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The risk factors affecting the development of ASD and the possibility for prenatal detection

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ABSTRACT

Autism Spectrum Disorder (ASD) is a multifaceted neurodevelopmental condition influenced by genetic, epigenetic, and environmental factors. Recent advancements in research have highlighted the polygenic nature of ASD, with key genetic markers such as chromosomal deletions and specific gene mutations contributing significantly to risk. Epigenetic mechanisms and maternal health factors during pregnancy, including infections, medication use, and nutritional deficiencies, further modify ASD susceptibility. Emerging diagnostic tools, including prenatal ultrasound and chromosomal microarray analysis, offer promising avenues for early detection by identifying structural abnormalities, genetic variations, and other risk indicators. Despite these advancements, challenges persist, including limited specificity and sensitivity of diagnostic methods, ethical and social concerns, and the high cost and limited accessibility of genetic testing. Additionally, the multifactorial origins of ASD necessitate larger, longitudinal studies to validate early markers and refine detection strategies. This study underscores the importance of addressing these limitations to improve early intervention and support for high-risk pregnancies, ultimately aiming to enhance outcomes for individuals with ASD.

Introduction

As to the DSM 5, autism spectrum disorder (ASD) is a neurobehavioral condition marked by enduring impairments in social and communication functioning, difficulties in forming, comprehending, and sustaining relationships, and atypical and persistent interests and repetitive activities. Compared to females, Males are four to five times more likely to develop ASD (Zayed Higher Organization for People with Determination & Ural Federal University, 2024). This disorder is among the most prevalent illnesses that affect children. The reason appears to be the outcome of genetic and environmental interactions. (Kobayashi et al., 2016; Ohkawara et al., 2015; American Psychiatric Association, 2013). The latest estimate, reported in 2020, found that 1 in 54 children were diagnosed with ASD. Just one year later, the reported estimate increased to 1 in 44 (Maenner et al.,2021). ASD overlaps to varying degrees with clinical symptoms and genetic risk factors (Geschwind and State, 2015). Twin studies show that while discordant twins are significantly less concordant, homozygous twins are extremely concordant. In the process of looking for particular genes linked to ASD, it was discovered that there are potential ASD-related genes on multiple chromosomes. It seems that genetic factors and the prenatal and postnatal environments combine resulting in ASD (Ohkawara et al., 2015).

Recent research suggests that changes that occur during pregnancy, and possibly delivery may increase the likelihood of autism in children who are genetically predisposed to the disorder (Stoner et al., 2014). An increasing amount of research indicates that early childhood and probably even prenatal periods are when the first indications of ASD appear (Regev et al.,2021; Whitehouse et al.,2011; Mizejewski, Lindau-Shepard and Pass, 2013; Hazlett et al.,2017). In fact, some prenatal studies have shown preliminary evidence of abnormal brain development and higher rates of structural anomalies in the renal system of both ASD fetuses and children with specific genetic syndromes associated with ASD. Additionally, recent postnatal studies have found indications of the prenatal onset of abnormal neurodevelopment in children with ASD. There is emerging interest in examining the prenatal organ development of fetuses later developing into children diagnosed with ASD (Regev et al.,2021; Unwin et al.,2016; Blanken et al.,2018; Fulceri et al.,2018; Gamliel et al.,2012; Caly et al.,2021; Hobbs et al.,2007; Bonnet-Brilhault et al.,2018; Whitehouse et al.,2011; Vianna et al., 2019; Jiang et al., 2017; Jing et al.,2019; Handrigan et al.,2013; Loirat et al.,2010; Courchesne, Gazestani and Lewis, 2020).

In the following study, some of the articles that have been reviewed in recent years in the world investigate the risk factors and possibility of detecting autism in the fetus and during pregnancy from the angles of environmental, health, genetic and hereditary effects. By investigating the interplay between genetic and environmental variables, particularly during the prenatal and perinatal stages, this study aims to evaluate the multifaceted roots of autism spectrum disorder (ASD). It seeks to identify early indicators of ASD, such as abnormal brain development and structural abnormalities on prenatal ultrasounds, to determine when early signs of the disorder appear. In addition, the research will review the development of vital organs, including the brain and kidney system, in fetuses who later develop ASD. Ultimately, the goal is to increase understanding of the earliest indicators and contributing factors of ASD to facilitate early detection of possible indicators and targeted interventions during pregnancy and early childhood.

Elsevier, PubMed, and Google Scholar were among the databases from which the publications used in this study were chosen. The search was conducted using keywords like "ultrasound," "prenatal diagnosis of autism," "Autism," "fetal ultrasound," and "diagnosing." Predetermined inclusion and exclusion criteria were used to screen the studies. Only papers that addressed the genetic, environmental, or epigenetic factors that contribute to prenatal diagnostic indications of autism spectrum disorder (ASD) were considered. Excluded were studies that did not address ASD indicators or had no bearing on the prenatal or perinatal period. Additional keywords pertaining to genetic and environmental risk factors, such as "maternal health," "epigenetics," and "gene-environment interaction," were included to the search technique in an attempt to guarantee thorough coverage of all relevant components. In order to efficiently mix terms, the chosen databases were searched using Boolean operators as part of a methodical search procedure. For example, phrases such as "genetic risk factors AND autism" or "maternal health AND prenatal diagnosis" were used to find research that focused on certain facets of the etiology of ASD. To guarantee agreement with the study's goals, pertinent papers were then screened based on their abstracts and complete contents. To find more pertinent research, the reference lists of important papers were also carefully examined. This thorough approach made sure that relevant and high-quality research addressing both prenatal ASD detection markers and contributory variables was included

Literature review

Recent research highlights those disruptions in neurotransmitter systems play a critical role in the development of autism spectrum disorder (ASD).

Among the various neurotransmitters, gamma-aminobutyric acid (GABA) and glutamate are central to maintaining the balance between neural excitation and inhibition, a balance that is crucial for normal brain development and function.

Dysregulation of this excitatory/inhibitory (E/I) balance has been proposed as a core mechanism underlying the behavioral and cognitive symptoms characteristic of ASD (Bjørklund & El-Ansary, 2024). Multiple studies have demonstrated that individuals with ASD often show reduced GABAergic activity and alterations in glutamatergic signaling. These changes can impair synaptic plasticity, leading to abnormalities in social behavior, communication, and repetitive actions. Evidence from animal models further supports this notion, where deficits in GABAergic signaling are associated with autistic-like behaviors, and GABA supplementation has shown partial improvements in social interaction (Alabdali, 2025). Additionally, biomarker studies have revealed that disruptions in neurotransmitter systems can be detected early, suggesting that measurements of GABA, glutamate, and related neurochemical markers could aid in early diagnosis and the development of personalized treatment strategies (El-Ansary et al., 2024).

The medical and scientific communities have worked extremely hard to identify the risk factors and etiology of autism since it was first diagnosed. It is widely recognized that ASD is a condition resulting from intricate interactions between genetics and environment, with heritability estimates varying between 40 and 80% (Chaste and Leboyer, 2012). The prenatal identification of autism involves the potential identification of ASD symptoms and/or characteristics during pregnancy and before birth (in utero) (Zhang,2020). There were compelling scientific indications by the 1980s that ASD was in fact heritable (Newschaffer, Fallin and Lee, 2002).

Another key biological component in ASD research is oxytocin (OXT)—a neuropeptide that regulates social bonding and emotional processing. Evidence shows that individuals with ASD often exhibit dysregulated oxytocin pathways, which may underlie some of the core symptoms such as reduced eye contact, empathy, and social reciprocity. Studies utilizing oxytocin administration have reported enhanced social cognition and brain connectivity in both neurotypical individuals and ASD patients, although outcomes can vary depending on genetic and developmental factors (Bjørklund & Bhat, 2024).

Risk Factors for ASD

In 1977, Folstein and Rutter found that monozygotic twins shared more diagnoses of ASD than dizygotic twins, suggesting a genetic influence (Folstein and Rutter, 1977). The heritability of ASD in monozygotic twins is high (0.62-0.94) and siblings of affected individuals have a high probability of relative recurrence (10.1%) (Geschwind and State, 2015). Le Couteur et al., supported this finding, recording 60% concordance for monozygotic twins versus no concordant dizygotic twins. Additionally, it has been found that a child's likelihood of developing ASD is proportional to the percentage of their genome they share with an affected sibling or parent (Constantino et al., 2010; Risch et al., 2014; Sandin et al., 2014; Le Couteur et al., 1995). After examining 2,388 families, Freed and Pevsner discovered an ascertainment bias for pathogenic mosaic variants in ASD patients in comparison to siblings who were not afflicted. Large-scale sequencing investigations of post-zygotic mutations have revealed scores of additional risk genes and established somatic mosaicism as a major role in the etiology of ASD. They have also confirmed previously implicated candidate genes, such as SCN2A (Lim et al., 2017; Freed and Pevsner, 2016).

