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Plant tropisms: providing the power of movement to a sessile organism

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ABSTRACT In an attempt to compensate for their sessile nature, plants have developed growth responses to deal with the copious and rapid changes in their environment. These responses are known as tropisms and they are marked by a directional growth response that is the result of differential cellular growth and development in response to an external stimulation such as light, gravity or touch. While the mechanics of tropic growth and subsequent development have been the topic of debate for more than a hundred years, only recently have researchers been able to make strides in understanding how plants perceive and respond to tropic stimulations, thanks in large part to mutant analysis and recent advances in genomics. This paper focuses on the recent advances in four of the best-understood tropic responses and how each affects plant growth and development: phototropism, gravitropism, thigmotropism and hydrotropism. While progress has been made in deciphering the events between tropic stimulation signal perception and each characteristic growth response, there are many areas that remain unclear, some of which will be discussed herein. As has become evident, each tropic response pathway exhibits distinguishing characteristics. However, these pathways of tropic perception and response also have overlapping components – a fact that is certainly related to the necessity for pathway integration given the ever-changing environment that surrounds every plant.

KEY WORDS: *phototropism, gravitropism, thigmotropism, hydrotropism*

When circumstances become unfavorable for optimal growth and development of animals, they can respond accordingly by moving to a more favorable environment. Plants are not afforded this luxury. Due to their sessile nature, plants are forced to make the most of their immediate surroundings, which means adapting to an ever-changing environment (Liscum, 2002). Darwin described some of these responses to environment more than a century ago in his book *The Power of Movement in Plants* (Darwin, 1880). Darwin noted that plants had a tendency to sense their environment so as to orient themselves for optimal growth and development.

Plants are constantly being bombarded with changes in their environment. Temperature fluctuations, poor light and low water content in the soil are just a few of the factors to which plants must be able to respond. Moreover, plants must respond to physical forces of nature such as gravity or touch stimulation. Over evolutionary time, plants have adapted to their surroundings with a high degree of plasticity, affording them the ability to respond to ever-changing conditions that provide constant stimulation. Plant tropisms are operationally defined as differential growth responses that reorient plant organs in response to direction of physical stimuli. Tropisms can be negative, such as a stem bending away from a gravity stimulation (Blancaflor and Masson, 2003), or they can be positive, as in a stem bending toward a light stimulation (Liscum, 2002). Tropisms are different from nastic plant movements, such as the diurnal movement of leaves or the opening and closing of flowers, in that nastic growth is not directional in relation to a stimulation (Findlay, 1984). With tropic growth, the direction of the stimulation is very important.

Although it has been shown that each tropic response is governed by generally divergent genetic systems, it has become evident in recent years that at least some of the mechanistic features inherent to tropic responses may be shared. It is also apparent that different tropic responses function in coordinating and overlapping ways to give rise to adaptive responses necessary for normal plant growth and development. So how are very different physical stimulations, or inputs, perceived and responded to in such a way to yield outputs - differential growth responses - that are virtually the same? As we are finding in most areas of biology, nothing functions in vacuum. Much of the overlap has to do with the action of plant hormones and how each modulates cell growth. In each case it appears that it is the redistribution of plant hormones

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in response to signal perception that precedes and likely stimulates the differential growth response.

As already mentioned the operational definition of a tropic response is the curve of a plant organ toward or away from a directional stimulation. This can only be accomplished through a differential growth response in which certain cells are actively elongating at a greater rate in one region of the responding organ relative to an opposing position within that same organ. As most work in the area of tropic response has shown, curvature can only be properly manifested through the coordinated activity of hormones. Small fluctuations in the cellular concentration of hormones can have a drastic effect on whether or not a cell is going to rapidly expand or continue to grow at a normal growth rate.

While plants do not exhibit cell migration—the one example being pollen tube growth—they do have the ability to move hormones and other signal molecules between cells as well as over long distances. In plants, the story is this: The cell may not move, but the signal can. In animal systems, hormones may or may not work at the site they are synthesized, but this is not always the case in plant systems. Auxin, for example, is synthesized in the shoot apex but is effective as a morphogen from "tip to tail" (or from shoot apex to root apex). The specific cellular concentration is what will determine what effect the hormone will have at a particular time and place. As in real estate, it's location that matters.

Just like animals, plant hormones are small organic molecules that are most effective at certain concentrations on a cell-to-cell basis. Hormones, being potent growth regulators, tend to be most effective in promoting growth and development at small concentrations. Indeed, large concentrations of certain plant hormones such as auxin or ethylene can actually be growth retarding. But hormones aren't the whole story. Each tropic response has its own special suite of molecules that are necessary for proper signal perception, signal amplification and attenuation and elaboration of the growth response. While the establishment of hormone gradients is a required step in each response, it's not the hormone that does the dirty work. Auxin, for example, acts indirectly through many different proteins to induce a growth response.

Plants have evolved to respond to a variety of environmental circumstances. This review will focus on the four best-characterized tropic responses: phototropism (response to directional light), gravitropism (response to gravity stimulation), thigmotropism (response to touch) and hydrotropism (response to water availability).

