Spontaneous electrical activity is seen as a vital component to the developing nervous system in a wide variety of species. In chickens, before phototransduction is even possible, embryonic chick retinas fire highly coordinated bursts of spontaneous action potentials called retinal waves. These waves are first seen during the early developmental stages of the retina when local circuits are being formed and distal connections are being made from the retina to subcortical structures. Neuronal nicotinic acetylcholine receptors (nAChRs) are believed to be vital for triggering and propagating retinal waves and interestingly, certain nAChR subtypes have been observed to be more critical than others to retinal wave propagation. Previous studies have shown that by blocking retinal waves during development, dendritic growth and synaptogenesis within the retina are commonly disrupted. In this study, we are curious to see if administering nicotine (the agonist for nAChRs) to chick embryos during the early phases of spontaneous activity alters the expression of α3, α7, β3 and β4 nAChRs subtypes and their respective endogenous prototoxins (lynx1, lynx2, PSCA) in the developing retina. In addition, we want see if nicotine also disrupts the normal arborization of RGC dendrites during the early periods of initial synaptogenesis in the retina.