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Ethylene and apical dominance

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Abstract

Minireview

Ethylene is involved in at least two discrete mechanisms in the control of apical dominance: the release of apical buds from inhibition and their subsequent growth and development. Generally, high levels of diffusible ethylene in the apical region of the shoot are conducive to lateral bud outgrowth, while high ethylene levels in the region of the lateral buds themselves tend to be inhibitory. Threshold ethylene levels concerned with the release of buds from inhibition and in the growth that follows may differ between species. Thus, in some species (e.g. *Gossypium*) lateral bud growth proceeds in the continuing presence of ethylene supplied to the whole plant, whereas in others (e.g. *Petunia*) the growth of the released lateral buds occurs only when the ethylene is removed.

When ethylene production in *Pisum* nodal sections is enhanced by exogenous auxin, growth of the attached buds is suppressed. In the intact plant system, unequivocal evidence has not been established for a role of endogenous ethylene acting directly on lateral buds to effect their inhibition. Apical dominance is not affected by the application of ethylene antagonists to the lateral buds of intact plants. Results from different studies have been inconsistent regarding the changes in endogenous levels in the node/lateral bud tissue when the plant is decapitated or when auxin is applied to the stump of the decapitated plant to maintain lateral bud inhibition.

While exogenous ethylene supplied to the lateral bud generally increases inhibition, the availability of ethylene, regulated endogenously, is essential to the released bud on the decapitated plant in order to sustain its subsequent development into a lateral shoot. There is evidence that, in certain instances, endogenous ethylene may also be essential in the initial stages of bud development, e.g. in the early growth that is promoted by auxin in *Phaseolus* or by kinetin in *Avena.*

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Ethylene is involved in at least two discrete mechanisms in the control of apical dominance: the release of lateral buds from inhibition and their subsequent growth and development. Generally, high levels of freely diffusible ethylene in the apical region of the shoot are conducive to lateral-bud outgrowth, while high ethylene levels in the region of the lateral buds themselves tend to be inhibitory. Threshold ethylene levels concerned with the release of buds from inhibition and with the growth that follows may differ between species. Thus, in some species (e.g. *Gossypium*) lateral-bud growth proceeds in the continuing presence of ethylene supplied to the whole plant, whereas in others (e.g. *Petunia*) the growth of the released lateral buds occurs only when the ethylene is removed.

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Introduction

In dicotyledonous plants the primary meristems of the shoot are located in the shoot apex and lateral buds. Under favourable environmental conditions, the apical bud of vegetative herbaceous plants develops actively; the development of the lateral buds, on the other hand, is generally inhibited by the presence of the apex or young apical leaves. In the event that the apical bud is lost or incapacitated by damage, a lateral shoot assumes dominance.

Inhibition of lateral buds by the apex is one of several forms of correlative relationships which involve interactions between different organs and tissues of plants in the regulation and co-ordination of form and function. Plant growth substances are thought to play an important role in the correlative inhibition of lateral buds. Among the major groups of plant growth substances, ethylene is ubiquitous in the higher plants and influences numerous aspects of plant growth and differentiation (see Abeles 1973, Lieberman 1979).

Hitherto, research on ethylene in relation to the correlative inhibition of lateral buds has been largely neglected. Whereas attempts have been made from various physiological viewpoints to explain apical dominance, studies aimed specifically at elucidating the role

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of ethylene in this phenomenon are few. Nevertheless, ethylene involvement may be indirectly implicated or hypothesized from the results of other studies on apical dominance, but further investigations will be necessary to substantiate and expand on these incidental observations.

This paper reviews the research that has been carried out on the involvement of ethylene in the correlative inhibition of lateral-bud growth. The role of ethylene in apical dominance is further explored by references drawn from the known interactions and relationships of ethylene with other growth regulators.

Abbreviations – ACC, 1-aminopropane-1-carboxylic acid; AVG, aminoethoxyvinyl glycine; IAA, indole-3-acetic acid; TIBA, triiodobenzoic acid; vpm, volumes per million.

Ethylene-induced outgrowth of lateral buds

Hall et al. (1957) reported various abnormalities in the growth of cotton plants in the field which were later attributed to pollution of the air by ethylene. The symptoms shown by the affected plants included a marked loss of apical dominance. In subsequent laboratory tests, growth of lateral buds of cotton plants was similarly observed when the plants were treated with 10 vpm ethylene.

Following the exposure of *Petunia* plants to 100 vpm ethylene for 2–12 h, outgrowth of the normally inhibited lateral buds was recorded over the next 7 days (Burg 1973). The effect of exogenous ethylene in the release of apical dominance was comparable to that of decapitation of the plant. Lateral-bud development was triggered off only upon removal of the ethylene source; the gas inhibited outgrowth of the buds while it was present. Thus, no bud growth occurred in a treatment where the plants were exposed to ethylene continuously for 7 days. A similar effect of an ethylene pulse treatment has been reported in potato plants (Catchpole and Hillman 1976). Plants treated with 5 vpm ethylene for 8 days showed a loss of apical dominance 2 days after the ethylene supply was removed.

Besides supplying ethylene in its gaseous form, ethylene treatment can be effected by treatment with ethylene-releasing chemicals, e.g. ethephon (2-chloroethylenephosphonic acid). Ethephon increases lateral-bud development in a wide variety of ornamentals when applied at 0.7–35 mol m^{-3}, and in some rhizomatous species when applied at rates of 1.1–8.9 kg $hectare^{-1}$ (see review by de Wilde 1971).

