Conservation of boundary extension mechanisms between plants and animals

Jaideep Mathur
Molecular Cell Biology Lab, Department of Plant Agriculture, University of Guelph, Guelph, Ontario N1G 2W1, Canada

Locomotion clearly sets plants and animals apart. However, recent studies in higher plants reveal cell-biological and molecular features similar to those observed at the leading edge of animal cells and suggest conservation of boundary extension mechanisms between motile animal cells and nonmotile plant cells.

Boundary extension: a necessity for living organisms
Survival requires a constant refurbishment of resources. Therefore, living organisms persistently explore their environment for extending their boundaries into favorable regions. One fundamental difference we are taught early on between motile animal and nonmotile plant cells is the way in which they carry out their boundary extension. Motile cells achieve whole-body displacement or locomotion, which allows them to leave resource-depleted domains behind while moving on to new areas. Plant cells, however, become rooted to a spot and are only able to extend outwards from it in response to different stimuli.

Two major conditions have to be met for achieving locomotion: a flexible bounding layer of the cell, which allows for rapid change in its shape, and the ability to retract one end of the cell while extending the other (Small et al., 2002a). Plant cells do not fulfill these requirements because they are encased in a relatively rigid cellulosic cell wall and, clearly, they do not have rear-end retractability. However, the cell wall of actively expanding plant cells is actually quite labile in contrast to its later, rigid nature in a mature, fully expanded cell (Mathur, 2004). Further, though lacking rear-end retractability, the forward extension of a plant cell, like that of an animal cell, also involves membrane protrusion (Vidali et al., 2001).

Recent cell-biological and molecular evidence from higher plants suggests that, despite their sessile and wall-encased nature, plant cells possess a core machinery similar to the one required for forward motility of animate cells.

Boundary extension requires a leading edge
Although usually used in the context of crawling amoeboid locomotion, here I use the term “leading edge” for both animal and plant cells as the part of a cell that extends or “leads.”

Though descriptions vary from cell to cell, the generalized leading edge of an amoeboid cell comprises a 2–5-μm-wide veil-like, organelle free, cytoplasmic extension called the lamellipodium. The leading edge has an actin-rich zone comprising of a fine F-actin mesh followed by a microtubule-rich region (Etienne-Manneville, 2004) (Fig. 1 A). A few pioneering microtubules do extend into the F-actin mesh (Small et al., 2002b; Raftopoulou and Hall, 2004).

A very similar intracellular zonation is seen in plants (Fig. 1, B–D; Mathur, 2004). For plant cells, two kinds of expansion modes are recognized; one called tip-growth, where the growth processes is limited to a small region that extends to form a tubular structure, and the second, designated diffuse growth, where the process of growth is dispersed over a large area of the cell (Mathur, 2004). Tip-growing cells (Fig. 1, B and D) are best typified by elongating root-hair and pollen tubes and, like lamellipodia, exhibit an apical region with a stretched plasma membrane, followed by an organelle-depleted clear zone (Vidali and Hepler, 2001). A fine, labile F-actin zone is defined next and leads into a dense F-actin region where the filaments become progressively bundled (Fu et al., 2001; Ketelaar et al., 2003). In active tip-growing cells, most cytoplasmic microtubules extend only to the edge of the fine F-actin mesh (Sieberer et al., 2002). A large vacuole fills the rest of the cell (Carol and Dolan, 2002). By comparison, the volume occupied by vacuoles is quite large in diffuse-growing cells so that the cytoplasm is compressed into a thin layer against the plasma membrane (Fig. 1, B and C; Mathur et al., 2003a,b). Although this thin cytoplasmic layer has not allowed intracellular zonation in these cells to be appreciated as clearly as in tip-growing cells, a fine cortical mesh, underlying cytoplasmic F-actin bundles (Fu et al., 2002; Mathur et al., 2003b), and microtubules (Saedler et al., 2004a) observed in different diffuse-growing cells suggest a very similar zonation (Fig. 1 C). The apparent similarity in zonal relationship between the plasma membrane and cytoskeletal elements in the region of active protrusive growth between animal and plant cells (Fig. 1) extends to the molecular mechanisms responsible for the creation of a leading edge.
Creating the leading edge

A considerable body of information exists about the multiple protein complexes and regulatory molecules that get activated to create the leading edge in an animal cell (Kraynov et al., 2000; Pollard and Borisy, 2003; Rodriguez et al., 2003). Three key interacting components—members of the Rho/rac/Cdc42 superfamily of GTPases, and actin and microtubule cytoskeletons—stand out. Although Rho-GTPase loops interact with and regulate both actin and microtubule cytoskeletons and microtubules provide directionality to the leading edge, it is actin dynamics that play a major role in membrane protrusion (Small et al., 2002b; Pollard and Borisy, 2003). One of the proposed pathways for actin–cytoskeleton regulation in animal cells involves Cdc42/Rac–GTPase–triggered activation of a suppressor of cAMP receptor from Dictyostelium (SCAR)/Wiskott–Aldrich syndrome protein family verprolin-homologous protein (WAVE) complex. This complex in turn activates a 7-subunit actin-related protein (ARP2/3) complex to enhance actin polymerization (Millard et al., 2004; Vartiainen and Machesky, 2004). The final outcome is a fine dendritic mesh of filamentous actin seen at the leading edge (Fig. 1).