I saw the light

Phototropism is the directional growth of a plant organ toward (or away from) a blue-light stimulation. Stems exhibit positive phototropism (growth towards the stimulation), while roots exhibit negative phototropism (growth away from the stimulation). As proposed, this occurs because of a greater rate of cellular elongation on the shaded side of the plant as opposed to the rate on the lit side. This phenomenon has been documented for more than 140 years. In the 19th Century, Darwin postulated that there was "something" being moved from the tip of the plant to the shoot that enabled it to bend toward the light stimulation. In the early portion of the 20th Century, Cholodny (1927) and Went and Thimann (1937), working independently, proposed that it was due to a redistribution of a growth-promoting substance from one side of a plant to the other that lead to the phototropic response. They named this substance "auxin" which is Greek for "to increase" – an appropriate name given its properties to promote cell elongation. It would be many years before the substance was purified and a structure determined, but auxin would become the first plant substance to be termed a "hormone."

How does the perception of photons of light energy lead to a differential growth response that is potentially based on a hormone gradient? First the photons must be perceived by the plant. Blue light-induced phototropic responses utilize a class of chromoproteins known as the phototropins (Figure 1). While other families of photoreceptors such as the phytochromes (Parks *et al.,* 1996; Janoudi *et al.,* 1997) and cryptochromes (Whippo and Hangarter *et al.,* 2003) have been shown to play varying roles in phototropic responses, only the actions of the predominant phototropins will be discussed here. Moreover, most of the genetic and physiological studies discussed here will be limited to those performed in the model plant *Arabidopsis thaliana*.

There are two phototropins in *Arabidopsis*, designated *PHOT1* and *PHOT2*. *PHOT1* was the first of the phototropins to be identified through a screen for mutants that showed impaired phototropic curvature under low-fluence rate blue light (Liscum and Briggs, 1995). Under high fluence blue light, however, *phot1* mutants exhibited a normal phototropic response, indicating the action of another photoreceptor under high light conditions (Sakai *et al*., 2000). The most obvious candidate for a second receptor would be one related to PHOT1. PHOT2 was initially identified through sequence homology to PHOT1 (Jarillo *et al.,* 1998). Its potential role as the second phototropic receptor was cemented when Sakai and colleagues (2001) determined that *phot1phot2* double mutants lack phototropic response in both low and high fluence rate blue light. However, *phot2* single mutants retained an essentially wildtype response under all fluence rates tested (Sakai *et al*., 2001). It was therefore concluded that phot1 and phot2 function redundantly as high light receptors, while phot1 acts as the low-light photoreceptor (Sakai *et al.,* 2001). The phototropins are members of a larger family of sensor proteins known as the LOV domain family (Crosson *et al*., 2003). The family name is derived from the function of the LOV domain as a sensor for light, oxygen or voltage (Huala *et al.,* 1997, Zhulin and Taylor, 1997; Taylor and Zhulin, 1999; Crosson *et al.,* 2003). Each photoropin contains two LOV domains, termed LOV1 and LOV2 (Huala *et al.,* 1997). The non-phototropin members of the LOV family contain just a single LOV domain (Crosson *et al*., 2003). The phototreceptive properties of the photoropins is derived from the non-covalent binding of one flavin mononucleotide (FMN) molecule to each of its LOV domains (Christie *et al.,* 1998; Figure 1). Phototropins are activated through light absorption and subsequent formation of a covalent adduct between the conserved C(4)a atom of the FMN and a conserved cysteine residue within the LOV domain. This adduct formation is thought to initiate downstream signaling through de-repression of the carboxy terminal serine/threonine kinase domain of the phototropin (Christie *et al.,* 2002; Harper *et al.,* 2003, 2004; Figure 1). Interestingly, there are differences in the functional properties of each LOV domain within a given phototropin. Amino acid replacement experiments performed with either the LOV1 or LOV2 domain of phot1 demonstrated that within phot1, only LOV2 abduct formation is necessary for phototropic function (Christie *et al.,* 2002). To date, no clear function has been assigned to the LOV1 domain although recent studies by Salomon and colleagues (2004) suggest that LOV1 may serve as a dimerization domain.

The de-repression of the kinase domain of the phototropins would seem to imply a role for protein phosphorylation in the transduction of the active signal to downstream events necessary for altered growth and development with respect to the phototropic response. There are currently no known phosphorylation substrates for the phototropins aside from the phototropins themselves (Liscum, 2002; Briggs and Christie, 2002). Phototropin interacting partners have been identified. The first phot1-interacting protein to be identified was NPH3 (Motchoulski and Liscum, 1999; Figure 2). *nph3* was another mutant isolated in the same screen that yielded phot1 (Liscum and Briggs, 1995) with null mutations in NPH3 showing a complete loss of phototropic response (Liscum and Briggs, 1996; Motchoulski and Liscum, 1999). *NPH3* turns out to be a member of a large 33-member family *Arabidopsis*, designated the *NRL* (NPH3/RPT2-like) gene family (Motchoulski and Liscum, 1999; S. Joo and E. Liscum, unpublished). While most members of the *NRL* family exhibit a conserved domain structure (Motchoulski and Liscum, 1999; Sakai *et al.,* 2000; E. Liscum, unpublished), the protein structure of NPH3 provides little clue about a potential biochemical function. It has been hypothesized that NPH3 acts as a scaffolding or adaptor protein to assemble a signaling complex containing phot1 and other unidentified proteins at the plasma membrane (Motchoulski and Liscum, 1999). A critical role for NRL proteins in phototropism is further suggested by the finding that mutations in *RPT2* also lead to phototropic defects (Okada and Shimura, 1992, 1994; Sakai *et al*., 2000) Moreover, RPT2, like NPH3, interacts with phot1 *(*Inada *et al.,* 2004; Figure 2). RPT2 has also been shown to form heterodimers with NPH3, suggesting a dynamic and complicated signaling complex (Inada *et al.,* 2004; Sakai *et al*., 2000). This plasma-membrane-associated complex could be directly coupled to changes in auxin transport that might be regulated via changes in phosphorylation status (Celaya and Liscum, 2005; Stone *et al.,* 2004; Figure 2).