Control by ethylene in the apical region of the shoot

Repeating the earlier work of Mulder (1941), Leong et al. (1976) induced branching in young rubber plants (*Hevea brasiliensis*) by covering the apical portion of the shoot with a paper bag. Folding and securing the uppermost whorl of leaves over the shoot apex achieved the same effect. Besides the well-documented enhancement of ethylene emanation by the wounding of plant tissue (see Abeles 1973, Yang and Pratt 1978), various mechanical stimuli shown to influence specific aspects of plant growth and develoment have also been attributed to ethylene (Goeschl et al. 1966, Turgeon and Webb 1971, Jaffe 1976, Mitchell 1977). As such, mediation by ethylene in the release of correlative inhibition arising from a physical stimulus in the apical portion of the shoot was suspected. Evidence in support of this proposition was derived from studies on *Phaseolus vulgaris*, where vigorous lateral-bud growth was recorded when the apical portion of the shoot was physically restricted (Hillman and Yeang 1979, Yeang and Hillman 1981b). Retardation of the growth of the shoot was observed, this being a common response in plants to ethylene. Analyses of internal ethylene (i.e. ethylene in the gaseous extract of the tissue – see Beyer and Morgan 1970) in the apical tissues provided further evidence for ethylene involvement, since the ethylene in physically confined shoots increased 2.4-fold, from 0.34 to 0.82 vpm. Enhancement of the rate of ethylene emanation in confined shoots was even more pronounced. Release of ethylene from the confined shoots was 8.28 $mm^3\ kg^{-1}\ h^{-1}$, compared with 1.81 $mm^3\ kg^{-1}\ h^{-1}$ in control shoots – a 4.6-fold increase.

The role of ethylene acting on the apical region of the shoot in releasing correlative inhibition was tested further by enclosing the apical portion of the shoot in a glass vessel supplied with 0.5 vpm ethylene (Hillman and Yeang 1979). The vessel was large enough not to constrict the shoot while it was treated with ethylene. Rapid outgrowth of lateral buds basipetal to the enclosure ensued, whereas the buds remained inhibited in the intact control and in those controls where the vessel was flushed with air or where endogenous ethylene released by the enclosed shoot was absorbed by mercuric perchlorate. Analyses of ethylene from ethylene-treated shoots and the various controls showed no significant difference in their rates of ethylene emanation (Yeang and Hillman 1981b). Internal ethylene, however, was higher in all enclosed shoots (irrespective of whether ethylene was supplied) than in intact, unenclosed controls. The effect of ethylene on the apical region of the shoot on correlative control of lateral-bud growth was again demonstrated by ethephon treatment. Dilute (0.35–1.4 mol m^{-3}) aqueous solutions of ethephon painted on the apical bud or expanding apical leaves of *Phaseolus vulgaris* resulted in the outgrowth of basipetally situated axillary buds (Hillman and Yeang 1979). Similarly, Leong et al. (1976) induced branching in young rubber plants by spray applications of 7–14 mol ethephon m^{-3} to the topmost flush of leaves.

Application of the ethylene-synthesis inhibitor aminoethoxyvinyl glycine (AVG) or of the silver ion (Ag^+) to the apical region of the shoot of *Phaseolus* had no effect on the inhibition of lateral buds. AVG reduced, by about half, the rate of ethylene emanation and internal ethylene. Ag^+ had no significant effect on ethylene

Tab. 1. The relationship between lateral-bud growth and three forms of endogenous ethylene from the apical portion of the shoot of *Phaseolus vulgaris*. Only in the case of intercellular ethylene is an increase consistently accompanied by an enhancement of lateral-bud growth, and *vice versa* (indicated by squares that are either completely filled or completely empty). a, 0.5 vpm ethylene; b, ethylene absorbed by mercuric perchlorate; c, vessel enclosing the shoot ventilated to preclude accumulation of endogenously released ethylene; d, 0.7–1.4 mol ethephon m^{-3}; e, 0.5 mol $Ag\,NO_3$ m^{-3}; f, 0.1 mol AVG m^{-3}. Adapted from Yeang and Hillman (1981b). ◤, Lateral-bud growth enhanced; ◢, endogenous ethylene enhanced; ◿, endogenous ethylene not determined.

Treatment to apical portion of the shoot	Endogenous ethylene		
	Internal	Emanated	Intercellular
Constriction	■	■	■
Ethylene-enhanced air[a]	■	◪	■
Ethylene-depleted air[b]	◪	□	□
Ventilation[c]	◪	□	□
Ethephon[d]	◪	◪	■
Ag^+[e]	◪	□	□
AVG[f]	□	□	□

emanation, it being an inhibitor of ethylene action rather than of ethylene synthesis. Ag^+ did, however, raise the level of internal ethylene in the treated shoot tissue (Yeang and Hillman 1981b).

