Razi Med J. 2025;1(3):212-221
https://doi.org/10.69667/rmj.25318
Razi Medical Journal
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Systematic article

Cytogenetic Study of Autism: A Systematic Review

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Abstract
Study of autism via a methodical search strategy predominantly through the Scopus database. The search employed terms including "autism," "autism spectrum disorder," "ASD," alongside "cytogenetics," "chromosome," "chromosomal abnormality," "copy number variation," "CNV," and "aneuploidy." The inquiry was confined to English, peer-reviewed publications without temporal limitations. The inclusion criteria emphasized original research, reviews, and case reports that elucidate cytogenetic or chromosomal investigations in persons with autism, encompassing classic karyotyping, aCGH, and SNP arrays, accompanied by explicit descriptions of findings and diagnoses. The exclusion criteria eliminated research focused on single-gene mutations lacking a cytogenetic component, non-English publications, editorials, and studies in which chromosomal mosaicism was a secondary observation. The findings indicated a vigorous scientific production in autism research, with annual publications continuously over 300 from 2021 to 2023; however, a decrease was observed in 2024 and 2025. The United States prominently led in publications, with over 600 documents, followed by Italy, the United Kingdom, China, and Canada. Prominent authors such as J.D. Buxbaum and A. Kolezvon significantly influenced research productivity. The review methodologically emphasized the growing integration of advanced genetic testing, such as Chromosomal Microarray Analysis (CMA), Whole Exome Sequencing (WES), and Whole Genome Sequencing (WGS), in conjunction with behavioral evaluations. Chromosomal Microarray Analysis (CMA) has become the recommended initial genetic assessment, detecting harmful copy number variations (CNVs) in 10-20% of autism spectrum disorder (ASD) cases, with elevated rates among individuals with intellectual disabilities. WES functioned as an ancillary instrument, providing diagnoses in 15-30% of ASD cohorts by identifying de novo pathogenic single-nucleotide variants (SNVs) and copy number variations (CNVs). Whole Genome Sequencing (WGS), albeit costly, provided the most thorough genomic perspective, detecting complex structural variants and copy number variations (CNVs) overlooked by alternative methods.
Keywords. *Cytogenetic Study, Autism, Candidate Regions, Cytogenetic Abnormalities.*

Received*: 02/06/25*
Accepted*: 09/08/25*
Published*: 17/08/25*

Introduction

Autism spectrum disorder (ASD) is a multifaceted neurodevelopmental syndrome marked by enduring impairments in social communication and interaction, alongside restricted and repetitive behavioral patterns, hobbies, or activities. The incidence of Autism Spectrum Disorder (ASD) has been progressively rising, with recent projections from the Centers for Disease Control and Prevention (CDC) indicating a prevalence of 1 in 36 children in the United States [1]. The etiology of ASD remains incompletely understood; however, it is generally acknowledged to involve a confluence of genetic and environmental elements. 2 Genetic variables are deemed to have a substantial influence, with heritability estimates between 70% and 90% [2]. Cytogenetic investigations, which examine chromosomes for abnormalities in quantity or structure, have proven essential in elucidating the genetic foundations of ASD. Three Initial cytogenetic studies concentrated on detecting significant chromosomal anomalies, including aneuploidies (Trisomy 21 in Down syndrome) and substantial structural alterations (translocations, deletions, and duplications). The research indicated that a modest yet noteworthy proportion of patients with ASD exhibit identifiable chromosomal abnormalities, especially in areas linked to neurodevelopmental problems [3].

The emergence of high-resolution cytogenetic techniques, such as array comparative genomic hybridization (aCGH) and single-nucleotide polymorphism (SNP) arrays, has significantly enhanced the resolution for identifying genetic alterations. Four of these approaches facilitate the detection of minor, submicroscopic chromosomal anomalies termed copy number variations (CNVs), which consist of deletions or duplications of DNA sequences. Five Multiple studies have shown an increased prevalence of uncommon and de novo CNVs in persons with ASD relative to the general population [4]. These CNVs frequently encompass genes essential for synaptic function, brain development, and chromatin remodeling, offering significant insights into the molecular processes altered in ASD. For instance, copy number variations in the 16p11.2, 15q11-q13, and 22q13.3 loci have been consistently linked to autism spectrum disorder

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https://doi.org/10.69667/rmj.25318
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[5]. Cytogenetic research has progressed from discovering big chromosomal aberrations to detecting small, functionally relevant submicroscopic copy number variations (CNVs). This advancement has been crucial in establishing a robust genetic basis for ASD, pinpointing specific chromosomal areas and genes linked to the disorder, and establishing the groundwork for future investigations into the intricate genetic structure of autism.

