Andersen Lab

Alzheimer's disease (AD) is characterized by progressive memory loss and cognitive decline and is the leading cause of dementia in those over the age of 65. Despite a growing number of afflicted individuals and rising economic costs leading to a sharp increase in research funding, the precise molecular basis for the disease remains elusive. Several growing lines of evidence suggest that defects in mitochondrial function constitutes a major factor underlying AD. In an effort to identify novel therapeutics capable of counteracting this loss in mitochondria function, the Andersen laboratory recently identified a novel drug-like molecule capable of enhancing autophagic mitochondrial turnover (mitophagy) which we have called 'mitophagy inducing compound' or MIC (Chamoli et al., Nature Aging, 2023). Excitingly, MIC engages a molecular target associated with this hallmark in a unique fashionwhich we have shown is also shared by another well-known mitophagy inducing agent, urolithin A (UA). Both drugs successfully mitigate neuropathology and cognitive losses associated with a pre-clinical model of AD, the triple transgenic 3xTg AD, underscoring their potential benefits in the context of human AD (Ballencestros et al., Geroscience 2023, unpublished data). Current work in the laboratory is towards interrogating newly identified direct drug binding targets in order to better understand mechanisms underlying the ability of these agents to regulate mitophagy function with a view towards future human clinical trials.