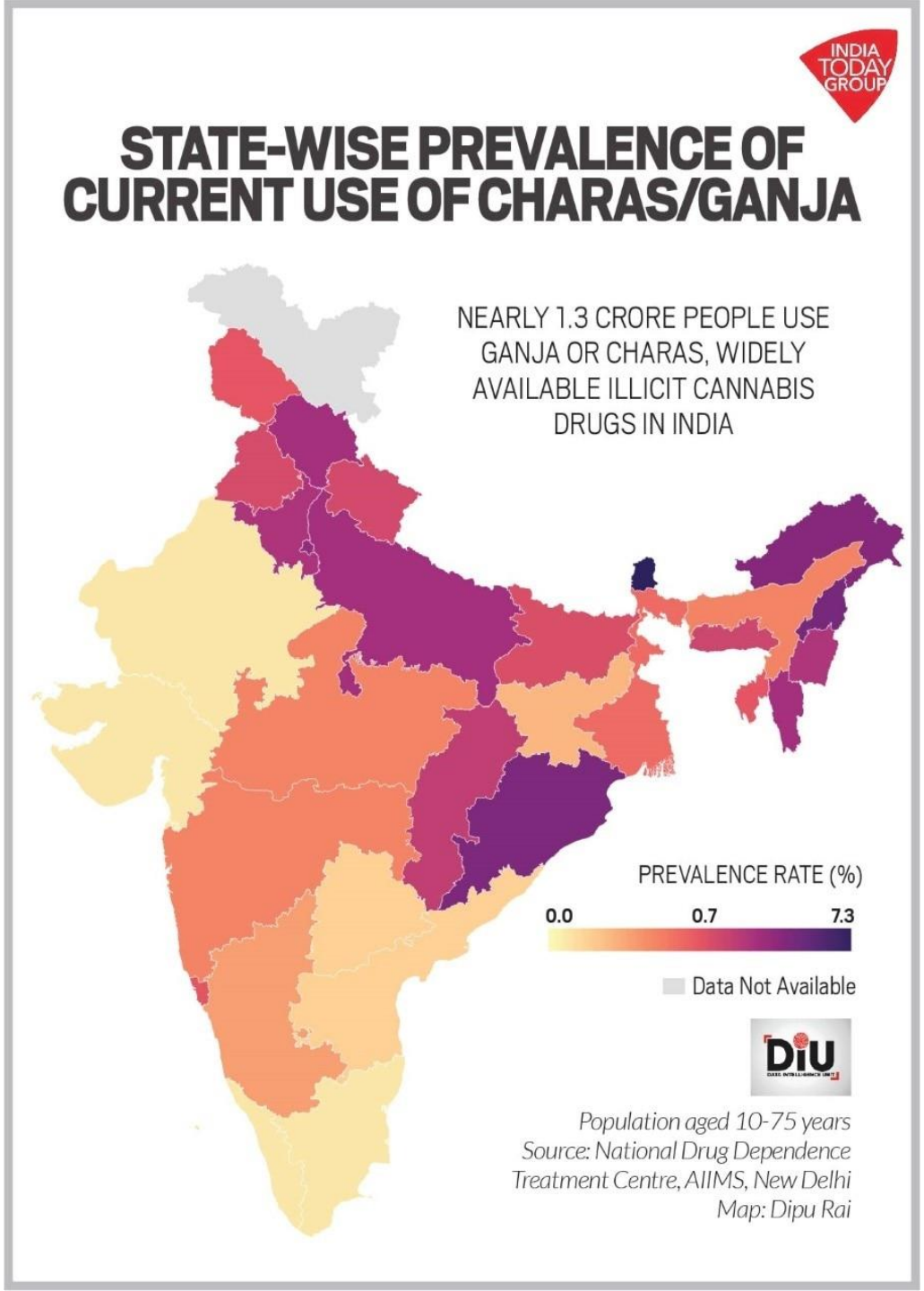
Cannabis Withdrawal

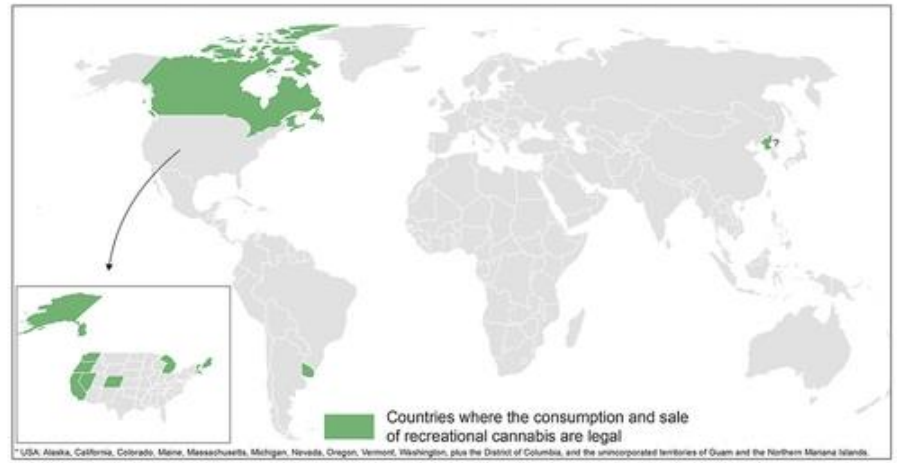


Cannabis in India

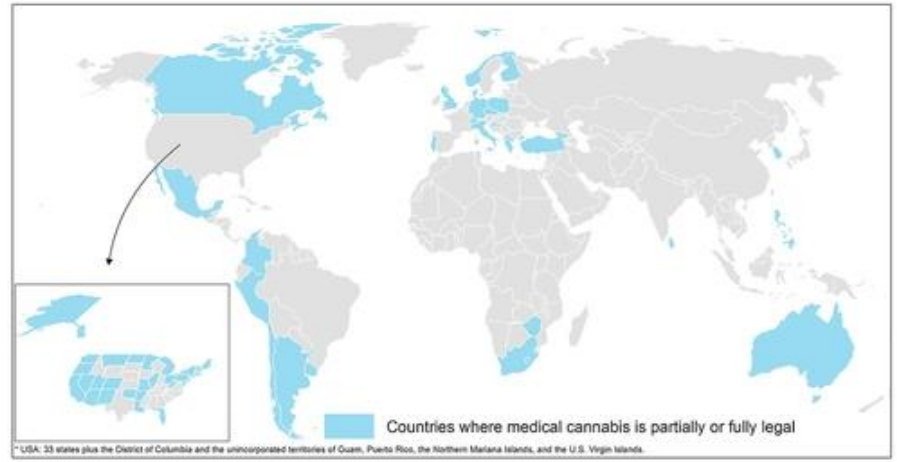
- Narcotic Drugs and Psychotropic Substances Act (1985) recognizes cannabis as a source of biomass, fiber, and high-value oil – can be cultivated for industrial use
  - GOI allows cultivation of cannabis with low THC content.
  - Bhang is not illegal - wet grinding of the leaves of the plant

- Bhang is banned in states of Maharashtra, Karnataka, Assam





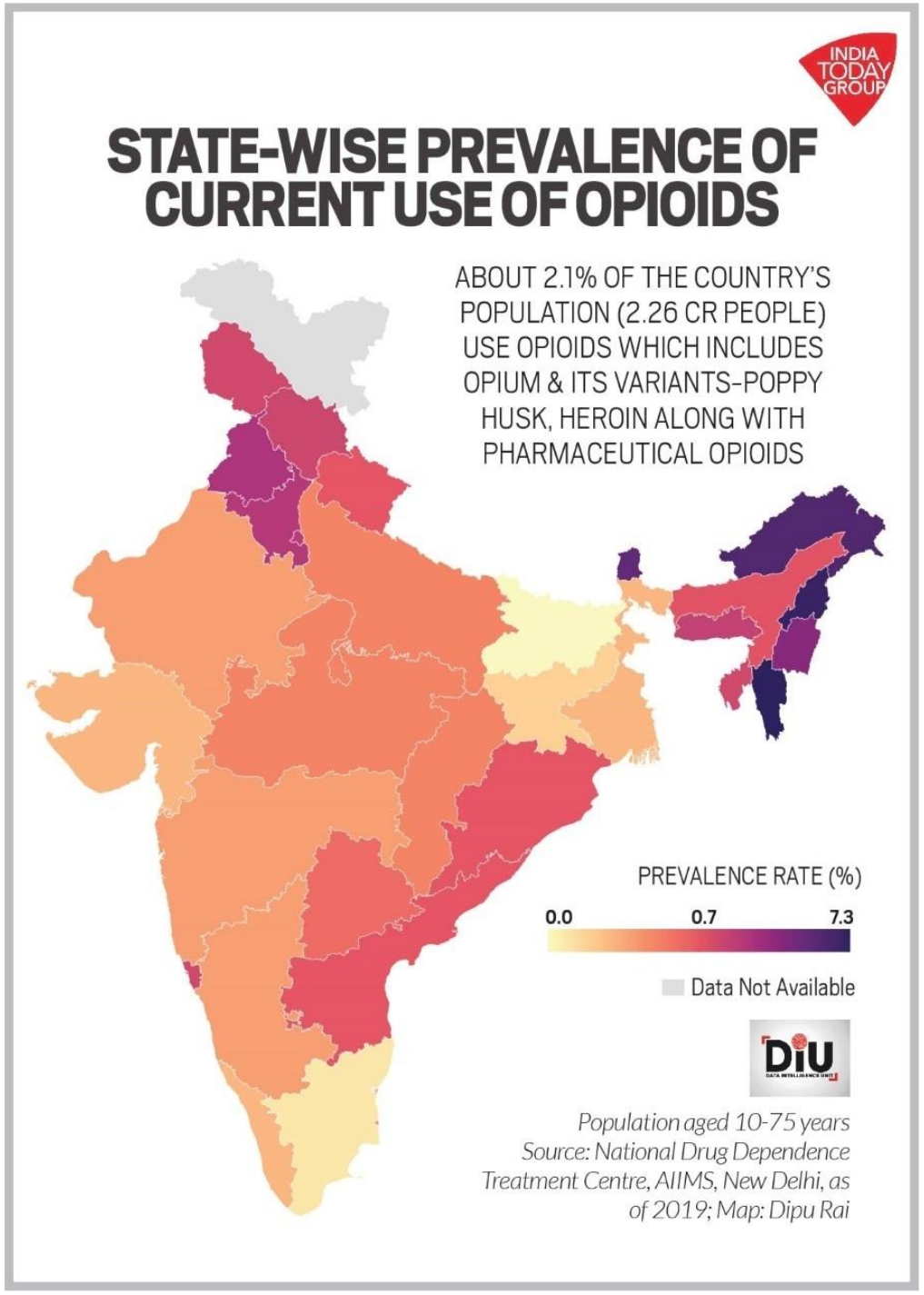
* USA: Alaska, California, Colorado, Maine, Massachusetts, Michigan, Nevada, Oregon, Vermont, Washington, plus the District of Columbia, and the unincorporated territories of Guam and the Northern Mariana Islands.



* USA: 33 states plus the District of Columbia and the unincorporated territories of Guam, Puerto Rico, the Northern Mariana Islands, and the U.S. Virgin Islands.

Sources: Research by Pierre-Arnaud Chouvy, January 2019

- All drugs discussed so far are illegal for consumption in India
  - Opium
  - Cocaine – doesn't grow in India
  - Cannabis



Why are SSRIs legal and MDMA/ecstasy illegal ?

3,4-Methylenedioxymethamphetamine (MDMA)

SEROTONIN is a chemical in the brain which affects a number of things, including mood. People suffering from clinical depression have lower than average levels of serotonin in their brains. Prozac gradually restores serotonin to its proper level, then maintains that level. It has no effect on mood in people who are not suffering from depression. Ecstasy, in contrast, releases a sudden excess or rush of serotonin which produces an elevated mood for several hours afterwards. In fact, the massive release of serotonin may leave nerve cells depleted and cause irreversible brain damage. Mood can be thought of as a light bulb and serotonin as the voltage which keeps it glowing. Too little voltage and it goes dim (depression). Prozac restores the voltage and brightness to normal. Ecstasy produces a blinding flash as the bulb burns out. Finally, prozac is a strictly controlled medicine, whereas the manufacture and sale of Ecstasy is unregulated and dangerous.

Dr A Simpson (Medical Director, Eli Lilly), Basingstoke, Hants
(Part of the group that manufactures Prozac)

SSRIs prevent "used" serotonin from being reabsorbed, allowing it to hang around the synapse longer than normal with the goal of correcting a chemical imbalance.

MDMA, on the other hand, actually causes your brain to pump out "extra" serotonin, producing the lovely feelings of "ecstasy" MDMA is known for.

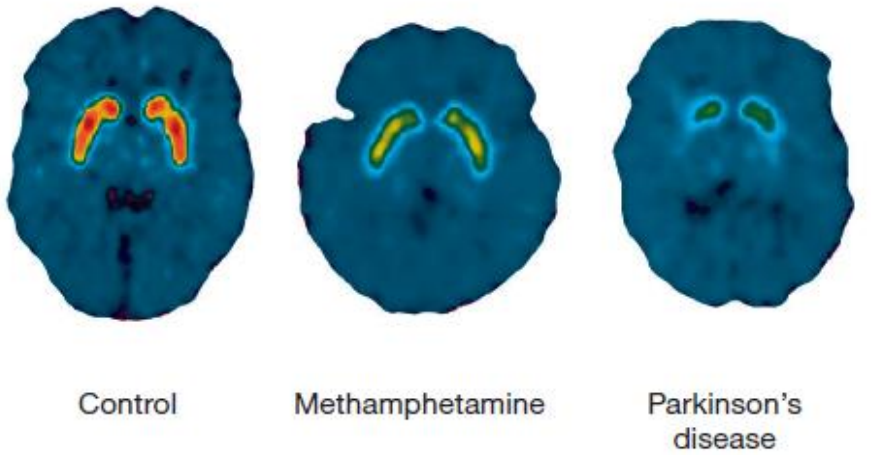
https://www.theguardian.com/notesandqueries/query/0,,-2140,00.html

- Amphetamine/methamphetamine (meth) – synthetic drug

- inhibits the reuptake of dopamine and also stimulates the release of dopamine from terminal buttons.

- Meth can also damage terminals of serotonergic axons and trigger death of neurons in the cerebral cortex, striatum, and hippocampus

- Increased risk of Parkinson's disease



Legal stimulants

- Alcohol
- Tobacco (nicotine)
- Caffeine

Alcohol

- At low doses → mild euphoria, reduces anxiety
- At moderate doses → cognitive, perceptual, verbal, and motor impairment, loss of cognitive control, socially unacceptable behaviour
- At higher doses → stupor , unconsciousness, and if blood levels reach 0.5%, there is a risk of death

- Alcohol produces dependence and tolerance
- Addiction – to alleviate body pain and mental stress

- Withdrawal symptoms can be seen even after single bout of drinking
  - Nausea, headache, vomiting, tremors → hangover

- Withdrawal symptoms of moderate to heavy drinkers are severe

BAC	Predictable Effects
.02% to .04%	Lightheaded – Relaxation, sensation of warmth, "high," minor impairment of judgment
.05% to .07%	Buzzed– Relaxation, euphoria, lower inhibitions, minor impairment of reasoning and memory, exaggerated emotions (good and bad)
.08% to .10%	Legally Impaired – Euphoria, fatigue, impairment in balance, speech, vision, reaction time and hearing, judgment and self-control are impaired
.11% to .15%	Drunk – "High" reduced and depressive effects (anxiety, depression or unease) more pronounced, gross motor impairment, judgment and perception severely impaired
.16% to .19%	Very Drunk – Strong state of depression, nausea, disorientation, dizzy, increased motor impairment, blurred vision, judgment further impaired
.20% to .24%	Dazed and Confused – Gross disorientation to time and place, increased nausea and vomiting, may need assistance to stand/walk, impervious to pain, blackout likely
.25% to .30%	Stupor – All menal, physical and sensory functions are severely impaired, accidents very likely, little comprehension, may pass out suddenly
.31% and up	Coma – Level of surgical amnesia, onset of coma, possibility of acute alcohol poisoning, death due to respiratory arrest is likely in 50 % of drinkers

BAC – Blood Alcohol Concentration

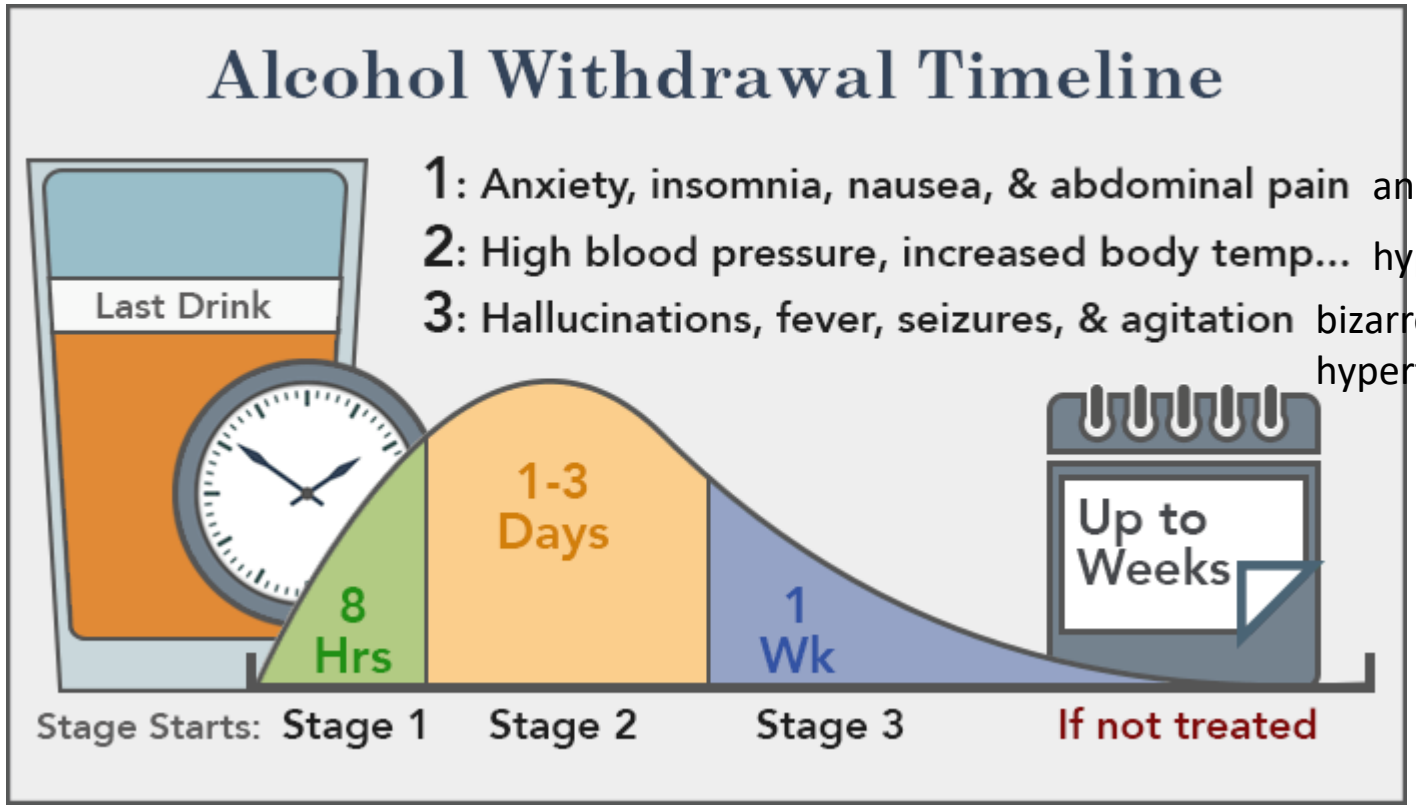
Effects of Alcohol addiction

- $GABA_A$ receptor – agonist – anxiolytic, sedative effect
- NMDA receptor (glutamate) - antagonist – disrupts learning and memory
  - Long term suppression of NMDA receptors makes them more sensitive to glutamate
  - Sudden withdrawal leads to spike in NMDA activity which can cause seizures
  - Drug controlled withdrawal – controlled dosage

- Fetal Alcohol Syndrome (FAS)→ Prenatal alcohol exposure (alcohol consumption by mother or father)
  - brain damage, intellectual disability, poor coordination, poor muscle tone, low birth weight, delayed growth, and/or physical deformity, birth defect

- alcohol consumption can produce *transgenerational epigenetic effects*

Korsakoff's syndrome (a neuropsychological disorder)
- by memory loss, sensory and motor dysfunction, and in advanced stages - severe dementia
- thiamine (vit B1) deficiency (inflammation of the gut lining due to excessive alcohol consumption, which reduces the body's ability to absorb vitamins and nutrients from the gut)
- general loss of cortical white and gray matter


Alcohol Withdrawal Timeline
1: Anxiety, insomnia, nausea, & abdominal pain and tachycardia (rapid heartbeat)
2: High blood pressure, increased body temp... hyperactivity, insomnia, and hallucinations
3: Hallucinations, fever, seizures, & agitation bizarre delusions, disorientation, agitation, confusion, hyperthermia, and tachycardia.
Last Drink
8 Hrs
1-3 Days
1 Wk
Up to Weeks
Stage Starts: Stage 1 Stage 2 Stage 3 If not treated

Nicotine



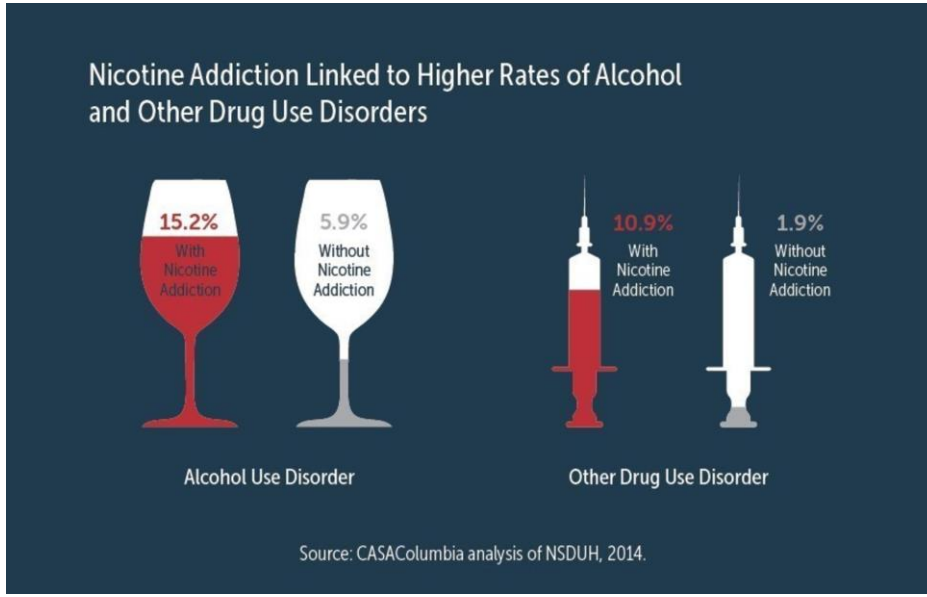

- Tobacco → major psychoactive ingredient → **nicotine**
- Nicotine → in lower quantities in tomato, potato, eggplant (brinjal), and green pepper, dark chocolate, tea
- two common methods of nicotine inhalation:
  - (1) **smoking**—inhaling the smoke from the burning of tobacco (e.g., *cigarettes, cigars*)
  - (2) **vaping**—inhaling a vapor that contains nicotine (e.g., *e-cigarettes*).
  - (3) chewing – especially in India

- Nicotinic receptors are also found in the brain (but fewer than those found at the neuromuscular junction)
  - Nicotine binds to nicotinic Ach receptors located on dopaminergic neurons in the VTA – increased release of dopamine in the Nucleus Accumbens → increased activity in prefrontal cortex, amygdala, hippocampus

- In the body, nicotine triggers the adrenal gland to release epinephrine - **stimulant**

- Short term effects of nicotine - Alertness, improved concentration, better memory, increased heart rate, loss of appetite
- Long term effects of nicotine – high blood pressure, hardening of arteries, respiratory disorders (smoking)

Nicotine – the gateway substance

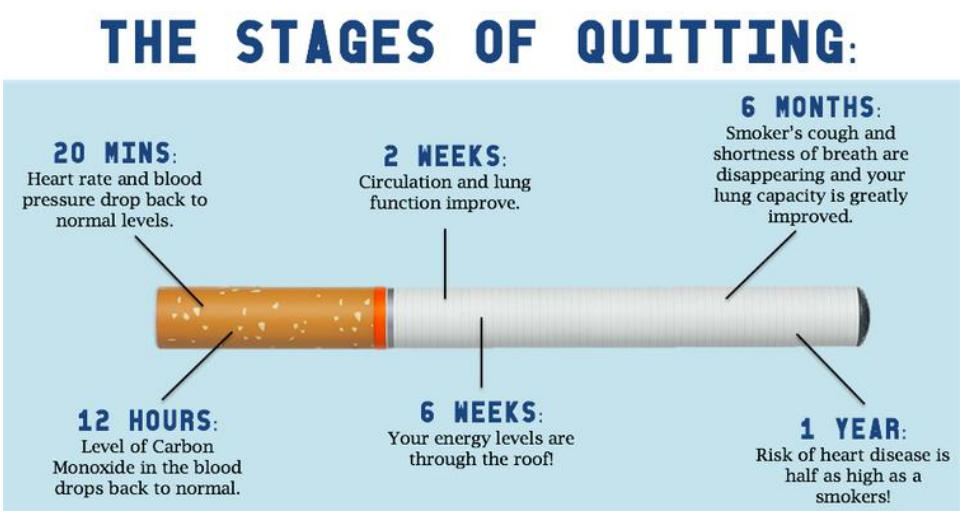


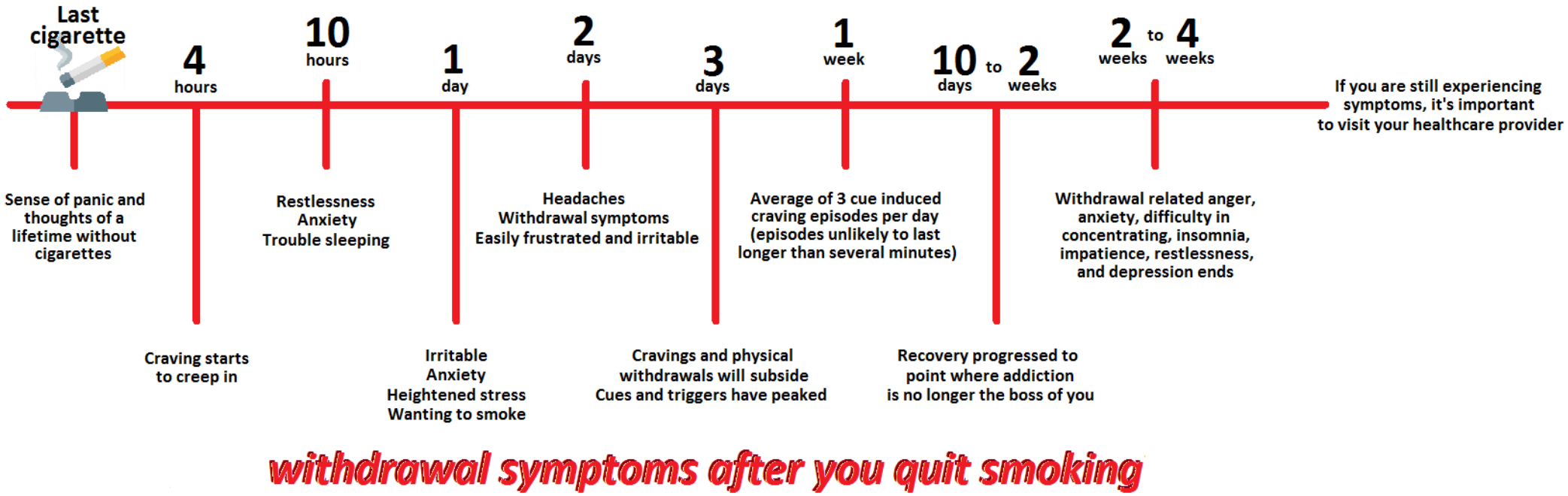
- Mild drug → Does not produce a high, euphoria or any impairment like other drugs
- Because of the heightened alertness and concentration, performance improves → more frequent use
- Nicotine primes the brain towards addiction of other drugs including alcohol → makes it easier for other drugs of abuse to alter brain activity

- Develops tolerance and addiction
- Chronic tobacco use – respiratory infections, heart disease, risk of stroke, cancer of lungs, mouth, throat, or esophagus
- Individuals who live or work with smokers are also likely to develop heart disease and cancer
- Smoking during pregnancy increases the likelihood of miscarriage, stillbirth, early death of the child, psychiatric disorders during adolescence
- Higher cause of death than alcohol and other drugs

Nicotine Withdrawal

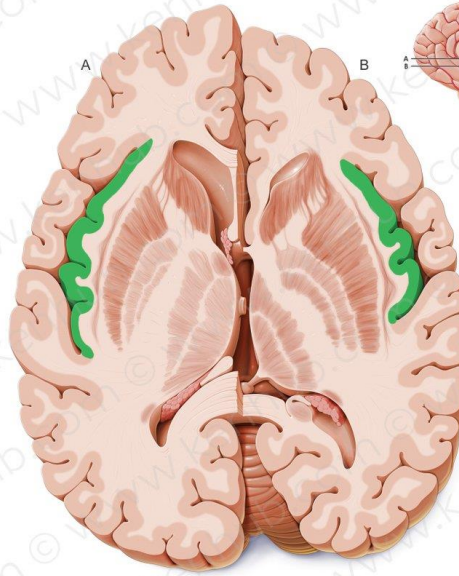
- Typical withdrawal effects, such as depression, anxiety, restlessness, irritability, constipation, and difficulties in sleeping and concentrating.
- Weight gain (increases appetite)





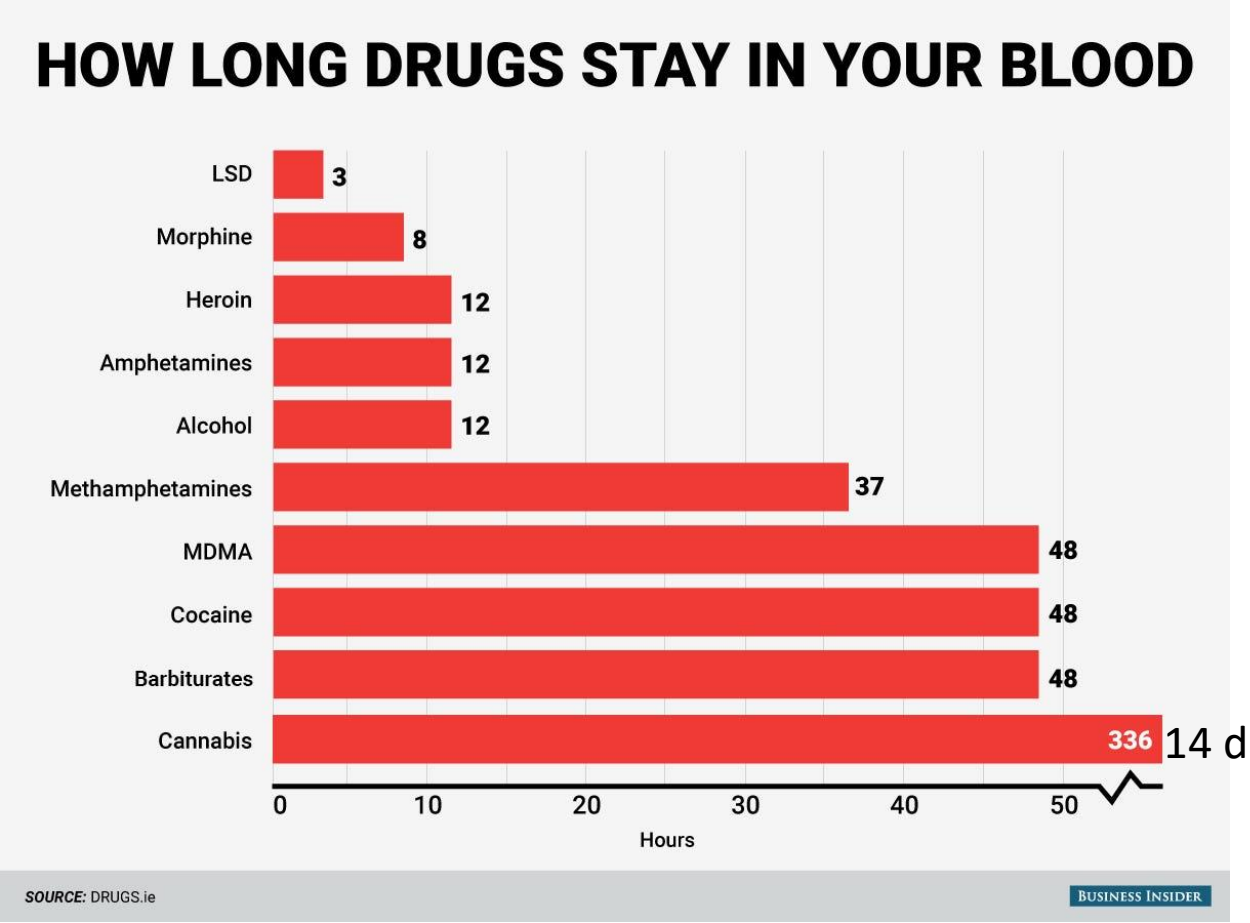
Insula’s role in addiction

- Insula is larger in smokers – plays a role in addiction
- Damage or stroke in the insula can suddenly halt addiction (without relapse or craving
- Patient N story (pg 622-623) helped to understand the role of insula in addiction



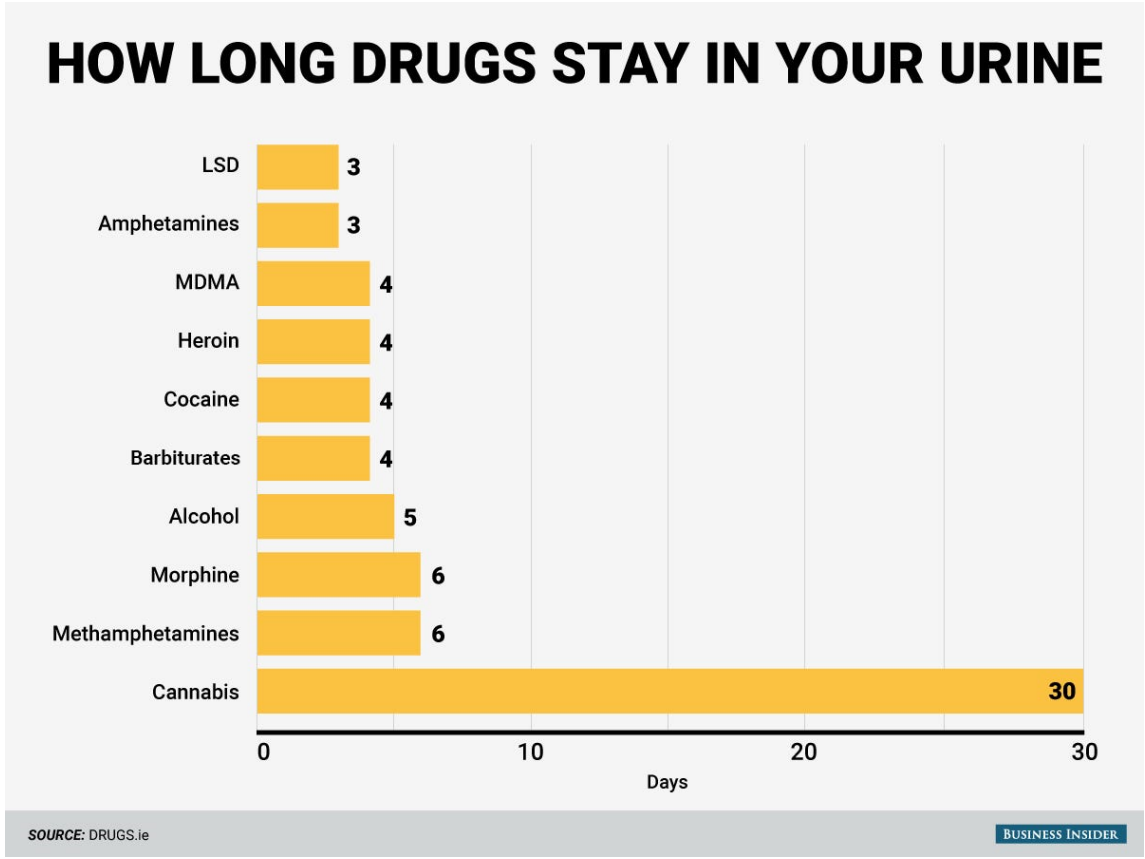
- Role for the insula →
  - conscious drug urges
  - translating bodily signals (perception of sensations inside the body) into conscious feelings,
  - behavioral biases during decision making that involves uncertain risk and reward.

- In rat experiments – inhibitory drugs injected in insula help in nicotine deaddiction

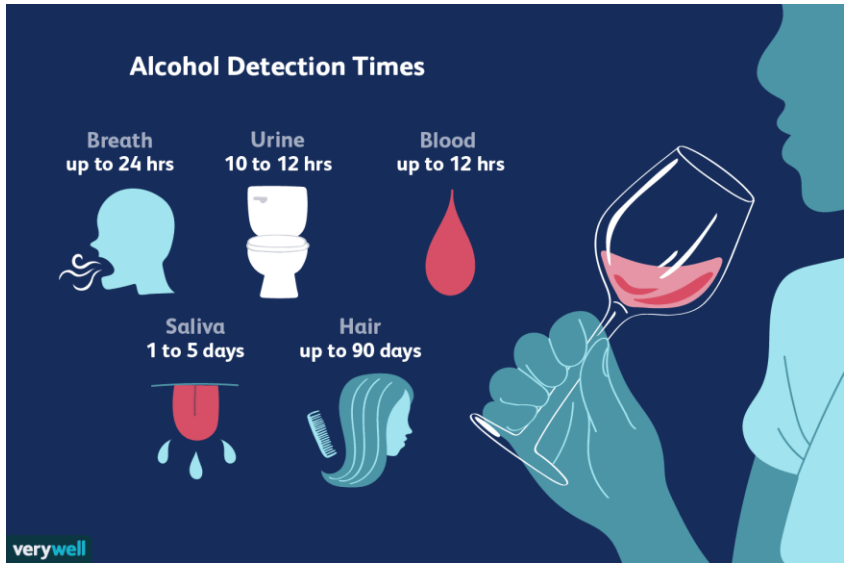


14 days

After a single use of the drug/alcohol

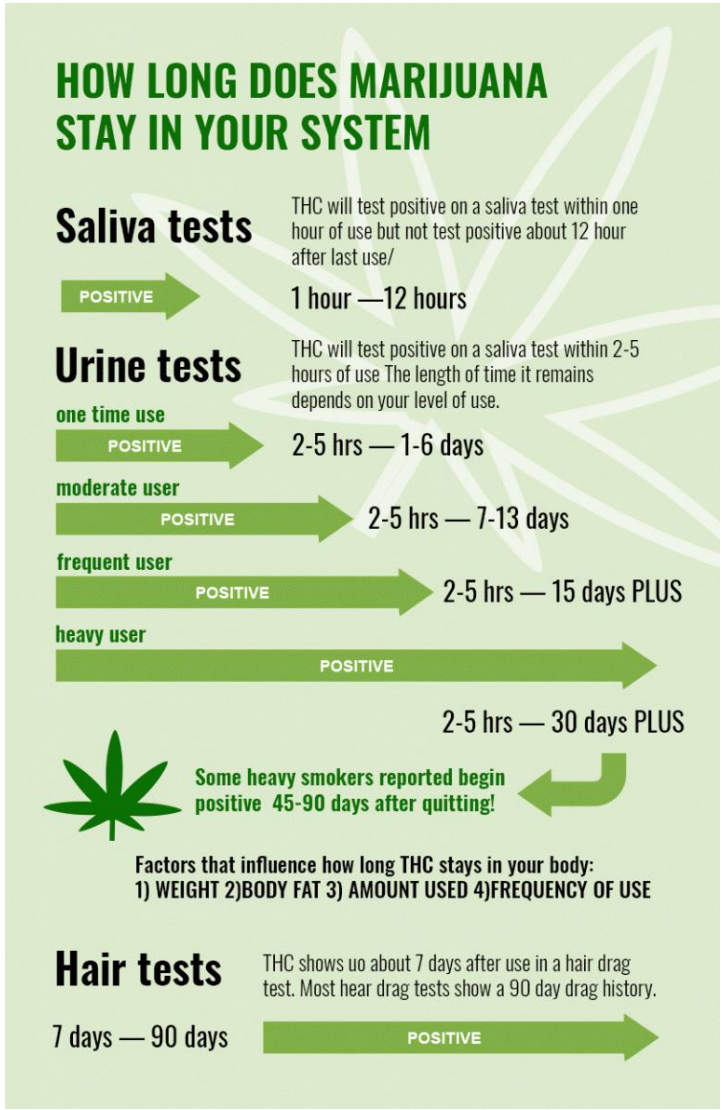



COCAINE IN THE HUMAN BODY
Cocaine remains in one's system anywhere from 3.3–5.5 hours.
EVIDENCE OF COCAINE
Blood Test: up to 24–48 hours
Saliva Test: up to 12–48 hours
Urine Test: up to 2–4 days
Hair Follicle Test: up to 90 days
Note: This is a general guide. Factors like frequency of use can affect the amount of time the drug is evident in your system.


Alcohol Detection Times
Breath up to 24 hrs
Urine 10 to 12 hrs
Blood up to 12 hrs
Saliva 1 to 5 days
Hair up to 90 days
verywell

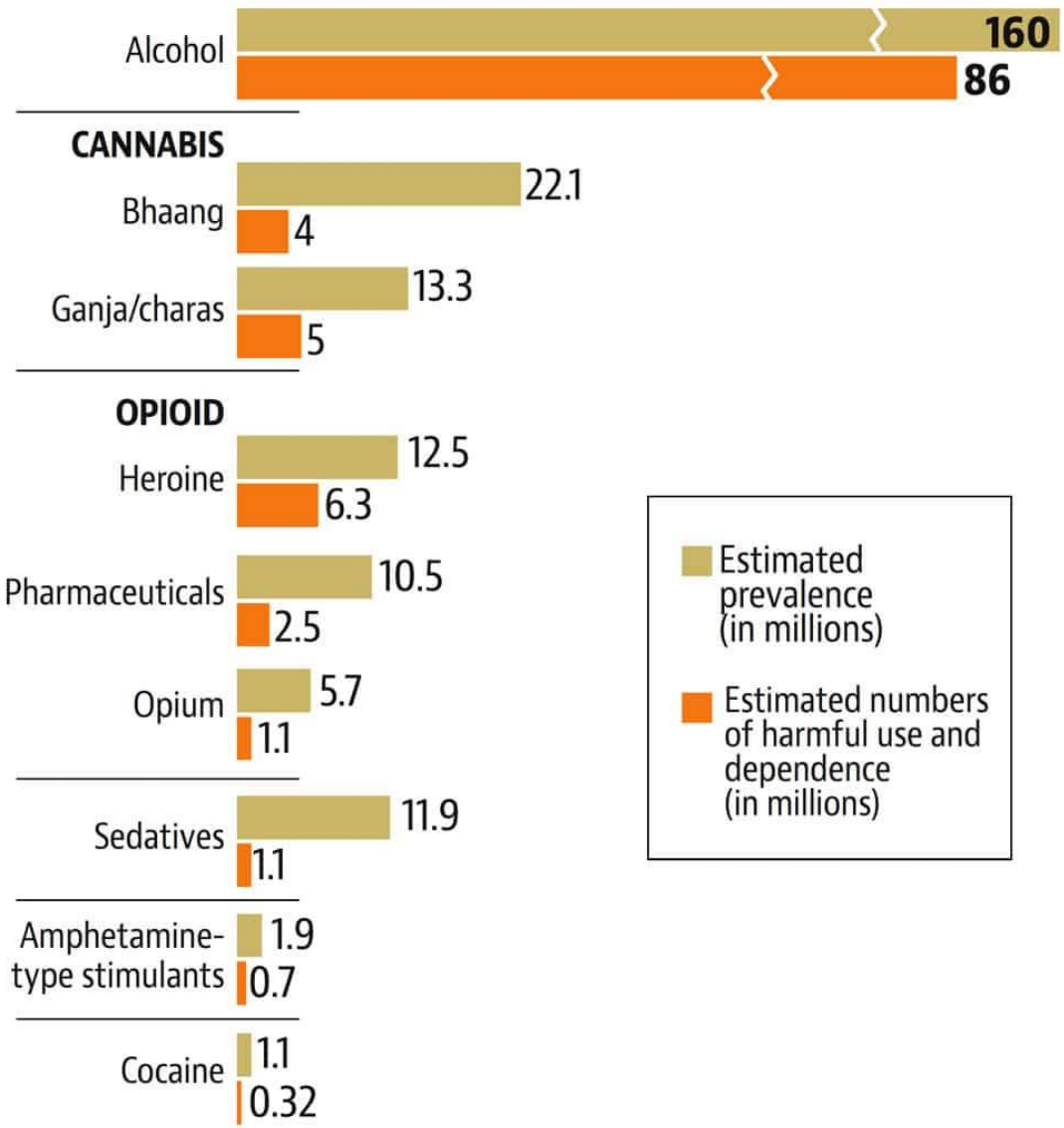

How Long Does Heroin Stay in the System?
Testing Negative
• Urine : 2-7 Days
• Saliva : 5 Hrs.
• Blood : 6 Hrs.
• Hair : Up to 90 Days


Nicotine
HAIR
SALIVA
URINE
BLOOD
90 DAYS
4 DAYS
4 DAYS
2-3 DAYS


HOW LONG DOES MARIJUANA STAY IN YOUR SYSTEM
Saliva tests
THC will test positive on a saliva test within one hour of use but not test positive about 12 hour after last use/
POSITIVE
1 hour —12 hours
Urine tests
THC will test positive on a saliva test within 2-5 hours of use The length of time it remains depends on your level of use.
one time use
POSITIVE
2-5 hrs — 1-6 days
moderate user
POSITIVE
2-5 hrs — 7-13 days
frequent user
POSITIVE
2-5 hrs — 15 days PLUS
heavy user
POSITIVE
2-5 hrs — 30 days PLUS
Some heavy smokers reported begin positive 45-90 days after quitting!
Factors that influence how long THC stays in your body:
1) WEIGHT 2)BODY FAT 3) AMOUNT USED 4)FREQUENCY OF USE
Hair tests
THC shows uo about 7 days after use in a hair drag test. Most hear drag tests show a 90 day drag history.
7 days — 90 days
POSITIVE

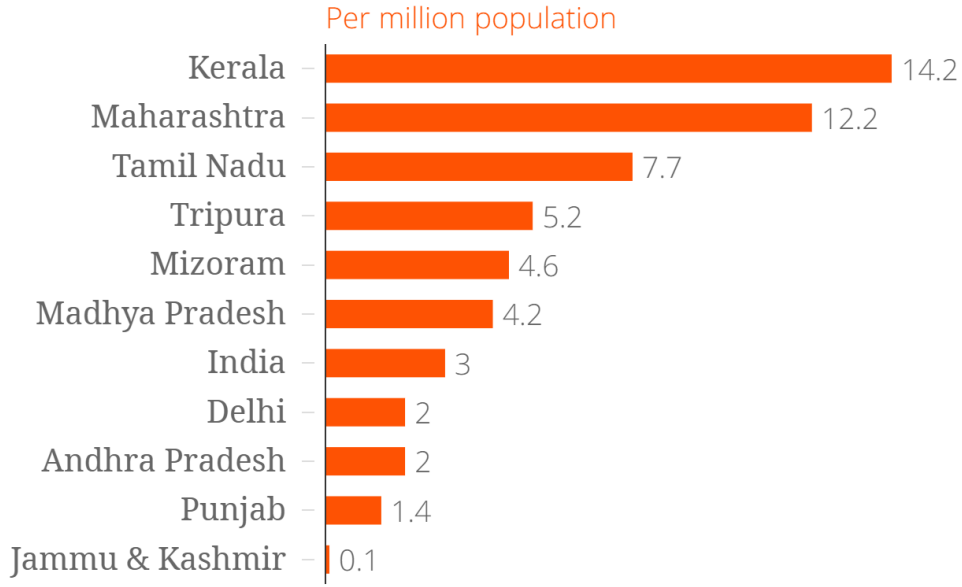
Scale of substance abuse in India

Alcohol is the most commonly used legal psychoactive substance, with about about 14.6% of population using it



(Source: Magnitude of Substance Abuse in India Report, 2019)

Drug addiction or abuse related suicides in some Indian states



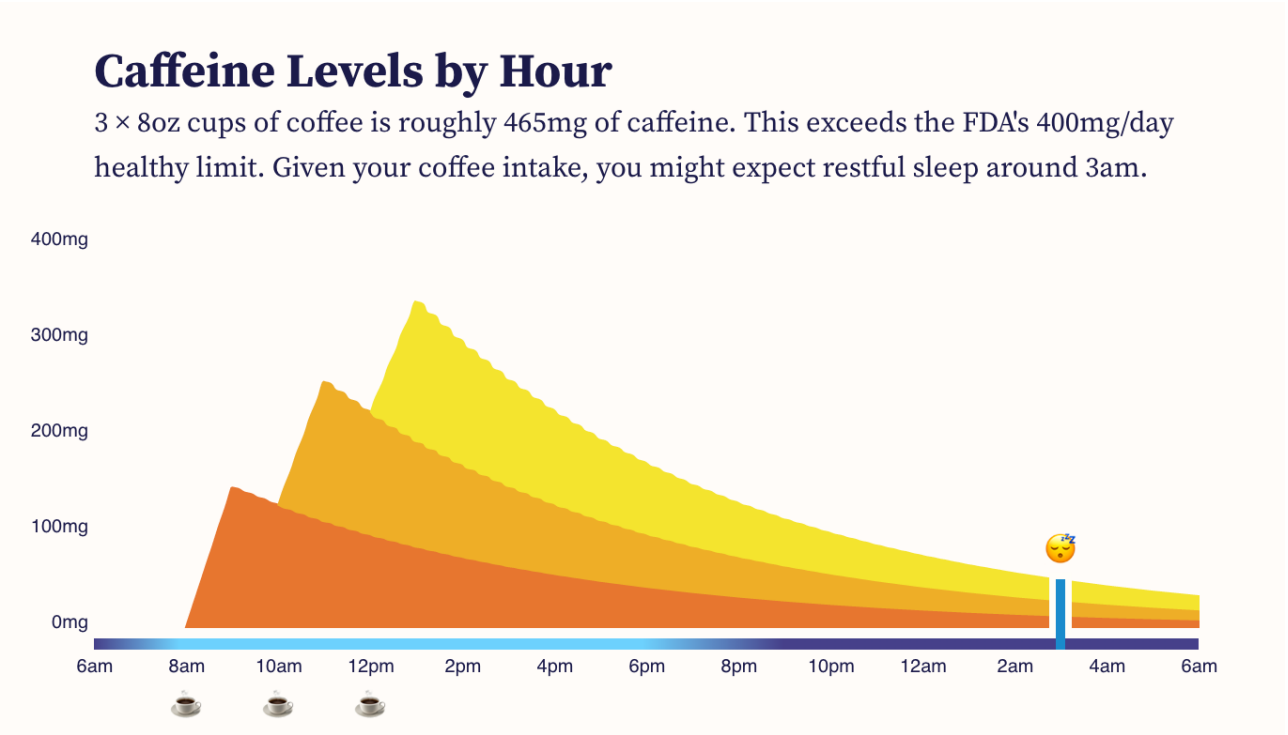
Scroll.in

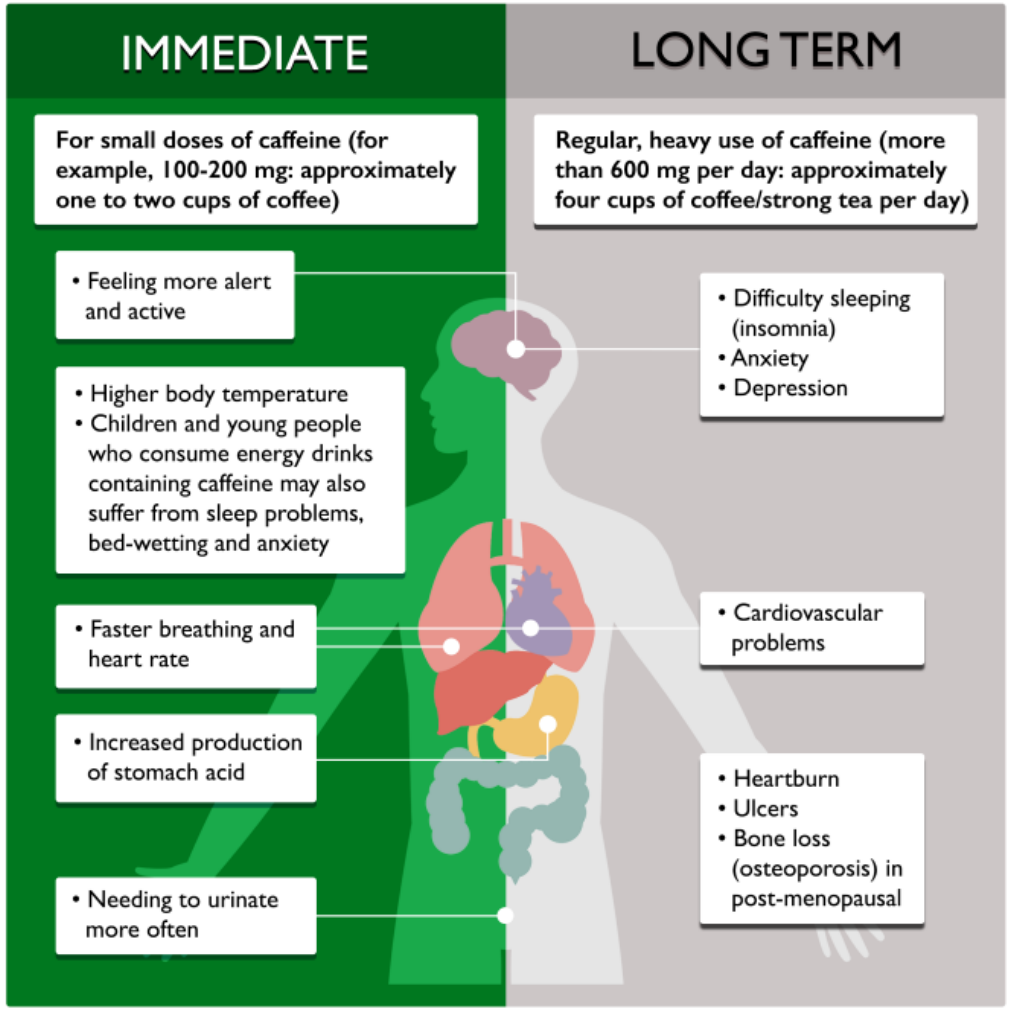
Data: Ministry of Social Justice and Empowerment

2014

Caffeine

- Stimulant – Makes you alert and active
- Caffeine can cross the blood brain barrier
- Mild use is helpful but chronic intake is harmful
- Effect persists for 4-6hrs
- Caffeine binds to
  - Increases dopamine and norepinephrine - addictive
  - Adenosine receptors antagonist – prevents sleepiness





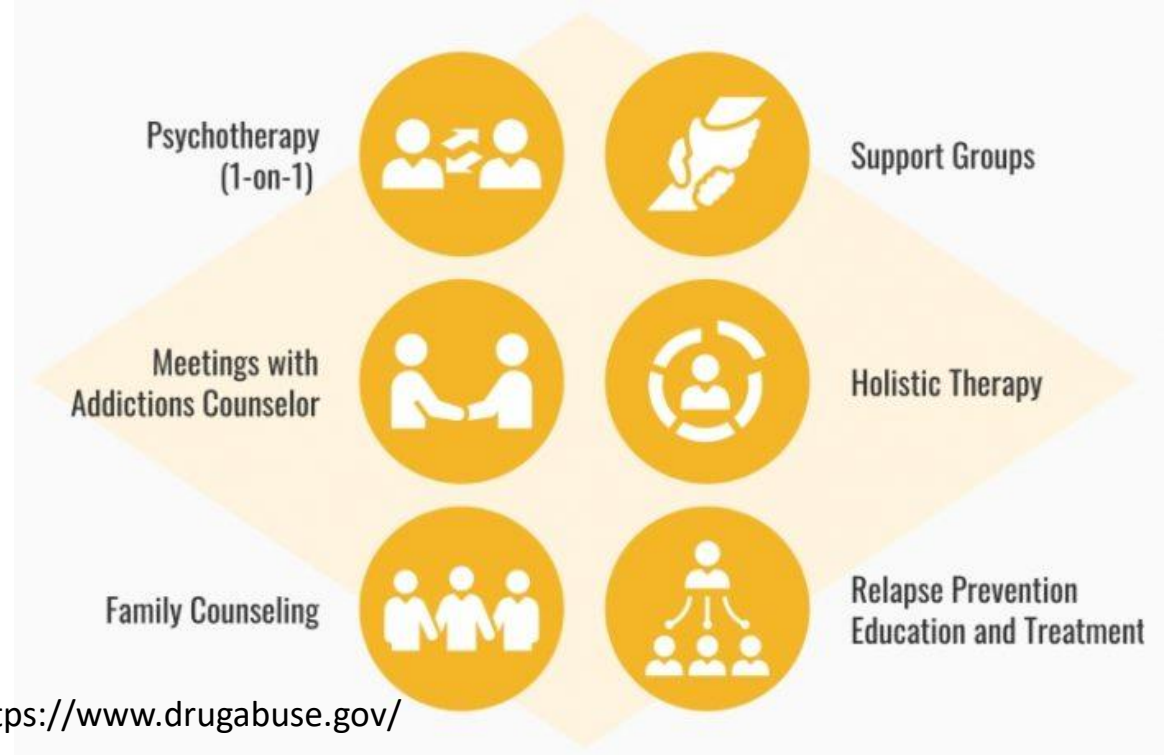
Can cause dehydration

https://www.brown.edu/campus-life/health/services/promotion/content/what-are-effects-caffeine

Treatment for substance abuse

Behavioral therapies help patients:

- modify their attitudes and behaviors related to drug use
- persist with other forms of treatment, such as medication
- be productive in the family, at work, and in society
- increase healthy life skills



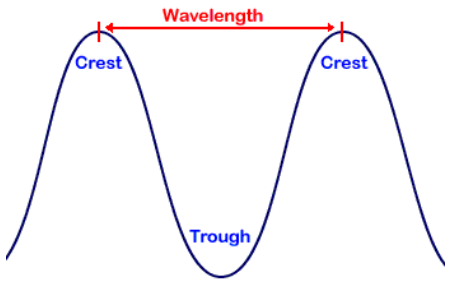
https://www.drugabuse.gov/

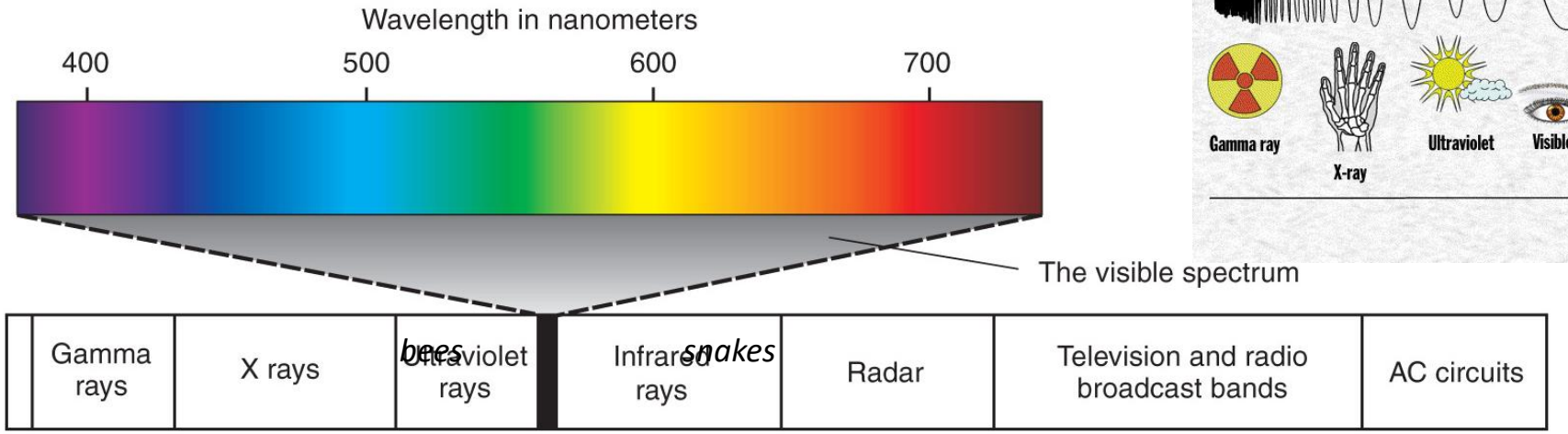
Gateway Foundation

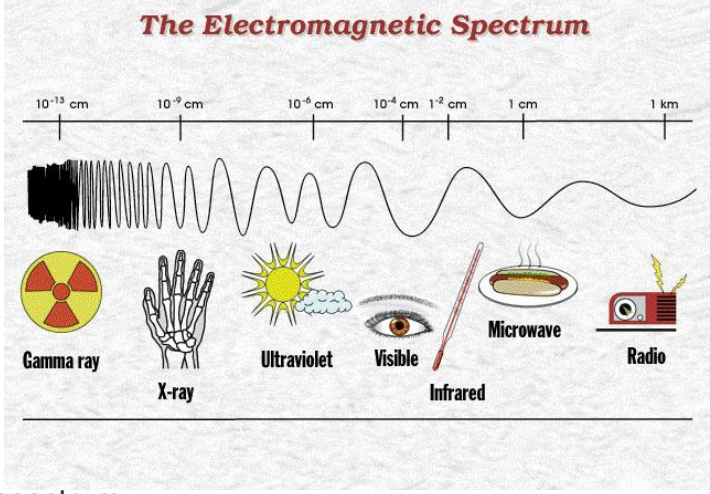
- Any particular pattern of that you observed for legal vs illegal drugs (esp. related to neurotransmitters)?