Plants, too, possess a unique subfamily of Rho-family GTPases, called Rho-related GTPase of plants (ROP; Vernoud et al., 2003), whose members localize to areas of active growth (Fu et al., 2001, 2002; Fig. 1, C and D) and play numerous roles, including that of actin–cytoskeleton regulation (Gu et al., 2004). Further, different components of a putative SCAR/WAVE-like complex (Millard et al., 2004; Vartiainen and Machesky, 2004), namely SCAR-related proteins (Frank et al., 2004), NAP125/GNARLED (Brembu et al., 2004; Deeks et al., 2004; El-Din El-Assal et al., 2004; Li et al., 2004; Zimmermann et al., 2004), PIR121/KLUNKER/PIROGI (Basu et al., 2004; Brembu et al., 2004; Li et al., 2004; Saedler et al., 2004b), HSPC300/BRICK1 (Frank and Smith, 2002), and Ab1-1-like proteins (Deeks et al., 2004), have been cloned from Arabidopsis. At least one ROP, AtROP2, has been shown to interact with PIR121, strongly suggesting that a SCAR/WAVE-like pathway mediates the ROP–actin interaction in plants (Basu et al., 2004). Finally, homologues of the different subunit of the ARP2/3 complex have been cloned from plants and in some cases shown to be interchangeable with their animal orthologues (Le et al., 2003; Li et al., 2003; Mathur et al., 2003a,b; El-Assal et al., 2004; Saedler et al., 2004a).

In animal cells, changes in actin-mesh density are believed to affect microtubule plus-end growth by removing a stearic hindrance that allows cytoplasmic microtubules localized access to cortical domains (Rodriguez et al., 2003). In plants, studies on actin–microtubule interactions are just beginning (Mathur, 2004). However, observations of an aberrant aggregation and stabilization of cytoplasmic microtubules in actin-compromised cells suggests an actin control over microtubules very similar to that observed in animal cells (Saedler et al., 2004a). Alternatively, microtubule control over the actin cytoskeleton has also been demonstrated in Drosophila by providing evidence that EB1, a microtubule plus-end–binding protein, mediates the delivery of DRhoGEF2, an activator of Rho1 to cortical domains for its actomyosin-related functions in cell retraction (Rogers et al., 2004). Though not yet demonstrated, similar microtubule–ROP interactions could be envisaged in plants too, as several microtubule plus-end–binding proteins (Bisgrove et al., 2004) as well as a putative ROP-GEF, SPIKE1, have been cloned from Arabidopsis. Interestingly, cells of the spike1 mutant display an aberrant microtubule cytoskeleton (Qiu et al., 2002).

Numerous other modulators (Table I) involved in fine-tuning of the core elements and their interactions appear to be conserved between animals and plants (Goode et al., 2000; Rodriguez et al., 2003; Wasteneys and Yang, 2004) (Table I).
Defects at the leading edge: mutant phenotypes

Compromised function of the three key molecular components, as mentioned in the previous section, has major implications for both motility and cell shape. A brief comparison of cellular phenotypes resulting from defects in the core elements underscores the commonalities. Actin-dependent lamellipodium protrusion and cell motility get greatly attenuated in animal cells with an aberrant actin cytoskeleton (Etienne-Manneville, 2004). Likewise, cell expansion is significantly reduced in actin-compromised plant cells (Baluska et al., 2001). This is especially apparent in Arabidopsis mutants such as nap125/gnarled, pir121/klunker/pirogi (Brembu et al., 2004; Deeks et al., 2004; El-Assal et al., 2004; Li et al., 2004; Saedler et al., 2004b; Zimmermann et al., 2004), and hspc300/brick1 (mutant in maize; Frank and Smith, 2002), whose respective gene products possibly feed into the ARP2/3 complex regulatory pathway. Mutations in different subunit homologues of the putative plant ARP2/3 complex such as arp2/wurm, arp3/distorted1, arp2/distorted2, and arp3/crooked also exhibit similar phenotypes (for review see Mathur, 2005). Each of these mutants exhibits aberrant F-actin organization and characteristic cellular deformations resulting from abnormal, random expansion. However, as mentioned earlier in this paper, actin cytoskeleton activity is heavily dependent upon the stimulation provided by Rho-GTPases. Animal cells with aberrant Rho-GTPase activity display numerous defects in pseudopod extension and chemotaxis (Chung et al., 2000; Raftopoulou and Hall, 2004). Overexpression of ROPs in plants results in increased fine F-actin meshworks and a swollen cell morphology, suggesting an overall reduction in growth polarization (Molendijk et al., 2001; Fu et al., 2002; Jones et al., 2002). Because loss of polarity is a characteristic feature of microtubule defective cells too, in both animal (Small et al., 2002b) and plant cells (Mathur, 2004) these observations again point to a conserved, intimate relationship between Rho-family GTPases, and the actin and microtubule cytoskeletons.

