# Through form to function: root hair development and nutrient uptake

Simon Gilroy and David L. Jones

**Root hairs project from the surface of the root to aid nutrient and water uptake and to anchor the plant in the soil. Their formation involves the precise control of cell fate and localized cell growth. We are now beginning to unravel the complexities of the molecular interactions that underlie this developmental regulation. In addition, after years of speculation, nutrient transport by root hairs has been demonstrated clearly at the physiological and molecular level, with evidence for root hairs being intense sites of $H^+$-ATPase activity and involved in the uptake of $Ca^{2+}$, $K^+$, $NH_4^+$, $NO_3^-$, $Mn^{2+}$, $Zn^{2+}$, $Cl^-$ and $H_2PO_4^-$.**

The tube-like growth pattern of root hairs is essential to their function in root anchorage and for increasing the area of soil exploitable by the plant[1]. Root hairs form from root epidermal cells. Their development occurs in four phases: cell fate specification, initiation, subsequent tip growth and maturation (Fig. 1). Because of these distinct stages of development, root hairs have been used as a model system to begin to understand how plants:

- Specify cell fate (whether a cell is destined to make a root hair or not).
- Encode positional information (where on a cell the root hair will form).
- Localize growth (how the cellular machinery is organized to make hair-like growth).

The root hairs' role as major nutrient-uptake sites has also made them an obvious site to study the molecular basis of nutrient transport activities in the root. We stand at a particularly exciting point in our understanding of root hair form and function. Molecular candidates for regulators of all stages of root hair development are beginning to emerge. In addition, defined root hair nutrient transport activities have begun to be identified. We are seeing the beginnings of a comprehensive molecular characterization of the developmental and transport activities that underlie the adaptations of the root hair for exploitation of the soil.

## Root hair development

### *Cell fate specification*

Although it has been known for decades that only some root epidermal cells are destined to develop hairs (trichoblasts)[2], the past few years have seen an explosion of information about how this occurs. The decision to become a trichoblast or not happens early in development. From the time of their formation in the meristematic zone, trichoblasts can be distinguished from atrichoblasts by differences in their cytoplasmic structure (e.g. reduced vacuolation)[3]. Two basic schemes describe root-hair-fate specification (Fig. 1). In plants, such as *Phleum* and *Hydrocharis*, trichoblasts form from an asymmetrical division of a protodermal cell. A second theme of development is seen in *Arabidopsis*. In this plant, trichoblasts form from epidermal cells overlying the junction of two cortical cells. This patterning leads to files of trichoblasts interspersed with files of atrichoblasts[4] (Fig. 1), and suggests intricate cell-to-cell communication soon after formation in the meristem. The cortical cells might relay positional information to the overlying epidermal cells to lay down these precise patterns of cell fate. Recent evidence suggests genes, such as *TRANSPARENT TESTA GLABRA* (*TTG*) and *GLABRA2* (*GL2*), are also involved in fate specification in the shoot epidermis, and are negative transcriptional regulators of root hair formation[5–7]. Conversely the myb-like transcription factor encoded by the *CAPRICE* gene is thought to be a negative regulator of non-hair fate[8].

Mutants that suppress root hair formation, or those that lead to the production of ectopic root hairs, also indicate possible hormone (ethylene and auxin)-related mechanisms of cell fate specification. For example, in the *ctr-1* mutant of *Arabidopsis*, all root epidermal cells produce root hairs. *CTR-1* encodes a protein kinase of the Raf super-family that is involved in ethylene signal transduction[9]. Similarly, activators and inhibitors of ethylene synthesis alter trichoblast formation[10], and the *rhd6* root hair developmental mutant, which fails to initiate root hairs correctly, can be rescued by auxin or ethylene, and phenocopied by ethylene synthesis inhibitors[11]. Exactly how these hormones act is not known. It is likely that hormones act either independently or later than *TTG* and *GL2* (Ref. 12), perhaps fixing the cell fate once it is specified. Recent work also suggests that trichoblasts and atrichoblasts might have differential hormone sensitivities[13]. We anticipate that the identification of signaling elements, such as the *CTR-1* kinase, coupled to the continuing characterization of the remarkable array of root-hair-development mutants, identified by the efforts of many research groups, is just the beginning of the process of defining a molecular regulatory pathway for trichoblast fate specification and root hair formation.