For more than 100 years, scientists have centered the differential growth necessary for the phototropic curve squarely on the shoulders of auxin. This dependence on auxin is best typified by the Cholodny-Went hypothesis (Cholodny, 1927; Went and Thimann, 1937). In brief, this hypothesis holds that increases in auxin concentration in the shaded flank (relative to the opposing lit flank) of a phototropically-stimulated stem (Figure 2) would result in a shoot that bends toward the light due to auxin-induced growth (Cholodny, 1927; Went and Thimann, 1937). Such a differential accumulation of auxin requires active movement of the hormone. As already mentioned, the plasma-membrane-associated complex including phot1 and other proteins (such as NPH3 or RPT2) could influence auxin transport. A phot1-signalling complex could be working through modification of auxin transporter localization. For example, Blakeslee and colleagues (2004) have recently found that upon blue-light stimulation, PIN1, a facilitator of polar auxin transport (Geldner *et al.,* 2001), delocalizes from the basal wall of the plant cell and that this delocalization does not occur in cells of *phot1* null mutants (Blakslee *et al.,* 2004; Figure 2).

What happens once the auxin reaches the shaded side of the plant? In the same screen that yielded *phot1* and *nph3*, a third aphototropic mutant, *nph4*, was recovered (Liscum and Briggs, 1995,1996) that shows severely altered auxin responsiveness (Watahiki and Yamamoto, 1997; Stowe-Evans *et al.,* 1998). *NPH4* was cloned and found to encode the auxin-responsive transcription factor ARF7 (Harper *et al.,* 2000) ARF7 is a member of a multigene family in *Arabidopsis*, consisting of as many as 23 members (Liscum and Reed, 2002). The finding that an auxin-responsive transcription factor is necessary for proper phototropic curvature gives credence to the long held notion that the phototropic response is based on an auxin gradient and further suggest that changes in gene expression are a necessary component of the phototropic response system (Liscum, 2002; Figure 2).

ARFs can be either transcriptional repressors or transcriptional activators, depending on their variable middle region (MR). ARF7 contains a Q-rich middle MR often associated with transcriptional activators and has indeed been shown to function as an activator (Tiwari *et al*., 2003). ARF proteins also contain a C-terminal dimerizartion domain (CTD) that allows them to homodimerize or

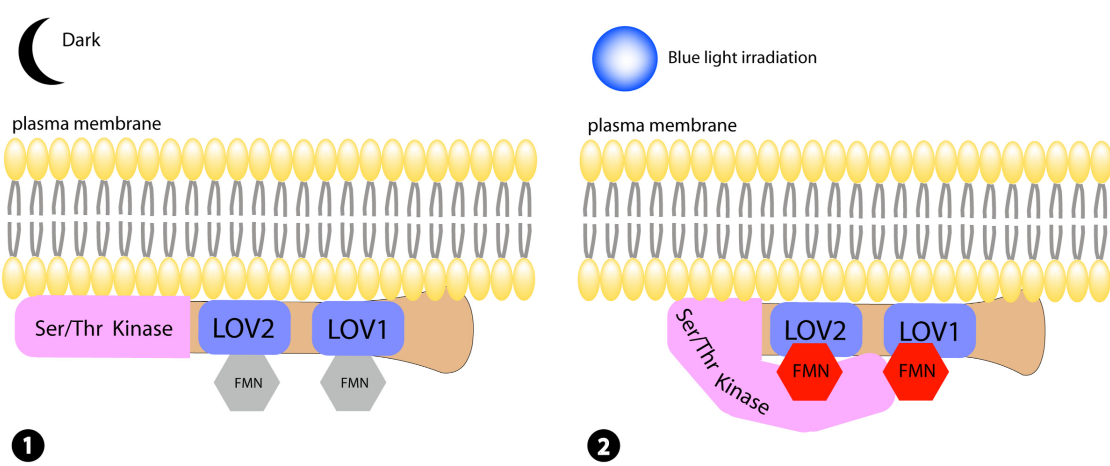

Fig. 1. Model of blue light-dependent activation of phototropins. Arabidopsis *phototropins are plasmalemma associated proteins containing conserved LOV (light, oxygen or volatage) domains which are part of the PAS superfamily and a serine/threonine kinase domain. In the dark one flavin mononucleotide (FMN) is bound non-covalently to each of two LOV domains (left panel) and are activated under blue light irradiation (right panel). LOV domains form covalent adduct with the C(4)a atoms of FMN thus initiating downstream signaling through de-repression of carboxy Ser/Thr kinase domain. It has been revealed that these novel phototropins are their own substrates thus undergoing autophosphorylation and initiating cascade of phototropic signaling events.*

heterodimerize with other ARF family members or to heterodimerize with Aux/IAA family members that share the C-terminal dimerization domain (Ulmasov *et al.*, 1999; Hagen and Guilfoyle, 2002; Tiwari *et al.*, 2003). Lastly, ARF protiens contain a DNA binding domain (DBD) that exhibits homology to the VP1 class of transcription factors (Ulmasov *et al.*, 1999) and allows ARFs to bind to auxin response elements (AuxREs), which can be found in the promoter region of target genes (Ulmasov *et al.*, 1997). There is currently very little known about the function of most ARF family members other than *NPH4/ARF7* with the exception of a small handful. Through loss of function mutant analysis we know that *ETTIN(ET)/ARF3* is necessary for auxin-dependent pattern formation of the gynoecium (Nemhauser *et al.*, 2000; Sessions *et al.*, 1997) and that *MONOPTEROS(MP)/ARF5* plays a role in vascular tissue patterning and differentiation (Hardtke and Berleth, 1998).

