The International Journal of Indian Psychology
ISSN 2348-5396 (Online) | ISSN: 2349-3429 (Print)
Volume 13, Issue 1, January- March, 2025
DIP: 18.01.084.20251301, DOI: 10.25215/1301.084
https://www.ijip.in

Research Paper

Current Trends and Future Directions for Diagnosing and Managing Autism Spectrum Disorders (ASD)

Shoban Kumar Pakalapati[1], Dr. Shahnawaz Mushtaq Mangral[2]*

ABSTRACT

Autism Spectrum Disorder (ASD) is a prevalent neurodevelopmental disorder in youngsters. The age-based classification of ASD is inaccurate, as it often includes stereotypical behavior and hindered social communication. The illness is thought to be caused by a complex interplay of environmental, genetic, and epigenetic variables that lead to cerebral dysfunction. As the global prevalence of ASD exceeds 1%, governments, schools, and healthcare professionals are developing solutions to address this complex illness. Children with low school performance, bad behaviors, speech difficulties, or developmental delays require ongoing monitoring. Using a systematic technique makes it easy to evaluate a youngster with probable ASD. Early intervention improves social communication, imitation, and cognitive performance. This study examines the epidemiology, etiology, social-economic effects, and existing techniques for diagnosing and managing ASD. This paper outlines future diagnostic and therapeutic strategies for ASD.

Keywords: *Autism Spectrum Disorder (ASD), behaviour, diagnosis, management, speech or developmental delay*

ASD is a neurodevelopmental disease characterized by speech difficulty, social difficulties, and repetitive or restricted behaviors [1]. Autism Spectrum Disorder (ASD) begins in childhood and persists throughout adolescence and maturity. These diseases typically manifest within the first five years of life. ASD persons often have comorbidities such as depression, anxiety, ADHD, and epilepsy. Individuals with ASDs exhibit a wide range of intellectual functioning, from severe handicap to higher levels [2]. ASD affects one in every 700-1000 persons, with one in every 1000 experiencing classic autism symptoms. Globally, ASD affects 3-4 boys per girl. Autism spectrum disorder encompasses five clinical groups: Asperger's syndrome, autistic disorder, pervasive developmental disorder, Rett syndrome, and childhood disintegrative disease. These conditions impact emotional, cognitive, and social skills. Autism's etiology and neurological basis are unknown, however it is thought to be caused by genetic and environmental factors [3, 4]. ASD affects approximately 5 million Americans, with a 1.7% prevalence among children [5]. The symptoms of this condition are predominantly behavioral, with varying severity and cognitive function. They include disruptions in social interaction and

[1]Research scholar, Shri Venkateshwara University
[2]Research guide
*Corresponding Author

Received: January 04, 2025; Revision Received: February 09, 2025; Accepted: February 13, 2025

communication, a loss of interest in activities, and stereotyped behaviors. Symptoms typically include sensory processing abnormalities, impaired adaptive function, self-injury, or violence. Clinical diagnosis of central symptoms is typically based on DSM-5 parameters. Early and precise diagnosis is crucial for providing children with optimal care and counsel, leading to a better quality of life [6]. This review examines current ASD diagnostic and management practices, as well as potential future directions.

Epidemiology

ASD affects around one out of every 160 children globally [2]. According to the Centers for Disease Control and Prevention (CDC), around 1.68% of 8-year-old children in the US are diagnosed with ASD [5, 7]. According to the World Health Organization (WHO), the global prevalence of autism spectrum disorder (ASD) is 0.76 percent, accounting for around 16% of the global child population [8]. The Autism and Developmental Disabilities Monitoring Network (ADDM) indicates that the prevalence of ASD in the US more than quadrupled from 2000-2002 to 2010-2012 [5]. While analyzing trends in the US may be premature, the prevalence of ASD stabilized between 2014 and 2016, with no statistically significant rise [7]. Although ASD affects all socioeconomic, racial, and cultural groups, it is not a universal diagnosis. Caucasian children with ASD are more frequently diagnosed than Hispanic or black youngsters [5]. Genetic diagnoses such as Rett syndrome, Down syndrome, tuberous sclerosis, and fragile X have a higher incidence of comorbidities with ASD compared to the general population. However, these genetic conditions only account for a small proportion of ASD cases [4, 9, 10]. Research suggests that male children with sex chromosomal aneuploidy have a higher risk of autism due to a certain social functioning profile [11-14]. Increased usage of chromosomal microarrays has been linked to an increased risk of ASD at specific chromosome locations, including X, 2, 3, 7, 15, 16, 17, and 22 [15]. preterm and parental age are additional risk factors for ASD [16]. Older parents are more likely to experience complications during pregnancy, including preterm [17].

