NOVEL IMMUNOTHERAPIES IN CHRONIC NEUROLOGICAL DISEASES – REDUCING PATHOGENESIS AND IMPROVING HEALING

https://neurodegenerationresearch.eu/survey/novel-immunotherapies-in-chronic-neurological-diseases-reducing-pathogenesis-and-improving-healing/

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Contact information of lead PI Country

Sweden

Title of project or programme

NOVEL IMMUNOTHERAPIES IN CHRONIC NEUROLOGICAL DISEASES - REDUCING PATHOGENESIS AND IMPROVING HEALING

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Swedish Research Council

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4

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Research Abstract

In Multiple Sclerosis, Glioblastoma multiformes and Alzheimers Disease we hypothesise that disease chronicity is due to dysregulation of immunological mechanisms associated with macrophage/microglia (Mf/mG) activation in the CNS. Our research program aims to develop novel immunotherapies for treatment of these neurological diseases, and we use a multifaceted approach with myeloid cell therapy, vaccination and tolerance induction. We developed a

myeloid cell therapy concept based on injection of specifically in vitro re-programmed Mf/mG in order to reset the immunological balance in vivo. Our focus in MS is to study healing of the damaged tissues, specific effects on both regulatory T cell function and neural stem cell development within the affected tissues. Combining immunomodulatory/regenerative therapies will be tested in chronic MS models. Our data demonstrate that transfer of pro-inflammatory activated Mf can cure mice of GBM and we will expand on this by refining the treatment and characterising the immunological mechanisms operating within the tumour environment. We test a novel CNS targeting strategy involving intra-arterial delivery of Mf/mG that circumvents the requirement of surgically opening the skull. In AD we will test the therapeutic effects of adoptive cell transfer of Mf/mG in amyloid and Tau models and use specific Mf/uG transient depletion in the CNS and blood to investigate the relative roles of these cell populations in pathogenic and healing responses.

Further information available at:

Investments < €500k
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Years: 2016
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Database Tags:

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Types: