2023 Buck Summer Scholar: Lea Baskin Monk

My name is Lea Baskin Monk. I am a 4th-year biochemistry major at UCLA, where I work as an undergraduate researcher in the Thomas Rando Lab. In the Rando Lab, I study muscular aging. A significant manifestation of skeletal muscle aging is a decline in regeneration upon injury, which results from diminishing muscle stem cell activity and an altered muscle niche environment. I study the mechanisms behind these changes in the skeletal muscle with the goal of better understanding skeletal muscle aging and informing therapeutic interventions.

As a Buck Summer Scholar, I worked in Judith Campisi's lab. The Campisi Lab studies cellular senescence, a fascinating cell state that is likely evolutionarily selected for to prevent early-life cancer and promote tissue repair. However, as an individual ages, senescent cells accumulate and are associated with chronic inflammation and age-related diseases such as Alzheimer's, osteoarthritis, and (paradoxically) late-life cancer. In response to a stressor, such as DNA damage, senescent cells halt cell division by upregulating tumor suppression pathways and secreting inflammatory factors. These factors are collectively referred to as the senescence-associated secretory phenotype (SASP) and magnify the influence of senescent cells on their environment by interacting with nearby cells. While senescent cells have been identified in many tissues, they have different senescence profiles, driven by their tissue context and the type of senescence-inducing stressor. This heterogeneity prevents a single definition from describing all senescent cells, but opens a rich and yet-to-be-studied diversity of cellular senescence in disease.

This summer, I worked with PhD student Jun-Wei Brendan Hughes to study cellular senescence in idiopathic pulmonary fibrosis (IPF). IPF is a highly degenerative and age-associated disease in which scar tissue accumulates in the lung, causing progressive pulmonary failure. The average individual survives only three to five years post-diagnosis. While IPF is the most common type of pulmonary fibrosis, its underlying causes are unknown, and few treatments exist. Recently, the accumulation of senescent cells in the lung was implicated in the progression of IPF. This summer, I studied the relationship between cellular senescence in the lung and IPF using RNA sequencing techniques. We are particularly interested in the heterogeneity of senescence profiles in IPF and healthy lung cells. Elucidating the effects of senescence in IPF is essential to better understand IPF progression and develop more effective therapeutics.

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