

A Comparison of Apolipoprotein E and β -Amyloid in Senile Plaques in Alzheimer's Disease

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Research in the field of Alzheimer's disease has shown that there are many potential factors involved in the onset and manifestation of the disease. Pathologically, the brain tissue of patients with Alzheimer's disease shows the presence of senile plaques and neurofibrillary tangles. Two of the proteins that have been found to deposit in these plaques are Apolipoprotein E and β -Amyloid. Individuals who possess at least one copy of the ϵ -4 allele of Apolipoprotein E have been shown to have an increased incidence of Alzheimer's disease compared to individuals who only have copies of the ϵ 2 or ϵ 3 alleles. It has also been found that individuals who have mutations in the gene for APP, the precursor protein of β -Amyloid, develop Alzheimer's disease at a very early age. Because of the potential roles both proteins seem to play in the development of Alzheimer's disease, we chose to compare the relative amounts of these two proteins in the plaques of Alzheimer's brains. We proposed that a correlation would exist between the amount of Apolipoprotein E and the amount of β -Amyloid present in these plaques.

In order to carry out this study we stained sections of tissue from the temporal lobe of brains with Alzheimer's disease and brains of controls. Alternating sections were stained for Apolipoprotein E and β -Amyloid. These sections were imaged and analyzed. The value obtained for each image represented the percentage of the image which was occupied by either Apolipoprotein E or β -Amyloid.

In our study we found a significant increase in the amount of both Apolipoprotein E and β -Amyloid deposited in the Alzheimer's brains compared to the controls. However, no significant correlation was found between the amount of Apolipoprotein E and the amount of β -Amyloid present in these deposits. The results of this study indicate that there is an increased deposition of Apolipoprotein E and β -Amyloid in Alzheimer's brains. However, it appears that the mechanisms by which these two proteins are deposited in the brain occur independently of one another.