Natural Resources for Human Health
eISSN: 2583-1194

Original Research

Received 22 July 2026
Revised 28 Aug 2026
Accepted 10 Sep 2026

Natural Resources for Human Health 2026, Vol. 6 (22s): 1075–1086

KEYWORDS:
Autism spectrum disorder; Diagnostic biomarkers; Electroencephalography; Neuroimaging; Neuromodulation; Personalized medicine

Diagnostic Markers of Autism Spectrum Disorder and Emerging Neuromodulation Interventions: A Translational Review

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ABSTRACT: Autism spectrum disorder (ASD) is a complex developmental disorder with a variety of social communication and restricted and repetitive behaviours. The clinical manifestations are variable, which makes early diagnosis and personalized therapy difficult. In this review, emerging biomarkers and neuromodulatory strategies to diagnose ASD with a focus on mechanistic relationships and possible clinical utility are discussed. The studies on behavioural, electroencephalographic, neuroimaging, genetic, immune, and multimodal biomarkers are reviewed, as are studies on transcranial magnetic stimulation (TMS), transcranial direct-current stimulation (tDCS), and vagus nerve stimulation (VNS). Results suggest that multiple biomarkers hold promise of detecting differences in the brain or predicting treatment outcomes, but none of the currently identified biomarkers is sufficiently reliable by itself for an independent clinical diagnosis. There are some preliminary results for discrete social, cognitive, and behavioural outcomes that suggest that neuromodulation interventions may be beneficial; however, the evidence is sparse and limited by a small number of patients, different protocols, and few studies having a long enough follow-up period. The combination of the use of biomarkers with neuromodulation could help to achieve more precise stimulation targeting, objective monitoring of neural response, and individualized treatment strategies. However, methodological limitations, barriers to access, safety issues and ethical concerns remain significant obstacles to the application of clinical practice. There is a need for additional large-scale, longitudinal, and well-controlled studies to validate biomarkers, define a consistent stimulation protocol, and assess the long-term efficacy and safety of using biomarkers for guiding neuromodulation in ASD.

1. INTRODUCTION

Autism or Autism spectrum disorder (ASD) is defined as a neurodevelopmental disorder characterized by deficits in social communication and the presence of restricted interests and repetitive behaviors (Hodges et al., 2020). With an increasing prevalence of about 1 in 31 (3.2%) children aged about 5 years, ASD has been identified according to estimates from CDC's ADDM Network. Autism Spectrum Disorder is not specific to any racial or socioeconomic groups. Though there seems to be a clear gender bias, with the gender ratio of boys to girls with autism being nearly 3:1 in most countries (Shaw, 2025). Every year from 2002 to 2022, there has been an increase in the annual reported cases of autism, from 1 in 150 (2002) to 1 in 31 (2022) (CDC, 2025).

As described by Nielsen (2009), autism has many varied symptoms and characteristics. Not all people with autism manifest every characteristic. The following areas and specific behaviors are typical:

Sr. No.	Characteristics	Symptoms
1	**Social Interactions and Relations**	• Significant difficulty developing nonverbal communication skills (*e.g., eye-to-eye gaze, facial expression*) • Failure to establish friendships with children the same age. • Lack of interest in sharing enjoyment, interests, or achievement with other people. • Appearing to be unaware of others. • Lack of empathy • Difficulty relating to people
2	**Verbal and Nonverbal Communication**	• Delay in or lack of learning to talk • Problem taking steps to start and/or continue a conversation • Nonspeech vocalizations (*Eg, grunting, humming*) • Stereotyped and repetitive use of language (*Eg., echolalia, repeating what one has heard)* • Difficulty understanding listener's perspective every day (*Eg., does not understand humor, takes comments literally (communicates word for word), fails to catch implied meaning*)
3	**Activities and play**	• An unusual focus on pieces (e.g., focus on the wheels on the toy car rather than on the entire car) • Using toys and objects in an unconventional manner • Preoccupation with topics (*e.g., fascination with train schedules, Weather patterns, Numbers*) • A need for sameness and routines. (*Insists that environment and routine remain unchanged; insists on driving the same route every day*) • Stereotyped behaviors (*Body rocking, Hand flapping, Self-stimulatory behavior, Self-injurious behavior (head banging), Preoccupation with hands*)

