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A review on autism

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Autism is a congenital neurological disorder characterized by impairment of socialization, abnormalities of communication, limited activity and curiosity. Clinical signs of Autism Spectrum Disorders (ASD) are frequently presented at 3 years of age with abnormalities in social, communication and play behaviour, though early indicators of autism can be detected as early as 14 months of age. Repetitive, stereotyped, and obsessive compulsive–like behaviours are also prominent features of the disorder and are often accompanied by cognitive impairment, seizures or epilepsy, gastrointestinal complaints, disordered sleep, and others are frequent problems in the clinical profiles of patients with autism. There are certain factors that contribute to the pathogenesis of ASD, such as dysfunctions of the serotonergic system, have been implicated in autism. The serotonergic system appears to be developmentally dis-regulated in autism. The study suggested that the clinical onset of autism appears to be preceded by two phases of brain growth abnormalities: a reduced head size at birth, then a sudden and excessive increase between 1–2 months and 6–14 months of age. Neuroimaging studies have shown that an abnormal pattern of brain overgrowth also occurs in areas of the frontal lobe, cerebellum and limbic structures between 2 and 4 years of age, a pattern that is followed by abnormal slowness in brain growth. Another study revealed that the oxidative stress might be unregulated in patients with ASDs, possibly due to decreased ability to neutralize free radicals. One study of autistic children and controls reported that plasma S –adenosyl homocysteine, which was used as an indicator of methylation ability, was significantly lower in autistic children. Another study found reduced plasma levels of the key endogenous antioxidant S–adenosyl methionine. Other reviews suggested that the Erythrocyte Superoxide Dismutase (SOD) and the endogenous antioxidants plasma glutathione peroxidase and erythrocyte glutathione peroxidase (GSH-Px) were also significantly reduced in autistic children. Increased mitochondrial metabolism and oxidized mitochondrial proteins in temporo-cortical gray matter in post-mortem samples from autistic patients as compared to controls was also seen. Calcitriol down-regulates production of inflammatory cytokines in the brain, which have been associated with autism. Vitamin D deficiency impairs glutathione metabolism, which may explain the link between autism and oxidative stress, as well as autism and mercury accumulation. Consumption of vitamin D containing fish during pregnancy reduces autistic symptoms in children. It seems probable that autism's neuro-developmental defect is 'multi-domain' in origin (rather than a single anomaly) and is, hence, distributed across numerous levels of study (genetic, immune-pathogenic, etc.). A more definitive understanding of the pathogenesis could facilitate the development of better treatments for this complex psychiatric disorder.

Biography

Juhi Tiwari is an MPharm in pharmacology from Jayoti Vidya Peeth Womens' University, Jaipur, India. She has attended National and International conferences and has published research paper on antipyretic effect.

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