REVIEW / SYNTHÈSE

Trichome cell morphogenesis in *Arabidopsis*: a continuum of cellular decisions[1]

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Abstract: In keeping with the myriad functions carried out by plants, their component cells display an amazing diversity of shapes and sizes. How is a precise cell form achieved? In recent years, the single-celled, branched, aerial epidermal trichome of *Arabidopsis thaliana* L. (Heynh) has emerged as a model cell for understanding the cell biological and molecular basis underlying the development of cell shape in plants. Here, I critique the recent information gleaned from dissecting trichome cell morphogenesis in *Arabidopsis* and identify areas and questions that can be further addressed using this unique cell type.

Key words: morphogenesis, trichome, *Arabidopsis*, cytoskeleton, microtubules, ARP2/3 complex.

Résumé : En entretenant les myriades de fonctions réalisées par les plantes, leurs constituantes cellulaires montrent une étonnante diversité de formes et de dimensions. Comment se réalise une forme de cellule précise? Au cours des récentes années, les cellules individuelles et ramifiées du trichome épidermique aérien de l'*Arabidopsis thaliana* L. (Heynh), se sont imposées comme modèle de cellule pour comprendre la base cytologique, biologique et moléculaire, du développement de la forme chez les cellules des plantes. L'auteur analyse l'information récente obtenue en disséquant la morphogenèse cellulaire, chez l'*Arabidopsis* et identifie des domaines et des questions susceptibles d'être étudiés en utilisant ce type de cellule unique.

Mots clés : morphogenèse, trichome, *Arabidopsis*, cytosquelette, microtubules, complexe ARP2/3.

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Introduction

A casual observation of plants reveals an amazing diversity of cell forms. Considering that material is not added on from outside, one wonders how a plant cell shapes itself to execute its functions most efficiently. Clearly, the key to understanding the intricacies of plant form and function lies in studying the complex behavior and interactions of subcellular structures and compartments. Observations on several model cell types, such as root hairs, stigmatic papillae, epidermal pavement, and trichome cells, have provided cell biological and molecular-genetic information related to plant cell morphogenesis (Mathur 2004). Whereas each model cell type provides interesting morphogenetic traits, *Arabidopsis thaliana* trichome cells have recently provided some interesting insights into the morphogenesis of diffuse growing cells. Kotzer and Wasteneys (2006) present a review on the development of puzzle-shaped pavement cells, another diffuse-growing cell type, in this issue.

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Leaf trichomes in *Arabidopsis* are unicellular, endoreduplicating (up to 32 °C), epidermal outgrowths. Trichome cell specification and patterning have been exhaustively reviewed by Szymanski et al. (2000), Hülskamp (2004), and Schellmann and Hülskamp (2005) and will not be discussed here. A fully expanded leaf surface displays a nonrandom distribution of trichomes at different stages of development (Fig. 1). Although a mature *Arabidopsis* trichome is between 300 and 500 μm tall, at its earliest stage of morphogenesis, it can be distinguished, upon comparison with its neighbors, as a slightly larger bulging cell with a diameter of 10–15 μm (Fig. 1). Within 6–10 h after its initiation, the cell grows out into a tubular shape and soon delineates subapical domains that serve as branching initiation points. Two or three branches elongate rapidly through diffuse growth in precise genetically determined orientations that display a tight correlation with the main axis of leaf expansion (Folkers et al. 1997). Growth processes of individual branches are directed to the flanks rather than to the branch apex, resulting in each branch ending in a pointed tip. Although the majority of trichomes on *Arabidopsis* leaves are stellate and 2–3 branched, a subset of the trichome population may remain unbranched. As the trichome cell approaches the end of its growth phase, secondary thickenings raise the cell wall into papillae (Fig. 1). A group of socket cells is arranged around the base of the mature trichome.

Although clearly a continuous process, trichome cell dif-

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ferentiation, nevertheless, appears to proceed through a succession of different shapes. Precise genetic interactions govern the progression from one stage to the next, and numerous mutants possessing trichomes arrested at different stages of development have been isolated in *Arabidopsis* (Table 1) (Hülskamp et al. 1994; Hülskamp 2004). A molecular-genetic as well as cell biological dissection of trichome cell morphogenesis has thus been possible. For convenience of studying, trichome morphogenesis has been divided into five stages (Szymanski et al. 2000). The following sections deal with some of the questions and recent findings pertinent to each stage of trichome cell morphogenesis.

