Chapter 3: Prenatal Fluoride and Autism

**Adhesion Molecules, Autism, and Fluoride**
Autism involves early brain overgrowth and dysfunction, an excess of neurons in the prefrontal cortex caused by a prenatal disruption of developing brain architecture as early as the second trimester. Research by Lahiri et al. (2013) suggests that brain enlargement in autism is likely due to cell adhesion dysfunction.

Neural cell adhesion molecules (NCAM) are widely expressed in the nervous system, where they are involved in axon growth and guidance – fundamental processes that underlie formation of the synaptic connections crucial to brain development. Significantly lower serum levels of several types of adhesion molecules, including NCAM, have been found in ASD. Neural pathways involving synaptic cell adhesion are disrupted in some people with autism, including alterations in the structure and expression of NCAM.

Fluoride exposure has been shown to cause a dose-dependent decrease in NCAM expression levels in rat hippocampal neurons. In particular, the NCAM-140 protein expression level was significantly lower in response to the lowest dose of fluoride used. NCAM-140 is found in migrating growth cones that are crucial to the formation of synaptic connections.

**Fluoride Adversely Affects Synaptic Development**
Autism and Alzheimer’s are increasingly being linked to defects in the organization and number of synapses, the tens of trillions of tiny yet complex structures that link neurons so they can communicate with each other. A molecule that helps create and maintain the scaffolding around which a synapse is built is postsynaptic density protein-95 (PSD-95). Neuronal synapses with less PSD-95 are likely to be weakened or lost.

PSD-95 is concentrated at synapses where it regulates adhesion and enhances maturation of the presynaptic terminal. Research demonstrates that PSD-95 orchestrates synaptic development and plays an important role in synapse stabilization and plasticity.

In rats that drank water with added fluoride for several months, the fluidity of brain synaptic membranes and the expression level of PSD-95 decreased in a dose-dependent manner. Rats anesthetized for 4 hours with 2.5% sevoflurane, a fluoride-based anesthetic, showed long-term deficits in hippocampal function and decreased hippocampal PSD-95 expression.
References


