A molecular mechanism involved in cellular proliferation characterized

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Researchers from Guillermo Montoya's team at the Spanish National Cancer Research Centre (CNIO), in collaboration with Isabelle Vernos' Group from the Centre for Genomic Regulation (CRG), have uncovered the molecular interaction between TACC3 and chTOG, key proteins in forming the internal cellular framework that enables and sustains cell division. Published today in *Nature Communications*, the observations may help to optimise current oncological therapies specifically designed to fight against this framework, named by the scientific community as microtubules.

Key molecules for cellular proliferation

"During cell division, alterations in microtubule formation may bring about chromosome instability and aneuploidy. In other words, alterations in the number of chromosomes, which can lead to a tumour process," explains Montoya. "This is an underlying cause of tumours."

While the role of chTOG in microtubule assembly during cell division has been widely studied, not much is known regarding TACC3 and its contribution to the process.

The team of researchers uncovered the molecular basis of the interaction between these proteins, and how TACC3 recruits chTOG to the microtubules during cellular division. "Our results indicate that TACC3's function completely depends on this interaction, so that mutations in the latter prevent chTOG from correctly incorporating into the microtubules," states Montoya. Analyses were performed on the frog *Xenopus laevis*, an animal model widely used by researchers from around the world to study laws governing cellular division in depth.

Relevance in cancer research

One of the most used and most effective strategies in cancer treatment are drugs targeting microtubules, which halt the growth of tumour cells and induce apoptosis or programmed cellular death.

"Our study on the TACC3-chTOG interaction will allow cellular biologists and researchers on microtubule dynamics to better understand how microtubule assembly is regulated during cellular division," says Montoya, and he anticipates that "it could also help in developing new anti-microtubule drugs, providing more effective therapeutic options in cancer treatment."

More information: The XTACC3-XMAP215 association reveals an asymmetric interaction promoting microtubule elongation. Gulnahar B. Mortuza, et al. *Nature Communications* (2014)

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