Stage 1. Cell enlargement

In diverse organisms, including higher plants, cell size and nuclear DNA content show strong correlations (reviewed by Umen 2005). The large size of the trichome initial (Fig. 1) apparently results from the first round of endoreduplication that has taken place in the initial (Hülskamp et al. 1994). Whether increased nuclear DNA content and cell turgor are the sole reasons for enlargement and bulging of the trichome cell at this stage or there is an involvement of other local events such as pH alterations, fluctuations in Ca^{2+} gradients, and early expression of expansin genes, as documented for bulge domain creation in root trichoblasts and pollen tube tips (Hepler 2005), remains unclear. However, this key step differentiates the trichome initial from its neighbors and suggests the activation of strong intracellular polarization factors. Continued maintenance of intracellular polarization is required for further commitment to trichome cell fate and allows the initial to grow into the next stage of morphogenesis. Alternatively, an inability to maintain such intracellular commitment would hinder further progression in trichome differentiation. The latter situation is observed in the *glabra2* mutant where a trichome cell becomes specified, undergoes changes in its ploidy level, and embarks on cell enlargement but does not proceed further (Rerie et al. 1994). Instead, as the leaf expands, the *gl2* trichome initial appears to fall flat and subsequently is visible as a large, slightly bulged cell with normal looking pavement cells bordering it. The GLABRA2 protein shows sequence similarity to homeodomain transcription factors and is expressed early in trichome development. Rerie et al. (1994) speculated that *GL2* regulates the expression of later-acting morphogenesis genes. More recently, Ohashi et al. (2003) identified a phospholipase-D (AtPLDζ1) as a downstream target of *GL2*, and a p90 PLD from tobacco has been found to bind to microtubules and the plasma membrane (Gardiner et al. 2001). Further, manipulation of phospholipase-D activity leads to a rapid reorganization of the microtubule cytoskeleton (Dhonukshe et al. 2003). Taken together, these observations suggest the possibility that phospholipase-D-mediated plasma membrane modifications leading to alterations in membrane–cytoskeletal interactions might be required for further progression in, and a continued commitment to, trichome cell morphogenesis.

Stage 2. Tubular outgrowth

The bulged trichome initial extends into a nearly perpendicular, tubular form. As with the earlier stage, the intracellular events contributing to this development are not well understood except that the vacuolar compartment appears to become more prominent and the nuclear DNA content changes through another round of endoreduplication (Hülskamp et al. 1994). Based on the superficial resemblance to tip-growing root hair and pollen tube cells, it has been speculated that this stage may involve similar actin-mediated tip growth mechanisms (Szymanski et al. 2000). However, no ultrastructural evidence supports this suggestion and neither an accumulation of vesicles or a cytoskeletal organization characteristic of tip growing cells has been observed in trichomes at this stage of morphogenesis (Schwab et al. 2003). Further, although the length of tubular, unbranched trichomes may extend up to 600 μm, their tips, in sharp contrast with the rounded apex of tip growing cells, are strongly tapered. These observations indicate that the tubular stage of trichome morphogenesis does not involve a tip growth mechanism but a more general growth targeted to the sidewalls of the expanding cell. Mutants in the *SCD1* gene could be interesting in this respect, since their trichomes do not

Fig. 1. Trichome cell morphogenesis in *Arabidopsis*. The different shapes through which a trichome cell passes (from the bottom of the figure to the top) can be observed on a single expanding leaf. The dark arrows between stages emphasize continuity of the process and indicate key turning points in morphogenesis as the cell changes its direction of growth. The mature trichome has characteristic, papillate surface decorations. Scale bar = 20 μm.

