Alzheimer's: Aging and Dysfunctional Glia? Giving a "DAM"!

Name: Victor Lau

SFU Faculty/Major: Faculty of Science, Behavioural Neuroscience

Presentation Description:

Alzheimer's Disease has harmed many that we know and love, and continues to burden society both in health care and personal loss. Future perspectives should not approach this dementia through traditional means, but rather understand and target Alzheimer's as a disease of aging and non-neuronal, glial brain cells.

Abstract:

Alzheimer's Disease (AD) leads to progressive memory loss, mood disturbances, and being unable to take care of oneself. For such a severe decrease in quality of life, no current treatments currently halt AD. Major AD theories include unhealthy protein aggregation and dying brain cells, especially the notable "neuronal" type. However, because these explanations have not produced sufficient solutions, we must revise our understanding of AD to more accurately and successfully treat it. Here, summarizing literature in neuroimmunology and biochemistry, it is posited that AD is primarily caused by aging and dysfunction in the brain's glia, or non-neuronal cells. From recent rodent models and post-mortem investigation of AD patients, studies agree that aging processes naturally accumulate damaging, oxidizing products. This buildup gradually wears down glia; as glia normally support neuronal cells and each other, this further favours accumulation of more damaging oxidants and unhealthy protein aggregation. These aging-dependent processes also create glia subtypes that add to AD pathology; many such subtypes are defined by impaired clearance of protein aggregates, and release molecules that causes pathological inflammation seen in AD. Two major AD glia subtypes are explored: multiple glia subtypes grouped together into a "senescent" label, and a specific "disease-associated" microglia (DAM), or a glia subtype that prematurely kills neuronal cells. As DAM may act as a key switch between pre-symptomatic and clinical AD, modulating these cell types and their induction by aging processes are vital.

References/Acknowledgments:

Thank you to Dr. Leanne Ramer, Dr. Marie-Ève Tremblay, and everyone from the Tremblay Lab that has helped manifest this into reality. Thank you to my parents, and friends as well - especially my Mom, for guiding the motivation and journey towards cultivating these ideas.