CONCEPTS AND TERMINOLOGY OF APICAL DOMINANCE

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Apical dominance is the control exerted by the shoot apex over lateral bud outgrowth. The concepts and terminology associated with apical dominance as used by various plant scientists sometimes differ, which may lead to significant misconceptions. Apical dominance and its release may be divided into four developmental stages: (I) lateral bud formation, (II) imposition of inhibition on lateral bud growth, (III) release of apical dominance following decapitation, and (IV) branch shoot development. Particular emphasis is given to discriminating between Stage III, which is accompanied by initial bud outgrowth during the first few hours of release and may be promoted by cytokinin and inhibited by auxin, and Stage IV, which is accompanied by subsequent bud outgrowth occurring days or weeks after decapitation and which may be promoted by auxin and gibberellin. The importance of not interpreting data measured in Stage IV on the basis of conditions and processes occurring in Stage III is discussed as well as the correlation between degree of branching and endogenous auxin content, branching mutants, the quantification of apical dominance in various species (including Arabidopsis), and apical control in trees.

Key words: apical control; apical dominance; Arabidopsis; auxin; branching; cytokinin; lateral bud outgrowth; shoot apex.

Apical dominance may be defined as the control exerted by the shoot apex over the outgrowth of the lateral buds (Cline, 1994). This is a classical example of a developmental correlation where one organ of a plant affects another organ (Sachs, 1991). Apical dominance is also referred to as “correlative inhibition” (Hillman, 1984) or in the dormancy literature as “paradormancy,” a type of growth control involving a biochemical signal from another structure (Lang, 1990).

The terminology and concepts associated with apical dominance as used by molecular biologists, geneticists, ecologists, foresters, agronomists, botanists, horticulturists, and plant physiologists often vary in meaning. For instance, do “release of apical dominance,” “bud initiation,” “sprouting,” “tillering,” “regrowth,” “compensatory growth,” “release of meristems,” “branching,” “feathering,” “lateral shoot development,” “axillary bud outgrowth,” “activation of dormant buds,” and “branch induction” all have precisely the same meaning? There is generally little difficulty as long as there is understanding as to the context with which the terms are used. However, in some cases significant misconceptions may arise.

It is the objective of the present essay to explore some of these misconceptions and their implications and to offer some suggestions on the quantification of apical dominance that may be helpful. Although the scope here is general, specific discussions on Arabidopsis branching and apical control in trees are also included.

As an apical shoot meristem of a herbaceous plant develops, axillary buds “arise exogenously from superficial cell layers in the axils of leaf primordia” (Fahn, 1990). These embryonic lateral buds may have inhibition imposed upon them shortly after their formation or after a brief period of growth. As Steeves and Sussex (1989) describe, “...commonly at this time [after bud formation] the dominance of the main apex is expressed in the inhibition of further development of the lateral apices and they remain as axillary buds, often for long periods and sometimes permanently unless the main apex is removed.” Although the elongation of the buds may be essentially inhibited, they remain metabolically active (Stafstrom, 1995). As Hillman (1984) points out, “In some species, inhibited or quiescent buds may be fairly rudimentary whereas in others the buds may be well formed...” If the shoot apex is subsequently decapitated (also sometimes referred to by such terms as “apex removal or excision,” “clipping,” “topping,” “defoliation,” “pruning,” “trimming,” “tipping,” “shearing,” “heading,” “herbivory,” “browsing,” “meristem destruction,” and “pinching”), apical dominance is released and one or more of these lower axillary buds begins to grow out.

Within a few hours after apex removal, measurable increases in the length of the emerging lateral bud can be detected in some species. In other species the lag period may be longer depending upon the degree of inhibition and the stage of the cell cycle at the time of inhibition (Tamas, 1987). In the days and weeks following decapitation, subsequent elongation and development of the lateral bud into a branch shoot occur.

These foregoing developmental steps, which will vary with species and will apply more to dicotyledons than to monocotyledons, may be conveniently divided into four stages (Fig. 1): lateral bud formation (Stage I), imposition of inhibition (apical dominance) (Stage II), initiation of lateral bud outgrowth following decapitation (Stage III), and subsequent elongation and development of the lateral bud into a branch (Stage IV). Even though there is some overlap between the four stages, they each involve some different processes and are affected differently by plant hormones. Hence, each stage should be considered more or less separately. The degree of inhibition imposed in

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Stage II may vary. If it is negligible as with Arabidopsis or greenhouse-grown Coleus (Cline, 1996), then apical dominance is essentially nonexistent and the lateral bud continues to grow out from the time of its formation into Stage IV without the necessity of release via decapitation. This is similar to sylleptic growth, which occurs in many tropical woody species and in a few temperate trees (Halle, Oldeman, and Tomlinson, 1978; Wheat, 1980) and in some young fruit trees (Barlow, 1970; Tromp, 1996) where there is no rest or dormancy period between bud formation and outgrowth.

