World Journal of Advanced Engineering Technology and Sciences
eISSN: 2582-8266
Cross Ref DOI: 10.30574/wjaets
Journal homepage: https://wjaets.com/

(REVIEW ARTICLE)

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Autism Spectrum Disorder: Etiology and Gender Differences in STEM Education Choices

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World Journal of Advanced Engineering Technology and Sciences, 2025, 17(01), 454-464

Publication history: Received on 22 September 2025; revised on 27 October 2025; accepted on 30 October 2025

Article DOI: https://doi.org/10.30574/wjaets.2025.17.1.1445

Abstract

The term Autism refers to a group of developmental disorders characterized by deficits in social interaction and communication, as well as a markedly restricted repertoire of activities and interests (American Psychiatric Association, 2000). While 6–10% of individuals with Autism present with combined neurodevelopmental syndromes, for the majority the cause remains unknown. It is likely that multiple etiological factors contribute to this symptomatic constellation. However, the complex interaction between genetic makeup, hormonal activity, and environmental influences enables researchers and clinicians to better understand the causality of the autism spectrum and its differentiation between the two sexes. A combination of social difficulties and sensory sensitivities, together with significant impairments in executive functioning and the protective attitudes of families, appears clinically to lead higher-functioning individuals with autism to perform below the potential suggested by their normal intelligence. The perception that men are inherently more talented and interested in science is a widespread cultural stereotype. Research indicates that interest in academic studies and careers in STEM fields is largely shaped by environmental influences, as social, psychological, and sociocultural perspectives offer clearer explanations regarding gender differences.

Keywords: Autism; STEM; Genter; Inclusion; Pedagogy; Neurogenetics

1. Introduction

Autism Spectrum Disorder (ASD) is a neurodevelopmental condition characterized by social deficits, repetitive behaviors, and a lack of empathy. Its significant genetic heritability and potential comorbidities often lead to diagnostic and therapeutic challenges. ASD is diagnosed more frequently in males, with a ratio of 4:1, suggesting a possible bias in autism research toward males and a risk of underdiagnosis in females.

Synaptic gene function (including the balance between excitation and inhibition) is influenced by gene mutations, which significantly affect the cognitive and behavioral manifestations of the autism spectrum. The role of small RNAs (miRNAs), and particularly a novel mutation in miRNA-873, impacts a set of core synaptic genes—such as neurexin, neuroligin, and density-related proteins—associated with autism spectrum pathology.

Autism, or Autism Spectrum Disorder (ASD), is defined in terms of abnormalities in social and communicative development, combined with pronounced repetitive behavior and limited imagination (APA, 1994). It is characterized by deficits in social communication, as well as repetitive and restricted movements and behaviors (Tager-Flusberg, 2015; Simmons, Paul, & Volkmar, 2014; Pang, 2016). Asperger Syndrome (AS) is defined as a condition in which an individual meets the same criteria as autism but without a history of delays in cognitive or language development, and

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without meeting the criteria for Pervasive Developmental Disorder (PDD) (World Health Organization, 1994). Language delay is defined as the absence of single words by the age of 2, and/or phrases by the age of 3.

Autism is a severe childhood neuropsychiatric condition with a significant genetic basis. There is growing evidence that autism and Asperger syndrome (AS) are of genetic origin, with the strongest support coming from twin and family genetic studies (Bailey et al., 1995; Gillberg, 1991; Bolton & Rutter, 1990; Folstein & Rutter, 1977, 1988). Subsequent twin and family studies (Lauritsen & Ewald, 2001; Rutter, 2000) also provide evidence of a genetic contribution to autism and Asperger syndrome. There is also a hypothesis—still under discussion—that autism and Asperger syndrome lie along a spectrum of social-communication disability, with Asperger syndrome serving as a bridge between autism and typical development (Baron-Cohen, 1995; Frith, 1991; Wing, 1981, 1988). The spectrum concept shifts us away from categorical diagnosis and toward a quantitative approach.