### *Root hair initiation*

In the trichoblast, the first morphological indications of root hair formation become evident when the cell begins to form a highly localized expansion from one side to form a bulge in the cell wall (Fig. 1). This is the process of root hair initiation. The site on the lateral wall of the trichoblast is precisely regulated. In *Arabidopsis*, for example, root hairs always form at the end of the cell nearest the root apex[14]. This site can be shifted by ethylene or auxin treatment[11], suggesting again that these hormones are critical regulators of root hair formation. The ability to shift the initiation site also implies that it is continuously and actively specified before root hair emergence rather than being the result of a marker laid down at the beginning of trichoblast development.

Microtubule rearrangements are associated with the formation of the bulge that will become the root hair[15]. The subsequent wall bulging at the initiation site appears to be intimately linked to the acidification of the cell wall[16]. Thus, blocking the localized drop in pH at the initiation site reversibly arrests initiation. How this acidification is maintained is not known, but localized $H^+$-ATPase activity is an obvious candidate, especially as cytoplasmic $H^+$


1360 - 1385/00/$ – see front matter  PII: S1360-1385(99)01551-4

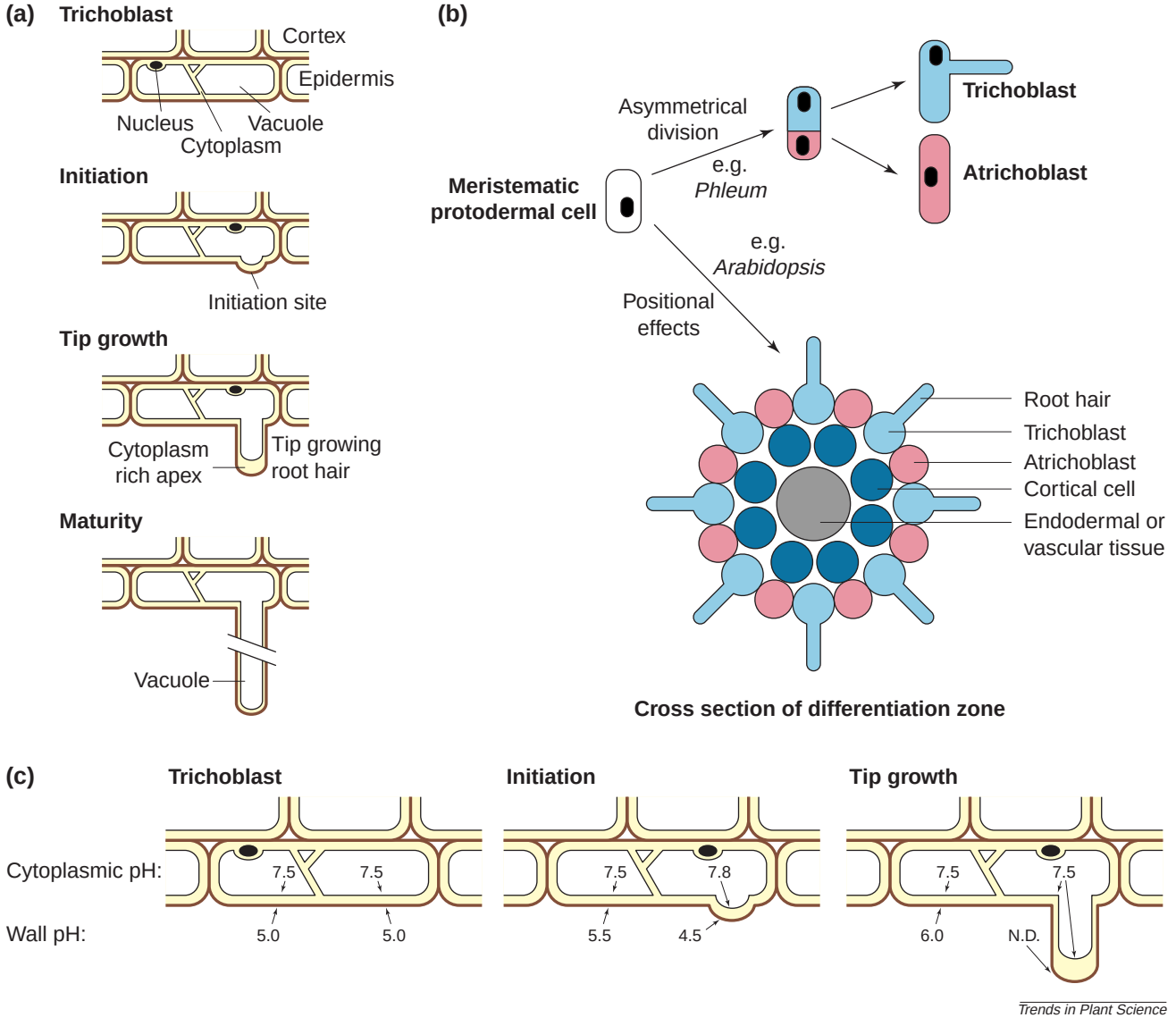


**Fig. 1.** Patterns of root hair development. (a) Cross section of a trichoblast (epidermal cell that will produce a root hair) during root hair development. Note the nuclear movements accompanying root hair emergence and changes in the organization of the cytoplasm at each new developmental phase. (b) Divergence of root epidermal cell fate to trichoblast or atrichoblast might be determined by asymmetrical division and differences in subsequent differentiation of the daughter cells or by positional effects relative to underlying cell layers. (c) pH profiles of the cytoplasm and the wall in a trichoblast before, during and after the initiation phase of root hair emergence. Wall and cytoplasmic pH were monitored by confocal ratio imaging[16]. N.D., not determined.