To arrive at a working hypothesis compatible with all their findings pertaining to the status of ethylene in the apical region of the shoot, Yeang and Hillman (1981b) considered 3 ways by which ethylene levels in the apical portion of the shoot might be expressed: rate of ethylene emanation, intercellular ethylene [intercellular ethylene is similar to "free" ethylene described by Yeang and Hillman (1981b)] and internal ethylene. Intercellular ethylene may be enhanced either exogenously or endogenously. Treating the plant tissue with ethylene (or with ethephon) would obviously increase intercellular ethylene. Tissues showing an increase in the rate of ethylene release are deemed also to have a corresponding increase in intercellular ethylene since endogenous ethylene emanating from cells must first diffuse into the intercellular spaces before dissipating into the atmosphere. Internal ethylene, which refers to the concentration of ethylene in gases recovered from the plant tissue by extraction under reduced pressure (Beyer and Morgan 1970), incorporates intercellular ethylene, intracellular ethylene, and ethylene that is "compartmented" or otherwise "bound" (i.e. ethylene that is not readily released from the tissue; see Jerie et al. 1979). The occurrence of increased ethylene in the apical portion of the shoot of *Phaseolus* in relation to apical dominance is summarized in Tab. 1. Outgrowth of lateral buds was not dependent upon an increased rate of ethylene release from the apical portion of the shoot since vigorous bud growth was observed following ethylene treatment of the shoot, even though ethylene release from the same tissue was not increased. Internal ethylene was also not critical to the onset of lateral-bud outgrowth as internal ethylene was similarly enhanced by treatments which released apical dominance as well as those which did not. It was consistently observed, however, that release of correlative inhibition accompanied an increase in intercellular ethylene. It appears from the collated experimental data that the onset of lateral-bud growth is related to an availability of freely diffusible ethylene in the apical region of the shoot.

Ethylene in the region of lateral buds

Whereas the apical portion of the shoot has a major role in maintaining lateral buds in their inhibited state, the correlative signal that is transmitted might act on the buds themselves. Ethylene involvement in apical dominance might thus involve its control of lateral-bud development at the location of the bud and/or adjacent tissue.

Burg and Burg (1968a, b) found that exogenous ethylene, even in low concentrations, could suppress the growth of axillary buds of pea plants released from apical inhibition. Buds on isolated nodal sections or decapitated stem cuttings were partially inhibited by about 0.2 vpm ethylene and completely inhibited when the ethylene concentration was increased to 2 vpm or higher. Where ethylene was supplied for less than 3 days, buds on the nodal sections expanded normally when ethylene was subsequently withdrawn, indicating that no permanent damage had occurred. (Treatment for more than 3 days rendered the buds incapable of expansion when the ethylene was removed.) With *Phaseolus vulgaris*, 0.7–1.4 mol ethephon m^{-3} painted on the axillary bud and adjacent tissue inhibited outgrowth of the bud when the plant was decapitated (Yeang and Hillman 1982).

When it was first observed that ethylene evolution in etiolated pea plants was higher at the nodes than at the internodes, Burg and Burg (1968a) proposed that inhibition of the axillary buds situated at the nodes was due to endogenous ethylene. In a subsequent paper (Burg and Burg 1968b), they reported that although ethylene emanation from the node decreased upon decapitation of the plant, this decrease was not observed when the scale leaves at the node were first removed. A noteworthy point regarding the determination of endogenous ethylene in the inhibited bud is that, in many species, isolating the bud from the plant apex for this purpose effectively releases it from inhibition. In *Pisum*, subsequent growth increment and mitotic activity in the bud can take place within 6–12 h (Wardlaw and Mortimer 1971, Nagao and Rubinstein 1976). Hence, if the period between bud isolation and ethylene sampling were prolonged (e.g. 21 h in Burg and Burg's experiment), there would be uncertainty as to whether the ethylene levels

relate to the condition of sustained bud inhibition or release of the bud from inhibition.

Yeang and Hillman (1982) re-examined the role of ethylene at the nodal region in relation to lateral-bud inhibition, taking the precaution of carrying out the analyses of endogenous ethylene within 27 min of excision of the nodal and internodal test tissues from *Phaseolus vulgaris* plants. Unequivocal evidence for the participation of ethylene in lateral-bud inhibition was not found. No consistent differences were observed between the rates of ethylene emanation from nodal and internodal tissues. Internal ethylene in the nodal tissues was, as a rule, higher than in internodal tissues, but the difference (ca 25%) was not marked and might be attributed largely to morphological differences between nodal and internodal tissues (see Yeang and Hillman 1981a, 1982). While Burg and Burg (1968b) found no decrease in ethylene emanation from the node (with scale leaves removed) when *Pisum* plants were decapitated, both ethylene emanation and internal ethylene in nodal and internodal tissues declined when *Phaseolus* plants were decapitated. It must be noted, however, that the decline in endogenous ethylene was not limited specifically to the node; decapitation essentially brought about a decrease in endogenous ethylene in the stem as a whole. This development is comparable to the decrease in the rate of ethylene release reported in *Phaseolus* hypocotyls following decapitation (Abeles and Rubinstein 1964).

An abstract of the findings of Blake and Reid (1981) indicated a decrease in ethylene evolution from the nodal region of *Pisum* plants when the plants were decapitated; experimental details were not given in this note.

If the development of lateral buds were inhibited by endogenous ethylene present in the region of the bud, it might be supposed that treatment with anti-ethylene agents would overcome the inhibition. However, endogenous ethylene is closely associated with the processes of active plant growth and development. Non-selective interference of essential ethylene-related processes at the cellular level could suppress normal growth and development. It was perhaps for this reason that AVG (0.01 mol m^{-3}) or Ag^+ (0.1 to 1 mol m^{-3}) applied to the lateral buds of intact *Phaseolus* plants had no effect on apical dominance (H. Y. Yeang, 1980. Thesis, Univ. of Glasgow, Glasgow, U.K.) and that higher dosages (0.4 mol AVG m^{-3} or 4 mol Ag^+ m^{-3}) inhibited active growth of the buds on decapitated plants (Yeang and Hillman 1982).