Because ASD is so common, there is a great deal of interest in studying its biological foundation, genetic risk factors, and the risk genes that promote ASD pathways and symptoms. It has been demonstrated that uncommon genetic mutations play a significant influence in the genetic heterogeneity and strong genetic component of ASD (Masi et al.,2017; Jiang et al., 2014; Jeste and Geschwind, 2014). In a review published in Nature Reviews Neurology in 2014, Shafali and Geschwind noted that chromosomal microarray analysis, a clinical diagnostic tool for identifying microdeletions or duplications linked to congenital and neurodevelopmental disorders, is a useful genetic test that is "warranted and clinically indicated for all suspected cases of ASD" (Batzir, Shohat and Maya, 2015; Jeste and Geschwind, 2014). Study findings (2018) showed that rare inherited structural variants in cis-regulatory elements (CRE-SVs) contribute to autism spectrum disorder (ASD). Specifically, paternally inherited CRE-SVs were more likely to be transmitted to children with ASD compared to their healthy siblings, suggesting that these non-coding genetic variants, particularly those inherited from the father, may increase the risk of developing ASD (Brandler et al., 2018). Another study found that measuring sperm mosaicism can stratify the recurrence risk of ASD due to de novo mutations. By using deep whole-genome sequencing, the researchers identified specific mosaic variants in the father's sperm that were linked to ASD risk (Breuss et al., 2020). Genetic variation in CRs (chromatin remodelers) has been found to be strongly related with disease in GWAS on neurodevelopmental disorders, including intellectual disabilities and ASD (Kim and Leventhal, 2015). There is evidence linking the DbetaH (DBH) gene to autism in

families whose children have low serum dopamine β-hydroxylase levels. The attributable risk was substantial (42%) despite the odds ratios indicating only a modest significance for the DBH-allele as a risk allele, suggesting this allele is a significant influence in predicting the likelihood of having an autistic child (Robinson et al., 2001; Caglayan, 2010).

Spatial-temporal clustering provides an efficient way to identify exposure to intergenerational environmental dangers that may have epigenetic impacts. In an investigation of the intergenerational effects of autism spectrum disorder, 3,957 children and family trees from residence areas of ASD cases in Utah were used to detect Spatial-temporal clusters of ancestors. found statistically significant spatiotemporal clusters during key developmental windows linked to the offspring's likelihood of developing ASD. An effective method for researching transgenerational effects that might be epigenetic in nature is the spatial and temporal grouping of family pedigrees spanning more than three generations (Richards Steed et al., 2022). Genes with epigenetic-modulating functions are highly involved in ASD susceptibility. A recent review of 215 candidate genes estimated that 19.5% are epigenetic regulators, suggesting the potential for diverse disease phenotypes from a few pathogenic variants (Duffney et al., 2018).

Early Markers of ASD

A significant study published in the American Journal of Human Genetics indicates that there is a significant increased risk of autism and schizophrenia associated with deletion of the chromosomal region 17q12. According to the study, there is a 14-fold higher likelihood of developing one of those problems among those who do not have this significant region. This study discovered that 24 out of 75,719 people had chromosomal area 17q12 deleted; 20 of these people had autism or delayed development, and 4 had schizophrenia. This means that the 17q12 region and the 15 genes it contains are formally linked to autism for the first time (Moreno-De-Luca et al.,2010). It is in line with findings that indicate schizophrenia and autism may both result from the same genetic defects (Carroll and Owen, 2009). The Gilboa study reports a prenatal identification of hyperechogenic kidneys in a normal-sized fetus, which leads to the detection of autism spectrum disorder and 17q12 deletion syndrome. Seven fetuses were recognized with hyperechogenic renal parenchyma over a nine-year period, and they were monitored prospectively. A 17q12 deletion was detected molecularly in five of the seven fetuses. Four children exhibited symptoms that were consistent with neurodevelopmental abnormalities during a long-term follow-up. Prenatal pracogenic kidneys, 17q12 microdeletion, and autism spectrum disorder have been found to be highly correlated (Gilboa et al.,2016).

Chromatin modifiers have been linked to autism spectrum disorder in recent research due to the discovery of frequent de novo gain-of-function mutations in afflicted people. Chromosome remodeling factor CHD8 may operate as a "master regulator" of a shared etiology for ASD. According to systems biology techniques, mid-fetal development is a pivotal period in the etiology of ASD, as evidenced by the enrichment of CHD8 and other putative ASD risk genes (Barnard, Pomaville and O'Roak, 2015). The chromodomain helicase DNA-binding protein CHD8 is among the most frequently found de-novo mutations in autism (Sanders et al.,2012; O'Roak et al.,2012a; O'Roak et al.,2012b; Neale et al.,2012).

Lately researchers have shown that many copy number variations (CNVs), which are deletions or duplications of a DNA sequence, are present in individuals who suffer from autism or schizophrenia (Zhao et al., 2007; Sebat et al.,2007; Zayed Higher Organization for People with Determination & Ural Federal University, 2024). It is now recognized that CNV plays a significant role in an individual's vulnerability to ASD; estimates suggest that around 10% of ASD cases are directly caused by these changes (Geschwind, 2011). A study by Anderson et al. found that placental trophoblast inclusions are associated with ASD (Anderson et al., 2007). Park et al. assessed placental shape characteristics in a sample of younger siblings of children with ASD and a sample of peers who were not at risk in a more recent study about placental gross form. They discovered that the siblings of the children with ASD had placental thickness measures that were somewhat higher and rounder, more regular placentas around the circumference. Although they persisted in both genders, the disparities were more noticeable in the women. The siblings of the children with ASD also exhibited less heterogeneity in placental shape (Park et al., 2018). It seems that fetal exposure to high androgens increases the probability of ASD. Rather than being the origin of the condition, in utero hyperandrogenemia may suggest poor neurogenesis associated to autism that has already happened and is impacting fetal androgen homeostasis. In an Israeli cohort of 437,222 children born between 2013 and 1999, the relationship between conditions related to maternal hyperandrogenism—specifically, polycystic ovarian syndrome, or PCOS—and ASD was examined. The findings showed that the likelihood of ASD in children of mothers with PCOS was greater than those of mothers without PCOS (odds ratio = 1.42, 95% confidence interval: 1.24,1.64), and the covariates taken into consideration only partially moderated this impact. There were also increased chances for additional conditions linked to

hyperandrogenemia. The results provide credence to the direct role that maternal hyperandrogenemia plays in the genesis of ASD (Rotem et al.,2021). During fetal development, the development of the heart and brain happens concurrently. One organ's development may be impacted by disruptions to organogenesis in the other. Congenital heart disease (CHD) children have intrauterine maldevelopment because of their aberrant circulations; nevertheless, prior to, during, or following surgery, they frequently experience postnatal brain insults due to hypoxia or ischemia. Children who have cardiac surgery are more likely to have ASD. There is growing evidence that suggests a probable link between ASD and CHD. Several population-based studies have found that children with CHD have a greater likelihood of developing ASD (Sigmon et al.,2019; Tsao et al.,2017; Calderon, Bellinger and Newburger, 2019; Miller et al., 2007; McQuillen and Miller, 2010; Wier et al.,2006; Dizitzer et al.,2020; Dawson et al.,2009; Bean Jaworski et al., 2017; Hultman, Sparén and Cnattingius, 2002; Razzaghi, Oster and Reefhuis, 2015). Findings also point to a favorable correlation between the severity of ASD and prenatal structural abnormalities. Congenital defects are in fact more common in people with autism and intellectual difficulties; children with ASD who have wider-set eyes or CHD have greater language, cognitive, and attention deficits than other ASD children (Timonen-Soivio et al., 2015; Bean Jaworski et al., 2017; Tsao et al.,2017; Aldridge et al., 2011).

A collection of conditions known as fetal alcohol spectrum disorders (FASD) are linked to the consequences of alcohol exposure during pregnancy and are distinguished by somatic and cognitive changes. Given that alcohol has an impact on all phases of brain development, several writers postulated that exposure to alcohol during pregnancy might raise the chance of ASD in those who are genetically vulnerable (Carpita et al.,2022). Autism affects males more than females, giving rise to the idea that the influence of steroid hormones on early fetal brain development may be one important early biological risk factor (Gutiirrez-Sacristán et al., 2022). In a study, all males born between 1993 and 1999 who were subsequently recognized with autism or Asperger syndrome (n = 128) were identified from their amniotic fluid samples when matched usually developing controls were used. The study also made use of the Danish Psychiatric Central Register and the Danish Historic Birth Cohort. Progesterone, 17α-OHP , androstenedione, testosterone, and cortisol concentration levels were examined. The group with autism had increases in latent generalized steroidogenic factor for all hormones. These increases could play a significant role in epigenetic prenatal programming pathways and interact with other key pathophysiological elements in autism (Baron-Cohen et al.,2015). Steroid hormones are thought to be an important early fetal environmental factor that may interact with other risk factors for autism and other neurodevelopmental conditions that affect the sexes asymmetrically, given their ability to exert epigenetic programming effects during early critical periods of brain development (Baron-Cohen et al.,2011; Geschwind and Galaburda, 1985; Lombardo et al.,2012; Glover,2011; Auyeung, Lombardo and Baron-Cohen, 2013). Autistic traits were documented to be increased following prenatal exposure to abnormally high levels of testosterone caused by congenital adrenal hyperplasia (KnickMeyer et al., 2006). The main mechanism via which environmental variables likely act is epigenetic regulation.

