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Article

Genetic Basis of Autism Spectrum Disorder: A Review

Enas Muhsin Abbass Alkhafagy[1], Dena Mohammed Alsahaf[2], Faris K. Khadir[3], Hiba A. Abdulhussein[4]*

1. Al-Mustansiriyah University, College of medicine, Chemistry and Biochemistry Department, Baghdad, Iraq
2. Almustafa University, College of Health and Medical Technology, Radiology Technology Department, Baghdad, Iraq
3. Physiology Department, College of Medicine, University of Baghdad, Baghdad, Iraq
4. University of Hilla, College of Pharmacy, Babylon, Iraq

*Correspondence: hiba.abbas@hilla-unc.edu.iq

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Abstract: Autism spectrum disorder (ASD) is a highly heritable neurodevelopmental condition, yet it is also markedly heterogeneous at the molecular level, with contributions ranging from rare, highly penetrant single-gene mutations and copy-number variants to common polymorphisms in genes encoding neurotransmitter and neuropeptide systems. Objectives to review the genetic basis of ASD across four complementary lines of evidence – family and twin studies, single-gene and copy-number variation, epigenetic mechanisms, and functional candidate genes in the serotonin, oxytocin and melatonin pathways – and to integrate these findings with a case-control genotyping study of oxytocin-pathway polymorphisms performed by the authors in Iraqi children. A narrative review of the English-language literature indexed in PubMed, Scopus and Google Scholar was conducted using the terms "autism", "genetics", "twin study", "copy number variation", "epigenetics", "serotonin transporter", "oxytocin receptor" and "melatonin", used alone and in combination, together with the authors' own case-control study of OXT/rs2770378 and OXTR/rs53576 polymorphisms in 60 Iraqi male children with autism and 26 controls. Twin studies show markedly higher concordance in monozygotic than dizygotic twins, confirming strong heritability, although a substantial shared-environment component has also been demonstrated. Genetic heterogeneity is extensive, with more than 100 genes and dozens of recurrent copy-number variants implicated, alongside epigenetic mechanisms exemplified by MECP2 in Rett syndrome and FMR1 in fragile X syndrome. Among functional candidate genes, the serotonin transporter (SLC6A4), the oxytocin/oxytocin-receptor system (OXT/OXTR) and the melatonin-synthesis gene ASMT show biologically plausible but inconsistently replicated associations with ASD. In the authors' Iraqi cohort, the OXT rs2770378 G/G genotype was associated with significantly higher autism risk and lower serum oxytocin, particularly in severe autism, while OXTR rs53576 showed no significant overall association. The genetic architecture of ASD spans rare, highly penetrant syndromic causes and common, functionally modest variation in neurotransmitter and neuropeptide pathways. Continued integration of family-based, genomic and candidate-gene approaches, including ethnically diverse case-control data such as the authors' own, is required to refine the genetic map of autism and to move toward clinically useful genetic biomarkers.

Keywords: Autism Spectrum Disorder, Genetics, Copy Number Variation, Epigenetics, Oxytocin, Serotonin Transporter, Melatonin

Introduction

Autism spectrum disorder (ASD) is a neurodevelopmental condition characterized by persistent deficits in social communication and social interaction together with restricted, repetitive patterns of behavior, interests or activities, typically evident before three years of age [1]. ASD is now recognized as one of the most heritable neuropsychiatric conditions, yet it is simultaneously one of the most genetically heterogeneous, such that no single variant or mechanism can account for more than a small fraction of cases across the population [4]. This heterogeneity has led research to proceed along several complementary tracks: quantifying overall heritability through family and twin studies; cataloguing rare, highly penetrant single-gene mutations and copy-number variants (CNVs); characterizing epigenetic mechanisms that alter gene expression without changing the underlying DNA sequence; and testing common polymorphisms in biologically plausible candidate genes, particularly those governing neurotransmitter and neuropeptide signaling.

This review summarizes the evidence from each of these tracks, with particular attention to three functional candidate systems – the serotonin transporter (SLC6A4), the oxytocin and oxytocin-receptor genes (OXT/OXTR), and the melatonin-synthesis gene ASMT – and integrates this literature with the results of the authors' own case-control genotyping study of OXT/rs2770378 and OXTR/rs53576 in Iraqi children with autism.

Materials and Methods

This is a narrative review of the English-language literature indexed in PubMed, Scopus and Google Scholar, using the search terms "autism", "genetics", "twin study", "copy number variation", "epigenetics", "serotonin transporter", "oxytocin receptor", "OXTR", "ASMT" and "melatonin", used alone and in combination; reference lists of retrieved articles were also screened for additional relevant publications. Landmark family and twin studies, genomic and candidate-gene association studies, and narrative or systematic reviews addressing the genetic basis of ASD were included. Where relevant, the findings of the authors' own case-control study of 60 Iraqi male children with autism (20 each with mild, moderate and severe disease) and 26 age- and gender-matched controls, in which serum oxytocin was measured by enzyme-linked immunosorbent assay and OXT/rs2770378 and OXTR/rs53576 genotyping was performed by polymerase chain reaction–restriction fragment length polymorphism (PCR-RFLP), are integrated into the discussion for illustrative purposes [26].

