An in vitro Study of the Effectiveness of Cinnamon Compounds on the Degradation of Amyloid-B and Tau Protein in Alzheimer's Disease

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There are an estimated 5.1 million Americans currently suffering from Alzheimer's Disease, a neurodegenerative disorder, and by 2050, it is estimated that over 15 millions will be diagnosed (AD Sheet). Alzheimer's occurs when Amyloid-B plaques and Tau Neurofibrillary Tangles (NFT's) form in the hippocampus region, resulting in deteriorating cognition. Cinnamon, a common spice, has displayed characteristics in degrading these proteins (Qin 2010). Cinnamaldehyde, the main constituent of cinnamon, has been shown to bind to the cysteine residues in tau, preventing NFT's. Additionally, epicatechin, also found in cinnamon, has shown to prevent the formation of reactive oxygen species, which inhibits Amyloid-B plaques (RC George 2013). Both cinnamaldehyde and epicatechin have shown to pass the blood-brain barrier and remain effective against Alzheimer's (Peterson 2009). This experiment tested the treatment of cinnamaldehyde and epicatechin on Amyloid-B and Tau through SDS and Urea-PAGE to examine the effectiveness of these compounds in degrading the proteins. In this experiment, all proteins and compounds were diluted to their natural concentrations. Aggregation was induced in several preparations to analyze the effectiveness of the cinnamon compounds on aggregated and non-aggregated proteins. The proteins were then treated with cinnamon compounds and SDS and Urea-PAGE was used to determine the effectiveness of the cinnamon compounds after incubation. Both hypotheses were rejected. Not only were the cinnamon compounds overall ineffective on the degradation of non-aggregated proteins, they also did not degrade the aggregated proteins. In the future, I would like to investigate how cinnamon compounds can prevent Alzheimer's, instead of trying to inhibit it's progression.