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Short Communication

Diagnostic and Clinical Significance of Autism Spectrum Disorder

James Feldon*

Department of Psychology, University of Rutgers, New Haven, USA

DESCRIPTION

Autism Spectrum Disorder (ASD) is a one of the most common neurodevelopmental disorders. ASD is highly genetically diverse and can be brought on by *de novo* or inheritable gene mutations. Hundreds of genes have been found to be involved in the severe communication, social cognition, and behavior issues that patients frequently experience. Patients with comparable pathogenic variations might be analyzed on totally different levels of the range. Genetic modifiers like copy number variation, single nucleotide polymorphisms, and epigenetic alterations likely play a key role in modulating the phenotypic spectrum of ASD patients. Additionally, the genetic landscape of ASD genetic modifiers can change convergent signaling pathways, which can make it harder to build neural circuits. The clinical implications of sex-linked modifiers are depending on both comprehending ASD and developing novel therapies [1-3].

Schizophrenia, bipolar disorder, and autism are among the mental health conditions whose causes are strongly linked to genetics. The genetic predisposition to autism is described in monozygotic and dizygotic twins. The model that better depicts DASs is multifactorial with a concordance of 60%-92% in monozygotic twins and 0%-10% in dizygotic twins. The multifactorial model is supported by differences observed in evaluation involving monozygotic twins, demonstrating the significance of environmental factors.

Examination of monozygotic and dizygotic twins, adopted children and their families helped the foundation of a strong genetic component in autism spectrum disordres. In any case, examination performed on the genome of autists flopped in the foundation of reliable indications of linkage. According to Mendel's laws, none of the cognitive and affective diseases as well as some psychoses follow a heritage pattern, supporting the hypothesis that multiple genes are involved. [4-6].

It is a complex issue with the inclusion of a few variants, each contributing with a reduced risk of phenotype, the identified genes have been difficult. In addition, chromosomal changes that are not always sufficient for disease development are the basis for hypotheses that support the existence of synergism and epistases between multiple candidate genes. Therefore, the majority of traditional linkage methods fail to detect even minute genetic effects in typically small samples. The type of coded proteins that play a significant role in the development of autism must then be taken into consideration when selecting candidate genes [7,8].

Translocations and inversions affect a significant number of involved chromosomes. These progressions bring about interference of genes along with deletions and duplications that are responsible for differences in gene expression. Duplications in 15q11-q13 and deletions in 2q37 are the most common mutations, most of which cannot be detected by analyzing the karyotype [9].

In the cytogenetic evaluation of autism, Freitag asserts that some chromosomal deletions in 2q37, 7q31, 22q11, and 22q13.3 are also significant. Chromosomic micro deletions liable for specific disorders, are related with the presence of secondary autism are Velocardiofacial disorder, DiGeorge condition and Facial Conotruncal abnormality disorder. Ion-channel-coding genes, among many others, appear to be involved in the onset of autism development [10].

CONCLUSION

The evaluation of autism has undergone significant shifts as a result of a number of developments in the examination of autism genetics. The majority of genetic analysis concentrate on particular chromosome regions based on the associated genes are related to the observed characteristics of the disease. The information on genetic or environmental factors is vital for right determination and treatment and may permit avoidance of the disorders. Diagnosis criteria used for autistic spectrum disorders, have been improved. The best way to diagnose autism would be to find the genetic variants that cause particular autistic phenotypes.

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Correspondence to: James Feldon, Department of Psychology, University of Rutgers, New Haven, USA, E-mail: jamesfeldon0213@gmail.com

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