The Effect of Oleic Acid on Intracellular Calcium through Liposome Formation

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Alzheimer's Disease (AD) is a degenerative brain disorder resulting in neuronal death and loss of memory and cognition. However, the exact etiology of AD is not completely understood. This novel research elucidates the role of fatty acid accumulation, and particularly oleic acid (OA), reported in post mortem murine models of AD. Interestingly, excess OA has been shown to increase production of intracellular liposomes, which are known calcium storage sites in neuroepithelial cells. Also, calcium dysregulation is suggested to lead to excitotoxicity, with a concurrent accumulation of amyloid-beta and tau common in AD pathology. It was therefore hypothesized that at the in-vitro onset of excitotoxicity, administration of exogenous OA would further increase characteristics of AD pathology, including decreased viability, increased amyloid-beta and tau production, and increased intracellular calcium potentially facilitated by increased liposome production. The onset of excitotoxicity was induced with 5mM Ca+2 in SHSY5Y amyloid-producing neuroblastoma cells, rendering a 20% decrease in viability ($p<0.001$). Subsequently, cells were given combined treatment of Ca+2 (5mM) with innocuous OA concentrations (1-125μM). Combination treatment reduced cell viability by greater than 20% when compared to cells treated with OA alone ($p<0.01$). Combined treatment also increased liposome formation ($p<0.01$) and intracellular calcium concentrations ($p<0.01$), with increased extracellular amyloid-beta and tau observed at high OA concentrations ($p<0.001$). These results suggest treatment of OA in conjunction with calcium may lead to liposome aggravated potentiation of AD pathology. This research also suggests OA may act as a novel biomarker and may, in fact be significant in the etiology of AD.

Awards Won:

Second Award of $2,000