Autism: Understanding Autism Spectrum Disorder from a Neuroscience Perspective

Allison Cummings

Antioch College

The point of this paper is to evaluating and understanding Autism Spectrum Disorder from a Neuroscience Perspective. The exploration will consist of an introduction to Autism Spectrum Disorder, followed by what a neuroscience perspective of Autism encompasses, then lead into atypical developmental features and basic brain chemistry, a genetic background component, and the role of neurotransmitters. The basic question will look at how the Human brain and physiology shape and control behavior differently in an individual with Autism Spectrum Disorder.

Brock (2011) classifies Autism Spectrum Disorder as a, “pervasive developmental disorder, affecting multiple aspects of cognition and behavior” (p. 645). Autism Spectrum disorders consist of deficiencies in three functional main areas: social exchange and interactions, communication and language skills and nonfunctional repetitive behaviors as well as egocentric interests (Eigsti, 2003, p. 206). Autism Spectrum disorder can be connected with intellectual disability, complications in motor coordination and attention as well as physical health issues such as sleep and gastrointestinal disturbances (“What is Autism,” 2016). Autism origin occurs in early brain development, but the typical symptoms of Autism appear around age two or three (“What is Autism,” 2016).

With the publication of the DSM-5 diagnostic manual, all Autism disorders were combined into one diagnosis of Autism Spectrum Disorder (“What is Autism,” 2016). Prior to the DSM-5, subtypes of Autistic disorders were utilized. These included, Childhood Disintegrative Disorder, Autistic Disorder, Asperger Syndrome and other Pervasive Developmental Disorders (“What is Autism,” 2016). All the disorders with the exception of Childhood Disintegrative Disorder are categorized by communicative and social deficiencies that occur form the start (Eigsti, 2003, p. 206). While Eigsti (2003) proposes that, “Disintegrative Disorder encompass a period of apparently typical growth and development followed by a signiﬁcant loss of skills” (p. 206).

Every individual with Autism Spectrum Disorder has unique developmental features and many of them have extraordinary abilities in music, academic and visual skills (“What is Autism,” 2016). Forty percent of Autistic individuals have average or above intellectual capabilities while others may have a significant disability and are unable to live independently (“What is Autism,” 2016). Autism Spectrum disorder allows for vast heterogeneity by being based on the definitive of observable behavior (Brock, 2011, p. 645). Stated by Brock (2011) this heterogeneity occurs because, “Some form of social impairment is required for an autism diagnosis, entailing that social dysfunction is a universal amongst diagnosed individuals” (p. 646). This suggest that individuals on the Autism Spectrum have similar fundamental neurobiological mechanisms (Brock, 2011, p. 646).

“What is Autism” (2016) articulates that, “Autism Spectrum Disorder affects over three million individuals in the United States and tens of millions worldwide”. Research statistics show that occurrence rates of Autism have increased ten to seventeen percent yearly in recent history (“What is Autism,” 2016). Environmental influences and improved diagnosis rates are assumed to be the cause of the prevalence increase (“What is Autism,” 2016).

 Neuroscience deals with the Anatomy and physiology of the brain and nervous system. When looking at Autism from a neuroscience perspective, it includes the incorporation of origins, development, and the intricacies of Autism (Berger, 2008, p. 792). Berger (2008) specifically states that neuroscience looks at the “complexity of the phenomena and the developmental ramiﬁcations of the autism disorders” (p. 792).

 Autism being a heterogeneous disorder is likely to have multiple etiologies, but Eigsti (2003) said that there are, “clear effects of neurodevelopmental differences that will likely point to the etiology” (p. 207). Current research on the structure of the brain has elicited awareness of anatomical abnormalities as well as early developmental changes in growth and pruning of neural tissue (Eigsti, 2003, p. 206). Cerebellar and brain volume abnormalities have both been confirmed by this research (Eigsti, 2003, p. 208-210).

Magnetic resonance imaging (MRI) results have shown an increase in temporal, parietal, and occipital lobes in individuals with Autism Spectrum Disorder (Eigsti, 2003, p. 210). Egstil (2003) suggested, “the frontal lobes may be the most abnormal in volume” (p. 210). This indicates an overall brain volume enlargement compared to a non-Autistic individual. Data also has been provided to demonstrate cerebellar nuclei abnormalities that are atypical in Autism (Eigsti, 2003, p. 208). Also, declared by Carré (2015), “Abnormal functioning of primary brain systems that express and modulate basic emotional drives are increasingly considered to underlie mental disorders including autism spectrum disorders” (p. 3351).

Neurodevelopmental disconnections also have been assumed to cause behavioral alterations in Autistic individuals (Alaerts, 2014, p. 1592). Alaerts (2014) provided, “converging experimental evidence that underconnectivity between key areas in the temporal and frontal-parietal lobes underlie the social deficits in emotion recognition associated with Autism Spectrum Disorder (p.1598). A strong link between neural abnormalities and emotion perception deficits in Autistic Individuals resulted from this study (Alaerts, 2014, p. 1598).

A genetic link has also been suggested as a cause of Autism Spectrum Disorder. Research has identified mutations and rare gene changes associated with Autism (“What is Autism,” 2016). However, genes don’t seem to cause Autism alone, but when combined with environmental factors seem to influence early brain development (“What is Autism,” 2016). Genetic risk factors before and during birth with prenatal teratogens or hazards effect an infant’s development and may lead to Autism (“What is Autism,” 2016). Possible etiologies of Autism from a genetic component according to Eigsti (2003) are, “Differential gene expression from chromosomes of different parental origin” as well as “Genetic liability combined with an environmental factor” (p. 209). For example, prenatal exposure to the toxin thalidomide may attribute to Autism (Eigsti, 2003, p. 209).