Control of bud outgrowth by auxin-induced ethylene

Interactions between ethylene and other classes of phytohormones are known, and the most significant interactions occur with auxin. The promotion of ethylene production in plant tissue by auxin is a well-studied and widespread phenomenon. Several (but by no means all) of the effects of auxins in the plant have been attributed to auxin-induced ethylene (see Abeles 1973, Lieberman 1979). The many research findings implicating auxin in the correlative control of lateral-bud growth lends speculation to the possibility of a role of auxin that is mediated *via* ethylene.

In experiments where excised pea nodal sections were incubated in different concentrations of indole-3-acetic acid (IAA) solutions, Burg and Burg (1968b) found that the degree and duration of inhibition of bud outgrowth correlated well with the induction of ethylene formation by the various IAA concentrations. As already mentioned, treatment of the buds with ethylene alone produced a similar inhibitory effect. Burg and Burg (1968a) reported on this basis that ethylene production could account for the inhibitory action of applied auxin. There is a basis also to suppose, therefore, that in the intact plant, apically synthesized IAA could be translocated to the region of the lateral bud where it promotes ethylene production and thus maintains growth inhibition. This mode of auxin action would also explain the oft-repeated demonstration of sustained apical dominance in the decapitated plant by the substitution of the shoot apex with exogenous IAA or other auxins. Consistent with this proposition, Bourbouloux (1978) showed that IAA applied to the cut stem of decapitated *Faba vulgaris* had a tendency to be translocated basipetally and accumulate at the nodes. Moreover, isolated segments of nodal tissue of the pea plant appeared to be more sensitive than internodal tissue to the stimulation of ethylene production by auxin (Burg and Burg 1968b). Parenthetically, ethylene production by scale leaves at the node accounted to a considerable extent for the difference in response to auxin between nodal and internodal tissue. But even with the scale leaves removed, nodal tissue was still better predisposed to the stimulation of ethylene production, especially at higher (e.g. 0.1 mol m^{-3}) IAA concentrations.

Yeang and Hillman (1982) investigated the involvement of auxin-induced ethylene in the correlative inhibition of lateral buds of *Phaseolus vulgaris* by measuring endogenous ethylene from nodal and internodal tissues of decapitated plants with IAA applied to the cut-stem surfaces. Their findings did not substantiate the hypothesized auxin-ethylene interaction in maintaining apical dominance. As expected, ethylene emanation from internodal tissue near the site of IAA application was enhanced some 3-fold as compared with the same tissue in decapitated plants where IAA was not supplied. However, there was no increase in ethylene emanation from nodal tissue which was located further down the stem. Internal ethylene level in the nodal tissues of IAA-treated plants was similarly comparable to the corresponding tissues in decapitated plants not supplied with IAA. These findings are compatible with the report of Hall and Hillman (1975) who noted that while tritiated IAA applied to the cut surface of the decapitated stem inhibited axillary-bud growth in *Phaseolus*, basipetal translocation of IAA into the axillary buds was poor and

breakdown of the auxin was extensive during the 48 h following removal of the apex.

Contrary to the findings in *Phaseolus*, Blake and Reid (1981) who carried out a similar type of experiment on *Pisum*, reported that ethylene evolution in the nodal region of the decapitated stem increased greatly when IAA was supplied to the stump. Full experimental details were not available in this abstract.

Although the effect of auxin on lateral-bud growth is, in the main, inhibitory, 0.1 mol IAA m^{-3} applied to the lateral bud of *Phaseolus* promoted bud growth over the first 2 days, the growth increment approaching that due to decapitation (Yeang and Hillman 1982). This enhanced growth was not sustained thereafter, and it was suspected that the growth-promoting effect of IAA was curtailed by the antagonistic effect of IAA-induced ethylene production from the treated tissue. It was thought, therefore, that simultaneous treatment with AVG would overcome the hypothesized ethylene inhibition. Unexpectedly, instead of a sustained growth enhancement, the growth-promoting effect of IAA was lost altogether when combined with 0.01 mol AVG m^{-3}. Treatment with AVG did not affect the normal slow growth increment of the inhibited bud on the intact plant. Only the additional growth over the first 2 days that was due to IAA was eliminated. These results have hence not established any grounds to suggest that accelerated development of the lateral bud resulting from IAA treatment was prevented by induced endogenous ethylene. On the contrary, growth promotion was prevented by AVG, suggesting that the promotion was ethylene-dependent and perhaps brought about by IAA-induced ethylene in the first instance. There is some doubt, however, as to whether growth promotion in IAA-treated buds, even in the initial phase, is similar to that in decapitated plants. In the first instance, IAA-promoted growth of the axillary buds was not sustained beyond 2–3 days (although this could be due to a depletion of the auxin as IAA application was not repeated). Secondly, the enhancement of growth by IAA was completely annulled by AVG while that due to decapitation was unaffected. The transient, accelerated growth of IAA-treated buds is perhaps more comparable to the promotion of extension growth in internodes of inhibited shoots (Sachs and Thimann 1967), in view of the fact that the *Phaseolus* bud was well developed and possessed a defined internode.