Evidence from family and twin studies

The first systematic evidence for a genetic contribution to autism came from twin studies. In the landmark 1977 study of 21 same-sexed twin pairs, the concordance rate for autism was 36% in monozygotic (MZ) twins compared with 0% in dizygotic (DZ) twins, and concordance for a broader cognitive phenotype reached 82% in MZ versus 10% in DZ pairs [2]. Subsequent, larger twin studies confirmed a substantially higher concordance in MZ than DZ twins, generally in the range of 60–90% versus 0–31%, providing strong indirect evidence for genetic involvement in autism susceptibility [4]. However, a large population-based Californian twin study using contemporary diagnostic standards estimated that shared environmental factors common to twins could account for as much as 55% of the liability to ASD, a finding that tempered earlier, purely genetic interpretations of twin data and highlighted the importance of gene–environment interaction in autism risk [3]. Family studies have complemented twin data by demonstrating an elevated recurrence risk of autism and autism-related traits among siblings of affected probands, further supporting a heritable, though clearly multifactorial, basis for the disorder [6].

Single-gene mutations and copy number variation

Beyond the aggregate heritability demonstrated by twin and family studies, molecular genetic work has identified specific rare, highly penetrant mutations and structural genomic variants in a meaningful subset of individuals with ASD. Single-gene mutations reported in autistic patients include FMR1, MECP2, CNTNAP2, PTEN, TSC2, NF1, ARX, UBE3A, NLGN3, NLGN4, NRXN1, FOXP1, FOXP2 and SHANK3, several of which cause recognizable syndromic forms of intellectual disability in which autism is a core or associated feature [4]. Copy-number variation – submicroscopic chromosomal deletions or duplications – has been identified at numerous recurrent loci, including 1q21.1, 7q11.23, 15q11-13, 16p11.2, 17q12 and 22q13.33, among others. A comprehensive review of the clinical and research genetics literature identified more than 100 distinct disease genes and 44 recurrent genomic loci implicated in ASD or autistic behavior, most of which are also causally linked to intellectual disability, underscoring the shared genetic architecture between these two neurodevelopmental phenotypes [5]. This degree of genetic heterogeneity supports the view that autism is not a single clinical or molecular entity, but rather a common behavioral manifestation of a very large number of distinct, mostly rare, genetic and genomic disorders [5].

Epigenetic mechanisms in autism

In addition to changes in DNA sequence, epigenetic modifications – including DNA methylation and post-translational histone modification – provide a further mechanism through which gene expression relevant to ASD can be regulated, often in response to environmental exposures. The clearest example is Rett syndrome, a progressive neurodevelopmental disorder classified among the ASDs, which is caused by mutations in the methyl-CpG-binding protein 2 gene (MECP2); because MeCP2 protein itself functions as an epigenetic regulator of gene expression, Rett syndrome represents a genetic mutation that produces its effects through epigenetic dysregulation [7]. A parallel example is fragile X syndrome, caused by expansion of a CGG trinucleotide repeat in the 5′ untranslated region of FMR1; this expansion triggers epigenetic silencing of the gene, again illustrating how a primary genetic lesion can act through secondary epigenetic mechanisms to produce an ASD phenotype. Epigenetic regulation of genes such as RELN, which encodes a protein important for neuronal positioning during cortical development, has similarly been proposed as a contributor to the broader ASD phenotype, reinforcing the concept that genetic and epigenetic mechanisms act together rather than independently in the pathogenesis of autism.

Functional candidate genes in neurotransmitter and neuropeptide pathways

Serotonin transporter (SLC6A4)

Elevated whole-blood serotonin is found in approximately one-quarter of individuals with autism and is familial, making the serotonergic pathway an attractive candidate system. The serotonin transporter gene (SLC6A4/SERT) has received particular attention because selective serotonin reuptake inhibitors, which target this transporter, are used clinically to treat anxiety and repetitive behaviors in ASD. In a large family-based study, SLC6A4 showed strong evidence of genetic linkage to autism, driven predominantly by allele-sharing in affected males, and multiple coding and non-coding variants were preferentially transmitted to affected individuals and correlated with more severe rigid-compulsive behaviors, supporting SLC6A4 as a genuine, allelically heterogeneous susceptibility locus rather than a single common risk variant [8].

