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Anti-TNF-α reduces amyloid plaques and tau phosphorylation and induces CD11c-positive dendritic-like cell in the APP/PS1 transgenic mouse brains.

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Abstract

Inflammation plays an important role in the pathogenesis of Alzheimer's disease (AD). Overexpression of tumor necrosis factor-α (TNF-α) occurs in the AD brain. Recent clinical studies have shown that the anti-TNF-α therapy improves cognition function of AD patients rapidly. However, the underlying mechanism remains elusive. The present study investigates the effects of intracerebroventricular injection of the monoclonal TNF-α antibody, Infliximab, on the pathological features of AD in the APP/PS1 double transgenic mice. We found that Infliximab administration reduced the levels of TNF-α, amyloid plaques and tau phosphorylation as early as three days after daily injection of 150μg Infliximab for three days. The number of CD11c-positive dendritic-like cells and the expression of CD11c were found to be increased concurrently after Infliximab injection. These data suggested that the CD11c-positive dendritic-like cells might contribute to the Infliximab-induced reduction of AD-like pathology. Further, our results support the use of anti-TNF-α for the treatment of AD.

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