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### Neuroinflammation and Alzheimer's Disease: new data boost the hypothesis and the possibility of a treatment

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A recent paper and a commentary published on the January issue of the freely available *Journal of Neuroinflammation* ([Tobinick, Gross 2008](#) e [Griffin 2008](#)) provide new insight into the issue of the relationship between inflammation and Alzheimer's disease. The data are focused on TNFalpha and sound relevant and convincing even if the numbers of treated patients are small and the reported case history describes just one patient. TNFalpha increases interleuchin-1 which in turn favours the formations of plaques and tangles. Moreover TNFalpha is a gliotransmitter, which is released from glial cells surrounding the synapse and modulates synaptic activity. Several literature data suggest that inhibition of TNFalpha may have beneficial effects in AD.

The authors published in 2006 a [paper](#) investigating the effect of etanercept treatment (antagonizing TNFalpha) in AD patients. In order to do so, the authors developed a new administration technique, perispinal extrathecal administration in the posterior neck, which was hypothesized to improve delivery of etanercept to the brain via the cerebrospinal venous system. It is worth to remember that etanercept is a fusion protein neutralizing TNF alpha, used in rheumatoid arthritis, which does not cross the blood brain barrier when administered by conventional ways. Moreover, during the 6-month trial the authors noticed that patients treated with etanercept showed quick improvement of mental performance within minutes from the weekly administration of the drug.

In the January 2008 issue of neuroinflammation they describe in depth one of such cases accurately monitoring the acute effects of the treatment, which improved several cognitive domains, including executive function. The quick effect may well be due to inactivation of TNFalpha in turn leading to a relief of the inhibitory action of this cytokine on synaptic activity. It is an important information that brings back the attention of the researchers on acute synaptic activities in Alzheimer's disease (as we did for beta amyloid, see: [Preda et al. 2007](#)) and indicates the possibility that targeting rapid synaptic functions with drugs may ameliorate the symptoms of the disease.

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