CARNEGIE MELLON UNIVERSITY BME 2022 FALL SEMINAR SERIES

Potential Role of Cytomegalovirus in Alzheimer's Disease



PRESENTED BY

SCHEDULE

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Currently afflicting more than 6.2 million Americans, Alzheimer's Disease (AD) is a chronic neurodegenerative condition that results from pathological brain aging and is the most common cause of cognitive dysfunction among older adults. The lack of comprehensive understanding regarding drivers of AD initiation and progression represents a critical barrier to progress for the field and has contributed to the current lack of viable treatment options. Pathogens are emerging as a potential contributor to the etiology of AD. Neurological manifestations and AD-associated phenotypes following acute infection with a variety of pathogens have been well documented. The long-term consequences of repeated infection are inadequately studied, though emerging epidemiological and preclinical evidence suggests that a higher lifetime infection burden impairs cognition, especially among organisms carrying AD genetic risk. That pathogens impact cellular metabolism may represent a key AD-related mechanism by which infection accelerates AD progression. What remains unknown is 1) whether a higher lifetime exposure frequency to pathogens, especially those that have limited neuronal tropism, can promote an AD phenotype, 2) how advanced age may potentiate this risk, and 3) the extent to which altered metabolism, due to infection, contributes to these effects. Our preliminary data demonstrates that intermittent infection induces altered metabolic phenotypes throughout the brain. Specifically, mitochondrial dysfunction and oxidative stress in brain microvascular endothelial cells (BMEC) correlates with reduced cognitive function. Defining the contributions of pathogen-associated metabolic rewiring in the brain provides novel therapeutic targets for prevention of AD.



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