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Sommario/riassunto

Over the last years it has become evident that many neurological diseases of the central nervous system (CNS) are induced by a specific adaptive immune response directed against molecules expressed on CNS-resident cells. Well-recognized examples are anti-N-Methyl-D-Aspartate Receptor (NMDAR) encephalitis which is characterized by the presence of antibodies against neuron-expressed NMDAR, or neuromyelitis optica (NMO), induced by antibodies to astrocyte-expressed aquaporin-4. Many more examples exist, and antibodies, and T or/and B cells have increasingly been associated with CNS disease. Often the symptoms of these diseases have not been typically reported to have an immune aetiology. Beside classical neurological symptoms like ataxia, vision disturbance, and motor or sensory symptoms, these can include cognitive disturbances, behavioral abnormalities, or/and epileptic seizures. Although much has been learned regarding the pathophysiology of prototypic examples of these disorders, there are still major gaps in our understanding of their biology. This may be due to the fact that they are rare diseases, and their therapies are still very limited. This research topic includes contributions addressing the analysis of the adaptive immune response driving disease including target antigens, molecular epitope mapping, and factors involved in the disease pathogenesis such as complement activation cascades, genetic and genomic regulation, as well as environmental triggers. Diagnostic criteria and methods, and treatment

are also discussed. The overall aim of the volume is to review progress in our pathophysiological understanding of immune-mediated CNS disorders in order to advance diagnostic and therapeutic approaches, and ultimately improve outcomes for patients.