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Root Meristem Establishment and Maintenance: The Role of Auxin

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ABSTRACT

Auxin has a central role in the establishment and elaboration of pattern in root meristems. Regulation of root development by auxin begins early in embryogenesis, perhaps even as early as the establishment of polarity in the zygote, and persists throughout the lifetime of a root. Auxin-regulated development depends on a balance of synthesis/import and metabolism/export/sequestration. The overall result of these processes is to establish a state of auxin homeostasis which we hypothesize is required for normal root meristem patterning and development.

Key words: Root meristems; Auxin; *Zea mays*; Auxin maximum

INTRODUCTION

Perhaps the most significant conclusion to emerge from the past 10 years of research on roots is the increasing importance of, and central role for, auxin in many aspects of root development, from embryogenesis to senescence. Much of the new data come from work with *Arabidopsis*, especially from its many mutants. But substantial insights of the involvement of auxin have also come from studies of maize which, because of its relatively larger root, has proven amenable to biochemical analysis and to experimental manipulation. This review focuses on root meristems, with particular emphasis on the involvement of and role for auxin in the development and activities of three populations; the root cap (RC), the quiescent center (QC), and the proximal meristem (PM). In terms of auxin, we consider recent views on how these populations originate during embryogenesis, how they interact and ''communicate'' with each other, and how they function to establish and to elaborate fundamental patterns.

The basic structural features of the root are generally described in reference to a median longitudinal section (Figure 1). In all angiosperm roots the terminal structure is the root cap. Subterminal (proximal) to the cap is the root proper which is composed of files of cells that converge in a region just proximal to the root cap junction (Figure 1). Analyses of the arrangements of cell lineages in roots of many species of angiosperms suggested that the functional, rapidly dividing initials should lie at the point of convergence of the files. Countering this view was the work of Clowes (1954) who instead reasoned that cells located at the point of lineage convergence need not divide very frequently in order for the root to maintain its characteristic architecture. Rather, he concluded that the population of cells located just proximal (basal) to the point of lineage convergence comprised the zone of active initials (Figure 1). By making use of the then recently available radiolabeled DNA pre-

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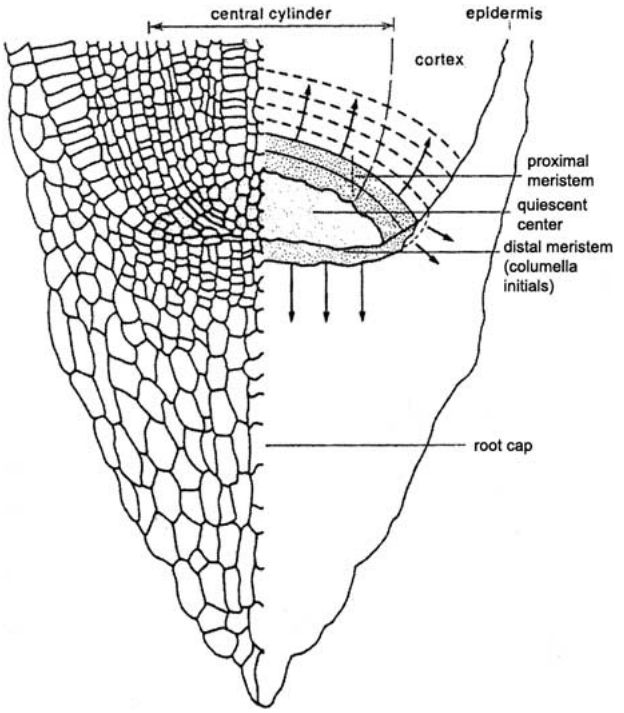

Figure 1. Diagram of a maize root apex in longitudinal section indicating specific cell types and populations, and their relationships to each other. (Modified from Torrey and Feldman 1976).