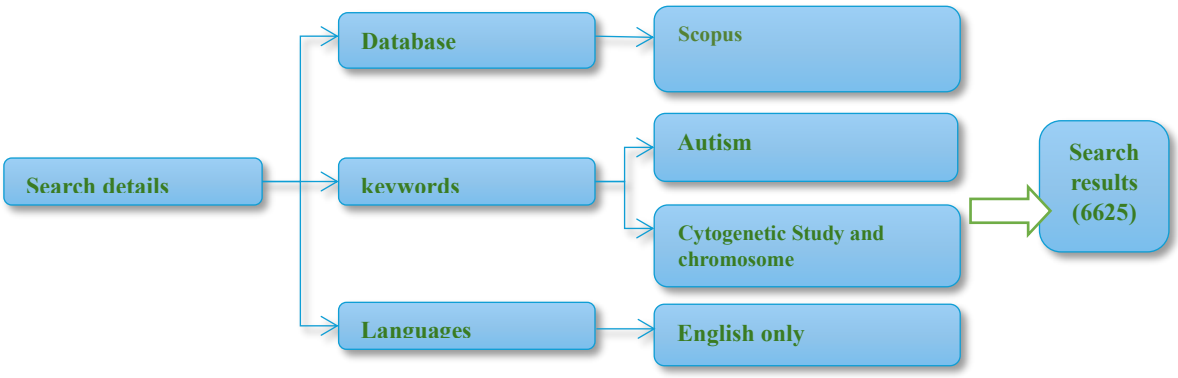
Methods

A thorough literature review was performed to identify and consolidate academic literature on the cytogenetic study of autism. The review method was structured to be systematic and replicable, adhering to recognized norms for academic evaluations where relevant.

Data source and research process

The principal database for this review was Scopus, owing to its extensive coverage of peer-reviewed literature in the biomedical and biological sciences. A systematic search was conducted utilizing a combination of the following keywords with various Boolean operators. Main Terms: "autism", "autism spectrum disorder," "ASD", Genetic Terms: "cytogenetics," "chromosome," "chromosomal abnormality," "copy number variation," "CNV," "aneuploidy". The search query was constructed to capture relevant literature broadly, for example: (autism OR "autism spectrum disorder" OR ASD) AND (cytogenetics OR chromosome OR "chromosomal abnormality). The search was limited to peer-reviewed articles published in English, with no date restrictions to ensure all relevant historical and recent findings were included. The search and analysis procedure are illustrated in (Figure 1).

Identification



Selection

Exclusion Criteria	Inclusion Criteria
-Studies that only focused on single-gene mutations without any cytogenetic component. -Documents not available in English. -Editorials, letters, conference abstracts (unless a full paper was subsequently published), and non-peer-reviewed materials. -Studies focusing on chromosomal mosaicism or other conditions where the cytogenetic abnormality is a secondary finding and not the primary focus of the autism research.	-Original research articles, review articles, and case reports focused on cytogenetic or chromosomal studies in individuals with autism. -Studies reporting on classic karyotyping, high-resolution techniques (e.g., array comparative genomic hybridization [aCGH], SNP arrays), or other methods for detecting chromosomal abnormalities. -Documents that provide a clear description of the cytogenetic findings and the clinical diagnosis of autism.

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Analysis

- **Publication Details:** Author(s), year of publication, journal.
- **Study Design:** review.
- **Sample Characteristics:** Number of participants, age, and method of ASD diagnosis (e.g., DSM-IV, DSM-5, ADI-R, ADOS).
- **Cytogenetic Method:** Karyotyping, FISH, aCGH, or other.
- **Key Findings:** A summary of the cytogenetic abnormalities reported (type of abnormality, chromosomal location, size of deletion/duplication).

Figure 1: Flowchart of the document collecting and selection process utilizing a research framework.