A study by Harrison and Kaufman (1982) on correlative inhibition in *Avena* provides more information in the requirement of ethylene by the lateral bud during the early stages of its release from inhibition. Increased endogenous ethylene accompanied the onset of bud swelling preceding tiller outgrowth, both when the plants were placed in a horizontal position as well as during inflorescence emergence. The outgrowth of buds also occurred when stem segments in a sucrose medium were treated with kinetin during the first 24 h after isolation. This development was inhibited by the ethylene inhibitors AVG or carbon dioxide. Conversely, pulse dosages of ethylene or the ethylene precursor 1-aminocyclopropane-1-carboxylic acid (ACC) could partially replace the initial kinetin requirement Harrison and Kaufman (1982) suggested that ethylene could act by promoting the swelling phase of bud development and by stimulating kinetin-promoted elongation in the tiller shoots.

Exogenous application of IAA inhibited kinetin-induced bud development in *Avena* stem segments, as did the continuous presence of ethylene. This response was similar to that of isolated *Pisum* buds (Burg and Burg 1968a, b) but it was not determined if endogenous ethylene in *Avena* stem segments was enhanced by the IAA treatment.

An overview

In as far as the phenomenon of apical dominance is concerned with the processes governing the inhibition of lateral-bud growth and the onset of growth following removal of the inhibition, it is not difficult to visualize ethylene playing a crucial regulatory role. Ethylene is both an inhibitor of cell division and extension growth, while at the same time, it is also essential to cytodifferentiation in certain plant tissues and is a natural cellular product of active growth and development (see Lieberman 1979). Because ethylene has such diverse effects on the control of physiological mechanisms in the plant, it is not always easy to devise experiments which isolate and demonstrate the specific action of ethylene, devoid of extraneous influences arising from ethylene interactions in other areas. For example, when test-plant materials are supplied with ethylene (especially in high, non-physiological dosages), it may not be immediately clear if a resultant inhibitory response is an accurate simulation of natural correlative inhibition, or if it arises from a generalized effect of ethylene. Similarly, it may be ambiguous whether the application of ethylene inhibitors specifically modifies correlative control, or if the observed effects arise from the interference, and hence suppression, of cellular growth and development. These considerations should be accorded appropriate weight in the interpretation of experimental results relating to the involvement of ethylene in apical dominance.

The observation that lateral buds in undecapitated *Petunia* and potato plants are able to develop rapidly when treated with ethylene, but do so only when the stimulatory ethylene supply is subsequently removed, points towards at least two discrete mechanisms by which apical dominance is released. In the first instance, the physiological signal which imposes and maintains inhibition of lateral-bud development is nullified by ethylene. Subsequently, in the absence of inhibitory levels of ethylene, onset of growth of the lateral buds occur. It is not universally the case that lateral buds do not sprout in the presence of a concentration of ethylene sufficient to release apical inhibition. In the case of cotton plants,

onset of lateral-bud development occurs even in the continuing presence of the exogenous source of ethylene supplied to the whole plant (Hall et al. 1957). This might be indicative of a higher threshold level at which ethylene inhibits bud growth in cotton plants as compared with, for instance, *Petunia* or potato plants. Ethephon treatment of "whole plants" has also been reported to be effective in removing apical inhibition of lateral-bud growth (see de Wilde 1971). It is possible that in many such cases the effect of ethephon was essentially in the apical tissues. Ethephon, being primarily translocated in the acropetal direction (Weaver et al. 1972), can conceivably accumulate in the apical tissues where it acts to release apical dominance. Especially where overhead spray applications were employed, the lateral buds themselves could well be protected by foliage from substantial direct contact with the chemical.

Where does ethylene act to release correlative inhibition and where does it act to prevent bud break in released buds under experimental conditions? The term "apical dominance" itself already denotes the widely held belief that control of lateral-bud growth originates from the apical region of the shoot. Control of apical dominance by ethylene acting on the apical tissues of *Phaseolus vulgaris* has been experimentally verified by treating the apical portion of the shoot with ethylene but leaving the basipetally situated lateral buds free of ethylene (Hillman and Yeang 1979, Yeang and Hillman 1981b). The promotion of lateral-bud development apparently depends on the availability of freely diffusible ethylene in the treated apical tissues. It may be inferred from the reports of Burg and Burg (1968a, b) and Yeang and Hillman (1982) that at least some control of the outgrowth of lateral buds, once they are released from inhibition, is localized at the bud itself and/or adjacent nodal tissue. In the general case, high levels of freely diffusible ethylene in the apical region of the shoot are conducive to lateral-bud outgrowth while high ethylene levels at the region of the lateral bud itself are inhibitory.

One salient feature of ethylene treatment of the apical portion of the shoot is the reduction in growth of the treated tissue. This may be compared with the effects of other experimental treatments which induce lateral-bud growth but where decapitation is not carried out, e.g. removal of a ring of bark from the stem (Phillips 1975), applying auxin-transport inhibitors such as triiodobenzoic acid (TIBA) or morphactins to the stem (Panigrahi and Audus 1966, White and Hillman 1972), or treating the apical region of the shoot with abscisic acid (Aung and Byrne 1978). In these instances, growth of the apical portion of the shoot is also inhibited. It might be argued, therefore, that the inhibition of apical growth is the underlying cause of lateral-bud outgrowth, and that ethylene treatment is merely one of several methods of achieving inhibition of apical growth. It is conceivable that with the reduction of shoot growth, nutrients and other growth factors are then channelled to the lateral buds where compensation growth subsequently takes place; a mechanism essentially conforming to the "nutrient theory" (see McIntyre 1977). This proposition remains a possibility. If compensation growth does not initiate lateral-bud development, it would at least contribute toward the rapid growth of released buds. On the other hand, it has been observed that enclosure of the apical portion of the shoot of *Phaseolus* (without physical constriction and without adding ethylene) can significantly inhibit growth of the enclosed shoot without promoting axillary bud growth (Hillman and Yeang 1979). Conversely, a large proportion of lateral buds of the rhizomatous species *Agropyron* can be induced to sprout when the plant is teated with ethephon, yet inhibition of the apex is not always apparent (Parker 1976).

