My name is Emily Moon and I'm a student here at Wesleyan University. I work in the Aaron Lab and the O'Neil Lab here at Wesleyan. This past summer I made a poster titled “Altered Electrophysiology and Calcium Dynamics in SOD1-Associated Amyotrophic Lateral Sclerosis.” My project over the summer was a literature review and I created four different sections looking at various different ways in which neurons degenerate in amyotrophic lateral sclerosis, or ALS. I have an introduction section, a background, an electrophysiology section, and then I review the major theories of neurodegeneration. Under the main theories of neurodegeneration, I have “Glutamate-Mediated Excitotoxicity and Calcium-Related Apoptosis,” “Mitochondrial Dysfunction,” and “Protein Aggregation.” Under “Electrophysiology,” I have “Hyperexcitability” and “Hypoexcitability.” In my presentation, I review background from the summer, two summers ago, when I was able to conduct research in person. From my results in this Figure 1, I found that both resting membrane potential as well as threshold for neurons treated with SOD1 aggregates are significantly higher than healthy, untreated neurons. They also have a significantly decreased ability to fire repetitive action potentials. Moving on to the “Theories of Neurodegeneration” section, I created three figures. The first figure, Figure 2, is cited from Chang and Martin et al. They show increased high voltage calcium currents in mice that are ALS models and have a G93A-SOD1 mutation. The second figure relates to mitochondrial dysfunction and is a schematic showing the various pathways in which mitochondrial dysfunction can lead to neuron death. And the final figure under “Theories of Neurodegeneration" is about protein aggregation from Benkler et al from in 2018. This shows that when motor neurons are treated with SOD1 aggregates, they have significantly lower survival rates than healthy motor neurons and non-motor neuron cells. Finally, in the “Electrophysiology” section, I have two figures. The first figure cited from Wanger et al 2014 shows hyperexcitability as an early phenotype in ALS patient-derived motor neurons. The second figure derived from Naujock et al illustrates hypoexcitability in, again, motor neurons derived from patients with ALS with both SOD1 and FUS mutations. At the bottom of my poster, I have an “Acknowledgements” section and a “References” section and again, I would like to just acknowledge the Wesleyan Research in the Sciences program as well as Josephine Park who helped me on this poster and helped me in my project.