If the imposition of inhibition is partial as in bean or petunia, then the apical dominance could be termed as "intermediate" with a certain level of lateral bud outgrowth occurring even without decapitation. If the imposition of inhibition is complete as with Helianthus, Tradescantia, or indoor-grown Ipomoea, then apical dominance is strong and no release occurs without decapitation or some drastic restriction of apical shoot growth. Sachs (1991) describes this as the most extreme form of apical dominance and that the norm involves some lateral outgrowth even in the intact plant. Speaking more generally he states: "Many physiological studies stress an all-or-none approach, but this is a result of a careful choice of experimental systems which is dictated by good reasons of experimental design, not an expression of the common reality of plant development."

Any treatment given to a plant 24 h after apex removal (Stage IV) will have a much different effect on lateral bud outgrowth than if it is given immediately following decapitation (Stage III). Wickson and Thimann (1958) found only a small repression of outgrowth of isolated pea buds when auxin was added after a 24-h delay. Several workers (Shein and Jackson, 1971; Stimart, 1983) have pointed out a disturbing aspect of the apical dominance literature is that a distinction is usually not made between the initiation of axillary bud growth and the subsequent axillary shoot elongation, which may be under the control of different hormone factors as demonstrated by Sachs and Thimann (1967).

With respect to hormone effects, the differences in stages may be tentatively defined as follows: I (Cytokinin promoted), II (Auxin repressed), III (Cytokinin released), and IV (Auxin and gibberellin promoted).

When considering the mechanism of apical dominance, the physiologist is particularly interested in the processes involved in the imposition and release of apical dominance (Stages II and III). A common mistake is to attempt to interpret data measured several days or weeks after decapitation (during Stage IV when branch development
is well under way) on the bases of physiological processes occurring within the first few hours (during Stage III at apical dominance release when the lateral bud first begins to grow out). Branch development involves much more than apical dominance release and is affected differently by hormones as mentioned above. What happens in Stage IV is important and valuable to know, but it should not be confused with what happens in Stage III during apical dominance release.

The perspective taken in this essay is physiological. There certainly are other valid and useful perspectives, e.g., genetic, ecological, etc. Whereas the physiologist focuses directly on the processes (i.e., the imposition/release of inhibition in stages II and III), the developmental geneticist may begin by defining the branching gene in Stage IV and then work back toward the earlier stages in order to understand the mechanisms involved (e.g., Napoli’s work on petunia, 1996).

BRANCHING AND AUXIN CONTENT

Workers will often use the relative amount of branching (Stage IV) not only as an indicator of the degree of apical dominance release but also as a suggestive indicator of endogenous auxin content. There is nothing wrong with using branching as an indicator of the strength of apical dominance as long as it is realized in the case of branching released by decapitation that the hormonal and other conditions necessary for this release are present only during Stage III. Even though low auxin content in a shoot is correlated with the release of apical dominance in Stage III and the release of apical dominance is a prerequisite for branching, it does not follow that the presence of heavy branching in Stage IV is necessarily accompanied by low auxin content. In fact, as the developing branches produce their own auxin (Thimann and Skoog, 1934), the auxin content in Stage IV may well approach normal levels. That transgenic iaaL tobacco plants deficient in auxin exhibit reduced apical dominance (Romano, Cooper, and Klee, 1991) probably can be attributed to the low auxin content at the very early stages of bud formation.

The same misconception can occur with the employment of nondecapitated branching mutants (including their use in grafting experiments) for the same reasons as noted above. Beveridge, Ross, and Murfet (1994) found that branching pea mutants had consistently higher endogenous free IAA levels than the nonbranching wild types. Heavily branching plants should not necessarily be expected to reflect low endogenous auxin levels. Also, the relative concentration of cytokinins and other plant hormones as well as the sensitivity of tissues to them cannot be ignored.

QUANTIFICATION OF APICAL DOMINANCE AND ITS RELEASE

In many journal papers (particularly those in molecular biology) that in some way involve apical dominance research, no actual quantitative data on lateral bud outgrowth are given. A photograph showing branched and unbranched plants side by side may be included or, more often, the degree of dominance or branching under various conditions are verbally described with such terms as “increased branching,” “lateral growth,” “lateral shoot formation,” “bushy,” “shoozy phenotype,” “reduced or decreased apical dominance,” “nonbranching,” “unbranched growth habit,” “strong apical dominance,” or “restored apical dominance.”