The diagnosis and treatment of ASD are challenging since autism presents differently in every case. An individual's symptoms can vary in severity, development, language ability, intellectual functioning, and behaviour. This heterogeneity leads to individuals with the same diagnosis requiring very different responses to treatment (Hobson & Petty, 2021; Mottron & Bzdok, 2020). Another major challenge is the gap between early detection and access to effective intervention (Camarata, 2014). Although signs of ASD may be recognised during early childhood, identification does not always lead to immediate or appropriate treatment or intervention. Moreover, children's responses may differ to the same intervention, making it difficult to determine which approach will be most effective. Recent research has identified several potential ASD biomarkers, including differences in brain structure, functional connectivity, electrophysiological activity, and molecular processes (Clairmont et al., 2022). However, findings remain inconsistent because of clinical heterogeneity and differences in study methods, meaning that

no single biomarker is currently sufficient for diagnosis or treatment prediction (Louise Bogéa Ribeiro & Filho, 2023). Emerging neuromodulation techniques, such as repetitive transcranial magnetic stimulation and transcranial direct-current stimulation, may improve selected cognitive, behavioural and social outcomes (Ameis et al., 2020). However, the evidence remains preliminary because many studies use small samples and variable stimulation protocols (Han et al., 2023).

2. DIAGNOSTIC MARKERS OF ASD

Although previously, ASD was diagnosed mainly based on persistent differences in communication and restricted, repetitive communication behaviour, contemporary research has shown that clinical identification of ASD cannot rely solely on behavioural markers (Vargason et al., 2020). Instead, efforts in diagnosis have expanded towards a biomarker framework that includes behavioural, neurophysiological, neuroimaging, genetic, immune, metabolic, and multimodal indicators (Ansel et al., 2019). This change is driven by the understanding that ASD is a very diverse condition in both individuals and development, and that there is no single marker that will capture all of the diagnostic phenotype (Shen et al., 2020).

Though behavioural markers cannot be the only factor considered, they remain a factor considered in ASD diagnosis. There are standardized instruments such as the Autism Diagnostic Observation Schedule, Second Edition (ADOS-2) and the Autism Diagnostic Interview-Revised (ADI-R) which are widely used to structure observations of social reciprocity, communication, and restricted or repetitive behaviour. Meta-analyses of diagnostic biomarkers have been reported, indicating that assessments based on the ADOS demonstrate a range of accuracy, with pooled sensitivity and pooled specificity reported in various studies, highlighting its limited use as a stand-alone assessment but also its clinical utility. Hence, these instruments are considered as frameworks to support skilled clinical judgment and not as a definitive biological test (Ansel et al., 2019).

One of the key difficulties in the behavioural diagnosis is the high degree of heterogeneity found in ASD (Mottron & Bzdok, 2020). The clinical presentation is variable across individuals and is dependent on their age, language level, cognitive ability, and co-occurring conditions, making it difficult to arrive at a single behavioural phenotype across the spectrum of this disorder (Hobson & Petty, 2021). Research suggests that there is wide lifespan variability in the structure and function of ASD, with a focus on the dynamic behavioural presentation. Others suggest that the diagnostic category encompasses a range of different patterns of expression in symptoms, making it difficult to diagnose at a younger age, and contributing to the fact that some children are diagnosed later than others (Wallis, 2021). For this reason, behavioural markers are now considered as probabilistic indicators and must be interpreted taking into account the child's development and collateral history.

