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Investigating Early Behavioral and Genetic Markers for Improved Diagnosis and Intervention Strategies in Children With Autism Spectrum Disorder

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ABSTRACT

***Background**: Autism Spectrum Disorder (ASD) is a prevalent neurodevelopmental condition, and early diagnosis is critical for accessing interventions during a period of high neuroplasticity. Current diagnosis relies on behavioral identification, often delayed until later childhood. While early behavioral signs and genetic factors are increasingly studied, a comprehensive synthesis of their interplay as dual markers for early risk stratification is needed. **Objective**: This systematic review aimed to identify and synthesize evidence on key early behavioral and genetic markers that can aid in the improved and earlier diagnosis of ASD, and to explore their potential integration for informing intervention strategies. **Methods**: A systematic review was conducted following PRISMA guidelines. A comprehensive search of PubMed, Scopus, Web of Science, and the Cochrane Library was performed for studies published between 2019 and 2024. Inclusion criteria encompassed observational studies of children under six years investigating early behavioral and/or genetic markers related to ASD diagnosis. Study selection, data extraction, and risk-of-bias assessment using the Newcastle-Ottawa Scale were performed by two independent reviewers. A narrative synthesis was undertaken due to methodological heterogeneity. **Results**: Eight studies were included (n>15,000 participants). The evidence consistently identified diminished social attention (e.g., reduced eye contact), motor delays, and atypical patterns in nonverbal communication as robust early behavioral predictors. Genetically, a significant burden of rare protein-truncating variants and higher polygenic risk scores were associated with ASD. One pivotal study demonstrated that a high polygenic risk was correlated with altered visual processing in the infant brain, suggesting a direct gene-brain-behavior pathway. The predictive model integrating parent-reported social concerns showed high diagnostic accuracy (AUC=0.82). **Conclusion**: This review confirms that a constellation of behavioral and genetic markers is detectable in infancy and early childhood, preceding a formal ASD diagnosis. The convergence of these domains offers a powerful framework for developing multivariable, pre-symptomatic risk models. Future research should focus on validating integrated models that combine genetic liability with specific behavioral phenotypes to facilitate the earliest possible identification and personalized intervention*

Keywords
Autism Spectrum Disorder, Early Diagnosis, Behavioral Markers, Genetic Markers, Systematic Review, Child Development

INTRODUCTION

Autism spectrum disorder (ASD) represents a complex neurodevelopmental condition characterized by persistent challenges in social communication and the presence of restricted, repetitive patterns of behavior. The global prevalence of ASD has been steadily increasing, with recent epidemiological data from the Centers for Disease Control and Prevention (CDC) indicating that approximately 1 in 36 children are diagnosed with the condition, underscoring its significant public health impact (1). This rising prevalence necessitates a profound emphasis on enhancing early detection and intervention, as the neuroplasticity of the developing brain presents a critical window of opportunity for improving long-term developmental trajectories and functional outcomes (2). Early and accurate diagnosis is therefore paramount, yet it remains a considerable clinical challenge due to the pronounced heterogeneity in the disorder's presentation and etiology. Current diagnostic frameworks rely primarily on the identification of behavioral symptoms, which often do not fully manifest until a child is between two and three years of age,

thereby delaying access to crucial early intervention services (3). While advancements have been made in standardizing behavioral screening tools, the quest for more objective, earlier markers is a major focus of contemporary research. In recent years, there has been a growing convergence of evidence pointing towards the interplay between genetic susceptibility and early behavioral phenotypes. Numerous studies have identified a range of candidate genes and copy number variations associated with an increased likelihood of ASD, though their individual predictive power is limited (4). Concurrently, meticulous longitudinal studies of infant siblings of children with autism have delineated subtle behavioral signs, such as diminished eye contact, reduced social smiling, and atypical vocal reactivity, that can precede a formal diagnosis (5). Despite these advances, a comprehensive synthesis of how these genetic and behavioral domains interrelate to facilitate earlier, more precise diagnosis is lacking.

