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Forbidden fruit: dominance relationships and the control of shoot architecture

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ABSTRACT

Plants continually integrate environmental information to make decisions about their development. Correlative controls, in which one part of the plant regulates the growth of another, form an important class of regulatory mechanism, but their study has been neglected and their molecular basis remains unclear. In this review, we examine the role of negative correlative controls or 'dominance' phenomena in the regulation of shoot architecture. Apical dominance, in which actively growing shoot branches inhibit the growth of other branches, is perhaps the most famous example of this. We discuss the recent progress made in understanding the mechanistic basis for apical dominance, and three plausible models for shoot branching control. We then use the apical dominance paradigm to explore other dominance phenomena, including seed-seed inhibition (carpic dominance), seed-to-meristem inhibition, and the control of maternal senescence by seeds. We propose that apical and carpic dominance may share a common mechanistic basis rooted in auxin transport canalization. Conversely, we conclude that seed-to-meristem inhibition and seed-driven senescence may not be 'true' correlative controls, but rather more complex phenomena in which seed-set plays a permissive rather than instructive role. Overall, we attempt to develop a coherent framework for understanding the developmental and regulatory mechanisms that control shoot architecture, and provide new insights into the end of flowering, fruiting and growth.

KEYWORDS

Shoot architecture, shoot branching, apical dominance, auxin, canalization, strigolactones, correlative controls, senescence, seed set

1. Introduction: decision-making in plants

One of the key ways in which plants respond to the environment is through continuous, modular and plastic post-embryonic development, which allows the plant to match its growth to prevailing conditions. We have come to understand the environmental inputs and developmental outputs of these response mechanisms in great detail, but the process by which inputs are integrated and translated into outputs is less well understood, and in some instances positively mysterious. We use the term 'decision-making' to describe this general process. Although 'decision' might seem anthropomorphic, we believe that this term ('a choice or judgement') is entirely justified. Plants do not inertly wait for challenges, they are proactive rather than reactive regarding their development; they plan their life-cycles with impressive precision (Figure 1.1).

1.1 What decisions do plants make, and why?

In accordance with standard evolutionary theory, traits that maximise the fitness of individuals will tend to be selected for; we should therefore expect that decision-making processes in plants are geared to maximise fitness. However, this does not imply that plant decisions are geared to maximizing reproduction. Despite the ingenious mechanisms used by some plants to promote long distance seed (or spore) dispersal, there is a very strong probability that offspring will grow close to the parental plant. Thus a plant which strips its locality of nutrients to maximise its own reproduction is likely to directly penalize the growth of its offspring. Similarly, producing an excessively large number of offspring is likely to cause unnecessary intraspecific competition for resources in the next generation. These inter- and intra-generational considerations mean that is unlikely that plants actively maximise either growth or reproduction relative to available resources. Rather, plant decisions are shaped to optimise growth and reproduction in a manner that is sustainable for the species over evolutionary timescales.

Fundamentally, development in plants comes down to making decisions on the quantity, type, size and location of organs that should be produced. Or, to epitomise it further; "How many organs and which ones?" Across all land plants, and both shoot-like and root-like systems, the same basic choices are available; to invest in new organs, to further invest in existing organs, or to divest from existing organs. In this review, we will focus on decision making in the shoot systems of angiosperms (flowering plants), primarily because our knowledge of regulatory mechanisms is much more developed in these species than in non-flowering plants.

Although shoot morphology amongst flowering plants is incredibly diverse, the basic decision-making processes that determine shoot architecture are relatively few in number:

1) Organogenesis: active shoot axes can initiate new lateral organs (leaves, etc.) from the shoot apical meristem (SAM) at a greater or lesser rate, and/or with altered phyllotaxis.

2) Organ growth: lateral organs can grow to different sizes and shapes depending on environmental and developmental cues.

3) Shoot growth: shoot axes can elongate to a greater or lesser extent, or arrest altogether.

4) Shoot branching: new shoot axes can initiate, and either enter dormancy, or activate.

5) Reproduction: flowers can initiate in greater or lesser numbers, be fertilised, and produce greater or lesser numbers of fruit and seed.

6) Senescence: any of these structures can undergo senescence and/or abscission

1.2 What factors influence shoot architectural decisions?

To understand the decisions that plants make, it is necessary to understand the factors that potentially impinge on shoot architecture. Most obviously, this includes internal factors such as developmental status; for instance, the floral transition usually triggers wholesale changes in shoot architecture. It also obviously includes the availability of sufficient resources to grow. Here, we will use 'resources' to broadly encapsulate light, photosynthate, and mineral nutrients whether external or internal to the plant. We will use 'assimilates' to describe those resources already acquired by the plant, and 'nutrients' to describe resources potentially available to the plant, but as yet unassimilated.

There are also a suite of other, less obvious factors that affect shoot architecture. For instance, the mutual interdependency of shoot and root systems means that there is a strong need to balance shoot development with root development. Furthermore, vegetative shoot growth must also be balanced against reproductive growth; the plant must not invest too much or too little in vegetative growth that it cannot support an optimal reproductive effort. Plants must also bet-hedge, and reserve resources to replace or repair damaged organs, or to mitigate against changes in conditions. It is far better to successfully produce some seed, than to over-commit and produce none. Plants also have to make trade-offs; defense versus growth being a commonly cited example (Karasov et al, 2017). Balance, bet-hedging and trade-offs all contribute to the non-maximization of plant growth; maximizing growth is simply a poor strategy that does not maximise long-term fitness. Plants also likely face temporal constraints on their growth; for instance, to take advantage of pollinator availability, or to avoid poor growing conditions. This may require an increased rate of development that requires other trade-offs to be made. For instance, production of branches allows for an increased rate of organ production, but makes development less efficient in terms of resources invested in the organs

themselves. Finally, hierarchical developmental constraints also play a key role in determining shoot architecture; for instance, in order to produce flowers, a plant must first produce branches.

1.3 Information processing: long distance signals and dominance phenomena

Plants must thus detect and integrate a wide range of environmental and developmental stimuli, and use this information to plan and balance their development, both in the present and in the future. To solve these equations would be a difficult enough challenge for a human, even backed up with considerable computing power, and yet plants do so without any sort of central information processing system. While it can be debated whether plants are 'intelligent', they are certainly very good at mathematics, and can consistently make good decisions, sometimes for thousands of years in a row. So how do plants manage these astounding feats of calculation?

In our opinion, there are three crucial components (Figure 1.3). Firstly, plants use highly reductive long-distance signalling mechanisms to distribute environmental information across the plant body. A very small number of well-defined phytohormones seem to account for much environment-to-development signalling, with auxin, cytokinins and strigolactones the most ancient and widespread of these. These signals might act as 'consolidated information', whereby complex environmental inputs are translated into generic signals that do not carry specific instructions, but convey simple information (Bennett & Leyser, 2014).

Secondly, developmental decision-making is partially devolved to each individual organ; this is a form of 'distributed computing' (Leyser, 2011). Each organ thus integrates local environmental information together with systemic long-distance signals to decide on an appropriate growth response. For instance, a given root meristem in a nutrient poor 'patch' may cease growing if soil conditions are generally good, or may grow strongly if soil conditions are uniformly bad (Li et al, 2014). Thirdly, for many growth responses, plants possess mechanisms that coordinate growth among organs; so that the 'growth potential' of a given set of environmental conditions is focused into an appropriate number of organs, rather than divided among all possible organs. These coordinating mechanisms undoubtedly exist, but remain enigmatic – how can a plant make such coordinated decisions without a centralised processing system?

So-called 'correlative controls' are one major class of these coordinating mechanisms, in which one part of the plant exerts control over the development of another, either positively or negatively (Nooden, 1984). Perhaps the best known example of this is 'apical dominance', in which actively growing shoot branches inhibit the activity of other shoot branches (Went & Thimann, 1937). Other

negative correlative controls include the ability of seeds to inhibit further seed production, and the ability of seed to promote senescence of maternal tissues. The main focus of this review will be to examine progress in understanding the mechanisms behind these 'dominance' phenomena, and their role in coordinating development across the angiosperm shoot system.

1.4 Sources, sinks and signalling

Traditional explanations for dominance phenomena tended to hinge on organ nutrition and source-sink relationships. Thus, it has been proposed that plants make as many organs as resources allow, and that organs become dominant because they are stronger sinks for assimilates, and outcompete other organs for their import (Molisch, 1929). Practically speaking, it is difficult to absolutely prove or disprove this 'nutrient diversion' hypothesis, because assimilate flux does tend to correlate with dominance patterns. However, this does not mean that the assimilate flux causes dominance. Purely nutritional mechanisms would result in competition between organs only occurring where assimilates are limiting, whereas active signalling would involve inhibition of developing organs when resources are not limiting. Where evidence is available, this does seem to be the case; dominance precedes competition for assimilates, or occurs without assimilates being a limiting factor (Nooden, 1984; Bangerth, 1989).

Overall, simplistic nutritional models imply that the plant is largely a passive actor, and simply grows as much as possible for the given levels of assimilate. This clearly does not reflect the plants ability to intricately plan and execute developmental programs. Rather, it is clear that active signalling is used to determine which organs grow, and to determine how resources are allocated. Two major forms of active signalling can be distinguished. Firstly, assimilates themselves can trigger active signalling events, independently of their nutritional role. The level of photosynthetic assimilate (hereafter generically referred to as 'sugar') available to the plant is perceived through several signalling pathways, including TARGET OF RAPAMYCIN (TOR) kinase and Snf1-RELATED KINASE1 (SnRK1) (Lastdrager et al, 2014). Similarly, assimilated nitrate triggers major transcriptional changes in both root and shoot systems through active signalling (Forde, 2014; Medici & Krouk, 2014; Krapp et al, 2014). Modern models of source-sink regulation of plant growth thus include these active signalling components (e.g. Yu et al, 2015). The previously discussed hormonal signals act as a second level of active signalling, in many cases acting as proxy for resource availability both internal and external to the plant.

In the case of apical dominance, there is overwhelming evidence that dominance is caused by active signalling, principally driven by long-distance hormonal signals, and not simply as a result of

assimilate availability (reviewed in Domagalska & Leyser, 2011). Furthermore, genetic analysis has identified numerous mutants which make more branches, biomass or seed than normal with the same set of resources, showing that resources are not necessarily a limiting factor per se (de Freitas Lima et al, 2017). Another problem with simple nutritional models is that they assume decision making is driven solely by assimilates, rather than all resources (i.e. both internal assimilates and as-yet-unassimilated resources in the environment). Again, there is clear evidence that plants integrate external resource availability into developmental decisions (e.g. Guan et al, 2017; Franklin et al, 2014). Active source-sink nutrient signaling undoubtedly contributes to the establishment or maintenance of dominance relationships, but in the following we will largely focus on the evidence for dominance phenomena as hormonally regulated processes.