However, neurophysiological markers (EEG and ERPs) are a promising and non-invasive method for detecting abnormal brain processing in ASD, and these signals can also serve as a useful alternative to behavioural markers. It has been especially appealing for early detection because it is cost-effective, readily available, and able to record the activity patterns of the infant's neurons. Bosl, Tager-Flusberg, and Nelson (2018) conducted a developmental study where they compared EEG recordings from infants whose older sibling was diagnosed with ASD with infants without an older sibling diagnosed with ASD. After examining the nonlinear characteristics of the signals and applying statistical learning algorithms, the authors determined it was feasible to predict future diagnoses from the EEG measurements they collected from the 3-month-old babies. This shows the EEG's power to identify early indicators of ASD prior to the onset of behavioural symptoms, and can be useful in the diagnosis of the condition. The most consistent evidence for ERPs is in the area of ASD's auditory processing (typical M100 latency). More specifically, Robert et al. found that the latency delay, or delay in response, was greater in the right hemisphere for M100, which could be a marker for atypical encoding of simple auditory stimuli in children with ASD. This deviation can be used to aid in the differentiation of ASD from other developmental disorders that present with similar symptoms (Roberts et al., 2010). Other ERP studies have also shown increased latencies and deviations from normal amplitudes in response to novelty or to target stimuli, indicating irregularities in orienting response and attention processes (Sokhadze et al., 2009). Although the evidence is positive, the study of auditory ERP abnormalities in ASD is still not fully understood and is very complex. Although most studies find delayed neural responses to auditory stimuli in individuals with ASD, recent studies did not find this (Shan et al., 2023). For this reason, it is not possible to develop individualized ERP-based assessments, as there is a lack of evidence that backs up this form of intervention. Overall, both EEG and ERP data can be used in combination with other methods to aid in the detection of ASD.

Moreover, neuroimaging studies have played a significant role in the understanding of ASD by highlighting abnormal brain morphology, function, and connectivity. Total brain volume, regional volume, cortical thickness, cortical surface area, and patterns of cortical maturation have been measured using structural magnetic resonance imaging (MRI) studies. Meanwhile,

Functional MRI (fMRI) has been used to investigate the activity and connectivity within networks that were associated with social cognition, language, executive functioning, and sensory processing. Ecker et al. (2015) define ASD as a neurodevelopmental system-level disorder characterized by deviations in brain structure and function throughout development. Neuroimaging studies have suggested that the abnormalities are not localized to one brain region but are spread throughout multiple regions across the brain and that these abnormalities vary with age (Ecker et al., 2015). Some studies investigating functional connectivity have revealed altered coordination in large-scale networks, both in terms of direction and location of this coordination across age groups of participants (Ecker et al., 2015). There is cross-study variability that restricts the clinical use of MRI-based markers immediately, since variations in the selection of participants, imaging protocol, sample size, preprocessing, and/or statistical analysis can come into play. Consequently, nowadays, neuroimaging is more of a biological characterization and a tool used for the identification of groups instead of a common diagnostic tool.

Additionally, genetic studies demonstrate that ASD results from the interaction of numerous genetic and developmental factors. High-impact variants, however, and common variants with moderate effects, do not usually make much of a contribution to susceptibility to ASD. ASD affects genes that regulate organization of synapses, communication between neurons, regulation of chromatin structure, and early brain development. With some exceptions, such as SHANK3 and CNTNAP2, whose research results show the convergence of more than one molecular pathway. One of the largest exome-sequencing studies of ASD was performed by Satterstrom and colleagues (2020), who analysed 35,584 samples including 11,986 individuals with ASD. The study managed to find 102 genes associated with ASD, with it being revealed that these genes were related to two aspects in particular: developmental processes and neuronal functions. The results are consistent with a model in which genetic risk impacts various biological processes that could all, in the end, influence synapses and neural connectivity. Another approach is a polygenic risk score for inherited liability, which calculates the risk of an inherited predisposition by summing the impact of numerous common variants (Satterstrom et al., 2020). The current predictive level is rather limited, though; other measures are being investigated. For example, oxidative stress and mitochondrial indicators are being investigated, but they require further replication before they can be used clinically.

Moreover, another potential source of ASD biomarkers is abnormalities in immune and cytokine profiles. Research has shown both central nervous system inflammation and peripheral immune signaling (including changes in cytokines such as interleukin-1β, interleukin-6, tumor necrosis factor-α, and interleukin-8), suggesting that some individuals with ASD might show altered pro-inflammatory and anti-inflammatory signaling (Zhao et al., 2021). However, cytokine findings are inconsistent across numerous studies, as cytokine levels are influenced by age, sex, medication, sleep, infection, gastrointestinal problems, and other co-occurring conditions. As a result, immune markers similar to behavioural markers should be considered as an indicator of biological subgroups rather than a definitive diagnostic test. In the future, they have the potential to be used to benefit individuals with inflammatory profiles and to enable specifically targeted clinical monitoring or treatment for them.

