

The Shape-Shifting Mechanics of Cells

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Cell biologists at Johns Hopkins have discovered how tiny molecular motors within cells work together with other structural players to coordinate critical cell shape changes that accompany cell division. The work appears in the April 8 issue of [*Current Biology*](#).

"Cell division is a classic movement that all cells must do, so we studied how several key proteins move during cell division to learn more about the mechanics of cell shape change," says [Douglas Robinson, Ph.D.](#), an associate professor of cell biology at the Johns Hopkins School of Medicine.

Some cells like yeasts divide by pinching themselves into two, like cinching a purse string. The purse string is made up of rope-like rings of the actin protein within the cell. But Robinson's team studies the social amoeba *Dictyostelium*, whose cells are similar to human cells but are much easier to manipulate genetically. Using high-power microscopes, the researchers discovered that rather than forming purse string-like rings, actin in *Dictyostelium* cells organizes into mesh-like structures in the region where the cell will divide.

"This was a surprise," says Robinson, "because the textbooks teach us that the myosin-II motor slides along and pulls these actin purse strings together to pinch a dividing cell, but we couldn't imagine a way that the same mechanics could work with short segments of actin in a weblike structure."

To figure out how the myosin-II motor contracts an actin mesh during cell division, the research team looked at cells containing, instead of normal myosin-II, an altered form of myosin-II that works 10 times slower than normal, cells lacking myosin-II altogether and normal cells. They found that cells lacking myosin-II divided the fastest, which suggested that myosin-II isn't the only thing controlling cell shape change during division.

"Cells are like liquid-filled balloons with a consistency thicker than water and closer to honey or Jell-O," says Robinson, "so we decided to look at the deformability of each of these different cells." To do this, they used tiny capillary pipettes that "like straws sucking on the surface" can measure the hardness or softness of the cell.

The researchers found that cells lacking myosin-II are more deformable than normal cells. Normal cells in the process of dividing, however, were stiffer near the division site than elsewhere in the cell.

The team then examined the deformability of cells containing or lacking so-called actin crosslinkers, proteins that bind to both the actin meshwork and myosin-II. They found that cells lacking the crosslinkers were more deformable, suggesting that myosin-II works with crosslinkers to increase tension and, with that, increase elasticity to enable the cell to change shape where it's dividing.

"Structure itself is not the critical part," says Robinson. Instead, structure may be the effect rather than the cause of the shape change.

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Authors on the paper are Elizabeth M. Reichl, Michael Delannoy, Janet C. Effler, Kristine D. Girard, Srikanth Divi, Pablo A. Iglesias, Scot C. Kuo, and Douglas N. Robinson, all of Johns Hopkins, and Mary K. Morpew of the University of Colorado.

On the Web:

<http://www.hopkinsmedicine.org/cellbio/robinson/doug.html>

<http://www.current-biology.com/>