CellPress

Plant Cell Shape: Trafficking Gets Edgy

Ramin Rahni[1] and Kenneth D. Birnbaum[1,*]
[1]Department of Biology, Center for Genomics and Systems Biology, New York University, 12 Waverly Place, New York, NY 10003, USA
*Correspondence: ken.birnbaum@nyu.edu
http://dx.doi.org/10.1016/j.devcel.2016.02.005

Polyhedral-shaped plant cells have faces, corners, and edges that can have different material properties. As Kirchhelle et al. (2016) now show, RAB-A5c reveals a trafficking compartment that localizes to the edges where two cell walls meet, with a potential role in mediating local wall stiffness.

Plants exhibit a stunning diversity of organ shapes that are generated, in part, from the geometries of their constituent cells. While animal cells can be likened to beanbags that achieve shape through the internal support of their cytoskeleton, plant cells are like soda cans or cardboard pizza boxes, in which rigid exterior walls provide structural support and, along with turgor pressure, dictate shape. The plant cell wall must also resist or respond to changes in tension, compression, and shear, due to cell division and expansion as organs take shape during development. Previous studies have suggested that the junctions of two planar cell walls—the geometric edges—may have special mechanical properties, perhaps to deal with these dynamic forces (Routier-Kierzkowska et al., 2012). However, little is known about the trafficking mechanisms that could regulate plant cell geometry.

In the current issue of *Developmental Cell*, Kirchhelle et al. (2016) uncover properties of a RAB-GTPase, RAB-A5c, that suggest a role at the edges where planar cell walls meet (Figure 1). The Rab family of Ras-like small GTPases is known to function in intracellular transport in both plants and animals (Nielsen et al., 2008). RAB-A5c is preferentially expressed in the shoot meristem and emerging lateral root meristem, which rapidly attains a conical organ shape that pushes through the outer layers of the parent root. Lateral roots, which the authors used for their in vivo analysis, are feasibly under extreme and dynamic mechanical forces during their development (i.e., this tissue experiences an internal rise in turgor pressure that enables the lateral root primordium to push through the outer cell layers [Péret et al., 2012] and a reciprocal mechanical force applied by the overlying tissues [Lucas et al., 2013]).

Interestingly, quantitative analysis of the distribution of the tagged version of the protein under its own regulatory elements—YFP-RAB-A5c—showed preferential localization to the geometric edges of cells in the developing lateral root. RAB-A5c was previously characterized in pollen, where it appeared to localize to the trans-Golgi network (TGN) (Ueda et al., 1996). In the current study, the authors showed that in young lateral roots, in addition to localization in the TGN and cell plate, RAB-A5c also localized to unique domains, distinct from endosomal, Golgi, or prevacuolar compartments. They speculate, through a series of localization and perturbation experiments, that the protein traffics from the TGN to an edge-localized compartment via the exocytic pathway (Figure 1).

RAB-A5c is not the first protein shown to preferentially localize to plant cell edges. CLASP proteins were previously shown to play a role in stabilizing cortical microtubules at sharp cell edges (Ambrose et al., 2011). However, the authors showed that localization of RAB-A5c is not altered in CLASP mutants, suggesting an independent mechanism of edge regulation. Altogether, RAB-A5c appears to mark a specialized vesicle population that traffics to cell edges.

To explore the function of RAB-A5c in the face of apparently high redundancy in the gene family, the authors used an inducible mutation in a YFP-RAB-A5c construct that appeared to sequester the protein in the TGN, away from its edge-localized compartments, acting as a dominant perturbation. In this background, the shapes of initiating lateral root cells were severely abnormal, often losing their box-like appearance in favor of more globular shapes. Cell walls also showed abnormal angles, consistent with the presence of RAB-A5c in the TGN, where it is involved in cell plate formation. Importantly, the wild-type version of YFP-RAB-A5c, which had initially defined the edge compartments, could quantitatively rescue the dominant-mutant form when expressed at higher levels. The work implicates an edge-trafficking mechanism for RAB-A5c, which, in turn, appears to have a role in cell shape. The intriguing possibility is that RAB-A5c uncovers a role for a trafficking pathway that regulates the mechanical properties of edges, which would allow the cell to attain proper shape. For instance, recent work has shown that modification of pectins in the cell wall can alter cell wall elasticity during organ formation (Peaucelle et al., 2011).

Many critical questions remain about the role of RAB-A5c and its specificity to edge mechanics. In probing the connection between edge integrity and overall cell shape, the authors used a finite element model to explore the effect of loosening cell wall edges; the model showed that edge loosening led to radial distension of cells, similar to the blob-like cells seen with the dominant-mutant form of RAB-A5c. This shows that softening edges could feasibly lead to a structural breakdown that affects whole-cell morphology. Still, previous work using automated indentation techniques to measure wall stiffness has shown that the edge where two cell wall faces meet actually already exhibits reduced stiffness in wild-type cells (Routier-Kierzkowska et al., 2012). This may be a contradiction or could simply reflect the fact that edge stiffness needs to be tightly regulated. Further work is needed to show that cell-shape deformations in the RAB-A5c perturbations are due to "edge effects" rather than whole cell wall defects.

