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Role of endocrine disturbing chemicals in development of autism spectrum disorders**Omar Bagasra**

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Autism Spectrum Disorder (ASD) is a set of complex developmental disorders whose etiology is unknown. Although the symptoms may vary from person to person, they include impairment or loss of speech, lack of empathy and social interaction deficiency. The cases of ASD have continued to increase drastically each year, with the CDC estimating 1:45 children diagnosed, from 1 in 10,000 40 years ago. It is believed that the ASD is caused by a combination of genetic and environmental factors, but recent studies suggest that epigenetic factors as well as exposure to endocrine disturbing environmental chemicals, to which expecting mothers are exposed on a daily bases, may play a critical role in its pathogenesis. Although there are no biomarkers for the disease, low levels of Oxytocin (OXY) and Arginine Vasopressin (AVP) have been reported. These neuropeptides play a critical role in neurodevelopment of social interaction. Social interaction deficiencies are a principal sign of ASD in children. OXY, is involved in social recognition, pair bonding and anxiety, as well as being linked to autism. Numerous studies have shown that children with autism have plasma levels of OXY and AVP that are significantly lower than average. The importance for normal OXY and OXY receptor function in males may explain how hormonal malfunction leads to ASD male bias. Also, mothers of ASD children have lower levels of OXY and AVP and, in typical children, lower concentrations of OXY in plasma are associated with lower social and cognitive functioning. There is an inexplicable bias toward males in classical autism by a ratio of ~4:1, and ~10:1 in Asperger's Syndrome (AS). The clinical picture is very heterogeneous and the etiology is unknown. The heritability is high in ASD but no individual gene variants exerting a major impact on susceptibility have as yet been identified. The mechanism for gender bias in autism is unknown although several hypotheses have been advanced including: (1) Epigenetic mechanisms 'the extreme male brain' hypothesis of Baron-Cohen which postulates that elevated fetal testosterone is a risk factor for ASD, (2) genetic mechanisms which involves X or Y chromosome inactivation and (3) recently, Hu, et al. have shown that retinoic acid-Related Orphan Receptor Alpha (RORA) is reduced in the brain and lymphoblastoid cell lines of multiple cohorts of individuals with ASD. This gene targets CYP19A1 (aromatase), in a gender-dependent manner that can also lead to elevated testosterone (or male hormones-like chemicals) levels, a proposed risk factor for autism. To date, none of these hypotheses have been either proven or disproven. Given the high clinical heterogeneity of ASD, it is possible that each of these mechanisms for gender bias may apply to specific cohorts of individuals with ASD. We will present data on the neuro-modifying effects of several endocrine disturbing chemicals on developing human brain neurons and their effects at morphologic, immunologic and at molecular levels.

Biography

Omar Bagasra has completed his PhD from University of Louisville, Kentucky, USA and his MD from UACJ, Mexico. He has completed his Post-doctorate at Union University, Albany, New York and Residency and Fellowships at Hahnemann University, Temple University, Schools of Medicine. He is the Founding Director of South Carolina Center for Biotechnology at Claflin University, South Carolina, USA. He has published more than 140 papers in reputed journals and published over 10 books. His recent book describes the role of environmental factors in autism. He has been serving as Editorial Board Member of over 15 journals.

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