Causes

ASD is a neurological disease influenced by both genetic and environmental factors. Although research on the etiology of ASD is ongoing, no clear trigger has been identified. Epidemiological research does not support a causal link between ASD, rubella vaccination, mumps, or measles. Previous studies found methodological difficulties in establishing a causal relationship [20, 21]. There is no clear evidence that childhood vaccines increase the risk of ASD. Evidence suggests that the use of aluminum adjuvants and thiomersal in inactivated vaccines may not increase the incidence of ASDs [2]. Limited research on neuropathological variables has identified variances in limbic system deficits, cerebellar architecture and connections, and cortical alterations in the frontal and temporal lobes, among other abnormalities (9, 20, 21). In a short study of young children's neocortical architecture, most patients had a cortical laminar architecture with focal disruption, indicating issues with neuronal differentiation and layer creation [22]. Children with ASD exhibit brain enlargement, including increased extraaxial fluid and cortical size. Research on biomarkers and their underlying causes is ongoing (23, 24). Siblings of ASD patients are more likely to be diagnosed than the general population due to genetic factors [25-27]. Whole-exome sequencing and genome-wide association studies have improved our understanding of ASD susceptibility genes. Understanding their functions can shed light on potential biological mechanisms [28]. ASD potential genes include those involved with neuronal excitability, brain development, and neurotransmitter function [29, 30]. ASD-related genetic abnormalities typically affect activity-dependent neural alterations or specific

synaptic proteins, including transcription factors [25, 31]. Research indicates that advanced mother and paternal age increases the likelihood of having a kid with ASD [32]. Maternal history of autoimmune illnesses, such as psoriasis, thyroid disease, or diabetes, has been proposed, although research findings are varied [33, 34]. Recent research suggests that immunological activation during pregnancy or maternal illness may pose a risk factor [35-37]. Both longer and shorter inter-pregnancy periods have been linked to an increased incidence of ASD [38]. Premature birth is associated with an increased risk of ASD and other neurodevelopmental problems [38]. A previous epidemiological investigation found that low birth weight, premature delivery, low Apgar scores, caesarian delivery, and uterine bleeding were more consistently associated with autism [39]. A meta-analysis found that several risk factors, including maternal and paternal age, race, antepartum hemorrhage, threatened abortion, gestational diabetes, and gestational hypertension, were significantly associated with the risk of ASD during the prenatal period. During the perinatal era, autism risk variables included fetal distress, hypertension, breech presentation, no labor, induced labor, spontaneous labor, parity ≥4, gestational age ≤36 weeks, and caesarian delivery. Autism risk factors after birth include brain anomalies, male gender, maternal hemorrhage, and low birth weight [40]. Research on the factors related with ASD risk is ongoing, but no definitive conclusions have been reached [41].

Social and economic impacts

ASDs can significantly limit an individual's ability to participate in society and perform ordinary activities. ASDs can lead to unfavorable social, educational, and employment outcomes. Autism Spectrum Disorders (ASDs) can cause significant financial and emotional strain on individuals and their families. Caring for children with severe ASD can be challenging, especially when parental support and access to assistance is limited. Caregivers play a crucial role in caring for children with autism spectrum disorders. Some individuals with ASD can live independently, while others require extensive care and ongoing treatment [2].

Diagnosis

Early intervention can effectively detect ASD in children as young as two years old. The usual diagnosis age ranges from 3 to 6 years. Diagnosing these disorders at a young age is believed to be a contributing factor to the delay. A number of variables contribute to this: a) Language delays and social deficiencies may not be recognized until the child begins to interact with peers in pre-school. b) Symptoms are complicated and age-dependent [42, 43]. The American Academy of Neurology (AAN) recommends a dual method for detecting and diagnosing autism spectrum disorder (ASD) (Figure 1). To detect changes in children's neurodevelopment that require strict monitoring or deviations from the norm, it's crucial for specialists to remain vigilant from infancy. The second level of ASD diagnosis involves identifying the child's clinical characteristics, followed by three assessment phases [44]. The first part is identifying possible cases. The objective can be "to check the parents observations" rather than "to identify the manifestations regarding the social interaction and the child's communica- tion and behaviour" [44]. Second phase, worldwide evaluation: Examining a child's behavior and neurological symptoms can validate the results of referring physicians or parents' concerns [44]. The third phase, specific diagnosis, outlines the precise ASD diagnosis and its type. To link the data with the DSM-5 diagnostic manual criteria, the information of specialists and parents who have seen the patient is compared, and necessary tests are performed [44]. The DSM-5 is considered the gold standard for diagnosing ASD and closely aligns with implanted cardioverter-defibrillators (ICDs). The DSM-5 defines

