J Plant Growth Regul (2001) 20:255–264
DOI: 10.1007/s003440010028

J P G R
Journal of Plant Growth Regulation

Dual Auxin Signaling Pathways Control Cell Elongation and Division

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ABSTRACT

Auxin regulates plant cell division, elongation, and differentiation through signal transduction that probably embodies a complex network rather than a linear pathway. Evidence has emerged that auxin-binding protein 1 (ABP1) is a component of this signal network by acting as a high-affinity auxin receptor mediating cell expansion. On the other hand, high auxin concentrations stimulate cell division via a second, unidentified auxin pathway, possibly mediated by a low-affinity auxin receptor. Evidence is presented that this second pathway is coupled with a heterotrimeric G-protein. Molecular genetic evidence supports the conclusion that both the ABP1 and G-protein pathways must crosstalk to bring about normal development.

Key words: Auxin; Auxin-binding protein 1 (ABP1); BY-2; Cell division; Cell elongation; G-protein

INTRODUCTION

Auxin regulates three essential cellular fates in morphogenesis: elongation, division, and differentiation. The endogenous auxin, indole-3-acetic acid (IAA), is shaped into apical/basal and radial gradients throughout plant organs via specific, polarized transport proteins, conferring positional information (Meinhardt 1984; Lomax and others 1995; Uggla and others 1996). In the etiolated shoot of a seedling, cell division, elongation, and differentiation proceed linearly and roughly sequentially from apex to base. Exogenous auxin can cause rapid increases in steady state transcript levels of a small set of genes, alters ion flux at the plasma membrane, and stimulates changes in the cell wall (Taiz 1984; Guilfoyle 1986; Theologis 1986; Blatt and Thiel 1994; Abel and others 1995). Although auxin effects have been extensively studied, the molecular mechanisms of auxin action remain poorly understood. Biochemical, pharmacological, electrophysiological, and molecular genetic evidence support a role for auxin-binding protein 1 (ABP1) being an auxin receptor, but it remains controversial how this protein works and what it interacts with (Jones 1994; Napier 1995; Napier and Venis 1995; Napier 1997). Emerging evidence implicates the presence of diverse auxin signaling pathways, with ABP1 operating in only one. It has been suggested that there are at least two distinct auxin receptors because auxin-induced changes in gene expression and the change at the cell wall-plasma membrane interface can be uncoupled from growth in the short term (Vanderhoef and Dute 1981; Prasad and Jones 1991; Jones and Prasad 1992). In this perspective, it will be proposed how two fundamental cellular processes, cell elongation and division, are separable, and how ABP1 fits in the cell elongation pathway. Evidence that the parallel division pathway involves a het-

Online publication 8 November 2001
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erotrimeric G-protein will be discussed as well as how these two pathways must crosstalk to bring about normal morphogenesis.

Auxin Induces Separable Cellular Responses

It is well known that auxin controls both cell division and elongation. In the early 1960s, the concept was developed that these two responses are separable (Haber 1962). Auxin can have dual actions on growth: it can influence growth by expansion, and it can influence mitotic activity.

Distinct separable auxin-regulated responses were demonstrated in the *alf4-1* mutant (Celenza and others 1995). One response induces lateral root primordia and one inhibits root elongation. Lateral roots are initiated from coordinated cell divisions along the primary root in the pericycle, a layer of cells that surrounds the vascular tissue. These clusters of mitotically active cells, lateral root primordia, first appear as a ball-like structure within the primary root, divide, then protrude through the epidermis of the primary root. The *alf4-1* mutant fails to make lateral roots, and auxin fails to stimulate any lateral root induction. However, root elongation of the *alf4-1* mutant could be inhibited by auxin. These two observations suggest distinct signal transduction pathways.

