INTRODUCTION

Axial, meristematic growth imposes a unique set of constraints upon cell behavior during plant development. Owing to the perpetually embryonic nature of meristematic activity, cell division plays a relatively more constant role throughout development in higher plants than it does in animals. During early embryogenesis in Arabidopsis, for example, stereotyped, formative cell divisions block out domains in which symmetries, axes, and apices arise (Jürgens, 1995; Laux and Jürgens, 1997, in this issue). From late embryogeny forward, the products of embryonic and apical mitoses contribute to ongoing organogenesis by leaving behind anlagen and primordia along an axis composed of nascent dermal, ground, and vascular tissues in roughly concentric domains (see Clark, 1997; Kerstetter and Hake, 1997, in this issue). Although the fundamental role of meristematic cell division is maintained throughout a plant's postembryonic life, the spatial patterns and consequences of these divisions can and do shift, as seen in heterophyllic plants and during floral morphogenesis in all species.

Cell versus Organism

What is the nature of the signal–response circuitry through which cell reproduction is enlisted to perform a plant's genetically mandated morphogenetic plan? In light of lineage-based animal paradigms and the rigidity of plant cell structure, one might presume that a genetically programmed internal engine prompts meristematic cells to divide in stereotypical, spatially appropriate patterns that are essential for generating final morphology. Although this appears to be true in a very limited number of cases, an alternative paradigm relieves the meristem of most of this morphogenetic burden. At the organ level and above, this developmental model falls under the rubric of the so-called organismal theory of plant development (Kaplan, 1992). According to this view, it is not the autonomic division and expansion of meristematic cells and their products, respectively, that drive morphogenesis but the anisotropic expansion of organs whose constituent cells merely partition the resultant volumes via mitoses. Organ morphogenesis is therefore driven by some yet to be defined supracellular agency that need not be encoded in the geometry of cells themselves (Barlow, 1991; Cooke and Lu, 1992).

A cellular corollary can be extracted from the organismal theory and expressed as cell size homeostasis. As the expanding organism creates volumes and surface areas too large for a single nucleus to service, the cell duplicates its organelles, distributes them to longitudinal poles, and partitions the cellular volume with a new wall, thus supplying a second set of organelles to govern the space. Correlative studies suggest that within a given plant tissue, the cellular volume and surface area serviceable by a single nucleus are limited and strictly correlated with the C-value (number of haploid genomes) of that nucleus (Boeken and Van Oostveldt, 1977; Melaragno et al., 1993).

Therefore, the organismal theory and its cellular corollary hold that expansion creates the need for and thus drives mitotic partitioning. However, this informational vector should not be construed as the exclusive force behind plant cell division, even within a single organism. Indeed, data discussed below suggest that plant cells can also enlarge as a consequence of being driven to divide by direct activation of the cell-cycle engine, independent of prior cell expansion. Thus, division can drive expansion, expansion can drive division, and context is all.

If mitosis can act either as master of or slave to development, depending on context, then the signal transduction pathways that link the conserved cell-cycle engine to outer regulatory layers of plant cell physiology and development must be relatively more flexible or diverse than are their animal cell counterparts. However, molecular data that directly address such questions are scarce. Thus, this review uses recent research reports as starting points from which to reexamine assumptions about the place that cell division occupies within the developmental plans of higher plants. In so doing, we hope that future studies of biochemical mechanisms underlying the complex interplay between plant cell division and expansion can be guided by better-informed hypotheses. For additional perspectives on these and related issues, the reader is referred to recent reviews (Barlow,
MITOSIS LEADS, EXPANSION Follows

The angiosperm root is a suitable system in which to examine the relationship between cell division and cell expansion during plant development (Erickson and Sax, 1956a, 1956b; Scheres et al., 1996; Schiefelbein et al., 1997, in this issue). The root apical meristem is basipetally displaced from the organ's distal terminus by the root cap and acropetally displaced from the rest of the plant by the bidirectional expansion of its proximal mitotic products (Figure 1). Cells generated by apical mitoses are contributed distally to the columella of the root cap and proximally to the protoderm, ground meristem, and procambium of the root axis. Cell divisions are most highly concentrated in the promeristem, peripheral to the meristem's quiescent center (Schiefelbein et al., 1997, in this issue).

