# Targeting of Microglia in Neurogenerative Diseases

## Innovative approach to modulating non-plaque-associated microglia for the treatment of neurodegenerative diseases such as Alzheimer's disease

# Technology

This technology introduces a pioneering microglia-specific therapy targeting non-plaque-associated microglia (non-PAM) via the Colony-Stimulating-Factor-1-Receptor (CSF1R) to slow Alzheimer's neurodegeneration.

Unlike conventional approaches focused on plaque-associated microglia, it modulates reactive non-PAM populations—highly responsive to environmental cues like gut microbiome dysbiosis or LPS (Lipopolysaccharids)—that clonally expand toward amyloid plaques and actively drive pathology. Microglia, resident CNS macrophages with phenotypic heterogeneity (characterized via transcriptomics, single-cell analysis, multicolor fluorescence mapping, epigenetics, and immunohistochemistry), are key players in Alzheimer's, affecting over 55 million people worldwide through irreversible neuronal loss from plaques, tau tangles, and neuroinflammation.

CSF1R, a tyrosine kinase receptor expressed mainly in myeloid cells, binds CSF1/IL-34 to regulate survival, proliferation, differentiation, development (migration/differentiation), perinatal synaptic pruning, and adult maintenance. Dysregulation impairs neuronal function in neurodegeneration; current amyloid-cascade therapies fail, while genetic factors (TREM2/APOE) underscore microglia's potential. Evidence shows antibiotics reduce non-PAM expansion, LPS promotes it—offering precise modulation points without broad immunosuppression.

This innovative technology needs industrial partners for the further development and market readiness.

**Responsible Scientist**

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## Innovation

- **Non-PAM Targeting:** Focus on non-plaque-associated microglia as a novel therapeutic target.
- **Dynamic Modulation:** Clonal expansion modulable via microbiome/LPS
- **Spatial Heterogeneity:** Distinguishes reactive non-PAM from PAM for precise therapy.

## Application

- **Primary Application:** Therapy for Alzheimer's disease through targeted modulation of non-PAM to slow neurodegeneration
- **Extended Indications:** Treatment of further neurodegenerative diseases via modulation of microglial dynamics.
- **Combination Therapy Potential:** Integration with microbiome modulators (e.g., antibiotics/LPS inhibitors) for synergistic effects.

## Development Status

- Early stage

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