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Global Congress on NEUROSCIENCE, PSYCHIATRY AND MENTAL DISORDER

March 09 , 2022 | Webinar

Tumor Necrosis Factor Alpha (TNF Alpha) blockade and Multiple Sclerosis (MS), exploring new avenues

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Multiple sclerosis (MS) is the most common disabling disease of the central nervous system (CNS) with a progressive neurodegenerative pattern. It is characterized by demyelination of white matter in CNS and apoptosis of oligodendrocytes. Tumor necrosis factor (TNF) alpha is a major cytokine in the pathogenesis of MS. However, the failure of TNF alpha inhibitors in preclinical and clinical trials disapproved of their use in MS patients. Nevertheless, failures and misses sometimes open avenues for new hits. In the later years, it was discovered that TNF signalling is mediated via two different receptors, TNFR1 and TNFR2, both of which have paradoxical effects. TNFR1 mediates demyelination and apoptosis, while TNFR2 promotes remyelination and neuroprotection. This explained the cause of the failure of non-selective TNF alpha-blockers in MS. It also enlightened researchers that repurposing the previously formulated non-selective TNF alpha-blockers using a receptor-selective approach could lead to discovering novel biologic agents with a broader spectrum of indications and better safety profiles. This review focuses on a novel premier TNFR1 blocker, atrosab, which has been tested in animal models of MS, experimental autoimmune encephalomyelitis (EAE), where it demonstrated a reduction in symptom severity. The early promise shown by atrosab in preclinical studies has given us hope to find another revolutionary drug for MS in future. Clinical trials are still going on which will finally decide whether this drug can be used as a better therapeutic agent for MS or not.

Biography

Maryam Zahid is a medical doctor by profession who is interested in pursuing her clinical career in the field of neurology. She is also enthusiastic in neurological research focused on finding better treatment options for currently untreatable neurological disorders. She recently published a review article about selective inhibitors of TNF alpha as a possible disease modifying agent for multiple sclerosis in future.