Two separable auxin responses have also been demonstrated with cultured cells. Tobacco BY-2 cells (Hasezawa and Syono 1983; Nagata and others 1992) require auxin for proliferation in culture and these cells can be synchronized and grow rapidly. Auxin concentrations affect cell growth. At relatively low concentrations of auxin, such as 0.3 μM IAA, BY-2 cells elongate without dividing, whereas at higher concentrations, BY-2 cells divide with regenerative expansion. Cell division and elongation are clearly separable in these cells. Cell expansion was maximum at 0.3 μM IAA, whereas cell division and [^{3}H]-thymidine incorporation (DNA synthesis) were maximal at 30 μM (Figure 1). The *ABP1* antisensed line lacks auxin-induced cell elongation, but retains auxin-induced cell division (Chen and others 2001a, b).

The questions remaining are how the plant cell perceives auxin, and how auxin at different concentrations can trigger different responses. One solution would be that the auxin receptor has two different binding sites, high- and low-affinity, respectively, mediating cell elongation and division. The best auxin receptor candidate, ABP1, having only a single high-affinity binding site, does not fit this criterion. Evidence discussed below will support a hypothesis that ABP1 mediates only the cell elongation component of this auxin signal network. Consequently, we need to ask if there is another auxin receptor mediating auxin-regulated cell division.

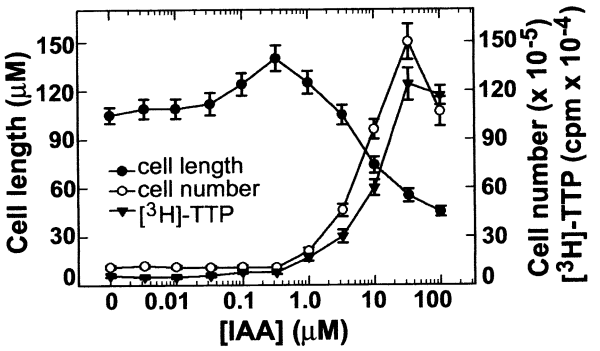

Figure 1. Regulation of cell expansion and division by auxin in tobacco BY-2 cells. The biphasic dose response to auxin suggests two auxin pathways, one leading to expansion mediated by a high-affinity auxin receptor, and one leading to cell division mediated by a low-affinity auxin receptor. Seven-day-old cells were washed in hormone-free media and transferred to media containing the indicated concentrations of IAA. After 4 days, cell length and cell number were determined and [^{3}H]-thymidine was added. Twelve hours later, [^{3}H]-thymidine incorporation was determined. The values represent the mean of cell length (●), cell number (○) and [^{3}H]thymidine incorporation (▼) ± SEM of three independent experiments from 50 ml cells.

ABP1 Mediates Auxin-Regulated Expansion

ABP1 is a low-abundance, pioneer protein found in greatest amounts in the elongation zones of aerial tissues (Jones 1994; Napier 1995; Napier and Venis 1995). It binds most auxins with affinities correlating with their potency to induce growth of cells in the etiolated shoot. ABP1 is predominantly an endoplasmic reticulum lumenal protein, but data from electrophysiological, biochemical, and cytological studies suggest that ABP1, together with its ligand, may interact with a plasma membrane factor where it alters ion fluxes (Klambt 1990; Ruck and others 1993; Thiel and others 1993; Barbier-Brygoo and others 1989, 1991; Claussen and others 1997; LeBlanc and others 1999).

Substantial evidence points to a role for ABP1 in mediating auxin-induced expansion. Ectopic and inducible expression of *Arabidopsis ABP1* in tobacco leaves confers auxin-dependent cell expansion in cells normally lacking auxin responsiveness (Jones