It will be critical going forward to further probe the role of the RAB-A5c

compartments in edge trafficking specifically. This will require additional characterization of these edge compartments, as well as finding perturbations that can target them exclusively. How does the RAB-A5c compartment expressly affect the mechanical properties of cell edges in terms of edge loosening or stiffening? In addition, the precise route and destination of the RAB-A5c compartments remains somewhat unclear. And, critically, if these compartments are specifically bound for the plasma membrane or nearby regions, what is their cargo?

Interestingly, the Rab-A clade of highly conserved RAB GTPases has undergone extensive radiation in plants. One possibility is that some of this radiation, including RAB-A5c, may represent innovations in ancestral trafficking mechanisms that evolved to cope with cell wall requirements. Thus, RAB-A5c has the potential to shed light on trafficking mechanisms that can be targeted to highly specific regions of the cell—a problem common to both plant and animal developmental cell biology. In addition, the RAB-A5c compartments could shed light on how plants adjust the mechanical properties of cell walls locally to maintain stable shapes.

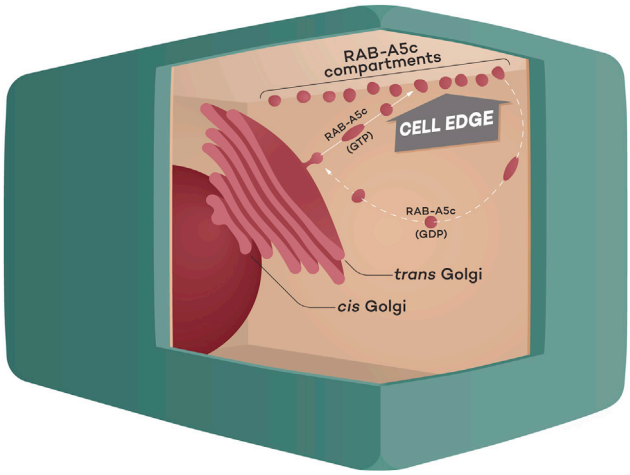

Figure 1. Localization and Speculated Trafficking Route of RAB-A5c

Quantitative microscopy showed that a tagged version of RAB-A5c localized to the geometric edges of cells where two planar walls meet. Experiments based on mutated forms of RAB-A5c suggested that the GDP-bound form may be recruited from the cytoplasm to the TGN. There, interaction with its nucleotide exchange factor would lead to a GTP-bound state, with subsequent trafficking on an exocytic pathway to the edge compartments. Following GTP hydrolysis, the protein would cycle back to the cytosol. (Graphic by Ramin Rahni.)

REFERENCES

Ambrose, C., Allard, J.F., Cytrynbaum, E.N., and Wasteneys, G.O. (2011). Nat. Commun. *2*, 430.

Kirchhelle, C., Chow, M.C., Foucart, C., Neto, H., Stierhof, Y.D., Kalde, M., Walton, C., Fricker, M., Smith, R.S., Jérusalem, A., et al. (2016). Dev. Cell *36*, this issue, 386–400.

Lucas, M., Kenobi, K., von Wangenheim, D., Vobeta, U., Swarup, K., De Smet, I., Van Damme, D., Lawrence, T., Peret, B., Moscardi, E., et al. (2013). Proc. Natl. Acad. Sci. USA *110*, 5229–5234.

Nielsen, E., Cheung, A.Y., and Ueda, T. (2008). Plant Physiol. *147*, 1516–1526.

Peaucelle, A., Braybrook, S.A., Le Guillou, L., Bron, E., Kuhlemeier, C., and Höfte, H. (2011). Curr. Biol. *21*, 1720–1726.

Péret, B., Li, G., Zhao, J., Band, L.R., Voß, U., Postaire, O., Luu, D.T., Da Ines, O., Casimiro, I., Lucas, M., et al. (2012). Nat. Cell Biol. *14*, 991–998.

Routier-Kierzkowska, A.L., Weber, A., Kochova, P., Felekis, D., Nelson, B.J., Kuhlemeier, C., and Smith, R.S. (2012). Plant Physiol. *158*, 1514–1522.

Ueda, T., Anai, T., Tsukaya, H., Hirata, A., and Uchimiya, H. (1996). Mol. Gen. Genet. *250*, 533–539.

A GPCR Handles Bacterial Sensing in Chemotaxis and Phagocytosis

Hiroshi Senoo,[1] Hiromi Sesaki,[1] and Miho Iijima[1,*]
[1]Department of Cell Biology, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA
*Correspondence: miijima@jhmi.edu
http://dx.doi.org/10.1016/j.devcel.2016.02.006

In this issue of *Developmental Cell*, Pan et al. (2016) identified in cells of the social amoeba *Dictyostelium* a G protein-coupled receptor (GPCR) that recognizes a chemoattractant secreted by bacteria. This work uncovers a mechanism by which a single GPCR mediates pseudopod extension during cell migration and bacterial engulfment.

Chemotaxis and phagocytosis are important processes for removing infecting bacteria and clearing dying apoptotic cells in the human body (Bloes et al., 2015; Muñoz et al., 2010). For example, immune cells such as macrophages, neutrophils, and dendritic cells sense, chase, and eat bacteria through directed cell migration toward bacteria (chemotaxis) and engulfment of bacteria (phagocytosis). These two mechanisms involve common processes such as the