The positive and useful contributions that digital technologies provide to the field of education should be highlighted as a final point. Mobile devices (62-64), a range of ICT apps (65-69), AI & STEM ROBOTICS (70-71), and games (72-73) are some examples of the technologies that enable and improve educational processes including evaluation, intervention, and learning. Additionally, the use of ICTs in conjunction with theories and models of metacognition, mindfulness, meditation, and the development of emotional intelligence (74-87), accelerates and improves educational practices and outcomes, especially for STEM and Gender education.

2. Causality of Autism Spectrum Disorder

Autism Spectrum Disorder (ASD) is a multifaceted neurodevelopmental condition that frequently overlaps or co-occurs with other psychiatric disorders, such as attention-deficit/hyperactivity disorder (ADHD) and obsessive-compulsive behaviors. It encompasses a broad spectrum of symptoms with varying levels of severity, resulting in a wide range of behavioral manifestations and potential comorbidities, including epilepsy, intellectual disability, and gastrointestinal disorders. The absence of reliable biomarkers for ASD complicates both diagnosis and treatment.

Historically, intellectual disability was strongly associated with autism. However, toward the end of the millennium, it became established that intellectual disability is not a defining characteristic of ASD. Scientific consensus has identified the core features of autism spectrum disorders as difficulties in social interaction, repetitive behaviors, and challenges in recognizing and responding to emotions (American Psychiatric Association, 2013). Intellectual disability is now considered an important comorbidity, occurring in approximately 40% of individuals with autism (Hajri et al., 2022).

Regarding autism prevalence rates in Europe and the United States, during the 1960s and 1970s the rates were estimated at 2–4 per 10,000 children, leading to the perception of autism as a rare childhood disorder (Lotter, 1966; Treffert, 1970). This was followed by a dramatic increase in diagnosed cases, with data from the Centers for Disease Control and Prevention (CDC) showing prevalence rising from 1 in 150 children in 2000 to a concerning 1 in 36 by 2020. Most children are diagnosed after the age of four, although signs are typically evident by the age of two (Gibbs et al., 2019).

These particularly alarming rates have, over the last five years, heightened awareness among both scientists and society regarding the autism spectrum. This awareness has reinforced the understanding that ASD is not a uniform disorder but varies significantly across individuals. Only through an in-depth understanding of the condition can effective therapeutic interventions be developed and strategies designed to meaningfully support individuals on the autism spectrum.

Despite decades of extensive research, a definitive causal factor for Autism Spectrum Disorder remains elusive, highlighting the complexity of its origins. Nevertheless, the prevailing view is that genetics play a pivotal role, with current estimates suggesting that heritability accounts for approximately 80% of the risk (Bai et al., 2019). While twin studies provide strong evidence for the genetic basis of autism, the wide variability in observed genetic mutations further complicates its etiology (Monteiro & Feng, 2017).

No single gene mutation has been identified as the primary causal factor for autism (Abrahams & Geschwind, 2008). Instead, a wide range of distinct mutations and genetic variants across numerous genes contributes to the heterogeneity of autism, making direct comparisons between individuals challenging. Consequently, the genetic landscape of autism is characterized by broad variability, reflecting the wide-ranging impact of these mutations on neurodevelopment and functioning. The fact that more than 1,000 genes have been associated with autism underscores the complexity and heterogeneity of the condition (Geschwind & State, 2015). Recent advances in gene-screening techniques have identified approximately 1,404 genes and 2,274 copy number variation (CNV) loci linked to ASD (Basu et al., 2009).

These genetic mutations can vary greatly in scale, ranging from single nucleotide polymorphisms (SNPs) to large segments of DNA containing thousands or even millions of bases, known as CNVs. Research has shown that rare, de novo CNVs occur ten times more frequently in individuals with autism compared to the general population (Marshall et al., 2008; Sebat et al., 2007).

