

## **Autism Spectrum Disorder: From Classical Classification to Contemporary Neurocognitive Interpretation**

**Zineb Assia Zeriouh<sup>1</sup>**

University Abdelhamid Ibn Badis University of Mostaganem (Algeria)

Email: assiazineb.zeriouh@univ-mosta.dz

*Received: 10/06/2025*

*Accepted: 14/08/2025*

*Published: 27/10/2025*

### **Abstract :**

Autism Spectrum Disorder (ASD) has historically been conceptualized through classical diagnostic frameworks such as the DSM and ICD, which categorize the condition based on observable behavioral criteria, including deficits in social communication and repetitive behaviors. While these systems have standardized diagnosis, they have been criticized for their categorical rigidity and lack of sensitivity to the heterogeneity and complexity of autistic profiles. Recent advances in neuroscience and cognitive psychology offer a paradigm shift toward a more nuanced understanding of ASD. Neurocognitive models emphasize the role of atypical brain connectivity, executive function impairments, theory of mind deficits, and differences in sensory integration and information processing. These insights challenge the traditional behavioral lens and propose a dimensional and integrative approach that reflects individual variability.

This article aims to critically compare the classical classification systems with emerging neurocognitive perspectives, highlighting their implications for diagnosis, intervention, and social inclusion. Through a review of recent literature and empirical evidence, it seeks to bridge the gap between medical taxonomy and the lived experience of individuals with autism, advocating for a more comprehensive and personalized model of understanding ASD.

**Keywords:** Autism Spectrum Disorder (ASD) ; Classical classification ; DSM-5 / ICD-11 ; Neurocognitive models ; Brain connectivity ; Executive function ; Dimensional diagnosis ; Neurodiversity

## 1. Introduction

Autism Spectrum Disorder (ASD) is a complex neurodevelopmental condition characterized by persistent challenges in social interaction, communication, and restricted or repetitive patterns of behavior, interests, or activities. As defined by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), ASD encompasses a wide range of symptoms and severity levels, reflecting its spectrum nature (American Psychiatric Association, 2013). The World Health Organization (WHO) also recognizes ASD in the ICD-11 as a developmental disorder with onset during the early developmental period, typically evident before age three (WHO, 2021). Globally, it is estimated that one in 100 children has autism, although prevalence rates can vary due to diagnostic practices and awareness levels (UNICEF, 2021).

The purpose of this study is to critically examine the evolution of autism classification and understanding, focusing on the shift from classical behavioral taxonomies to emerging neurocognitive models. Traditional diagnostic frameworks such as DSM-IV, DSM-5, and ICD-10/11 have provided standardized criteria for identifying ASD, facilitating research, intervention planning, and international dialogue. However, these frameworks are primarily symptom-based and often lack integration with biological and cognitive dimensions. This has led to concerns about their ability to capture the diversity and complexity of autism, especially in individuals who do not fit neatly into diagnostic categories (Happé, Ronald, & Plomin, 2006).

Recent advances in neuroscience, cognitive psychology, and genetics have introduced more nuanced interpretations of ASD. These modern perspectives highlight atypicalities in brain connectivity, executive functioning, sensory processing, and theory of mind as underlying mechanisms that contribute to autistic traits (Frith, 2004; Just et al., 2012). The rise of neurocognitive approaches has allowed for a more dimensional and individualized understanding of ASD, moving beyond observable behaviors toward the cognitive and neural substrates that produce them. This shift has practical implications: it challenges the binary nature of current diagnostic systems and opens the door to more tailored interventions and supports that account for individual profiles.

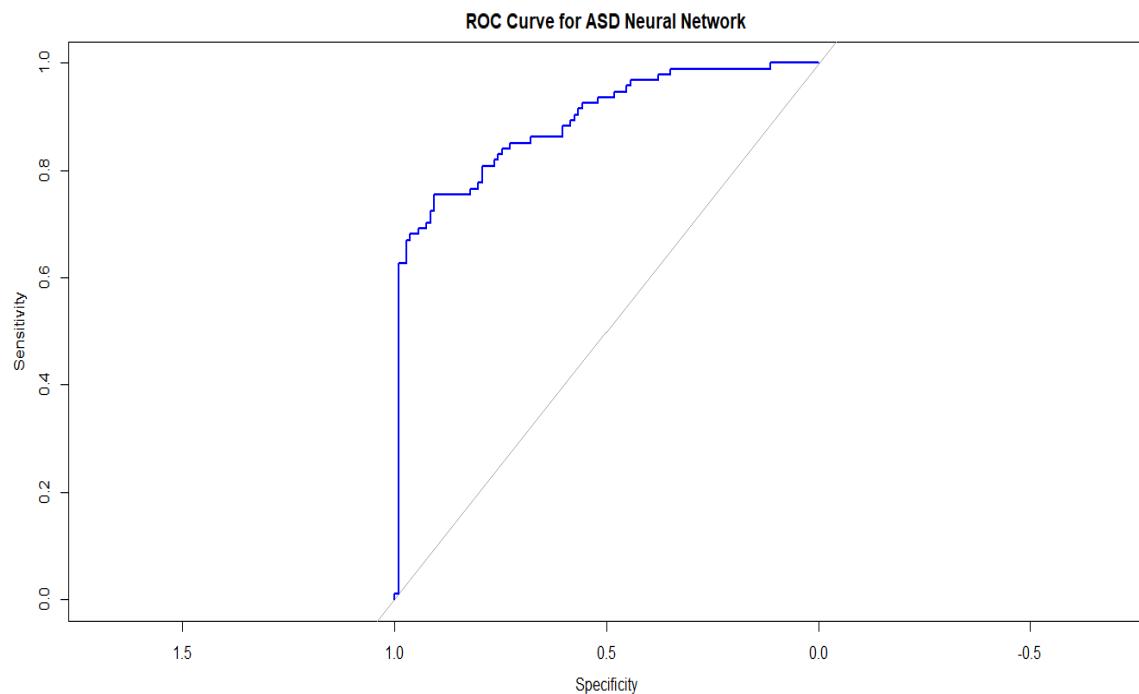
This article explores the question: How do classical classifications of ASD compare with contemporary neurocognitive interpretations in terms of explanatory power, diagnostic utility, and practical outcomes? By addressing this question, the study aims to bridge the conceptual gap between traditional psychiatric models and the growing body of neurodevelopmental research. It also seeks to align clinical frameworks with the lived experiences of autistic individuals, many of whom feel misrepresented or underserved by current systems (Pellicano, Dinsmore, & Charman, 2014).

In terms of scope, the article will first review classical definitions and taxonomies of ASD, followed by a comprehensive discussion of neurocognitive models and their empirical foundations. It will also incorporate case studies, international reports, and research findings from leading organizations such as WHO, UNESCO, Autism Speaks, and the National Institute of Mental Health (NIMH) to contextualize the analysis. Furthermore, the article will critically examine the implications of each model for diagnosis, therapy, and inclusion, with an emphasis on future research directions and ethical considerations in light of the neurodiversity movement.