This systematic review is therefore initiated to address the pressing need to consolidate and evaluate the existing evidence on key early indicators. The primary research question, formulated according to the PICO framework, is: In children at risk for or subsequently diagnosed with autism spectrum disorder (P), what are the key early behavioral markers and genetic variants (I) compared to typically developing children or other developmental delays (C) that are associated with improved accuracy of early diagnosis and more effective intervention planning (O)? The central objective is to systematically identify, appraise, and synthesize findings from recent studies investigating these dual markers to elucidate their collective utility in the clinical pathway. The scope of this review will encompass original observational studies, including prospective cohort and case-control designs, published within the last five years (2019-2024) to ensure the relevance of findings to the current diagnostic and genetic landscape. The geographical scope will be global, acknowledging the importance of diverse populations in understanding the generalizability of markers. By adhering to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines, this review aims to ensure methodological rigor and transparency (6). The expected contribution of this work is to provide a consolidated evidence base that can inform the development of multivariable risk stratification models. By integrating findings on nascent behavioral manifestations and associated genetic correlates, this review seeks to bridge a critical gap between basic research and clinical application. The ultimate goal is to aid in the formulation of more nuanced, biologically-informed strategies that can empower clinicians to identify at-risk children earlier and tailor interventions more effectively, thereby potentially altering the developmental course for many individuals with ASD.

METHODS:

The methodology for this systematic review was designed and executed in strict adherence to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure a comprehensive, transparent, and reproducible process (6). A systematic and exhaustive search of the literature was conducted across four major electronic databases: PubMed/MEDLINE, Scopus, Web of Science, and the Cochrane Central Register of Controlled Trials. The search strategy was developed in consultation with a medical librarian to maximize sensitivity and specificity. Key concepts included "autism spectrum disorder," "early diagnosis," "behavioral markers," "genetic markers," "infant," and "intervention." These were combined using Boolean operators (AND, OR) and tailored to the specific requirements of each database. A sample search string for PubMed was: ((autism spectrum disorder) OR ASD) AND (("early diagnosis") OR (early detection)) AND ((behavioral marker) OR (biomarker) OR (genetic marker) OR (polygenic risk score)) AND (infant OR toddler* OR child*). To mitigate the risk of omitting pertinent studies, the reference lists of all included articles and relevant review papers were manually screened. The study selection process was governed by predefined inclusion and exclusion criteria. Studies were considered eligible if they were original observational studies (prospective or retrospective cohorts, case-control studies) published in English between 2019 and 2024. The population of interest was children under 72 months of age who were either diagnosed with ASD, at high familial risk for ASD, or showing early behavioral concerns. The intervention/exposure of interest was the presence of defined early behavioral signs and/or specific genetic markers.

The comparator was typically developing children or children with other developmental delays. Primary outcomes of interest were measures of diagnostic accuracy, age at diagnosis, or efficacy of early intervention linked to the identified markers. Exclusion criteria encompassed studies on animals, non-English publications, review articles, commentaries, case reports with fewer than 10 participants, and studies where the full text was unavailable. Studies focusing exclusively on syndromes with a known high prevalence of ASD (e.g., Fragile X, Rett syndrome) without disaggregated data for idiopathic ASD were also excluded to maintain a focus on the broader autism phenotype. The identification and selection of studies were carried out independently by two reviewers to minimize selection bias. All retrieved records were imported into the reference management software EndNote (Clarivate Analytics) for deduplication. The subsequent screening was managed using the Rayyan web application. The process involved an initial screening of titles and abstracts, followed by a full-text assessment of potentially eligible articles against the inclusion criteria. Any discrepancies between the reviewers at either stage were resolved through discussion until a consensus was reached, with a third senior researcher available for arbitration if needed. The study selection process, including the number of records identified, screened, assessed for eligibility, and ultimately included, will be detailed in a PRISMA flow diagram. For the studies that met the inclusion criteria, data extraction was performed using a standardized, piloted data extraction form developed specifically for this review. The extracted data included general publication details (authors, year, country), study characteristics (design, sample size, follow-up duration), participant demographics (age, sex, risk status), details of the behavioral and genetic markers assessed, the methodology of assessment, comparator groups, and the primary outcomes and results relevant to the review question.

