

ABSTRACT

THESIS: USING ASTAXANTHIN AND VITAMIN E IN THE PROMOTION OF HIPPOCAMPAL CELL GROWTH AND PREVENTION OF BETA-AMYLOID AGGREGATION UNDER HYPERGLYCEMIC CONDITIONS

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Alzheimer's disease (AD) is one of the most prevalent neuro-degenerative disorders in the world, with 1 in 9 people age 65 years or older being affected. AD has been studied for comorbidities. A common comorbidity is type 2 diabetes mellitus (T2DM) which is characterized by prolonged levels of hyperglycemia. The purpose of this study was to investigate if under hyperglycemic conditions, would the combination of astaxanthin (ATX) and vitamin E (VE) result in decreased β -amyloid ($A\beta$) aggregation and promote growth of hippocampal cells, using an NE-4C cell line. The cells were analyzed for growth, $A\beta$ fibril degradation, and mitochondrial reactive oxygen species (ROS) production. These variables were measured using XTT, Thioflavin T, and MitoSOX assays. Data was analyzed using ImagePro 6 and/or Excel ($p < 0.05$). VE+ATX addition was expected to decrease hippocampal cell death while decreasing $A\beta$ aggregation under hyperglycemic conditions *in vitro*. The results of this study concluded that hippocampal cells in the presence of $A\beta$ did decrease ROS production at 5mM glucose +ATX+VE. In fact, the combination of ATX+VE decreased cell growth at all glucose concentrations. Decreased $A\beta$ fibrilization was significant at 5mM, 10mM and 25mM glucose +ATX+VE when compared to control cell treatments

that included the A β protein. Therefore, the results of this study suggest that patients with hyperglycemia may be able to take either ATX or VE or ATX+VE for neuroprotection. However, individuals with AD and hyperglycemia may not experience the same benefit, as the combination of ATX+VE may cause more harm to hippocampal cells than benefit when A β is present.