1.5 Life strategies, developmental transitions and shoot architecture

Life-history strategies vary considerably among flowering plants, which can be divided into the broad categories of annuals, whose life-cycle is completed within a single year, and perennials, which live for multiple years. Perennial plants can be further divided into monocarps, which reproduce only once at the climax of the lifecycle, and polycarps, which undergo repeated flowering during the lifecycle; annual plants are by definition monocarpic. Within these broad categories, the timing of key developmental transitions (e.g. germination, entry and exit from the reproductive phase, winter dormancy) varies greatly between different species in order to take advantage of, or to mitigate against environmental conditions. It is important to bear in mind that these different strategies and developmental transitions also have considerable consequences for the decisions made in the control of shoot architecture, and particularly for the expression of dominance phenomena. We will use comparisons between annual and perennial, and between monocarpic and polycarpic plants to help build a coherent picture of this area.

2. Apical dominance and the control of shoot branching

2.1 The theory of shoot branching

As perhaps the most visually obvious aspect of shoot architecture in many plants, understanding how shoot branching is regulated has been long-standing question in the field. Branches allow plants to produce more tissue in a given time frame. In the case of vegetative branches, this both allows greater leaf production and greater exploration of space to increase light harvesting. In the case of reproductive branches (inflorescences) it more simply allows increased numbers of flowers to be produced. However, branches come at a cost of the resources used to produce the branch itself, and branches (especially inflorescences) thus represent a trade-off between allowing faster production of organs and producing organs in a less resource-efficient way. We should therefore expect that, for a

given set of resources, plants produce the maximum number of lateral organs from the minimum number of branches. That is to say, branching is an inherently conservative process.

This undoubtedly underlies the predominantly binary behavior of axillary meristems (AMs), which tend to either become completely active or remain dormant as an 'axillary bud'; it is more resource efficient to have one fully developed branch than several short ones. Axillary buds can subsequently activate if conditions change, and conversely actively growing branches can also be re-inhibited (Morris, 1977). The mechanisms that regulate branching must therefore 'calculate' how many buds it is optimal to activate for a given set of resources. Plants are astonishingly consistent in their decision making in this respect; for instance, plants grown in the same soil environment tend to make a similar number of branches, irrespective of quite significant changes in life history or light conditions (Figure 2.1). Furthermore, these regulatory mechanisms must also determine which buds to activate for optimum productivity, especially in the case of light-harvesting vegetative branches. While there is a default pattern of bud activation in many species, this can (and should) be altered in response to local information. If, for instance, a bud is shaded, it is unlikely to make an efficient contribution to light harvesting. Shoot branching must therefore be regulated by a system in which bud outgrowth is globally coordinated in response to both systemic and local stimuli.

2.2 Resource-related signals and shoot branching

Unsurprisingly, given the resources required to build a new branch, many of the key signals that regulate branching are associated with the availability (actual or potential) of mineral nutrients and photosynthetic carbon. For instance, nitrogen (N) and phosphorous (P) availability in the soil, as perceived by the roots, strongly promotes shoot branching (Troughton, 1977; Cline 1997; de Jong et al, 2014). Root-shoot signalling is thus a key element in shoot branching control, and cytokinins (CKs) and strigolactones (SLs), have been implicated as signals that couple branching to resource availability in the soil. CK synthesis in the roots is upregulated by N-availability, and CK is transported into the shoots, where it acts to promote bud outgrowth (Takei et al, 2002; Muller et al, 2015). CK treatment promotes outgrowth of buds in both excised nodal segments and whole plants (Wickson & Thimann, 1958; Sachs & Thimann, 1965; Chatfield et al, 2000), while CK synthesis and signalling mutants fail to increase their branching in high N conditions (Muller et al, 2015). Conversely, strigolactone synthesis in roots is downregulated by both N- and P-availability (Yoneyama et al, 2007; Lopez-Raez et al, 2008). Strigolactone is also translocated into the shoot, where it acts to repress bud outgrowth (Umehara et al, 2008; Gomez-Roldan et al, 2008). SL treatment inhibits outgrowth of buds in both excised nodal segments and whole plants (Umehara et al, 2008;

Gomez-Roldan et al, 2008), while SL synthesis and signalling mutants have constitutively high branching that is insensitive to low N or P conditions (Kohlen et al, 2011; de Jong et al, 2014).

Having been neglected due to a lack of suitable molecular genetic tools, the role of sugar in shoot branching has recently attracted renewed attention (Barbier et al, 2015a). As a direct indicator of available energy, it is perhaps unsurprising that sugar is integrated into the shoot branching regulatory network. In Arabidopsis, pea and rose, treatment with various sugars promotes activation of buds in an excised nodal assay, in a dose-dependent manner (Barbier et al, 2015b). Unlike mineral nutrients, sugar availability does not appear to be translated into a hormonal signal, but nor does it appear to act solely as an energy source, since non-metabolisable sugar analogues trigger the same developmental responses (Barbier et al, 2015b), Rather, sugar appears to have an active signalling role in shoot branching, which may be transduced through one of the several defined sugar signalling pathways, although it is currently unclear which one (Barbier et al, 2015a).

It is clear that CK, SL and sugars act systemically to couple shoot branching to resource availability, and that between them, they determine how many buds should be activated. However, as systemic signals that are available to, and apparently perceived by every bud, these signals cannot determine which buds grow out. An extra layer of regulation is thus required to explain which buds are 'selected' for outgrowth.

2.3 Apical dominance: a classic problem

Apical dominance refers to the ability of actively-growing shoots (and specifically their SAMs) to inhibit the outgrowth of other buds within the shoot system; removal of these dominant apices ('decapitation') results in activation of additional buds. Thus, apical dominance is a classic example of a correlative control. In Arabidopsis, the number of branches which activate after complete decapitation is essentially the same as would be produced without decapitation, suggesting that apical dominance is a manifestation of the system that determines which buds grow out, but not of the system controlling how many buds activate (Figure 2.1). As demonstrated during the 'golden era' of plant physiology, replacing excised shoot apices with agar blocks containing auxin prevents the outgrowth of additional buds (Thimann & Skoog, 1934). It has therefore been clear for over 80 years that the auxin produced in, and exported from the SAMs of actively growing branches maintains their dominance within the shoot system (Went & Thimann, 1937). However, it is has been clear for almost as long that this inhibition is not direct (Went, 1938); apically-derived auxin does not move into dormant buds and applying auxin to buds does not maintain their dormancy (Hall & Hillman, 1975; Brown et al, 1979; Everat-Bourbouloux and Bonnemain, 1980; Prasad et al, 1993; Booker et al,

2003). Historically, much shoot branching research has therefore focused on understanding this 'dominant' effect of apical auxin on buds, but there remain three major unanswered questions. Firstly, how does apical auxin result in inhibition of inactive buds? Secondly, how does this system 'select' which buds to activate or inhibit? Thirdly, how is this system integrated with the systemic signals that determine how many buds should grow?

2.4 The direct action model of signal integration

Broadly speaking, two models of shoot branching control have been proposed and developed over the last decade, which attempt to answer these questions in an integrated fashion. The 'direct action' model derives from the older 'second messenger' model that specifically relates to apical dominance. Since apical auxin does not enter buds directly, the second messenger model proposes that apically-derived auxin in the stem regulates the production of a mobile signal (i.e. the second messenger), which itself enters the buds and regulates their outgrowth (reviewed in Domagalska & Leyser, 2011). CK has been a long-standing candidate for a second messenger, since it can directly activate bud outgrowth (Wickson & Thimann, 1958; Sachs & Thimann, 1964) and its synthesis is negatively regulated by auxin in many tissues, including both the root and the stem (Nordstrom et al, 2004; Tanaka et al, 2006). More recently, SLs have also been proposed as potential second messengers, since their synthesis is positively regulated by auxin in both root and stem (Dun et al, 2012; Dun et al, 2013), and since they have a potent inhibitory effect on bud outgrowth (Umehara et al, 2008; Gomez-Roldan et al, 2008). However, the second messenger model has recently fallen out of favour because it over-emphasizes the importance of apical dominance relative to systemic signalling.

The direct action model develops these themes, but treats CK and SL, along with sugars, as 'primary messengers' in branching control, rather than adjuncts of the apical dominance system. The model proposes that CK, SL and sugars are systemically transported into buds, and directly affect the ability of the bud to grow out; thus, the relative abundance of these signals determines whether a bud activates or remains dormant (Brewer et al, 2015). The model is consistent with observations that direct treatment of buds with SL, CK or sugars can directly inhibit or activate their outgrowth (Dun et al, 2012; Mason et al, 2014; Brewer et al, 2015). Indeed, it has been suggested that sugar translocation into buds after decapitation is the earliest signal driving bud activation, since certain pea buds start growing long before the auxin levels at those nodes drops (Morris et al, 2005). It is proposed that by removing a major sugar sink (the primary apex), sugar is now available to be transported into buds and promote their outgrowth, an idea supported by sugar treatments and defoliation experiments (Mason et al, 2014). However, it should be noted that if auxin is applied to the decapitated apex, the sugar influx to the buds still occurs, along with the initial growth of the bud,

but sustained bud outgrowth does not occur (Mason et al, 2014). Thus sugar influx is not sufficient to drive bud outgrowth, although it may be necessary to do so.

Based on work in pea and Arabidopsis, the TCP transcription factor BRANCHED1 (BRC1) has been proposed as a central integrator for these systemic signals. In both Arabidopsis and pea, mutations in BRC1 result in increased branching levels, showing that BRC1 negatively regulates bud outgrowth (Aguilar-Martinez et al, 2007; Braun et al, 2012). BRC1 transcription increases in response to SL treatment in a fast and translation-independent manner, and is likewise decreased in response to CK and sugar treatment (Dun et al, 2012; Mason et al, 2014), consistent with it acting to integrate inputs from systemic signals and thereby regulating a local grow/no grow decision. In the direct action model, the effect of apically-derived auxin is to regulate the abundance of the systemic primary messengers, either distally in the roots, or proximally in the stem, consistent with the known effects of auxin on CK and SL synthesis (Brewer et al, 2015). The direct action model also allows for local light availability cues to be integrated into branching decisions. BRC1 transcription is modulated by light availability in a PHYTOCHROME B (PHYB)-dependent manner, and increases in response to shading (i.e. a low red:far red light ratio)(Kebrom et al, 2006; Kebrom et al, 2010; Finlayson et al, 2010; Gonzalez-Grandio et al, 2013). This local regulation allows potentially unproductive buds to be inhibited, even if the bud would ordinarily activate.

The direct action model presents a simple and logical framework for the role of systemic resource-related signals on branching. However, it is at best a partial explanation for global shoot branching control. Since all integration of primary messengers in the model occurs locally, the model does not contain any mechanism for coordinating which buds actually grow. All buds should behave independently, and in the same manner as each other if they are exposed to the same systemic concentrations of SL, CK and sugars. Clearly, this does not occur, because there is a system coordinating bud outgrowth across the shoot system, of which apical dominance is a very visible manifestation. Another manifestation is the competition that occurs between two buds on an excised nodal segment; generally speaking, one bud will manage to inhibit the growth of the other (Snow, 1929; Snow, 1931; Ongaro et al, 2008). Furthermore, the stereotyped patterns of activation that occur in many species are not directly explained by the direct action model. Thus, the primary problem with the direct action model is that it treats apically-derived auxin as part of the quantitative system controlling how many buds activate (i.e. through regulation of primary messengers), rather than the system by which growth is focused into the most appropriate buds.

