Body

The IND?s Kampmann lab [1] conducted the first genome-wide CRISPR-based screens in human neurons and uncovered a new pathway that protects neurons from oxidative stress. The findings were published in *Nature Neuroscience* in May.

Surprisingly, the screens found a neuron-specific role for the lysosomal protein prosaposin in controlling survival during oxidative stress. Prosaposin loss caused the formation of lipofuscin, a hallmark of aging, trapping iron and thereby generating ROS and triggering ferroptosis. Mutations in prosaposin have been previously linked to neurodegenerative diseases.

The project was led by Ruilin Tian, a graduate student in the Kampmann lab who recently started his own lab as an Assistant Professor at the Southern University of Science and Technology in Shenzhen, China.

The Kampmann lab collaborated with Data Tecnica International to create CRISPRbrain [2], a data commons that enables the scientific community to access, explore, visualize, and cross-compare the results of CRISPR screens in different human cell types.

Read the UCSF press release here.[3]

Read a discussion of this research by the Alzheimer?s Forum here [4].

Reference: Ruilin Tian+, Anthony Abarientos, Jason Hong, Sayed Hadi Hashemi, Rui Yan, Nina Dräger, Kun Leng, Mike A. Nalls, Andrew B. Singleton, Ke Xu, Faraz Faghri, Martin Kampmann+. Genome-wide CRISPRi/a screens in human neurons link lysosomal failure to ferroptosis. Nat Neurosci (2021). https://doi.org/10.1038/s41593-021-00862-0[5]

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Links
[1] http://kampmannlab.ucsf.edu/
[2] https://crisprbrain.org/
[3] https://www.ucsf.edu/news/2021/05/420576/full-genome-crispr-screen-reveals-surprising-ways-neurons-survive-oxidative
[4] https://www.alzforum.org/news/research-news/dysfunctional-lysosomes-cause-ferroptosis-neurons
[5] https://doi.org/10.1038/s41593-021-00862-0