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Development of the abscission zone

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Abstract

Purpose of review: This review focuses on the genetic mechanisms that underlie differentiation of tissues specialised for cell separation in three well-studied models: the abscission zone (AZ) of the flower and fruit pedicel, floral organ AZ, and the related dehiscence zone of the fruit.

Findings: Nearly all research on abscission has concentrated on the activation stage, when cells of the AZ become competent to respond to abscission-promoting signals, or the separation stage, characterised by loss of cell-cell adhesion. There have been relatively few studies on mechanisms responsible for the initial differentiation of the AZ during organ development. The comparable architectures of AZs in various developmental circumstances suggest patterning by analogous mechanisms and involvement of similar activities of genes.

Directions for future research: The control of postharvest losses through abscission at the level of AZ development deserves more attention. A mechanistic model for AZ development will require the identification of additional genes, through genetic or molecular approaches, as well as the characterisation of interactions between these genes.

Keywords: abscission; dehiscence

Abbreviation

AZ Abscission Zone

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Introduction

Postharvest control of abscission in fruits, vegetables and flowers has focused almost exclusively on repressing activation of the abscission zone (AZ) and the accompanying cell-cell separation within the abscission layers, either through specific storage regimes or use of plant growth regulators [1]. However, abscission ultimately relies on the cellular architecture of the AZ, set up by patterning events during organ formation and maturation. Although the sequence of morphological, cytological and biochemical events that occur upon activation of the AZ have been studied extensively, the developmental processes leading to the differentiation of the AZ have not. These developmental mechanisms are obvious and attractive targets for abscission control. In fact, domestication of major human food crops including wheat, rice and maize is thought to have involved selection for aberrant AZ development [2–4**, 5**]. Improvement of other crops through manipulation of AZ development similarly should be feasible through breeding or genetic engineering. However, to date, the genetic mechanisms that drive these processes remain poorly understood. This review will concentrate on the genes and genetic mechanisms influencing

development in the three most thoroughly studied models – the AZ of the flower/fruit pedicel, AZ of the leaf/floral organs, and the analogous dehiscence zone of some dry fruits.

Development of the abscission layers

The presence of a subtending AZ is a common feature of many diverse types of plant organs and parts [1], suggesting that it reflects the output of a common patterning mechanism adapted for specific developmental events. The rationale behind this interpretation may be most obvious when comparing leaf AZs to floral organ AZs such as sepals, petals and stamens. Botanically, these floral parts are recognised to be elaborations of leaves [6]. Mechanisms that underlie leaf AZ development may be more difficult to extend to AZs that subtend flowers and fruits, because the latter are considered highly modified shoots rather than leaves. Thus, morphology may be a more appropriate starting point for potential functional comparisons between botanically distinct organs.

Many, but not all, AZs are morphologically distinct prior to the onset of abscission. Of those that are, AZs in very early stages of development are invariably described as tightly localised regions of relatively small cells lacking large vacuoles and any aspect of differentiation. These are arranged transversely to the axis of the pedicel (flower or fruit), petiole (leaf), or long axis of the floral organs [1, 7, 8]. Specialised cell types that may otherwise be distributed continuously along the axis of the organ, such as the sclerenchyma fibres that provide mechanical support, are typically absent across this region. Vasculature is continuous through the AZ, but may undergo a localised disruption in arrangement, and tracheids are less lignified [1].

The apparent absence of differentiated cell types within the AZ, other than those directly associated with the vasculature, suggests that cell growth and differentiation is arrested at an early stage. Thus, the small and dense cells typically observed in AZs might simply be remnants of the mitotic proliferation that generated the organ, having remained relatively unchanged as surrounding cells expanded and differentiated. The formation of rudimentary vasculature, which is seen very early in organ development [9], may precede such a developmental halt. This idea is consistent with the morphological distinction of this region at very early stages of development observed in many species, and the lack of the highly ‘layered’ appearance that is manifested in the AZ at more advanced stages [10].