Although the similarities described above emphasize conserved mechanisms for membrane protrusion between animal and plant cells, they also reflect the profound impact the mechanism of boundary extension has on the survival ability of an organism. For motile cells, loss of motility leads to an inability to move to resource-rich areas and ultimately leads to death. Consequently, many animal mutants for the core elements display lethal phenotypes. For the nonmotile but photosynthetic (and therefore relatively self-sufficient) plant cells, a change in cell shape does not lead to death directly. As a result, many comparable plant mutants, though misshapen and underweight (El-Assal et al., 2004; Li et al., 2004), are able to grow and complete their life cycle, giving the erroneous impression that the core genes are not as vital for plants as for animals. However, when grown in a population of siblings, the inability of mutant plants to extend properly becomes a severe handicap that affects their survival ability.

Conclusions and perspectives

A comparison of key cell biological and molecular features in two apparently disparate processes, namely, forward motility of animal cells and extension growth of plant cells, reveals a core machinery that has been conserved by both motile and nonmotile life forms for achieving boundary extension; an essential requirement for survival. Given this realization, future studies on boundary extension mechanisms of different life forms should prove exciting, not only for uncovering the extent of similarities, but also for discovering the range of cell biological and molecular variations, and the adaptations and innovations introduced by different organisms for surviving in their specialized niches.

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References

Baluska, F., J. Jasik, H.G. Edelmann, T. Salajova, and D. Volkman, 2001. Latrunculin B-induced plant dwarfism: plant cell elongation is F-actin-
dependent. Dev. Biol. 231:113–124.

Basu, D., S.E. El-Assal, J. Le, E.L. Mallory, and D.B. Szymanski. 2004. Interchangeable functions of Arabidopsis PIROGI and the human WAVE complex subunit SRA1 during leaf epidermal development. Development. 131:4345–4355.

Bisgrove, S.R., W.E. Hable, and D.L. Kropf. 2004. +TIPs and microtubule regulation. The beginning of the end plus in plants. Plant Physiol. 136:3855–3863.

Brembu, T., P. Winge, M. Seem, and A.M. Bones. 2004. NAPP and PIRP encode subunits of a putative wave regulatory protein complex involved in plant cell morphogenesis. Plant Cell. 16:2335–2349.

Carol, R., and L. Dolan. 2002. Building a hair: tip growth in Arabidopsis thaliana root hairs. Philos. Trans. R. Soc. Lond. B Biol. Sci. 357:815–821.

Chung, C.Y., S. Lee, C. Briscoe, C. Ellsworth, and R.A. Firtel. 2000. Role of Rac in controlling the actin cytoskeleton and chemotaxis in motile cells. Proc. Natl. Acad. Sci. USA. 97:5225–5230.

Deeks, M.J., P.J. Hussey, and B. Davies. 2002. Formins: intermediates in signal cascades that affect cytokinetic reorganization. Trends Plant Sci. 7:492–498.

Deeks, M.J., D. Kaloriti, B. Davies, R. Malho, and P.J. Hussey. 2004. Arabidopsis NAP1 is essential for Arp2/3-dependent trichome morphogenesis.Curr. Biol. 14:1410–1414.

El-Din El-Assal, S., J. Le, D. Basu, E.L. Mallory, and D.B. Szymanski. 2004. DISTORTED2 encodes an ARPC2 subunit of the putative Arabidopsis ARPC/ARP2/3 complex. Plant J. 38:526–538.

El-Assal, S.E., J. Le, D. Basu, E.L. Mallory, and D.B. Szymanski. 2004. Arabidopsis GNARLED encodes a NAP125 homolog that positively regulates ARP2/3. Curr. Biol. 14:1405–1409.

Ettiene-Manneville, S. 2004. Actin and microtubules in cell motility: which one is in control? Traffic. 5:470–477.

Frank, M.J., and L.G. Smith. 2002. A small novel protein highly conserved in plants and animals promotes the polarized growth and division of maize epidermal leaf cells. Curr. Biol. 12:849–853.

Frank, M., C. Egile, J. Dyachok, S. Djakovic, M. Nolasco, R. Li, and L.G. Smith. 2004. Activation of Arp2/3 complex-dependent polymerization by plant proteins distantly related to Scar/WAVE. Proc. Natl. Acad. Sci. USA. 101:16379–16384.

