Protein damage control: regulation of toxic protein aggregation in aging-associated neurodegenerative diseases

https://neurodegenerationresearch.eu/survey/protein-damage-control-regulation-of-toxic-protein-aggregation-in-aging-associated-neurodegenerative-diseases/

Principal Investigators
Institution
Contact information of lead PI
Country

European Commission

Title of project or programme

Protein damage control: regulation of toxic protein aggregation in aging-associated neurodegenerative diseases

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€ 1,450,249

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5.0

The project/programme is most relevant to:

Neurodegenerative disease in general

Keywords
Research Abstract

The worldwide aging population will lead to a dramatic increase in the number of people with Alzheimer’s disease and other incurable age-related neurodegenerative diseases over the next few decades. By 2050 over 115 million are expected to suffer from these devastating diseases. The major pathological hallmark of these disorders is the accumulation of aggregation-prone disease proteins in aggregates in the brain. To understand the disease mechanisms and to identify targets for treatment, I aim to uncover the cellular pathways that regulate disease-protein toxicity and aggregation (proteotoxicity).

Opening up such exciting new avenues for research is our recent identification of a modifier of aggregation, which we named MOAG-4, as a general regulator of age-related proteotoxicity in worm (C. elegans) models for neurodegenerative diseases. MOAG-4 and its human counterpart SERF act independently of classical pathways that degrade proteins or prevent their aggregation, but their molecular role remains to be determined. I hypothesize that MOAG-4/SERF represents a new regulatory pathway of age-related proteotoxicity in neurodegenerative diseases.

With an ERC starting grant, I will uncover the pathway in which MOAG-4/SERF is operating, establish the mechanism by which the pathway regulates proteotoxicity, establish the evolutionarily conservation of the pathway in human cells, and establish its potential as a therapeutic target in patient-derived cells. By combining the power of C. elegans genetics with the development of cell-biological tools to visualize and monitor aggregation and toxicity in living and aging worms and in patient-derived cells, we will be at the forefront of providing new insights into disease-mechanisms. Our discoveries will offer new starting points for research and for the development of therapeutic interventions in the early molecular events of aging-associated neurodegenerative diseases.

Lay Summary
Further information available at:

Types:
Investments > €500k

Member States:
European Commission

Diseases:
Neurodegenerative disease in general

Years:
2016

Database Categories:
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