# Article information:

Enhancing GluN2A-type NMDA receptors impairs long-term synaptic plasticity and learning and memory - PubMed  
<https://pubmed.ncbi.nlm.nih.gov/35484243/>

# Article summary:

1. A rare variant in the NMDAR subunit GluN2A (K879R) was identified in a patient with intellectual disability.

2. Expression of GluN2A\_K879R in mouse hippocampal CA1 neurons enhanced the excitatory postsynaptic currents mediated by GluN2A-NMDAR but suppressed those mediated by GluN2B-NMDAR and the AMPA receptor.

3. GluN2A\_K879R knock-in mice showed impaired learning and memory, as well as severely impaired long-term synaptic plasticity.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article is generally reliable and trustworthy, as it is based on research conducted by a team of experts from multiple institutions, including universities and hospitals. The authors have provided evidence to support their claims, such as data from experiments conducted on mice, which adds credibility to their findings. Furthermore, the authors have provided references to other relevant studies that support their conclusions.

However, there are some potential biases that should be noted. For example, the study only focused on one particular mutation in the NMDAR subunit GluN2A (K879R), so it may not be applicable to other mutations or variants of this gene. Additionally, since the study was conducted using animal models, its results may not be directly applicable to humans. Finally, while the authors have provided references to other relevant studies that support their conclusions, they do not explore any counterarguments or alternative explanations for their findings.

# Topics for further research:

* NMDAR GluN2A mutation
* NMDAR GluN2A K879R
* NMDAR GluN2A K879R effects
* NMDAR GluN2A K879R in humans
* NMDAR GluN2A K879R alternative explanations
* NMDAR GluN2A K879R counterarguments

# Report location:

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