At a mechanistic level, there are also problems with the direct action model. Recent analysis in Arabidopsis demonstrates that decreased BRC1 expression is not sufficient to induce bud outgrowth, and that increased BRC1 expression is not sufficient to repress bud outgrowth; BRC1 activity is also not necessary to repress bud outgrowth in many contexts (Seale et al, 2017). Furthermore, genetic analysis indicates that much of the effect of SL on shoot branching does not require BRC1 activity (Seale et al, 2017). In grass species, the direct action model is even more problematic. Although homologues of BRC1, including FINE CULM1 (FC1) in rice and Teosinte Branched1 (TB1) in maize, clearly regulate branching levels in these species, the transcription of these genes does not respond to strigolactone treatment, and is not altered in strigolactone mutants (Arite et al, 2007; Guan et al, 2012; Minakuchi et al, 2010). However, it is plausible that other proteins could act to integrate systemic signals in grass buds. For instance, a recent report suggests that activity of the rice IDEAL PLANT ARCHITECTURE1 (IPA1) transcription factor, a member of the SPL family of transcription factors that inhibits branching, is activated by SL signalling (Song et al, 2017).

2.5 The canalization model

The canalization model of branching regulation builds on several earlier ideas in the field, and posits that the highly-regulated transport of auxin through the plant body controls bud outgrowth (Prusinkiewicz et al, 2009). Morris (1977) showed that the ability of a bud (or branch) to grow is tightly correlated with its ability to export auxin. Bangerth expanded on this idea, by proposing that the export of auxin from an actively growing organ is able to inhibit the export of auxin from other organs (‘auxin transport auto-inhibition’, thereby preventing their outgrowth (Bangerth, 1989; Li & Bangerth, 1999). This idea was then further developed by Prusinkiewicz et al (2009), who united it with the canalization hypothesis of Sachs, which was originally proposed to explain the patterning of vascular initiation in plants (Sachs, 1969; Sachs, 1981). The canalization hypothesis has two central tenets: 1) that auxin is actively transported from source to sink and 2) that established routes of auxin transport become progressively narrower and more polarised; the resulting ‘canalised’ transport routes determine the positioning of vascular elements (Sachs, 1969; Sachs, 1981). There is strong phenomenological and experimental evidence to support the canalization hypothesis, even though its mechanistic basis remains unclear (reviewed in Bennett et al, 2014).

Modern treatments of canalization tend to re-state the hypothesis in terms of the behaviour of auxin transport proteins. At apoplastic pH, auxin (a weak acid) is predominantly protonated, and can move passively through the plasma membrane into cells. Conversely, at cellular pH auxin is negatively charged and cannot diffuse out of cells; the chemiosmotic hypothesis thus postulated the existence of auxin transport proteins to mobilize auxin from cells, and further that polar localization of these

transporters would lead to the observed polar auxin transport (Rubery & Sheldrake, 1974; Raven 1975). Consistent with this hypothesis, the PIN family of auxin efflux carriers have been identified as transmembrane proteins that are required for efficient cell-cell transport of auxin, and which often have polar localizations (reviewed in Bennett et al, 2014). For instance, PIN1 is polarly localized to the basal plasma membrane in cells of the stem, and in pin1 mutants auxin transport through the stem is greatly reduced (Bennett et al, 2016). While there has been considerable debate around the issue, it is now generally accepted that PIN proteins are transporters for auxin, and not simply regulators of auxin transport. They appear to be secondary transporters, using an electrochemical gradient to mobilise auxin, rather than ATP (Zazimalova et al, 2010). PIN proteins often display highly dynamic patterns of re-localization in cells, particularly in the hypocotyl and root meristem in response to tropic stimuli (Adamowski & Friml, 2015). Thus, canalization is now generally thought to arise through positive-feedback regulation of PIN protein expression and localization, which becomes more focused and polar as auxin is transported through tissue, consistent with the canalization hypothesis (Sauer et al, 2006; Scarpella et al, 2006).

As applied to shoot branching, the canalization hypothesis proposes that buds need to export auxin in order to grow (following Bangerth, 1989), and that this export can only occur if the bud creates a canalised link between the bud and the vascular-associated 'polar auxin transport stream' (PATS) in the stem. The ability to form this link depends on the bud being a sufficiently good auxin source, and the stem being a sufficiently good auxin sink (Prusinkiewicz et al, 2009). Key to the development of the canalization model was the observation that SL negatively regulates the abundance of the PIN1 auxin efflux carrier in stems, and that this increased PIN1 and auxin transport is causally related to the increased branching seen in SL-deficient mutants (Bennett et al, 2006; Crawford et al, 2010; reviewed in Waters et al, 2017). Essentially, SL-deficient mutants are proposed to make more branches because the stem is a better sink for auxin, and more buds can therefore create a canalised link to the stem (Prusinkiewicz et al, 2009). Further evidence for the canalization model of branching has come from observation of other Arabidopsis mutants affected in auxin transport processes (Prusinkiewicz et al, 2009; Shinohara et al, 2013; Bennett et al, 2016), mathematical modelling (Shinohara et al, 2013), and direct observation of canalization in activating pea buds (Balla et al, 2011; Balla et al, 2016). Recent work has demonstrated that auxin transport potential in stems is much more widespread than previously thought, with a 'connective auxin transport' (CAT) system linking wider stem tissues to the PATS (Bennett et al, 2016). CAT is associated with the PIN3, PIN4 and PIN7 efflux carriers, and appears to facilitate the outgrowth of buds and communication between them (Bennett et al, 2016).

The elegance of the canalization model is that it provides a straightforward explanation for the conversion of systemic signals regulating how many buds activate into a system regulating which buds activate. Essentially, by altering the properties of the auxin transport system, systemic signals limit the number of buds that can activate in a canalization-dependent manner, and the buds which are the strongest auxin sources out-compete the other buds and are able to activate. The canalization model thus provides a simple explanation for apical dominance; actively growing branches prevent activation of buds because they decrease the auxin sink strength of the stem, and removal of branches allows new activation by increasing the sink strength (Prusinkiewicz et al, 2009). The canalization model is also able to explain the bi-direction bud-bud competition that occurs in excised 2-node segments (Balla et al, 2016). The canalization model thus presents an integrated framework, in which branches are able to communicate, and through which branching is coordinated, as a consequence of the properties of the auxin transport system.

However, the canalization model has also been extensively criticised (e.g. Dun et al, 2006; Brewer et al, 2009; Brewer et al, 2015). Many of these criticisms arise because of confusion about what the canalization model actually states. Crucially, canalization is a dynamic process that only requires relative differences in auxin levels between tissues. Creating dynamic and relative changes in auxin is highly challenging, and thus experiments that create static and absolute differences in auxin levels are used to either support or challenge the canalization hypothesis (Bennett et al, 2006; Brewer et al, 2009; Brewer et al, 2015). The canalization model is also mechanistically enigmatic, which has made it difficult to test via molecular genetic approaches (Bennett et al, 2014); suitable genetic tools are also scarce, although the recent identification of roles for PIN3, PIN4 and PIN7 in shoot branching control may improve this situation (Bennett et al, 2016). The most important criticisms of the canalization model, however, are that it does not straightforwardly explain why direct application of CK and sugar to buds promotes their outgrowth, nor why direct SL treatment can inhibit buds even when auxin transport is completely inhibited (Brewer et al, 2015). Furthermore, the canalization model does not account for of the activity of BRC1, nor the ability of SL, CK and sugar to regulate BRC1 expression.

2.6 A hybrid model of shoot branching control

It is clear that neither the direct action nor canalization models are satisfactorily able to explain all the available data. Whilst the debate surrounding the veracity of the models has become rather polarised, the models are in no way mutually exclusive or irreconcilable, and a hybrid model seeking to incorporate elements of both direct action and canalization has previously been proposed (Seale et

al, 2017; Waters et al, 2017). Here we expand upon this proposal, and seek to reconcile current data into a single plausible model of shoot branching control (Figure 2.6).

We propose that there are four key processes in the regulation of branching; priming, auxin transport re-modelling, committed outgrowth and competition. Priming is the bud-autonomous integration of developmental status, systemic signals, and local cues. As the direct action model proposes, it is very likely that many of these cues are integrated through regulation of BRC1 expression. Thus, buds are primed for activation by CK and sugar availability by decreased BRC1 expression, and de-primed by SL (at least in eudicots) and poor light by increased BRC1 expression. Developmental status may also be integrated through BRC1, with different basal levels of BRC1 expression determining the 'activation potential' of different buds. For instance, in rice, higher levels of FC1 expression in basal buds is correlated with their greater inhibition (Arite et al, 2007). Furthermore, the higher activity of cauline buds in Arabidopsis might be determined in part by lower BRC1 expression relative to the rosette buds. We propose that cumulative decreases in BRC1 expression prime the buds, increasing their 'activation potential' and pre-disposing them towards outgrowth. We propose that priming does not itself determine whether buds can undergo committed activation, but can result in partial activation, and substantial growth of the bud. This is consistent, for instance, with the early sugar-induced growth of pea buds after decapitation (Mason et al, 2014). In line with the canalization hypothesis, we propose that one key effect of priming is to increase the auxin source strength of the buds, but priming may also have other effects such as increased cell division within the bud, which might itself lead to increased auxin levels (Figure 2.6).

In addition to priming (or de-priming) buds, we propose that systemic signals also re-model the auxin transport system in stems (Figure 2.6). This alters the sink strength of the stem for auxin, and thereby alters the ease with which canalised auxin transport links are formed between bud and stem. Effectively, remodeling of the auxin transport system sets the 'activation threshold' that buds need to achieve to activate. As discussed above, there is strong evidence that SL remodels the auxin transport system by removing PIN1 from the basal plasma membrane of cells in the stem, and that this directly alters the ability of buds to grow (Bennett et al, 2006; Crawford et al, 2010; Shinohara et al, 2013). There is also evidence for both CK and sugar regulating auxin transport (Marhavy et al, 2014; Simaskova et al, 2015; Barbier et al, 2015), but more work is required to establish the extent and relevance of this to shoot branching.

We propose that committed outgrowth can occur if the activation potential of a bud exceeds the necessary activation threshold. A simple re-statement of this premise in terms of canalization would

be that committed activation only occurs if buds are a sufficiently strong auxin source that they can form a canalised link to the auxin sink in the stem, resulting in the active export of auxin from the buds. However, we believe that the reality is slightly more nuanced. One of the key roles of BRC1 appears to be to generate the binary, switch-like behavior of buds in response to the auxin landscape (Seale et al, 2017). brc1 mutant buds are not completely inhibited by auxin treatment, but nor are they as active as untreated buds, essentially displaying a continuous response to the auxin, rather than a binary one (Seale et al, 2017). BRC1 activity thus seems to be particularly important to prevent the partial activation of those buds that would otherwise be able to weakly canalise to the stem, and weakly export auxin. Although Brewer et al (2015) argued that complete inhibition of auxin transport in buds does not prevent their outgrowth, it should be noted that the buds in these experiments are not necessarily undergoing committed activation, but rather a gradual growth consistent with uncommitted growth.