concentration falls at the point of initiation (Fig. 1). However, artificially acidifying the entire trichoblast wall does not alter the site of root hair formation, indicating that it must be defined by other factors in addition to wall pH changes[16]. Defining precisely what these additional factors might be is a challenge for future research.

*Tip growth*

Following initiation, the root hair commences tip growth (Fig. 1), a process that is genetically[11,14] and physiologically[17] distinct from root hair initiation. In the tip of the growing root hair, the deposition of new plasma membrane and cell wall material is confined to the expanding tip, leading to an elongated hair-like morphology.

Regulation of the direction in which the secretory apparatus operates appears to be linked intimately to $[Ca^{2+}]_{cyt}$ at the apex. The resting level of $[Ca^{2+}]_{cyt}$ in eukaryotic cells is between 100 and 400 nM. However, the $[Ca^{2+}]_{cyt}$ at the tip of elongating cells as diverse as fungal hyphae, algal rhizoids, pollen tubes and root hairs, is elevated to several micromolar[17–23] (Fig. 2). The steepness of the $[Ca^{2+}]_{cyt}$ gradient correlates well with the growth rate of individual root hairs, being most pronounced in the rapidly elongating root hairs. Root hairs that have stopped elongating, either by reaching their final mature length, or because of some experimental manipulation, show no elevation of $[Ca^{2+}]_{cyt}$ at the tip[17–23]. In addition, when root hairs are forced to produce new tips or to redirect their growth, for example, as a result of Nod-factor treatment[19] or disruption of their microtubule cytoskeleton[22], the new growing tips always posses a tip-focused gradient in $[Ca^{2+}]_{cyt}$. Electrophysiological studies using a self-referencing (vibrating) microelectrode also show that $Ca^{2+}$-influx is higher at the tip than at the base or sides of growing root hairs[23–25] (Fig. 3). Disrupting