Due to the limitations of individual markers, the development of multimodal diagnostic approaches has been greatly encouraged. These approaches include both high-impact variants and common variants with relatively small effect sizes, rarely contributing to profiles (Vargason et al., 2020). There are machine-learning algorithms that are built to analyse large, high-dimensional datasets and to identify complex patterns that are not apparent through conventional statistical methods used often. Research on ASD biomarkers indicate that multimodal models provide a greater predictive accuracy than any means of single modal diagnostic approaches (Ameis et al., 2020). Though promising, these multimodal approaches still require further validation in larger, more diverse, and independent samples. Smaller datasets with methodological variation,limited interpretability, and population bias might produce models that perform well in research and ideal settings but not as well in clinical practice. Therefore, multimodal markers should be developed as a tool that complements but does not entirely replace clinical expertise. With more clinical trials and improvements, by integrating behavioural, neurophysiological, neuroimaging, genetic, and immune information, individual approaches to ASD assessment and intervention can be more accurate and provide more opportunities for early detection.

3. NEUROMODULATION INTERVENTIONS IN ASD

Neuromodulation interventions have become an emerging potential for treatments of ASD due to growing evidence of the involvement of dysregulation of large-scale brain networks. Studies on neuroimaging and electrophysiology shinterleukin-1β of individuals with ASD often show atypical connectivity, excitation-inhibition balance, and cortical plasticity patterns, particularly in regions that support social cognition, language, attention, and behavioural flexibility. These neural differences are what suggest that targeted modulation of brain activity might help normalize network function and reduce the severity of core

symptoms (Wu et al., 2025). Furthermore, non-invasive neuromodulation interventions are exceptionally suitable for ASD because they do not require surgery and can be administered in outpatient settings while generally having favourable safety profiles (Oberman et al., 2024). Contrasting with some pharmacological interventions that target broad neurotransmitter systems and often cause systemic side effects, neuromodulation can be directed towards specific cortical regions and/or neural circuits implicated in ASD (Oberman et al., 2015). The spatial precision, combined with neuromodulation interventions' ability to adjust stimulation parameters across sessions, makes it a promising addition to behavioral and educational interventions (Zhang & Zhang, 2022).

One such example of a neuromodulation intervention is transcranial magnetic stimulation (TMS), particularly repetitive TMS (rTMS), which has been investigated as a treatment for ASD in multiple clinical trials. rTMS uses magnetic pulses to induce electrical currents in cortical tissue, and its effects depend on the stimulation frequency, intensity, and target region (Enticott et al., 2021). Low-frequency rTMS (generally 1Hz or lower) is categorized as inhibitory, whereas high-frequency rTMS (generally 5Hz and above) tends to be excitatory. As of recent rTMS studies, most of them target the dorsolateral prefrontal cortex (DLPFC), which is the region responsible for executive function, emotional regulation, and social cognition. Results show that low-frequency rTMS over the DLPFC can reduce repetitive behaviors and irritability, while high-frequency stimulation of the same region may enhance social functioning and reduce anxiety. Other targets that were tested include the supplementary motor area and premotor cortex, where low-frequency stimulation has been associated with improvements in movement-related cortical potentials and sensorimotor integration.