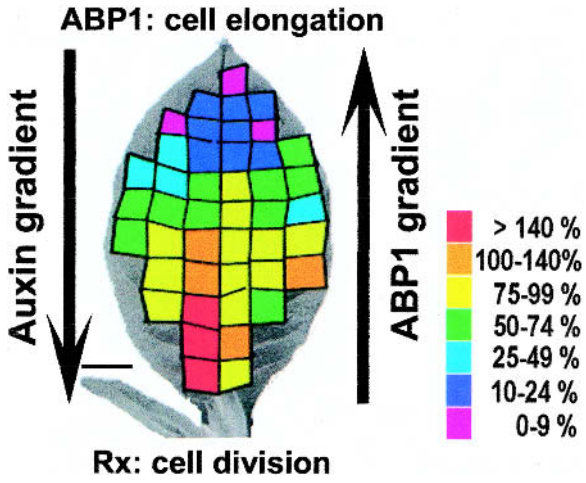

Figure 2. Auxin and ABP1 gradients in a developing tobacco leaf. Cell division moves basipetally followed by cell expansion. The leaf shown is at a stage where expansion is beginning toward the base and waning toward the tip. Growth kinetics of young tobacco leaves (10–20 % of full size) were measured digitally at 24-h intervals. Growth at each position was expressed as the percent increase in area over each 24-h interval (color coded and mapped along the leaf). Bar, 1 cm. Leaves supporting both expansion and division showed a basipetally increasing longitudinal gradient of free IAA with the lowest level at the tip where elongation occurred, and highest at the base where cells were actively dividing. In contrast, the amount of ABP1 is highest at the tip and lowest at the base. It is proposed that ABP1 acts at relatively low concentrations of auxin to mediate cell elongation, whereas high levels of auxin stimulate cell division via an unidentified receptor (Rx). Adapted from Chen and others (2001a).

and others 1998). Over-expression of maize ABP1 in tobacco leaf cells enhances auxin sensitivity (Bauly and others 2000). *Arabidopdsis abp1* null mutant embryos arrest at a time during embryogenesis when cell expansion normally drives the transition from globular to heart-stage, the point when the embryo visibly obtains its axiality (Chen and others 2001b). Antisense suppression of ABP1 in BY-2 cells blocks auxin-induced cell elongation (Chen and others 2001a, b), and inducible antisense suppression of *ABP1* reduces cell elongation in *Arabidopsis* hypocotyls (J.G. Chen and A. M. Jones, unpublished results).

Furthermore, studies on tobacco leaf growth demonstrate that ABP1 acts at relatively low auxin levels to mediate cell elongation (Chen and others 2001a). Leaf morphogenesis is driven by both cell division and expansion. Division begins at the apex of the primordium and moves basipetally ahead of a wave of cell expansion (Figure 2). Free IAA levels were equally high all along the length of the lamina where cell division occurred at all positions. A basipetally increasing longitudinal gradient of IAA with the lowest level at the tip, the site of elongation and highest at the base where cells were actively dividing, was found in developing leaves (Figure 2). The IAA gradient was the inverse of the ABP1 gradient and these gradients collapsed as the leaf aged. Leaves that were nearly fully expanded had low but equal amounts of IAA (with high ABP1) at all leaf positions.

The gradients of auxin and ABP1 in the leaf are also consistent with the spatial distribution of auxin-inducible cell expansion and cell size (Chen and others 2001a). In a young leaf, only cells in the tip region (lowest auxin and highest ABP1) are able to respond to auxin in terms of epinastic curvature (Keller and van Volkenburgh 1997; Jones and others 1998), and cells in the tip are larger than the cells in the middle and base regions. In a mature leaf, cells in the tip, middle, and base regions (low and equal amounts of auxin, high and equal amounts of ABP1) respond maximally to auxin, and no significant differences in epidermal cell sizes were found in these regions. These correlations imply that cell division induced by high auxin is not likely to be mediated by ABP1.

The data suggest that ABP1 is a high affinity receptor mediating auxin-regulated cell elongation at low auxin levels. Little is known about the role of ABP1 in cell division but the current evidence suggests that ABP1 has an indirect effect on division via pathway crosstalk. For example, *abp1* null mutant embryos consistently show aberrant cell divisions, errors shared by some auxin mutants, but this may be an indirect effect from a lack of cell expansion (Chen and others 2001b).

One Auxin Receptor Or More?

Auxin signal transduction embodies a complex network that has frustrated many researchers, leading some to call them "mysterious molecules" (Leyser and Berleth 1999). If ABP1 does not directly mediate auxin-regulated cell division, there must be another auxin receptor. Recent evidence provides insight on what that receptor may be.