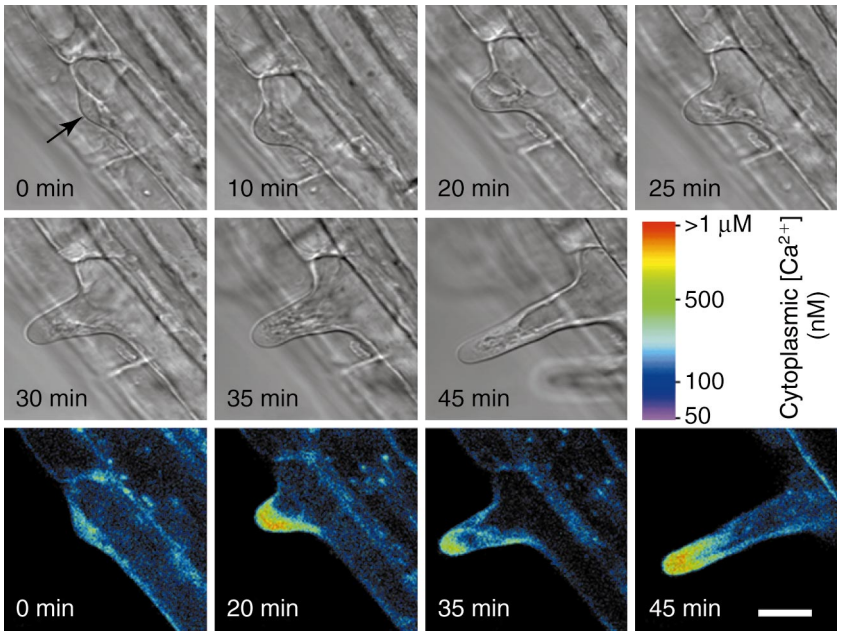


**Fig. 2.** Gradients in cytoplasmic $Ca^{2+}$ associated with tip growth of *Arabidopsis* root hairs. There are no detectable $Ca^{2+}$ gradients associated with the initiation process (0 min), but on the commencement of tip growth, gradients focused on the elongating tip are always seen. Upon cessation of growth the gradient dissipates. Images are confocal ratio images of *Arabidopsis* root hairs loaded with the fluorescent $Ca^{2+}$ sensor Indo-1 and monitored using a confocal microscope[17]. $Ca^{2+}$ levels in the cytoplasm have been color coded according to the inset scale. The arrow indicates the initiation site. Scale bar = 20 μm. Reproduced, with permission, from Ref. 17.

these tip-focused gradients in $[Ca^{2+}]_{cyt}$ by the addition of $Ca^{2+}$-ionophores, $Ca^{2+}$-channel blockers, or by the microinjection of $Ca^{2+}$ buffers, which diffuse any cytoplasmic gradient, all inhibit root hair growth[17–23]. Interestingly, imposing an artificial tip-focused $[Ca^{2+}]_{cyt}$ gradient reorients root hair growth towards the new gradient[18]. These observations are all consistent with a model whereby a localized increase in $[Ca^{2+}]_{cyt}$ is specifically associated with growth, and appears to be capable of organizing and driving growth at the root hair apex.

What might the $Ca^{2+}$ gradient be affecting? The cytoskeleton is an obvious candidate. Treatment with drugs that disrupt actin filaments arrests apical growth in most tip-growing cells, including root hairs[22]. In growing root hairs, actin runs along the length of the root hair but flares into fine bundles sub-apically. These bundles are excluded from the vesicle-rich apex (apical clear zone)[26]. In non-growing root hairs, the actin bundles extend throughout the tip. This ordered actin array might be needed to move secretory vesicles towards the apical clear zone. The tip-focused $Ca^{2+}$ gradient might not only keep the clear zone free of cytoskeletal arrays but might also facilitate the movement and fusion of vesicles once they are in this area. Consistent with this model is the observation that when the $[Ca^{2+}]_{cyt}$ gradient is lost, actin microfilaments protrude to the root hair tip[26]. In addition, elevated $[Ca^{2+}]_{cyt}$ is known to promote secretory vesicle fusion with the plasma membrane[27], inhibit cytoplasmic streaming, fragment F-actin and depolymerize microtubules[28] – all activities that would promote stabilization of the apical clear zone.

The microtubule cytoskeleton also appears to be important for root hair elongation, although not for maintaining tip growth. Thus, in *Arabidopsis*, microtubule-depolymerizing agents do not arrest tip growth but do cause them to adopt a waving growth habit[22] (Fig. 4). These observations suggest that although the microtubule cytoskeleton is not required for tip growth to proceed, it is involved in stabilizing the site of the apical growth machinery. How this stabilization occurs is unknown but the disruption of kinesin-like microtubule motors leads to waving growth patterns in tip-growing fungal hyphae[29] and altered branching patterns in trichomes[30], providing a candidate for a microtubule-associated protein that could help stabilize the tip growth machinery.

In addition to facilitating tip growth, calcium probably has many other roles in root hairs. For example, the initial interactions between rhizobial bacteria and root hairs, leading to the formation of nitrogen-fixing root nodules, is accompanied by signaling-related changes in cytoplasmic[31] and nuclear[32] $Ca^{2+}$, as well as by many other root hair-related changes. In addition, $Ca^{2+}$ and the cytoskeleton are far from being the only candidates for the regulatory elements of the tip-focused growth machinery. Other possibilities include annexins, calmodulin, GTPases, protein kinases and putative integrin-like proteins spanning the cell wall–plasma membrane–cytoskeleton continuum. Most of these elements have been identified in tip-growing pollen tubes, but their role in root hair growth remains to be unraveled. This is exciting because we now have a series of defined molecular targets with which to generate testable models to determine how the tip growth machinery operates and how it is regulated.

