

A Short Note on Neurodevelopmental Disorders Include Autism

Setsuki Congo*

Department of Psychiatry, Nairobi University, Kenya

Abstract

Neurodevelopmental disorders include autism spectrum disorders (ASDs). Although the exact cause of ASD is unknown, a number of genetic and non-genetic risk factors have been identified that, either on their own or in combination, may contribute to the disorder's onset. There are currently no diagnostic biomarkers, and the diagnosis of ASD is based on typical characteristics such as impaired social communication and interaction and repetitive behaviors. These behaviors have been suggested to be caused by a number of pathomechanisms, including synaptic defects and changes in brain development and function. The clinical phenotype and severity may also be altered by processes that occur outside the central nervous system. This chapter provides a synopsis of the clinical characteristics of ASD, draws attention to significant genetic and non-genetic risk factors for ASD, and introduces the most recent understanding of the pathological processes that take place both inside and outside the brain.

Keywords: Neurodevelopmental disorders; Autism spectrum disorders

Introduction

A multidimensional and early treatment approach is required for personality disorders in their early stages and preliminary forms. In addition, Robert F. Krueger's review of their research indicates that some children and adolescents do experience clinically significant syndromes that resemble adult personality disorders, and that these syndromes have meaningful correlates and are consequential. Personality development disorder is considered to be a childhood risk factor or early stage of a later personality disorder in adulthood. The adult personality disorder constructs from Axis II of the Diagnostic and Statistical Manual have served as the framework for much of this research. As a result, the first risk they identified at the beginning of their review is less likely to occur: The PD concept is not simply being avoided by researchers and clinicians working with youth. However, the second risk they mentioned could occur to them: under-recognition of these syndromes' developmental context. Consequently, PD constructs are probabilistic predictors despite their historical continuity; Youths with PD symptoms do not always progress into adult cases.

The triad of speech impairments, social interaction impairments, and the presence of restricted or repetitive behaviors are the clinical characteristics that have historically been associated with ASD. However, ASD is also linked to a wide range of psychological and physiological co-morbidities.

ASD is typically diagnosed by the age of three in most cases, when symptoms first appear in childhood. A child's inability to respond to their name and difficulty maintaining eye contact are early signs of ASD. The majority of ASD symptoms persist into adulthood, particularly in terms of cognitive ability and social functioning. As the individual reaches adolescence and adulthood, symptoms, particularly communication abilities, may improve over time. Over time, intellectual functioning and IQ tend to stay the same. Although some aspects of ASD remain relatively stable, a person's quality of life can be enhanced through other accommodations and interventions or the development of a robust social support network.

The clinical diagnosis of ASD is based on the presence of key characteristics like delayed social development and recurrent interests and behaviors. ASD frequently results in delays in speech development, learning disabilities, and difficulties with social interaction. In people with ASD, executive function and organizational skills suffer greatly.

They rarely take the initiative to engage in social or other interactions with their surroundings. They likewise frequently experience the ill effects of hardships with handling improvements and arranging out the means of an action and may show ritualized or unbending ways of behaving. As a result, ASD is characterized by difficulties initiating independent behaviors and tasks. The capacity to apply one's skills to a variety of contexts is another area that is frequently impacted by ASD. People often have trouble adjusting to new people, things, and environments because of probably very specific stimuli [1-5].

Discussion

Repetitive or restricted behaviors are the third fundamental characteristic of ASD. These can be verbal behaviors like saying the same thing over and over again or stereotypical actions like shaking one's hands or rocking one's body. Additionally, individuals with ASD typically have restricted or specific interests. For instance, a strong interest in a particular subject, sensory fixations on a specific object, or adherence to a particular routine or approach to a task.

Aggression, hyperactivity, impulsivity, and the occurrence of comorbidities like anxiety and depression are secondary symptoms of ASD. The diversity of ASD's clinical features is a major feature. Numerous psychological and physiological comorbidities, in addition to a wide range of symptoms, may be present. The variety of these symptoms and comorbidities is depicted. Anxiety, intellectual disability, and attention-deficit hyperactivity disorder (ADHD) are examples of psychological comorbidities. ASD and ADHD frequently occur together, and the two conditions share numerous neurological and behavioral similarities. For instance, both ASD and ADHD have social deficits and hyperactivity. Between 30% and 50% of people with ASD also exhibit ADHD symptoms. This is taken into consideration in the fifth edition of the Diagnostic and Statistical Manual of Mental

*Corresponding author: Setsuki Congo, Department of Psychiatry, Nairobi University, Kenya, E-mail: ongosetski@edu.ke

Received: 04-Jan-2023, Manuscript No: jcalb-23-86468; **Editor assigned:** 06-Jan-2023, Pre-QC No: jcalb-23-86468 (PQ); **Reviewed:** 20-Jan-2023, QC No: jcalb-23-86468; **Revised:** 23-Jan-2023, Manuscript No: jcalb-23-86468 (R); **Published:** 30-Jan-2023, DOI: 10.4172/2375-4494.1000487

Citation: Congo S (2023) A Short Note on Neurodevelopmental Disorders Include Autism. J Child Adolesc Behav 11: 487.

Copyright: © 2023 Congo S. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Disorders, which now allows for a dual diagnosis of ADHD and autism. Because of the challenges that people with ASD face in their day-to-day lives, such as social isolation and communication difficulties, conditions like anxiety and depression frequently co-occur. However, overlapping biological processes that are disrupted in these disorders may also account for the co-occurrence. Schizophrenia is thought to be linked to ASD, which was once thought to be a form of schizophrenia in children. Behavior and brain biochemistry, as well as a few risk factors, appear to be shared by the two diseases. The individual may also exhibit other abnormalities, such as aggression and self-harming behaviors.