Auxin-regulated Cell Division Involves a Heterotrimeric G-Protein

Auxin is involved in the cell cycle (Francis and Sorrell 2001). The G protein alpha subunit (GPA1) is a positive modulator of cell division. GPA1 is present through all stages of development and in all organs with the exception of mature seeds (Weiss and oth-

ers 1993). GPA1 is present at a high level in the root meristem and elongation zone, in the shoot and floral meristems, and in the leaf primordium and floral organ primordia, where cell division is active (Ma and others 1990). Antisense suppression of G-alpha in rice caused dwarfism (Fujisawa and others 1999) which was due to shortening of all internodes. Microscopic observations revealed no significant differences in the length of internode cells between the dwarf transformants and the control plants, suggesting that the internode shortening may be attributed to a decrease in the number of cells per internode and so to a reduction in the frequency of cell division. *GPA1* knockouts in *Arabidopsis* showed reduced cell division, whereas *GPA1* -overexpressing lines showed ectopic cell division (Ullah and others 2001). This suggests that GPA1 couples signals that modulate cell division.

When an extracellular stimulus activates a cell surface G-protein-coupled receptor (GPCR), GTP binds to the α subunit (Gα) in exchange for a bound GDP. GTP binding causes dissociation of Gα from the βγ dimer (Gβγ). Gα, Gβγ, or both then activate a downstream target protein, which results in the relevant cellular response. Endogenous GTPase activity returns it to an inactive GDP-bound sate, accompanied by reassociation with Gβγ (Hepler and Gilman 1992). Circumstantial evidence supports a role for G-proteins mediating the auxin signal. The very first evidence of G-protein involvement in mediating auxin response comes from the work of Zaina and others (1990), who found that IAA increased binding of [^{35}S] GTP-γ-S, a non-hydrolyzeable ^{35}S labeled GTP analog, to rice coleoptile membrane vesicles twofold compared with no-auxin control. This indicated that auxin stimulated the exchange of GDP for GTP. GTP-γ-S also reduced binding of [^{3}H] IAA to rice coleoptile membrane vesicles. These results may suggest that binding of GTP-γ-S activated the G-protein which led to the desensitization of the receptor (Ma 1994).

Auxin-induced K^+ channel expression is also an essential step in maize coleoptile growth and gravitropism (Philippar and others 1999). G-proteins in plants regulate both the inward- and outward-rectifying K^+ channels (Fairley-Grenot and Assmann 1991; Li and Assmann 1993; Saalbach and others 1999; Wang and others 2001). GTP-γ-S reduced an inward K^+ current, and GDP-β-S (inactive G-protein) enhanced the current (Fairley-Grenot and Assmann 1991). However, the picture is not straightforward. Both cholera toxin (activates Gα) and pertussis toxin (inactivates Gα) inhibited the inward K^+ current. More confusing both GTP-γ-S and pertussis toxin induced stomatal opening in the *Commelina communis* guard cells (Lee and others 1993). A peptide that mimics the portion of GPCR that activates G-protein, mas7, strongly inhibited the inward K^+ currents in a manner similar to GTP-γ-S (Armstrong and Blatt 1995), suggesting the presence of a GPCR in *V. faba* guard cells. It is poorly understood how G-proteins modulate these auxin-regulated K^+ channels. Obviously, missing from the picture is the GPCR or some other component that activates the G-protein.

Is There a GPCR for Auxin?

In mammals, G-proteins mediate a wide diversity of signaling through membrane delimited GPCRs. Neurotransmitters, hormones, light, and compounds having taste and smell utilize GPCRs. A few known downstream effectors are adenylate cyclase, cGMP phosphodiesterase, phospholipase C, phospholipase A2, and potassium channels. However, only one plant homolog of GPCR, GCR1, has been identified (Josefsson and Rask 1997; Plakidou-Dymock and others 1998), but its function remains unknown (Humphrey and Botella 2001; Kanyuka and others 2001). Although Armstrong and Blatt (1995) provided evidence that there may be a GPCR mediating *V. faba* guard cells K^+ channels, such a GPCR has yet to be identified.

Is There an Alternative Auxin-Signaling Pathway Mediated by G-Protein?

Obviously ABP1 does not fit the criteria of a GPCR, and there is no compelling evidence yet that there is going to be a GPCR for auxin. Is there an alternative signaling pathway mediated by G proteins?