How is ARF function connected to auxin? Liscum and Reed (2002) have presented a relatively simple model to explain auxin-regulated ARF function. First, ARFS are thought to bind to AuxREs of target genes as inactive heterodimers with Aux/IAA proteins (Tiwari *et al.*, 2001, 2004; Figure 2). Next, as the auxin concentration rises, turnover of the IAA proteins occurs via SCF^{TIR1} -dependent proteoelysis (Gray *et al.*, 2001; Ramos *et al.*, 2001; Zenser *et al.*, 2001, 2003; Kepinski and Leyser, 2004), allowing ARF-ARF heterodimers to form resulting in active complex (Figure 2). An ARF-ARF7 complex could thus lead to the increased (or decreased in case of repressor ARFS) transcription of target genes in response to increased auxin levels (Liscum and Reed, 2002; Figure 2). Interestingly, ARF7 seems to be targeting its own repressor in the hypocotyl, *IAA19* (Tatematsu *et al.*, 2004) a member of the early auxin response Aux/IAA family of proteins (Theologis *et al.*, 1985). Dominant mutations in *IAA19* that stabilize the resultant protein lead to an aphototropic phenotype reminiscent of *nph4* (Tatematsu *et al.*, 2004). This is in agreement with biochemical results that suggest dominant mutations in Aux/IAA family members lead to a decrease in auxin-stimulated transcription (Tiwari *et al.*, 2001). By increasing the stability of IAA19, protein turnover through the proteosome is decreased and IAA19 remains bound to ARF7, leaving the complex inactive in an increased auxin environment.

But what are the potential targets of NPH4/ARF7 transcriptional activity, besides *IAA19/MSG2* ? Given that the output of the phototropic response is a differential growth response based on differing rates of cell elongation, potentially the targets are enzymes either directly or indirectly involved in loosening of the cell wall (Stone *et al.*, 2004; Figure 2). NPH4/ARF7 could be acting on primary or secondary expansion molecules. This would allow for a greater rate of cell elongation on the shaded side as opposed to the lit side. Some candidate genes might be members of the α-expansin family (Cosgrove, 2000) or perhaps members of the *GH3* and *SAUR* gene families (Hagen and Guilfoyle, 2002). Interestingly, there are AuxREs in the promoter regions of all the previously mentioned gene families (Hagen and Guilfoyle, 2002). What remains unknown is whether NPH4/ARF7 is directly activating genes involved in cell wall modification or if it is activating other transcription factors that in turn may be acting on the cell wall modification enzymes. It is also possible that NPH4/ARF7 is acting in conjunction with another ARF family member through the CBD to lead to transcription of given target genes. Recently, it was reported by Tian and colleagues (2004) that mutations in the *ARF8* gene showed a slight decrease in phototropic response (about 20% in