### Root hairs and nutrient uptake

The complex developmental processes outlined above lead to the hair-like growth pattern of root hairs. This in turn increases the volume of soil in contact with, and therefore exploitable by, the root. Indeed, it has been speculated for many decades that the primary reason for root hair existence is to increase the efficiency of nutrient ion uptake from the soil. The evidence for this was simply based on the observation that the number and density of root hairs apparently increases under nutrient stress. Because of the lack of suitable molecular and physiological techniques, the mechanisms involved in nutrient capture by root hairs have become clear only recently. Although evidence for the uptake of most major- and micronutrients by root hairs now exists ($NH_4^+$ $NO_3^-$, $K^+$, $Ca^{2+}$, $H_2PO_4^-$, $Cl^-$, $Zn^{2+}$, $Mn^{2+}$), the signalling pathways involved in regulating these transport mechanisms remain elusive. Furthermore, it is known that within plant species there is considerable genetic variability in root hair responses to nutrient stress. Improving plants to make root hairs more effective at nutrient capture should reduce the environmental impact of agriculture (e.g. eutrophication) and increase crop production and sustainability in reduced input systems.

The driving force for most nutrient uptake in plants is the electrochemical gradient across the plasma membrane, a major proportion of which is generated by the $H^+$-ATPase. In support of the theory that root hairs are centers of nutrient uptake, high levels of expression of $H^+$-ATPase genes in root hairs has been demonstrated in *Nicotiana*[33], with the strongest expression occurring in developing root hairs and reduced expression in mature root hairs.

Although the spatial localization of $H^+$-ATPase proteins within the root hairs themselves is unknown, evidence obtained with vibrating pH-sensitive microelectrodes indicates a strong $H^+$ efflux from the base of the root hair and an apparent tip-localized $H^+$ influx. This suggests that the proximity of $H^+$-ATPases to the zone of new growth is closely regulated[25] (Fig. 3).

*Phosphorous uptake*

Phosphorus is extremely immobile in soil and is frequently growth-limiting. Considering the significant genetic variability in P efficiency that exists within species, research efforts are now being focused on understanding the mechanistic basis of P efficiency, to develop crops that require less input. Experiments carried out in soil, where only root hairs were allowed to penetrate $^{32}$P-labeled soil hotspots, have confirmed that root hairs can satisfy >60% of the plant's P demand[34]. In P-deficient soil, the length and density of *Arabidopsis* root hairs increase massively, expanding the root's surface area from 0.21 $mm^2\ mm^{-1}$ root under P-sufficient conditions to 1.44 $mm^2\ mm^{-1}$ root under P-starvation conditions, with the root hairs constituting 91% of the total root's surface area[35]. In a P-deficient medium, the root hairs grow nearly twice as fast as those in a medium with low levels of P, with full root hair expansion taking 8 h. Although *Arabidopsis* root hair growth is stimulated by low levels of P, other nutrient deficiencies (K, B, Cu, Fe, Mg, Mn, S and Zn) fail to produce a similar phenotype. This is particularly surprising considering that the uptake of micronutrients, such as Cu, Zn and Fe, is frequently diffusion-limited like P.

In spite of extensive experiments showing the depletion of P within the root hair zone of the soil, the exact mechanisms involved in P uptake by root hairs were elucidated only recently. Recent cloning of a high-affinity P transport gene from tomato (*LePT1*) has indicated a high degree of expression in root hairs, as well as in other root tissues with upregulation under P deficiency[36]. Based on yeast expression studies, the transporter can be classed as a $H^+$/symporter with $H_2PO_4^-$ appearing to be the primary ionic species transported, with neither $HPO_4^{2-}$, $NO_3^-$ nor $SO_4^{2-}$ capable of transportation. Although the signal transduction cascades, which lead to P-mediated increases in root hair length and density, have yet to be elucidated, evidence suggests that control might be at the cellular level, with parallel signalling pathways involving hormones, such as ethylene and IAA (Ref. 37).