If, in promoting lateral-bud development, it is supposed that ethylene plays a more specific role in the apical tissues than merely inhibiting apical growth, what would this role be? With the physiology of ethylene action still a subject of much debate, a proposed role of ethylene in this instance will be largely a matter of conjecture. It would appear that the absence of physiological transport of ethylene (Zeroni et al. 1977, Jerie et al. 1978) renders it improbable that ethylene applied to the apical region of the shoot is transferred to the bud. Any assertion of the lack of ethylene transport should, however, be qualified by the amount of ethylene that is regarded as physiologically significant. The concentration of ethylene required to restore normality of growth habit to the ethylene-deficient mutant tomato plant (Zobel 1973) and that required for cytodifferentiation in lettuce pith cells (Zobel and Roberts 1978) are of an order of 0.005 vpm. Thus, while it can be demonstrated that mass transport of ethylene does not take place in the plant, movement of small, yet physiologically active quantities of ethylene or ethylene metabolites can certainly pass undetected by presently available analytical techniques. Even assuming the absence of ethylene transport, high ethylene concentration at one location of the plant is able to induce an increase in ethylene production elsewhere (Zeroni et al. 1977, Bradford and Yang 1980), possibly through the translocation of ethylene precursors (Bradford and Yang 1980).

The negative effects on bud growth when ethylene or ethephon is applied to the bud directly (Burg and Burg 1968a,b, Yeang and Hillman 1982) argues against a direct involvement of ethylene at the bud in promoting its outgrowth. Zobel (1974) encountered a broadly comparable situation in his work with the ethylene-deficient tomato mutant. He found that while treatment of the entire plant with a low concentration of ethylene induced normal development of the otherwise non-branching roots, ethylene treatment to isolated roots in culture did not produce a similar effect. He concluded that (a) ethylene induced the production of an active "substance" that was produced in the shoot and transported basipetally to the roots and (b) the roots did not produce this substance themselves. Drawing a parallel in the present study, ethylene in the apical region of the shoot may

be modified in some manner or may modify other factors to bring about a change in the correlative signal to the basipetal lateral buds. Taking a hypothetical stance, ethylene can act indirectly through its effect in reducing auxin transport. Auxin plays a central role in both the "direct" and "indirect" theories of auxin-induced bud inhibition (see Phillips 1975). Auxin also forms the basis of the "hormone-directed transport" theory, and a paper by Patrick and Wareing (1978) proposes that a continuous auxin stream from apex to inhibited bud is necessary for the maintenance of IAA-promoted acropetal transport of metabolites. Application of ethylene to the shoot could cause the breakdown of these auxin-mediated mechanisms which maintain bud inhibition. In fact, a range of other auxin-transport inhibitors besides ethylene, viz. morphactins (Schneider 1970), TIBA (Panigrahi and Audus 1966), α-naphthylphthalamic acid (naptalam; Morgan 1964), 3-phenacylidine phthalide hydrate (Brown et al. 1972) and 3,3a-dihydro-2-(*p*-methoxyphenyl)-8H-pyrazolo-(5,1*a*)-isoindol-8-one (DPX 1840; Beyer 1972), have all been shown to release apical dominance. It is tenable that the effect of ethylene on correlative inhibition might be by a similar mechanism.

Direct application of ethephon to the inhibited lateral bud of intact (undecapitated) *Phaseolus vulgaris* showed that bud outgrowth was not stimulated by ethylene even when steps were taken to avoid donating a supra-optimal dosage (Hillman and Yeang 1979). The application of the ethylene inhibitors Ag^+ and AVG to the lateral bud of the decapitated plant also inhibited development of the bud. In most of the buds a limited initial growth took place before further development was curtailed. It appears that the requirement for ethylene at the location of the bud was not so much associated specifically with the onset of axillary-bud outgrowth, but with the processes relating to subsequent tissue development; for example, the inhibitory effect of ethylene on cell division is known (Apelbaum and Burg 1972). In general, therefore, ethylene in the region of the lateral bud is essential for maintaining its normal growth on the decapitated plant, but the absence of bud outgrowth in the intact plant is not due to a lack of freely diffusible ethylene.

Because of the extensive range of physiological activities in the plant which are mediated by ethylene, many aspects of ethylene-related physiology which could have a bearing on apical dominance are yet to be investigated. For example, red and far-red light treatments affect apical dominance in some plants such as *Nicotiana* (Kasperbauer 1971) and *Xanthium* (Tucker and Mansfield 1972). Apical dominance in *Pisum* is diminished by increasing the carbon dioxide content of the air (Andersen 1976). Both the treatments with red/far-red light (Goeschl et al. 1967, Saimy 1978) and with carbon dioxide (Aharoni et al. 1979) modify ethylene production and/or action in plants. The phenomenon of gravimorphism, in which growth is promoted in the upward-orientated lateral buds on stems and branches trained into a horizontal position, might be mediated through stress-induced ethylene. Abeles and Gahagan (1968) reported that *Coleus* plants placed in a horizontal position produced more ethylene than plants in the normal upright position. Branches of *Pinus strobus, Pyrus malus* and *Prunus persica* tied in arcs showed significant increases in their internal ethylene content (Leopold et al. 1972). As already mentioned, hormone-directed transport of nutrients and other growth factors has been proposed as a mechanism for the apical control of lateral-bud growth. Some work on ethylene involvement in this area has already been initiated by Mullins (1970) although the usefulness of the study is compromised by the very high concentrations of ethylene used (undiluted or 500 vpm).

