

Body

Spreading of tau aggregates between cells is thought to be an important mechanism by which Alzheimer's disease and several other neurodegenerative diseases progress. A major question is which cellular pathways mediate or counteract this process.

In a project led by postdoc John Chen in the Kampmann lab [1], a CRISPR-based genetic screen revealed an important role for the ESCRT pathway to prevent the escape of internalized tau aggregates from the endolysosomal pathway into the cytosol, where they can seed more tau aggregation. Collaborators included the Gestwicki [2] and Southworth [3] labs at the IND, the Grinberg [4] lab at UCSF, and the Leonetti [5] lab at the Chan Zuckerberg Biohub.

The study was published in the *Journal of Biological Chemistry*. [6]

Reference:

Chen JJ, Nathaniel DL, Raghavan P, Nelson M, Tian R, Tse E, Hong JY, See SK, Mok SA, Hein MY, Southworth DR, Grinberg LT, Gestwicki JE, Leonetti MD, Kampmann M. Compromised function of the ESCRT pathway promotes endolysosomal escape of tau seeds and propagation of tau aggregation. *J Biol Chem*. 2019 Oct 2. pii:jbc.RA119.009432. doi: 10.1074/jbc.RA119.009432. [Epub ahead of print] PubMed PMID: 31578281.

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Links

[1] <https://kampmannlab.ucsf.edu/>

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[4] <https://grinberglab.ucsf.edu/>

[5] <https://www.czbiohub.org/manuel-leonetti-intracellular-architecture/>

[6] <http://www.jbc.org/content/early/2019/10/02/jbc.RA119.009432.abstract>