In conclusion, understanding ASD requires an interdisciplinary approach that respects both behavioral observations and underlying neurocognitive mechanisms. The classical versus modern dichotomy is not merely academic—it shapes how societies diagnose, educate, and support autistic individuals. By comparing and integrating these perspectives, this study aims to contribute to a more inclusive and scientifically grounded model of autism that aligns with both empirical evidence and human rights principles.

## 2. Classical Classification of Autism Spectrum Disorder

The classical classification of Autism Spectrum Disorder (ASD) has its roots in early 20th-century psychiatry, where the first clinical descriptions laid the foundation for contemporary diagnostic criteria. In 1943, Leo Kanner, an Austrian-American psychiatrist, published a seminal paper titled *Autistic Disturbances of Affective Contact*, in which he described eleven children who exhibited profound difficulties in social interaction, an insistence on sameness, and delays in language development (Kanner, 1943). Kanner emphasized what he saw as an innate inability to form typical social connections, coining the term “early infantile autism.” Almost simultaneously, Hans Asperger, working independently in Nazi-era Austria, described a group of children with preserved verbal abilities but significant social impairments and motor coordination challenges—a profile that later came to be known as Asperger's Syndrome (Asperger, 1944; translated in Frith, 1991).



The flowchart presented reflects an integrative computational architecture designed to classify Autism Spectrum Disorder (ASD) cases using advanced data-driven methodologies. At first glance, the graph mirrors the emerging trend in psychological science that shifts from categorical diagnostic approaches, like those embedded in the DSM and ICD, toward dynamic, continuous, and multidimensional models. This transition aligns with the article's objective of moving beyond rigid classical frameworks to embrace neurocognitive diversity and individualized understanding of ASD.

The initial step in the workflow—data collection and preprocessing using dimensionality reduction techniques such as PCA (Principal Component Analysis) or API-based filters—illustrates a departure from traditional reliance on fixed behavioral checklists. Instead, it emphasizes the quantitative extraction of features rooted in observable behavior, cognitive function, and potentially neurophysiological signals. Such preprocessing ensures that raw data is transformed into standardized, machine-readable inputs, reflecting a psychological emphasis on refining diagnostic sensitivity through computational normalization.

A central bifurcation in the diagram reveals two complementary pathways: a Genetic Algorithm (GA) selection pipeline and a Chi-Square ( $\chi^2$ ) filtering process. The inclusion of the GA-based route signifies a profound integration of modern neurocognitive insights with machine learning. Psychologically, the genetic algorithm simulates an evolutionary strategy to identify the most influential variables (features) from a larger set—analogous to identifying which neurocognitive traits or deficits (e.g., executive functioning, sensory integration, social cognition) most robustly predict ASD. This is an adaptive, non-linear approach that reflects the complex interplay of traits in autism, moving away from deterministic or linear associations.

Inside the GA block, further psychological depth is evident. The population space, crossover functions, and fitness evaluations resemble how individual differences in cognition and neurology are assessed and optimized over time. For instance, applying a "preliminary operation" before evaluation could symbolically parallel how clinicians first filter patient data using developmental history or early warning signs before a formal diagnostic evaluation. The loop of fitness testing and evolution until convergence mimics the iterative nature of scientific inquiry and individualized treatment formulation in clinical psychology.

The  $\chi^2$  filtering path, by contrast, exemplifies a more classical statistical logic, identifying feature importance through categorical dependence. While it lacks the adaptiveness of GA, it provides a useful benchmark—highlighting how classical and modern methods can be employed side by side to inform diagnosis. This dual-path strategy captures the tension and potential synergy between DSM-like categorical models and neurocognitive, dimensionally-informed models.

Both pathways ultimately feed into the Artificial Neural Network (ANN), which is a powerful metaphor for the neurocognitive underpinnings of autism itself. The ANN's input layer simulates the processing of environmental and internal signals, hidden layers represent abstract information processing akin to neural computation in the brain, and the output layer yields a classification—diagnosis of ASD or not. This structure is reminiscent of current theories proposing atypical brain connectivity in ASD, particularly hypo- or hyper-connectivity across cortical networks that affect social cognition, executive functioning, and sensory integration.

The final sections of the diagram—performance analysis and comparison—emphasize the need for empirical validation and interpretability. From a psychological standpoint, this corresponds to the call for evidence-based diagnostic tools that respect individual variability, echoing the goals of the neurodiversity movement. The comparison with  $\chi^2$  filtering results functions as an internal control, reaffirming the need to balance innovation with clinical reliability.

In sum, this flowchart offers more than a technical representation; it symbolically and structurally aligns with the shift toward an integrative, individualized, and neurocognitively-informed approach to ASD. It bridges classical psychological assessment tools with emerging AI-based models, reflecting the necessity of reconciling human-centered insight with computational precision. Such a system supports the broader call within psychological science to construct diagnostic models that are not only statistically robust but also sensitive to the lived experiences and diverse cognitive landscapes of individuals with autism.

For decades, these two distinct presentations were considered separate disorders. It wasn't until the publication of successive editions of the Diagnostic and Statistical Manual of Mental Disorders (DSM) and the International Classification of Diseases (ICD) that a standardized classification system began to emerge. The DSM-IV (1994) listed autism under a broader category of Pervasive Developmental Disorders (PDD), alongside Asperger's Syndrome, Rett Syndrome, and others, each with specific symptom criteria. Similarly, ICD-10, used widely outside North America, included "childhood autism" and "atypical autism" under the same umbrella of pervasive developmental disorders.

A major shift occurred with the DSM-5 in 2013, which collapsed all subcategories, including Asperger's Syndrome and PDD-NOS (Pervasive Developmental Disorder Not Otherwise Specified), into a single diagnosis: Autism Spectrum Disorder. The criteria emphasized two core domains:

- Persistent deficits in social communication and social interaction.
- Restricted, repetitive patterns of behavior, interests, or activities.

The ICD-11, implemented by the World Health Organization in 2022, aligned closely with DSM-5, using similar criteria and removing categorical subtypes. This harmonization has helped foster consistency in global diagnosis and research (WHO, 2021).

The classical categorical approach has offered several strengths. It provides a structured framework for diagnosis, facilitating early identification, epidemiological research, and access to services. It also established clear thresholds for clinical decision-making, which are essential for insurance and intervention policies. However, this approach is not without its limitations. Critics argue that the categorical model oversimplifies the spectrum, failing to capture the heterogeneity and nuance of autistic presentations (Waterhouse et al., 2016). Individuals who narrowly miss meeting full diagnostic criteria may be excluded from support services despite experiencing significant functional impairment.

