Clinical Psychiatry
ISSN: 2471-9854

Open access Perspective

Schizophrenia is a Chronic and Disabling Brain Disorder, thought to be a Heterogeneous Syndrome

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INTRODUCTION

Schizophrenia is a chronic, debilitating brain disorder classified as a heterogeneous syndrome. With a lifetime prevalence of approximately 1%, the onset of the disease is often early in adulthood. Many studies point to oxidative stress and redox dysregulation as risk factors for the development of schizophrenia. In addition to genetic predisposition to disease, quantitative and qualitative stress tolerance interacts with environmental influences and genetic factors, leading to metabolic imbalances in the body. Although several potential candidate genes and chromosomal binding sites for schizophrenia have been identified, little information is available on their molecular and cellular intermediates. Dicarbonyls such as methylglyoxal (MG), a potent protein saccharifying agent, are formed from sugars, lipids, and amino acids. This dicarbonyl accumulation, termed carbonyl stress, modifies proteins and ultimately leads to the formation of advanced glycation end products (AGEs). AGE formation is associated with three different pathways *in vivo*: The Maillard reaction, the polyol pathway, and lipid peroxidation.

DESCRIPTION

Several reports in humans suggest elevated AGEs in her in various diseases, including diabetes, hemodialysis, and psychiatric disorders. Cellular removal of dicarbonyls and AGEs is mediated by a glutathione-dependent glyoxalase, glyoxalase I. This system is also known to interact with multiple metabolic cascades, some of which have been described as possible causative factors. MG is a reactive glycosylation agent and a precursor of important quantitative adducts formed from proteins. Accumulation of MG induces increased protein activation and degradation, DNA mutagenesis, and cytotoxicity. Therefore, efficient metabolism and detoxification of MG is required to prevent intracellular protein and DNA damage. The glyoxalase pathway is the major pathway for MG metabolism and is assisted by aldose reductase and aldehyde dehydrogenase. As expected, heterozygous frameshift carriers in schizophrenia showed increased plasma pentosidine concentrations. A decrease in pentosidine and an increase in Glo1 enzymatic activity were observed in discharged patients. These pentosidine levels and her Glo1 enzymatic activity suggest that upregulation of Glo1 metabolism leads to suppression of carbonyl stress and ameliorate some symptoms in schizophrenic patients. If we can find bioactive substances that ameliorate the dysfunction of Glo1 metabolism, this too may prove to be of therapeutic value. Drug-naïve patients with mental status disorder (ARMS) with elevated plasma pentosidine levels and elevated carbonyl stress underscored the possibility that carbonyl stress was present prior to disease onset. The clinical features of schizophrenia with carbonyl stress suggest targeting this stress as a novel therapeutic for psychiatric disorders. Pyridoxamine, a non-toxic water-soluble vitamin B, is considered a new drug for carbonyl stress schizophrenia, mainly because it inhibits the formation of her AGEs.

CONCLUSION

Although further studies are needed to understand the molecular mechanisms triggered by carbonyl stress in the central nervous system, carbonyl stress undoubtedly represents a new target for drugs without neurotransmitter-based approaches in the treatment of schizophrenia. Pentosidine levels may benefit clinically from pyridoxamine treatment. A better understanding of the molecular mechanisms that drive the pathophysiology of carbonyl stress-related schizophrenia may lead to improvements in difficult-to-treat negative symptoms and cognitive impairment, thereby improving the quality of life of patients. Future omics research combining extensive molecular and clinical information will lead to new discoveries that will enable more precise diagnostic subdivision and subsequent treatment planning of schizophrenia.

Received:	02-January-2023	**Manuscript No:**	IPCP-23-15612
Editor assigned:	04-January-2023	**PreQC No:**	IPCP-23-15612 (PQ)
Reviewed:	18-January-2023	**QC No:**	IPCP-23-15612
Revised:	23-January-2023	**Manuscript No:**	IPCP-23-15612 (R)
Published:	30-January-2023	**DOI:**	10.35841/2471-9854.23.9.004

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Citation Kim D (2023) Schizophrenia is a Chronic and Disabling Brain Disorder, thought to be a Heterogeneous Syndrome. Clin Psychiatry. 9:004.

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ACKNOWLEDGEMENT

None.

CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.