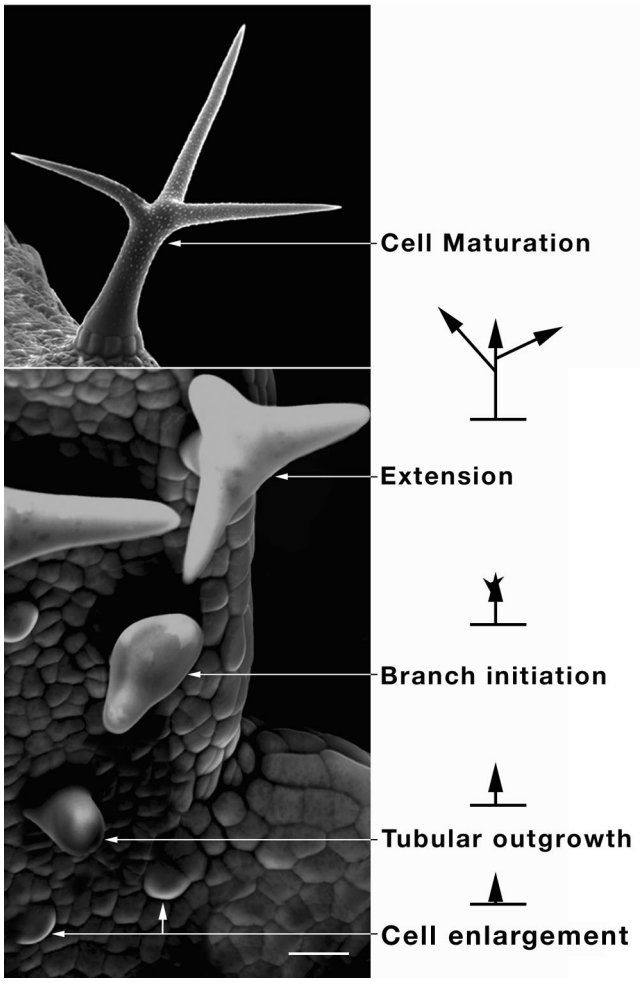


Table 1. Summary of molecularly characterized *Arabidopsis* genes leading to an understanding of trichome morphogenesis.

Gene/AGI No.[a]	Product	Trichome defect in mutant	Reference(s)
ANGUSTIFOLIA At1g01510	CtBP/BARS-like protein	2-branched, MT link proposed	Folkers et al. 2002; Kim et al. 2002
CROOKED[b] At4g01710	ARPC5, smallest p15 subunit of ARP2/3 complex	Distorted, MF aberrant	Mathur 2005; Szymanski 2005)
DISTORTED1[b] At1g13180	Actin-related protein 3, subunit of ARP2/3 complex	Distorted, MF aberrant	Mathur 2005; Szymanski 2005
DISTORTED2[b] At1g30825	ARPC2, p35 subunit of ARP2/3 complex	Distorted, MF aberrant	Mathur 2005; Szymanski 2005
DISTORTED3/IRREGULAR TRICHOME BRANCHING1 At2g38440	Plant-specific SCAR2 homolog	Distorted, branch positioning and expansion irregularities	Basu et al. 2004; Zhang et al. 2005
FASS/TONNEAU2 At5g18580	Domain homology to type2A protein phosphatases	Reduced branching, slightly swollen	Camilleri et al. 2002
GLABRA2 At1g79840	Homeodomain-containing protein	Loss of commitment to trichome cell fate	Rerie et al. 1994
GNARLED[b] *At2g35110*	NAP125 homolog, ARP2/3 complex regulator	Distorted, MF aberrant	Mathur 2005; Szymanski 2005
KATANIN-p60[b] LUE1/FRA2/ BOT1*/ERH3* At2g34560	Subunit of microtubule-severing protein katanin	Less branched, swollen	Bouquin et al. 2003 and other groups
KIESEL At2g30410	Tubulin-folding cofactor A	Swollen, MT associated	Kirik et al. 2002*a*
KINESIN-13A At3g16630	Member of MCAK/kinesin subfamily	4–5 branches, MT–MF-dependent Golgi association	Lu et al. 2005
KCBP-interacting Ca^{2+}-binding protein (KIC) At2g46600	Novel, single EF-hand motif, Ca^{2+} binding	Overexpression reduces trichome branching	Reddy et al. 2004
KLUNKER/*PIROGI*[b] At5g18410	PIR121 homolog, ARP2/3 complex regulator	Distorted, MF aberrant	Mathur 2005; Szymanski 2005
LEFTY1 At1g04820	Alpha tubulin 6	Reduced trichome branching	Thitamadee et al. 2002; Abe et al. 2004
LEFTY2 At1g04820	Alpha tubulin 4	Reduced trichome branching	Thitamadee et al. 2002; Abe et al. 2004
MICROTUBULE *ORGANIZATION/GEMINI POLLEN1* At2g35630	HEAT repeat-containing XMAP215 homolog	Swollen upon exposure to temperature above 29 °C	Whittington et al. 2001
PORCINO At3g10220	Tubulin-folding cofactor C	Swollen, MT associated	Kirik et al. 2002*b*
SPIKE1 At4g16340	Adaptor protein CDM family, putative GEF	Highly elongated, unbranched, abnormally swollen or short-stalked 2-branched	Qiu et al. 2002
STICHEL At2g02480	Domain homology to ATP-binding eubacterial DNA-polymerase-III gamma subunit	Needle like, straight, unbranched, dosage-dependent manner	Ilgenfritz et al. 2003
STOMATAL CYTOKINESIS-DEFECTIVE1 At1g49040	Novel, regulatory protein in intracellular protein transport and or signaling pathways	Mechanically unstable, unbranched blobs	Falbel et al. 2003
WURM[b] *At3g27000*	Actin-related protein 2, large subunit of ARP2/3 complex	Distorted, MF aberrant	Mathur 2005; Szymanski 2005
ZWICHEL At5g65930	Kinesin-like calmodulin-binding protein (KCBP)	Short stalk, swollen, less branched	Oppenheimer et al. 1997