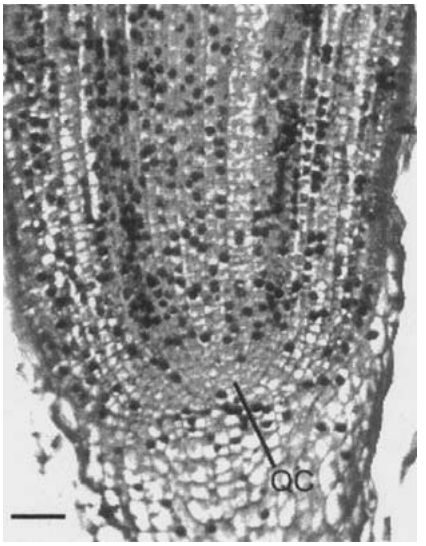

Figure 2. An autoradiograph of a maize root apex as seen in longitudinal section. This root was provided with ^{3}H-thymidine for 12 hours and then this autoradiograph was prepared. The black''dots'' overlay (indicate) nuclei which were synthesizing DNA, and hence preparing to divide. Note the relative absence of labeled nuclei at the point of lineage convergence, the region of the quiescent center (QC). Scale bar = 100 μm.

cursors and the technique of autoradiography, Clowes found that in a growing root, as he had hypothesized, the most highly labeled cells were distributed in an arc spanning the cells located at the point of lineage convergence (Figure 2). Depending on the species, he, and then later others, found that cells comprising the arc divided on an average of about every 20 hours, whereas the non-labeled cells within the arc, including those just proximal to the root cap, divided much less frequently, perhaps every 150–200 hours. Clowes called the group of slowly dividing cells surrounded by this arc the quiescent center (QC) and suggested that the QC is a ubiquitous feature of all angiosperm root apices for some or all of their lifetime. However, he was unable to determine a function for the QC, although he noted that because cells of the QC divided infrequently they would less likely be damaged by environmental extremes (for example, cold or drought), compared to adjacent, more rapidly cycling cells in the arc overlaying the QC. Hence, the QC might serve as a reservoir for replacement of damaged initials in the overlying arc.

Discovering a function of the QC, and the mechanism of its formation, are currently topics of considerable interest to a significant number of investigators. Today, it is generally accepted that in a rapidly growing root, the initials producing the body of the root are many in number and that they form a relatively wide band of cells, of varying depth, and are arranged in arcs overlaying the proximal face of the QC (Torrey and Feldman 1976). Collectively these initials have been termed the proximal meristem (PM) (Figure 1), and in a mature root give rise to all the cells and tissues of the root proximal to the QC. Thus, understanding the structure and activity of the PM are central to comprehending root development. Yet even today not much progress has been made in characterizing this cell-forming region of the root, mainly because of the difficulty in defining its limits. Evidence suggests considerable cross-talk occurs between the QC and PM (Feldman and Torrey 1976). When the QC is caused to diminish in size, as can be done experimentally, so too does the PM contract and narrow, often resulting in marked developmental changes to the root. In circumstances such as extreme cold causing damage to cells of the PM, cells of the QC increase their frequency of division and replace the damaged cells, thereby reforming a new PM. As discussed subsequently, we have recently gained some insight into the nature of communication between populations of cells comprising the root apex (van den Berg and others 1995, 1997).

As is evident from this brief introduction, the root apex consists of dynamic, interacting populations of

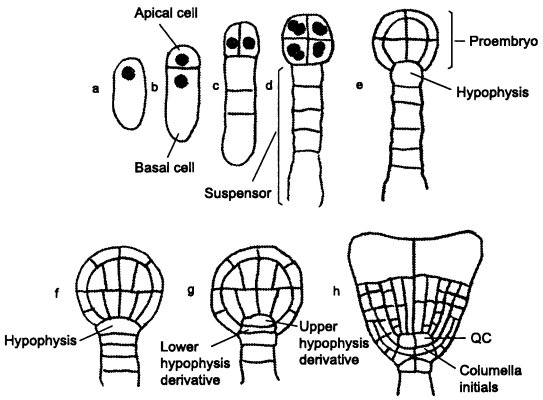

Figure 3. Schematic diagrams of early stages of *Arabidopsis* embryogenesis. After Hanstein (1870).

cells, the sum of whose activities determine patterning and architecture in the root. Recent data now suggest that many aspects of root development can be explained in terms of auxin, with particular emphasis on: (1) the locale of the auxin maximum (maxima) in the root tip; (2) elucidating the circumstances that result in a shift or decrease in the auxin maxima; (3) how auxin is metabolized in the root. These new data on auxin have particular relevance in addressing questions related to embryonic root meristem origin and organization, QC formation and function, and the many postulated roles for the root cap in root development.