If the AZ does represent lack of differentiation, then the question arises of how the cells in this region could escape the developmental programming that patterns the surrounding tissues. A common theme in developmental biology, exemplified by the segmentation of the fruit fly embryo along the proximal-distal axis, is the specification of specific cell types as a response to morphogens. Morphogens are developmentally important molecules that act in a concentration-dependent and combinatorial manner to regulate expression

of various subsets of genes. In this way, the AZ can be conceptualised as defining a position where proximal-distal patterning signals are absent or are ineffective to promote differentiation of specific cell types. Studies of cell proliferation and differentiation during leaf initiation and early development at the shoot apical meristem may also provide clues as to how AZ differentiation could be arrested [11, 12]. Cells in the apical meristem are prevented from differentiating through the activity of the KNOX family of transcription factors, which, among other functions, promote cytokinin signalling. Initiation of leaf primordia, which occurs in a regular pattern (phylotaxy), involves the localised accumulation of auxin at a defined point on the meristem. Auxin directly or indirectly may suppress the expression of KNOX genes, allowing development to begin. Following initiation, interaction between auxin and re-established KNOX gene expression contributes to lateral patterning by repressing differentiation in defined regions along the leaf lamina [13]. Extending these observations to the AZ, it is tempting to speculate that the apparent lack of development results from either persistence of KNOX expression in this region, or its reestablishment following organ initiation. This scenario would predict that KNOX genes, their downstream effectors (genes involved in cytokinin signalling) and upstream regulators (possibly, genes involved in auxin signalling) would affect AZ development. A lack of such predicted involvement could reflect that these genes have a broader role in organ initiation, an event that precedes AZ formation.

Development of the protective layers

A second essential feature of the AZ that must precede cell separation is the development of the protective layers. Organ abscission creates a situation whereby formerly internal tissues are exposed directly to the external environment. The new exterior surface must adopt the important functions of the epidermis, including providing a barrier to water loss and pathogen infection. The protective layer forms basal to the separation layers and is continuous with the periderm of the stem [1]. In most cases observed, the development of the protective layer involves transverse cell division, which may produce daughter cells with epidermal identity. Transverse cell division to generate the protective layers may precede activation of the AZ, and might define the ‘layered’ appearance typical of AZs [10]. The number of final layers is highly variable, ranging from as little as two (at the base of the Arabidopsis petal) to 50 or more (in the leaf rachis of the elderberry, *Sambucus nigra*) [14]. Where cell division is not observed, the exposed tissues may undergo a program of transdifferentiation, or a reprogramming of function. In either case, cell wall modification can involve the deposition of lignin and suberin into the walls of the nascent epidermal cells, as well as modification of the wall through covalent cross linking of proteins associated with peroxidase activities [15]. Thus, development of the protective layers is expected to be associated with the expression of genes involved in pathogen defence and cell wall modification. In fact, in the few studies that have been attempted so far on gene expres-

sion in the AZ, these classes of gene are strongly represented in the gene sets reported as abscission-related [16, 17]. Cell-wall modifying genes previously associated with abscission, and presumed to act in cell separation, may have an alternative and less obvious activity in remodelling the walls of the protective layers. In support of this, studies in the bean leaf showed that an increase in endo- β -1,4-glucanase activity occurred in a domain that extended beyond the separation layers [18].

Genetic control of AZ development

Development of the pedicel AZ in tomato

There have been very relatively few genes identified that have an unambiguous role in the development of the AZ. Probably the best known of these few genes are *Jointless pedicel* (*J*) and *J-2*, which promote the formation of the flower/fruit pedicel AZ in tomato [19, 20**]. Most flowers and fruits exhibit AZs at the junction between the pedicel and the main shoot, and often between the pedicel and the calyx. Typical tomato varieties exhibit a swollen ‘joint’ on the pedicel at a point midway between the main shoot and calyx; this structure encompasses the abscission layers and is the point at which aborted flowers, or ripened fruit, separate from the plant. In plants homozygous for *j* or *j-2*, pedicels lack morphological evidence of a complete joint. This trait has obvious agronomic value, as fruits remain attached to the plant upon ripening, minimising preharvest fruit drop and allowing for more efficient harvesting, especially by mechanical methods. In addition, when fruits are harvested, the calyx and pedicel segments remain attached to the plant, eliminating both pathogen invasion into the calyx region and damage to neighbouring fruit during transport and storage. Indeed, alleles of both *j* and *j-2* have been widely utilised to develop varieties optimised for the processing industry [21].

