The downside of microtubule stability

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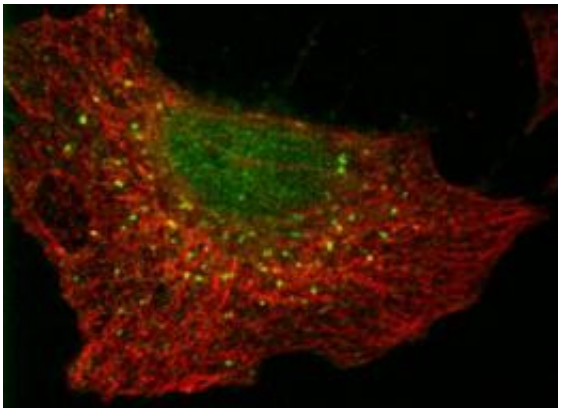

In a cell lacking dynamin 2, the pre-Golgi vesicles (green spheres) remain dispersed. Credit: Tanabe, K., and K. Takei. 2009. J. Cell Biol. doi:10.1083/jcb.200803153.

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 microtuble dynamics during the cell cycle.

Source: Rockefeller University Press