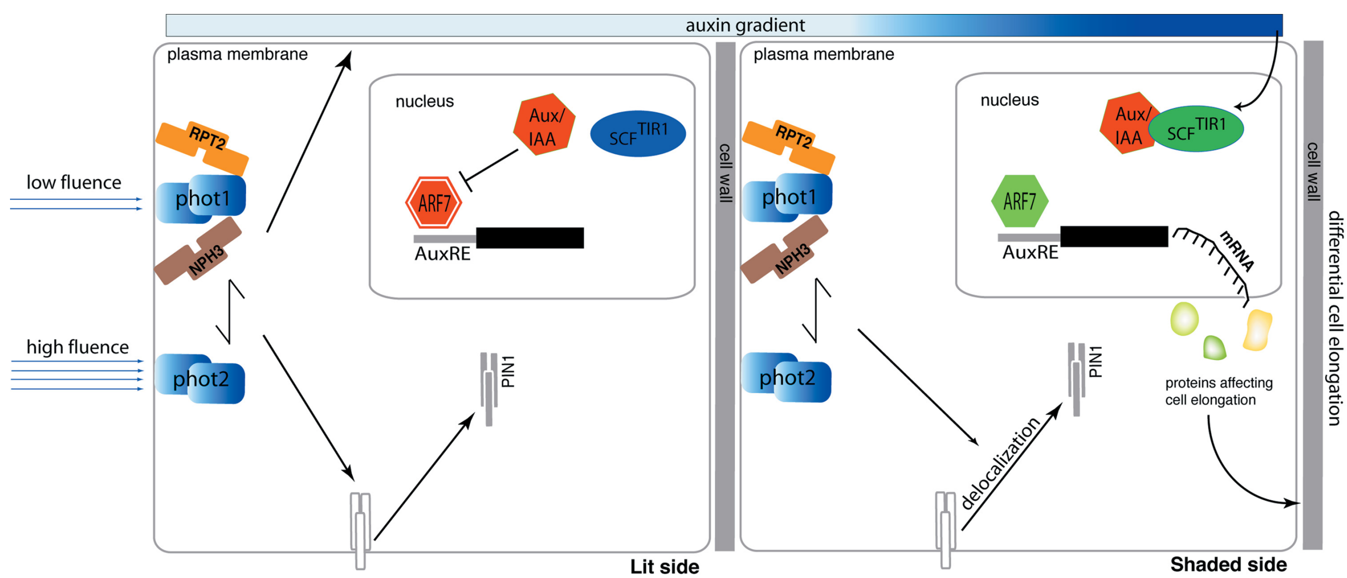

Fig. 2. Scheme of signaling pathway mediated by phototropin associated complex leading to phototropism. *Phototropins (phot1 and phot2) are known to act redundantly as high-light receptors, while phot1 acts as a low-light photoreceptor. NPH3 and its homologue RPT2 are plasma membrane associated proteins known to physically interact with the phot1, thus forming a photo-signaling complex. Under blue light irradiation this phototropin-complex undergoes change in phosphorylation states on the lit side leading to unequal lateral auxin gradient through the tissue with higher auxin concentration in the shaded side (gradient bar, top). This buildup of auxin on the shaded side initiates the SCF^{TIR1} based proteasome degradation of Aux/IAA's that are repressors of auxin response factor 7 (ARF7). In the absence of a repressor, ARF7 bound to an auxin response element (AuxRE) in the promoter region of given target genes is allowed to activate transcription. The gene products of these target genes lead to differential cell elongation on the shaded side thus providing a phototropic curvature. It is also seen under blue light simulation there is delocalization of PIN1, a facilitator of polar auxin transport, from basal wall of the plant cell. Arrows and bars represent promotion and repression activities, respectively.*

comparison to wild type) and that certain *GH3* family members show a decrease in transcript accumulation in a mutant background. While the evidence supplied is not enough to implicate ARF8 as the transcription factor in sole control of these auxin responsive genes transcription level, it is tempting to hypothesize about a potential role ARF8 may play together with NPH4/ARF7 in relation to these targets. Interestingly, recent data would also suggest potential overlap between *MP/ARF5* and *NPH4/ARF7* given the even more drastic vascular defects seen in *mp/nph4* double mutants (Hardtke *et al.,* 2004) However interesting the possibilities for NPH4/ARF7 activity - or for any of the 23 ARF family members - much work remains to determine what the transcriptional targets are for this dynamic transcription factor and its relationship to the phototropic response (Liscum and Reed, 2002).

Center of gravity

As previously stated, plants maintain optimal growth and development despite environmental conditions that are constantly changing. They accomplish this through integration of the many signals to which they are exposed. This includes changes in the direction of gravity stimulation due to changes in growth axis direction. For example, if you rotate a plant 90° from its original growth orientation it will perceive a 90° change in the gravity vector and will, over time, reorient its main growth axis so that it is once again growing vertical relative to the gravity vector. This is a gravitropic response to a change in a plant's gravity field and such a response is one way a plant maintains a proper gravitational set-point angle (GPSA) for a given organ. Each plant organ has a specific GPSA that is wholly dependent upon the age of that organ, the type of organ, what stage it is at developmentally and the environment in which the plant is growing (Blancafor and Masson, 2003). When there is a deviation from the GPSA, a plant responds to the stimulation accordingly through differential cellular elongation on the side away from the stimulation. This results in tip curvature and ultimately the GPSA is regained (Firn and Digby, 1997). How does a change in gravity stimulation lead to a differential growth response? First, there must be signal perception or a sensing of the gravity alteration. Second, there must be signal transduction that ultimately leads to the third step; a directional growth response resulting from differential cellular elongation on opposing flanks of the organ in question.

The most popular explanation for how plants perceive changes to their gravity environment is the starch/statolith hypothesis, whereby starch-filled amyloplasts are displaced when the gravity stimulation changes (Kiss *et al.,* 1989). Amyloplasts are found in the columella cells of the root cap (statoliths) and in the endodermal cells of the shoot (statocytes). When laser ablation was used to remove the central root columella cells in *Arabidopsis*, a large inhibitory effect was seen with respect to root curvature in response to a gravity stimulation (Blancaflor *et al.,* 1998). Genetic studies using mutants that have few or no endodermal cells, lack amyloplasts, or have a problem in sedimentation of amyloplasts have proven to be useful tools in establishing the necessary role of the organelle in a plant's ability to respond to a change in gravity stimulation (for review, see Boonsirichai *et al.,* 2002).

But how does sedimentation of amyloplasts lead to a gravitropic curve? One current idea is that the sedimentation of amyloplasts disrupts the plant cytoskeleton by breaking through the dense local networks of actin microfibrils linked to the plasma membrane (Blancaflor and Masson, 2003). This physical perturbation is proposed to lead to an activation of mechanosensitive ion channels in the plasma membrane (Yoder *et al.,* 2001). Although an initial study in which latrunculin-B was used to disrupt the actin cytoskeleton of maize roots suggested that actin might not be directly involved in the gravitropic response (Yamamoto and Kiss, 2002), a more recent study with this inhibitor indicated that actin is important for gravitropism through modulation of the timing and duration of the response (Blancaflor *et al.,* 2003). Hou and colleagues (2003) have shown that latrunculin-B treated *Arabidopsis* seedlings exhibit persistent increase lateral auxin accumulation accompanied by an increased duration of alkalinization upon gravistimulation. These results have been interpreted as implicating the cytoskeleton in a regulatory capacity that acts antagonistically to the persistent gravity stimulation by constantly resetting the gravitropic-signaling system (Hou *et al.,* 2003).

