Analysis of genetic modulators for mitochondrial dysfunction in alpha-synuclein models for Parkinson's disease

https://neurodegenerationresearch.eu/survey/analysis-of-genetic-modulators-for-mitochondrial-dysfunction-in-synuclein-models-for-parkinsons-disease/

Question

Principal Investigators

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Related Institution

Université du Luxembourg

Contact information of lead PI Country

Luxembourg

Title of project or programme

Analysis of genetic modulators for mitochondrial dysfunction in alpha-synuclein models for Parkinson's disease

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FNR

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€ 172,990

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15/04/2014

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3

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Research Abstract

The importance of alpha-synuclein and mitochondrial dysfunction is well established in both familial and sporadic forms of Parkinson?s disease (PD). Increasing evidence is also indicating a multitude of functional interactions between alpha-synuclein and mitochondrial dysfunction in

PD. In this project we will analyse the modulation of alpha-synuclein based mitochondrial dysfunction by other genes involved in familial forms of Parkinson?s disease. The use of an already in house developed baculovirus system for the traceable analysis of double genetic perturbations will be used to overexpress alpha-synuclein variants and to knockdown familial PD genes. The moderately high number of resulting perturbation combinations and the complexity of mitochondrial phenotypes motivates the use of endpoint measurements via high content screening as main tool for an efficient multiparametric mitochondrial phenotype analysis. Hits from this screen will undergo further validation experiments targeting the time resolved analysis of gene expression patterns and mitochondrial physiology. Finally, the most important findings from the study will be validated in dopaminergic neurons derived from induced pluripotent stem cells.

Further information available at:

https://www.fnr.lu/projects/analysis-of-genetic-modulators-for-mitochondrial-dysfunction-in-%CE%B1-synuclein-models-for-parkinsons-disease-2/

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Investments < €500k

Member States:

Luxembourg

Diseases:

N/A

Years:

2016

Database Categories:

N/A

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