Caerin 1.9: A Possible Treatment for Alzheimer's Disease? Investigating the Effects of the Caerin 1.9 Peptide on Amyloid-beta Aggregation and Phagocytosis by Cultured Microglia

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Alzheimer’s Disease (AD) is a global epidemic, affecting approximately 47 million people worldwide, increasing to an estimated 76 million by 2030. AD is the most common form of dementia and is the second leading cause of death in Australia, plaguing almost 350,000 people. Currently there is no cure, nor highly effective treatment, leaving millions of people worldwide with a drastically reduced quality of life. A major aspect of AD is the deposition of a protein called amyloid-beta and current therapies aimed at reducing production or aggregation of this protein have been largely unsuccessful. This experiment endeavoured to provide a new possible treatment for AD by investigating a way to increase the transport and clearance of amyloid-beta. Caerin 1.9 is a peptide found in the skin glands of the Australian tree frog, Litoria chloris, and has been studied for its antibiotic properties for many years. However this experiment looked at whether caerin 1.9 would be able to alter the uptake of amyloid-beta into microglia through phagocytosis. It was hypothesized that caerin 1.9 would increase this uptake - this was shown to be correct through an overall 35% increase in the clearance of amyloid-beta. The exact mechanisms of how caerin 1.9 affected this uptake could not be determined due to time constraints. However this experiment provided promising evidence for the effectiveness of caerin 1.9 as a potential therapeutic treatment, which would markedly reduce the medical, social and financial impacts on a patient and their loved ones.

Awards Won:

Third Award of $1,000