# Article information:

Frontiers | Altered Sensory Neuron Development in CMT2D Mice Is Site-Specific and Linked to Increased GlyRS Levels
<https://www.frontiersin.org/articles/10.3389/fncel.2020.00232/full>

# Article summary:

1. Charcot-Marie-Tooth (CMT) is a hereditary peripheral neuropathy that affects 1 in 2,500–5,000 people and can be categorized as CMT1, CMT2, or intermediate CMT.

2. Mutations in more than 100 different genetic loci have been linked to CMT, with the largest protein family implicated being the aminoacyl-tRNA synthetase enzymes.

3. GARS1 is the first and best-studied ARS gene linked to CMT2D and mouse models for this condition display loss of lower motor neuron connectivity and disturbed neurotransmission at the neuromuscular junction, as well as pre-natal perturbation of sensory neuron fate in dorsal root ganglia.

# Article rating:

Appears well balanced: The article presents the information in a reliable and balanced way, without biases and prejudices. The claims made in the article are well supported and, where applicable, all sides of the argument are given opportunity to present their point of view. The article appears trustworthy and reliable.

# Article analysis:

The article provides an overview of Charcot-Marie-Tooth disease (CMT), its categorization into three types (CMT1, CMT2, and intermediate CMT), and its prevalence among people. It also discusses mutations in more than 100 different genetic loci that have been linked to CMT, with the largest protein family implicated being the aminoacyl-tRNA synthetase enzymes. The article then focuses on GARS1 as the first and best-studied ARS gene linked to CMT2D and how mouse models for this condition display loss of lower motor neuron connectivity and disturbed neurotransmission at the neuromuscular junction, as well as pre-natal perturbation of sensory neuron fate in dorsal root ganglia.

The article appears to be reliable overall; it cites relevant research studies throughout its text to support its claims about CMT's prevalence, categorization into three types, mutations in more than 100 different genetic loci that have been linked to it, GARS1's role in CMT2D specifically, and how mouse models for this condition display certain symptoms. The article does not appear to contain any promotional content or partiality towards any particular point of view; instead it presents both sides equally by discussing both pathogenesis hypotheses (loss-of-function vs gain-of-function) for GlyRS mutants causing neuropathy. Furthermore, possible risks are noted when discussing wild type GARS1 overexpression having no discernible rescue effect on neuromuscular pathologies while increased dosage of disease causing Gars alleles causes more severe neuropathy.

The only potential bias present in the article is that it does not explore counterarguments or missing points of consideration regarding its claims about mutations in more than 100 different genetic loci being linked to CMT or GARS1's role in CMT2D specifically; however this does not detract from its overall reliability since these points are outside the scope of what is discussed within the article itself.

# Topics for further research:

* Charcot-Marie-Tooth disease pathogenesis
* Charcot-Marie-Tooth disease treatment
* Charcot-Marie-Tooth disease symptoms
* GARS1 gene mutation
* GARS1 gene expression
* Neuromuscular junction pathology

# Report location:

<https://www.fullpicture.app/item/9ac3e39580caa5dd466c3353d00f35d3>