As in the canalization hypothesis, we propose that when a bud undergoes committed outgrowth, the exported auxin from the bud lowers the auxin sink strength of the stem (Figure 2.6). This creates competition between buds, as each successive activated bud makes bud-stem canalization more difficult for all other buds. Thus, although many buds may exceed the initial activation threshold of the system, cumulative bud activation raises the threshold until no more buds can activate. Overall, we therefore propose that through combined and coherent effects on both bud priming and auxin source strength (activation potential) and stem auxin sink strength (activation threshold), systemic signals are able to determine the total number of buds in the system that can activate.

This still leaves open the question of how the system determines which buds grow. It would be theoretically possible for the system to be balanced in just such a way as to be self-selecting; i.e. only the precise number of buds required are primed to exceed the activation threshold. However, such a system would lack flexibility, and does not reflect the actual properties of shoot branching, in which buds that are clearly primed to grow can nevertheless be inhibited. An alternative explanation, rooted in the canalization hypothesis, would be that, at any given time, the buds which are the best auxin sources (i.e. most highly primed) activate, in approximate sequence, until no more buds are able to export their auxin. Thus, the local cues that result in differential priming between buds would ultimately determine which buds activate.

However, this is an unsatisfactory answer as to how distinctive stereotyped patterns of bud activation occur. For instance, to explain the basipetal (top-down) sequence of bud activation in Arabidopsis branching (Hempel & Feldman, 1994), it would be necessary to suppose that the buds were

differentially primed in exactly that pattern – a supposition for which there is no obvious explanation. Prusinkiewicz et al (2009) dealt with this problem in their model by assuming that vegetative meristems are highly dominant, but once they transition to inflorescence meristems, they no longer produce as much auxin. Thus, the conversion of the primary SAM to an inflorescence releases the inhibition on the uppermost cauline bud, which begins to grow, but which then converts to an inflorescence and releases the inhibition on the second cauline bud; and so on. However, there are several problems with this idea, not least that there is little experimental evidence for weakened dominance after floral transition. On the contrary, there is reasonable evidence that inflorescence meristems remain dominant; the main inflorescence meristem controls the growth rate and angle of the primary branches (Roychoudhury et al, 2013), while those branches in turn are able to inhibit the growth of their own secondary branches. We believe that the best explanation for this pattern is that the activation threshold is not systemically constant, but varies in space and time. We thus propose that basipetal bud activation occurs due to a basipetal gradient in activation threshold, rather than any differences between the buds themselves. This is consistent with observations of the CAT system in Arabidopsis inflorescences, in which the youngest tissues have high expression of PIN4 and PIN7, which rapidly declines with age (Bennett et al, 2016). This creates a highly-canalization conducive environment for the upper cauline nodes, allowing their early activation relative to subtending buds. We thus propose that both local variations in bud activation potential and stem activation threshold underlie observed patterns of bud activation.

3. Carpic dominance and fruit-fruit communication

When Bangerth proposed his auxin transport auto-inhibition model he was particularly inspired not by apical dominance, but by the correlative inhibition that occurs between fruits on the same plant. This correlative inhibition is a well-known and long-discussed phenomenon, particularly in the fields of horticulture and floriculture, where ‘dead-heading’ and/or prompt picking of fruit is required to stimulate further flower and fruit production in many species. However, as far as we can establish, this phenomenon has never been formally named. Since the phenomenon is actually driven by the developing seeds (discussed below), we will henceforth describe it as ‘carpic dominance’, by analogy with apical dominance.

3.1 The nature of carpic dominance

Dominance amongst fruits has been described in a wide range of species, including cucumber (de Stigter, 1969), wheat (Fisher, 1973), soybean (Nooden et al, 1984), oilseed rape (Pechan and Morgan, 1985), and tomato (Bangerth, 1989). The phenomenon is expressed in a variety of ways; ranging from mild inhibition of growth to complete abortion or abscission of developing fruits. Cucumber provides

a particularly striking example, in which a few pollinated fruit can completely inhibit further fruit-set from otherwise viable, pollinated ovaries. In oilseed rape, fruit-set ceases after the first ~50% of flowers are pollinated, but the plant continues to open flowers for another 10 days or so; these will not develop even if pollinated (Tayo & Morgan, 1976). In soybean and other legumes, pods that have started to develop after pollination are often shed in substantial numbers, under the influence of older developing fruits (Nooden, 1984). This is echoed in many spring-blossoming fruit trees, which shed excess fruits during the 'June drop'. Much milder effects are also possible; for instance tomatoes exhibit repressed growth of later-pollinated fruits within an inflorescence (truss) (Bangerth, 1989). There does not seem to be any carpic dominance in Arabidopsis – there is no obvious inhibition of late-set fruit growth, and no fruit abscission occurs – which might be related to its ruderal habit and the strategy of producing hundreds of small fruits. Altenatively, or in addition, the almost certain self-pollination of Arabidopsis flowers may render carpic dominance unnecessary, because fruit set can be determined entirely by the rate of flower production.

Although carpic dominance tends to be manifested at the level of fruits, it has long been understood that it is the development of seeds that inhibits new fruits (and therefore seeds) from being formed (Bangerth, 1989). Thus, seedless, parthenocarpic plants produce far more fruits than their seeded counterparts (Pandolfini, 2009; Ostergaard, 2009; Heuvelink and Korner, 2001). Indeed, parthenocarpic fruits are typically cultivated in horticultural industry as a method of producing higher-yielding plants with more consistency between fruit size. As with apical dominance, the role of carpic dominance seems to be to determine which fruits grow, focussing available resources into an appropriate number of seeds. As discussed above, many species make many extra flowers that do not ultimately produce fruits, or make extra fruits that will ultimately not be maintained. Presumably, since pollination of any given flower is not guaranteed, 'over-flowering' has evolved as a mechanism to ensure that a minimum number of fruits are set. Carpic dominance then acts as corresponding mechanism to restrict the number of fruits that are actually maintained. This yield-limiting process directs resources towards the development of fewer fruits than might be supported given sustained favourable conditions. However, one of the main roles of carpic dominance is presumably to ensure that sufficient viable fruits and seeds will be still be produced even if access to resources is dramatically reduced. Consistent with these ideas, the severity of carpic dominance is influenced by environmental factors, allowing plants to proactively adjust fruit numbers both during and after seed set (Bangerth, 2000).

Compared to shoot branching, control of fruit/seed set – taken as whole – seems to be much less of a binary process, with varied outcomes for individual fruits including inhibition, abscission or a

continuum of growth. However, a more binary version of carpic dominance may underlie the production of heteromorphic fruit, a trait which has evolved independently in a number of angiosperm families (Lenser et al, 2016). For instance, in the desert plant Aethionema arabicum (Brassicaceae), plants produce two distinct fruit morphs; small indehiscent fruits that only contain a single seed and abscise intact from the plant, and larger dehiscent fruits with multiple seeds that open on the plant (Lenser et al, 2016). This appears to be part of a bet-hedging strategy in which Ae. Arabicum makes seeds available for immediate germination (to exploit e.g. rainfall) and places other seeds into a long-term seed bank (Lenser et al, 2016). Intriguingly, the ratio of dehiscent:indehiscent fruit is much higher on the main inflorescence than on primary and secondary branches, but removal of dehiscent fruits on the main inflorescence increases the dehiscent:indehiscent ratio on the branches (Lenser et al, 2016). This suggests that the indehiscent fruit are correlatively inhibited by dehiscent fruit through carpic dominance, and that the parameters of the system are fine-tuned in such a way as switch fruits from one highly uniform fruit type to a distinct, smaller morph.

3.2 Possible mechanisms for carpic dominance

The carpic dominance phenomenon makes it clear that there must be a mechanism for communication and coordination of growth between fruits. Since inhibition of fruits can occur acropetally and basipetally within or between inflorescences, this communication outwardly appears to be multi-directional (Bangerth, 1989). As with shoot branching, a source-sink driven nutrient diversion hypothesis was initially proposed to explain carpic dominance. Beyond the fundamental argument that plants do not passively wait for nutrient limitation to occur, this is not likely to be the case for a variety of reasons. For instance, carpic dominance is apparent from early in fruit development, although at this stage the requirement for assimilates is likely to be very low (Nooden et al, 1984; Bohner & Bangerth, 1988). Furthermore, the removal of dominant fruits in some species resulted in yield over-compensation later in the season (Ojehomon, 1970).

As alluded to above, Bangerth (1989) argued instead that carpic dominance, like apical dominance, was most likely driven through auxin transport auto-inhibition. He proposed that the higher export of auxin from a dominant early-induced fruit inhibits the export of auxin from later-induced, inhibited fruits. This idea was supported by his work on the effect of pollination timing on the size and auxin export capacity of tomatoes (Bangerth, 1989). Fruits from tomato flowers pollinated in their normal sequence decrease in size, with older fruits being larger than their younger counterparts on the same truss. The level of auxin export from each fruit correlated to its size, consistent with previous work showing that the rate of auxin transport from a fruit is related to the number of seeds it contains (Sjut and Bangerth, 1984). Furthermore, the removal of a dominant fruit from a truss rapidly results in

increased polar auxin transport from the remaining fruit, regardless of whether they are seeded or pathenocarpic (Kim et al, 1992). Conversely, as has been frequently observed within horticulture, the near-simultaneous pollination of flowers on a single truss results in the production of fruits of a comparable size and auxin transport (Bangerth, 1989). As with shoot branching, Bangerth's model points towards a canalization-dependent mechanism driving fruit/seed-set. Unlike apical dominance, carpic dominance is poorly studied, and there is currently relatively little data either in support or contradiction of this idea; however, the idea certainly warrants testing. One major difference between apical and carpic dominance is the non-binary nature of the latter. In terms of canalization-based explanation, this would suggest that fruit that exceed the activation threshold (i.e. are not inhibited) display a continuous linear relationship between auxin export and growth, rather than simple switch-like behavior (Figure 3.2).

An interesting consequence of a canalization model for carpic dominance is that fruit absicission/pod shed would be a natural outcome of the process. Development of fruits from ovaries that have not been pollinated (or in 'excess' pollinated ovaries) would be an unnecessary use of resources, and ethylene production in these tissues normally promotes their absicission. This ethylene production is inhibited through the action of auxin, which is synthesized and exported from seeds after fertilisation (Pomares-Viciana et al, 2017). Intriguingly, it has been shown that the auxin content of parthenocarpic courgettes is twice that of non-parthenocarpic courgettes (Pomares-Viciana et al, 2017), and many other parthenogenic crop cultivars exhibit high auxin content (Kim et al. 1992). Furthermore, mutation of AUXIN RESPONSE FACTOR8 (ARF8) results in parthenocarpic fruits in both Arabidopsis and tomato (Goetz et al, 2006; Goetz et al, 2007). Thus, pathenocarpy might arise at least in part from increased auxin export from unfertilised ovaries, preventing abscission and allowing pseudo-dominance of the fruit. In general, abscission zones are well-known to form where there is a lack of auxin transport out of organs (Kim et al, 1992; Carbonell-Bejerano et al, 2011; Martinez et al, 2013; Pomares-Viciana et al, 2017). Thus canalization-driven inhibition of auxin export from fruits would in many cases result in the absicission of fruits without further requirement for any additional decision making process.