Initiation and Establishment of the Embryonic Root in *Arabidopsis*

In *Arabidopsis* the primary root originates and is elaborated as a consequence of a regular, predictable series of cell divisions (Dolan and others 1993; Hamann 2001). Following fertilization, the zygote elongates about 3-fold and then divides unequally, forming a small apical cell and a larger basal cell (Figure 3). The apical cell undergoes three rounds of mitoses to form an 8-celled, two-tiered proembryo. The upper tier gives rise to the shoot meristem and most of the cotyledons, while derivatives of the lower tier contribute to the cotyledons, the hypocotyl and most of the root, including the initials of the root meristem and lateral regions of the root cap. While this is taking place the basal cell divides to form the suspensor. The uppermost cell of the suspensor, which is in direct contact with the proembryo, is designated the hypophysis which subsequently divides producing a lens-shaped upper cell and a larger basal cell. The upper cell forms the QC (4 cells in *Arabidopsis*) while derivatives from the lower cell become the initials for the columella region of the cap. Thus, the root apex is derived from two different cells whose origins can be traced to the very first division of the zygote. In the mature root these two origins are not evident.

Auxin and Establishment of the Embryonic Root Meristem

In *Arabidopsis*, auxin regulation of root meristem establishment begins in early embryogenesis, certainly by the 4-celled proembryo stage, and perhaps even earlier. Although it is not known where or when in the embryo auxin synthesis first takes place, evidence from both mutants and drug studies indicates the importance of auxin in establishing the root meristem (Hadfi and others 1998). Mutants of *BODENLOS* (which is auxin resistant) (Hamann and others 1999), *MONOPTEROS* (which encodes an auxin response factor and affects polar auxin transport) (Berleth and Jürgens 1993; Hardtke and Berleth 1998), and *AUXIN RESISTANT1* (which shows reduced sensitivity to auxin, Leyser and others 1993) do not form an embryonic root. For two of these mutants, *monopteros* (*mp*) and *bodenlos* (*bdl*), the initial regulation by auxin of root meristem development may be indirect. For *mp* mutants, the first recognizable morphological effects of the mutation are seen at the octant stage which forms four rather than two tiers of embryonic cells. For *bdl* mutants, defects are first observed at the 2-cell proembryo stage in which the apical cell undergoes a transverse, instead of a longitudinal division.

Because these defects are observed first in the apical cells of the 2-celled embryo, and only later does a root meristem fail to develop, it has been concluded that it is the apical proembryo cells that are responding to auxin, and subsequently a signal is sent to the basal cells of the proembryo, programming them to form a root meristem (Hamann 2001). The nature of this hypothetical signal is not known, though auxin itself has not yet been excluded. However, the fact that *mp* can be phenocopied by treating isolated embryos (of *Brassica juncea*) with the auxin transport inhibitor NPA (1-N-napthylphthalamic acid) suggests that this signal could be auxin (Hadfi and others 1998). Taken together, the work with auxin-associated mutants and auxin transport inhibitors supports the notion that auxin is involved early in embryogenesis in the establishment of the embryonic root meristem, and that interference with auxin transport causes defects in embryonic patterning.