Note: ARP2/3 complex, A 7-subunit actin-related protein 2/3 complex (ARPC1 to ARPC5 are the five small subunits of the complex); CDM, Ced-5, Dock180 and Myoblast city proteins that activate Rac; CtBP/BARS, C-terminal binding protein/ brefeldinA-ADP ribosylated substrate; EF-hand, a highly conserved helix–loop–helix calcium-binding motif; GEF, guanine–nucleotide-exchange factor; MCAK, mammalian mitotic centromere-associated kinesin; NAP125, Nck-associated protein 125; PIR121, p53–121F induced; SCAR, suppresor of cAMP receptor from *Dictyostelium*; XMAP215, *Xenopus* microtubule associated protein 215; WAVE, Wiskott–Aldrich syndrome protein family verprolin-homologous protein.

[a]Systematic designation given by the *Arabidopsis* Genome Initiative.

[b]The gene has been independently cloned and reported by several laboratories. Recent reviews by Mathur (2005) and Szymanski (2005) list the multiple references associated with the gene.

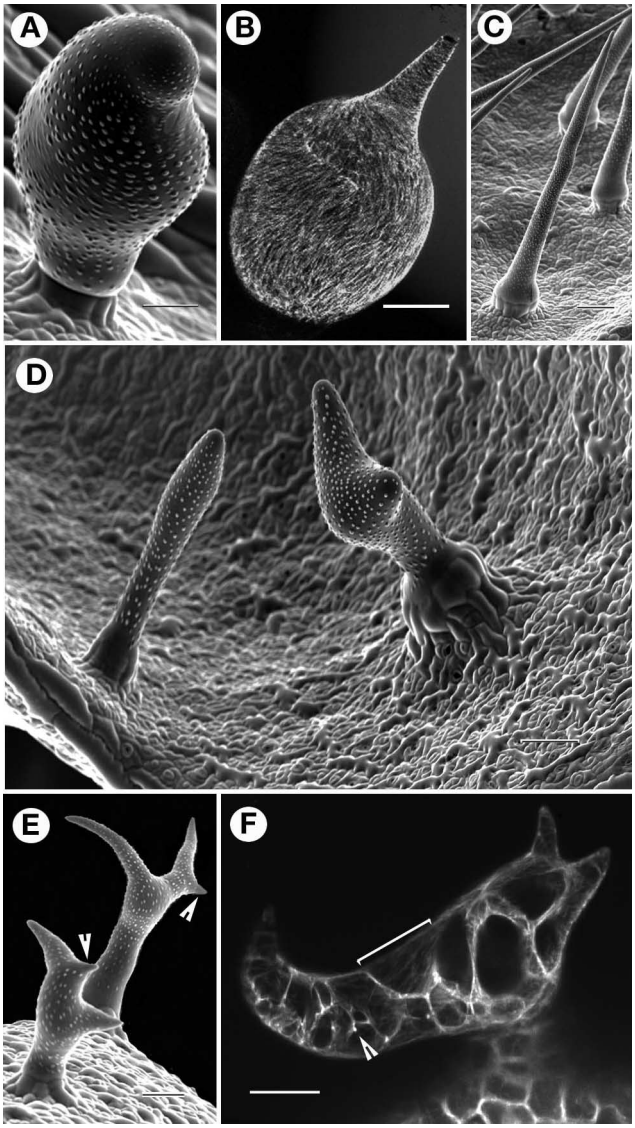


Fig. 2. The actin and microtubule cytoskeletons play an important role in trichome cell shape development in *Arabidopsis.* (A) Trichome cells undergo rapid loss of polarization and expand isotropically upon disturbance with the microtubule cytoskeleton. A single bloated trichome after 12 h of incubation in taxol (1 μmol/L), a microtubule-stabilizing drug. Compare with the untreated trichome shape in Fig. 1. (B) A trichome cell treated with the microtubule-depolymerizing drug oryzalin and allowed to recover for 24 h. Although the cell shape has changed considerably (compare with the untreated trichome (Figs. 1 and 3B), cortical microtubule arrays visualized using a green fluorescent frotein fused to the microtubule binding domain of the human microtubule-associated protein 4 (GFP–MAP4) appear to realign themselves obliquely similar to those in untreated trichomes (compare with Fig. 3B). (C) Unbranched trichomes of the *stichel* mutant (Ilgenfritz et al. 2003). (D) A short treatment with taxol (15 μmol/L) followed by a recovery phase alters growth directionality at the tip of the previously unbranched *stichel* trichome. The trichome shown on the left exhibits only a slight swelling, whereas the right one has developed branch-like structures (Mathur and Chua 