Numerous environmental variables, including maternal difficulties or infections during pregnancy, prenatal exposure to anticonvulsant medicines, and parental age, particularly paternal age, have been suggested to potentially increase this risk (Rasalam et al., 2005; Kong et al., 2012; O'Roak et al., 2012; Ohkawara et al., 2015).Many studies have indicated that although genetic factors are important for ASD, maternal immune system responses during pregnancy can also be related to the pathogenesis of ASD. Zimmerman et al. looked at maternal anti-brain antibodies in autism and identified specific serum antibodies that recognize prenatally expressed brain antigens in the mothers of autistic children. They concluded that there were distinctive patterns of antibody reactivity to fetal brain in mothers and children, which could be relevant to autism pathogenesis (Zimmerman et al., 2007). A study released in March 2019 and published in JAMA Psychiatry analyzed 1,791,520 Swedish children. It found that if a pregnant person suffered from a severe infection, their child was 79% more likely to be detected with autism spectrum disorder. The increase was found with both major infections (like sepsis, pneumonia, meningitis, and flu) and minor infections (like urinary tract infections) (Scutti,2019). Data reveal that maternal viral infection in the first trimester and bacterial infection in the second trimester are associated by identifying autism in the offspring (Atladóttir et al., 2010). Due to prenatal viral infection, maternal immune activation might result in elevated levels of IL-6 (interleukin 6) and changed gene expression, which might result in autistic behavior and neuropathology in the fetus later in pregnancy or after delivery (Parker-Athill and Tan, 2011). The rubella virus was the first known risk factor for autism. Additionally, measles and mumps viruses can induce encephalitis, which might eventually end in autism (Chess, 1977; Chess, 1971; Ziring, P, 1977; Sandler et al., 2001). The encephalitis-causing viral infections that lead to autism frequently happen in utero. On the other

hand, herpes virus-induced encephalitis has been shown to induce autism in elderly people. When combined, these findings indicate that some viruses can result in autism (Ratajczak,2011). Researchers have discovered a possible connection between a mother's use of medications during her pregnancy and her child's likelihood of developing autism. Autism development has been linked to maternal usage of thalidomide as an environmental factor. Also in a study, they found that most autistic thalidomide victims had abnormalities in the outer areas of their ears, but not in their arms or legs. This trend indicated that many people started having symptoms 20–24 days after conception, during the beginning of the pregnancy. Many cases of autism begin early in pregnancy. There is a link between various early-pregnancy medications and autism (Strömland et al., 1994; Landrigan, 2010). Misoprostol, an analog of prostaglandin that is used to treat stomach ulcers and as an abortifacient in some countries, has been linked to autism after failed efforts at abortion. The sixth week after conception was the typical exposure time. Prenatal exposure to the anti-convulsant valproic acid, which took place three to five weeks after conception, was a factor in autism. Moreover, autism was linked to mother rubella infection, which was most common in the first eight weeks following pregnancy. There has also been evidence linking acetaminophen to autism. Mothers of autistic children frequently have higher fevers and bacterial and viral infections during pregnancy, which may harm the developing fetus and increase the risk of autism in the offspring. Acetaminophen is frequently used by these moms to treat their illnesses. If acetaminophen is administered to a newborn on a regular basis, it can deplete sulfate and glutathione, which might lead to a regression into autism (Schultz et al., 2008; S.T., 2010; Meyer et al., 2006; Rodier, 2000; Torres, 2003; Ratajczak,2011; Kidd, 2002). Following the MMR II vaccination, children administered acetaminophen had a notably greater likelihood of developing autism compared to those administered ibuprofen (Good, 2009). Numerous studies have linked the use of antidepressants, particularly SSRIs, to autism; however, it is unclear if this relationship is due to the medications themselves or the mother's depression (Man et al.,2015). Additionally, valproate, a drug used to treat epilepsy and other neurological problems, has been demonstrated in research published in the Journal of the American Medical Association to raise the likelihood of autism (Christensen et al.,2013).

According to a 2011 epidemiological study, taking prenatal vitamins at least the first month of pregnancy and three months before conception reduced the likelihood that child might develop autism by half. Mathers who skipped vitamins and had a significant genetic predisposition to the condition increased the likelihood of developing autism by up to seven times (Schmidt et al.,2017). Due to 2014 research published in the American Journal of Epidemiology, there is a five times higher likelihood of autism in children whose mothers are iron deficient. If the mother is 35 years of age or older, or if she has a metabolic disease like diabetes, high blood pressure, or obesity, the risk goes up (Schmidt et al.,2014). These results were supported by JAMA research published in April 2015, which showed that mothers with gestational diabetes at 26 weeks of pregnancy had a 63 percent higher likelihood of having an autistic child. That means for every 1,000 women with gestational diabetes, seven of them may have a child with autism. Interestingly, the study discovered children delivered to mothers with type 2 diabetes before conception did not have a higher likelihood of autism, maybe because the mothers were on medication to regulate their blood sugar levels (Xiang et al.,2015). Based on the neurobiology of developing genes, a theory concerning the etiology of autism posits that it may be a problem of very early fetal development (around days 20–24 of gestation), with environmental exposures during pregnancy either causing or contributing to autism (London and Etzel, 2000).

Research using rodent and primate models has demonstrated that MIA (Maternal Immune Activation) can produce offspring exhibiting social and cognitive impairments, closely resembling ASD phenotypes. These models provide essential insights into both mechanistic pathways and potential therapeutic interventions for reducing the impact of prenatal immune dysregulation (Bjørklund et al., 2024).

Chromosomal microarray (CMA) has promising clinical validity, higher detection rates, and better diagnostic yields compared to other testing methods (e.g., karyotype).The most popular and first-tier technique in prenatal settings for identifying inherited or de novo copy number variations linked to ASD is CMA (Robert et al.,2017; Schaefer and Mendelsohn, 2013; Zhao et al., 2018; Soorya et al.,2013; Bean Jaworski et al., 2017; Armengol et al.,2012; Hillman et al.,2013; Klugman et al.,2014; Miller et al., 2010). Prenatal genetic testing, or PGT, offers benefits for identifying ASD susceptibility genes in fetuses. It can help parents make intelligent choices by informing them about the possibility that their unborn child will have ASD. The PGT findings might enable these parents to prepare for early intervention or other measures for their children who might be impacted by ASDs (Chen et al., 2015; Chen et al., 2012). Nevertheless, PGT has limitations. Using PGT to detect ASD susceptibility genes presents significant ethical, legal, and social challenges. These include the potential for controversial decisions about pregnancy termination

based on genetic findings, the moral implications of selecting embryos with certain genetic traits, and the societal impact of stigmatizing individuals with ASD. Additionally, there are concerns about the accuracy and interpretation of test results, which might lead to undue stress or misinformed decisions for prospective parents (Zhao et al., 2018; Chen et al., 2015; Chen et al., 2012). In 2018, researchers postulated that a poly- "omic" RNA panel derived from saliva could differentiate between children with ASD and those with neurotypical and non-ASD developmental delays. According to Hicks et al., this method of measuring salivary poly-omic RNA could be a precise and noninvasive way to increase the specificity of ASD referrals or offer unbiased support for the identification of ASD (Hicks et al.,2018).

Over the past years, advances in technology and equipment availability have made the use of ultrasound an integral part of clinical care in pregnancy. Among the applications of gynecology and obstetrics ultrasound, the following can be mentioned: identification of placenta previa. assessment of gestational age; Identification of the number and position of embryos; documentation of fetal life; Assessment of amniotic fluid volume; and biophysical assessment of the fetus. Ultrasound has also been used to evaluate the uterus, cervix, uterine adnexa, and fetal anatomy and has been useful in identifying abnormalities as early as the 12th week of pregnancy. As a result, abnormalities that may indicate genetic and developmental issues that need more testing and monitoring may be revealed. Structural abnormalities and "soft markers" that might point to genetic abnormalities or other non-genetic embryonic traumas such intrauterine infections are among the abnormalities that the survey can identify. When structural abnormalities or "soft markers" are found during the prenatal anatomy survey, a detailed evaluation of the fetal anatomy and the possibility of further diagnostic testing for chromosomal abnormalities are typically prompted. (Edwards and Hui, 2018; Cargill et al.,2009; Ali et al., 2012; Wickstrom et al.,1996; Maizels et al.,2006; Chen et al., 2001).

A 2024 study analyzed the results of 219 ultrasounds performed at -12, -20, and between 26 and 30 weeks of pregnancy, examining brain development at multiple points in pregnancy and its relationship to Early Neurodevelopmental Outcomes Examined in Autism. Tricuspid Diameter (TCD) in the 2nd trimester was significantly correlated with autistic traits measured by the Q-CHAT at 18 to 20 months. Longitudinal ultrasound data showed variations in fetal brain growth rates between trimesters, with head circumference (HC) and TCD showing significant correlations, particularly in the 3rd trimester. Males exhibited larger brain measurements in the late 2nd trimester. The study posited that early cerebellar dysfunction could influence autism by disrupting sensory integration and social network maturation. Prenatal factors, including estradiol levels, may influence both TCD growth and autistic traits (Aydin et al., 2024).

To look at the connection between fetal ultrasonography anomalies and ASD, research including 659 children (229 ASD, 201 typically developing siblings TDS, and 229 TDP) born in southern Israel between 2004 and 2018 produced the following findings: 29.3% of children with ASD had abnormalities on fetal ultrasonography, compared to only 15.9% and 9.6% in the TDS and TDP groups. At the time of ultrasonography, there was a noticeably higher prevalence of fetal anomalies among sufferers of ASD. The most common ultrasound abnormalities linked to the detection of ASD were those relating to the heart, head, and brain. The frequency of concomitant fetal abnormalities on sonograms was greater in females with ASD (43.1% vs. 25.3%, $P = 0.013$) than in males with ASD (Regev et al.,2022). The topic of whether prenatal ASD testing is feasible persists despite prior studies demonstrating that autism is detectable even in infancy. Deborah Brauser reported in a 2014 WebMD article that minor research comparing the head and stomach diameters of 40 infants who were subsequently recognized with ASD at 20 weeks in utero to 120 healthy children revealed that the ASD children had bigger heads and abdomens than the healthy children (Brauser, 2014).

Overall, we might say that hyperechogenic kidneys, placental anomalies, and ultrasound-detectable indicators like head size and tricuspid diameter are likely to suggest risk for ASD. The presence of congenital conditions, especially congenital heart diseases, has been seen in some cases of ASD. Advances in diagnostic tools such as Chromosomal microarray (CMA) and prenatal genetic testing (PGT) can effectively help identify markers of ASD. Non-invasive tools, such as ultrasound and salivary RNA panels, are being developed as potential early detection of possible indicators aids for ASD.

Maybe it can be said that in all the above-mentioned subjects, diagnosis methods require time or laboratory tools, which sometimes do not reach definitive results. Finding a way to detecting autism during pregnancy, which can be done using common and widely used and perhaps less expensive methods, can open up a bigger world of possible treatment options. These findings highlight genetic, epigenetic, and prenatal health factors as critical in ASD risk and point toward promising diagnostic tools for earlier, non-invasive detection.

