2024 Buck Summer Scholar: Anshuman Das

My name is Anshuman Das, I am a rising senior at Vassar College in Poughkeepsie, New York. At Vassar College, I am majoring in Cognitive Science with a focus on Evolutionary Biology. I am involved in a research project under the guidance of Dr. Lori Newman, where I work to understand the role of astrocytes in synaptic plasticity for spatial working memory in rat models using DREADDs (Designer Receptors Exclusively Activated by Designer Drugs). At the Buck Institute, I am working in the Ellerby Lab under the supervision of Dr. Kenneth Wilson, a postdoctoral fellow, and Dr. Lisa Ellerby. My work in the lab focused on the role of the intercellular transport system and its connection to cellular senescence, which is when cells stop dividing and accumulate in the body. Senescent cells can cause a myriad of irregular functions and lead to the development of neurodegenerative diseases. Senescence has been connected to many neurodegenerative diseases such as Alzheimer's Disease (AD).

The Ellerby Lab is specifically focused on neurodegeneration, which is a type of disease that causes progressive damage to the neural cells. This damage, in turn, has trickle-down effects throughout the rest of the body, sometimes leading to changes in mood, behavior, and cognition. I am particularly interested in the disease models of Alzheimer's, Parkinson’s, and Huntington’s disease. All of these diseases can be attributed in part to the formation of toxic protein clumps. The retromer, one of the many cellular transportation systems, is responsible for the efficient movement of macromolecules around the cell, including proteins. It connects different cellular compartments, or organelles, together. In cases where this transport system breaks down, it can lead to the accumulation of these toxic clumps, which can be disastrous to cell viability. These dysfunctions in the retromer are specifically tied to the onset of neurodegenerative diseases. A component of interest within the transport system is OXR1, which is a structural protein responsible for holding the retromer together. OXR1 is lost in people with age, and the loss of OXR1 has been connected to neurological defects. More specifically, on a gene expression level, loss of OXR1 shows a similar pattern to Alzheimer’s disease. This loss can serve as an indication of DNA damage, which is a hallmark of aging. DNA damage can lead to cellular senescence.

In our project, I wanted to solidify the connection between the retromer and the onset of senescence. Therefore, in my research I am focused on highlighting senescence in the mouse model brains with OXR1 overexpression. I am using human neurons to understand the impact of knocking down OXR1 and also overexpressing it to see if the overexpression would rescue cellular senescence. These experiments would in turn help solidify the connection between senescence and retromer dysfunction.