Therapeutic potential of boosting astrocytic Nrf2-mediated antioxidant signalling in combating white matter damage, amyloid pathology and cognitive deficits caused by chronic cerebral hypoperfusion

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Country

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Title of project/programme

Therapeutic potential of boosting astrocytic Nrf2-mediated antioxidant signalling in combating white matter damage, amyloid pathology and cognitive deficits caused by chronic cerebral hypoperfusion

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Alzheimer's Research UK

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€ 319,950

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3.0

The project/programme is most relevant to:

Alzheimer's disease & other dementias

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Cell Death | Oxidative Stress

Research Abstract
Prolonged modest reductions in blood supply to the brain (chronic hypoperfusion) are an early feature of Alzheimer’s disease (AD). There is evidence, including our work, that sustained hypoperfusion leads to over-production of harmful molecules (free radicals) causing cell damage through oxidative stress. This leads to increased production of amyloid, a key molecule implicated in AD. In normal healthy brain, there are defence mechanisms (anti-oxidant) to prevent damage to brain cells. In particular a new molecule has been discovered called Nrf2, which can switch on several antioxidant molecules and exert protection. This project will test the hypothesis that hypoperfusion leads to increased oxidative stress, amyloid production and brain damage associated with impaired cognition which can be ameliorated by boosting the anti-oxidant capacity of the brain through targeting Nrf2. Using experimental model systems, we will firstly determine the effects of hypoperfusion or increased amyloid on Nrf2 related signalling pathways, the extent of oxidative damage and whether this is exacerbated when levels of Nrf2 are reduced. Central to this proposal we will address whether boosting anti-oxidant mechanisms either by drugs, or by using specialised genetically modified mice in which anti-oxidant capacity is enhanced, can protect against the effects of hypoperfusion and amyloid.

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