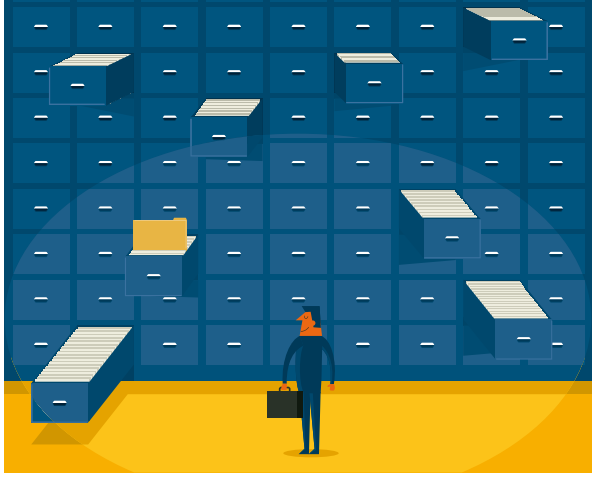

Credit: Sorbetto/Getty

This study suggests that acetylated histones and their binding proteins, but not transcription, help establish a BRD4-NUT nuclear subcompartment, and that high levels of gene expression can be achieved merely by residing in this subcompartment. MZ1 is similar to compounds used to treat NUT carcinomas and other cancers, and the data indicate that the mechanism of action of these anti-cancer drugs could involve the disruption of chromatin folding and nuclear subcompartments.

Eytan Zlotorynski

ORIGINAL ARTICLE Rosencrance, C. D. et al. Chromatin hyperacetylation impacts chromosome folding by forming a nuclear subcompartment. *Mol. Cell* **78**, 112–126 (2020)

> reduction in mitochondrial fragmentation … was accompanied by failure of membrane repair

the injury site. This was associated with decreased mROS levels following injury. Notably, global increase in mROS generation in *Drp1*-knockout MEFs did not rescue the membrane repair deficiency in these cells, highlighting the importance of localized mROS release in this process.

In summary, plasma membrane injury induces localized mitochondrial fission and mROS signalling, which is key to efficient membrane repair. Mitochondrial dynamics, including the opposing processes of fusion and fission, are tightly coupled with cell function and behaviour. It will be interesting to learn to what extent dynamic changes to the mitochondrial network contribute to other localized cellular processes.

Paulina Strzyz

ORIGINAL ARTICLE Horn, A. et al. Mitochondrial fragmentation enables localized signaling required for cell repair. *J. Cell Biol.* **219**, e201909154 (2020)
RELATED ARTICLES Giacomello, M. et al. The cell biology of mitochondrial membrane dynamics. *Nat. Rev. Mol. Cell Biol.* **21**, 204–224 (2020) | Sies, H. & Jones, D. P. Reactive oxygen species (ROS) as pleiotropic physiological signalling agents. *Nat. Rev. Mol. Cell Biol.* https://doi.org/10.1038/s41580-020-0230-3 (2020)

Journal Club

LOOKING DEEPER INTO TISSUE ELONGATION

Epithelia are the primary tissues that, through stretching, bulging, folding and layering, give animals their forms. Much of the study of morphogenesis has focused on the versatile shape-change toolkit epithelia use to accomplish these rearrangements. These studies are dominated by the use of advanced microscopy techniques. However, epithelial cells are not the near-2D pancakes that microscopists prefer, and the problem of light scattering deep in tissues is a tough one. Consequently, we often take the 3D problem of cell rearrangements and see how much of it we can account for with the events happening just under the coverslip.

One model in which to study epithelial tissue rearrangements is the embryonic ectoderm of *Drosophila melanogaster*. This epithelium rapidly becomes narrower and longer to extend the body axis through a process called convergent extension, which occurs during the development of many animals. To do this, cells move between their neighbours preferentially along the shortening axis (Irvine & Wieschaus, 1994). In the early fly embryo, the apical surface of the ectoderm is most accessible for imaging. Because of this, most studies to date have focused on investigating molecular drivers of convergent extension in this plane, revealing that neighbour exchanges are driven by myosin-dependent contraction of adherens junctions along the shortening axis and formation of new junctions along the elongating axis (Bertet et al., 2004; Zallen & Wieschaus, 2004).

> both the formation of basal protrusions and apical junction contraction are required for full convergent extension

An exciting paper from Sun et al. (2017) shows that there is more to this story. Using two-photon microscopy to image whole cell volumes over time, they found that neighbour exchange often initiates at the basal surface, and apical junction exchange lags behind. At the basal surface, neighbour exchange is driven by polarized localization of phosphorylated Src42A kinase, which acts upstream of the Rho GTPase Rac1 to generate basal protrusions that cells extend between their neighbours in the direction of ultimate neighbour exchange. After these protrusions make contact, the junction zippers upwards, assisted at the apical surface by contraction of cell–cell junctions as described above. Notably, both the formation of basal protrusions and apical junction contraction are required for full convergent extension. These findings extend work from vertebrate systems (see especially Williams et al., 2014) and provide the strongest case yet for the contribution of both apical and basal cell behaviours to convergent extension. This paper is a nod to the under-appreciated dynamicity of the epithelial basal surface. It is also a nice reminder to look at the processes we study from new perspectives — there is a lot going on beneath the surface.

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ORIGINAL ARTICLE Sun, Z. et al. Basolateral protrusion and apical contraction cooperatively drive *Drosophila* germ-band extension. *Nat. Cell Biol.* **19**, 375–383 (2017)
RELATED ARTICLES Bertet, C., Sulak, L. & Lecuit, T. Myosin-dependent junction remodelling controls planar cell intercalation and axis elongation. *Nature* **429**, 667–671 (2004) | Irvine, K. D. et al. Cell intercalation during *Drosophila* germband extension and its regulation by pair-rule segmentation genes. *Development* **120**, 827–841 (1994) | Williams, M. et al. Distinct apical and basolateral mechanisms drive PCP-dependent convergent extension of the mouse neural plate. *Dev. Cell* **29**, 34–46 (2014) | Zallen, J. A. & Wieschaus, E. Patterned gene expression directs bipolar planar polarity in *Drosophila*. *Dev. Cell* **6**, 343–355 (2004)