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References

Abeles, F. B. 1973. Ethylene in Plant Biology. – Academic Press, New York and London.
– & Gahagan, H. E. 1968. Accelerated abscission of *Coleus* petioles by placing plants in a horizontal position. – Life Sci. 7: 653–655.
– & Rubinstein, B. 1964. Regulation of ethylene evolution and leaf abscission by auxin. – Plant Physiol. 39: 963–969.
Aharoni, N., Anderson, J. D. & Lieberman, M. 1979. Production and action of ethylene in senescing leaf discs. Effect of indoleacetic acid, kinetin, silver ion and carbon dioxide. – Plant Physiol. 64: 805–809.
Andersen, A. S. 1976. Regulation of apical dominance by ethephon, irradiance and CO_2. – Physiol. Plant. 37: 303–308.
Apelbaum, A. & Burg, S. P. 1972. Effect of ethylene on cell division and deoxyribonucleic acid synthesis in *Pisum sativum*. – Plant Physiol. 50: 117–124.
Aung, L. H. & Byrne, J. M. 1978. Hormones and young leaves control development of cotyledonary buds in tomato seedlings. – Plant Physiol. 62: 276–279.
Beyer, E. M., Jr. 1972. Auxin transport: a new synthetic inhibitor. – Plant Physiol. 50: 322–327.
– & Morgan, P. W. 1970. A method for determining the concentration of ethylene in the gas phase of vegetative plant tissue. – Plant Physiol. 46: 352–354.
Blake, T. J. & Reid, D. M. 1981. Ethylene, IAA and apical dominance in peas. – Plant Physiol. 67 (suppl.): 52.
Bourbouloux, A. 1978. Auxin transport and apical dominance. – In Abstract of Papers: Inaugural Meeting of the Federation of European Societies of Plant Physiology, Edinburgh. pp. 105–107.
Bradford, K. J. & Yang, S. F. 1980. Xylem transport of 1-aminocyclopropane-1-carboxylic acid, an ethylene precursor, in waterlogged tomato plants. – Plant Physiol. 65: 322–326.
Brown, B. T., Johansen, O. & Sasse, W. H. F. 1972. New inhibitors of auxin transport. – Experientia 28: 1290–1291.
Burg, S. P. 1973. Ethylene in plant growth. – Proc. Natl. Acad. Sci. USA 70: 591–597.
– & Burg, E. A. 1968a. Auxin stimulated ethylene formation: Its relationship to auxin inhibited growth, root geotropism and other plant processes. – *In* Biochemistry and Physiology