*Nitrogen uptake*

With respect to $NH_4^+$ and $NO_3^-$, there is now clear electrophysiological and molecular evidence that root hairs can transport N-compounds. Cloning of $NH_4^+$ and two putative low-affinity $NO_3^-$ transporters from tomato[38] has revealed that the expression of two of these genes is root-hair-specific (*LeNRT1-2* and *LeAMT1*) and regulated by an external N supply. In the case of the $NH_4^+$ transporter *LeAMT1*, it is constitutively expressed and possibly down-regulated by the presence of $NO_3^-$. Indirect evidence using pH-sensitive fluorescent dyes has suggested that $NH_4^+$ uptake occurs immediately after the addition of $NH_4^+$ to N-starved roots, in agreement with the molecular studies[39]. By contrast, the $NO_3^-$ transporters encoded by *LeNRT1-1* and *LeNRT1-2* are both upregulated in the presence of available $NO_3^-$, as might be expected for low-affinity transporters. In addition, results from voltage clamp studies have revealed that the high-affinity $NO_3^-$ transporter in *Arabidopsis* root hairs is greatly upregulated under $NO_3^-$ deficiency, suggesting that root hairs have multiple strategies for providing adequate nutrients to the root[40].

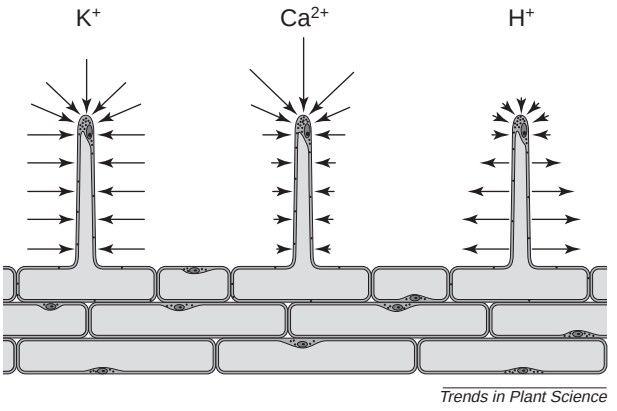


**Fig. 3.** Ion fluxes around root hairs of *Limnoboium stoloniferum*. Ion-selective self-referencing (vibrating) microelectrodes were used to map the ion influx or efflux around elongating root hairs[25]. The length and direction of arrows reflects the relative magnitude and the direction of flux of each ion.

## Prospects for the future

Many open questions remain as to how the trichoblast cell fate is specified, the cues that determine when and where a root hair will form, and precisely what molecules make up the tip growth machinery of the elongating root hair. Answering these questions using the model system of the root hair will undoubtedly reveal insights into pattern formation, cell-fate specification, axis formation and localization of growth that is applicable to other systems. The combination of the vast array of *Arabidopsis* developmental mutants in root hair formation and the imminent completion of the *Arabidopsis* genome-sequencing project bode well for rapid and significant steps toward answering some of these questions. Further, the potential exists for altering nutrient efficiency in crops by the manipulation of root hair transport processes. We are clearly far from understanding the complexities of nutrient transporter activities in the root hair. However, we can anticipate a much

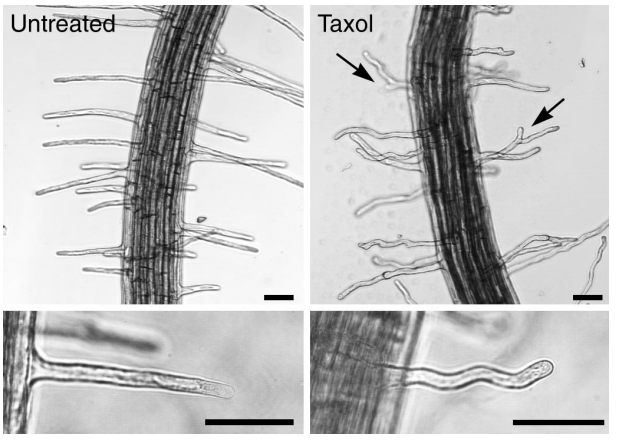


**Fig. 4.** Effect of the microtubule antagonist taxol on the growth habit of *Arabidopsis* root hairs. The root hairs in the right panels have been treated for 4 h with 10 μM taxol. Note the waving growth habit and the formation of multiple growing points in a single root hair (arrows) in the taxol-treated root, versus straight growth with a single tip growth-point in untreated roots. Scale bars = 25 μm.

more complete characterization of the $H^+$-ATPase and the N- and P-transporter systems now identified in root hairs. Such data is clearly critical for building a model of how the root hair contributes to the nutrient status of the plant. An important challenge for the future will be to see how generally applicable the insights emerging from these studies are for understanding the mechanisms whereby root hairs contribute to the growth of a range of agronomically important crop plants.