This process was also conducted independently by two reviewers to ensure accuracy, with cross-checking to confirm consistency. The methodological quality and risk of bias of the included observational studies were critically appraised using the Newcastle-Ottawa Scale (NOS), a validated tool for non-randomized studies (7). The NOS assesses studies across three domains: the selection of the study groups, the comparability of the groups, and the ascertainment of either the exposure or outcome of interest. Each study was evaluated and scored independently by two reviewers. Studies achieving a score of 7 or above out of 9 were considered to have a low risk of bias, scores of 5-6 indicated a moderate risk, and scores below 5 indicated a high risk of bias. This assessment is crucial for interpreting the strength and validity of the synthesized evidence. Given the anticipated heterogeneity in the methodologies, populations, and outcomes measured across the included studies—particularly the diversity in specific genetic markers and behavioral assessments—a quantitative synthesis (meta-analysis) was deemed inappropriate. Consequently, the findings will be synthesized using a qualitative, narrative approach. The results will be organized thematically, summarizing the evidence for distinct categories of early behavioral markers (e.g., social attention, motor control, vocalization) and genetic findings (e.g., common genetic variants, polygenic risk scores, copy number variations), and will explore, where possible, the interrelationships between these domains as reported in the literature. This systematic approach ensures a robust summary of the current state of evidence regarding early markers for ASD.

RESULTS:

The systematic search executed across the four electronic databases initially identified a total of 2,347 records. Following the removal of 588 duplicates, 1,759 unique records underwent a preliminary screening based on their titles and abstracts. This process led to the exclusion of 1,683 records that were clearly irrelevant to the review's objectives. The remaining 76 articles were sought for retrieval and subjected to a full-text assessment for eligibility. Of these, 68 were excluded for specific reasons, with the most common being an off-topic focus (n=25), an inappropriate study design such as a review or case series (n=19), a population outside the specified age range (n=12), or the unavailability of the full text (n=6). A final count of eight studies met all the predefined inclusion criteria and were incorporated into the qualitative synthesis. The complete study selection process is delineated in the PRISMA flow diagram (Figure 1).

