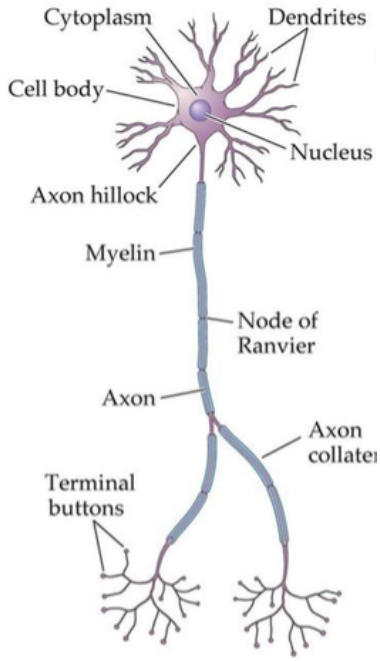
Neuropharmacology

The neuron:

- First thought fluids in the brain transcend muscle movements, then Galvani discovered electrical stimulation of muscles, Golgi then stained the neural membrane (led to neuron theory that brain made up of neuron cells that communicate with each other)
- Information > within neurons (electrical activity) via action potentials
- Information > between neurons (chemical signals) via neurotransmitters diffuse across synapses
- Dendrites: receive info from other neurons via cell terminals
- Cell body: machinery to keep cell alive and functioning
- Axon hillock: input from other neurons are used to determine whether the cell fires or not
- Myelin sheath: insulation layer increased speed of conduction down axon
- Node of Ranvier: allow electrical charge to jump to each node
- Terminal buttons: pass electrical signals across synapse to dendrite of target cell



Action potential:

- Huxley used giant squid axon to discover how cells pass electrical signal down their length (action potential)
- Occurs when a neurotransmitter causes Na channels to open. Na+ into cells create brief positive charge inside cell Na is then pumped out due to K-, cell becomes –ve again
- The brief +ve charge = ion channels open = electrical signal passed down the neuron
- Myelin sheath as electrical insulator, blocking ions across cell membrane (allows for faster speed)

Neurotransmitters:

- Action potential > axon terminal stimulates release of neurotransmitters > cross synaptic gap to bind to receptor on receiving neuron (allowing charged atoms to enter to excite/inhibit new action potential)
- Sending neuron absorbs excess called reuptake
- Excitatory: open Na+ channels to create +ve charge increasing action potential
- Inhibitory: open K+ channels releasing ions trapped = -ve cell = decrease action potential

Agonist vs. antagonist drugs:

- Agonists: fit the receptor perfectly to open/close linked channel
- Antagonists: don't fit the receptor, thus block action of neurotransmitter

Dopamine – reward:

- Three pathways: nigrostriatal, mesolimbicand mesocortical
- Mesolimbic: Ventral tegmental area (VTA) to nucleus accumbens (substrate in drug reward)
- Olds and Milner's used technique called intracranial self-stimulation where stimulating electrode inserted into brain. Rats favored the location of the box where stimulation was activated (then rats learned to press lever for stimulation as rewarding, rate increased > underpin addiction)
- Cocaine (blocks dopamine transporters, dopamine not cleared from cleft) and amphetamines (reverse the transporter) increase dopamine across synaptic cleft
- Wise measured rats rate of lever pressing for stimulation of different frequencies. Higher stimulation was more rewarding therefore higher lever pushing (increase dopamine=less push needed, small amount = more pushes)
- Rats push level repeatedly means it's rewarding, drug must activate dopamine in order to sustain self-administration behavior