The *j* allele was first recognised in a domestic cultivar, Rouge Naine Hative [19]. This gene encodes a presumed transcription factor belonging to the MADS-domain class [22], which has been greatly expanded in plants and includes other members with such diverse roles as timing of flowering, floral organ identity and fruit ripening [23]. The commitment of this class of protein to important developmental events is consistent with the striking phenotypic effect conditioned by *j*. However, recent work in tomato suggests that the influence of *J* on AZ formation may be an unanticipated, indirect effect of a function in shoot/inflorescence architecture [24]. Loss of *J* converts the typical sympodial shoots of tomato to indeterminate shoots with partial inflorescence identity [25]. Additional evidence for an indirect effect is the observation that *J* is not expressed in developing AZs, but rather is active in the shoot, inflorescence and floral meristems [24]. *J* affects development of the AZ in a cell non-autonomous manner [26], but whether this could explain an indirect effect on AZ formation is unclear. Interestingly, joints were formed at various positions along the pedicel in transgenic plants with altered expression of *J*, suggesting that correct dosage of the *J* protein is required to correctly position the AZ [22].

The *J-2* gene was discovered through observations of an accession of a wild species, described as *Lycopersicon pimpinellifolium*, from the Galapagos Islands. The studied *j-2* allele was incompletely recessive in F1 hybrids with a domestic variety, with joints forming abnormally [20**]. Similar to *j*, *j-2* has pleiotropic effects associated with shoot architecture: *j-2* plants often produce bifurcate inflorescences producing an abnormally large number of flowers [27]. Additionally, *j-2* is associated with conversion of sepals to leaf-like structures, resulting in a ‘leafy’ calyx. Genetic mapping has identified a transcription-related gene as a candidate for *J-2*. This gene, *CPL1* (*C-TERMINAL DOMAIN PHOSPHATASE-LIKE 1*), encodes a protein closely related to budding yeast Fcp1, a phosphatase that targets phosphorylated serines within the heptapeptide repeats in the carboxyl-terminal domain of the largest subunit of RNA polymerase II (Pol II) [28]. The activity of Fcp1 is believed to be essential for recycling Pol II at the end of the transcription cycle, thus generating the non-phosphorylated form of Pol II required for recruitment and initiation [29]. *CPL1* is a member of a large family of *FCPI*-related genes in tomato. Various members of this gene family in tomato and other plants may be expressed in unique tissues or may target unique subsets of genes, possibly explaining how disruption of a core transcriptional regulator results in relatively mild phenotypic effects. Further investigation on *J-2* awaits confirmation that *J-2* is *CPL1*.

Other genes that affect development of the abscission joint in tomato are *MACROCALYX* (*MC*) and *BUSHY* (*BU*) [30]. In *mc* plants, a functional but structurally aberrant joint is formed. The *mc* allele also conditions pleiotropic effects similar to those seen in *j-2*, including inflorescence indeterminacy and a leafy calyx [20**, 31], suggesting a close functional relationship between *J-2* and *MC*. Like *J*, the *MC* gene encodes a MADS-domain transcription factor, but of a subclass that is not closely related with *J*. In plants carrying the *bu* allele, a normal joint forms, but at a more apical position near the calyx [30]. The aberrant positioning of the joint reported in transgenic plants expressing abnormal levels of *J* suggests a functional link between *BU* and *J*. Identification of the *BU* gene has not yet been reported.

Whether the mechanism of AZ differentiation involving these genes is conserved among plants remains unclear. Arabidopsis, the most popular model for understanding plant development, does not exhibit an AZ on the pedicel of the flower or fruit. The closest homologs of *J* in Arabidopsis are *AGL24* and *SVP* [22], two MADS-box genes involved in flowering timing and meristem identity [32]. Thus, an answer to this question will await identification of genes involved in AZ formation in other species. In this respect, the domestic apple represents an attractive opportunity, considering its genetic diversity, genomic resources, domestication of various flowering and fruiting habits, and extreme variability in fruit abscission [33].

Development of the leaf and floral organ AZs

The developmental genetics of leaf abscission, arguably the most ubiquitous abscission-related phenomenon, remains largely untouched, likely because the most accessible plant genetic models (*Arabidopsis* and maize) do not show leaf abscission. In contrast, the abscission of floral organs has been extensively characterised, mostly through genetic screens in *Arabidopsis* for loss of petal abscission. Similar to that seen in the leaf, which abscises from the main shoot at the point of attachment of the petiole, the AZ of floral organs is found at the point of attachment to the receptacle [1].