The probability of a cell's committing to a full turn of the mitotic cycle declines as it is displaced from the promeristem (Erickson and Sax, 1956b). At a species- and tissue-specific distance from the root tip (2.5 mm in maize [Erickson and Sax, 1956b]), the probability of additional mitoses within a lineage drops to zero (Figure 1). This occurs despite the fact that cells in this zone have attained what in the meristem would have been a size sufficient to permit (or, after the organismal theory, to drive) another mitotic division. The relevant cellular and biochemical differences between the meristem proper and the postmeristematic domains are unknown, although such distinctions constitute key functional elements of developmental control.

Do cells basipetal to this point run short of an essential cell division factor, or does the balance of a cell's mitotic promoters and inhibitors shift toward the latter as cells mature?

Ectopic Expression of a Mitotic Cyclin Increases Cell Number

One experimental approach to the question posed above is to express, in various tissues, a protein hypothesized from correlative data to be the limiting factor for cell division and to determine the location of any ectopic mitoses that result. There is a rough correlation between mitotic activity and the presence of cyclin protein in plants (HirI et al., 1992; Fobert et al., 1994). Therefore, one candidate for a protein that limits mitotic activity is CYC1At, an Arabidopsis cyclin expressed exclusively in mitotically active cells (Shaul et al., 1996). Cyclins are regulatory subunits that impart substrate specificity, intracellular localization, and the capacity for catalytic activity upon their otherwise lifeless catalytic partners, the cyclin-dependent kinases (CDKs). The protein CDK1, previously known as CDC2 after the cell division cycle mutant that originally defined it in fission yeast, is the prototypical CDK. When appropriately phosphorylated and partnered with a mitotic (i.e., B-type) cyclin subunit, CDK1 catalyzes the cell's entry into mitosis.

Peter Doerner's group has ectopically expressed cyc1At in transgenic Arabidopsis by placing it under the control of the cdc2aAt promoter. cdc2aAt encodes a likely but unconfirmed Arabidopsis homolog of CDK1. The particular choice of promoter was designed to render the spatial and temporal expression patterns of the regulatory and catalytic subunits of an Arabidopsis CDK complex artificially congruent (although this was not confirmed by in situ hybridization, nor have the products of the cdc2aAt and cyc1At genes been shown to form an active CDK complex in vivo). The experiment was guided by the hypothesis that cells in wild-type plants expressing cdc2aAt but not cyc1At cease to divide for their widowed CDKs' want of cyclin partners. This hypothesis was supported by the observation that roots of transgenic cdc2aAt::cyc1At plants grew faster than those of wild-
type plants, with no significant alteration in mature cell sizes. However, neither cell-cycle times nor the distribution of cell divisions was directly measured in plants ectopically expressing cycl1At. Instead, the conclusion that the longer roots observed in cdc2aAt::cycl1At plants were attributable to elevated cell reproduction rates was deduced from measurements of overall organ length and final cell sizes (Doerner et al., 1996).

The phenotypes of cdc2aAt::cycl1At plants offer two lessons: (1) mitotic cyclin protein concentration can be limiting for cell reproduction rates in the angiosperm root axis; and (2) cells ectopically driven to divide, at least in this developmental context, proceed to expand without being explicitly engineered to do so. The significance of the latter observation can best be appreciated when one considers the seemingly plausible alternative outcome—that the supernumerary cells produced in cdc2aAt::cycl1At roots may not have expanded to produce elongated organs. Unless cyclin protein has some unexpectedly gibberellin-like function in plant cells, elongation seems to have been the default pathway for the extra cells in cdc2aAt::cycl1At plants.

Finding the Source of “Extra” Cells in cdc2aAt::cycl1At Plants

From which anatomical source did new cells arise in cdc2aAt::cycl1At plants? We can narrow the field to root tissues in which the cdc2aAt promoter is active—dividing cells of the root apical meristem (but not the quiescent center), the pericycle, and the vascular parenchyma (Martínez et al., 1992; Hemerly et al., 1993). Ectopic division and expansion of cells comprising the latter two internal tissues would require that the overlying epidermal (and perhaps cortical) layer follow suit. Mitogenic induction between histogenic layers in plants has been documented in studies of chimeric plants (Szymkowiak and Sussex, 1992) and the KNOTTED locus of maize (Smith et al., 1994; Fuerst et al., 1996) are driven by promoters with hand, more refined hypotheses can be fashioned and tested by using new constructs in which cycl1At expression or that of a cyclin active in the G1 phase of the cell cycle (Soni et al., 1994; Fuerst et al., 1996) are driven by promoters with different spatial patterns of expression.