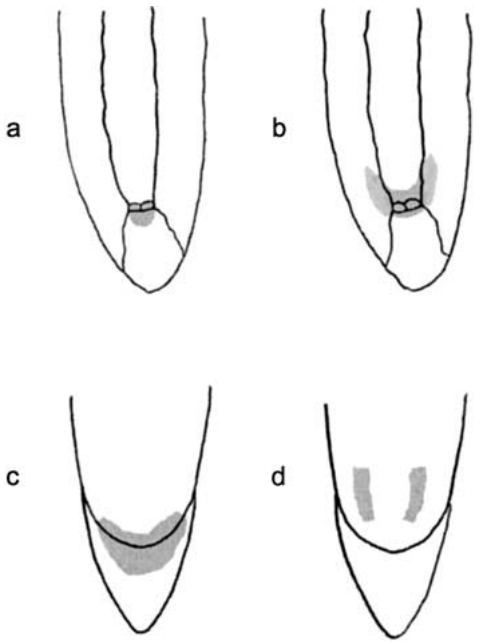

Figure 4. Diagram showing distribution of auxin in *Arabidioposis* (**a** and **b**) and in maize (**c** and **d**) root tips before (**a** and **c**) or after (**b** and **d**) NPA treatment.

Auxin and the Elaboration of the Root Meristem

Following establishment of the embryonic axis, elaboration of the root meristem continues. Even though we are now only beginning to understand the nature of root meristem morphogenesis, it is clear that auxin plays a central role. By using *Arabidopsis* lines transformed with an auxin-responsive promoter (DR5) linked to GUS (DR5:GUS), Sabatini and others (1999) established that auxin is asymmetrically distributed in the root tip, with an apparent high concentration (auxin maximum) in the columella initial/QC region (Figure 4a). Later work by Friml and others (2002) demonstrated that a peak could be detected as early as the heart-shaped stage of embryogenesis, well before the root pole acquired typical root meristem architecture. Sabatini and others (1999) were able to alter the location and shape of this maximum by either using the auxin transport inhibitor NPA, or by using *Arabidopsis* mutants perturbed in either auxin sensitivity or polar auxin transport. Roots treated with NPA showed a remarkable relocation of the auxin maximum; in treated roots the DR5 peak expression not only included the columella initials/QC site, but the peak enlarged and became cup-shaped, incorporating flanking proximal cortical cells (Figure 4b). Similarly, in mutants perturbed in auxin transport, such as *AtPIN1* and *EIR1* (genes encoding proteins mediating cellular auxin efflux), the spatial distribution of DR5 expression was variously affected, and usually involved ectopic DR5 peak levels in unusual positions. In maize too, NPA applications caused shifts in the auxin maximum from the region of the columella initials and the QC to a more proximal location (Figures 4c,d), Thus, interfering with polar auxin transport causes a relocation of the auxin maximum.

Changes in the auxin maximum and distribution also correlate with a change in the status and/or location of the QC. Treating *Arabidopsis* for long periods with NPA not only shifts the auxin maximum to a more basal region of cortical cells, but also leads to the acquisition of QC identity in former epidermal, endodermal, and cortical cells, as indicated by the expression of the QC-specific promoter, QC46 (Sabatini and others 1999). This suggests that the root meristem responds to atypical auxin accumulations by a respecification of cell types. However, unlike in *Arabidopsis*, in NPA-treated maize roots the shifted auxin maximum did not include the previously auxin-rich QC and columella initials. These differences in responses of *Arabidopsis* and maize to NPA may be attributable to the different ways the two roots were treated with NPA or, more likely, the methodology of auxin detection.

In maize an antibody to auxin was used reflecting (within certain limits) the presence of free auxin. This differs from *Arabidopsis* in which one is observing the presence of auxin by its ability to activate a specific auxin-responsive promoter. With this latter approach it is possible to miss detecting some of the auxin if the transcription factors that regulate DR5:GUS expression vary in concentration and type. Some cells may lack transcription factors or signaling components required for DR5:GUS expression, or may contain factors that prevent expression (Guilfolye personal communication). In addition, if transcription factors are very stable, GUS staining may not be an accurate picture of current auxin distribution. Moreover, sequestering of auxin may make it detectable to the antibody but not to DR5:GUS expression. Nevertheless, given these limitations, both the DR5:GUS construct and the antibody can provide information about auxin distribution on a cellular/tissue level.

