Autism Insights: A New Hope Part I



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New insights into physical, biochemical, and neurological differences in children with Autism Spectrum Disorders provides hope for new interventions and lifestyle changes that may reduce symptoms and improve attention, socialization, and attachment development.

Autism Spectrum Disorder [ASD] is associated with disorders of attachment as well as anomalies in neurocognitive, neuroendocrine, and neurobiological processes (Sivaratnam, Newman, Tonge, & Rinehart, 2015; Teague, Newman, Tonge, et al., 2018). Children with ASD are at a greater risk for interruption of attachment development, motor dysfunction, and attenuated socioemotional development (Marko et al., 2015; Sivaratnam et al., 2015; Teague et al., 2018). A bidirectional relationship exists between attachment development of children with ASD and the quality of parenting and mental health caregiving environment (Teague et al., 2018). The findings of Teague et al. support the exigence for early and vigorous intervention of attachment development in the population of children with ASD.

Among the varied anomalies identified in children who later develop ASD is an increase in cerebrospinal fluid [CSF] in the sub-arachnoid space from 6 to 24 months of age (Shen et al., 2017). Severity of ASD-related symptomology was directly related to increased extra-axial volume of CSF (Shen et al., 2017). The increase in CSF is suspected to be a contributory factor to brain enlargement and increase in head circumference of infants who later develop ASD (Shen et al., 2013). Future investigation is required to determine whether the increased CSF volume is a marker or contributing factor in the later development of ASD (Shen et al., 2017).

Neurons in the fusiform gyrus of children with ASD are smaller and fewer in number (van Kooten et al., 2008). Meanwhile, neuroplastic changes in morphology of the fusiform gyrus were documented following a face-evoked activity with individuals with ASD (Perlman, Hudac, Pegors, Minshew, & Pelphrey, 2011). As the fusiform gyrus is the brain structure associated with facial perception and memory (Kanwisher & Yovel, 2006), intervention to increase size and functionality of the fusiform gyrus may facilitate more effective facial recognition capacities in children with ASD; which may in turn mediate improved social functioning.

Investigation of changes in the cerebellum of children with ASD revealed decreased volume of the anterior cerebellum where it extends into lobules VI and VIII as compared to typical children (Marko et al., 2015). These cerebellar differences correlate with patterns of proprioceptive and visual errors that are associated with anomalies in motor learning, providing an explanation of the linkage between brain and behavior anomalies in children with ASD (Marko et al., 2015). Autoimmune disorder [AD] is another suspected linkage to ASD (Edmiston, Ashwood, & Van de Water, 2017; Wu et al., 2015). Investigation of families with history of AD revealed a significantly higher risk of ASD of offspring (Wu et al., 2015). Risk of ASD proved to be associated with various forms of AD including rheumatoid arthritis,

hypothyroidism, and diabetes (Wu et al., 2015). High levels of some antineuronal antibodies in mothers is positively associated with increased risk of ASD of offspring (Ali, Khalaf, Al-Asadi, & Abed, 2016).

In addition to ASD, brain inflammation plays a role in the pathogenesis of some neuropsychiatric disorders (Theoharides, Stewart, Panagiotidou, & Melamed, 2016).

Theoharides, Asadi, Panagiotidou, and Weng (2013) posited that mitochondrial components secreted by mast cells during degranulation may be the *missing link* between autoimmunity and ASD. Due to the close proximity of some mast cells [MC] to microglia and neurons, especially in the median eminence, hypothalamus, thalamus, and leptomeninges, MC release of neurotoxic and inflammatory mediators can interfere with the blood-brain barrier [BBB], leading to stimulation of microglia and focal inflammation (Theoharides et al., 2018). Under stress, the hypothalamus secretes corticotropin-releasing factor [CRF], which acts synergistically with neurotensin [NT] to stimulate MCs; as the MC's respond to the continued stimulation, vascular permeability is increased (Theoharides et al., 2018). The impact of these and other responses of activated MCs may be ASD, headache, and brain fog (Theoharides et al., 2018). Activation of mast cells in Type 2 Diabetes mediates inflammation (Conti, Ronconi, Kritas, Caraffa, & Theoharides, 2018).

Further investigation of the various associated and concomitant conditions and anomalous morphological changes in children with ASD is required to identify a concise therapeutic intervention. In the meantime, a significant number of studies point to the connection between ASD and inflammatory processes. While no single inflammatory process has been identified as the primary aggravating or causative factor, a number of logical connections have been made between ASD and such pro-inflammatory conditions as diabetes, mast cell activation

syndrome, maternal autoimmunity, and hypo-thyroiditis. Practical strategies to promote overall health of the individual with ASD may be trialed and documented to identify mitigating factors or strategies that attenuate some of the symptomology associated with the autism condition.

