Beyond Amyloid: Dr. Grace Stutzmann's Fresh Approach to Studying Alzheimer's Disease

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The cause of Alzheimer's disease (AD) and the quest for its cure remain elusive, even in a modern and progressive time like the 2020s. Technology has advanced considerably since the early days of research on neurodegenerative disease, and it often seems as if the scientific community is publishing breakthroughs in AD regularly. For years, scientists have supported popular theories such as the amyloid-beta and tau hypotheses. However, neuroscientists wonder if they must look at different avenues to address this debilitating disease.

Cue Dr. Grace Stutzmann, Ph.D., a professor of neuroscience and director of the Center for Neurodegenerative Disease and Therapeutics at Rosalind Franklin University. A pioneer in studying neurodegenerative diseases, focusing on Alzheimer's disease, Dr. Stutzmann has long pondered alternative approaches to tackling the question, "Is there something else going on that potentially facilitates AD-linked cellular pathology?" Dr. Stutzmann began her work on Alzheimer's disease as a postdoctoral fellow at institutions like Yale University and UC Irvine. She continues her research with her lab at RFU, using various models such as human-induced neurons and transgenic mice. Notably, her main focus is on the role of calcium and its dysregulation before histopathology and memory loss. Calcium is essential for many biological processes like gene transcription, membrane excitability, and synapse plasticity. Elevated Increased calcium levels are closely associated with crucial AD characteristics, like beta-amyloid plaques, tau hyperphosphorylation, and synaptic dysfunction. Particularly concerning is that these histopathological characteristics of AD can further increase calcium levels, which can rapidly accelerate in a feed-forward system as the disease progresses. Therefore, the Stutzmann lab hypothesizes that stabilizing calcium signaling can disrupt disease progression earlier, presenting an innovative approach to understanding and potentially treating AD.

Dr. Stutzmann and her lab, in collaboration with the Ginsberg laboratory at New York University Grossman School of Medicine, are pursuing new research into the relationship between Alzheimer's disease and individuals with Down syndrome (DS), as this group has an increased risk of developing the disease, with nearly all adults showing common AD symptoms as they grow older. This results from the location of a specific protein called amyloid precursor protein on chromosome 21, which individuals with Down syndrome have an extra copy of. This protein is widely known to facilitate the formation of amyloid-beta plaques, a hallmark feature of AD. Amyloid aggregation is observed in nearly every individual with DS over 40 years old. Therefore, studying the similarities and differences between these two disorders can shed more light on the changes in the brain's chemistry and pathways.

Working as a member of the Stutzmann lab through the LFC-RFU Summer Scholars Program has enabled me to challenge traditional assumptions and evaluate new ideas presented in the media. Notably, when it related to the "promising" new treatments for AD, Dr. Stutzmann encouraged me to look at the raw data of such treatments and see to what extent they improve cognition and memory rather than merely reducing amyloid formation. Dr. Stutzmann and her team's emphasis on calcium dysregulation in AD pathogenesis, particularly in the context of Down syndrome- marks a fresh and potentially promising approach to understanding this common neurodegenerative disease.