Paralleling shifts in the shape/location of the auxin maximum were patterning defects in the *Arabidopsis* root meristem, including a distorted organization of the columella (Sabatini and others 1999). In maize, interfering with auxin movement also caused a change in the pattern of the root apex.

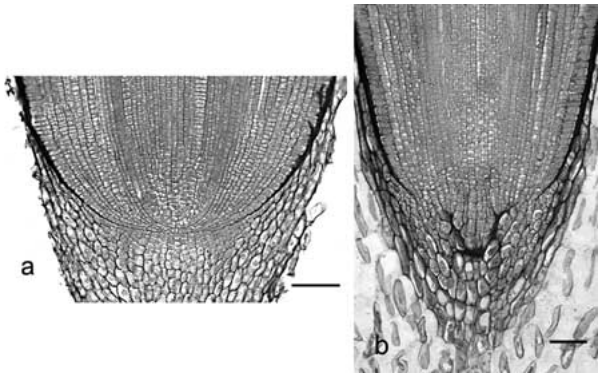

Figure 5. Activation of the quiescent center in maize roots treated with NPA. Control (**a**) and after 2 days NPA treatment (**b**). Note that the (former) quiescent center grows into the root cap (**b**). Scale bar = 100 μm

The QC activates (that is, cells increase their rate of division) and the QC grows (penetrates) into the root cap and eventually displaces it (Figure 5). Thus, there appears to be a linkage between the location of the auxin maximum and patterning of the root meristem.

In summary, from many studies using both mutants and auxin-antagonistic drugs we can conclude that interference with auxin transport alters root meristem morphogenesis (Liu and others 1993; Hadfi and others 1998), and that auxin distribution (gradients in auxin) has a role in patterning root meristems (Sabatini and others 1999). How such gradients arise and are perpetuated are not known, but if this view is correct we must consider how a maximum is developed and maintained in localized regions (such as the QC and columella initials), how the level of auxin is regulated (synthesis, breakdown, polar transport), and whether this hypothesized auxin-controlled patterning involves only a signal imposed from the outside (auxin), or some other signal(s) originating from within the QC or surrounding tissue. In addition, we need to consider what are the intermediate steps/processes linking auxin with the development of the QC? Towards addressing these questions we need to consider the fact that cells take on specific characteristics because of their positions, not because of any inherent, special properties, and that positional signals may originate from more mature regions of the root (van den Berg and others 1995). These results, and those from work with auxin antagonists, are consistent with the likelihood that positional signals move towards the root tip and that the positional signal may be auxin.

Establishment of the Auxin Maximum

The finding that auxin moves polarly in an acropetal direction (probably in vascular parenchyma, towards the root tip) and that it is then redirected basipetally (probably via the epidermis or underlying cortical cells), indicates that the establishment of the maximum reflects a balance between auxin import and export (Rashotte and others 2000). However, in addition to the importance of transport, cellular auxin levels can be regulated by other processes including synthesis, degradation/conjugation, and sequestration. Kerk and others (2000) showed that the root apex is very efficient at auxin breakdown. Thus, the establishment of an auxin maximum involves a precise and localized balance between breakdown and synthesis/import of auxin. Towards deciphering how the maximum forms we need to consider a most unusual characteristic of auxin, namely, that auxin can create a sink towards which more auxin moves (Hertel and Flory 1968; Rayle and others 1969; Veen 1969). In roots this was demonstrated by removal of the root cap (decapping) of maize, which resulted in a 50% decrease in polar auxin transported toward the tip (Feldman 1981). However, if the cap is excised and then replaced with a small amount of auxin (10^{-9} M), not only is polar transport restored, but the amount of auxin moving towards the tip can exceed that in intact (non-decapped) roots. The mechanism by which auxin acts as a sink for more auxin isn't known.