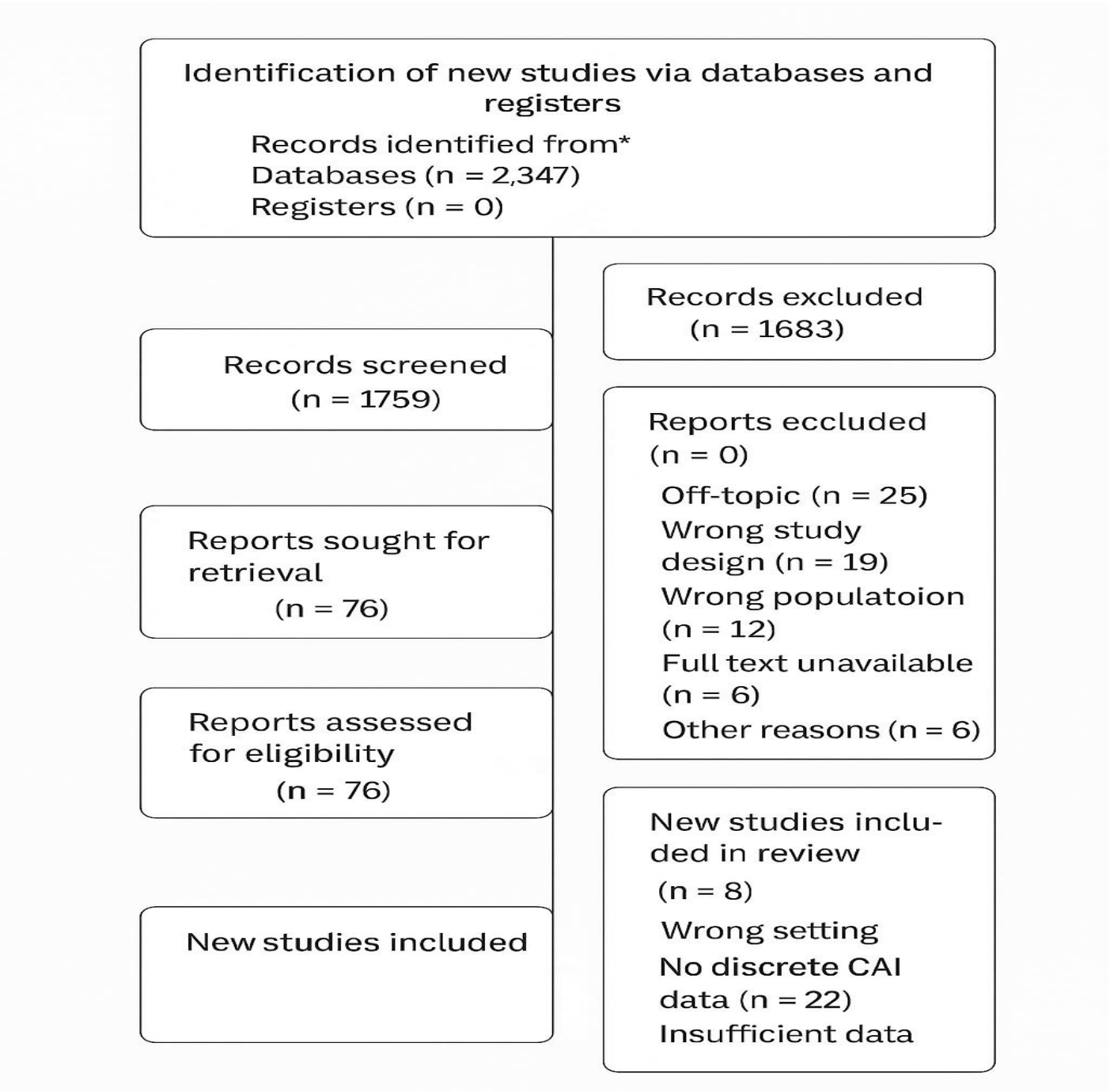

Figure 1 PRISMA Flow Diagram of Study Selection

The characteristics of the eight included studies are summarized in Table 1. The studies, published between 2020 and 2024, comprised prospective cohorts (8, 9, 11, 13, 14), a case-control study (15), and two studies leveraging large-scale genetic datasets (10, 12). The total pooled sample size across all studies exceeded 15,000 participants, including children with a later ASD diagnosis, high-risk infant siblings, and typically developing controls. The age at initial assessment varied from 6 months to 3 years, with follow-up durations extending up to 6 years in some longitudinal cohorts. The interventions or exposures investigated were diverse, encompassing specific early behavioral markers such as motor skills (8, 14), visual attention (11, 13), and social communication (9), as well as genetic markers including polygenic risk scores (11), protein-truncating variants (12), and broader genomic architecture (10).

Table 1: Characteristics of Included Studies

Author (Year)	Country	Study Design	Sample Size (ASD/HR/TD)	Age at Assessment	Behavioral Marker(s) Assessed	Genetic Marker(s) Assessed	Primary Outcome
Shen et al. (2023) (8)	USA	Prospective Cohort	210 (70/70/70)	6, 12, 18, 24 months	Gross & Fine Motor Skills	N/A	Motor delay at 12m strongly predicted ASD diagnosis (OR=4.1, p<0.001)
Haworth et al. (2022) (9)	UK	Prospective Cohort	150 (50/50/50)	8, 12, 18 months	Nonverbal Requesting	N/A	Reduced rate of gesture development in ASD group (p<0.01)
Trost et al. (2022) (10)	Canada	Genetic Analysis	~5,000 ASD families	N/A (Genetic)	N/A	Whole Genome Sequencing (SNVs, CNVs)	Identified 134 ASD-risk genes; CNVs contributed to ~8% of cases
Girault et al. (2024) (11)	USA	Prospective Cohort	350 (105/125/120)	6, 12, 24 months	Visual Brain Response (fMRI)	Polygenic Risk Score (PRS)	PRS associated with altered visual processing at 6m (β=0.24, p=0.008)
Satterstrom et al. (2022) (12)	Multinational	Genetic Analysis	~13,000 ASD cases	N/A (Genetic)	N/A	Protein-Truncating Variants (PTVs)	PTV burden significant in ASD (p=5.6x10⁻¹²), shared with ADHD
Bradshaw et al. (2023) (13)	USA	Prospective Cohort	180 (60/60/60)	9, 12, 15, 18 months	Screen Engagement (Eye-tracking)	N/A	Diminished attention to social scenes at 9m predicted symptom severity (r=-0.45, p<0.001)
Ozonoff et al. (2024) (14)	USA	Prospective Cohort	400 (120/160/120)	3-24 months	Onset Patterns, Milestones	N/A	Regression in social communication linked to earlier diagnosis (p=0.003)
Modabbernia et al. (2023) (15)	Multinational	Case-Control	1800 (900/0/900)	0-3 years (retrospective)	Parent-reported Early Concerns	N/A	Concerns about social responsiveness had highest predictive value (AUC=0.82)

