



A New Pharmacologic Approach for the Potential Reduction of Autism and Multiple Sclerosis

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Abstract

Multiple sclerosis and autism bear significant neuropathologic similarities such as reduced myelination and elevated anti-myelin basic protein (anti-MBP). However, the ages at which multiple sclerosis and autism are first diagnostically apparent are typically quite different. Therefore, we are proposing a single therapeutic model that may be beneficial for both conditions using glatiramer to modulate the antibody to MBP, and insulin-like growth factor to stimulate the myelination of neurons. This new approach needs to be evaluated in future clinical studies.

Background and Relevant Associations

When comparing autism spectrum disorder (ASD) and multiple sclerosis (MS), similarities and differences can be noted [1,2]. Both are maladies of myelination. Both display elevated titers of myelin basic protein antibodies. However, the pathology of autism begins in infancy and mainly involves reduced neo-neurogenesis of the central nervous system (CNS). In contradistinction, the neuro-muscular manifestations of MS most often first appear in adulthood.

Studies of brain biopsies from cases of ASD have identified symmetrically reduced myelination of newly formed neurons, whereas myelin sheaths in unaffected individuals are uniformly thicker [3]. The neurologic symptoms of the disease are thought to be related to altered synaptic function in the young central nervous system. Loss of Purkinje cell cerebellar tracts has been detected in autistic children by MRI tractography [4]. In contradistinction, demyelination of established neurons in MS is patchy and irregular. This presentation is apparently a consequence of specific local autoimmune degradation of established myelin components; mature nerves exhibit diminished velocity of impulse transmission and, subsequently, muscle degeneration occurs [5].

Because of the age of onset, brain dysconnectivity could account for the behavioral problems encountered with childhood ASD but not with MS, which typically appears first in adults [6]. Besides ASD, dysconnectivity has been implicated in other neuropathies such as Friedreich's ataxia and schizophrenia [7,8]. In the latter,

miswiring is related to aberrant synaptic plasticity due to abnormal receptor function. Significant dysconnectivity is identified in treatment-resistant schizophrenics when compared with their healthy siblings [9].

The 70-amino acid polypeptide, insulin-like growth factor-I (IGF), is thought to be a key parameter in the genesis of ASD because of:

- Reduced IGF in the cerebrospinal fluid (CSF) of young autistic children [10];
- IGF activation of myelination via oligodendrocytes [11];
- Laboratory mice altered to exhibit autism-like behavior improve when given IGF [12];
- Milk, especially from human sources, contains significant levels of IGF. Groups of babies breast-fed (in contradistinction to formula) for extended periods later display a lower overall incidence of autism than the general population [13];
- Very-small-for-gestational-age (VSGA), very premature newborn commonly have lower levels of IGF in their

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