Fe65/APP signalling and brain-derived neurotrophic factor in Alzheimer’s disease

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Country

United Kingdom

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Fe65/APP signalling and brain-derived neurotrophic factor in Alzheimer's disease

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Research Abstract

The amyloid precursor protein (APP) is strongly linked to Alzheimer’s disease. This is principally because it is cleaved by enzymes to produce amyloid beta peptide (Abeta) that is deposited in Alzheimer’s disease brains. However, cleavage of APP to produce Abeta is likely to change APP function and this may also contribute to Alzheimer’s disease. One function of APP is in nuclear signalling. This is where proteins in cells are moved into the compartment that stores the cells genetic material. Nuclear signalling causes alterations to many processes within cells. This function of APP is mediated by binding to another protein, Fe65. Recently we have found that brain-derived neurotrophic factor (BDNF), a substance produced in the brain induces

changes to Fe65 termed phosphorylation. BDNF has been shown to be protective in some Alzheimer’s disease models but its mechanism of action is unclear. This project is to investigate whether BDNF-induced Fe65 phosphorylation influences APP-Fe65 nuclear signaling. As such, it may provide insight into the mechanisms of protection of BDNF and so reveal new therapeutic targets for Alzheimer’s disease.

Further information available at:

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