Data Analysis and Visualization

The collected data were systematically arranged to provide a thorough overview of the cytogenetic landscape of autism. The results were thematically synthesized, categorizing analogous chromosomal abnormalities or recurrent CNV regions. Visual representations, including figures, were employed to emphasize significant trends, such as the most commonly implicated chromosomal areas and the types of cytogenetic abnormalities identified. The findings were subsequently integrated to examine the progression of cytogenetic research in autism, from the identification of large-scale aberrations to the detection of submicroscopic CNVs, and their significance for comprehending the genetic framework of the condition.

Results and Discussion

Publications per year

This bar chart Figure 2 illustrates scholarly papers on autism from 2020 to 2025. The x-axis represents the year of publication, while the y-axis indicates the quantity of publications, ranging from 0 to 350. The findings indicate a substantial quantity of autism research conducted over time. A modest rise to 318 publications in 2021 from 300 in 2020 signifies sustained scientific engagement. Following a little decline to 298 publications in 2022, output increased to 312 in 2023. Publications decreased from 255 in 2024 to 147 in 2025. In 2021, 2022, and 2023, publications on autism research consistently exceeded 300, signifying a robust and active domain. The decline in 2024 and 2025 may necessitate additional analysis to determine whether it represents a genuine decrease in research output, a change in undocumented publication sites, or an artifact of data collection methodology, particularly for 2025. The scientific output in autism research is dynamic, as illustrated by this image.

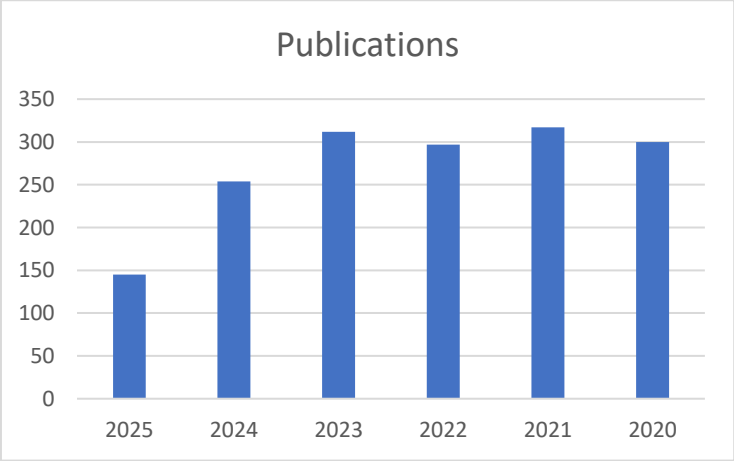

Figure 2: Change in the number of articles over the years

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Countries/Regions Publications

(Figure 3) The bar chart illustrates the geographical distribution of autism publications, presumably reflecting the scientific literature contributions from various countries. The x-axis represents ten countries, while the y-axis indicates a range of 0 to 700 documents. The United States' contributions surpass all others, totaling approximately 600 documents. Italy, the United Kingdom, China, and Canada produce 130 to 170 documents after this substantial output. Germany, the Netherlands, France, Japan, Spain, and Australia are classified as third-tier nations, possessing between 65 and 125 papers. This distribution indicates that North American and European nations predominantly lead in autism research publications, particularly the United States. The findings reveal that these regions excel in research infrastructure and output, whereas other developed nations contribute notably, though to a lesser extent. This graphic encapsulates global research productivity on autism.

