

„Autoimmunity, inflammation and cytokine networks in psychosis“

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The field of “psychoneuroimmunology” has gained increasing interest and research work during the last 20 years. Several epidemiological studies demonstrate a co-occurrence of autoimmune diseases, chronic inflammatory conditions, and mental disorders. Since in many cases the elevated prevalence of autoimmune/chronic inflammatory diseases existed before the onset of psychosis, the hypothesis was put forward that the onset of psychosis could be induced by an inflammatory process elicited by the autoimmune reaction. Animal models of psychotic conditions (maternal stress and inflammation paradigms) suggest that monocyte/microglia activation can be the result of a combination of genetic predisposition and/or first hit early stimulations of the monocytes and microglia, leading to developmental brain abnormalities. This is followed by later second hit, which activates the microglia and leads to severe functional abnormalities of the neuronal circuitry in the brain. However, the majority of the available clinical studies in psychosis are not focused on the cells of the myelomonocytic and T cell lineage, but on levels of cytokines, chemokines, and other compounds mainly produced by these or related cells.

The aim of our study was to analyze the serum concentrations of type-1, type-2, type-17 and regulatory cytokines in drug-naive patients with first episode psychosis and schizophrenia in relapse. We have demonstrated that type-1 and type-17 responses are blunted and type-2 overweight in schizophrenia. Our results also implicate anti-inflammatory response through TGF- β production. Analysis showed that TGF- β and IL-23 can be valuable markers for psychosis. The presence of enhanced anti-inflammatory/immunosuppressive activity in schizophrenia may be an attempt to counteract or limit ongoing pro-inflammatory processes and downregulating chronic inflammation. A chronic pro-inflammatory stage with a robust type-2 response, including high IL-6 levels, may also predominate in later stages and may possibly lead to autoimmunity. Our results suggest that immune pattern in schizophrenia can be similar with those in atopic disorders and this cytokine disbalance can be corrected with antipsychotics.