Discussion

Genetic Risk Factors

Considering that the earliest possible detection greatly facilitates the earliest start of specific development and a higher degree of effectiveness, recent research has increasingly focused on mapping the genetic background of autism spectrum disorder (ASD). However, despite decades of genetic research, no single genetic marker has been identified as universally reliable for genetic screening. Current efforts focus on identifying common variants and understanding the polygenic nature of ASD. While no definitive genetic marker for ASD exists at present, prenatal genetic testing may still be considered for parents of children with ASD in subsequent pregnancies, offering some potential for early intervention and risk assessment. Genetic studies, particularly those involving twins and families, have pointed to a polygenic nature of ASD, where multiple genes contribute to risk. These studies also highlight the complex interplay between genetic factors and environmental influences on the expression of ASD (Hallmayer et al., 2011). In addition, recent research suggests that structural brain changes may be associated with genetic risk factors for ASD. For example, the measurement of brain structure through imaging techniques in the second trimester of pregnancy has shown promise in predicting ASD risk. Studies by Hobbs suggested that second-trimester measurements of biparietal diameter (BPD) are important for assessing the risk of later ASD development, further underlining the potential for prenatal assessments to aid in early identification (Hobbs et al.,2007).

In the late 1900s, researchers conducted many studies, including twin and family studies, pointing to the apparent polygenetic and heterogenetic nature of ASD and the unclear environmental influence on expression. As Anderson argued, early phenotypes associated with the disorder can aid in the identification of candidate genes, early screening, or risk assessment for ASD (Anderson et al.,2007). Various hypotheses about biomarkers for ASD include postnatal changes in head size, brain volume anomalies, neuroanatomical differences in brain regions like the amygdala, differences in the serotonergic system, immune system irregularities, and distinctive neuropeptide or neurotrophin profiles. Studies have suggested that these biological anomalies could be detectable early in life and serve as useful biomarkers for ASD. Moreover, some of these neurobiological markers are believed to have prenatal origins. Research increasingly supports the theory that the neuropathologic processes underlying ASD may begin in utero, and that prenatal environmental or genetic factors contribute significantly to the early development of ASD (Newschaffer, Fallin, & Lee, 2002). Previously, atypical Chemokine and Cytokine profiles were also found in both amniotic fluid and neonatal bloodspots of individuals with a detection with autism in the HBC (Abdallah et al., 2013; Abdallah, Larsen, Grove, et al., 2012; Abdallah, Larsen, Mortensen, et al., 2012).

Environmental Risk Factors

One study in the New England Journal of Medicine found differences in the brains of children with autism as early as the second trimester of pregnancy. This study by Stoner highlighted significant changes in the brain structure of children who would later be diagnosed with ASD. They observed that these differences included altered brain patterns and neuroanatomical features as early as the second trimester, suggesting that early neurodevelopmental changes may be associated with ASD. However, while these findings indicate that brain development anomalies are present early in pregnancy, the study emphasized that the causes of these changes remain complex and multifactorial. Current understanding points to a combination of genetic, environmental, and possibly epigenetic factors influencing the development of ASD (Stoner et al.,2014). A 2014 French study conducted on mice and published in the journal Science suggests that the use of spinal anesthesia during labor results in higher concentrations of chloride or salt in the brains of newborn mice. Schmidt conducted a study in mice where they found that spinal anesthesia could affect the developing brain by increasing chloride levels in the neurons. This disruption was associated with neurodevelopmental abnormalities. The authors hypothesized that similar processes might occur in humans, suggesting a potential environmental risk factor for ASD. While the study was conducted on mice, the researchers raised concerns about the potential implications for human prenatal development, specifically in terms of how anesthesia could impact the developing brain and increase ASD risk (Schmidt et al.,2014).

Emerging research links abnormalities in fetal ultrasound (US) parameters to ASD and other neurodevelopmental disorders (NDDs). Several studies have suggested that abnormalities detected via prenatal ultrasound, such as changes in fetal brain development, could be linked to an increased risk of ASD and other neurodevelopmental disorders. Advances in three-dimensional (3D) and four-dimensional (4D) ultrasound have enabled more detailed assessments of fetal brain structure and function. These technologies provide high-resolution images that can capture dynamic fetal movements and measure brain volume more precisely. Research by Fulceri highlighted the potential of these technologies to identify at-risk fetuses for early intervention. Given the low cost and relatively easy access to ultrasound, it could become

a valuable tool for early risk assessment in pregnancies with potential neurodevelopmental concerns (Fulceri et al., 2018). Obstetrical ultrasound can also help monitor fetal exposure to potential risk factors for ASD, such as maternal inflammation or certain medications taken during pregnancy. Prenatal exposure to certain factors, such as maternal infections, stress, or medications, has been associated with an increased risk of ASD. Ultrasound technology can monitor the impact of these factors on fetal development. For example, maternal inflammation or the use of certain drugs during pregnancy may affect brain development and increase the risk of ASD. Tracking such exposures through ultrasound can provide critical information for managing pregnancies at higher risk. Although ultrasound itself cannot directly diagnose ASD, it can assist in identifying structural anomalies and ensuring closer monitoring of high-risk pregnancies. However, it is important to note that not all pregnancies with ultrasound-detected abnormalities result in autism, and many children diagnosed with ASD show no detectable abnormalities on prenatal ultrasound (Christensen et al., 2023).

Early Markers

A blood test has been investigated for its potential to detect large genetic mutations linked to autism spectrum disorder (ASD). A pilot study, as discussed by Zeliadt, demonstrated that certain mutations, particularly those associated with ASD, could be identified through maternal blood sampling. This non-invasive approach holds promise for early detection, especially for identifying large chromosomal mutations that may increase the likelihood of ASD in a fetus. The technique is still in its early stages, and researchers have expressed plans to extend this method to detect smaller genetic variations, such as those affecting a single DNA base (Zeliadt, 2019). These advances may help refine genetic screening protocols and improve the understanding of the genetic underpinnings of ASD.

Combining multimodal biomarkers—such as imaging, genetic screening, and metabolic profiling—may improve diagnostic precision. The integration of such markers into clinical practice could facilitate the implementation of personalized precision medicine for ASD, targeting interventions based on the individual's biological profile (Bjørklund et al., 2024).

However, while blood tests can provide insights into the genetic risks, diagnosing ASD in the early stages remains challenging. Ultrasound imaging has been explored as a tool for identifying structural abnormalities that could signal an increased risk for neurodevelopmental disorders, including ASD. Several studies have reported that abnormal fetal ultrasound findings, such as changes in brain size or atypical brain structure, may be correlated with ASD in some cases. For example, 3D and 4D ultrasounds have been used to measure brain development and monitor fetal movement, offering a more detailed view of neurodevelopmental processes (Fulceri et al., 2018). Despite these advancements, predicting ASD based solely on ultrasound findings is complex. Research shows that many children with ultrasound-detected brain abnormalities, such as changes in head size or abnormal cortical folding, do not go on to develop autism. Conversely, some children diagnosed with ASD show no observable ultrasound abnormalities, further complicating the ability to rely on imaging for prediction (Stoner et al., 2014; Fulceri et al., 2018). Thus, while ultrasound can help in monitoring fetal development, its predictive value for ASD remains limited, and it cannot serve as a definitive diagnostic tool for autism.

Ultrasound has emerged as a valuable tool for prenatal assessments due to its non-invasive nature, widespread availability, and relatively low cost. Advances in three-dimensional (3D) and four-dimensional (4D) ultrasound technology allow for precise measurements of fetal brain development and dynamic imaging of fetal movements, which can provide important insights into neurodevelopmental risks (Fulceri et al., 2018). These methods enable clinicians to monitor structural and functional development of the fetal brain and detect anomalies that may be associated with ASD, such as changes in head circumference or cortical folding (Christensen et al., 2023). Ultrasound is also critical in identifying fetal exposure to environmental risk factors. It can detect placental irregularities or measure fetal organ development, which might indicate maternal health issues such as inflammation or infections during pregnancy, both of which are linked to ASD risk (Regev et al., 2022). Obstetrical ultrasound can be employed to monitor high-risk pregnancies more closely, ensuring timely interventions if abnormalities are detected. Additionally, ultrasound's wide accessibility makes it an ideal research tool for studying neurodevelopment in diverse populations. Its ability to capture early developmental milestones and potential markers for ASD highlights its potential role in early detection efforts, particularly when combined with genetic and environmental data (Hobbs et al., 2007).

Despite the promise of prenatal diagnostic tools such as ultrasound and blood tests in identifying autism spectrum disorder (ASD) risk, several significant limitations constrain their utility. One major challenge is the insufficient specificity and sensitivity of these methods, which limits their ability to provide definitive diagnoses. For instance, while prenatal ultrasounds can detect structural abnormalities in the fetal brain or other organs, these findings are not consistent predictors of ASD. Many

children with such abnormalities do not develop ASD, and conversely, many children diagnosed with ASD show no detectable abnormalities during prenatal imaging (Christensen et al., 2023). The use of genetic screening methods, such as chromosomal microarray (CMA) or blood tests for detecting large mutations, also presents challenges. These methods are limited by their accessibility and cost, particularly in resource-constrained settings. Ethical and social concerns further complicate their use, with potential risks including the misuse of genetic information, stigmatization, or decisions regarding pregnancy termination based solely on genetic risk factors (Zeliadt, 2019). A significant limitation in current research is the small sample sizes of many studies, which restricts the generalizability of findings. Large-scale, longitudinal studies are necessary to validate early markers such as head circumference or tricuspid diameter (TCD) measurements as reliable predictors of ASD (Aydin et al., 2024). Additionally, environmental factors influencing neurodevelopment, such as maternal infections or medication use, add complexity to interpreting results, as their direct impact on ASD risk is not always clear (Stoner et al., 2014).