In more thoroughly understood signaling systems from animals, signals are often amplified by release of second messengers from intracellular stores, such as the role calcium ions play in G-protein linked signaling cascades or the role of cyclic AMP in some hormone-induced signaling mechanisms (Alberts *et al.*, 1989). Two ions represent the most likely gravitropic second messengers; namely calcium ions and protons (Blancaflor and Masson, 2003). Cytoplasmic calcium ($[Ca^{2+}]_{cyt}$) fluctuations have been linked to the transduction of a number of signals, both endogenous and exogenous (for a review of the many affects of $[Ca^{2+}]_{cyt}$ in plants, see Sanders *et al.,* 2002). Unfortunately, it is not trivial to monitor changes in intracellular calcium between different stores. Investigators have had to resort to very indirect methods to gauge the impact of Ca^{2+} on gravitropism such as application of Ca^{2+} channel blockers or Ca^{2+} chelators or by removing/altering the function of certain calcium regulatory proteins such as calmodilin-like proteins (for review see Fasano *et al.,* 2002). Recently, however, Plieth and Trewavas (2002) used a luminescent Ca^{2+} reporter aequorin to look at transient increases in $[Ca^{2+}]_{cyt}$. The intensity of the aequorin luminescence is roughly proportional to the concentration of $[Ca^{2+}]_{cyt}$ and thus serves as an excellent tool to look at increases (or decreases) in ion concentration. Plieth and Trewavas (2002) reported that after gravitropic stimulation seedlings exhibit an intense period of luminescence followed by a steady drop off. Interestingly, other mechanical stimulations don't have the same effect on $[Ca^{2+}]_{cyt}$ spiking (Plieth and Trewavas, 2002). Future experiments should include using this biosensor for calcium in conjunction with amyloplast mutants to see whether a link can truly be drawn between the sedimentation of starch molecules and the transient changes in $[Ca^{2+}]_{cyt}$ (Plieth and Trewavas, 2002; Fasano *et al.,* 2002). Inositol-1,4,5-triphosphate (IP3) is another potential second messenger. When using the cereal pulvuni of oat and maize as a system to study gravitropically stimulated ion fluxtuation, IP3 levels were shown to increase as much as five fold within 10 seconds of gravity stimulation (Perera *et al.,* 2001).

Changes in pH due to fluxes in protons (H+) has also been implicated as a signaling mechanism in gravitropism. An alkilization of the cytoplasm of columella cells has been shown to occur within minutes of gravity stimulation (Scott and Allen, 1999). This is concomitant with an increase in the acidity of the columella apoplast (Fasano *et al.,* 2001). These pH changes are absent in mutants that fail to make amyloplasts or are less sensitive to gravity

(Fasano *et al.,* 2001; Blancaflor and Masson, 2003). A change in pH could depend upon the changing auxin environment as the rates of pH change seem to follow the rate of auxin transport (Monschausen and Sievers, 2002). Thus, as the auxin environment changes, pH changes occur in the root columella, perhaps triggering a feedback mechanism that influences the activity and distribution of auxin transporters allowing for signaling amplification.

It appears from the aforementioned studies that signaling is ultimately coupled to auxin transport and response. Based on indirect evidence using auxin-inducible promoter elements, several researches have shown that there does seem to be a lateral flow of auxin that is manifested upon gravity stimulation (Rashotte *et al.,* 2001, Boonsirichai *et al.,* 2003; Ottenschlager *et al.,* 2003). This lateral flow would thus lead to a differential growth response, that results in a gravitropic curvature. As will be discussed below, much of the evidence supporting the role of auxin in gravitropism has come from studies of *Arabidopsis* mutants.

It should be obvious that for auxin to accumulate in one region of an organ relative to another that new synthesis and/or directional transport of the hormone is required. Since auxin is generally believed to be synthesized only in rapidly dividing regions of the the shoot apex and newly emerged leaves (Bartel, 1997), directional transport must be the mechanism by which lateral auxin accumulation occurs. In unstimulated plants auxin normally travels by two routes from the source of synthesis to rest of the plant where it is utilized: First, via passive diffusion through the phloem cells of the vasculature and second via a polar transport system that links multiple root and shoot tissues. The polar transport system requires transmembrane transporters that can either function to take in auxin from the apoplast (influx carriers) or can serve to shuttle auxin out of a cell (efflux carriers). For an excellent review on auxin transport the reader is referred to a recent review by Friml (2003). The identified influx carriers belong to the AUX/LAX family of proteins related to amino acid transporters carriers (Swarup *et al.,* 2004) while components of the efflux carrier system belong to the AGR/PIN family and MDR-like family of transporters (Noh *et al.,* 2001, 2003). Many members of the PIN family have been implicated in the gravitropic response of roots and shoots (Friml *et al.,* 2002, 2003; Noh *et al.,* 2003; Geldner *et al.,* 2001; Galweiler *et al.,* 1998; Müller *et al.,* 1998), as has AUX1 of the AUX/LAX family of auxin influx carriers (Swarup *et al.,* 2001; Marchant et al. 1999). PIN1 and AUX1 appear to function in transport of auxin from the vasculature to the root tip where PIN4 regulates the channeling of auxin towards more apical columella cells. Once in the columella cells, the presence of AUX1 ensures that auxin will be taken up while the presence of another PIN family member, PIN3, ensures auxin efflux will occur when necessary (Swarup *et al.,* 2004; Friml *et al.,* 2002).