ASD based on repetitive behaviors, restricted social communication, and impairments, with severity grades ranging from 1 to 3. This is similar to how Kanner children were first identified. Children with milder symptoms and "requires support" fall under group 1. Category 3 refers to children with severe symptoms who require extensive support. Clinical data often accompany diagnoses, including language impairments, intellectual impairments, environmental variables, hereditary problems, and medical conditions. The ICD and DSM have expanded their diagnostic criteria, with the DSM-5 combining PDD-NOS and Asperger's into a single ASD diagnosis [45]. Autism lacks biological markers, hence the diagnosis is solely dependent on behavioral symptoms as defined by the DSM-5. According to the DSM-5 manual, ASD disorders are characterized by chronic social impairment, reciprocal communication (criterion A), repetitive activities, interests, or restricting behavioral patterns (criterion B). Criteria C and D indicate that these problems have been limiting daily function since early childhood. The disorder's symptoms vary according on chronological age, degree of autism, and developmental level. The level of noticeable functional deterioration varies based on the individual's characteristics and surroundings [46, 47].

Diagnosis role in research

The broad diagnostic criteria for ASD resulted in a diverse range of participants in the study. Lynn Waterhouse's book Rethinking Autism [49] suggests that the DSM-5 ASD diagnosis should be abandoned for further investigation. Some studies advocate expanding the DSM-5 to include additional ASD subgroups [50]. SPARK, a significant ASD research effort by the Simons Foundation [51], uses questionnaires to exclude people with epilepsy or genetic diseases, without reclassifying them. Future of Diagnostics ASD is typically diagnosed by submicroscopic copy number variations (CNV) detection and microarray for large cases, followed by whole-genome sequencing (WGS) or whole-exome sequencing (WES). Genomic techniques, including as epigenomics and transcriptomics, were employed to study the genetic mechanisms of ASD and guide clinical treatment. Cell-free DNA (cf-DNA) testing is gaining importance in diagnostics. This test is also known as the Non-Invasive Prenatal Test (NIPT). Fetal cfDNA in maternal plasma can be examined for genetic diseases or related conditions using single nucleotide polymorphisms or next-generation sequencing techniques. Using clinical genomics or genetic testing as an in-vitro screening tool can help identify individuals at risk of autism. Advances in ASD clinical care rely on collaborative, multidisciplinary research. Efforts are underway to define functional subgroups of genetic-ontological methods for potential clinical intervention. This includes studying gene disruption-related symptoms that share molecular characteristics, such as CHD8 protein-regulated genes [52, 53]. Identifying gene abnormalities that cause observable neurological effects, like as electrophysiological (EEG) signals, can lead to functional biomarkers of ASD for therapeutic trials [60]. Developing therapeutics for genetic subtypes, such as SCN2A, requires more understanding of the reversibility and timing of genetic expression in neurodevelopmental disabilities. Continued thorough phenotyping is crucial for the success of the therapeutic trial on genetic ASD subtypes [52].

Management of ASD

The goals of ASD management include treating co-morbid conditions, reducing maladaptive behavior, promoting learning and problem-solving, addressing educational placement, communication and social skills, building cognitive skills, and providing coping assistance to families (Figure 2). Prepare a multidisciplinary team with tailored management. 1) Consult a clinician (psychiatrist, general practitioner, or competent pediatrician) for a

comprehensive assessment and pharmacological treatment. 2) Consult a clinical psychologist or developmental pediatrician for assessment of: i) Core symptoms include the Indian Scale for Assessment of Autism and the INCLEN: ASD Consensus Clinical Criteria [54, 55]. Both rating measures are not widely used among children [56]. Internationally recommended techniques include the Autism Diagnosis Observation Schedule General (ADOS-G) and the Autism Diagnosis Interview-Revised (ADI-R). In India, specialized training is underutilized due to limited suppliers and high costs. The CA-RS is commonly used to determine the severity of autism. Two Indian diagnostic technologies are now being evaluated. To effectively assess cognitive capacity, use tools that evaluate nonverbal and verbal competencies independently, such as Leiter's and Mullen's scales. iii) Adaptive function: The Vineland Adaptive Behavior Scale (second edition) assesses the quality and frequency of abilities observed throughout everyday activities (ADL). Regardless of quality, it outperforms growth metrics that solely assess skill. 3) Speech-language pathologists do language assessments to help with eating challenges and provide other communication options or speech therapy. 4) Consult a pediatric neurologist or geneticist if necessary. 5) Occupational Therapist: Provides ADL-based skill training and Sensory Integration (SI) therapy for individuals with sensory difficulties. SI improves children's responses to movement, sight, sound, and touch through controlled sensory inputs during play. 6) Behavior analyst for behavior change treatments. 7) Special educators create an Individualized Education Strategy and determine placement in education (integrated/inclusive, normal school, or special). 8) Adolescent and pediatric psychiatrist. 9) Psychiatric social workers provide family support. Unfortunately, there are few multidisciplinary centers that provide all of these services. There are insufficient skilled and trained staff to provide high-quality interventions. Professionals with experience and certifications working with ASD children are essential. Experts may have to multitask [48].

