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IMMUNOLOGY OF MULTIPLE SCLEROSIS*

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■ Abstract Multiple sclerosis (MS) develops in young adults with a complex predisposing genetic trait and probably requires an inciting environmental insult such as a viral infection to trigger the disease. The activation of CD4⁺ autoreactive T cells and their differentiation into a Th1 phenotype are a crucial events in the initial steps, and these cells are probably also important players in the long-term evolution of the disease. Damage of the target tissue, the central nervous system, is, however, most likely mediated by other components of the immune system, such as antibodies, complement, CD8⁺ T cells, and factors produced by innate immune cells. Perturbations in immunomodulatory networks that include Th2 cells, regulatory CD4⁺ T cells, NK cells, and others may in part be responsible for the relapsing-remitting or chronic progressive nature of the disease. However, an important paradigmatic shift in the study of MS has occurred in the past decade. It is now clear that MS is not just a disease of the immune system, but that factors contributed by the central nervous system are equally important and must be considered in the future.

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