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Depletion and Replacement of Brain Border Myeloid Cells

Tech ID: 34040 / UC Case 2025-772-0

BRIEF DESCRIPTION

A novel method for selectively targeting and modulating brain border-associated myeloid cells for the treatment of neurological disorders.

FULL DESCRIPTION

This technology involves the use of colony-stimulating factor 1 receptor (CSF1R) inhibitors to selectively deplete and replace brain border-associated myeloid cells (BAMs) such as meningeal macrophages, without affecting microglia. It enables the targeted manipulation of BAMs, potentially allowing for the delivery of therapeutic genes directly to the brain's border regions. This approach is particularly relevant for treating neurological disorders where myeloid cell dysfunction is implicated.

SUGGESTED USES

- » Treatment of neurological disorders such as Alzheimer's disease, multiple sclerosis, and lysosomal storage diseases.
- » Gene therapy applications for neurological conditions with identified genetic deficiencies or pathologies.
- » Development of more targeted and less invasive therapeutic delivery methods for brain disorders

ADVANTAGES

- » Selective targeting of brain border-associated myeloid cells (BAMs) without altering microglia populations.
- » Potential for delivering therapeutic genes directly to critical areas of the brain.
- » Utilizes FDA-approved CSF1R inhibitors, ensuring a level of safety and efficacy.
- » Offers a less invasive delivery method compared to direct intracranial injection.
- » Addresses the challenge of drug delivery across the blood-brain barrier.

PATENT STATUS

Patent Pending

RELATED MATERIALS

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OTHER INFORMATION

CATEGORIZED AS

- » **Materials & Chemicals**
 - » Biological
 - » Chemicals
- » **Medical**
 - » Delivery Systems
 - » Disease: Central Nervous System
 - » Gene Therapy
 - » Research Tools
 - » Therapeutics
- » **Research Tools**
 - » Antibodies
 - » Other
 - » Reagents

» Hofsfield, L. A., et al. Green, K. N. (2020). Effects of long-term and brain-wide colonization of peripheral bone marrow-derived myeloid cells in the CNS. J. Neuroinflammation. 17.

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