However, the recent finding that AtPIN4 is important in *Arabidopsis* in generating an auxin sink distal to the QC, suggests that this protein may be part of this mechanism, and that this gene may also be involved with the recently discovered phenomenon of basipetal auxin transport (Friml and others 2002; Rashotte and others 2000). AtPIN4 is first detected during the late globular stages of embryogenesis, at which time it is localized to the hypophysis and its sister, the basal cell. In *Atpin4* mutants normal auxin distribution is perturbed, resulting in serious patterning defects in both embryonic and seedling root meristems. In this mutant aberrant cell divisions occur at the presumptive root pole and the QC specific marker, QC25, normally expressed only in the 4 cells of the QC, is expressed in a much broader domain. Thus, an inability to generate a presumed auxin sink in *Atpin4* embryos prevents the auxin maximum from developing at the presumptive root pole and can lead to a change in cell fate specifications.

These results suggest that an important element in the establishment of an auxin maximum is the creation of an auxin sink, and that in *Arabidopsis*, auxin sink establishment occurs early in embryogenesis, perhaps before the QC has formed, and before a root apex has organized. In this context it is believed that AtPIN4 functions either as an auxin (efflux) carrier or as a regulator of auxin transport (Friml and others 2002). But how this presumed function could translate mechanistically into the development of the auxin maximum is not known. A possible explanation may lie in the observations that in the root proper, AtPIN4 is asymmetrically distributed in cells along the walls/membranes facing the root pole, whereas in the root cap, AtPIN4 showed a nonpolar localization in the columella and columella initials and was distributed uniformly on all walls (Friml and others 2002). Because earlier work suggested that auxin is synthesized at low levels in the cap (Feldman 1981) there is also the possibility that both the distribution of AtPIN4 and the AtPIN4-dependent auxin gradient could be mediated by auxin itself.

Communication Between the Root Cap and the QC is Necessary for the Establishment of the Auxin Maximum

Several recent reports suggest that the QC can affect events in the cap, including events in columella initials (Ponce and others 2000; van den Berg and others 1997). In *Arabidopsis*, laser ablation of one or more of the presumptive QC cells changes the fate of adjacent columella initials, which now undergo differentiation. From this work, van den Berg and others (1997) concluded that a signal must normally come from the QC, maintaining the adjacent cells as initials. Thus, destruction of the QC would prevent the formation of this signal, or interrupt polar auxin transport; hence, the surrounding initials, including those for the columella, would undergo differentiation. Whether a signal does indeed originate in the QC, or whether the QC affects columella initial status in some other way is still an open question. In maize, if the root cap is excised, the QC activates and cells of the QC bordering on the former root cap begin to differentiate starch grains (amyloplasts), allowing the root to sense and respond to gravity in the absence of a normal cap. Hence, removal of the cap causes a re-specification of QC cells. This could also be interpreted to mean that, in addition to putative signals moving from the QC to the cap, the reverse can occur and a signal moves from the cap to the QC to maintain QC cells in a relatively undifferentiated, mitotically inactive state. Currently there is no evidence for this suggestion.

Interestingly, removal of the cap leads to profound changes in auxin distribution in the decapped root; auxin accumulates to high levels in the region of the activated QC. It is possible that this auxin accumulation causes or is related to the reprogramming of QC cells to form starch. Moreover, we suggest that this accumulation of auxin is necessary for the development of a (new) auxin sink in the reforming cap. Only after this sink is reformed can ''correct'' auxin patterning, establishment of the auxin maximum, and QC reformation occur. No doubt the formation of the auxin sink and maximum are complicated events. Scheres (2000) notes that factors controlling cell polarity (for example, the distribution of auxin transport proteins) are certainly also important in understanding how the auxin maximum is established. There is also the suggestion that in some cases auxin distribution is related to the placement of vascular bundles (Sabatini and others 1999), which seems logical given the likely path of polar transport (vascular parenchyma). Clearly additional factors could be considered.

