Editorial

Monoclonal Antibody for Reducing Memory and Learning Problems in Schizophrenia

Schizophrenia is a chronic debilitating psychiatric illness that accounts for a significant portion of the burden caused by mental illnesses worldwide. Primary negative symptoms of schizophrenia are not secondary to extrapyramidal, depressive or positive symptoms [1,2]. Negative symptoms are the core features of the illness which are associated with long-term functional disability and poor outcome [1,3]. These symptoms include deficits in social and emotional functioning, blunted affect and lack of spontaneity. There is a growing body of evidence for the role of inflammation and immune system dys-regulation in psychiatric disorders [4]. Although the precise pathophysiology of schizophrenia is not completely known, a number of recent studies support the probable pathologic role of immunologic dysfunction in this disorder. Assessing serum cytokine levels such as interleukin 1 (IL-1), IL-2, IL-6, and chemokine CCL11 in schizophrenic patients demonstrates profound alterations compared to healthy matched controls [4]. Furthermore, increased cyclooxygenase-2 (COX-2) expression as well as prostaglandin E2 production in schizophrenia, are among other postulated etiologies supported by recent studies [4]. On the other hand, it has been shown that immune response imbalance is associated with decreased activity of indoleamine 2, 3-dioxygenase enzyme which subsequently leads to accumulation of kynurenic acid, an endogenous antagonist of glutamate N-methyl-D-aspartate (NMDA) receptor. Compared with anitinflammatory agents like celecoxib and NAC, monoclonal antibodies also have more potent anti-inflammatory properties. Indeed, COX-2 inhibitors and N-acetylcysteine have moderate efficacy in treatment of schizophrenia and autism [1,2,5]. British scientists have begun testing a radically new approach to treating schizophrenia based on emerging evidence that it could be a disease of the immune system. Evidence for prenatal and premorbid immune risk factors for the development of schizophrenia in the offspring is highlighted [6,7]. Then key evidence for immune dysfunction in patients with schizophrenia is considered. A collaboration between the Medical Research Council (MRC) and King's College London, is based on emerging evidence that schizophrenia may be an immune disease. The drug, natalizumab, works by targeting microglia, a type of immune cell residing in the brain which are thought to be overactive in people at risk of developing schizophrenia [6,7].

References

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