The intracellular localization of PIN3 appears to depend on the root's orientation relative to the gravity vector. PIN3 has been shown to relocalize from a basal to a lateral position within 2 minutes of gravity stimulation (Friml *et al.,* 2002). Interestingly, *pin3* mutants show only a small loss of gravitropic responsiveness (Friml *et al*., 2002). suggesting redundant function for one or more additional PIN family member (Friml *et al.,* 2002). In contrast, *aux1* mutations show dramatic defects in response to gravity stimulation (Chen *et al.,* 1998). Another protein that appears to function in formation of the lateral auxin gradient in response to gravity is ARG1, a ubiquitously expressed J-domain protein (Sedbrook *et al.,* 1999; Boonsirichai *et al.,* 2003). *arg-1* mutants fail to redistribute auxin in the root cap when compared to wild-type plants and they also do not show the characteristic change in pH that is associated with gravitropic stimulation (Boonsirichai *et al.,* 2003). It is currently unknown how ARG1 regulates auxin movement. For example, could ARG1 directly interact with and regulate PIN protein function. One way to address this question would be to examine the localization of PIN family members in an *arg1* mutant background.

The *sgr* (shoot gravitropism) class of mutants exhibit severely impaired (or lack) inflorescence shoot gravitropism and represent another set of mutants that have provided significant new insights into the mechanisms of gravitropic signal response (Fukaki *et al*., 1996; Yamauchi *et al.,* 1997). Many of the *SGR* genes identified via mutant phenotype have now been cloned. *SGR3* encodes a syntaxin-like protein that appears to be targeted to the prevacuolar and vacuolar compartments (Yano *et al.,* 2003) while *SGR4* encodes a SNARE-like protein that is homologous to a yeast protein that is involved in transport of vesicles to the vacuolar compartments (Kato *et al.,* 2002). *SGR3* and *SGR4* were shown to form a SNARE complex that may be involved in vesicular trafficking to the vacuole (Yano *et al.,* 2003). How the vacuole might be involved in the gravitropic response remains undetermined. However, it is possible that the vacuole might serve as a necessary conduit for auxin redistribution, or the interaction of amyloplasts and vacuole during sedimentation might lead to altered tensions in the vacuolar membranes (Blancaflor and Masson, 2003).

But how does redistributed auxin lead to the expansion of only certain plant cells in response to the gravitropic stimulation? The answer is probably through the acitivity of ARF and Aux/IAA proteins through a mechanism like that discussed for phototropism. The finding that *nph4/arf7* mutants show a lack of gravitropic response in the stem is consistent with this notion (Liscum and Briggs, 1995, 1996). Recent microarray studies from the Sederoff lab corroborate this notion of Aux/IAA and ARF activity in the gravitropic response as they found stimulation of *Aux/IAA* and *SAUR* family members within 5 minutes of gravity stimulation in the root tip (Kimbrough *et al*., 2004).

Touch and go

Thigmotropism is the response of a plant organ to a mechanical stimulation. Intuitively, one can imagine that the gravitropic and thigmotropic responses of roots might be intimately related. In fact, a recent study from Massa and Gilroy (2003) suggest that proper root tip growth requires the integration of both a gravity response and a touch response (Massa and Gilroy, 2003). As with the previously discussed responses, thigmotropism requires perception of a stimulus, a signal transduction cascade that amplifies the signal and finally the ability to respond to the touch stimulation through a differential growth response. In 1990, Bramm and Davis initiated the first comprehensive screen to identify components of the mechanosensory response system in *Arabidopsis*. From this screen they found a small group of five genes they termed the *TOUCH* (TCH) family. *TCH1* encodes a calmodulin (CaM), while *TCH2* and *TCH3* encode calmodulin-like genes (Sistrunk *et al.,* 1994). Calmodulin is a highly conserved protein that serves to modulate certain target enzymes under the influence of calcium ions (Allan and Hepler, 1989) and thus one can propose, as was the case for other tropic responses, that Ca^{2+} may play an important second messenger role (Legue *et al.,* 1997).

TCH3 represents a particularly interesting TOUCH protein. First, external calcium application was enough to lead to the increased expression of *TCH3*, suggesting a role for calcium in the feed-back regulation of *TCH3* (Braam, 1992). The fact that *TCH3* accumulates in the cells of the expanding root and shoot and that *TCH3* expression could be artificially induced via exogenous auxin treatment argues for a potential role in cell growth and expansion (Antosiewicz *et al.,* 1995). Recently it has been shown that TCH3 binds to PINOID (PID), a protein serine/threonine kinase, in a yeast two-hybrid assay (Benjamins *et al.*, 2003). PINOID had previously been shown to be necessary for proper auxin signaling (Bennet *et al.,* 1996; Christensen *et al.,* 2000) and a recent study suggests that it acts as a switch to regulate intracellular localization and the function of the PIN familu of auxin efflux regulators (Friml *et al.,* 2004). TCH3 protein appears to bind PID and regulate the ability of the kinase to phosphorylate substrates in response to changing calcium ion levels (Benjamins *et al.,* 2003). While TCH3 has been shown to be phosphorylated, or at least under the repressive activity of a phosphatase, it is apparently not a substrate for PID itself (Wright *et al.,* 2002; Benjamins *et al.,* 2003). The interaction of a potential calcium-signaling intermediate and a protein involved in regulation of auxin transport represents an attractive link between the two signaling mechanisms most commonly associated with tropic responses. Interestingly, not all hormones appear to play a role in mechanostimulation, which is in accordance with previous findings for other tropic responses.

