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A Fatty Solution to Autism

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Abstract

Our previous studies led to the suggestion that in those with autism spectrum disorder (ASD) may have deficiency of arachidonic acid (AA) and docosahexaenoic acid (DHA) and an imbalance in the production and action of pro- and anti-inflammatory metabolites of these fatty acids. This could be responsible for increased amounts of free radicals and cytokines observed in ASD. Since perinatal inflammatory events could enhance the risk of ASD, it is suggested that supplementation of AA and DHA from the second trimester of pregnancy till the first 5 years of childhood may decrease or abrogate ASD. This is especially so as AA and DHA and their metabolites lipoxin A4, resolving, protections and maresins can suppress the production of excess of IL-6 and TNF- α , free radicals and facilitate wound healing and inhibit inappropriate inflammation and thus, protect neurons from the cytotoxic actions of both endogenous and exogenous insults.

It is noteworthy that AA and DHA enhance neuronal generation, synapse formation, neurotransmission, and memory. TNF- α despite being a pro-inflammatory molecule is needed for strengthening the synapse and thus has a role in proper neuronal function. It is envisaged that AA, DHA and TNF- α will interact with each other to ensure appropriate synapse formation. Both AA and DHA are needed for syntax in action and thus regulate exocytosis of various neurotransmitters. This implies that AA and DHA regulate the secretion of various neurotransmitters. As essential components of cell membrane both AA and DHA regulate fluidity of neuronal cell membranes and the expression and affinity of various proteins/growth factors and neurotransmitters to their receptors. These and other actions of AA and DHA suggest that they have an important fundamental role in ASD. This may explain the high AA and DHA content of human brain and their high concentrations in human breast milk explaining as to why breast-fed children have low incidence of ASD.