

DISSECTING THE MECHANISMS OF MICROGLIA RESPONSE, DETERMINANTS AND BIOMARKERS IN AMYOTROPHIC LATERAL SCLEROSIS

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Funder

FCT

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Country

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Title of project/programme

DISSECTING THE MECHANISMS OF MICROGLIA RESPONSE, DETERMINANTS AND BIOMARKERS IN AMYOTROPHIC LATERAL SCLEROSIS

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Keywords

Research Abstract

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease due to selective loss of motor neurons (MN), mainly in spinal cord (SC), but other cell types as microglia are involved. Pathogenesis is not clarified nor the biomarker(s), although SOD1 and TDP-43 mutations, as well as neuroinflammation and excitotoxicity, are comprised. Microglia certainly contributes to disease progression. Although the mechanisms involved are unknown soluble factors released by microglia seem to be relevant to MN injury and astrocyte reactivity. We will investigate microglia phenotype and immunological alterations along ALS disease stages in an integrated neuron-glia context, the determinants implicated and the most consistent translational biomarkers. MN-like NSC-34 cell line with mutant SOD1 induced with doxycycline, SC astrocytes treated with r-mSOD1G93A, astrocyte- and MN-microglia co-cultures, and organotypical cultures depleted or non-depleted in microglia, and SC of TgSOD1G93A mice, among other ALS animal models, will be used. Analysis of microglia and MN cross-talk in synaptic plasticity and retrograde neurodegeneration will be performed in models of sciatic nerve denervation and in TgSOD1G93A mice.

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Fellowships

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