Such qualitative visual or verbal evidence may be sufficient for many purposes. But in some cases, more quantitative and definitive evidence may be required. When comparing two plants, what does it mean to say that one plant has “more branching” than another? It is probably meant that one plant has more branches than the other. As experience in Christmas tree selection can attest, outward appearance of bushiness can be deceiving. A short plant with the same number of branches as a tall plant will appear bushy because of shorter internodes. A plant with long branches, particularly if they are floppy, may appear bushier than a plant with the same number of shorter branches. Secondary and inflorescence branching will further complicate the analysis. One plant could have more branches than another plant if the one plant had more nodes, assuming all buds at all nodes on both plants grew out. Or, one plant could have more branches if a greater percentage of its buds had sprouted.

Probably the most common way of detecting apical dominance release is to measure the outgrowth (i.e., bud length or fresh mass) of lateral shoots following decapitation or other restrictive treatments of the shoot apex. In the case of many herbaceous plants, measurable elongation of the lateral bud begins within 3–10 h following decapitation. The determination of whether or not apical dominance release at the morphological level has occurred depends upon whether or not the lateral bud begins to grow out. If the lateral bud has not begun to grow out, then it would be presumed that the release of apical dominance had not occurred. It is understood, of course, that earlier molecular and mitotic events associated with the release must occur before bud elongation can begin.

Since studies on the mechanisms of apical dominance release are aimed at understanding these underlying cellular, biochemical, and molecular events, it is critical that the appropriate determinations and measurements of these processes be made both before decapitation in Stage II and within minutes and hours after decapitation during Stage III. Continuous measurements of these processes and bud growth can be carried out during the following days (Stage IV), but it must be recognized that these data may not have strict correlation with the mechanisms of apical dominance release.

EVALUATING APICAL DOMINANCE IN ARABIDOPSIS

Arabidopsis is a difficult plant for apical dominance analysis for two reasons: Firstly, its shoot is an inflorescence with extremely weak apical dominance (i.e., very early branching in the intact bolting shoot). Secondly, there are two types of branches: (1) the inflorescence lateral branches, which begin to grow out at the same time as the main shoot bolts from a rosette base and (2) secondary or axillary inflorescences, which emerge shortly after the bolting of the main shoot. Can the release of apical dominance even be detected following decapitation of the main shoot? The answer to this question is yes, if
the decapitation is carried out immediately after bolting begins. There are, of course, quite a range of branching types among the various mutants and lines of *Arabidopsis*. The vast genetic data available on *Arabidopsis* certainly constitute a major advantage for the use of this system in branching studies. Mutants with relatively strong apical dominance include the strain 2100 (Torii and Komeda, 1993) and the CS 1072 Chi-O (Ohio State University *Arabidopsis* Biological Resource Center), whereas those with extremely weak apical dominance include the auxin resistant *axl*1–12 (Lincoln, Britton, and Estelle, 1990), the *trp*1–1 (Last and Fink, 1988), and the *amp1* (Chaudhury et al. 1993).

Branching from the main shoot is not much affected by decapitation in strain CS 1072 Chi-O (Cline, 1996) because of the extremely early lateral bud outgrowth in the intact plant. However, the lateral buds below the point of decapitation grow out longer than those of the controls. Nevertheless, the effect of decapitation of the main shoot on hastening the bolting of and increasing the number of bolting secondary inflorescences is significant and can be detected in this line. These results probably have general application to many other lines of *Arabidopsis*. However, Lincoln, Britton, and Estelle (1990) reported little difference in the total number of inflorescences between the bushy auxin-resistant *axl* mutant and the wild type, although there is an increase in the number of lateral shoots in the former.

In most plant species apical dominance release or lateral bud outgrowth are usually defined by periodic measurements of bud length. In the case of *Arabidopsis*, such measurements could become cumbersome because of the large number of secondary inflorescences that could be encountered. Romano, Cooper, and Klee (1993) used total fresh mass of secondary inflorescences which may be a viable alternative, although it does not distinguish between inflorescence number and branch length.

Although Romano, Cooper, and Klee (1993) did report inhibition of branching in transgenic *iaaM* auxin-over-producing plants, Cline (1996) was not able to restore apical dominance in *Arabidopsis* by exogenous auxin in lanolin treatment via the classical Thimann-Skoog experiment or by aqueous spray. There is concern about the general health of such high auxin-level transgenic plants, which exhibit severe leaf epinasty. Likewise, C. Lincoln (personal communication, Biology Department, Indiana University) was unable to restore apical dominance via exogenous auxin treatments in *Arabidopsis*. For future apical dominance studies with *Arabidopsis*, the availability of mutants or lines with strong apical dominance (e.g., *NAB 1*, Bohnert and Benning, 1996) would be helpful.

**CRITERIA FOR APICAL DOMINANCE ANALYSIS**

The ideal plant for analyzing apical dominance release with respect to carrying out measurements of lateral bud elongation is one with a rapidly growing shoot, moderately strong apical dominance (i.e., with inhibited lateral buds that will respond immediately to decapitation), lateral buds that are widely separated by large internodes so as to be easily observed and measured, and a shoot in which only the highest lateral bud will grow out following decapitation (providing for more uniformity in treatments and for a shorter transport distance for chemicals applied to the decapitated stump).

