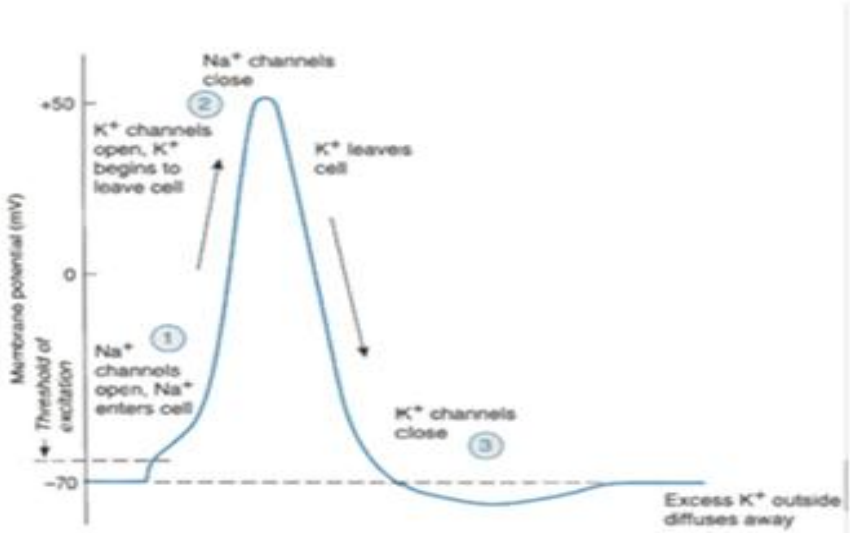
larger than normal electrical current is required to produce an action potential. Absolute refractory period = the state of the axon during repolarisation when a new action potential can not be generated. The refractory period ensures the action potential travels in one direction only.

- Myelin sheath - Insulates the axons, only allows action potentials in unmyelinated areas known as the nodes of Ranvier. Results in saltatory conduction - action potential jumping from node to node, increases speed action potential can travel along axon.



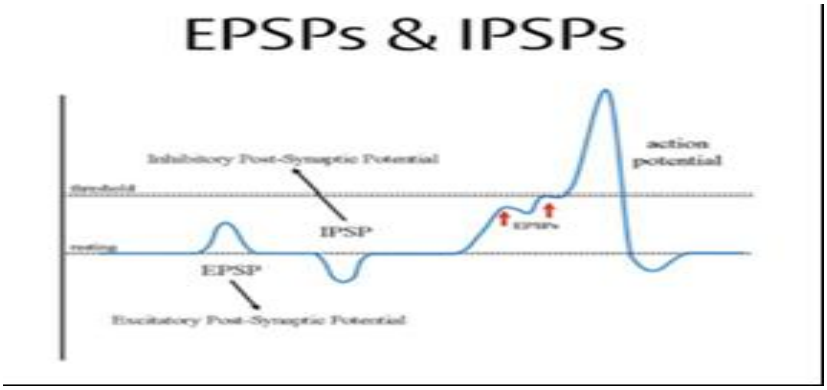
Week 3 - Chemical Brain

- Chemical signalling - between neurons. The action potential sent down the an axon reaches the axon terminal. It must then cross over to the next neuron (or e.g., onto a muscle if in PNS)
- Synaptic junctions - Axon terminals from one neuron are separated from the dendrite of an adjacent neuron. The small (~ nanometres) gap = synaptic cleft. The synaptic cleft is the space between the presynaptic membrane (axon) and postsynaptic membrane (dendrite)
- Chemical synapse - The synapse is like a break in the electrical circuit and is therefore a barrier that the electrical signal can not cross. The electrical signal is converted to a chemical signal to link the action potential of one neuron with a synaptic potential in another neuron.
- Electrical synapse - neurons connect with each other, meet at gap junctions which are ion channels connected across 2 neurons. Electrical signal can pass directly between neurons and eliminates the ~5ms it takes for chemical signalling.
- Neurotransmitters - Neurotransmitters are chemicals released from the presynaptic neuron onto a target postsynaptic neuron. They can have an excitatory or inhibitory effect. The process by which neurotransmitters pass on the information signal is called neurotransmission.
- Neurotransmission - 4 steps in transmitting information across a chemical synapse:
  - Neurotransmitter synthesis and storage - Some neurotransmitters are synthesised in the soma and are transported to the axon terminal along microtubules. Other neurotransmitters are synthesised in the axon terminal. In the axon terminal, all neurotransmitters are stored in vesicles. Vesicles are phospholipid bilayer compartments.
  - Neurotransmitter release - When the action potential reaches the axon terminal, voltage changes on the presynaptic membrane set off a series of processes to release neurotransmitter, held in vesicles, into the synaptic cleft: The presynaptic membrane has many voltage-sensitive calcium (Ca2+) channels that open in response to the membrane voltage change (from the incoming action potential) The opening of Ca2+ channels causes and

influx of Ca2+ into the axon terminal. Ca2+ binds to a protein calmodulin which causes vesicles to move to the presynaptic membrane. The vesicles fuse with the presynaptic membrane to release their contents into the synaptic cleft = exocytosis.

- Receptor binding - The released neurotransmitter diffuses across the synaptic cleft. It binds with specialised protein receptors that are embedded in the postsynaptic membrane. The receptors only bind specific neurotransmitters. There are 3 effects receptor binding can have on the postsynaptic membrane. Depolarisation of the postsynaptic membrane to excite the postsynaptic neuron. Hyperpolarisation of the postsynaptic membrane to inhibit the postsynaptic neuron. Initiate modulatory chemical reactions, that will indirectly produce excitation or inhibition of the postsynaptic neuron.
- Neurotransmitter deactivation - Neurotransmitter in the synaptic cleft must be removed to stop the occupation and stimulation of receptors. Can occur in 5 ways - Diffusion: some of the neurotransmitter diffuses away from the synaptic cleft. Degradation: enzymes in the synaptic cleft break down neurotransmitters. The components are then returned to the axon terminal to be recycled back into neurotransmitter. Re-uptake: transporters on the presynaptic membrane bind neurotransmitters and return them to the presynaptic terminal for re-use. Autoreceptor binding: receptors on the presynaptic membrane bind neurotransmitters that inhibit their further release. Glial uptake: neurotransmitters can be taken up by nearby glial cells which can then be returned to the axon terminal.

- Postsynaptic effect of neurotransmission - Action potentials not produced in postsynaptic terminal straight away. The result can be graded potentials that increase or decease the likelihood of an action potential being produced. Neurotransmitter receptors can open ion channels to produce an excitatory postsynaptic potential (EPSP) via Na+ influx (depolarisation). An inhibitory postsynaptic potential (IPSP) is produced via Cl- influx or K+ efflux (hyperpolarisation).
- EPSPs and IPSPs - EPSPs and IPSPs travel along the dendrites to the membrane of the cell body, but action potentials are not produced here (because the cell body does not have voltage-gated Na+ and K+ channels). These graded potentials must reach the axon hillock (the start of the axon) in order for an EPSP to produce an action potential. An action potential is produced when enough EPSPs sum to reach the activation threshold.
- Summation - Temporal summation: Graded potentials that occur at approximately the same time on the membrane are summed and produce a large single EPSP (or IPSP). Spatial summation: Graded potentials that occur at approximately the same location on a membrane are summed.



Week 4 - Neurotransmission

- Synapses in learning and memory - Synapse structure can change, synaptic plasticity is the neural basis of learning. Learning is a relatively permanent change in behaviour that results