

# Endoplasmic reticulum stress modulation by S-glutathionylation in Parkinson's disease

<https://neurodegenerationresearch.eu/survey/endoplasmic-reticulum-stress-modulation-by-s-glutathionylation-in-parkinsons-disease/>

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## **Funder**

FCT

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## **Country**

Portugal

## **Title of project/programme**

Endoplasmic reticulum stress modulation by S-glutathionylation in Parkinson's disease

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FCT

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€ 116,640

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01/01/14

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## **The project/programme is most relevant to:**

Parkinson's disease & PD-related disorders

## **Keywords**

## **Research Abstract**

The Endoplasmic Reticulum (ER) has important cellular functions in protein folding and maintenance of calcium homeostasis. Mitochondrial dysfunction and proteolytic pathways failure, common pathological features of Parkinson's disease (PD), may lead to accumulation of misfolded oxidized proteins causing ER stress. S glutathionylation, a reversible post-

translational modification can protect proteins against irreversible oxidation thus being instrumental in correct protein folding. Glutathione S-Transferase pi (GSTP) protects cells from reactive oxygen species (ROS) by several mechanisms including the modulation of protein S glutathionylation.

This project aims to contribute to the clarification of the role of ER stress response modulation, namely by S glutathionylation of ER stress-related proteins, in PD neurodegenerative processes. The neuroprotective role of GSTP in PD should also be further elucidated. The mechanism of action of tauroursodeoxycholic acid (TUDCA) as an ER stress response modulating agent will be explored with particular emphasis on the potential regulation of S glutathionylation, which can lead to novel therapeutic strategies.

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