4. A unified dominance mechanism?

Bangerth explicitly proposed his auxin transport auto-inhibition model as a generalised explanation for all correlative controls, which he suggested formed a unified phenomenon of 'primigenic dominance'. We concur with Bangerth in proposing that the same basic mechanism underlies both

apical and carpic dominance, but are they truly the same phenomenon? That is to say, is there a single unified form of dominance mechanism, and if so, what are the consequences of this?

The classic study of Hensel et al (1994) on correlative control in Arabidopsis provides apparent support for this idea. Hensel et al observed that Arabidopsis inflorescences undergo a quasi-simultaneous arrest after approximately 20 days of flowering, but that the male-sterile ms1-1 mutant did not cease flowering until much later. Thus, the seeds of developing Arabidopsis inflorescences appear to inhibit the activity of the inflorescence meristems. Interestingly, the cessation of flowering in wild-type was shown to be brought about by a regulated and reversible 'global proliferative arrest' (GPA), whereas the ms1-1 line eventually arrested due to an irreversible accumulation of morphological abnormalities (Hensel et al, 1994). The arrested meristems in wild-type essentially enter in a dormant state that is equivalent to axillary bud dormancy (Hensel et al, 1994; Wuest et al, 2016). Continuous de-podding during flowering prevented GPA, and de-podding after GPA caused reactivation of arrested meristems, firmly implicating the fruits as the cause of the phenomenon. Further analysis showed that mutations reducing seed production by greater than 50% produce a larger number of total flowers and do not typically achieve GPA; it was therefore concluded that GPA is ultimately controlled by the developing seeds (Hensel et al, 1994).

Hensel et al proposed two models to explain GPA; a classic source-sink model, and one in which a cumulative, seed-derived signal acts to inhibit meristematic activity (Hensel et al, 1994). Recently, Wuest et al (2016) revisited this topic, and on the basis that the transcriptome of arrested apices resembles that of dormant buds, and that removal of fruits stimulates axillary shoots which were previously dormant, proposed that GPA is brought about by seed-driven domination of the inflorescence meristems. Wuest et al (2016) suggested a gradual transfer of dominance from shoot apices to fruits as seeds develop, but did not suggest a causative agent for this hypothesized process. Given the above discussions of plausible mechanisms for apical and carpic dominance, an obvious hypothesis would be an interchange of dominance between seed and shoot apices based on the canalization model (Figure 3.2). As flowers are fertilised and seeds develop, auxin export from fruits would gradually increase, decreasing the auxin sink strength of stems. Once a threshold was reached, the auxin exported by the seeds would collectively outcompete the apical meristems, leading to their inhibition.

5. The end of flowering, fruiting and growth

5.1 A cross-species round up

As Henry Louis Mencken famously commented, "for every complex problem there is an answer that is clear, simple, and *wrong."* The problem with the GPA model is that, while it would elegantly explain events in Arabidopsis, it is a very poor explanation for post-flowering events in most other species. Examination of a handful of common agricultural species quickly demonstrates that there can be no singular process by which seed-driven carpic dominance inhibits flowering and proliferation. As discussed above, in oilseed rape, a close relative of Arabidopsis, initiation of productive fruit pods only occurs in approximately 50% of flowers, and flowering (and pollination) may continue for at least 10 days after the final productive pods are set (Tayo & Morgan, 1976). Carpic dominance is the likely explanation for the failure to initiate productive pods from all pollinated flowers, but clearly cannot explain the termination of flowering in this case. A converse situation occurs in many legume species (e.g. soybean), where productive pods are initiated from most flowers, but many pods are subsequently shed through an active abscission process, as a result of carpic dominance (Nooden, 1994). In this case the end of flowering precedes the end of the seed-set process.

Spring blossoming fruit trees such as apple, pear and cherry provide perhaps the most comprehensive demonstration that separate post-floral processes are at work in flowering plants. In these species, floral transition occurs in the autumn, and the 'extent' of flowering is determined by the number of inflorescence buds that are initiated, and which subsequently over-winter in a dormant state. These buds then synchronously activate in spring (bud-break) producing a blossom whose extent was determined in the previous autumn; these trees will usually initiate far more fruits than will ever be sustained. As mentioned above, this results in the 'June drop' as smaller/less viable fruits are shed from the tree, again as a result of carpic dominance (Bangerth, 1989). Crucially, these carpic dominance effects do not cause the end of flowering (which occurred the previous autumn), and seed-set and fruit growth happens simultaneously with vigorous spring vegetative growth in the trees. Thus, developing seeds do not necessarily inhibit flowering or proliferation in these species. This said, heavy fruiting in these trees can limit the extent of flowering in the subsequent autumn, thus limiting the following year's crop, or indeed causing the tree to skip flowering for a year entirely; seed-set and flowering thus are clearly closely intertwined. The perennial alpine plant Arabis alpina, another close relative of Arabidopsis, provides a further example of de-coupling of flowering, fruiting and growth. This is most dramatically demonstrated in perpetual flowering 1 (pep1) mutants of A. alpina, in which active vegetative branches, active floral branches, arrested floral branches and senescing floral branches can all be seen on the same plant (Wang et al, 2009). Thus, in Arabis alpina

the production of seed on one branch does not necessarily inhibit flowering or proliferation on other branches.

This short survey is sufficient to demonstrate that a singular canalization-driven model cannot reasonably explain post-floral development in flowering plants. While there is clear evidence for carpic dominance as a fruit-to-fruit phenomenon, there is much less evidence that the end of flowering or proliferation arises through domination of meristems by fruits or seeds. We would certainly not rule out this process operating in Arabidopsis, especially since it displays little obvious carpic dominance; seed-to-meristem inhibition might replace carpic dominance as a limit on seed-set in Arabidopsis. However, we propose that at least two more processes, which are not directly seed-driven, are required alongside carpic dominance to explain post-floral development across the angiosperm group in general.

5.2 Floral arrest

In late spring the brilliant yellow fields of oilseed rape seen across much of the northern temperate zone suddenly turn green, as the flowering period draws to a close. As discussed above, this 'floral arrest' does not seem to depend directly on carpic dominance effects, and occurs some days after the last pods have set. Random hand pollination of both oilseed rape and the closely related Brassica rapa grown in controlled environments results in a wide distribution of pod and seed numbers per plant (Figure 5.2). However, floral arrest nonetheless occurs synchronously in these plants, suggesting that cumulative seed production does not drive the phenomenon; only in totally unpollinated plants does floral arrest fail to occur. This suggests that floral arrest is not directly seed driven, but does requires at least some seed-set in order to occur. These results are thus compatible with those of Hensel et al (1994), because de-podded and male-sterile plants have no pods, and thus do not undergo floral arrest, while complete de-podding reverses floral arrest. In our hands, wild-type Arabidopsis plants (Col-0) grown in long-day conditions reliably undergo floral arrest after ~7 weeks of growth. This still occurs if plants are partially de-branched or de-podded, suggesting that partial seed set is also sufficient to trigger floral arrest in Arabidopsis (Bennett lab, unpublished data).

If floral arrest is not driven by cumulative seed-set, then how then do we explain the phenomenon, in particular its precise timing? Two obvious possibilities are that floral arrest is driven by a 'timing' mechanism that measures the absolute time since germination and/or flowering, or that it is driven by environmental factors such as light or temperature. Given the floral transition is tightly regulated by light and temperature cues to ensure flowering at the optimal time of year, it would seem logical that the same signals also trigger floral arrest when conditions are no longer optimal. However, floral

arrest in Arabidopsis still occurs in constant growth conditions that are still inductive for flowering, suggesting changes in conditions are not necessary to trigger arrest. Although individual oilseed rape fields stop flowering quasi-synchronously, nearby fields (experiencing the same changes in day-length and temperature) may stop flowering up to two weeks later (Figure 5.2), suggesting that it is not changes in absolute day length or temperature that cause arrest. Furthermore, individual inflorescences in Arabis alpina seem able to arrest independently of each other, suggesting that some sort of timing mechanism can work locally in inflorescences (Wang et al, 2009). Although the evidence is patchy at the moment, the answer may lie in a combination of all these factors – that is, a photo-thermal timing mechanism, integrating cumulative light and/or temperature information across the growth season to trigger arrest – either locally in individual inflorescences, or globally across the plant. It is notable that the only mutants identified by Hensel et al (1994) as delaying GPA were the late-flowering circadian clock-associated mutants (gigantea (gi) and luminidependens (ld)), but only in the Ws background, and not in Col-0 or Ler. Since Ws is a phyD mutant, this suggests that a combination of circadian clock and light inputs may determine the timing of floral arrest.

While the mechanism that lies behind floral arrest is currently unknown, the floral arrest signal is presumably perceived or integrated in the inflorescence meristems, and leads to imposition of a quiescent but not differentiated state. As discussed above, the arrested meristems in Arabidopsis appear to be transcriptionally equivalent to dormant axillary buds (Wuest et al, 2016). It thus appears that floral arrest re-imposes dormancy on inflorescence meristems to bring an end to flowering.

5.3 Vegetative arrest and local versus systemic post-floral effects

In monocarpic plants with indeterminate inflorescences, such as Arabidopsis and oilseed rape, the quasi-synchronous arrest of all meristems leads to the impression that the arrest is a global effect caused by a systemic signal (Hensel et al, 1994). However, as discussed above, comparison with Arabis alpina (and other perennials) shows that the floral arrest signal does not impose dormancy on vegetative meristems. While floral arrest causes complete proliferative arrest in Arabidopsis, this is probably because all the active shoot meristems are inflorescence meristems (Woolhouse, 1983), rather than because this is an inherent effect of floral arrest itself. This implies that there is a separate set of signals that can trigger 'vegetative arrest' (for instance, entry into winter-dormancy) in perennials. Presumably, this vegetative arrest is not necessarily a canalization-dependent dominance process, but is imposed by environmental stimuli; qualitatively, the effect is the same however, with an entry into dormancy. Two hypothesizes are suggested by this; either the floral arrest signal is systemic, but is not perceived by vegetative meristems; or the floral arrest signal is only produced and active locally with inflorescence branches. Arguments can be made for both possibilities; the

quasi-synchronous behaviour of inflorescences in Arabidopsis and oilseed tends to suggest a systemic signal, but the temporally-independent behaviour of inflorescences in Arabis alpina tends to suggest a local signal. Indeed, it is possible that the floral arrest signal may vary between different species in terms of systemic vs local activity; clearly more work is needed to identify the signal in the first place before this question can be properly answered.

The issue of local versus systemic signals is closely paralleled in the case of carpic dominance. If carpic dominance and apical dominance are indeed driven by the same basic canalization mechanism, it is difficult to imagine how carpic dominance can act anything other than locally in species such as tomato or apple, where determinate inflorescences are distributed on pseudo-vegetative branches. There is no obvious way of deconvoluting canalization effects to keep systemic branch-branch and inflorescence-inflorescence signals separate, but branches do not inhibit fruits or vice versa in these species. Rather, it seems likely that carpic dominance effects are localised within inflorescences in these species, consistent with visible local effects on fruit growth. However, in species such as cucumber, carpic dominance seems to be truly systemic, and it is thus possible that the effective range of carpic dominance varies considerably between species. Again, further insights into the nature of carpic dominance will be needed to understand its local or systemic effects, and interaction with other correlative controls.