Why Did cdc2aAt::cycl1At Expression Not Produce Tumors?

What prevented elevated CYC1AT levels in the root promeristem from generating tumors or a phenotype analogous to that of clavata1 (clv1), a mutant of Arabidopsis that exhibits hypertrophy of the shoot apical meristem (Clark et al., 1993; Clark, 1997, in this issue)? Perhaps CYC1AT levels are simply not rate limiting for cell reproduction in the root promeristem. Alternatively, hypertrophy of the root apical meristem in cdc2aAt::cycl1At plants may have been averted by the efficient proteolysis of cyclins that occurs at anaphase in actively dividing cells (King et al., 1996). Or perhaps the simple linearity of root geometry accommodates extra cells more gracefully than does the more complex apical organization of the shoot. It would be interesting to cross cdc2aAt::cycl1At transgenics with mutants incapable of maintaining a primary root apical meristem, such as those of the root meristemless1 (rml1) or rml2 genotypes (Cheng et al., 1995). Perhaps ectopic expression of a mitotic cyclin can bypass a developmental
signaling defect in the *rml* mutants, thus placing RML1 and/or RML2 upstream of cyclin activation in the mitotic regulatory circuitry of root promeristems.

**What Makes Plant Cells Stop Dividing?**

Although the *cdc2aAt::cyclAt* construct provides a means to entice root cells into unscheduled mitoses, it does not necessarily bring us closer to the mechanism by which root cells normally withdraw from the cell cycle. In vertebrates, proliferative withdrawal is directly mediated by CDK inhibitors (CKIs; Harper and Elledge, 1996). As antagonistic subunits of CDK/cyclin complexes (Russo et al., 1996), these small proteins block the cell’s commitment to a new round of division by impeding G1/S passage. Thus, CKIs play a pivotal role in diverting cells in the G1 phase into one of two alternative nonmitotic pathways: apoptosis (programmed cell death) or differentiation.

Given that a CKI appears to be involved in mitotic arrest during maize endosperm development (Grafi and Larkins, 1995), it is not unreasonable to suspect that a related mechanism may operate in the transition between mixed proliferation/elongation and pure elongation that occurs in the root. If this is so, then how did ectopic *cyc1At* expression override the effect of the CKI? Because CKIs and cyclins do not compete for the same site on CDKs (Russo et al., 1996), a simple kinetic argument is not adequate. Perhaps CKI-arrested cells of the root were simply not part of the population that responded to *cdc2aAt::cyclAt* by dividing ectopically.

CKIs are not the only agents of proliferative arrest in eukaryotic cells. Plants carry an array of genes whose products antagonize cell reproduction within specific developmental contexts (Meyerowitz, 1997). These include *no apical meristem* (*nam*; Souer et al., 1996), *SUPERMAN* (*SUP*; Sakai et al., 1995), *CLV1*, and *CLV3* (Clark et al., 1993). In addition, the retinoblastoma gene product, Rb, plays an antiproliferative role in animal cells, and a plant cDNA encoding a member of the Rb family has been reported (Grafi et al., 1996). Thus, proliferative withdrawal along the root axis may involve a multistep process elicited by an upstream *nam/SUP/CLV*-like function that triggers Rb dephosphorylation (Weinberg, 1995) and blocks cyclin synthesis or CKI production (Wang et al., 1997).

**EXPANSION LEADS, MITOSIS FOLLOWS**

In the above-described transgenic Arabidopsis plants, new daughter cells dutifully expanded and the root elongated after cell division was artificially promoted by the ectopic expression of a mitotic cyclin. A plant may well achieve the same effect by promoting cell expansion directly. This prompts the question, if meristematic cells receive an “elongate!” signal, do they elongate and then divide, thereby obeying the “division serves elongation” corollary of the organismal theory? Or are they driven prematurely into the “elongation only” zone of the axis?