**Acknowledgements**
We would like to thank Dr Sarah Swanson for critical reading of the manuscript. This work was supported by grants from the National Science Foundation, NASA, NATO and The Royal Society.

**References**

**1** Peterson, R.L. and Farquhar, M.L. (1996) Root hairs: specialized tubular cells extending root surfaces. *Bot. Rev.* 62, 2–33
**2** Cormack, R.G.H. (1949) The development of root hairs in angiosperms. *Bot. Rev.* 15, 583–612
**3** Galway, M.E. *et al.* (1997) Growth and ultrastructure of *Arabidopsis* root hairs: the *rhd3* mutation alters vacuole enlargement and tip growth. *Planta* 201, 209–218
**4** Dolan, L. *et al.* (1993) Cellular organization of the *Arabidopsis thaliana* root hair. *Development* 119, 71–84
**5** Galway, M.E. *et al.* (1994) The *TTG* gene is required to specify epidermal cell fate and cell patterning in the *Arabidopsis* root. *Dev. Biol.* 166, 740–754
**6** Di Chrisitina, M. *et al.* (1996) The *Arabidopsis ATHB10* (*GLABRA2*) is a HD-ZIP protein required for repression of ectopic root hair formation. *Plant J.* 10, 393–402
**7** Masucci, J.D. *et al.* (1996) The homeobox gene *GLABRA2* is required for position-dependent cell differentiation in the root epidermis in *Arabidopsis thaliana*. *Development* 122, 1253–1260
**8** Wada, T. *et al.* (1997) Epidermal cell differentiation in *Arabidopsis* is determined by a Myb homolog, CPC. *Science* 227, 1113–1116
**9** Kieber, J.J. *et al.* (1993) CTR1, a negative regulator of the ethylene response pathway in *Arabidopsis*, encodes a member of the Raf family of protein kinases. *Cell* 72, 427–441
**10** Tanimoto, M. *et al.* (1995) Ethylene is a positive regulator of root hair development in *Arabidopsis thaliana*. *Plant J.* 8, 943–948
**11** Masucci, J.D. and Schiefelbein, J.W. (1994) The *rhd6* mutation of *Arabidopsis thaliana* alters root hair initiation through an auxin and ethylene associated process. *Plant Physiol.* 106, 1335–1346
**12** Massucci, J.D. and Schiefelbein, J.W. (1996) Hormones act downstream of *TTG* and *GL2* to promote root hair outgrowth during epidermis development in the *Arabidopsis* root. *Plant Cell* 5, 1505–1517
**13** Cao, X.F. *et al.* (1999) Differential ethylene sensitivity of epidermal cells is involved in the establishment of cell pattern in the *Arabidopsis* root. *Physiol. Plant.* 106, 311–317
**14** Schiefelbein, J.W. and Somerville, C. (1990) Genetic control of root hair development in *Arabidopsis thaliana*. *Plant Cell* 2, 235–243
**15** Emons, A.M.C. and Derksen, J. (1986) Microfibrils, microtubules, and microfilaments of the trichoblast of *Equisetum hyemale*. *Acta. Bot. Neerl.* 35, 311–320
**16** Bibikova, T.N. *et al.* (1998) Localized changes in apoplastic and cytoplasmic pH are associated with root hair development in *Arabidopsis thaliana*. *Development* 125, 2925–2934
**17** Wymer, C.L. *et al.* (1997) Cytoplasmic free calcium distributions during the development of root hairs of *Arabidopsis thaliana*. *Plant J.* 12, 427–439
**18** Bibikova, T.N. *et al.* (1997) Root hair growth in *Arabidopsis thaliana* is directed by calcium and an endogenous polarity. *Planta* 203, 495–505
**19** DeRuijter, N.C.A. *et al.* (1998) Lipochito-oligosaccharides re-initiate root hair tip growth in *Vicia sativa* with high calcium and spectrin-like antigen at the tip. *Plant J.* 13, 341–350
**20** Felle, H. and Hepler, P.K. (1997) The cytosolic $Ca^{2+}$ concentration gradient of *Sinapis alba* root hairs as revealed by $Ca^{2+}$-selective microelectrode tests and fura-dextran ratio imaging. *Plant Physiol.* 114, 39–45
**21** Jones, D.L. *et al.