Schizophrenias ankyrin 3 gene-based genetic modality of working memory deficiencies.

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Introduction

Schizophrenia is a complex and enigmatic mental disorder that has intrigued researchers, clinicians, and society for centuries. Characterized by a profound disruption of thought processes, emotions, and behaviors, this psychiatric condition affects millions of people worldwide, making it one of the most challenging mental health disorders to understand and treat effectively. Schizophrenia's symptoms often manifest during late adolescence or early adulthood, significantly impacting an individual's ability to function in everyday life and interact with others. Despite significant advances in medical research and neuroscience, the exact causes of schizophrenia remain elusive, leading to ongoing efforts to unravel its underlying mechanisms and develop innovative therapeutic approaches [1].

Due to its many different forms and enigmatic etiology, schizophrenia is a complicated and multidimensional mental condition that has long baffled researchers. The study of the ankyrin 3 (ANK3) genes and its potential role in working memory impairments associated with schizophrenia is one exciting research area that has developed in recent years. The ability of people with schizophrenia to process and retain important information for daily tasks is frequently hindered. Working memory is a vital cognitive function that is responsible for temporary storage and manipulation of information. Working memory deficiencies have been connected to anatomical and functional anomalies in the prefrontal cortex and hippocampus, two parts of the brain. These regions, which are crucial for working memory functions, have been carefully examined in relation to schizophrenia. However, until recent studies into the ANK3 gene, the precise genetic pathways behind these cognitive impairments remained obscure [2].

Ankyrin-G, which is encoded by the chromosome 10 gene ANK3, is essential for preserving the form and functionality of neuronal cells. Ankyrin-G contributes to the development and stabilization of axons and dendrites, laying the structural groundwork for effective electrical signal transmission between neurons. Ankyrin-G is therefore essential for normal synaptic plasticity and neuronal transmission in the brain areas related to working memory. Numerous genetic research, including genome-wide association studies (GWAS), have uncovered intriguing links between specific ANK3 gene variations and a higher risk of developing schizophrenia. Single-nucleotide polymorphisms (SNPs), which are genetic variations, may have an impact on ankyrin-G expression or function, altering neuronal connections and impairing working memory functions [3].

The molecular processes by which working memory problems in schizophrenia are caused by ANK3 gene variations are still being investigated. Changes in ankyrin-G expression or function have the potential to impair the growth and upkeep of neuronal circuits essential for working memory. This might result in a decreased capacity for synchronizing and integrating information, which would cause cognitive impairments typical of the condition [4].

It has been demonstrated that ankyrin-G interacts with other proteins involved in synaptic plasticity and transmission. The effectiveness of neuronal transmission and information processing in the brain may be further impacted by disruptions in these connections brought on by genetic differences. A fascinating and promising line of inquiry into the working memory deficits associated with schizophrenia has been revealed by studies into the significance of the ANK3 gene. We are getting a little bit closer to comprehending the intricate mechanisms at work in this puzzling condition as we delve deeper into the genetic mode driving cognitive abnormalities in schizophrenia. The link between the ANK3 gene and working memory issues sheds light on the neurological foundations of schizophrenia and may have implications for future therapeutic approaches [5].

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***Received:** 31-Jul-2023, Manuscript No. AACBM-23-109368; **Editor assigned:** 04-Aug-2023, PreQC No. AACBM-23-109368(PQ); **Reviewed:** 18-Aug-2023, QC No. AACBM-23-109368; **Revised:** 24-Aug-2023, Manuscript No. AACBM-23-109368(R); **Published:** 31-Aug-2023, DOI: 10.35841/aacbm-5.4.160*

***Citation:** Mossman M. A Schizophrenias ankyrin 3 gene-based genetic modality of working memory deficiencies. J Cell Biol Metab. 2023;5(4):160*