Genetic alterations can lead to changes in DNA sequence or chromosomal structure, subsequently affecting the protein-coding regions of the entire genome. Genome-wide association studies (GWAS) have identified common genetic variants associated with autism (Kim et al., 2008; Sebat et al., 2007), highlighting the importance of critical genes involved in synaptic function and neural connectivity. The strong presence of rare, large CNVs in individuals with autism—particularly those encompassing recognized synaptic genes linked to the disorder (Fregeac et al., 2016)—suggests that disruptions in synaptic function play a pivotal role in shaping the autism phenotype.

2.1. Causality of Autism Spectrum Disorder at the Gender Level

Historically, males have been diagnosed with Autism Spectrum Disorder (ASD) more frequently than females, with a ratio of approximately 4:1 (Elsabbagh et al., 2012; Loomes et al., 2017). Current diagnostic criteria and tools are primarily based on male-centered data, resulting in many women on the spectrum receiving delayed diagnoses or remaining undiagnosed—often because their symptoms do not align with the stereotypical presentation of autism, which has been largely defined through male cases (Kreiser & White, 2014). This androcentric perspective in understanding biology is also widespread in physiological and pharmacological research, as highlighted by Rebecca Shansky (Shansky, 2019).

Autism also manifests differently in females and may present with more subtle social communication differences and fewer overt repetitive behaviors. Behavioral inhibition, involving the prefrontal cortex, the amygdala, and the brain's cingulate gyrus, shows sex-specific differences between females and males (Laine et al., 2024).

Females may exhibit distinct cognitive and behavioral profiles compared to males (Ratto et al., 2018). Combined with their ability to skillfully mask autistic tendencies and behaviors, this complicates autism recognition, often leading to misdiagnoses or underdiagnoses (Kreiser & White, 2014; Ratto et al., 2018).

Moreover, there is growing awareness of the "female protective effect" (FPE) (Robinson et al., 2013), suggesting that females may have a higher genetic threshold for developing ASD (Li et al., 2020). They may require a greater genetic load to manifest behavioral pathology, potentially due to genetic and hormonal factors. The presence of two X chromosomes in females, along with their hormonal environment (notably higher estrogen levels), may confer neuroprotective effects against autism. Additionally, social and environmental factors, including early socialization, may provide females with compensatory tools to counteract subtle imbalances associated with the autism spectrum.

There is a trend toward a decreasing male-to-female ratio in ASD diagnoses, with some studies suggesting a ratio closer to 2:1. According to findings from the Autism and Developmental Disabilities Monitoring (ADDM) Network for 2020, the reported ratio was 3.8:1, closely aligning with the traditionally estimated 4:1 ratio.

However, this adaptive behavior in females—skillfully masking autistic tendencies and behaviors—often results in higher rates of anxiety and depression, stemming from constant social pressures and the ongoing mental effort required to conceal symptoms and meet societal expectations (Mandy et al., 2012; Rincón-Cortés et al., 2019; Shansky, 2020). In contrast, males typically display more overt autistic traits and an externally directed behavioral profile, primarily experiencing challenges in behavioral adjustment, communication difficulties, and distress in noisy environments (de Giambattista et al., 2021). They also exhibit atypical social approaches, social avoidance, and reduced empathy.

Research focusing on females has gained momentum, emphasizing the need for targeted studies. Nevertheless, there is a lack of research examining the impact of gene mutations in females, as genetic studies largely focus on male or mixed-gender samples, limiting understanding of female-specific genetic contributions (Klin et al., 2003; Szatmari et al., 2009; Werling, 2016). These findings underscore the complexity of diagnosing autism across sexes and highlight the necessity for nuanced approaches in both clinical practice and research.

2.2. Sex Neurogenetic Basis of Autism Predisposition

Heritability is a significant factor in autism risk; however, the genetic liability for developing the disorder arises from the cumulative effect of numerous small variants across hundreds to thousands of genes (Grove et al., 2019; Mattheisen, 2022; Ramaswami, 2020). These subtle changes in gene expression can collectively exert a substantial impact on an individual's likelihood of exhibiting autistic traits (Dong et al., 2022; O'Brien et al., 2018; Song et al., 2019). Such

variations in gene expression are critical for determining the pathogenesis and progression of autism, underscoring the importance of related studies for understanding this condition.