* (1998) Effect of aluminum on cytoplasmic $Ca^{2+}$ homeostasis in root hairs of *Arabidopsis thaliana* (L.). *Planta* 206, 378–387
**22** Bibikova, T.N. *et al.* (1999) Microtubules regulate tip growth and orientation in root hairs of *Arabidopsis thaliana*. *Plant J.* 17, 657–665
**23** Herrmann, A. and Felle, H.H. (1995) Tip growth in root hair cells of *Sinapis alba* L.: significance of internal and external $Ca^{2+}$ and pH. *New Phytol.* 129, 523–533
**24** Schiefelbein, J.W. *et al.* (1992) Calcium influx at the tip of growing root-hair cells of *Arabidopsis thaliana*. *Planta* 187, 455–459
**25** Jones, D.L. *et al.* (1995) Role of calcium and other ions in directing root-hair tip growth in *Limnobium stoloniferum*. I. Inhibition of tip growth by aluminum. *Planta* 197, 672–680
**26** Miller, D.D. *et al.* (1999) The role of actin in root hair morphogenesis: studies with lipochito-oligosaccharide as a growth stimulator and cytochalasin as an actin perturbing drug. *Plant J.* 17, 141–154
**27** Carroll, A.D. *et al.* (1998) $Ca^{2+}$, annexins, and GTP modulate exocytosis from maize root cap protoplasts. *Plant Cell* 10, 1267–1276
**28** Cyr, R.J. (1994) Microtubules in plant morphogenesis: role of the cortical array. *Annu. Rev. Cell Biol.* 10, 153–180
**29** Wu, Q. *et al.* (1998) A fungal kinesin required for organelle motility, hyphal growth, and morphogenesis. *Mol. Biol. Cell* 9, 89–101
**30** Oppenheimer, D.G. *et al.* (1997) Essential role of a kinesin-like protein in *Arabidopsis* trichome morphogenesis. *Proc. Natl. Acad. Sci. U. S. A.* 94, 6261–6266
**31** Felle, H.H. *et al.*(1998) The role of ion fluxes in Nod factor signaling in *Medicago sativa*. *Plant J.* 13, 455–463
**32** Ehrhardt, D.W. *et al.* (1996) Calcium spiking in plant root hairs responding to *Rhizobium* nodulation signals. *Cell* 85, 673–681
**33** Moriau, L. *et al.* (1999) Expression analysis of two gene subfamilies encoding the plasma membrane $H^+$-ATPase in *Nicotiana plumbaginifolia* reveals the major transport functions of this enzyme. *Plant J.* 19, 31–41
**34** Gahoonia, T.S. and Nielsen, N.E. (1998) Direct evidence on participation of root hairs in phosphorus ($^{32}P$) uptake from soil. *Plant Soil* 198, 147–152
**35** Bates, T.R. and Lynch, J.P. (1996) Stimulation of root hair elongation in *Arabidopsis thaliana* by low phosphorus availability. *Plant Cell Environ.* 19, 529–538
**36** Daram, P. *et al.* (1998) Functional analysis and cell-specific expression of a phosphate transporter from tomato. *Planta* 206, 225–233
**37** Lynch, J. and Brown, K.M. (1997) Ethylene and plant responses to nutritional stress. *Physiol Plant.* 100, 613–619
**38** Lauter, F.R. *et al.* (1996) Preferential expression of an ammonium transporter and of two putative nitrate transporters in root hairs of tomato. *Proc. Natl. Acad. Sci. U. S. A.* 93, 8139–8144
**39** Kosegarten, H. *et al.* (1997) Differential ammonia-elicited changes of cytosolic pH in root hair cells of rice and maize as monitored by 2′,7′-bis-(2-carboxyethyl)-5 (and -6)-carboxyfluorescein-fluorescence ratio. *Plant Physiol.* 113, 451–461
**40** Meharg, A.A. and Blatt, M.R. (1995) $NO_3^-$ transport across the plasma-membrane of *Arabidopsis thaliana* root hairs – kinetic control by pH and membrane voltage. *J. Memb. Biol.* 145, 49–66

Simon Gilroy* is at the Biology Dept, The Pennsylvania State University, 208 Mueller Laboratory, University Park, PA 16802, USA; David Jones is at the School of Agricultural and Forest Sciences, University of Wales, Bangor, Gwynedd, UK LL57 2UW.

*Author for correspondence (tel +1 814 863 9626; fax +1 814 865 9131; e-mail sxg12@psu.edu; http://www.bio.psu.edu/faculty/gilroy).