6. Senescence

Fittingly, the final correlative control we will discuss is the proposed inter-generational conflict in which developing seeds bring about the senescence of the maternal plant in monocarpic plants. As with the other correlative controls, this is an observation that dates back to the golden era of plant physiology, but which remains poorly understand (Molisch, 1929; Wuest et al, 2016). It has long been assumed that senescence of plant tissues allows for recycling of nutrients (Hildebrand, 1881), and there is excellent evidence this indeed occurs (Nooden et al, 1984). Senescence is often associated with increased demand for nutrients in sink tissues, and/or environmental scarcity, and presumably is a sound adaptive strategy to maximise resource utilization. At least four senescence syndromes can be outlined; 1) sequential, in which older organs are gradually turned over during the life of the plant; 2) autumnal, in which the leaves of deciduous trees are simultaneously turned over before winter; 3) monocarpic, in which the vegetative leaves are collectively sacrificed during the reproduction effort, thus committing a plant to a monocarpic habit; and 4) terminal, in which the whole plant dies at the end of its life cycle (e.g. Wingler, 2011). There is good evidence that sequential senescence is not under correlative control, and rather seems to be driven by leaf age and/or darkness (Nooden, 1984; Hensel et al, 1993). Autumnal senescence is also not associated with correlative controls, but rather

obviously with environmental conditions. In contrast, both monocarpic and terminal senescence have been proposed to be correlatively controlled, since removal of seeds leads to extended life-span (either of vegetative leaves or of the plants as a whole) in many monocarpic plants (reviewed in Nooden, 1984).

Overall, the status of monocarpic senescence as a correlatively-regulated process is unclear. Nooden's work on soybean suggested that developing seeds exert strong dominance over leaf longevity, but only through very local signalling (Nooden, 1984). However, in Arabidopsis and the Brassicaceae more generally, there is no obvious effect of seeds on leaf longevity (Nooden & Penney, 2011). Furthermore, in dioecious spinach, the male plants also undergo monocarpic senescence despite not setting seed (Leopold et al, 1959). In all species, entry into the reproductive phase seems to at least be a pre-requisite for monocarpic senescence. Indeed, flowering and monocarpic senescence seem to be co-regulated processes at the molecular level, and, for instance, cues such as vernalisation that induce flowering in Arabidopsis also induce senescence (Wingler, 2011). Thus, it is possible that monocarpic senescence in many species may be jointly regulated with flowering, rather than correlatively controlled. Since monocarpy has evolved convergently on many occasions, it is possible that monocarpic senescence has no common regulatory thread, and that in some species it is correlatively controlled, and in some species not (Woolhouse, 1983). An alternative possibility is that monocarpic senescence is the same conserved developmental module as autumnal senescence, but that in monocarpic plants this module is activated in response to different environmental or developmental stimuli (both compared to perennial plants, and compared to other monocarpic plants).

The status of terminal senescence as a seed-controlled process is equally unclear. Like floral arrest, seed set seems to be a pre-requisite for terminal senescence, which is delayed in de-podded plants and sterile mutants (Nooden & Penney, 2011). However, this does not mean that the seeds actively drive terminal senescence, and there are plenty of examples where monocarpic plants have full seed set and do not proceed to terminally senesce. For instance, in the UK oilseed rape plants have full seed set by the end of May, but do not terminally senesce until late July, with many farmers resorting to using glyphosate to expedite the process. A strong possibility is that the process of whole plant senescence is actually driven by the need to dry out seeds and fruits for dispersal, and is therefore only triggered when the seeds are fully mature, and not as a general function of nutrient demand. If this is the case, we might expect to see significant differences in the extent of terminal senescence between plants with shatter-dispersing seeds in which fruit must dry, and in herbivore-dispersed seeds, in which fruits must remain fleshy. Comparing end-of-life in e.g. tomato or courgette with e.g. pea or wheat tends to support this idea, but more work will be needed to test it. Finally, it is worth

noting that complete inflorescences can undergo terminal senescence in perennial plants (e.g. Arabis alpina), without affecting vegetative tissues (Wingler, 2011). Thus terminal senescence is not unique to monocarpic plants, suggesting that the underlying developmental program may be conserved across flowering plants.

Taken together, it remains to be established whether any senescence programs are widely regulated by correlative control in flowering plants. Nevertheless, it is clear that these senescence syndromes are controlled by the same general decision-making principles as for other shoot architectural traits. Senescence is clearly connected to resource availability, is responsive to plant hormone levels, and is coordinated by global demand for nutrients (Nooden, 1984). For instance, cytokinin, an indicator of soil nutrient availability, strongly inhibits monocarpic senescence (Zwack & Rashotte, 2013), while strigolactones, an indicator of nutrient stress, promote sequential senescence in many species (Yamada & Umehara, 2015).

7. Perspectives

Correlative controls form an important class of regulatory mechanisms for plant development, but their study has been neglected and their molecular basis remains unclear. We have examined dominance phenomena (negative correlative controls), and conclude that they may be less widespread than previously thought. We propose that apical and carpic dominance may share a common mechanistic basis rooted in auxin transport canalization. However, canalization itself remains mechanistically enigmatic, and much more work will be needed to test these ideas. Conversely, we have proposed that proliferative arrest (both vegetative and floral) may not be under 'true' correlative controls, but rather that they are more complex phenomena in which seed-set plays a permissive rather than instructive role. Overall, we have attempted to develop a coherent framework for understanding the developmental and regulatory mechanisms that control shoot architecture, and to provide new insights into the end of flowering, fruiting and growth (Figure 7.1).

Producing enough food to feed a growing global population is a critical challenge currently faced by humanity. As we have discussed, plants have evolved to grow conservatively given their available resources for a number of key reasons. This includes trade-offs that allow long-term survival of the species within the environment, and constraining growth to promote individual survival when environmental conditions become less favourable. However, in the context of increasing crop production, this conservativism is probably acting as a significant limitation on yields. For instance, the first 50% of fertiliser application in winter wheat in the UK accounts for 90% of crop yield, with the only 10% additional yield from the remaining fertiliser (Sylvester-Bradley et al, 2015). The plants

are therefore being provided with enough resources to produce higher yields, but their yield remains mechanistically constrained, through ‘reluctance’ to commit to production of additional tillers and ears. The constrained nature of fruit production is more dramatic in apple, with the abscission of many fruits during ‘June drop’ (Abruzzese et al, 1995). Similarly, oilseed rape constrains its own yield by inhibiting growth of later-pollinated flowers, and through the abscission of excess pods later in the season (Child et al, 1998). Clearly, oilseed plants grown in the field must contend with issues that are absent in the controlled conditions; for instance, reduction in green leaf area and stem integrity to Leptosphaeria maculans (Phoma), or loss of unopened flower buds to pollen beetle. While the crop must compensate for these losses through production of extra organs, individual plants still also produce multiple secondary branches late in the season, which do not produce viable seed, effectively wasting resources.

Fully understanding the mechanisms through which apical and carpic dominance act could have substantially enhance our ability to increase crop yields with minimal (or no) increase of inputs. The poor nitrogen use efficiency of most crops makes it clear that they do not fully utilise all of the nutrients they are provided with, and we believe it should be possible to produce increased yields in a wide range of crop species without increasing inputs, but by ‘persuading’ the plants to abandon their inherent caution.

Figures

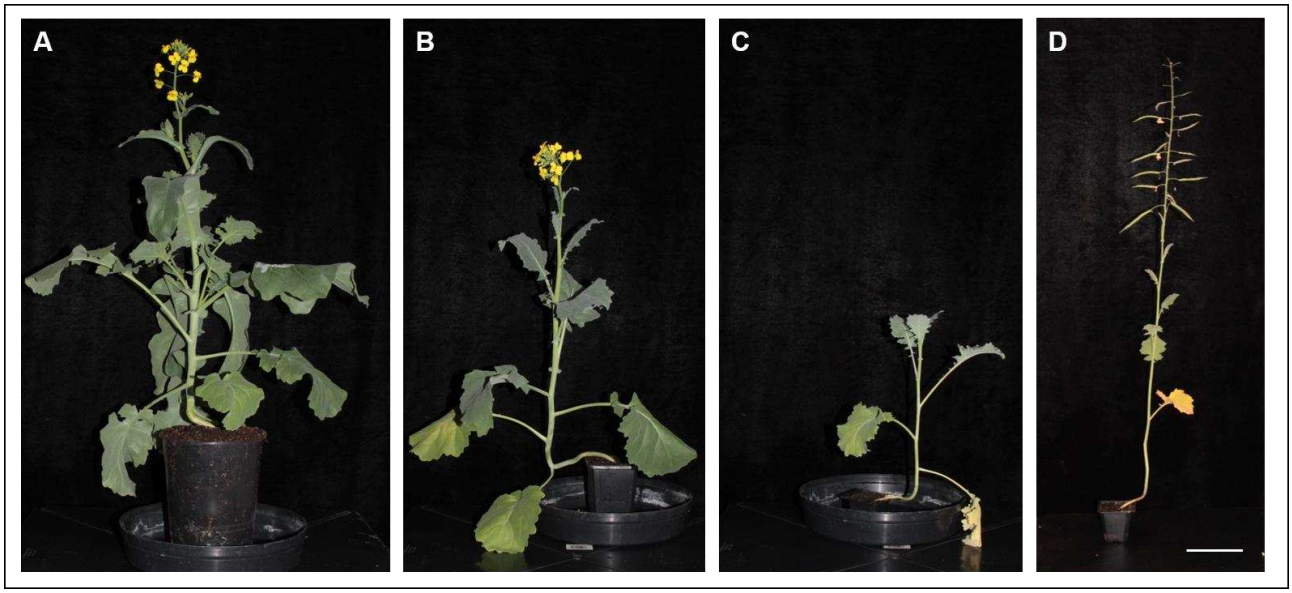

Figure 1.1: Decision making in oilseed rape.

(A-C) 7-week old oilseed rape plants grown in 3 different pot sizes. The plants have adapted their growth to the availability of resources. A= 2 litres of soil, B= 0.75 litres of soil, C, D = 0.15 litres of soil. The small habit of (C) is not caused by lack of nutrients; rather, the plant actively makes developmental decisions that enable it to successfully complete its life cycle (D).

