

Abstract

Neurodegeneration associated with Alzheimer's disease is thought to be associated with the aggregation of a peptide, β -amyloid ($A\beta$) that accumulates in senile plaques during the disease. A variety of researchers have shown that aggregated $A\beta$ is associated with cell toxicity in vitro, and that strategies that modify $A\beta$ aggregation also prevent toxicity of the peptide. In this work, we examine the effects of Aspartame and a variety of other dipeptides at altering $A\beta$ aggregation. Initial work shows that aggregation of $A\beta$ in the presence of Aspartame results in $A\beta$ fibrils that are less stable than fibrils formed in the absence of Aspartame. We will show how dipeptide charge and hydrophobicity are related to ability of these dipeptides to alter $A\beta$ fibril formation, fibril stability, and fibril toxicity to cultured cells. This work may offer insight in the design of agents which alter $A\beta$ aggregation associated with Alzheimer's disease.