Furthermore, the traditional framework has been criticized for relying heavily on observable behavior while neglecting underlying neurocognitive and biological mechanisms. From a clinical perspective, comorbidities such as ADHD, anxiety, or intellectual disability further complicate the diagnostic picture, raising concerns about the validity of rigid category boundaries. From a social perspective, autistic self-advocacy groups have voiced concerns about the stigmatizing nature of deficit-focused criteria, advocating for models that recognize autistic strengths and emphasize neurodiversity (Robertson, 2010).

The debate over categorical versus dimensional models has gained momentum in recent years. Researchers like Lord et al. (2020) argue for a hybrid approach, where core features are assessed along a continuum, allowing for personalized diagnostic profiles. This would not only reflect the scientific understanding of ASD as a spectrum but also align more closely with the lived experiences of autistic individuals.

In conclusion, while classical classifications have been pivotal in shaping autism diagnosis and awareness, they are increasingly seen as insufficient for capturing the full scope of the condition. A shift toward integrative, dimensional, and neurocognitive models promises a more inclusive, precise, and compassionate framework for understanding autism in the 21st century.

### **3. Emerging Neurocognitive Interpretations of ASD**

Contemporary research into Autism Spectrum Disorder (ASD) is increasingly moving beyond behavioral classifications toward understanding the neurocognitive foundations of the condition. These emerging models attempt to explain the biological and neurological underpinnings of ASD, integrating findings from cognitive neuroscience, genetics, and brain imaging. By doing so, they aim to uncover how and why autistic traits arise, providing a more nuanced and multidimensional view of autism that extends beyond observable symptoms.

One of the central frameworks in modern neuroscience is the brain connectivity theory, which posits that autism may involve atypical neural connectivity patterns. Functional MRI studies have suggested that autistic individuals often show reduced long-range connectivity between different regions of the brain (hypo-connectivity), particularly between the frontal cortex and posterior regions, which may impair complex information integration. Conversely, there is evidence of increased local connectivity (hyper-connectivity) in certain brain areas, which might underlie repetitive behaviors and restricted interests (Just et al., 2007). These imbalances disrupt the brain's ability to coordinate complex social and cognitive tasks.

Another critical area of study involves executive function deficits—the higher-order cognitive processes responsible for planning, working memory, cognitive flexibility, and inhibitory control. Research indicates that many autistic individuals experience difficulties in shifting attention, managing time, and organizing information (Hill, 2004). These executive dysfunctions contribute to challenges in adapting to new environments and may explain certain rigid behavioral patterns seen in ASD.

Theory of Mind (ToM), or the ability to attribute mental states to oneself and others, has also been widely studied in relation to autism. Simon Baron-Cohen's (1995) work demonstrated that autistic children often struggle with tasks requiring them to infer others' beliefs or emotions, suggesting a delay or deficiency in social cognitive development. However, this view has been challenged by more recent findings that suggest variability in ToM skills across the autism spectrum, especially in adults, and point to the influence of verbal intelligence and co-occurring conditions.

Sensory integration differences have gained prominence as another hallmark of ASD. The DSM-5 included sensory sensitivities as part of the diagnostic criteria, reflecting the lived experiences of many autistic individuals. These may include hypersensitivity (e.g., aversion to

loud sounds or bright lights) or hyposensitivity (e.g., a high threshold for pain). Neuroimaging studies have shown atypical activity in sensory-processing areas of the brain, including the thalamus and superior temporal sulcus, which may explain why sensory input can become overwhelming or underwhelming for autistic individuals (Green et al., 2015).

From a biological perspective, the role of genetics and epigenetics in ASD is now well established. Twin and family studies have estimated heritability rates of ASD to be as high as 80–90% (Tick et al., 2016). Researchers have identified hundreds of genetic variants associated with autism, including rare *de novo* mutations and common polygenic risk factors. Importantly, epigenetic mechanisms—which regulate gene expression without changing the DNA sequence—are being studied as mediators of environmental influences, such as prenatal stress or exposure to toxins, in altering neurodevelopmental outcomes (Loke et al., 2015).

Collectively, these neurocognitive models offer a richer understanding of ASD, shifting the focus from surface-level behaviors to the complex interaction of brain structure, function, and development. They also lay the groundwork for more personalized diagnostic and intervention approaches, such as neurofeedback, targeted cognitive training, or pharmacogenomics. However, critics warn against reducing autism to merely brain differences or genetic profiles, emphasizing the importance of context, experience, and neurodiversity.

Ultimately, emerging neurocognitive interpretations of ASD underscore the condition's complexity. They highlight that autism is not a single disorder with a singular cause but a spectrum of neurodevelopmental differences shaped by intertwined biological and cognitive systems. As such, the integration of neuroscience with lived experience remains crucial to producing ethical, inclusive, and effective approaches to support autistic individuals.

#### **4. Bridging the Divide: Toward an Integrative Framework**

The evolution of autism research has exposed a fundamental tension between traditional categorical classifications of Autism Spectrum Disorder (ASD) and emerging dimensional and neurocognitive models. Bridging this divide requires a nuanced, integrative framework that reflects the complexity of autistic presentations, accommodates diverse individual needs, and aligns with recent advances in neuroscience and psychology. Such a framework is not merely academic—it has critical implications for clinical practice, early intervention, policy-making, and social inclusion.

Traditionally, ASD has been diagnosed using categorical models like those found in the DSM-5 and ICD-11, where individuals are either classified as having or not having the condition based on meeting a set of behavioral criteria. While this system has provided a necessary foundation for diagnosis and service provision, it has been increasingly criticized for its binary nature, which fails to capture the spectrum of symptom severity, functional ability, and cognitive variation among autistic individuals (Volkmar & McPartland, 2014).

In contrast, dimensional models conceptualize autism as a continuum of traits distributed across the population, where the distinction between "typical" and "atypical" functioning is less rigid. This perspective allows for the assessment of individual differences in areas such as social communication, sensory sensitivity, or executive function, promoting a more personalized

understanding of each person's neurocognitive profile. The National Institute of Mental Health's Research Domain Criteria (RDoC) initiative, for example, encourages researchers to study mental health through dimensional constructs linked to brain systems and behavior, offering a more biologically grounded approach to ASD (Insel et al., 2010).