In exploring the neurobiology of autism, one compelling causal hypothesis that has gained traction is the "extreme male brain" theory (Baron-Cohen, 2002). This theory posits hyper-masculinization of cognitive and behavioral traits associated with the autism spectrum and suggests that the molecular architecture of the male brain may inherently tilt toward the autistic phenotype. Consequently, a smaller accumulation of genetic and environmental risk factors may be required to trigger autistic characteristics in the male brain compared to the female brain.

This hyper-masculinization appears to be reflected in brain transcription patterns, with the model predicting that baseline transcription in a typical male brain mirrors the transcriptional signatures observed in postmortem brains of individuals with autism. Previous research has revealed that the male bias in autism may be influenced by innate intersexual processes rather than by sex-specific regulation of autism risk genes. Postmortem studies have identified downregulation of neuron- and synapse-related genes and upregulation of microglial and immune-related genes in the brains of individuals with autism compared to controls, indicating subtle but significant differences in gene expression (Gupta et al., 2014; Velmeshev et al., 2019; Willsey et al., 2022).

Analysis of 137 adult brains revealed that 2.6% of all genes expressed in the brain exhibit differential expression levels between males and females (Trabzuni et al., 2013). This variation is evident across all major brain regions, indicating a broad and consistent pattern of sex differences in gene activity, with each analyzed region showing small but significant differences (Trabzuni et al., 2013).

The hippocampus is a critical brain region for learning and memory, involved in the encoding, consolidation, and retrieval of memories—essential cognitive functions for learning. In individuals with autism, significant deficits are observed in spatial reasoning, working memory, and executive functioning. Structural and functional alterations in the hippocampus are consistently reported in individuals with autism (Reinhardt et al., 2020; Won et al., 2012).

A related study analyzing human brain proteomes found that 5.5% of the genes assessed exhibited sex-differentiated expression at both the mRNA and protein levels in the brain, impacting psychiatric and neurological disorders (Wingo et al., 2023). A recent study using large samples of brain tissue observed distinct sex-specific gene expression patterns (Fass et al., 2023). Female brains showed increased expression of genes involved in vascular regulation, immune responses, and zinc homeostasis, whereas male brains exhibited higher expression of genes related to neurotransmitter transporters, ion channels, synaptic signaling, and neural plasticity (Fass et al., 2023). These findings suggest heightened immune system activity in the female brain (Hanamsagar et al., 2017; McCarthy et al., 2017; Trabzuni et al., 2013).

Importantly, disruptions in the expression of genes predominant in the "male brain" may disturb critical cellular and synaptic functions, particularly in males. Studies focusing on differences in cellular states rather than cell number provide valuable insights into the fundamental sex neurogenetic basis of autism predisposition.

Animal studies and animal models can also provide valuable insights into the sex neurogenetic basis of autism predisposition. A study in rats reported a differential expression of up to 1,680 genes in the hippocampus between male and female controls, with 983 genes showing higher expression in males (Biala et al., 2011). This study also found that biological processes such as axonogenesis, synaptogenesis, and synaptic maturation appeared to be more active in males compared to females. In contrast, genes more highly expressed in females were primarily associated with maintenance and neurogenesis (Biala et al., 2011). Findings from animal research can help clarify the complex genetic underpinnings observed in human studies and contribute to a more comprehensive understanding of autism.

