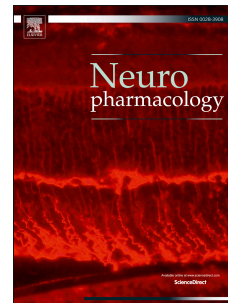


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Autoantibody-mediated diseases of the CNS: Structure, dysfunction and therapy

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**Autoantibody-mediated diseases of the CNS: Structure, dysfunction and therapy**

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**Abstract**

The field of neuronal autoantibody associated diseases of the central nervous system has expanded dramatically in the last few years. The range of identified neuronal and glial antibody targets has led to the accurate classification of a number of syndromes which each associate with characteristic clinical features. These diseases are especially important due to their frequent response to immunotherapies. Antibodies against the N-methyl, D-aspartate receptor (NMDAR) and leucine-rich glioma inactivated 1 (LGI1) are the commonest autoantibodies known in patients with autoimmune forms of encephalitis. Patients with NMDAR-antibodies often present with psychiatric symptoms and a movement disorder, whereas patients with LGI1-antibodies have frequent seizures and prominent amnesia. In contrast, aquaporin-4 and myelin-oligodendrocyte glycoprotein antibodies are found in patients with inflammation of the spine and optic nerves. The antigenic-specificities appear to determine the associated clinical syndromes, hence the detection of these antibodies informs clinical practice and the biology of these diseases. Indeed, the mechanisms of antibody action are being intensively studied *in vitro* and *in vivo*. Overall, these studies

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