Studies in the past several years have identified both ethylene-dependent and ethylene-independent mechanisms of floral organ abscission in *Arabidopsis* [34, 35]. The role of ethylene has been best documented as promoting expression of genes presumed to function in dissolution of the cell wall [36]. These studies suggest ethylene acts at a relatively late stage of the abscission process, raising the possibility that ethylene-independent mechanisms could be involved in the establishment of an AZ that is responsive to the ethylene signal, either through mediating the correct architecture of the AZ itself, or by facilitating the efficacy of the ethylene-signalling pathway. Several genes have been identified with apparent ethylene-independent roles. However, most appear to function late in abscission, and these are discussed in another chapter in this volume [35].

The only genes identified so far with clear roles in development of the floral organ AZ are *BLADE-ON-PETIOLE1* (*BOP1*) and *BOP2*. Plants dysfunctional for both genes lack morphological evidence of the AZ [37–39]. *Bop1/2* plants also lacked evidence of the vestigial AZs that form at the point of attachment of the cauline leaves and pedicels to the inflorescence [39, 40], suggesting a more expansive role in AZ development. Both genes are closely related to *NPRI*, a gene that functions in systemic acquired resistance by activating pathogenesis-responsive genes. Consistent with a function in development, at least *BOP1* is expressed at the base of developing sepals, petals and stamens [39]. *BOP1/2* also act together to repress leaf blade formation along the petiole, and to suppress development of floral bracts (which are normally absent in *Arabidopsis*) [37, 38]. Thus, these genes are not committed to AZ specification but appear to have a wider role in repressing differentiation.

Development of the dehiscence zone

In many non-fleshy fruits, seed dispersal is enabled upon maturity by the splitting open of the fruit, a process termed fruit dehiscence. While technically not true abscission, fruit dehiscence nevertheless provides interesting insight into programmed cell separation relevant to flower/fruit and floral organ abscission. Many clear parallels can be drawn. Indeed, Addicott [1] suggests abscission and dehiscence can be viewed as “two aspects of the same basic process”. Ultimately, both processes are dependent on tissue breakdown, associated with the expression of an overlapping set of cell

wall-modifying genes. In both processes, this occurs within a tightly restricted zone of cells that is specified at predetermined positions during organ development. In addition, both the floral organ AZ and dehiscence zone exhibit juxtaposition of ‘separation layer’ cells with layers of cells that show extensive modification of the cell walls [1]. In contrast to the floral organ abscission AZ, however, there is substantial information on the genetics of the early development of the dehiscence zone, mostly from studies in *Arabidopsis*. Such work has huge agricultural implications because in many important crop plants, such as canola or soybean, premature fruit dehiscence — called pod shatter — causes considerable yield losses.

The fruit of *Arabidopsis* is a silique, composed of two semi-circular walls, called valves, which are derived from the congenitally fused carpels of the flower, and replum, which separates the valves [41]. The valves exhibit an outer epidermis, called the exocarp, several layers of parenchymous mesocarp, and two layers of internal epidermis called endocarp (en) a and en b. Along the length of the fruit, between the valves and on either side of the replum, the so-called valve margin or dehiscence zone is differentiated. A specialised separation layer forms on the replum side of the valve margin, while a layer of lignified cells forms on the valve side of the margin. This lignified layer is continuous with an adjacent layer of lignified cells present in the valves, the en b layer. When the valve tissues become desiccated and shrink late in development, the lignified tissue stays rigid, and spring-like tension allows the silique to pop open following tissue breakdown in the separation layer [41].

Formation of the dehiscence zone (valve margin) in the developing fruit is accomplished through a mechanism involving a transcription factor called JAGGED (*JAG*), acting with the organ-polarity transcription factors FILAMENTOUS FLOWER (*FIL*) and YABBY3 (*YAB3*) [42]. In mutants dysfunctional for all of these genes, abnormal, indehiscent fruit develop that lack valve margins [42]. *JAG*, *FIL* and *YAB3* together promote the expression of genes that help to define the organisation of the valve-replum boundary: *SHATTER-PROOF* (*SHP*) 1 and 2, and *FRUITFULL* (*FUL*).