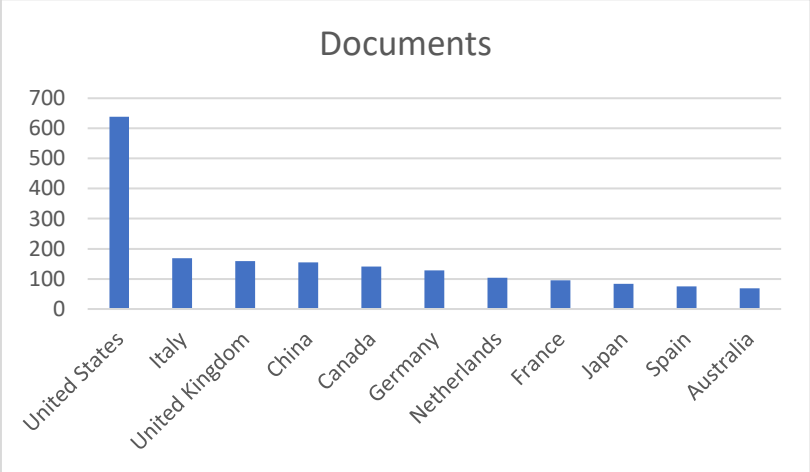

Figure 3: Global Distribution of Research Contributions by Country

Publications by Authors

The bar chart (Figure 4) illustrates the publication output of the ten leading autism researchers. The x-axis enumerates the authors, whereas the y-axis delineates their document counts, ranging from 0 to 25.

The results indicate that a few numbers of prolific authors dominate research output. J.D. Buxbaum and A. Kolezvon have authored 23 and 21 documents, respectively. Approximately 18 documents are meticulously overseen by C.E. Bearden. The second group—S.P.M. Siper, T. Takumi, M.G. Butler, S.W. Scherer, and T. Werge—has between 11 and 12 publications. T. Levy, A. Thurm, and D. Halpern produced 9–10 documents as a tertiary group. This graphic encapsulates the key individual contributions to the discourse on autism research. The distribution illustrates how prolific scholars influence the volume and trajectory of publications in this specialized domain.

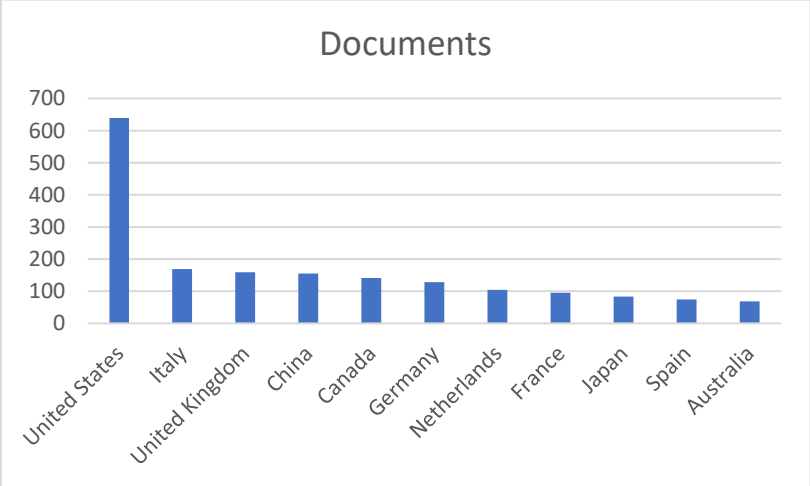

Figure 4. Top 10 publications by authors

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Methods of Autism Spectrum Disorder (ASD) Diagnosis

Behavioral assessment is essential; however, there is an increasing acknowledgment and incorporation of advanced genetic testing, specifically Chromosomal Microarray Analysis (CMA), Whole Exome Sequencing (WES), and the expanding significance of Whole Genome Sequencing (WGS), to uncover underlying etiologies in a considerable number of individuals. Emerging technologies, including artificial intelligence (AI) and telemedicine, are rapidly gaining prominence as prospective methods for improving diagnostic precision, accessibility, and efficiency.

Diagnostic Manuals: The Definitive Criteria

The diagnosis of ASD is standardized and consistent across clinical and research environments through the application of global categorization approaches. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision: The DSM-5 serves as the global standard for diagnosing autism spectrum disorder (American Psychiatric Association, 2013). Research and clinical protocols have demonstrated their enduring significance and extensive influence on diagnostic practices after 2020. The DSM-5 consolidates the DSM-IV's Autistic Disorder and Asperger's Disorder into a unified "autism spectrum disorder," acknowledging its many manifestations and severity. The essential diagnostic criteria for ASD remained unaltered in the 2022 DSM-5 language revision; nevertheless, it incorporated clarifications, enhanced descriptive terminology, and presented fresh information to align with contemporary scientific insights (Research, Society and Development, 2024) [6]. There are two primary diagnostics
domains for ASD:

Deficits in social communication

These encompass socio-emotional reciprocity, nonverbal communication, and the establishment of relationships.
Repetitive behaviors, interests, or activities: Stereotyped or repetitive motor movements (hand flapping, spinning), insistence on consistency or inflexible adherence to routines (significant distress at minor alterations, rigid cognitive patterns), highly restricted, fixated interests that are abnormal in intensity or focus, and hyperactivity are all requisite in this domain. Initial symptoms must result in significant social, occupational, or other impairments and cannot be attributed to intellectual disability or global developmental delay. The DSM-5 and DSM-5-TR include specifiers for intellectual or language disability, medical or genetic associations, and co-occurrence with other neurodevelopmental, mental, or behavioral disorders, thereby enhancing diagnostic precision and comprehensiveness. The International Classification of Diseases, 11th Edition (ICD-11) recognizes autism spectrum disorder (ASD) and associated diseases globally (WHO, 2023). The diagnostic criteria for ASD align with the DSM-5, facilitating global clinical practice, epidemiological data gathering, and research collaboration (ResearchGate, 2025). Similar to DSM-5, ICD-11 incorporated Asperger's Syndrome into Autism Spectrum Disorder (ASD).

Standardized Diagnostics: CBCAs

Specialized standardized methods for methodically evaluating ASD-specific behaviors and obtaining developmental history are considered "gold standards", surpassing diagnostic manuals due to their empirical rigor. Clinical practice from 2020 to mid-2025 depends on these instruments for comprehensive assessment.

The Autism Diagnostic Interview-Revised (ADI-R)

The Autism Diagnostic Interview-Revised (ADI-R) is a semi-structured interview designed for parents or primary caregivers of individuals suspected of having Autism Spectrum Disorder (ASD), as noted by [7]. It methodically records historical and current development in the primary ASD symptoms of reciprocal social interaction, communication, and restricted/repetitive behaviors.
Since 2020, the ADI-R has demonstrated its capacity to furnish longitudinal data and facilitate diagnosis across various age groups and intellectual capabilities[7]. Comprehensive clinical assessments necessitate the collection of medical history and symptoms. Second edition of the Autism Diagnostic Observation Schedule [8] characterized the ADOS-2 as a semi-structured observational evaluation of communication, social interaction, play, and restricted and repetitive behaviors. Specialized therapists evaluate predetermined activities to provoke behaviors associated with ASD. The components of ADOS-2 are determined by age (12 months to maturity) and expressive language [9]. Since 2020, research and clinical guidelines have endorsed the ADOS-2 for the assessment of ASD, yielding substantial observational data in a structured environment [10]. Experts assert that the ADOS-2, in isolation, is insufficient for diagnosis and must be complemented by further clinical assessments.

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Comprehensive Clinical Evaluation (2020–2025)

ASD is seldom diagnosed with a singular test or piece of information. This necessitates a multidisciplinary, multisource examination. Guidelines and literature underscore this comprehensive approach [10]. A standard element is inquiring with parents or caregivers regarding developmental milestones, skill regressions, and the beginning and progression of symptoms. Cognitive and Language Evaluations: Evaluating intellectual capabilities, linguistic proficiency, and adaptive behaviors to contextualize ASD diagnosis, identify concurrent intellectual impairments, and facilitate customized intervention planning. Medical and Genetic Assessment: Identify genetic etiologies and exclude associated disorders [11]. Advanced genomic testing is on the rise.

Ascendance of Genetic Methods (2020–2025)

Given the genetic variability of ASD, genetic testing is becoming progressively vital for diagnosis. Nonetheless, behavioral assessment constitutes the cornerstone of diagnosis. Chromosomal microarray analysis (CMA) is the primary genetic assessment for autism spectrum disorder (ASD), developmental delays, and intellectual disabilities [12]. Twenty-one. This rapid, high-resolution genomic analysis identifies submicroscopic copy number variations (CNVs), including deletions or duplications of DNA segments. Chromosomal microarray analysis (CMA) has demonstrated the ability to identify harmful copy number variations (CNVs) in 10-20% of persons with autism spectrum disorder (ASD) [11]. 23 In patients with autism spectrum disorder who exhibit intellectual disability, dysmorphic features, or other congenital anomalies, the diagnostic yield is elevated. CNVs related to ASD, including 16p11.2, 22q11.2, and 15q13.3, are frequently identified, and investigations persist to improve clinical interpretation. WES sequences the protein-coding exons of the genome. It can identify single-nucleotide variants (SNVs), small insertions/deletions, and copy number variations (CNVs) that chromosomal microarray analysis (CMA) overlooks or fails to detect due to its threshold limitations.