Developing reliable, accessible, and ethically sound prenatal detection methods for ASD remains a challenge. Diagnostic methods like prenatal genetic testing and CMA depend on advanced technologies and laboratory resources, making them less practical in low-resource settings. Current prenatal markers and ultrasound-detectable abnormalities, such as placental shape variations or TCD measurements, are not definitive indicators. The polygenic and complex genetic basis of ASD complicates efforts to identify a single conclusive marker. Ethical and social dilemmas, such as decisions about pregnancy continuation and the impact on families when high ASD risk is identified, further underscore the need for caution in applying these methods. Moreover, many findings regarding potential prenatal markers or genetic factors are derived from small-scale studies. To improve the reliability of early ASD evaluations, larger and longer-term investigations are urgently required.

Conclusion

In conclusion, recent research has deepened our understanding of the intricate interactions among genetic, environmental, and prenatal factors in the development of autism spectrum disorder (ASD). Advances in identifying genetic markers, such as chromosomal deletions and specific gene mutations, have underscored the heritable aspects of ASD, while epigenetic mechanisms and maternal health during pregnancy, including exposure to infections, medications, and nutritional imbalances, have been shown to significantly influence risk. These findings highlight the multifactorial nature of ASD and the need for comprehensive approaches to study its origins. Emerging diagnostic tools, especially non-invasive techniques like ultrasound and prenatal genetic testing, present promising opportunities for early detection. These methods can potentially identify structural abnormalities, genetic variations, and other risk factors during pregnancy, paving the way for timely interventions. However, significant limitations persist, including the low specificity and sensitivity of current markers, ethical and accessibility challenges, and the need for larger-scale studies to validate findings.

The future of ASD research lies in addressing these challenges by improving the accuracy, affordability, and ethical application of diagnostic tools. Longitudinal studies with larger sample sizes are essential to confirm early markers and refine risk assessment strategies. By overcoming these hurdles, research can move toward developing reliable and accessible prenatal diagnostic approaches, ultimately supporting earlier interventions and improved outcomes for children with ASD. These efforts will also provide vital insights for high-risk pregnancies, helping families make informed decisions and better prepare for the future.

References

1. Abdallah, M.W., Larsen, N., Grove, J., et al. (2012) ‘Amniotic fluid chemokines and autism spectrum disorders: An exploratory study utilizing a Danish Historic Birth Cohort’, Brain, Behavior, and Immunity, 26(1), pp. 170–176. Available at: https://doi.org/10.1016/j.bbi.2011.09.003.

2. Abdallah, M.W., Larsen, N., Mortensen, E.L., et al. (2012) ‘Neonatal levels of cytokines and risk of autism spectrum disorders: An exploratory register-based historic birth cohort study utilizing the Danish Newborn Screening Biobank’, Journal of Neuroimmunology, 252(1–2), pp. 75–82. Available at: https://doi.org/10.1016/j.jneuroim.2012.07.013.

3. Abdallah, M.W. et al. (2013) ‘Amniotic fluid inflammatory cytokines: Potential markers of immunologic dysfunction in autism spectrum disorders’, World Journal of Biological Psychiatry, 14(7), pp. 528–538. Available at: https://doi.org/10.3109/15622975.2011.639803.

4. Aldridge, K. et al. (2011) ‘Facial phenotypes in subgroups of prepubertal boys with autism spectrum disorders are correlated with clinical phenotypes’, Molecular Autism, 2(1). Available at: https://doi.org/10.1186/2040-2392-2-15.

5. Ali, M.K. et al. (2012) ‘Ultrasonographic soft markers of aneuploidy in second trimester fetuses’, Middle East Fertility Society Journal, 17(3), pp. 145–151. Available at: https://doi.org/10.1016/j.mefs.2012.04.007.

6. Alabdali, A. N. (2025). Effects of GABA Supplementation on Grooming Behavior and Social Interaction in a Propionic Acid-Induced Rat Model of Autism. International Journal for Autism Challenges & Solution, 2(1), 4–11. DOI: https://doi.org/10.54878/sgp7fp60

7. American Psychiatric Association (2013) 'Diagnostic and Statistical Manual of Mental Disorders', Diagnostic and Statistical Manual of Mental Disorders [Preprint]. Available at: https://doi.org/10.1176/appi.books.9780890425596.

8. Anderson, G.M. et al. (2007) 'Placental Trophoblast Inclusions in Autism Spectrum Disorder', Biological Psychiatry, 61(4), pp. 487–491. Available at: https://doi.org/10.1016/j.biopsych.2006.03.068.

9. Armengol, L. et al. (2012) 'Clinical utility of chromosomal microarray analysis in invasive prenatal diagnosis', Human Genetics, 131(3), pp. 513–523. Available at: https://doi.org/10.1007/s00439-011-1095-5.

10. Atladóttir, H.Ó. et al. (2010) 'Maternal infection requiring hospitalization during pregnancy and autism spectrum disorders', Journal of Autism and Developmental Disorders, 40(12), pp. 1423–1430. Available at: https://doi.org/10.1007/s10803-010-1006-y.

11. Auyeung, B., Lombardo, M. V. and Baron-Cohen, S. (2013) 'Prenatal and postnatal hormone effects on the human brain and cognition', Pflugers Archiv European Journal of Physiology, 465(5), pp. 557–571. Available at: https://doi.org/10.1007/s00424-013-1268-2.

12. Aydin, E., Tsompanidis, A., Chaplin, D., Hawkes, R., Allison, C., Hackett, G., Austin, T., Padaigaitė, E., Gabis, L. V., Sucking, J., Holt, R., & Baron-Cohen, S. (2024). Fetal brain growth and infant autistic traits. Molecular Autism, 15(1). https://doi.org/10.1186/s13229-024-00586-5

13. Barnard, R.A., Pomaville, M.B. and O'Roak, B.J. (2015) 'Mutations and modeling of the chromatin remodeler CHD8 define an emerging autism etiology', Frontiers in Neuroscience, 9(DEC). Available at: https://doi.org/10.3389/fnins.2015.00477.

14. Baron-Cohen, S. et al. (2011) 'Why are Autism Spectrum conditions more prevalent in Males?', PLoS Biology, 9(6). Available at: https://doi.org/10.1371/journal.pbio.1001081.

15. Baron-Cohen, S. et al. (2015) 'Elevated fetal steroidogenic activity in autism', Molecular Psychiatry, 20(3), pp. 369–376. Available at: https://doi.org/10.1038/mp.2014.48.

16. Batzir, N.A., Shohat, M. and Maya, I. (2015) 'Chromosomal microarray analysis (CMA) a clinical diagnostic tool in the prenatal and postnatal settings', Pediatric Endocrinology Reviews, 13(1), pp. 448–454.

17. Bean Jaworski, J.L. et al. (2017) 'Rates of autism and potential risk factors in children with congenital heart defects', Congenital Heart Disease, 12(4), pp. 421–429. Available at: https://doi.org/10.1111/chd.12461.

18. Blanken, L.M.E. et al. (2018) 'A prospective study of fetal head growth, autistic traits and autism spectrum disorder', Autism Research, 11(4), pp. 602–612. Available at: https://doi.org/10.1002/aur.1921.

19. Bonnet-Brilhault, F. et al. (2018) 'Autism is a prenatal disorder: Evidence from late gestation brain overgrowth', Autism Research, 11(12), pp. 1635–1642. Available at: https://doi.org/10.1002/aur.2036.

20. Bjørklund, G., & El-Ansary, A. (2024). GABAergic Dysregulation in Autism Spectrum Disorder: Collaborative Insights on Neurobiological Complexity. International Journal for Autism Challenges & Solution, 1(1), 72–78. DOI: https://doi.org/10.54878/4v2w1f14

21. Bjørklund, G., Bhat, R. S., & El-Ansary, A. (2024). Maternal Immune Activation and Autism Spectrum Disorder: Complex Interactions and Therapeutic Possibilities. International Journal for Autism Challenges & Solution, 1(1), 39–50. DOI: https://doi.org/10.54878/bgxvwf58

22. Bjørklund, G., & Bhat, R. S. (2024). The Role of Oxytocin in Autism Spectrum Disorder: Current Evidence and Therapeutic Implications. International Journal for Autism Challenges & Solution, 1(2), 4–17. DOI: https://doi.org/10.54878/h8j48873

23. Brandler, W. M., Antaki, D., Gujral, M., Kleiber, M. L., Whitney, J., Maile, M. S., Hong, O., Chapman, T. R., Tan, S., Tandon, P., Pang, T., Tang, S. C., Vaux, K. K., Yang, Y., Harrington, E., Juul, S., Turner, D. J., Thiruvahindrapuram, B., Kaur, G., … Sebat, J. (2018). Paternally inherited cis-regulatory structural variants are associated with autism. Science, 360(6386), 327–331. https://doi.org/10.1126/science.aan2261

24. Brauser, D. (2014) 'Routine ultrasounds may detect autism in utero', in International Congress of the Royal College of Psychiatrists (RCPsych). London, United Kingdom: Medscape Psychiatry. Available at: https://www.medscape.com/viewarticle/827333?form=fpf.

25. Breuss, M. W., Antaki, D., George, R. D., Kleiber, M., James, K. N., Ball, L. L., Hong, O., Mitra, I., Yang, X., Wirth, S. A., Gu, J., Garcia, C. A. B., Gujral, M., Brandler, W. M., Musaev, D., Nguyen, A., McEvoy-Venneri, J., Knox, R., Sticca, E., … Gleeson, J. G. (2020). Autism risk in offspring can be assessed through quantification of male sperm mosaicism. Nature Medicine, 26(1), 143–150. https://doi.org/10.1038/s41591-019-0711-0

26. Caglayan, A.O. (2010) 'Genetic causes of syndromic and non-syndromic autism', Developmental Medicine and Child Neurology, 52(2), pp. 130–138. Available at: https://doi.org/10.1111/j.1469-8749.2009.03523.x.