Although the inductive regulation of root cell fate has received some notable attention recently (Van den Berg et al., 1995; Schiefelbein et al., 1997, in this issue), the physiological and genetic control of root cell elongation per se has been relatively less studied (Baskin et al., 1995; Wu et al., 1996). At the other end of the plant, the dramatic stimulation of shoot elongation in rosette plants by gibberellin has been investigated for decades (reviewed in Kende and Zeevaart, 1997, in this issue). Yet, despite the impressive responses elicited in rosette plants, an alternative experimental system is beginning to yield some clues to more questions regarding the relationships between cell division and cell expansion in plant development.

**Elongation of Deepwater Rice Plants**

Deepwater rice plants (*Oryza sativa*) elongate dramatically when they are submerged (Figure 2A). Their stunning growth spurt results from the increased lengths that are attained by preexisting cells as well as those newly formed in the intercalary meristems (Kende, 1987). The signal transduction pathway that promotes this response is well defined at the upstream end. Reduced partial pressure of oxygen (due to flooding) at the youngest aerial internode promotes ethylene biosynthesis, which in turn raises that tissue’s sensitivity to the elongation-promoting effects of endogenous gibberellin. The adaptive justification for this response is that deepwater rice plants must extend themselves above rapidly rising floodwaters during annual monsoon rains to avoid succumbing to anoxia.

What is the initial investment plan implemented by the rice genome to achieve such rapid internodal growth? Acquire more units of cellular capital and then make them grow? Or stretch the organ’s existing cellular portfolio and issue stock splits secondarily, in accordance with our cellular corollary of the organismal theory? Internode elongation is first detected 40 min after gibberellin treatment of rice stems is initiated (Sauter and Kende, 1992), and a rise in the abundance of a transcript encoding a rice CDK1 homolog is detectable after 1 hr (we return to this intriguing datum below). During the next 2 hr, the levels of cyclin mRNA and histone H1 kinase activity (indicative of CDK activation) increase (Sauter et al., 1995). Two cardinal attributes of cell-cycle acceleration are seen much later. A decline in the number of G2 phase nuclei, which is indicative of the semisynchronous passage of meristem cells into mitosis, occurs at 4 hr, and DNA synthesis, representing entry into S phase, does not increase until 6 hr after the onset of organ elongation (Sauter and Kende, 1992). Although these data fall short of proving that expansion of meristematic cells was a necessary prerequisite for their subsequent mitotic activation, the reported order of events argues for cell division being a secondary
consequence of cell enlargement. Thus, when the rice plant urgently needs to generate a greater aerial presence, it calls on gibberellin to stimulate the elongation of existing cells; only later do mitoses follow.

Is Gibberellin a Mitogen?

An alternative to the above-mentioned “mitoses by default” in deepwater rice is that gibberellin may play a direct role in stimulating cell division in the intercalary meristem. After all, the increase in CDK1 transcript abundance is more or less coincident with the first signs of stem elongation (Sauter and Kende, 1992; Sauter et al., 1995). But during its half century in the spotlight of plant physiology, has gibberellin ever displayed any indication that it may function as a primary mitogen?

In the late 1950s, Sachs and Lang published a series of studies in which data were interpreted to support a mitogenic role for gibberellin (Sachs and Lang, 1958; Sachs et al., 1959a, 1959b). Every 24 hr, they injected the hormone into the bases of rosette leaves proximal to the shoot apices of *Hyoscyamus niger* and *Samolus parviflorus*. Apices were sectioned, and mitotic figures were mapped and enumerated 24 to 72 hr after the start of treatments (Figure 2B). The widely reproduced sketches from these articles show dramatic increases in the frequency and longitudinal distribution of mitotic figures during this time interval. However, two aspects of the observations reported in these studies argue against the authors’ conclusions regarding the mitogenicity of gibberellin.

First, Sachs and Lang’s data suggest that longitudinal extension of the meristem and its proximal flank had occurred before cell sizes were seen to decrease (Sachs et al., 1959b). Because full-size cells are not added to plant axes like bricks to a wall and because greater cell numbers were reported in these tissues after gibberellin treatment, apical cells must have elongated concomitant with division. Yet Sachs and Lang discounted the contribution of elongation, reporting that “the entire increase in stem length must be attributed to the formation of new cells” (Sachs et al., 1959b). Second, mitotic spindles in virtually all “gibberellin-induced” cell divisions were oriented longitudinally, that is, cells that divided as a consequence of gibberellin treatment invariably positioned their new cross-walls so as to bisect an extending longitudinal dimension. This behavior suggests a drive to maintain cell volumes under some maximum dimension in the face of hormonally induced axial extension.