A meta-analysis found that TMS improved certain symptoms of ASD such as social withdrawal, repetitive behaviours, and executive function in adolescents with ASD who had an IQ greater than 65 (Hao et al., 2026). Moderate but significant reductions were also observed in repetitive and stereotyped behaviours and social interactions (although inconsistent) (Watt et al., 2008). Further, new protocols of TBS have been investigated recently, and some evidence suggesting improvement in social communication and sensory processing has been found (Tan et al., 2026). Sample sizes, however, are still small, and long-term durability of effects has not yet been determined, but there is a wide variation in protocols. A thorough roadmap for future TMS research in ASD was outlined by Oberman and Thatberg (2017) that highlighted the need for standardized protocols, larger sample sizes, and rigorous sham-controlled designs. They concluded that, although TMS holds promise for investigating neural mechanisms and the treatment of symptoms in ASD, methodological inconsistencies and a lack of statistical power have been observed. As a result, they recommended a consensus on stimulation parameters, objective outcome measures, and systematic sharing of data for meaningful cross-study comparisons to enable researchers to make inferences. (Oberman et al., 2015)

Another type of neuromodulatory intervention is direct current stimulation (tDCS), which involves applying weak currents via electrodes placed on the scalp to influence the excitability of the cortex (Thair et al., 2017). In ASD, tDCS has been applied to regions of the brain including DLPFC, temporoparietal junction and Broca's area, all of which are involved in aspects of social cognition, language processing and behavioural control. Low-frequency tDCS has been shown to enhance neuronal excitability, while cathodal tDCS has been shown to reduce neuronal excitability. TDCS is effective for improving social and communication skills, social awareness, and some of the behavioural symptoms in children and adolescents with ASD. The most consistent results have been shown for anodal stimulation of the left DLPFC, where it has shown benefits for emotional recognition, theory of mind, and social responsiveness (Alabbad et al., 2026). In a randomized controlled trial, bilateral tDCS enhanced autism symptom severity and theory of mind (Zemestani et al., 2022), and emotion regulation was also enhanced in comparison with sham stimulation. There are other studies that have investigated cathodal stimulation of the left DLPFC and cognitive training and found improvements in information processing related to social-movement (Chan et al., 2023).

One of the first randomized, double-blind, cross-over trials of tDCS in ASD was carried out by Amatachaya et al. (2014), who used anodal stimulation over the left DLPFC for 20 minutes for 5 successive days. The study also demonstrated that there were statistically significant gains in Childhood Autism Rating Scale, Autism Treatment Evaluation Checklist, and Children's Global Assessment Scale ratings for the 20 children with ASD following active stimulation as opposed to sham stimulation. The results offer early proof-of-concept evidence suggesting that tDCS could be a valuable clinical tool in ASD, especially in the long term, to reduce the overall severity of symptoms and enhance global functioning (Amatachaya et al., 2014). Despite these findings, protocols seem heterogeneous, and variability in outcome measures. Other studies have reported no difference between active and sham groups in language skills (Kessler et al., 2012). Therefore, additional reviews are warranted, and a greater focus should

be given to standardizing stimulation parameters, as well as additional trials to elucidate the optimal protocol and to identify who might be best suited to benefit from this intervention.

Moreover, a newer technique is vagus nerve stimulation (VNS), where the vagus nerve is electrically stimulated, which projects to the brainstem and affects widespread cortical and subcortical regions (Austelle et al., 2024). There are two ways of doing VNS: invasive and non-invasive. Where invasive VNS requires surgical implantation of a pulse generator, typically used for treatment-resistent epilepsy while non-invasive approaches include transcutaneous auricular VNS (taVNS), which stimulates the auricular breach of the vagus nerve through the ear, and cervical VNS devices applied to the neck.

In particular, VNS has been a point of interest in ASD research due to its involvement in autonomic regulation, mood, attention, and social behaviour. Some patients with epilepsy and ASD have benefitted from VNS for epilepsy and improvements in mood and behaviour. In preliminary studies, the non-invasive electrical stimulation of the vagus nerve (taVNS) was found to induce improvements in cognitive emotion regulation, social attention, as well as adaptive control (Elger et al., 2000). The causes of these effects may relate to heightened parasympathetic activity (which tends to pacify the body) and alterations in brain chemicals that affect attention, mood, and emotions.

Van Hoorn et al. (2019) conducted a review of the studies on vagus nerve stimulation (VNS) and behavioural outcomes in individuals with ASD, which led them to identify 11 studies of relevance, most being case series or case reports, and found some preliminary evidence for the use of VNS for epilepsy to also benefit behavioural symptoms in people with ASD (van Hoorn et al., 2019). The authors pointed out that there was limited evidence available and that more rigorous controlled studies were needed, though.