2.3. Sex Hormonal Contribution to Autism Predisposition

Focusing on sex-based neural differences, hormonal imbalances—particularly the role of the sex steroids testosterone and estradiol—play a central role in the differential gene expression observed throughout the brain. Testosterone, especially during prenatal development, is critical in establishing numerous physiological sex differences, including physical characteristics, regional brain volume, neurotransmitter levels, cellular differentiation, and neurogenesis (Simerly, 2002). Relevant research suggests a link between higher fetal testosterone levels and the development of autism. Additionally, studies have observed that autistic females tend to exhibit elevated testosterone levels (Auyeung et al., 2009; Knickmeyer & Baron-Cohen, 2006).

The critical phase of fetal development can significantly shape the neurophysiological and behavioral tendencies observed throughout an individual's life on the autism spectrum. Autism-related genes are involved in processes such

as synapse formation, maintenance, and cellular adhesion. Furthermore, autism traits have been associated with abnormalities in steroid hormones (Baron-Cohen et al., 2021; Baron-Cohen et al., 2020; Gillberg et al., 2017; Lai et al., 2015). The unique mechanism and lipid-soluble nature of steroids allow them to directly regulate genes by crossing cell membranes, interacting with their receptors, and directly entering the nucleus. This process adapts cells for specific activities. The ability to control gene activation is critical and explains why certain genes, although present in all cells, are expressed differently between cell types and sexes. For example, genes crucial for muscle function may remain dormant in neurons where they are not needed. Such selective gene activation, largely regulated by various molecules including hormones like testosterone—which tends to be more active in males than in females—leads to distinct physiological and behavioral tendencies in males and females.

The complex interplay between genetic makeup, hormonal activity, and environmental factors helps researchers and clinicians better understand the autism spectrum. Moreover, ASD is more common in children of mothers with polycystic ovary syndrome (PCOS) (Cherskov et al., 2018; Kosidou et al., 2016), as these women generally have higher testosterone levels, including during pregnancy (Jiang et al., 2021; Palomba et al., 2012).

3. Bias in Autism Assessment and Impartiality of ASD Models

The literature highlights significant concerns that assessment tools may function differently depending on the sex of the respondent, which helps explain the historical overrepresentation of males and boys in autism research. Assessment tools may therefore exhibit sex-based bias, resulting in invalid measurements and underestimations of autism prevalence in females and girls (Belcher, Marucha, Vitoratou, Ford, & Zamir, 2023). In psychometrics, this is referred to as measurement bias or non-invariance (non-equivalence) or differential item functioning. Non-invariance in assessment tools can lead to different clinical decisions and complicate conceptual comparisons between groups, as the evaluation is not objective (Belcher, Marucha, Vitoratou, Ford, & Zamir, 2023).

Despite increasing public awareness of autism in females, girls and women continue to receive diagnoses significantly later in life, on average, compared to boys and men. This may partly be due to a different presentation of autistic traits in females, including fewer social communication difficulties and fewer restrictive and repetitive behaviors and interests. Evidence suggests that males may be more likely to exhibit autism due to gene mutations and pronounced structural changes, as well as the involvement of sex-determining chromosomes and hormonal effects. In contrast, females may remain undiagnosed because they experience greater social pressure to “camouflage” and conceal their autistic traits.

Approximately three males are diagnosed with autism for every female; however, the actual sex difference in autism prevalence is likely lower when undiagnosed cases are considered. Missed or delayed diagnoses are concerning given the increased risk of symptomatic psychiatric disorders and suicidal behaviors experienced by individuals with autism. Many adults see multiple healthcare professionals before receiving an autism diagnosis, which occurs more frequently in females. Early diagnosis and support can have a substantial positive impact on quality of life in adulthood and facilitate better psychiatric management of symptomatic mental health difficulties later in life.

To date, limited research has examined measurement non-invariance with respect to sex across different versions of the Autism-Spectrum Quotient (AQ). The Autism-Spectrum Quotient (Baron-Cohen et al., 2001) is a validated self-report measure used to quantify autistic traits in individuals with average or above-average IQ. It is a self-assessment tool designed to measure the extent to which a neurotypical individual exhibits autistic characteristics. The AQ was developed to be brief and easy to administer, consisting of 50 questions across five domains: social skills, attention switching, attention to detail, communication, and imagination.

