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Understanding the role of immunological derangements in the pathophysiology of autism

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Autism is a neurodevelopmental disorder characterized by communication deficits, repetitive behaviors and impairment in language, cognition and socialization. Males are more affected by autism than females, showing a ratio of 4:1. The prevalence of autism has been increased dramatically over the past 30 years, before the 80s, the prevalence was estimated at 1:2,000 children to 1:100-200 in 2009. Most recent record is 1 in 50 children which is currently the most widely used figure. The need to clarify the causes behind the increase of autism prevalence and the underlying pathophysiology has become more urgent since the number of diagnosed cases has risen dramatically in recent years. Autism behavioral symptoms are frequently accompanied by immunological derangements including dysregulated cellular immune response, chronic inflammatory states represented as elevated tumor necrosis factor-α (TNF-α) and interferon-γ (IFN-γ), abnormal pro-inflammatory/anti-inflammatory cytokines (e.g., Il6/IL10), elevated chemokines and neuroimmune alterations. Currently, the involvement of the immune pathology in autism remains unclear and better understanding would be beneficial for earlier diagnosis and interventions. This presentation aims to highlight the most current aspects regarding the etiology of autism with particular reference to the involvement of inflammatory events occurring in the periphery and into the brain and how they can affect the early development of the brain and induce autism phenotype. Relationship between immunological derangements, glutamate excitotoxicity, mitochondrial dysfunction and oxidative stress as the most four common mechanisms related to the pathophysiology of this neurodevelopmental disorder will be discussed. Association between neuroimmune alteration and severity of autism measured as social responsive scale (SRS) and childhood autism rating scale (CARS) will be clarified through considering ours and others most recent published work.

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Mir193a expression pattern in lymph, spleen and brain samples and cell cultures of experimental autoimmune encephalomyelitis induced mice

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Tau is naturally a neuron-specific soluble protein that promotes microtubule assembly and stabilization. When pathologically modified, tau dissociates from microtubules and becomes insoluble aggregates called neurofibrillary tangles (NFTs). The NFTs formation is one of the main significant pathological signatures in Alzheimer's disease (AD) and multiple neurodegenerative disorders classified as tauopathies. NFTs are accumulated in axons and dendrites, thereby causing degeneration of tangle-bearing neurons. In addition, tau oligomers propagate in neurons and acting as a seed for native tau aggregation. Accordingly, great efforts have been made to investigate the mechanism of tau aggregation and to identify the pathogenic tau species. Diverse intracellular modifications make soluble tau to be a susceptible substrate for the soluble tau oligomers and insoluble filamentous aggregates. Due to the implications of tau pathology in many neuro-degenerative disorders, preventing the pathological tau aggregation become an important therapeutic strategy to halt the disease. However, progress has been slow due to the lack of understating tau aggregation mechanism. Tau aggregation is a multi-step process regulated by complicated cellular pathways. In tau pathology, diverse tau modifications including phosphorylation, oxidation, acetylation and truncation promote tau aggregation in a defected neuron. At the same time, the neuron activates cellular defense mechanisms such as glycosylation. We are currently focusing on diverse tau modifications associated with tau aggregation, strategy of tau aggregation inhibition, small molecule for tau imaging, and cell-based model that could monitor and quantify tau aggregation process in living cells.

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