*Pisum sativum* probably has been the most widely used species in apical dominance studies (Wickson and Thimann, 1958; Beveridge, Ross, and Murfet, 1994; Stafstrom 1995). These plants are rapidly growing and offer many advantages. Japanese Morning Glory (*Ipomoea nil*) is also an excellent system (Cline, 1996). The shoot elongates at the rate of ~10 cm/d in constant light. The lateral buds in a 20- to 25-d-old plant average ~2–3 mm in length at the first through the fifth nodes (counting from the base). Following decapitation just above the fourth or fifth node, the beginning of elongation can be detected within 5–8 h (Yang and Cline, unpublished data). The lateral bud at the fourth node (which is similar to that of the fifth node) will have increased in length by about 2, 5, 17, and 329 mm after the 1st, 2nd, 3rd and 7th d, respectively (Cline and Riley, 1984). Plants with weak apical dominance, like *Coleus*, are difficult to study because buds on the intact plant are only slightly inhibited and, hence, are continually growing out. Decapitation only slightly hastens bud outgrowth and exogenous auxin added to the shoot stump of greenhouse-grown *Coleus* plants has no repressive effect (Jacobs et al., 1959; Cline, 1996).

An ideal branching mutant would be one that is exactly the same as the wild type in height, node number, and other characteristics, except that all the lateral buds of intact mutant would grow out, whereas all the lateral buds of the intact wild type would remain inhibited and could only be released by decapitation or by some other definitive treatment. If the hormone content or some type of molecular/cellular activity is to be compared between the branching mutant and the wild type plants, it must be remembered that in most situations the branching mutant will be similar in many respects to the Stage IV plants, which are far beyond the apical dominance release stage (III).

**APICAL DOMINANCE VS. APICAL CONTROL**

Although there is some disagreement in distinguishing between the meaning of the terms “apical dominance” and “apical control” (Martin, 1987; Loreti and Pisani, 1990; Wilson, 1990), the former is usually used in a narrow sense with respect to herbaceous plants to specify control (or correlative influence) by the shoot apex over auxillary bud outgrowth and in a broader sense also to include control over orientation of lateral branches and leaves as well as over the growth of branches, rhizomes, stolons, and tubers and, finally, correlative influence over leaf abscission (Hillman, 1984).

Brown, McAlpine, and Kormanik (1967) introduced the use of the term “apical control” for woody plants because of the complex branching associated with periodic sprouting of buds from year to year. Wareing (1970) has stated, “…the classical theory of apical dominance does not seem applicable to shoot systems of woody plants beyond the first year.” Brown used “apical control” to describe the physiological conditions that give rise to the overall shape and form of the tree crown via various branching patterns. The excurrent conifer (e.g.,
pine) has a single dominant central leader shoot and strong apical control, giving an attenuated-shaped crown. The individual shoots have weak apical dominance and the lateral buds of the current year grow out to varying degrees during the first season. If the growing point is somehow destroyed, then one of the remaining upper branches, in a Stage IV type response, replaces it by bending more vertically via processes in the reaction wood and by elongating. Ethylene, gibberellins, and auxin (Blake, Pharis, and Reid, 1980) may play a role here.

In the recurrent hardwoods (e.g., oak) with no dominant leader shoot and with weak apical control (except when very young), the lateral shoots outgrow the weak leader shoot to give a crown with a round shape. The individual shoots presumably have strong apical dominance and the lateral buds of the current season do not grow out until the following spring. Hence, when used with respect to trees, apical dominance is thought to refer to the control of lateral bud outgrowth by the apex in an individual branch during the current season’s growth (associated with the Stage III response), whereas apical control refers to the control over branch elongation and orientation (Stage IV events) and is concerned with “the influence of the main growing point on all branches of a perennial plant” (Martin, 1987) or as Bollmark et al. (1995) describe it, “the influence of the top of the tree on the branches lower down...”. However, as Davidson and Remphrey (1990) have pointed out, “The architecture of tree crowns may be more complex than the excurrent−decurrent dichotomy and its relationship to apical control.”

CONCLUSION

In their historical evaluation of adventitious rooting research, Haissig and Davis (1994) point out that “...there are no rewards, and often criticisms for those who attempt to develop terminologies.” In the present essay there has been no presentation of new terms. The perspective has been physiological and the attempt has been to redefine some of the old terms that relate to apical dominance such as “apical dominance release” and “branching” along with methods of quantification and to elucidate their usage in a way that, hopefully, will help generate discussion for the future and to avoid misconceptions.

LITERATURE CITED


