

CeBioN

Cellular Bioenergetics in Neurodegenerative Diseases: A system-based pathway and target analysis

Mitochondria are parts of every cell that provide energy to enable cells to perform their day-to-day duties. However mitochondria can also kill cells by producing damaging molecules or by initiating a cell suicide program. These processes are believed to play a key role in the degenerative changes associated with Alzheimer's disease (AD), Parkinson's disease (PD) and Huntington's disease (HD).

CeBioN aims to understand if impairments in mitochondrial function are sufficient to trigger these neurodegenerative disorders, or if they represent an additional risk factor ('second hit' hypothesis) for developing the disease. It is also important to test whether mitochondria and different ways to 'boost' the cell's energy represent 'true' therapeutic targets.

CeBioN assembles a team of international experts with a track record in analysis of mitochondrial functions and energetics research. To determine the contribution of mitochondria and cell bioenergetics to disease progression in AD, PD and HD we will use two complementary approaches:

- 1) Analysis of common disease mechanisms focusing on alterations in mitochondrial functions and in the signalling pathways regulating bioenergetics.
- 2) High-throughput screen to identify drugs enhancing mitochondrial functions and demonstrating neuroprotection.

The CeBioN consortium will deliver much required insights into the contribution of mitochondria dysfunctions and bioenergetics to neurodegeneration in AD, PD and HD and will deliver novel therapeutic strategies. There are no disease modifying drugs currently on the market and therefore the CeBioN initiative is of highest importance as part of the work to understand the disease mechanisms and to develop efficient treatments for these devastating diseases.

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