To date, we have identified ten promising strategies that have shown promising results in some children with Autism Spectrum Disorder. The great news is that each of these strategies is developmentally appropriate and health supportive. So even if the strategy does not improve Autism-specific symptoms, your child is likely to benefit from overall increased health, learning, and development. Our new PNE Model helps identify the best next step to try for your child; check back for the next paper in this Autism Hope series.

About the Author



Darleen Claire Wodzenski, MS ESE, MA CMHC, DD, QPPE, PhD Candidate has dedicated her life to working with children with Attachment Disorders, Autism Spectrum Disorders, and Emotional and Behavioral Disorders. As a Clinical Mental Health Counselor, Special Educator, and Non-Clinical Psychoneuroeducational Psychologist, Dr. Darleen shares valuable information to promote health, learning, growth, and development of children – no matter the circumstances.

A national speaker and author, her books help parents and children navigate challenges with learning, metal health, behavior, development, and attachment. Check out some of these titles on Amazon.com or visit OrchardHumanServices.org to access the information library:

Marmalade Jam

Part I of Therapeutic Children's Series

Juno's Butter Knife [to be released soon]

Part II of Therapeutic Children's Series

Dead Children Can't Read

Classrooms of Compassion

Raising Social Children

A Guide for Parents Who Suspect Their Child May Have A Delay in Social Development

Self-Harm Guide

A Guide for Youth, Families, Educators, Human Services Workers

References

- Ali, N.H., Khalaf, S.K., Al-Asadi, J.N., & Abed, A.H. (2016). Maternal antineuronal antibodies and risk of childhood autism spectrum disorders: A case-control study. *Journal of the Chinese Medical Association*, 79(12), 661-664. https://doi.org/10.1016/j.jcma.2016.08.003
- Conti, P., Ronconi, G., Kritas, S.K., Caraffa, A., & Theoharides, T.C. (201). Activated mast cells mediate low-grade inflammation in type 2 diabetes: Interleukin-37 could be beneficial.

 Canadian Journal of Diabetes, 2(5), 568-573. (Accession No. 184932)
- Edmiston, E., Ashwood, P., & Van de Water, J. (2017). Autoimmunity, autoantibodies, and autism spectrum disorder. *Biological Psychiatry*, 8(5), 383-390. https://doi.org/10.1016/j.biopsych.2016.08.031
- Kanwisher, N, & Yovel, G. (2006). The fusiform face area: A cortical region specialized for the perception of faces. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 361, 21092128. doi:10.1098/rstb.2006.1934
- Marko, M.K., et al. (2015). Behavioral and neural basis of anomalous motor learning in children with autism. *Brain*, *138*(3), 784-797. doi:10.1093/brain/awu394
- Perlman, S. B., Hudac, C. M., Pegors, T., Minshew, N. J., & Pelphrey, K. A. (2010).

 Experimental manipulation of face-evoked activity in the fusiform gyrus of individuals with autism. *Social Neuroscience*, 6(1), 22-30.
- Shen, M.D., et al. (2013). Early brain enlargement and elevated extra-axial fluid in infants who develop autism spectrum disorder. *Brain*, *136*, 2825-2835. doi:10.1093/brain/awt166

- Sivaratnam, C.S., Newman, L.K., Tonge, B.J., & Rinehart, N.J. (2015). Attachment and emotion processing in children with autism spectrum disorders: neurobiological, neuroendocrine, and neurocognitive considerations. *Review Journal of Autism and Developmental Disorders*, 2(2), 222-242. https://doi.org/10.1007/s40489-015-0048-7
- Teague, S.J., Newman, L.K., Tonge, B.J., et al. (2018). Caregiver mental health, parenting practices, and perceptions of child attachment in children with Autism Spectrum Disorder. *Journal of Autism and Developmental Disorders*, 48(80), 2642-2652. https://doi.org/10.1007/s10803-018-3517-x
- Theoharides, T.C., Asadi, S., Panagiotidou, S., & Weng, Z. (2013). The "missing link" in autoimmunity and autism: Extracellular mitochondrial components secreted from activated live mast cells. *Autoimmunity Reviews*, 12(12), 1136-1142. doi:10.1016/j.autrev.2013.06.018
- Theoharides, T.C., Stewart, J.M., Panagiotidou, S., & Melamed, I. (2016). Mast cells, brain inflammation and autism. *European Journal of Pharmacology*, 778, 96-102. doi: 10.1016/j.ejphar.2015.03.086
- Van Kooten, I.A., et al. (2008). Neurons in the fusiform gyrus are fewer and smaller in autism.

 *Brain, 131(4), 987-999. doi:10.1093/brain/awn033
- Wu, S., Ding, Y., Wu, F., Li, R., Xie, G., Hou, J., & Mao, P. (2015). Family history of autoimmune diseases is associated with an increased risk of autism in children: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, 55, 322-332. https://doi.org/10.1016/j.neubiorev.2015.05.004