# Article information:

Sarcosine (glycine transporter inhibitor) attenuates behavioural and biochemical changes induced by ketamine, in the rat model of schizophrenia | SpringerLink  
<https://link-springer-com.libproxy.ucl.ac.uk/article/10.1007/s00221-022-06530-4>

# Article summary:

1. Schizophrenia is a neurological disorder that affects behavior and quality of life, characterized by positive, negative, and cognitive symptoms.

2. The glutamatergic hypothesis proposes NMDA receptor hypofunction as a cause of schizophrenia, leading to the investigation of glycine as a co-agonist to NMDA receptors.

3. Sarcosine, a natural amino acid and glycine transporter inhibitor, was found to attenuate ketamine-induced behavioral impairments in a rat model of schizophrenia by alleviating oxidative stress, neuroinflammation, and mitochondrial dysfunction.

# Article rating:

Appears moderately imbalanced: The article provides some useful information, but is missing several important points or pieces of evidence that would be required to present the discussed topics in a balanced and reliable way. You are encouraged to seek a more balanced perspective on the presented issues by exploring the provided research topics and looking at different information sources.

# Article analysis:

The article titled "Sarcosine (glycine transporter inhibitor) attenuates behavioural and biochemical changes induced by ketamine, in the rat model of schizophrenia" discusses the potential therapeutic effects of sarcosine, a glycine transporter inhibitor, in treating schizophrenia. The study was conducted on rats that were induced with schizophrenia-like symptoms using ketamine.

The article provides a comprehensive overview of schizophrenia and its symptoms, highlighting the limitations of current antipsychotic medications in treating all three symptom domains. The authors propose the glutamatergic hypothesis as a possible target for treatment, specifically targeting NMDA receptor hypofunction through positive modulation of glycine co-agonist effect.

The study found that sarcosine reversed ketamine-induced behavioral impairments and ameliorated oxidative and nitrosative stress, mitochondrial dysfunction, and neuroinflammation. The authors suggest that sarcosine may attenuate the behavioral symptoms of schizophrenia by alleviating oxidative stress, neuroinflammation, and mitochondrial dysfunction established by ketamine.

While the study provides valuable insights into the potential therapeutic effects of sarcosine in treating schizophrenia, there are some limitations to consider. Firstly, the study was conducted on rats and may not necessarily translate to humans. Secondly, while the authors suggest that sarcosine may alleviate all three symptom domains of schizophrenia, only positive symptoms were evaluated in this study. Further research is needed to evaluate its effects on negative and cognitive symptoms.

Additionally, while the article provides a comprehensive overview of schizophrenia and its current treatment options, it does not explore other potential targets for treatment beyond NMDA receptor hypofunction. It also does not provide any counterarguments or limitations to the glutamatergic hypothesis.

Overall, while this study provides promising results for sarcosine as a potential treatment option for schizophrenia, further research is needed to fully evaluate its efficacy and safety in humans.

# Topics for further research:

* Alternative targets for treating schizophrenia beyond NMDA receptor hypofunction
* Limitations of current antipsychotic medications in treating negative and cognitive symptoms of schizophrenia
* Mechanisms of oxidative and nitrosative stress in schizophrenia
* Neuroinflammation and its role in the pathophysiology of schizophrenia
* Mitochondrial dysfunction in schizophrenia and its potential as a therapeutic target
* Clinical trials evaluating the efficacy and safety of sarcosine in treating schizophrenia in humans

# Report location:

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