2024 Buck Summer Scholar: Xinran (Jess) Liu

Hello everyone! My name is Xinran (Jess) Liu. I'm a rising junior majoring in Molecular, Cell, and Developmental Biology and minoring in Neuroscience at the University of California, Los Angeles (UCLA). At UCLA, I work in Dr. Daniel C. Lu's Neuroplasticity & Repair Lab under the guidance of Dr. Ruyi Huang. The lab aims to elucidate the mechanisms behind motor and respiratory functions encoded in the spinal cord using rodent models. We also investigate how neuromodulation on the spinal cord can restore lost functions and translate these findings to clinical trials for spinal cord injury patients. This summer at the Buck Institute, I joined Dr. Eric Verdin's Lab to study the interplay between aging, metabolism, and the immune system. Working with postdoctoral fellow Dr. Jingqi Fang, I am investigating the role of CD38, a major nicotinamide adenine dinucleotide (NAD)-consuming enzyme, in brain aging.

NAD is a crucial coenzyme involved in cellular metabolism. In aging, NAD levels decrease, which is shown to be linked causally to various age-associated diseases, including cancer, metabolic disorders, brain aging, neurodegeneration, and more. Recent research suggests CD38 increases with age and may contribute to the age-related decline in NAD levels. While studies have shown that CD38-removed mice are protected against obesity, metabolic syndromes, and mitochondrial dysfunction during aging, the role of CD38 in the brain has not yet been explored. Our lab has found that CD38 is predominantly expressed in the choroid plexus (ChP) of the brain. The ChP is a critical yet under-researched structure that produces cerebrospinal fluid (CSF) and forms the blood-cerebrospinal fluid barrier (BCSFB) through junctions between epithelial cells. The passive filtration and active transport of blood components via this barrier enable the production of CSF, which is essential for maintaining brain homeostasis, providing immune surveillance, and removing metabolic waste products. Researchers have reported that with aging, there are decreases in barrier integrity and changes in CSF composition, which are correlated with age-related cognitive decline and neurodegenerative disease symptoms. Given the crucial role that ChP plays in brain aging, it is important to investigate how CD38 is involved in this process.

My project aims to explore how CD38 influences ChP-mediated brain aging. Since CD38 levels increase with age and are predominantly expressed in the ChP, we hypothesize that the increased CD38 levels may be one of the causes of ChP functionality impairment with aging, potentially contributing to age-related brain symptoms. To investigate this, we will compare mice with CD38 removed to mice with normal CD38 expression using multi-dimensional techniques. Our goal is to determine if the loss of CD38 has a protective effect on ChP integrity and brain health. If a correlation is found, we will further investigate the underlying mechanisms. Insights into the role of CD38 in brain aging could be crucial for developing therapeutic strategies to protect overall brain health.