Two papers relevant for Namenda / Glutamate. The second is a formulated trial.

Prostaglandins Leukot Essent Fatty Acids. 2009
Jul 14. [Epub ahead of print] Links

Novel plasma phospholipid biomarkers of autism: Mitochondrial dysfunction as a putative causative mechanism.

Pastural E, Ritchie S, Lu Y, Jin W, Kavianpour A, Khine Su-Myat K, Heath D, Wood PL, Fisk M, Goodenowe DB. Phenomenome Discoveries Inc., 204-407 Downey Road, Saskatoon, Saskatchewan, Canada S7N 4L8.

Autism is a neurological disorder that manifests as noticeable behavioral and developmental abnormalities predominantly in males between the ages of 2 and 10. Although the genetics, biochemistry and neuropathology of this disease have been extensively studied, underlying causal factors to this disease have remained elusive. Using a longitudinal trial design in which three plasma samples were collected from 15 autistic and 12 non-autistic age-matched controls over the course of 1 year, universal and unambiguous alterations in lipid metabolism were observed. Biomarkers of fatty acid elongation and desaturation (poly-unsaturated long chain fatty acids (PUFA) and/or saturated very long chain fatty acids (VLCFA)-containing ethanolamine phospholipids) were statistically elevated in all autistic subjects. In all 8 of the affected/non-affected sibling pairs, the affected sibling had higher levels of these biomarkers than the unaffected sibling. Exposure of neurons, astrocytes and hepatocytes in vitro to elevated extracellular glutamate levels resulted in lipid biomarker changes indistinguishable from those observed in autistic subjects. Glutamate stress also resulted in in vitro decreased levels of reduced glutathione (GSH), methionine and cysteine, in a similar way to the decreases we observed in autism plasma. Impaired mitochondrial fatty acid oxidation, elevated plasma VLCFAs, and glutamate toxicity as putative causal factors in the biochemistry, neuropathology, and gender bias in autism are discussed.

http://www.ncbi.nlm.nih.gov/pubmed/19608392

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J Child Neurol. 2007 May;22(5):574-9.

Memantine as adjunctive therapy in children diagnosed with autistic spectrum disorders: an observation of initial clinical response and maintenance tolerability.

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Autism and Pervasive Developmental Disorder Not Otherwise Specified are common developmental problems often seen by child neurologists. There are currently no cures for these lifelong and socially impairing conditions that affect core domains of human behavior such as language, social interaction, and social awareness. The etiology may be multifactorial and may include autoimmune, genetic, neuroanatomic, and possibly excessive glutaminergic mechanisms. Because memantine is a moderate affinity antagonist of the N-methylD-aspartic acid (NMDA) glutamate receptor, this drug was hypothesized to potentially modulate learning, block excessive glutamate effects that can include neuroinflammatory activity, and influence neuroglial activity in autism and Pervasive Developmental Disorder Not Otherwise Specified. Open-label add-on therapy was offered to 151 patients with prior diagnoses of autism or Pervasive Developmental Disorder Not Otherwise Specified over a 21-month period. To generate a clinician-derived Clinical Global Impression Improvement score for language, behavior, and self-stimulatory behaviors, the primary author observed the subjects and questioned their caretakers within 4 to 8 weeks of the initiation of therapy. Chronic maintenance therapy with the drug was continued if there were no negative side effects. Results showed significant improvements in open-label use for language function, social behavior, and self-stimulatory behaviors, although self-stimulatory behaviors comparatively improved to a lesser degree. Chronic use so far appears to have no serious side effects.
http://www.ncbi.nlm.nih.gov/pubmed/17690064