The only proven interventions for ASD (TMS, tDCS, VNS) are in the process of further testing and development; several emerging interventions are being tested for ASD. A technique used is Deep brain stimulation (DB,), which appears to be very variable, with electrodes placed in specific subcortical structures (Marini et al., 2023). This type of intervention has been used in animal studies and case reports; however, it is still experimental in the field of ASD. DBS could be used to modulate circuits related to repetitive behaviors, anxiety, and social motivation, but has limited potential because of ethical issues and safety considerations, particularly in children.

In the same way, focused ultrasound is another promising emerging neuromodulation technique, which involves the non-invasive modulation of neural activity by acoustic energy (Arulpragasam et al., 2022). While TMS or tDCS requires a surgical opening, focused ultrasound can reach multiple brain structures without surgery. Preliminary research has shown that ultrasound can affect synaptic plasticity and the dynamics of the networks, but real-life applications in ASD are still in their developmental stages (McLennan, 2016). These are possible future methods but need to be tested and approved for safety to be used in clinical practice.

Overall, non-invasive neuromodulation techniques seem to be well tolerated in patients with ASD. The following side effects are reported in some of the treatments: Mild headache, discomfort in the scalp, brief fatigue; serious side effects include seizures from rTMS. These seldom happen when safety procedures are strictly adhered to. According to most studies, tDCS does not lead to any serious side effects, except for minor tingling, itching, or skin irritation under the electrodes. There are surgical risks and side effects, including voice change, coughing, or difficulty breathing, particularly with invasive VNS.

In ASD, considering age is extremely important. Very little neuromodulation research has taken place among young children, and a lot more has been done among adolescents and adults. Neurological development is ongoing in children, and the impact of repeated stimulation on neural plasticity is not known. So ethical issues are stressed, including informed consent, particularly for persons with communication difficulties or intellectual disabilities. Neuromodulation interventions are recommended to be viewed as an additional form of intervention and not a replacement for evidence-based behavioural and educational interventions.

Although neuromodulation offers a promising and new avenue for treating core and associated symptoms of ASD, it is still experimental. While early diagnosis will help to prevent millions of future cases, larger, well-controlled trials with standardized protocols are needed to prove efficacy, optimize parameters, and to ensure long-term safety in the wide range of the population of ASD.

4. MECHANISTIC LINKS BETWEEN MARKERS AND INTERVENTIONS

With the integration of diagnostic biomarkers with neuromodulation, there will be real-life potential for individualized approaches to ASD treatment. Currently, neuromodulation protocols are often selected according to broad clinical categories like age, symptom severity, or the presence of repetitive behaviour, but individuals with AD differ widely in brain connectivity, cortical excitability, sensory processing, and developmental profile. Due to these factors, the same stimulation target and parameters might not apply to all individuals, and by doing so, there may be variable or limited therapeutic effects. Biomarkers which are derived from,EEG, fMRI, genetics, and behavioural assessment could help identify the neural characteristics. These are individual and help in the selection of stimulation targets.

In particular, EEGs are deemed especially useful due to their relatively inexpensive and portable nature while also carrying the capability to measure neural activity with high temporal resolution. Certain measures like oscillatory power, functional connectivity, ERP latency, and cortical complexity provide information about atypical sensory processing; considering altered alpha, theta, and gamma activity can be used to define one's neurophysiological percentile before starting treatment (Chen et al., 2026). Changes in EEGs after tDCS or TMS might allow for an objective measure of target engagement and how the patient might respond to the treatment. Research has reported considerations to modify EEG features, including peak alpha frequency and EEG microstates, and those changes might be associated with improvement in behavioural symptoms (Wei et al., 2025). Similarly, N170 responses and other ERP measures have been proposed in research as potential indicators of social processing differences and changes after treatment.

Additionally, functional MRIs complement the EEG by identifying atypical activity or connectivity in networks involved in social cognition, language, executive control, and sensory processing;well-controlled, individualized fMRI connectivity maps could guide TMS towards cortical regions that are connected to dysregulated networks. Instead of stimulating the same anatomical location in every patient, this approach would allow treatment to be based on the person's individual functional organisation. Recent research suggests that MRS protocols informed by fMRI have an improvement in precision of stimulation as it accounts for individuals' connectivity profiles (Hugdahl et al., 2012).

