Protection against reactive oxygen species in neurodegeneration

https://neurodegenerationresearch.eu/survey/protection-against-reactive-oxygen-species-in-neurodegeneration/ Principal Investigators Institution

Contact information of lead PI

Country

European Commission

Title of project or programme

Protection against reactive oxygen species in neurodegeneration

Source of funding information

European Commission FP7-Seventh Framework Programme

Total sum awarded (Euro)

€ 1,328,956

Start date of award

01/02/2013

Total duration of award in years

5.0

The project/programme is most relevant to:

Motor neurone diseases

Keywords Research Abstract Oxidative stress (OS) is implicated in the pathogenesis of some of the most devastating neurological diseases. Therefore, identification of pathways that counteract oxidative damage may lead to therapeutic strategies applicable to a wide range of disorders. I have recently identified the importance of the mitochondrial oxidation resistance 1 (Oxr1) gene for neuronal survival; loss of Oxr1 causes neurodegeneration in mice, whereas over-expression in vitro is able to confer protection against OS.

Deregulation of this protein is also observed in human ALS and additional mouse models of in neurodegeneration. Despite the apparent high level of conservation of Oxr1-related proteins, virtually nothing is known about their function in vivo. This proposal will establish an independent research programme to understand the role of Oxr1 and related proteins as part of novel pathways that provide protection against both OS and neurodegeneration.

Preliminary data I have generated has identified proteins that interact with Oxr1 in neurons; significantly, a number of these binding partners are mutated in neurodegenerative disease. Therefore, using a combination of mutational and biochemical analysis the relationship of these interactions to neuronal cell death will be investigated. Studies will also focus on the role of Oxr1 in mitochondria, examining the significance of this localisation to the control of the OS response. This will be complemented by in vivo studies in the mouse to to determine whether the loss or disruption of Oxr1-related proteins is critical for CNS function.

Using the latest techniques in molecular genetics and in vivo modelling this project has major translational opportunities and will provide an excellent opportunity to answer one of the key questions in neurodegenerative research.

Lay Summary Further information available at:

Types: Investments > €500k

Member States: European Commission

Diseases: Motor neurone diseases

Years: 2016

Database Categories: N/A

Database Tags: N/A