

The journey of a cluster of GPI-anchored proteins around the cell membrane includes occasional stops (arrows).

membrane. Scientists suspect that these structures permit communication, but they do not understand the mechanism.

By treating fibroblasts with gold particles coated in antibodies, Chen et al. forced two kinds of GPIAPs to clump. These clusters halted for up to 10 seconds, a behavior dependent on cholesterol and Src kinases.

The authors hypothesize that, during the stops, proteins in the patches connect to the underlying cytoskeleton. Supporting that conclusion, the cystic fibrosis transmembrane conductance regulator, which does span the membrane, also shows the intermittent stops. But a version that cannot bind to the cytoskeleton just keeps drifting. The researchers propose that clusters unite GPIAPs with a membrane-bridging molecule that links to the cytoskeleton. The identity of this connector remains elusive, however. **JCB**

Brief encounter

At first glance, some of the cell's communication proteins seem ill-suited for the job. They are embedded in the outer layer of the plasma membrane and have no direct connection to the cell interior. Yet they manage to relay signals across the membrane. The proteins tend to congregate, and on page 169, Chen et al. show that this gregariousness might enable them to link to the cytoskeleton, a step necessary to pass on messages.

The study focused on GPI-anchored proteins (GPIAPs), some of which interconnect and spur cell division. Cross-linked GPIAPs band together with lipids and other molecules to form clusters that travel together around the

Division of labor

More than 100 proteins collaborate to build the kinetochore. Using RNAi, Liu et al. (page 41) sort out the responsibilities of key proteins in this process.

The kinetochore is a three-layered disc that sits on either side of the centromere. It links to spindle fibers and helps align and separate mitotic chromosomes. Although researchers have teased out the roles of some kinetochore proteins, they lacked a comprehensive picture of how these molecules interact to assemble the structure.

Liu et al. picked 20 putative kinetochore big shots and knocked them down, one at a time, using RNAi. The team then merged its results with past findings to sketch a map of the connections. Sitting atop the protein hierarchy is CENP-A, which permanently resides on centromeres. At the next level, three branches split off: two are headed by other centromere fixtures, CENP-I and CENP-C, and the third under the direction of the Aurora B kinase, a passenger protein crucial for chromosome separation.

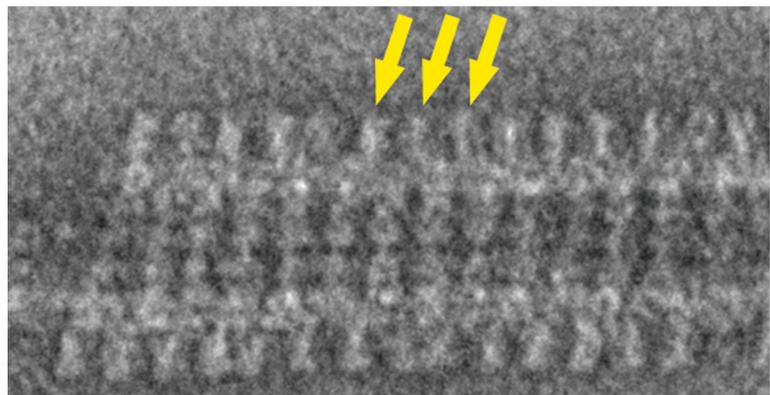
Each branch takes on a different task. CENP-I establishes the three-layered organization, for example. Multiple cross-links tie the branches together, however, so there is no linear chain of command. The researchers are now investigating whether interacting proteins make direct contact or whether other molecules serve as intermediaries. The interaction map might be useful to pharmaceutical researchers developing anticancer drugs that disrupt kinetochore proteins. The map may point the way to biomarkers for monitoring the drugs' effects. **JCB**

Ring around the tubule

The kinesin protein family is in the trucking business, hauling vesicles and other cellular cargo along the microtubules. The family's black sheep is kinesin-13, which disassembles the filaments while riding on them. Tan et al. report on page 25 that kinesin-13 is unusual in another way. The protein forms rings and spirals around microtubules. The structures might help it maintain its position as a tubule shrinks.

By shortening microtubules, kinesin-13 helps chromosomes to go their separate ways during mitosis. But how kinesin-13 performs the job is mysterious. The researchers spotted the rings and spirals when they combined microtubules with the motor domain of kinesin-13, the protein segment that latches onto the filament. Other types of kinesin did not coil up, the scientists showed. Molecular reconstructions suggest that each ring consists of several kinesin-13 molecules encircled by a strand of free tubulin, the building block of microtubules.

Why kinesin-13 gets into a twist is not certain, but Tan et al. speculate that the formation slides along the microtubule like a sleeve, keeping kinesin-13 at the end of the shortening tubule. To test that possibility, the researchers tagged kinesin-13 with green fluorescent protein. As the microtubule depolymerized, its tip glowed brighter, indicating that the rings were bunching up at the end. **JCB**



Coils of kinesin-13 (arrows) surround a microtubule.