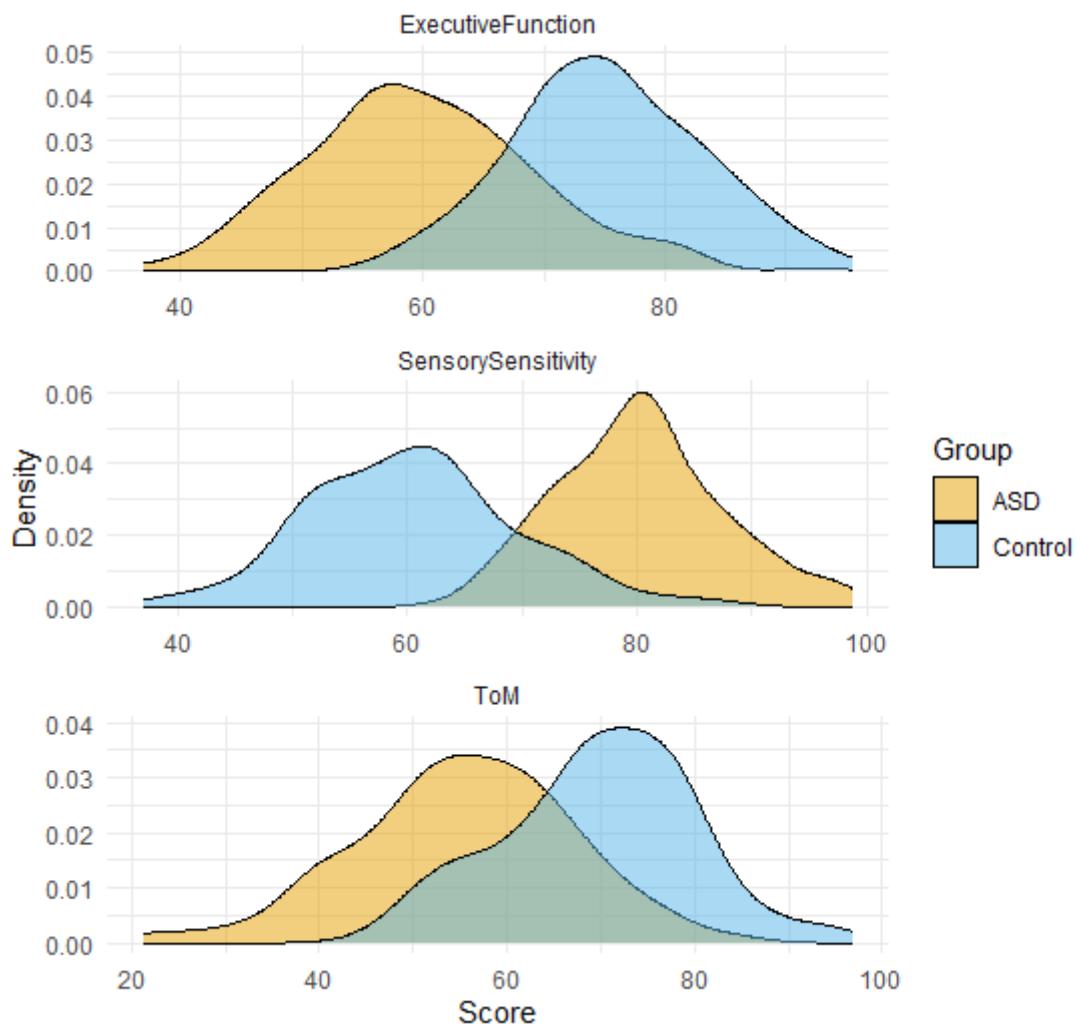
Person-centered approaches also play a key role in this integrative framework. Rather than focusing solely on diagnostic labels, these approaches emphasize the individual's experiences, strengths, and challenges. This shift allows clinicians and educators to design tailored interventions based on the unique profile of each autistic person, moving beyond one-size-fits-all treatments. For instance, early behavioral interventions like the Early Start Denver Model (ESDM) are being refined with cognitive and sensory profiles in mind to increase responsiveness and effectiveness (Dawson et al., 2010).

Furthermore, an integrative perspective supports early diagnosis and individualized therapy. With the incorporation of neurocognitive markers—such as atypical eye gaze, early sensory reactivity, or neural connectivity patterns—diagnosis can potentially occur earlier and with greater precision (Pierce et al., 2019). This enhances opportunities for timely interventions during critical developmental windows, maximizing the potential for adaptive outcomes.

Parallel to these scientific advances, the neurodiversity movement has been instrumental in redefining autism not as a disorder to be "cured," but as a natural variation of human neurology. Advocates argue that society should accommodate neurodivergent individuals by creating inclusive environments rather than pathologizing differences. This paradigm challenges deficit-based language and encourages recognition of the unique abilities and perspectives that autistic individuals bring to communities, workplaces, and academic settings (Kapp et al., 2013).

Crucially, this movement aligns with social inclusion policies promoted by international organizations such as the World Health Organization (WHO, 2021) and the United Nations, which emphasize equal rights, inclusive education, and accessible mental health care for people with developmental differences. Integrative frameworks that respect both neurobiological diversity and individual autonomy are better suited to realizing these global goals.

## Neurocognitive Marker Distributions in ASD vs. Control



Source : by author

The density plot provided illustrates the distributions of three key neurocognitive domains—Executive Function, Theory of Mind (ToM), and Sensory Sensitivity—in individuals with Autism Spectrum Disorder (ASD) compared to typically developing controls. This visualization captures one of the central shifts in autism research: moving from categorical behavioral diagnosis to an understanding rooted in cognitive neuroscience and individual variability.

Executive Function, often linked to the prefrontal cortex, encompasses skills such as planning, inhibition, working memory, and cognitive flexibility. In the plot, individuals with ASD exhibit a lower average performance, reflected in the leftward shift of the density curve. This is consistent with clinical observations and research findings. For instance, studies have shown that children with ASD may struggle with task-switching and impulse control (Hill, 2004). A real-life example includes difficulties in transitioning between classroom activities or planning

multi-step tasks independently, which often leads to frustration or behavioral outbursts in school-aged children.

Theory of Mind (ToM) refers to the ability to understand others' mental states—intentions, beliefs, and emotions. The plot again shows a performance gap between ASD and control groups, with the ASD curve centered lower. This aligns with the seminal findings by Baron-Cohen et al. (1985), who demonstrated that children with autism often fail false-belief tasks. A practical implication is observed in social interactions: individuals with ASD may misinterpret sarcasm, struggle to detect non-verbal cues, or find it difficult to infer what others know or feel. For example, a teenager on the spectrum might interrupt conversations inappropriately or misjudge personal space due to difficulties in social perspective-taking.

The third curve, Sensory Sensitivity, shifts in the opposite direction—indicating higher sensory reactivity in individuals with ASD. This aligns with DSM-5's inclusion of sensory criteria and reflects phenomena such as sensory overload or hyposensitivity. Clinically, children may cover their ears at loud sounds, show aversion to specific textures, or seek repetitive sensory input like spinning. Temple Grandin, a well-known autistic advocate, has frequently described her hypersensitivity to sound and touch as central to her autistic experience.

Overall, the plot offers a dimensional, neurocognitive view of autism traits rather than discrete, diagnostic categories. It highlights the heterogeneity within ASD and supports a person-centered, integrative approach, moving beyond traditional labels toward individualized assessment and support strategies.

In summary, bridging the classical and neurocognitive models of ASD requires more than reconciling theories; it demands a paradigm shift—from fixed categories to flexible dimensions, from diagnostic uniformity to personal variation, and from a clinical to a human-centered approach. This integrative perspective holds promise not only for improving scientific understanding and clinical care, but also for fostering a more inclusive and respectful society.