Vision

Our Visual stimulus







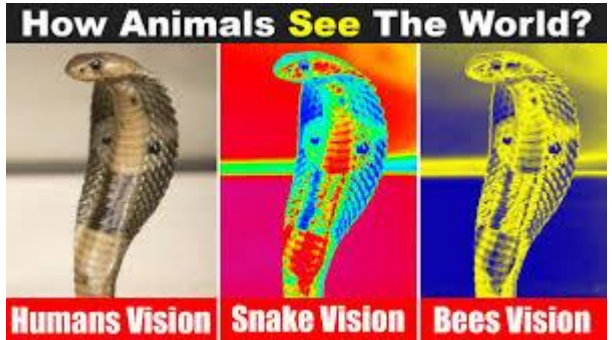
The ability to see ultraviolet (UV) helps guide bees to the pollen containing parts of flowers

Human View
(No UV Sensitivity)

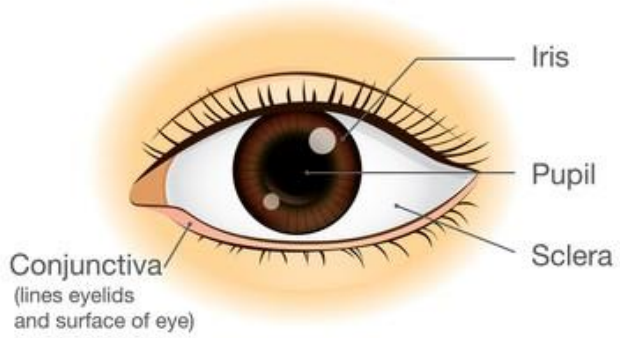
Bee View
(UV Sensitivity)

Human View
(No UV Sensitivity)

Dog View
(Some UV Sensitivity)

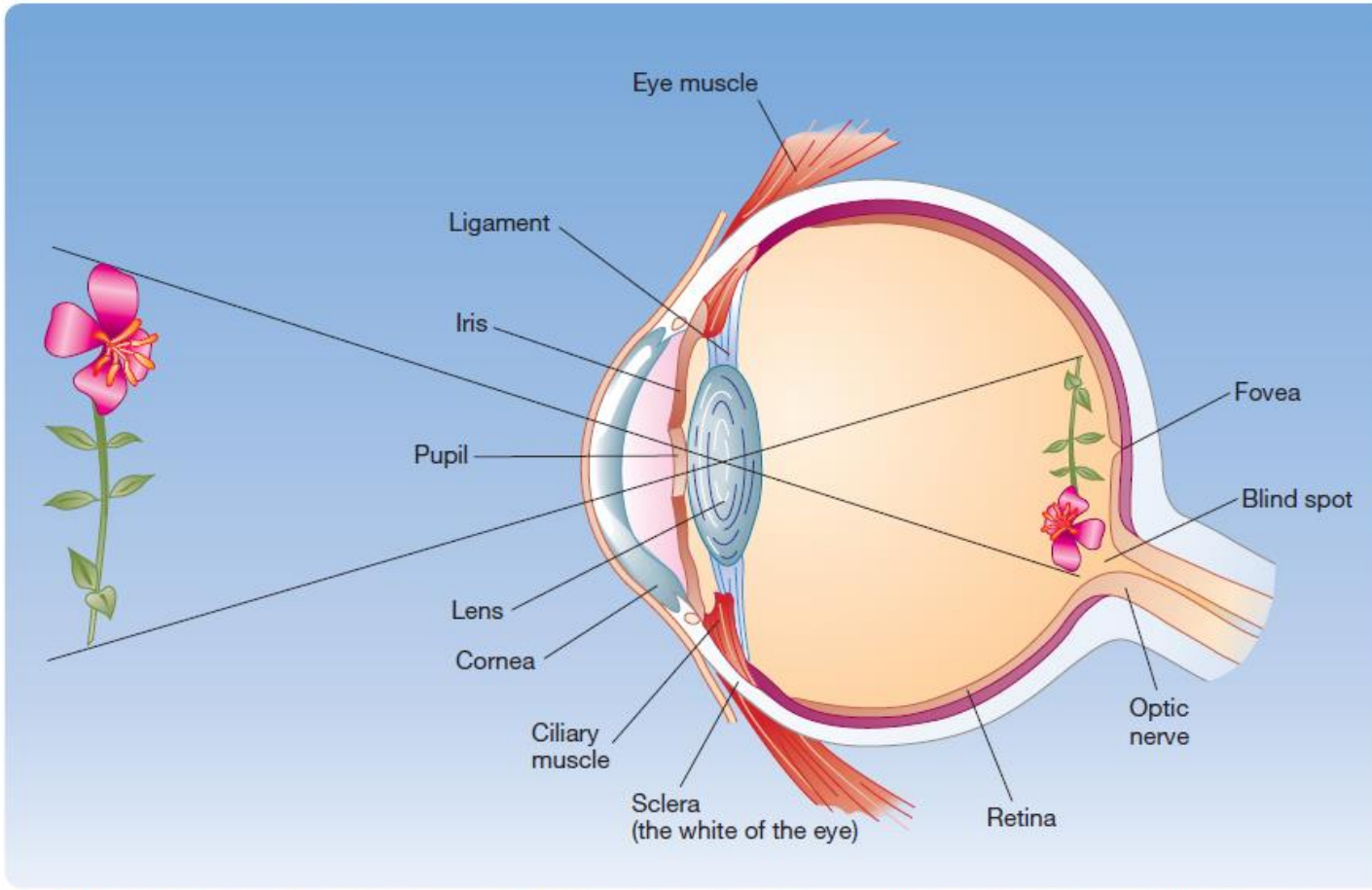


Anatomy of the eye



The iris gives the eye its characteristic color—blue, brown, or other.

Figure 6.4 The human eye, a product of approximately 600 million years of evolution.

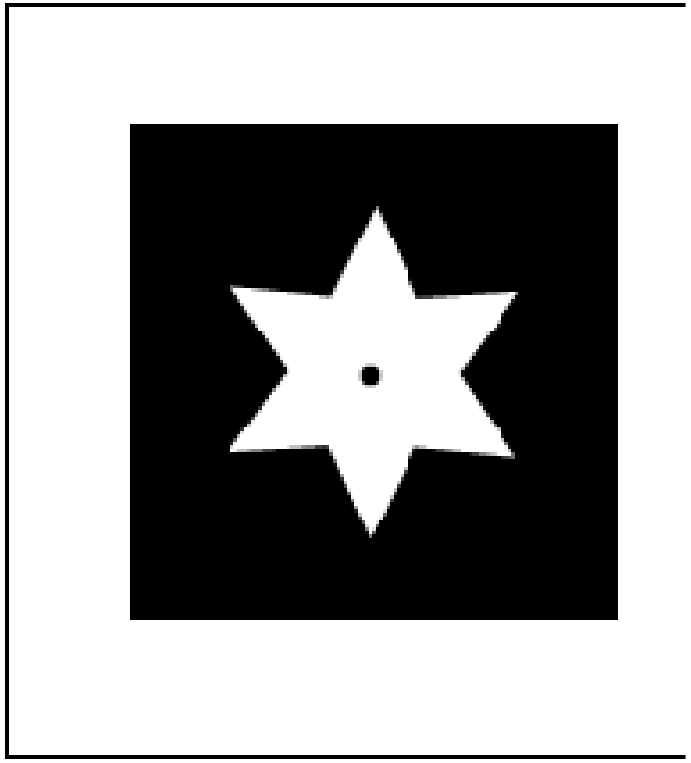


Based on Lamb, T. D., Collin, S. P., & Pugh, E. N. (2007). Evolution of the vertebrate eye: Opsins, photoreceptors, retina and eye cup. *Nature Reviews Neuroscience*, 8, 960–975.

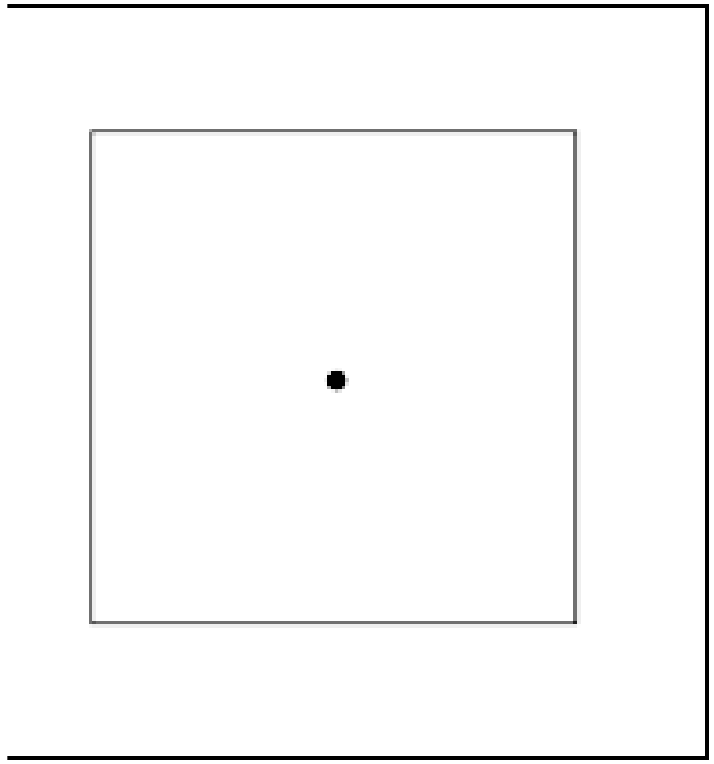
- Light enters the eye through the pupil, whose size is regulated by the iris.
- Light travels into the lens all the way to the retina

- **Fovea** – central region of the retina which contain only cones and mediates the most acute vision

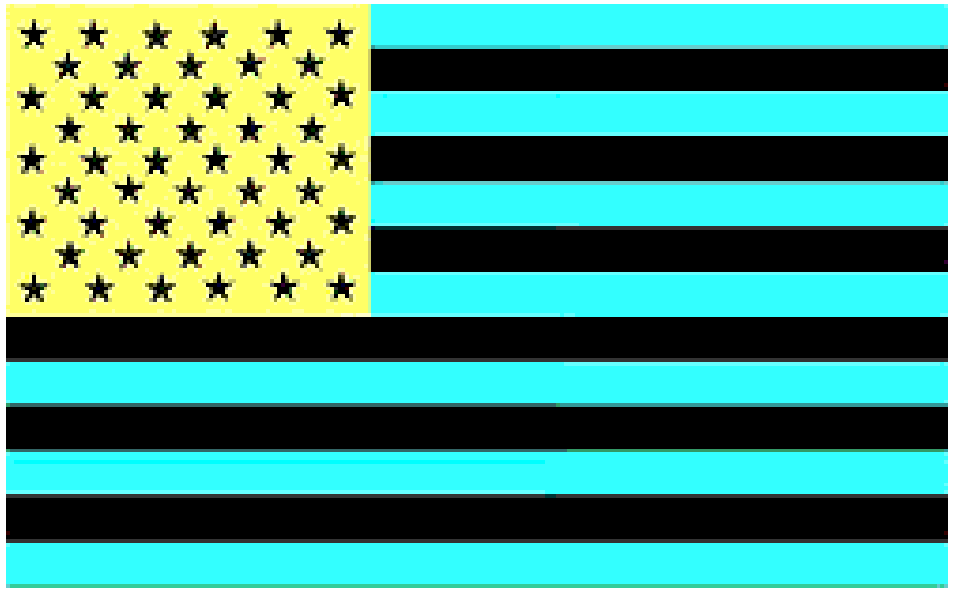
Look at the dot inside the star for 60 secs

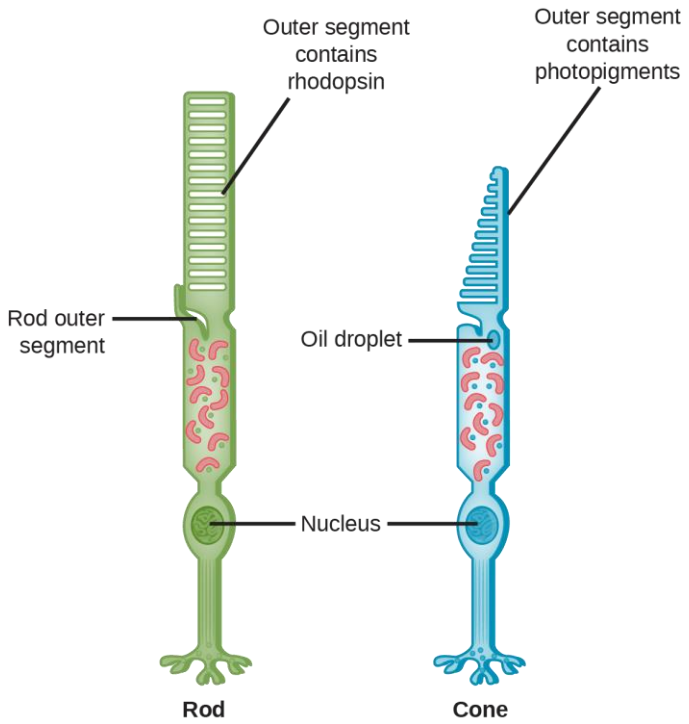


look at the dot inside the white box



Look at the flag for 60 secs and then look at the white/blank area



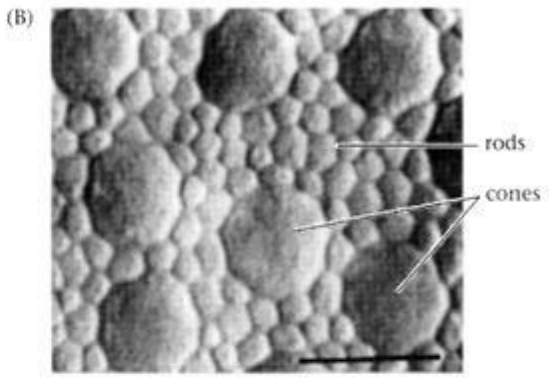


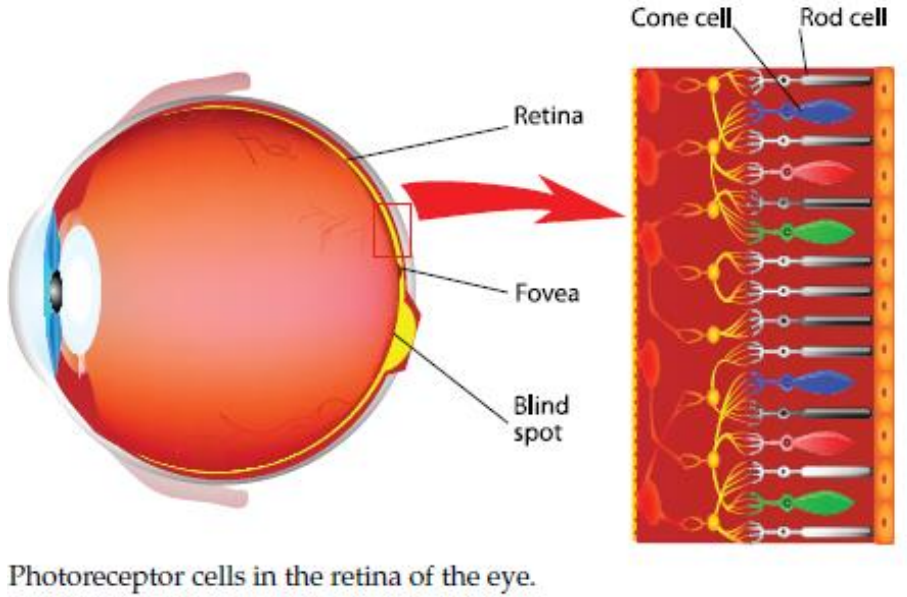
- Retina has two types of photoreceptors
  - **Rods** (12 million) – sensitive to light intensity (active in low light)
  - **Cones** (6 million) – sensitive to color/wavelength (active in normal daylight)

- Light passes through the transparent layers to the retina to stimulate the photoreceptors – rods/cones

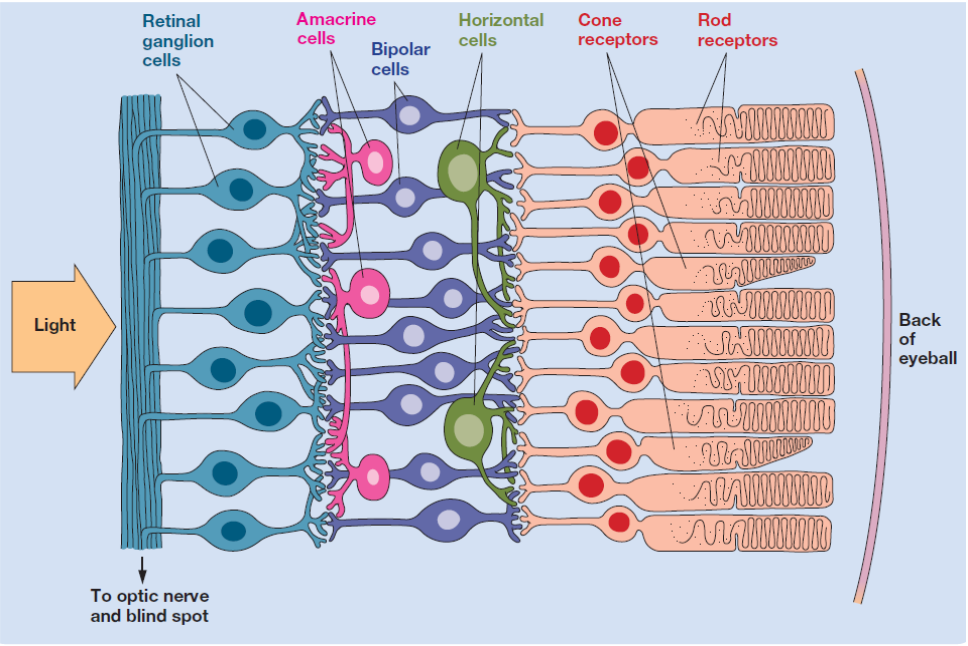
- Photoreceptors transduce light energy into electrical impulses (by release of different neurotransmitters)

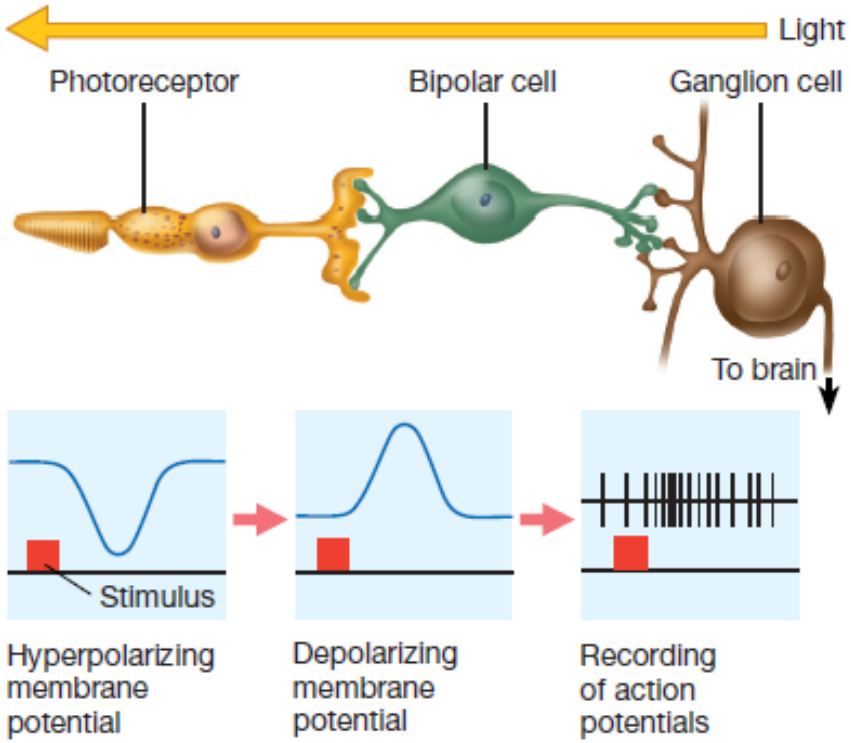
- Rods work in dim light conditions
- Cones function under good light intensity (daylight).

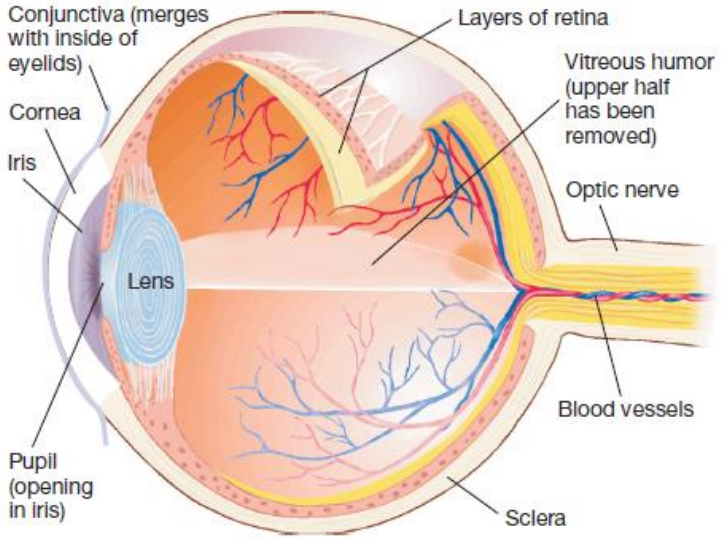




Photoreceptor cells in the retina of the eye.



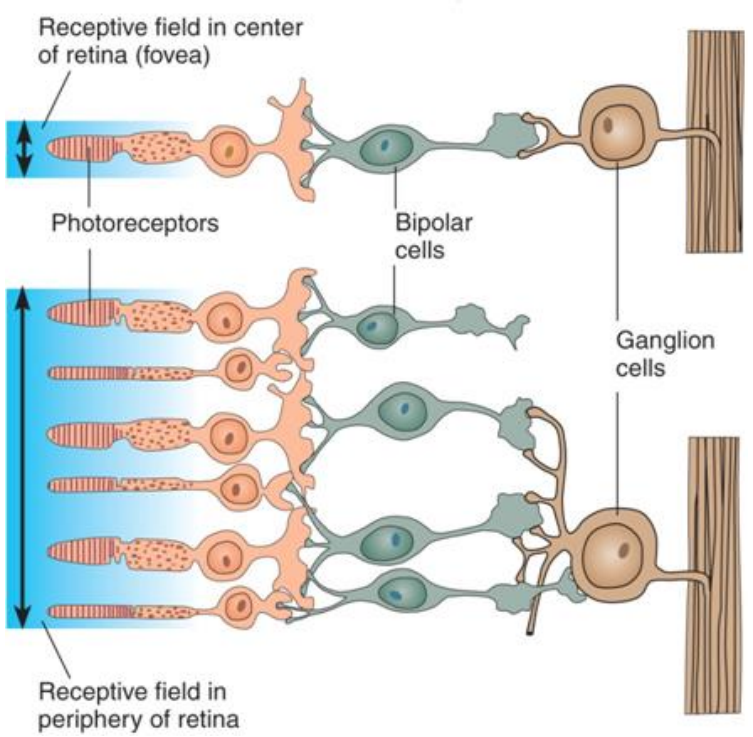




Neural circuitry in the retina

- Rods have rhodopsin that are turned off (hyperpolarized) when light falls on them. (detect light with closed eyelids)
- Cones have *photopigments* that are bleached (pigments change their color) when light falls on them. The bleaching causes hyperpolarization which changes the rate of glutamate neurotransmitter released.
- Hyperpolarization of the photoreceptor (PRs) results in reduced release of neurotransmitter (PRs are always ON and light falling on them reduces their activity or turns them OFF).
- Glutamate normally hyperpolarizes (inhibits) the bipolar cell. When light falls on the photoreceptors, the reduction of glutamate release results in depolarization (de-inhibits) of the bipolar cells. This depolarization causes the bipolar cells to excite the subsequent cells (ganglion cells) – increasing its firing rate
- The ganglion cell axons form the optic nerve

General versus peripheral acuity: ganglion cells in the fovea receive input from a smaller number of photoreceptors than in the periphery and hence provide more visual acuity



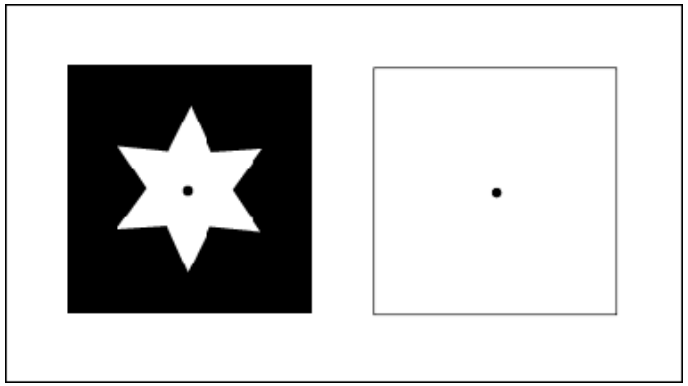
- Receptive field of a neuron is that part of the visual field that the neuron “sees”

- If a neuron receives info. from PRs located in the fovea, its receptive field will be the fixation point – the point where the eye is looking.

- Fovea has equal no. of ganglion cells and cones, so the 1:1 ratio gives high acuity at the fixation point.

- The ratio decreases for the peripheral areas where the visual preciseness is less, i.e. more no. of PRs spread over a larger area are required to code information from the periphery.

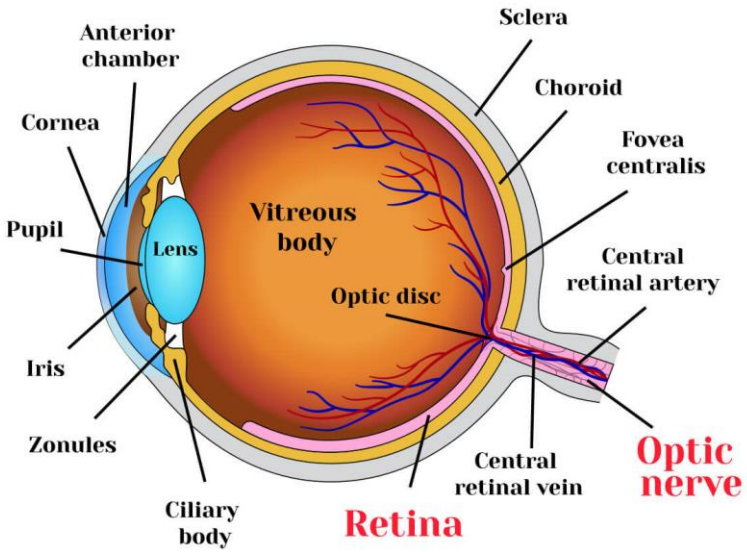
Afterimage



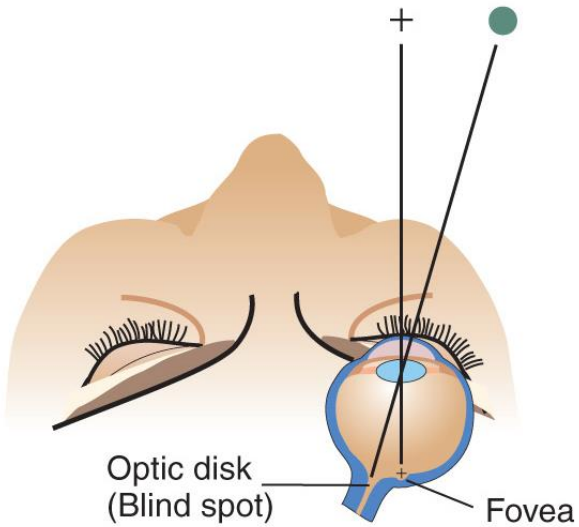
Negative afterimages - occur when the rods and cones, which are part of the retina, are overstimulated and become desensitized. This desensitization is strongest for cells viewing the brightest part of the image, but is weakest for those viewing the darkest. When you look away, the least depleted cells react strongest, and vice versa, and you see an image with colors that are the reverse of how the image originally appeared.

A positive afterimage ?

Blind Spot



Blind spot - small area of the retina where no receptors (no rods or cones) are present. This is where the optic nerve exits the eye carrying visual information to other brain areas.



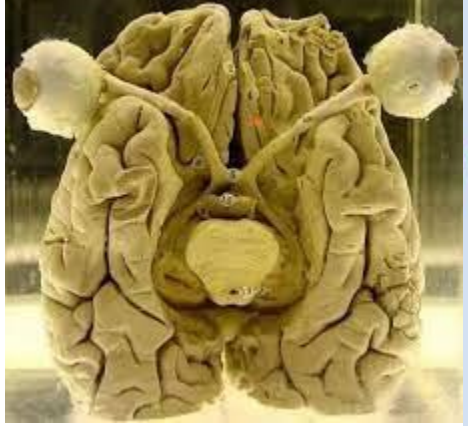
Check It Out

Your Blind Spot and Completion

First, prove to yourself that you do have areas of blindness that correspond to your retinal blind spots. Close your left eye and stare directly at the A below, trying as hard as you can to not shift your gaze. While keeping the gaze of your right eye fixed on the A, hold the text at different distances from you until the black dot to the right of the A becomes focused on your blind spot and disappears at about 13 centimeters (5 inches).

- If the blindsplot on the retina has no photoreceptors why don’t we see a black/blind/empty spot when we see the world?

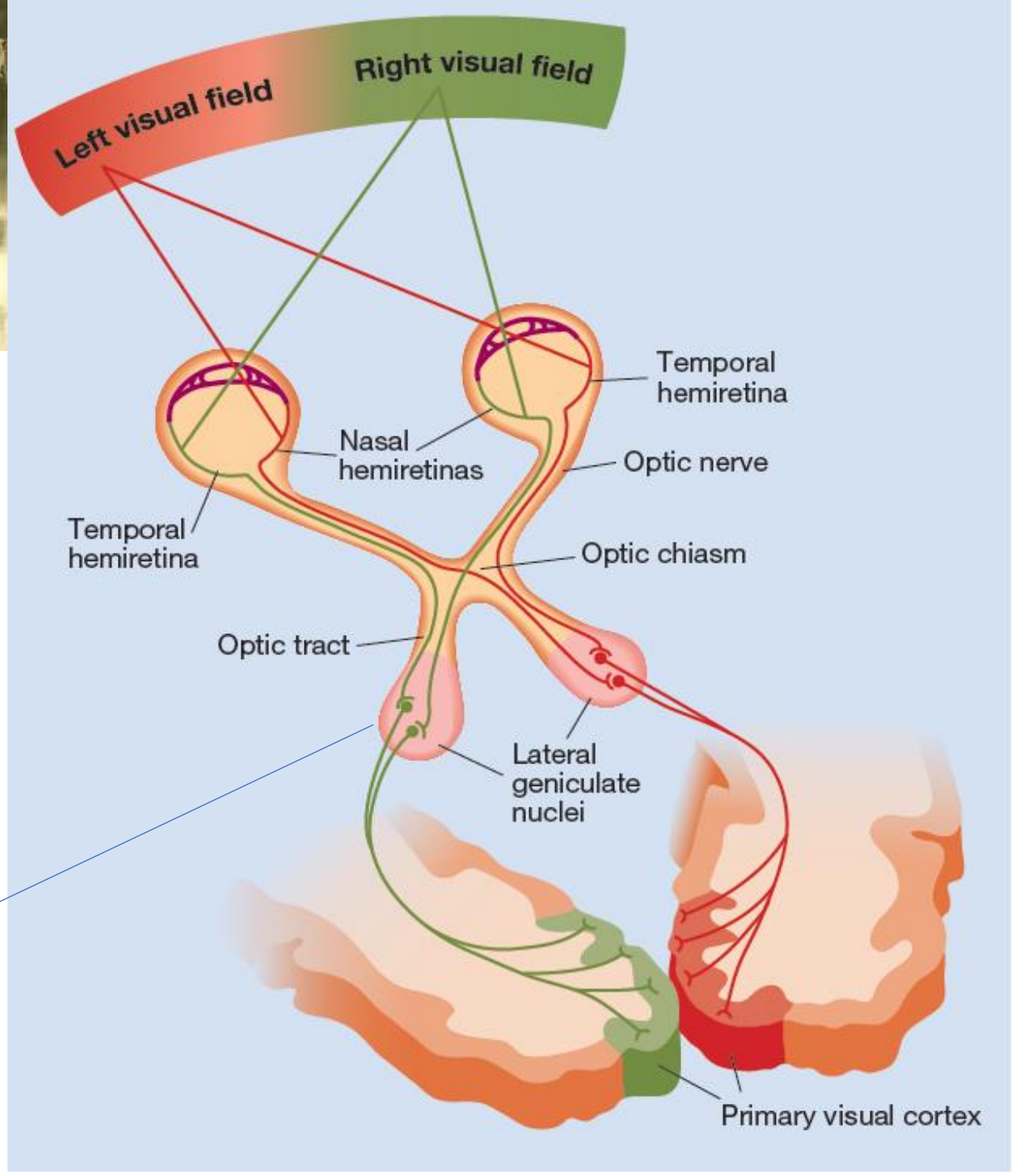
If each eye has a blind spot, why is there not a black hole in your perception of the world when you look at it with one eye? You will discover the answer by focusing on B with your right eye while holding the text at the same distance as before. Suddenly, the broken line to the right of B will become whole. Now focus on C at the same distance with your right eye. What do you see?



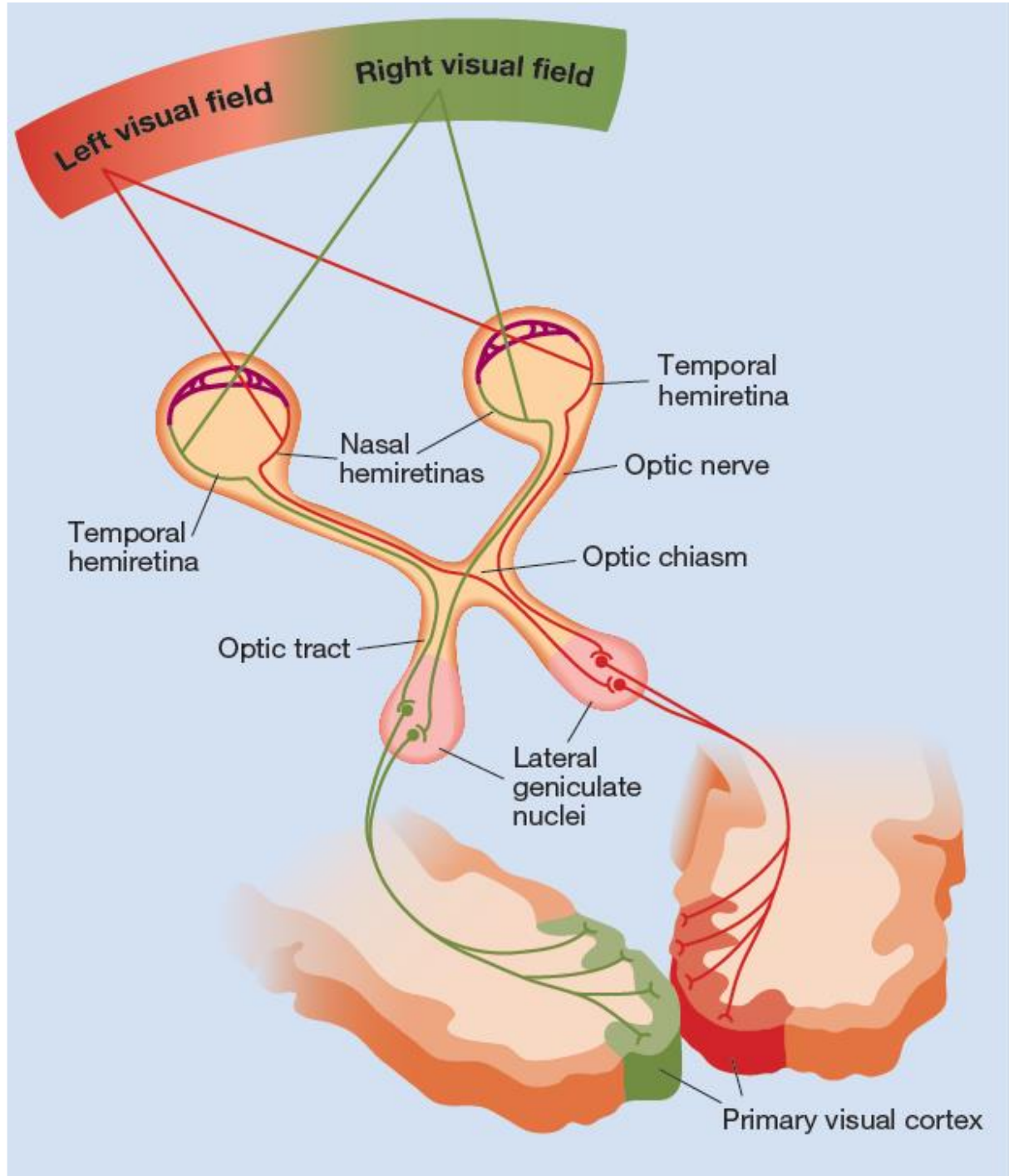
Information from retina via optic nerve → optic chiasm → LGN → primary visual cortex

- All information from the lateral (outer) side of a retina enters the ipsilateral optic tract
- All information from the nasal (inner) side of the retina crosses over at the chiasm and enters the contralateral optic tract.

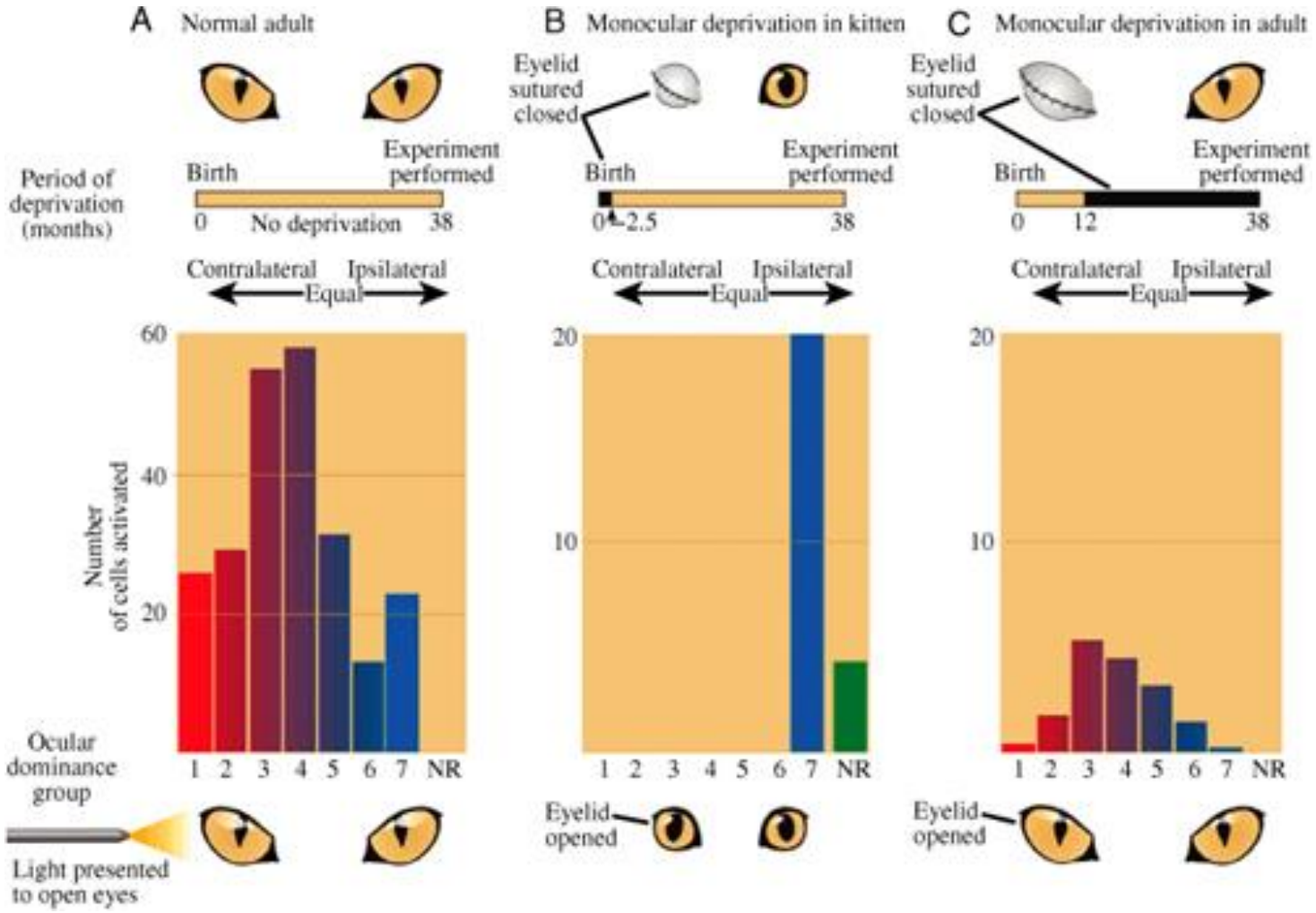
thalamus

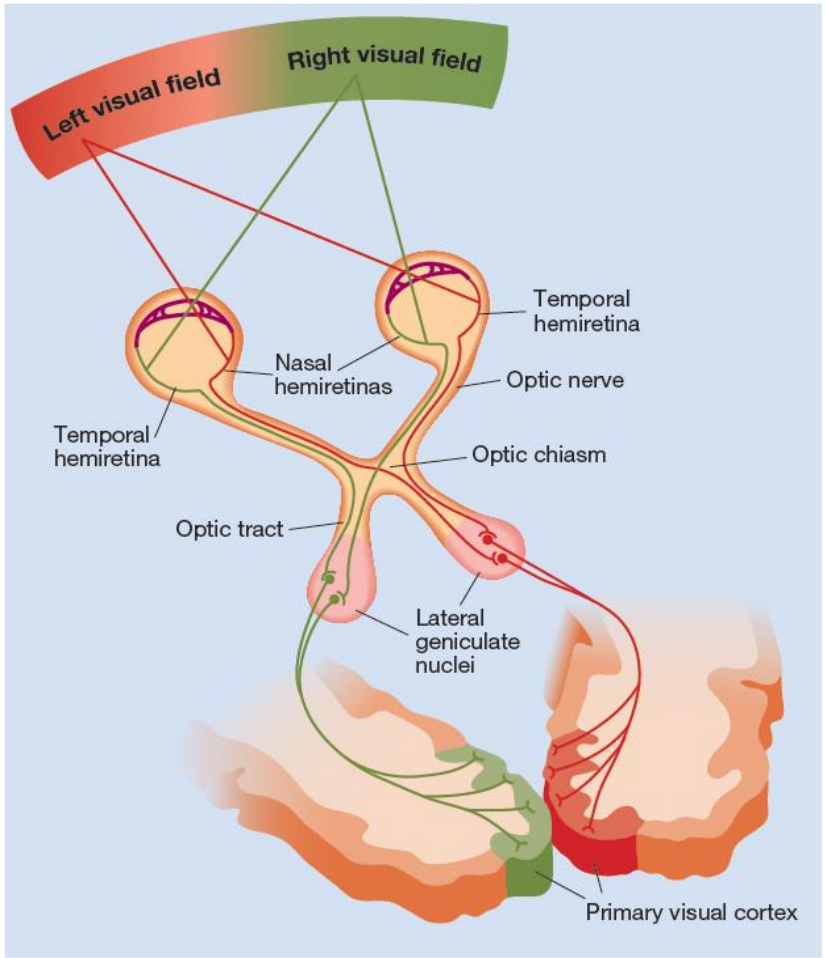


Visual fields and Primary visual pathway


Left visual field
Right visual field
Temporal hemiretina
Nasal hemiretinas
Optic nerve
Temporal hemiretina
Optic chiasm
Optic tract
Lateral geniculate nuclei
Primary visual cortex

Visual experience drives the wiring in the visual cortex





https://www.informit.com/articles/article.aspx?p=1431818

- How do we perceive depth?
- What allows us to perceive depth?

Depth perception

Veronica Lara/Shutterstock

Binocular cues help us to perceive depth.

Check It Out

The Position of Eyes

Here you see three animals whose eyes are on the front of their heads (a human, an owl, and a lion) and three whose eyes are on the sides of their heads (an antelope, a canary, and a squirrel). Why do a few vertebrate species have their eyes side-by-side on the front of the head while most species have one eye on each side?

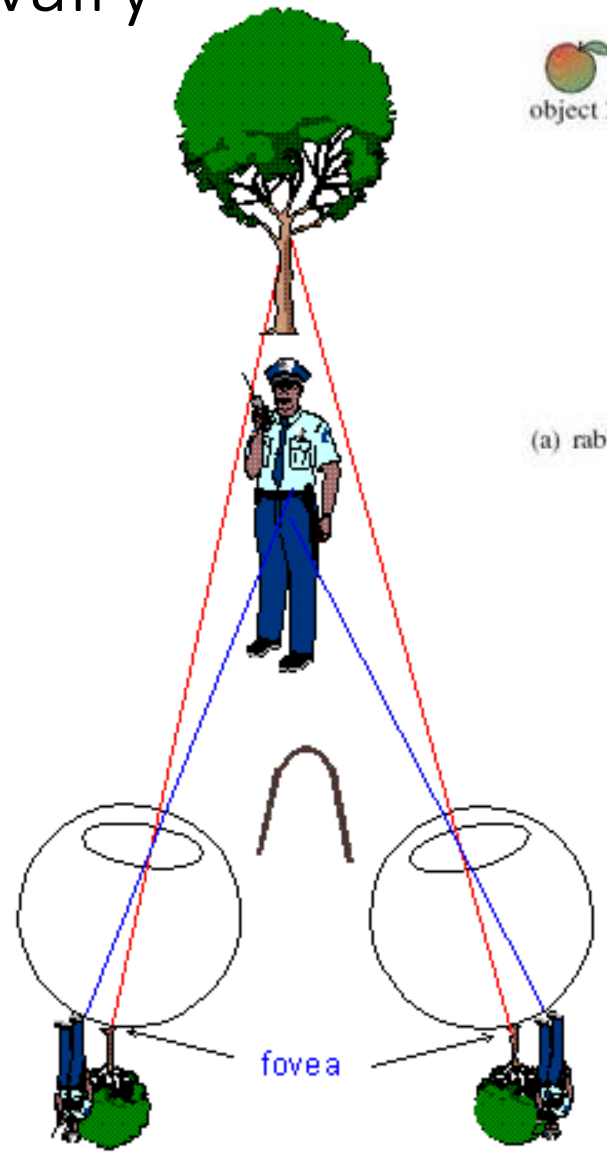
In general, predators tend to have the front-facing eyes because this enables them to accurately perceive how far away prey animals are; prey animals tend to have side-facing eyes because this gives them a larger field of vision and the ability to see predators approaching from most directions.

Top row from left: Guiziou Franck/Hemis/Alamy Stock Photo; Matthew Cuda/Alamy Stock Photo; C.K. Lorenz/Science Source
Bottom row from left: Naomi Engela Le Roux/123RF; Vasiliy Vishnevskiy/123RF; Colin Varndell/Nature Picture Library

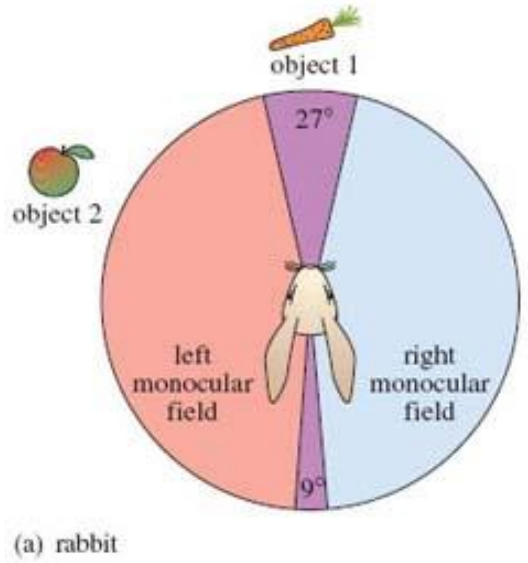
Binocular Disparity or Rivalry (Depth perception) 3D vision

The brain uses binocular disparity to extract depth information from the two-dimensional retinal images

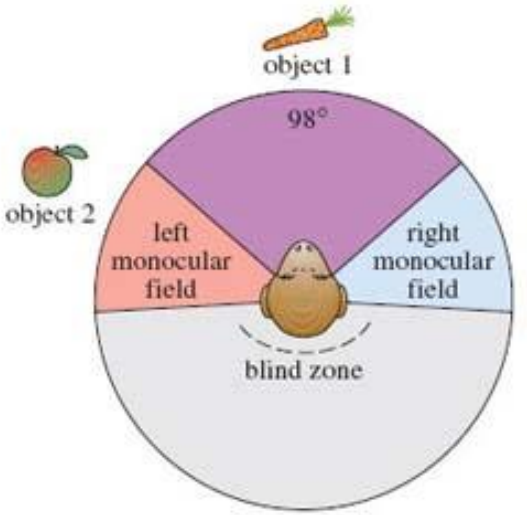
Both of your eyes perceive the same object at slightly different locations or at slightly different angles, but your brain can merge the two images into one 3-D image (stereoscopic vision)



Binocular Disparity



(a) rabbit



(b) monkey

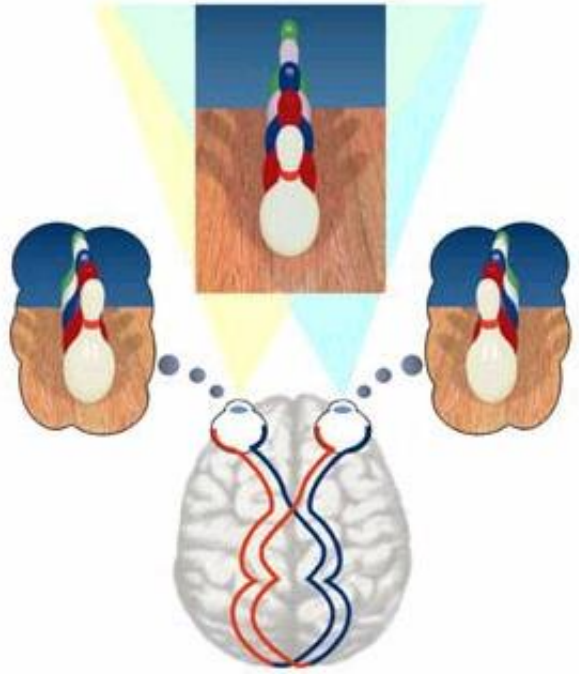
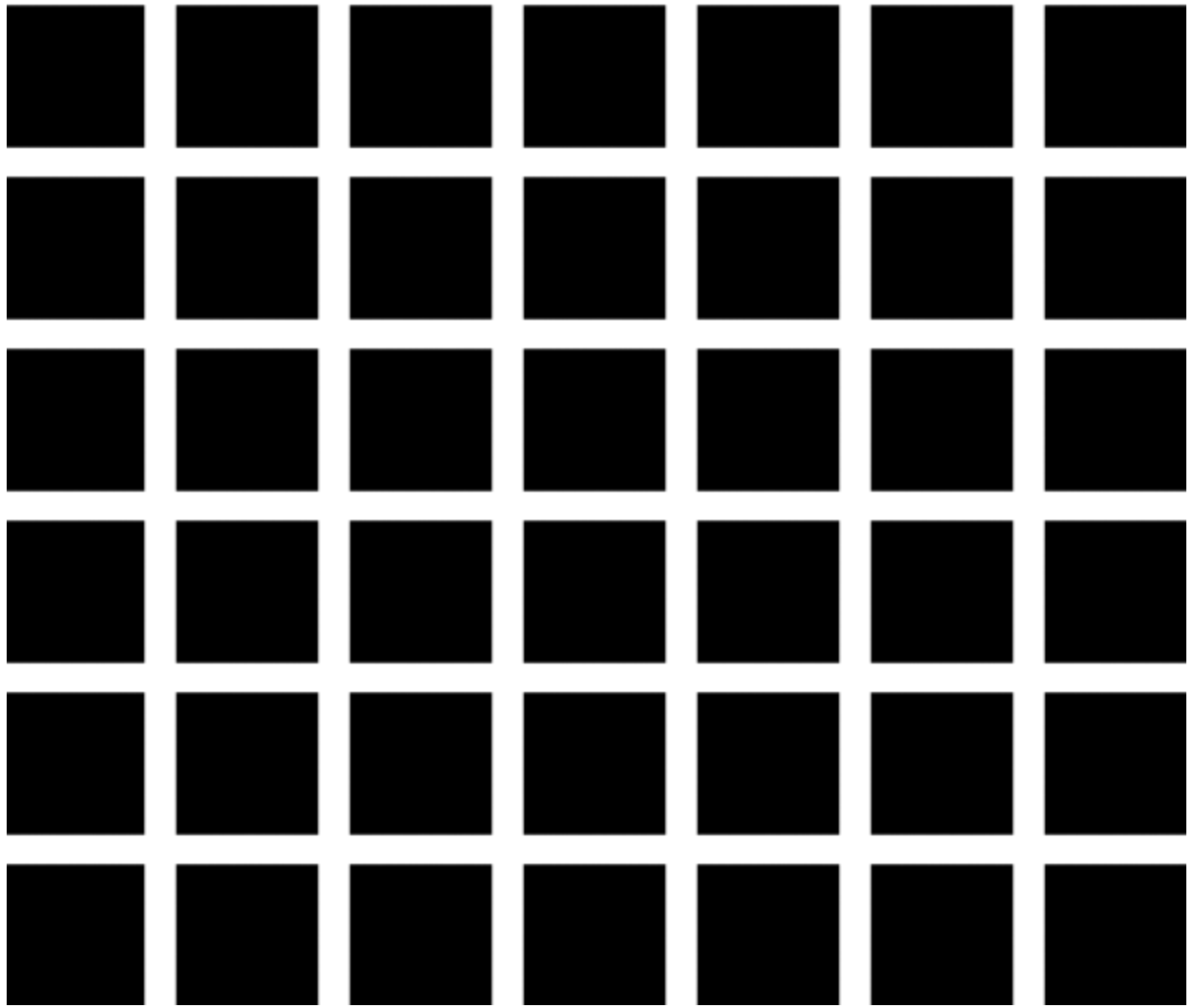
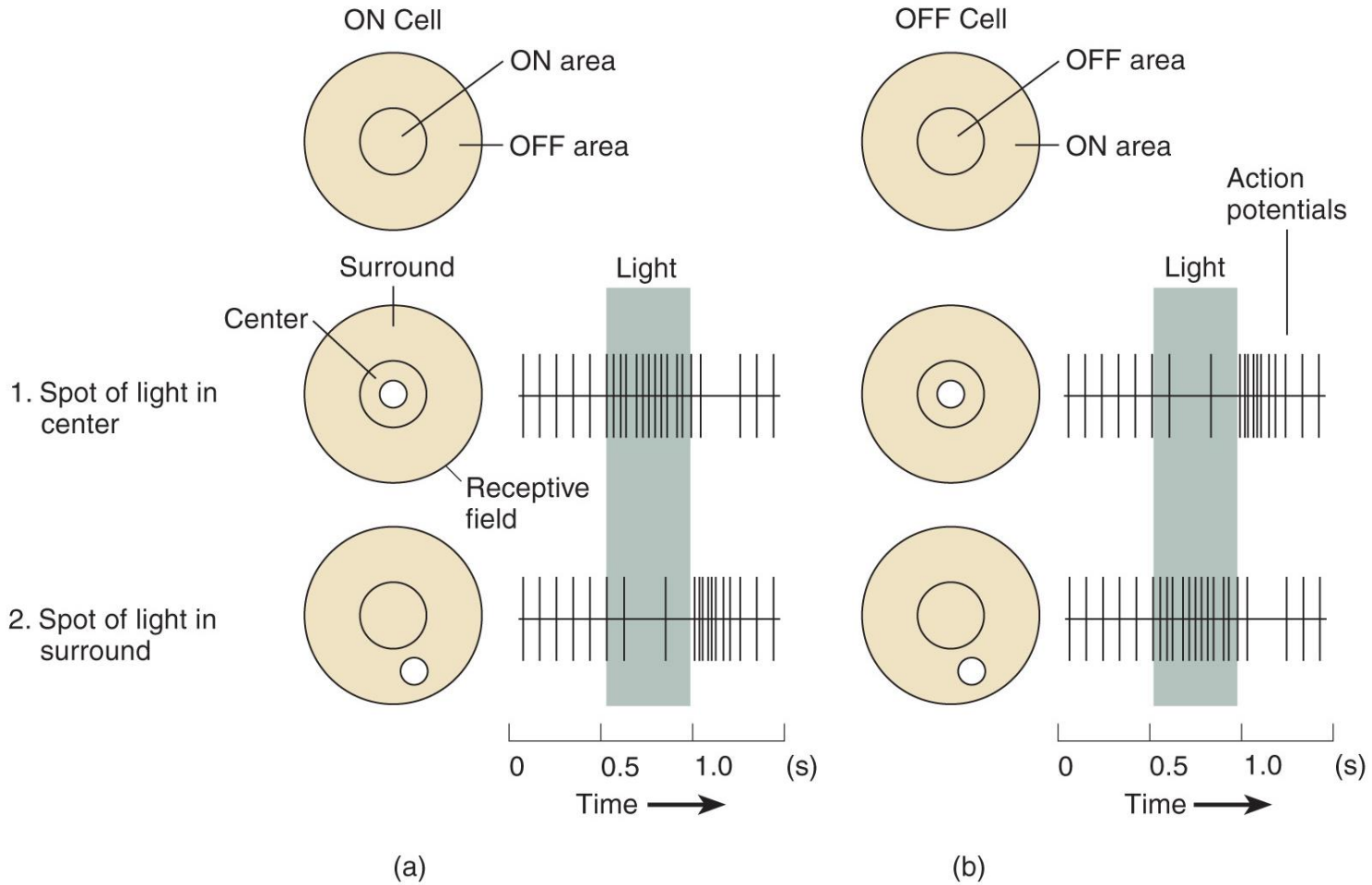


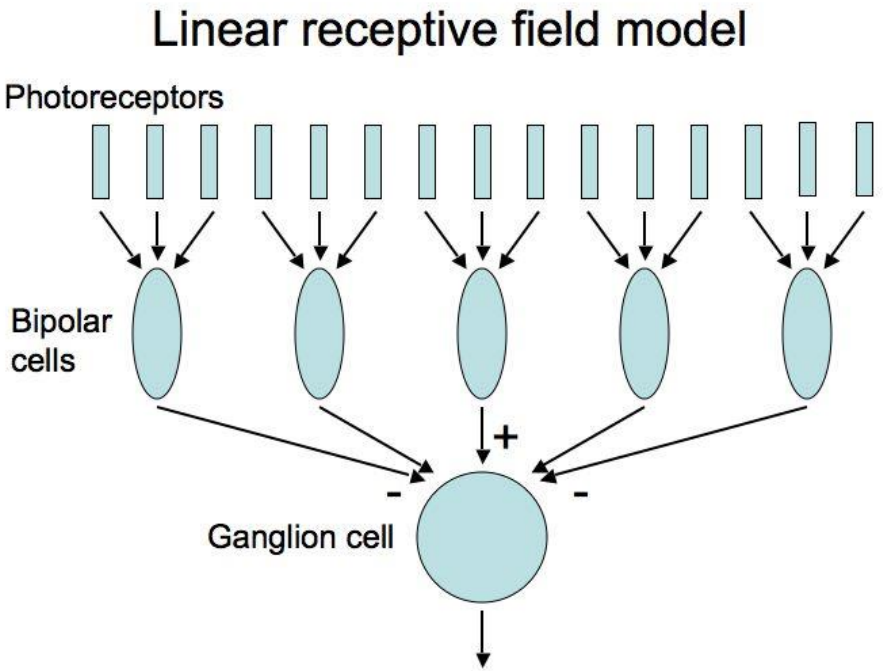
Figure ground perception

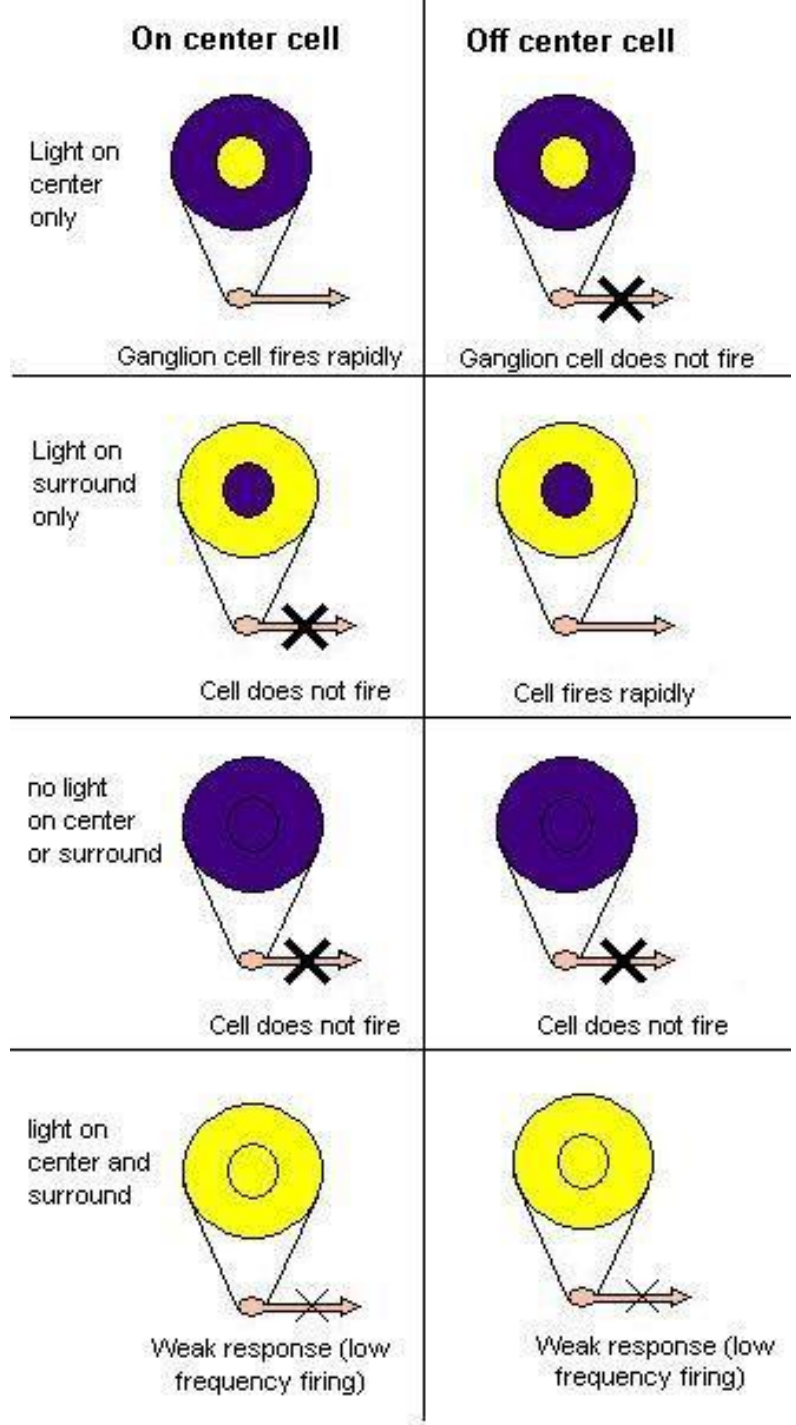


Coding light and dark

Retinal ganglion cells (circular receptive fields)






On center cell
Off center cell
Light on center only
Ganglion cell fires rapidly
Ganglion cell does not fire
Light on surround only
Cell does not fire
Cell fires rapidly
no light on center or surround
Cell does not fire
Cell does not fire
light on center and surround
Weak response (low frequency firing)
Weak response (low frequency firing)

Retinal ganglion cells respond to edges

Input image
(cornea)

"Neural image"
(retinal ganglion cells)

Center-surround receptive fields: emphasize edges.