Recently, Christensen and others (2000) cloned *PINOID* (*PID*). The sequence of PID is predicted to encode a serine-threonine protein kinase with noteworthy similarity to known G-protein coupled receptor kinase (GRK), although mostly within the catalytic domain. GRK is involved in regulation of hormone signaling occurring via GPCRs. Prolonged stimulation of GPCR leads to rapid waning of signals termed as desensitization of the GPCR. The GRKs negatively regulate G-protein-coupled signal transduction by phosphorylating agonist-occupied GPCRs (Pitcher and others 1998). Protein kinase C (PKC) is also involved in the heterologous desensitization of GPCRs. The PID sequence shows similarity to almost all known GRKs (Figure 3A), though the GPCR binding site has yet to be identified. The sequence also showed similarity with various known subtypes of PKC (Figure 3B). This indicates that PID could have GRK or PKC activity and negatively regulate

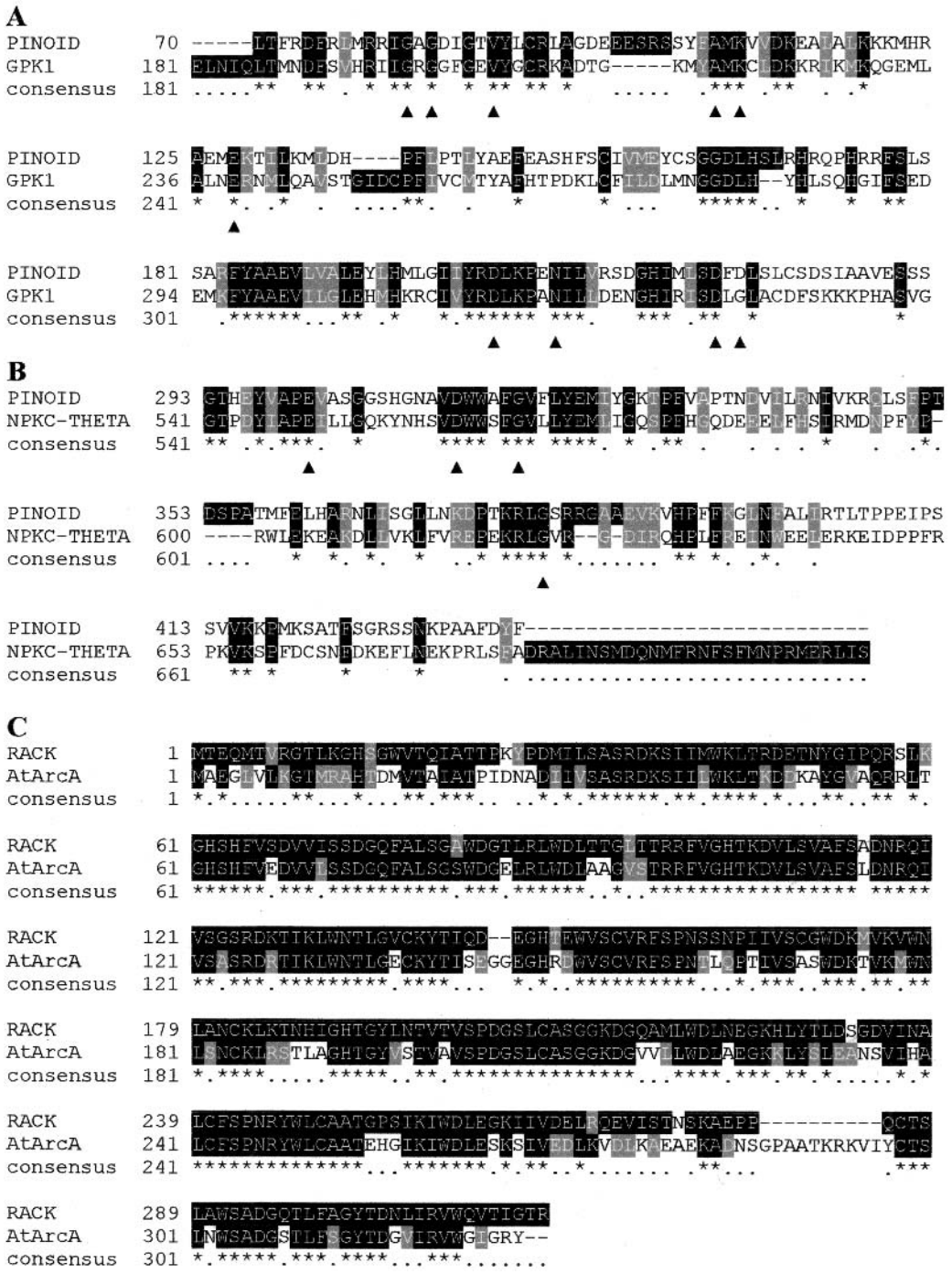

Figure 3. The sequence similarities between PINOID and GRK (**A**), PINOID and PKC (**B**), and ArcA and RACK (**C**). Arrowheads indicate invariant protein kinase residues that define the catalytic domain. There is a replacement of an invariant glycine with aspartate at position 225 in PINOID. (**A**) Partial lineup between PINOID and *Drosophila melanogaster* GPK1 (accession no. P32865, G protein-coupled receptor kinase 1). PINOID has 30% identity with GPK1 within 350 amino acids. (**B**) Partial lineup between PINOID and human NPKC THETA (accession no. Q04759, protein kinase C, THETA type). PINOID has 29% identity with NPKC THETA within 358 amino acids. (**C**) Lineup between ArcA and *Oreochromis niloticus* RACK (accession no. O42249, receptor of activated protein kinase C). ArcA has 65% identity with RACK overall.

