Pediatric Neurology: Chapter 111. Mendelian predisposition to herpes simplex encephalitis (Handbook of Clinical Neurology)

Shen-Ying Zhang, Laurent Abel, Jean-Laurent Casanova

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Herpes simplex encephalitis (HSE) is the most common sporadic viral encephalitis in the Western world. The pathogenesis of HSE, which affects a small minority of HSV-1-infected individuals, has long remained elusive. Mendelian defects in the TLR3-interferon (IFN) and IFN-responsive pathways were recently shown to predispose to HSE, at least in some children. Autosomal recessive STAT-1 deficiency and X-linked NEMO deficiency were found in children with both mycobacterial disease and HSE. Autosomal recessive UNC-93B deficiency and autosomal dominant TLR3 deficiency were then described in children with isolated HSE. These discoveries provided proof-of-principle that HSE may result from a novel group of single-gene inborn errors of interferon (IFN)-mediated immunity. The TLR3–UNC-93B-dependent production of IFN-α/β and IFN-λ is essential to confer protective immunity to HSV-1 in the central nervous system during the course of primary infection in childhood.

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