Epilepsy, sleep disorders, and issues with the gastrointestinal (GI) tract are examples of physiological comorbidities associated with ASD. ASD and epilepsy co-occur for unknown reasons, but it is likely that they are caused by brain pathologies like synaptic defects and similar risk factors (like prenatal environment). Strangely, people with ASD who also have an intellectual disability are more likely to get epilepsy than people who don't have an intellectual disability.

Another comorbidity of ASD is dysfunction in the immune system, as evidenced by an increase in cytokine levels and inflammation. The scientific literature has long recognized a connection between ASD and some autoimmune disorders. In addition, the possibility that the pathogenesis of ASD is influenced by the transfer of maternal autoantibodies to the developing fetal brain, affecting neurodevelopment, was mentioned. Additionally, it appears that maternal immune activation is a significant ASD risk factor.

More severe autism core symptoms, which typically range from profound, in which an autistic person may be nonverbal and unable to function without significant support, to relatively "high-functioning," are frequently correlated with the presence of a comorbidity. In the past, Asperger's syndrome was thought to be a high-functioning form of autism; however, this is no longer the case. According to DSM-V, Asperger's syndrome falls under the umbrella term ASD. Asperger's syndrome can manifest later, with an average age of seven years for diagnosis, and its symptoms are typically less severe than those of typical autism. Most of the time, people with Asperger's have average to above-average intelligence and don't have any delays in the development of their speech. However, other ASD shares symptoms like significant social impairment and restricted behaviors and interests.

When taken as a whole, ASD's clinical features and co-occurring conditions are highly variable and heterogeneous. These co-morbidities can have an impact on a variety of body systems and functional areas, from physiological to psychiatric. In recent years, researchers have begun to investigate the role that ASD alters other organ systems beyond the brain. The gastrointestinal (GI) system is one system heavily linked to ASD, and many people with ASD report GI dysfunction. Depending on the study, estimates of the autistic population's prevalence of GI disorders range from 20 to 86%. In addition to the fact that people with ASD have a higher prevalence of GI dysfunction than people who are not autistic, the severity of GI abnormalities appears to be correlated

with the severity of ASD, suggesting that the GI system may play a role in both the modification of ASD behavior and the etiology of ASD. The most frequently mentioned GI issues are abdominal pain, bloating, diarrhea, constipation, or gastroesophageal reflux [6-10].

Conclusion

It has been established that the gut-microbiome-brain axis influences behavior and contributes to neurodevelopment. Numerous studies have described dysbiosis of the gut microbiome in ASD patients and animal models. Stool and feces samples from ASD subjects and non-autistic individuals showed different levels of microbial diversity when the microbiome was studied. People with ASD not only have abnormal bacterial diversity, but they also have abnormal microbial composition, which may make GI pathology and inflammatory processes worse. Several studies report changes in the microbiota of Bacteroides and Firmicutes on a phylum level, despite conflicting reports due to differences in general methodology, sample size, the inclusion or exclusion of participants with known GI dysfunction, and possibly different diets due to different countries of origin. Additionally, individuals with autism have Actinobacteria that differ from those of control subjects in terms of phylum. The neuropathology of ASD is exacerbated by changes in the gut microbiota and abnormal function of the intestinal epithelial barrier (also known as "leaky gut"), both of which directly or indirectly trigger inflammatory processes that have an effect on cerebral function.

References

1. Skovgaard AM, Houmann T, Christiansen E, Landorph S, Jørgensen T, et al. (2007) The prevalence of mental health problems in children 1(1/2) years of age? The Copenhagen Child Cohort 2000. *J Child Psychol & Psychiat* 48: 62-70.
2. Egger HL, Angold A (2006) Common emotional and behavioral disorders in preschool children: presentation, nosology, and epidemiology. *J Child Psychol Psychiatry* 47: 313-337.
3. Wichstrøm L, Berg-Nielsen TS, Angold A, Egger HL, Solheim E, et al. (2012) Prevalence of psychiatric disorders in preschoolers. *J Child Psychol Psychiatry* 53: 695-705.
4. Wurmser H, Laubereau B, Hermann M, Papoušek M, Kries R (2001) Excessive infant crying: often not confined to the first three months of age. *Early Human Development* 64:1-6.
5. Becker K, Holtmann M, Laucht M, Schmidt MH (2004) Are regulatory problems in infancy precursors of later hyperkinetic symptoms? *Acta Paediatr* 93: 1463-1469.
6. Angold A, Egger HL (2007) Preschool psychopathology: lessons for the lifespan. *J Child Psychol & Psychiat* 48: 961-966.
7. Cierpka M (2014) *Beratung und Psychotherapie für Eltern mit Säuglingen und Kleinkindern*. Heidelberg: Springer Frühe Kindheit 0-3.
8. Stern D (1985) The interpersonal world of the infant.
9. Papousek H, Papousek M (1983) Biological basis of social interactions: Implications of research for understanding of behavioural deviance. *J Child Psychol Psych* 24: 117-129.
10. Trevarthen C, Aitken KJ (2001) Infant Intersubjectivity: Research, theory, and clinical applications. *J Child Psychol & Psychiat* 42: 3-48.