Whole exome sequencing (WES) serves as a secondary genetic assessment for autism spectrum disorder (ASD) when chromosomal microarray (CMA) results are negative and there is a clinical suspicion of a genetic etiology [13]. Whole exome sequencing diagnostic yields in autism spectrum disorder datasets from this period range from 15% to 30%, generally identifying de novo pathogenic mutations. Whole exome sequencing (WES) discovered more than 230 new genes associated with autism spectrum disorder (ASD).

Whole Genome Sequencing (WGS)

This involves sequencing the complete genome, encompassing both coding and non-coding regions. This method identifies 27 single-nucleotide variants (SNVs), small insertions and deletions, copy number variations (CNVs), and intricate structural alterations (ResearchGate, 2025a). Notwithstanding its elevated expense and the complexity of data analysis, whole genome sequencing (WGS) is gaining traction in autism spectrum disorder (ASD) research and treatment [14]. Whole Genome Sequencing (WGS) can identify intricate structural changes that Chromosomal Microarray Analysis (CMA) and Whole Exome Sequencing (WES) cannot, hence improving diagnostic efficacy in instances when traditional tests are inadequate (2021-2025) (ResearchGate, 2025a). Research is being conducted on the ability to identify alterations in regulatory non-coding regions.

Emerging Diagnostic Technologies (2020–2025)

Research is being conducted on AI and ML to enhance the accuracy, efficiency, and accessibility of ASD diagnostics. 28 Applications from 2020 to 2025 encompass the identification of digital biomarkers for autism spectrum disorder through the analysis of facial features, eye-tracking patterns, vocalizations, movement data, and biological data. AI methodologies are being devised to assess home videos for subtle behavioral cues, enhance diagnostic uniformity, and minimize diagnostic latency.

Telemedicine

The utilization of remote diagnostic examinations, sometimes using digital tools and virtual platforms, has surged significantly since 2020 as a result of the COVID-19 pandemic. Protocol-guided video recordings and parental interviews enhance diagnostic validity and consistency for disadvantaged populations relative to in-person assessments [15]. Tele-ASD-PEDS is a validated remote assessment for toddlers.

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Cytogenetic Abnormalities in Autism Spectrum Disorder: Foundational Methods

Conventional Karyotyping

Historically, traditional karyotyping offered initial insights into substantial chromosomal abnormalities (often >5-10 Mb). Despite its poor resolution, it remains pertinent for identifying significant structural or numerical alterations [16]. Abnormality Type: Aneuploidies (Trisomy 21), extensive translocations, inversions, and deletions/duplications. Chromosomal Location and Size: These anomalies are, by definition, substantial and may affect any chromosome. Ongoing Significance (2021-2025): Recent guidelines confirm the importance of karyotyping in instances exhibiting distinct dysmorphic characteristics or suspected syndromic disorders that other tests do not adequately elucidate, although its diagnostic yield for isolated ASD is typically minimal, generally below 5% [11]. Its principal function during this phase is generally to augment higher-resolution tests rather than to act as a primary screening instrument

Fluorescence In Situ Hybridization (FISH)

Fluorescence In Situ Hybridization (FISH) is a precise molecular cytogenetic method that uses fluorescent DNA probes to identify certain sequences [17]. It enhanced the resolution from around 50 kilobytes to several megabytes. Abnormality Type: Targeted microdeletions and microduplications. Chromosomal Position and Dimensions: Ongoing Application (2021-2025): The application of FISH is predominantly for the confirmation of clinically suspected, clearly delineated microdeletion/microduplication syndromes (22q13.3 deletion [Phelan-McDermid syndrome], 15q11-q13 duplications) or for elucidating ambiguous results from extensive genomic analyses [17]. It is not a comprehensive screening instrument but a valuable confirmatory or diagnostic method.

High-Resolution Genomic Screening: The Gold Standard

Chromosomal Microarray Analysis (CMA) / aCGH

Chromosomal Microarray Analysis (CMA), frequently using Comparative Genomic Hybridization (aCGH) and SNP arrays, has established itself as the preferred initial genetic test for individuals with ASD, developmental delay, or intellectual disability from 2021 to 2025 [18]. Its capacity to identify CNVs throughout the entire genome with resolutions as fine as tens of kilobases has transformed ASD genetics.

