1 Notes on "Ionic Channels of Excitable Membranes" by Bertil Hille

1.1 Chapter 4: Calcium Channels

1.1.1 Crustacean muscles can make Ca^{2+} action potentials

Fatt and Katz (1958) discovered that crustacean muscles don't need Na channels in order to create action potentials.

Fatt and Ginsborg (1958) identified that Ca^{2+} ions were facilitating the spiking. Ca^{2+} has a greater Nernst equilibrium potential than Na. The use of TBA and TEA in their experiments blocked the K channels. This stopped the repolarisation of the cells, which caused the Ca^{2+} channels to remain open and depolarise the cells regenratively.

Hagiwara and Naka (1964) found that reducing the amount of intracellular Ca^{2+} restored Ca spiking mechanism.

Every type of excitable cell has Ca channels. They are similar to Na and K channels in that they are voltage dependent, they open in response to depolarisation with some delay, and they close rapidly after repolarisation.

1.1.2 Ca Channels activate with depolarisation

In general, Ca channels activate when the membrane is depolarised. They need a larger depolarisation than Na channels need. They activate more slowly than Na channels also. I_{Ca} is usually quite a bit smaller than a typical I_{Na}. If depolarisation is maintained, the I_{Ca} deactivates slowly, and usually doesn't completely stop. I_{Ca} is usually small, and slowly decaying. This causes pure Ca action potentials to rise slowly, conduct slowly, and have a long duration.

Even within the same species, different cells use Ca^{2+} in different ways. For example, in crayfish muscles or mammalian hearts, the I_{Ca} makes a major contribution to functionality. But in frog muscles, the I_{Ca} is negligable relative to the I_{Na}, which does all the work.