27. Calderon, J., Bellinger, D.C. and Newburger, J.W. (2019) 'Autism and congenital heart disease: Evidence and unresolved questions', Pediatrics, 144(5). Available at: https://doi.org/10.1542/peds.2019-2752.

28. Caly, H. et al. (2021) 'Machine learning analysis of pregnancy data enables early identification of a subpopulation of newborns with ASD', Scientific Reports, 11(1). Available at: https://doi.org/10.1038/s41598-021-86320-0.

29. Cargill, Y. et al. (2009) 'Content of a Complete Routine Second Trimester Obstetrical Ultrasound Examination and Report', Journal of Obstetrics and Gynaecology Canada, 31(3), pp. 272–275. Available at: https://doi.org/10.1016/S1701-2163(16)34127-5.

30. Carpita, B. et al. (2022) 'Autism Spectrum Disorder and Fetal Alcohol Spectrum Disorder: A Literature Review', Brain Sciences, 12(6). Available at: https://doi.org/10.3390/brainsci12060792.

31. Carroll, L.S. and Owen, M.J. (2009) 'Genetic overlap between autism, schizophrenia and bipolar disorder', Genome Medicine, 1(10). Available at: https://doi.org/10.1186/gm102.

32. Chaste, P. and Leboyer, M. (2012) 'Autism risk factors: Genes, environment, and gene-environment interactions', Dialogues in Clinical Neuroscience, 14(3), pp. 281–292. Available at: https://doi.org/10.31887/dcns.2012.14.3/pchaste.

33. Chen, C.P. et al. (2001) 'Prenatal diagnosis of de novo distal 11q deletion associated with sonographic findings of unilateral duplex renal system, pyelectasis and orofacial clefts', Prenatal Diagnosis, 21(4), pp. 317–320. Available at: https://doi.org/10.1002/pd.42.

34. Chen, L.S. et al. (2012) 'Chinese Americans' views of prenatal genetic testing in the genomic era: A qualitative study', Clinical Genetics, 82(1), pp. 22–27. Available at: https://doi.org/10.1111/j.1399-0004.2012.01871.x.

35. Chen, L.S. et al. (2015) 'Autism spectrum disorders: A qualitative study of attitudes toward prenatal genetic testing and termination decisions of affected pregnancies', Clinical Genetics, 88(2), pp. 122–128. Available at: https://doi.org/10.1111/cge.12504.

36. Chess, S. (1971) 'Autism in children with congenital rubella', Journal of Autism and Childhood Schizophrenia, 1(1), pp. 33–47. Available at: https://doi.org/10.1007/BF01537741.

37. Chess, S. (1977) 'Follow-up report on autism in congenital rubella', Journal of Autism and Childhood Schizophrenia, 7(1), pp. 69–81. Available at: https://doi.org/10.1007/BF01531116.

38. Christensen, D., Pazol, K., Overwyk, K. J., England, L. J., Alexander, A. A., Croen, L. A., Dowling, N. F., Schieve, L. A., Tian, L. H., Tinker, S. C., Windham, G. C., Callaghan, W. M., & Shapira, S. K. (2023). Prenatal ultrasound use and risk of autism spectrum disorder: Findings from the case-control Study to Explore Early Development. Paediatric and perinatal epidemiology, 37(6), 527–535. https://doi.org/10.1111/ppe.12998

39. Christensen, J. et al. (2013) 'Prenatal valproate exposure and risk of autism spectrum disorders and childhood autism', Jama, 309(16), pp. 1696–1703. Available at: https://doi.org/10.1001/jama.2013.2270.

40. Constantino, J.N. et al. (2010) 'Sibling recurrence and the genetic epidemiology of autism', American Journal of Psychiatry, 167(11), pp. 1349–1356. Available at: https://doi.org/10.1176/appi.ajp.2010.09101470.

41. Courchesne, E., Gazestani, V.H. and Lewis, N.E. (2020) 'Prenatal Origins of ASD: The When, What, and How of ASD Development', Trends in Neurosciences, 43(5), pp. 326–342. Available at: https://doi.org/10.1016/j.tins.2020.03.005.

42. El-Ansary, A., et al. (2024). The Relative Usefulness of the Identification and Analysis of Biomarkers for the Diagnosis of Autism Spectrum Disorders in Early Childhood and the Implementation of Personalized Precision Medicine. International Journal for Autism Challenges & Solution, 1(1), 51–71. DOI: https://doi.org/10.54878/c0yn1911

43. Le Couteur, A.L. et al. (1995) 'Autism as a strongly genetic disorder evidence from a british twin Study', Psychological Medicine, 25(1), pp. 63–77. Available at: https://doi.org/10.1017/S0033291700028099.

44. Dawson, S. et al. (2009) 'Birth defects in children with autism spectrum disorders: A population-based, nested casec-control study',

American Journal of Epidemiology, 169(11), pp. 1296–1303. Available at: https://doi.org/10.1093/aje/kwp059.

45. Dizitzer, Y. et al. (2020) 'Comorbidity and health services' usage in children with autism spectrum disorder: A nested case-control study', Epidemiology and Psychiatric Sciences [Preprint]. Available at: https://doi.org/10.1017/S2045796020000050.

46. Duffney, L.J. et al. (2018) 'Epigenetics and autism spectrum disorder: A report of an autism case with mutation in H1 linker histone HIST1H1E and literature review', American Journal of Medical Genetics, Part B: Neuropsychiatric Genetics, 177(4), pp. 426–433. Available at: https://doi.org/10.1002/ajmg.b.32631.

47. Edwards, L. and Hui, L. (2018) 'First and second trimester screening for fetal structural anomalies', Seminars in Fetal and Neonatal Medicine, 23(2), pp. 102–111. Available at: https://doi.org/10.1016/j.siny.2017.11.005.

48. Folstein, S. and Rutter, M. (1977) 'Genetic influences and infantile autism [13]', Nature, 265(5596), pp. 726–728. Available at: https://doi.org/10.1038/265726a0.

49. Freed, D. and Pevsner, J. (2016) 'The Contribution of Mosaic Variants to Autism Spectrum Disorder', PLoS Genetics, 12(9). Available at: https://doi.org/10.1371/journal.pgen.1006245.

50. Fulceri, F. et al. (2018) 'Antenatal ultrasound value in risk calculation for Autism Spectrum Disorder: A systematic review to support future research', Neuroscience and Biobehavioral Reviews, 92, pp. 83–92. Available at: https://doi.org/10.1016/j.neubiorev.2018.05.016.

51. Gamliel, M. et al. (2012) 'Minor fetal sonographic findings in autism spectrum disorder', Obstetrical and Gynecological Survey, 67(3), pp. 176–186. Available at: https://doi.org/10.1097/OGX.0b013e31824bb5d6.

52. Geschwind, D.H. (2011) 'Genetics of autism spectrum disorders', Trends in Cognitive Sciences, 15(9), pp. 409–416. Available at: https://doi.org/10.1016/j.tics.2011.07.003.

53. Geschwind, D.H. and State, M.W. (2015) 'Gene hunting in autism spectrum disorder: On the path to precision medicine', The Lancet Neurology, 14(11), pp. 1109–1120. Available at: https://doi.org/10.1016/S1474-4422(15)00044-7.

54. Geschwind, N. and Galaburda, A.M. (1985) 'Cerebral Lateralization: Biological Mechanisms, Associations, and Pathology: I. A Hypothesis and a Program for Research', Archives of Neurology, 42(5), pp. 428–459. Available at: https://doi.org/10.1001/archneur.1985.04060050026008.

55. Gilboa, Y. et al. (2016) 'Prenatal diagnosis of 17q12 deletion syndrome: from fetal hyperechogenic kidneys to high risk for autism', Prenatal Diagnosis, 36(11), pp. 1027–1032. Available at: https://doi.org/10.1002/pd.4926.

56. Glover, V. (2011) 'Annual research review: Prenatal stress and the origins of psychopathology: An evolutionary perspective', Journal of Child Psychology and Psychiatry and Allied Disciplines, 52(4), pp. 356–367. Available at: https://doi.org/10.1111/j.1469-7610.2011.02371.x.

57. Good, P. (2009) 'Did acetaminophen provoke the autism epidemic?', Alternative Medicine Review, 14(4), pp. 364–372.

58. Gutiirrez-Sacristán, A. et al. (2022) 'Multi-PheWAS intersection approach to identify sex differences across comorbidities in 59 140 pediatric patients with autism spectrum disorder', Journal of the American Medical Informatics Association, 29(2), pp. 230–238. Available at: https://doi.org/10.1093/jamia/ocab144.

59. Hallmayer, J. F., Cleveland, S., Risch, N., et al. (2011). Genetic Heritability and Shared Environmental Factors Among Twin Pairs With Autism. Archives of General Psychiatry, 68(11), 1095-1102.

60. Handrigan, G.R. et al. (2013) 'Deletions in 16q24.2 are associated with autism spectrum disorder, intellectual disability and congenital renal malformation', Journal of Medical Genetics, 50(3), pp. 163–173. Available at: https://doi.org/10.1136/jmedgenet-2012-101288.

61. Hazlett, H.C. et al. (2017) 'Early brain development in infants at high risk for autism spectrum disorder', Nature, 542(7641), pp. 348–351. Available at: https://doi.org/10.1038/nature21369.

62. Hicks, S.D. et al. (2018) 'Validation of a Salivary RNA Test for Childhood Autism Spectrum Disorder', Frontiers in Genetics, 9. Available at: https://doi.org/10.3389/fgene.2018.00534.

