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Perspective

Genetic Causes and Components of Autism Spectrum Disorder

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DESCRIPTION

The complex neurodevelopmental disorder known as Autism Spectrum Disorders (ASDs) is characterised by a trio of core behavioural deficits, including narrow interests, frequently accompanied by repetitive behaviour, communication and language impairments, and a lack of ability to interact with others in a reciprocal manner. Although ASD has a complicated origin and is among the most heritable illnesses, it is not a straightforward condition with a single pathology. ASD, schizophrenia, epilepsy, and intellectual disability are just a few of the neuropsychiatric illnesses that are thought to be caused by abnormalities in synapse formation, development, and stability. Synaptic dysfunction is causal in the pathophysiology of ASD, according to biological characterization of an expanding range of synaptic mutations in different model species.

The identification of molecular targets for drug discovery is dependent on the discovery of previously obscure molecular pathways for Autistic Spectrum Disorders (ASD). The heredity of ASD is now clearly demonstrated, and genetic research into the disorder has advanced at an exponential rate over the past ten years. With common genetic variation conferring a low risk, unusual chromosomal structural rearrangements and rare sequence variants have been implicated in particular. Rare genetic changes that have a significant impact provide specific targets for the investigation of underlying molecular pathways. To uncover associated functional pathways in ASD, systems biology approaches like data mining of massive genomic data sets are being employed.

ASD is a feature of the clinical picture in some genetic models that resemble established human diseases like fragile X, whereas other models lack well characterized human syndromes. The most prevalent environmental ASD model in rodents is that caused by Valproic Acid (VPA), either prenatally or in the early postnatal period. Following a single exposure to VPA at various stages of brain development, autism-like behaviours are induced, suggesting that the mechanism of action is *via* a more widespread biological mechanism such epigenetic alterations. In both human and animal models, maternal infection and inflammation are linked to ASD. Due to its extreme genetic heterogeneity, SD can result from both inherited and spontaneous gene mutations. In the last ten years, hundreds of genes have been found to play a role in the severe behavioural, social cognition, and communication problems that patients frequently experience. These cases make up only 10–20% of ASD cases, and persons with the same causal mutations may have significantly varied spectrum diagnoses. The genetic makeup of ASD will be described in this paper, along with how genetic modifiers including copy number variation, single nucleotide polymorphisms, and epigenetic changes probably have a significant impact on how the phenotypic spectrum of ASD patients are developed. Studies have concentrated on improving understanding of ASD and developing risk prediction and prevention because there is currently clinical and epidemiological evidence for an ASD cure. There are numerous related genes and environmental risk factors, making the causes of ASD complicated and multifaceted. Several maternal factors, including advanced age (35 years), chronic hypertension, preeclampsia, gestational hypertension, and being overweight before or during pregnancy, were significantly and without evidence of bias associated with the risk of ASD, according to a prior Umbrella Review (UR) of environmental risk factors for ASD. The concordance rate between monozygotic twins is higher (60-90%) than that between dizygotic twins (0-30%), indicating that genetic variables play important roles in ASD, according to accumulating twin- and family-based studies.

CONCLUSION

Studies have shown that autism has a genetic foundation, and several organisations are actively looking for susceptibility genes. The MZ concordance rate for a precise diagnosis of autism is just 36%, however twin studies have indicated that the co-twin frequently shows cognitive abnormalities, typically involving language delay. The MZ concordance rate increases to 82% when the autism spectrum diagnosis is defined more broadly to include language delay. Studies on the genetic links to autism show various potential causes. Human genetic research aim to establish the link between particular genetic variants (genotype) and observed traits (phenotype). This can assist specify which genotypes to look at for disease-related consequences and which phenotypic characteristics are probably most important for genetic investigations.

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