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Molecular basis of multiple sclerosis explains the disease pathophysiology

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Multiple sclerosis (MS) is a complex, multifactorial autoimmune disorder of the central nervous system (CNS) that causes inflammation, demyelination and neurodegeneration. The increased prevalence of this disease in Arabian Gulf Countries (AGCs) has captivated many over the last several years. To explain the disease pathophysiology, one must consider the smallest variant within the body; the cell. Understanding the plethora of cellular variables involved is critical to help clarify why such disease tends to increase in these populations. MS develops from the interaction of different genetic and environmental factors. Genetic, epigenetic and even mitochondrial genomic variants are all associated with immune response initiation, facilitation of migration through the blood-brain barrier (BBB), inflammatory molecule mediation and the attack of cellular components. Collectively, this culminates in CNS demyelination and predisposition to MS symptoms. More so, environmental modulators such as vitamin D, UVR, EBV infection, smoking and obesity, influence disease pathophysiology through modulation of gene transcription, thereby predisposing to MS. Detailed knowledge of susceptibility factors underlying any disease is essential to properly understand disease pathophysiology, especially if correlated with population-related variables.

Biography

Eiman M A Mohammed has completed her MSc in Molecular Biology from Kuwait University, College of Medicine. She is currently working at the Immunology Laboratory in the Kuwait Cancer Control Centre, the only referable laboratory for organ transplantation testing, autoimmune disease testing and HLA typing. She has published two papers on multiple sclerosis.

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