Psychological and behavioural management

After diagnosing ASD, a team-based approach is used to address deficiencies [4]. Regular treatments include play therapy, speech, behavioral, and occupational training. There are no pharmacological treatments for ASD, however medicine can help manage comorbidities including ADHD and seizures. Some schools provide support services for children with intellectual disabilities and learning problems [57]. Families and caregivers may struggle to connect with individuals with ASD due to communication problems. As part of their care, they are often urged to prioritize mutual pleasure and teamwork. In addition to standard therapy, families and individuals with ASD may seek community support. This may include online support groups or webpages. Autism Speaks [58] is a popular website where people may interact, gain moral support, and access the newest research. The prognosis for individuals with ASD varies depending on their diagnosis. Aspergers individuals with an ordinary to high intelligence quotient (IQ) can succeed in mainstream education, the workplace, and social interactions with the right support. High-achieving individuals, including those previously diagnosed with Asperger's, often embrace and profit on their unique neurodevelopment [59, 60]. Obsessive focus on items can hinder social connection, creativity, and adaptability. These qualities can be especially beneficial in fields that require limited focus, precision, and tight adherence to routines. Serious diagnoses have a less favorable prognosis. Severe instances may never develop communication skills, leading to isolation from classmates and family. ASD treatment seeks to enhance patients' and others' quality of life. Families and caregivers of individuals with ASD may struggle with communication and should engage in activities that promote bonding (61, 62). Improved prognoses include early psychotherapy intervention and the development of communicative

language before the age of six for children who score high on intensive nonverbal IQ tests and have minimal mental impairment. Collaboration among professionals, including educators, occupational therapists, speech therapists, psychologists, neuroscientists, psychiatrists, and neurologists, is vital for fostering understanding and addressing patients appropriately [63-65].

Future direction

Gene therapy shows potential as an effective treatment for autism spectrum disorder. The first preclinical investigations on monogenic ASD have been published, involving gene silencing and replacement. This work can be applied to polygenic ASD to better understand its genesis [66]. Genetically modified mice with monogenic disorders and disease-causing copy number variants, such as Rett syndrome (RTT) [67], Tumorous Sclerosis Complex (TSC) [68], and Phelan-McDermid syndrome (PMDS) [69], support the hypothesis that ASD has a common pathophysiology involving changes in cellular properties that lead to abnormal neural network activity and behavioral deficits [31]. Genome editing holds promise as a revolutionary medicine. CRISPR-gene editing strategies can improve neurodevelopment and mimic normal physiology in ASD models. Recent research utilized CRISPR-Gold, a new nanoparticle delivery technique, to target striatal mGluR5 levels in Fragile X syndrome (FXS) animals. The study demonstrated that reducing mGluR5 levels effectively restored repetitive behaviors in FXS mice [70].

CONCLUSION

Finally, ASD is very common in children. Screening for ASD in all young children should be implemented uniformly. This paper analyzed available tools for diagnosing and managing ASD. The study offers recommendations for future ASD diagnosis and management, assisting doctors in determining the most effective teraputic practices.

REFERENCES

[1] Hyman SL, Levy SE and Myers SM. Identification, evaluation and management of children with autism spectrum disorder. Pediatrics 2020; 145: e20193447.
[2] World Health Organization. Autism spectrum disorders. World Heal Organ 2019.
[3] Oviedo N, Manuel-Apolinar L, de la Chesnaye E and Guerra-Araiza C. Aspectos geneticosy
[4] Yoo H. Genetics of autism spectrum disorder: current status and possible clinical applications. Exp Neurobiol 2015; 24: 257-72.
[5] Baio J, Wiggins L, Christensen DL, Maenner MJ, Daniels J, Warren Z, Kurzius-Spencer M, Zahorodny W, Robinson Rosenberg C, White T, Durkin MS, Imm P, Nikolaou L, Yeargin-Allsopp M, Lee LC, Harrington R, Lopez M, Fitzgerald RT, Hewitt A, Pettygrove S, Constantino JN, Vehorn A, Shenouda J, Hall-Lande J, Van Naarden Braun K and Dowling NF. Prevalence of autism spectrum disorder among children aged 8 years - autism and developmental disabilities monitoring network, 11 sites, United States, 2014. MMWR Surveill Summ 2018; 67: 1-23.
[6] Ellison-Wright Z and Boardman C. Diagnosis and management of ASD in children and adolescents. Prog Neurol Psychiatry 2015; 19: 28-32.
[7] Xu G, Strathearn L, Liu B and Bao W. Prevalence of autism spectrum disorder among us children and adolescents, 2014-2016. JAMA 2018; 319: 81.
[8] Baxter AJ, Brugha TS, Erskine HE, Scheurer RW, Vos T and Scott JG. The epidemiology and global burden of autism spectrum disorders. Psychol Med 2015; 45: 601-13.