63. Hillman, S.C. et al. (2013) 'Use of prenatal chromosomal microarray: Prospective cohort study and systematic review and meta-analysis', Ultrasound in Obstetrics and Gynecology, 41(6), pp. 610–620. Available at: https://doi.org/10.1002/uog.12464.

64. Hobbs, K. et al. (2007) 'A Retrospective Fetal Ultrasound Study of Brain Size in Autism', Biological Psychiatry, 62(9), pp. 1048–1055.

Available at: https://doi.org/10.1016/j.biopsych.2007.03.020.

65. Hultman, C.M., Sparén, P. and Cnattingius, S. (2002) 'Perinatal risk factors for infantile autism', Epidemiology, 13(4), pp. 417–423. Available at: https://doi.org/10.1097/00001648-200207000-00009.

66. Jeste, S.S. and Geschwind, D.H. (2014) 'Disentangling the heterogeneity of autism spectrum disorder through genetic findings', Nature Reviews Neurology, 10(2), pp. 74–81. Available at: https://doi.org/10.1038/nrneurol.2013.278.

67. Jiang, Y.H. et al. (2014) 'Genetic diagnosis of autism spectrum disorders: The opportunity and challenge in the genomics era', Critical Reviews in Clinical Laboratory Sciences, 51(5), pp. 249–262. Available at: https://doi.org/10.3109/10408363.2014.910747.

68. Jiang, Y.L. et al. (2017) '[Prenatal diagnosis of 17q12 microdeletion syndrome in fetal renal abnormalities].', Zhonghua fu chan ke za zhi, 52(10), pp. 662–668. Available at: https://doi.org/10.3760/cma.j.issn.0529-567X.2017.10.004.

69. Jing, X.Y. et al. (2019) 'Prenatal diagnosis of 17q12 deletion syndrome: a retrospective case series', Journal of Obstetrics and Gynaecology, 39(3), pp. 323–327. Available at: https://doi.org/10.1080/01443615.2018.1519693.

70. Kidd, P.M. (2002) 'Autism, an extreme challenge to integrative medicine. Part 1: The knowledge base', Alternative Medicine Review, 7(4), pp. 292–316.

71. Kim, Y.S. and Leventhal, B.L. (2015) 'Genetic epidemiology and insights into interactive genetic and environmental effects in autism spectrum disorders', Biological Psychiatry, 77(1), pp. 66–74. Available at: https://doi.org/10.1016/j.biopsych.2014.11.001.

72. Klugman, S. et al. (2014) 'Clinical utility of chromosomal microarray analysis in prenatal diagnosis: Report of first 6 months in clinical practice', Journal of Maternal-Fetal and Neonatal Medicine, 27(13), pp. 1333–1338. Available at: https://doi.org/10.3109/14767058.2013.858243.

73. Knickmeyer, R. et al. (2006) 'Androgens and autistic traits: A study of individuals with congenital adrenal hyperplasia', Hormones and Behavior, 50(1), pp. 148–153. Available at: https://doi.org/10.1016/j.yhbeh.2006.02.006.

74. Kobayashi, T. et al. (2016) 'Autism spectrum disorder and prenatal exposure to selective serotonin reuptake inhibitors: A systematic review and meta-analysis', Reproductive Toxicology, 65, pp. 170–178. Available at: https://doi.org/10.1016/j.reprotox.2016.07.016.

75. Kong, A. et al. (2012) 'Rate of de novo mutations and the importance of father-s age to disease risk', Nature, 488(7412), pp. 471–475. Available at: https://doi.org/10.1038/nature11396.

76. Landrigan, P.J. (2010) 'What causes autism? Exploring the environmental contribution', Current Opinion in Pediatrics, 22(2), pp. 219–225. Available at: https://doi.org/10.1097/MOP.0b013e328336eb9a.

77. Lim, E.T. et al. (2017) 'Rates, distribution and implications of postzygotic mosaic mutations in autism spectrum disorder', Nature Neuroscience, 20(9), pp. 1217–1224. Available at: https://doi.org/10.1038/nn.4598.

78. Loirat, C. et al. (2010) 'Autism in three patients with cystic or hyperechogenic kidneys and chromosome 17q12 deletion', Nephrology Dialysis Transplantation, 25(10), pp. 3430–3433. Available at: https://doi.org/10.1093/ndt/gfq380.

79. Lombardo, M. V. et al. (2012) 'Fetal programming effects of testosterone on the reward system and behavioral approach tendencies in humans', Biological Psychiatry, 72(10), pp. 839–847. Available at: https://doi.org/10.1016/j.biopsych.2012.05.027.

80. London, E. and Etzel, R.A. (2000) 'The environment as an etiologic factor in autism: A new direction for research', Environmental Health Perspectives, 108(SUPPL. 3), pp. 401–404. Available at: https://doi.org/10.1289/ehp.00108s3401.

81. Maenner, M.J. et al. (2021) 'Prevalence and Characteristics of Autism Spectrum Disorder Among Children Aged 8 Years — Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2018', MMWR Surveillance Summaries, 70(11), pp. 1–16. Available at: https://doi.org/10.15585/MMWR.SS7011A1.

82. Maizels, M. et al. (2006) 'Late second trimester assessment of pyelectasis (SERP) to predict pediatric urological outcome is improved by checking additional features', Journal of Maternal-Fetal and Neonatal Medicine, 19(5), pp. 295–303. Available at: https://doi.org/10.1080/14767050600553225.

83. Man, K.K.C. et al. (2015) 'Exposure to selective serotonin reuptake inhibitors during pregnancy and risk of autism spectrum disorder in children: A systematic review and meta-analysis of observational studies', Neuroscience and

Biobehavioral Reviews, 49, pp. 82–89. Available at: https://doi.org/10.1016/j.neubiorev.2014.11.020.

84.Masi, A. et al. (2017) 'An Overview of Autism Spectrum Disorder, Heterogeneity and Treatment Options', Neuroscience Bulletin, 33(2), pp. 183–193. Available at: https://doi.org/10.1007/s12264-017-0100-y.

85.McQuillen, P.S. and Miller, S.P. (2010) 'Congenital heart disease and brain development', Annals of the New York Academy of Sciences, 1184, pp. 68–86. Available at: https://doi.org/10.1111/j.1749-6632.2009.05116.x.

86.Meyer, U. et al. (2006) 'The time of prenatal immune challenge determines the specificity of inflammation-mediated brain and behavioral pathology', Journal of Neuroscience, 26(18), pp. 4752–4762. Available at: https://doi.org/10.1523/JNEUROSCI.0099-06.2006.

87.Miller, D.T. et al. (2010) 'Consensus Statement: Chromosomal Microarray Is a First-Tier Clinical Diagnostic Test for Individuals with Developmental Disabilities or Congenital Anomalies', American Journal of Human Genetics, 86(5), pp. 749–764. Available at: https://doi.org/10.1016/j.ajhg.2010.04.006.

88.Miller, S.P. et al. (2007) 'Abnormal Brain Development in Newborns with Congenital Heart Disease', New England Journal of Medicine, 357(19), pp. 1928–1938. Available at: https://doi.org/10.1056/nejmoa067393.

89.Mizejewski, G.J., Lindau-Shepard, B. and Pass, K.A. (2013) 'Newborn screening for autism: In search of candidate biomarkers', Biomarkers in Medicine, 7(2), pp. 247–260. Available at: https://doi.org/10.2217/bmm.12.108.

90.Moreno-De-Luca, D. et al. (2010) 'Deletion 17q12 is a recurrent copy number variant that confers high risk of autism and schizophrenia', American Journal of Human Genetics, 87(5), pp. 618–630. Available at: https://doi.org/10.1016/j.ajhg.2010.10.004.

91.Neale, B.M. et al. (2012) 'Patterns and rates of exonic de novo mutations in autism spectrum disorders', Nature, 485(7397), pp. 242–246. Available at: https://doi.org/10.1038/nature11011.

92.Newschaffer, C.J., Fallin, D. and Lee, N.L. (2002) 'Heritable and nonheritable risk factors for autism spectrum disorders', Epidemiologic Reviews, 24(2), pp. 137–153. Available at: https://doi.org/10.1093/epirev/mxf010.

93.O'Roak, B.J. et al. (2012) 'Multiplex targeted sequencing identifies recurrently mutated genes in autism spectrum disorders', Science, 338(6114), pp. 1619–1622. Available at: https://doi.org/10.1126/science.1227764.

94.O ' Roak, B.J. et al. (2012) 'Sporadic autism exomes reveal a highly interconnected protein network of de novo mutations', Nature, 485(7397), pp. 246–250. Available at: https://doi.org/10.1038/nature10989.

95.Ohkawara, T. et al. (2015) 'Maternal viral infection during pregnancy impairs development of fetal serotonergic neurons', Brain and Development, 37(1), pp. 88–93. Available at: https://doi.org/10.1016/j.braindev.2014.03.007.

96.Park, B.Y. et al. (2018) 'Placental gross shape differences in a high autism risk cohort and the general population', PLoS ONE, 13(8). Available at: https://doi.org/10.1371/journal.pone.0191276.

97.Parker-Athill, E.C. and Tan, J. (2011) 'Maternal immune activation and autism spectrum disorder: Interleukin-6 signaling as a key mechanistic pathway', NeuroSignals, 18(2), pp. 113–128. Available at: https://doi.org/10.1159/000319828.

98.Rasalam, A.D. et al. (2005) 'Characteristics of fetal anticonvulsant syndrome associated autistic disorder', Developmental Medicine and Child Neurology, 47(8), pp. 551–555. Available at: https://doi.org/10.1017/S0012162205001076.

99.Ratajczak, H. V. (2011) 'Theoretical aspects of autism: Causes-A review', Journal of Immunotoxicology, 8(1), pp. 68–79. Available at: https://doi.org/10.3109/1547691X.2010.545086.

