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ANTI-INFLAMMATORY TREATMENT APPROACHES IN MAJOR DEPRESSION

N. Müller, M.J. Schwarz
Psychiatry and Psychotherapy, Ludwig Maximilians University, München, Germany

Proinflammatory cytokines, such as IL-6, IL-1 and TNF-a appear to be elevated at least in the peripheral blood of depressed patients. Thus, the activity of the enzyme IDO, which is driven by pro-inflammatory cytokines and regulates the tryptophan/kynurenine metabolism may be enhanced in depressed patients through these cytokines. Although IL-6 does not directly act on IDO, its elevated levels in serum may contribute to IDO activation within the CNS by the stimulatory effect on PGE_2, which acts as cofactor in the activation of IDO. This fits with a report on the correlation of increased in vitro IL-6 production with decreased tryptophan levels in depressed. Due to the increase of proinflammatory cytokines and PGE2 in some psychiatric patients, antiinflammatory treatment would be expected to show advantagous effects in schizophrenic and depressed patients. Cyclo-oxygenase-2 inhibitors have been evaluated in major depression. We were able to demonstrate a statistically significant therapeutic effect of the COX-2 inhibitor on depressive symptoms in a randomized double blind pilot add-on study using the selective COX-2 inhibitor celecoxib in MD. Another randomized double-blind study in fifty depressed patients suffering from MD also showed an statistically significant better outcome of the COX-2 inhibitor celecoxib plus fluoxetine compared to fluoxetine alone. Additionally, results of the clinical study of celecoxib add-on to sertraline and the effects of this anti-inflammatory therapy approach to inflammatory markers planned by the MOODINFLAME consortium will be presented as far as available. Further on, alternative therapeutic strategies based on immune-modulatory effects will be discussed.

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