2000) (E) As shown here, random alterations in trichome shape are characteristic of the *distorted* class. Compare with the three-branched, mature, wild-type trichome in Fig. 1. Some of the branches (arrowheads) remain stumpy and fail to extend, while others expand abnormally. (F) Internally, many of the *distorted* mutant trichomes have an aberrant actin organization comprising fine F-actin meshworks (area indicated by the line) interspersed with randomly placed dense F-actin patches (arrowhead). F-actin was visualized using a GFP fused to the F-actin domain of the mouse *Talin* gene (GFP–*mTalin*). Compare with the F-actin organization in the wild-type trichome shown in Fig. 3A. Scale bars = 20 μm.

grow beyond the tubular stage and collapse as the leaf expands. *SCD1* encodes a novel regulatory protein affecting vesicle transport (Falbel et al. 2003). Also of interest is the fact that in wild-type *Arabidopsis*, a subset of the trichomes on leaves and a majority of trichomes on flowering stems and sepals remain tubular. Although mechanisms behind the development of a single tubular trichome versus a branched one (see following section) remain obscure, strong alleles of the *stichel* mutant display needle-like, unbranched, tapering trichomes only, while weak *sti* alleles display branch initials (Ilgenfritz et al. 2003). *STI* might be a major regulator in this process. However, cell biological characterization of trichomes in *stichel* fails to provide hints about why certain trichomes remain unbranched while others move into the next stage of morphogenesis to initiate branching. Molecular characterization of *STICHEL* as a novel protein exhibiting sequence similarity to the ATP-binding eubacterial DNA-polymerase III gamma-subunits (Ilgenfritz et al. 2003) also does not provide a direct clue about its strongest mutant phenotype.

Nevertheless, a number of studies strongly suggest a role for the microtubule cytoskeleton during this stage of trichome morphogenesis. Drug- or genetic-lesion-induced alterations in microtubule dynamics result in fat, rounded rather than tubular trichome cells (Figs. 2A and 2B) (Mathur and Chua 2000). In contrast, destroying the actin cytoskeleton produces short but still tubular cells (Mathur et al. 1999).