Edge detection

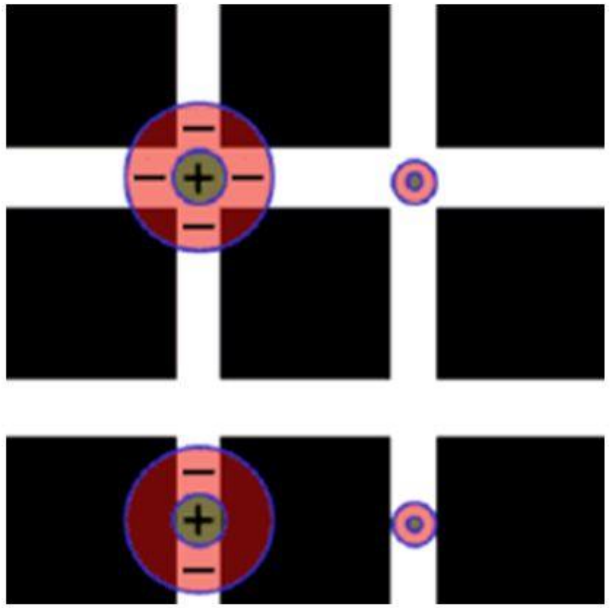
http://www.cns.nyu.edu/~david/courses/perception/lecturenotes/ganglion/ganglion.html

Hermann Grid Illusion is Due to Lateral Inhibition?

We see phantom dark spots at the intersections to the side of the focus of vision because .....

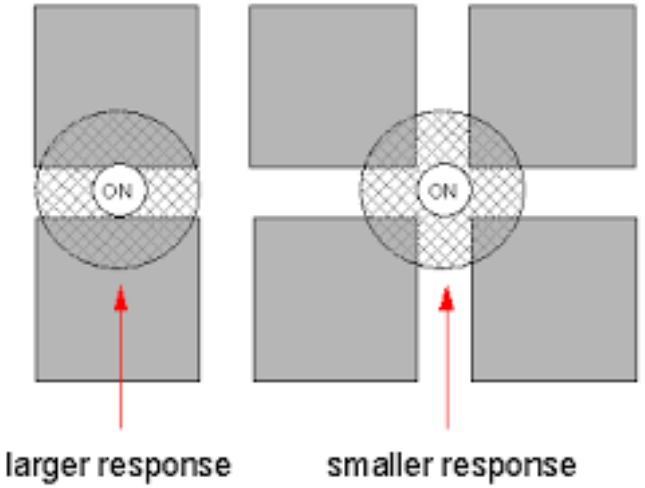
There is more lateral inhibition at the intersection than in the middle of a white bar.

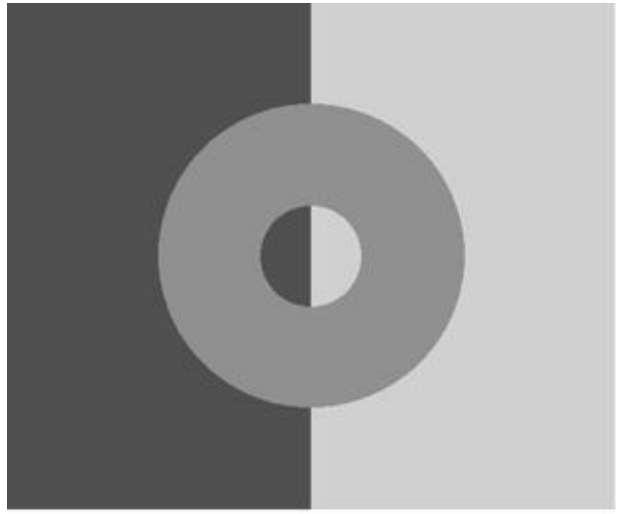
More inhibition from surrounding cells



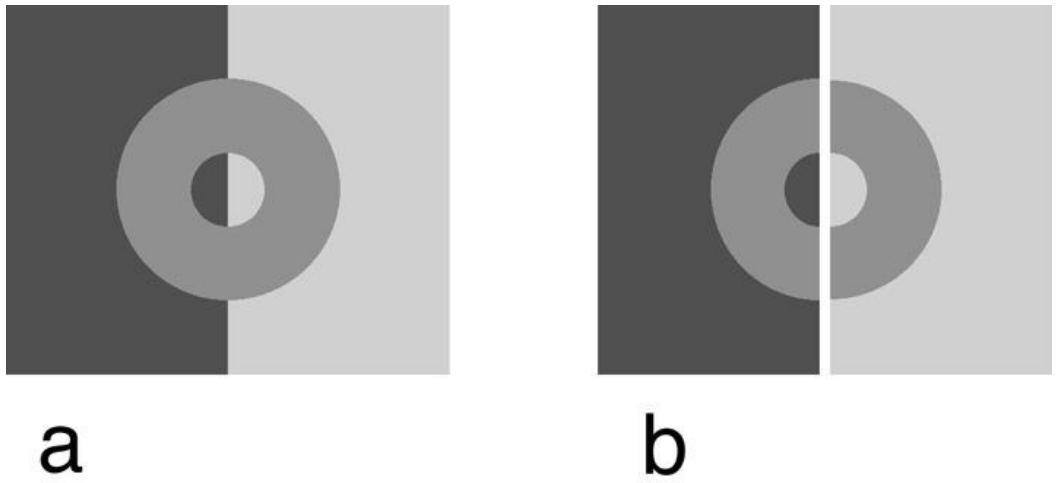
Less inhibition from surrounding cells

Receptive fields are smaller in the fovea (center of vision) than away from the fovea.

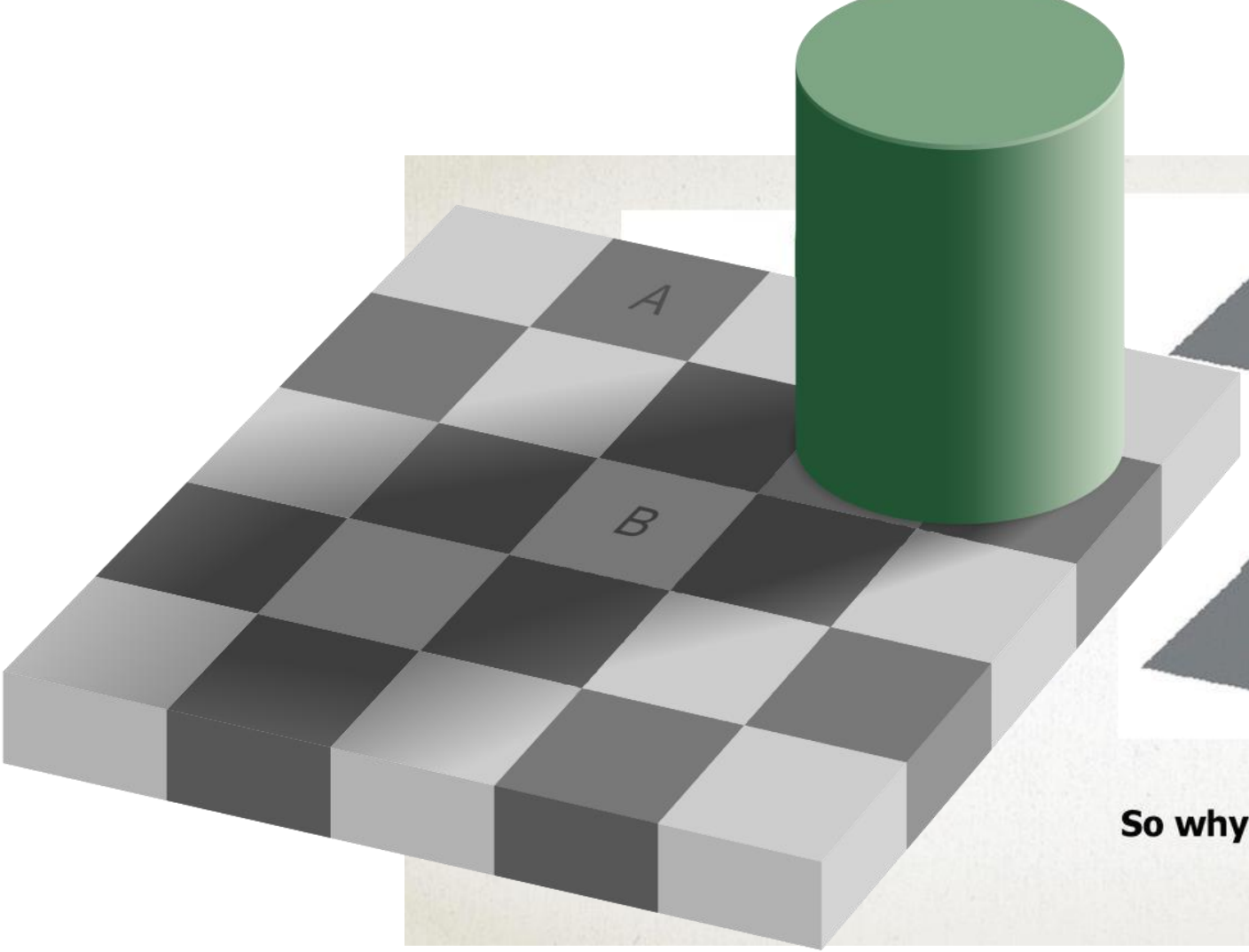


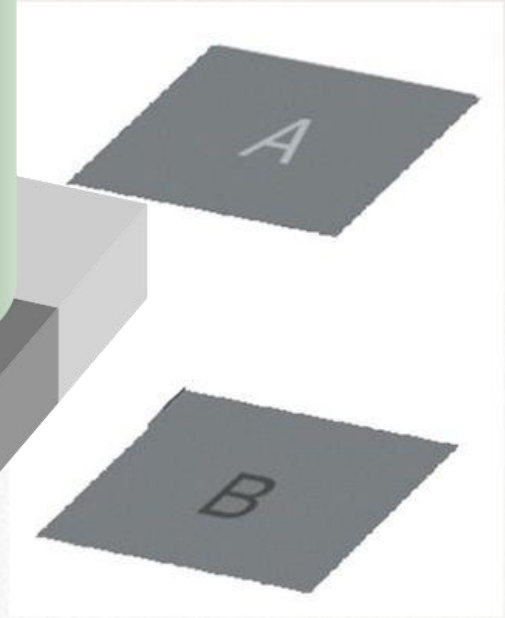


a

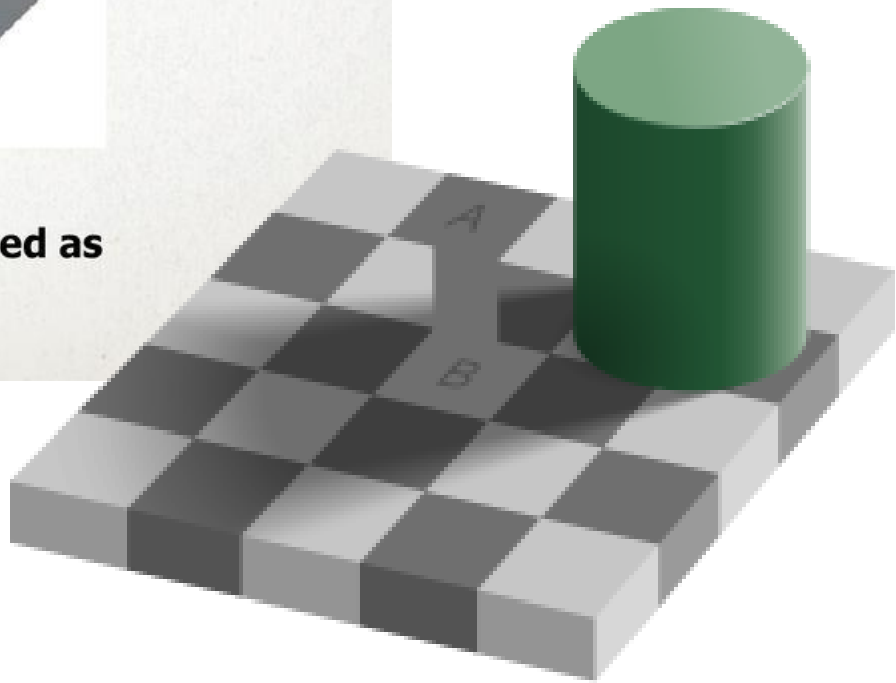


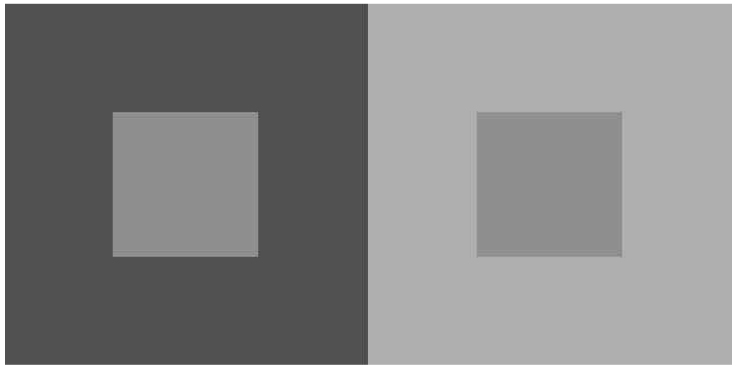
Variants on the Koffka ring.
(a) The ring appears almost uniform.
(b) When split, the two half-rings appear distinctly different.
(c) When shifted, the two half-rings appear quite different.



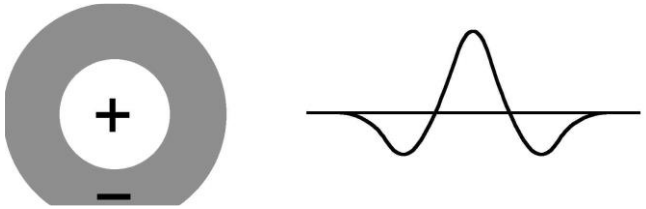


So why is B perceived as lighter?

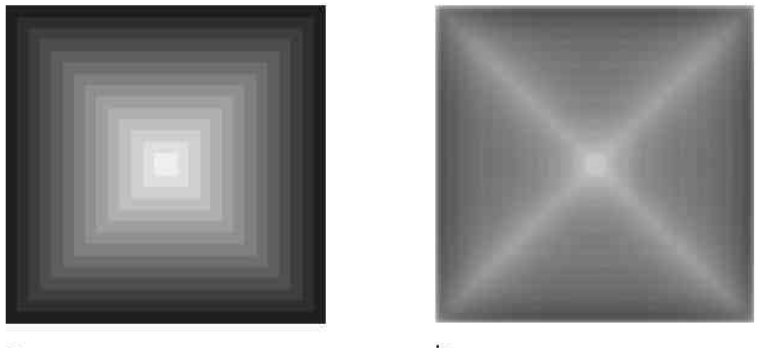




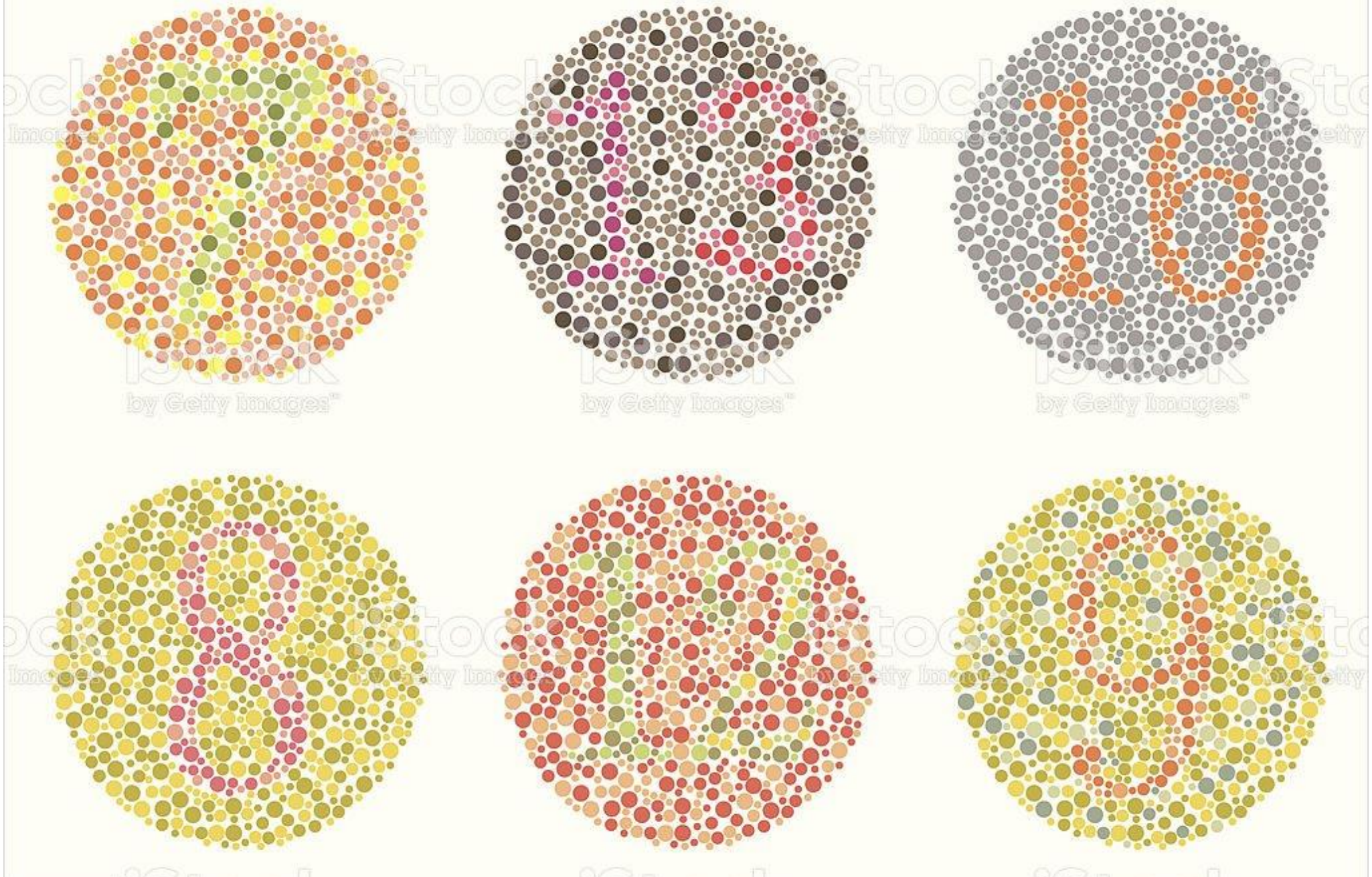
Simultaneous contrast

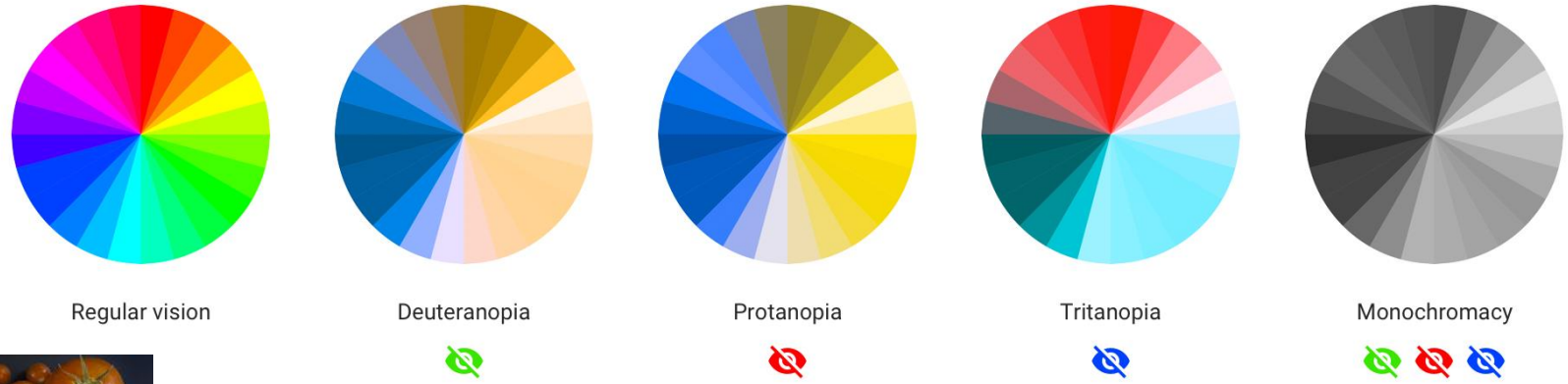


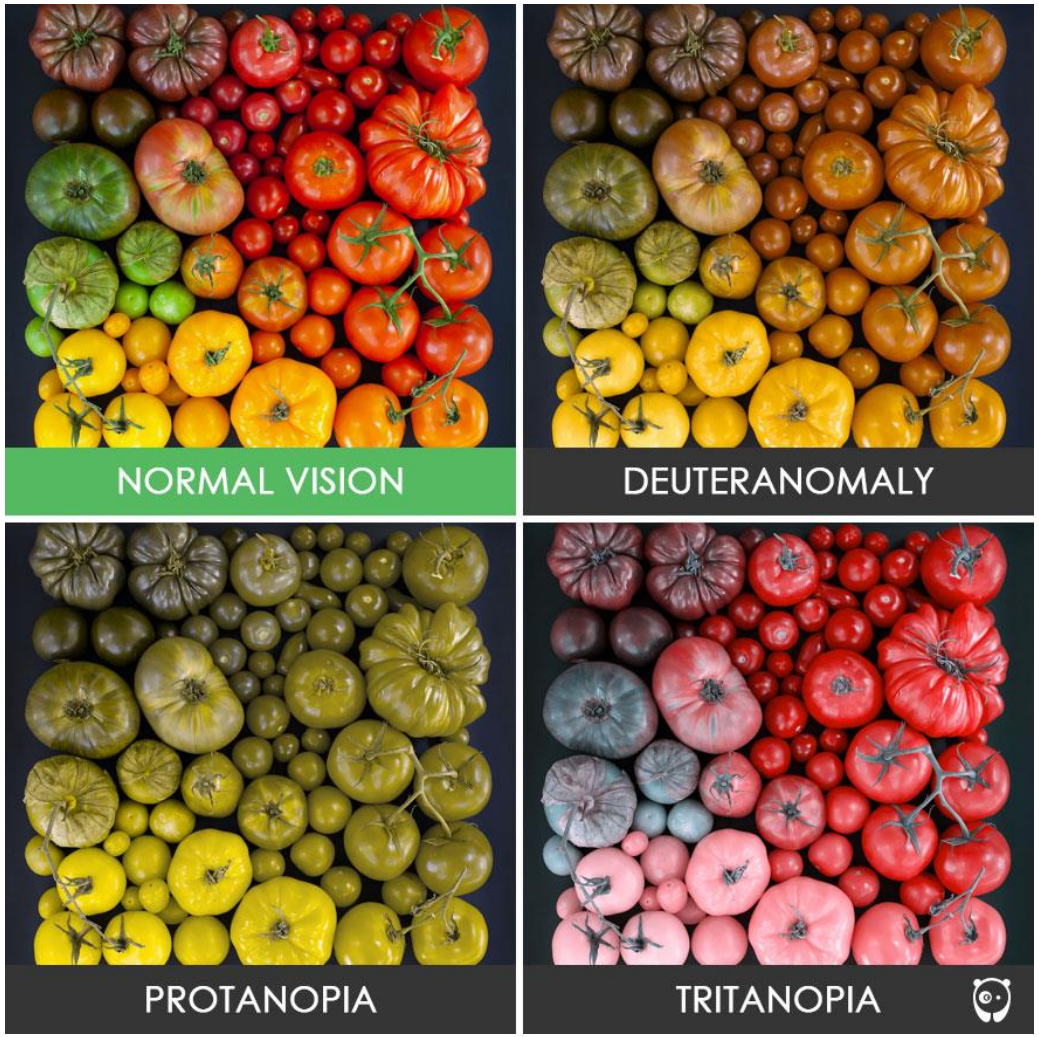
Center-on, surround-inhibit

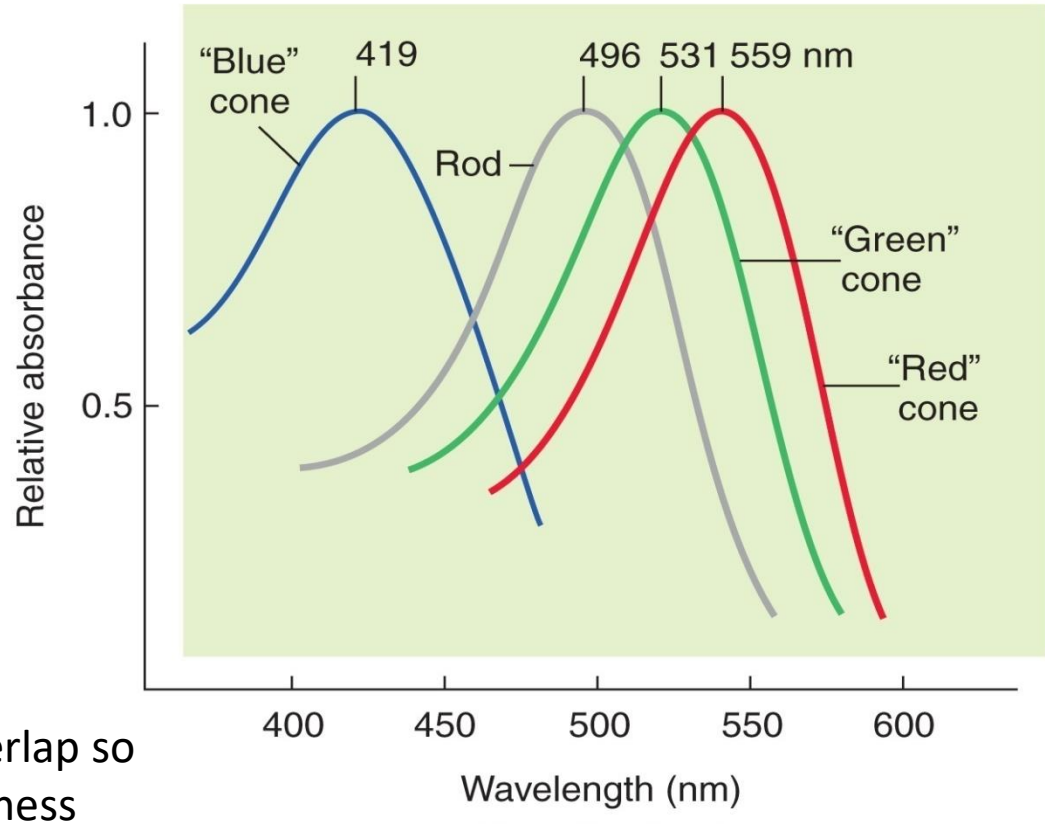


Band pass filter


iStock
by Getty Images
470424759



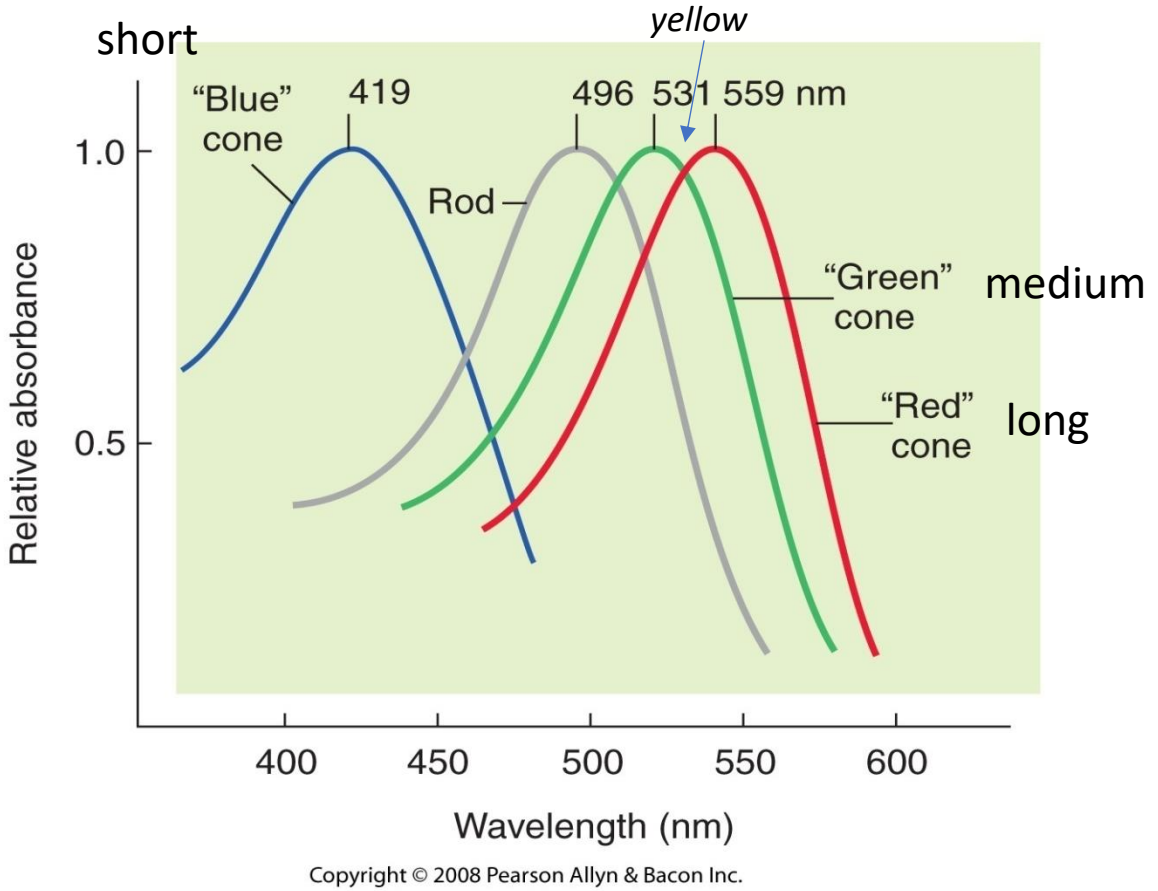


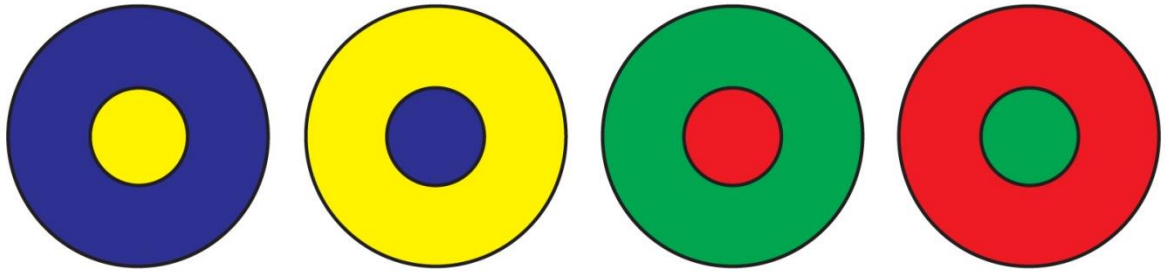


Red and green wavelengths overlap so their color blindness effects can be similar

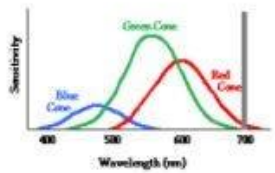
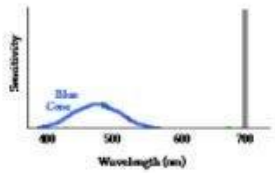
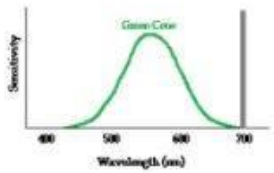
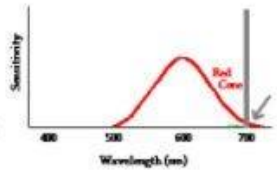
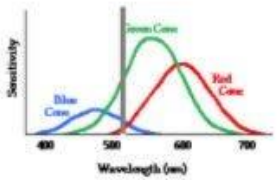
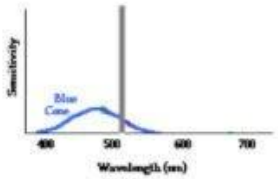
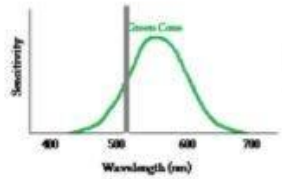
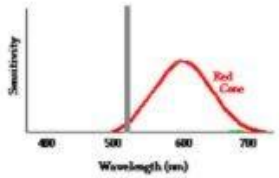
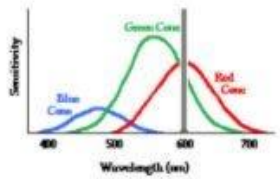
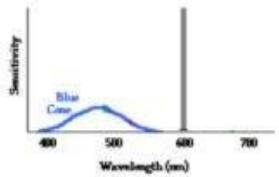
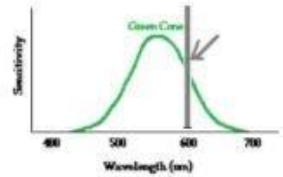
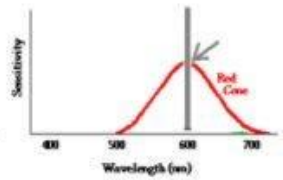
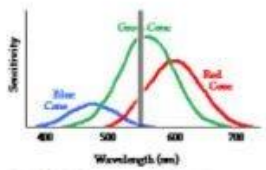
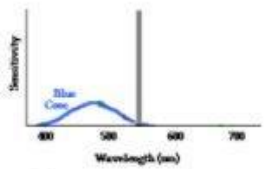
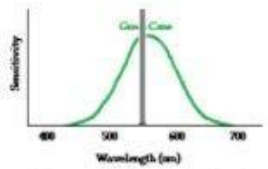
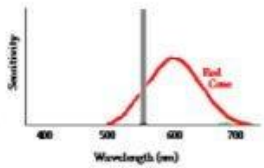
Color Coding

Retinal ganglion cells:
Opponent process coding

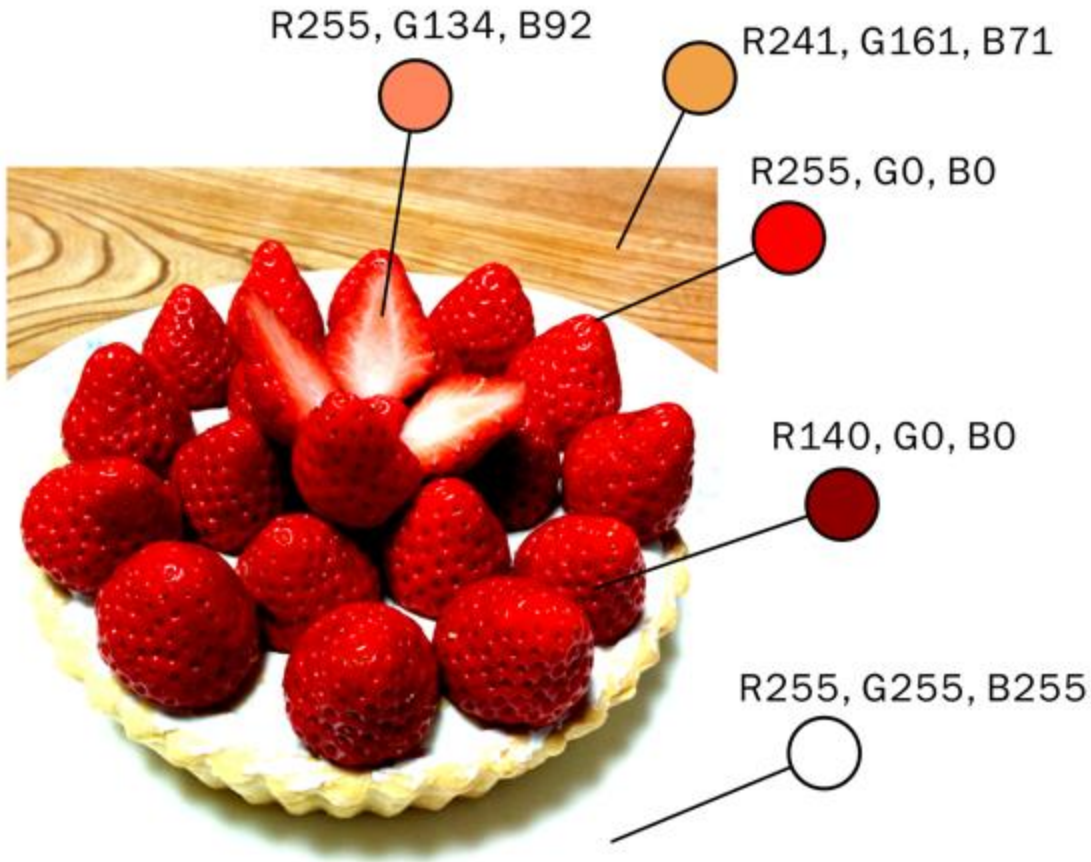




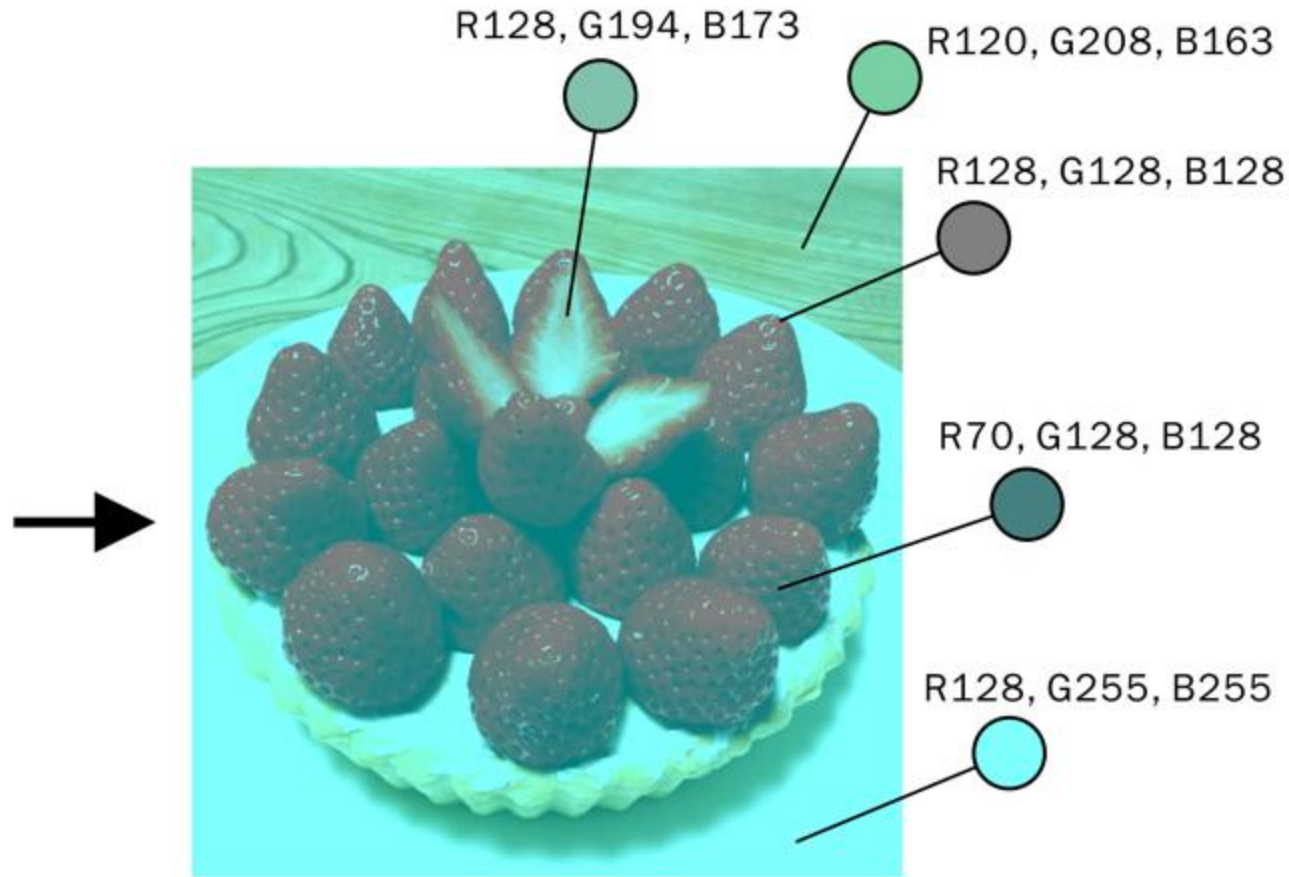
Let's do some color perception examples using the graph:

Wavelength of light shining at your eye	What the Blue Cone detects	What the Green Cone detects	What the Red Cone detects	What color the brain perceives
 A 700 nm light is shining (gray bar)	 The Blue Cone doesn't see it	 The Green Cone doesn't see it	 The Red Cone has a small signal	Only the Red Cone detected anything, so it must be **Red**
 A light of 510 nm (gray bar)	 Blue Cone detects some signal	 Green Cone has medium signal	 Red cone has a tiny signal	Blue and Red Cones detected a little, and Green detected a medium amount, so the color must be **Green**
 A 600 nm light (gray bar)	 The Blue Cone can't see it	 The Green Cone has a medium signal	 The Red Cone has a big signal	It's a lot of Red Cone, a medium amount of Green Cone, and no Blue Cone, so it must be **Orange**
 A 550 nm light (gray bar)	 Blue Cone detects a small signal	 Green Cone detects a large signal	 Red Cone detects a medium signal	It's a lot of Green Cone, a little bit Blue, and a medium amount of Red, so it must be **Yellow**

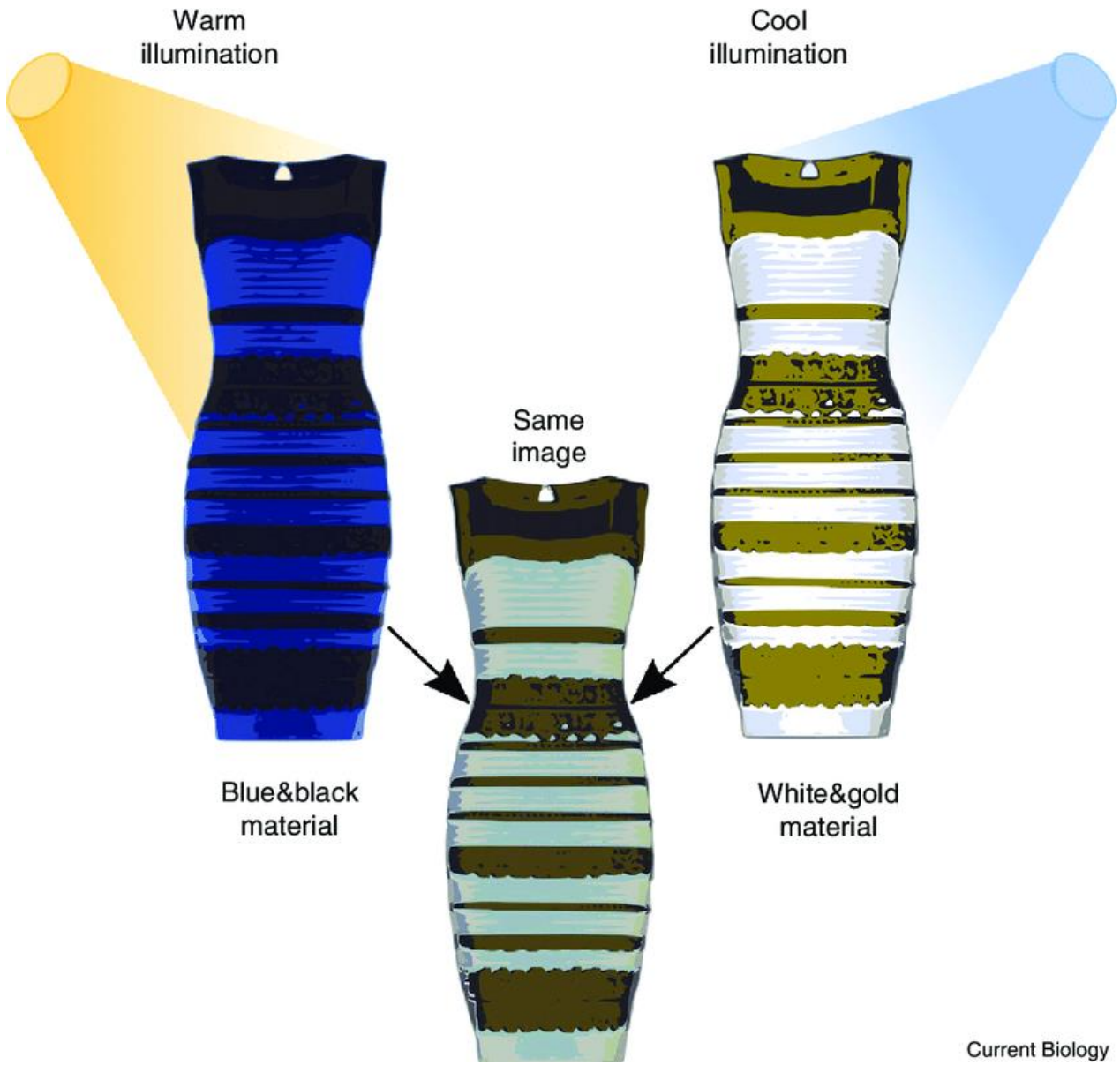
Yellowish green



Original image

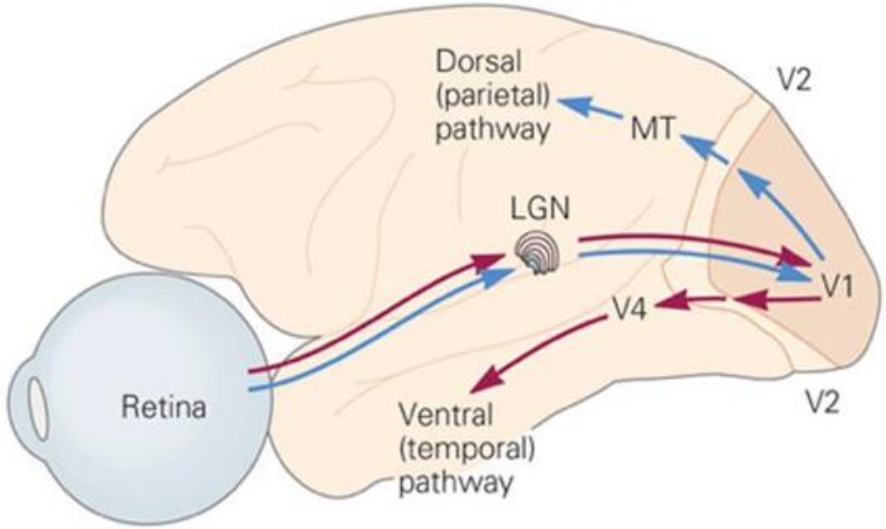


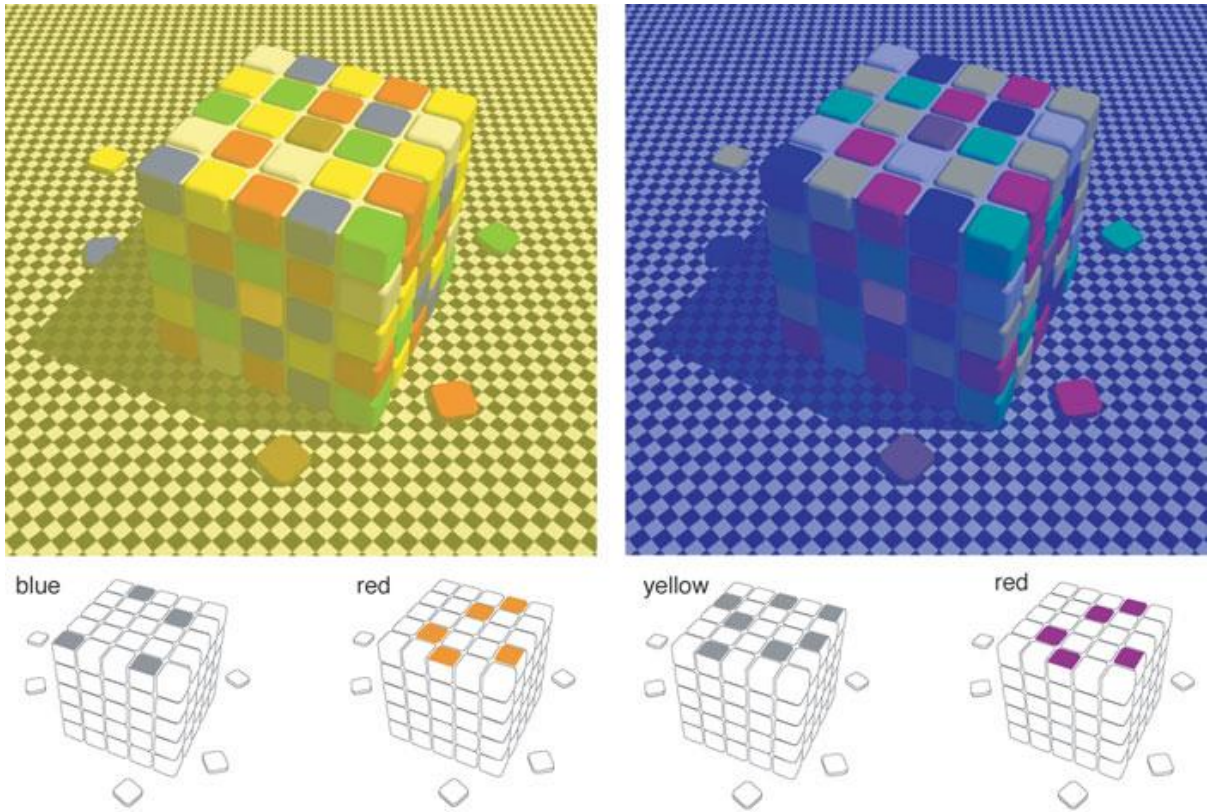
Additive color change
with cyan 50% (nonlinear model)


Warm
illumination
Cool
illumination
Same
image
Blue&black
material
White&gold
material
Current Biology


NBC

Color constancy



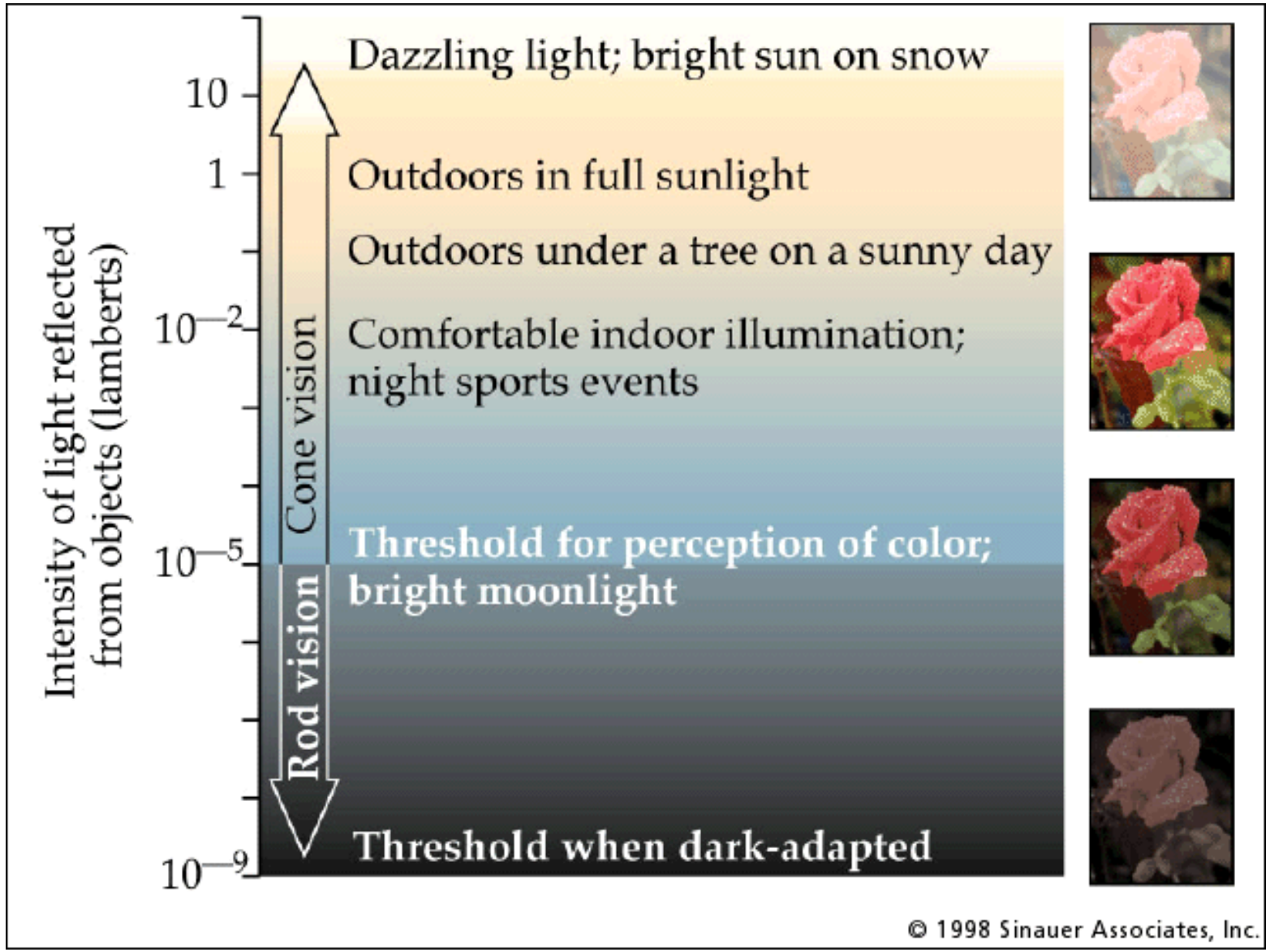


The relative constant appearance of the colors of objects viewed under varying lighting conditions

- Area V4 → perception of color and color constancy.
- Lesion in V4 area → Achromatopsia i.e. vision without color

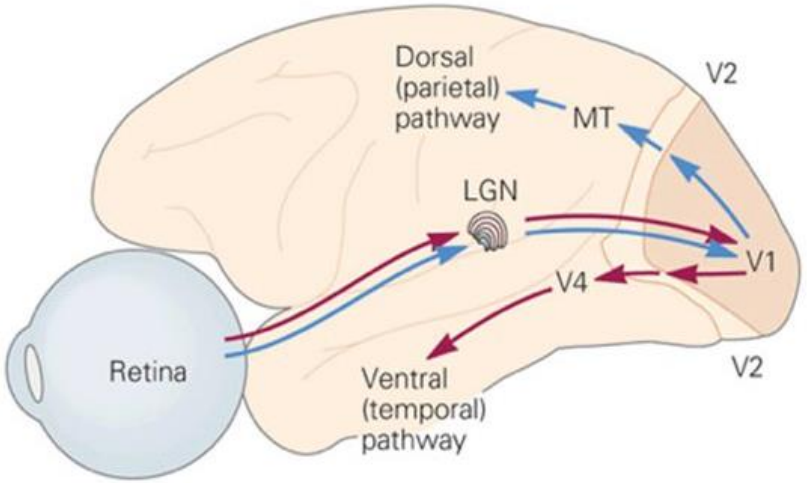

ORIGINAL


ORIGINAL

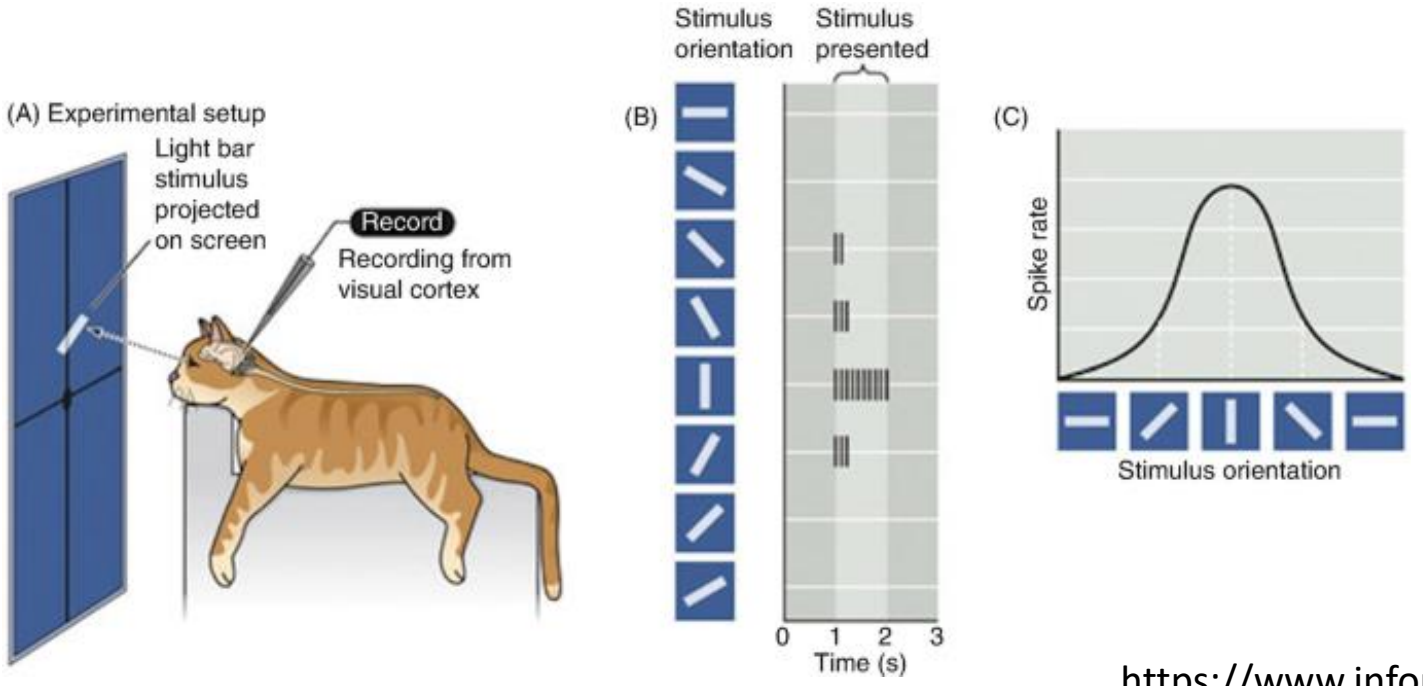

Intensity of light reflected from objects (lamberts)
10
1
10⁻²
10⁻⁵
10⁻⁹
Cone vision
Rod vision
Dazzling light; bright sun on snow
Outdoors in full sunlight
Outdoors under a tree on a sunny day
Comfortable indoor illumination; night sports events
Threshold for perception of color; bright moonlight
Threshold when dark-adapted
© 1998 Sinauer Associates, Inc.

