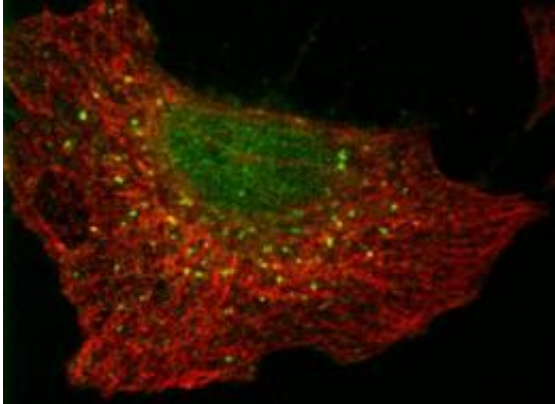


The downside of microtubule stability

June 15 2009



In a cell lacking dynamin 2, the pre-Golgi vesicles (green spheres) remain dispersed. Credit: Tanabe, K., and K. Takei. 2009. *J. Cell Biol.* doi:10.1083/jcb.200803153.

Stalled microtubules might be responsible for some cases of the neurological disorder Charcot-Marie-Tooth (CMT) disease, Tanabe and Takei report in the *Journal of Cell Biology*. A mutant protein makes the microtubules too stable to perform their jobs, the researchers find.

The mutations behind CMT disease slow nerve impulses, reduce their strength, or both. One of these [mutations](#) leads to production of faulty dynamin 2, a [protein](#) that is crucial for endocytosis but also latches onto microtubules. Tanabe and Takei investigated how defective dynamin 2 hampers cells.

Normal microtubules are continually extending and shrinking. But

microtubules from cells that made the faulty version of dynamin 2 were abnormally stable, as measured by how many acetyl groups were attached to them. The researchers also found that blocking normal dynamin 2 with RNAi had the same effect as the mutation, confirming that one of dynamin 2's functions is to promote microtubule turnover.

Removing dynamin 2 shattered the Golgi complex, Tanabe and Takei discovered. Dynamic microtubules help construct the Golgi complex in two ways: they capture the vesicles that combine to form a mature Golgi complex; and they provide a track along which these vesicles can travel to their rendezvous point near the [nucleus](#). By breaking up the Golgi apparatus and then watching the fragments reunite, the researchers found that dynamin 2 was essential for the capture step, not for transportation. Dynamin 2 also clings to microtubules of the mitotic spindle, and the team next wants to determine whether the protein regulates microtubule dynamics during the cell cycle.

Source: Rockefeller University Press

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