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Dependence of Action Potential Propagation Delay and Synaptic Delay on Extracellular Calcium Ion Concentration

Time for a neural signal to travel through a neuron can be described in two parts, action potential propagation delay and synaptic delay. Action potential propagation delay is defined as the time period between action potential induction and action potential arrival at the nerve terminal. Synaptic delay is the time period required for transmission of the signal from the presynaptic cell to the postsynaptic cell. Synaptic delay consists of three main segments, release of neurotransmitters, diffusion of neurotransmitters through synaptic cleft, and induction of a postsynaptic signal. Among the three main segments, the first segment – neurotransmitter release – makes up most of the synaptic delay. Then the factors that facilitate neurotransmitter release, such as calcium ions, should significantly change, ideally reduce, synaptic delay; but earlier reports state that there is no significant correlation between synaptic delay and calcium ion concentration. The seeming contradiction led to this project, which aims to study the dependence of synaptic delay on the extracellular calcium ion concentration. Furthermore, studying synaptic delay under different calcium ion concentration allows for acquisition of the minimum calcium-independent synaptic delay as the calcium ion concentration increases to a point from which point on the synaptic delay cannot decrease further. The dependence of action potential delay on the extracellular calcium ion concentration is also examined to study how calcium ions affect both parts of neural transmission. Single terminal extracellular recording was used to measure action potential delay and synaptic delay separately under different extracellular calcium ion concentrations using neuromuscular junctions of freshwater crayfish (*Procambarus Clarkii).* The results were then plotted and analyzed, which shows that the increase in extracellular calcium ion concentration decreases synaptic delay and increases action potential delay. Calcium ions speed up synaptic signal transmission by helping to fuse the neurotransmitter vesicle membrane with the nerve terminal plasma membrane. Calcium ions slow down action potential propagation by opening the calcium-ion dependent potassium channels and hyperpolarizing the cell. In conclusion, calcium ions are shown to speed up synaptic transmission and obstruct action potential propagation.