Class 5
Vision

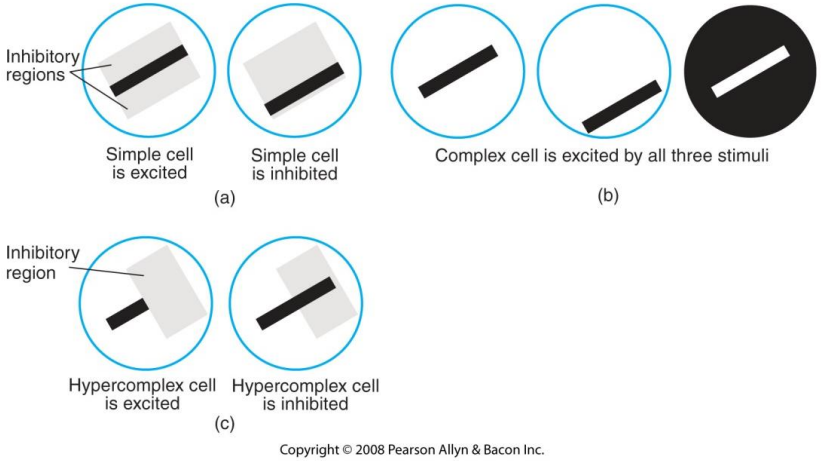
Orientation and movement (Area V1)



Hubel and Wiesel, 1959



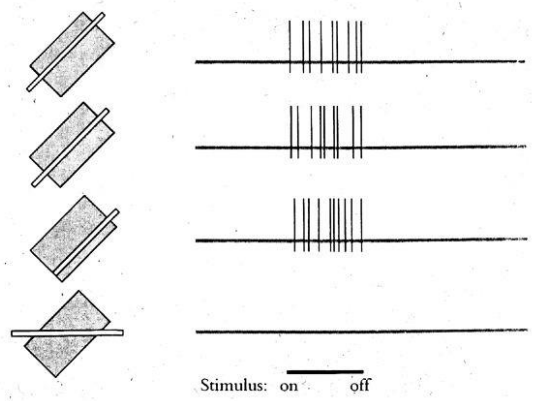
https://www.informit.com/articles/article.aspx?p=1431818



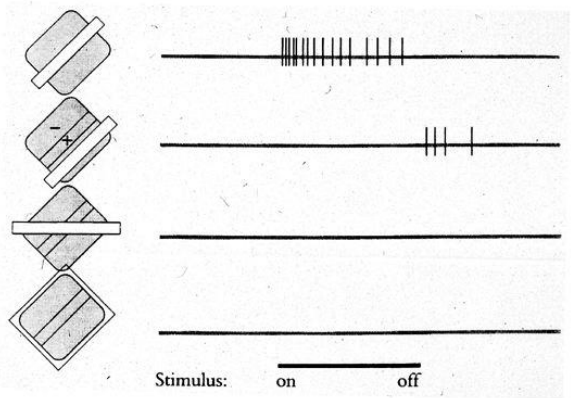
- **Simple cell**: sensitive to particular orientation of light
- **Complex cell**: sensitive to orientation and motion of light in a particular direction, esp. perpendicular to the orientation

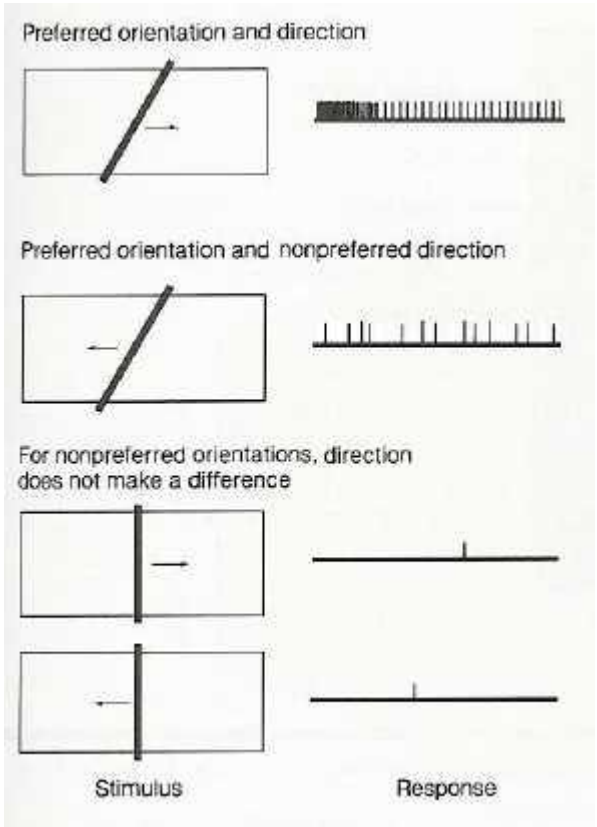
  (movement detectors)
- **Hypercomplex cell**: sensitive to orientation and end of line

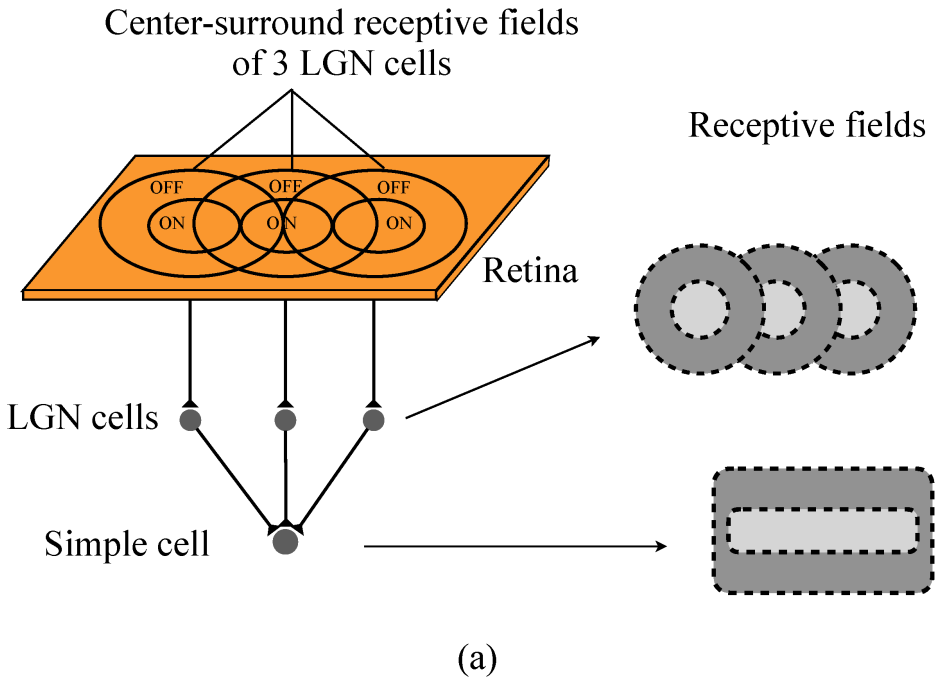
Complex cell



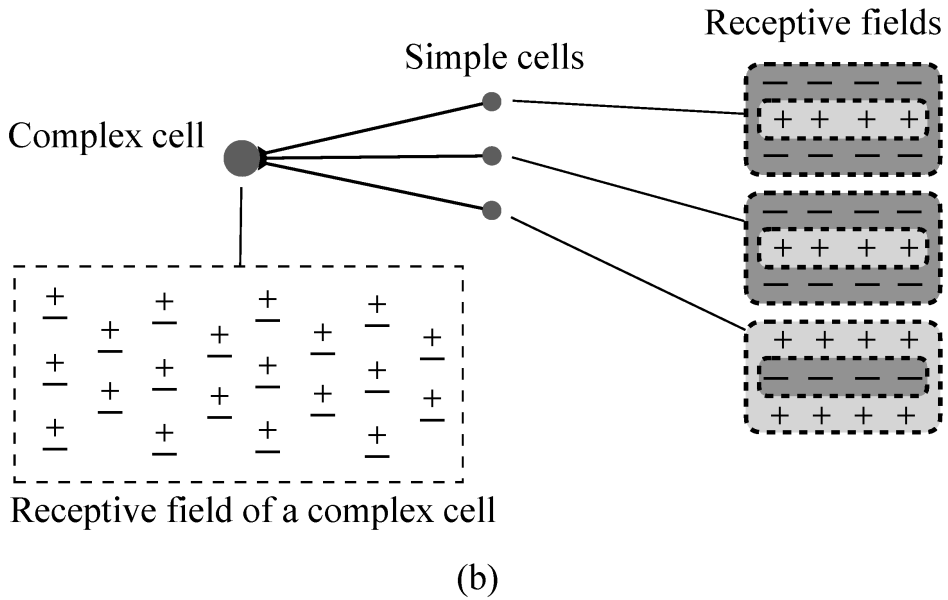
Simple cell







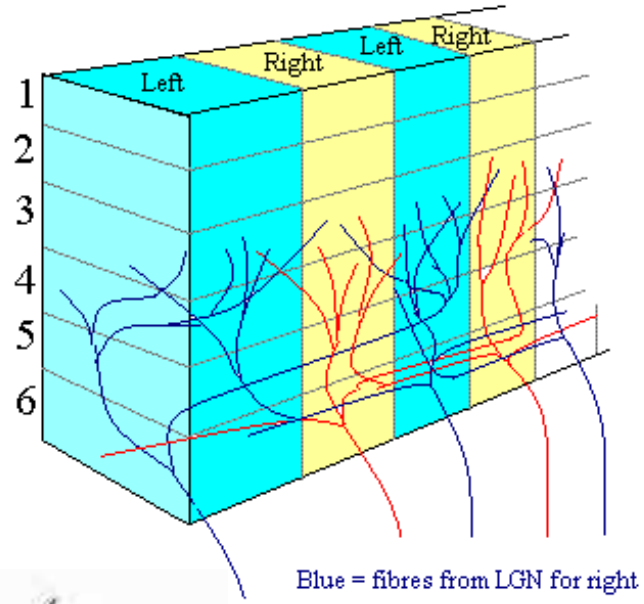
(a)



(b)

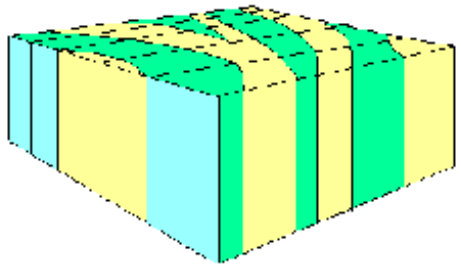
https://www.mdpi.com/2076-3425/12/4/470

Cortical Columns

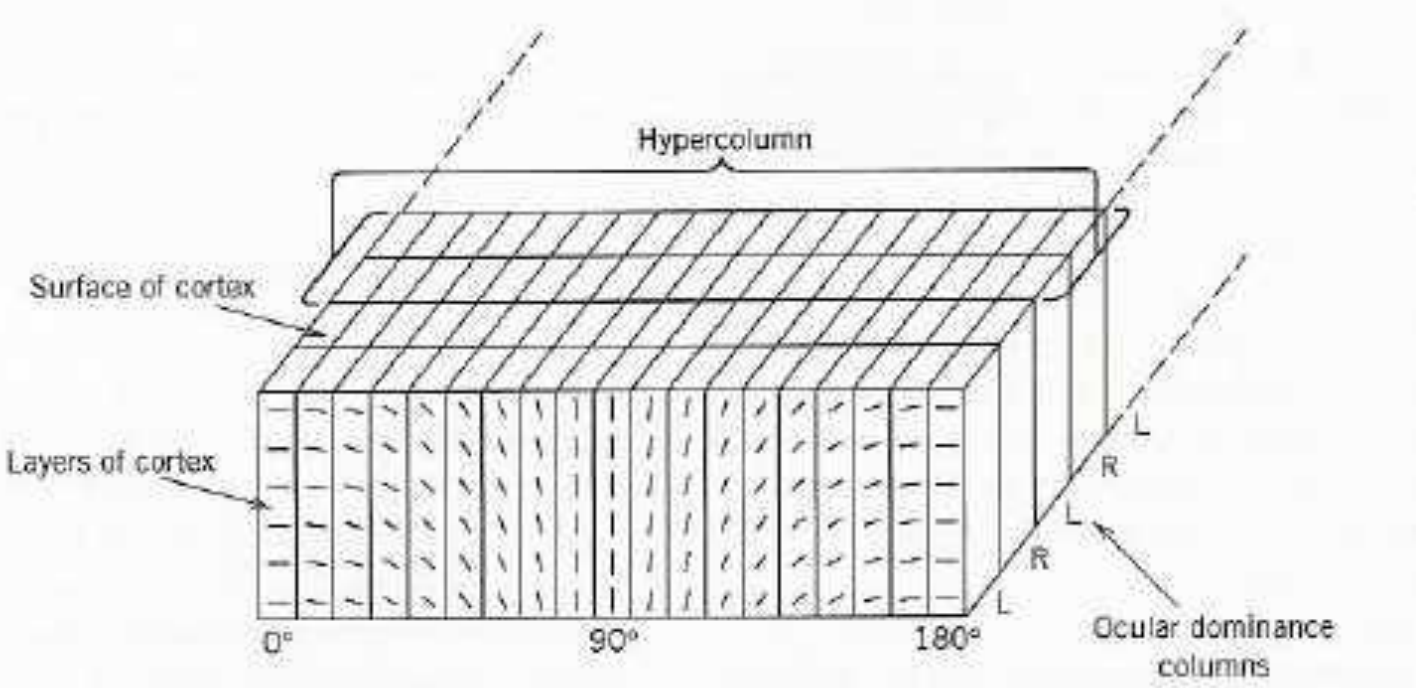


Blue = fibres from LGN for right eye

Red = fibres from LGN for left eye

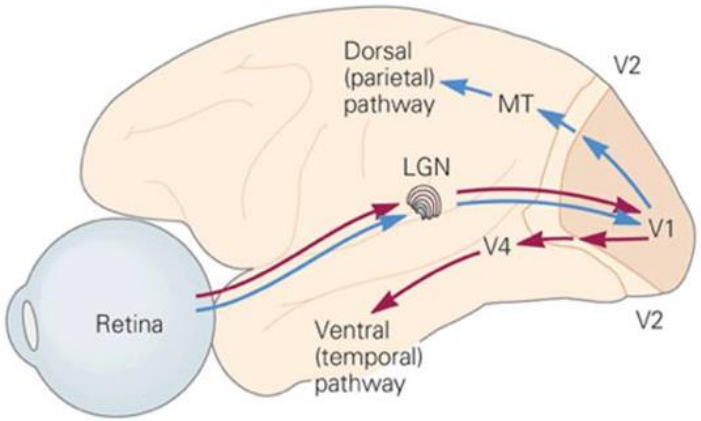


The columns are rather disorderly on the cortex and form a pattern like a finger print.



Columns of cells with different orientation preferences

Spatial frequency



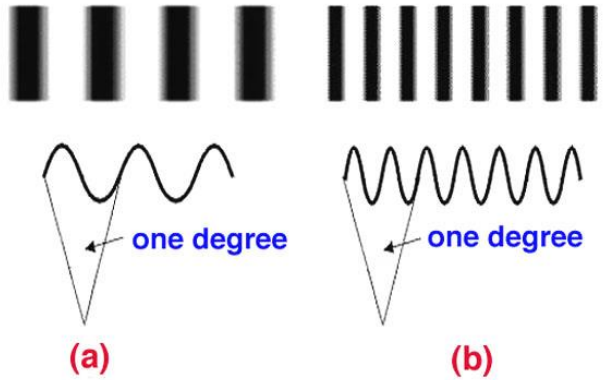


Figure 22. Spatial frequency is a measure of the number of cycles subtended at the eye per degree. (a) One cycle per degree. (b) Two cycles per degree.

Spatial frequency processing occurs in V1 (primary visual cortex)

Note: physical distance in visual physics is measured in units of visual angle. Cycles per degree of visual angle constitute spatial frequency

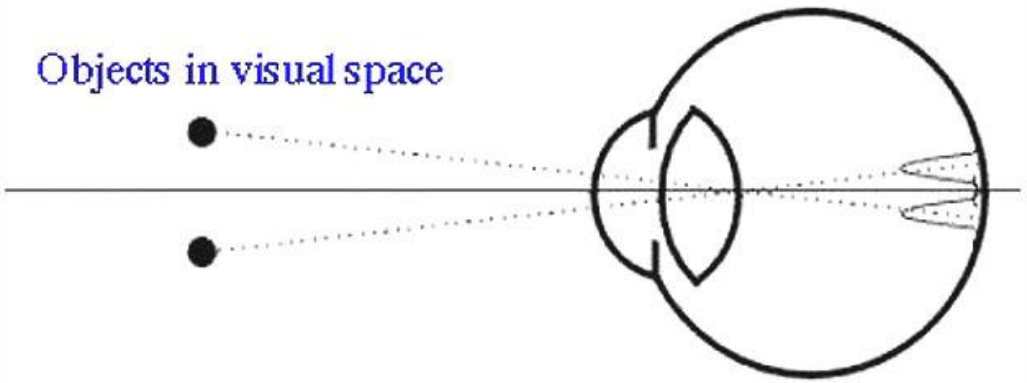


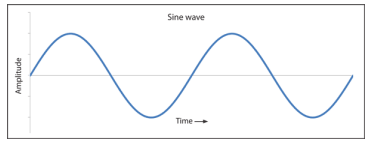
Figure 10. Two point sources and their point spread function at the back of the eye.

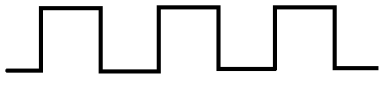
Object is too close to eyes

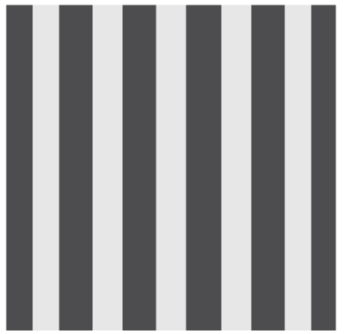
Object is far from eyes

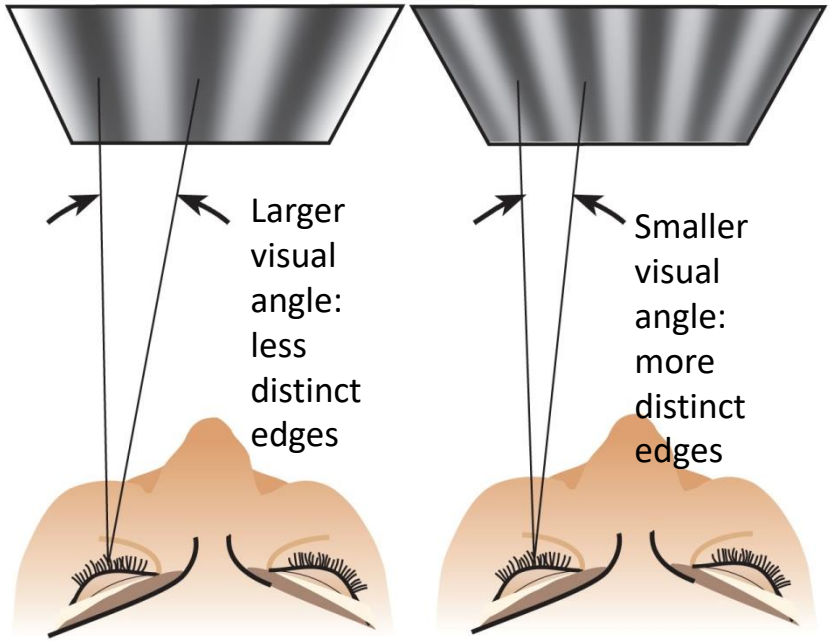
Sine wave grating

Square wave grating









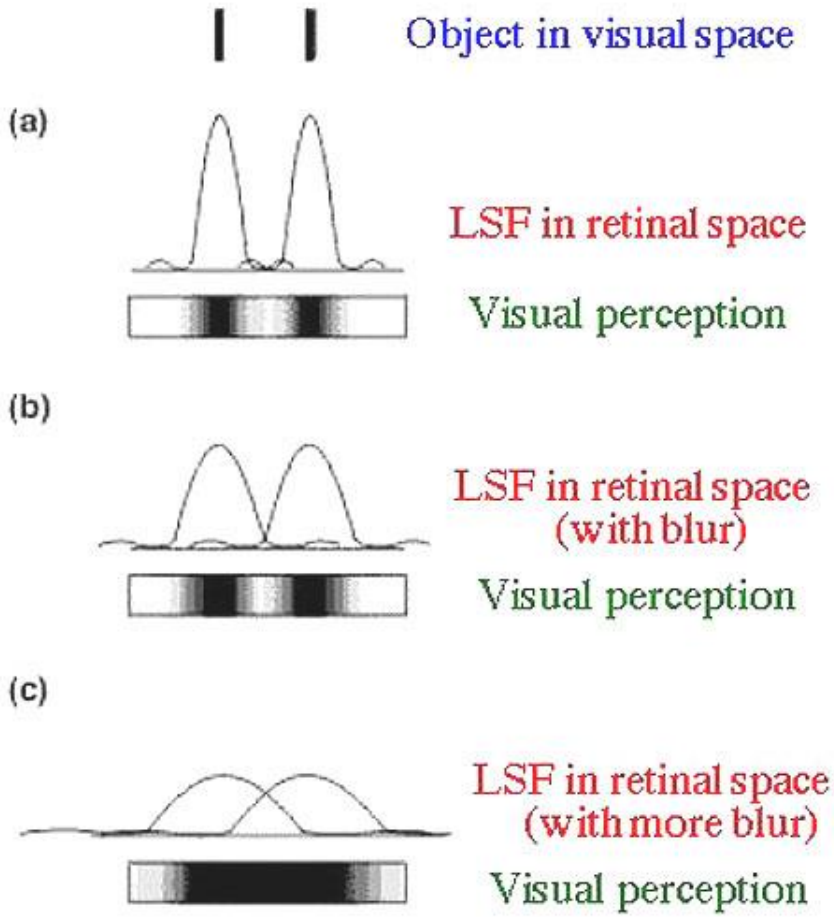


Figure 16. Line spread function (LSF) of two lines with varying amounts of blur. With increasing blur, the discrimination of the two lines is lost.

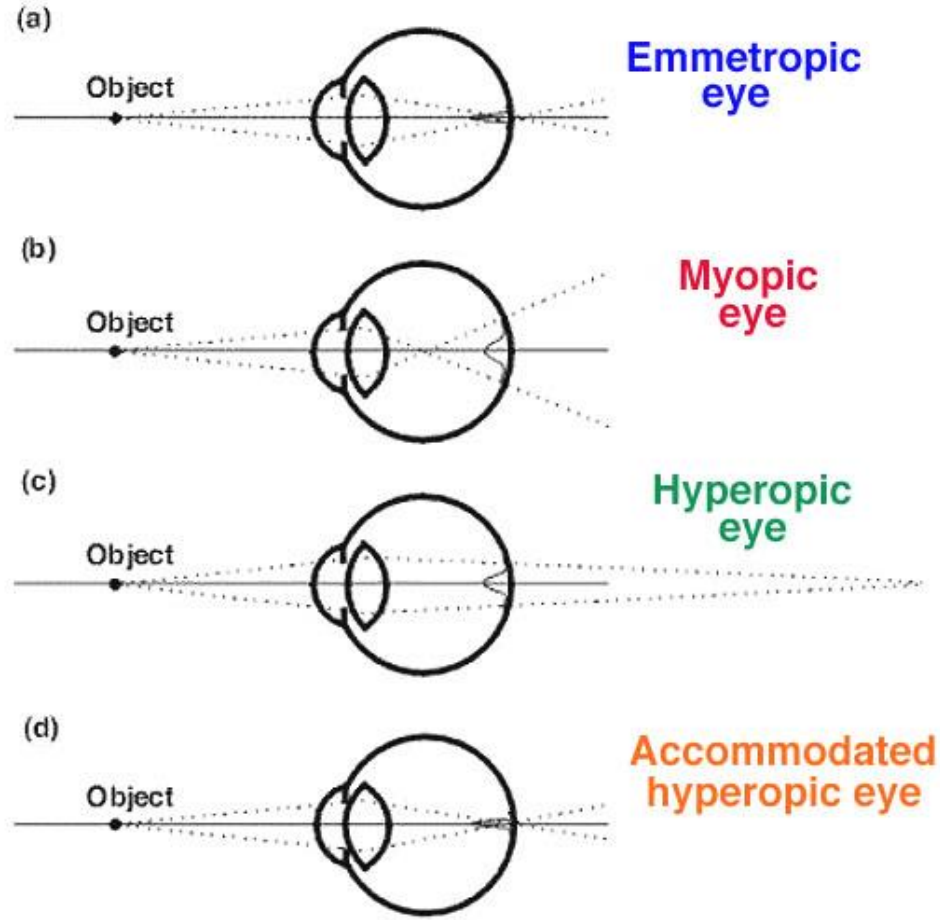
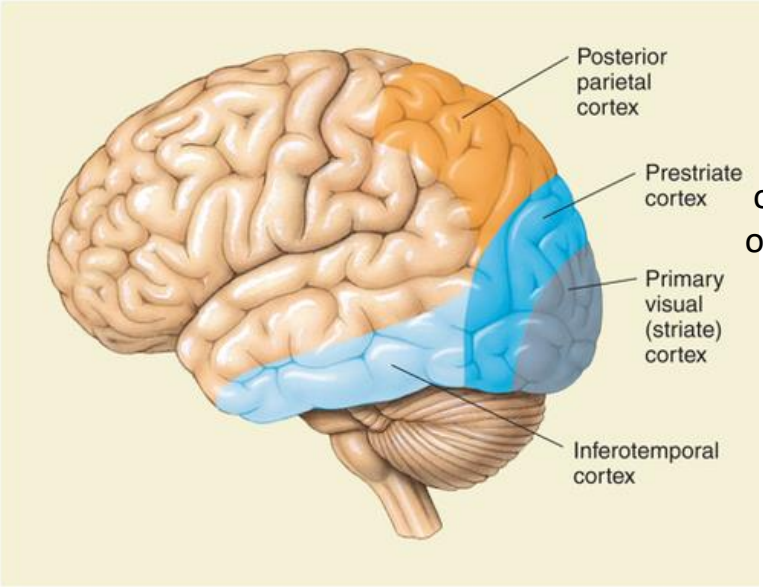


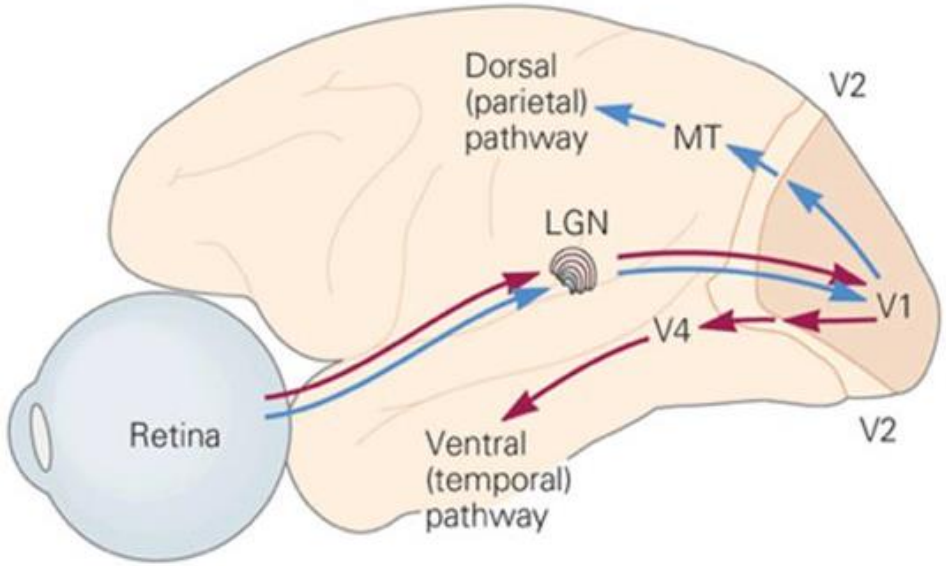
Figure 17. Point spread function at the back of the eye with different refractive errors.

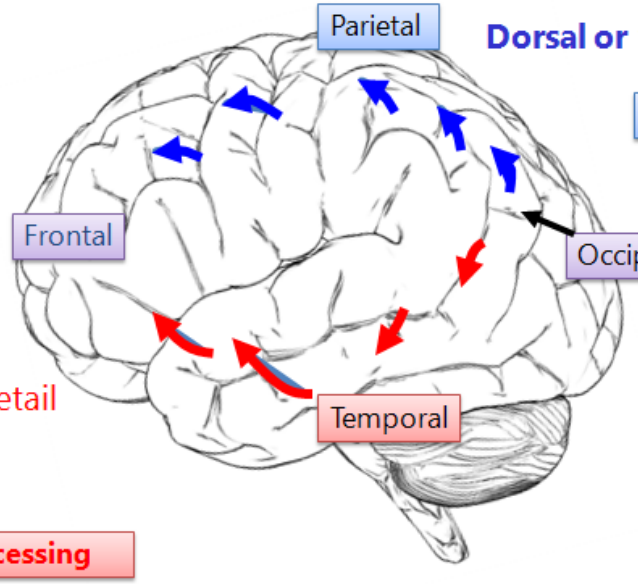
https://webvision.med.utah.edu/book/part-viii-psychophysics-of-vision/visual-acuity/

Visual pathway



or Extrastriate cortex
or Visual Association cortex





Dorsal or "where" stream

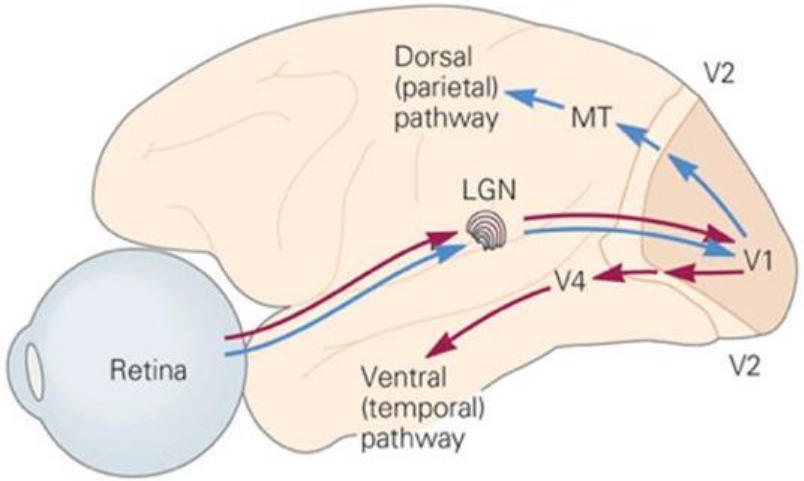
Spatial processing

location
movement
spatial transformations
spatial relations

color
texture
pictorial detail
shape
size

Object processing

Ventral or "what" stream



Dorsal Stream

middle temporal (MT)

medial superior temporal (MST)

V2
(thick stripes)
- Speed
- Direction
- Spatial frequency
- Temporal frequency

MT
- Speed
- Direction
- Spatial frequency
- Temporal frequency
- Local/Global motion

MST
- Expansions
- Contractions
- Rotations
- Translations
- Spirals
- Optic Flow

Parietal Regions
- Heading
- Optic flow
- Self motion
- Multi-modal integration

Primary Visual (Striate) Cortex

V1
- Speed
- Direction
- Spatial frequency
- Temporal frequency
- Orientation
- Color

Extrastriate cortex
(Second level of visual processing)

(Inferior temporal Cortex)

V2
(thin stripes, interstripes)
-Edges
-Illusory edges
- Border ownership
- Color

V4
- Angles
- Curvature
- Perceived color
- Kinetic contours
- Motion

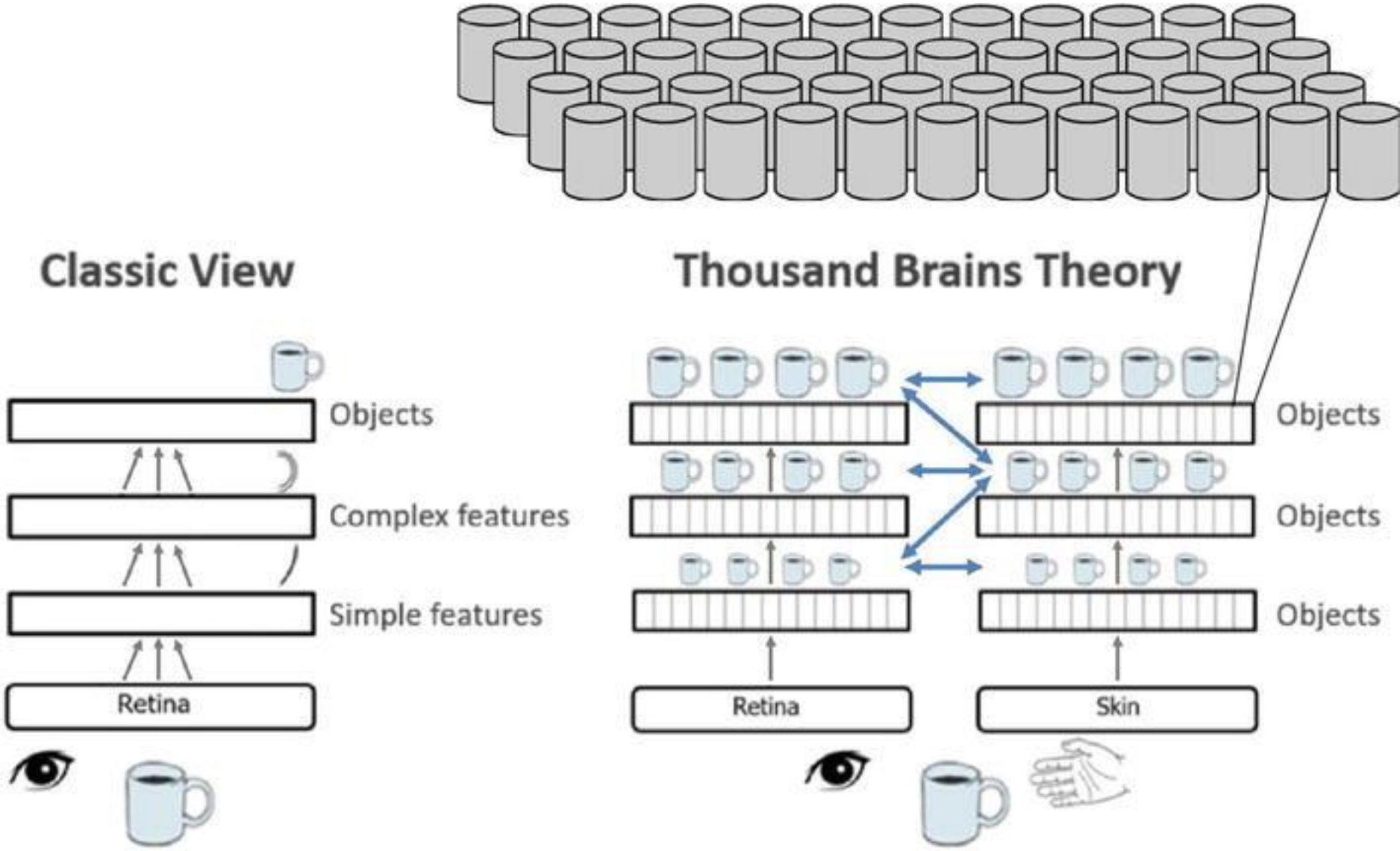
TEO/PIT
- Simple shapes

TE/AIT
- Complex shapes/ body parts
- Object recognition
- Object invariance

Ventral Stream

Visual Processing does not happen in isolation

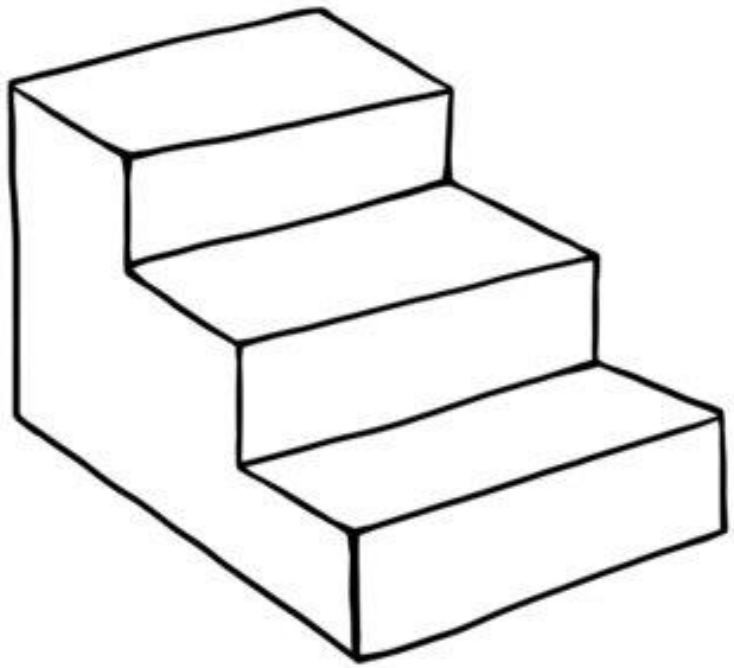
We still need neuroscientific data to confirm this theory



https://www.intechopen.com/chapters/86375

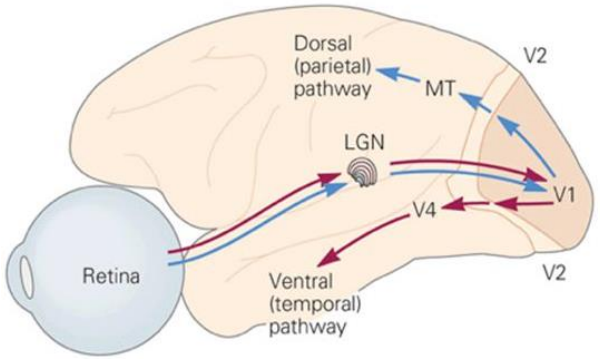
How do we resolve this conflict and see a stable perception?

How do we perceive 2D as 3D in our brain?



ERICH LESSING/ART RESOURCE

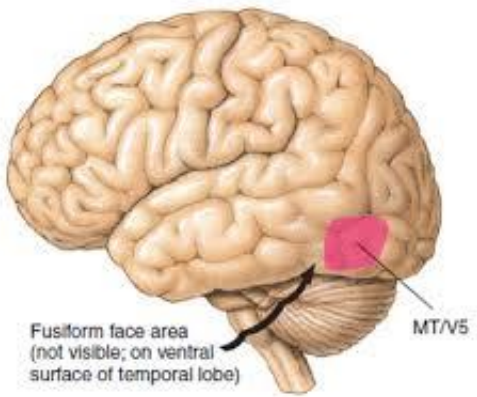
Perception of Form

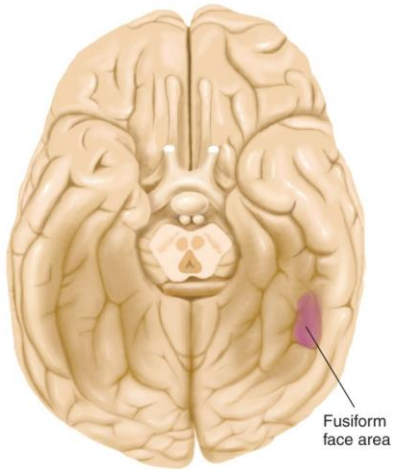


- ***Agnosia –*** *"failure to know"*

- ***Visual agnosia (damage to the ventral stream)***
  - Visual Agnosia video

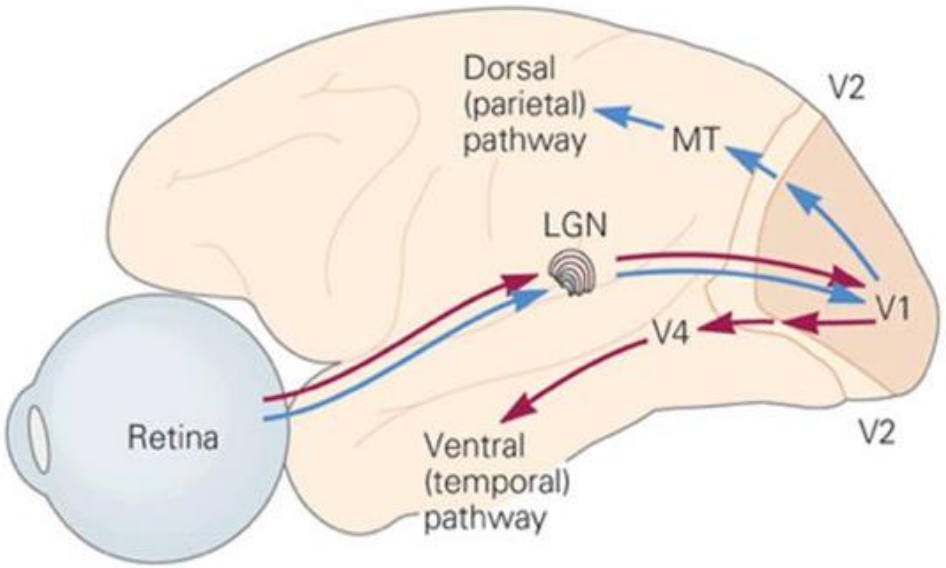
- ***Prosopagnosia (damage to fusiform area (FFA))***
  - Prosopagnosia video





Visual agnosia or prosopagnosia ?

Visual Agnosia

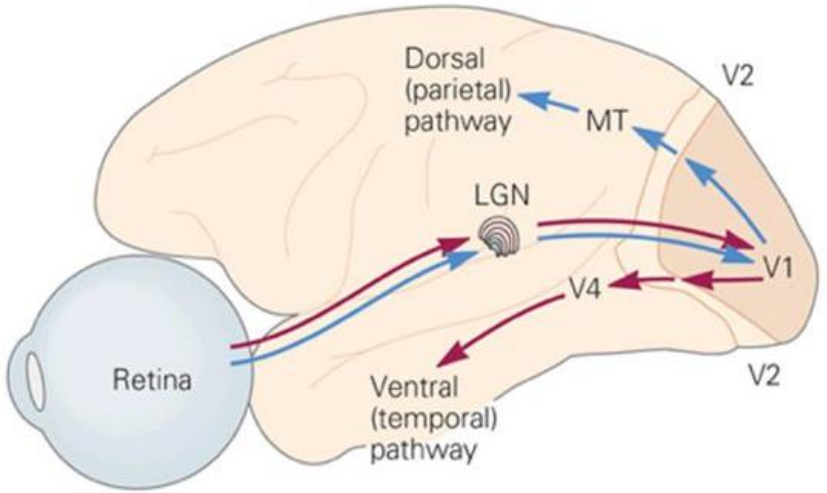


patient S. B., a 30-year-old man whose ventral stream was damaged extensively bilaterally by an infection when he was 3 years old. As a result, he was unable to recognize objects, faces, textures, or colors. However, he could perceive movement and could even catch a ball that was thrown to him. Furthermore, he could recognize other people's arm and hand movements that mimed common activities such as cutting something with a knife or brushing one's teeth, and he could recognize people he knew by their gait.

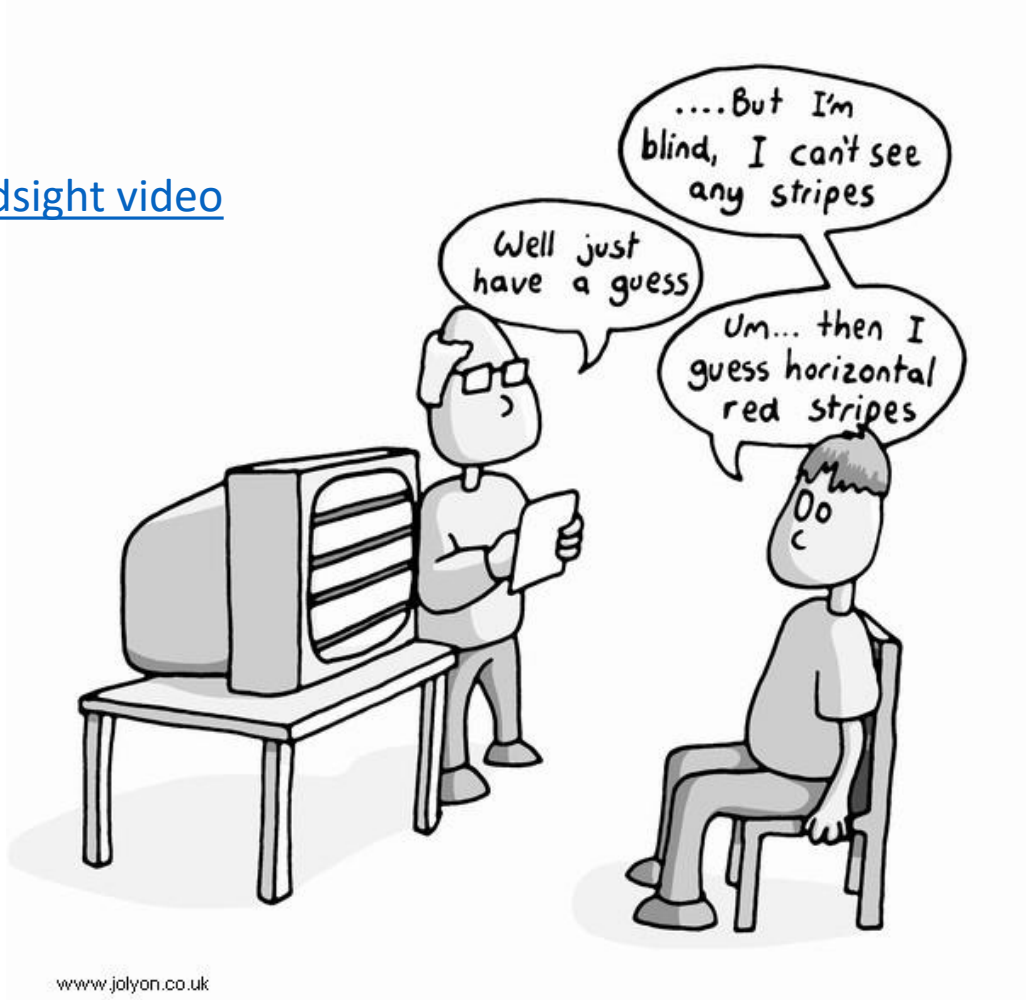
Blindsight

Perception without awareness

Damage to?



[Blindsight video]

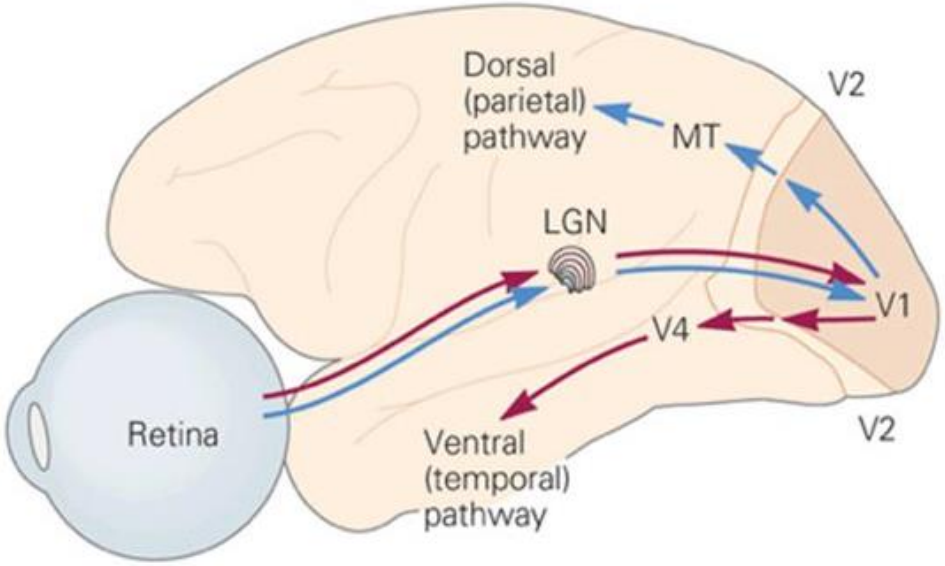


The Case of D.B., the Man Confused by His Own Blindsight

D.B. had no awareness of "seeing" in his blind left field. Despite this apparent left-field blindness, he could accurately reach for visual stimuli in his left field and could accurately differentiate between a horizontal or diagonal line in his left field if forced to "guess." When he was questioned about his vision in his left field, his most usual response was that he saw nothing. When he was shown a video of his accurate left-field performance through his good, right field, he was astonished and insisted he was just guessing.

Perception of Movement

- Area V5 (Medial temporal (MT)) – responds to movement
- Damage to V5 - ***Akinetopsia*** – inability to perceive movement
- Form from motion: ability to perceive 3D forms
  - people with visual agnosia for objects can recognize actions (motion recognition) performed by those objects but cannot recognize the objects

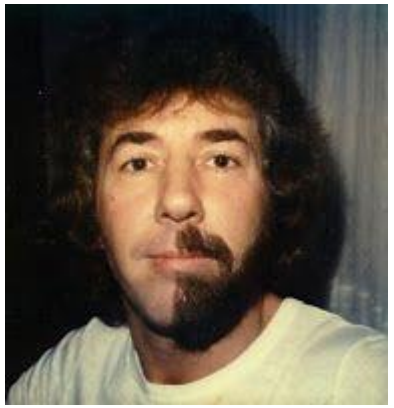


Patient L. M. had an almost total loss of movement perception. She was unable to cross a street without traffic lights because she could not judge the speed at which cars were moving. Although she could perceive movements, she found moving objects very unpleasant to look at. For example, while talking with another person, she avoided looking at the person's mouth because she found its movements very disturbing. When the investigators asked her to try to detect movements of a visual target in the laboratory, she said, "First the target is completely at rest. Then it suddenly jumps upwards and downwards" (Zihl et al., 1991, p. 22–44). She was able to see that the target was constantly changing its position, but she was unaware of any sensation of movement.

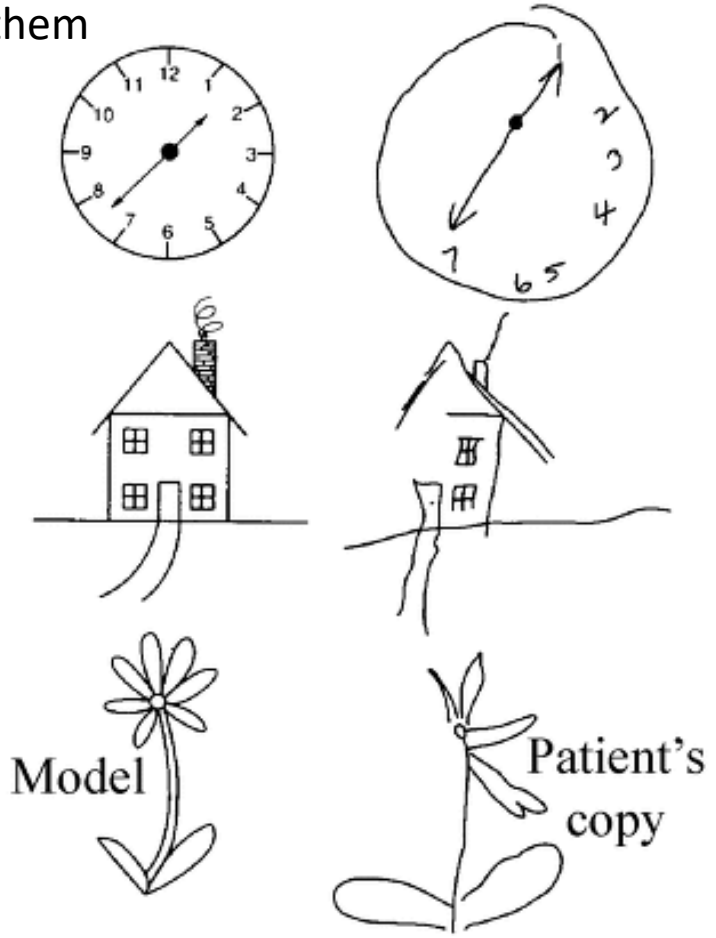
You can perceive objects but cannot act on them or interact with them

Hemispatial neglect

P.S. was presented simultaneously with two line drawings of a house, in one of which the left side was on fire. She judged that the drawings were identical; yet when asked to select which house she would prefer to live in, she reliably chose the house that was not burning.



One sided world



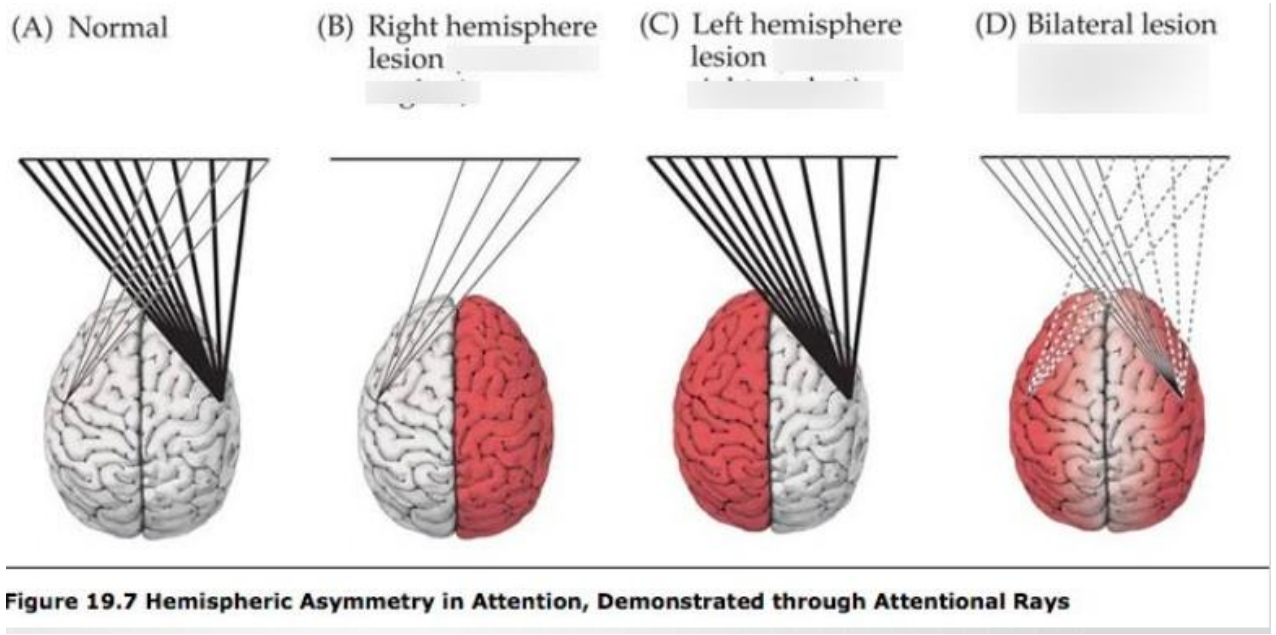
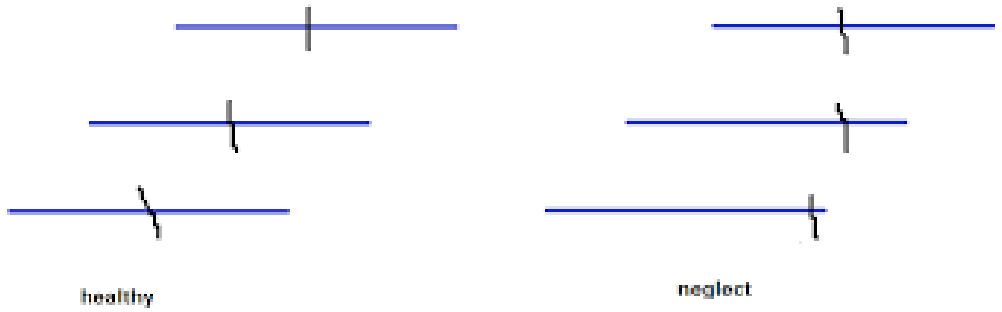
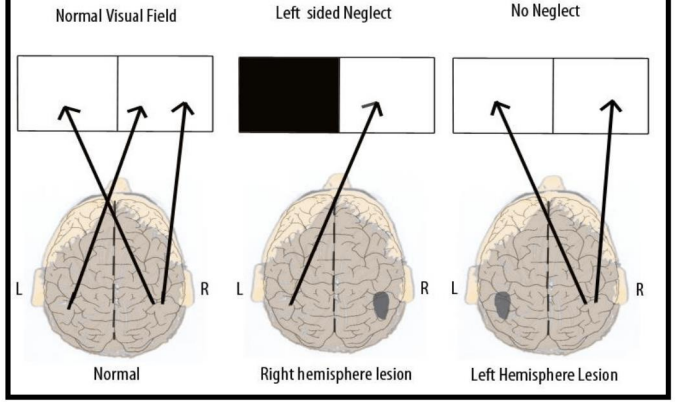
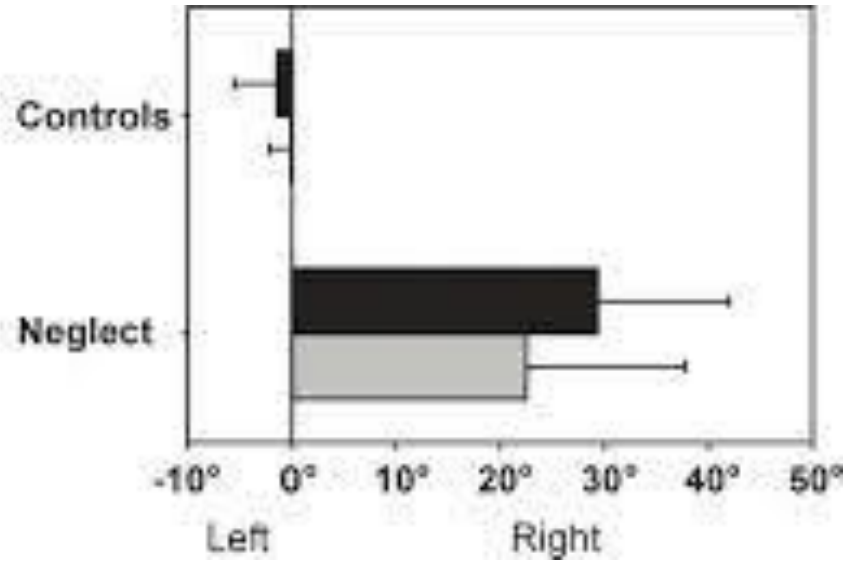


Figure 19.7 Hemispheric Asymmetry in Attention, Demonstrated through Attentional Rays



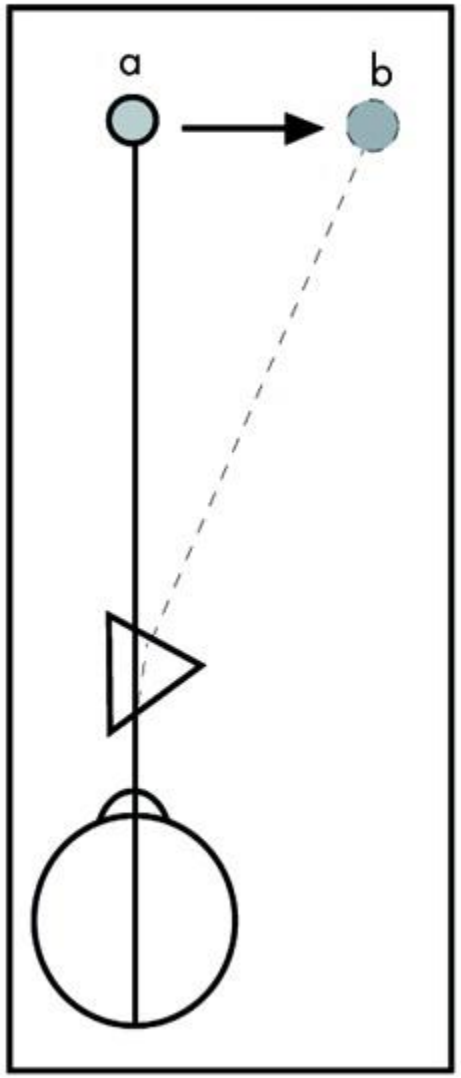


Right Left Right Left



The most prominent deficit in patients with spatial neglect is a bias of their active behaviour, i. e. a deviation of exploratory movements towards the right. When searching for targets, copying, or reading, the patients direct their eye and hand movements towards the ipsilesional side, leading to neglect of the contralesional side. The present study investigated whether spatial neglect is predominantly linked with such active behaviour or if it is obvious also without any explicit requirements, namely in the patients' spontaneous eye and head position. To address this issue we investigated the patients' spontaneous resting position while "doing nothing", i. e. just sitting and waiting for an experiment to start. Using magnetic search coil technique, we recorded spontaneous eye–in–head and head–on–trunk orientation in that waiting period in 24 patients with and without spatial neglect. In contrast to controls, neglect patients showed a marked deviation of spontaneous eye and head orientation of about 30° (= gaze position) towards the right. The findings strengthen the view that one component of the behaviour in neglect patients is due to a very elementary disturbance of spatial information processing. The deviation of eye and head may be understood as a pathological adjustment of the subject's normal resting position to a more rightward position. While the position in healthy subjects is in line with trunk orientation, this "default position" is shifted to a new origin in patients with spatial neglect.

Optical effect of rightward prism induced shift



Start of prism adaptation period

Prisms on

End of prism adaptation period

Prisms on

Post-adaptation (after effect)

Prisms off

- How does attention interact with vision?

Change blindness

Change blindness

Inattention Blindness

This is an example of what is called “inattention blindness” or “change blindness.” The idea is that people often miss large changes in their visual field.

Visit the ‘DIY’ link on S:EI to test your change blindness

Change blindness is a surprising perceptual phenomenon that occurs when a change in a visual stimulus is introduced and the observer does not notice it. For example, observers often fail to notice major differences introduced into an image while it flickers off and on again. People’s poor ability to detect changes has been argued to reflect fundamental limitations of human attention.

