Effect of GLP1 analogue, Liraglutide on cerebral glucose metabolism and microglial activation in Alzheimer’s disease

https://neurodegenerationresearch.eu/survey/effect-of-glp1-analogue-liraglutide-on-cerebral-glucose-metabolism-and-microglial-activation-in-alzheimers-disease/

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United Kingdom

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Effect of GLP1 analogue, Liraglutide on cerebral glucose metabolism and microglial activation in Alzheimer's disease

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Research Abstract

Alzheimer’s disease (AD) is a progressive neurodegenerative disease for which there is only minimal treatment available. Insulin resistance has been identified as a risk factor for AD. The newer anti diabetic drug, GLP-1 incretin analogue, Liraglutide has shown wide range of neuroprotective properties in mouse models of AD in extensive preclinical studies. Liraglutide crosses the blood-brain barrier, decreases microglial activation (50%), reduces amyloid plaque

formation in the murine brain, reduces levels of soluble amyloid oligomers, normalises synaptic plasticity in the hippocampus, protects the cognitive abilities of mice, prevents progressive neuronal damage as evidenced by maintaining cerebral glucose metabolism, and finally increases the proliferation of neuronal progenitor cells and the number of new neurons in the dentate gyrus. We therefore propose to test the effect of Liraglutide in patients with early Alzheimer's dementia on microglial activation and cerebral glucose metabolism using Positron Emission Tomography (PET). TSPO ([11C]PBR28/[18F]PBR111)-PET measures the microglial activation and [18F]FDG-PET measures the cerebral glucose metabolism. Liraglutide can be easily administered through daily subcutaneous injections and does not cause hypoglycaemia in non-diabetic individuals. 22 subjects with a diagnosis of AD will have detailed neurological and neuropsychometric assessment, T1 and T2 MRI, TSPO-PET and [18F]FDG-PET at baseline, followed by 1.2mg of daily sub-cutaneous injection of Liraglutide for one year with regular assessments. PET and MRI scans will be repeated at the end of one year treatment. We hope to find a reduction in microglial activation and stable glucose metabolism over the course of treatment.

Further information available at:

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