The cause of autism is still under debate, but research suggests variations in certain neurotransmitter in the brain and spinal fluid could attribute a crucial role in Autism Spectrum Disorder (Drenthen, 2016, p. 25644). Serotonin is perceived to be one of the neurotransmitters that attributes to Autism (Kaplan, 2016, p. 6631). Serotonin usually thought as an influence of sleep, mood and certain sensory perceptions lie hunger and regulating body temperature (Kaplan, 2016, p. 6631). However, serotonin also affects the rate at which hormones are released (Kaplan, 2016, p. 6632). According to Kaplan (2016), “Individuals with Autism are known to have increased levels of serotonin in their blood” (p. 6632). In Kaplan (2016) study the objective was to “To determine associations regarding prenatal selective serotonin reuptake inhibitor use and the risk for autism spectrum disorders in children” (p. 6638).

Autistic individuals not only produce, but absorb serotonin differently and abnormalities have been found in serotonin synthesis in parts of the brain and overall the serotonin levels of the whole brain ((Kaplan, 2016, p. 6639). Results of Kaplan’s (2016) study, “demonstrated a significantly increased risk of Autism Spectrum Disorder in the children whose mothers were prenatally exposed to selective serotonin reuptake inhibitor during different exposure time windows” (p. 6642). Based on this research it is predicted that medication that mirrors serotonin may be helpful in targeting characteristics of Autism (Kaplan, 2016, p. 6643).

However, more neurotransmitter associations have been suggested to also attribute to Autism Spectrum Disorder. The study by Drenthen (2016) aimed to, “assess Glu and GABA neurotransmitter concentrations in HFA” (p. 25645). Which means they were looking at glutamate and gamma-aminobutyric acid imbalanced levels in high-functioning Autistic children. The results of their study indicates that these neurotransmitters imbalanced levels of excitation and inhibition are linked with high functioning Autistic children (Drenthen, 2016, p.25649).

According to Drenthen (2016), “Proton magnetic resonance spectroscopy can provide valuable information about abnormal brain metabolism and neurotransmitter concentrations” (p. 25644). However, very few studies have been conducted utilizing this method as well as looking for neurotransmitters relation to Autism in general. Also, so far studies have purely focused on the high functioning autism population and limits its finding to intellectual impairments instead of Autism (Drenthen, 2016, p. 25645).

In summary, Autism Spectrum Disorder is an umbrella term for a group of persistent developmental disorders that result in social and communication impairments with repetitive behaviors. Individuals with Autism Spectrum Disorder show a significant difference in brain anatomy and physiology compared to non-Autistic individuals and there is no universal cause of Autism. A variety of theories to what exactly causes Autism remains elusive. Some current theories focus on the levels of certain neurotransmitters in the brain and spinal cord as well as genetic components with environmental hazards included.

 It is important to understand and spread education of Autism Spectrum Disorder for many reasons. One reason is because Children on the Autism Spectrum do not follow typical patterns of child development. So, it is imperative that we learn these developmental differences in order to provide accurate, early diagnosis. The earlier diagnosed the better outcome of treatment interventions. Autism is found worldwide, it ignores racial, economic, and ethnic backgrounds; making it possible for it to develop anywhere. So, by studying and educating ourselves about Autism, we can figure out the best course prevention and treatment options.

References

Alaerts, K., Woolley, D. G., Steyaert, J., Di Martino, A., Swinnen, S. P., & Wenderoth, N. (2014). Underconnectivity of the superior temporal sulcus predicts emotion recognition deficits in autism. Social Cognitive & Affective Neuroscience, 9(10), 1589-1600.

Berger, M. (2008). Review of Understanding autism: From basic neuroscience to treatment. Journal Of Child Psychology And Psychiatry, 49(7), 792. doi:10.1111/j.1469-7610.2007.01767.x

Brock, J. (2011). Commentary: Complementary approaches to the developmental cognitive neuroscience of autism—Reflections on Pelphrey et al. (2011). Journal Of Child Psychology And Psychiatry, 52(6), 645-646. doi:10.1111/j.1469-7610.2011.02414.x

Carré, A., Chevallier, C., Robel, L., Barry, C., Maria, A., Pouga, L., & ... Berthoz, S. (2015). Tracking Social Motivation Systems Deficits: The Affective Neuroscience View of Autism. Journal Of Autism & Developmental Disorders, 45(10), 3351-3363.

Drenthen, G. S., Barendse, E. M., Aldenkamp, A. P., van Veenendaal, T. M., Puts, N. A., Edden, R. A., & ... Jansen, J. F. (2016). Altered neurotransmitter metabolism in adolescents with high-functioning autism. Psychiatry Research: Neuroimaging Section, 25644-49. doi:10.1016/j.pscychresns.2016.09.007

Eigsti, I., & Shapiro, T. (2003). A systems neuroscience approach to autism: Biological, cognitive, and clinical perspectives. Mental Retardation & Developmental Disabilities Research Reviews, 9(3), 206-216.

Kaplan, Y. C., Keskin-Arslan, E., Acar, S., & Sozmen, K. (2016). Prenatal selective serotonin reuptake inhibitor use and the risk of autism spectrum disorder in children: A systematic review and meta-analysis. Reproductive Toxicology, 6631-43. doi:10.1016/j.reprotox.2016.09.013

What Is Autism? What is Autism Spectrum Disorder? (2016). Retrieved November 29, 2016, from https://www.autismspeaks.org/