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Keywords

Peripheral Neuropathy – Charcot-Marie-Tooth disease – Crispr/Cas9 – transgenic mouse models – pharmacology

Science

Charcot-Marie-Tooth (CMT) disease is the most frequent hereditary peripheral neuropathy with a prevalence of 1:2500 individuals. Patients suffer from progressive motor and/or sensory disabilities, often need walking aids and become severely disabled early in life. To date, there is no cure for this neurological disorder.

CMT disease is divided into a demyelinating form (CMT1) and an axonal form (CMT2), depending on where the primary deficit takes place. Over 40 genes have been identified that can be causative for CMT neuropathy. Especially the axonal form of CMT shows great genetic heterogeneity with more than 140 mutations in 26 genes known to date and still over 50% of the patients with an unknown genetic variant.

How such a vast set of genes, causative for axonal CMT2, leads to exclusive degeneration of the peripheral nervous system is unknown. Various molecular processes and cellular pathways have been linked to axonal CMT, with several reoccurring themes: cytoskeleton and endoplasmic reticulum (ER) dynamics, mitochondrial homeostasis, axonal transport, endosomal sorting and mRNA processing. However, this list is growing quickly and, so far, these themes seem only applicable to small subsets of the causative genes. It therefore remains to be elucidated whether different CMT2 causal genes affect common underlying pathways leading to peripheral neurodegeneration.

This project will study common cellular mechanisms by means of parallel proteome analysis in models of axonal CMT.

Selected Publications

NextGen Voices: Science advocacy. *Science*: **344** (6179), 34-37. (2014)