A recent study (Belcher, Marucha, Vitoratou, Ford, & Zamir, 2023) involving a large sample of adults from the general population of the United Kingdom (5,246 females, 1,830 males, totaling 7,179 participants) found that 20 items consistently exhibited bias favoring males, while 21 items consistently exhibited bias favoring females. Females with autism displayed fewer social communication difficulties compared to males on standard AQ scales, despite exhibiting similar levels of autistic traits in childhood. Furthermore, autistic girls were more likely to engage in reciprocal conversation and age-appropriate imaginative play than boys. This difference may be partly explained by the ways in which girls and boys are socialized, as well as by gender stereotypes often reinforced by caregivers who encourage females to be more social and empathetic.

Although there is consensus across different nationalities regarding autism diagnostic criteria, research suggests that cultural differences may exist in what is considered neurotypical development. Cultural factors can also contribute to variations in social communication, which is a core feature of autism.

It is also important to recognize the disparities in obtaining autism diagnoses among ethnic and racial minority groups. There is an imbalance in diagnosis between high-income countries and low- and middle-income countries due to a lack of culturally appropriate diagnostic tools. Evidence indicates that children from ethnic and racial minority groups are more likely to receive a misdiagnosis or to be diagnosed later compared to white children, due to increased social and cultural stigma, lack of education, and limited access to diagnostic assessments and interventions within these communities.

4. Biological Sex and STEM Education

Despite significant progress toward gender equality, sex-based disparities remain a defining feature of STEM education. Gender gaps continue to influence perceptions of ability and are often cited as evidence of men's innate superiority in STEM fields (Correll, 2001; Nosek et al., 2002; Hyde, 2005).

Recent studies, however, have shown gender parity or a slight female advantage in basic computation and mathematical concept comprehension across all grade levels (Hyde et al., 1990; Hedges & Nowell, 1995). Research also documents female advantages in certain tasks related to spatial abilities (Hyde, 2005; Spelke, 2005; Halpern et al., 2007). Nevertheless, related analyses challenge the notion that observed gender differences in STEM achievement are fixed or socially meaningful. The societal significance of documented gender disparities in STEM performance remains unclear (Xie & Shauman, 2003; Weinberger, 2005) and warrants further investigation.

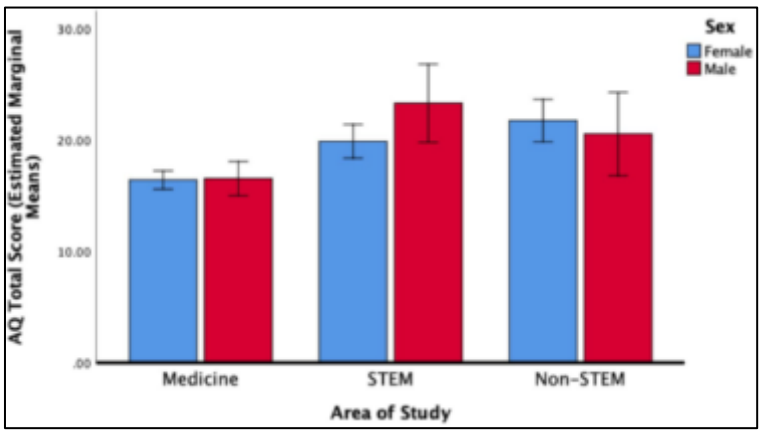

Figure 1 Biological Sex and STEM Education. Adapted from Turner, E., Aitken, E., & Richards, G. (2021). Autistic Traits, STEM, and Medicine: Autism Spectrum Quotient Scores Predict Medical Students' Career Specialty Preferences