Oxytocin and oxytocin receptor genes (OXT/OXTR)

Oxytocin (OXT) is a nine-amino-acid neuropeptide, synthesized principally in the hypothalamus, with a well-established role in social affiliation, bonding and the recognition of social cues, making its gene and that of its receptor (OXTR) compelling functional candidates in ASD [12]. OXT is located on chromosome 20p13 and OXTR on chromosome 3p26 [14]. Clinically, plasma oxytocin has been reported to be reduced in a subset of autistic children, and randomized trials of intranasal oxytocin have shown improvements in social cognition and repetitive behaviors in some individuals

with ASD, providing a biological rationale for examining OXT/OXTR genetic variation as a source of this dysregulation [13], [15].

Genetic association studies of OXT and OXTR polymorphisms – principally rs2770378 in OXT and rs53576 in OXTR – have produced heterogeneous results across populations. OXTR variation has been linked to autism in Caucasian cohorts, to Asperger syndrome diagnosis, and to a distinct "social" subgroup within broader ASD samples [21], [17], [19], and a large meta-analysis of rs53576 found an overall association with sociality-related traits, albeit with substantial heterogeneity across ethnicities [18]. Conversely, some case-control studies, including a Slovakian cohort, found no significant association between OXTR polymorphisms and autism [22], and a Swedish twin study found that OXT rs2770378 was associated with autistic-like traits only in female, not male, twins [20]. Non-clinical studies further support a broader role for OXTR rs53576 in empathy, stress reactivity and depressive symptomatology, situating this variant within a wider framework of social-emotional regulation rather than autism specifically [24], [25], and related work on genes governing sex steroids and neural growth has shown overlapping associations with autistic traits and empathy, suggesting that OXT/OXTR variation should be interpreted within a broader, pathway-based genetic context [23].

To illustrate how such genotype-phenotype relationships can be examined directly, the authors previously conducted a case-control study of OXT/rs2770378 and OXTR/rs53576 in 60 Iraqi male children with autism (20 each with mild, moderate and severe disease) and 26 age- and gender-matched controls, using PCR-RFLP genotyping and enzyme-linked immunosorbent assay measurement of serum oxytocin [26]. Although overall genotype and allele frequencies for OXT/rs2770378 did not differ significantly between autistic patients and controls, children carrying the G/G genotype had a significantly higher risk of autism than those with the A/A genotype (odds ratio 1.98), a risk that increased markedly among children with severe autism (odds ratio 9.89), and the G/G genotype was also associated with significantly lower serum oxytocin than the A/A genotype. For OXTR/rs53576, genotype and allele frequencies again did not differ significantly between patients and controls, and this polymorphism had no significant effect on serum oxytocin, although a non-significant trend toward a higher A/A frequency was observed in severe autism[26]. These findings illustrate a more consistent genotype–biomarker relationship for OXT than for OXTR in this population, and they exemplify the kind of ethnicity-specific data needed to complement the larger, more heterogeneous international literature summarized in Table 2.

Melatonin synthesis pathway (ASMT)

Melatonin, synthesized from serotonin via the enzyme acetylserotonin O-methyltransferase (ASMT), regulates circadian and seasonal physiological rhythms, and reduced nocturnal melatonin secretion has repeatedly been reported in children with autism. In a mutation-screening study of 250 individuals with ASD and 255 controls, non-conservative ASMT variants – including a splice-site mutation – were identified specifically in the ASD group, and two promoter polymorphisms (rs4446909 and rs5989681) were significantly more frequent in ASD and were associated with markedly reduced ASMT transcript levels; biochemical analyses confirmed significantly lower ASMT enzymatic activity and melatonin levels in the ASD group, supporting a primary deficit in melatonin synthesis, driven in part by ASMT genetic variation, as a risk factor for autism [9]. As with OXT/OXTR, however, not all subsequent studies have replicated significant ASMT associations, again illustrating the broader pattern of biologically plausible but inconsistently replicated candidate-gene findings in ASD genetics.

Table 1. Overview of the main categories of genetic evidence in autism spectrum disorder

Category of genetic evidence	Representative examples	Key evidence / findings
Twin and family studies	Monozygotic vs. dizygotic concordance; sibling recurrence risk	Concordance markedly higher in monozygotic (up to ~90%) than dizygotic twins (0–31%), establishing strong heritability

Category of genetic evidence	Representative examples	Key evidence / findings
Single-gene (monogenic) causes	FMR1, MECP2, PTEN, TSC2, NF1, SHANK3, CNTNAP2, NLGN3/NLGN4, NRXN1, ARX, UBE3A	Rare, highly penetrant mutations account for a subset of cases, often as part of a recognisable syndrome
Copy-number variation (CNV)	Deletions/duplications at 1q21.1, 7q11.23, 15q11-13, 16p11.2, 17q12, 22q13.33 and other loci	More than 100 genes and dozens of recurrent genomic loci implicated across clinical and research genetics literature
Epigenetic mechanisms	MECP2 (Rett syndrome), FMR1 CGG-repeat expansion (fragile X syndrome), RELN promoter methylation	DNA methylation and chromatin modification alter gene expression without changing DNA sequence, contributing to syndromic ASD
Functional candidate genes – neurotransmitter/neuropeptide pathways	SLC6A4 (serotonin transporter), OXT/OXTR (oxytocin/receptor), ASMT (melatonin synthesis)	Common variants associated with altered protein function or expression; inconsistent replication across populations but biologically plausible