[9] Sztainberg Y and Zoghbi HY. Lessons learned from studying syndromic autism spectrum disorders. Nat Neurosci 2016; 19: 1408-17.
[10] Reddy KS. Cytogenetic abnormalities and fragile-x syndrome in autism spectrum disorder. BMC Med Genet 2005; 6: 3.
[11] Demily C, Poisson A, Peyroux E, Gatellier V, Nicolas A, Rigard C, Schluth-Bolard C, Sanlaville D and Rossi M. Autism spectrum disorder associated with 49, XYYYY: case report and review of the literature. BMC Med Genet 2017; 18: 9.
[12] Tartaglia NR, Wilson R, Miller JS, Rafalko J, Cordeiro L, Davis S, Hessl D and Ross J. Autism spectrum disorder in males with sex chromosome aneuploidy. J Dev Behav Pediatr 2017; 38: 197-207.
[13] Ross JL, Roeltgen DP, Kushner H, Zinn AR, Reiss A, Bardsley MZ, McCauley E and Tartaglia N. Behavioral and social phenotypes in boys with 47, XYY syndrome or 47 ,XXY klinefelter syndrome. Pediatrics 2012; 129: 769-78.
[14] Bardsley MZ, Kowal K, Levy C, Gosek A, Ayari N, Tartaglia N, Lahlou N, Winder B, Grimes S and Ross JL. 47,XYY syndrome: clinical phenotype and timing of ascertainment. J Pediatr 2013; 163: 1085-94.
[15] Johnson CP and Myers SM. Identification and evaluation of children with autism spectrum disorders. Pediatrics 2007; 120: 1183-215.
[16] Agrawal S, Rao SC, Bulsara MK and Patole SK. Prevalence of autism spectrum disorder in preterm infants: a meta-analysis. Pediatrics 2018; 142: e20180134.
[17] Parner ET, Baron-Cohen S, Lauritsen MB, Jørgensen M, Schieve LA, Yeargin-Allsopp M and Obel C. Parental age and autism spectrum disorders. Ann Epidemiol 2012; 22: 143-50.
[18] Maisonneuve H and Floret D. Affaire Wakefield: 12ans d'errance car aucun lien entreautisme et vaccination ROR n'a été montré. Presse Med 2012; 41: 827-34.
[19] Dyer C. Lancet retracts Wakefield's MMR paper. BMJ 2010; 340: c696.
[20] Skefos J, Cummings C, Enzer K, Holiday J, Weed K, Levy E, Yuce T, Kemper T and Bauman M. Regional alterations in purkinje cell density in patients with autism. PLoS One 2014; 9: e81255.
[21] Stoodley CJ, D'Mello AM, Ellegood J, Jakkamsetti V, Liu P, Nebel MB, Gibson JM, Kelly E, Meng F, Cano CA, Pascual JM, Mostofsky SH, Lerch JP and Tsai PT. Altered cerebellar connectivity in autism and cerebellar-mediated rescue of autism-related behaviors in mice. Nat Neurosci 2017; 20: 1744-51.
[22] De Rubeis S, He X, Goldberg AP, Poultney CS, Samocha K, Cicek AE, Kou Y, Liu L, Fromer M, Walker S, Singh T, Klei L, Kosmicki J, ShihChen F, Aleksic B, Biscaldi M, Bolton PF, Brownfeld JM, Cai J, Campbell NG, Carracedo A, Chahrour MH, Chiocchetti AG, Coon H, Crawford EL, Curran SR, Dawson G, Duketis E, Fernandez BA, Gallagher L, Geller E, Guter SJ, Hill RS, Ionita-Laza J, Jimenz Gonzalez P, Kilpinen H, Klauck SM, Kolevzon A, Lee I, Lei I, Lei J, Lehtimäki T, Lin CF, Ma'ayan A, Marshall CR, McInnes AL, Neale B, Owen MJ, Ozaki N, Parellada M, Parr JR, Purcell S, Puura K, Rajagopalan D, Rehnström K, Reichenberg A, Sabo A, Sachse M, Sanders SJ, Schafer C, Schulte-Rüther M, Skuse D, Stevens C, Szatmari P, Tammimies K, Valladares O, Voran A, Li-San W, Weiss LA, Willsey AJ, Yu TW, Yuen RK; DDD Study; Homozygosity Mapping Collaborative for Autism; UK10K Consortium, Cook EH, Freitag CM, Gill M, Hultman CM, Lehner T, Palotie A, Schellenberg GD, Sklar P, State MW, Sutcliffe JS, Walsh CA, Scherer SW, Zwick ME, Barett JC, Cutler DJ, Roeder K, Devlin B, Daly MJ and Buxbaum JD. Synaptic, transcriptional and chromatin genes disrupted in autism. Nature 2014; 515: 209-15.