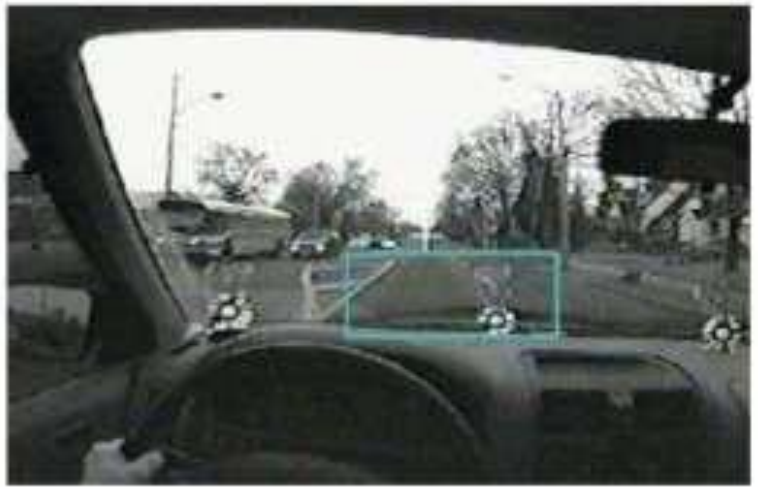
Source: Pavlov.tech

S:EI @ScienceEvidenceIntelligence

Inattention Blindness

A narrowed scope

Where drivers not using a hands-free cell phone looked.



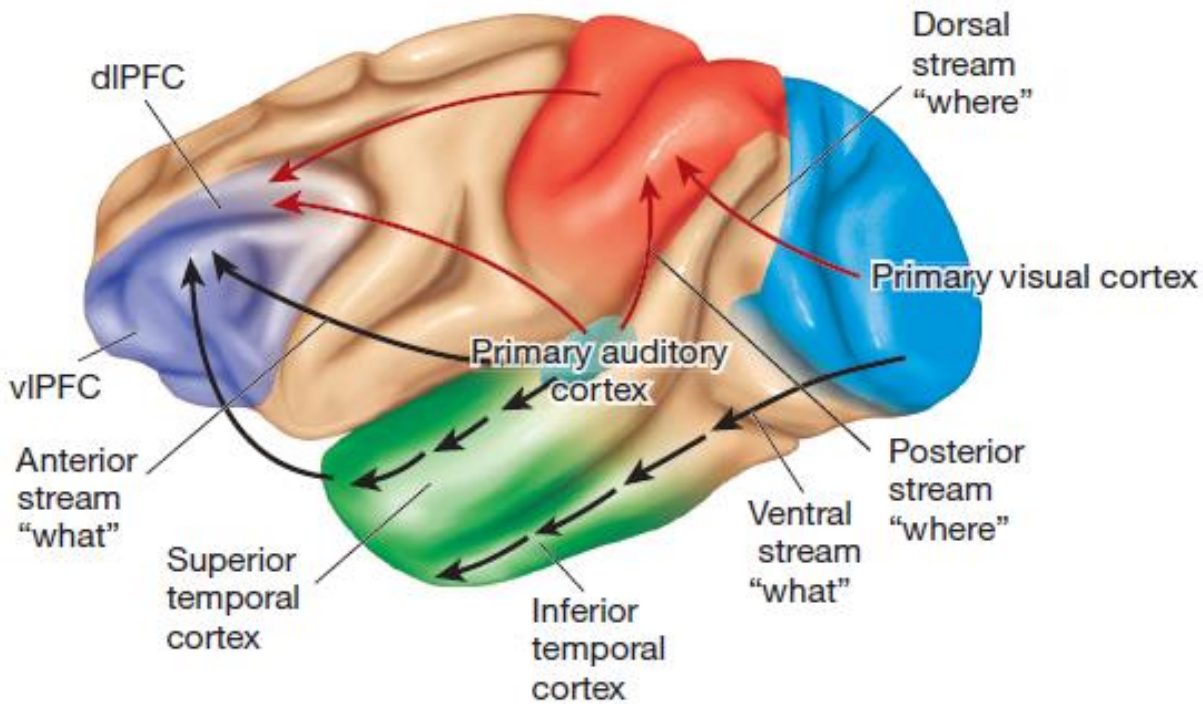
Where drivers using a hands-free cell phone looked.

Source: Transport Canada

Does perception need awareness?

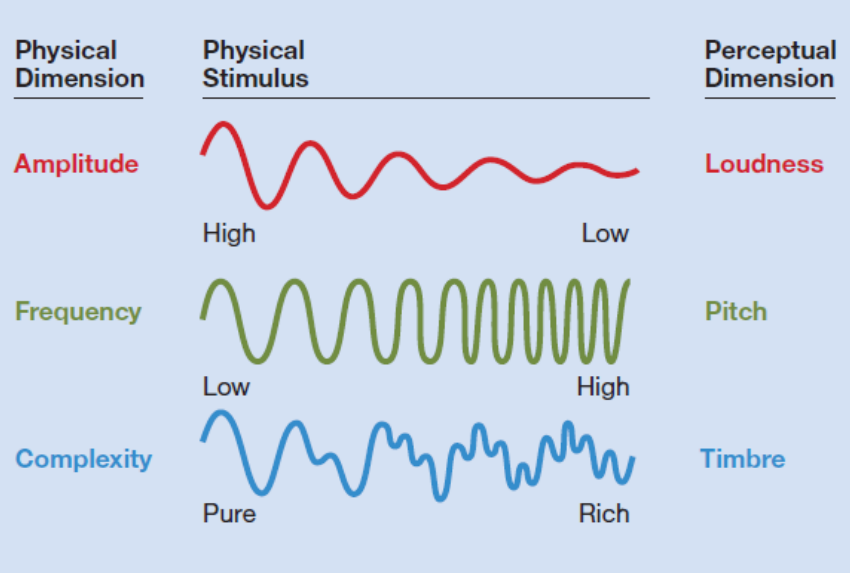
- In healthy normal brain

What would happen if someone becomes blind?



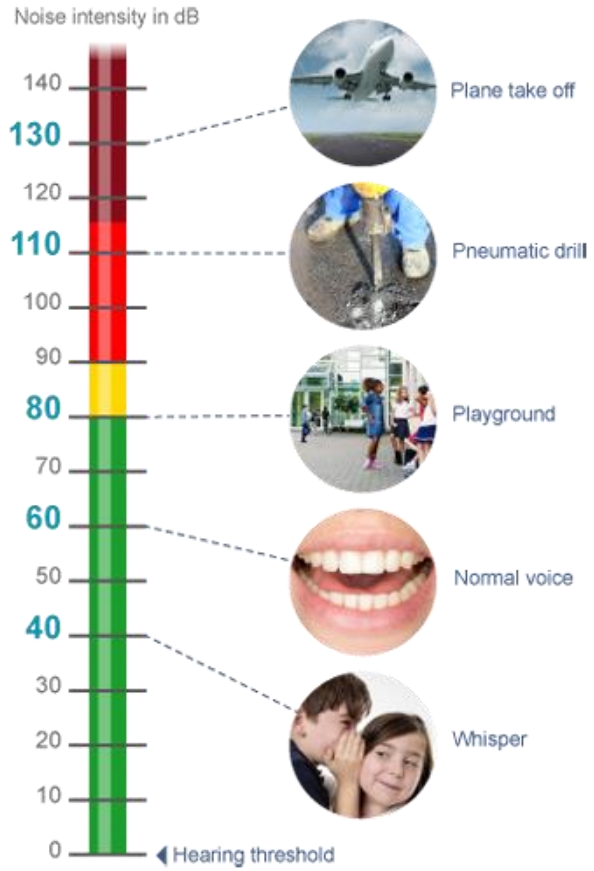
Enhanced responsiveness to auditory stimuli in blind people (auditory stimuli may engage the visual association areas in blind individuals)

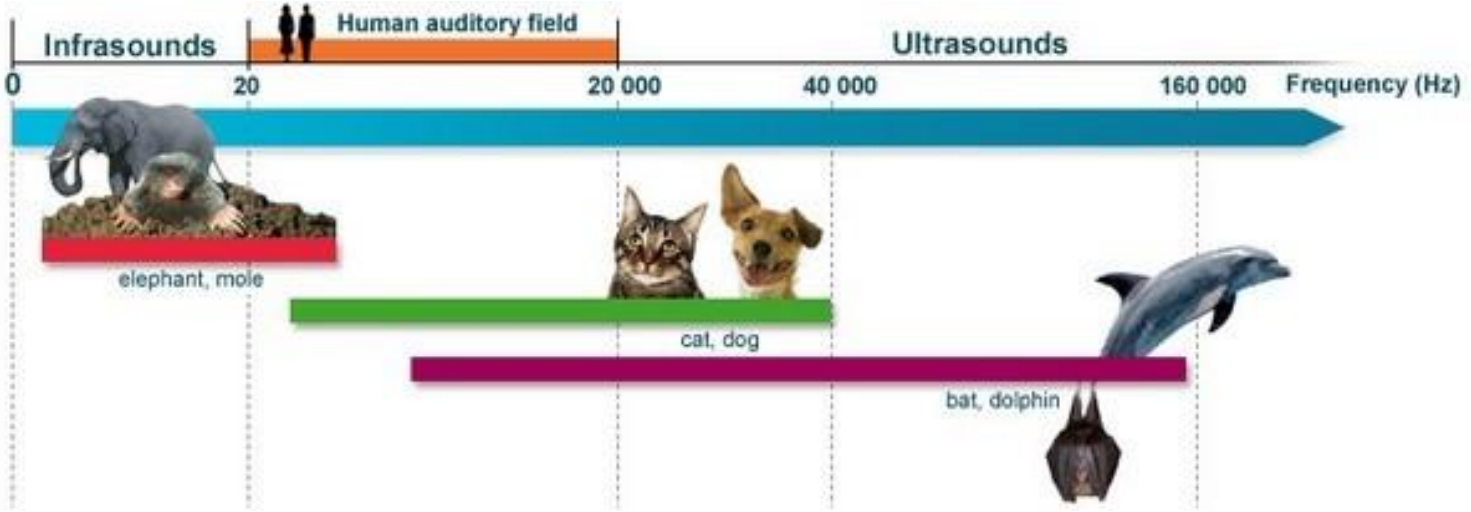
Audition



Decibels (dB)

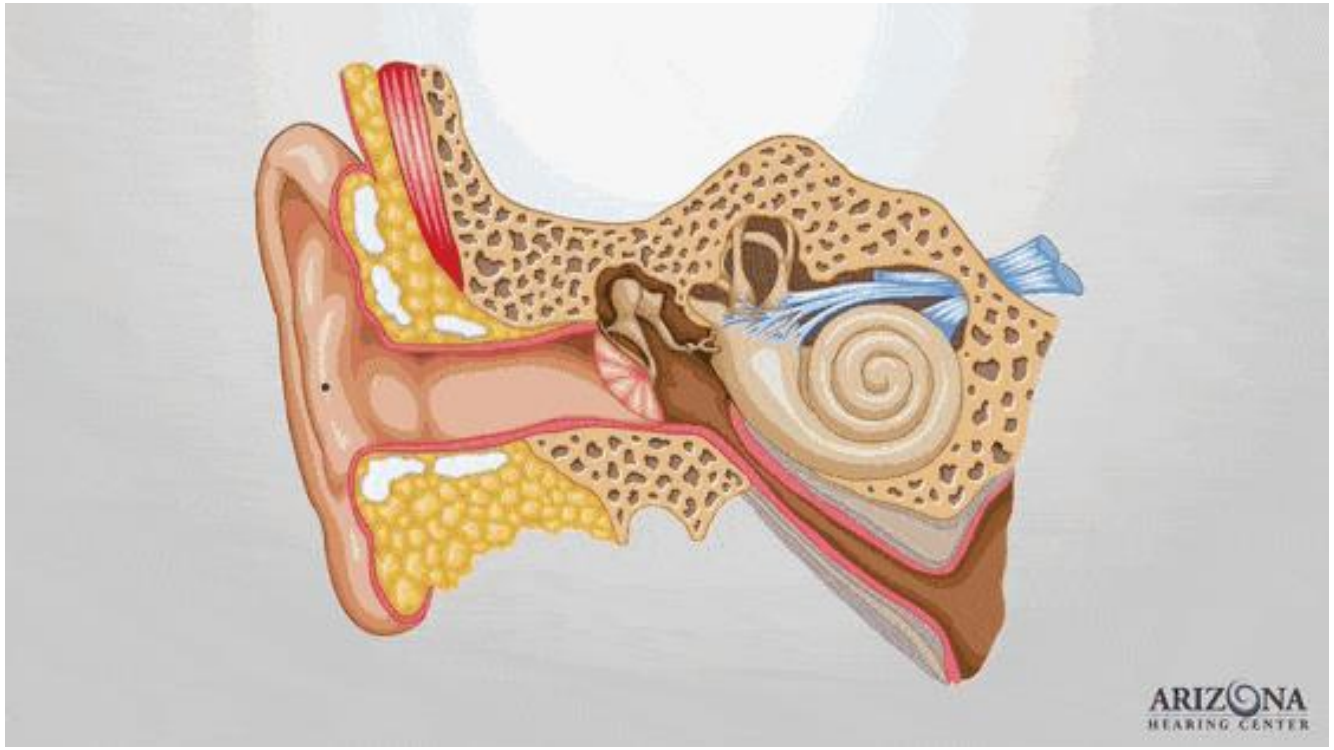
Hertz (Hz)

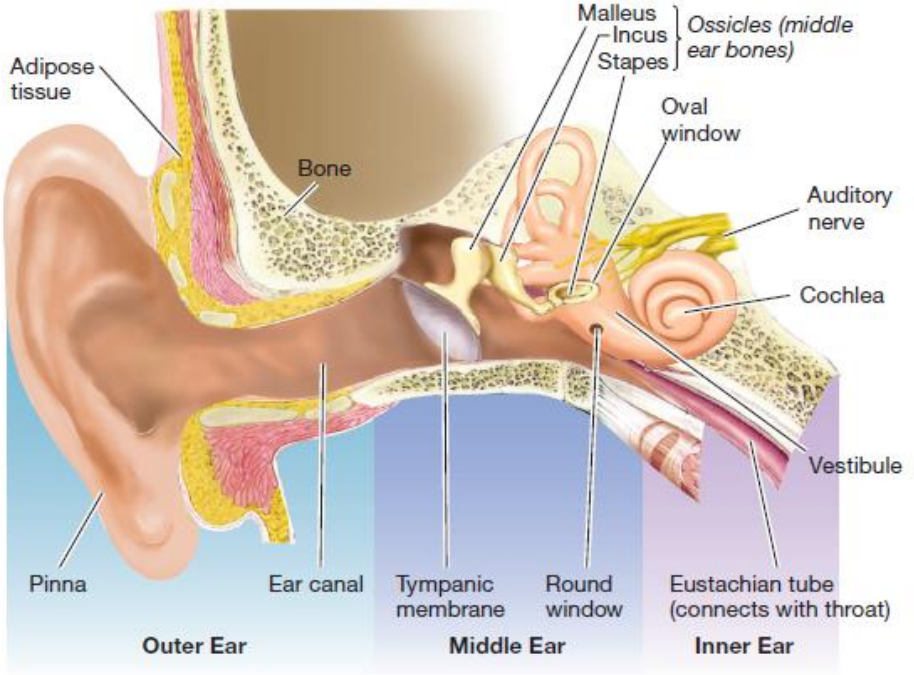


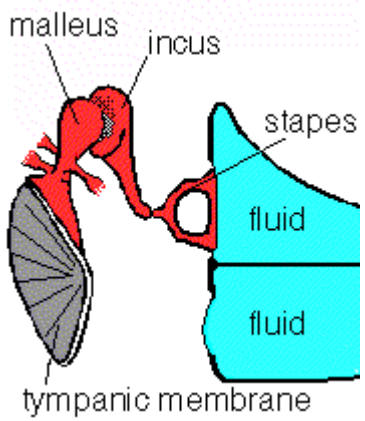


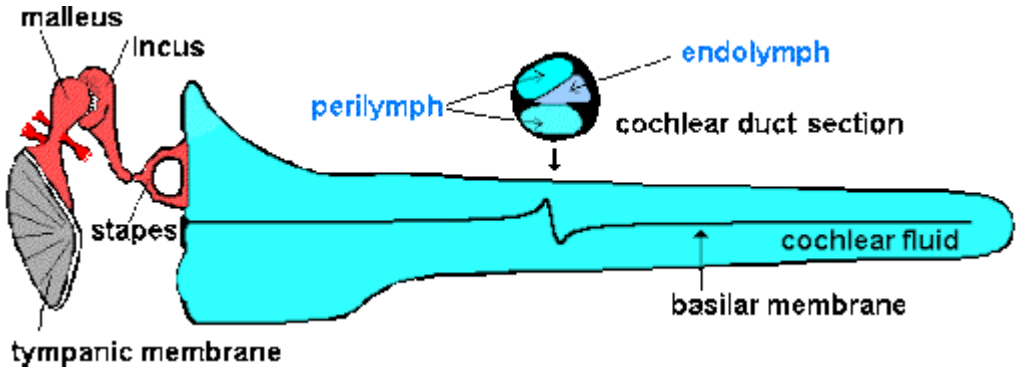
Humans are sensitive to vibrations between 20 and 20,000 times per second.
Most sensitive to 3000 Hz

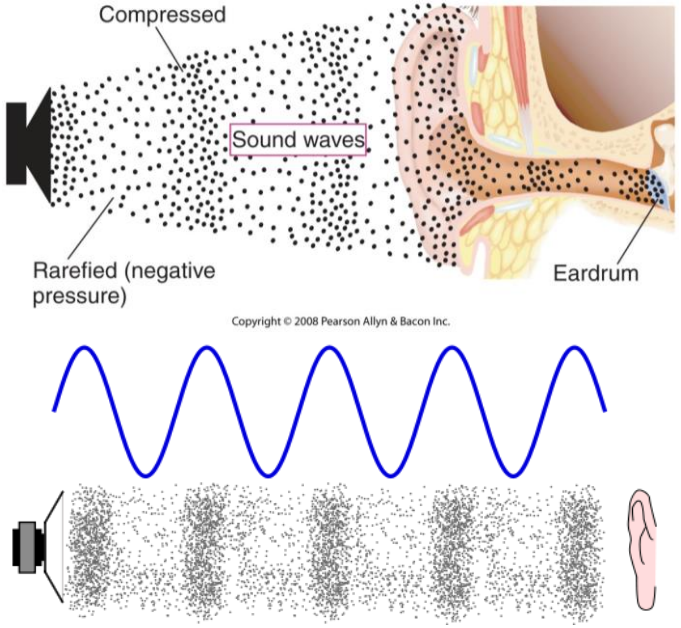
Anatomy of the ear





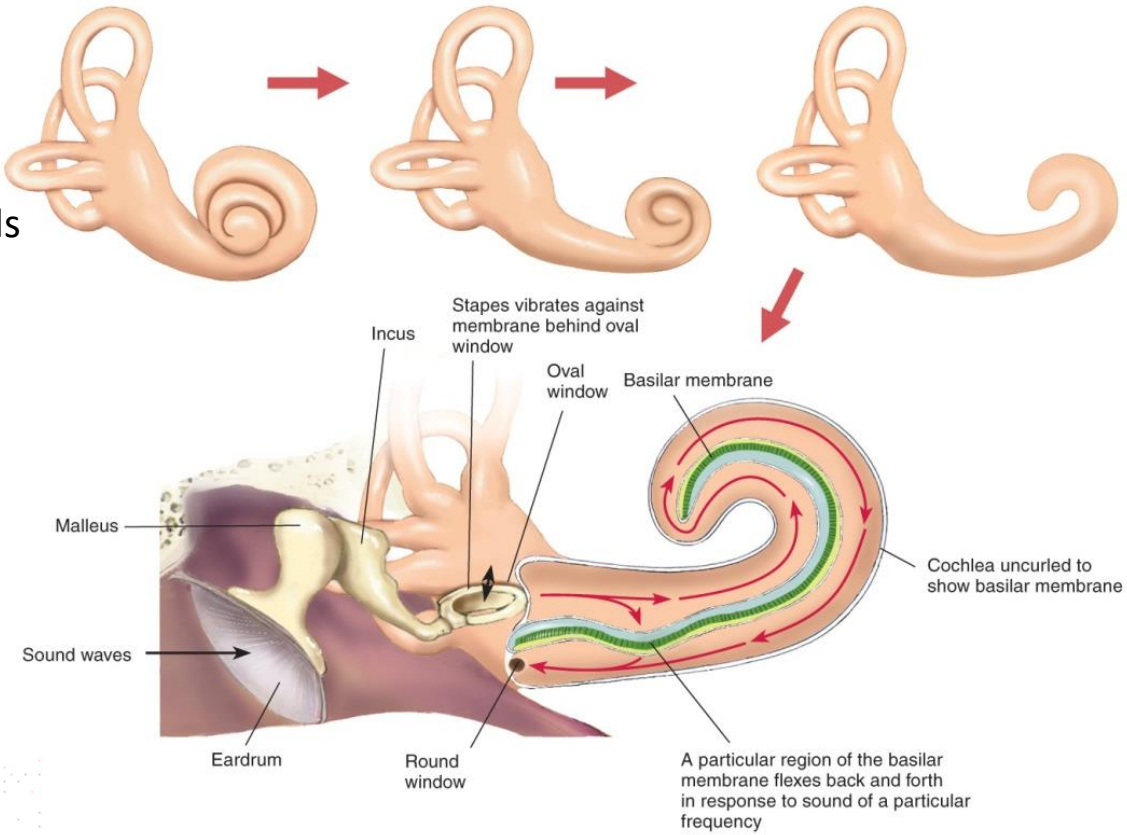




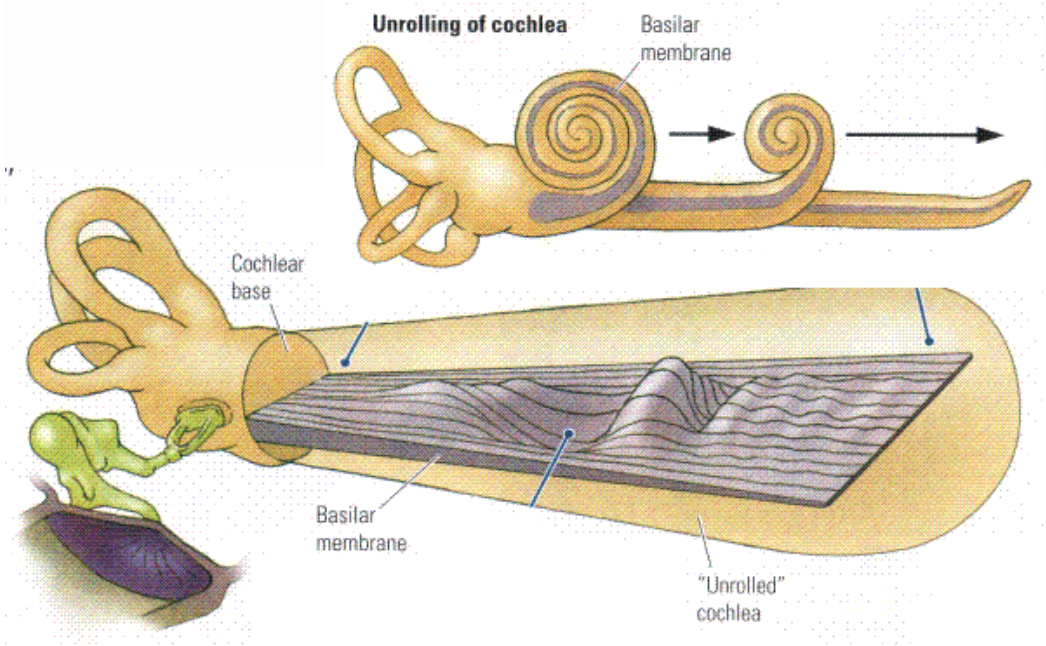


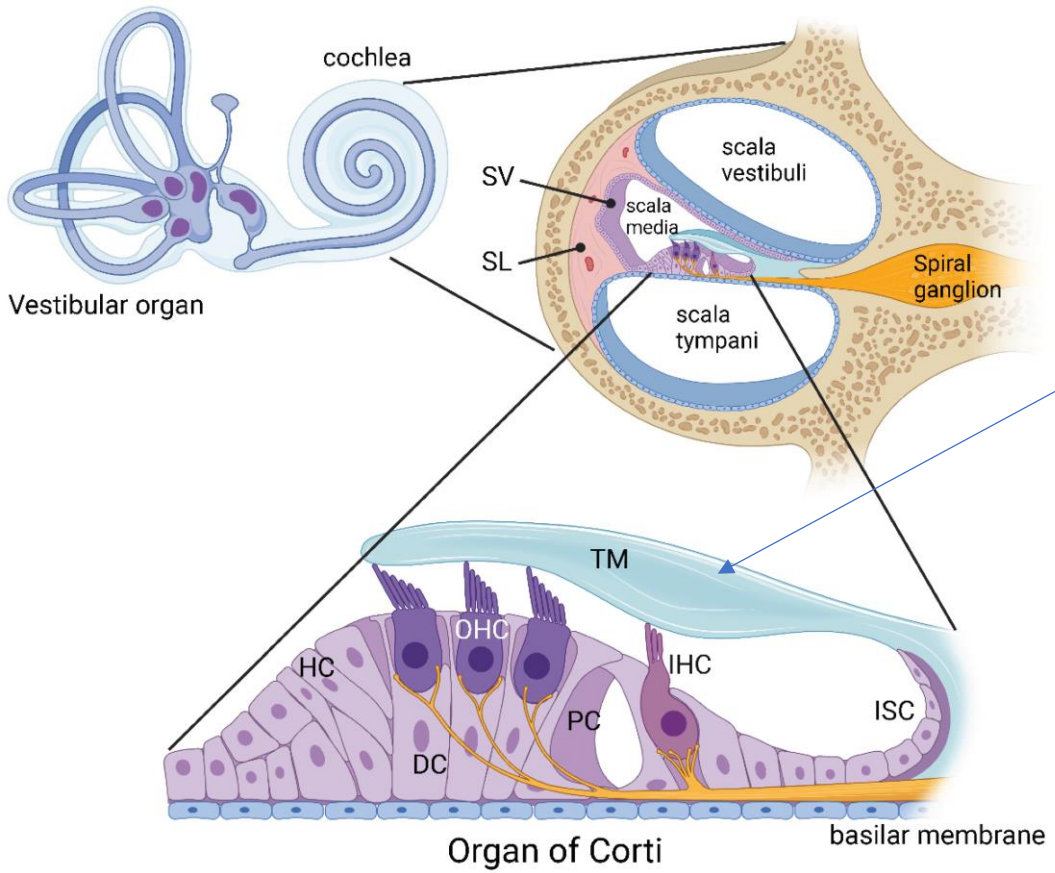
Sound transmitted from air is converted in to a forceful push by the ossicles to vibrate the fluid in the cochlea.

Kinetic energy (air)→ mechanical vibrations → liquid vibrations → electrical signals

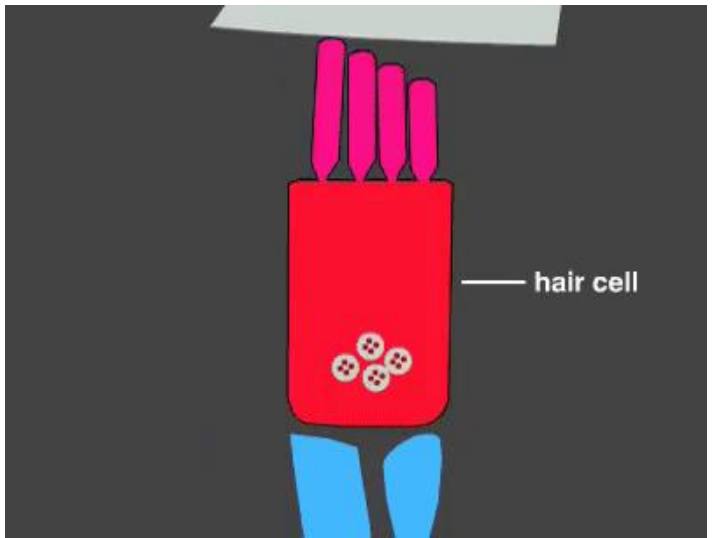


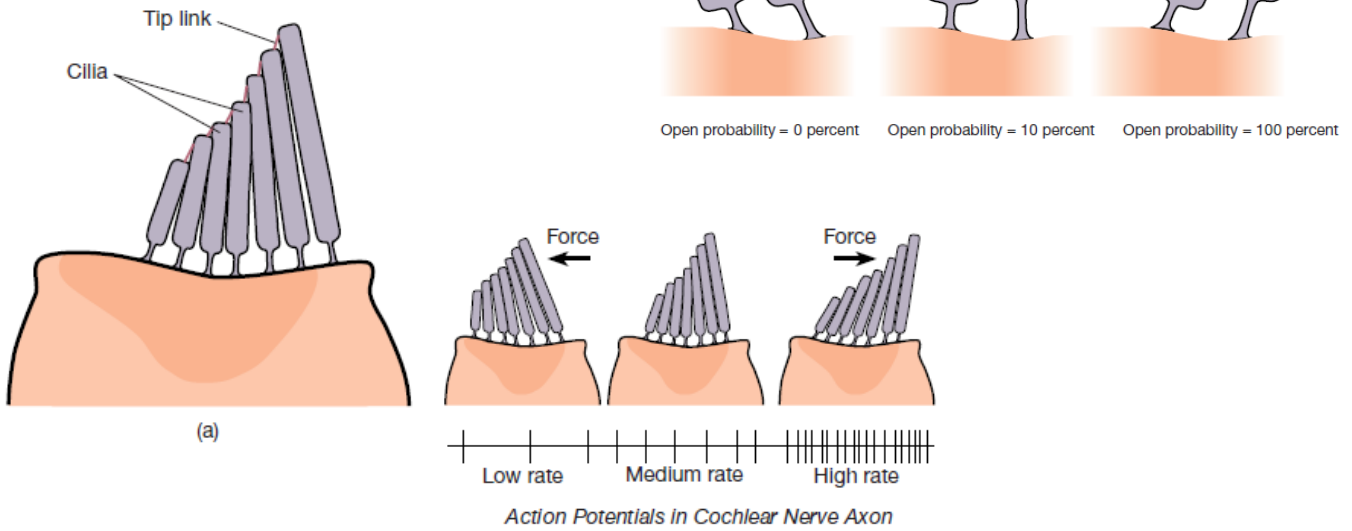
- In the absence of vibrations the cochlea is curled and no vibrations are transmitted in this position
- When the fluid vibrates, the pressure changes in the fluid produce vibrations in the basilar membrane
- High-frequency sounds cause the base of the membrane - the end nearest the oval window—to bend.
- Low frequency sounds flex the tip of the basilar membrane.





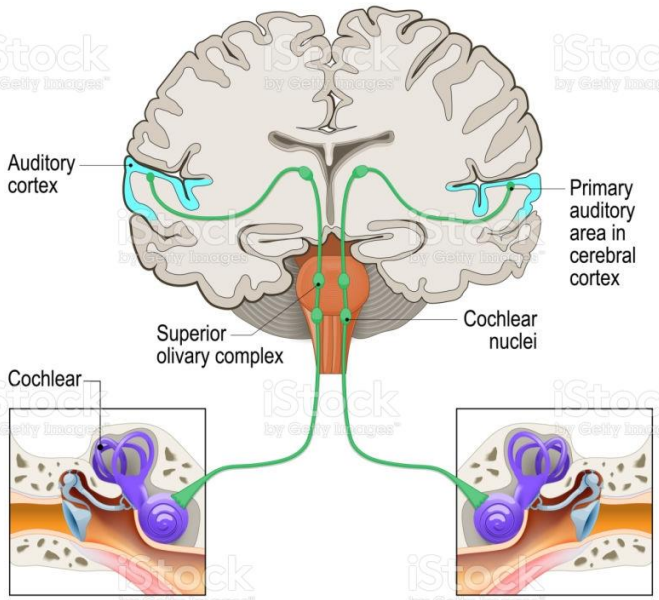
- The basilar membrane has hair cells which act as receptors
- Sound waves cause the basilar membrane to move relative to the **tectorial membrane (TM)**, which bends the cilia (eyelashes) of the hair cells.
- When the cilia bend towards the tallest one, the ion channels open, much like a trap door. This mechanism transduces mechanical energy into electrical impulses.
- The hair cells synapse on the auditory nerve that carries the signal to the brain

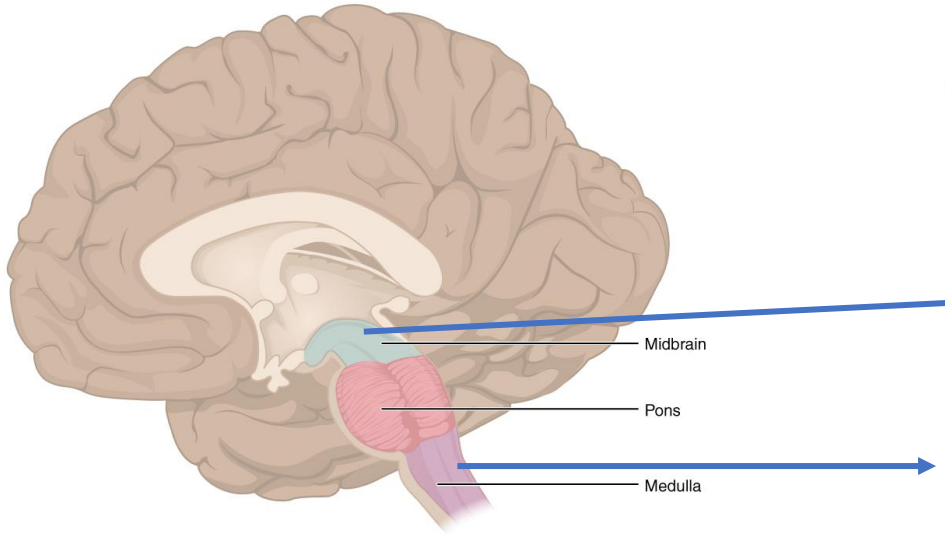


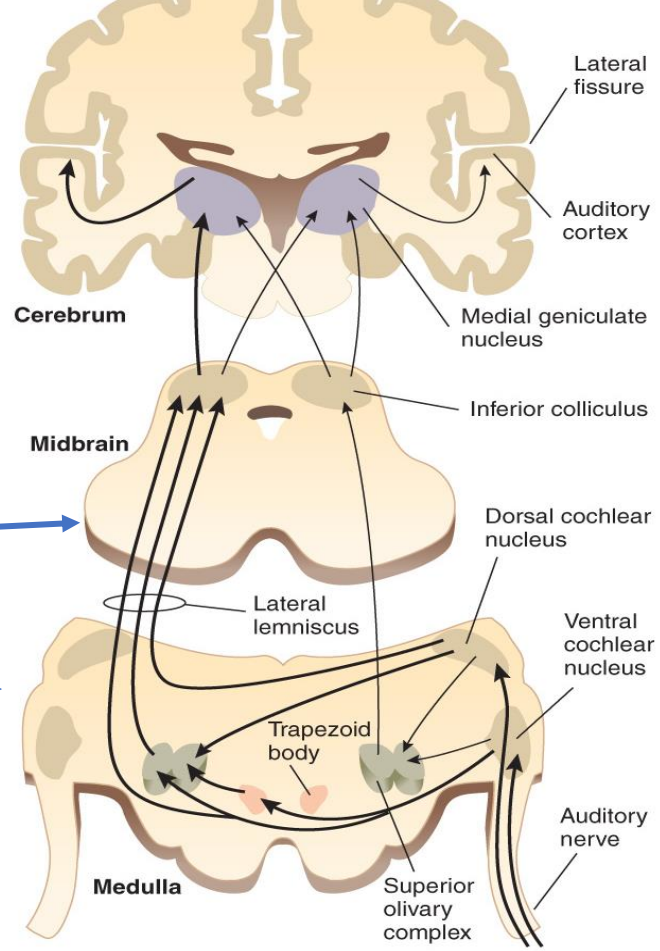


- Where should the sound signals go from the ear (auditory nerve)?

Auditory pathway







- Auditory nerve → cochlear nucleus (medulla) → superior olivary nucleus (medulla) → inferior colliculus (midbrain) → MGN (thalamus) → primary auditory cortex (temporal lobe)

pathways of the auditory system are inherently complex, and they have many more synapses than the other senses

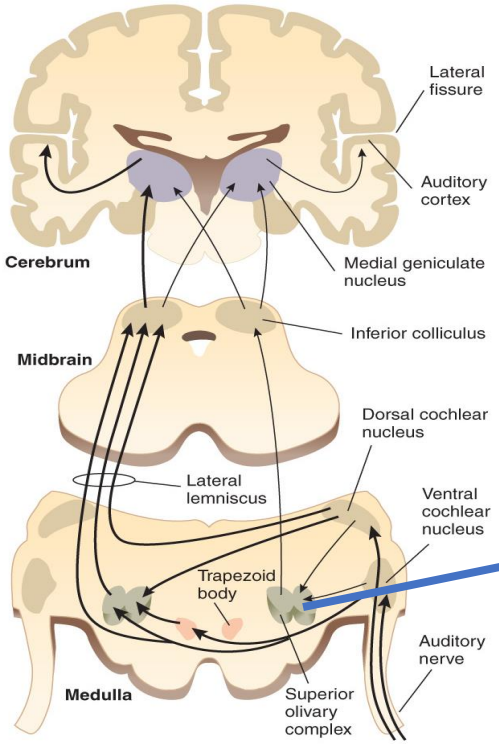
- The primary auditory cortex receives input from both ears and processes the quality of sound
  - More contralateral fibers than ipsilateral nerves

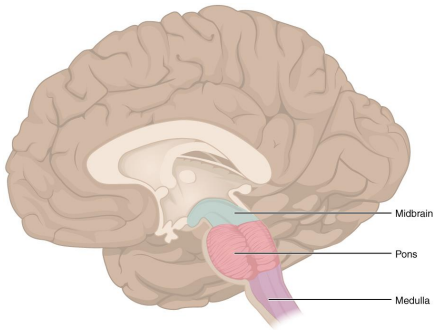
https://hearinghealthmatters.org/pathways/2017/contralateral-ear-effect-tests-central-auditory-function-introduction/

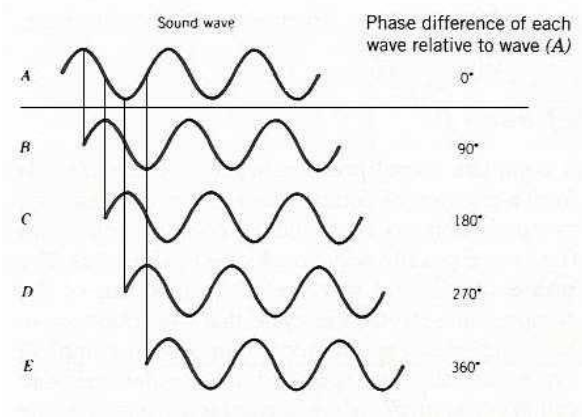
Perception of Spatial Location (in medulla)

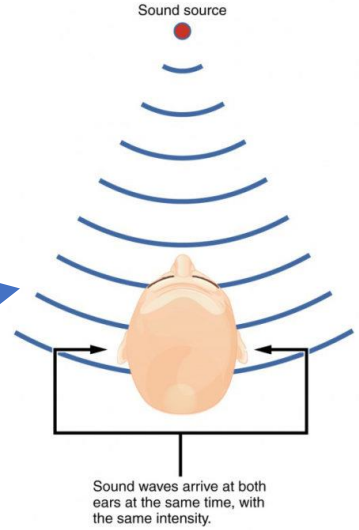
Phase difference is calculated by the superior olivary complex

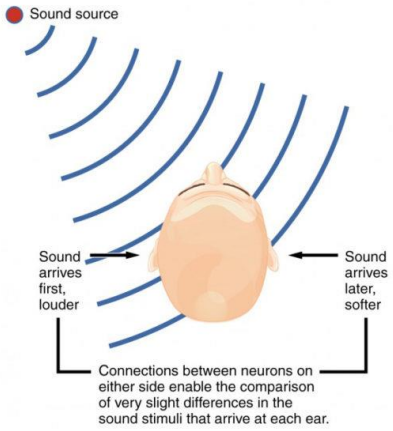
- If sound is located on one side, it will reach the other ear 180° out of phase
- If sound is located in front of us, it reaches both ears in the same phase (0 ° out of phase)

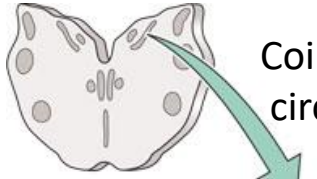




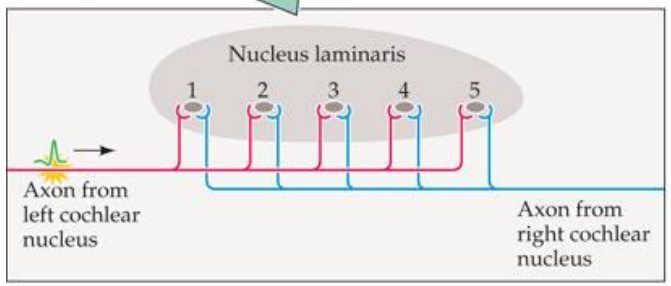


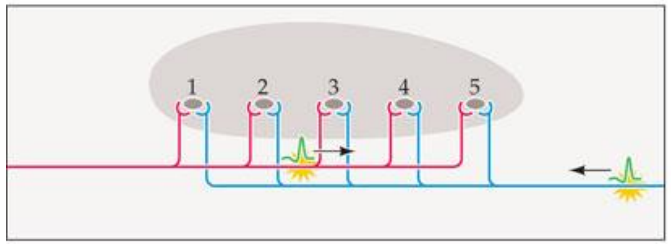


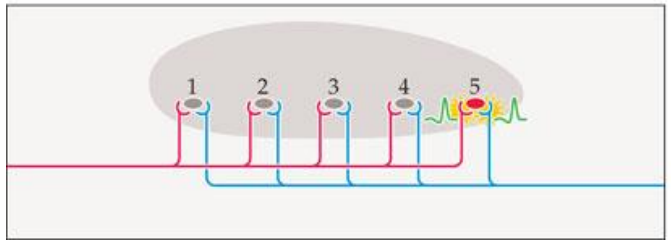




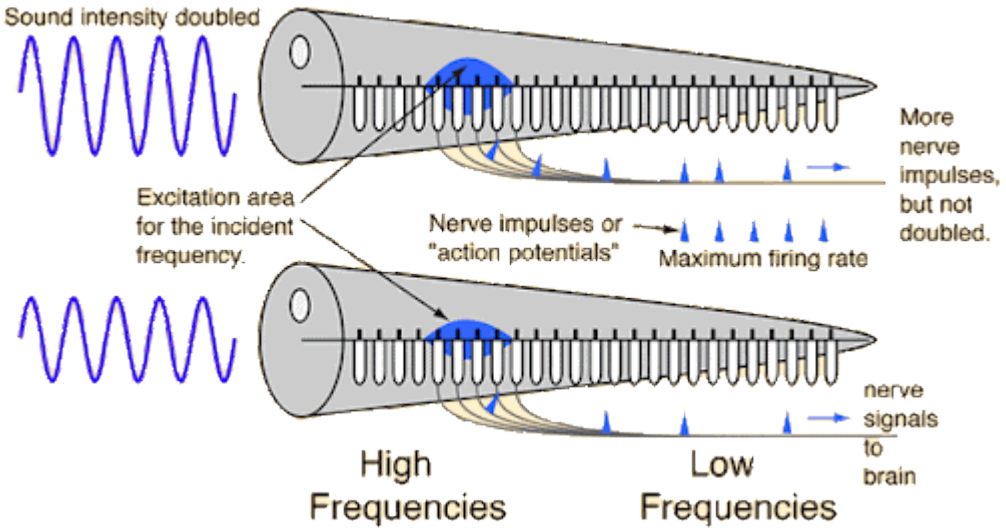
Coincidence detector circuit

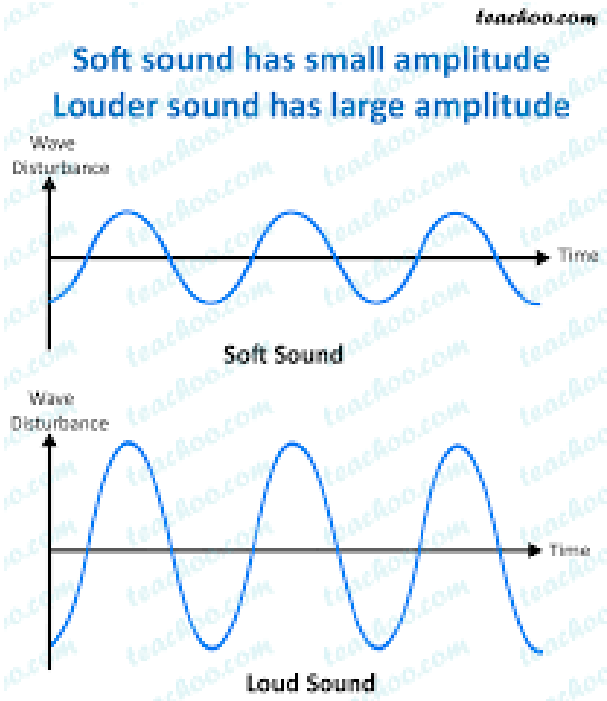






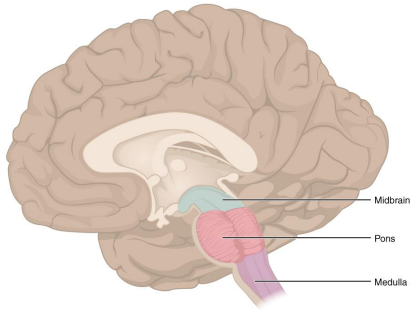
Perception of loudness



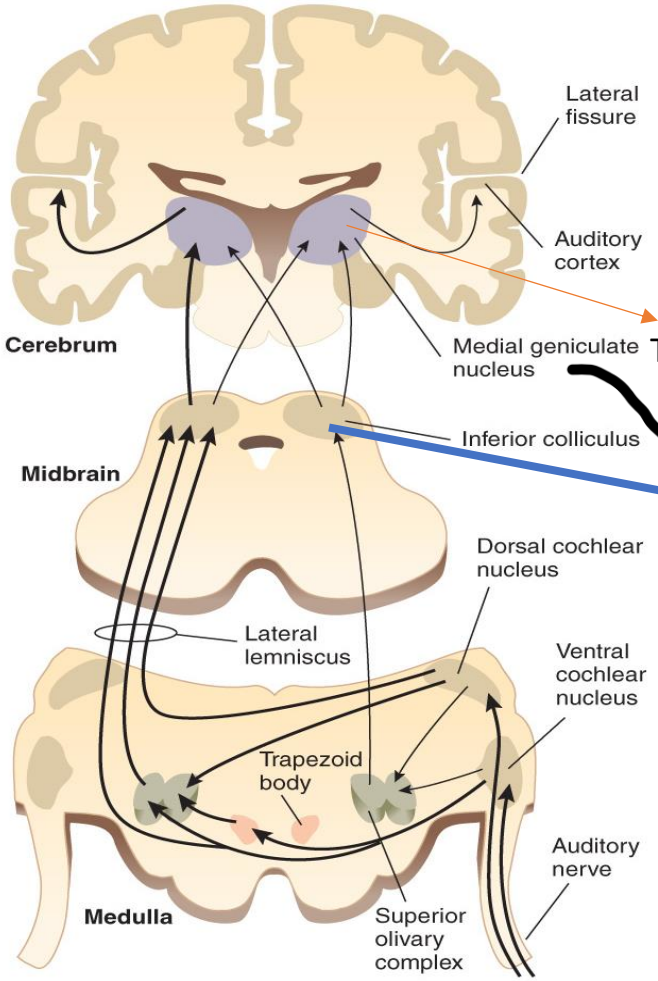


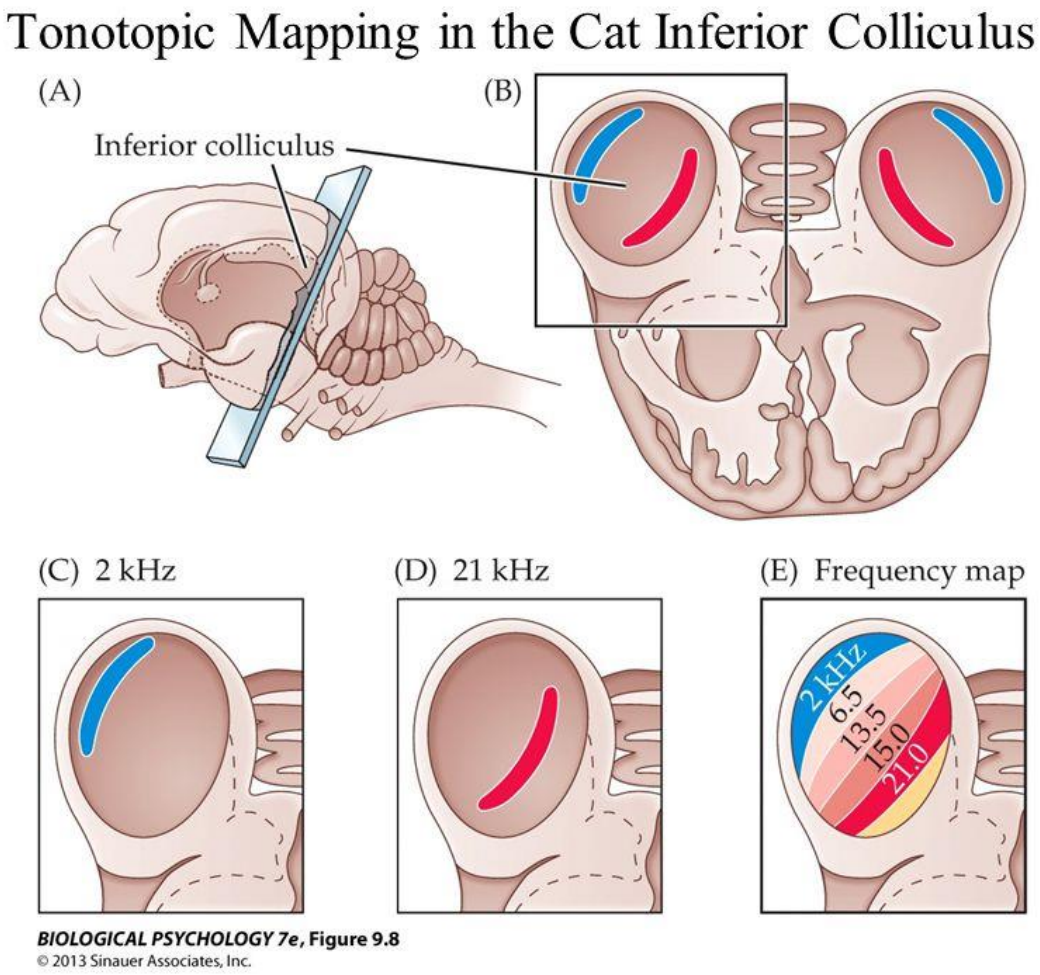
Louder sounds produce more intense vibrations of the eardrum and ossicles, which produce a more intense shearing force on the cilia of the auditory hair cells. As a result, these cells release more neurotransmitter, producing a higher rate of firing by the cochlear nerve axons

Loudness in not linear, it is logarithmic (twice the amplitude does not mean twice as loud)



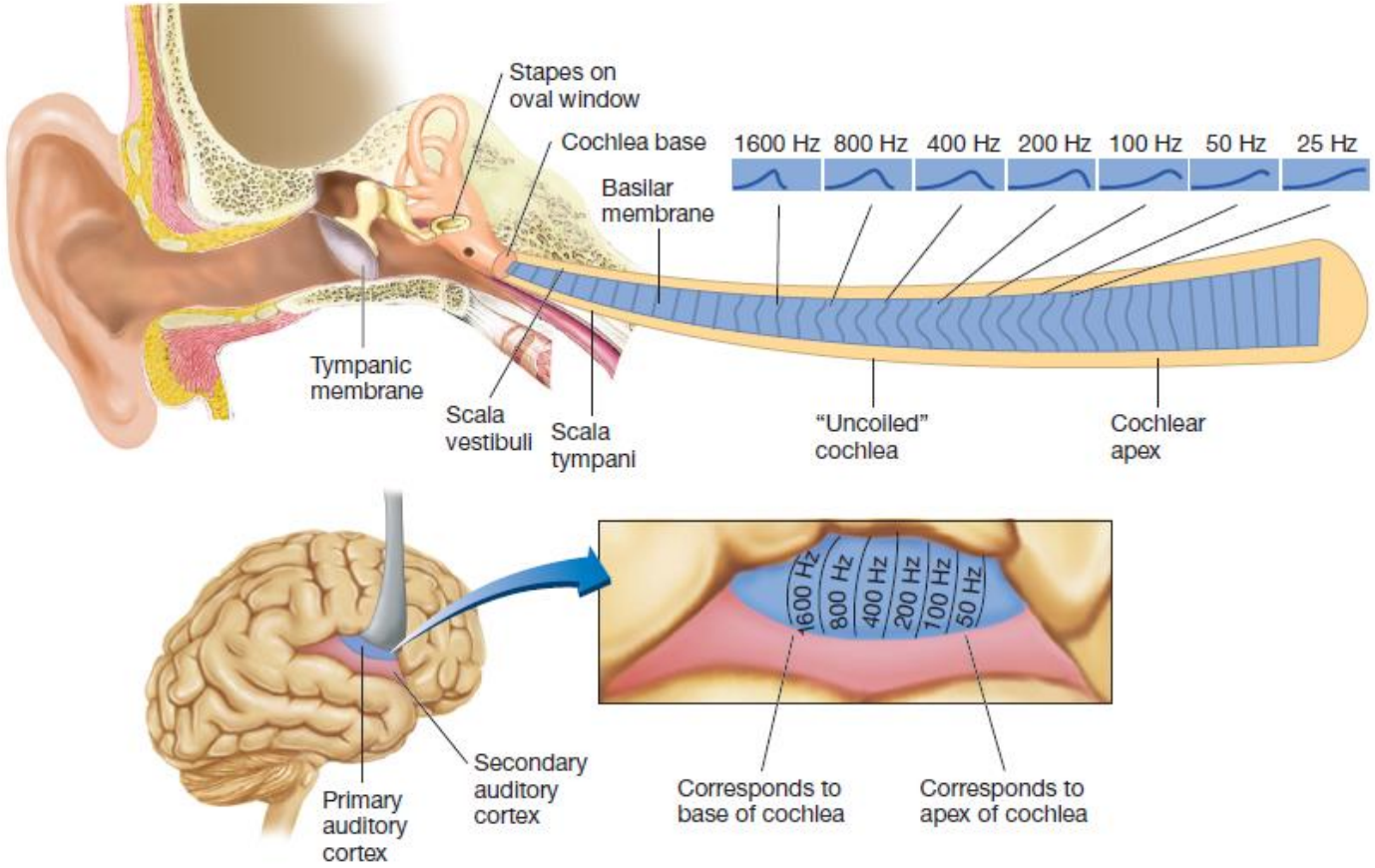
Rudimentary Sound perception in the Midbrain



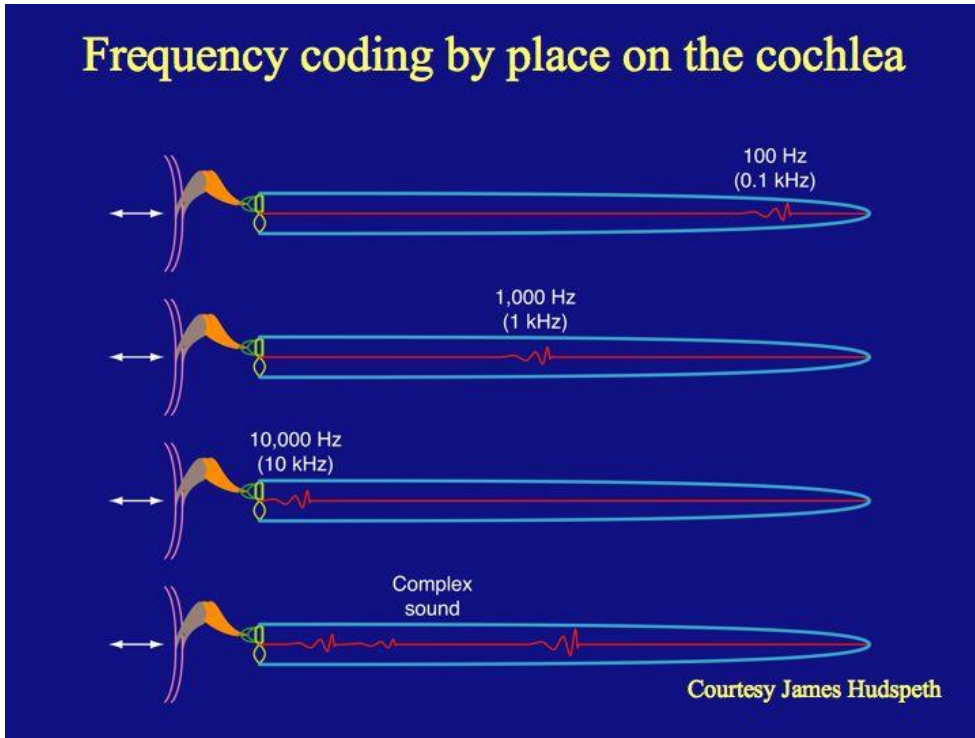


Frequency mapping

The frequency map (tonotopic representation) of the basilar membrane is preserved through processing in the subcortical structures and mapped in the primary auditory cortex.



Different frequencies are processed in their corresponding area of the auditory cortex



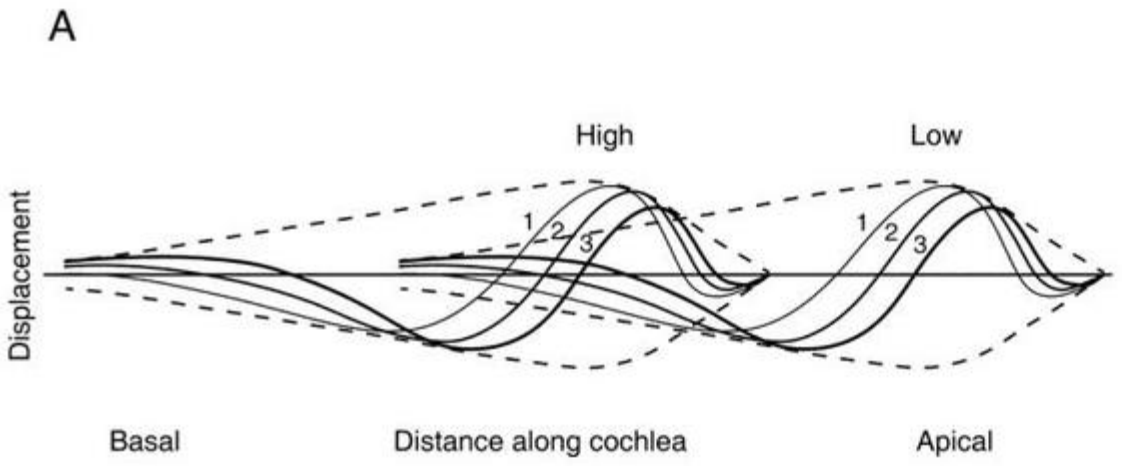
Perception of Pitch

- What happens when you combine frequencies?
- Which frequency do you really hear?

The case of the missing fundamental

Perception of timbre

When the basilar membrane is stimulated by complex sounds, different portions of the membrane respond to each of the overtones. This response produces a unique anatomically coded pattern of activity in the cochlear nerve, which is subsequently identified by circuits in the auditory association cortex



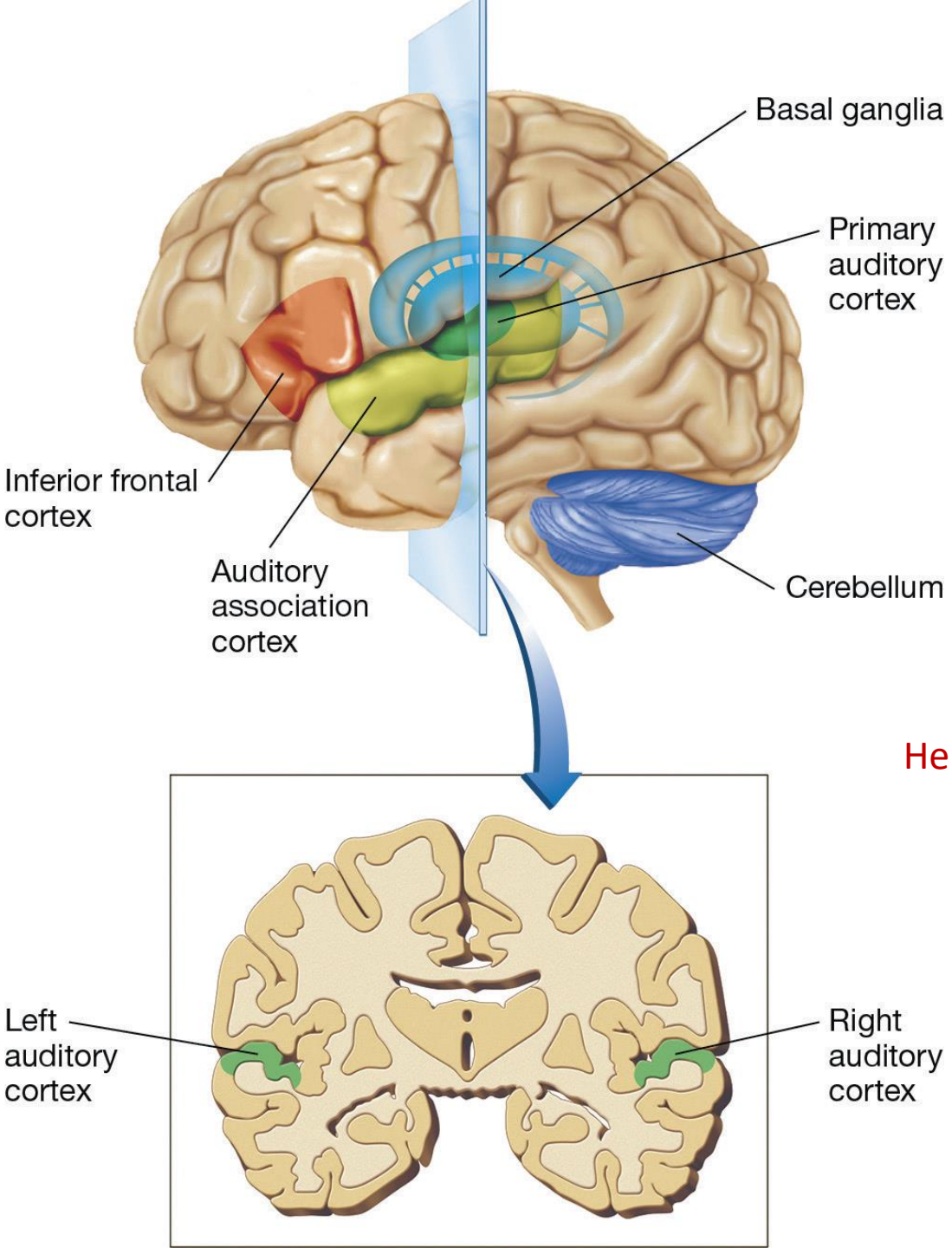
Special characteristics of neurons in the auditory pathway afford the processing of timbre.

e.g. hollow or solid door
e.g. different voices of friends

Music perception

a melody is recognized by the relative intervals between its notes, not by their absolute value.

the inferior frontal cortex appears to be involved in recognition of harmony



the cerebellum and basal ganglia are involved in timing of musical rhythms, as they are in the timing of movements.