G-protein-coupled signal transduction. Phenotypes from the mutations in *PID* share a similarity with other known auxin mutants, in *PIN,* several *ARFs* (*ETTIN*), *MP* (*ARF5*), and *Aux/IAA* transcription factors (*IAA3*). Though there is no experimental evidence to tie the mutants directly to G-protein-coupled signal transduction, shared phenotypes suggest a common pathway. This raises the possibility that PID is involved in G protein-mediated auxin signaling, but how might PID accomplish this?

This brings us to *ArcA,* a WD-40 repeat, G protein beta subunit like protein, cloned from the tobacco BY-2 cells (Ishida and others 1993). The expression of *ArcA* is regulated by auxin. ArcA shows extensive sequence similarity with a group of receptors of activated protein kinase C (RACK) (Figure 3C). RACK, a protein kinase C scaffolding protein, is a membrane-associated protein that binds the C2 domain of PKC and is related in sequence to the beta subunit of heterotrimeric G-proteins. Considering PID's similarities to GRK and PKC, ArcA may bind to PID and bring it to the target protein, possibly GPA1 or a GPCR.

It should also be pointed out that there are other alternative modes of stimulus input into heterotrimeric G-proteins that do not require direct interaction of the G-protein with GPCR. For example, in *Saccharomyces cerevisiae,* two mammalian proteins, AGS2 and AGS3 (activators of G-protein signaling), can activate the pheromone response pathway at the level of heterotrimeric G-proteins in the absence of a typical receptor (Takesono and others 1999). It was found that the mechanisms of G-protein activation by AGS2 and AGS3 were distinct from that of a typical GPCR (Takesono and others 1999). Although possible homologs of AGS2 and AGS3 have not been identified in the *Arabidopsis* genome, it should be possible that such a receptor-independent G-protein activation signaling pathway exits in plant considering there is only one Gα, one Gβ, two Gγ, and only one putative GPCR identified so far.

Fill the Black Box

As discussed above, it is possible that auxin controls cell elongation and division separately, through ABP1 and a G-protein signaling pathway. However, the up- and down-stream elements remain even more elusive.

