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GABA and Glutamate Imbalance in Autism and Their Reversal as Novel Hypothesis for Effective Treatment Strategy: An update

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ABSTRACT

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by reduced social communication and repetitive behaviors. The etiological mechanisms of ASD are still unknown; however, the GABAergic system has received considerable attention due to its potential as a therapeutic target. Based on the fact that individuals with autism demonstrate altered gene expression concomitant with impaired blood brain barrier (BBB), and gut barrier integrities, so increased glutamate levels in the blood and platelets of ASD patients can be related to lower numbers of cerebellar GABAergic neurons, less active GABA-synthesizing enzymes, and decreased brain GABA levels. Excitotoxic levels of released glutamate trigger a cascade of deleterious cellular events leading to delayed neuronal death. According to our understanding of glutamate excitotoxicity, GABA supplementation could theoretically be useful to treat certain autistic phenotypes. While there is still no effective and safe medication for glutamate-related cell damage and death, combined efforts will hopefully develop better treatment options. Here I hypothesize that an integrated treatment strategy with GABA supplements, regulation of chloride (Cl-) and magnesium (Mg2+) levels, vitamin D supplements, probiotics to enhance GABAA receptor and glutamate decarboxylase (GAD) expression, and memantine to activate glutamate transporters and inhibit NMDA receptors, could collectively reduce glutamate levels, maintain functional GABA receptors and thus treat repetitive behavior, impaired social behavior, and seizure activity in individuals with autism.