Unlike TCH1, TCH2 and TCH3 that encode calmodulin and calmodulin-like proteins, *TCH4* encodes a xyloglucan endotranglucosylase/hydrolase (XTH). *TCH4* transcript accumulates rapidly (30 minutes) upon touch stimulation and then declines almost as rapidly (in 1 to 3 hours) (Braam and Davis 1990). There are more than 33 *XTH* gene family members in *Arabidopsis* that show relatively varied degrees of sequence homology (Xu *et al.,* 1996; Rose *et al.*, 2002). *TCH4* has also been shown to be upregulated by brassinosteroid (BR) and auxin treatment, but not all *XTH* family members show induction by these hormones (Xu *et al.,* 1995, 1996). Recently it was shown that BR perception is not required for *TCH4* expression, leaving open an interesting question as to how BR is affecting *TCH4* (Iliev *et al.,* 2002).

In vitro, XTHs have been shown to catalyze the cleavage of xyloglucan polymers in the expanding cell wall (Campbell and Braam, 1999; Steele *et al.,* 2001). Xyloglucan is believed to be a tether that holds cellulose microfibrils together in the cell wall, providing tensile strength and restraining cell expansion (Rose *et al.,* 2002). In response to mechanostimulation TCH4 (and/or other XTH family members) could be acting to break the xyloglucan chains, allowing for more elasticity in the wall and thus providing the cell with the capacity to expand and grow with respect to the touch stimulation. These are precisely the kind of wall modification enzymes that might be necessary for a differential growth response under the influence of an auxin gradient, which makes the fact that *TCH4* transcript levels increase in response to auxin application especially interesting. Unfortunately, there is still not much known about the physiological ramifications of these enzymes. There are genome projects underway to try and decipher function for each of the 33 XTH family members (Rose *et al.,* 2002), but much work remains in identifying function of the TCH family members as well as the XTH family members.

Water, water everywhere …

Hydrotropism can be defined as growth or movement in a sessile organism toward or away from water. The best example of this is in plants is the preference of roots for soil with a higher water potential (Takahashi *et al.,* 2002). The idea that plant roots penetrate the soil in search of highest water potential has been long held as truth (Darwin, 1880; Hooker, 1915), however there is very little known about how this actually happens. While we now know that gravity is the driving force behind a root's downward growth and that this growth is modulated by mechansostimulation of soil particles (Massa and Gilroy, 2003), the search for highest water potential is likely playing some role in the integrated growth response. The difficulty in studying hydrotropic growth comes in separation of this response from other tropic responses, gravitropism chiefly among them and drought responses that can occur if plants are water stressed. Further complications arise due to the root cap as the proposed signal integration center for both the gravitropic and hydrotropic responses (Takahashi *et al.,* 2002).

Most studies of hydrotropism have been done using either pea mutants (Takahashi *et al.,* 1991, 1993; Steinmetz *et al.,* 1996), ABA, auxin, or agravitropic mutants of *Arabidopsis* (Takahashi *et al.,* 2002), or maize roots (Takahashi and Scott, 1993) From early work, it is known that calcium is important for a hydrotropic response, as is auxin and potentially other plant hormones (for a review on early work in hydrotropic studies, see Takahashi, 1997). Recently, however, research has focused on using screens for *Arabidopsis* mutants that do not show a hydrotropic response making use of a water potential gradient system. To date, two large-scale screens have been initiated in *Arabidopsis*. The first screen yielded *no hydrotropic response 1* (*nhr1*) (Eapen *et al.,* 2003), while a second screen has yielded 12 putative mutants termed *root hydrotropism* (*rhy*) (Takahashi *et al.,* 2003). Both screens made use of differing water potentials to find mutants that did not show a preference for higher water potential. *nhr1* is a semi-dominant mutation that seems to increase root growth sensitivity to abscisic acid (ABA), a plant hormone known to be involved in drought response (Ishitani *et al.,* 1997). Given the embryonic arrest of homozygous *nhr1* mutants, it is difficult to assign a potential function to *nhr1,* although the authors argue for a role in cell proliferation (Eapen *et al.,* 2003). The *rhy* mutants all show varying degrees of loss of hydrotropic response, but most do not have drastically altered responses to other tropic stimulations, although *rhy4* does seem to have a slight reduction in phototropic response, perhaps signaling an area of overlap between these two tropic responses (Kobayashi *et al.,* 2003; Takahashi *et al.,* 2003). To date, none of the twelve *rhy* mutants has been cloned. Molecular characterization of the *NHR1* and *RHY* loci should be informative as to the mechanism of hydrotropic responsiveness and thus their role in growth and development.

Conclusion

There is still a lot of work to be done before we truly understand how each tropic stimulation impacts a plant and leads to a specific differential growth response. However, recent advances in the various "-omics" should allow for targeted studies that will provide new insights into molecular and biochemical responses of plants exposed to tropic stimuli. The application of mutant analysis to the lesser-studied tropic responses will also shed more light on essential proteins. One thing is certain: As we learn more about each response, we will continue to be amazed by our distant relatives' ability to adapt to changing environments.

All plants move, but they don't usually pull themselves out of the ground and chase you. - Day of the Triffids (1963)

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