Synapses

In the previous section we discussed action potentials, the voltage pulses that travel along axons and carry signals from neuron to neuron. What we didn't discuss was how the signals got from the axon of one neuron to the dendrite of the next. The answer is synapses.

In discussing the action potential we mentioned that it is a largely stereotypical signal, the action potentials from a neuron all have roughly the same amplitude and shape. The synapses, in contrast, are diverse. They have different strengths, which can change over time. Beyond these variations in strength, synapses can have different dynamics, with differences in the time course of how the synapse responds to a spike, or in how the synapse responds to spikes coming in quick succession.

The complicated and variable behaviours of synapses are possible because synapses do not simply connect the axon to the dendrite, they are not simply holes or pores through which ions flow. In fact, there are synapses, called **gap junctions** that are like that, these gap junctions are the only synapses in some simple creatures like jellyfish and are found in the mammalian brain. However, the synapses we usually have in mind are **chemical synapses** in which the signal transfer from **pre-synaptic** axon to **post-synaptic** dendrite involves a complex chemical cascade.

Chemical synapses

In Fig. 1 is a cartoon of a synapse. Note that the dendrite and axon don't actually touch; there is a little gap, called the **synaptic cleft** between the two. Of course, the axon and dendrite are held together by glial cells, cells which also surround the cleft and keep everything in place.

Importantly, electrical charge does not flow directly from dendrite to axon. When a spike arrives at the synapse it triggers a chain of events. The sudden change in voltage opens channels in the membrane of the synapse that allow calcium to flow into the **terminal bouton**, the part of the synapse on the axon side of the cleft (it is an amazing property of neurons that they contain ion-specific channels). Calcium flows into the synapses and, by way of complicated chemical reactions, this causes some little ($\approx$40 nm) membrane-bound spheres called **vesicles** to fuse with the wall of the cleft and burst, re-

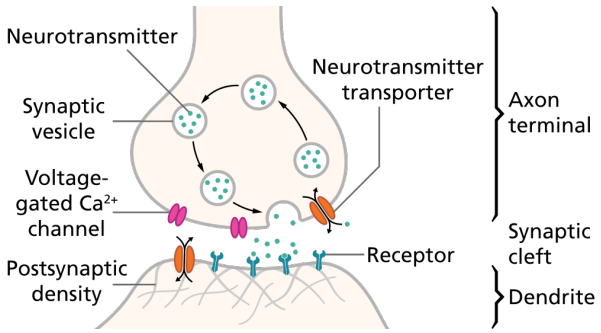

Figure 1: **A diagram of a synapse**; this cartoon shows the main parts of the synapse; the axon of the pre-synaptic neuron is coming in from the top, the dendrite of the post-synaptic neuron is leaving through the bottom. Figure from `wikipedia`

leasing specialised molecules called **neurotransmitters** into the cleft.

These neurotransmitters, in turn, bind with channels ("receptors") on the opposite face of the cleft, that is, with channels in the membrane of the post-synaptic dendrite. These channels then open in response to binding with the neurotransmitter. Depending on the type of synapse, they either allow ions to flow into, or out of, the dendrite, either increasing its voltage, or decreasing it. As a crude first approximation, we might summarise the immediate effect of a synapse as either **excitatory**: increasing the voltage of the post-synaptic neurons, or **inhibitory**: decreasing it.

Each type of synapse has different channels in the dendritic face of the cleft and different neurotransmitters that bind to these channels. In an excitatory synapses these channels allow sodium ions into the cell; Since sodium ions are positive ions this increases the charge inside the cell. Some excitatory channels may also allow influx of positively charged calcium ions. In an inhibitory synapse the channels either allow chlorine ions into the dendrite (chlorine ions are negative so this decreases the voltage), or they allow potassium ions out of the cell (potassium ions are positive, so this decreases the voltage).

Ion channels that open because they have bonded with a neurotransmitter are called **ligand-gated channels**; a ligand is a molecule that binds to things. These channels act as gates, sometimes allowing ions through and

sometimes not and they do so depending on whether or not they are bound to a ligand. This is in contrast with the **voltage-gated channels** that open and close depending on voltage. We will see later that voltage gated channels are important in understanding how spiking happens.

The binding between ligand and ligand-gated channel is quite loose and the molecules are bathed in a warm fluid; random Brownian movements will unbind the ligand allowing the channel to close. The timescale for this unbinding is different for different channels; for typical excitatory synapses it is of the order of tens of milliseconds. The neurotransmitter in the cleft is also cleared away by little pumps; "reuptake pumps", and is repackaged into vesicles ready for the arrival of later spikes.

The final part of this story is the dendrite itself. Consider first the situation with an excitatory neuron: Current flow into a neuron increases its voltage; This ramps up as more and more ligand-gated channels open before decaying away again as they close. The resulting positive pulse is called an **excitatory post-synaptic potential** or EPSP. For an inhibitory synapse the pulse is negative and is called an **inhibitory post-synaptic potential** or IPSP. Example PSPs are showing Fig. 2.

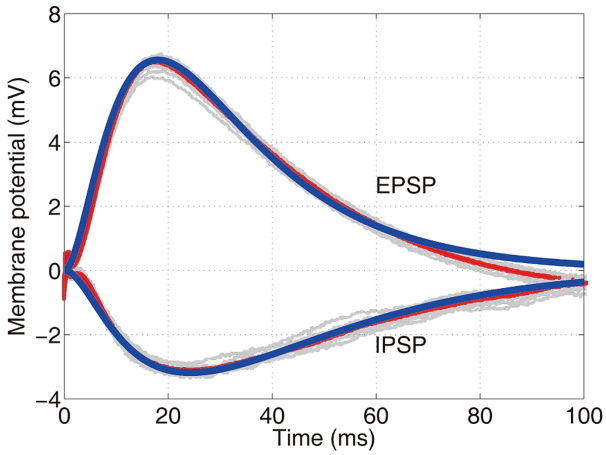

Figure 2: **Some PSPs**; the grey lines are recordings for individual PSPs with the red lines giving averages and blue lines demonstrate a model; we will look at simple models of PSPs later. Figure from [**?**]

In general dendrites lack the voltage-gated channels needed for action po-

tentials and the PSPs in dendrites propagate passively towards the axon. Dendrites are typically shorter than axons and thicker, so this conductance allows the change in voltage at the dendrite to propagate in to the soma. Even in the soma, where there are voltage gates channels an individual EPSP won't change the voltage enough to cause a spike. However, if lots of PSPs arrive at around the same time, the voltage in the soma will increase until it reaches a tipping point, around -55 mV is typical value of where this tipping point is. At the tipping point the opening of voltage-gated channels will cause a spike, usually at the point the axon joins the soma, and this spike will head off down the axon.

1 Summary

Here we described synapses; this is a complicated story we will go through again. When the spike arrives at the synapse some of the vesicles, little bags of neurotransmitter, fuse to the membrane of the synapse closest to the dendrite and burst, emptying neurotransmitter into the synaptic cleft. This, in turn, causes channels to open in the dendrite, changing the potential there. This change in potential, a PSP, flows into the soma causing a small change in the potential in the soma. If these small changes add up to push the soma potential to a threshold, the neuron spikes, sending out an action potential along the axon.