# Article information:

CMT2D neuropathy is linked to the neomorphic binding activity of glycyl-tRNA synthetase - PubMed  
<https://pubmed.ncbi.nlm.nih.gov/26503042/>

# Article summary:

1. CMT2D is a type of peripheral neuropathy caused by mutations in the ubiquitously expressed enzyme glycyl-transfer RNA (tRNA) synthetase (GlyRS).

2. Mutations in GlyRS cause selective neuronal loss, leading to deficits in distal motor function.

3. The neomorphic binding activity of GlyRS(CMT2D) antagonizes an essential signalling pathway for motor neuron survival, linking the selective pathology of CMT2D to this aberrant interaction.

# 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 provides evidence for its claims through experiments and data analysis. The authors have conducted hydrogen-deuterium exchange analysis to compare P234KY-GlyRSCMT2D and GlyRSWT in solution, which showed a global increase (15%) in deuterium incorporation for the mutant GlyRS indicating overall structural opening. Additionally, genetic reduction of Nrp1 in mice worsened CMT2D symptoms, whereas enhanced expression of VEGF improved motor function. These findings provide strong evidence that the neomorphic binding activity of GlyRS(CMT2D) antagonizes an essential signalling pathway for motor neuron survival.

The article does not appear to be biased or one-sided, as it presents both sides equally and does not contain any promotional content or partiality. It also notes possible risks associated with the findings presented, such as potential side effects from treatments targeting the VEGF-Nrp1 signalling axis. However, there are some missing points of consideration that could be explored further, such as potential long-term effects of treatments targeting this signalling axis or other potential pathways that may be involved in CMT2D pathology. Additionally, more evidence could be provided to support the claims made regarding the neomorphic binding activity of GlyRS(CMT2D).

# Topics for further research:

* Long-term effects of VEGF-Nrp1 signalling axis treatments
* Alternative pathways involved in CMT2D pathology
* Neomorphic binding activity of GlyRS(CMT2D)
* Hydrogen-deuterium exchange analysis
* Genetic reduction of Nrp1 in mice
* Motor neuron survival pathways

# Report location:

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