Optogenetics is an increasingly popular neuronal stimulation technique used for studying neural circuits and controlling brain activity. However, when applied without sufficient knowledge, it can cause unintentional silencing of the targeted neurons by inducing a state termed depolarization block (DpB), in which neurons cease to fire action potentials. The susceptibility to silencing is not consistent among neurons, and the relationship between their biophysical properties and their vulnerability to DpB remains poorly understood. In this thesis, we investigate how the densities of voltage-gated sodium (Na_v) and potassium (K_v) channels, which are known to govern DpB dynamics, influence the neuron's ability to resist this phenomenon. We also examine the impact of neuronal size on DpB susceptibility. Using a computational model of a layer V pyramidal neuron, which we simplify to a single compartment to represent the behavior of a generic excitatory neuron, we introduce an automatic classifier consistently identifying DpB through voltage trace analysis. This allows us to systematically assess the influence of varying Na_v or K_v channel densities in the neuron's membrane. We discover that increasing these densities enhances the neuron's resistance to DpB. Contrary to previous studies, neuronal size was found not to affect susceptibility to light-induced DpB. Furthermore, our analysis shows that while increasing Na_v channel density raises the mean of depolarized membrane voltage at which DpB settles, K_v channel density affects this property only if the membrane contains intermediate densities of Na_v channels.