The Auxin Maximum and the Proximal Meristem

If the QC is, as hypothesized (Kerk and others 2000), a site for auxin breakdown and action, not only should this influence auxin distribution in the root cap/columella initials, but also one might predict consequences for the adjacent proximal meristem (PM), which borders on the side of the QC opposite the root cap. Although the boundary between the cap and the QC can be very discrete and ''fixed'', as in maize, the border between the QC and PM is not sharp, and can fluctuate in location, as usually happens in roots as they age. Depending upon where the border is located, cells at the juncture of the QC and PM can thus change their fate, sometimes being part of the QC and sometimes being part of the PM. How might we understand these fluctuations in QC size? Previously we noted that in roots treated with the auxin transport inhibitor NPA, the auxin maximum shifts from the root cap/QC boundary to a more proximal location, resulting in the expression of QC markers in the cells newly exposed to high levels of auxin (Sabatini and others 1999). Moreover, moving this maximum not only imposes a new QC identity on additional

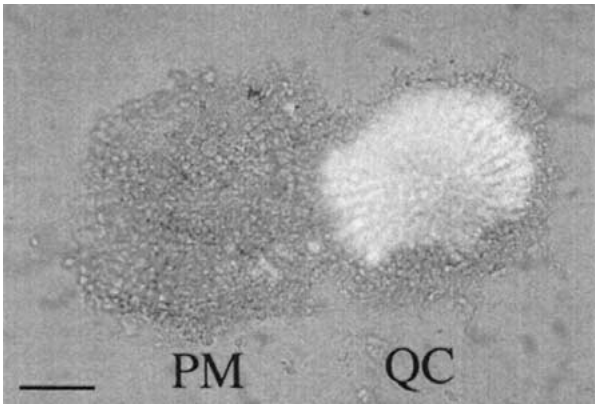

Figure 6. Whole mounts of excised quiescent center (QC) and the adjacent proximal meristem (PM) from roots pre-treated 2–3 hours with the redox-sensitive dye carboxy H2DCFDA. Green color indicates a relatively oxidizing environment/status in the QC. Scale bar = 100 μm.

cells, but the cells from which the auxin maximum has been shifted (removed) lose their QC identity (Sabatini and others 1999). In maize this loss of QC identity is evident with the onset of cell division and consequent diminution in size of the QC (Feldman 1975). Thus, there appears to be a clear correlation between QC size and the location of the auxin maximum, suggesting that cell fate at the QC/PM interface may be directly dependent on placement of the auxin maximum.

An Hypothesis for Auxin Action

Thus far considerable evidence has been presented for a role for auxin in meristem patterning and in QC establishment and elaboration. An assumption for much of this review is that certain levels of auxin translate into or influence the quiescent state. How this may happen is not certain, but perhaps contributing to this understanding are recent findings suggesting that auxin can affect cellular redox status, which in turn influences the quiescent state. The link between polarly transported auxin and cellular redox status is based on several observations: (1) auxin stimulates the synthesis of ascorbic acid oxidase (AAO), the enzyme which converts ascorbic acid, a major cellular redox regulator, from the reduced to the oxidized form (Kerk and Feldman 1995); (2) that AAO is able to degrade auxin (Kerk and others 2000); and (3) that the overall redox status of the QC is highly oxidizing (Feldman and others 2002). Detailed biochemical measurements have been made on both the QC and the adjacent proximal meristem in maize roots. These data show that the status of two major cellular redox regulators, ascorbic acid and glutathione, differ between these two cell populations. In the QC the oxidized forms of both ascorbic acid and glutathione predominate, whereas in the adjacent PM the reverse holds, with relatively high levels of the reduced forms and low levels of the oxidized forms. The redox status of tissues can also be visualized using membrane-permeable dyes which are colorless in the reduced state, but which fluoresce when oxidized. As indicated by its bright fluorescence, the QC has a relatively more oxidized status, and the PM, which shows no fluorescence, thus has a relatively reduced status (Figure 6). However, if polar auxin transport is blocked with NPA, thereby shifting the auxin maximum from the QC to the region of the PM, the reverse fluorescence pattern is observed; the QC is colorless (=a reduced status) and the PM fluoresces (=an oxidized status).