[23] Shen MD, Kim SH, McKinstry RC, Gu H, Hazlett HC, Nordahl CW, Emerson RW, Shaw D, Elison JT, Swanson MR, Fonov VS, Gerig G, Dager SR, Botteron KN, Paterson S, Schultz RT, Evans AC, Estes AM, Zwaigenbaum L, Styner MA, Amaral DG, Piven J, Hazlett HC, Chappell C, Dager S,Estes A, Shaw D, Botteron K, McKinstry R, Constantino J, Pruett J, Schultz R, Zwaigenbaum L, Elison J, Evans AC, Collins DL, Pike GB, Fonov V, Kostopoulos P, Das S, Gerig G, Styner M, Gu H and Piven J; Infant Brain Imaging Study Network. Increased extra-axial cerebrospinal fluid in high-risk infants who later develop autism. Biol Psychiatry 2017; 82: 186-93.

[24] Hazlett HC, Gu H, Munsell BC, Kim SH, Styner M, Wolff JJ, Elison JT, Swanson MR, Zhu H, Botteron KN, Collins DL, Constantino JN, Dager SR, Estes AM, Evans AC, Fonov VS, Gerig G, Kostopoulos P, McKinstry RC, Pandey J, Paterson S, Pruett JR, Schultz RT, Shaw DW, Zwaigenbaum L and Piven J; IBIS Network; Clinical Sites; Data Coordinating Center; Image Processing Core; Statistical Analysis. Early brain development in infants at high risk for autism spectrum disorder. Nature 2017; 542: 348-51.

[25] Kim H, Keifer C, Rodriguez-Seijas C, Eaton N, Lerner M and Gadow K. Quantifying the optimal structure of the autism phenotype: a comprehensive comparison of dimensional, categorical and hybrid models. J Am Acad Child Adolesc Psychiatry 2019; 58: 876-886, e2.

[26] Sandin S, Lichtenstein P, Kuja-Halkola R, Larsson H, Hultman CM and Reichenberg A. The familial risk of autism. JAMA 2014; 311: 1770.

[27] Risch N, Hoffmann TJ, Anderson M, Croen LA, Grether JK and Windham GC. Familial recurrence of autism spectrum disorder: evaluating genetic and environmental contributions. Am J Psychiatry 2014; 171: 1206-13.

[28] Walsh CA, Morrow EM and Rubenstein JL. Autism and brain development. Cell 2008; 135: 396-400.

[29] McDougle CJ, Erickson CA, Stigler KA and Posey DJ. Neurochemistry in the pathophysiology of autism. J Clin Psychiatry 2005; 66: 9-18.

[30] Rubenstein JL and Merzenich MM. Model of autism: increased ratio of excitation/inhibition in key neural systems. Genes Brain Behav 2003; 2: 255-67.

[31] Zoghbi HY. Postnatal neurodevelopmental disorders: meeting at the synapse? Science 2003; 302: 826-30.

[32] Croen LA, Najjar DV, Fireman B and Grether JK. Maternal and paternal age and risk of autism spectrum disorders. Arch Pediatr Adolesc Med 2007; 161: 334.

[33] Xiang AH, Wang X, Martinez MP, Page K, Buchanan TA and Feldman RK. Maternal type 1 diabetes and risk of autism in offspring. JAMA 2018; 320: 89.

[34] Croen LA, Grether JK, Yoshida CK, Odouli R and Van de Water J. Maternal autoimmune diseases, asthma and allergies and childhood autism spectrum disorders. Arch Pediatr Adolesc Med 2005; 159: 151-7.