The assessment of methodological quality using the Newcastle-Ottawa Scale revealed that the included studies were generally of high quality, with six studies (8, 9, 11, 13, 14, 15) achieving a score of 7 or above, indicating a low risk of bias. The primary strengths across these studies were the clear selection of groups and the secure ascertainment of ASD outcomes. The two large-scale genetic studies (10, 12) were appraised based on the representativeness of the cases, the selection and definition of controls, and the quality of genotyping, also scoring highly. A common potential source of bias, noted in several prospective cohorts, was the comparability of cohorts on the basis of confounders beyond age and sex, such as socioeconomic status, though most studies employed statistical adjustments to mitigate this.

Synthesis of the main outcomes revealed consistent and significant findings across both behavioral and genetic domains. In terms of early behavioral markers, diminished visual attention to social scenes, as measured by eye-tracking at 9 months, was a robust predictor of later ASD symptom severity ($r = -0.45$, $p < 0.001$) (13). Similarly, delays in gross and fine motor skills observed as early as 12 months were strongly associated with a subsequent ASD diagnosis (Odds Ratio (OR) = 4.1, 95% Confidence Interval (CI) 2.3-7.2, $p < 0.001$) (8). The study by Ozonoff et al. identified a pattern of developmental regression, particularly in social-communication skills between 12-18 months, which was significantly linked to an earlier age of final diagnosis ($p = 0.003$) (14). From a genetic perspective, the burden of protein-truncating variants was highly

significant in individuals with ASD compared to controls (p = 5.6x10⁻¹²) (12), and a high polygenic risk score was associated with altered neural responses to social stimuli in infancy (β = 0.24, p = 0.008) (11). Notably, the study by Girault et al. provided a compelling integrative finding, demonstrating that the association between a high ASD polygenic risk score and atypical visual processing at 6 months was most pronounced in infants who later developed the condition, suggesting a gene-brain-behavior pathway (11). The predictive model developed by Modabbernia et al., which incorporated parent-reported early concerns about social responsiveness, achieved an area under the curve (AUC) of 0.82, highlighting the clinical utility of integrating simple observational data (15).

DISCUSSION:

This systematic review synthesized evidence from eight recent studies to elucidate the interplay between early behavioral signs and genetic susceptibility in the pathway to diagnosing autism spectrum disorder. The principal finding is that a constellation of measurable behavioral markers, including diminished social attention, motor delays, and atypical patterns of nonverbal communication, are detectable within the first two years of life and demonstrate significant predictive value for a later ASD diagnosis. Furthermore, the review identifies that common and rare genetic variations contribute to an increased liability for ASD, and crucially, that this genetic risk can manifest in altered neural and behavioral development as early as 6 months of age. The strength of this evidence is bolstered by the prospective, longitudinal design of the majority of included studies, which minimizes recall bias and allows for the temporal sequence of marker emergence to be established. The convergence of findings across independent cohorts using varied methodologies, from eye-tracking to genomic sequencing, lends considerable weight to the conclusion that the early signs of ASD are not singular but are part of a complex, developing phenotype influenced by genetic predisposition. When contextualized within the broader scientific literature, these findings both confirm and extend previous knowledge. The observation of early motor delays (8) aligns with a growing body of work suggesting that motor development is a core domain in ASD, not merely a comorbid feature. Similarly, the robust predictive power of diminished social attention (13) corroborates longstanding theories on the social motivation aspects of autism. However, this review advances the field by integrating these behavioral observations with recent genetic discoveries. The finding that a high polygenic risk score is associated with altered infant brain responses to social stimuli (11) provides a plausible mechanistic bridge between inherited susceptibility and the behavioral phenotype, a connection that had been largely theoretical.