Heritability of musical abilities is high

the left auditory cortex appears to be involved in perception of rhythmic patterns that are superimposed on the rhythmic beat

right auditory cortex appears to be involved in perception of the underlying beat in music

Auditory processing disorder (APD)

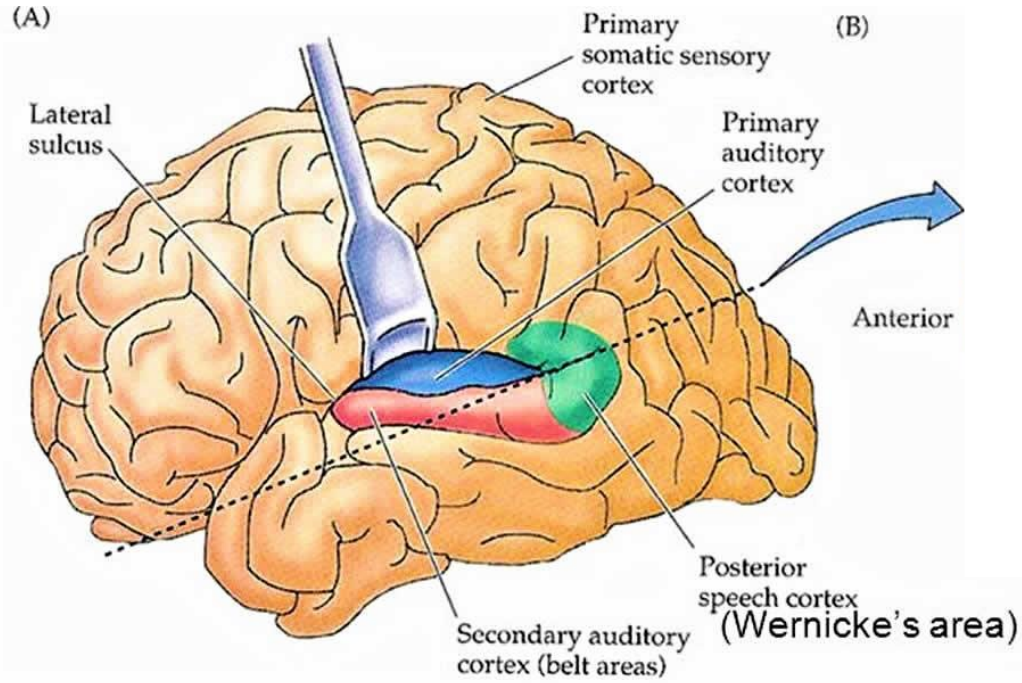
Someone says, "Please raise your hand," and you hear something like "Please haze your plan."

APD is a rare hearing problem that affects school-aged children.

- Auditory process matures fully by 13 years
- The child may grow out of the disorder once they reach that age.
- Listening skills usually develop as the auditory system matures.
- It usually takes around 12-15 years of age to have complete auditory processing maturity.

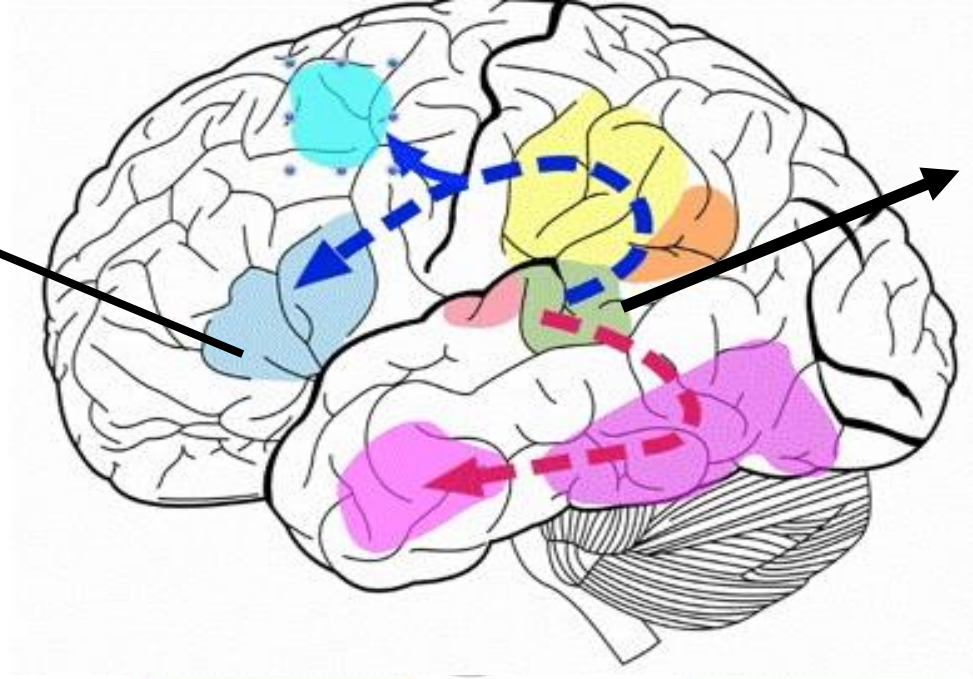
- Cause - genetic, brain trauma during childbirth, infection of the ear.
- Currently, APD is recognized as a “specific learning disability”

Auditory Pathways



Planning speech output

auditory comprehension



Dorsal pathway

auditory-motor integration

verbal repetition

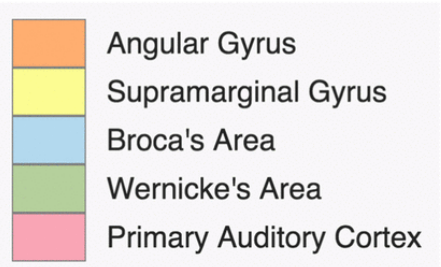
sound localization

Ventral pathway

auditory-conceptual mapping

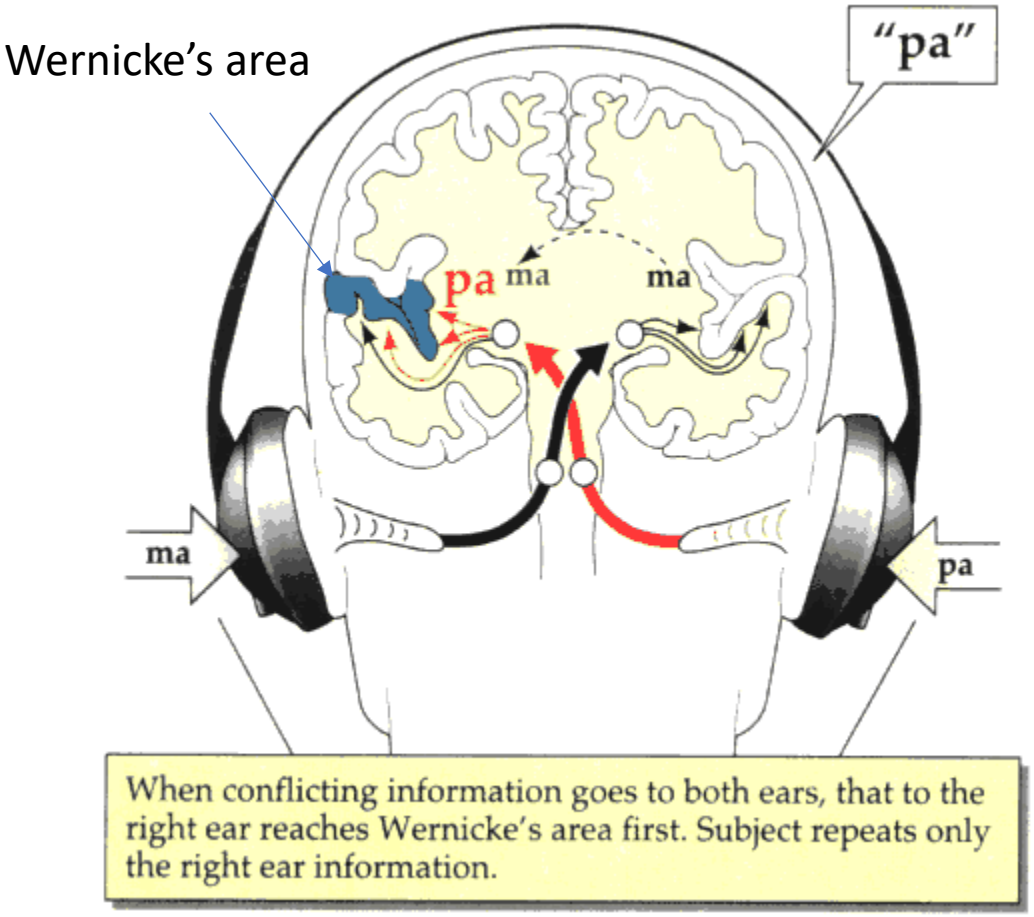
auditory comprehension

analysis of complex sounds



Asymmetries of the Auditory System

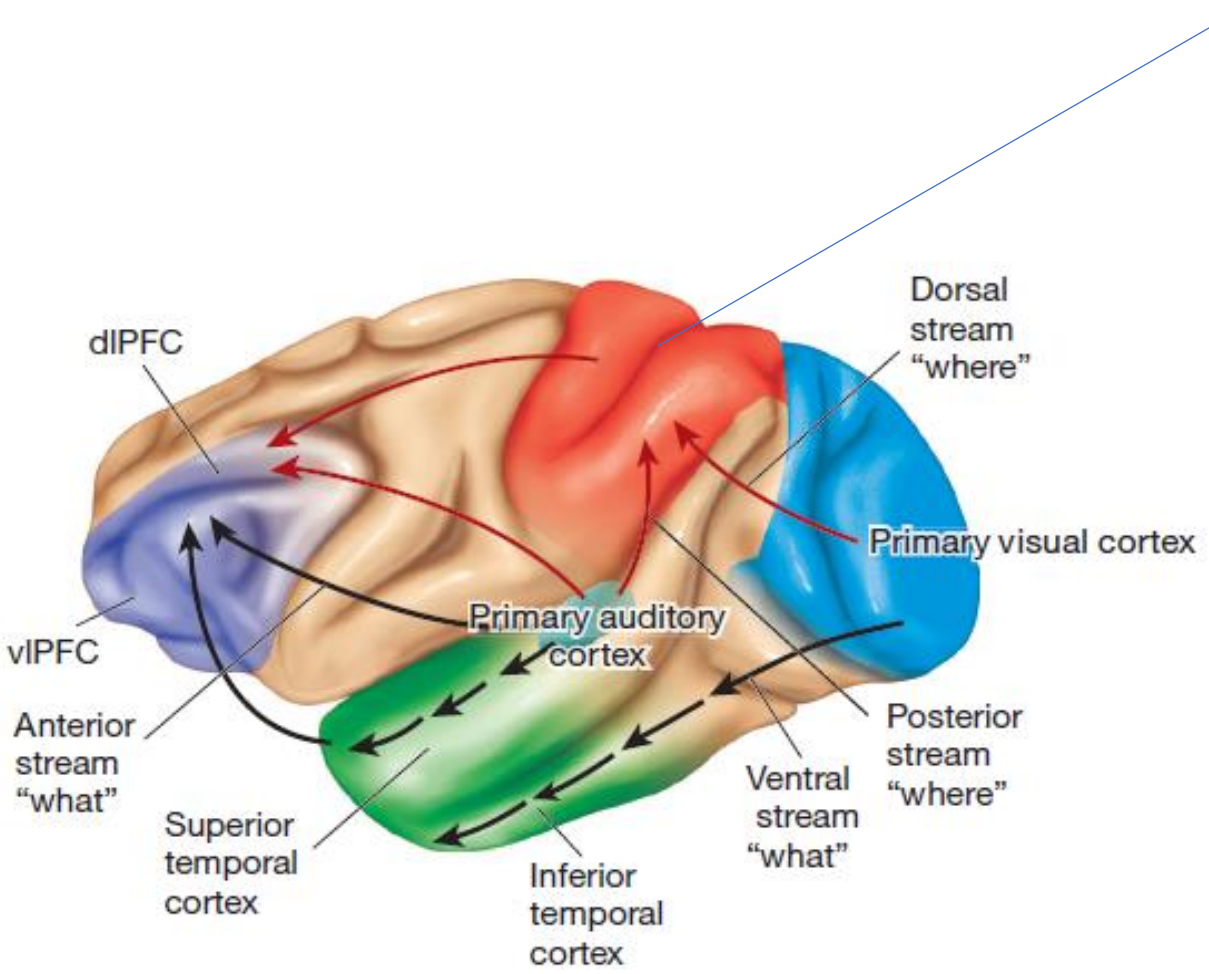
- In normally hearing individuals, anatomical and functional observations from the cochlea up to the cortex are in favor of a right ear advantage (REA), a feature hypothetically linked to the fact that in almost all right handed and most left handed people, speech is processed predominantly in the left cerebral hemisphere.
- Both afferent and efferent auditory pathways show asymmetrical features which suggests that competing signals from both ears are processed with a REA which enables the left hemisphere to process speech appropriately in difficult listening situations.
- Stimuli with complex speech-like acoustic properties, including rapid spectrotemporal changes, yield greater activation in auditory cortex over the left hemisphere, regardless of whether right ear, left ear, or binaural stimulation is used.
- The left hemisphere is specialized from birth for processing specific properties of speech and children exhibit the right ear advantage as early as the first year of life.



Right Ear Advantage

https://hearingreview.com/inside-hearing/research/remarkable-history-right-ear-advantage

Overlap of auditory and visual dorsal pathways



the visual and auditory "where" streams overlap in the parietal lobe.

We can use the convergence of sight and sound to recognize which of several objects in the environment is making a noise.

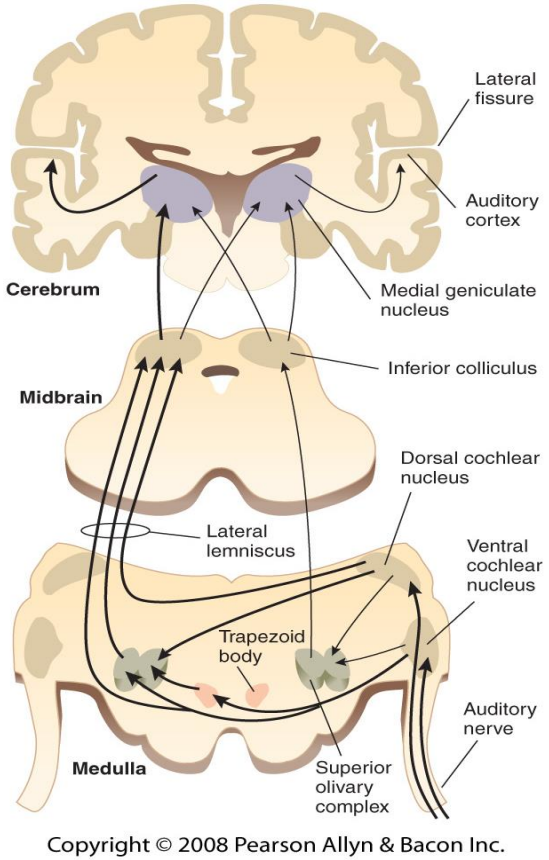
we can learn the association between the sight of an object and the sounds it makes – learning and memory

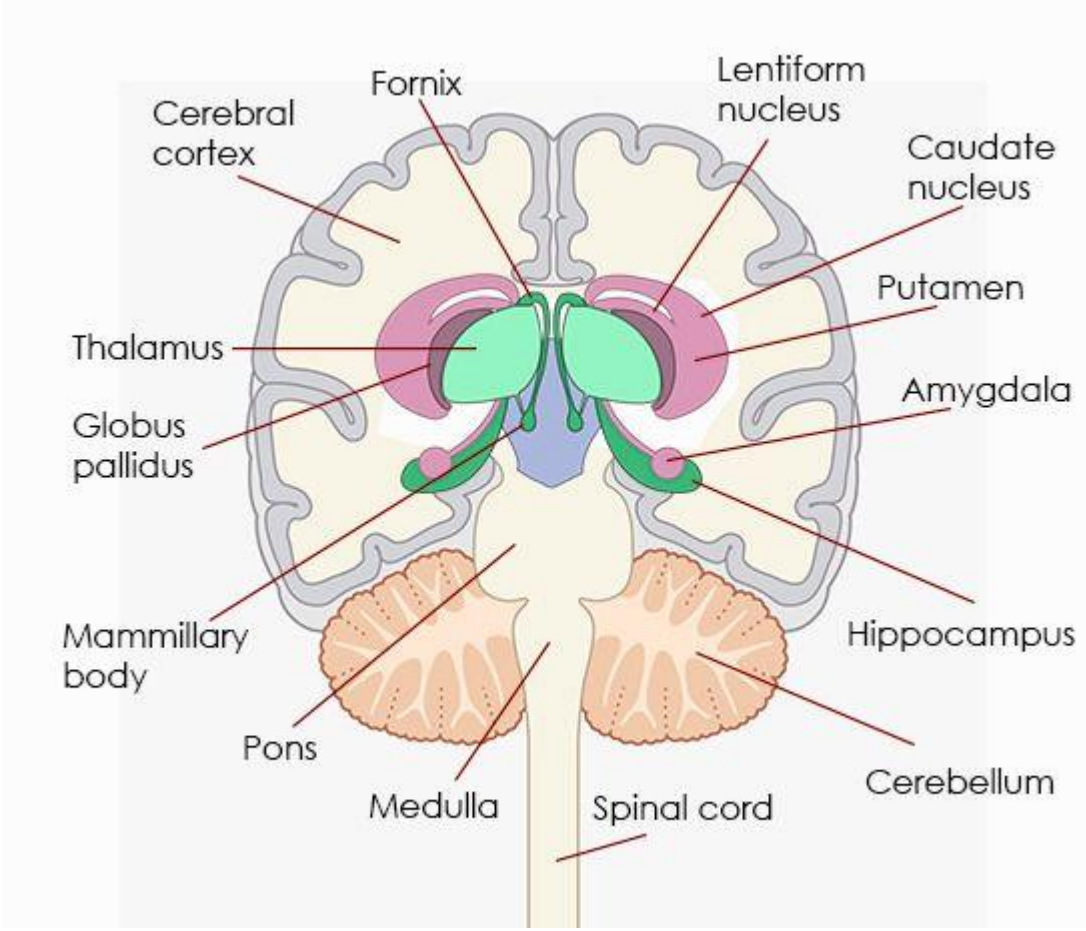
Enhanced responsiveness to auditory stimuli in blind people (auditory stimuli may engage the visual association areas in blind individuals)

- Can you detect additional information from sounds?

Auditory fear processing

Thalamus → amygdala





Types of Deafness

trouble identifying sounds → damage to? → auditory 'what' pathway

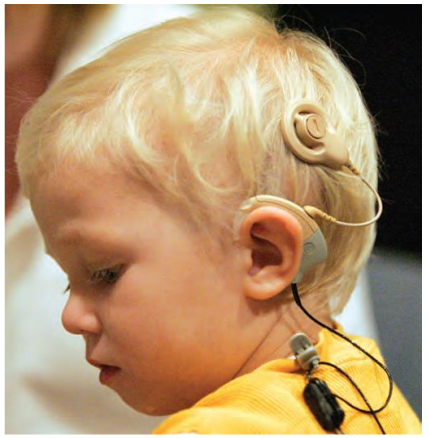
difficulty localizing sounds → damage to ? → auditory 'where' pathway

Aging related deafness → damage to?→ basilar membrane neurons (esp high freq - "s," "f," and "t" sounds)

Noise (loud) induced hearing loss→ damage to the tympanic membrane or hair cells or base of basilar membrane that receives most signals first

Complete deafness → damage to ? → bilateral 'primary' auditory cortex

Congenital deafness → damage to ? → cochlea → cochlear implants



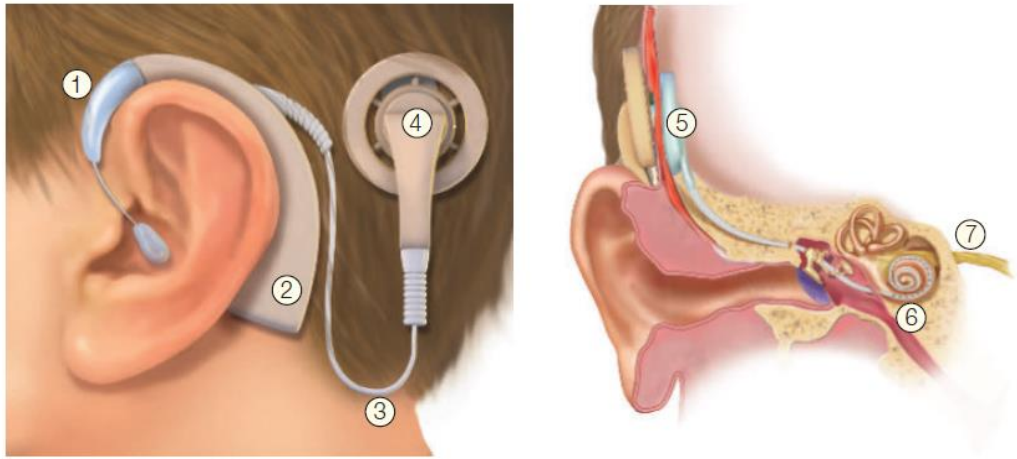
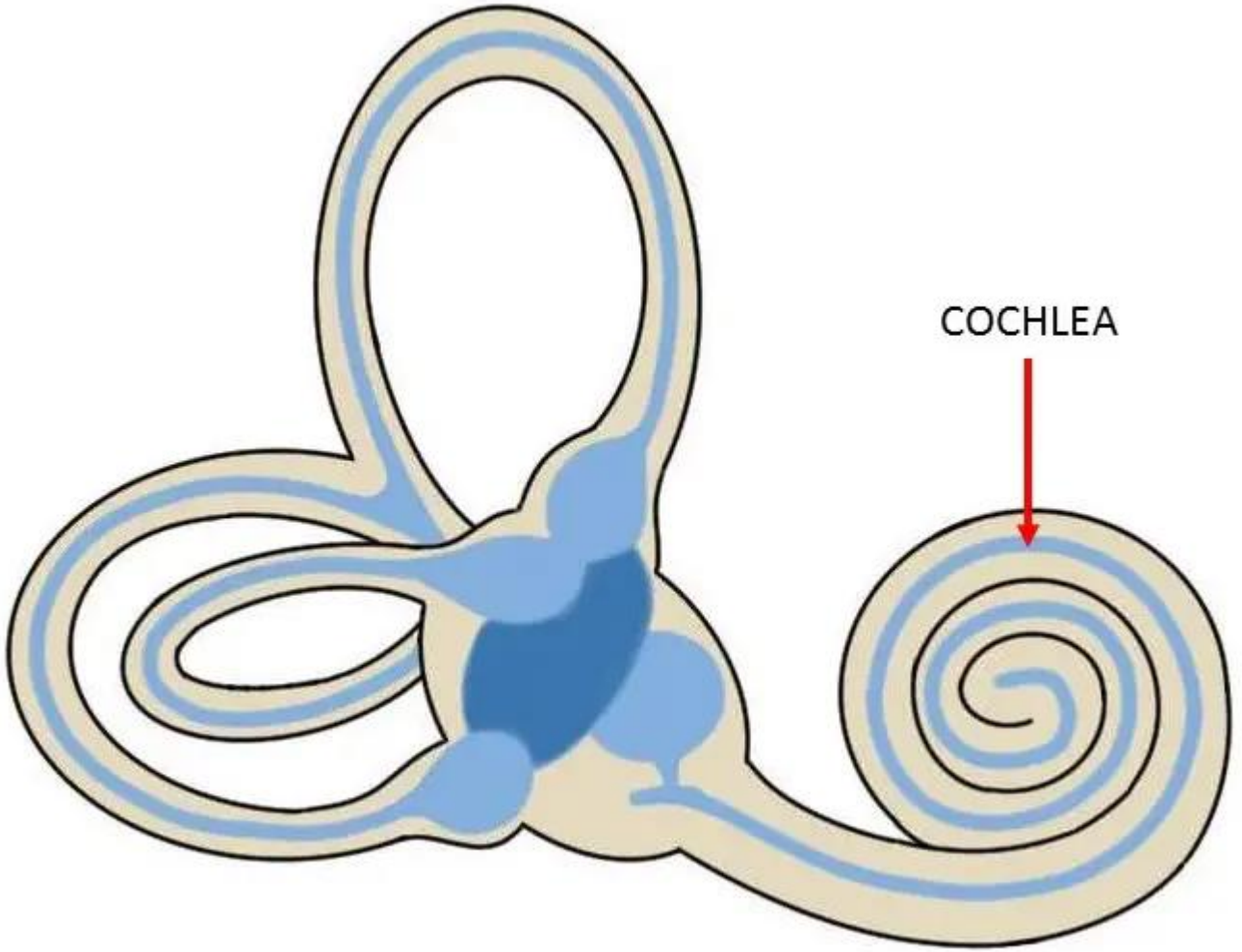


FIGURE 5.46 Cochlear implant.
Sound is picked up by a small microphone (1) located behind the ear and converted into an electrical signal. An external processor (2) converts the signals into complex digital representations of the sound, which travel by wire (3) to an external transmitter (4), which transmits them as radio waves to the internal processor (5). Here they are reconverted to electrical signals that travel by wire (6) to the cochlea, where 22 electrodes are placed. The electrodes stimulate the auditory nerve (7).

- What do these circular/angular canals do?



Vestibular System

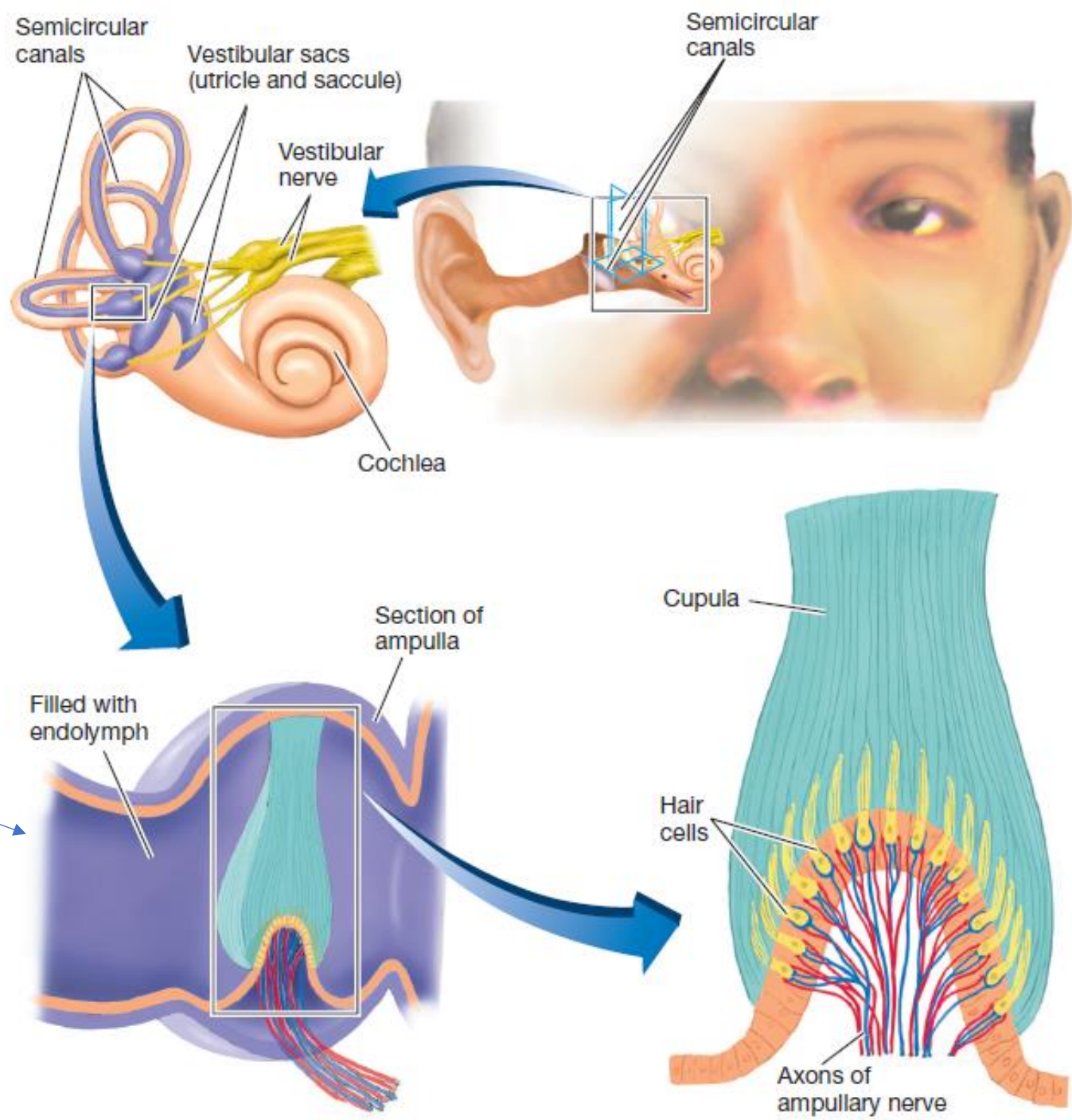
Function - balance, maintenance of the head in an upright position, and adjustment of eye movement to compensate for head movements.

Semi-circular canals

- Angular acceleration → electrical signals
- changes in the rotation of the head
- Arranged in a 3D manner

Vestibular Sacs

- force of gravity → electrical
- inform the brain about the head's orientation

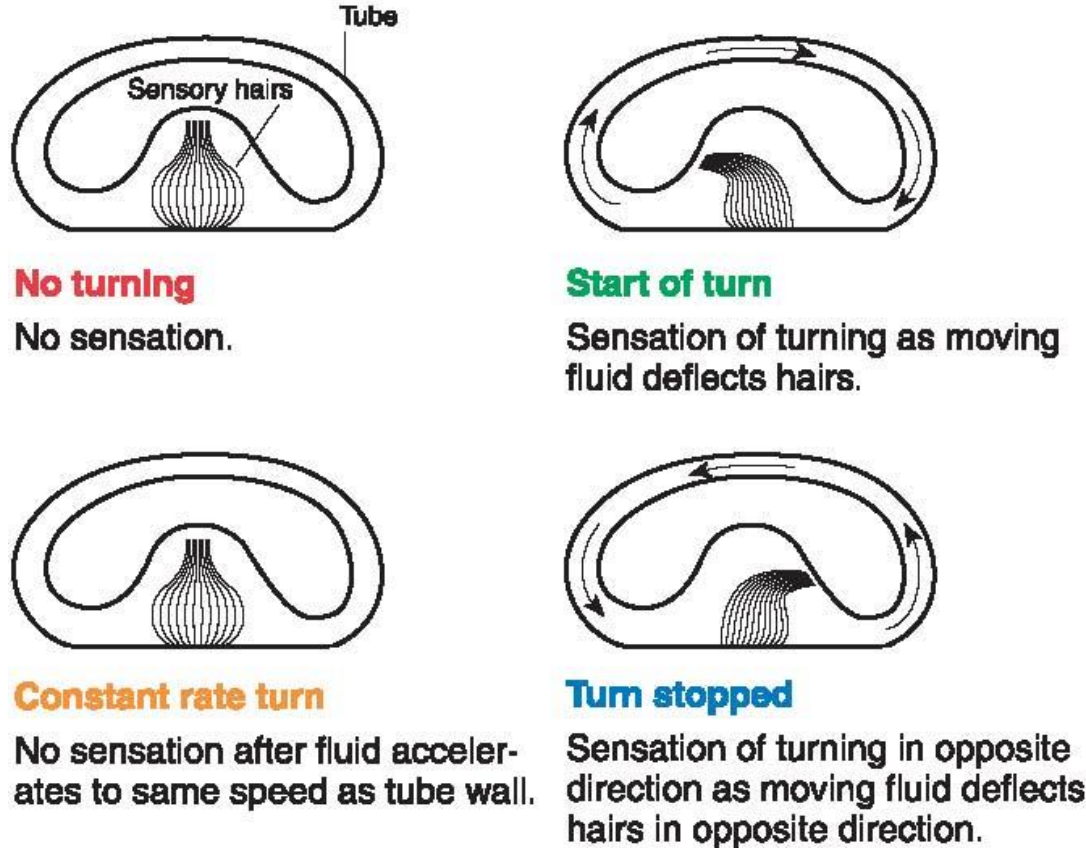


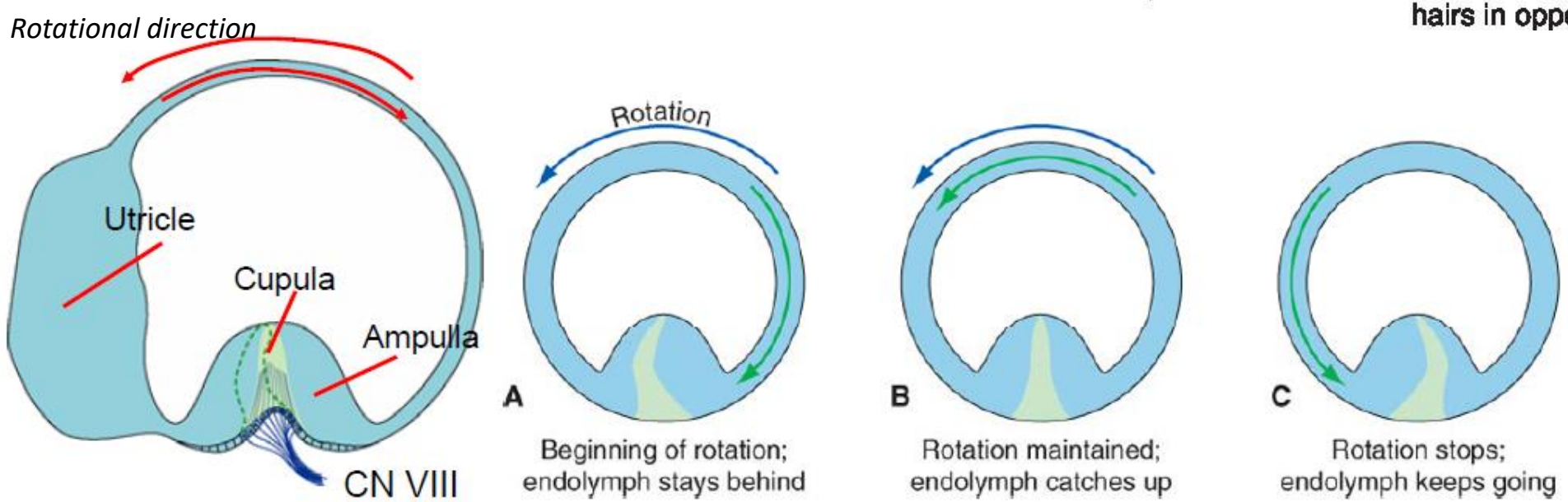
Working of semicircular canals

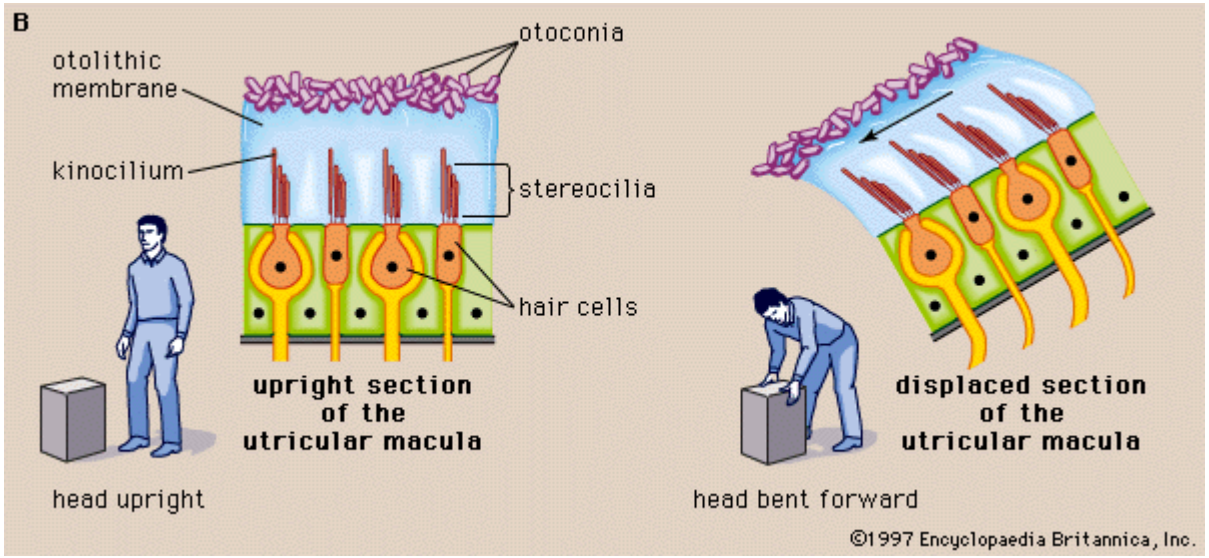
- Rotation of the head → fluid flows in the opposite direction → triggering receptor potentials in the hair cells

- Stimulation of the semi-circular canals can produce dizziness and rhythmic involuntary eye movements (nystagmus)

- Angular acceleration → electrical signals



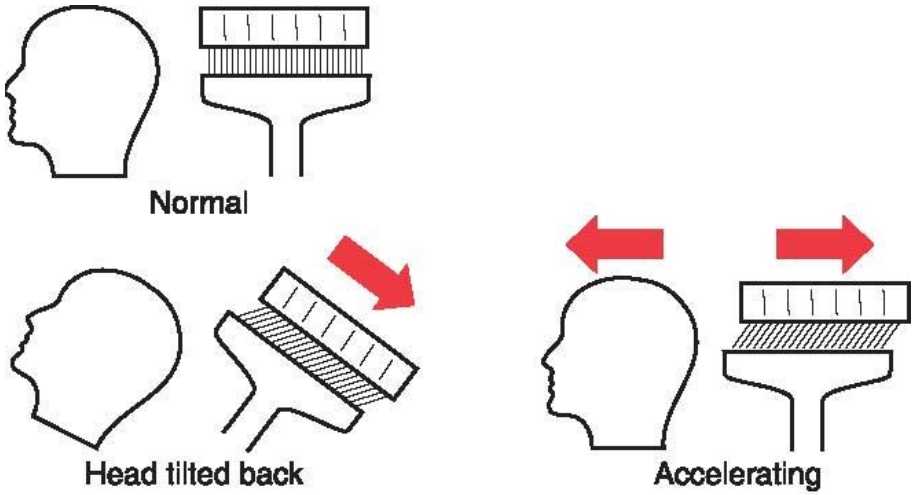




Working of vestibular sacs

- The weight of the crystals causes the gelatinous mass to shift in position as the orientation of the head changes. This movement produces a shearing force on the cilia of the receptive hair cells

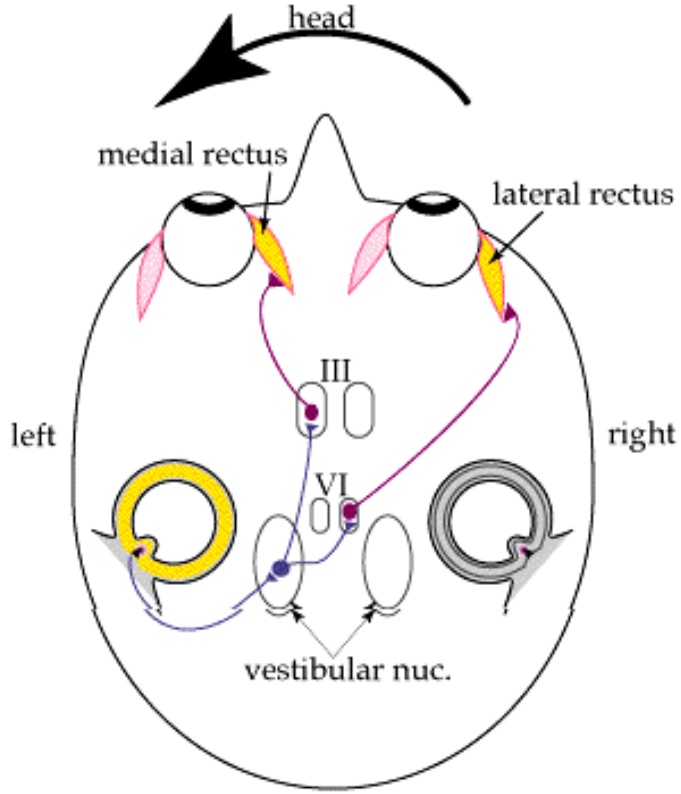
- Low frequency stimulation of the vestibular sacs can produce nausea



Vestibular Pathway:

- To prevent our vision from blurring while walking or running, the brain stem nuclei hold the head upright and control eye movements to compensate for sudden head movements

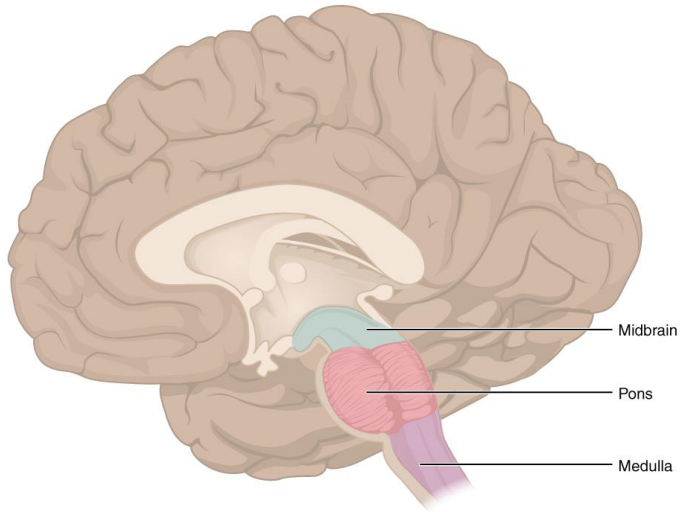
- Activity of vestibular pathway projections to the lower brain stem (medulla) can produce nausea and vomiting that may accompany motion sickness



Connection of vestibular nuclei to eye movements

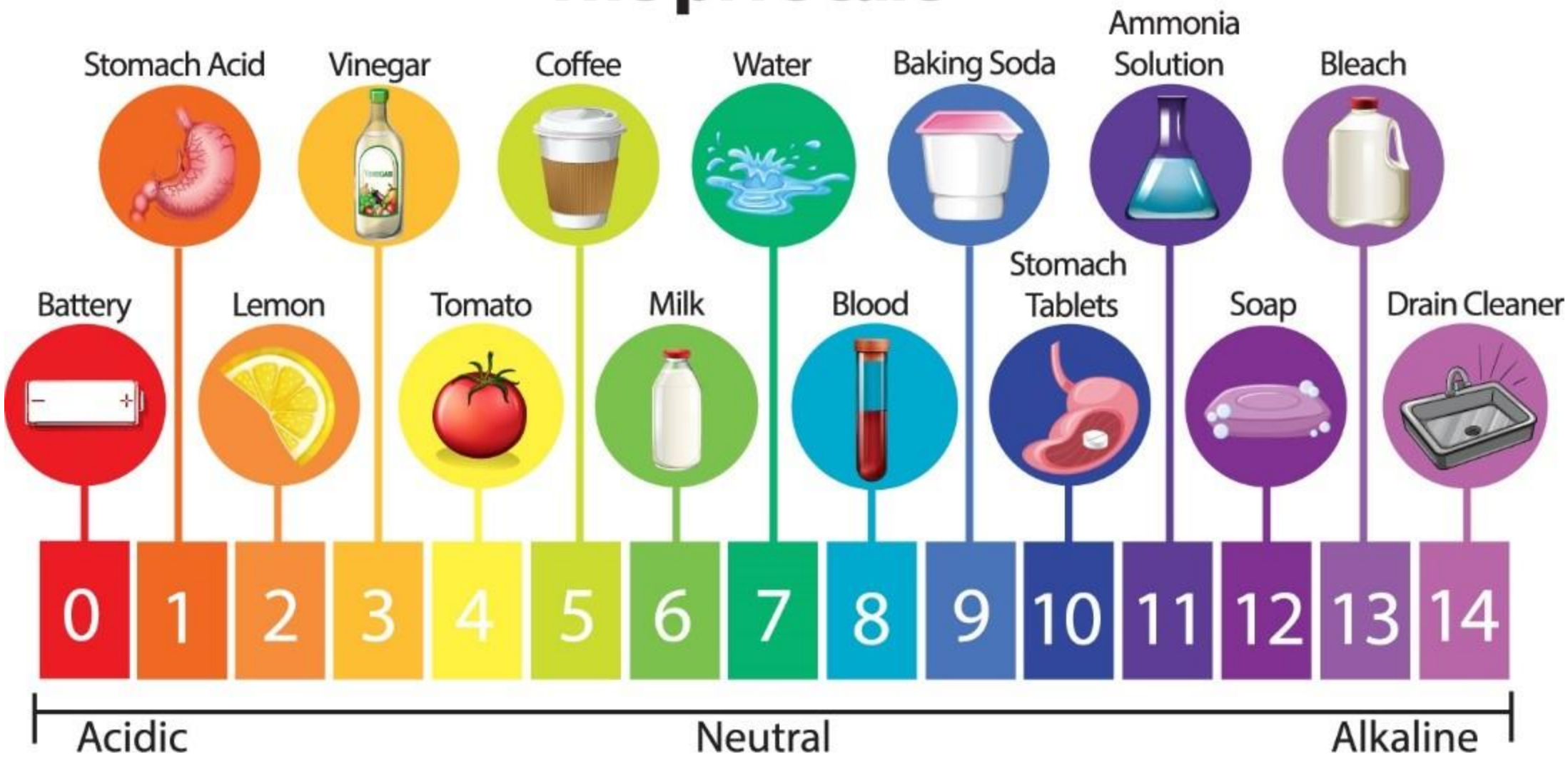
Functions of the vestibular system

- Balance
- Maintenance of head in an upright position
- Adjustment of eye movement to compensate for head movement



Olfaction
Gustation
Somatosenses

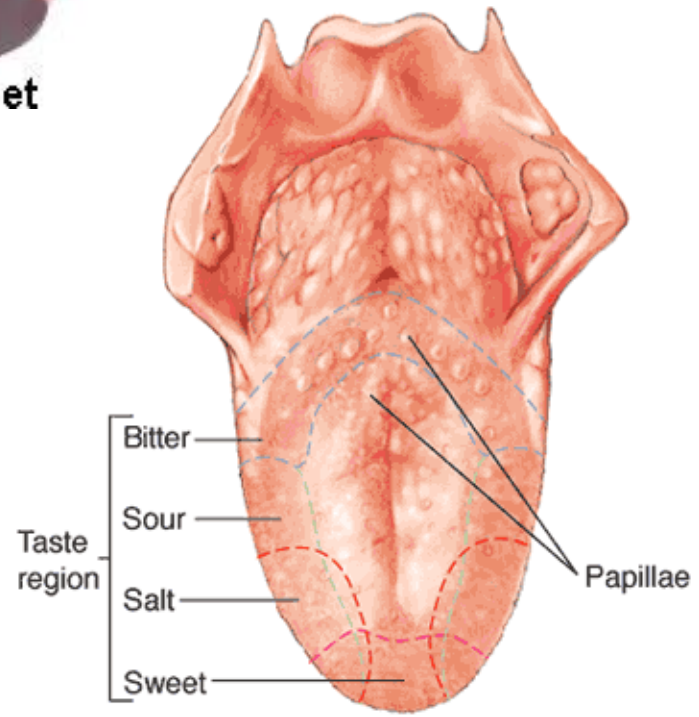
The pH Scale


Stomach Acid
Vinegar
Coffee
Water
Baking Soda
Ammonia Solution
Bleach
Battery
Lemon
Tomato
Milk
Blood
Stomach Tablets
Soap
Drain Cleaner
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
Acidic
Neutral
Alkaline

- Why do we have taste?
- Function?
- Covid effects?
- Why does food taste bland when you are sick?

Gustation

- Substance molecules act like chemicals that activate the taste receptors (chemoreceptors)
- There are 5 qualities of taste:
  1. Bitterness (alkaline, caffeine, polyphenols)
  2. Sourness (Acidic)
  3. Sweetness
  4. Saltiness
  5. Umami: (*Japanese word*) means good taste refers to the taste of monosodium glutamate (MSG), a substance that is found naturally and often used as flavor enhancer in Asian cuisine
     - Umami receptors detect the presence of glutamate, an amino acid found in proteins
- Sweet and salty tastes (amino acids) are preferred over sour (acidic) and bitter (alkaline) tastes
  - Foods that rot due to bacteria are acidic and therefore, taste sour
  - Alkaloids produced by plants to prevent them from being eaten by animals taste bitter

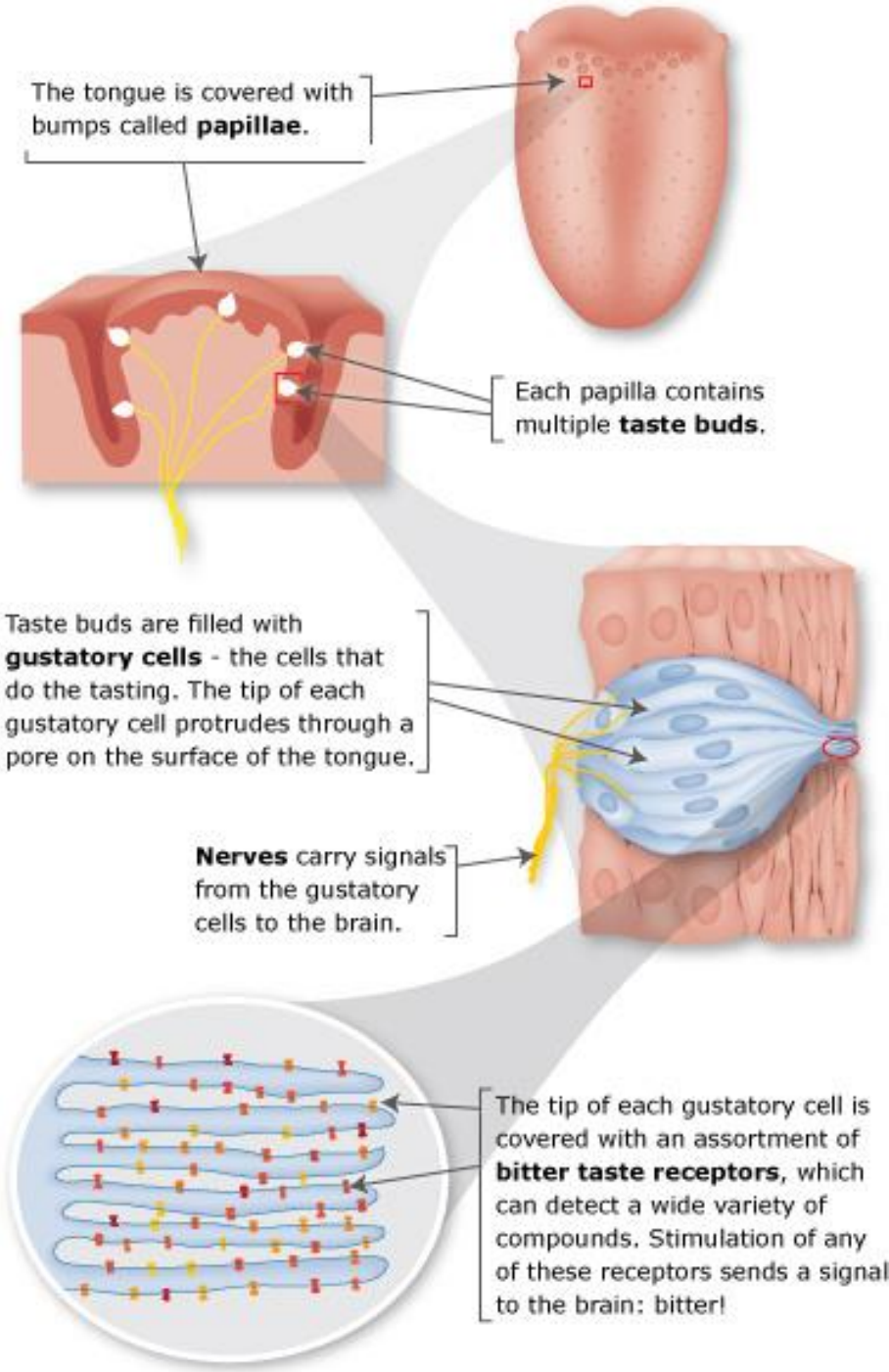


Umami

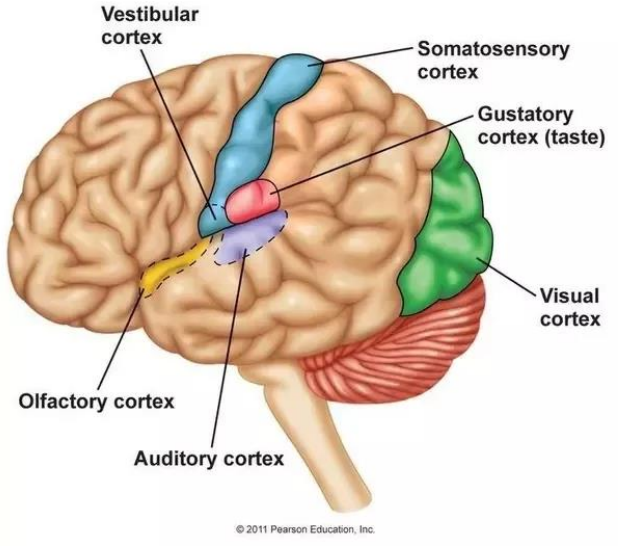
Fat???

Anatomy of taste buds:

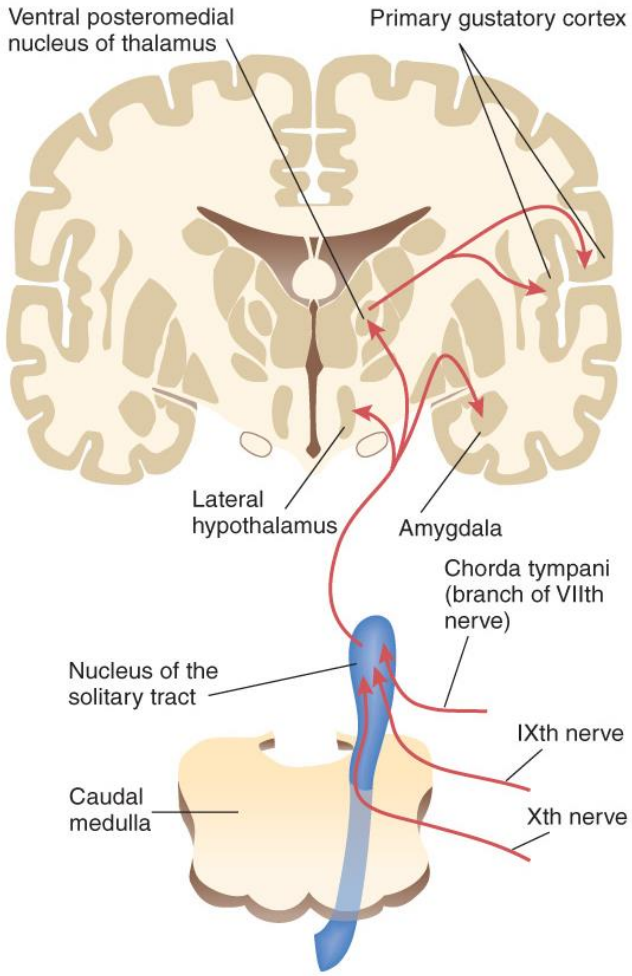
- Papillae on the surface of the tongue contain taste buds
- Taste buds contain groups of 20-50 receptor cells arranged like segments of an orange
- Taste receptor cells are have a life span of only 10 days (renewed regularly)
    - If you burn your tongue you cannot taste food for a few days but you recover your tasting ability after a few days



Gustatory Pathway



Gustatory cortex

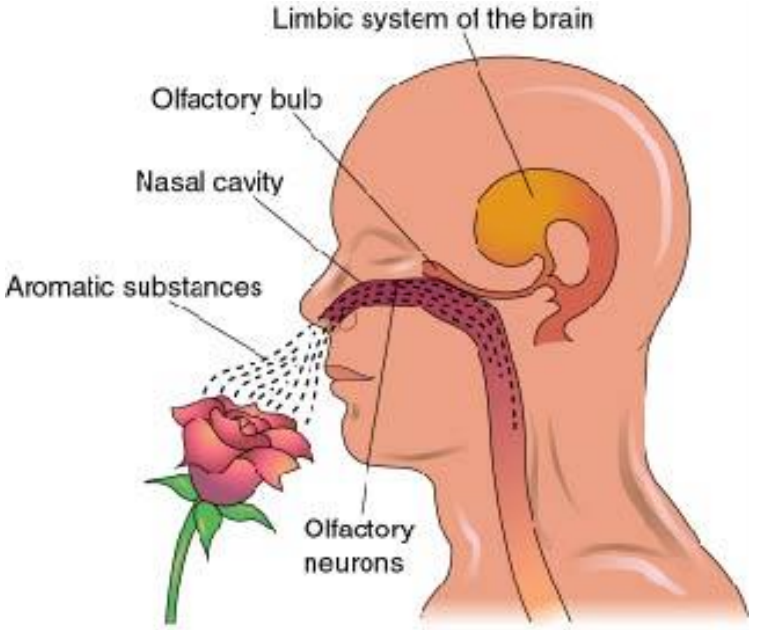


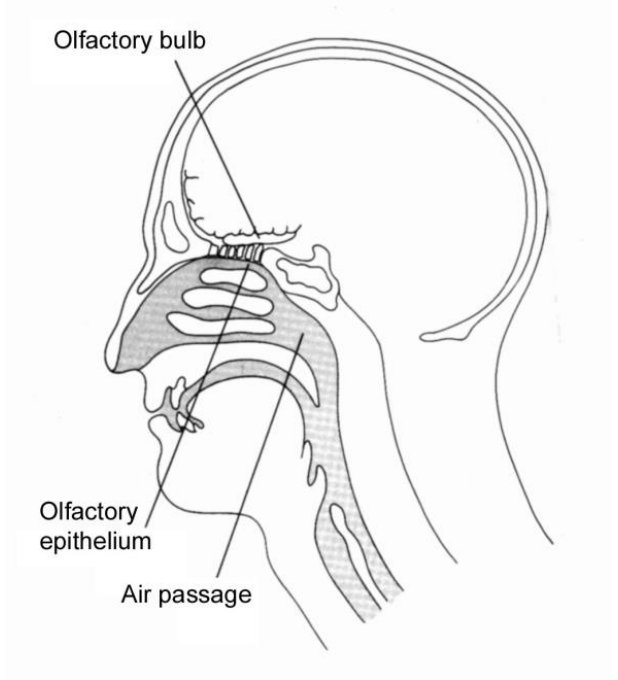
- Ipsilateral
- Different regions in the gustatory cortex code different tastes
- Gustatory information also reaches the amygdala and hypothalamus which may play a role in reinforcing a tastes and feelings of pleasure after eating preferred food
- The gustatory cortex also receives thermal, visceral, and nociceptive (painful) stimuli which plays a role in determining the palatability of food

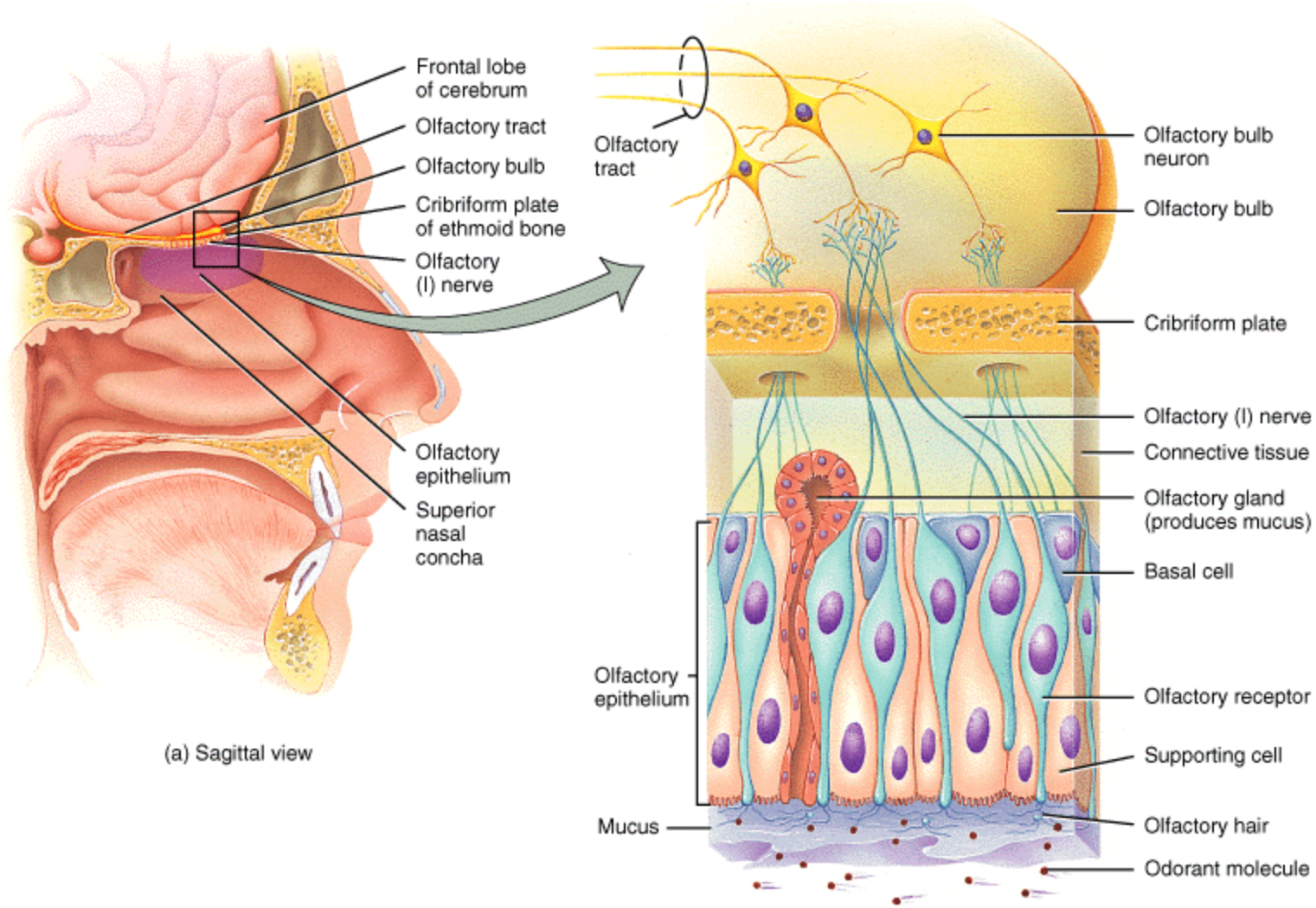
- What is purpose of smell?