The relationship between cell extension and cell partitioning in the axial growth of plants is dynamic and complex. Determinations of elemental expansion and partitioning rates can best be made in organs such as roots, in which growth can be treated experimentally as one dimensional and can be monitored continuously (Erickson and Sax, 1956a, 1956b; Green, 1976). On the other hand, activity of shoot apical meristems is more three dimensional, serially organogenic, hidden by expanding leaves, and visible only
by destructive, micrographic snapshots. Thus, the appropriate kinematic analyses were probably not possible in the Hyoscyamus and Samolus systems.

A Transition Zone in the Shoot Meristem?

Such reinterpretations notwithstanding, one aspect of the early studies that is worth noting is the cellular district in which the presumed gibberellin-induced mitoses were observed. This region was invariably described and diagrammed as “subapical” (Sachs et al., 1959a, 1959b), reminiscent of the root’s transition zone, described above. Thus, the shoot apex includes a zone immediately proximal to the meristem proper that has special properties with respect to cell growth and division. As in the analogous domain of the root, the distal portion of the shoot’s rib meristem (the “méristème médulaire” [Buvat, 1955]) straddles the border between proliferating and strictly elongating cell populations. In rosette plants, it is this transitional population that responds dramatically to gibberellin treatment by accelerating elongation and increasing mitotic activity. Similarly, in the deepwater rice system, 90% of elongation in the first 2 hr is attributable to the expansion of cells within the intercalary meristem itself (Sauter and Kende, 1992). The latter observation reflects the now widespread recognition that in plant organs, cell division and elongation do not occur in strictly segregated zones but are often concomitant processes.

Cell Size Homeostasis in Plants

The proposed relationship between the acceleration of expansion and the induction of mitosis in both deepwater rice and presumably rosette plant apices suggests the involvement of a regulatory system that ensures size homeostasis. Such systems have been elegantly demonstrated in yeasts (Fantes and Nurse, 1977; Hartwell and Unger, 1977). In these unicellular eukaryotes, the duration of a cell’s mitotic cycle is inversely proportional to its size at birth (i.e., emergence from mitosis and entry into G1 phase). Genetic control of size homeostasis in fission yeast is exercised by the wee1+ gene, mutations in which lead to premature entry into mitosis and abnormally small daughter cells. In subsequent cycles, wee1+ cells compensate for their diminutive birth mass by prolonging G1 phase until they have grown sufficiently to pass a size threshold checkpoint and gain entry into S phase (Murray and Hunt, 1993). A related observation has been made in plants, where the larger of two unequal daughter cells reenters M phase sooner than does its smaller sibling (Armstrong and Francis, 1985).

The wee1+ gene of fission yeast encodes a protein kinase that phosphorylates a tyrosine on CDK1, inhibiting mitotic passage until the requirements (sufficient size not being the only one) for M phase entry are met, whereupon WEE1 is inactivated and CDK1 is dephosphorylated by CDC25. Exactly how the parameter of cell size is perceived and transduced to WEE1 and CDC25 is not known. Functional homologs of wee1+ have been identified in vertebrates, but they have yet to be found in plants. Evidence has been presented for mitotic control by reversible tyrosine phosphorylation of CDKs in cultured plant cells (Zhang et al., 1996).

If in fact gibberellin’s beguiling mitogenicity arises from its ability to accelerate expansion of meristematic cells past a size homeostasis checkpoint, how does the hormone accomplish this task? The rate of plant cell expansion is a function of turgor pressure, yield threshold, and wall extensibility (Kutschera, 1991; see also Cosgrove, 1997, in this issue). It is believed that gibberellin affects, at minimum, the latter either by reorienting cortical microtubules in a transverse disposition or by selectively locking into place those (from a rapidly cycling pool) that already happen to be in that orientation (Shibaoka, 1991). Because the orientation of cellulose microfibrils in the cell wall tends to parallel that of underlying microtubules, then transverse microtubules (and perhaps other factors [Giddings and Staehelin, 1991]) would dictate transverse cellulose reinforcement, thereby limiting turgor-driven expansion to the longitudinal dimension.

