Identifying effectors of mutant C9Orf72 ALS/FTD to combat neurodegeneration

https://neurodegenerationresearch.eu/survey/identifying-effectors-of-mutant-c9orf72-alsftd-to-combat-neurodegeneration-2/

Principal Investigators

K.Talbot, G.Haase

Institution

Multiple

Contact information of lead PI
Country

United Kingdom|France

Title of project or programme

Identifying effectors of mutant C9Orf72 ALS/FTD to combat neurodegeneration

Source of funding information

CoEN

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€ 465,586

Start date of award

01/06/2018

Total duration of award in years

2

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Research Abstract
Hexanucleotide repeat expansions (HRE) are a type of genetic mutation. When they occur in the C9orf72 gene, they are the most frequent cause of amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD). Recent studies have identified several different potential triggers of nerve cell death related to this type of genetic mutation, but the specific pathways leading to ALS/FTD remain to be discovered.

The project aims to determine the pathways which trigger nereve damage by combining the power of studying human motor neurons grown in the lab, and other disease models including

yeast and zebrafish. To identify and characterise potential neurodegeneration pathways we willl compare the expression of large numbers of genes in motor neurons grown from patients with C9orf72 HRE mutations, cells from such patients where the genetic mutation has been corrected, and age-matched healthy controls. We will then study these effects in more detail in yeast and test whether or not these pathways are good targets for drug therapy in a zebrafish model of ALS/FTD.

In order to achieve these ambitious aims, we have gathered three leading European teams with documented expertise in the field of stem cell biology, ALS pathology and systems biology in this highly innovative and translational project.

Further information available at:

Types:
Investments < €500k

Member States:
France, United Kingdom

Diseases:
N/A

Years:
2016

Database Categories:
N/A

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