Abnormality Type: Genome-wide identification of submicroscopic deletions and duplications (CNVs). CMA reliably detects pathogenic CNVs in roughly 10-20% of ASD cases, with increased detection rates (up to 25%) in people exhibiting co-occurring intellectual disability or other syndromic characteristics. This period has enhanced our comprehension of both established and novel recurrent CNV regions: 1 2 deletion/duplication: Continues to be one of the most commonly detected and well-replicated copy number variation regions. Deletions (about 600 kb) correlate with macrocephaly, obesity, and heightened ASD risk, whereas duplications (around 600 kb) are associated with microcephaly, underweight, and ASD. 22q11 2 deletion syndrome (about 3 Mb) are significantly correlated with autism spectrum disorder (ASD), intellectual incapacity, and several mental comorbidities, with consistent findings reported during this timeframe (Bassett et al., 2021). The 15q13.3 deletion, approximately 1.5 Mb in size and frequently. CHRNA7, remains a significant risk factor for autism spectrum disorder, intellectual disability, and epilepsy deletion/duplication: These copy number variations (CNVs), ranging from hundreds of kilobases to several megabases linked to several neurodevelopmental abnormalities, including autism spectrum disorder (ASD). Copy number variations (CNVs) associated with synaptic genes: Deletions and duplications impacting genes essential for synaptic function, including SHANK2 (11q13.2), SHANK3 (22q13.3), and NRXN1 (2q16.3), have been consistently validated and further characterized, underscoring their pivotal role in the pathogenesis of ASD [13] utilizing extensive population datasets and functional research [13].

Next-Generation Sequencing (NGS) and CNV

The rapid advancement and enhanced accessibility of Next-Generation Sequencing (NGS) technologies, especially Whole Exome Sequencing (WES) and Whole Genome Sequencing (WGS), have profoundly influenced CNV discovery in autism spectrum disorder (ASD). Although largely recognized for identifying single-nucleotide variations (SNVs) and minor insertions/deletions, their functionalities also encompass copy number variation (CNV) detection, frequently complementing or even exceeding chromosomal microarray analysis (CMA) for specific variant types.

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Whole Exome Sequencing (WES)

WES focuses on protein-coding areas, providing excellent sensitivity for single-nucleotide variants (SNVs) and minor insertions and deletions (indels), while also possessing considerable proficiency in copy number variation (CNV) identification. Abnormality Type: SNVs, minor indels, and CNVs, encompassing those that may be intragenic or beneath CMA resolution.

Utility (2021-2025)

WES has solidified its position as a secondary diagnostic tool for ASD, particularly in instances of negative CMA results [11]. Research from this period regularly indicates diagnostic yields for whole exome sequencing (WES) between 15-30% in autism spectrum disorder (ASD) populations, frequently uncovering de novo pathogenic single-nucleotide variants (SNVs) and copy number variations (CNVs) (ResearchGate, 2025). WES has identified numerous de novo CNVs that are smaller and less recurrent than those usually detected by CMA, suggesting a wider array of implicated genes [11]. The incorporation of advanced CNV calling algorithms into WES pipelines has enhanced their capacity to identify these structural variants.

Whole Genome Sequencing (WGS)

WGS offers the most exhaustive perspective of the genome, identifying nearly all forms of genetic variants. **Abnormality Classification**: All forms of genetic variations, encompassing single-nucleotide variants (SNVs), tiny insertions and deletions (indels), and intricate structural variants (including copy number variations, CNVs) in both coding and non-coding regions (Figure 5).

Utility (2021-2025): Although not yet often employed as a primary option due to financial constraints and complexities in data interpretation, whole genome sequencing (WGS) is progressively utilized in research and certain specialized therapeutic environments for autism spectrum disorder (ASD) [19]. Research throughout this era underscores WGS's enhanced capacity to identify intricate structural variations and CNVs in non-coding or GC-rich areas that may be overlooked by CMA or WES [19]. The diagnostic yield is anticipated to significantly improve beyond whole exome sequencing, especially in difficult instances when other tests produce negative results. The years 2021-2025 have witnessed intensified endeavors to develop clinical pipelines for whole genome sequencing (WGS), with its extensive implementation expected to substantially enhance the copy number variation (CNV) landscape in autism spectrum disorder (ASD).