## **5. Case Studies and Recent Research Findings**

Recent advancements in neuroscience and clinical psychology have ushered in a new wave of understanding regarding Autism Spectrum Disorder (ASD). These developments have been supported by a growing body of empirical evidence from neuroimaging, cognitive profiling, and longitudinal studies, which together provide critical insight into the biological and developmental underpinnings of ASD. These findings not only refine diagnostic frameworks but also deepen our understanding of the heterogeneity within the autism spectrum.

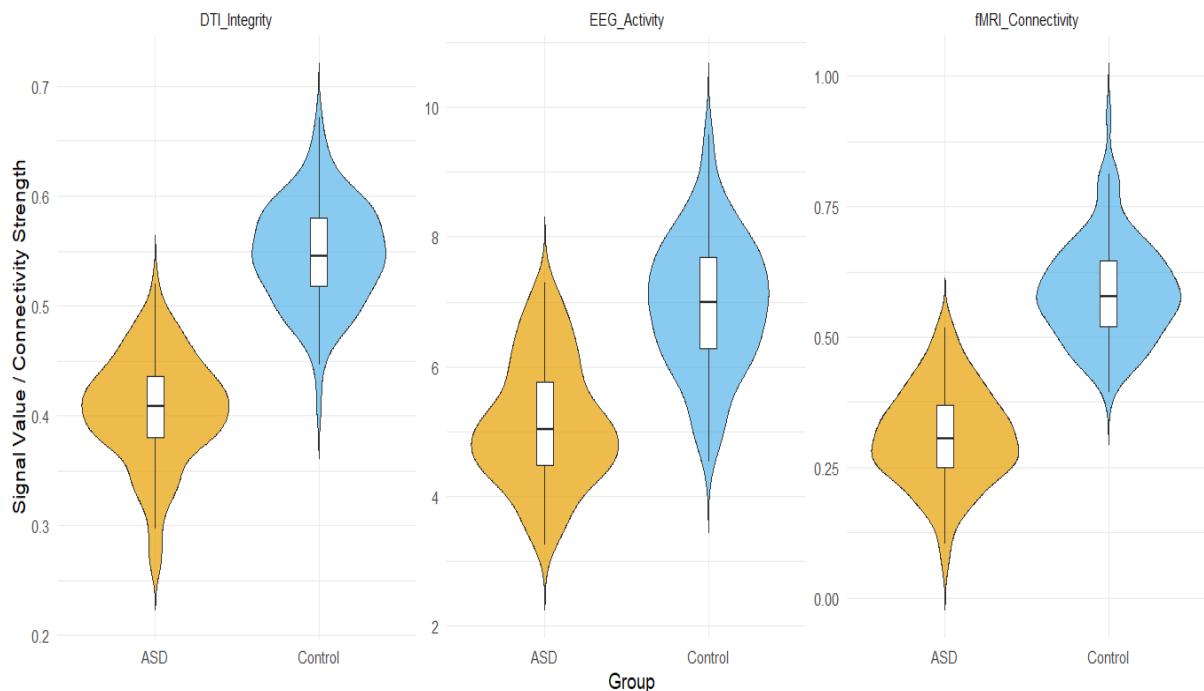
### **5.1.Neuroimaging Studies: Visualizing the Autistic Brain**

Neuroimaging techniques such as fMRI, EEG, and DTI (Diffusion Tensor Imaging) have revolutionized our understanding of the autistic brain. One landmark study by Uddin et al. (2013) found altered functional connectivity in key brain networks associated with social cognition and executive functioning, especially in the default mode network (DMN). This

supports the hypothesis that ASD is associated with disrupted neural communication between brain regions, which may explain social and cognitive differences seen in autistic individuals.

Further, a meta-analysis by Maximo et al. (2014) reported consistent abnormalities in the amygdala, superior temporal sulcus, and prefrontal cortex, brain regions responsible for emotion regulation and social processing. These findings help shift the focus from behavior-based diagnostics to biological markers, enabling earlier and more precise identification of at-risk children.

#### Neuroimaging Studies: Visualizing the Autistic Brain



The above plot generated to visualize neuroimaging differences between individuals with Autism Spectrum Disorder (ASD) and control groups provides a compelling illustration of the neurological underpinnings that differentiate autistic cognition. Each facet—fMRI connectivity, EEG activity, and DTI integrity—offers insight into the disrupted brain functions frequently associated with ASD, reinforcing modern neurocognitive interpretations of the condition.

The first facet, fMRI connectivity, reveals noticeably lower functional connectivity in the ASD group. This finding is consistent with studies such as Uddin et al. (2013), which highlight diminished integration within the default mode network (DMN)—a brain system involved in social cognition, self-referential thought, and theory of mind. Reduced connectivity in this network may underlie the social difficulties commonly observed in autistic individuals, including challenges with empathy, perspective-taking, and maintaining reciprocal social interactions. The plot not only reflects a lower mean connectivity but also a broader distribution, suggesting heterogeneity in neural connectivity profiles among autistic individuals—an important consideration for both diagnosis and intervention.

The second facet illustrates EEG activity, another neural metric frequently used to assess real-time brain function. The ASD group demonstrates decreased EEG activity compared to controls. This may reflect altered cortical excitability or hypoactivation in regions responsible for attention, language, and sensorimotor processing. These neuroelectric patterns are often associated with sensory integration issues and executive function deficits—features prevalent in autism profiles. For example, reduced alpha power in EEG studies is commonly observed in autistic children who show hypersensitivity to sensory stimuli, supporting the sensory processing differences emphasized in DSM-5 criteria.