Stage 3. Branch initiation

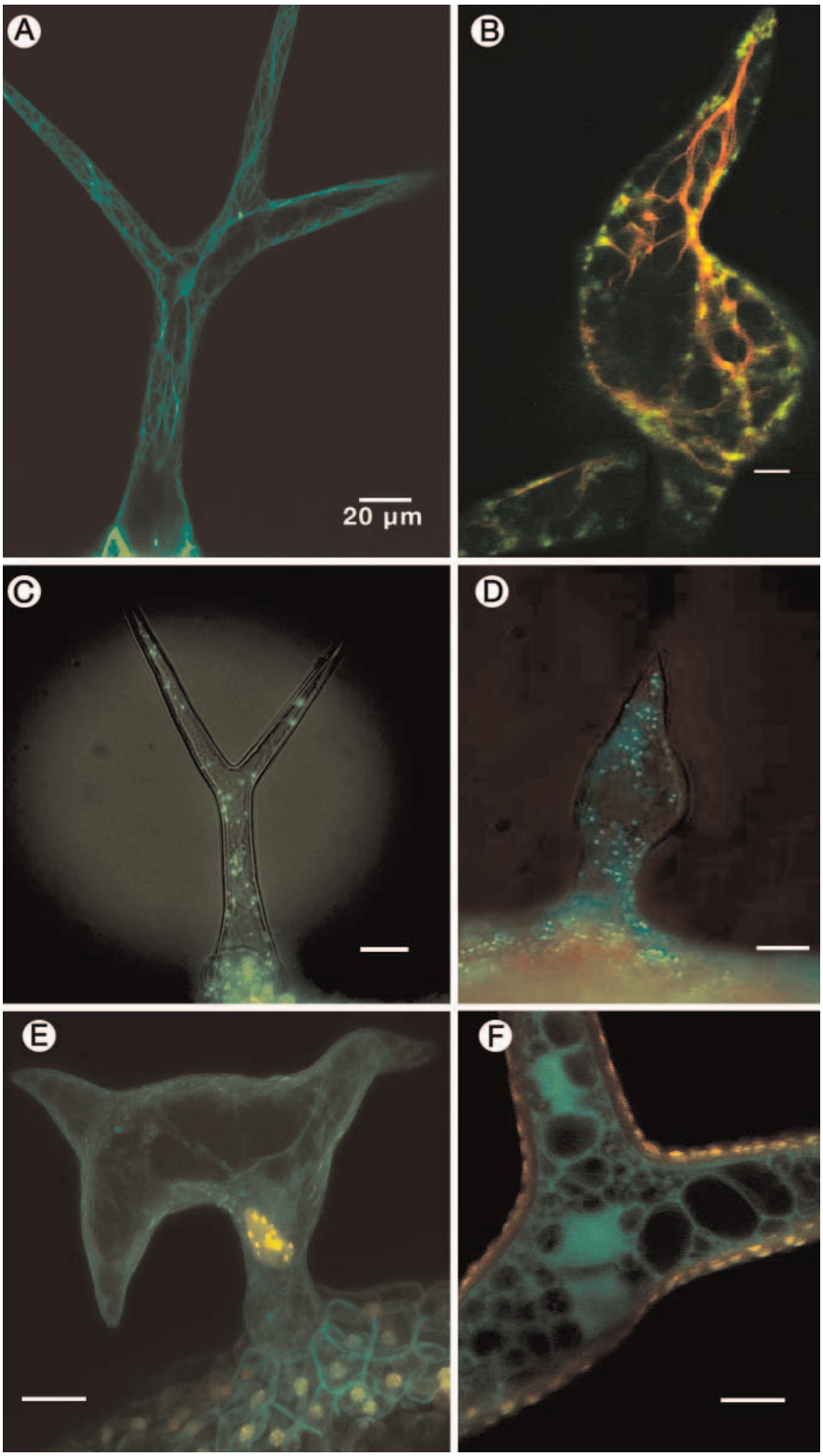
A large number of branching mutants have been identified in *Arabidopsis* and their characterization and genetic interactions reviewed recently (Hülskamp 2004; Schellmann and Hülskamp 2005). From the viewpoint of this review, it is the demarcation of apical domains per se in the tubular initial rather than the number of such domains that provides an interesting aspect of trichome morphogenesis. The cell splits its single axis of growth (stage 2) and recommits itself to creating multidirectional growth. How is this achieved? One cell biological approach to this question exploited the ability to manipulate the growth directionality of trichome cells by microtubule-interacting drugs (Mathur and Chua 2000). Briefly, in this study, the tubular trichome cells were taken to represent an anisotropic growth condition, as they grow in one direction only (Figs. 1 and 2). A drug-induced alteration of microtubule dynamics made these cells lose their strong polarization and expand isotropically (Fig. 2A). The response range between anisotropic and isotropic growth suggested that branch initiation from a tubular form could be viewed as a local and transient switchover towards iso-

Fig. 3. Use of the nearly transparent *Arabidopsis* trichomes to understand organelle dynamics and interactions in living cells. (A) F-actin meshwork visualized in trichomes using a GFP fused to the F-actin-binding domain from the mouse *Talin* gene (GFP–*mTalin*) was used to understand the role of the actin cytoskeleton during trichome cell morphogenesis (Mathur et al. 1999). (B) Yellow fluorescent protein (YFP) – *mTalin* labeled F-actin (false colored red) and ERD2–GFP endoplasmic retention signal defective protein 2 fused to GFP (Boevink et al. 1998) labeled Golgi bodies (in green) visualized in trichomes of the *crooked* mutant suggested that the F-actin mesh could act as a local barrier for organelle motility (Mathur et al. 2003*a*). (C) Peroxisomes visualized using a YFP-carrying a peroxisome targeting signal 1 at its carboxy terminus (YFP–PTS1) appear as punctate motile bodies in an *Arabidopsis* trichome cell (Mathur et al. 2002). (D) Peroxisomes labeled with YFP–PTS1 remained motile in a trichome cell that exhibited a bloated form owing to microtubule depolymerization. This suggested that peroxisome motility did not depend on the microtubule cytoskeleton (Mathur et al. 2002). (E) GFP–*mTalin* and SYTO25 stain (Molecular Probes Inc.) aided in the simultaneous visualization of F-actin and the nucleus in a *distorted* mutant trichome. (F) A trichome cell treated with actin inhibitor latrunculin-B (5 nmol/L)was placed in fluorescein diacetate (FDA) to stain the cytoplasm and highlight the vacuolar compartments as dark areas. Some mutants of the *distorted* class exhibit similar small, unfused vacuoles (Mathur et al. 2003*b*). Size bars = 20 μm.