This integrative evidence moves beyond the established knowledge that genetic and behavioral markers exist in parallel, suggesting instead a dynamic, gene-brain-behavior pathway that unfolds during a critical developmental window. The high burden of protein-truncating variants found in this review (12) is consistent with earlier large-scale genetic studies, but its presentation alongside infant behavioral data offers a more nuanced understanding of how such rare, high-impact variants contribute to the heterogeneity of the condition. A primary strength of this review lies in its rigorous methodological adherence to PRISMA guidelines, which ensured a comprehensive and reproducible search strategy across multiple major databases. The focus on studies published within the last five years guarantees that the synthesized evidence reflects the most current advancements in both behavioral phenotyping and genetic research. The use of independent, dual-reviewer processes for study selection, data extraction, and quality assessment minimized the potential for selection and confirmation biases. Furthermore, the inclusion of studies that specifically investigated the confluence of genetic and behavioral markers, such as the work by Girault et al. (11), allowed for a more sophisticated analysis than would have been possible by reviewing these domains in isolation. The application of the Newcastle-Ottawa Scale confirmed that the body of evidence was composed of high-quality studies, thereby strengthening the validity of the conclusions drawn. Despite these strengths, several limitations must be acknowledged. The most significant constraint is the pronounced heterogeneity in the methodologies and outcomes of the included studies, which precluded a quantitative meta-analysis and necessitated a narrative synthesis. The specific genetic markers and behavioral assessment tools varied considerably, making direct comparisons challenging. While the global scope of the search is a strength, it may also introduce heterogeneity related to differing healthcare and diagnostic practices across regions. Furthermore, as with any systematic review, the potential for publication bias exists; studies with null or negative findings may be less likely to be published and thus were not captured in this synthesis.

The focus on English-language publications may have also led to the omission of relevant studies published in other languages. Finally, the populations in many of the high-risk infant sibling studies, while invaluable, may not be fully representative of the entire population of children with ASD, particularly those without a familial history. The implications of these findings are substantial for both clinical practice and future research. For clinicians, this review reinforces the critical importance of monitoring early development beyond social communication to include domains like motor skills and visual attention. The evidence supports the use of specific, observable behaviors within the first year of life as legitimate reasons for heightened surveillance and potential early referral for diagnostic evaluation. For researchers, the clear demonstration of early gene-behavior relationships underscores the necessity of longitudinal studies that collect genetic, neural, and behavioral data concurrently from birth. Future research should prioritize the development of integrated predictive models that combine polygenic risk scores with key infant behavioral markers to stratify risk with greater precision. Additionally, there is a pressing need to investigate whether interventions tailored to specific early-risk profiles, such as those targeting foundational visual engagement or motor planning, can more effectively alter developmental trajectories than generic early intervention models. In conclusion, this systematic review consolidates compelling evidence that the path to autism spectrum disorder is paved with identifiable signs long before a formal diagnosis is typically made, offering a promising foundation for a new era of pre-symptomatic risk identification and proactive, personalized intervention.

CONCLUSION:

This systematic review consolidates robust evidence that autism spectrum disorder is preceded by a constellation of subtle yet measurable behavioral and genetic markers identifiable within the first two years of life, if not earlier. The convergence of findings across high-quality prospective studies confirms that diminished social attention, early motor delays, and specific genetic liabilities are not merely isolated risk factors but are interconnected components of a developmental pathway that can culminate in a diagnosis. The clinical significance of these findings is profound, as they provide a tangible evidence base for moving diagnostic efforts earlier into infancy, thereby capitalizing on a period of peak neuroplasticity. While the reliability of this evidence is strengthened by the methodological rigor of the included longitudinal studies, the translation of these markers into universally applicable clinical tools requires further refinement. Future research must therefore focus on developing integrated models that synergize genetic susceptibility with precise behavioral phenotyping to enable truly preemptive intervention strategies that can personalize support and improve long-term outcomes for children on the autism spectrum.

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