It has been shown that K^+ channels are involved in auxin-induced cell elongation (Claussen and others 1997). Overexpression of ABP1 enhanced the guard cell K^+ channel sensitivity to auxin (Bauly and others 2000), supporting a role for K^+ channels in the auxin-regulated cell elongation pathway. Recently, Yang and Poovaiah (2000) provided molecular and biochemical evidence that calcium and calmodulin (CaM) are directly involved in auxin-mediated signal transduction. A CaM-binding protein, ZmSAUR1, was found to have sequence similarity with SAURs (small auxin up RNAs). The CaM antagonist inhibited the auxin-induced cell elongation but not the auxin-induced expression of *ZmSAUR1* (Yang and Poovaiah 2000), indicating that calcium/CaM has a role only in the auxin-regulated cell elongation pathway.

Substantial evidence supports the likelihood of crosstalking. For example, G-proteins are likely to act in both cell division and elongation pathways (Fairley-Grenot and Assmann 1991; Li and Assmann 1993; Saalbach and others 1999, Wang and others 2001). Ectopic expression of *Arabidopsis ABP1* in tobacco led to larger but fewer leaf cells but did not change leaf morphology or growth kinetics (Jones and others 1998), suggesting that enhanced cell elongation is compensated by reduced cell division.

One other intergrating possibility is a regulatory role for MAP kinases (MAPK). Another is for phosphatidylinositol turnover. As early as 1984, Morre and others reported that auxin caused rapid phosphatidylinositol turnover in isolated soybean membranes. Ettlinger and Lehle (1988) showed that auxin could generate transient changes in inositol-1,4,5-trisphosphate (IP3) within minutes in *Catharanthus roseus* cells arrested in G1. Recently, Perera and others (1999) showed that the gravity response—a typical auxin-mediated process—is preceded by a transient and sustained increase in IP3 resulting from phosphatidylinositol turnover in gravistimulated internodal maize pulvinus. The possible involvement of a MAPK in auxin signaling was also implicated from biochemical experiments in cultured tobacco cells (Mizoguchi and others 1993, 1994). Auxin starvation arrests cell division whereas readdition starts the cell cycle in tobacco cell culture. During this process, a MAPKK and a protein kinase that has the properties of a MAPK were activated, suggesting that a MAPK pathway is involved in auxin signal transduction. However, Tena and Renaudin (1998) claimed that it is cytosolic acidification, not auxin, that is the activator of MAP kinases in tobacco cells. Recently, Mockaitis and Howell (2000) found auxin treatment of *Arabidopsis* roots could transiently induce increases in protein kinase activity with characteristics of mammalian ERK-like MAPKs. The MAPK response was the result of hormonal action of biologically active auxin rather than a stress response because auxin-induced MAPK pathway signaling was distinguished genetically in

the auxin response mutant *axr4,* in which MAPK activation by auxin, but not by salt stress, was significantly impaired (Mockaitis and Howell 2000). Furthermore, inhibitors of a mammalian MAPKK blocked auxin-activated transgene expression in BA3-GUS seedlings, while potentiating higher than normal levels of MAPK activation in response to auxin. These results indicate that MAPK pathway signaling is positively involved in auxin response and that interactions among MAPK signaling pathways influence plant responses to auxin (Mockaitis and Howell 2000). It also appears that the signaling pathways leading to auxin and salt stress activation of MAPKs in roots are genetically separable. However, in a mesophyll protoplast system, Kovtun and others (1998) showed that transient overexpression of a tobacco MAPKKK, NPK1, activates a MAPK cascade that leads to the suppression of early auxin response gene transcription, indicating that MAPK signaling is negatively regulating auxin responses. A putative ortholog of NPK1, *Arabidopsis* ANPs interfered with auxin-inducible transcription under conditions in which it was activated *in vivo* by H_2O_2 treatment (Kovtun and others 2000), suggesting that oxidative stress signaling through this MAPKKK negatively influences auxin signaling to the *GH3* promoter. Further experiments like testing for constitutive MAPK activity in G-protein knockout plants will strengthen the evidence for involvement of MAPK in auxin signaling pathways.