[35] Croen LA, Qian Y, Ashwood P, Zerbo O, Schendel D, Pinto-Martin J, Daniele Fallin M, Levy S, Schieve LA, Yeargin-Allsopp M, Sabourin KR and Ames JL. Infection and fever in pregnancy and autism spectrum disorders: findings from the study to explore early development. Autism Res 2019; 12: 1551-1561.

[36] Choi GB, Yim YS, Wong H, Kim S, Kim H, Kim SV, Hoeffer CA, Littman DR and Huh JR. The maternal interleukin-17a pathway in mice promotes autism-like phenotypes in offspring. Science 2016; 351: 933-9.

[37] Estes ML and McAllister AK. Immune mediators in the brain and peripheral tissues in autism spectrum disorder. Nat Rev Neurosci 2015; 16: 469-86.

[38] Schieve LA, Tian LH, Drews-Botsch C, Windham GC, Newschaffer C, Daniels JL, Lee LC, Croen LA and Danielle Fallin M. Autism spectrum disorder and birth spacing: findings from the study to explore early development (SEED). Autism Res 2018; 11: 81-94.
[39] Newschaffer CJ, Croen LA, Daniels J, Giarelli E, Grether JK, Levy SE, Mandell DS, Miller LA, Pinto-Martin J, Reaven J, Reynolds AM, Rice CE, Schendel D and Windham GC. The epidemiology of autism spectrum disorders. Annu Rev Public Health 2007; 28: 235-58.
[40] Wang C, Geng H, Liu W and Zhang G. Prenatal, perinatal and postnatal factors associated with autism. Medicine (Baltimore) 2017; 96: e6696.
[41] Ruzzo EK, Pérez-Cano L, Jung JY, Wang LK, Kashef-Haghighi D, Hartl C, Singh C, Xu J, Hoekstra JN, Leventhal O, Leppä VM, Gandal MJ, Paskov K, Stockham N, Polioudakis D, Lowe JK, Prober DA, Geschwind DH and Wall DP. Inherited and de novo genetic risk for autism impacts shared networks. Cell 2019; 178: 850-866, e26.
[42] Giangaspro EC and Pertejo MA. Instrumentos para la deteccion precoz de los trastornos del espectro autista. Pediatr Aten Primaria: Asociacion Espaola de Pediatria de Atencion Primaria 2007; 9: 301-15.
[43] Kim SK. Recent update of autism spectrum disorders. Korean J Pediatr 2015; 58: 8-14.
[44] Alvarez-Alcantara E. Trastornos del espectro autista. Rev Mex Pediatr 2007; 74: 269-76.
[45] Association AP. Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Publishing 2013.
[46] Blazer-Dan, Burke-Jack, William C, Castellanos X and Joel D. Trastorno del espectro autista. Manual diagnóstico y estadístico de los trastornos mentales. España 2014; 50-9.
[47] Cabrera. Generalidades sobre el autismo. Rev Colomb Psiquiat 2007; 36: 208-20.
[48] Mukherjee SB. Autism spectrum disorders - diagnosis and management. Indian J Pediatr 2017; 84: 307-14.
[49] Waterhouse L. Rethinking autism: variation and complexity: Academic Press 2013.
[50] Posar A, Resca F and Visconti P. Autism according to diagnostic and statistical manual of mental disorders 5th edition: the need for further improvements. J Pediatr Neurosci 2015; 10: 146-8.
[51] Sparks A. Examining the consequential validity of using an English language proficiency assessment for reclassification decisions; 2019.
[52] Cotney J, Muhle RA, Sanders SJ, Liu L, Willsey AJ, Niu W, Liu W, Klei L, Lei J, Yin J, Reilly SK, Tebbenkamp AT, Bichsel C, Pletikos M, Sestan N, Roeder K, State MW, Devlin B and Noonan JP. The autism-associated chromatin modifier CHD8 regulates other autism risk genes during human neurodevelopment. Nat Commun 2015; 6: 6404.
[53] Sugathan A, Biagioli M, Golzio C, Erdin S, Blumenthal I, Manavalan P, Ragavendran A, Brand H, Lucente D, Miles J, Sheridan SD, Stortchevoi A, Kellis M, Haggarty SJ, Katsanis N, Gusella JF and Talkowski ME. CHD8 regulates neurodevelopmental pathways associated with autism spectrum disorder in neural progenitors. Proc Natl Acad Sci U S A 2014; 111: E4468-77.
[54] Juneja M, Mishra D, Russell PS, Gulati S, Deshmukh V, Tudu P, Sagar R, Silberberg D, Bhutani VK, Pinto JM, Durkin M, Pandey RM, Nair MK and Arora NK; INCLEN Study Group. INCLEN diagnostic tool for autism spectrum disorder (INDT-ASD): development and validation. Indian Pediatr 2014; 51: 359-65.