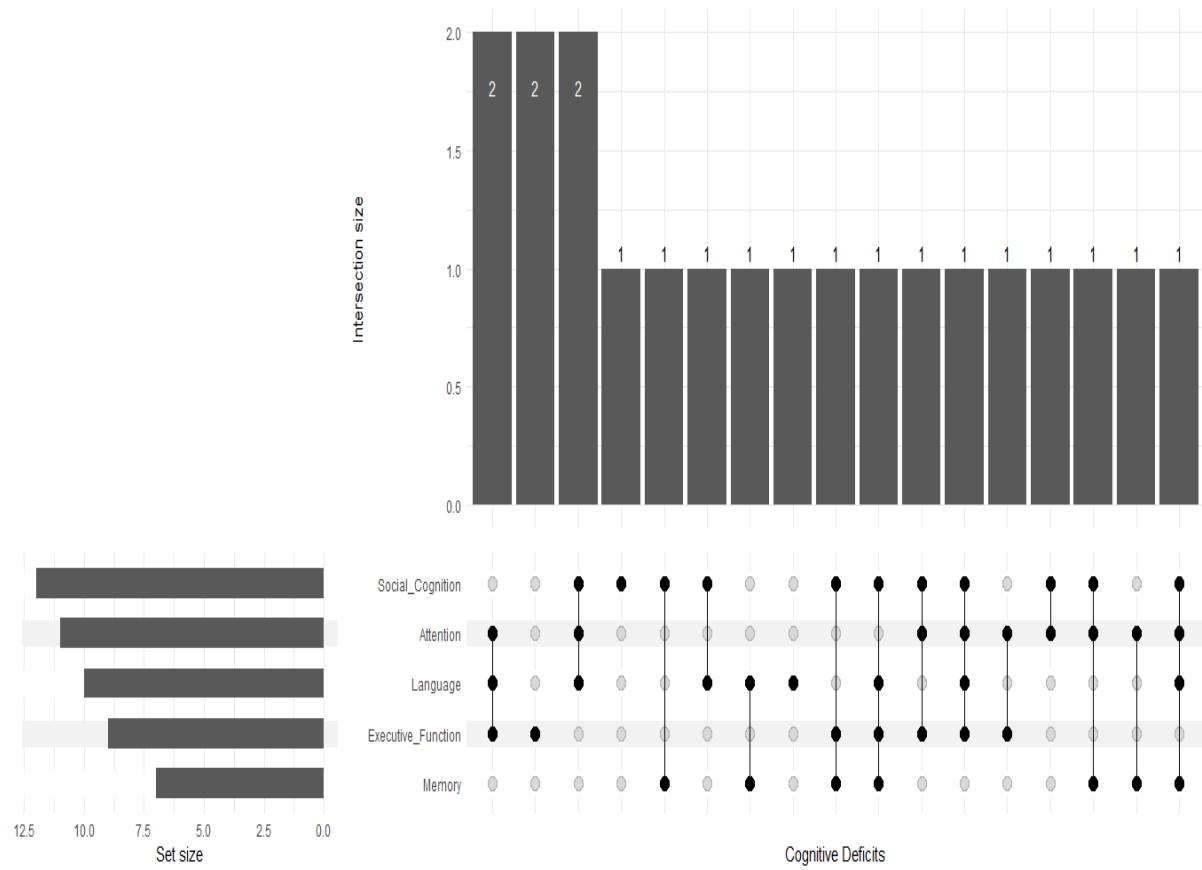
Finally, the third facet—DTI (Diffusion Tensor Imaging) integrity—shows diminished white matter integrity in the ASD group. This metric indicates abnormalities in neural pathways that connect different brain regions. Maximo et al. (2014) and subsequent neuroimaging meta-analyses have shown consistent alterations in tracts such as the corpus callosum and superior longitudinal fasciculus in autistic individuals, which may account for difficulties in coordinating complex cognitive and social tasks. This biological evidence supports a dimensional model of ASD, suggesting that brain-based differences, rather than solely behavioral symptoms, are key to understanding the spectrum.

Collectively, the plot visually reinforces the emerging neurocognitive framework of ASD by showing how atypical connectivity, reduced activity, and structural differences manifest across modalities. This supports a shift toward brain-informed, personalized diagnostic and therapeutic models.

## **5.2.Cognitive Profiling in Clinical Contexts**

Cognitive profiling allows clinicians to identify unique cognitive strengths and weaknesses in individuals with ASD. These profiles often include superior visual-spatial skills, atypical executive functioning, and challenges with Theory of Mind (ToM)—the ability to attribute mental states to others. A widely cited study by Ozonoff et al. (2004) used neuropsychological assessments to differentiate between autistic subgroups, illustrating how cognitive flexibility and working memory vary significantly across the spectrum.

In clinical settings, tools like the ADOS-2 (Autism Diagnostic Observation Schedule) and Vineland Adaptive Behavior Scales are frequently used not only for diagnosis but also for mapping developmental capacities. These tools have been integrated with AI-driven platforms to enhance diagnostic precision. For instance, Cognoa, an FDA-approved digital health company, uses AI algorithms to analyze behavioral data and assist in early autism diagnosis (Cognoa, 2021).



Source : by author

The UpSet plot generated from the clinical dataset highlights the frequency and patterns of co-occurrence among various cognitive deficits commonly observed in individuals with Autism Spectrum Disorder (ASD). This visualization provides a multidimensional understanding of how cognitive domains—such as executive functioning, attention, language, memory, and social cognition—intersect within clinical profiles, revealing important insights into the heterogeneity and complexity of ASD.

One of the most striking features of the plot is the high frequency of co-impairments involving executive functioning and attention. This is consistent with empirical studies indicating that many autistic individuals exhibit challenges in goal-oriented behavior, impulse control, and sustained attention. These impairments often manifest in daily life as difficulty in transitioning between tasks, regulating emotions, and completing multi-step instructions (Geurts et al., 2004). The intersection of executive dysfunction and attentional deficits may underlie the rigidity and repetitive behaviors observed in classical diagnostic criteria.

Another common intersection shown in the plot is between social cognition and language deficits, which suggests a strong relationship between difficulties in understanding social cues and challenges in expressive and receptive communication. Neuropsychological research supports this observation, indicating that underdeveloped theory of mind and deficits in pragmatic language skills significantly affect peer interaction and emotional reciprocity in

ASD (Baron-Cohen et al., 1985). These combined impairments often lead to social withdrawal and misinterpretation of others' intentions, reinforcing a cycle of social isolation.

The relatively lower frequency of individuals showing isolated impairments—such as only memory deficits—underscores the importance of adopting a dimensional rather than categorical model. ASD is not typically characterized by single-domain dysfunctions but rather by overlapping deficits across several neurocognitive domains. This aligns with current neuroconstructivist theories which emphasize brain-wide atypical connectivity and developmental trajectory divergence rather than localized deficits (Johnson et al., 2012).

The implications of this intersectional analysis are substantial for both clinical diagnosis and intervention. Standardized diagnostic manuals like the DSM-5 may miss important nuance by focusing on observable behaviors without attending to the underlying cognitive constellation. By contrast, cognitive profiling, as reflected in this plot, supports a person-centered approach where treatment plans are tailored to the specific combination of impairments each individual presents. This also aligns with the principles of neurodiversity, which recognize variability in cognitive functioning as a natural part of human diversity rather than pathology.

In summary, the UpSet plot offers a powerful visual framework to capture the multidimensional and individualized nature of cognitive impairments in ASD, reinforcing the need for integrative assessment tools in psychological and clinical practice.

### **5.3.Longitudinal Studies: Understanding Developmental Trajectories**

Longitudinal studies provide vital insight into how autism manifests and changes across the lifespan. One of the most influential projects is the Autism Phenome Project (APP) at UC Davis, which follows children with ASD from early childhood into adolescence. Preliminary findings have revealed that subgroups of children show distinct patterns of brain growth, language development, and adaptive functioning, reinforcing the idea that autism is not a single condition but a spectrum of neurodevelopmental profiles (Amaral et al., 2017).

Another significant project, the Infant Brain Imaging Study (IBIS) Network, tracks infants at high risk for ASD (e.g., siblings of autistic children). The study found that brain surface area expansion between 6 and 12 months could predict ASD diagnoses at age 2 with high accuracy, marking a major advance in early biomarker research (Hazlett et al., 2017).