tropic growth. Following this reasoning, nonbranching trichomes of the *stichel* mutant could be experimentally manipulated to initiate branching points through a transient drug-mediated stabilization of microtubules (Mathur and Chua 2000) (Figs. 2C and 2D). The results suggested that microtubule stabilization could be involved in fixation of polar growth of trichomes, and any changes in growth directionality, such as those required for branch point establishment, required microtubule cytoskeleton reorganization. In principle, local reorganization of microtubules should be able to recreate the same effect. Observations of short, less-branched or rounded trichomes in diverse mutants exhibit branch initiation defects and a tendency towards isotropic growth. These include mutations in *ZWICHEL*, *PORCINO*, *KIESEL*, *LEFTY1*, and *LEFTY2* encoding a calmodulin-dependent kinesin-like protein (Oppenheimer et al. 1997) and tubulin cofactors A and C (Kirik et al. 2002*a*, 2002*b*), alpha tubulin genes (Thitamadee et al. 2002; Abe et al. 2004), respectively, as well as temperature-sensitive alleles of *mor1* (Whittington et al. 2001), an internal kinesin motor (Lu et al. 2005), and katanin-p60 mutants (Bouquin et al. 2003 and other groups, cited in Mathur 2004). Further, the *ANGUSTIFOLIA* gene has also been implicated in functions requiring or having an impact on the microtubule cytoskeleton and the *an* mutant does not exhibit secondary branching (Folkers et al. 2002; Kim et al. 2002). Given the large number of genes affecting the branching process (Hülskamp et al. 1999) (Table 1), the molecular hierarchy that underlies microtubule array organization and its disintegration is presently unclear. Mechanistically, this stage of trichome differentiation clearly involves selective stabilization of microtubules at the plasma membrane (e.g., through the activity of microtubule-associated proteins like MOR1), local stimulation of microtubule-severing proteins (like katanin), and the microtubule-organizing/organelle-interacting properties of motor proteins like ZWICHEL (Oppenheimer et al. 1997), its regulator KIC (Reddy et al. 2004), and kinesin-13A (Lu et al. 2005). In addition, given the precise placement of branch domains, the process is probably subject to the stringent laws of angular interactions (Dixit and Cyr 2004) that govern cortical microtubule dynamics and local organization.

Stage 4. Trichome branch extension

The trichome cell achieves a characteristic regular shape (Fig. 1) as the branches extend outwards. Consequently, even subtle alterations in branch extension are detectable and lead to trichome shape distortion. A drug-based interference with the actin, but not the microtubule cytoskeleton, creates random areas of local expansion in trichome branches and thereby alters trichome shape considerably (Mathur et al. 1999; Szymanski et al. 1999). The actin–drug treatments phenocopy the twisted trichomes of eight different *Arabidopsis* mutants belonging to the "distorted" class (Fig. 2E) (Hülskamp et al. 1994). Much of the recent excitement in the plant morphogenesis field has come from the molecular and cell biological characterization of different members of this *DIS* group (Table 1). As described in detail in recent reviews (Mathur 2005; Szymanski 2005) and briefly below, actin dynamics play a pivotal role in trichome cell expansion. Among the *DIS* genes, *CROOKED*, *DISTORTED1*, *DISTORTED2*, and *WURM* encode protein subunits of a highly conserved seven-subunit actin cytoskeleton modulator, the ARP2/3 complex (Mathur et al. 2003*a*, b; Le et al. 2003; Li et al. 2003; Li et al. 2004; Saedler et al. 2004*a*; El-Assal et al. 2004*a*). The ARP2/3 complex enhances actin polymerization as it creates dendritic F-actin arrays. In other organisms, this complex has been implicated in leading edge dynamics of motile cells, the motility of subcellular structures such as mitochondria and vesicles, and the rapid rocketing motility of certain pathogenic microbes (Vertiainen and Machesky 2004). Upstream regulators of the ARP2/3 complex have been identified in other organisms, and homology-based searches for the respective genes in *Arabidopsis* led to the identification of *GNARLED*, *KLUNKER*, and *DISTORTED3*/*IRREGULAR TRICHOME BRANCHING* genes as different proteins (Table 1) (Li et al. 2004; Brembu et al. 2004; Deeks et al. 2004; El-Assal et al. 2004*b*; Basu et al. 2004; Saedler et al. 2004*b*; Zimmermann et al. 2004; Zhang et al. 2005) belonging to the SCAR/WAVE-containing ARP2/3 regulatory complex. Trichomes in the different *distorted* class mutants of *Arabidopsis* exhibit varying degree of actin organization defects. Mutant trichomes frequently have pockets of aberrantly organized F-actin rather than the characteristic continuous mesh of fine cortical F-actin observed in normally expanding trichomes (Fig. 2F) (Mathur et al. 1999). As this stage of trichome morphogenesis involves rapid extension of the branches, it is suggested that the abnormal F-actin organization interferes somehow with the growth process. Observations of unusual accumulations of F-actin bundles in