SHP1/2 redundantly promote valve margin development [43]. In *shp1/2* double mutants, the valve margin is completely absent, including both the separation layer and the layer of lignified cells. The vascular bundle, which tracks along the length of the replum, is largely unaffected [43], indicating that there is no gross morphological alteration of the fruit structure that might drive this change indirectly. Thus, *SHP1* and *SHP2* seem to have a fairly specific function in development of the valve margin. Both genes are members of the large class of MADS-domain presumed transcription factors, suggesting that they act in a regulatory capacity for the activity of other genes. In contrast to *SHP1/2*, *FUL*, also a MADS-box gene, promotes valve development [44]. In *ful* mutants, the valves are reduced and the valve marginal tis-

sues are expanded; this usually results in the fruits rupturing prematurely as the seeds develop. *FUL* is expressed only in valves, and together these data suggest that *FUL* promotes valve development while negatively regulating valve margin development [45]. Consistent with this, constitutive expression of *FUL* represses the formation of the valve marginal tissue, converting the entire fruit surface to valve tissue [46].

The *REPLUMLESS (RPL)* gene encodes a homeodomain protein required for proper development of the replum [46]. In plants lacking *RPL* activity, valve margin cells occupy the domain normally occupied by the replum. This suggests that *RPL* normally represses the expression of valve margin cell identity and the genes that determine that identity [46]. Therefore, although *FUL* and *RPL* have similar function as repressors of valve margin development, they act in nonoverlapping domains—*FUL* in the valve and *RPL* in the replum.

The *INDEHISCENT (IND)* gene is functionally equivalent to *SHPI/2*, with loss of function totally eliminating valve margins including both the separation layer cells and the lignified cell layer [47]. The epistatic relationship between *SHPI/2* and *IND* isn't clear, and data suggest they could work together or in parallel but interdependent pathways. *IND* encodes a bHLH-type transcription factor [47]. Another bHLH transcription factor acting in valve margin development is *ALCATRAZ (ALC)*. As the gene name implies, fruit from *alc* mutant plants is indehiscent [48]. However, in contrast to *shp* or *ind* mutants, which lack the entire valve margin, in *alc* mutants the separation layer is absent but the lignified cell layer is still differentiated. Thus, *ALC* is involved in only a subset of functions carried out by the *SHP* and *IND* genes; this places *ALC* genetically downstream from these genes. In accordance with a specific function in dehiscence, *ALC* gene expression is confined to the dehiscence zone [48].

The model developed from these and other studies is that both *RPL* and *FUL* are required to limit *SHPI/2*, *IND* and *ALC* expression to a narrow strip of cells so that the dehiscence zone differentiates precisely at the valve/replum boundary. The studies suggest that dehiscence zone development is the end result of a cascade of transcriptional regulators, from the initial patterning activities of *FIL* and *YAB3*, to the separation layer specification function of *ALC*.

Conclusion and perspectives

Obviously, much remains to be learned about developmental mechanisms of AZ formation if these are to be easily manipulated as a point for control of postharvest losses. The pedicel AZ in tomato would seem to be an attractive system for further analysis of the genetics of AZ development, given its morphological simplicity, and defined, exposed position. The genetic or molecular interactions between the genes identified so far have not been well characterised, and this offers exciting prospects for further work. The findings that *J*, *MC*, and possibly *J-2* encode transcriptional regulators

suggests the opportunity to identify genes that are direct targets and that likely play more specific roles in AZ specification.

The mutant screens in Arabidopsis that have been so productive for the identification of genes functioning at the activation stage of floral organ abscission should continue to discover genes such as *BOPI/2* involved in differentiation. Such genes might also be identified through molecular methods. Transcriptional profiling provides an entry point for the characterisation of gene regulation during AZ development [16, 49]. Next-generation sequencing techniques now permit this type of analysis for traditional abscission models, such as bean or *S. nigra*, that are not currently supported by genomics resources.

Finally, opportunities exist even in the area of fruit dehiscence, where more knowledge has accumulated. While numerous regulatory genes have been identified, their downstream targets remain mostly unknown. Based on its narrow and committed role to abscission layer development, the *ALC* gene is a particularly attractive entry point for downstream molecular events in the abscission layers.

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