To date, the increase in women's participation in STEM fields has been driven primarily by the overall rise in female enrollment (Mann & DiPrete, 2013), particularly regarding persistence in the "science pipeline" (Miller & Wai, 2015). Consequently, while the number of women earning academic degrees in STEM disciplines has steadily increased, the proportional representation of women in many STEM fields has not grown since the 1980s (England & Li, 2006; England et al., 2007; DiPrete & Buchmann, 2013; Mann & DiPrete, 2013) and may even be declining in certain engineering fields (Mann & DiPrete, 2013). In the United States and other industrialized countries, women have earned the majority of degrees in biological and social sciences since the 1980s, yet they remain significantly underrepresented among degree recipients in engineering, physical sciences, mathematics, and computer science (Charles & Bradley, 2002, 2006, 2009; Xie & Shauman, 2003; Xie & Killewald, 2012; DiPrete & Buchmann, 2013).

The perception that men are naturally more talented and more interested in science is a widely held cultural stereotype (Nosek et al., 2009; Leslie et al., 2015). Although most people consciously reject this notion (Hyde et al., 1990), implicit association studies confirm the pervasive presence of the "math = male" stereotype across age, race/ethnicity, sex, and country (Nosek et al., 2002; Kiefer & Sekaquaptewa, 2007; Nosek et al., 2009; Cvencek et al., 2011). Reflecting this belief, girls consistently report lower self-assessments of quantitative skills, lower confidence in mathematical abilities, less interest and motivation in learning mathematics and science, higher levels of math anxiety than their male peers, and reduced interest in STEM careers (Correll, 2001, 2004; Fredricks & Eccles, 2002; Watt, 2004, 2006; Jacobs et al., 2006; Else-Quest et al., 2011; Sadler et al., 2012; Wang et al., 2013). Girls are also more likely than boys to express interest in people-oriented careers, perceive science as inconsistent with these orientations, and view a scientific lifestyle as less attractive (Miller et al., 2006).

Causal explanations for women's career choices often attribute them to biologically based sex predispositions. A prominent theory posits that prenatal hormonal exposure predisposes women to a natural affinity for interaction with people and caregiving, whereas men show an innate interest in objects, technical, and mechanical systems (Baron-Cohen, 2003; Su et al., 2009; Schmidt, 2011). Another theory suggests that the gender gap in interests is linked to women's biological reproductive roles, which prioritize family responsibilities over occupational roles (Ceci et al., 2009; Ceci & Williams, 2010, 2011).

Recent studies do not support these essentialist explanations. Research indicates that interest in STEM is largely shaped by environmental influences (Cheryan et al., 2009, 2011; Murphy et al., 2007; Stout et al., 2011). Social-psychological and sociocultural perspectives provide more nuanced explanations for the gender gap. Studies show that macro-level cultural conditions influence gender differences in STEM interest through a variety of causal mechanisms, which are encoded and transmitted via the attitudes and expectations of parents, teachers, and other influential individuals.

5. Conclusions

Despite years of extensive research, a definitive causal factor for Autism Spectrum Disorder (ASD) remains elusive, highlighting the complexity of its origins. Nevertheless, it is widely accepted that genetics plays a decisive role, with current estimates suggesting that heritability accounts for approximately 80% of the risk (Bai et al., 2019). The extensive variability in observed genetic mutations further complicates the disorder's etiology (Monteiro & Feng, 2017). No single gene mutation has been identified as the primary causal factor for autism (Abrahams & Geschwind, 2008). Instead, a multitude of discrete mutation sites across various genes contributes to a diversity that makes direct comparisons between individuals with autism difficult. Consequently, the genetic landscape of autism is characterized by a broad spectrum of manifestations, reflecting the widespread effects of these mutations on neurodevelopment and function. Nevertheless, the complex interaction among genetic makeup, hormonal activity, and environmental factors significantly contributes to a deeper understanding of the causality of the autism spectrum.

Compliance with ethical standards

Acknowledgments

The Authors would like to thank the Department of Pedagogy and Primary Education, School of Education, University of Athens, Greece. Team, for their support.

Disclosure of conflict of interest

The Authors proclaim no conflict of interest.

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