The role of TMEM106B and CHMP2B in neuronal lysosomal trafficking in frontotemporal dementia

https://neurodegenerationresearch.eu/survey/the-role-of-tmem106b-and-chmp2b-in-neuronal-lysosomal-trafficking-in-frontotemporal-dementia/

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The role of TMEM106B and CHMP2B in neuronal lysosomal trafficking in frontotemporal dementia

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Research Abstract

Frontotemporal dementia (FTD) is the second most common form of dementia in individuals under the age of 65. Mutations in a number of genes can cause FTD. These genes are known to have quite distinct functions. How mutations in these diverse genes contribute to development of the same disease (FTD) is unknown. A recently published paper has shown that TMEM106B, a gene that increases the risk of developing FTD, causes defects in the ability of

cellular components to traffic to their correct destination within neurons. Preliminary data from our lab reveals that the FTD-causing gene CHMP2B causes similar defects in the same trafficking pathway. We would like to further characterise this defect, and investigate whether these two genes function in the same pathway. If this is the case, this would be the first data to suggest a common pathway leading to FTD, which would be a significant progression in understanding the mechanism of neurodegeneration in FTD.

Further information available at:

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