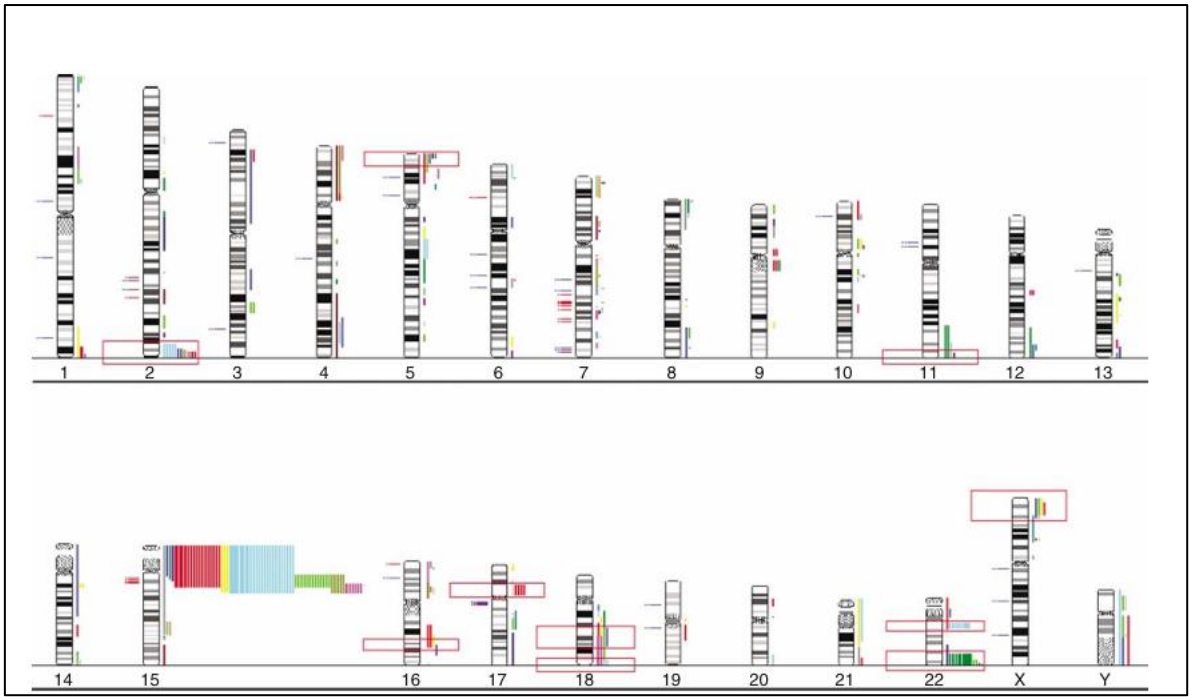

Figure 5: Comprehensive examination of all Cytogenetic Regions of Interest (CROIs) linked to the autism phenotype throughout the human genome.

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On the left side of each chromosome, linkage findings (LOD ≥ 2.0) are represented by blue dashed lines, while significant association findings ($P < 0.05$) are shown by red solid lines. The CROIs are depicted by colored bars on the right side. Adjacent bars of identical hue signify the same CROI documented in several case reports, while thicker bars denote more than two cases within a single case report. Red boxes denote probable novel regions where over four case reports converge at the same locus, absent prior linkage and/or association studies.

Conclusion

This study delineates the research focal points and potential trends of cytogenetics in the diagnosis of autism. Cytogenetics holds significant potential for enhancing the diagnosis and treatment of autism. Nevertheless, it is imperative to collect extensive data over an extended period to confirm their efficacy and safety. Further illustrated its ability to deliver the most extensive genomic perspective, facilitating the identification of complex structural variants and copy number variations (CNVs). These findings collectively underscore the significant progress in clarifying the genetic foundations of ASD, propelled by more advanced cytogenetic and genomic methodologies. This study will assist pertinent researchers, clinicians, and governmental bodies in comprehending the latest advancements in cytogenetics related to Autism and identifying optimal applications of cytogenetics in future clinical practice for societal advantage.

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