ABSTRACT

THESIS: Effects of Vitamin E on Hippocampal NE-4C Neuronal Cell Growth

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Vitamin E (alpha-tocopherol) is a lipid-soluble vitamin found within a variety of oils, nuts, and leafy greens. In a balanced diet, individuals receive approximately 10mg of Vitamin E a day. Over the counter supplements contain approximately 180mg to 450mg of synthetic Vitamin E, with no toxicity effects. Vitamin E is a common antioxidant that inhibits inflammation and increases protection of the plasma membranes. Recently, Vitamin E has been proposed to protect DNA and certain proteins against antioxidant activity. Vitamin E also protects against beta amyloid plaque formation and hyperglycemia *in vitro.* Since vitamin E is beneficial to cell survival, Vitamin E supplementation has the potential to influence cell culture growth. However, no studies have yet been conducted that examine the effects that Vitamin E may have on the initial growth stage of hippocampal neurons. In this study, we evaluated the effects of Vitamin E on NE-4C hippocampal neuronal cells, *in vitro*. NE-4C cell lines were incubated under normal glycemic conditions (5mM). Vitamin E was added to NE-4C cells at either 20μM, 30μM, or 40μM concentrations. Control groups consisted of either untreated cells or cells treated with 5mM glucose. The experiments were run in triplicate with consistent placement using a microplate reader. The cells were examined for their cell growth and concentration of reactive oxygen species, using an XTT Assay and MitoSOX Assay, respectively. Data was analyzed using a single-factor

ANOVA and 2-Tailed T-test. The results of this study concluded that hippocampal cells treated with either 20μM or 40μM Vitamin E in 5mM glucose possessed significantly less reactive oxygen species than cells treated with 5 mM glucose or 30μM Vitamin E and 5mM glucose. The cell growth analysis showed no significant difference in cell growth between treatment conditions. These results suggest that although additions of Vitamin E may not affect initial cell growth, treatments of 20μM and 40μM Vitamin E decrease mitochondrial reactive oxygen species production allowing for the prolongment of hippocampal neuronal function.