Heterochromatin gets dynamic

The sleeping giant of the cell, heterochromatin, is really a bustling complex whose components turn over rapidly, say two groups led by Richard Festenstein (Imperial College, London) and Thierry Cheutin and Tom Misteli (National Cancer Institute [NCI], Bethesda, MD).

Both groups used fluorescence recovery after photobleaching (FRAP) to track the behavior of heterochromatin protein 1 (HP1). HP1 was not static but moving around, as Misteli puts it, in the “same sort of range” as transcription factors. Although both groups found that HP1 was dynamic, Festenstein’s mobility values were lower than those found by Misteli. Part of the difference may lie in varying bleaching and microscopy methods, but both researchers suggest the importance of the different cell types used. Festenstein looked in primary T cells—a resting cell type—and when he stimulated the T cell receptor to activate these cells, his new values for HP1 mobility increased almost to the level seen by Misteli in his transformed cells. Thus, an increase in heterochromatin protein dynamics may help kick-start a T cell as it takes on invading microbes.

The details of how cell activation increases mobility remain to be determined, but the overall high level of mobility is already causing a rethinking of standard models of heterochromatin. The important point, says Misteli, is not to confuse stable with static. “We think most stable structures are dynamic,” he says. “You can generate stable structures from dynamic components.” The dynamic behavior may, however, indicate that dimers or small oligomers of HP1 may be the building blocks of heterochromatin, rather than the large oligomeric networks portrayed in many textbooks.

Also up for grabs is the accessibility of both heterochromatic DNA and the ever-important histone tails (the target of HP1 binding). With the dynamic behavior of HP1, says Festenstein, “now you get an opportunity for access.” Thus, heterochromatin is not dormant but subject to constant competition for binding between HP1 and the activators and remodeling factors that seek to bring DNA into the open.

References: Cheutin, T., et al. 2003. Science. 299:721–725.
Festenstein, R., et al. 2003. Science. 299:719–721.

Life on a bed of needles

Cells pull on their environment during adhesion, contraction, or movement, but mapping those forces is no easy task. Past methods have relied on the wrinkling or deformation of a pliant substrate—akin to the scrunching of a sheet when someone sits down on a bed. Now, John Tan, Christopher Chen, and colleagues (Johns Hopkins University, Baltimore, MD) have put forward an alternative method using arrays of microfabricated, bendy posts.

The tops of the posts are coated with fibronectin using microprinting, thus keeping the cells restricted to the tops. This may be more physiological than it sounds. “The way cells seek out and find these posts may be similar to the way cells seek out collagen fibers in a loose network,” says Chen. Limited microprinting restricts the spreading of the cells. Such cells could not contract in response to serum, yet still responded to activated RhoA.

Reference: Tan, J.L., et al. 2003. Proc. Natl. Acad. Sci. USA. 10.1073/pnas.0235407100.