This demonstration of a linkage between auxin and redox may be important for understanding the onset and maintenance of quiescence. For it is now known that cellular redox status can affect cell cycle progression, and that many cell cycle checkpoints are sensitive to oxidative stress (Bijur and others 1999; Reichheld and others 1999). Thus, one potential mode of auxin action may be by mediating the redox status of the presumptive QC, and thereby, indirectly affecting the frequency of cell division. In this way, by regulating redox status at a local level, auxin may act as a positional signal (Berleth and Sachs 2001) and mediate meristem patterning, including QC development. In this regard, the recent report of the *Arabidopsis* mutant *ROOTMERISTEMLESS* (*RML1*) provides strong support for a linkage between redox status and root meristem organization. This mutation leads to a reduction in levels of glutathione and plants are unable to form an active post-embryonic root meristem. But these mutants can be rescued (an organized root meristem reforms) by providing seedlings with reduced glutathione (Vernoux and others 2000).

A Biochemical Explanation for Auxin-Regulated QC Development

What might be the biochemical/molecular pathway(s) by which auxin affects redox status? From studies of root gravitropism in *Arabidopsis*, Joo and others (2001) demonstrate that auxin can stimulate reactive oxygen species (ROS) production in graviresponding roots, and others report that

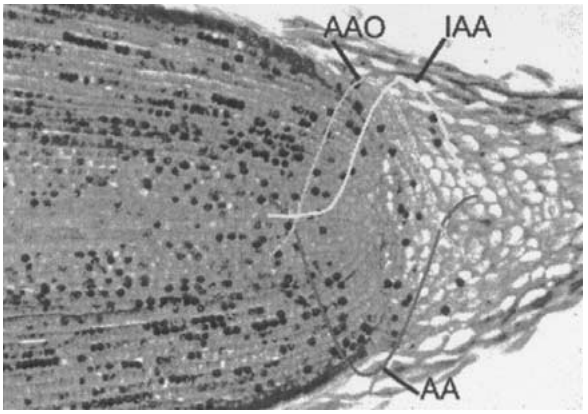

Figure 7. Schematic view of the distribution (location of maxima and minima) of auxin (IAA), ascorbic acid oxidase (AAO), and ascorbic acid (AA) in relation to the quiescent center.

auxin herbicides can lead to overproduction of H_2O_2 or other ROS (Grossmann and others 2001; Pfeiffer and Hoeftberger 2001), though in neither case are the mechanisms for ROS formation proven. But insight into this mechanism may come from studies of auxin regulation of enzymes associated with the formation and/or regeneration of redox intermediates, such as ascorbic acid and glutathione. In most tissues the levels of ROS are kept low by interactions with the reduced forms of glutathione and ascorbate. In the QC, however, there is relatively little reduced ascorbate or glutathione and the enzymes responsible for the regeneration of these reduced intermediates are also at low levels (Feldman and others 2002). However, if polar auxin transport is inhibited by NPA the levels of these key enzymes increase markedly and this corresponds with an increase in the reduced forms of ascorbate and glutathione in the QC, a lowering of the levels of ROS, and an activation of the QC. Thus, as hypothesized earlier (Kerk and Feldman 1995), auxin can regulate key enzymes involved in the maintenance of cellular redox and in this way influence or impose the quiescent state. In roots, therefore, the consequences of ROS generation perhaps should be viewed as requisite for the differentiation and development of the QC.

Summary

Auxin is central to the establishment and maintenance of a root meristem. Within the meristem, auxin is changed (broken down/complexed) or redirected towards the shoot. When auxin transport in the root is perturbed the architecture of the root meristem is affected and the meristem may disorganize. When the root apex is damaged, or the quiescent center caused to activate, transport of auxin in the root is perturbed and auxin is atypically localized in the damaged meristem. We hypothesize that a feedback loop exists between auxin and the root meristem: (1) auxin is necessary for root meristem development, and (2) a meristem is a requisite for auxin breakdown and, hence, for the development of an auxin maximum and auxin homeostasis (Figure 7).

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