Given such a causal chain linking gibberellin to anisotropic cell expansion, how might the process cross-talk with the cell’s mitotic engine? The nearly simultaneous rise in CDK1 transcript abundance and the first measurable extension of young internodes in deepwater rice suggest that lines of communication must be short indeed. It is almost as if meristematic cells knew that gibberellin would eventually push them through the putative size homeostasis checkpoint.

A reinterpretation of plant CDK cell biology offers one element of a possible mechanism here. Plant CDKs are associated with the preprophase band of microtubules in onion roots (Mineyuki et al., 1991) and maize leaves and roots (Colasanti et al., 1993). These observations have been interpreted to suggest that the CDK brands (presumably by phosphorylation) a substrate in the cortical cytoplasm with a mark to which the phragmoplast is somehow attracted during cell plate formation. But could the signal be traveling in the opposite direction? Perhaps CDKs are sequestered at the preprophase band in the cortical cytoplasm only to be released for mitotic activation duty when appropriately signaled by the wall or plasma membrane. In this way, the CDK-preprophase band interaction would represent a node in the regulatory network that links cell growth to cell partitioning.

An alternative interpretation of the CDK-preprophase band correlation is suggested by the recent report that microinjection of active CDKs into Tradescantia stamen hair cells results in disassembly of the cells’ preprophase bands (Hush et al., 1996). The low frequencies of CDK-preprophase band association reported in the maize and onion studies may therefore reflect the transient association of a kinase with one of its substrates rather than the more stable association of the CDK with other proteins in a multimeric complex.
SYNTHESIS AND PROSPECT

The organismal theory of plant development has recently gained support from novel phenotypes manifested at the supracellular level (Hemerly et al., 1995; Smith et al., 1996). If cell division serves merely to subdivide space in growing plants, then we must model a signal transduction chain that can link the passage of plant cells through a size threshold to the CDK-driven cell-cycle engine. The validity of the size threshold concept need not be compromised if, during normal development, cell division is ever found to proceed in the complete absence of cell expansion. Indeed, axillary buds, quiescent meristems of seeds, and lateral root anlagen of the root pericycle consist of dense populations of relatively unexpanded cells whose postmitotic fates must have been directed by different signals than those that guide the cellular products of active, vegetative meristems.

A size homeostasis paradigm carries a number of implications, a few of which are mentioned here. First, if such a rule guides cell division within apical meristems, then Arabidopsis cells driven to divide by ectopically expressing a mitotic cyclin would have illegally bypassed such a checkpoint. Therefore, one might expect to find mitotic catastrophes and perhaps reduced fertility in such plants (Campbell et al., 1995). If this is not seen, then how did these plants accommodate such a regulatory infraction? Was cell elongation stimulated as well? If so, by what signal transduction pathway?

Second, if the drive for size homeostasis contributes to the timing of cell division in plant meristems, then the existence of the mitotically quiescent center of the root apex might be explained simply by the cells there being hemmed in, unable to elongate sufficiently to bypass the size threshold for mitosis.

Third, such a simple rule would need to be governed secondarily to accommodate the diversity of developmental contexts in which cell division occurs in plants. The growing inventory of genetic and biochemical functions that limit cell division in plants (i.e., CKls, nam, SUP, CLV1, CLV3, and Rb [Meyerowitz, 1997]) suggests that the default behavior of expanding cells is to divide and that the marvelous diversity observed in plant morphogenesis is, at least in part, achieved by selectively applying the brakes to cell proliferation. Thus, morphogenetic (formative) cell divisions may result from these limiting genetic controls being superimposed over a size homeostasis mechanism that otherwise operates relatively independently in proliferative mitoses.

If cell expansion (gibberellin stimulated or otherwise) drives plant cells past a cell-cycle checkpoint and cyclin elevation alone can promote cell division in roots, then we have two points between which to sketch a signal transduction pathway linking cell expansion with cell division. Such a pathway would form part of the network that shifts cells between two essentially cytoskeletal states: growth (spindles, phragmoplasts, and traverse cortical microtubules) and maturation (oblique and longitudinal microtubules). A convergence of genetics, molecular biology, and cell biology has brought us this far, and we can look forward to new connections as these fields close in on mechanisms in the future.

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