Mechanisms of cell dysfunction by aggregation dynamics of polyQ-containing proteins

https://neurodegenerationresearch.eu/survey/mechanisms-of-cell-dysfunction-by-aggregation-dynamics-of-polyq-containing-proteins/

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Contact information of fellow
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Mechanisms of cell dysfunction by aggregation dynamics of polyQ-containing proteins

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The project/programme is most relevant to:

Neurodegenerative disease in general

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Research Abstract
Protein aggregation is a hallmark of several ageing-related neurodegenerative diseases, such as Huntington’s disease (HD), Alzheimer’s disease and prion-mediated diseases. Different

cellular pathways influence the rate of aggregation, and clearance of intermediate protein species and aggregates in the cell (for example, autophagy or proteasomal degradation). Many signalling pathways regulate these processes. These signalling events, and the molecular pathways downstream are not completely understood. In HD there is an inverse correlation between the number of CAG triplets found in mutated huntingtin (htt) and the age-of-onset of the symptoms. However, there is a wide variation in the age-at-onset of the disease among carriers of short mutant glutamine tracts, suggesting that the genetic background strongly influences the severity of the disease. Hence the broad objective of this proposal is to find molecules that modulate protein aggregation.
We will use in vivo (C. elegans) and in vitro (mammalian cells) models of HD to find new molecules and pathways that modulate aggregation and toxicity induced by polyglutamines. The second objective of this proposal is to understand the mechanism by which mHtt toxic species alter cellular processes, with special focus on pre-synaptic function.

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Fellowships

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