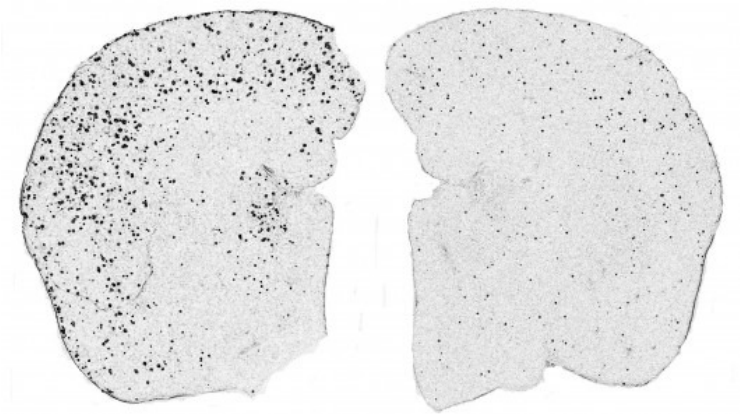
New Treatment for Alzheimer's Disease



Plaque deposits in the brain tissue of an untreated mouse (left), and one treated in the study

A research Organization has developed a new therapeutic approach in fighting Alzheimer's disease, the invention details are as below :

Evidence was found that modulation of cytokines IL-12 and/or IL-23 or its receptors impacts Aβ-plaque burden in Alzheimer's disease (AD). This is highlighted by the following aspects:

- It was found that myeloid cells, namely microglia, typically surrounding Aβ-plaques and known to be a major source of inflammatory cytokines in the brain, showed an robust upregulation e.g. of IL-12/IL-23 p40 and IL-23 p19 in Alzheimer's APPPS1 mice when compared to age-matched wild-type controls.
- Genetic ablation of IL-12 and/or IL-23 or its receptors (e.g. of p40, p19, p35 and il12rb1) resulted in a consistent, strong and significant reduction in the Aβ-plaque load of Alzheimer's APPPS1 mice at various time points investigated (120 and 250 days of age).
- In order to demonstrate the feasibility of manipulating IL-12 and/or IL-23 or its receptors for treating AD, blocking anti-p40 antibodies were injected into Alzheimer's APPPS1 mice and induced a robust and statistically plaques when given prior to plaque significant reduction of A development, or reduced cognitive decline and soluble Aβ species in aged APPPS1 with already established plaque pathology
- When comparing p40 protein levels in cerebrospinal fluid (CSF) specimens of subjects with Alzheimer's disease (n = 39) to disease control cases (n = 20), we found a significant linear correlation of the cognitive performance assessed by the mini-mental score evaluation (MMSE) and CSF p40 values, suggesting that the IL-12/IL-23 pathway poses a novel pharmacological target to combat Alzheimer's disease.
- Since clinical trials already used anti-p40 blocking antibodies for the treatment of various autoimmune diseases, data on the safety and tolerance of anti-p40 blocking antibodies exists.

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