Olfaction

- Another chemical sense that helps identify food and avoid spoiled/rotten food
- Taste is not a highly discriminating sense, and there is functional overlap between taste receptor cells. In contrast, odor is highly discriminating.
- Helps other species to track prey
- In humans, smells can evoke memories



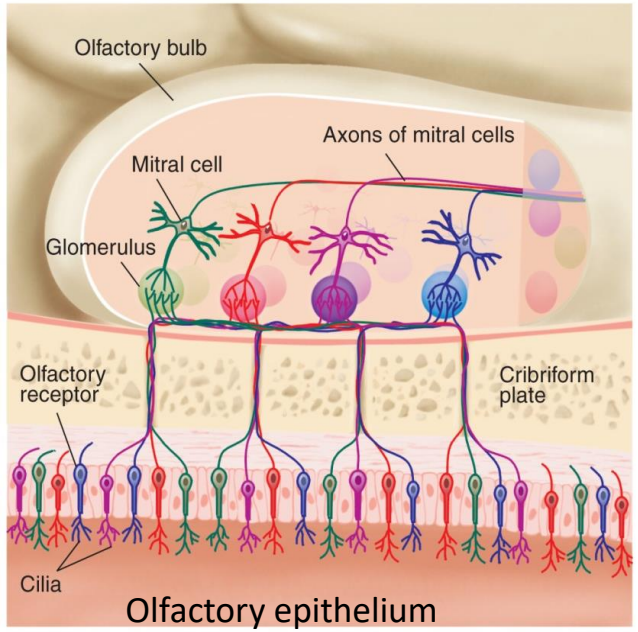




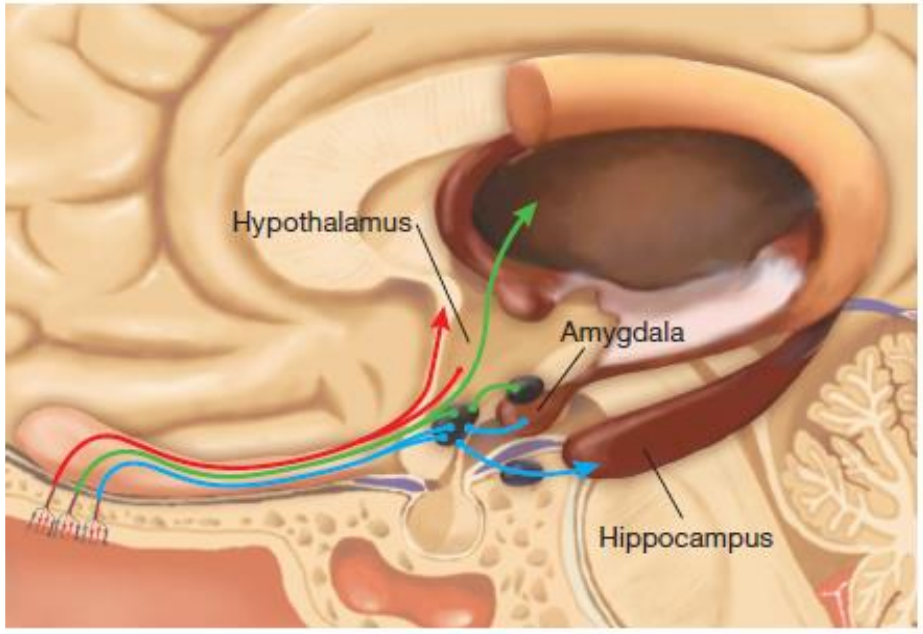
Anatomy of Olfactory System

- We have 6 million olfactory receptor cells on the mucous membrane (olfactory epithelium) inside our nose

- detect painful odorants such as ammonia



Olfactory receptor cells → glomerulus → mitral cells → amygdala (emotional valence) + hypothalamus (reception or rejection of food) + hippocampus (memories)



Encoding Odor

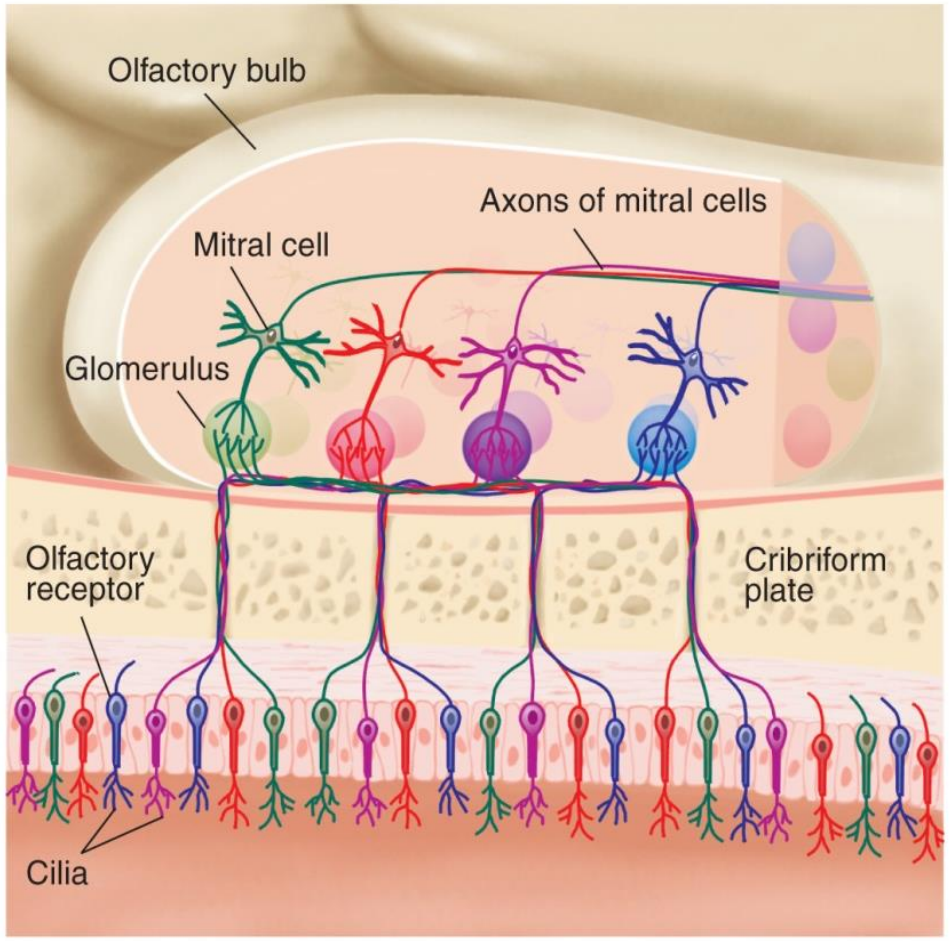
- Each glomerulus receives axons from specific type receptor cells that encode a specific odor.

- each glomerulus receives information from approximately 2,000 olfactory receptor cells detecting the same odor.

- Glomeruli can also inhibit activity in other glomeruli (more active inhibit less active glomeruli)
  - which is why we can mask odors (air fresheners, reuse old food with spices)

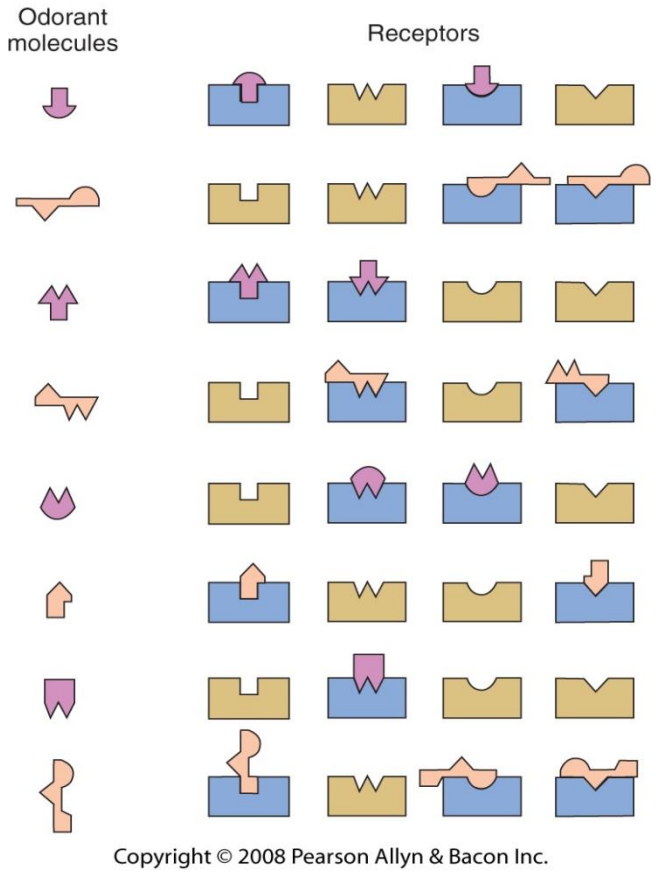
- Humans can recognize up to 10,000 different odors but with only

339 different olfactory receptors. How?

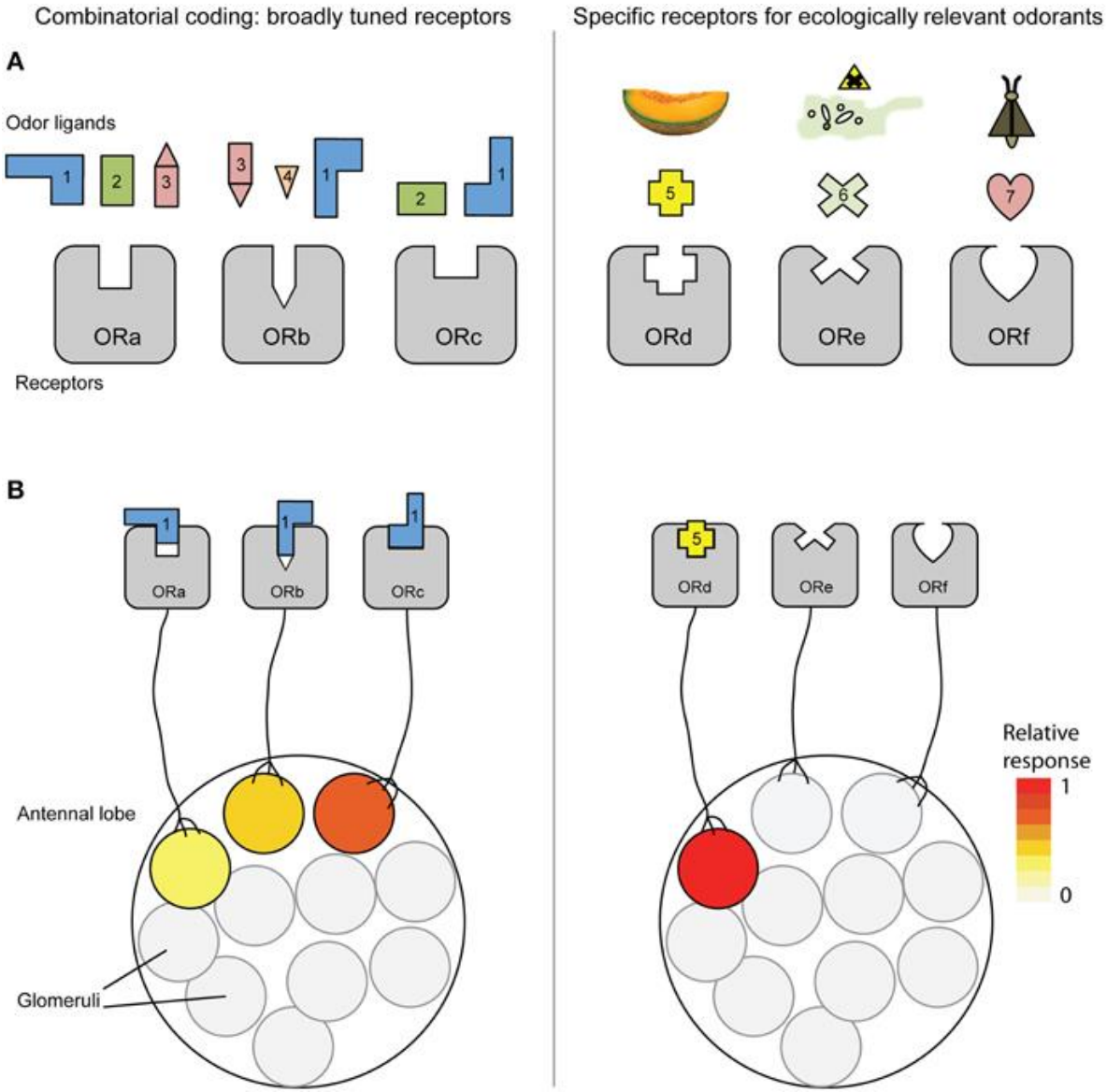


A hypothetical explanation of coding of olfactory information

Discriminating odor



-
- Identification of an odorant is a "lock and key” (different shapes and sizes of molecules) relationship between the odorant and the membrane receptor.
- Odorants bind to more than one receptor – high, moderate or weak binding
- Molecules of an odorant bind with olfactory receptors and open sodium channels and produce depolarizing receptor potentials
- Every odor creates a specific pattern of activation in different glomeruli
- Every pattern represents an odor which is identified by the olfactory cortex



Odorant receptors	1	2	3	4	5	6	7	8	9	10	11	12	13	14	Description
A					●										rancid, sour, goat-like
B		●				●									sweet, herbal, woody
C	●			●	●		●			●	●				rancid, sour, sweaty
D		●			●	●									violet, sweet, woody
E	●			●	●		●	●		●	●	●			rancid, sour, repulsive
F				●	●		●			●					sweet, orange, rose
G	●			●	●		●	●		●		●		●	waxy, cheese, nut-like
H				●	●		●			●		●			fresh, rose, oily floral

MODIFIED AFTER LINDA BUCK AND COLLEAGUES IN CELL VOL 96, MARCH 5, 1999

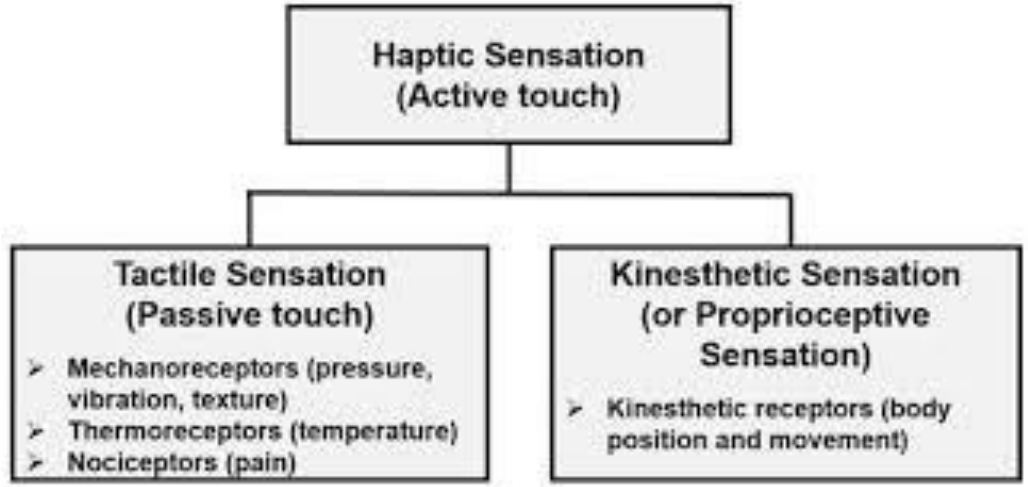
Flavour = olfaction + gustation.

- What is somatosense?
- Function?

Somatosenses

Somatosense	Function
Cutaneous Senses	Provide information from the surface of the body.
Proprioception	Provide information about location of body in space.
Kinesthesia	Provide information about movement of body though space.
Organic Senses	Provide information from in and around internal organs.

- Cutaneous (skin) senses respond to a range of stimuli (Tactile):
  - Pressure, Temperature, Vibration, Pain
- Kinesthetic & Proprioception senses
  - Muscle, skin, and joint movement
- Organic senses
  - Stomach ache, muscle cramps
  - Drinking hot/cold liquid down your throat



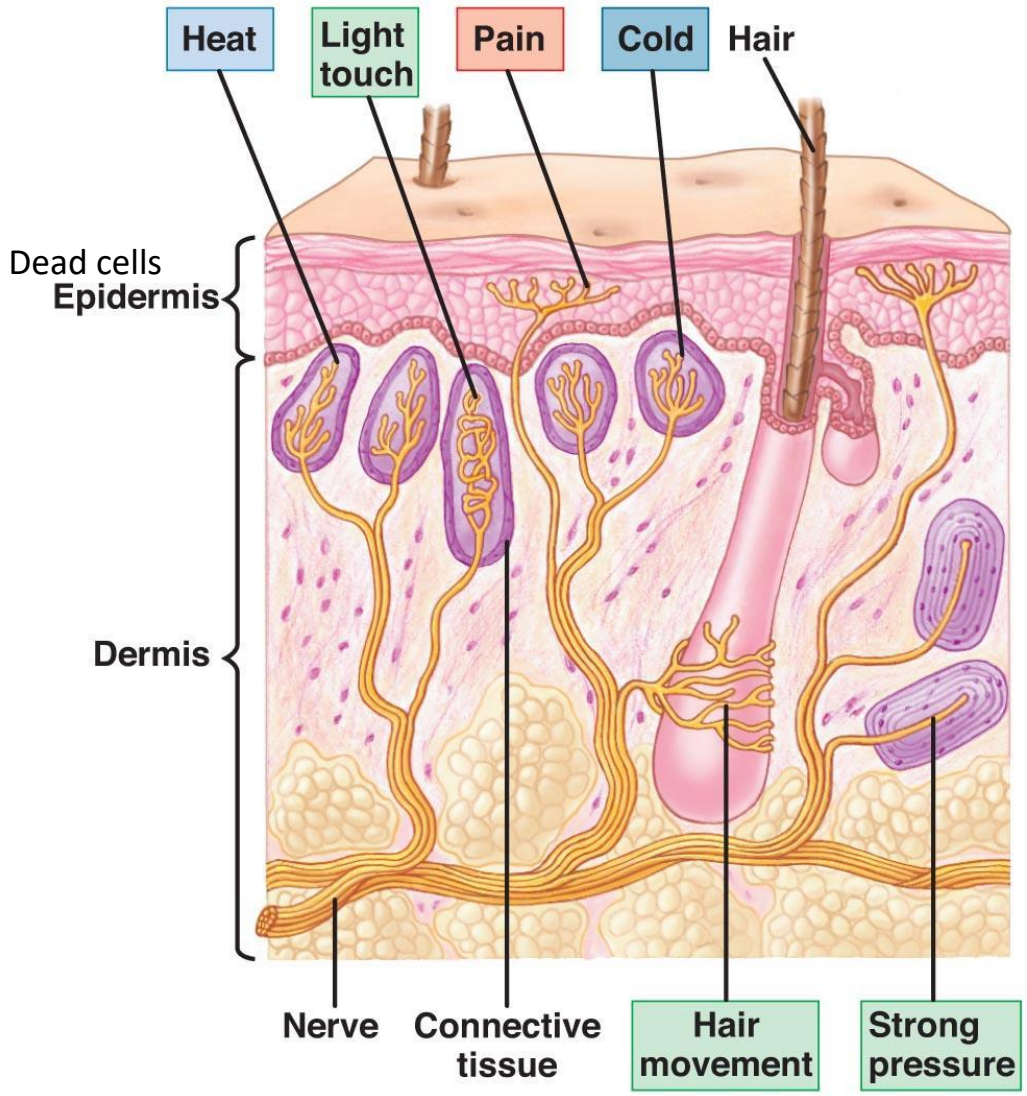
Skin Receptors

Hairless skin (glabrous skin) is found on our fingertips, palms and bottom of toes and feet

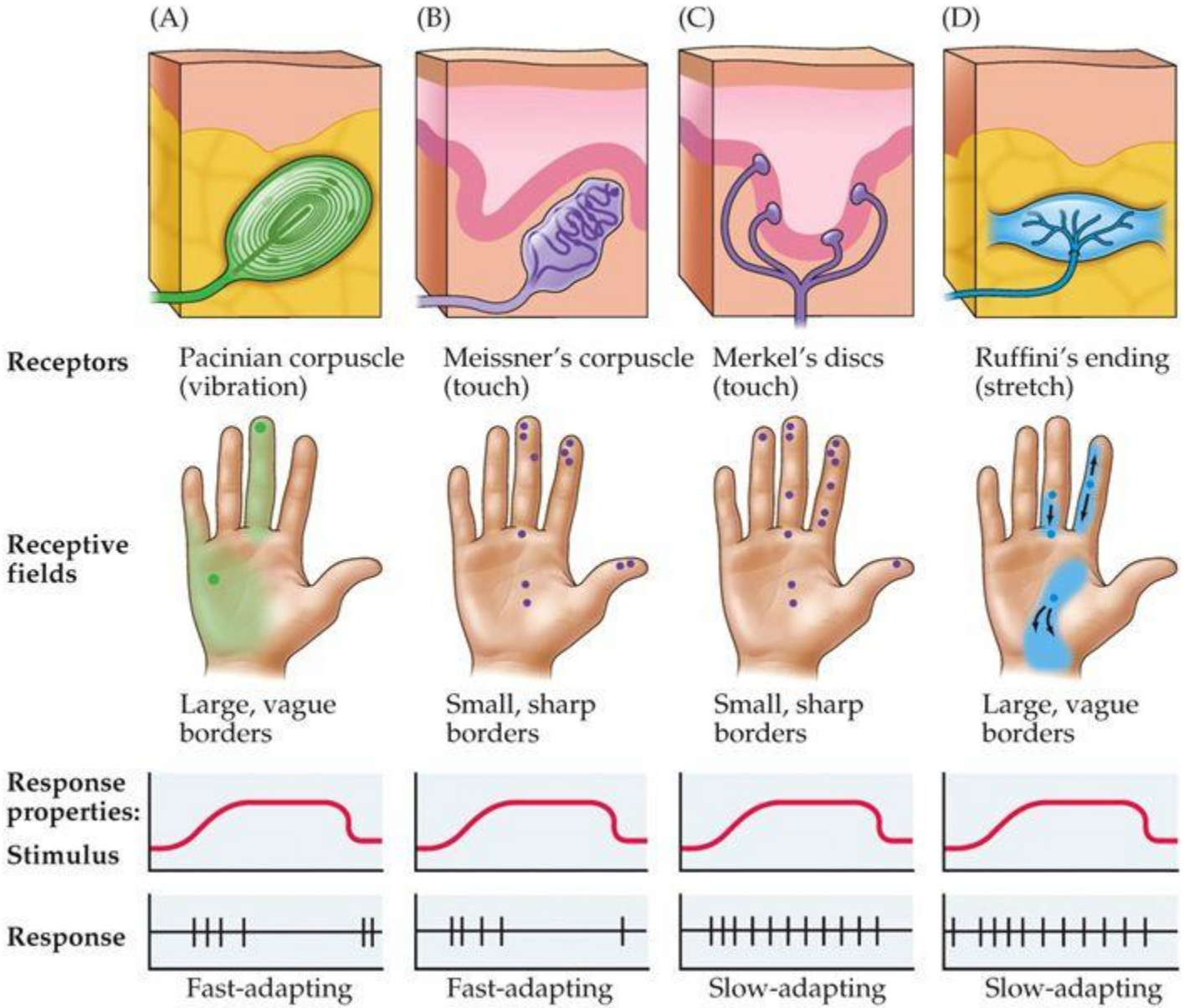
It has a complex mixture of receptors to explore the external environment with our palms and fingers – touching or holding objects

- Glabrous skin receptors
  - Free nerve endings – painful stimuli and temperature changes
  - Ruffini corpuscles – respond to pressure on skin
  - Pacinian - respond to rapid vibrations
  - Meissner's corpuscles – respond to low freq vibration and taps on the skin
  - Merkel endings – respond to touch and light pressures

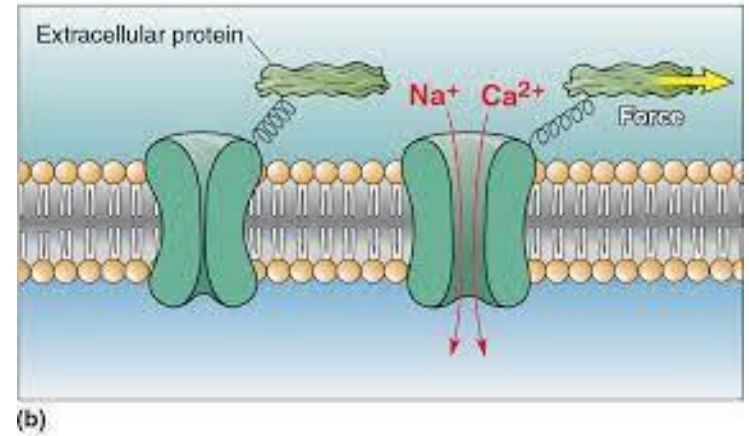
- Hairy skin receptors
  - Free nerve endings – at the base of hair shafts that detect movement of hair
  - Ruffini corpuscles – respond to pressure on skin
  - Pacinian - respond to rapid vibrations

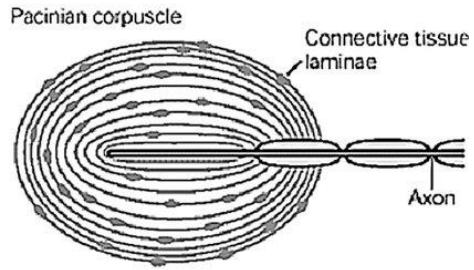


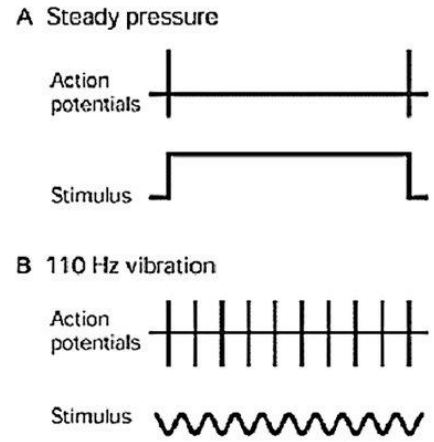
Properties of Skin Receptors Related to Touch

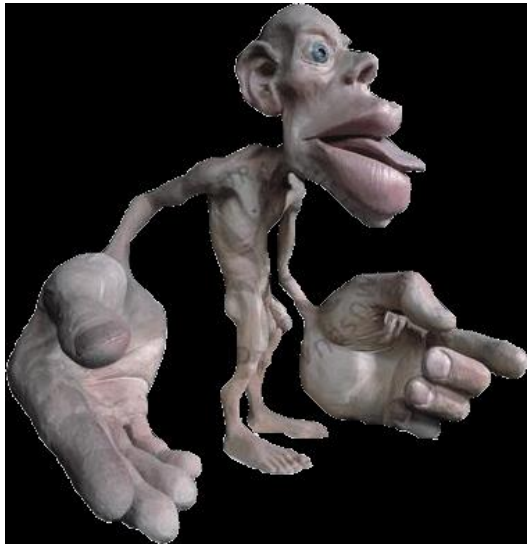


***BIOLOGICAL PSYCHOLOGY 7e*, Figure 8.13**

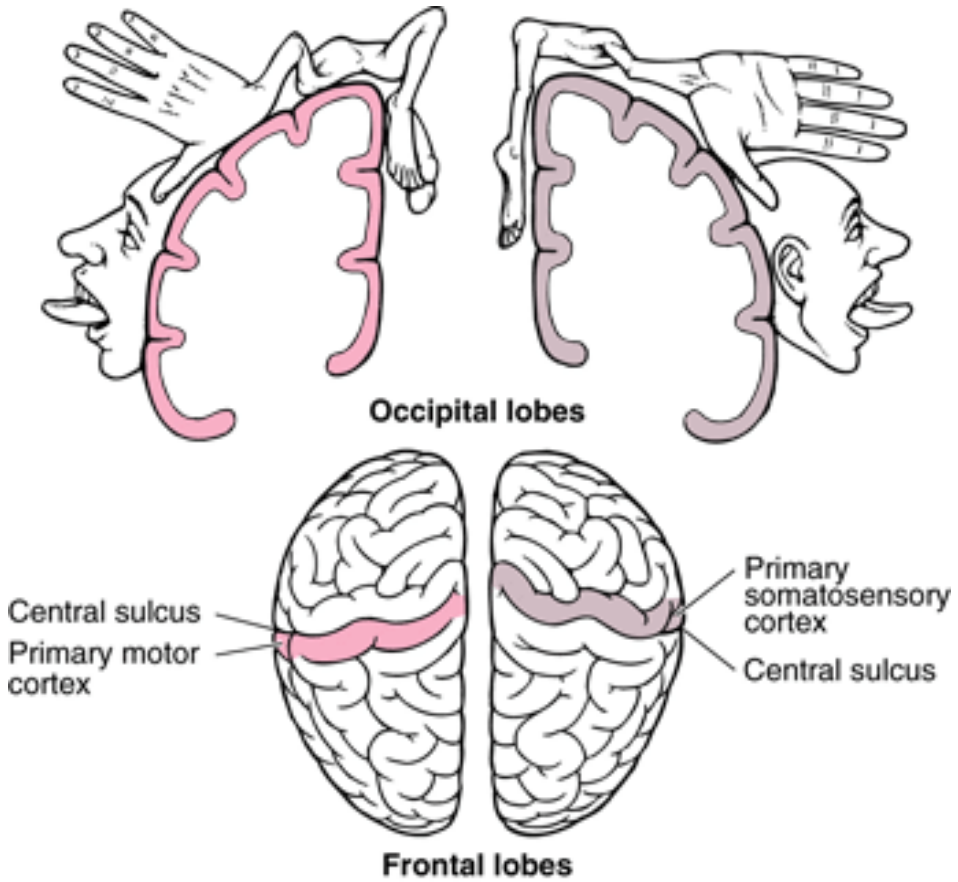


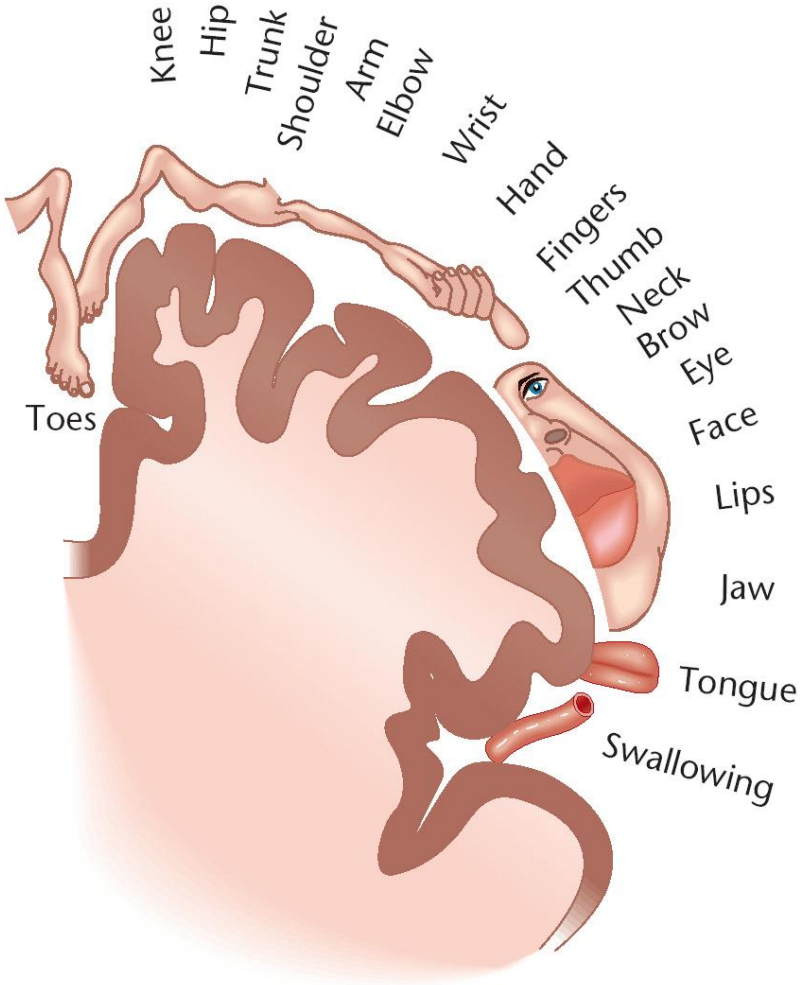


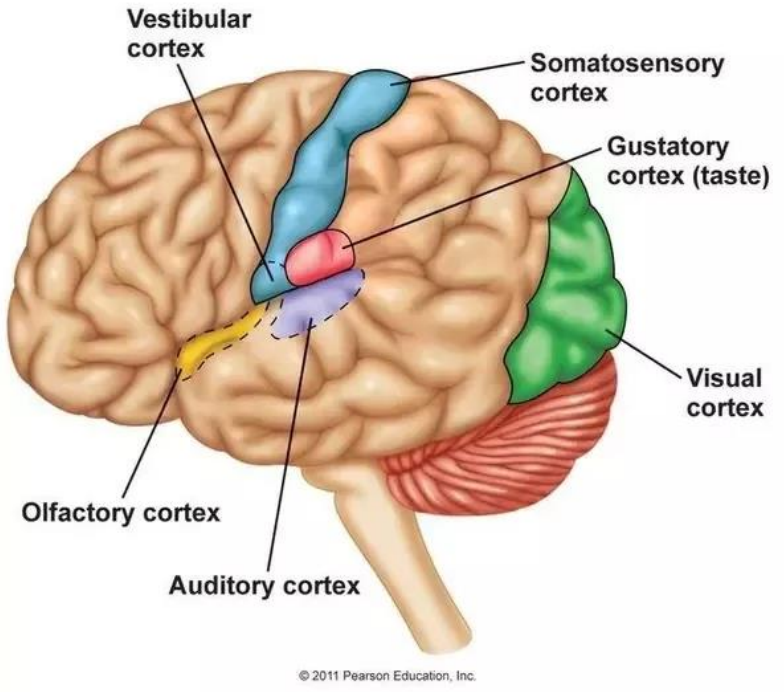


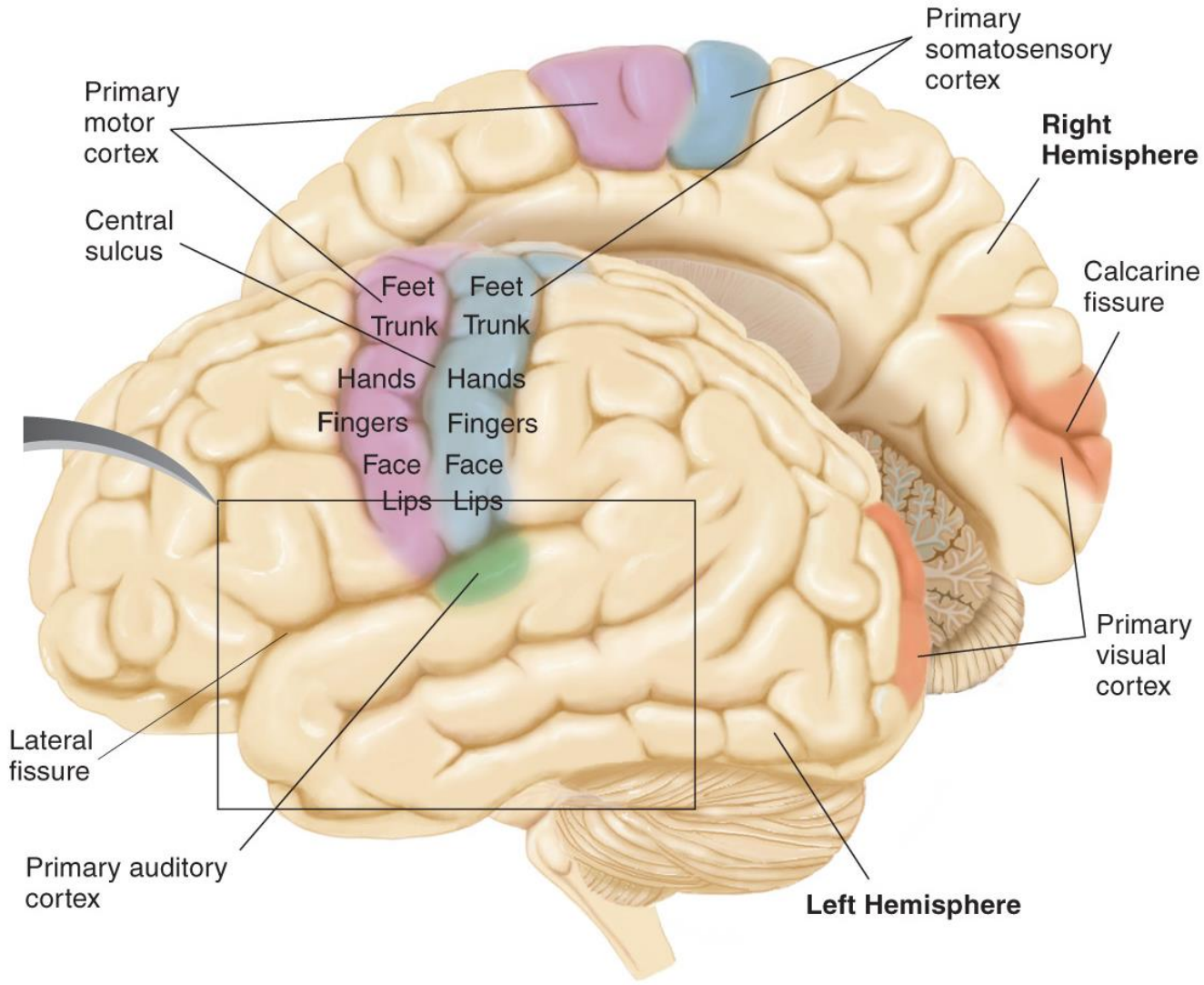


Homunculus – "little human"



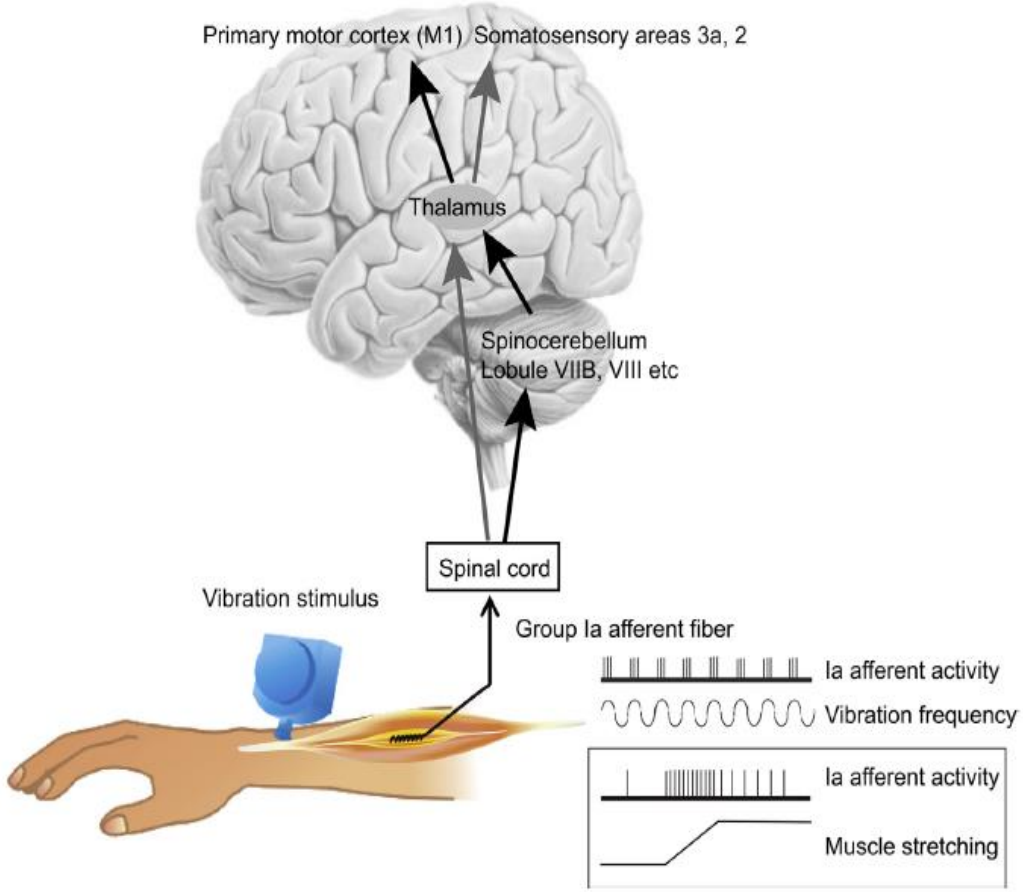






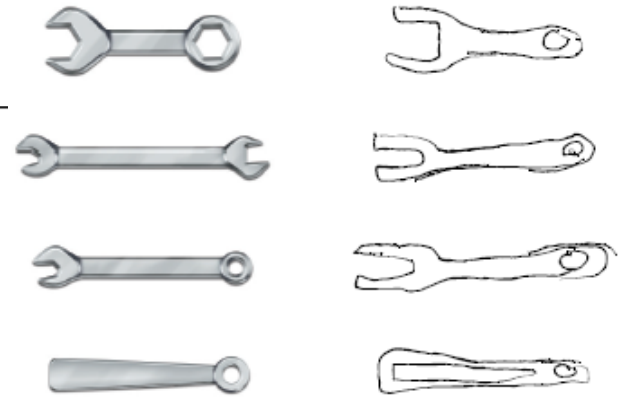
Perception of touch

- Mechanoreceptors respond to mechanical stimulation
- Dendrites of mechanoreceptors are stimulated by pressure and vibration on skin (mechanical energy → electrical energy)
- tactile sensation is localized- receptors are very specific to regions on the skin surface
- fingertips for precise movements like playing the violin need very accurate tactile and proprioceptive feedback
- corresponding contralateral hemispheres in such people allocate more cortex to accomplish such fine tasks

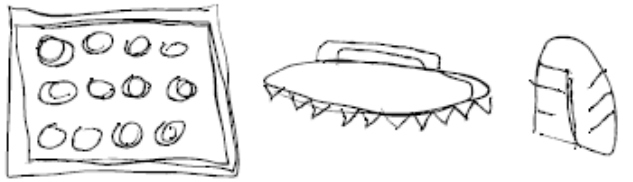


Tactile Agnosia

(a) Drawings of wrenches felt but not seen by M. T. Although the patient did not recognize the objects as wrenches, he was able to draw them accurately. (b) Drawings of objects felt but not seen by E. C. The patient could neither recognize the objects by touch nor draw them accurately.



(a)



Casette tape | Staple remover | Plug adaptor

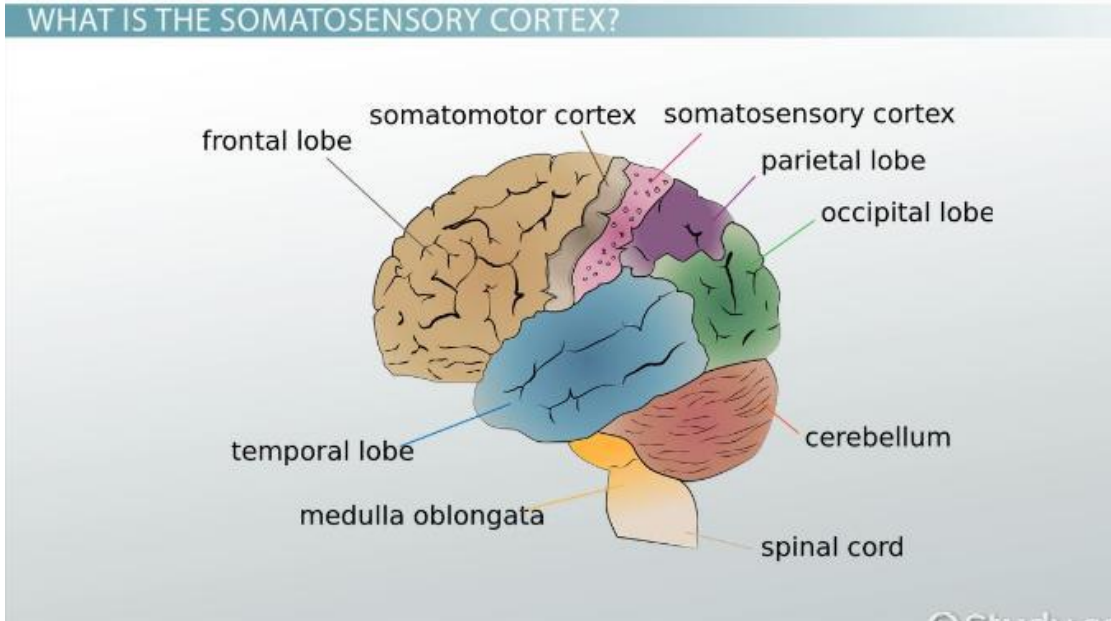
(b)

Tactile Agnosia (Astereognosis)

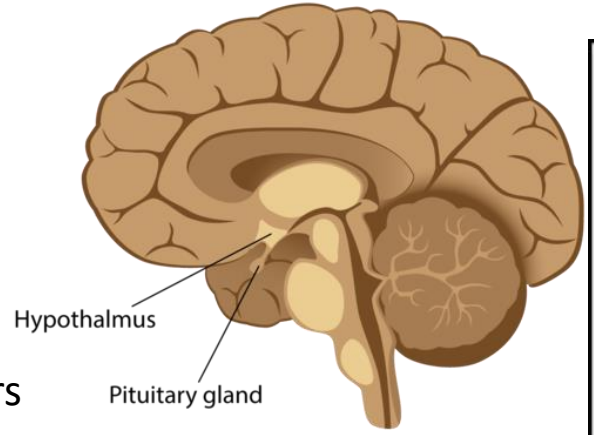
- Inability to identify object by touch (in the absence of visual input).
- Patient can name object by sight, draw object if asked to, and will reach for object if instructed to do so.

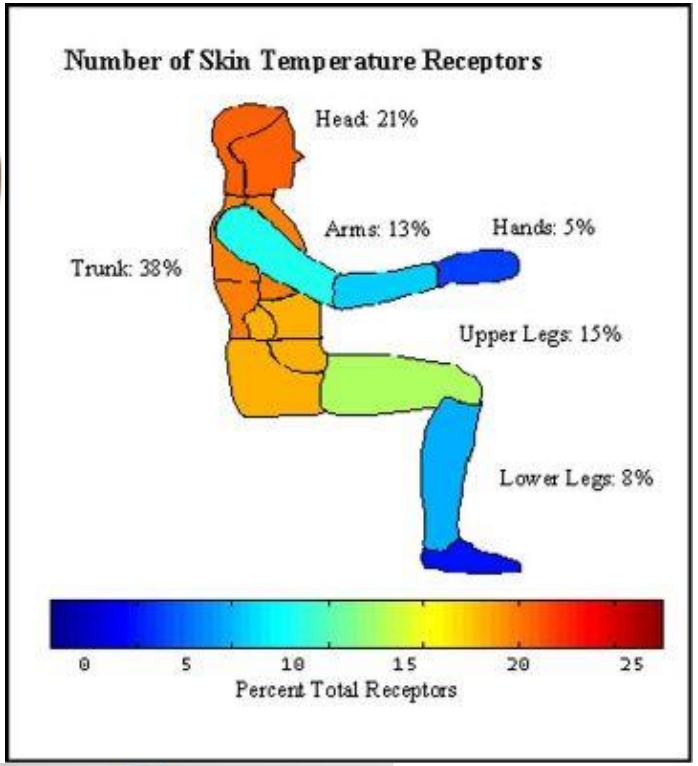
Tactile Agnosia – deficit in tactile perception

Damage to the somatosensory association cortex

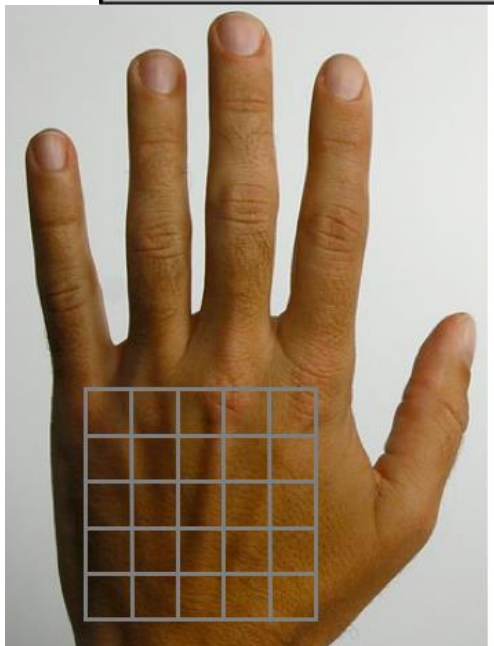


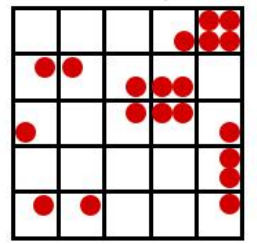
Perception of Temperature

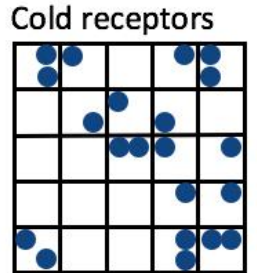


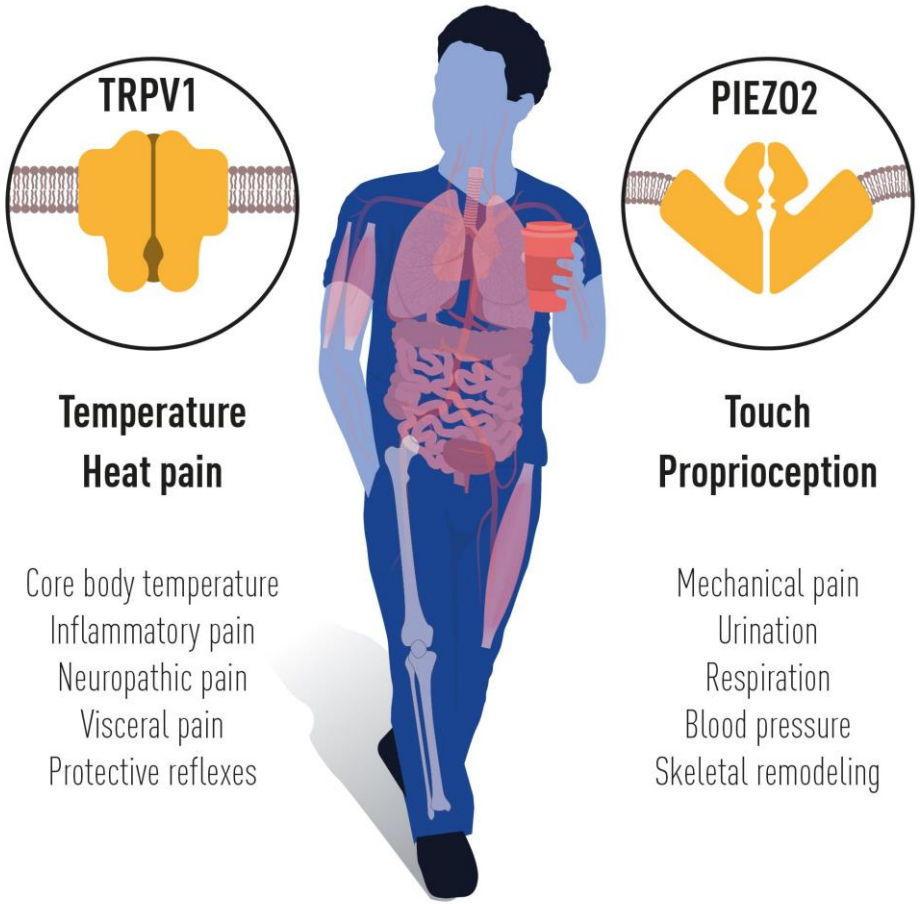


- Two types of thermal receptors – warmth and coolness receptors
- Humans can detect a wide range of temperatures
  - 8° C (noxious cold) to 52° C (noxious heat)
- Anterior hypothalamus → maintaining body temperature
- Our brain adjusts the core body temperature based predominantly upon feedback it receives from the temperature receptors in the torso and head
- Some chemicals can also produce the sensation of warmth or coolness – e.g. mint- coolness, capsaicin - heat
- Accomplished by free nerve endings

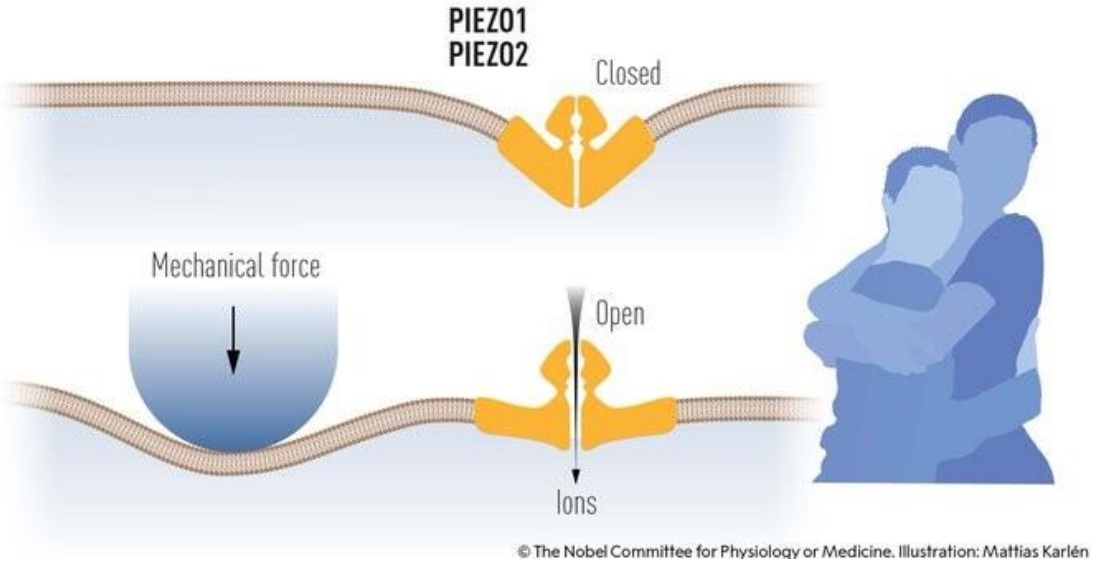


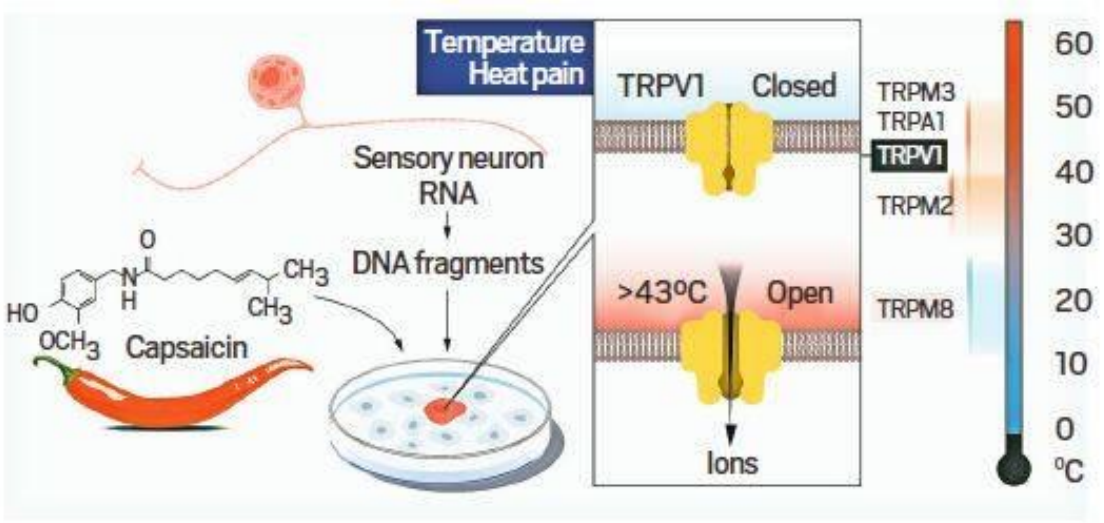






Nobel prize in Physiology/Medicine 2021 –

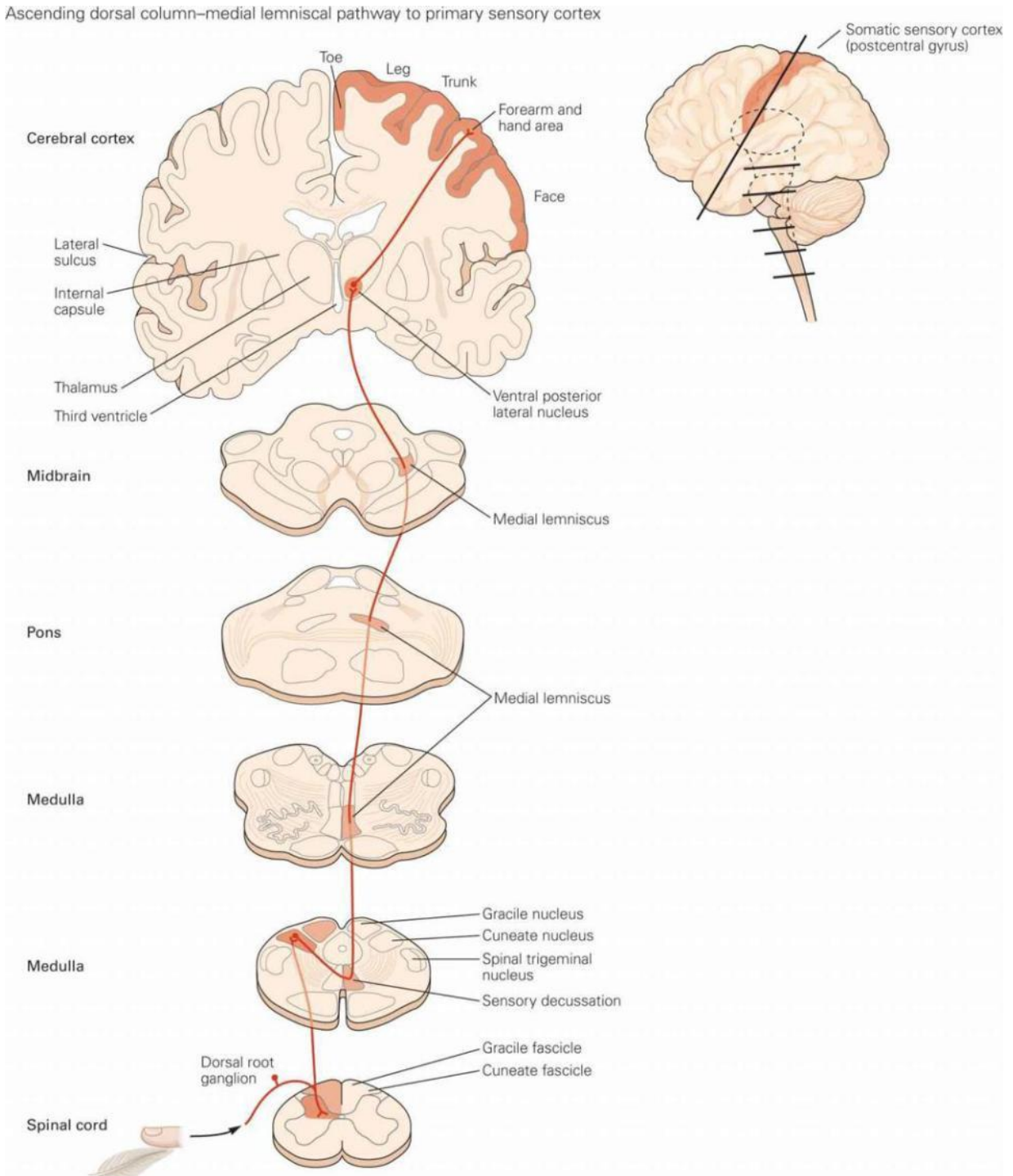




The discovery of the receptor TRPV1 paved the way to the unravelling of additional receptors, which together code for temperature sensation.
nobelprize.org

Perception of Pain

- 3 types of pain receptors
  1. Pain due to intense pressure – striking, excessive stretching or pinching of skin → high threshold
  2. Pain due to extremes of heat to acids and capsaicin (chili peppers) causing inflammation
  3. Pain due to internal injuries or disorders – migraine, damage to muscles, cancer, etc.
- Accomplished by free nerve endings



Types of pain

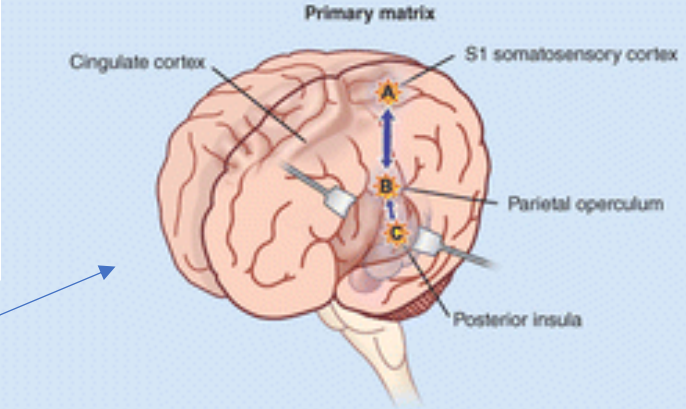
Effects of pain	Brain Regions Involved
Sensory component	Pathway from spinal cord to thalamus to primary/secondary somatosensory cortex
Immediate emotional consequences	Insular cortex, ACC, primary somato-sensory cortex
Long-term emotional consequences	Prefrontal cortex

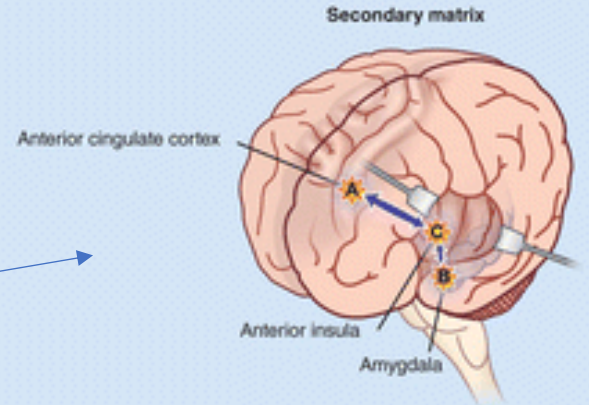
- Pain is a complex phenomenon
- Has 3 different perceptual and behavioral effects

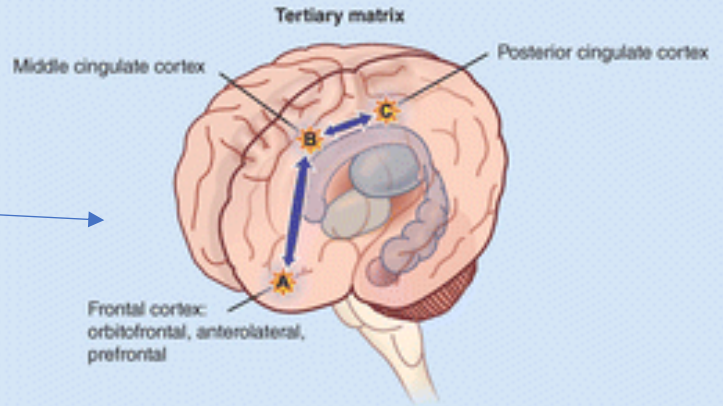
1. Sensory component
   - pure sensation of intensity of painful stimulus
   - Spinal nerves → thalamus → primary somatosensory cortex → secondary somatosensory cortex

2. Immediate emotional reaction
   - unpleasantness following the sensation
   - Spinal nerves → Thalamus → anterior cingulate cortex and insular cortex

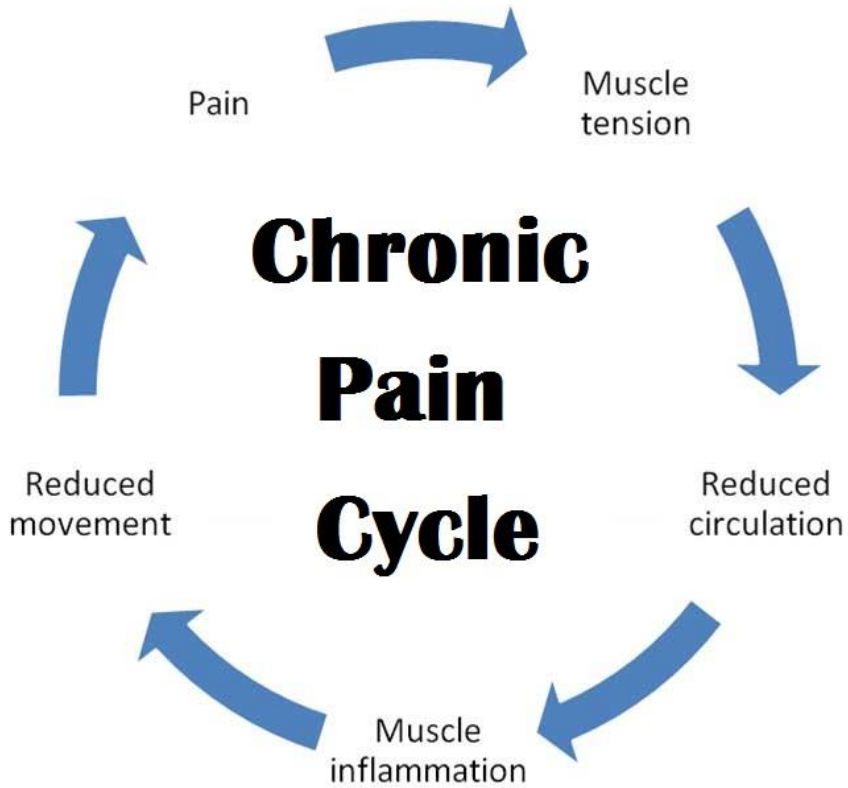
3. Long term emotional implications of chronic pain
   - Well being and comfort
   - Spinal nerves → Thalamus → frontal cortex







What is Chronic Pain?

