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Pathophysiological Concepts and Their Potential Impact on Patients

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Sommario/riassunto

Research and advances in the treatment of Multiple Sclerosis (MS) is a story of growing success. Great progress has been made in identifying the molecular mechanisms that can be utilized to develop highly efficacious treatments for MS patients. Since the middle of the last century, when studies using an inflammatory autoimmune model of MS i.e. experimental autoimmune encephalitis (EAE) were initiated, research on MS has since led to an enormous growth of knowledge. The limitations arising from these animal models have been partially compensated by insights generated from histopathological, immunological, genetic, imaging and clinical studies of MS. The importance of T cells and, recently of B cells, in MS has been elucidated and the significance of CNS resident cells, particularly in the progressive course of MS has been highlighted. The importance of environmental factors and genetic predisposition have also been recognized and the interaction between the individual and its environment seems to play key roles in the pathogenesis of this disease. However, definite causative agent(s) or single gene(s) involved in MS still remain unidentified. A deeper understanding of the immunological pathways involved goes hand in hand with further improving therapy for MS. Focal inflammation in MS, caused by de novo CNS infiltration, can be prevented effectively using peripherally-acting

drugs. However, diffuse CNS intrinsic inflammation and neurodegeneration are not targeted by the current arsenal of therapeutics and patients with progressive disease courses remain difficult to treat. Generating insights into pathophysiological processes and the clinical translation of this knowledge have been a formula of success for treating inflammatory diseases. The future will show whether this will hold true for CNS-trapped inflammation and neurodegeneration and whether the ultimate goal - truly curing MS - will be possible.