100.Razzaghi, H., Oster, M. and Reefhuis, J. (2015) 'Long-term outcomes in children with congenital heart disease: National Health Interview Survey', Journal of Pediatrics, 166(1), pp. 119-124.e1. Available at: https://doi.org/10.1016/j.jpeds.2014.09.006.

101.Regev, O. et al. (2021) 'Association Between Abnormal Fetal Head Growth and Autism Spectrum Disorder', Journal of the American Academy of Child and Adolescent Psychiatry, 60(8), pp. 986–997. Available at: https://doi.org/10.1016/j.jaac.2020.11.019.

102.Regev, O. et al. (2022) 'Association between ultrasonography foetal anomalies and autism spectrum disorder', Brain, 145(12), pp. 4519–4530. Available at: https://doi.org/10.1093/brain/awac008.

103.Richards Steed, R. et al. (2022) 'Evidence of transgenerational effects on autism spectrum disorder using multigenerational space-time cluster detection', International Journal of Health Geographics, 21(1). Available at: https://doi.org/10.1186/s12942-022-00313-4.

104.Risch, N. et al. (2014) 'Familial recurrence of autism spectrum disorder: Evaluating genetic and environmental contributions', American Journal of Psychiatry, 171(11), pp. 1206–1213. Available at: https://doi.org/10.1176/appi.ajp.2014.13101359.

105.Robert, C. et al. (2017) 'Role of genetics in the etiology of autistic spectrum disorder: Towards a hierarchical diagnostic strategy', International Journal of Molecular Sciences, 18(3). Available at: https://doi.org/10.3390/ijms18030618.

106.Robinson, P.D. et al. (2001) 'Genetically determined low maternal serum dopamine β-hydroxylase levels and the etiology of autism spectrum disorders', American Journal of Medical Genetics, 100(1), pp. 30–36. Available at: https://doi.org/10.1002/ajmg.1187.

107.Rodier, P.M. (2000) 'The early origins of autism.', Scientific American, 282(2), pp. 56–63. Available at: https://doi.org/10.1038/scientificamerican0200-56.

108.Rotem, R.S. et al. (2021) 'Associations of Maternal Androgen-Related Conditions with Risk of Autism Spectrum Disorder in Progeny and Mediation by Cardiovascular, Metabolic, and Fertility Factors', American Journal of Epidemiology, 190(4), pp. 600–610. Available at: https://doi.org/10.1093/aje/kwaa219.

109.S.T., S. (2010) 'Can autism be triggered by acetaminophen activation of the endocannabinoid system?', Acta Neurobiologiae Experimentalis, 70(2), pp. 227–231. Available at: https://doi.org/10.55782/ane-2010-1793.

110.Sanders, S.J. et al. (2012) 'De novo mutations revealed by whole-exome sequencing are strongly associated with autism', Nature, 485(7397), pp. 237–241. Available at: https://doi.org/10.1038/nature10945.

111.Sandin, S. et al. (2014) 'The familial risk of autism', Jama, 311(17), pp. 1770–1777. Available at: https://doi.org/10.1001/jama.2014.4144.

112.Sandler, A.D. et al. (2001) 'The pediatrician's role in the diagnosis and management of autistic spectrum disorder in children', Pediatrics, 107(5), pp. 1221–1226. Available at: https://doi.org/10.1542/peds.107.5.1221.

113.Schaefer, G.B. and Mendelsohn, N.J. (2013) 'Clinical genetics evaluation in identifying the etiology of autism spectrum disorders: 2013 guideline revisions', Genetics in Medicine, 15(5), pp. 399–407. Available at: https://doi.org/10.1038/gim.2013.32.

114.Schmidt, R.J. et al. (2014) 'Maternal intake of supplemental iron and risk of autism spectrum disorder', American Journal of Epidemiology, 180(9), pp. 890–900. Available at: https://doi.org/10.1093/aje/kwu208.

115.Schmidt, R.J. et al. (2017) 'Combined prenatal pesticide exposure and folic acid intake in relation to autism spectrum disorder', Environmental Health Perspectives, 125(9). Available at: https://doi.org/10.1289/EHP604.

116.Schultz, S.T. et al. (2008) 'Acetaminophen (paracetamol) use, measles-mumps-rubella vaccination, and autistic disorder: The results of a parent survey', Autism, 12(3), pp. 293–307. Available at: https://doi.org/10.1177/1362361307089518.

117.Scutti, S. (2019) Exposure to infection in the womb increases risk of autism and depression, study says, CNN Health & Medical. Available at: https://edition.cnn.com/2019/03/06/health/autism-depression-infection-in-utero-study/index.html.

118.Sebat, J. et al. (2007) 'Strong association of de novo copy number mutations with autism', Science, 316(5823), pp. 445–449. Available at: https://doi.org/10.1126/science.1138659.

119.Sigmon, E.R. et al. (2019) 'Congenital heart disease and autism: A case-control study', Pediatrics, 144(5). Available at: https://doi.org/10.1542/peds.2018-4114.

120.Soorya, L. et al. (2013) 'Prospective investigation of autism and genotype-phenotype correlations in 22q13 deletion syndrome and SHANK3 deficiency', Molecular Autism, 4(1). Available at: https://doi.org/10.1186/2040-2392-4-18.

121.Stoner, R. et al. (2014) 'Patches of Disorganization in the Neocortex of Children with Autism', New England Journal of Medicine, 370(13), pp. 1209–1219. Available at: https://doi.org/10.1056/nejmoa1307491.

122.Strömland, K. et al. (1994) 'Autism in Thalidomide Embryopathy: a Population Study', Developmental Medicine & Child Neurology, 36(4), pp. 351–356. Available at: https://doi.org/10.1111/j.1469-8749.1994.tb11856.x.

123. Timonen-Soivio, L. et al. (2015) 'The association between congenital anomalies and autism spectrum disorders in a Finnish national birth cohort', Developmental Medicine and Child Neurology, 57(1), pp. 75–80. Available at: https://doi.org/10.1111/dmcn.12581.

124. Torres, A.R. (2003) 'Is fever suppression involved in the etiology of autism and neurodevelopmental disorders?', BMC Pediatrics, 3.

Available at: https://doi.org/10.1186/1471-2431-3-9.

125. Tsao, P.C. et al. (2017) 'Additive effect of congenital heart disease and early developmental disorders on attention-deficit/hyperactivity disorder and autism spectrum disorder: a nationwide population-based longitudinal study', European Child and Adolescent Psychiatry, 26(11), pp. 1351–1359. Available at: https://doi.org/10.1007/s00787-017-0989-8.

126. Unwin, L.M. et al. (2016) 'A Prospective Ultrasound Study of Prenatal Growth in Infant Siblings of Children With Autism', Autism Research, 9(2), pp. 210–216. Available at: https://doi.org/10.1002/aur.1518.

127. Vianna, P. et al. (2019) 'Zika virus as a possible risk factor for autism spectrum disorder: Neuroimmunological aspects', NeuroImmunoModulation, 25(5–6), pp. 320–327. Available at: https://doi.org/10.1159/000495660.

128. Whitehouse, A.J.O. et al. (2011) 'Brief report: A preliminary study of fetal head circumference growth in autism spectrum disorder', Journal of Autism and Developmental Disorders, 41(1), pp. 122–129. Available at: https://doi.org/10.1007/s10803-010-1019-6.

129. Wickstrom, E.A. et al. (1996) 'A prospective study of the association between isolated fetal pyelectasis and chromosomal abnormality', Obstetrics and Gynecology, 88(3), pp. 379–382. Available at: https://doi.org/10.1016/0029-7844(96)00211-6.

130. Wier, M.L. et al. (2006) 'Congenital anomalies associated with autism spectrum disorders', Developmental Medicine and Child Neurology, 48(6), pp. 500–507. Available at: https://doi.org/10.1017/S001216220600106X.

131. Xiang, A.H. et al. (2015) 'Association of maternal diabetes with autism in offspring', JAMA - Journal of the American Medical Association, 313(14), pp. 1425–1434. Available at: https://doi.org/10.1001/jama.2015.2707.

132. Zayed Higher Organization for People with Determination, Ural Federal University. (2024). Emirati-Russian Psychology Dictionary. https://doi.org/10.54878/ERPD.124

133. Zeliadt, N. (2019) Ultrasensitive blood test may detect autism mutations in utero, Transmitter. Available at: https://www.thetransmitter.org/spectrum/ultrasensitive-blood-test-may-detect-autism-mutations-in-utero/?fspec=1.

134. Zhang, S. (2020) 'Prenatal Diagnosis of Autism', Encyclopedia of Autism Spectrum Disorders, pp. 1–4. Available at: https://doi.org/10.1007/978-1-4614-6435-8_102539-1.

135. Zhao, S. et al. (2018) 'A qualitative study exploring the attitudes toward prenatal genetic testing for autism spectrum disorders among parents of affected children in Taiwan', Research in Autism Spectrum Disorders, 48, pp. 36–43. Available at: https://doi.org/10.1016/j.rasd.2018.01.006.

136. Zhao, Y. et al. (2007) 'LIM-homeodomain proteins Lhx1 and Lhx5, and their cofactor Ldb1, control Purkinje cell differentiation in the developing cerebellum', Proceedings of the National Academy of Sciences of the United States of America, 104(32), pp. 13182–13186. Available at: https://doi.org/10.1073/pnas.0705464104.

137. Zimmerman, A.W. et al. (2007) 'Maternal antibrain antibodies in autism', Brain, Behavior, and Immunity, 21(3), pp. 351–357. Available at: https://doi.org/10.1016/j.bbi.2006.08.005.

138. Ziring,P, R. (1977) 'Congenital rubella: the teenage years', Pediatric Annals, 6(12), pp. 762–770.