Table 2. Selected genetic association studies of the oxytocin/oxytocin-receptor (OXT/OXTR) candidate genes in autism spectrum disorder

Study (author, year)	Population	SNP	Key finding
Jacob et al, 2007	Caucasian children and adolescents with autism, USA	rs2254298	OXTR polymorphism associated with autism; maternal transmission bias for the risk allele
Chakrabarti et al, 2009	Individuals with Asperger syndrome and controls, UK	OXTR SNPs	OXTR variants associated with autistic traits and empathy scores
Lakatosova et al, 2013	Autistic children, Slovakia	rs53576, rs2268491	No significant association of either polymorphism with autism
Di Napoli et al, 2014	Adults with Asperger syndrome, UK	OXTR SNPs	Significant association between OXTR variation and Asperger syndrome diagnosis
Hovey et al, 2014	Swedish twins (autistic-like traits)	OXT rs2770378	Association with autistic-like traits in female but not male twins
Harrison et al, 2015	ASD probands and families, USA	OXTR SNPs	OXTR variation associated with a distinct “social” ASD subgroup
Li et al, 2015 (meta-analysis)	Pooled samples, multiple countries	rs53576	Overall association with sociality-related traits, with heterogeneity across ethnicities

Study (author, year)	Population	SNP	Key finding
Abdulamir et al, 2016 (authors' study)	60 autistic boys (mild/moderate/severe) and 26 controls, Iraq	OXT rs2770378, OXTR rs53576	OXT G/G genotype linked to significantly higher autism risk and lower serum oxytocin, most marked in severe autism; OXTR rs53576 not significantly associated overall

Gene–environment interaction

Although this review has focused on genetic contributors, the incomplete concordance for autism even among monozygotic twins, together with the substantial shared-environment component demonstrated in large twin cohorts, indicates that genetic susceptibility in ASD is generally expressed in interaction with environmental exposures rather than in isolation [3], [6]. This gene–environment framework is consistent with the broader genetic evidence reviewed above: rare, highly penetrant mutations and CNVs can be sufficient to cause an ASD phenotype largely independent of environmental modifiers, whereas common functional variants in pathways such as oxytocin, serotonin and melatonin signaling likely confer graded susceptibility that interacts with prenatal, perinatal and early developmental environmental factors to determine the final phenotype and its severity.

Gaps in knowledge and future directions

Several limitations constrain the current genetic understanding of ASD. First, the sheer number of implicated genes and loci, each contributing to only a small fraction of cases, complicates the translation of genetic findings into individually useful diagnostic tests [5]. Second, candidate-gene association studies of functional genes such as OXT, OXTR and ASMT, including the authors' own, are frequently limited by modest sample sizes and single-population ascertainment, which likely explains much of the inconsistent replication seen across studies and populations [26], [22]. Third, relatively few studies combine genotyping with functional assessment of gene expression, protein activity or receptor signaling, so it often remains unclear whether an associated polymorphism is itself functional or merely in linkage disequilibrium with an unidentified causal variant. Future work should prioritize larger, multi-ethnic, adequately powered cohorts that combine genome-wide and candidate-gene approaches with functional and longitudinal biomarker data, and that examine interacting neurochemical pathways – such as oxytocin, serotonin and melatonin – jointly rather than in isolation, in order to build a more complete and clinically actionable picture of the genetic basis of autism.

Conclusion

The genetic basis of autism spectrum disorder is best understood as a spectrum of its own: at one end, rare, highly penetrant single-gene mutations and copy-number variants that are individually sufficient to produce an ASD phenotype, often as part of a recognized syndrome; at the other, common, functionally modest polymorphisms in genes governing neurotransmitter and neuropeptide signaling – exemplified here by SLC6A4, OXT/OXTR and ASMT – that confer graded susceptibility in interaction with environmental factors. Family and twin studies confirm strong overall heritability but also a substantial environmental contribution, and epigenetic mechanisms provide a further layer linking genetic and environmental influences. The authors' own findings in an Iraqi cohort, showing a more consistent association for OXT rs2770378 than for OXTR rs53576 with both autism risk and serum oxytocin, illustrate how population-specific candidate-gene data continue to refine this increasingly complex, but increasingly well-characterized, genetic landscape.

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