Aux/IAA and auxin response factor (ARF) proteins are other important elements in auxin signaling (for details, see perspectives by Guilfoyle and Hagen and Tian and Reed in this issue). Biochemical and molecular approaches have suggested that Aux/IAA proteins might be transcription factors involved in the secondary phase of auxin-responsive gene expression (Abel and others 1994; Abel and Theologis 1996). ARFs bind to auxin-response elements found in primary or early auxin-response gene promoters and contain conserved N-terminal DNA-binding domains and C-domains similar to those found in Aux/IAA proteins (Ulmasov and others 1997). It has been proposed that Aux/IAA proteins function along with ARFs in regulating early auxin-responsive gene transcription (Guifoyle 1998). However, it remains unclear how Aux/IAA and ARFs interact with other signaling elements in the auxin pathway.

The other important cascade in auxin signaling is the AXR1-TIR1 pathway (for details, see the article by Ward and Estelle in this issue). *AXR1* encodes a protein similar to the N-terminal half of ubiquitin activating enzyme E1 (Leyser and others 1993). *TIR1*encodes an F-box protein with a series of leucine-rich repeats (Ruegger and others 1998). How do AXR1 and TIR1 fit in the whole auxin signaling pathway? One possible model is as follows: in response to hormone, TIR1 recruits one or more repressors of auxin responses to an SCF ubiquitin-ligase complex. Ubiquitination of this response is dependent on the AXR1-ECR1-mediated RUB1 modification of ATCUL1. Subsequent degradation of the repressor via the 26S proteasome depresses the auxin-response pathway resulting in the expression of auxin-regulated genes and auxin-mediated growth and development (Gray and Estelle 2000). The COP9 signalosome has recently been shown to play an important role in mediating E3 ubiquitin ligase-mediated responses (Schwechheimer and others 2001). Leyser and Berleth (1999) hypothesized that, as ubiquitination requires phosphorylation of target proteins, auxin could regulate the phosphorylation state. Presence of MAPKKK (NPK1, ANP1) in the auxin signal transduction pathway raises the possibility of such phosphorylation, but a link to the first step of auxin signaling, auxin perception, is still missing. No evidence has shown the interaction of AXR1-TIR1 signaling elements to ABP1 or a GPCR, but in yeast and *C. elegans,* the F box domain (present in TIR1) is often associated with WD-repeat proteins, such as Gβ (Smith and others 1999).

Concluding Remarks

Cell division and elongation are fundamental cellular processes in the life cycle of plants. Auxin signal transduction is highly complex and cannot be explained by a simple linear signal transduction cascade. The possible existence of two or more types of receptors linked to distinct signaling pathways may help to explain the complexity of auxin responses (Figure 4). Because cell division and expansion both require new wall synthesis and movement of cell wall material through the endomembrane lumen, there may be elements of the pathways between division and elongation that are shared. The inhibition of cell division found in the *gpa1* mutants and the involvement of G-protein in the K^+ channel (which lead to cell expansion) also suggest the occurrence of cross-talk between these two pathways. These pathways may become integrated at some point to modulate proliferation.

ACKNOWLEDGMENTS

I am grateful to Alan Jones and Hemayet Ullah for stimulating discussions and critical reading of the manuscript and to Richard Napier, Jen Sheen, Pu-

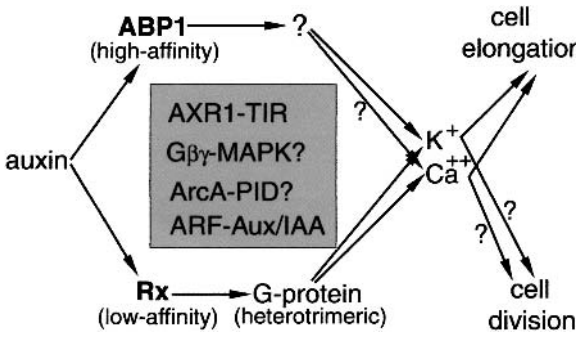

Figure 4. A model shows the complexity of auxin signaling pathways. A high-affinity auxin receptor complex, which probably includes ABP1, controls cell elongation, whereas a low-affinity unidentified auxin receptor (Rx) controls cell division. This complex involves AXR1-TIR, ARF-Aux/IAA, and possibly ArcA-PID and Gβγ-MAPK. The up- and down-stream elements are not yet known.

nita Nagpal, and Jason Reed for invaluable suggestions and comments.

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