Other than the above-mentioned uses, biomarkers also carry an additional function. They can also be utilized as predictive biomarkers to predict responder status. It is a measurable characteristic that indicates whether a patient is likely to benefit from a particular intervention. In ASD, potential predictors are: baseline EEG patterns, functional connectivity, age, genetic profile, symptom dimensions, and measures of cortical inhibition or excitation. Though evidence is still preliminary, a systematic review of ASD response biomarkers has identified hundreds of candidate measures (Parellada et al., 2023). There is still a lack of sufficient evidence to guide clinical trials and treatment selection due to various reasons such as inconsistent study designs, small samples, inadequate replication, and differences in participant characteristics. Therefore, biomarkers should currently be used as research tools rather than definitive clinical decision-making instruments, but with more research and evidence, biomarkers have the potential to revolutionise treatment and intervention.

From another perspective, neuroplasticity may be the common mechanism through which different treatments improve brain function. Neuroplasticity is the capacity of the nervous system to modify synaptic strength, network connectivity, and functional organization in response to experience or stimulation. ASD is associated with atypical synaptic development, altered excitation-inhibition balance, and differences in the maturation of functional brain networks, which contribute to restricted behavioural flexibility, atypical sensory processing, and difficulties in social communication (Chen et al., 2024). There is a possibility that TMS and tDCS can influence these mechanisms by altering neuronal excitability and promoting activity-dependent plasticity. Repeated stimulation can produce effects that extend beyond the time of stimulation, especially when stimulation is paired with behavioural or cognitive training. Low-frequency rTMS can reduce excessive cortical excitability, whereas high-frequency rTMS and anodal tDCS can enhance activity in underactive networks (Lage et al., 2016). In theory, these effects could help rebalance dysfunctional neural circuits that have a role in social cognition, attention, language, and behavioural regulation.

This relationship between the above-mentioned ASD pathology is definitely not a simple normalization. ASD is biologically heterogeneous, and the appearance of atypical patterns varies in individuals and may not even be considered dysfunctional. Hence, instead of focusing on fixing neural activity back to a "normal pattern", the focus should be on producing therapeutic effects through compensation, network reorganization, or improving coordination between brain regions using neuromodulation. This approach also creates a potential feedback system in which biomarkers are assessed before, during, and after treatment. First, to identify a suitable target, researchers can then confirm if that target has been affected, and initial post-

treatment measures may determine whether post-treatment effects correspond to behavioural improvement. A biomarker-guided system has the ability to make neuromodulation more adaptive and reduce unnecessary stimulation. Decision-making between neural change and clinical benefit is still uncertain, as a biomarker can change without producing any meaningful functional improvement and behavioural improvement can occur without measurable normalization of the original neural abnormality.

On the whole, the area of utilizing neuroplasticity, though plausible, still requires longitudinal studies to determine whether observed neural changes are causes, mechanisms, or consequences of treatment response.

5. CLINICAL TRANSLATION

A number of scientific, technical, economic, and ethical issues arise regarding translating biomarkers of ASD and neuromodulation into regular clinical practice. While there are many potential biomarkers, most are in the discovery phase, and a biomarker that is clinically useful will require both a statistical association with ASD or treatment response and reliability, sensitivity, and eventually cost-effectiveness and utility over time for activity-dependent therapies. At present, biomarker research is often based on small and highly selected samples, which makes it hard to decide if the findings from the research can be generalised to different criteria (Klin, 2018).

Standardisation is required because of the high frequency of variability. To compare the results between studies accurately, EEG studies must follow a uniform protocol in the way brain activity is recorded, processed, and analyzed. Likewise, MRI studies require standardized scanning protocols, quality control, and data analysis. If techniques are not standardized, variability from one study to another could be due to technique differences, not to biological differences. A recent study on AI-based neuroimaging biomarkers has also highlighted the need for standardized protocols for data collection, processing, and analysis before the models can be compared and applied in clinical settings (Evgenia Gkintoni et al., 2025).