[55] Ministry of Social Justice and Empowerment. Scientific report on research project for development of Indian scale for assessment of autism. 2009.
[56] Mukherjee SB, Malhotra MK, Aneja S, Chakraborty S and Deshpande S. Diagnostic accuracy of Indian Scale for Assessment of Autism (ISAA) in chidren aged 2-9 years. Indian Pediatr 2015; 52: 212-6.
[57] Guldberg K, Ashbee E, Kossyvaki L, Bradley R and Basulayyim A. Meeting the needs of pupils with autism in qatar: moving forward. Doha: Qatar WISE; 2017.
[58] Autism Speaks. Enhancing lives today and accelerating a spectrum of solutions for tomorrow; 2021.
[59] Cederlund M, Hagberg B, Billstedt E, Gillberg IC and Gillberg C. Asperger syndrome and autism: a comparative longitudinal follow-up study more than 5 years after original diagnosis. J Autism Dev Disord 2008; 38: 72-85.
[60] Howlin P, Goode S, Hutton J and Rutter M. Adult outcome for children with autism. J Child Psychol Psychiatry 2004; 45: 212-29.
[61] Fernell E, Eriksson and Gillberg C. Early diagnosis of autism and impact on prognosis: a narrative review. Clin Epidemiol 2013; 33: 33- 43.
[62] Styles M, Alsharshani D, Samara M, Alsharshani M, Khattab A, Qoronfleh MW and AlDewik NI. Risk factors, diagnosis, prognosis and treatment of autism. Front Biosci 2020; 25: 1682-717.
[63] Rogel-Ortiz. Autismo. Gac Méd Méx 2005; 141: 143-7.
[64] Calderon RS. El sindrome autista: un acercamiento a sus caracteristicas y generalidades. Rev Educ 2002; 26: 47-61.
[65] Gadia CA and Tuchman RF. Manejo de los ninos con trastornos del espectro autista. Rev Neurol 2003; 36: 166-73.
[66] Benger M, Kinali M and Mazarakis ND. Autism spectrum disorder: prospects for treatment using gene therapy. Mol Autism 2018; 9: 1-10.
[67] Chahrour M and Zoghbi HY. The story of Rett syndrome: from clinic to neurobiology. Neuron: Elsevier 2007; 56: 422-37.
[68] Sundberg M and Sahin M. Cerebellar development and autism spectrum disorder in tuberous sclerosis complex. J Child Neurol 2015; 30: 1954-62.
[69] Jiang Y and Ehlers MD. Modeling autism by SHANK gene mutations in mice. Neuron: Elsevier 2013; 78: 8-27.
[70] Lee JJ, Wedow R, Okbay A, Kong E, Maghzian O, Zacher M, Nguyen-Viet TA, Bowers P, Sidorenko J, Karlsson Linnér R, Fontana MA, Kundu T, Lee C, Li H, Li R, Royer R, Timshel PN, Walters RK, Willoughby EA, Yengo L; 23andMe Research Team; COGENT (Cognitive Genomics Consortium); Social Science Genetic Association Consortium, Alver M, Bao Y, Clark DW, Day FR, Furlotte NA, Joshi PK, Kemper KE, Kleinman A, Langenberg C, Mägi R, Trampush JW, Verma SS, Wu Y, Lam M, Zhao JH, Zheng Z, Boardman JD, Campbell H, Freese J, Harris KM, Hayward C, Herd P, Kumari M, Lencz T, Luan J, Malhotra AK, Metspalu A, Milani L, Ong KK, Perry JRB, Porteous DJ, Ritchie MD, Smart MC, Smith BH, Tung JY, Wareham NJ, Wilson JF, Beauchamp JP, Conley DC, Esko T, Lehrer SF, Magnusson PKE, Oskarsson S, Pers TH, Robinson MR, Thom K, Watson C, Chabris CF, Meyer MN, Laibson DI, Yang J, Johannesson M, Koellinger PD, Turley P, Visscher PM, Benjamin DJ and Cesarini D. Gene discovery and polygenic prediction from a genome-wide association study of educational attainment in 1.1 million individuals. Nat Genet 2018; 50: 1112-1121.

Acknowledgment
The author(s) appreciates all those who participated in the study and helped to facilitate the research process.

Conflict of Interest
The author(s) declared no conflict of interest.

How to cite this article: Shoban, K.P. & Shahnawaz, M.M. (2025). Current Trends and Future Directions for Diagnosing and Managing Autism Spectrum Disorders (ASD). *International Journal of Indian Psychology, 13*(1), 886-896. DIP:18.01.084.20251301, DOI:10.25215/1301.084