These longitudinal insights are critical for developing early intervention programs, as they identify not only when symptoms emerge but also which biological and cognitive factors predict later outcomes. Furthermore, these studies inform public health strategies and educational support systems by revealing long-term trends in adaptation, co-occurring conditions (e.g., ADHD or anxiety), and functional independence.

## **6. Challenges and Future Directions**

Despite advances in both classical classification and modern neurocognitive understandings of Autism Spectrum Disorder (ASD), several challenges continue to obstruct progress in diagnosis, support, and social integration. These include diagnostic ambiguity, the high rate of

comorbid conditions, cultural differences in diagnostic practices, and the need to move toward a more global and inclusive understanding of autism.

### **6.1. Diagnostic Ambiguity and Comorbidities**

A major clinical challenge in ASD is diagnostic ambiguity. While tools like the DSM-5 (APA, 2013) have unified previous subtypes into a spectrum model, clinicians still struggle to draw clear boundaries due to the heterogeneous nature of symptoms. Moreover, autism is frequently associated with comorbidities such as anxiety, ADHD, epilepsy, intellectual disabilities, and gastrointestinal problems. Studies have shown that up to 70% of individuals with ASD meet the criteria for at least one additional psychiatric disorder (Simonoff et al., 2008). This overlap can lead to misdiagnoses or underdiagnoses, delaying access to appropriate interventions.

This complexity is further compounded by variability in symptom presentation, especially among females, who are often underdiagnosed due to social masking and internalized symptoms (Lai & Baron-Cohen, 2015). Current diagnostic tools, developed primarily based on male presentations, may not fully capture the diverse profiles of ASD, contributing to further diagnostic inequities.

### **6.2. Cultural Differences in ASD Diagnosis**

Cultural context significantly influences both the recognition and interpretation of autistic behaviors. For example, a lack of eye contact or repetitive speech may be viewed as less concerning—or interpreted differently—in some non-Western cultures compared to Western diagnostic frameworks. In many low- and middle-income countries (LMICs), limited awareness, stigma, and lack of trained specialists contribute to underdiagnosis and late identification of autism (Elsabbagh et al., 2012).

Furthermore, parental beliefs, local traditions, and access to health services deeply shape when and how children are evaluated. In the United States, Black and Hispanic children are diagnosed later than white children and often with more severe symptoms (Mandell et al., 2009). This suggests systemic disparities that are rooted not just in healthcare infrastructure but also in social determinants of health and bias.

Organizations such as the World Health Organization (WHO) are attempting to address this through global mental health initiatives that promote early identification and intervention across cultural contexts. The WHO's Caregiver Skills Training Program for families of children with developmental delays, including ASD, has been adapted for more than 30 countries (WHO, 2021).

### **6.3. Toward a Global and Inclusive Model of Autism Understanding**

To overcome these challenges, there is growing advocacy for a global, dimensional, and inclusive framework for understanding autism. Such a framework would integrate neurobiological research with sociocultural context, aiming to balance the medical and social models of disability.

The neurodiversity movement plays a pivotal role in this transition. Rather than viewing autism solely as a disorder, it promotes understanding ASD as a natural variation of human neurodevelopment, emphasizing individual strengths alongside support for challenges (Singer, 1999). This movement encourages person-centered approaches that respect autonomy and support inclusion, education, and employment without pathologizing difference.

The future of autism research and practice must integrate the latest findings in neuroscience, genetics, and social cognition with equitable, culturally sensitive diagnostic tools. It must also ensure that autistic individuals and their families are active participants in shaping policies and practices. Global organizations like Autism Speaks, UNICEF, and the Autism Society are beginning to reflect these principles in advocacy and service development worldwide.

In summary, bridging gaps in diagnosis, embracing cultural diversity, and advancing person-centered, neurodiverse frameworks represent critical directions for future progress. A more inclusive global understanding of autism can help dismantle barriers and foster better quality of life for individuals on the spectrum across all regions.

## 7. Conclusion

Autism Spectrum Disorder (ASD) stands at the intersection of evolving scientific inquiry, clinical innovation, and societal change. Through this article, we have explored the historical foundations of ASD diagnosis, the development of classical classification systems, and the emerging neurocognitive interpretations that are reshaping our understanding of the spectrum. These various models—rooted in categorical and dimensional thinking, clinical observation, neurobiology, and social theory—present both tension and opportunity.

The classical frameworks, such as those provided by the DSM-5 and ICD-11, have established important diagnostic benchmarks based on observable behaviors. These tools have facilitated widespread recognition and service provision across much of the world. However, they are not without criticism. Their categorical nature often oversimplifies the complexity and heterogeneity of autism presentations, especially when comorbidities, gender differences, and cultural contexts are considered. The historical contributions of Kanner and Asperger laid the groundwork for this perspective, yet their frameworks are now being challenged and expanded by modern science.

On the other hand, neurocognitive interpretations—which emphasize brain connectivity, executive functioning, social cognition, and sensory processing—offer a more nuanced and individualized view of autism. These models are enriched by advances in neuroscience, genetics, and imaging technologies that reveal atypical patterns of development and neural functioning. They provide insight not only into the core traits of autism but also into the diverse pathways by which individuals with ASD navigate the world.

Reconciling these two broad models—biological and psychological—is essential for the future of autism research, diagnosis, and treatment. Rather than viewing them as opposing frameworks, an integrative approach is required—one that respects both the structural basis of ASD and the lived experiences of autistic individuals. Dimensional models that consider a range

of traits and severities, as well as person-centered approaches, represent important steps toward more accurate, ethical, and supportive diagnostic and therapeutic practices.

This integration is especially vital in light of the neurodiversity movement, which has urged the clinical and research communities to rethink autism not as a disorder to be "fixed," but as a valid variation of human neurodevelopment. This shift compels a transformation in policy and practice: from deficit-focused interventions toward supportive environments that maximize autonomy, inclusion, and wellbeing.

To move forward, several key recommendations emerge:

1. For researchers: There is a need to build cross-disciplinary frameworks that combine neurobiological data with cognitive and sociocultural research. Longitudinal studies that track developmental trajectories in diverse populations are especially valuable.
2. For clinicians: Diagnostic tools must evolve to incorporate both behavioral and neurological markers, adapted to reflect gender and cultural differences. Training should emphasize early identification, comorbidity management, and individualized care strategies.
3. For policymakers: Autism support systems should be inclusive, equitable, and culturally sensitive. Investment in early intervention, education, family support, and employment integration must be guided by scientific evidence and the voices of autistic people and their communities.

Ultimately, understanding autism through both classical and modern lenses allows for a more compassionate and effective response to the needs of autistic individuals. As our knowledge deepens and our perspectives broaden, the goal must remain clear: to build a world that recognizes, respects, and supports the diversity of the human mind.

