Essential Fatty Acids role in neurochemistry of the Central Nervous System
A guide to remedial repletion therapy.

In the previous article "Essential Fatty Acids role on the Neurochemistry of the Brain", an overview of the role of EFAs was considered, here the remedial repletion of EFA is discussed.

The previous article proposed the role played by lipids in the metabolism of Very Long Chain Fatty Acids, VLCFA.
At this point, we should consider the role of a part of the cell structure which is present in cells and whose sole purpose is to rid the cell of toxic substances. This component is the Peroxisome.
VLCFAs are shortened by oxidation to long chain fatty acids in Peroxisomes and further oxidised in mitochondria, converted to Acetyl-CoA, the enzyme that catalyzes the first step in oxidation. The build up of VLCFA leads to abnormal neuroanatomy primarily in the hippocampus and amygdala giving rise to development delay, identified by Bauman and Kemper in the Neuroanatomy of Autistic Brains.
The accumulation of VLCFA may constitute a minor part of overall fatty acids, according to Kane, ***but peroxisome deficiency disorders are deleterious to the brain and CNS.***

The peroxisome is also responsible for the formation inside the cell of plasmalogens (in simple terms another lipid) which is the most abundant lipid in myelin. A deficiency of plasmalogens causes profound abnormalities in the myelination of nerve cells, which is one of the reasons that many peroximonal disorders lead to neurological dysfunction's.
Peroxisomes are present in virtually all cells and are most prevalent in the liver and kidney cells where the peroxisomes detoxify various toxic substances that enter the blood.
Deficiency of the formation of plasmalogens can also cause severe brain disorders, leading to neurological dysfunction.

According to Kane, red cell lipid analysis of children with ASD have revealed, elevation of VLCFAs, depressed myelination markers, suppressed prostaglandin synthesis, omega 6 depletion, poor cell membrane integrity, and complex autoimmune derangement.
The autoimmune presentation of ASD may ***initially respond negatively*** to omega 3 marine oils, due to the competitive inhibition of omega 3s to omega 6s and poor cell membrane integrity. On this principle, omega 6s, the precursor PG1 (prostaglandin one) must be repleted and stabilized before omega 3 supplementation begins, usually 6-8 weeks.
The synthesis of prostaglandins is an oxidative process, introducing antioxidants, or the incorrect sequence of EFAs may have a negative effect on the ASD presentation.