Fu, Y., G. Wu, and Z. Yang. 2001. RopGTPase-dependent dynamics of Tip-localized F-actin controls tip growth in pollen tubes. J. Cell Biol. 152:1019–1032.

Fu, Y., H. Li, and Z. Yang. 2002. The ROP2 GTPase controls the formation of cortical fine F-actin and the early phase of directional cell expansion during Arabidopsis organogenesis. Plant Cell. 14:777–794.

Goode, B.L., D.G. Drubin, and G. Barnes. 2000. Functional cooperation between the microtubule and actin cytoskeletons. Curr. Opin. Cell Biol. 12:63–71.

Gu, Y., Z. Wang, and Z. Yang. 2004. ROP/RAC GTPase: an old master regulator in actin-related proteins 2 and 3 affect cell shape development in Arabidopsis. Plant Cell. 15:1632–1645.

Millard, T.H., S.J. Sharp, and L.M. Machesky. 2004. Signalling to actin assembly via the WASP (Wiskott-Aldrich syndrome protein)-family proteins and the ARP2/3 complex. Biochem. J. 380:1–17.

Molendijk, A.J., F. Bischoff, C.S. Rajendrakumar, J. Friml, M. Braun, S. Gilroy, and K. Palme. 2001. Arabidopsis thaliana Rop GTPase are localized to tips of root hairs and control polar growth. EMBO J. 20:2779–2788.

Pollard, T.D., and G.G. Borisy. 2003. Cellular motility driven by assembly and disassembly of actin filaments. Cell. 112:453–465.

Qiu, J.L., R. Jilk, M.D. Marks, and D.B. Szymanski. 2002. The Arabidopsis SPIKE1 gene is required for normal cell shape control and tissue development. Plant Cell. 14:101–118.

Raftopoulos, M., and A. Hall. 2004. Cell migration: Rho GTPases lead the way. Dev. Biol. 265:23–32.

Rodriguez, O.C., A.W. Schaefer, C.A. Mandato, P. Forscher, W.M. Bement, and C.M. Waterman-Storer. 2003. Conserved microtubule-actin interactions in cell movement and morphogenesis. Nat. Cell Biol. 5:599–609.

Rogers, S.L., U. Wiedemann, U. Hacker, C. Turck, and R.D. Vale. 2004. Drosophila RhogeF2 associates with microtubule plus ends in an EB1-dependent manner. Curr. Biol. 14:1827–1833.

Small, J.V., T. Stradal, E. Vignal, and K. Rottner. 2002a. The lamellipodium: where motility begins. Trends Cell Biol. 12:112–120.

Small, J.V., B. Geiger, I. Kaverina, and A. Bershadsky. 2002b. How do microtubules guide migrating cells? Nat. Rev. Mol. Cell Biol. 3:957–964.

Saedler, R., N. Mathur, B.P. Srinivas, B. Kernebeck, M. Hülskamp, and J. Mathur. 2004a. Actin control over microtubules suggested by DISTORTED2 encoding the Arabidopsis ARPC2 subunit homolog. Plant Cell Physiol. 45:813–822.

Saedler, R., I. Zimmermann, M. Mutondo, and M. Hülskamp. 2004b. The Arabidopsis KLUNKER gene controls cell shape changes and encodes the AGRA1 homolog. Plant Mol. Biol. 56:775–782.

Sieberer, B.J., A.C. Timmers, F.G. Lhuissier, and A.M. Emons. 2002. Endoplasmic microtubules configure the subapical cytoplasm and are required for fast growth of Medicago truncatula root hairs. Plant Physiol. 130:977–988.

Vartiainen, M.K., and L.M. Machesky. 2004. The WASP-Arp2/3 pathway: genetic insights. Curr. Opin. Plant Biol. 16:174–181.

Vernoud, V., A.C. Horton, Z.B. Yang, and E. Nielsen. 2003. Analysis of the small GTPase GNARLED gene superfamily of Arabidopsis thaliana. Plant Physiol. 131:1191–1208.

Vidali, L., and P.K. Hepler. 2001. Actin and pollen tube growth. Protoplasma. 215:64–76.

Vidali, L., S.T. McKenna, and P.K. Hepler. 2001. Actin polymerization is essential for pollen tube growth. Mol. Biol. Cell. 12:2534–2545.

Wasteneys, G.O., and Z. Yang. 2004. New views on the plant cytoskeleton. Plant Physiol. 136:3884–3891.

Zimmermann, I., R. Saedler, M. Mutondo, and M. Hülskamp. 2004. The Arabidopsis GNARLED gene encodes the NAP125 homolog and controls several actin based cell shape changes. Mol. Genet. Genomics. 272:290–296.