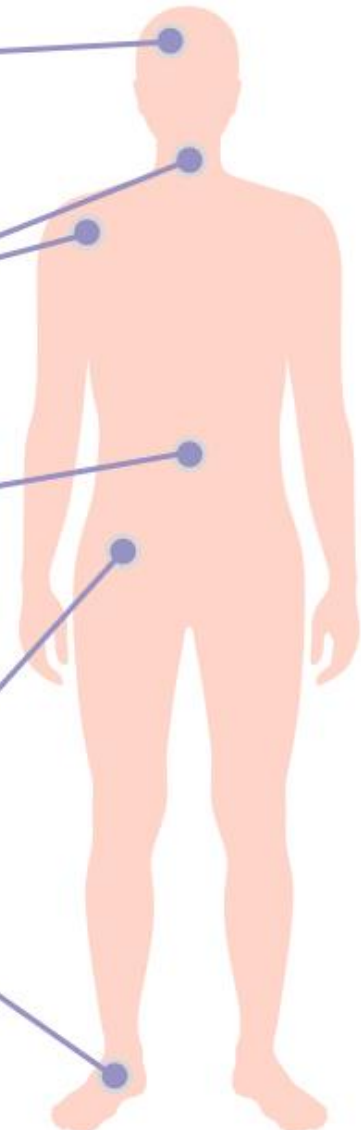


Nerve Pain (neuropathic pain, sciatica, diabetic neuropathy, etc.)

Muscle Pain (hips, legs, neck, shoulders, feet, etc.)

Mechanical/Compressive Pain (spinal cord compression, spinal disc degeneration, tumors, etc.)

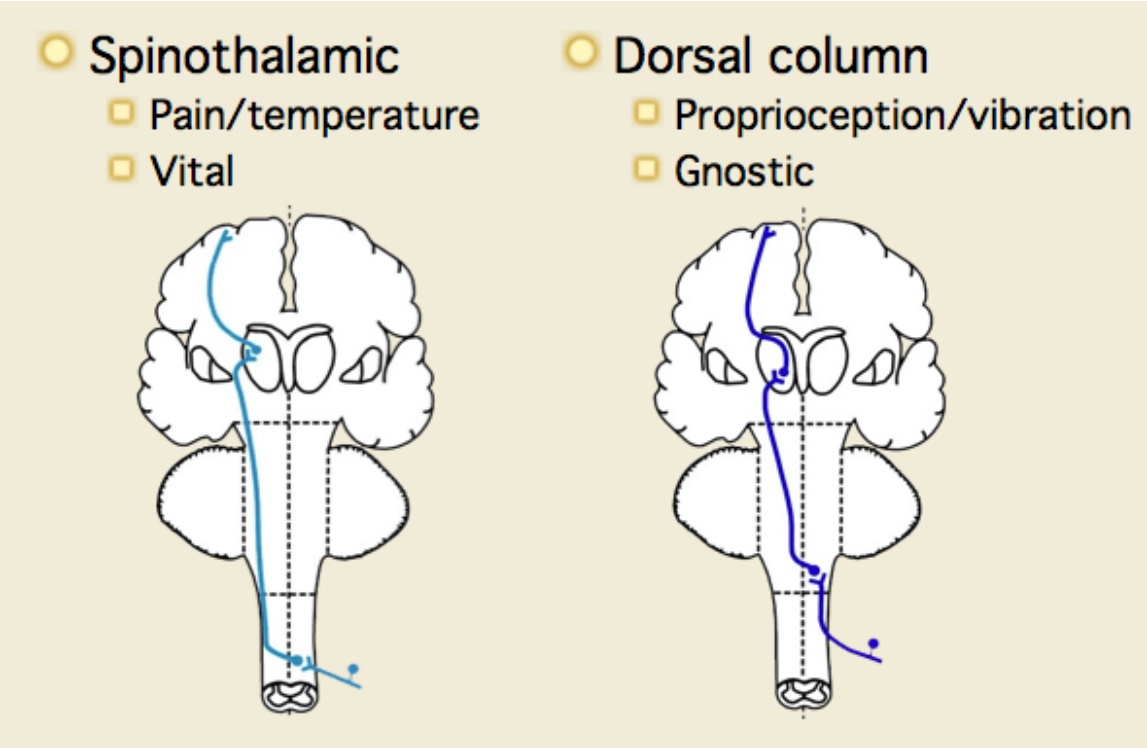
Inflammatory Pain (arthritis, infection, tissue injury, etc.)

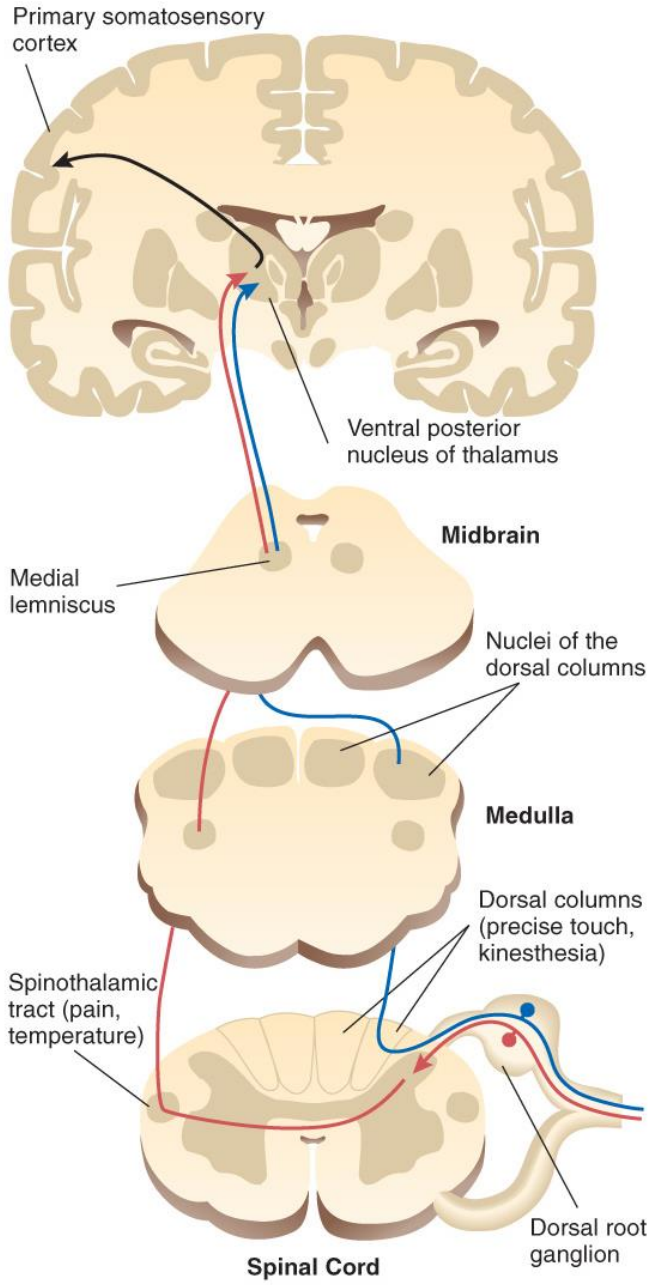


Somatosensory pathways

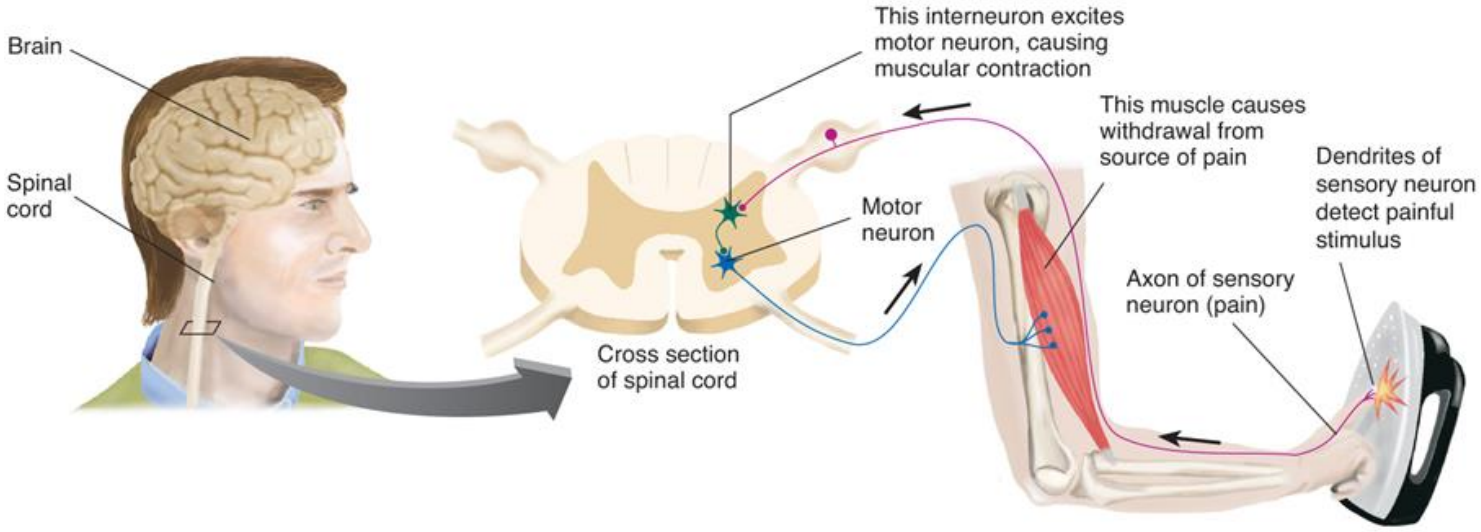
- Sensory information crosses the spinal cord at various levels to reach the contralateral hemisphere

Spinal cord injuries??

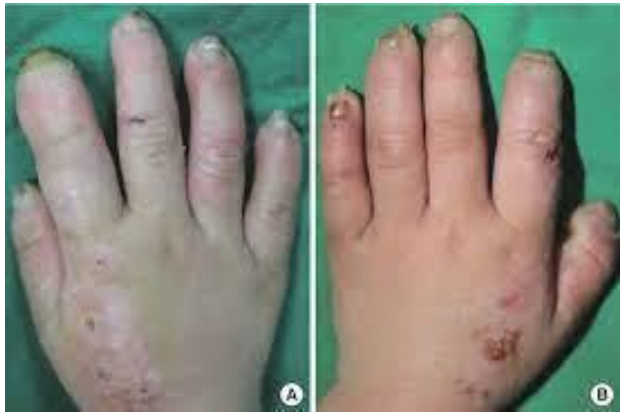




Simple withdrawal reflex



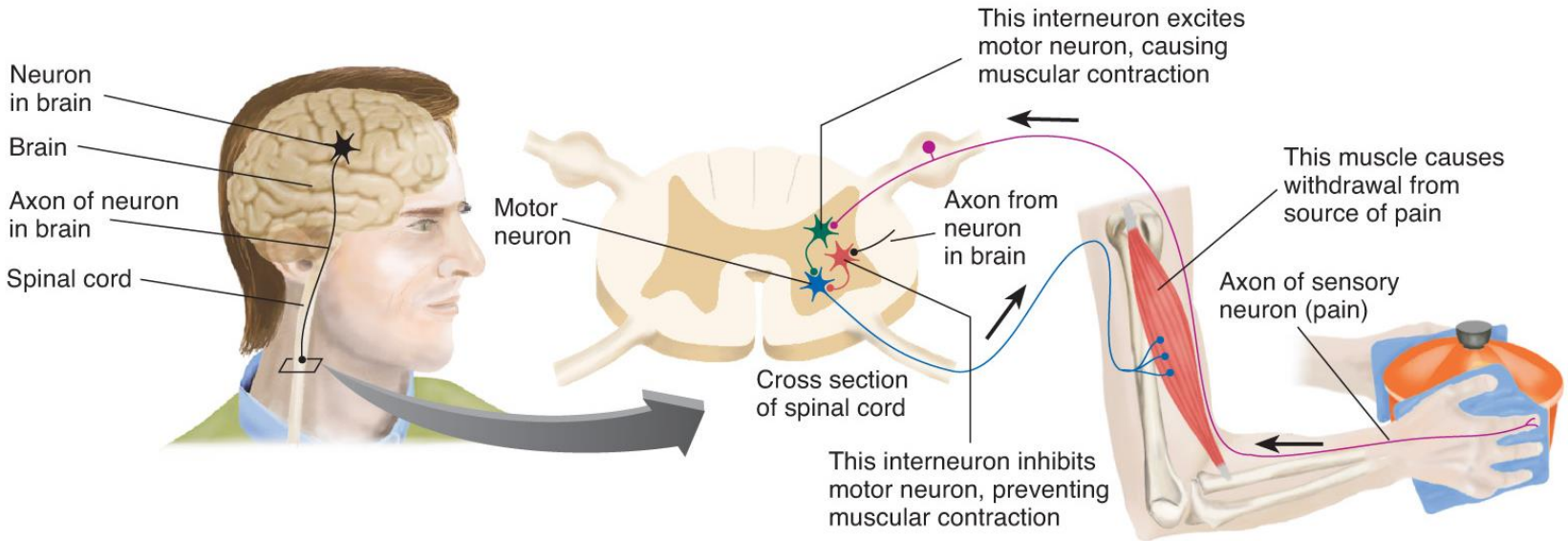
Congenital Insensitivity to Pain



The terminal boutons of the sensory neuron release a neurotransmitter that excites the interneuron, causing it to send messages down its axon. The terminal boutons of the interneuron release a neurotransmitter that excites the motor neuron. The axon of the motor neuron travels to join the muscle. When the motor neuron releases neurotransmitter, the muscle cells contract, causing the hand to move away from the hot object.

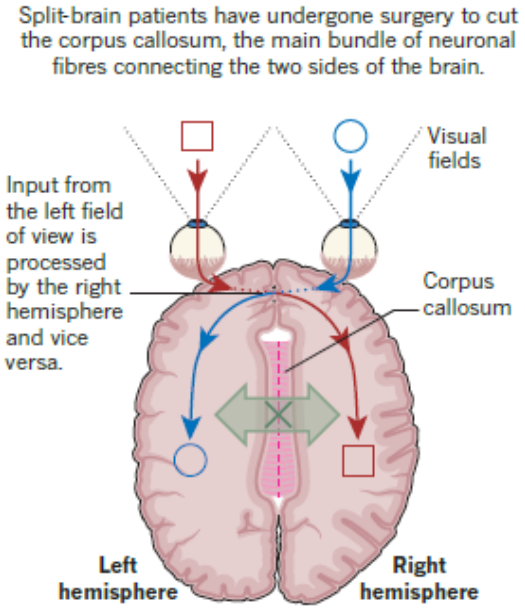
All synapses have **excitatory** effects

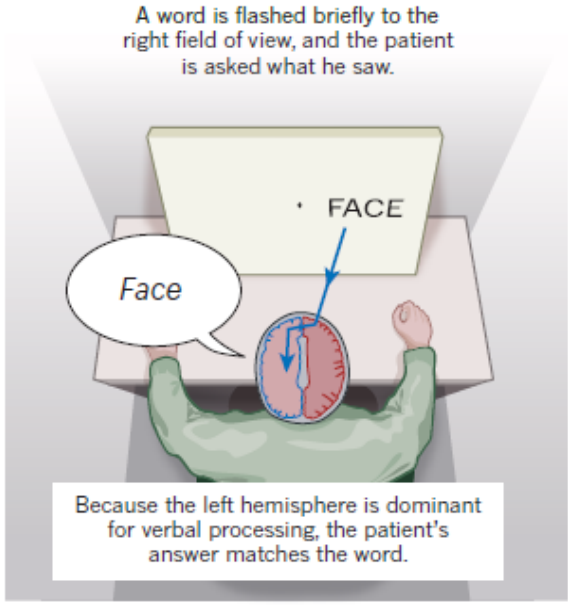
Preventing withdrawal reflex

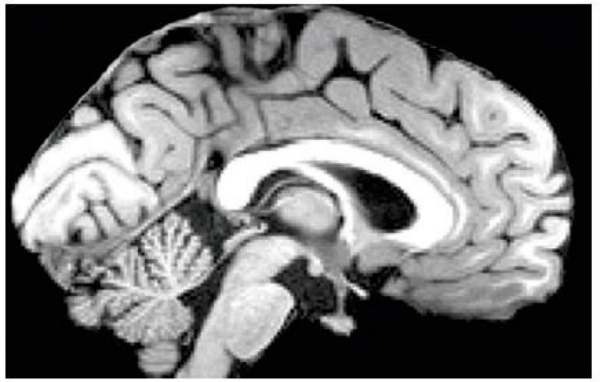


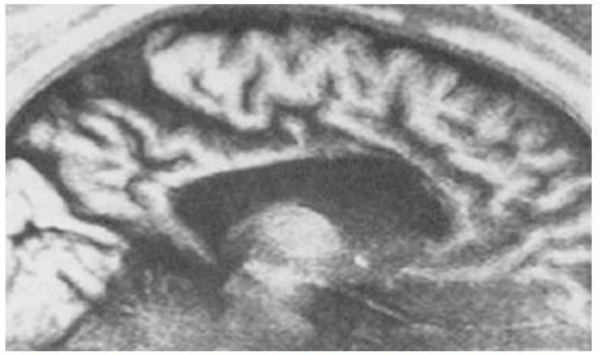
The pain from the hot casserole increases the activity of excitatory synapses in the withdrawal reflex, which tends to cause the hand to pull away from the casserole. However, this excitation is counteracted by **inhibition**, supplied by the brain. The brain contains neural circuits that recognize what a disaster it would be if you dropped the casserole on the floor. These neural circuits send information to the spinal cord that prevents the withdrawal reflex from making you drop the casserole. The brain basically excites an inhibitory interneuron which decreases the activity of the motor neuron that was going to withdraw the muscle, i.e. blocks the withdrawal reflex. This is an example of two competing tendencies

Split brain





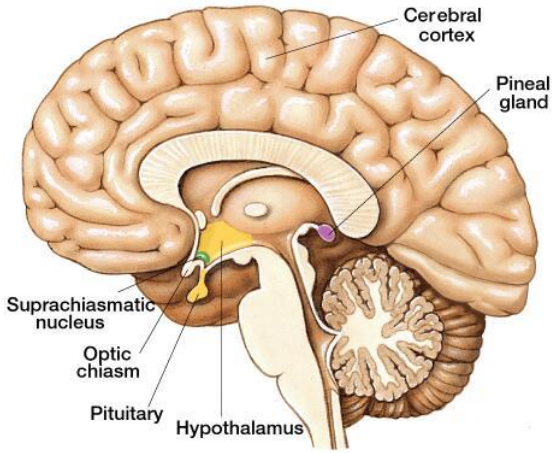


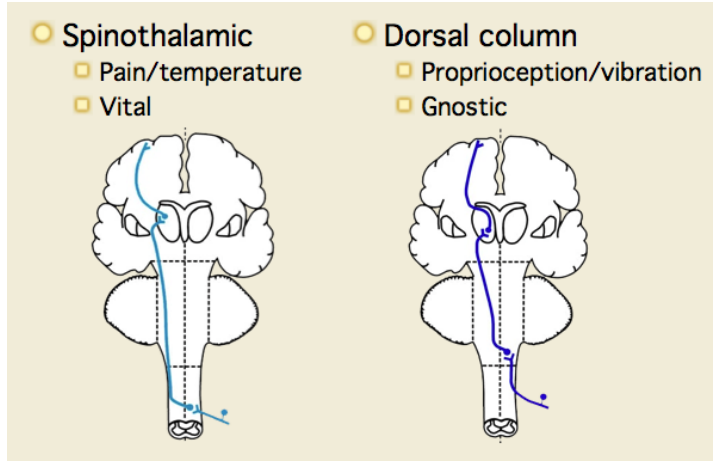


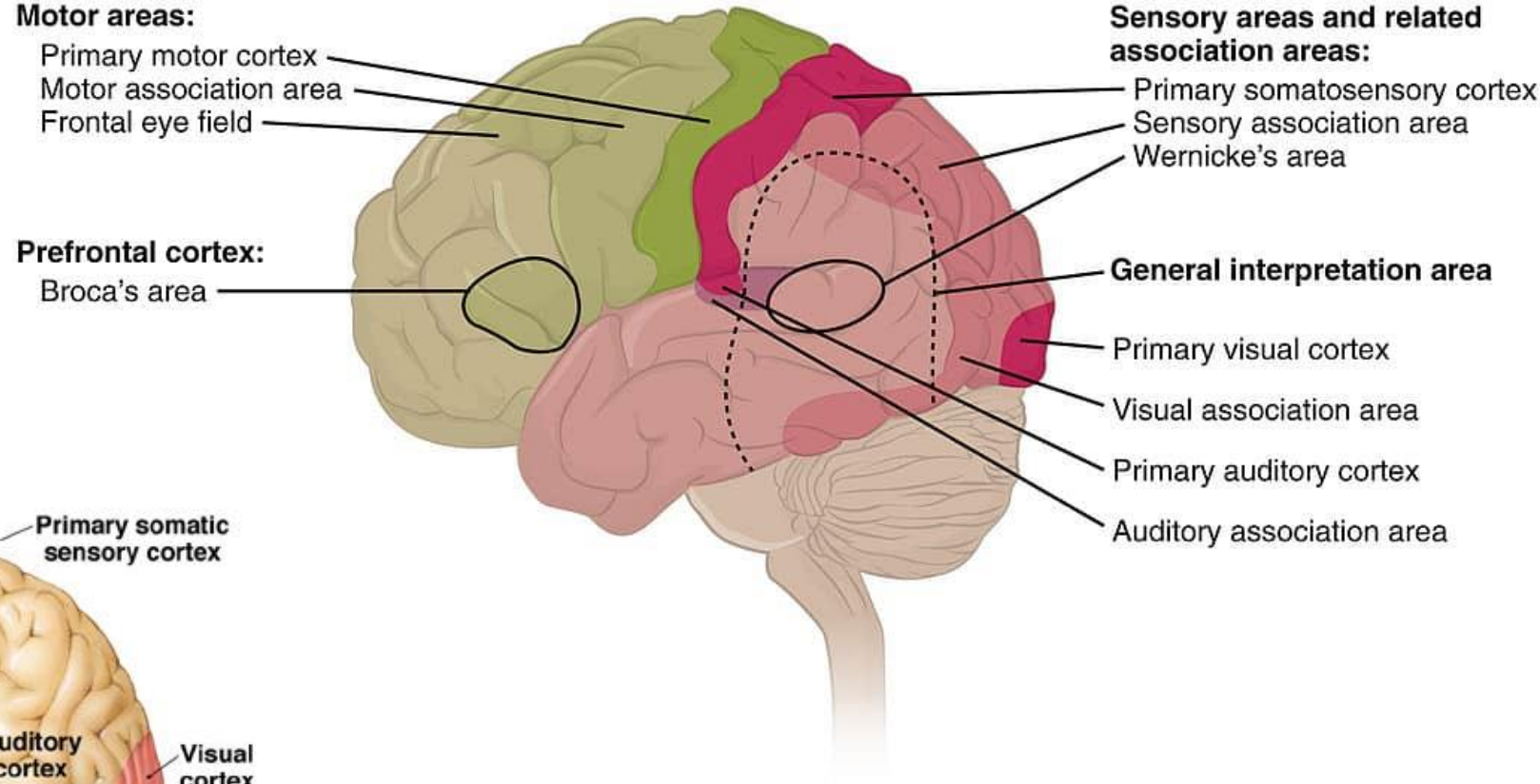
The callosum tissue seen in a healthy brain (bright white in top image) retracts after a corpus callosotomy, leaving just the ventricle (black).

Tactile and visual information reaches the contralateral hemispheres through nerves that cross over below the corpus collosum.

However, visual information in the right hemisphere travels to the language center in the left hemisphere via the corpus collosum







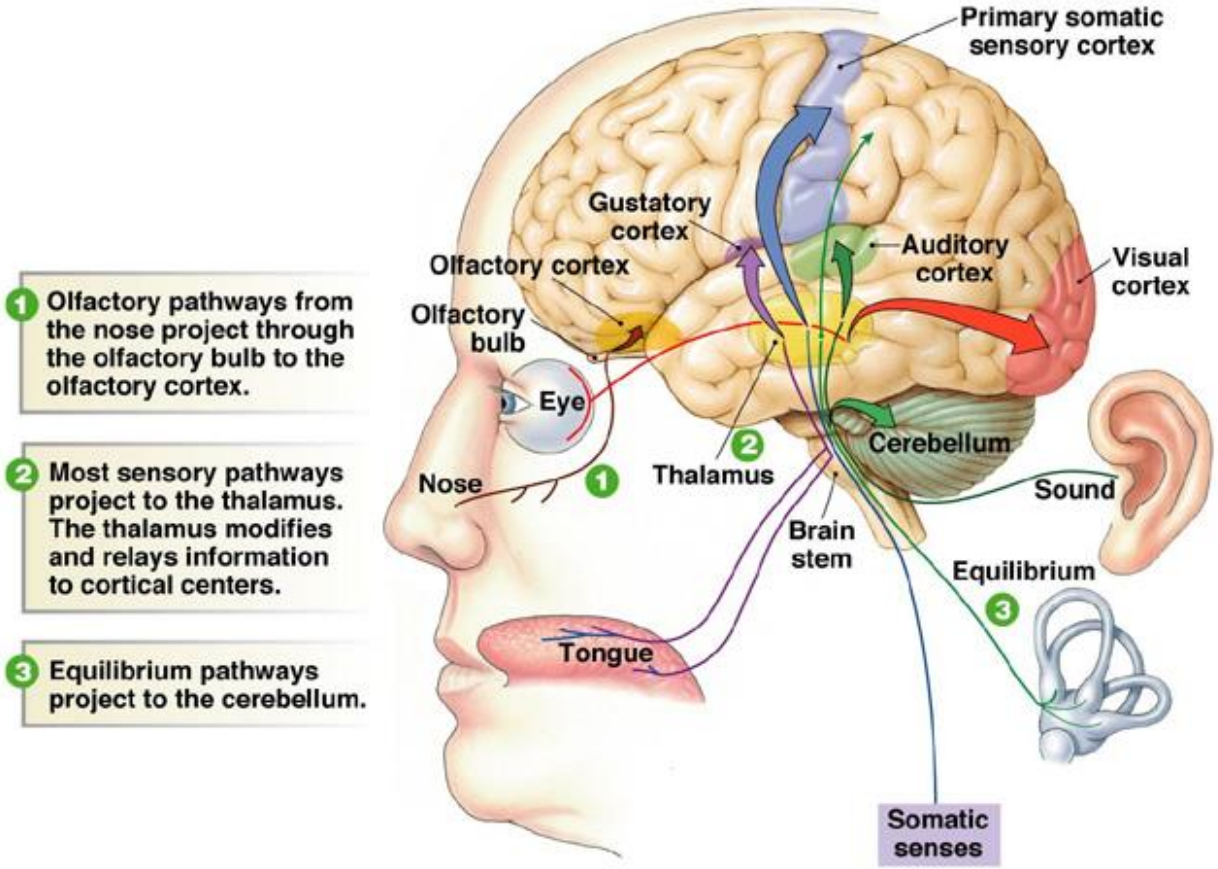
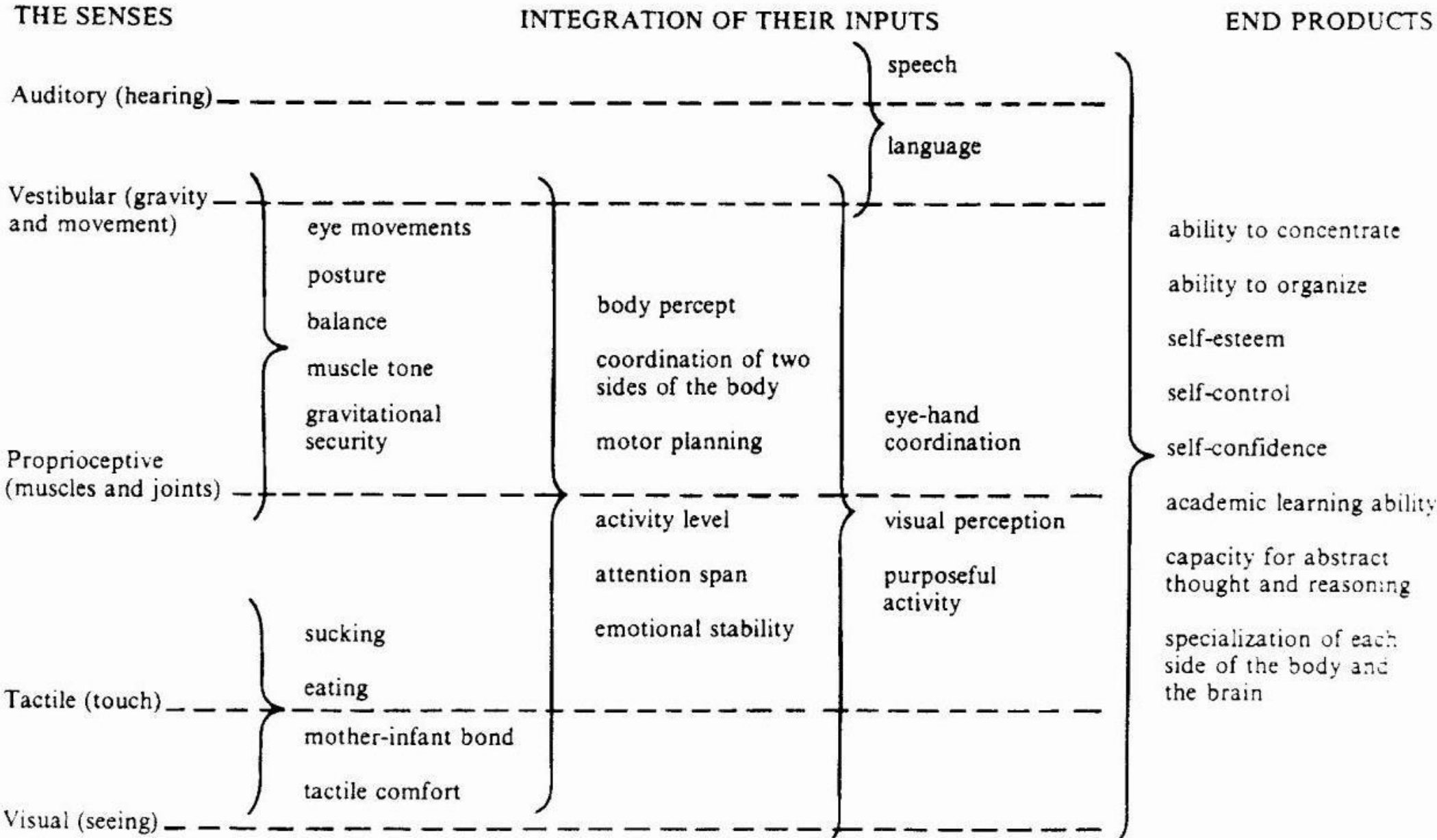
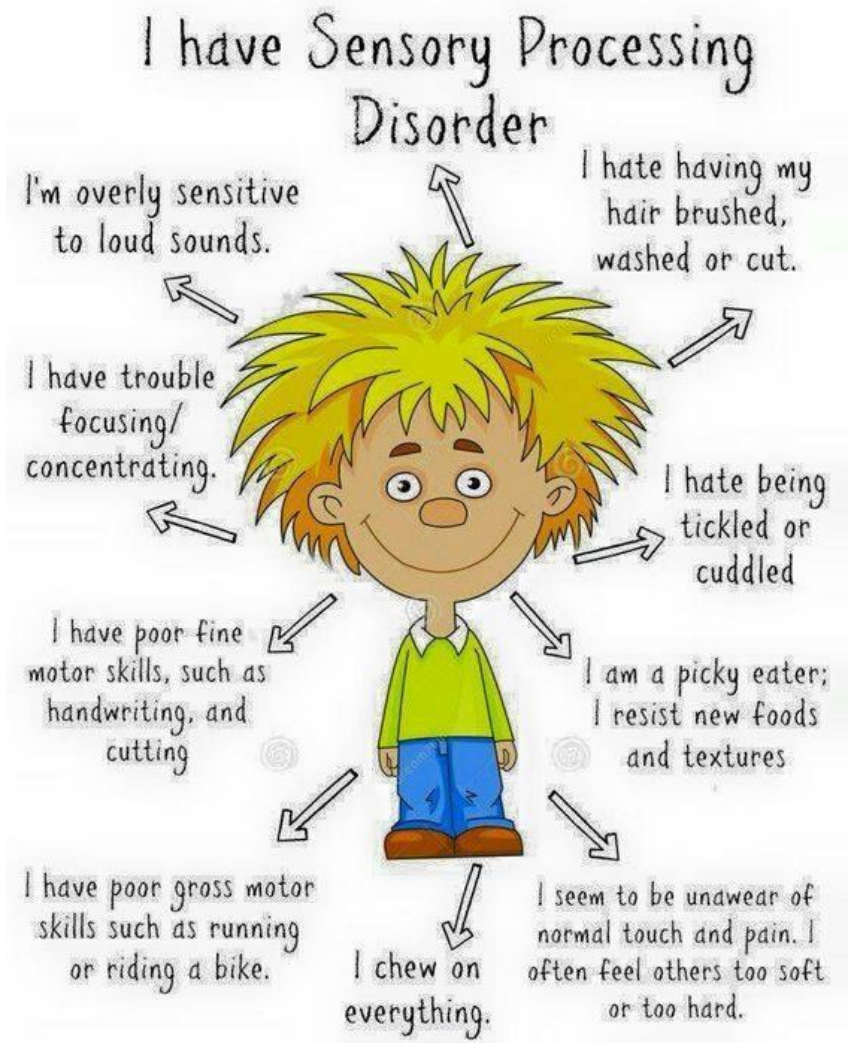


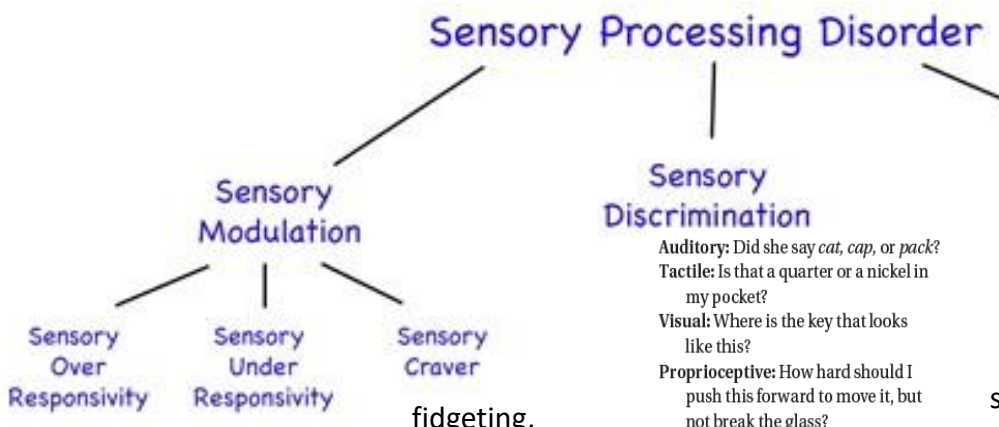
Fig. 10-4



https://www.griffinot.com/sensory-integration-sensory-processing/


I have Sensory Processing Disorder
I'm overly sensitive to loud sounds.
I hate having my hair brushed, washed or cut.
I have trouble focusing/ concentrating.
I hate being tickled or cuddled
I have poor fine motor skills, such as handwriting, and cutting
I am a picky eater; I resist new foods and textures
I have poor gross motor skills such as running or riding a bike.
I chew on everything.
I seem to be unawear of normal touch and pain. I often feel others too soft or too hard.

Treatment based on subtype of the



Hypersensitive
dislike for textures (fabrics, foods, grooming products), discomfort towards normal sounds, lights, temperature, movements, smells, tastes

Hyposensitive
tardiness, sluggishness, slow reaction

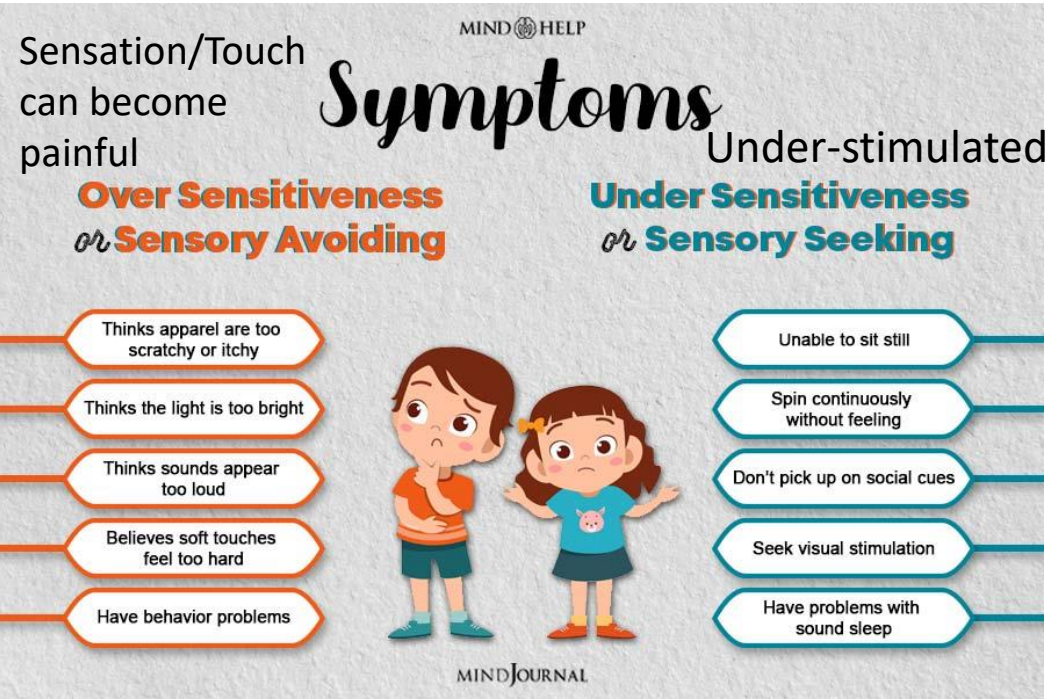
fidgeting, impulsiveness, seeking or making loud, disturbing noises

slow and uncoordinated movements or poor handwriting, dropping objects, stumbling,

- no internal motivation to move (e.g., run, jump, skip, hop)
- difficulty using both sides of the body simultaneously (e.g., catching a ball, rotating trunk to cross the midline of body needed to ride a bike or hit a baseball)
- challenges with following a moving object with eyes, noticing what happens around him using peripheral vision)
- inefficient vestibular (awareness of where he is in space)
- Inefficient proprioception (awareness of position of muscles and joints)

Dyspraxia is the inability or difficulty with three aspects of completing a motor action:
1) ideation,
2) sequencing, and/or
3) motor execution

Commonly observed in Autism Spectrum Disorder (ADD, ADHD, Asperger's syndrome) and other developmental disabilities



https://www.autism.org/sensory-integration/

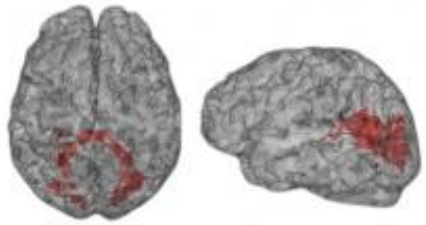
Mapping Sensory Processing Disorders in the Brain

Children with sensory processing disorders have decreased structural brain connectivity in specific sensory regions different than those in autism. Here's a closer look at the areas affected.

Sensory Processing Disorder only

Area Affected:

Splenium of the corpus callosum between left and right lateral occipital cortices



Autism Spectrum Disorder only

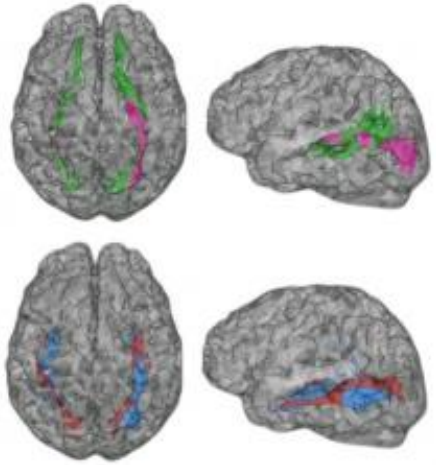
Areas Affected:

Inferior fronto-occipital fasciculus (IFOF)

Fusiform-amygdala

Inferior longitudinal fasiculus (ILF)

Fusiform-hippocampus

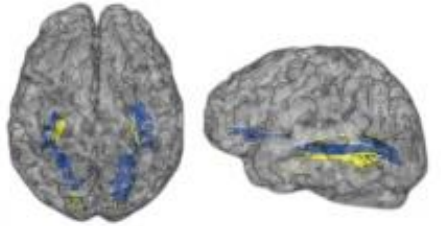


Autism Spectrum and Sensory Processing disorders

Areas Affected:

Posterior corona radiata (PCR)

Dorsal visual stream



https://www.ucsf.edu/news/2014/07/116196/kids-autism-sensory-processing-disorders-show-brain-wiring-differences

Example of activities a child can engage in to improve sensory discrimination

Children who have poor **sensory discrimination** need sensory-rich activities in the domain in which they have issues.

Visual: category games (e.g., find everything in the room that's a circle, while driving point to all food-related signs)

Interoceptive: Talk about how your body feels at times when you are happy versus worried. Say things such as, "I'm so happy, I can feel my heart beating fast!" or "I always feel nervous when I [fill in the blank]. My stomach feels like it is flipping over." That way the child will begin to understand the body sensations related to emotional content.

Auditory: Play the same-and-different game, "I'm going to say two words, and you tell me if they are the same or different. Then it will be your turn to try to trick me."

Proprioceptive: Play Simon Says or Mother May I? performing unusual, novel body movements.

Treatment for sensory processing problems is called sensory integration. The goal of sensory integration is to challenge a child in a fun, playful way so they can learn to respond appropriately and function more normally

Therapy video

Dance therapy

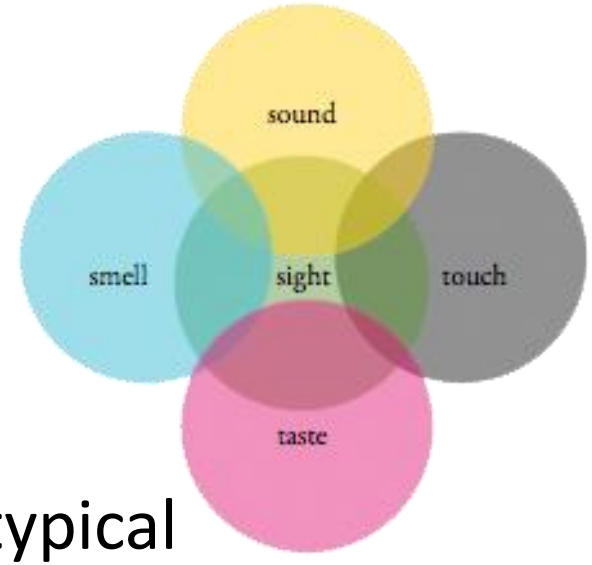
Repetitive movements
Costumes
Lights

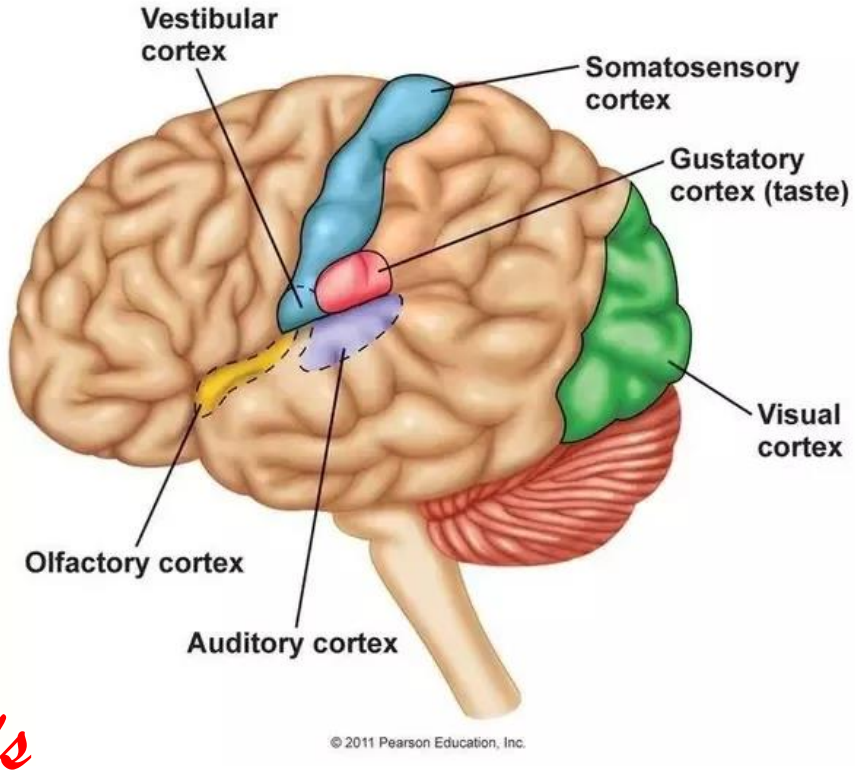
Art therapy

Concentration/engagement
Hand eye coordination
Sensitive touch

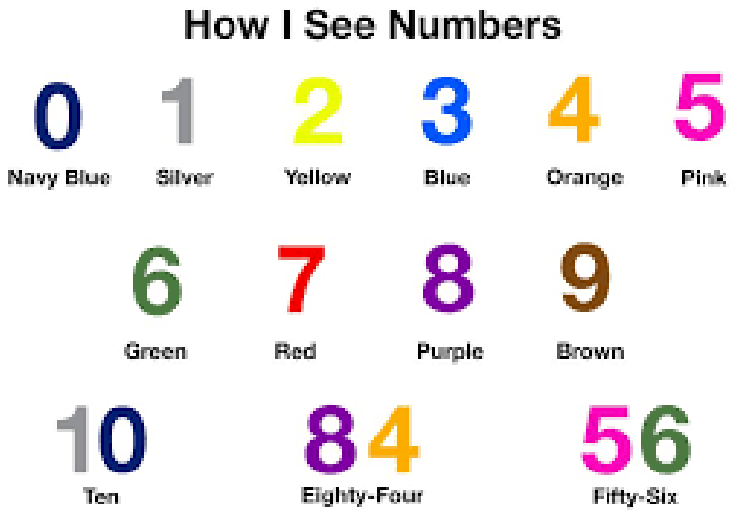
- Synesthesia?

Synesthesia





- Routing of senses is not typical

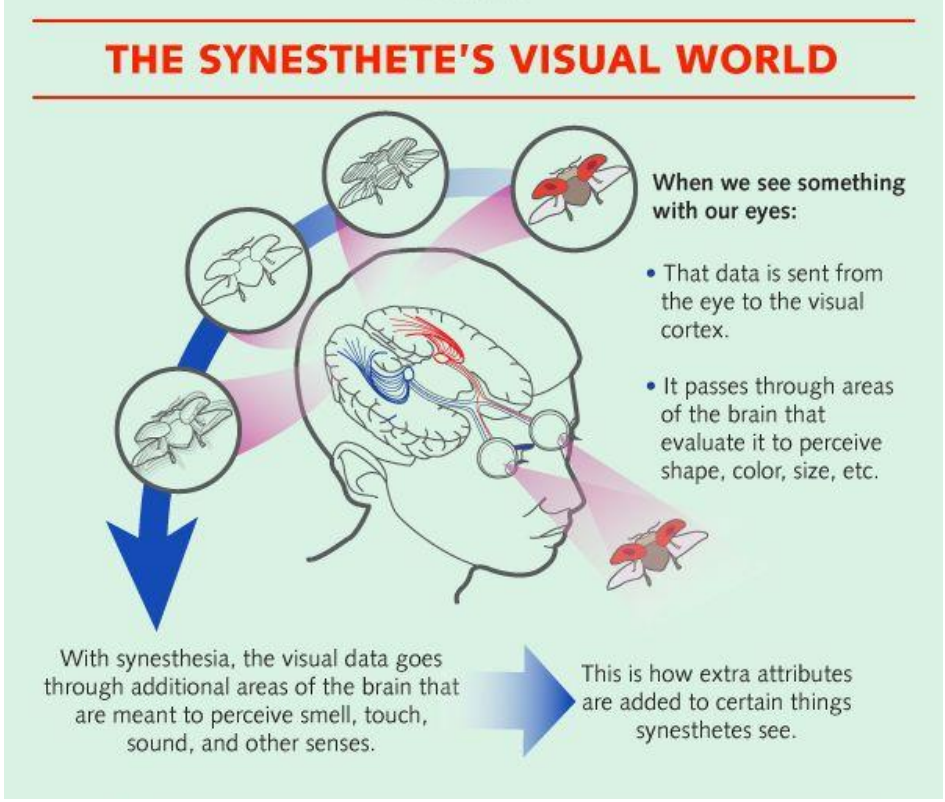


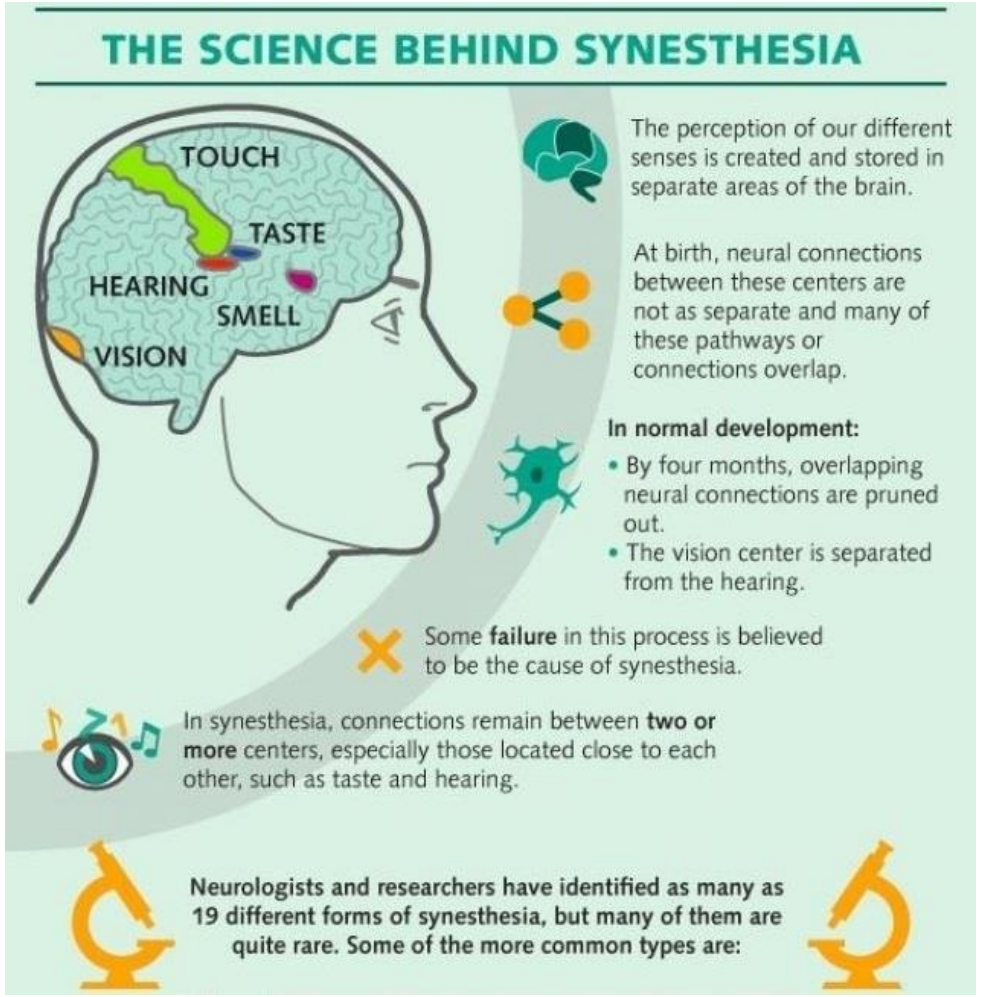
Tasty Words

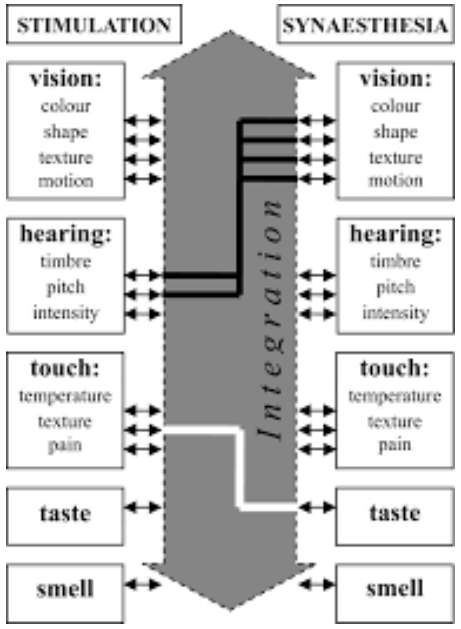
Colorful Sounds

Synesthesia

Synaesthesia is thought to have a genetic component, since it often runs in families and is more common in women







Emotions - > colors	Musical sounds - > colors	Sounds - > smells
Emotion - > smell	Odors - > colors	Sound - > temperatures
Emotion - > flavor	Orgasm - > colors	Sound - > touch
Emotion - > pain	Pain - > colors	Temperatures - > colors
Emotion - > smell	Pain - > flavor	Temperature - > flavors
Emotion - > temperature	Pain - > smell	Temperatures - > sounds
Emotion - > touch	Pain - > sound	Time units - > colors
Flavors - > colors	Personalities - > smells	Touch - > colors
Flavors - > sounds	Personalities - > touch	Touch - > emotions
Flavors - > temperatures	Personalities - > colors (auras)	Touch - > flavors
Flavors - > touch	Phoneme - > touch	Touch - > smell
General sounds - > colors	Phoneme - > flavor	Touch - > sounds
Graphemes - > colors	Phonemes - > colors	Touch - > temperatures
Grapheme - > flavor	Smells - > flavor	Vision - > flavors
Kinetics - > colors	Smells - > sounds	Vision - > kinetics
Kinetics - > sounds	Smells - > temperatures	Vision - > smells
Lexeme - > touch	Smells - > touch	Vision - > sounds
Musical notes - > colors	Sound - > flavors	Vision - > Temperatures
Musical notes - > flavors	Sounds - > kinetics	Vision - > touch

Hallucinations are a perception not based on sensory input, whereas illusions are a misinterpretation of a correct sensory input.

Synesthesia is not an illusion – it is an added perception to a stimulus (sound + color)