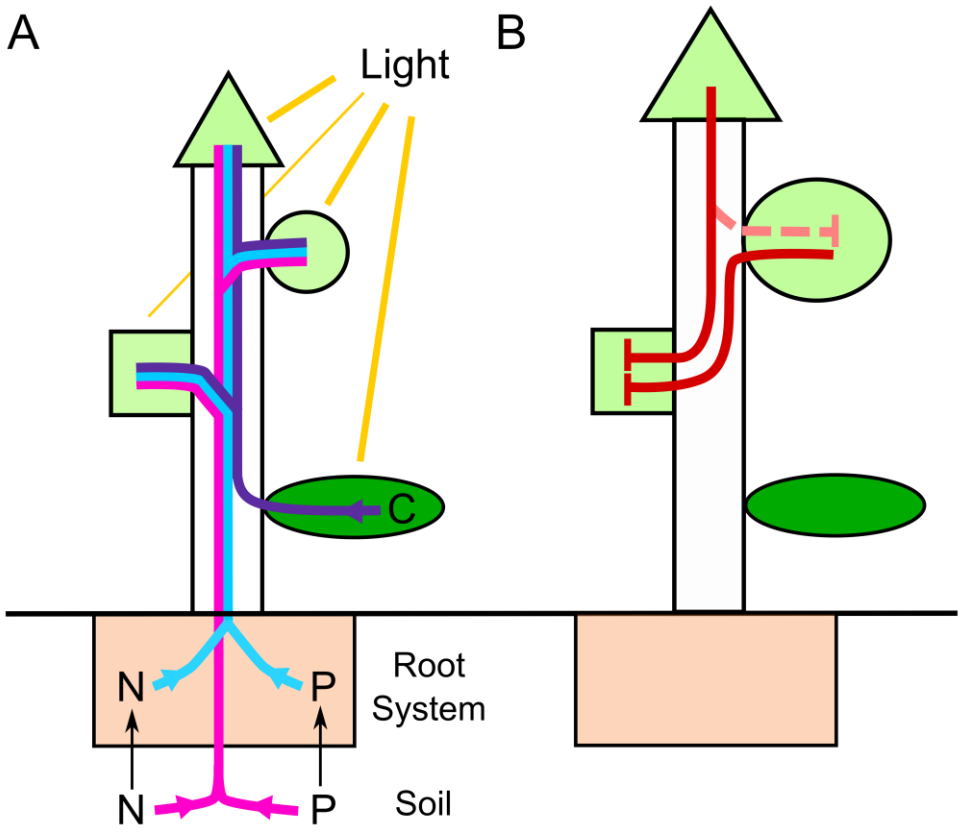

Figure 1.3: A simple model for decision making in plants

(A) Plants integrate external and internal nitrate (N) and phosphate (P) availability into simple systemic signals that move into the shoot system, and are detected by all organs (blue and pink lines). Photosynthetically fixed carbon (C) also acts as a systemic signal (purple lines). Individual organs integrate systemic cues with local information (e.g. light availability) to make a 'pre-decision' about their growth.

(B) Co-ordinating mechanisms determine which organs ultimately grow. These give the appearance of direct organ-organ communication (red lines), but signals do not necessarily move between the organs to mediate these 'correlative controls'.

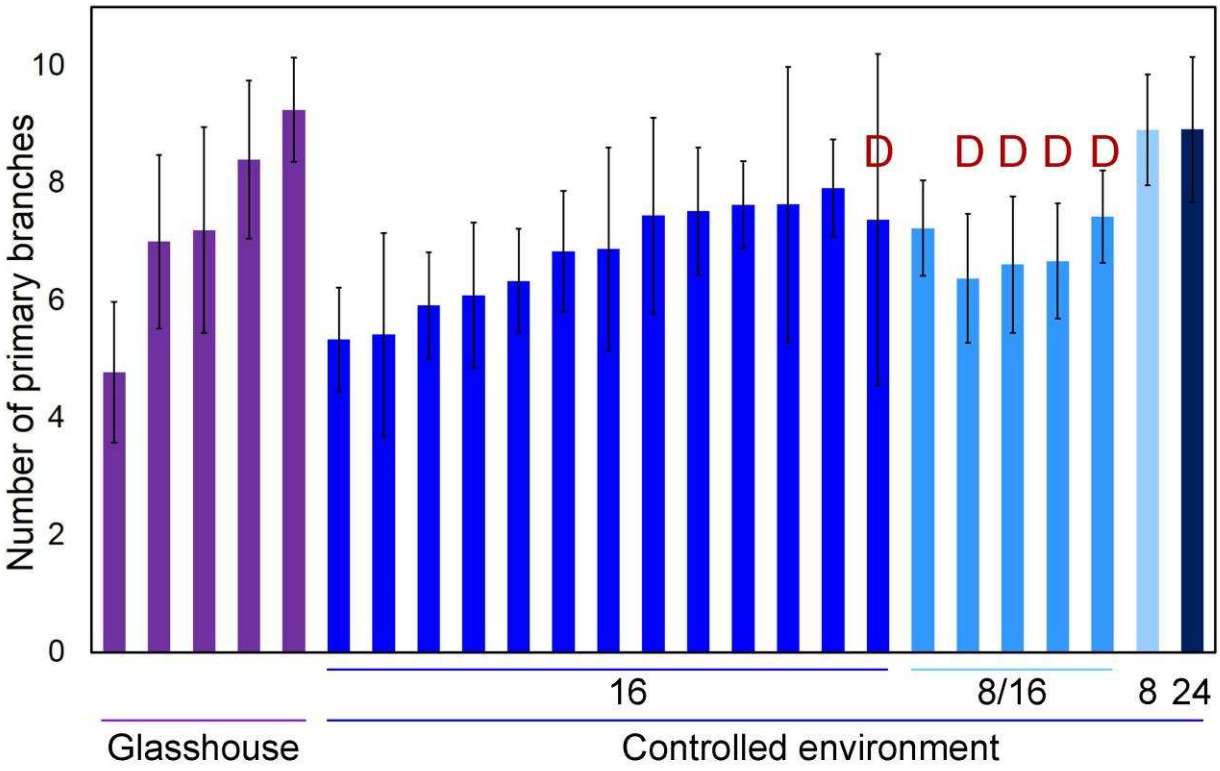

Figure 2.1: Decision making and apical dominance in Arabidopsis branching

Plants grown in the same soil volume tend to make the same number of primary branches, despite quite large changes in life-history and light conditions. Meta-analysis of 23 experiments, in which Col-0 plants were grown in ~150ml of soil, in either glasshouse or controlled environment chambers. Primary branch numbers were measured at the end of flowering. Each bar represents 1 experimental mean ± standard deviation, n per experiment = 8-24. For plants grown in controlled environment chambers, the number of hours of daylight is indicated. '8/16' plants were grown for 4 weeks in short days (8 hours of light) and then until the end of flowering in long days (16 hours of lights). In experiments marked with 'D', plants were decapitated after 2 weeks of flowering by complete removal of all inflorescences, then allowed to recover.

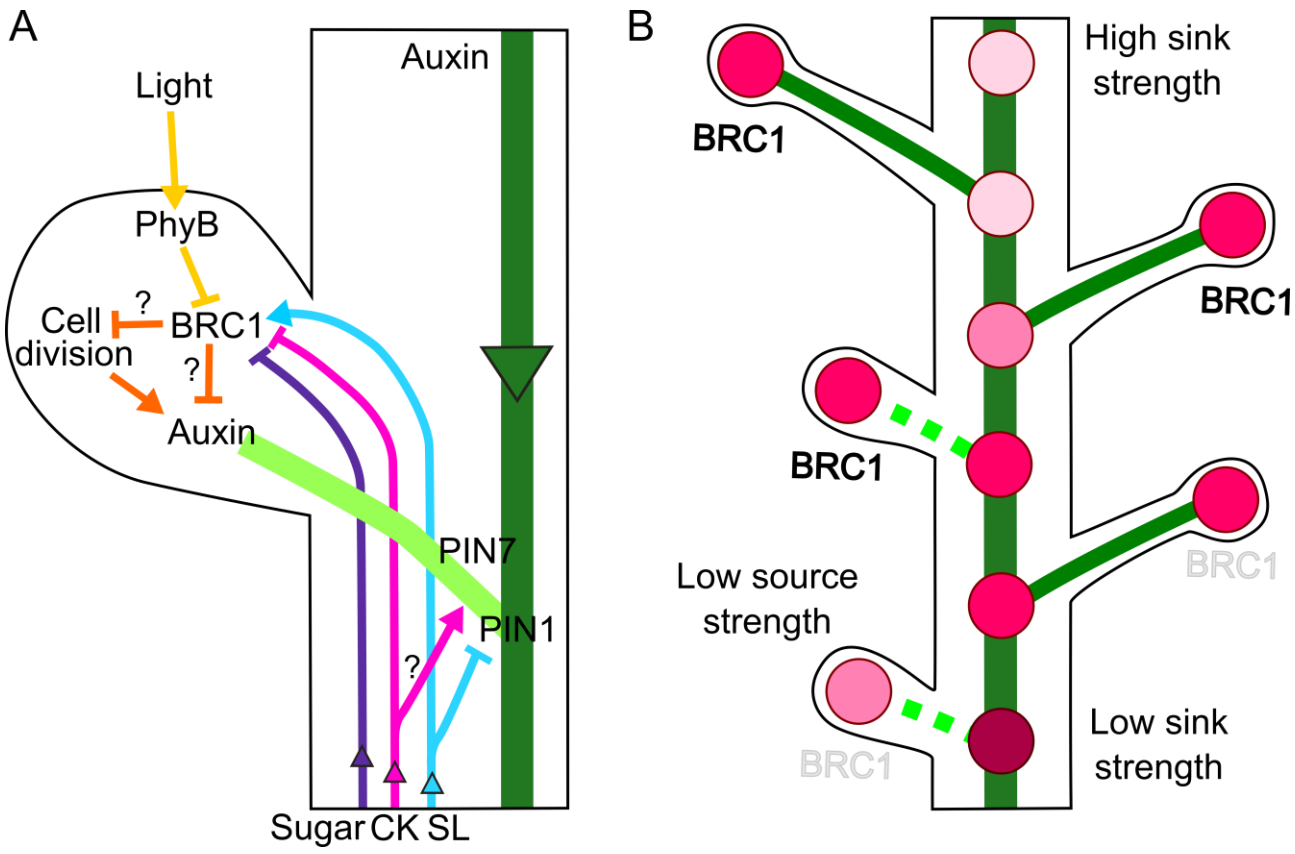

Figure 2.6: A hybrid model for shoot branching

(A) Systemic and local signals prime buds for activation and re-model auxin transport. Strigolactone (SL; pink line) and possibly cytokinin (CK; blue line) alter availability of PIN auxin efflux carriers in the stem. SL and CK also alter transcription of BRANCHED1 (BRC1) in the bud. Sugar (purple line) also alters BRC transcription, as does local light availability, transduced through PHYTOCHROME B (PhyB). BRC1 expression might alter cell division and/or alter auxin source strength of the bud directly or indirectly. Auxin transport is shown in green.

(B) Canalization-dependent competition determines bud outgrowth. Circles indicate auxin source strength of buds (darker colours = higher source strength) and auxin sink strength of the stem (darker colours = lower sink strength). Where buds are sufficiently strong sources and the stem is a sufficiently strong sink, buds are able to form canalised auxin transport links (green lines) to the main stem auxin transport stream, even in the presence of high BRC1 expression (black text). For buds of middling auxin source strength, the high (black text) or low (grey text) expression of BRC determines whether the buds activates or not. For buds of insufficient auxin strength, even low BRC1 expression does not result in activation.