of Plant Growth Substances (F. Wightman and G. Setterfield, eds), pp. 1275–1294. The Runge Press Ltd., Ottawa.
– & Burg, E. A. 1968b. Ethylene formation in pea seedlings: Its relation to the inhibition of bud growth caused by indole-3-acetic acid. – Plant Physiol. 43: 1069–1074.
Catchpole, A. H. & Hillman, J. R. 1976. The involvement of ethylene in coiled sprout disorder of potato. – Ann. Appl. Biol. 83: 413–423.
de Wilde, R. C. 1971. Practical applications of (2-chloroethyl) phosphonic acid in agricultural production. – HortScience 6: 364–370.
Goeschl, J. D., Rappaport, L. & Pratt, H. K. 1966. Ethylene as a factor regulating the growth of pea epicotyls subjected to physical stress. – Plant Physiol. 41: 877–884.
–, Pratt, H. K. & Bonner, B. 1967. An effect of light on the production of ethylene and the growth of the plumular portion of etiolated pea seedlings. – Plant Physiol. 42: 1077–1080.
Hall, S. M. & Hillman, J. R. 1975. Correlative inhibition of lateral bud growth in *Phaseolus vulgaris* L. Timing of bud growth following decapitation. – Planta 123: 137–143.
Hall, W. C., Truchelut, G. B., Leinweber, C. L. & Herrero, F. A. 1957. Ethylene production by the cotton plant and its effects under experimental and field conditions. – Physiol. Plant. 10: 306–317.
Harrison, M. A. & Kaufman, P. B. 1982. Does ethylene play a role in the release of lateral buds (tillers) from apical dominance in oats? – Plant Physiol. 70: 811–814.
Hillman, J. R. & Yeang, H. Y. 1979. Correlative inhibition of lateral bud growth in *Phaseolus vulgaris* L. Ethylene and the physical restriction of apical growth. – J. Exp. Bot. 30: 1079–1083.
Jaffe, M. J. 1976. Thigmomorphogenesis: A detailed characterization of the response of beans (*Phaseolus vulgaris* L.) to mechanical stimulation. – Z. Pflanzenphysiol. 77: 437–453.
Jerie, P. H., Zeroni, M. & Hall, M. A. 1978. Movement and distribution of ethylene in plants in relation to the regulation of growth and development. – Pestic. Sci. 9: 162–168.
–, Shaari, A. R. & Hall, M. A. 1979. The compartmentation of ethylene in developing cotyledons of *Phaseolus vulgaris* L. – Planta 144: 503–507.
Kasperbauer, M. J. 1971. Spectral distribution of light in a tobacco canopy and effects of end-of-day light quality on growth and development. – Plant Physiol. 47: 775–778.
Leong, W., Leong, H. T. & Yoon, P. K. 1976. Some branch induction methods for young buddings. – Rubber Research Institute of Malaysia, Kuala Lumpur, Malaysia. pp. 1–20.
Leopold, A. C., Brown, K. M. & Emerson, F. H. 1972. Ethylene in the wood of stressed trees. – HortScience 7: 715.
Lieberman, M. 1979. Biosynthesis and action of ethylene. – Annu. Rev. Plant Physiol. 30: 533–591.
McIntyre, G. I. 1977. The role of nutrition in apical dominance. – *In* Integration of Activity in the Higher Plant. The Society for Experimental Biology Symposium No. 31 (D. H. Jennings, ed.), pp. 251–273. Cambridge University Press. ISBN 0-521-21617-6.
Mitchell, C. A. 1977. Influence of mechanical stress on auxin-stimulated growth of excised pea stem section. – Physiol. Plant. 41: 129–134.
Morgan, D. G. 1964. Influence of α-naphthylpthalamic acid on the movement of indolyl-3-acetic acid in plants. – Nature 201: 476–477.
Mulder, H. H. 1941. De nieuwe "domper-kroon-ontwikkelingsmethode" bij jonge rubber. – Bergcultures 15: 472–474.
Mullins, M. G. 1970. Hormone directed transport of assimilates in decapitated internodes of *Phaseolus vulgaris* L. – Ann. Bot. 34: 897–909.
Nagao, M. A. & Rubinstein, B. 1976. Early events associated with lateral bud growth of *Pisum sativum* L. – Bot. Gaz. 137: 39–44.
Panigrahi, B. M. & Audus, L. 1966. Apical dominance in *Vicia faba*. – Ann. Bot. 30: 457–473.
Parker, C. 1976. Effects on the dormancy of plant organs. – *In* Herbicides: Physiology, Biochemistry, Ecology, 2nd Ed. (L. J. Audus, ed.), Vol. 1, pp. 165–190, Academic Press. ISBN 0-12-067702-4.
Patrick, J. W. & Wareing, P. F. 1978. Auxin promoted transport of metabolites in stems of *Phaseolus vulgaris* L. Effects remote from the site of hormone application. – J. Exp. Bot. 29: 359–366.
Phillips, I. D. J. 1975. Apical dominance. – Annu. Rev. Plant Physiol. 26: 341–367.
Sachs, T. & Thimann, K. V. 1967. The role of auxins and cytokinins in the release of buds from apical dominance. – Am. J. Bot. 54: 136–144.
Samimy, C. 1978. Effect of light on ethylene production and hypocotyl growth of soybean seedlings. – Plant Physiol. 61: 772–774.
Schneider, G. 1970. Morphactins: Physiology and performance. – Annu. Rev. Plant Physiol. 21: 499–536.
Tucker, D. J. & Mansfield, T. A. 1972. Effects of light quality on apical dominance in *Xanthium strumarium* and the associated changes in endogenous levels of abscisic acid and cytokinins. – Planta 102: 140–151.
Turgeon, R. & Webb, J. A. 1971. Growth inhibition by mechanical stress. – Science 174: 961–962.
Wardlaw, I. F. & Mortimer, D. C. 1970. Carbohydrate movement in pea plants in relation to axillary bud growth and vascular development. – Can. J. Bot. 48: 229–237.
Weaver, R. J., Abdel-Gawad, H. A. & Martin, G. C. 1972. Translocation of 1,2-^{14}C-(2-chloroethyl)-phosphonic acid (ethephon) in Thompson seedless grapes. – Physiol. Plant. 26: 13–16.
White, J. C. & Hillman, J. R. 1972. On the use of morphactin and triiodobenzoic acid in apical dominance studies. – Planta 107: 257–260.
Yang, S. F. & Pratt, H. K. 1978. The physiology of ethylene in wounded plant tissue. – *In* Biochemistry of Wounded Plant Storage Tissues (G. Kahl, ed.), pp. 595–622. Walter de Gruyter & Co., Berlin. ISBN 3-11-006801-X.
Yeang, H. Y. & Hillman, J. R. 1981a. Extraction of endogenous ethylene and ethane from leaves of *Phaseolus vulgaris* L. – J. Exp. Bot. 32: 381–394.
– & Hillman, J. R. 1981b. Control of lateral bud growth in *Phaseolus vulgaris* L. by ethylene in the apical shoot. – J. Exp. Bot. 32: 395–404.
– & Hillman, J. R. 1982. Lateral bud growth in *Phaseolus vulgaris* L. and the levels of ethylene in the bud and adjacent tissue. – J. Exp. Bot. 33: 111–117.
Zeroni, M., Jerie, P. H. & Hall, M. A. 1977. Studies on the movement and distribution of ethylene in *Vicia faba* L. – Planta 134: 119–125.
Zobel, R. W. 1973. Some physiological characteristics of the ethylene-requiring tomato mutant diageotropica. – Plant Physiol. 52: 385–389.
– 1974. Control of morphogenesis in the ethylene-requiring tomato mutant diageotropica. – Can. J. Bot. 32: 735–741.
– & Roberts, L. W. 1978. Effects of low concentration of ethylene on cell division and cytodifferentiation in lettuce pith explants. – Can. J. Bot. 56: 987–990.

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