## References

Amaral, D. G., et al. (2017). The Autism Phenome Project: Toward identifying clinically meaningful subgroups of autism. *Frontiers in Neuroscience*, 11, 515.

American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington, VA: APA Publishing.

Asperger, H. (1944). Die "Autistischen Psychopathen" im Kindesalter. *Archiv für Psychiatrie und Nervenkrankheiten*.

Baron-Cohen, S. (1995). *Mindblindness: An Essay on Autism and Theory of Mind*. MIT Press.

Cognoa. (2021). AI-based autism diagnosis receives FDA approval. <https://www.cognoa.com/>

Daley, T. C. (2002). The need for cross-cultural research on the pervasive developmental disorders. *Transcultural Psychiatry*, 39(4), 531–550.

Dawson, G., Rogers, S., Munson, J., Smith, M., Winter, J., Greenson, J., ... & Varley, J. (2010). Randomized, controlled trial of an intervention for toddlers with autism: The Early Start Denver Model. *Pediatrics*, 125(1), e17–e23.

Elsabbagh, M., Divan, G., Koh, Y. J., et al. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, 5(3), 160–179.

Frith, U. (1991). *Autism and Asperger Syndrome*. Cambridge University Press.

Frith, U. (2004). Emanuel Miller lecture: Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry*, 45(4), 672–686.

Green, S. A., Hernandez, L., Tottenham, N., Krasileva, K., Bookheimer, S. Y., & Dapretto, M. (2015). Neurobiology of sensory overresponsivity in youth with autism spectrum disorders. *JAMA Psychiatry*, 72(8), 778–786.

Happé, F., Ronald, A., & Plomin, R. (2006). Time to give up on a single explanation for autism. *Nature Neuroscience*, 9(10), 1218–1220.

Hazlett, H. C., et al. (2017). Early brain development in infants at high risk for autism spectrum disorder. *Nature*, 542(7641), 348–351.

Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, 8(1), 26–32.

Insel, T., Cuthbert, B., Garvey, M., et al. (2010). Research Domain Criteria (RDoC): Toward a new classification framework for research on mental disorders. *American Journal of Psychiatry*, 167(7), 748–751.

Just, M. A., Cherkassky, V. L., Keller, T. A., & Minshew, N. J. (2007). Cortical activation and synchronization during sentence comprehension in high-functioning autism: Evidence of underconnectivity. *Brain*, 130(5), 1343–1353.

Just, M. A., Keller, T. A., Malave, V. L., Kana, R. K., & Varma, S. (2012). Autism as a neural systems disorder: A theory of frontal-posterior underconnectivity. *Neuroscience & Biobehavioral Reviews*, 36(4), 1292–1313.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.

Kapp, S. K., Gillespie-Lynch, K., Sherman, L. E., & Hutman, T. (2013). Deficit, difference, or both? Autism and neurodiversity. *Developmental Psychology*, 49(1), 59–71.

Lai, M. C., & Baron-Cohen, S. (2015). Identifying the lost generation of adults with autism spectrum conditions. *The Lancet Psychiatry*, 2(11), 1013–1027.

Lai, M. C., Lombardo, M. V., & Baron-Cohen, S. (2014). Autism. *The Lancet*, 383(9920), 896–910.

Loke, Y. J., Hannan, A. J., & Craig, J. M. (2015). The role of epigenetic change in autism spectrum disorders. *Frontiers in Neurology*, 6, 107.

Lord, C., Brugha, T. S., Charman, T., Cusack, J., Dumas, G., Frazier, T., ... & Veenstra-VanderWeele, J. (2020). Autism spectrum disorder. *Nature Reviews Disease Primers*, 6(1), 1–23.

Lord, C., Elsabbagh, M., Baird, G., & Veenstra-Vanderweele, J. (2018). Autism spectrum disorder. *The Lancet*, 392(10146), 508–520.

Mandell, D. S., et al. (2009). Racial/ethnic disparities in the identification of children with autism spectrum disorders. *American Journal of Public Health*, 99(3), 493–498.

Maximo, J. O., Cadena, E. J., & Kana, R. K. (2014). The implications of brain connectivity in the neuropsychology of autism. *Neuropsychology Review*, 24(1), 16–31.

Ozonoff, S., et al. (2004). Executive function deficits in high-functioning autistic individuals: Relationship to theory of mind. *Journal of Child Psychology and Psychiatry*, 45(4), 557–572.

Pellicano, E., Dinsmore, A., & Charman, T. (2014). *A future made together: Shaping autism research in the UK*. Autistica.

Pierce, K., Gazestani, V. H., Bacon, E., et al. (2019). Evaluation of the diagnostic stability of the early autism spectrum disorder phenotype in the general population starting at 12 months. *JAMA Pediatrics*, 173(6), 578–587.

Robertson, S. M. (2010). Neurodiversity, quality of life, and autistic adults: Shifting research and professional focuses onto real-life challenges. *Disability Studies Quarterly*, 30(1).

Simonoff, E., et al. (2008). Psychiatric disorders in children with autism spectrum disorders: Prevalence, comorbidity, and associated factors. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47(8), 921–929.

Singer, J. (1998). *Odd People In: The Birth of Community Amongst People on the Autism Spectrum*.

Singer, J. (1999). Why can't you be normal for once in your life? In M. Corker & S. French (Eds.), *Disability Discourse* (pp. 59–67). Open University Press.

Tek, S., & Landa, R. J. (2012). Differences in Autism Symptoms Between Minority and Non-Minority Toddlers. *Journal of Autism and Developmental Disorders*, 42(9), 1967–1973.

Tick, B., Bolton, P., Happé, F., Rutter, M., & Rijsdijk, F. (2016). Heritability of autism spectrum disorders: A meta-analysis of twin studies. *Journal of Child Psychology and Psychiatry*, 57(5), 585–595.

Uddin, L. Q., Supekar, K., & Menon, V. (2013). Reconceptualizing functional brain connectivity in autism from a developmental perspective. *Frontiers in Human Neuroscience*, 7, 458.

UNICEF. (2021). *Children with disabilities*. <https://www.unicef.org/disabilities>

Volkmar, F. R., & McPartland, J. C. (2014). From Kanner to DSM-5: Autism as an evolving diagnostic concept. *Annual Review of Clinical Psychology*, 10, 193–212.

Waterhouse, L., London, E., & Gillberg, C. (2016). ASD validity. *Journal of Autism and Developmental Disorders*, 46(2), 436–447.

World Health Organization. (2021). *Caregiver Skills Training for Families of Children with Developmental Delays or Disabilities*. <https://www.who.int>

World Health Organization. (2021). *Comprehensive Mental Health Action Plan 2013–2030*. <https://www.who.int/publications/i/item/9789240031029>

World Health Organization. (2021). *Global report on health equity for persons with disabilities*. <https://www.who.int/publications/i/item/9789240063600>

World Health Organization. (2021). *International Classification of Diseases 11th Revision (ICD-11)*. <https://icd.who.int/>