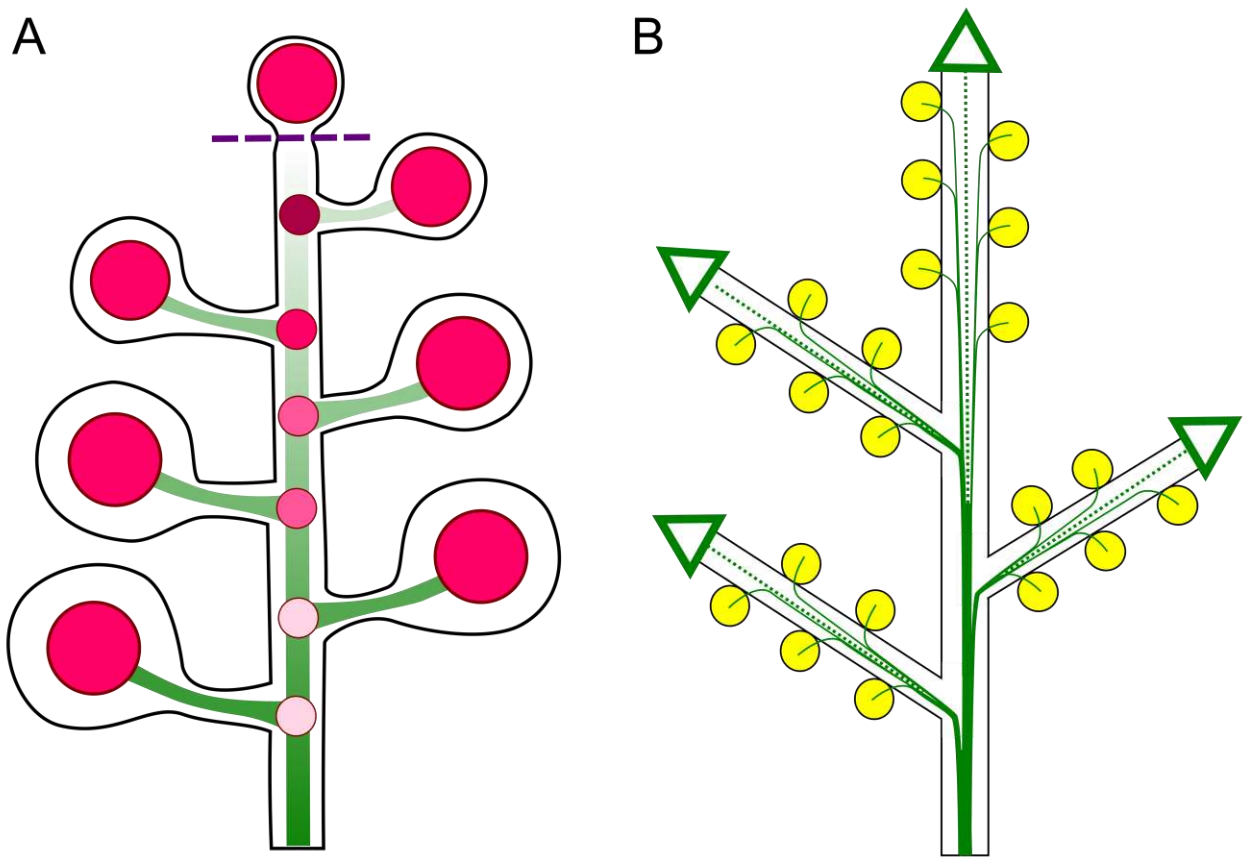

Figure 3.2: Seed-driven dominance

(A) A plausible model of carpic dominance within an inflorescence. Green lines = auxin transport routes, the depth of colour indicates strength of transport. Large circles indicate auxin source strength of fruit, small circles indicate auxin sink strength of the stem at the time the fruit initiated (darker colours = lower sink strength). Fruit growth displays a continuous relationship with auxin export strength, with earlier initiating fruits (at the bottom) exporting more auxin. The final fruit produced (top) does not export sufficient auxin, leading to formation of an abscission zone between fruit and stem (dashed line).

(B) A plausible model of GPA. Green lines = auxin transport routes. Auxin export from fruits (yellow circles) gradually weakens the sink strength of the stem for auxin, causing auxin export from active shoot apices (green trangles) to be inhibited (dashed lines).

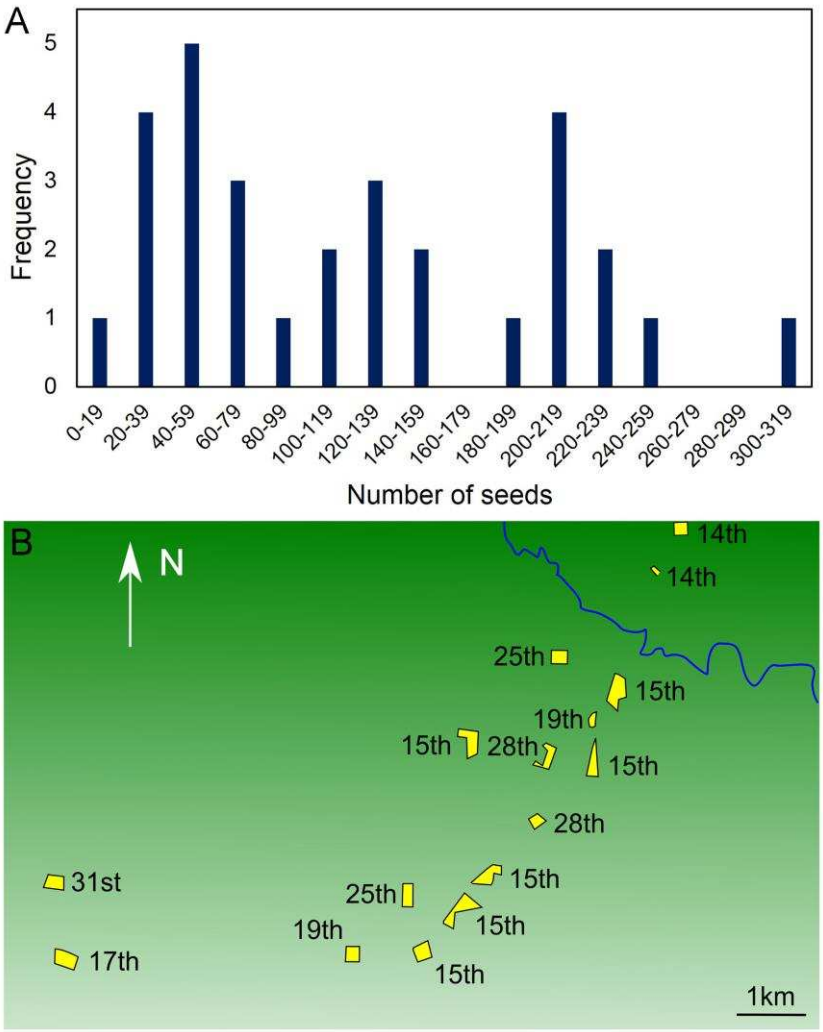

Figure 5.2: Floral arrest

(A) Frequency distribution of the number of seed produced per plant in randomly hand pollinated Brassica rapa plants, all of which underwent floral arrest.

(B) Map showing the relative position of 16 oilseed rape fields in Yorkshire, and the date in May 2017 in which the crops ceased flowering.

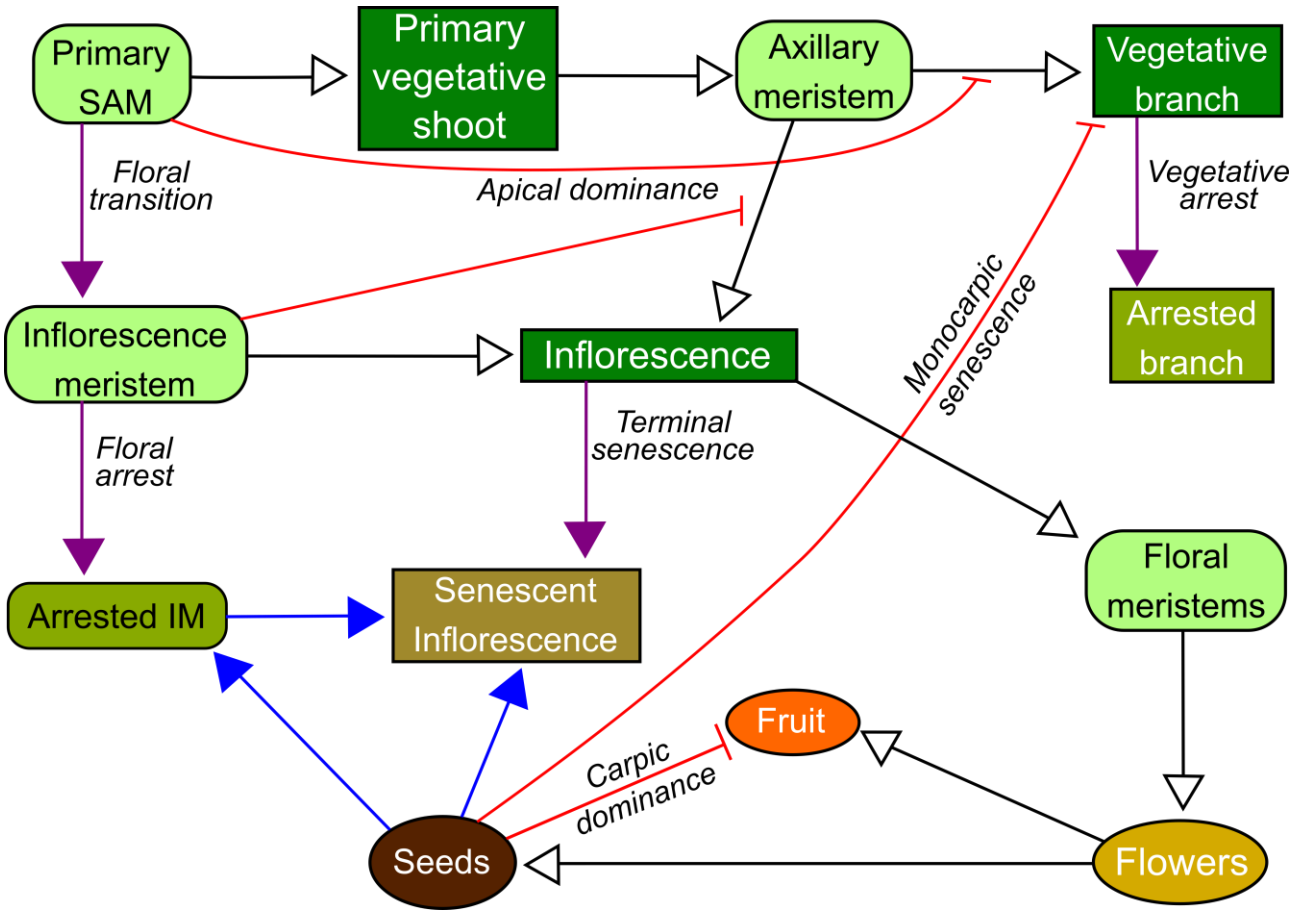

Figure 7.1: Overall model for shoot architectural processes

Flow diagram showing key stages, processes and regulatory mechanisms in shoot architecture. Developmental processes are shown with black arrows, developmental transition with purple arrows, and possible correlative controls with red arrows. Blue arrows indicate that one tissue/stage is a prerequisite for another developmental process or transition.

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