nonexpanding portions of mutant cells compared with fine F-actin meshworks in well-expanded regions suggest that the actin cytoskeleton might act as a physical barrier regulating subcellular motility and organelle interactions (Mathur et al. 2003*a*). Indeed, the motility of small organelles such as mitochondria, peroxisomes, and Golgi bodies appears to be compromised in mutant trichomes (Mathur et al. 2003*a*, 2003*b*). By extension, the movement of vesicles carrying material for membrane and cell wall refurbishment during rapid extension may also be inefficient or inadequate.

The actin cytoskeleton appears to affect the local dynamics of microtubules also (Saedler et al. 2004*a*; Zhang et al. 2005) and thereby seems to be responsible for the *distorted* trichome phenotype, which involves substantial alterations

in growth directionality of trichome branches. The cloning of the different *DIS* genes and putative upstream regulators has provided a major impetus to research on the actin cytoskeleton and its regulation (Fig. 2). It continues to stimulate fresh discoveries and discussions on the role of the cytoskeleton during single-cell morphogenesis.

Stage 5. Cell maturation

An indication of trichome cell morphogenesis coming to an end is the formation of papillate secondary wall thickenings. Presently, the molecular mechanisms governing this late occurrence are nearly unknown, although five mutants where the trichome surface decorations are suppressed or nearly nonexistent have been identified. These are grouped in the "glassy trichome" class and includes the *chablis*, *chardonnay*, and *retsina* (Hülskamp et al. 1994), *underdeveloped trichome* (*udt*) (Haughn and Somerville 1988), and *trichome birefringence* (*tbr*) (Potikha and Delmer 1995) mutants. The responsible genes are speculated to encode cell wall components (Hülskamp 2004). However, trichome cell wall maturation also appears to involve the actin cytoskeleton, since mild treatments with actin polymerization inhibitors produce trichomes with random smooth patches (J. Mathur, unpublished observations).

Tapping the potential of the trichome cell

The trichome cell is transparent and conveniently located on the epidermis (Fig. 1). Thus, besides its use in addressing questions on cell morphogenesis, it has also been used as a living laboratory for dissecting subcellular organelle behavior. The morphological alterations that take place in trichomes as a response to actin- and microtubule-specific inhibitors (Fig. 2) were used as an additional, independent criterion to prove that peroxisome motility in plant cells depends on the actin and not the microtubule cytoskeleton (Figs. 3A–3C) (Mathur et al. 2002). Given the large number of live-cell probes that are available now (Dhanoa et al. 2006) and the fact that a majority of the probes are expressed in trichome cells further increases the feasibility of using this robust cell type for visualizing subcellular processes and understanding organelle interactions in plants (Figs. 3D–3F). The development of methodology to free mature trichomes from other epidermal cells further increases their attractiveness for use in immunolocalization-based studies and for downstream applications such as cell wall analysis, measurement of DNA content, and proteomics (Zhang and Oppenheimer 2004). Trichomes are finding increased use in cell cycle studies on higher plants (Jakoby and Schnittger 2004; Verkest et al. 2005) and for understanding mechanisms of epidermal patterning (Schellmann and Hülskamp 2005). It may be safely concluded that with the growing number of genes being implicated in the regulation of *Arabidopsis* trichome development and rapid advances in live-cell imaging technology, this model cell type has much more to offer to modern plant cell biology.

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