Also, access to these interventions is an additional barrier. The disadvantage of EEGs is that they are not as portable or as expensive as MRI, but they still need to have proper equipment and an appropriately trained individual to read them. MRI-guided neuromodulation can offer biomarker-guided anatomical precision with high costs and low availability but is time-consuming. In addition, repeated appointments and care and monitoring with trained clinicians are required for both TMS and tDCS. These requirements have led to greater inequities in access, especially among families with financial, geographic, or health system barriers. Geographically, in the translation of neurotechnology, there needs to be a stronger focus on affordability, the capacity of the workforce, and the ability to include a more diverse population, in addition to technical accuracy (Landscape Analysis of the Opportunities and Challenges for Neurotechnology in Global Health, n.d.).

Similarly, regulatory aspects are also equally crucial. Neuromodulation devices should meet safety requirements and manufacturing quality standards. Typically, trials are designed to differentiate between treatment efficacy and placebo efficacy, and to determine the relationship between treatment biomarkers and patient-specific outcomes. For neurological devices, a biomarker needs to be demonstrated to truly reflect the disease or treatment effect (Brennan, 2016). This is more difficult in ASD due to the diversity of the disease and the lack of a single disease mechanism in ASD.

Lastly, ethical issues become a large factor when it comes to clinical translation of these interventions. Particularly regarding consent and child and/or communication impaired protection. Clear management of incidental neuroimaging findings and awareness of indeterminate benefits and/or potential side effects. Experimental neuromodulations should not be marketed as a guaranteed cure, but as a potential treatment to help alleviate symptoms. The patient's aesthetic requirements, physiological state, and ability to assent/consent should be considered when determining the course of treatment.

6. CONCLUSION

Researchers are studying many different types of signs that might help identify or understand ASD. These include behavior, brain electrical activity such as EEG, brain scans such as MRI, genes, metabolism, and immune-system activity. That being said, there are currently no biological markers/tests that can diagnose autism accurately enough by themselves. Doctors still need to use a clinical assessment, including behavior, development, and medical history. Neuromodulation interventions, including TMS, tDCS, and VNS, offer promising methods for modifying atypical cortical and network activity, but their efficacy remains restricted by small samples, differing protocols, and there is not enough research showing whether improvements last long-term. In the future, biomarkers could potentially help doctors determine which part of the brain should be stimulated and which patients are most likely to benefit from a particular treatment.

7. FUTURE PERSPECTIVES

The combination of diagnostic biomarkers and personalized neuromodulation interventions should be a focus of future studies on ASD. Using EEG, fMRI, genetic data, and behaviour together could help identify more biologically distinct subgroups and help predict a person's response to their treatment. Machine learning developments could further aid analysis of multimodal datasets and help to select stimulation targets and parameters more accurately. Furthermore, longitudinal studies are needed to assess if the changes in neural activity induced by neuromodulation are related to persistent improvements in social communication, cognitive function, and behavioural regulation. Efficacy, safety, and reproducibility of the new method should be further verified by larger randomized controlled trials using a standardized protocol and a more heterogeneous population. Developmental differences should be considered, particularly as they relate to young children, who are highly plastic. Accessible, ethical, and biomarker-driven interventions have the potential to help develop more individualized and effective clinical strategies for people with ASD.

Funding Information

The author received no external funding for the preparation of this review manuscript.

Author's contributions

- Conception and Design: Yukiko Yuasa
- Literature review and analysis: Eimi Yuasa
- Manuscript Drafting: Eimi Yuasa
- Review and editing: Yukiko Yuasa

Acknowledgement

I would like to thank my teachers, parents, and friends for their guidance and support.

Conflict of interest

The author declares no conflicts of interest to disclose.

Ethics Statement

This manuscript is a literature-based review of previously published research and does not involve the recruitment of human participants, collection of primary patient data, or experimentation on animals. Therefore, institutional ethics committee approval and informed consent were not required for the preparation of this review.

Data Availability Statement

Data supporting the findings of this study are available from the corresponding author upon reasonable request.

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