Investigating the Effect of Polyhydroxylated Small-Gap Fullerene as an Antioxidant for Amyloidß-Induced Free Radicals in NT2 Cells

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Alzheimer's Disease is currently an incurable disease that is growing over time. A current treatment-aim is to inhibit the neurotoxic effects of a protein peptide found in Alzheimer's disease. The neurotoxic protein in the brain of patients with Alzheimer's diesease, called Amyloid-beta (Abeta) results in the cells' production of free radicals. In Alzheimer's there are low levels of natural antioxidants that can potentially mitigate the effects of Abeta. The imbalance between free radicals and antioxidants in the brain is called oxidative stress. In this research, polyhydroxylated small-gap fullerene (PSGF), a basic, soluble carbon nanotube derivative, was studied as a potential antioxidant for free radicals produced by NT-2 (Neuron-like) cells in response to Abeta42 exposure. The PSGF (varying from C60 to C400, with peak distribution of C120) was hypothesized to be an effective antioxidant. In a 96-well plate NT-2 cells were cultured, and then DCFDA (fluorescence for free radicals) was applied to each well. Cells were exposed to Abeta42, or Abeta42 with different concentrations of PSGF. Using a fluorescence microplate reader, the fluorescence intensities of the wells were measured for quantification of free radicals present in wells, and effectiveness of PSGF as an antioxidant. Results indicated that fluorescence intensity values for cells exposed to Abeta42 with PSGF were similar to the fluorescence intensity value of cells not exposed to Abeta42. It can be concluded that PSGF is an effective antioxidant for NT-2 cells exposed to Abeta42. Potential use of PSGF in Alzheimer's patients can slow the progression of Alzheimer's disease.

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